



# Atlas of Gastrointestinal Endoscopy and Related Pathology

Klaus F. R. Schiller

Roy Cockel

Richard H. Hunt

Bryan F. Warren

Foreword by Peter B. Cotton

Second Edition

*b*  
Blackwell  
Science

**Atlas of  
Gastrointestinal  
Endoscopy and  
Related Pathology**

**To Sidney Truelove**

President, British Society for Digestive Endoscopy at its foundation,  
and later President, British Society of Gastroenterology, gastroenterologist,  
teacher, colleague and friend, in appreciation and with thanks.

# Atlas of Gastrointestinal Endoscopy and Related Pathology

**Klaus F.R. Schiller**  
**Roy Cockel**  
**Richard H. Hunt**  
**Bryan F. Warren**

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WITH THE COLLABORATION OF

**Martin G. Lombard**  
**Anthony I. Morris**  
**A. John Morris**  
**Thomas Rösch**

FOREWORD BY

**Peter B. Cotton**

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# Foreword

Like the authors of this book (and this writer), endoscopy has changed considerably over the last three decades. The early years of youthful excitement, pioneering (and some mistakes) led to a period of adult confidence and reasonable competence. Now, in maturity, there is the fun and obligation of reflection, and perhaps the beginnings of wisdom. What are now the main issues?

Endoscopy has become mainstream as other exciting new diagnostic and therapeutic techniques emerge and evolve towards practicality. Whilst we must embrace any developments which may have benefit for our patients, the imperative for endoscopy leaders must be to encourage enhanced efficiency and quality in endoscopic services. We are all aware that there are widespread problems of omission and commission, and that not all patients are optimally served.

There are three fundamental elements in this agenda: initial training, continuous quality improvement and patient empowerment. Initially we need to learn how to do endoscopy properly, and then continuously to strive to improve our efficiency and outcomes, and to make our patients partners in these endeavours.

Training programs are gradually becoming more thoughtful and structured, with less reliance on learning ‘by osmosis’ at the possible expense of our patients. Understanding what can be done, and what we are doing, is being facilitated by the increasing availability of community outcomes data derived from the wider use of structured endoscopy reporting systems. The fact that we and endoscopy are not perfect must be shared openly with our patients. Not even the experts claim 100% success and safety. Patients deserve to know more about the practice and competencies of individual endoscopists so that they can make informed choices. I strongly support the use of ‘report cards’, using fairly simple quality metrics, and the development of practice benchmarks.

Where does this new book fit in? Clearly, it is an important contribution for endoscopic trainees, and for all those involved in the endoscopy process. It is a clear and vividly illustrated guidebook to endoscopic appearances and to the major procedures, and the endoscopist will be well served by the inclusion of so much pathological material and the helpful introduction to endoscopic ultrasound. This book will undoubtedly find a place amongst other available learning resources.

I congratulate the authors on bringing this work to completion, and recommend it to the endoscopy community.

Peter B. Cotton  
Director, Digestive Disease Center  
Medical University of South Carolina, USA

# Preface

In 1986, three of us (KFRS, RC and RH) produced a volume entitled *A Colour Atlas of Gastrointestinal Endoscopy* which was published under the imprint of Chapman & Hall Medical. Since then there have been many developments in gastroenterology and endoscopy. We therefore felt that the time had come to update and to expand the previous Atlas and also to broaden our approach. With the addition of BFW, the original trio has become a quartet and we have a new title for this second edition, *Atlas of Gastrointestinal Endoscopy and Related Pathology*. We also have a new publisher, Blackwell Science.

As endoscopy is now universally established we felt that it was inappropriate to include, in an Atlas, any discussion on such topics as the principles of endoscope design, methods of recording visual data, the design of endoscopy rooms or the organization of an endoscopy service. These and other matters, including detailed descriptions of technique and of endoscope sterilization, are covered in other publications, many of which are listed in the section on Bibliography and Other Information.

A fuller discussion of the diseases mentioned, their differential diagnosis and of the various methods of investigation and treatment available is also beyond our remit. We do not dwell in depth on the merits of endoscopy versus classical barium radiology or the newer non-invasive radiological techniques. We do, however, recognize the contribution of endoscopic ultrasonography to clinical practice and a chapter is devoted to this subject.

The place of enteroscopy remains uncertain: it may never become established in all hospitals but is likely to find a permanent niche in reference centres. A chapter on this growing subject has therefore been included.

We could have added a chapter on Growing Points. This might, for example, have included such topics as virtual colonoscopy, endoscopic fluoroscopic spectroscopy, non-visual biosensors, the possible uses of very small robots, and experimental therapeutic techniques such as endoscopic gastroplasty. Every practising endoscopist should be aware of what is new in endoscopy, but we felt that none of these techniques had yet been sufficiently developed to merit further discussion in an Atlas.

Most endoscopists will not regard themselves simply as expert technicians but as members of a gastroenterological team carrying clinical responsibility during endoscopy and also subsequently when major decisions are made on all available evidence, including that from endoscopy. This principle is generally accepted the world over, and for this reason training programmes for gastroenterologists not only set targets for endoscopy but also insist on a working knowledge of gastrointestinal pathology.

To accommodate these developments the most important differences between the original Atlas and the present volume are the inclusion of a chapter on how the pathologist can help the endoscopist, and the presentation of histopathological appearances alongside endoscopic images. No attempt has been made to write a

comprehensive textbook of gastrointestinal pathology, but sufficient data are presented to underline the relevance to the endoscopist of some knowledge of pathology. This is reflected in the new title.

The views obtained when using fibre endoscopes and newer video endoscopes are generally similar, as the same regions and lesions are being surveyed. The older images, whether square or round, were often of excellent quality. In the event, most endoscopists now use electronic equipment and are more used to viewing a screen, and recording images on video tape or as video prints. There seemed little point in attempting to replace the better pictures of the original Atlas but we have tried to supplement these as appropriate with images obtained by the use of video endoscopes.

Although this Atlas includes some material from the original work, the text has been almost entirely rewritten and expanded, many images have been replaced and many new ones added, and each chapter has been restructured to suit the new contents and purposes of this venture. While this Atlas is a second edition, we present it to our readers as a new work. It certainly seems so to us, especially taking into account the amount of time and effort expended, we hope to good effect.

For whom is this Atlas intended? As we stated in the Preface to the original Atlas, we aim, firstly, at the less experienced endoscopist so that he or she may gain confidence by having available a range of appearances from which to learn and with which to compare findings. Secondly, more experienced endoscopists may wish to broaden their horizons and may be stimulated by seeing a wider spectrum of appearances than those with which they are familiar. We hope that this Atlas will find a place in the endoscopy room as a bench book, as we are told the previous edition did. Radiologists, pathologists and non-specialist physicians and surgeons may also be interested to examine what the endoscopist actually sees and does during diagnostic and therapeutic procedures. Furthermore, we hope that this publication will lead to a wider understanding of the place of endoscopy in gastroenterology, that it will help decide which patients are most appropriately referred for endoscopy, and not least that it will reveal some of the limitations of the technique. We believe that this new book should be of value to clinical students and their teachers during discussions on gastroenterological topics. With the increasing involvement of interventional radiologists in endoscopic procedures, we wish for them to be among our readers. Histopathologists are also very much a part of the team so it may be of help and interest to them to have a ready access to a collection of endoscopic appearances when handling the fruits of endoscopy. And, last but not least, there is the nurse endoscopist. The number of practitioners in this new specialty has risen rapidly and their breadth of experience has expanded. With increasing acceptance of the new role the number of such specialty nurses will continue to rise. To this new group of potential readers we also extend our welcome and hope that they will find the Atlas useful.

Our own experience, and with it our collection of endoscopic images, has grown over the years. Nevertheless, we could not have undertaken this task without the help of others, as acknowledged elsewhere. Producing an Atlas, like the successful practice of gastroenterology, relies heavily on the support of a good team. To all our collaborators and colleagues we express our thanks.

KFRS  
RC  
RHH  
BFW

# Acknowledgements

Our first acknowledgement goes to our collaborators, Dr Martin Lombard, Dr Anthony Morris and Dr Thomas Rösch for their chapter on endoscopic ultrasound, and Dr John Morris for his overview of enteroscopy.

Some illustrations have previously appeared in *A Colour Atlas of Gastrointestinal Endoscopy*, edited by three of us and published by Chapman & Hall Medical; in this Atlas we listed and thanked a number of endoscopists for their loan of images, some of which are now reused. We must reassure our readers that the majority of the figures that appear in this second edition originate in our own units. Nevertheless, we have been offered a wealth of additional material by old and new friends and colleagues.

We must extend special thanks to Dr Mark van Blankenstein and colleagues, Endoscopy Unit of Rotterdam University Hospital Dijkzigt, Prof. Neil Mortensen, Department of Colorectal Surgery, John Radcliffe Hospital, Oxford and Dr Christopher Williams, Wolfson Unit, Northwick Park and St. Mark's Hospital, London.

We also gratefully acknowledge help from Dr Monzur Ahmed, Prof. Duncan Colin-Jones, Prof. Massimo Crespi, the late Dr Jack Davies, Dr Richard Dickinson, Prof. Brian Gazzard, Mr Bruce George, Dr Peter Golding, Dr Godman Greywoode, Dr Rebecca Harrison, Dr. Stefan Hubscher, Prof. Gunar Jänerot, Dr Blair S. Lewis, Dr Duncan Loft, Dr Nick Mahy, Dr Tony Mee, Dr Peter Millard, Dr David Mutimer, Dr Simon Olliff, Dr Adam Padel, Dr Marios Panos, Dr Juan Piris, Dr Howard Rigby, Mr Alan Roe, Dr David Rowlands, Dr Scott Sanders, Dr Brian Saunders, Prof. Neil Shepherd, Prof. Janaka de Silva, Dr Subbaramiah Sridhar, Dr Tim Stephenson, Prof. Paul Swain, Dr Robin Teague, Dr Naomi Uemura, Dr Marjorie Walker and Dr Peter Willoughby.

A small number of images have been reproduced, with permission, from *Illustrated Case Reports in Gastroenterology*, the *Journal of Clinical Pathology* and the *American Journal of Surgical Pathology*.

It takes a long time to put together a useful collection of endoscopic images, and over this time we have had much help from medical, nursing, technical and secretarial staff of our Units. They deserve more than routine thanks. We must also thank KeyMed (Medical and Industrial Equipment) for the loan of some excellent equipment; Helene Beard for expert preparation of histological figures and Molly Harwood for help with the images drawn from the collection held in the Department of Colorectal Surgery, John Radcliffe Hospital, Oxford. The Department of Medical Illustration, Selly Oak Hospital, Birmingham, prepared many of the photographs of equipment. Special mention must be made of Robin Roberts-Gant, Medical Informatics Unit, Nuffield Department of Clinical Laboratory Sciences, University of Oxford, who so expertly handled a melange of old and new, round and square transparencies, prints, photographs and digital images of all sizes, and also helped in

the preparation of the greyscale images and many other illustrations. The line drawings in Chapter 6 were prepared by Jane Fallows.

The staff of Blackwell Publishing were throughout encouraging and supportive. In particular we must list Charlie Hamlyn, Marcela Holmes, Audrey Cadogan and Sally Lane and last but not least Andrew Robinson who was in overall charge, and ever helpful and patient.

When there are four editors, all of whom are also contributors, the manuscript inevitably has a troubled gestation, a difficult birth and a traumatic childhood. We could not have produced this Atlas without the patient, sustained and efficient input (in her spare time) from Ginny Schiller at the keyboard.

We cannot thank everyone who helped us enough. If the result is worthwhile it is in great measure due to their unstinting support.

KFRS  
RC  
RHH  
BFW

# Abbreviations

AIDS	acquired immunodeficiency syndrome
APC	argon plasma coagulation
AVM	arteriovenous malformation
CBD	common bile duct
CLO	columnar lined oesophagus
CMV	cytomegalovirus
CT	computerised tomography
DALM	dysplasia-associated lesion or mass
EATL	enteropathy-associated T-cell lymphoma
EGC	early gastric cancer
ERCP	endoscopic retrograde cholangiopancreatography
ESWL	extracorporeal shock wave lithotripsy
EUS	endoscopic ultrasonography
FAP	familial adenomatous polyposis
FB	foreign body
FNAB	fine needle aspiration biopsy (also FNA)
GAVE	gastric antral vascular ectasia
GIST	gastrointestinal stromal tumour
GORD	gastro-oesophageal reflux disease
HPF	high power field
MALT	mucosa-associated lymphoid tissue
MRC	magnetic resonance cholangiography
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
Nd-YAG	neodymium-yttrium aluminium garnet
NSAID	non-steroidal anti-inflammatory drug
OGD	oesophago-gastro-duodenoscopy
PAS	periodic acid Schiff
PCR	polymerase chain reaction
PEG	percutaneous endoscopic gastrostomy
PSC	primary sclerosing cholangitis
PTC	percutaneous transhepatic cholangiography
REAL	revised European and American lymphoma classification
TTS	through-the-scope
UC	ulcerative colitis
US	percutaneous ultrasonography

# Getting the Most out of your Pathologist

**Information 1****Suggestions for obtaining biopsy and cytological specimens 3****How do I send the biopsy specimens? 3****How quickly can I have an answer? 7****What does the laboratory do with the specimens? 7****Special techniques 8****Cytological specimens 13****Endoscopic resection specimens 14****Artefacts 15****Clinicopathological meetings 18**

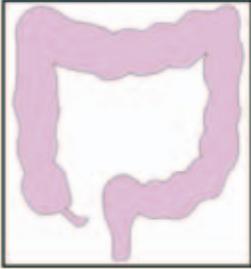
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This chapter discusses handling of biopsy, endoscopic resection and cytological specimens and how to present them to the pathologist to realise their optimum diagnostic potential. Mention is made of some techniques used by pathologists with which endoscopists should be familiar. Throughout this chapter emphasis is laid on ways clinicians and pathologists can work together. Views expressed in this chapter are based on practice in one of our centres. Readers in practice elsewhere will bear this in mind, for example regarding the section headed ‘How quickly can I have an answer?’

---

**Information****What do I tell the pathologist?**

The pathology request form is a request for a specialist opinion and as such must include adequate information about patient details including symptoms, results of other relevant investigations, endoscopic findings and, above all, the sites from which the samples have been taken. The request may be made on a simple card or a computerized document including part of the endoscopy report. An integrated request form for endoscopists (Fig. 1.1, adapted from D. Jenkins, 1988) has been developed by the British Society of Gastroenterology Guidelines Group for

COLORECTAL BIOPSY		CLINICAL DATA SET FOR SUSPECTED CHRONIC IDIOPATHIC IBD	
Patient Name:		Hospital:	
Date of birth:	Sex: F <input type="checkbox"/> M <input type="checkbox"/>		
Date of examination:			
Main symptoms:		<input type="text"/>	
Diarrhoea:	<input type="text"/>		watery <input type="checkbox"/> bloody <input type="checkbox"/>
Total duration of disease:	<input type="text"/>		
Duration of present episode:	<input type="text"/>		
Present clinical state:	<input type="text"/>		active <input type="checkbox"/> inactive <input type="checkbox"/>
Sigmoidoscopic appearance:	mucosal features: <input type="text"/>		
	extent of disease:		
	rectum	<input type="checkbox"/>	
	rectum & sigmoid	<input type="checkbox"/>	
	left side	<input type="checkbox"/>	
	substantial	<input type="checkbox"/>	
	extensive	<input type="checkbox"/>	
	pancolitis	<input type="checkbox"/>	
pattern	continuous	<input type="checkbox"/>	
	segmental	<input type="checkbox"/>	
Site of biopsy/biopsies:	<input type="text"/>		
Clinical opinion:	<input type="text"/>		
<b>ADDITIONAL INFORMATION (IF APPROPRIATE)</b>			
Include previous gastrointestinal surgery, therapy, results of stool culture, other diseases present	<input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/> <input type="text"/>		

1.1

Inflammatory Bowel Disease; this form serves as a good example of the use of tick boxes and may be used either as a sheet of paper or as part of a computerized request.

The exact site of origin of samples is often crucial, as for example in the stomach when gastritis of the antrum and pangastritis have quite different clinical connotations and risk potential; again, metaplasia in body-type mucosa as a consequence of inflammation and loss of specialized cells can cause it to look identical to antral mucosa. Another example is in the diagnosis of Barrett's oesophagus where it is essential to know whether the biopsy is truly from the oesophagus. Although the presence of underlying oesophageal mucous glands or ducts (Fig. 2.141) may help in identification they are not always present. If it is known with certainty that the biopsy came from the oesophagus and not from an hiatal hernia or the stomach, disorderly glandular mucosa with or without intestinal metaplasia is enough to corroborate the endoscopic diagnosis.

Relevant previous surgical operations must be mentioned on the request form as it is an intellectual challenge to the pathologist if a form is labelled

'oesophageal biopsy ?inflamed' when the patient has had an oesophagectomy with colonic interposition. Similarly, small bowel metaplasia might be reported in a defunctioned rectum when there had been a previous colectomy with ileo-colic anastomosis. Without knowledge of the previous anastomosis the biopsy taken from a remnant of normal small bowel would be misinterpreted.

It is helpful to include a copy of the endoscopy report and perhaps an endoscopic picture: pathologists find macroscopic appearances invaluable. This could be supplemented by occasionally inviting the pathologist to see lesions *in situ* at endoscopy. Such an approach improves working relations and makes the pathologist feel part of the team, with consequent increased enthusiasm for endoscopic specimens.

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## Suggestions for obtaining biopsy and cytological specimens

Certain general recommendations can be made. For example, for lesions where malignancy is suspected or is a possibility, whether such a lesion is raised, flat, depressed or ulcerated, up to 12 biopsy specimens should be collected. The target sites should include as many aspects of the lesion as possible, for example the rim as well as the ulcerated centre. When a solid lesion appears to be submucosal, superficial biopsies are often unhelpful and other methods of obtaining material, e.g. 'large particle'/snare biopsy, fine needle aspiration or the use of hot biopsy forceps should be considered.

When the interest is centred on an observed or possible mucosal abnormality, the biopsy forceps must of course be directed as appropriate but in any case multiple specimens should be sought as mucosal abnormalities are often patchy and may not be visible endoscopically. It is difficult to recommend a useful minimum number of specimens; suffice it to say that your interested, co-operative and involved pathologist will prefer too many to too few.

The requirements for screening and surveillance will differ from those for diagnosis. For example, a diagnosis of Barrett's oesophagus will be confirmed when one biopsy from a lower oesophagus clearly lined with columnar mucosa is positive in this respect. Conversely, when a patient with known Barrett's oesophagus attends for screening for dysplasia, it is recommended that quadrant biopsies should be taken at 2 cm intervals along the length of the Barrett's oesophagus. Again, in the follow-up of a patient with inflammatory bowel disease known to have had pancolitis, it is advisable to take biopsies at 10 cm intervals from caecum to rectum, although this is not always practicable.

When specimens for cytological study are collected, a sheathed brush must be used. Contamination of the biopsy channel of the endoscope with foreign material can lead to irreparable complications in patient management and to serious medicolegal problems. Other aspects of cytology are discussed later in this chapter.

The above is no more than a set of general recommendations. More specific and more detailed recommendations appear as appropriate in succeeding chapters.

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## How do I send the biopsy specimens?

### Histological processing cassette 6

### Frozen section diagnosis 6

Biopsies from different anatomical sites or lesions must be submitted in separate, labelled containers. The impossibility of distinguishing atrophic gastric body-type mucosa from antral mucosa has already been mentioned. The distribution of a colitis is a great diagnostic aid which is lost if all samples are floating in the same pot.

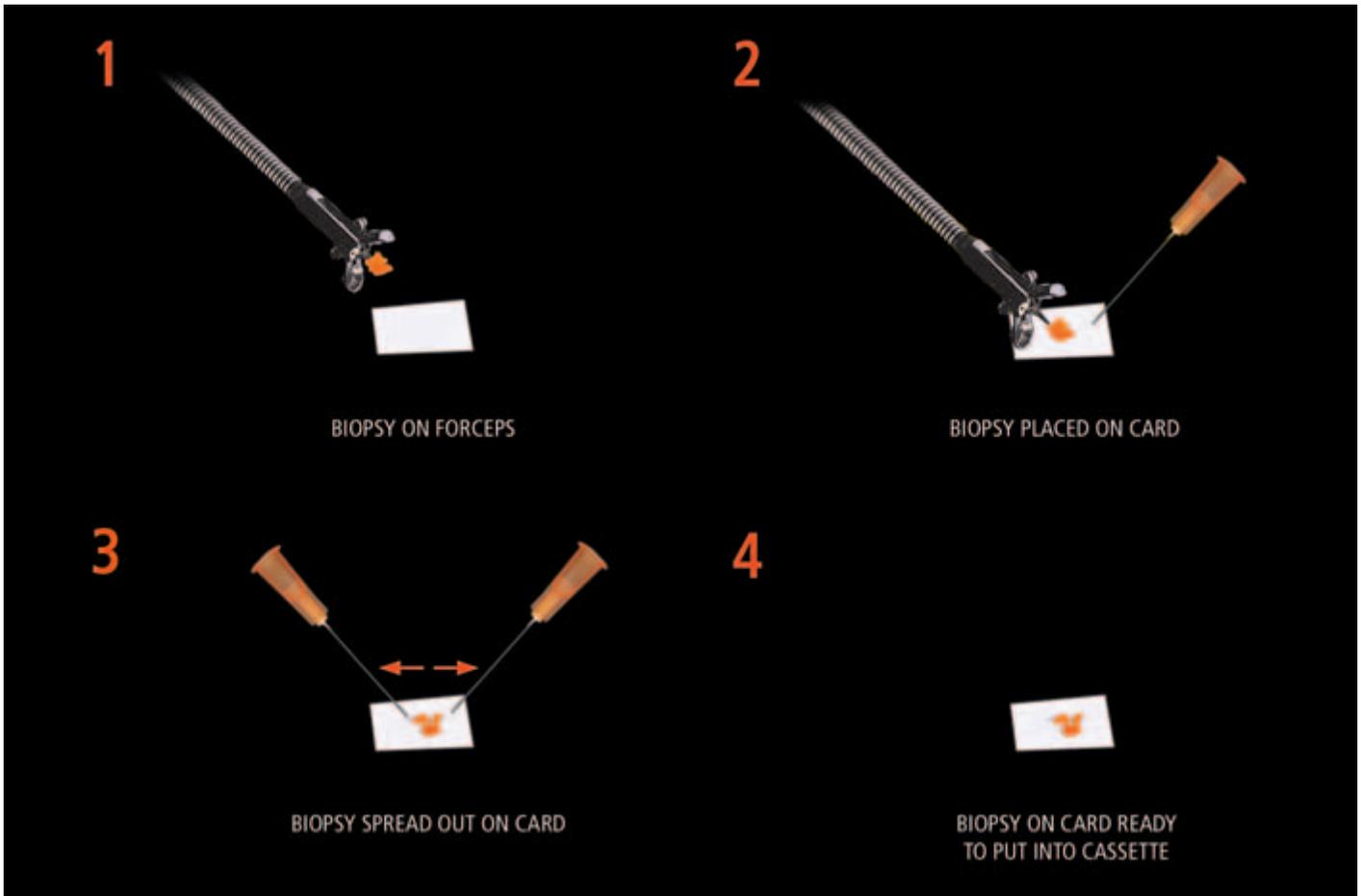
Similarly, a diagnosis of dysplasia or invasive malignancy is only of practical use if the site is known: a dermatologist would not place odd looking naevi from different sites in the same pot, in case one of the naevi should be a malignant melanoma needing further excision. Each pot should be labelled individually and placed in a plastic bag with the request form in a separate plastic pocket within the bag (Fig. 1.2) to avoid smudging and contamination of the form with potentially infected body fluids.



1.2

Most material for pathological study can be put into 10% buffered formalin, the amount of fixative required being a minimum of five times the volume of the tissue to be fixed. Tissue fixation involves a complex set of chemical reactions which are slowed by cooling. Thus there is no logic in the common practice of placing specimens in formalin in the refrigerator overnight. Glutaraldehyde is a good fixative for electron microscopic studies. Bouin's fixative gives better preservation of neuroendocrine cells but is not used in routine endoscopic diagnostic biopsy practice.

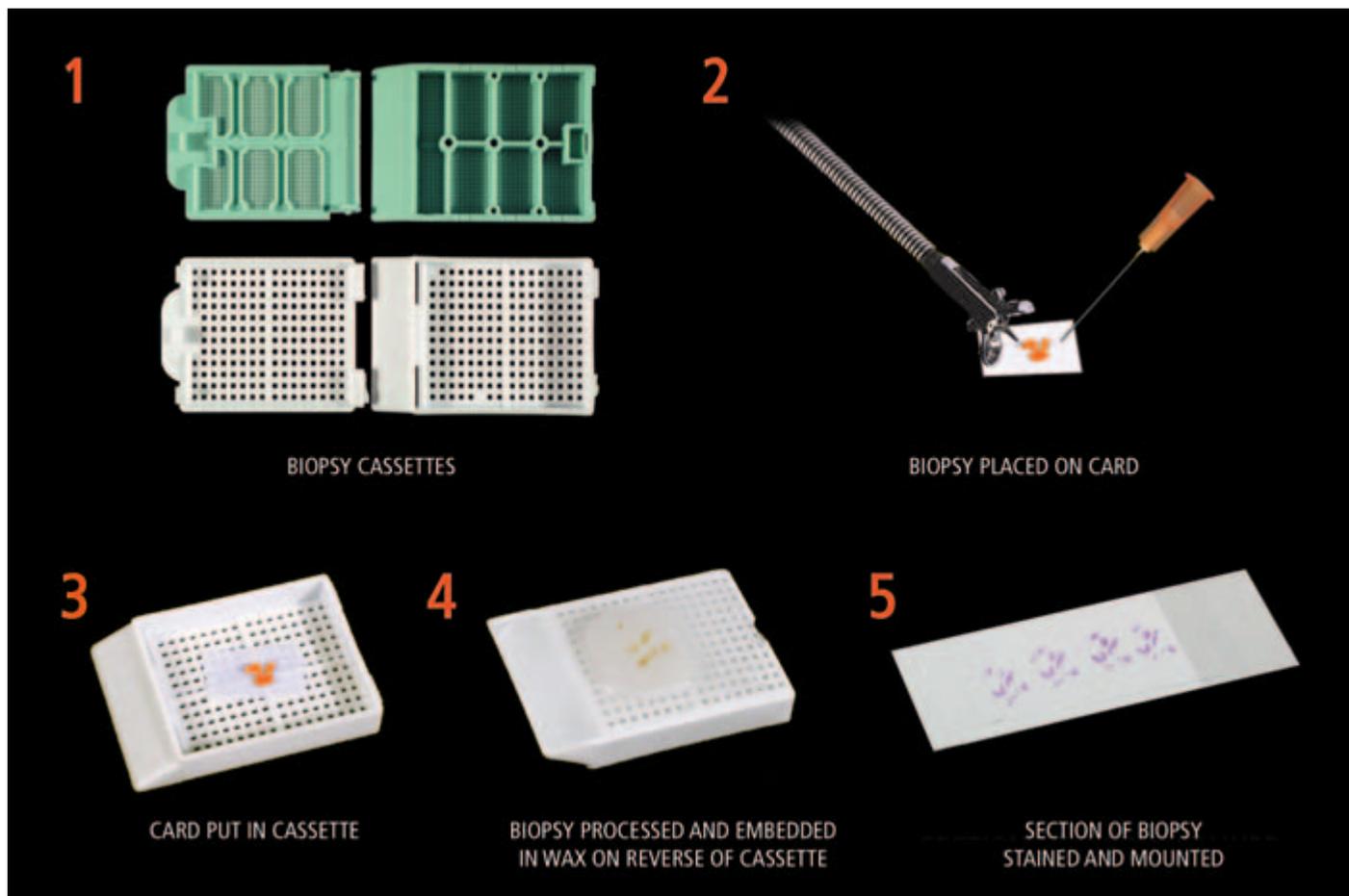
Biopsies should be extracted gently from the forceps using a needle. Although it takes more time, this is best done in a Petri dish of physiological saline to prevent drying artefacts. In a non-orientated mucosal biopsy contraction of the muscularis mucosae results in curling of the tissue with the mucosa on the outside of the ball. Teasing of very small biopsies in an attempt to orientate them may be difficult and potentially disrupting to the tissue, but orientation of larger, more easily visible pieces may be helped by placing the material on a strip of thin card, muscularis mucosae side down (Fig. 1.3). The biopsy will adhere to the card and will not roll into a ball as the muscularis mucosae contracts with fixation. Multiple biopsies may be put on one strip of filter paper so long as the strip is carefully labelled. This permits some degree of orientation at the postfixation stage. However some laboratories do not use this method routinely and rely on cutting an adequate number of sections at different levels through the block, which usually allows a well orientated view of the mucosal architecture. Orientation is especially important for accurate assessment of villous architecture, inflammation and dysplasia. In the absence of correct orientation, proper assessment of villous architecture in small bowel biopsies is impossible. Similar problems occur with recognition of inflammation in the large bowel and dysplasia anywhere in the gastrointestinal tract. Lymphocytes, plasma cells and eosinophils are normally present only in the superficial part of the lamina propria of the large bowel. An increase in chronic inflammatory cells is recognized by



1.3

noting the presence of plasma cells at the level of the muscularis mucosae and loss of the normal gradient of cell density between the upper and lower parts of the lamina propria. This is impossible if the view is of a transverse section of only the superficial part of the mucosa at more than one site. Recognition of dysplasia depends on many subtle and less subtle changes in the crypt epithelium. One reliable feature is failure of nuclear maturation and the presence of abnormal nuclear detail throughout the full length of the crypt. If the full length of the crypt cannot be viewed the presence and grade of dysplasia cannot be assessed.

Exceptionally, biopsies need to be presented fresh. Under such circumstances it is best to make arrangements for the pathologist to attend in person. Freshness is essential, for example, in the investigation of motility disorders, such as Hirschsprung's disease in children or slow transit constipation in adults so that specimens are in the best state prior to freezing. Fresh specimens are also useful to assess excision in larger polyps, in transanal endoscopic microsurgical excision and when schistosomiasis is suspected. Crushing a biopsy between two glass microscopy slides and viewing the unstained biopsy with ordinary light microscopy and between paired polarizing lenses may reveal the refractile wall of the schistosomes. When successful, this gives a very rapid answer; when unsuccessful, a potentially useful rectal biopsy has been destroyed. A duplicate specimen should always therefore be submitted in routine fixative.



1.4

### Histological processing cassette

Automated histological processing machines are now in common use. Tissue is placed into a small perforated plastic cassette. Figure 1.4 shows two types, one with six small compartments and one with a single large compartment. When processed, the tissue is embedded in paraffin wax and the wax block is adhered to the back of the tissue cassette. The cassette is then used to mount the block on a microtome for sectioning and subsequently for storage. The cassette is labelled with a unique identifying laboratory number. Laboratory handling is facilitated by the use of such cassettes in the endoscopy room. When this step is not used in the endoscopy room biopsies have to be extracted from a pot of formalin and placed in a cassette in the laboratory. Repeated transfer of tissue from one container to another may lead to fragments of tissue from one patient's specimen contaminating another. One way to reduce the number of specimen handling and transfer steps is for the endoscopist to place the biopsy in a labelled, lidded cassette, as described above, in the endoscopy room. Endoscopists may care to discuss this approach with their pathologist.

### Frozen section diagnosis

Frozen sections are rarely used in gastrointestinal endoscopic biopsy practice, with the exception of the very few specialized investigations, usually research orientated immunohistochemistry, with reagents which are not yet validated or are known not to work on paraffin processed material. As mentioned earlier, it may be useful in motility disorders.

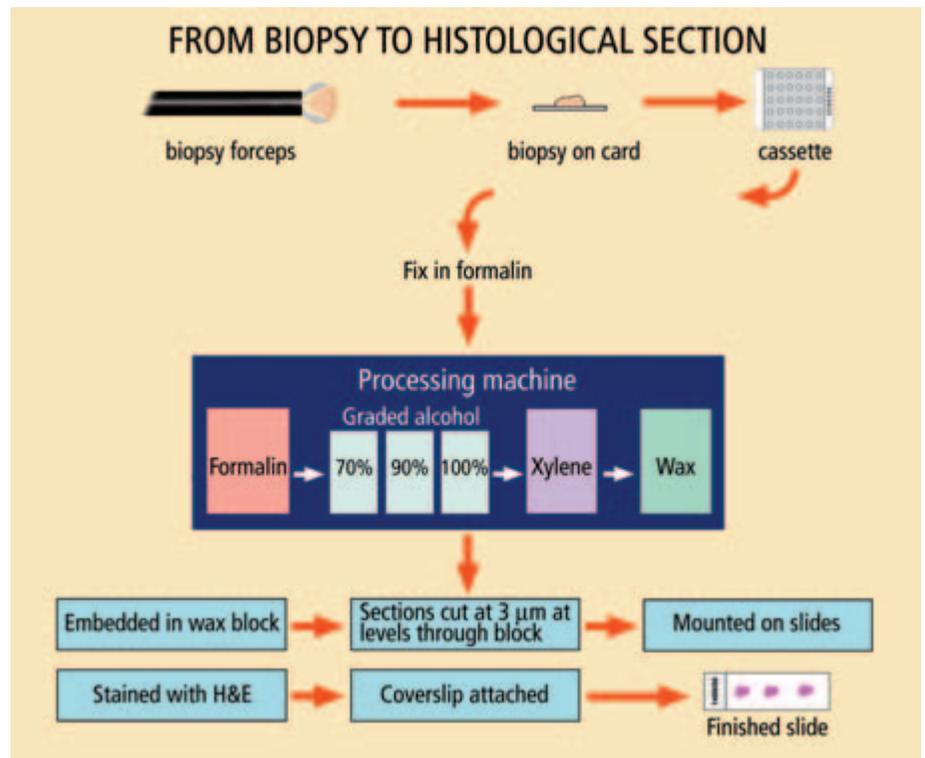
## How quickly can I have an answer?

This depends primarily on the size of the specimen sent to the laboratory, and the time of day it is sent. With very small biopsies, it is possible to provide at least a provisional report within the working day or overnight, depending on the time of receipt. If special staining techniques are required the process will be prolonged as described below. Cytological smears can often be reported on within 10–20 min of receipt by the pathology laboratory.

## What does the laboratory do with the specimens?

A flow chart illustrating the routine handling of a biopsy specimen appears below (Fig. 1.5).

In the first place this involves adequate fixation of the tissue, and processing of the specimens through to paraffin wax. Sections are then cut and stained appropriately for microscopic examination. Most gastrointestinal diagnoses are made using a routine haematoxylin and eosin stain, but specialized staining will cause delays. Simple tinctorial staining, e.g. for mucin, can be done on the same day and it is recommended that this should be performed routinely on all gastric, Barrett's and duodenal biopsies as isolated signet ring adenocarcinoma cells may be so easily missed. More complex staining, such as for immunohistochemistry, may take a further day. Initial immunohistochemical staining may indicate that further immunohistochemical stains are necessary, and this will take usually another full day.



1.5

## Special techniques

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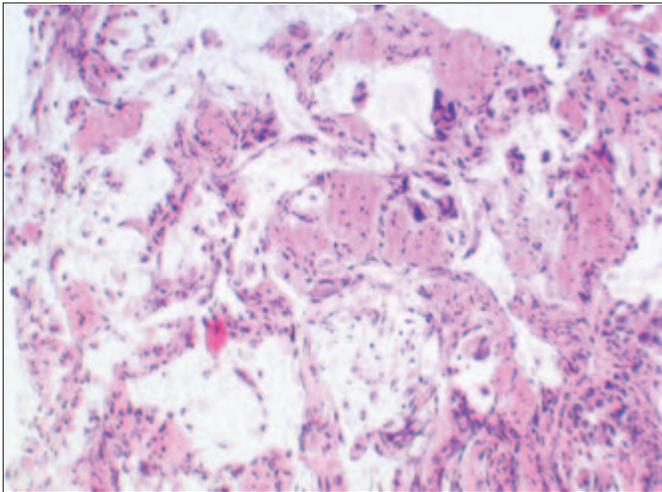
**In situ hybridization 13**

**Polymerase chain reaction (PCR) 13**

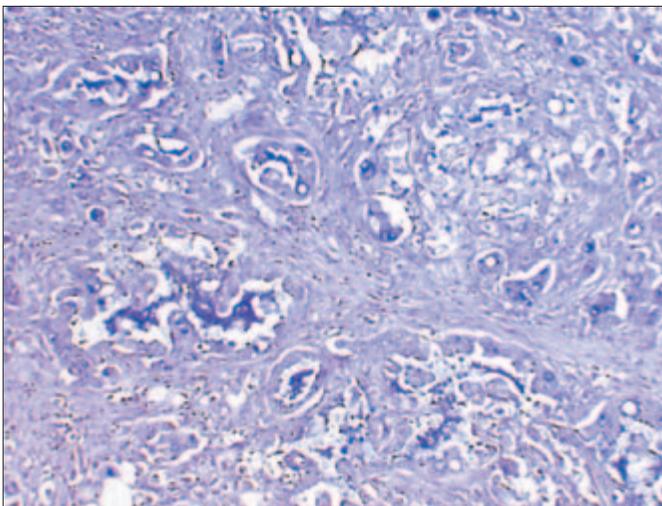
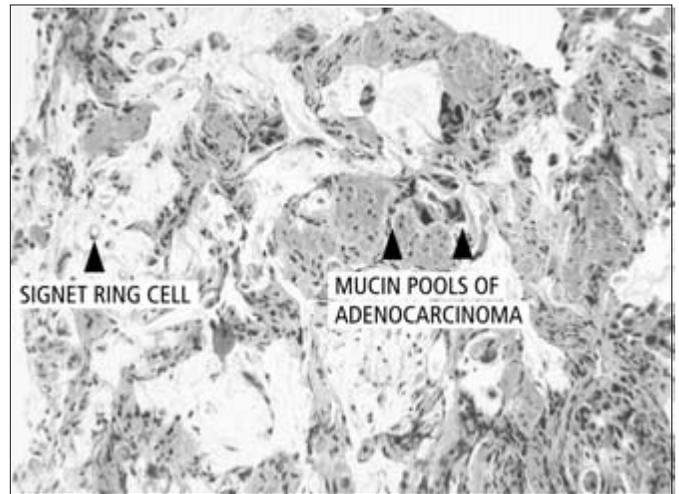
**Tissue typing 13**

### Additional commonly used tinctorial stains

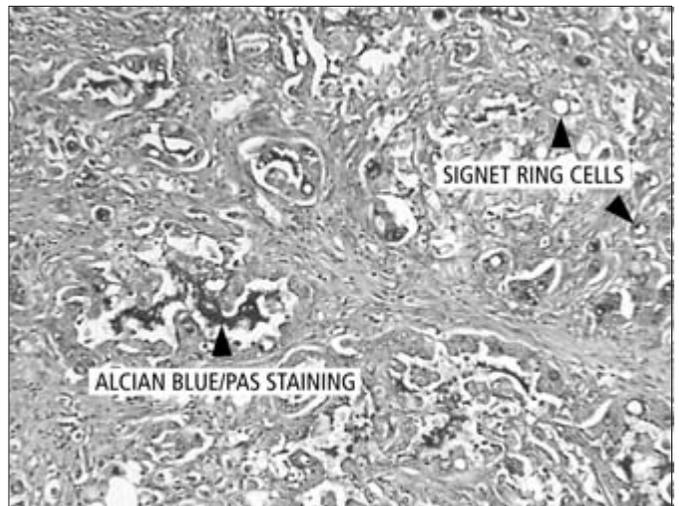
Alcian blue combined with periodic acid–Schiff (PAS) staining is essential in the evaluation of biopsies from the oesophagus, stomach and duodenum for identification of acid mucin/neutral mucin. Without this technique intestinal metaplasia and individual signet ring cancer cells may very easily be overlooked. In Fig. 1.6 the section has been stained with haematoxylin and eosin. When stained with Alcian blue and PAS the signet ring cells are easily seen (Fig. 1.7).



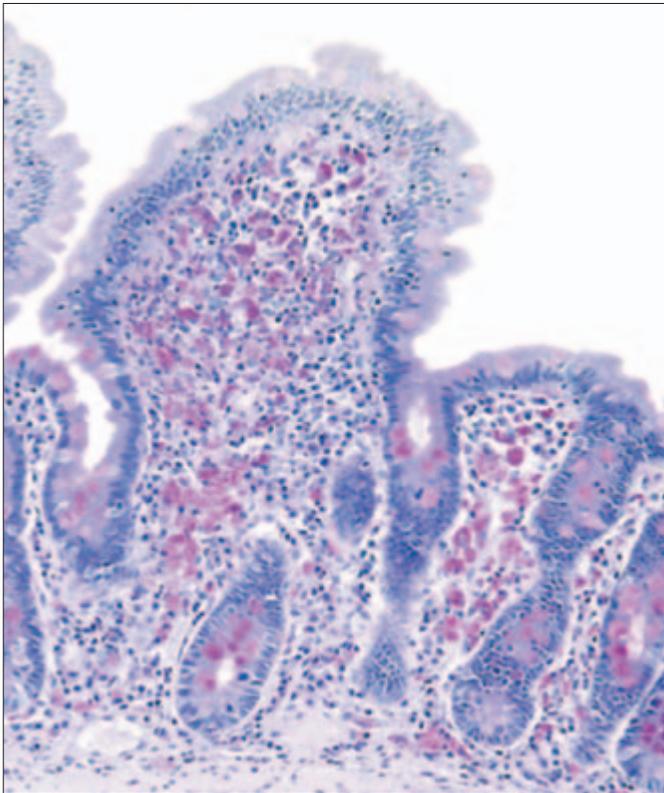
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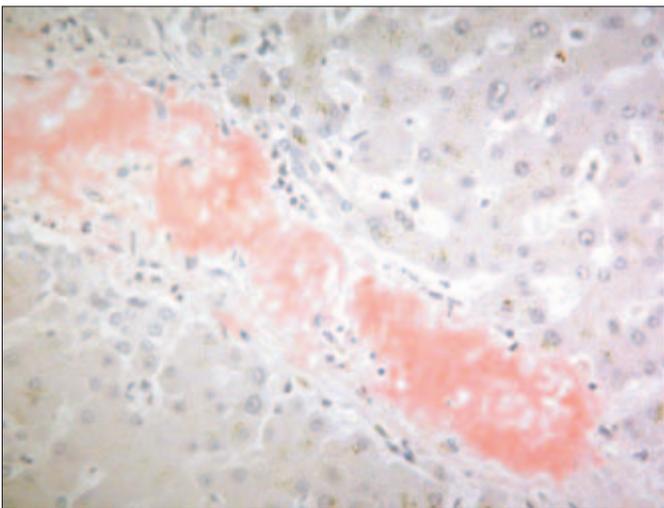
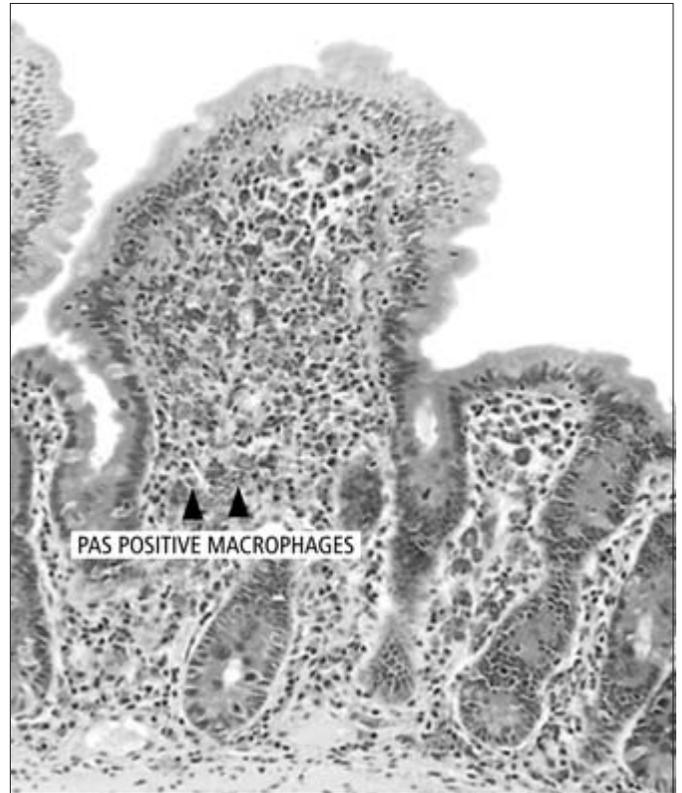
1.7



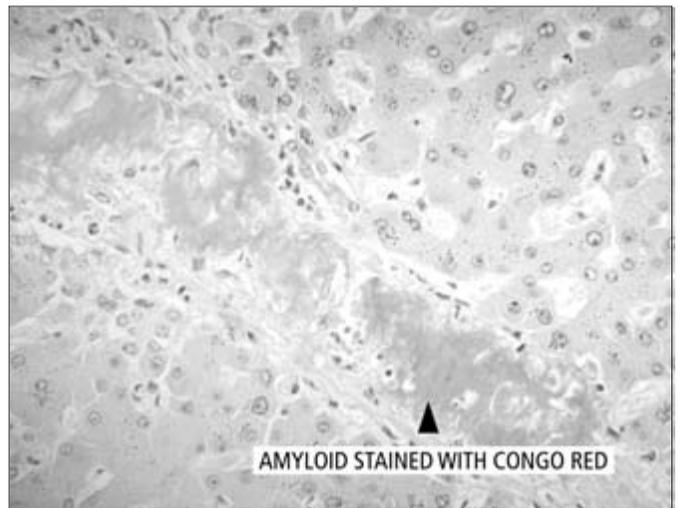
The PAS part of this combination also stains fungi, macrophages in Whipple's disease (Fig. 1.8) and amoebae in large bowel biopsies. Trichrome stains are useful for collagen if collagenous colitis is suspected. Sections stained with Sirius red or in this case with Congo red, when viewed under crossed polarizing lenses identify amyloid by the presence of apple green birefringence (Fig. 1.9).



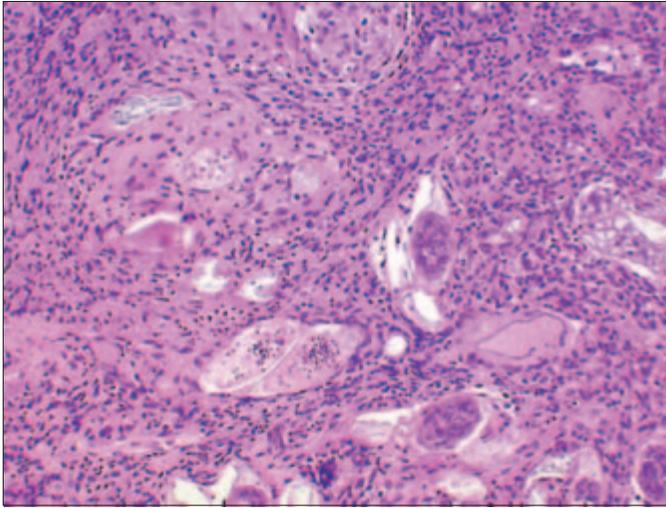
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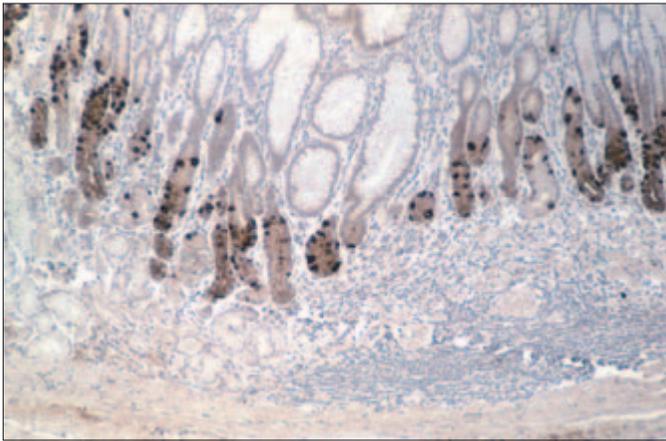
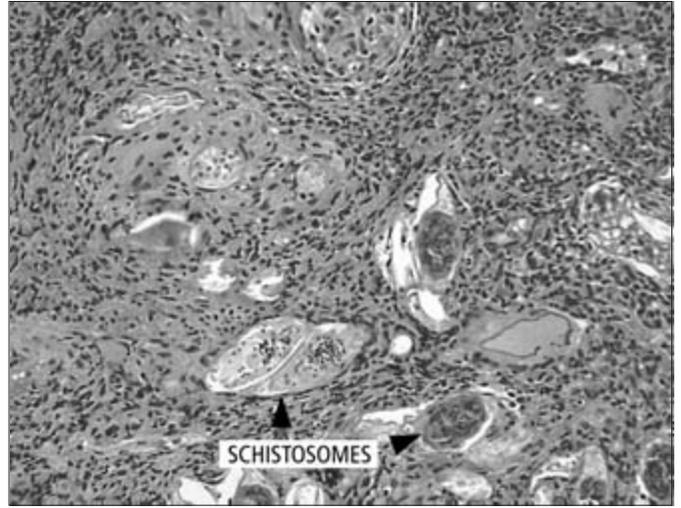
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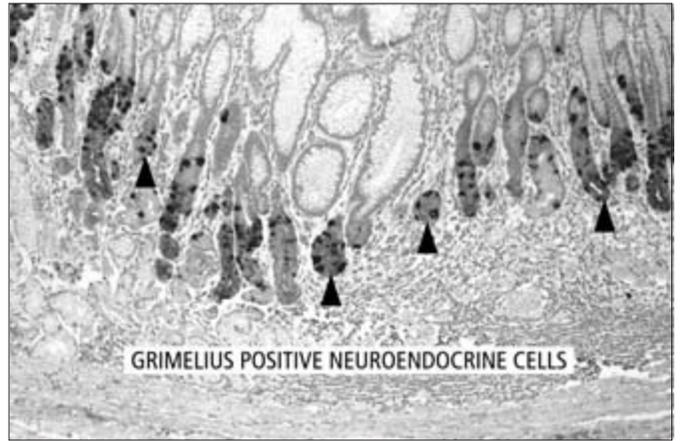
Ziehl–Neelsen stain, well known as a means of identifying mycobacteria, also stains schistosomes (Fig. 1.10). Grimelius is a silver stain for neuroendocrine cells (Fig. 1.11). There have been few new tinctorial stains but the recently introduced Genta stain shows mucin and *Helicobacter* as well as demonstrating tissue morphology.



1.10

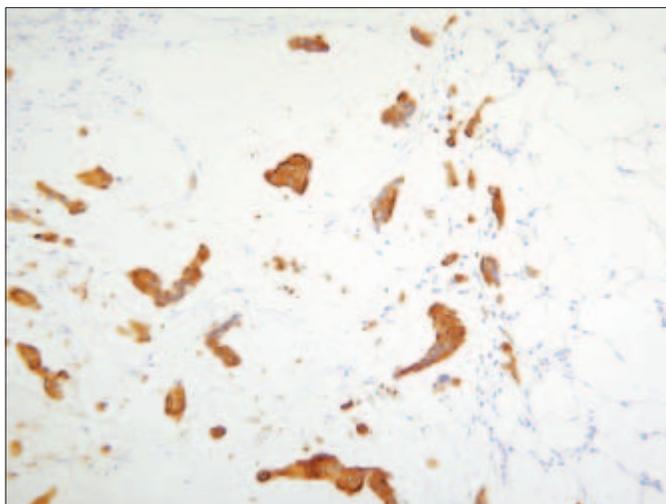


1.11

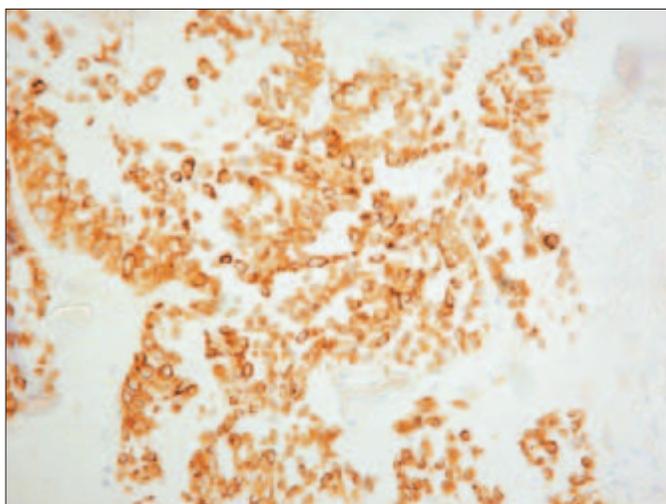
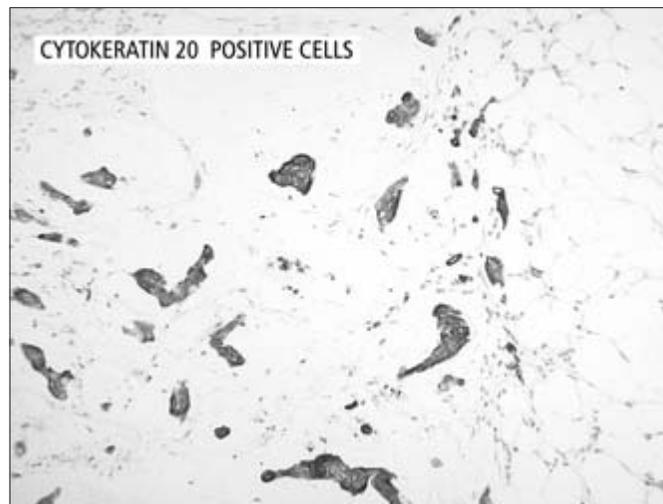


## Immunohistochemistry

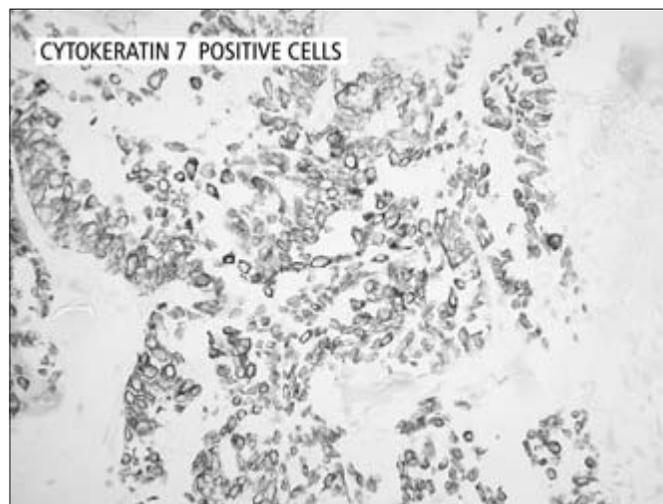
Immunohistochemical stains for cytokeratins, epithelial membrane antigen and carcinoembryonic antigen are used to detect abnormally sited epithelial cells when diagnosing invasive malignancy. Some caution is needed in interpretation since some cytokeratin stains may attach to new fibroblasts in ulcer bases as well as staining epithelial cells. Many cytokeratin stains are available and may be used to overcome this pitfall. Cytokeratin 20 is a sensitive marker for gastrointestinal adenocarcinoma cells (Fig. 1.12), whereas cytokeratin 7 is a sensitive marker for gynaecological adenocarcinomas; this pair of cytokeratin stains is therefore very useful for the distinction of a primary gastrointestinal carcinoma from a metastatic ovarian or endometrial carcinoma (Fig. 1.13). Lymphomas are best evaluated immunohistochemically. This will enable identification of T or B cells and will by staining for kappa and lambda light chains assess clonality in B cell infiltrates where the diagnosis of lymphoma is subtle. Cytokeratin staining will highlight the crypt epithelium and will enable the destructive lymphoepithelial lesions of a MALT lymphoma to be identified more easily. These are areas of crypt epithelium which are infiltrated by B lymphocytes causing epithelial destruction at that site. Chromogranin A will identify more than 90% of neuroendocrine cells. This is especially useful for carcinoid tumours. Immunohistochemical staining gives confusing results in

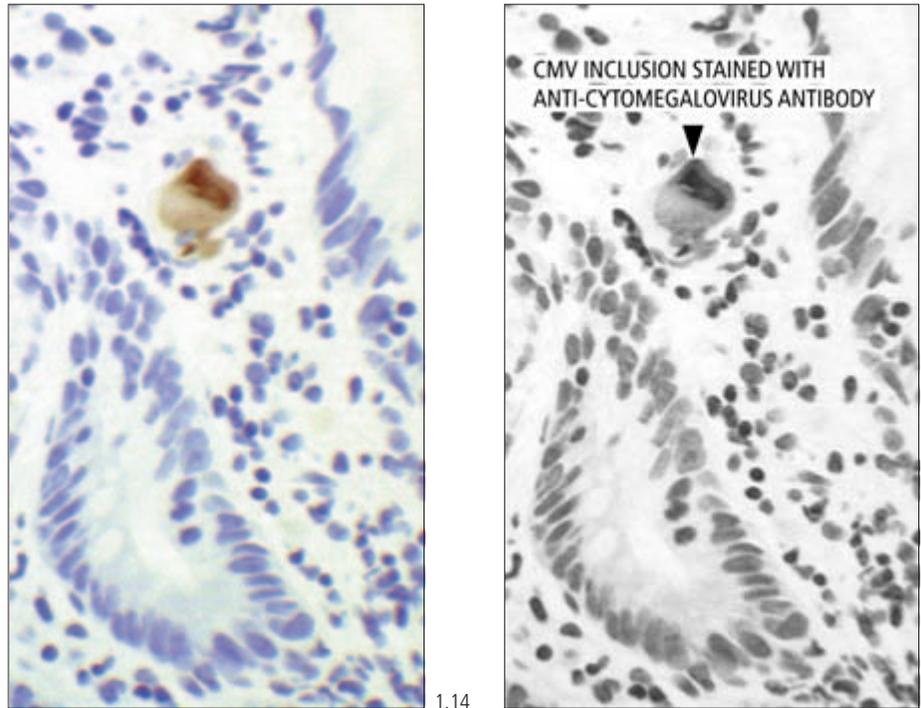


1.12



1.13





1.14

gastrointestinal stromal tumours (GISTs) but, whether they appear predominantly neural, muscular, mixed or neither on immunohistochemistry, most are positive with CD34, a vascular endothelial cell marker. Nearly all GISTs including those negative with CD34 will stain with antibody to Ckit proto-oncogene, which is thought to indicate a possible origin of these tumours from the interstitial cells of Cajal (whose normal function is as ‘pacemaker cells’ in the intestine). Different patterns of immunohistochemical staining in GISTs have sometimes been correlated with prognosis, but it is probably better to count mitotic figures as a guide to prognosis (Fig. 2.415 and Table 2.10).

Immunohistochemical stains for organisms such as cytomegalovirus (Fig. 1.14) are an essential part of the work-up of a biopsy from an immunocompromised individual. *Herpes simplex* antibody staining is useful in suspected herpes oesophagitis.

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### Electron microscopy

This is rarely used in gastroenterological practice but may help in identification of *Microsporidia* spp. in immunocompromised patients. It may occasionally be useful to subclassify rare tumours. Figures 5.50 and 5.51 demonstrate microerosions in NSAID-related enteropathy.

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### Flow cytometry

Flow cytometry has found favour particularly in Sweden for the early diagnosis of dysplasia in ulcerative colitis by the detection of aneuploidy. It has not as yet become routine in most laboratories.

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## In situ hybridization

*In situ* hybridization is used for demonstration of abnormal DNA, RNA and other abnormal protein products, and in the diagnosis of some viral infections.

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## Polymerase chain reaction (PCR)

PCR demonstrates small amounts of protein, as for example in the tissue detection of *Yersinia* or *Mycobacterium paratuberculosis*.

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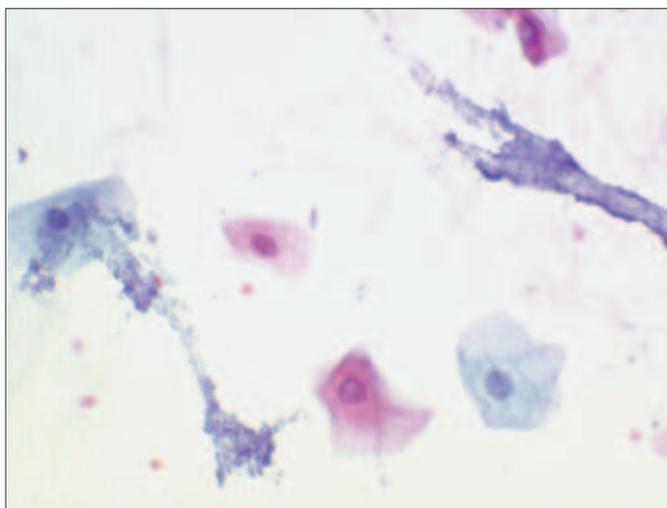
## Tissue typing

HLA tissue typing is useful in evaluation of 'carry over', when tissues may inadvertently originate from two patients. Anxiety, mismanagement or litigation may result if, for example, carcinoma or dysplasia is seen in a single fragment of blocked tissue when such a diagnosis was not expected. The problem may be resolved by referral of the block or sections to a laboratory specializing in tissue typing of small fragments.

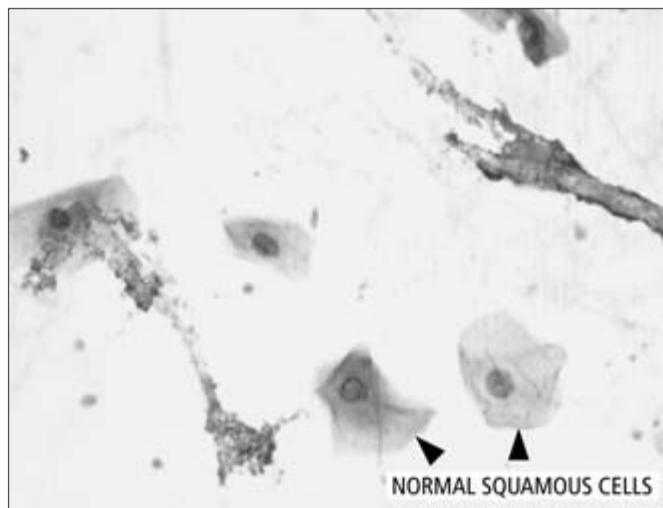
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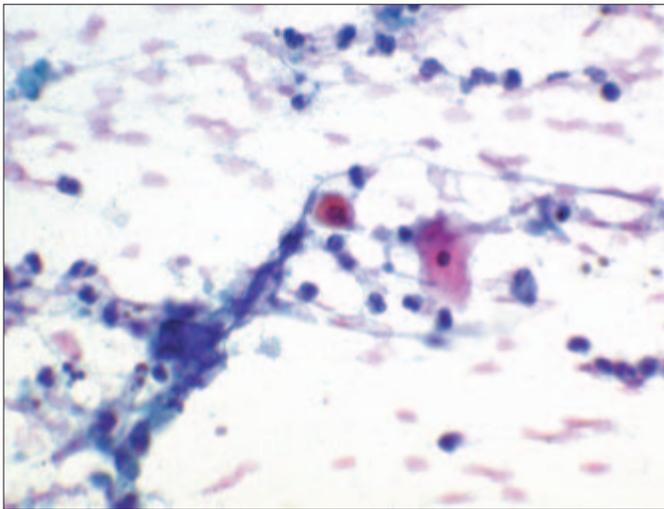
## Cytological specimens

Brush specimens taken for cytological examination are mainly employed in upper gastrointestinal tract diagnosis. The addition of cytology to biopsy may improve the positive diagnostic yield, especially for carcinoma of the oesophagus when compared with biopsy alone. Figure 1.15 shows the cytology from normal oesophagus and Fig. 1.16 from squamous cell carcinoma. Bile duct cytology is useful for lesions out of reach of biopsy forceps. Figure 4.43 illustrates brushings from a normal bile duct, in contrast to those taken from a bile duct carcinoma (Fig. 4.44). Anoscopy and cytology with a spatula and brush have become standard in some centres with a special interest in anal intraepithelial neoplasia. Brush cytology specimens generally have a higher yield than washings. It is essential that the pathologist should instruct the endoscopist and assistants in the technique of slide preparation. In particular, different staining techniques will require the appropriate method of slide preparation. Giemsa staining needs air dried slides, whereas most other stains need immediate smear fixation.

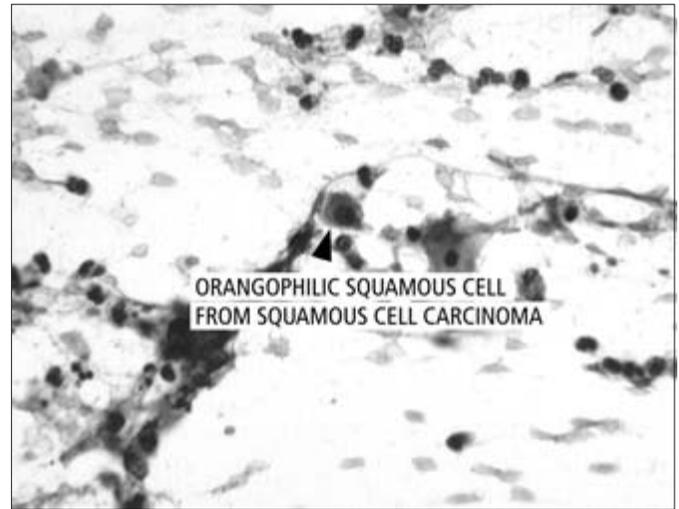


1.15





1.16



Fine needle aspiration cytology is not commonly used by endoscopists but may be of value in sampling lesions beneath the mucosa both for localized tumours and for attempting to improve the chances of tissue diagnosis in suspected linitis plastica.

## Endoscopic resection specimens

### Polypectomy 14

### Transanal endoscopic microsurgery 14

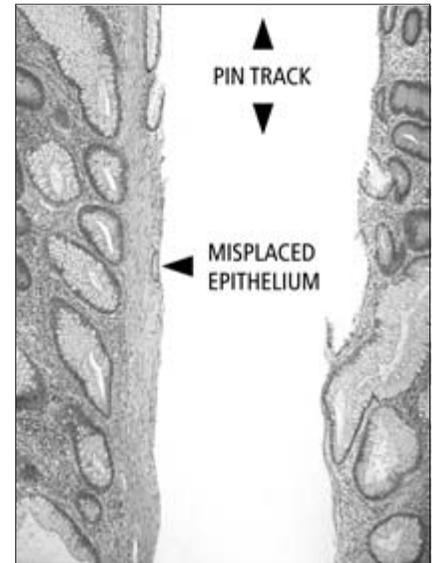
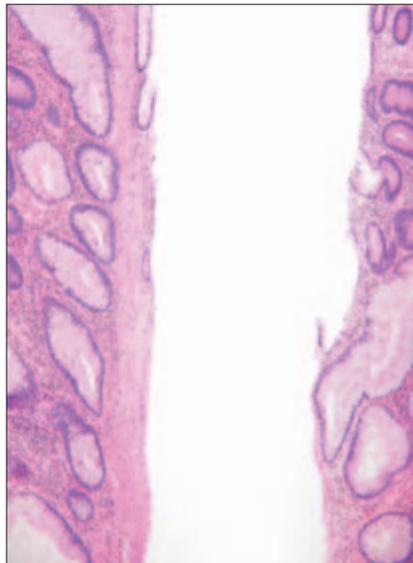
#### Polypectomy

The problems here are of orientation, diathermy artefact in the assessment of polyp type, stalk invasion and completeness of excision. A pedunculated polyp will shrink when fixed in formalin. Normal mucosa in the stalk shrinks more than the adenomatous mucosa which results in a stalk that was easy to visualize in the fresh state, retracting and disappearing into the polyp such that it is difficult or impossible to identify on the following day. Some endoscopists mark the site of excision on the excised polyp with ink or with a pin. If a pin is used it should pass from the stalk to the polyp, because insertion from the polyp towards the stalk may cause problems of misinterpretation of pseudoinvasion due to misplacement of epithelium into the stalk by passage of the pin, as shown in Fig. 1.17.

#### Transanal endoscopic microsurgery

Specimens from this technique need especially careful handling to provide the information required for subsequent patient management. The operation produces a square of full thickness rectal wall including tumour. Such specimens should be received fresh and pinned on to cork, to identify the narrow but crucial margin of normal mucosa which represents the mucosal resection margin.

Transanal endoscopic microsurgery requires the use of specialized equipment and cannot be performed via standard rigid or flexible endoscopes.

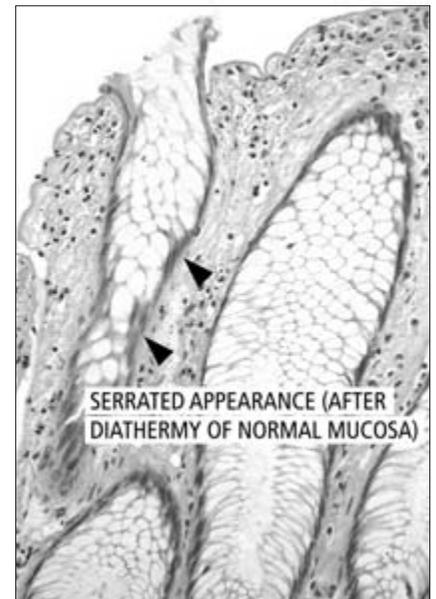


1.17

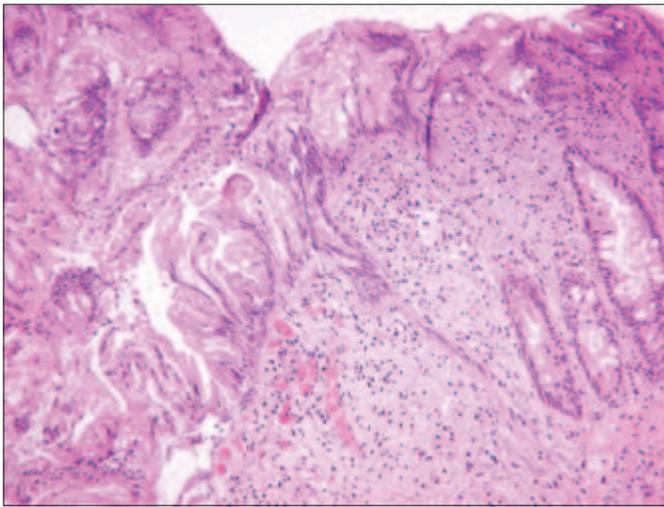
## Artefacts

It has been said that the histopathologist is merely a viewer of artefacts, i.e. that pathological specimens and stained histological sections are in themselves almost artefacts. To these must be added, for example, the damage to tissue during collection, handling and processing. These are some of the challenges to which the histopathologist tries to rise.

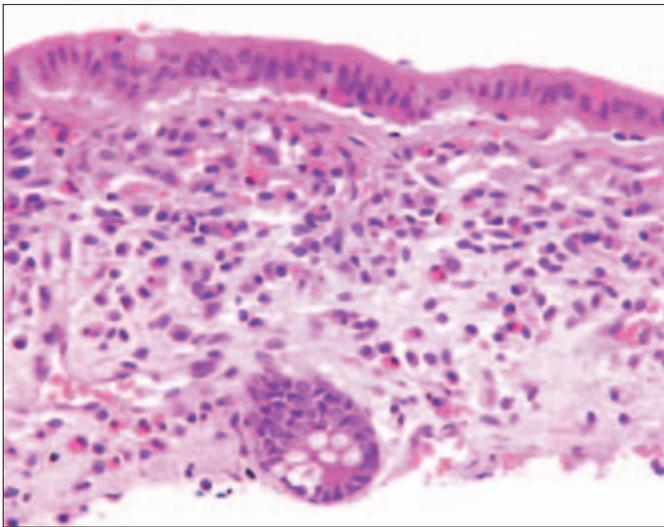
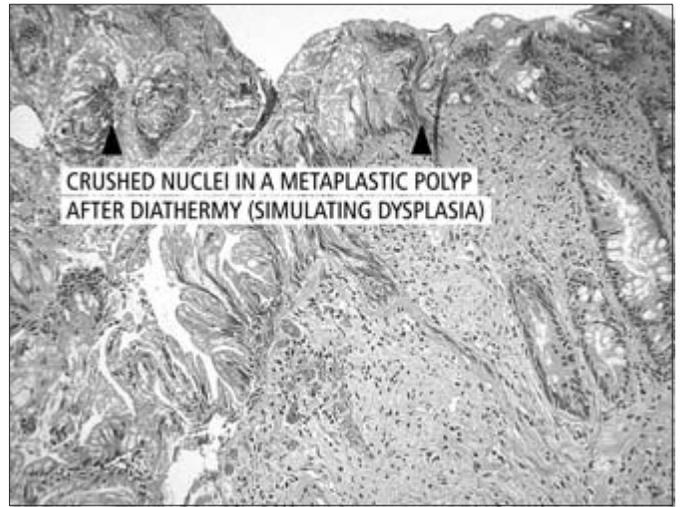
One of the commonest problems is the heat induced artefact seen after diathermy or the use of hot biopsy forceps. This makes normal mucosa look like a metaplastic polyp (Fig. 1.18).



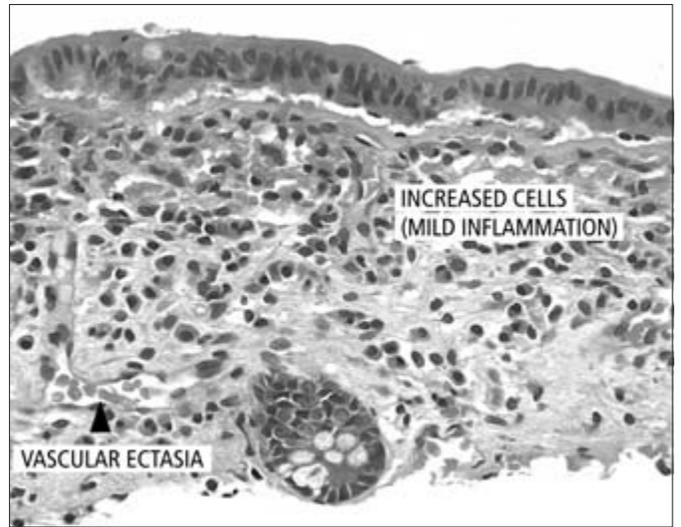
1.18



1.19



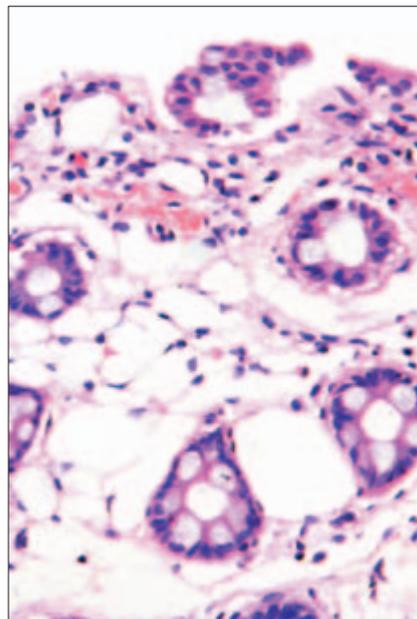
1.20



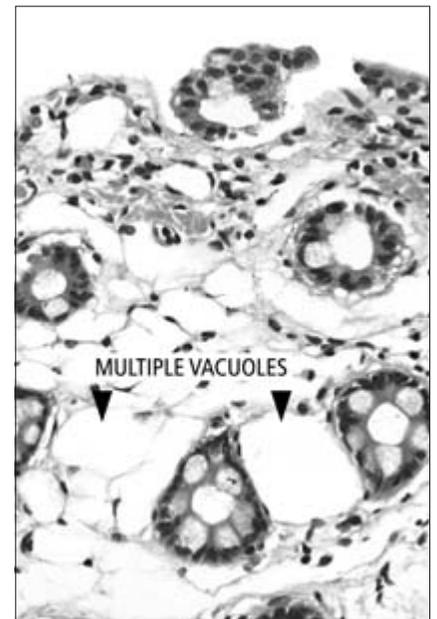
Metaplastic polyps may look like adenomas by crowding the nuclei together and simulating dysplasia (Fig. 1.19).

Following the use of heat, there may also be difficulty in the assessment of the completeness of excision of an adenoma or of a carcinoma within an adenoma.

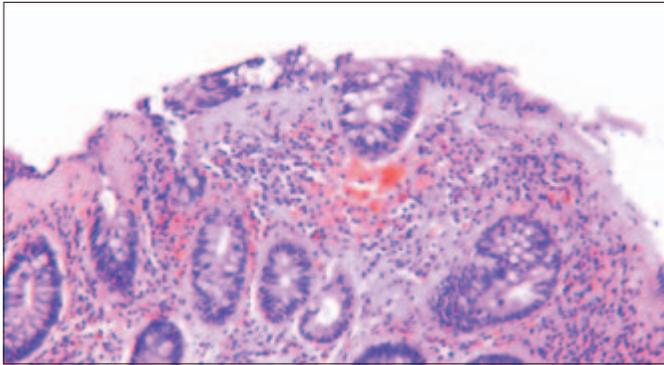
Preparation of the bowel for endoscopy using hyperosmolar solutions results in oedema and mucin depletion, and some authors report occasional appearances of inflammation, although this is not our experience. Mild inflammation may occasionally be seen following irritant enemas (Figs 1.20 and 3.336). White mucosal patches seen during the withdrawal of the colonoscope (Fig. 3.337) may be the result of hydrogen peroxide, occasionally used in endoscope cleaning, which causes a vacuolated appearance in the lamina propria (Fig. 1.21).



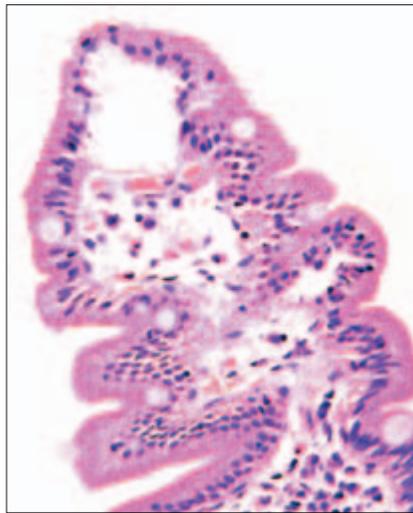
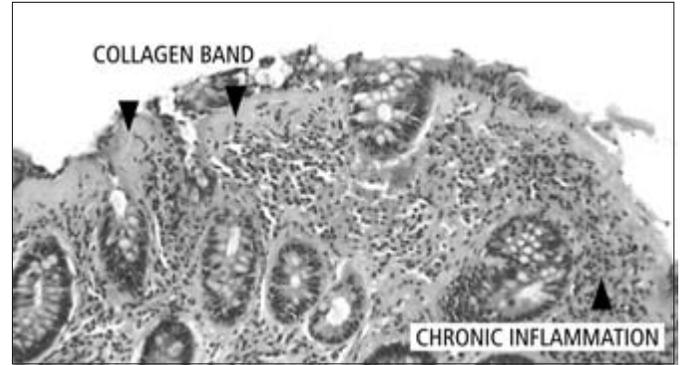
1.21



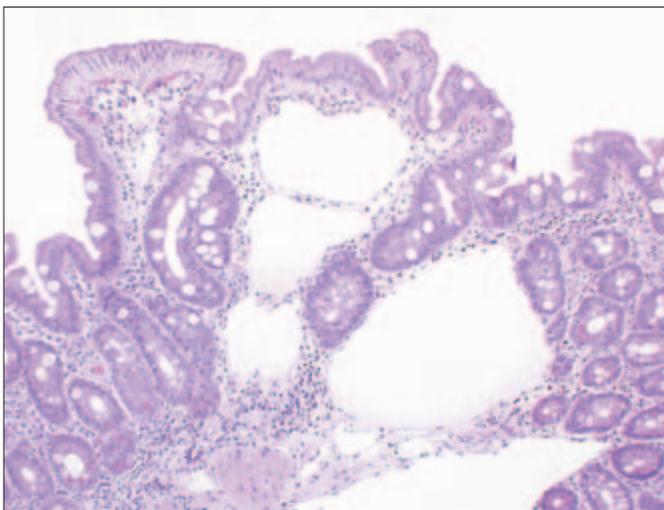
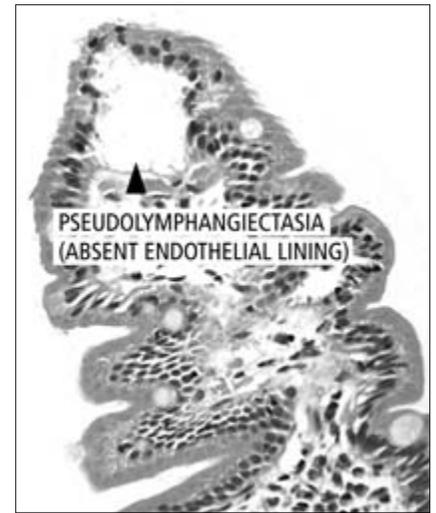
Disruption and telescoping of glands and separation of epithelium result in much confusion. Separation is a useful artefact in collagenous colitis when during processing and sectioning the surface of the epithelium lifts away from the abnormal collagen band (Fig. 1.22). Separation becomes a nuisance in the duodenum where pseudo-lymphangiectasia may result (Fig. 1.23); however, unlike those of true lymphangiectasia (Fig. 1.24), these spaces are not lined by endothelium. Retraction spaces also occur around tumour deposits which may make the diagnosis of vascular invasion difficult; special stains for vascular endothelial cells such as CD34 will usually resolve this problem.



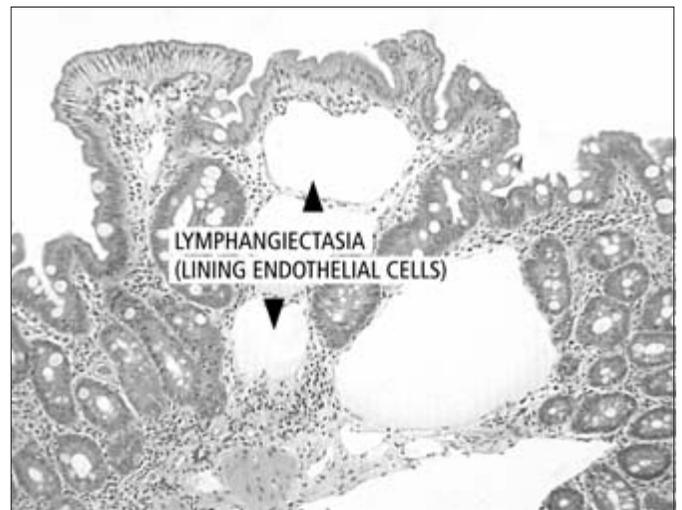
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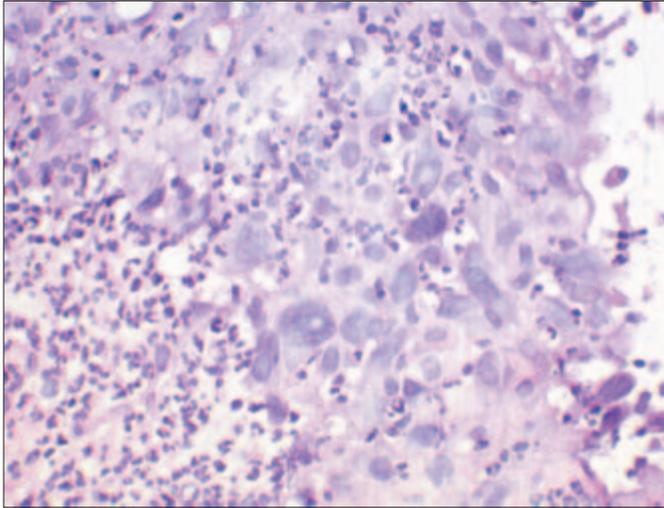
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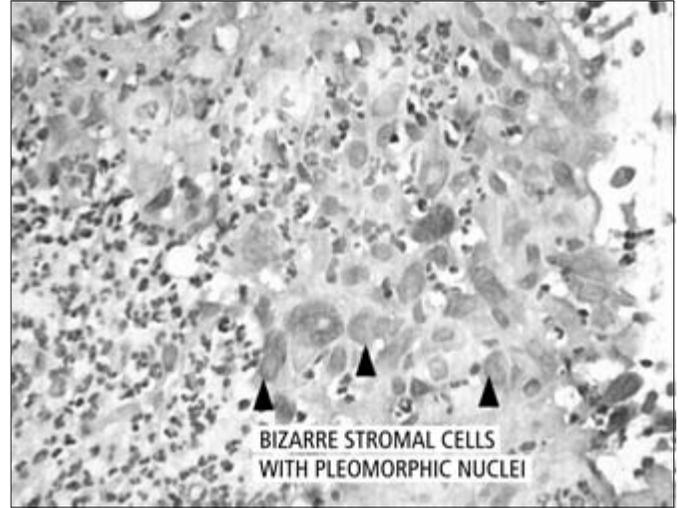
1.24



The most diagnostically dangerous artefact is crushing which in the biopsies from the stomach and oesophagus results in glandular crowding and nuclear pleomorphism simulating malignancy (Fig. 1.25). Whenever possible samples from the oesophagus should be taken before bougienage of strictures and if this is not feasible the pathologist should be informed appropriately. Smooth muscle tumours may be simulated by crush artefact producing a ball of smooth muscle from the muscularis mucosae.



1.25



### Clinicopathological meetings

Regular meetings should be held between clinical gastroenterologists, both physicians and surgeons, and their pathologist colleagues for clinical, educational and audit purposes. It may also be helpful if a radiologist can attend. At such meetings, routine material should be discussed as well as material from patients with unusual or rare conditions and those where clinical and pathological features do not correspond. The pathologist may wish to revise his/her diagnosis in the light of evidence emerging during discussion, or may be prompted to further investigation such as more levels or stains or to obtain previous biopsy material from his/her own file or from that of another hospital where the patient had previously been treated.

The presence of a formally timetabled clinicopathological meeting should never be a barrier to informal consultation in person. The gastroenterologist should be a welcome visitor to the pathology department at all times. Likewise, the pathologist should be an equally welcome visitor in the endoscopy suite, to familiarize him/herself with the challenges and limitations of endoscopy, to see interesting appearances, to help select biopsy numbers and sites, to collect unusual or urgent specimens, and to emphasize the close cooperation between clinician and pathologist which is essential to obtaining the best results.

# Upper Gastrointestinal Tract

<b>Normal appearances</b>	<b>19</b>
<b>Abnormal appearances</b>	<b>42</b>
<b>Postoperative appearances</b>	<b>168</b>
<b>Bleeding</b>	<b>177</b>
<b>Therapeutic procedures</b>	<b>187</b>
<b>Complications</b>	<b>226</b>

Upper gastrointestinal endoscopy is generally performed following pharyngeal anaesthesia and commonly employing light 'conscious' sedation; benzodiazepines are used most frequently. Careful clinical observation of the patient is essential throughout the procedure, and it is usual to employ additional devices such as a pulse oximeter.

## Normal appearances

Larynx  
Epiglottis  
Hypopharynx  
Trachea and bronchi  
Oesophagus  
Gastric mucosal prolapse during retching  
Lower oesophageal sphincter  
Oesophago-gastric mucosal junction  
Saliva obscuring clear view of stomach  
Residual gastric juice  
Appearance of stomach during inflation  
Cup-and-spill deformity: a normal variant  
Rugal and mucosal appearances: body and antrum

**Red lines in antrum**

**Antral contractions and the normal pylorus**

**Duodenal bulb**

**Duodenal mucosa**

**Upper duodenum: junction of bulb and second part**

**Bile in the duodenum**

**Duodenal froth**

**Circular folds**

**Congested duodenal lacteals**

**Papilla of Vater**

**Antrum, angulus and body from below**

**Gastro-oesophageal junction from below**

**Mucosa of body and fundus**

**Dark shadow-like appearances from adjacent viscera**

**Taking biopsy and cytological samples**

**Normal histological and cytological appearances**

It is common practice to introduce the endoscope 'blind', but many endoscopists perform this procedure under direct vision, allowing inspection of the pharynx and larynx. These areas are however, more commonly and more easily observed during extubation. The endoscopist must be familiar with the normal appearances of this region (Figs 2.1–2.7).

In the elderly or the heavily sedated patient, the endoscope may inadvertently slip into the trachea (Figs 2.8 and 2.9). The oesophagus is best examined during insertion (Figs 2.10–2.23) while it is easier to view the stomach in detail during withdrawal. Adequate distension of the stomach with air and removal of excess fluid by suction may be needed to enable gastric landmarks to be observed during introduction (Figs 2.24–2.31). The appearance and behaviour of most of the stomach and of the antropyloric area can easily be observed and noted (Figs 2.32–2.44) at this stage and it is advisable to complete this part of the examination before giving any gastroduodenal relaxant; at a later stage, suppression of peristalsis may be desirable. For routine upper gastrointestinal endoscopy (oesophago-gastro-duodenoscopy or OGD) a forward-viewing endoscope is used, though for special indications a fore-oblique or sideviewing instrument may be substituted. Normal upper duodenal appearances are shown in Figs 2.45–2.63. Using a forward-viewing endoscope it is rarely possible to see the papilla of Vater in detail (Figs 2.64 and 2.65) nor to advance the endoscope much beyond the papilla. (For enteroscopy specially designed instruments and particular techniques are available. Enteroscopy is described in Chapter 5.) The incisura and upper portions of the stomach are best viewed on withdrawal of the instrument. For this the upward-flexed or J-position (Figs 2.67–2.80) is favoured by some endoscopists. The endoscope must of course be straightened before being pulled back into the oesophagus.

It is necessary to be familiar with the technique of obtaining biopsy material and cytological brushings for microscopy. This, together with normal histological appearances, is discussed later in this chapter.

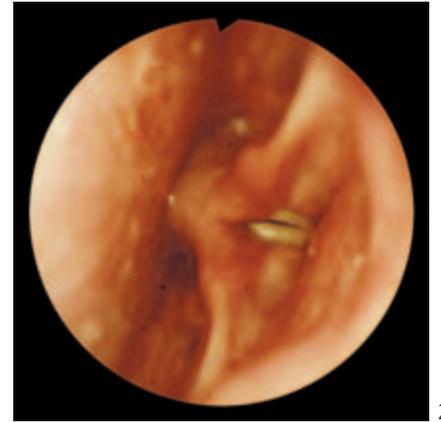
Familiarity with endoscopic routine and normal appearances is essential before the endoscopist can proceed with any confidence to the examination and interpretation of abnormalities, or therapeutic procedures.

### Larynx

As it is common practice to introduce the endoscope into the upper oesophagus 'blind' the larynx and hypopharynx are not usually seen at this stage. The area may, however, be inspected either during intubation or extubation. Movement of the vocal cords can be assessed by asking the patient to say 'ee' (Figs 2.1 and 2.2).



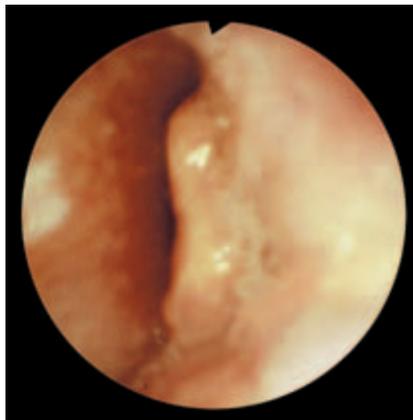
2.1



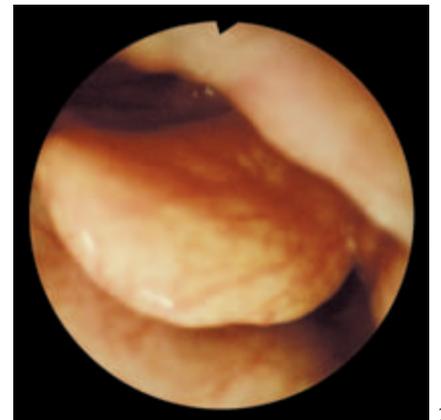
2.2

### Epiglottis

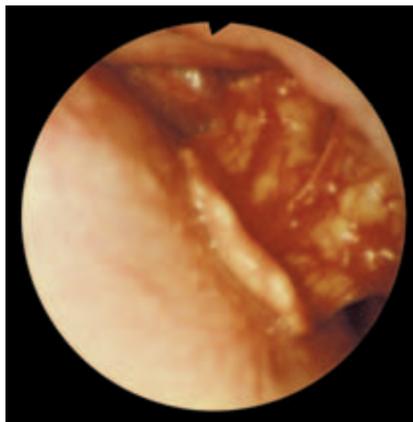
Figure 2.3 shows the epiglottis in its normal resting position, thrown forward. In Fig. 2.4 its anterior surface is shown after the epiglottis has moved backwards while in Fig. 2.5 the endoscope has been advanced showing the root of the epiglottis. Detailed examination of the epiglottis is not normally included in routine OGD.



2.3



2.4

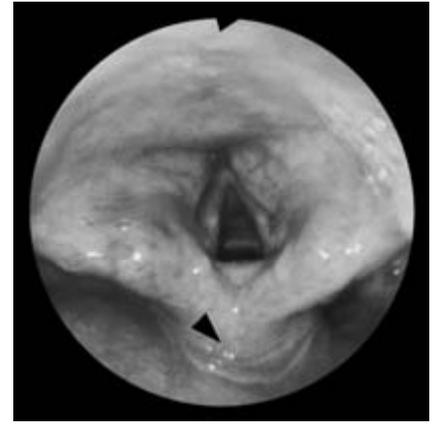


2.5

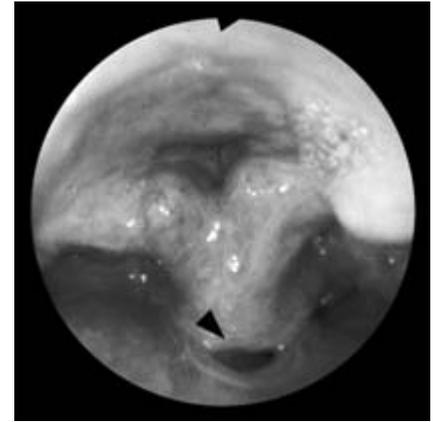
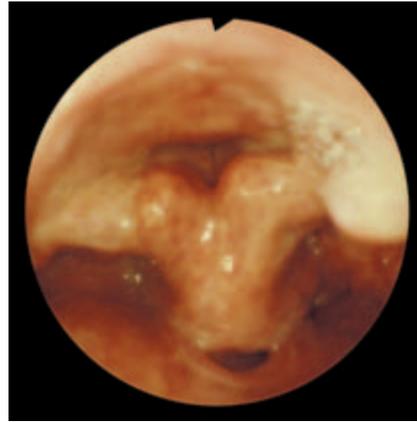
### Hypopharynx

Figure 2.6 demonstrates the posterior aspect of the larynx with the entrance to the oesophagus closed, while in Fig. 2.7 it has barely opened. The pyriform fossae lie each side of the oesophageal opening. The cricopharyngeal sphincter can be opened by asking the patient to swallow. The pyriform fossae are obliterated by this manoeuvre allowing the instrument safely to be advanced into the upper oesophagus.

It should be remembered that swallowing is difficult with the mouth open (as for example when a tooth guard is used), when the neck is extended or if the patient is 'chomping'.



2.6



2.7

### Trachea and bronchi

Occasionally the endoscope will enter the upper trachea, particularly in older heavily sedated patients or those who retch during intubation. If the instrument is immediately withdrawn to the pharynx this is unlikely to be of great consequence, although if coughing is provoked it is advisable to extubate the patient completely, and to restart the endoscopy after coughing has ceased. On occasions the trachea may be entered without this causing the patient to cough.

Figure 2.8 demonstrates the endoscopic appearances of the trachea, while in Fig. 2.9 the carina and main bronchi are illustrated.



2.8

2.9

## Oesophagus

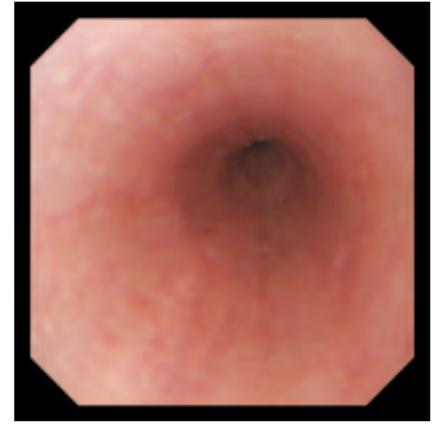
Figure 2.10 shows a normal oesophagus before distension with air. This will present as a whitish-pink tube, often with longitudinal folds. It is unusual for the upper oesophagus to show peristaltic movements though these are commonly seen in the middle and lower thirds.

The partially distended normal oesophagus is shown in Fig. 2.11. The appearances are often slightly lumpy and irregular, at first suggestive of an abnormal mucosa. Further inflation will, however, assuage such doubts.

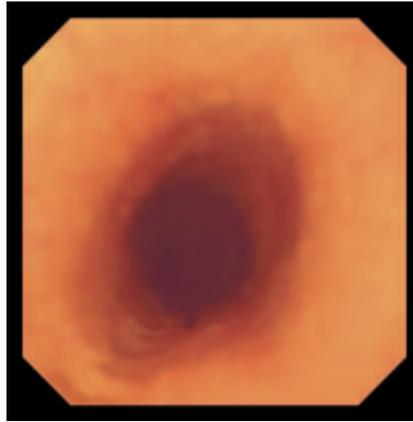
The fully distended normal oesophagus (Fig. 2.12) appears as a smooth featureless tube lined by pale pink mucosa. Small intraepithelial venules are commonly seen. The lower oesophagus is often whiter and the upper a little pinker, this contrast being more obvious in elderly subjects. The histological features of normal squamous oesophageal mucosa are seen in Fig. 2.13.



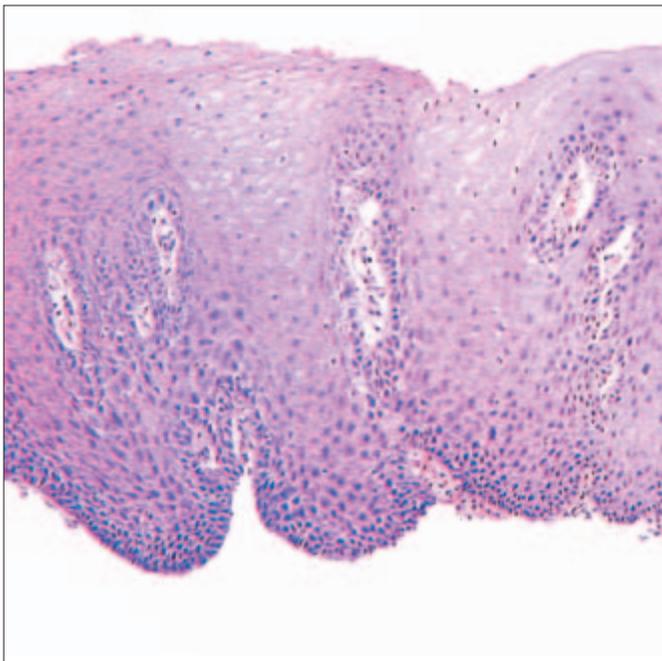
2.10



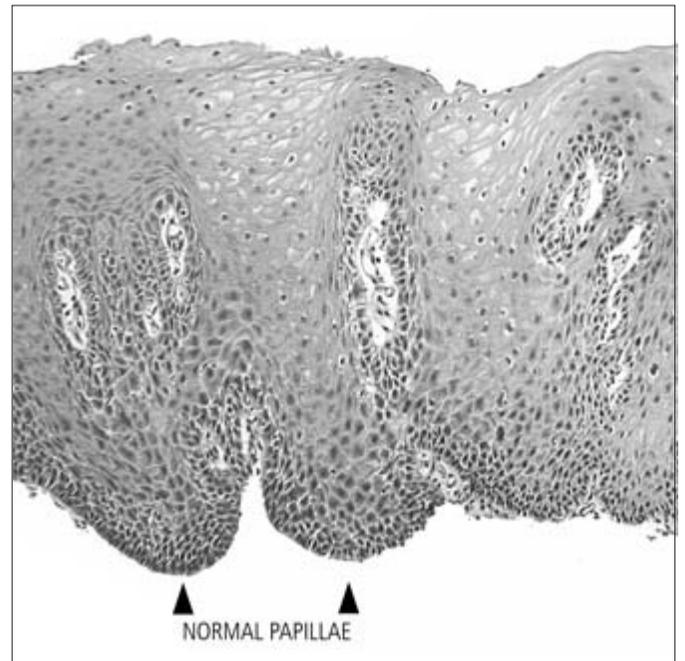
2.11



2.12



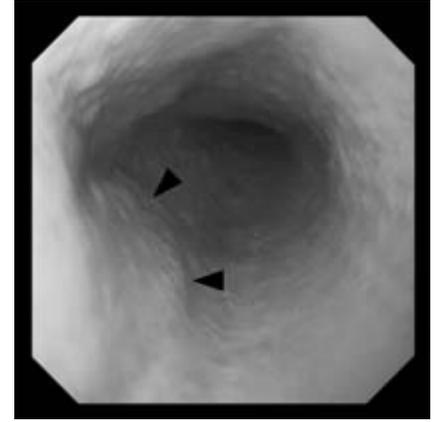
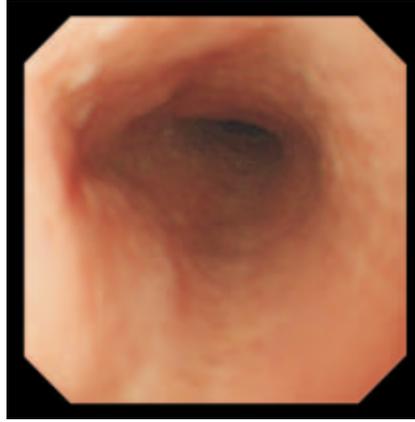
2.13



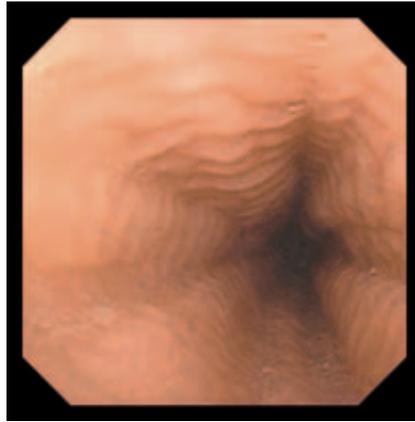
### Oesophagus (*cont.*)

The aortic indentation into the otherwise smooth oesophageal outline forms a useful landmark at about 25 cm from the incisors (Fig. 2.14). Being pulsatile it is easily distinguished from other extrinsic deformities. Transmitted cardiac pulsation is commonly seen in the lower oesophagus.

It is not unusual to see a multitude of tiny transverse ridges crossing the longitudinal folds (Fig. 2.15). These ridges disappear during peristaltic relaxation or as a result of distension with air. They are caused by reflex spasm of the muscularis mucosae and have been described as ‘oesophageal frisson’.



2.14



2.15

### Gastric mucosal prolapse during retching

Introduction of the endoscope into the oesophagus often causes retching. This brings about an increase in oesophageal movement and belching of air introduced by the endoscopist, making examination of the oesophagus difficult. With the tip of the endoscope situated in the lower oesophagus, momentary prolapse of the redder gastric mucosa can often be seen (Fig. 2.16). This process, when it occurs during frank vomiting, is associated with significant shearing strains on the gastric mucosa and is the basis of the Mallory–Weiss tear (Fig. 2.445) or so-called hernia gastropathy (Fig. 2.444).

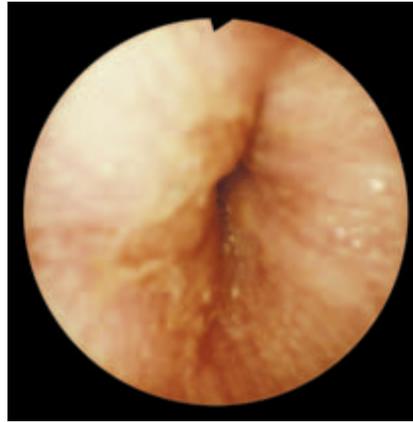


2.16

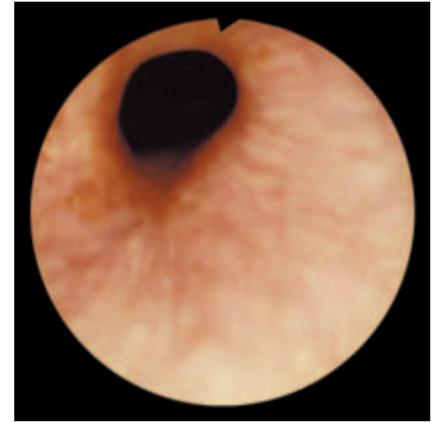
### Lower oesophageal sphincter

The oesophagus is seen to be distended above the closed sphincter (Fig. 2.17). These appearances are also called the oesophageal fleurette. Note the venous palisade mentioned below.

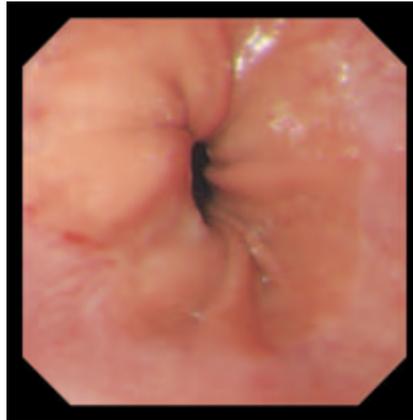
When the sphincter is relaxed, the lumen at the oesophago-gastric junction is normally round or nearly so. Particularly in the younger subject, a fine longitudinally running venous palisade is often seen just above the squamocolumnar epithelial junction. An exaggeration of normal or no clinical significance appears in Fig. 2.18. Figure 2.19 shows a less evident palisade.



2.17



2.18



2.19

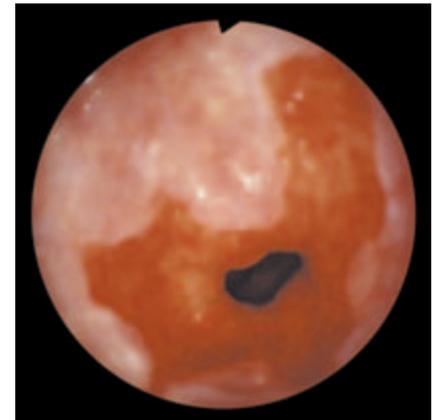
### Oesophago-gastric mucosal junction

It is not always easy, even in normal subjects, to see the squamocolumnar epithelial junction, also known as the dentate or Z-line. In older subjects, this line may be more pronounced because the lower oesophageal mucosa is often relatively paler in colour. The dentate line is sometimes regular, giving the appearance of a sharp ring-like demarcation (Fig. 2.20), though an irregular line is equally common (Fig. 2.21).

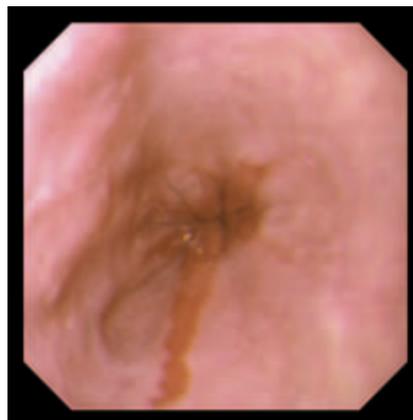
Sometimes there may be finger-like extensions of gastric mucosa running proximally (Fig. 2.22). Other normal variations include islands of oesophageal mucosa surrounded by gastric mucosa, and vice versa (Fig. 2.23). Similar variations in the appearance of the oesophago-gastric mucosal junction are seen in Barrett's oesophagus (Figs 2.143 and 2.145).



2.20



2.21



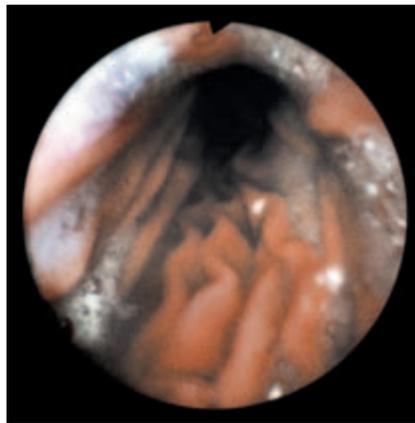
2.22



2.23

### Saliva obscuring clear view of stomach

It is not always possible to obtain good views when entering the stomach. Swallowed saliva (Fig. 2.24), or regurgitated bile-stained yellow froth (Fig. 2.57), may present difficulties. Bubbles may be dispersed by introduction of a silicone-containing defoaming agent or by suction to remove the offending material. Excessive suction may traumatize the mucosa causing petechiae or suction artefacts (Figs 2.447 and 2.448). Some endoscopists ask the patients to drink a small quantity of defoaming agent before proceeding to OGD.

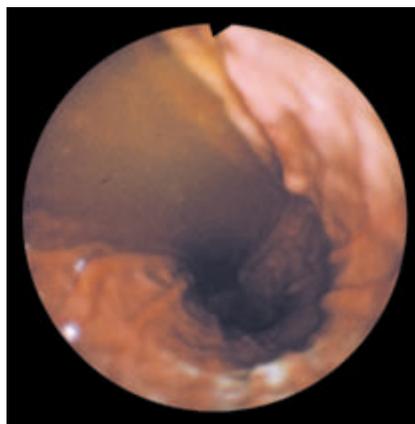


2.24

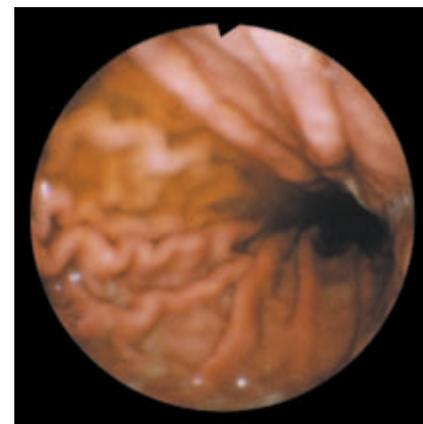
### Residual gastric juice

Saliva (Fig. 2.24) and regurgitated frothy duodenal contents (Fig. 2.57) are difficult to remove, whilst residual gastric juice (Fig. 2.25) can easily be aspirated through the endoscope. This enables a complete gastric survey to be undertaken with greater confidence (Fig. 2.26). Residual gastric juice is sometimes called 'the mucus lake'. When inflating the stomach, care should be taken to introduce the air above and not through residual juice, lest further bubbles be formed.

Excessive fluid seen in the stomach at this stage of the examination is commonly due to the patient having drunk fluids immediately before the procedure. Preparation for OGD should include a period during which no food or fluid is consumed. The minimum period of starvation is probably 4–6 h; an increasing number of endoscopists place no restrictions on the intake of clear fluids.



2.25

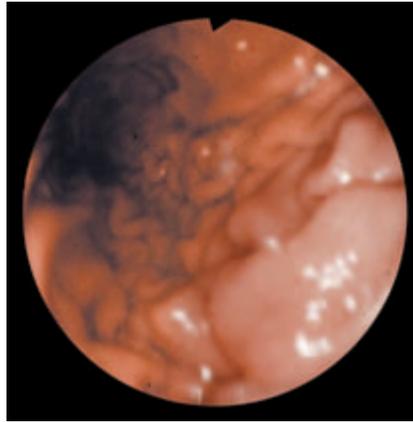


2.26

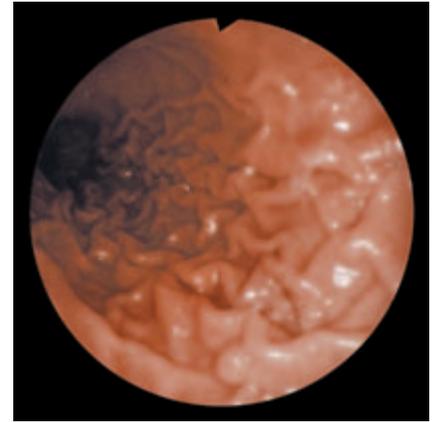
### Appearances of stomach during inflation

When first entering the stomach, the rugae appear lumpy or tangled and the way forward may not immediately be apparent (Fig. 2.27). During inflation the rugae straighten and appear more linear, running towards the antrum and causing the appearance of the so-called 'Magenstrasse' (Figs 2.28 and 2.29). The rugal pattern may disappear altogether with full gaseous distension (Fig. 2.30).

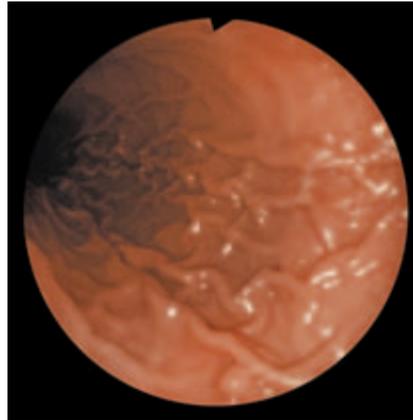
The relative prominence of the rugal folds varies between normal subjects and may be less obvious in the elderly.



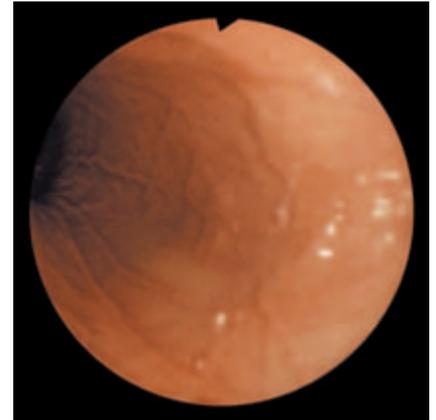
2.27



2.28



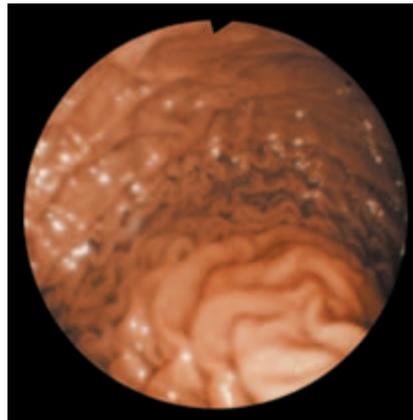
2.29



2.30

### Cup-and-spill deformity: a normal variant

When there is a cup-and-spill deformity, it is sometimes difficult to pass the endoscope from the fundus into the body of the stomach. Curling of the instrument may occur in the fundus causing disorientation. To overcome this problem, the instrument should be withdrawn so that the tip is just below the cardia; after identifying the likely way forward, slow advancement into the antrum is usually possible under direct vision. During this procedure, the stomach should be well distended so as to facilitate recognition of the usual landmarks. Figure 2.31 shows a well-marked ridge dividing the 'cup' from the body.



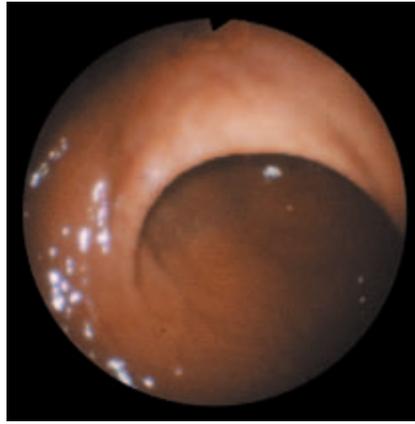
2.31

**Rugal and mucosal appearances:  
body and antrum**

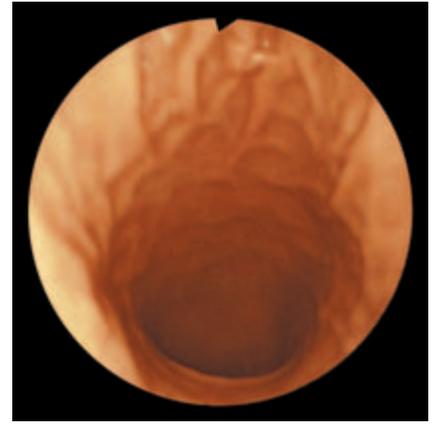
The rugae covering the greater curve and the posterior wall are more marked than those of the lesser curve.

It is not always possible to see the incisura (angulus) from above during introduction of the endoscope. In the relaxed distended stomach it usually appears as a distinct fold (Fig. 2.32), occupying about a third to a half of the circumference of the stomach at the junction with the body, where rugae are normally well seen, and the antrum, which is rather smoother. The incisura can easily be distinguished from a ring formed during antral contraction (Fig. 2.40). Figure 2.33 illustrates the gradual flattening of the rugae at the junctional area between the body and the antrum, in a patient where there was no easily recognized incisura.

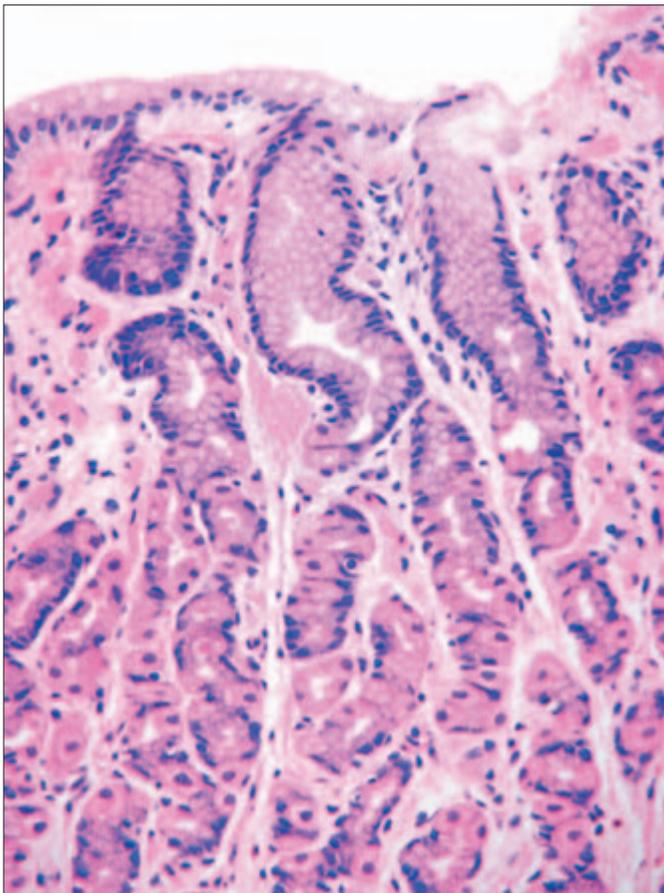
To the endoscopist the appearances of normal body and antral mucosa are indistinguishable. Histologically there are however, important differences (Figs 2.34 and 2.35).



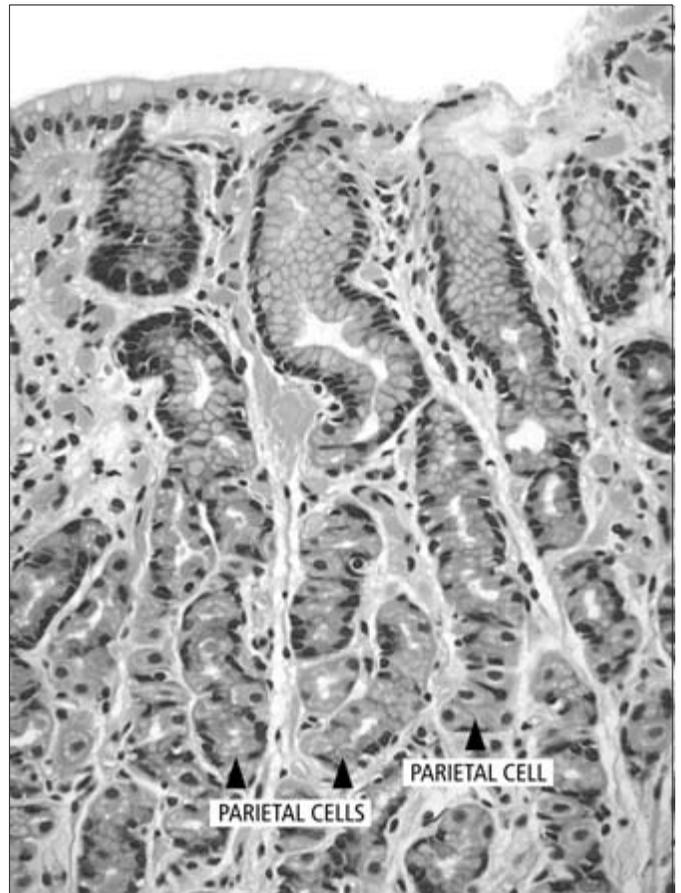
2.32

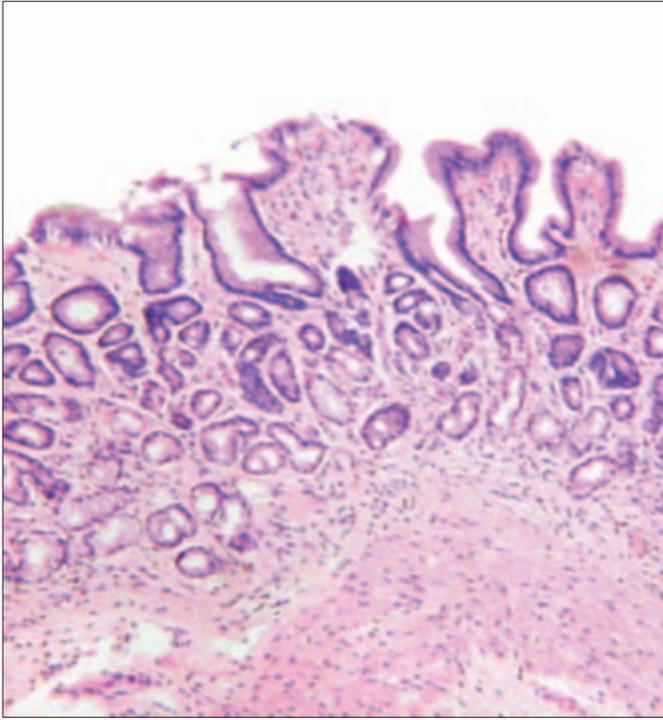


2.33

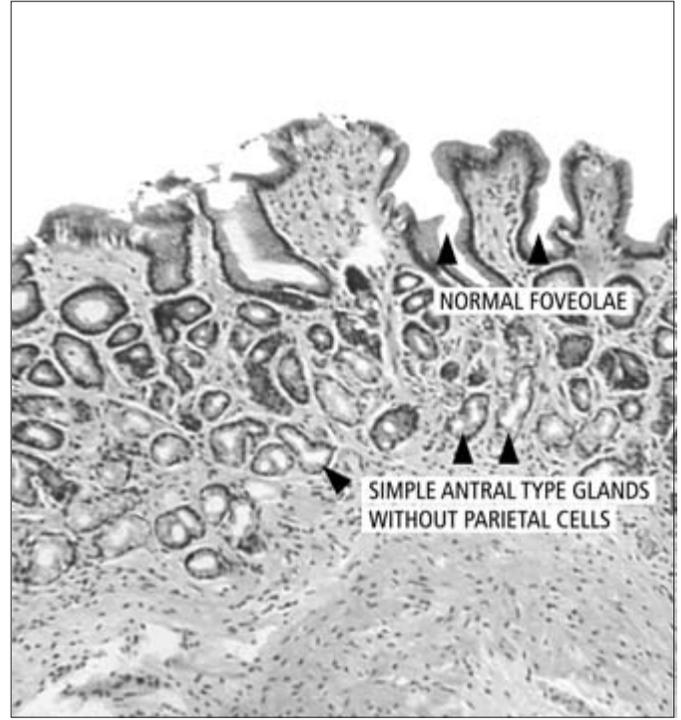


2.34





2.35



**Red lines in antrum**

The normal antrum shows longitudinal rugae only during antral contractions. It is common to find red crests along these rugae, appearing as thinner (Fig. 2.36) or thicker (Fig. 2.37) lines when the rugae disappear during antral relaxation. These appearances are a normal variant and of no significance.



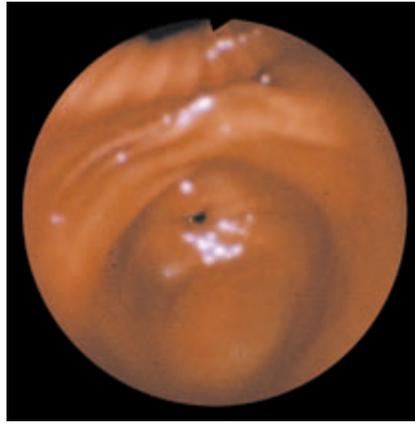
2.36



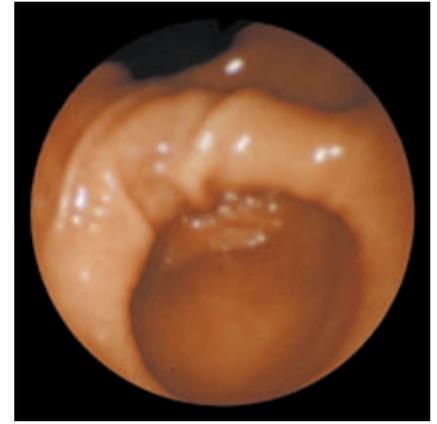
2.37

**Antral contractions and the normal pylorus**

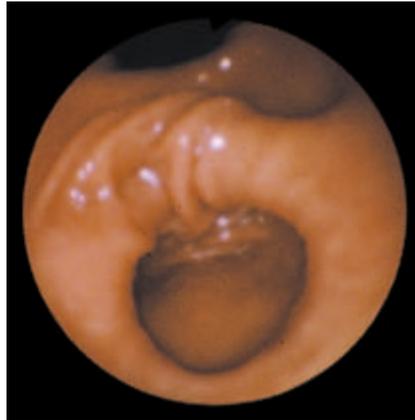
Antral contractions (Figs 2.38–2.42) are often seen during routine OGD. They occur more commonly in nervous subjects, and may be absent in the deeply sedated. The contractions occur approximately every 20 s and travel distally along the antrum in circumferential symmetrical fashion. During the brief period of antral and pyloric diastole the normal pylorus appears as a round aperture, sometimes on a flat surface (Fig. 2.43) and sometimes slightly raised (Fig. 2.44). It is commonly eccentric with respect to the longitudinal axis of the antrum, lying more towards the upper border. Antral contractions are eliminated by smooth muscle relaxants such as hyoscine *N*-butyl bromide.



2.38



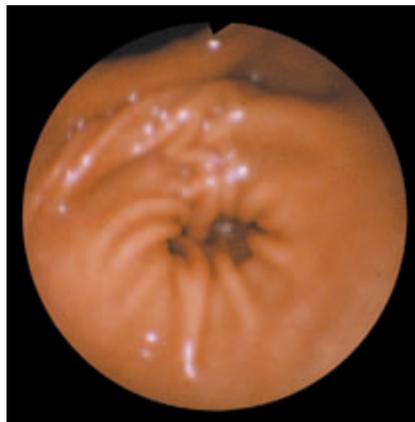
2.39



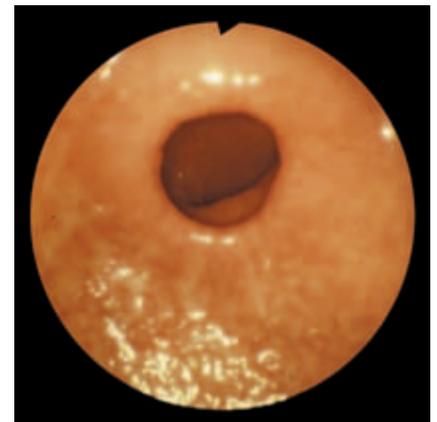
2.40



2.41



2.42



2.43

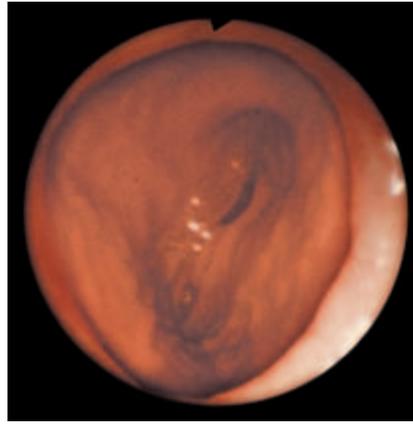


2.44

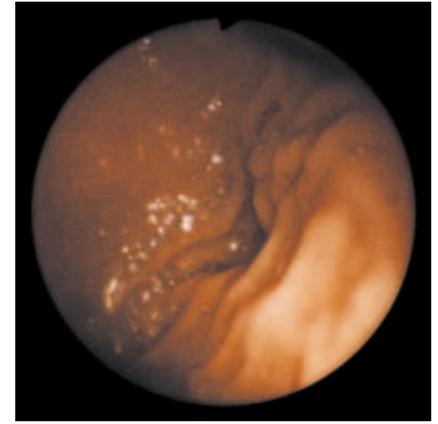
### Duodenal bulb

Ideally the tip of the endoscope should be held at the pyloric orifice before letting it slip into the bulb, so that an overall view of the bulb can be obtained (Fig. 2.45). This may be facilitated if a gut relaxant is given. The normal pylorus is a ring or diaphragm, rather than a channel; existence of a channel suggests the presence of ulcer, oedema or scarring.

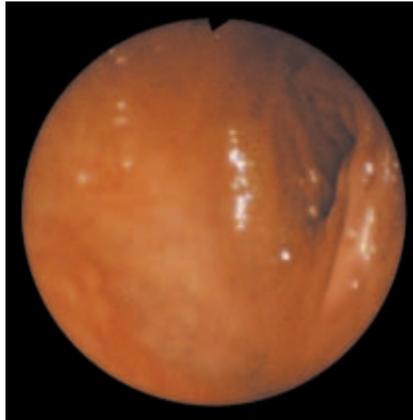
The first part of the duodenum (duodenal bulb or cap) can be seen quite well as the endoscope is advanced through the pyloric ring, and before distension with air will appear as a semicollapsed ridged structure (Fig. 2.46). After distension it assumes a bulb-like outline with smooth walls (Fig. 2.47) and a fold at the apex, the superior duodenal fold, marking the junction between the first and second parts of the duodenum. The bulb is usually seen to better advantage on withdrawal of the instrument from the more distal parts of the duodenum.



2.45



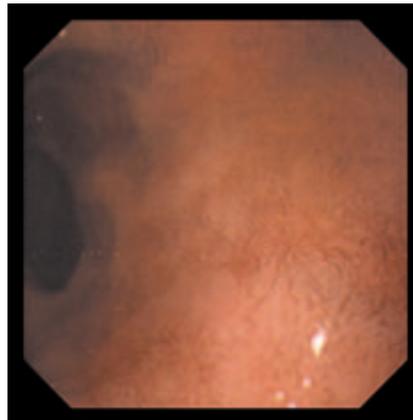
2.46



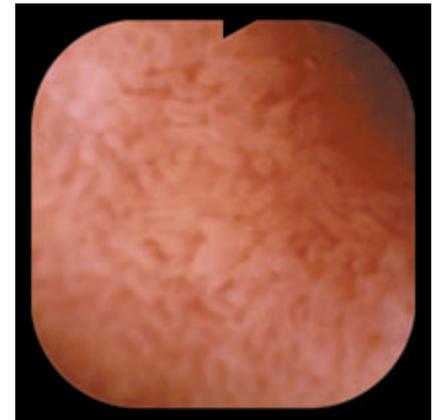
2.47

### Duodenal mucosa

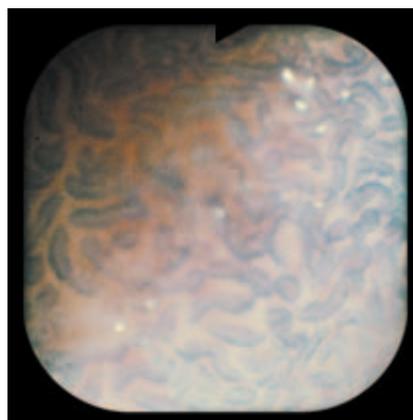
The surface of the duodenum is normally made up of digitate villi and, to a lesser extent, leaf and ridge forms. Although the bulbar mucosa, as seen with a standard endoscope, at first usually appears quite smooth, more careful examination may reveal a granular pattern (Fig. 2.48). With certain focusing and magnifying endoscopes this appearance can be more fully appreciated (Fig. 2.49), and the effect can be accentuated by the use of supravital staining techniques with, for example, methylene blue (Fig. 2.50).



2.48



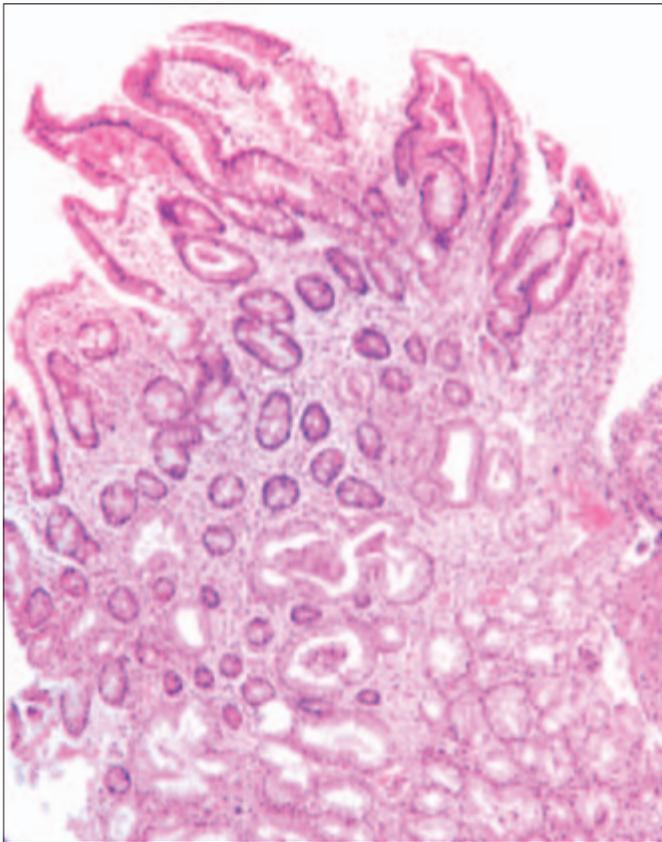
2.49



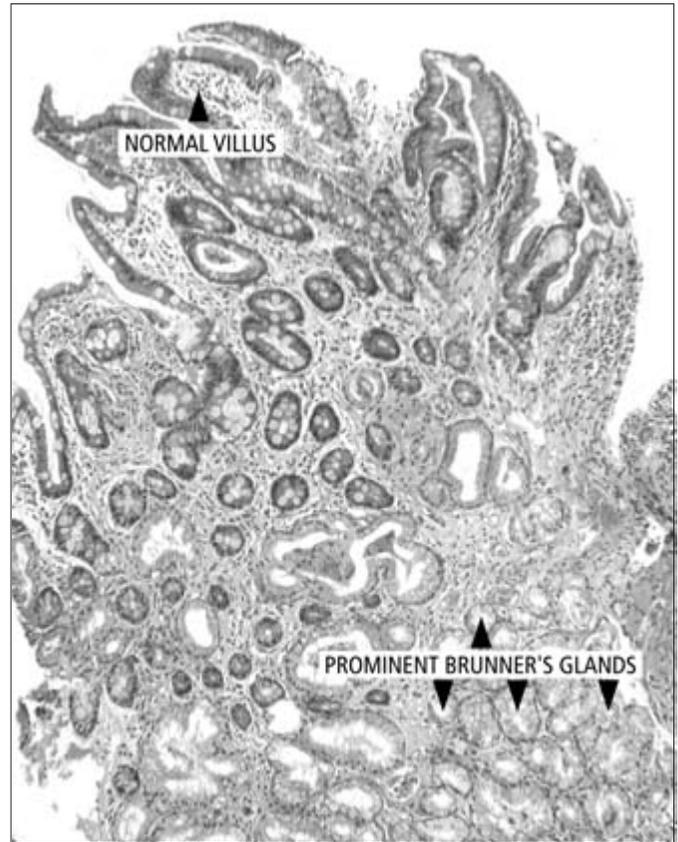
2.50

**Duodenal mucosa (cont.)**

The normal histology of the bulbar mucosa is shown in Fig. 2.51.

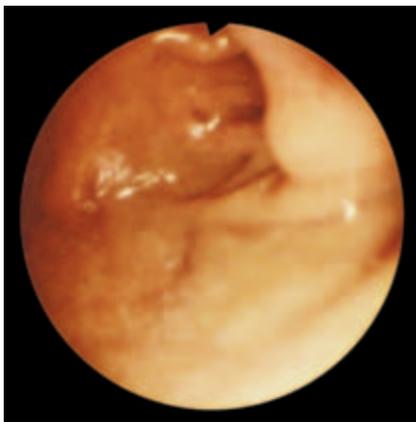


2.51

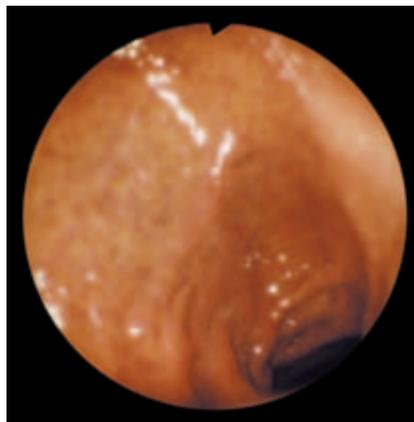


**Upper duodenum: junction of bulb and second part**

In Fig. 2.52 the apex of the duodenal bulb and the superior duodenal fold are seen in greater detail. If the tip of the endoscope is carefully advanced so that it lies under this fold, the uppermost portion of the second part of the duodenum will come into view (Fig. 2.53). When the endoscope first passes the superior duodenal fold and enters the descending duodenum, a series of crowded smooth folds is seen (Fig. 2.54). The pattern of these folds cannot at this stage of the procedure be distinguished.



2.52



2.53



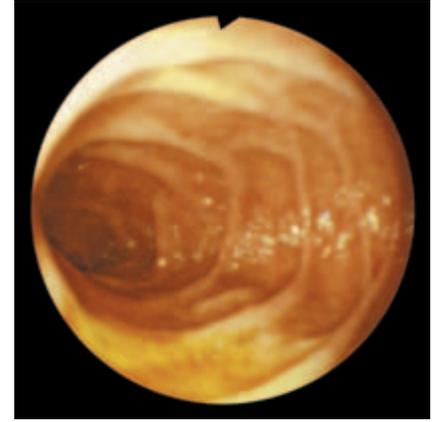
2.54

### Bile in the duodenum

With further distension the tubular structure of the duodenum becomes evident. The dependent spaces between the valvulae conniventes are often filled by bile (Fig. 2.55) which can easily be aspirated (Fig. 2.56). The small bowel mucosa may look browner than gastric mucosa, even after removal of bile.



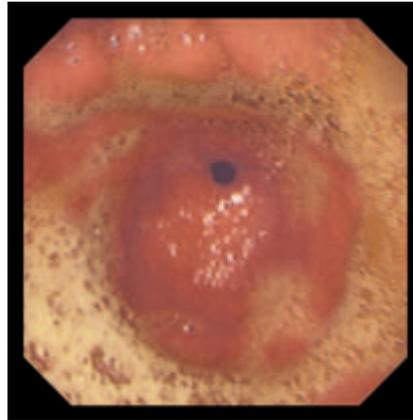
2.55



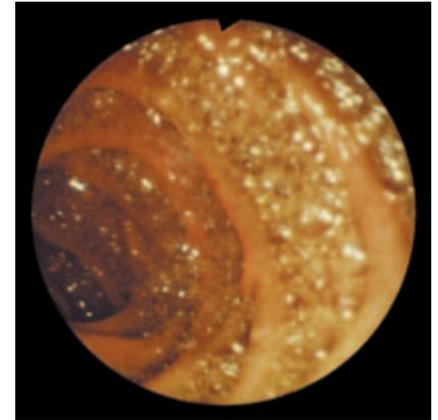
2.56

### Duodenal froth

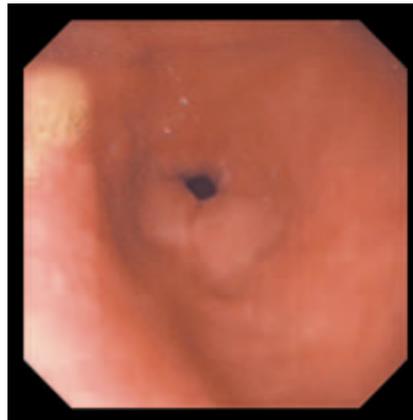
It is common to see yellow bile-stained froth in the antrum (Fig. 2.57) and in the upper duodenum (Fig. 2.58). Air insufflation makes the situation worse and often clear vision is impaired. Water washing may help and if this is not satisfactory instillation of a silicone-containing suspension will disperse the froth (Fig. 2.59).



2.57



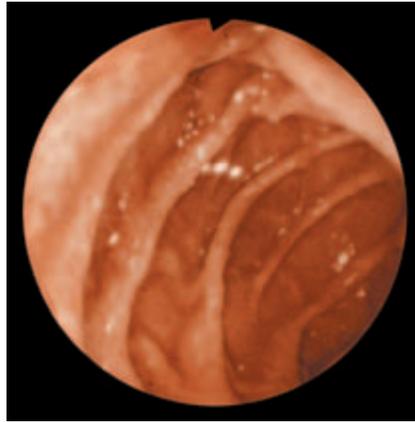
2.58



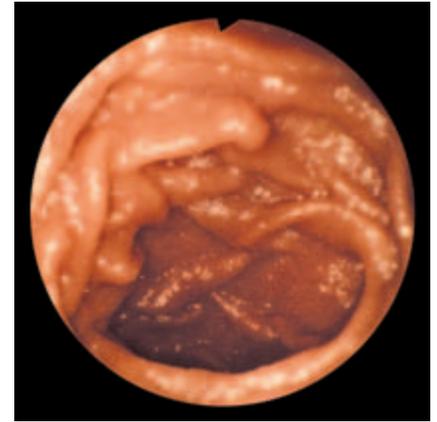
2.59

**Circular folds**

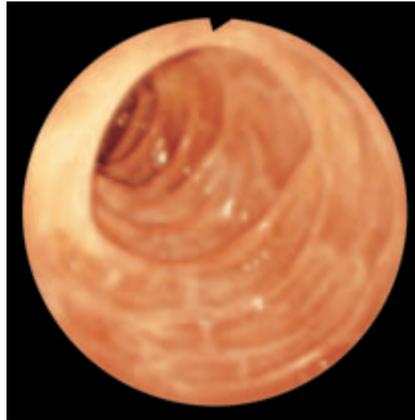
Circular folds, also known as valvulae conniventes and rings of Kerckring, occur below the duodenal bulb. These may form thin, ring-like structures (Fig. 2.60), or may be thicker and produce a more irregular pattern (Figs 2.61 and 2.62).



2.60



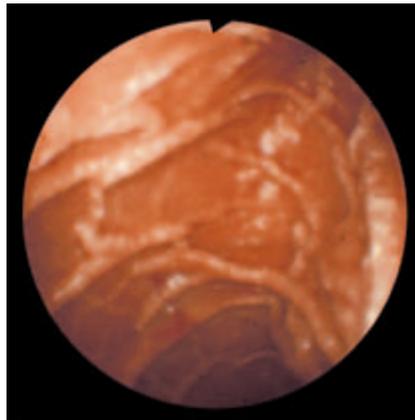
2.61



2.62

**Congested duodenal lacteals**

This normal variation (Fig. 2.63) is not commonly seen. It causes a carpet of tiny white spots.



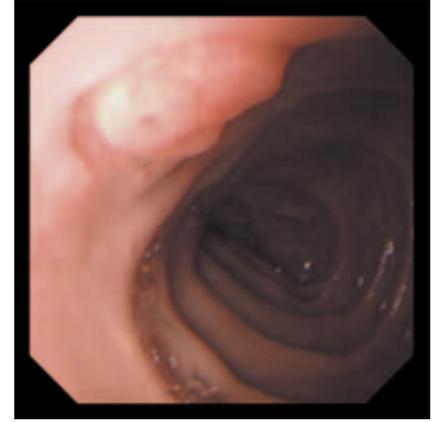
2.63

### Papilla of Vater

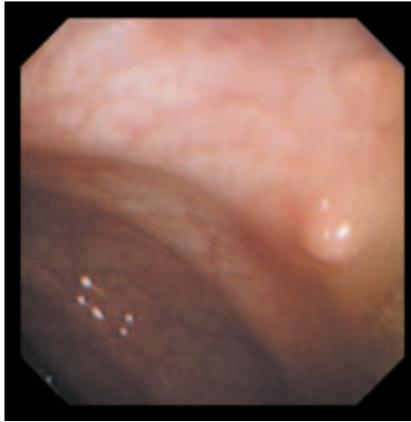
Using a forward-viewing endoscope, this is frequently overlooked as it may lie between folds, often at the proximal end of a short longitudinally running fold. When found it is nipple-like, granular and a pinkish colour (Figs 2.64 and 2.65). It is most unusual to see an accessory papilla during routine OGD. If present, it lies to the right but quite near the papilla of Vater. There may be only a blind polyp-like structure (Fig. 2.66). Proper inspection of the papillae requires the use of a side-viewing endoscope.



2.64



2.65

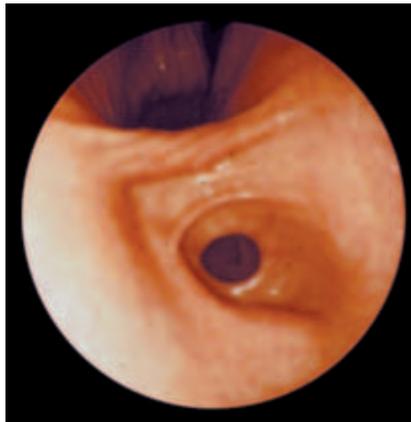


2.66

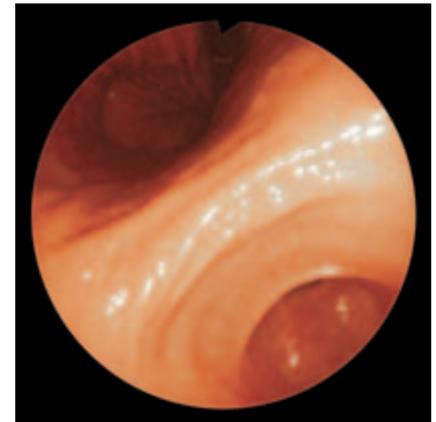
### Antrum, angulus and body from below

The flexible tip of the modern endoscope can invariably be angulated to 180° enabling a J-manoeuvre to be performed with ease. If this is done when the tip of the endoscope lies just proximal to the pylorus, the upper surface of the antrum and the angulus can be inspected from below.

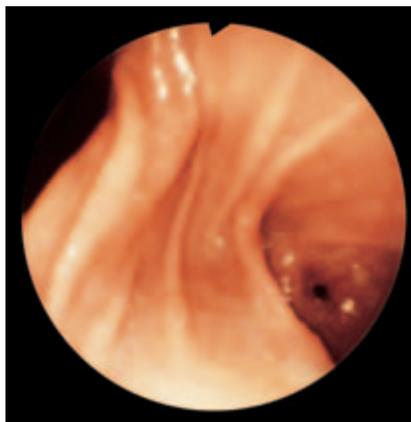
Figure 2.67 shows the pylorus and a well-defined angulus, or incisura. In Fig. 2.68 the endoscope is pulled back a little and the angulus is shown in greater detail. A common variant, a rather wider, flatter angulus, is shown in Fig. 2.69.



2.67



2.68



2.69

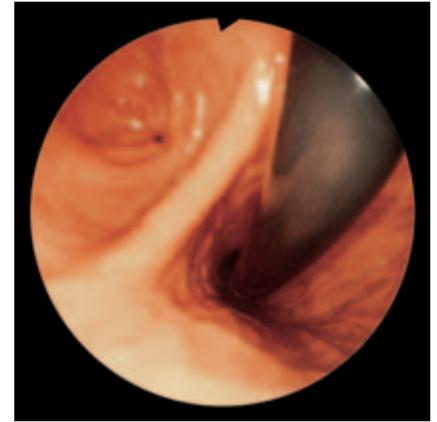
**Antrum, angulus and body from below (cont.)**

By withdrawing the endoscope further and if necessary rotating it as appropriate, it is usually possible to view simultaneously the antrum and pylorus (Fig. 2.70), and angulus and body of the stomach (Fig. 2.71).

Increasing insufflation and further withdrawal of the endoscope in the J-position will reveal first the partially collapsed (Fig. 2.72) and then the fully distended gastric body (Fig. 2.73). The shaft of the endoscope will lie prominently in the centre of the field. This is an especially useful manoeuvre for a detailed inspection of the lesser curve, gastro-oesophageal junctional area and fundus.



2.70



2.71



2.72

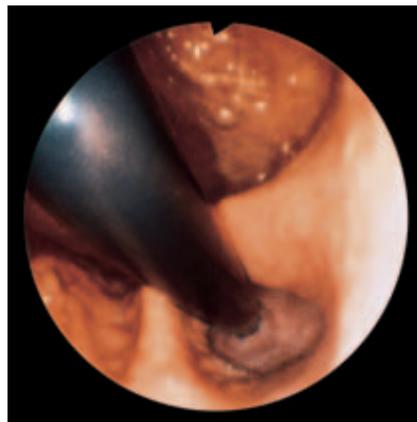


2.73

**Gastro-oesophageal junction from below**

The flap-valve normally covering the gastro-oesophageal junction is easily seen on inversion, with the shaft of the endoscope coming through the junction (Fig. 2.74). Pulling the endoscope back further reveals the gastro-oesophageal mucosal junction (Figs 2.75 and 2.76). The retroflexed endoscope should not normally be withdrawn beyond this point: impaction of the tip in the lower oesophagus can occur (when it may be necessary to pass a second instrument in parallel, for use as a 'rammer').

Although the flap-valve usually presents a somewhat flat appearance, a more rugose appearance (Fig. 2.77) is not uncommon.



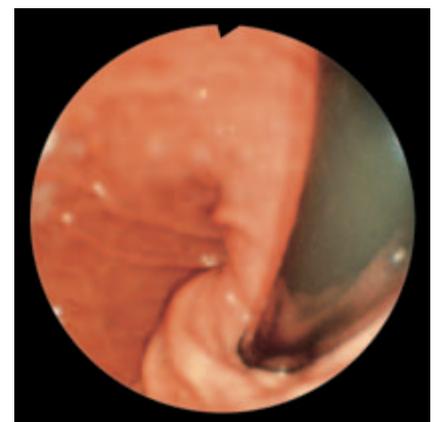
2.74



2.75



2.76

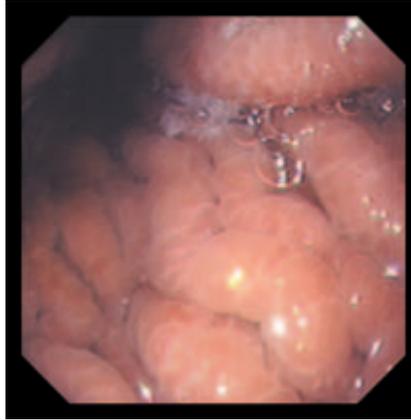


2.77

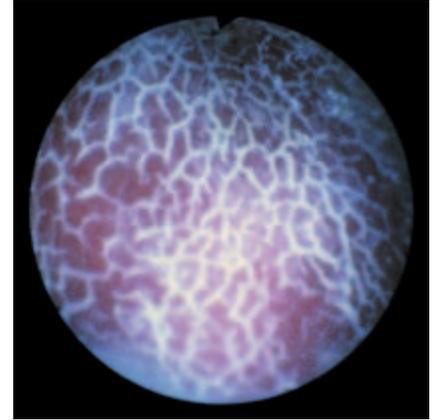
### Mucosa of body and fundus

The rugae of the body of the stomach usually appear uniformly smooth but, on closer inspection, the *areae gastricae* (Fig. 2.78) may be seen. Figure 2.79 shows these well outlined by residual barium suspension when endoscopy was performed shortly after an upper gastrointestinal series.

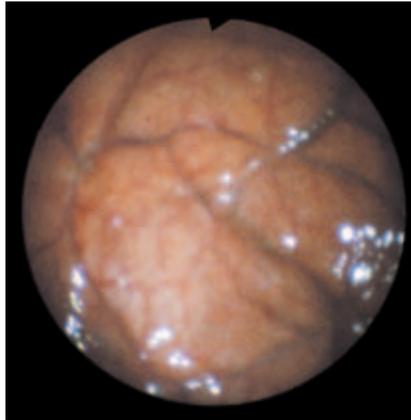
In contrast with the mucosal appearances of the body and antrum, the fundus (Fig. 2.80) shows a more marked vascular pattern somewhat resembling the appearances seen elsewhere in the stomach when there is gastric mucosal atrophy (Fig. 2.265).



2.78



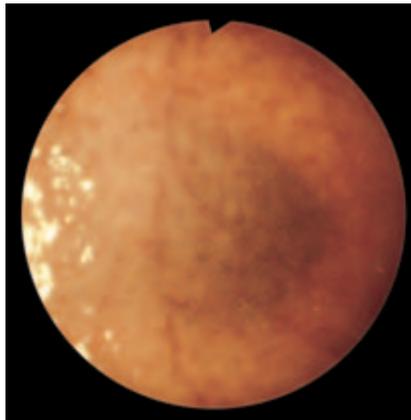
2.79



2.80

### Dark shadow-like appearances from adjacent viscera

Occasionally a dark, shadow-like appearance will be seen through the gastric (Fig. 2.81) or duodenal wall; depending on the exact site this may be liver or other normal or abnormal structures. It is important to differentiate between intramural lesions which will not alter shape or move with respiration, and extramural lesions which may do so; Figs 3.21 and 3.22 show such appearances during colonoscopy.



2.81

### Taking biopsy and cytological samples

Mention of this topic is included in the chapter on normal appearances as biopsies are often taken when appearances are normal to the endoscopist, though abnormalities may be evident to the pathologist. Important examples would include dysplasia in Barrett's oesophagus, some types of gastritis or inflammatory bowel disease.

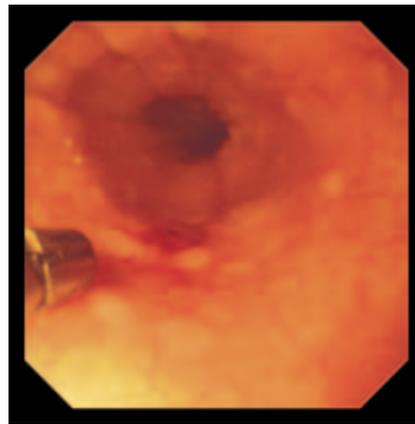
Biopsies are taken with spiked forceps (Fig. 2.82); the spike prevents the device from slipping along the smooth mucosal surface (Fig. 2.83). There will be tenting of the mucosa before the specimen separates (Fig. 2.84). A little bleeding, of no clinical significance, is common following mucosal biopsy (Fig. 2.85). (The forceps appear in this figure as another specimen is about to be taken.) The specimen when retrieved is held between the jaws of the closed forceps and on opening it is sometimes impaled on the spike. When necessary biopsies may also be taken using hot biopsy forceps (p. 331) and to obtain a so-called large particle biopsy (p. 332).

Cytological material is obtained by rubbing a cytology brush over the area of interest. Only sheathed cytology brushes (Fig. 2.86) should be used.

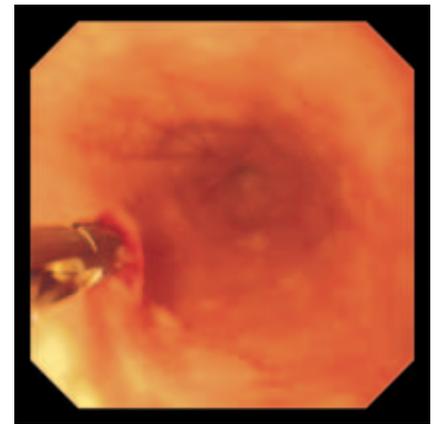
Taking biopsy and cytological samples is also referred to in various other sections of this Atlas as appropriate. In particular the reader is referred to the relevant sections of Chapter 1.



2.82



2.83



2.84



2.85

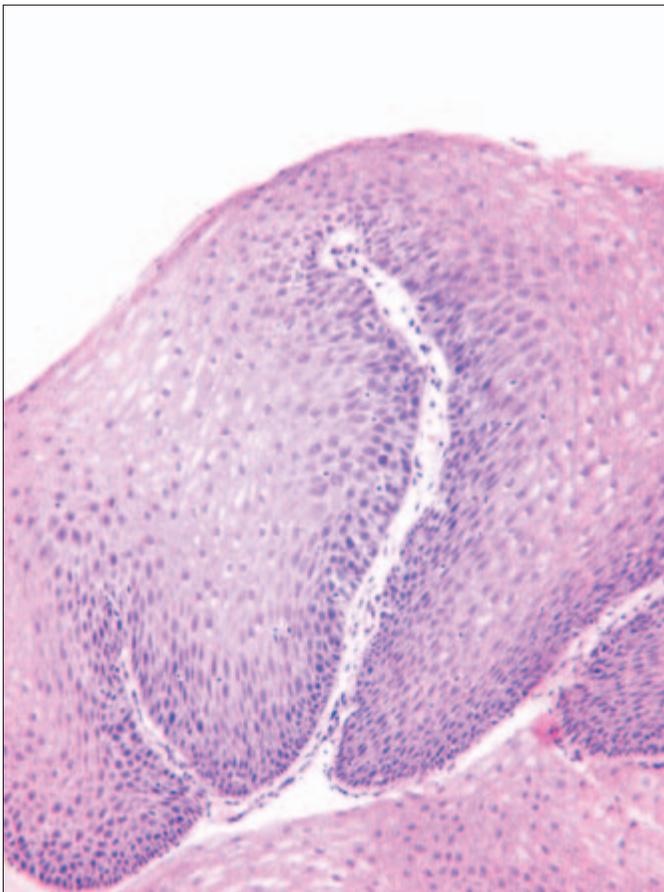


2.86

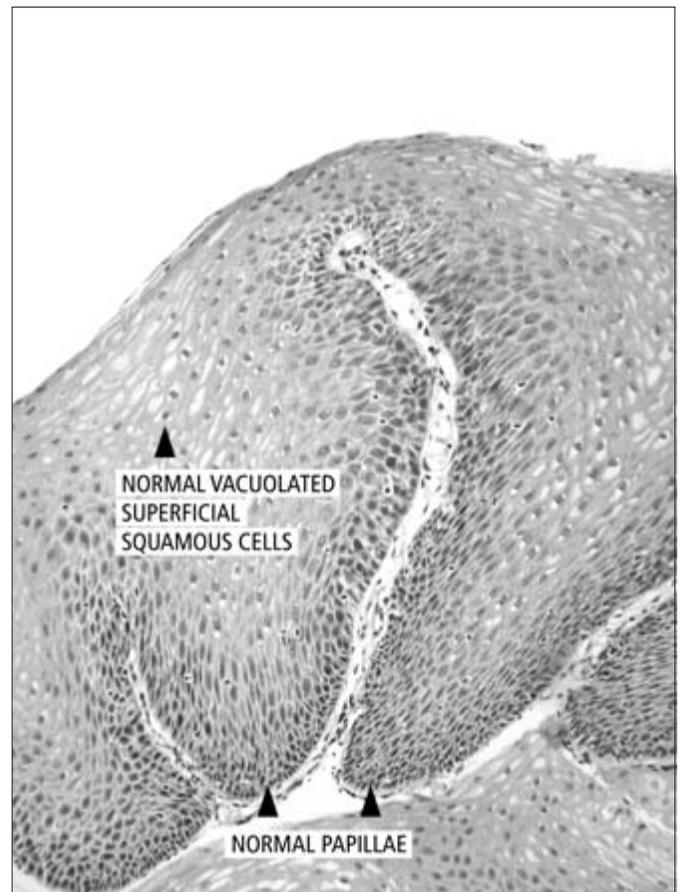
### Normal histological and cytological appearances

These are illustrated as follows: squamous oesophageal mucosa (Figs 2.13 and 2.87), mucosa from the gastric cardia (Fig. 2.88), body of the stomach (Figs 2.34 and 2.89) and the gastric antrum (Figs 2.35 and 2.90), and from the first (Figs 2.51 and 2.91) and third parts (Fig. 2.92) of the duodenum. Normal cytological appearances from oesophageal brushings appear in Fig. 1.15.

Normal oesophageal squamous mucosa has the characteristics of squamous epithelium elsewhere in the body with mild palisading of cells at the deeper part of the mucosa, and the cells expand as they reach the surface to include more cytoplasm. In many areas in the oesophageal squamous mucosa (as in the anal squamous mucosa), there is some vacuolation of the superficial squamous cells. Figure 2.87 shows these appearances from the normal papillae up to the vacuolated surface and also includes a small amount of underlying normal tissue.



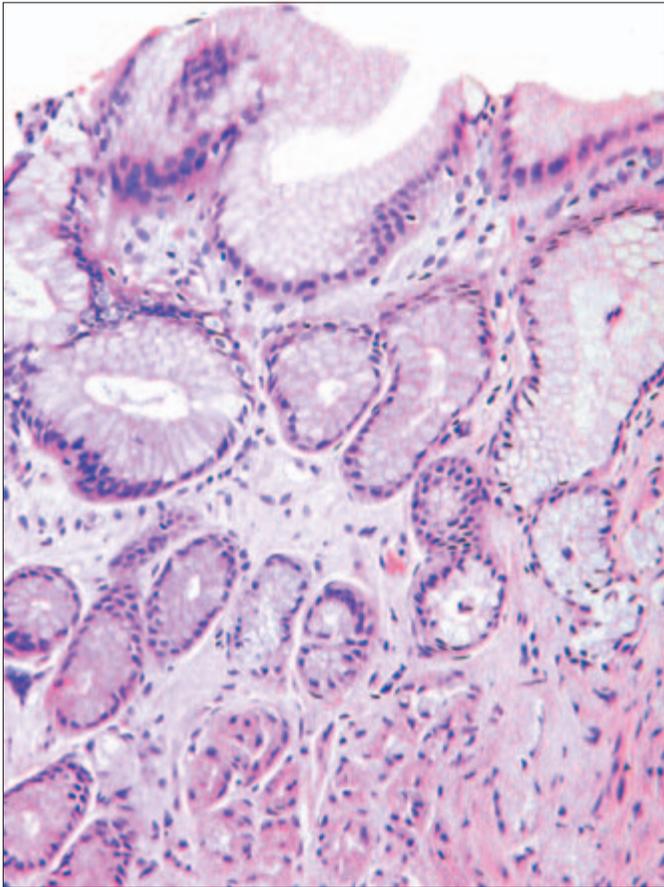
2.87



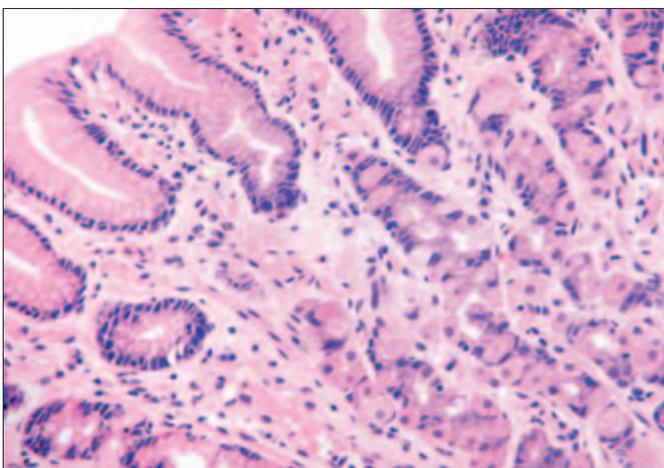
**Normal histological and cytological appearances (cont.)**

There has long been debate as to whether the mucosal appearances at the cardia represent a normal state, or whether they are the result of a pathological process. The type of mucosa found at the cardia is a simplified glandular mucosa which may include occasional parietal cells but is mainly comprised of simplified mucous glands (Fig. 2.88). Many authors now believe that cardiac type mucosa is a response to inflammation and injury; it is not present in neonates.

The mucosa of the body of the stomach has considerably more parietal cells than the cardia and certainly more than the antrum where only scattered parietal cells may be seen. The body mucosa has a rather compact appearance shown in Fig. 2.89; atrophy is identified by loss of parietal cells. Occasional small groups of lymphoid cells are seen within the normal gastric body.



2.88



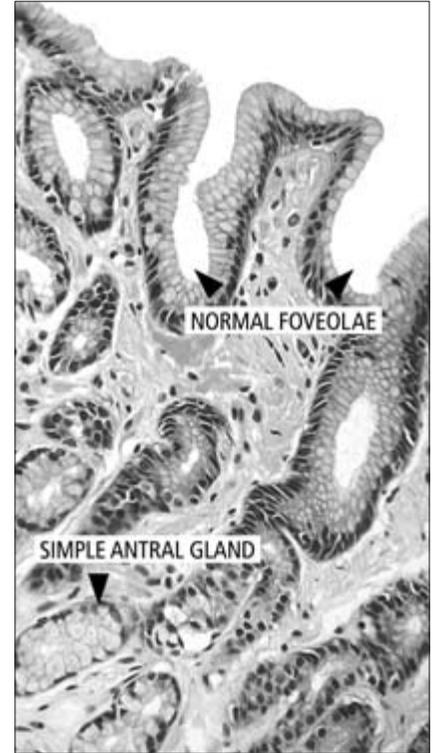
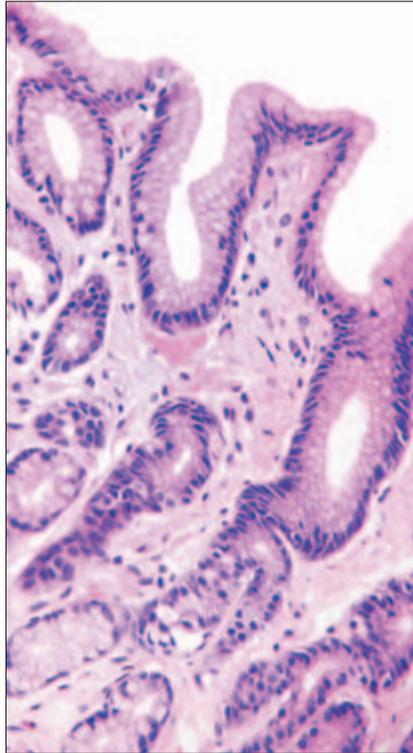
2.89



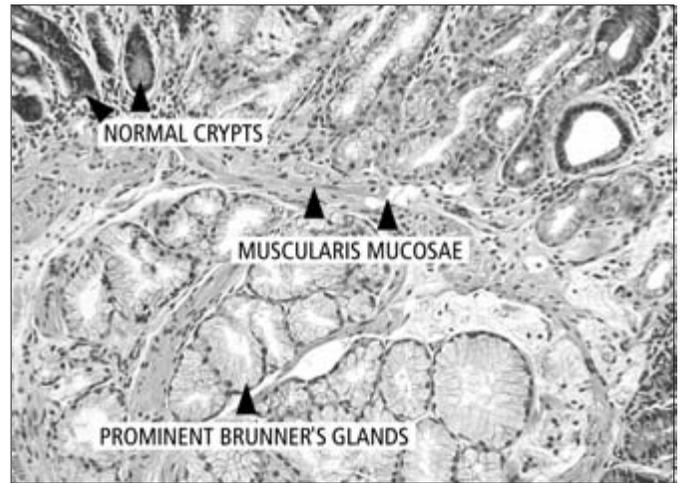
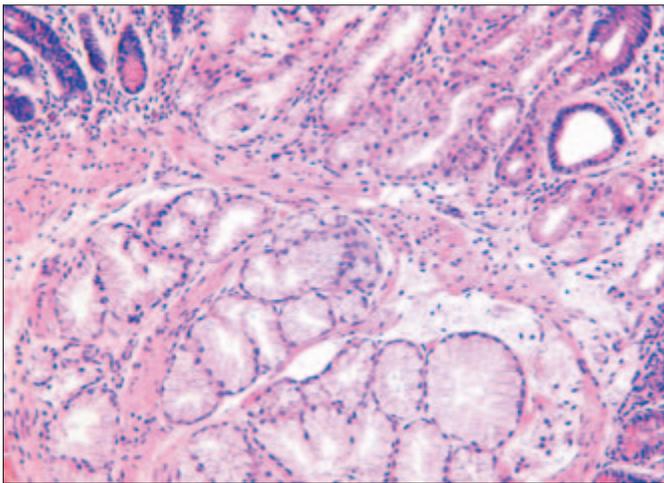
**Normal histological and cytological appearances (cont.)**

Gastric antral mucosa (Fig. 2.90) is a simplified glandular mucosa which has very sparsely scattered chronic inflammatory cells within the lamina propria; the foveolae are almost straight and the glands have a simplified appearance. There are only very occasional scattered parietal cells. In the duodenum the microscopic anatomy varies with site. In the first part of the duodenum, large and plentiful Brunner's glands are noted as shown in Fig. 2.91 and these are situated mainly below the muscularis mucosae.

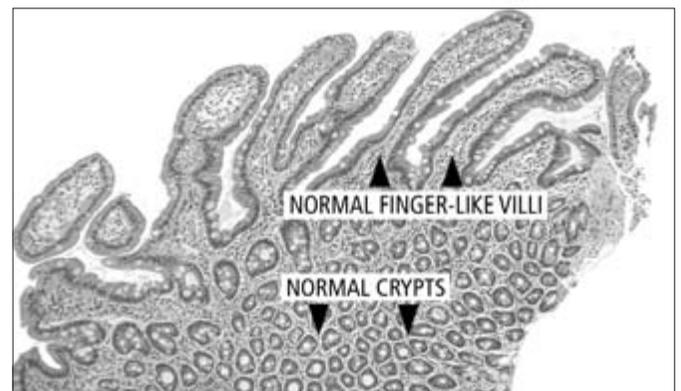
In the third part of the duodenum the Brunner's glands are lost (Fig. 2.92). This illustration of the superficial part of mucosa from the third part of the duodenum shows several villi of normal height and shape and a normal crypt/villus ratio. Some of the crypts are cross-cut but the normal villous architecture can still be identified.



2.90



2.91



2.92

---

## Abnormal appearances

**OESOPHAGUS 42**

**STOMACH 81**

**DUODENUM 140**

In the next section where abnormal appearances of the oesophagus, stomach and duodenum are discussed, some apparent duplication is inevitable as, for example, benign ulcers look similar wherever their site, though there may be differences. Where lesions appear identical, illustrations are not always duplicated in the various sections. Only passing reference is made in this section when a more detailed description appears elsewhere, e.g. in the section on bleeding.

### OESOPHAGUS

**Diverticula 42**

**Disorders of calibre and motility 44**

**Rings and webs 47**

**Hiatal hernia 48**

**Gastro-oesophageal reflux disease 49**

**Barrett's oesophagus 53**

**Benign and malignant tumours 59**

**Vascular abnormalities 68**

**Miscellaneous conditions 70**

**Suggested biopsy sites 80**

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#### Diverticula

**Congenital diverticula**

**Acquired diverticula**

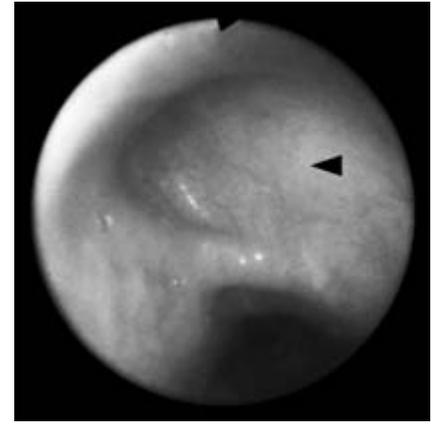
During endoscopy, there is a risk of perforating an unsuspected oesophageal diverticulum, especially a pharyngeal pouch (Zenker's diverticulum). When forewarned by the findings of a previous barium study, the endoscopist can approach areas of abnormality with greater confidence and should be able to avoid difficulty. However, barium studies are not usually available before upper gastrointestinal endoscopy but perhaps should be requested in complete dysphagia.

### **Congenital diverticula**

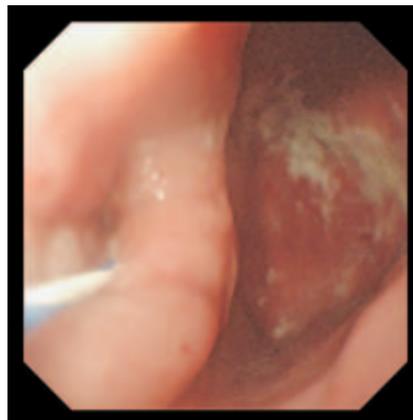
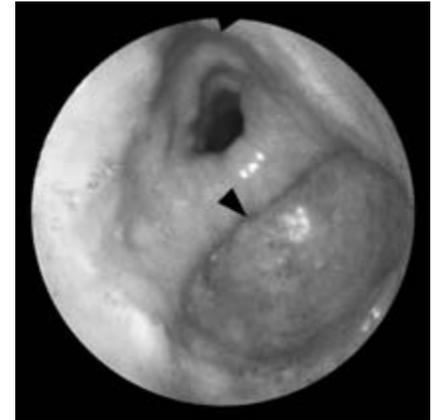
Congenital oesophageal diverticula may be quite shallow (Fig. 2.93) when they are easily missed endoscopically. Wide-mouthed deeper diverticula are more easily seen (Fig. 2.94). In Fig. 2.95 there is a large deep pouch. The oesophageal lumen was difficult to see at endoscopy; in this image, a guide wire has been passed to demonstrate the lumen and to aid onward passage of the endoscope. By contrast, Fig. 2.96 demonstrates a small but deep diverticulum in the lower oesophagus, an incidental finding in a patient with severe oesophagitis.



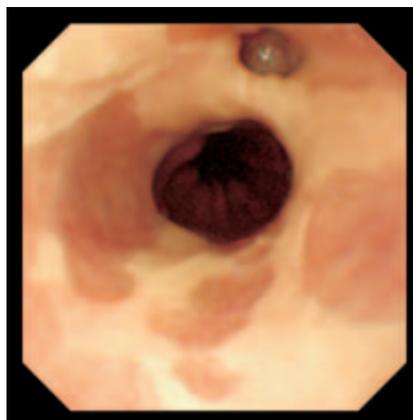
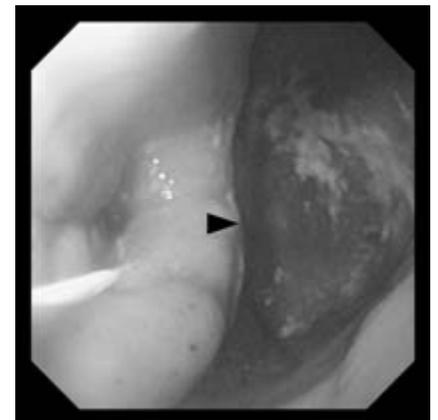
2.93



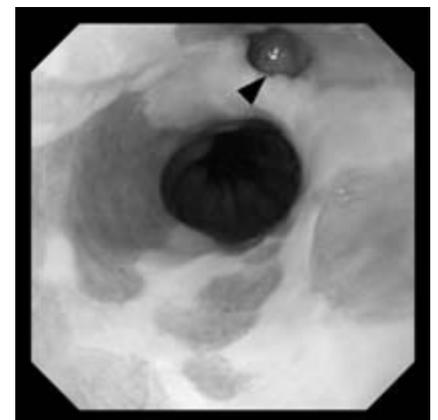
2.94



2.95



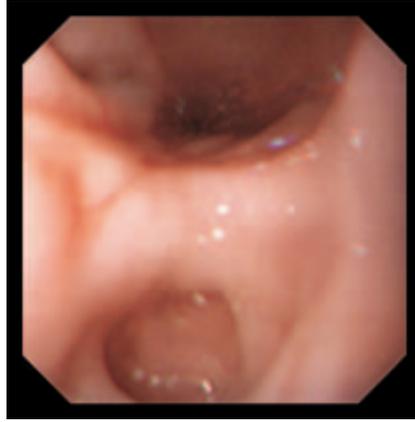
2.96



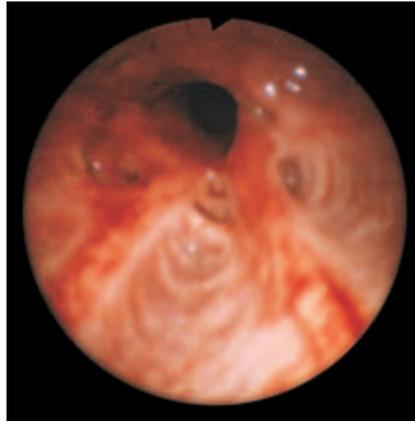
### Acquired diverticula

A diverticulum is sometimes associated with previous inflammation within the mediastinum, resulting in a so-called traction diverticulum. In patients with diffuse oesophageal spasm there may be pulsion diverticula.

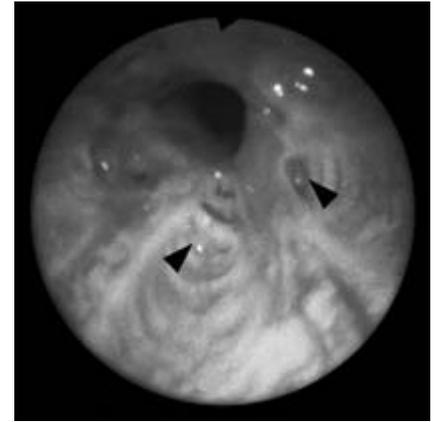
The inflammatory process which eventually leads to formation of a benign oesophageal stricture may also cause local diverticulum formation. Figure 2.97 shows a well-healed stricture with an associated simple diverticulum while in Fig. 2.98 there is a complex pattern of several diverticula lying between cicatricial ridges. The occasional presence of such diverticula associated with a benign stricture underlines the danger of blind as opposed to endoscopy-guided dilatation (see pp. 197–201).



2.97



2.98



## Disorders of calibre and motility

### Achalasia

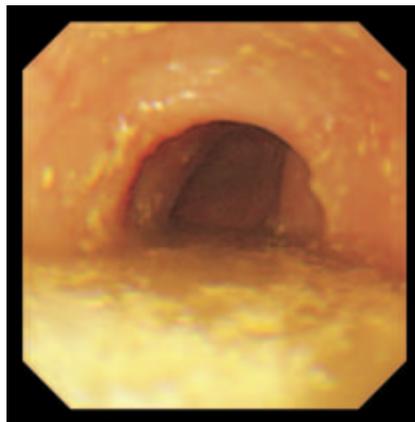
#### Progressive systemic sclerosis

#### Mixed connective tissue disease

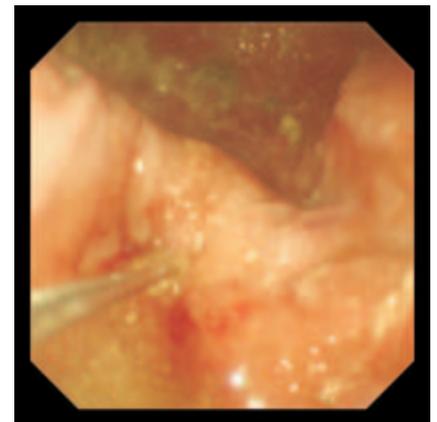
When oesophageal dysmotility is suspected, manometric, radiological and radionuclide-based investigations are more rewarding than endoscopy. Nevertheless, endoscopy has a place in the diagnosis and management of this group of conditions.

### Achalasia

In classical achalasia the body of the oesophagus is dilated and aperistaltic, and there is often evidence of retained food or fluid (Fig. 2.99). The spastic gastro-oesophageal junction can usually be passed with ease. Exceptionally it may be very tight in which case a guide wire, closed biopsy forceps or a polythene tube (Fig. 2.100) can be employed as a guide.



2.99



2.100

**Achalasia (cont.)**

Figure 2.101 shows a lower oesophageal squamous cell carcinoma complicating longstanding achalasia.

Vigorous achalasia is shown in Fig. 2.102, and a corkscrew oesophagus in Fig. 2.103.



2.101



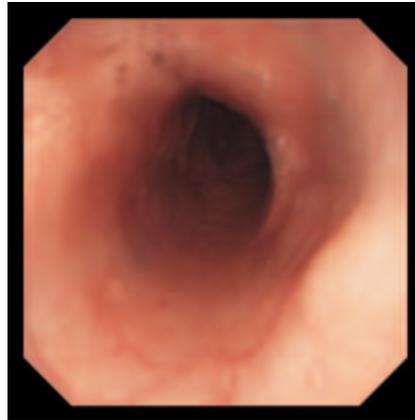
2.102



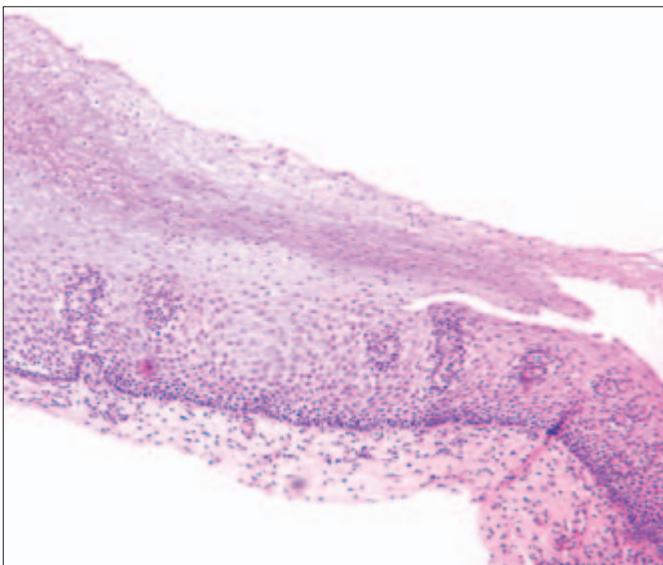
2.103

**Progressive systemic sclerosis**

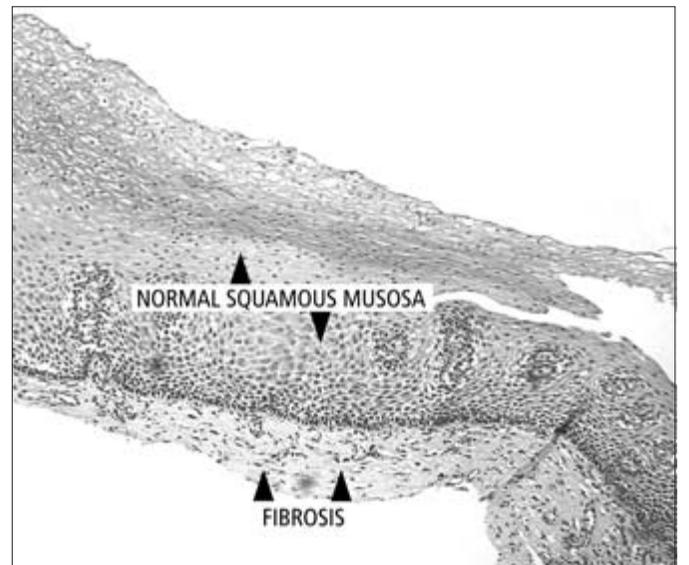
Oesophageal involvement in progressive systemic sclerosis (scleroderma) leads to a dilated atonic appearance (Fig. 2.104) simulating achalasia (Fig. 2.99) but without the spastic oesophago-gastric junction. The histopathological changes are shown in Fig. 2.105.



2.104

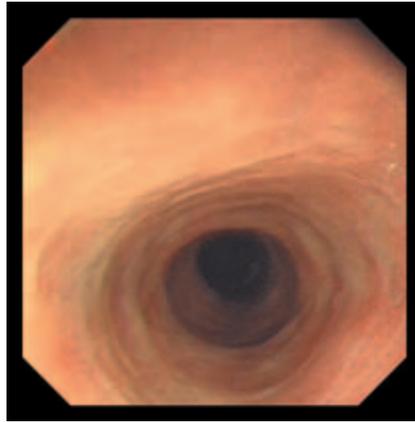


2.105



**Mixed connective tissue disease**

Figure 2.106 shows the endoscopic appearances. These are similar to those seen in progressive systemic sclerosis (Fig. 2.104) which is perhaps to be expected as the disorders have overlapping features. Figure 2.107 demonstrates the histological appearances.



2.106

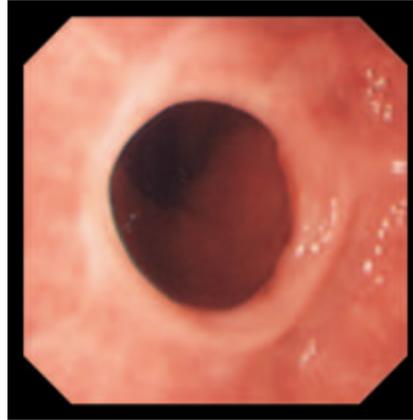


2.107

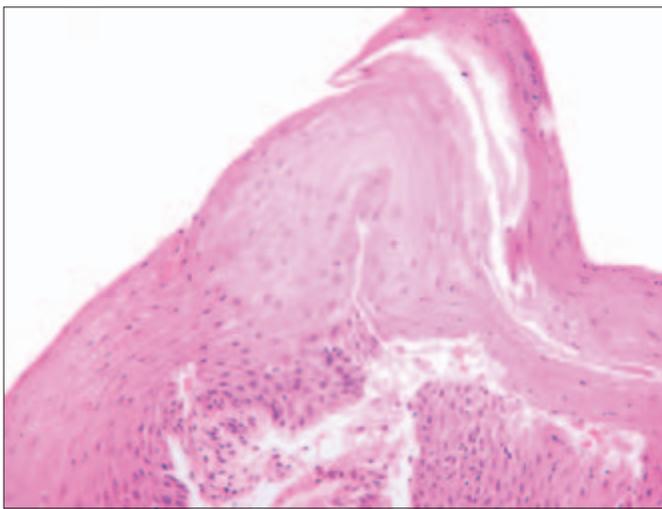
## Rings and webs

### Rings and webs

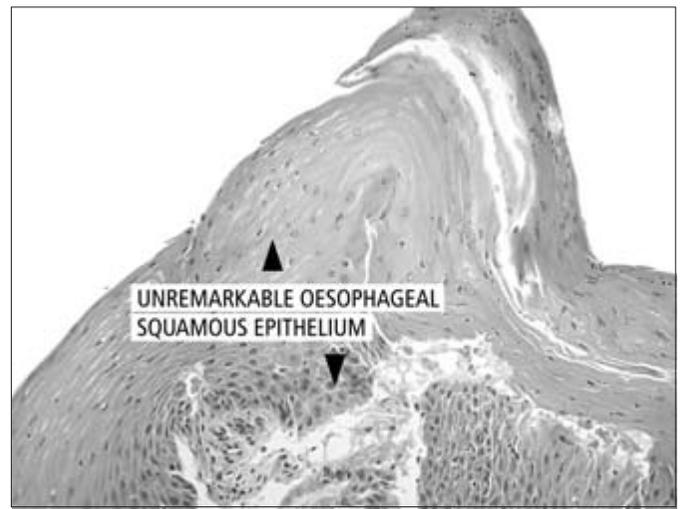
It is not always possible endoscopically to diagnose the nature of a ring or web. For example, the lesion shown in Fig. 2.108 resembles a benign stricture with scarring; this however, is unlikely as it was wide-mouthed and could easily be passed by the endoscope. Figure 2.109 shows the histological appearances of an oesophageal web.



2.108



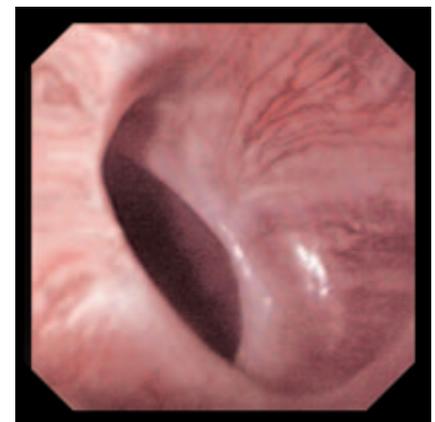
2.109



In this photograph (Fig. 2.110) the squamo-columnar junction was web-like, sufficient to give occasional dysphagia. The appearances are not dissimilar to those seen in patients with a Schatzki ring, except that in the latter there is usually an hiatal hernia below the ring, which may be tough and fibrous (Fig. 2.111).



2.110



2.111

### Rings and webs (cont.)

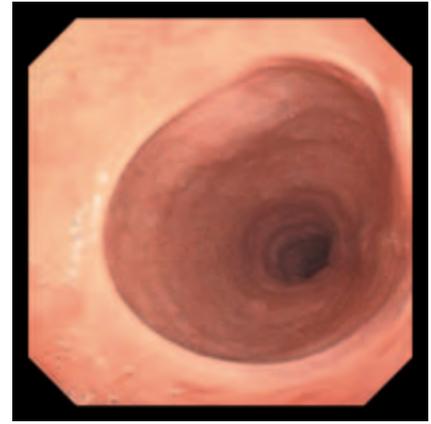
Figure 2.112 represents another view of a Schatzki ring.

Upper oesophageal webs are usually associated with the Patterson–Kelly (Plummer–Vinson) syndrome. Lower oesophageal webs of uncertain aetiology may be multiple, and may resemble rings. The lesion shown in Fig. 2.113 was seen at 20 cm and was softer and more pliable than the rings illustrated above.

Oesophageal webs may be difficult to demonstrate endoscopically, even when contrast studies have clearly shown them to be present.



2.112



2.113

## Hiatal hernia

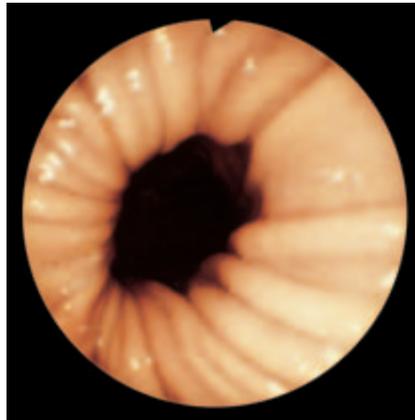
### Sliding hiatal hernia

#### Paraoesophageal (rolling) hiatal hernia

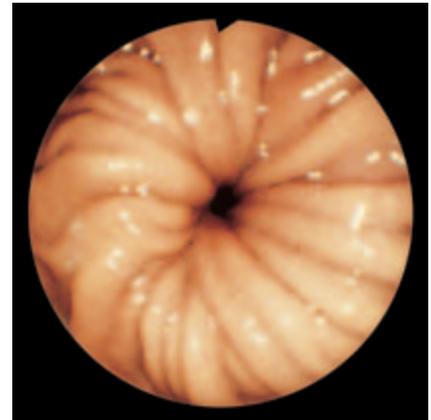
### Sliding hiatal hernia

In sliding hiatal hernia the herniated portion of stomach ascends into the thorax through a widened diaphragmatic hiatus in line with the oesophagus above. A sliding hiatal hernia is recognized as the endoscope passes from the tubular oesophagus, into a 'cavity' below the squamocolumnar junction but above the diaphragm. The gastric mucosa within the hernia is usually folded longitudinally, the folds running distally through the hiatal opening. With each breath this opening will increase (Fig. 2.114) and decrease (Fig. 2.115) in diameter and the rugae will 'roll' across it. Sometimes it can be difficult to negotiate the hiatus and the endoscope may curl in the hernia. Especial care is then required to avoid trauma and possible perforation.

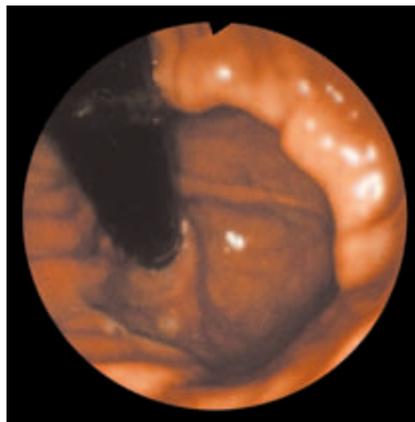
Employing inversion in the stomach, the J-manoeuvre, the mucosa can be seen 'rolling' across the hiatal circumference with each breath (Fig. 2.116). Further withdrawal of the endoscope reveals the squamocolumnar junction (Fig. 2.117). Under these circumstances there will be no flap valve.



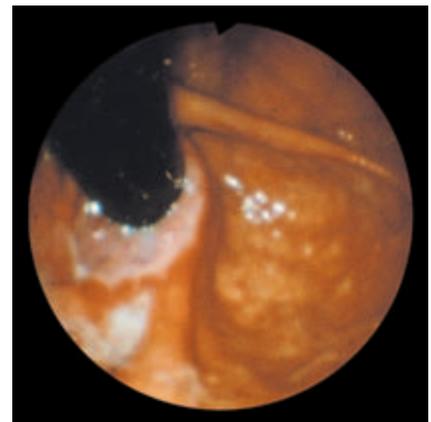
2.114



2.115



2.116

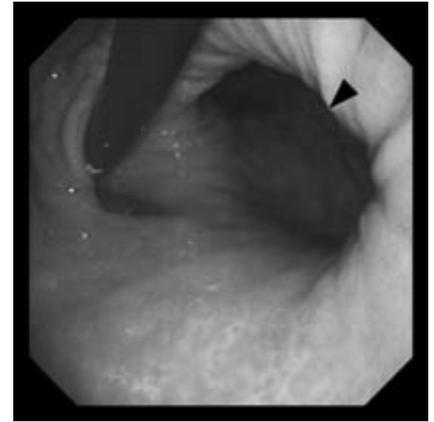
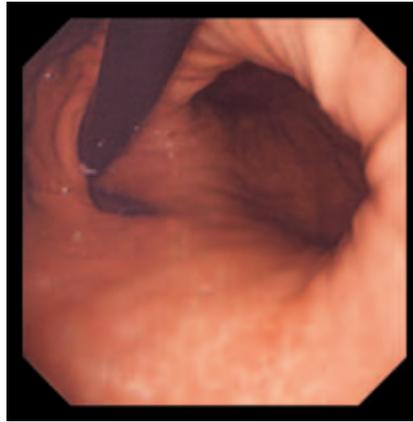


2.117

It is important to differentiate sliding hiatal hernia from Barrett's oesophagus (pp. 53–58).

**Paraoesophageal (rolling) hiatal hernia**

Paraoesophageal or rolling hiatal hernia is less common than sliding hiatal hernia. With the endoscope in the J-position in the stomach the apex or fundus of the hernia is seen to the right of the endoscope (Fig. 2.118). Paraoesophageal hernia may become incarcerated and even strangulated; also, it predisposes to gastric volvulus (Fig. 2.242).



2.118

**Gastro-oesophageal reflux disease**

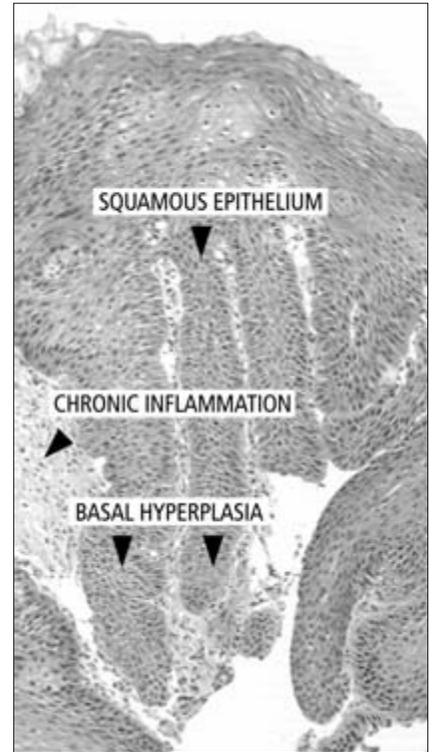
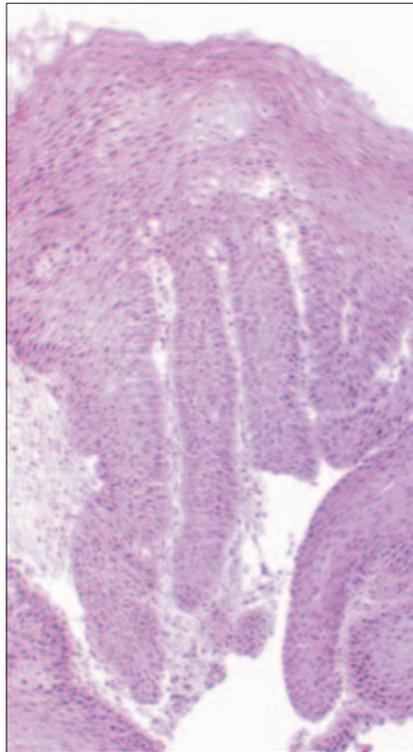
**Reflux oesophagitis**

**Ulcers in lower oesophageal and hiatal hernia**

**Benign oesophageal stricture**

**Reflux oesophagitis**

The gold standard for the assessment of gastro-oesophageal reflux is pH measurement. Endoscopy can demonstrate oesophagitis, but this correlates poorly with the results of pH measurements and symptoms. Histological appearances which have been associated with gastro-oesophageal reflux are basal hyperplasia, vascular prominence and chronic inflammation (Fig. 2.119). As with endoscopic appearances, correlation between histological appearances, pH measurements and symptoms is poor.



2.119

**Reflux oesophagitis (cont.)**

The classification of oesophagitis is contentious. The Savary–Miller scheme is widely used (Table 2.1) but other classifications have been suggested.

Oesophagitis occurs at and proximal to the squamocolumnar junction. In its mildest visible form there are variable red streaks running along the crests of the lower oesophageal folds, and in places these may be superficially ulcerated (Figs 2.120–2.122).

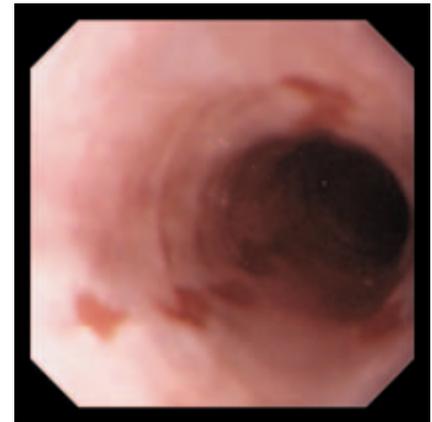
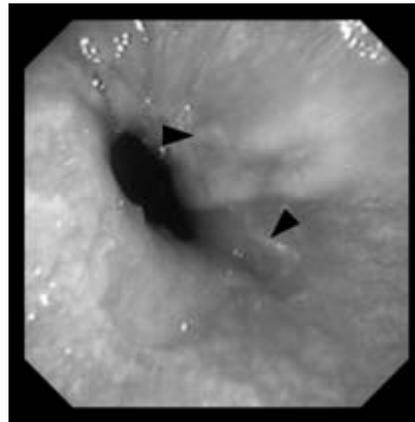
In moderate oesophagitis (Figs 2.123 and 2.124) there are tongue-like or flame-like areas of shallow ulceration overlain by yellow slough, surrounded by an erythematous zone. The pattern may be irregular. Merely touching these ulcers with the endoscope causes oozing of blood.

**Table 2.1** Classification of appearances of distal oesophagitis in gastro-oesophageal reflux disease (adapted from Savary and Miller, 1978)

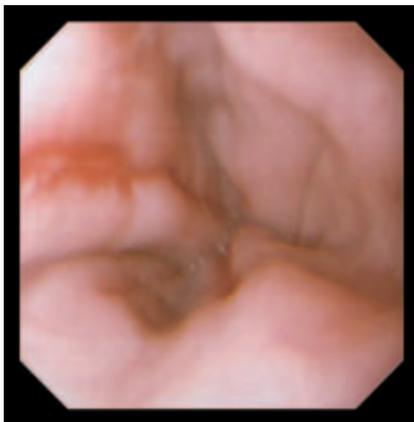
<i>Grade I</i>	One or more non-confluent erythematous spots or superficial erosions
<i>Grade II</i>	Confluent erosive or exudative mucosal lesions which do not extend around the entire oesophageal circumference
<i>Grade III</i>	Erosive or exudative mucosal lesions which cover the entire oesophageal circumference and lead to inflammation of the wall without stricture
<i>Grade IV</i>	Chronic mucosal lesions including oesophageal ulcer, mucosal scarring, mural fibrosis, stricture and Barrett's oesophagus



2.120



2.121



2.122



2.123

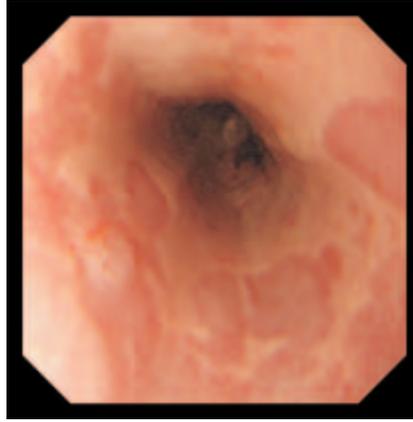


2.124

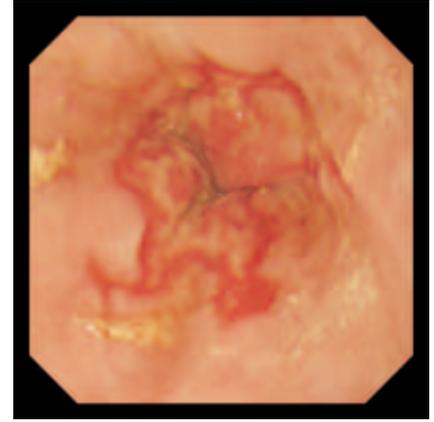
**Reflux oesophagitis (cont.)**

Severe oesophagitis (Figs 2.125 and 2.126) is characterized by deep irregular ulceration and oedema of the surrounding mucosa. The ulcers may become confluent involving in effect the whole of the lower oesophagus as in Fig. 2.127 where a strip of exudate has been lifted during the passage of the endoscope.

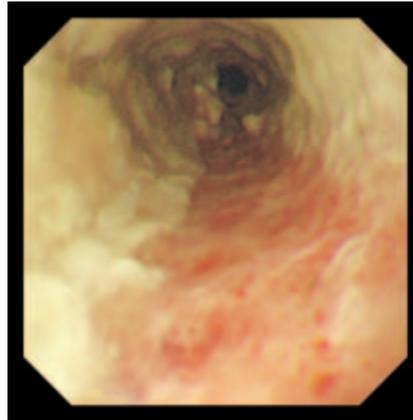
Rarely, severe oesophagitis may be complicated by the formation of multiple inflammatory polypoid lesions as shown in Fig. 2.128. These must be differentiated from carcinoma of the oesophagus which occurs most commonly in the lower third. Biopsy material from a carcinoma with such endoscopic appearances would certainly give a diagnostically positive result.



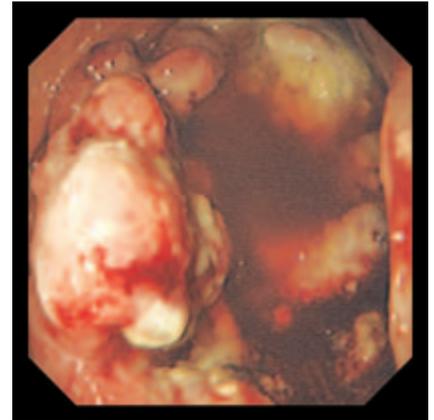
2.125



2.126



2.127

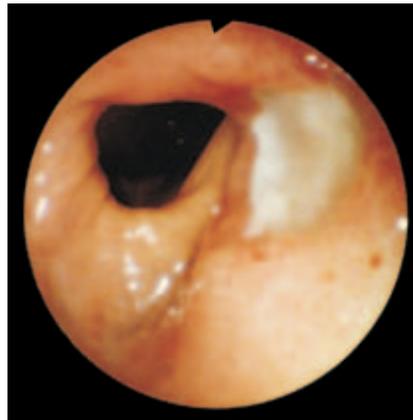


2.128

**Ulcers in lower oesophagus and hiatal hernia**

Oesophagitis as described above, with linear or patchy superficial ulceration, is more common than solitary oesophageal ulcers. The conditions are not usually found in the same patient. Discrete ulcers usually lie in the lower oesophagus and their shape and depth vary in similar fashion to gastric and duodenal ulcers. Figure 2.129 shows a rounded elongated ulcer.

A benign ulcer may occur in a hiatal hernia at the junction of oesophagus and hernia, or at its lower rim (riding ulcer). Such ulcers are similar to those found elsewhere in the stomach. They are often seen best during inversion using the J-manoeuvre (Figs 2.130 and 2.131).



2.129



2.130



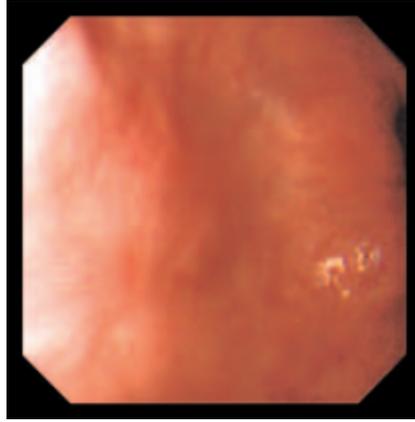
2.131

### Ulcers in lower oesophagus and hiatal hernia (cont.)

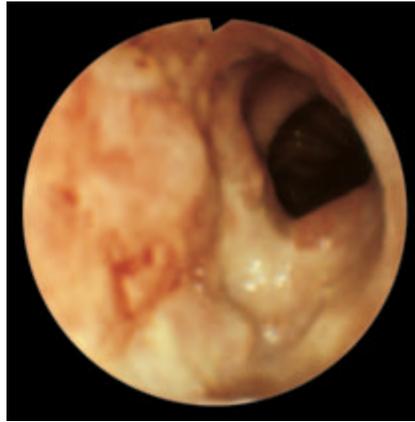
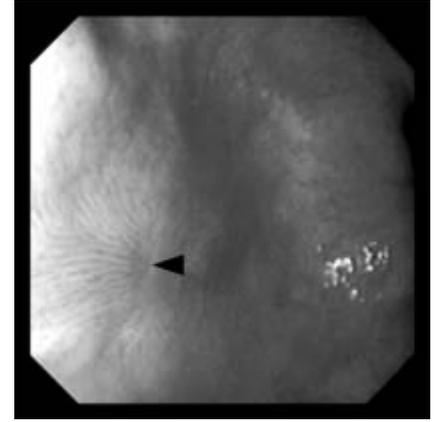
A healed oesophageal ulcer may leave a well-marked stellate scar (Fig. 2.132).

It may be difficult to draw the distinction between severe oesophagitis and multiple ulcers (Fig. 2.133). Indeed, this may be merely a matter of semantics.

Oesophageal ulcers, like strictures, cannot be deemed benign or malignant purely on the basis of visual endoscopic inspection. It is essential to obtain material for histopathological study.



2.132



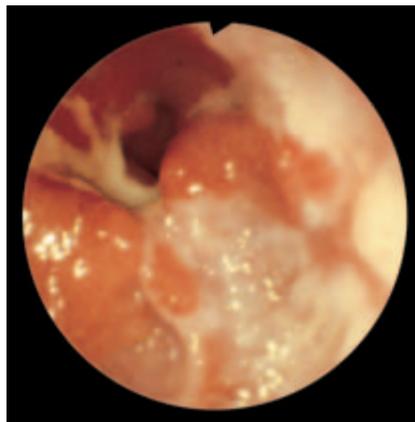
2.133

### Benign oesophageal stricture

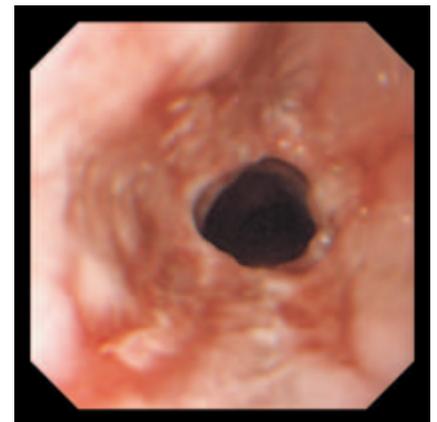
A benign oesophageal stricture is a tight fibrotic ring which does not vary on passage of a peristaltic wave.

Both endoscopy and radiology are used in the investigation of dysphagia, the commonest presentation of benign stricture. Although radiological diagnosis of carcinoma is usually correct, a false positive diagnosis is not rare and the possibility of carcinoma can never be excluded radiographically even in apparently smooth strictures. In significant dysphagia, endoscopy is indicated whether radiology is normal or not. Opinion is divided as to whether all patients with dysphagia should be investigated radiologically before endoscopy. Although helpful, prior radiological assessment should not be regarded as mandatory.

The commonest cause of benign oesophageal stricture is gastro-oesophageal reflux. Thus many strictures seen at endoscopy show associated oesophagitis. This may be very severe (Fig. 2.134), less severe (Fig. 2.135) or sharply localized to the area immediately surrounding the stricture (Fig. 2.136).



2.134

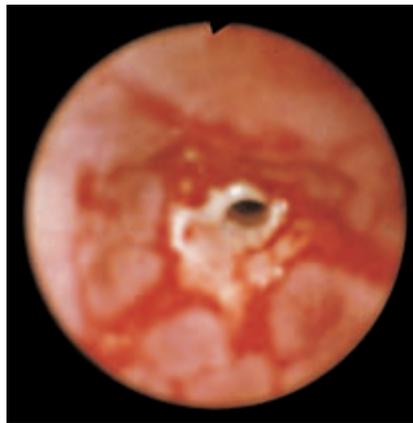


2.135

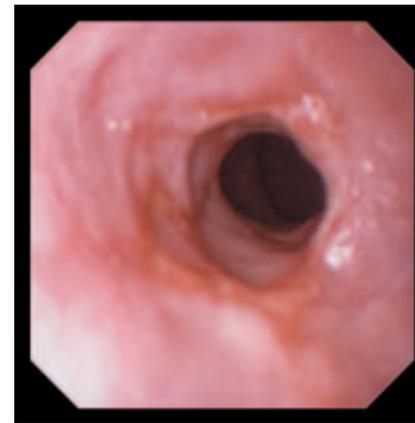
### Benign oesophageal stricture (cont.)

Oesophagitis may be minimal (Fig. 2.137) or there may be no obvious oesophagitis but varying degrees of scarring (Figs 2.97, 2.98 and 2.138). As there are no endoscopic features which will confidently exclude malignancy, it is essential always to obtain biopsy and cytological material. It is advisable to collect such specimens at diagnosis rather than after dilatation, when bleeding is likely to obscure good visualization and tissue trauma may make histological and cytological interpretation difficult. Sometimes, however, access to appropriate tissue for biopsy is only possible after dilatation.

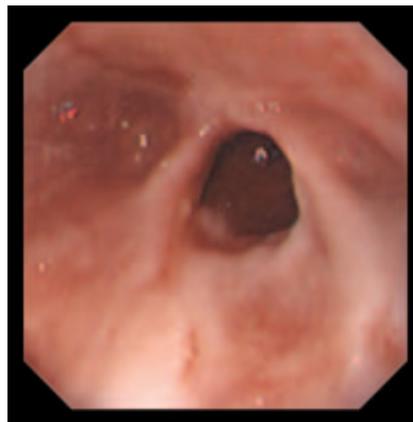
Figure 2.139 illustrates the use of a graduated flexible measuring device (Fig. 2.298) passed through the operating channel of the endoscope. It may be sufficient to estimate size by comparison with the spread of an opened pair of biopsy forceps.



2.136



2.137



2.138



2.139

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## Barrett's oesophagus

### Recognition and definition

#### Scarring and ulceration

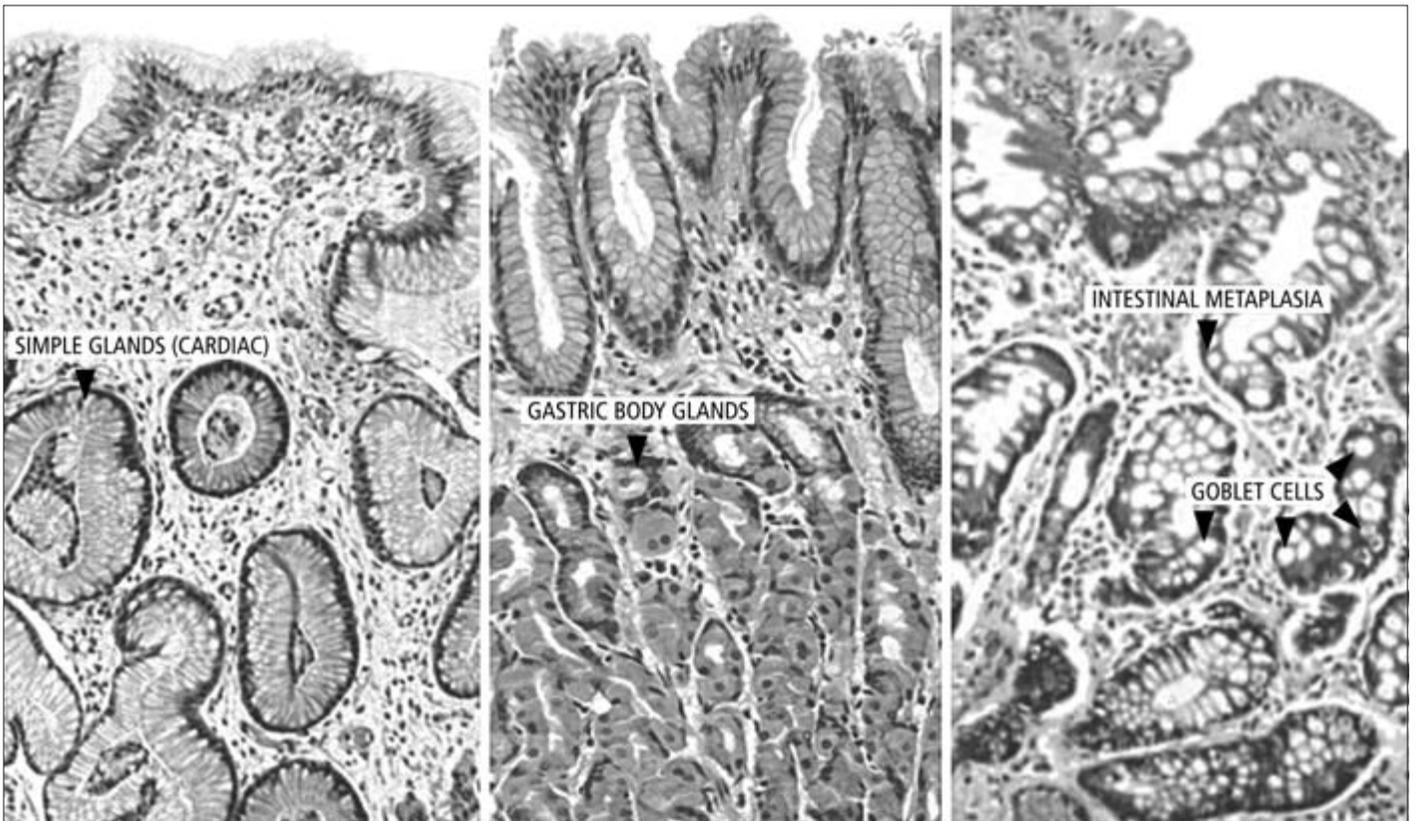
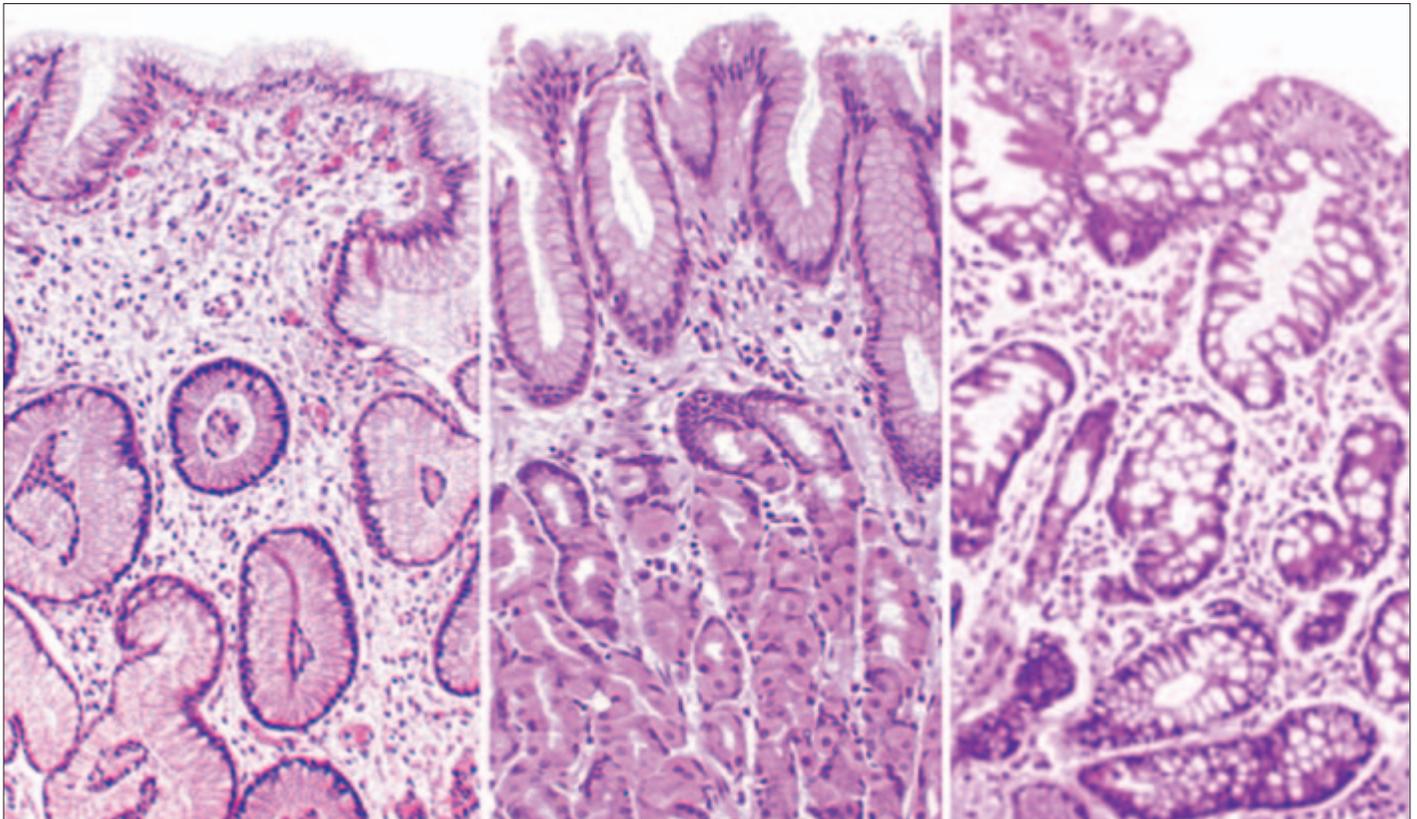
#### Dysplasia and carcinoma

### Recognition and definition

Although Barrett's oesophagus is considered to be a severe consequence of gastro-oesophageal reflux disease, it is currently the focus of special interest and will therefore be covered in some detail under a separate heading.

Barrett's oesophagus occurs when the tubular oesophagus is lined with columnar mucosa of either gastric or intestinal type rather than the usual squamous mucosa. Figure 2.140 illustrates the three types of mucosa seen in Barrett's oesophagus: simple glandular (so-called cardiac) mucosa, organoid gastric body type mucosa, and intestinal metaplasia. A workaday acceptable definition would be as follows: A columnar lined lower oesophagus of greater than 3 cm. Columnar lined oesophagus or CLO is an often preferred acceptable alternative description, having the advantage that it would include short segment Barrett's oesophagus, i.e. columnar lined oesophagus less than 2 cm. Ultrashort segment Barrett's oesophagus, that is, within the confines of the lower oesophageal sphincter, is a controversial concept, and is thought to equate with 'carditis', a *Helicobacter pylori*-related disease, as opposed to non-*H. pylori*-related reflux of acid and bile in Barrett's oesophagus both long and short segment.

Recognition and definition (cont.)



2.140

**Recognition and definition (cont.)**

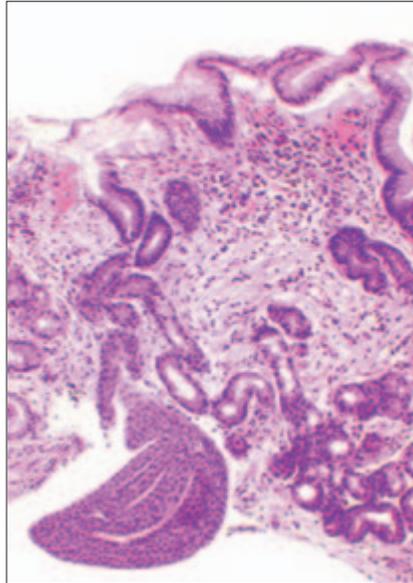
To the endoscopist, squamous epithelium appears whitish-pink while columnar epithelium has a redder hue, so that the transition is not usually difficult to recognize. There may however, be particular difficulty in assessing the length of lower oesophagus that is lined with columnar mucosa when there is a sliding hiatal hernia (p. 48). The extent of Barrett's oesophagus is usually recorded in centimetres from the squamocolumnar junction to the point at which the lumen changes from a tubular shape into a cavity.

In most cases Barrett's oesophagus is readily recognized and a single biopsy will show columnar mucosa which will confirm the endoscopic diagnosis. These circumstances will apply in five out of six cases of Barrett's oesophagus. In one of six cases the diagnosis may be made on histological evidence alone. This is when the pathologist sees glandular mucosa overlying pre-existing oesophageal glands or gland ducts within the biopsy. These structures are quite distinctive in their appearance (Fig. 2.141).

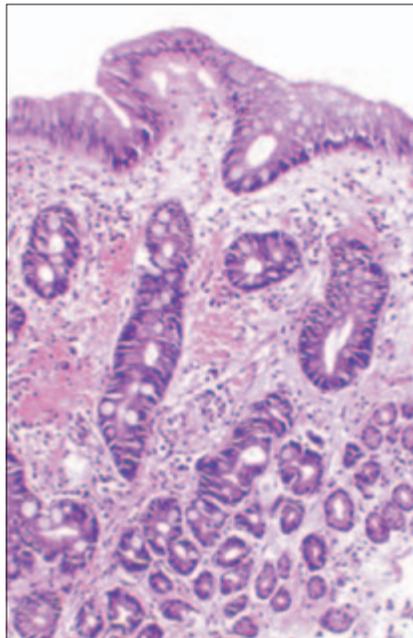
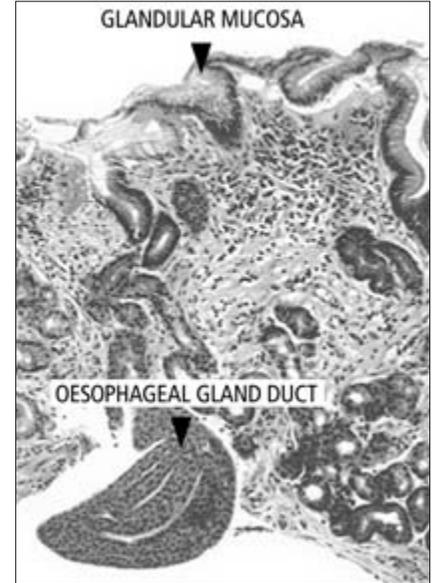
The reader may notice the lack of emphasis on intestinal metaplasia. Figure 2.142 shows intestinal metaplasia. It is our view, unlike that of most USA workers, that intestinal metaplasia, although an important precursor of dysplasia and adenocarcinoma, should not be relied on for the diagnosis of CLO. Intestinal metaplasia, although present in almost all cases, characteristically has a patchy distribution which means it may easily be missed on biopsy.

Intestinal metaplasia in Barrett's oesophagus cannot be recognized endoscopically.

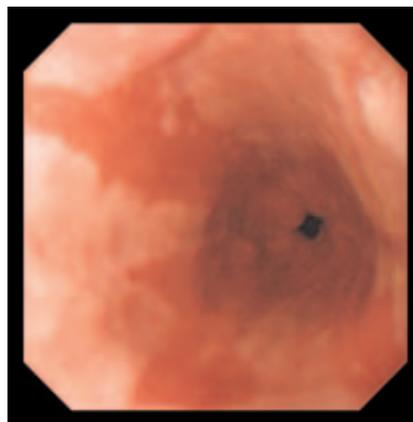
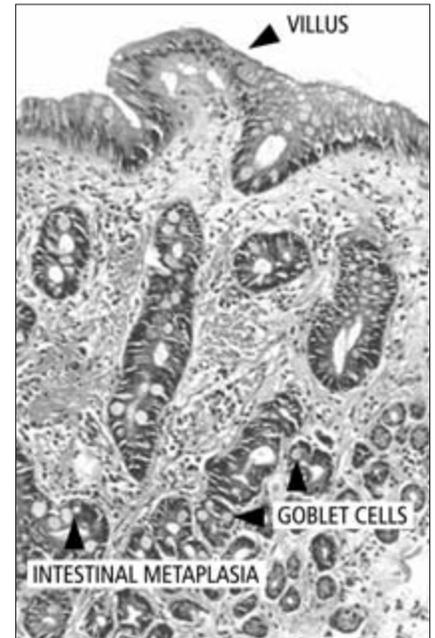
The squamocolumnar junction in Barrett's oesophagus is commonly irregular. Figure 2.143 shows such an irregular junction situated 10 cm above the hiatus; the lower portion of tubular oesophagus was lined entirely with fundal-type gastric mucosa.



2.141



2.142



2.143

**Scarring and ulceration**

There may be scarring in the gastric mucosa (Fig. 2.144) and there may be islands of white oesophageal mucosa (Fig. 2.145). Such islands (Fig. 2.146) are also seen after treatment with proton pump inhibitors or after laser treatment. Nodularity is not uncommon (Fig. 2.147) and such lesions should be biopsied.

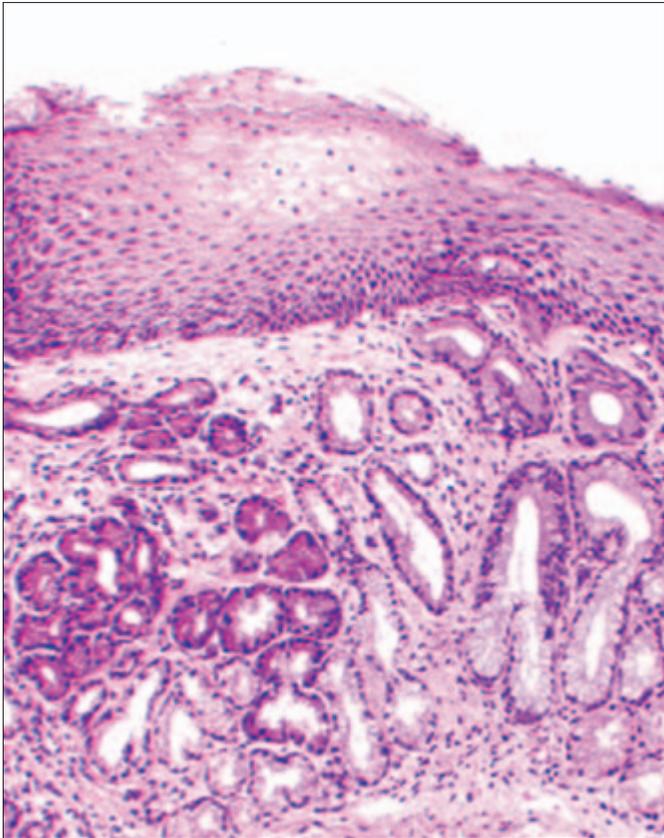
Figure 2.148 shows a discrete benign lower oesophageal ulcer arising in gastric-type mucosa, a so-called Barrett's ulcer. Figure 2.149 shows the stellate scar of a healed Barrett's ulcer.



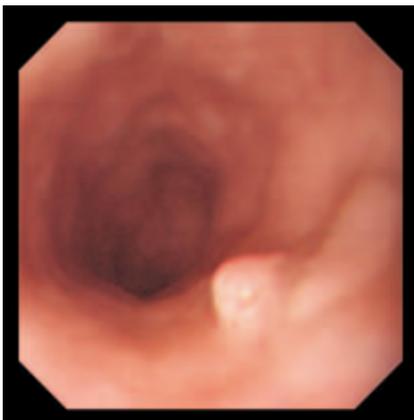
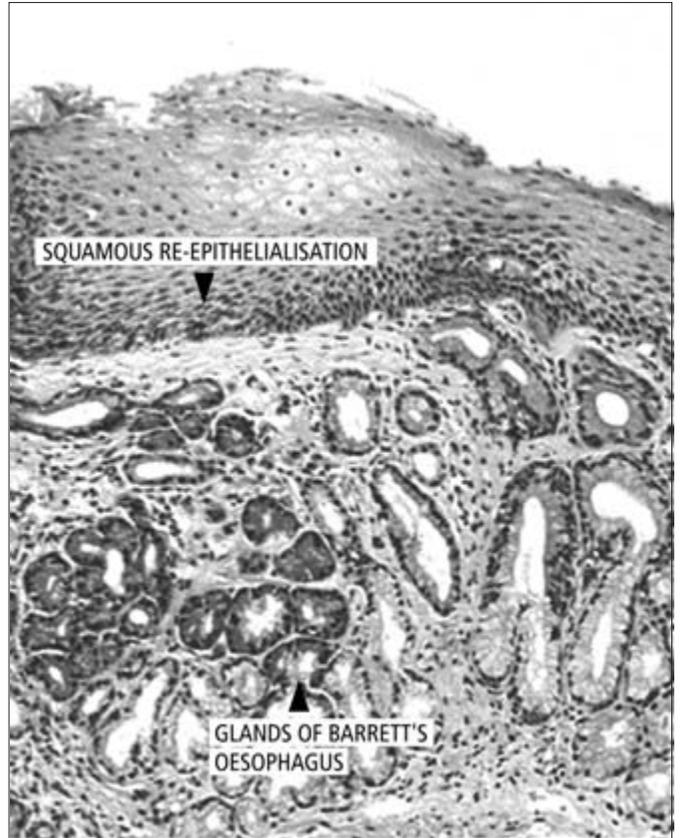
2.144



2.145



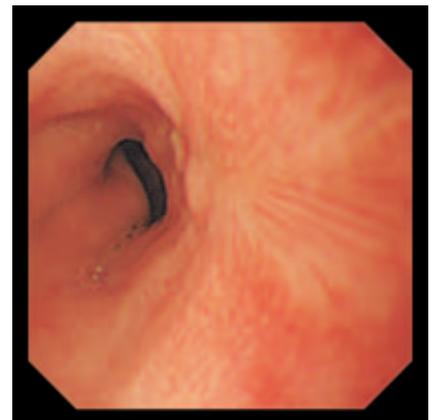
2.146



2.147



2.148



2.149

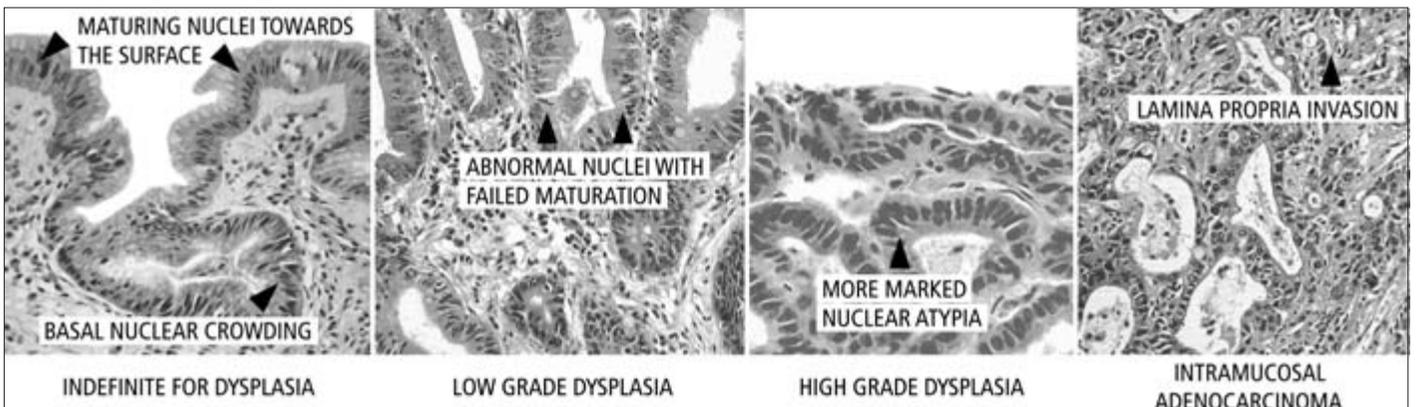
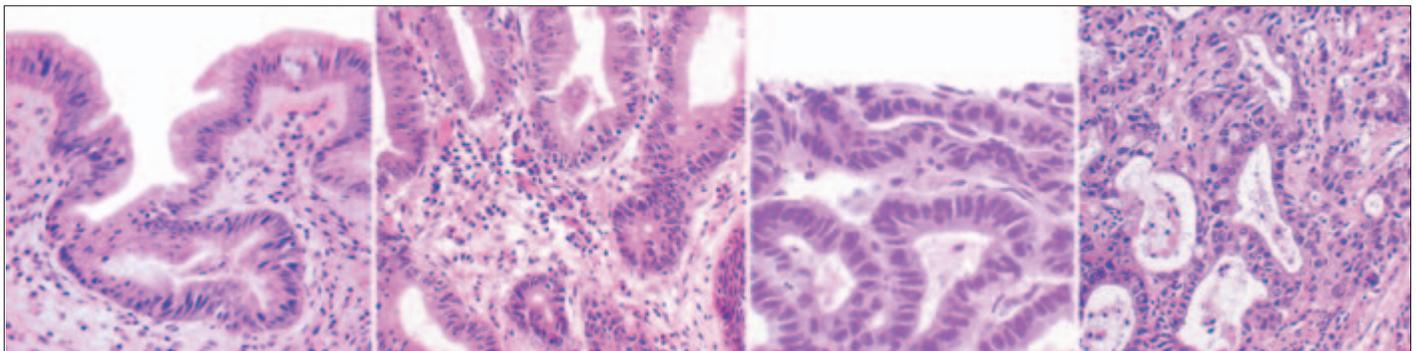
### Dysplasia and carcinoma

In patients with CLO there is an increased risk of dysplasia and subsequent carcinoma as dysplasia is the penultimate stage of carcinogenesis. Dysplasia is an histological diagnosis: dysplastic mucosa cannot be recognized endoscopically. Dysplasia may be a chance finding or may be found as a result of surveillance. To add to the clinical problem, it may occur in a patchy distribution. Lauwers and Riddell (1999) have proposed a new classification of gastric dysplasia and neoplasia, the Vienna classification (Table 2.8, p. 113). Further work may make it possible to employ this classification throughout the gastrointestinal tract.

In the search for dysplasia in patients with CLO the Seattle recommendations (Levine *et al.* 1992) may be followed: quadrant biopsies are taken every 2 cm throughout the length of the CLO and should then be assessed histologically (Fig. 2.150). It is recommended that, according to the severity of the changes found, action should be taken as outlined in Table 2.2. It should be borne in mind that up to 50% of patients with high grade dysplasia have an unsuspected adenocarcinoma.

**Table 2.2** Histological reporting of dysplasia in Barrett's oesophagus and action needed

Dysplasia	Action
Absent	Routine follow-up endoscopy
Indefinite	Urgent repeat endoscopy
Low grade	Full mapping biopsies and early repeat endoscopy
High grade	Repeat biopsies and review/confirmation by second pathologist; then consider for surgery and/or other appropriate treatment
Intramucosal carcinoma	Review/confirmation by second pathologist; then consider for surgery and/or other appropriate treatment

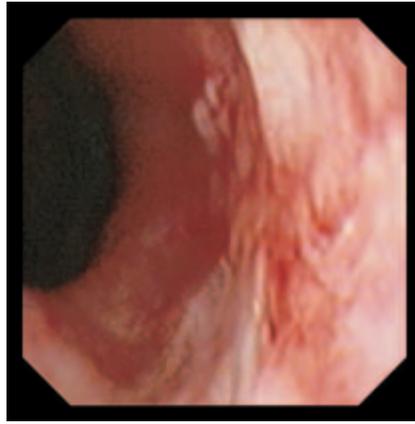


2.150

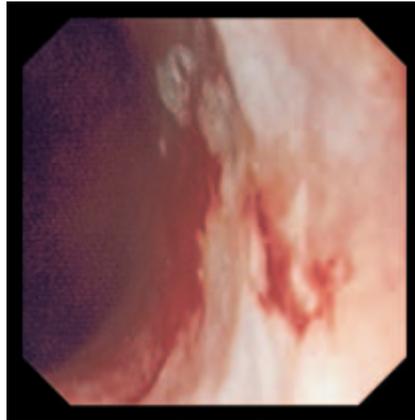
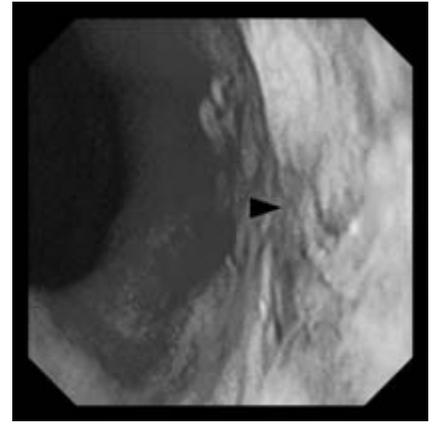
**Dysplasia and carcinoma (cont.)**

The following two images show such progressive changes. In Fig. 2.151 there is a suspicious area in a length of Barrett's oesophagus. Biopsies showed intestinal metaplasia but no dysplasia. Six months later repeat biopsies from the same suspect area revealed high-grade dysplasia and intramucosal carcinoma (Fig. 2.152).

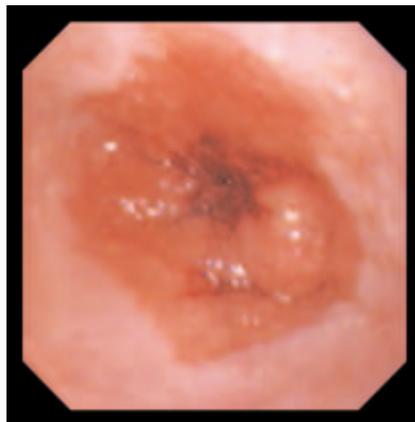
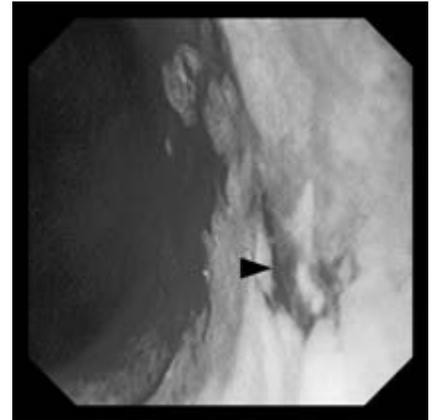
Figure 2.153 illustrates an exophytic carcinoma that was found complicating Barrett's oesophagus. A flatter ulcerated carcinoma appears in Fig. 2.154. Another patient known to have CLO with intestinal metaplasia presented with food impaction causing total dysphagia. After removal of the food bolus a circumferential carcinoma was identified (Fig. 2.155).



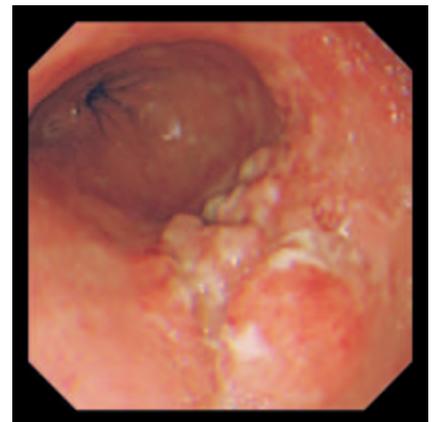
2.151



2.152



2.153



2.154



2.155

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## Benign and malignant tumours

### Classification

#### Nonspecific appearances

#### Squamous cell papilloma

#### Inflammatory polyp

#### Primary carcinoma

#### Direct spread of tumours from adjacent organs

#### Granular cell tumour

#### Lipoma

#### Kaposi's sarcoma

#### Mucosal vesicles

#### Sebaceous glands

### Classification

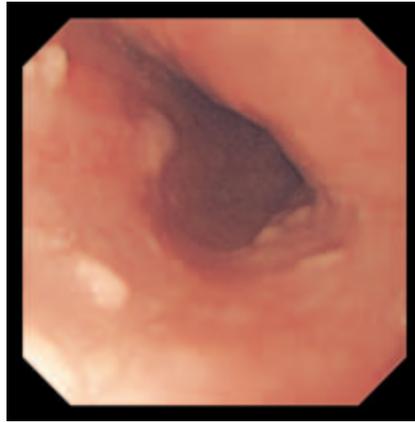
Small nonspecific looking polypoid lesions occur commonly in the oesophagus. Larger polyps, such as are found elsewhere in the gastrointestinal tract, are rare. A classification of oesophageal tumours appears in Table 2.3. In the selection of images there has been no attempt at comprehensiveness, but all the lesions here illustrated are listed and will be discussed in the order in which they appear in this table.

**Table 2.3** Benign and malignant tumours of the oesophagus (adapted from Day *et al.*, 1990)

<b>Benign epithelial tumours</b>
Squamous cell papilloma
Inflammatory polyps
Adenomas
Hyperplastic polyps
<b>Malignant epithelial tumours</b>
Early oesophageal carcinoma
Squamous carcinoma
Adenocarcinoma
Others
<b>Secondary and direct spread tumours</b>
Epithelial neoplasms
Leukaemias and lymphomas
Direct spread tumours
<b>Non-epithelial tumours</b>
Gastrointestinal stromal tumours (GISTs)
Granular cell tumours
Lipomas
Fibrous (fibrovascular) polyps
Kaposi's sarcoma
<b>Miscellaneous tumour-like lesions</b>
Mucosal vesicles
Sebaceous glands
Others

**Nonspecific appearances**

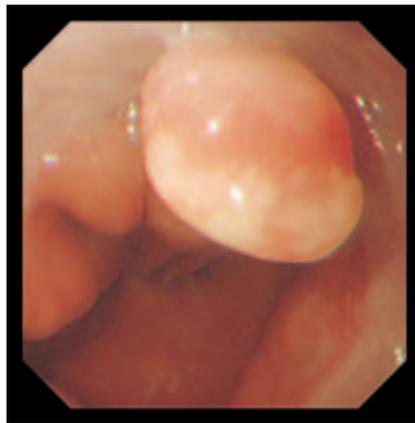
Visually, the nature of the lesions shown in Fig. 2.156 is uncertain but to the experienced endoscopist they appear benign. Such small ‘lumps and bumps’ occur on almost every endoscopy list. Judgement will develop with growing experience.



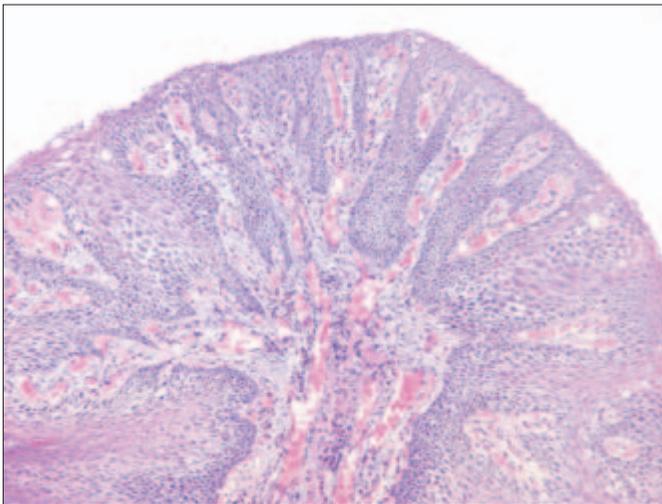
2.156

**Squamous cell papilloma**

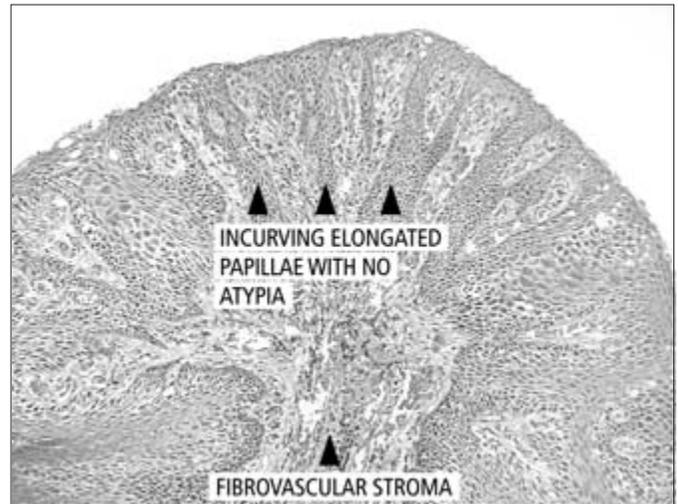
This uncommon lesion (Fig. 2.157) may undergo malignant change. In this instance it is smooth with a regular outline. Any lesion with such an appearance should always be extensively biopsied (Fig. 2.158), particularly if large, and the diagnosis of a verrucous (well differentiated) squamous cell carcinoma should be considered.



2.157

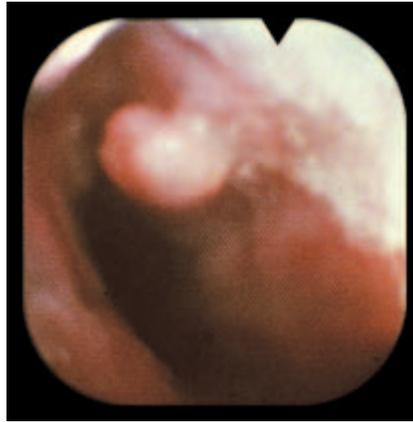


2.158



**Inflammatory polyp**

The polyp shown in Fig. 2.159 has nonspecific appearances and the diagnosis depends on what is shown on biopsy.



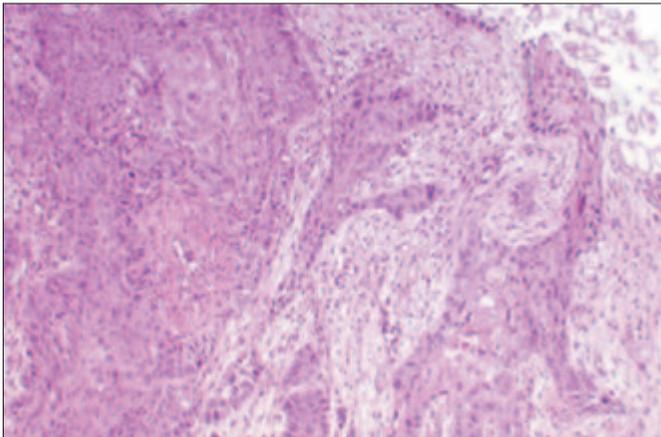
2.159

**Primary carcinoma**

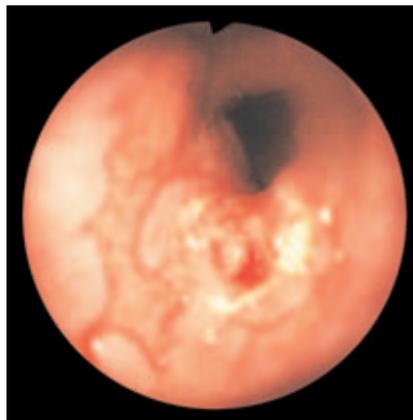
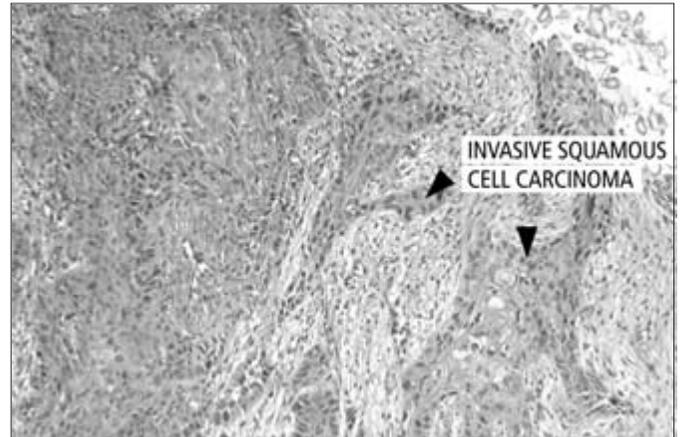
While squamous cell carcinoma (Figs 2.160 and 2.161) is the commonest type involving the upper and middle thirds of the oesophagus, a malignant lesion found in the lower third is most likely to be an adenocarcinoma (Figs 2.162a and 2.162b).



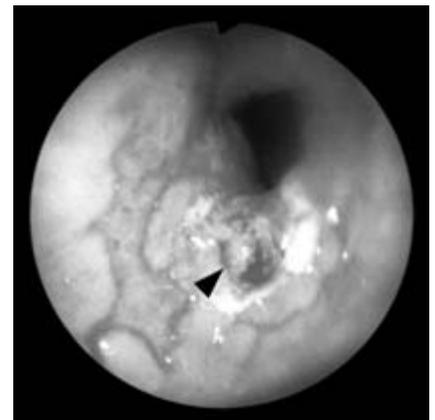
2.160

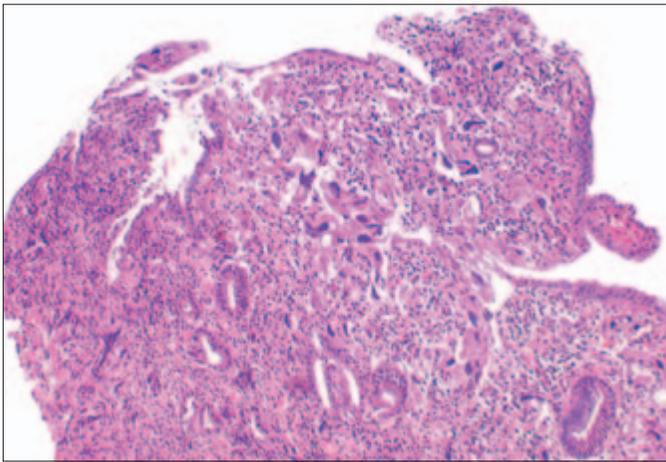


2.161

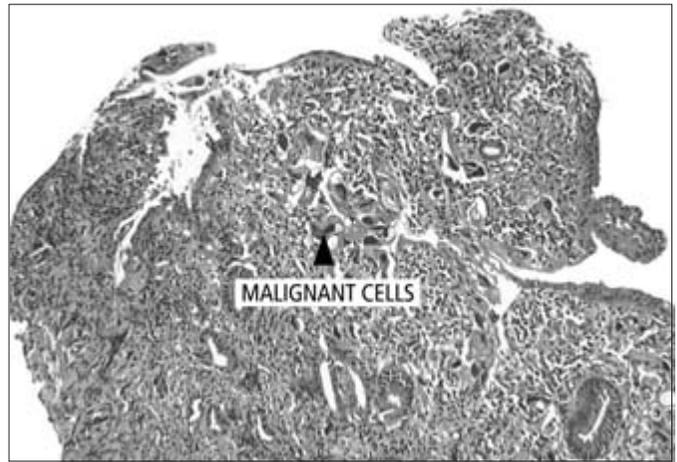


2.162a





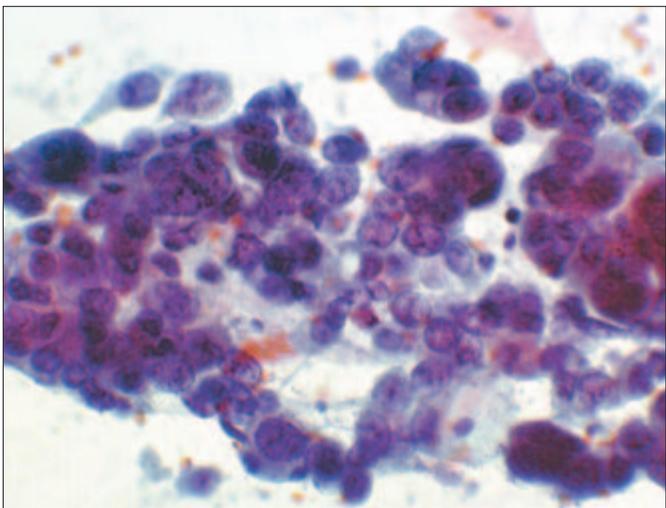
2.162b



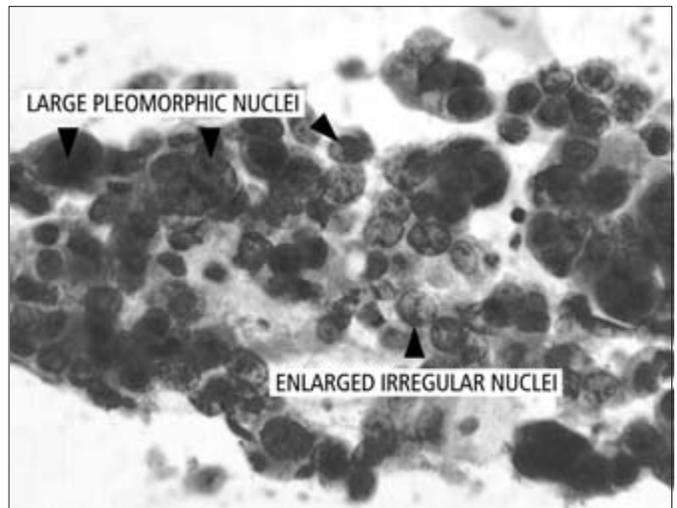
**Primary carcinoma (cont.)**

Endoscopically these types are indistinguishable and therefore brush cytology and forceps biopsies are essential. Specimens are usually easy to obtain. A repeat diagnostic endoscopy can be avoided if a sufficient number of biopsy and cytology specimens is obtained. Figure 2.163 shows the appearances of a brush cytology specimen obtained from an adenocarcinoma complicating Barrett's oesophagus.

The irregular relatively firm polypoid lesion illustrated in Fig. 2.164 is an adenocarcinoma. By contrast Fig. 2.165 shows a large friable obstructing growth.



2.163



2.164



2.165

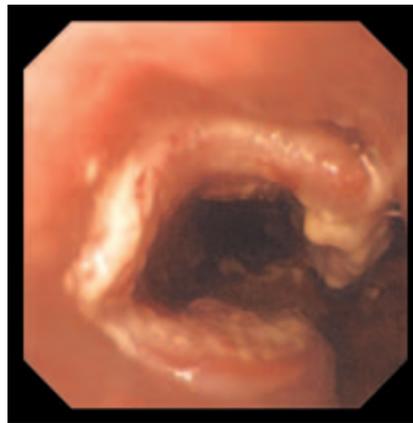
**Primary carcinoma (cont.)**

The encircling lesion shown in Fig. 2.166 caused mild painless dysphagia, while the nonencircling carcinoma appearing in Fig. 2.167 was a complication of achalasia, revealed at routine surveillance.

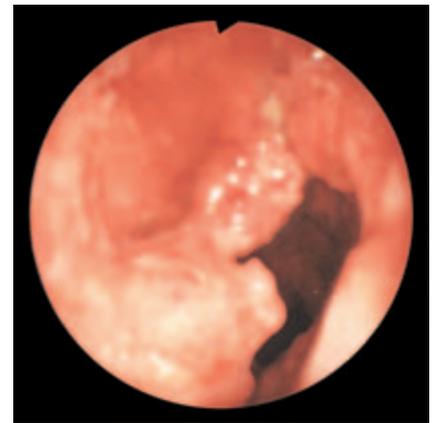
The lower oesophageal mass shown in Fig. 2.168 caused severe painless dysphagia. Note the diffusely ulcerated appearance. The lesion shown in Fig. 2.169 is a squamous cell carcinoma of the lower oesophagus. This large exophytic growth was friable, superficially ulcerated and bled easily on contact.

An adenocarcinoma involving the lower oesophagus at the oesophago-gastric junction may have arisen in the oesophagus or in the cardia. Figure 2.170 shows the rolled edge and ulcerated surface of such a lesion from the oesophageal aspect; from below it is seen to surround the junction (Fig. 2.171).

Carcinoma of the oesophagus commonly spreads submucosally and islands of malignant tissue (Fig. 2.172) may then be seen where the process has breached the mucosal surface.



2.166



2.167



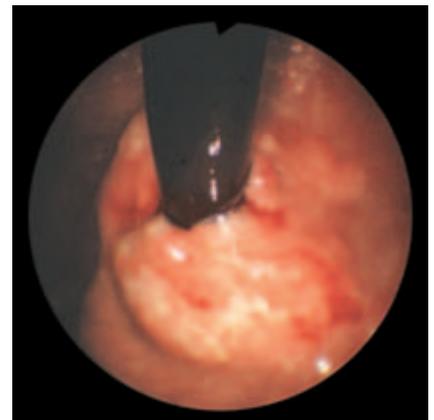
2.168



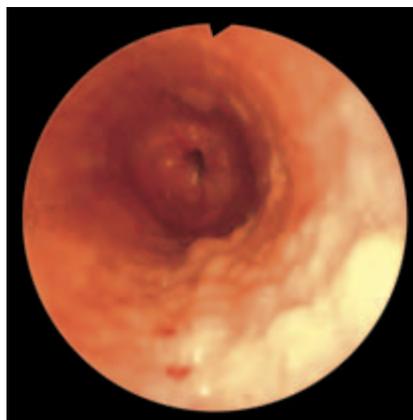
2.169



2.170



2.171



2.172

**Direct spread of tumours from adjacent organs**

Figures 2.173 and 2.174 illustrate direct invasion of the oesophagus by bronchial carcinoma. The lesion shown in Fig. 2.173 has not breached the mucosa, while there is superficial ulceration in Fig. 2.174. Not surprisingly biopsies in the first case were unhelpful, while in the second case they were diagnostic. Florid invasion of the oesophagus by a bronchial carcinoma appears in Fig. 2.175: this patient presented with severe dysphagia. Typical histopathological appearances of a bronchial small cell carcinoma are shown in Fig. 2.176.



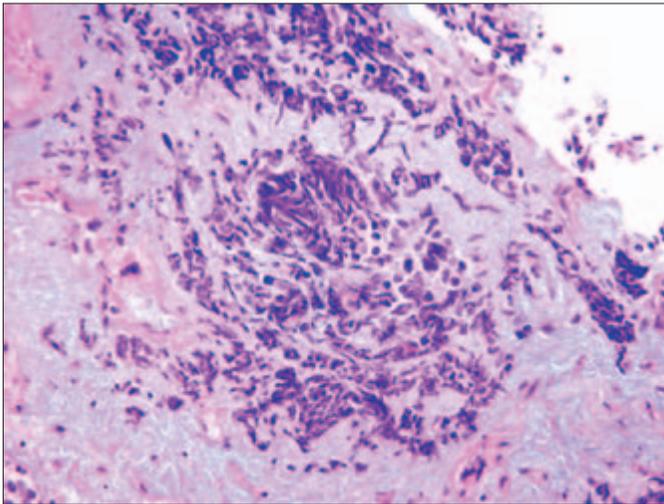
2.173



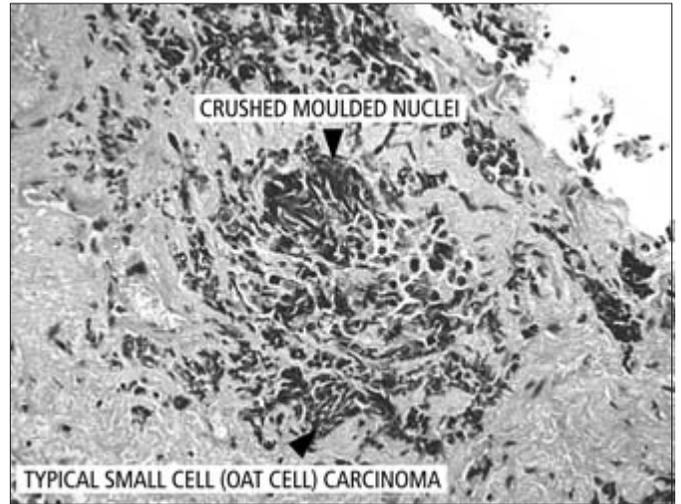
2.174



2.175



2.176



### Granular cell tumour

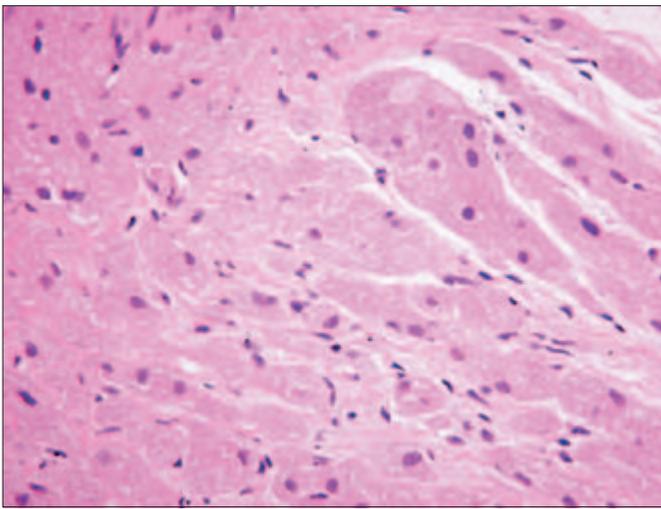
These usually small white raised lesions are benign and of no clinical importance other than in differential diagnosis. Despite the name the origin is probably a nerve sheath cell. Typical examples of multiple lesions are shown in Fig. 2.177. It is a matter of individual judgement whether all such small abnormalities should be biopsied, though a successful target biopsy will reveal their nature (Fig. 2.178). Figure 2.179 shows an unusually large granular cell tumour; with a lesion of this size biopsy is essential.



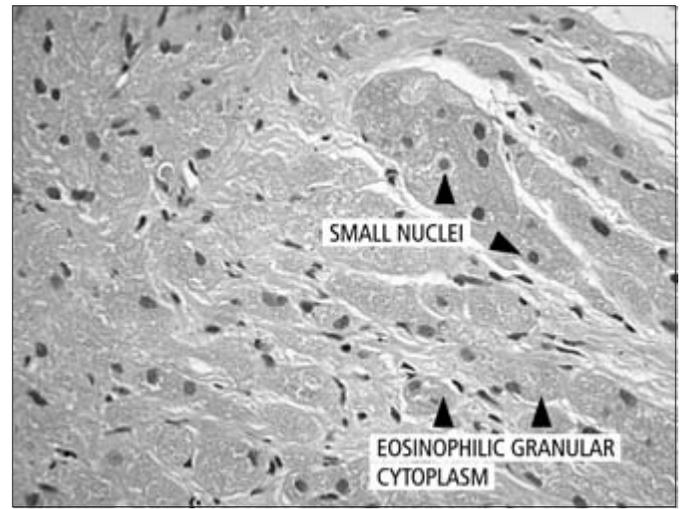
2.177



2.179

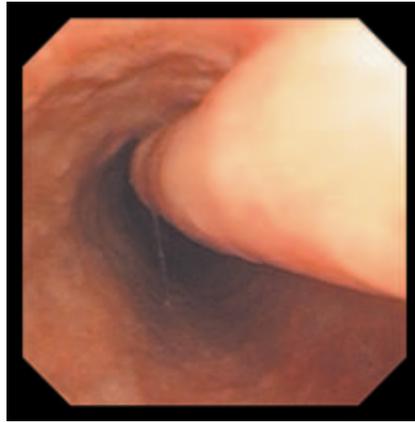


2.178



**Lipoma**

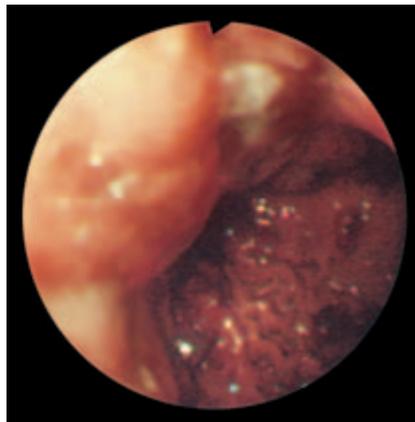
Such lesions are uncommon in the oesophagus and it is particularly unusual to find such a very large pedunculated exemplar (Fig. 2.180).



2.180

**Kaposi's sarcoma**

Although still uncommon, Kaposi's sarcoma which endoscopically mimics carcinoma is seen increasingly because of its association with AIDS (Fig. 2.181).



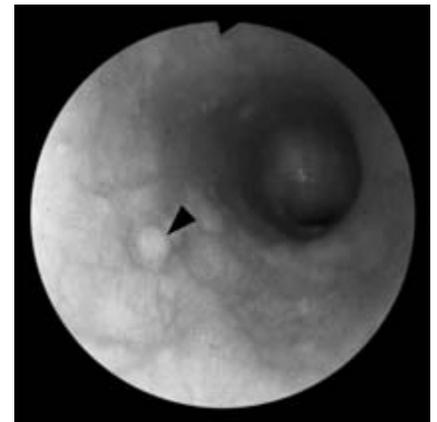
2.181

**Mucosal vesicles**

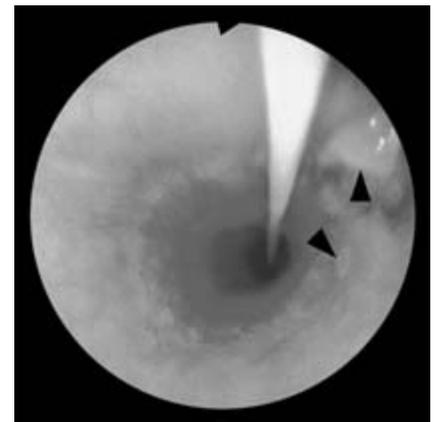
An isolated vesicle, at first glance suggestive of a tumour, may be a chance finding (Fig. 2.182) when further investigation may be thought inappropriate. The vesicular lesion in Fig. 2.183 was due to *Herpes simplex* in a patient with AIDS (see also Fig. 2.232).



2.182



2.183

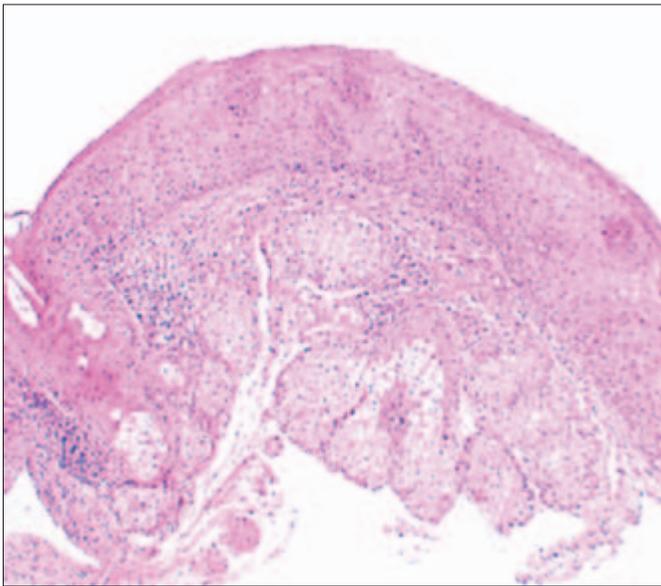


### Sebaceous glands

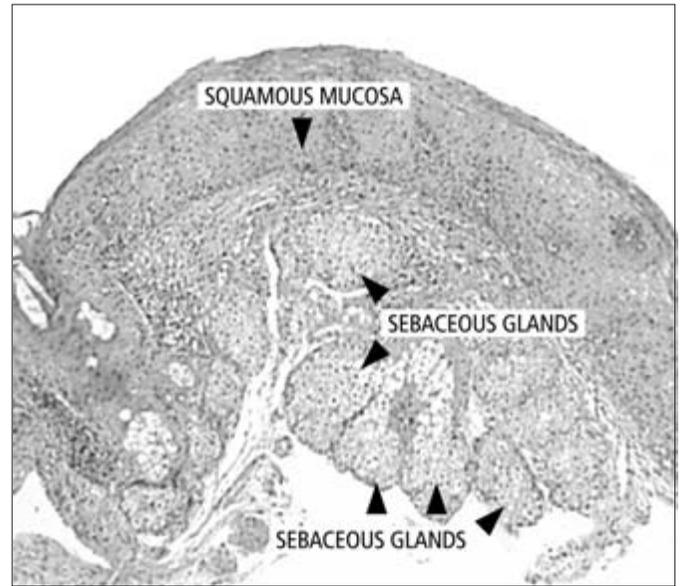
These are found within the oesophagus as a congenital anomaly in up to 2% of the population. An endoscopist can therefore be forgiven for not recognizing this very unusual condition (Fig. 2.184), when he will rely on the pathologist for the correct diagnosis (Fig. 2.185).



2.184



2.185



## Vascular abnormalities

### Varices

#### Minor vascular abnormalities

#### Varices

Recognition of varices if not bleeding seldom presents a problem, although occasionally difficulty may arise should they resemble normal oesophageal folds in colour and shape. They run upwards from the oesophago-gastric junction, rarely the full length of the oesophagus, and usually not higher than the aortic arch. Unusually, they arise above the oesophago-gastric junction, in this patient at 30 cm from the incisors (Fig. 2.186). The surface of varices tends to have a blue tinge, and their outline is usually beaded. The greater the diameter, the prouder they stand into the oesophageal lumen, sometimes virtually filling it.

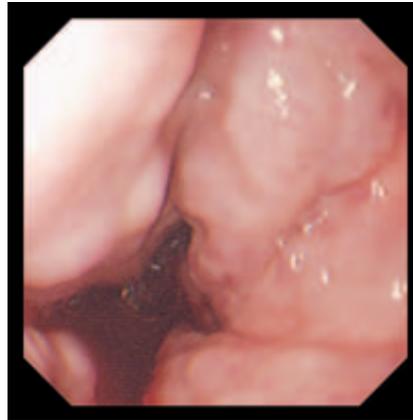
Rarely there is a single varix of small size (Fig. 2.187). More commonly, many varices are present which may be more or less beaded (Figs 2.188 and 2.189) and which may form a communicating pattern (Fig. 2.190). They may give the impression of being very thin-walled (Fig. 2.191).



2.186



2.187



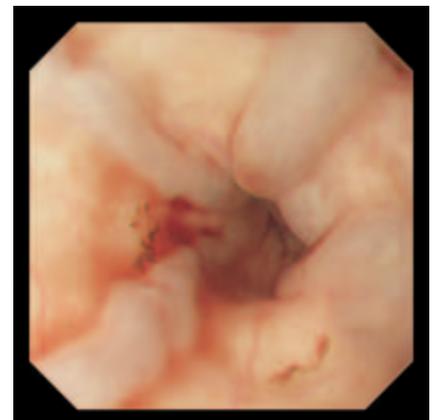
2.188



2.189



2.190



2.191

**Varices (cont.)**

Smaller veins may run over the surface of the varix or may occur above, below or between varices (Fig. 2.192). Cherry-red spots on the surface of the varices (Fig. 2.193) may indicate the site of recent haemorrhage and are associated with a high risk of subsequent bleeding.

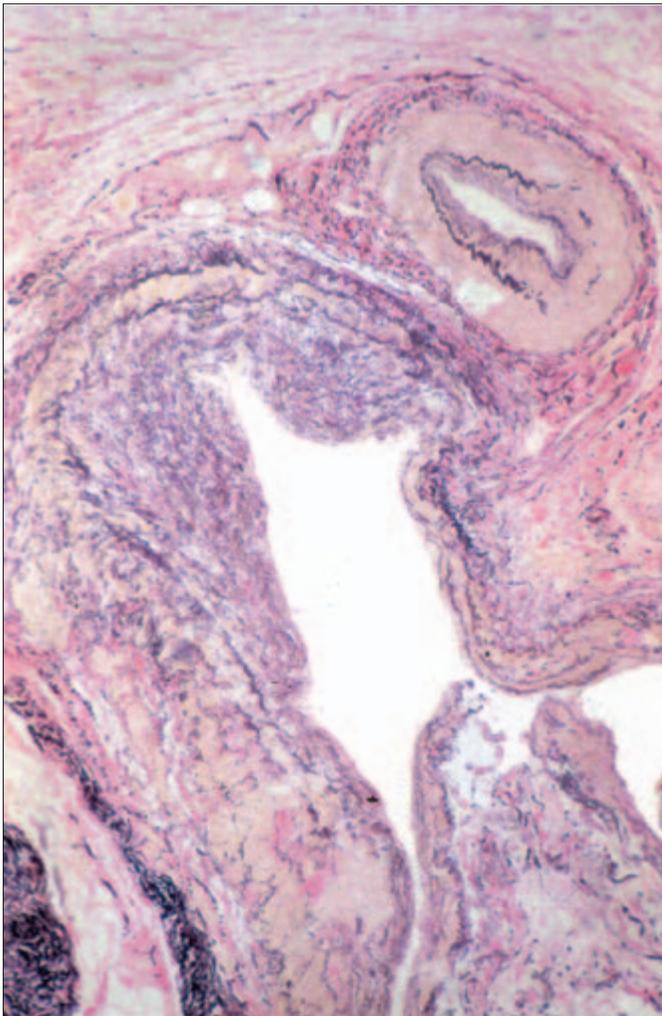
It is perhaps surprising that varices do not bleed more readily when it is appreciated how thin a variceal wall can be (see site of rupture in Fig. 2.194). Bleeding from varices is illustrated in Figs 2.665 and 2.666 and their management is discussed on pp. 208–213.



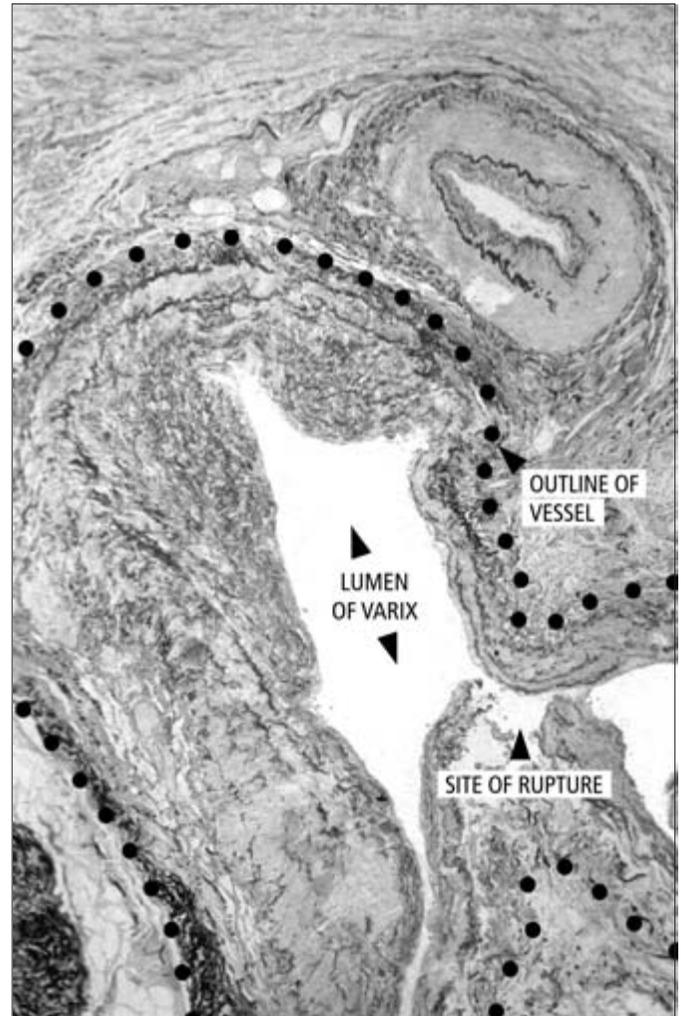
2.192



2.193

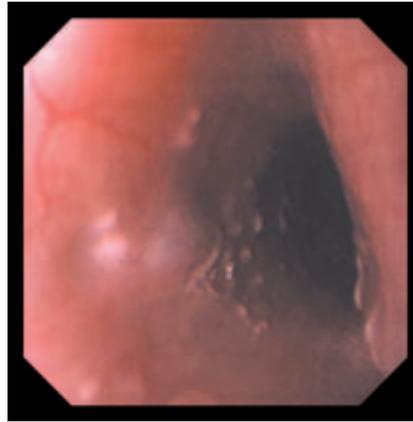


2.194

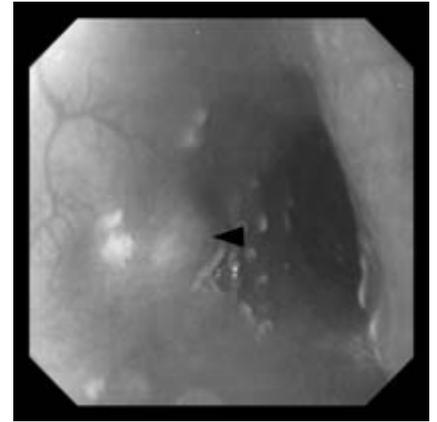


### Minor vascular abnormalities

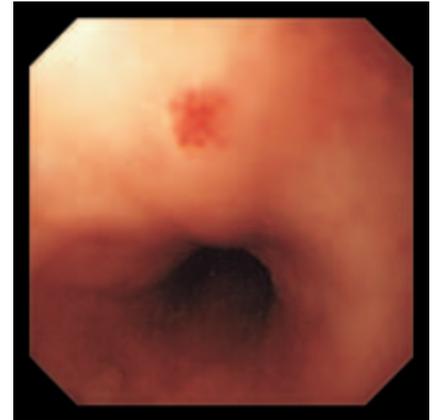
Minor vascular abnormalities may occur. Figure 2.195 illustrates a raised round bluish lesion, probably a cavernous haemangioma. It is not uncommon to see a blue submucosal patch in the oesophagus (Fig. 2.196) which may represent another variety of angioma. Figure 2.197 shows a haemangioma such as occurs in the Osler–Weber–Rendu syndrome; lesions of this type are commoner in the stomach (Figs 2.421 and 2.422) and small bowel (Fig. 2.569).



2.195



2.196



2.197

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### Miscellaneous conditions

#### Candidiasis

#### Appearances after barium radiography

Inlet patch in oesophagus (ectopic gastric mucosa)

Foreign bodies

Partial thickness tears

Acute necrotizing oesophagitis

Oesophagitis due to drugs and chemicals

Oesophagitis dissecans

Oesophageal hyperkeratosis

Crohn's disease

Tuberculosis

Herpes simplex

Fistulae

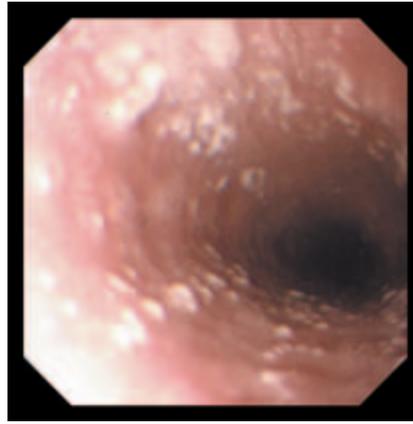
Xanthelasma

Glycogenosis

### Candidiasis

Candidiasis is often asymptomatic, and may be a chance finding of no clinical significance. It can however, be the cause of odynophagia or dysphagia. It is seen commonly in the elderly and in patients with immunoparesis or following antibiotic therapy.

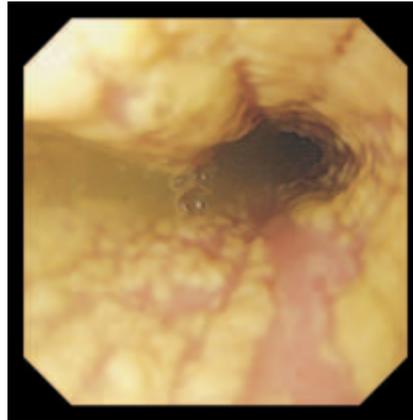
The appearance may be of small white spots (Fig. 2.198), white lines (Fig. 2.199), irregular yellow lines (Fig. 2.200) or confluent white (Fig. 2.201) or discoloured material (Fig. 2.202). If a lesion is lifted or washed off, the mucosa beneath is often ulcerated and may bleed (Fig. 2.203).



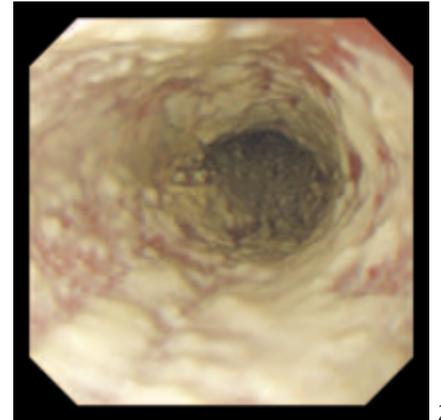
2.198



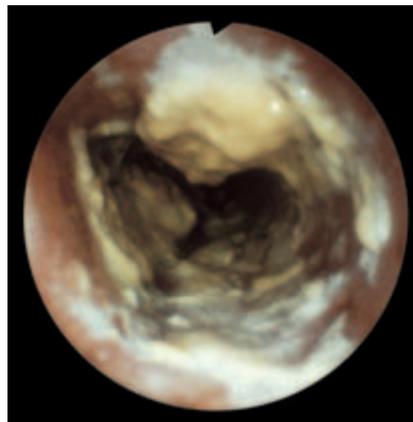
2.199



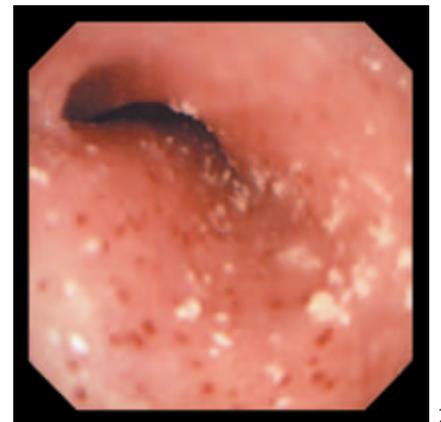
2.200



2.201



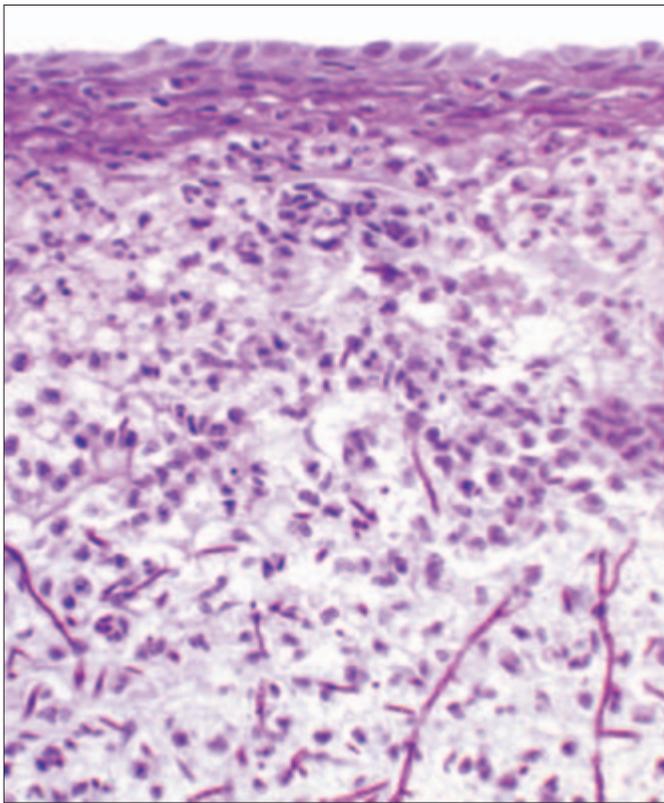
2.202



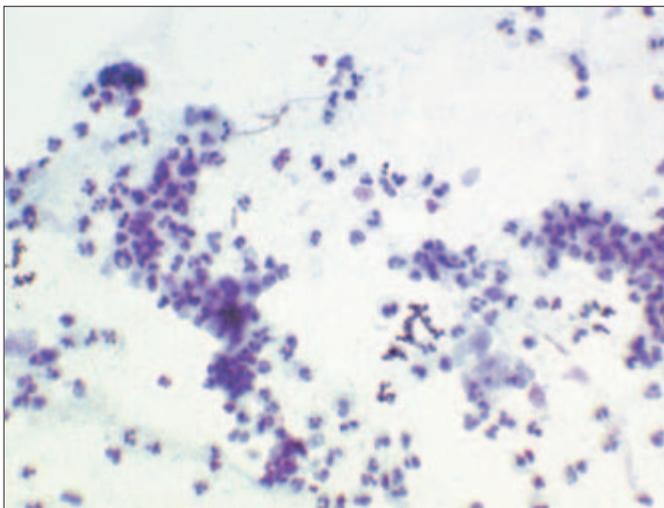
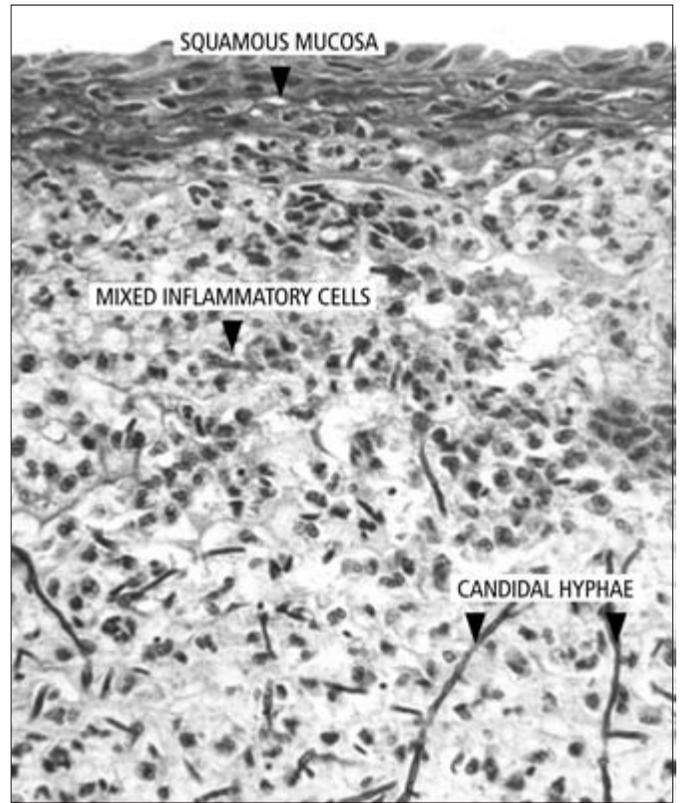
2.203

**Candidiasis (cont.)**

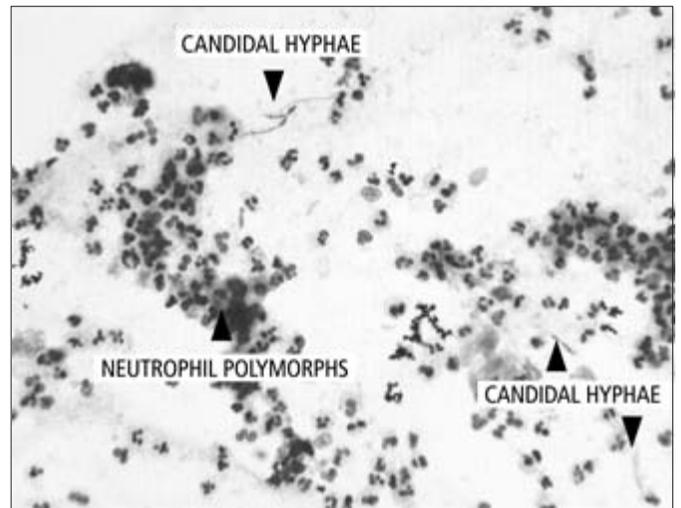
Microbiological, histological (Fig. 2.204) or cytological examination (Fig. 2.205) of the exudate is essential for accurate diagnosis.



2.204



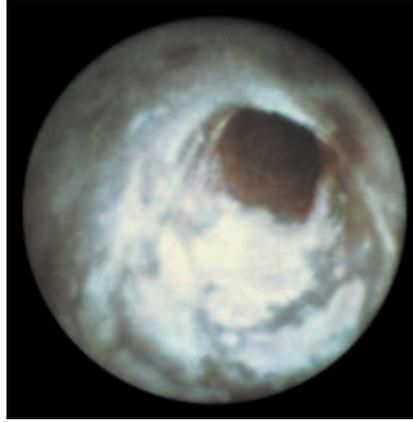
2.205



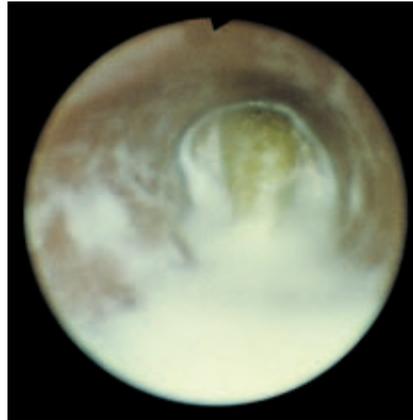
### Appearances after barium radiography

When endoscopy is performed too soon after barium studies, residual contrast material may give an appearance suggestive of candidiasis (Fig. 2.206). Moreover, mucosal coating will prevent adequate examination.

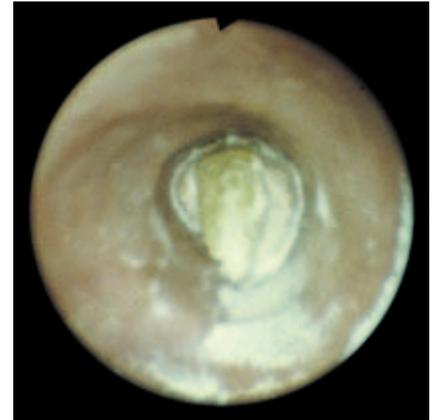
In complete dysphagia some endoscopists advise barium studies before endoscopy. In this patient, radiology showed food bolus obstruction. At subsequent endoscopy the bolus was at first not well seen (Fig. 2.207), but, after contrast was washed away, the plug of meat and vegetable (Fig. 2.208), was easily recognized and removed.



2.206



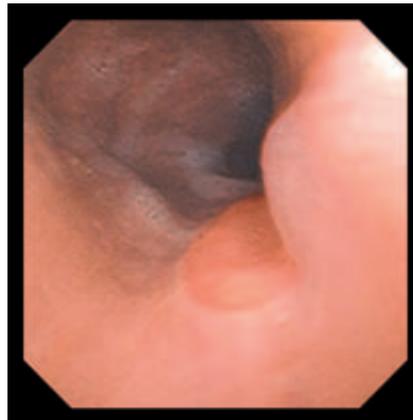
2.207



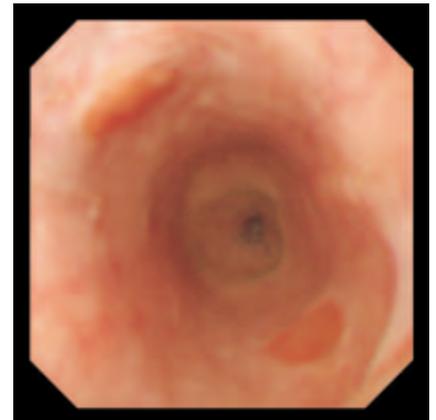
2.208

### Inlet patch in oesophagus (ectopic gastric mucosa)

Rarely, patches or larger areas of the upper third of the oesophagus will be noted to be unduly pink, these areas being sharply circumscribed. The appearances on closer inspection will suggest the presence of gastric type mucosa. Inlet patch in oesophagus may occur as single (Fig. 2.209) or multiple lesions (Fig. 2.210). The pathologist confirmed this diagnosis by finding normal gastric body type mucosa.



2.209



2.210

### Foreign bodies

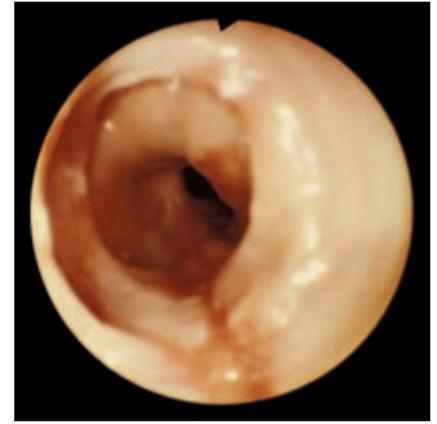
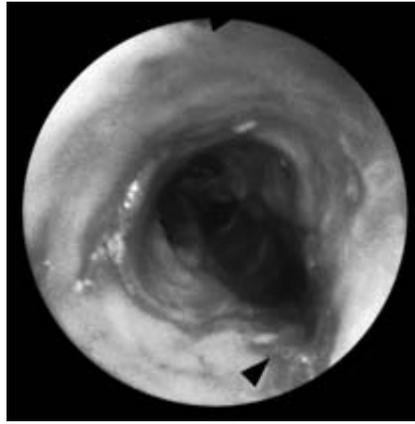
As an example of an oesophageal foreign body, Fig. 2.211 illustrates a retained tablet. However, the commonest foreign body encountered by the endoscopist is a food bolus. Management of foreign bodies is discussed on pp. 187–196 where many foreign bodies retained in the upper gastrointestinal tract and especially the oesophagus are illustrated.



2.211



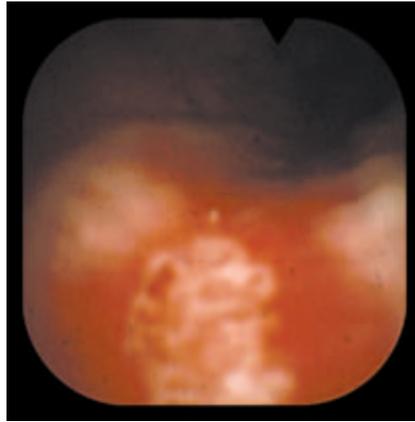
2.212



2.213

### Partial thickness tears

Figure 2.212 shows a mucosal tear after food impaction, while Fig. 2.213 illustrates this tear one month later almost healed. Figure 2.214 shows a longitudinal ulcerated tear surrounded by an erythematous flare. The patient had inadvertently swallowed too large a bite of apple and had experienced considerable retrosternal pain. More severe oesophageal injury and mucosal sloughing following inappropriate food ingestion are shown in Figs 2.215 and 2.216.



2.214



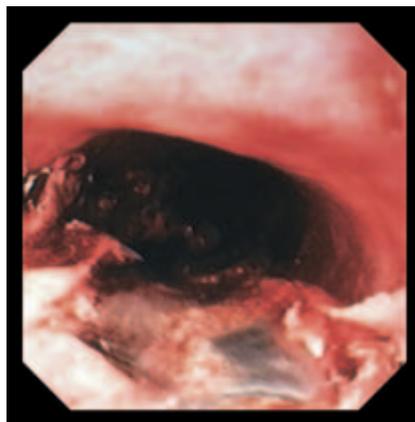
2.215



2.216

### Acute necrotizing oesophagitis

This unusual condition (Fig. 2.217) may be associated with severe reflux, ingestion of certain chemicals and antibiotics, irradiation and overwhelming infection. It may lead to perforation.



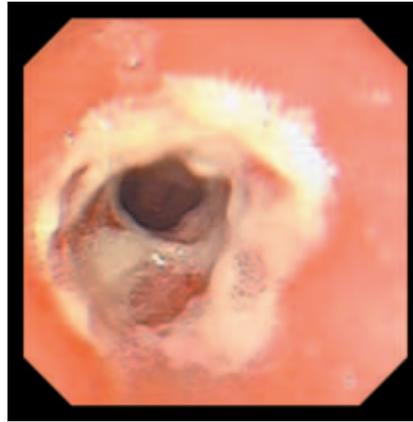
2.217

**Oesophagitis due to drugs and chemicals**

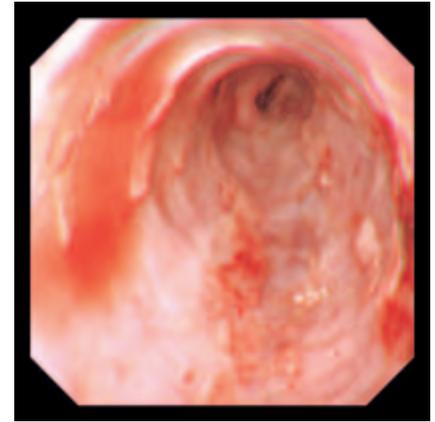
Alendronate is now in common use in the treatment of osteoporosis. Occasionally it may cause injury resembling oesophagitis, sometimes leading to stricture formation (Fig. 2.218). It is contraindicated in patients with gastro-oesophageal reflux disease.

Figure 2.219 illustrates severe confluent lower oesophagitis but in addition there is extensive deep ulceration, the mucosa apparently having been destroyed. The patient was suspected of having swallowed a toxic substance but denied this. The severe pan-oesophagitis shown in Figs 2.220 and 2.221 was unexplained, possibly chemically induced. The following two figures illustrate the sequelae of hydrochloric acid ingestion. Figure 2.222 shows extensive oesophageal mucosal damage, and Fig. 2.223 the appearance of the oesophago-gastric junction. Oesophageal damage due to caustic ingestion is now uncommon in the Western world but see Figs 2.468 and 2.469.

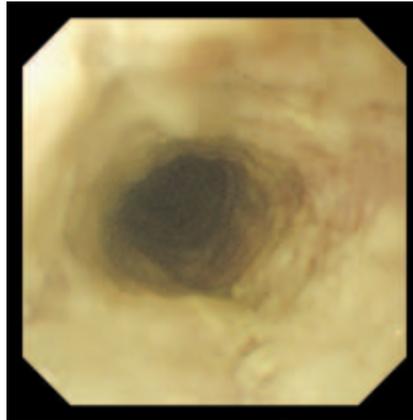
This appearance (Fig. 2.224), also described as the ‘washboard phenomenon’, is rarely seen. In this case, it is due to exposure to acetic acid.



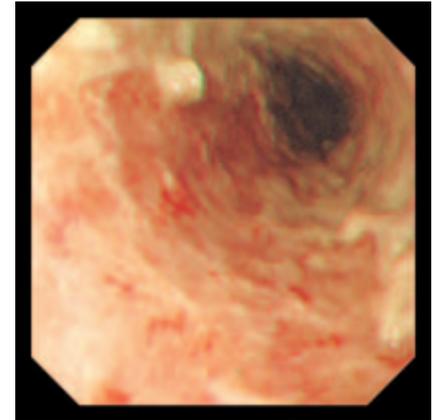
2.218



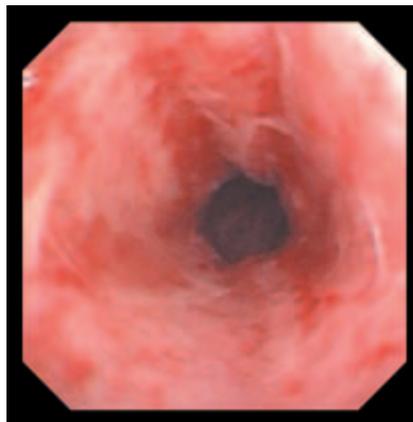
2.219



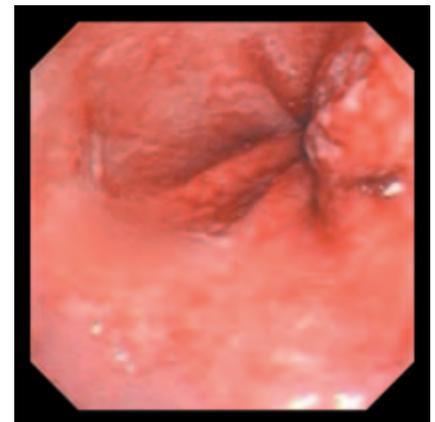
2.220



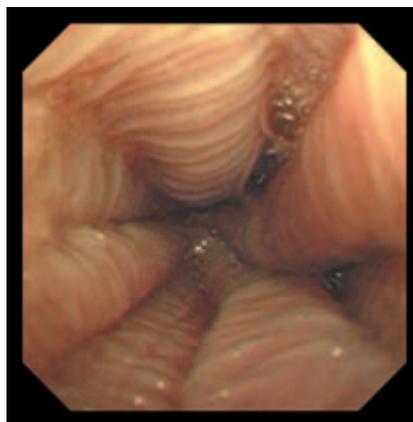
2.221



2.222



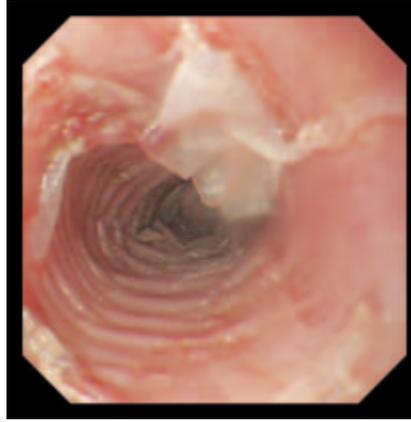
2.223



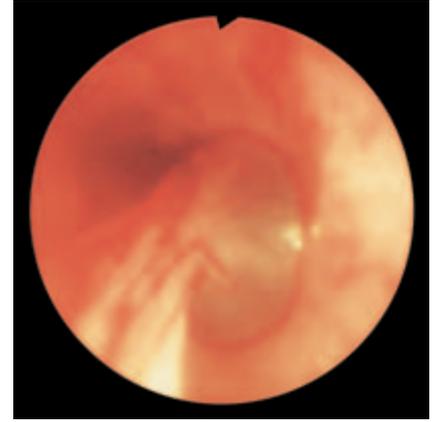
2.224

### Oesophagitis dissecans

In this rare condition a tube of apparently normal noninflamed squamous epithelium may spontaneously separate and be shed, with or without bleeding. Figure 2.225 shows the upper end of such an oesophageal cast still *in situ*. In Figs 2.226 and 2.227 it will be seen how easily the mucosa can be stripped; this could not be done in a normal oesophagus.



2.225



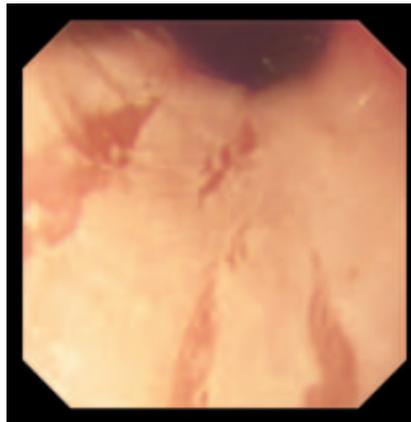
2.226



2.227

### Oesophageal hyperkeratosis

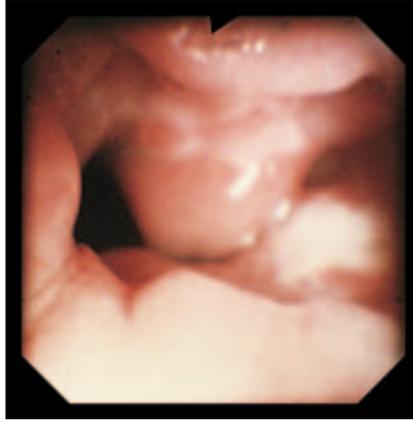
This uncommon condition appears in Fig. 2.228. It may be found in association with tylosis and other hyperkeratotic skin conditions. In this patient, the abnormality was confined to the oesophagus.



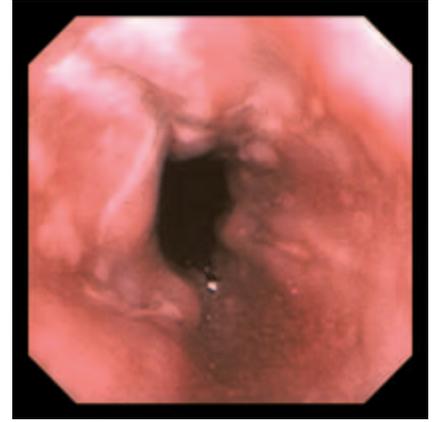
2.228

### Crohn's disease

The oesophagus is rarely involved and even more rarely involved in isolation; there are no specific endoscopic features. The changes may be minimal or well advanced. For example, in the patient shown in Fig. 2.229 the mucosa of the lower oesophagus was lumpy and irregular with marked luminal narrowing. In Fig. 2.230 there is a combination of ulcerative and polypoid changes, together with slight narrowing. The diagnosis may be equally difficult histopathologically due to the findings of nonspecific inflammation and the rarity of granulomas at this site. It is likely when Crohn's disease is known to be present elsewhere, and other causes of inflammation, especially fungal and viral infections, are excluded histologically.



2.229

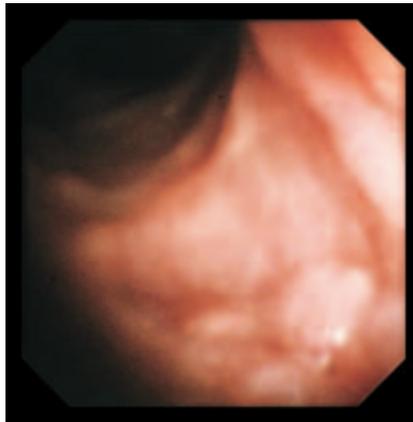


2.230

### Tuberculosis

Biopsies from the nodular lesion shown in Fig. 2.231 revealed mycobacteria, and *M. xenopi* was grown on culture. This unusual lesion healed on antituberculous therapy. Tuberculous oesophagitis is rare and usually accompanies severe untreated pulmonary tuberculosis.

An oesophago-bronchial fistula of tuberculous origin is illustrated in Fig. 2.234.



2.231

### Herpes simplex

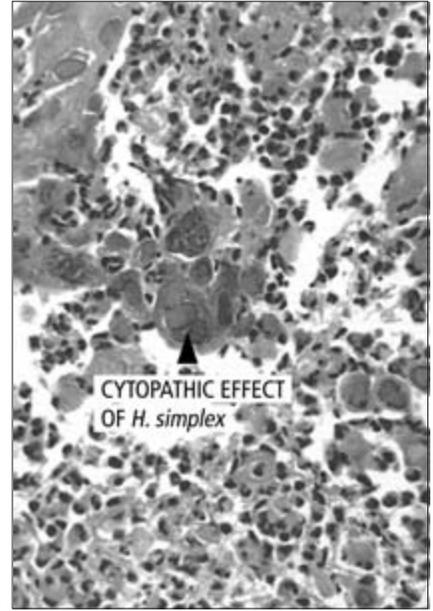
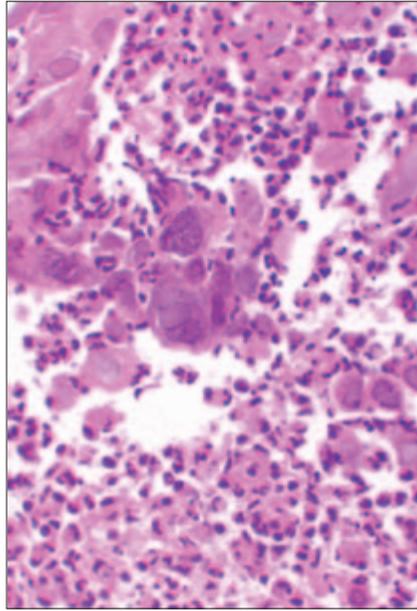
Herpetic lesions originate as vesicles (Fig. 2.183). When these collapse the appearance changes to shallow craters (Fig. 2.232).



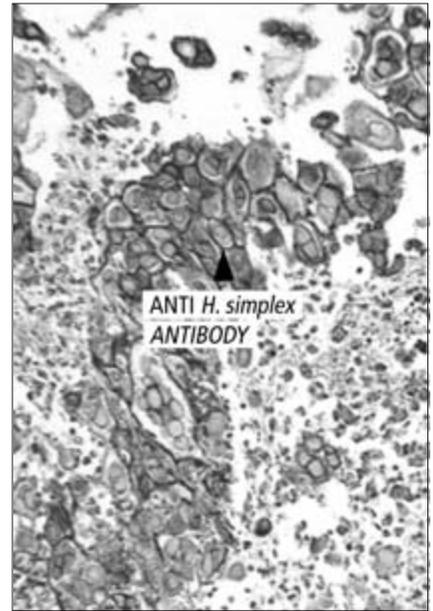
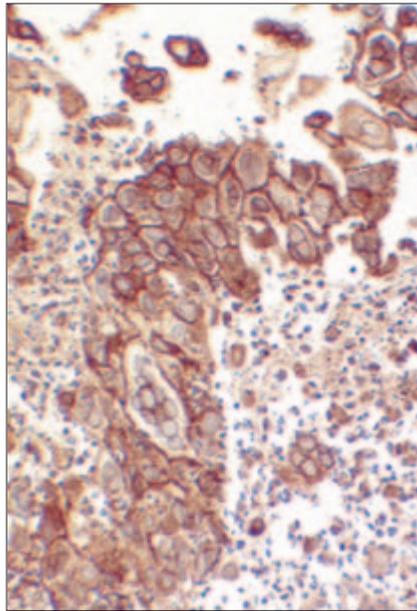
2.232

**Herpes simplex (cont.)**

Histopathological appearances of multinucleate cells with 'glassy nuclei' showing a typical cytopathic effect of *Herpes simplex* in the oesophageal squamous epithelium are shown in Fig. 2.233a. Figure 2.233b shows positive staining of anti-*H. simplex* antibody with immunohistochemistry.



2.233a

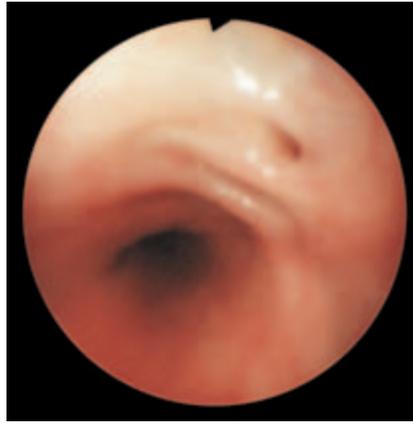


2.233b

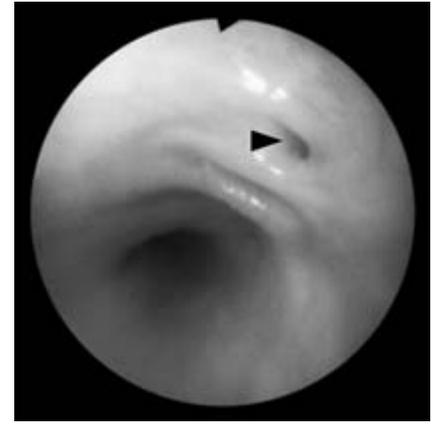
### Fistulae

In Fig. 2.234 there are two abnormalities. The proximal indentation denotes a fistula connecting the oesophagus with the right main bronchus. This had for many years caused coughing after drinking fluids. At surgical repair tissue samples were suggestive of a tuberculous aetiology. The abnormal appearances just distal to the fistula were due to a diverticulum; its proximity to a probably tuberculous lesion makes it likely that it was of the traction type.

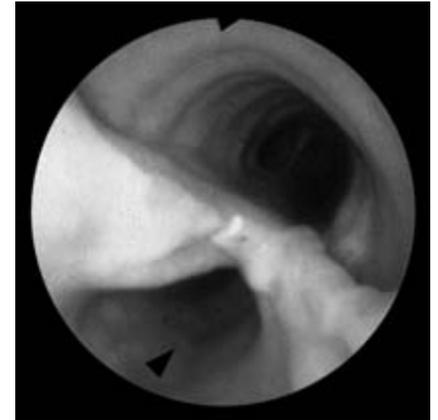
An oesophago-tracheal fistula is shown in Fig. 2.235. This occurred during endoscopy and was caused by the breakdown of neoplastic tissue following radiotherapy for squamous carcinoma arising in the postcricoid region. The upper lumen shows tracheal rings whilst the paler tissue is oesophageal mucosa.



2.234



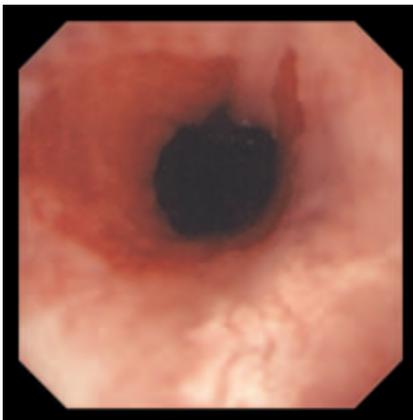
2.235



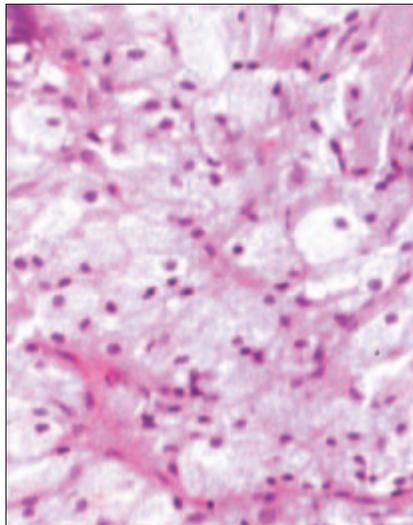
### Xanthelasma

Small white or yellowish lesions, representing a xanthelasma, may occur in any part of the gastrointestinal tract. The lesion shown in Fig. 2.236 was not biopsied but was probably a xanthelasma. Biopsy-proven lesions of this type are illustrated in subsequent sections.

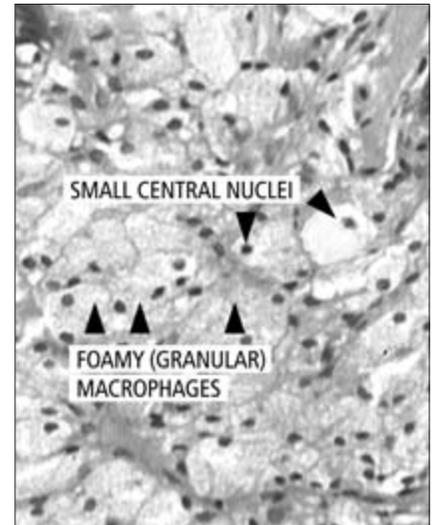
Histopathological appearances are shown in Fig. 2.237. For the pathologist xanthelasma is an important mimic of signet ring cell carcinoma. There are differences: the nuclei are small and central, the cytoplasm is granular, and signet ring cell carcinoma will stain with mucin stains.



2.236

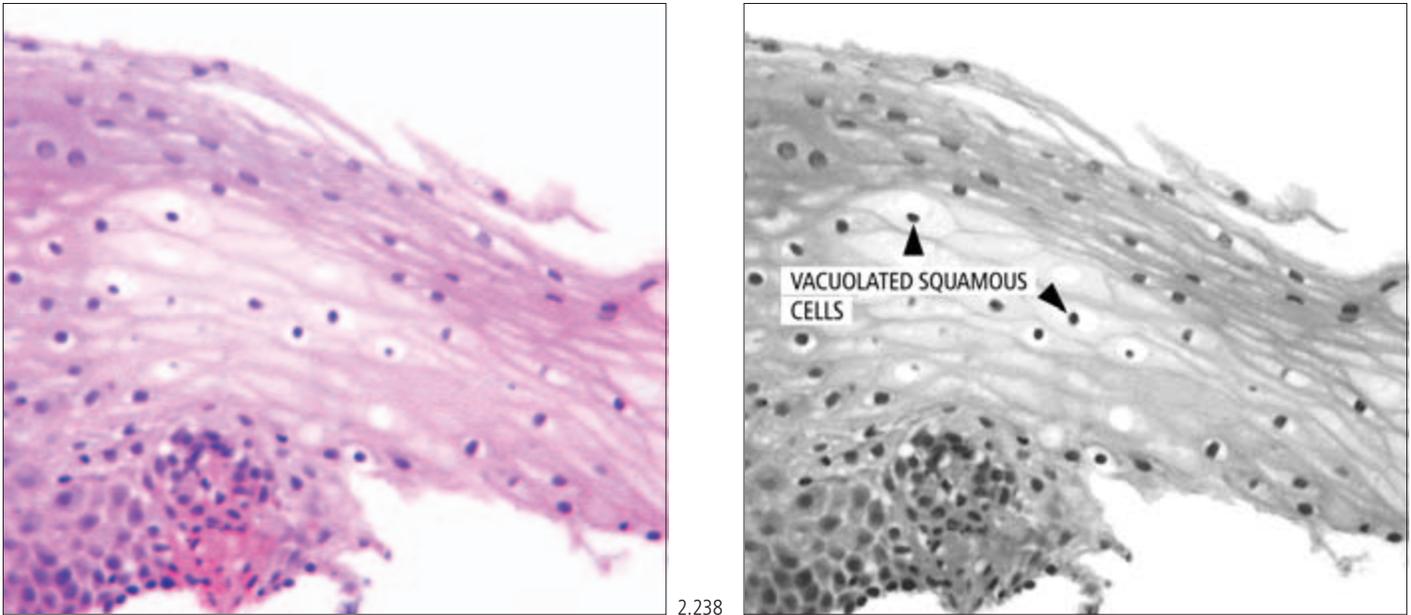


2.237



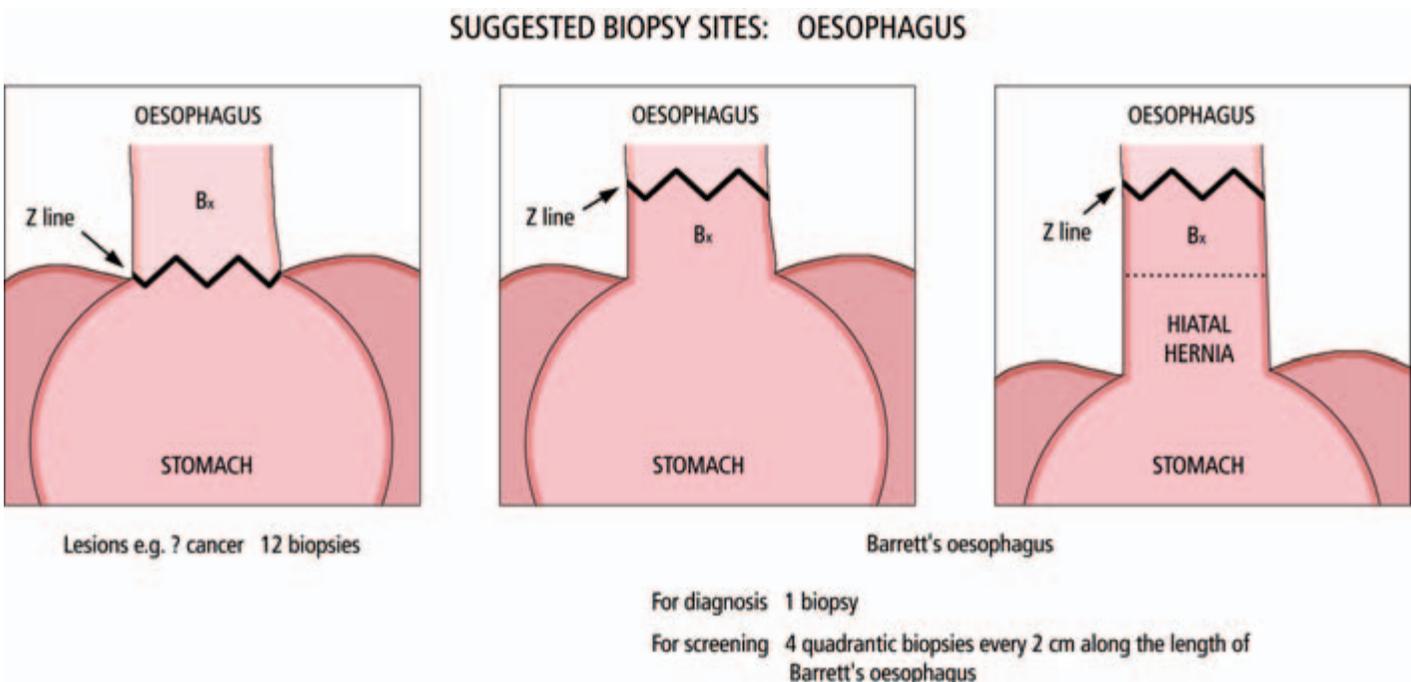
**Glycogenosis**

This is another cause of white patches in the oesophagus. Histologically this condition is characterized by vacuolated squamous cells with a basket weave appearance in the mucosa containing glycogen which can be stained with PAS (Fig. 2.238).



**Suggested biopsy sites**

Figure 2.239 shows suggested biopsy sites for various types of abnormalities and the number of samples that should be taken. For further details the reader is referred to the appropriate sections of this chapter.



2.239

## STOMACH

<b>Diverticula and other deformities</b>	<b>81</b>
<b>Gastritis</b>	<b>84</b>
<b>Erosions</b>	<b>95</b>
<b>Peptic ulcer</b>	<b>97</b>
<b>Benign and malignant tumours</b>	<b>106</b>
<b>Vascular abnormalities</b>	<b>127</b>
<b>Pancreas-related conditions</b>	<b>130</b>
<b>Vomiting and trauma</b>	<b>132</b>
<b>Miscellaneous conditions</b>	<b>134</b>
<b>Suggested biopsy sites</b>	<b>139</b>

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### Diverticula and other deformities

Congenital diverticula

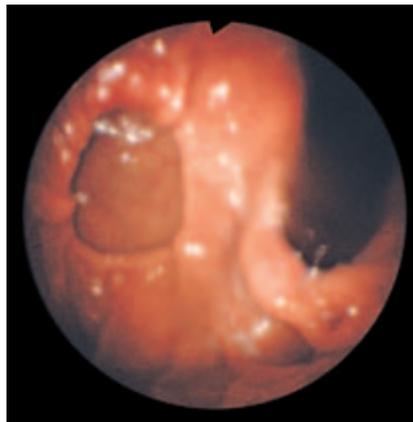
Volvulus

Hourglass deformity

Prepyloric and pyloric deformities

#### Congenital diverticula

Congenital gastric diverticula are usually situated in the upper portion of the stomach and are best seen when the endoscope is in the inverted position. A wide mouth (Fig. 2.240) is more common than a narrow mouth (Fig. 2.241). The walls of such diverticula are thin and, should the tip of the endoscope be forcibly introduced, there is a risk of perforation. When they occur in the fundus, diverticula must be differentiated from paraoesophageal hernia (Fig. 2.118).



2.240



2.241

**Volvulus**

The appearance of gastric volvulus with the endoscope in the J-position is shown in Fig. 2.242. Volvulus is more likely to occur in the presence of rolling hiatal (paraesophageal) hernia (Fig. 2.118).



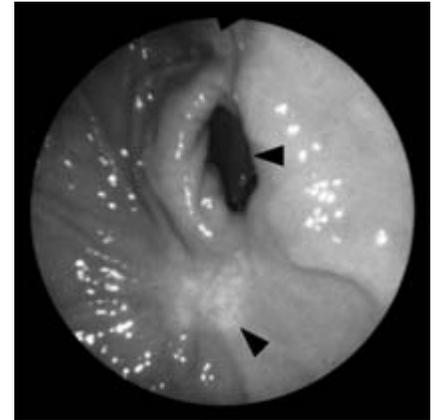
2.242

**Hourglass deformity**

Gastric ulcer healing is often accompanied by fibrosis. With the ulcer situated in the body of the stomach this may lead to the formation of a ring-like circumferential constriction, a so-called hourglass deformity (Fig. 2.243). A benign gastric ulcer lying in the constriction is shown in Fig. 2.244. Note the stellate scar of a previous ulcer proximal to the constriction (Fig. 2.243).



2.243



2.244

**Prepyloric and pyloric deformities**

Figure 2.245 shows a prepyloric pseudodiverticulum. This may result from previous duodenal ulceration causing an adhesion to form between the bulb and the antrum.

The normal pylorus when fully relaxed is round (Fig. 2.43). Past or present inflammation on or near it from, for example, a prepyloric ulcer, may cause a cicatricial deformity (Fig. 2.246).

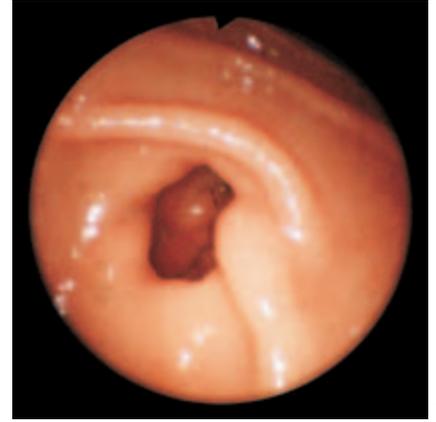
Figure 2.247 illustrates a sentinel prepyloric fold suggesting past or present ulceration distal to the pyloric ring.

Prepyloric ulcers occasionally perforate through into the duodenum and on healing leave a double pylorus (Fig. 2.248). Congenital double pylorus has a similar appearance.

Adult as opposed to infant hypertrophic pyloric stenosis, unrelated to peptic ulceration, probably does not occur. Duodenal ulceration may, however, lead to narrowing of the bulb and associated widening of the pylorus.



2.245



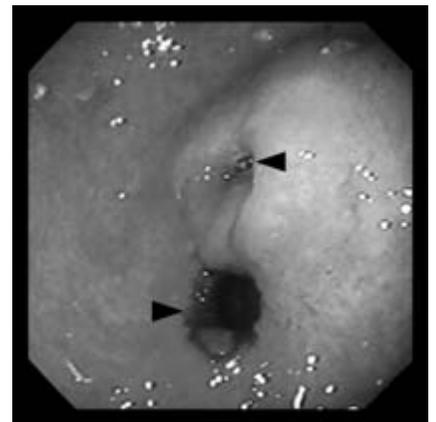
2.246



2.247



2.248



## Gastritis

### Non-atrophic gastritis

### Atrophic gastritis

### Chemical (reactive, reflux) gastritis

### Lymphocytic gastritis

### Crohn's disease

### Eosinophilic gastritis

### Gastritis in progressive systemic sclerosis

The term 'gastritis' should be used with circumspection. It implies an histological diagnosis. The endoscopist often observes areas of gastric mucosa which look redder or more mottled than expected, perhaps differing from adjacent mucosa and suggesting the diagnosis of gastritis, but this may not be confirmed when biopsy material is studied. Conversely, biopsies from normal-looking mucosa may be genuinely gastritic. Histopathology is the 'gold standard': endoscopic appearances are fallible.

Gastritis has been classified in many ways. The most useful current classification is probably the modified Sydney System (Table 2.4). Dixon *et al.* (1996) stress the importance of multifocal atrophy as a risk factor for dysplasia and malignancy. They also relate topography and histological appearances to the cause of the gastritis. These authors recommend biopsies from the antrum (2), body (2) and incisura (1).

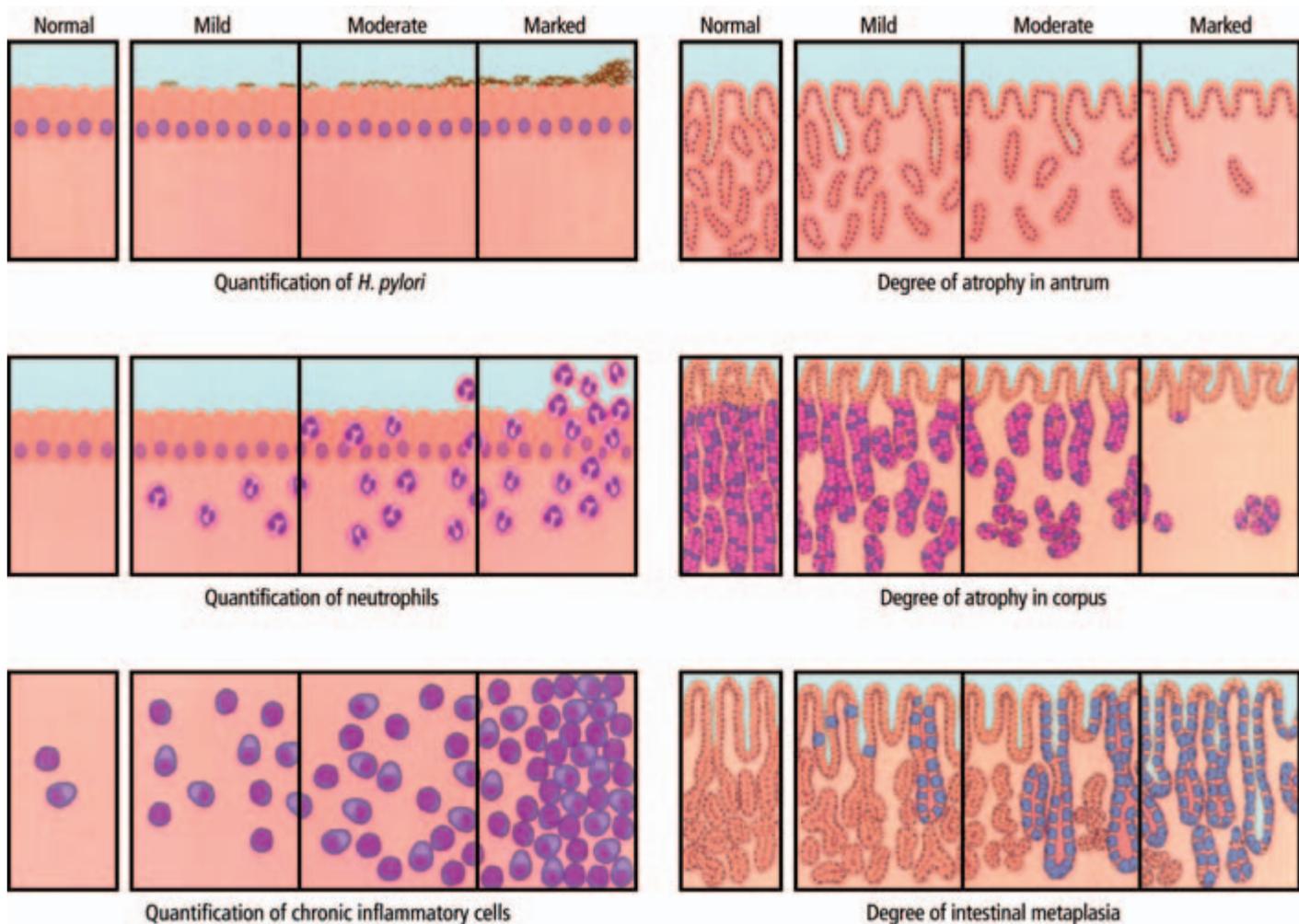
**Table 2.4** Classification of gastritis according to topography, morphology and aetiology (adapted from Dixon *et al.*, 1996)

Type	Aetiology	Site	Synonym
<b>Non-atrophic</b>	<i>Helicobacter pylori</i> and other <i>Helicobacter</i>	Antral	Type B
<b>Atrophic</b>			
Autoimmune	Autoimmunity	Corporal	Type A
Multifocal	<i>Helicobacter pylori</i>	Pangastritis	Type B
<b>Special types</b>			
Chemical	Chemical irritation Bile NSAIDs Other irritants	Antral, corporal	Type C, reactive, reflux, postoperative
Radiation	Radiation injury		
Lymphocytic	Gluten enteropathy Idiopathic		} Varioliform
Granulomatous	Crohn's disease (usually focal chronic active gastritis) Sarcoidosis Wegener's granulomatosis Churg–Strauss syndrome Idiopathic		
Eosinophilic	Food sensitivity Idiopathic		
Other infective	Bacteria (other than <i>H. pylori</i> ) Viruses Fungi Parasites		Acute phlegmonous

### Gastritis (cont.)

Each of the histological features should be graded in three categories as in the visual analogue scale shown in Fig. 2.249 adapted from Dixon *et al.* 1996. *Helicobacter pylori*-associated gastritis and reactive/chemical gastritis are the two commonest types in routine practice. Activity in *H. pylori*-associated gastritis is recognized by the presence of neutrophils in the epithelium. Chronic inflammatory changes may take up to one year to resolve following *H. pylori* eradication. The presence of neutrophils in a biopsy some time after treatment of *H. pylori* infection is a good indicator of persistence. If the pattern of inflammation, i.e. diffuse chronic inflammation with neutrophils in the epithelium, suggests the presence of *H. pylori* but organisms cannot be found, it may be because of very recent treatment or because of intestinal metaplasia which presents an unfavourable epithelial environment for *H. pylori*. If difficulty is encountered in identifying *H. pylori* from other bacteria on the surface of a gastric biopsy, anti-*H. pylori* antibody staining may be useful (Fig. 2.250). This is too expensive to use in every case, but is valuable when mixed organisms are present and when a diligent search using tinctorial stains has been unrewarding but the pattern of inflammation suggests *H. pylori* infection.

In reactive or chemical gastritis there may be absence or relative absence of inflammation. Such gastritis is caused by chemical irritation, usually bile reflux or NSAIDs. It is usually antral-predominant gastritis, as in many patients with *H. pylori*-related gastritis, but is also common in the operated stomach.

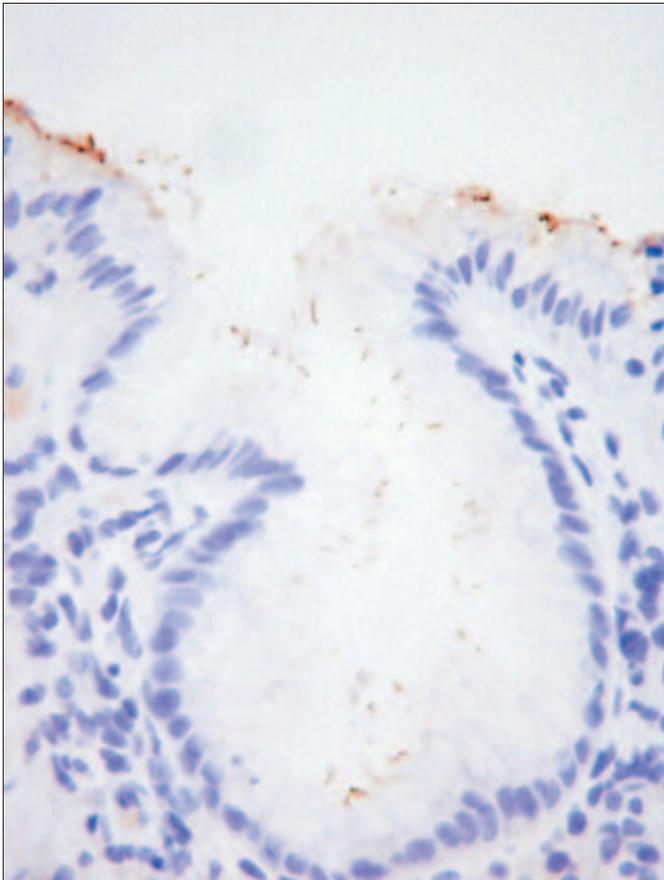


2.249

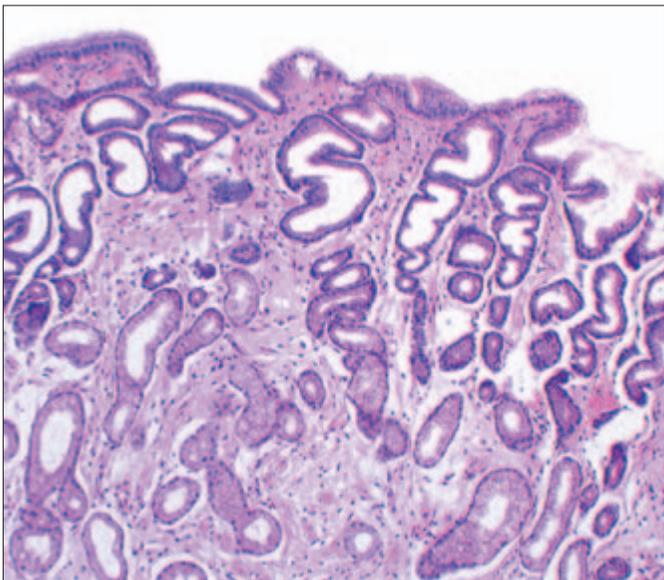
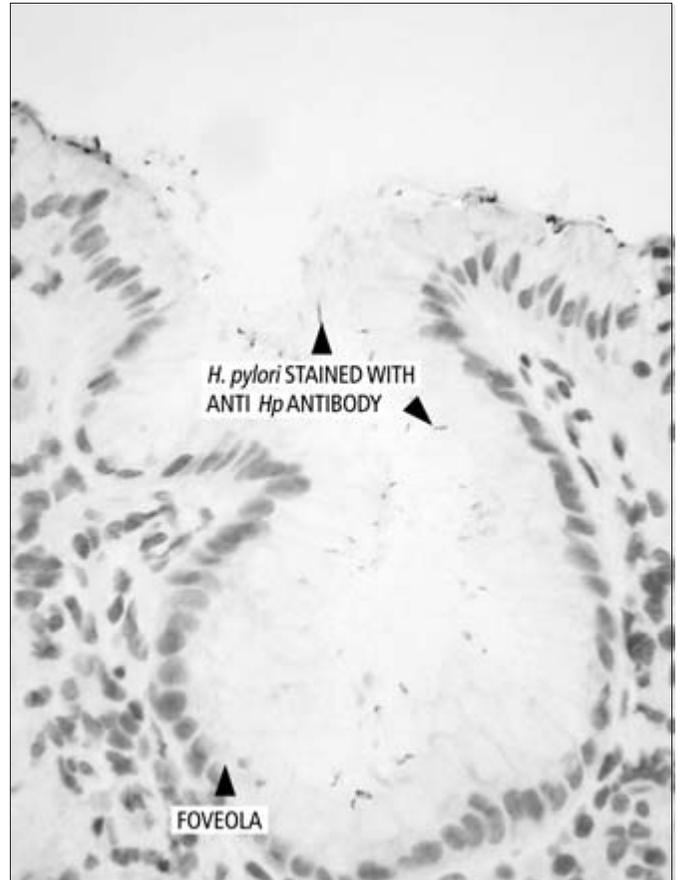
**Gastritis (cont.)**

The characteristic histological features (Fig. 2.251) are oedema, foveolar hyperplasia, vascular ectasia, smooth muscle fibres in excess in the lamina propria and relative absence of inflammation. The foveolar hyperplasia may be so marked as to mimic dysplasia—a differential diagnosis of some importance.

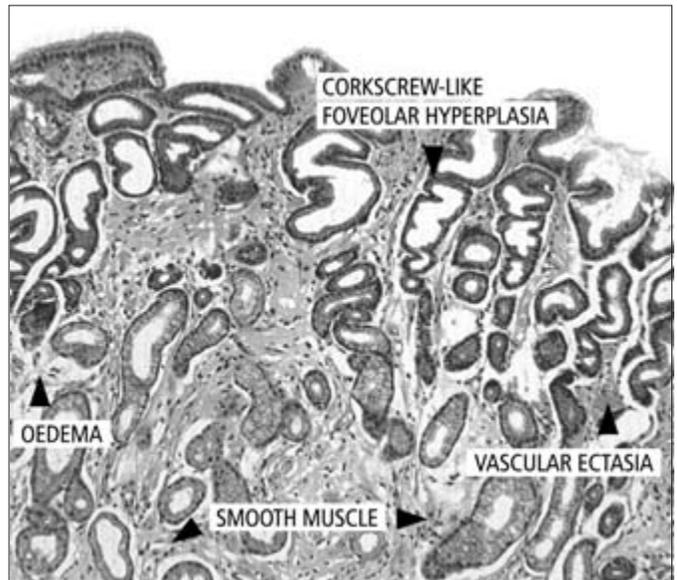
Other less common forms of gastritis are also listed in Table 2.4. Of great importance are lymphocytic gastritis and the recently recognized focal active gastritis of Crohn's disease.



2.250



2.251



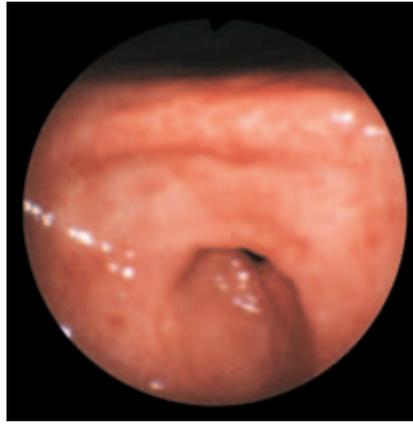
### Non-atrophic gastritis

The appearances shown in Figs 2.252 and 2.253 are typical: the mucosa is slightly reddened, granular and mottled.

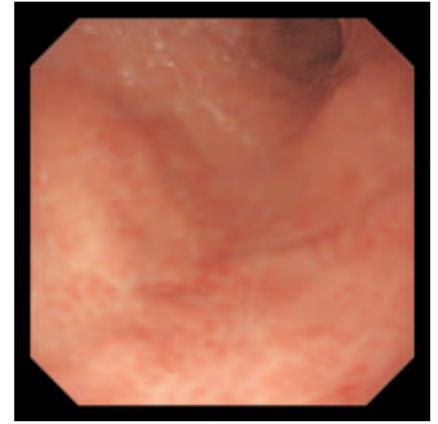
Such gastritis may be sharply localized, as in the example of antral gastritis shown in Fig. 2.254. Another example of localized acute antral gastritis is shown in Fig. 2.255; this patient had test results indicative of infection with *H. pylori*. Examples from another patient show pangastritis with mottling; Fig. 2.256 illustrates the antral and Fig. 2.257 the body appearances. Incomplete intestinal metaplasia was seen in the biopsies.

Nodular gastritis may occur in response to infection with various types of *Helicobacter*. The causative organism in the gastritis shown in Fig. 2.258 was *H. heilmannii*.

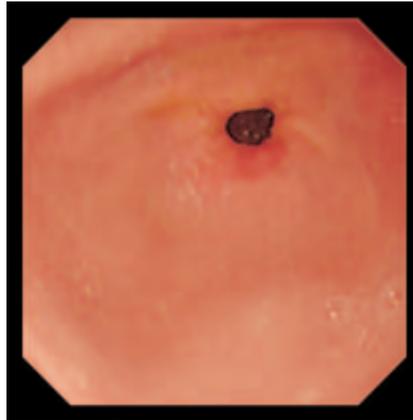
Hypertrophy of the gastric mucosa leads to hypersecretion. Figure 2.259 demonstrates the cobblestone appearance of hypersecretory body mucosa resulting from antral infection with *H. pylori*. Hypertrophy is less marked in Fig. 2.260.



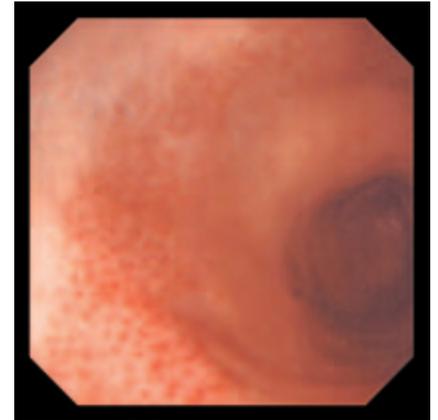
2.252



2.253



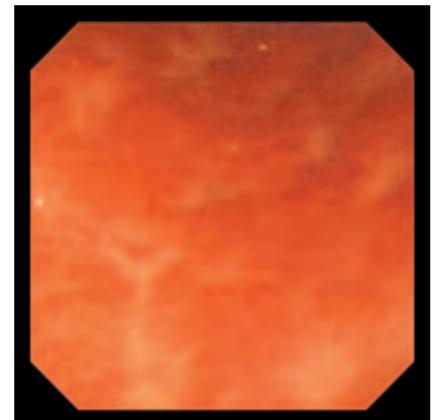
2.254



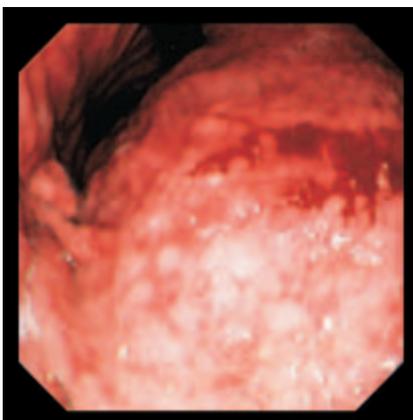
2.255



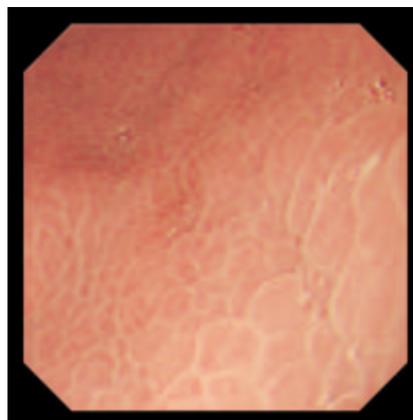
2.256



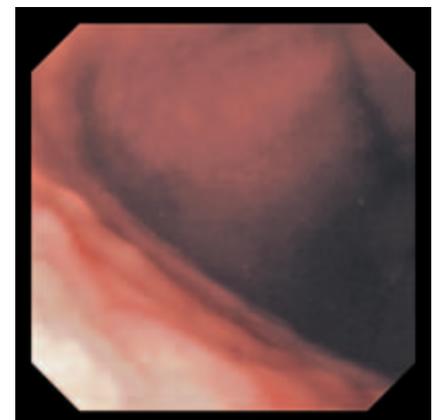
2.257



2.258



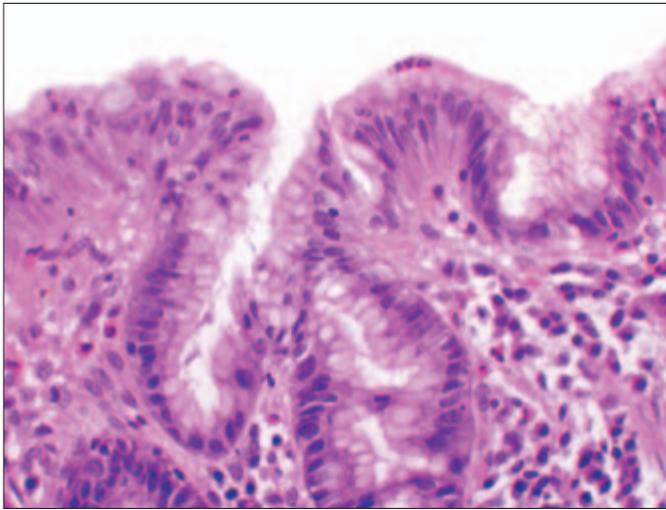
2.259



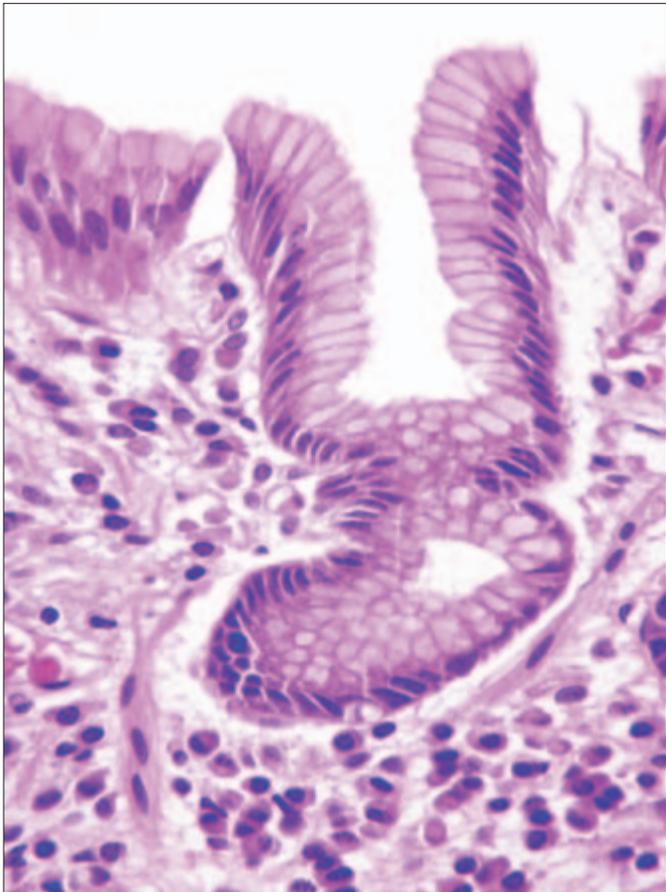
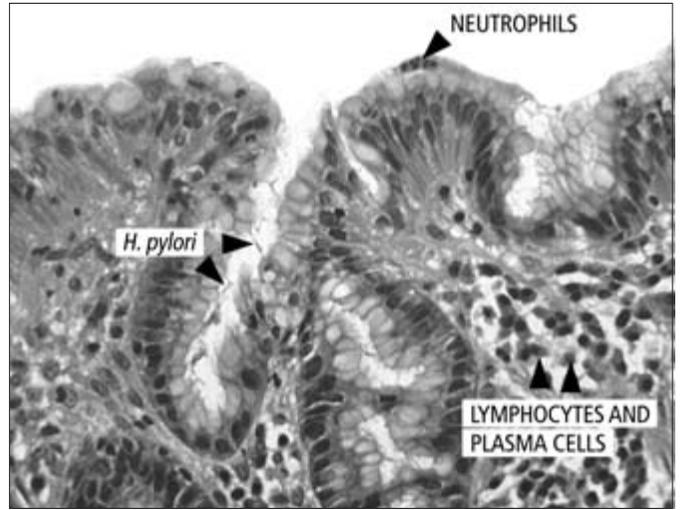
2.260

**Non-atrophic gastritis (cont.)**

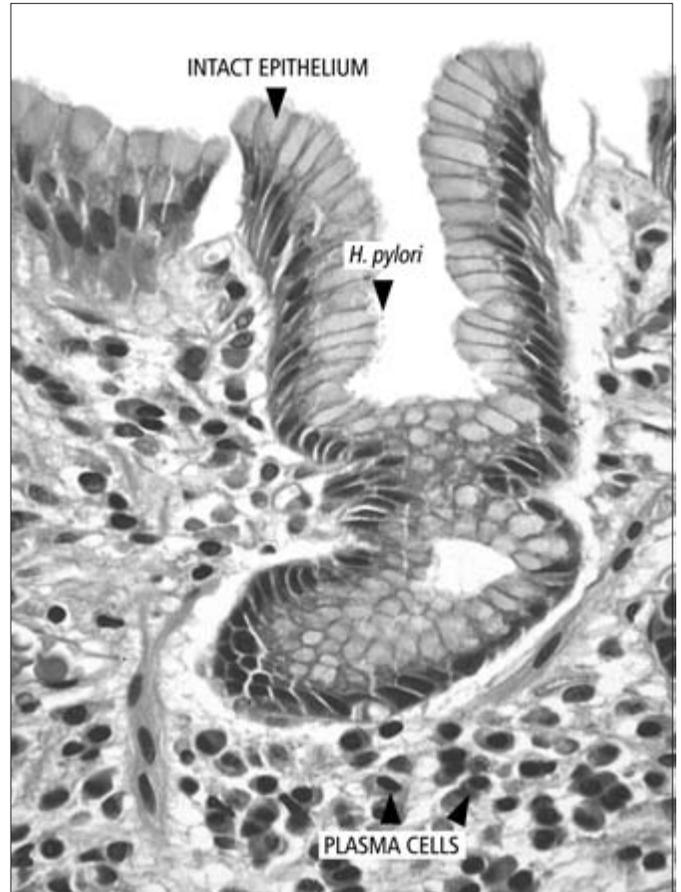
Figure 2.261 shows the histopathological appearances of active *H. pylori* infection associated with chronic antral gastritis, while Fig. 2.262 illustrates chronic antral gastritis in inactive *H. pylori* infection.



2.261



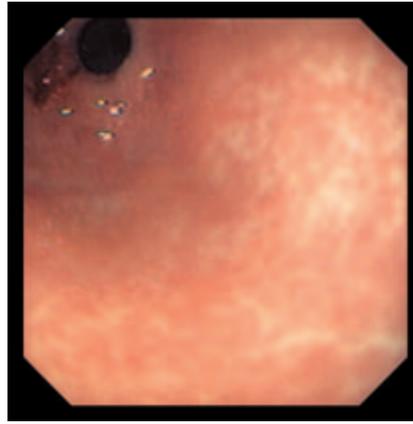
2.262



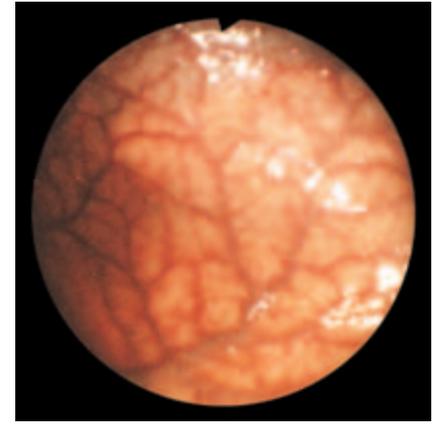
### Atrophic gastritis

In atrophic gastritis mottling is a common feature (Fig. 2.263). When the gastritic process has advanced to mucosal atrophy, the mucosa is thinned and the venous plexus can easily be seen (Fig. 2.264). Dysplastic changes and carcinoma may supervene. When endoscopic appearances resembling mucosal atrophy are limited to the fundus, biopsies often show no abnormalities.

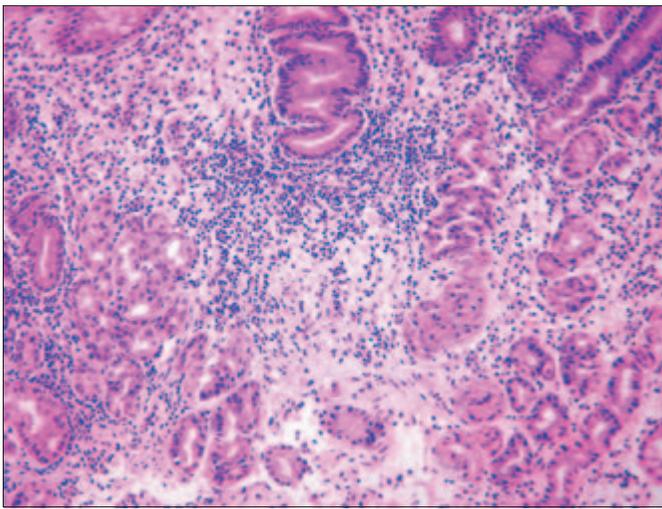
The pathology of gastric mucosal atrophy in body-type mucosa and in antral-type mucosa appears, respectively, in Figs 2.265 and 2.266.



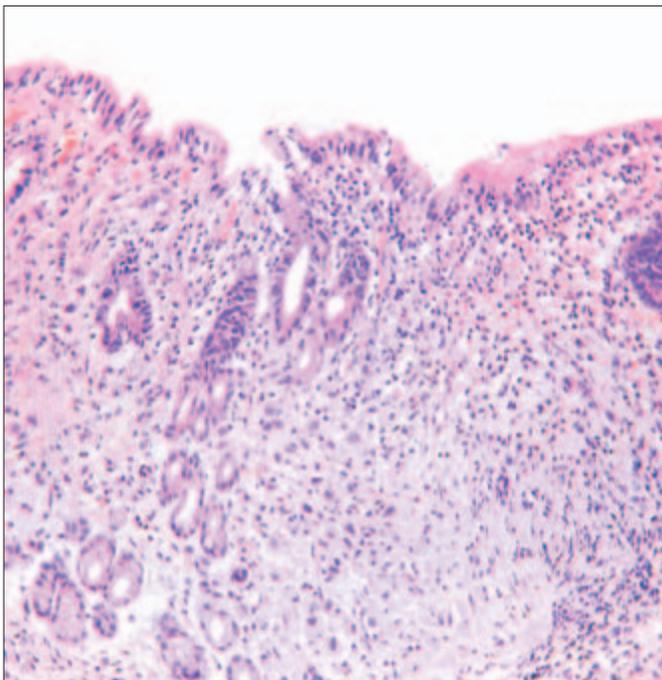
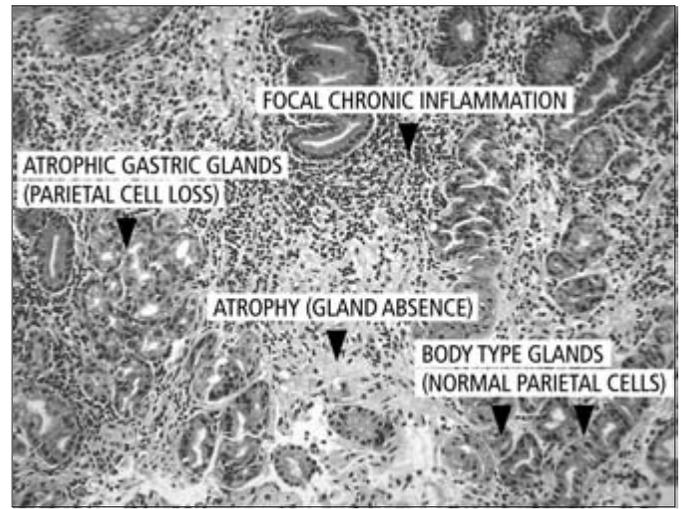
2.263



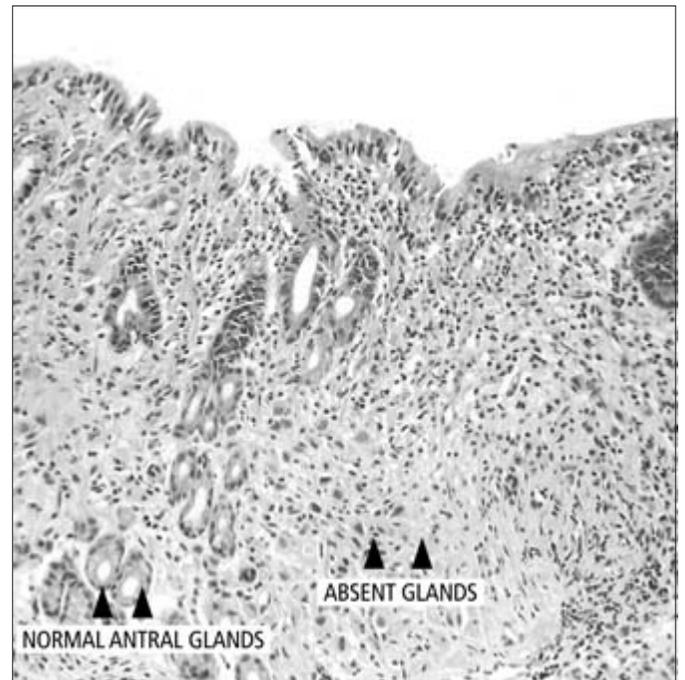
2.264



2.265

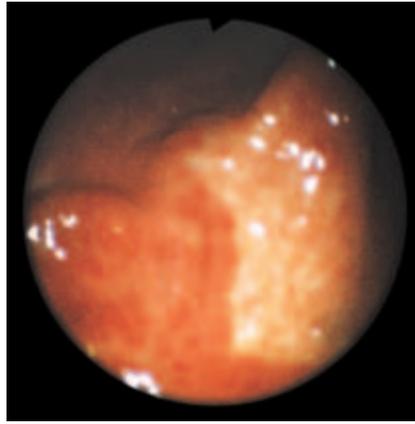


2.266

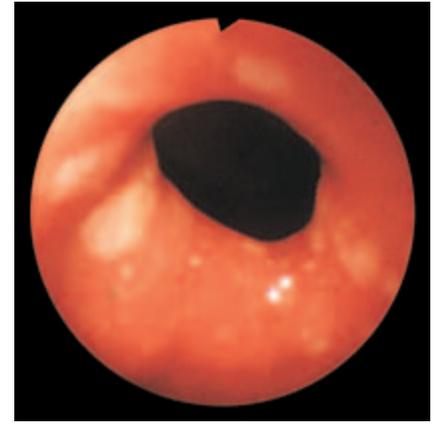


**Atrophic gastritis (cont.)**

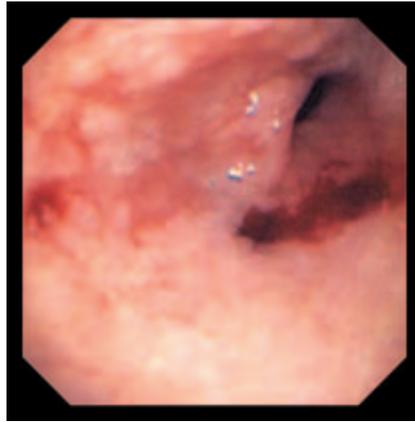
Intestinal metaplasia is commonly found in areas of atrophic gastritis, and is usually accompanied by some degree of mucosal atrophy. There are no specific endoscopic features. However, when there are larger (Fig. 2.267) or smaller (Fig. 2.268) whitish areas in normal-looking or reddened mucosa, intestinal metaplasia should be suspected. The appearances of intestinal metaplasia may also be nodular (Fig. 2.269). The pathological appearances are shown in Fig. 2.270. As this condition can presage malignant change, it is advisable to take multiple biopsies. The issue of long-term endoscopic and histological surveillance of atrophic gastritis and gastric intestinal metaplasia is controversial.



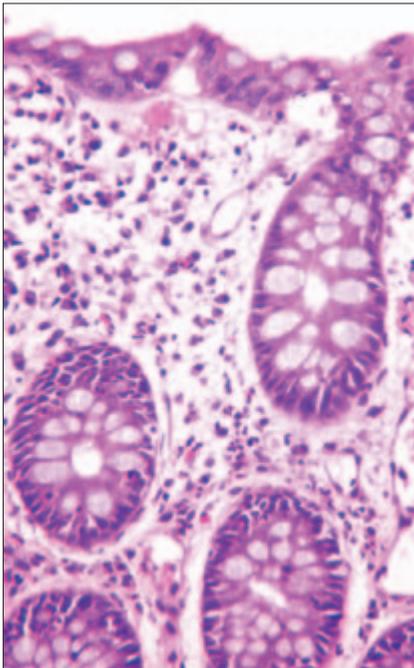
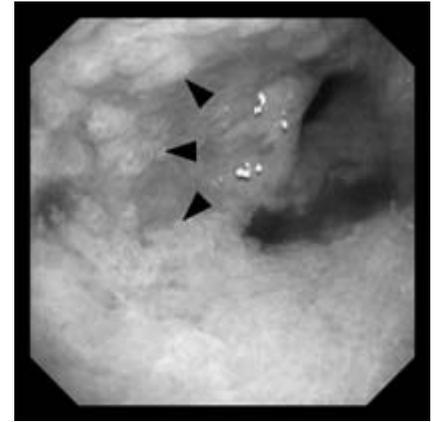
2.267



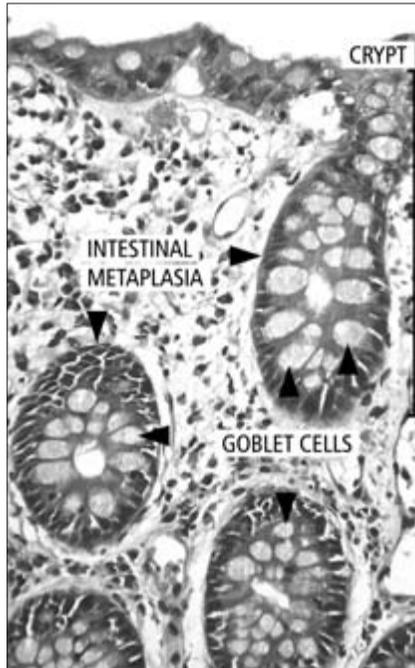
2.268



2.269

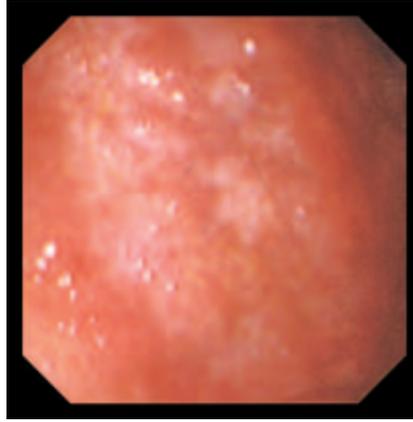


2.270

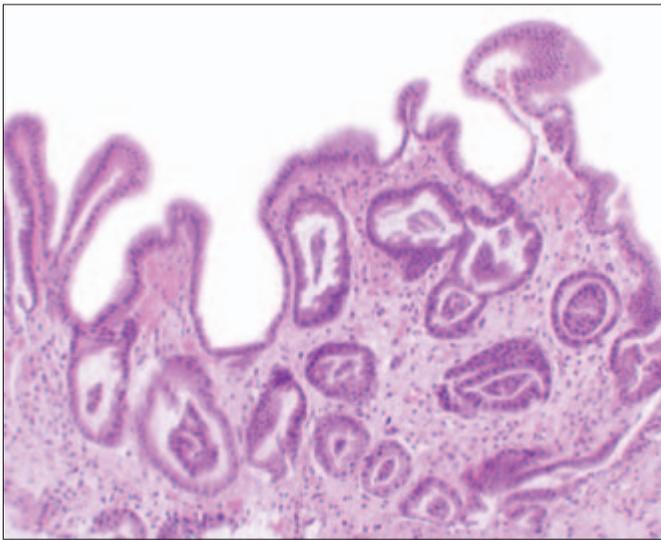


**Chemical (reactive, reflux) gastritis**

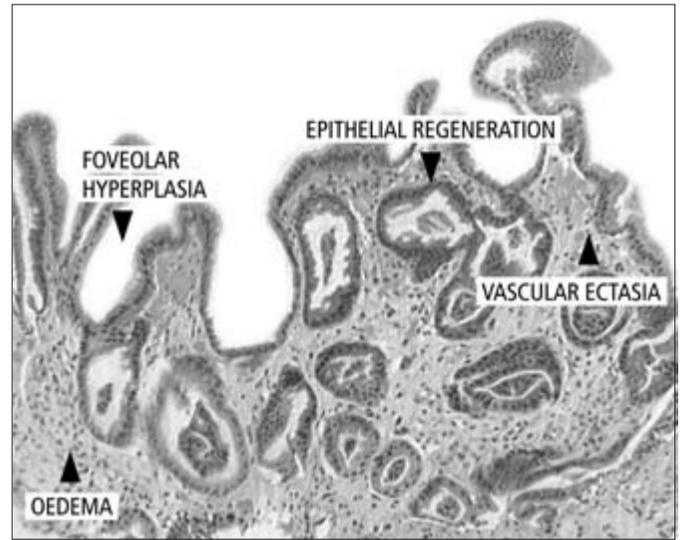
This is common in the postoperative stomach (Fig. 2.271). There are no specific endoscopic features. Bile reflux is often seen at endoscopy and may be causative; Fig. 2.272 shows the pathological appearances.



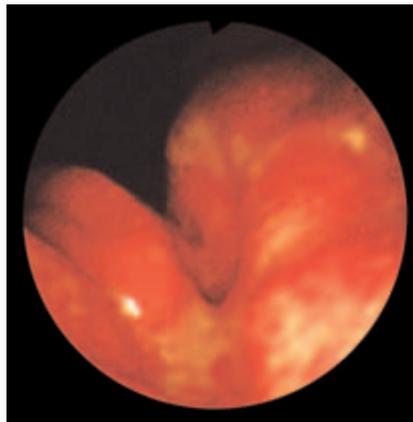
2.271



2.272



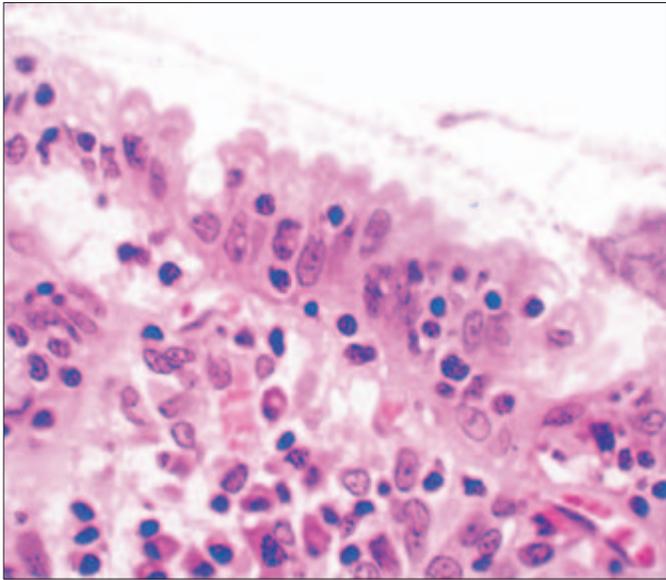
A form of gastritis (Fig. 2.273), again to the endoscopist nonspecific, may be observed in patients with gastric outlet obstruction, for example in pyloro-duodenal stenosis of whatever cause. This may histologically prove to be *H. pylori* gastritis, reactive gastritis or sometimes lymphocytic gastritis.



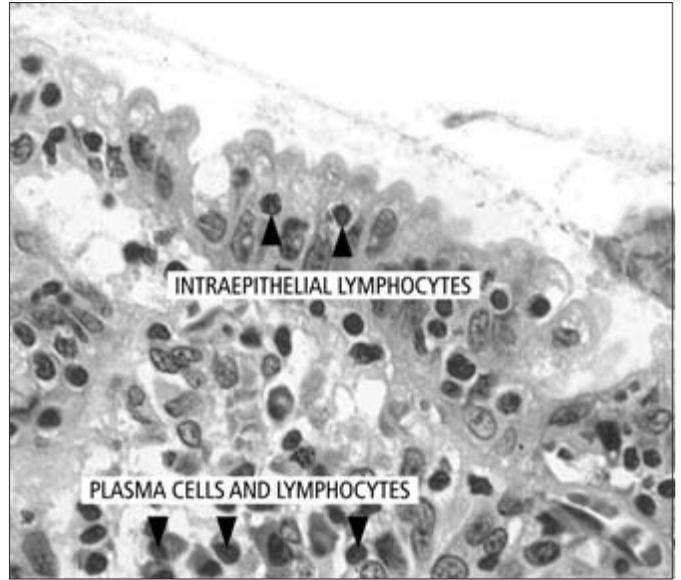
2.273

### Lymphocytic gastritis

Lymphocytic gastritis has increased numbers of intraepithelial lymphocytes (> 25 per 100 epithelial cells). It occurs in association with coeliac disease (especially when the gastritis is antral), or lymphocytic colitis. Occasionally it may be seen in stomachs that contain tumours, or in association with gastric outlet obstruction. The pathological features are shown in Fig. 2.274.

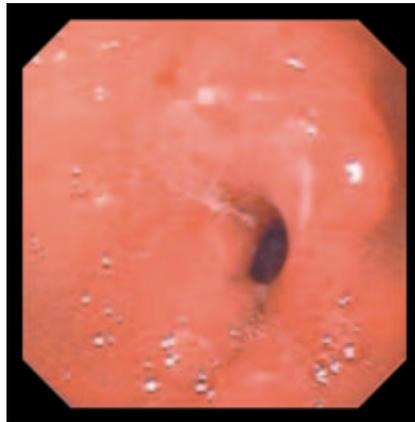


2.274

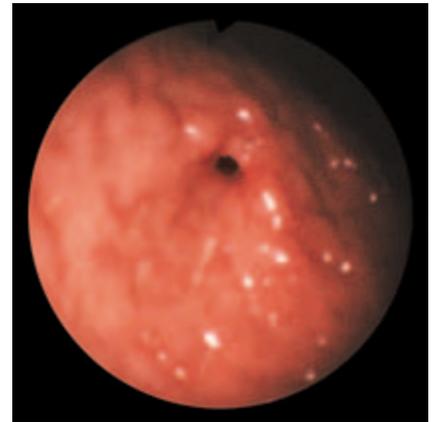


### Crohn's disease

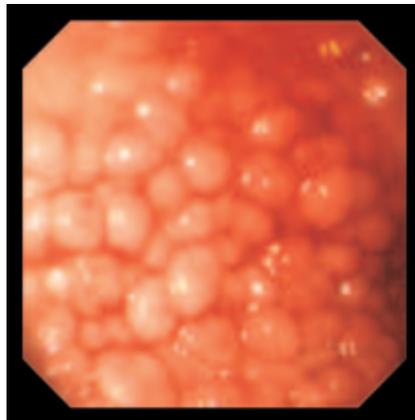
Crohn's disease of the stomach is manifested by changes similar to those seen elsewhere in the gastrointestinal tract. In Fig. 2.275 there is prepyloric ulceration. Figure 2.276 illustrates linear ulcers in an irregular polypoid mucosa. 'Cobble-stoning' is shown in Fig. 2.277; the whole stomach had a similar appearance in this patient. Gastric Crohn's disease is uncommon and is unlikely to be found without involvement of small or large bowel.



2.275



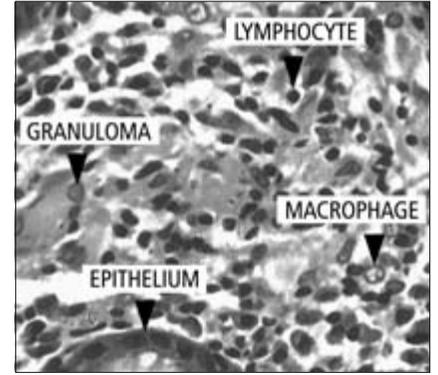
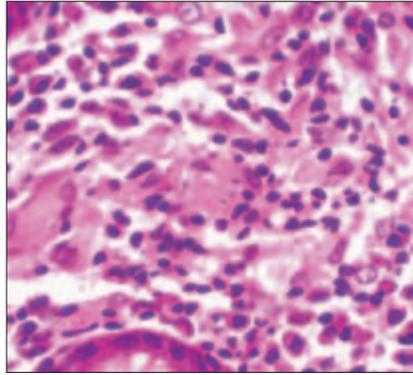
2.276



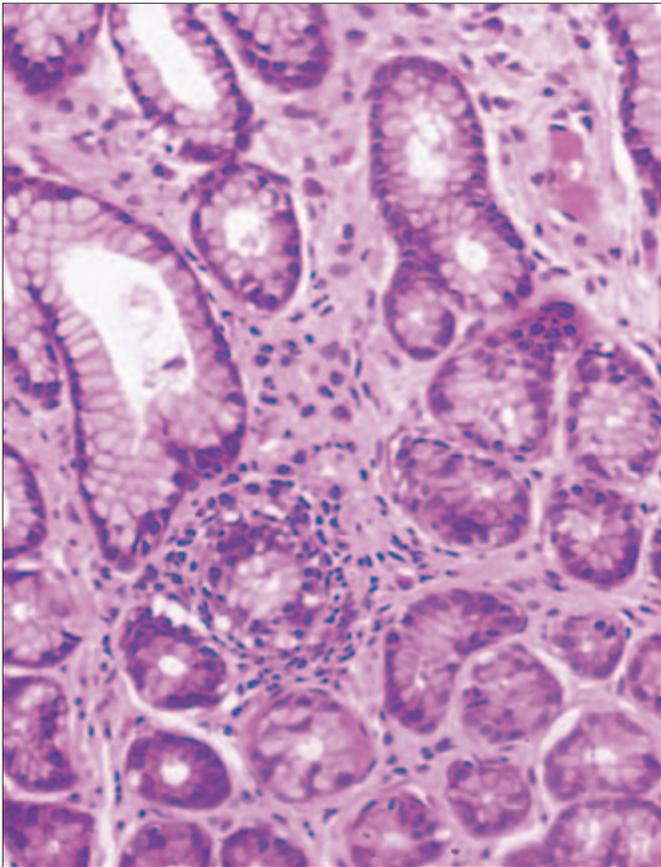
2.277

**Crohn's disease (cont.)**

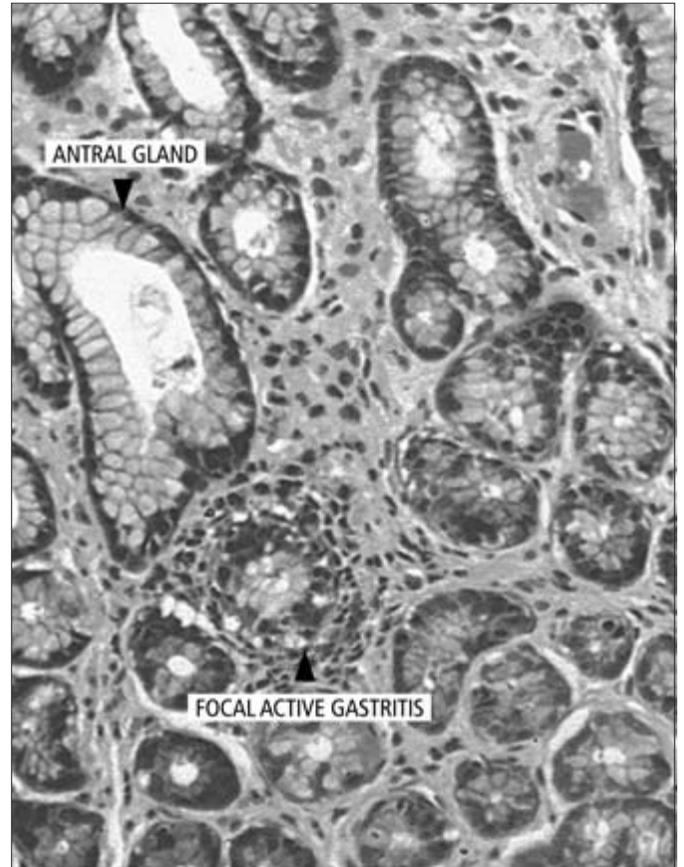
Histologically the findings are focal active gastritis, which may also be seen in endoscopically normal stomachs in a proportion of patients with known Crohn's disease elsewhere. The lesions comprise focal nondestructive lympho-epithelial aggregates in association with neutrophils infiltrating the same epithelial site; this may occur with (Fig. 2.278) or without (Fig. 2.279) granuloma formation. Thus gastric biopsy may be helpful in the differential diagnosis of a difficult colitis.



2.278

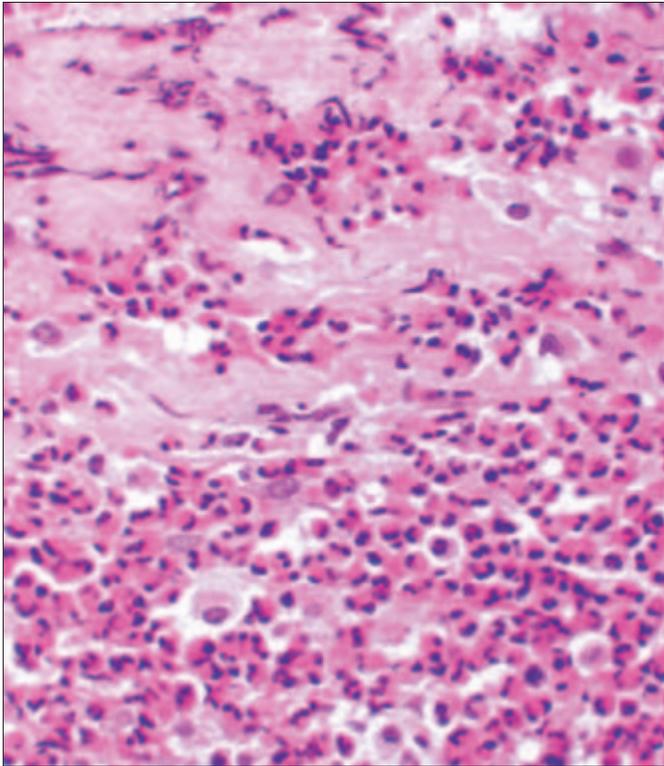


2.279

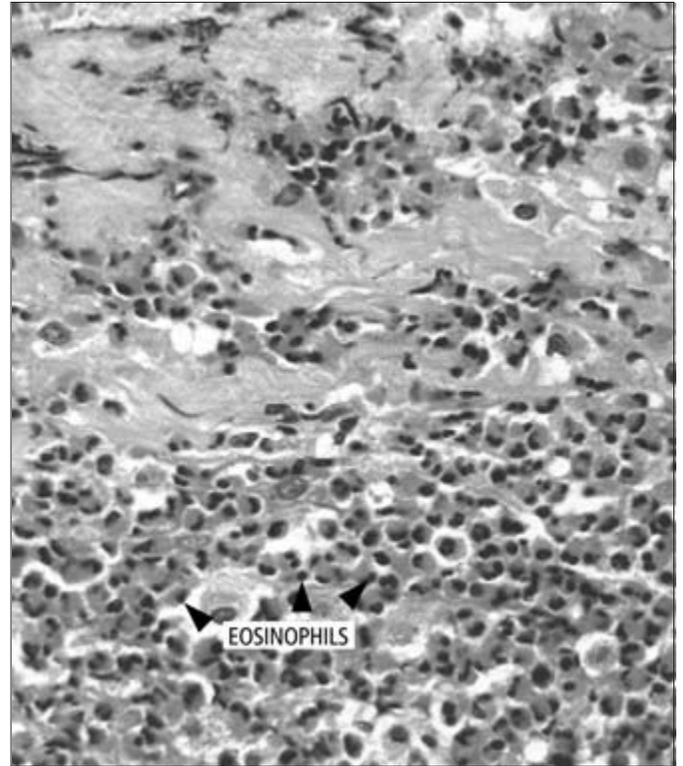


### Eosinophilic gastritis

This is an uncommon but important mimic of linitis plastica. It affects the antrum which becomes thickened and rigid with an irregular mucosa. This abnormality usually extends to involve the duodenum. By contrast, linitis plastica (p. 118) is usually limited to the stomach. In eosinophilic gastritis, eosinophils are seen throughout all layers of the wall (Fig. 2.280) and an associated peripheral eosinophilia is common. Care is needed to exclude an inflammatory reaction to adjacent adenocarcinomas, intestinal worms and eosinophilic vasculitis, as in the Churg–Strauss syndrome.

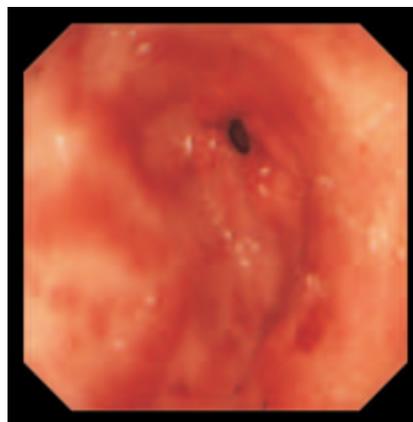


2.280



### Gastritis in progressive systemic sclerosis

Progressive systemic sclerosis (scleroderma) may lead to various gastrointestinal motility and mucosal changes. Figure 2.281 illustrates nonspecific but severe antritis in such a patient.



2.281

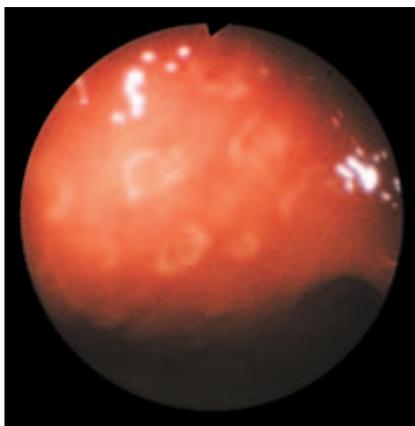
## Erosions

An erosion is a partial thickness defect in an epithelial surface, as distinct from an ulcer which is a full thickness breach of an epithelial surface. Both are pathological. Attempts have been made to classify erosions, but from the endoscopic point of view such terms as 'acute' or 'complete' may have little substance. Thus a descriptive approach may be best unless the causative factor is known or there are features on biopsy pointing to a specific diagnosis. Also, it is difficult to distinguish an erosion from a small gastric (or duodenal) ulcer solely on endoscopic appearances.

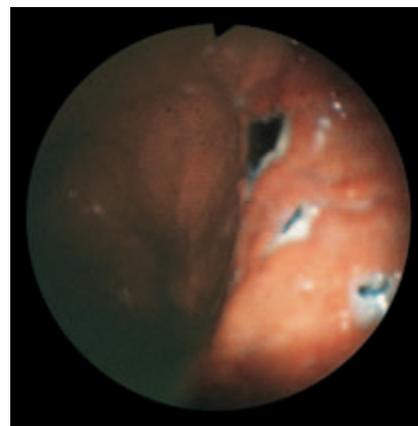
### Erosions

Erosions are usually multiple and may occur anywhere in the stomach. Figure 2.282 illustrates multiple flat erosions. The black base of the erosions shown in Fig. 2.283 suggests recent bleeding.

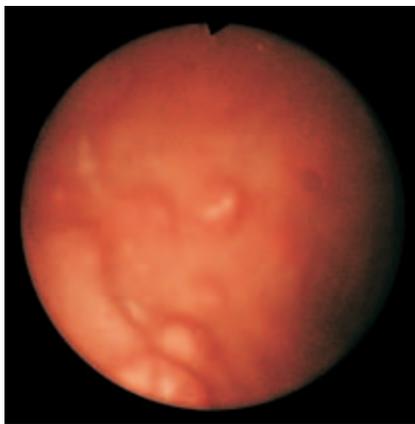
The scattered raised erosions appearing in Fig. 2.284 were found in the antrum where rugae are usually less prominent. The cause of the erosions in Fig. 2.285 was thought to be aspirin. Similar lesions may follow the ingestion of nonsteroidal anti-inflammatory drugs (NSAIDs). Gastric erosions are commonly found on the crests of rugae, and may be round (Fig. 2.286) or elongated (Fig. 2.287).



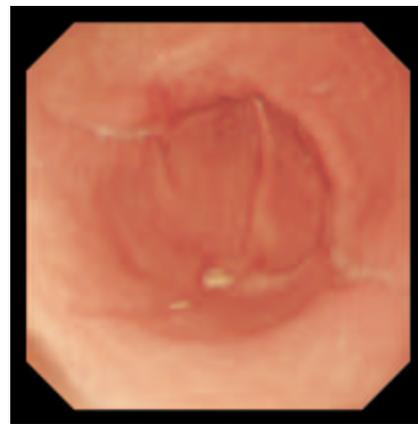
2.282



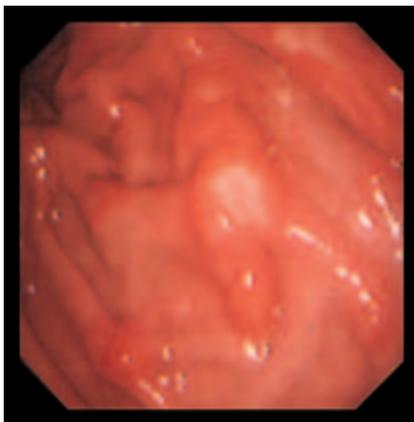
2.283



2.284



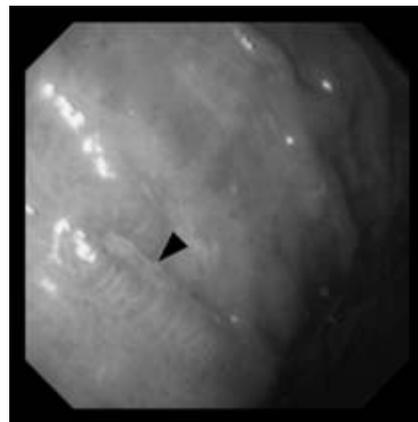
2.285



2.286

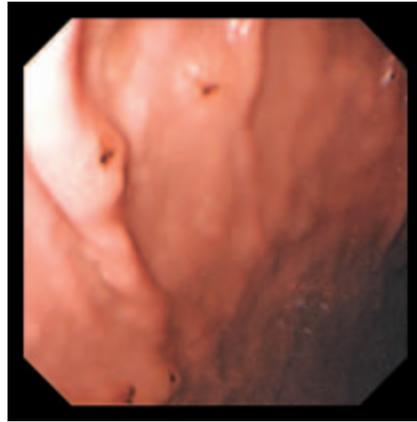


2.287

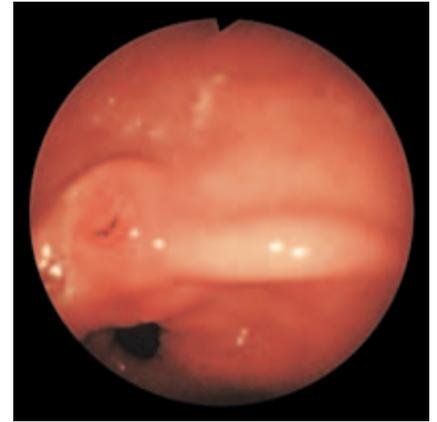


**Erosions (cont.)**

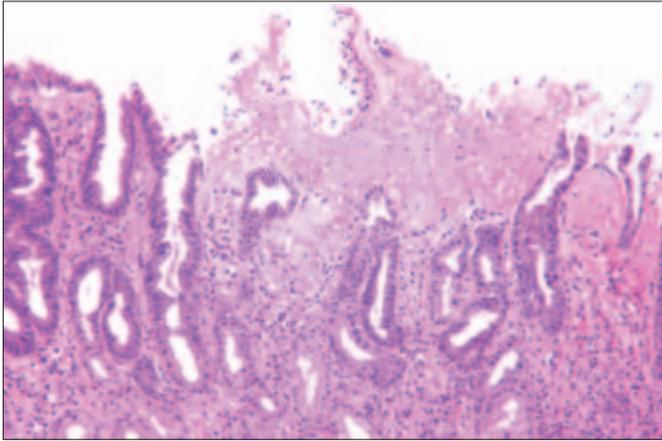
Minimal bleeding is commonly seen (Fig. 2.288) even when there is no other clinical evidence of this. There may be endoscopic evidence of inflammation (Fig. 2.289).



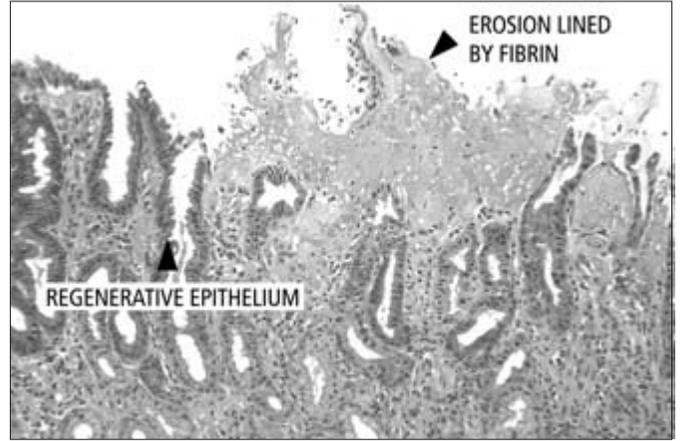
2.288



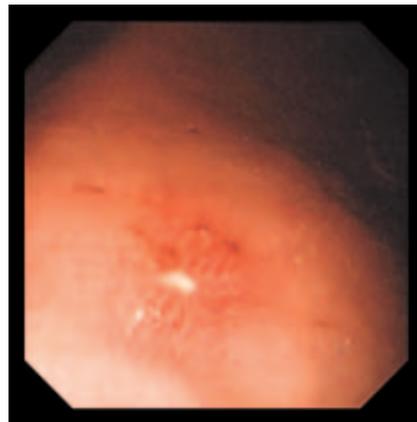
2.289



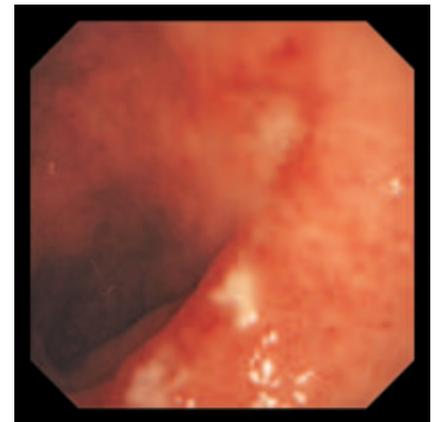
2.290



Typical nonspecific appearances of biopsy material from a gastric erosion are shown in Fig. 2.290. On endoscopic appearances alone there is uncertainty whether the isolated lesion shown in Fig. 2.291, or the multiple incisural lesions appearing in Fig. 2.292, are erosions or ulcers. Similar problems arise when commenting on appearances as shown in Figs 2.293–2.295: are these multiple antral erosions or gastric ulcers?



2.291



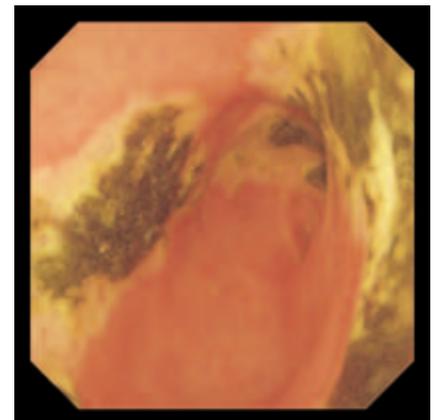
2.292



2.293



2.294



2.295

## Peptic ulcer

### Histopathology

Variations in shape and size

Variations in site

Multiple ulcers

Healing and scarring

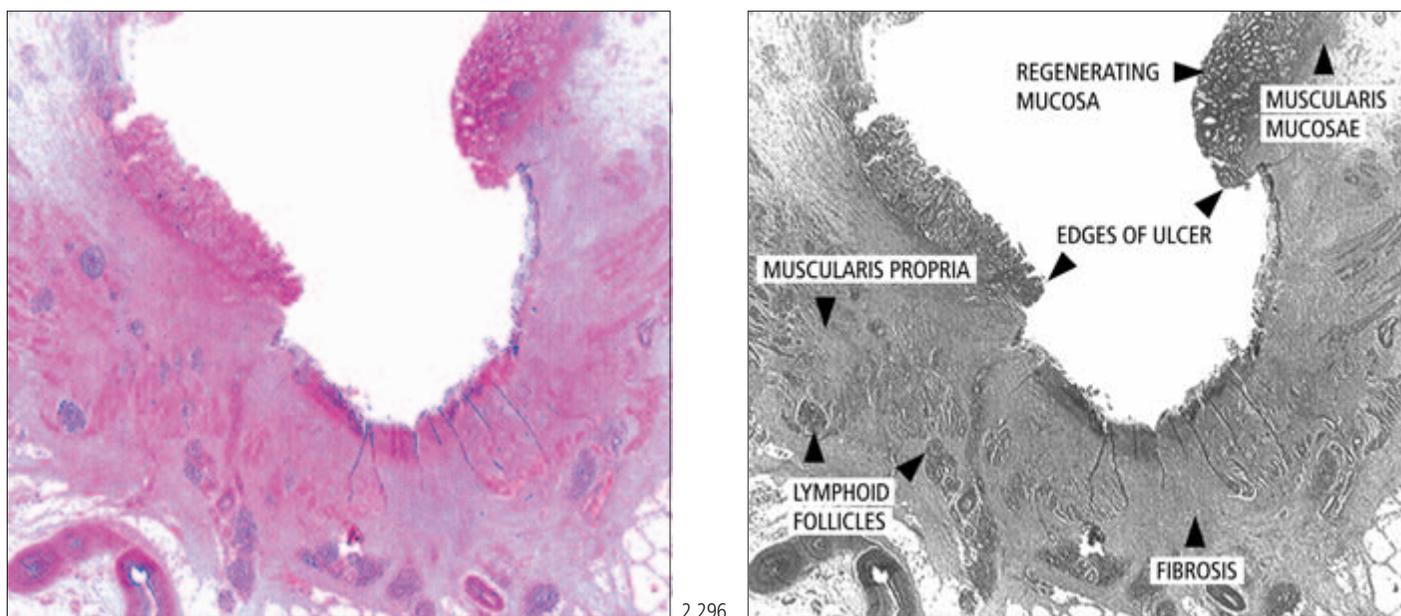
Perforation

Obtaining a clear view, measuring and sampling

Benign and malignant ulcer: similarities

### Histopathology

The typical histopathological appearances of benign gastric ulcer, irrespective of shape, size and site, are shown in Fig. 2.296.



### Variations in shape and size

As with oesophageal and duodenal ulcers, gastric ulcers come in all shapes and sizes.

Figure 2.297 illustrates the commonest appearance. The outline is only slightly irregular, and the junction between normal and ulcerated mucosa is well defined. The ulcer is shallow: the depth of the lesion can be easily appreciated by moving the tip of the endoscope and studying the slight changes in appearance. Size can be estimated in relation to

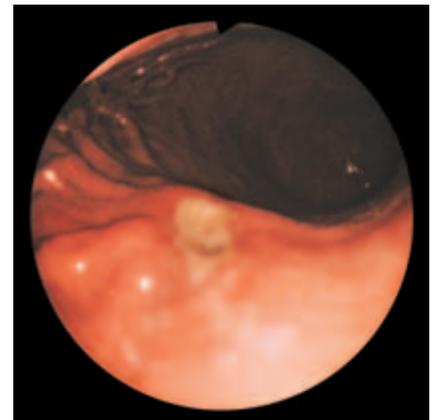
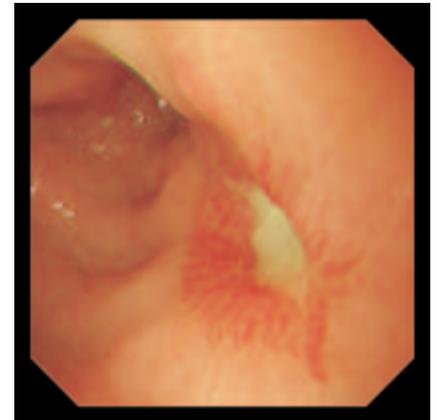
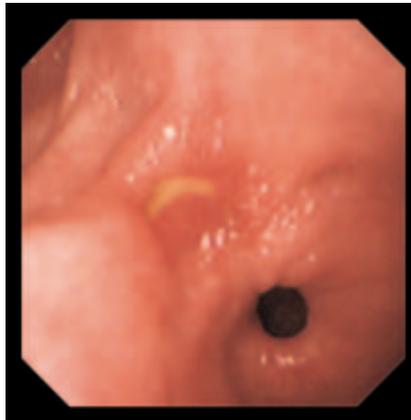


**Variations in shape and size (cont.)**

neighbouring features, in this case the pylorus, or by the use of the opened biopsy forceps. Endoscopic measuring devices (Fig. 2.298) are available.

The ulcer (or erosion) shown in Fig. 2.299 is crescentic, and demonstrates surrounding hyperaemia, also well seen in Fig. 2.300. A larger slightly irregular crescentic ulcer is seen in Fig. 2.301.

The small round ulcer on the incisura shown in Fig. 2.302 has a converging pattern of mucosal folds suggesting chronicity with underlying fibrosis.



**Variations in shape and size (cont.)**

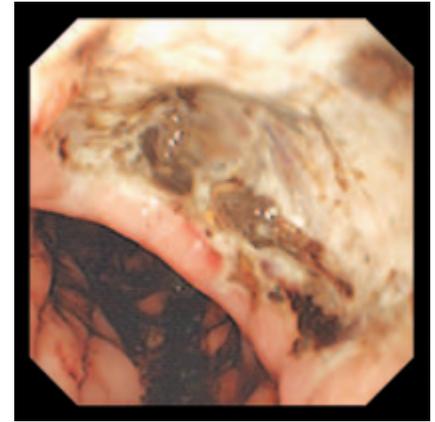
Figure 2.303 illustrates a larger deeper ulcer; the dark stains in the base are indicative of recent bleeding. The incisural ulcer appearing in Fig. 2.304 is very large. The commonly held belief, namely that the larger the gastric or duodenal ulcer the greater the chance that it is malignant is not based on fact: many 'giant' ulcers are benign.

Ulcers are sometimes quite lumpy (Fig. 2.305). While this raises the suspicion of malignancy, it may only represent an unusual degree of inflammation, or a healing stage.

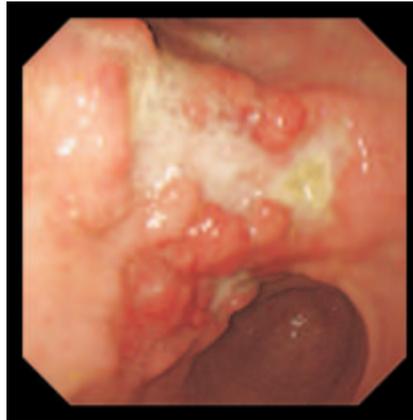
Linear ulcers, in this case just below the oesophago-gastric junction (Fig. 2.306), are less common.



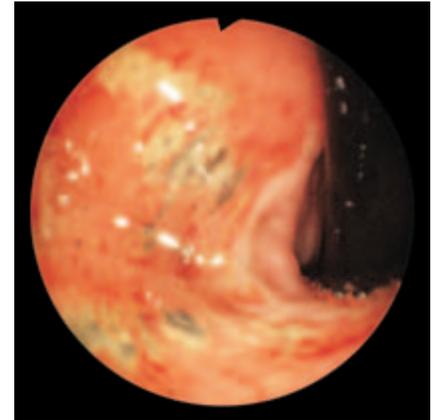
2.303



2.304



2.305



2.306

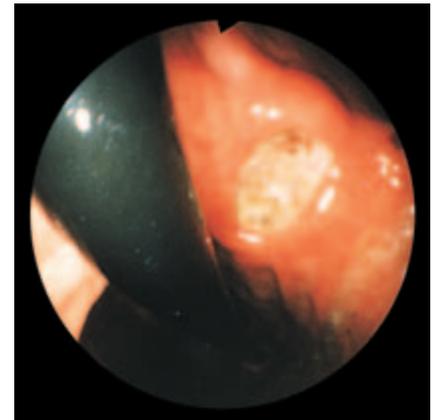
**Variations in site**

Benign gastric ulcers may occur at any site but are commonest on the incisura, on the lesser curve and in the prepyloric area.

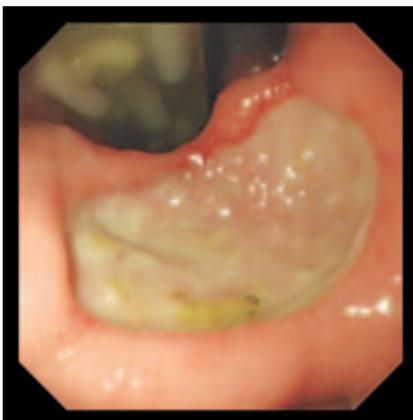
An incisural ulcer may be seen with the endoscope in the prograde position (Fig. 2.307) though inversion is sometimes necessary to get a good view (Fig. 2.308). In Fig. 2.309 another ulcer on the incisura is shown in greater detail. Inadequate distension may cause ulcer-like appearances at the incisura (Figs 2.310 and 2.311), a problem resolved on fuller insufflation of air.



2.307



2.308



2.309



2.310



2.311

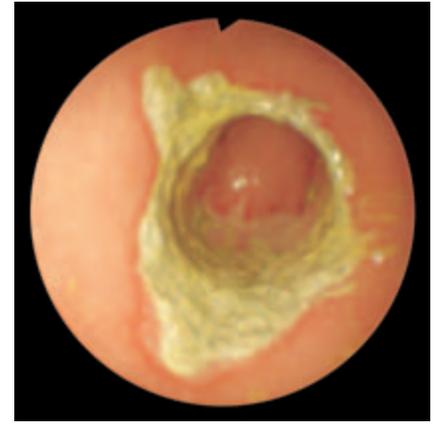
**Variations in site (cont.)**

Prepyloric ulcers are usually single and sharply circumscribed (Fig. 2.312). Rarely they may be multiple, or large and quite atypical. Figure 2.313 shows a giant circumferential benign gastric ulcer; this patient had been taking a NSAID.

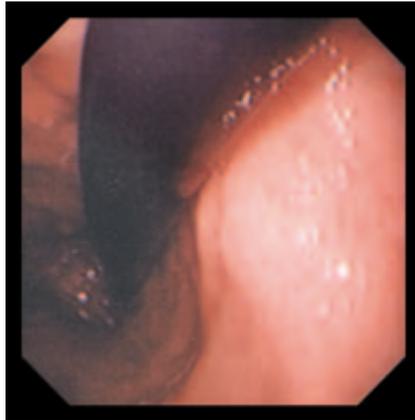
High lesser curve ulcers are often missed during introduction of the endoscope, and on inversion may be obscured by the shaft of the instrument (Fig. 2.314). Gentle rotation to enable complete inspection of the area may show such an ulcer (Fig. 2.315). Ulcers in this area may be linear, may present with bleeding (Figs 2.316 and 2.317) and simulate Mallory–Weiss tears (Fig. 2.445).



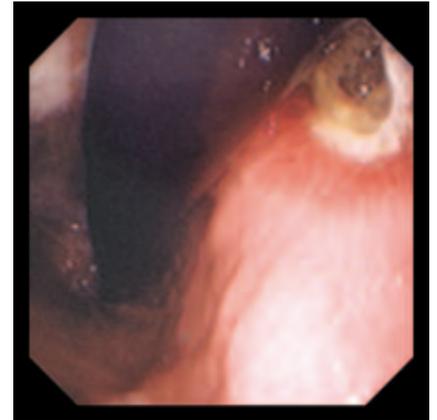
2.312



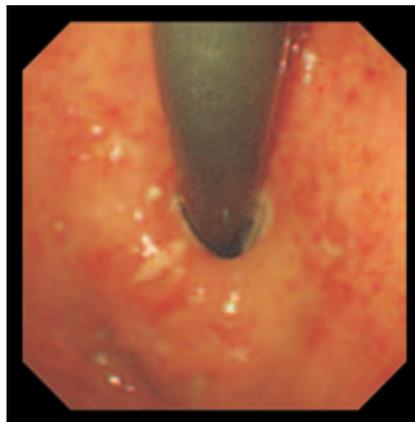
2.313



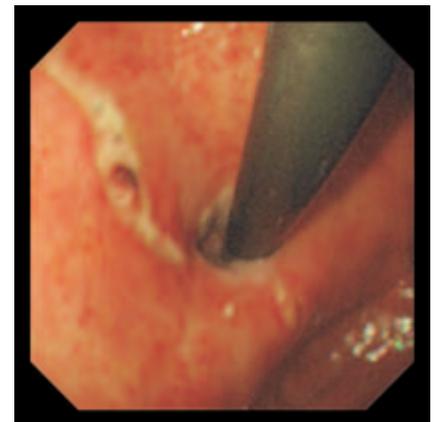
2.314



2.315



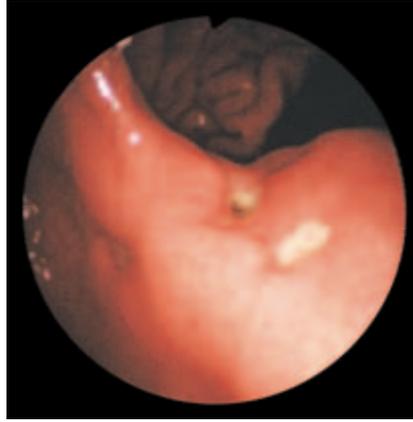
2.316



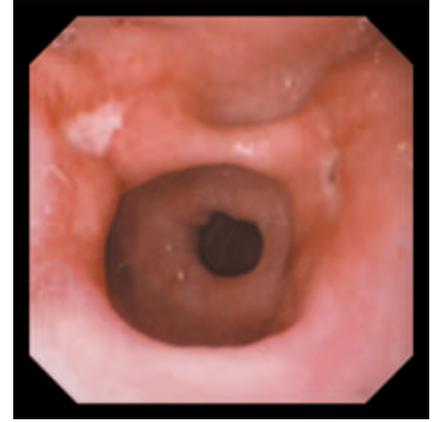
2.317

### Multiple ulcers

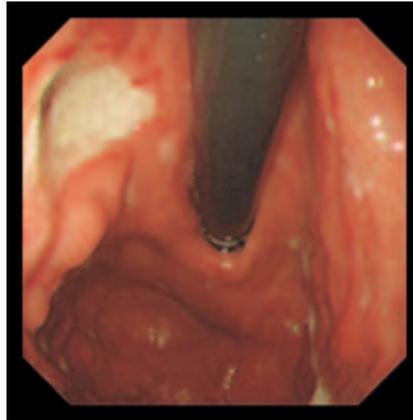
Multiple ulcers are common. Three lesions on the angulus can be seen in this photograph (Fig. 2.318), while Fig. 2.319 demonstrates two small ulcers linked by scarring. Another patient had a larger ulcer at the cardia with smaller ulcers in the body (Fig. 2.320) and a further ulcer on the incisura (Fig. 2.321).



2.318



2.319



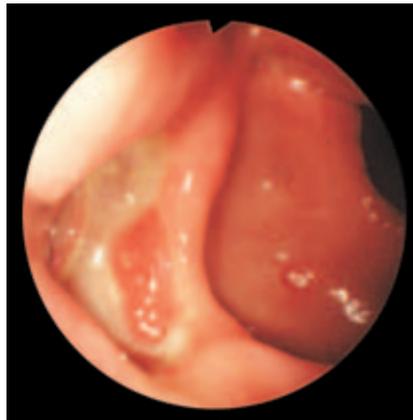
2.320



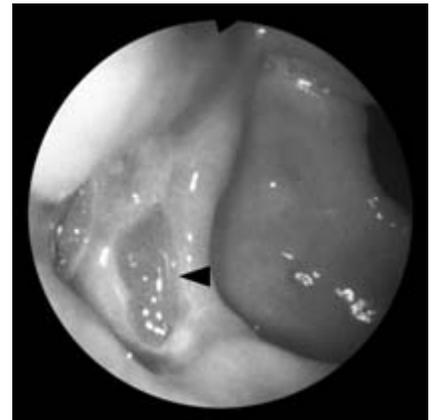
2.321

### Healing and scarring

Figure 2.322 shows granulation tissue in the base of a healing ulcer. The ulcer illustrated in Fig. 2.323 is surrounded by considerable scarring, possibly due to previous ulceration nearby. In Fig. 2.324 there is prepyloric narrowing in association with an ulcer in this position.



2.322



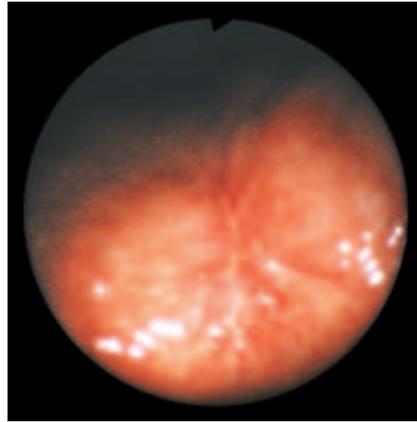
2.323



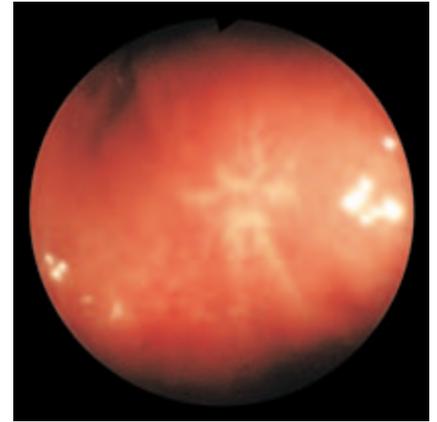
2.324

**Healing and scarring (cont.)**

During healing an ulcer may form a linear scar (Fig. 2.325). The scar may be stellate involving the mucosa only (Fig. 2.326) or leaving a converging pattern of rugae (Fig. 2.327). Occasionally there may only be superficial mottling (Fig. 2.328).



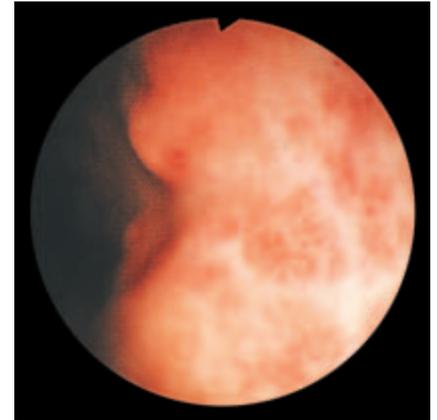
2.325



2.326



2.327

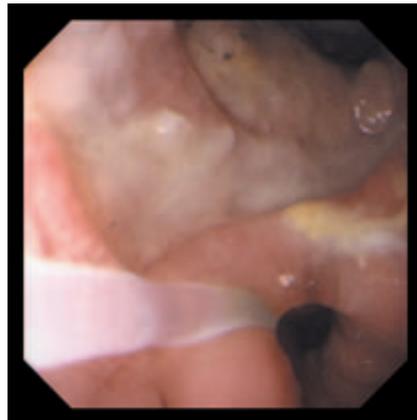


2.328

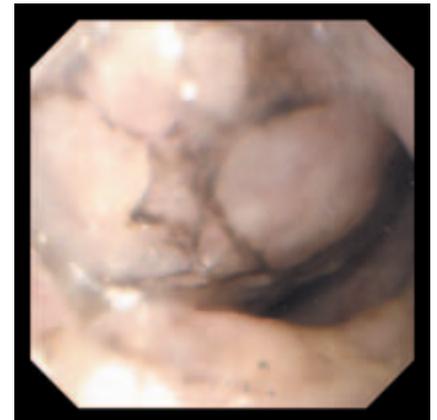
**Perforation**

Suspected or even possible perforation of a viscus is an absolute contraindication to planned endoscopy. However, the endoscopist may be inadvertently involved in this diagnosis.

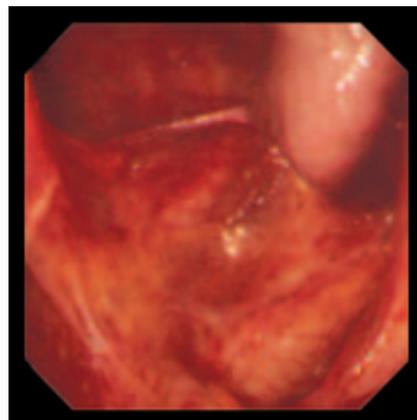
Figures 2.329 and 2.330 illustrate such a case which concerned a fit elderly patient with abdominal pain of uncertain origin. Figure 2.329 shows a large ulcerated area near the pylorus (here cannulated with a small diameter tube for identification). Exploration of the ulcer led to a deep irregular cavity biopsy which revealed granulation tissue and necrotic liver. (Though not relevant in this case, it is worth mentioning that histologically necrotic liver tissue may mimic carcinoma histologically). Figure 2.331 reveals a view more commonly obtained at laparoscopy: the patient had presented very atypically and, as with the previous patient, endoscopy seemed indicated in the search for the correct diagnosis. The endoscopist found evidence of a freely perforated gastric ulcer.



2.329



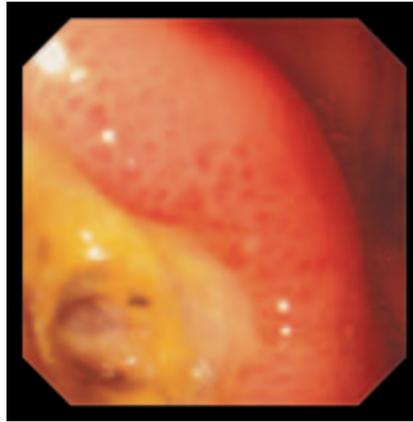
2.330



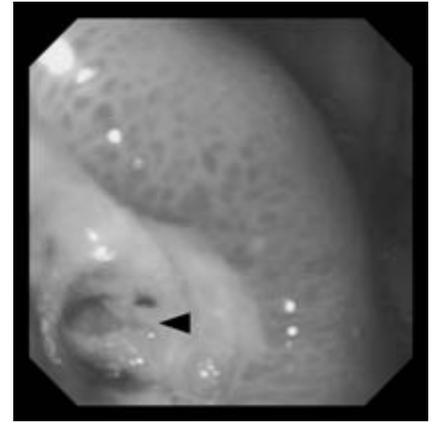
2.331

**Perforation (cont.)**

In contrast, Fig. 2.332 shows a perforated ulcer, in another patient, with prolapsed small bowel occluding the perforation site.



2.332



**Obtaining a clear view, measuring and sampling**

These are common problems which apply to endoscopy of any part of the gastrointestinal tract. It may be appropriate to discuss them here, with reference to gastric ulcers.

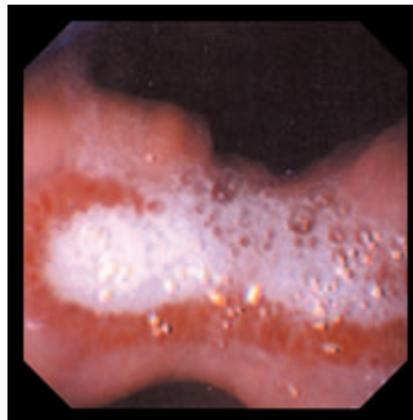
Antifoaming preparations are often helpful, and some endoscopists routinely ask patients to swallow an aliquot before endoscopy.

Saliva or froth may obscure detail. Figure 2.333 shows an incisural ulcer largely covered by saliva.

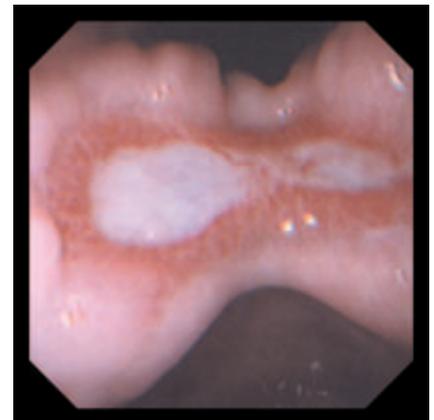
After spraying with foam dispersant excellent views were obtained (Fig. 2.334). Antifoam may be impelled directly through the biopsy channel of the endoscope, or via a tube with spray tip, passed through the biopsy channel.

Measuring devices (Fig. 2.298) may be used to assess the size of a lesion (Fig. 2.335), and multiple biopsies (Fig. 2.336) and cytological brushings (Fig. 2.337) can be taken.

When an apparently benign gastric ulcer is found, it is essential to exclude malignancy before medical treatment is commenced. The ulcer is first inspected visually. If required cytological samples, using a sheathed brush passed through the biopsy channel of the endoscope, are best obtained if the brush can be slid tangentially across the rim and bed of the ulcer. Some cytology brushes are rotatable within the sheath permitting very accurate sampling.



2.333



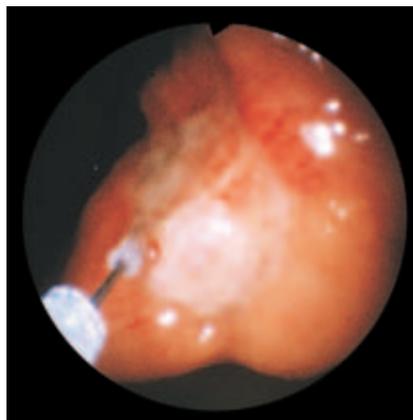
2.334



2.335



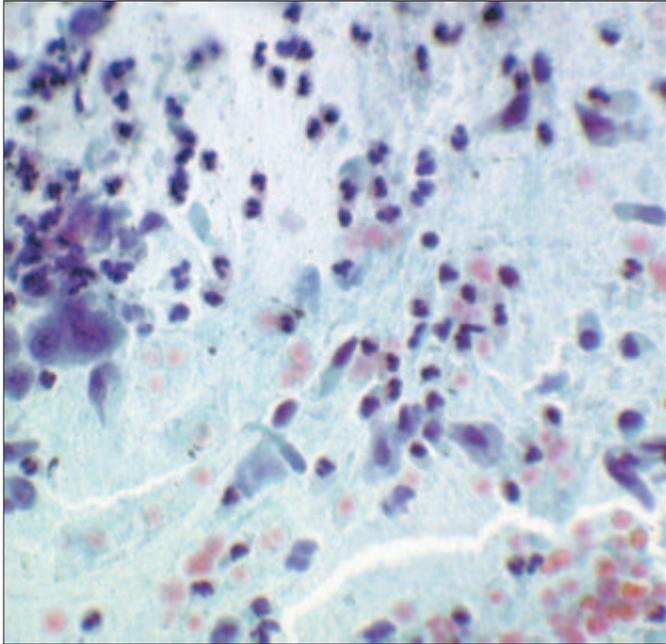
2.336



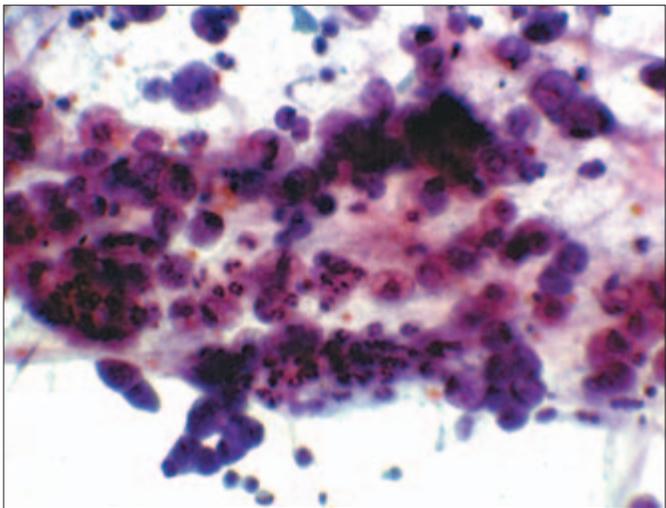
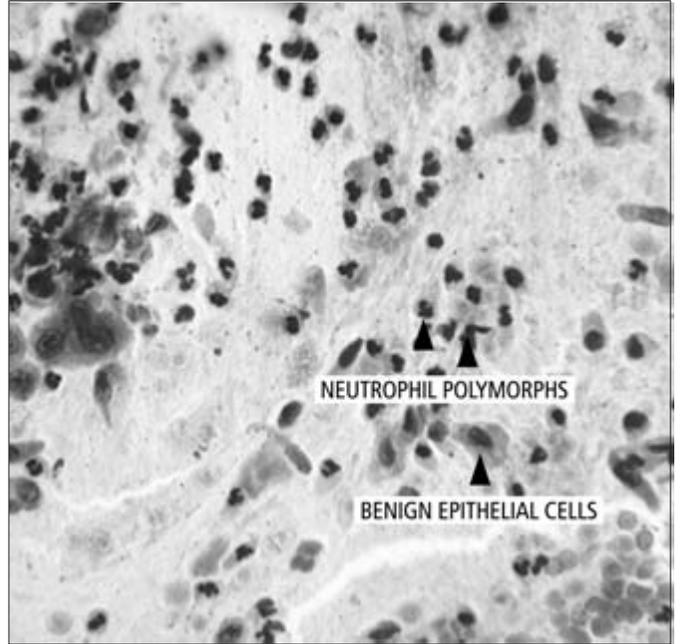
2.337

**Obtaining a clear view, measuring and sampling (cont.)**

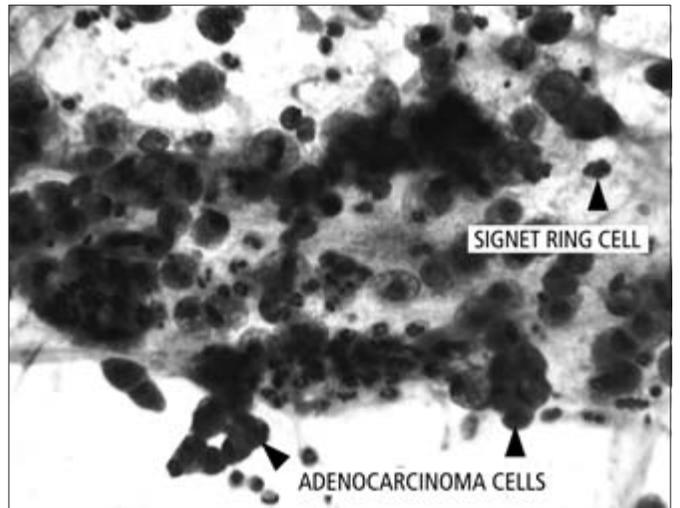
The next two figures illustrate brush cytological appearances in benign gastric ulcer (Fig. 2.338) and in gastric adenocarcinoma (Fig. 2.339). Multiple target biopsies should be taken from different quadrants of the rim and base (see also Fig. 2.470). For these manoeuvres it may be necessary to alter the position of the endoscope, or occasionally the patient. Healing of the ulcer should be checked by endoscopy after 4–8 weeks of medical treatment.



2.338

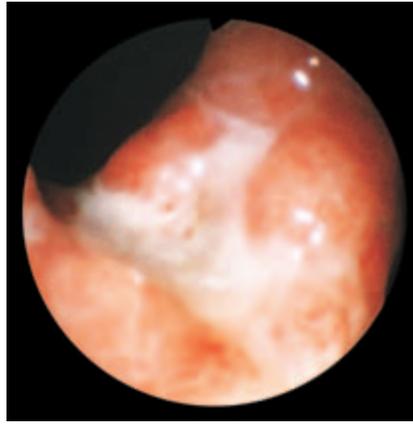


2.339

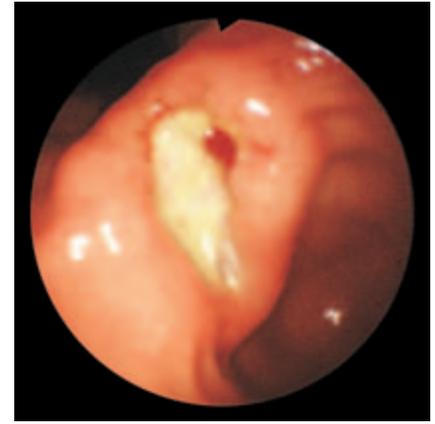


### Benign and malignant ulcer: similarities

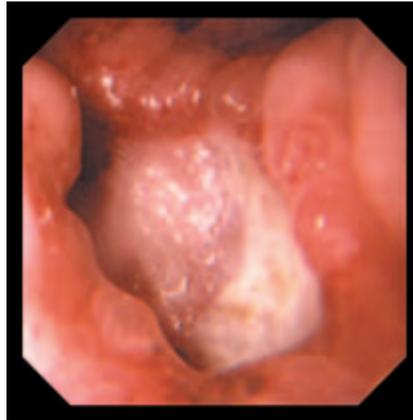
Variations in the appearance of benign gastric ulcer have been illustrated previously (Figs 2.297–2.331). In concluding this section on peptic ulcer, the occasional similarities between benign and malignant ulcers are emphasized. Lumpy irregular ulcers, e.g. Fig. 2.340, may on full assessment turn out to be benign. The benign looking ulcer shown in Fig. 2.405 was a lymphoma. The ulcer shown in Fig. 2.341 looked benign, but closer inspection revealed that the underlying tissue was standing slightly proud. In Fig. 2.342 the ulcer initially looked benign, but the irregular rolled and beaded edge suggested malignancy. In both cases carcinoma was confirmed. In Fig. 2.343 the ulcer itself looks benign. However, on review it is clear that it lies in a linear, irregular raised wedge of hyperaemic tissue; biopsies confirmed carcinoma. The outline of the ulcer shown in Fig. 2.344 was regular as in benign ulcers but it is very unusual for a benign ulcer to be so deep; biopsies confirmed carcinoma.



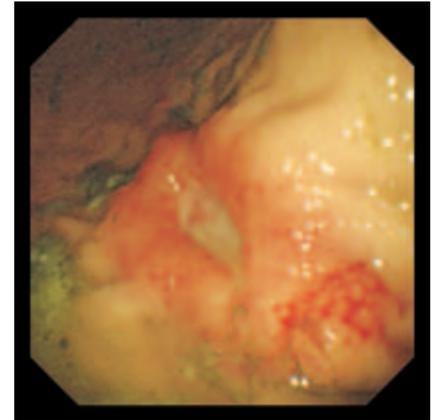
2.340



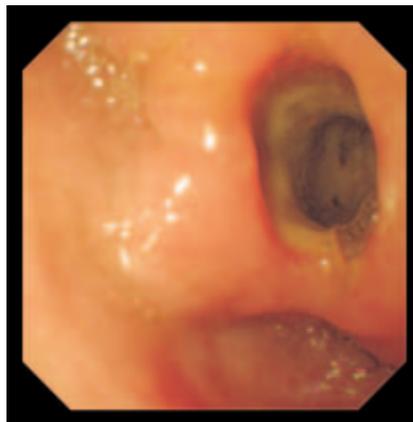
2.341



2.342



2.343



2.344

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## Benign and malignant tumours

### Classification

Nonspecific appearances of polypoid lesions

Hyperplastic (regenerative) polyps

Adenomas

Familial adenomatous polyposis (FAP)

Peutz–Jeghers polyps

Cronkhite–Canada syndrome

Dysplasia

Early gastric cancer (EGC)

Advanced cancer

Linitis plastica

Ulcer cancers

Endocrine cell tumours

Secondary carcinoma

Invading extra-gastric carcinoma

Lymphoma

Benign inflammatory fibroid polyp

Lipomas

Gastrointestinal stromal tumours (GISTs)

Kaposi's sarcoma

Inflammatory cap polyp

### Classification

Benign and malignant tumours of the stomach are common and to the endoscopist many may be indistinguishable. Classification is most usefully approached from a pathologist's standpoint as for example in Tables 2.5–2.7. The endoscopic appearances of various lesions, with related pathology as appropriate, will be shown in the order arising from these tables.

**Table 2.5** Benign epithelial tumours and polyps of the stomach (adapted from Day *et al.*, 1990)

#### Hyperplastic (regenerative) polyps

#### Neoplastic polyps

Adenomas

Familial adenomatous polyposis syndromes

#### Hamartomatous polyps

Peutz–Jeghers syndrome

Juvenile polyposis syndrome

Cronkhite–Canada syndrome

Fundic glandular cysts

**Table 2.6** Malignant epithelial tumours of the stomach (adapted from Day *et al.*, 1990)

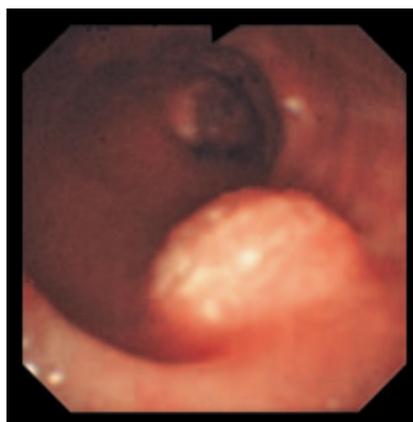
<p><b>Precancerous lesions and conditions</b></p> <ul style="list-style-type: none"> <li>Pernicious anemia</li> <li>The operated stomach</li> <li>Atrophic gastritis, intestinal metaplasia and epithelial dysplasia</li> </ul> <p><b>Early gastric cancer</b></p> <p><b>Advanced cancer</b></p> <ul style="list-style-type: none"> <li>Intestinal-type adenocarcinoma</li> <li>Diffuse-type adenocarcinoma</li> <li>Mixed-type adenocarcinoma</li> </ul> <p><b>Ulcer cancer</b></p> <p><b>Other types of primary epithelial tumour</b></p> <ul style="list-style-type: none"> <li>Endocrine cell tumours</li> <li>Others</li> </ul> <p><b>Secondary and direct spread carcinoma of the stomach</b></p>
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**Table 2.7** Non-epithelial tumours of the stomach (adapted from Day *et al.*, 1990)

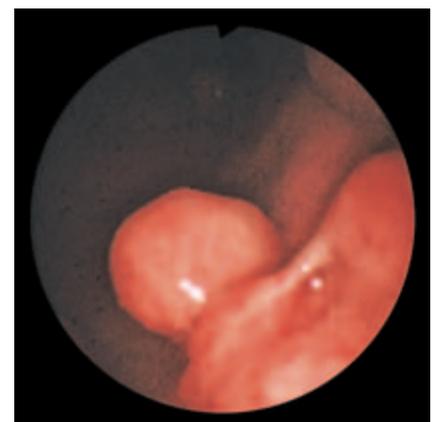
<p><b>Tumours of lymphoid tissue</b></p> <ul style="list-style-type: none"> <li>Focal lymphoid hyperplasia</li> <li>Lymphoma</li> <li>Leukaemia</li> </ul> <p><b>Inflammatory fibroid polyp</b></p> <p><b>Lipoma</b></p> <p><b>Gastrointestinal stromal tumours (GISTs)</b></p> <p><b>Kaposi's sarcoma</b></p> <p><b>Other non-epithelial tumours</b></p> <ul style="list-style-type: none"> <li>Inflammatory cap polyp</li> <li>Others</li> </ul>
--

**Nonspecific appearances of polypoid lesions**

The lesions illustrated here are endoscopically similar. In the event, Fig. 2.345 was an antral carcinoma and Fig. 2.346 a 'reactive' polyp.



2.345



2.346

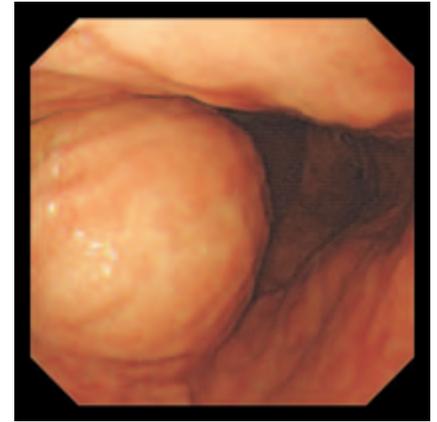
**Nonspecific appearances of polypoid lesions (cont.)**

Figure 2.347 was a prepyloric adenomatous polyp. This emphasizes the need to obtain material for histopathological evaluation: snare biopsies are more informative than forceps biopsies, but the risk of perforation following the use of a snare is believed to be greater than in the colon.

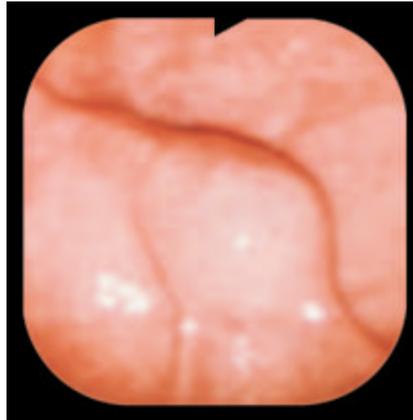
When a lesion is submucosal, the appearances are often even less specific. The nature of the lesions shown in Figs 2.348 and 2.349 is unknown. Figure 2.349 shows the bridging folds frequently seen in association with submucosal masses.



2.347



2.348



2.349

**Hyperplastic (regenerative) polyps**

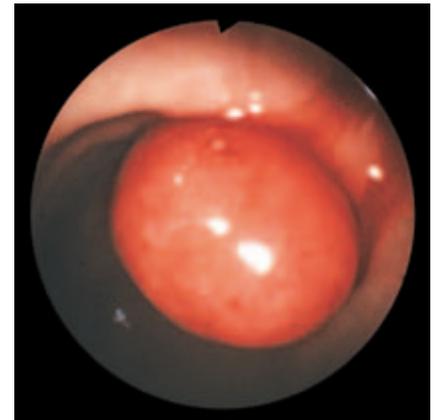
These are usually small (less than 1 cm), multiple and widely scattered throughout the stomach, often sparing the antrum (Fig. 2.350). They are normally a chance finding as they cause no symptoms. They have no malignant potential. Fundic glandular cysts (Fig. 2.366) are commonly coexistent and have a similar endoscopic appearance.

Single or larger hyperplastic polyps do occur. Figure 2.351 illustrates an example of such a polyp. The 2 cm hyperplastic polyp shown in Fig. 2.352 initially was discovered prolapsed through the pylorus into the duodenal cap. It was withdrawn to the antrum with biopsy forceps (the cause of bleeding) and found to be on a narrow stalk. Polypectomy was performed with diathermy snare.

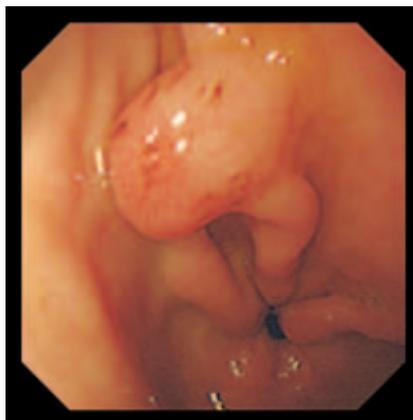
Figure 2.353 shows an irregular polyp which was successfully removed. Surprisingly, it was found to be regenerative, underlining the fact that appearances may be of little help in accurate diagnosis.



2.350



2.351



2.352



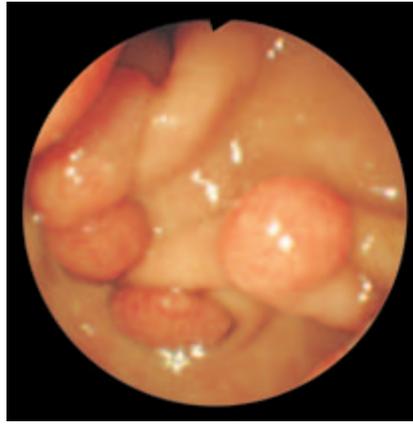
2.353

**Hyperplastic (regenerative) polyps (cont.)**

Rarely, hyperplastic polyps may appear following successful laser treatment of antral haemangiomas (see Fig. 2.427 and p. 217).

This patient developed multiple small hyperplastic polyps (Fig. 2.354) and a large pedunculated irregular hyperplastic polyp (Fig. 2.355) which caused gastric outlet obstruction and was successfully snared.

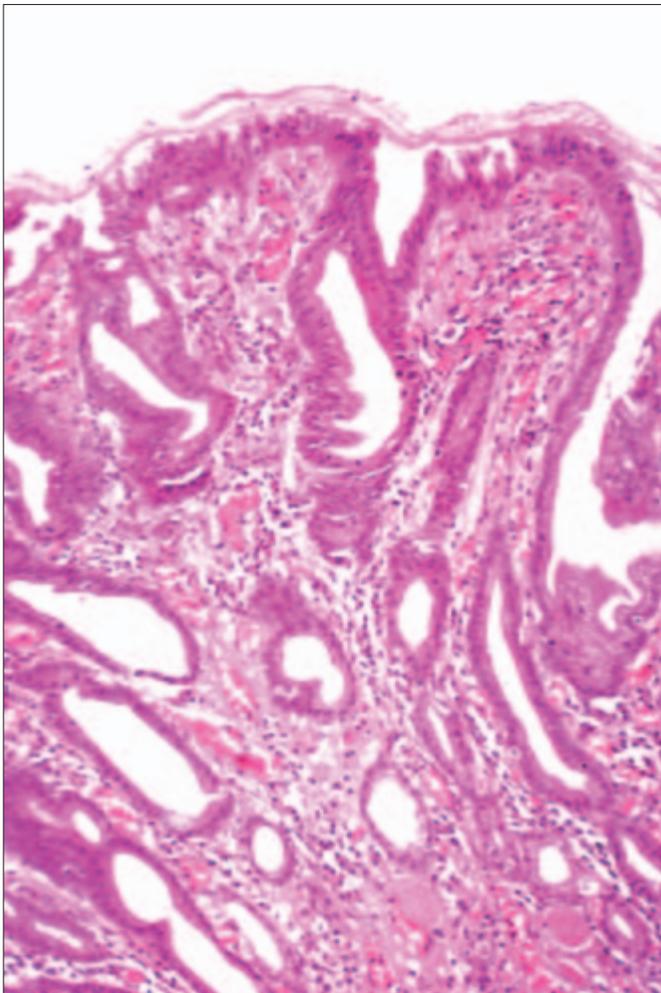
The histological features of hyperplastic polyps appear in Fig. 2.356.



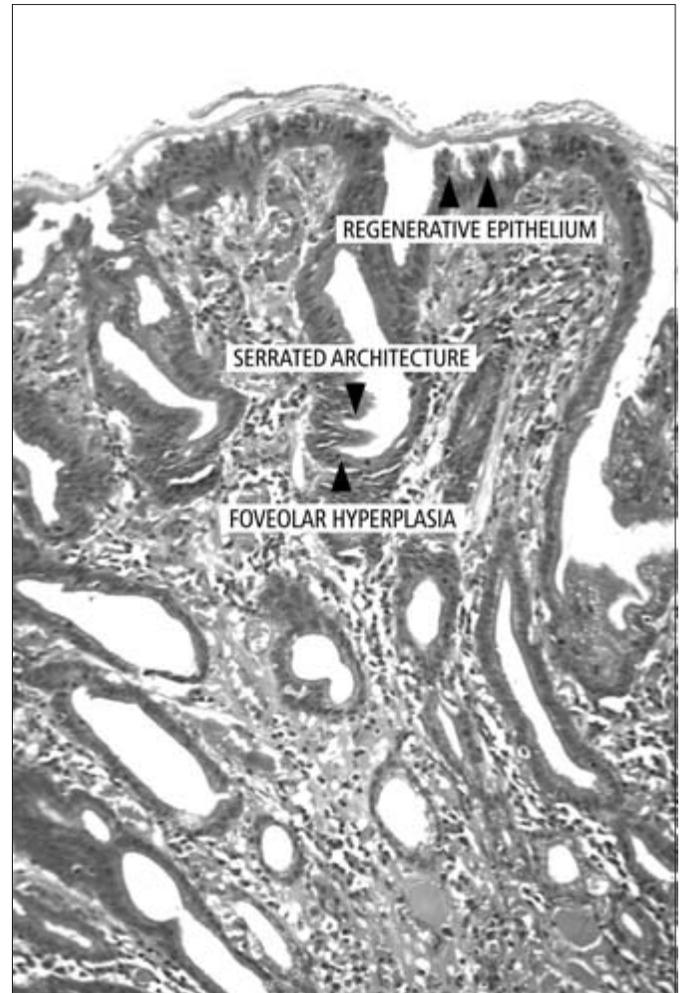
2.354



2.355



2.356



**Adenomas**

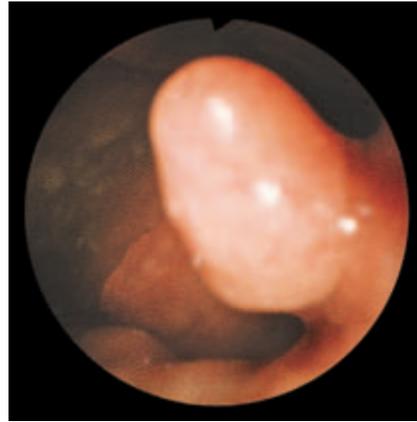
These tend to be single (Fig. 2.357) and may be pedunculated. It is also common for the head of the polyp to differ slightly in colour from the stalk and surrounding mucosa. The polyp illustrated in Fig. 2.358 is redder, and that in Fig. 2.359 paler, than the surrounding mucosa. The surface is usually smooth, but may be irregular: this patient had four polyps, two of which are visible in this figure (Fig. 2.360). Surface ulceration sometimes occurs. The histopathology of gastric adenoma is shown in Fig. 2.361.



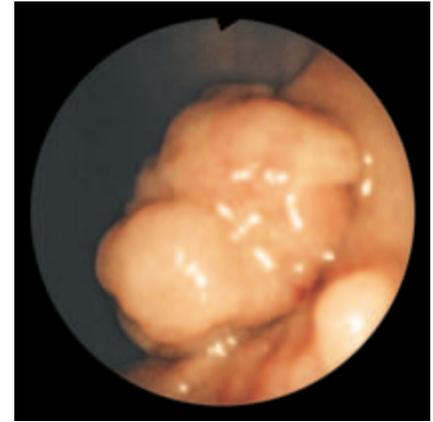
2.357



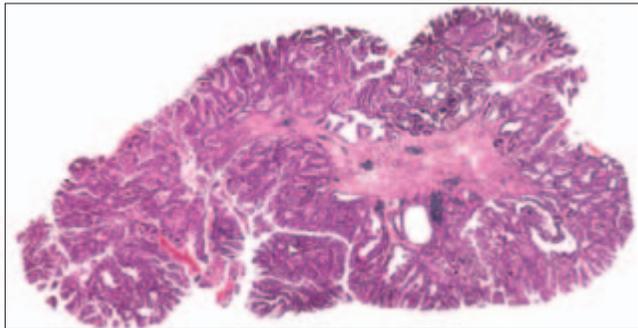
2.358



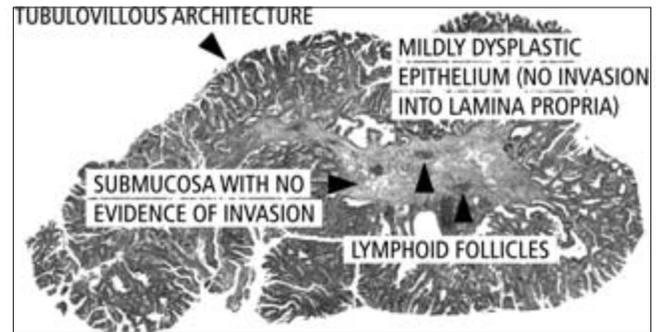
2.359



2.360



2.361



Gastric adenomatous polyps, like the much commoner colonic adenomas, may undergo malignant change. The polyp appearing in Fig. 2.362 showed severe dysplasia amounting to adenocarcinoma while in Fig. 2.363 there is invasive malignancy as a complication of adenoma. (The adenoma–carcinoma sequence is discussed more fully on p. 254.)



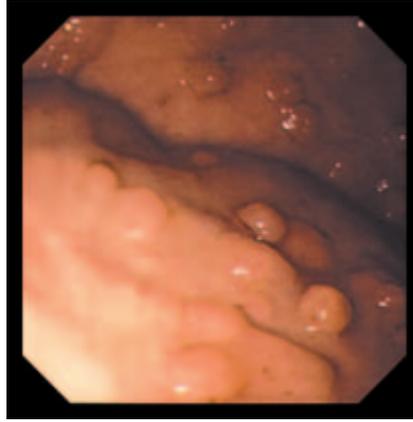
2.362



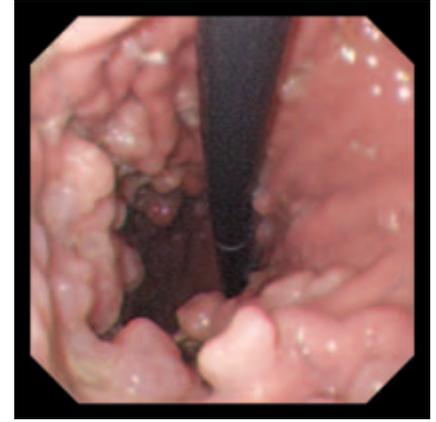
2.363

**Familial adenomatous polyposis (FAP)**

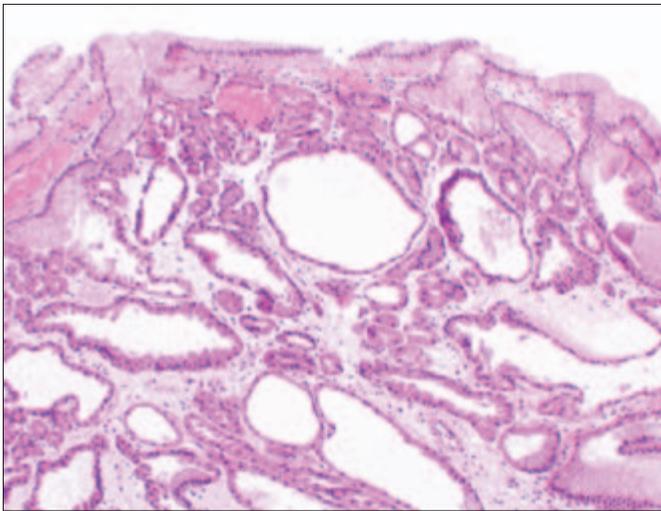
Multiple gastric lesions (Figs 2.364 and 2.365), duodenal (Fig. 2.525) and colonic (Figs 3.113–3.115) adenomatous polyps may occur in patients with familial adenomatous polyposis (FAP). The gastric lesions in FAP may be adenomatous polyps but are usually multiple fundic gland cysts; the endoscopic appearances are similar. These lesions have simple cystic dilatation of gastric glands in the absence of hyperplasia or dysplasia (Fig. 2.366).



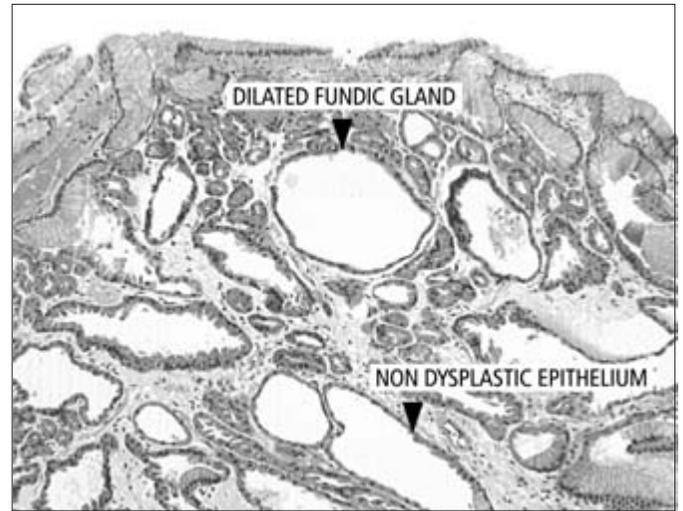
2.364



2.365

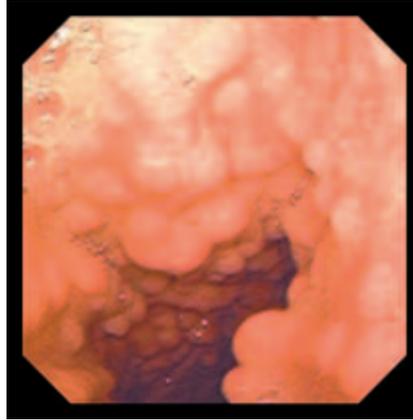


2.366



**Peutz–Jeghers polyps**

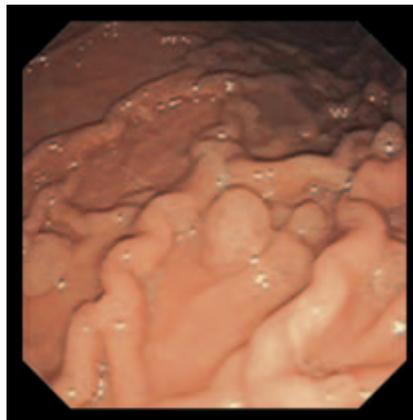
Polyps occurring in this syndrome may be single or multiple (Fig. 2.367); they have no pathognomonic endoscopic features. Histological appearances are shown in Figs 2.535 and 3.132.



2.367

**Cronkhite–Canada syndrome**

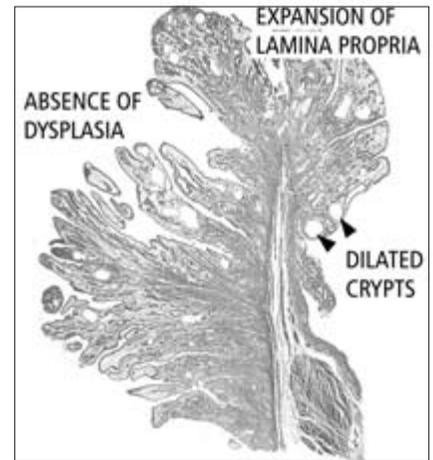
Polypoid lesions may be found in the stomach (Fig. 2.368) and less commonly elsewhere in the digestive tract (Fig. 3.133). There are no specific endoscopic features. The histological appearances (Fig. 2.369) are like those of juvenile polyps (Figs 3.125 and 3.126). Cronkhite–Canada lesions are distinguished by having similar glandular changes in non-polypoid flat mucosa. This condition, like FAP, exemplifies the need to take biopsies from flat mucosa, not just of the polyp.



2.368



2.369



## Dysplasia

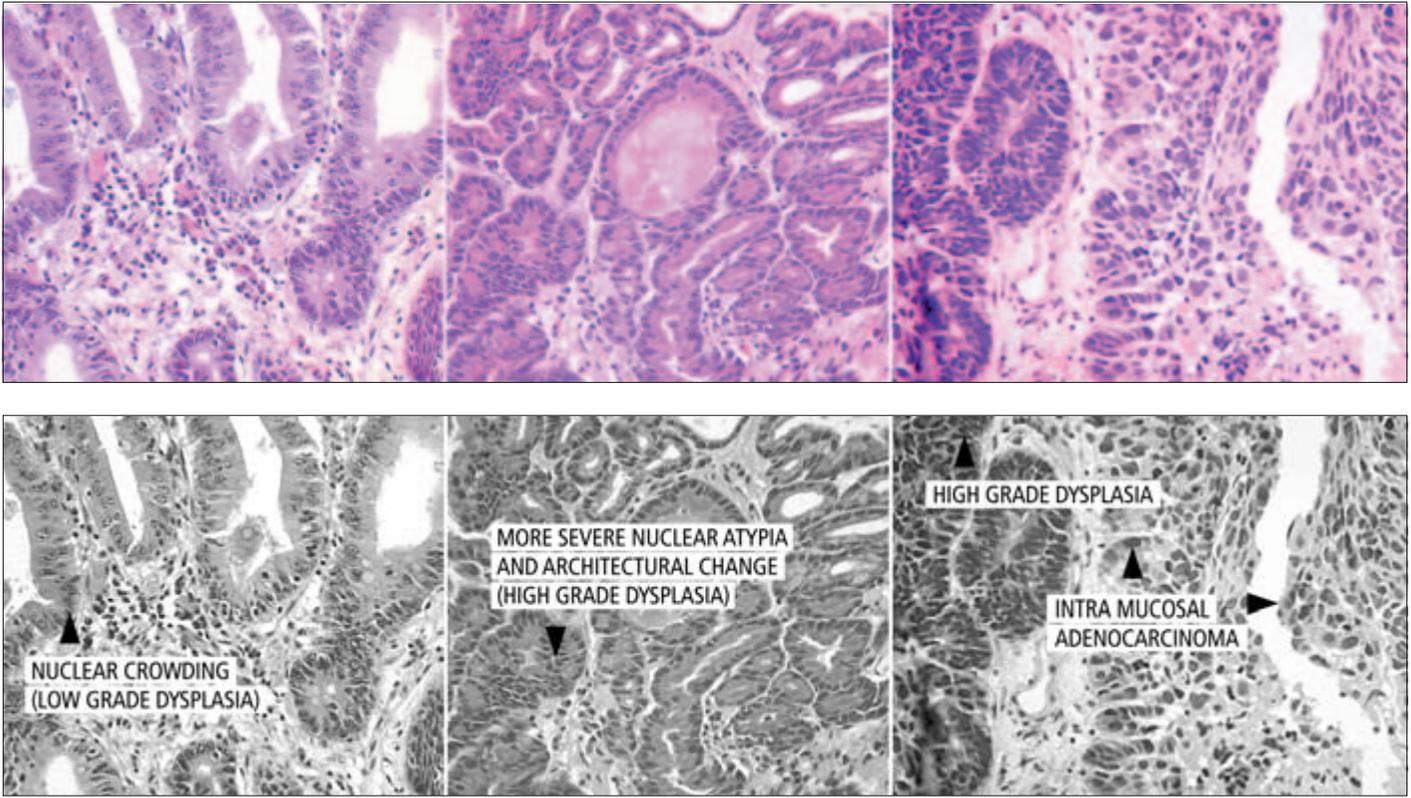
Dysplastic gastric mucosa cannot be recognized endoscopically. Dysplasia is, however, of great importance as it is a stage in carcinogenesis, and in the stomach may arise *de novo* or as a complication of chronic gastritis. Dysplasia has previously been mentioned in connection with Barrett's oesophagus (p. 57). The Vienna classification was also referred to *en passant*. The classification of gastrointestinal epithelial neoplasia/dysplasia has been controversial. While this new classification essentially addresses gastric dysplasia and neoplasia, it was hoped that it could be applied throughout the gastrointestinal tract to include squamous oesophagus, Barrett's oesophagus, stomach, small bowel and large bowel. It is presented in Table 2.8 in slightly modified form. Category 2 is of particular importance. The pathologist may be unable to confirm or deny the presence of dysplasia for one of the following reasons: the biopsy may be inadequate for assessment due to orientation, size, crushing or fixation artefacts; extreme regenerative changes in the face of severe inflammation in *H. pylori* gastritis may mimic dysplasia; and regenerative changes seen in the foveolar hyperplasia of chemical gastritis may also mimic dysplasia.

**Table 2.8** Classification of gastric epithelial neoplasia (adapted from Lauwers and Riddell, 1999): the Vienna classification

CATEGORY 1
Negative for neoplasia/dysplasia
CATEGORY 2
Indefinite for neoplasia/dysplasia
CATEGORY 3
Non-invasive neoplasia low grade (low grade adenoma/dysplasia)
CATEGORY 4
Non-invasive neoplasia high grade
4.1 High grade adenoma/dysplasia
4.2 Non-invasive carcinoma (carcinoma <i>in situ</i> )
4.3 Suspicious for invasive carcinoma
CATEGORY 5
Invasive neoplasia
5.1 Intramucosal carcinoma
5.2 Submucosal carcinoma or beyond

**Dysplasia (cont.)**

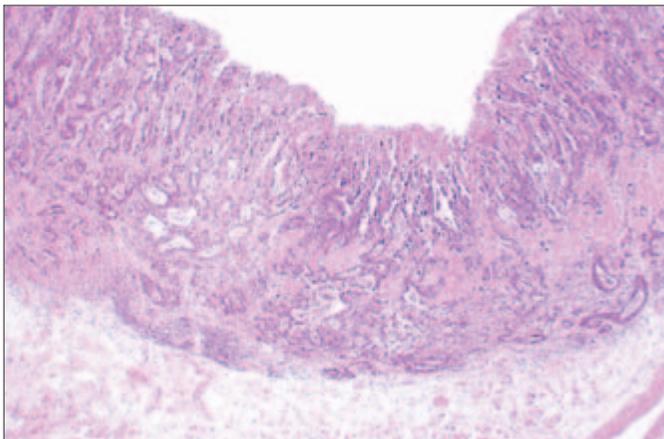
Figure 2.370 illustrates the histopathological appearances of dysplasia in gastric mucosa.



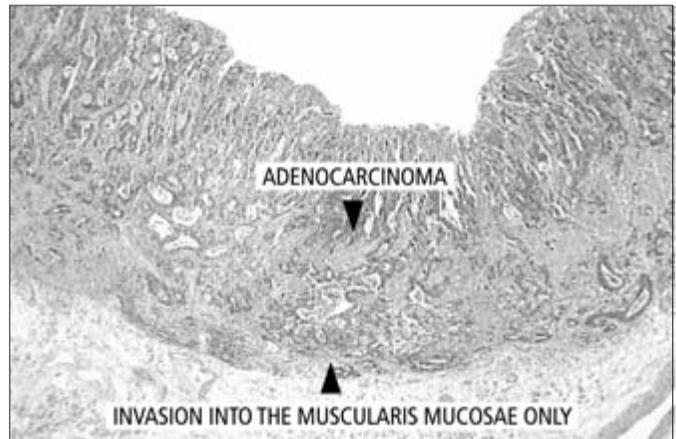
2.370

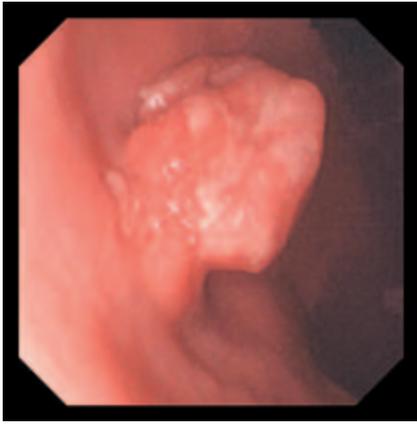
**Early gastric cancer (EGC)**

This is defined as adenocarcinoma limited to the mucosa, or the mucosa and sub-mucosa (Fig. 2.371), with or without lymph node involvement. There is a Japanese classification of EGC based on endoscopic appearances, and some evidence that prognosis may be related to endoscopic subtype. In contradistinction to advanced gastric cancer, EGC in general has an excellent 5-year survival following resection, even where there are nodal metastases at the time of surgery. EGC is not commonly found in the West, largely because there are no specific symptoms, and the relatively low incidence of gastric carcinoma makes screening programmes uneconomic. The histological subtypes are the same as in advanced gastric cancer (Fig. 2.376).



2.371

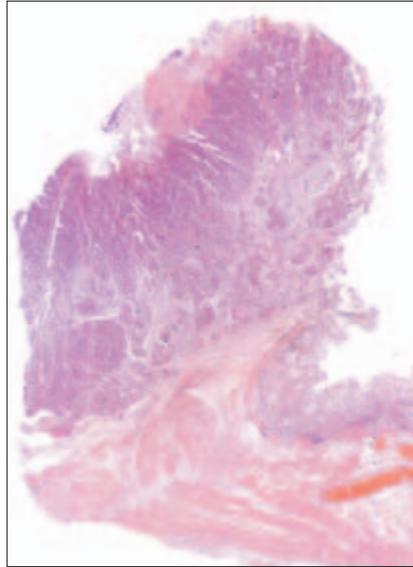




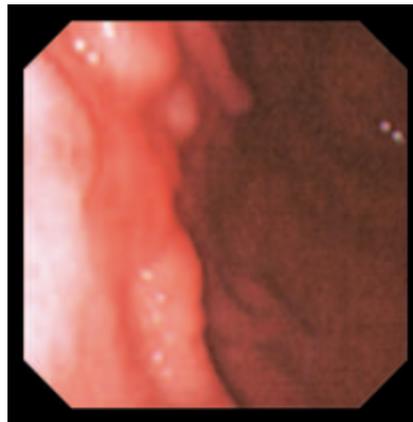
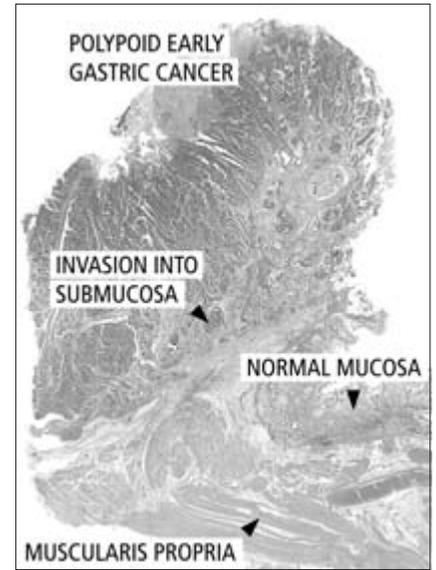
2.372

**Early gastric cancer (EGC) (cont.)**

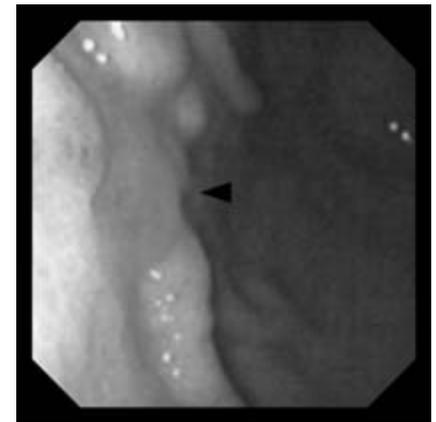
The lesion shown in Fig. 2.372 looked malignant. Histological examination of the operative specimen revealed invasion into but not below the submucosa characterizing it as an 'early' gastric cancer. Figure 2.373 illustrates the histopathological appearances in a patient with a similar tumour. A more diffuse lesion, subsequently shown to be an intramucosal signet ring cell carcinoma appears in Fig. 2.374. Figure 2.375 illustrates a small stellate antral abnormality at first erroneously suggestive of a scar; the excised specimen confirmed early gastric adenocarcinoma.



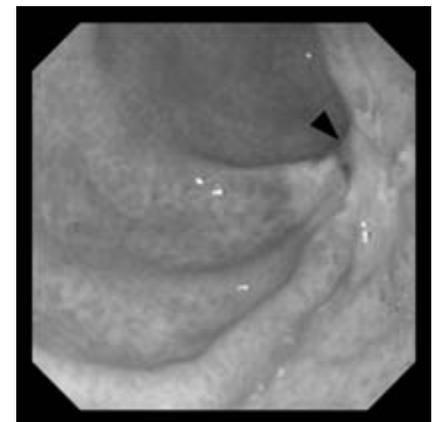
2.373



2.374



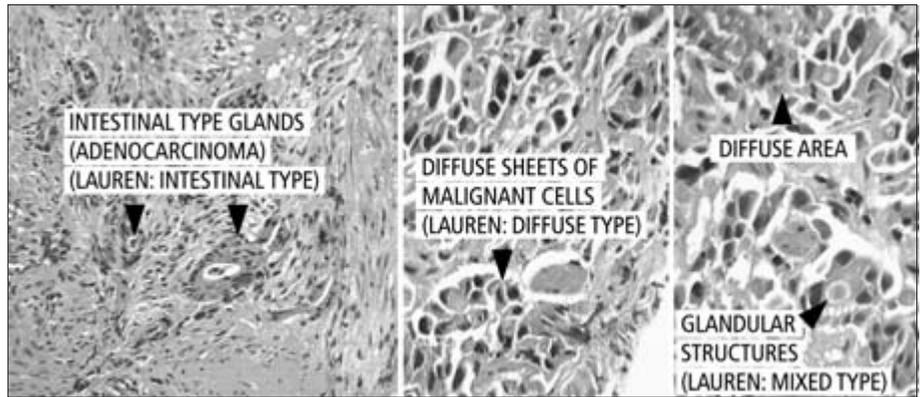
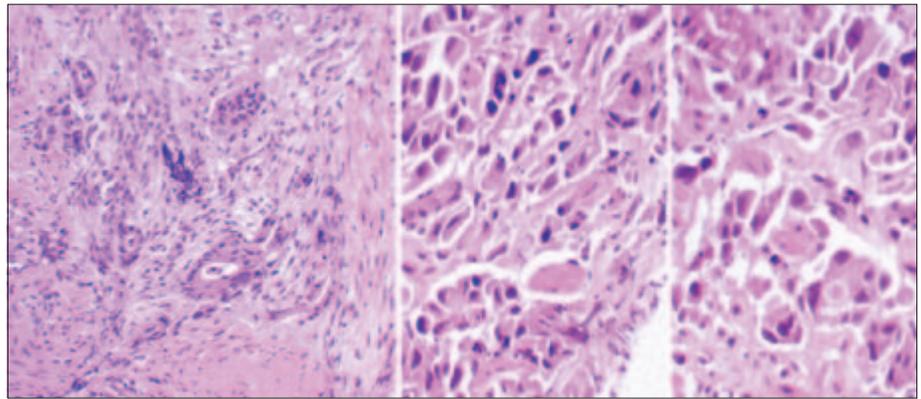
2.375



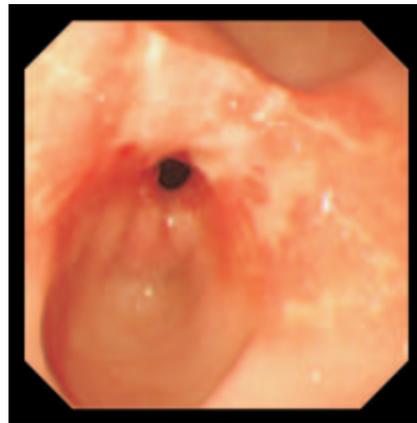
**Advanced cancer**

There is great variation in the incidence of gastric carcinoma in different parts of the world, and it is declining in the West. Gastric carcinoma may occur at any site and assume a multitude of appearances. Lesions may be flat, polypoid, ulcerated, obstructing, localized or spreading widely, submucosally infiltrating, or any combination of these. According to the Lauren classification there are intestinal, diffuse (including signet ring cell) and mixed type (Table 2.6 and Fig. 2.376).

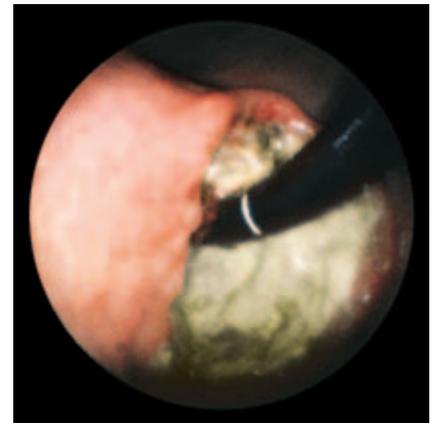
Figure 2.377 shows superficial ulceration of an antral carcinoma, while Fig. 2.378 illustrates a deeply ulcerated fundal carcinoma with lower oesophageal involvement. An ulcerated polypoid lesion is shown in Fig. 2.379 while Fig. 2.380 shows another polypoid carcinoma with less ulceration.



2.376



2.377



2.378



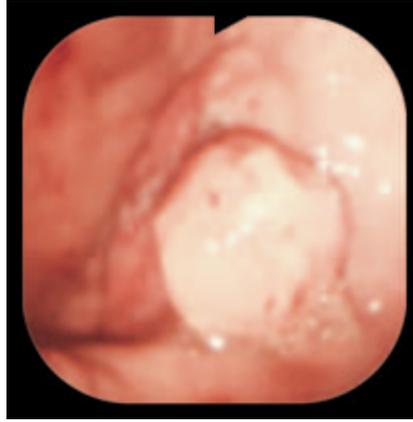
2.379



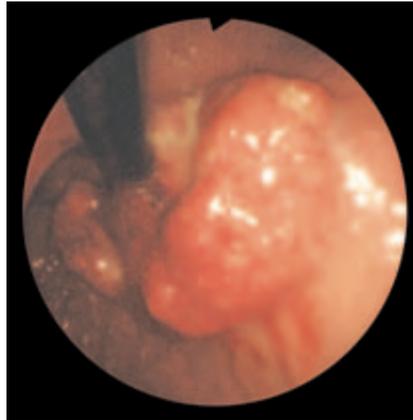
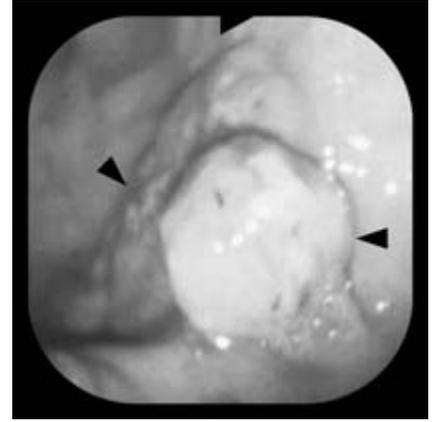
2.380

**Advanced cancer (cont.)**

A polypoid growth may assume almost any shape. Figure 2.381 shows an *en plaque* lesion with a superimposed round nodule. The polypoid lesion seen on inversion in Fig. 2.382 appears to be sharply localized with a reddened irregular surface while Fig. 2.383 shows a nonulcerated obstructing antral carcinoma. An extensive ulcerated fungating partially obstructing carcinoma of the body appears in Fig. 2.384.



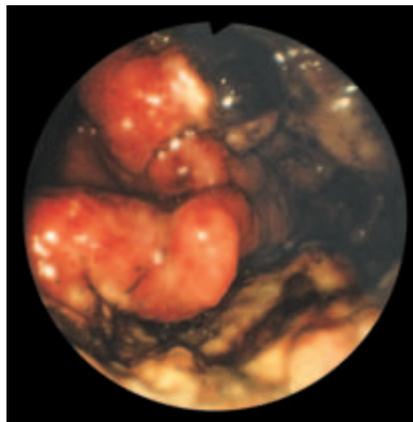
2.381



2.382



2.383

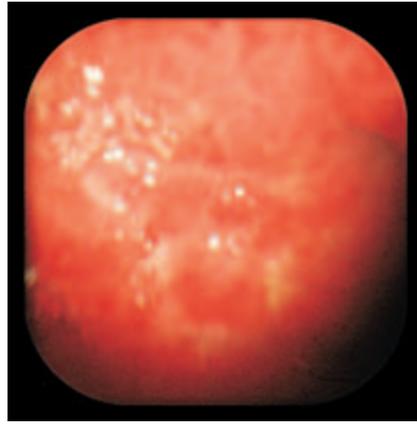


2.384

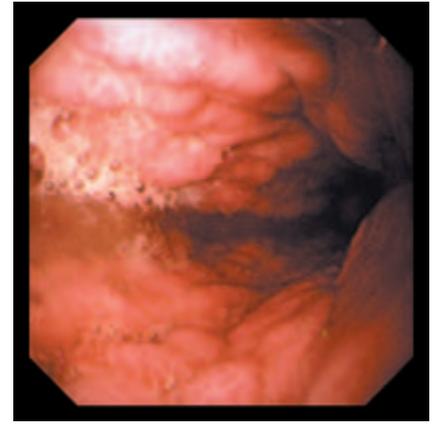
### Linitis plastica

This is not a common condition. The endoscopist may suspect linitis plastica when there is lack of distensibility of the stomach and the folds cannot be flattened by air insufflation. It is however, essential to know that the air channel of the endoscope is not blocked, as poor air flow may give the mistaken impression of poor distensibility. The endoscopic appearances are nonspecific. In Fig. 2.385 the mucosa is irregular and hyperaemic. Figure 2.386 shows the gastric mucosa thrown into irregular folds by submucosal tumour invasion, while the thickened folds in Fig. 2.387 are breached by white malignant tissue. Similar appearances are shown in Fig. 2.388. The stomach was diffusely infiltrated by an adenocarcinoma which in places was breaking through the surface in the form of pale, rice-like lesions.

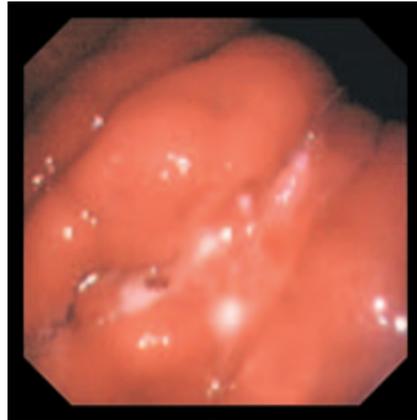
As the characteristic mode of spread is through the deeper layers of the gastric wall (Fig. 2.389), superficial mucosal biopsy is often unhelpful, unless malignant tissue has reached the surface.



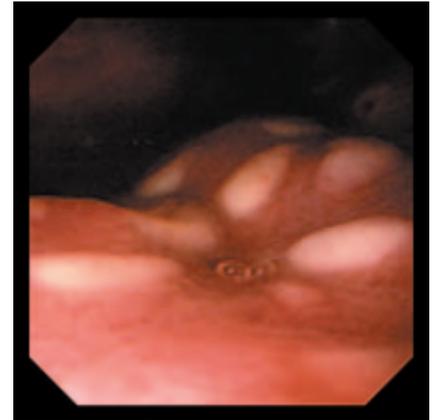
2.385



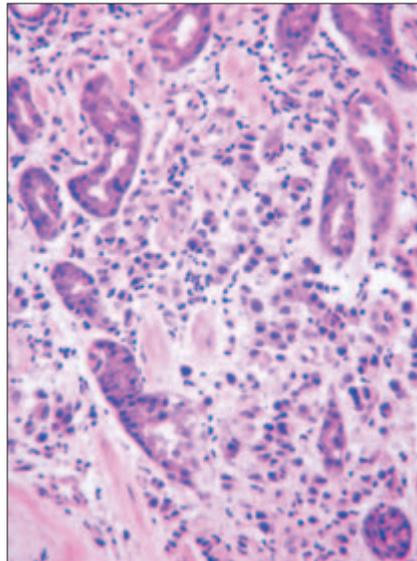
2.386



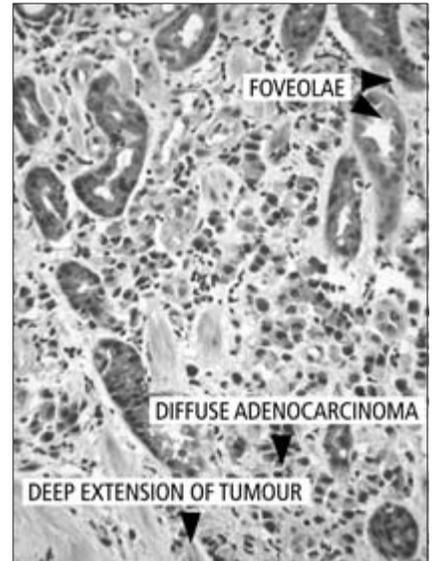
2.387



2.388



2.389

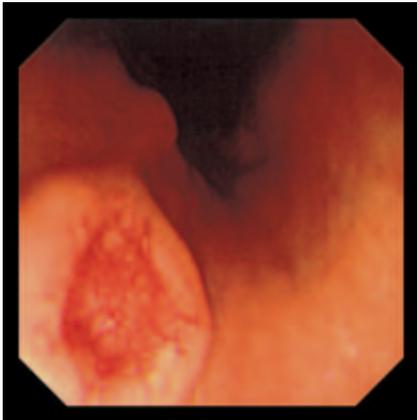


### Ulcer cancers

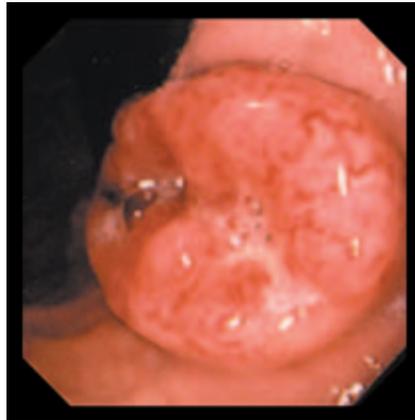
An ulcer cancer is not a cancer that looks like an ulcer. By definition, these are cancers that have arisen in and as a complication of gastric ulcers, emphasizing the need for every benign looking gastric ulcer to be biopsied extensively.

### Endocrine cell tumours

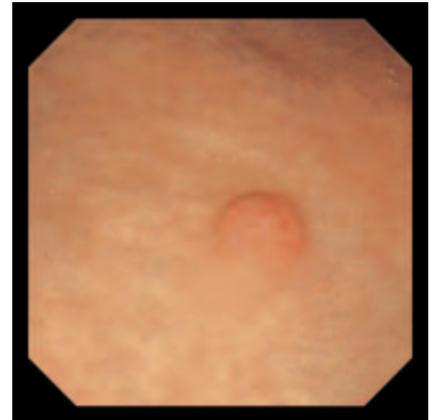
These tumours, also known as carcinoids, are uncommon in the stomach and occur more frequently in other parts of the gastrointestinal tract, for example the appendix. The endoscopic appearances are usually nonspecific. In this case (Fig. 2.390) the apex of the lesion was umbilicated and locally covered with small new vessels. In another patient there was, in addition to new vessel formation and umbilication, evidence of multiple superficial ulcerations (Fig. 2.391); the tumour proved to be deeply invasive. Single (Fig. 2.392) or multiple endocrine cell tumours may be seen in atrophic type A autoimmune gastritis. Figure 2.393 shows the histopathological appearances. Neuroendocrine cell hyperplasia and multiple microcarcinoids may also be seen (Fig. 2.394).



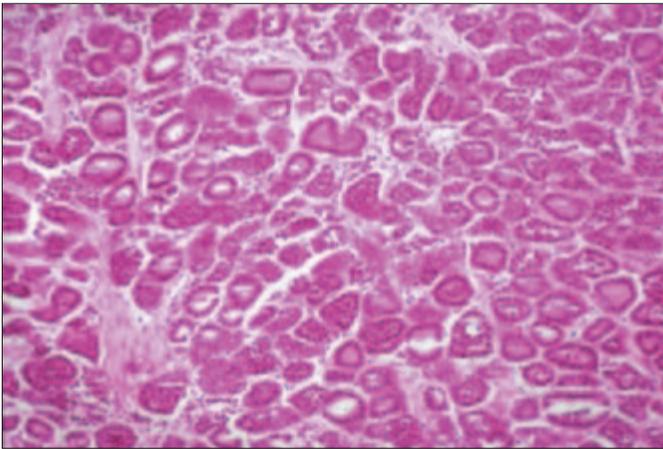
2.390



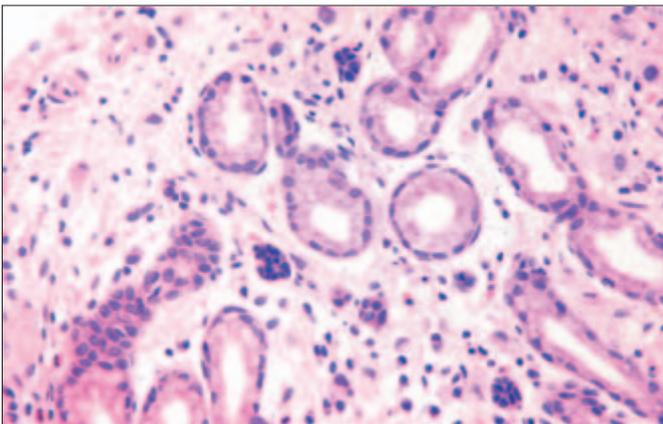
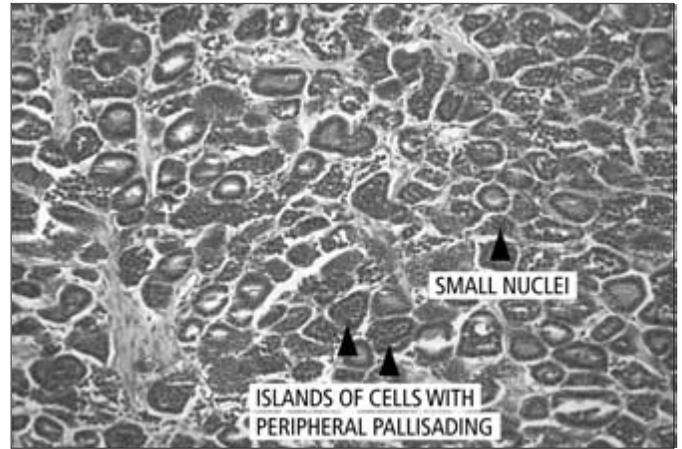
2.391



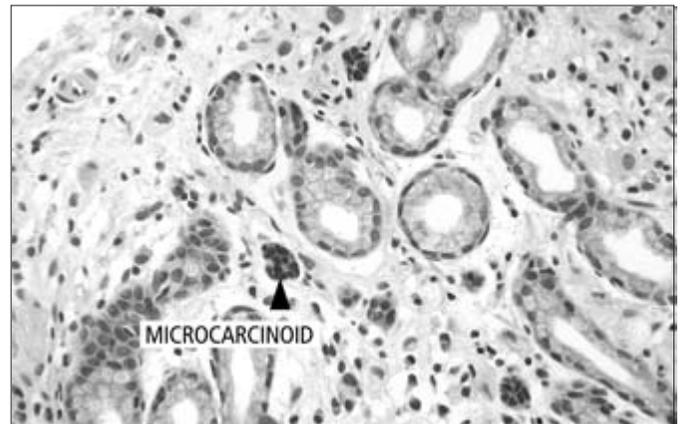
2.392



2.393



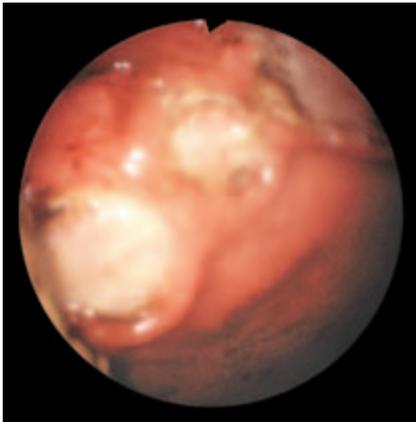
2.394



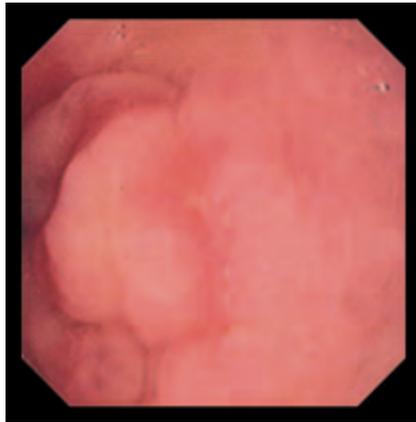
### Secondary carcinoma

Metastasis to the stomach is uncommon. Figure 2.395 shows ulcerated gastric metastases arising from the pancreas, while in Fig. 2.396 there is a metastasis from a breast tumour. Figure 2.397 illustrates a secondary deposit from an adenocarcinoma of the colon: note how the lesion appears to be bursting through the gastric mucosa.

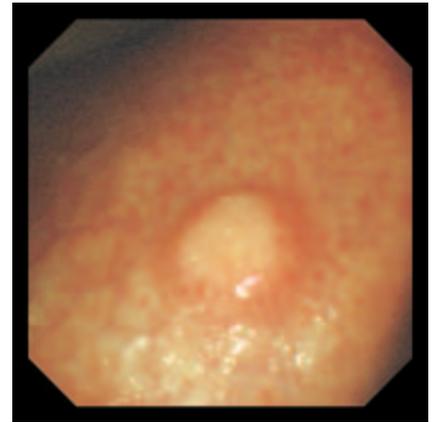
Secondary lobular carcinoma of the breast (Fig. 2.398) and diffuse-type gastric adenocarcinoma may appear similar not only histologically but also immunohistochemically since many primary gastric adenocarcinomas will be oestrogen-receptor positive.



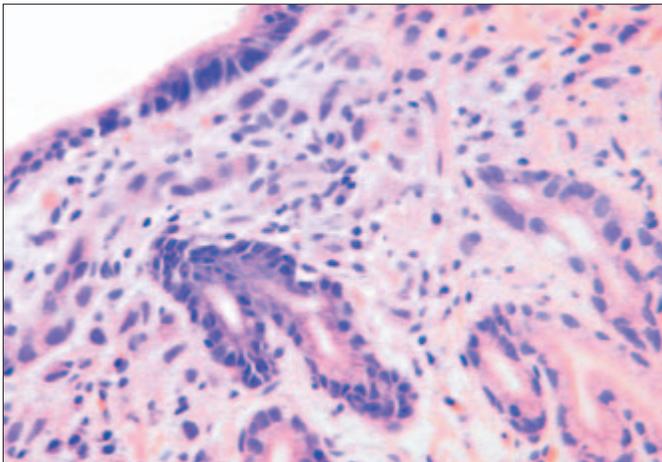
2.395



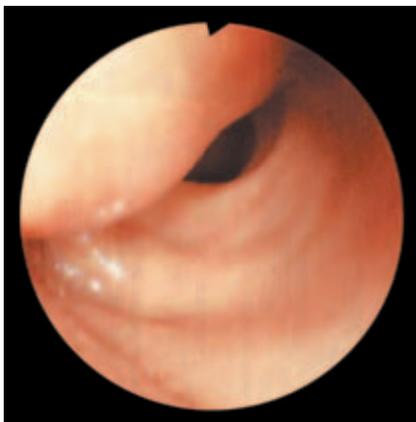
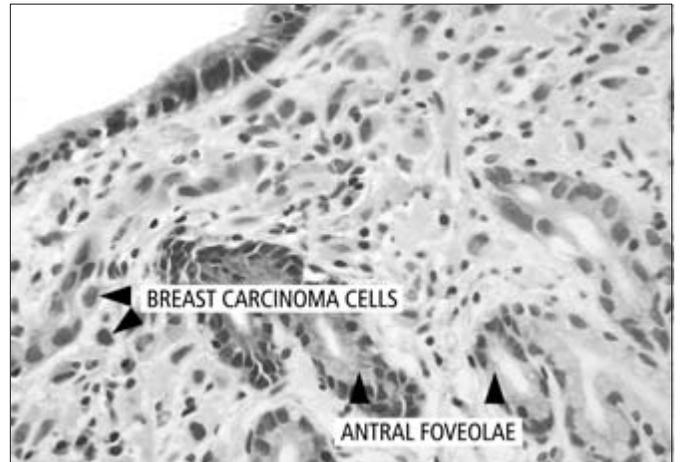
2.396



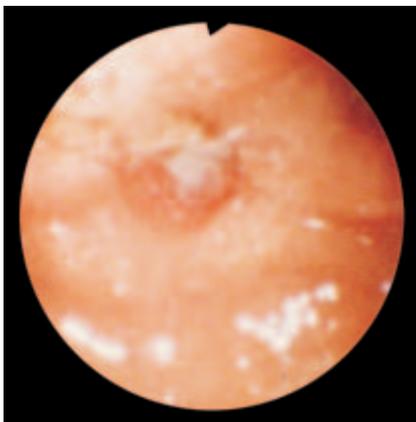
2.397



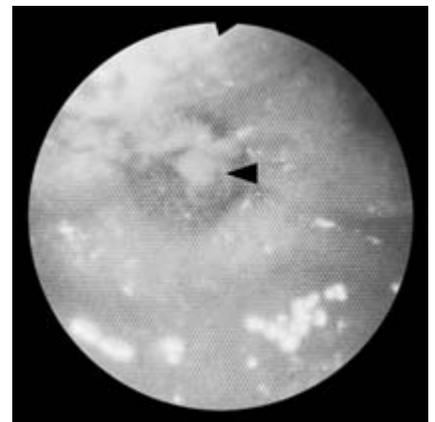
2.398



2.399



2.400



### Invading extra-gastric carcinoma

Figure 2.399 shows the proximal view of a carcinoma of the gallbladder, as a mass indenting the antral wall and covered with intact mucosa. As the endoscope was advanced a little and the tip tilted up, an ulcerated area was visualized; this surrounded a fistula connecting the cavity of the gallbladder to the antrum (Fig. 2.400).

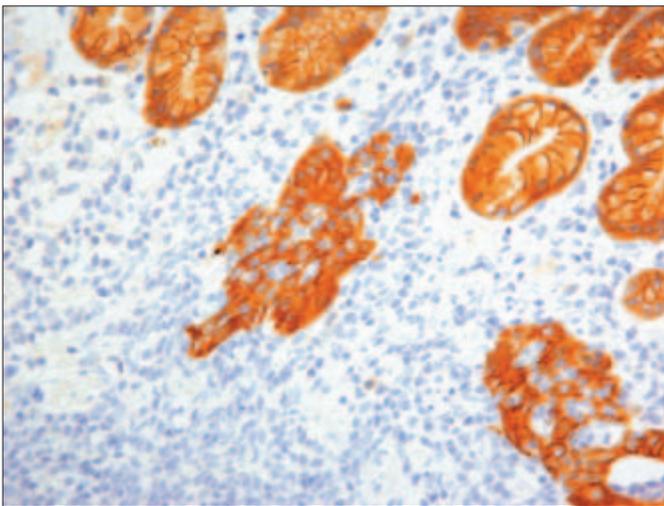
### Lymphoma

Lymphomas are now classified according to the Revised European and American Lymphoma classification, or REAL system and Table 2.9 illustrates how gastrointestinal lymphomas fit into this schema.

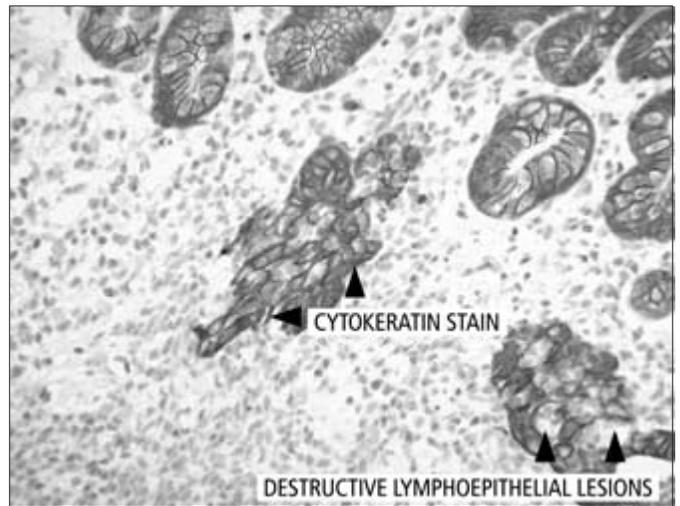
**Table 2.9** The REAL classification of lymphomas with special reference to lymphomas of the gastrointestinal tract (adapted from Mason and Gatter, 1998)

Precursor B cell neoplasia	
Peripheral B cell neoplasia	e.g. MALT lymphoma
Precursor T cell neoplasia	
Peripheral T cell neoplasia	e.g. enteropathy associated T cell lymphoma (EATL of coeliac disease)
Hodgkin's disease	

Upper gastrointestinal tract lymphoma may be limited to the bowel or may be part of widespread disease. The commonest type is a *Helicobacter*-driven B cell MALT lymphoma of the stomach (MALT being the acronym for mucosa-associated lymphoid tissue). This is usually a low grade lymphoma. Some of these lesions are curable with antibiotic therapy against *Helicobacter pylori* but some may develop into high grade lymphoma. MALT lymphomas are recognized histologically by the presence of destructive lymphoepithelial lesions of an abnormal clonal B cell population in the lamina propria. Immunohistochemistry using cytokeratin antibody will identify lymphoepithelial lesions of MALT lymphomas. Immunohistochemistry will also identify B and T cells and kappa and lambda light chain restriction. Figure 2.401 illustrates cytokeratin staining of a lymphoepithelial lesion in a MALT lymphoma.



2.401



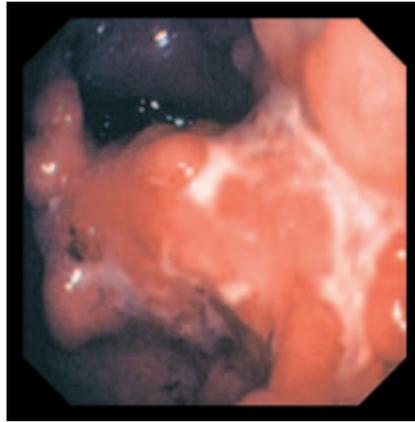
**Lymphoma (cont.)**

The next most common upper gastrointestinal lymphoma is probably the enteropathy-associated T cell lymphoma (EATL) of coeliac disease (see p. 157). This must be sought diligently in patients with coeliac disease found to have unexplained small intestinal masses or ulceration. Here the abnormal cell is the T cell.

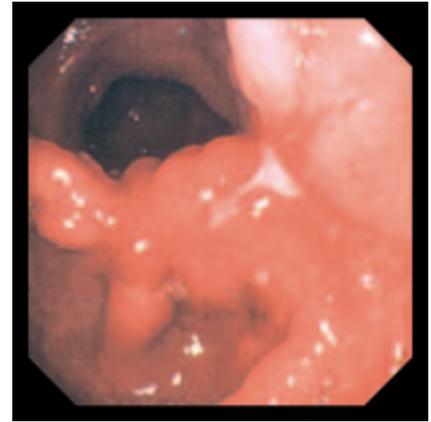
An untreated *H. pylori*-associated MALT lesion is shown in Fig. 2.402. There was a marked response to the appropriate anti-*H. pylori* treatment within 2 weeks (Fig. 2.403), with nothing more than an appearance suggestive of chronic gastritis 10 months later (Fig. 2.404).

Endoscopically, gastric lymphomas do not have pathognomonic features but certain characteristics are suggestive of this: lymphomatous ulcers may be large, well defined but shallow (Fig. 2.405). Other lymphomatous ulcers are shown in Figs 2.406, 2.407 and 2.408.

Less commonly, gastric lymphoma is manifested by widespread raised white plaques (Fig. 2.409).



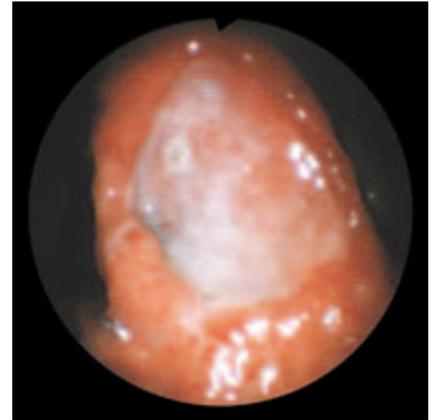
2.402



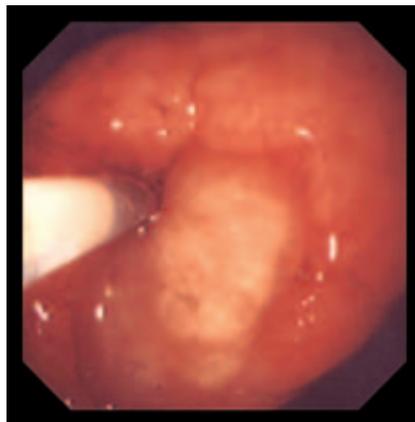
2.403



2.404



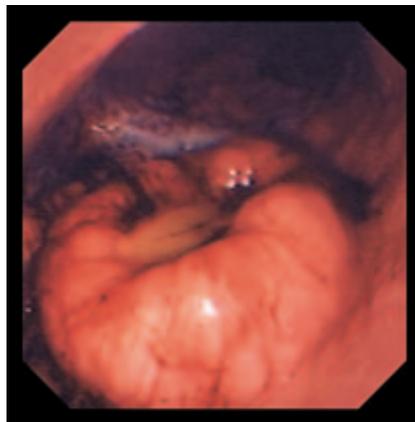
2.405



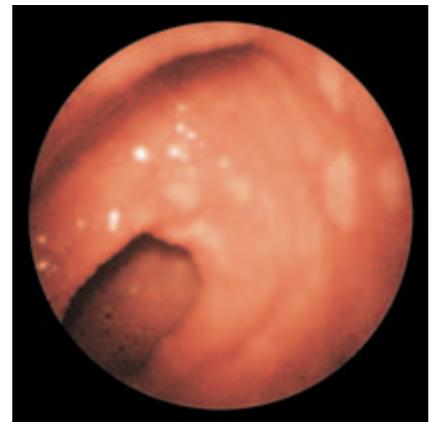
2.406



2.407



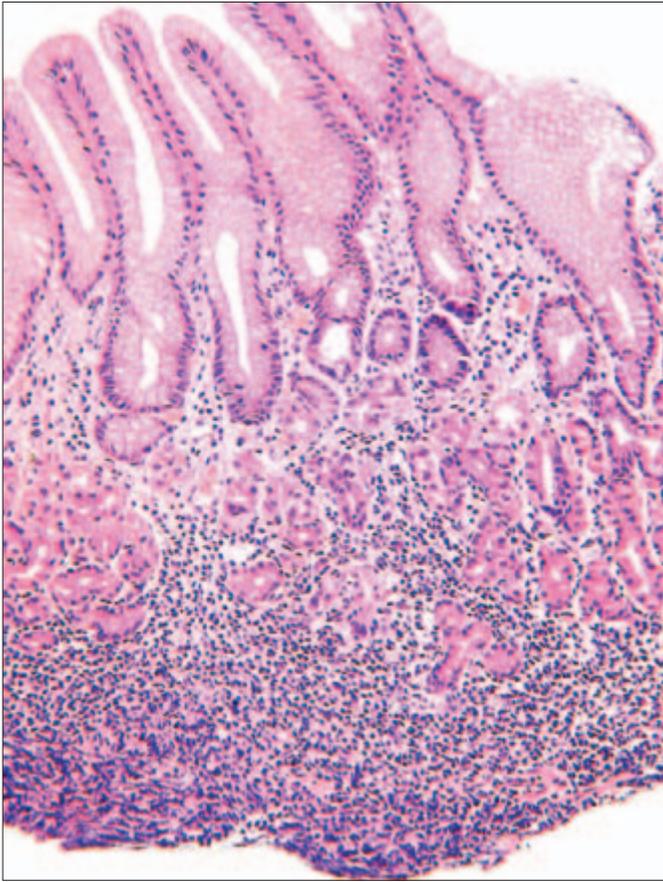
2.408



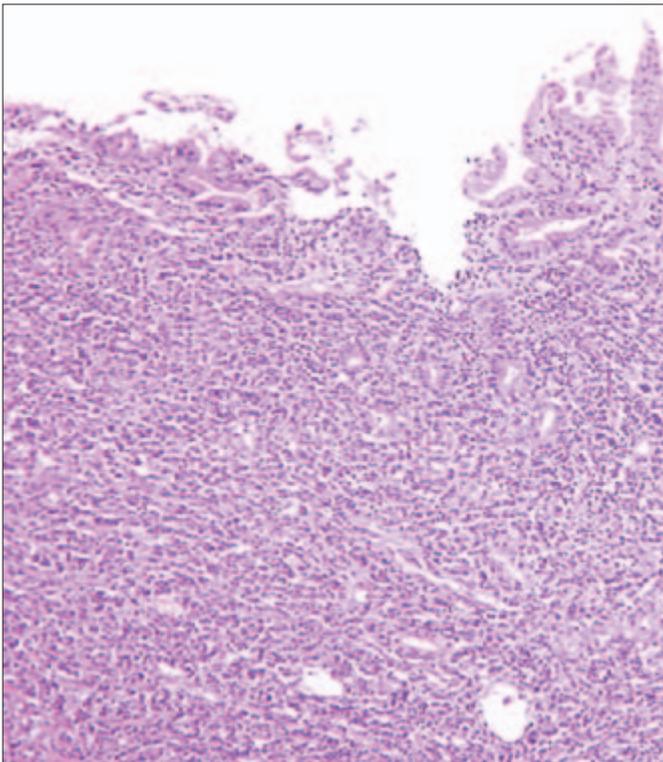
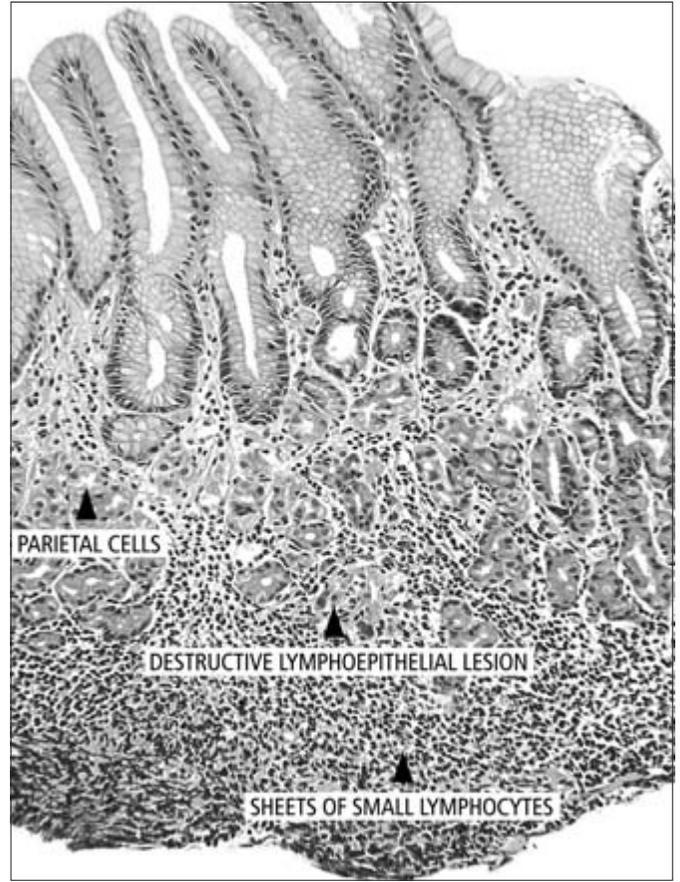
2.409

**Lymphoma (cont.)**

The pathological appearances of a low grade MALT lymphoma and a high grade gastric lymphoma are shown in Figs 2.410 and 2.411 respectively.



2.410



2.411



**Benign inflammatory fibroid polyp**

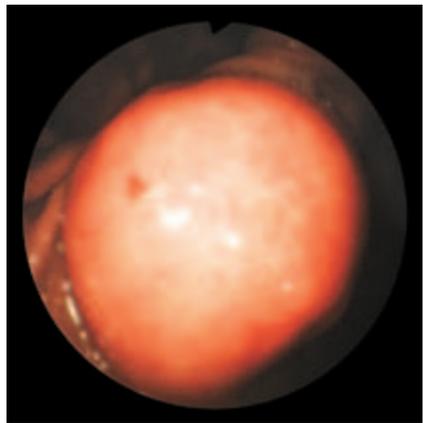
This uncommon lesion has no specific endoscopic features and rarely, as in this case, is pedunculated (Fig. 2.412).



2.412

**Lipomas**

Gastrointestinal lipomas are usually smooth globular lesions covered by normal mucosa (Fig. 2.413). The oesophageal lipoma shown in Fig. 2.180 is an obvious exception. Easy and persistent indentation with biopsy forceps (Fig. 2.414)—the ‘pillow sign’—is almost pathognomonic (see also Figs 3.179a and 3.179b).



2.413



2.414

**Gastrointestinal stromal tumours (GISTs)**

These lesions, formerly known as leiomyoma, leiomyosarcoma, peculiar tumour of Stout, epithelioid leiomyoblastoma and smooth muscle tumours of uncertain malignant potential (STUMPs), have been renamed. Terminology has changed due to emerging new immunohistochemical techniques, a variety of immunohistochemical staining characteristics and doubt as to the cell of origin. Some of these cells stain like smooth muscle cells. Others have a neural phenotype, some are mixed and some are non-staining. Most will be positive with Ckit. It is difficult to assess a benign as against a malignant GIST but a good guide is obtained from the histological type and mitotic count (Table 2.10).

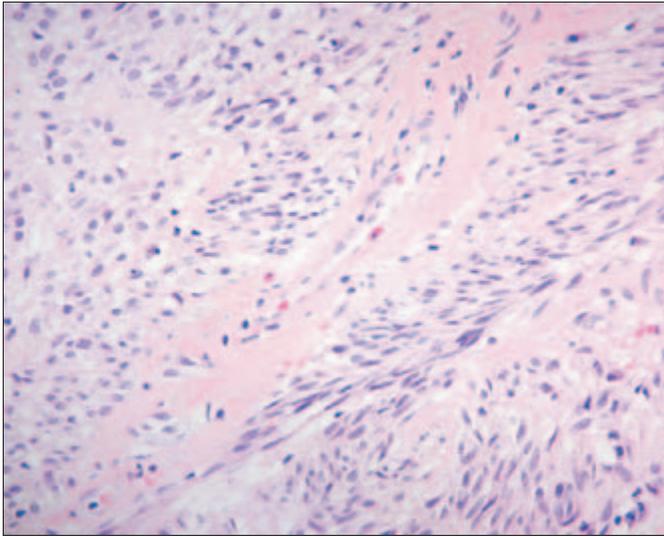
**Table 2.10** Histological criteria for grading gastrointestinal stromal tumours (from Newman *et al.*, 1991)

Benign	either	0–2 mitoses per 30 HPF	Spindle cell lesion, no atypia
	or	0 mitoses per 30 HPF	Epithelioid lesion
Borderline	either	2–3 mitoses per 30 HPF	Spindle cell lesion, mild pleomorphism/hyperchromasia
	or	3–4 mitoses per 30 HPF	Spindle cell lesion, no atypia
	or	1 mitosis per 30 HPF	Epithelioid lesion
Malignant	either	> 5 mitoses per 30 HPF	Spindle cell lesion, no atypia
	or	> 3 mitoses per 30 HPF	Spindle cell lesion, frank pleomorphism/hyperchromasia
	or	> 2 mitoses per 30 HPF	Epithelioid lesion

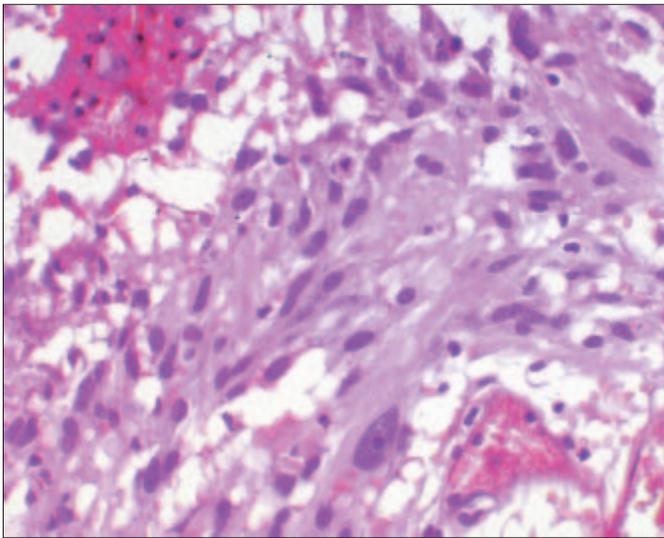
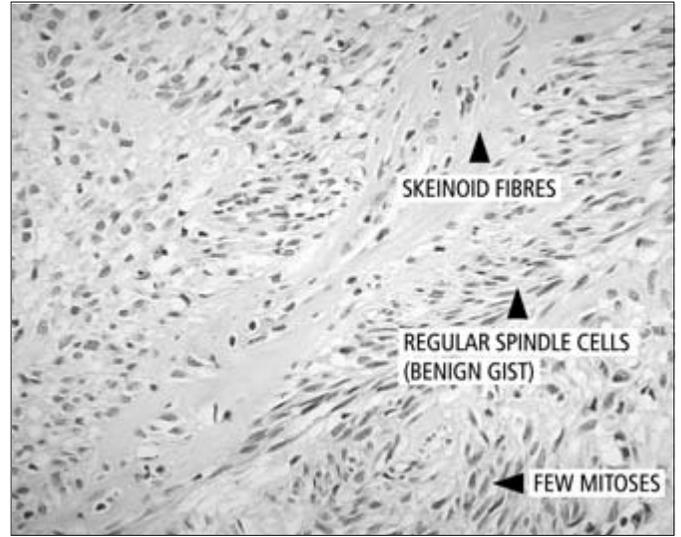
1 HPF (high power field) = 0.159 mm<sup>2</sup> on the microscopes used

**Gastrointestinal stromal tumours (GISTs) (cont.)**

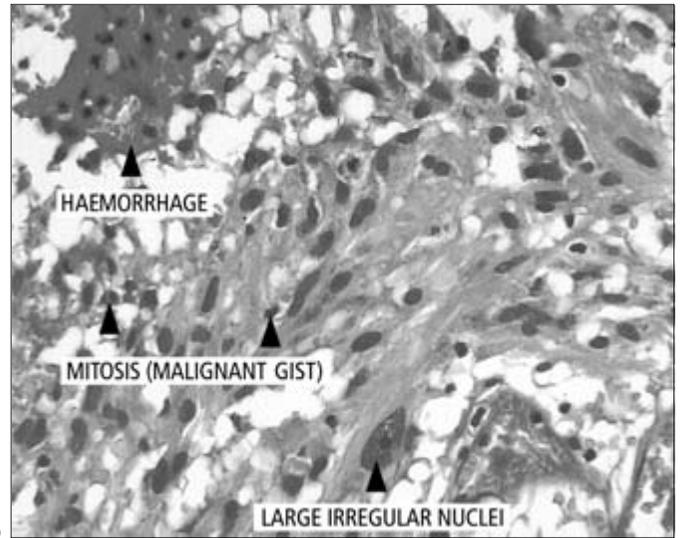
Figure 2.415 shows the histological appearances of a benign (Fig. 2.415a) and a malignant (Fig. 2.415b) GIST.



2.415a



2.415b



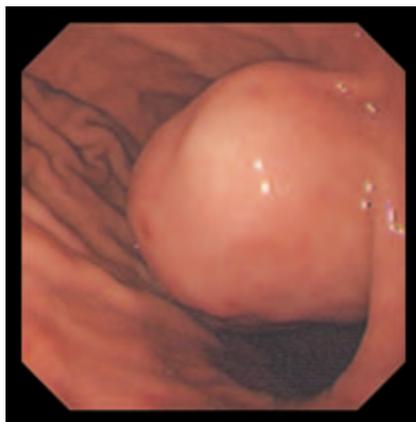
### Gastrointestinal stromal tumours (GISTs) (cont.)

These tumours are usually sessile and often ulcerate centrally giving a characteristic appearance (Fig. 2.416). Bleeding may occur from this ulcer. The tumour seen in Fig. 2.417 was situated high on the lesser curve and was stalked; it was better seen on inversion (Fig. 2.418).

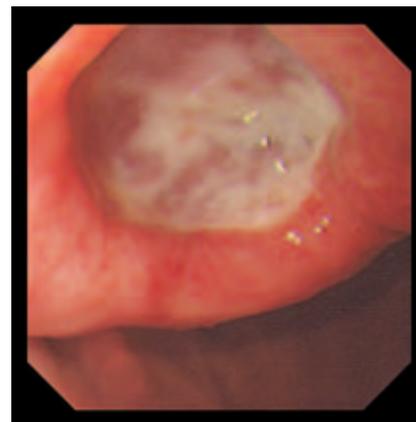
It is hazardous to attempt to remove a GIST endoscopically without full and appropriate imaging as a significant portion of the tumour may lie extraluminally.



2.416



2.417



2.418

### Kaposi's sarcoma

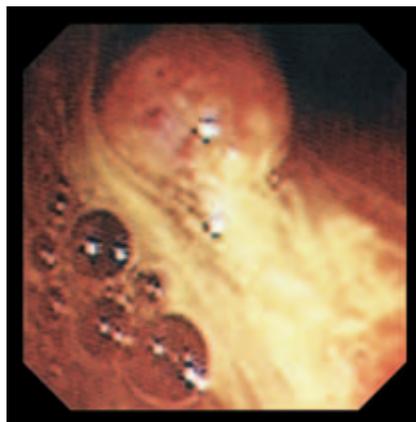
This has no specific endoscopic features to distinguish it from other malignant tumours (Fig. 2.419). The increasing incidence of AIDS should heighten the endoscopist's awareness of this possible diagnosis. Prognosis is poor.



2.419

### Inflammatory cap polyp

This is a rare polyp in the gastrointestinal tract and extremely rare in the stomach. Endoscopically, it may have inflammatory slough on the surface (Fig. 2.420) or may have no distinguishing features. Histologically, the features are those of prolapse (see also pp. 309–310).



2.420

## Vascular abnormalities

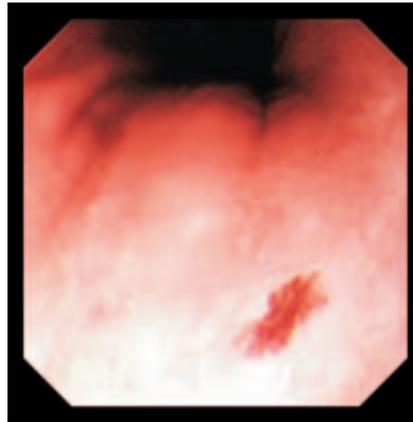
### Haemangioma and angiodysplasia

#### Portal hypertensive gastropathy

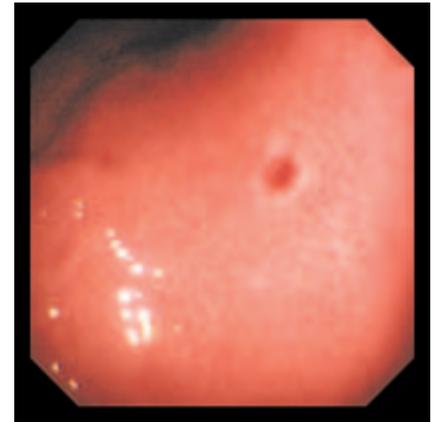
#### Varices

#### Haemangioma and angiodysplasia

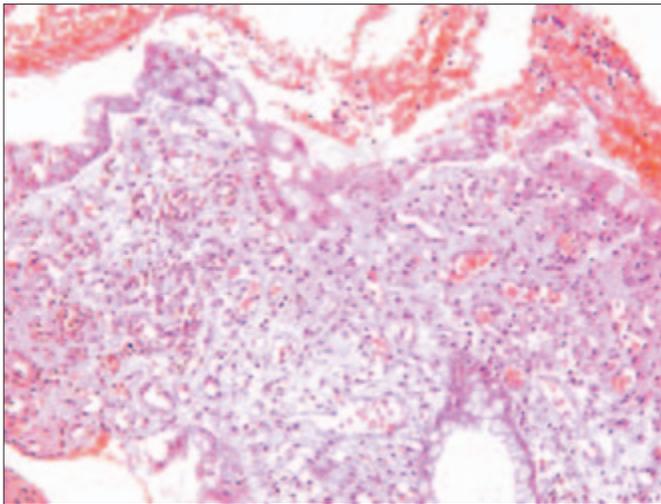
Figure 2.421 shows a capillary haemangioma. These occur singly or in larger numbers, particularly in the stomach and small bowel, as in the Osler–Weber–Rendu syndrome. Such lesions are often surrounded by a white ‘halo’ (Fig. 2.422) which aids in the differential diagnosis. Figure 2.423 shows the histopathological appearances.



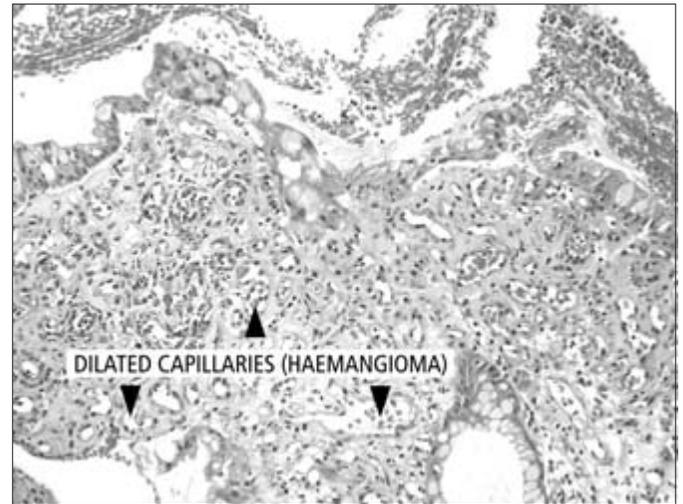
2.421



2.422



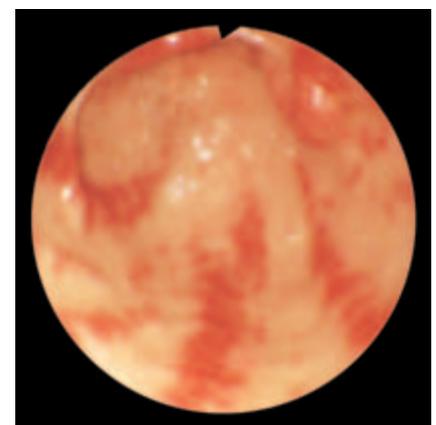
2.423



A diffuse form of antral haemangioma is shown in Fig. 2.424 (note argon beam electrode). Another type is illustrated in Fig. 2.425. These lesions are easily confused with the red streaks commonly seen in the antrum (Fig. 2.37) but are wider, and are a recognized cause of acute and chronic upper gastrointestinal bleeding.



2.424



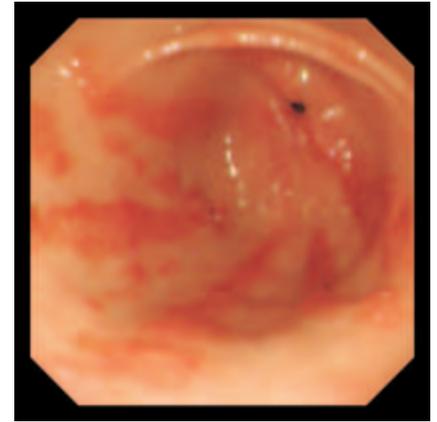
2.425

**Haemangioma and angiodysplasia (cont.)**

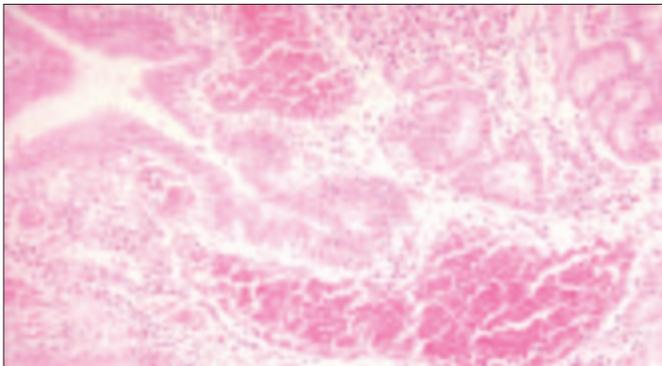
A more florid type of antral haemangioma (gastric antral vascular ectasia or GAVE), often described as an ‘octopus’ or ‘watermelon’ lesion, is shown in Fig. 2.426. Such lesions are commonly but not invariably confined to the antrum. The lesions seen in Fig. 2.427 involve the antrum and the body. The patient later underwent endoscopic therapy (Fig. 2.778). Figure 2.428 shows the pathological features.



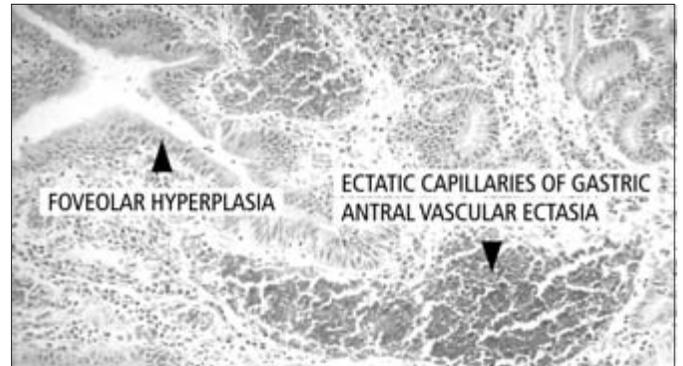
2.426



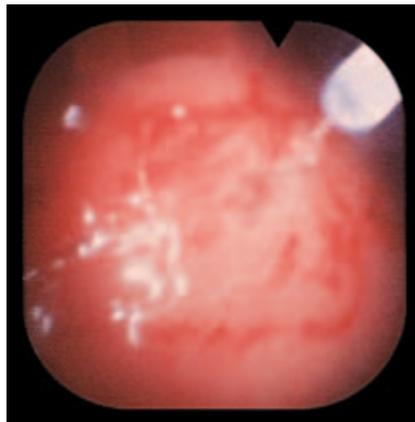
2.427



2.428



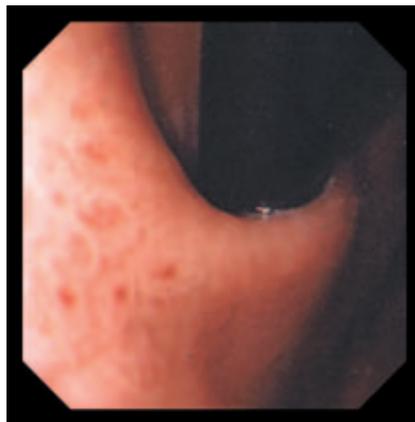
Angiodysplasia is represented in Fig. 2.429. This is uncommon in the stomach though relatively common in the colon (see pp. 297–298). In this patient the appearances were at first thought to represent local bleeding. However, washing with a jet of water—as illustrated—had no effect. Biopsy of the lesion revealed its nature.



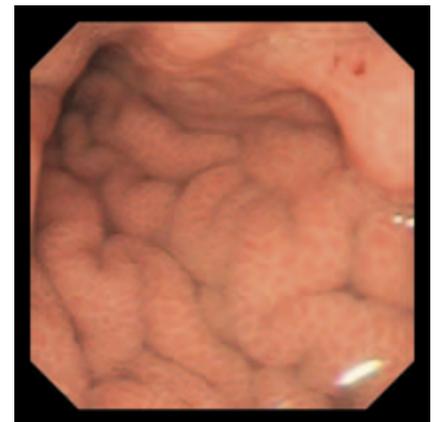
2.429

**Portal hypertensive gastropathy**

Portal hypertensive gastropathy often accompanies oesophageal or gastric varices; it may become more marked after variceal sclerotherapy or banding. It is seen most commonly in the fundus where in the mildest form there are prominent small vessels in the gastric mucosa (Fig. 2.430). When well established there is an irregular red mosaic pattern (Figs 2.431 and 2.432) due to dilated small vessels and intramucosal petechiae.



2.430

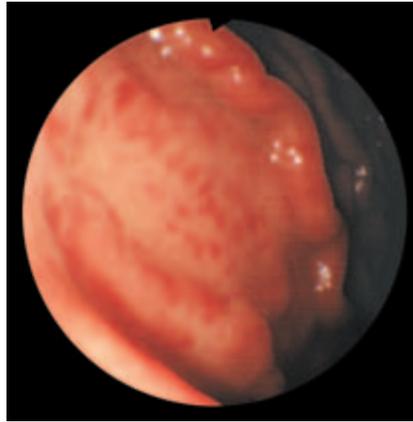


2.431

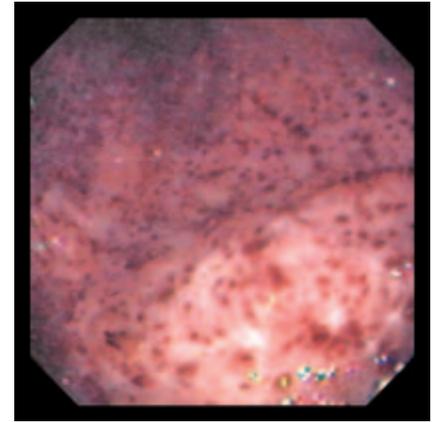
**Portal hypertensive gastropathy (cont.)**

Severe changes are shown in Fig. 2.433.

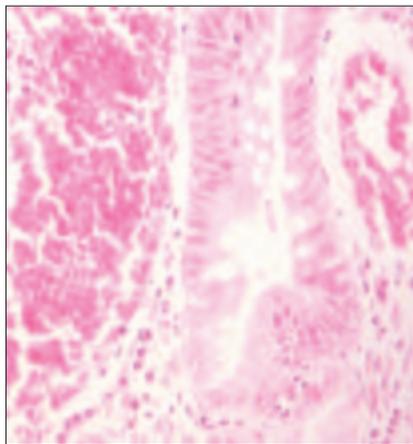
The histopathological changes (Fig. 2.434) are indistinguishable from those seen in gastric antral vascular ectasia which may coexist.



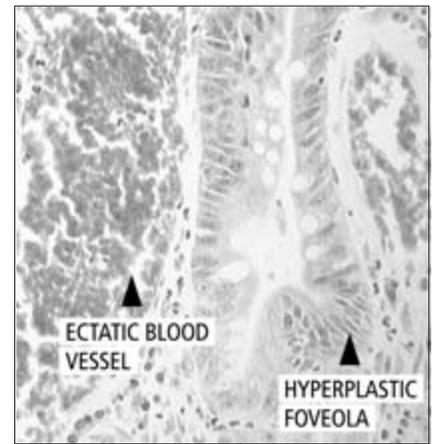
2.432



2.433

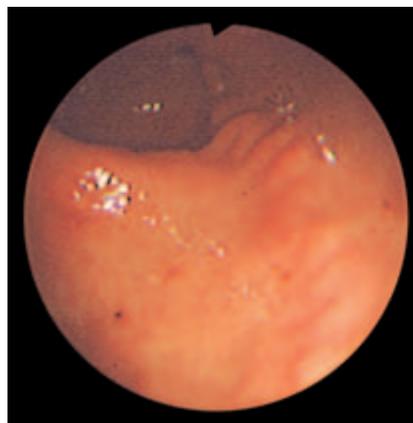


2.434

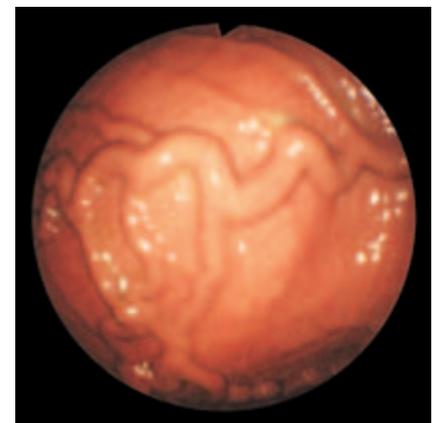


**Varices**

Gastric varices (Fig. 2.435) seldom occur in the absence of oesophageal varices but are less common. They may resemble normal rugae and are easily overlooked though they are often bluish in colour (Fig. 2.436).



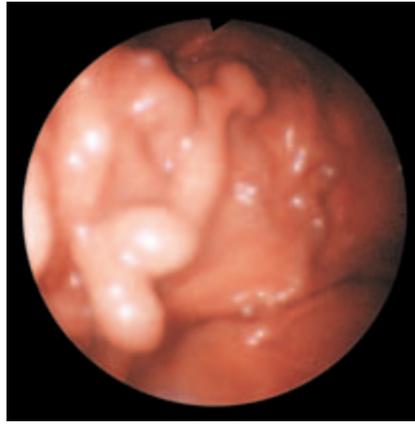
2.435



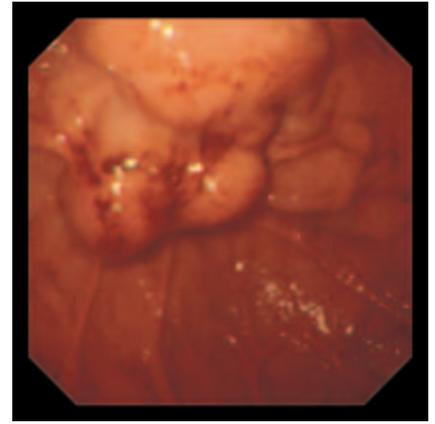
2.436

**Varices (cont.)**

There can be little doubt of the diagnosis when the appearances are as shown in Fig. 2.437 but when gastric varices present as a localized irregular polypoid lesion (Fig. 2.438) the endoscopist may face a diagnostic problem: do not take a biopsy if the lesion could be a gastric varix.



2.437



2.438

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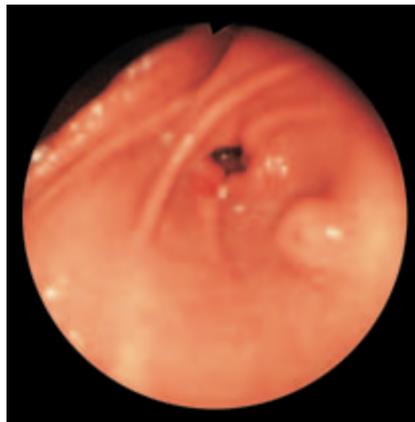
**Pancreas-related abnormalities**

**Heterotopic pancreatic tissue**

**Pancreatic pseudocyst**

**Heterotopic pancreatic tissue**

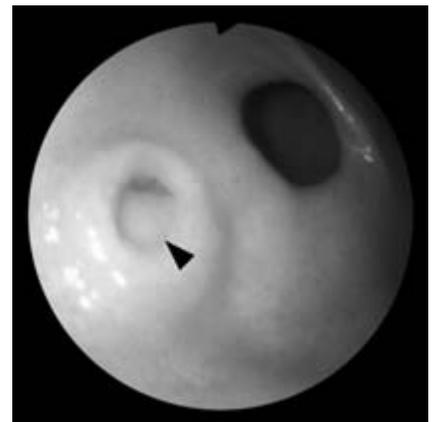
Typically, this rare condition presents as a small sessile antral polyp-like lesion with an apical dimple, believed to represent a rudimentary ductal orifice (Fig. 2.439). Occasionally a small apparently raised diverticulum in the antrum has a similar appearance (Fig. 2.440).



2.439



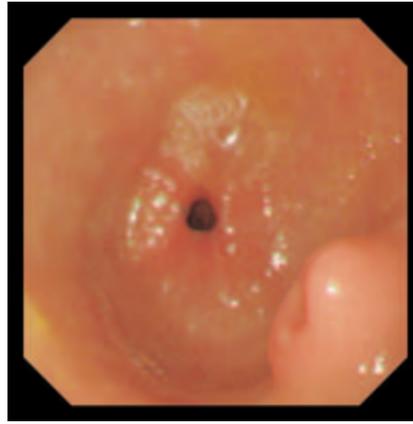
2.440



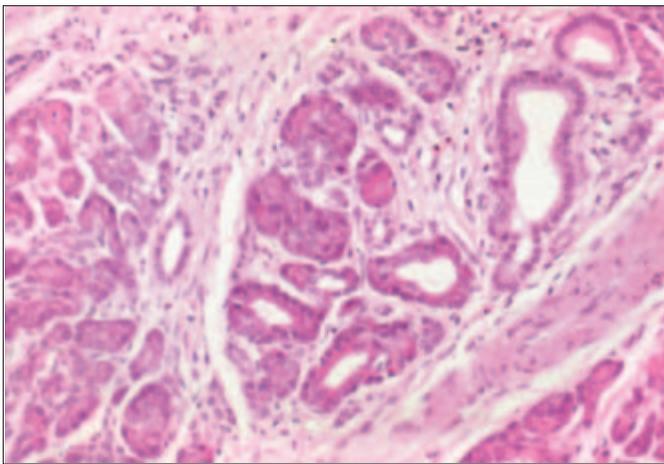
**Heterotopic pancreatic tissue (cont.)**

Figure 2.441 shows a very similar lesion but the dimple is superficially eroded; at first glance the appearances suggest heterotopic pancreatic tissue, but this lesion is probably a single raised erosion. GIST (cf. Figure 2.416) enters the differential diagnosis.

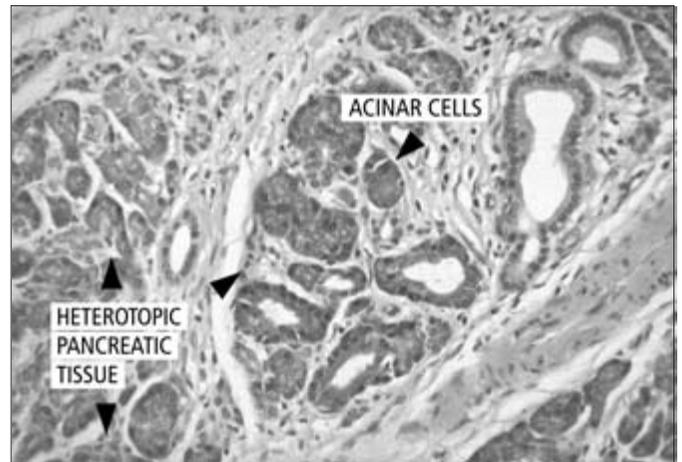
A confirmed diagnosis must rest on histopathological evidence (Fig. 2.442) but endoscopic mucosal biopsies are unlikely to be sufficient. When the endoscopic appearances are typical, strenuous endeavours to obtain material for histopathological study are not necessary.



2.441

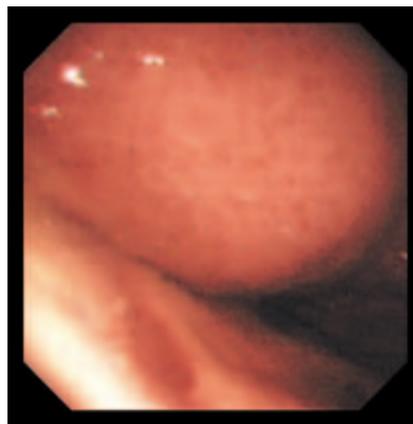


2.442



**Pancreatic pseudocyst**

A large pancreatic pseudocyst may make an extrinsic impression into the stomach (Fig. 2.443) but the appearance is quite nonspecific and confirmatory tests require endoscopic ultrasound or other imaging techniques. Sometimes a pseudocyst may point and discharge spontaneously into the gastric lumen. Endoscopic drainage may occasionally be indicated. Pancreatic pseudocysts are discussed more fully in Chapter 4.



2.443

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## Vomiting and trauma

### Effects of vomiting

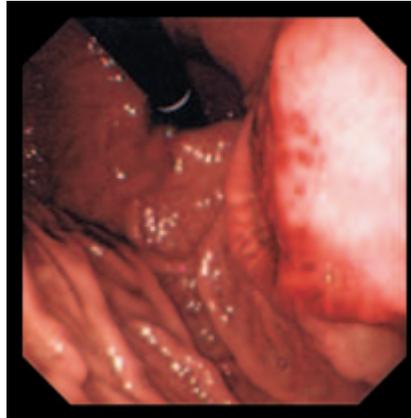
#### Mallory–Weiss tears

#### Trauma produced by the endoscope

#### Traumatic erosions

### Effects of vomiting

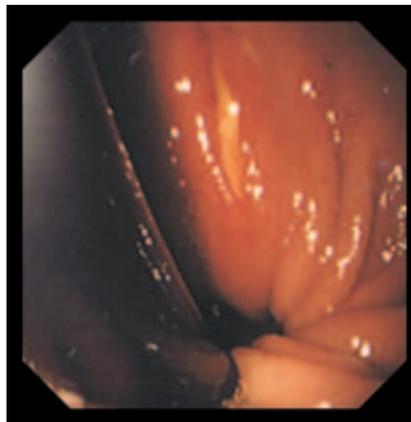
Localized trauma to the gastric mucosa may arise for a variety of reasons, of which vomiting is a common one. In Fig. 2.16 prolapse of gastric mucosa into the lower oesophagus during retching is illustrated. This mucosa may become significantly traumatized leading to appearances known as hernia gastropathy, as shown in Fig. 2.444, and less commonly to a Mallory–Weiss tear.



2.444

### Mallory–Weiss tears

The majority of Mallory–Weiss tears (Fig. 2.445) occur as a result of vomiting, although in some patients there is no such history. The lesion occurs in the region of the oesophago-gastric junction and usually consists of a short longitudinal superficial tear, often with yellow slough in its base, sometimes resembling an atypical gastric ulcer. Patients present with acute upper gastrointestinal bleeding. Figure 2.446 shows a fresh clot on a Mallory–Weiss tear which has recently bled.



2.445



2.446



### Trauma produced by the endoscope

Red spots may be caused by sucking a knuckle of mucosa into the biopsy channel. When this tissue is released it appears first as a cherry-red round raised lesion (Fig. 2.447) flattening over the course of a few seconds to a red ring (Fig. 2.448) which may fade rapidly unless petechial spots have been induced.

As well as causing suction marks, the endoscope tip may produce a variety of minor and occasionally frightening-looking mucosal marks and ecchymoses which are usually of little significance (Fig. 2.449). This occurs on the high lesser curve if the instrument strikes the mucosa after first entering the stomach. If not recognized at the time, these appearances may be puzzling when seen later in the examination. Clearly rough handling of the endoscope is more likely to produce such appearances.

Gastric perforation is a rare but serious complication of endoscopy (Figs 2.808 and 2.809). Superficial dehiscence is less serious (Fig. 2.450) but should suggest to the endoscopist that his technique is inappropriately rough.

### Traumatic erosions

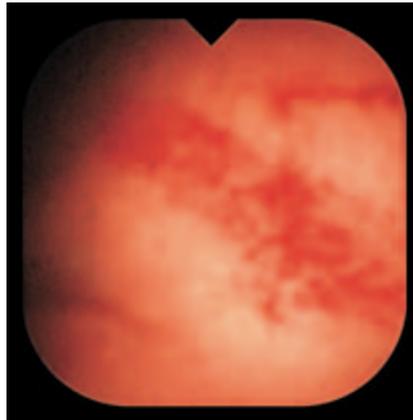
These appearances (Fig. 2.451) were caused by a nasogastric tube which had been left *in situ* for a number of days. The lesions were red, raised, multiple and superficially eroded and resembled endoscopic suction artefacts (Fig. 2.447).



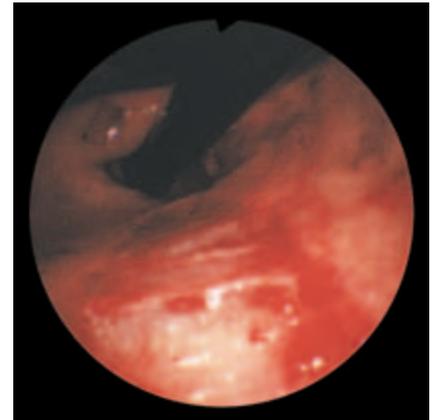
2.447



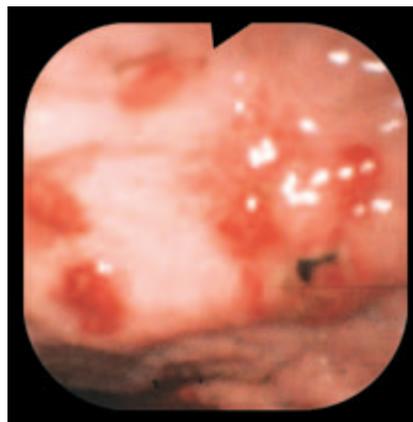
2.448



2.449



2.450



2.451

## Miscellaneous conditions

Amyloid

Ménétrier's disease

Xanthelasma

Thrombocytopenia

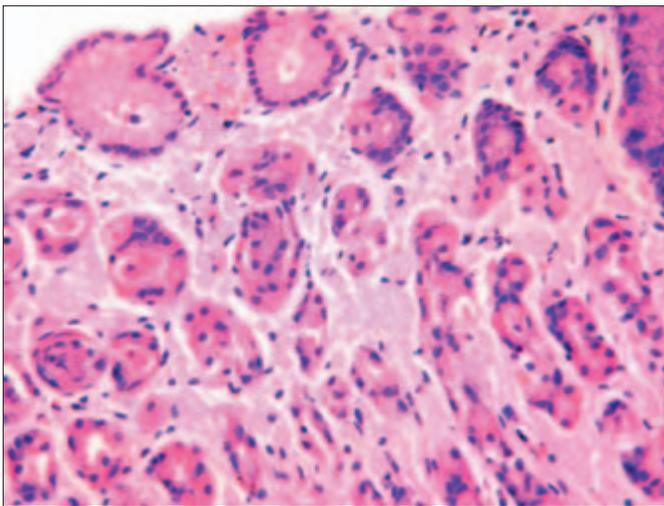
Food remnants, capsule and tablets

Foreign bodies

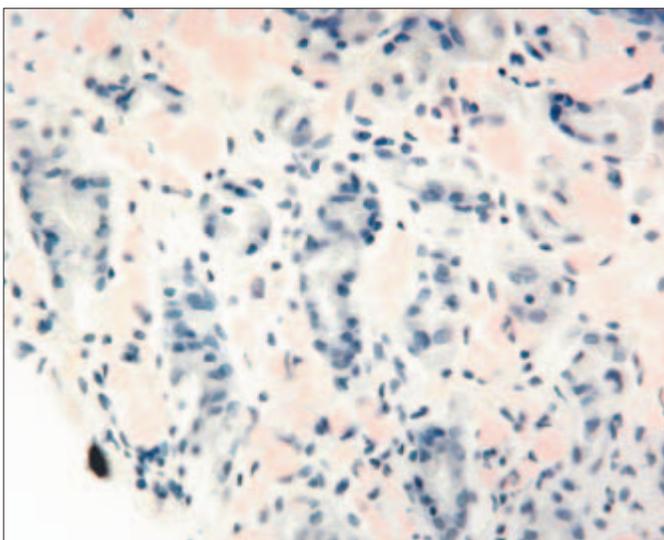
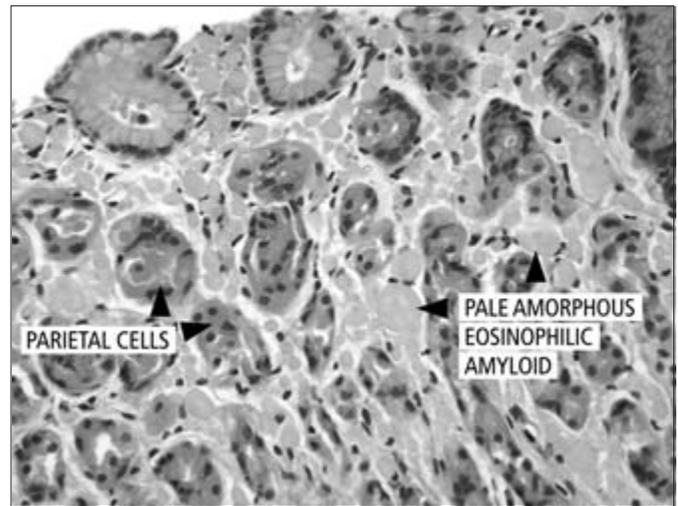
Chemical injury

### Amyloid

Amyloid of the stomach may be seen as part of systemic amyloidosis or, very rarely, as an isolated gastric finding. The endoscopic appearances may be nonspecific, or may resemble linitis plastica. Histopathologically the appearances of amyloid may also mimic those of linitis plastica. A deep biopsy may be required, and should be stained with haematoxylin and eosin (Fig. 2.452), and Congo red (Fig. 2.453) and viewed between polarizing lenses for apple-green birefringence.



2.452



2.453

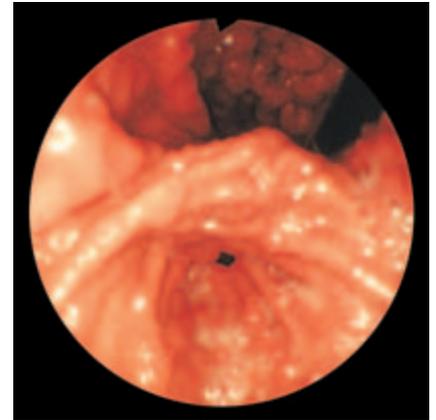
### Ménétrier's disease

There is great variation in the thickness and configuration of normal gastric rugae. While the appearances shown in Figs 2.454 and 2.455 might suggest an underlying abnormality, biopsy appearances were entirely normal in both instances. Endoscopists and radiologists tend to overdiagnose Ménétrier's disease.

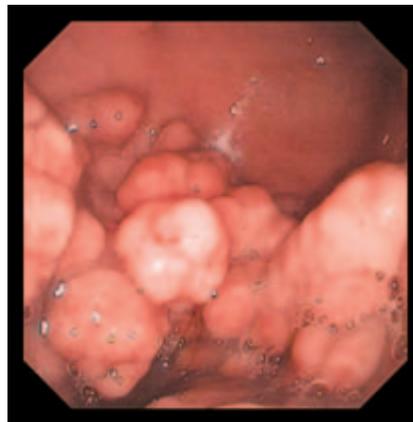
In this rare disorder there is marked thickening, tortuosity and irregularity of the mucosal ridges, occasionally, as in this case, giving the mucosa the appearance of polyposis (Fig. 2.456). Accurate diagnosis must be based on histopathological appearances (Fig. 2.457) of adequate deep biopsies, which may necessitate using a diathermy snare (see p. 332).



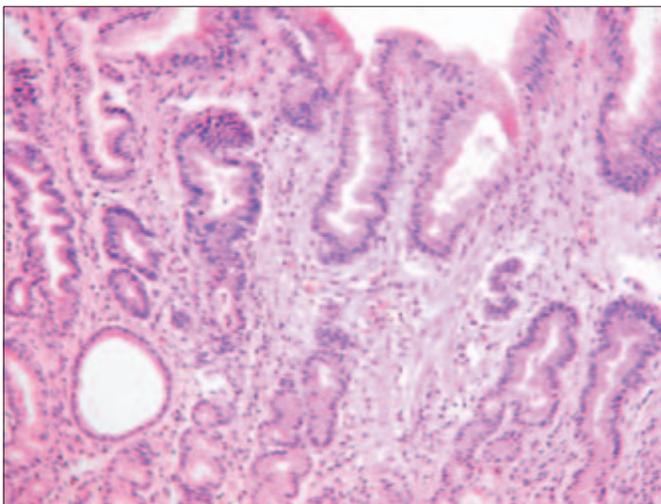
2.454



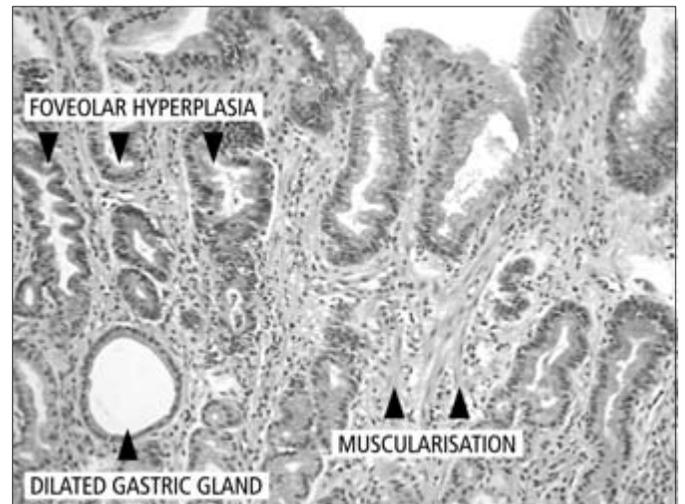
2.455



2.456

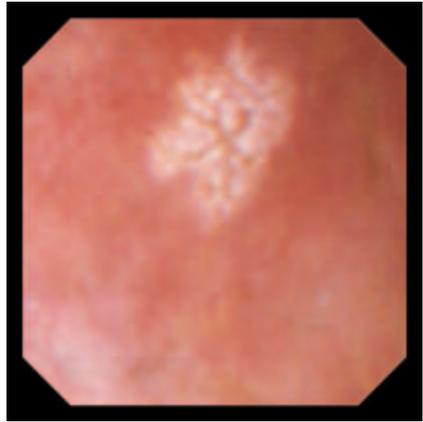


2.457

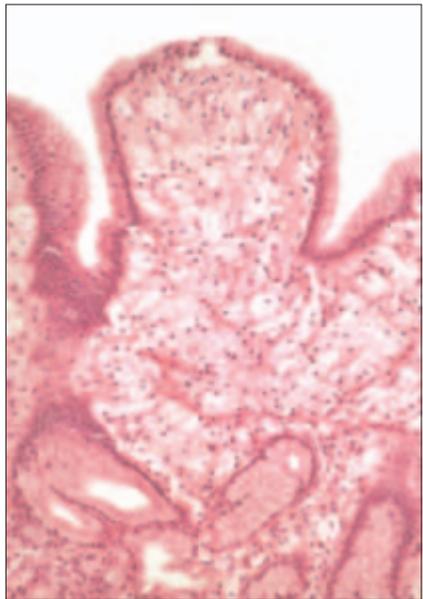


**Xanthelasma**

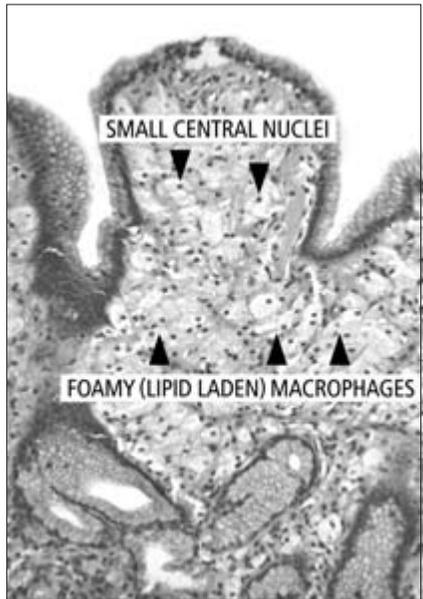
These lesions are commonly seen as small yellow or white plaques (Fig. 2.458), usually in the antrum, and may be multiple. Although they contain lipid-laden macrophages (Fig. 2.459), they do not correlate with the presence of hypercholesterolaemia. Their importance lies in differential diagnosis: histologically they may be confused with signet cell carcinoma.



2.458

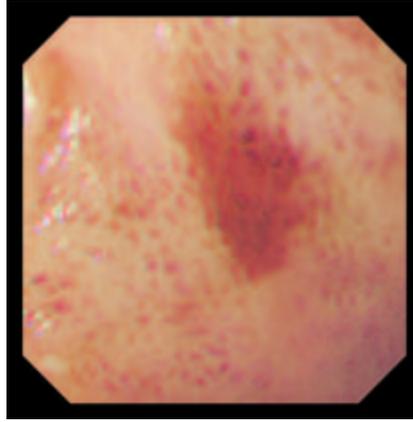


2.459



### Thrombocytopenia

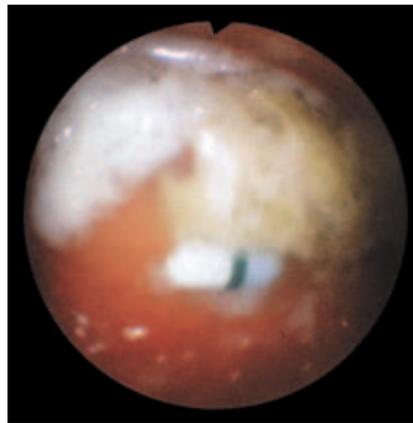
Figure 2.460 shows multiple petechiae, and a mucosal ecchymosis resulting from the endoscopy. The platelet count was  $7 \times 10^9$  per cu mm. Such appearances should alert the endoscopist to the possibility of a generalized haemorrhagic tendency.



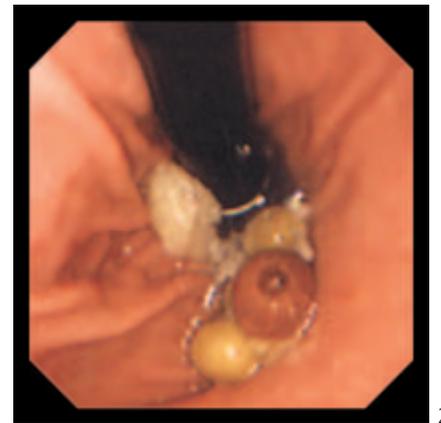
2.460

### Food remnants, capsule and tablets

Despite advice to take nothing by mouth for several hours before endoscopy, it is not uncommon to find food remnants, tablets or capsules. Figure 2.461 shows a capsule (and saliva), Fig. 2.462 a variety of berries and Fig. 2.463 a collection of grape pips. Such findings should be noted on the endoscopy report but are unlikely to be of significance unless there is gastric outlet obstruction. The objects in Fig. 2.464, seen under a pool of clear gastric juice, are not undigested rice or other food particles, but a collection of hyperplastic polyps. The small gastric bezoar made up of inspissated food remnants (Fig. 2.465) was found in a patient with pyloric stenosis.



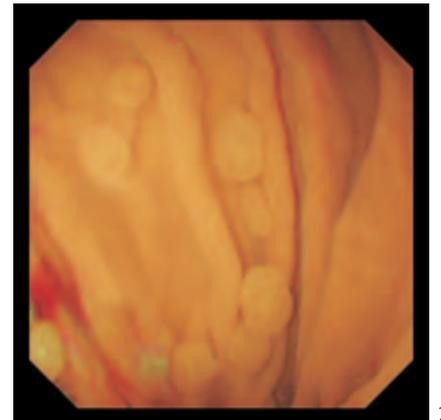
2.461



2.462



2.463



2.464



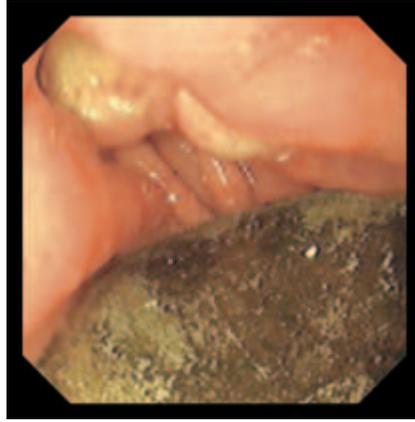
2.465

**Foreign bodies**

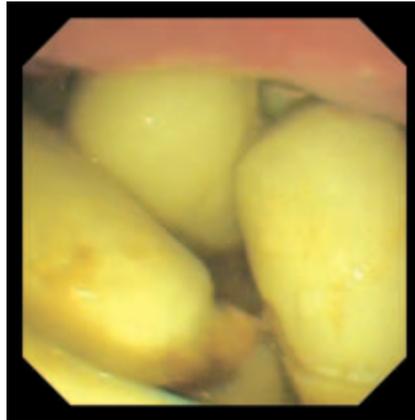
These are described and discussed more fully on pp. 187–196.

A complication of swallowing foreign bodies, in this instance a collection of spoons, is the development of a pressure ulcer (Fig. 2.466).

The difficulties, dangers and limitations of therapeutic endoscopy are evident in Fig. 2.467. The patient was a ‘body-packer’ who had swallowed a number of rubber containers filled with cocaine. Therapeutic endoscopy was contraindicated and the patient underwent laparotomy.



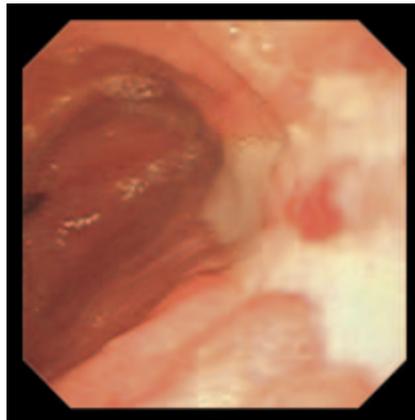
2.466



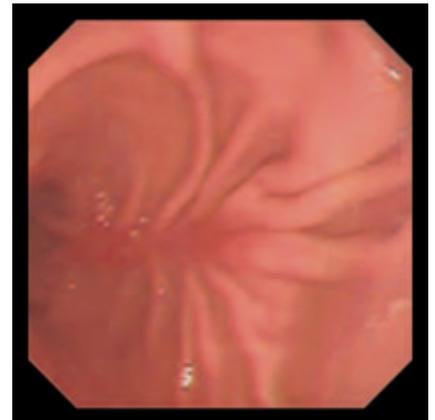
2.467

**Chemical injury**

Injury caused by ingestion of caustic soda is now a rarity in the West. Figure 2.468 shows the appearance of the antrum at the acute stage; within 6 weeks the ulcer had healed but there was considerable scarring (Fig. 2.469).



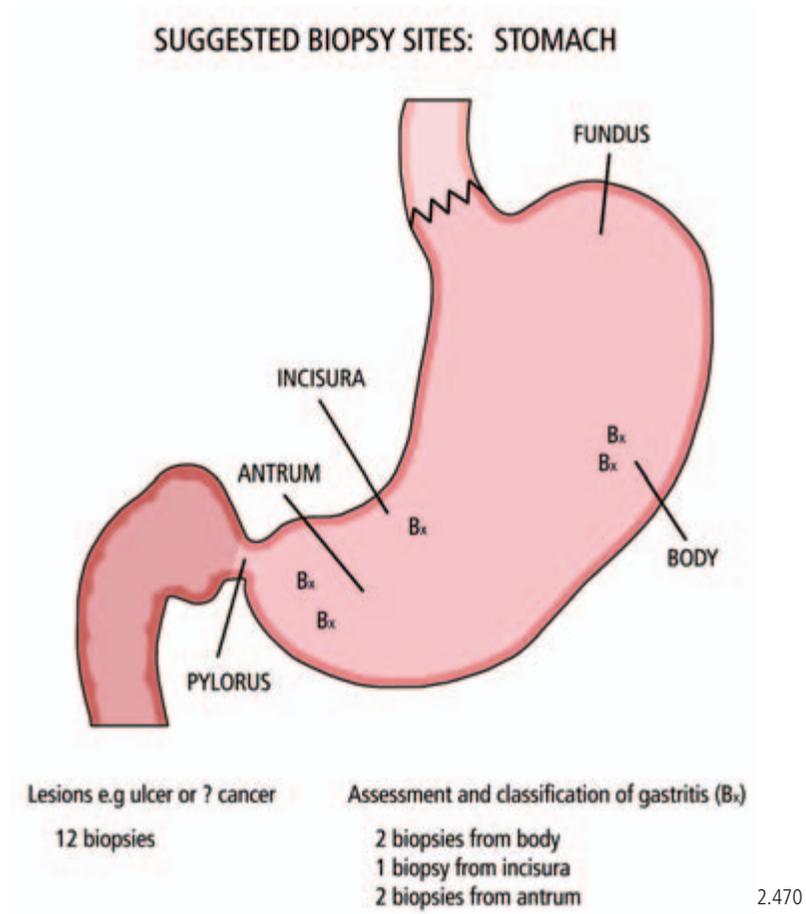
2.468



2.469

## Suggested biopsy sites

Figure 2.470 shows suggested biopsy sites for various types of abnormalities, and the number of samples that should be taken. For further details the reader is referred to the appropriate section of this chapter.



## DUODENUM

**Congenital abnormalities 140**

**Duodenitis 142**

**Peptic ulcer 144**

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**Celiac disease and associated conditions 158**

**Miscellaneous conditions 160**

**Suggested biopsy sites 167**

Many of the abnormalities encountered in the duodenum during OGD using a standard endoscope may also be seen during ERCP and enteroscopy. Some overlap with Chapters 4 and 5 is therefore inevitable. In similar vein when the same conditions occur in the duodenum and elsewhere in the gastrointestinal tract, the discussion on this topic may appear in another section or chapter.

### Congenital abnormalities

#### Diverticula

#### Duodenal reduplication

#### Diverticula

Congenital diverticula are commonly found on the medial wall of the duodenum adjacent to or involving the papilla of Vater (Fig. 2.471), and as a result are of particular relevance to ERCP (see Chapter 4). As the neck of the diverticulum is often narrow, the opening is usually small (Fig. 2.472).



2.471



2.472

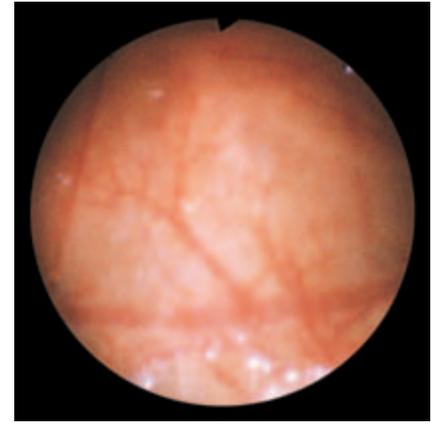


### Diverticula (*cont.*)

If the endoscope is advanced into the diverticulum, the mucosa will be found to look more vascular (Fig. 2.473) than the normal duodenal mucosa. Close inspection within the diverticulum (Fig. 2.474) shows no small intestinal circular folds but instead a thin, flat mucosa with an easily recognized vascular pattern. A duodenal diverticulum should only be entered with great care, as instrumental perforation must be avoided. An unusually wide mouthed diverticulum is shown in Fig. 2.475 and smaller multiple diverticula in Fig. 2.476.



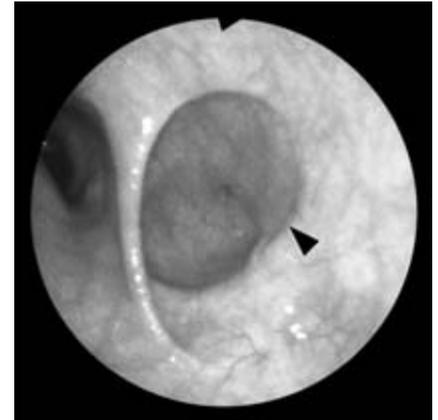
2.473



2.474



2.475



2.476

### Duodenal reduplication

Figure 2.477 illustrates the view from a megabulbus into the duodenal orifices.



2.477

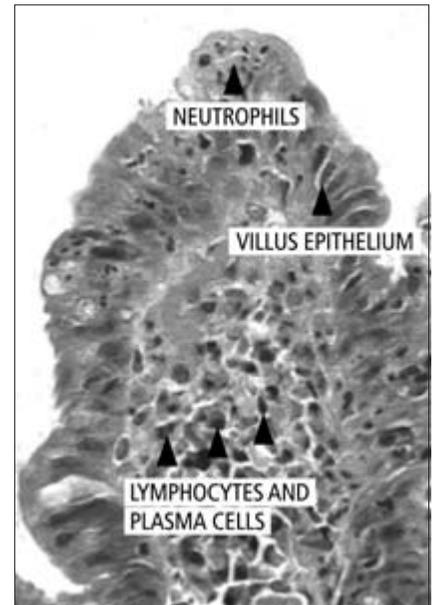
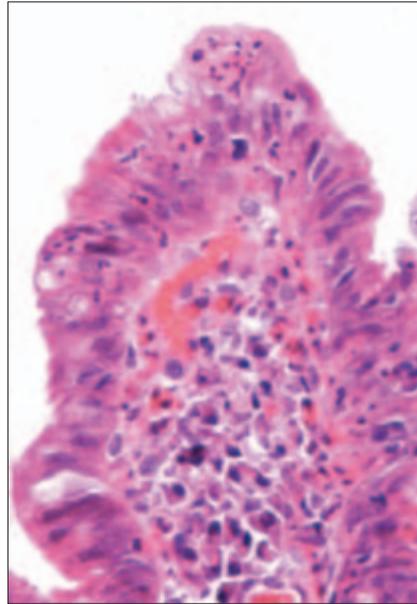
## Duodenitis

### Duodenitis

Frequently seen nonspecific minor abnormal mucosal appearances in the duodenal bulb and occasionally in the descending duodenum are by common usage described as 'duodenitis'. Biopsy material usually confirms the presence of inflammation (Fig. 2.478), although the correlation between endoscopic and histological appearances, as in 'gastritis', is less than perfect. When erythema alone is seen histological appearances can be normal, and by contrast an apparently normal mucosa may reveal histological evidence of inflammation.

Figure 2.479 illustrates what has been called 'the bumpy, blotchy bulb', that is, scattered reddened slightly raised noneroded patches in the bulb. In 'pepper-and-salt' or 'salami' duodenitis there are raised erythematous areas with small white erosions and, often, red spots, probably representing tiny petechiae (Figs 2.480 and 2.481).

Although the endoscopic appearances of duodenitis are usually confined to the bulb, they can extend into the second part. The ridges of the folds in Figs 2.482 and 2.483 are patchily erythematous.

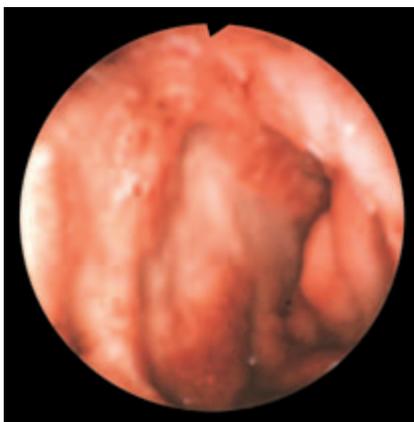


2.478

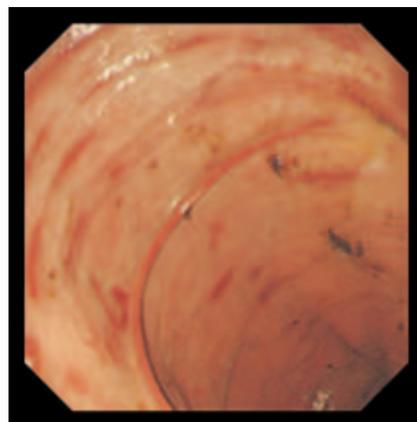


2.479

2.480



2.481



2.482



2.483

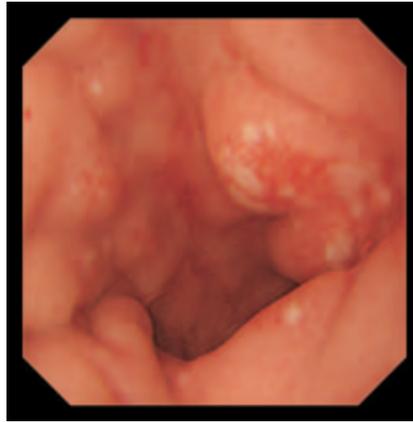
**Duodenitis (cont.)**

The ridges shown in Fig. 2.484 demonstrate superficial erosions.

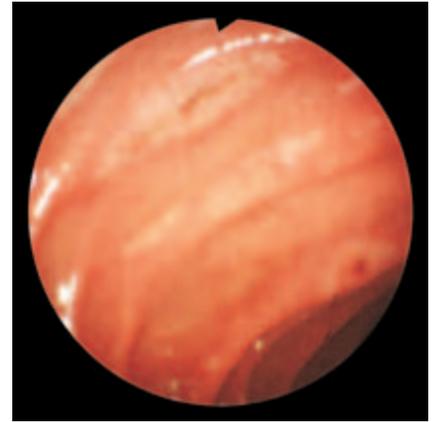
The erosions in Figs 2.485 and 2.486 are different in appearance, and reminiscent of a variant seen in the stomach.

The clinical significance of duodenitis is uncertain. In some patients with *Helicobacter pylori* it appears to be part of the peptic ulcer syndrome, preceding the development of a duodenal ulcer or persisting after the ulcer has healed, whilst in others it is apparently asymptomatic. Many patients with duodenitis have 'nonulcer dyspepsia', though the association may be fortuitous.

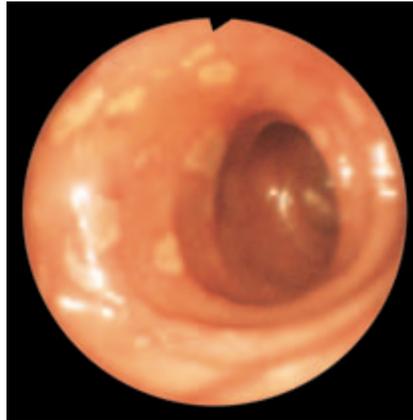
It is unusual to see non-*H. pylori* related duodenitis with a known cause. The lesions shown in Figs 2.487 and 2.488 were due to NSAIDs.



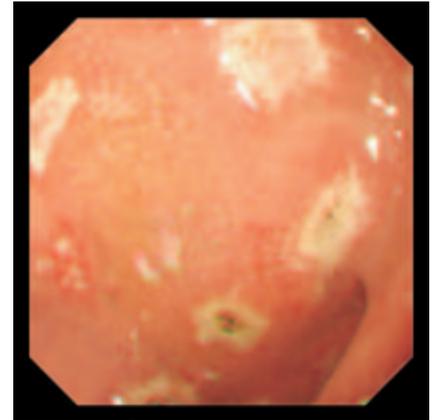
2.484



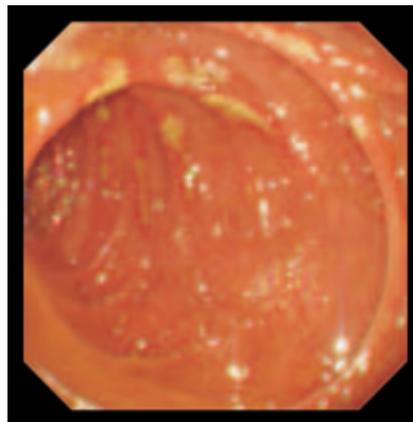
2.485



2.486



2.487



2.488

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## Peptic ulcer

### Cleaning the ulcer

### Shape, size and position

### Tell-tale fold

### Multiple ulcers

### Ulcer with duodenitis

### Ulcer and oedema

### Healing and scarring

### Supravital staining

### Cleaning the ulcer

In Fig. 2.489 bile-stained fluid has refluxed from the second part of the duodenum into the bulb. When this was aspirated, it became obvious that a small bulbar duodenal ulcer (Fig. 2.490) had been obscured by bile and an air bubble. Duodenitis is also present.

Obtaining a clear view, measuring and sampling are discussed more fully on pp. 103–104.



2.489



2.490

### Shape, size and position

There is considerable variation in shape and size of duodenal ulcers, as with benign ulcers elsewhere in the upper gastrointestinal tract. They may occur anywhere in the duodenal bulb and, much less commonly, in the second part of the duodenum.

Figure 2.491 shows that an apical ulcer can often be seen through the pylorus; this ulcer, which has an irregular outline, is again illustrated in Fig. 2.492 after the endoscope has been advanced into the duodenal bulb.



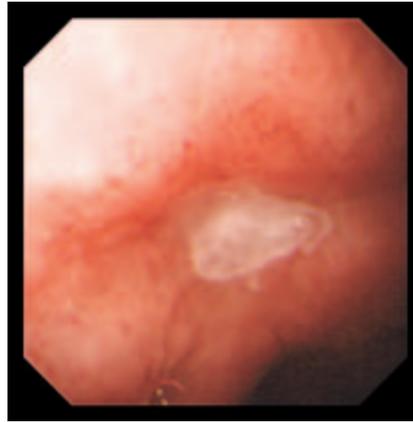
2.491



2.492

**Shape, size and position (cont.)**

Duodenal ulcers may assume any shape, varying from round and regular to grossly irregular. Figure 2.493 shows a superficial ovoid ulcer, while the ulcer portrayed in Fig. 2.494 is bigger and deeper. The small ulcer shown in Fig. 2.495 is surrounded by punctate erythema, a common finding. Figure 2.496 shows an irregular chronic duodenal ulcer. Because of its size it is difficult to record a giant ulcer but Fig. 2.497 represents an attempt. It is likely that the scarring which appears in Fig. 2.498 resulted from previous ulceration, now healed; a small recurrent ulcer is also shown.



2.493



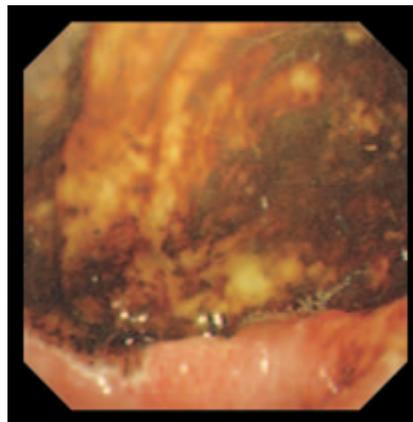
2.494



2.495



2.496



2.497



2.498

### Tell-tale fold

Ulceration is often accompanied by the formation of a tell-tale fold or guideline: Fig. 2.499 shows such a 'guideline' seen through the pylorus. When followed, it leads to a typical round duodenal ulcer (Figs 2.500 and 2.501) with a regular, swollen, smooth hyperaemic edge (Fig. 2.502). The fold is probably the result of traction on the mucosa due to ulcer scarring.



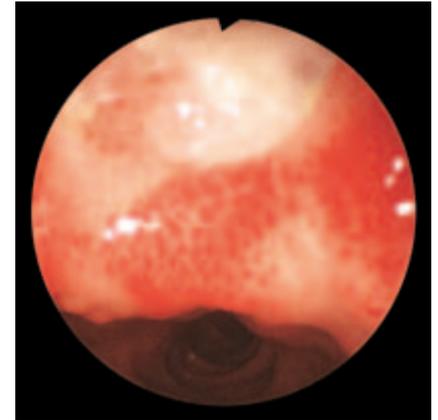
2.499



2.500



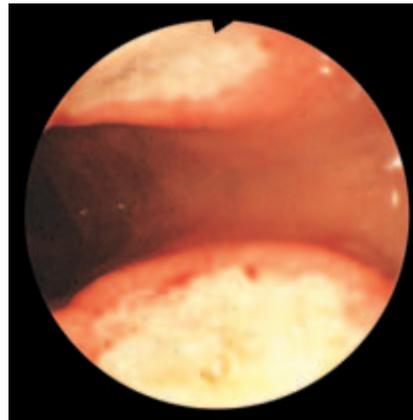
2.501



2.502

### Multiple ulcers

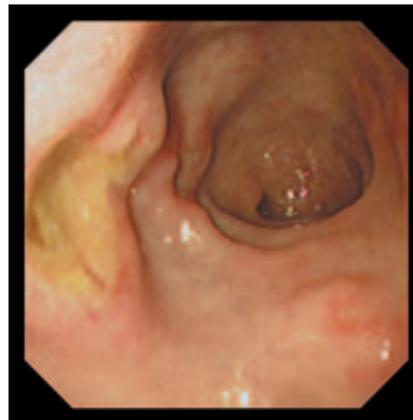
It is not uncommon to find more than one chronic ulcer; in this patient there were two ulcers (Fig. 2.503). When situated on opposite walls of the bulb they are sometimes referred to as 'kissing ulcers'. Multiple ulcers commonly occur in the Zollinger–Ellison syndrome, which should be considered when ulceration is seen distal to the duodenal bulb.



2.503

### Ulcer with duodenitis

Duodenitis may accompany peptic ulceration without (Fig. 2.504) or with a history of NSAID ingestion (Fig. 2.505). The significance of this association is uncertain. There is no predictable relationship between the healing of the ulcer and of the duodenitis. Sometimes both respond simultaneously to treatment, yet duodenitis often persists when the ulcer has healed. This may predispose to earlier recurrence of the ulcer if the underlying cause has not been eliminated.



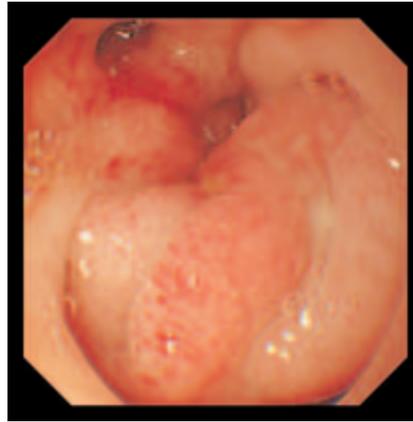
2.504



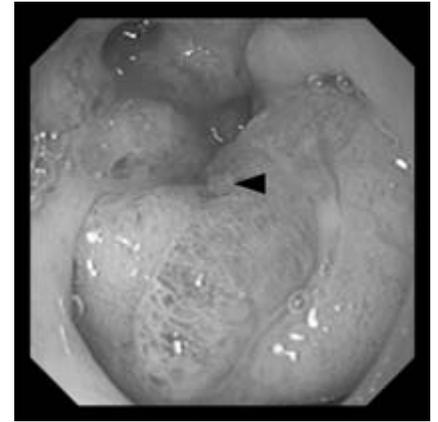
2.505

### Ulcer and oedema

Active chronic ulceration is often accompanied by considerable oedema. Figure 2.506 shows a small apical ulcer with disproportionate oedema. In Fig. 2.507 the ulcer base is unusually oedematous, while in Fig. 2.508, although the ulcer itself is very small and possibly healing, there is a considerable amount of oedema involving the whole of the duodenal apex.



2.506



2.507



2.508

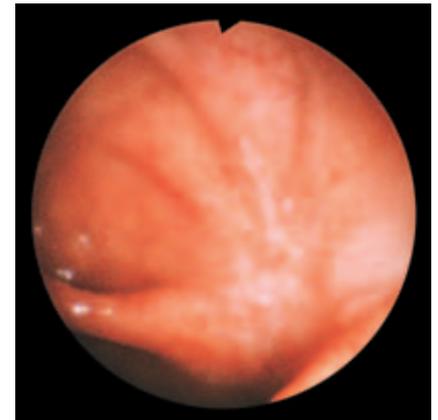
### Healing and scarring

Figure 2.509 shows a small shallow apical healing ulcer. In Fig. 2.510 there is stellate scarring with minimal persisting ulceration.

The crescentic ulcer shown in Fig. 2.511 is partially healed while that in Fig. 2.512 has arisen on an old scar.



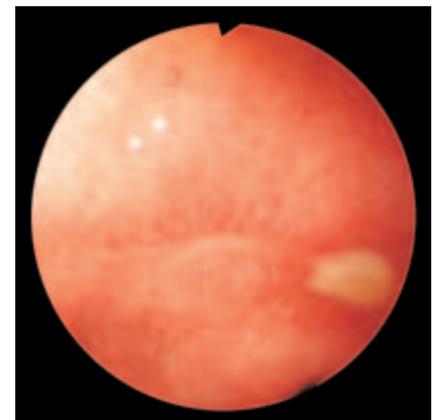
2.509



2.510



2.511

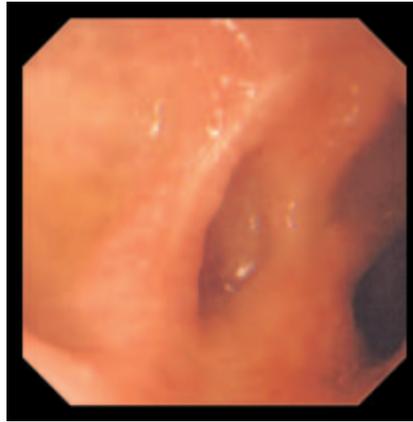


2.512

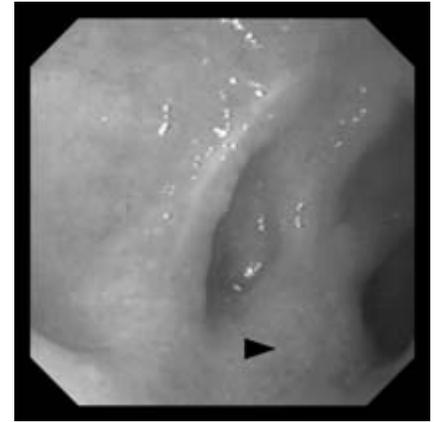
### Healing and scarring (cont.)

The duodenal bulb in Fig. 2.513 shows duodenitis and extensive old scarring.

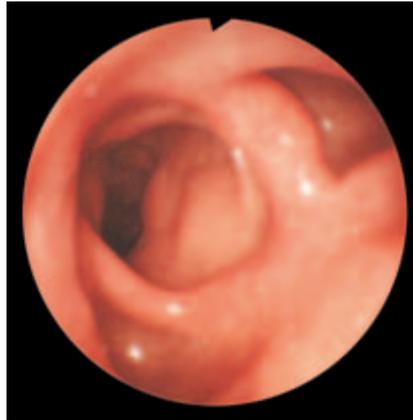
In Figs 2.514–2.516 no ulcer is present but severe scarring has led to various patterns of pseudodiverticulum formation. Such features explain the trefoil deformity characteristic of chronic duodenal ulceration as shown by barium meal radiology.



2.513



2.514



2.515

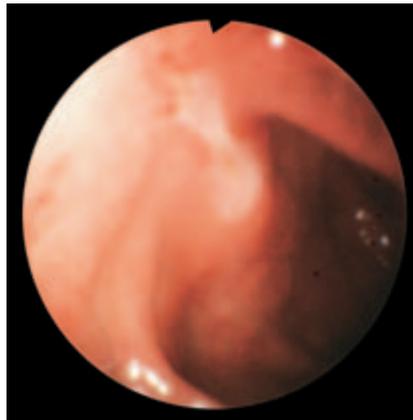


2.516

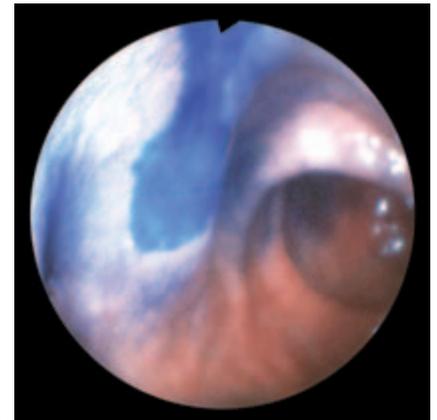
### Supravital staining

In routine clinical practice as opposed to clinical trials it is not necessary to check endoscopically or radiologically for healing of duodenal ulcer if symptoms have disappeared and the full course of medical treatment has been completed. If endoscopic follow-up is indicated supravital staining may help distinguish an ulcer scar from an incompletely healed ulcer.

Methylene blue can be used to delineate an ulcer as the dye is preferentially absorbed by slough. Figs 2.517a and 2.517b respectively show the stained and unstained appearances.



2.517a



2.517b

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## Testing for the presence of *Helicobacter pylori*

### Testing for the presence of *Helicobacter pylori*

As duodenal ulceration (and to a lesser extent gastric ulceration) is usually associated with colonization of the stomach by *H. pylori* diagnostic procedures to demonstrate this are routine. The simplest method is the rapid urease test in which a gastric antral biopsy is inserted into the agar well of a CLOtest®. In this context, CLO is an acronym for *Campylobacter*-like organism, a superseded name for *Helicobacter pylori*. A heavy organism load gives a rapid positive result within minutes, while the final interpretation should be delayed for 24 h. Figure 2.518 shows the yellow appearance of a negative test and the magenta appearance of a positive result. The 'gold standard' for demonstrating *H. pylori* in the stomach remains histological (p. 85) but this is not performed routinely. Serological and breath tests are alternative diagnostic methods but will not be discussed further here.



2.518

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## Benign and malignant tumours

### Classification

#### Nonspecific appearances of polypoid lesions

#### Adenomas

#### Familial adenomatous polyposis (FAP)

#### Gastric heterotopia

#### Gastric metaplasia

#### Hyperplasia of Brunner's glands

#### Peutz–Jeghers polyps

#### Juvenile polyps

#### Carcinoma

#### Invading extra-duodenal carcinoma

#### Lymphoma

#### Gastrointestinal stromal tumours

### Classification

This appears in Tables 2.11 and 2.12.

**Table 2.11** Benign and malignant epithelial tumours of the small bowel (adapted from Day *et al.*, 1990)

#### Benign epithelial tumours

- Adenomas
- Familial adenomatous polyposis
- Gastric heterotopia
- Gastric metaplasia
- Hamartomas and hamartomatous polyps
  - Hamartomas of Brunner's glands
  - Peutz–Jeghers syndrome
  - Juvenile polyposis syndrome
  - Cronkhite–Canada syndrome

#### Malignant epithelial tumours

- Carcinoma
- Endocrine cell tumours
- Secondary and direct spread tumours

**Table 2.12** Non-epithelial tumours of the small bowel (adapted from Day *et al.*, 1990)

#### Disorders and tumours of lymphoid tissue

- Lymphoid hyperplasia of the terminal ileum
- Lymphoma
- Leukaemia

#### Gastrointestinal stromal tumours (GISTs)

#### Fibromas

#### Neurogenic tumours

#### Lipomas

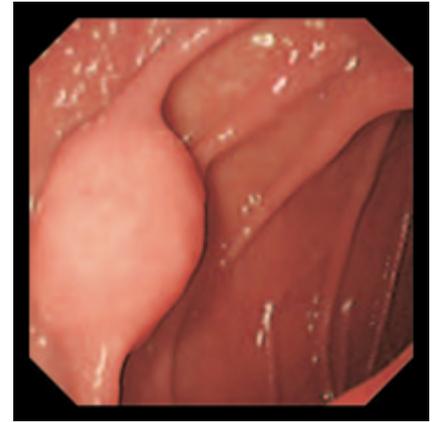
#### Others

**Nonspecific appearances of polypoid lesions**

As elsewhere in the gastrointestinal tract, polyps without specific distinguishing features may be found in the duodenum (Fig. 2.519). A submucosal polyp, shown in Fig. 2.520 is reminiscent of a similar lesion seen in the stomach (Fig. 2.349). The nature of duodenal polyps as elsewhere can only be determined by the pathologist. Caution is required as polypectomy in this region may be more hazardous than in other parts of the bowel.



2.519



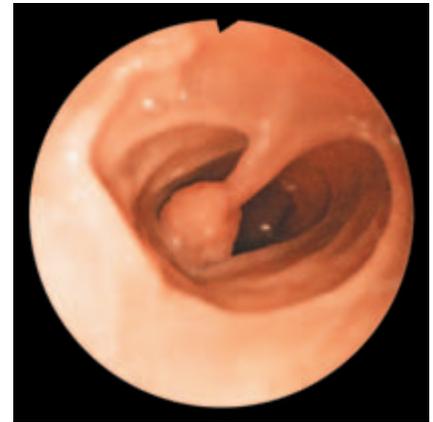
2.520

**Adenomas**

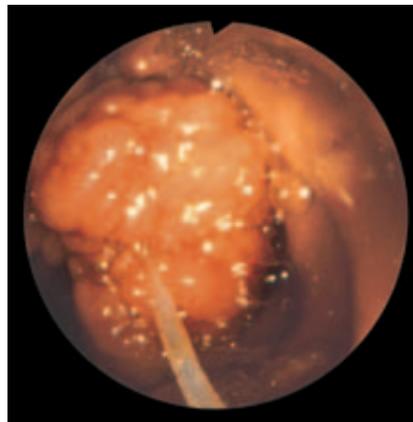
Not surprisingly, the varying appearance of adenomatous polyps of the duodenum, where they are rare, mirrors the pattern seen in other parts of the gastrointestinal tract. Such polyps may be small, smooth and sessile (Fig. 2.521), pedunculated (Fig. 2.522), or large or irregular in shape (Fig. 2.523). Figure 2.524 shows a fairly extensive sessile villous adenoma.



2.521



2.522



2.523



2.524

**Familial adenomatous polyposis (FAP)**

Multiple duodenal polyps (Fig. 2.525) may be found in patients with FAP (where the emphasis is usually on colonic polyposis). These duodenal adenomas have a significant malignant potential and endoscopic removal should be considered. It is suggested that patients with FAP should be routinely screened by OGD, aiming at cancer prevention.

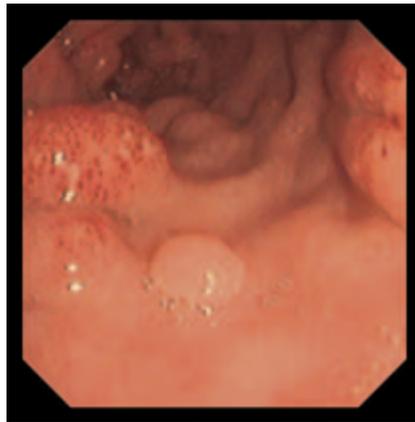


2.525

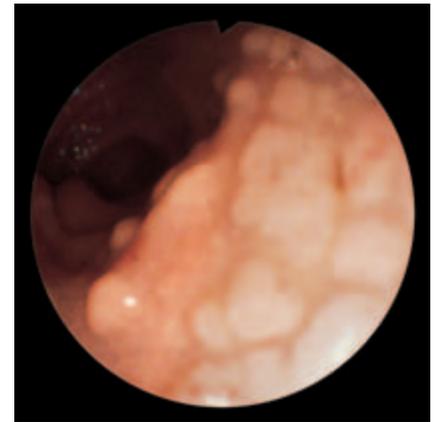
**Gastric heterotopia**

Such lesions are found not uncommonly in the duodenum. They may be single (Fig. 2.526) or multiple (Fig. 2.527) and are usually polypoid and well circumscribed. Figure 2.528 shows the histological appearances of gastric heterotopia.

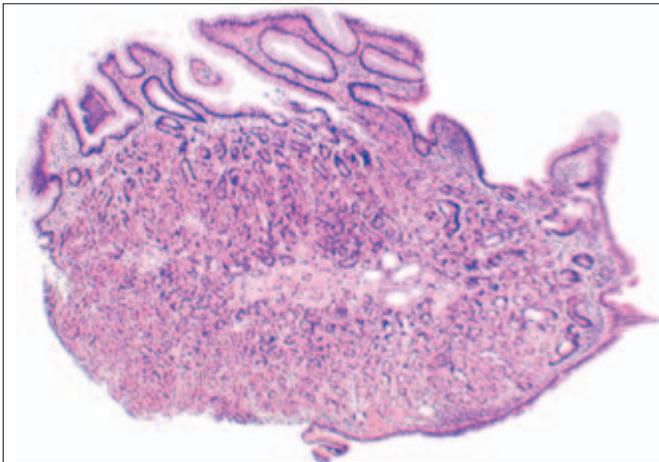
Rarely, cysts may develop as a direct result of gastric heterotopia (Fig. 2.529).



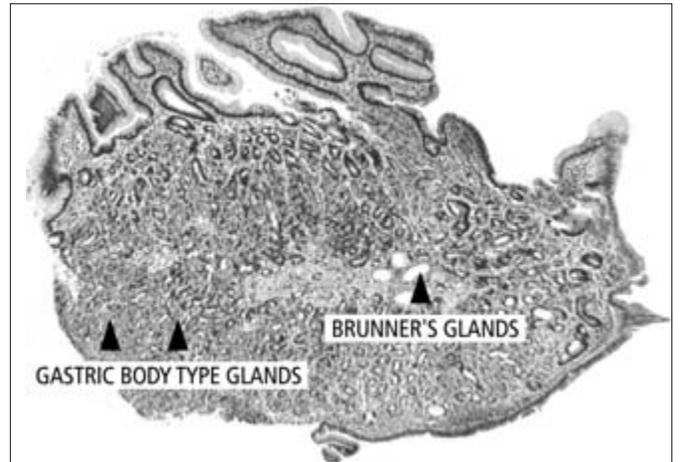
2.526



2.527



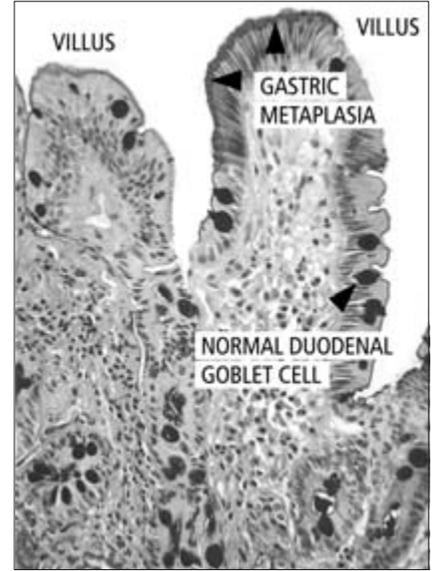
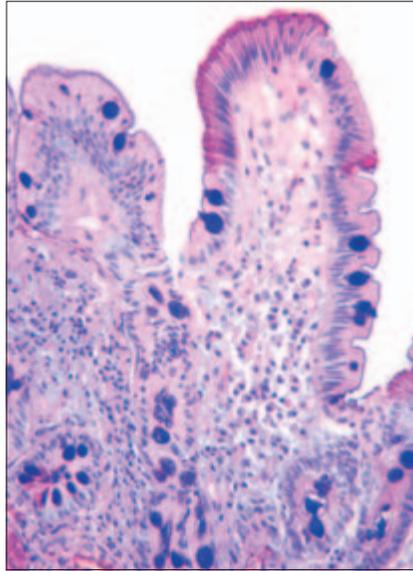
2.528



2.529

### Gastric metaplasia

In contrast to gastric heterotopia, gastric metaplasia in the duodenum is not usually visible endoscopically. The gastric-type epithelial cells have a mucin component with a finger nail like shape and neutral (pink) mucin by Alcian blue/PAS staining (Fig. 2.530).



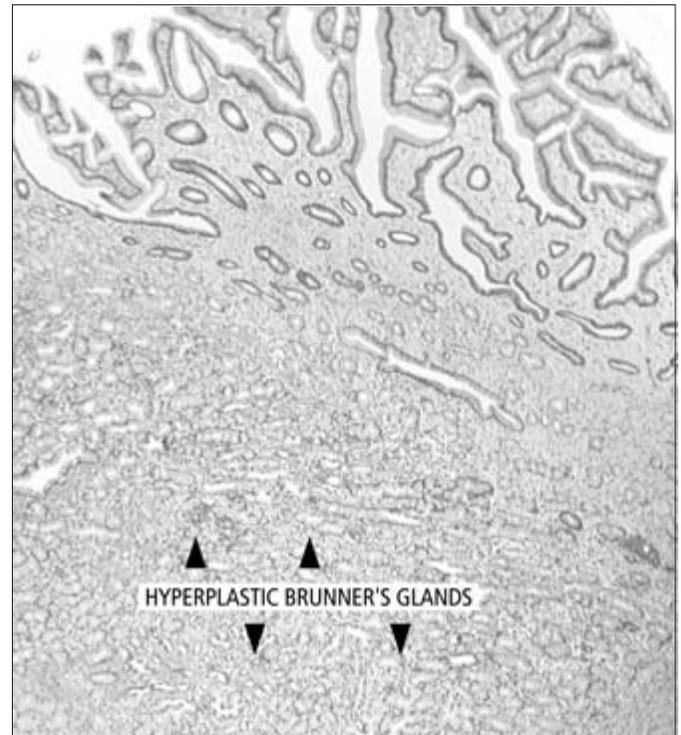
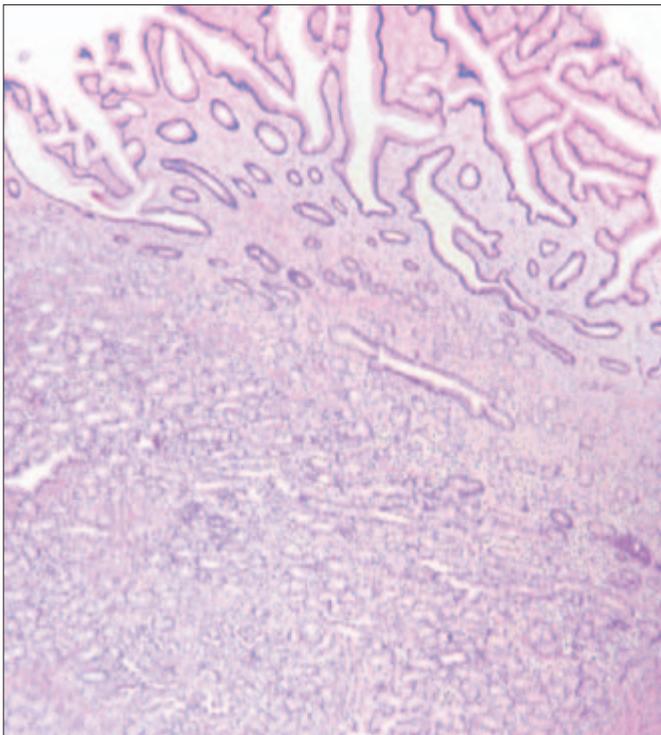
2.530

### Hyperplasia of Brunner's glands

These lesions are usually small, and may be single (Fig. 2.531) or multiple, and are commoner in the second part of the duodenum. The histopathology appears in Fig. 2.532.



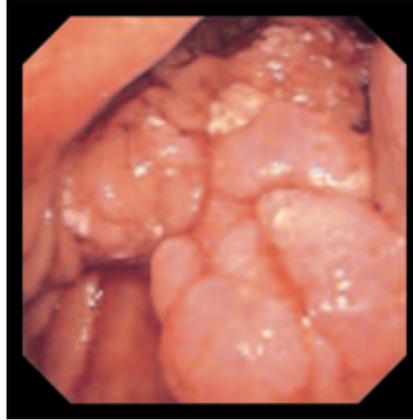
2.531



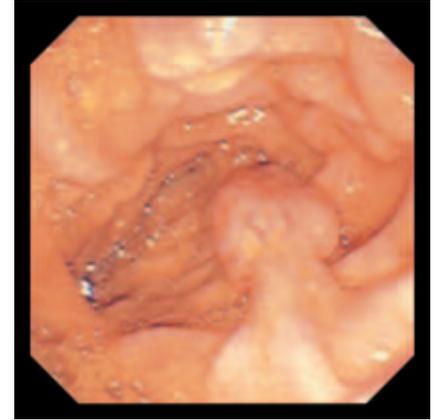
2.532

**Peutz–Jeghers polyps**

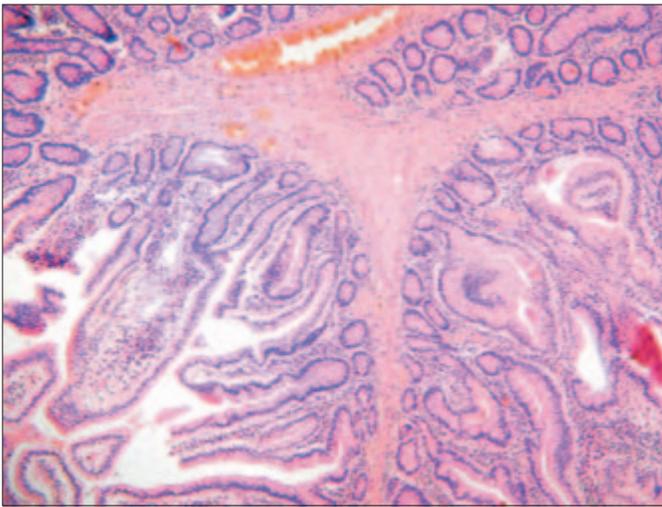
Polyps of this type, which may also occur in many other sites in the gastrointestinal tract, have no specific endoscopic features (Figs 2.533 and 2.534). Figure 2.535 shows their histology.



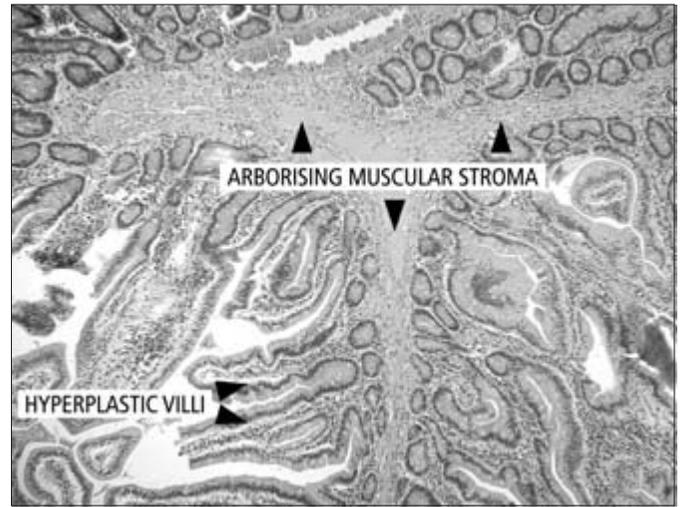
2.533



2.534

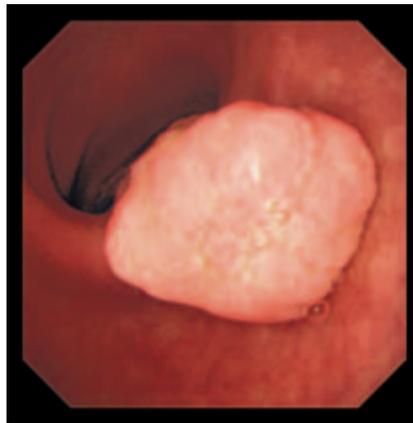


2.535



**Juvenile polyps**

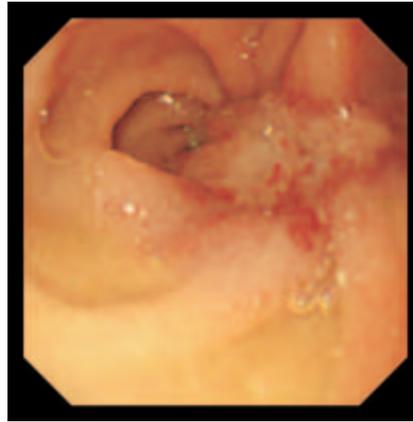
In the duodenum (Fig. 2.536) these polyps do not have the mottled surface often seen in colonic juvenile polyps (Figs 3.125 and 3.126).



2.536

### Carcinoma

Primary carcinoma of the duodenum (Figs 2.537 and 2.538) is rare though the incidence per unit length of bowel is similar throughout the small intestine. The endoscopic appearances are variable as is to be expected. It is not usually possible at routine OGD using a standard instrument to advance beyond the ligament of Treitz. Figure 2.539 however, shows a jejunal carcinoma recorded in this way (see also Chapter 5). The appearances in Fig. 2.539 are unusual: there are granular plaques of malignant tissue, hence the multiple highlights.



2.537



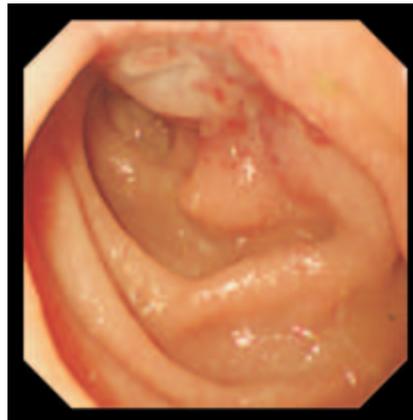
2.538



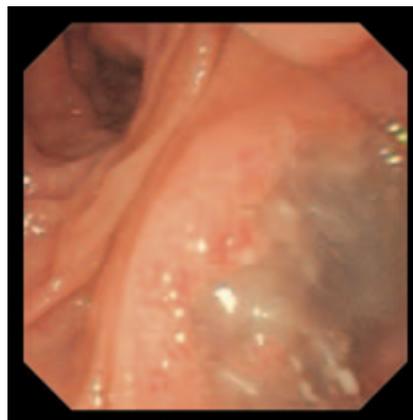
2.539

### Invading extra-duodenal carcinoma

Figure 2.540 shows invasion of the duodenum by pancreatic carcinoma, while in Fig. 2.541 the duodenal wall has been penetrated by a mucus secreting pancreatic adenocarcinoma.



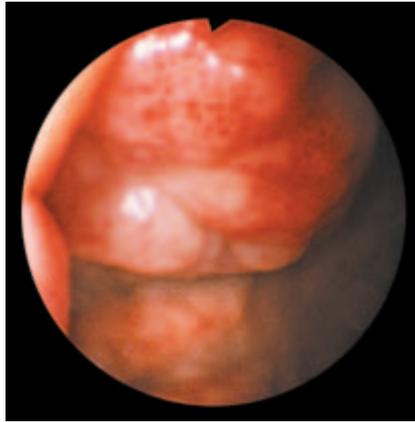
2.540



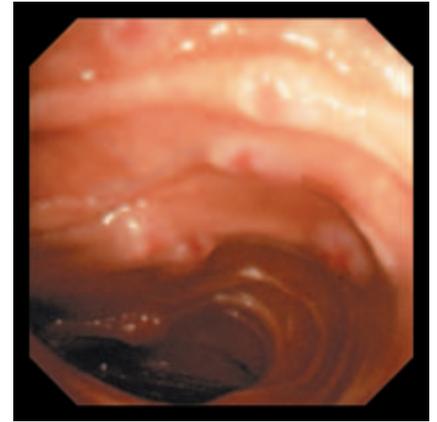
2.541

**Lymphoma**

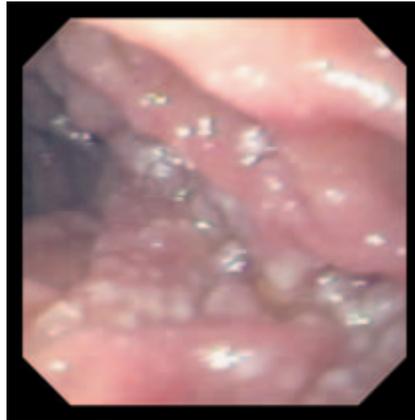
Figure 2.542 demonstrates an exuberant mass of lymphomatous tissue encroaching on the duodenal lumen. Other appearances sometimes encountered include widespread nodularity and discrete ulceration (Fig. 2.543); this patient suffered from AIDS. The nodular lesion shown in Fig. 2.544 was diagnosed as a MALT duodenal lymphoma. The histology of a high grade duodenal lymphoma appears in Fig. 2.545.



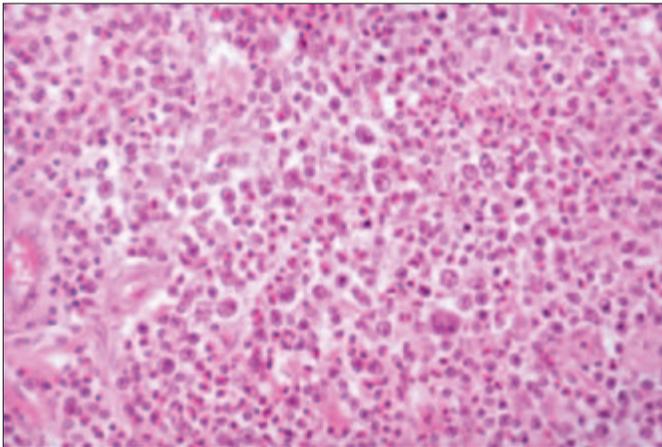
2.542



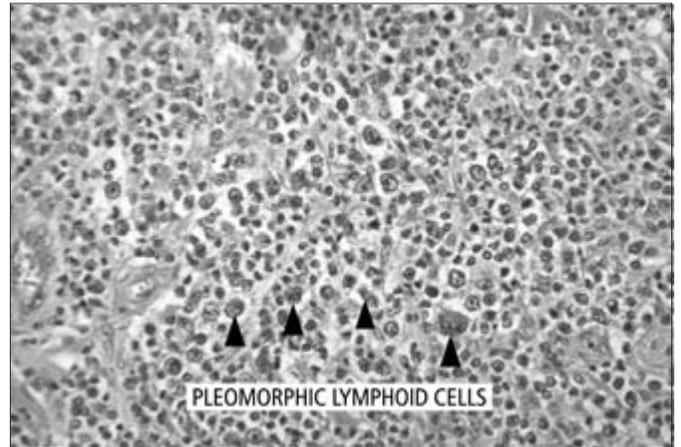
2.543



2.544



2.545



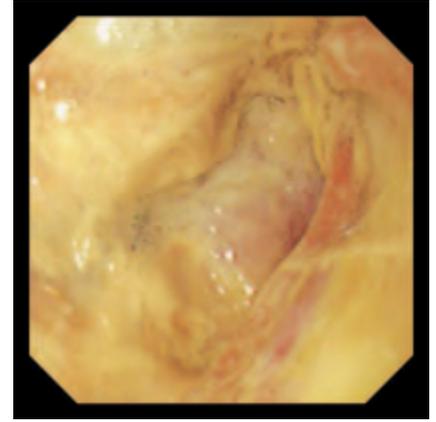
**Lymphoma (cont.)**

Coeliac disease may be complicated by T cell lymphoma. Figure 2.546 shows the upper edge of such a lesion in the duodenal cap, and Fig. 2.547 the same tumour in the descending duodenum. This condition is known as enteropathy-associated T cell lymphoma or EATL. The histopathology is shown in Fig. 2.548.

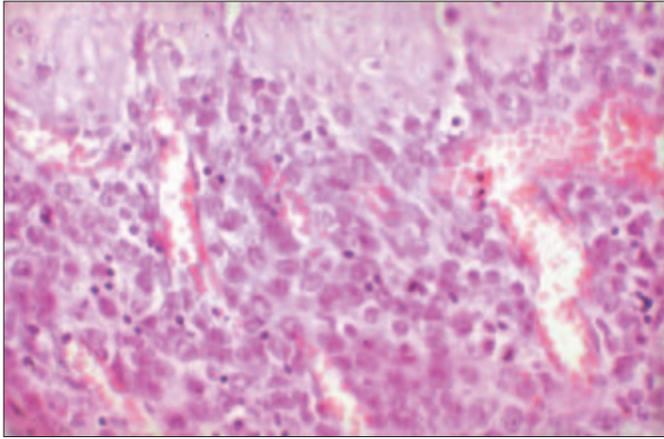
Gastrointestinal lymphoma is more fully discussed on pp. 121–123.



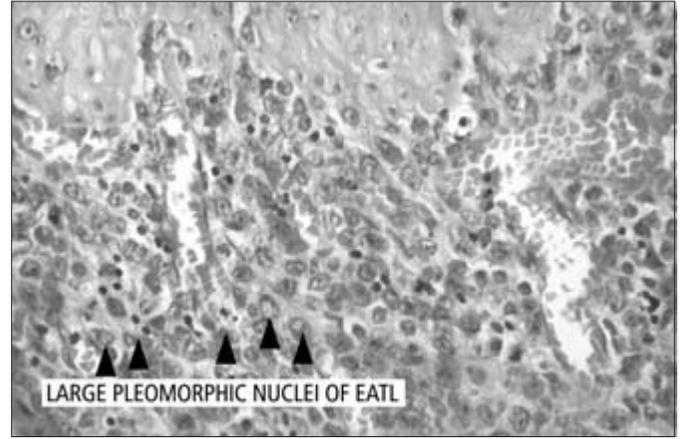
2.546



2.547

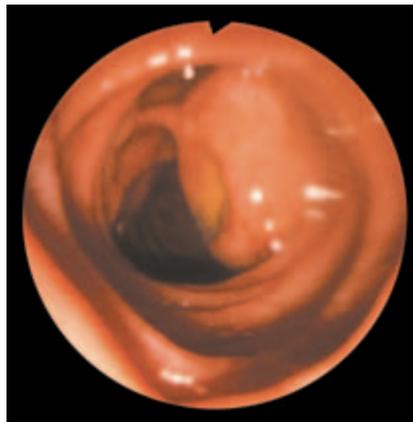


2.548

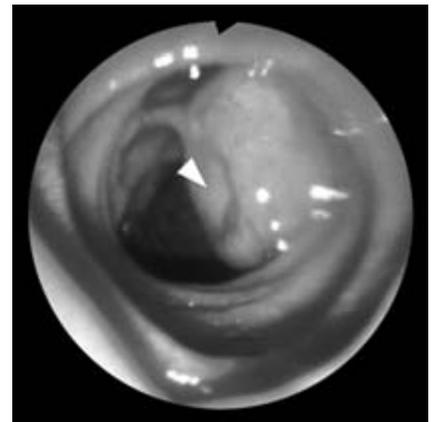


**Gastrointestinal stromal tumours**

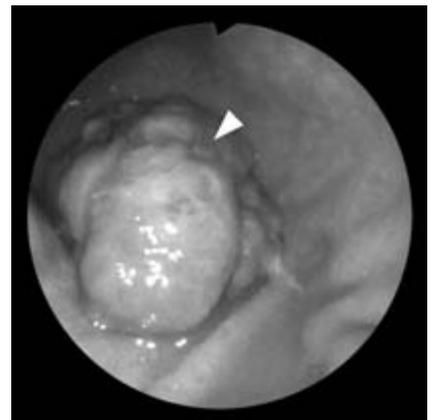
The characteristics of duodenal GISTs (Figs 2.549 and 2.550) are similar to those already described. A fuller discussion appears on pp. 124–126.



2.549



2.550



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## Coeliac disease and associated conditions

### Coeliac disease

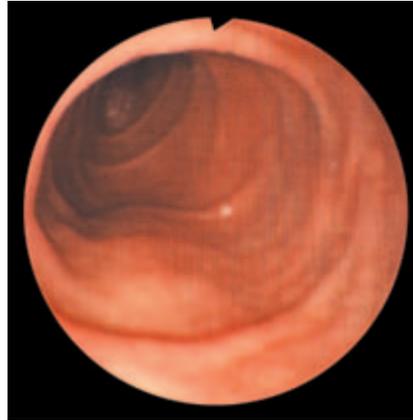
#### Ulcerative jejuno-ileitis

#### Common variable immunodeficiency

#### Enteropathy associated T cell lymphoma

### Coeliac disease

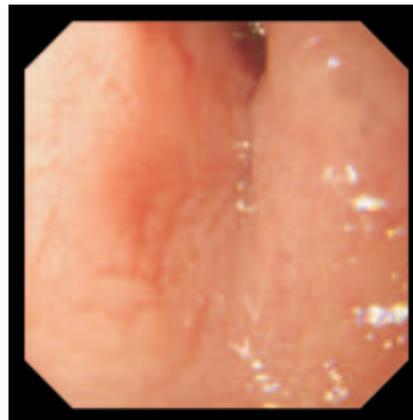
The mucosa often appears featureless (Fig. 2.551) and the mucosal folds may be scalloped (Fig. 2.552). The mosaic pattern familiar from the dissecting microscope is well seen in Figs 2.553 and 2.554. Supravital staining with methylene blue may lead to easier recognition (Fig. 2.555).



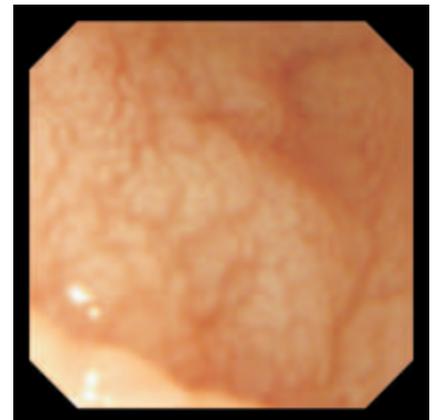
2.551



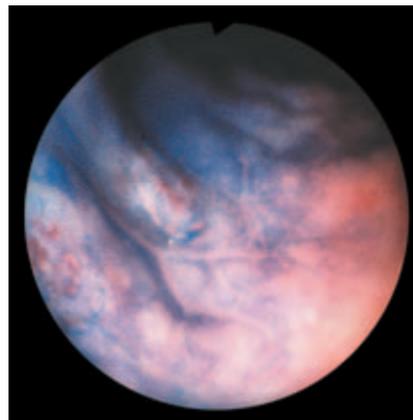
2.552



2.553



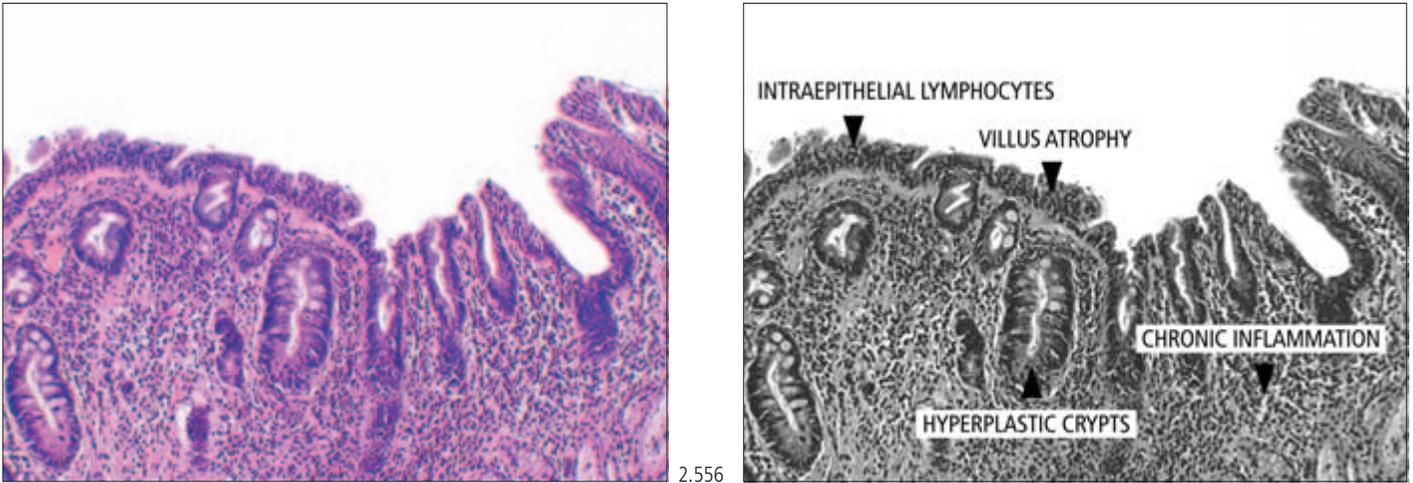
2.554



2.555

**Coeliac disease (cont.)**

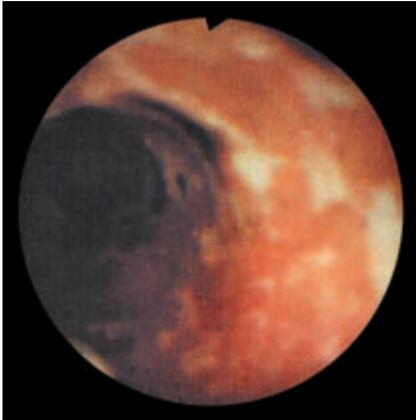
The characteristic histopathological appearances are shown in Fig. 2.556. Four biopsies are necessary since the histological changes of coeliac disease in the duodenum may be patchy, especially when associated with dermatitis herpetiformis.



2.556

**Ulcerative jejuno-ileitis**

Figure 2.557 illustrates the appearances in ulcerative jejuno-ileitis, sometimes seen in coeliac disease. In this case the patient responded to a strict gluten-free diet. This is not so when such lesions are due to enteropathy-associated T cell lymphoma (see p. 157).



2.557

**Common variable immunodeficiency**

The endoscopic appearances in common variable immunodeficiency are identical to those seen in coeliac disease (Fig. 5.37).



5.37

**Enteropathy associated T cell lymphoma**

This is discussed on p. 157.

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## Miscellaneous conditions

Crohn's disease

Systemic mastocytosis

Progressive systemic sclerosis

Henoch-Schönlein syndrome

Haemangioma

Xanthelasma

Mycobacterium avium intracellulare

Cryptosporidiosis

Lymphangiectasia

Abnormal papillary appearances

Gallstone

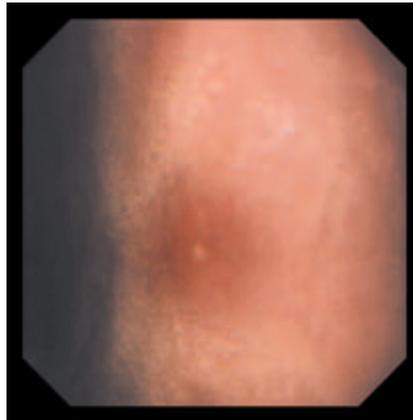
Duodenal obstruction

Hepatic impression

### Crohn's disease

Crohn's disease limited to the duodenum is rare but duodenal involvement is not uncommon when this condition affects other parts of the gastrointestinal tract.

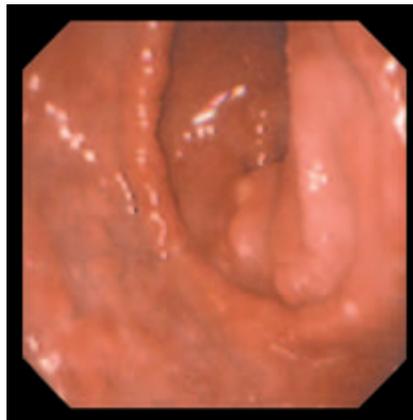
In Fig. 2.558 there is a single aphthoid ulcer. The valvulae conniventes show multiple ulcers in Fig. 2.559 and nodularity with minimal ulceration in Fig. 2.560.



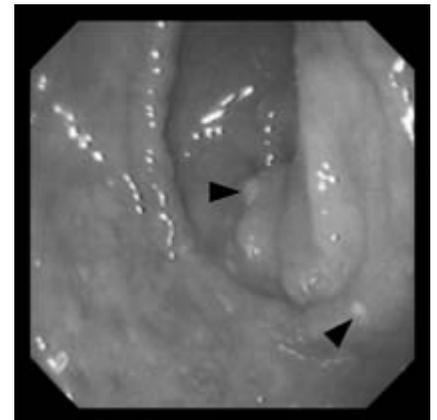
2.558



2.559

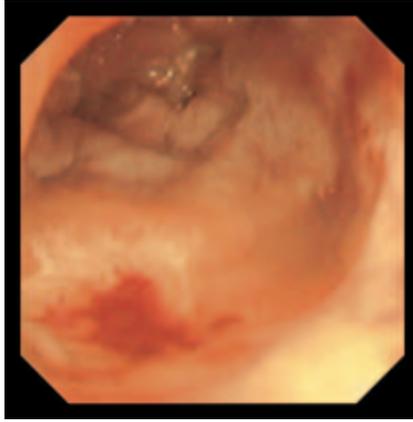


2.560



**Crohn's disease (cont.)**

There is a larger ulcer in Fig. 2.561, while Fig. 2.562 shows a duodenocolic fistula.



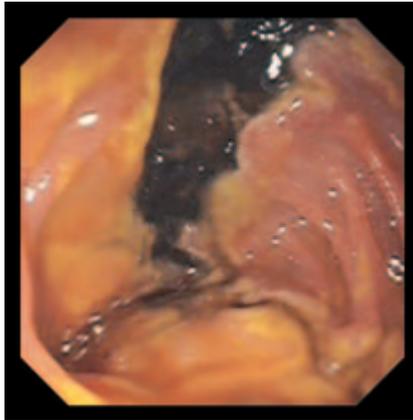
2.561



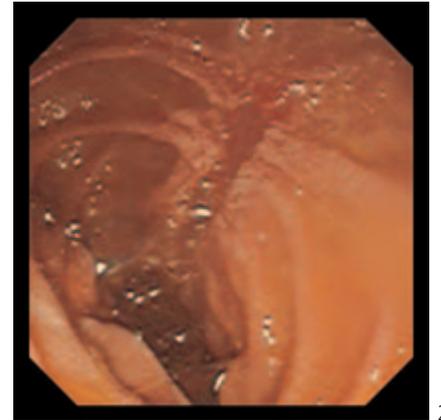
2.562

**Systemic mastocytosis**

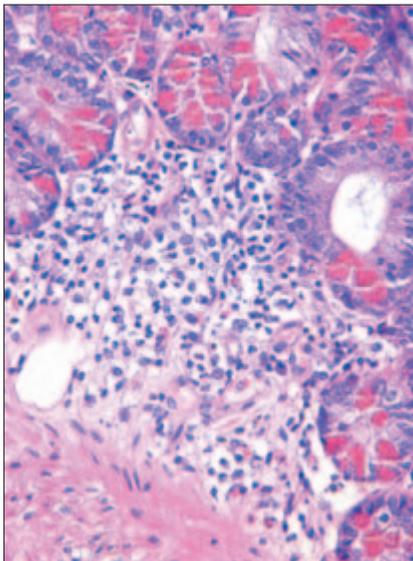
Figure 2.563 demonstrates postbulbar ulceration in this condition. The lesion healed (Fig. 2.564) following treatment with a proton pump inhibitor. The histology of systemic mastocytosis is shown in Fig. 2.565.



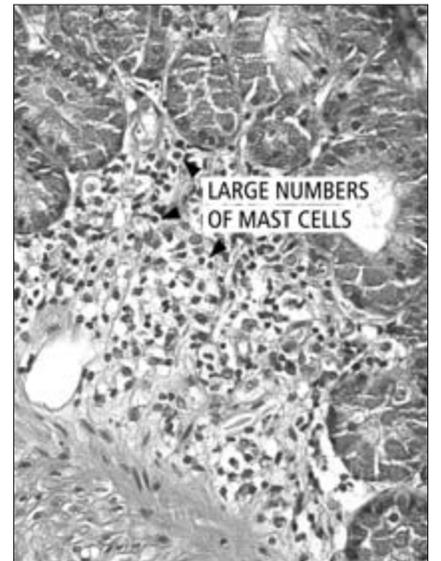
2.563



2.564

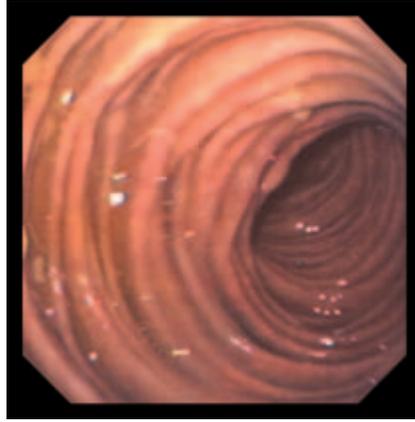


2.565



**Progressive systemic sclerosis**

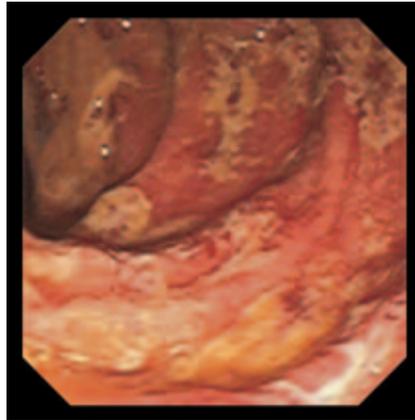
In progressive systemic sclerosis (also known as scleroderma) the pathological changes in the small bowel may lead to the endoscopic appearances shown in Fig. 2.566, suggestive of a concertina. In addition to the crowded mucosal folds, the lumen is dilated.



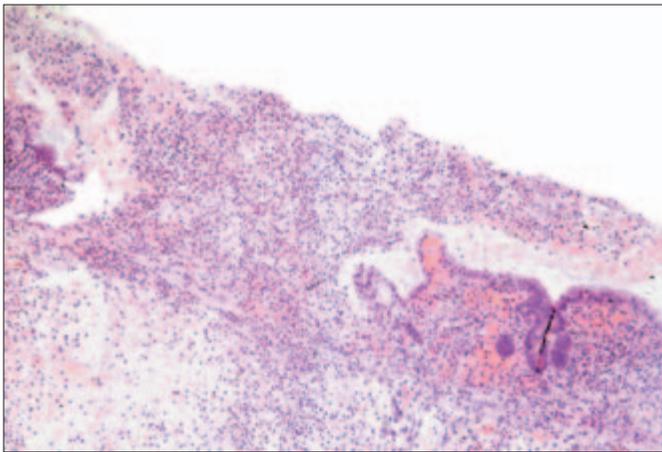
2.566

**Henoch–Schönlein syndrome**

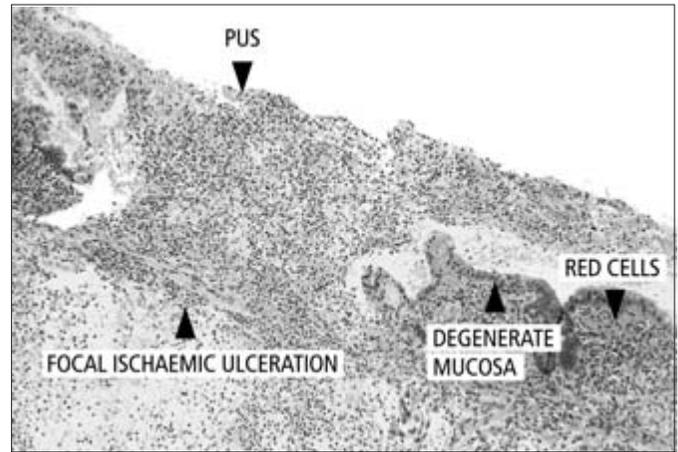
Intramural haemorrhage and superficial ulceration in the small bowel can cause intestinal blood loss. Figure 2.567 illustrates swollen ulcerated folds of the upper small bowel. At duodenoscopy the circular folds were involved to the full limits of vision. Figure 2.568 shows the histopathological appearances.



2.567

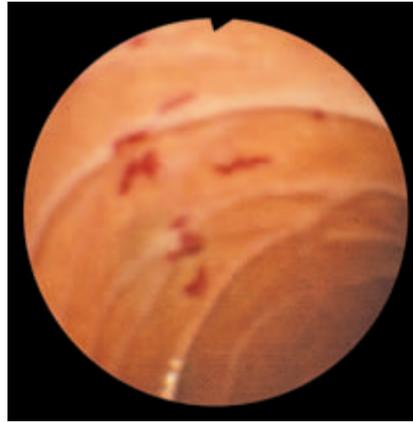


2.568



### Haemangioma

Haemangiomas appear similar in all parts of the gastrointestinal tract. In the duodenum they are often multiple (Fig. 2.569) but occasionally single. Duodenal haemangiomas are similar in appearance to those seen in the oesophagus (Fig. 2.197) and stomach (Figs 2.421 and 2.422). They may be the cause of significant upper gastrointestinal bleeding, often presenting considerable diagnostic difficulties.



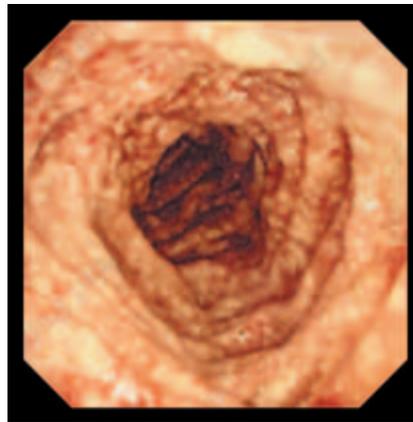
2.569

### Xanthelasma

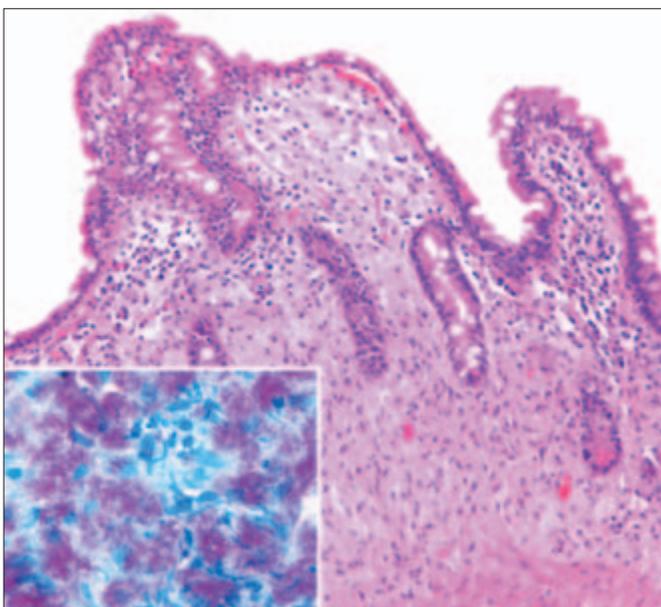
Lesions similar to those described for other parts of the gastrointestinal tract (Figs 2.236 and 2.458) are common and of no known clinical importance.

### Mycobacterium avium intracellulare

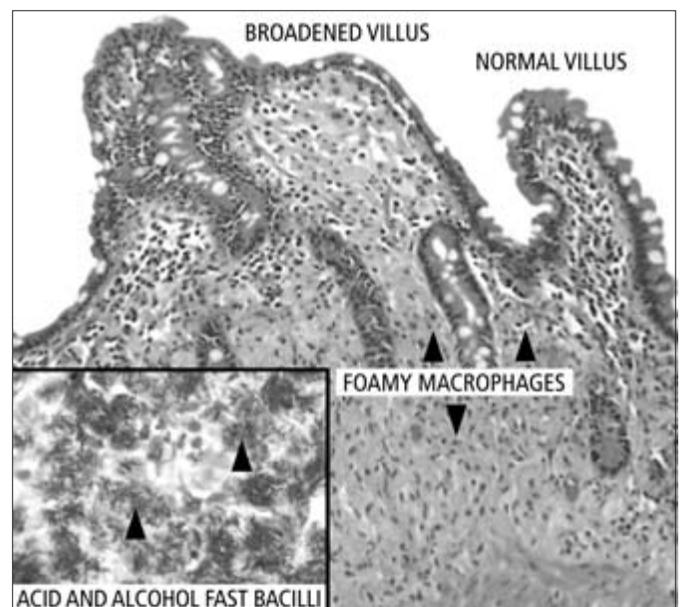
This organism is not normally pathogenic but in patients with AIDS infiltration of the submucosa by macrophages packed with bacteria may produce endoscopically visible abnormalities. The appearances of diffuse nodularity with enlarged and visible villi (Fig. 2.570) may mimic Whipple's disease. Figure 2.571 shows the typical histological features.



2.570

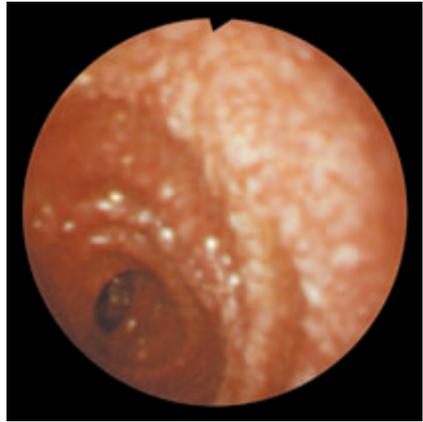


2.571

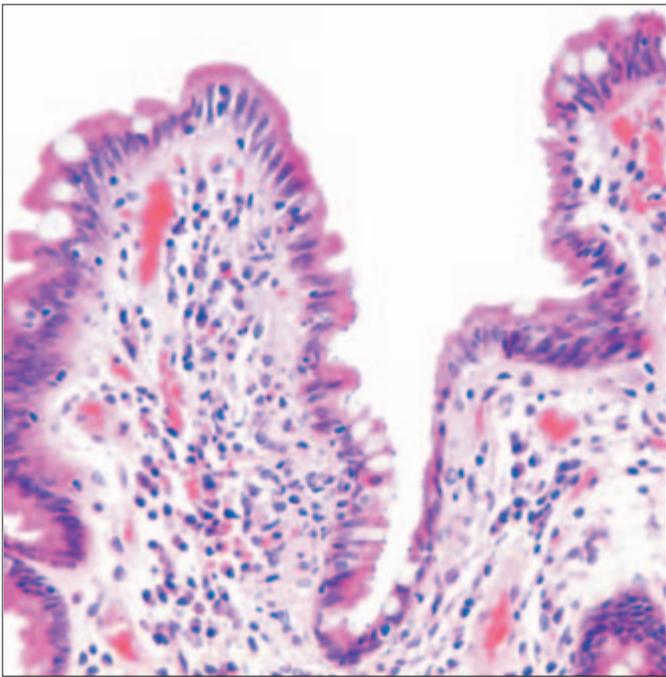


**Cryptosporidiosis**

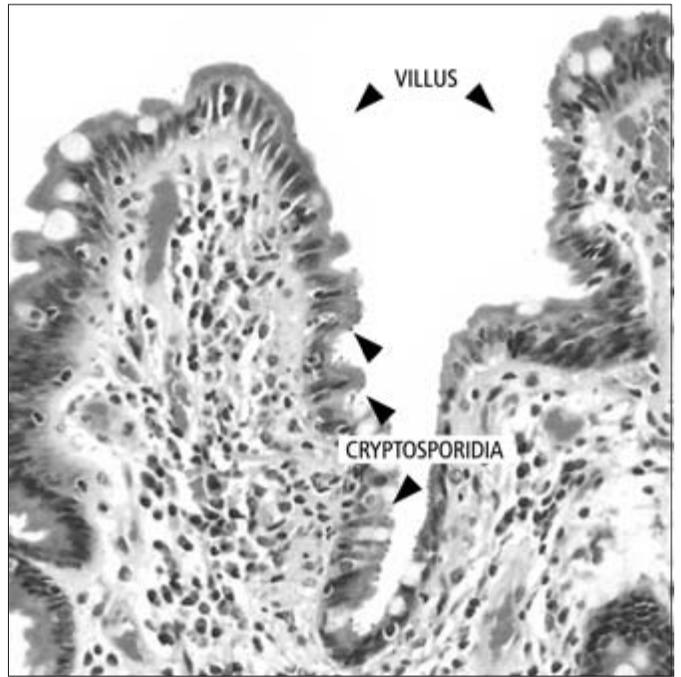
Patients with AIDS commonly acquire cryptosporidiosis. Figures 2.572 and 2.573 show the typical endoscopic and histological appearances.



2.572

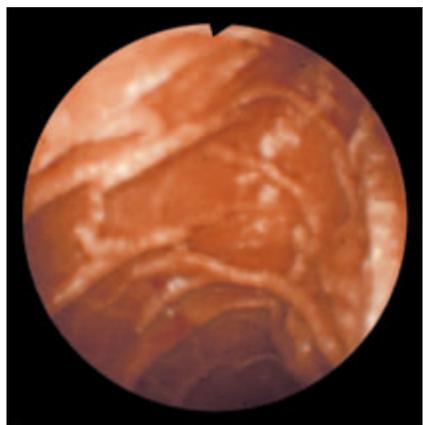


2.573

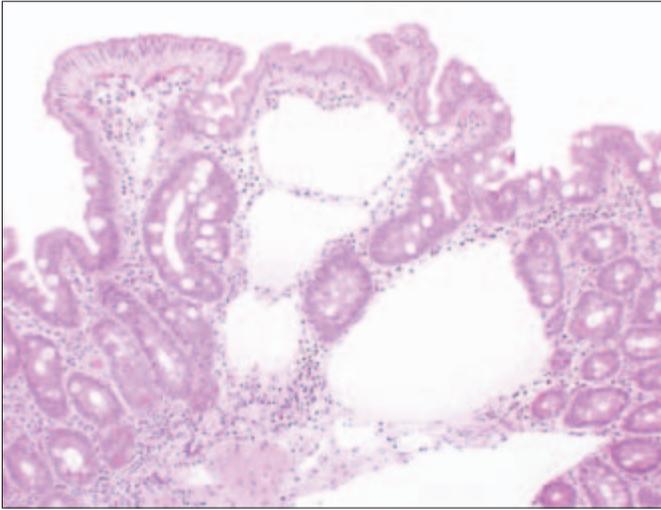


**Lymphangiectasia**

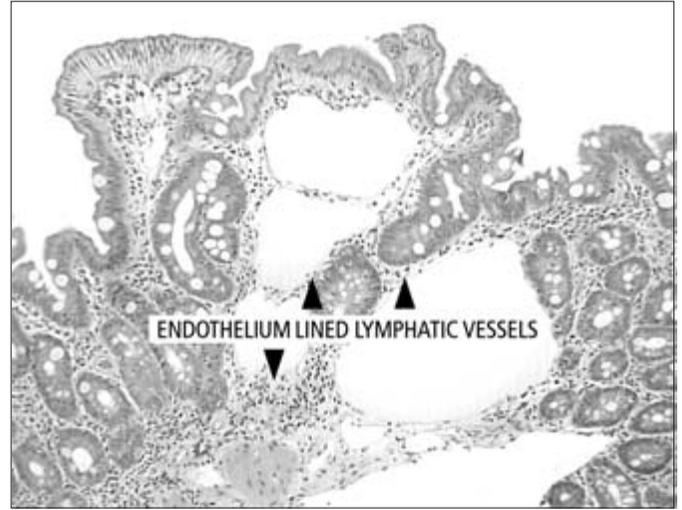
White tips to the intestinal villi are commonly seen but even after histological examination the cause is not always clear. Often this is due to endoscopically visible normal filled lacteals as shown in Fig. 2.63. Recent ingestion of fat-containing fluids or food is the likely explanation in most cases. It may in some patients be associated with obstruction to lymphatic flow.



2.63



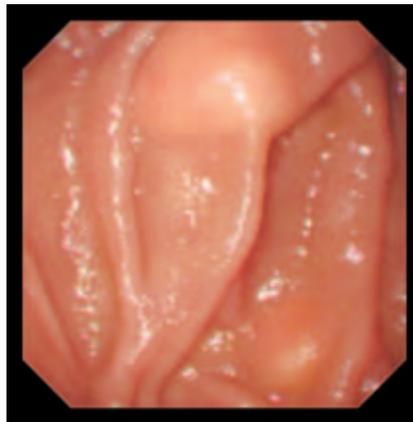
2.574



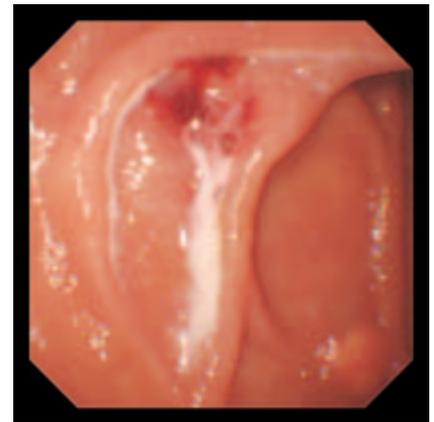
**Lymphangiectasia (cont.)**

The histopathological appearances of lymphangiectasia with endothelium-lined lymphatics is shown in Fig. 2.574.

The nature of white submucosal areas as shown in Fig. 2.575 was at first uncertain: on biopsy chyle was released (Fig. 2.576) indicating dilated lacteals or a lactocele.



2.575



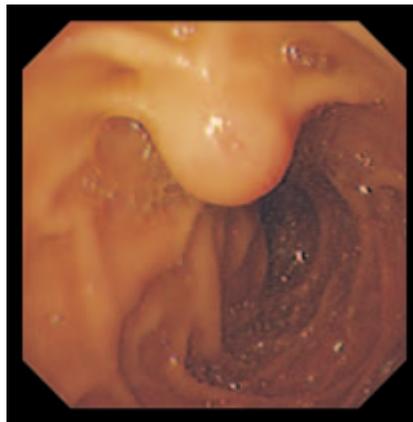
2.576

**Abnormal papillary appearances**

These are listed and discussed more fully in Chapter 4. Two examples are mentioned here in passing.

*Bulging papilla*

Comparing Fig. 2.577 with normal appearances (Figs 2.64 and 2.65) it is clear that the papilla is bulging. ERCP and sphincterotomy confirmed the cause to be a small impacted gallstone.

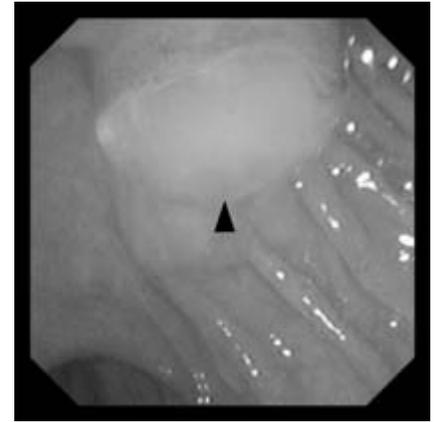
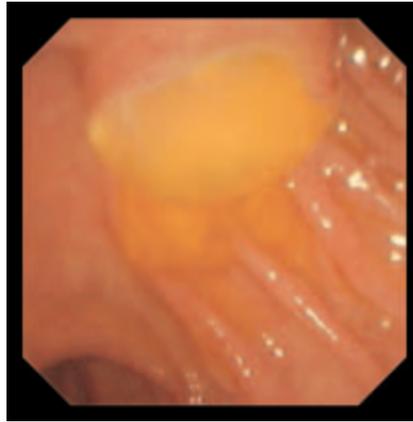


2.577

**Abnormal papillary appearances (cont.)**

*'Snotty nosed' papilla*

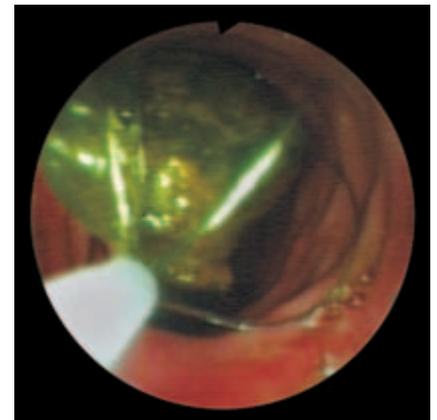
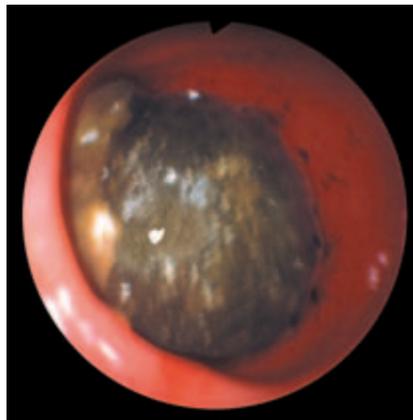
These appearances (Fig. 2.578) were observed in a patient with a mucus-secreting pancreatic carcinoma.



2.578

**Gallstone**

Rarely, a gallstone may be seen lying in the upper duodenum (Fig. 2.579), usually as a result of migration through a cholecyst-duodenal fistula. The stone seen in Fig. 2.580 was removed with a wire basket.

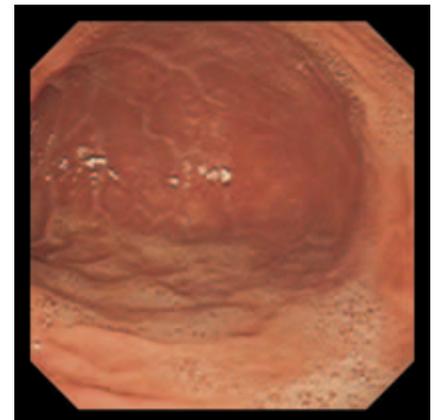
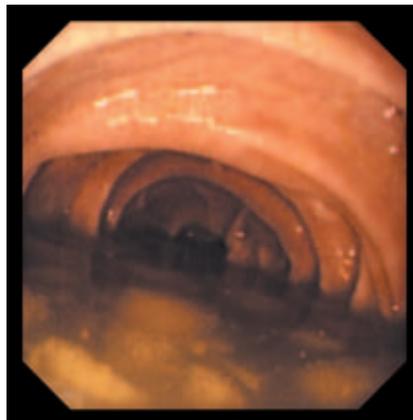


2.579

2.580

**Duodenal obstruction**

At routine endoscopy, there is normally little fluid in the upper duodenum. The presence of excess fluid (Fig. 2.581) suggests more distally lying obstruction. There may be dilatation proximal to the site of the obstruction: Fig. 2.582 illustrates the appearances of duodenal megabulbus in association with an annular pancreas.

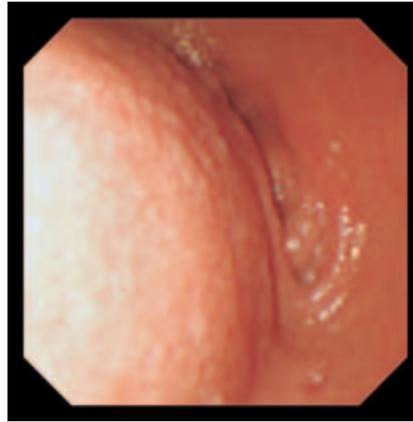


2.581

2.582

### Hepatic impression

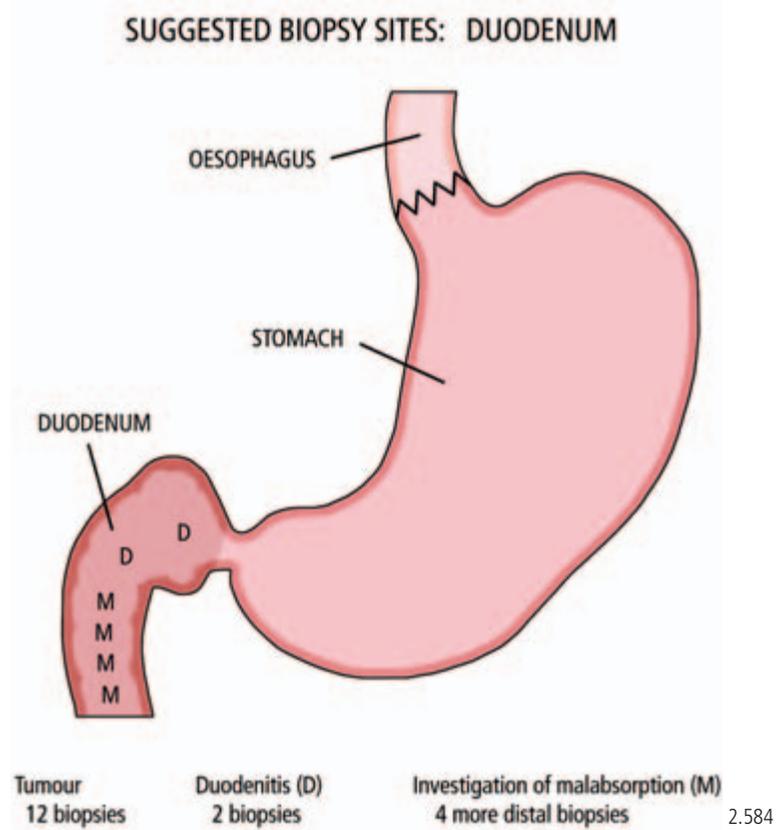
Any enlarged or abnormal structure lying against the duodenum may cause a visible bulge without a breach in the duodenal mucosa. The appearances shown in Fig. 2.583 were due to a nodular cirrhotic liver.



2.583

### Suggested biopsy sites

Figure 2.584 shows suggested biopsy sites for various types of abnormalities, and the number of samples that should be taken. For further details the reader is referred to the appropriate section of this chapter.



## Postoperative appearances

Oesophageal anastomosis

Nissen fundoplication

Band gastroplasty

Gastrotomy scar

Pyloroplasty

Gastroduodenal and gastrojejunal anastomoses: normal appearances

Anastomotic ulceration

Intestinal metaplasia

Stump carcinoma

Retained antrum following Polya (Billroth II) partial gastrectomy

Retained suture material, staples and swabs

Cholecyst-duodenostomy

Choledocho-duodenostomy

Angelchik prosthesis

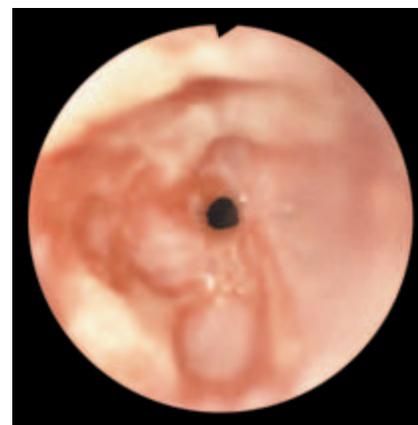
Surgery may alter the anatomy of the upper gastrointestinal tract in many ways. Scarring and adhesions can cause deformity and fixation. Consequently radiological appearances are often confusing and may simulate disease. For these reasons endoscopy is the preferred method of investigation, more particularly following pyloroplasty or partial gastrectomy. Nevertheless, important complementary information, especially of an anatomical type, can sometimes be obtained from a barium meal examination when the layout is delineated, for example if the nature of the previous surgery is not known. In general terms, when considering the symptomatic postoperative patient, endoscopy is better when investigating recurrent pain or bleeding, whilst for vomiting or bloating, radiology is superior.

### Oesophageal anastomosis

The sequelae of acid reflux may appear after an oesophago-gastric anastomosis. Figure 2.585 shows an oesophago-gastric anastomosis with minimal oesophagitis. In Fig. 2.586 there is moderate oesophagitis and a stricture.



2.585



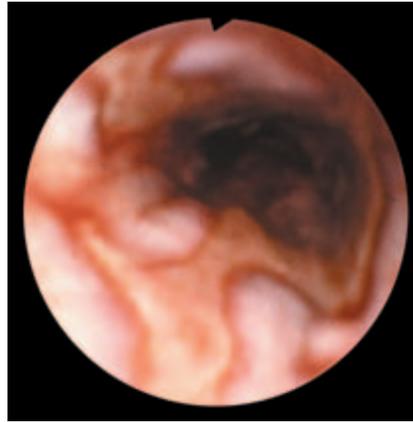
2.586

**Oesophageal anastomosis (cont.)**

Oesophagitis may also follow total gastrectomy (Fig. 2.587).

Figure 2.588 demonstrates oesophagitis, and a stricture with converging cicatricial folds. In Fig. 2.589 the typical appearances of jejunal mucosa are seen distal to the anastomotic site; besides showing a normal well-healed anastomosis, the orifice of a postoperative oesophago-pleural fistula is seen in the lower part of Fig. 2.589.

In Fig. 2.590 there is a retained suture (see also Figs 2.704 and 2.706), and in Fig. 2.591 a retained staple.



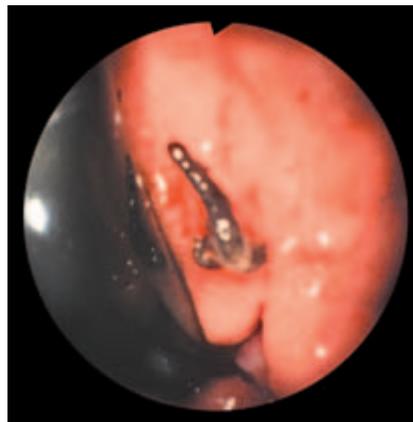
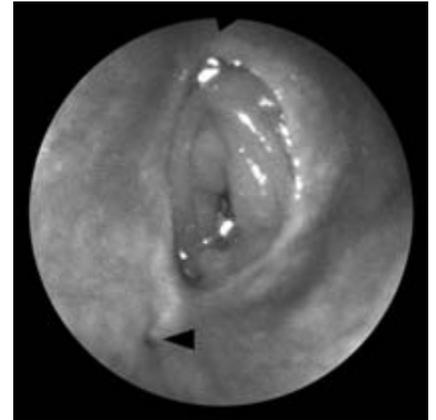
2.587



2.588



2.589



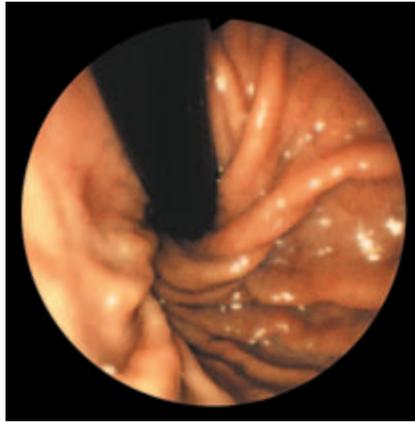
2.590



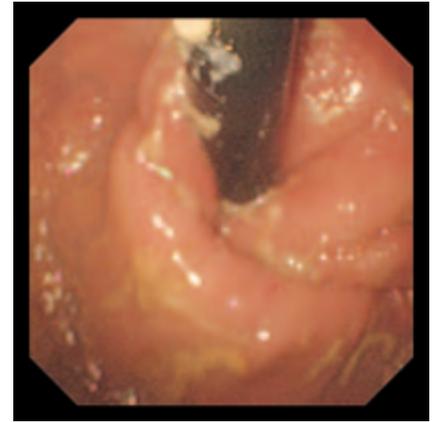
2.591

**Nissen fundoplication**

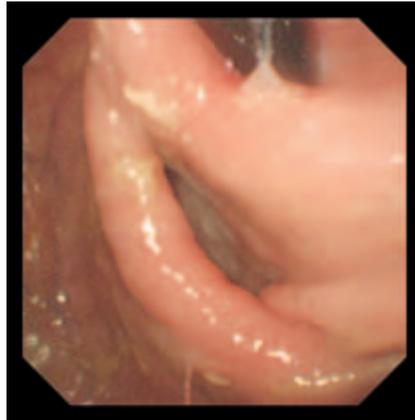
Typical appearances following successful fundoplication are shown in Figs 2.592 and 2.593. Sometimes a diverticulum is simulated (Fig. 2.594) by the ‘wrap’ or ‘inkwell’; other deformities and scarring may occasionally occur (Fig. 2.595).



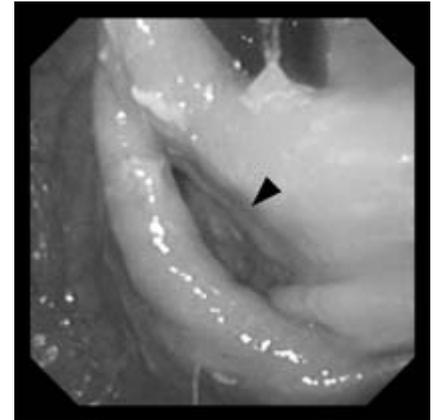
2.592



2.593



2.594



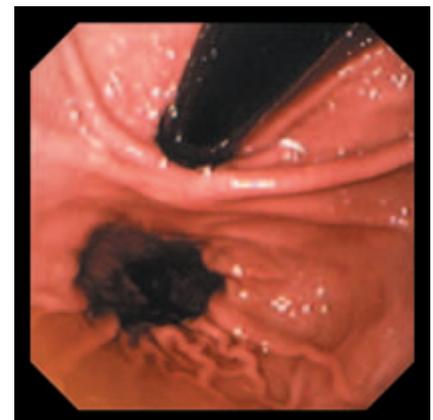
2.595

**Band gastroplasty**

The vertical band narrows the proximal stomach to a slender tube (Fig. 2.596). A small diameter endoscope passed through the ‘stoma’ between the proximal portion and the body of the stomach has viewed the gastroplasty by inversion (Fig. 2.597).



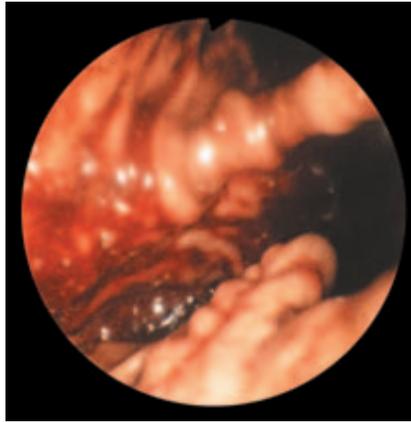
2.596



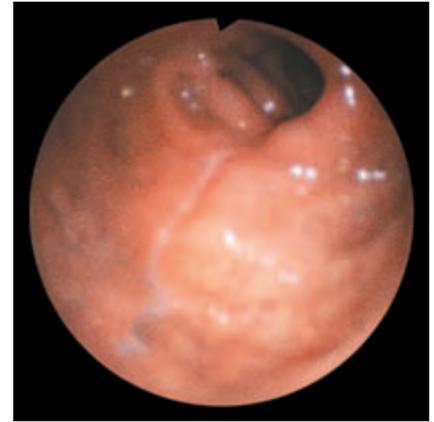
2.597

### Gastrotomy scar

Following gastrotomy the suture line can be clearly seen at an early postoperative stage when the edges are still raised (Fig. 2.598). Later the linear scar in an otherwise normal stomach (Fig. 2.599) is whitish-yellow and similar to, although longer than, that resulting from a healed gastric ulcer (see also Fig. 2.325).



2.598



2.599

### Pyloroplasty

The appearances of an uncomplicated pyloroplasty are shown in Fig. 2.600. In Fig. 2.601 a small recurrent duodenal ulcer is visible through the pyloroplasty, while in Fig. 2.602 a large ulcer has recurred just proximal to the papilla following an extensive pyloroplasty. Sometimes after pyloroplasty the anatomy is so altered that no vestige of the pyloric ring remains and the gastric antrum funnels directly into the duodenum.

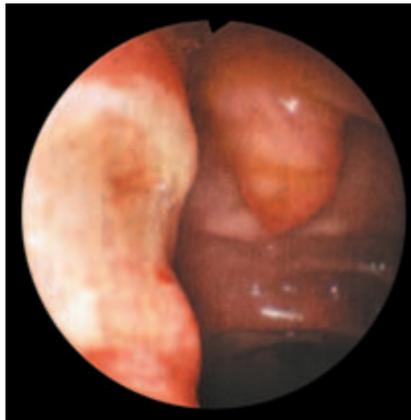
An unusual complication of no clinical significance is illustrated in Fig. 2.603 where a mucosal bridge was found at the mouth of an otherwise successful pyloroplasty. The photograph also shows an exploratory probe used during the endoscopic examination, more clearly to display the bridge. This is another example of 'double pylorus' (see also Fig. 2.248).



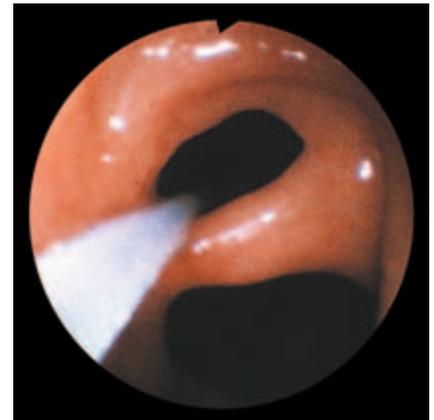
2.600



2.601



2.602



2.603

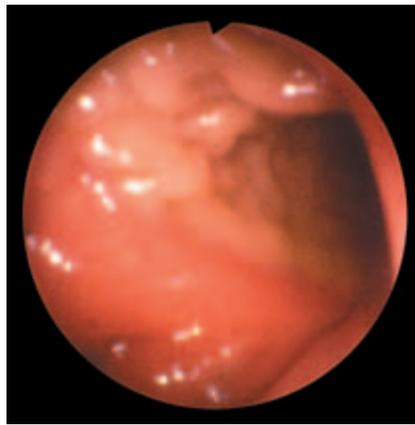
**Gastroduodenal and gastrojejunal anastomoses: normal appearances**

Following almost any gastric operation and especially partial gastric resection, the gastric mucosa near the operative site will appear redder than normal. This may merely represent hyperaemia though reactive gastritis may be present (Fig. 2.271); a firm diagnosis of gastritis should rest on histopathological appearances (see pp. 84–94). In contrast with gastric mucosa, small intestinal mucosa looks brownish.

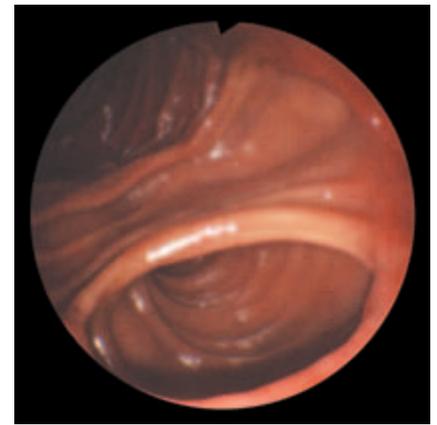
Following Billroth I resection the gastric lumen may give the impression of funnelling into the proximal duodenum (Fig. 2.604).

The orifices of the afferent and efferent loops following a Polya (Billroth II) resection are shown in Fig. 2.605. The effects on the stoma of afferent loop peristalsis appear in Figs 2.606 and 2.607, and a close-up view of the gastrojejunal mucosal junction appears in Fig. 2.608.

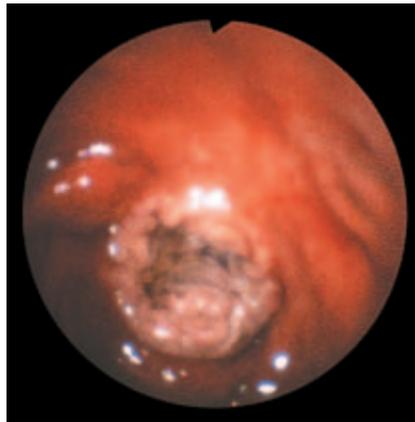
When it is difficult for the endoscope to enter one or the other loop it may help to pass a plastic catheter (Fig. 2.609) or a wire through the orifice as a guide along which to thread the endoscope.



2.604



2.605



2.606



2.607



2.608



2.609

**Anastomotic ulceration**

Anastomotic ulcers may occur anywhere on or near gastroduodenal or gastrojejunal anastomoses.

Figure 2.610 represents an early postoperative view. The suture line is irregular and slightly ulcerated.

In Fig. 2.611 there is a small anastomotic ulcer. That seen in Fig. 2.612 is of moderate size but has a regular outline; irregular (Fig. 2.613) and multiple (Fig. 2.614) anastomotic ulcers also occur.

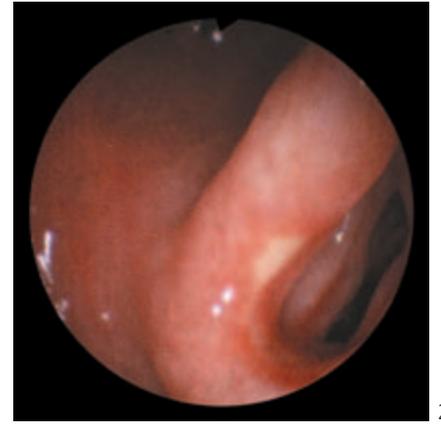
Large multiple ‘carinal’ ulcers are shown in Fig. 2.615. The areas of hyperaemic granulation tissue seen in Fig. 2.616 suggest that this ulcer is healing.

Rarely, an anastomotic ulcer can be better seen when approached indirectly. In Fig. 2.617 a large jejunal ulcer has been visualized after the endoscope has been passed via the duodenum into the afferent loop.

As with chronic duodenal ulcers, anastomotic ulcers may be associated with more distally lying duodenitis (Fig. 2.618).



2.610



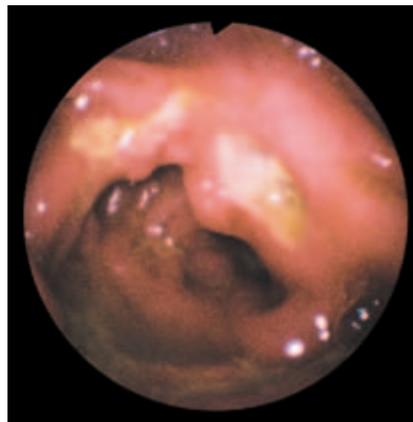
2.611



2.612



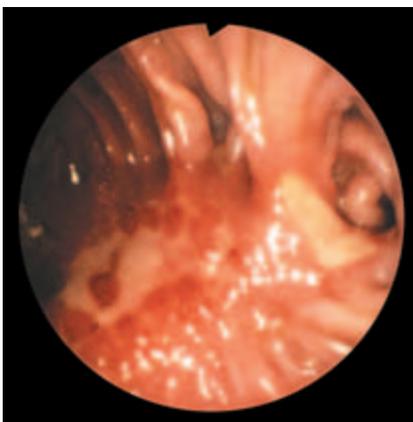
2.613



2.614



2.615



2.616



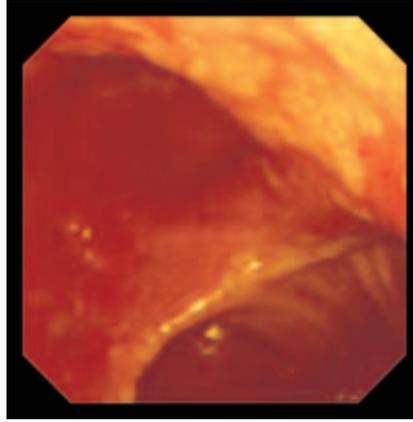
2.617



2.618

### Intestinal metaplasia

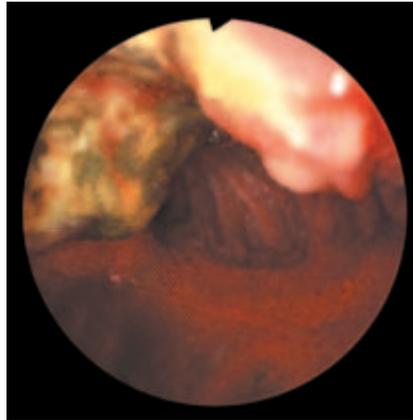
Intestinal metaplasia may develop in the gastric remnant proximal to the resection margin (Fig. 2.619) and typically appears as pale 'mucoid' patches (see also Fig. 2.268).



2.619

### Stump carcinoma

Figure 2.620 shows a carcinoma just proximal to a gastroenterostomy. This patient had undergone Polya (Billroth II) partial gastrectomy 40 years previously. There is an increased incidence of carcinoma in the operated stomach.

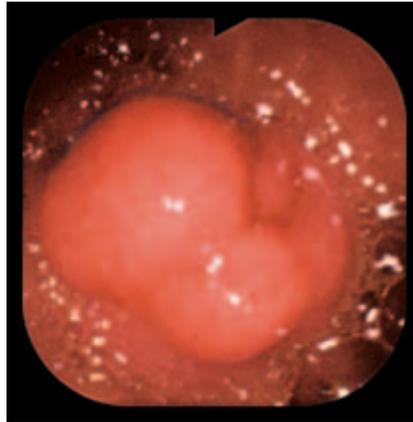


2.620

### Retained antrum following Polya (Billroth II) partial gastrectomy

At the time of Polya partial gastrectomy a small portion of gastric antrum may inadvertently be left behind within the supposed duodenal stump. Due to being in a persistently alkaline environment the mucosa of this portion of stomach may continue to secrete gastrin without inhibition. The resultant massive hypergastrinaemia can result in acid hypersecretion from the remaining main portion of the stomach leading to stomal ulceration.

Postoperative endoscopy via the afferent loop (Fig. 2.621) shows smooth reddish gastric mucosa at the apex of the blind duodenal stump. Target biopsy and plasma gastrin levels will confirm the diagnosis.



2.621

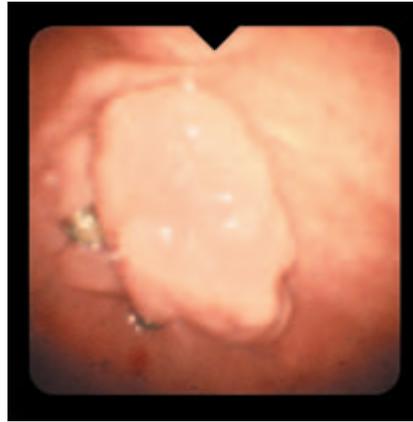
**Retained suture material, staples and swabs**

Retained silk suture material, now little used, often assumes an unusual colour due to bile staining and may also become covered with adherent detritus. Sutures may be found by chance when the operated upper gastrointestinal tract is inspected for whatever reason. Such material may be associated with local probably insignificant ulcer formation, and can be removed endoscopically (see also p. 196).

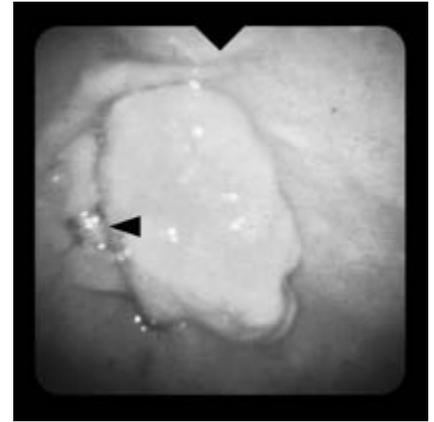
Figure 2.622 shows the internal appearances of the duodenal stump closed by a purse string suture following Polya partial gastrectomy. Note the invaginated portion of the duodenum and the persistent unabsorbed suture material.

In Fig. 2.623 a retained suture is illustrated, while Fig. 2.624 shows a staple associated with minor ulceration.

Very rarely a swab may inadvertently be left in the abdominal cavity at operation. In this patient a swab retained in the caecal area had ulcerated through a caecoduodenal fistula and was visible at endoscopy of the upper gastrointestinal tract (Fig. 2.625).



2.622



2.623



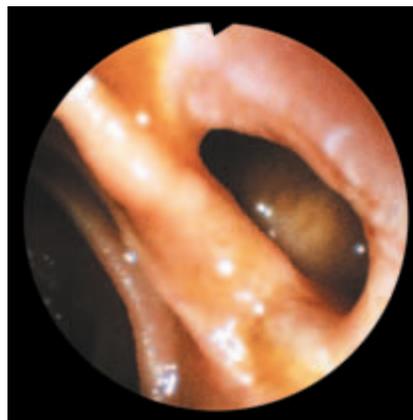
2.624



2.625

**Cholecyst-duodenostomy**

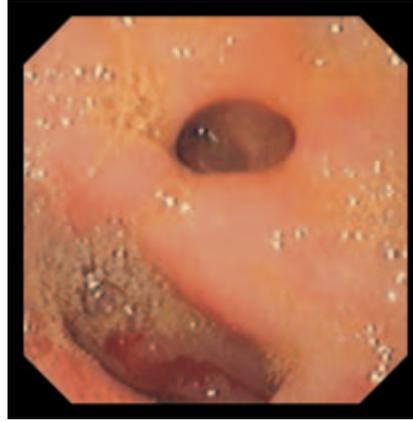
The anastomotic site (Fig. 2.626) resembles the opening of a congenital diverticulum.



2.626

### Cholecho-duodenostomy

This is shown in Fig. 2.627. Using a small diameter endoscope it was possible to examine the biliary tree (Fig. 2.628).



2.627



2.628

### Angelchik prosthesis

In this procedure a soft silicone ring is tied around the lower oesophagus at the level of the oesophago-gastric junction in an attempt to recreate the intra-abdominal oesophagus and so reduce gastro-oesophageal reflux. Because of the frequency and severity of complications this operation has lost favour, but occasional patients who underwent the procedure may come to endoscopy for a variety of reasons.

Figure 2.629 illustrates the appearances of the gastro-oesophageal junction from below; this patient had no complications and was investigated for other unrelated symptoms. Note the typical tyre-like appearances. There is no evidence of mucosal ulceration, displacement of the device or of perforation, some of the complications which have been described.



2.629

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## Bleeding

**Blood and blood clot with varying degrees of freshness**

**Active arterial bleeding**

**Blood issuing from the duodenum**

**Slow bleeding from a visible lesion**

**Visible vessel and 'red spot'**

**Fresh adherent blood clot**

**Dieulafoy lesion**

**Old adherent blood clot**

**Dark material in the base of the lesion**

**Active venous (variceal) bleeding**

**Fresh adherent variceal blood clot**

**Ulcerated gastric varix**

**Oesophageal submucosal haematoma**

Endoscopy is the method of choice in the investigation of upper gastrointestinal bleeding and in addition it plays a major role in treatment. Endoscopists must therefore be familiar with the indications for endoscopy, methods of patient preparation, the interpretation of various findings, how to deal with certain incidental problems such as removal of blood clot, and with the various therapeutic approaches available.

It is mandatory that the patient is fully resuscitated before commencing endoscopy and careful clinical observation is essential throughout the procedure to ensure safety.

Emergency endoscopy is more difficult than an elective procedure and should only be undertaken after the endoscopist is competent in the latter. In recent years there have been considerable advances in endoscopic therapy for patients with acute gastrointestinal haemorrhage (pp. 208–217). However, a full and detailed discussion of therapeutic techniques in the bleeding patient is beyond the scope of this atlas and for such details the reader is referred to the bibliography.

This section considers the appearance of bleeding, blood clot, altered blood and the causative lesions. There are illustrations of the visible vessel and other stigmata of recent haemorrhage. It is essential to distinguish these accurately as the risk of rebleeding is closely related to the presence and type of stigmata. With improvement in the results of therapeutic endoscopy, an accurate diagnosis assumes even greater relevance.

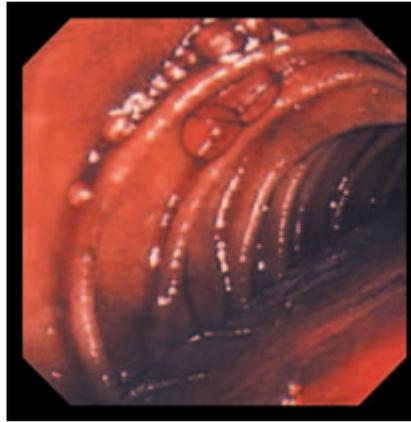
### Blood and blood clot with varying degrees of freshness

After an acute haemorrhage, blood may be slow to clear from the upper gastrointestinal tract. Clearing may be hastened by the use of metoclopramide or a similar agent. In the first instance, the endoscopist must be familiar with the various appearances of blood and clot in the organs to be surveyed.

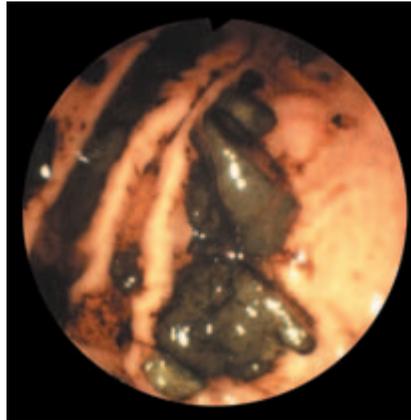
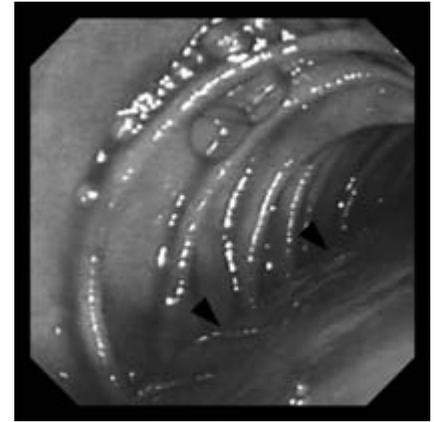
In Fig. 2.630 there is a very large and fresh blood clot in the duodenum. Old blood clot (Fig. 2.631) appears darker than fresh clot. It is common to find a large amount of clot lying in a pool of altered blood in the fundus. Such clots cannot be aspirated through the endoscope. With careful air distension it is usually possible to steer the tip of the endoscope past clot for inspection of the greater part of the stomach and duodenum. If vision is poor it may be possible to tip the patient so that retained blood is moved away from an area previously hidden. If this fails, clots can sometimes be washed out by passing a large bore stomach tube, the endoscope having been redrawn. Not uncommonly blood clot is vomited up during the examination making it easier for the endoscopist to proceed to an accurate diagnosis. Care must be taken lest bronchial aspiration should occur.

Figure 2.632 shows traces of fresh and slightly altered brown blood near the superior duodenal fold. It is mainly bright red in colour but it is surprising how quickly the colour changes to brown in an acid environment.

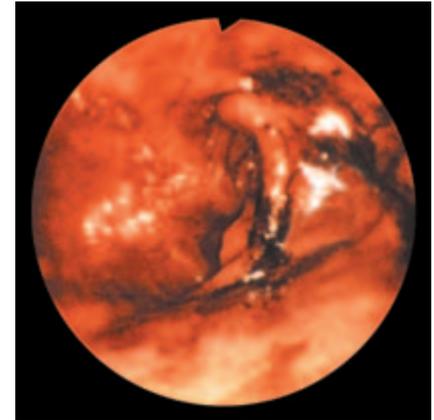
Later dark altered blood in the form of streaks (Fig. 2.633) and clots (Fig. 2.631) may be found in the oesophagus, stomach or duodenum. They can usually be displaced quite easily with a jet of water allowing the underlying mucosa to be inspected.



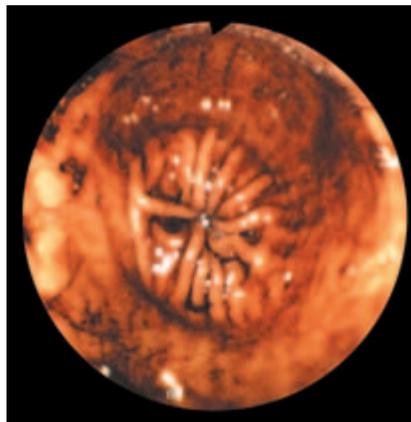
2.630



2.631



2.632

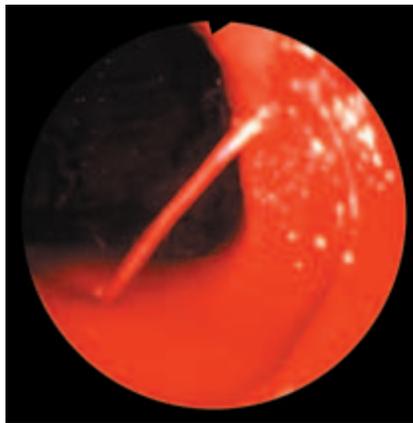


2.633

### Active arterial bleeding

Arterial bleeding is not commonly seen as 'immediate' urgent endoscopy is not usually regarded as necessary. Figure 2.634 shows active arterial bleeding from a lesser curve gastric ulcer.

The probable steps leading to arterial bleeding are described below (see p. 180).



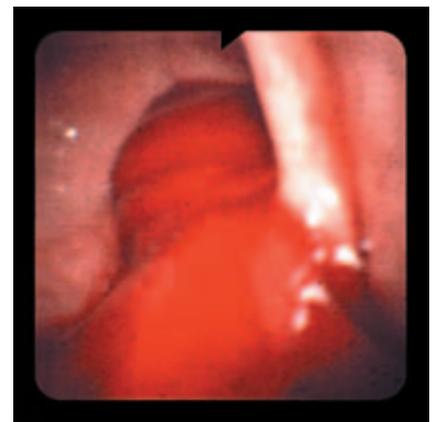
2.634

### Blood issuing from the duodenum

A flow of fresh blood may obscure its site of origin. In this case, blood is seen issuing from the pylorus (Fig. 2.635) stemming most likely from a duodenal lesion. Under such circumstances it is often impossible to aspirate blood quickly enough through the endoscope and only a presumptive rather than a certain diagnosis can be made. Fig. 2.636 shows a similar situation, with fresh blood and clot emerging through the pylorus.



2.635

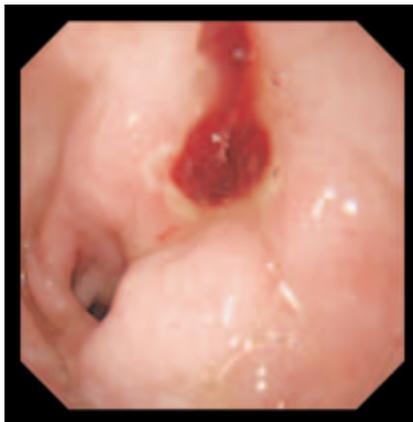


2.636

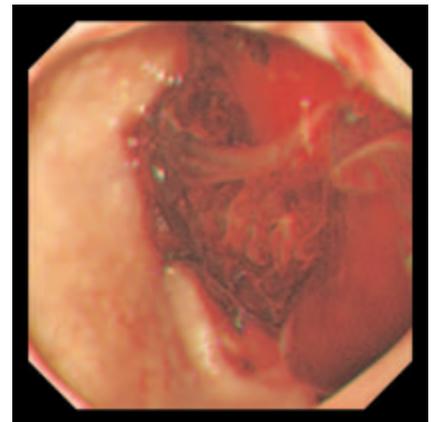
### Slow bleeding from a visible lesion

If there is oozing from a lesion, it can often be effectively washed clean by a jet of water from a flushing catheter passed through the operating channel of the endoscope. The lesion can then be well seen, accurately diagnosed and effectively treated. Sometimes the ooze can be seen to emerge from a lesion without washing.

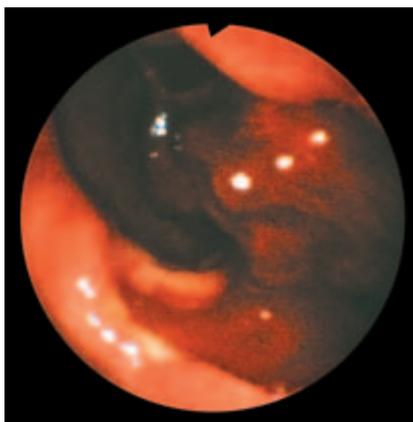
Figure 2.637 shows oozing from the base of a gastric ulcer. Figures 2.638 and 2.639 show fresh clot adhering to duodenal ulcers where oozing has barely ceased.



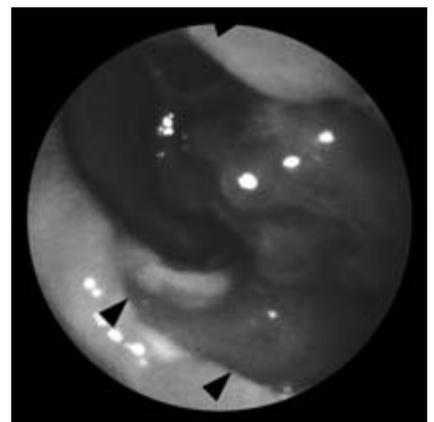
2.637



2.638



2.639

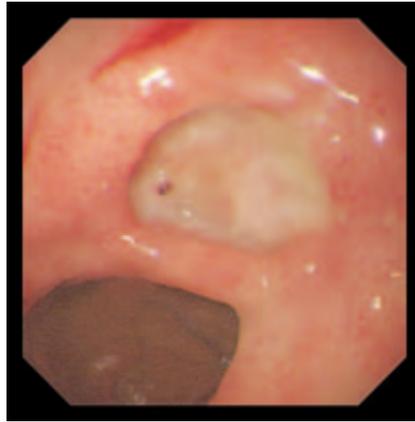


**Visible vessel and 'red spot'**

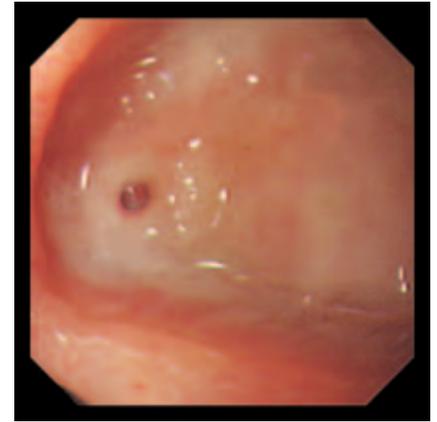
A visible vessel when identified, and this can present difficulties, should alert the endoscopist to the fact that the patient is very likely to rebleed.

The well circumscribed, round raised red spot in an ulcer base shown in Fig. 2.640 is a good example of a visible vessel; a close-up view appears in Fig. 2.641. Urgent endoscopic treatment would be indicated in the presence of such a finding. The diagnosis of 'visible vessel' is often difficult: for example, when there are several discolored areas in the base of an ulcer as shown in Fig. 2.642, only one of which is recognizable as a visible vessel.

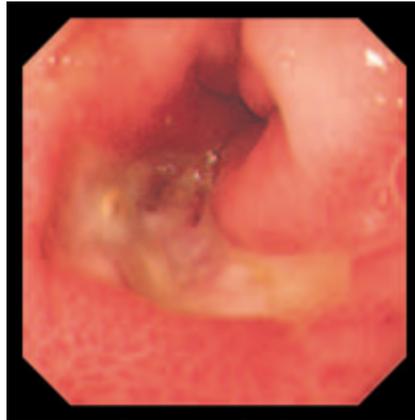
A visible vessel probably evolves in the following manner. An adjacent artery becomes involved in the inflammatory process in the base of the ulcer and appears near the surface. The arterial wall is weakened and, due to the relatively high intra-arterial pressure, begins to bulge. Such an aneurysmal deformity can sometimes be seen endoscopically. This being the weakest point in the involved artery, it is the one most likely to rupture. Figure 2.643 illustrates the histopathological appearances after rupture, at which stage the aneurysmal deformity is not always visible.



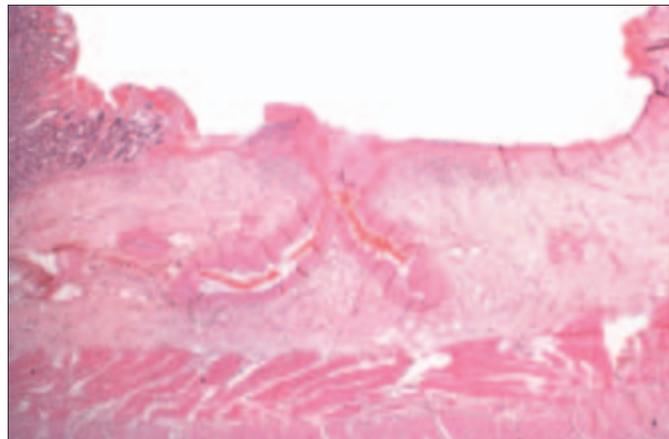
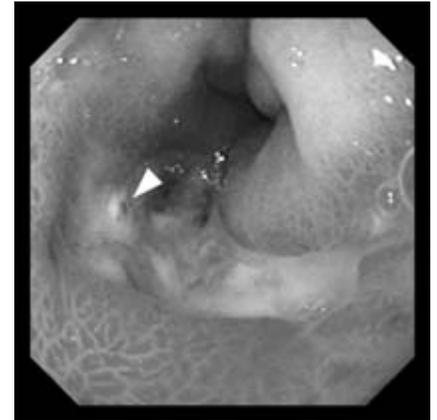
2.640



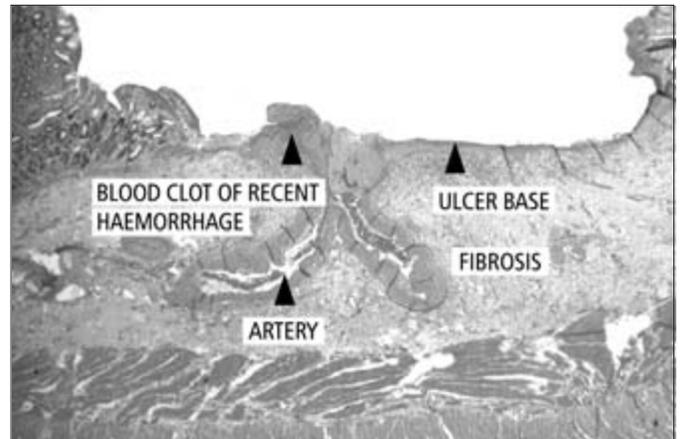
2.641



2.642

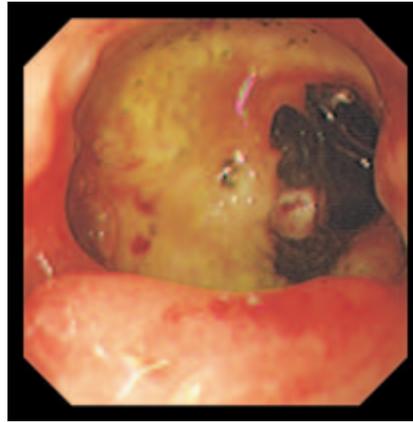


2.643

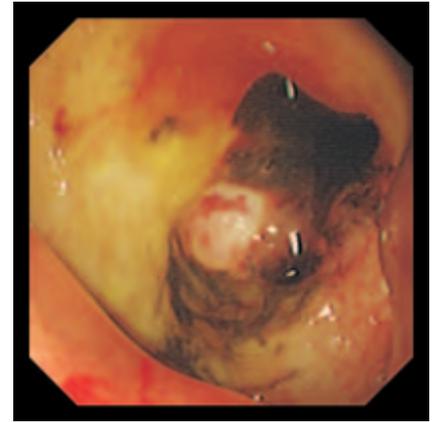


**Visible vessel and 'red spot' (cont.)**

Figure 2.644 shows a duodenal ulcer after haematemesis. There is aneurysmal bulging in the wall of the artery in its base seen better in striking close up (Fig. 2.645).



2.644



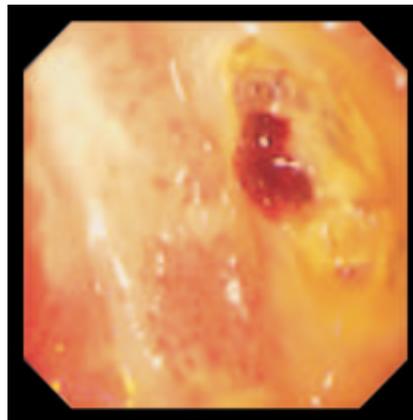
2.645

**Fresh adherent blood clot**

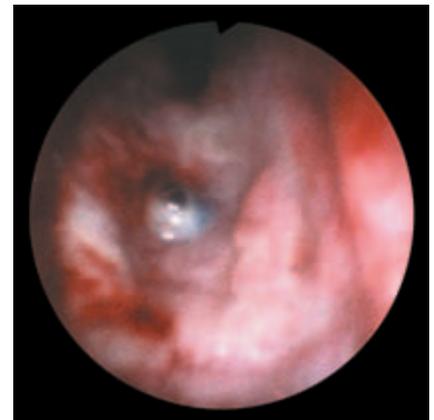
Endoscopic differentiation of a visible vessel from fresh blood clot may present difficulties.

Figure 2.646 shows a raised reticulated fibrinous clot on a yellow ulcer base. At the apex of the clot there is a red spot, probably indicating a very fresh clot on a prominent but not quite visible vessel.

Figure 2.647 shows a raised pale coloured lesion with a dark apex, while in Fig. 2.648 there is a small protuberant darker lesion. The spot in Fig. 2.649 resembles the lesion in Fig. 2.641 but is considerably darker. All three probably represent proud-standing vessels with fresh adhering clot.



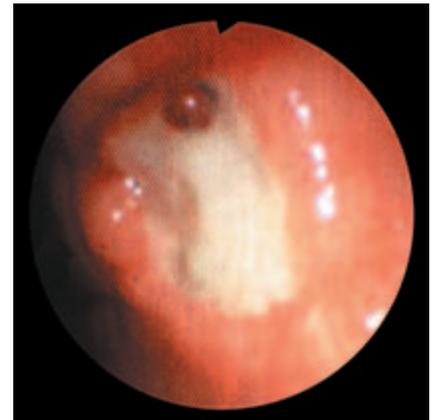
2.646



2.647



2.648

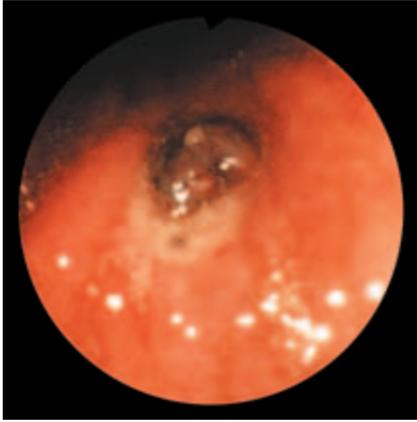


2.649

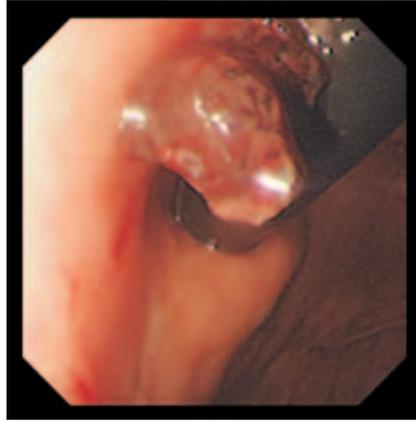
**Fresh adherent blood clot (cont.)**

Fresh clot is easily recognized in Figs 2.650 and 2.651. There is a strong suggestion of a 'red spot' surrounded by fresh clot in the base of the ulcer appearing in Fig. 2.652.

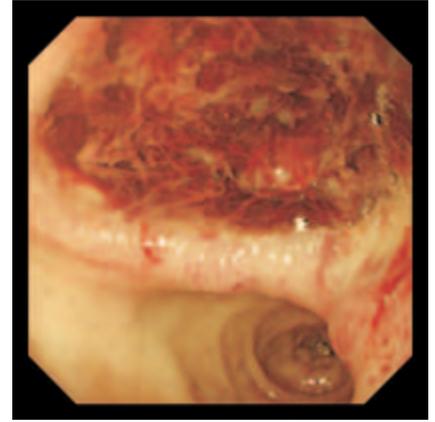
If endoscopic therapy is contemplated it may be necessary to attempt to wash off adherent clot to reveal the underlying lesion. This is discussed in greater detail on p. 179.



2.650



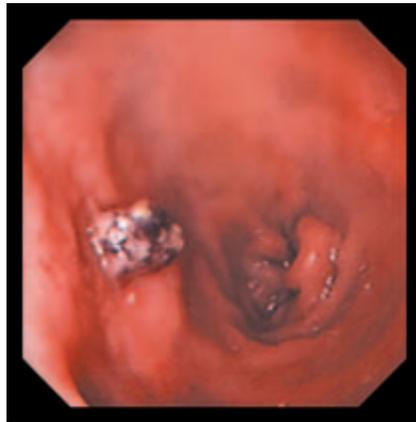
2.651



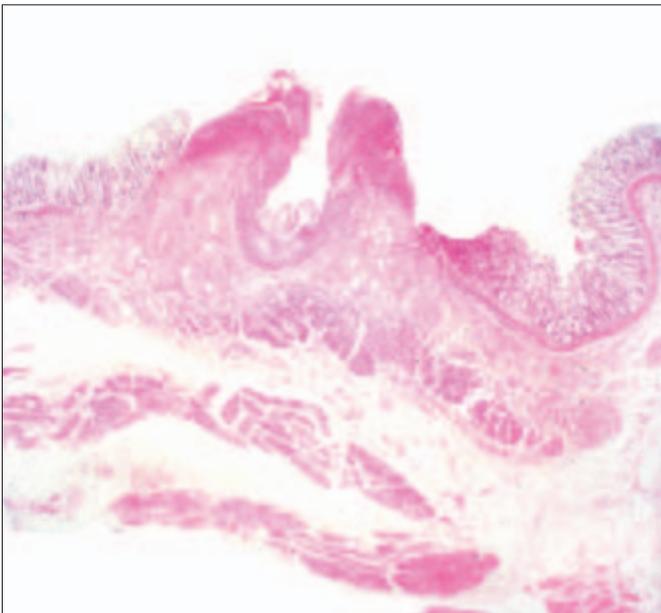
2.652

**Dieulafoy lesion**

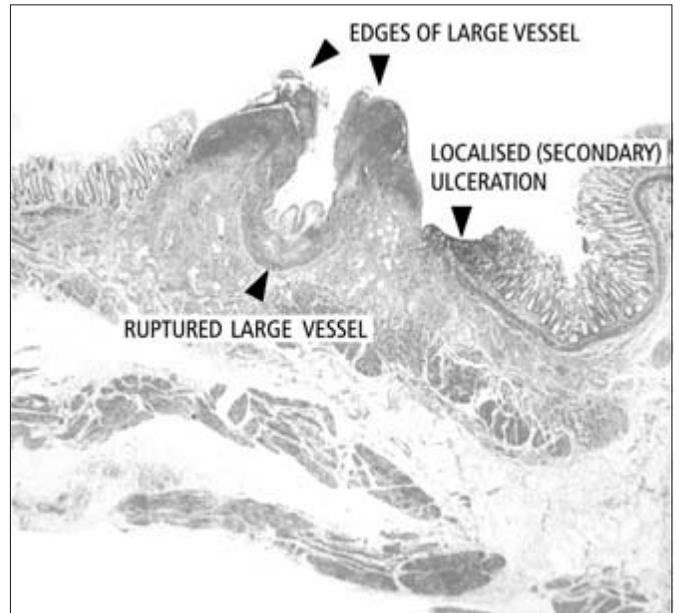
The Dieulafoy lesion is a blood vessel which has ruptured into the gastrointestinal tract, but without previous or associated peptic ulceration. It occurs most commonly in the gastric fundus. When such a lesion is not bleeding it is difficult to see endoscopically. In this case (Fig. 2.653) there is a moderately fresh adherent clot. Figure 2.654 shows the histopathological appearances.



2.653

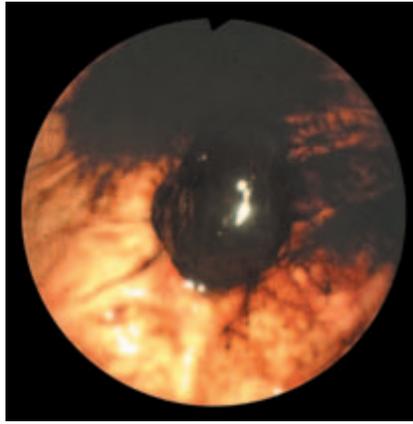


2.654



**Old adherent blood clot**

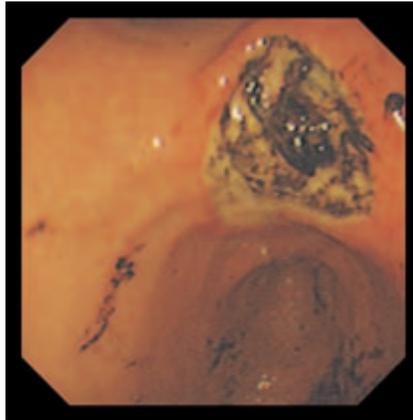
An older darker egg-shaped clot overlying a small gastric ulcer is seen in Fig. 2.655. In Fig. 2.656 there is a dark irregular clot on a gastric ulcer: the irregular shape of this blood clot and its colour suggest that it has been there for several hours. Smaller quantities of clot remain on another ulcer (Fig. 2.657); probably such appearances precede those shown in Fig. 2.658.



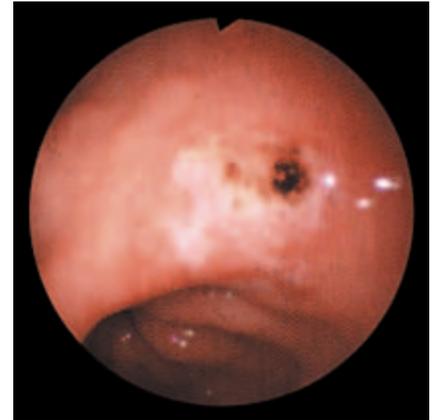
2.655



2.656



2.657



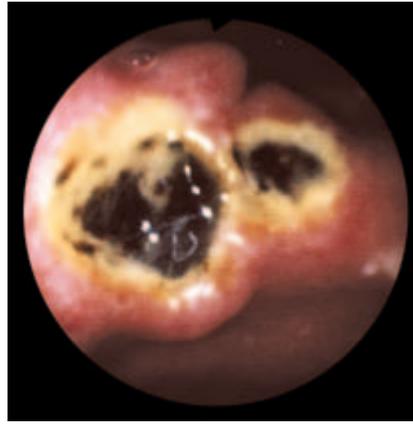
2.658

**Dark material in the base of the lesion**

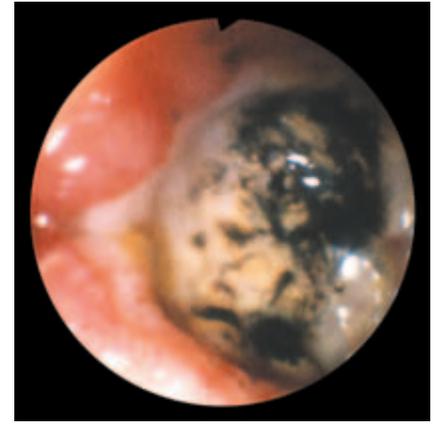
This represents altered blood in the slough covering the base of the lesion.

The altered blood shown in Fig. 2.658 is dark red rather than black and is therefore of recent origin. The black areas appearing in the ulcer base in Figs 2.659 and 2.660 represent older blood. When such appearances are present there is little chance of recurrent bleeding. Most endoscopists would therefore advise against active endoscopic therapy.

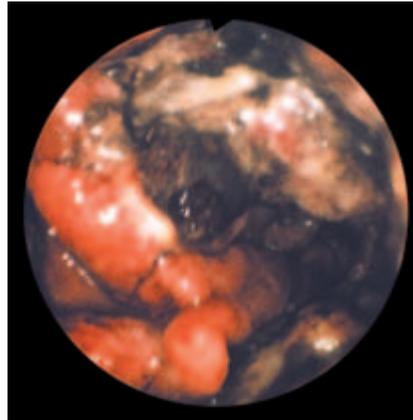
Figure 2.661 shows evidence of old blood in a malignant ulcer, while Fig. 2.662 and 2.663 show old blood in the base of an acute erosion and a Mallory–Weiss tear, respectively (see also Figs 2.445 and 2.446).



2.659



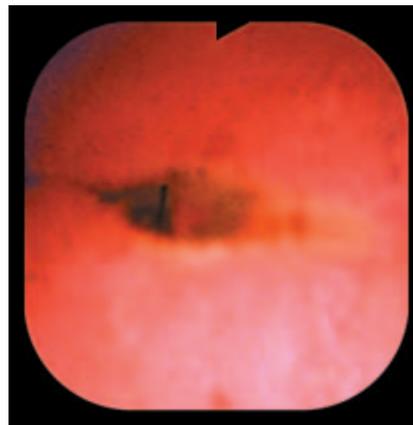
2.660



2.661



2.662

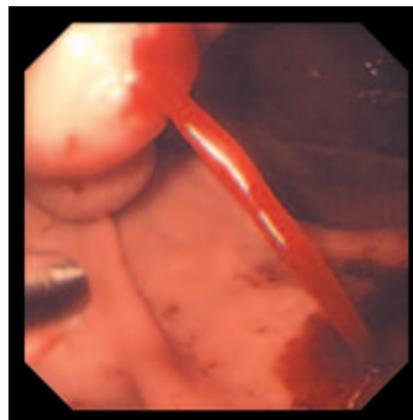


2.663

**Active venous (variceal) bleeding**

A venous source of bleeding will have a lesser head of pressure compared with an arterial source, though this may not be immediately obvious, as when the appearances of Figs 2.664 and 2.634 are compared.

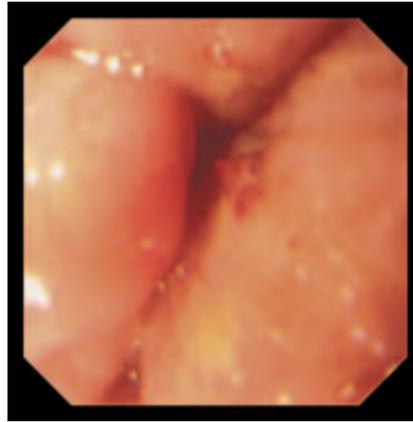
Bleeding from a gastric varix is shown in Fig. 2.664. Bleeding from oesophageal varices is similar in appearance but not usually as easily seen as blood drains away more slowly.



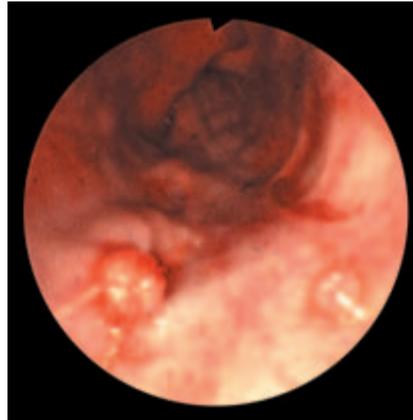
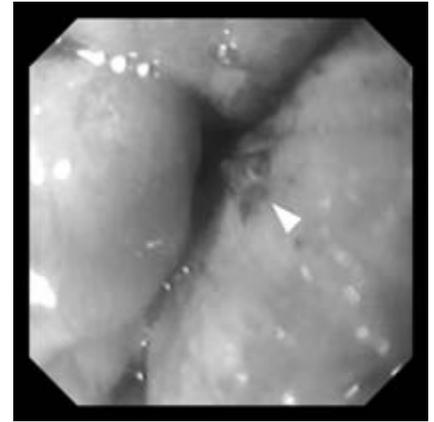
2.664

**Fresh adherent variceal blood clot**

This is shown in Figs 2.665 and 2.666.



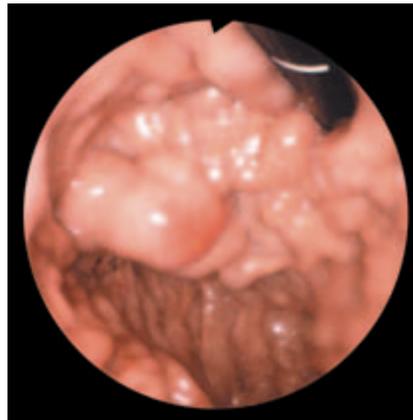
2.665



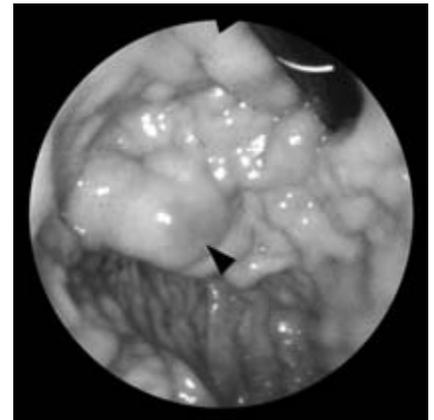
2.666

**Ulcerated gastric varix**

In the centre of this photograph (Fig. 2.667) there is a small ulcerated area, surrounded by a red flare. As the patient had recently bled, it is reasonable to assume that this represents the healing area from which the bleeding originated.



2.667

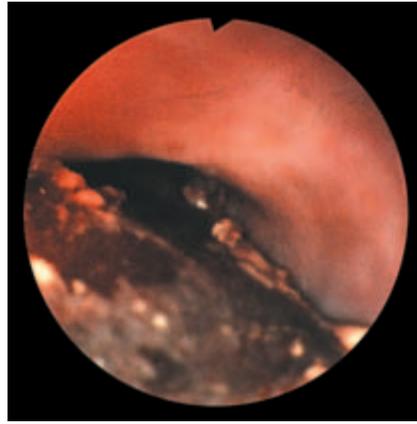


**Oesophageal submucosal haematoma**

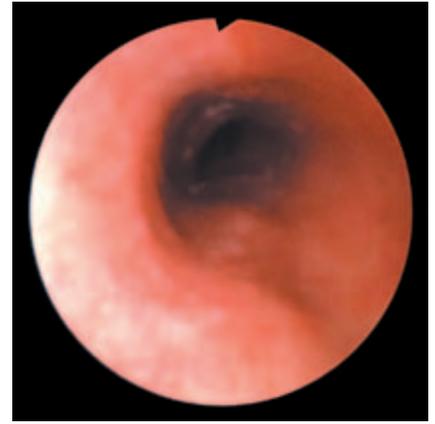
Figure 2.668 shows an haematoma extending almost throughout the length of the oesophagus reducing the lumen to a slit. This condition followed an episode of violent retching which was accompanied by a small haematemesis without subsequent dysphagia. Repeat endoscopy 2 weeks later showed complete resolution (Fig. 2.669) except for a small linear discoloration in the region of the oesophago-gastric junction. This sequence of events has been ascribed to submucosal bleeding from an incomplete tear not unlike a Mallory–Weiss lesion.

Figure 2.670 shows a similar though more circumscribed haematoma of which there were a number. Such lesions may ulcerate. During healing the rolled edges of the resolving lesions are easily identified (Fig. 2.671).

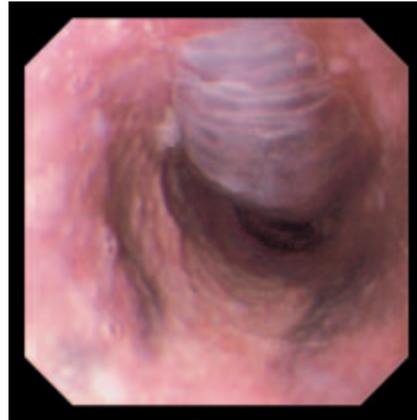
Oesophageal submucosal haematoma is also a recognized complication of cardiopulmonary resuscitation.



2.668



2.669



2.670



2.671

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## Therapeutic procedures

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Numerous therapeutic techniques have developed in the wake of diagnostic endoscopy. Today it is essential that the simpler and more frequently performed therapeutic procedures are available for patient care in all departments performing gastrointestinal endoscopy.

This section does not set out to describe methods in detail, nor does it purport to be comprehensive; for further information on endoscopic therapy the reader is referred to the bibliography. Nevertheless we believe it is appropriate to mention techniques in outline and to include some illustrations of the necessary accessories. Without this, the relevant endoscopic views would have had less meaning.

Before undertaking therapeutic procedures it is essential for the endoscopist to be fully competent in the appropriate diagnostic techniques.

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### Removal of foreign bodies

**Appliances commonly used for the removal of ingested foreign bodies**

**Impacted foreign bodies simply dislodged**

**Dissolution of foreign bodies**

**Foreign bodies retrieved using forceps**

**Foreign body removal using snares**

**An overtube in the removal of ingested foreign bodies**

**Magnet for removing ferrous metallic objects**

**A special device for small round objects**

**Broken endoscopic equipment**

**Psychiatrically disturbed patients, prisoners and unusual foreign bodies**

**'Body-packing'**

**Bezoars**

**Non-absorbable suture material**

Most swallowed foreign bodies (FBs) pass uneventfully through the gastrointestinal tract to be voided with the faeces. Objects suitable for removal are those causing or liable to cause obstruction of the lumen, those which are sharp and may become impacted or cause perforation or when passage from the stomach is unduly delayed. Certain FBs may contain chemicals that are dangerous to the gastrointestinal tract locally if released whilst others may have serious systemic toxic effects. Illegal drugs are sometimes concealed by swallowing in condoms or other containers. Careful consideration must be given in each case as to whether the object may pass spontaneously, may cause some complication and whether attempts at endoscopic removal may inflict damage upon the patient.

Ingested objects require considerable skill and patience for successful removal. A range of accessories is needed and ingenuity may be called upon to grasp and deliver some unusual FBs. Occasionally improvisation will be required. With the appropriate technique, virtually all ingested FBs can be safely and successfully removed endoscopically without recourse to surgery, unless full-thickness penetration of the gastrointestinal tract has already occurred by the time of presentation.

#### **Appliances commonly used for the removal of ingested foreign bodies**

Forceps for FB removal are purpose designed with recurved tips or jaws which grip (Fig. 2.672). Biopsy forceps are unsuitable as hard objects slip when the jaws are closed. As an emergency measure if special forceps are not immediately available,

a 'V' can be filed across the jaws of an old pair of biopsy forceps but such modified forceps are far from satisfactory.

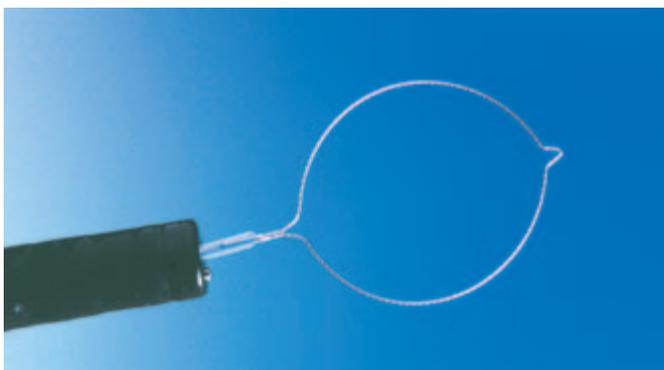
A snare loop (Fig. 2.673) designed for endoscopic polypectomy is useful for removing certain types of FBs. A tightly closed snare may bite into the FB so that it cannot easily be disengaged if for some reason this is necessary; it is therefore advisable to apply a snare gently until the endoscopist is certain that it is in good position and that the FB can be safely delivered.

Wire baskets of various sizes and configurations are useful for small ovoid or near-spherical objects (Fig. 2.674).

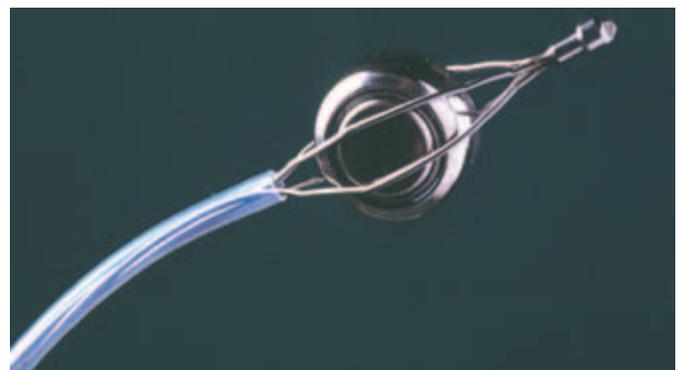
All endoscopy units must possess a wide range of retrieving devices such as those here described.



2.672



2.673



2.674

### Impacted foreign bodies simply dislodged

Large lumps of food may stick in the oesophagus above a stricture or due to hasty swallowing of unchewed material, especially meat. A bolus of jacket potato (Fig. 2.675) lodged above a stricture which was known not to be very tight. After disruption the bolus was guided into the stomach with the tip of the endoscope. The stricture was subsequently dilated.

A lump of unchewed meat prematurely swallowed lodged in the lower oesophagus (Fig. 2.676). Another piece of meat, impacted in an hiatal hernia (Fig. 2.677) had caused pain and complete obstruction. Attempts at withdrawal failed because the forceps cut out, and a snare could not be placed. Firm pressure with the tip of the endoscope enabled the bolus to pass into the body of the stomach (Fig. 2.678). Sometimes careful passage of a fine guide wire, perhaps under fluoroscopy, helps to find a lumen alongside the impacted bolus. The endoscope or a fine dilator may then be passed over the wire. Using this manoeuvre it may be possible safely to dislodge the stuck object.

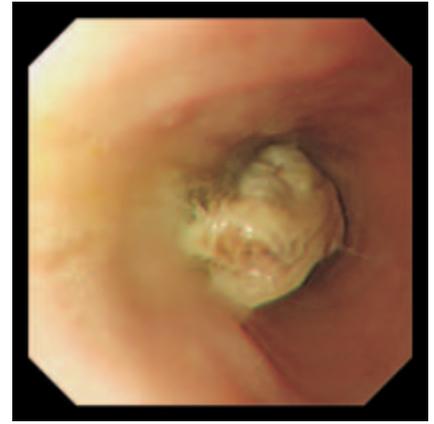
Great care should be taken before it is decided to push rather than to extract. A superficial tear or full thickness rupture may occur, especially if an hitherto unrecognized stricture is present.

### Dissolution of foreign bodies

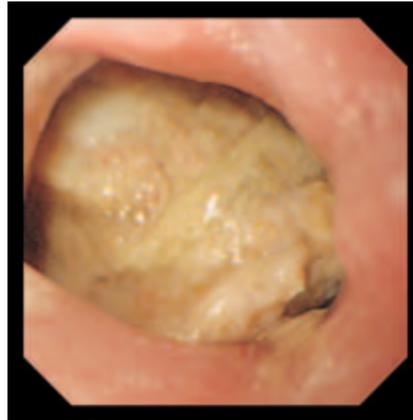
Attempts have been made to soften certain types of impacted or retained foreign bodies by direct endoscopic injection. For example, papaine has been used for meat impactions, and cellulase for phytobezoars (see p. 195). This approach may be ineffective and is not without risk.



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2.678

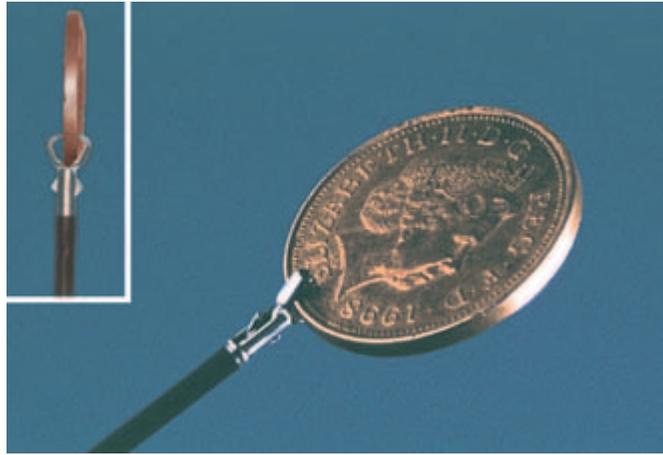
**Foreign bodies retrieved using forceps**

Figure 2.679 shows a coin grasped in special forceps. The rim possessed by most coins ensures firm engagement by the forceps.

A single coin in the distal antrum (Fig. 2.680) and a collection of coins lodged in the lower oesophagus (Fig. 2.681) above a mild stricture were easily removed with grasping forceps although it was necessary in the latter case to pass the endoscope repeatedly. The orange object seen to the left of Fig. 2.681 is a small piece of carrot.

Needles, pins and open safety pins may cause perforation. Removal may safely be undertaken so long as sharp ends are kept well away from the mucosa. Protection may be provided by trailing the point or by using an oversheath (Fig. 2.682) as described below (see p. 192). Careful subsequent observation for signs of perforation is important. In this case (Fig. 2.683) a dental file embedded into the antral wall was withdrawn into the lumen and the point trailed during removal.

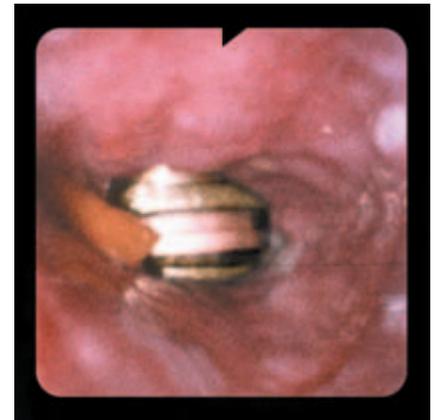
Keys on a ring grasped by forceps (Fig. 2.684) present no problem. There is little risk of damage from the keys trailing behind. The ring dilates the oesophago-gastric junction during withdrawal.



2.679



2.680



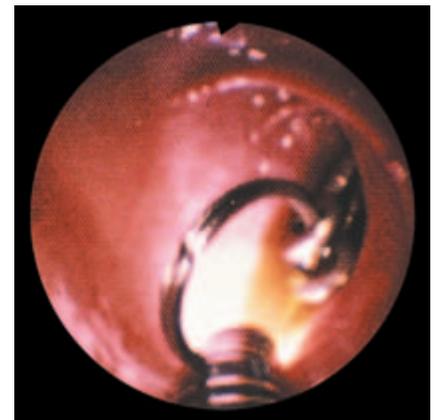
2.681



2.682



2.683



2.684

### Foreign body removal using snares

Food lodged in the oesophagus may become soft and difficult to remove. If it is reasonably firm, such as a piece of meat, it may be possible to pass a snare over it and remove it *in toto* (Figs 2.685 and 2.686). Minor trauma and ulceration of the oesophageal wall (Fig. 2.687) may result from the object itself or from the removal process. Pressure necrosis is a rare but serious complication of food bolus impaction. A partial denture, with one false tooth visible, impacted in the mid-oesophagus (Fig. 2.688): it is simple to grip dentures with a snare loop but subsequent removal may be dangerous. First the object must be freed by angulation of the tip of the endoscope pressing the mucosa just above the FB, lifting the mucosa away. This allows inspection for possible damage and facilitates removal (Fig. 2.689). Especial care and patience are required when withdrawing such objects through the pharyngeal region: this should be done if possible under direct vision. Meat and fish bones are treated in a similar manner.

A clinical thermometer lying in the stomach (Fig. 2.690) was grasped near the end by a snare (Fig. 2.691) and successfully removed. Gentle snare tightening is essential to avoid breakage of a glass thermometer and gripping near one end allows easy passage through narrow regions.

It may be safer when extracting this type of FB to consider the use of a protective overshath (Fig. 2.682).



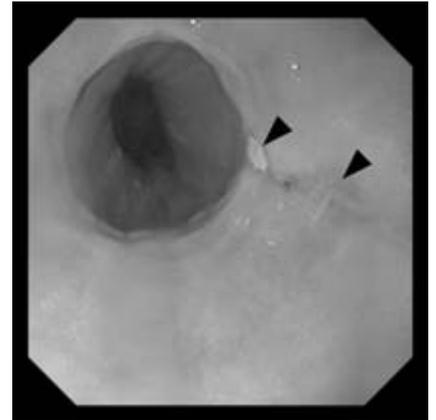
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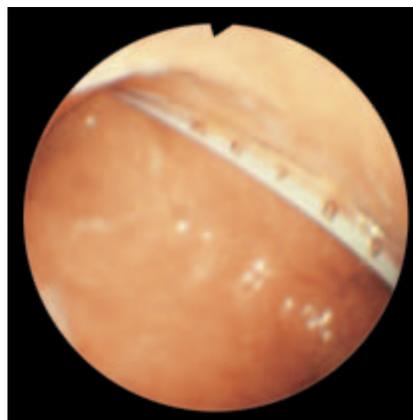
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2.688



2.689



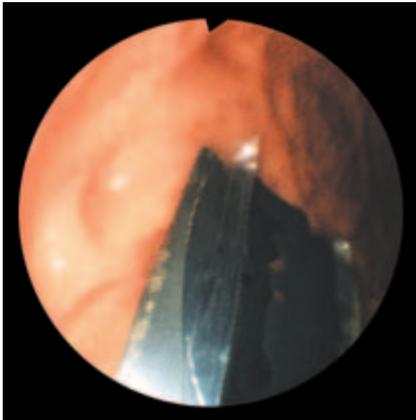
2.690



2.691

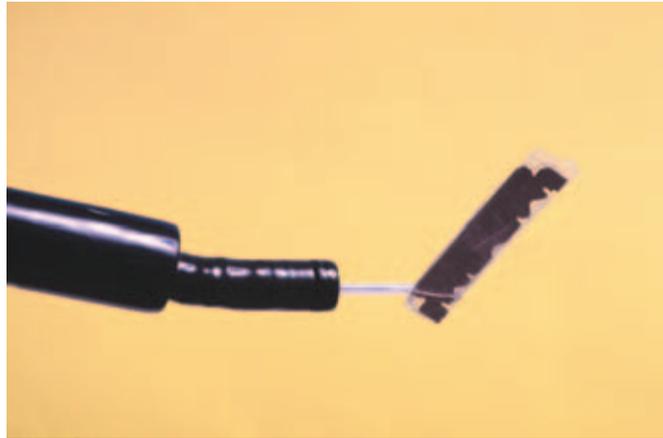
### An overtube in the removal of ingested foreign bodies

Certain types of FBs such as razor blades (Fig. 2.692) are removed more safely by initially withdrawing them into an overtube which has been passed with the endoscope into the oesophagus or stomach as appropriate. It will be noted that in this instance the patient covered the cutting edge of the razor blade with adhesive tape for swallowing.



2.692

Figures 2.693–2.695 illustrate how half a razor blade is removed using this technique. The sheath may be obtained commercially or prepared easily and cheaply from a length of soft plastic tubing about 50 cm long and 2 cm in diameter. It is back-loaded on to the endoscope before insertion and must be well lubricated both inside and out. When the endoscope has reached its destination it is straightened and the overtube is carefully advanced so as to provide a chamber into which the FB can be drawn, if necessary after crushing or breaking with the snare. After this, the firmly held FB, sheath and endoscope are removed together. As those who ingest such dangerous objects may react adversely to endoscopy with an increased risk of injury during the procedure, it may be advisable to perform this type of extraction under full anaesthesia.



2.693



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2.695

**An overtube in the removal of ingested foreign bodies (cont.)**

An overtube long enough to be passed into the stomach as described above must not be confused with a shorter overtube designed to go no further than the upper oesophagus as an aid to easy reintroduction of endoscopes or other appliances (Fig. 2.696).



2.696

**Magnet for removing ferrous metallic objects**

Some small metallic objects such as hearing aid and watch batteries (Fig. 2.697) may easily be withdrawn using a magnetic device passed alongside the endoscope (Fig. 2.698). This device is not very powerful and unless the FB is small and slides easily through the cardia there is a risk of it becoming detached and falling back into the stomach.



2.697



2.698

**A special device for small round objects**

Standard accessories sometimes fail or are not available, and improvisation may be required. A 4-year-old child had a stainless steel ball bearing in his stomach for 6 weeks. Snares and wire baskets were unsuccessful and therefore a 'trawling net' was made from a latex condom attached to the endoscope (Fig. 2.699). The ball was rolled into the appliance by tipping the patient, and was successfully removed. Alternative trawling nets would be the tip of a nylon stocking or the net commercially available for polyp retrieval (Fig. 3.442).



2.699

**Broken endoscopic equipment**

Endoscopic equipment occasionally breaks during use and the retained portion may need to be removed. Figure 2.700 shows a diathermy snare which became detached whilst looped over a polyp in the gastric antrum during polypectomy. A second snare was passed to complete polyp removal. The first snare was removed with the polyp.

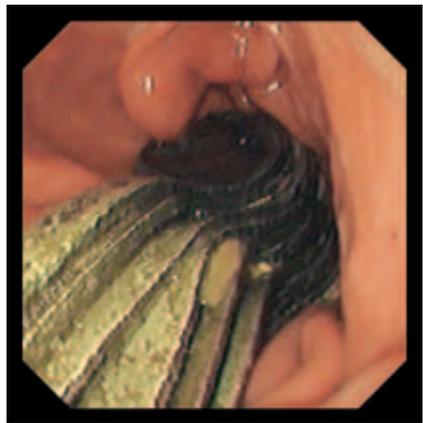


2.700

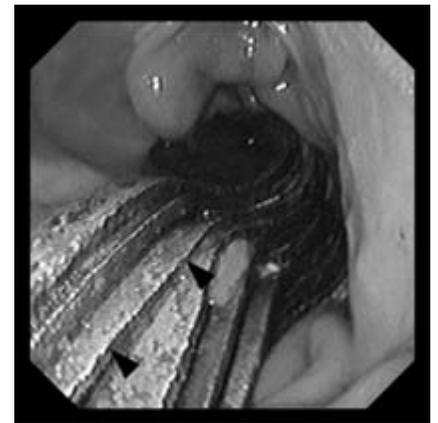
**Psychiatrically disturbed patients, prisoners and unusual foreign bodies**

Psychiatrically disturbed patients may present unusual problems in the nature of objects swallowed, an increased likelihood of repeating the performance and by failure of cooperation. Non-cooperation may demand the use of general anaesthesia both to avoid damage to the patient and to obviate the risk of the endoscope being bitten. Prisoners pose similar problems, ingestion of a FB being a deliberate attempt temporarily to escape detention.

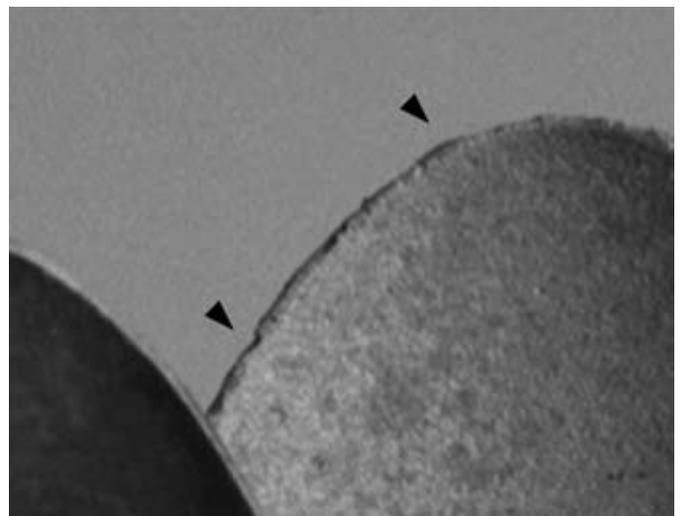
Figure 2.701 shows several spoons lying in such a patient's stomach. Following acid corrosion some objects of this type may become extraordinarily sharp along the edges which can become serrated (Fig. 2.702). Extreme caution is required for removal. Open surgery may be preferable.



2.701



2.702



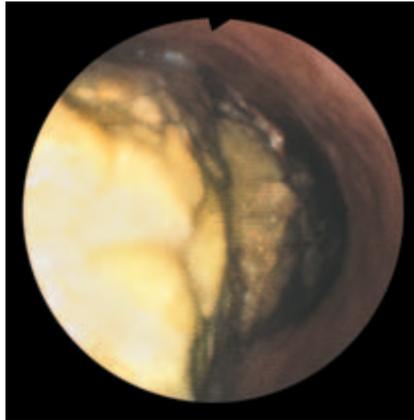
### **'Body-packing'**

'Body-packing', the ingestion of condoms or balloons filled with illicit substances to avoid detection by customs officials, is occasionally practised. Diagnostic endoscopy may be indicated (Fig. 2.467) but the risk of rupture of the balloon with release of potentially fatal doses of drugs makes endoscopic removal hazardous. If spontaneous passage does not occur and laxatives have proved ineffective, surgical removal is the only safe option.

### **Bezoars**

Large quantities of certain vegetable fibres (notably persimmon) or hair may accumulate in the gastrointestinal tract to form a bezoar. These FBs can cause abdominal pain, bloating or intermittent obstruction. Whilst mentally disturbed females constitute the majority of patients with hair balls (trichobezoars, and the Rapunzel syndrome) patients who have undergone gastric surgery, especially vagotomy, are prone to develop phytobezoars, as are patients with diabetic gastropathy.

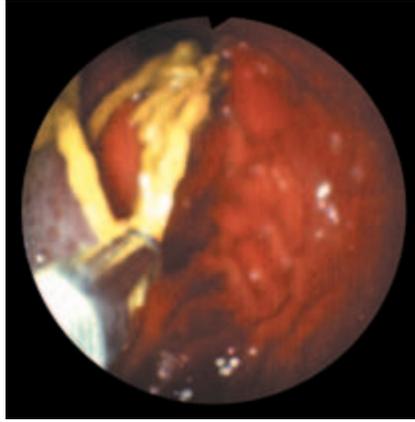
The patient whose appearances are illustrated in Fig. 2.703 had been known to have a phytobezoar for 5 years before the photograph was taken; 10 years previously he had undergone Billroth I partial gastrectomy for peptic ulceration. There was no anastomotic narrowing. The bezoar was resistant to enzymic dissolution but was easily removed piecemeal with a snare and suction using the overtube technique. The cause of bezoars in such patients is due to reduced motility in the operated stomach.



2.703

**Nonabsorbable suture material**

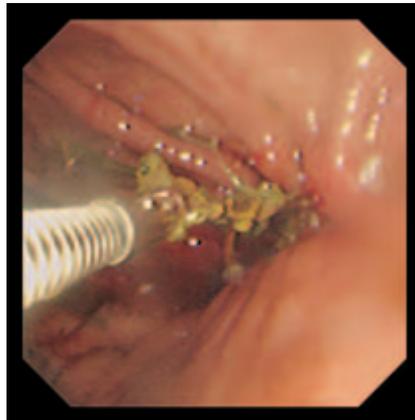
If this has been used for a gastro-intestinal anastomosis it may occasionally be seen protruding through the mucosa into the intestinal lumen. It acts as a foreign body and sometimes an ulcer crater surrounds the suture material. Whether the foreign material causes the ulcer or merely dictates the site at which re-ulceration occurs after surgery is unclear, but it seems reasonable to attempt removal when such associated ulceration is seen. Firm traction by means of biopsy (Fig. 2.704) or grasping forceps is usually successful. If this fails specially designed endoscopic scissors (Fig. 2.705) may release knots and aid removal (Fig. 2.706). Today, absorbable material is usually employed making this finding is less common (see also Figs 2.590, 2.622 and 2.623).



2.704



2.705



2.706

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## Dilatation of benign and malignant oesophageal strictures

### Equipment

#### Dilatation: endoscopic and radiographic appearances

Endoscopically guided dilatation of benign oesophageal strictures is a simple procedure with a low risk of complications: totally 'blind' dilatation is no longer acceptable. Several types of dilator are available, the majority based upon a similar principle. The tip of the endoscope is positioned just above the stricture and a guide wire is passed via the operating channel of the endoscope through the stricture, under direct visual control, the flexible tip of the guide wire being positioned in the stomach. When the wire is satisfactorily placed, the endoscope is removed whilst the position of the tip of the wire is maintained in the patient's stomach. The wire can now act as a guide over which bougies of increasing diameter or a balloon catheter can be passed, so dilating the stricture without the risk of creating a false channel. There is debate regarding the diameter to which benign strictures should be dilated, and whether full dilatation should be attempted at the first session or in stages.

When a stricture is smooth and straight even if tight, the flexible finger is easily guided through, although it passes out of view. If passage is simple it is safe then to proceed directly to dilatation. However, if there is resistance or a feeling of hold-up it is a wise precaution to use fluoroscopy to aid correct positioning of the wire. Less severe strictures can often be passed with a narrow diameter endoscope, when it is preferable to position the tip of the guide wire in the gastric antrum under direct vision.

After dilatation, endoscopy is usually repeated to check the appearance of the stricture and to complete examination of the stomach and duodenum if this was not previously possible.

Another popular technique employs a through-the-scope (TTS) balloon which as its name implies is passed through the endoscope channel. It has a soft flexible silicone rubber tip and an integral guide wire. This balloon permits dilatation under direct vision ahead of the endoscope which may then be advanced through the area directly after dilatation.

The same dilatation techniques are employed for malignant strictures but complications, especially perforation, are much more likely if a malignant stricture is dilated beyond 10–12 mm. Palliative endoscopic intubation for inoperable malignancy may be performed immediately after dilatation.

Some endoscopists recommend routinely taking a plain chest radiograph after any procedure involving oesophageal dilatation to avoid overlooking a silent perforation (see also p. 206).

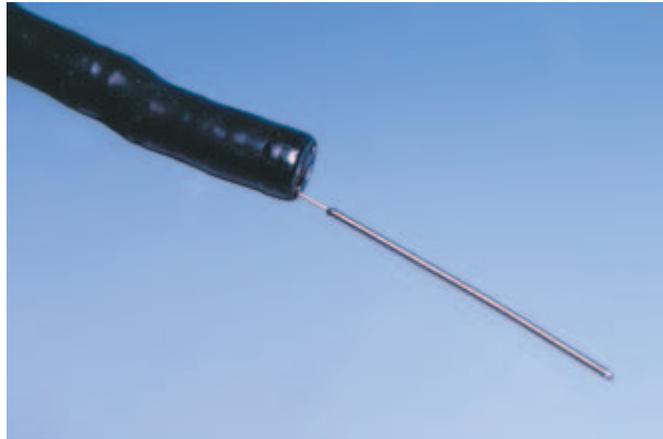
Opinions differ as to whether cytological smears and biopsy material should be obtained before or after dilatation. Biopsy immediately before dilatation might cause an area of least resistance through which a tear could develop during dilatation but there is little evidence to support this fear. Biopsies and cytological samples taken after dilatation often demonstrate confusing crush artefacts, but improved access after dilatation may have countervailing advantages.

### Equipment

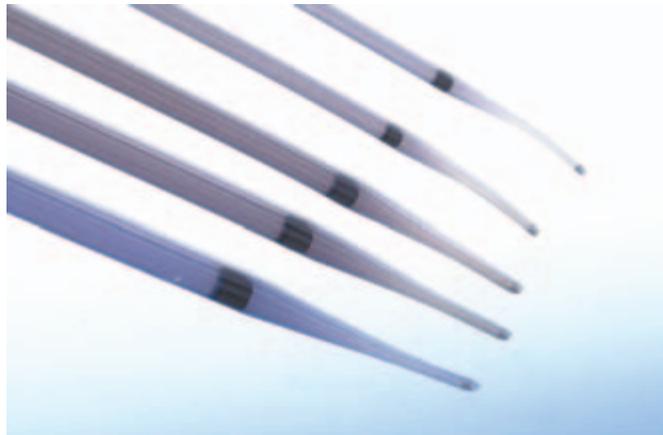
Figure 2.707 shows the flexible finger on the end of a stainless steel guide wire protruding from the tip of an endoscope. For safety and success guide wires must be in good condition without kinks in the wire or angulation of the flexible tip. Damaged equipment can cause complications: an angulated finger may negate the safety of a floppy tip, and a kinked wire may cause snagging of the bougie so that it is difficult to judge whether resistance on introducing the bougie is transmitted from the wire or the stricture. Furthermore, kinked wires are difficult to hold in position during both introduction and especially the withdrawal of bougies. Damage to wires results from careless or forcible use and from maltreatment. Damaged wires cannot be fully straightened and must be discarded.

Several types of dilator are available and there is little evidence for clinical superiority of any type; choice often depends upon individual experience. Figure 2.708 shows a series of Savary–Gilliard dilators from 7 to 18 mm in diameter. They are constructed from a silicone material and are rendered partially radio-opaque. There is a metal sleeve within the distal end to act as a firm stop when it abuts against the shoulder of the terminal flexible finger of the guide wire, so preventing advancement beyond the end of the wire. Smoothly tapered bougies such as these may not give a very positive ‘feel’ when passing through the stricture.

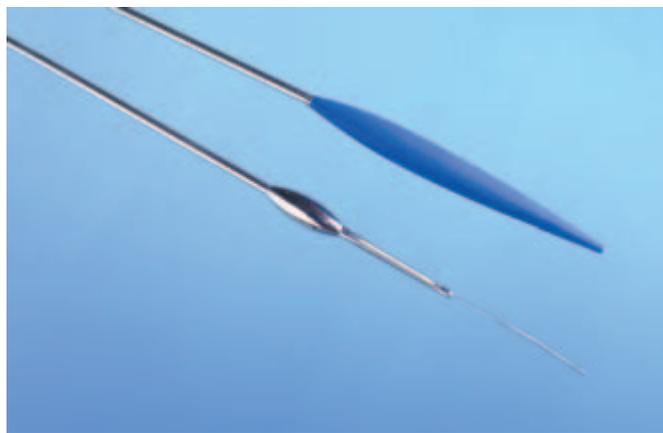
Figure 2.709 illustrates an assembled Eder–Puestow metal olive dilator on a guide wire and a Key-Med Advanced Dilator (KAD). For many years Eder–Puestow olives were the commonly used dilators, mainly as with increasing size it was possible to have a distinct ‘feel’ of the degree of resistance while dilating. Knowing the diameter of a given olive, the endoscopist could gauge the appropriate amount of pressure to be exerted safely. However, due to its rather cumbersome nature the Eder–Puestow dilator has been superseded for most purposes. The KAD shares some properties with olive and soft dilators as it has a tapered olive shape to give ‘feel’ yet is made of firm flexible plastic material which is easier to use.



2.707



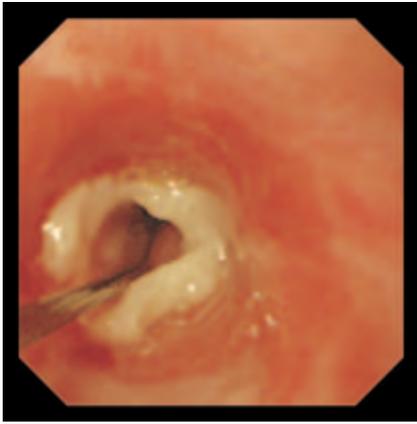
2.708



2.709

**Dilatation: endoscopic and radiographic appearances**

Figure 2.710 shows a guide wire passed through a tight stricture of approximately 5 mm diameter.



2.710

The position of a guide wire may be checked by fluoroscopy (Fig. 2.711). The endoscope is situated above the stricture and the wire has been passed well down into the stomach in a smooth curve. Fluoroscopy is only required when difficulty is expected or encountered. It is essential to pass several centimetres of wire, in addition to the flexible tip, beyond the stricture to allow enough run off for the dilator to pass fully through.

Figure 2.712 illustrates an Eder-Puestow assembly within a benign oesophageal stricture. (Note: this view is not normally seen as the actual dilatation is performed 'blind'; this picture was specially taken by passing a slim endoscope alongside the dilator.)



2.711



2.712

**Dilatation: endoscopic and radiographic appearances (cont.)**

Figure 2.713 shows a radiographic check to ensure that the bougie, in this case a Savary–Gilliard dilator, has successfully passed the stricture and that the guide wire remains correctly placed. Radiology during bougienage is particularly useful when passage of the wire through the stricture was difficult but also when the lumen is limited beyond the stricture or if there is a suspicion of distal coiling of the guide wire.

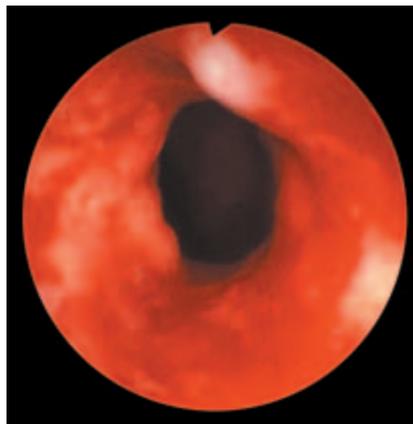
Endoscopic appearances immediately after dilatation are illustrated in Fig. 2.714. Some bleeding is usual but seldom of consequence. The area can easily be cleaned of blood by washing with water through a plastic tube passed via the endoscope or directly through the washing channel. After use of the larger dilators it is almost invariably possible to pass a standard-diameter endoscope through the stricture to complete the examination.

Figure 2.715 illustrates a small area of mucosal dehiscence as a result of dilatation of a benign stricture. There was no clinical evidence of perforation. This degree of mucosal damage is unusual, and might be regarded as due to too vigorous dilatation. Note: the guide wire is still in place to aid passage of the endoscope for completion of the examination beyond the stricture. Overenthusiastic bougienage can result in perforation.

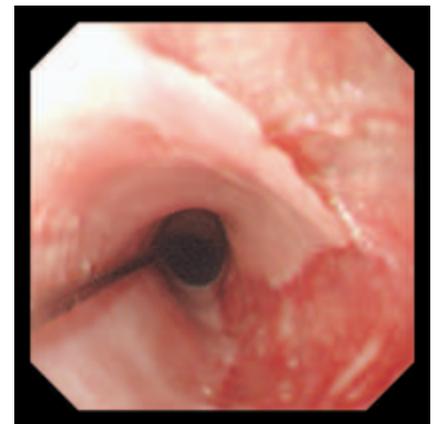
Balloon dilatation is preferred by many endoscopists as there might in theory be a safety advantage to stretching radially rather than forcing a bougie longitudinally. There is no convincing evidence, however, of the safety advantages of either standard method. Figure 2.716 shows balloons for use through the scope (TTS) or placed over a guide wire as with bougies. Unless fluoroscopy is employed there is always doubt as to the diameter of dilatation achieved with balloons as the waist (caused by the stricture) may persist despite firm inflation pressure.



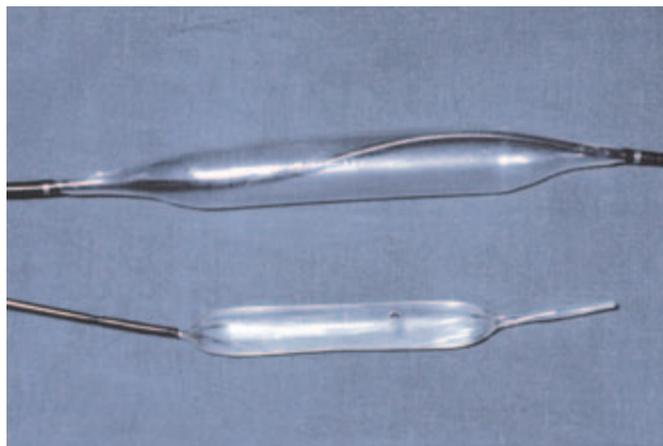
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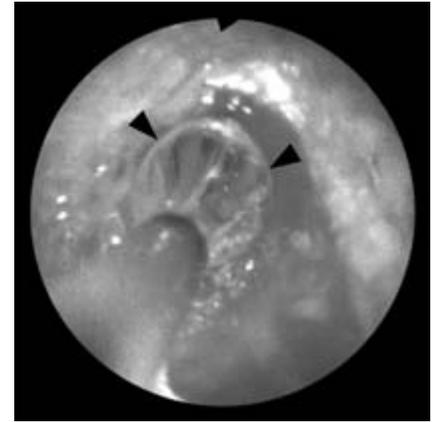
### Dilatation: endoscopic and radiographic appearances (*cont.*)

An obvious advantage of the TTS balloon is that dilatation is performed under direct vision (Fig. 2.717) and the endoscope may often be advanced immediately without the need for re-intubation, as is required with the other methods.

Balloons are costly and have a limited life span whilst bougies may be used indefinitely with obvious cost advantages. The clinical results are probably identical.



2.717



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## Palliation of inoperable oesophageal carcinoma

### Tumour reduction

#### Intubation (insertion of endoprosthesis)

Progressive dysphagia due to carcinoma of the oesophagus or neighbouring organs is a most distressing symptom. All patients should be carefully evaluated to decide upon the most appropriate method of treatment. Whenever possible surgical resection must be performed as offering the best hope of cure, but so often the disease has progressed beyond operability, or the patient's general condition precludes surgery. In such cases radiotherapy, chemotherapy or endoscopic palliation are indicated, either individually or in combination.

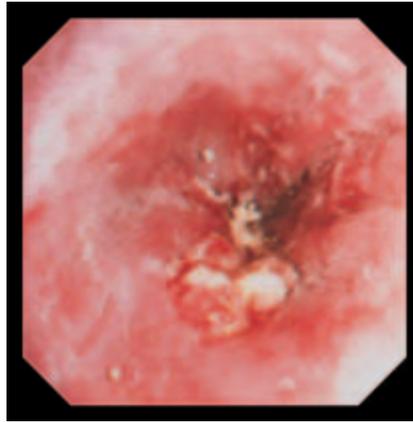
Endoscopic methods include tumour reduction by laser, argon beam coagulation or ethanol injection, and insertion of an endoprosthesis. Debulking is most successful for exophytic tumours projecting into the lumen but is of limited value in other cases. Patients with diffuse narrowing of the oesophagus or extrinsic compression as for example from a carcinoma of the bronchus, are usually best treated by intubation of the stricture with a prosthesis. Expanding metal prostheses need much less preliminary dilatation before placement than do semirigid silicone rubber or latex prostheses and are therefore easier and safer to insert. On the other hand the cost of a nonmetallic prosthesis is at present one tenth of the cost of an expanding metal prosthesis. Clinical and economic factors are in direct competition.

Tumour reducing treatments need to be repeated every few weeks but when successful the quality of swallowing may be superior to that achieved by intubation. By contrast, intubation is usually a once only treatment unless the tube migrates, food impacts or there is tumour overgrowth. The management plan must be decided individually depending upon the patient's acceptance of repeat endoscopy, the local availability of expertise and perhaps financial considerations.

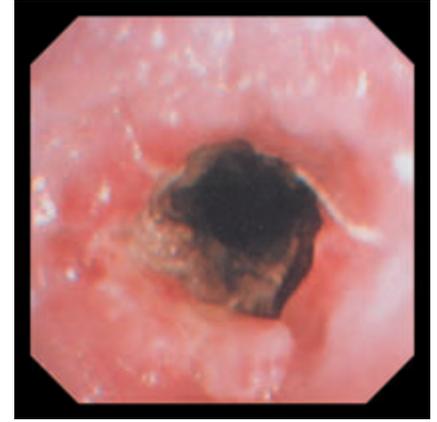
## Tumour reduction

### *Ethanol injection*

Injection of absolute (dehydrated) ethanol in 1 mL aliquots into the tumour tissue may effectively reduce the size of an exophytic carcinoma. The total volume needed to produce a satisfactory result varies with the size of the lesion but may be as much as 30 mL. The effect is not immediately visible and experience is essential to gauge the volume required. Figure 2.718 shows the obstructed lumen before treatment, and Fig. 2.719 shows the result subsequently achieved.



2.718



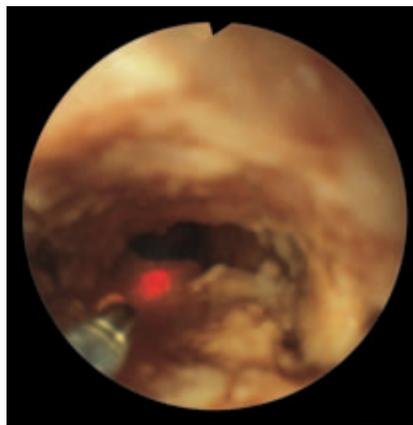
2.719

### *Laser therapy*

Endoscopic laser treatment is performed using a quartz fibre passed via the operating channel of the instrument. The laser source provides a coaxial jet of carbon dioxide around the fibre to prevent combustion of tissue. The gas jet may be useful additionally to blow away blood or debris from the site under treatment.

Exophytic tumours may be reduced in size by Nd-YAG laser treatment. The emission from this laser is invisible; an aiming beam, seen as a red spot in Fig. 2.720, is therefore required to direct the treatment. As the lesion is either desiccated or vaporized by the laser beam the effect can be seen immediately (Fig. 2.721) but its full extent will only become apparent after 24–48 h when the superficial area has sloughed. The beam penetrates to about 5 mm; the extent of destruction is therefore greater than that seen directly.

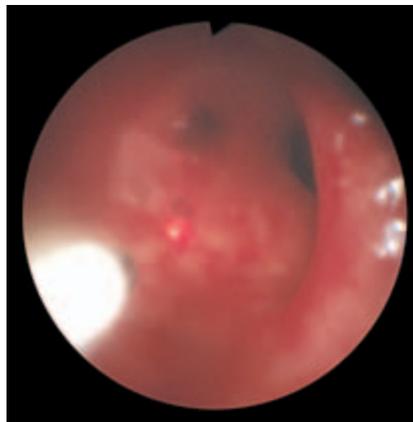
Figure 2.722 shows a large oesophageal carcinoma before and (Fig. 2.723) after treatment with the Nd-YAG laser. The lumen of the involved part of the oesophagus has been considerably enlarged.



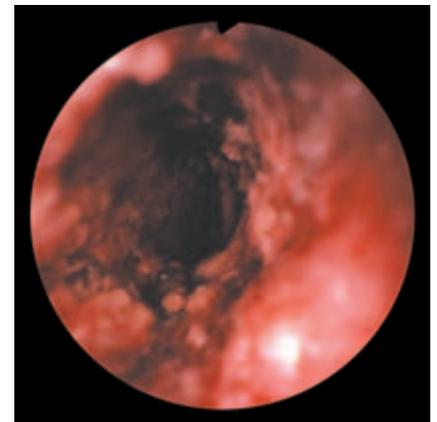
2.720



2.721



2.722

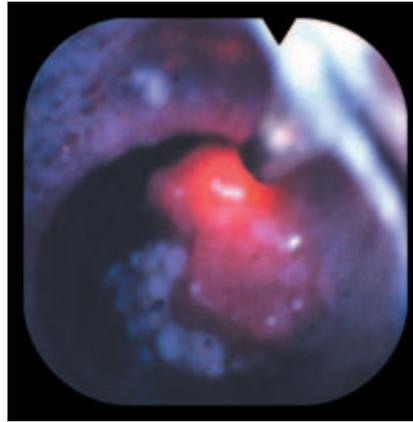


2.723

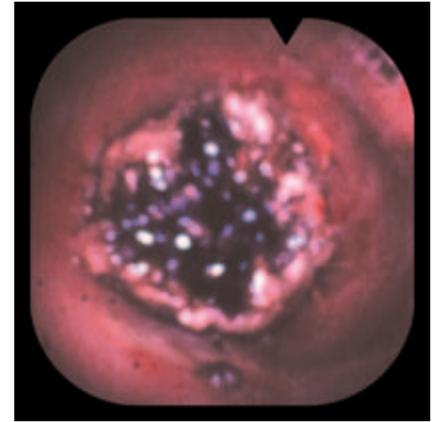
### Tumour reduction (cont.)

#### Laser therapy (cont.)

A smaller oesophageal carcinoma was successfully treated as shown in Figs 2.724 and 2.725.



2.724



2.725

#### Argon plasma coagulation (APC)

Argon plasma coagulation (argon beamer treatment) applies heat to tissue using a jet of argon gas as a noncontact electrode for diathermy. The depth of tissue penetration is only about 1–3 mm and targeting is less precise than with a laser. On the other hand APC is much less expensive in terms both of capital outlay and disposables. A distinct advantage compared with laser is that the gas jet can pass laterally as well as *en face* so that positioning may often be easier. It may be used for surface coagulation both for tumour reduction and haemostasis.

### Intubation (insertion of endoprosthesis)

#### Equipment

Plain and plastic-coated expanding metal stents are shown in Fig. 2.726. They are available in different lengths and diameters. It must be remembered that, with some metallic mesh stents, during expansion in width considerable shortening in length may occur so that it is essential that manufacturers' measurements of length are observed when selecting an appropriate stent. During introduction the compressed spring metal stent is contained within the delivery system from which it is released when correctly positioned. The stent takes on the shape of the stricture which it steadily dilates during the following hours or days. Uncoated stents embed themselves into the oesophageal wall, thus maintaining their position. Plastic coating (in the form of a sheath) is



2.726

provided on some stents to prevent ingrowth of tumour through the wire mesh. One of the consequences is that retention is reduced giving an increased risk of migration. To overcome this some coated stents are provided with hooks or barbs to fix

them in the stricture. In general, once an expanding metal stent has been placed it is not removable so that correct initial positioning is crucial. Plastic coated expanding metal stents are also used to occlude fistulae and perforations.

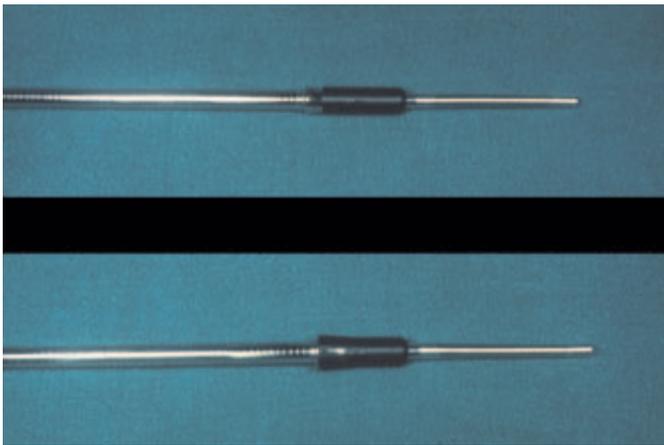
**Intubation (insertion of endoprosthesis)  
(cont.)**

*Equipment (cont.)*

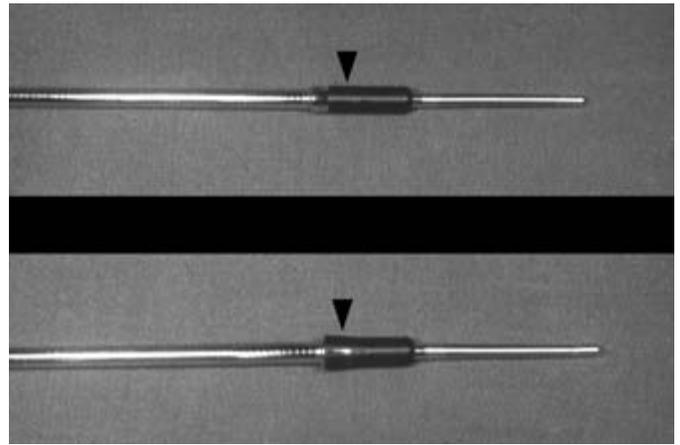
Figure 2.727 shows semirigid Atkinson prosthetic tubes for endoscopic intubation, made of radio opaque silicone rubber and available in different lengths. The proximal end is expanded into a soft funnel to collect food, and does not press too firmly against the oesophageal wall to obviate the risk of pressure necrosis; at the distal end there is a retaining device to prevent proximal migration. These tubes may be moved a little after insertion and can if necessary be removed.



2.727



2.728



The Nottingham introducer (Fig. 2.728) is used with the Atkinson tube (see also Fig. 2.727). At the distal end of the flexible spiral steel introducer there is an expanding sleeve which in the open position (*lower picture*) fits firmly into the prosthesis. The proximal end of the introducer has a locking device to hold the sleeve in the open or closed position. Following preliminary dilatation the assembly is passed over a guide wire which has been left *in situ* through the stricture. An optional rammer, not usually required, can be inserted into the funnel end to prevent slipping of the prosthesis.

Figure 2.729 illustrates a Celestin latex tube and introducer; this type of prosthesis is now rarely used.



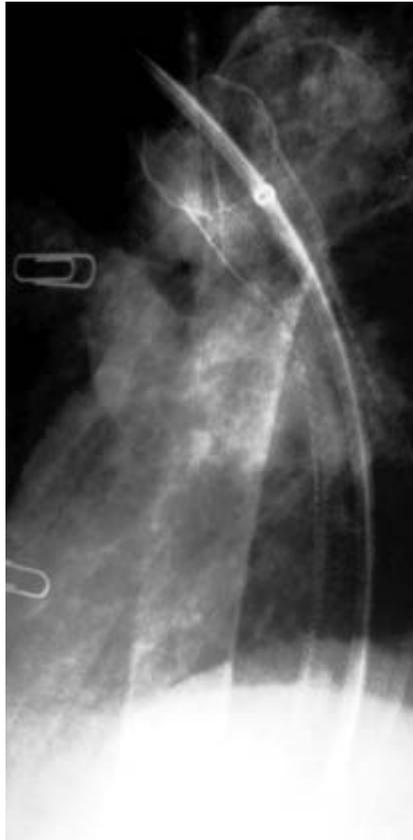
2.729

**Intubation (insertion of endoprosthesis)  
(cont.)**

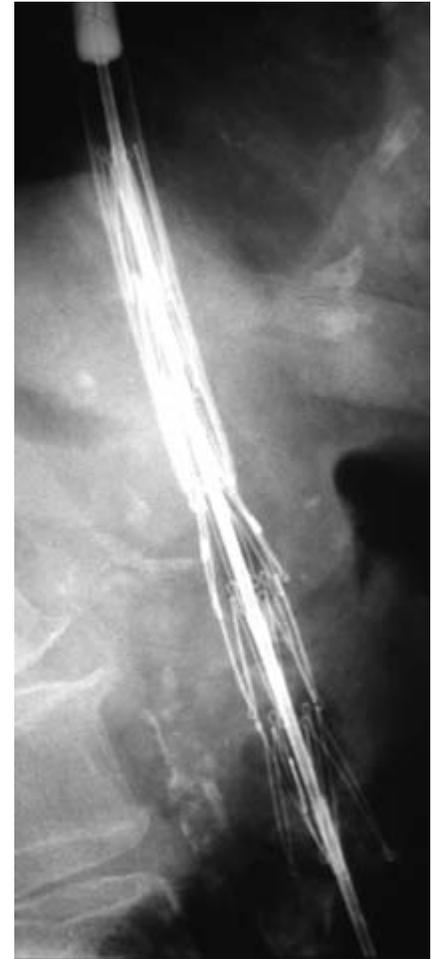
*Procedure*

After dilatation of the stricture to the diameter appropriate to accept the stent, the precise position and length of the tumour must be measured endoscopically to determine the required length of stent. The upper and lower ends of the tumour are then marked in some way to guide positioning. Possible methods are injection of radio-opaque contrast material into the oesophageal wall using an endoscopic injector, application of clips to the mucosa or use of external skin markers (Fig. 2.730) or combinations of these. Positioning of the prosthesis must be checked radiographically when employing either expanding metal stents or the Nottingham introducer. Figures 2.730 and 2.731 show deployment of metal stents (which vary between different manufacturers). When the stent is satisfactorily placed, the delivery system is released according to the instructions, and is withdrawn after separation has been confirmed by fluoroscopy.

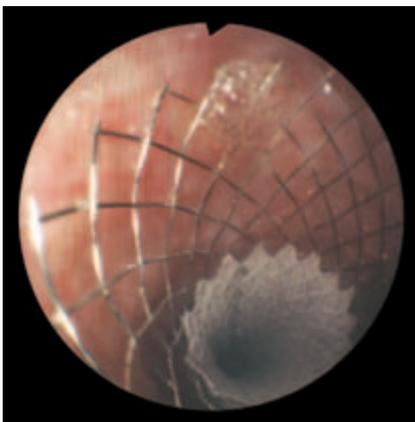
After placement, the prosthesis is inspected from above (Fig. 2.732 shows a plastic coated stent), from within (Fig. 2.733 shows an uncoated metal stent) and in some circumstances from below by passing a narrow diameter endoscope through the lumen and if necessary performing a J-manoeuvre in the stomach (Fig. 2.734 shows a Celestin tube).



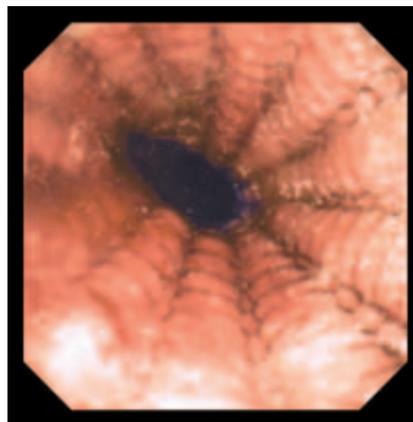
2.730



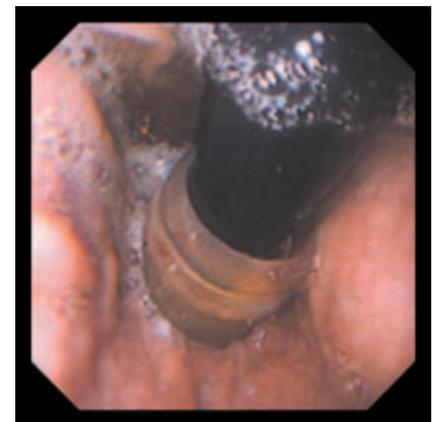
2.731



2.732



2.733



2.734

**Intubation (insertion of endoprosthesis)  
(cont.)**

*Procedure (cont.)*

This check ensures that the tumour is completely bridged. If this is not the case, repositioning is possible with the Atkinson type of tube by once more passing the guide wire followed by the introducer to pick up the prosthesis and move it. If a metal stent does not completely cover the tumour a second one will need to be placed within the first, extending the length as appropriate.

Plain radiographs of the chest show the position of prosthetic tubes: a well-penetrated film aids visualization. Atkinson tubes are wholly radio-opaque (Fig. 2.735). A chest radiograph should be taken after tube insertion for two reasons: firstly, it will serve as a baseline record of the position of the recently inserted prosthesis to compare with later radiographs if displacement is suspected; and secondly, it serves as an immediate check for the presence of surgical emphysema, a sign of complicating perforation which it must be remembered may be asymptomatic.



2.735

*Complications*

Recurrence of severe dysphagia is often due to the tube becoming blocked with food despite advice to chew thoroughly and to wash through with an effervescent drink after eating. Figure 2.736 shows a blocked tube. It is usually a simple matter to break up the debris with biopsy forceps, a small dilator (Fig. 2.737) or a snare (Fig. 2.738) and then to push the residue through into the stomach using a narrow endoscope in the manner of a piston. Rarely is a tube so firmly blocked with food residue as to require any alternative action.



2.736

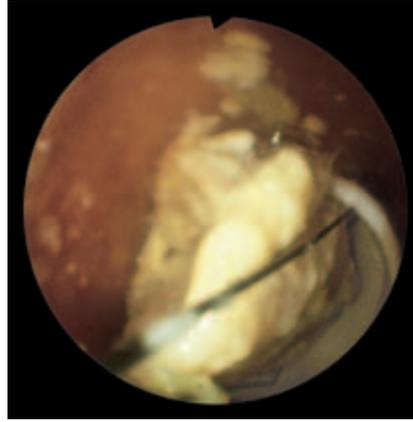


2.737

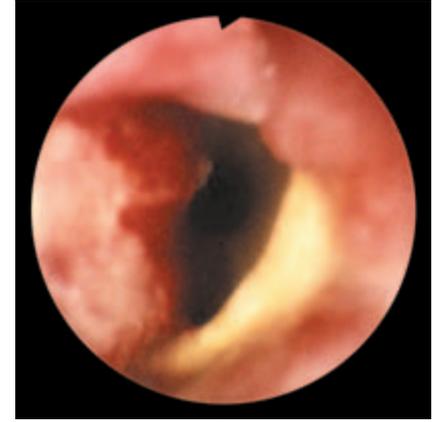
**Intubation (insertion of endoprosthesis)  
(cont.)**

*Complications (cont.)*

Proximal end overgrowth of a prosthetic tube by extension of carcinoma may eventually occur (Fig. 2.739). Blockage from this cause can sometimes be overcome if tumour tissue is destroyed by injection of alcohol or by ablation using laser therapy. An Atkinson tube may occasionally be moved slightly upwards or downwards as appropriate, and can sometimes be removed and replaced by a longer one. Insertion of a second metal stent within the first to lengthen the stented portion of oesophagus is possible except in high oesophageal lesions.



2.738



2.739

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**Forced dilatation for achalasia**

**Forced dilatation for achalasia**

There are two widely practised treatments for achalasia of the oesophagus: surgical myotomy (Heller's operation) and forced dilatation for which a range of equipment is available. Only the Microvasive® balloon device will be mentioned here. The recommended assembly includes a nondistensible polyurethane balloon 30, 35 or 40 mm in diameter (Fig. 2.740) which is passed over a guide wire, a syringe for injecting fluid for inflation and a pressure gauge. The guide wire is positioned with its tip well below the oesophagogastric junction, as described on pp. 198–199. After withdrawal of the endoscope, the balloon catheter is threaded onto the wire and passed under fluoroscopic control so that the radio-opaque markers lie either side of the diaphragm.



2.740

### Forced dilatation for achalasia (*cont.*)

Dilute water soluble radiographic contrast material is injected into the system and the position of the balloon adjusted under X-ray control so that the waist caused by the cardia is in the centre of the balloon (Fig. 2.741). When the position is correct more fluid is forcibly injected to expand the balloon until the sides are parallel (Fig. 2.742). This ensures that dilatation reaches the predetermined size of the balloon. After deflation, reflation is usually achieved at a much lower pressure indicating that the cardia has been stretched. On removal there is often a little blood on the balloon.



2.741



2.742

Perforation of the lower oesophagus occurs more commonly when treating achalasia than during dilatation of benign strictures, probably because the necessary extent of dilatation is greater. Thus very close observation is maintained after the procedure. When there is no pain or other cause for anxiety a drink of water is allowed after three or four hours, and as long as there is no concern the patient is allowed home to start a soft diet on the following day. On the other hand should there be pain or any other feature to suggest a possible perforation, a water soluble contrast X-ray swallow must be performed and the patient admitted to hospital. If a perforation has occurred surgical repair is appropriate if it is 'free' whilst contained perforation is usually managed conservatively.

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## Endoscopic management of oesophageal varices

### Injection sclerotherapy

### Rubber band ligation

### Ulceration following the treatment of oesophageal varices

Acute haemorrhage from oesophageal varices can often be arrested by injection sclerotherapy performed as an emergency procedure. Application of rubber bands is an alternative but more difficult technique in the acute situation. Sometimes compression of varices using a modified Sengstaken–Blakemore tube will be necessary to control the bleeding and allow resuscitation of the patient before endoscopic or other methods can safely be employed. The Sengstaken tube may also be used if endoscopic treatment fails or exacerbates bleeding at the first session.

The frequency of recurrent bleeding from varices can be considerably reduced by obliteration of the veins using repeated sclerotherapy or banding until all of the varices are seen to have been eradicated. Banding is superior both in terms of the proportion of cases controlled and also as a lesser number of sessions is required to achieve obliteration.

There is increasing evidence that banding may have a significant place in primary prophylaxis; injection sclerotherapy is not recommended for this purpose.

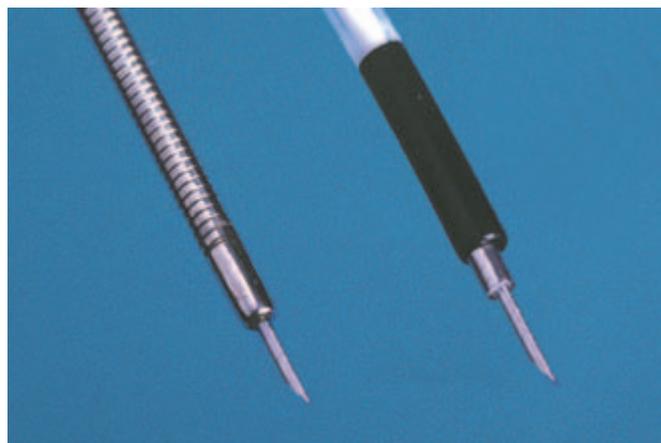
### **Injection sclerotherapy**

The usual method is to inject sclerosant into the lumen of the varix through a needle passed via the operating channel of a standard end-viewing or oblique-viewing endoscope. Usually this is an uncomplicated procedure, although on withdrawing the needle there may be minor bleeding from the puncture site. More severe bleeding may occur and is often controlled by passing the endoscope into the stomach so that the shaft overlies the bleeding point to provide tamponade. It is usual to make several injections during a session giving 1–2 mL intravenously (depending on the sclerosant used) at several sites along each of the varices starting just above the cardia. Repeated injections obliterate varices throughout their length in the oesophagus. The optimal interval between treatments is undecided; 2–4 weeks are often allowed to elapse permitting ulcers, should they have occurred, to heal between treatments. Sclerotherapy is repeated until all the varices have been obliterated; usually two to six sessions suffice. Interval follow-up to detect recurrence is recommended. Gastric varices are not normally injected, as sclerotherapy is not usually effective at this site, and injection may provoke severe haemorrhage.

Although intravariceal injection is the aim described above, some injections miss the target and are perivenous. Indeed, a smaller volume of sclerosant may be injected around the vein without danger and helps with eradication by inducing a tissue reaction which in itself may cause thrombosis of the varix. Furthermore such injections into the tissues produce local fibrosis, another aid to variceal elimination. This technique is favoured as first choice by some endoscopists who recommend multiple small perivenous injections round the oesophageal circumference at several levels, extending proximally to the extent of the varices, with the aim of inducing generalized sclerosis of the oesophagus.

### *Endoscopic injection needles*

Two types of endoscopic injection needle are illustrated in Fig. 2.743. The needle tips are retractable, but are here shown protruded to about 5 mm. Such needles are used for all endoscopic injection techniques.

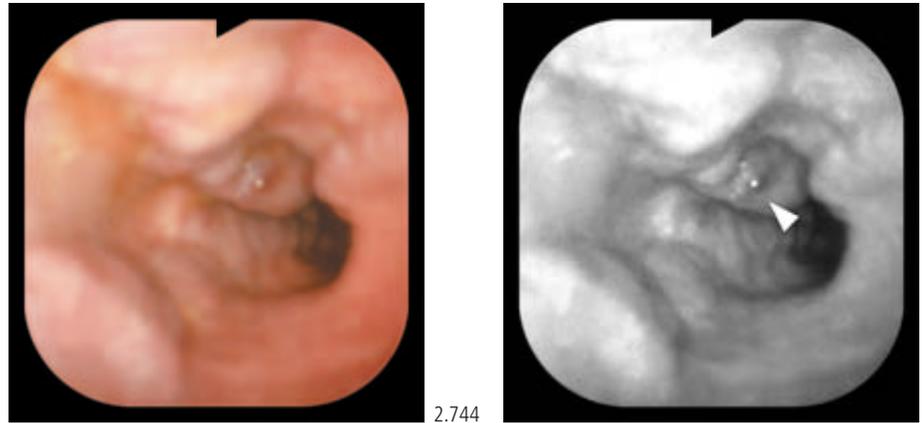


2.743

### **Injection sclerotherapy (cont.)**

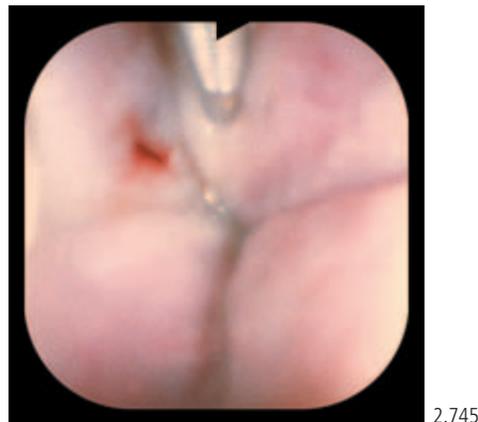
#### *Variations in variceal appearances*

Oesophageal varices bulge into the lumen (Fig. 2.744) even when air distension is adequate: a necessary diagnostic prerequisite to avoid confusion with longitudinal mucosal folds which are flattened with air distension. If the overlying mucosa is thin, varices may appear blue; there will be less colour difference when the overlying mucosa is thicker. Often there is evidence of recent or potential haemorrhage in the form of red spots on the surface of varices (Fig. 2.193). The number, distribution, relative size and longitudinal extent of the oesophageal varices may be recorded according to the clock convention, anterior representing 12 o'clock. Such recording is useful when assessing the results of treatment. The varices in this patient (Fig. 2.744) are situated at 7 and 11 o'clock, the endoscope having been correctly placed, and extend upwards from the cardia to 25 cm from the incisors. Photographic or videoprint images may be useful to record progress of a course of treatment. (Variations in variceal appearances are also discussed on pp. 68–69.)



#### *Injection of varix*

The endoscopic injector has been applied to the varix 3 cm above the cardia by angulating the endoscope tip slightly (Fig. 2.745). The injection needle has been advanced to puncture the varix to inject intravenously. The sclerosant must be injected by an assistant as both of the endoscopist's hands are employed maintaining a stable position with a clear view and manipulating the injection catheter. (As sclerosants are irritant, special care is required from the assistant to ensure that the junction between the syringe and needle is secure to avoid spraying the patient or staff: corneal ulcers have been produced. A Luer lock type of fitment is preferred.) The volume of sclerosant injected varies according to the agent and technique applied. A previous injection site is shown by a red mark at 10 o'clock.



### Injection sclerotherapy (*cont.*)

#### *Injection sites after withdrawal of needle*

In the presence of Barrett's oesophagus injection sites may be below the squamo-columnar mucosal junction but will still be within the tubular oesophagus (Figs 2.746 and 2.747).

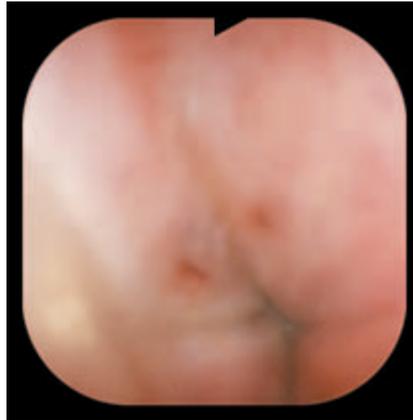
Two injections have been given and there was no significant haemorrhage (Fig. 2.748). More obvious bleeding is shown in Fig. 2.749.



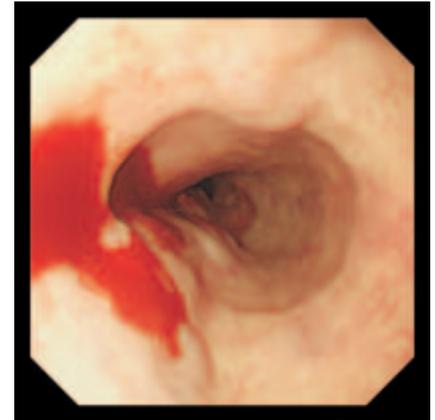
2.746



2.747



2.748



2.749

### *Complications*

Bleeding as a complication of sclerotherapy has already been mentioned. Retrosternal pain during, and sometimes following the procedure, is not uncommon. Pneumothorax and mediastinitis are rare but serious complications, as is stricture formation. Ulceration, which is a common sequel but could be regarded as a complication, is discussed on pp. 212–213.

### **Rubber band ligation**

By a method analogous to that long established for treatment of haemorrhoids, small rubber bands applied to varices eradicate them efficiently, safely, and more rapidly than does sclerotherapy. Earlier techniques used one band at a time necessitating multiple re-intubations and an overtube (Fig. 2.696) and were therefore cumbersome and difficult to employ. Development of multiband ligation devices (Fig. 2.750) has revolutionized the technique allowing a series of bands to be applied following a single intubation, without the need to employ an overtube.



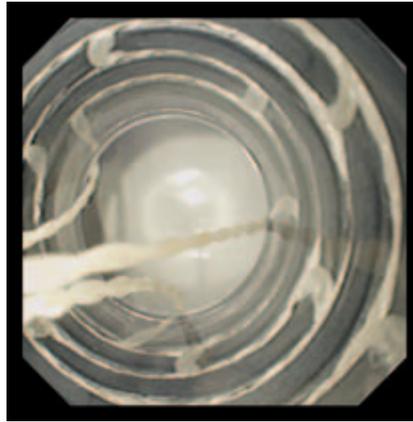
2.750

**Rubber band ligation (cont.)**

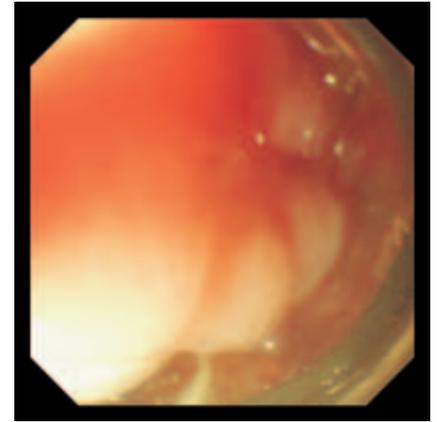
Multiple bands are preloaded on the barrel of the device which attaches to the end of the endoscope and from which they are ‘fired’ onto the varix. The presence of the barrel restricts the view (Fig. 2.751), especially early in the procedure when many bands are loaded but vision progressively improves as the bands are deployed, as is seen in the following figures.

The technique involves aspirating the varix deeply into the end of the barrel (Fig. 2.752) when the firing mechanism is operated. This releases a band onto the varix, which then retracts from the barrel (Fig. 2.753). Multiple varices (Fig. 2.754) or many sites along a varix (Fig. 2.755) may be treated in turn during a single session. Therapeutic sessions are repeated at intervals of 1–4 weeks until the varices are eradicated (Fig. 2.756).

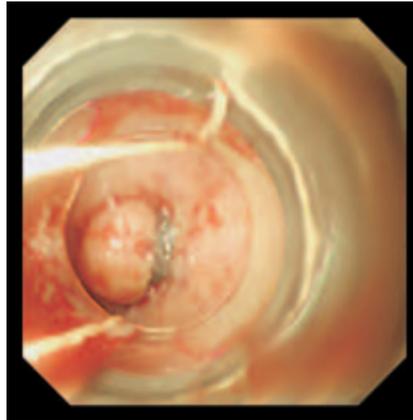
Usually there are no complications although postprocedural pain is common and some ulceration and secondary haemorrhage can occur.



2.751



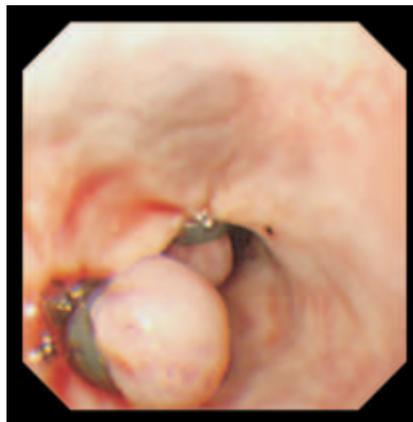
2.752



2.753



2.754



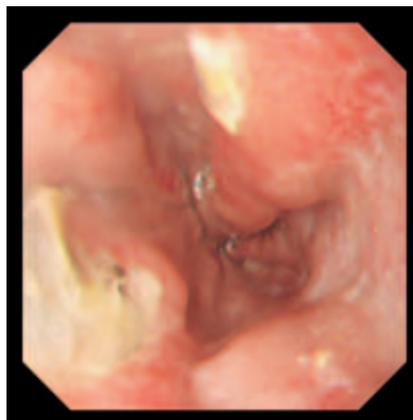
2.755



2.756

**Ulceration following the treatment of oesophageal varices**

Small areas of mucosal ulceration immediately overlying the injection site (Fig. 2.757) occur commonly. Ulcers usually heal within 2–4 weeks. Sometimes larger areas of ulceration may result with formation of much slough (Fig. 2.758).



2.757



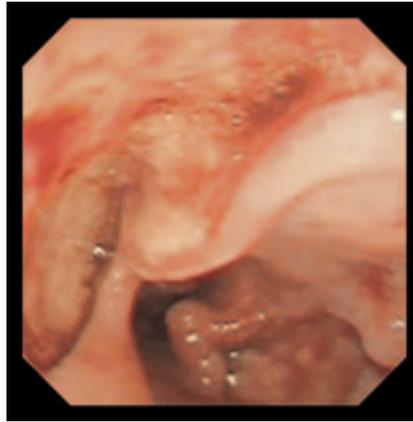
2.758

### Ulceration following the treatment of oesophageal varices (*cont.*)

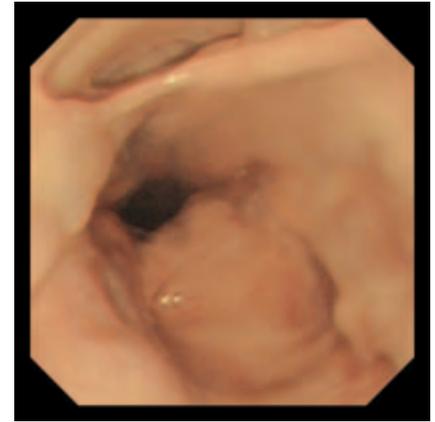
Figures 2.759 and 2.760 show evolution from acute ulceration to scarring with mucosal deformity over an eight month period. Should a stricture result (Fig. 2.761), it is treated endoscopically as described on pp.197–201. Ulceration is common after large volume extra-variceal injection but is also seen following intravenous injection, when the exposed lumen of the vein may be visible with thrombus within.

Rubber band ligation of varices can also result in ulceration of the site from which the varix has been eradicated.

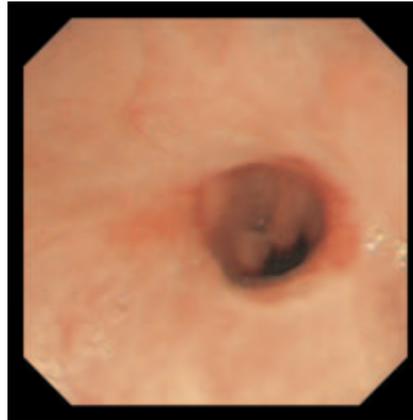
A further cause of iatrogenic mucosal damage or ulceration (Fig. 2.762) is the use of a Sengstaken–Blakemore tube to control haemorrhage either before or after sclerotherapy.



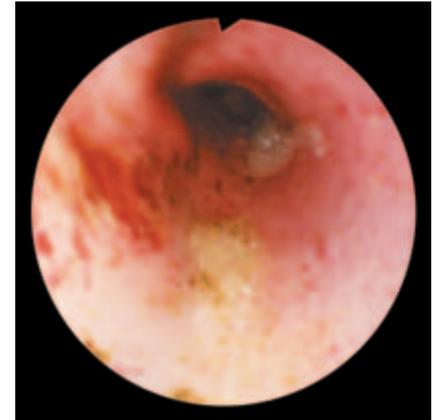
2.759



2.760



2.761



2.762

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## Treatment of nonvariceal vascular and bleeding lesions

### Injection therapy of bleeding peptic ulcer

### Treatment by heater probe

### Laser treatment

### Use of haemoclips

### Therapy of mucosal vascular lesions

Despite advances in the treatment of peptic ulceration, acute bleeding from gastric or duodenal ulceration remains a common clinical problem which requires urgent endoscopy both for diagnosis and therapeutic intervention. Injection therapy and application of heat by means of heater probe or laser have been shown by controlled clinical trials to improve outcome. Other techniques including application of clips, use of tissue adhesives or endoscopic suturing have not yet gained wide acceptance.

### Injection therapy of bleeding peptic ulcer

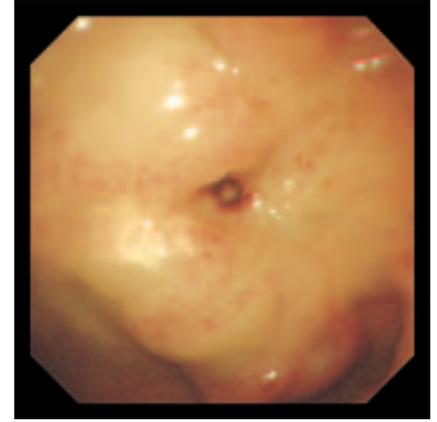
Using a standard endoscopic injection needle (Fig. 2.743) 1 mL aliquots of dilute adrenaline solution (usually 1 : 10 000) are placed around the visible vessel (Fig. 2.763) or actual bleeding point. Blanching and/or cyanosis of the surrounding mucosa occurs and there is tamponade of vessels due to increased tissue pressure from the volume injected. Figure 2.764 shows blanching and oedema surrounding a minute prepyloric ulcer (or Dieulafoy lesion). Figures 2.765 and 2.766 show blanching and cyanosis around injected duodenal ulcers and Fig. 2.767 extensive blanching around a distal lesser curve gastric ulcer. There is uncertainty as to how much adrenaline is required or the optimum strength of solution to use, or whether alternative agents or sclerosants alone or in combination are superior. Further controlled clinical trials should define the optimum regimen. Similarly there is debate as to whether the bleeding point itself should be touched or injected.

Spurting vessels (Fig. 2.634) may require application of a heater probe or perhaps sclerosant in addition to a vasoconstrictor agent to effect control of bleeding.

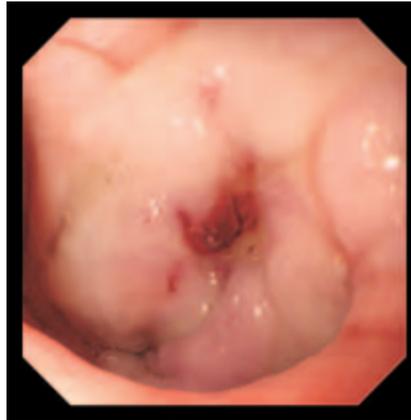
As alternatives or additions to injection of adrenaline thrombophilic agents such as thrombin or fibrin glue have been used with reported success but as yet have not been accepted into routine practice. Similarly tissue adhesives have not achieved general approval. Nevertheless it is probable that advances will be made in the employment of haemostatic agents in the control of this common acute medical emergency.



2.763



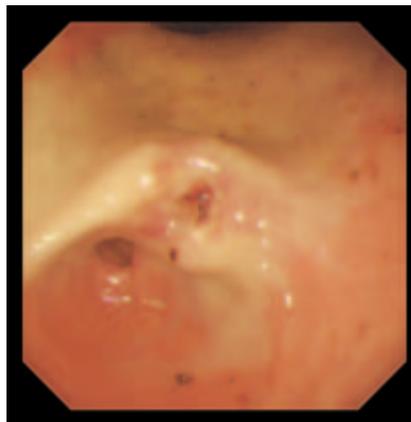
2.764



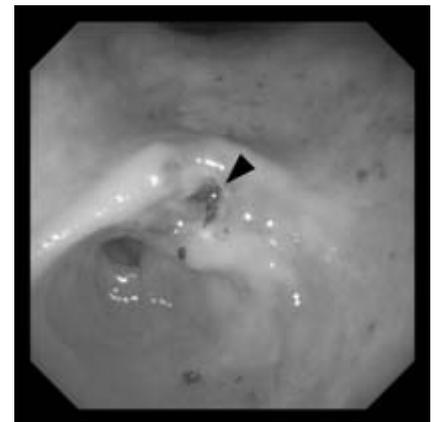
2.765



2.766



2.767

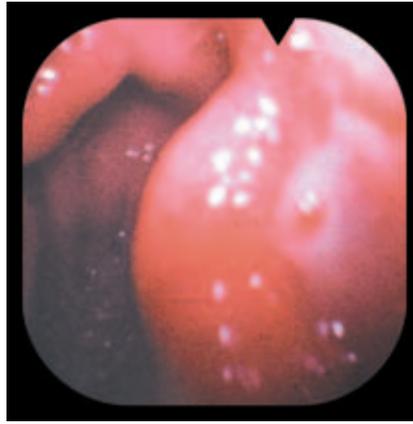


**Treatment by heater probe**

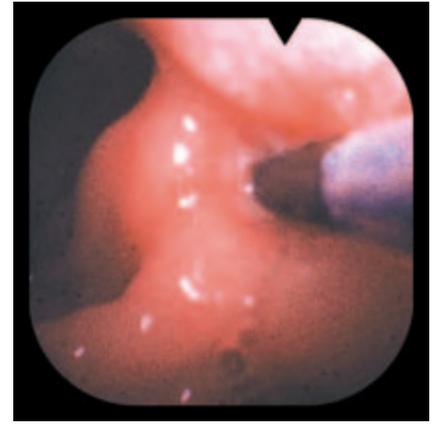
The heater probe is one of several available thermal devices. Figure 2.768 shows a raised red area, representing a visible vessel in a gastric ulcer before treatment, and in Fig. 2.769 the tip of the heater probe is applied to this vessel.

A bleeding gastric ulcer seen in Fig. 2.770 was successfully treated by heater probe (Fig. 2.771). The ulcer was subsequently found to be carcinomatous, and the patient underwent surgery.

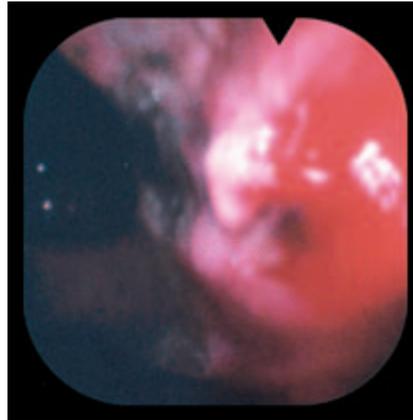
Unipolar and multipolar diathermy (Fig. 2.772) have also been used as methods of applying heat to bleeding lesions but these techniques will not be discussed further here.



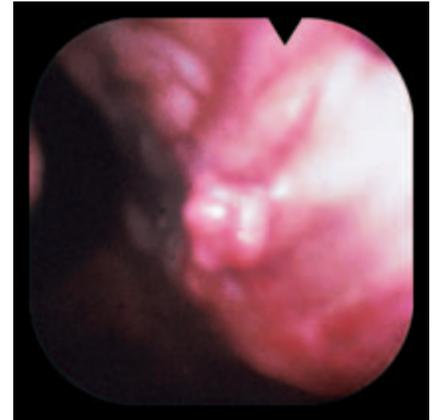
2.768



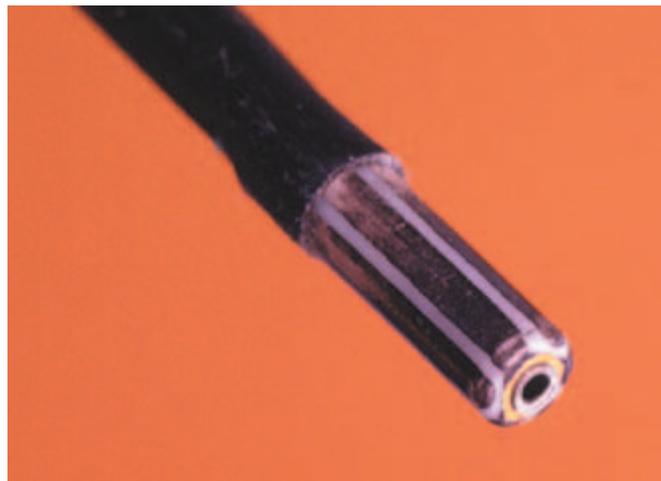
2.769



2.770



2.771

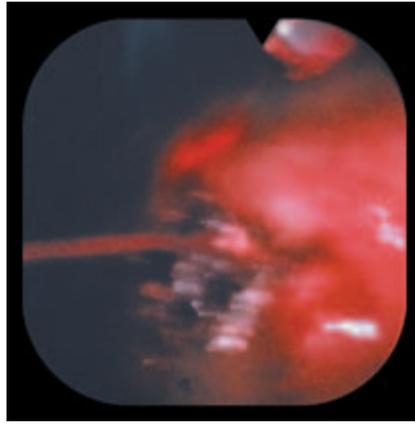


2.772

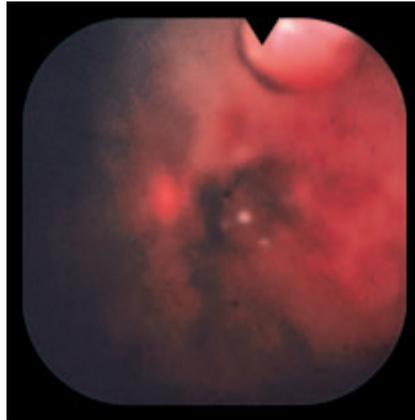
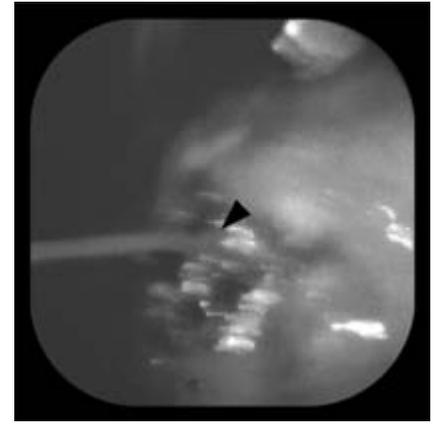
### Laser treatment

Laser therapy (usually Nd-YAG) is an alternative method of applying heat to coagulate the tissue adjacent to a bleeding point so aiding control of haemorrhage. Figures 2.773 and 2.774 show the effect of the Nd-YAG laser on acute arterial bleeding from a gastric ulcer. The red spot (aiming beam) marks the area at which the invisible laser beam is aimed. Laser treatment is much more expensive in terms of capital outlay and fibres and is not widely available outside specialist units.

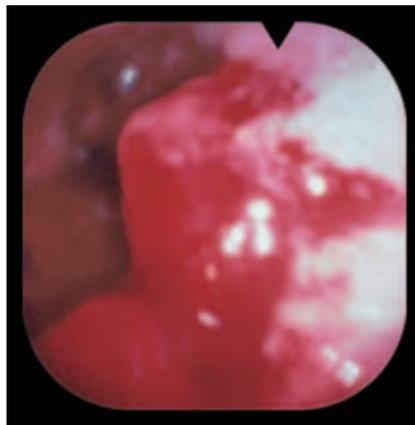
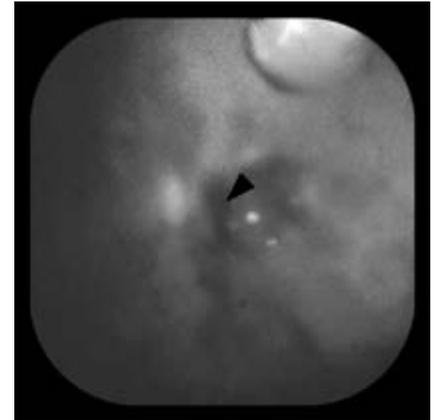
Other actively bleeding lesions may be treated in a manner similar to peptic ulceration. In Figs 2.775 and 2.776 oozing from a gastric carcinoma is arrested by the same method.



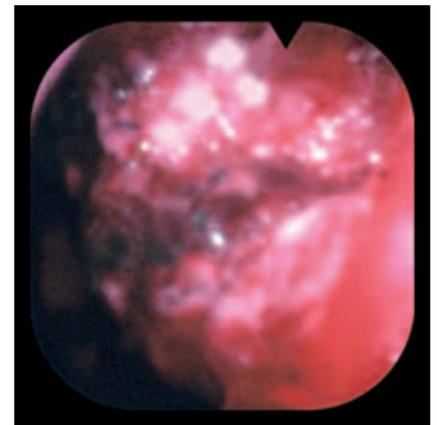
2.773



2.774



2.775



2.776

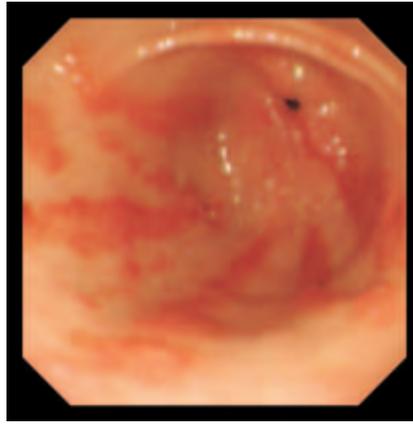
### Use of haemoclips

5 mm haemoclips can be applied to the gastrointestinal mucosa in the hope of controlling a bleeding point. Numerous individual cases have been observed where this technique has stopped a spurting vessel but experience is limited and results are too unpredictable at present to make this a first line treatment.

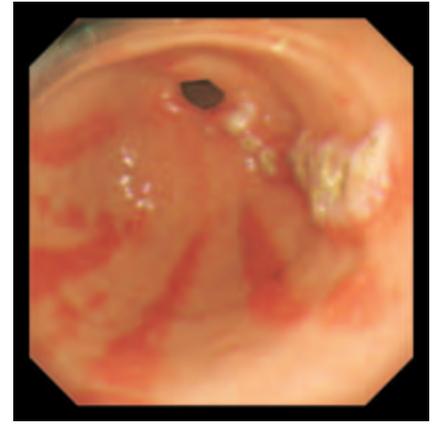
### Therapy of mucosal vascular lesions

Endoscopic therapy may be successful in the eradication of many vascular lesions notably small angiomata, areas of angiodysplasia and in hereditary haemorrhagic telangiectasia (Osler-Rendu-Weber syndrome). Antral vascular ectasia ('water melon' stomach) also is amenable to endoscopic treatment. Small vascular lesions may be controlled in a single session but large lesions especially water melon stomach may require multiple endoscopies to control and deal with recurrences.

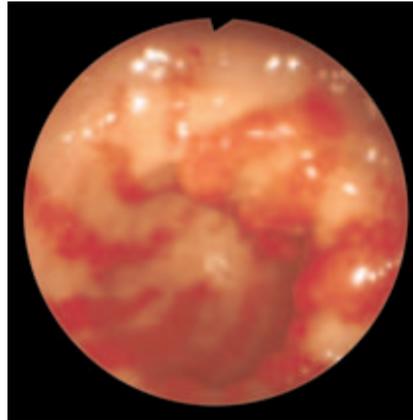
Figure 2.777 shows antral vascular lesions before and Fig. 2.778 after treatment of one row with argon plasma coagulation. Figures 2.779 and 2.780 show similar before and after appearances using the Nd-YAG laser. Ulcers created by these techniques (Fig. 2.781) usually heal in two to four weeks. Sometimes mucosal scarring, deformity and rarely hyperplastic polyps (Figs 2.354 and 2.355) result from this treatment.



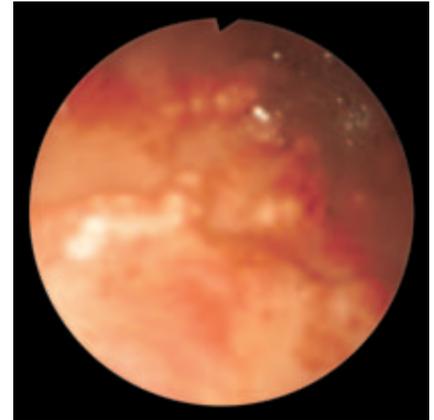
2.777



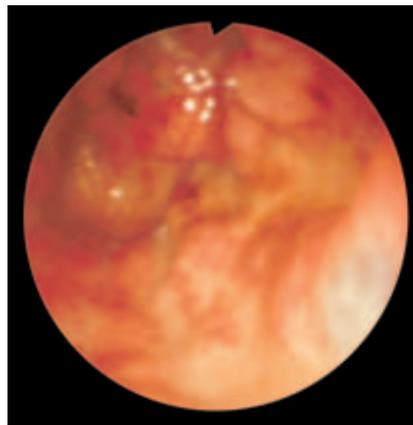
2.778



2.779



2.780



2.781

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## Intubation for enteral nutrition

### Simple tube placement

#### Passage of tube over guide wire

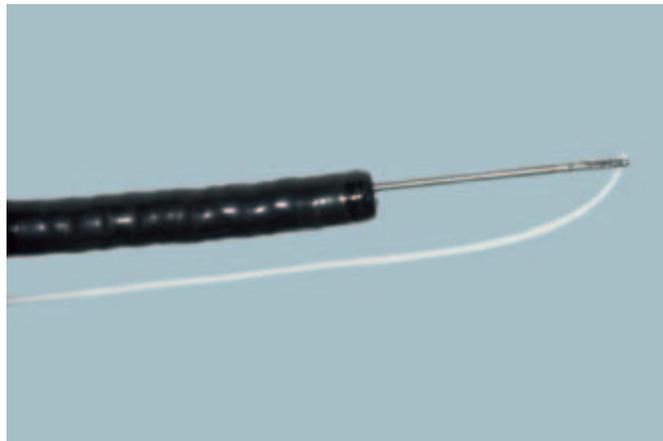
#### Percutaneous endoscopic gastrostomy (PEG)

Increasing use of enteral nutrition has stimulated development of endoscopic techniques of tube placement. Three basic methods are employed, namely grasping a feeding tube in forceps and passing it with the endoscope, passing a guide wire endoscopically, leaving it in place and then inserting a tube over the wire and, thirdly, percutaneous endoscopic gastrostomy (PEG) or percutaneous endoscopic jejunostomy. Detailed indications for the use of enteral nutrition and selection of techniques are not considered in this work.

### Simple tube placement

Occasionally a feeding tube cannot be passed through a stricture, a surgical anastomosis or the pylorus despite using a guide wire. Endoscopic guidance may overcome these difficulties.

The tube is held in a grasping forceps previously passed through the operating channel of the endoscope (Fig. 2.782). The instrument, with tube attached, is advanced to the stricture. Figure 2.783 shows the grasping forceps taking the feeding tube through the stricture. When correctly positioned the tube is released, and the forceps and endoscope withdrawn. Care must be taken lest friction between the tube and the endoscope results in these being inadvertently withdrawn together. Generous lubrication reduces friction and temporary use of a guide wire by increasing the rigidity of the tube may help avoid this problem.



2.782

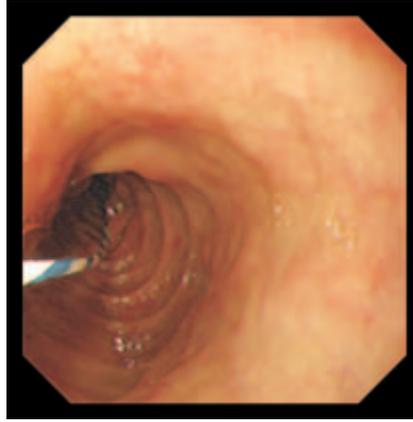


2.783

If so desired, the feeding tube can be passed by the nasal route. Initially it is advanced transnasally to the pharynx where, using a laryngoscope and Magill's forceps, the tip is grasped and pulled out through the mouth. It is then inserted into the endoscopic grasping forceps as described above and placed appropriately.

### Passage of tube over guide wire

It is possible to pass some types of fine bore or double lumen (naso-jejunal) tubes into the jejunum over a guide wire. The wire is passed through the endoscope into the distal duodenum (Fig. 2.784) or beyond the ligament of Treitz and left in place whilst removing the endoscope. The proximal end of the wire is then re-routed through the nose as described on p. 430. Next, a well lubricated feeding tube is passed through the nose over the guide wire and advanced as far as possible, which should be into the small bowel, as long as the wire has not retracted. The position of the tube is checked radiographically (Fig. 2.785) and if satisfactory the guide wire is removed. If the tube has retracted endoscopic adjustment may be helpful although often it is better to start again.



2.784



2.785

### Percutaneous endoscopic gastrostomy (PEG)

Long-term enteral nutrition can conveniently be administered through a PEG tube, which avoids the discomfort of a tube passed via the nose and is aesthetically more acceptable for outpatient or nursing home care.

#### Equipment

Several commercial kits are available comprising the feeding tube, its various fittings, a trocar and cannula set and a thread or wire for drawing the tube into position (Fig. 2.786). Some kits provide a longer inner portion to allow positioning beyond the pylorus or a flush fitting 'button' (Fig. 2.787) which avoids the long external portion.



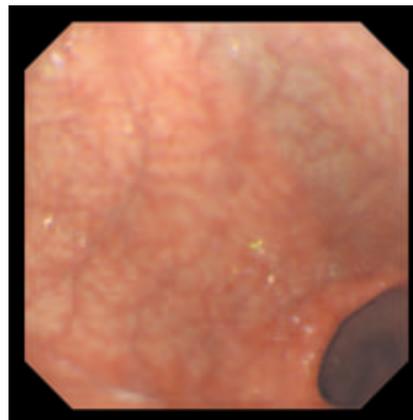
2.786



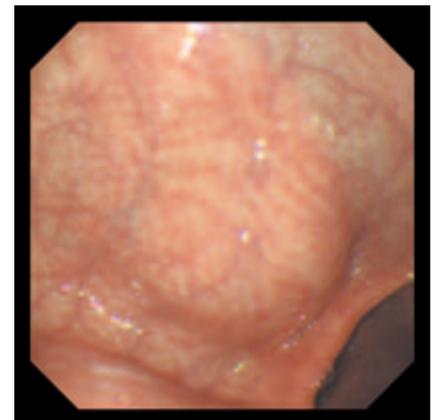
2.787

#### Technique

After the endoscope has been passed into the stomach, the site for insertion is identified by indenting the stomach wall. Figure 2.788 shows the endoscopic appearance before indentation, and Fig. 2.789 after indentation with finger pressure on the upper abdomen.



2.788



2.789

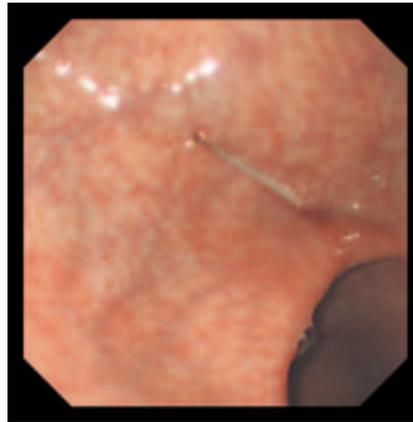
**Percutaneous endoscopic gastrostomy (PEG) (cont.)**

*Technique (cont.)*

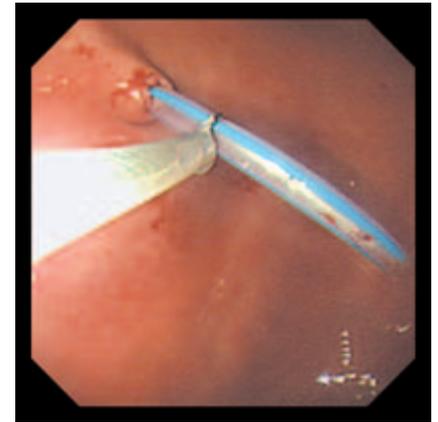
At the same site and time transillumination of the abdominal wall (Fig. 2.790) from the endoscope must be obtained to ensure that there is no intervening organ. When this has been ascertained the abdominal wall is punctured by a fine needle which is used for infiltrating local anaesthetic (Fig. 2.791). If the position is satisfactory, local anaesthesia is completed and the trocar and cannula are inserted through the abdominal wall into the stomach. The cannula is temporarily grasped with an endoscopic snare (Fig. 2.792) and the trocar removed. A fine thread or wire is introduced through the cannula (Fig. 2.793) from which it is drawn out into the endoscope using the snare. The thread is now brought out through the mouth by removing the endoscope and snare together. The PEG tube is next attached to the oral end of the thread, by gentle traction on the distal end of the thread, currently protruding from the cannula, the PEG tube is pulled through the mouth and down the oesophagus until the tapered end of the tube impacts in the lumen of the cannula. Next, the thread and cannula are withdrawn, delivering the PEG tube through the abdominal wall. The internal flange *in situ*; patency of the device can be confirmed by injection of water (Fig. 2.796). After fixing to the skin according to the manufacturers' instructions the device is ready for use under the guidance of a dietician.



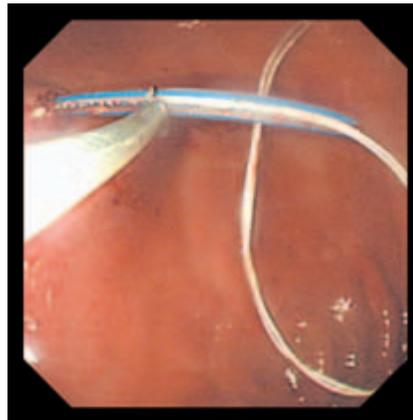
2.790



2.791



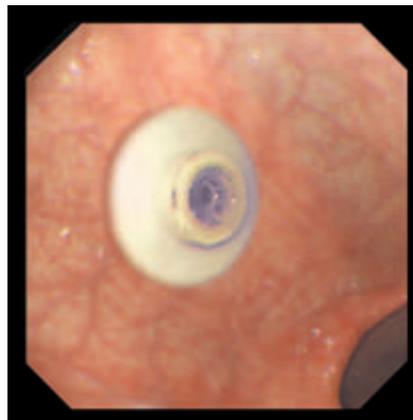
2.792



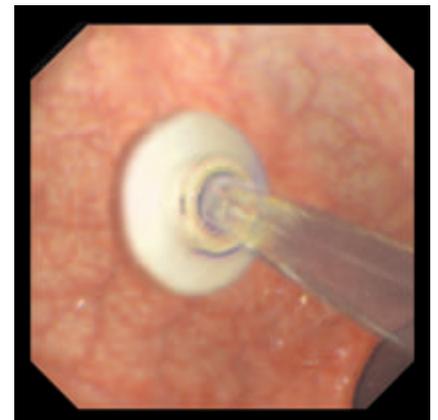
2.793



2.794



2.795



2.796

## Percutaneous endoscopic gastrostomy (PEG) (cont.)

### Technique (cont.)

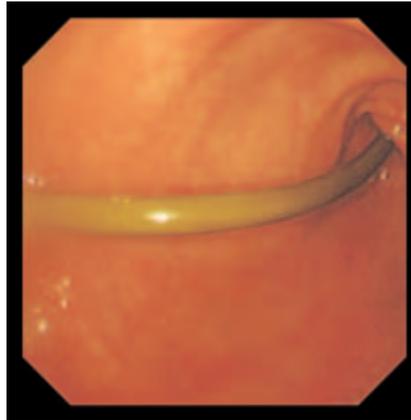
Serious complications of PEG placement (haemorrhage, perforation, puncture of the wrong viscus, gastro-colic fistula or overwhelming infection) and usage are rare, but minor sepsis around the puncture site is quite frequent. Irritation of the gastric mucosa by the internal part of the PEG may occur and cause *contre-coup* ulceration (Fig. 2.797). Deterioration of the plastic material and fungal colonization of the internal portion may occur after prolonged intubation (Fig. 2.798). Rarely the internal disc may become buried in the gastric wall due to mucosal overgrowth, only a small punctum remaining. Periodic releasing of the external fixation and ensuring the mobility of the established PEG device should prevent this occurring. However if the fixation is too loose the disc or balloon may be drawn through the pylorus by peristalsis (Fig. 2.799).



2.797



2.798



2.799

### Removal of PEG tube

Removal should not be contemplated before 10–14 days have elapsed to allow a secure track to form between the gastric lumen and the skin without risk of peritoneal leakage. Depending upon the type of PEG tube used, removal may be possible by external traction or may need a further endoscopy to snare the inner flange (Fig. 2.800). The fistula remaining (Fig. 2.801) after removal of the PEG tube, seen above a snare in this figure, closes rapidly, usually within a few hours.



2.800



2.801

## Endoscope-guided intestinal biopsy using a Crosby or Watson capsule

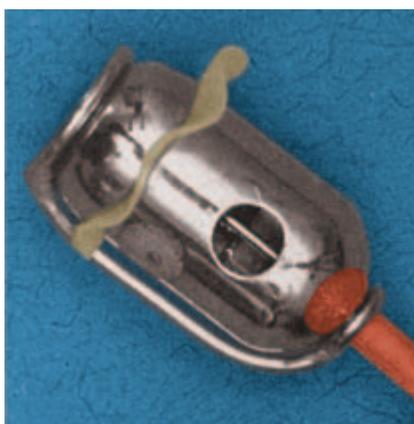
### Endoscope-guided intestinal biopsy using a Crosby or Watson capsule

Although not strictly a therapeutic procedure it is convenient to describe here this technique which provides samples of duodenal mucosa larger than can be obtained by forceps biopsy. Most pathologists find four standard forceps biopsies of the distal duodenum adequate to exclude coeliac disease and similar disorders. The use of the Crosby capsule has declined except when larger mucosal samples are required or mucosa from beyond the ligament of Treitz is essential for diagnosis and enteroscopy is not available. It may be performed rapidly by muzzle loading a Crosby or Watson capsule on to an endoscope. This is done by threading the suction tube retrogradely through the biopsy channel from which the entry valve is removed.

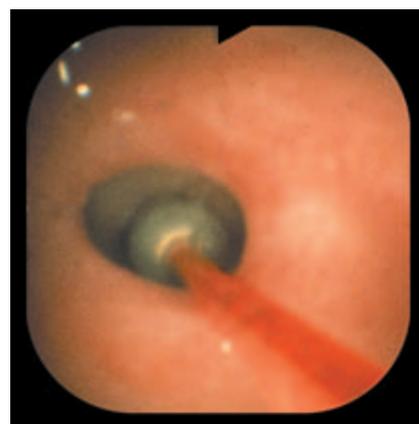
The endoscope and attached capsule (Fig. 2.802a) are passed into the oesophagus in the usual manner. If desired, the capsule can be pushed a short distance ahead to allow better vision.

Usually the combined assembly can be passed rapidly through the pylorus (Fig. 2.802b) to the duodenum, following which the capsule is advanced further and out of sight to take the sample. Fluoroscopy can be performed to check positioning if required.

It is convenient to employ a short overtube (Fig. 2.696) during this procedure so that should the biopsy be inadequate the assembly can be reintroduced easily with little discomfort to the patient. Multiple samples may be obtained by the same method.



2.802a

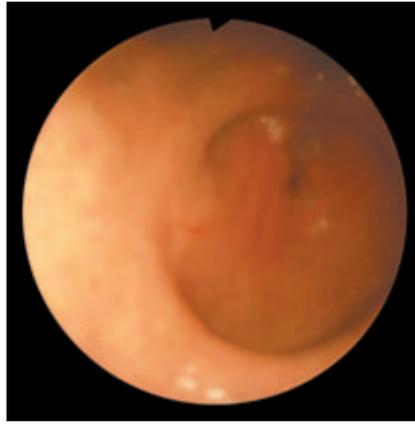


2.802b

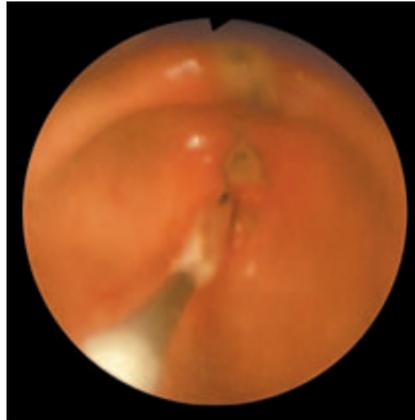
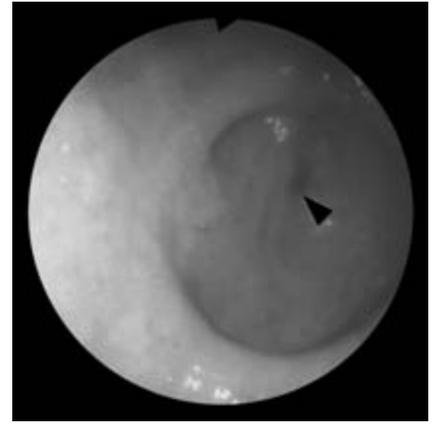
## Balloon dilatation of pyloro-duodenal stenosis

### Balloon dilatation of pyloro-duodenal stenosis

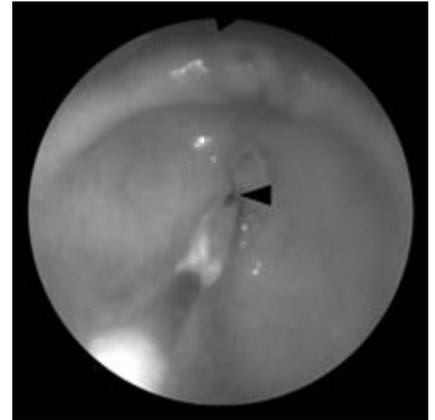
Strictures of the pyloro-duodenal region causing gastric outflow obstruction may complicate chronic peptic ulceration. Whilst surgical treatment is needed for densely fibrotic stenoses, some cases respond satisfactorily to balloon dilatation using the through-the-scope (TTS) technique. Figure 2.803 shows the narrowed pylorus through which a balloon catheter was threaded (Fig. 2.804). The large gastric residue had previously been aspirated but note vigorous antral peristalsis. When the site of narrowing is endoscopically visible, dilatation using a fluid filled balloon may be observed directly (Fig. 2.805). The result of dilatation with a little bleeding is seen in Fig. 2.806 when the lumen was large enough to allow passage of the endoscope into the duodenal cap (Fig. 2.807). Should the narrowing be in the duodenum, where curvature makes vision difficult, it is better to employ fluoroscopy to follow progress with obliteration of the waist of the balloon on inflation (as already described on p. 208). The usual medical therapy is needed for the underlying duodenal ulceration. Dilatation may need to be repeated to obtain a satisfactory clinical result.



2.803



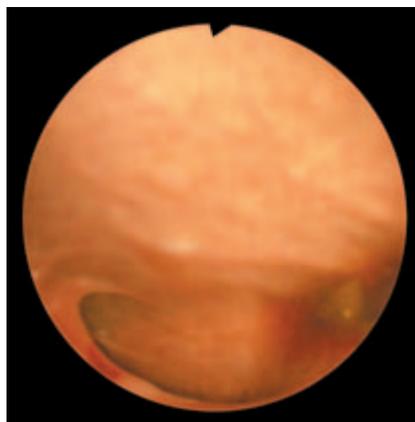
2.804



2.805



2.806



2.807

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## **Polypectomy**

### **Polypectomy**

The technical aspects of upper and lower gastrointestinal polypectomy are similar. As the technique is employed most frequently in colonoscopy, it is described and illustrated on pp. 327–335.

The endoscopic appearances of polypoid lesions of the upper gastrointestinal tract are shown earlier in this chapter. Adenomatous polyps in this region are relatively rare. Many polypoid lesions are due to submucosal benign tumours such as GISTs and are often sessile and may be extraluminal. Consequently, an attempt at polypectomy may fail to give adequate material for tissue diagnosis, may not eradicate the lesion and may be dangerous. Careful consideration should precede a decision to attempt endoscopic removal of sessile lesions in the oesophagus, stomach and duodenum: the gain may be slight and the risks of severe haemorrhage and perforation are greater than in the colon. Pedunculated lesions however, can usually be removed safely.

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## **Mucosal resection**

### **Mucosal resection**

The loose attachment of the mucosa to the underlying muscular coats of the intestinal wall creates the potential to remove large pieces of mucosa without risk of bowel perforation. In this manner flat superficial tumours or premalignant lesions can be resected completely and delivered for pathological examination to ensure a correct diagnosis and completeness of excision. This method is suitable for removing early gastric cancer when surgery is contraindicated or refused, for flat polyps or when looking for localized high grade dysplasia. The procedure is described and illustrated on p. 332.

## Complications

### Perforation

#### Fistula

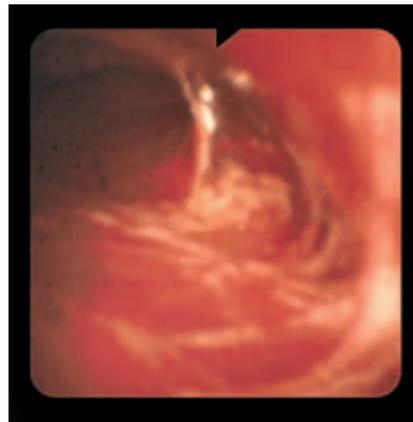
The risk of perforation has been alluded to on many occasions in previous sections. Fortunately complications occur infrequently during diagnostic upper gastrointestinal endoscopy though they are commoner following therapeutic procedures. It is possible to cause excessive bleeding, for example during a biopsy manoeuvre, and other endoscopically visible complications are described. It is not surprising that in the heat of the moment, especially in the sick patient, these are not always photographically recorded. Complications that may be seen through the endoscope include perforation into the mediastinum, pleural cavity and peritoneal cavity.

In addition there are many complications of endoscopy that photography cannot record. These include, for example, the cardio-respiratory risks of sedation such as hypoxia and hypotension, and other cardiovascular problems such as dysrhythmias.

#### Perforation

This is usually recognized immediately if severe pain, surgical emphysema or shock develop. Nevertheless, a small tear is not always apparent at the time, and later onset of these symptoms is sinister. Plain radiographs demonstrate mediastinal air; the site of perforation can usually be shown by use of a water-soluble contrast examination. To detect unsuspected perforation after oesophageal dilatation or intubation some endoscopists routinely examine the oesophagus by contrast radiology before allowing oral feeding.

Figure 2.808 shows the appearance of bleeding loose areolar tissue in the upper mediastinum distended with air, the perforation having occurred high in the oesophagus. Figure 2.809 illustrates blood-stained fatty omentum; in this case the stomach was perforated just above a benign gastric stricture.



2.808



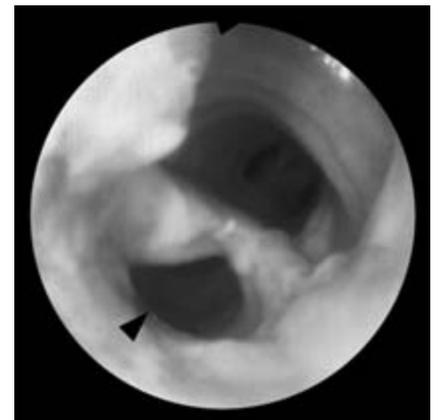
2.809

#### Fistula

The oesophago-tracheal fistula seen in Fig. 2.810 occurred during endoscopy following radiotherapy for a squamous cell carcinoma of the postcricoid region. The tumour had responded to treatment but had become necrotic, breaking down during the passing of the endoscope.



2.810



# Lower Gastrointestinal Tract

<b>Normal appearances</b>	<b>229</b>
<b>Abnormal appearances</b>	<b>240</b>
<b>Postoperative appearances</b>	<b>318</b>
<b>Therapeutic procedures</b>	<b>327</b>
<b>Complications</b>	<b>341</b>

Total colonoscopy allows examination of rectum, colon and caecum, and usually the terminal ileum. A shorter instrument, the flexible sigmoidoscope, may be used when it is planned only to inspect the large bowel distal to the splenic flexure.

A double-contrast barium enema examination is often performed as the initial investigation of colonic symptoms. Colonoscopy is indicated if radiological appearances are equivocal, when biopsy or polypectomy are necessary, and when radiology is normal despite significant symptoms, especially rectal bleeding. Increasingly, colonoscopy is undertaken as the first investigation and for surveillance.

Before examination begins all faecal matter must be evacuated. Good preparation is essential for satisfactory visualization (Figs 3.1–3.5), to permit intubation under optimal conditions, and not least to improve the aesthetic aspects of colonoscopy. There is a wide choice of preparatory regimens. Oral fluids only for 24 h or more before the procedure, cathartics (irritant or osmotic) and rectal washouts or enemas, either alone or in combination, are used. Preparation may occasionally influence mucosal appearances (p. 309).

Colonoscopy is usually performed under light sedation often with an analgesic added as the procedure may cause pain due to over-insufflation, stretching of bowel loops or mesentery. Powerful narcosis or general anaesthesia are best avoided as the lightly sedated patient can warn the endoscopist if too forceful a technique is used.

After preliminary digital examination which helps to lubricate and dilate the anal sphincter, the instrument is inserted into the rectum by pressing its tip through the sphincter. Advancement around the colon is achieved under direct vision. Careful insufflation of air or CO<sub>2</sub>, which is more rapidly absorbed, distends the colon to allow recognition of the lumen. Over-distension should be avoided as it is the most common cause of pain and may extend loops making angulations between free and fixed segments, such as descending and sigmoid colon, so that insertion is much more difficult.

Care must be taken to differentiate between the lumen and diverticular orifices (Fig. 3.57). Introduction of the instrument tip into or over-insufflation of a diverticulum may cause perforation (see p. 342).

During the insertion phase of colonoscopy, attention is usually concentrated on advancement of the instrument though appearances must be noted, whilst meticulous examination is generally performed during withdrawal. Landmarks are less evident than in the upper gastrointestinal tract and are more difficult to recognize. Nevertheless, the different fold patterns in the descending (Figs 3.16–3.18), transverse (Figs 3.23 and 3.24) and ascending (Fig. 3.26) regions of the colon are normally recognizable. The flexures are usually distinct and a bluish colouration may be seen through the bowel wall where it overlies the spleen (Figs 3.21–3.22) or liver. The caecum is typified by the confluence of the three taenia coli and the appendicular orifice (Figs 3.28–3.33), and the ileocaecal valve (Figs 3.34–3.40). These appearances confirm that the caecum has been reached.

It is sometimes stated that the only certain way to document total colonoscopy is to take a confirmatory biopsy from the terminal ileum (Figs 3.41–3.46).

Additional help in locating the tip of the instrument comes from the position of the light from the colonoscope transilluminating the abdominal wall (Fig. 3.27). Indentation of the colon can be seen through the colonoscope when the abdominal wall is digitally compressed, particularly over the caecum. Fluoroscopy may be employed to locate the colonoscope tip and to show the configuration of any loops which have formed. Screening is not essential but can be valuable during a colonoscopist's training period and in difficult cases. With modern colonoscopes it is however, rarely used. Electromagnetic imaging for monitoring the position of the colonoscope is under active development.

Detailed technical aspects of colonoscopy are beyond the scope of this Atlas but a few basic rules may be stated. Minimal insufflation of air or CO<sub>2</sub> should be used. The instrument should be kept as 'straight' (short) as possible avoiding the formation of loops. Repeated short insertions aid pleating ('concertina-ing') of the colon over the instrument. Aspiration of gas, especially at flexures, allows the colon to shorten over the instrument which may then advance spontaneously. Manual pressure on the abdomen by the assistant may prevent the formation of loops in the sigmoid or transverse colon and may greatly aid advancement beyond the splenic and hepatic flexures, respectively.

Familiarity with normal appearances and variations is essential for recognition of pathological processes. As in the upper gastrointestinal tract, mucosal biopsy is important for accurate pathological diagnosis. A peculiarity of the colon is its propensity to form polypoid lesions, especially adenomas. Mucosal biopsy is seldom adequate for accurate diagnosis, as small areas of adenocarcinoma may be missed on biopsy of an adenoma. Accurate diagnosis of polypoid lesions depends upon the histological examination of multiple sections of the whole polyp. For this reason familiarity with the technique of polypectomy (see pp. 327–335) is essential for the practice of colonoscopy.

The use of screening and surveillance colonoscopy is increasing though as yet there is little agreement on the indications, methods and frequency, for example in inflammatory bowel disease, following colonoscopic polypectomy, and the asymptomatic elderly. There is probably a significant role for flexible sigmoidoscopy in population screening programmes for colonic polyps, and it is possible that in future much of this work will be carried out by nurse endoscopists.

Significant advances have been made in the development of 'virtual colonoscopy', reconstruction of colonic images obtained from spiral CT, and of robots capable of surveying the colonic lumen. It is likely, however, to be several years before these new techniques are incorporated into standard clinical practice. In any case, gastroenterologists employing these techniques will need to have a detailed knowledge of colonic appearances in health and disease, and will continue to have to be expert colonoscopists for therapeutic purposes.

## Normal appearances

Poor bowel preparation

Normal mucosal appearances

Rectum

Sigmoid and descending colon

Splenic flexure

Transverse colon and hepatic flexure

Ascending colon

Caecum

Ileocaecal valve

Ileum

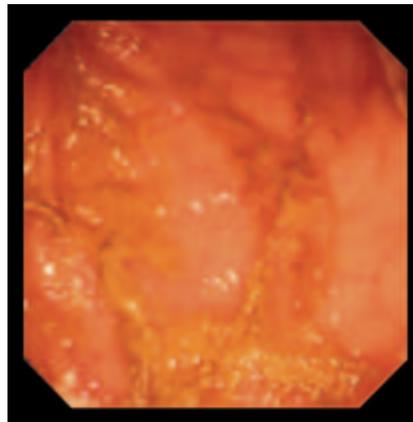
Inversion of colonoscope in rectum

Minor trauma during colonoscopy

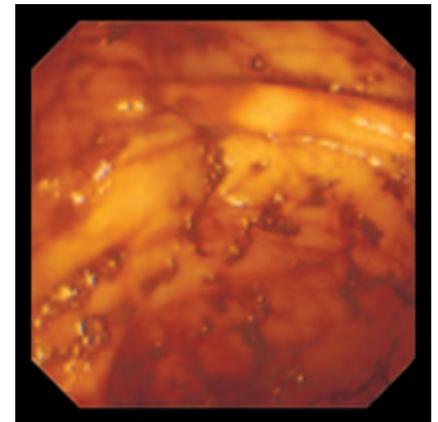
### Poor bowel preparation

When the bowel is poorly prepared there will be technical difficulties. Minor faecal remnants (Figs 3.1 and 3.2) may obscure mucosal detail, making it difficult to be certain whether abnormalities such as small polyps, aphthae or vascular lesions are present. A larger amount of faeces (Fig. 3.3) will totally obscure the view especially when the material is fluid (Fig. 3.4).

It is sometimes possible to wash away small amounts of faecal residue by injecting water either directly through the washing channel of the colonoscope or through a plastic tube passed through this channel; the latter method has the advantage that the washing jet can be more accurately aimed. Small hard faecal pellets can often be pushed to one side as the tip of the colonoscope is advanced. A mass of soft adherent faeces or the presence of fluid stools make it impossible to proceed. The washing facility of the colonoscope may not have the power to clean a lens covered by sticky material. Occasionally by manipulation of the tip, it is possible to clean the lens by sliding it against the colonic mucosa but more often withdrawal for cleaning and reinsertion is necessary.



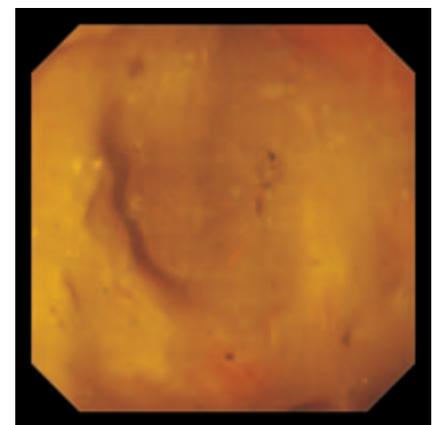
3.1



3.2



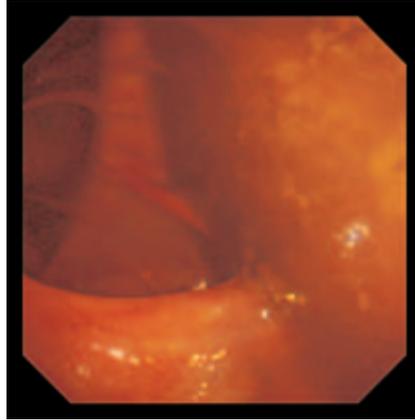
3.3



3.4

**Poor bowel preparation (cont.)**

When the right side of the colon is reached, it is not uncommon to find residual faeces. Liquid matter (Fig. 3.5) can be aspirated but if there is a large quantity of solid it may be preferable to withdraw and repeat the examination after improved preparation.

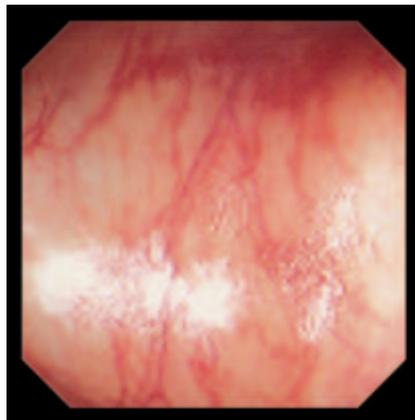


3.5

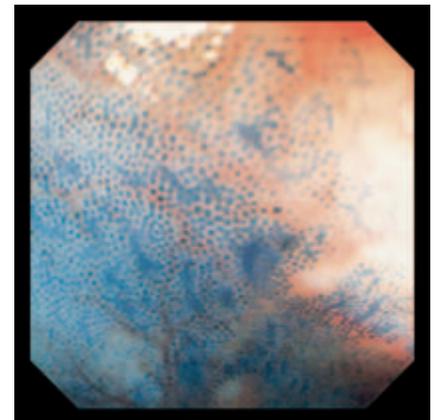
**Normal mucosal appearances**

The colonic mucosa appears pink and smooth with occasional minor irregularities. Sometimes there is the suggestion of a mosaic pattern which on the 'enhanced' video setting may be more pronounced (Fig. 3.6). This is caused by reflections from the mucosal crypts which are more apparent when dye spraying has been employed (Fig. 3.7).

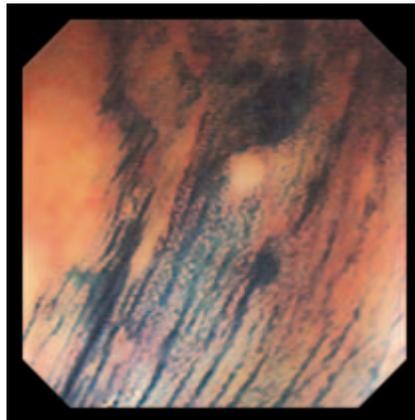
Figure 3.8, in addition to mucosal crypts, shows the innominate grooves and some lymphoid follicles.



3.6



3.7



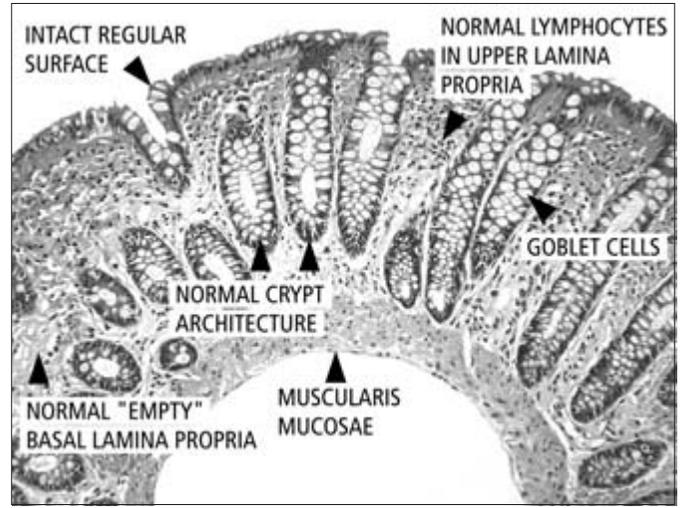
3.8

**Normal mucosal appearances (cont.)**

The histological appearances of normal large bowel mucosa, other than caecal and low rectal, are shown in Fig. 3.9.



3.9



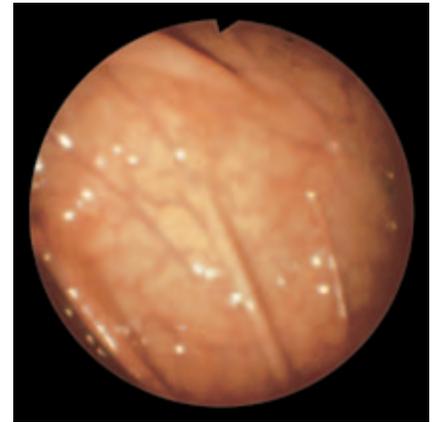
**Rectum**

The rectum, as its name suggests, may be relatively straight, but even the experienced colonoscopist may have a little difficulty in moving the tip of the instrument past the many large mucosal folds (Fig. 3.10). Folds are present throughout the large bowel and may persist even after full distension with air (Fig. 3.11).

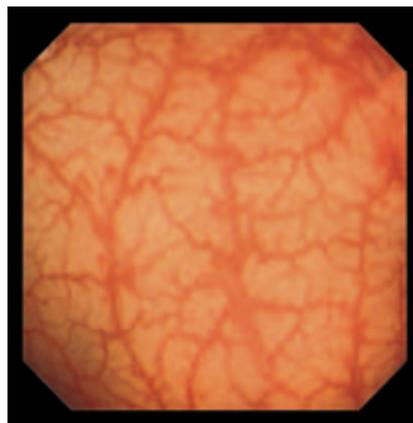
The vascular pattern is prominent in the rectum (Fig. 3.12) and varies according to the method of preparation used. Certain types of preparation may induce hyperaemia. It is not uncommon to see more than one layer of small vessels (Fig. 3.13). Submucosal arterioles and venules tend to fan out together.



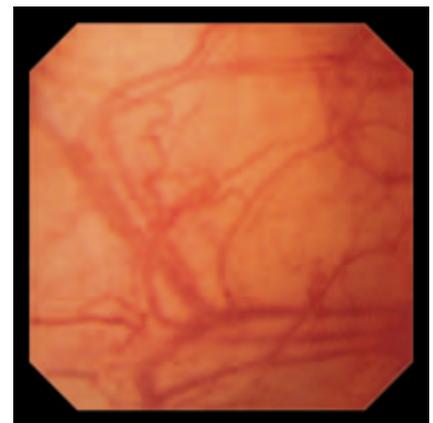
3.10



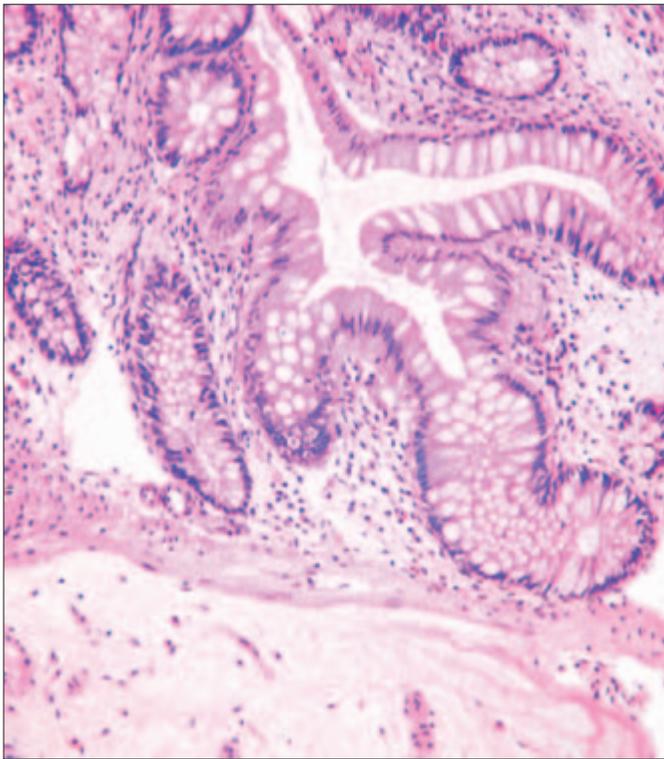
3.11



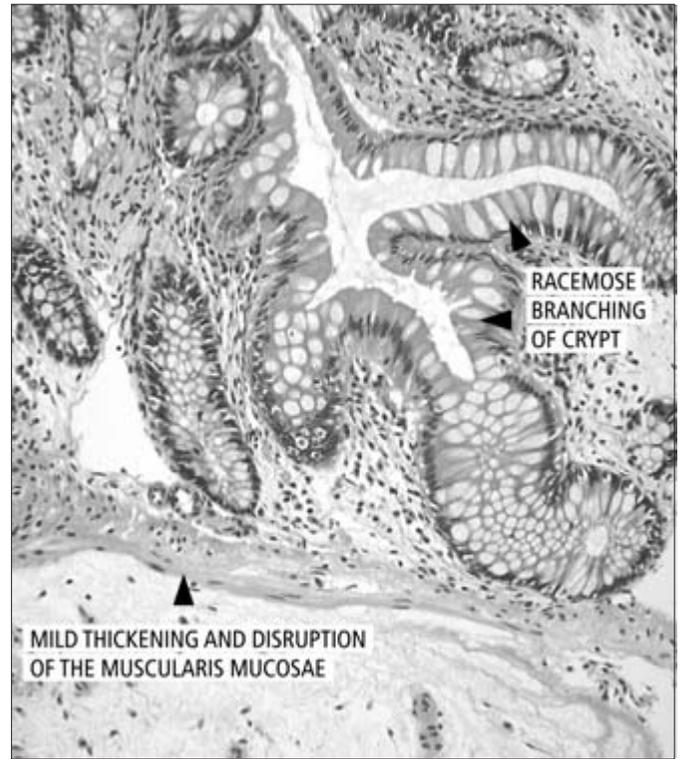
3.12



3.13



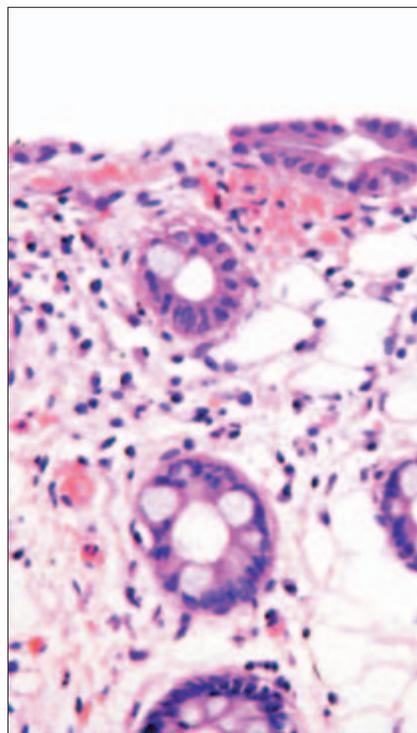
3.14



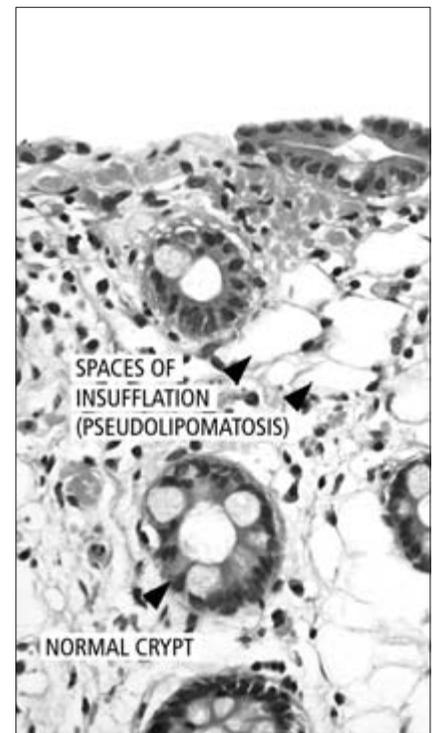
**Rectum (cont.)**

Histologically, low rectal biopsies from the normal rectum (Fig. 3.14) may have a degree of crypt distortion, basal lymphoid aggregates and muscularization of the lamina propria. To the unwary a low rectal biopsy may therefore simulate quiescent inflammatory bowel disease or mucosal prolapse.

Another warning to the unwary: in colonic biopsies in general and rectal biopsies in particular the histopathologist may find within the lamina propria spaces with no lining epithelium or surrounding cellular reaction. These appearances, described as pseudolipomatosis (Fig. 3.15) are thought to be an artefact related to air insufflation at colonoscopy and are of no clinical significance.



3.15



### Sigmoid and descending colon

Muscular activity creates prominent circular folds (Fig. 3.16) which may be diminished by anticholinergic drugs (Fig. 3.17).

The lumen in the sigmoid is often tortuous needing repeated changes of direction during insertion of the endoscope. Progress is made most satisfactorily by frequent short advances followed by withdrawal, so gathering mucosal folds on the instrument in a concertina-like fashion. It is in the region of the sigmoid and descending colon that loops commonly form during colonoscopy.

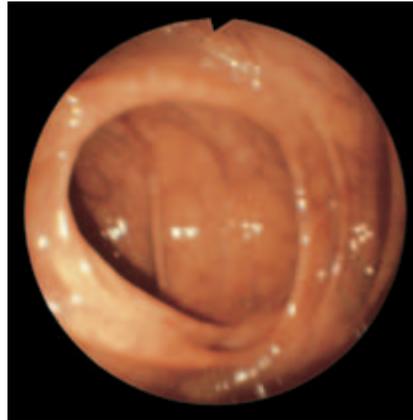
Figure 3.18 shows the descending colon. As in the sigmoid, encircling folds are seen throughout much of the descending colon which usually appears round. The lumen tends to straighten as the instrument is advanced.



3.16



3.17

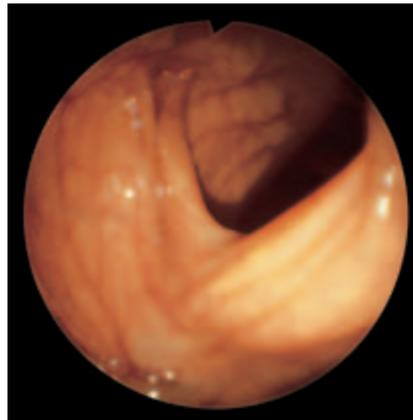


3.18

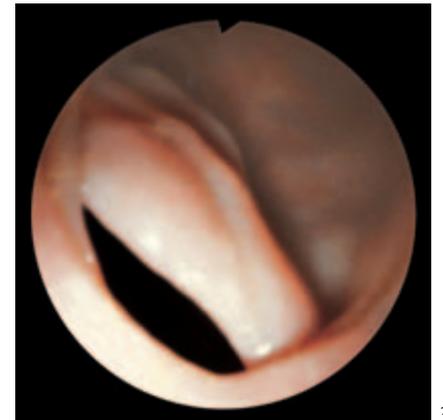
### Splenic flexure

A sudden change of direction is often noted at the splenic flexure (Fig. 3.19) and advancement of the colonoscope may be temporarily halted. A distinct fold is commonly seen just below the flexure (Fig. 3.20). A similar appearance may be sometimes found at the hepatic flexure or when there are redundant loops on the mesentery in the transverse or descending colon.

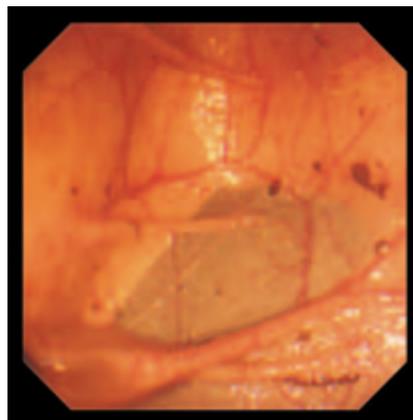
The spleen and liver when seen through the colonic wall present as a patch of blue colour, often with an obvious impression as the bowel wall is compressed (Figs 3.21 and 3.22). Although characteristic of the flexures this appearance may be seen elsewhere when a mobile loop is pressed against a solid viscus or a pathological mass.



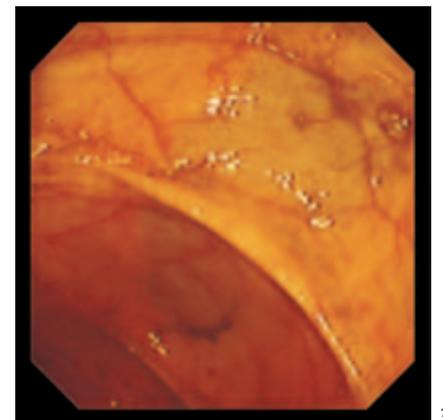
3.19



3.20



3.21



3.22

### Transverse colon and hepatic flexure

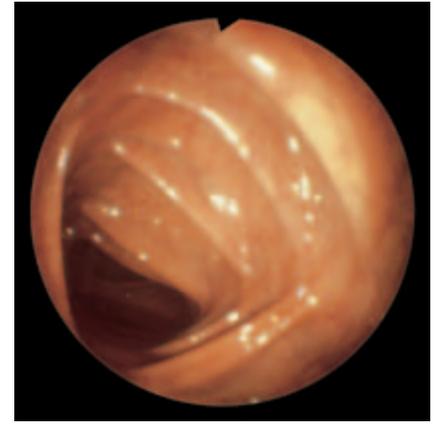
Haustra in the transverse colon (Figs 3.23 and 3.24) typically exhibit a distinct appearance described as ‘rounded triangles’, somewhat reminiscent of Toblerone® chocolate. This is caused by the taenia coli. This region is often the most typical and easily recognized section of the colon.

The blue colouration caused by the liver is often angular, with a distinct edge which can be seen to move with respiration. As a considerable length of colon may be in contact with the liver this appearance may be present over several centimetres.

The hepatic flexure has no specific features and is not always clearly identified. Advancement through this region may present technical problems, but often aspiration of air shortens the loop allowing the instrument tip to pass the flexure.



3.23

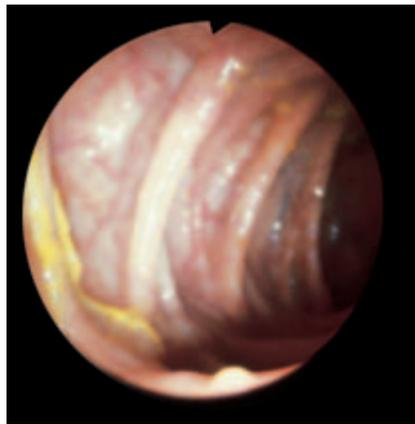


3.24

### Ascending colon

Here the pattern of the mucosal folds is again different; although well defined they do not encircle the lumen completely and may appear thicker than in the transverse colon (Figs 3.25 and 3.26).

Even in a well-prepared colon, there may be considerable fluid residue (Fig. 3.5) and some faecal material adherent to the mucosa. Often aspiration of the contents is needed to allow a satisfactory view of the caecum. Aspiration of air may aid the advancement of the colonoscope tip from the hepatic flexure down to the caecal pole.



3.25



3.26

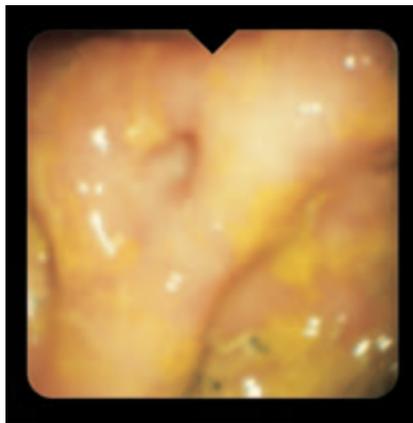
### Caecum

When the tip of the colonoscope is in the caecum, transillumination in the right iliac fossa is frequently seen (Fig. 3.27); these appearances are enhanced by darkening the room. Together with local indentation by a palpating finger, and recognition of the appendiceal orifice and ileocaecal valve, this confirms that total colonoscopy has been achieved. At this stage the distance marking on the colonoscope shaft shows about 70 cm when redundant loops have been straightened, but if the endoscope is looped, especially in the sigmoid, a greater insertion measurement will be seen.

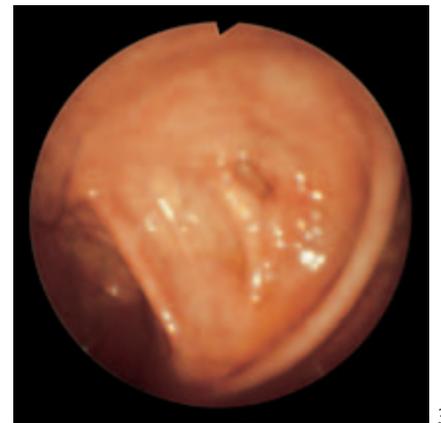
Figure 3.28 shows the caecal pole marked by the confluence of the three taeniae coli and the appendiceal orifice. Sometimes this confluence is less apparent (Fig. 3.29); in this case, the appendiceal orifice is more slit-like. It may be more circular (Fig. 3.30) and may even reveal the lumen of the appendix (Fig. 3.31). Rarely it assumes a piled up or 'Chelsea bun' appearance (Fig. 3.32).



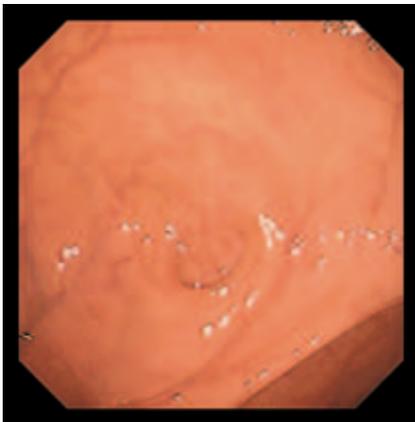
3.27



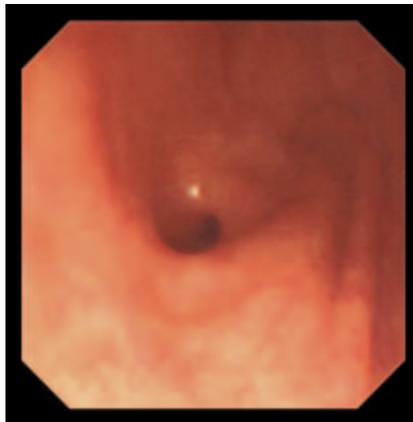
3.28



3.29



3.30



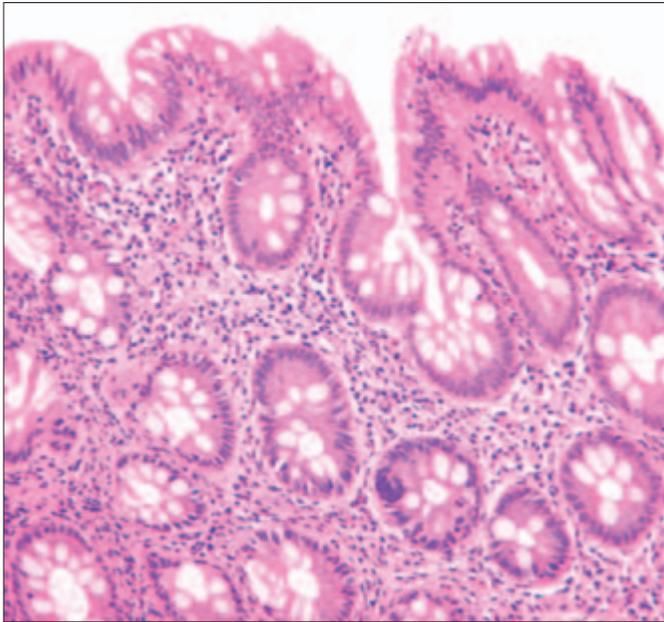
3.31



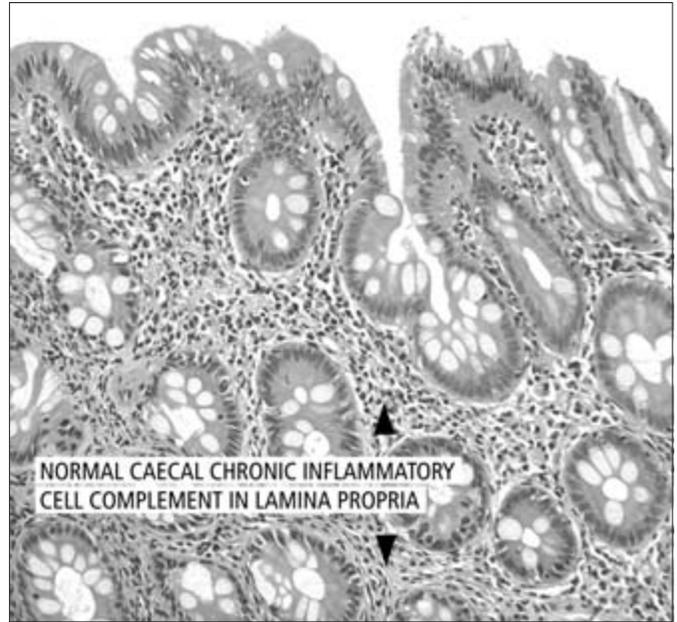
3.32

### Caecum (cont.)

The normal caecal mucosa (Fig. 3.33) contains considerably more chronic inflammatory cells than any other site in the normal large bowel. Here mononuclear cells occupy the full thickness of the lamina propria, and may lead to an erroneous histological interpretation; in particular a wrong assessment of the extent of ulcerative colitis or the over-diagnosis of microscopic colitis may result.



3.33



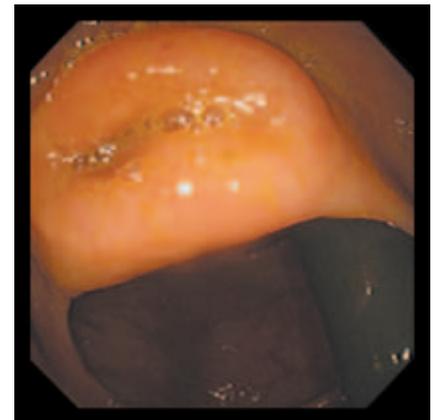
### Ileocaecal valve

Some colonoscopists assert that total colonoscopy cannot claim to have been achieved if the terminal ileum has not been entered. This presupposes that the ileocaecal valve has been found and correctly identified.

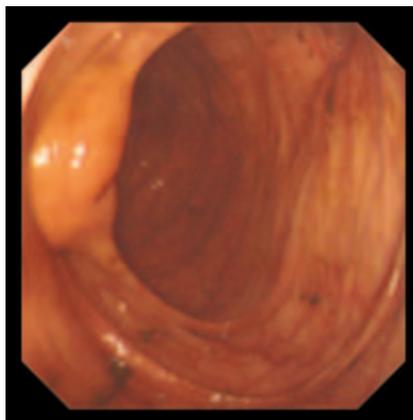
It is located on the medial wall of the caecum several centimetres above its pole, and is commonly hidden below a prominent fold (Fig. 3.34). On occasions it reveals itself when fluid or froth are expelled from the terminal ileum (Fig. 3.35). Its appearances are variable and may change during observation. It may appear as an ovoid swelling (Fig. 3.36), with puckering converging onto the opening (Fig. 3.37), or as an oval opening with a smooth regular outline (Fig. 3.38).



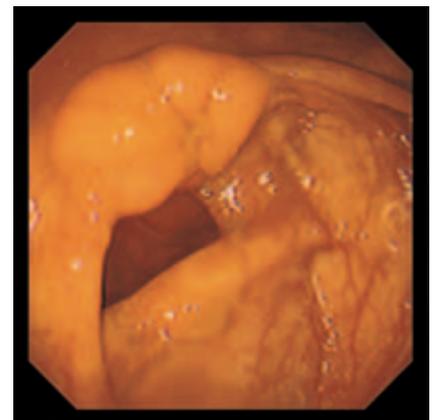
3.34



3.35



3.36



3.37

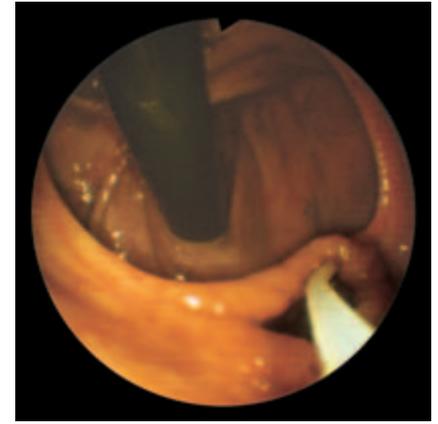
### Ileocaecal valve (*cont.*)

To gain access to the terminal ileum, especially if the valve is hidden below the fold, it is best to advance the colonoscope beyond the valve or this fold. The tip is then angulated towards the valve and the shaft gently withdrawn. When the valve is clearly seen, the tip is straightened and readvanced. Several attempts may be required to execute this manoeuvre successfully.

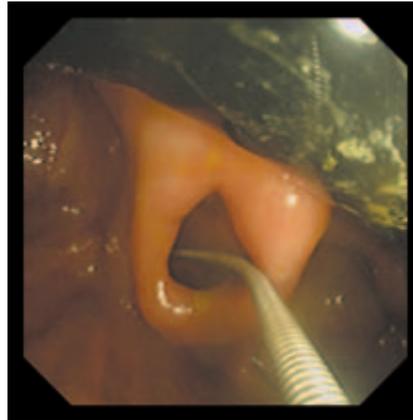
If entry is difficult the colonoscopist may explore the valve with a plastic washing tube (Fig. 3.39) or closed biopsy forceps (Fig. 3.40) using this as a 'guide wire' for the colonoscope.



3.38



3.39



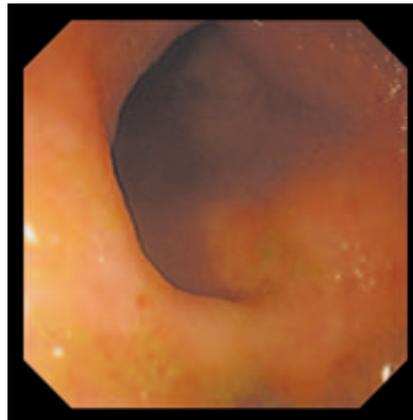
3.40

### Ileum

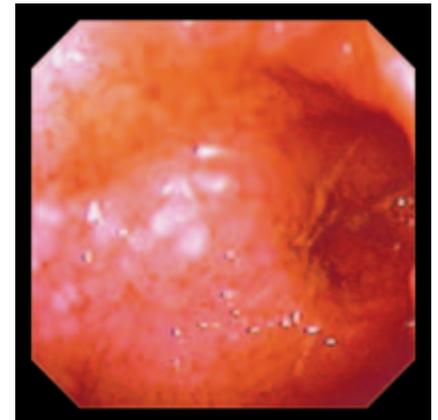
The ileum is best surveyed by enteroscopy (Chapter 5). At colonoscopy it is not usually possible to visualize more than the lowest portion of the terminal ileum.

The mucosa (Fig. 3.41) is not as translucent as that of the colon. It may be faintly granular in appearance. It is unusual to obtain good views of the villous pattern though this is sometimes possible during routine colonoscopy (Fig. 3.42). Dye spraying will enhance these appearances (Fig. 3.43).

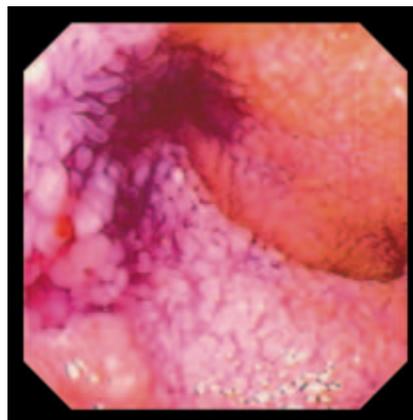
With suitable positioning it is possible to instil water through the biopsy channel into the part of the bowel under review. This causes distension of the lumen and allows underwater inspection and photography. In Fig. 3.44 the normal villous pattern of the terminal ileum is demonstrated much more clearly than by standard techniques.



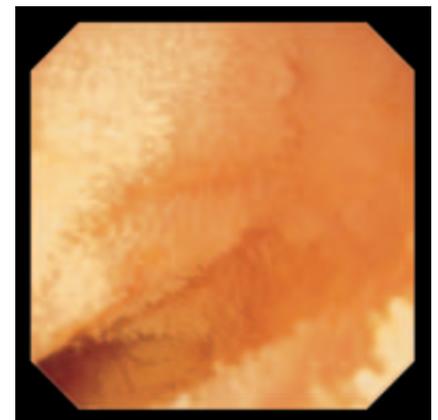
3.41



3.42



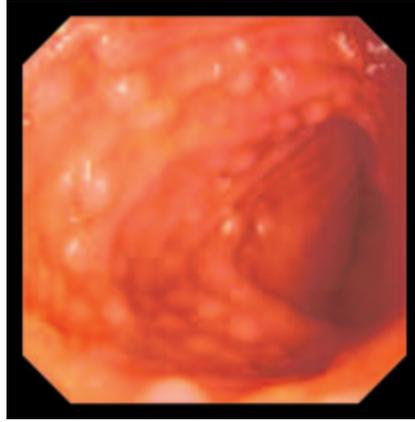
3.43



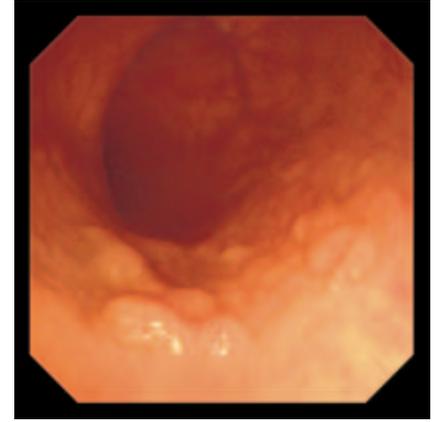
3.44

**Ileum (cont.)**

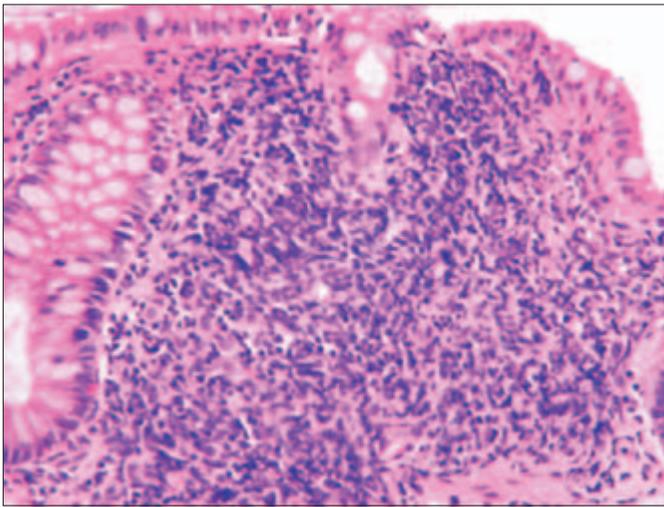
Prominent lymphoid follicles are commonly seen in the terminal ileum in children. Figure 3.45 illustrates the appearances in a 6–7-year-old boy. A Peyer’s patch in the terminal ileum is shown in Fig. 3.46. The histological appearances of biopsies from a lymphoid follicle and a Peyer’s patch appear in Figs 3.47 and 3.48.



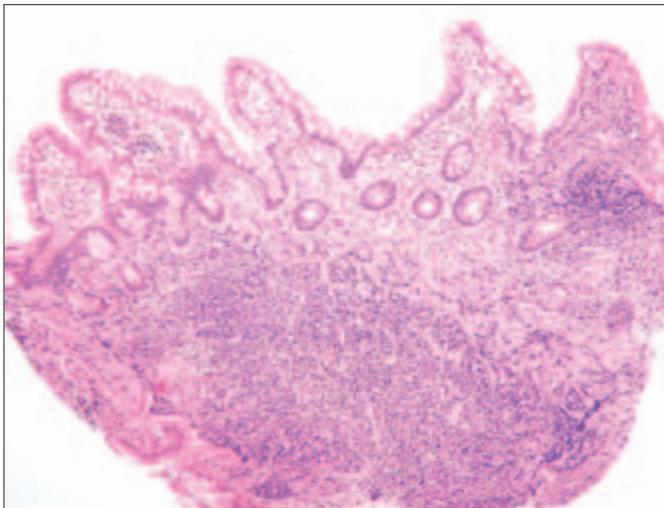
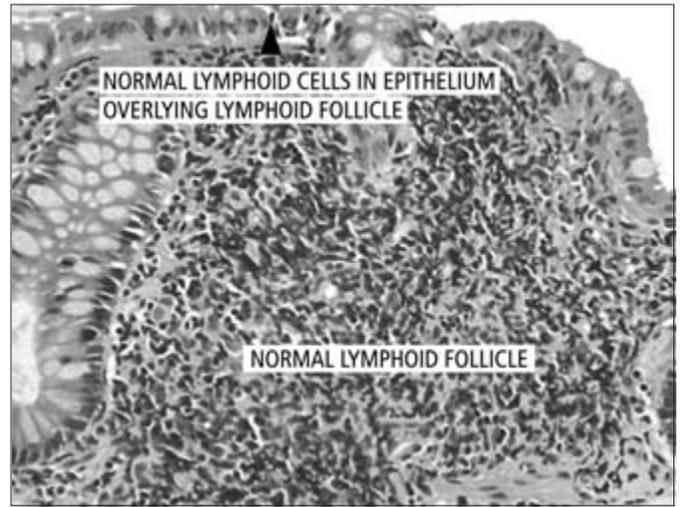
3.45



3.46



3.47



3.48



### Inversion of colonoscope in rectum

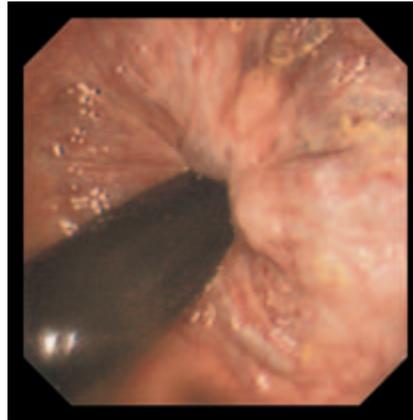
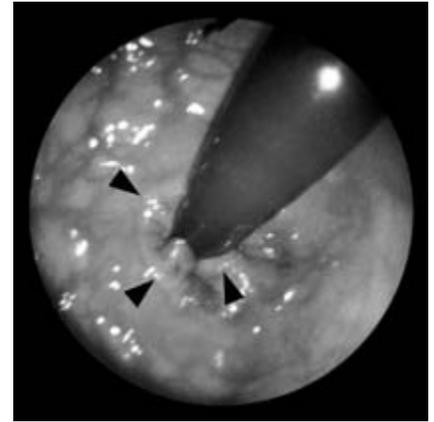
Distensibility of the normal rectum permits retroflexion of the instrument tip which allows the anal margin to be viewed from within. The dentate line and anal papillae can be seen (Fig. 3.49).

Internal haemorrhoids are well shown if present (Fig. 3.50), though more dramatic views can be obtained from below especially if the colonoscope or flexible sigmoidoscope is passed through a prepositioned rigid proctoscope (Fig. 3.51).

Caution must be exercised when attempting inversion in a diseased rectum. It is however, useful in cases of unexplained rectal bleeding. Inversion can be attempted, with due care, in any part of the colon. It may be especially useful during various therapeutic procedures such as in best placement of a snare for polypectomy.



3.49



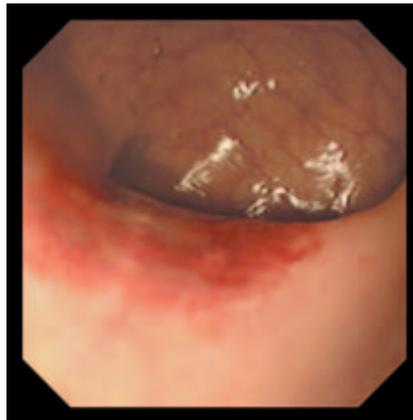
3.50



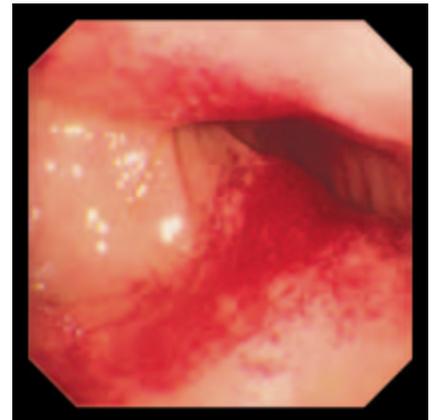
3.51

### Minor trauma during colonoscopy

It is common for minor mucosal trauma to occur during the procedure, varying from small ecchymoses, especially on the crests of folds (Fig. 3.52), to longer streaks, here seen at the splenic flexure (Fig. 3.53). Such minor trauma can reasonably be listed under 'normal appearances' and is of little importance. Major trauma is discussed under Complications (pp. 341–342).



3.52



3.53

## Abnormal appearances

**Blood 240**

**Diverticula, diverticulosis and diverticulitis 241**

**Benign and malignant tumours 244**

**Inflammatory bowel disease 270**

**Infections and infestations 290**

**Vascular abnormalities 296**

**Bleeding 301**

**Miscellaneous conditions 301**

### Blood

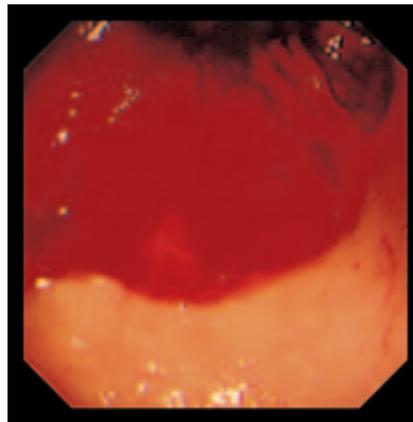
#### Blood

The need for adequate preparation for colonoscopy has already been stressed; faecal remnants can make examination difficult if not impossible. Colonoscopy in the presence of active or recent bleeding may be equally unrewarding.

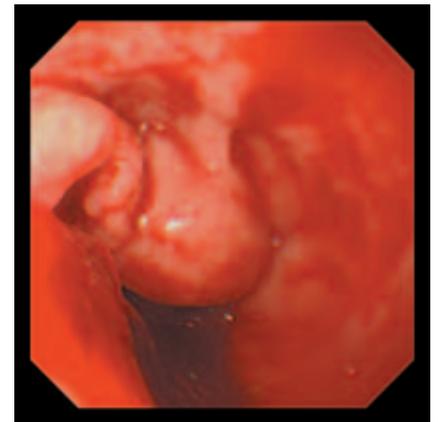
Figure 3.54 shows normal colonic mucosa, and a sharply defined lower margin of blood covered mucosa where detail is obscured. In Fig. 3.55 the mucosa is grossly abnormal and clearly irregular but again no details can be seen.

The casual reader may be surprised by the appearances shown in Fig. 3.56 and at first glance may think that the printer is at fault, or that this image should have been omitted. This is not so. When so much blood is present, light will be absorbed such that the endoscopist will have virtually no view.

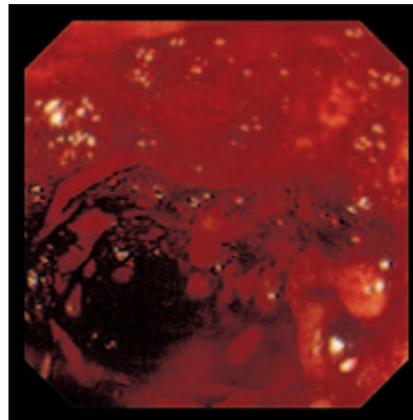
Active rectal bleeding or a history of recent severe bleeding are relative contraindications to colonoscopy, and it is advised where possible that time should be allowed for evacuation of clot before examination is attempted.



3.54



3.55



3.56

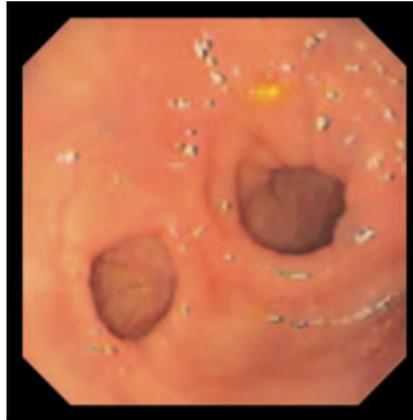
## Diverticula, diverticulosis and diverticulitis

### Diverticula, diverticulosis and diverticulitis

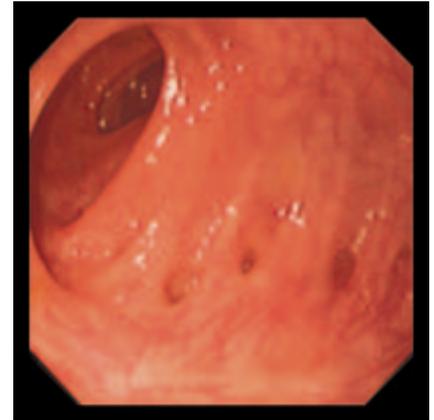
Diverticula of the large bowel are common and are found more frequently with advancing age. They may be single or multiple and, although occurring anywhere in the colon, are found most commonly in the sigmoid. They are usually asymptomatic and an incidental finding when the patient is under investigation for another condition. The endoscopist must always differentiate between the opening of a diverticulum and the lumen of the bowel: diverticula are thin-walled and may be perforated.

Figure 3.57 shows a single large upper rectal diverticulum, an uncommon condition, and the rectal lumen. In Fig. 3.58 there are multiple lower sigmoid diverticula. Some of those in Fig. 3.59 contain faecal pellets. In contrast to the appearances shown in Fig. 3.58 the diverticula in Fig. 3.60 are separated by thickened folds which represent the circular muscle hypertrophy of diverticular disease.

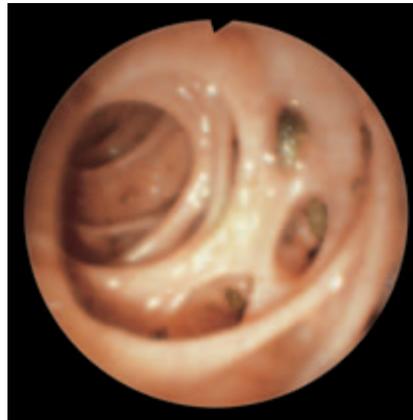
The diverticulum shown in Fig. 3.61 is filled with faeces. When such a pellet is removed, a clean deep diverticulum will be revealed (Fig. 3.62).



3.57



3.58



3.59



3.60



3.61



3.62

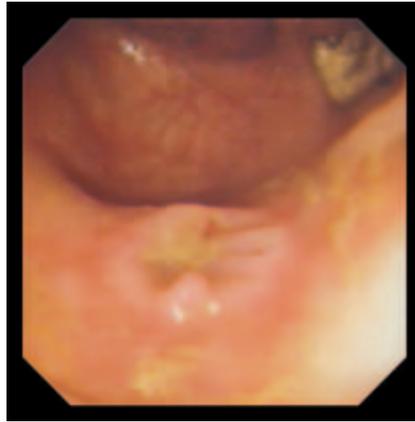
**Diverticula, diverticulosis and diverticulitis (cont.)**

Occasionally, when intracolonic pressure is reduced by aspiration of air, a diverticulum may collapse (Fig. 3.63) or even invert, resembling a sessile smooth polyp.

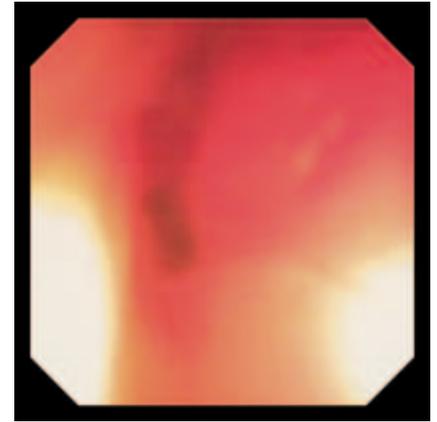
In severe diverticular disease it may be difficult to trace the colonic lumen due to muscular barring. When the tip of the colonoscope is pressed against the lumen the view becomes fuzzy (Fig. 3.64) and it may be hazardous to proceed. Water may be introduced to distend the colon, allowing clearer views and facilitating advancement of the instrument tip (Fig. 3.65).

Figure 3.66 shows severe diverticulitis. Colonoscopy is usually contraindicated in acute diverticulitis but occasionally the condition is encountered inadvertently. The lumen is narrowed or distorted by one or more inflamed, bulging masses, the surfaces of which are friable and often coated with mucopus. The mucosa may show nonspecific acute inflammatory changes for several centimetres on either side of the actual diverticular abscess.

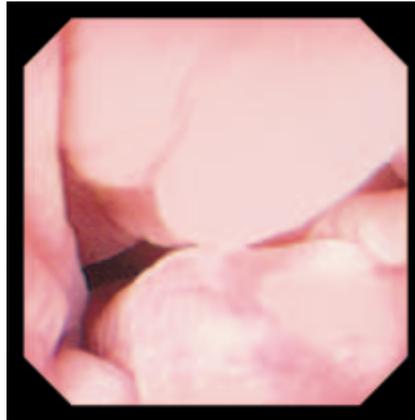
Figure 3.67 illustrates a milder form with hyperaemic changes confined to the orifices of several diverticula and in Fig. 3.68 inflammatory changes round one orifice. In Fig. 3.69 there is no surrounding erythema but pus can be seen issuing from a diverticulum.



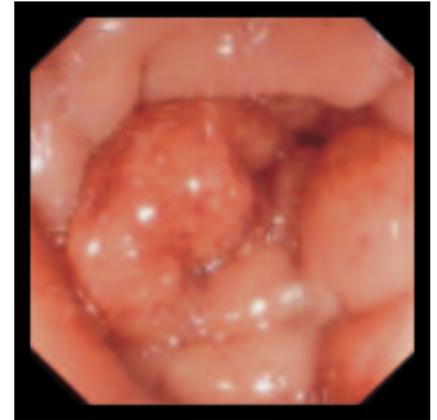
3.63



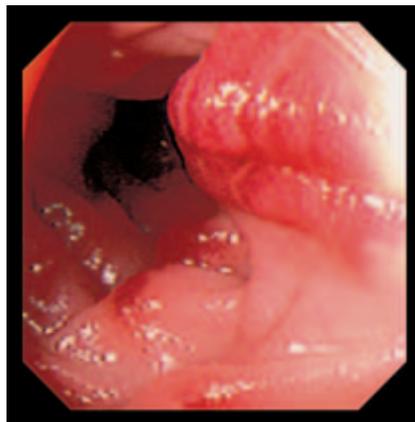
3.64



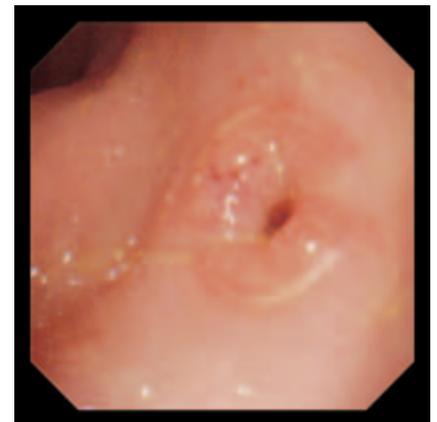
3.65



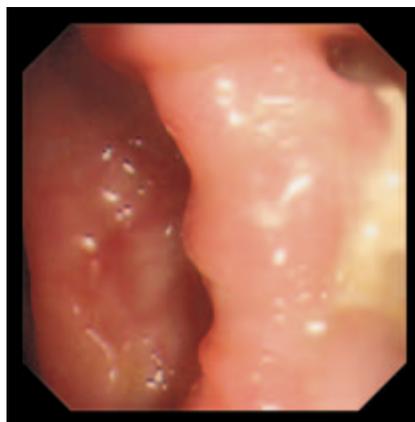
3.66



3.67



3.68

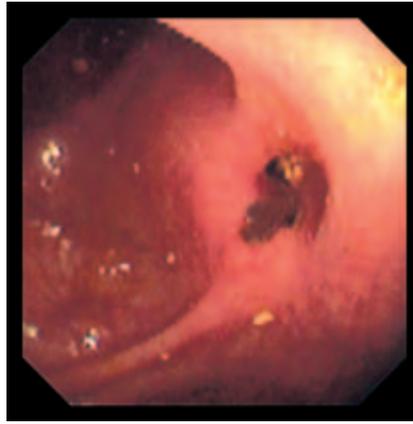


3.69

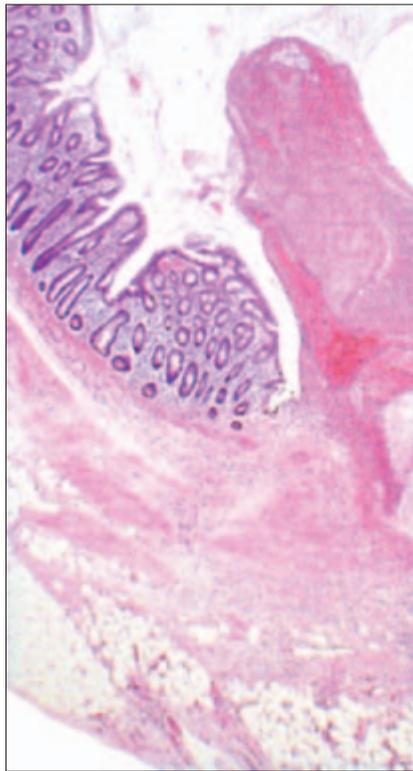
**Diverticula, diverticulosis and diverticulitis (cont.)**

The 'colitis' associated with diverticular disease may be diffuse but often causes a characteristic appearance of crescentic erythema on the mucosal folds adjacent to the diverticula: 'crescentic fold disease' or 'crescentic colitis'. Biopsies from these areas may present difficulties for the histopathologist as the appearances can mimic ulcerative colitis, Crohn's disease, mucosal prolapse and microscopic colitis.

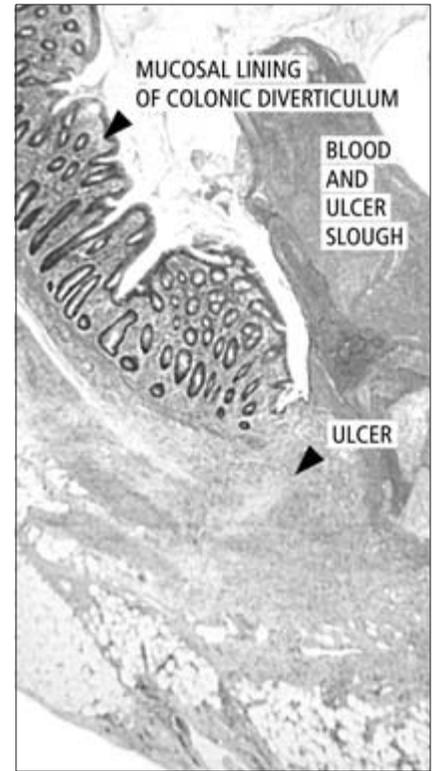
The diverticulum shown in Fig. 3.70 contains blood clot. In patients with diverticular disease it is important to ascertain whether bleeding is actually coming from the diverticular segment, as blood from above commonly accumulates within diverticula often leading to misdiagnosis. The histological appearances of a bleeding diverticulum are shown in Fig. 3.71.



3.70



3.71



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## **Benign and malignant tumours**

### **Classification**

#### **Nonspecific endoscopic appearances**

#### **Lesions with polyp-like appearances**

#### **Adenomas**

#### **Familial adenomatous polyposis**

#### **Dysplasia-associated lesions or masses (DALM)**

#### **Hyperplastic (metaplastic) polyps**

#### **Juvenile polyps**

#### **Peutz–Jeghers polyps**

#### **Cronkhite–Canada polyps**

#### **Inflammatory polyps**

#### **Adenocarcinoma**

#### **Kaposi's sarcoma**

#### **Secondary and direct spread of tumours**

#### **Lymphoma**

#### **Gastrointestinal stromal tumours (GISTs)**

#### **Neuroendocrine tumours**

#### **Lipomas**

The identification and management of polyps and cancers is a common problem for the colonoscopist. The term 'polyp' is a macroscopic description of an elevated localized lesion which may be almost flat, sessile or pedunculated (stalked). Polyps most often consist of tissue of epithelial origin although submucosal lesions can protrude into the bowel lumen and may even become pedunculated whilst still covered with normal mucosa.

Up to 25% of polyps of significant size may be missed if reliance is placed on radiological rather than endoscopic techniques, but even colonoscopy will fail to pick up all the polyps all of the time. However, on current evidence, colonoscopy is the investigation of choice when polyps enter the differential diagnosis.

It is not possible to determine the pathological character of a polyp with any degree of accuracy from the endoscopic appearances alone; histological examination is essential. Superficial mucosal biopsy is inadequate as cell type may vary within a polyp and because normal mucosa may be adjacent to or overlie neoplastic or mesodermal tissue. To overcome this problem and provide adequate material for histological interpretation, the whole polyp must be removed if possible and retrieved for examination. There are well established techniques for this purpose. Polypectomy is described on pp. 327–335.

Polyps may be present anywhere in the colon but are most frequent on the left side. They may be single or multiple scattered tumours, or may occur in the form of polyposis, that is when there are more than 100 polyps in the colon. The term 'polyposis' is descriptive only, and does not imply polyp type or malignant potential.

Many polyps are symptomless and are discovered incidentally during endoscopy or barium enema radiology, or during screening or surveillance. Occult bleeding is common. Overt bleeding may occur when the surface of a polyp ulcerates or when torsion occurs with autoamputation and haemorrhage from the stalk. Large polyps

may intussuscept or prolapse through the anus. Slow or intermittent blood loss may lead to iron-deficiency anaemia. Screening programmes for the presence of polyps or carcinoma, e.g. those based on faecal occult blood testing, or a once-only flexible sigmoidoscopy, after the age of 50 years, are under evaluation.

It is currently accepted that there is a sequence progressing from a local benign adenomatous cellular proliferation to sessile or pedunculated adenomatous polyp and culminating in invasive carcinoma, the so-called adenoma-carcinoma or polyp-cancer sequence. Malignant change probably starts in the polyp head with local spread and subsequent invasion down the stalk and into the bowel wall before transmural, local lymphatic spread and metastasis occur. It is in anticipation of preventing this progression that resection of adenomatous polyps is performed as a means of cancer prevention. There is however, some uncertainty as to the rate of growth of polyps. It is accepted that certain types of polyp, for example metaplastic polyps, grow little if at all whilst others such as adenomas usually grow slowly, with a proportionate increase in the likelihood of malignant change.

Resected polyps must be examined histologically in their entirety with particular attention to possible malignant change. If tumour involves the excision line or is within 2 mm of this line, or has 'bad' histological prognostic features, further polypectomy, or surgical resection to assess lymph node status may be indicated. 'Bad' features include high Haggitt level of invasion (see Fig. 3.110), poor differentiation, and vascular invasion.

### Classification

It is essential to determine the pathological nature of a tumour and to know its likely natural history in order to provide appropriate management. Tables 3.1 and 3.2 show a classification of benign and malignant tumours of the large intestine. These tables form the basis of the remainder of this section.

**Table 3.1** Benign and malignant epithelial tumours of the large bowel (adapted from Day *et al.*, 1990)

Benign epithelial tumours	Malignant epithelial tumours
Adenoma	Adenocarcinoma
Adenomas	Other primary malignant tumours
Familial adenomatous polyposis	Secondary and direct spread of tumours
Dysplasia-associated lesions or masses (DALM)	
Hyperplastic (metaplastic) polyps	
Juvenile polyposis syndrome	
Peutz-Jeghers syndrome	
Cronkhite-Canada syndrome	
Cowden's syndrome	
Inflammatory polyps	
Others	

**Table 3.2** Non-epithelial tumours of the large bowel (adapted from Day *et al.*, 1990)

<b>Tumours of lymphatic tissue</b>
Benign lymphatic lesions
Lymphoma
Leukaemia
<b>Gastrointestinal stromal tumours (GISTs)</b>
<b>Vascular tumours</b>
<b>Neuroendocrine tumours</b>
<b>Lipomas</b>
<b>Others</b>

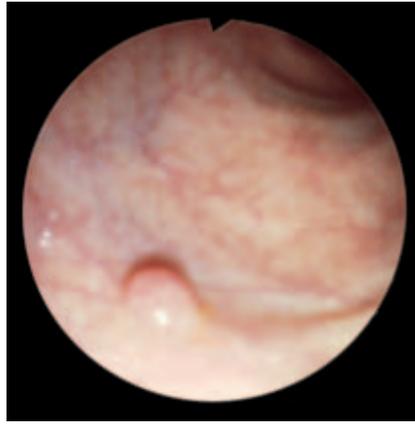
### Nonspecific endoscopic appearances

Small sessile adenomas and hyperplastic (metaplastic) polyps cannot reliably be distinguished visually. The lesions in Figs 3.72 and 3.73 were adenomatous polyps while Fig. 3.74 illustrates a metaplastic polyp. That shown in Fig. 3.75 without specific features is an inflammatory polyp.

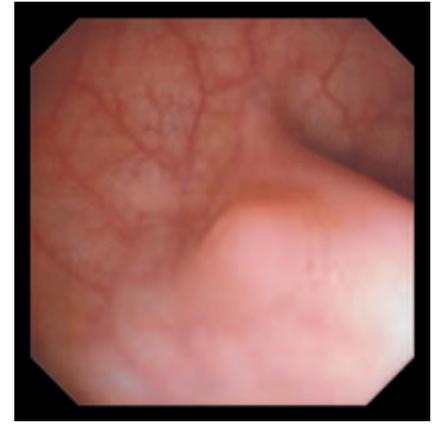
The small polyp in the centre of Fig. 3.76 is difficult to see. This demonstrates the need for good preparation to clear faecal residue before colonoscopy. Even when larger, the difficulty of presumptive diagnosis remains. The sessile polyp 1.5 cm in diameter (Fig. 3.77) is hyperplastic while that shown in Fig. 3.78 is adenomatous.

Occasionally a small polyp may resemble an anal tag; Fig. 3.79 shows a small low rectal abnormality which on biopsy was seen to be an adenomatous polyp.

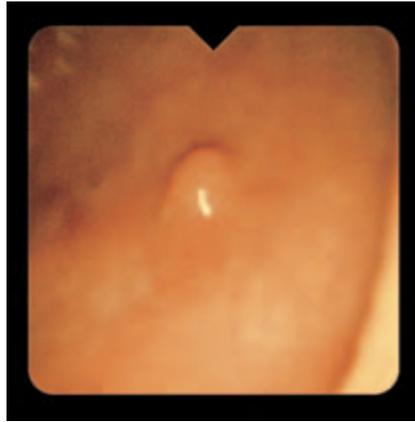
Polypectomy, retrieval and histological evaluation are essential for accurate diagnosis and satisfactory treatment. Small polyps less than 5 mm in diameter may be left *in situ*, but kept under surveillance.



3.72



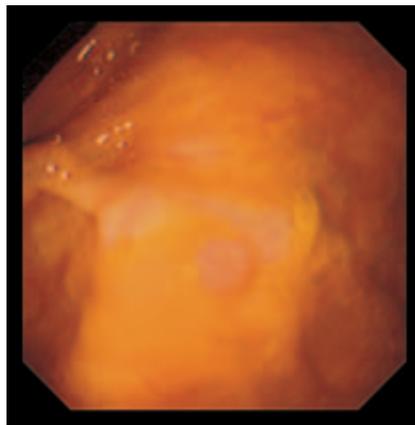
3.73



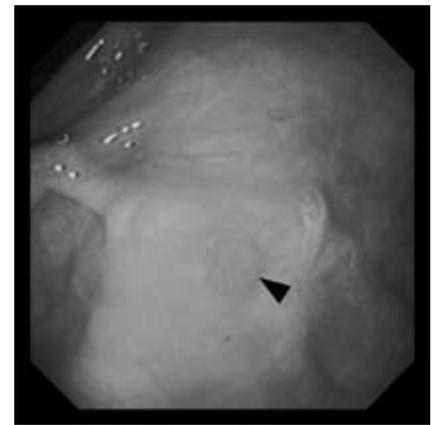
3.74



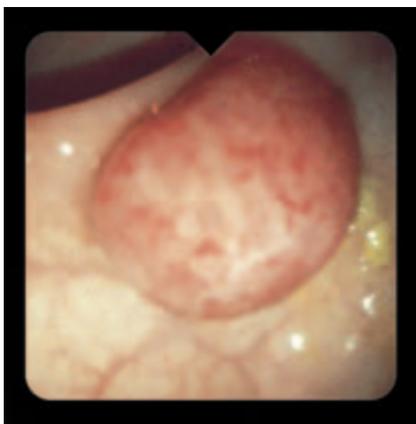
3.75



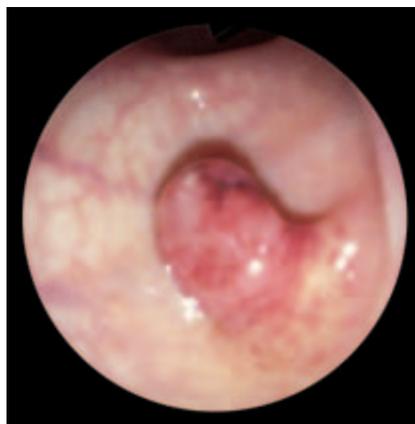
3.76



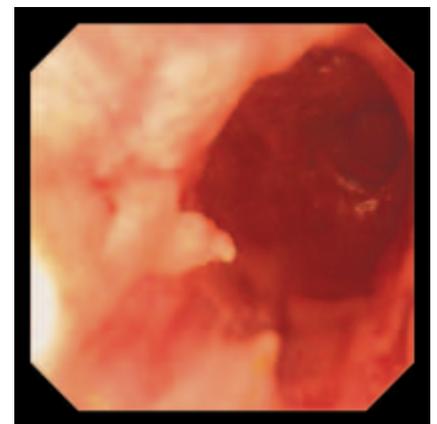
3.79



3.77



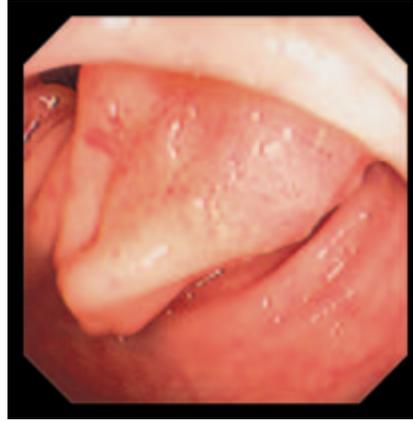
3.78



3.79

### Lesions with polyp-like appearances

It is not unusual for the endoscopist to be misled by the appearances of normal haustra: folds in the normal colonic mucosa may simulate polyps, especially when insufflation is suboptimal. Occasionally, a normal mucosal fold may assume an unusual form (Fig. 3.80) and this can be misleading, as can other normal structures, such as an ileo-caecal valve (Fig. 3.36) or an inverted diverticulum (Fig. 3.63), and many abnormal structures, for example lesions associated with bilharzia (Figs 3.272 and 3.273) or ureteric transplantation (Fig. 3.399).

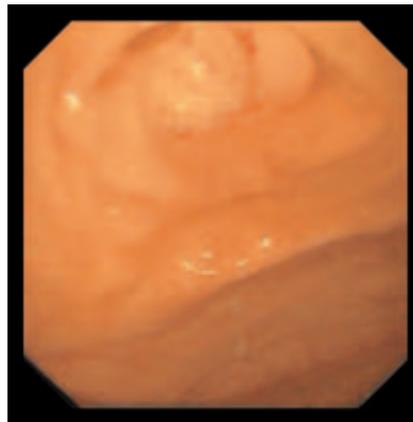


3.80

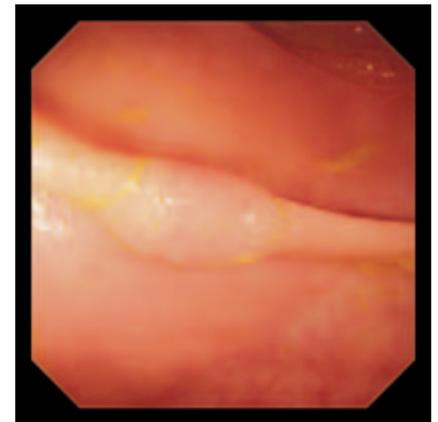
### Adenomas

Adenomas are classified according to their histological appearances (Figs 3.103–3.107) as tubular (approximately 50%), tubulovillous (approximately 40%) and villous (approximately 10%). Rarer morphological types of adenomas include serrated and flat. Tubular and tubulovillous adenomas are most likely to be stalked or pedunculated, while villous adenomas are often sessile. Adenomas may occur anywhere in the colon though they are commoner on the left. Figure 3.81 shows a rare site, an adenoma of the appendix.

A flat adenoma (Fig. 3.85) has been defined as less than twice the thickness of the normal mucosa, or less than 1.3 mm thick. The problem with flat adenomas lies in their recognition. A smooth almost flat adenoma is easier to spot on a mucosal fold (Fig. 3.82) or just beyond (Fig. 3.83).



3.81



3.82



3.83

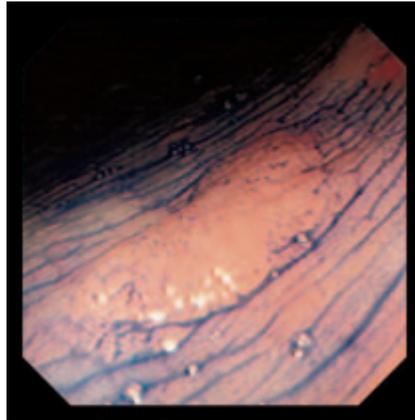
**Adenomas (cont.)**

Such lesions are most easily seen when thrown into relief either by peristalsis, or by changing the contour slightly with the closed biopsy forceps (Fig. 3.84). Dye spraying will display flat and very small adenomas well. Figure 3.85, obtained in a patient with familial adenomatous polyposis, shows a small flat and several very small polyps.

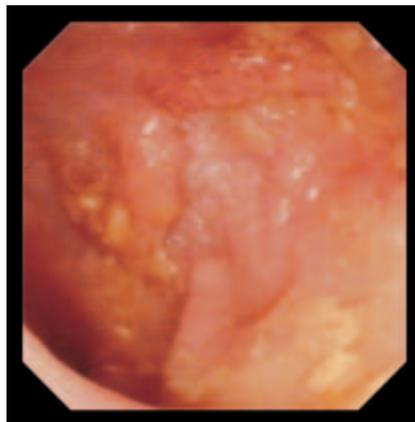
A sessile adenoma normally has no specific distinguishing features (Fig. 3.86). A pedunculated polyp has a smooth stalk of varying length, covered with normal colonic mucosa (Fig. 3.87) and therefore of the same colour as the surrounding colon. There is normally a sharp line of demarcation between stalk and head, which is made up of adenomatous tissue and is often darker than normal mucosa. Classically the outline of the head is regular and smooth, or slightly irregular, with a pattern reminiscent of the cerebral surface or brain coral (Figs 3.87 and 3.88).



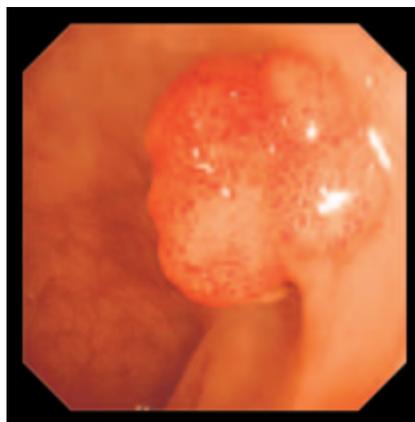
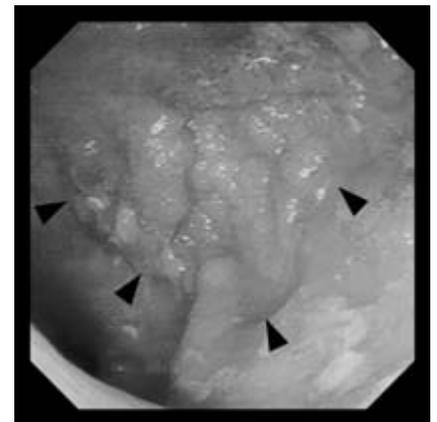
3.84



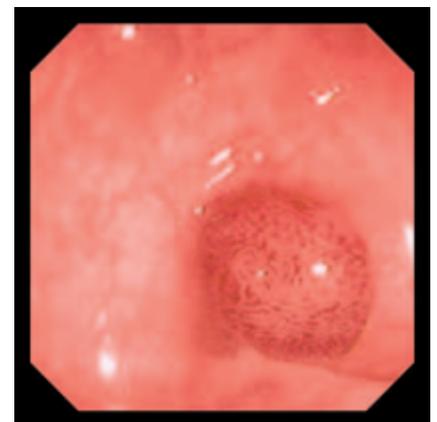
3.85



3.86



3.87



3.88

**Adenomas (cont.)**

This appearance is less well marked in the polyp shown in Fig. 3.89 which however, has many of the typical features already described. Less commonly the stalk is relatively thinner (Fig. 3.90) or the head irregular (Figs 3.91 and 3.92) or patchily discoloured (Fig. 3.93).

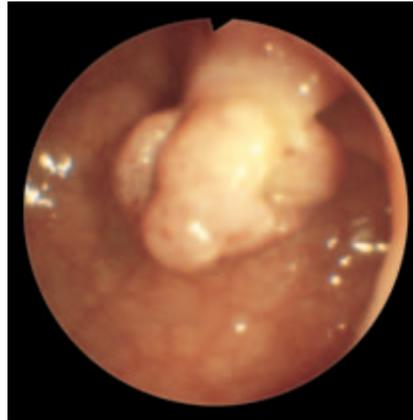
At first sight a stalked polyp may appear sessile (Fig. 3.94) but a stalk is revealed by altering the positioning of the colonoscope (Fig. 3.95) or by lifting the polyp with forceps or a snare (Figs 3.96 and 3.97).



3.89



3.90



3.91



3.92



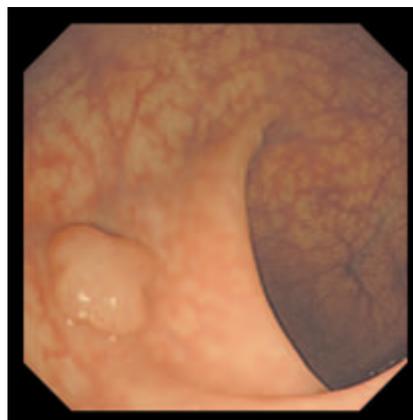
3.93



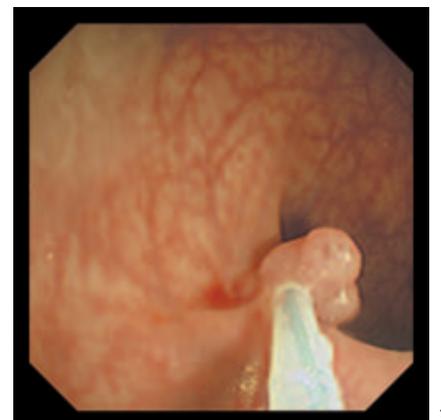
3.94



3.95



3.96



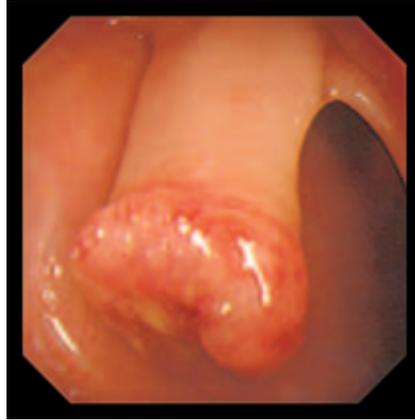
3.97

**Adenomas (cont.)**

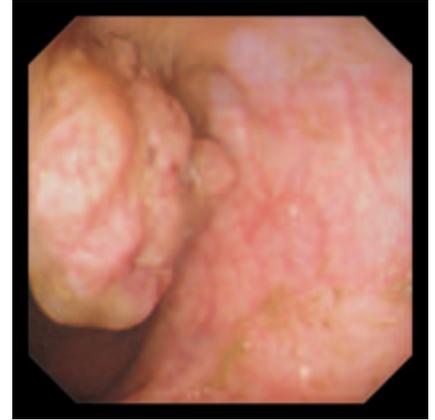
When an adenoma arises in a patient with melanosis coli the stalk will be pigmented but the head, being neoplastic, will be pink (Fig. 3.362).

The head of the stalked adenomatous polyp shown in Fig. 3.98 is regular in outline except at its apex: this notched appearance may be seen in partial infarction, or may indicate stromal tethering by focal invasive adenocarcinoma.

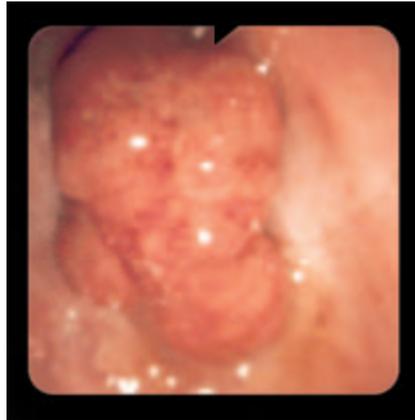
Breadth of base in sessile adenomas varies (Figs 3.99 and 3.100). The broad based polyp shown in Fig. 3.101 is a typical villous adenoma, a diagnosis confirmed after polypectomy. Villous adenomas in close up (Fig. 3.102) often have a tufted appearance.



3.98



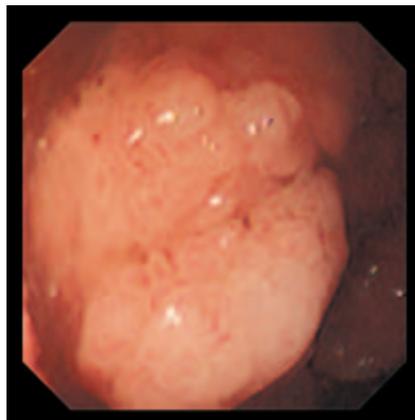
3.99



3.100



3.101



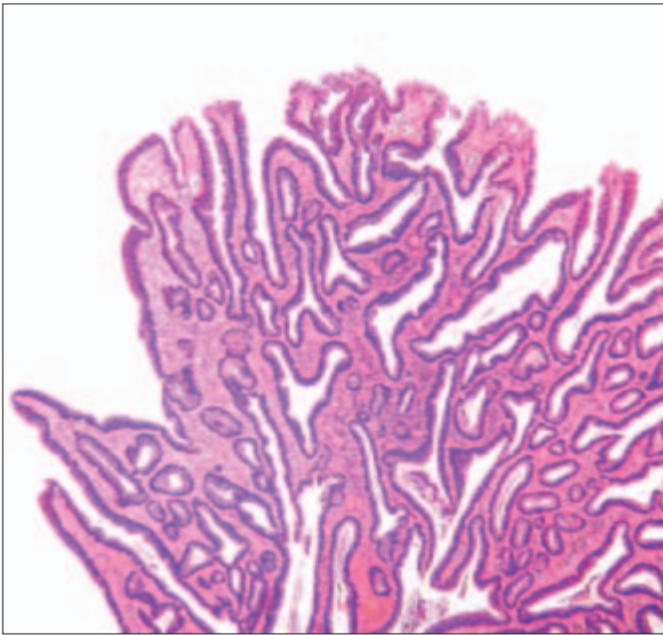
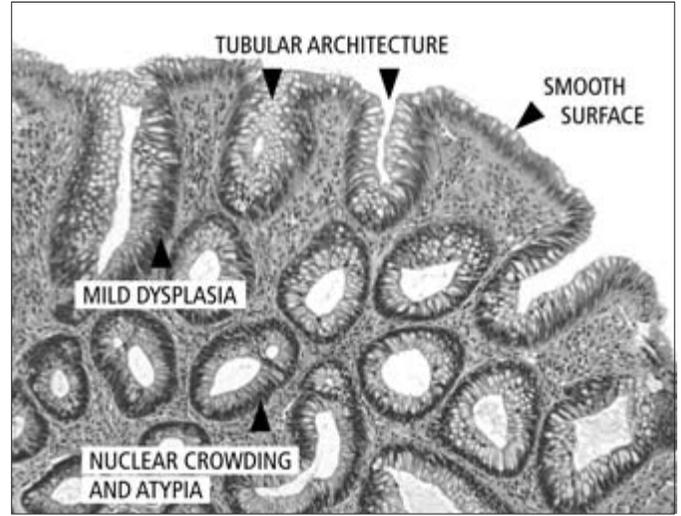
3.102

**Adenomas (cont.)**

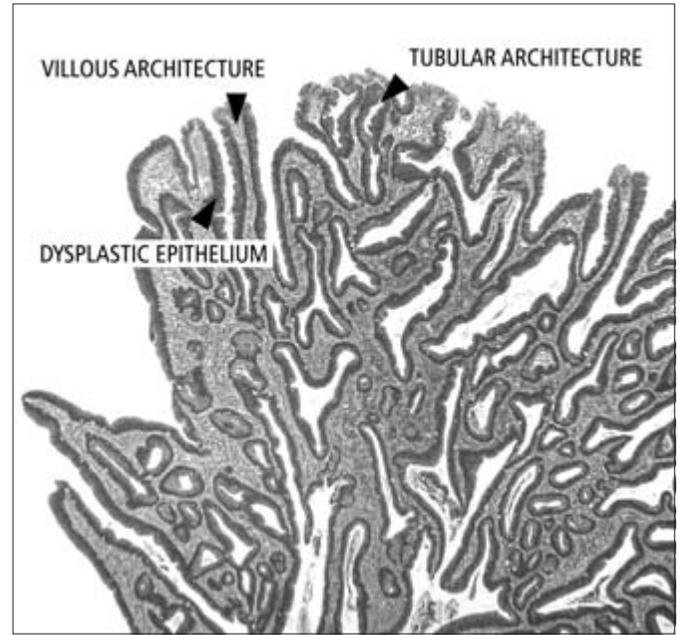
The histopathological appearances of the main types of adenoma are shown in the following figures: tubular (Fig. 3.103); tubulovillous (Fig. 3.104);



3.103

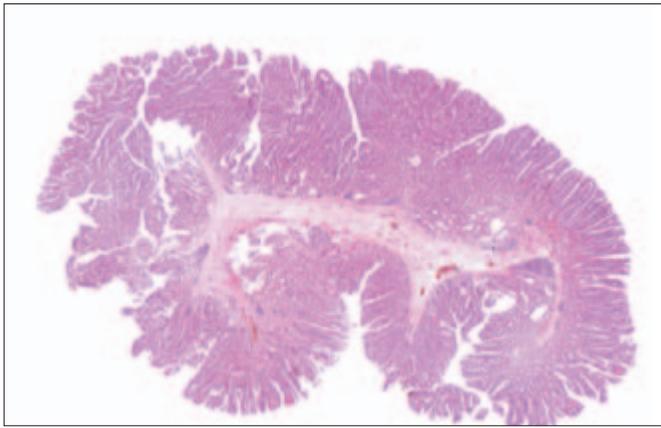


3.104

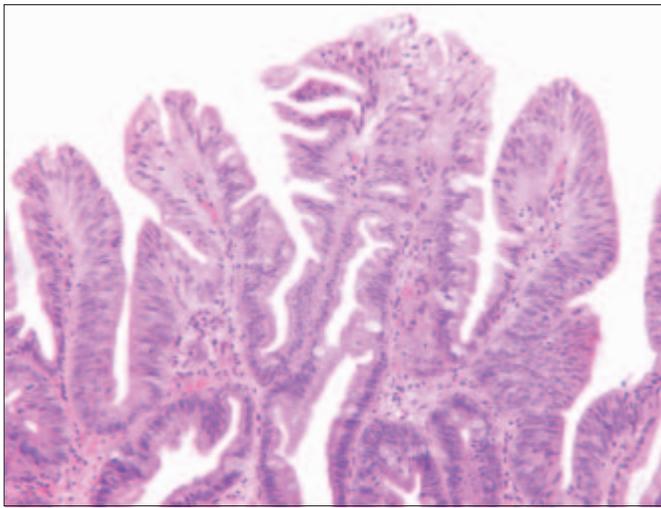
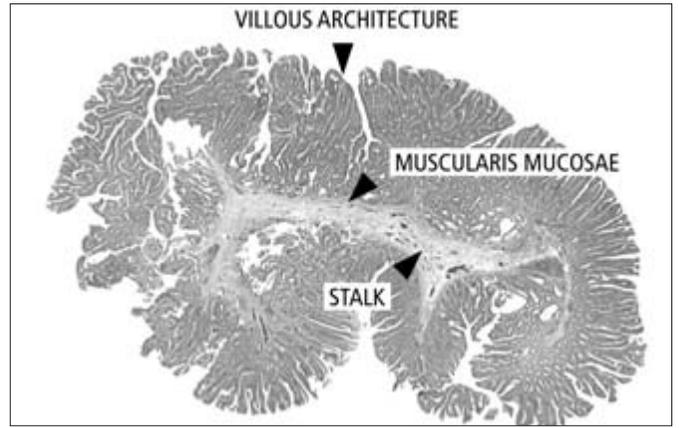


**Adenomas (cont.)**

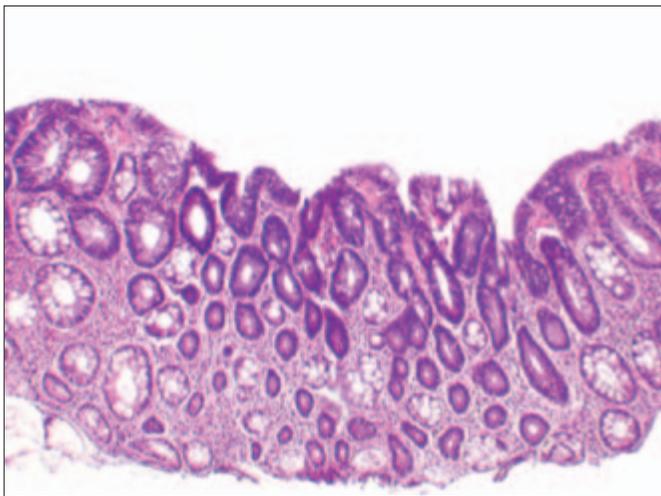
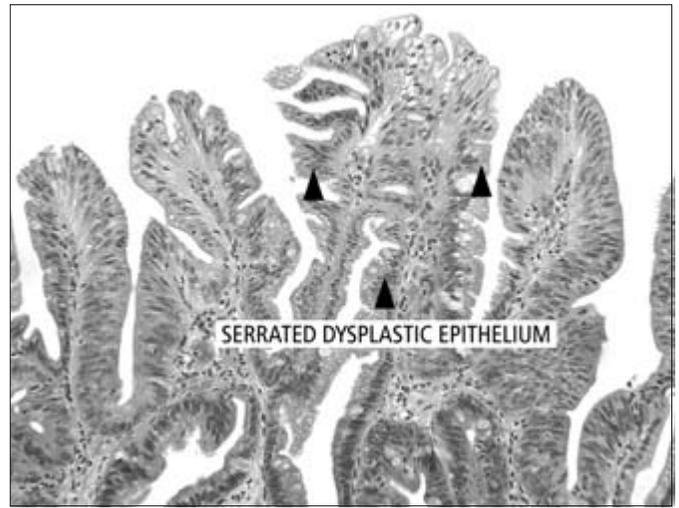
villous (Fig. 3.105); serrated (Fig. 3.106); and flat (Fig. 3.107).



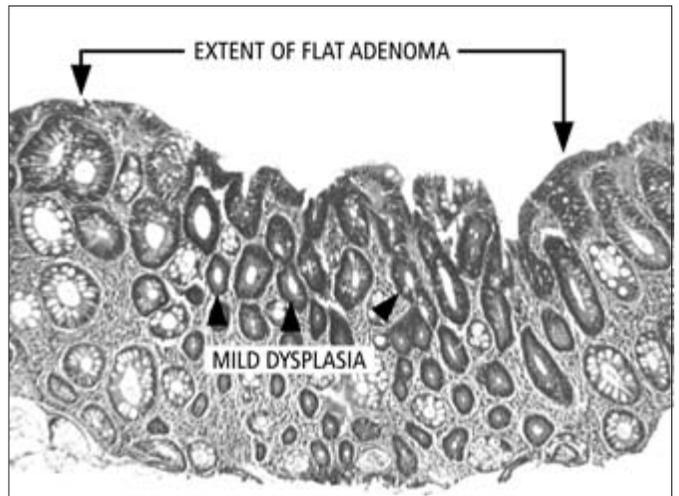
3.105

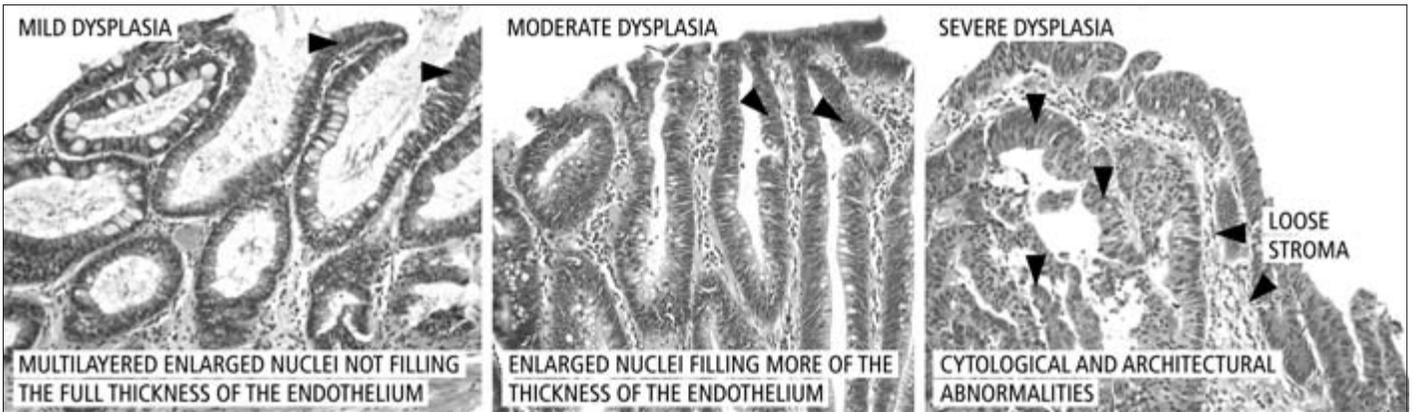
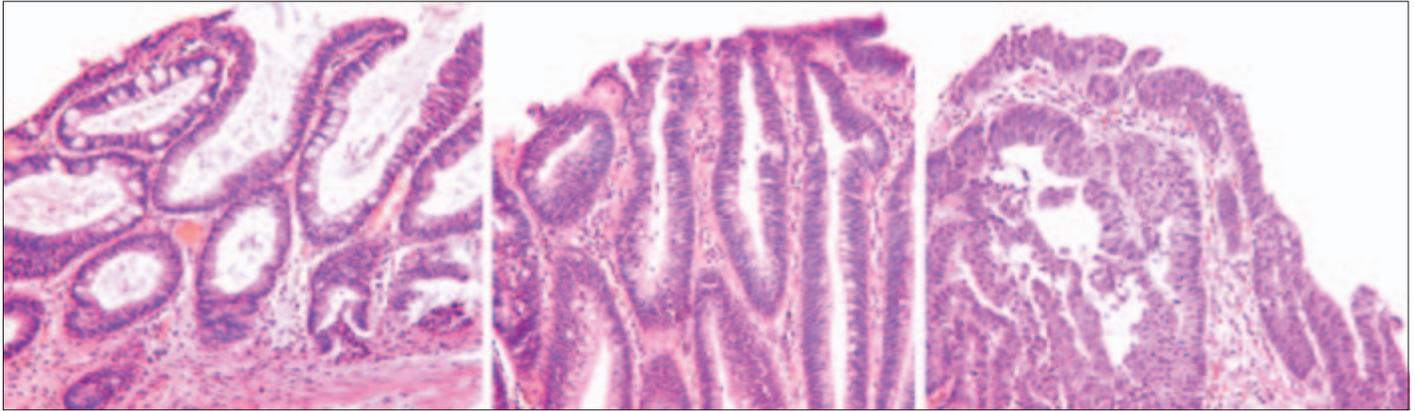


3.106



3.107



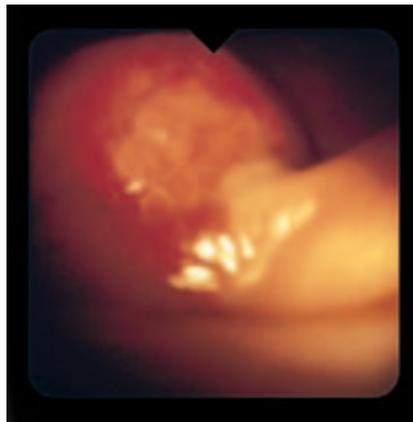


3.108

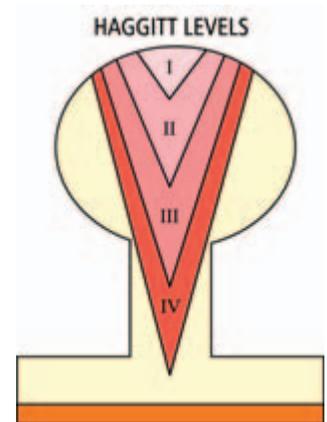
**Adenomas (cont.)**

Dysplasia in adenomas is graded (Fig. 3.108) as mild, moderate and severe. The adenoma-carcinoma sequence describes the development of carcinoma from an adenoma which is, by definition, already dysplastic, and becomes increasingly more dysplastic, finally developing invasive adenocarcinoma.

The misleading endoscopic appearance of a polyp is exemplified by the stalked polyp shown in Fig. 3.109. Although appearing to be a typical tubulovillous adenoma, confirmed after polypectomy, the head was extensively invaded by carcinoma; there was no involvement of the stalk. The Haggitt levels of invasion are shown in Fig. 3.110.



3.109

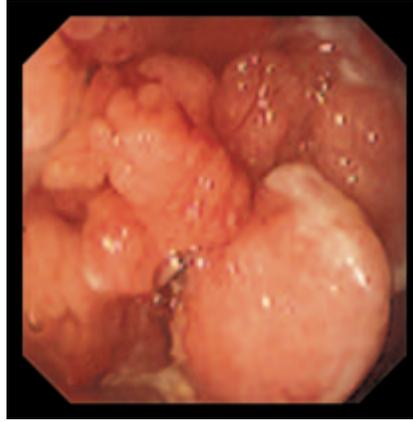


3.110

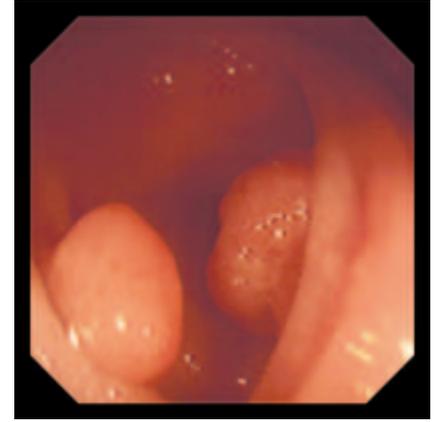
**Adenomas (cont.)**

On the other hand the sessile polyp shown in Fig. 3.111 looks frankly malignant but on resection proved to be a simple villous adenoma.

Adenomas may occur singly or may be scattered, multiple (Fig. 3.112) or in the form of polyposis.



3.111



3.112

**Familial adenomatous polyposis**

Patients with familial adenomatous polyposis (FAP) may have polyps in other parts of the gastrointestinal tract and various extra-gastrointestinal lesions (Gardner's syndrome). There are other familial polyposis syndromes, when much smaller numbers of adenomatous polyps are formed (Lynch syndromes). The adenomatous polyps in all of these conditions, like sporadic adenomas, have malignant potential.

Figure 3.113 shows several small adenomas in a patient with FAP. The polyps in another case (Fig. 3.114) are larger and pinker. Polyps in FAP may be confluent, causing the appearance of 'polyp carpeting' (Fig. 3.115).



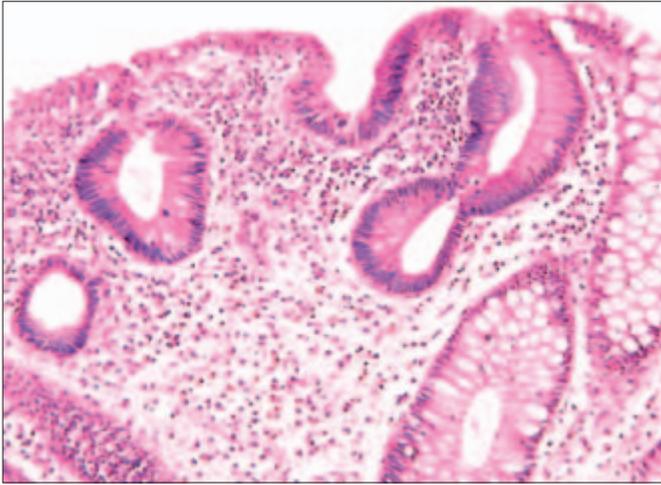
3.113



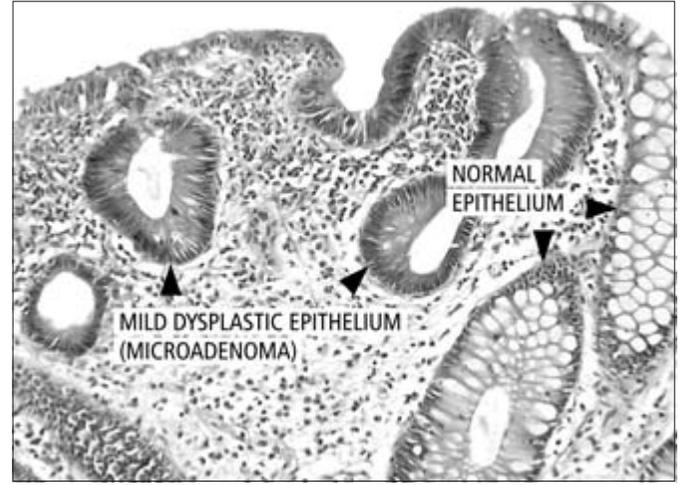
3.114



3.115



3.116



**Familial adenomatous polyposis (cont.)**

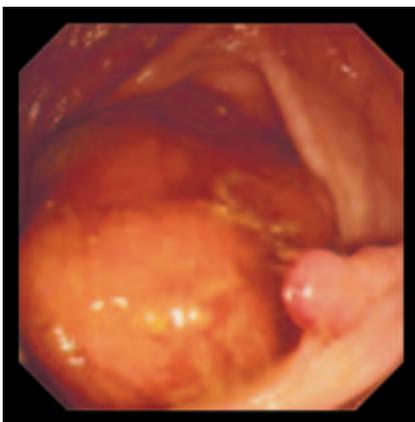
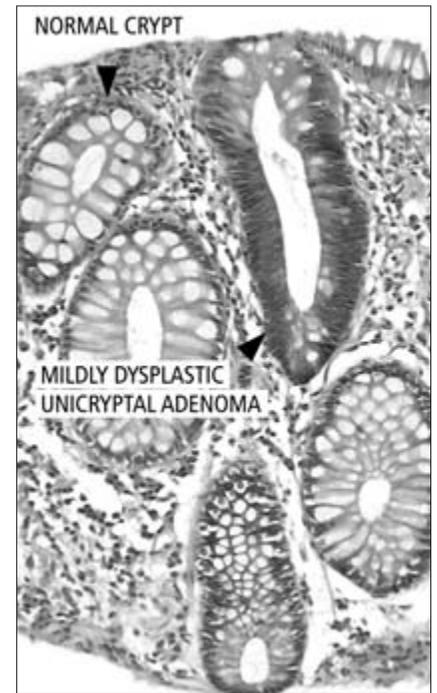
Biopsy of endoscopically normal looking mucosa in a patient with FAP may yield microadenomas (Fig. 3.116) or unicryptal adenomas (Fig. 3.117).

The risk of complicating malignancy correlates with increasing polyp size (whether the patient has FAP or not). This is shown in Fig. 3.118 where the smaller proximal lesion is a simple adenoma, whilst Fig. 3.119 shows an ulcerated carcinoma with many surrounding small polyps.

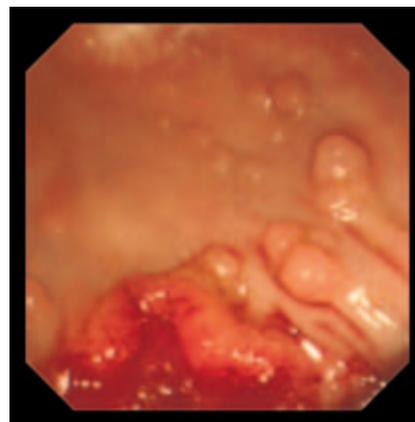
Routine colonoscopy underestimates the number of polyps in polyposis coli. Spraying the mucosa with various dyes, such as methylene blue or indigo-carmin, enhances visibility of small polyps which otherwise may go undetected (Fig. 3.120).



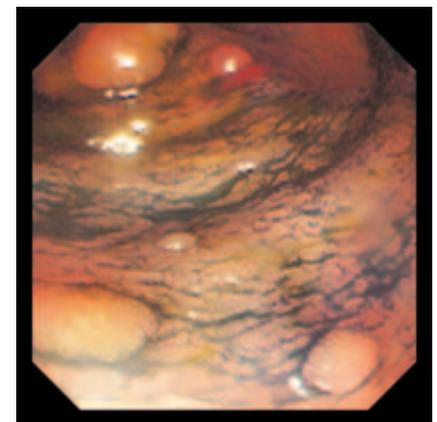
3.117



3.118



3.119



3.120

### Dysplasia-associated lesions or masses (DALM)

DALMs are seen in ulcerative colitis and therefore are discussed in the section on this disorder (p. 180).

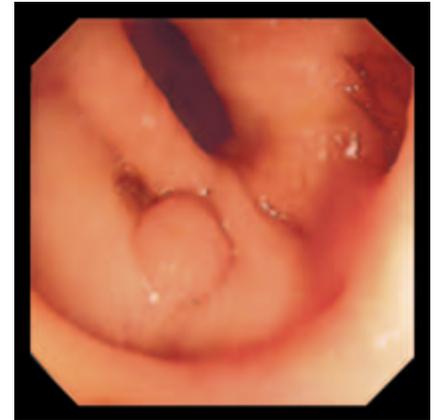
#### Hyperplastic (metaplastic) polyps

Hyperplastic polyps may occur singly or in large numbers. Left-sided metaplastic polyps, which are commonest in the rectosigmoid, are usually small, sessile, regular hemispherical lesions of normal mucosal colour or slightly pinkish (Figs 3.121 and 3.122). They are less common in the right colon where they may vary in colour and shape (Fig. 3.123). Unusually, there may be single or multiple larger metaplastic polyps. Multiple colonic metaplastic polyps occur as part of the non-inherited metaplastic polyposis syndrome. It has recently been recognized that a very small proportion of larger hyperplastic polyps may have dysplastic potential. The clinical implications of this are as yet uncertain.

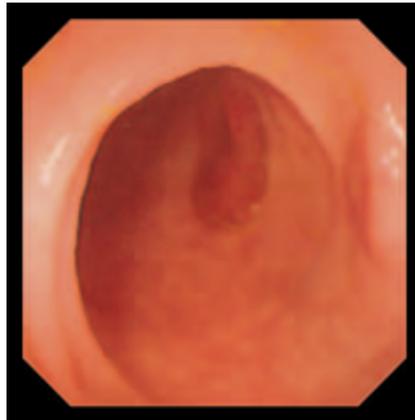
Figure 3.124 shows the histological appearances of a metaplastic polyp.



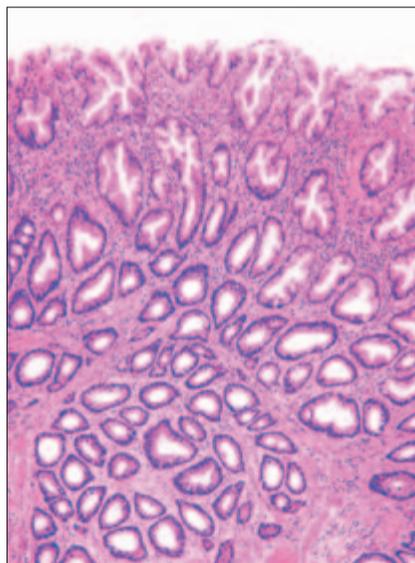
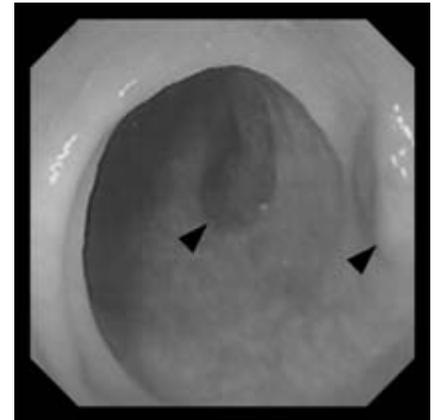
3.121



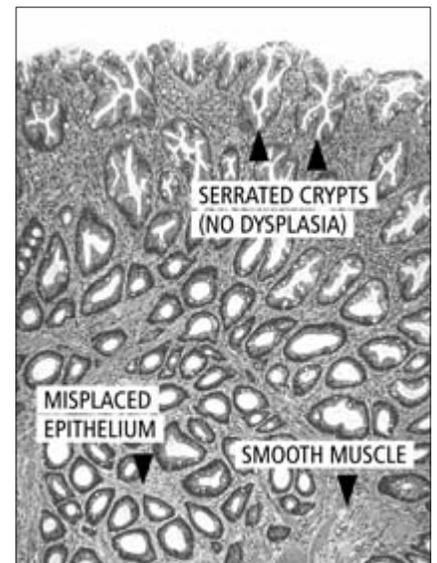
3.122



3.123

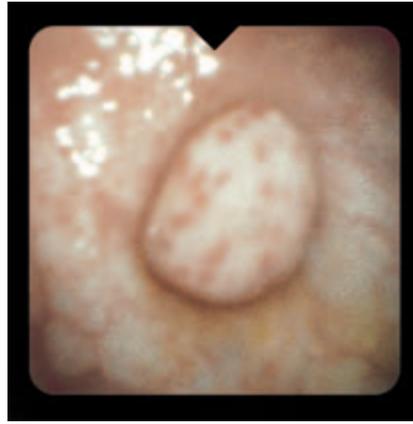


3.124

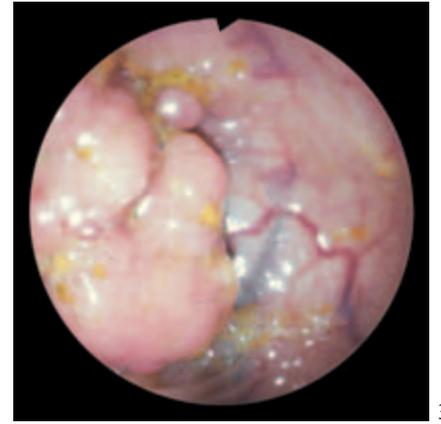


### Juvenile polyps

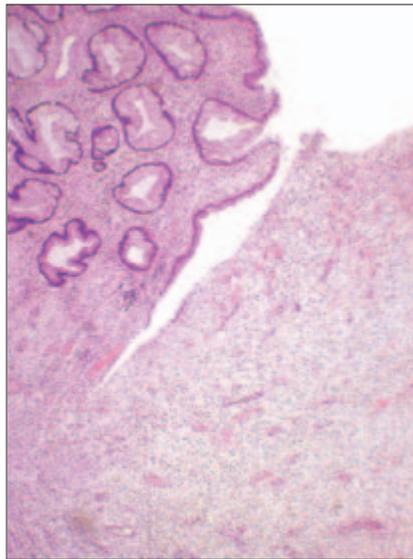
These hamartomatous lesions (Fig. 3.125) have a characteristic mottled appearance when small. Larger juvenile polyps may lose this individuality (Fig. 3.126) becoming indistinguishable from other types of polyp; resection then becomes essential for diagnosis. Figure 3.127 shows the histological appearance. The malignancy risk in juvenile polyps is low and is confined to patients with juvenile polyposis rather than with one or a few polyps.



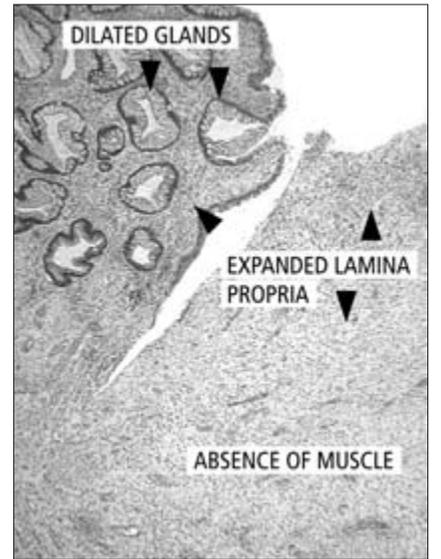
3.125



3.126



3.127

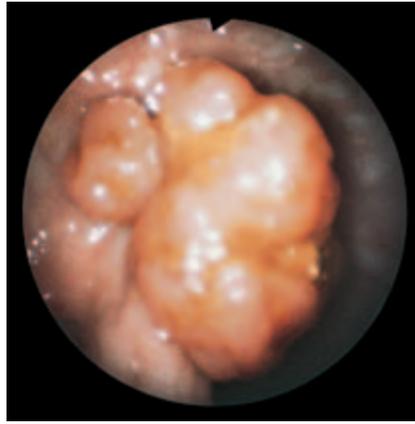


### Peutz–Jeghers polyps

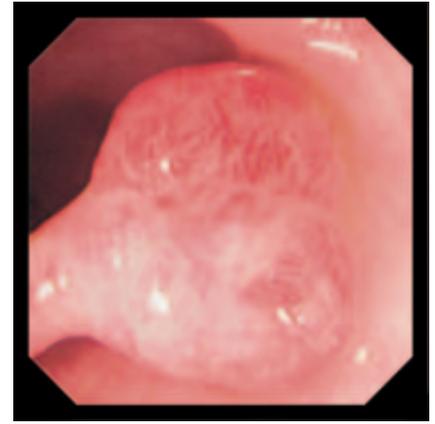
In the Peutz–Jeghers syndrome, which is inherited in an autosomal dominant fashion, intestinal hamartomas occur in association with mucocutaneous pigmentation. These colonic lesions, which have no specific colonoscopic features, have a low malignant potential.

A Peutz–Jeghers polyp is shown in Fig. 3.128. Another appears in Fig. 3.129; note the ‘cerebral’ surface patterns more suggestive of adenoma. There are several Peutz–Jeghers polyps in Fig. 3.130; the largest, which is about to be removed, shows a pattern of mucosal crypts (cf. Figs 3.6 and 3.7). The polyp appearing in Fig. 3.131 has a notched ulcerated defect.

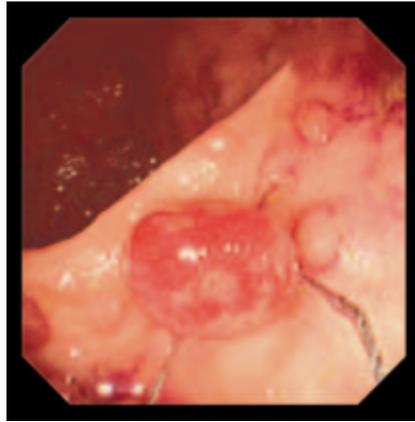
The histology of a Peutz–Jeghers polyp is shown in Fig. 3.132.



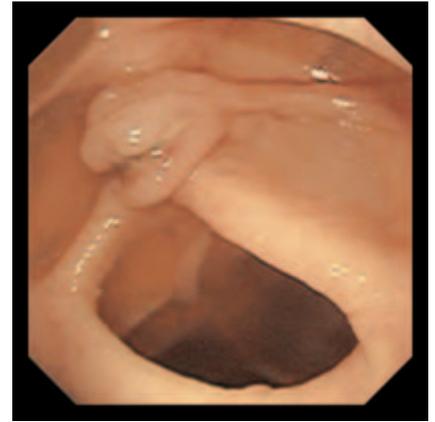
3.128



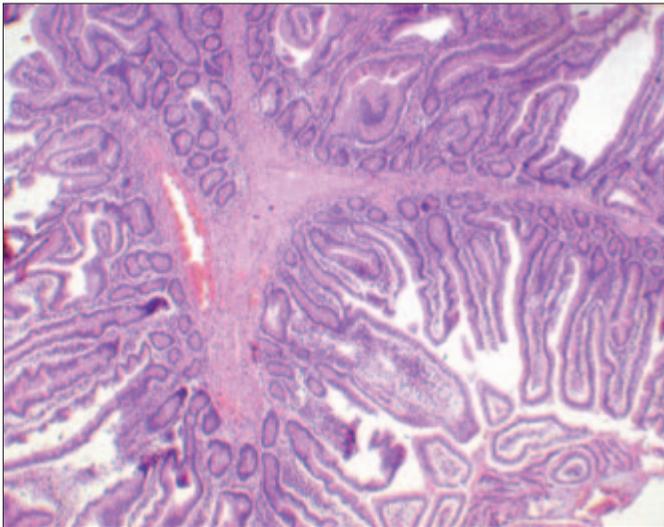
3.129



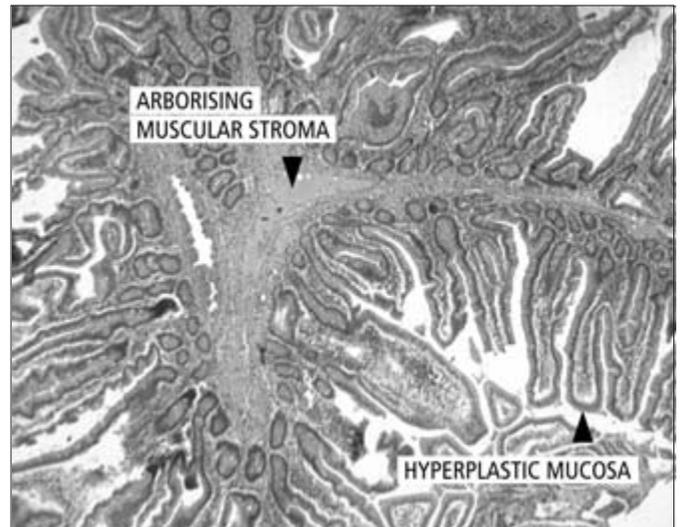
3.130



3.131

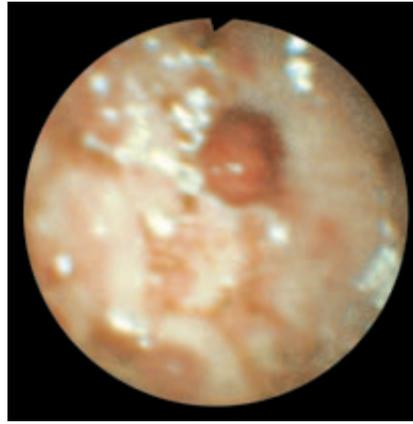


3.132

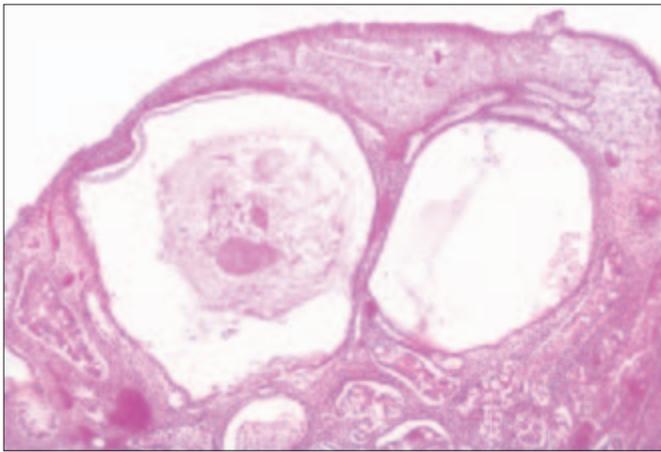


### Cronkhite–Canada polyps

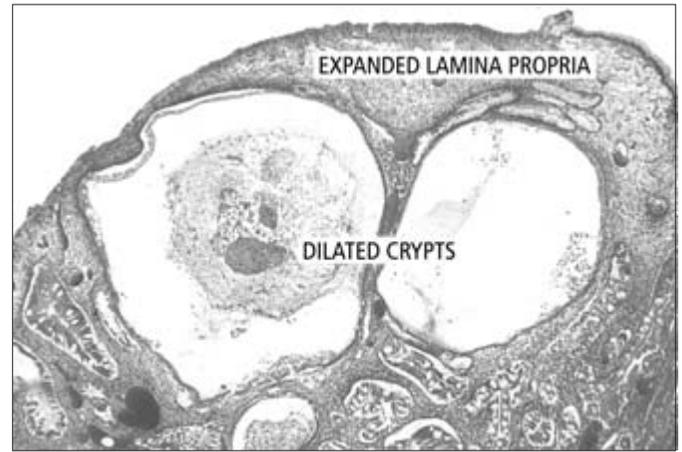
Polyps which are similar histologically to juvenile polyps occur in the Cronkhite–Canada syndrome (Fig. 3.133). Associated features include severe diarrhoea, alopecia, nail dystrophy and skin pigmentation, together with gastric and intestinal polyposis, electrolyte disturbances and hypoproteinaemia. The aetiology is unknown and there seems to be no familial tendency. Figure 3.134 shows the histology.



3.133



3.134



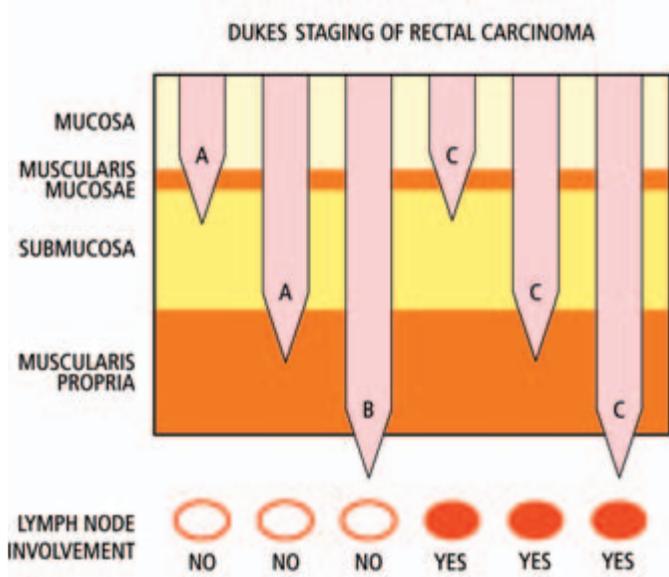
### Inflammatory polyps

This type of polyp may occur in isolation or as part of inflammatory bowel disease. When seen in isolation such polyps may be related to mucosal prolapse. For further description the reader is referred to the section on inflammatory bowel disease (pp. 270–289).

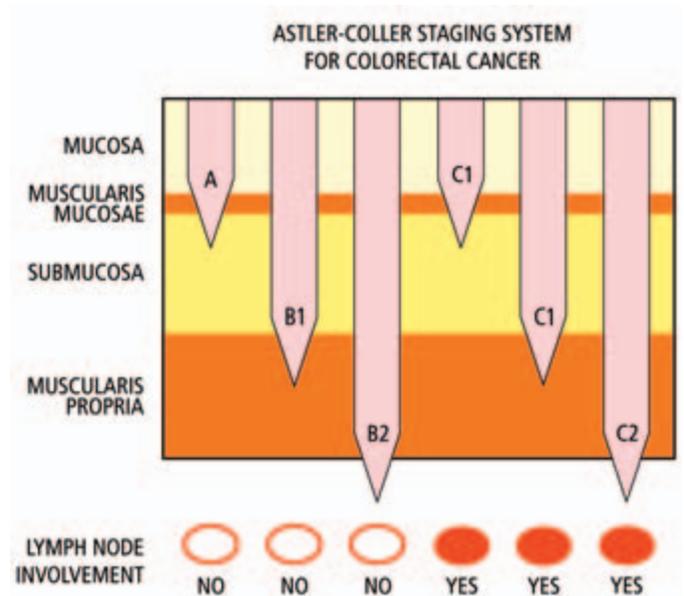
### Adenocarcinoma

Colorectal carcinoma is the second commonest malignancy in the United Kingdom after lung cancer, and in the United States after skin cancer. As the anatomical distinction between rectum and colon is sometimes arbitrary, exact incidence are necessarily uncertain. There is some evidence that the incidence of colon cancers is steadily increasing compared with rectal cancers. In adenocarcinoma of the colon and rectum, prognosis is dependent upon the extent of invasion, the degree of differentiation, and whether there are lymph node metastases. The classic staging system in common use in the United Kingdom is that described by Dukes (Fig. 3.135) for carcinoma of the rectum but now applied to carcinoma occurring anywhere in the large bowel. In a Dukes A stage case the carcinoma has spread through the muscularis mucosae but not beyond the outer surface of muscularis propria and there is no involvement of the lymph nodes. In Dukes B the growth has spread beyond the muscularis propria and there are no lymph node metastases. Once the lymph nodes are involved the tumour is categorised as Dukes C irrespective of the depth of invasion. The original Dukes classification has been variously modified so that Dukes stage C1 denotes local lymph node involvement while Dukes stage C2 describes apical (high tie) lymph node involvement; there is also a further modification, Dukes stage D, denoting distant metastases. Possible confusion arises with the American Astler–Coller system which uses the letters A, B and C but in a different way (Fig. 3.136).

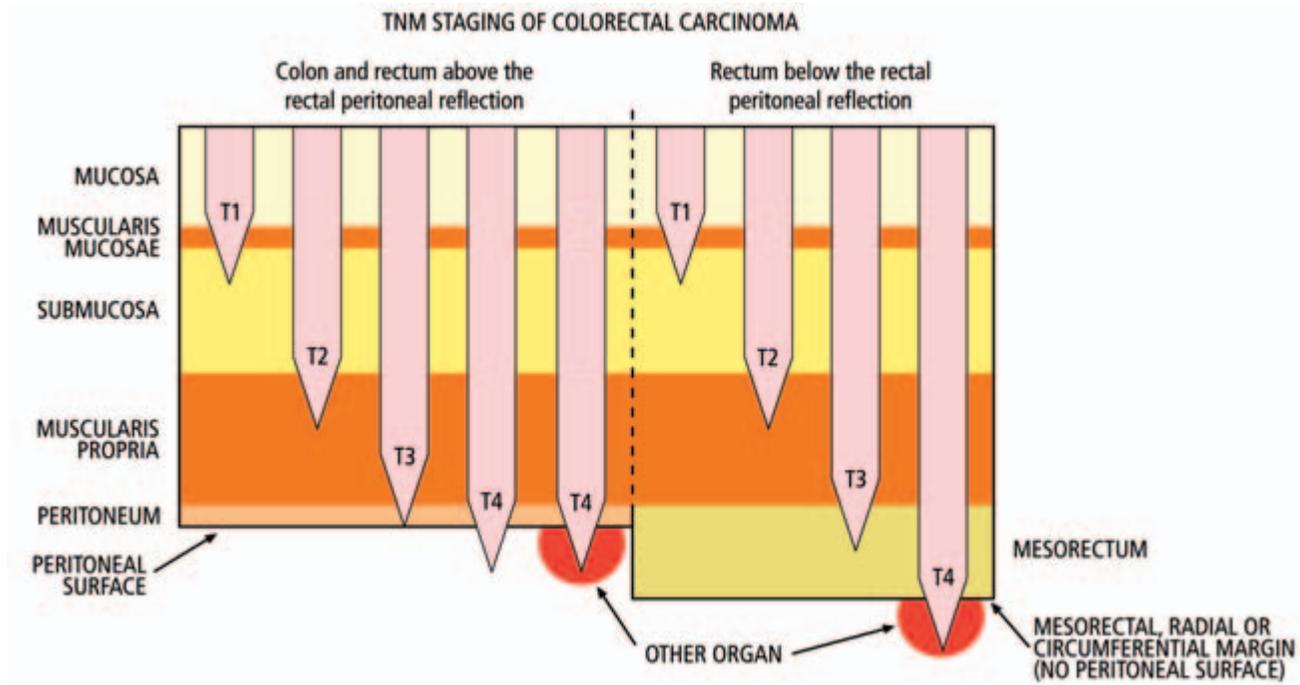
The current international standard is the TNM system as shown in Fig. 3.137, where T denotes tumour, N lymph nodes and M metastases. The classification is applied differently for colon and rectum above the peritoneal reflection, and rectum below this. In the first group T4 denotes involvement of the peritoneal surface or other organs by direct spread, while for the distal rectum a T4 lesion involves another organ beyond the mesorectal, radial or circumferential margin, there being no peritoneal surface. N0 signifies no nodes involved, N1 one to three nodes involved and N2 four or more nodes involved. M0 denotes no evidence of distant metastases while M1 signifies histologically confirmed distant metastases. Under the TNM system there is also an additional staging of surgical resection margin involvement: this refers to the radial, circumferential or mesorectal margin in the rectum, and the mesocolic margin in the colon, and does not refer to the peritoneal surface. R0 is used to describe a clear margin of resection, R1 microscopic involvement of the margin and R2 visible residual tumour at the resection margin.



3.135



3.136



3.137

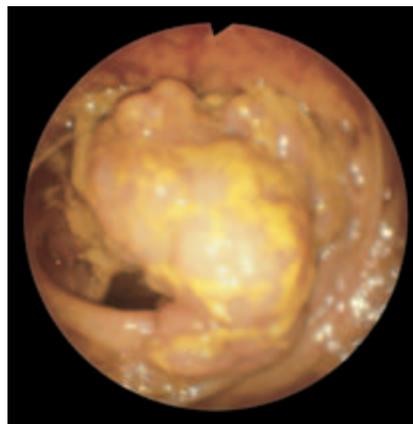
### Adenocarcinoma (cont.)

Detailed consideration of prognosis are beyond the scope of this Atlas. Suffice it to say that the 5-year survival after formal surgical resection of a Duke's A carcinoma of the colon is 90%, reducing to 30% for a Duke's C lesion.

Forceps biopsies are likely to be diagnostic when the appearance of the lesion is typical of carcinoma (Fig. 3.138) but multiple bites should be taken as there may be coexisting adenomatous tissue making erroneous diagnosis possible. If necessary snare biopsies can be taken to ensure adequate material for interpretation.

Carcinomas vary in shape, size and appearance, though there are no aspects of appearance which are specific to any part of the large bowel. They occur more commonly on the left side, and presenting features may vary with site, as does the differential diagnosis.

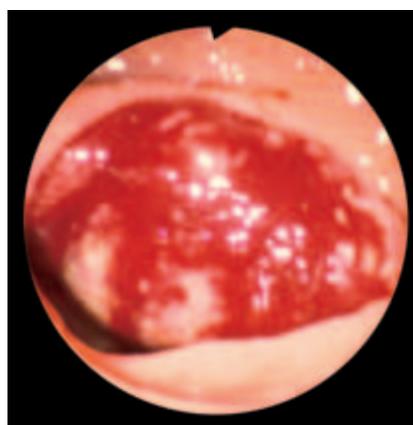
Obvious irregular polypoid carcinoma in the sigmoid (Fig. 3.139) and transverse colon (Fig. 3.155) presents little difficulty in diagnosis. The haemorrhagic polypoid carcinoma in Fig. 3.140 is almost completely obstructing the lumen at the splenic flexure.



3.138



3.139



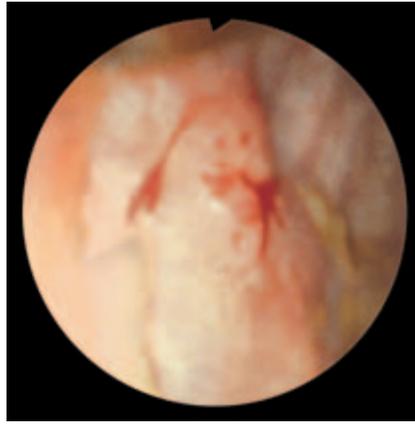
3.140

**Adenocarcinoma (cont.)**

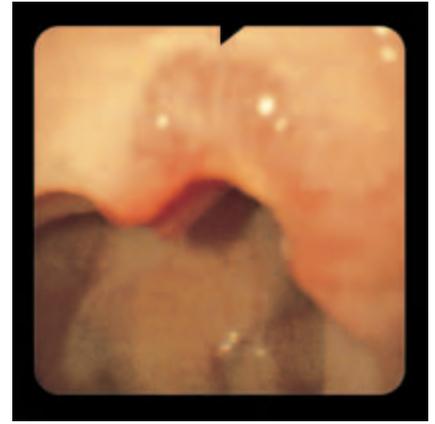
The flat lesion seen in Fig. 3.141 is not obviously a carcinoma and the abnormal superficial vasculature might be confused with a small vascular lesion. Distortion of the mucosal fold in Fig. 3.142 raises the suspicion of malignancy which was confirmed by biopsy. Small discoid lesions such as these may have arisen from polyps which have undergone necrosis after the stalk has been invaded by malignant tissue, but more probably are small primary growths. A larger discoid carcinoma is shown in Fig. 3.143.

The distal margin of a colonic carcinoma when first seen colonoscopically often appears pinker than the normal large bowel mucosa, irregular in shape but with an unbroken surface (Fig. 3.144). In the variant shown in Fig. 3.145 there is marked ulceration of the distal margin. The rectal carcinoma illustrated in Fig. 3.146 is irregular in form and bled easily on contact with the colonoscope. Spontaneous bleeding is frequently seen.

Appearances of the distal margin offer little clue to what will be seen as the endoscope is advanced. Figure 3.147 shows a raised, irregular and ulcerated lip but higher views (Figs 3.148 and 3.149) revealed a deeply ulcerated lesion.



3.141



3.142



3.143



3.144



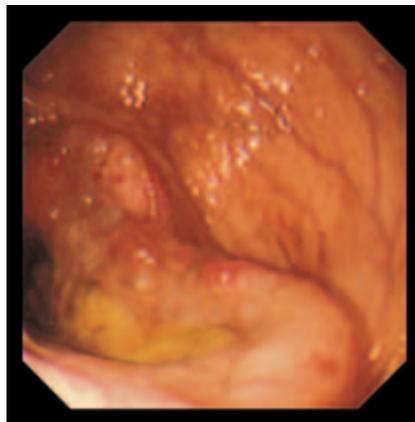
3.145



3.146



3.147



3.148



3.149

### Adenocarcinoma (cont.)

The lesion demonstrated in Fig. 3.150 was virtually obstructing the colon. Attempts to advance the colonoscope failed and led to some localized bleeding. It was clearly not possible to enter the obstructing lesion shown in Fig. 3.151.

When considering the best method of treatment, it is helpful to know the estimated length of the lesion. If this cannot be assessed using a standard width colonoscope, consideration should be given to substituting a smaller diameter instrument with which it may be possible to traverse the stricture.

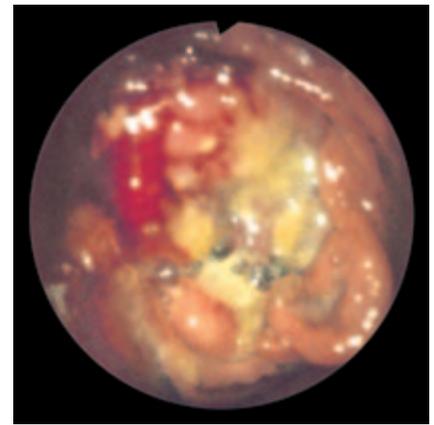
Carcinoma occurs less commonly on the right than the left, but the variable appearances of tumours are similar throughout the colon. Nevertheless, the site is of some diagnostic and prognostic importance. The lesion shown in Fig. 3.152 at first glance resembles an ileocaecal valve, though on closer inspection it is clearly an irregular malignant lesion. Patients with right-sided carcinoma seldom have intestinal symptoms such as a change in bowel habit or obstruction, but iron deficiency anaemia is the commonest presentation, as was the case in the patient whose carcinoma appears in Fig. 3.153. Bleeding was spontaneous and not caused by the instrument, yet there was no clinical history of overt bleeding.

Severe acute rectal bleeding makes colonoscopy difficult and perhaps unrewarding. It is however, the investigation of choice, superior to radiology, during the subsequent work up. Figure 3.154 illustrates bleeding from a colonic carcinoma. As commonly happens the presence of blood led to a somewhat fuzzy picture.

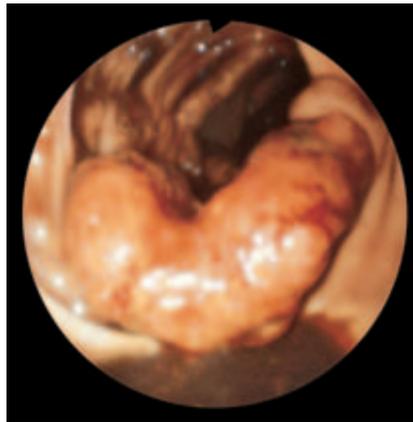
Synchronous neoplastic lesions, either benign adenomas or a second carcinoma, are found in up to 25% of patients with colorectal carcinoma.



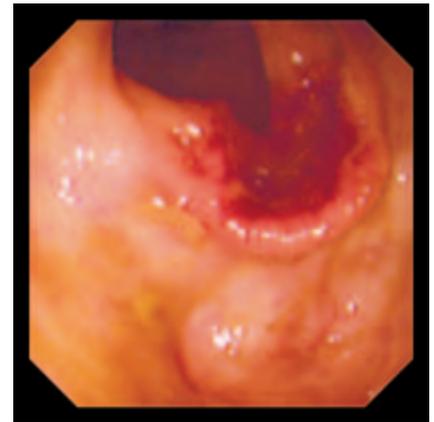
3.150



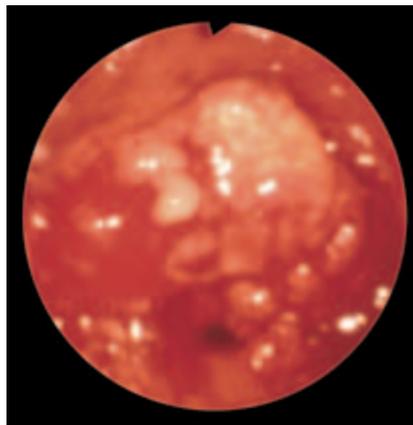
3.151



3.152



3.153



3.154



**Adenocarcinoma (cont.)**

This stenosing ulcerated carcinoma of transverse colon (Fig. 3.155) was accompanied by a small adenomatous polyp in the sigmoid. Because of the high rate of synchronous lesions patients with colorectal carcinoma should undergo colonoscopy (or barium enema) before surgery. Total colonoscopy is not always possible if there is an obstructing lesion. When a complete examination of the colon has not been made before surgery, follow up colonoscopy should be undertaken after three months and perhaps regularly thereafter.

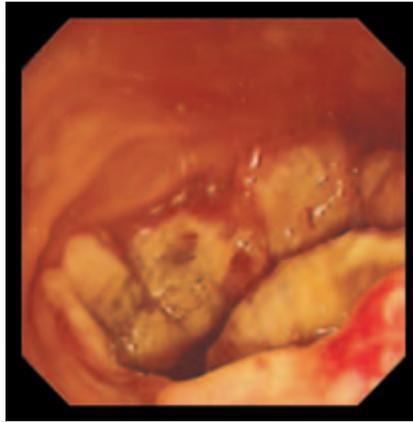
Carcinoma complicating familial adenomatous polyposis (FAP) has already been discussed (see pp. 254–255). Figure 3.156 is another illustration of a complicating carcinoma in this condition.

Extensive diverticular disease presents the radiologist with problems. Often a confident opinion cannot be given with regard to possible coexistent disease, especially carcinoma. Colonoscopy is valuable in this situation. Figure 3.157 shows a large polypoid carcinoma in the sigmoid colon affected by diverticular disease.

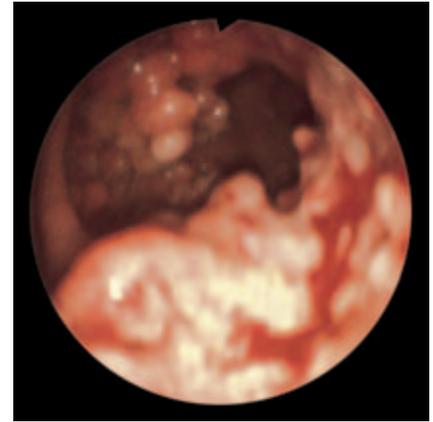
Carcinoma in ulcerative colitis is discussed in the section on inflammatory bowel disease (pp. 274–281). Figures 3.158 and 3.159 are typical examples. Crohn’s disease of the large bowel may sometimes lead to cancer. Carcinomas complicating inflammatory bowel disease may be indistinguishable from those seen in patients without this condition.

**Kaposi’s sarcoma**

This previously rare tumour is today seen most frequently in patients with AIDS (Fig. 3.160). There are no specific colonoscopic features to distinguish it from carcinoma. Figure 3.161 shows the histological appearances.



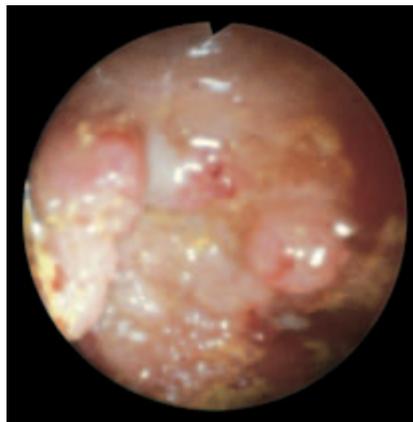
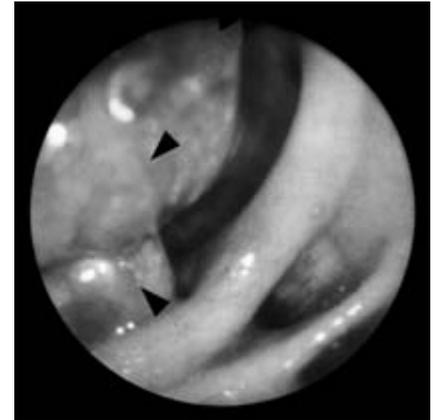
3.155



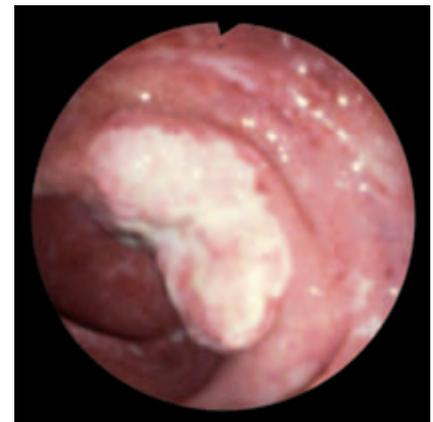
3.156



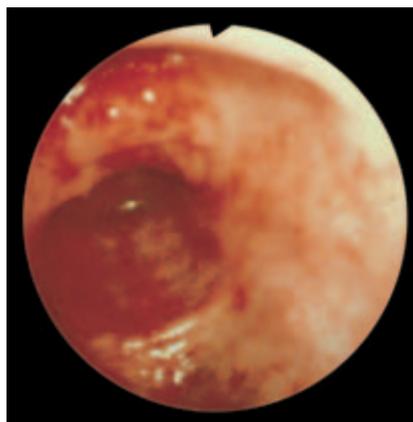
3.157



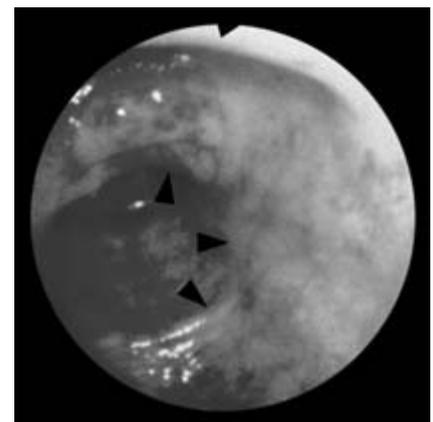
3.158



3.159



3.160



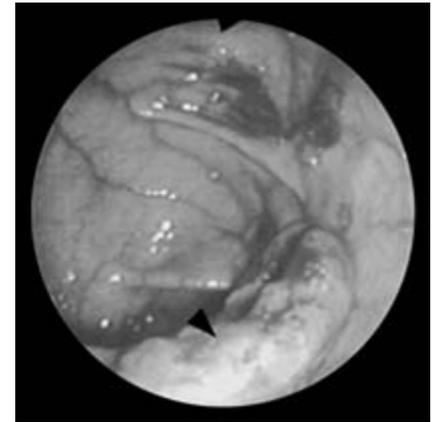
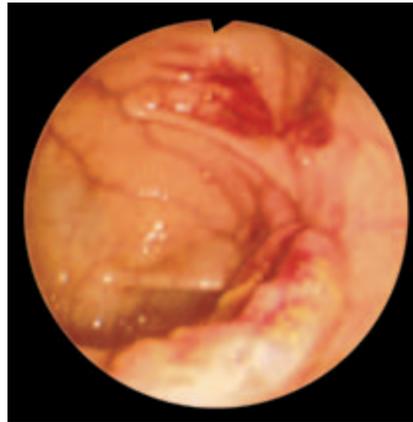
**Kaposi's sarcoma (cont.)**



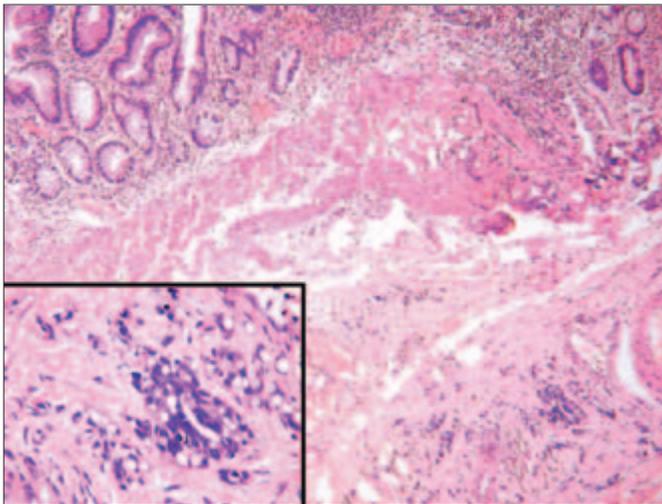
3.161

**Secondary and direct spread tumours**

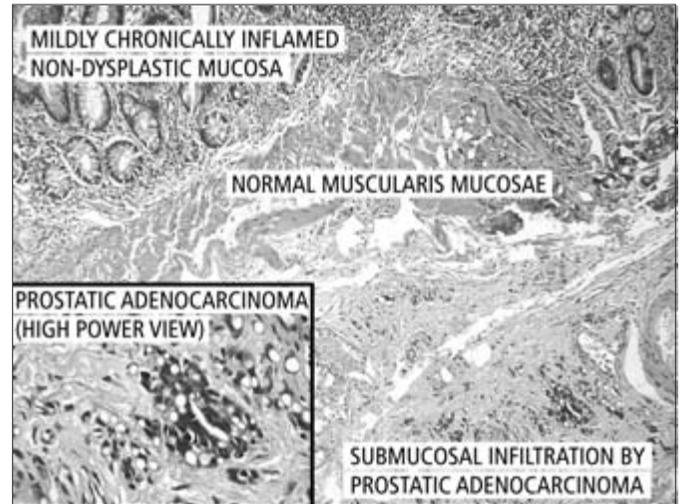
Tumours may rarely metastasize to the colon and may then be visible through the colonic mucosa or may breach this. Figure 3.162 illustrates a secondary deposit from a primary in the breast, while Fig. 3.163 shows the histological appearances of an invasive prostatic adenocarcinoma of the rectum stained with prostate specific antigen.



3.162



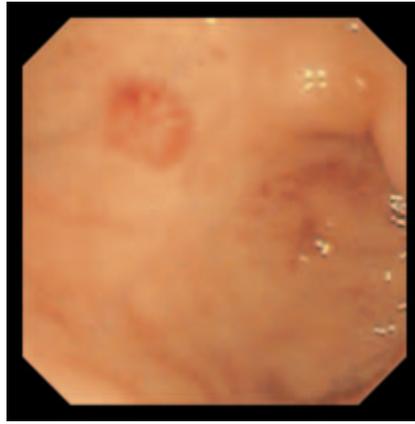
3.163



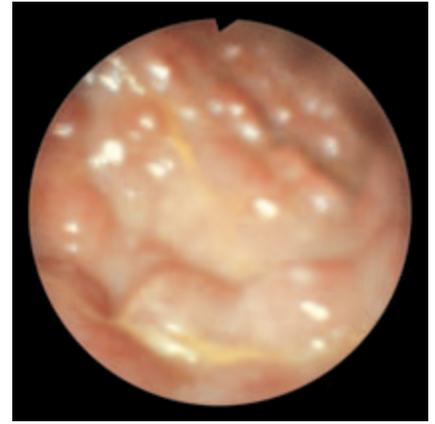
### Lymphoma

Large bowel lymphoma may present a variety of appearances to the colonoscopist. Figure 3.164 shows a single small nodule. There are multiple nodules in Fig. 3.165. In Fig. 3.166 there is an ulcerated exophytic lesion with a regular outline while Fig. 3.167 illustrates an extensive irregular ulcerated lesion. A large polypoid lesion may rarely lead to intussusception (Fig. 3.168).

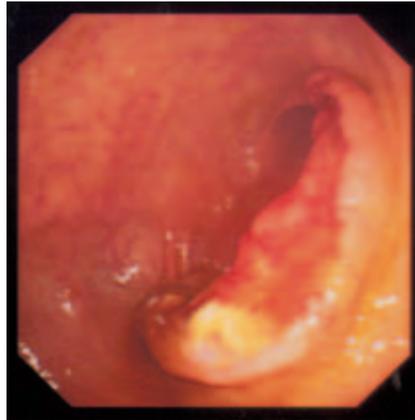
The terminal ileum can often be entered at colonoscopy; Fig. 3.169 shows a MALT lymphoma of the terminal ileum. Multiple lymphomatous polyposis can endoscopically mimic Crohn's disease. This is a mantle zone lymphoma. Figure 3.170 shows mantle zone lymphoma with abnormal lymphoid follicles and a mucosal infiltrate.



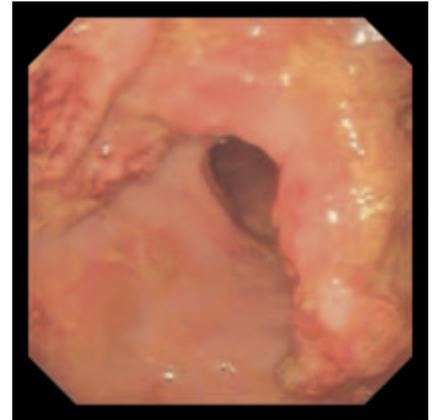
3.164



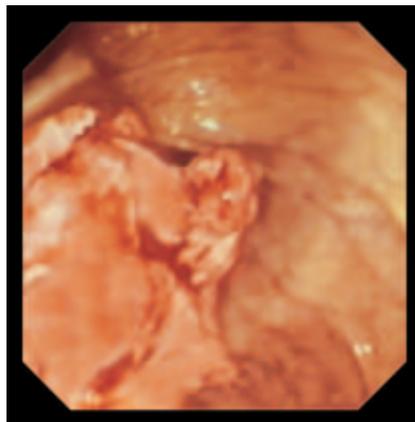
3.165



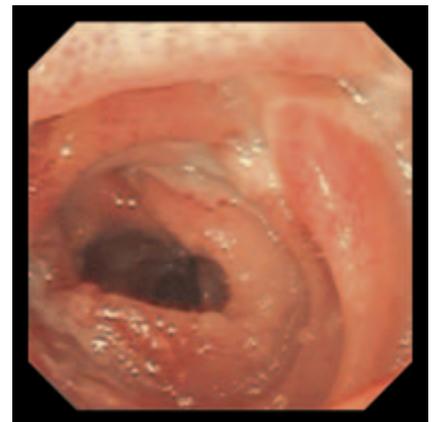
3.166



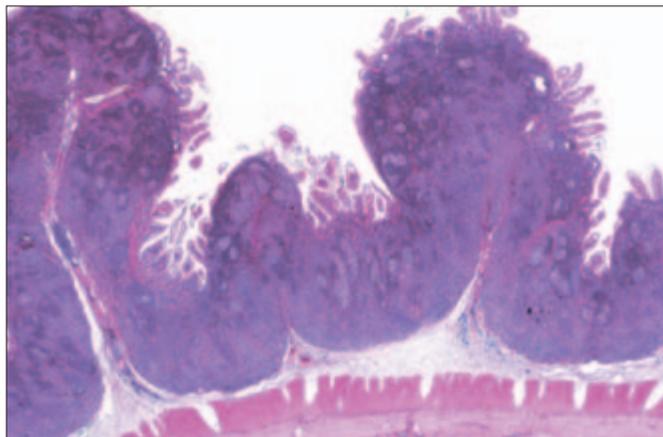
3.167



3.168



3.169



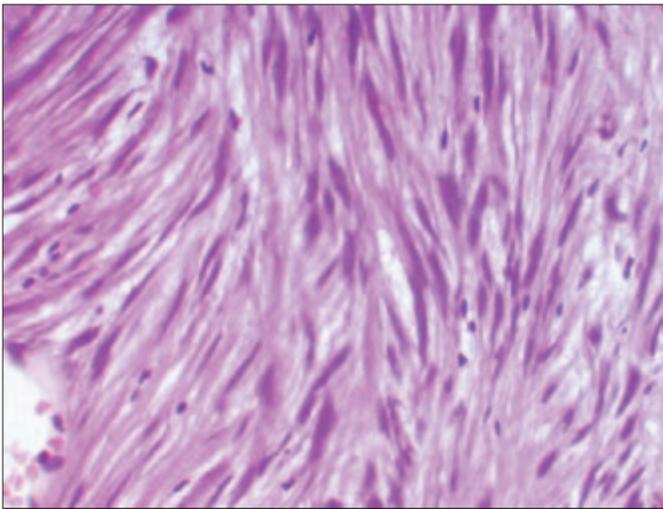
3.170

**Gastrointestinal stromal tumours (GISTs)**

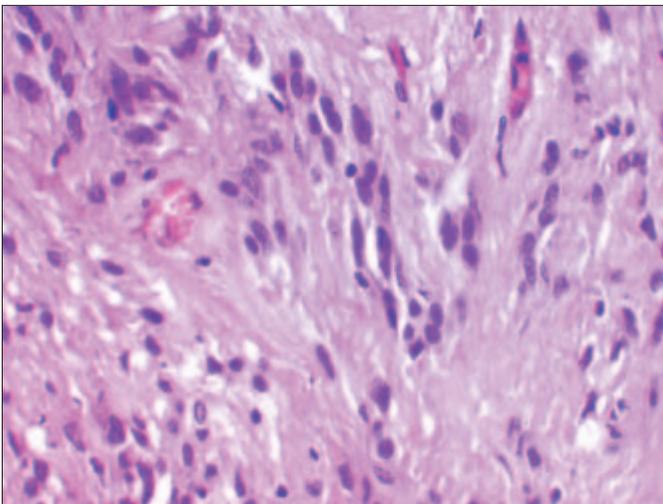
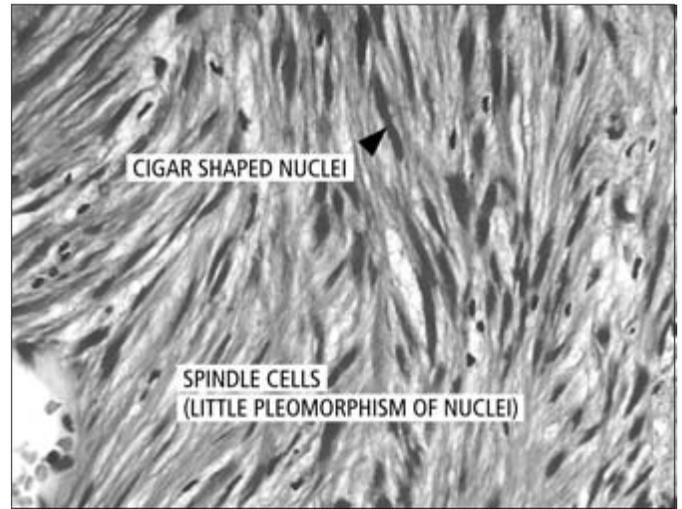
The small submucosal polyp shown in Fig. 3.171 has no pathognomonic features. Biopsies revealed the typical appearances of a GIST. Figure 3.172 demonstrates the histological features of a benign GIST while a malignant GIST is illustrated in Fig. 3.173. These lesions are discussed more fully on pp. 124–126.



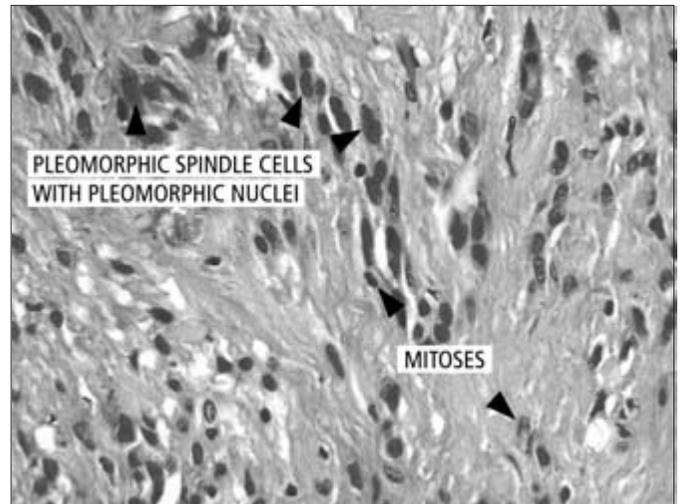
3.171



3.172

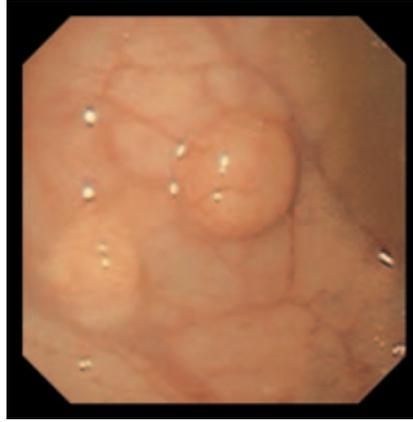


3.173

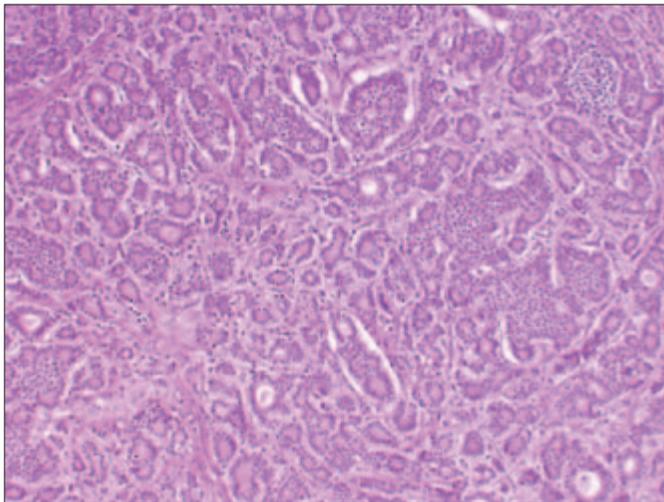


### Neuroendocrine tumours

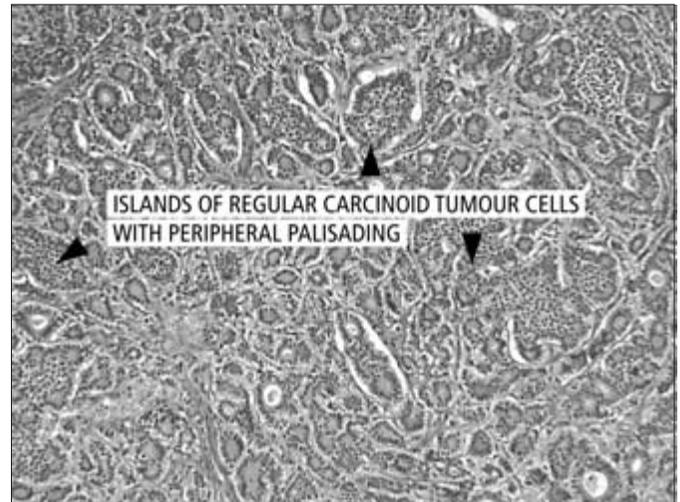
This uncommon lesion, also known as a carcinoid tumour, is another example of a polypoid lesion without specific endoscopic appearances. Lesions may be single or multiple. Two such tumours appear in Fig. 3.174. The pathological appearances of a carcinoid tumour of rectum are shown in Fig. 3.175 (see also p. 119).



3.174



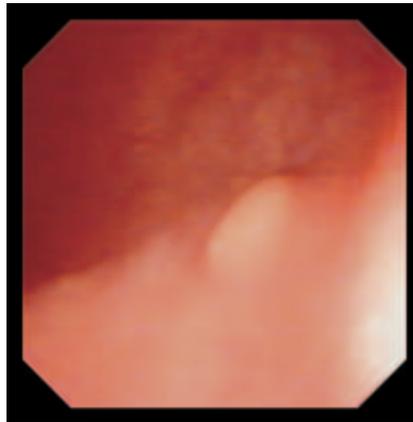
3.175



### Lipomas

These are probably the commonest submucosal polyps occurring in the large bowel. Being covered by normal mucosa they do not usually differ significantly in colour from the surrounding tissue. Small lesions are normally sessile while some large submucosal polyps become pedunculated. Inspection does not permit differentiation between tissue types and superficial biopsy usually only yields normal mucosa. Cell type can be determined only following polypectomy.

Figure 3.176 shows a small sessile lipoma in the caecum just above the ileocaecal valve which lies behind the fold beyond the tumour.



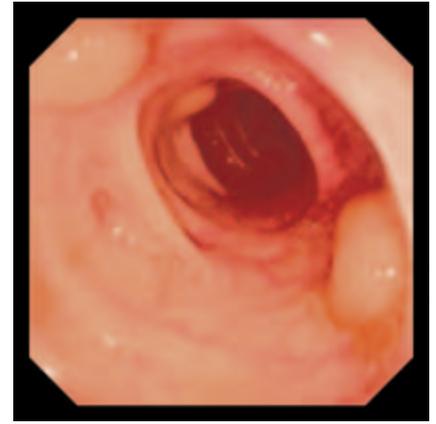
3.176

**Lipomas (cont.)**

A larger typical pedunculated lipoma appears in Fig. 3.177. Rarely, lipomas may be multiple (Fig. 3.178). They often show the 'pillow sign' where a dimple is left after compression with the tip of the biopsy forceps (Figs 3.179a and b). Histology of a lipoma appears in Fig. 3.180.



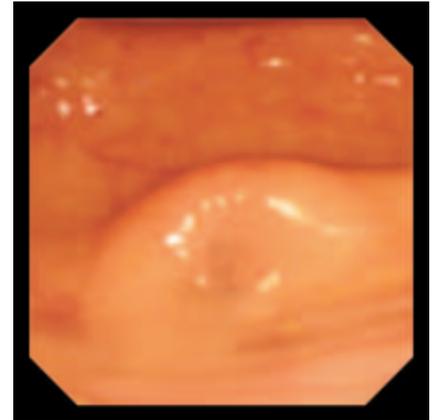
3.177



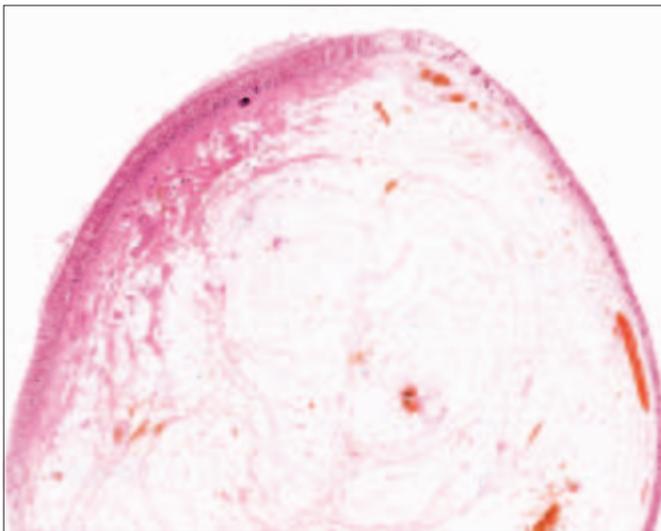
3.178



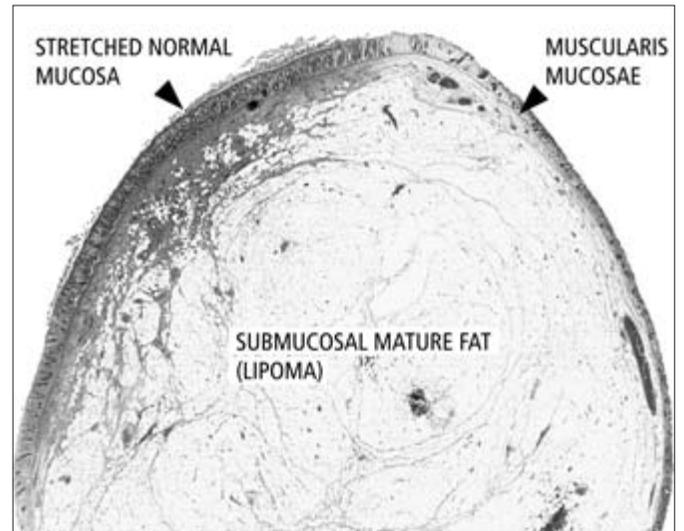
3.179a



3.179b



3.180



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## Inflammatory bowel disease

### Nonspecific nature of endoscopic appearances

#### Indeterminate colitis

#### Minimal change colitis

#### Microscopic colitis

#### Ulcerative colitis

#### Crohn's disease

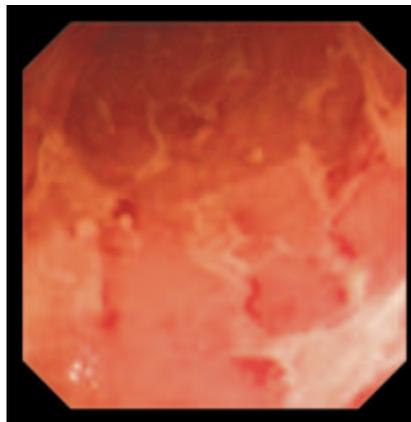
While a good quality double contrast barium enema is often suggestive of the correct diagnosis and permits assessment of the apparent extent of colonic involvement, colonoscopy is now the investigation of choice in inflammatory bowel disease particularly as it enables biopsy material to be obtained.

In acute colitis, of whatever cause, colonoscopy is preferably avoided as it is not often necessary for diagnosis and may result in perforation. In subacute and chronic disease the extent of colitis as assessed by endoscopy and biopsy is usually greater than when judged by radiological appearances, and occasionally a pancolitis may be detected when radiographic appearances are normal.

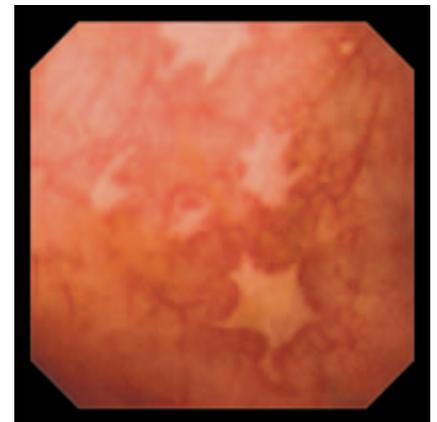
An important role for colonoscopy is in the surveillance of longstanding extensive ulcerative colitis where there is an increased risk of developing carcinoma. Here regular and systematic examination with mucosal biopsies taken at intervals throughout the colon to detect premalignant changes in the form of dysplasia is helpful in the selection of cases for prophylactic colectomy. The use of surveillance in Crohn's disease is not established. Colonoscopy is also valuable for assessment of polypoid lesions or strictures demonstrated radiographically in patients with inflammatory bowel disease.

### Nonspecific nature of endoscopic appearances

The endoscopic manifestations of inflammation of the colonic mucosa are often nonspecific and the final diagnosis must rest on all available evidence, combining clinical history, findings on physical examination, results of all investigations and the pathologist's interpretation of biopsy appearances. Figures 3.181 and 3.182 show nonspecific ulceration; the apparent absence of mucosal inflammation between these lesions makes ulcerative colitis unlikely.



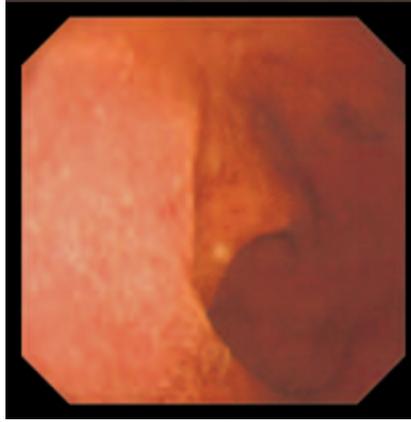
3.181



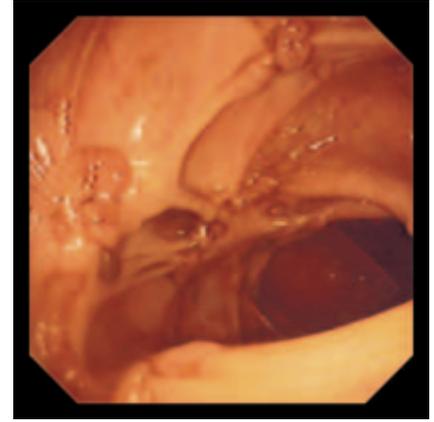
3.182

**Nonspecific nature of endoscopic appearances (cont.)**

Conversely, Fig. 3.183 illustrates generalized mucosal inflammation without discrete ulceration, suggesting ulcerative colitis, though in the event this patient had Crohn's disease. Figure 3.184 shows severe scarring of a type commonly seen in Crohn's disease, though the biopsies only showed nonspecific features.



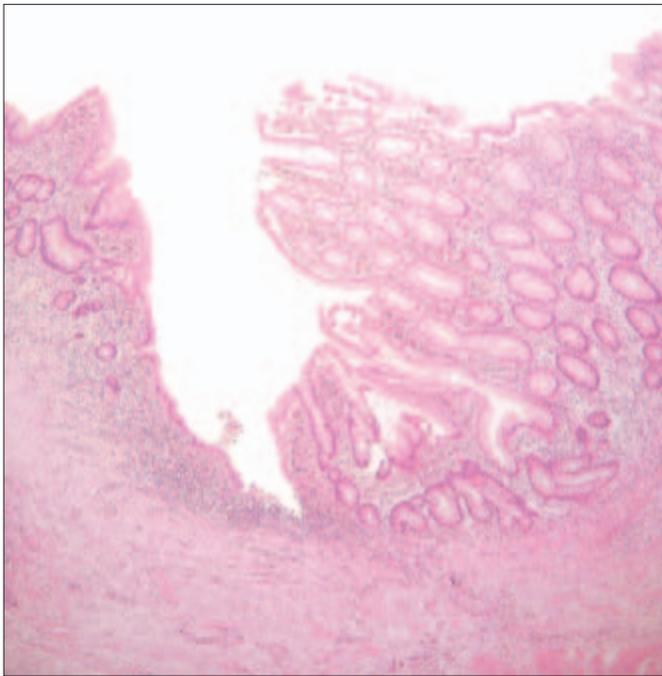
3.183



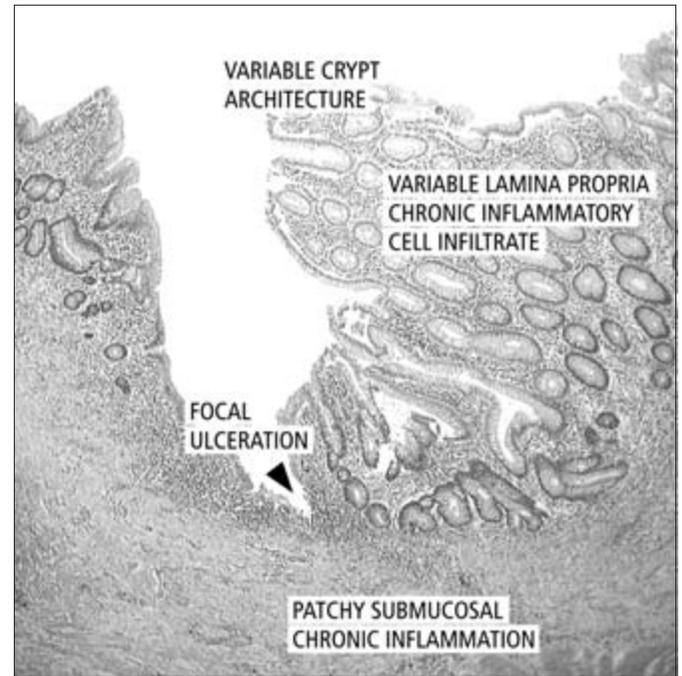
3.184

**Indeterminate colitis**

Sometimes it may be impossible on the basis of clinical features and colonoscopic appearances to decide whether the diagnosis is ulcerative colitis or Crohn's disease. There may be clear histopathological evidence of inflammatory bowel disease but the changes may be too nonspecific for the pathologist to subclassify the inflammatory bowel disease (Fig. 3.185). The majority of patients carrying this label will over a period of time develop clinical, radiological and histological features by which they will be reclassified as having ulcerative colitis or Crohn's disease.

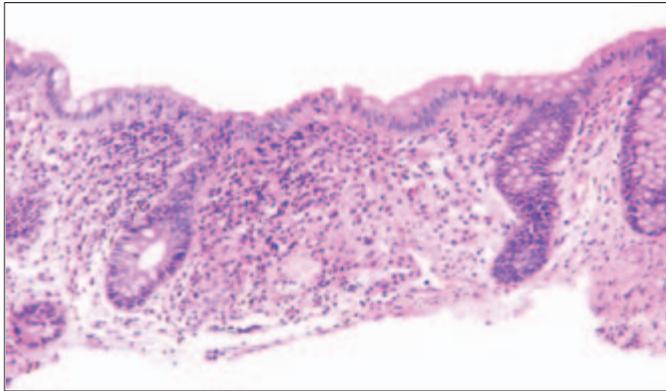


3.185

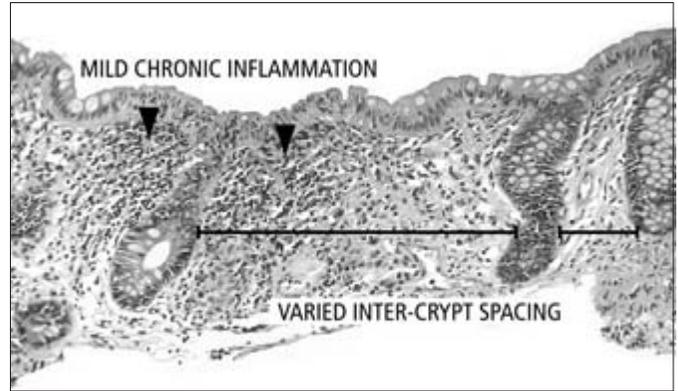


### Minimal change colitis

The occasional patient presenting with symptoms suggestive of inflammatory bowel disease shows no or only minimal evidence of any mucosal abnormality on colonoscopy. When abnormality is seen, it is in the blood vessels which develop a 'leaves on the trees' appearance. Nevertheless biopsy material shows changes of chronic inflammatory bowel disease (Fig. 3.186).



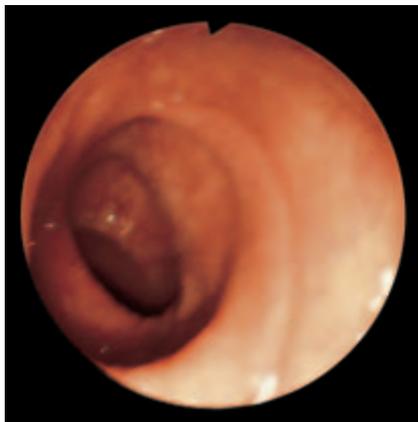
3.186



### Microscopic colitis

Patients with this disorder present, usually in late middle age, with persistent watery diarrhoea but have no evidence of bleeding. In 30% of cases the mucosa shows minor abnormalities, e.g. minimal erythema, oedema and altered vascular pattern, but in the great majority endoscopy is visually normal. The diagnosis rests on biopsy appearances. Microscopic colitis is subclassified according to its histological appearances (Table 3.3).

The mild endoscopic changes which may be seen in collagenous colitis are shown in Fig. 3.187.



3.187

**Table 3.3** Working classification of microscopic colitides compared with minimal change colitis

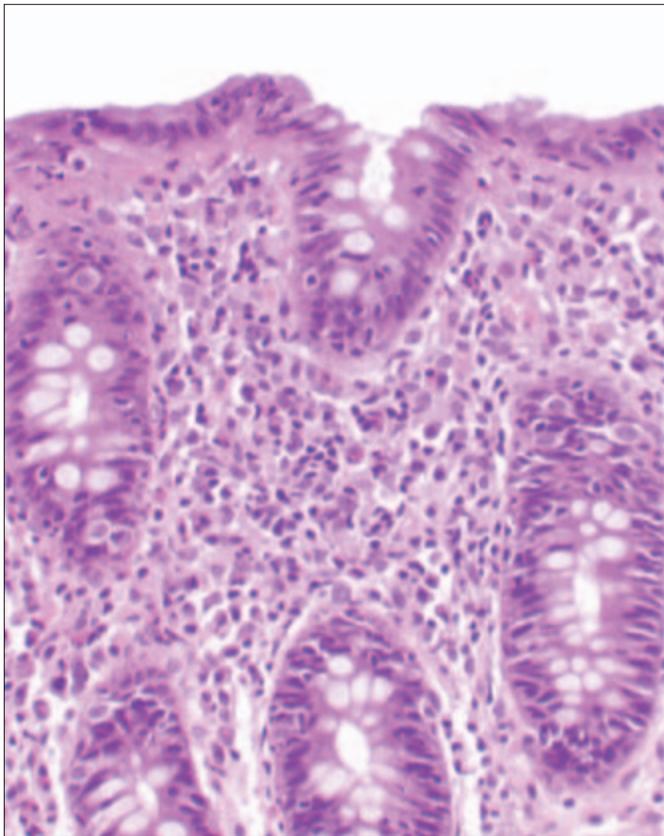
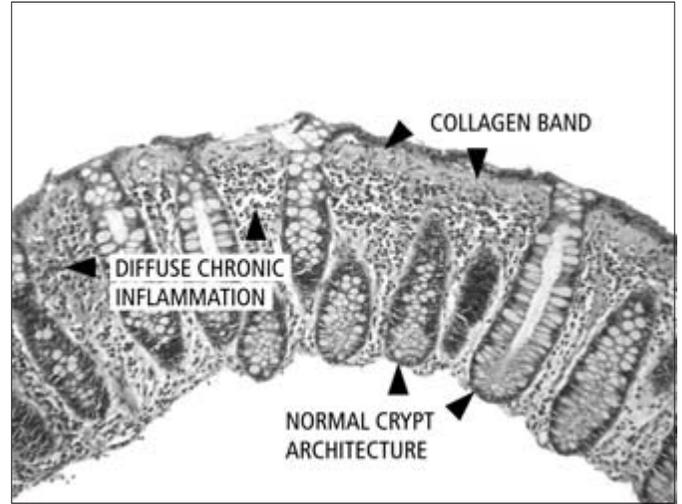
	Minimal change colitis	Collagenous colitis	Lymphocytic colitis	Microscopic colitis not otherwise classified
Colonoscopic appearances	Normal or mildly abnormal	Normal or mildly abnormal	Normal or mildly abnormal	Normal or mildly abnormal
Histopathological appearances				
Crypt architectural distortion	Present	Absent	Absent	Absent
Acute inflammation	Often present and varies with activity	Absent	Absent	Absent
Chronic inflammation	Present	Present	Present	Present
Intraepithelial lymphocytes	Absent	Sometimes present	Increased	Usually absent
Collagen band	Absent	Present	Absent	Absent
Associated conditions	Those of ulcerative colitis	Coeliac disease Lymphocytic gastritis NSAIDs	Coeliac disease Lymphocytic gastritis NSAIDs	Resolving infections Developing IBD, lymphocytic or collagenous colitis NSAIDs

**Microscopic colitis (cont.)**

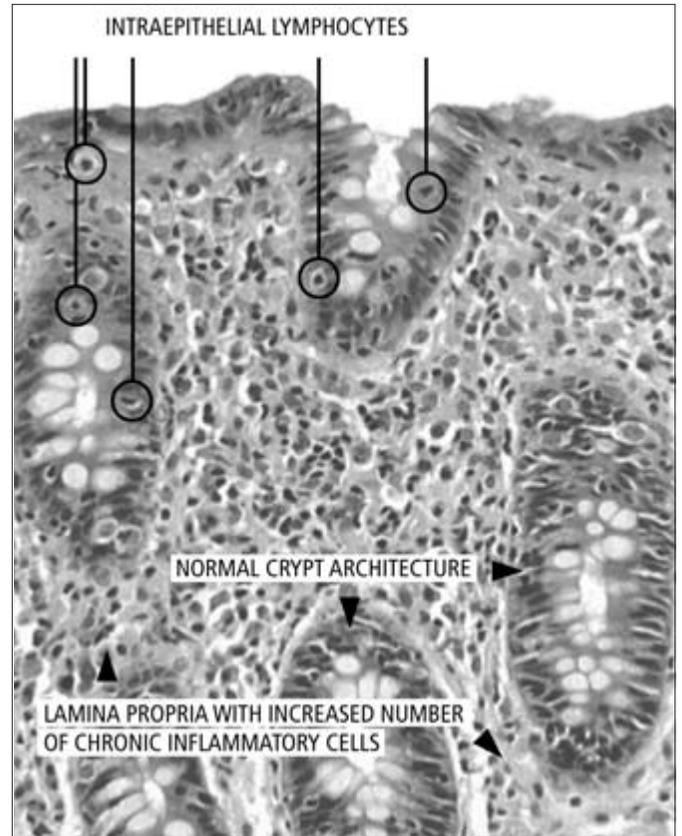
Histological appearances of collagenous colitis are illustrated in Figs 1.22 and 3.188, while lymphocytic colitis appears in Fig. 3.189.



3.188



3.189



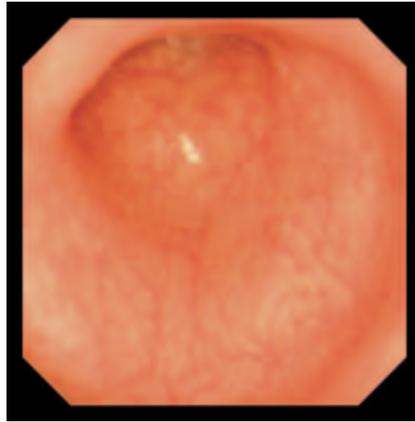
## Ulcerative colitis

### Early active disease

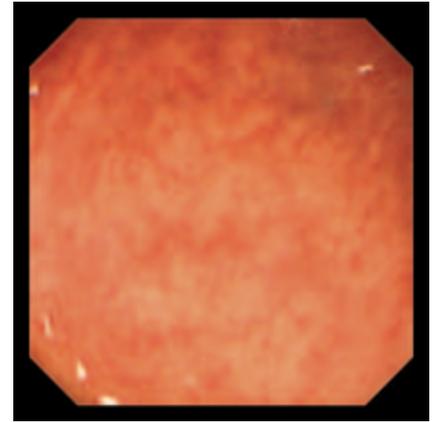
The earliest endoscopic manifestation of ulcerative colitis (UC) is an abnormal vascular pattern. Subtle vascular changes are shown in Fig. 3.190. Figure 3.191 shows a later stage. Diffuse inflammation may cause an opaque granular mucosa with loss of normal vascular pattern, patchy erythema and some mucopus (Fig. 3.192). At this stage radiological appearances are commonly normal.

Granularity of the mucosa (Fig. 3.193) causes multiple light reflections. Small superficial mucosal vessels are no longer visible and mucosal friability is marked. The histological changes of early active UC are shown in Fig. 3.194.

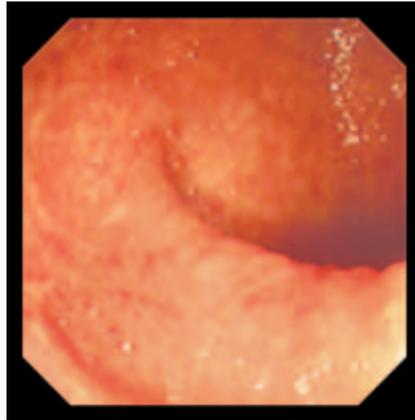
The generalized changes described above are less commonly seen in early Crohn's disease where the lesions are usually patchily distributed. The histology (Fig. 3.227) differs from that seen in early UC (Fig. 3.194).



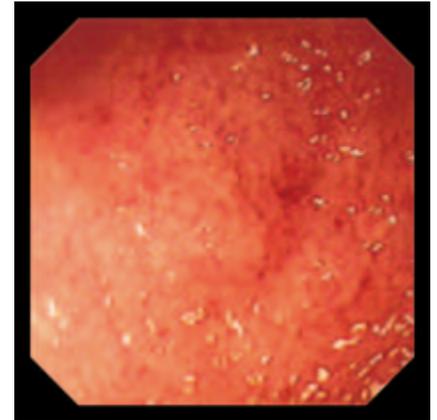
3.190



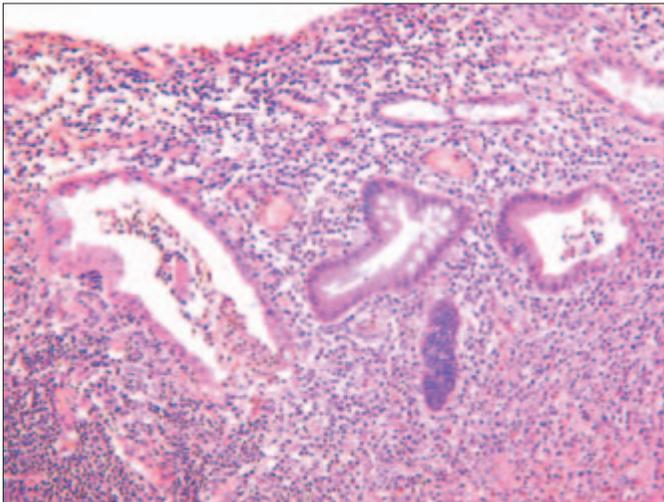
3.191



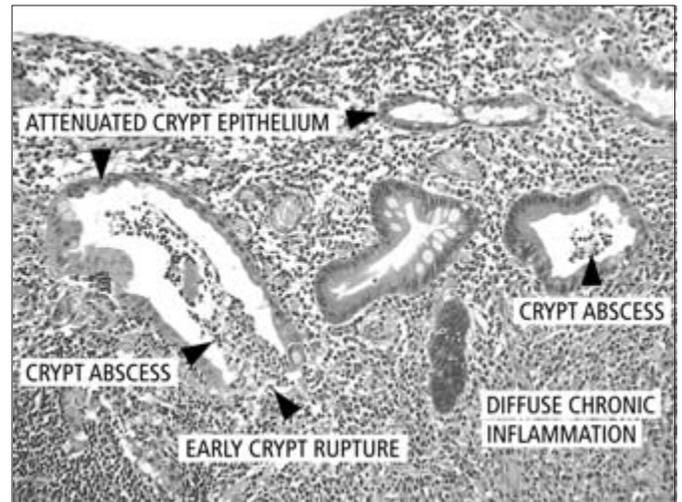
3.192



3.193



3.194

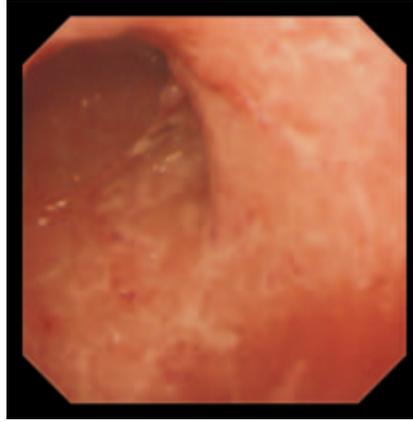


**Ulcerative colitis (cont.)**

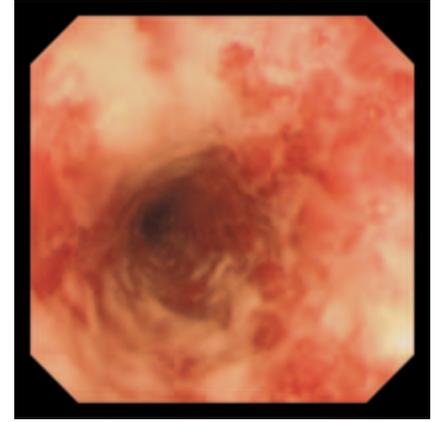
*Advanced and severe active disease*

When ulcerative colitis is more advanced and severe, the inflamed mucosa is often covered with purulent exudate (Figs 3.196 and 3.197).

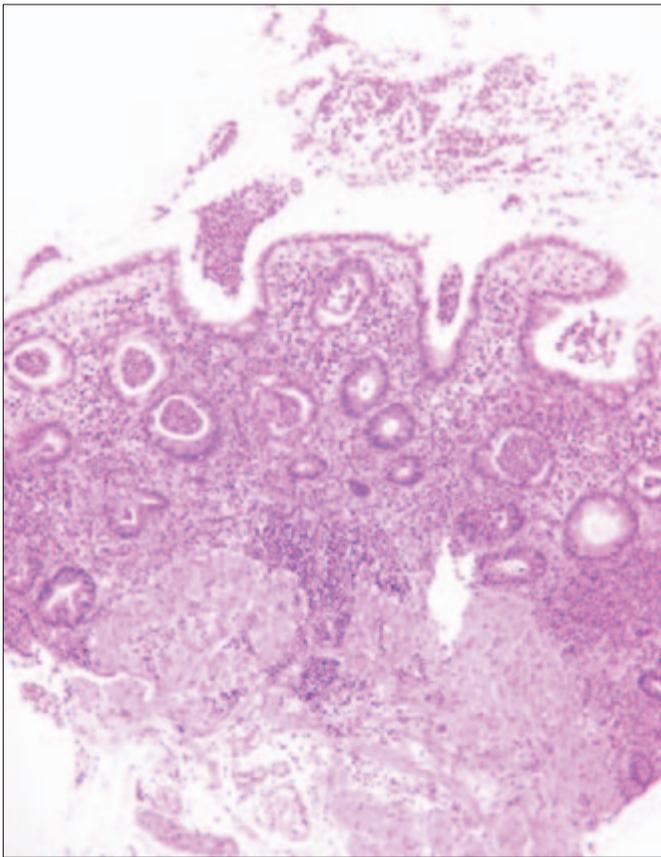
Figure 3.198 illustrates the histology.



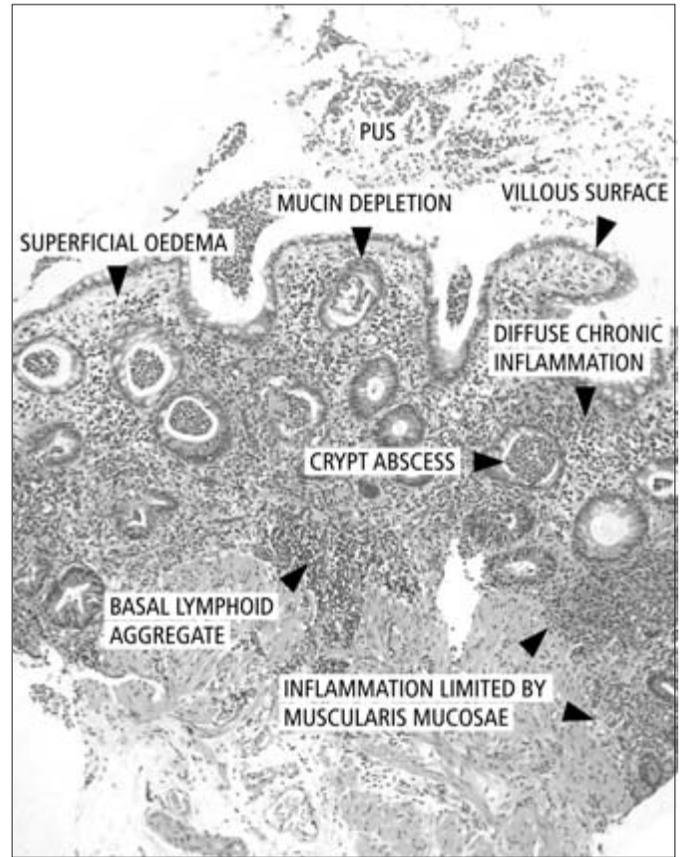
3.196



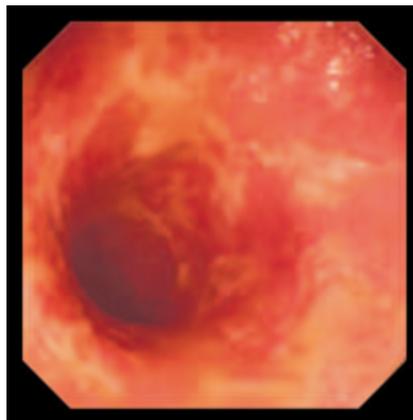
3.197



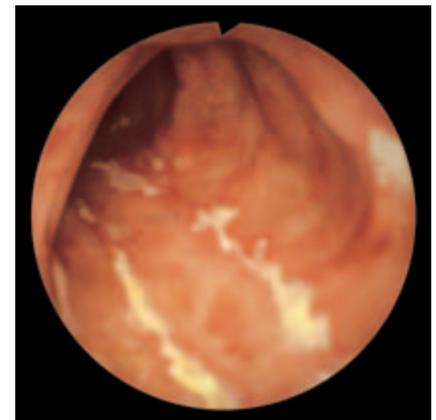
3.198



There may be free blood in the lumen and the mucosa is extremely fragile (Fig. 3.199). Special care must be taken if such appearances are discovered lest perforation of the colon should occur. Typical bear-claw ulcers may be seen (Fig. 3.200). These may also occur in Crohn's disease although other types of ulceration are commoner (Figs 3.239–3.241).



3.199



3.200

**Ulcerative colitis (cont.)**

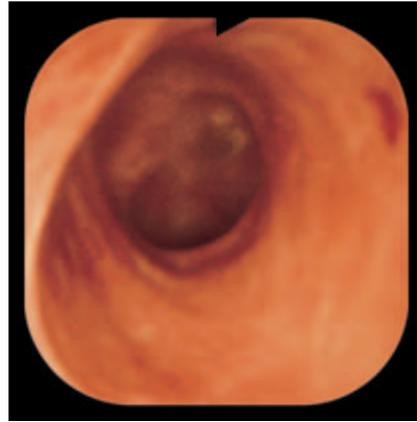
*Chronic ulcerative colitis*

Haustration is commonly lost so that the colon becomes tube-like (Fig. 3.201) and shortened. Consequently colonoscopy is often relatively simple. The caecal mucosa becomes flattened and the ileocaecal valve is often gaping and incompetent (Fig. 3.202). The mucosa in both these cases shows diffuse inflammation but without surface exudate or free bleeding; mucosal friability persists.

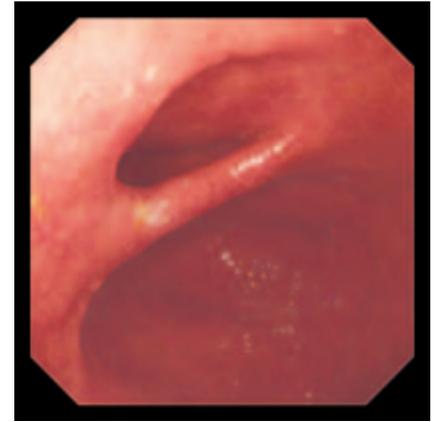
The histological changes in severe chronic UC appear in Fig. 3.203.

In Fig. 3.204 the tube-like appearance of the colon is well shown in addition to which there is an extensive but discontinuous purulent exudate. In Fig. 3.205 the exudate lies on a scarred mucosa.

Figure 3.206 illustrates an unusual mucosal appearance in chronic UC, reminiscent of the 'cerebral' pattern often seen in tubulovillous adenomas (Fig. 3.88).



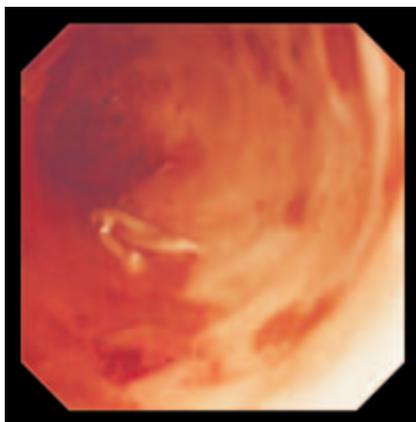
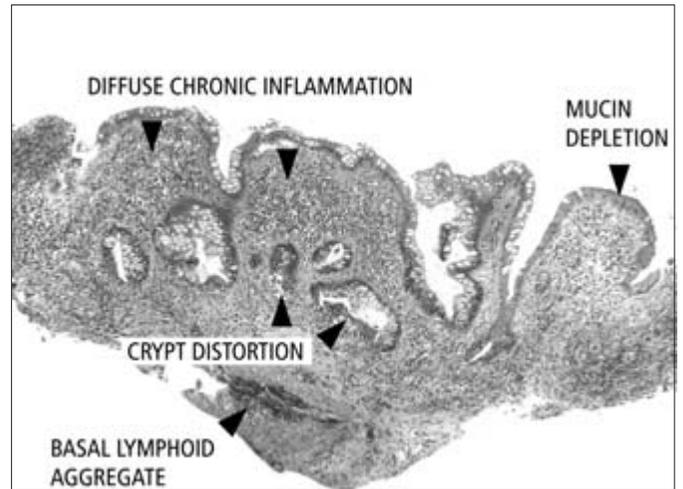
3.201



3.202



3.203



3.204



3.205



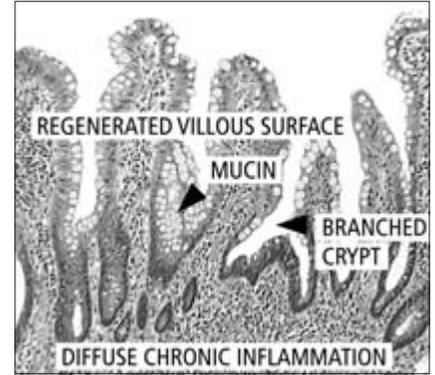
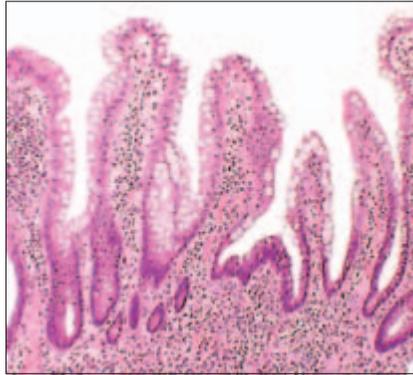
3.206

**Ulcerative colitis (cont.)**

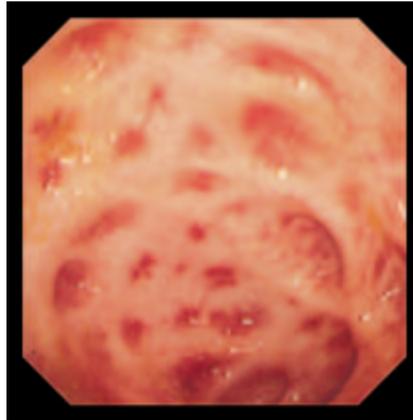
*Inactive or burnt-out colitis*

When an attack of colitis settles the mucosa may regain a normal endoscopic appearance although microscopic abnormalities usually persist (Fig. 3.207). Residual scarring may remain. Figure 3.208 shows white scarring with pink regenerative mucosa and an abnormal vascular pattern.

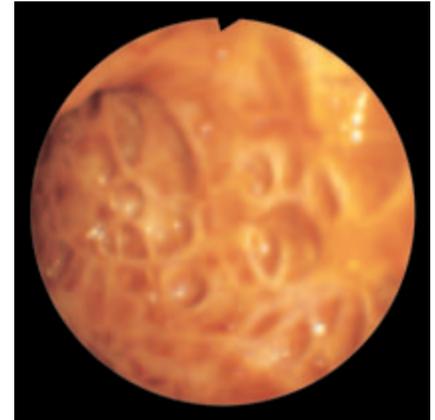
The pattern of the severe scarring seen in Fig. 3.209 is unusual.



3.207



3.208

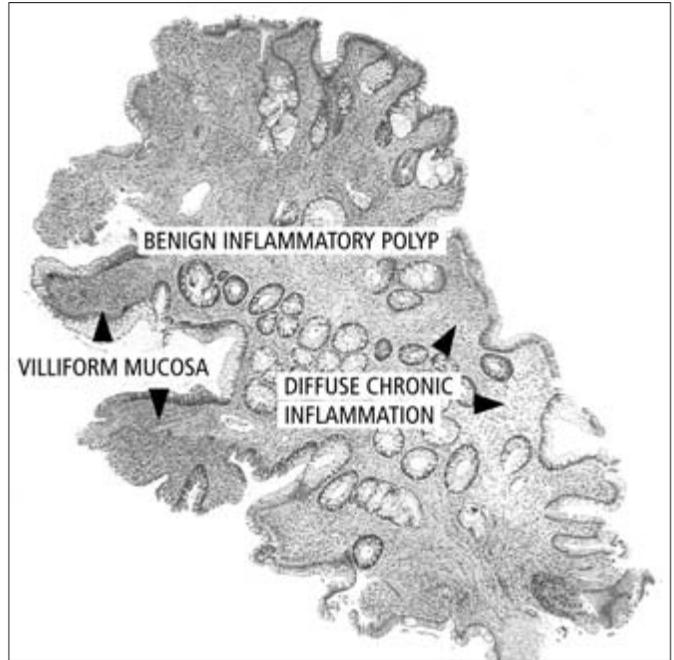
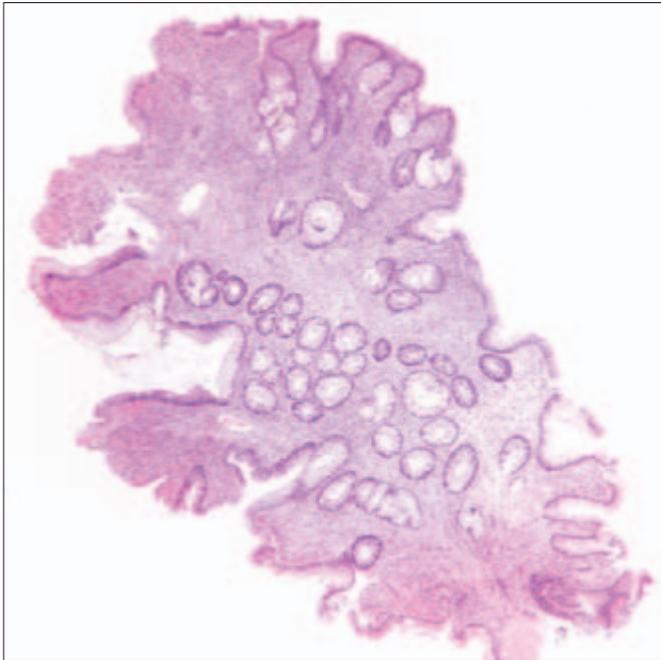


3.209

**Ulcerative colitis (cont.)**

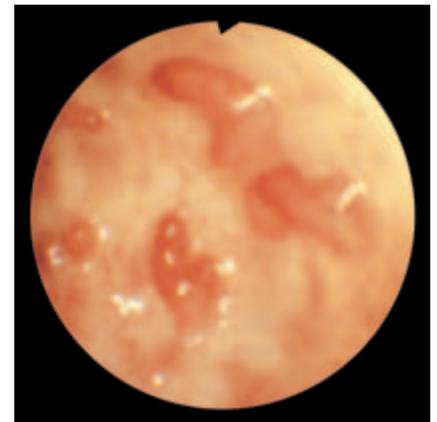
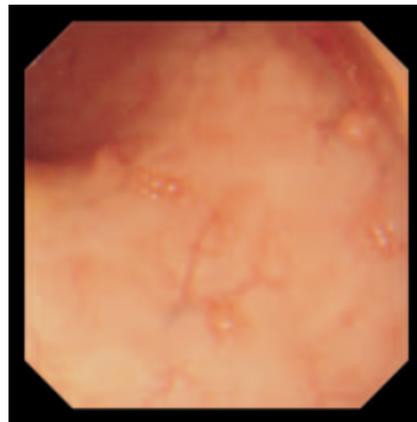
*Inflammatory polyps (pseudopolyposis)*

Pseudopolyposis is a term hallowed by usage. It has, however, been suggested that there is nothing ‘pseudo’ about these polyps which should, more appropriately, be referred to as benign inflammatory polyps (Fig. 3.210) or regenerative polyps, depending on the histopathological appearances.



3.210

Figure 3.211 illustrates a number of these pink lesions. Note the pale scarred mucosa and abnormal vascular pattern. The finger-like lesions in Fig. 3.212 are less uniform in colour. The polyps demonstrated in these two figures are mainly mucosal.



3.211

3.212

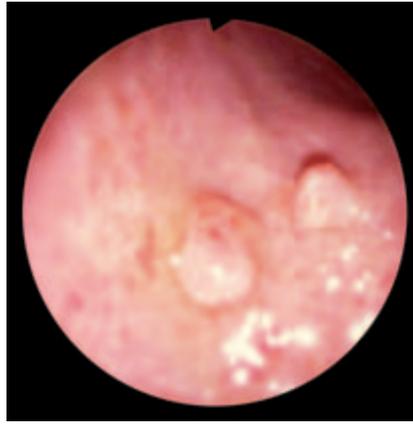
**Ulcerative colitis (cont.)**

*Inflammatory polyps (pseudopolyps) (cont.)*

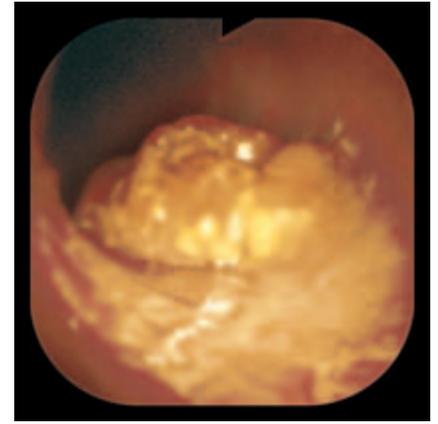
By contrast Figs 3.213–3.215 show polyps made up primarily of granulation tissue. These when contiguous may fuse causing irregular masses (Fig. 3.215) and, on healing, occasional mucosal bridges (Fig. 3.216; see also Fig. 3.253).

Biopsy or polypectomy of lesions greater than 1 cm in diameter or different in appearance or colour from their fellows is advisable to enable definition of polyp type and for exclusion of malignancy.

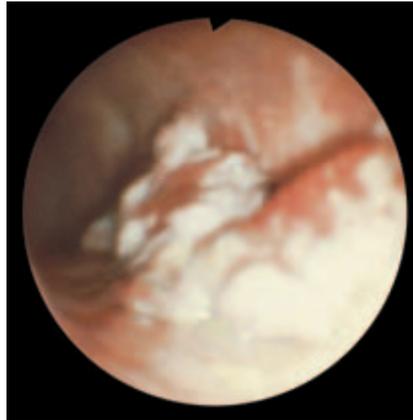
Adenomatous polyps may occasionally be seen in patients with UC, though there is no aetiological relationship.



3.213



3.214



3.215



3.216

**Strictures**

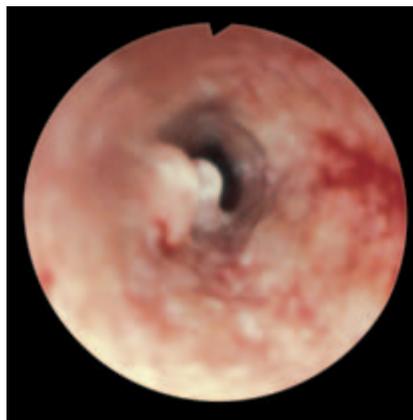
Chronic ulcerative colitis can be complicated by strictures which may be malignant. Colonoscopy is essential to determine the nature of such strictures which will, however, in most cases be benign. For diagnosis, biopsy whenever possible and, failing this, brush cytology through the stenosis is mandatory. Figure 3.217 shows a benign fibromuscular stricture in quiescent colitis and Fig. 3.218 some inflammatory narrowing in active colitis. The stricture in Fig. 3.219 is associated with inflammatory pseudopolyps which simulate carcinoma. The lesion in Fig. 3.220 is a true carcinomatous stricture at the hepatic flexure in a patient with chronic ulcerative colitis.



3.217



3.218



3.219



3.220

**Ulcerative colitis (cont.)**

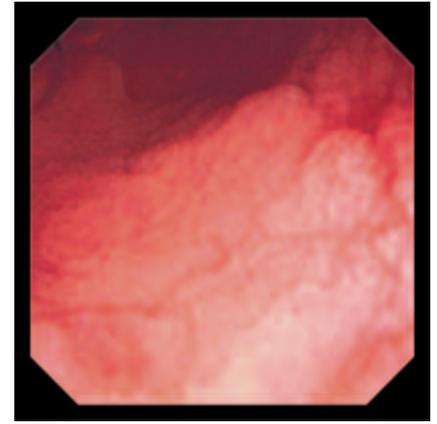
*Dysplasia-associated lesion or mass (DALM)*

A critique of surveillance programmes is beyond the scope of this atlas. Suffice it to say that random biopsies may reveal the changes of low grade or high grade dysplasia. The colonoscopist cannot usually distinguish flat dysplastic from normal mucosa, and the diagnosis therefore depends on the histopathological appearances. Dysplastic mucosa may however, be endoscopically visible as a DALM. Figure 3.221 shows the typical colonoscopic appearances which are better seen in the same patient using the underwater technique (Fig. 3.222). Another similar lesion, mildly traumatized by the colonoscope, appears in Fig. 3.223. Figure 3.224 shows the histopathology of a DALM.

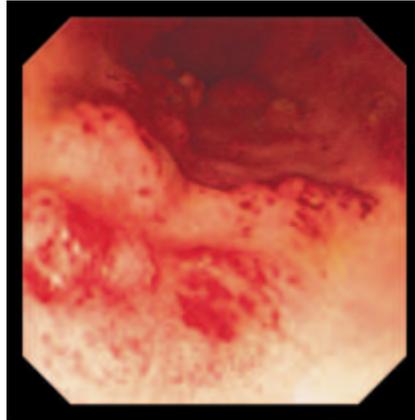
Older patients with ulcerative colitis may develop adenomas in the left colon (see also p. 279). These may be difficult to distinguish from DALMs. The distinction is helped by taking biopsies from the surrounding flat mucosa to look for dysplasia elsewhere. This important distinction necessitates the endoscopist and pathologist working closely together to decide between polypectomy and total colectomy.



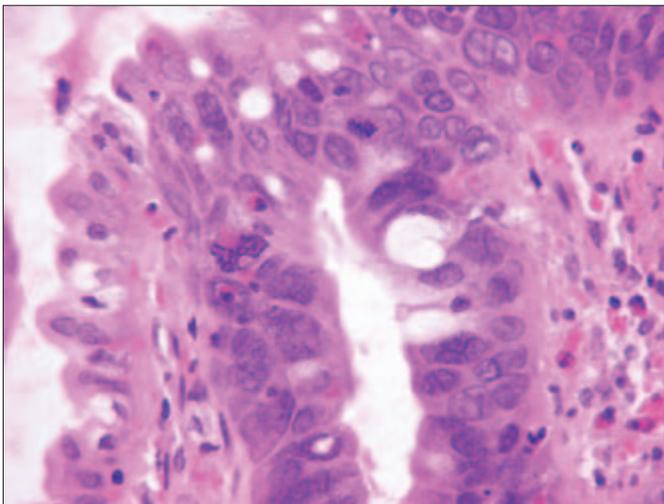
3.221



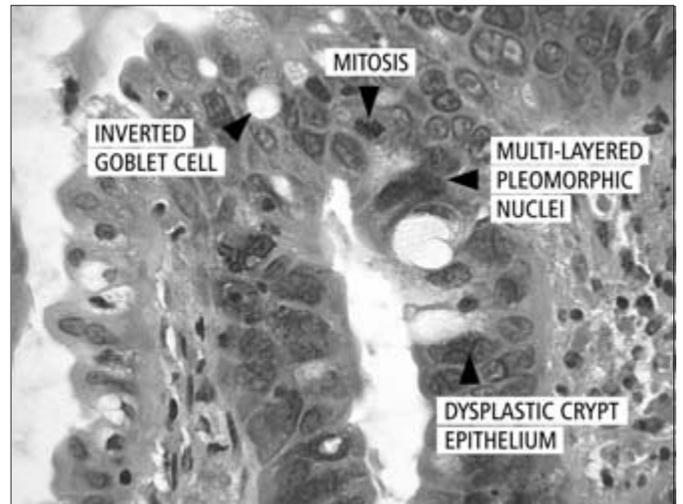
3.222



3.223



3.224

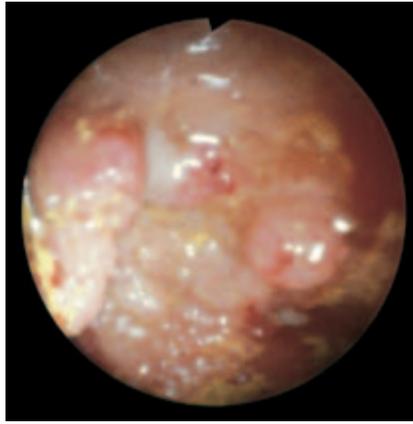


### Ulcerative colitis (*cont.*)

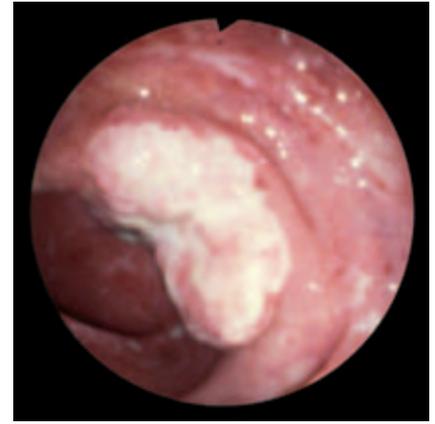
#### *Carcinoma*

Longstanding extensive UC is associated with a significantly increased risk of developing colon cancer. Colonoscopic surveillance is increasingly used in this group of patients.

Figure 3.158 shows a flat villous adenocarcinoma in longstanding UC, whilst the ulcerated lesion in Fig. 3.159 occurred in a patient with previously unrecognized chronic total ulcerative colitis. Carcinoma in UC is not infrequently multifocal. Such lesions may be synchronous or metachronous.



3.158



3.159

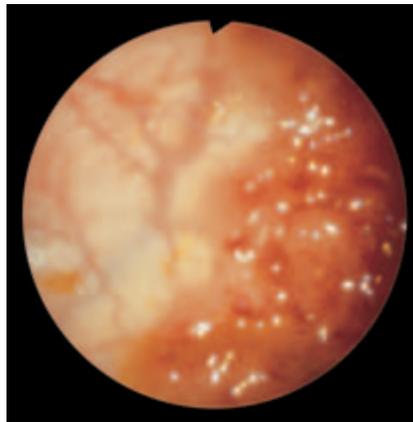
### Crohn's disease

The endoscopic appearances of the main types of inflammatory bowel disease may be similar or indeed indistinguishable, a problem referred to earlier. With increasing experience the endoscopist learns the strengths and limitations of colonoscopy in these common disorders.

#### *Segmental changes*

Ulcerative colitis is commonly confined to rectum or distal colon, and the change from diseased bowel to normal appearance proximally is quite abrupt, as seen by the colonoscopist, though to the histopathologist the transition may be slower. In Crohn's disease the bowel may be affected in several quite separate segments.

Figure 3.225 illustrates the sudden change from chronically inflamed mucosa with a polypoid appearance to normal looking mucosa. This is also shown in Fig. 3.226; this patient had several other so-called skip lesions.



3.225



3.226

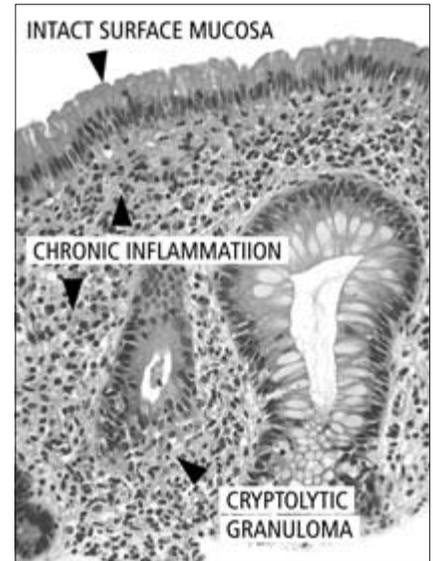
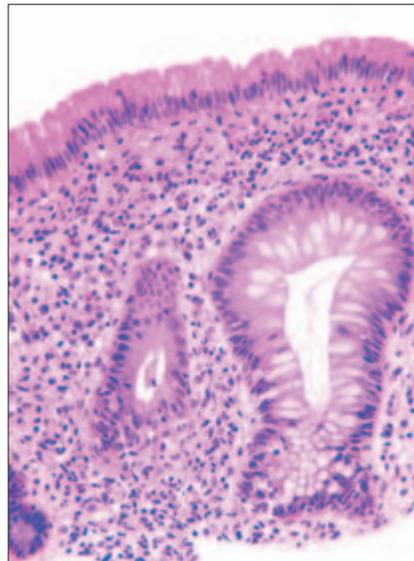
**Crohn's disease (cont.)**

*Early active disease*

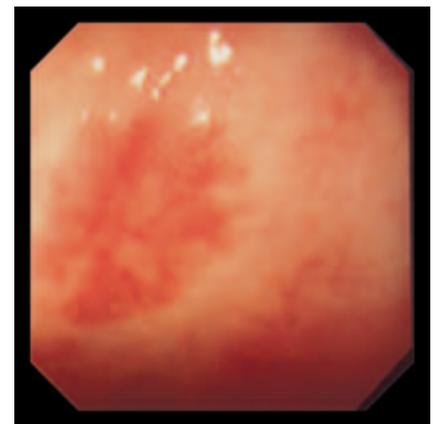
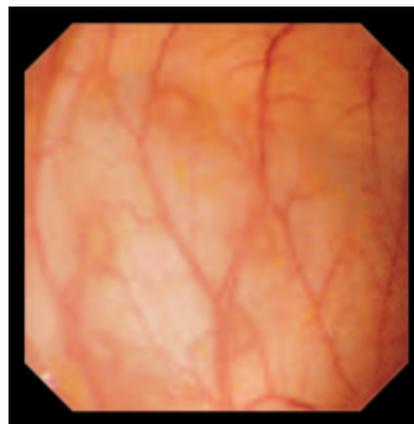
In contrast to ulcerative colitis where changes are diffuse, early lesions of Crohn's disease are discrete, often occurring within an otherwise normal mucosa. Figure 3.227 shows the histopathology of early Crohn's disease.

The earliest endoscopic change is the presence of small oedematous areas obscuring the normal vascular pattern (Fig. 3.228). Small non-ulcerated hyperaemic spots are occasionally encountered, as illustrated in Figs 3.229 and 3.230.

The best known endoscopic finding in early active disease is the aphthoid ulcer shown in Figs 3.231–3.234. These lesions have a wide differential diagnosis besides Crohn's disease, including NSAIDs (Fig. 3.312), amoebiasis (Fig. 3.327), herpes, Behçet's disease, infection with *Yersinia* (Fig. 3.267), and tuberculosis. In addition to biopsy, culture and serological tests may be required in some cases.

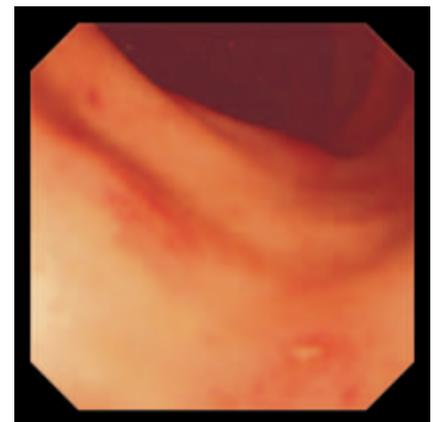
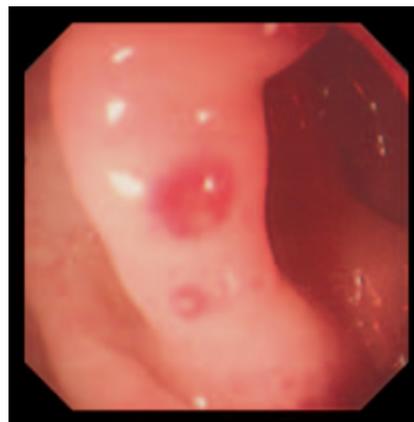


3.227



3.228

3.229



3.230

3.231

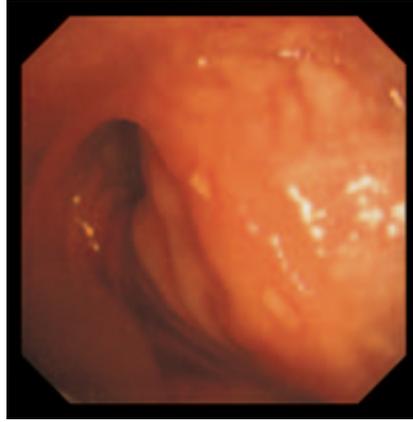
**Crohn's disease (cont.)**

*Early active disease (cont.)*

In Figs 3.231 and 3.232 there are scattered small ulcers, each surrounded by an hyperaemic flare. The aphthoid ulcer in Fig. 3.233 is situated at the apex of a raised lesion, a less common variant. The multiple ulcers shown in Fig. 3.234 lie mainly on the haustral folds; this siting is common in Crohn's disease and may also be apparent in the small bowel (see Chapters 2 and 5).

The histology of aphthoid ulceration is shown in Fig. 3.235.

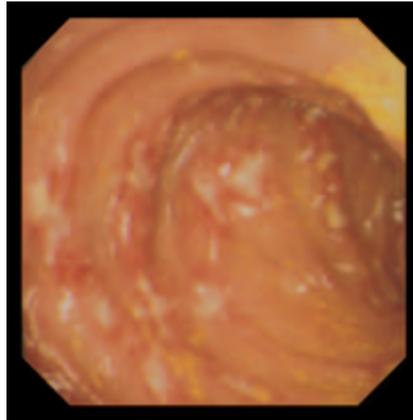
While discrete ulcers are typical of early Crohn's disease, they are also found adjacent to severe active disease.



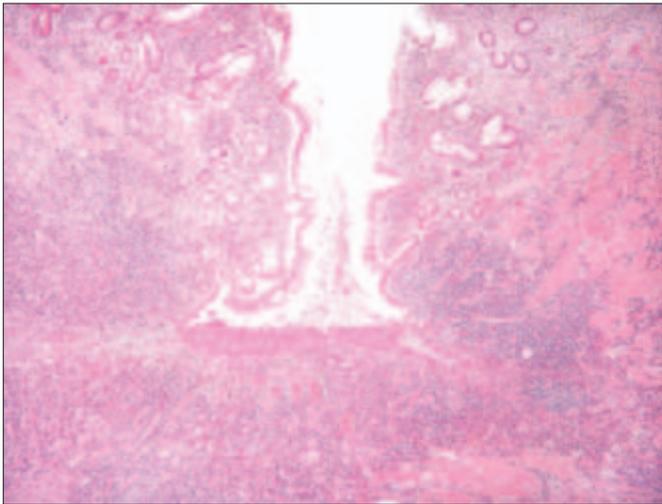
3.232



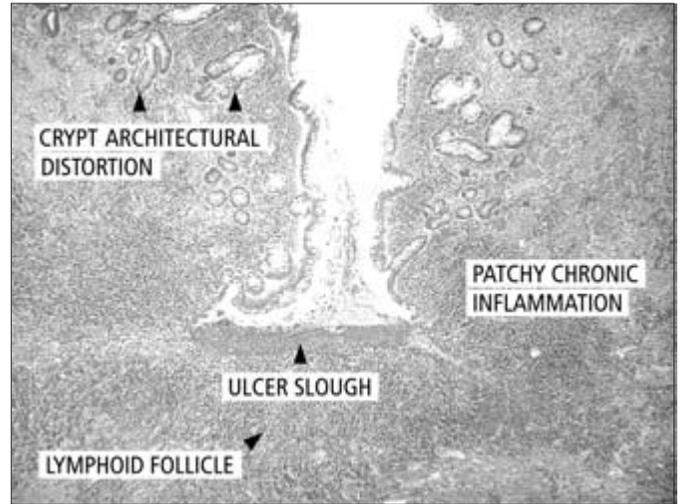
3.233



3.234



3.235

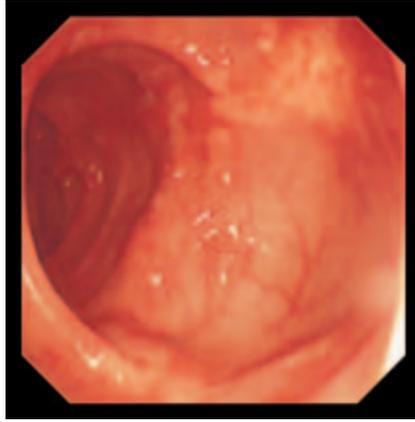


**Crohn's disease (cont.)**

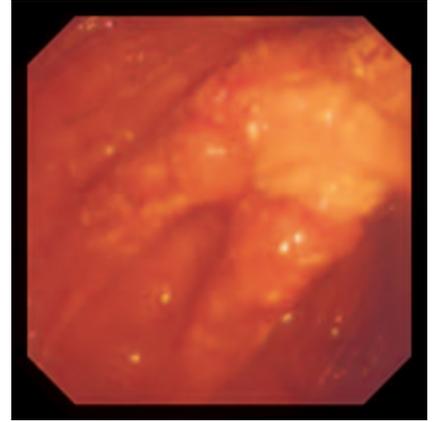
*Advanced and severe disease*

The extensive superficial ulceration seen in Figs 3.236 and 3.237 contrasts with the preceding illustrations. The ulcers in Fig. 3.238 are superficial, irregular and nearly confluent.

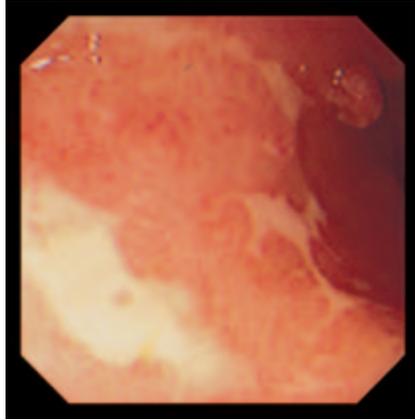
The appearances in Fig. 3.239 are typical of Crohn's disease. The ulcer runs longitudinally, it is deeper than aphthoid ulcers, and the edges are raised. The ulcer shown in Fig. 3.240 is deep, irregular and with some undermining of the surrounding mucosa, but is more transverse than longitudinal. Figure 3.241 illustrates extensive, near circumferential ulceration.



3.236



3.237



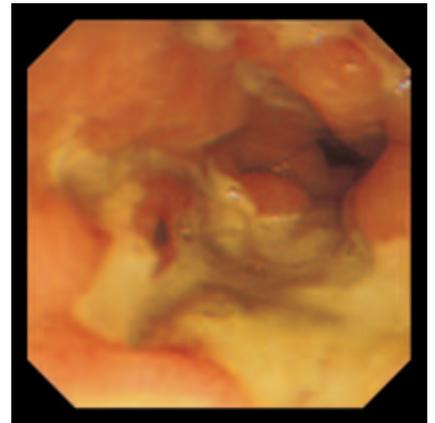
3.238



3.239



3.240



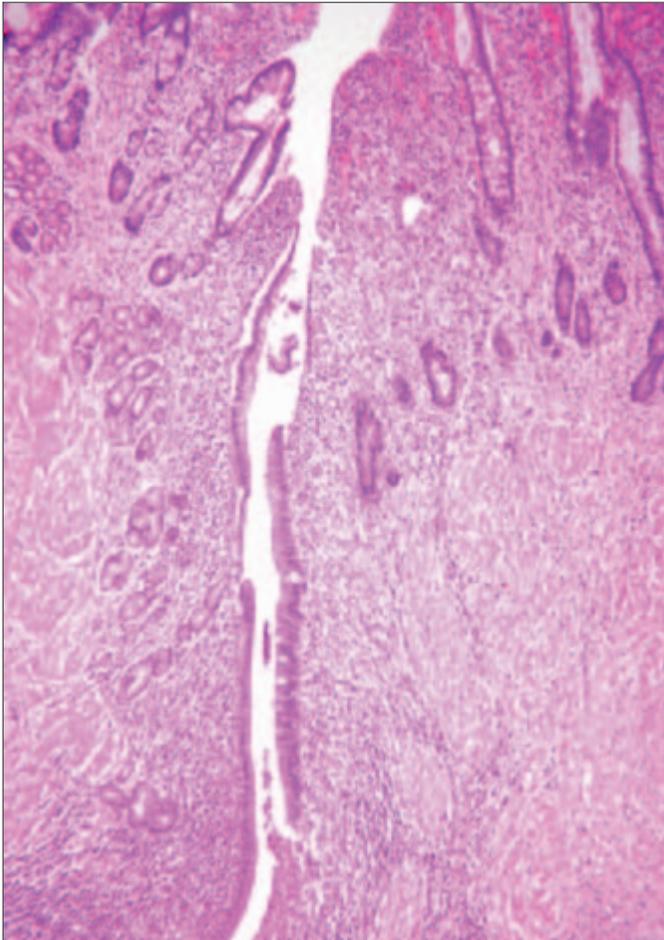
3.241

**Crohn's disease (cont.)**

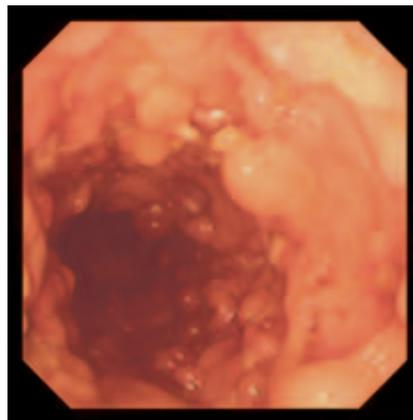
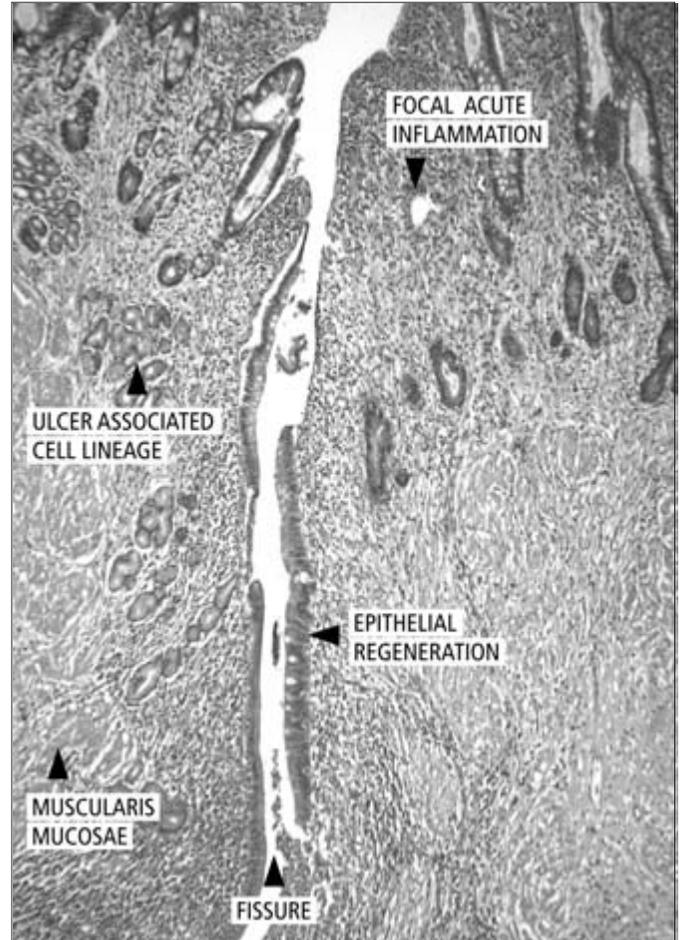
*Advanced and severe disease (cont.)*

Figure 3.242 shows the histopathological changes of advanced Crohn's disease. There is a fissure with adjacent disordered mucosal architecture and disordered muscularis mucosae. Within the mucosa there are islands of ulcer associated cell lineage. This was previously known as pyloric or pseudopyloric metaplasia. It is found adjacent to ulcers or at sites of healed ulcers in the gut, and is not specific for Crohn's disease. The muscularis mucosae is grossly thickened and blends to a muscularized submucosa.

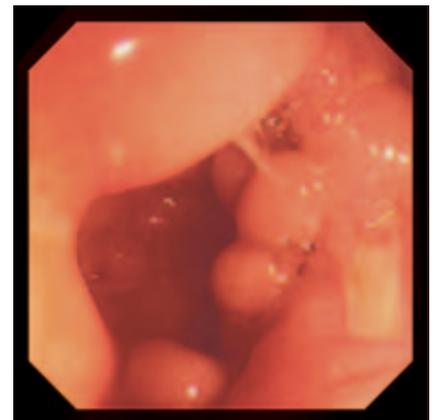
The cobblestone-like appearance, wherever it may occur (also seen in Figs 2.277 and 2.560) is almost diagnostic of Crohn's disease. Figure 3.243 shows typical colonic cobblestoning, which may be associated with persistent ulceration (Fig. 3.244).



3.242



3.243

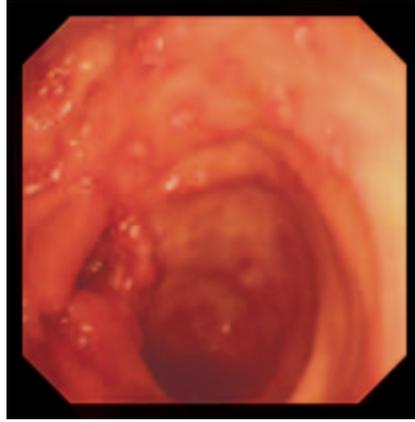


3.244

**Crohn's disease (cont.)**

*Advanced and severe disease (cont.)*

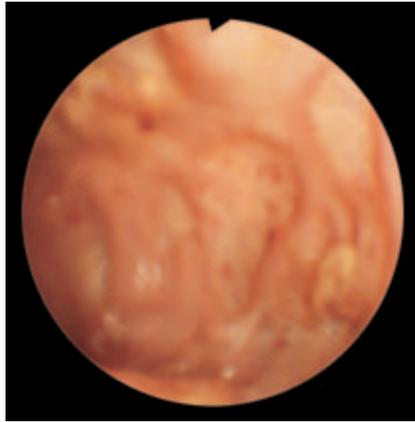
In Fig. 3.245 there are many scattered aphthoid ulcers. In addition the mucosal folds of the ileocaecal valve are inflamed and oedematous, leading to a fixed gaping of the valve. These appearances are clearly related to cobblestoning.



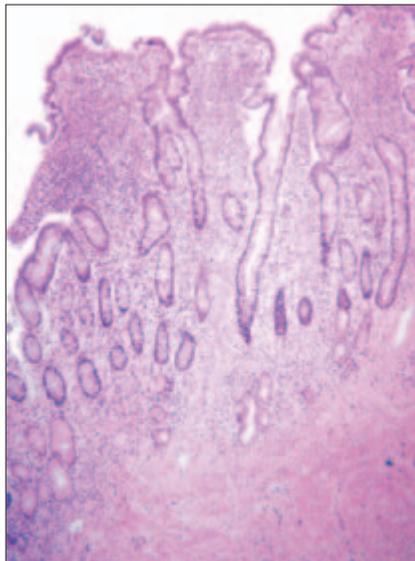
3.245

*Chronic disease*

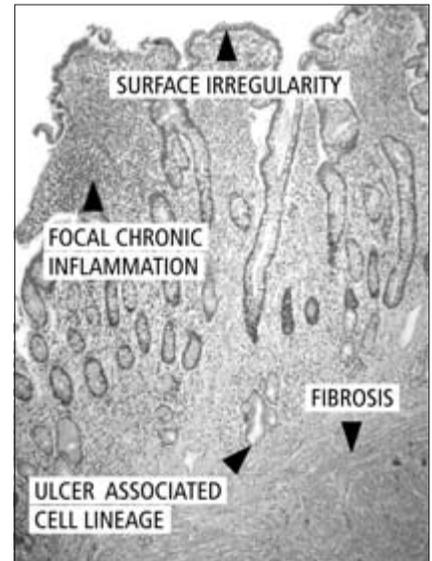
There are no clear features differentiating acute from chronic or burnt-out disease; rather, one stage merges into the next. Figure 3.246 shows a grossly irregular mucosa with evidence of deep but re-epithelialized ulcers, and some residual active ulceration. Figure 3.247 shows the histopathology.



3.246



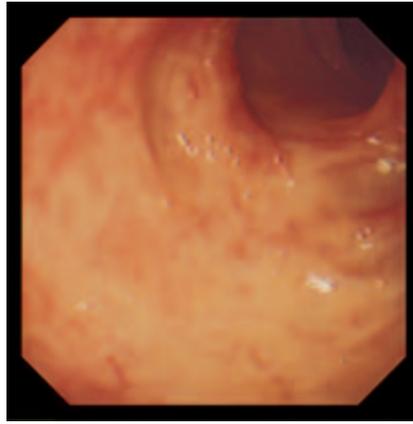
3.247



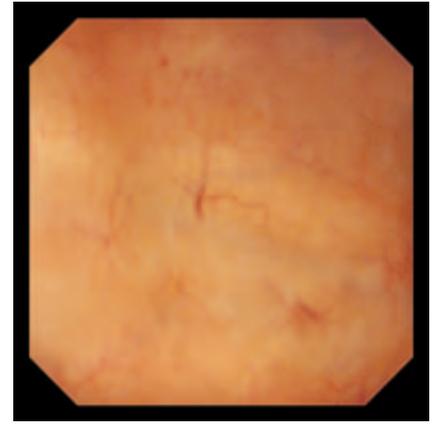
**Crohn's disease (cont.)**

*Inactive or burnt-out disease*

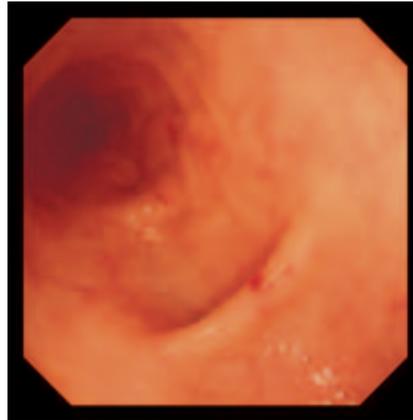
In both these figures (Figs 3.248 and 3.249) the mucosa appears smooth, white and scarred with occasional abnormal blood vessels. Non-specific red spots may be apparent (Fig. 3.250). Scarring may be non-uniform (Fig. 3.251) and may leave a reticular pattern (Fig. 3.252); there are also some inflammatory polyps. The healing process may lead to the formation of mucosal bridges (Fig. 3.253). Similar appearances can be seen in inactive burnt-out UC as illustrated in Fig. 3.216.



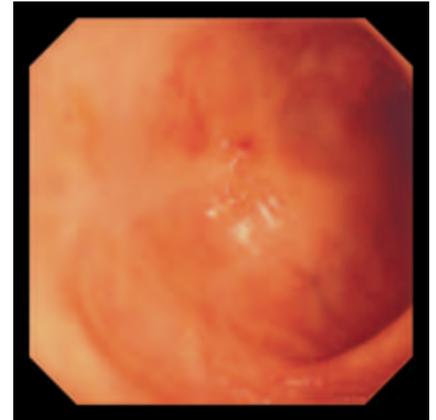
3.248



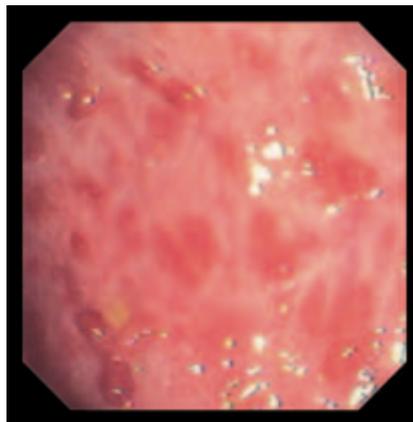
3.249



3.250



3.251



3.252

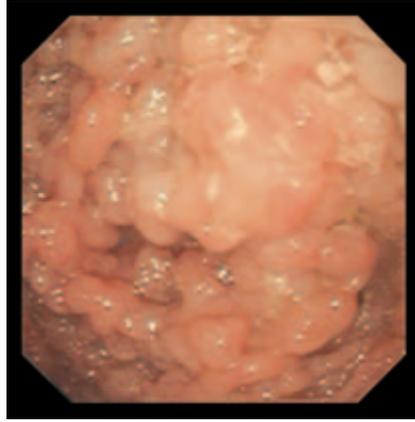


3.253

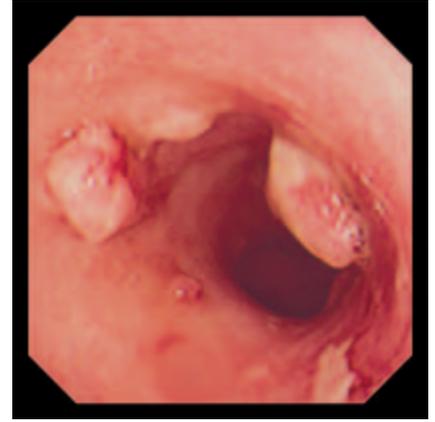
**Crohn's disease (cont.)**

*Inflammatory polyps*

In contrast to the relatively uniform appearance of a cobblestone mucosa, inflammatory polyps may be very variable in size and shape (Fig. 3.254). Figure 3.255 shows multiple small polyps. Occasionally inflammatory polyps are large, with a thick stalk (Fig. 3.256). When appearances are atypical removal and pathological examination is advisable. Typical histology appears in Fig. 3.257.



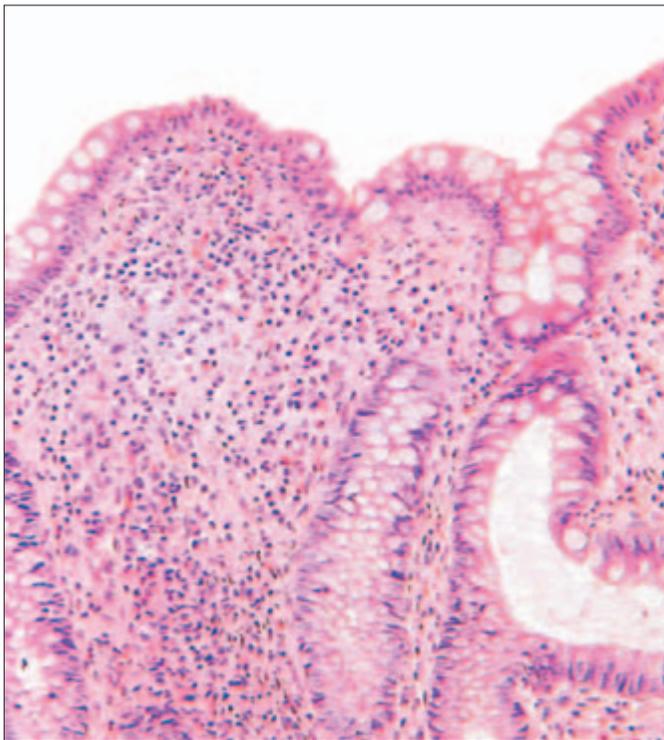
3.254



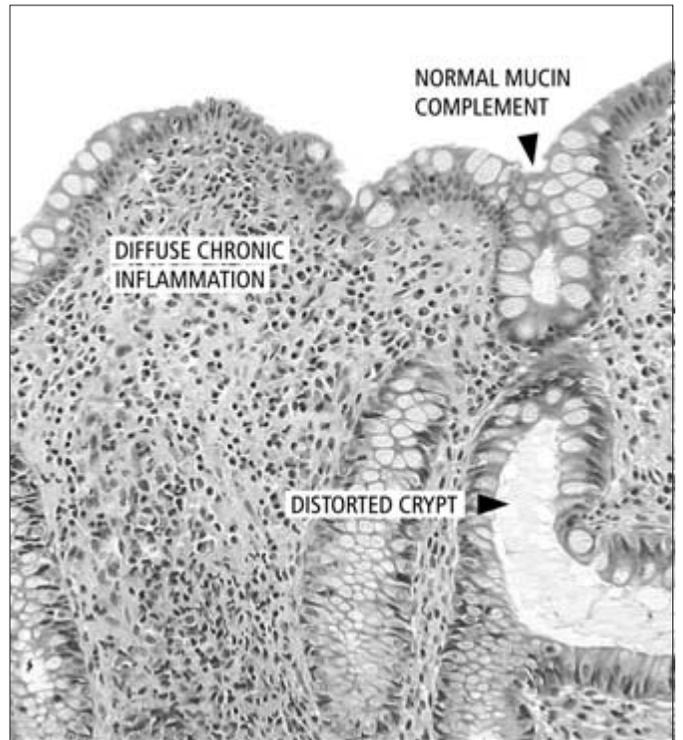
3.255



3.256



3.257

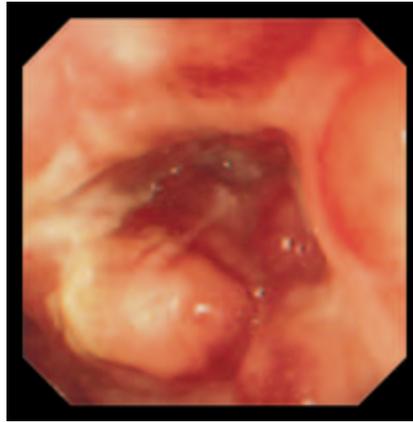


## Crohn's disease (cont.)

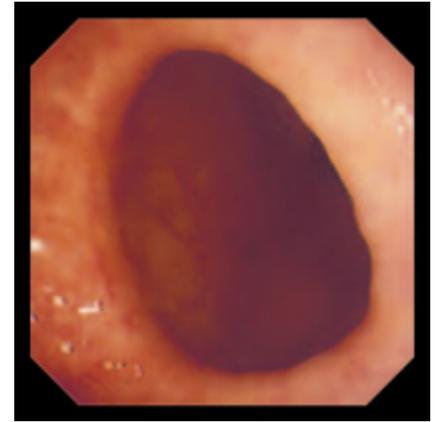
### Strictures

Strictures in Crohn's disease may be single, multiple, short or long. Figure 3.258 shows a stricture in the rectum with evidence of persistent active disease. Rarely, Crohn's disease may be complicated by carcinoma; rectal Crohn's disease creates a higher risk.

The stricture illustrated in Fig. 3.259 is regular, almost circular and was found in a patient whose disease activity settled some years before and who at the time of this examination had shown no more than some mucosal scarring and two non-obstructing almost round strictures in the descending colon.



3.258

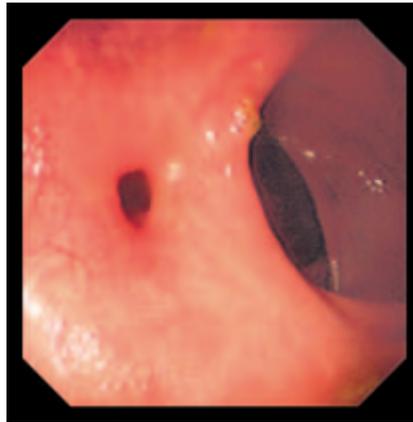


3.259

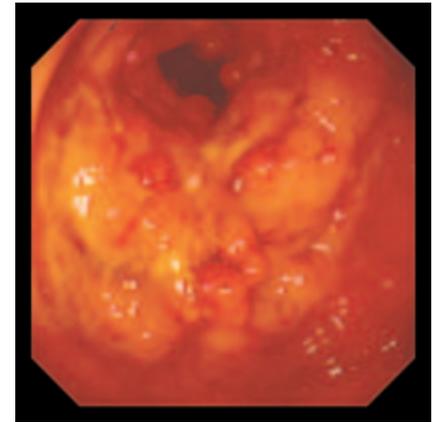
### Fistulae

Fistulae commonly complicate Crohn's disease. Their clinical significance is variable and depends upon the location. The mere finding of a fistula does not necessarily mean that any further action need be taken.

Figure 3.260 shows a small enterocolic fistula with little colonoscopic evidence of active disease. No fistula was visible when the colonoscopic abnormalities demonstrated in Fig. 3.261 were discovered. Biopsies were reported as showing nonspecific inflammatory changes only. Further investigation by radiology showed this to be the site of an enterocolic fistula with active Crohn's disease in the loop of small bowel which had fistulated into the colon.



3.260



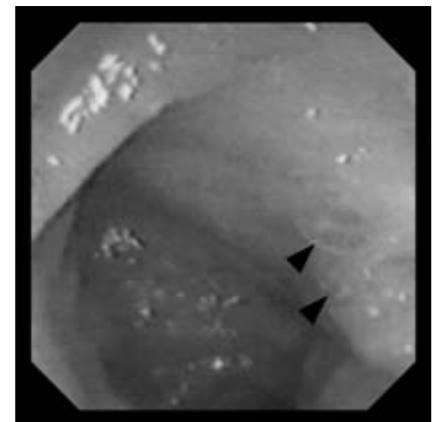
3.261

### Ileal Crohn's disease

The ileum is the commonest site of Crohn's disease. The terminal ileum may be inspected during colonoscopy and occasionally during enteroscopy (see Chapter 5). Figure 3.262 shows an ulcer of the terminal ileum in a patient with colonic Crohn's disease when the examination was extended to include limited ileoscopy.



3.262



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## Infections and infestations

### Acute bacillary dysentery

### Amoebic colitis

### Bilharzia

### Tuberculosis

### Cytomegalovirus colitis

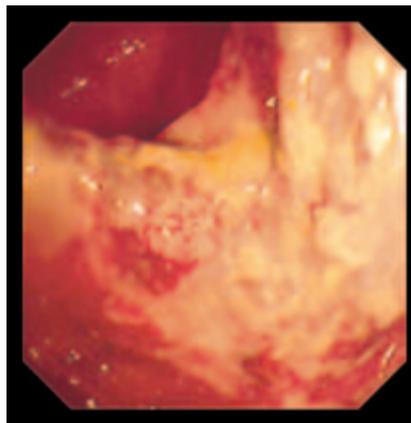
### Threadworms and tapeworms

Acute infections of the lower gastrointestinal tract commonly cause minor nonspecific mucosal abnormalities, and less frequently appearances suggestive of inflammatory bowel disease. Diagnosis is usually made by microbiological examination of faeces. Most episodes of acute bacillary infections of the lower bowel are either self-limiting or respond to antimicrobial chemotherapy. Rigid proctosigmoidoscopy may be performed to obtain biopsies but colonoscopic examination is rarely undertaken as it has little to offer. Occasionally, colonoscopy is performed when symptoms are slow to resolve or when a firm diagnosis has not been made. However, with the recognition of the 'gay bowel syndrome' and the rising incidence of AIDS the indications for colonoscopy in infections of the large bowel are broadening.

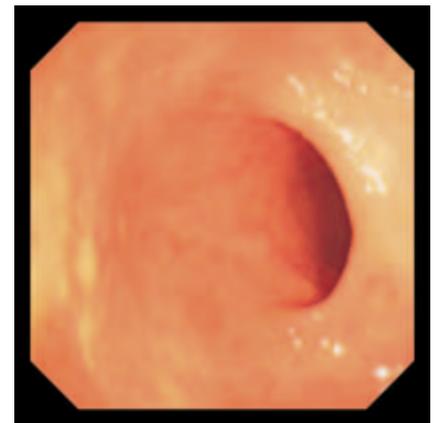
Chronic infections of the colon commonly mimic inflammatory bowel disease. Biopsy specimens may yield diagnostic information, and in addition microbiological examination of material taken at colonoscopy can be helpful.

### Acute bacillary dysentery

This patient, with no previous history of bowel problems, was investigated for severe bloody diarrhoea. Although the endoscopic appearances (Fig. 3.263) were suggestive of inflammatory bowel disease, stool cultures confirmed infection with *Salmonella*. The ileum (Fig. 3.264) appeared normal. Figure 3.265 shows similar appearances in a patient with *Klebsiella* infection.



3.263



3.264



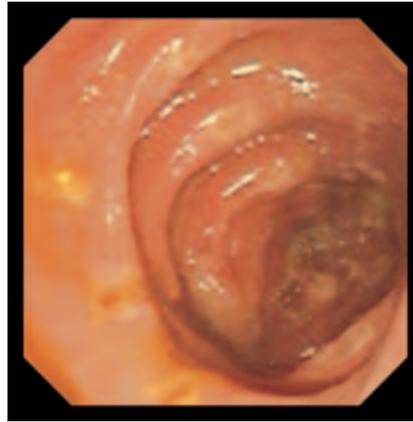
3.265

**Acute bacillary dysentery (cont.)**

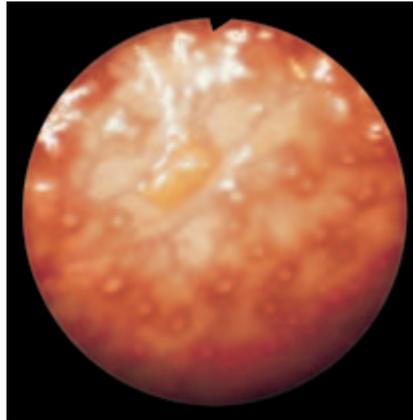
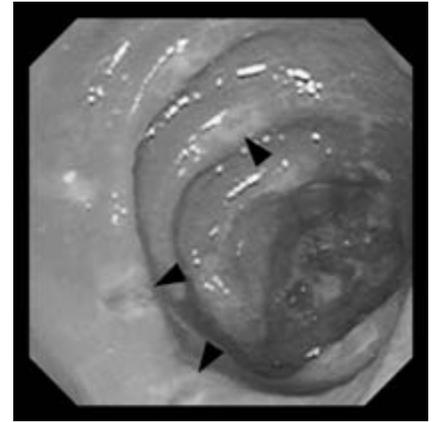
The appearances of the ileum in a patient with *Campylobacter* infection are illustrated in Fig. 3.266.

Figure 3.267 shows the classical appearance of acute colonic yersiniosis. There is a multitude of small shallow ulcers, each surrounded by an erythematous halo. The infection is always segmental in distribution and may involve the terminal ileum. Endoscopically, it may be indistinguishable from inflammatory bowel disease.

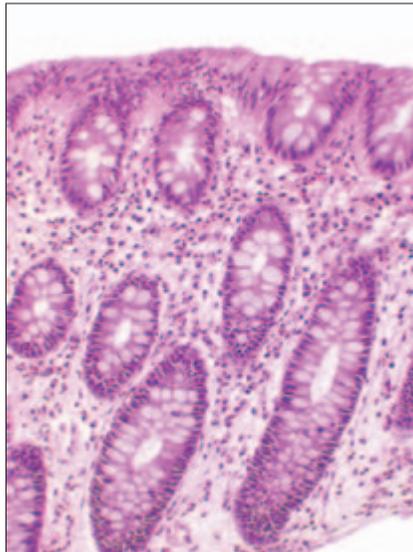
The histological appearances of an acute self-limiting bacterial colitis appear in Fig. 3.268.



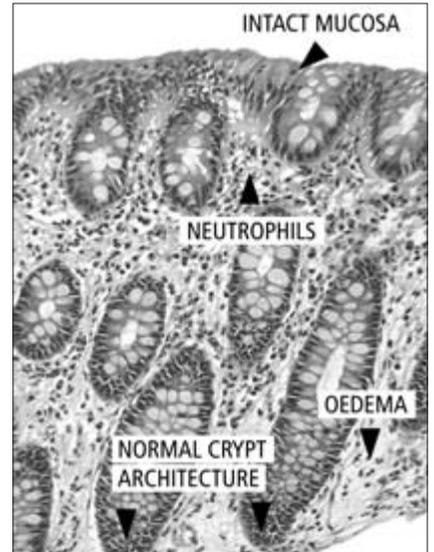
3.266



3.267



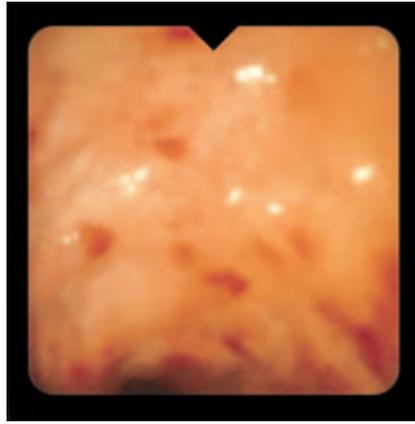
3.268



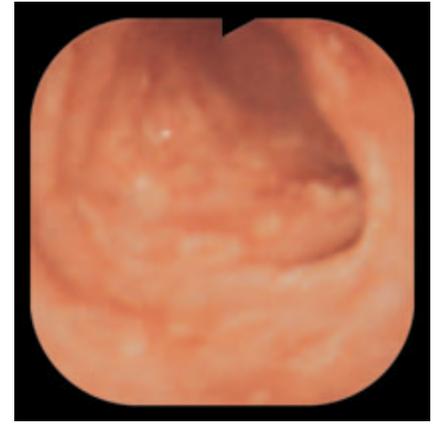
**Amoebic colitis**

In the early acute stage the changes in amoebic colitis are not specific, ranging from the mild abnormalities shown in Fig. 3.269 to severe confluent inflammation, indistinguishable endoscopically from ulcerative colitis.

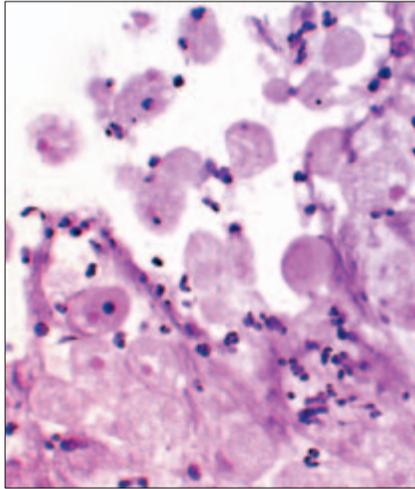
Chronic amoebic colitis manifests a very characteristic appearance with discrete punched-out ulcers (Fig. 3.270) as shown here in the rectosigmoid region. Biopsy from the edge of the ulcers usually reveals mature parasites (Fig. 3.271). They are seen best in the haematoxyphilic ulcer slough, but may only be present in small numbers necessitating meticulous histological examination. PAS staining may be helpful.



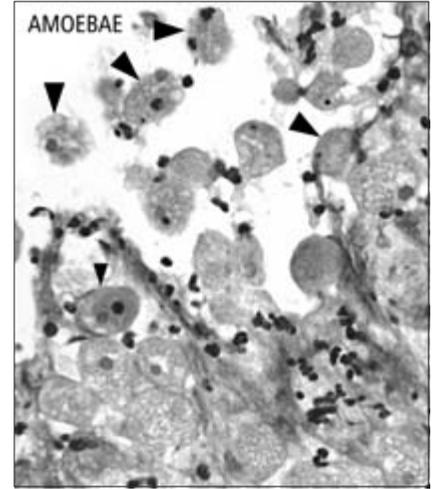
3.269



3.270



3.271

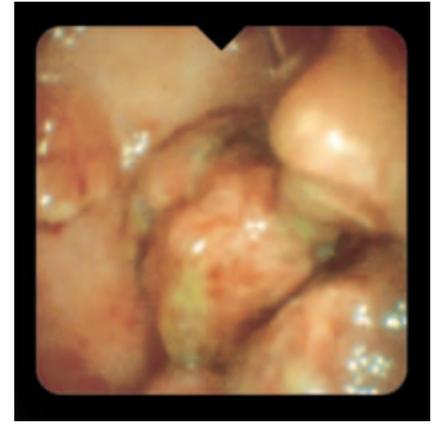


**Bilharzia**

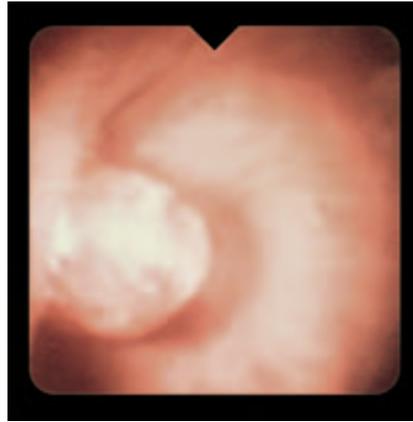
Infection with *Schistosoma mansoni* sometimes results in inflammatory polyps which contain ova. Isolated polyps in the region of the splenic flexure are shown in Fig. 3.272; extensive polyposis may occur (Fig. 3.273). In chronic infection polyps may calcify (Fig. 3.274). Figures 3.275 and 1.10 demonstrate the histological features.



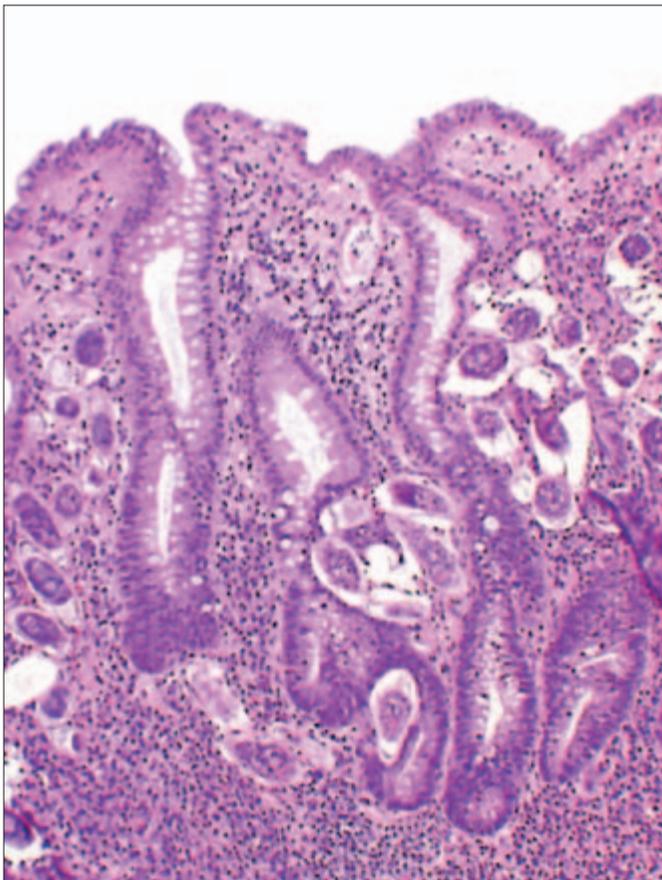
3.272



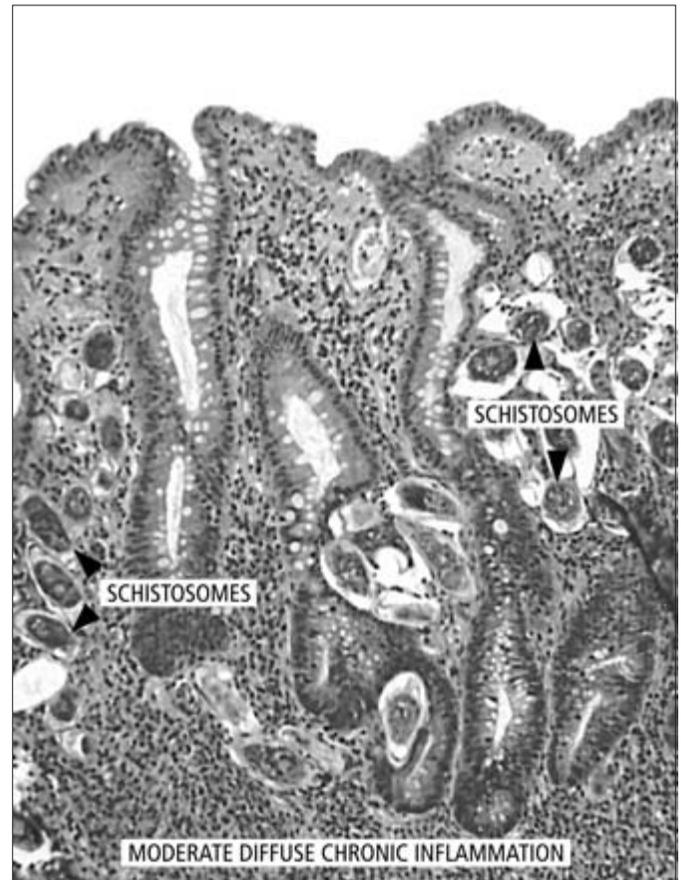
3.273



3.274

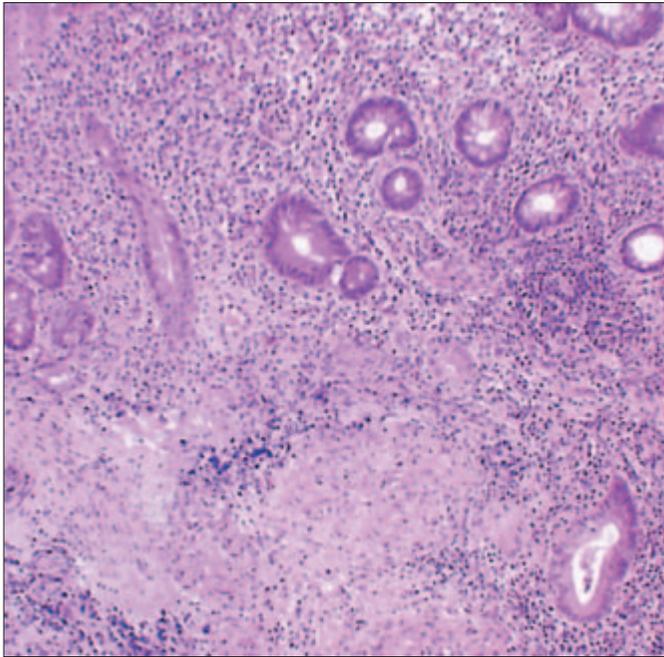


3.275



### Tuberculosis

Tuberculous infection of the large bowel or ileocaecal region may cause any of a variety of appearances, sometimes mimicking a tumour, and therefore represents a diagnostic challenge to the endoscopist. The diagnosis may be confirmed by mucosal biopsy showing coalescence of granulomas with (Fig. 3.276) or without caseation. Acid and alcohol-fast tubercle bacilli may be seen on Ziehl–Neelsen staining, or by culture from the lesions, especially if ulcerated.



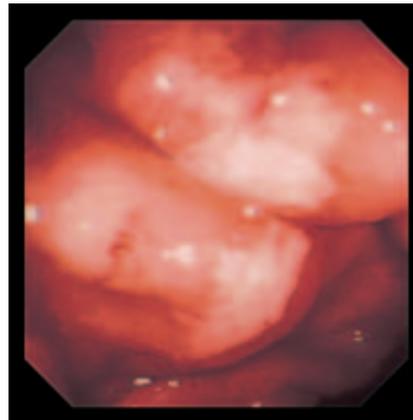
3.276



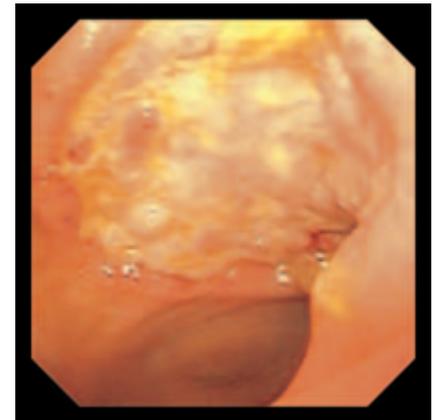
Figure 3.277 shows indolent tuberculous rectal ulceration. The large ulcer demonstrated in Fig. 3.278 was observed in a patient who had undergone renal transplantation. Cobblestoning (Fig. 3.279) may simulate the appearance of Crohn's disease (cf. Figs 3.243 and 2.244). The circumferential ulcer on a caecal stricture shown in Fig. 3.280 revealed caseating necrosis and tubercle bacilli; the stricture resolved with anti-tuberculous chemotherapy. Some colonic tuberculous lesions present as masses when malignancy enters the differential diagnosis.

Positive identification of terminal ileal tuberculosis, which is commoner, may be possible by examination of material obtained at colonoscopy when the tip of the instrument has been passed into the terminal ileum, or blindly by passing biopsy forceps through the ileocaecal valve.

It is worth remembering, in passing, that the first descriptions of Crohn's disease arose from studies on patients believed to have gastrointestinal tuberculosis.



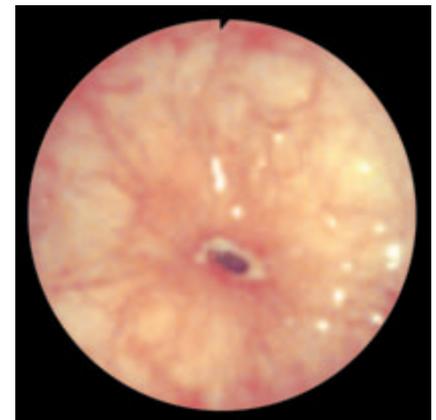
3.277



3.278



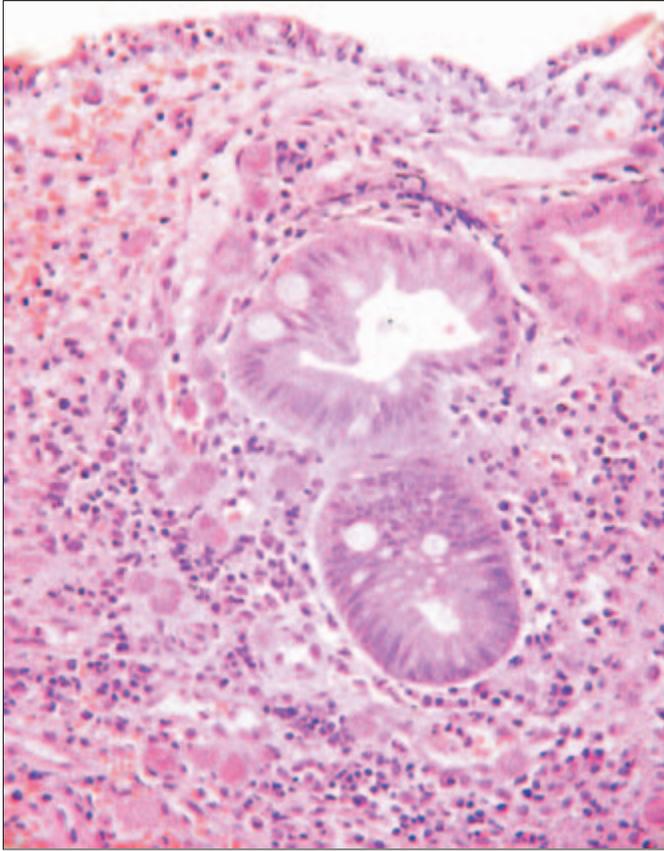
3.279



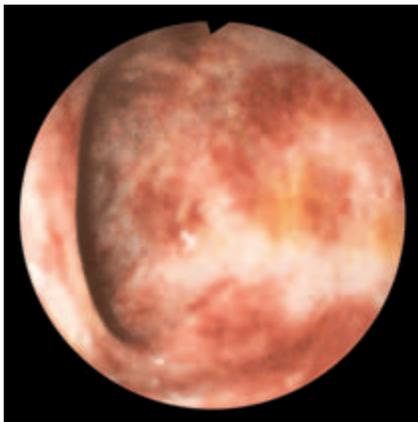
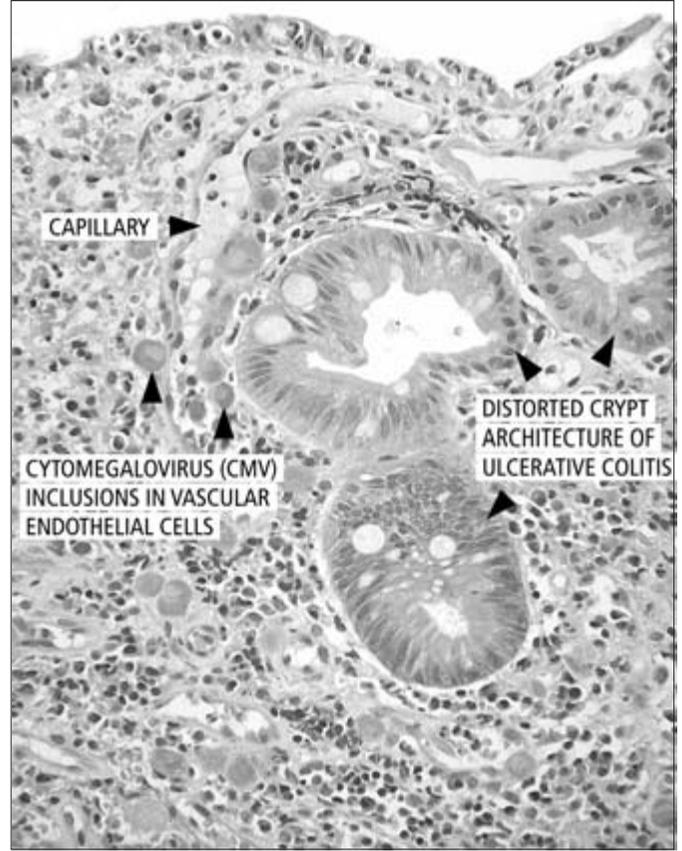
3.280

### Cytomegalovirus colitis

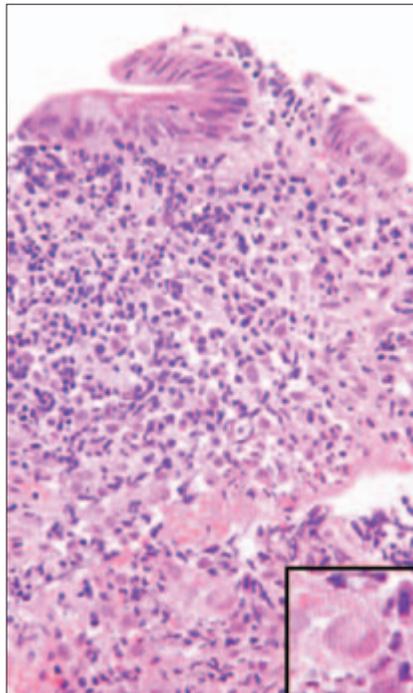
Colonic biopsies from patients with inflammatory bowel disease may incidentally show the presence of cytomegalovirus (CMV) (Fig. 3.281). The infection is commonly seen in patients with AIDS. Figure 3.282 shows multiple erythematous patches, typical of this condition. Mucosal ulceration is also reported. Figure 3.283 shows the histopathology of CMV colitis.



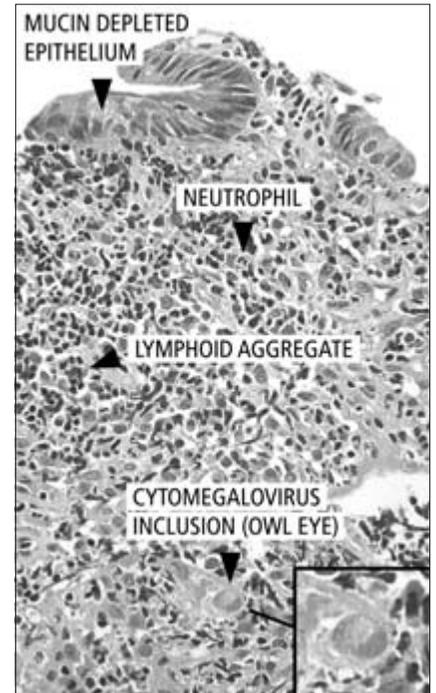
3.281



3.282



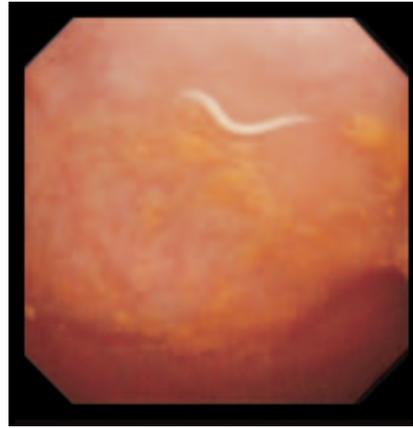
3.283



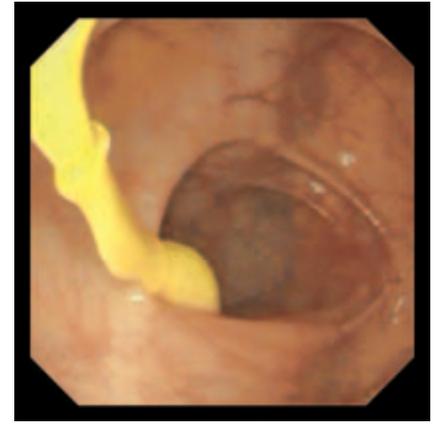
### Threadworms and tapeworms

Threadworms (*Enterobius vermicularis*) are sometimes found incidentally in the right colon and ileocaecal region, especially in children (Fig. 3.284). The mucosa is normal and many active worms may be present despite colonic preparation. They appear to tolerate bright lights and colonoscopic inspection poorly, and tend to scurry for cover. Figure 3.285 shows such a worm in an excised appendix.

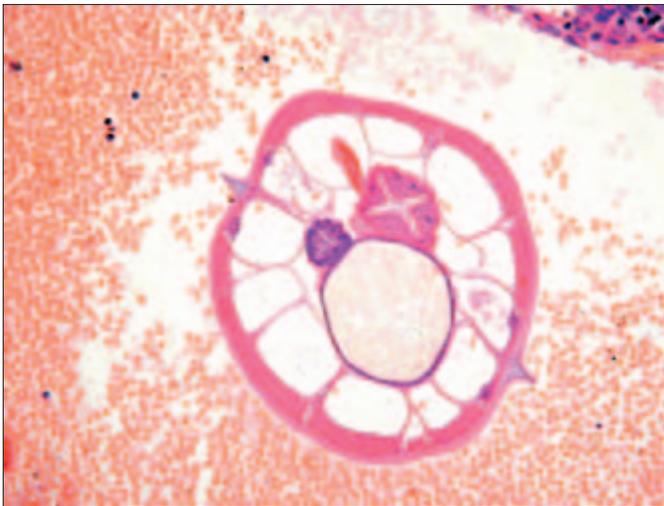
Tapeworms (Fig. 3.286) are not commonly seen at colonoscopy.



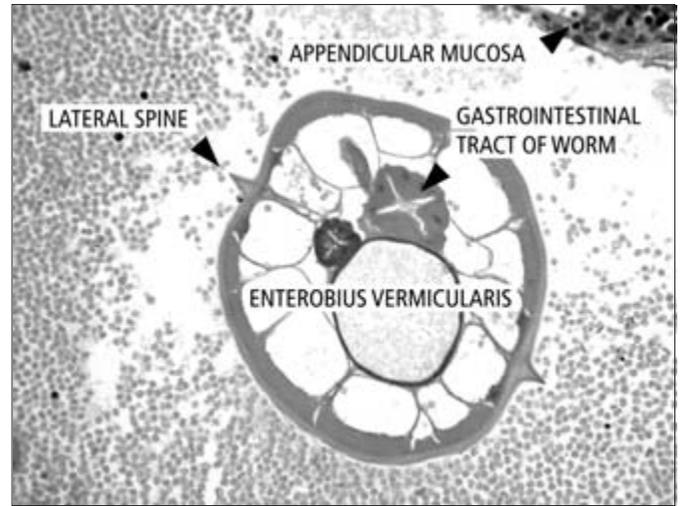
3.284



3.286



3.285



### Vascular abnormalities

Vascular ectasia (angiodysplasia)

Hereditary haemorrhagic telangiectasia

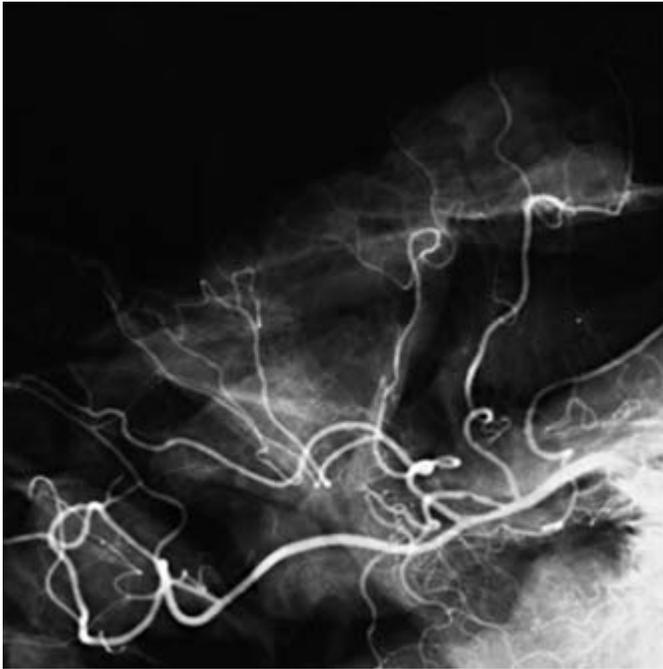
Blue rubber bleb naevus syndrome

Cavernous haemangioma of the rectum

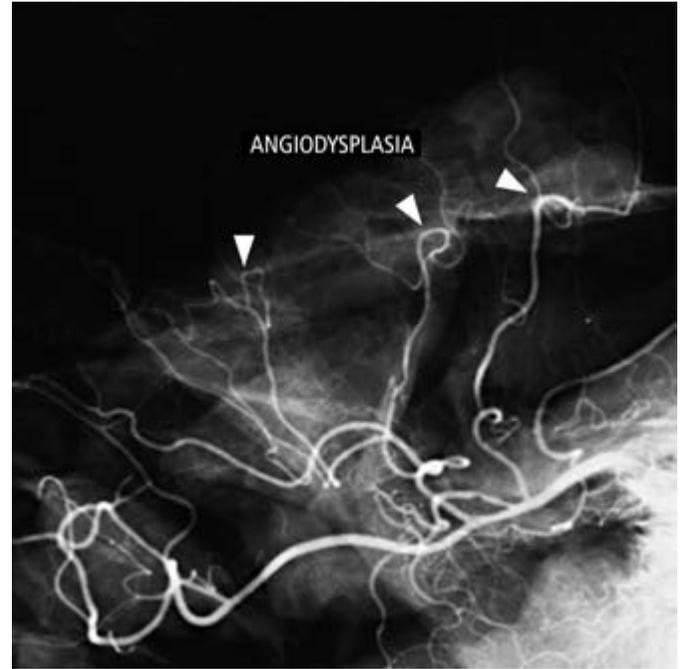
Diffuse intestinal haemangiomatosis

Varices

Oesophago-gastro-duodenoscopy, colonoscopy and latterly enteroscopy have greatly increased our knowledge of gastrointestinal vascular abnormalities. The endoscopic appearances are frequently nonspecific and it may therefore require the pathologist to affix the correct label. The following images are grouped under the diagnostic heading which seemed most appropriate to the colonoscopist at the time of the examination.



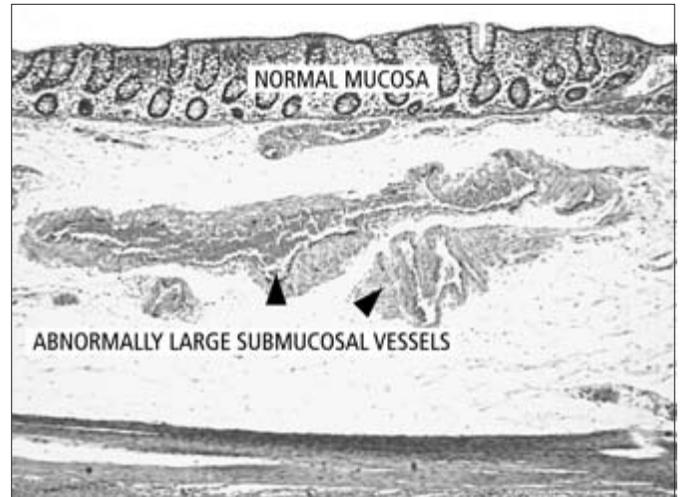
3.287a



ANGIODYSPLASIA



3.287b



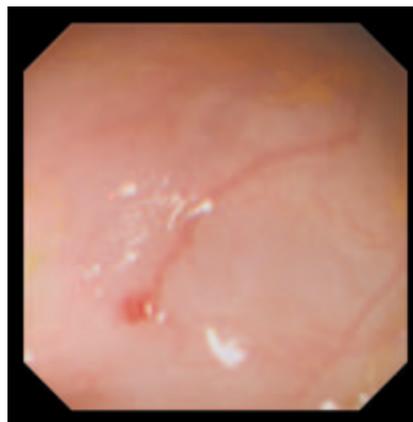
NORMAL MUCOSA

ABNORMALLY LARGE SUBMUCOSAL VESSELS

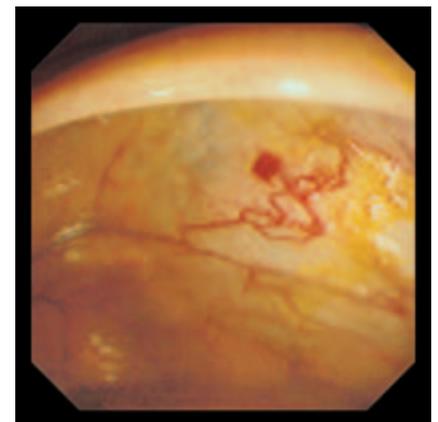
**Vascular ectasia (angiodysplasia)**

Vascular ectasia should be regarded as something of an umbrella term. Angiodysplasia is found mainly in elderly subjects and most frequently in the caecum and the ascending colon. It may present with bleeding or as a chance finding. Figure 3.287a shows the angiographic appearances in an excised specimen, and Fig. 3.287b the histopathology.

Figures 3.288 and 3.289 illustrate minimal lesions.



3.288



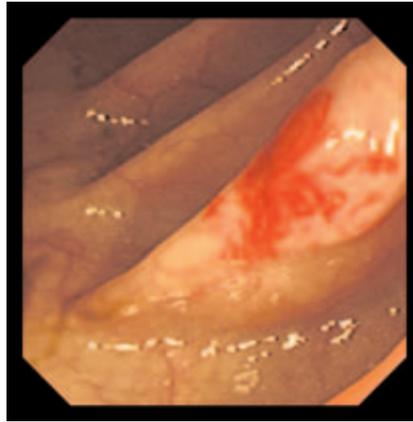
3.289

**Vascular ectasia (angiodysplasia) (cont.)**

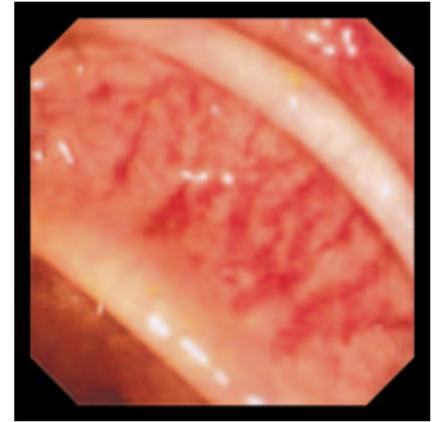
Larger multiple lesions appear in Fig. 3.290 while a markedly more extensive variant of angiodysplasia is illustrated in Fig. 3.291.

In Fig. 3.292 there is bleeding from an area of angiodysplasia. There are multiple white ulcerated areas and some fresh overlying blood clot.

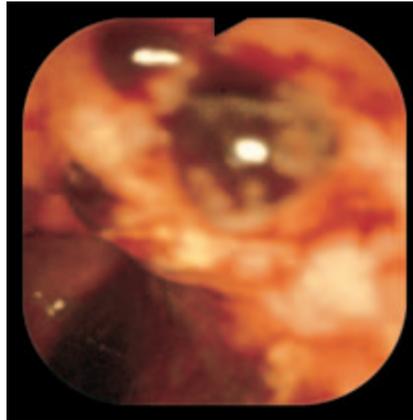
The grossly abnormal superficial venules seen in Figs 3.293 and 3.294 are striking. It is likely that in both cases these venules connect with deeper lying veins explaining the blue tinge. Figure 3.295 shows another extensive abnormality.



3.290



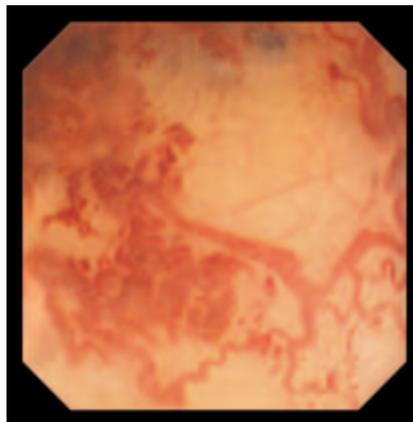
3.291



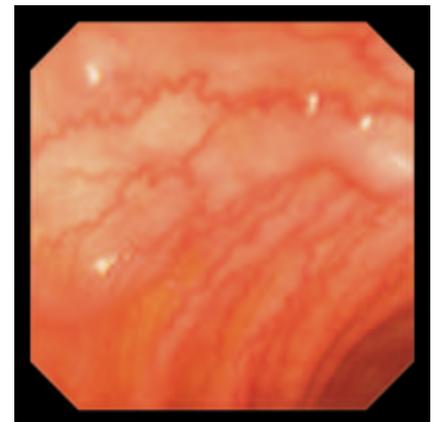
3.292



3.293



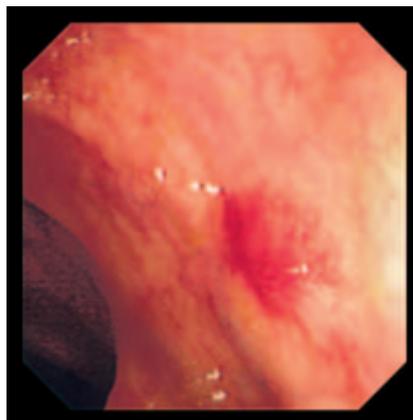
3.294



3.295

**Hereditary haemorrhagic telangiectasia**

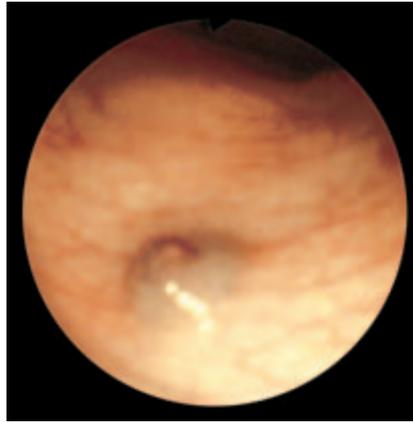
In this familial disorder, known as Osler–Weber–Rendu disease, small angiomatous lesions are found in a number of sites including the skin, lips and the whole gastrointestinal tract. Figure 3.296 shows a colonic lesion (see also Figs 2.421, 2.422 and 2.569).



3.296

**Blue rubber bleb naevus syndrome**

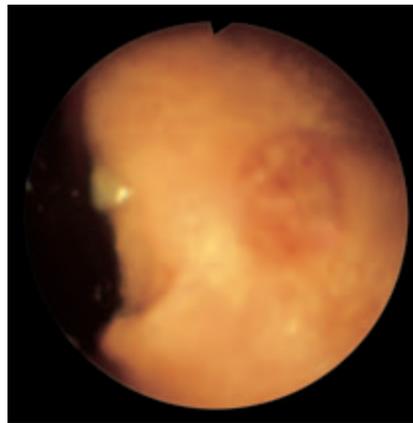
This is a rare condition where cavernous haemangiomas with a bluish colour may be found in the skin and the gastrointestinal tract including the colon (Fig. 3.297). The syndrome is also known as cutaneous and intestinal cavernous haemangiomatosis.



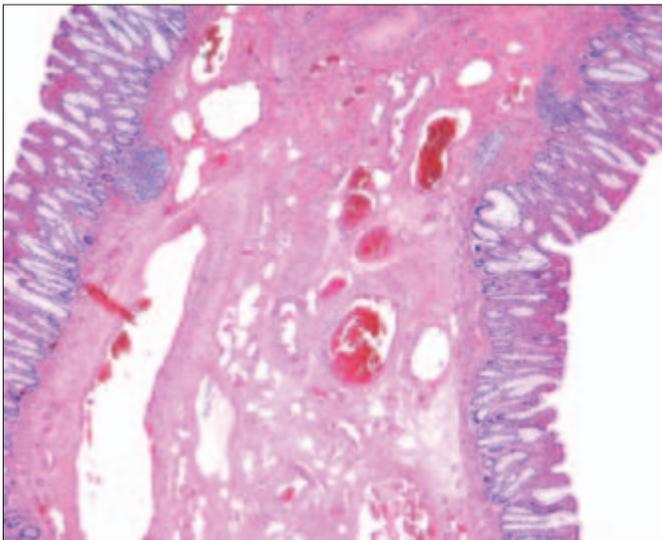
3.297

**Cavernous haemangioma of the rectum**

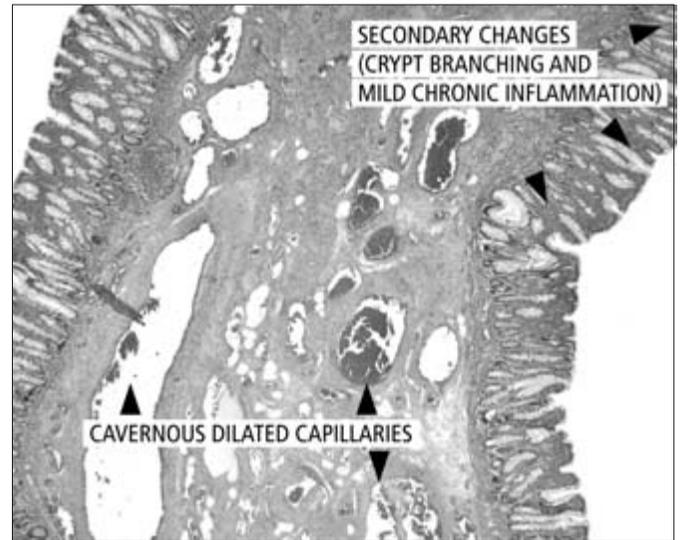
These are solitary lesions of varying size. The lesion shown in Fig. 3.298 is small. Such lesions may be very extensive and can cause severe bleeding. Figure 3.299 illustrates the histology.



3.298

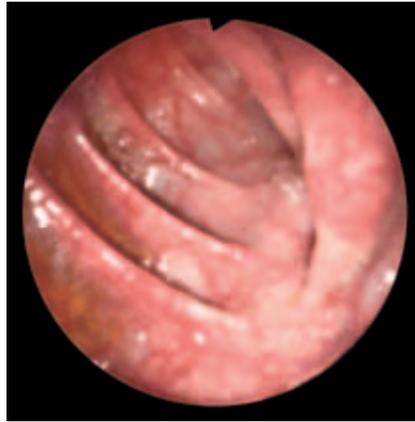


3.299



**Diffuse intestinal haemangiomatosis**

Figure 3.300 shows an extensive vascular malformation which was present throughout the length of the transverse colon, and was found in association with discrete cavernous haemangiomas elsewhere in the colon.

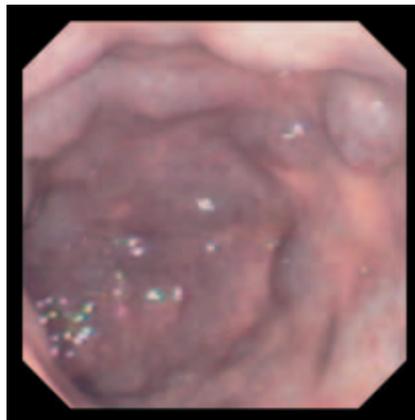


3.300

**Varices**

Varices may be found as a localized abnormality in any part of the colon when the cause may be uncertain. More commonly they occur in the rectum in patients with portal hypertension. Appearances are unpredictably variable as elsewhere in the gastrointestinal tract (e.g. Figs 2.186–2.193 and Figs 2.435–2.438).

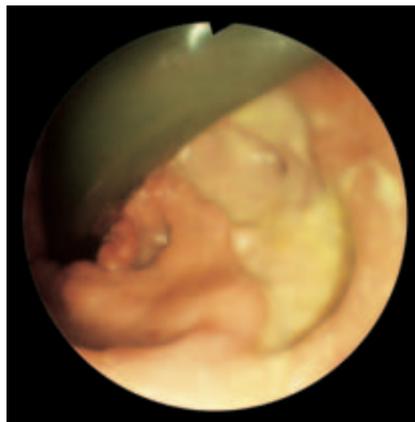
The rectal varices shown in Fig. 3.301 are markedly blue, a feature not present in another patient with this condition (Fig. 3.302). Figure 3.303 shows a superficial ulcer at the site of injection of a sclerosant (cf. Fig. 2.757). Unusually large rectal varices are shown in Figs 3.304 and 3.305.



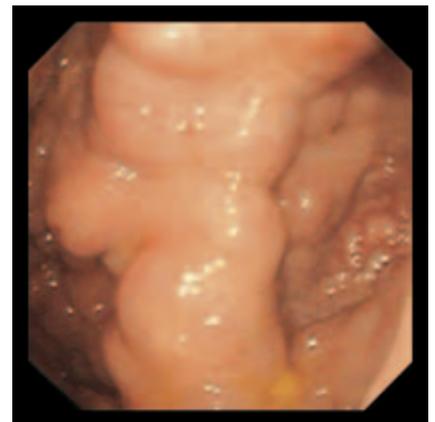
3.301



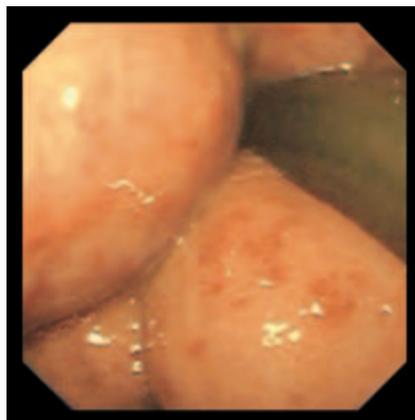
3.302



3.303



3.304



3.305

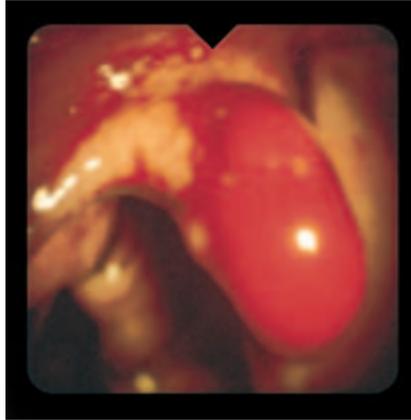
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## Bleeding

### Bleeding

Colonoscopy during severe rectal bleeding is often unrewarding due to poor vision. It should if possible be deferred until bleeding has stopped. It is, however, very useful in the subsequent search for a cause of which there are many, including vascular abnormalities, ulcerative colitis, diverticular disease, carcinoma and polyps.

Figure 3.306 shows a small haemorrhagic polyp. Occasionally bleeding may follow colonoscopic polypectomy (Figs 3.443 and 3.444).



3.306

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## Miscellaneous conditions

Pseudomembranous colitis

Colonic disease and nonsteroidal anti-inflammatory drugs (NSAIDs)

Ischaemic colitis

Radiation proctocolitis

Chemical proctitis

Mucosal prolapse syndrome

Ectopic gastric mucosa

Leukoplakia

Pneumatosis cystoides intestinalis

Colitis cystica profunda

Deformities due to external pressure

Intussusception

Melanosis coli

Muciphage mucosa

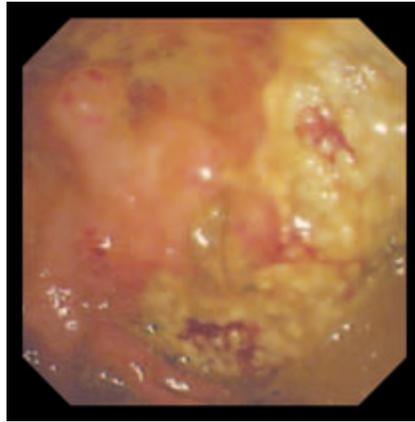
Retained foreign bodies

Faecal impaction

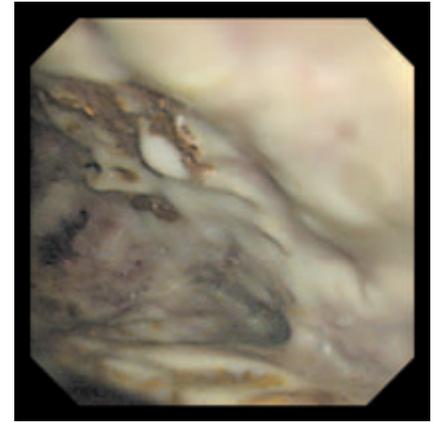
Megacolon

**Pseudomembranous colitis**

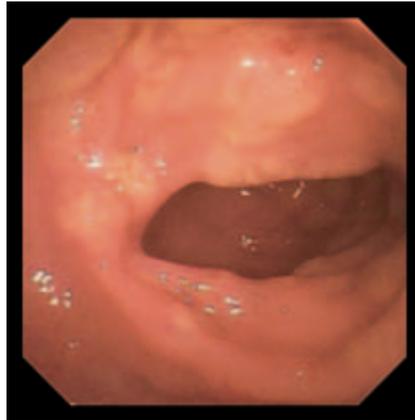
In this condition, which is usually left sided, a yellow or grey, patchy (Fig. 3.307) or confluent (Fig. 3.308) membrane overlies intensely inflamed mucosa. If the pseudomembrane is lifted off with biopsy forceps the underlying inflammation is clearly seen. In another case a membrane was not seen endoscopically (Fig. 3.309) but the diagnosis was confirmed histopathologically. Endoscopic appearances may simulate ulcerative colitis but the almost invariable history of antibiotic ingestion (hence the synonyms antibiotic-induced or antibiotic-related colitis) and the typical histological findings of the volcano lesion (Fig. 3.310) should permit accurate diagnosis. *Clostridium difficile* or more commonly its toxin may be isolated from the faeces.



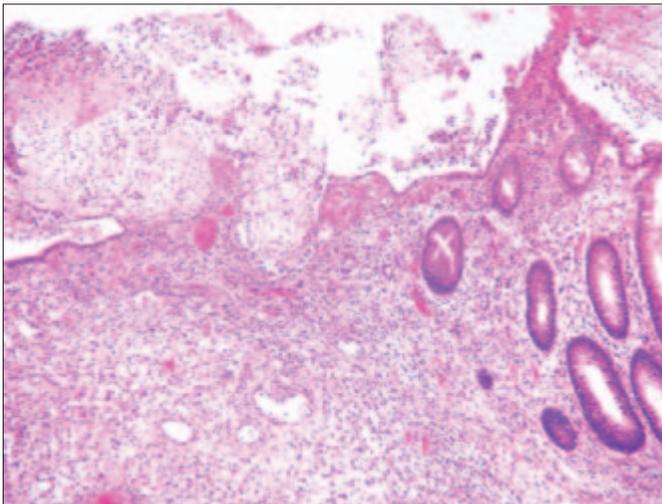
3.307



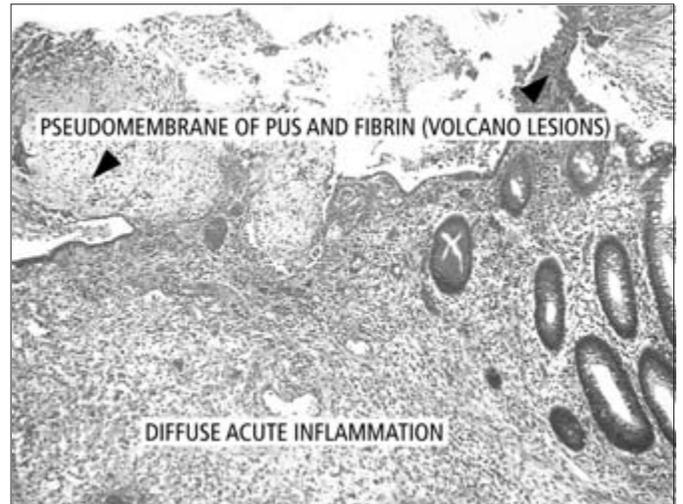
3.308



3.309



3.310

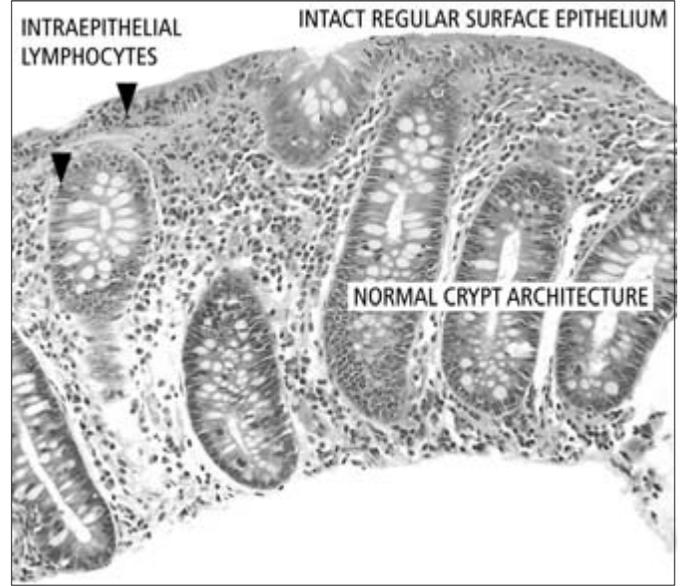


### Colonic disease and nonsteroidal anti-inflammatory drugs (NSAIDs)

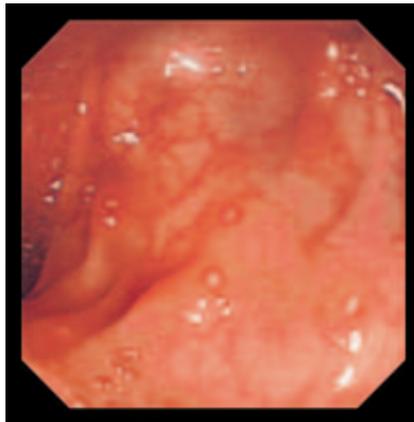
The effects of NSAIDs on the colon are less well appreciated than those in the upper gastrointestinal tract. Nevertheless, there is strong evidence of direct toxicity leading to exacerbations of ulcerative colitis or causing a disorder endoscopically indistinguishable from ulcerative colitis. It is possible that the whole range of appearances seen in the small bowel and attributed to NSAIDs may be mirrored in the colon. The histology of lymphocytic colitis due to NSAIDs appears in Fig. 3.311.



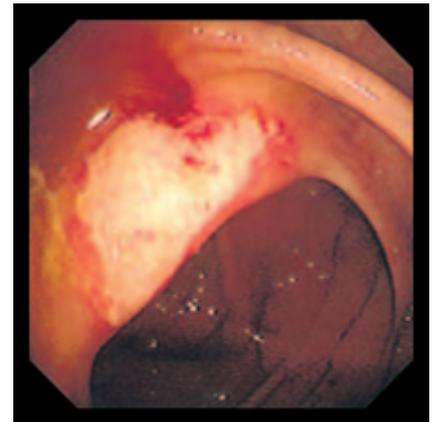
3.311



NSAIDs may cause multiple colonic aphthoid ulcers (Fig. 3.312) similar to NSAID-related lesions in other parts of the gastrointestinal tract, for example the duodenal erosions shown in Fig. 2.488. The differential diagnosis is extensive (see p. 282) and includes early colonic Crohn's disease (Fig. 3.232). Figure 3.313 shows an isolated irregular superficial colonic ulcer attributed to the ingestion of naproxen.



3.312



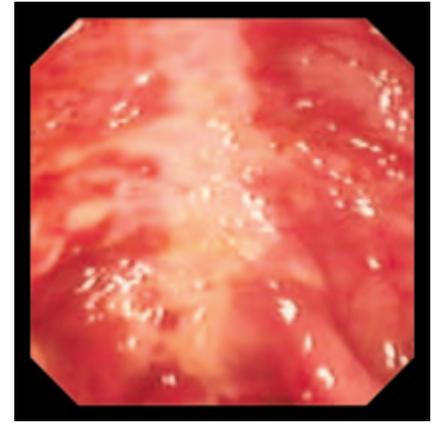
3.313

**Colonic disease and nonsteroidal anti-inflammatory drugs (NSAIDs) (cont.)**

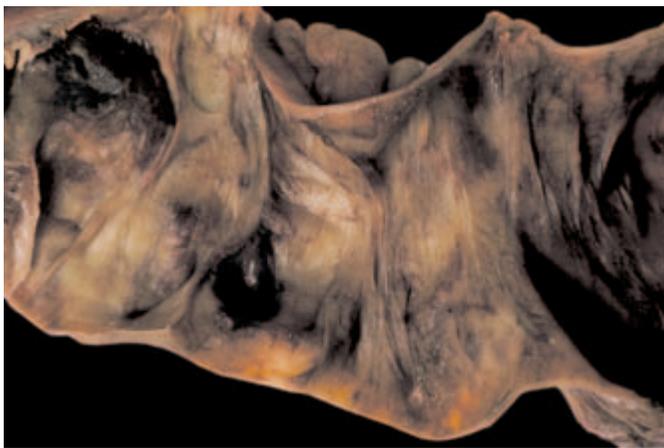
A non-steroidal drug was thought to be the cause of multiple colonic ulcers including a stellate caecal ulcer (Fig. 3.314). Appearances of intense inflammation and ulceration as shown in Fig. 3.315 developed 36 h after a single injection of diclofenac; toxic dilatation and perforation necessitated total colectomy, and the resected specimen revealed severe pancolitis. Figure 3.316 shows a stricture, one of many, similar to the diaphragm lesions which may occur in the small bowel (Fig. 5.45).



3.314



3.315



3.316

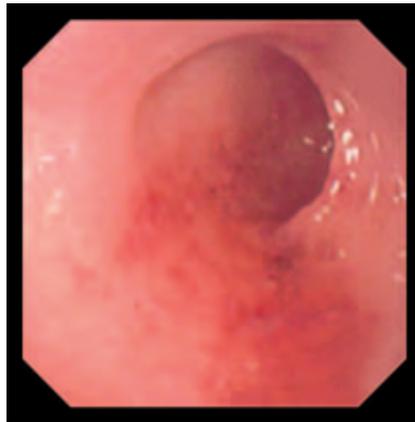


**Ischaemic colitis**

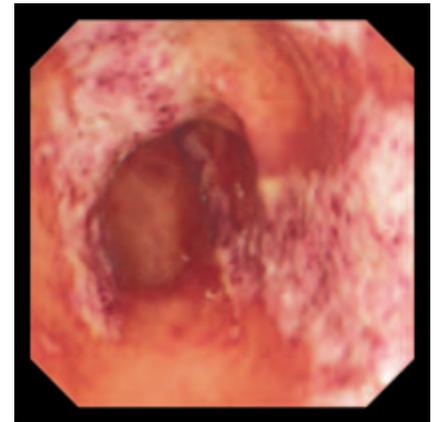
The acute condition is encountered during investigation of sudden rectal bleeding of uncertain cause. Endoscopy should not be considered if there is any suspicion of gangrene of the bowel.

The abnormalities found in ischaemic colitis are variable and depend upon the severity of the ischaemia, its duration and the time interval between the ischaemic episode and colonoscopic examination.

The earliest lesion is a pale mucosa with multiple petechial haemorrhages (Fig. 3.317) followed by streaks of superficial necrosis (Fig. 3.318).



3.317



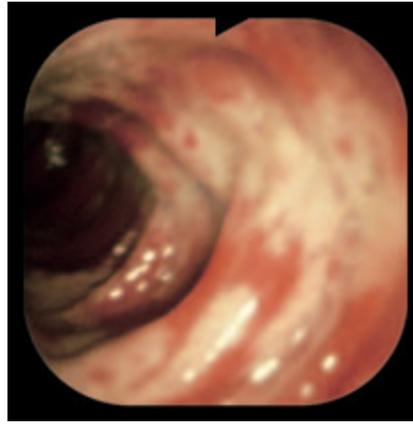
3.318

**Ischaemic colitis (cont.)**

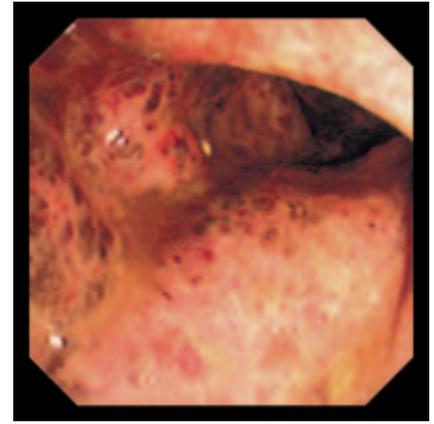
Later serpinginous ulcers develop (Fig. 3.319), similar to those seen in Crohn's disease, but shallower.

In severe disease the mucosa is oedematous with petechial haemorrhages and ulceration (Fig. 3.320). Severe mucosal oedema leads to localized areas of swelling (Fig. 3.321); in its most severe form (Fig. 3.322) such swellings form the 'thumb-printing' seen radiologically. Superficial sloughing results in a pseudomembrane (Fig. 3.323) which is sometimes grey or green in colour.

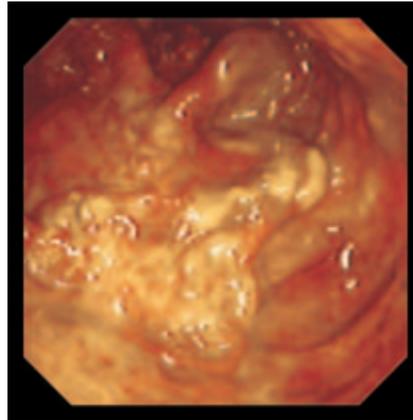
After the acute phase has passed, an extensive pseudomembrane, here white (Fig. 3.324), can be formed. With healing a smooth stricture may result (Fig. 3.325). Healing may also be accompanied by pseudopolyp formation (Fig. 3.326), though more commonly there is no residual scarring. During the healing phase, isolated ischaemic ulcers may be surrounded by extensive petechiae (Fig. 3.327).



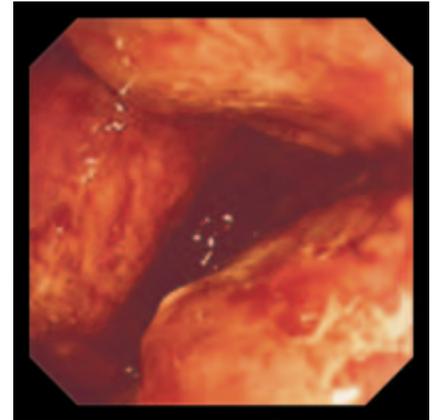
3.319



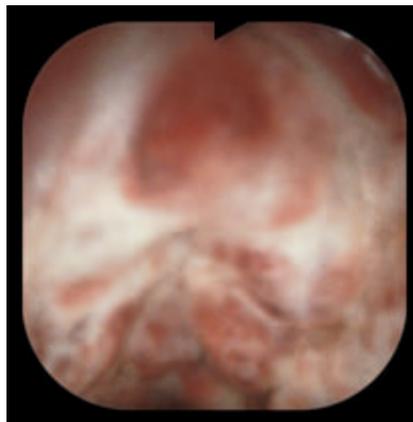
3.320



3.321



3.322



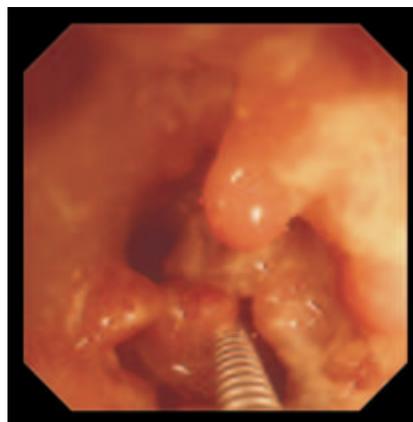
3.323



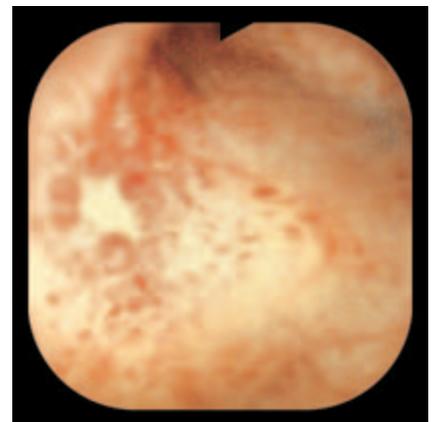
3.324



3.325



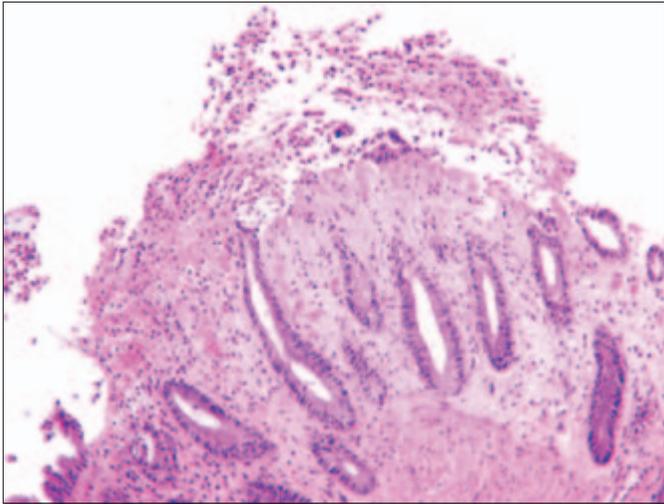
3.326



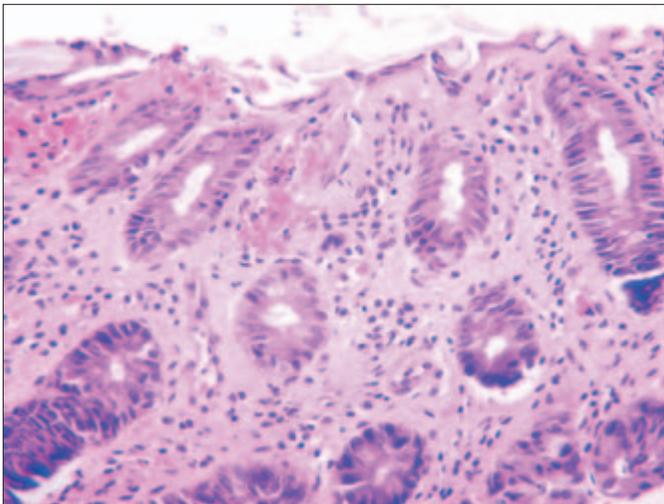
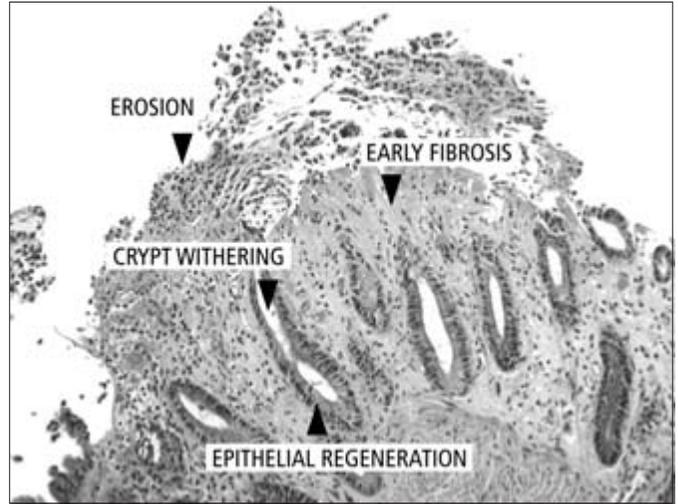
3.327

**Ischaemic colitis (cont.)**

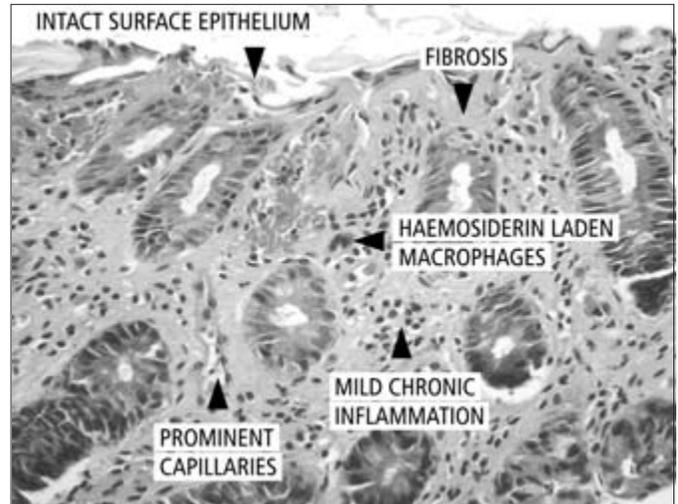
Figures 3.328 and 3.329 show the early and late stages in this condition: note the haemosiderin laden macrophages typical of the condition.



3.328



3.329



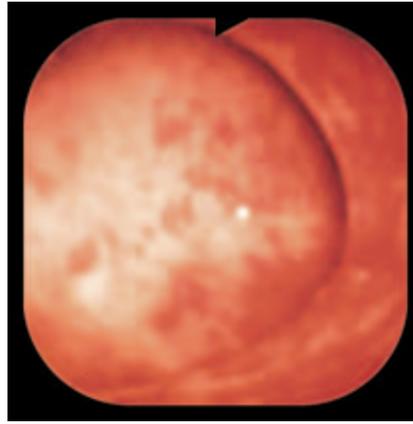
### Radiation proctocolitis

Colonoscopy must be undertaken with caution in patients with suspected radiation-induced proctocolitis as the colon is likely to be both fragile and fixed within the pelvis, making perforation a real hazard. Most commonly the condition is encountered at a late stage when investigating rectal bleeding or abnormalities shown by barium enema examination.

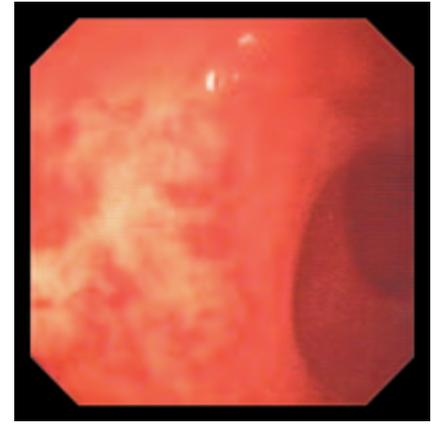
The changes of acute radiation proctocolitis (Fig. 3.330) are diffuse bleeding, severe mucosal oedema and often extensive irregular ulceration.

Later radiation proctocolitis appears as a thin atrophic mucosa with multiple irregular telangiectases (Fig. 3.331), resembling radiation effects on the skin.

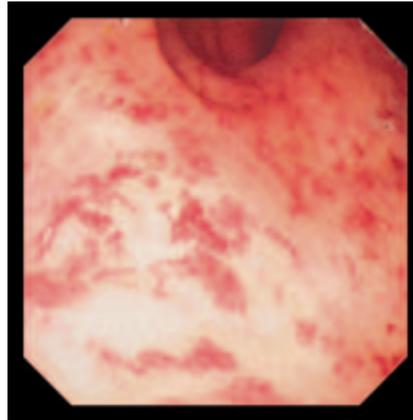
The appearances of late stage rectal irradiation proctocolitis appear in Fig. 3.332. Note the pale mucosa, the absence of normal vessels, and the mass of telangiectases. Strictures may also be found at this stage (Fig. 3.333).



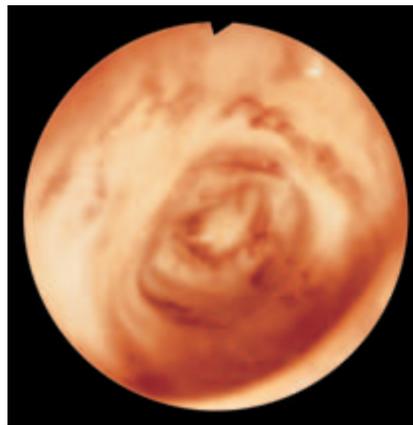
3.330



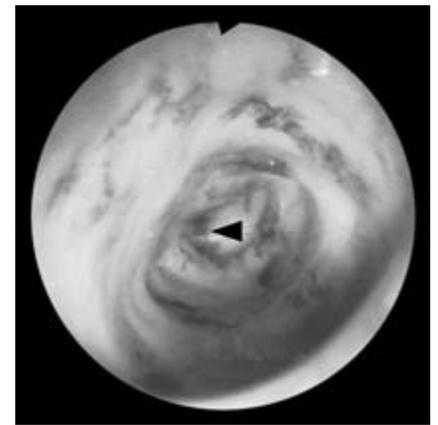
3.331



3.332

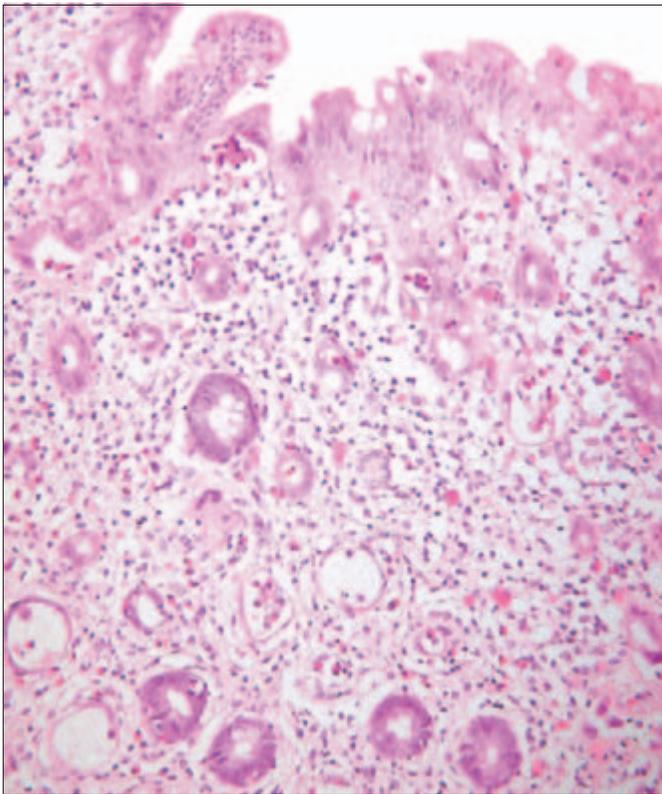


3.333

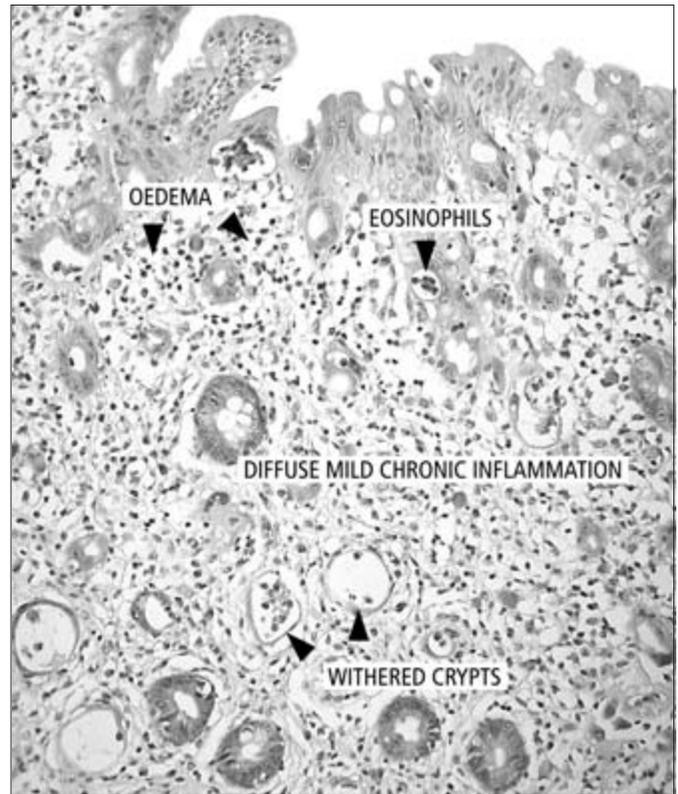


**Radiation proctocolitis (cont.)**

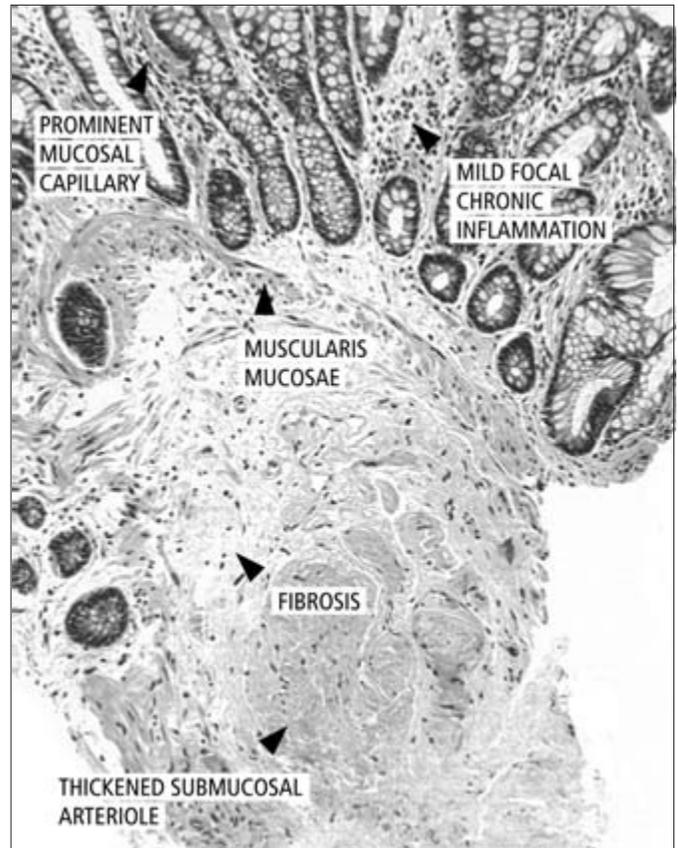
Figures 3.334 and 3.335 show the histology of the early and late stages of radiation proctocolitis.



3.334



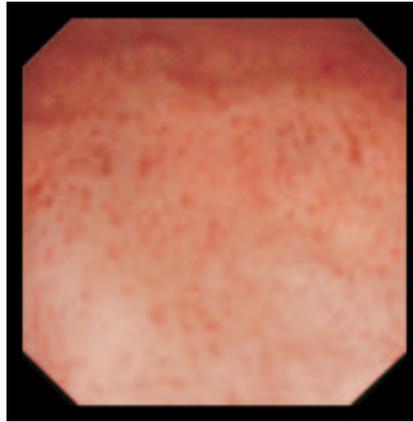
3.335



### Chemical proctitis

Diarrhoea and bleeding due to mucosal ulceration has been reported following exposure of the colon to glutaraldehyde: glutaraldehyde-induced colitis. If glutaraldehyde, used for disinfecting the colonoscope, has not been completely removed in the washing process, it may cause transient endoscopic and histological (Fig. 1.20) mucosal changes. Similar findings may be seen following the use of a phosphate enema (Fig. 3.336).

Hydrogen peroxide has infrequently been used for cleaning colonoscopes. The white slightly raised lesions (Fig. 3.337) seen during withdrawal of the instrument were caused by residual hydrogen peroxide running on to the mucosa from the water channel. Such appearances are visually striking but clinically unimportant. The histological changes are shown in Fig. 1.21.



3.336

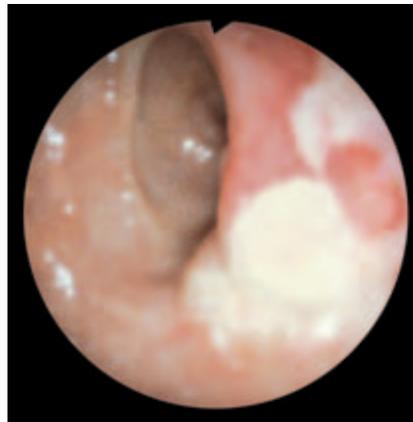


3.337

### Mucosal prolapse syndrome

Solitary rectal ulcer, the best known example of this syndrome, usually occurs on the anterior rectal wall at 7–10 cm from the anal margin. It is situated at the apex of a fold of mucosa which may be damaged during prolapse of the rectal mucosa. Such ulcers are not necessarily solitary: when many are present they may coalesce to form a single butterfly shaped ulcer.

Typical single ulcers are seen in Figs 3.338 and 3.339; there are multiple ulcers in Fig. 3.340. Mucosal prolapse may also be polypoid, with or without ulceration (Fig. 3.341). Such lesions may have characteristic endoscopic features.



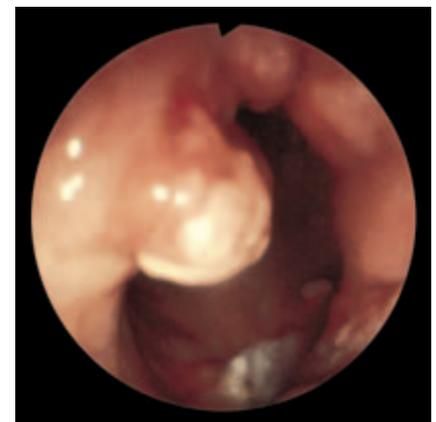
3.338



3.339



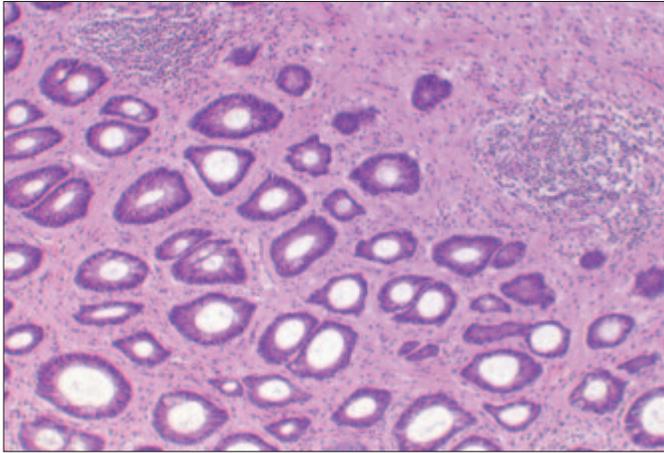
3.340



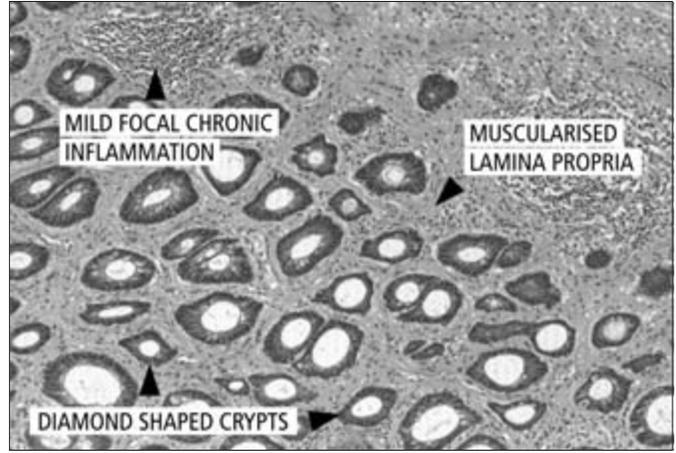
3.341

### Mucosal prolapse syndrome (cont.)

The histological appearances (Fig. 3.342) of the mucosal prolapse syndrome are diagnostic and differentiate the condition from other ulcerative diseases of the rectum, especially carcinoma, which it may mimic endoscopically, and from other polypoid lesions.

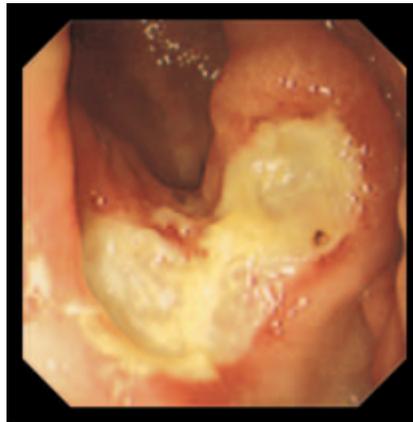


3.342

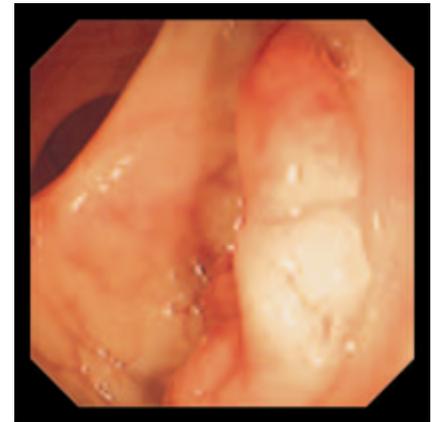


### Ectopic gastric mucosa

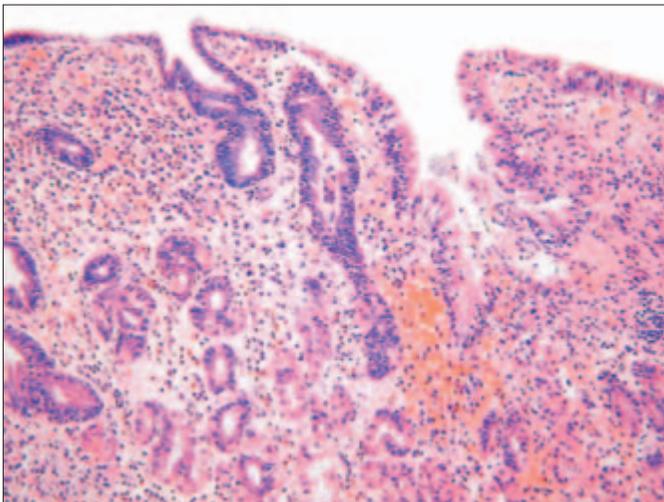
Ectopic gastric mucosa may occur at various sites in the gastrointestinal tract. The best known of these is in or near a Meckel's diverticulum in the terminal ileum. Rarely, there may be a patch in the large bowel and, as in Meckel's diverticulum (Fig. 5.19), ulceration may develop in this ectopic mucosa (Fig. 3.343). Under the influence of a proton pump inhibitor the ulceration healed but the abnormal patch of mucosa is still clearly visible (Fig. 3.344). Figure 3.345 shows the histopathological appearances of ectopic gastric mucosa.



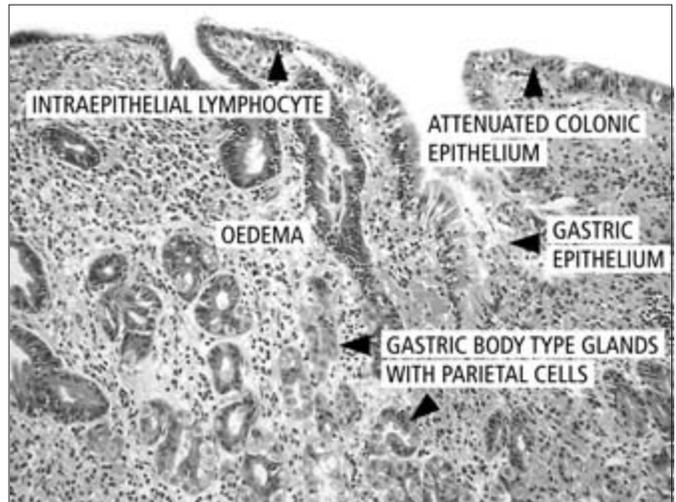
3.343



3.344



3.345

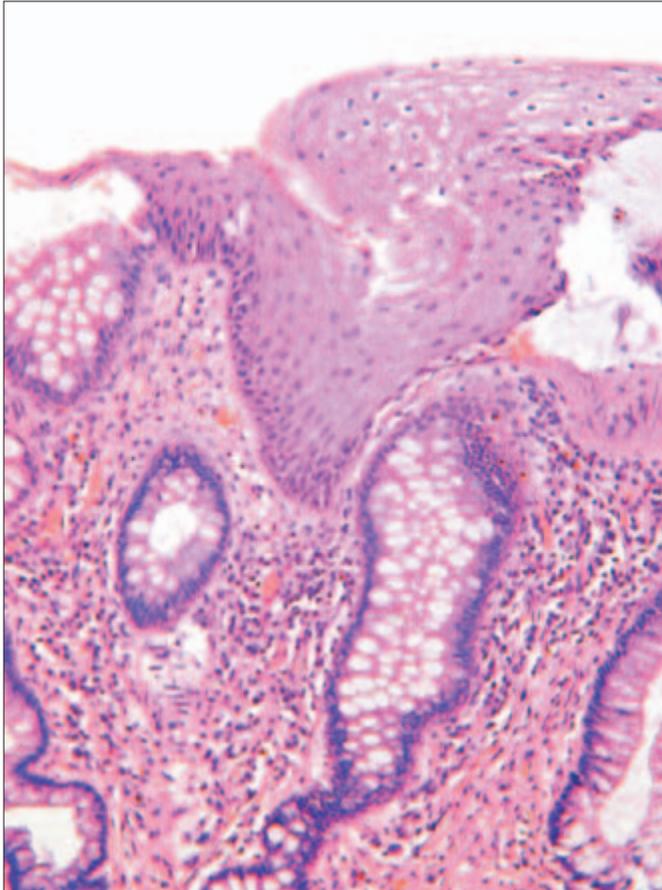


**Leukoplakia**

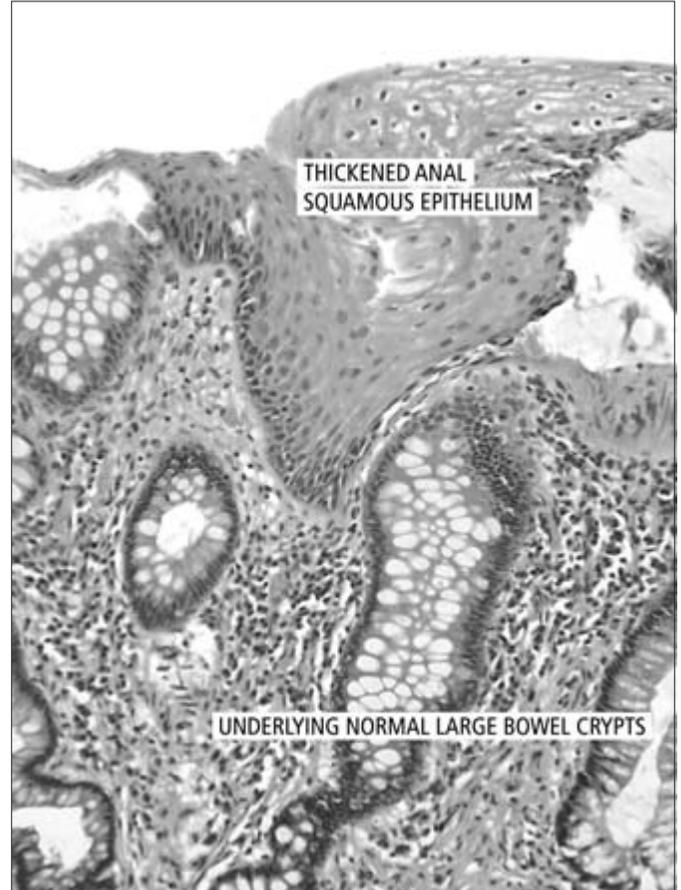
Minor degrees of rectal leukoplakia due to upgrowth of squamous epithelium from the anus over ulcerated rectal mucosa (Fig. 3.346) may occur in prolapsing haemorrhoids. More extensive leukoplakia, sometimes with evidence of self-inflicted trauma, has been described, but is uncommon. In this case (Fig. 3.347) it was associated with unusual sexual practices.



3.347



3.346

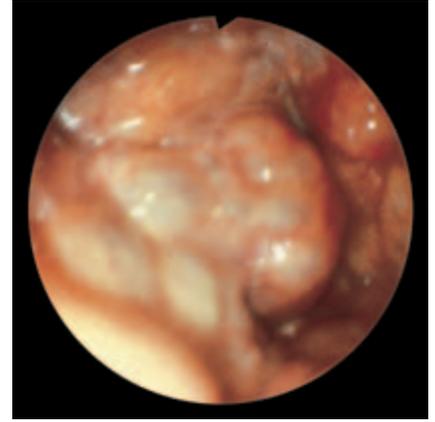


**Pneumatosis cystoides intestinalis**

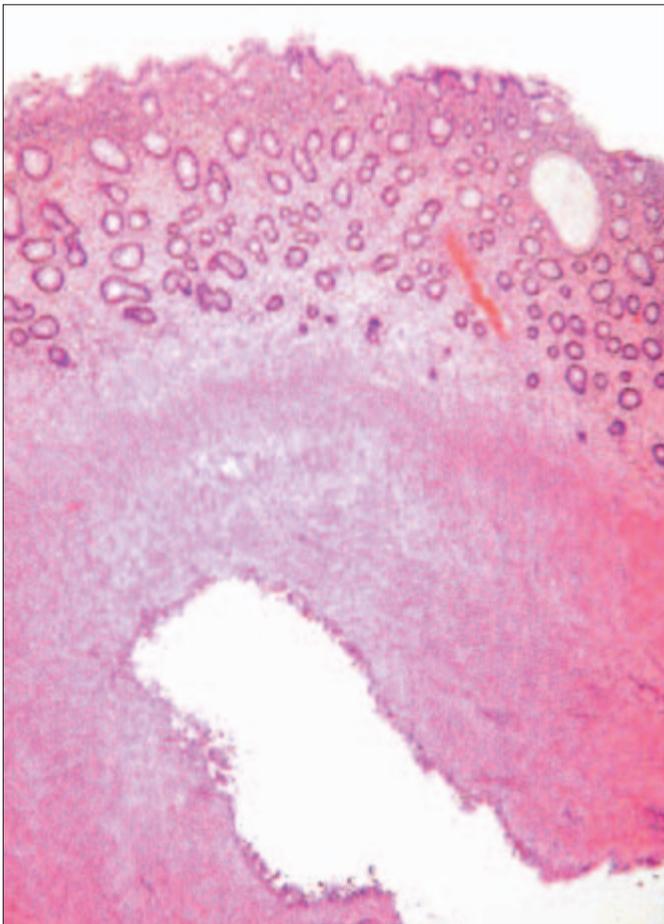
Innumerable gas-containing cysts occur in the bowel wall in this condition, often in association with chronic respiratory disease. The cysts may occur at any site in the bowel and appear from the luminal aspect as multiple often translucent broad based 'polyps' (Fig. 3.348). Appearances are however, somewhat variable and the transparent nature of the polyp-like lesions is sometimes less marked (Fig. 3.349). The gas-containing blebs are punctured when mucosal biopsy is performed, often with an audible 'pop'. The histology appears in Fig. 3.350.



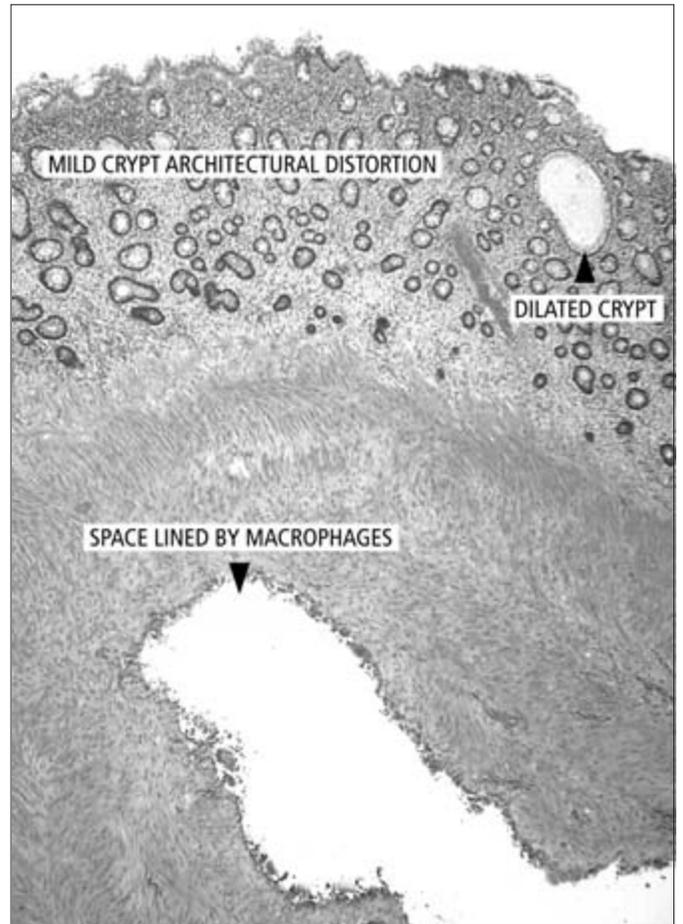
3.348



3.349



3.350

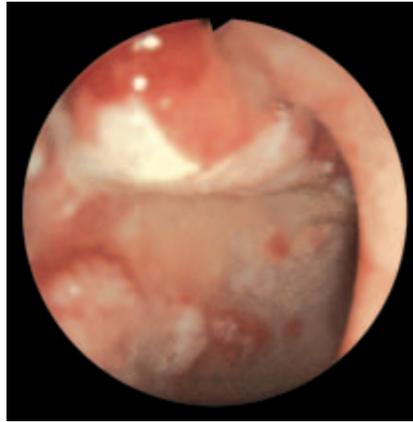


### Colitis cystica profunda

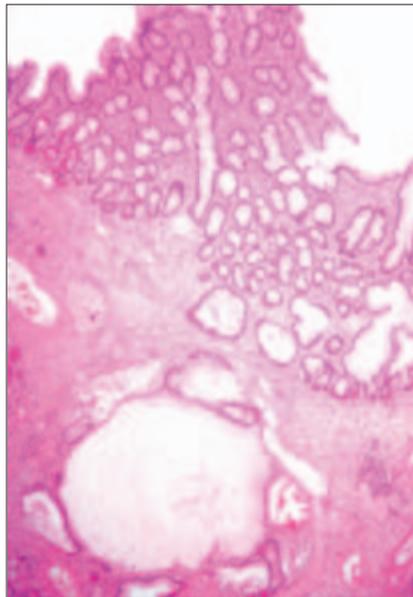
Multiple mucus-filled cysts, situated below the muscularis mucosae, produce sessile polyps about 1 cm in diameter (benign misplaced epithelium). These may be seen both in mucosal prolapse, in the rectum and in ulcerative colitis elsewhere.

Deep biopsies allow distinction from other polypoid lesions. The histological differential diagnosis is from adenocarcinoma.

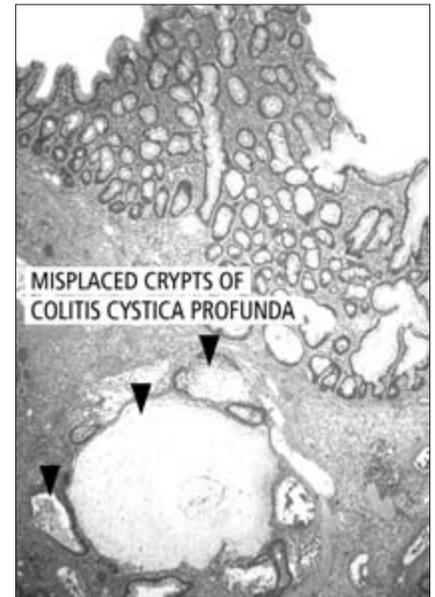
Figure 3.351 shows the typical appearance in prolapse and Fig. 3.352 the histopathological appearances. NB misplaced epithelium is not dysplastic.



3.351



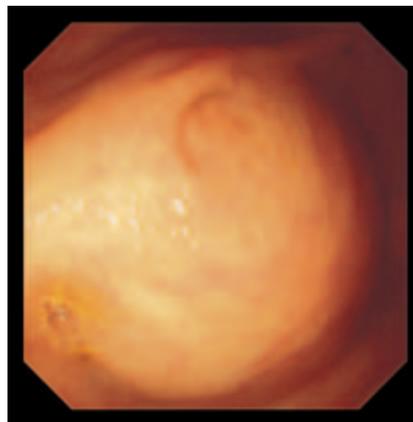
3.352



### Deformities due to external pressure

Any organ or mass against which the normal colon lies may cause a deformity of the colonic wall. It is common, for example, to see peristaltic movement or the impression of a looped colonoscope in an adjacent loop of bowel. Similarly, in the identification of the caecum it is useful to 'walk' the fingers in the right ileac fossa towards the inguinal area and to observe the indentations colonoscopically.

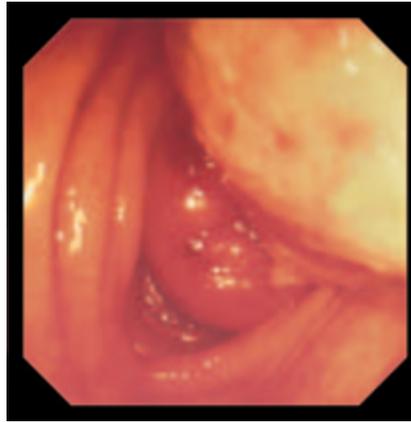
Figure 3.353 shows a rounded indentation over which it was possible to move the unattached caecal mucosa. This was due to a large underlying uterine fibroid.



3.353

### Intussusception

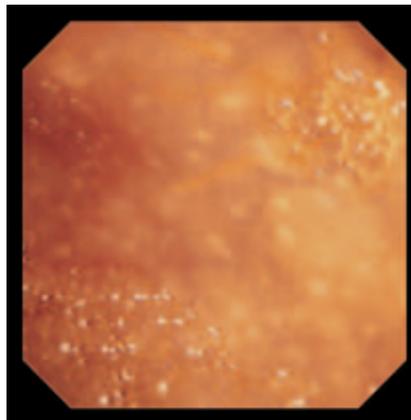
In the adult a benign or malignant tumour forms the lead point in up to 90% of cases. The large, round, unusually yellow lesion seen in Fig. 3.354 is a colonic lipoma with a short stalk which had caused an intussusception. The pink rim at the base of the lesion is a ring of intussuscepting large bowel mucosa which is engorged and oedematous.



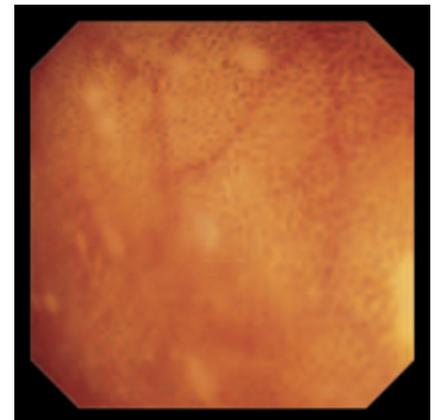
3.354

### Melanosis coli

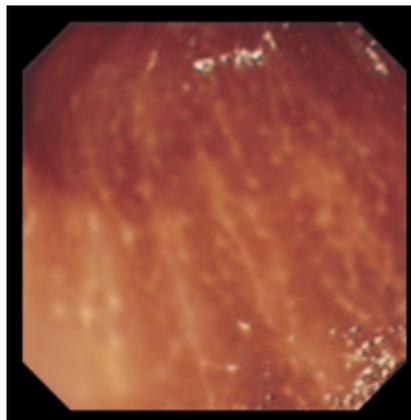
Prolonged ingestion of anthracene containing laxatives or NSAIDs causes apoptosis with deposition of lipofuscin pigment in the colonic mucosa and macrophages: melanosis coli. This may result in a variety of appearances (Figs 3.355–3.364). In Fig. 3.359 the mucosal crypts are nonmelanotic and easily recognized (cf. Figs 3.6 and 3.7). When changes are more advanced, diffuse blackening occurs making visualization and especially endoscopic photography difficult (Fig. 3.360). By contrast melaena and iron-stained faeces may result in black adherent material which can easily be washed off the mucosa.



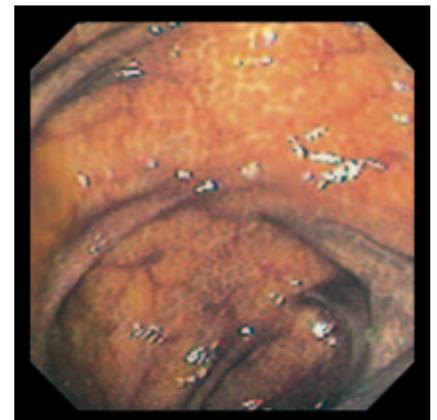
3.355



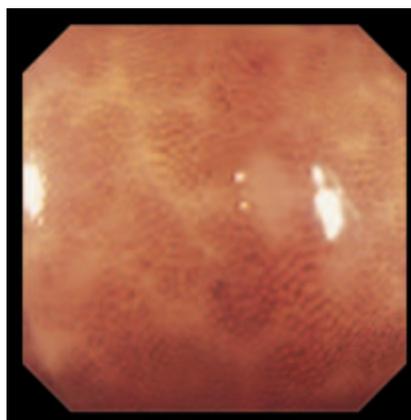
3.356



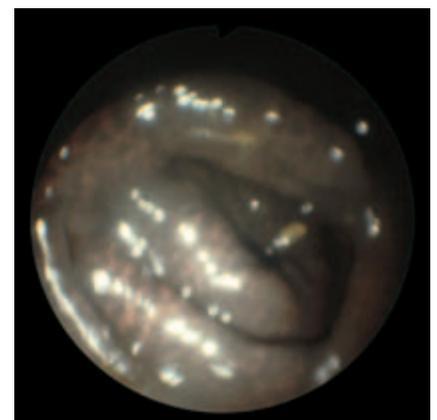
3.357



3.358



3.359

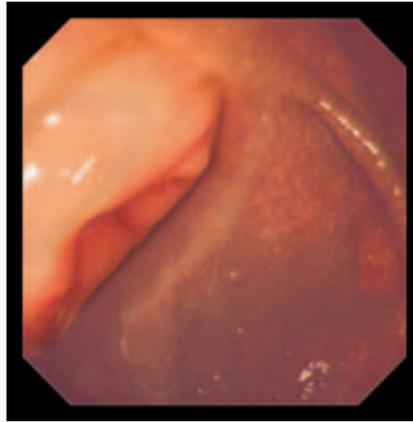


3.360

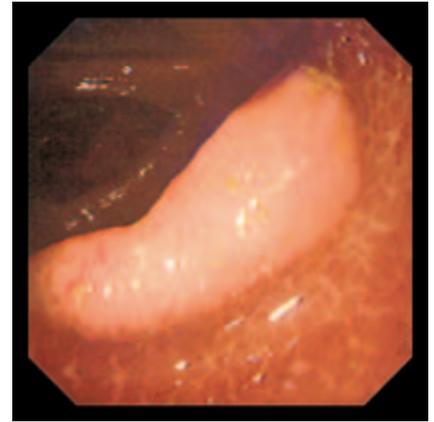
**Melanosis coli (cont.)**

The ileocaecal valve is not involved in melanosis coli (Fig. 3.361), neither is neoplastic tissue: in Fig. 3.362 there is a tubulovillous adenoma, in Fig. 3.363 three metaplastic polyps and in Fig. 3.364 a carcinoma.

Figure 3.365 shows the histopathological appearances of melanosis coli.



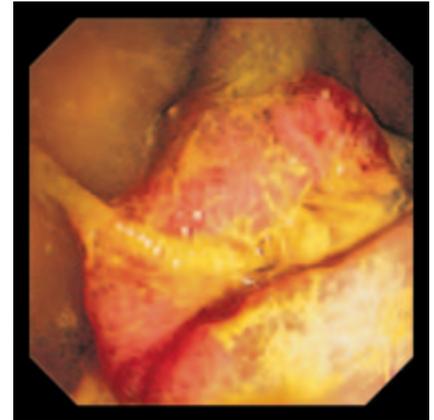
3.361



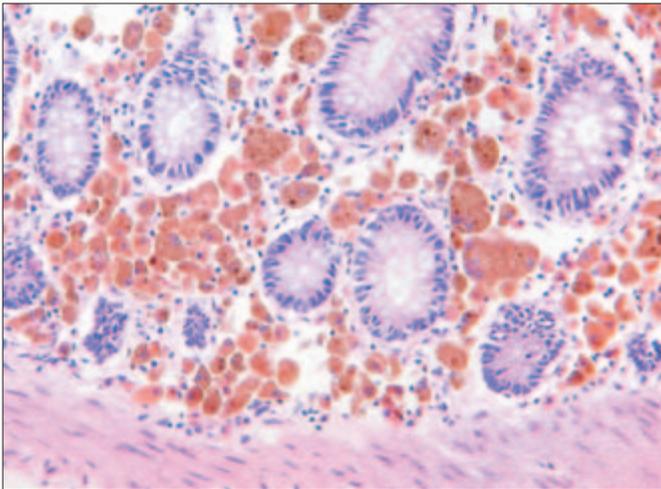
3.362



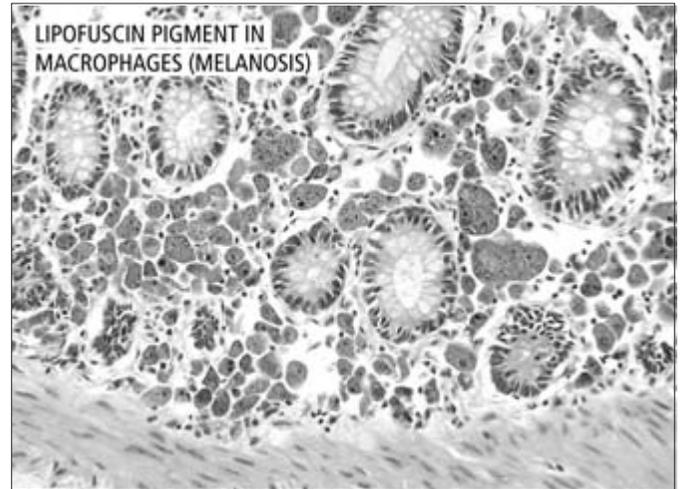
3.363



3.364

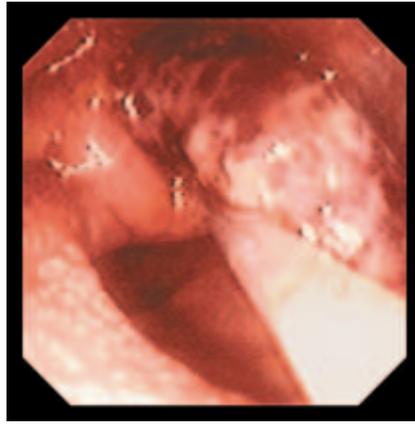


3.365

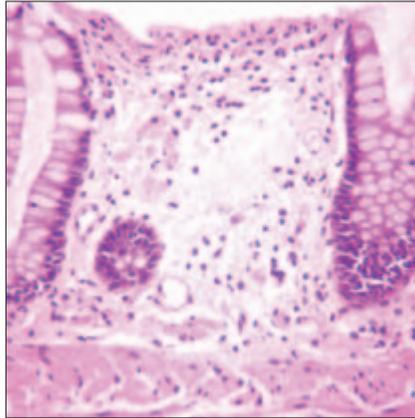
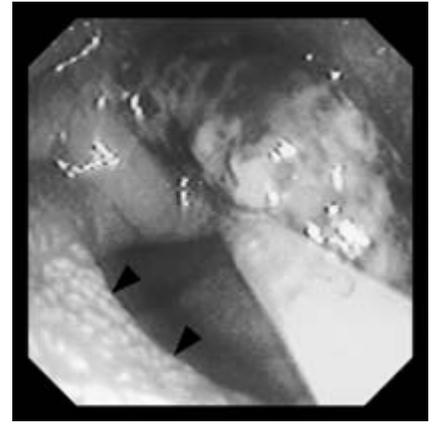


### Muciphage mucosa

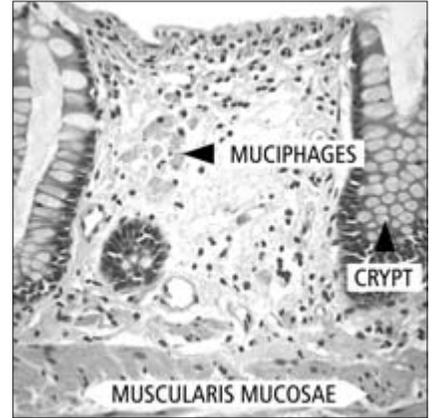
This condition is most commonly seen in association with a polyp. In Fig. 3.366 a polyp is pushed to one side with a polypectomy snare to expose the typical mottled appearances of muciphage changes in the mucosa. Figure 3.367 shows the histology.



3.366



3.367



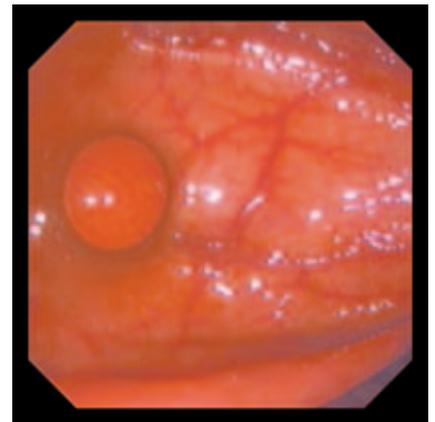
### Retained foreign bodies

It may be necessary for a patient to continue on tablet treatment despite imminent colonoscopy; alternatively the patient may not follow the usual precolonoscopy instructions or colonic preparation may be inadequate. In any case, it is not unusual to find tablets lying, apparently loose, in the caecum (Fig. 3.368) or elsewhere in the colon. A red tablet (Fig. 3.369) can easily be confused with a polyp.

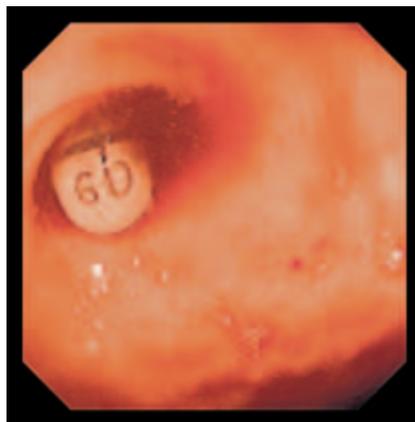
Tablets may become impacted in the appendiceal orifice (Fig. 3.370) or, more rarely, in a diverticulum (Fig. 3.371).



3.368



3.369



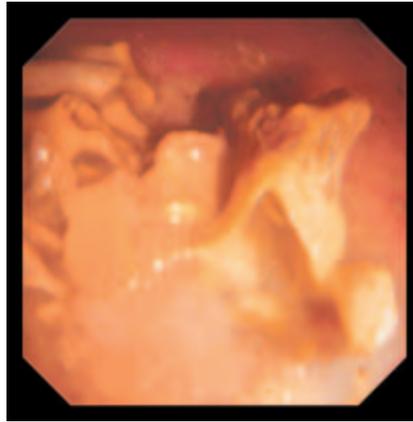
3.370



3.371

### Retained foreign bodies (*cont.*)

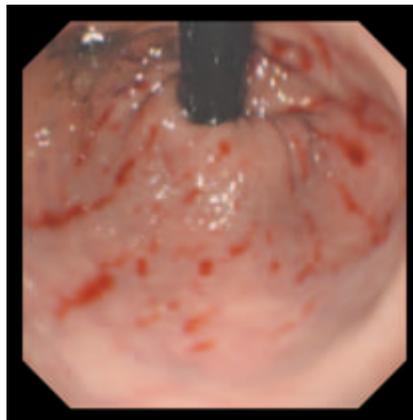
It is less common to see retained chicken bones, an appearance which has been described flippantly as Kentucky residues (Fig. 3.372).



3.372

### Faecal impaction

After removal of an impacted rectal faecal bolus, colonoscopy showed haemorrhagic mucosal tears (Fig. 3.373). The patient had presented with constipation and rectal bleeding.



3.373

### Megacolon

Sometimes unintentional retroflexion of the colonoscope occurs during colonoscopy in the absence of megacolon, especially when attempting to negotiate an acute flexure. In Hirschsprung's disease the instrument may easily be retroflexed in the dilated lumen (Fig. 3.374).

Pseudo-obstruction of the colon is an uncommon condition. Figure 3.375 shows the appearances after deflation during colonoscopy: note the crumpled, otherwise normal looking mucosa.

These conditions should not be confused with toxic megacolon (acute toxic dilatation) complicating inflammatory bowel disease when the bowel wall is thinned and friable. The presence of toxic megacolon is usually regarded as a contraindication to endoscopy, although colonoscopic aspiration of gas and fluid may be of occasional therapeutic value to deflate a dilated colon (see p. 340).



3.374



3.375

## Postoperative appearances

- Exposed suture material and staples
- Post-polypectomy scar
- Colotomy scar
- Appearances after closure of colostomy
- Colo-colic anastomosis
- Ileo-colic anastomosis
- Colo-anal pouch
- Ileo-anal anastomosis and pouch formation
- Recurrent Crohn's disease after resection
- Recurrent carcinoma at colonic anastomosis
- Post-appendicectomy appearances
- Uretero-sigmoidostomy
- Diversion colitis and proctitis
- Endoscopy of stomata

### Exposed suture material and staples

Exposed suture at colonic anastomosis (Fig. 3.376): silk suture material used in the seromuscular layers of an anastomosis may penetrate through to the lumen. An unusual appearance is shown in Fig. 3.377; small concretions have formed on unabsorbed sutures. Figure 3.378 demonstrates retained metal staples following excision of a large adenoma.

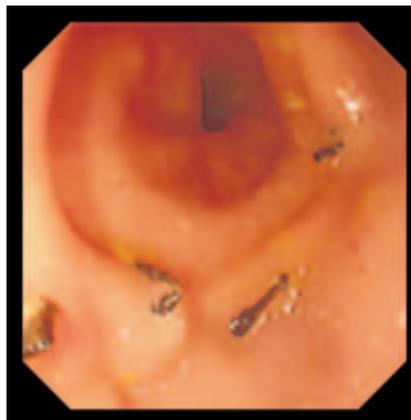
There is no clinical significance to the finding of retained suture material and staples, and no indication for their routine endoscopic removal.



3.376



3.377

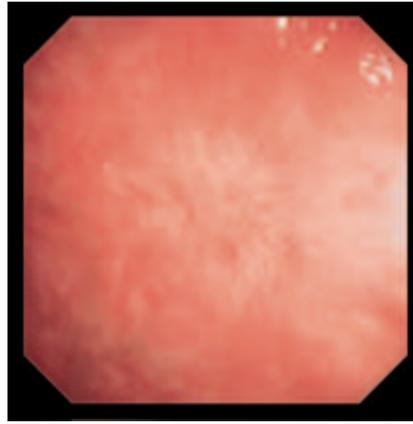


3.378

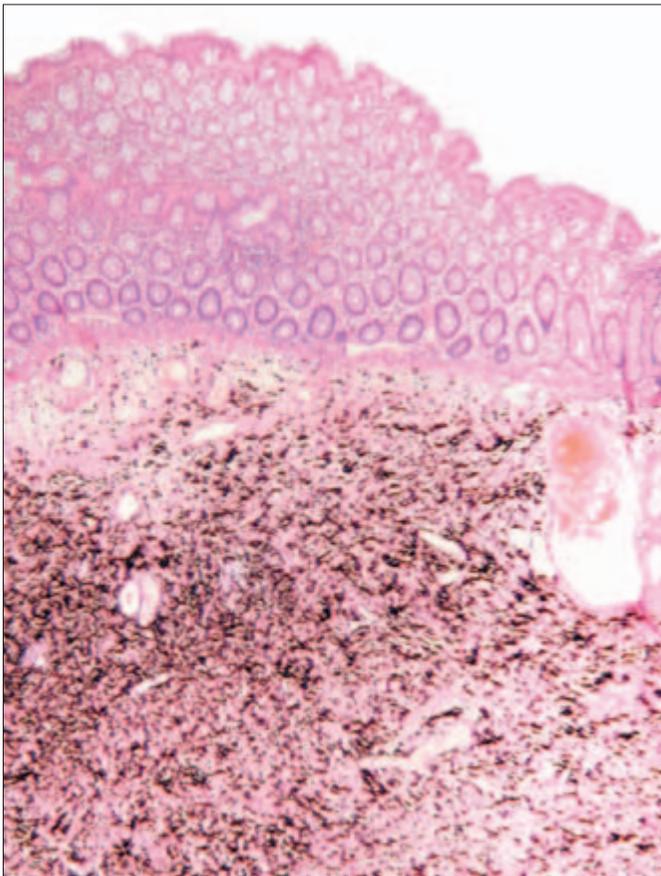
**Post-polypectomy scar**

Following colonoscopic polypectomy a flat stellate scar (Fig. 3.379) may form, reminiscent of the scar that may follow healing of a benign gastric ulcer (Fig. 2.326) (see also Fig. 3.438).

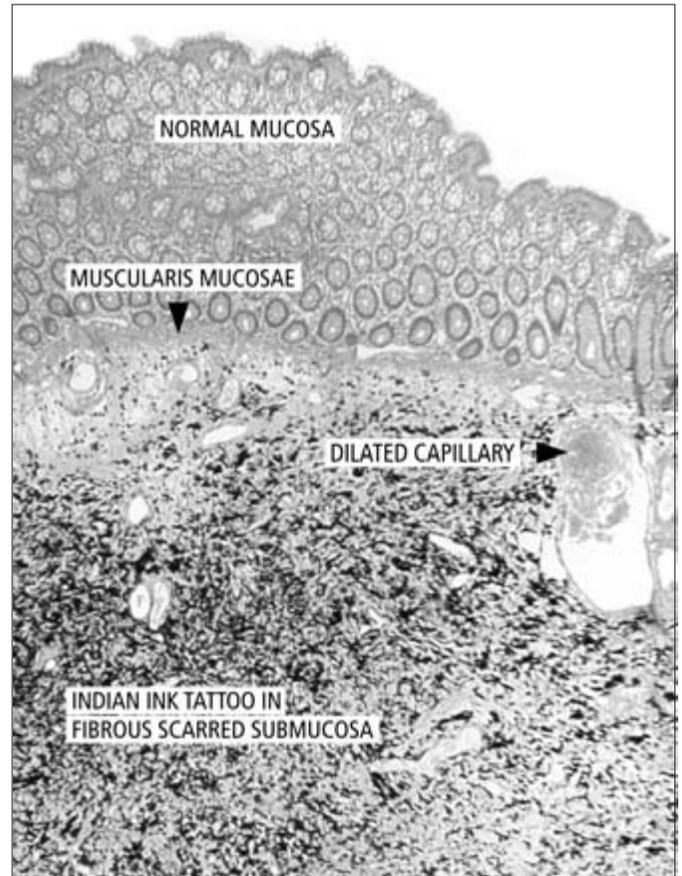
Some colonoscopists mark the site of a polypectomy with a tattoo injection. Figure 3.380 shows the histopathological appearances of a postpolypectomy scar with such ink tattooing (see also Fig. 3.439).



3.379

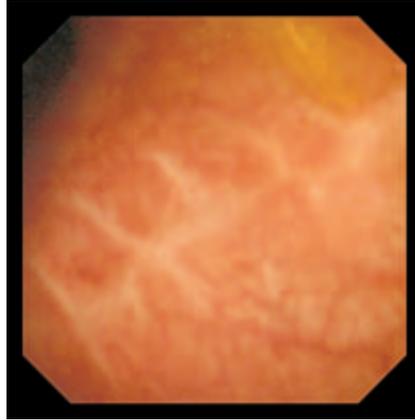


3.380



**Colotomy scar**

Figure 3.381 illustrates the appearances after simple colotomy performed for local excision of a benign lesion without segmental resection or anastomosis.



3.381

**Appearances after closure of colostomy**

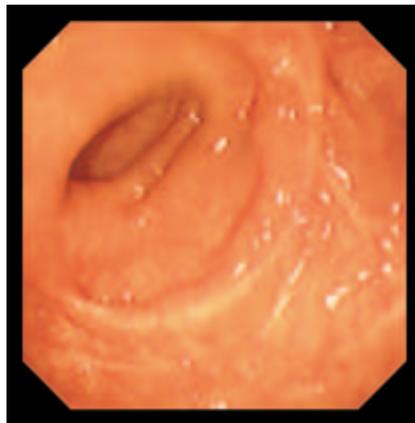
Figure 3.382 illustrates the slight narrowing and cicatricial pattern that may ensue.



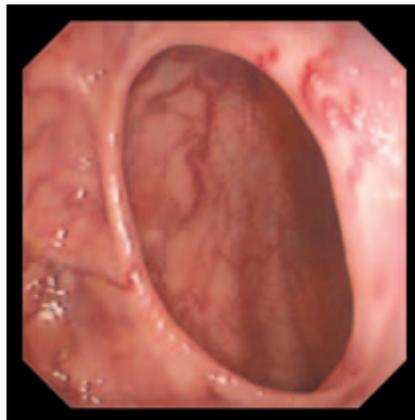
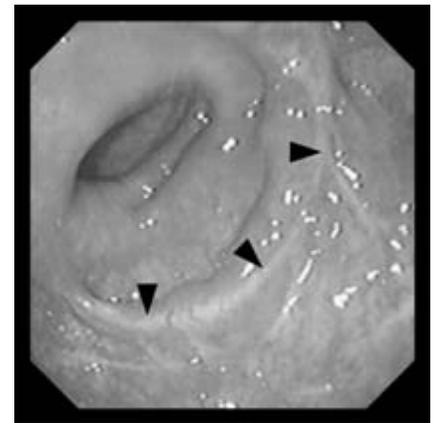
3.382

**Colo-colic anastomosis**

Healthy anastomosis after segmental colectomy for villous adenoma (Fig. 3.383): there is a smooth curvilinear scar without change in bowel diameter or mucosal pattern. Figure 3.384 shows minimal narrowing and new vessel formation, an uncommon finding. Often, the site of a previous anastomosis is only detected with difficulty, if at all.



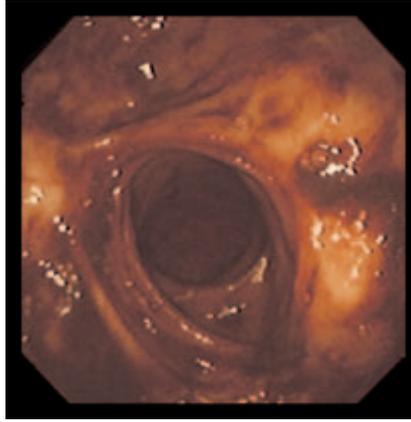
3.383



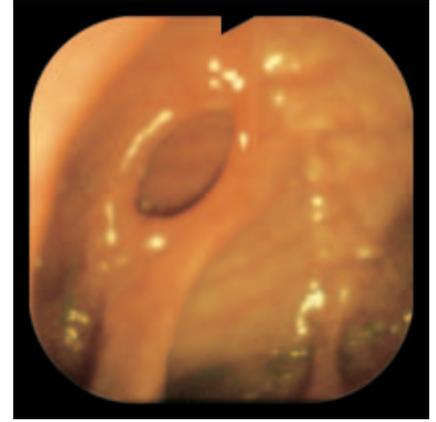
3.384

### Ileo-colic anastomosis

Figure 3.385 shows an ileo-colic anastomosis 3 years after right hemicolectomy for colonic carcinoma. Although the anastomosis is somewhat scarred there is no frank stenosis; the bowel was poorly prepared. An end-to-side ileo-colic anastomosis sometimes takes on the appearance of a patulous ileocaecal valve (Fig. 3.386).



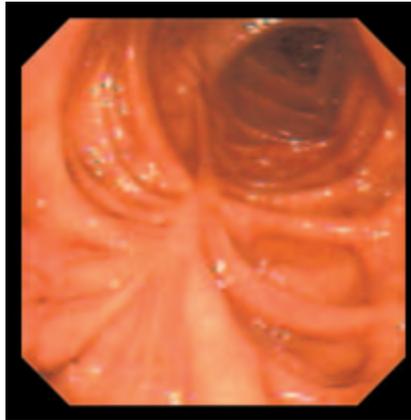
3.385



3.386

### Colo-anal pouch

Figure 3.387 demonstrates the appearance after resection of a rectal carcinoma.

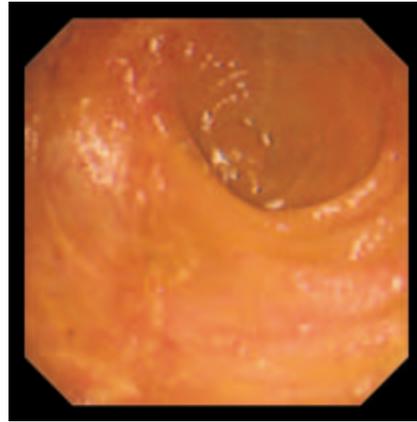


3.387

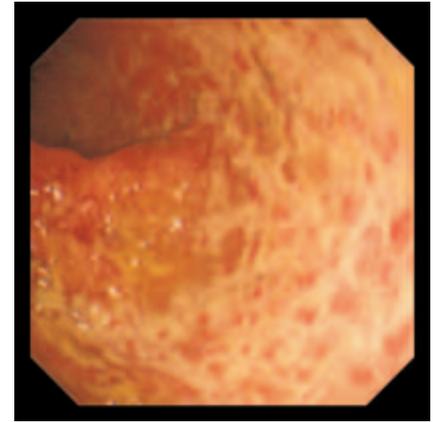
**Ileo-anal anastomosis and pouch formation**

Colonic resection with ileo-anal pelvic ileal reservoir pouch formation is now widely used in the management of familial adenomatous polyposis and ulcerative colitis, though most colorectal surgeons regard this procedure as contraindicated in Crohn’s disease.

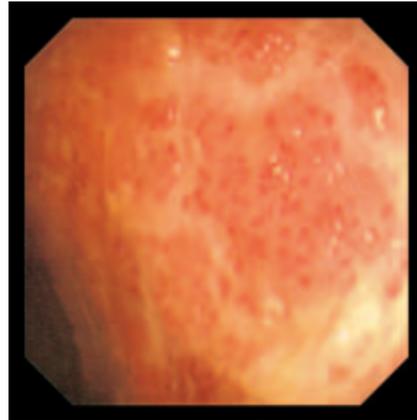
Pouchitis is a syndrome of clinical, endoscopic and histopathological findings resulting from severe acute inflammation and ulceration within the pouch. Figures 3.388–3.391 show varying degrees of pouchitis. In the same patient immediately proximal to the pouch, the histology of adaptive changes within ileo-anal pouch mucosa is shown in Fig. 3.392, while Fig. 3.393 shows pouchitis.



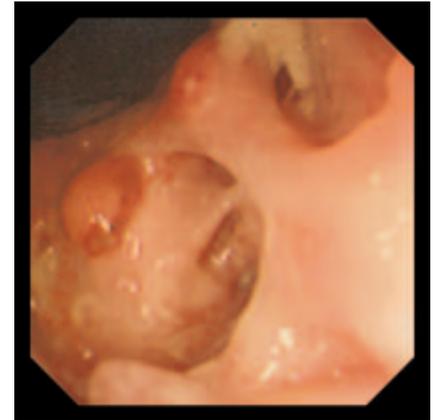
3.388



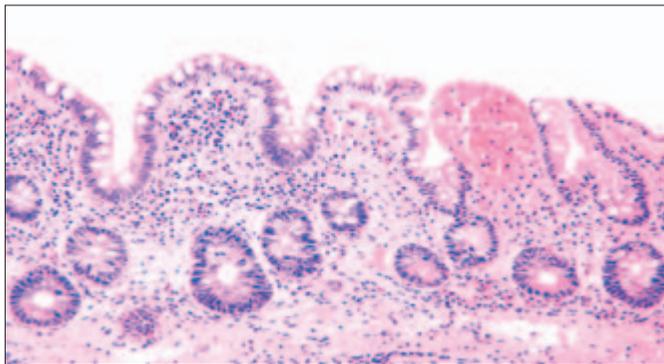
3.389



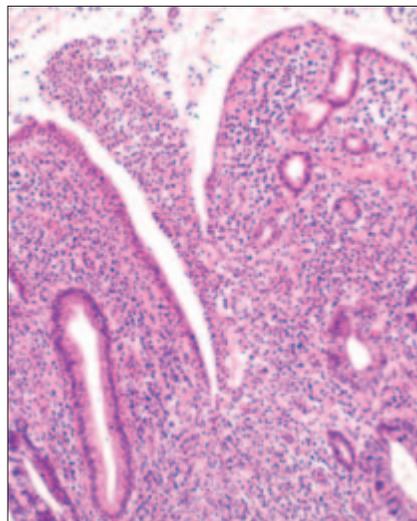
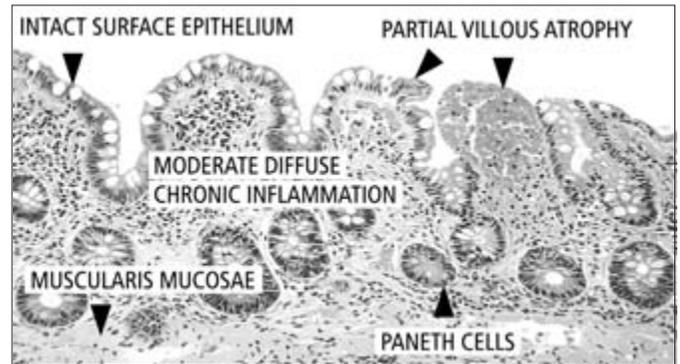
3.390



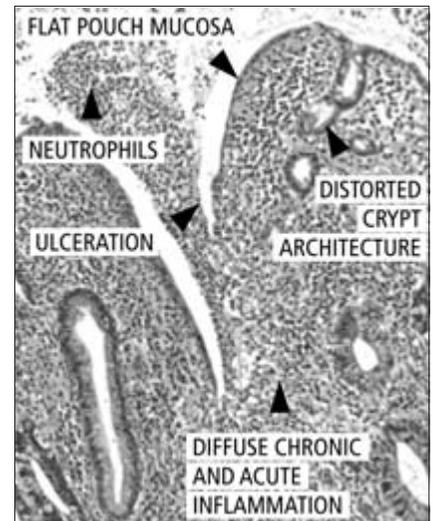
3.391



3.392



3.393



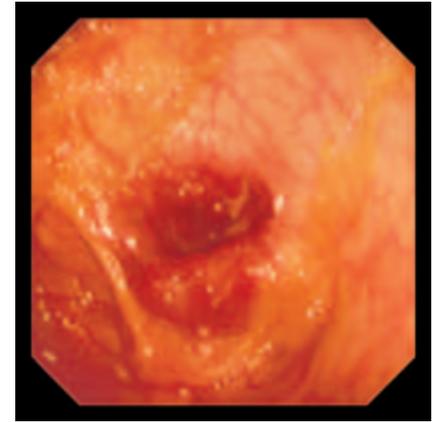
### Recurrent Crohn's disease after resection

Colonoscopy is a very effective method of detecting recurrent postoperative Crohn's disease, which frequently occurs at or just proximal to the line of surgical anastomosis. Direct visualization allows the distinction between simple anastomotic deformity or stricture, and recurrent disease. The severity of the postoperative lesions runs parallel to the clinical severity of the disease. Follow-up studies suggest that, in most cases, there is a gradual worsening of postoperative lesions with time.

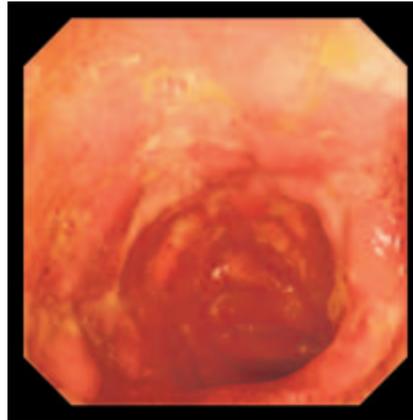
The ulceration in Fig. 3.394 has occurred at an ileocolic anastomosis without narrowing. Figure 3.395 shows an inflamed slightly narrowed ileocolic anastomosis with obvious recurrence in the ileum (Fig. 3.396).



3.394



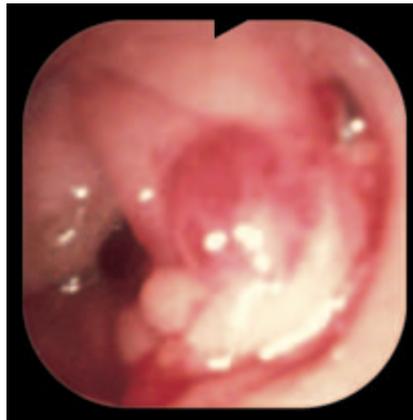
3.395



3.396

### Recurrent carcinoma at colonic anastomosis

A small piece of exposed suture material marks the site of the previous anastomosis where a carcinoma has recurred (Fig. 3.397).



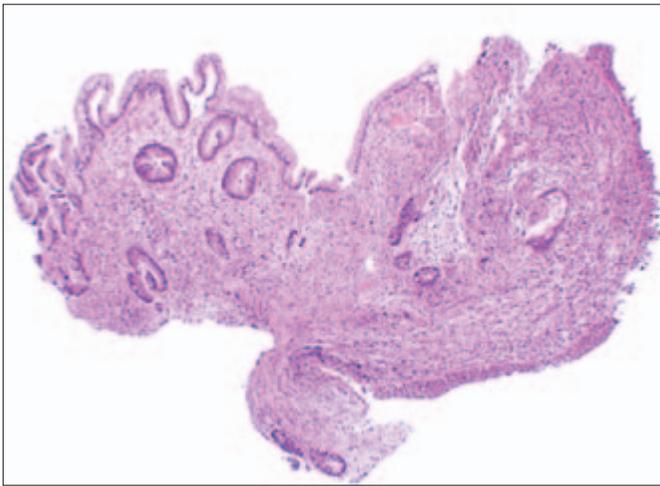
3.397

### Post-appendectomy appearances

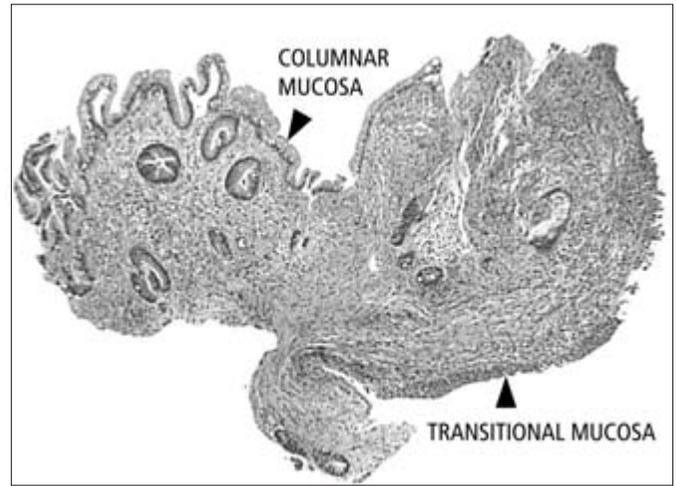
When performing an appendectomy the surgeon may leave a relatively long stump. Figure 3.398 shows inversion of such a stump into the caecum.



3.398



3.400

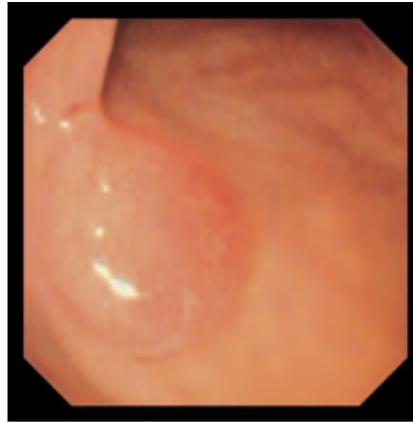


### Uretero-sigmoidostomy

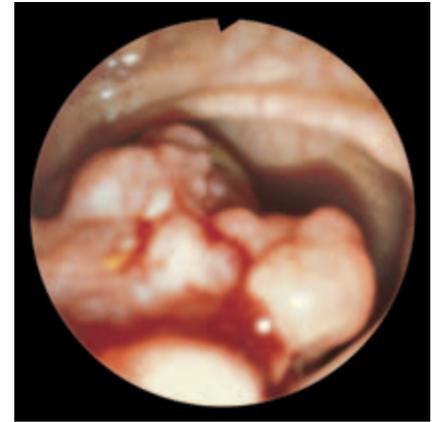
After surgical implantation of ureters into the colon the anastomotic area forms a polypoid protrusion at the apex of which an orifice may be visible (Fig. 3.399). Histologically the junction between the transitional and columnar mucosae is seen (Fig. 3.400).

Because of the propensity of this area to undergo malignant change colonoscopic surveillance has been suggested. Figure 3.401 shows a carcinoma which has developed at the site of an uretero-sigmoidostomy. Forceps biopsy is recommended as the use of a snare can result in perforation at the site of the implant.

Uretero-sigmoidostomy is no longer standard practice.



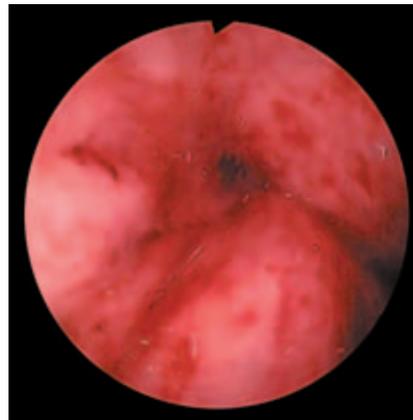
3.399



3.401

### Diversion colitis and proctitis

Diversion colitis and proctitis may result from diversion of the faecal stream via an ileostomy or colostomy in the absence of previous inflammatory bowel disease. Figure 3.402 shows the endoscopic appearances following a Hartmann procedure for colonic carcinoma;

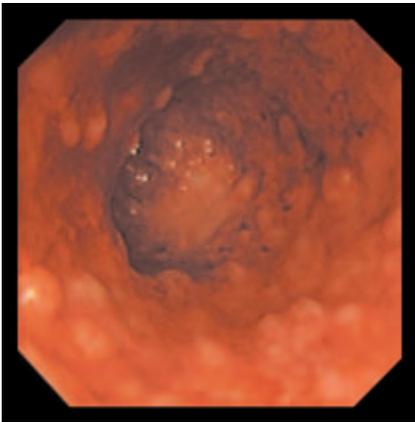


3.402

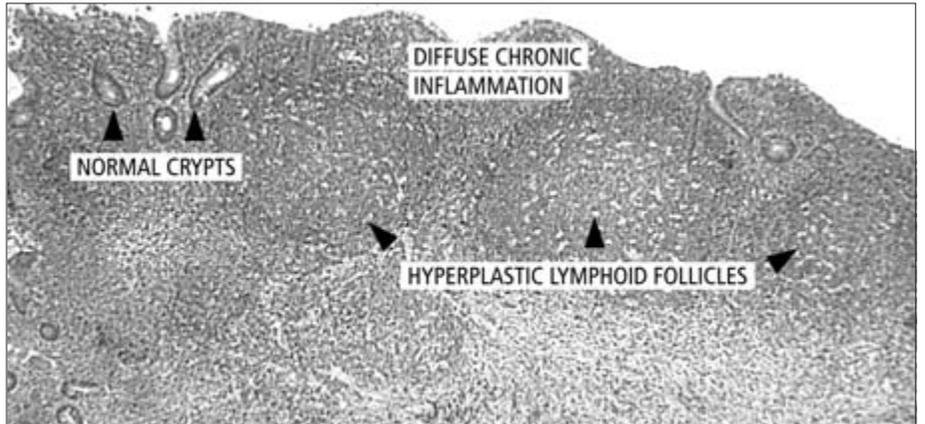
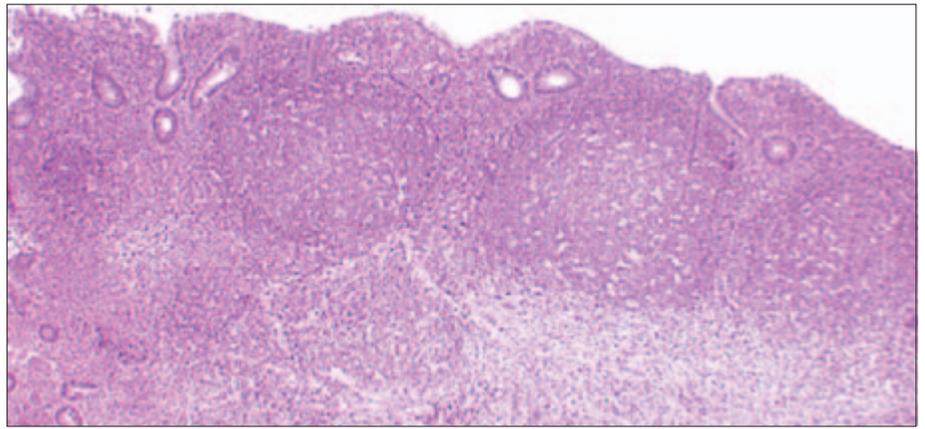
**Diversion colitis and proctitis (cont.)**

the underlying histopathological abnormalities, lymphoid follicular hyperplasia, and mild diffuse chronic inflammation in the absence of crypt architectural distortion, are illustrated in Fig. 3.403.

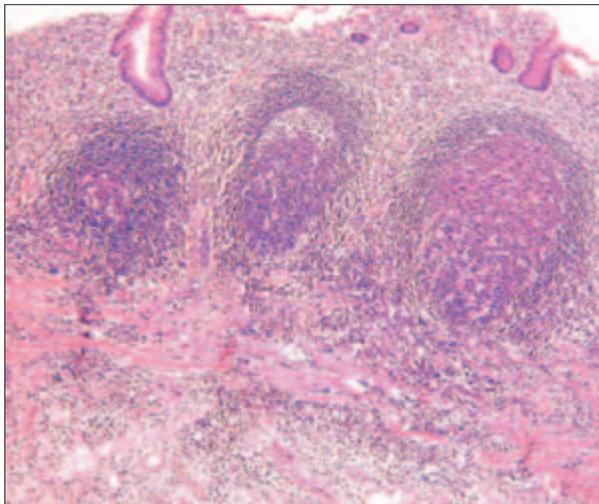
The effect of diversion in Crohn's disease is usually to ameliorate the pre-existing colonic disease, whereas in ulcerative colitis the pre-existing inflammation persists or becomes worse (Fig. 3.404) and lymphoid follicular hyperplasia is particularly prominent (Fig. 3.405).



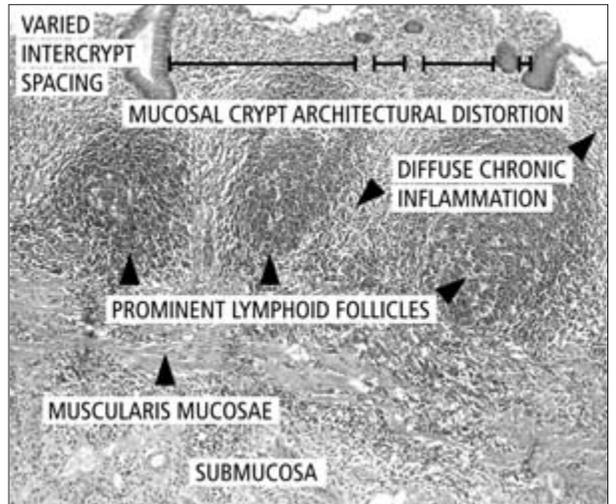
3.404

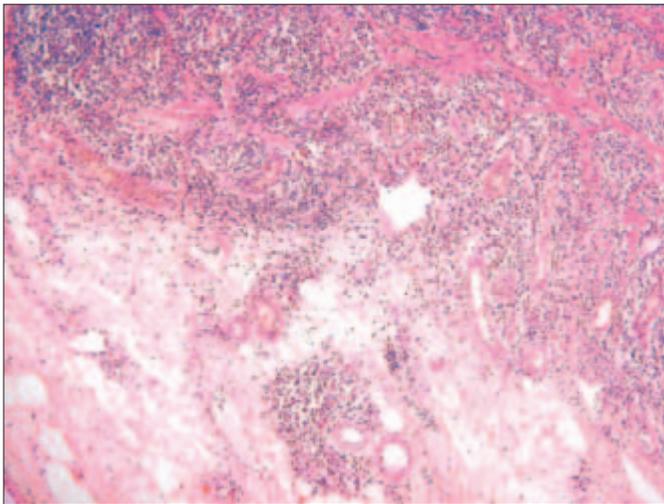


3.403

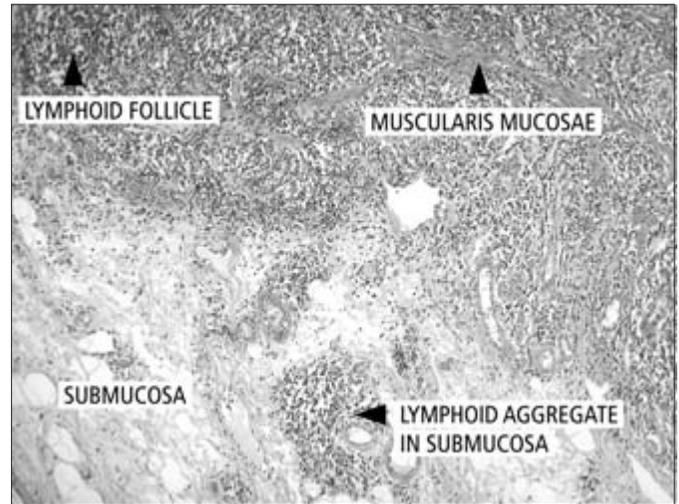


3.405





3.406



### Diversion colitis and proctitis (cont.)

When a patient with ulcerative colitis is considered for a pouch operation, one approach is the three stage pouch procedure. The first stage comprises a colectomy and the formation of a temporary ileostomy, leaving a diverted rectal stump *in situ*. The histological changes in the diverted rectal stump (Fig. 3.406) may include granulomas and transmural inflammation in the form of lymphoid aggregates, and may closely mimic Crohn's disease. It is important to realise this when selecting ulcerative colitis patients for pouch surgery, as Crohn's disease is usually regarded as a contraindication for this procedure since in most reported series the results are poor in comparison with those obtained in UC. Crohn's disease-like changes in a diverted rectum in a patient known to have ulcerative colitis do not alter the diagnosis and are not a contraindication to pouch surgery. The previous colectomy specimen and the whole patient, especially the small bowel and anus, should be studied prior to changing the diagnosis.



3.407

### Endoscopy of stomata

'Ileoscopy' and 'colonoscopy' are feasible and often valuable when performed via a stoma. Extreme caution must be exercised when examining through a stoma as narrowing and peristomal adhesion can cause technical problems. Sometimes an oblique-viewing or a narrow diameter ('paediatric') endoscope will be needed.

It may be clinically helpful to find normal ileal appearances when inspecting the ileum through an ileostomy. In Fig. 3.407 there is recurrent Crohn's disease seen through an ileostomy.



3.408

Figure 3.408 illustrates an unusual problem. There were clinical indications for an endoscopic inspection of the colon distal to a temporary colostomy. This was attempted through the stoma but severe angulation of the colon due to adhesions made full distal colonoscopy impossible. The examination was completed using a second colonoscope from below, hence the unusual face-to-face encounter.

<b>Polypectomy</b>	<b>327</b>
<b>Dilatation of strictures</b>	<b>335</b>
<b>Treatment of bleeding lesions</b>	<b>336</b>
<b>Palliation of malignant lesions</b>	<b>338</b>
<b>Decompression of megacolon</b>	<b>340</b>
<b>Removal of foreign bodies</b>	<b>340</b>

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**Polypectomy****Instrumentation and procedure****Larger polyps****Smaller polyps****Mucosal resection****Post-polypectomy appearances****Retrieval of resected polyps****Bleeding after polypectomy**

In general, complete removal and full histological examination are essential to confirm the diagnosis of all colonic polypoid lesions. Various exceptions to this rule will be found in the following section; for example, modified policies will dictate the approach to submucosal and very small polyps.

Using a snare loop and coagulation diathermy this is easily accomplished in the case of pedunculated polyps of up to 2 cm in diameter, which constitute the majority of such lesions.

Sessile polyps of 5 mm or less are best treated with the Williams 'hot biopsy' forceps. Slightly larger sessile lesions can be tented on to a pseudopedicle by snaring and lifting away from the mucosa before sectioning with diathermy. Flat or depressed 'polyps' may be removed by the technique of saline injection polypectomy or endoscopic mucosal resection. Large polyps, whether sessile or pedunculated, present special problems and caution is required lest cavalier attempts at polypectomy result in perforation, haemorrhage or diathermy injury to the bowel wall.

Besides obtaining tissue for diagnosis, polypectomy provides an effective method of treatment for adenomatous polyps and contributes to colorectal cancer prevention.

Detailed considerations of technique are beyond the scope of this Atlas but a selection of representative images is included.

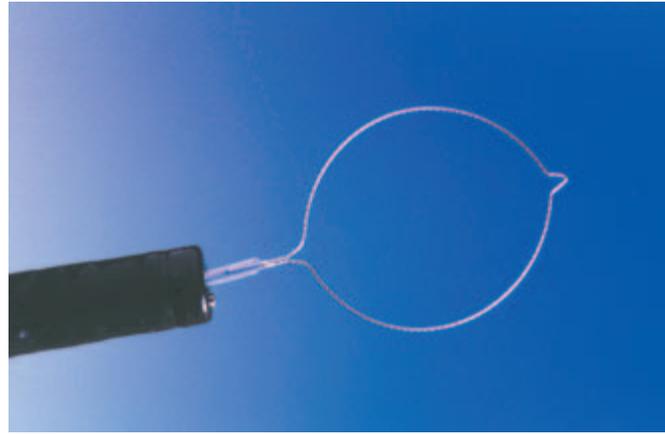
### Instrumentation and procedure

Figure 3.409 shows a braided wire snare loop protruded from its insulation sheath after passage through the operating channel of the colonoscope. Current is supplied by a specialized endoscopic diathermy unit with contacts for endoscope earthing. Manufacturers' instructions should be followed carefully. Standard high-power surgical diathermy units are quite unsuitable for endoscopic work.

It is useful to have available a variety of snares differing in size and shape. Simple loops are suitable for most purposes, but rotatable snares may be helpful when positioning is difficult and an eccentric snare may be better if working in a confined space. A snare with sharp serrations on the inner aspect of the loop, the so-called 'crown of thorns' snare, is able to bite into the smooth mucosa surrounding tissue elevated by submucosal saline injection (see p. 332).

Before commencing polypectomy it is essential to ensure that (a) the view is good with a stable endoscope position; (b) free fluid remaining from preparation is aspirated; and (c) air or CO<sub>2</sub> insufflation is adequate to maintain the view. Loss of vision during polypectomy is frustrating and potentially hazardous.

In the following sequence a 1 cm polyp has been chosen so that the view of the snare is not obscured as so often occurs with larger lesions. The snare is first fully opened beyond the polyp (Fig. 3.410) and the loop manipulated so that it loosely surrounds the polyp stalk (Fig. 3.411). Next the snare is gradually tightened and its position adjusted by advancing the insulating sheath until it touches the stalk just below the polyp head (Fig. 3.412). When the position is satisfactory the snare is further tightened, but with care not to apply so much force that the snare wire 'cheese pares' the polyp stalk. At this stage darkening or cyanosis of the polyp head is commonly observed (Fig. 3.413). The polyp is then lifted away from the mucosa and short bursts of low power coagulation current are applied.



3.409



3.410



3.411



3.412



3.413

**Instrumentation and procedure (cont.)**

Care must be taken to avoid polyp or wire contact with the opposite side of the colonic lumen as a contralateral burn could result. When blanching of the stalk is observed (Fig. 3.414) the snare is slowly tightened as further short bursts of current are applied. Gradually the stalk is coagulated and sectioned. Too rapid tightening of the snare or too high a current may result in rapid section of the stalk and inadequate coagulation with consequent risk of haemorrhage.

To complete the process of polypectomy the resected polyp must be retrieved (see p. 334) and sent to the laboratory for histological examination.

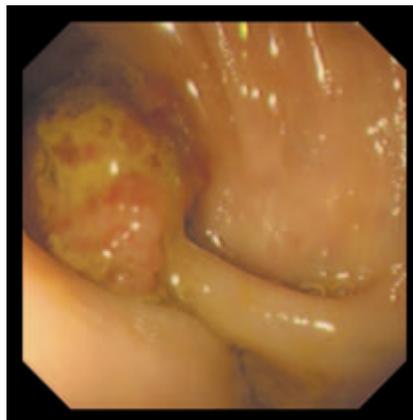


3.414

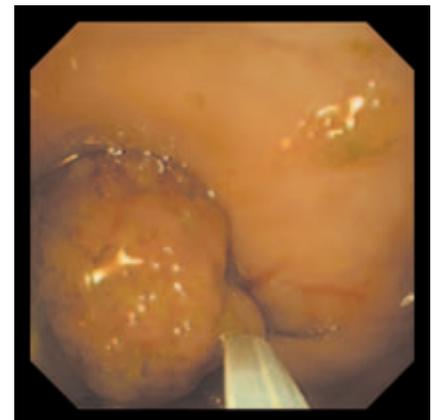
**Larger polyps**

Larger stalked polyps are treated in a manner similar to that previously illustrated (Figs 3.410–3.414) but there may be difficulties with snare placement, and due to the size of the polyp much of the loop may be hidden from view. Caution must be exercised when considering endoscopic removal of large or broad-based polyps. Over-ambitious polypectomy must be resisted: detailed considerations are beyond the scope of this work. Occasionally a snare is placed over a polyp and the decision is made for some reason not to resect. Usually it is possible to disengage the snare by opening widely and jiggling so that it comes free. Rarely the snare has cut in too deeply to allow this and a dilemma results—proceed with attempted polypectomy or remove the endoscope and seek surgical assistance? Sometimes the best plan is simply to remove the snare handle leaving the loop over the polyp and withdrawing the endoscope and proceeding to surgical polypectomy or segmental resection of the colon.

Figure 3.415 shows a larger stalked polyp before and Fig. 3.416 after application of a snare (further views of this case during later stages of polypectomy are shown in Figs 3.434 and 3.435).



3.415



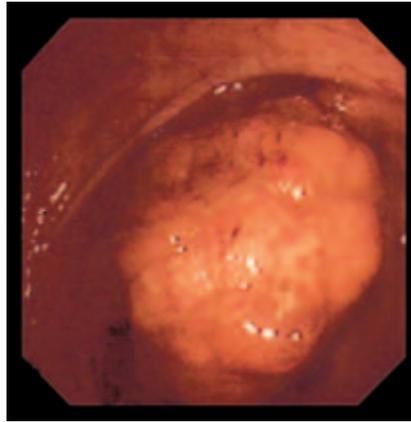
3.416

**Larger polyps (cont.)**

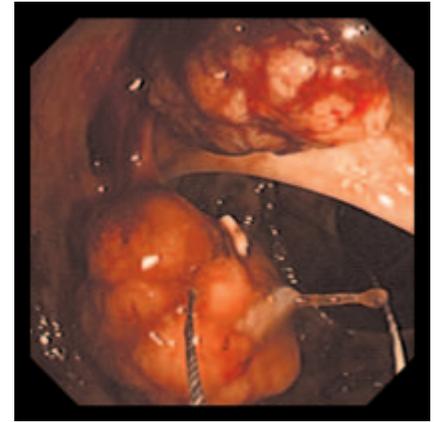
Some large broad based polyps (Fig. 3.417), as long as they are mobile and not fixed to the deeper layers, may be suitable for piecemeal polypectomy. The polyp head is sectioned gradually (Fig. 3.418) until the base or stalk can be clearly seen (Fig. 3.419) and then safely ensnared to allow complete removal (Fig. 3.420). Sometimes it is better if vision is poor to interrupt the procedure, completing the polypectomy a few days later.

A flatter lobulated polyp is shown in Fig. 3.421 and the large area denuded of mucosa after piecemeal polypectomy, revealing the underlying muscularis mucosae, is seen in Fig. 3.422.

It goes without saying that every effort must be made to retrieve all resected material. An important disadvantage of piecemeal polypectomy is that destruction of resected tissue may make histological assessment difficult and can even mask malignant change.



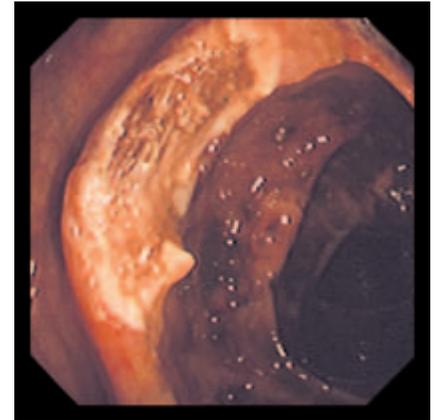
3.417



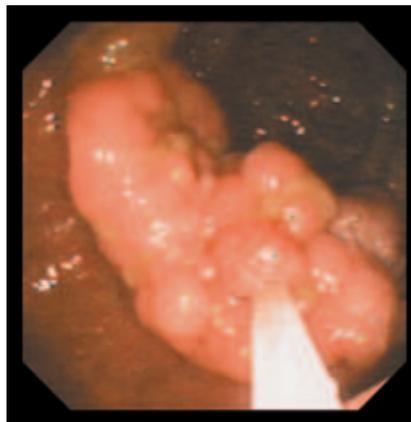
3.418



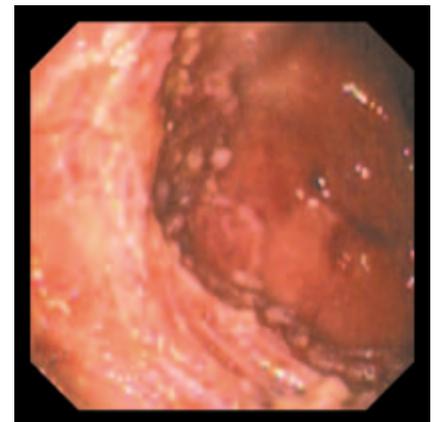
3.419



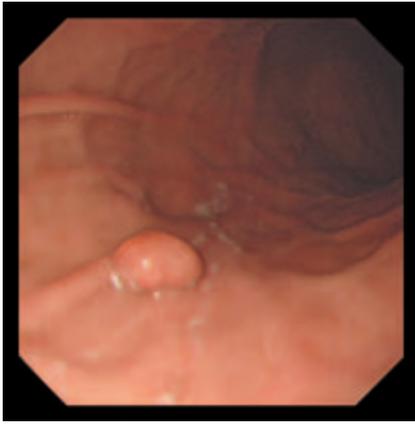
3.420



3.421



3.422



3.423



3.424



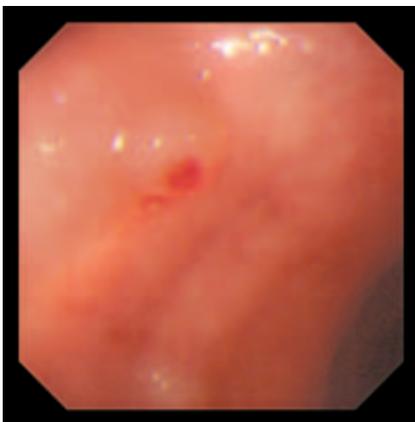
3.425

### Smaller polyps

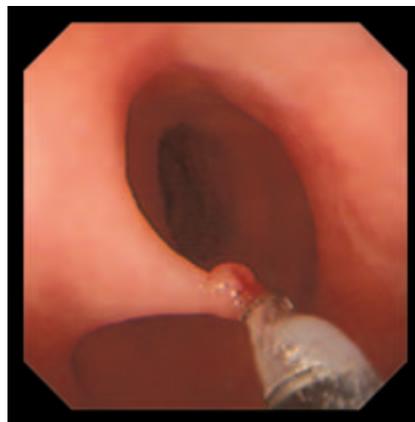
Polyps of about 5 mm in diameter or less (Fig. 3.423) may be removed by snare (Figs 3.424 and 3.425) but more easily with Williams 'hot biopsy' forceps (Fig. 3.426), which consist of a metal diathermy forceps insulated within a plastic sheath. The polyp (Fig. 3.427) is grasped firmly and lifted, tensioning the pedicle, or creating a pseudo-pedicle (Fig. 3.428) in the case of sessile polyps. Coagulation diathermy current causes blanching of the stalk (Fig. 3.429) and separation at its narrowest point giving the so called 'Mount Fuji' appearance. The tissue contained within the forceps cups is not damaged, and so is suitable for histological examination. A word of caution: necrosis following the use of hot biopsy forceps may be deeper than colonoscopic views suggest; very rarely necrosis may be transmural resulting in perforation.



3.426



3.427



3.428



3.429

### Mucosal resection

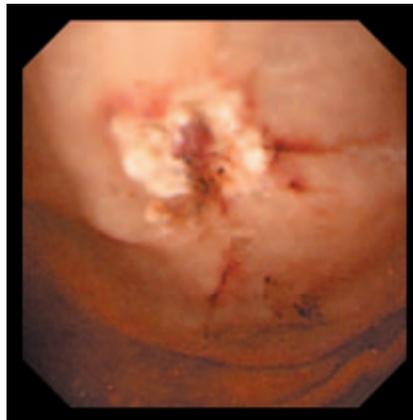
An alternative technique for removal of unstalked, flat or barely elevated polyps is saline injection polypectomy or mucosal resection. This takes advantage of the fact that normal mucosa may be separated from the muscular coats by injection of fluid beneath the mucosa causing a truly mucosal lesion to 'float' clear of the underlying muscle layers (Fig. 3.430). (In this instance a small dose of adrenaline 1 : 10 000 was added to the saline, hence the blanched appearance of the pedestal.) The first step is to raise the lesion on a bed of saline by submucosal injections starting beyond the further margin so as to tip the lesion into vision rather than obscure it from view. A standard endoscopic injector is used and several punctures are made with injections around the lesion. If this 'floats' satisfactorily it may then be excised either by grasping with a normal diathermy snare (Fig. 3.431) or a 'crown of thorns' snare (see p. 328), or cutting around the lesion with a needle knife (Fig. 4.163). Should the lesion not lift satisfactorily it suggests either that it originates or extends more deeply or that invasive malignancy has developed making resection by this method unwise and potentially dangerous. Figure 3.432 shows the appearance after successful complete mucosal resection. Submucosal injection of saline may also be usefully employed to facilitate piecemeal polypectomy of large broad based lesions as in Fig. 3.421. This manoeuvre is, in addition, appropriate for harvesting larger mucosal samples.



3.430



3.431



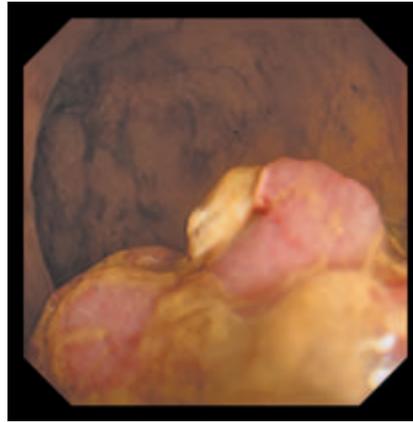
3.432

### Post-polypectomy appearances

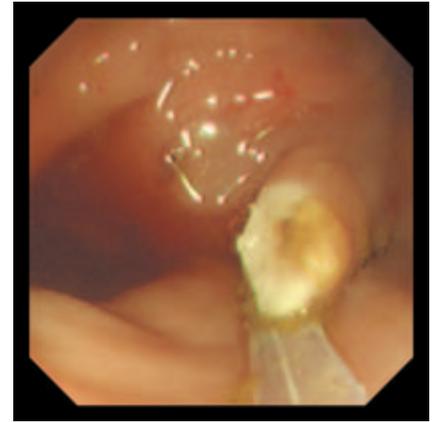
Immediately after resection, polyps usually fall free into the lumen (Fig. 3.433). The coagulated stalk, often with thrombosed blood vessels, is clearly visible (Fig. 3.434). Sometimes the polyp head adheres temporarily to its coagulated stalk despite complete division (Fig. 3.435). It is easily removed by the snare without further current. Free-lying polyps quickly become cyanosed, and so can usually be distinguished from those still receiving a blood supply.

Subsequently involution of the stalk occurs (Figs 3.436 and 3.437) and the site eventually heals with scarring (Fig. 3.438, also Fig. 3.379). Before healing an ulcer may persist at the polypectomy site for 1–2 weeks.

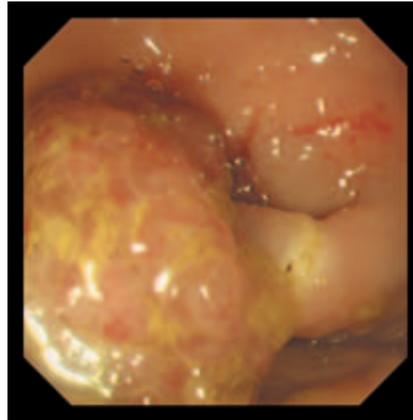
To mark the site of a resected polyp for later re-inspection either endoscopic or surgical, a small quantity of Indian ink may be tattooed into the adjacent mucosa (Fig. 3.439) at the time of polypectomy. It is also helpful to the surgeon and pathologist if the endoscopist marks the site of excision on an excised and retrieved polyp with Indian ink (see p. 14).



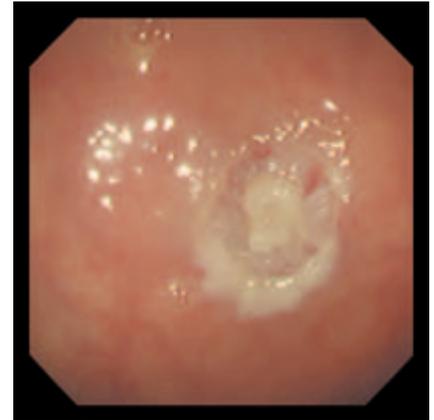
3.433



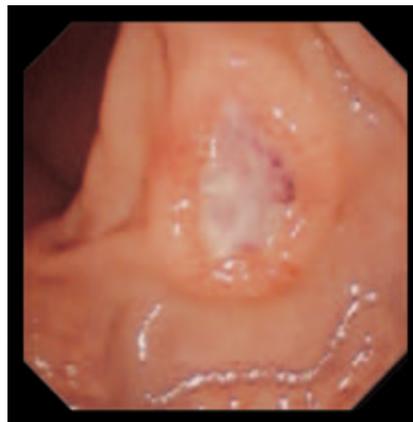
3.434



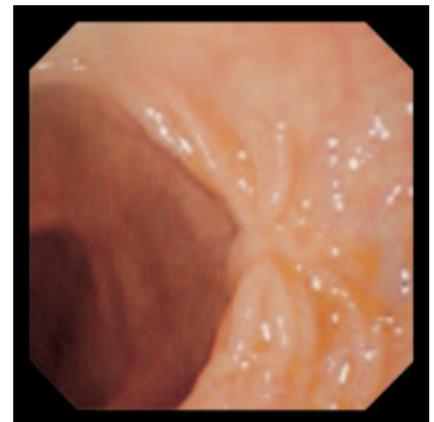
3.435



3.436



3.437



3.438



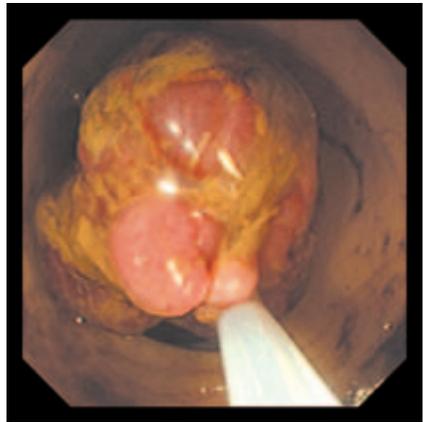
3.439

### Retrieval of resected polyps

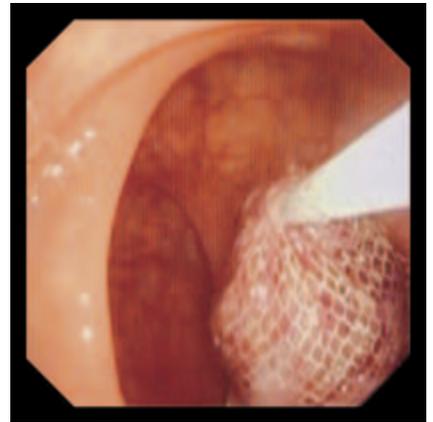
Various types of grasping forceps (Figs 2.672 and 3.440) can be used to retrieve the resected polyp although it is usually a simple matter to capture a free lying polyp with the snare which was used for its removal (Fig. 3.441). A wire basket or a special net (Fig. 3.442) for collecting multiple polyps are useful alternatives. Very small polyps may be aspirated through the biopsy channel into a trap placed in the suction line. A larger polyp can be sucked on to the end of the colonoscope as it is withdrawn although this obscures the view so is not suitable unless the examination in other respects has been completed. When many polyps have been resected an alternative method is to collect and filter through gauze the faecal fluid which is voided after colonoscopy is complete. To aid this procedure a phosphate enema may be given through the colonoscope before withdrawal.



3.440



3.441



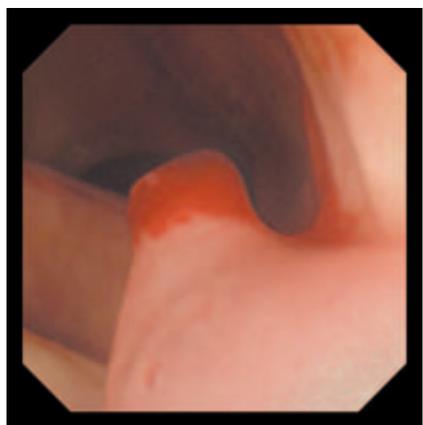
3.442

### Bleeding after polypectomy

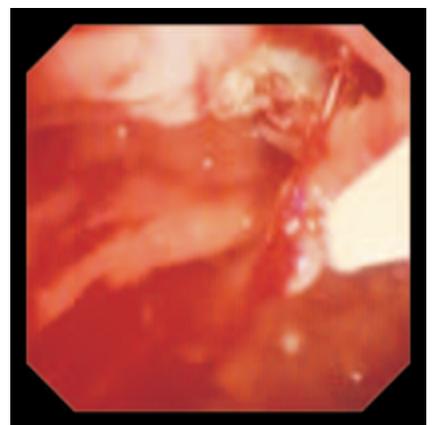
Haemorrhage may result if the snare loop is tightened too rapidly without applying adequate current to coagulate the vessels in the stalk. This complication is more likely when the power setting is too high, if cutting current is used or with polyps on a thick pedicle.

In Fig. 3.443 there is oozing from a polyp pedicle. Control of haemorrhage can usually be obtained by re-snaring the residual stalk and applying a gentle squeeze for several minutes; current is not applied lest further section of the stalk results. Locally applied iced water, vasoconstrictors or haemostatic drugs can also be employed.

Figure 3.444 shows brisk arterial bleeding following polypectomy when



3.443



3.444

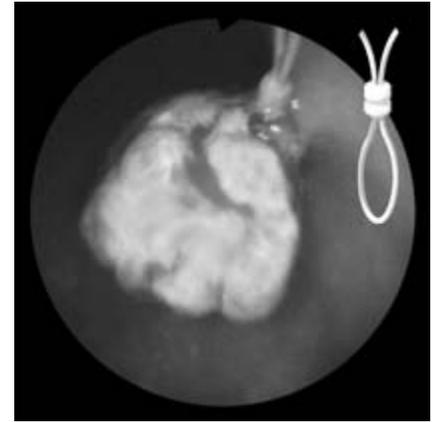
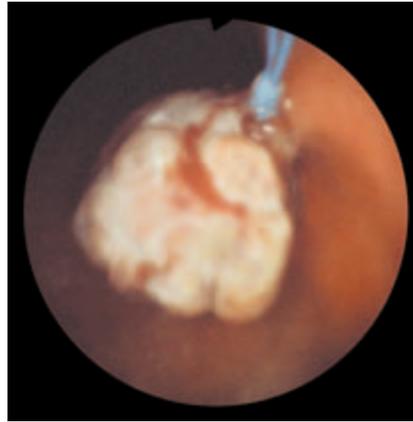
the stalk was cut too short giving little chance of endoscopic control of bleeding.

Post-polypectomy bleeding is most likely to occur immediately, or some

hours after the procedure. Rarely it may supervene after an interval of some days.

### Bleeding after polypectomy (cont.)

Methods possibly reducing the risk of haemorrhage include preliminary application of an Endoloop™, a device which is placed around the polyp stalk before polypectomy and tightened to occlude the blood supply and which remains in place after resection of the polyp (Fig. 3.445). Another approach is to inject 1–2 mL of 1 : 10 000 adrenaline into the polyp stalk to induce vasoconstriction before applying the diathermy current.

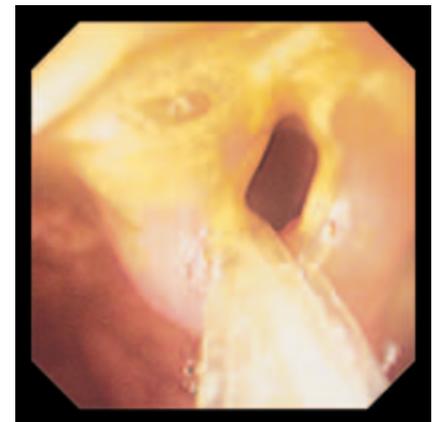
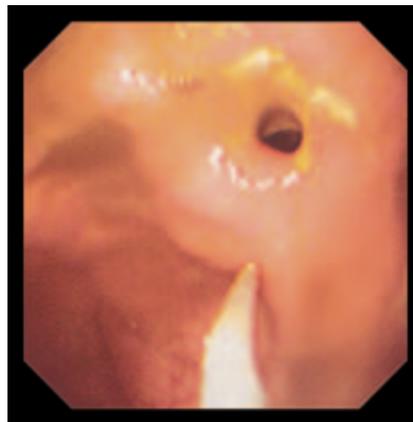


3.445

### Dilatation of strictures

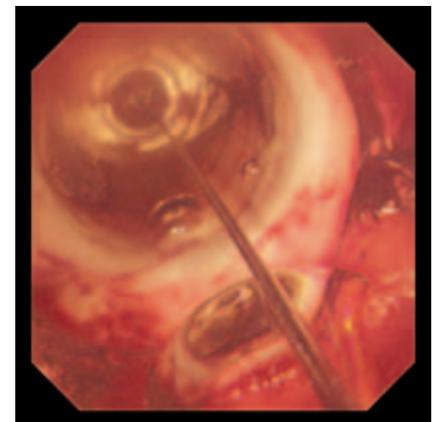
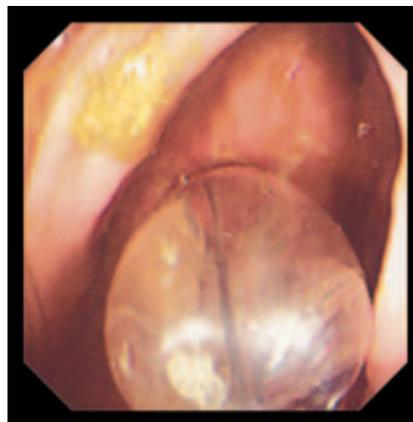
#### Dilatation of strictures

Balloon dilatation of benign strictures in the colon is as appropriate, although less well established, as dilatation of such strictures in the upper gastrointestinal tract (see pp. 200–201). At any level accessible to a colonoscope dilatation by the through-the-scope (TTS) technique is possible. This allows treatment of postoperative anastomotic strictures and fibrous or inflammatory strictures associated with inflammatory bowel disease. Figures 3.446–3.450 show balloon dilatation of a short anastomotic stricture with a TTS balloon. Radiographic screening during the procedure helps to establish that full dilatation has been achieved should it not be possible to pass the colonoscope through the lumen after dilatation. The view into a stricture, through the balloon during dilatation, is well seen in Fig. 3.449.



3.446

3.447



3.448

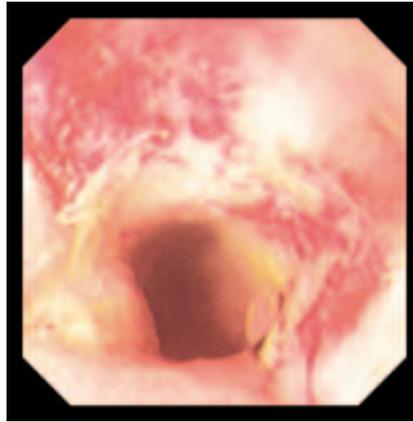
3.449

### Dilatation of strictures (*cont.*)

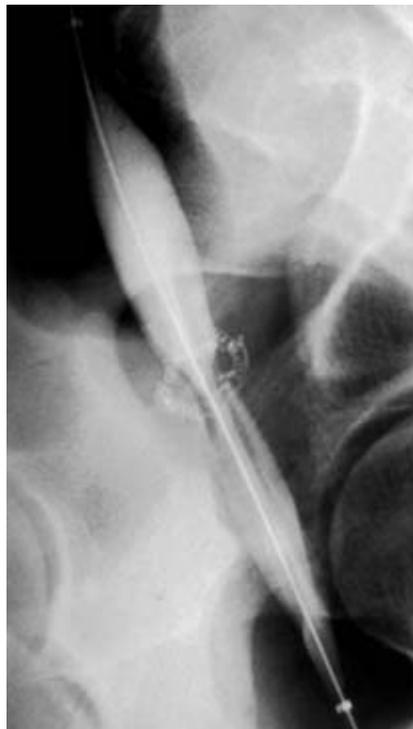
Fig. 3.450 shows the typical appearances after balloon dilatation.

Low anastomotic strictures following anterior resection are readily treated using a balloon catheter passed over a guide wire. Figure 3.451 shows a balloon catheter placed within a postoperative stricture: the ring of metal staples at the anastomosis is clearly seen. After dilatation (Fig. 3.452) the walls of the balloon are shown to be parallel and the ring of staples is expanded.

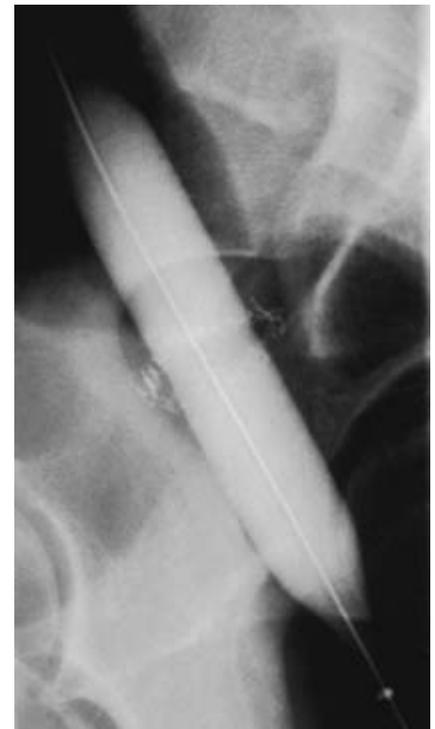
Often in this situation a single dilatation is effective in allowing normal defaecation but repeat procedures may be required. The presence of staples surrounding a postoperative stricture should not normally be regarded as a contraindication to balloon dilatation.



3.450



3.451



3.452

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## Treatment of bleeding lesions

### Fulguration

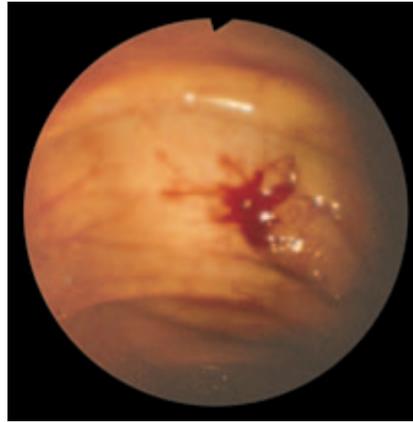
### Laser treatment

### Argon plasma coagulation (APC)

Colonic vascular lesions responsible for overt or occult bleeding may be considered for endoscopic treatment by the methods described for upper gastrointestinal haemorrhage (see pp. 208–219). However, as the colon is thinner walled, particularly in the caecum, there is a greater risk of transmural injury. Caution is thus mandatory in the use of any of these techniques lest damage is inflicted: surgical resection may be the safer option for extensive lesions.

### Fulguration

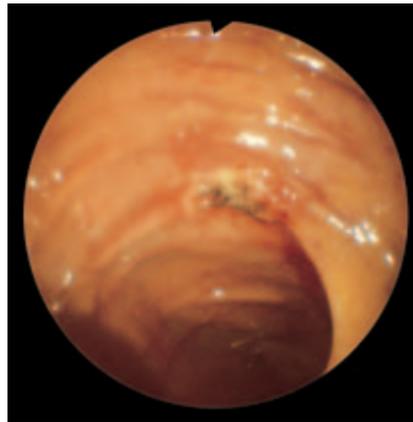
Small lesions (Fig. 3.453) can be obliterated using 'hot biopsy' forceps (Fig. 3.454). The vascular mucosa is gently picked up at the edge of the lesion, and a short burst of coagulation current applied. It is preferable to treat at more than one site rather than to attempt complete coagulation from a single place. Coaptation, folding the lesion upon itself with the diathermy forceps whilst applying slight pressure, may aid obliteration of vessels but carries a risk of transmural injury. After coagulation the whole area appears whitened without significant bleeding (Fig. 3.455). Any residual vessels can be treated by a further application of current.



3.453



3.454

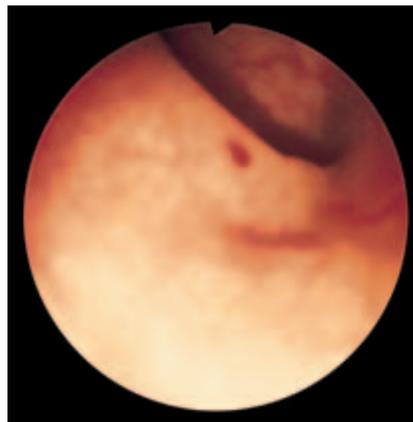


3.455

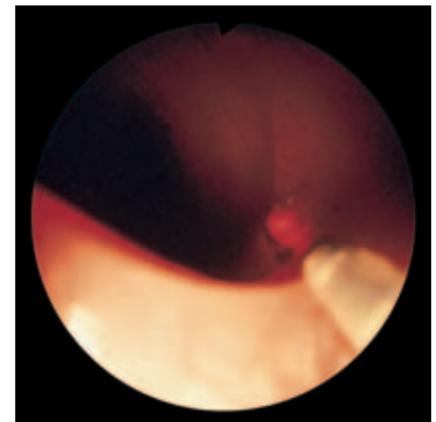
### Laser treatment

The Nd-YAG laser has been used extensively for treatment of vascular lesions in the upper gastrointestinal tract (see p. 216), and to a lesser extent in the colon.

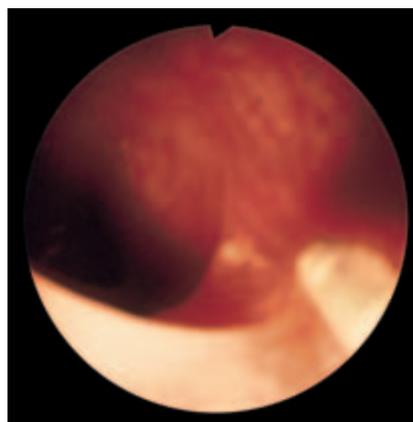
A colonic telangiectasis before treatment is shown in Fig. 3.456. Figure 3.457 illustrates the use of the aiming beam (cf. Fig. 2.720). The white area in Fig. 3.458 is the site of the lesion after exposure to the laser beam.



3.456



3.457



3.458

### Argon plasma coagulation (APC)

Vascular lesions in the colon, like those in the upper gastrointestinal tract (see Figs 2.777 and 2.778), may be obliterated by APC. There may be a greater margin of safety coupled with a greater ease of use as the depth of penetration is less than that of the laser beam, and by contrast with the hot biopsy method no pressure is applied. APC is increasingly favoured for treatment of angiodysplasia and angiomas.

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## Palliation of malignant lesions

### Laser treatment and argon plasma coagulation (APC)

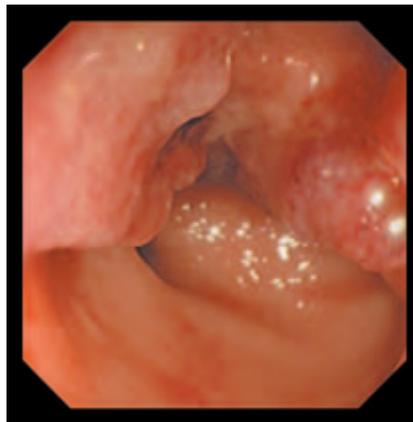
#### Expanding metal stents

Malignant obstruction of the colon may be treated by methods similar to those employed for oesophageal carcinoma (see pp. 201–207). Definitive curative treatment is surgical but if surgery is deemed inappropriate endoscopic measures may sometimes be used. Additionally, urgent endoscopic treatment of obstructing colon cancer may be a helpful preliminary by relieving the obstruction before elective surgical resection is undertaken. If resection is not possible palliation may be effected in some cases by repeated treatment with laser, argon plasma coagulation (APC), alcohol injection or by placement of an expanding metal stent.

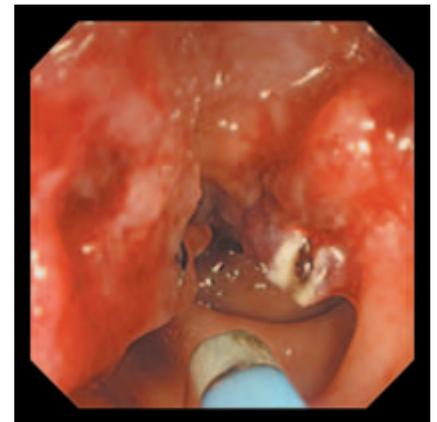
### Laser treatment and argon plasma coagulation (APC)

The Nd-YAG laser can be used for tumour destruction (as in the upper gastrointestinal tract pp. 202–203), enlarging the lumen to relieve obstruction and possibly permitting satisfactory defaecation. APC is an alternative method of addressing the same problem but as penetration is shallower it may be less successful for larger lesions although appropriate for earlier cases (Figs 3.459–3.461).

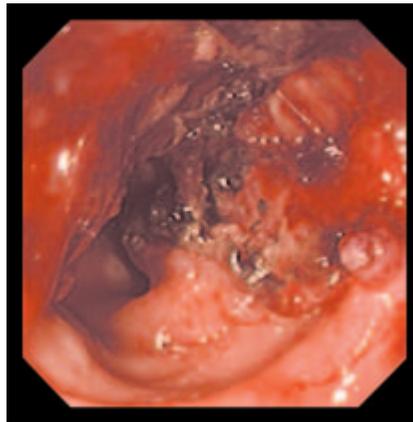
APC and laser treatment may also occasionally be appropriate for benign tumours unsuitable for polypectomy by standard methods, or residual tissue remaining after snare polypectomy.



3.459



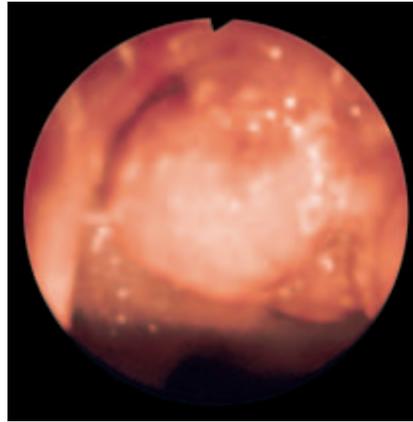
3.460



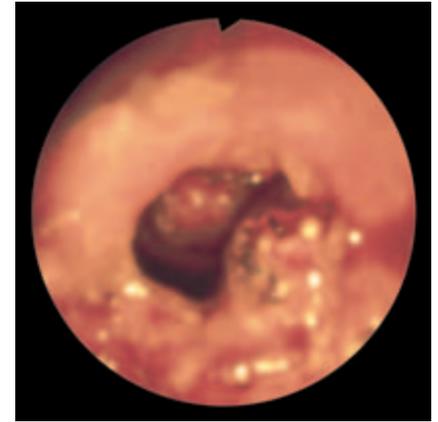
3.461

**Laser treatment and argon plasma coagulation (APC) (cont.)**

Figures 3.462 and 3.463 show a villous adenoma of the rectum before and after laser treatment. A second untreated tumour can be seen further up the bowel in Fig. 3.463. Multiple small adenomas occurring in the residual rectum of patients with familial adenomatous polyposis may be ablated with laser or APC after total colectomy and ileo-rectal anastomosis.



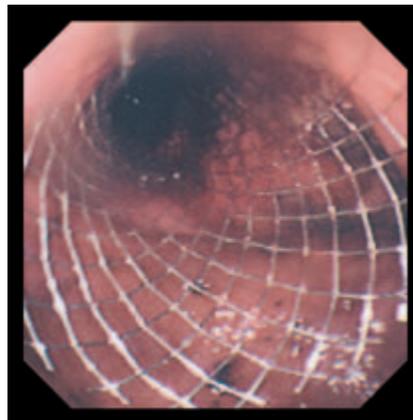
3.462



3.463

**Expanding metal stents**

Placement of an expanding metal stent through an obstructing malignant stricture may offer valuable palliation. With current delivery systems only lesions in the rectum and sigmoid are accessible (Fig. 3.464). Expanding metal stents are discussed in more detail on p. 203.



3.464

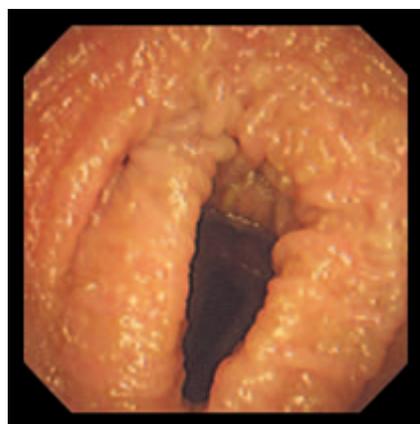
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## Decompression of megacolon

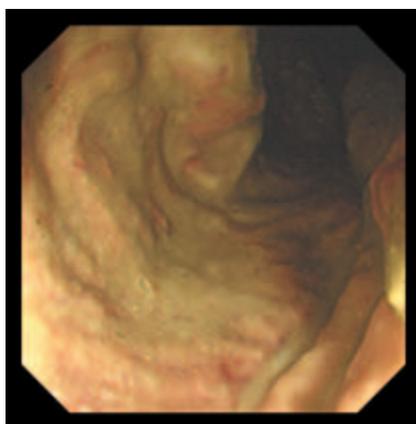
### Decompression of megacolon

Distension of the colon by gas and fluid may sometimes be effectively treated by colonoscopic aspiration and/or intubation. Sigmoid volvulus or intestinal pseudo-obstruction initially respond well to these techniques although recurrence or progression of the disease may necessitate alternative measures.

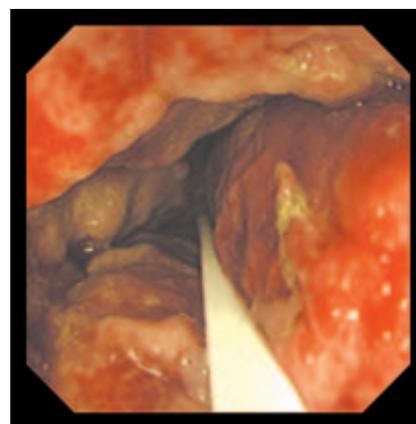
Figure 3.465 shows a colon partially decompressed in a case of pseudo-obstruction. Large quantities of mucus as well as gas had been aspirated and the mucosa shows a crenated appearance after relief of the distension. Acute dilatation of the colon in inflammatory bowel disease may very exceptionally be decompressed in a similar manner but as the colonic wall is intensely inflamed and at risk of perforation surgical treatment is preferred. However in antibiotic-induced (pseudomembranous) colitis colonoscopic decompression may prevent progression of the disease and perhaps pre-empt surgery. Figure 3.466 shows active colitis after decompression and Fig. 3.467 a guide catheter left behind after withdrawing the colonoscope. Over this fine catheter a larger tube may be deployed to allow continual deflation until the acute episode resolves or alternative therapy is undertaken.



3.465



3.466



3.467

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## Removal of foreign bodies

### Removal of foreign bodies

The problem of foreign bodies is commoner in the upper than in the lower gastrointestinal tract. Suffice it to say that relatively few foreign bodies retained in the colon and rectum lend themselves to colonoscopic extraction. Where an attempt at endoscopic removal seems appropriate, similar methods to those described on pp. 187–196 should be considered. Surgery is indicated if there is a perforation. Otherwise operative peranal instrumental extraction under spinal or general anaesthesia is usually successful. Prior anal dilatation may be necessary. In view of the large variety of (usually inserted) foreign bodies that may be found in the lower bowel, a wide range of appropriate instruments including obstetric forceps should be available.

## Complications

It is essential for the colonoscopist to have a good knowledge of the complications of both diagnostic and therapeutic procedures, and how to avoid them. Only a small proportion of such complications lend themselves to endoscopic photography.

The commonest serious complications related to colonoscopy are electrolyte imbalance and dehydration due to inadequate rehydration during preparation with cathartics. Additional nonendoscopic complications are possible over-sedation and associated hypoxia. Strenuous efforts must be made to ensure appropriate levels of sedation and adequate oxygenation controlled by close monitoring during the procedure.

### Mucosal damage

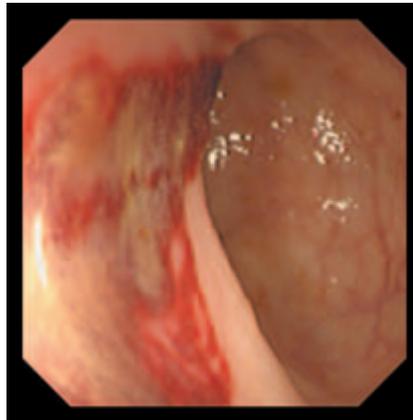
### Perforation

### Explosions

### Post-polypectomy bleeding

### Mucosal damage

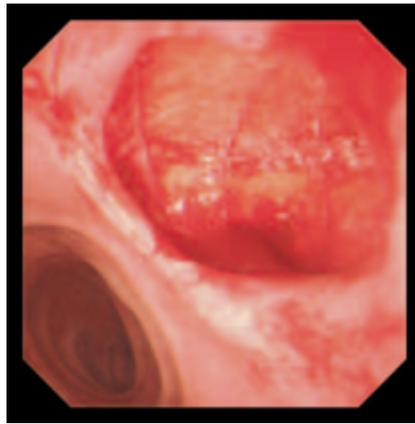
Advancing the endoscope relatively forcibly, especially when the view is not entirely clear, may lead to mucosal damage. Figure 3.468 shows a partial mucosal tear in the rectum only recognized on withdrawing the colonoscope.



3.468

### Perforation

Perforation of the colon, particularly in the sigmoid region, is the most frequent instrumental complication of diagnostic colonoscopy. It results from excessive stretching of the sigmoid when a large loop is formed, although introduction of the colonoscope tip into, or over-inflation of, a diverticulum can cause rupture. Figure 3.469 illustrates perforation of a low diverticulum sustained during inversion of the colonoscope tip in the rectum. Rare examples of right-sided perforation have been recorded, probably due to gaseous over-distension. Bruising of the sigmoid mesentery or rupture of its vessels may occasionally result from excessive looping. Damage to the spleen and liver have also been reported. Fixity of colonic loops in the pelvis, particularly following gynaecological surgery, predisposes to perforation or tearing of peritoneal adhesions or other trauma; such cases therefore demand extra caution.



3.469

### Explosions

Explosions during diathermy procedures have occurred on rare occasions. These are due to inflammable bowel gases (hydrogen and methane) resulting from poor preparation or bacterial action on mannitol or other bowel evacuants.

### Post-polypectomy bleeding

Polypectomy can result in bleeding (Figs 3.443 and 3.444), a transmural burn with peritonism or, rarely perforation.

Other therapeutic methods have their own uncommon complications, such as perforation during dilatation of strictures and bowel wall necrosis following laser therapy. Certain complications have already been mentioned and, as appropriate, illustrated in the relevant sections of this chapter.

# Endoscopic Retrograde Cholangiopancreatography

<b>Instrumentation</b>	<b>345</b>
<b>Endoscopic appearances commonly encountered</b>	<b>346</b>
<b>Abnormal endoscopic appearances</b>	<b>350</b>
<b>Technique of cannulation</b>	<b>355</b>
<b>Pancreas</b>	<b>358</b>
<b>Biliary system</b>	<b>383</b>
<b>Therapeutic procedures</b>	<b>419</b>
<b>Complications</b>	<b>451</b>

Besides the direct methods of examining the gastrointestinal lumen, endoscopy provides a valuable means of investigating and treating diseases of the pancreas and biliary system. The technique of endoscopic retrograde cholangiopancreatography (ERCP) complements other imaging procedures such as endoscopic ultrasonography (EUS), computerised tomography (CT), magnetic resonance imaging (MRI) including magnetic resonance cholangiopancreatography (MRCP), percutaneous transhepatic cholangiography (PTC) and radionuclide scanning.

ERCP provides excellent radiological images of the biliary and pancreatic ductal systems but gives less information about the size and shape of the organs themselves. It is demanding of personnel both in terms of number and technical skill, and is usually limited to hospitals with well developed endoscopy services. As a difficult technique it should only be undertaken by endoscopists expert in both routine diagnostic and therapeutic upper gastrointestinal endoscopy. The procedure is generally conducted in the department of radiology as high quality image intensification and facilities for taking good radiographs are essential. To facilitate best use of radiological equipment and optimal interpretation of results a radiologist should ideally be present during ERCP. If this is not possible, an expert radiographer is acceptable provided he or she has urgent access to a radiologist for guidance if required. This chapter includes endoscopic and radiographic appearances, emphasising the contributions of both disciplines but does not give descriptions of all aspects of technique.

ERCP is performed under light conscious sedation with pharyngeal anaesthesia as usually employed for OGD. However, as the duration of the procedure is longer the drug doses employed are generally higher. The position of the patient is important and different from other endoscopic examinations. As radiography employs the

prone position, the patient is prepared for this by starting off in the left lateral position but with the left arm behind the body. This allows the patient easily to be rolled prone when required.

A side-viewing duodenoscope is used to locate and enable cannulation of the papilla of Vater which is situated on the medial wall of the descending duodenum. A shaped plastic catheter previously filled with radiological contrast medium is passed through the operating channel of the endoscope and directed at the apex of the papilla. The biliary and pancreatic ducts usually have a common orifice into which the cannula is passed. Contrast material is injected under fluoroscopic control and radiographs are taken as appropriate. Care should be taken to use the correct strength of contrast material. Initially full strength is employed to define which duct has been entered and may be used for completion of a normal sized pancreatogram. If however, a dilated pancreatic duct or biliary system is entered, it is advisable to use a diluted contrast agent to avoid obscuring small calculi or other subtle abnormalities. With the tip of the cannula in the papilla both ducts may fill simultaneously, while deeper penetration achieves selective opacification of one duct or the other. Repositioning of the cannula is required to demonstrate the duct which was not entered initially.

ERCP is essentially a procedure undertaken with the specific objective of pancreatic and/or biliary duct cannulation for diagnosis and therapy. A full upper gastrointestinal examination cannot simultaneously and satisfactorily be performed with a side-viewing instrument although excellent views may be obtained of the distal stomach and duodenum.

The indications for ERCP have been broadened with the increasing use of laparoscopic cholecystectomy. It facilitates preoperative diagnosis and definition of anatomy, and can be used for removal of common duct stones. Often endoscopic removal of ductal stones is combined with laparoscopic cholecystectomy to obviate the need for 'open' surgery, the procedures usually being performed on separate occasions.

Magnetic resonance cholangiopancreatography (MRCP) is a non-invasive alternative to ERCP for demonstration of the bile ducts and main pancreatic duct. It can be combined with magnetic resonance imaging (MRI) of the surrounding structures and vessels. MRCP has the advantage of showing the ducts and segments beyond a stricture with no risk of iatrogenic infection or pancreatitis. ERCP offers better resolution of very fine details, and MRCP cannot be used for the assessment of drainage or flow without the injection of secretin. It can be performed on some patients when ERCP is not possible or does not provide adequate diagnostic data. MRI-based procedures are contraindicated in patients with pacemakers and some implanted surgical devices or with metal fragments in the eye or other vulnerable areas.

**Instrumentation**

Figure 4.1 shows the distal ends of standard and large channel side-viewing duodenoscopes with ERCP cannulae projecting from the operating channels. To obtain good views of the papilla of Vater suitable for cannulation a side-viewing endoscope is essential: end-viewing endoscopes only show the papilla in profile, if at all, as it is often masked by valvulae conniventes. To pass a cannula through a side-viewing endoscope a forceps raising bridge (elevator) is necessary. This also acts as an additional aid to manipulation.

Many ERCP cannulae are banded at the tip to indicate the depth of penetration and may be radio-opaque (often with a metal insert) to enhance fluoroscopic identification.

Figure 4.2 shows various cannulae: standard, fine-tip, tapered, and ball-tipped. Sometimes the tip of the standard cannula (1.7 mm) is too wide to allow insertion, for example if the papilla is stenosed or when it is necessary to cannulate the accessory papilla (Fig. 4.15). In such situations a fine, tapered or metal tipped cannula may be successful. The disadvantage of using these smaller cannulae routinely is that the pointed end may cause local trauma with bleeding or mucosal dissection, occasionally leading to the formation of a false passage. These complications are rarely hazardous to the patient but may result in the endoscopist's view being obscured, so preventing a satisfactory procedure. Moreover narrow cannulae may restrict the use of guidewires which are so often employed for therapeutic techniques.



4.1



4.2

**Approaching the papilla 346**

**Normal and accessory papilla 348**

**Duodenal diverticula 349**

### Approaching the papilla

Using a side-viewing endoscope

Pylorus

Upper duodenum

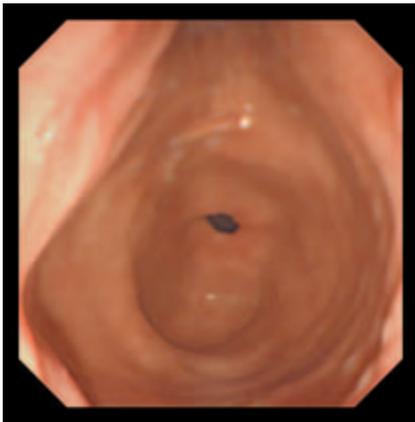
Finding the papilla

Using a side-viewing endoscope

The technique of using a side-viewing endoscope is quite different from that employed with a standard end-viewing instrument. Insertion as far as the stomach is 'blind'. If unexpected resistance is felt during the introduction of the instrument, it is best to withdraw this. A search for a possible cause can be made with a standard forward-viewing endoscope, and if no significant explanation is found, a further attempt with a side-viewing instrument can be made. As the field of view is at right angles to the long axis of the instrument, in order to see the area ahead the tip must be angled down (back) by as much as 90°. After seeing the way forward, the tip is straightened and the instrument is gently advanced. Frequent repetition of this manoeuvre allows almost continuous smooth insertion from the stomach to the duodenum. This method is used for passing the pylorus.

**Pylorus**

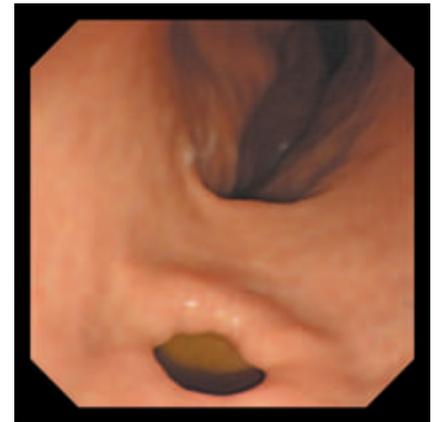
In Fig. 4.3 the pyloric orifice is viewed from the mid-antrum. Figures 4.4 and 4.5 show successive views as the instrument tip is advanced and gradually straightened. The pylorus 'sinks like the setting sun'. With further tip elevation the pylorus disappears from view and the advancing instrument enters the duodenum after a temporary loss of vision.



4.3



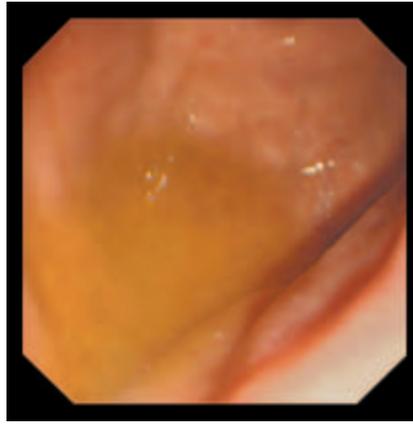
4.4



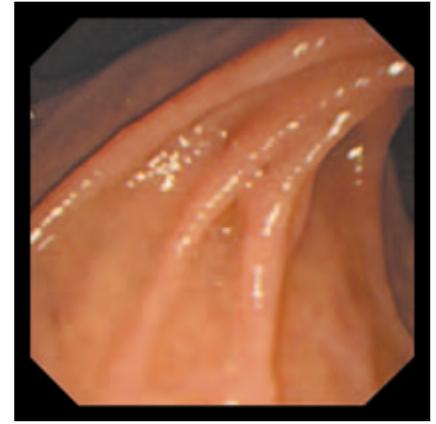
4.5

### Upper duodenum

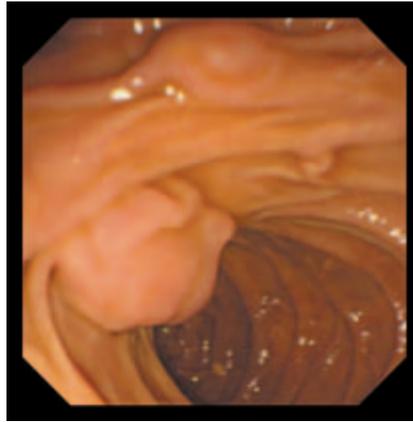
After passing the pylorus the initial view of the duodenal cap is shown in Fig. 4.6. The apex of the bulb with some bile occupies most of the field and the superior duodenal angle is in the lower part. Downward angulation displays the cap more satisfactorily and the way ahead is clear (Fig. 4.7). Advancement is achieved by angling up again, rotating the shaft clockwise and gently inserting. As the second part of the duodenum is entered the valvulae conniventes are seen (Fig. 4.8) and glide by as the lumen is followed. Note the accessory papilla in the upper part of the picture.



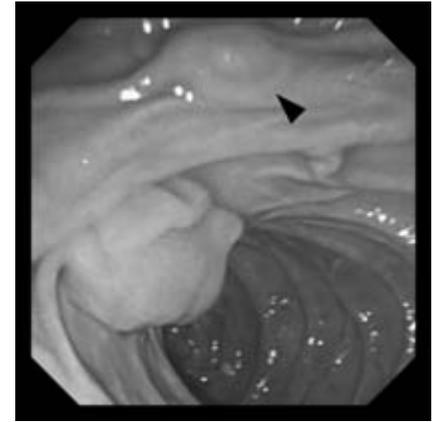
4.6



4.7

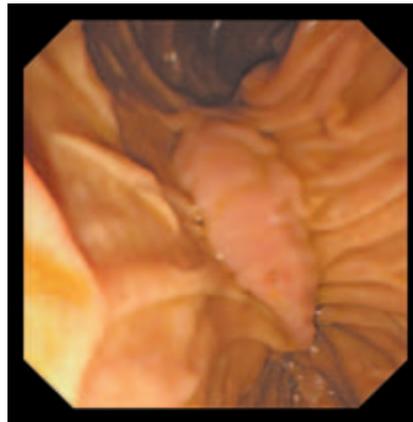


4.8

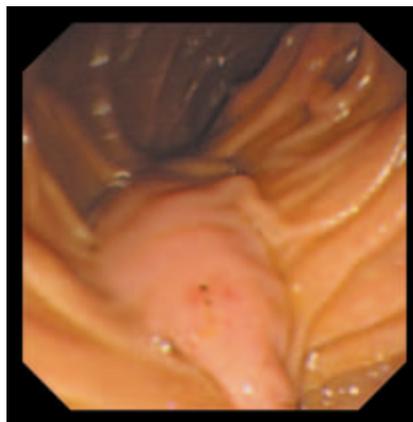
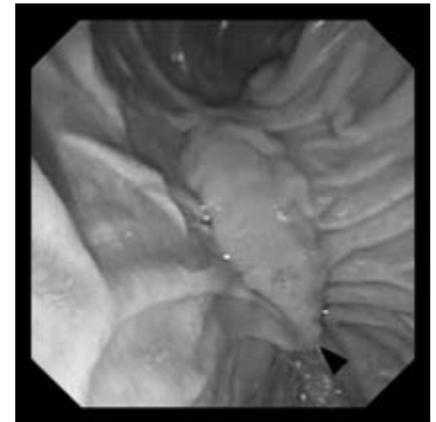


### Finding the papilla

When the descending or second part of the duodenum has been entered the angling knobs of the instrument should be locked, and the instrument gently withdrawn to straighten any redundant loops in the stomach. Paradoxically the instrument tip often advances further at this stage. Careful examination between the mucosal folds on the medial wall usually reveals a longitudinal fold, seen in the lower right corner of Fig. 4.9. Gentle repositioning of the endoscope tip allows a good view of the papilla to be obtained at the proximal end of this longitudinal fold (Fig. 4.10). Emphasis on gentle movement is deliberate, as delicate and small changes in position greatly facilitate the procedure by minimising patient discomfort so increasing co-operation.



4.9



4.10

## Normal and accessory papilla

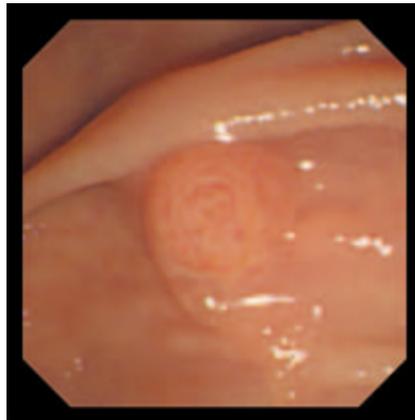
### Normal papilla

### Accessory papilla

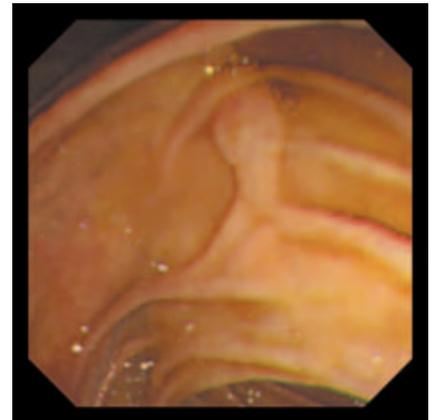
#### Normal papilla

Situated on the medial wall of the descending duodenum at the proximal end of a longitudinal fold, the normal papilla is an often granular nipple-like projection about 5 mm in diameter (Fig. 4.11). The apparent size, shape and prominence may vary greatly with papillary muscular activity. The circular valvulae conniventes are usually interrupted by the papilla, which is often hooded by a mucosal fold (Fig. 4.12) and may be completely hidden from view, when use of a cannula to lift the mucosal folds in turn may reveal the hidden papilla. A flatter papilla (Fig. 4.13) is not uncommon.

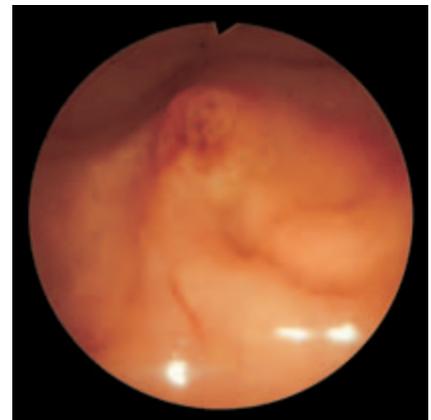
A single orifice of the common ampulla on the apex of the papilla is often visible and discharge of bile may be apparent. Rarely, separate openings of the pancreatic and bile ducts are seen on a single papilla, and



4.11



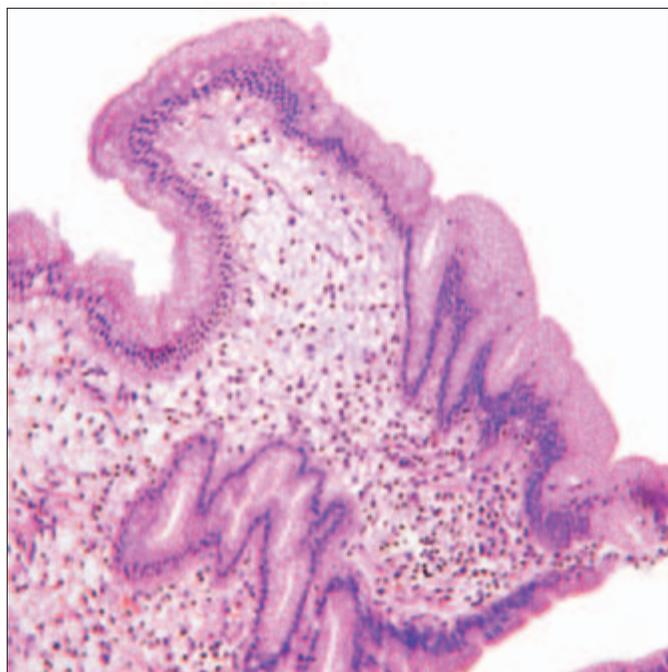
4.12



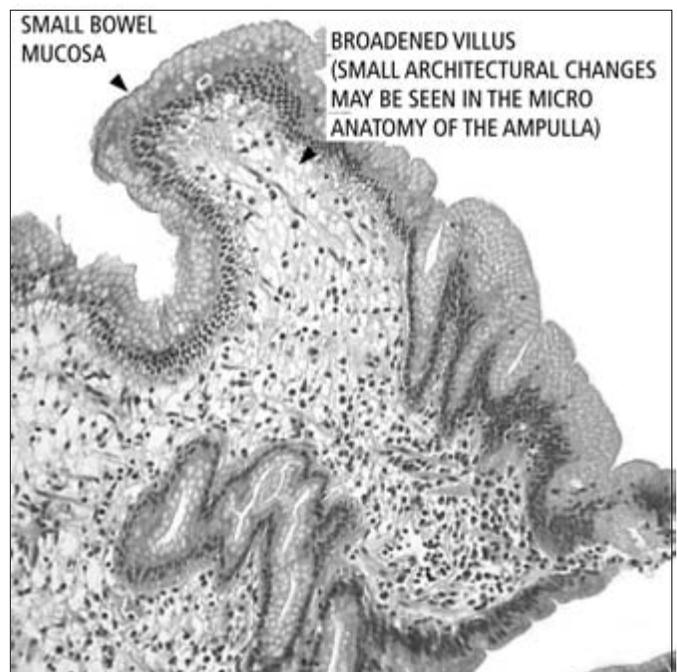
4.13

even less commonly on two separate papillae.

Figure 4.14 shows the normal histological appearances. Biopsies are not usually taken from a normal papilla; these appearances are shown here as a baseline for comparison with biopsies from abnormal tissue.



4.14

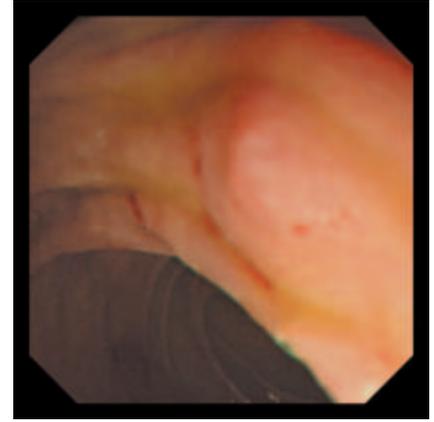


### Accessory papilla

An identifiable accessory papilla (Figs 4.8 and 4.15) is present in a large proportion of subjects. It is situated slightly proximal (1–2 cm) and anterior to the main papilla. It varies in size from just discernible to equal to that of the main papilla (Fig. 4.16), for which it is occasionally mistaken. The accessory papilla marks the embryological site of the duodenal orifice of the duct of Santorini which in many cases may persist into adulthood.



4.15



4.16

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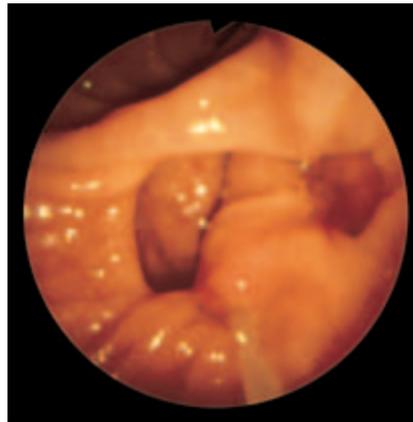
### Duodenal diverticula

#### Papilla associated with duodenal diverticulum

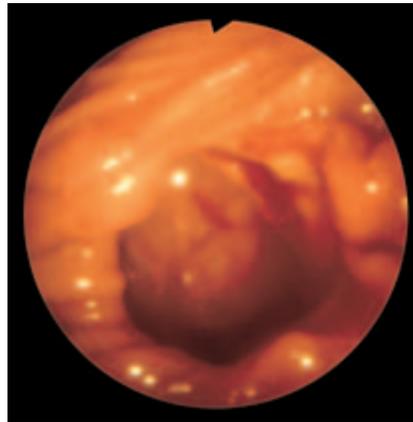
In Fig. 4.17 a ridge which contains the bile duct is seen running across the diverticulum which partially surrounds the papilla (which has been cannulated). In Fig. 4.18 the papilla lies within a diverticulum, a situation likely to present difficulties with cannulation. The longitudinal fold is seen entering the diverticulum from the right. A little blood marks the site of the papillary orifice which was slightly traumatised by earlier cannulation.

Duodenal diverticula are also illustrated in Figs 2.471–2.476.

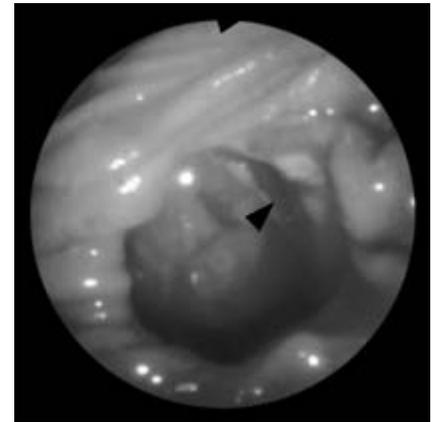
Common duct stones often occur in patients with juxtapaillary diverticula; it is unclear whether the relationship is causal.



4.17



4.18



## Abnormal endoscopic appearances

**Bulging intramural common bile duct**

**Papilla obstructed by a gallstone**

**Patulous papilla**

**Inflammation of papilla**

**Pus exuding from inflamed papilla**

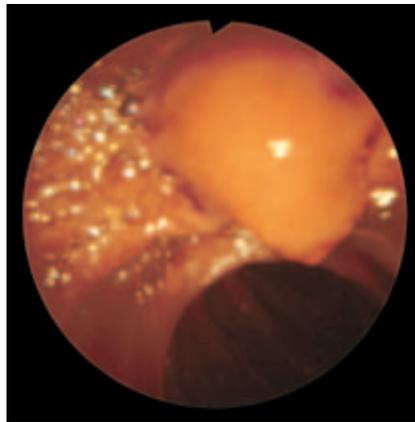
**Suprapapillary choledochoduodenal fistula**

**Carcinoma and adenoma of papilla**

**Carcinoma of head of pancreas**

### **Bulging intramural common bile duct**

A stone in the common bile duct has caused intermittent obstructive jaundice complicated by attacks of cholangitis and an inflamed papilla. The common bile duct is bulging into the medial wall of the duodenum (Fig. 4.19) above the papilla which now points downwards (see also Fig. 2.577). This appearance may simulate a periampullary tumour (see below).



4.19

### **Papilla obstructed by a gallstone**

The papilla is congested and slightly haemorrhagic due to impaction of a gallstone, which is seen protruding through the orifice (Fig. 4.20). This appearance is common when endoscopy is performed during an acute attack of gallstone-associated pancreatitis. Removal of the stone by duodenoscopic sphincterotomy when indicated (see pp. 419–422) is usually followed by resolution of pancreatitis.

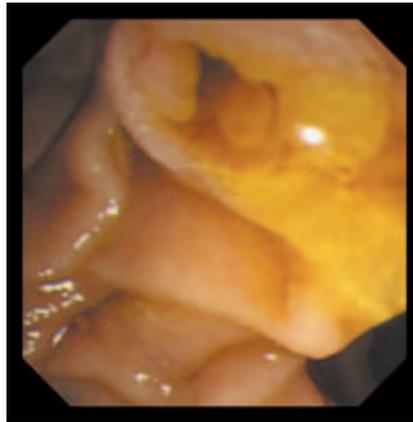


4.20

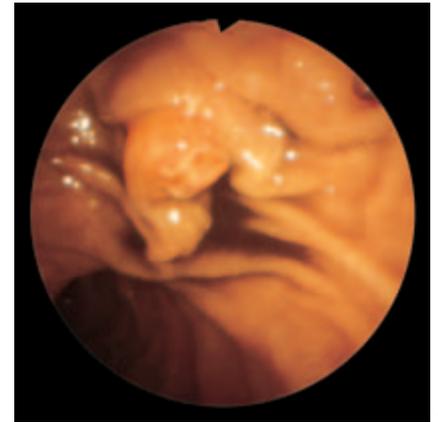
When the stone passes spontaneously the papilla may remain inflamed, friable, patulous or ductal mucosa may prolapse (Figs 4.21, 4.22, 4.24 and 4.25).

### Patulous papilla

A dilated papillary orifice commonly follows spontaneous passage of a calculus (Fig. 4.21). Previous surgical bougienage may result in a similar appearance.



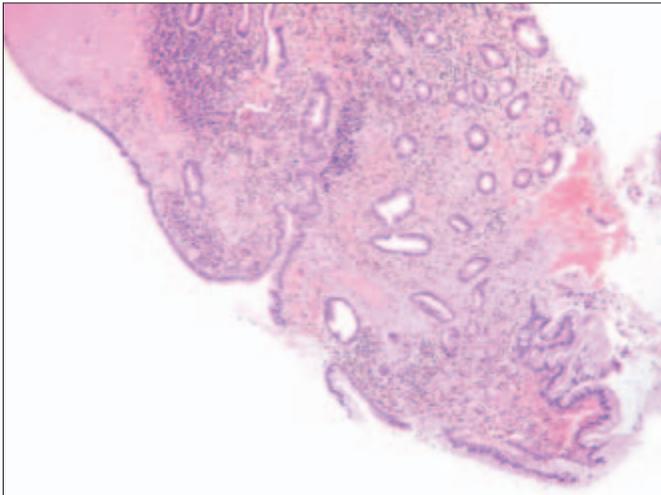
4.21



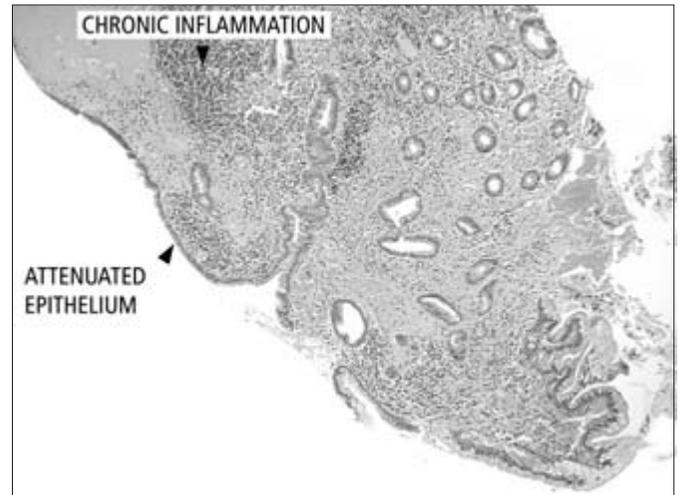
4.22

### Inflammation of papilla

Sometimes an inflamed papilla has a 'fronded' appearance (Fig. 4.22) due to local oedema. Differentiation from a papillary tumour (Fig. 4.29) may require biopsy. Figure 4.23 shows the histopathological appearances of papillary inflammation.



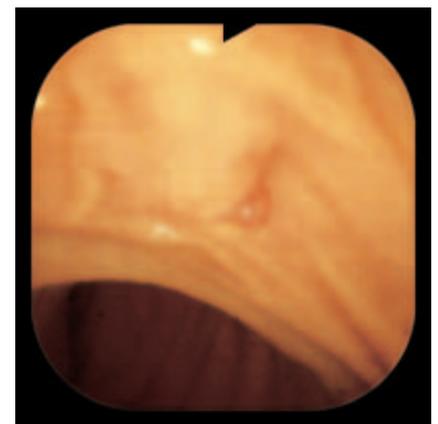
4.23



Ampullary mucosa, especially if inflamed and rendered oedematous for example due to the presence of biliary sludge, may prolapse through the papilla. These serial figures show intermittent prolapse which recurred rhythmically (Figs 4.24 and 4.25).



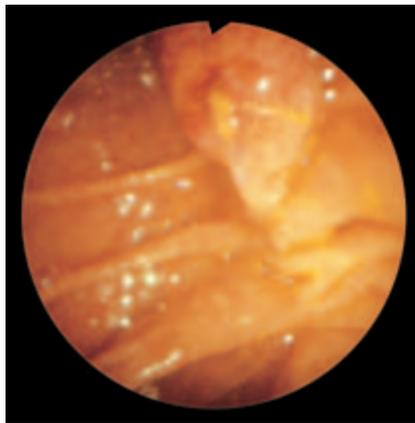
4.24



4.25

### **Pus exuding from inflamed papilla**

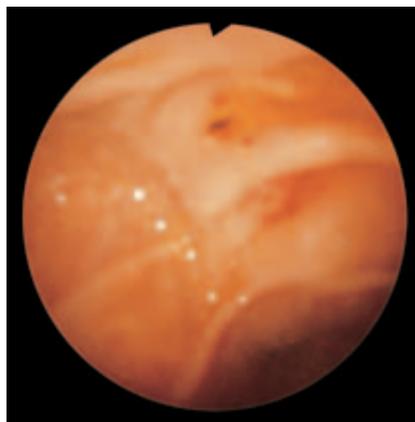
Acute suppurative cholangitis may be recognised by inflammation of the papilla from which pus is exuding (Fig. 4.26). In such patients ERCP will usually show common duct stones. Emergency sphincterotomy (see pp. 419–422) under antibiotic cover is the preferred treatment to allow drainage of the duct and removal of the stones. If this is not possible drainage by means of a stent (pp. 433–439) or a nasobiliary catheter (p. 430) is desirable.



4.26

### **Suprapapillary choledochoduodenal fistula**

Ulceration of a gallstone through the wall of the lower end of the bile duct into the duodenum or previous surgical bougienage of the common bile duct from above may create a choledochoduodenal fistula just above the papilla. In Fig. 4.27 the orifice is seen proximal to the mucosal fold which hoods the papilla. In Fig. 4.28 a cannula has been passed into the papilla and the tip has emerged through the fistula. It is easy to cannulate the bile duct selectively through such a fistula. In this case the fistula had resulted from ulceration by a gallstone.



4.27

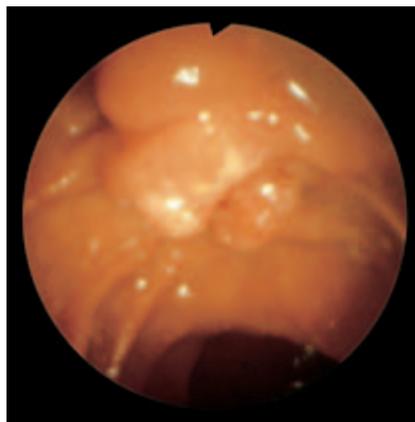


4.28

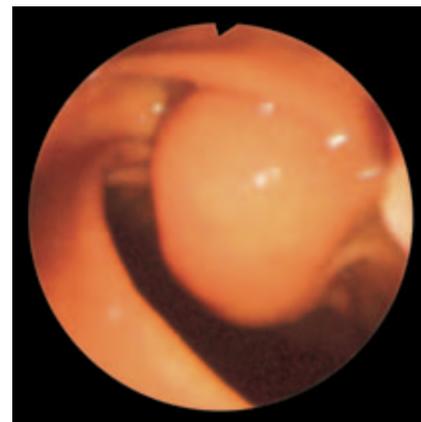
### **Carcinoma and adenoma of papilla**

In Fig. 4.29 the papilla is seen to be enlarged and distorted, with nodules of submucosal tumour adjacent to the orifice. In such circumstances the pathological appearances of biopsy and cytological specimens from within and around the papilla are usually diagnostic.

A tumour within the papilla or arising in adjacent tissues can cause an hemispherical mass on the medial wall of the duodenum (Fig. 4.30). The mass is either the tumour itself or is due to a grossly distended bile duct which has been blocked at the papilla. Mucosal biopsy will then reveal no abnormality. A benign adenoma of the papilla, a rare condition, may present a similar appearance and endoscopic differentiation is difficult. The histological appearances at lower power are shown in Fig. 6.97.



4.29



4.30

**Carcinoma and adenoma of papilla  
(cont.)**

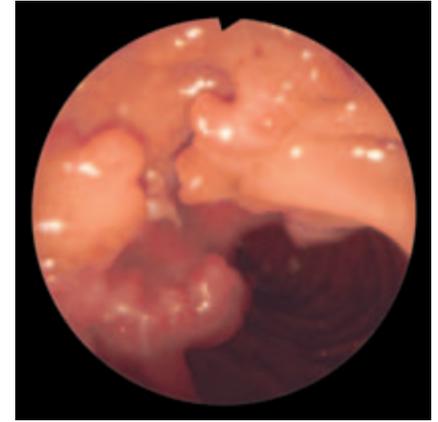
Sometimes diagnostic material can be obtained after the papilla has been ‘opened’ by means of a small sphincterotomy. Aspiration cytology using an endoscopic injection needle may help with diagnosis of submucosal tumours.

Local spread from the papilla to the adjacent duodenal wall may cause loss of the normal fold pattern with irregularity but without ulceration (Fig. 4.31).

Advanced disease causes a larger polypoid lesion which may undergo necrosis resulting in a typical carcinomatous ulcer with overhanging edges (Fig. 4.32). At this stage biopsy and cytological appearances are usually positive. Location and



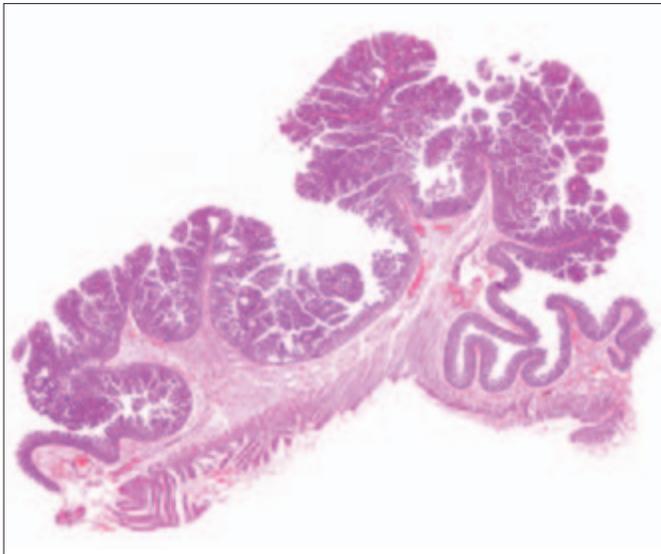
4.31



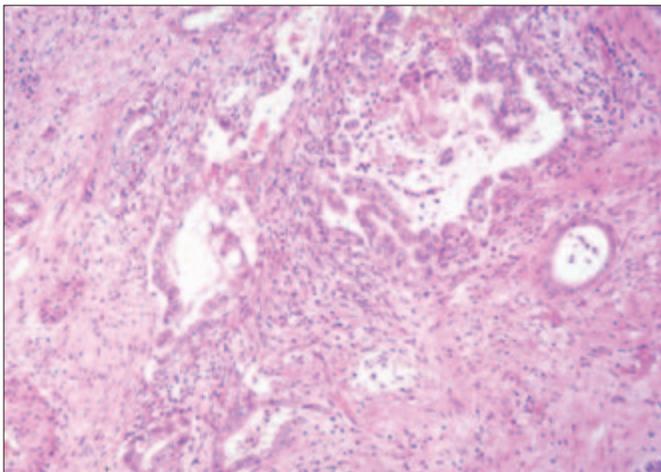
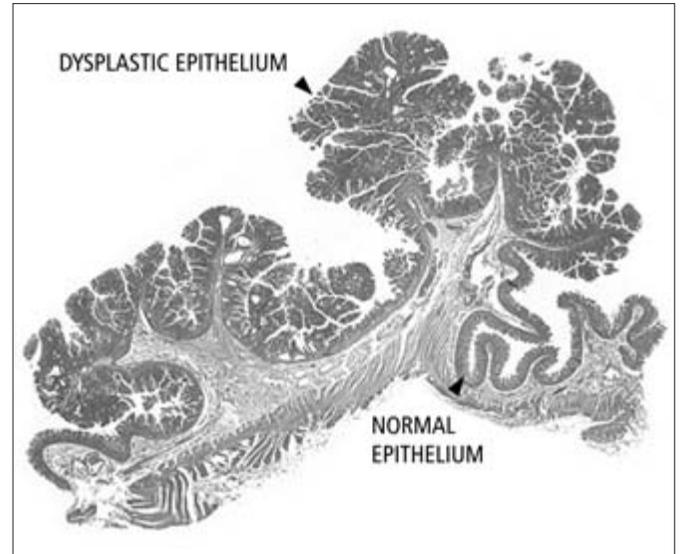
4.32

cannulation of the ductal orifices may be difficult or even impossible.

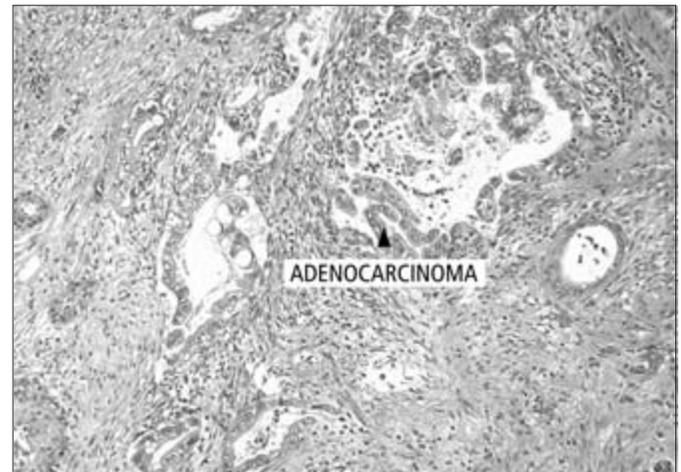
The histopathological appearances of adenoma and carcinoma of the papilla are shown respectively in Fig. 4.33a and Fig. 4.33b.



4.33a

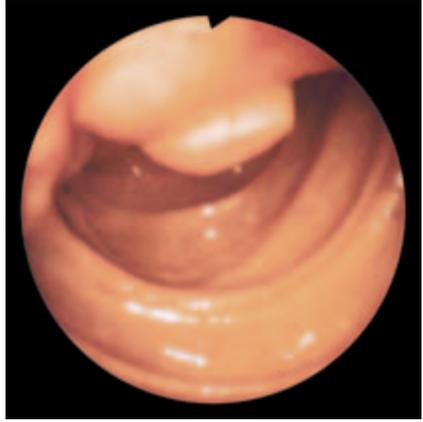


4.33b



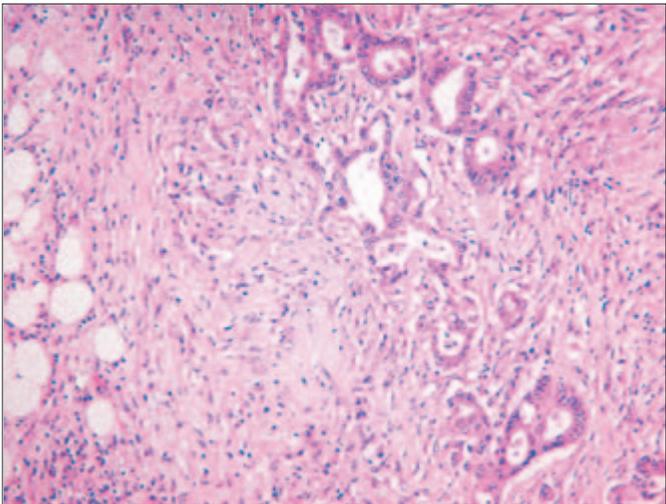
**Carcinoma of head of pancreas**

A neoplastic mass in the head of the pancreas may encroach upon the duodenal lumen (Figs 2.540 and 2.541) causing compression and sometimes resulting in obstruction. In this case (Fig. 4.34) the tumour is just beneath the papilla which is elevated on a mound. The tumour may erode into the duodenal lumen creating a malignant ulcer. Due to reduced space for manoeuvre cannulation of the papilla can be difficult or even impossible.

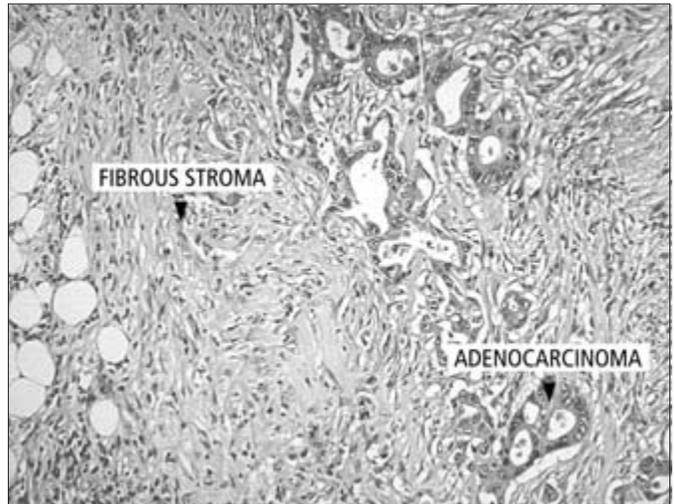


4.34

The histological appearances shown in Fig. 4.35 are of pancreatic adenocarcinoma which was diagnosed from a duodenal biopsy.



4.35



## Technique of cannulation

### Normal approach

#### Approach via the afferent loop

#### Cannulation in the presence of a duodenal diverticulum

#### Biopsy and cytology

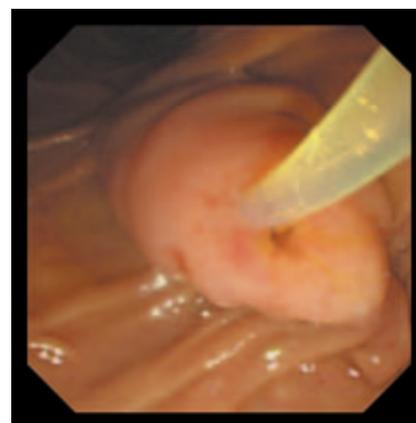
### Normal approach

The endoscope has been placed so that the papillary orifice lies in the most favourable position for cannulation, which has been successfully achieved (Fig. 4.36). The depth of penetration is judged by the single visible dark ring at the end of the cannula, the others (see also Fig. 4.2) being hidden. The papilla in this case is slightly enlarged and inflamed due to recent cholangitis associated with common duct calculi. At this stage it is not possible to be sure which duct has been entered; contrast injection and fluoroscopy are required. In general, cannulation at right angles to the long axis of the duodenum favours the pancreatic duct whilst upwards angulation of the catheter, at or immediately after cannulation (and therefore a more cephalad orientation), favours the bile duct.

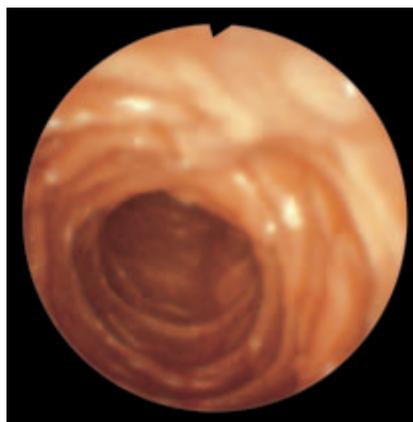
In Fig. 4.37 the cannula has passed several centimetres in the line of the bile duct, which has been entered. It is often possible to pass the catheter into an hepatic duct within the liver. Deep penetration of the pancreatic duct occurs less often: usually the cannula impacts within 3 or 4 cm, where commonly there is a sharp angle in the duct, at the junction of pancreatic head and body (Fig. 4.45). Aspiration through the cannula may reveal clear or bile-stained fluid, depending upon placement, but this is not a reliable method of duct identification.



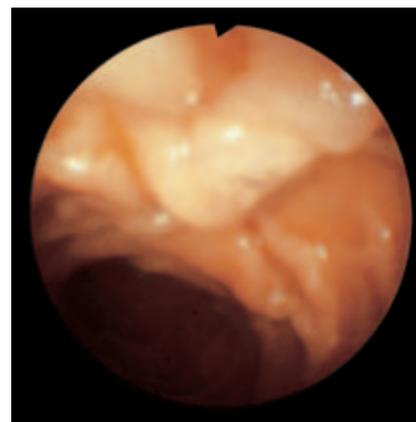
4.36



4.37



4.38



4.39

### Approach via the afferent loop

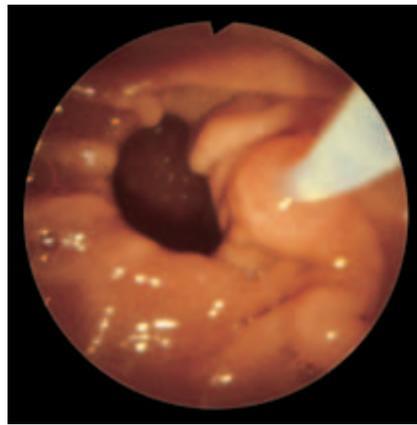
Following Polya (Billroth II) partial gastrectomy the papilla must be approached from below via the afferent loop. This presents many difficulties which are not always overcome. An end-viewing (as here) or

fore-oblique viewing endoscope makes entry to the afferent loop simpler and sometimes allows easier cannulation of the papilla from below.

Figure 4.38 illustrates the longitudinal fold below the papilla, which in turn is shown in Fig. 4.39.

### Cannulation in the presence of a duodenal diverticulum

Juxtapapillary duodenal diverticula are common. In this example (Fig. 4.40) the diverticulum poses no technical problems but when the papilla is situated on the edge of a diverticulum or within it cannulation may present a challenge. The papilla is likely to be intradiverticular when bile is seen discharging from the diverticulum or when the telltale longitudinal fold runs over the edge into the diverticulum (Fig. 4.18). It may be possible to insert the tip of the endoscope into the diverticulum to obtain a satisfactory cannulating position. It must be remembered that the wall of a diverticulum is thin, and that instrumental perforation can occur. Sometimes cannulation may be facilitated by lifting the papilla from within the diverticulum using the tip of the cannula or by means of gentle suction.



4.40

### Biopsy and cytology

Samples for pathological examination are easily obtained from the region of the papilla using standard endoscopic biopsy forceps or a sheathed cytology brush. Obtaining material from within the pancreas or biliary system is much more difficult as direct vision is not routinely possible and guidance for sampling depends upon fluoroscopy. It is preferable to use special ERCP forceps (Fig. 4.41) which are narrower and can be 'shaped' to permit insertion into the ducts. A novel technique allows forceps to be guided into the ducts through a wide bore cannula placed after opening the papillary orifice by means of a sphincterotomy. This is a complex procedure which is not used widely at present. Positive biopsy results are helpful but negative findings must be interpreted with caution. A wire guided cytology brush (Fig. 4.42) provides more satisfactory samples as it is usually possible to pass the guide-wire and in turn the brush through strictures in the biliary system (Fig. 4.207) and sometimes in the pancreas to obtain rich cytological smears.



4.41



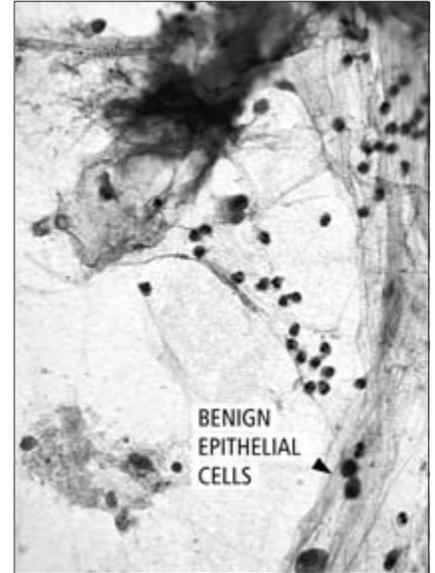
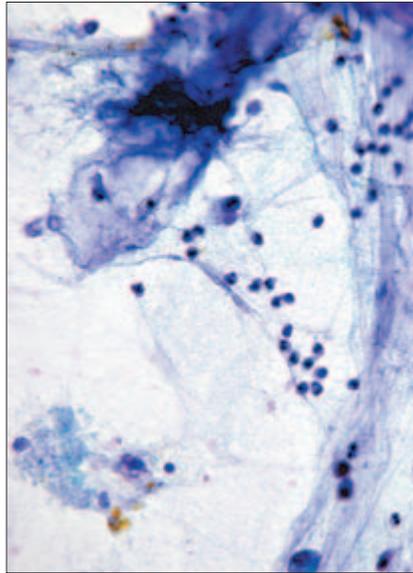
4.42

**Biopsy and cytology (cont.)**

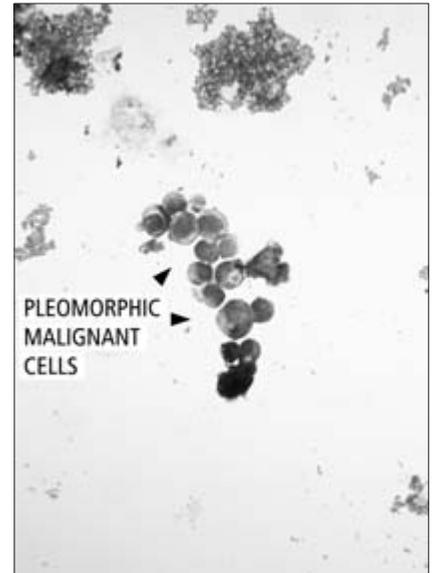
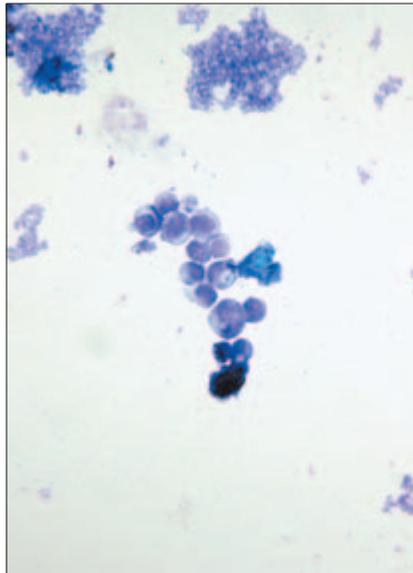
The success of the technique depends greatly upon the care with which material is collected and the interpretative skills of the cytologist. Contamination of the cytological material with radiological contrast medium may alter cell morphology and can cause difficulties in interpretation.

Figures 4.43 and 4.44, respectively, demonstrate the cytological appearances of normal and malignant bile duct epithelial cells.

Pure pancreatic juice aspirated through the cannula after giving intravenous secretin may also be examined cytologically.



4.43



4.44

## Pancreas

Normal radiological appearances 358

Congenital anomalies 362

Abnormal radiological appearances 365

### Normal radiological appearances

Normal pancreatogram

Pseudostricture

Contrast draining via Santorini's (accessory) duct

#### Normal pancreatogram

The volume of contrast material required to demonstrate the pancreatic duct is often less than 5 ml and careful radiological screening during filling should show when an adequate amount has been instilled. Care must be taken lest overfilling should occur causing pain, an acinar blush (Fig. 4.53) and possibly rupture of acini with the risk of post-ERCP pancreatitis. Radiographs are usually taken with the cannula in place, to allow further injection of contrast medium as appropriate; contrast normally drains quickly into the duodenum leaving the duct within 5 min or less. Contrast spilling into the duodenum may obscure radiographic detail and also tends to stimulate duodenal peristalsis which can make the procedure more difficult. Late films taken after removal of the cannula show whether drainage is satisfactory: delay is usually due to a stricture or chronic pancreatitis.

Figures 4.45 and 4.46 illustrate typical normal pancreatograms: the main duct has been filled to the tail and fine side branches are demonstrated. Normally the duct tapers gradually to the tail where a bifurcation is commonly seen. The maximum diameter is less than 5 mm, increasing slightly with age: the appearances shown in Fig. 4.47 are within normal limits for an older person.



4.45

**Normal pancreatogram (cont.)**

The course of the duct is usually sigmoid or pistol shaped but little significance is attached to minor variations. An example is shown in Fig. 4.48.



4.46



4.47



4.48

**Normal pancreatogram (cont.)**

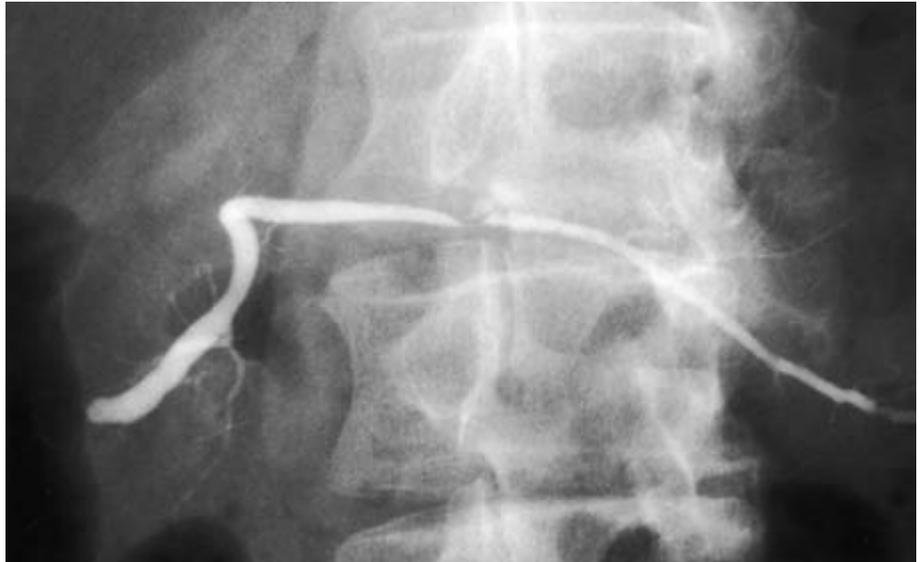
Commonly a juxta-papillary diverticulum fills with contrast material spilling from the papilla during instillation. When small as in Fig. 4.49 no diagnostic difficulties arise, but when diverticula are very large or multiple confusion with cysts in the head of the pancreas may occur.



4.49

### **Pseudostricture**

Underfilling of the pancreatic duct may cause pseudolesions, such as apparent stricture (Fig. 4.50) or blockage. Firm injection pressure caused the pseudostricture shown here to fill out. This appearance, particularly in the mid-portion of the duct, is more common in the elderly. It is probably caused by angulation of the duct as it crosses the aorta. Sometimes there is difficulty filling the duct to the tail with the patient in the prone position. This may often be overcome by instilling contrast medium and then quickly taking supine films after contrast has run 'downhill' into the tail. This would usually be done at the conclusion of an investigation to allow removal of the endoscope before re-positioning the patient.



4.50

### **Contrast draining via Santorini's (accessory) duct**

Patency of the accessory duct (of Santorini), in direct communication with the main pancreatic duct, has allowed contrast medium to leak into the duodenum, causing a small pool adjacent to the endoscope, whilst the tail of the gland is being filled with the cannula in the major papilla (Fig. 4.51). Occasionally leakage via Santorini's duct is so profuse that it is not possible to opacify the tail. Supine films as described above are sometimes successful.



4.51

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## Congenital anomalies

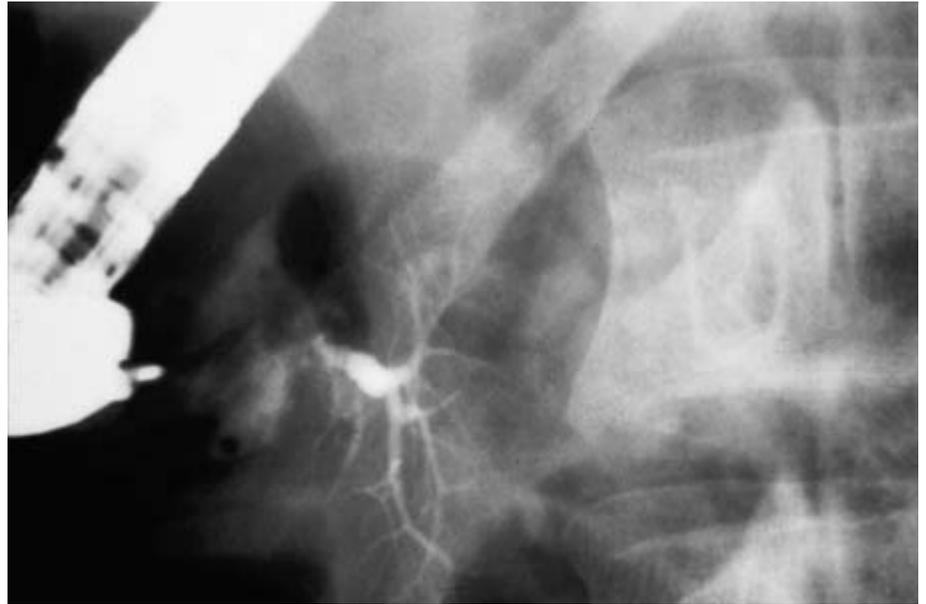
### Pancreas divisum

#### Incomplete fusion of ventral and dorsal portions

#### Annular pancreas

### Pancreas divisum

The embryonic ventral and dorsal pancreatic buds usually fuse in the seventh week of fetal life when the duct of the ventral portion takes over drainage of the dorsal portion thus forming the main pancreatic duct (of Wirsung), draining via the major papilla. The portion of the dorsal duct nearest the duodenum disappears, or remains small and becomes the accessory duct (of Santorini), draining via the accessory papilla. Failure of fusion occurs in about 5% of the population. Cannulation of the ventral pancreas through the major papilla then reveals a small duct draining only the head and uncinate lobe (Fig. 4.52). The duct is not continuous with that of the dorsal pancreas. Owing to the small capacity of the ventral pancreatic duct, an acinar blush due to overfilling is commonly seen (Fig. 4.53).



4.52



4.53

**Pancreas divisum (cont.)**

The possibility of pancreas divisum should always be remembered when filling of the major duct is not seen on fluoroscopy after apparently successful cannulation. Radiographs should be taken and examined carefully lest a small ventral pancreas be overlooked. An example is illustrated in Figs 4.54 and 4.55 where although biliary opacification was the aim of the examination it was only on review of the films that the small ventral pancreas was noted.

To confirm pancreas divisum cannulation of the accessory papilla is required. In Fig. 4.56 a fine-tipped cannula is resting in the accessory papilla and a normal dorsal pancreatic duct has been filled to the tail. The unfused ventral pancreas and biliary system were opacified earlier by cannulation of the main papilla.



4.54



4.55



4.56



### Incomplete fusion of ventral and dorsal portions

Following cannulation of the major papilla, a fine thread of pancreatic duct is seen joining a rudimentary ventral duct and well-developed dorsal duct (Fig. 4.57) draining through the accessory papilla. It is important to distinguish this condition from stricture of the pancreatic duct due to carcinoma (Fig. 4.83) or pancreatitis (Fig. 4.62).



4.57



### Annular pancreas

This uncommon congenital anomaly may cause stenosis of the duodenal lumen (Fig. 2.582). In the following patient duodenal narrowing made visualisation of the papilla difficult. The duct from the annular portion of the pancreas is seen angling acutely and passing behind the duodenoscope (Fig. 4.58). Acinar filling has occurred in the head and annular portions of the gland.



4.58



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## Abnormal radiological appearances

Acute pancreatitis

Chronic pancreatitis

Pancreatitis associated with pancreas divisum

Pseudocyst

Abscess

Leaking duct

Complex pancreatic disease

Pancreatitis causing biliary stricture

Carcinoma of the pancreas

Carcinoma of the papilla of Vater

### Acute pancreatitis

Inflammation and oedema of the duodenal wall associated with acute pancreatitis can make location and identification of the papilla difficult. Unless therapeutic intervention is contemplated in acute gallstone pancreatitis ERCP is best delayed until the acute phase of the disease has subsided. Diagnostic pancreatography is not normally appropriate in acute pancreatitis which is best diagnosed by serum amylase (or lipase) measurement and solid organ imaging techniques. However should ERCP be performed in this situation acinar opacification may occur with low injection pressure (Fig. 4.59). Endoscopic pancreatography performed in the presence of acute pancreatitis can exacerbate this condition.



4.59

## Chronic pancreatitis

Chronic pancreatitis or the effects of an acute attack of pancreatitis may cause structural changes to the pancreatic ductal system which are recognisable by ERCP. The features of minimal change pancreatitis are shown in Fig. 4.60 whilst more severe disease is illustrated in Figs 4.61–4.80.

### *Minimal change pancreatitis*

In Fig. 4.60 the duct has been filled throughout its length and shows minor variations in calibre, especially in the tail. Some of the branch ducts are irregular and slightly dilated with small collections of contrast medium appearing in the parenchyma. These appearance are described as minimal change pancreatitis. The cause in this patient was not known.

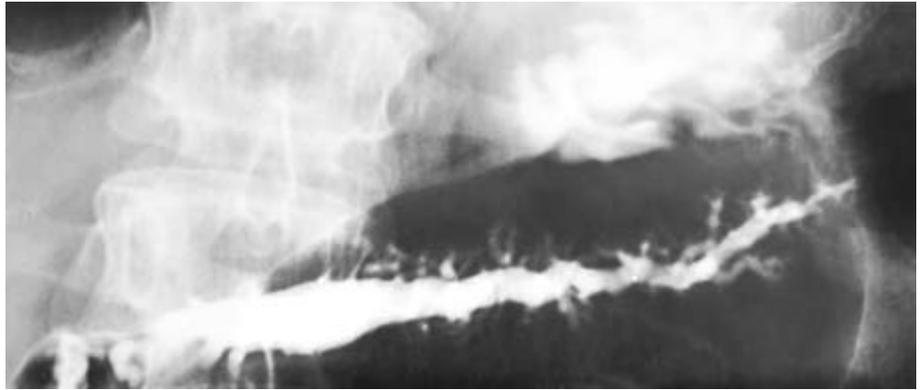


**Chronic pancreatitis (cont.)**

*Severe chronic pancreatitis*

More severe abnormalities are seen in a patient with chronic alcoholism (Fig. 4.61). The whole pancreatic duct is dilated with a maximum diameter of approximately 1 cm and is of markedly irregular calibre. Branch ducts are dilated and disorganised with small cystic areas in the parenchyma. Drainage of contrast medium is delayed until long after withdrawal of the endoscope and repositioning the patient for radiography.

In Fig. 4.62 the pancreatic duct in the head is of normal calibre but is blocked abruptly at the junction with the body of the gland. Acinar filling is shown in the parenchyma of the head. There are also flecks of calcification beyond the block. These appearances are strongly suggestive of chronic pancreatitis but associated malignancy cannot be excluded on ERCP appearances alone.



4.61



4.62

**Chronic pancreatitis (cont.)**

*Severe chronic pancreatitis (cont.)*

The ductal system shown in Fig. 4.63 has a bifurcation in the body but is otherwise unremarkable, yet there is extensive fine calcification in the parenchyma. A preliminary plain film of the abdomen (scout film) is a wise precaution as fine calcification either in the parenchyma or within the ducts may be obscured after contrast has been injected.



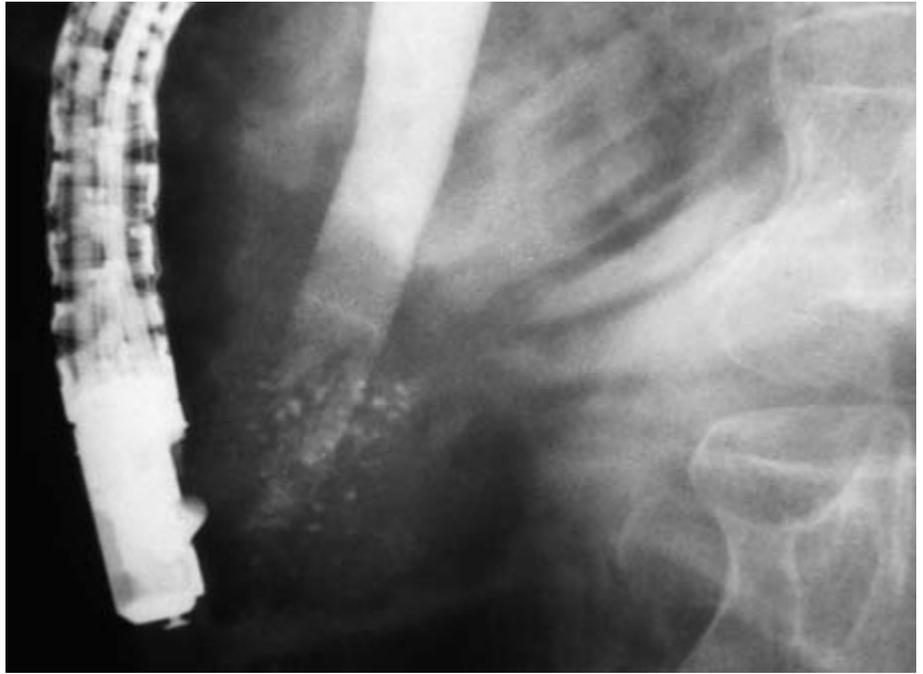
4.63

**Chronic pancreatitis (cont.)**

*Severe chronic pancreatitis (cont.)*

Moreover other radio-opaque objects may be misinterpreted as pancreatic calcification. Figure 4.64 shows barium residues in a foam mattress used on the X-ray table. During a previous examination barium sulphate had seeped into the mattress through a perforation in the cover. Such small opacities apparently overlying the pancreas might possibly be misinterpreted as pancreatic calcification.

In Fig. 4.65 multiple calculi appear as filling defects within the dilated ductal system of this patient who was a chronic alcoholic. The plain film showed some calcification but more defects were revealed by ERCP; it should be remembered that a proportion of pancreatic calculi are radiolucent.



**Chronic pancreatitis (cont.)**

*Severe chronic pancreatitis (cont.)*

Delayed drainage of the main pancreatic duct (Fig. 4.66) is due in this instance to a stricture in the head of the gland confirmed at laparotomy; similar abnormalities may be seen with carcinoma of the head of the pancreas (Fig. 4.85). Residual contrast material outlines the bile duct, cystic duct and gallbladder.

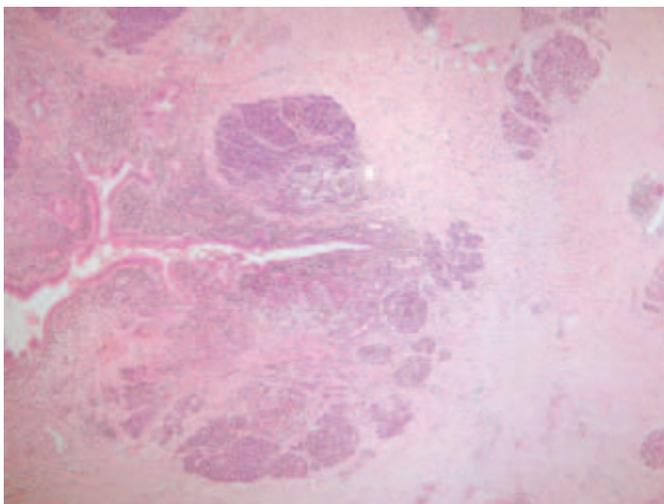
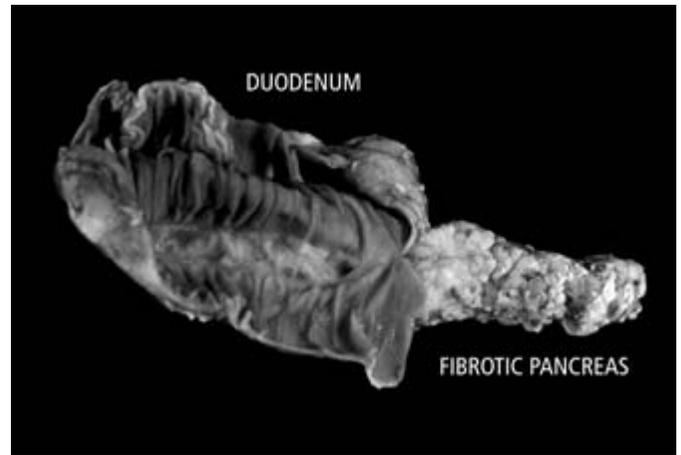
Macroscopic and microscopic appearances of chronic pancreatitis are illustrated respectively in Fig. 4.67a and 4.67b, a severe example in which there is considerable replacement of pancreatic tissue by fibrous tissue. Existing acini are abnormal with zymogen granule loss and vacuolation. Perineural chronic inflammation (possibly a contributory cause of the pain in chronic pancreatitis), islet hyperplasia and hyperplasia of ductal epithelium are seen.



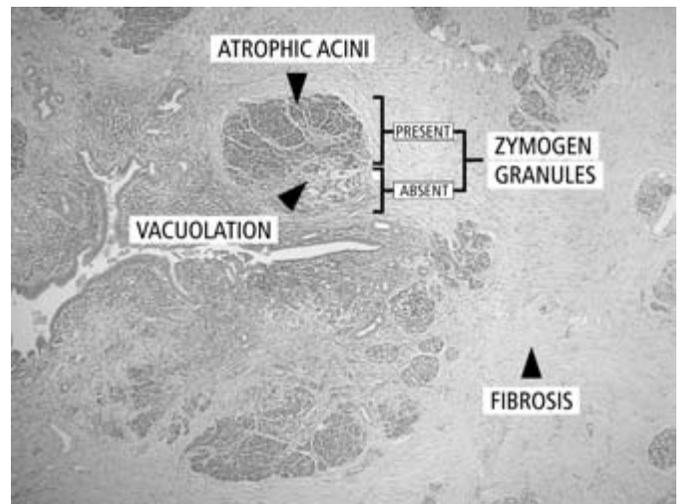
4.66



4.67a



4.67b



**Pancreatitis associated with pancreas divisum**

The congenital anomaly of pancreas divisum (pp. 362–363) is sometimes associated with the development of pancreatitis especially in the dorsal portion. The explanation is unknown; it is suggested that back pressure may result from inadequate drainage through the small accessory papilla. Figure 4.68 shows early acinar opacification after cannulation of the accessory papilla in a patient with a recent attack of acute pancreatitis. When the pancreas is inflamed acinar filling occurs at lower injection pressure and does not necessarily imply that too large a volume of contrast material has been used.

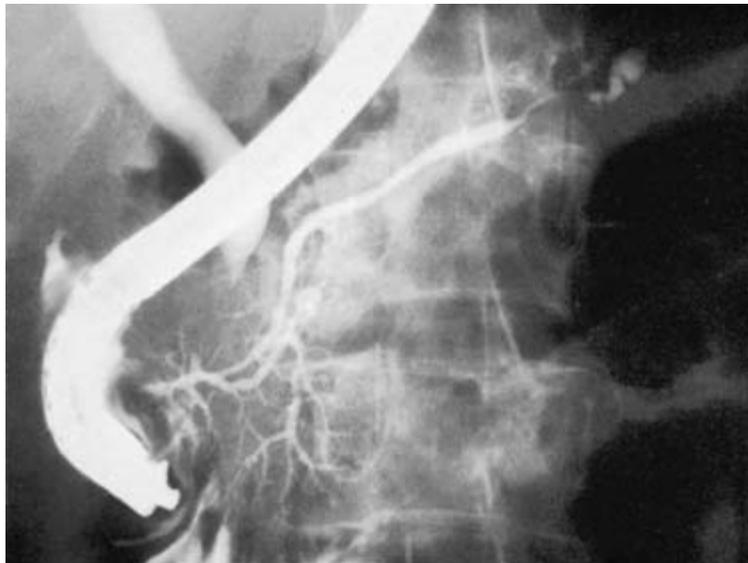


4.68

**Pseudocyst**

When it is necessary to perform ERCP in the presence of a suspected pseudocyst (for example, to assist in surgical planning), or on discovering such a cyst incidentally, ERCP carries a serious risk of introducing infection. This can lead to the development of a pancreatic abscess, a potentially lethal complication. Use of broad spectrum antibiotic cover is recommended. Surgical or percutaneous drainage may be necessary if an abscess develops following ERCP.

In Fig. 4.69 a stricture is shown at the junction of the body and tail. A small quantity of contrast material has passed beyond the stricture into the dilated tail which contained a pseudocyst. A second cyst is shown in the body.



4.69

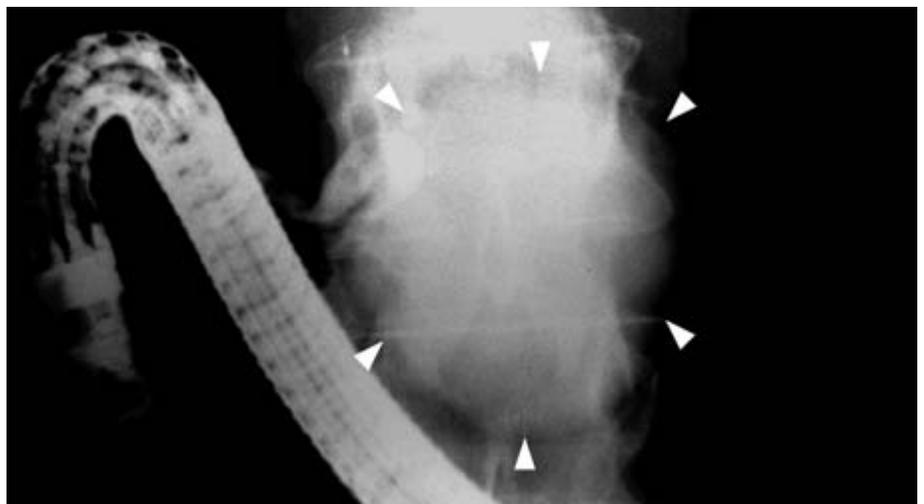
**Pseudocyst (cont.)**

In Fig. 4.70 multiple cysts communicating with the main duct are demonstrated in the markedly enlarged pancreatic head; the tail of the gland appears normal.

The large pseudocyst shown in Fig. 4.71 was filled inadvertently. Antibiotics were given and percutaneous drainage undertaken. Due to continuing pain and sepsis surgical intervention became necessary.



4.70



4.71

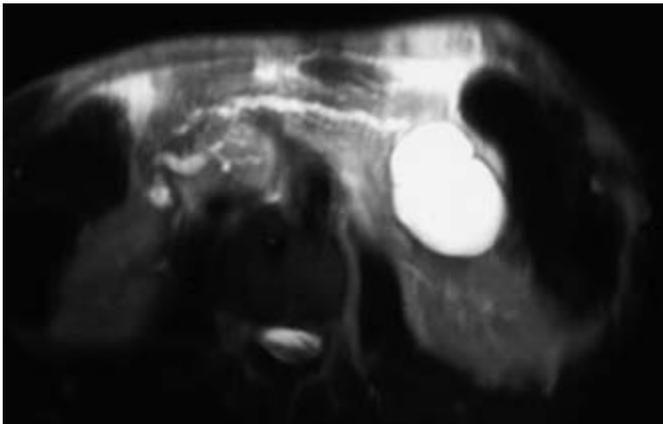
**Pseudocyst (cont.)**

Non-communicating cysts may exert a mass effect, compressing or distorting the main pancreatic duct (Fig. 4.72). In more advanced pancreatitis ERCP may show blockage of the pancreatic duct and alternative imaging techniques are required to determine what lies beyond the occlusion. CT or MRCP, as here, may demonstrate a cyst which is shown in both transverse (Fig. 4.73) and coronal (Fig. 4.74) sections.

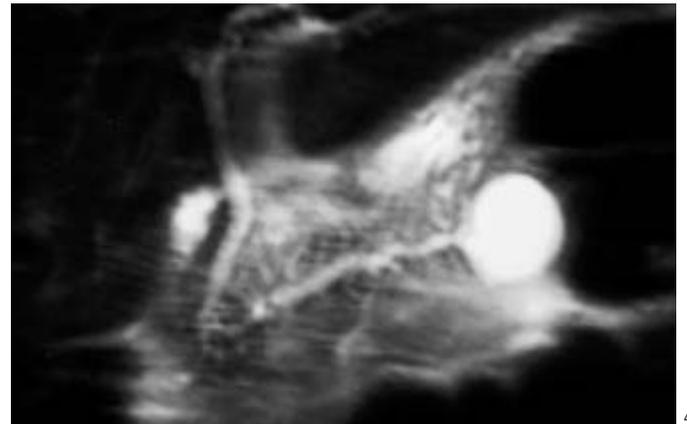
Figure 4.75 shows the macroscopic appearances of a pancreatic pseudocyst.



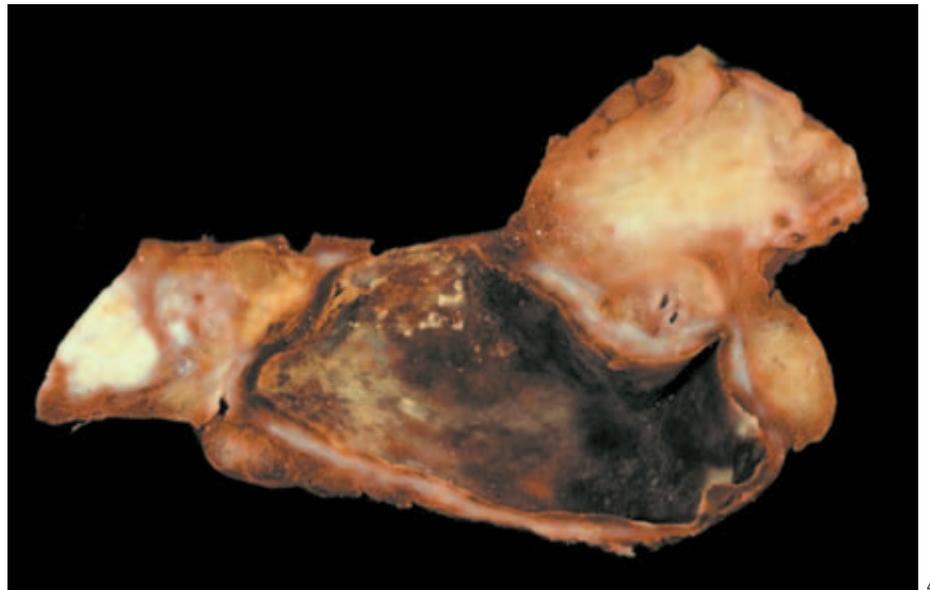
4.72



4.73



4.74



4.75

**Abscess**

An alcoholic presented with abdominal pain, fever and a left pleural effusion with high amylase content. ERCP (Fig. 4.76) showed a stricture in the neck of the gland, with ductal dilatation in the body and tail. Adjacent to the stricture and lying outside the duct there was a collection of contrast which remained after the ductal system had drained (Fig. 4.77). Urgent laparotomy showed a pancreatic abscess. Partial pancreatectomy was performed and the patient recovered well.



4.76



4.77

**Abscess (cont.)**

A stricture associated with a chronic well defined abscess cavity, in another patient, is shown in Fig. 4.78.



4.78

The radiographic distinctions between cyst, pseudocyst and abscess are not clearly defined. Additional clinical, anatomical and investigative information is required to resolve the differential diagnosis.

**Leaking duct**

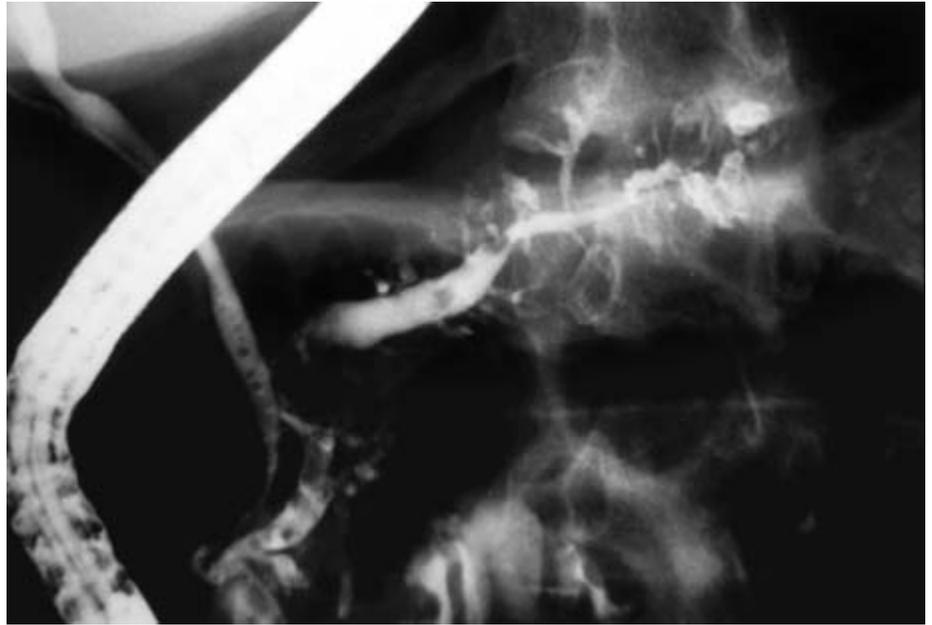
This patient developed ascites with a very high amylase content following acute pancreatitis. Pancreatography outlined the duct to the tail and showed slight irregularity of calibre. The striking abnormality was leakage of contrast from the pancreatic duct just to the right of the spine into the peritoneal cavity (Fig. 4.79). Rapid dilution prevented good definition but spillage can be seen overlying the upper lumbar vertebrae. Such evidence of leakage can also be demonstrated following rupture of the pancreatic duct by blunt abdominal trauma.



4.79

**Complex pancreatic disease**

Complex pancreatic disease may occur with various combinations of the abnormalities previously described. Figure 4.80 shows strictures, calculi, abnormal branch ducts, local cyst formation and leakage into the peritoneal cavity.



4.80

**Pancreatitis causing biliary stricture**

Fibrosis of the head of the pancreas due to chronic pancreatitis may surround and narrow the lower end of the common bile duct (Fig. 4.81) resulting in obstructive jaundice. Differentiation from obstruction by carcinoma of the head of the pancreas is difficult. Endoscopic and fine needle percutaneous aspiration cytology may be helpful although a negative smear does not exclude malignancy. In this case there were striking radiographic changes consistent with chronic pancreatitis and these, together with the smooth tapering biliary stricture, suggested the diagnosis which was confirmed at a later laparotomy. Temporary internal biliary drainage for relief of jaundice may be accomplished by stent placement (pp. 434–438).



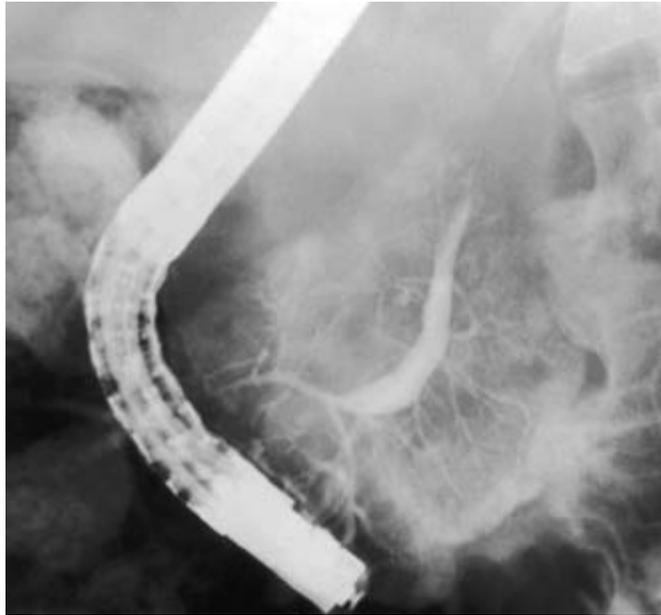
4.81

### **Carcinoma of the pancreas**

#### *Blockage of pancreatic duct*

Figure 4.82 shows abrupt blockage of the main pancreatic duct due to carcinoma of body. High injection pressure is revealed by fine duct and acinar filling in the head of the gland. This is deliberate as incomplete opacification may be due to inadequate force of injection failing to push the contrast into the tail when the patient is lying prone (cf. pseudostricture Fig. 4.50). Similar appearances may result from obstruction due to pancreatitis (Fig. 4.62), but often ductal changes in the head of the gland or presence of calcification will help with differentiation. This is in contrast to the appearance in pancreas divisum where the small portion of pancreatic duct opacified is usually narrower and tapers gradually (Figs 4.52 and 4.55).

In Fig. 4.83 a similar appearance has been shown in the head of the pancreas but the obstruction is incomplete so that contrast material has also filled a grossly dilated duct in the body and tail.



4.82

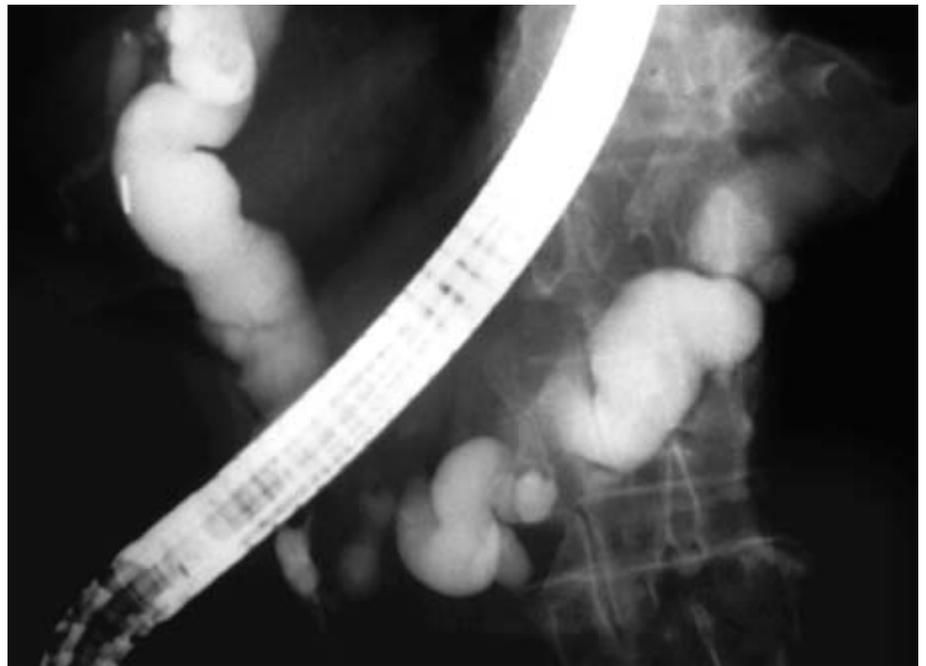


4.83

**Carcinoma of the pancreas (cont.)**

*Blockage of pancreatic duct (cont.)*

Carcinoma in the head of the pancreas may cause dilatation of almost the whole pancreatic duct (Fig. 4.84) and due to local spread also dilatation of the biliary system.



4.84

*Delayed drainage of pancreatic duct*

Failure of drainage of contrast material from the dilated body and tail portions of the duct after withdrawal of the cannula is diagnostic of stricture (Fig. 4.85) whatever the cause. Without pathological confirmation the distinction between cancer and pancreatitis is uncertain (see also Fig. 4.66).



4.85

**Carcinoma of the pancreas (cont.)**

*Biliary obstruction: the 'double duct' sign*

In Figs 4.86 and 4.87 the portions of the pancreatic and bile ducts nearer the duodenum are of normal diameter but a cancer in the head of the pancreas has involved the adjacent bile duct resulting in stricture, the 'double duct sign'. Consequently the upstream portions of the biliary and pancreatic duct systems are dilated due to obstruction caused by the tumour. Figure 4.88 shows the macroscopic appearance of double duct dilatation in a resected specimen.

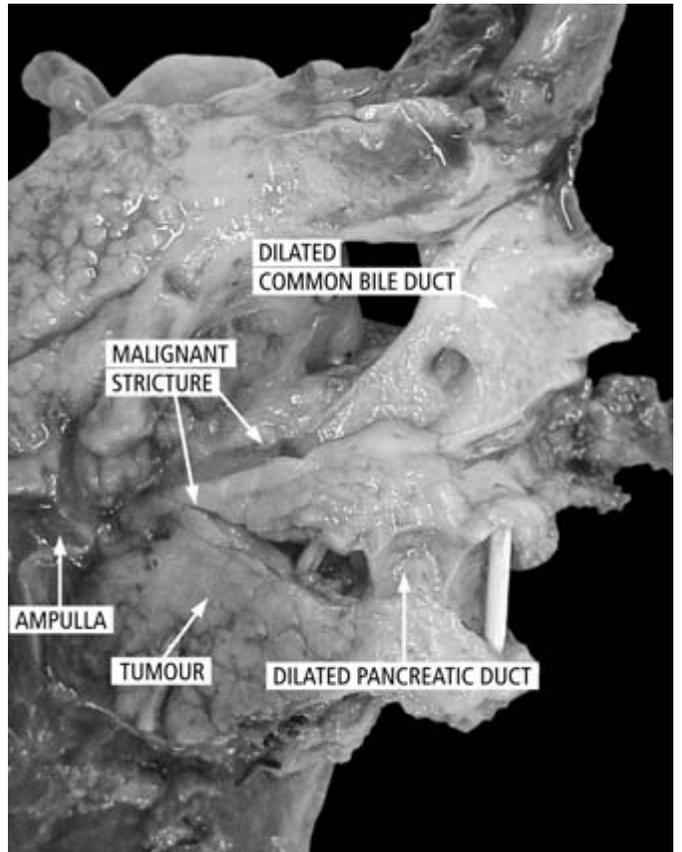




4.87



4.88



**Carcinoma of the pancreas (cont.)**

*Multiple hepatic metastases*

The carcinoma in Fig. 4.89 has caused complete blockage of the pancreatic duct in the body with non-filling of the tail. The liver is grossly enlarged, yet firm injection into the biliary system showed a paucity of intrahepatic ducts although contrast flowed freely into the gallbladder. The ducts that have filled display distorted contours compatible with the presence of multiple hepatic metastases. Previous ultrasound scanning had suggested the diagnosis which was confirmed by liver biopsy.

**Carcinoma of the papilla of Vater**

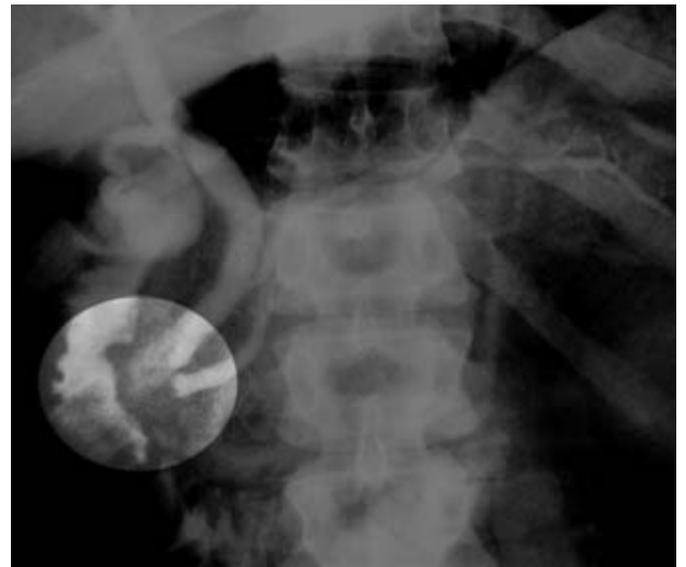
A tumour originating in the region of the papilla (see Figs 4.29–4.32 for endoscopic views) often causes obstruction of both pancreatic and biliary ducts. The ERCP appearances depend upon the location of the tumour, on the degree of obstruction and on its duration. At an early stage (Fig. 4.90) both ducts may be of relatively normal calibre although there is often a ‘filling defect’ apparently separating them from the duodenal lumen. Drainage of contrast material may be delayed.



4.89



4.90



**Carcinoma of the papilla of Vater (cont.)**

Later both ducts become markedly dilated (Fig. 4.91). At this stage there is usually a large tumour involving the medial wall of the duodenum and it may not be possible to cannulate the ducts. The papilla may be completely destroyed and it may be quite impossible to locate its orifice (Fig. 4.32).

When obstruction in the periampullary region is complete and ERCP fails, diagnostic images may readily be obtained by MRCP. Figure 4.92 shows dilatation of the whole biliary system and the pancreatic duct due to a tumour mass in the head of the pancreas.



4.91



4.92

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## Biliary system

**Volume of contrast material 383**

**Normal radiological appearances 383**

**Radiological appearances of congenital anomalies 386**

**Abnormal radiological appearances 388**

**Appearances following surgery 407**

**ERCP and liver transplantation 413**

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### Volume of contrast material

#### Volume of contrast material

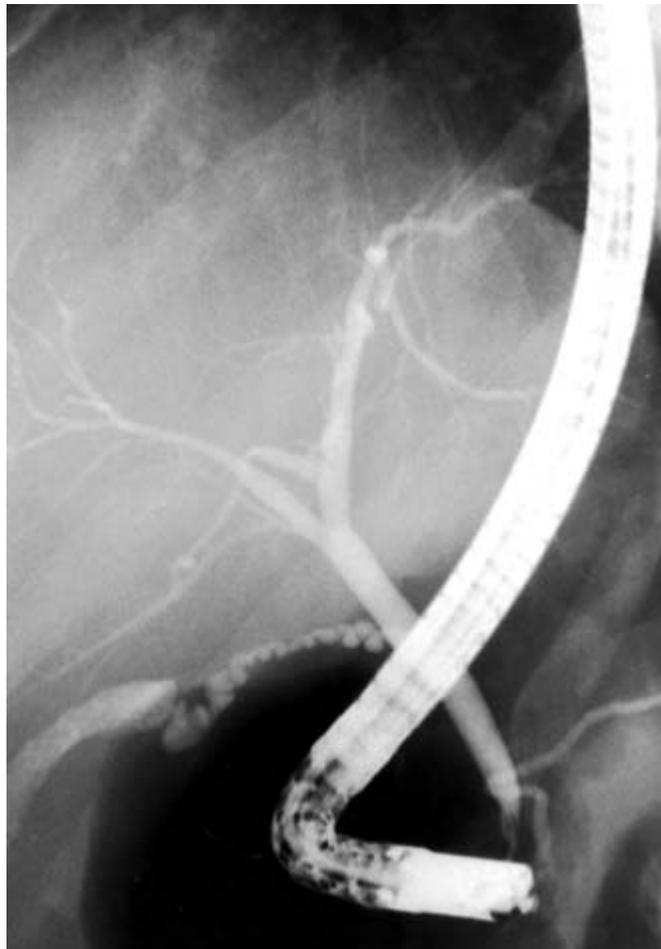
The volume of contrast material required to opacify the biliary system varies greatly depending upon whether dilatation is present and whether the gallbladder is present and/or fills. A dilated system may readily accept 50 ml or more. The quantity needed can only be determined by careful observation during biliary filling. Instillation of contrast material is usually painless but if distension of the biliary system is produced by overfilling, or there is further distension of an already obstructed system, pain may occur. Some patients with motility disorders of the pancreatico-biliary system or papillary stenosis may be particularly sensitive to the volume of contrast injected.

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### Normal radiological appearances

#### Normal biliary system

In Fig. 4.93 the common bile duct is well shown and of normal calibre; the endoscope acts as a scale for comparison. The upper limit in diameter of the normal common bile duct is about 1 cm. The cystic duct has filled and contrast material is entering the gallbladder. At this stage the main intrahepatic ducts and some of the smaller radicles are also clearly seen. Good filling of the whole biliary tree is essential for complete interpretation.



4.93

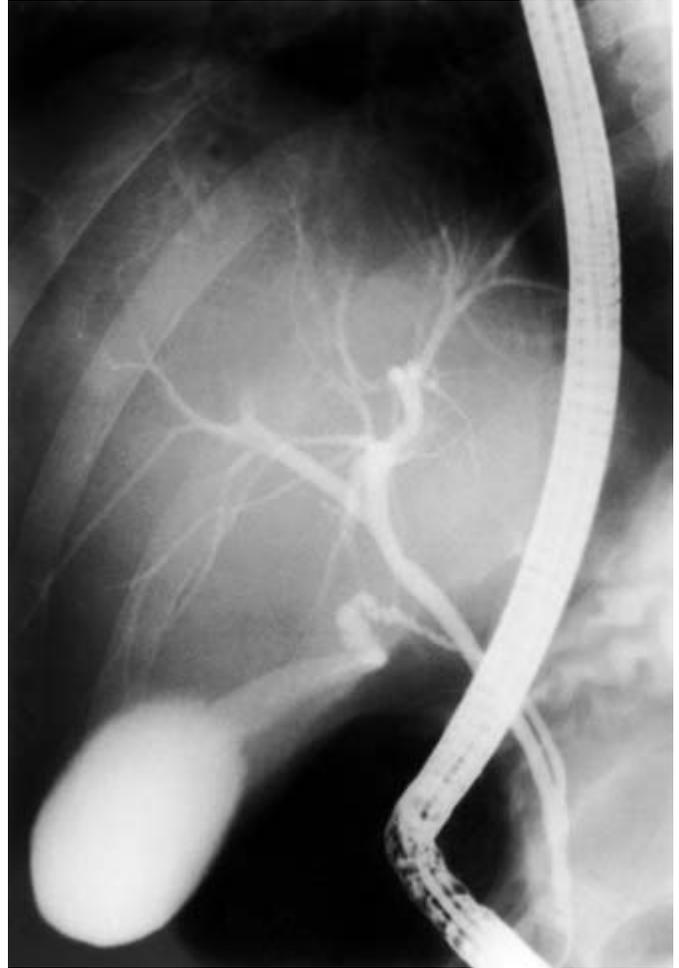
**Normal biliary system (cont.)**

Extensive fine duct opacification is seen in Fig. 4.94 although the gallbladder is not yet well shown.

In Fig. 4.95 almost the whole of the biliary system has filled. Note the low insertion of the cystic duct in this case, a normal variant. Sometimes difficulty may be experienced achieving complete biliary opacification due to gravitational factors: changing the patient's position may help in obtaining optimal filling.



4.94



4.95

**Normal biliary system (cont.)**

After withdrawal of the endoscope further radiographs of the biliary system may be taken under fluoroscopic control (Fig. 4.96) to show the gallbladder and common bile duct, especially when searching for small stones. The pancreatic duct empties of contrast rapidly. Emptying of the biliary system, even in normals, is much slower. Usually therefore, even after removal of the endoscope, there is ample time to obtain good diagnostic radiographs.

Motility of the lower end of the common duct may be observed during fluoroscopy and representative films taken to elucidate. As this region varies greatly in configuration from moment to moment, sometimes being tapered and sometimes blunt (Figs 4.97 and 4.98), judgements should not be made on the appearance of a single film.



4.96



4.97



4.98

The marked variations in the manner in which the biliary system fills and empties emphasise the need for close collaboration between endoscopist and radiologist in order to demonstrate satisfactorily both the structure and behaviour of the bile ducts and gallbladder.

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## Radiological appearances of congenital anomalies

### Choledochal cyst

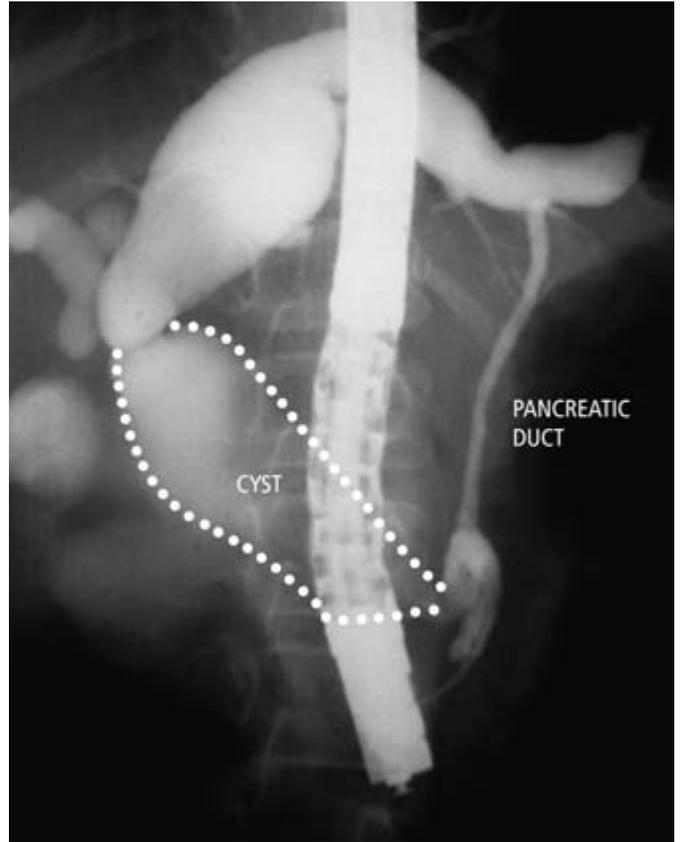
#### Intrahepatic cystic dilatation (Caroli's disease)

### Choledochal cyst

There are several types of choledochal cyst involving various portions of the bile duct, often in association with a long common channel (pancreatico-bile duct). The most frequent variant is diffuse dilatation of the common bile duct and/or the common hepatic duct (Fig. 4.99). This film also shows dilatation of the left hepatic duct.



4.99



### Choledochal cyst (*cont.*)

Other variants are a saccular dilatation of the common bile duct, rather like a diverticulum (Fig. 4.100), or a localised dilatation at the lower end of the bile duct or of the common channel (Fig. 4.101). In this case both the pancreatic and common bile ducts filled from the cyst.



4.100



4.101

### Intrahepatic cystic dilatation (Caroli's disease)

In this condition multiple intrahepatic cysts occur in communication with the biliary radicles (Fig. 4.102); congenital hepatic fibrosis is usually associated. In polycystic liver disease the intrahepatic cysts, by contrast, do not connect with the bile ducts; therefore there are no characteristic cholangiographic appearances in this condition.



4.102

Caroli's disease could be regarded as a variant of choledochal cyst disease, with cysts restricted to the ducts within the liver. There are reports of intra- and extrahepatic cysts occurring in the same patient. All varieties of choledochal cyst and Caroli's disease carry an increased risk of complicating malignant biliary disease.

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## Abnormal radiological appearances

### Stones in the biliary tract

#### Hepatic abscess

#### Sclerosing cholangitis

#### Hepatic cirrhosis

#### Cholangiocarcinoma

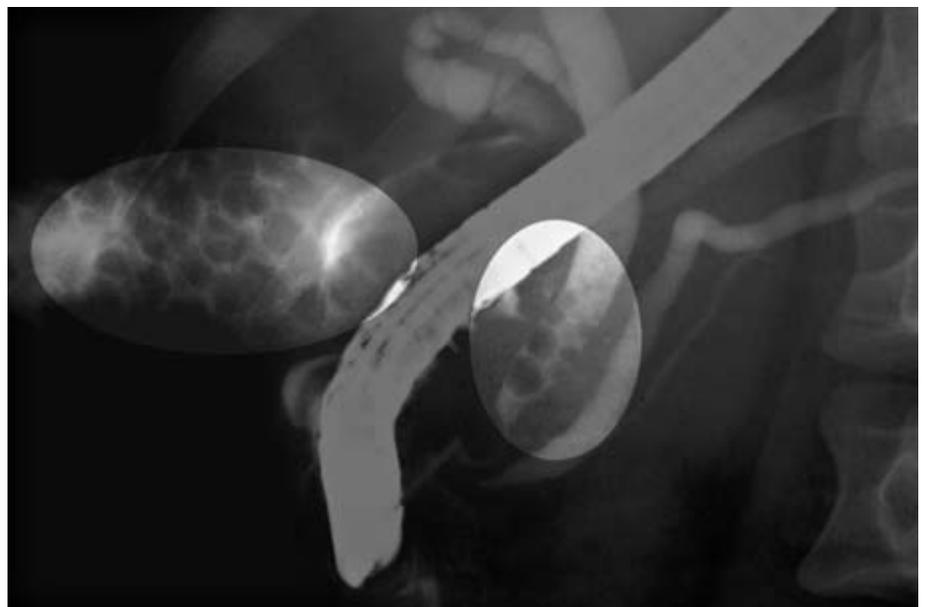
#### Carcinoma of the gallbladder

#### Carcinoma of the cystic duct

#### Differential diagnosis of fixed intraductal 'tumours'

### Stones in the biliary tract

Stones in the biliary system appear as lucencies within the radio opaque contrast medium instilled during ERCP. Their size and shape varies greatly as can be seen in the illustrations following. Gallstones are usually slightly irregular in shape or faceted rather than truly spherical. Calcification may be noted on the preliminary film and can sometimes be seen within stones outlined by contrast media. Distinction from air bubbles is clearly important as air injected through the cannula during ERCP or entering via the papilla or a fistula will also cause a similar appearance. Air bubbles can sometimes be expelled through the papilla during contrast injection and may be aspirated back through the cannula, differentiating them from stones. Single air bubbles are much more often spherical than stones and may be broken by agitating the cannula. Multiple air bubbles can sometimes be recognised as 'lather' with adjacent angular margins. Air floats on bile and may change position on tipping the patient but so may gallstones. Consideration of these various factors usually allows satisfactory distinction between air bubbles and stones, but even so doubt sometimes remains. Figure 4.103 shows multiple stones in the gallbladder but most, if not all of the negative shadows in the bile duct are due to air bubbles.



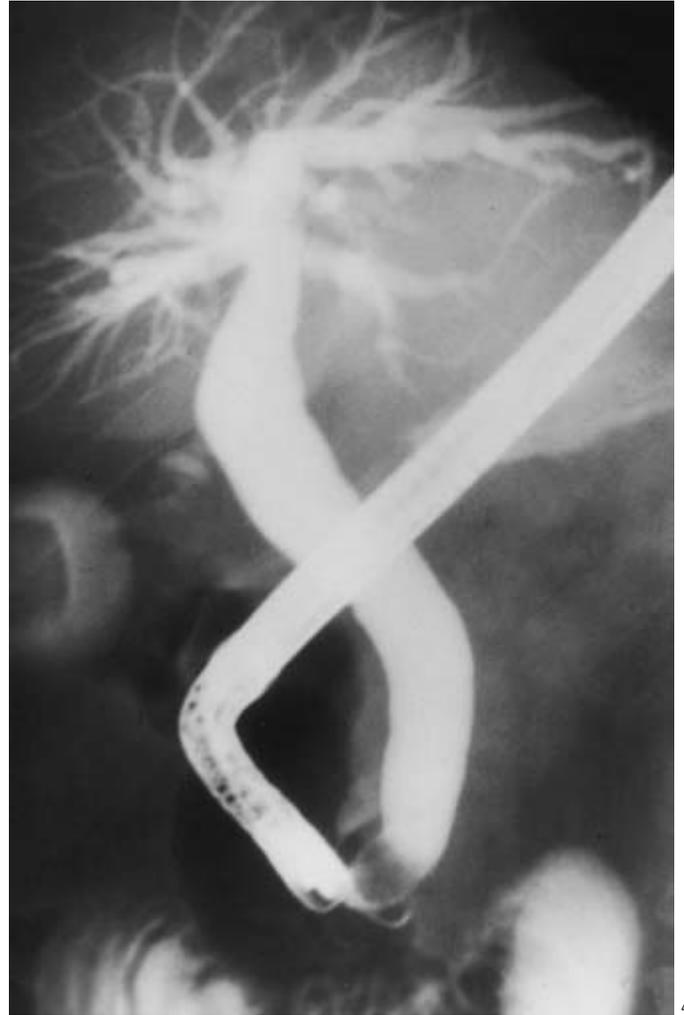
4.103

### **Stones in the biliary tract (*cont.*)**

Figure 4.104 shows the appearance of multiple gallstones in the gallbladder and a solitary stone in a slightly dilated bile duct. The extrahepatic and intrahepatic biliary ducts may be dilated due to stones in the common bile duct (CBD). In Fig. 4.105 there is a large radiolucent stone at the lower end of the CBD and another in the gallbladder. Dense contrast medium has been used which may in a dilated system obscure stones. For this reason it is recommended that a diluted solution of contrast medium is used when it becomes apparent that the bile ducts are dilated.



4.104



4.105

**Stones in the biliary tract (cont.)**

Myriad small stones are seen in the bile ducts in Fig. 4.106; the gallbladder had been removed previously. Some air is visible in the intrahepatic ducts following a sphincterotomy.

Dilatation of the biliary system, including the cystic duct, due to a stone impacted at the lower end of the bile duct is shown in Fig. 4.107. The gallbladder has not filled. Provided adequate injection pressure is achieved, non-filling of the gallbladder is usually due to calculous obstruction of the cystic duct except of course after cholecystectomy. Filling of the gallbladder if present usually occurs relatively late during ERCP, even if there is no distal obstruction.



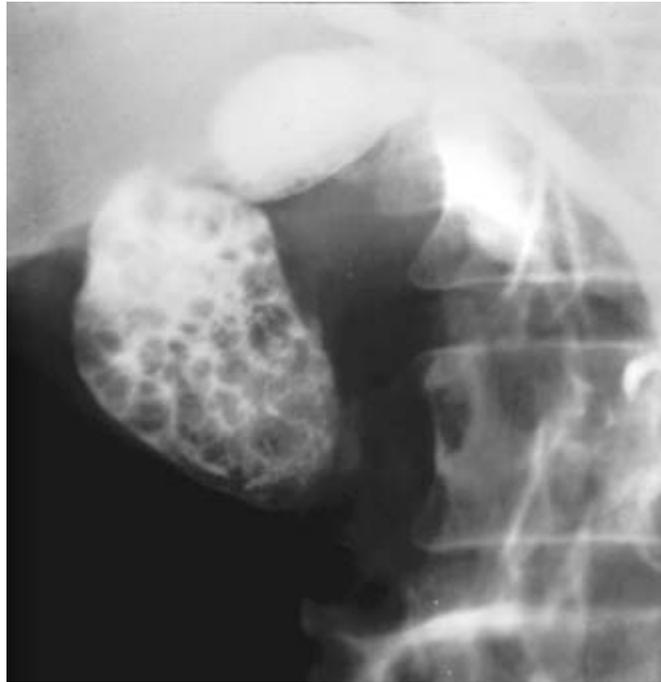
4.106



4.107

**Stones in the biliary tract (cont.)**

Sometimes biliary calculi may be associated with strictures. Figure 4.108 shows multiple stones trapped in the fundus of the gallbladder by fibrotic narrowing of the central part. Local inflammation may cause stones to become fixed to the wall of the bile duct or be held above a stricture (Fig. 4.109). The appearance shown in Fig. 4.110, whilst simulating a stricture, was in fact due to muscular activity at the lower end of the bile duct as the narrowing was seen to be inconstant during fluoroscopy (see also Figs 4.97 and 4.98).



4.108



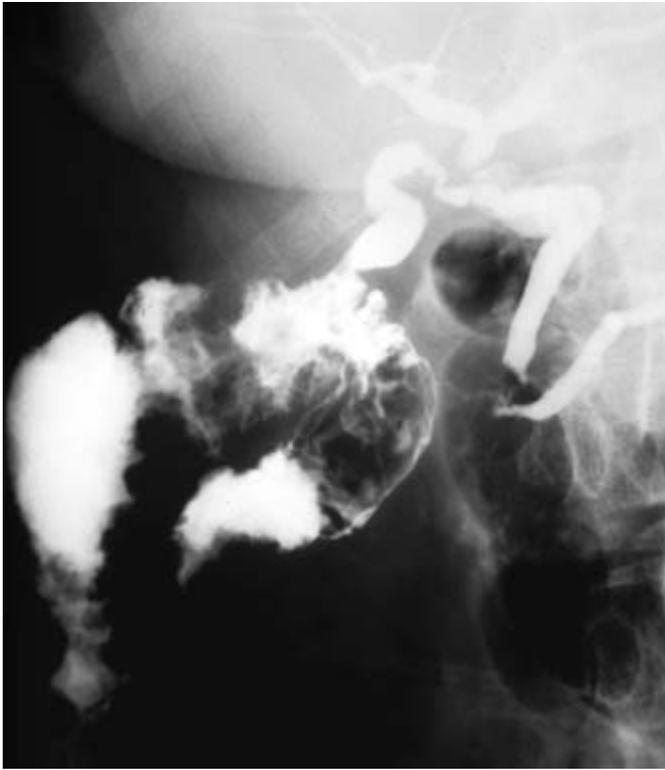
4.109



4.110

**Stones in the biliary tract (*cont.*)**

Local inflammation of the gallbladder due to calculi occasionally causes a spontaneous fistula from the fundus into an adjacent loop of bowel. Figure 4.111 shows a cholecysto-colic fistula. A stone which has passed into the duodenum may be seen directly by endoscopy (Fig. 2.579).



4.111



### **Stones in the biliary tract (*cont.*)**

Dilatation of the CBD usually results from calculi, but even when suspected, stones can be elusive. This may be due to the use of contrast material which is too dense, to very small stones or because the complexity of the biliary system with several overlying ducts prevents its adequate demonstration. The case shown in Fig. 4.112 illustrates the point. A careful search and multiple radiographs failed to show the cause of dilatation, and in particular no calculi were seen. A sphincterotomy was performed because of recent cholangitis, and after drainage of some contrast material a spherical gallstone was easily seen (Fig. 4.113) and subsequently extracted. These figures emphasise the uncertainty of diagnosis when biliary dilatation is found in apparent isolation. Dilatation of the bile duct may be permanent, remaining after spontaneous passage of stones or even after their surgical removal. Some dilatation of the CBD is common after cholecystectomy, even without a history of CBD stones.



4.112

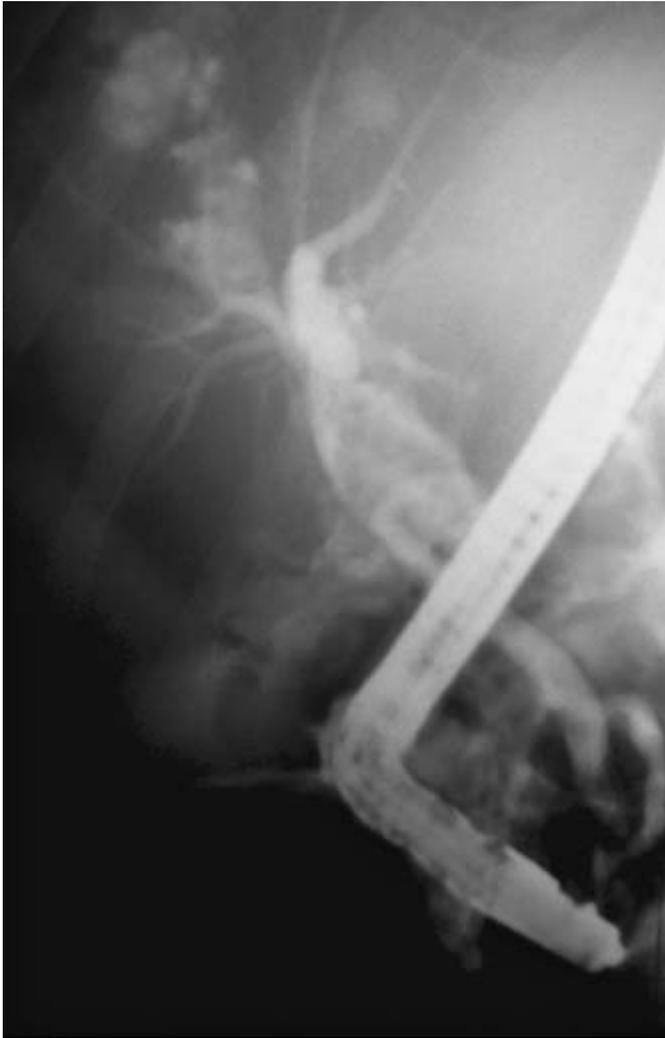


4.113

Multiple gallstones throughout the biliary system, particularly in the intrahepatic ducts, are found commonly in the Far East ('oriental cholelithiasis'). The explanation is unclear but the composition of the stones is different from those encountered in the West with pigment and mixed stones being more frequent. Endoscopic management perhaps employing the combined endoscopic and percutaneous technique (pp. 444–445) may be successful but often surgical intervention is required and, due to the difficulty of clearing the intrahepatic ducts, re-operation may be necessary.

### Hepatic abscess

Figure 4.114 shows large hepatic abscesses. This patient presented with acute pancreatitis and subsequently developed jaundice with septicaemia. ERCP showed the appearance of large abscesses both at the hilum and within the liver, and pus exuded from the papilla. The biliary system was drained by endoscopic sphincterotomy as an emergency.



4.114



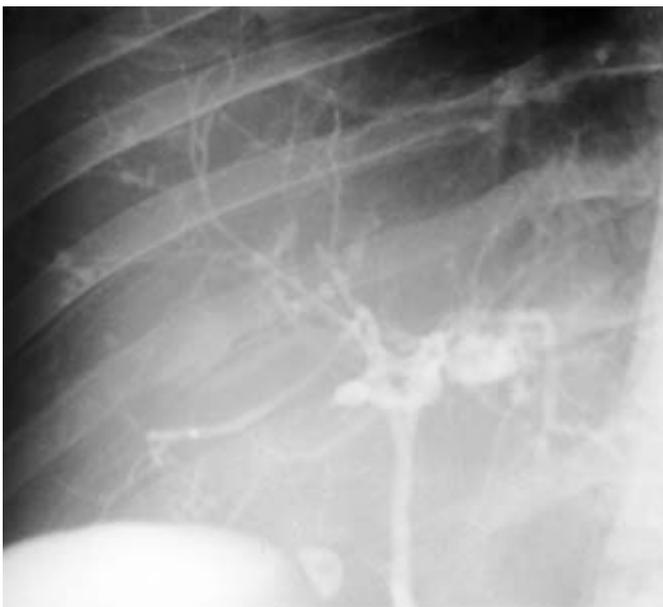
**Hepatic abscess (cont.)**

A repeat examination (Fig. 4.115) after clinical improvement revealed multiple stones in the gallbladder, and another above a stricture in the left hepatic duct. Elective surgery proved successful.

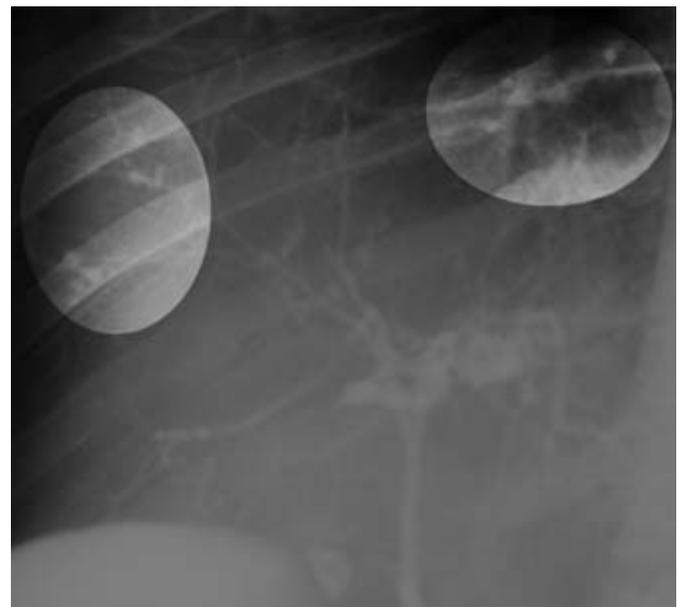
Multiple intrahepatic abscesses are shown in the peripheral regions of the biliary tree of a patient who presented with a pyrexia and abnormal liver function tests (Fig. 4.116). Resolution followed antibiotic therapy. The most common organisms found in liver abscesses are *E. coli*, *Streptococcus* spp. and anaerobes.



4.115



4.116

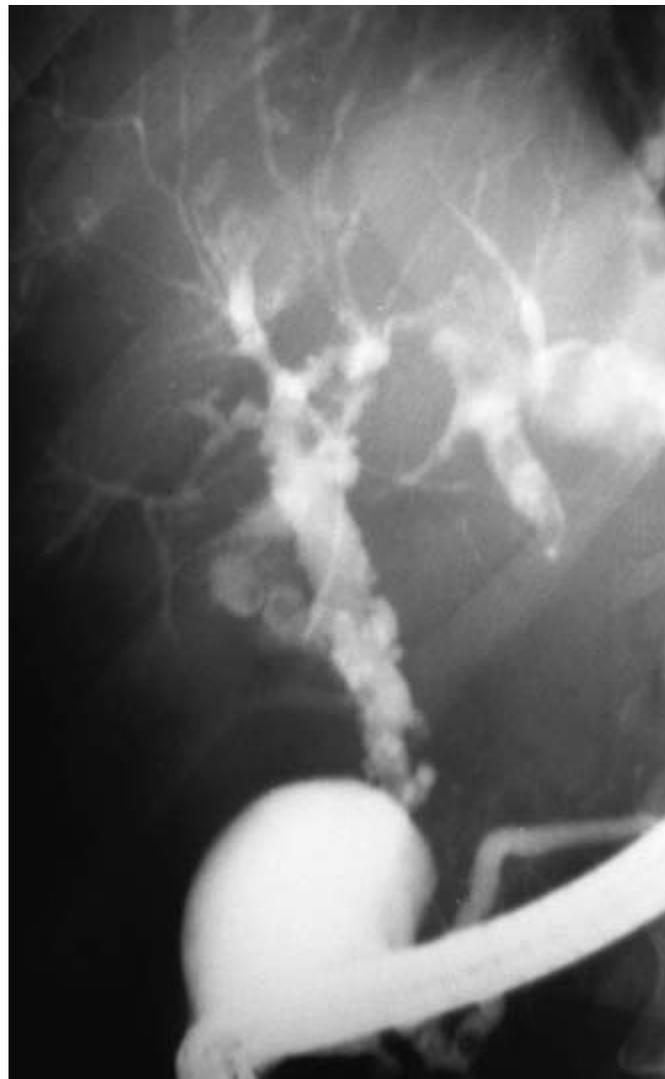


### Sclerosing cholangitis

Multiple intrahepatic and extrahepatic strictures associated with areas of dilatation are characteristic of this condition. The biliary tree may be grossly distorted and filling irregular and sometimes impossible. When occurring in isolation or in association with inflammatory bowel disease the term 'primary' sclerosing cholangitis (PSC) is used. When gallstones or another hepatobiliary disease coexist it is 'secondary' and presumed to be the result of cholangitis. Calculi often form in poorly drained but dilated segments of the intrahepatic tree in PSC, further complicating the terminology.

It may be very difficult to differentiate sclerosing cholangitis from cholangiocarcinoma which if multifocal can give a similar appearance, although intrahepatic and extrahepatic tumours rarely occur together. Malignant change can supervene in established sclerosing cholangitis when the radiological picture may be extremely complex and puzzling.

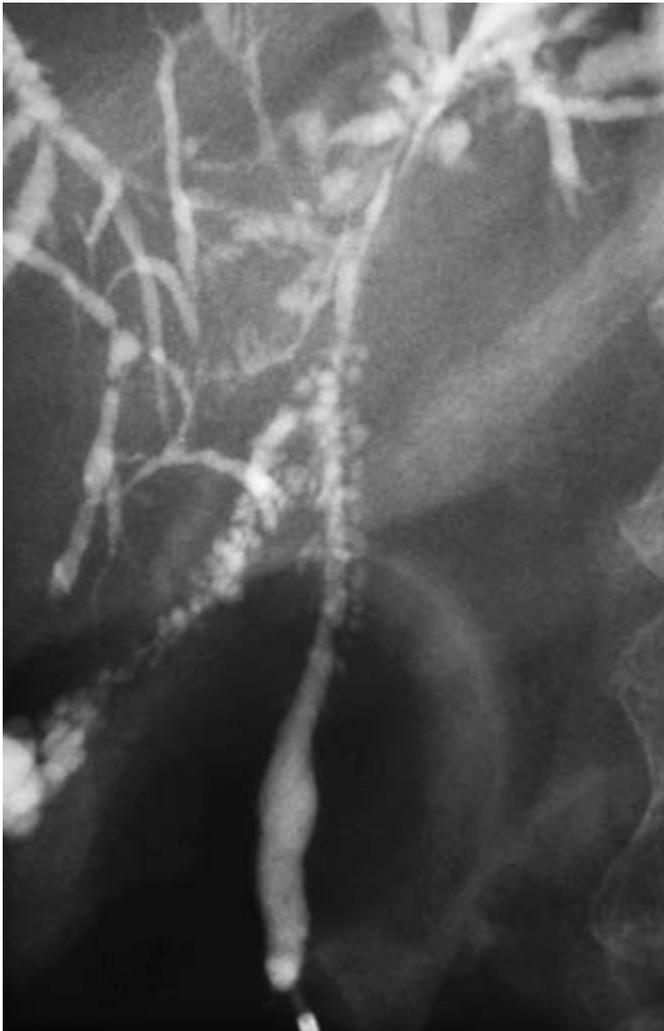
Minor changes of PSC are usually observed as slight irregularities in calibre of the intrahepatic ducts and sometimes there may be considerable difficulty distinguishing early changes from variations of normal anatomy. Figure 4.117 shows definite changes. There is filling of the irregular common ducts with contrast entering a narrowed left hepatic duct, dilatation of the peripheral part of the left hepatic duct and irregularity of other intrahepatic ducts.



4.117

**Sclerosing cholangitis (*cont.*)**

Sacculation of the large ducts, a characteristic feature, is well shown in Fig. 4.118. Often the gallbladder is strikingly enlarged in PSC (Fig. 4.119).



4.118

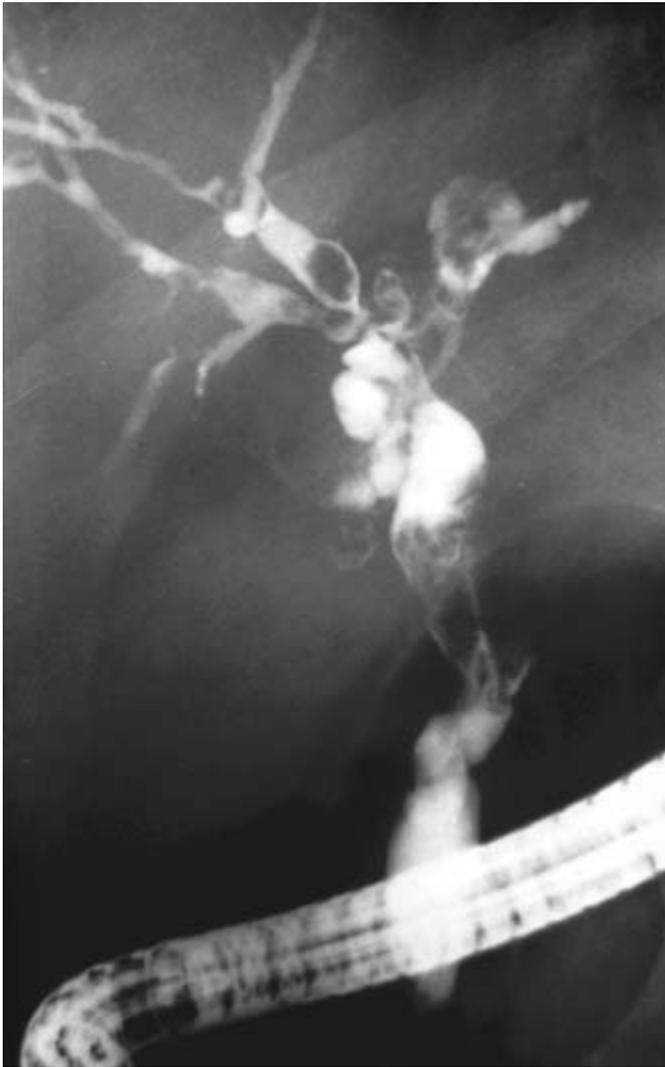


4.119

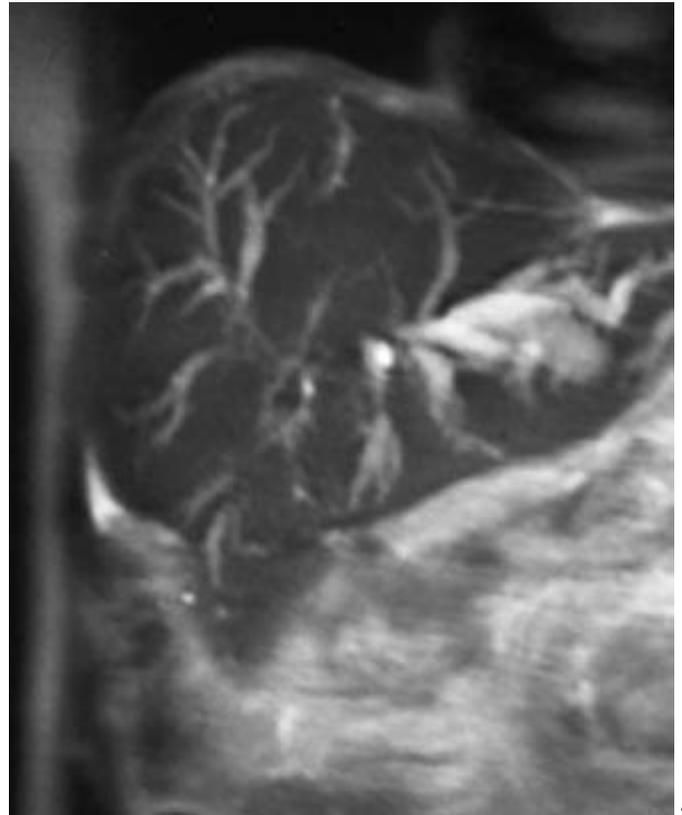
### Sclerosing cholangitis (*cont.*)

Figure 4.120 shows multiple stones in ducts of various sizes within the liver. Here it is impossible to say whether the stones are causative of or secondary to the strictures.

There is a serious risk of introducing secondary infection into the biliary tract when performing ERCP in sclerosing cholangitis of whatever cause. Magnetic resonance cholangiography (MRCP) may therefore be preferred for anatomical imaging reserving ERCP and percutaneous procedures for those cases where brush cytology or therapeutic intervention are required. Figure 4.121 is an MRCP image showing marked irregularity of the intrahepatic ducts with striking dilatation on the left. The ducts at the hilum and the common hepatic duct are not seen.



4.120



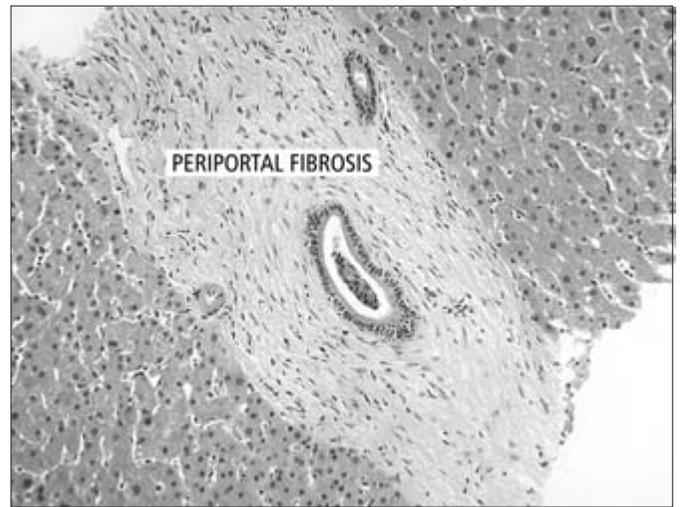
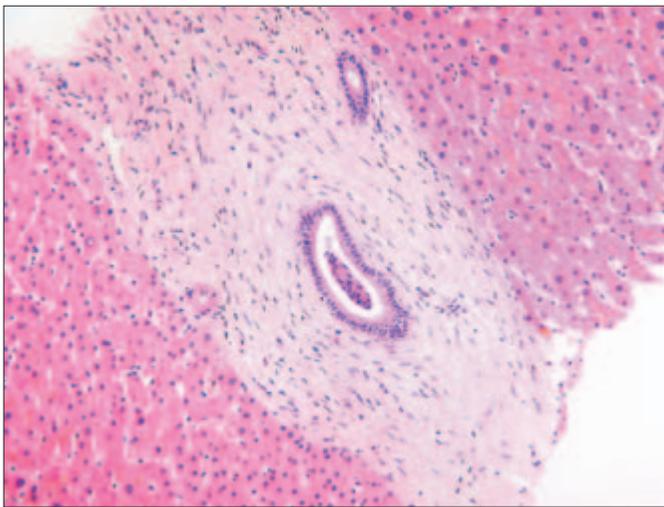
4.121

### Sclerosing cholangitis (*cont.*)

Figure 4.122a shows the macroscopic appearance of primary sclerosing cholangitis, complicated by cirrhosis. The histopathology appears in Fig. 4.122b.



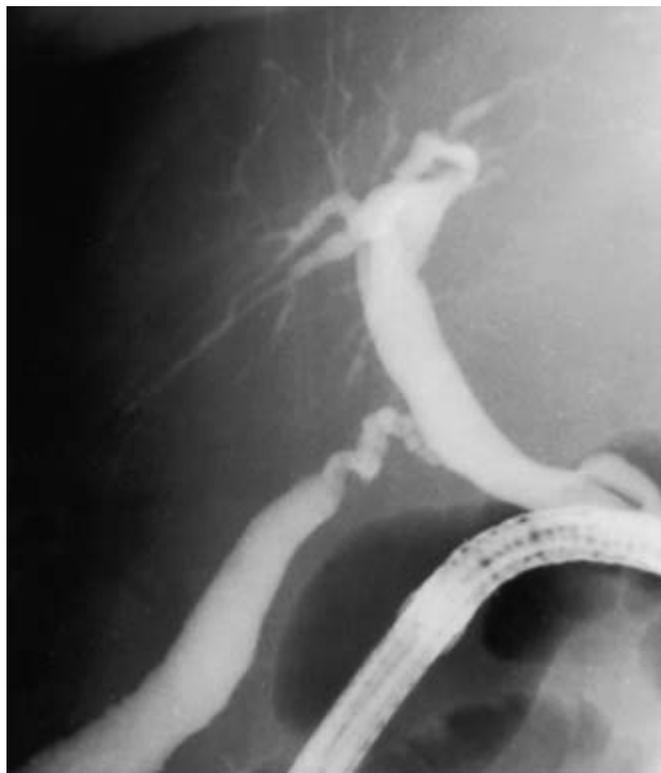
4.122a



4.122b

### Hepatic cirrhosis

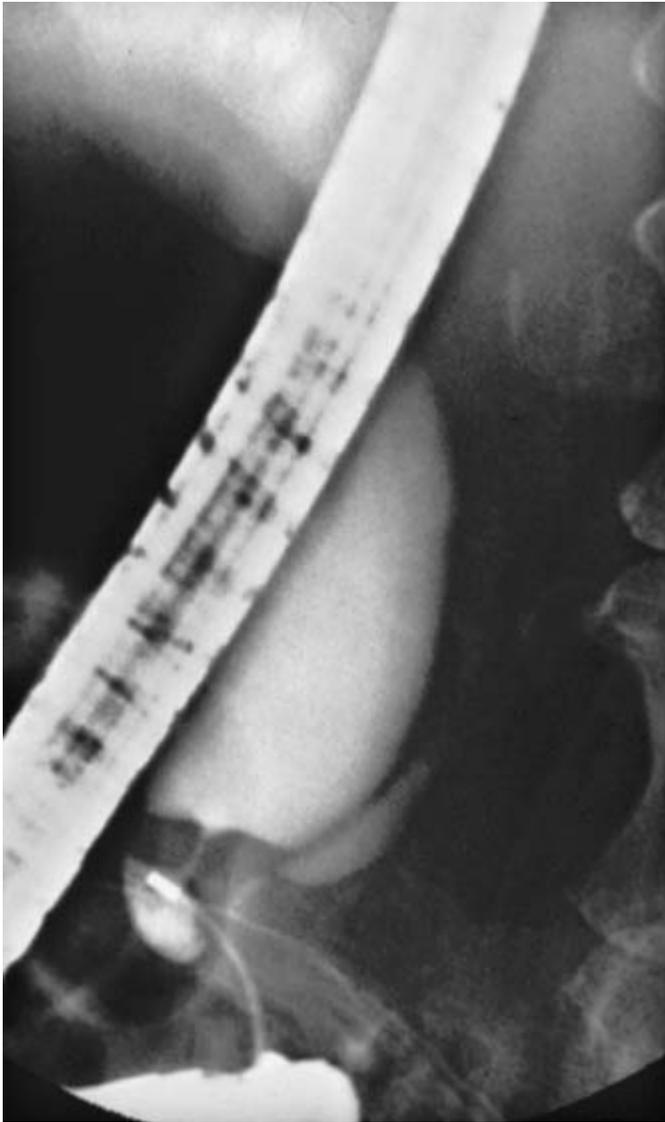
In cirrhosis the liver is often shrunken. The intrahepatic biliary system can then be smaller than normal with a 'pruned tree' appearance (Fig. 4.123). Whilst this is not diagnostic, it should when present suggest the diagnosis of hepatic cirrhosis.



4.123

### Cholangiocarcinoma

Cholangiocarcinoma may involve any part of the biliary system. Short and long malignant strictures involving the CBD at its lower and middle segments, respectively, are shown in Figs 4.124 and 4.125.



4.124



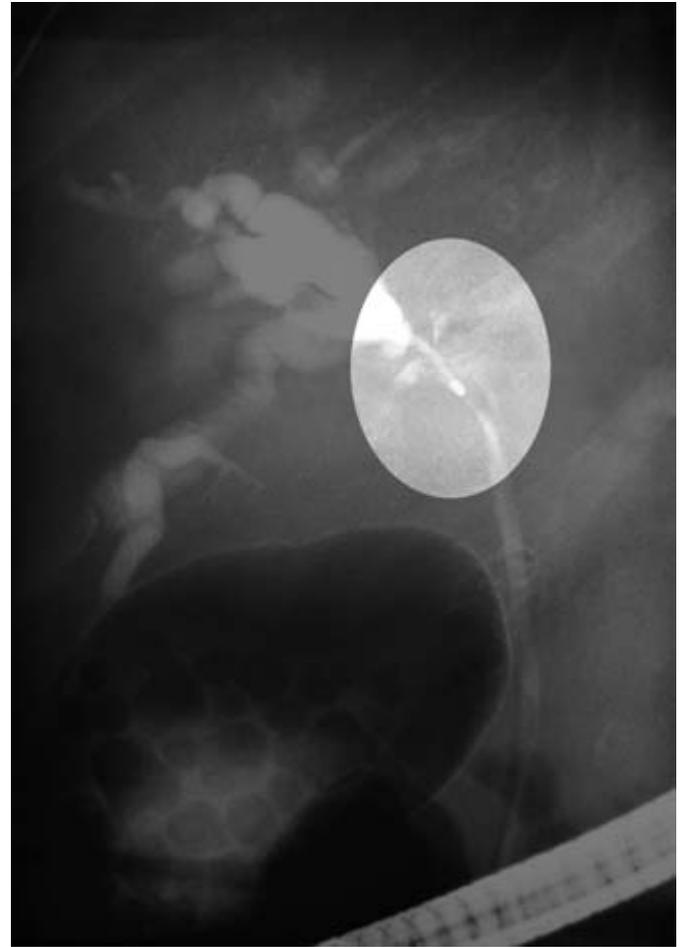
4.125

**Cholangiocarcinoma (cont.)**

Hilar cholangiocarcinoma (Klatskin tumour) may block the confluence of the hepatic ducts and/or the intrahepatic ducts to a varying extent. Figure 4.126 shows a tumour involving the common hepatic duct with just a trickle of contrast passing upwards into the liver.



4.126



**Cholangiocarcinoma (cont.)**

The tumour in Fig. 4.127 has blocked the common hepatic duct completely and an hydrophilic guide wire (see Fig. 4.206) was needed to negotiate the stricture before a catheter could be passed through (Fig. 4.128). Endoscopically derived cytological smears from the biliary tree may confirm malignancy (Fig. 4.44); forceps biopsy is less successful. Alternatively percutaneous fine needle aspiration cytology may be employed.



4.127

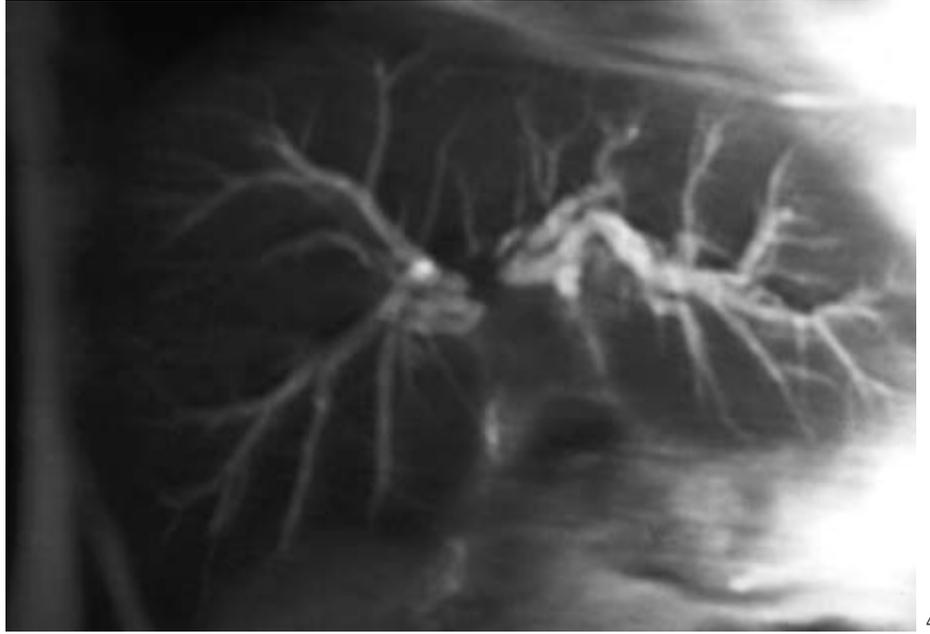


4.128

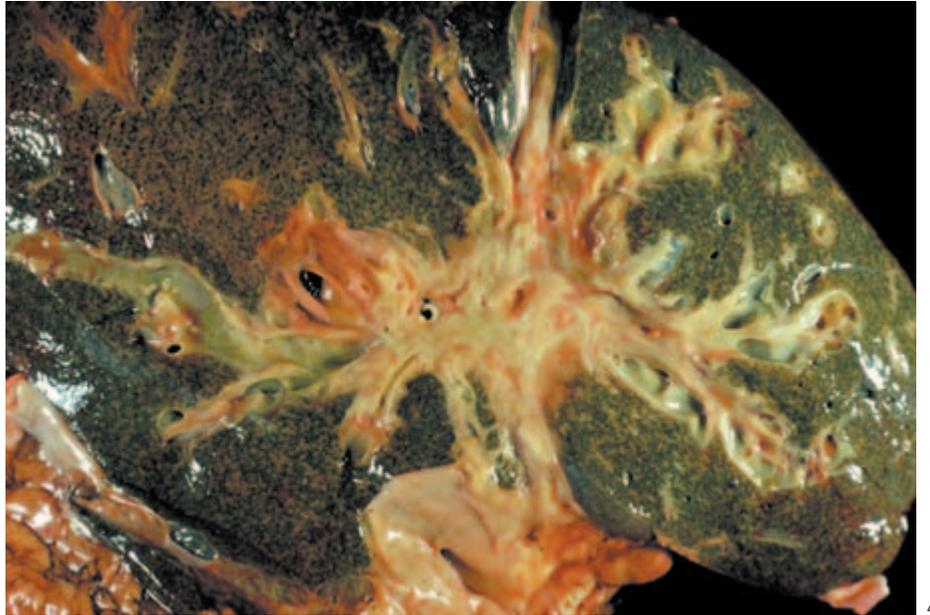


**Cholangiocarcinoma (*cont.*)**

An MRCP picture of an hilar carcinoma is shown in Fig. 4.129. Figure 4.130 illustrates the macroscopic appearances.



4.129



4.130

**Carcinoma of the gallbladder**

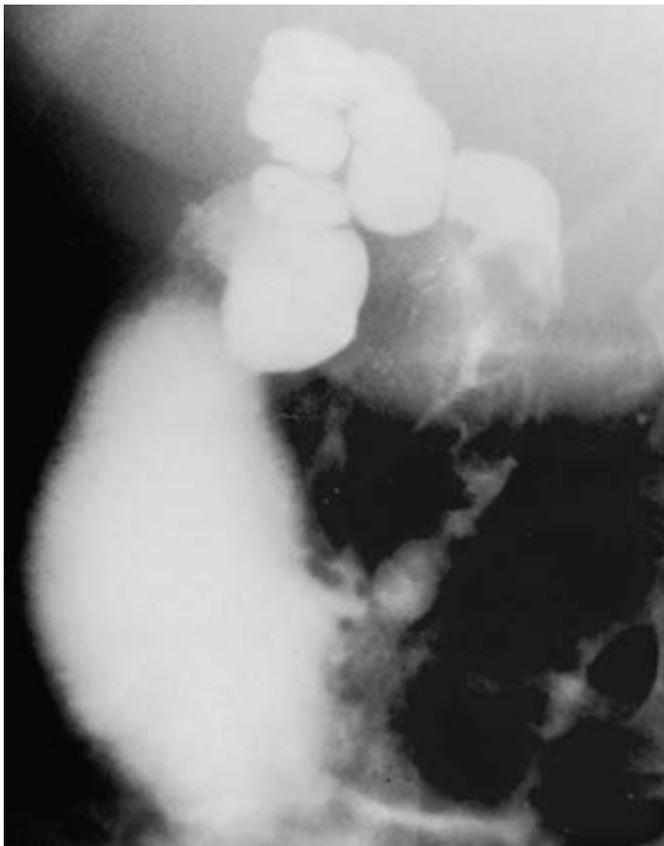
The radiographic appearances of carcinoma of the gallbladder are very variable as the tumour may extend to involve not only the gallbladder but also the cystic duct, common hepatic duct or common bile duct. The tumour shown in Fig. 4.131 has caused obstructive jaundice by invasion of the common hepatic duct and also involves the cystic duct which has not filled. Gallstones are often associated and may be causal.



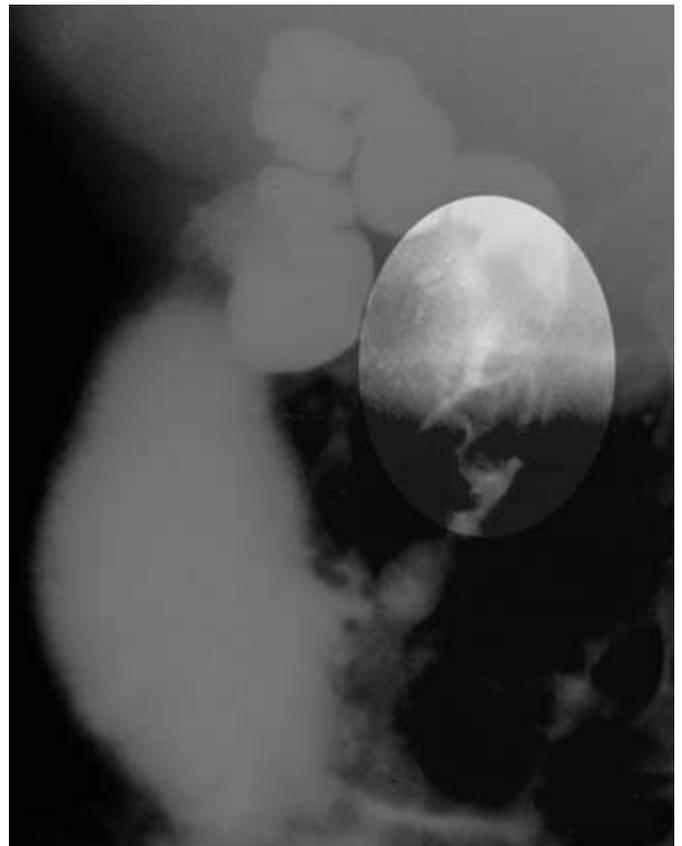
4.131

**Carcinoma of the cystic duct**

Carcinoma of the cystic duct (Fig. 4.132) may cause partial obstruction of the duct with delayed drainage.



4.132



**Differential diagnosis of fixed intraductal 'tumours'**

Fixed filling defects within the bile ducts may be due to benign or malignant polypoid tumours, varices associated with portal hypertension or even blood clot. Differentiation from calculi can be difficult but is clearly most important both in terms of management and prognosis. Balloon ballotment to see whether movement is possible and to gauge the hardness of the lesions may help. Brush cytology is valuable in the detection of malignancy. However blind biopsy and attempts to snare the lesion or grasp it in a basket should be used with the greatest caution lest bleeding or other damage ensues. The following show confirmed diagnoses: Fig. 4.133 a large intraductal tumour and Fig. 4.134 bile duct varices.



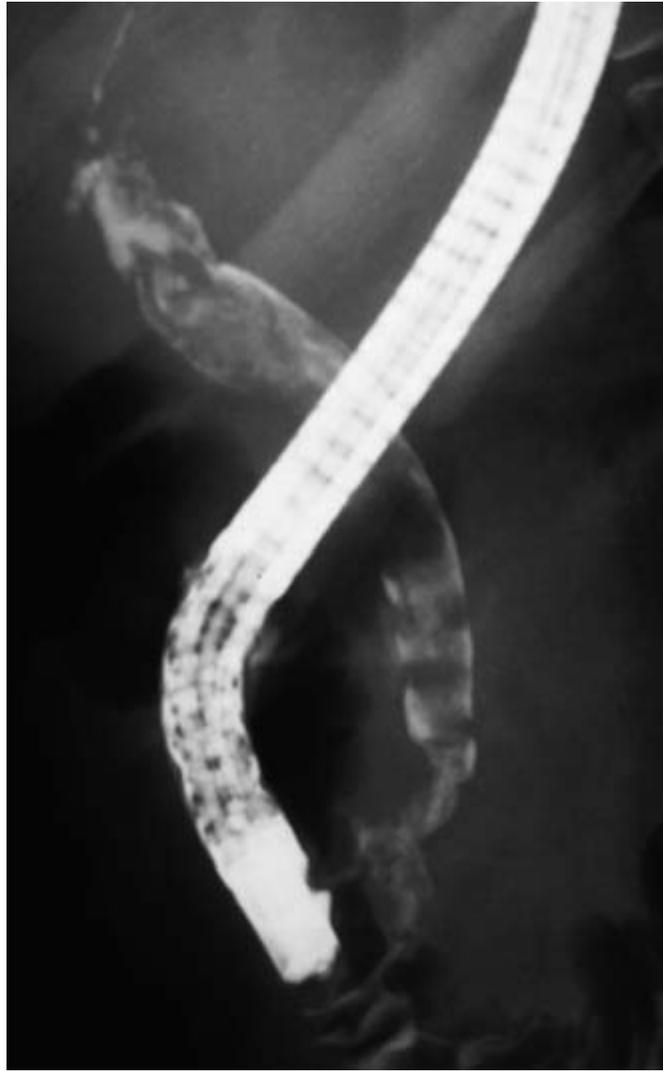
4.133



4.134

**Differential diagnosis of fixed  
intraductal 'tumours' (cont.)**

Figure 4.135 shows blood clot  
resultant from haemorrhage following  
endoscopic sphincterotomy (see  
p. 451).



4.135

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## Appearances following surgery

### ERCP after Polya partial gastrectomy

Unobstructed biliary system after cholecystectomy

Postoperative leak from common bile duct

Operative damage to duct with local collection of bile

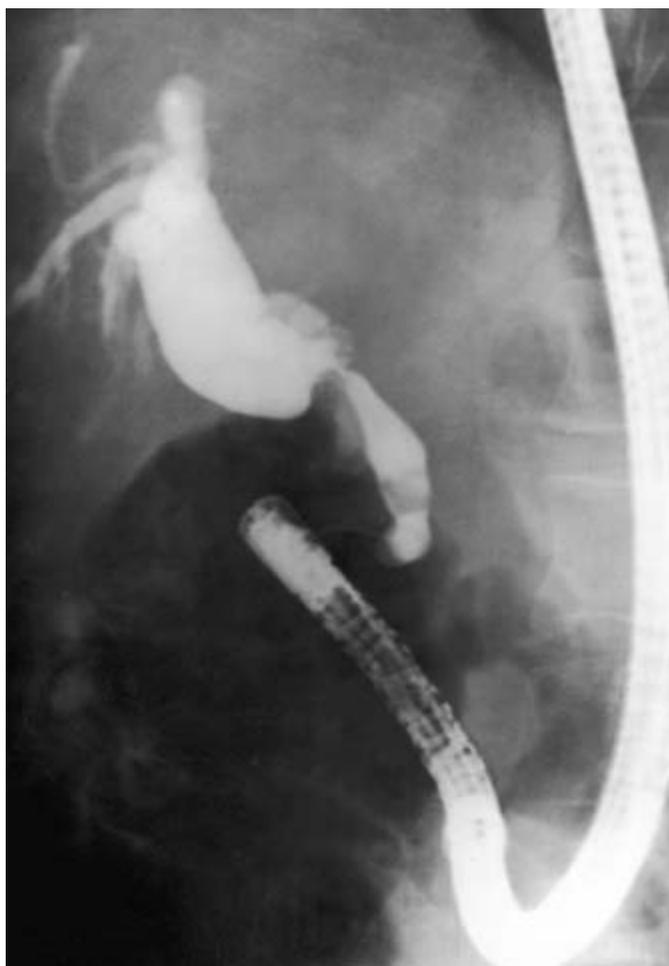
Retained stone in common bile duct (T-tube in situ).

Stricture of common bile duct

Post-laparoscopic cholecystectomy problems

### ERCP after Polya partial gastrectomy

Figures 4.136 and 4.137 show the biliary tree in a patient who had previously undergone Polya (Billroth II) partial gastrectomy. Note the configuration of the endoscope which has resulted from the approach to the papilla via the afferent loop. The initial film (Fig. 4.136) shows a calculus in the common bile duct, and a dilated common hepatic duct above a stricture. Later films reveal in addition multiple stones in the gallbladder (Fig. 4.137). There is no causal relationship between partial gastrectomy and the formation of gallstones.



4.136



4.137

### Unobstructed biliary system after cholecystectomy

ERCP is frequently performed to investigate abdominal pain after cholecystectomy. In Fig. 4.138 the remaining normal tree has been filled; apparent dilatation of the common hepatic duct is due to the overlying cystic duct stump. Injection of contrast may provoke pain, especially when the gallbladder is not present as a 'safety valve' to accommodate excess contrast material. Sometimes a 'sensitive' biliary system may be the result of papillary dysfunction (biliary dyskinesia). On occasions the gallbladder is removed without compelling evidence that the previously diagnosed stones were the cause of the symptoms. In such patients the persistence of pain after operation should not cause surprise. If stones were present in the bile duct at the time of cholecystectomy and removed, the biliary system often remains dilated (Fig. 4.139); no abnormality other than radio-opaque surgical clips on the cystic vessels was shown at ERCP.

Some authorities believe that the CBD may dilate physiologically after cholecystectomy, taking on a gallbladder function, though this is not generally agreed.



4.138



4.139

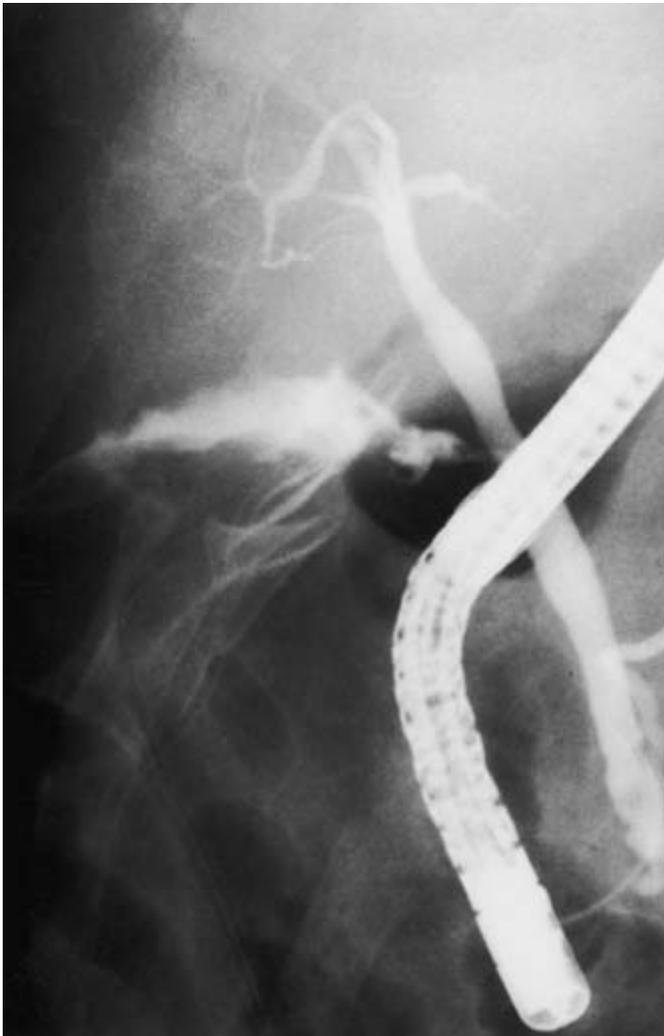
**Postoperative leak from common bile duct**

If jaundice occurs during the recovery period following cholecystectomy ERCP may reveal the cause. Such causes are a retained stone, inadvertent ductal ligation or trauma, nearby haematoma or abscess, or leak of bile into the tissues or peritoneal cavity.

Figure 4.140 shows leakage from the cystic duct stump possibly due to a slipped ligature. The leak settled spontaneously. If there is additionally a stone or a stricture preventing free bile drainage a postoperative leak will not close off unaided.

**Operative damage to duct with local collection of bile**

Damage to the right hepatic duct has resulted in a leak with a collection of bile, sometimes called a 'bile-oma' (Fig. 4.141) which may develop into an abscess. There is also left sided duct narrowing with dilatation beyond suggesting a mass effect due to the collection at the hilum.



4.140



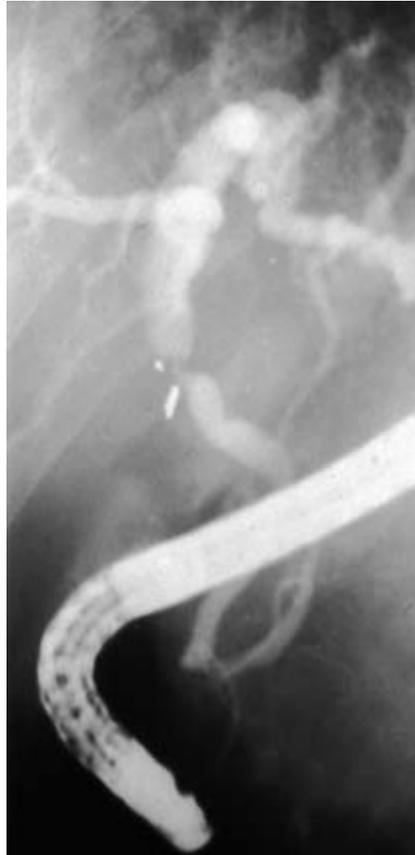
4.141

### **Retained stone in common bile duct (T-tube in situ).**

Even in the best hands a stone is sometimes left behind following cholecystectomy and may be shown on a T-tube cholangiogram. Several non-surgical methods are available for removal including solvent dissolution via the T-tube or endoscopic sphincterotomy at an early stage. After an interval of 5–6 weeks when the track has ‘matured’ it may be possible to remove the stone(s) percutaneously via the T-tube track using a retrieval basket, either under radiological guidance (the Burhenne procedure) or under direct vision using a choledochoscope.

### **Stricture of common bile duct**

After cholecystectomy a stricture of the CBD may result causing pain, jaundice or cholangitis. The stricture shown here (Fig. 4.142) is at the level where clips were applied to the cystic duct and vessels, the most common site for this complication. It is generally assumed that vascular factors are the cause of the majority of postoperative strictures although on occasions direct ductal damage can result from serious surgical errors.



4.142

### Post-laparoscopic cholecystectomy problems

Despite its many advantages, laparoscopic cholecystectomy is more likely than open surgery to result in damage to the bile ducts. Thermal injury and inaccurate clip placement may both be demonstrated by ERCP. Figure 4.143 shows blockage of the right hepatic duct due to diathermy injury. Figure 4.144 clearly shows a surgical clip which has been placed across the common bile duct causing complete obstruction.



4.143



4.144

**Post-laparoscopic cholecystectomy problems (cont.)**

A percutaneous transhepatic cholangiogram (PTC) in the same case (Fig. 4.145) shows dilatation of the bile duct and irregularity above the site of the occlusion. The role of ERCP (and PTC) in this context is predominantly diagnostic and is of help in planning corrective surgery. However, biliary leaks and some strictures may be amenable to endoscopic therapy (see pp. 440–442).



4.145

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## ERCP and liver transplantation

### 'Normal' duct-to-duct anastomosis

#### Gallbladder conduit

#### Anastomotic stricture

#### Leakage at biliary anastomosis

#### Hepatic ischaemia

#### Recurrent or persistent disease

A new role for ERCP has developed following the establishment of liver transplantation programmes. Sometimes diagnostic procedures are required in the work-up before surgery but it is particularly in the diagnosis and management of postoperative problems that ERCP is valuable.

Before considering ERCP after liver transplantation it is essential to obtain information about the type of anastomosis between donor duct and the recipient. If a long Roux loop of jejunum has been created ERCP is unlikely to be successful or useful, but when the biliary anastomosis drains through the recipient's papilla access for ERCP is normal.

### 'Normal' duct-to-duct anastomosis

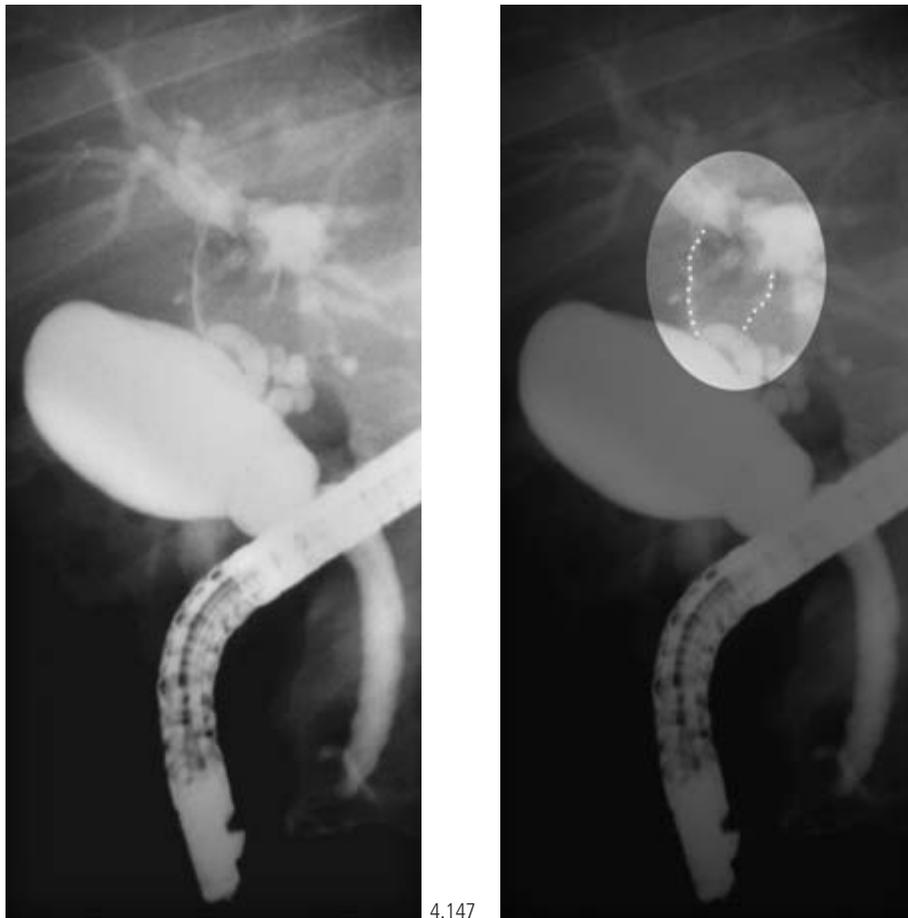
Whenever possible the donor bile duct is joined to the recipient's as shown in Fig. 4.146. The slight difference in ductal diameter as between donor and recipient is to be expected. The intrahepatic ducts are all normal and there is a short cystic duct stump.



4.146

### Gallbladder conduit

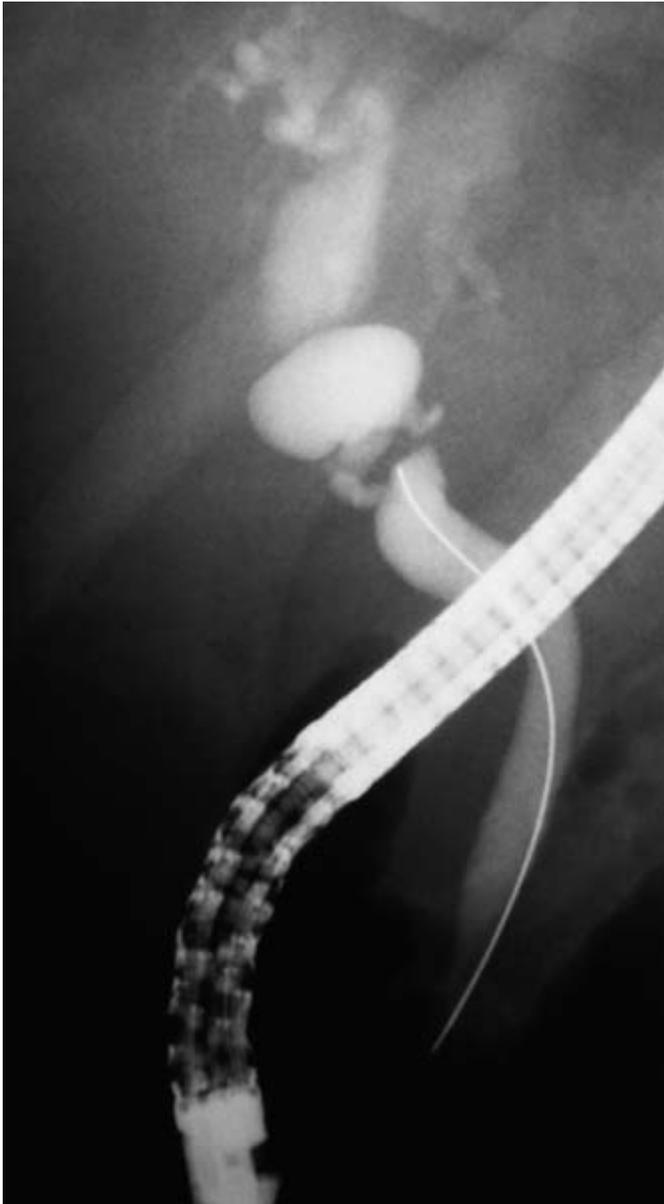
Earlier liver transplant operations often used the donor gallbladder as a conduit anastomosed to the recipient gallbladder or bile duct. Late complications were frequent with strictures and calculi being particularly troublesome. Figure 4.147 shows a large stone in the donor common hepatic duct.



4.147

**Anastomotic stricture**

Anastomotic strictures (Fig. 4.148) are typically tortuous and frequently not negotiable with an endoscopic guidewire. For therapeutic procedures the percutaneous approach may be more satisfactory. In Fig. 4.149 a PTC demonstrates the complexity of anastomotic strictures (and another at the hilum) and also complete obstruction by a gallstone at the lower end of the recipient's common bile duct. Neither endoscopic nor interventional radiological therapy was successful and surgical reconstruction was required.



4.148



4.149

### Leakage at biliary anastomosis

Leakage at the bile duct anastomosis, which predisposes to infection and stenosis, may be demonstrated by ERCP. In the presence of a stricture combined endoscopic and radiological procedures (see pp. 444–445) may sometimes be helpful. Here a stricture and a small leak at the duct-to-duct junction are shown (Fig. 4.150). Internal drainage was provided using a stent (p. 434) which was removed when the leak had closed off.

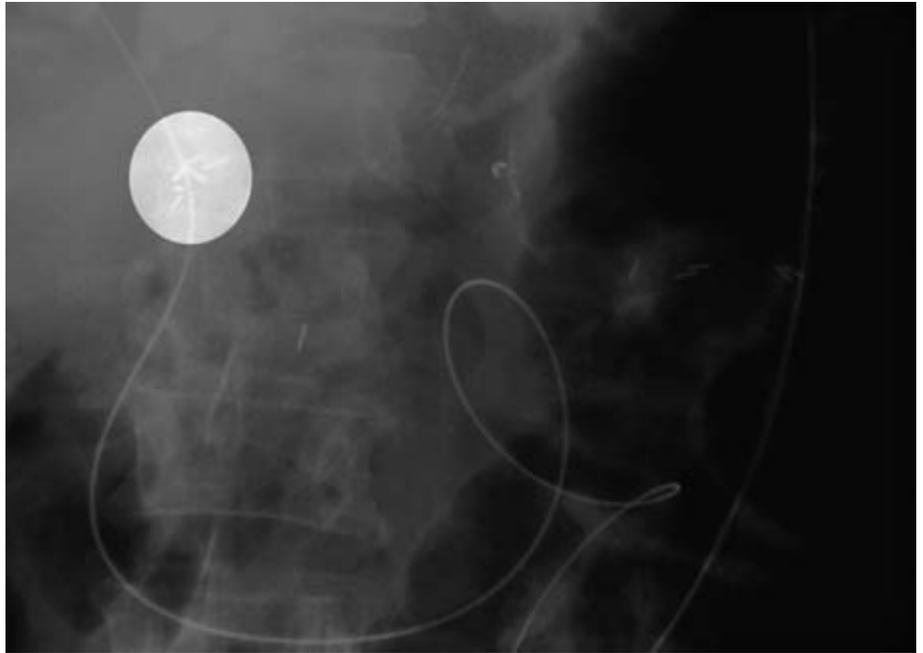
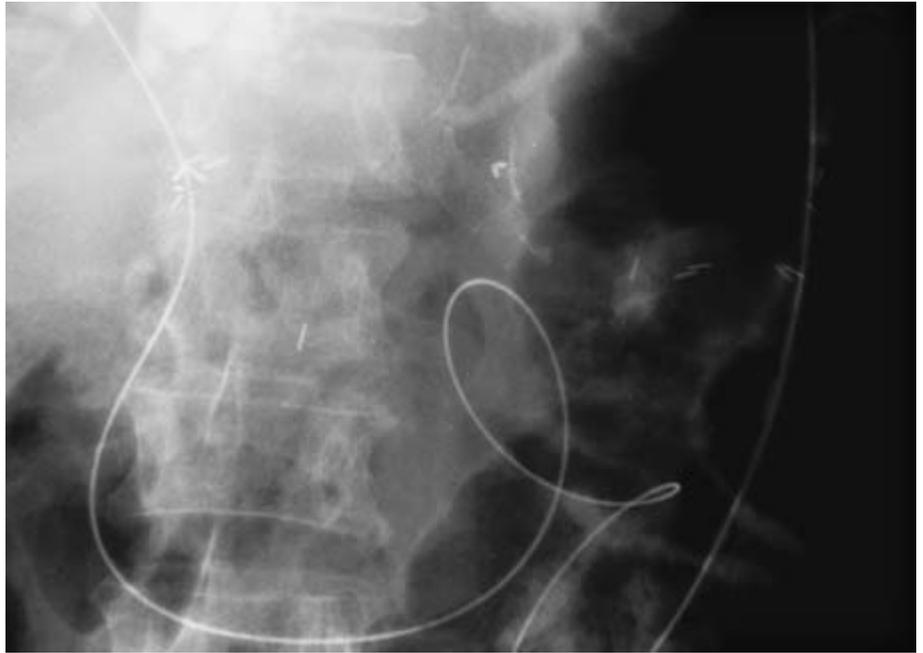


4.150



**Leakage at biliary anastomosis (cont.)**

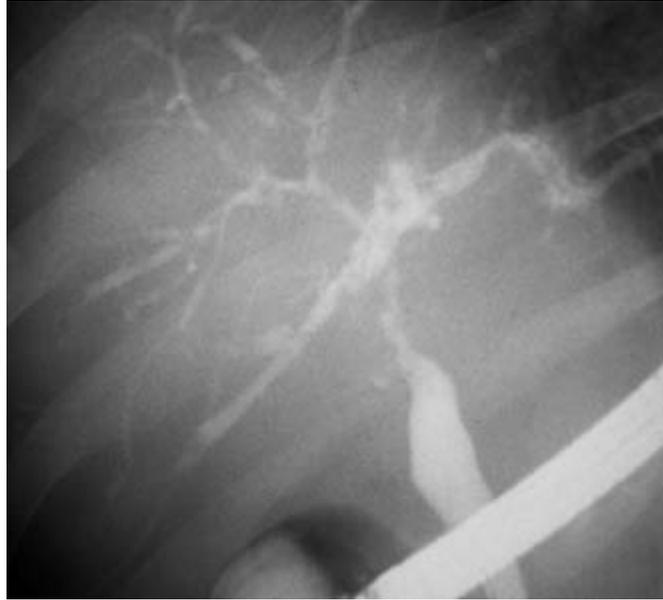
Figure 4.151 shows a guide wire placed percutaneously through an anastomotic stricture following choledocho-jejunostomy-en-Y. The wire has been passed as far as the stomach with a view to leading an endoscope along the jejunal loop back to the stricture. The complex configuration of the jejunal loop demonstrates how unlikely it would be for direct endoscopic access without the lead provided by such a guide wire to be successful in reaching the biliary anastomosis.



4.151

**Hepatic ischaemia**

The irregular narrowing and indistinct outlines of the hepatic ducts within the transplanted organ (Fig. 4.152) are characteristic of ischaemic liver damage.



4.152

**Recurrent or persistent disease**

Sometimes the disease for which transplantation was performed recurs within the graft. Persistence of disease in the recipient bile duct may also be seen. Here (Fig. 4.153) the changes of sclerosing cholangitis, narrowing and sacculation, are noted in the lower bile duct whilst the upper portion and the intrahepatic ducts are normal.



4.153

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## Therapeutic procedures

**Sphincterotomy 419**

**Stone removal 423**

**Nasobiliary drainage 430**

**Management of difficult or large gallstones 432**

**Internal biliary drainage: stent (endoprosthesis) placement 434**

**Balloon dilatation of strictures 440**

**Suprapapillary bile duct puncture 443**

**Combined or 'rendezvous' procedure 444**

**Therapy of bile duct injuries 446**

**Endoscopic therapy of pancreatic disease 448**

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### Sphincterotomy

Instrumentation and technique

Appearances after sphincterotomy

#### Instrumentation and technique

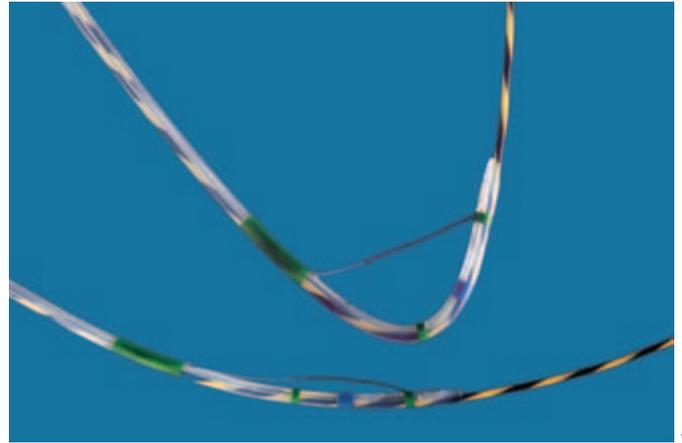
Techniques evolved from diagnostic ERCP are used for treating several disorders of the papillary region, the biliary system and pancreas. Duodenoscopic sphincterotomy (endoscopic papillotomy) of the papilla of Vater can be performed by a diathermy incision using a standard side-viewing duodenoscope. More specialised instruments with a larger operating channel facilitate instrumentation of the papilla.

Before considering sphincterotomy the patient's coagulation status must be assessed and deficiencies corrected as the procedure is contraindicated if there is a bleeding tendency, often present with jaundice. As an alternative to incision of the papilla, the orifice may be dilated to some extent using a balloon catheter. These procedures allow access to the ducts for removal of calculi or insertion of a stent (endoprosthesis) for drainage of bile or pancreatic juice.

Endoscopic techniques provide a cost-effective, often safer and more rapid alternative to surgical operation. The detailed indications for and selection of patients for treatment by these methods are beyond the scope of this work as are details of patient preparation and the finer points of instrumentation and technique. A few representative examples are illustrated.



4.154

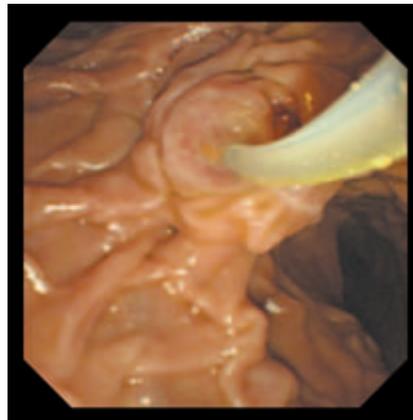


4.155

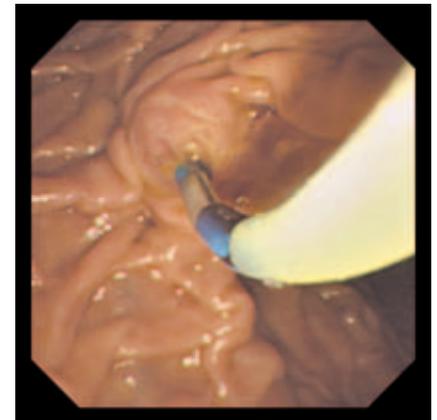
### Instrumentation and technique (cont.)

Incision through the papilla of Vater is performed with a diathermy wire (papillotome) exposed at the distal end of a Teflon tube of diameter similar to that of a diagnostic ERCP cannula. Several designs are available commercially, the most popular being the Erlangen type. Papillotomes vary with respect to tip characteristics, and in the length and position of exposed wire (Fig. 4.154). Cannulation is performed with the wire relaxed. Once positioned the wire is bowed (Fig. 4.155) and elevated, exerting gentle pressure on the roof of the papilla during incision.

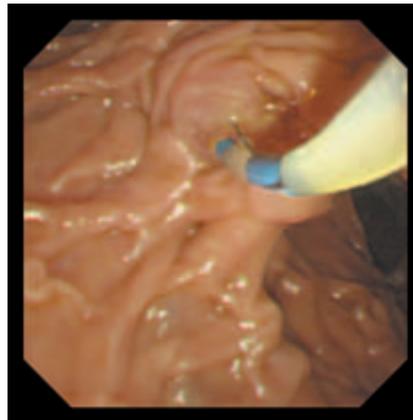
Selective cannulation of the common bile duct is an essential prelude to satisfactory biliary sphincterotomy. The papillotome is advanced deeply (Fig. 4.156) into the common bile duct and its position is checked fluoroscopically. After verification, the papillotome is gradually withdrawn until the diathermy wire becomes visible (Fig. 4.157). The position is adjusted until about half of the wire is exposed or to the level indicated by markers on the papillotome. The wire is next gently tensioned (Fig. 4.158). Short bursts of diathermy current are applied to make an incision in stages into the lower end of the bile duct (Figs 4.159 and 4.160). Control is provided by the endoscopist raising the bridge or elevating the tip of the endoscope. Reliance on tension in the bowed papillotome leads to unpredictable and potentially dangerous results.



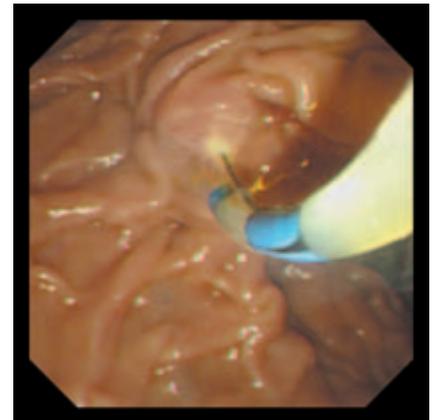
4.156



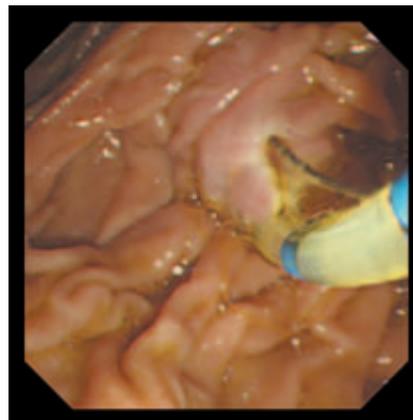
4.157



4.158



4.159



4.160

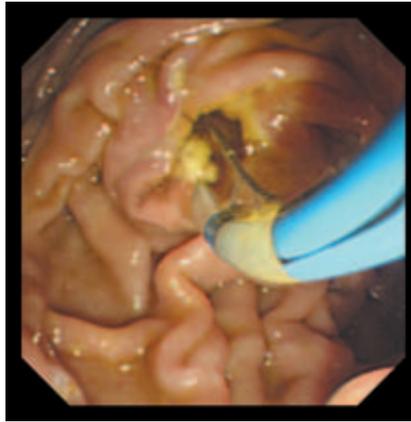
### Instrumentation and technique (cont.)

The size of the sphincterotomy is tailored according to its purpose, in particular to the size of a stone to be removed. Figure 4.161 shows an opening approximately 1.5 cm in length through which the bowed papillotome can easily be inserted and withdrawn. Precise estimation of the size of a sphincterotomy incision may be difficult as the surface cut may appear larger than the deeper opening created. Measurement using an inflated balloon catheter of known dimensions drawn through the aperture is a useful aid.

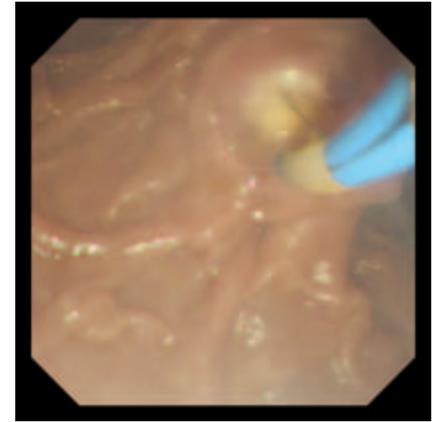
During the procedure the view may be partially obscured by steam and smoke (Fig. 4.162) resulting from the heat generated by the diathermy current. Often there is a gush of bile, or pus in the presence of cholangitis. Slight bleeding is common. Serious haemorrhage, which is the most frequent complication of this procedure, occurs in about 5% of cases. Complications of ERCP are discussed further on p. 451.

Sometimes it is not possible to advance the papillotome sufficiently deeply into the orifice to perform a standard sphincterotomy. In this situation, for therapeutic but not routine diagnostic purposes, it is acceptable to use a precut papillotome or needle-knife (point diathermy) (Fig. 4.163) to incise the papilla so as to cut a channel into the bile duct. This procedure should be employed with extreme caution and only after consideration of the possible risks of uncontrolled and blind incision or puncture through the ducts or duodenal wall. Once entry has been achieved the standard papillotome is re-inserted and sphincterotomy completed.

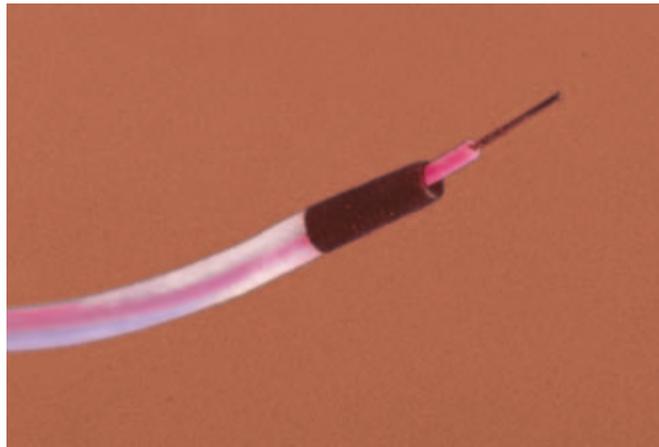
Sphincterotomy via the afferent loop after Polya (Billroth II) partial gastrectomy presents problems in orientation of the incision. Special cannulae have been designed for cutting in a cephalad direction.



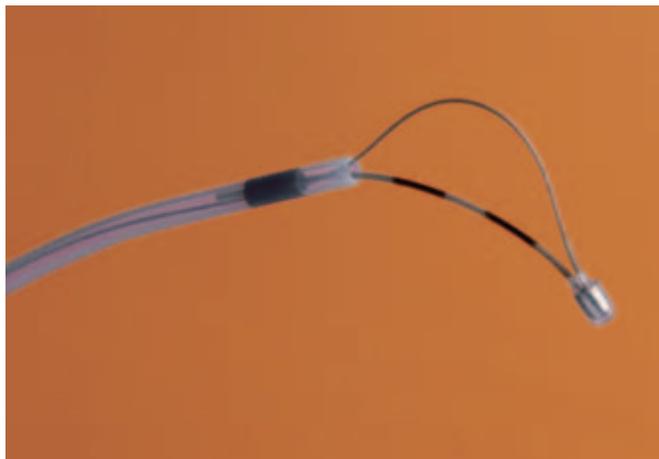
4.161



4.162



4.163



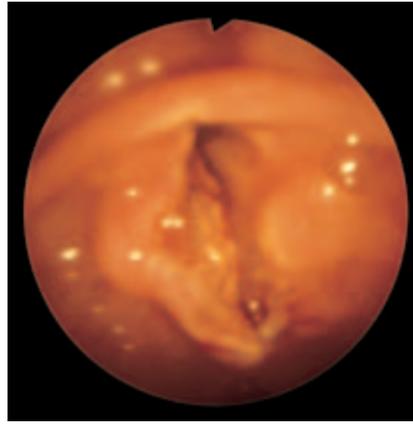
4.164

Examples are 'shark's fin' and reversed curve cannulae (Fig. 4.164). An alternative technique is to insert a short plastic 5F stent through the papilla into the bile duct initially, and then to perform sphincterotomy by cutting down onto this stent with a needle knife.

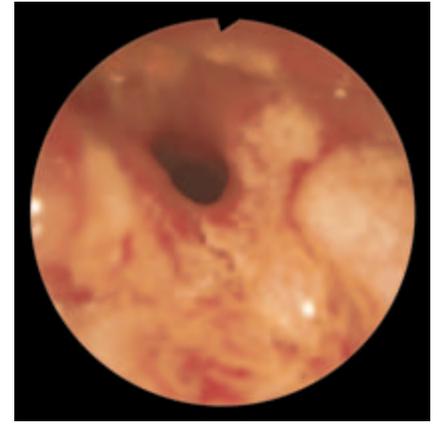
### Appearances after sphincterotomy

Immediately after sphincterotomy a wide opening may be apparent, allowing a view into the lower end of the bile duct (Figs 4.165 and 4.166). Even in the presence of a duodenal diverticulum good access may still be possible (Fig. 4.167). Often due to coagulation of tissue in the region of the incision or to slight bleeding, a free passage though present, is not visible (Fig. 4.168); satisfactory stone extraction is still possible provided the incision is large enough.

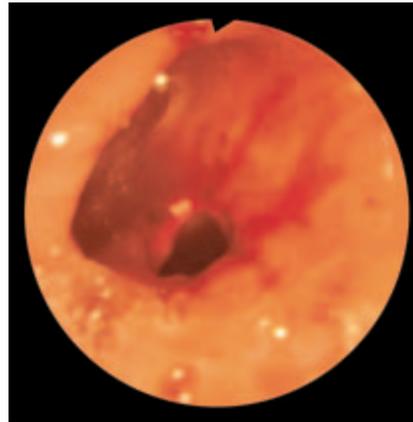
During the weeks after sphincterotomy re-epithelialisation of the incision occurs and its size is reduced, but stenosis is uncommon. Figure 4.169 shows the typical appearance some weeks after sphincterotomy with an apparently intact papilla and a circular 'fistula' above. Insertion of a cannula through the papillary orifice in such cases usually demonstrates a slit-like defect communicating with the 'fistula'. The incision through the papilla rarely closes completely. Although stenosis after surgical sphincterotomy is not uncommon, it is unusual following endoscopic papillotomy.



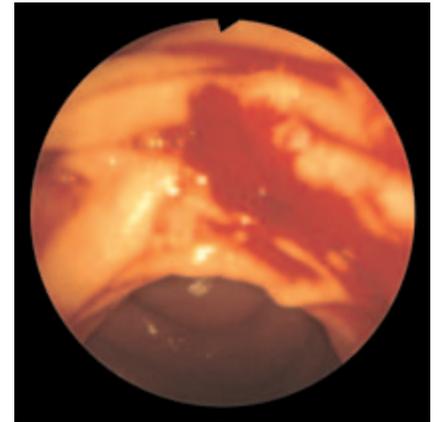
4.165



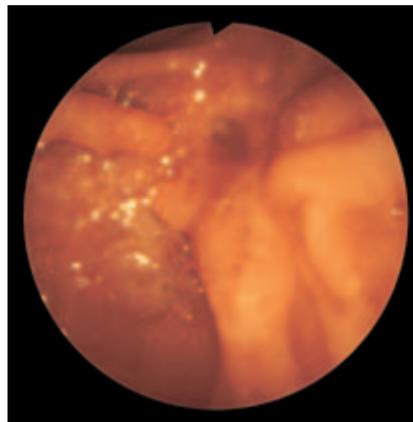
4.166



4.167



4.168



4.169

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## Stone removal

### Instrumentation and technique

### Endoscopic appearances

### Lithotripsy

### Check cholangiography

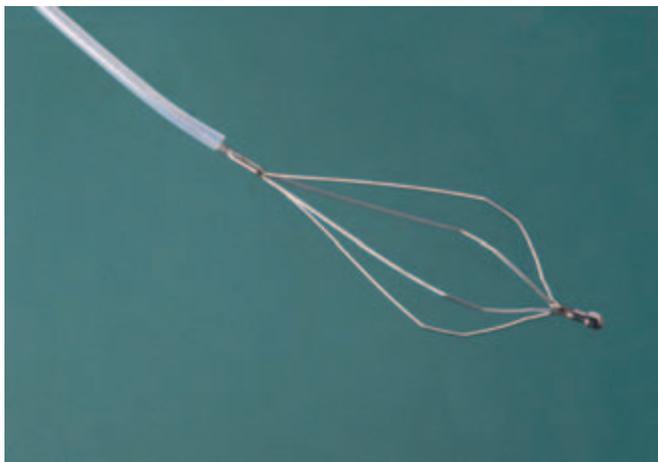
### Occlusion cholangiography

#### Instrumentation and technique

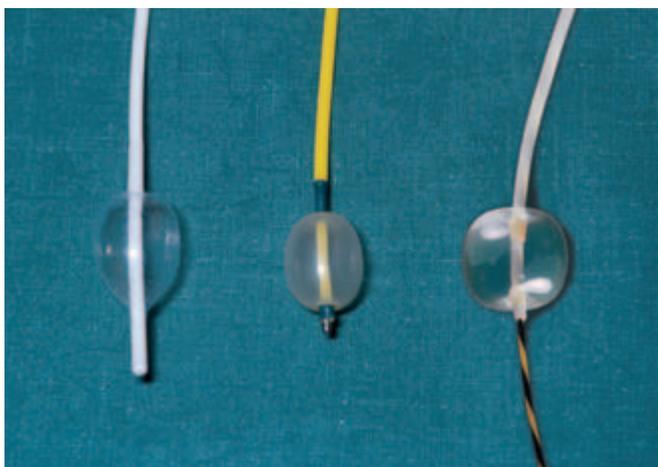
Gallstones are usually extracted from the bile ducts immediately after sphincterotomy using a wire basket (Fig. 4.170) or balloon catheter (Fig. 4.171). Alternatively stones may be allowed to pass spontaneously during the following days when a later cholangiogram will be required to check duct clearance.

Wire baskets of various sizes and designs are the most popular stone-removing devices. They are particularly suitable for larger stones which require active extraction.

Balloon catheters are fragile and easily damaged during attempts to remove large or impacted calculi. However, a balloon is an excellent device for extracting multiple small stones through a widely patent sphincterotomy opening. An additional use is as a gauge to assess adequacy of the incision. If an inflated balloon of appropriate size can be withdrawn easily through the sphincterotomy it is reasonable to expect spontaneous or assisted passage of stones of similar or smaller size.



4.170



4.171

**Instrumentation and technique (cont.)**

To engage stones the closed basket-catheter is advanced beyond the stone and opened. As the basket is withdrawn the wires should trap the calculus (Fig. 4.172). The trapped stone is then pulled down into the lower bile duct with the endoscope in the angled up position (Fig. 4.173); endoscopic vision is often lost at this stage.



4.172



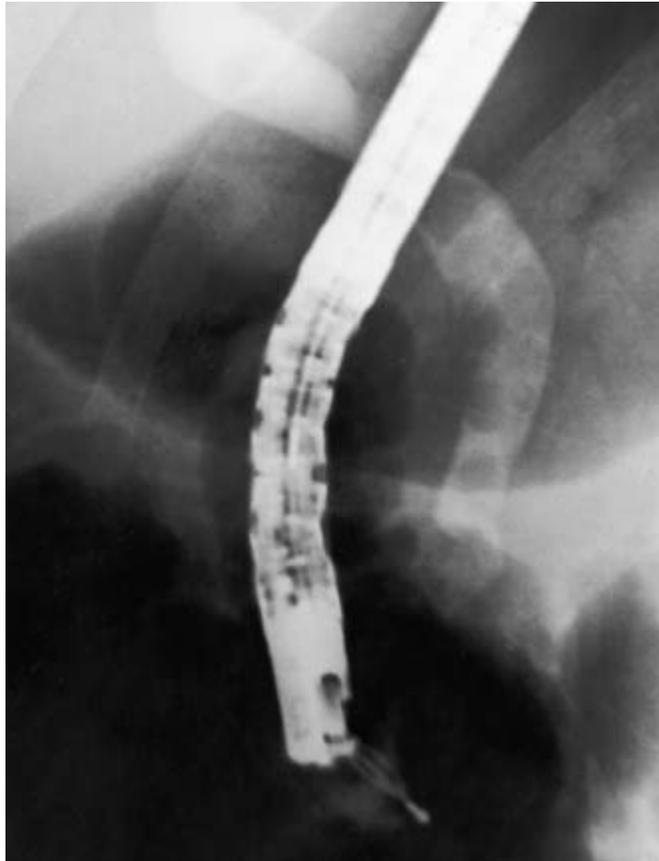
4.173

### Instrumentation and technique (cont.)

Extraction is effected by angling down the tip of the endoscope (Fig. 4.174) which flicks the basket and the contained stone out through the sphincterotomy, after which the basket can be opened to release the stone. As this patient had several stones, repeated introductions of the basket were necessary to obtain duct clearance. An adequately sized incision is essential for satisfactory stone removal and to avoid stone impaction. Following trapping, small stones are usually easily trailed through the sphincterotomy.

It is preferable not to close the basket firmly as the wires may cut into calculi preventing disengagement, which may be necessary if a stone becomes wedged in the sphincterotomy. Should this occur an 'emergency' lithotripter may be used. The handle is removed from the impacted basket with wire cutters and the endoscope is withdrawn over the cut end which is left protruding from the patient's mouth. The stout sheath of the lithotripter is then advanced over the wire until fluoroscopy shows it to abut on to the stone (Fig. 4.175) which can then be crushed using the special handle (Fig. 4.176). The basket is now withdrawn into the sheath (Fig. 4.177) and safely removed.

After extraction stones are usually allowed to pass through the gastrointestinal tract to be voided with the faeces. There is little point in routinely withdrawing stones via the mouth. Rarely following sphincterotomy a large stone may impact in the small intestine causing so called gallstone ileus. Very large stones are too big to be trapped in conventionally sized baskets and are more suitably treated by other methods such as lithotripsy, stent insertion or surgery.



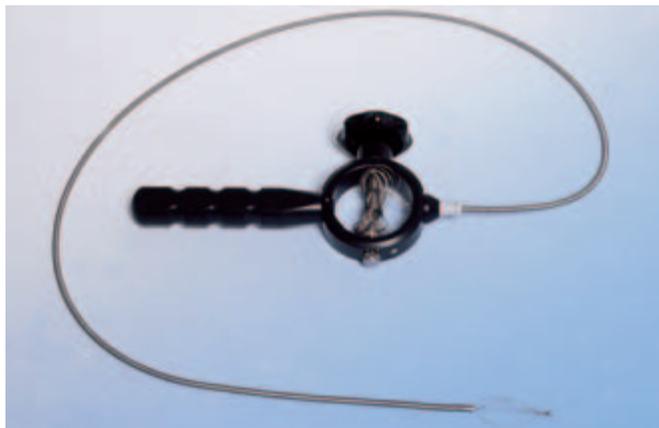
4.174



4.175



4.177



4.176

### Endoscopic appearances

Stones may be brought out with the papillotome (Fig. 4.178) immediately after the sphincterotomy. Small stones are easily withdrawn under direct vision after entrapment in the basket. Figure 4.179 shows a spherical yellow cholesterol stone whilst the dark stone seen in Fig. 4.180 is composed of pigment.

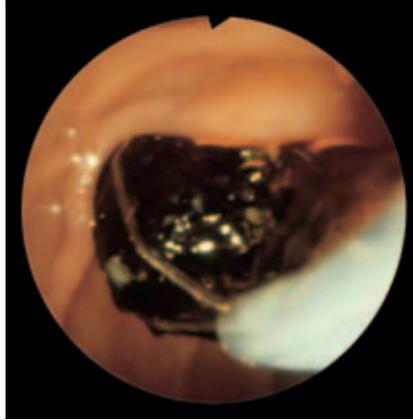
An irregular stone, with a little blood on the surface (Fig. 4.181) is seen escaping from the basket after being withdrawn through the sphincterotomy. Often endoscopic vision is lost during extraction and when the view is re-established the stone may be seen lying free in the duodenal lumen. Figure 4.182 shows a spherical cholesterol stone, adjacent to the recent sphincterotomy. Following papillotomy small stones commonly pass spontaneously (Fig. 4.183).



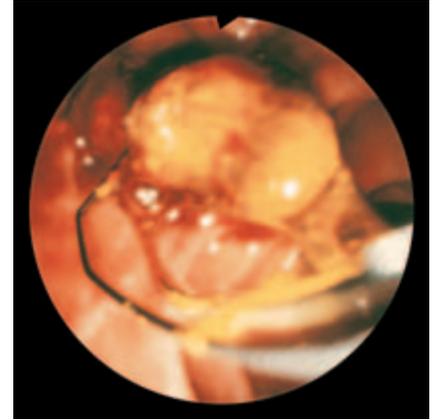
4.178



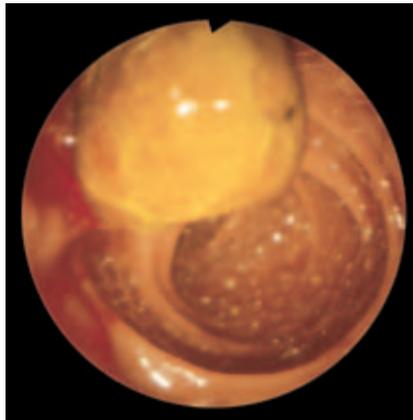
4.179



4.180



4.181



4.182



4.183

### Lithotripsy

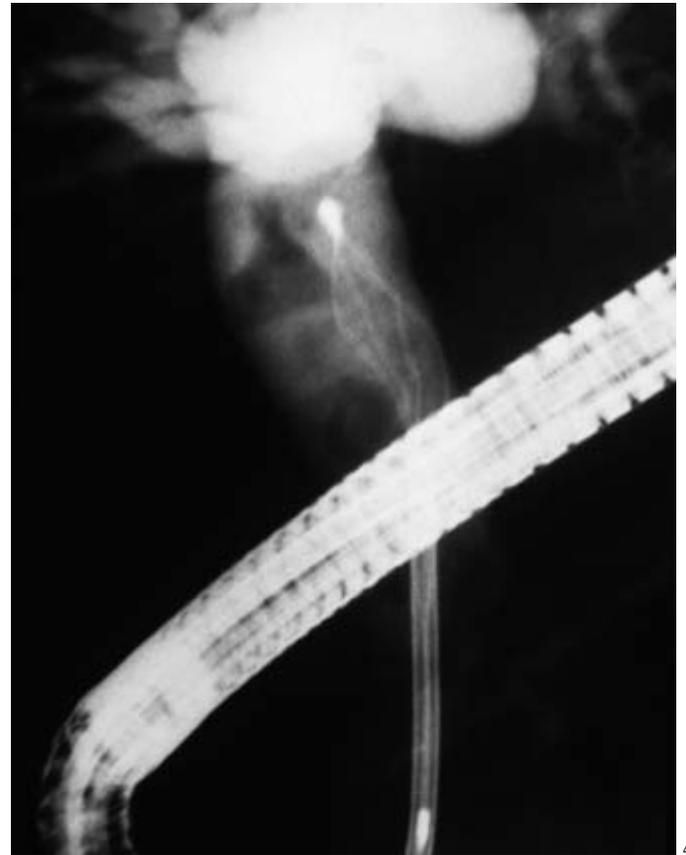
Stones too large for safe extraction through a reasonably sized sphincterotomy, or retained above a narrowing of the bile duct can often be fragmented by lithotripsy. The most frequently employed technique (mechanical lithotripsy) involves grasping the stone(s) in a robust wire basket passed through a rigid sheath (Fig. 4.184) against the end of which the stone is crushed (Figs 4.185 and 4.186) using a special geared or ratchet handle (Fig. 4.187). Other methods fragment stones utilising diathermy, shockwave or laser energy applied by various delivery systems.



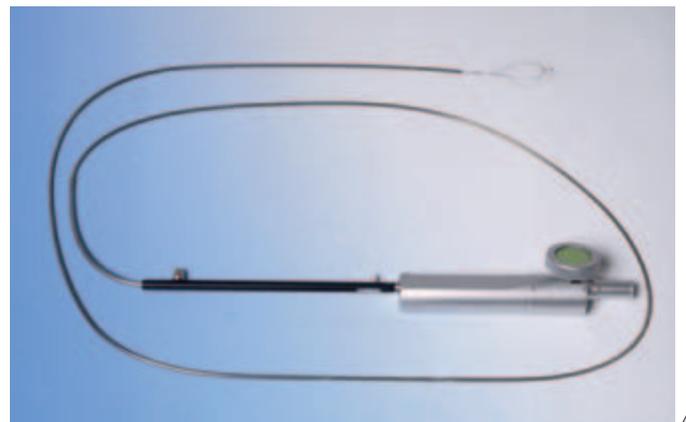
4.184



4.185



4.186



4.187

**Lithotripsy (cont.)**

Electrohydraulic shock wave lithotripsy employs a probe applied to stones under direct vision. To permit this a narrow diameter endoscope is passed up the bile duct following a previous sphincterotomy. The special equipment, ‘mother-and-baby scopes’ (Fig. 4.188), is fragile, requires two skilled endoscopists, is difficult to use and has not gained wide acceptance.



4.188

**Check cholangiography**

When doubt remains about duct clearance after sphincterotomy for stones, check cholangiography and if necessary secondary stone extraction is desirable. Placement of a nasobiliary tube (pp. 430–431) at the time of sphincterotomy may avoid the need for further endoscopy. Alternatively after 2–4 weeks a repeat ERCP is performed. Preliminary films in the presence of a patent sphincterotomy may show an air cholangiogram (Fig. 4.189). Presence of air in the biliary system may pose problems of interpretation (see also Fig. 4.103). To avoid difficulties it is helpful, if possible, first to displace the air. With the patient tipped slightly head-down a cannula is advanced deeply into the biliary system, perhaps into an intrahepatic duct. Saline is infused to flush out the air which bubbles from the sphincterotomy. Next, opacification of the ducts is carried out and a careful search is made for residual stones. Figure 4.190 shows the confusing appearance which may be seen before all the air bubbles have been displaced.



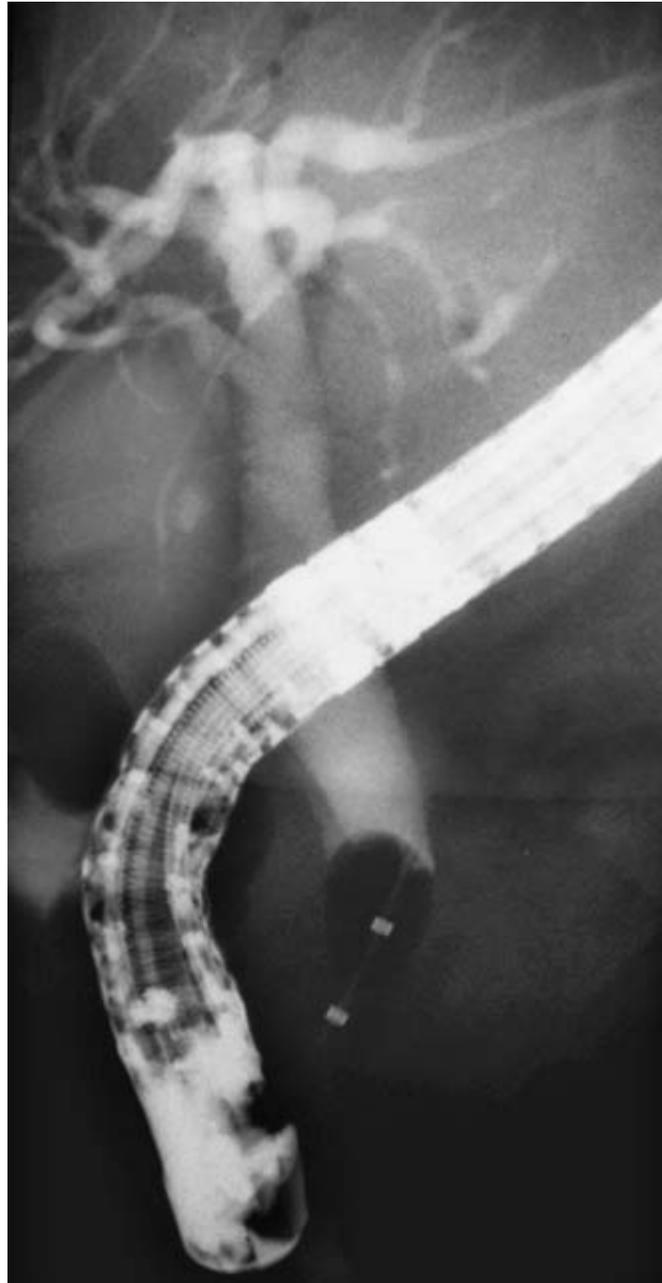
4.189



4.190

**Occlusion cholangiography**

Cholangiography using a balloon catheter allows a tight seal after sphincterotomy (Fig. 4.191) and may be helpful when air cannot easily be displaced as above. Occlusion cholangiography is also valuable after choledochoduodenostomy when sometimes it is very difficult to obtain good quality images.

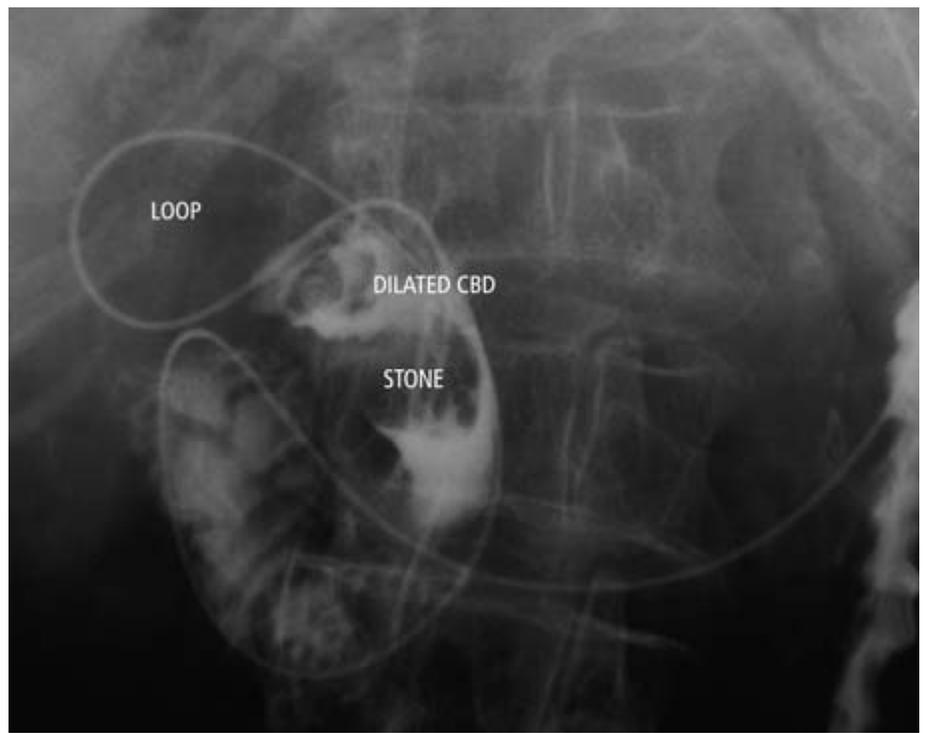
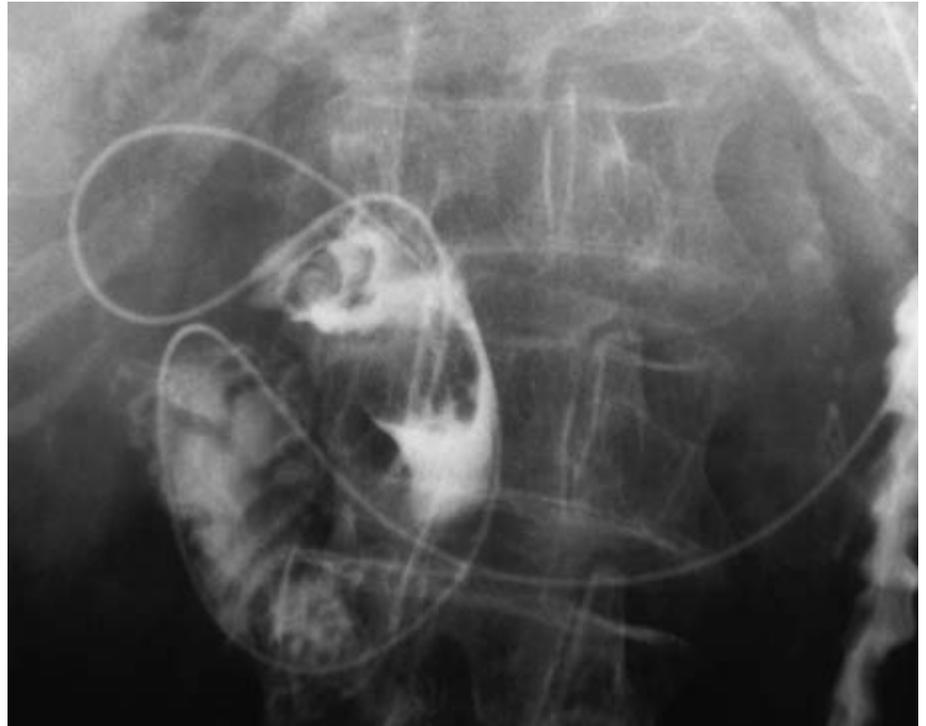
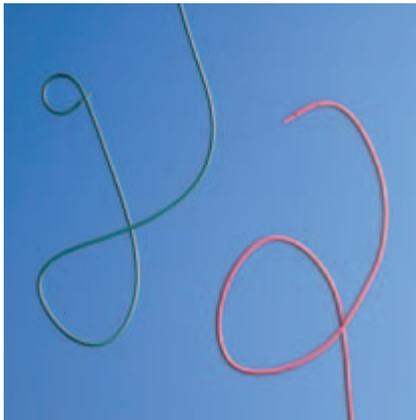


4.191

## Nasobiliary drainage

### Nasobiliary drainage

An indwelling nasobiliary tube (nasobiliary catheter) may be used to perform cholangiography, to drain a partially obstructed or an infected biliary system, or to infuse stone solvents or antibiotics, when primary stone extraction is not possible and if there is doubt as to whether spontaneous passage of calculi will occur. A catheter about twice the length of the endoscope is passed through the operating channel and into the biliary tree; retention within the biliary system is obtained either by forming a loop in the bile duct (Fig. 4.192) or using a self-retaining preformed catheter (Fig. 4.193). After positioning the catheter appropriately, the endoscope is carefully withdrawn, at the same time feeding the catheter down through the operating channel. The proximal end of the catheter, now emerging from the mouth, is re-routed via the nose after back-threading it into a nasogastric tube passed in through the nose and pulled out through the mouth (using a laryngoscope and Magill's forceps).



**Nasobiliary drainage (cont.)**

The nasobiliary cholangiogram shown in Fig. 4.194 was performed 7 days after that shown in Fig. 4.192; in the interim the stone had passed spontaneously.

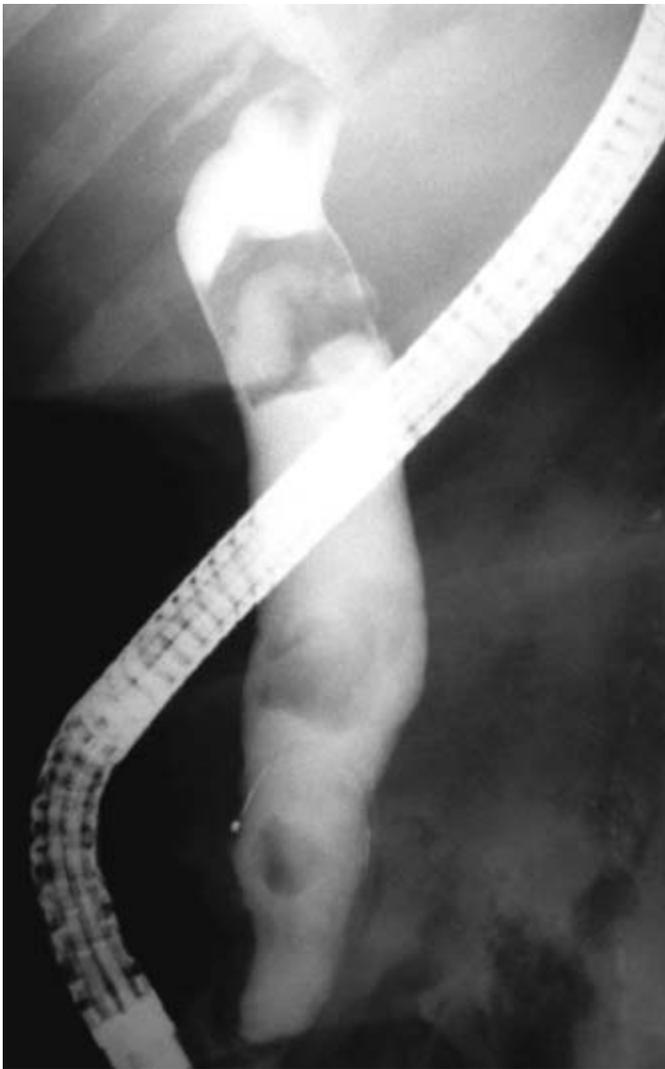


4.194

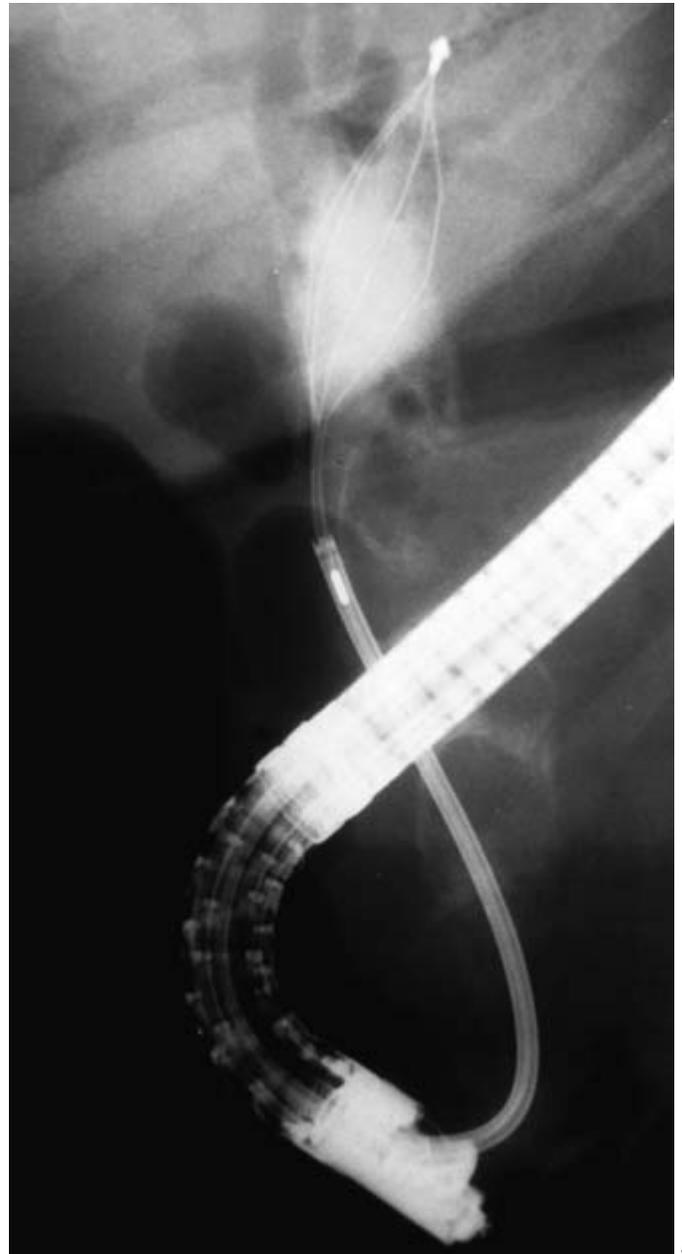
## Management of difficult or large gallstones

### Difficult or large gallstones

When duct clearance is not possible immediately after sphincterotomy and there is serious doubt whether the stone(s) will pass spontaneously (Fig. 4.195) it is preferable to insert a naso-biliary tube (as above) or a temporary stent (see below). This prevents stone impaction in the ampulla and allows biliary drainage so reducing the risk of cholangitis. If a naso-biliary tube was inserted, a check cholangiogram is performed after 24–48 h and if the stone(s) has passed the tube is removed. If stones remain further steps, as below, will be indicated. If a temporary stent was inserted there is less urgency and a check ERCP may be delayed for 2–4 weeks, and if the stones have passed the stent is removed. If stones remain consideration should be given to extending the sphincterotomy to allow removal of the stone(s) or lithotripsy (Fig. 4.196) may be preferred.



4.195



4.196

**Difficult or large gallstones (cont.)**

Occasionally when it is not feasible to clear the bile ducts of stones, long-term palliation with stents may be considered. Figure 4.197 shows two pigtail stents placed so as to prevent the large residual stones from becoming impacted in the lower portion of the bile duct. The endoscopic appearance of these stents from the duodenal aspect is shown in Fig. 4.198. Whilst this may be effective management in some cases the risk of cholangitis and its complications remains. It is therefore recommended that duct clearance should be achieved whenever possible.

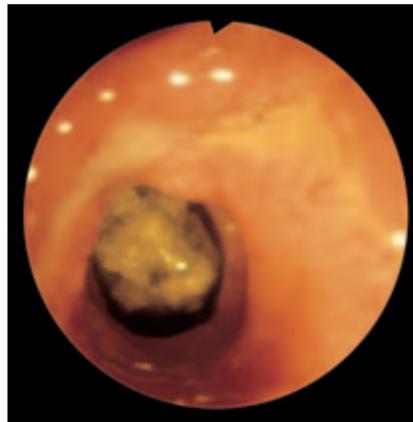
Cholesterol gallstones are soluble in organic solvents and may be dissolved in some cases by infusion of solvent through a surgically placed T-tube, or a naso-biliary or transhepatic catheter. The rate of dissolution tends to be slow and the results unpredictable; as a result the technique is not often used. Figures 4.199 and 4.200 show biliary debris softened and removable after infusion of mono-octanoin via a naso-biliary catheter. Note the clear view into the lower common bile duct; such clear access is necessary should lithotripsy using the 'mother-and-baby' endoscope be considered.



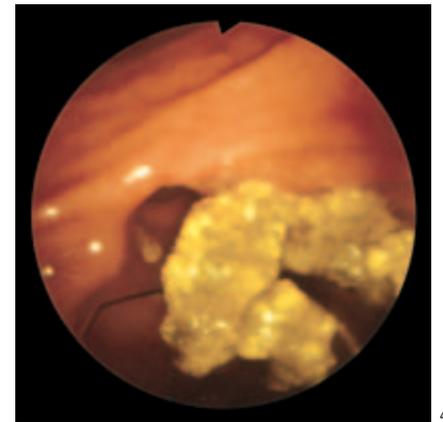
4.197



4.198



4.199



4.200

Despite the wide range of endoscopic possibilities, open surgical removal after exploration of the bile duct may be the best method for some patients with very large stones.

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## Internal biliary drainage: stent (endoprosthesis) placement

### Instrumentation

### Technique

### Endoscopic appearances

### Stent removal

### Instrumentation

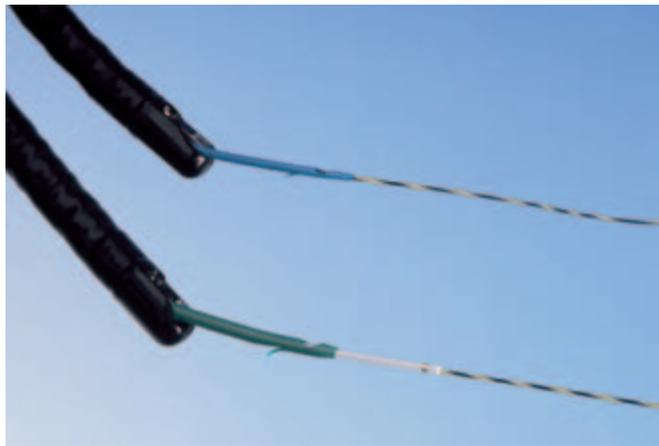
Internal endoscopic drainage is used for palliation of malignant obstruction of the bile duct, some benign biliary strictures and in other situations where there is unsatisfactory biliary drainage. For this purpose the endoscopist may use a stent.

As the word 'stent' is not now used outside medicine, readers may be interested in a little background gleaned from the *Compact Oxford English Dictionary* (2nd edn, 1991). 'A staple or hole to receive the end of a bar', 'a stake for stretching fishing nets upon in a river', 'tin mining rubble', and 'stiffening for a doublet' do not help much. 'To set up, erect (a tomb)' is, one hopes, premature. To confuse the issue further, it appears that a dentist called Stent devised a substance used for dental purposes. Hence 'all stents must be removed daily and cleaned; a pipe stem cleaner is effective in cleaning the tube'. More relevant would seem to be *stenton*, thought to be the same word, which in mining is 'a passage between two winning headways'. Finally we come to a more modern definition: 'a tube implanted temporarily in a vessel or part'. The most relevant paragraph contains a 1978 quotation: 'a soft Teflon tube called a stent is placed in the vessel to keep the lumen open and facilitate the suturing', and ends thus: 'At the time of the surgery, the physician lacerates the common bile duct and the liver. Both are successfully repaired but the common bile duct, of course, requires a stint [sic]'. A *stentmaster*, incidentally, is 'an official appointed to fix the amount of tax payable by the inhabitants of a town or parish', not necessarily an endoscopist.

### Instrumentation (cont.)

Standard sized duodenoscopes permit insertion of stents (biliary endoprotheses) of up to 8F (F indicates French gauge, the circumference in millimetres) and larger 'jumbo' duodenoscopes allow passage of 10F or 12F stents. For comparison of relative sizes Fig. 4.201 shows a standard duodenoscope with an 8F stent protruded, and a large channel duodenoscope with a 12F prosthesis passed over a guidewire and an intermediate (guiding) catheter which facilitates placement.

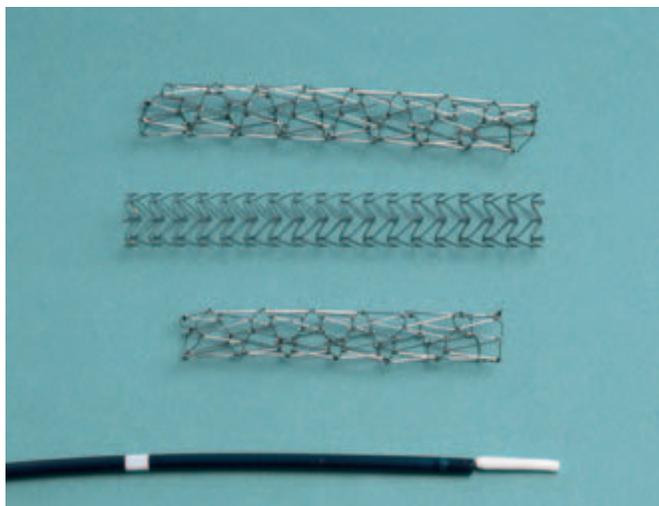
Stents of various sizes and designs are shown in Fig. 4.202. Plastic stents are self-retaining either by means of flaps cut into straight tubing or as the result of preformed 'pigtailed'. Expanding metal stents (Fig. 4.203) are retained by moulding themselves to fit the contours of the stricture. Metal stents, although much more expensive than plastic, stay patent for longer and may be preferred in some circumstances; this continues to be a matter of debate. It should be borne in mind that, metal stents once inserted, cannot be removed, whilst plastic stents can always be removed (see pp. 439–440).



4.201



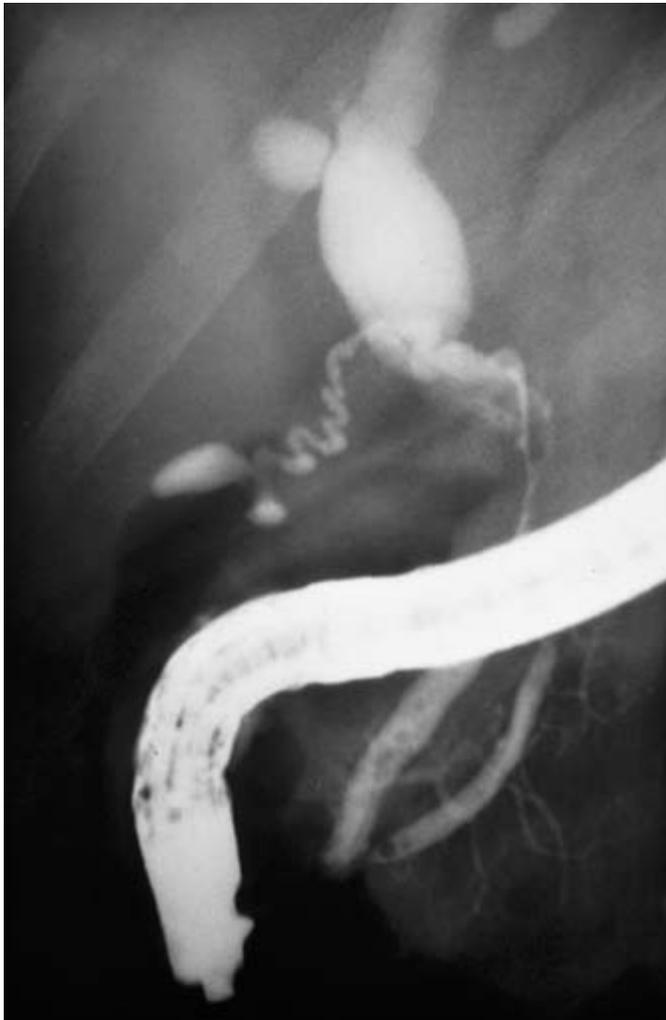
4.202



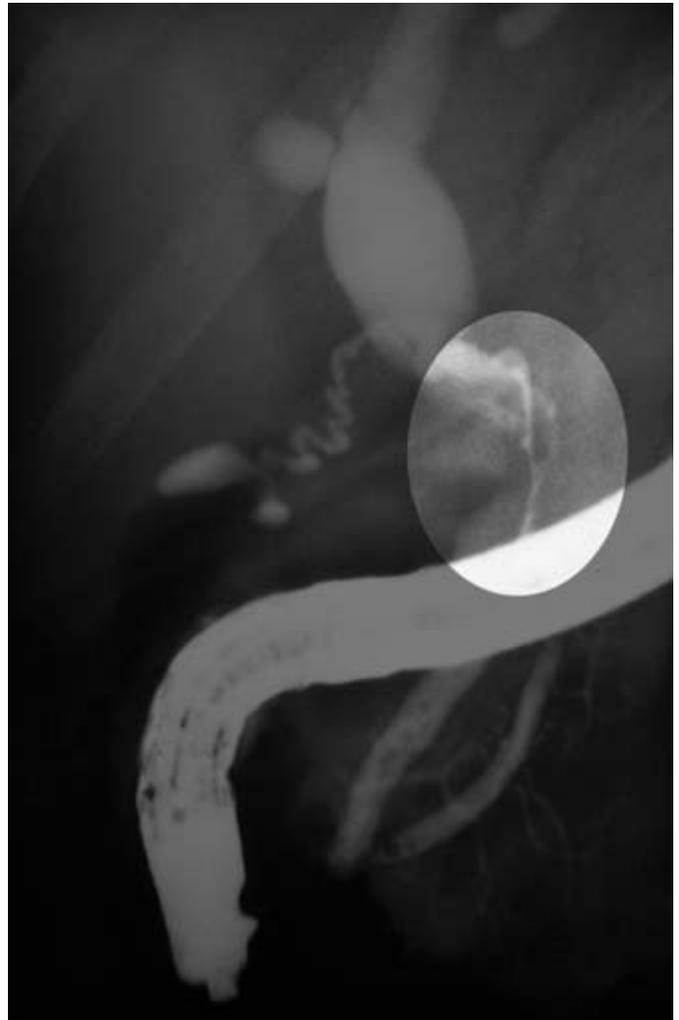
4.203

### Technique

Various techniques for the placement of stents have been described. All are broadly similar. Sometimes a small sphincterotomy is made, as described previously (pp. 419–421), to facilitate the procedure. This is not essential although it may aid drainage of pancreatic juice around a biliary stent which is eventually left protruding from the papilla. The introducing (delivery) system with its guidewire is passed up the bile duct to the stricture through which the wire is patiently negotiated. Figure 4.204 shows a tight stricture due to invasion by a carcinoma of the pancreas ('the double duct' sign, as described on pp. 379–380, is evident).

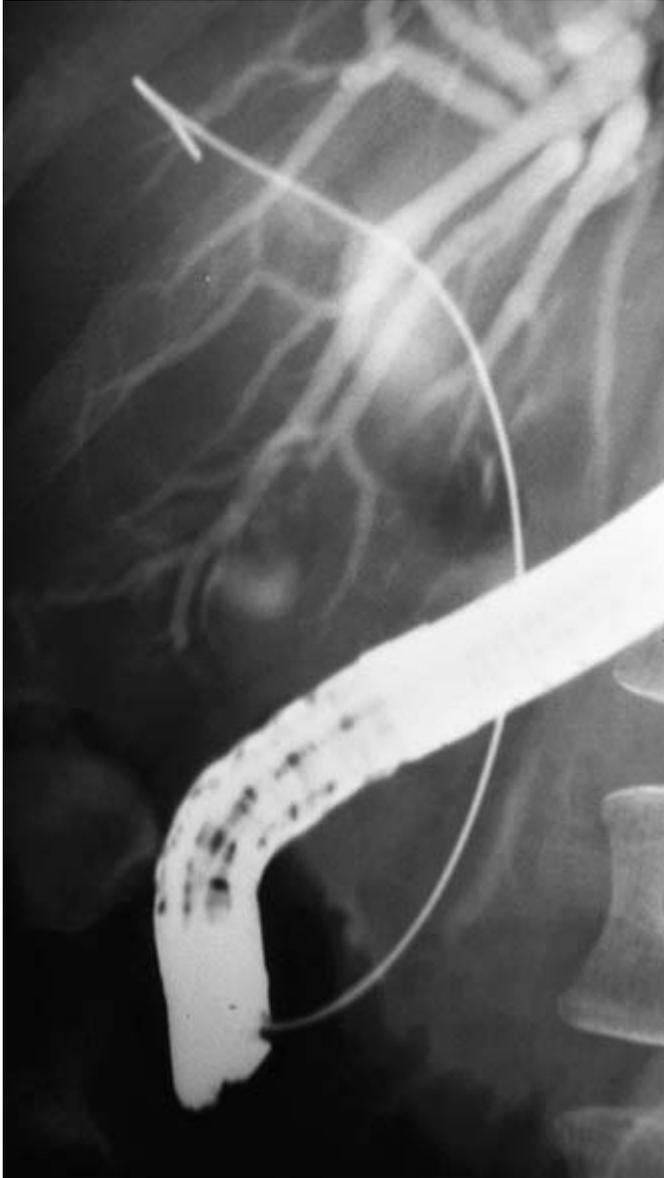


4.204



**Technique (cont.)**

Figure 4.205 shows the eventual passage of a guide wire. Note that the contrast material has drained from the lower common bile duct during the procedure. If it is difficult to pass a standard guide wire, changing to a hydrophilic ('slimy') wire (Fig. 4.206), may facilitate tracing the lumen of a stricture. When the wire is satisfactorily placed, brush cytology (pp. 356–357) may be performed (Fig. 4.207) to aid pathological diagnosis.



4.205



4.206



4.207

**Technique (cont.)**

After the collection of cytological material a stent of appropriate length is passed over the introducer and pushed into the bile duct by means of a pusher tube. Positioning is checked by frequent recourse to fluoroscopy (Fig. 4.208). When the stent is in position the wire is withdrawn. Figure 4.209 shows the radiographic appearance of the stent *in situ* at the completion of the procedure. Very tight strictures, benign or malignant, may require preliminary dilatation before a stent can be introduced (see pp. 440–441).



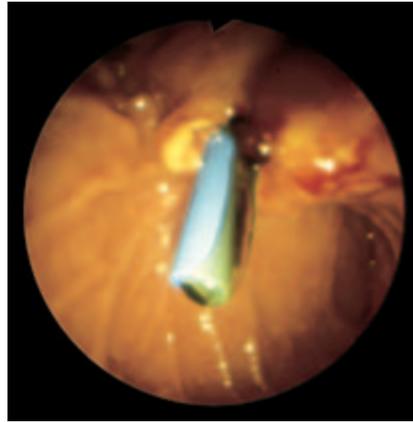
4.208



4.209

### Endoscopic appearances

In Fig. 4.210 a single 11.5F stent is shown protruding from the papilla, with bile draining. Small stents are inadequate to allow good bile drainage and are liable to early blockage but may sometimes be expedient when a large one cannot be inserted initially, or for short-term drainage. As an alternative to the use of a large stent two or more small tubes can be placed (Fig. 4.211).



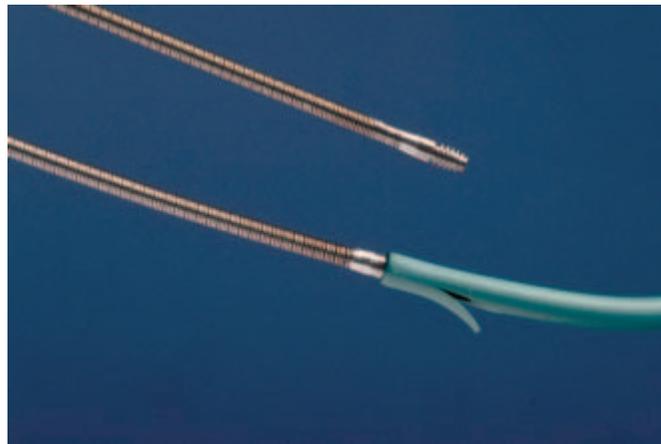
4.210



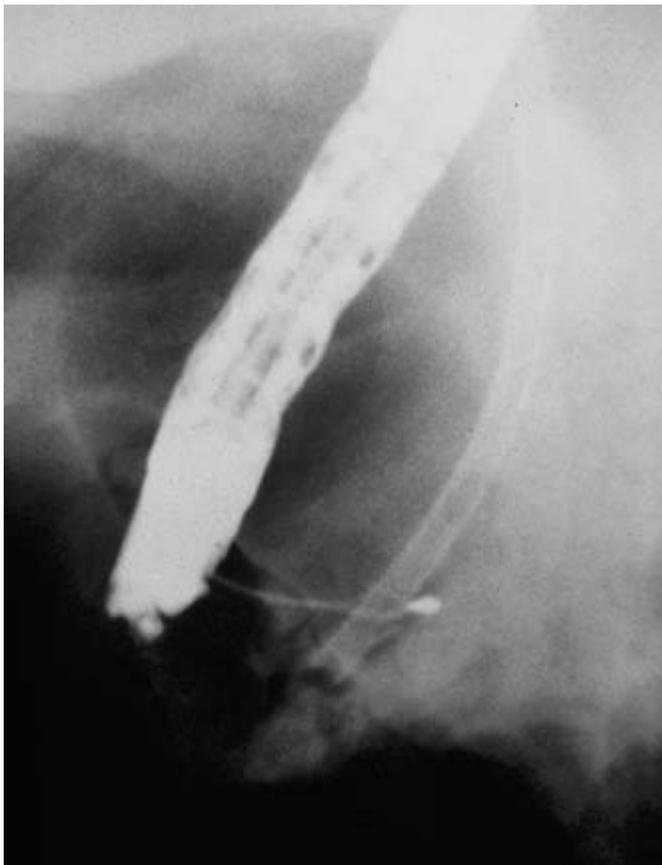
4.211

### Stent removal

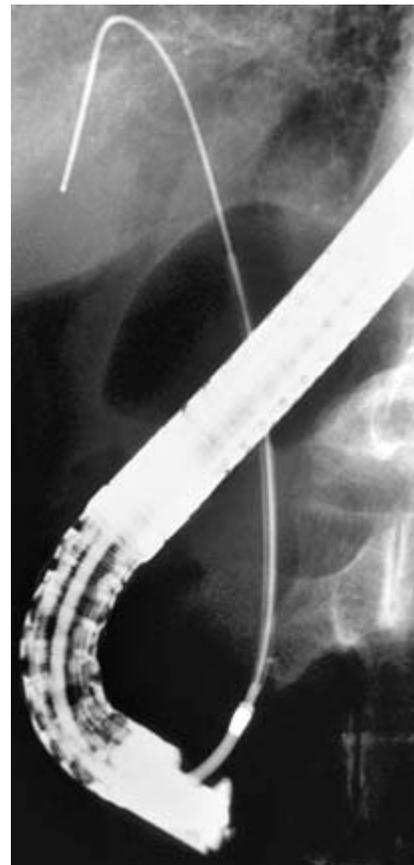
The simplest method is to grasp the protruding end with a snare or basket (Fig. 4.212) and withdraw it; retrieval of a narrow stent can be effected through the channel of a large endoscope. Alternatively a threaded removal device, as described by Soehendra (Fig. 4.213), can be screwed into place over a guide wire passed through the old stent (Fig. 4.214). With this method a guide wire should remain in the biliary system to allow a new stent to be placed.



4.213



4.212



4.214

### Stent removal (cont.)

Sometimes a balloon catheter passed into the lumen of the stent and inflated (Fig. 4.215) gives adequate grip for stent removal through the endoscope.



4.215

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## Balloon dilatation of strictures

### Instrumentation and technique

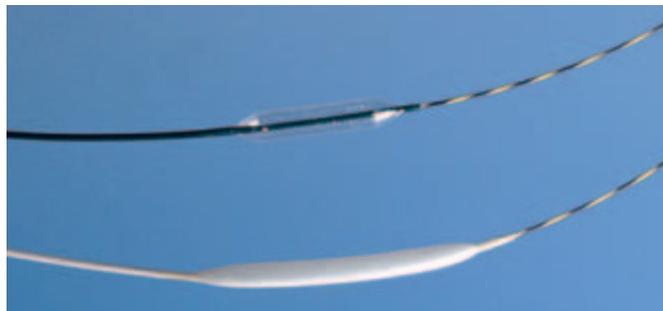
### Endoscopic and radiographic appearances

#### Instrumentation and technique

A balloon catheter passed over a guidewire, in a manner similar to that used for angioplasty, may be used successfully to dilate the papilla, or strictures within the pancreatic or biliary ducts. Such catheters are non-compliant (i.e. can only be distended to a predetermined size and shape) so preventing overdistension and reducing the risk of ductal damage. Various constructions and types of balloon material are available and those with a 'low profile' are favoured for ease of use. The material is either rigid (Gruntzig type) or low profile (Olbert

type) (Fig. 4.216). The balloon is filled with dilute radiographic contrast material so that positioning can be monitored and the waisting at the site of the stricture can be seen to disappear on inflation of the balloon. Use

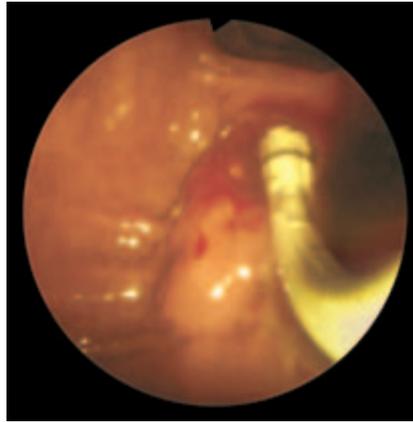
of fluid, which is non-compressible, ensures that adequate force is applied to the narrowed segment for efficient dilatation. Biliary dilatation may be very painful so adequate analgesia should be given.



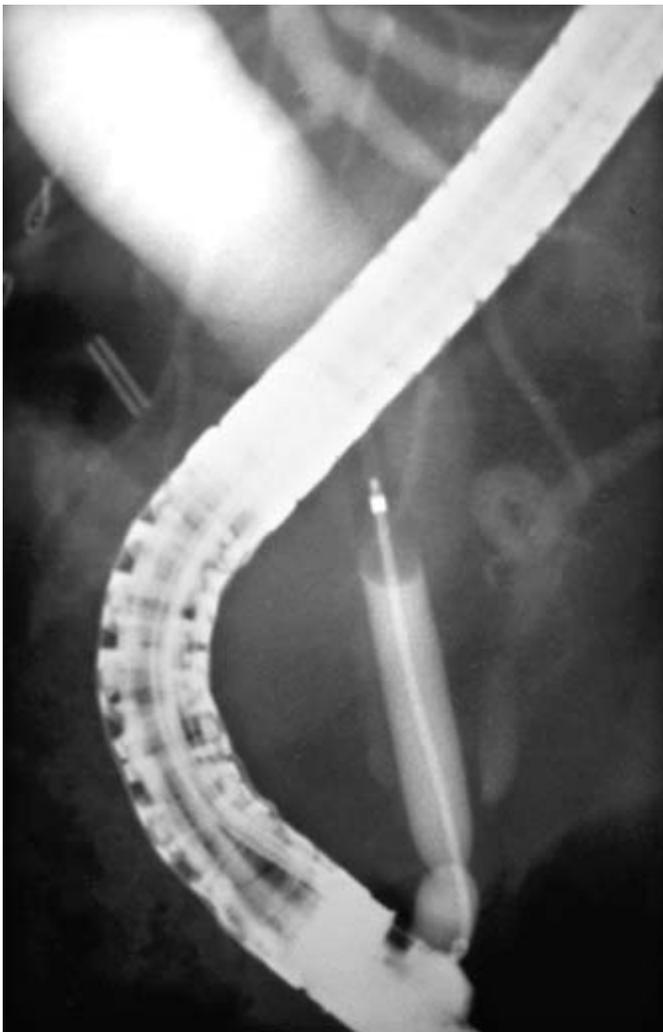
4.216

**Endoscopic and radiographic appearances**

Figure 4.217 shows a balloon catheter protruding from the papilla during dilatation of a low stricture of the common bile duct. Figure 4.218a shows the radiographic appearance of a balloon catheter placed across a bile duct stricture and Figure 4.218b the dilatation procedure.



4.217



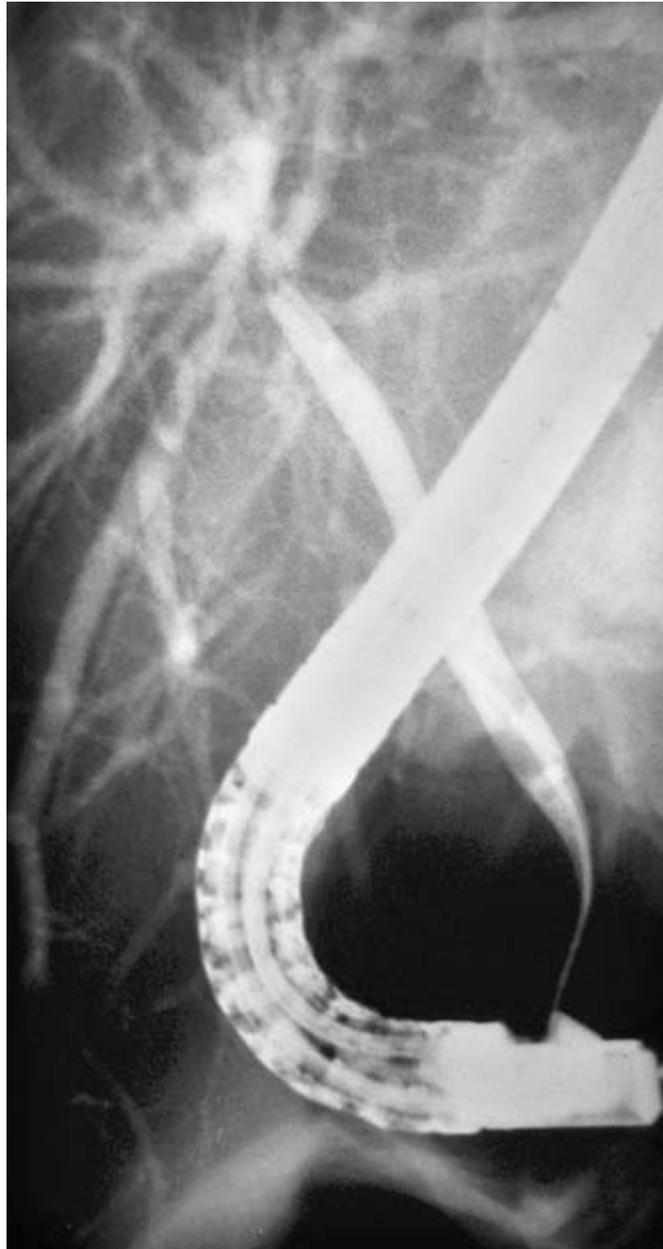
4.218a



4.218b

**Endoscopic and radiographic appearances (*cont.*)**

Low strictures are often easy to dilate but high strictures are technically more challenging as the relatively rigid catheter assembly can be difficult to position. Figure 4.219 shows dilatation of a long stricture in a patient with sclerosing cholangitis. Collaboration with a radiologist using a percutaneous approach employing the 'combined procedure' (pp. 444–445) may be successful.



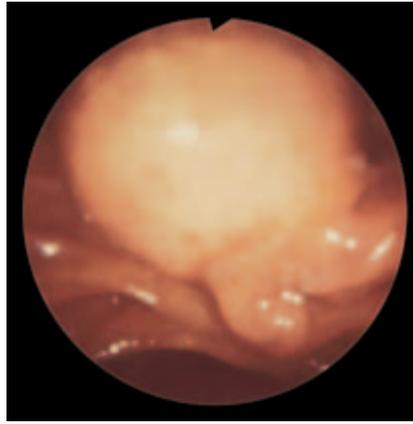
4.219

## Suprapapillary bile duct puncture

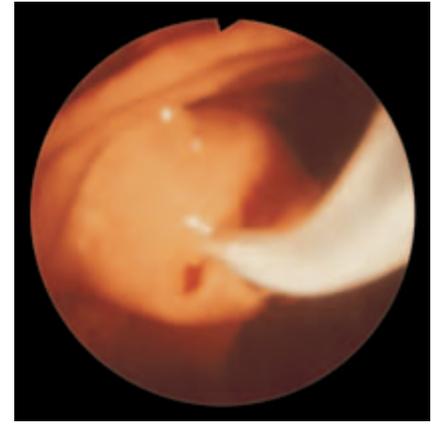
### Suprapapillary bile duct puncture

Rarely, when there is jaundice due to complete obstruction at the papilla and conventional sphincterotomy has failed, an alternative method of biliary drainage can be used. Figure 4.220 shows distension of the intramural portion of the common bile duct due to a lesion at the papilla. It was impossible to pass a papillotome and incision of the papilla with a needle-knife did not open the bile duct. The suprapapillary portion of the bile duct was punctured with the needle-knife to permit placement of the papillotome through the newly created orifice (Fig. 4.221).

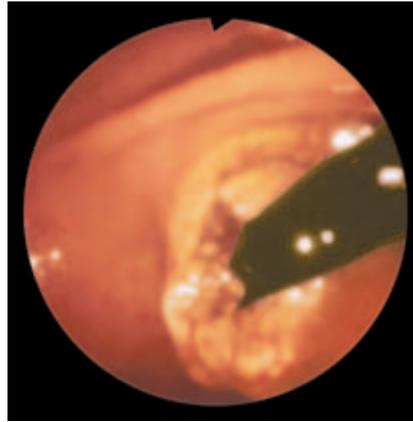
Incision into the bile duct wall caused a gush of bile (Fig. 4.222) and subsequently jaundice resolved. Biopsy of the tissue exposed by incision yielded adenocarcinoma. The tumour was later resected surgically. A similar technique may be employed if a stone impacted at the papilla causes distension of the intramural bile duct.



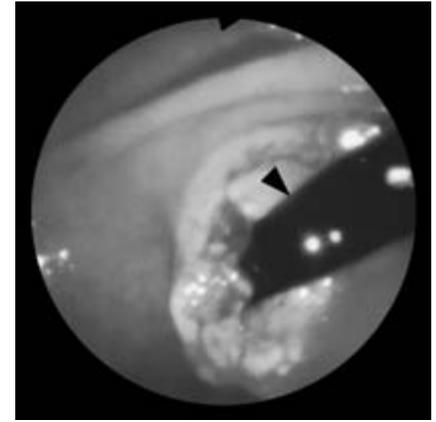
4.220



4.221



4.222



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## Combined or 'rendezvous' procedure

### Combined or 'rendezvous' procedure

When endoscopic access to the biliary system, or a segment of it, has not been achieved it is sometimes appropriate to use this technique in collaboration with an interventional radiologist. By the percutaneous transhepatic approach a catheter is placed in a suitable intrahepatic duct. A 4-m wire is passed through the catheter via the biliary system, down the bile duct and out through the papilla. Using a snare, the endoscopist picks up the wire, pulling the end of it through the endoscope and out of the biopsy port. The wire is held firmly in place by the radiologist and the endoscopist's assistant so that endoscopic catheters can be passed over the wire in the usual manner (Fig. 4.223) to allow sphincterotomy, balloon dilatation (Fig. 4.224) or stent placement (Fig. 4.225).



4.223



4.224

**Combined or 'rendezvous' procedure  
(cont.)**

After the procedure has been completed, so long as internal biliary drainage is good, the percutaneous catheter may be removed.

The combined procedure has two advantages. Firstly, percutaneous manipulation is often more successful in passing strictures and secondly direct puncture may provide access to segments of the biliary system which the endoscopist is unable to enter.

The most frequent indications for the combined procedure are when the papilla is hidden in a diverticulum, when a stricture cannot be passed from below or a segment of the biliary system must be drained but is not accessible endoscopically.



4.225

## Therapy of bile duct injuries

### Therapy of bile duct injuries

Injuries to the bile ducts or cystic duct sustained at surgical cholecystectomy (either laparoscopic or 'open') may sometimes be treated endoscopically. Small leaks from the bile duct (Fig. 4.226) or cystic duct stump (Fig. 4.227) may close satisfactorily after placement of a temporary stent across the sphincter of Oddi and perhaps also across the leak (Fig. 4.228). Figure 4.229, taken 6 weeks later, shows closure of the leak seen previously in Fig. 4.226.



4.226



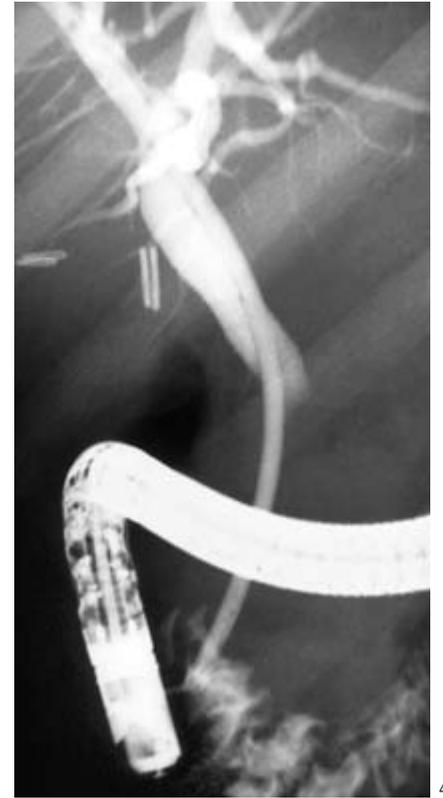
4.227



Therapy of bile duct injuries (*cont.*)



4.228



4.229

Post-cholecystectomy bile duct strictures may respond well to balloon dilatation provided it is performed early after surgery before dense fibrosis occurs. Late strictures are usually difficult to dilate and recurrence is common.

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## Endoscopic therapy of pancreatic disease

### Pancreatic sphincterotomy at the major papilla

#### Sphincterotomy at the minor papilla

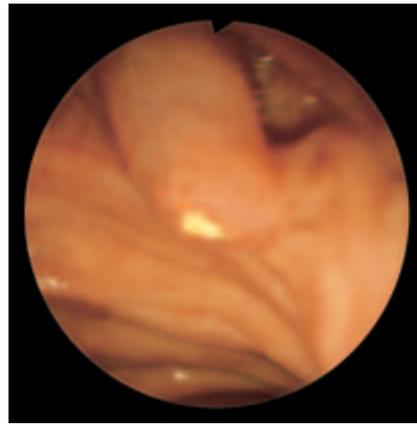
#### Dilatation of pancreatic ductal strictures

#### Endoscopic drainage of pancreatic pseudocysts and abscesses

Indications for therapeutic intervention in pancreatic diseases are less well defined than those for biliary diseases and the techniques are less well developed and evaluated. Detailed consideration is beyond the scope of this work.

### Pancreatic sphincterotomy at the major papilla

Small calculi in the main pancreatic duct (Figs 4.230 and 4.65) in the head of the gland may sometimes be removed by balloon or basket following selective pancreatic sphincterotomy. Before attempting to open the pancreatic duct it is usual to perform a biliary sphincterotomy so that the opening of the pancreatic duct is exposed directly thus increasing precision of incision into the pancreas. Larger stones or those in the pancreatic body (Fig. 4.80) may be treated by extracorporeal shock wave lithotripsy (ESWL) but calcification in the parenchyma is not amenable to treatment.



4.230

Pancreatic sphincterotomy may sometimes be performed for recurrent pancreatitis when it is caused by papillary stenosis, papillary dysfunction or inflammation of the sphincter of Oddi. These diagnoses should be based on the results of pressure studies of the sphincter of Oddi, a technique restricted to specialist centres.

### Sphincterotomy at the minor papilla

Accessory papillary sphincterotomy in pancreas divisum may be indicated for pancreatitis involving the dorsal portion of the gland. This difficult technique is not commonly performed.

### Dilatation of pancreatic ductal strictures

Strictures of the main pancreatic duct due to scarring following pancreatitis (Fig. 4.231) or trauma may be dilated with a balloon catheter or by placement of a stent (Fig. 4.232) as for strictures of the bile duct. As recurrence is common the stent is usually left *in situ* for several weeks to hold open the stricture in the hope that a more permanent result will be achieved.



4.231



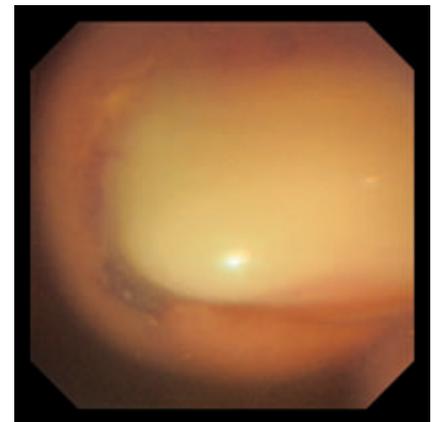
4.232

### Endoscopic drainage of pancreatic pseudocysts and abscesses

Sometimes a pancreatic pseudocyst or abscess discharges spontaneously into the stomach or duodenum. To imitate this process it may be possible to drain a mature pancreatic pseudocyst into the stomach. After precise localisation (preferably by endoscopic ultrasound Fig. 6.91) to ensure that the cyst is in direct contact with the gastric wall, the stomach and the cyst are punctured with a needle knife (Fig. 4.233) to allow discharge of the contained fluid (Fig. 4.234). A stent is commonly inserted through the incision to further aid drainage.



4.233



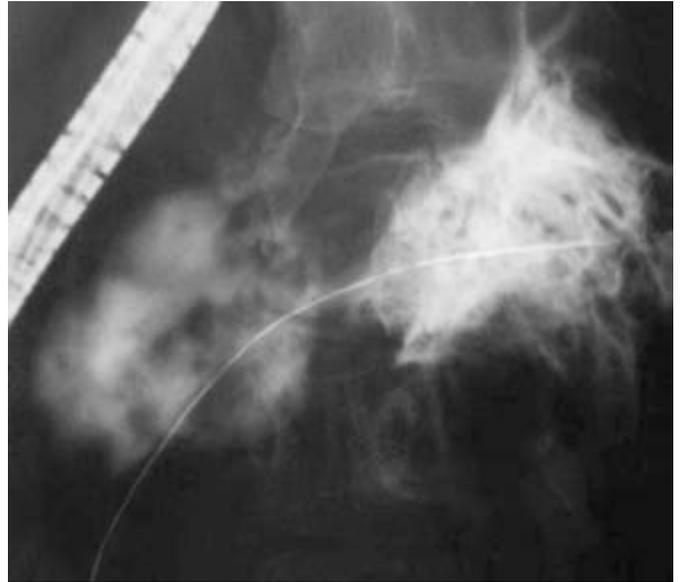
4.234

**Endoscopic drainage of pancreatic pseudocysts and abscesses (cont.)**

An alternative route of drainage is into the duodenum via the papilla. Figure 4.235 shows a stent draining from a cyst in the head of the pancreas into the duodenum. Less often it may be possible to pass a guide wire along the pancreatic duct into a cyst in the body or tail of the gland (Fig. 4.236) so allowing drainage by means of a long stent (Fig. 4.237). After resolution of the cysts the stents are removed.



4.235



4.236



4.237

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## Complications

ERCP has several specific complications in addition to those of upper gastrointestinal endoscopy.

Minor trauma to the papilla and adjacent mucosa commonly results from probing with the cannula. Mucosal dissection by the tip of the cannula may lead to injection of contrast material into the tissues. Neither of these occurrences has any serious consequences.

Mild hyperamylasaemia with or without abdominal pain is common following pancreatic duct opacification especially when acinar filling has occurred. Usually this is of little clinical consequence but occasionally severe acute pancreatitis can occur, this being more likely when the pancreas is overfilled by forceful injection, or when the patient has suffered previous attacks of acute pancreatitis. Patients with papillary stenosis or spasm are prone to develop acute pancreatitis especially after pancreatic pressure studies (manometry) have been performed; extreme caution is required.

Infection introduced by ERCP is a very real risk in the presence of pancreatic cysts or an obstructed biliary system and is potentially fatal. Thus, meticulous disinfection of endoscopes, sterilisation of reusable accessories or use of disposables is mandatory to minimise this hazard. Should a pancreatic pseudocyst be opacified the possibility of infection must always be considered and appropriate measures taken: antibiotic therapy and perhaps drainage of the cyst either percutaneously or surgically.

Obstructed bile ducts and particularly those which contain calculi are prone to infection with the risk of suppurative cholangitis and septicaemia. To reduce this risk prophylactic broad-spectrum antibiotics are given whenever ERCP is performed in the presence of impaired biliary drainage.

Insertion of a temporary stent or a nasobiliary drain is a wise additional precaution if at the end of a procedure there is doubt about free drainage.

Sphincterotomy has a serious complication rate of 5–10% and a mortality of 0.5–1%, but these figures compare favourably with those associated with the surgical procedures which it replaces. The advantages of endoscopic sphincterotomy over surgery are seen to be even greater when it is recognised that endoscopic sphincterotomy is commonly performed on elderly or debilitated patients considered unfit for operation. The most frequent and serious problem is haemorrhage; perforation, infection, basket impaction and pancreatitis constitute the remainder. Balloon dilatation of the papilla is less likely than sphincterotomy to cause bleeding but a local haematoma or marked papillary oedema may be produced. This may cause temporary biliary obstruction, pancreatitis or an attack of cholangitis.

The newer therapeutic techniques all have their own complications which will not be detailed here.

**Instrumentation and techniques 453****Normal appearances 457****Abnormal appearances 458**

Until recently, the small intestine was the only region of the digestive tract which could not be examined endoscopically. This was due in part to the anatomy of the small bowel whose multiple unsupported loops precluded deep intubation using conventional instruments and techniques, and also to differences amongst clinicians and experts in endoscopy as to the perceived need for endoscopic examination of the jejunum and ileum. These hindrances have gradually been replaced by enthusiasm as the value of this technique has been demonstrated.

The first evaluations of prototype small intestinal endoscopes were published in the mid-1970s. Today special techniques using purpose designed instruments allow high quality examination of the whole small intestine. It is possible that endoscopy of the small bowel may, like oesophago-gastro-duodenoscopy (OGD) and colonoscopy, gradually replace radiological methods of examination and will provide a major route for therapeutic intervention in the small intestine.

This chapter reviews current practice in the field of enteroscopy and illustrates normal endoscopic appearances of the small bowel and diseases of the jejunum and ileum. The upper duodenum is within easy reach of standard upper gastrointestinal endoscopes: normal and abnormal duodenal appearances are dealt with in Chapter 2.

**Instrumentation and techniques****Sonde enteroscopy 454****Push enteroscopy 455**

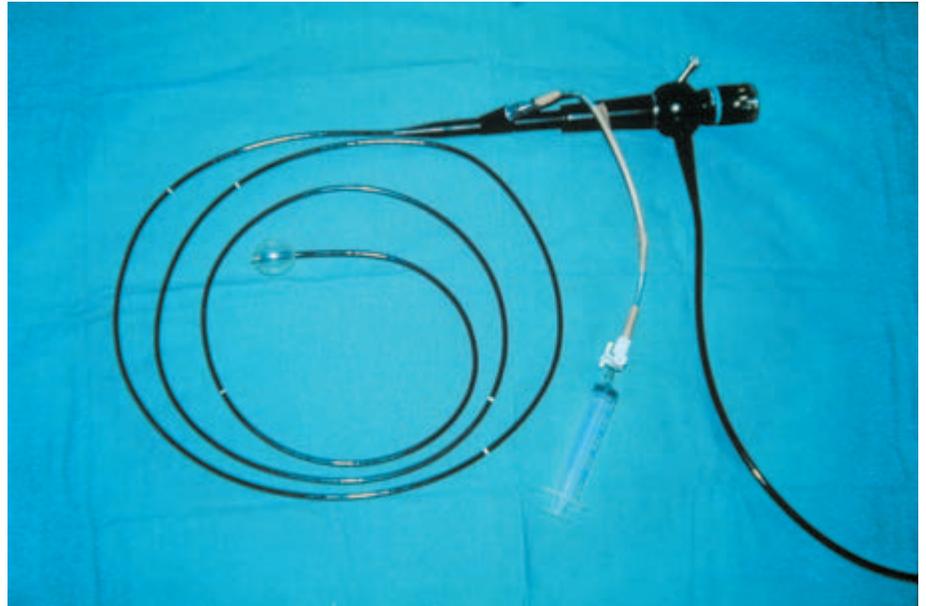
There are two techniques of endoscopic examination of the small bowel: sonde and push enteroscopy. These depend upon purpose designed instruments which enable examination of different areas of the small intestine. Facilities for simultaneous radiological screening are essential for safe and successful sonde enteroscopy and can be helpful to the less experienced endoscopist during push enteroscopy. Preparation and sedation for enteroscopy are essentially the same as for OGD, but as the procedure may be prolonged and sometimes uncomfortable, higher doses of sedation and the use of analgesics may be required.

## Sonde enteroscopy

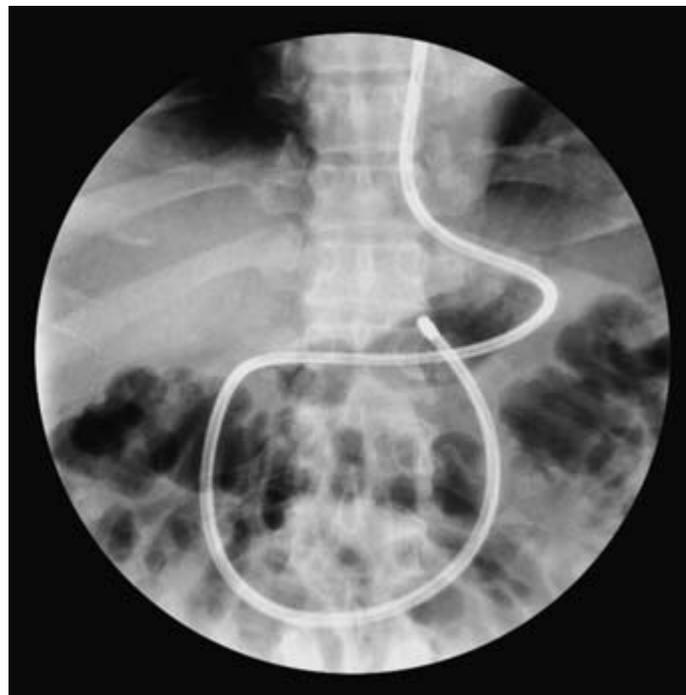
The sonde enteroscope is highly flexible, 5 mm in diameter and 2.7 m long. It is designed to negotiate the convoluted loops of the distal small bowel. It has two channels, one for insufflation of air or water from the tip and the second for inflation of a balloon covering the distal 3 cm of the instrument. Figure 5.1 shows a sonde enteroscope with a syringe inflating the balloon. Sonde enteroscopy was the first method used in clinical practice and was pivotal in the development of small bowel endoscopy.

Sonde enteroscopy differs from conventional gastrointestinal endoscopic techniques in three respects. Firstly, the enteroscope is not advanced by the endoscopist but relies on traction by peristalsis to allow deep intubation of the ileum. Secondly, the instrument is introduced through the nose and is advanced in stages by the patient or an assistant. Finally, examination of the bowel occurs only during withdrawal of the instrument with the balloon in the deflated state. A plain abdominal radiograph (Fig. 5.2) shows a sonde enteroscope at the ligament of Treitz during insertion. Figure 5.3 shows the tip of the instrument having passed to the terminal ileum while Fig. 5.4 reveals the sonde in mid-intestine during withdrawal.

A major problem with the sonde technique is the long time taken to achieve deep intubation of the small bowel. This period can be shortened by helping the enteroscope through the pylorus using a 'piggyback' technique, where a string on the distal tip of the enteroscope is grasped by biopsy forceps from a second conventional endoscope passed through the mouth and alongside the sonde. The tip of the enteroscope is manoeuvred into the duodenum using the biopsy forceps before inflation of



5.1



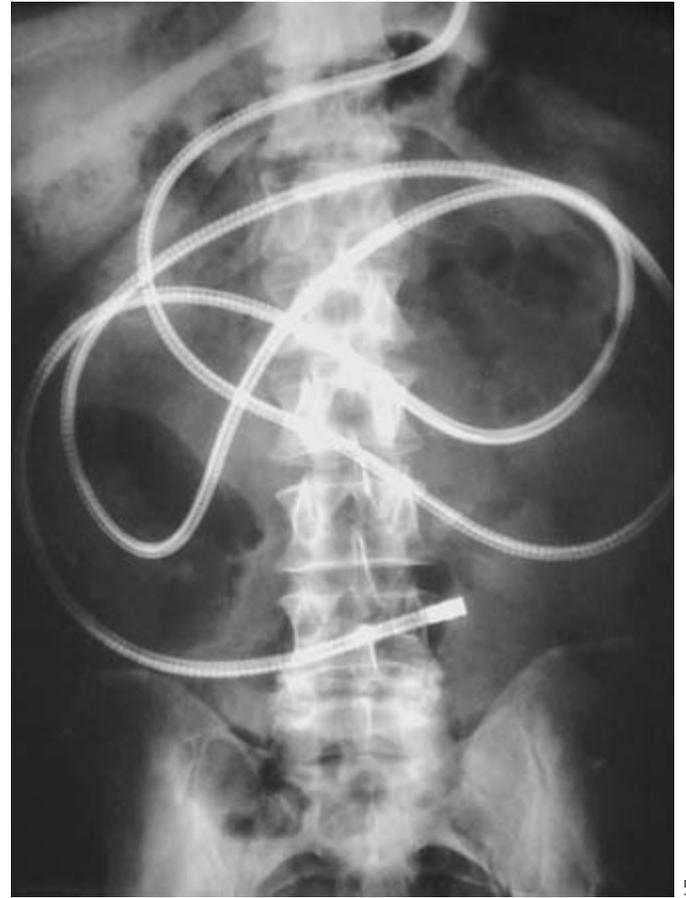
5.2

the distal balloon. The second endoscope is then withdrawn. Even with this modified technique the whole procedure may take 6–8 h. Sonde enteroscopy is therefore normally reserved for occasions where examination of the ileum is felt to be

essential for patient management. As there is no biopsy channel, it is not possible to obtain specimens for pathological examination, and the absence of a steering mechanism for tip deflection may result in incomplete mucosal inspection.



5.3



5.4

### Push enteroscopy

The push enteroscopy is a much more familiar instrument having a biopsy/therapeutic channel and a fully steerable tip. Figure 5.5 shows a typical video push enteroscope 2.5 m in length, with a specially flexible distal section to facilitate passage around bends in unsupported loops of small bowel. Successful push enteroscopy depends on the ability to propel the instrument into the small bowel by direct pressure. A potential problem is looping in the stomach. To overcome this a stiffening overtube is backloaded onto the shaft (Fig. 5.6) before intubation. The overtube has two sections, a stiffer proximal portion and a flexible Gore-Tex<sup>®</sup> distal section, designed to be positioned through the pylorus in the duodenal loop. Deployment of the overtube was originally thought to necessitate the use of fluoroscopy, but with increasing experience this is now often dispensed with.

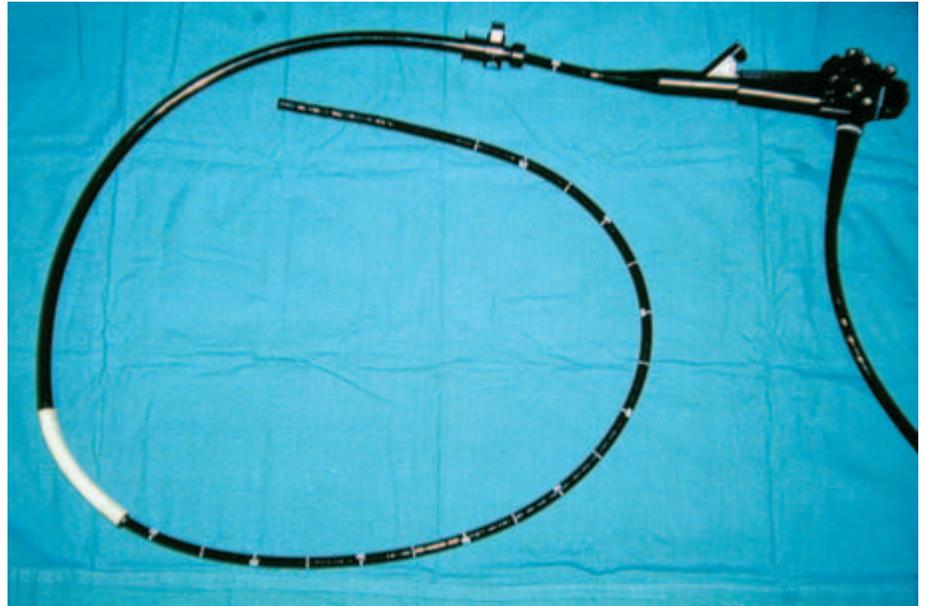


5.5

**Push enteroscopy (cont.)**

When the enteroscope has been advanced to the ligament of Treitz the loop in the stomach is removed by pulling back, which allows the overtube to be safely advanced over the straightened enteroscope, through the stomach and into position, with the flexible part in the duodenum. This facilitates direct transmission of pressure to further advance the tip of the enteroscope. Figure 5.7 taken during fluoroscopy shows the distal portion of the overtube in the stomach before entering the duodenum.

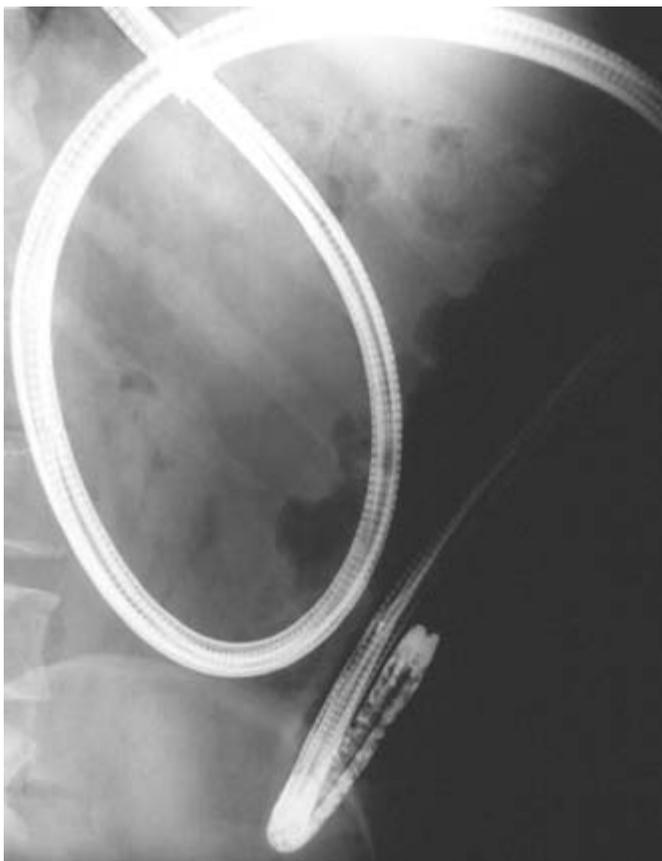
Whilst sonde enteroscopy allows examination of the entire small intestine the push enteroscope can only reach the distal jejunum or at best the proximal ileum. On the other hand the image quality of narrow sonde fibre-enteroscopes is poor when compared with that obtained with the latest push video enteroscopes as is clearly evident in the images in this chapter.



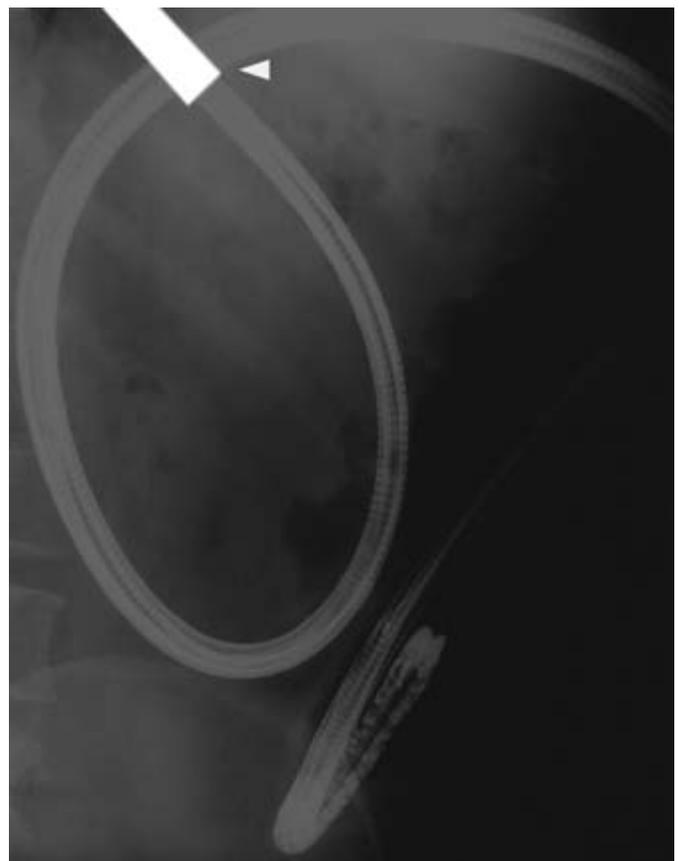
5.6

As appearances in the duodenum and the remainder of the small intestine are similar there is inevitably some overlap between this chapter and Chapter 2. We have minimized duplication both with respect to

endoscopic and histological images. It is hoped that what follows will be seen as an appropriate introduction and that endoscopists will more fully appreciate the potential of diagnostic and therapeutic enteroscopy.



5.7

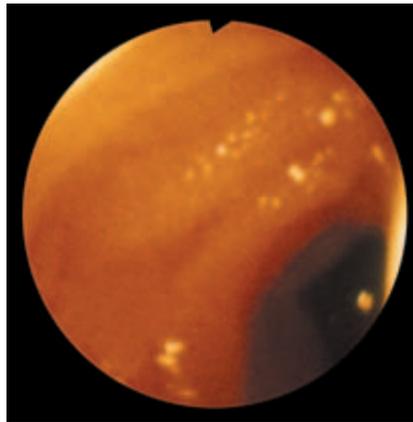


## Normal appearances

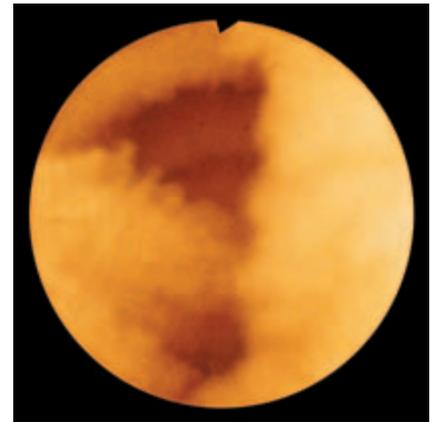
The small bowel is characterized by prominent circular folds and multiple villi which are not normally seen when the bowel is distended with air. Figures 5.8 and 5.9 show the jejunum using the sonde enteroscope. In Fig. 5.8 the bowel is distended by air and the villi are collapsed whilst following instillation of water via the enteroscope they are clearly identified as multiple finger-like extensions at the luminal surface (Fig. 5.9). Similar appearances may be seen in the terminal ileum during colonoscopy (Fig. 3.44).

Clear landmarks are not found in the small bowel but with experience the enteroscopist will recognize a reduction in circular folds and number of villi as the more distal small bowel is examined. Figure 5.10 shows a view of the ileum (sonde enteroscope) illustrating this subtle change. Note that circumferential views are rarely obtained with the sonde enteroscope due to the limited angle of view provided by the currently available lenses.

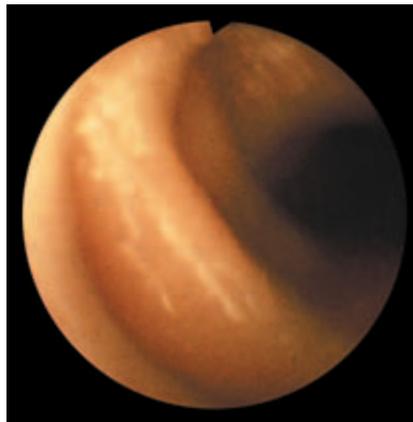
With the use of external palpation the tip can be displaced to allow more complete examination. The view of the jejunum (Fig. 5.11) using the push enteroscope is far superior and with tip deflection circumferential examination is possible. Figure 5.12 shows underwater views of the villi in the jejunum following water instillation.



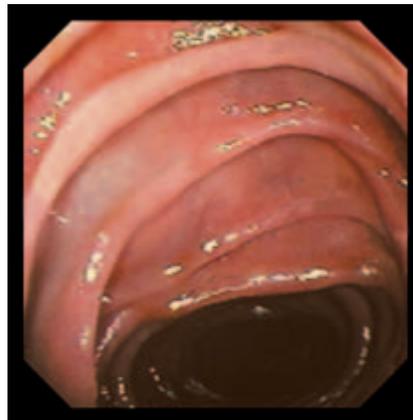
5.8



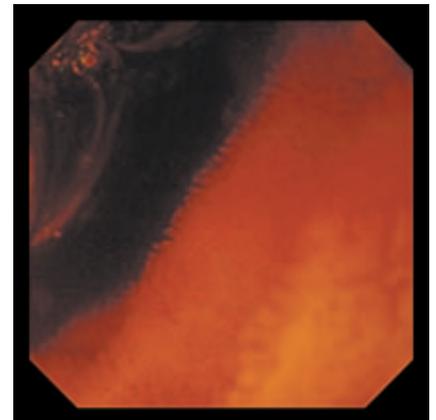
5.9



5.10



5.11



5.12

## Abnormal appearances

**Bleeding and vascular lesions 458**

**Benign and malignant tumours 459**

**Miscellaneous lesions 462**

### Bleeding and vascular lesions

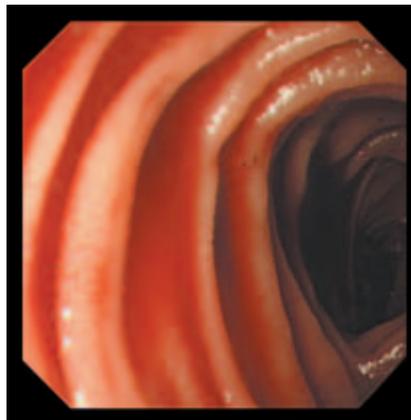
#### Arteriovenous malformations (AVMs)

#### Miscellaneous bleeding causes

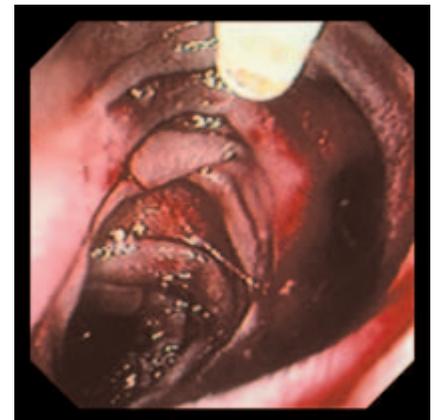
In 5–10% of all patients presenting with significant gastrointestinal haemorrhage, a source of blood loss will not be identified during OGD and colonoscopy. Before the introduction of enteroscopy these patients were assumed to have small bowel bleeding, but investigation frequently failed to localize the source. Enteroscopy is invaluable in patients who might otherwise consume substantial resources by repeated hospital admission for blood transfusions or re-investigation. Initial reports suggest that it is possible to find the cause of suspected small bowel bleeding in up to three-quarters of patients using push enteroscopy.

#### Arteriovenous malformations (AVMs)

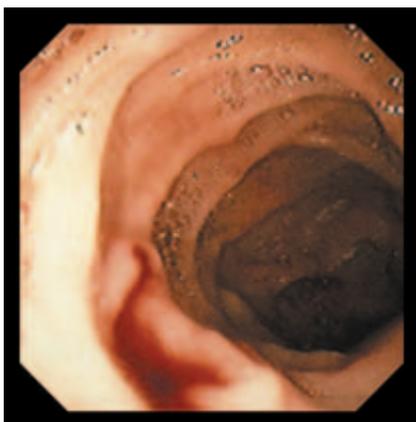
The most common lesions identified by enteroscopy in such patients are AVMs. Figure 5.13 shows blood in the proximal jejunum from a bleeding AVM and Fig. 5.14 a heater probe passed via the biopsy channel of the enteroscope prior to washing and ablation of such a lesion. Figures 5.15–5.17 illustrate a bleeding AVM before and after successful ablation by heater probe. Note the increase in bleeding before complete haemostasis: this is a



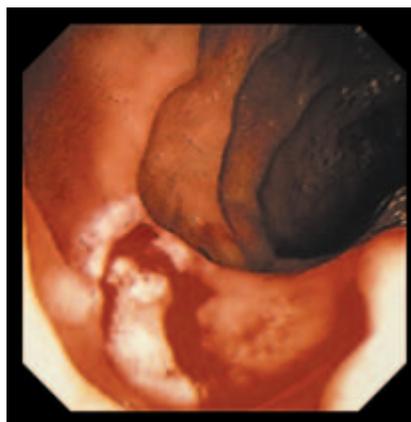
5.13



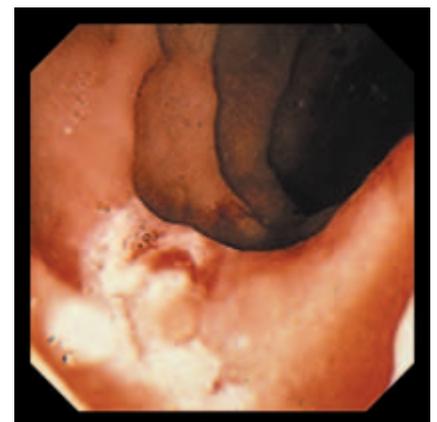
5.14



5.15



5.16



5.17

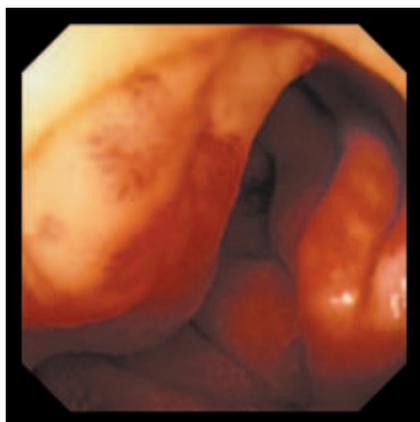
### Arteriovenous malformations (AVMs) (cont.)

frequent occurrence suggesting larger submucosal feeding vessels underlying small mucosal lesions. It is suggested that the heater probe is used at maximum 10 J setting for a total of only three applications per lesion because of the risk of perforation of the thin walled bowel; higher settings may be used elsewhere, for example in the stomach.

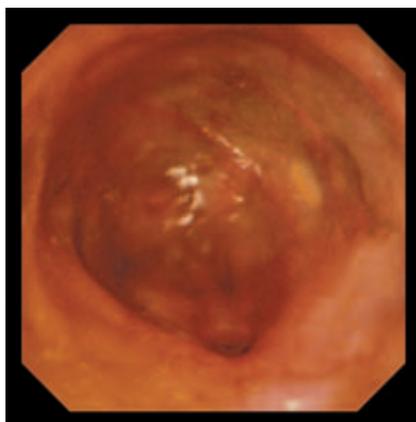
### Miscellaneous bleeding causes

Other causes of bleeding include portal hypertensive jejunopathy (Fig. 5.18) seen in a patient with advanced liver disease, when dilated superficial vessels and congested mucosa are easily identified.

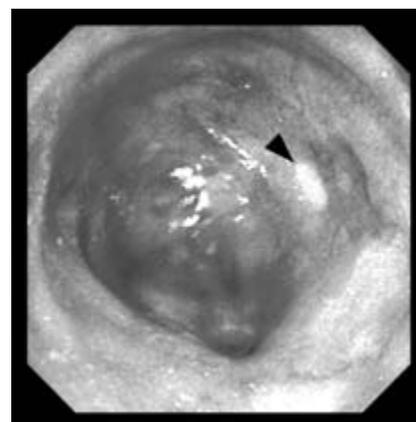
An ulcer in a Meckel's diverticulum (Fig. 5.19) may cause serious blood loss and should be considered as a rare cause of small bowel bleeding in younger patients.



5.18



5.19



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## Benign and malignant tumours

### Hereditary polyposis syndromes

#### Peutz–Jeghers syndrome

#### Adenoma

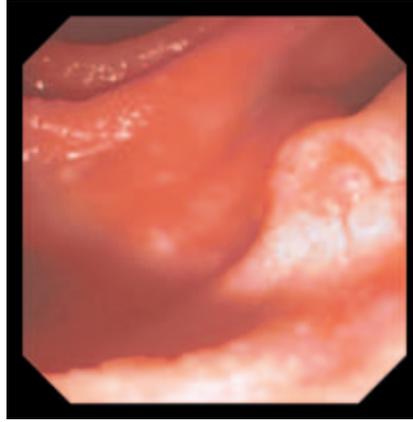
#### Gastrointestinal stromal tumours (GISTs)

#### Secondary deposits of tumours

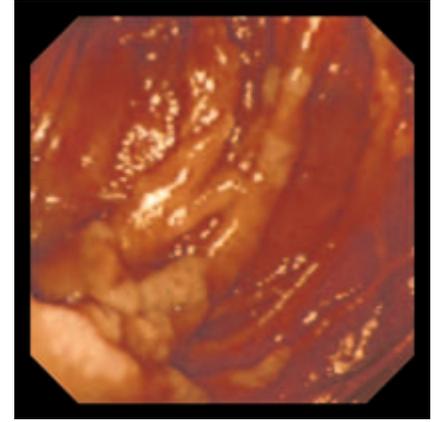
In younger patients, small bowel tumours are a major cause of small intestinal blood loss. If a patient under 50 years of age who is not receiving non-steroidal anti-inflammatory drugs (NSAIDs) presents with bleeding presumed to be from the small intestine, examination by enteroscopy, possibly combined with laparoscopy, is indicated.

### Hereditary polyposis syndromes

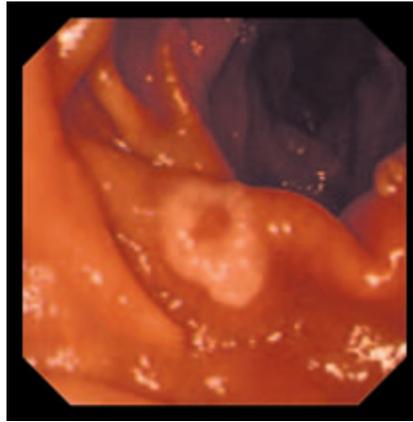
Enteroscopy now plays a leading role in the investigation, screening and treatment of patients with hereditary polyposis syndromes. Patients with familial adenomatous polyposis (FAP) are particularly prone to develop an adenoma of the papilla of Vater (Fig. 5.20); malignant change is common. Figures 5.21 and 5.22 illustrate multiple non-ulcerated and ulcerated adenomas in the jejunum beyond the reach of the type of endoscope used in routine upper gastrointestinal examinations. Biopsies revealed the presence of moderate dysplasia. The finding of small bowel adenomas is almost universal in such patients and may suggest an indication for enteroscopic surveillance of patients with FAP after colectomy has removed the risk of colon cancer. However, complicating carcinoma occurs more commonly in the duodenum than in the remainder of the small bowel.



5.20



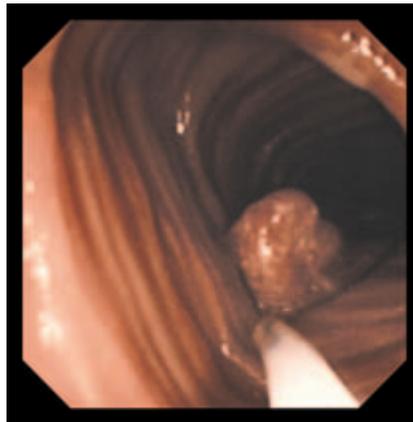
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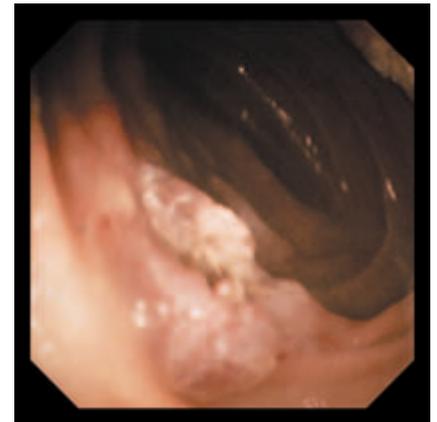
5.22

### Peutz–Jeghers syndrome

In Peutz–Jeghers syndrome enteroscopic polypectomy has been shown to relieve abdominal pain and to reduce the need for surgical intervention. Figure 5.23 shows the diathermy snare *in situ* prior to excision and retrieval of the polyp. Great care is needed to avoid deep excision and possible perforation of the jejunum: note the shallowness of excision in Fig. 5.24.



5.23



5.24

### Adenoma

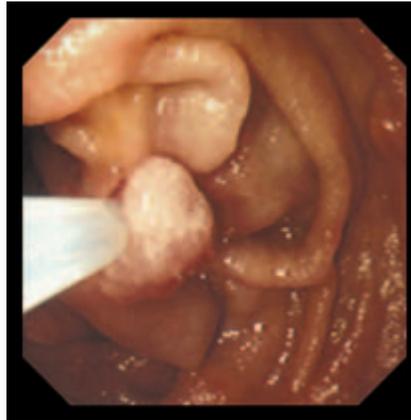
Figures 5.25–5.28 illustrate removal of a sessile jejunal adenoma using the technique of mucosal preinjection. Small adenomas can present with vigorous bleeding (Fig. 5.29). Endoscopic polypectomy is covered more fully in Chapter 3.



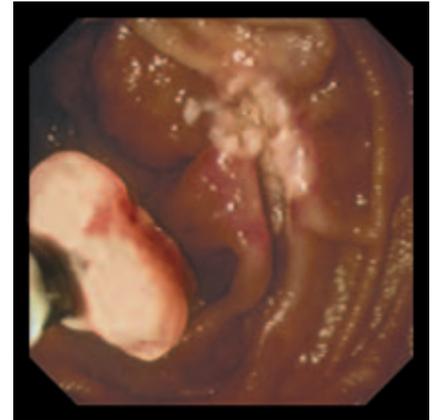
5.25



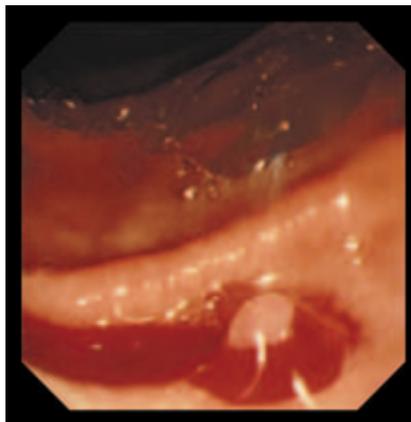
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5.27



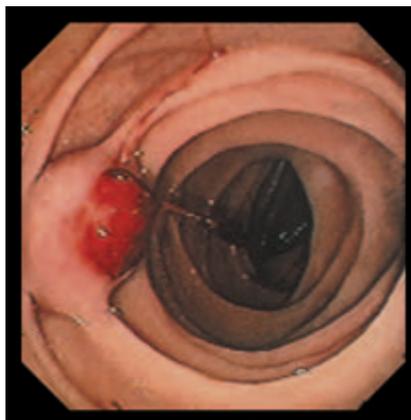
5.28



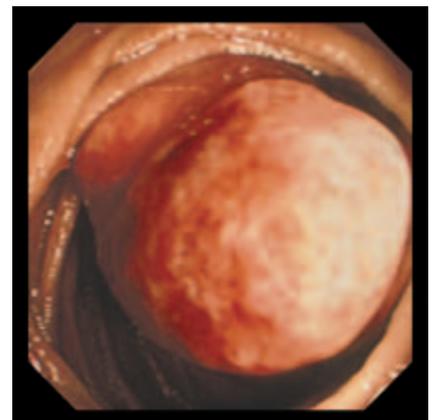
5.29

### Gastrointestinal stromal tumours (GISTs)

A bleeding GIST (Fig. 5.30), in the event malignant, was identified by enteroscopy and excised at simultaneous laparoscopy. A larger GIST is shown in Fig. 5.31. The current classification of such tumours formerly known by various names such as leiomyoma and leiomyosarcoma is discussed in greater detail in Chapter 2.



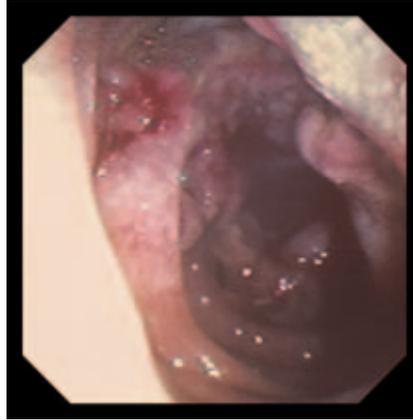
5.30



5.31

### Secondary deposits of tumours

Such deposits in the small bowel are uncommon and may arise from a variety of primary tumours including malignant melanoma. Figure 5.32 illustrates such a metastasis in an elderly patient from whom a renal cell carcinoma had been resected 7 years previously.



5.32

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### Miscellaneous lesions

Crohn's disease

Coeliac disease

Non-steroidal anti-inflammatory drug (NSAID) enteropathy

Graft vs. host disease

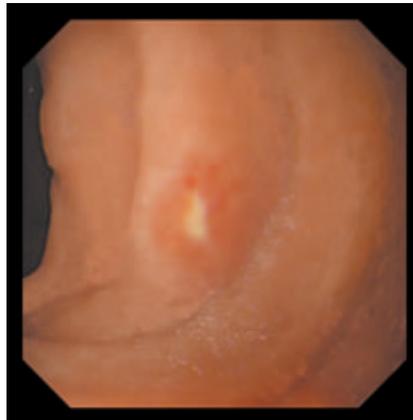
Ischaemic enteropathy

Cytomegalovirus infection

Abnormal mucosal folds

### Crohn's disease

Early lesions of Crohn's disease appear as aphthous ulcers (Fig. 5.33) as elsewhere in the gastrointestinal tract. More severe ulceration of the proximal jejunum is shown in Fig. 5.34.



5.33



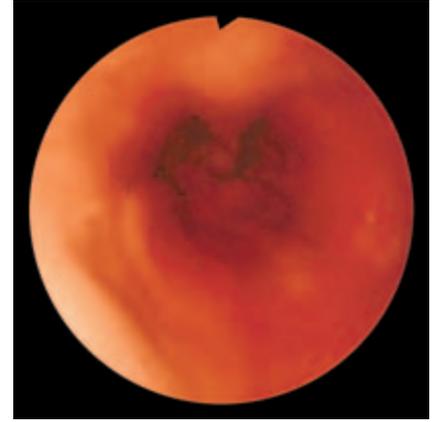
5.34

### Crohn's disease (cont.)

In other patients with Crohn's disease stenosis of the lumen can be observed (Fig. 5.35) and the diagnosis confirmed by biopsy. With sonde examination such lesions may prevent further progress of the enteroscope and limit mucosal inspection (Fig. 5.36).



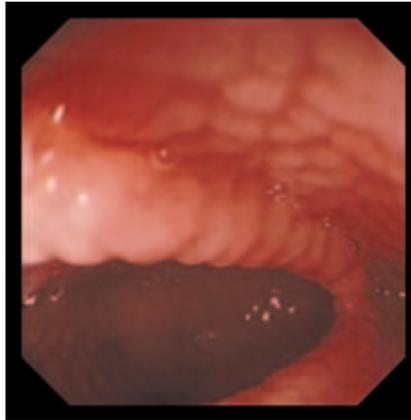
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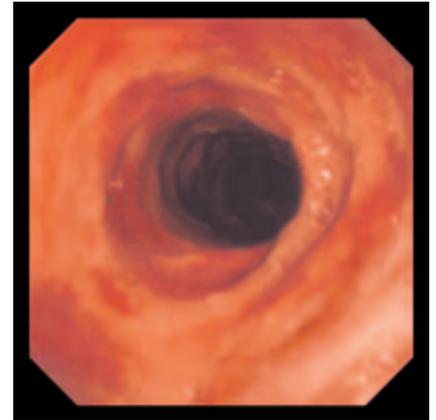
5.36

### Coeliac disease

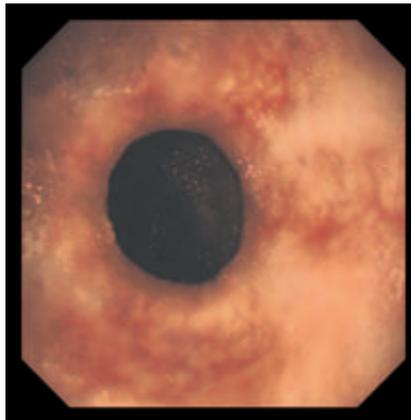
Whilst coeliac disease may cause characteristic endoscopic appearances in the duodenum (Figs 2.551–2.554) these findings can easily be overlooked. Endoscopic views of the jejunum in a patient with coeliac disease reveal a striking mosaic appearance with obvious scalloping of the folds (Fig. 5.37). Enteroscopy may also detect complications of coeliac disease. Figure 5.38 shows severe ulcerative jejunitis, Fig. 5.39 a benign stricture (jejunal diaphragm) and Fig. 5.40 a focal lymphoma (see also pp. 121–123). Enteroscopy offers the possibility of long-term surveillance of patients with ulcerative jejunitis. (Coeliac disease is also discussed on pp. 158–159.)



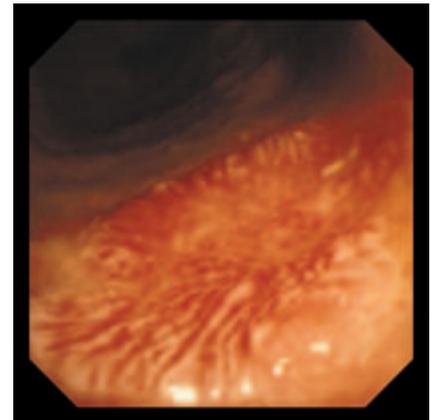
5.37



5.38



5.39



5.40

### Non-steroidal anti-inflammatory drug (NSAID) enteropathy

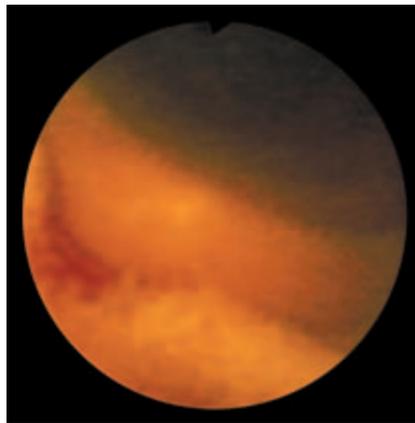
NSAIDs are the cause of a wide variety of lesions throughout the gastrointestinal tract. The best known are gastric and duodenal erosions and ulcers which are illustrated in Chapter 2. The frequency of NSAID-associated lesions in the small intestine is less well appreciated yet some studies suggest that abnormalities may be more prevalent here than in the stomach or duodenum. Mucosal damage has been reported to occur in approximately two thirds of patients on these drugs and is a major cause of occult and overt blood loss. Sonde and push enteroscopy have greatly expanded knowledge of NSAID enteropathy.

**Non-steroidal anti-inflammatory drug (NSAID) enteropathy (cont.)**

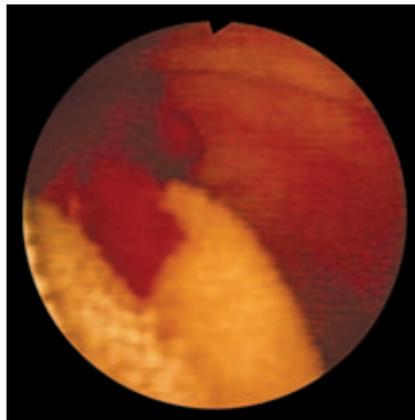
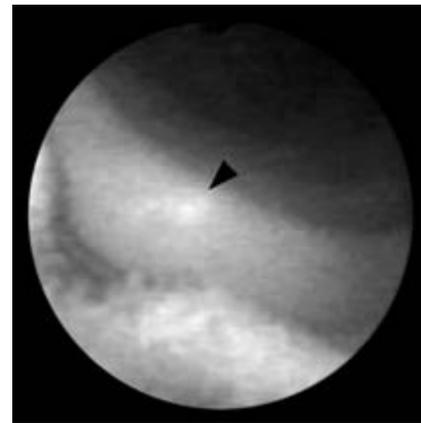
Figure 5.41 shows NSAID ulcers detected by sonde enteroscopy. Figure 5.42 reveals an underwater view of a bleeding NSAID ulcer during sonde examination: note the use of fluid to enhance luminal distension.

Ulceration of the jejunum occurs predominantly on the crests the circular folds (Fig. 5.43). With severe ulceration, adhesions and fibrosis may occur, perhaps leading to stenosis of the bowel lumen (Fig. 5.44). Such a process may be the cause of the well known 'diaphragm disease'. These diaphragms (Fig. 5.45) are typically only 3 mm in thickness and are easily missed radiographically due to the difficulty in distinguishing them from normal. They are characteristic of NSAID enteropathy.

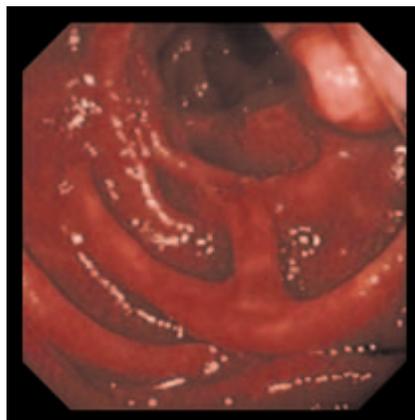
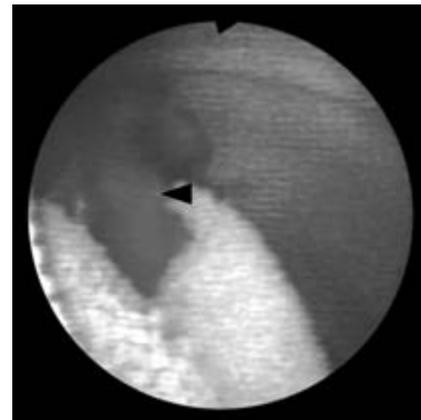
Push enteroscopy allows the use of biopsy (Fig. 5.46) which has permitted identification *inter alia* of novel pathological aspects of NSAID enteropathy.



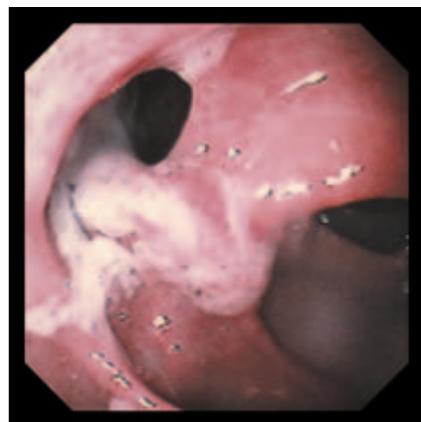
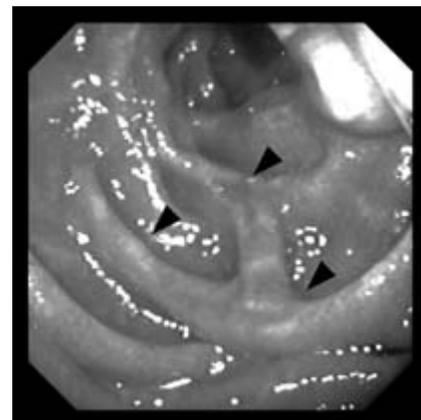
5.41



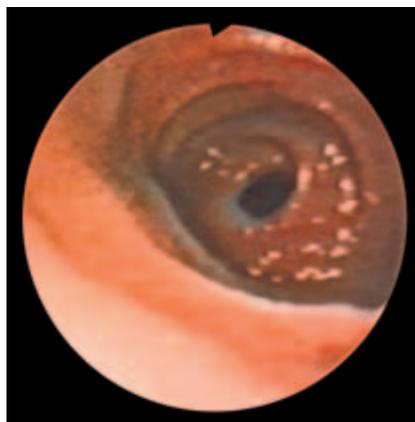
5.42



5.43



5.44



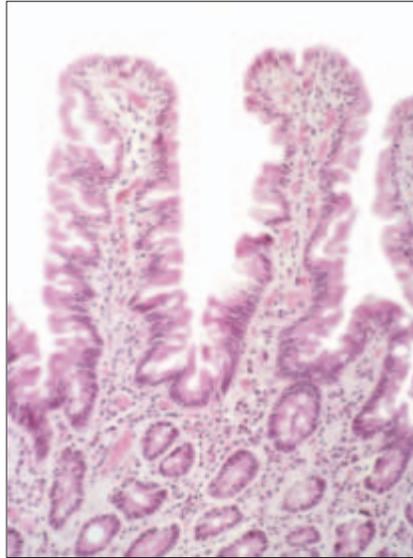
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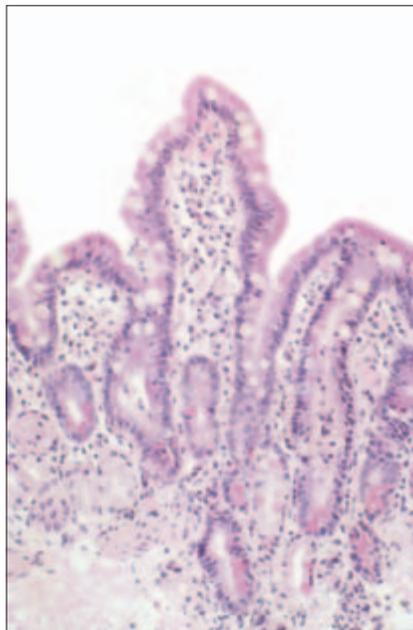
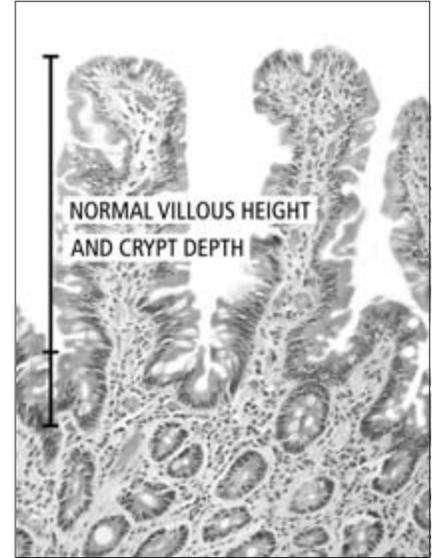
5.46

**Non-steroidal anti-inflammatory drug (NSAID) enteropathy (cont.)**

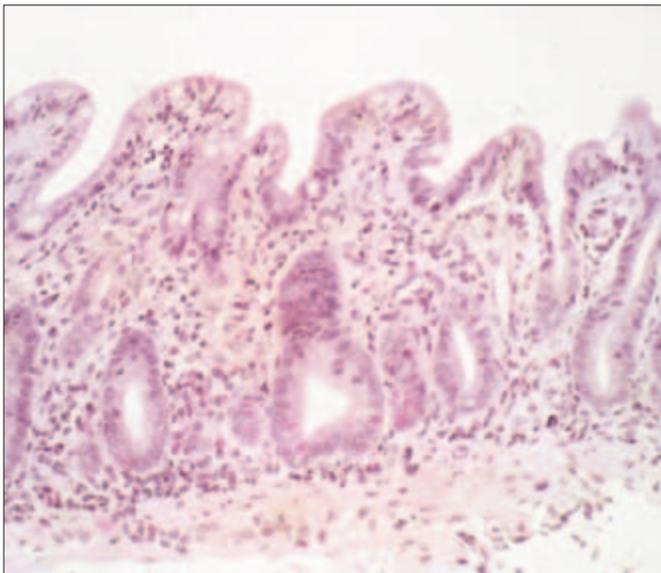
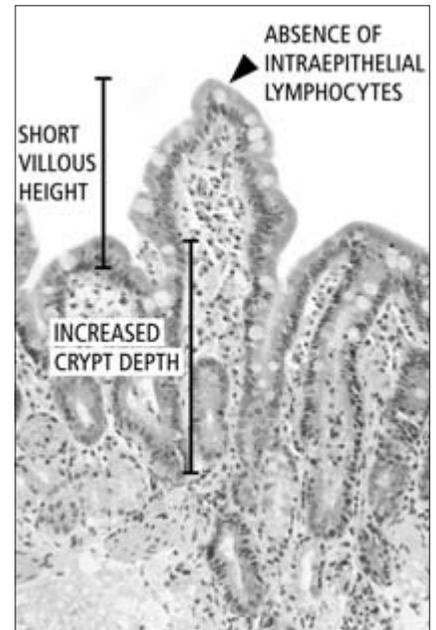
Figure 5.47 shows the normal histological appearance of small bowel. Figure 5.48 illustrates the appearance found in many patients on NSAID therapy. There is obvious reduction in the villus/crypt ratio, and villous blunting without evidence of increased intraepithelial lymphocytes (such as may be seen in coeliac disease); in Fig. 5.49 there is villous atrophy.



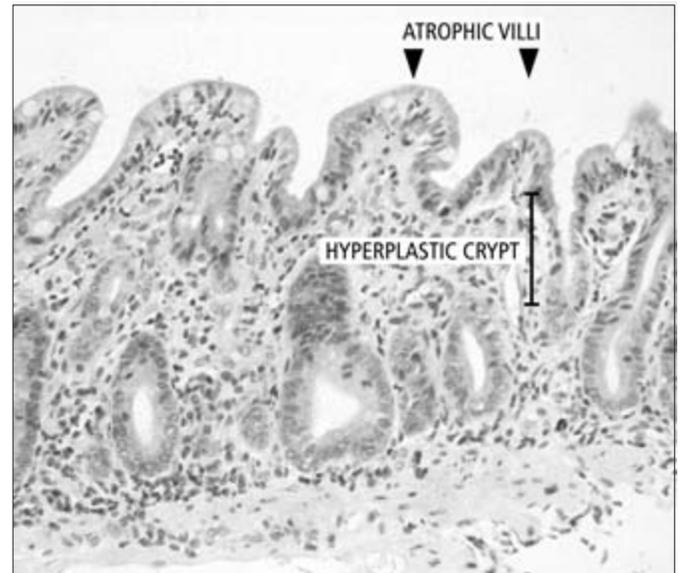
5.47



5.48



5.49

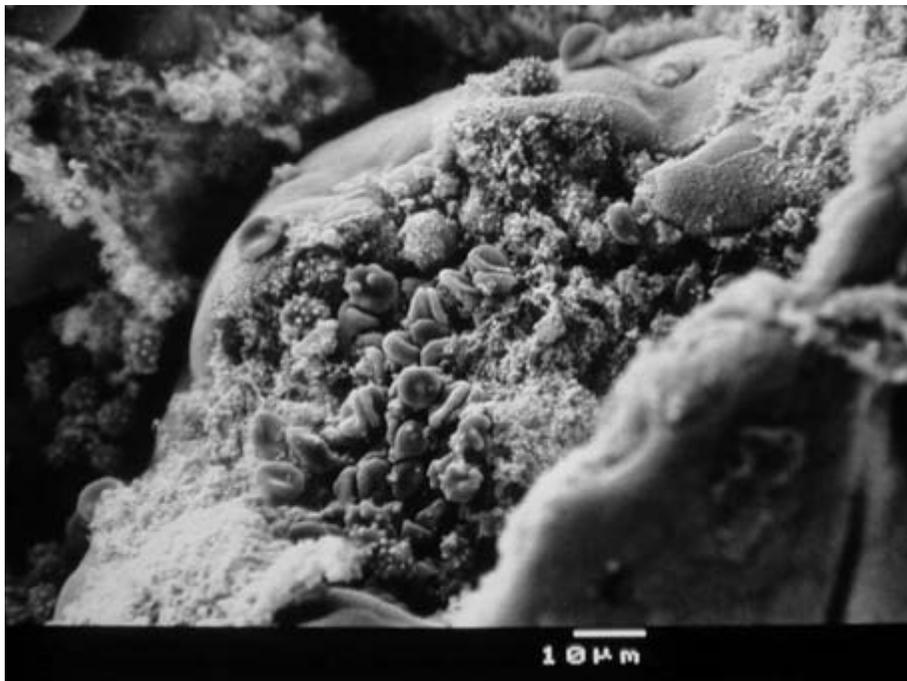


**Non-steroidal anti-inflammatory drug (NSAID) enteropathy (cont.)**

At higher magnification scanning electron microscopy identifies microerosions (Figs 5.50 and 5.51) which probably explain the occult blood loss in patients on these drugs. Random multiple jejunal biopsy is therefore recommended in all patients on NSAIDs attending for small bowel endoscopy.



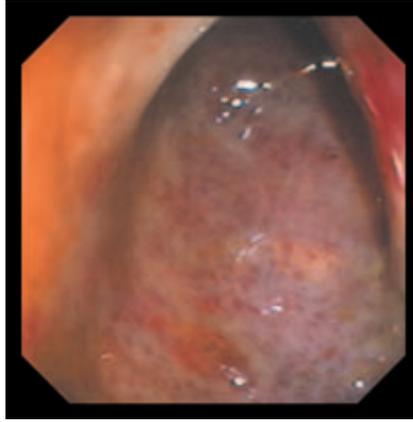
5.50



5.51

**Graft vs. host disease**

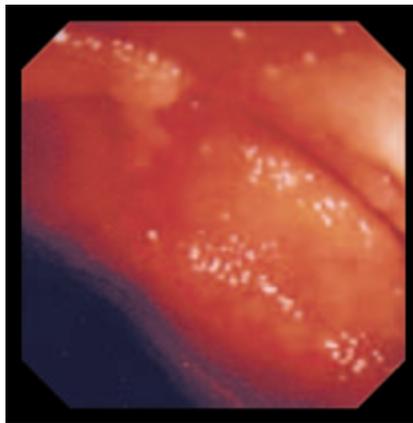
Figure 5.52 illustrates mucosal sloughing and punctate haemorrhages in this disorder.



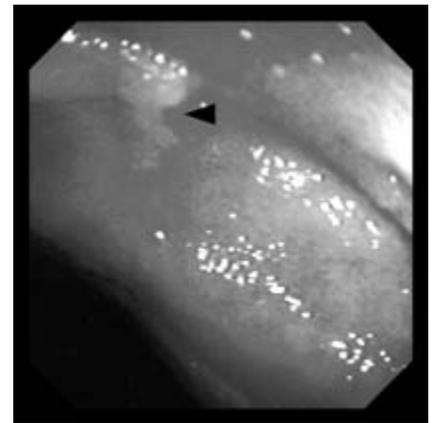
5.52

**Ischaemic enteropathy**

Figure 5.53 shows superficial jejunal ulceration due to subcritical obstruction of the superior mesenteric artery.



5.53



**Cytomegalovirus infection**

Macroscopic disease caused by infection is unusual in the small bowel. Cytomegalovirus infection (Fig. 5.54), here in an immunocompromised patient, causes punched out ulcers.



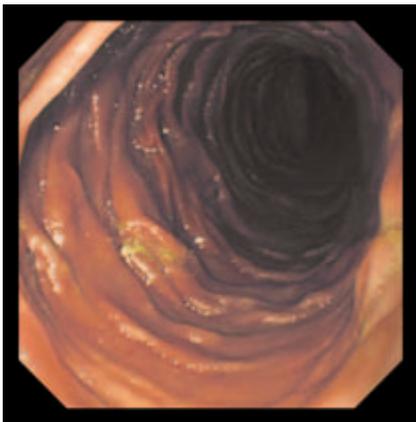
5.54

### Abnormal mucosal folds

Abnormal patterns of intestinal mucosal folds demonstrated by barium meal radiology or CT scanning can be investigated by enteroscopy. Many conditions including coeliac disease, progressive systemic sclerosis (scleroderma), amyloid and other infiltrative disorders may cause such changes. Mucosal biopsy sometimes allows a precise diagnosis. Figure 5.55 shows thickened mucosal folds seen radiographically whilst the enteroscopic appearance is seen Figure 5.56. The cause in this case was widespread infiltration by non-Hodgkin's lymphoma.



5.55



5.56

Applications of enteroscopy are limited only by availability of the necessary expertise and equipment. As more patients are examined, the many apparently unusual conditions will be commonly diagnosed. Descriptions of new diseases, novel perspectives on common disorders and the ability to target therapy for small bowel disease are the aims of enteroscopy today.

# Endoscopic Ultrasonography in Gastroenterology

<b>Principles of ultrasound imaging</b>	<b>470</b>
<b>Overview of technique and normal findings</b>	<b>476</b>
<b>Selected gastrointestinal indications</b>	<b>495</b>
<b>Selected indications for pancreatico-biliary disorders</b>	<b>511</b>
<b>EUS-guided fine needle aspiration biopsy</b>	<b>520</b>
<b>Miscellaneous uses</b>	<b>521</b>
<b>Future prospects</b>	<b>524</b>

Endoscopic ultrasonography (EUS) was initially developed to overcome the limitations of percutaneous ultrasonography (US) in the diagnosis of pancreatic disorders. US visualization of the pancreas is poor because of overlying gas and indifferent ultrasonic image resolution and is thus of little value in the diagnosis of pancreatic disease. More complex techniques such as endoscopic retrograde cholangio-pancreatography (ERCP), computed tomography (CT) and magnetic resonance imaging (MRI) either visualize only the pancreatic ductal system or delineate intrapancreatic abnormalities poorly. There was therefore an abundantly clear need for an imaging procedure by which the appearances of the pancreas could be shown in great detail.

Early in the development of EUS of the upper gastrointestinal tract it became apparent that it was possible to visualize the various layers of the wall of the viscus under investigation. This led directly to the second important application of EUS, namely in the diagnosis of submucosal lesions and the staging of gastrointestinal malignancies. Latterly, EUS has been shown to be significantly superior to CT and MRI in the delineation of gastrointestinal wall disorders.

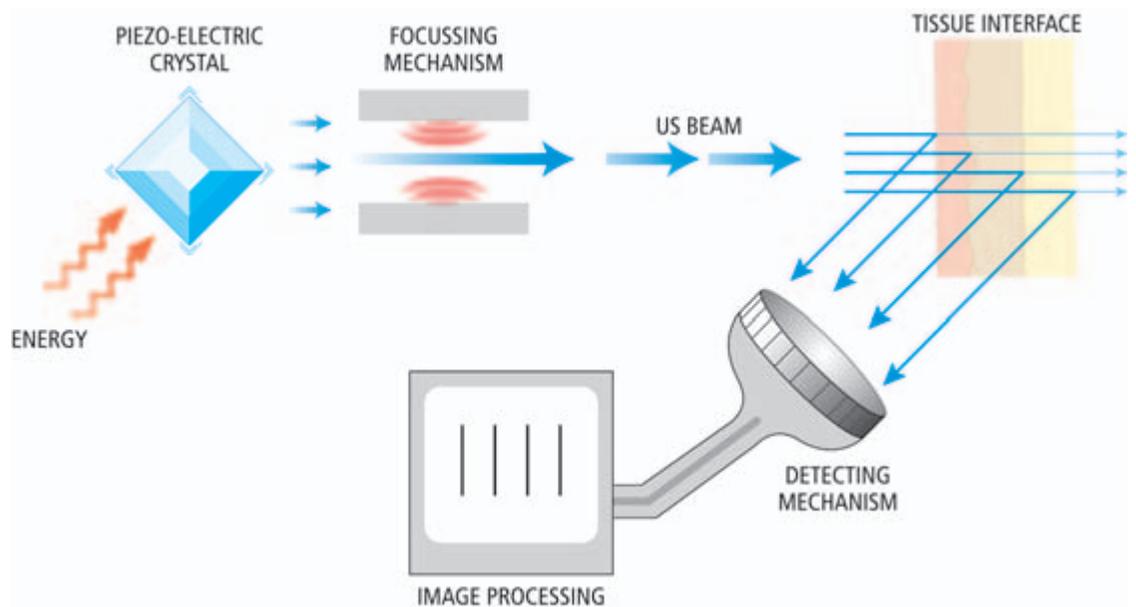
Following earlier pioneering work there have been major developments in US and other imaging techniques, such as spiral CT with phased-contrast imaging and in the field of MRI. However, EUS has similarly advanced, both in terms of the expertise of its practitioners and in the quality of imaging. Recent technological developments include sonographic enhancing agents and 3-dimensional imaging. The advent of colour Doppler EUS has also proved most helpful.

The third major role of EUS is for biopsy and therapeutic applications. Such procedures require the US examination to be in a different mode as explained later. EUS-guided fine needle aspiration (FNA) has proved useful in the diagnosis of lesions in both the abdomen and thorax and is likely to gain wider acceptance as experience grows. EUS-guided aspiration of cysts and pseudocysts and EUS-guided management of oesophageal varices and pancreatico-biliary disease are similarly likely to undergo developments.

## Principles of ultrasound imaging

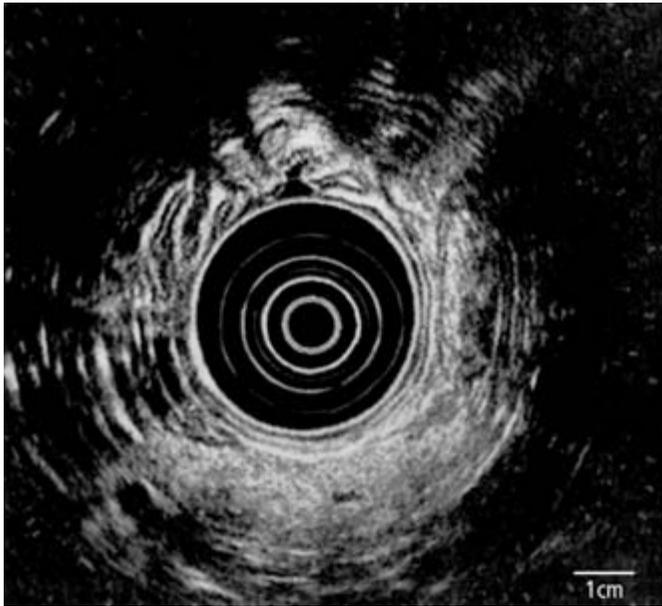
Sonographic (ultrasound) technology involves the generation of a high frequency energy wave by electrical vibration of a piezoelectric crystal. The wave energy can be pulsed, focused and directed to pass through tissue. Part of the high frequency pulse will be transmitted through or absorbed by an interface of two materials of differing densities and a proportion will be reflected and scattered from the interface. The characteristics of this wave energy are described by its wavelength ( $\lambda$ ) and frequency (MHz). The reflected energy wave can be detected and its intensity or amplitude represented on a visual display. It is important to realize that the visual display is a representation of the time taken for the sound waves to return from an interface, i.e. the further the interface, the longer the separation of sound waves which therefore can indirectly reflect distance between different interfaces.

Figure 6.1 is a diagrammatic depiction of the principles of sonography. Pulses of high frequency ultrasound are directed at tissue and energy is transmitted through, absorbed and/or reflected at each interface, the relative proportions being determined by the nature of the interface and the adjacent structures. The intensity or 'brightness' of echo reflection is determined by interface characteristics, with amount of air, gaseous or liquid material and scatter being important additional factors.

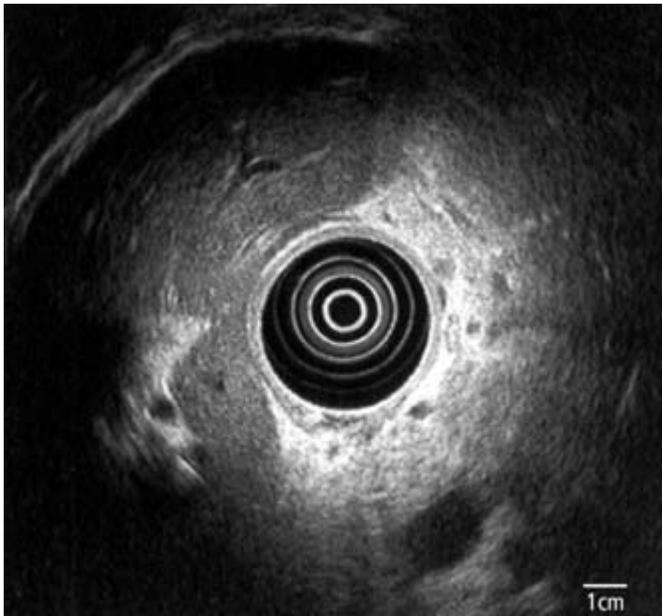
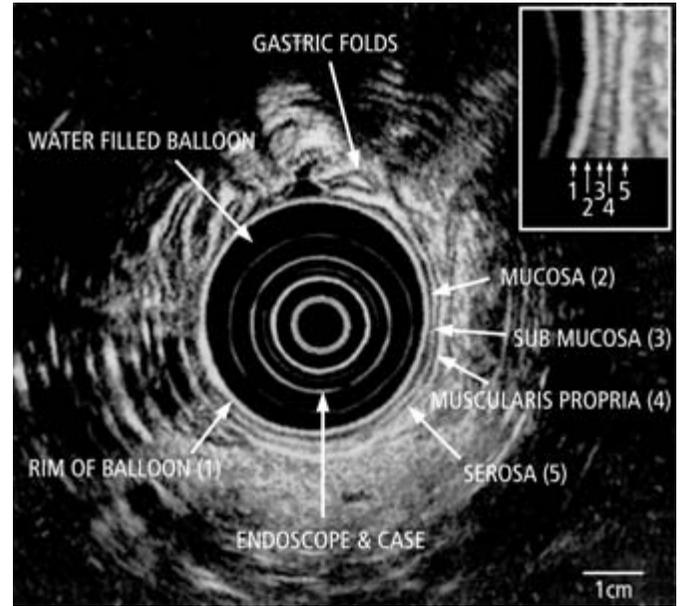


6.1

The angle of incidence of the transmitted wave and its reflection is also important. Several of these issues are illustrated in Fig. 6.2 which shows the reflective layers of the gastric mucosa and the bright echo pattern of fat in the liver (Fig. 6.3).



6.2



6.3

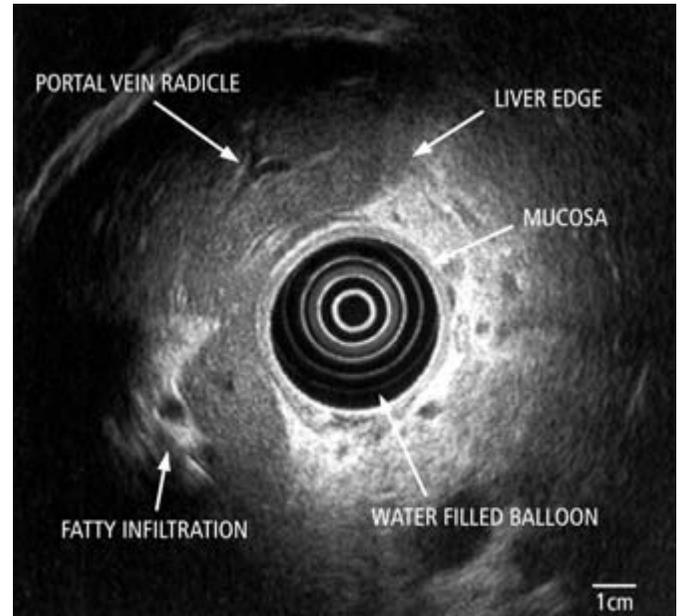
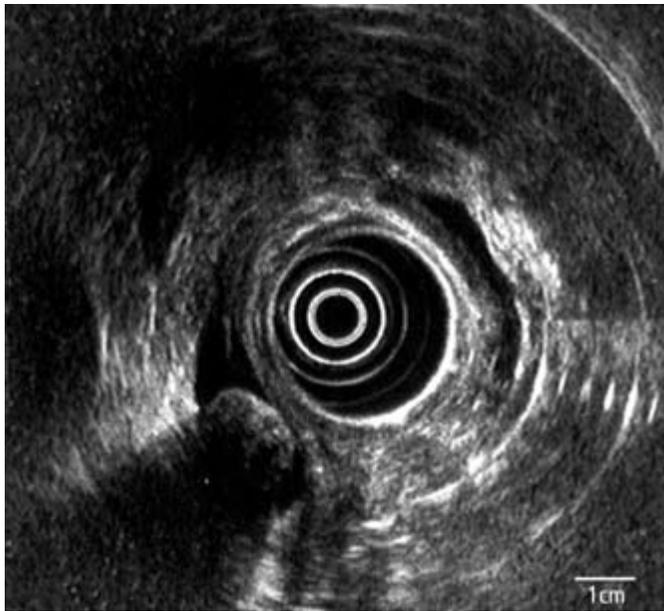
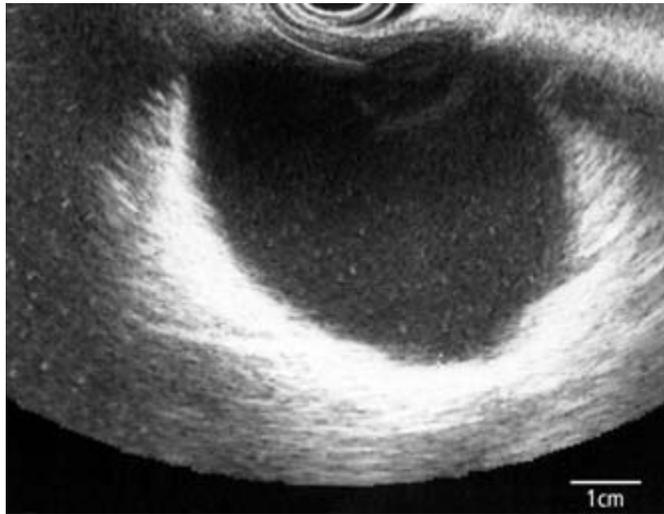
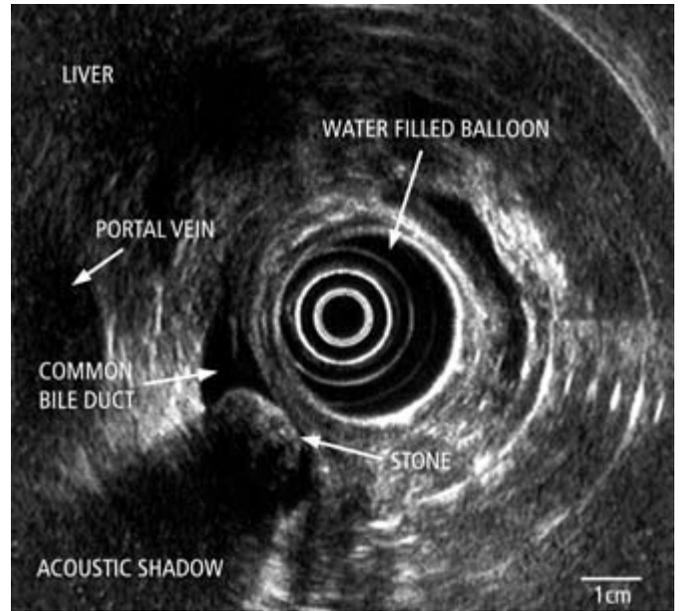


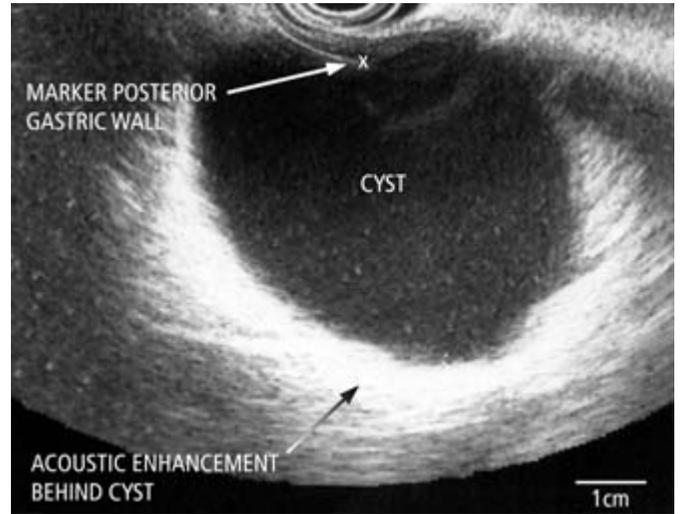
Figure 6.4 shows the acoustic 'shadow' cast by a gallstone and Fig. 6.5 the lack of echoes in a rounded structure with 'enhancement' behind it being the characteristics of a cyst. Enhancement occurs because sound waves which have passed through the cyst are not as attenuated as they would be if they had to pass through the adjacent tissue.



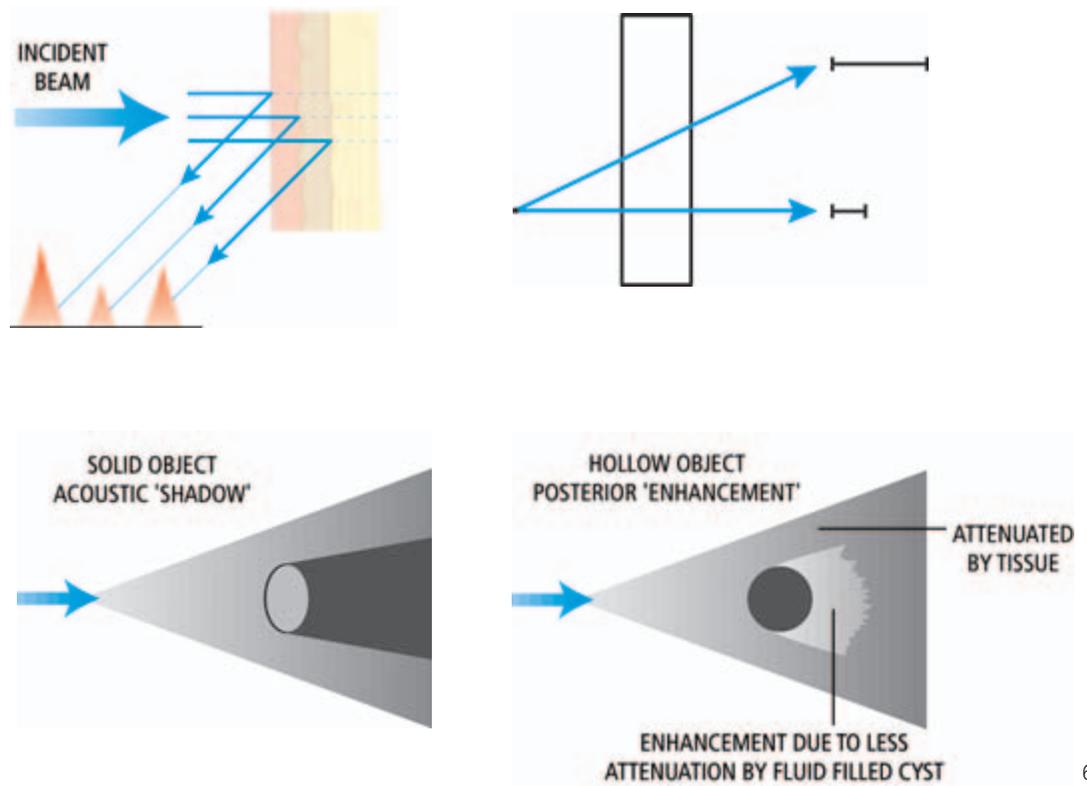
6.4



6.5

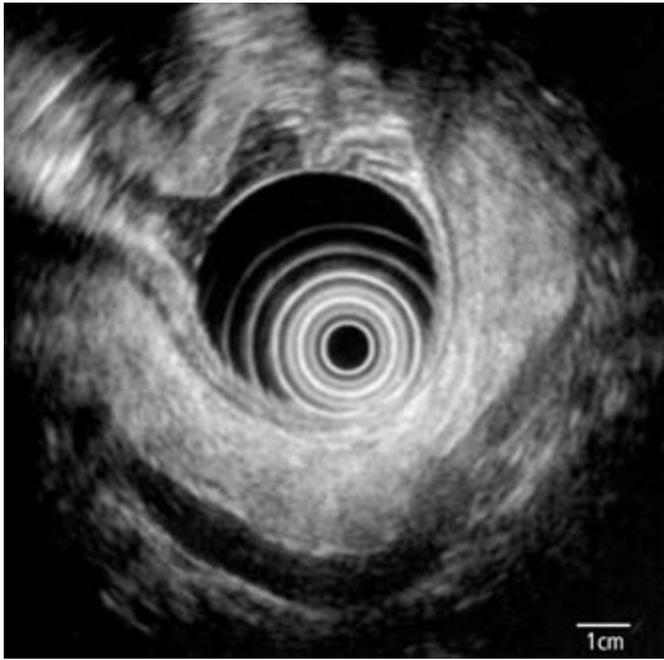


Some of the physics of these features are illustrated in Fig. 6.6. The endoscope, its casings and the water-filled balloon surrounding its tip produce their own appearances on the EUS image. These are marked up on Fig. 6.2 but are not routinely marked on subsequent figures.

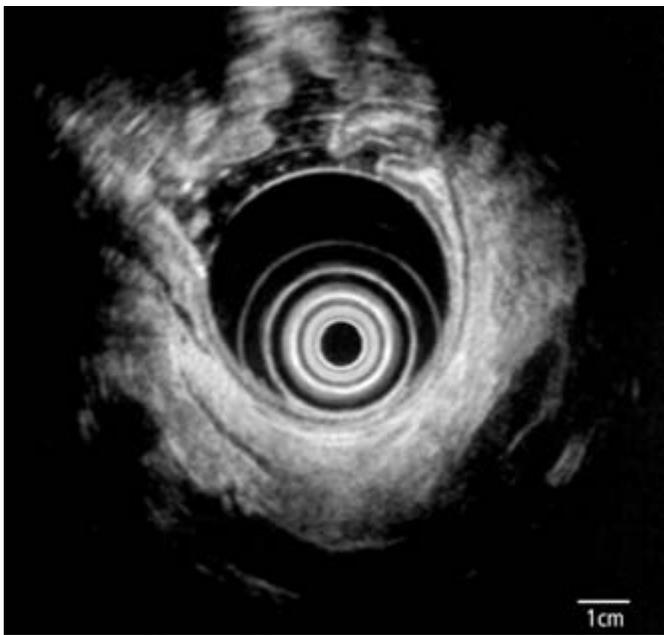
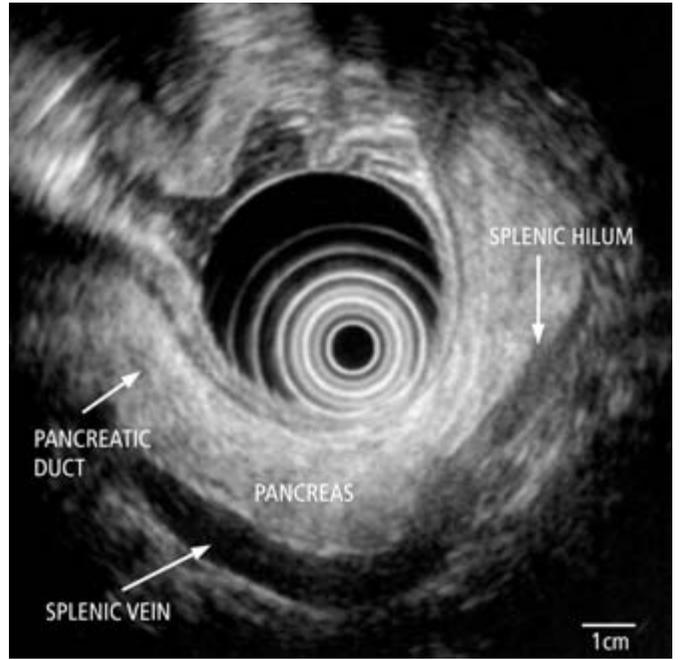


6.6

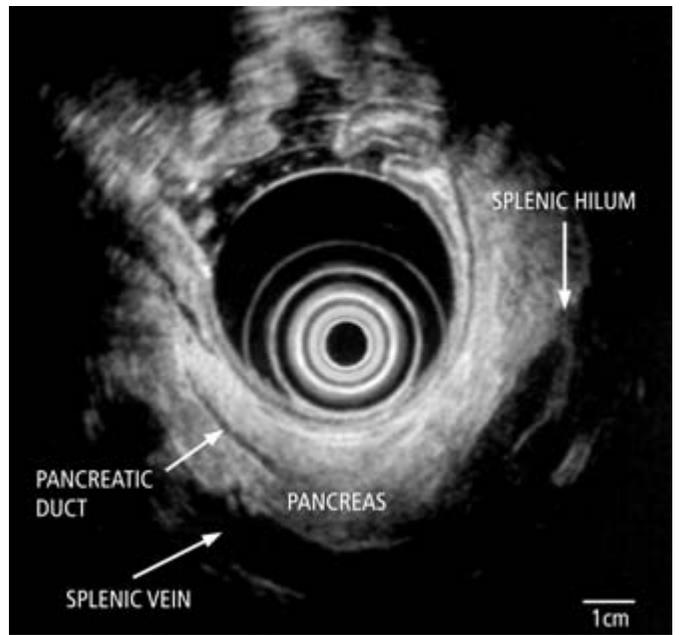
The frequency and wavelength of transmitted sound will determine penetration, clarity and sharpness of an image. Lower frequencies have deeper penetration but less sharp images are obtained due to reflection and scatter. This is illustrated in Fig. 6.7 (7.5 MHz) and Fig. 6.8 (12 MHz) showing different levels of detail of the same structure. At 7.5 MHz the retroperitoneal view from the stomach shows good penetration behind the splenic vein. The same view at 12 MHz demonstrates the gastric mural structures in finer detail, and the pancreas and pancreatic duct are clearly seen; penetration however is poorer.



6.7

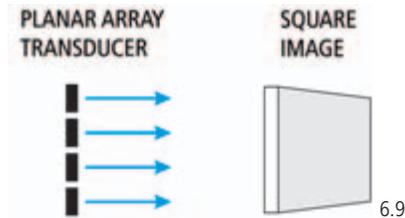


6.8

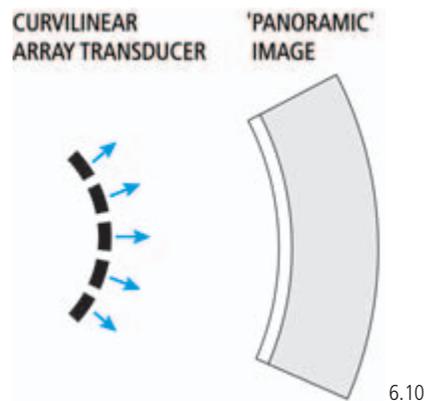


Detail of transmural anatomy depends upon good contact between the probe and the mucosa, without the presence of luminal air, and on the frequency of the probe being used. To avoid intraluminal air, either a water-filled balloon is used with its outer rim lying against the mucosal surface, or the viscus can be filled with water, remembering of course that in certain anatomical situations there is a risk of aspiration.

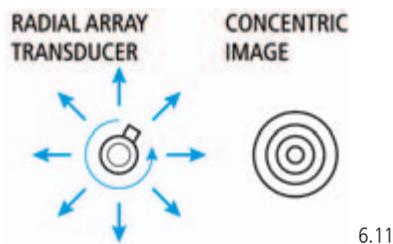
The plane of imaging is determined by the type of EUS probe employed. Sometimes a number of probes are used together to produce an array of images. The simplest is the planar array which transmits in one direction and produces a representative image of parallel structures (Fig. 6.9).



A curvilinear array is similar but can obtain image data over a wider area, which can be helpful as images have to be interpreted by relating one structure with another (Fig. 6.10).



The radial array is an extension of this curvilinear concept and is usually defined by the amount of sectoring or degrees field of 'vision'. The most commonly used radial arrays are 270° or 360° (Fig. 6.11).



**Who should perform EUS? 476****How is the patient prepared for EUS? 476****Instrumentation 476****Technique, visualization and normal findings 477**

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**Who should perform EUS?**

EUS is practised by gastroenterologists, surgeons specializing in gastroenterology and radiologists. Each of these specialties has advantages and disadvantages in terms of learning how to perform EUS. Surgeons and radiologists tend to be more familiar with retroperitoneal vascular anatomy and anatomical relationships in general. Radiologists clearly have an edge in terms of interpretation of EUS images as they are already more familiar with US images. Gastroenterologists, particularly those with a background in ERCP, may have an advantage in being more familiar with 3-dimensional orientation in relation to endoscope position. At present in the United Kingdom, the EUS users group is split equally amongst these disciplines.

There is no need for more personnel than for routine gastrointestinal endoscopy, although physicians and surgeons may welcome the presence of a radiologist during the learning phase. A radiographer or specialist technician is not normally required.

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**How is the patient prepared for EUS?**

Preparation is as for routine upper gastrointestinal endoscopy or ERCP, although explanations offered to obtain informed consent will be a little different. Pharyngeal topical anaesthesia is given in the usual way, and the patient is sedated for example with midazolam, occasionally with the addition of pethidine or, if an anaesthetist is available, with propofol. Preparation for anal and rectal endoluminal endoscopy is discussed on pp. 505–507.

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**Instrumentation**

Echoendoscopes for scanning the upper gastrointestinal tract and the retroperitoneum are side-viewing and carry a small ultrasonic transducer in the rigid tip. The use of higher frequencies (5–20 MHz, mostly 7.5 MHz) than those employed for transabdominal US provides better image resolution at the cost of limited depth of penetration (maximum 4–5 cm). The mode of ultrasonic scanning is either radial where usually a 360° ultrasound roundview is generated around the shaft of the instrument, or linear, in line with the instrument, where the ultrasound view varies from a section of 90° to 270°, depending on the instrument used. A range of flexible Olympus echoendoscopes is shown in Fig. 6.12. For anal and rectal ultrasonography it is usual to use rigid probes.

When using an echoendoscope, the optical views are similar to those obtained during conventional endoscopy but slightly oblique, and the flexion range of the tip is limited. This enables the presence of gross abnormalities of the gastrointestinal tract to be confirmed or excluded: for a detailed optical endoscopic examination of all areas the current range of echoendoscopes is not optimal.



6.12

The instrument channel of echoendoscopes enables biopsies to be taken as appropriate and can also be used for fine needle aspiration biopsy (FNAB; see p. 520) and injection therapy.

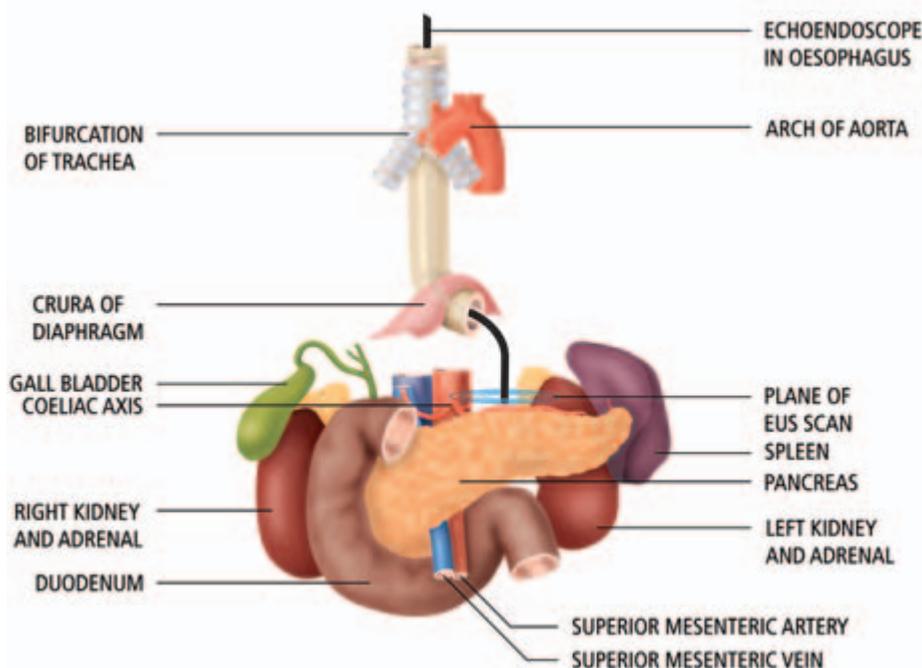
The EUS images shown in this chapter other than those of rectal and anal appearances were taken in the main via an Olympus 240 echoendoscope which gives a 360° radial roundview.

### Technique, visualization and normal findings

As EUS has a limited penetration depth due to the high frequencies used, the pancreas and the extrahepatic biliary system are the only extraluminal structures which are close enough for full visualization. It follows that complete delineation of other organs such as liver, spleen and kidneys is beyond the capability of EUS. For visualization of the various parts of the oesophageal and gastric wall, and of the various parts of the pancreas, there are a number of more or less standardized instrument positions which may, however, be modified appropriately in the face of anatomical variations.

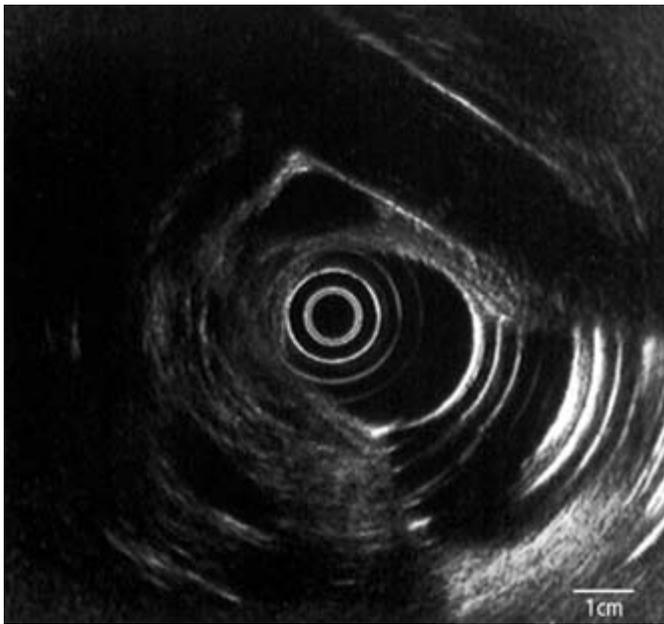
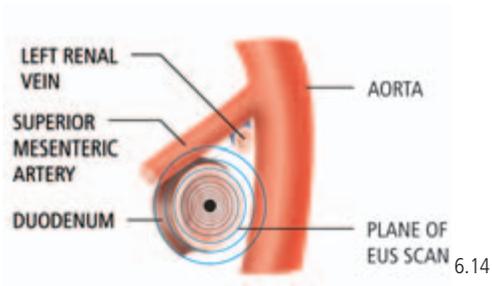
#### Organ structures

Organ structures such as the pancreas and biliary tract are viewed with a radial array endoscope usually at 7.5 MHz. Structures are recognized by their relationship to anatomical landmarks. Crucial to interpretation are the 'fixed' retroperitoneal vascular structures which display little variation between individuals, and the relationship of anatomical structures such as the aorta, carina and diaphragm to the oesophagus (Fig. 6.13).

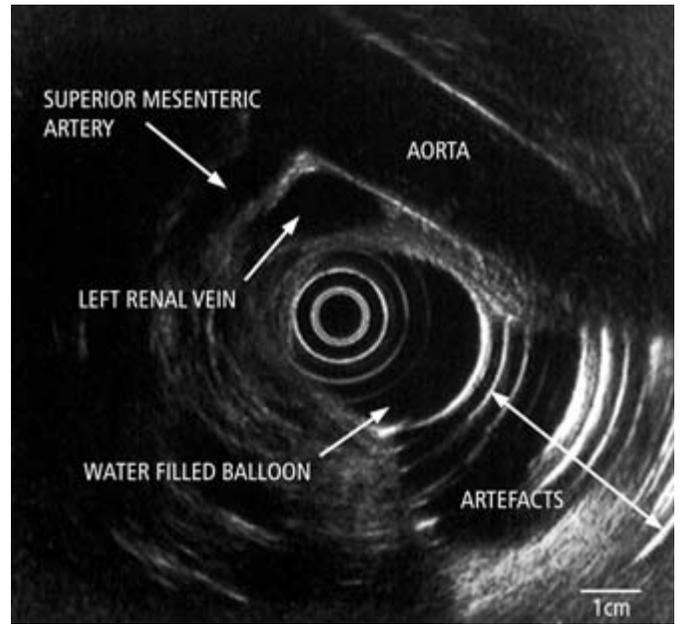


6.13

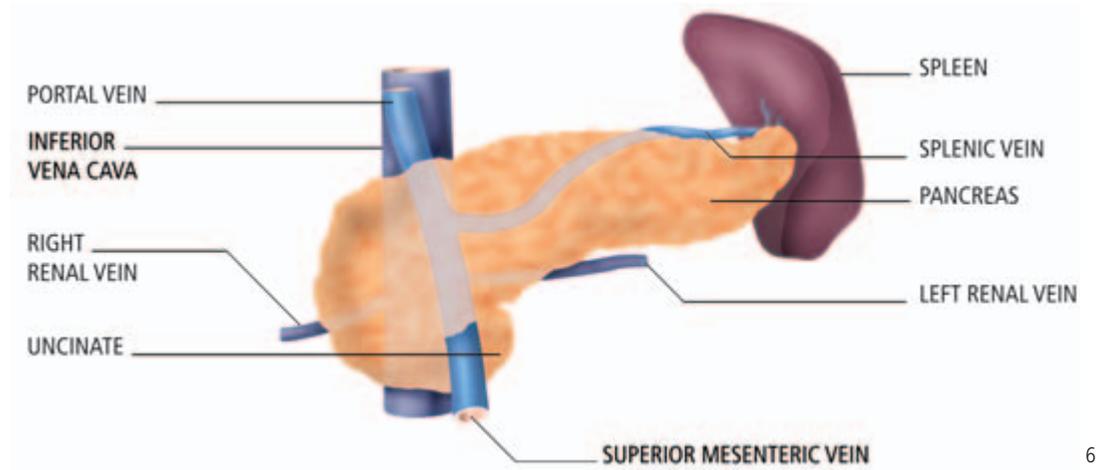
Thus, for example, an examination beginning within the third part of duodenum with the endoscope tip in a near-horizontal position should during gradual withdrawal should demonstrate in order: firstly, the origin of the superior mesenteric artery from the aorta (Figs 6.14 and 6.15).



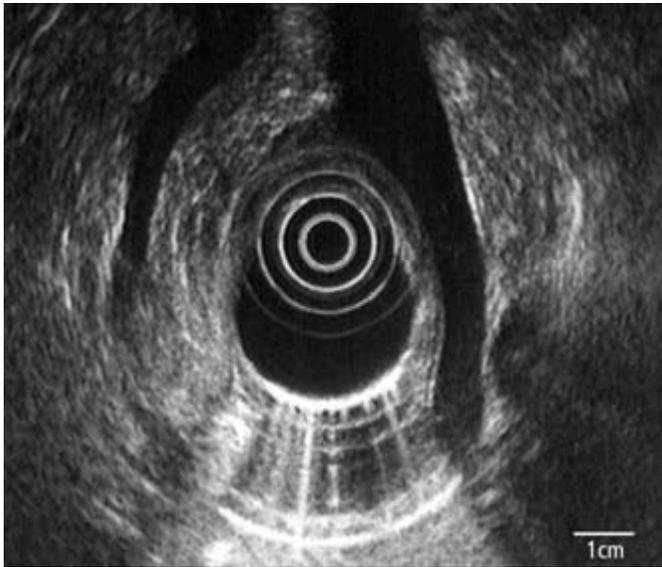
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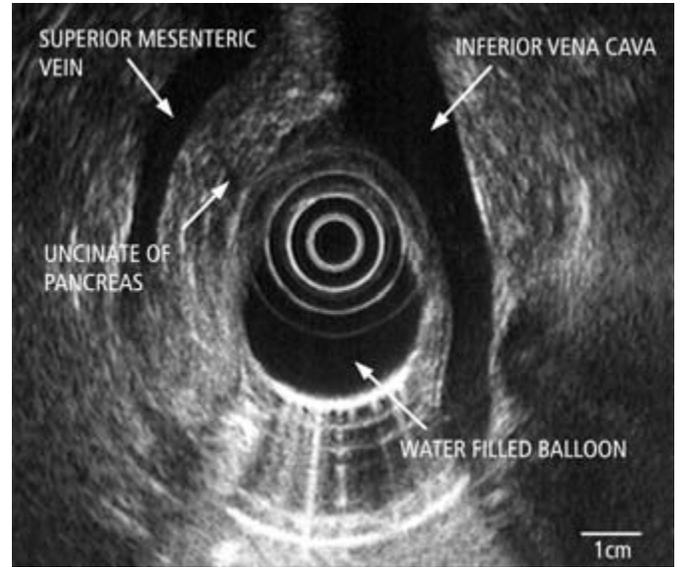
Secondly, the parallel courses of the superior mesenteric vein and inferior vena cava 'sandwiching' the uncinate process of pancreas (Figs 6.16 and 6.17).



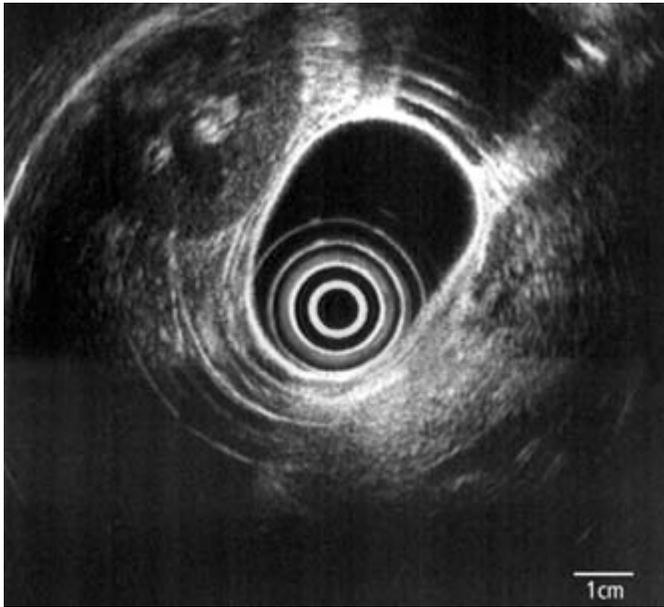
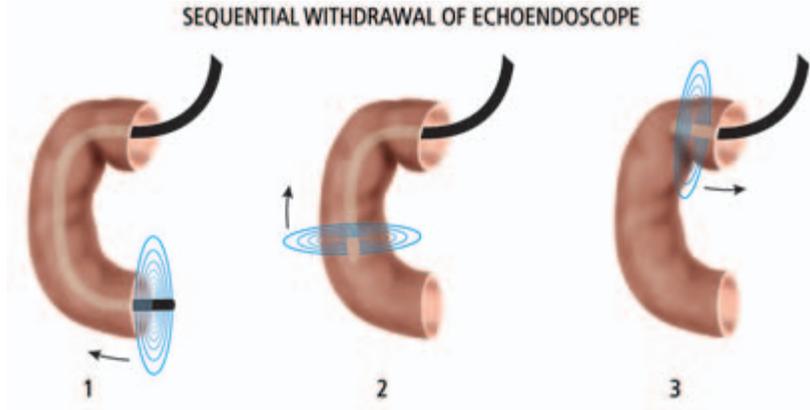
6.16



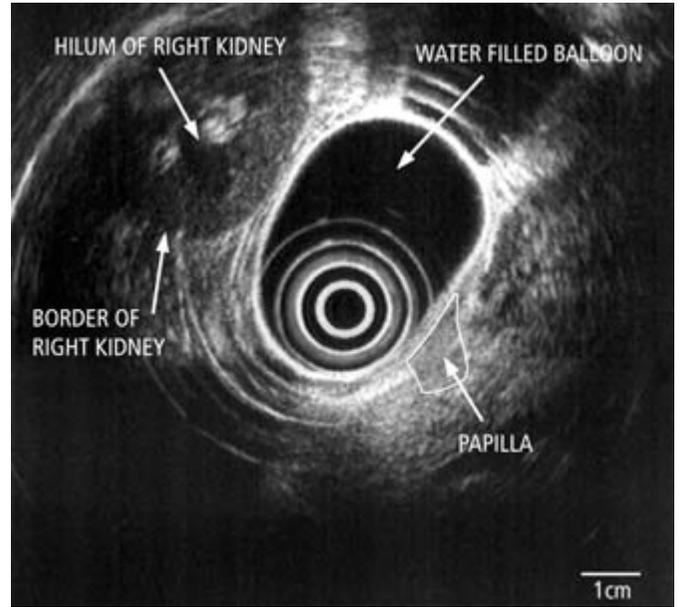
6.17



As the endoscope is further withdrawn the tip is gradually rotated through 180° (in the coronal plane) around the curve of the duodenum into the bulb and should demonstrate the right kidney, major papilla (of Vater), head of pancreas and subsequently the bile duct (Figs 6.18–6.20),



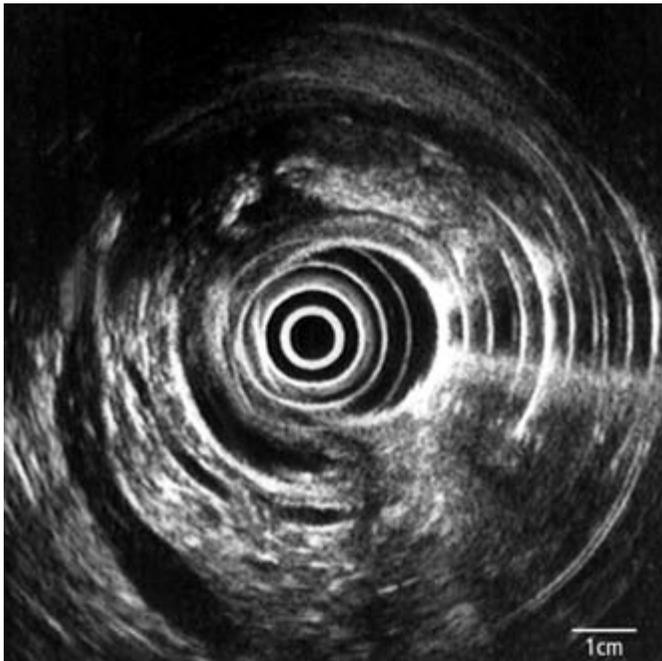
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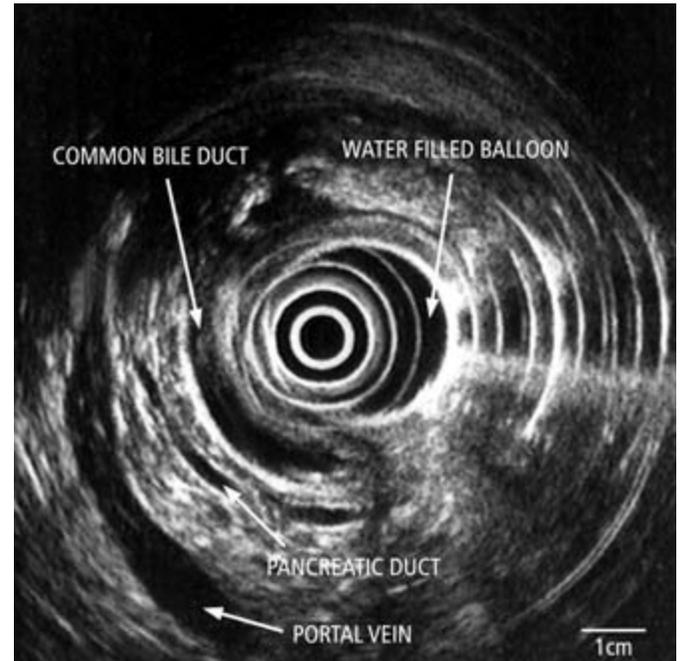
6.20



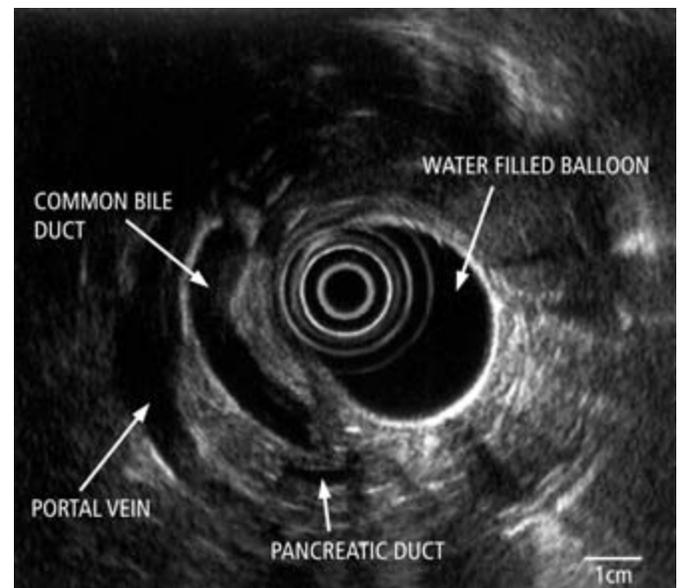
the pancreatic duct, parallel to the common bile duct, as it courses through the head of pancreas and across the midline, and the portal vein behind, running parallel to the bile duct into the liver (Fig. 6.21). Small adjustments of the position of the endoscope tip in each of these positions allows a thorough examination of the head of pancreas, bile duct and portal vein (Figs 6.22–6.24). Figure 6.22 shows the so-called ‘stack sign’, where the common bile duct, pancreatic duct and portal vein appear briefly as a parallel stack.



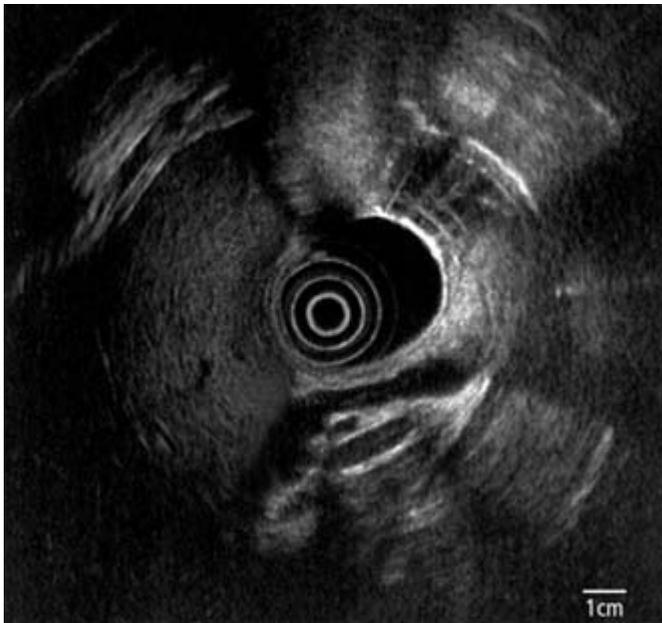
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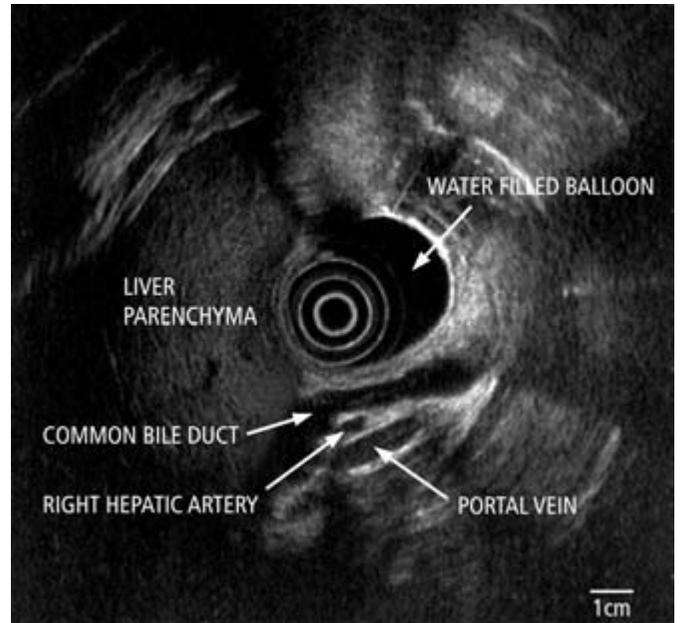
6.22



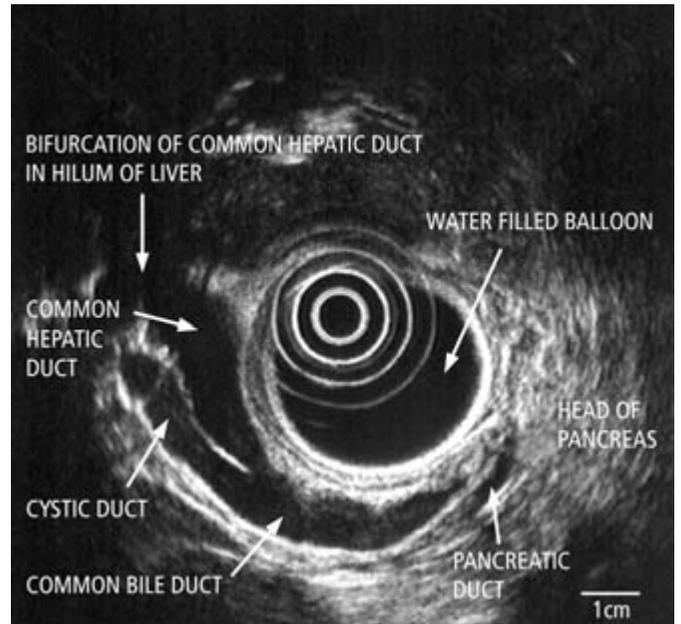
With the endoscope in the duodenal bulb just below the hilum of the liver (Fig. 6.23) the right hepatic artery can be seen between the common bile duct and the portal vein at the hilum. Slightly altering the position of the endoscope reveals the pancreatic duct in the head of the pancreas and a considerable portion of the biliary tree (Fig. 6.24).

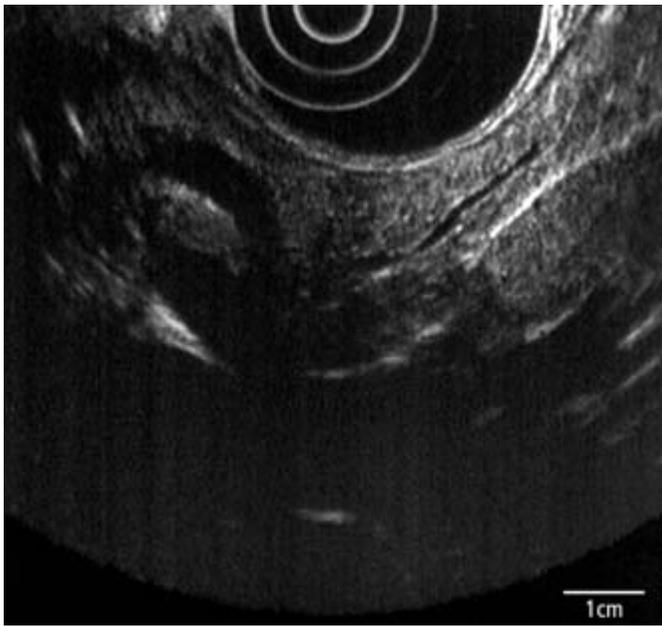


6.23

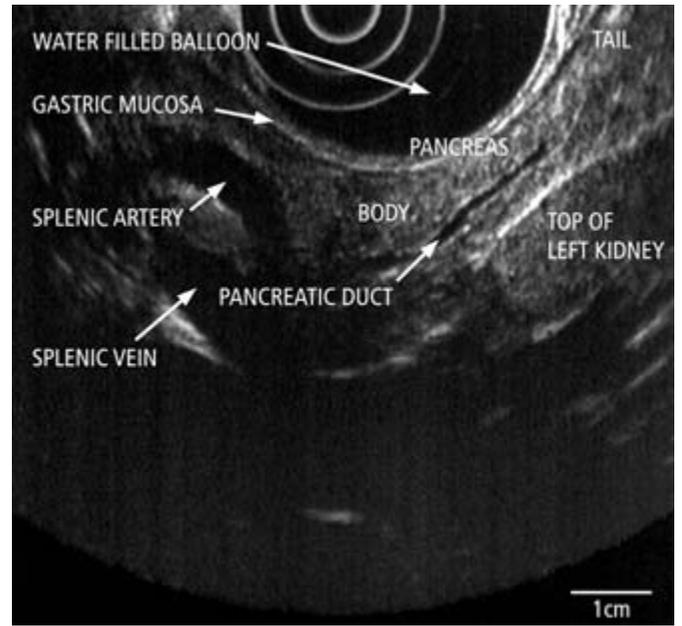


6.24

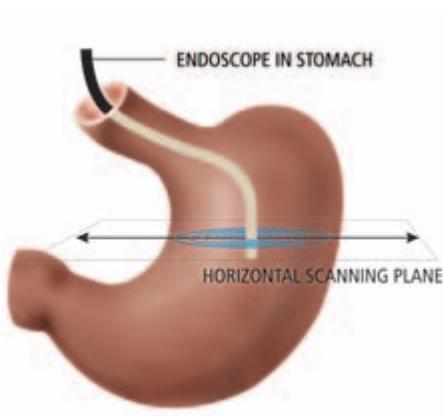




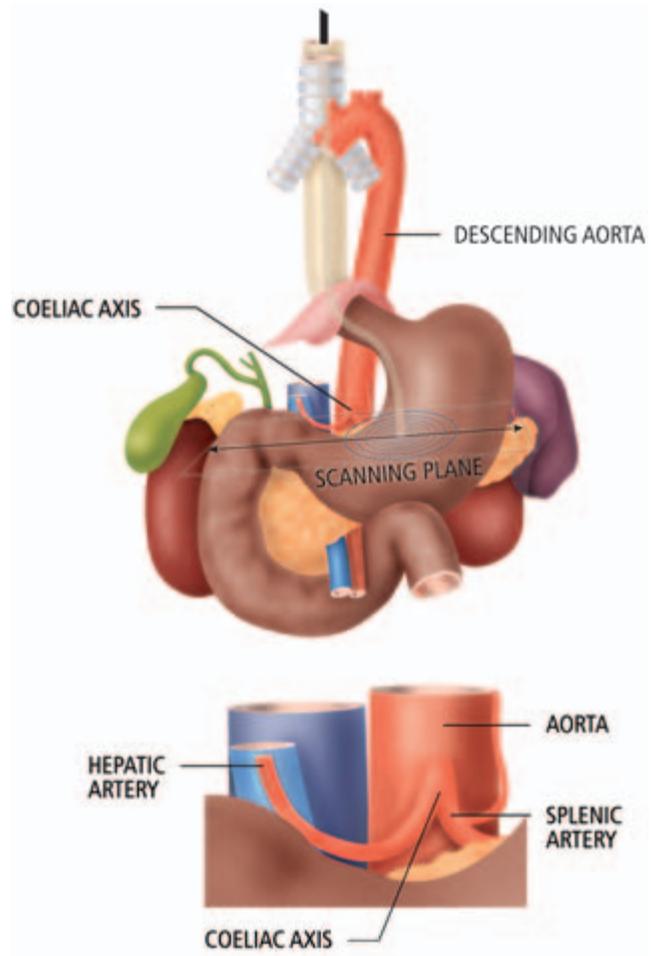
6.26



In the stomach with the endoscope tip in the vertical plane, a horizontal section through the body and tail of pancreas can be obtained with the portal venous confluence and splenic vein behind (Figs 6.25 and 6.26).

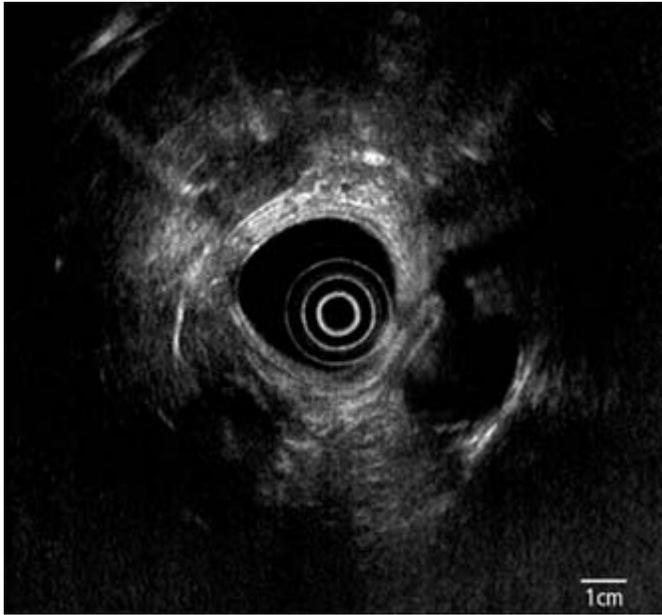


6.25

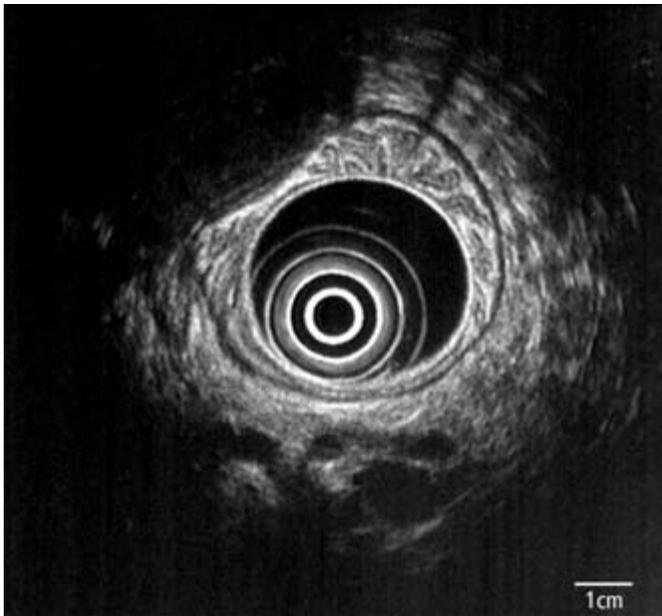
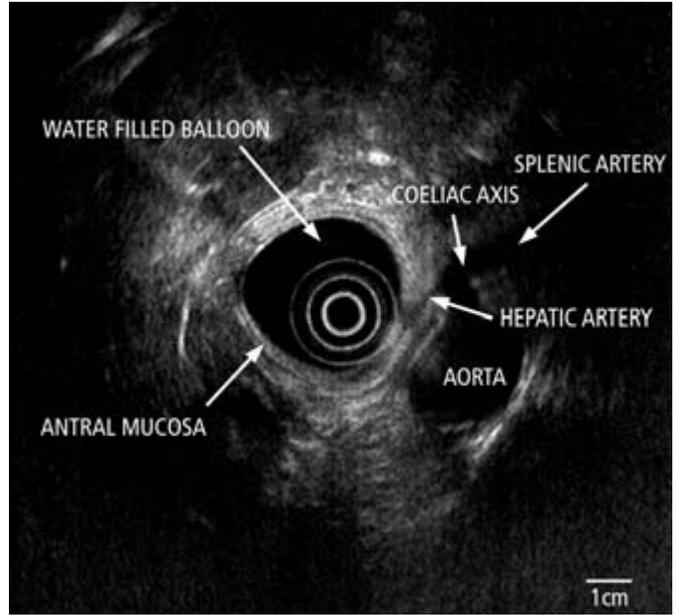


6.27

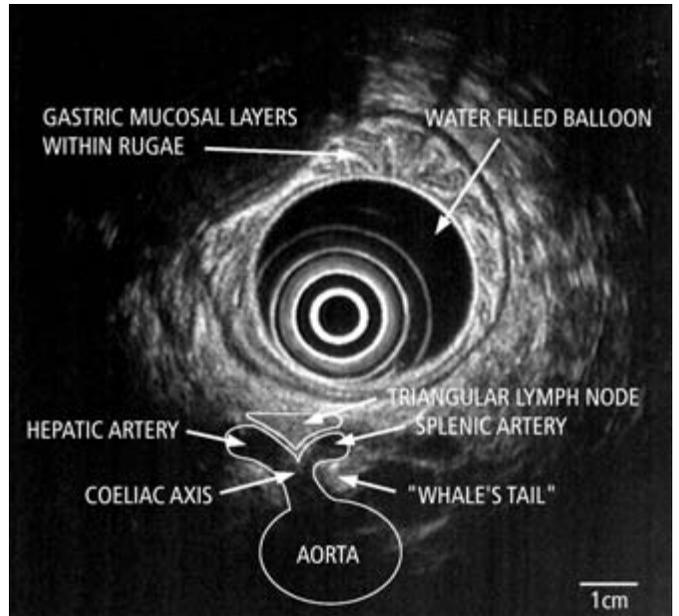
By subtle manipulation of the tip, the coeliac axis, its constituent arteries and its proximate lymph nodes can be identified, and the left lobe of liver can be examined (Figs 6.27–6.30). Another view of the coeliac axis (Fig. 6.29) demonstrates the ‘whale’s tail’ appearance.

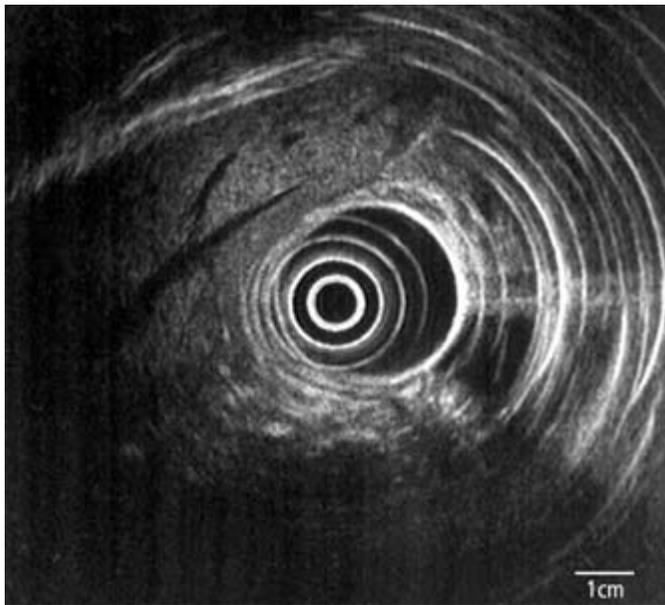


6.28

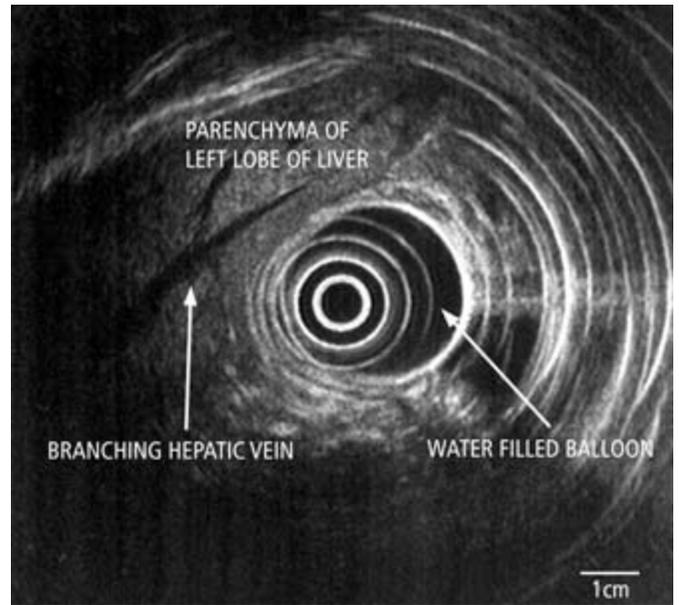


6.29

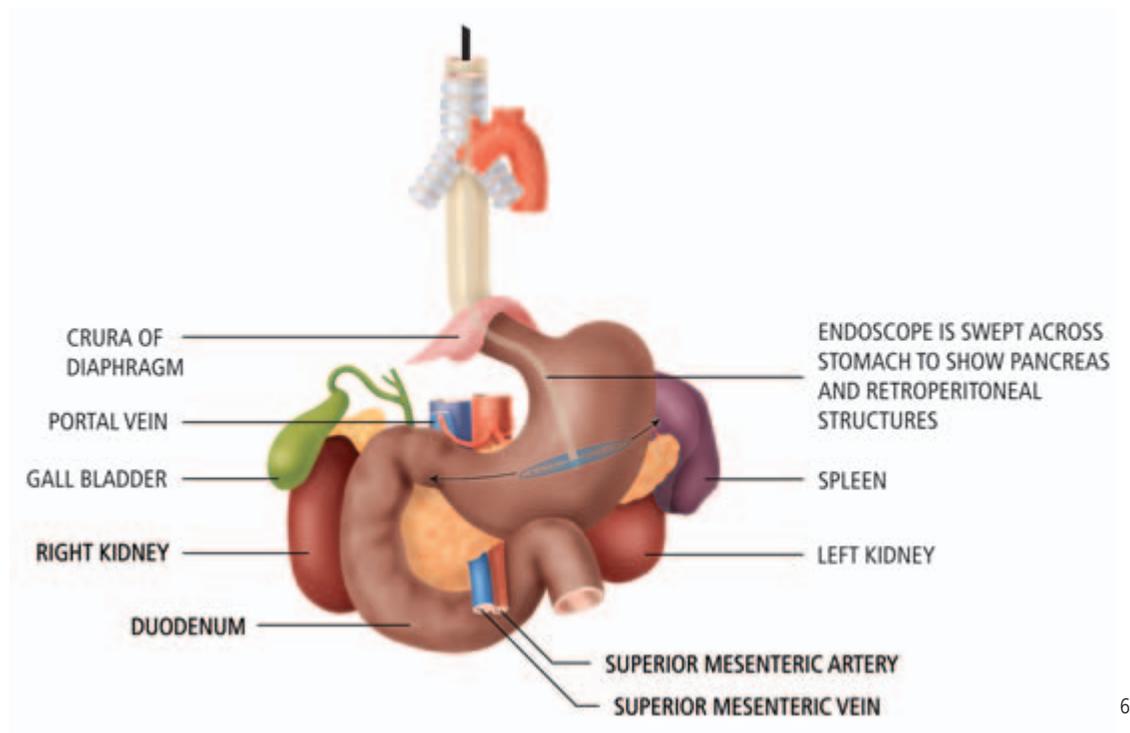




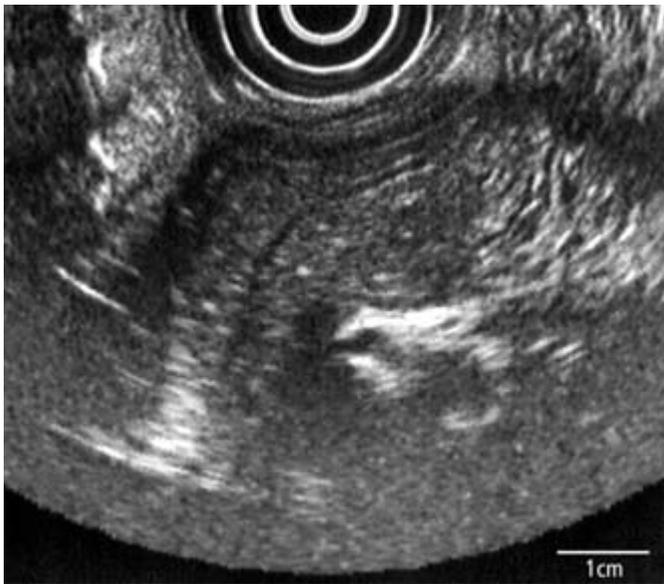
6.30



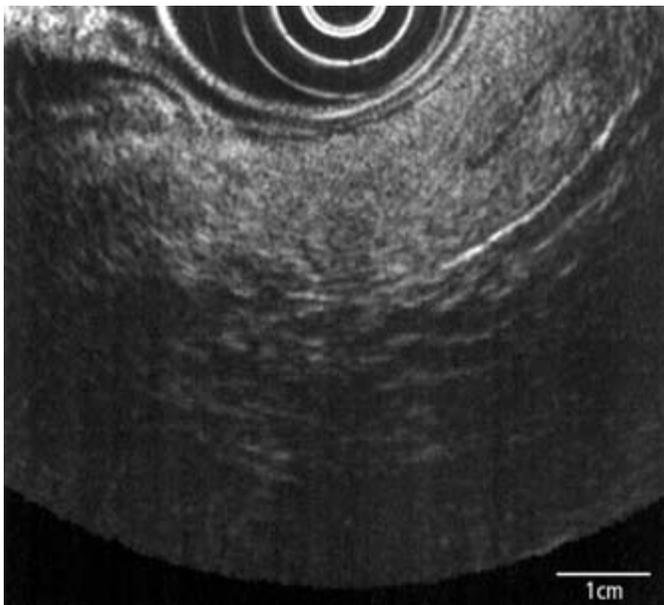
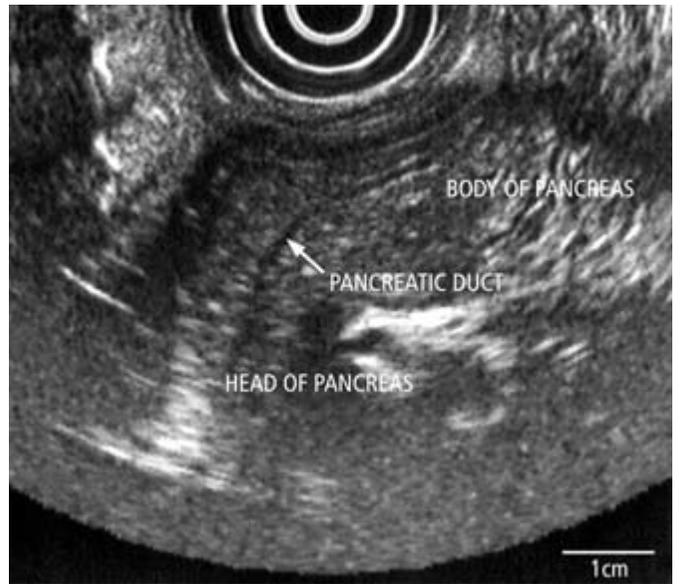
The entire body and tail of pancreas can be seen with a 'sweeping' motion when the pancreas is shown in front of the splenic vein with the splenic artery coursing in and out of the frames of view (Figs 6.31–6.33).



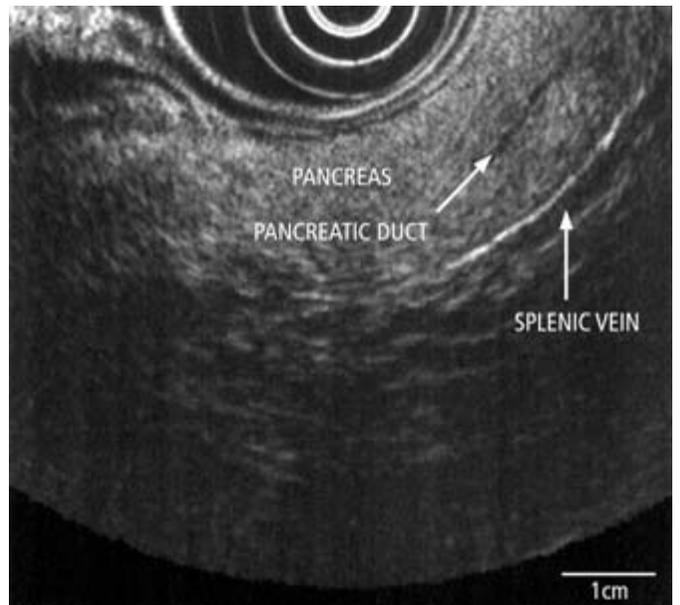
6.31



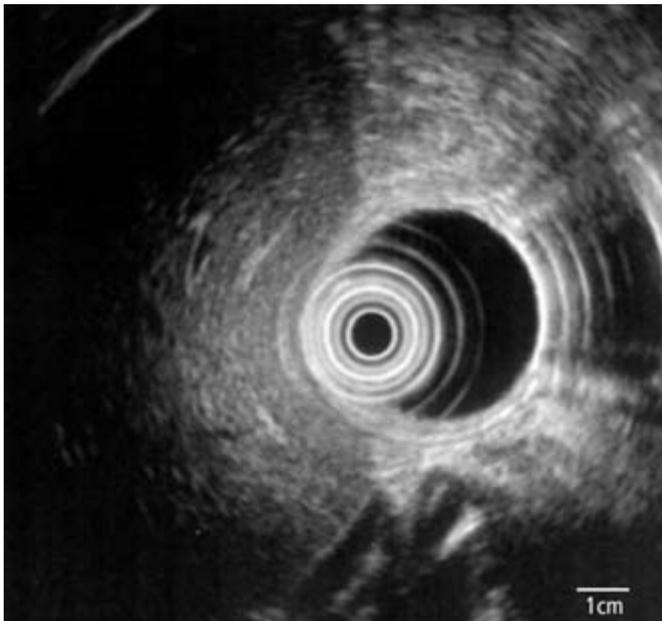
6.32



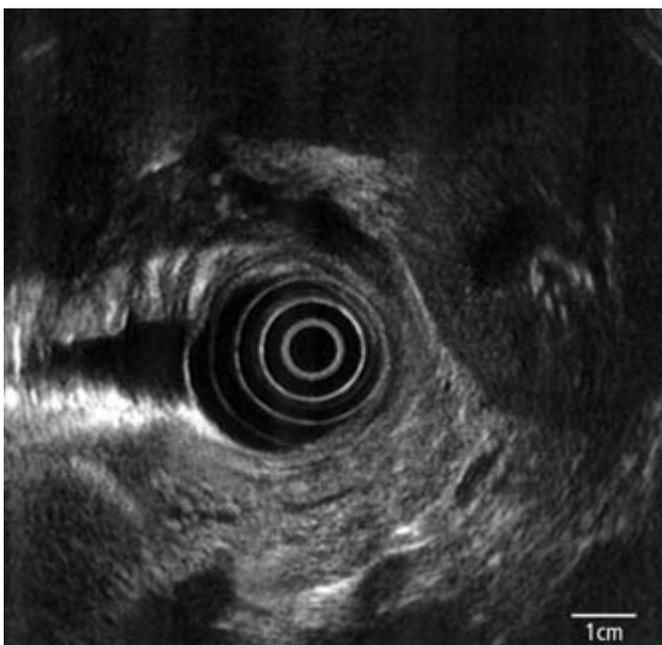
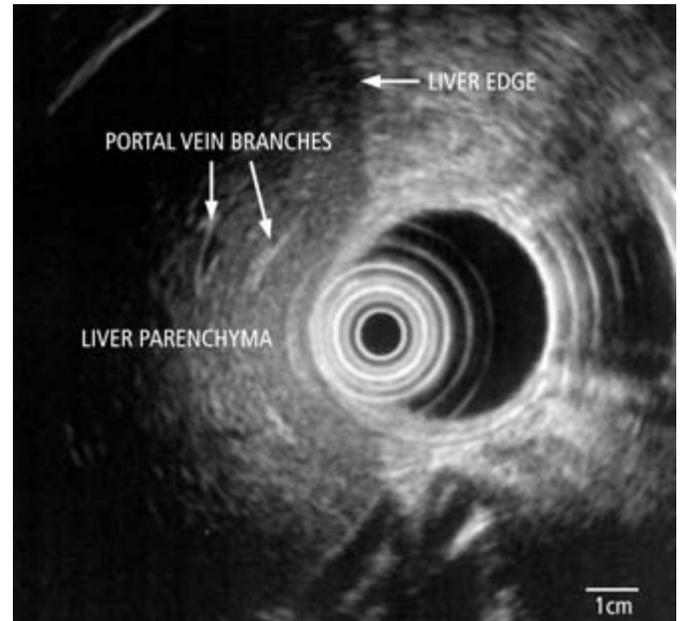
6.33



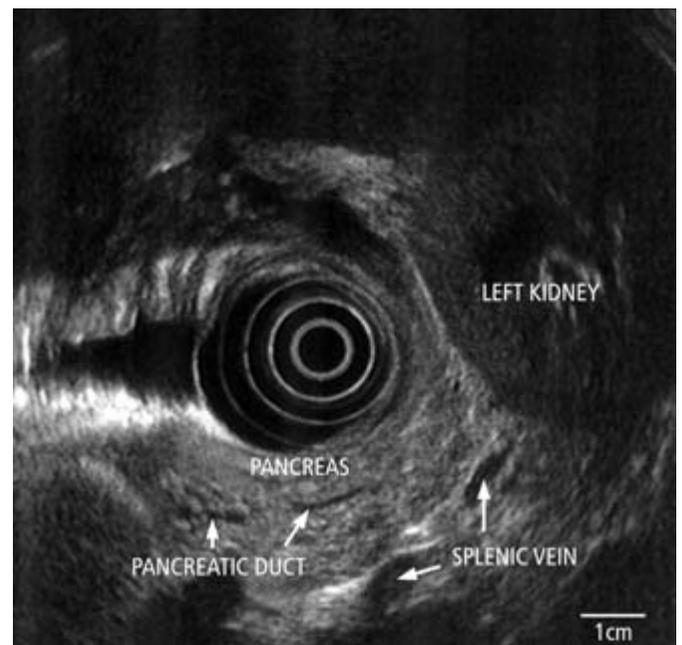
Broadly speaking, the normal pancreas has been described as having an homogeneous echo pattern slightly brighter than that of the adjacent liver (Figs 6.34 and 6.35); the ventral part of the head is usually less bright than the rest of the pancreas. Considered in more detail, the liver on EUS (Fig. 6.34) is hypoechoic, containing tubular structures with hyperechoic margins (portal veins) and a fine granular pattern in the parenchyma. The hepatic margins are well defined. The pancreas (Fig. 6.35) is slightly more hyperechoic in comparison to the liver. It is bordered by the splenic vein behind but otherwise its borders are often indistinct. It is usually possible to identify the pancreatic duct and follow its course. The pancreas has a more finely granular pattern than the liver, often referred to as a 'salt-and-pepper' appearance.



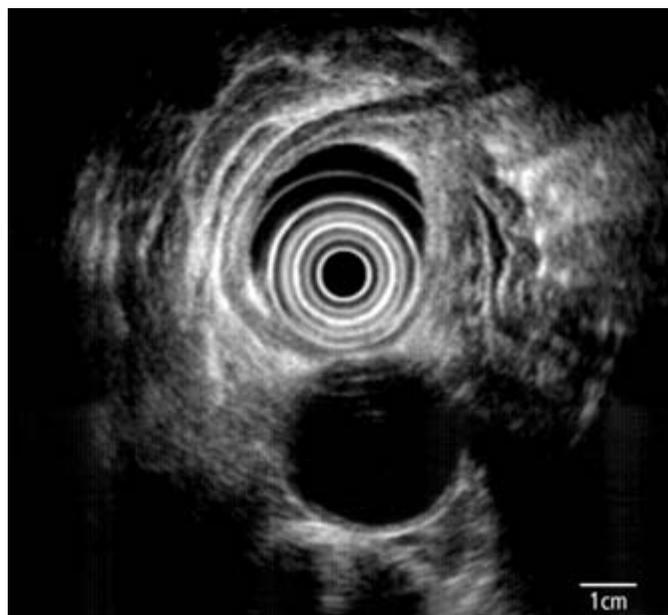
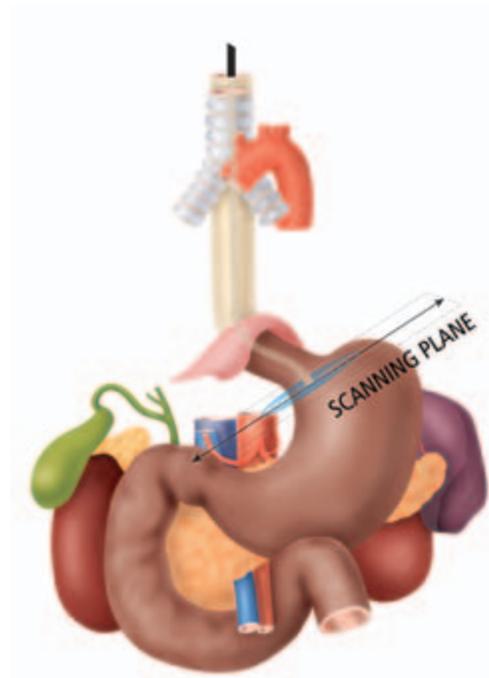
6.34



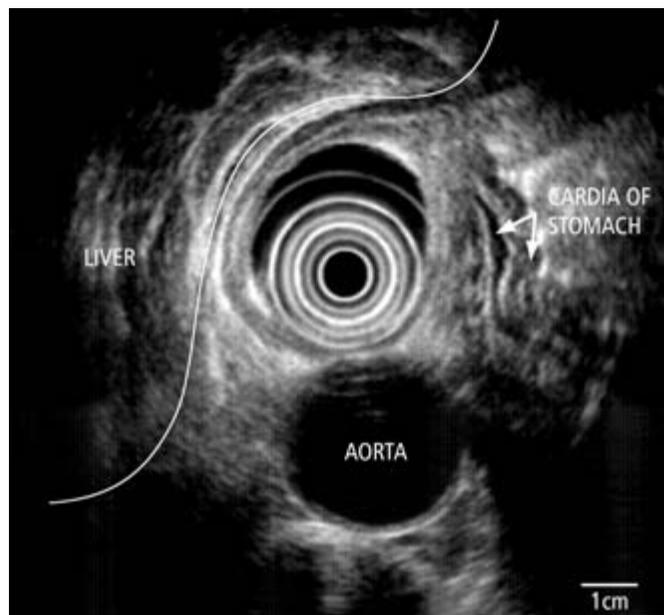
6.35

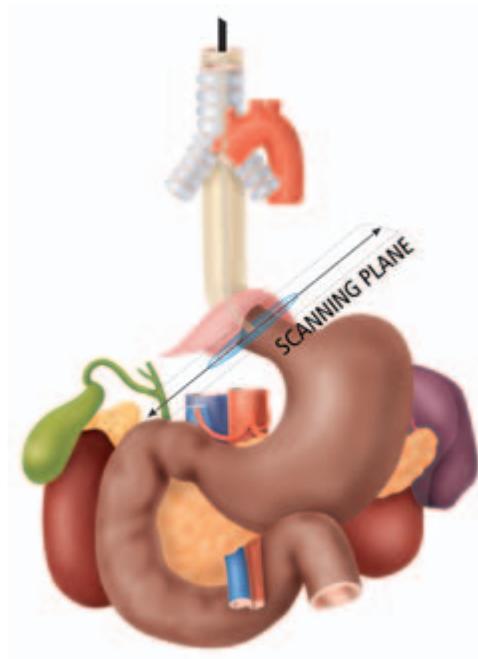


Further withdrawal of the endoscope brings into view the aorta and cardia of the stomach (Figs 6.36 and 6.37), the crura of the diaphragm (Figs 6.38 and 6.39), lymph nodes in the aorto-pulmonary window (Figs 6.40 and 6.41) and the bronchi and carina (Figs 6.42 and 6.43).

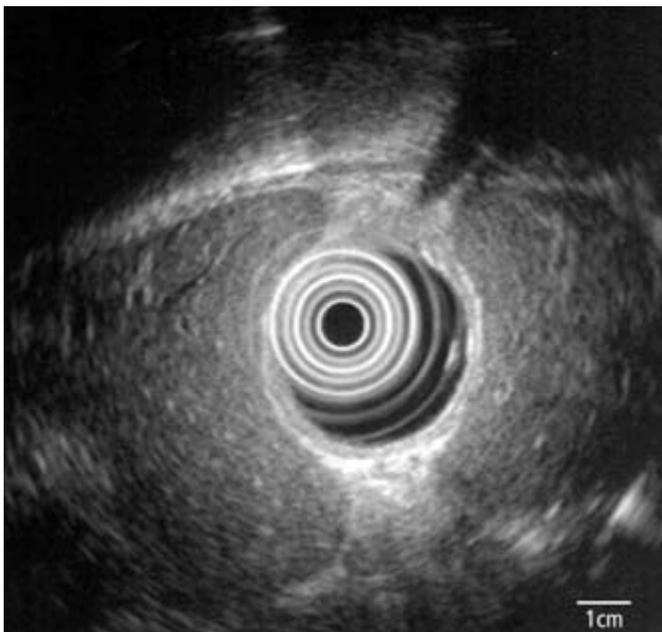


6.37

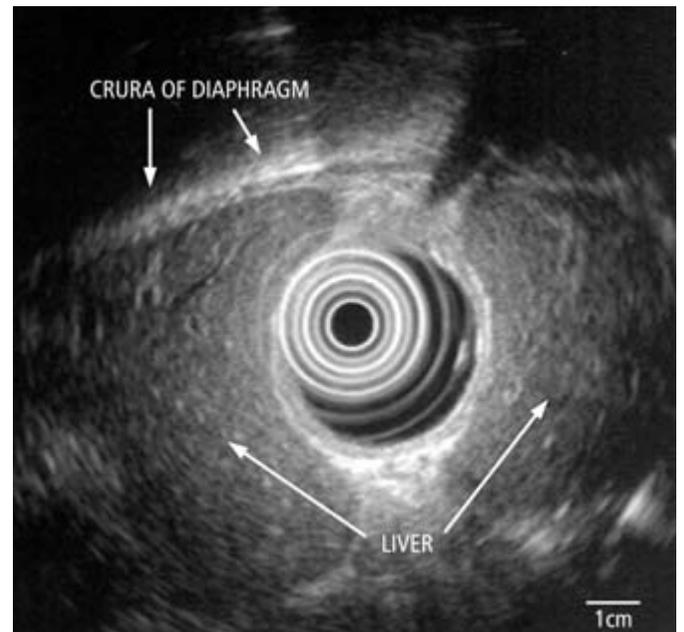


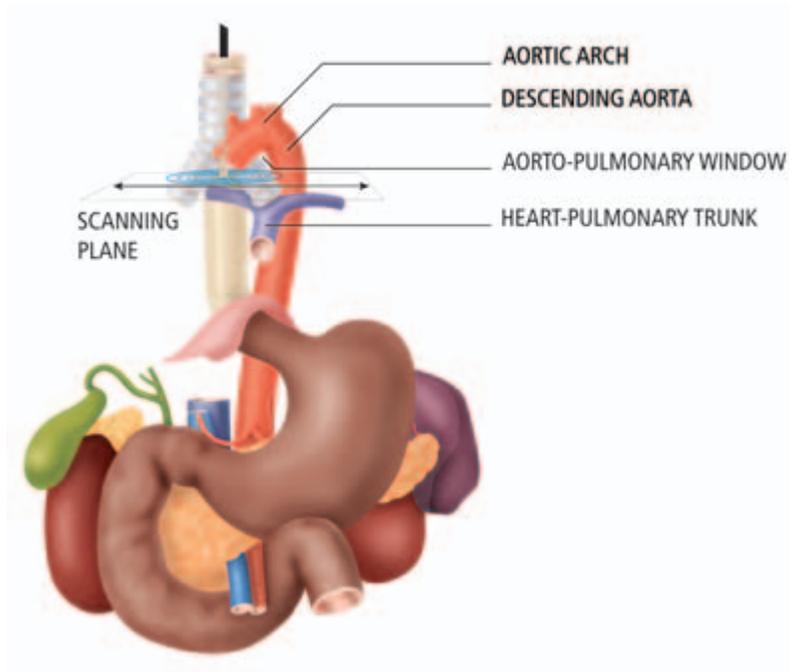


6.38

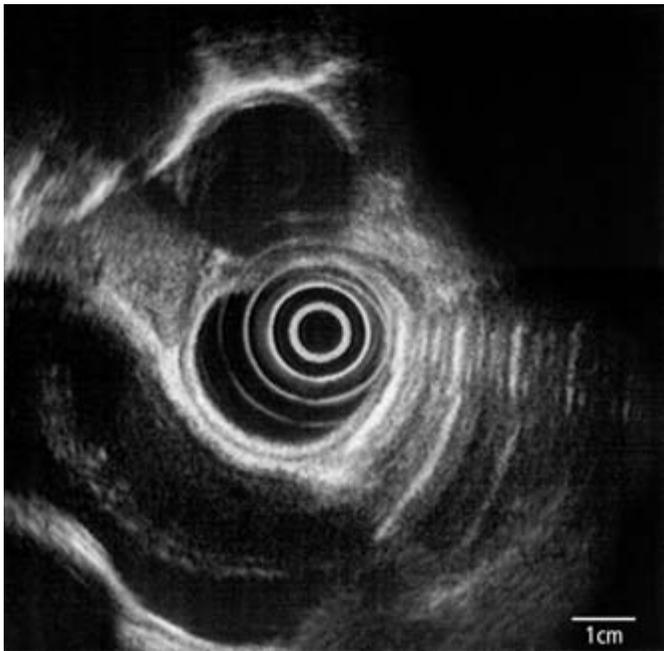


6.39

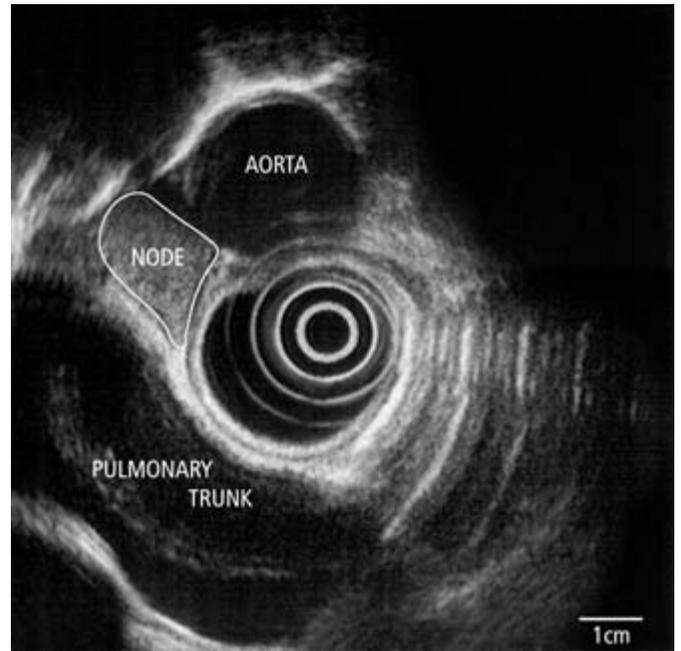


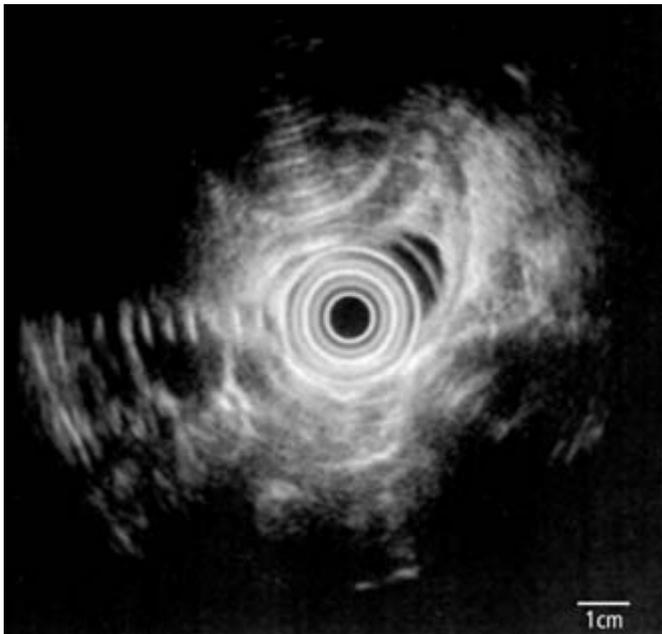
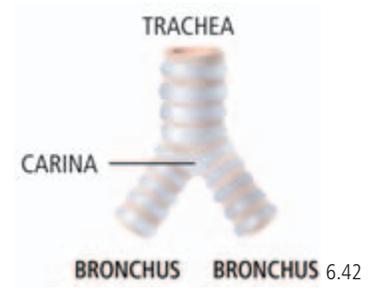
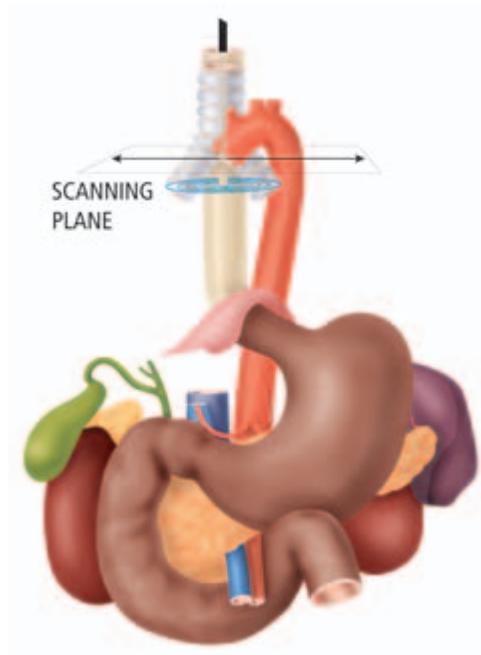


6.40

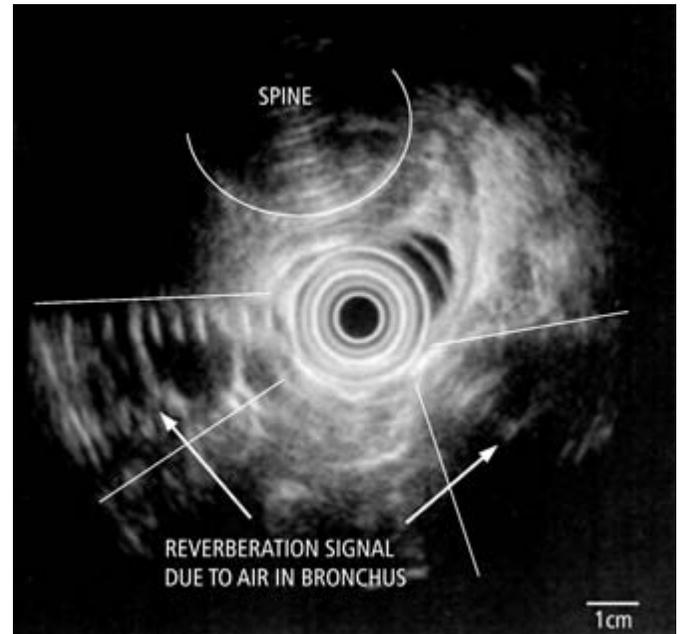


6.41



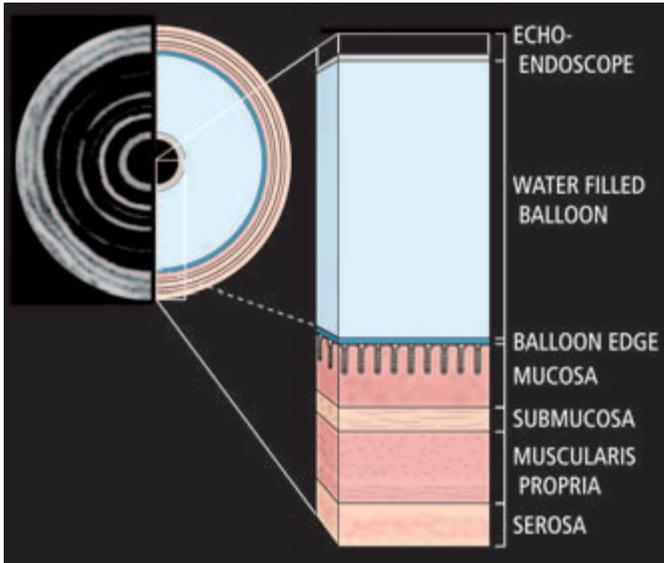


6.43

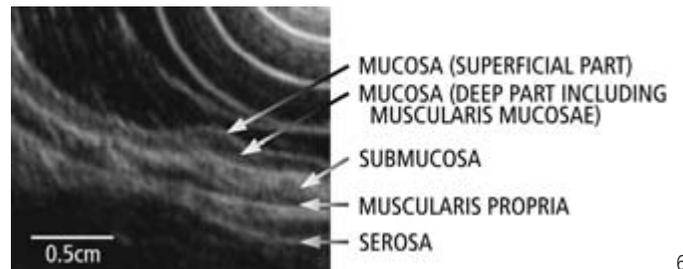


### Mural anatomy of the gastrointestinal tract

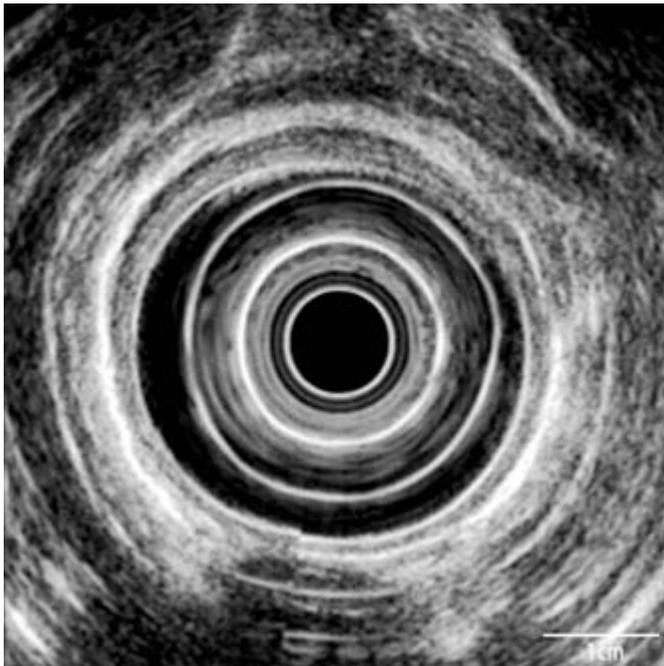
The mural anatomy of the gastrointestinal tract is usually examined with a 12–20 MHz probe. The gastrointestinal wall consists of five distinct echo-layers, which roughly correspond to mucosa (inner two layers), submucosa, muscularis propria (usually one layer; two layers visualized with higher frequency) and adventitia/serosa (Figs 6.2 and 6.44–6.47). Figure 6.45 shows mural detail of the oesophagus scanned at 7.5 MHz, and Fig. 6.46 the results of screening at 12 MHz. The normal EUS layer structure of the gastric wall (12 MHz) appears in Fig. 6.47.



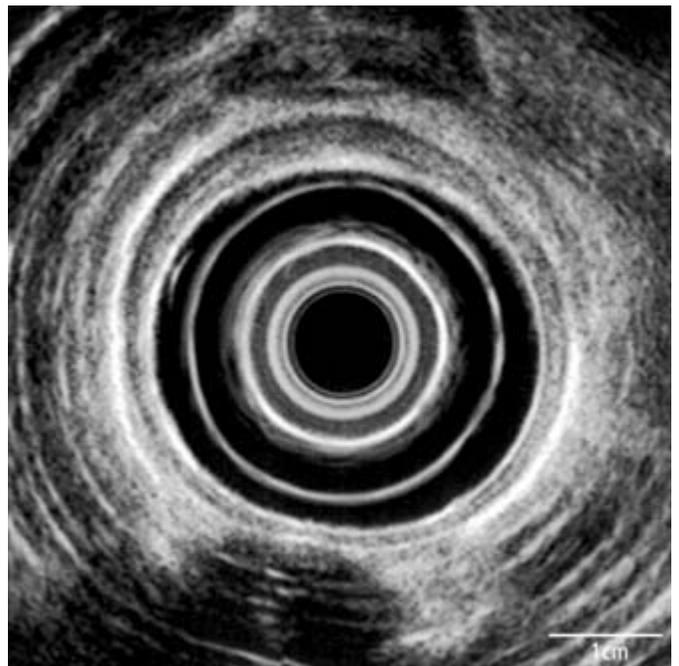
6.44



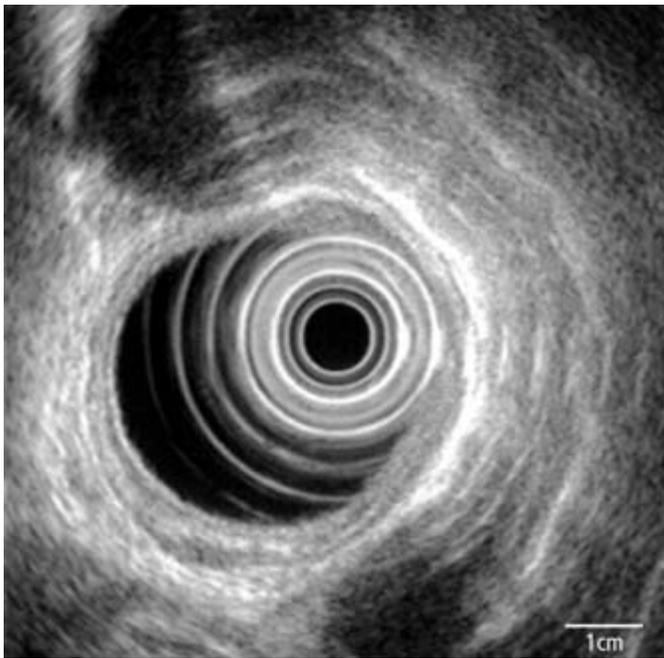
6.47



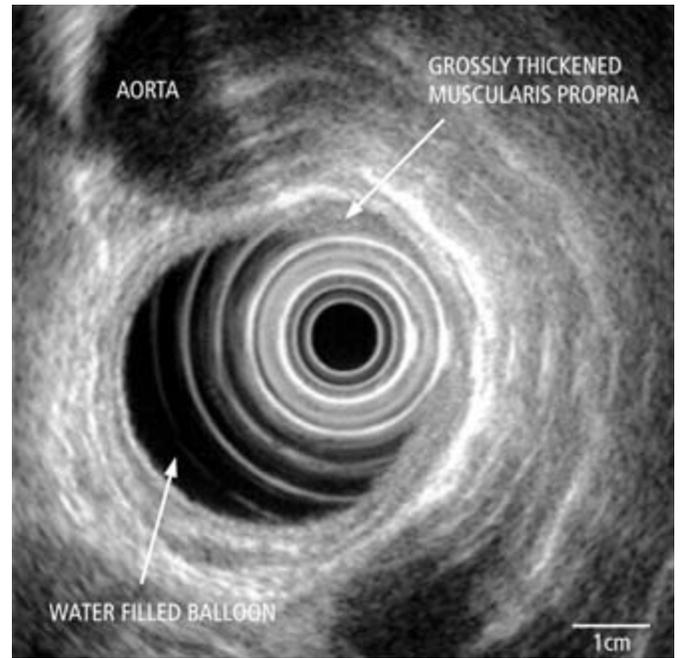
6.45



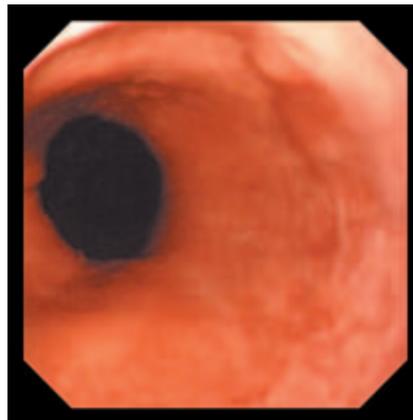
6.46



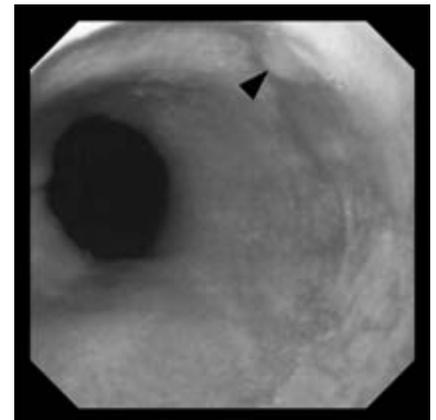
6.48



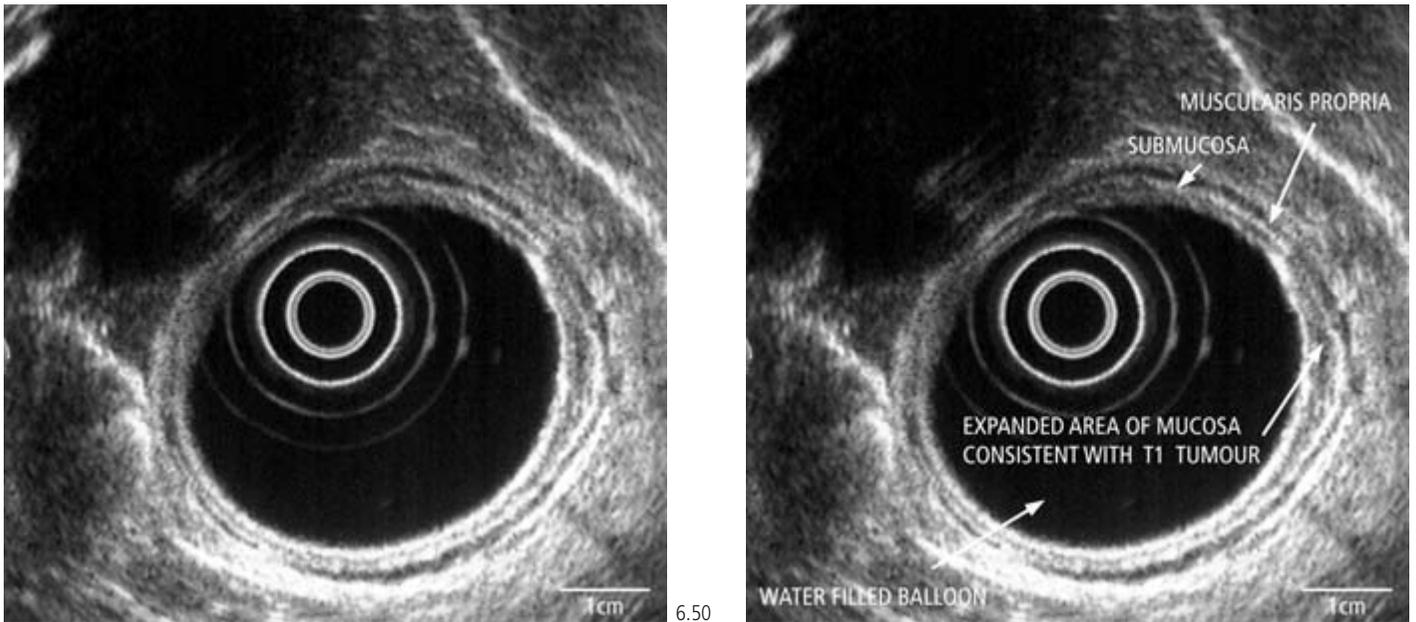
Thickening of the layers in the oesophagus may, for example, be seen in achalasia where the outer hypoechoic layer (muscularis propria) is thickened (Fig. 6.48), or Barrett's oesophagus with some superficial irregularity (Fig. 6.49).



6.49



The inner hypoechoic layer, if thickened, may signify development of carcinoma as in this T1 N0 example (Fig. 6.50).



With high-frequency probes, up to 12 layers have been described, but in reality few of these layers can be seen on a regular basis, and often not involving the whole circumference or length of the segment of the gastrointestinal tract being examined.

## Selected gastrointestinal indications

**Local staging of oesophageal, gastric and colorectal cancer 495**

**Gastric lymphoma and enlarged gastric folds 501**

**Submucosal lesions 504**

**Endoluminal ultrasonography of the colon, rectum and anus 505**

### Local staging of oesophageal, gastric and colorectal cancer

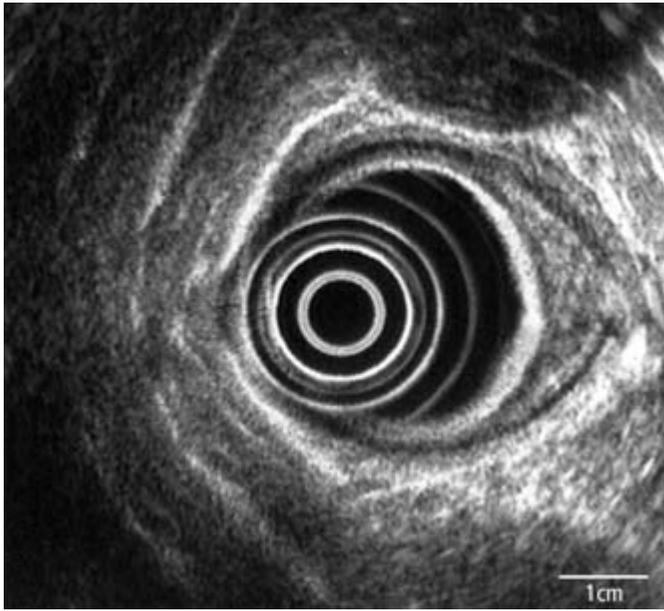
Current oncological practice uses multimodal treatments based on clinical staging. In general, this implies local excision in early cancers, conventional surgery in moderate growths and pretreatment with chemoradiotherapy in more advanced disease. The best example of such a differential approach is in rectal cancer, where T1 lesions can be treated by local excision, T2 and T3 lesions by conventional radical surgery, whereas T4 cancers should undergo prior radiotherapy in an attempt to downstage the tumour for later resection. Such management protocols require accurate preliminary staging, and currently EUS is the best method of assessing local tumour spread. Rectal cancer is a good example of how EUS can influence the choice of treatment. In the large bowel, much experience in the staging of rectal malignancy was gained with a rigid probe passed through a rectoscope. An example is shown in Fig. 6.75. Flexible echoendoscopes combining optical visualization with EUS will become available for use in the lower bowel but are more costly and have less impact clinically in the abdominal part of the colon. Rectal and anal endoluminal ultrasonography are discussed in greater detail on pp. 505–510.

Based on the five-layer structure of the gastrointestinal wall (seven layers in the stomach), EUS can demonstrate the extent to which a tumour has involved the various layers, or has spread further to involve other structures, thus enabling accurate staging. Table 6.1 for example shows how abnormalities demonstrated on EUS examination of the oesophagus relate to the anatomical layer involved, and the T staging in oesophageal carcinoma. A further discussion of the TNM classification (Sobin L.H. & Wittekind C., 1997) is however beyond the scope of this chapter.

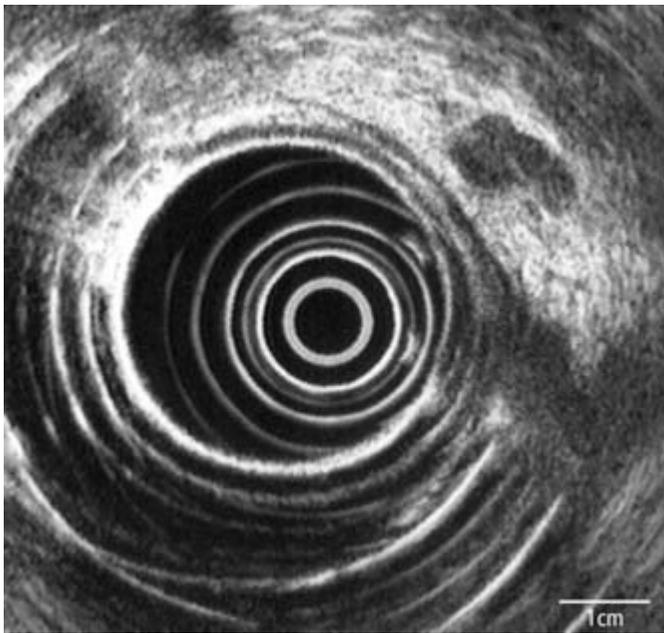
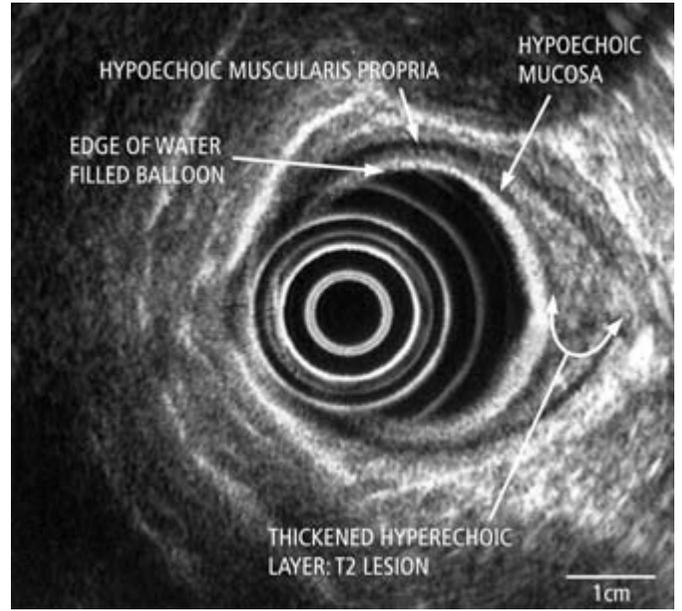
**Table 6.1** Staging for oesophageal carcinoma.

Carcinoma stage	Anatomical layer involved	Abnormality of EUS layer
Tis	Carcinoma <i>in situ</i>	Not seen
T1	Lamina propria, muscularis mucosae and submucosa	Lesion does not extend into outer hypoechoic layer
T2	Muscularis propria	Lesion extends into outer hypoechoic layer
T3	Adventitia	Lesion extends beyond outer hypoechoic margin and through outer hyperechoic layer
T4	Adjacent structures	Full wall involvement and into surrounding tissue and adjacent structures

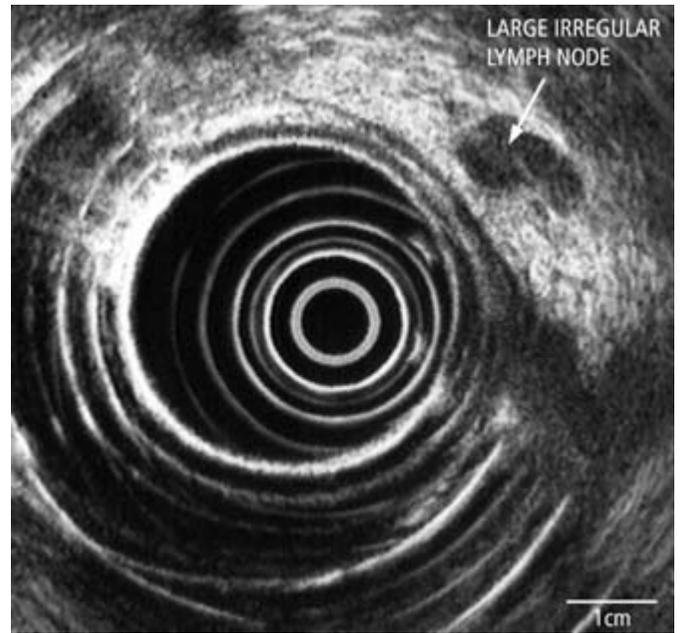
Examples of oesophageal and gastric staging of carcinoma are shown as follows. The thickened middle hyperechoic layer (Fig. 6.51) and enlarged irregular lymph node just below it (Fig. 6.52) staged this lesion at T2 N1.



6.51



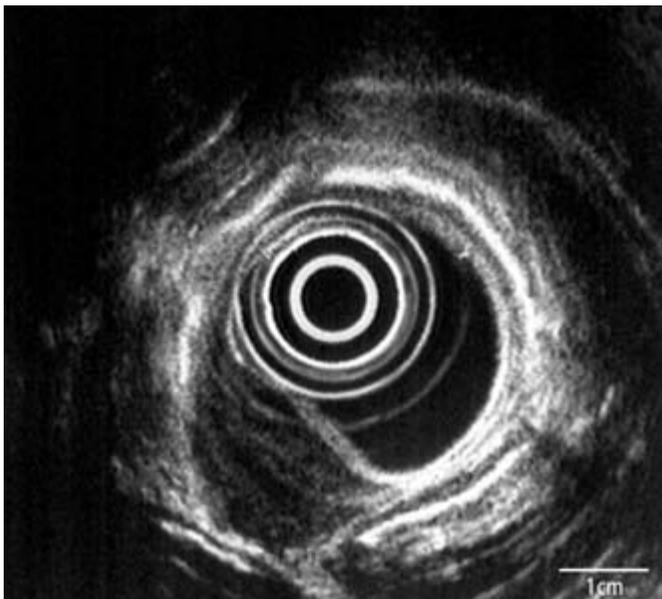
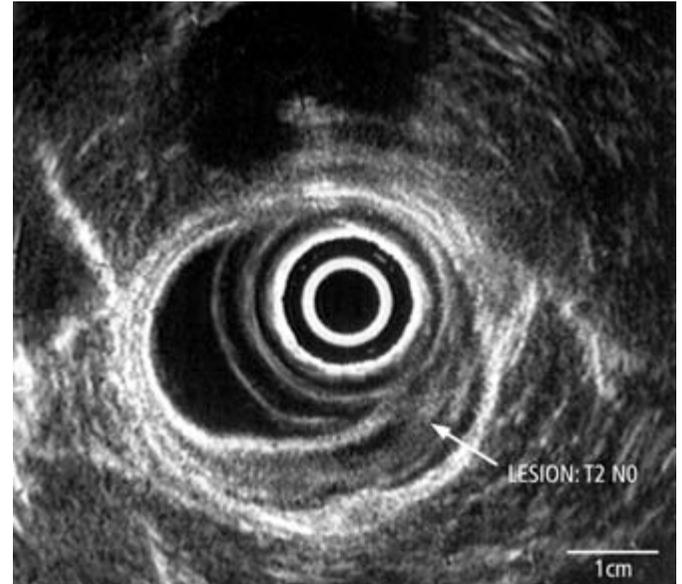
6.52



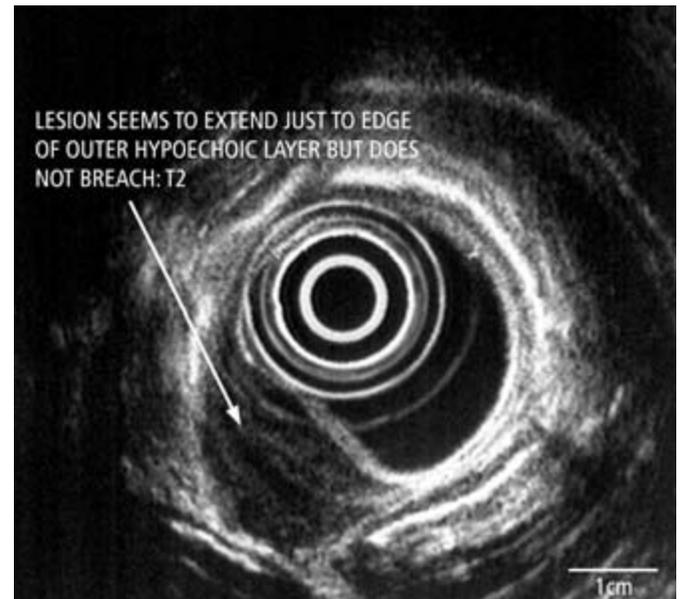
Even though it looks more extensive, the lesion in Fig. 6.53 was not accompanied by lymphadenopathy and is staged as T2 N0. The demonstration of lymph nodes *per se* is not diagnostic of metastatic disease but an increase in lymph node diameter beyond 10 mm is suggestive. (The appearances of lymph nodes detected by EUS are also discussed on pp. 522–523.) The lesion shown in Fig. 6.54 seems to extend just to the edge of the outer hyperechoic layer but does not breach it, and was therefore staged at T2.

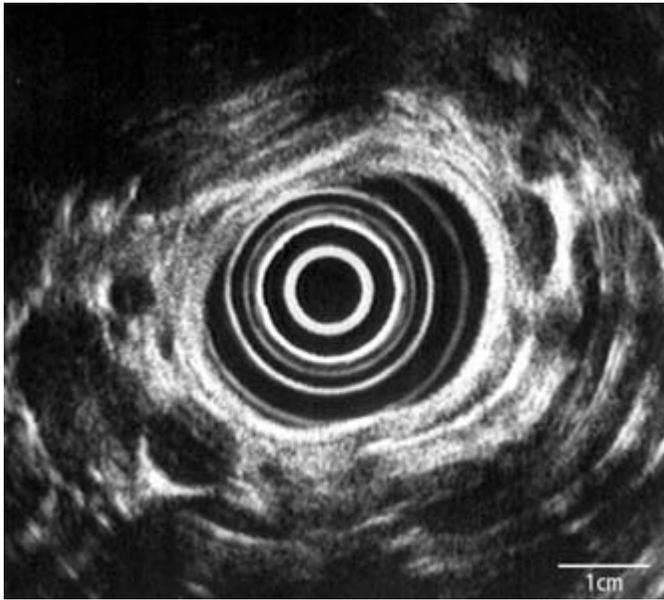


6.53

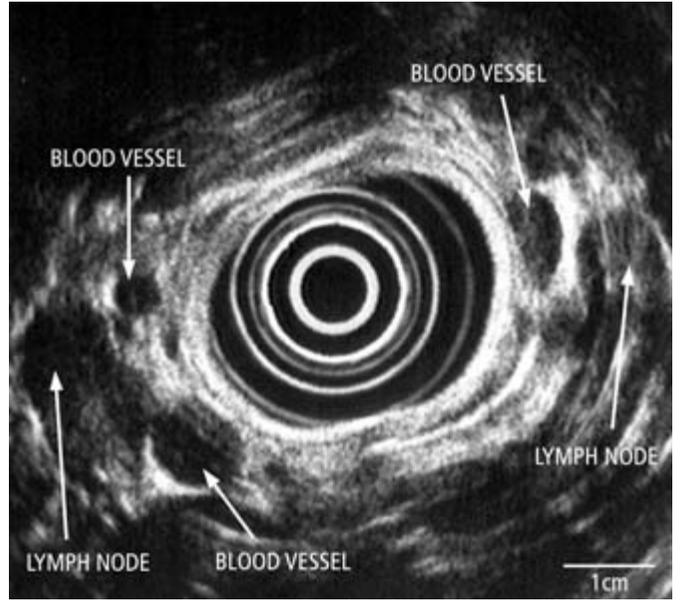


6.54

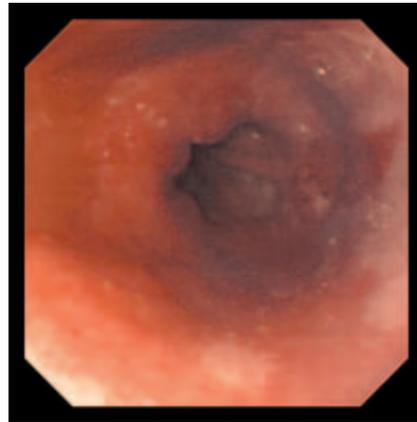




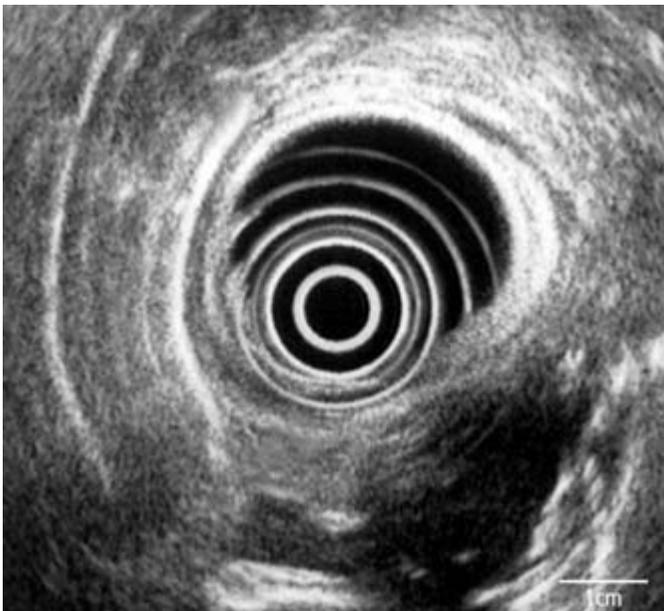
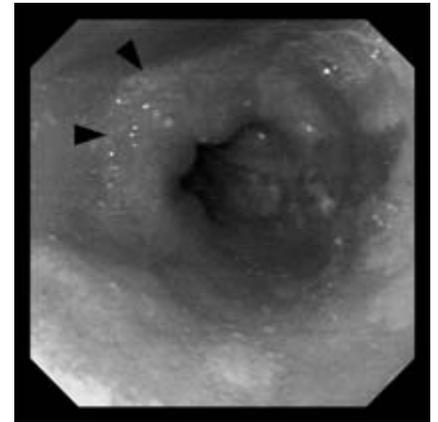
6.55



There were however, large associated lymph nodes (Fig. 6.55), leading to a more complete staging of T2 N1. More advanced tumours cause progressive loss of the outer hypoechoic margins and blurring of the borders with adjacent structures. Figure 6.56 is an endoscopic view of a lesion arising from the mucosa in Barrett's oesophagus which is seen on EUS (Fig. 6.57) to be a T3 tumour.



6.56



6.57

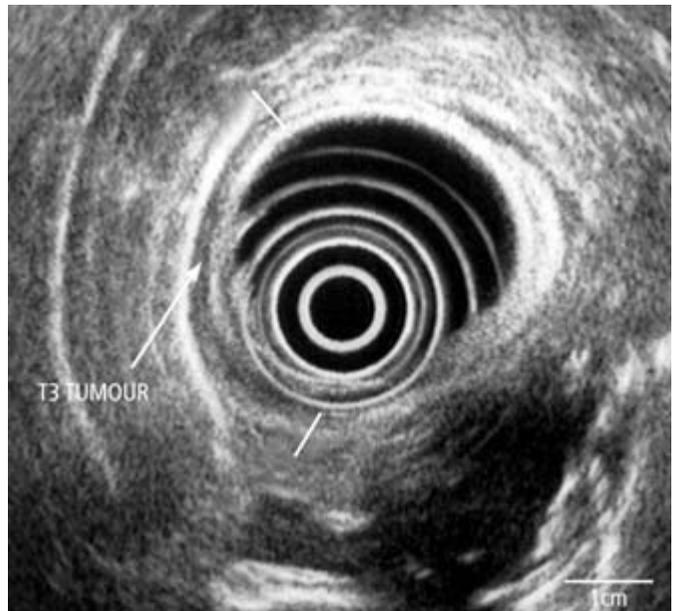


Figure 6.58 shows an extensive tumour with disruption of the outer hyperechoic layer which, however, does not appear to be breached; the lesion was therefore staged at T2.

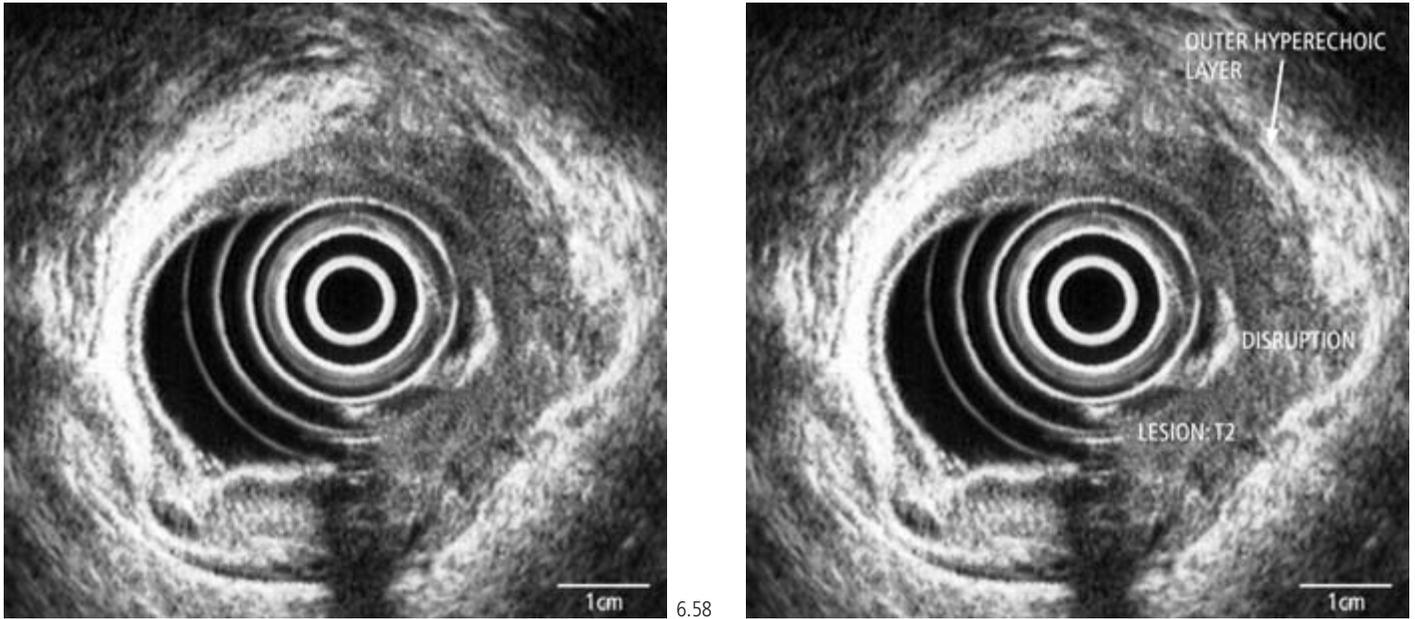
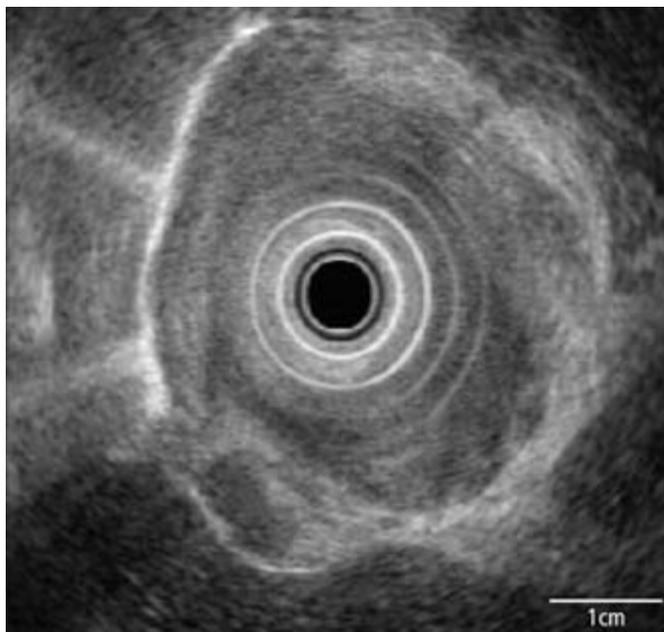
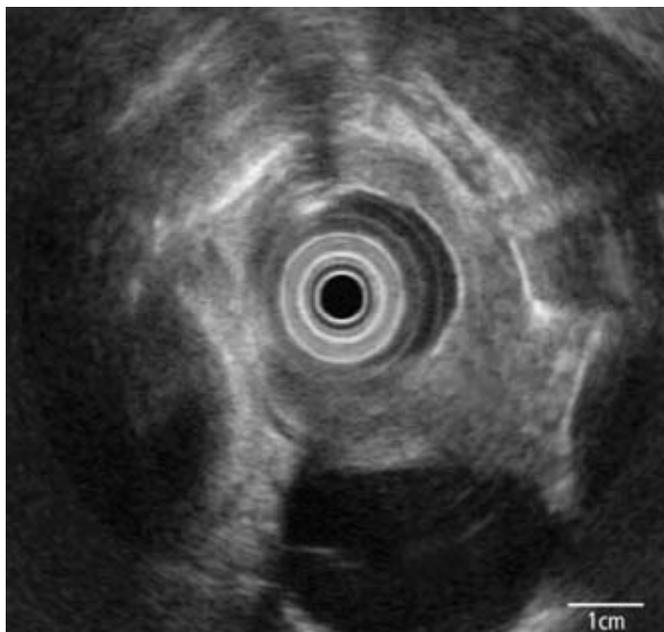
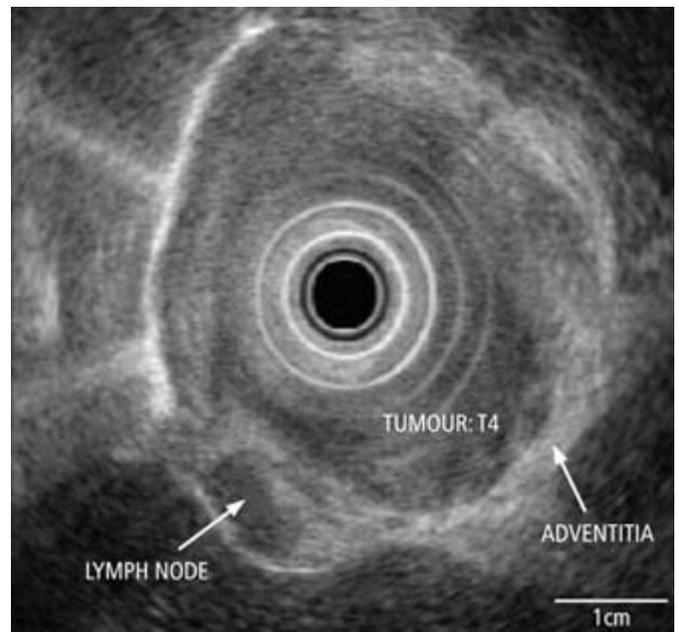


Figure 6.59 shows an oesophageal tumour infiltrating all wall layers circumferentially; in the presence of enlarged paramural lymph nodes the lesion was staged T4 N1. The oesophageal tumour portrayed in Fig. 6.60 has infiltrated the pleura and therefore fulfils the criteria of a T4 lesion. It should be emphasized that differentiating between T3 and T4 lesions by EUS alone can be difficult.

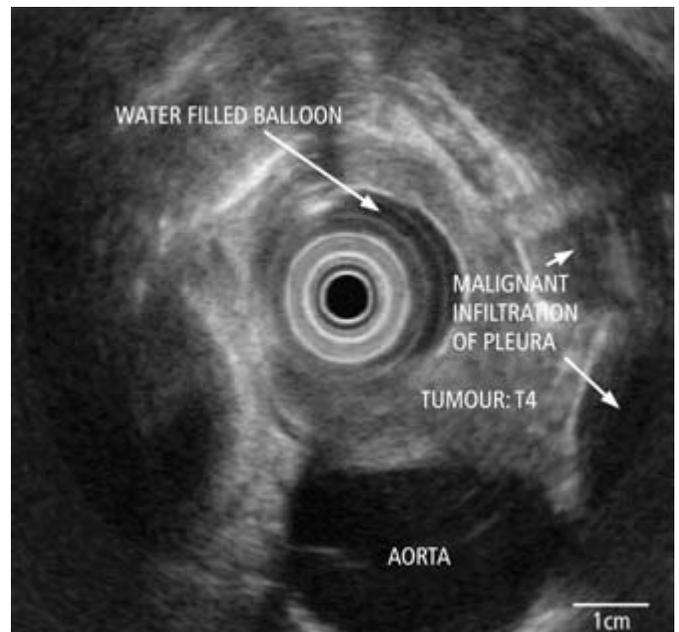
Various studies have demonstrated the higher accuracy of T and N staging based on EUS, compared with CT. *In vitro* studies in cases of oesophageal and rectal cancer have shown that usually only 30% of the lymph nodes present in resection specimens are detected by EUS. 'Blind' subjective and computer-aided objective analysis of those nodes visualized by EUS and appropriately marked in the resection specimen have shown that echo features cannot reliably differentiate benign from malignant nodes, and that there is a considerable overlap in features said to be distinctive. EUS-guided FNAB can be helpful in assessing malignant involvement, and this is discussed on p. 520.



6.59



6.60



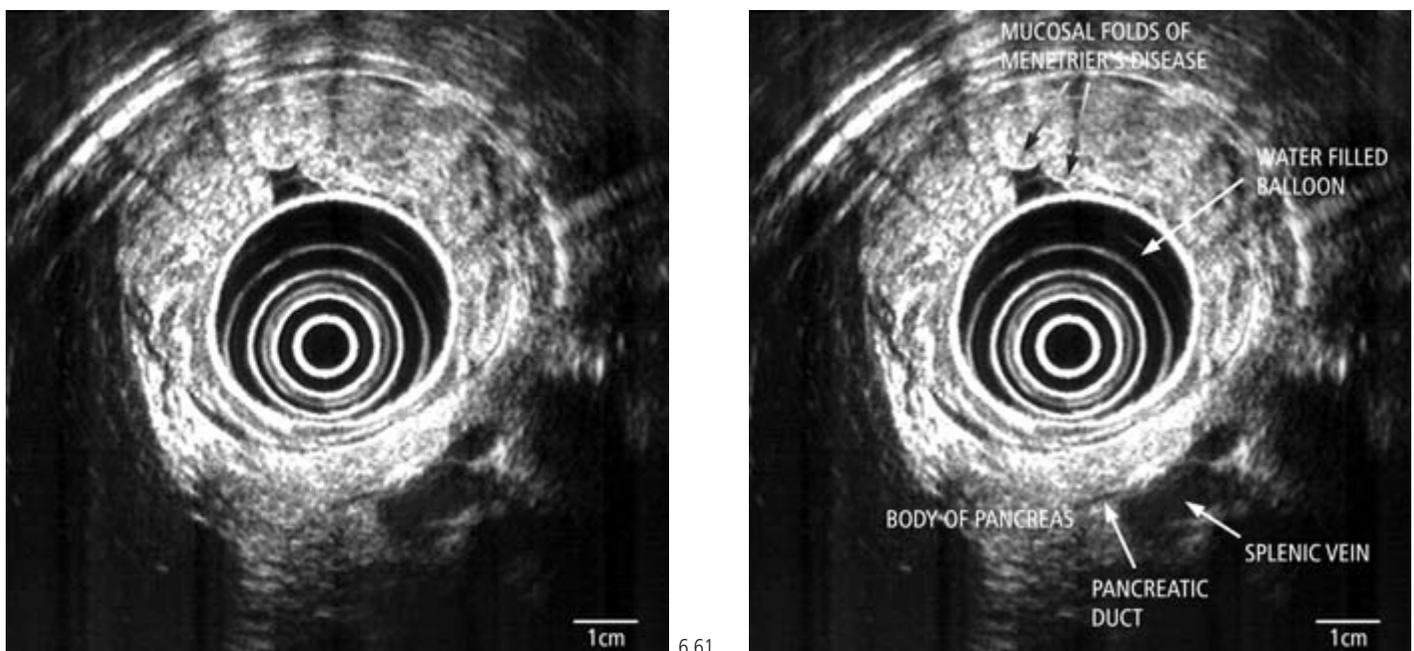
When a malignant oesophageal stricture is present, EUS is of limited value if the stricture cannot be passed, which occurs in approximately 30% of cases. However, in over 85% of such cases, the tumour will already have reached stage T3 or T4. When detailed staging is considered clinically important, EUS assessment after dilatation, or the use of a smaller 'blind' probe of 7 mm diameter (miniprobe), should be considered (Fig. 6.12). (A miniprobe is a narrow diameter instrument carrying a small ultrasonic transducer at its tip; there are no optical facilities, hence the descriptive term 'blind'.) Some studies have suggested that if the echoendoscope will not pass without dilatation of the oesophageal stricture the lesion is likely to be T3 N1.

Many studies have indicated that EUS is almost 90% accurate for staging oesophageal tumours with a 15% tendency to overstage T1 and T2 (which tend to be understaged or missed by CT). It may also understage 5–10% of T2–T4 lesions. Differential staging becomes particularly important when protocols are used to help decide on the most appropriate further treatment, for example palliative local therapy, or chemoradiotherapy with or without surgery. Biopsy with linear array echoendoscopes is likely to become most useful in surveillance programmes such as for patients with Barrett's oesophagus or tylosis. For the stomach, EUS is not as accurate for local tumour staging (overall accuracy just under 80%) as in the oesophagus and has particular difficulty in differentiating T2 and T3 lesions. However, in studies comparing EUS directly with CT or other techniques, so far it is consistently superior for all degrees of severity except stage T4 and M (presence of metastases).

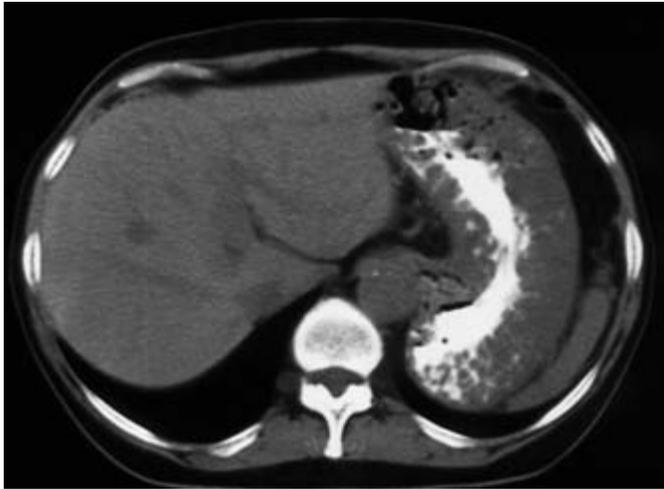
The role of EUS in restaging immediately after chemoradiotherapy and its value in the early diagnosis of anastomotic recurrences seems to be limited. Its use following local laser or photodynamic therapy and following mucosectomy is being evaluated.

### Gastric lymphoma and enlarged gastric folds

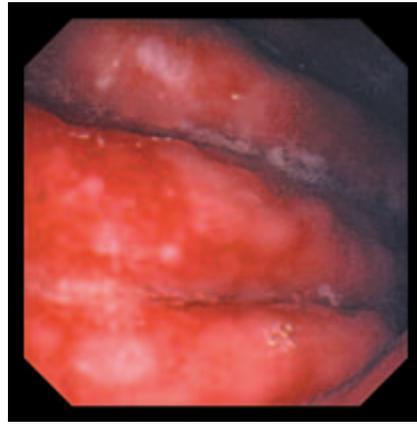
When gastric folds are enlarged, EUS can demonstrate which wall layers are causing the thickening and whether or not the normal layer structure is preserved. Thickening of the mucosa is found in various types of gastritis, Ménétrier's disease and some patients with early lymphoma. The EUS appearances of Ménétrier's disease are shown in Fig. 6.61.



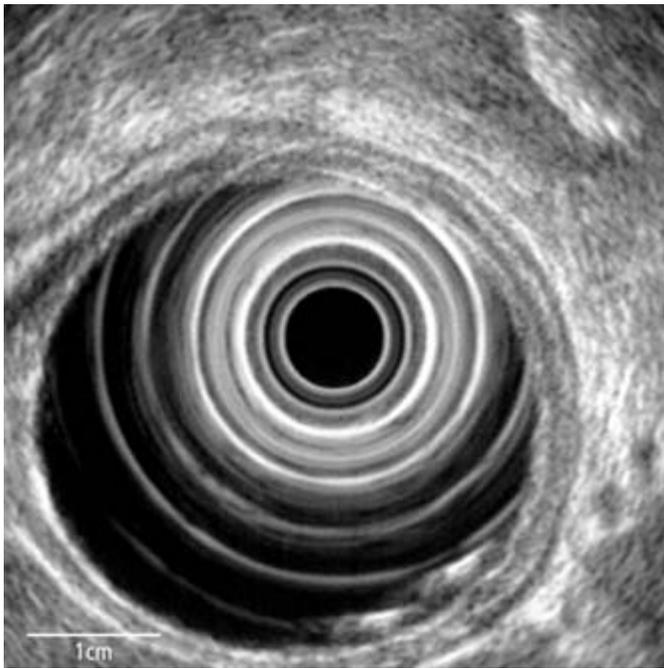
A CT image of this condition is shown in Fig. 6.62 and the endoscopic appearances in Fig. 2.456. In another patient endoscopy suggested thickened mucosal folds (Fig. 6.63) but on EUS (Fig. 6.64) all the layers in the stomach were normal; biopsies showed the appearances of a MALT lymphoma.



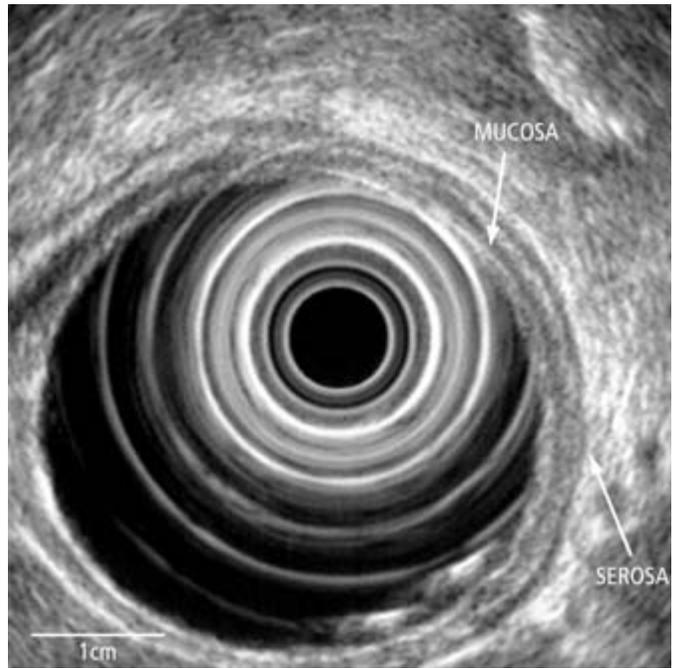
6.62



6.63

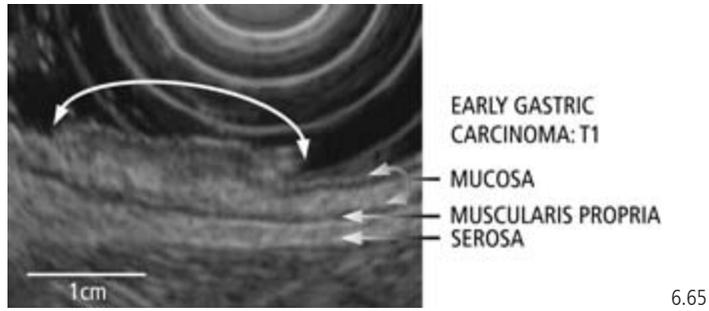


6.64

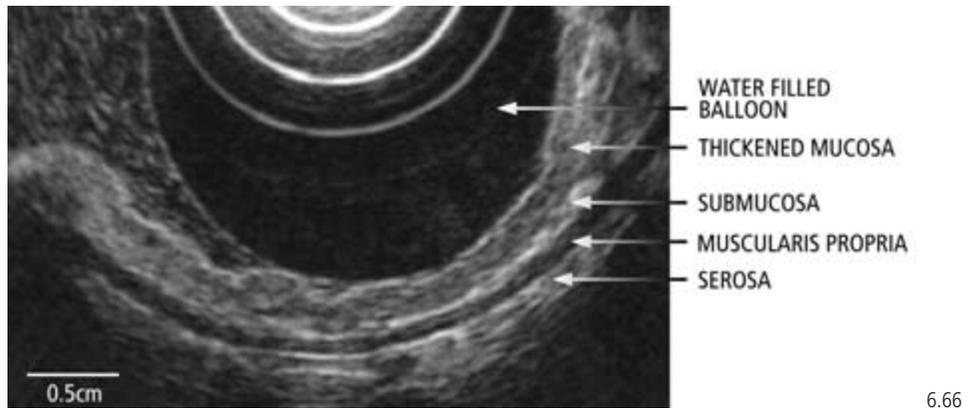


By contrast in Fig. 6.65 there is an early gastric carcinoma characterized by localized malignant infiltration of the mucosa and submucosa (first three layers) with preservation of the intactness of the muscularis propria, therefore staged as T1.

Generalized thickening with loss of the layer structure suggests malignancy, such as advanced lymphoma or linitis plastica. However, a reliable diagnosis cannot usually be made on the basis of EUS alone, and it does not replace histopathological assessment. EUS is of help in suggesting the diagnosis and prompting more aggressive procedures such as mucosal snare biopsy (see p. 332). If EUS shows the presence of large intramural vessels as the cause of the wall thickening, large particle or snare biopsy procedures are clearly contraindicated.



In biopsy-proven gastric lymphoma, EUS may be used for local staging to delineate tumour growth through the wall and to diagnose lymph node involvement. The value of EUS in primary staging, in monitoring the effects of radio- or chemotherapy and in assessing the effects of *H. pylori* eradication on gastric wall thickness in early gastric lymphoma (Fig. 6.66) is currently under intensive study. There is marked thickening of the second layer of the mucosa in Fig. 6.66.

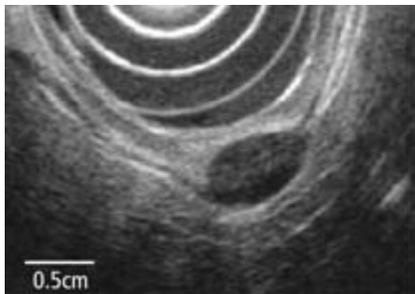


## Submucosal lesions

In bulging appearances of the upper gastrointestinal tract, endoscopy often cannot reliably distinguish between extramural impressions caused by normal or pathological structures and true (intramural) submucosal tumours nor can it determine their pathological nature. Such differentiation is, however, of great diagnostic and therapeutic importance, and EUS has repeatedly been shown to be highly accurate in resolving such dilemmas. EUS also shows size, margins, echopattern and layer of origin of a submucosal tumour. This information can be used to suggest the possible nature of such a tumour, and whether it is more likely, for example, to be a leiomyoma (gastrointestinal stromal tumour or GIST) or a lipoma. Figure 6.67 shows the EUS appearances of an oesophageal GIST; note the hypoechoic tumour has arisen from the fourth layer of the wall. A typical prepyloric GIST appears in Fig. 6.68 while Fig. 6.69 represents the EUS features of such a lesion.

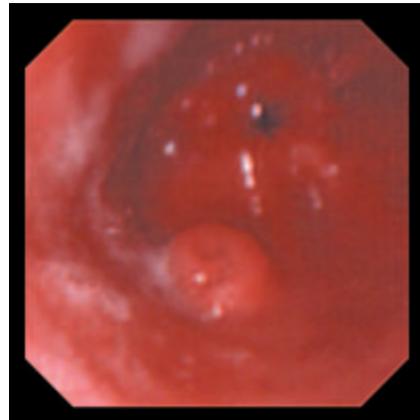
Firm differentiation between benign and malignant submucosal tumours cannot be made by the EUS appearance alone, although it is the best method of delineating risk criteria such as size, margin, and lymph nodes in assessment of the likelihood of malignancy.

EUS-guided fine needle aspiration cytology as discussed below may be helpful in evaluating these lesions but further evidence is required.

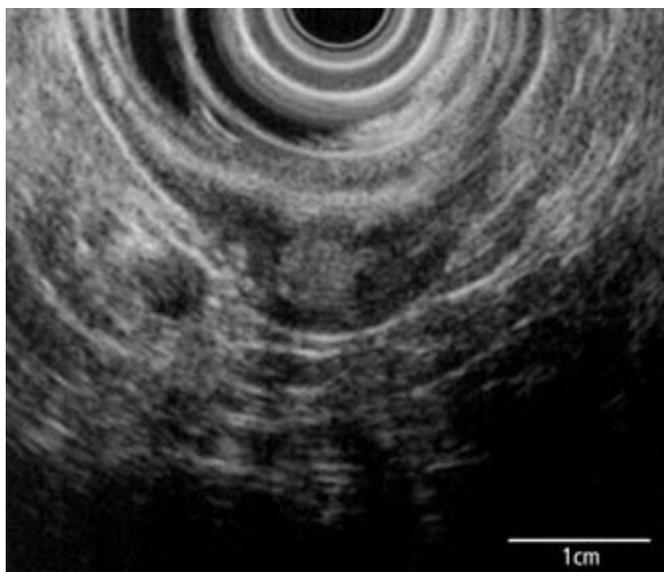


GASTROINTESTINAL  
STROMAL TUMOUR  
(GIST)

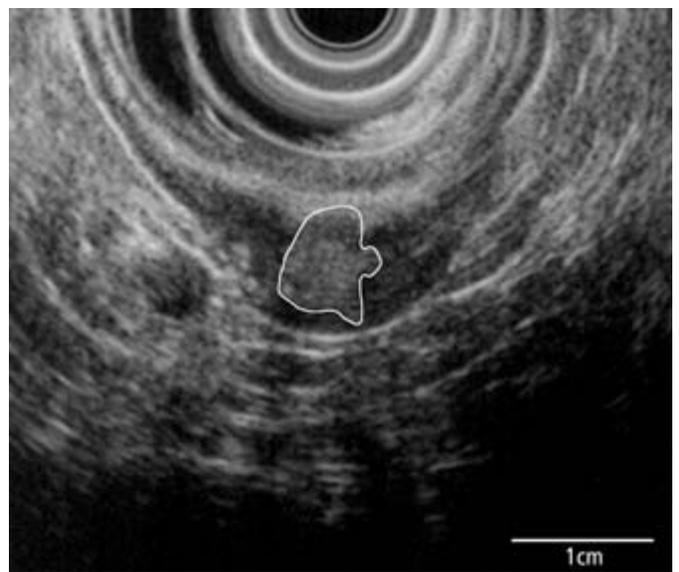
6.67



6.68



6.69



## Endoluminal ultrasonography of the colon, rectum and anus

The earliest endoluminal ultrasound examinations of the lower gastrointestinal tract were carried out through the anal canal, when it became clear that probes used to image the prostate across the rectal wall could also be helpful in looking at the rectal wall itself. At first 5 MHz, then 7 MHz and now 10 MHz probes mounted on a rigid rotating motor can be passed blind into the anal canal or via a rectoscope to the area of interest in the rectum. Although colonoscopes have been fitted with rotating transducers, such instruments are much more expensive than rigid probes and their clinical value has not yet been established. Most polyps in the colon will be snared and retrieved or resected, irrespective of staging, whereas in the rectum important clinical decisions may be informed by accurate imaging and are accessible to a rigid transducer. Flexible endoscopic echoprobes may have a role in determining the nature of strictures and perhaps increasingly in the future with technological refinements of local excision within the intra-abdominal colon.

### Anus

A 10-MHz Bruel and Kjaer transducer and probe with a sonolucent plastic water-filled nose cone is the most widely used equipment. The rectum need not be prepared but a small enema is usually given beforehand. The probe is lubricated for insertion. The anal sphincter is scanned from above downwards and representative images taken from the upper, mid and lower anal canal. A typical normal anal scan is shown in Fig. 6.70. The internal anal sphincter appears as an inner dark ring while the brighter outer ring is the external anal sphincter.

The principal indications for anal endoluminal ultrasonography are:

- incontinence, assessing the effect of obstetric injury or previous surgery;
- fistula, defining the anatomy of tracks using hydrogen peroxide bubble enhancement;
- anal cancer.

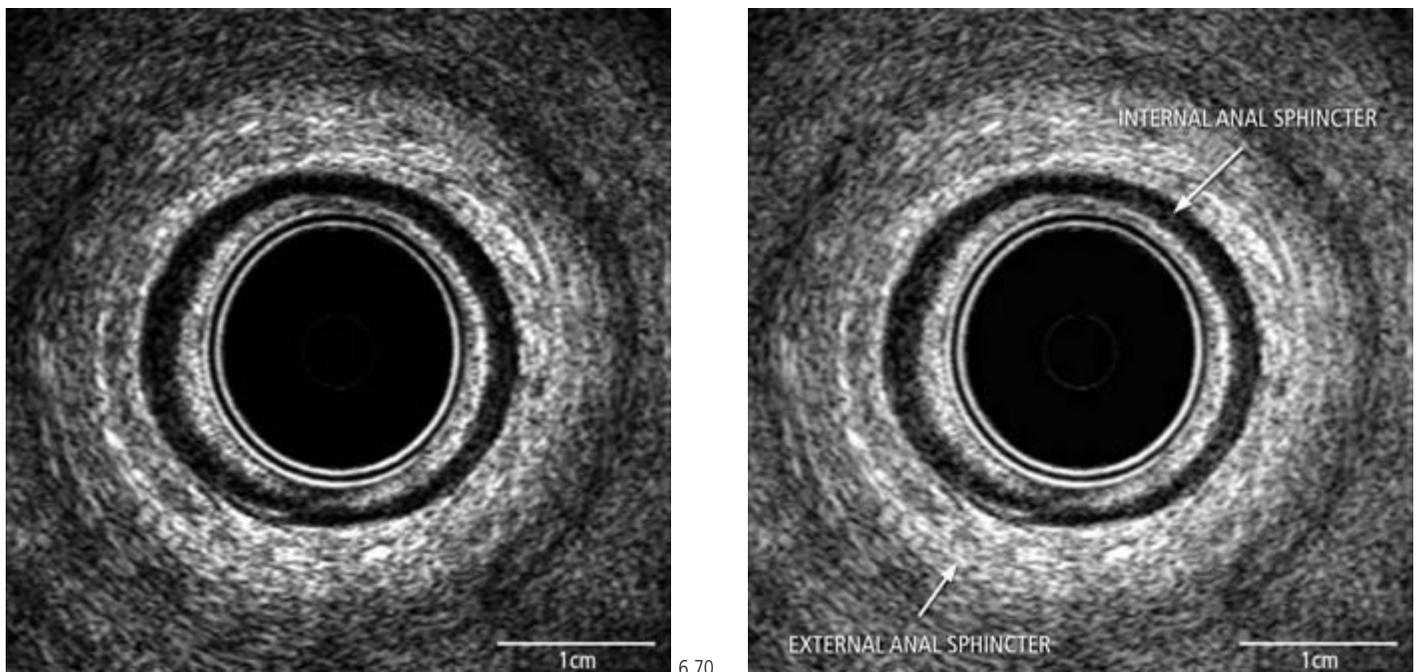
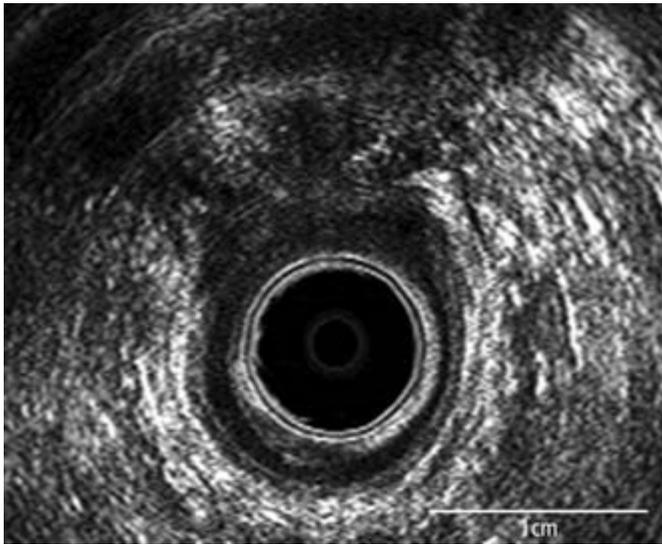
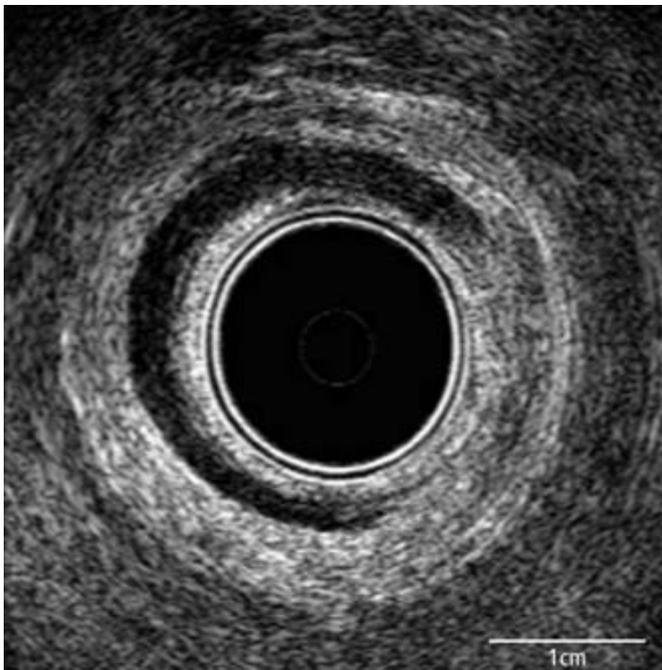
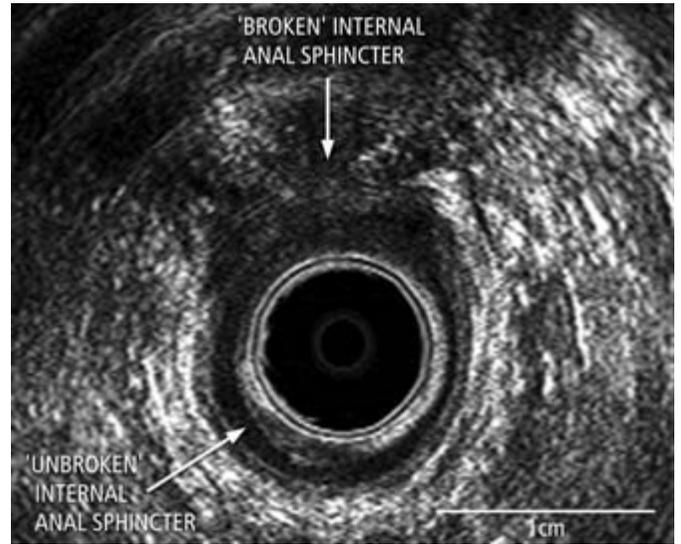


Figure 6.71 illustrates an obstetric injury to the external anal sphincter. The bright ring is 'broken' anteriorly and a scar shown as a dark mixed echo. The underlying internal anal sphincter is undamaged. The results of sphincterotomy appear in Fig. 6.72: there is a well-marked gap in the internal anal sphincter.

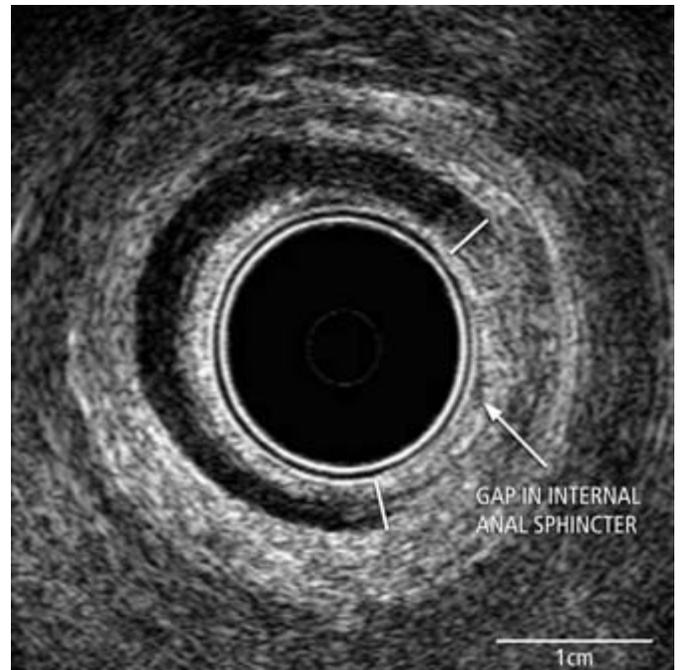
In the female, the sphincter complex is shorter anteriorly than posteriorly and this can give rise to diagnostic error.



6.71



6.72

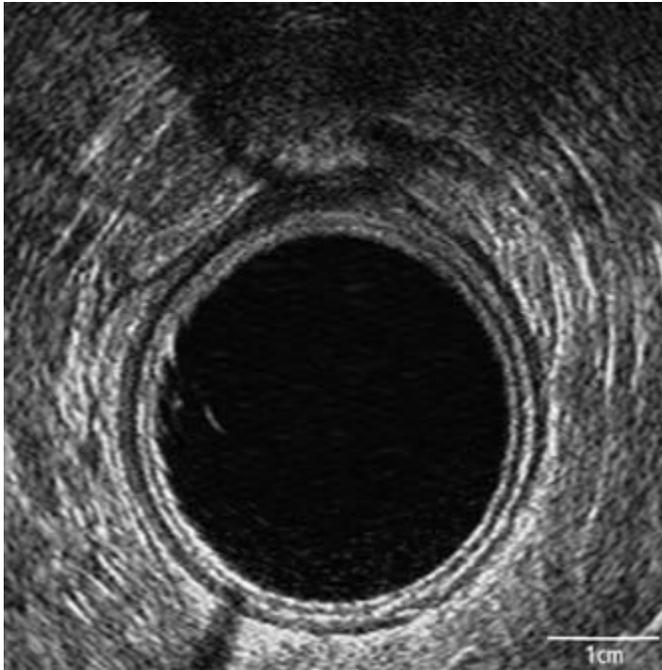


## Rectum

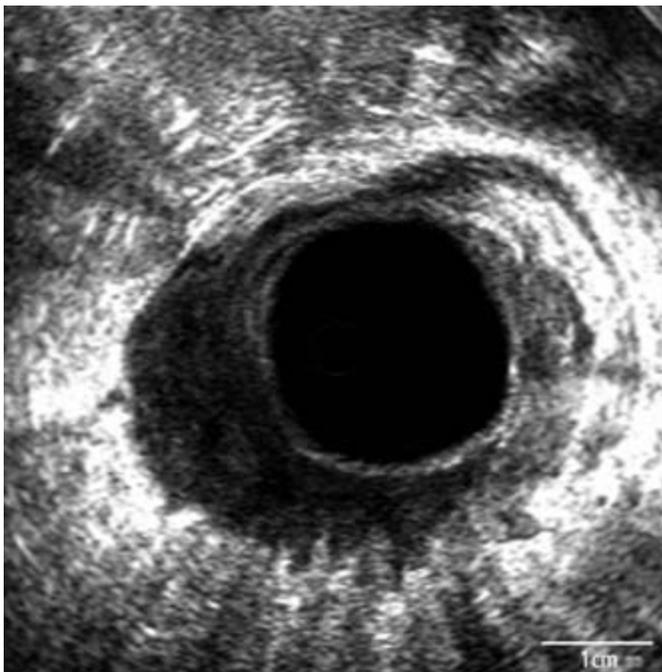
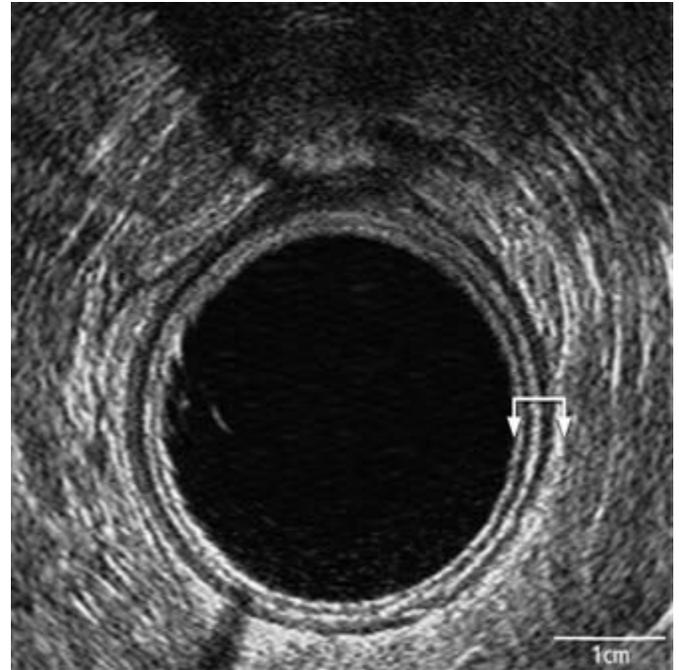
A 10-MHz Bruel and Kjaer rigid probe is the most widely used. A water-filled balloon is inflated around the transducer to ensure good acoustic contact. The rectum needs to be very well prepared. The transducer is placed above the cancer or polyp via a rigid rectoscope which can then be partially withdrawn. As with all endoluminal

ultrasound examinations the volume in the balloon is varied to give the best images. There may be problems negotiating rigid narrowed cancers. The same sonographic anatomy is seen in the rectum as elsewhere in the gut: the typical five layer appearance (Fig. 6.73, rectal ultrasound, 10 MHz) is similar to that described for the upper gastrointestinal tract, e.g. Figure 6.44.

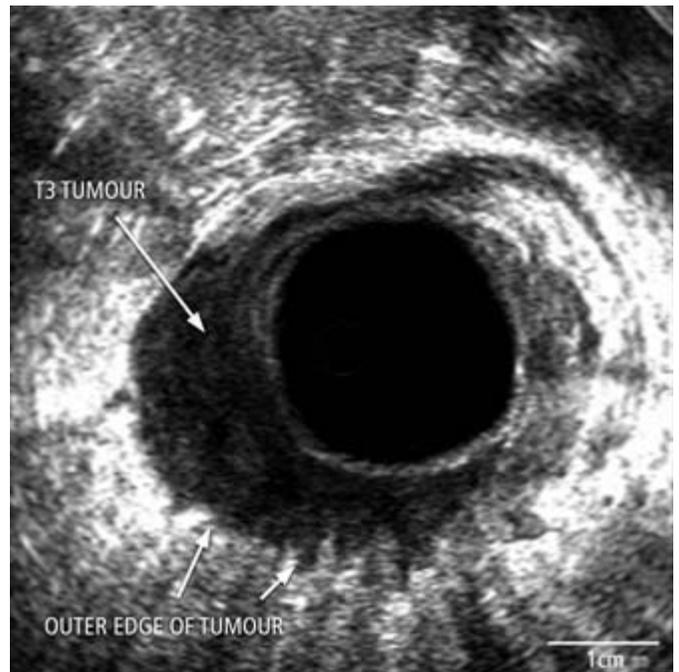
The darkest outside layer representing the muscularis propria is the most important for determining spread of tumours beyond the bowel wall: an example of a T3 cancer is shown in Fig. 6.74. There is an advanced stenotic T3 cancer. None of the usual layers are visible. The outer dark edge has a serrated appearance suggesting infiltration into the mesorectal fat.



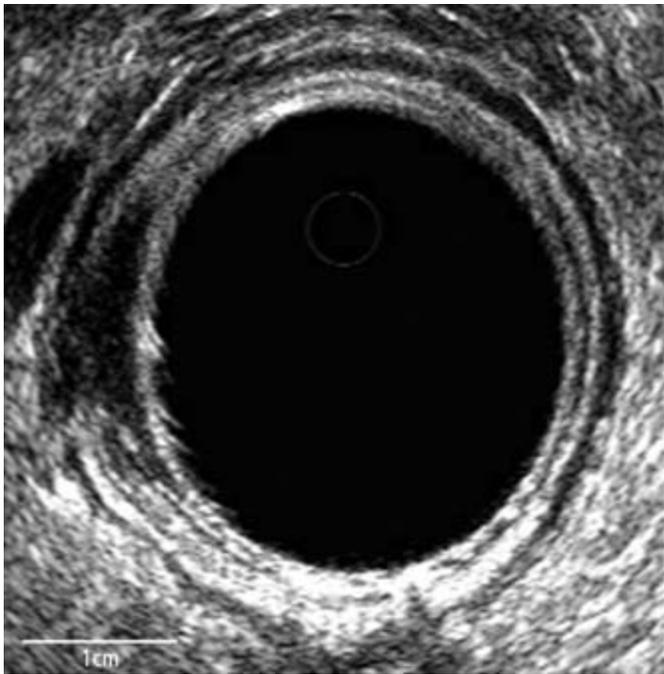
6.73



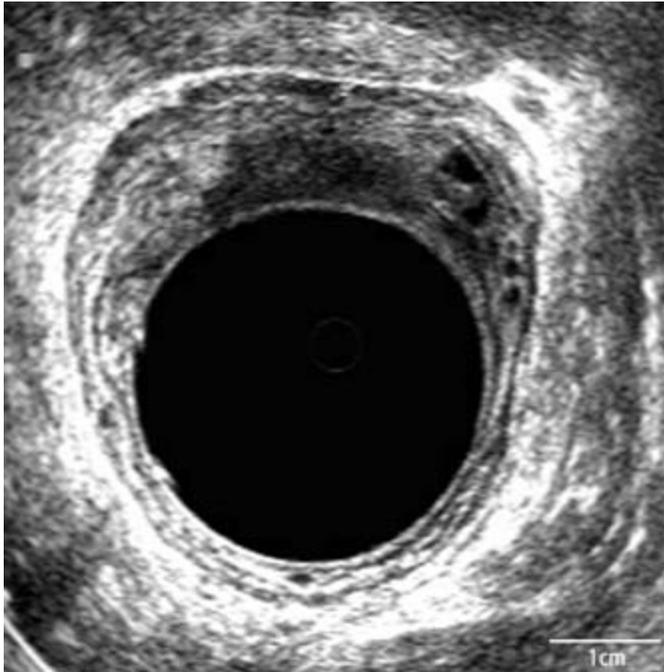
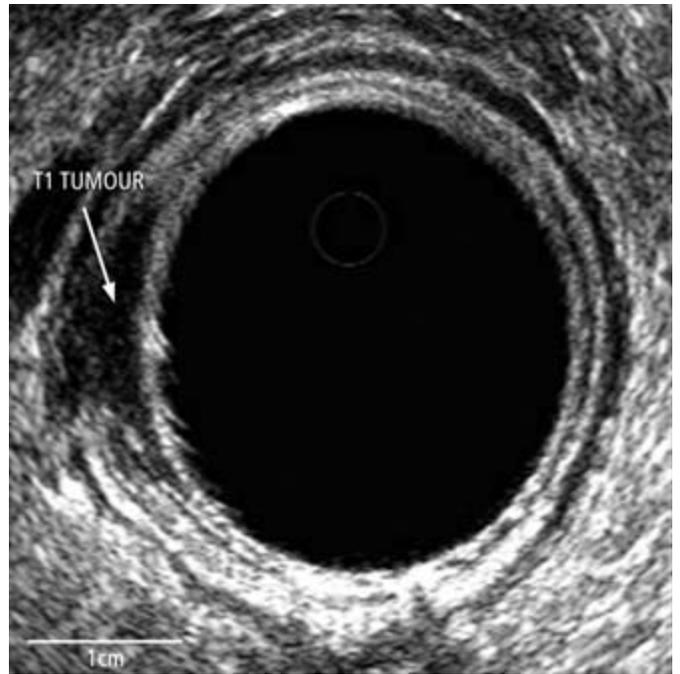
6.74



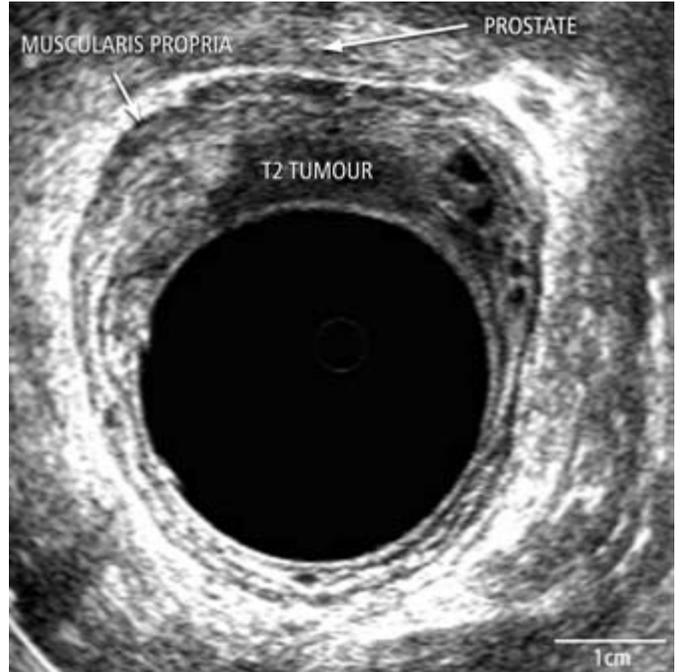
Distinguishing between T1 and T2 cancers can be difficult. In Fig. 6.75 there is a T1 carcinoma. The submucosa is filled out but the muscularis propria is intact. A T2 carcinoma appears in Fig. 6.76: note how the submucosa has been disrupted, and the muscularis propria invaded but not breached. Anteriorly, Denonvilliers' fascia is intact with a clearly separate prostate.



6.75



6.76

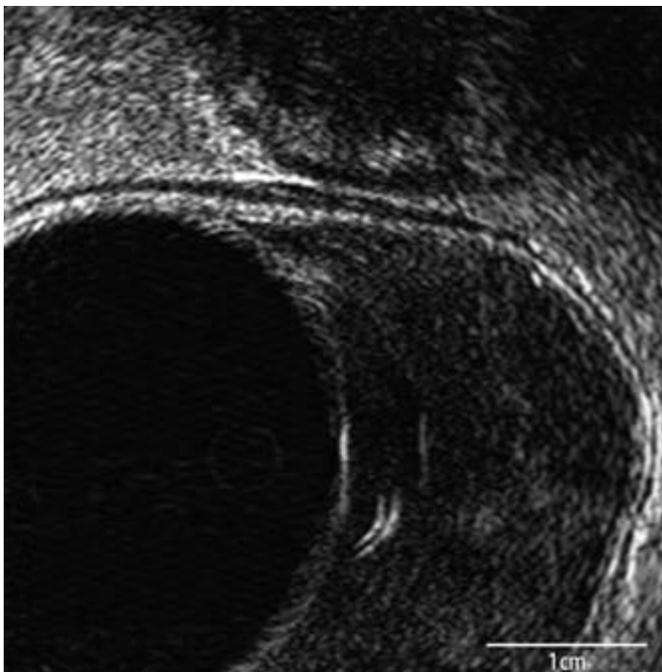


Large villous tumours become overfolded and squashed and may cause diagnostic errors. A benign tumour, in this instance a villous adenoma, is shown in Fig. 6.77. The speckled pattern is typical of villous adenoma and is caused by trapped air bubbles. The submucosal layer is intact. The features of another benign polyp appear in Fig. 6.78; again, the intact submucosal layer is well shown.

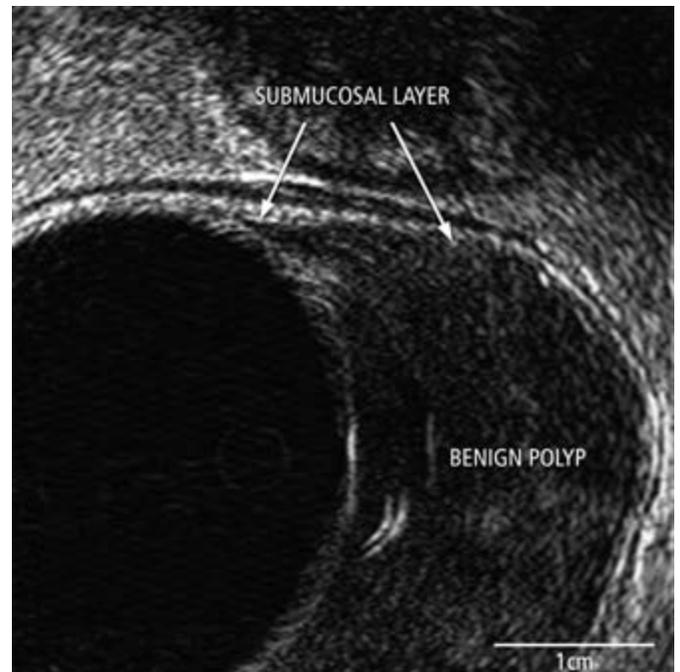
In general, quoted staging accuracy is 80–90% for primary tumours, but this is considerably less when looking at very early T1 invasion.



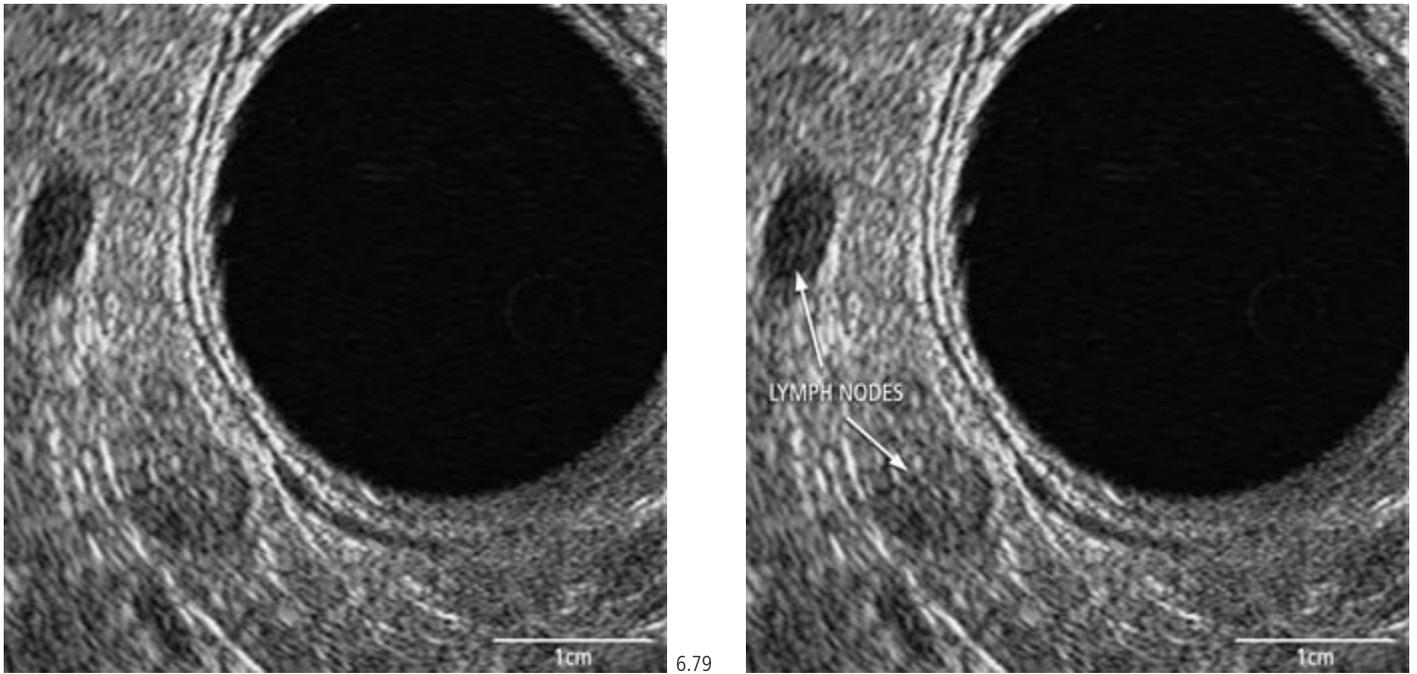
6.77



6.78



Lymph node imaging is reported as being around 70% accurate. Figure 6.79 shows two lymph nodes in the mesorectal fat, one less echodense than the other. The underlying rectal wall at this point shows the normal five layers. Local invasion of surrounding structures is better assessed by MRI.



## Selected indications for pancreatobiliary disorders

**Diagnosis and staging of pancreatic cancer 511**

**Chronic pancreatitis 514**

**Endocrine tumours of the pancreas 517**

**Common bile duct stones 518**

**Ampullary lesions 519**

### Diagnosis and staging of pancreatic cancer

In the diagnosis of pancreatic mass lesions, ERCP, spiral CT and EUS give comparable results. EUS is probably the best method currently available for staging (Table 6.2) and assessment of resectability of pancreatic tumours. EUS is better for the assessment of venous than arterial involvement in a malignant pancreatic lesion.

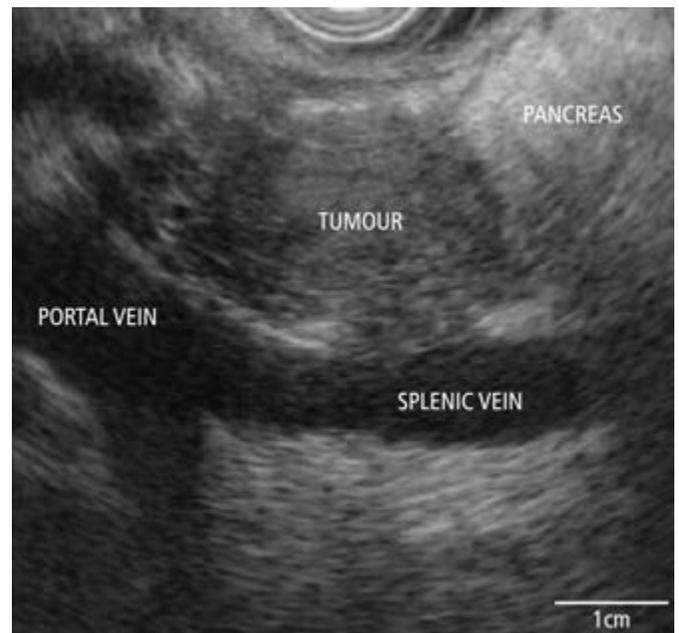
**Table 6.2** Staging of pancreatic carcinoma.

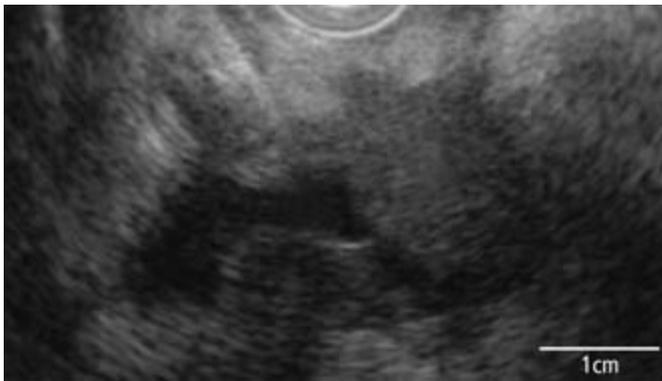
Carcinoma stage	Size, location on EUS
Tis	<i>in situ</i> , not seen
T1	Tumour limited to pancreas; greatest diameter $\leq$ 2cm
T2	Tumour limited to pancreas; greatest diameter $>$ 2cm
T3	Tumour extends into any of the following: duodenum, bile duct, peripancreatic tissues
T4	Tumour extends directly into any of the following: stomach, spleen, colon, adjacent large vessels

EUS shows a pancreatic cancer as having a non-homogeneous and echo-poor pattern with irregular outer margins and pseudopodia, features often associated with malignant tissue (Figs 6.80 and 6.81).

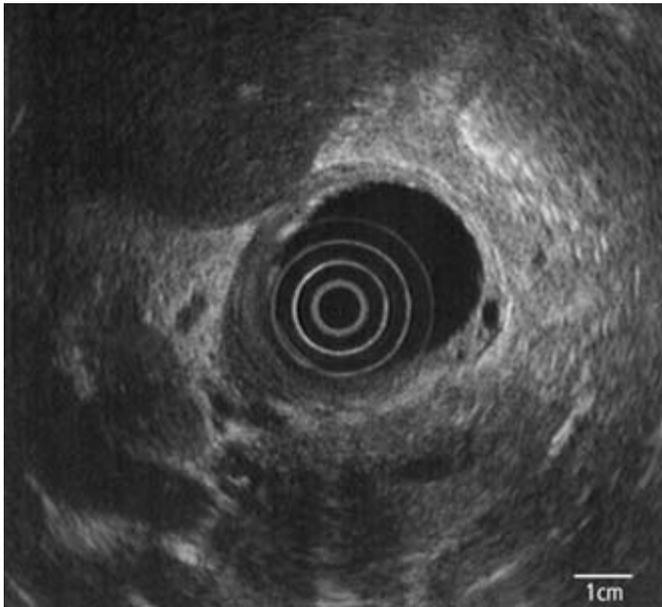
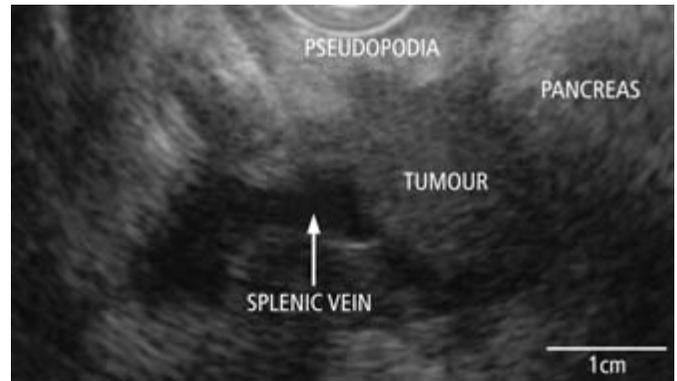


6.80

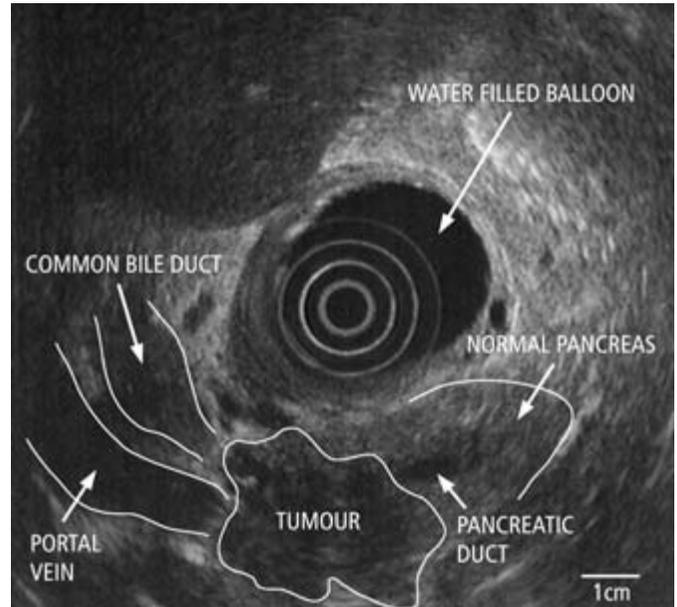




6.81



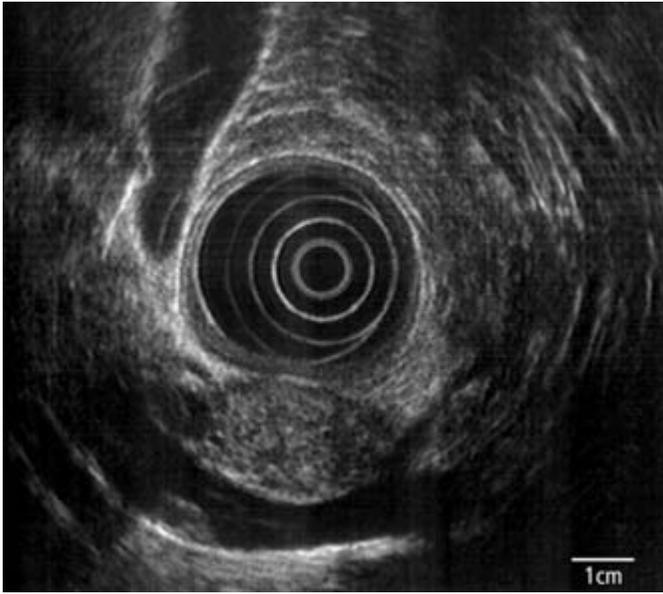
6.82



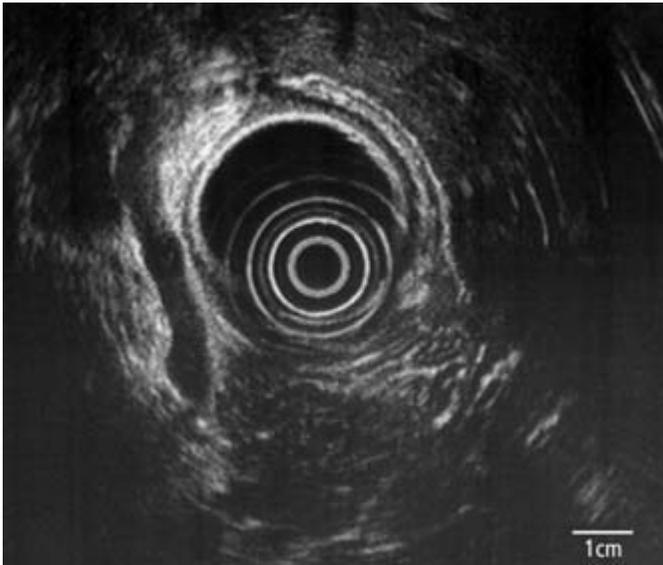
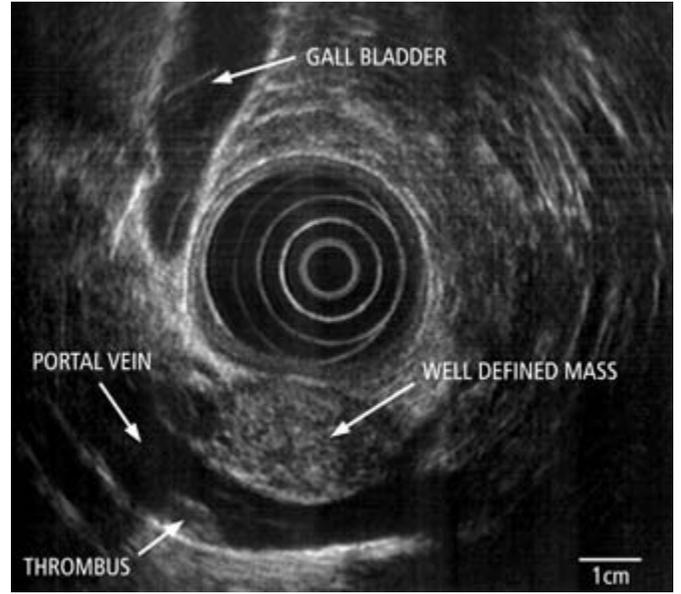
The tumour shown in Fig. 6.82 is invading the splenic vein. EUS is very sensitive for detecting invasion of portal and splenic veins but less so for involvement of superior mesenteric artery or vein. In Fig. 6.82 the portal vein and common bile duct can be followed into but not through the tumour in the head of the pancreas. The tumour appears hypoechoic and irregular in outline.

However, the EUS features of pancreatic inflammatory masses are often similar to those seen in malignancy. The well-defined area in the neck of the pancreas shown in Fig. 6.83 represents an inflammatory mass in a patient with chronic pancreatitis; the mass lies anterior to the portal vein which contains thrombus. In Fig. 6.84 there is an inflammatory hypoechoic mass with hyperechoic foci in the pancreatic head. Figure 6.85 shows a malignant mass in chronic pancreatitis.

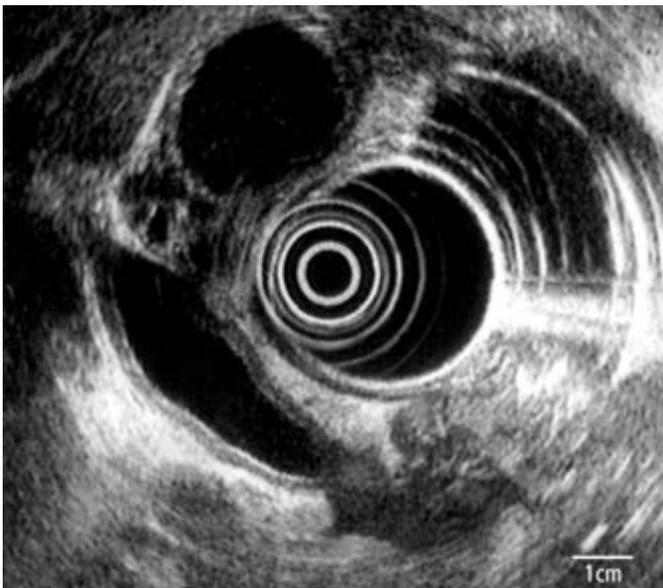
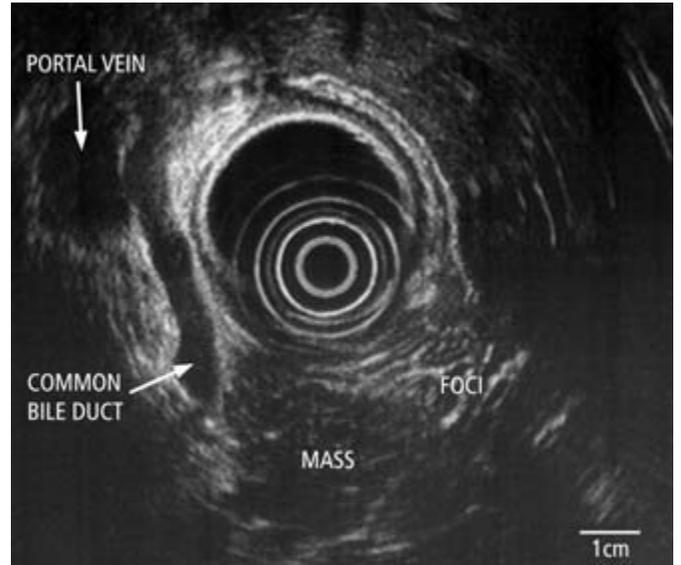
Reports focusing on the EUS appearances of cancer and focal chronic pancreatitis have shown conflicting results. Some authors have claimed a relatively high sensitivity (74–96%) with a low specificity (52–88%) in diagnosing cancer vs. chronic pancreatitis. However, combined results from several prospective and retrospective studies suggest that EUS is the best method for the diagnosis of pancreatic mass lesions and especially small cancers, when compared with other diagnostic modalities with high sensitivity and specificity.



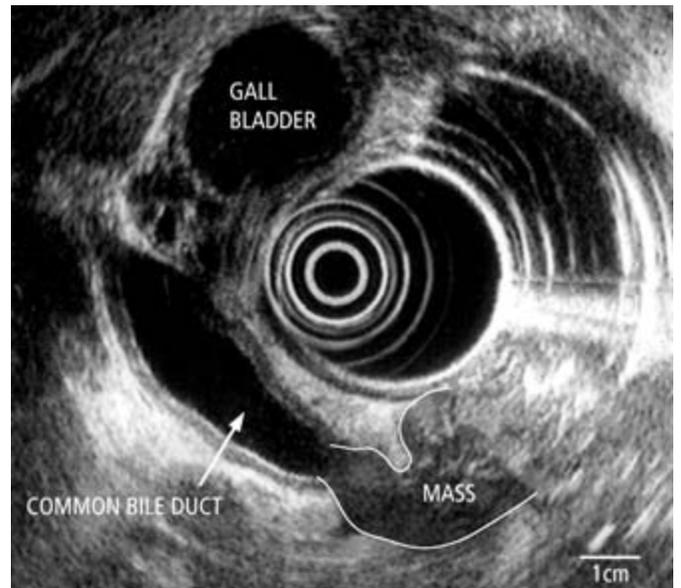
6.83



6.84

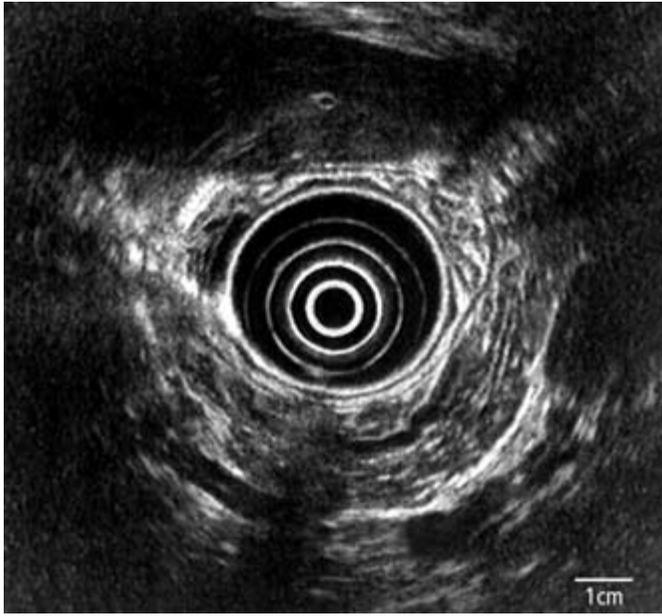


6.85

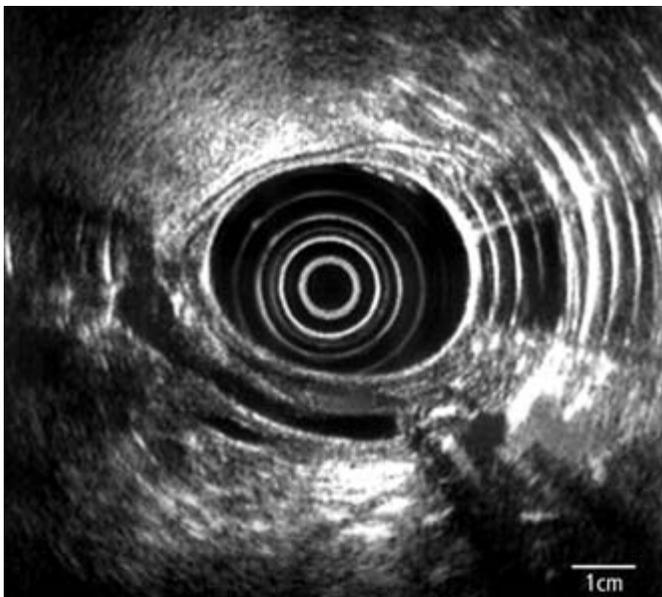
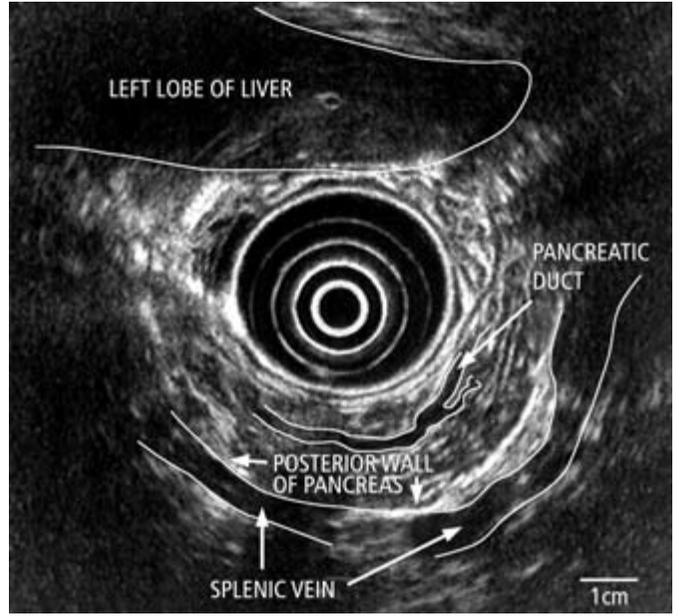


## Chronic pancreatitis

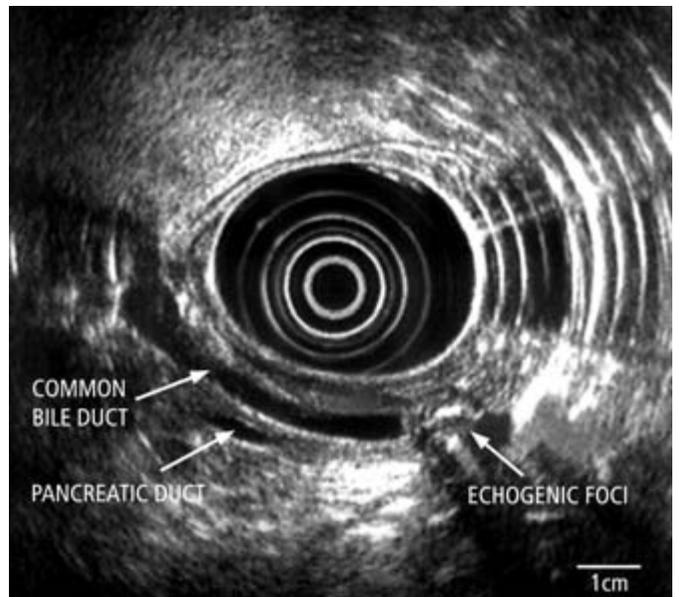
EUS is sensitive for the detection of chronic pancreatitis but not, as with other methods of investigation, in the early stages and it can lack specificity. The detection of distortion of the ductal system (Fig. 6.86), multiple echogenic lesions (Figs 6.87 and 6.88), ductal stones, calcification, and cysts (Figs 6.89 and 6.90) or pseudocysts (Fig. 6.91) correlate well with the results of using other diagnostic modalities (see also Chapter 4).

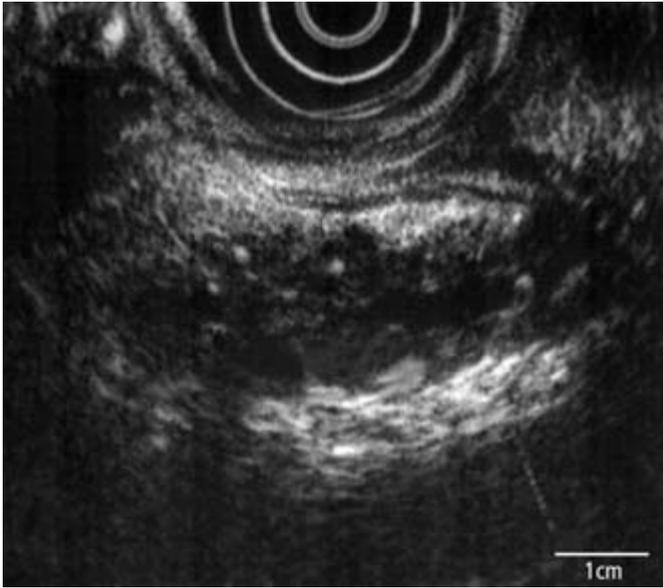


6.86

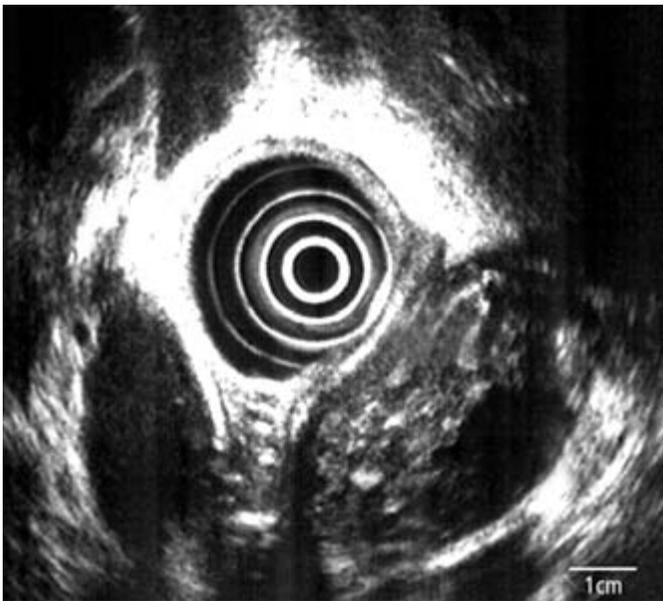
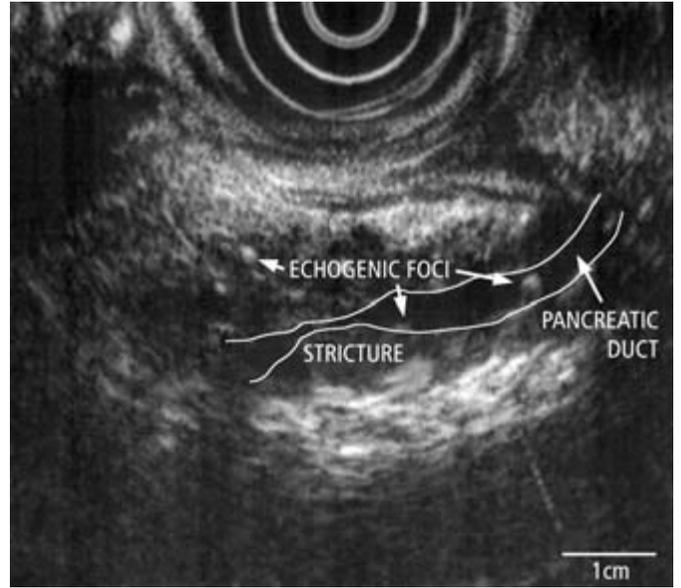


6.87

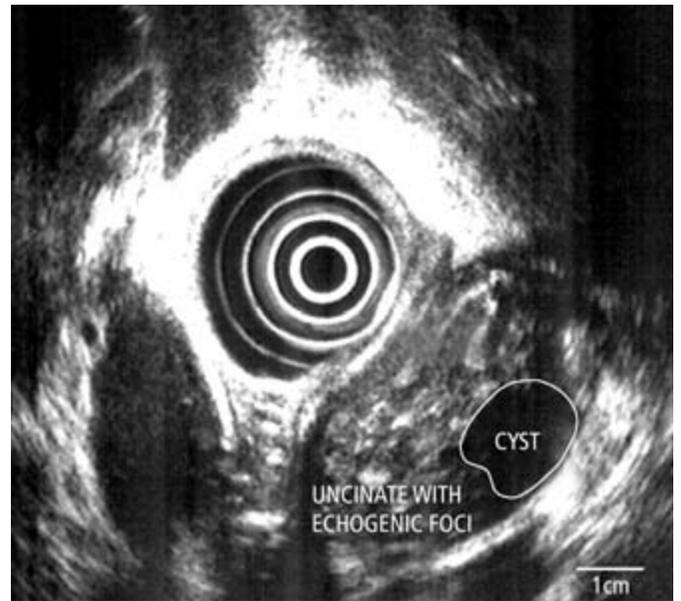


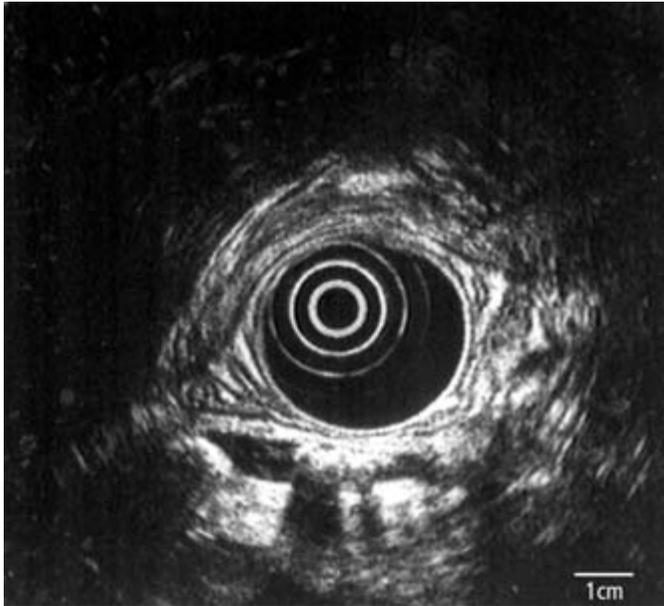


6.88

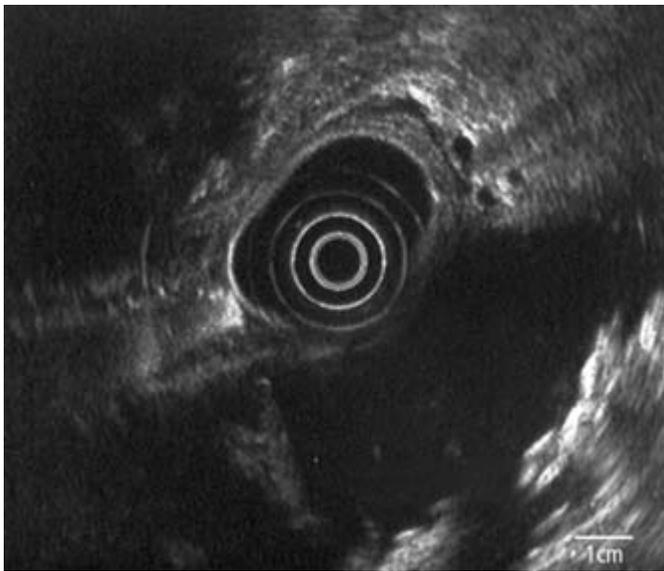
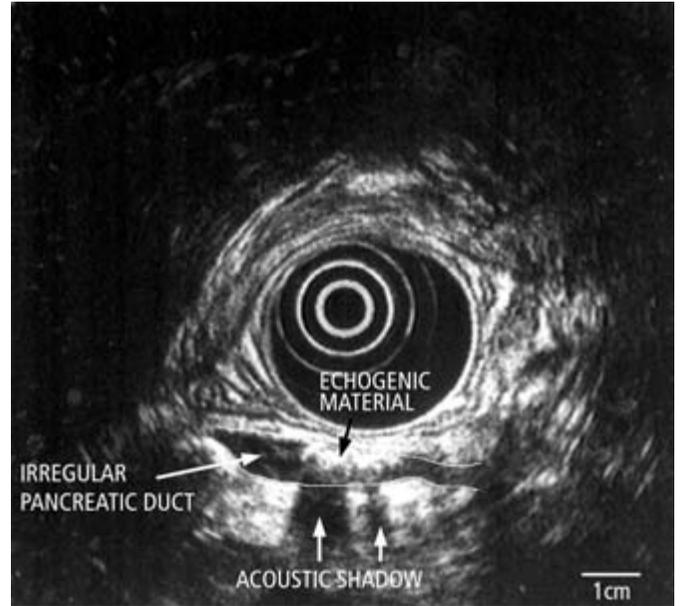


6.89

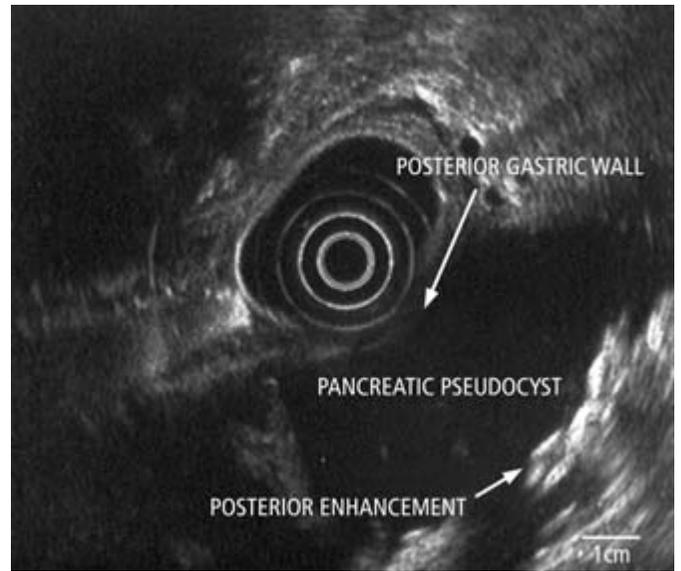




6.90



6.91

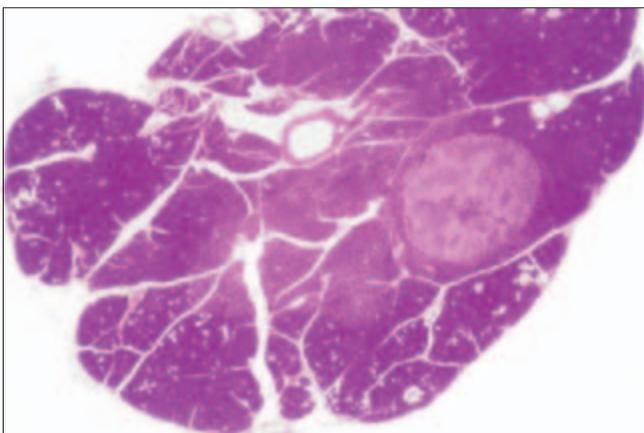


## Endocrine tumours of the pancreas

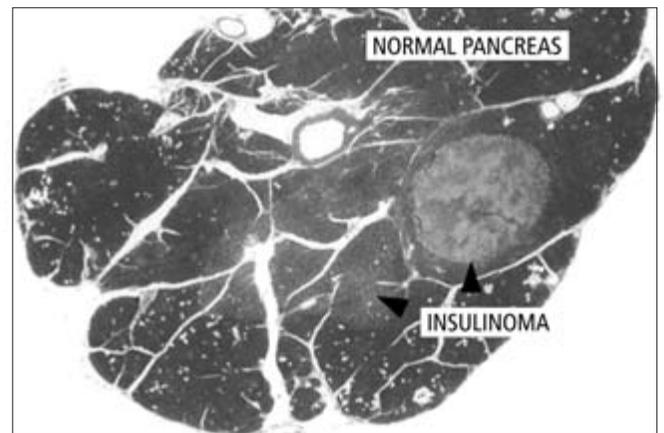
Typically, pancreatic endocrine tumours, such as gastrinomas and insulinomas, are well circumscribed, homogeneous and echo-poor masses within pancreatic tissue (Fig. 6.92). The low-power histopathological appearances are shown in Fig. 6.93. By contrast with pancreatic cancer, EUS in pancreatic endocrine tumours is used to localize the tumour after the diagnosis has been established on the basis of clinical features and laboratory tests. A high level of accuracy can be achieved both with radial and linear echo endoscopes. Compared with somatostatin receptor scintigraphy, EUS is superior in insulinomas but no better in gastrinomas. Small duodenal gastrinomas not detected by careful diagnostic endoscopy using both forward and side-viewing endoscopes are rarely detected by EUS.



6.92

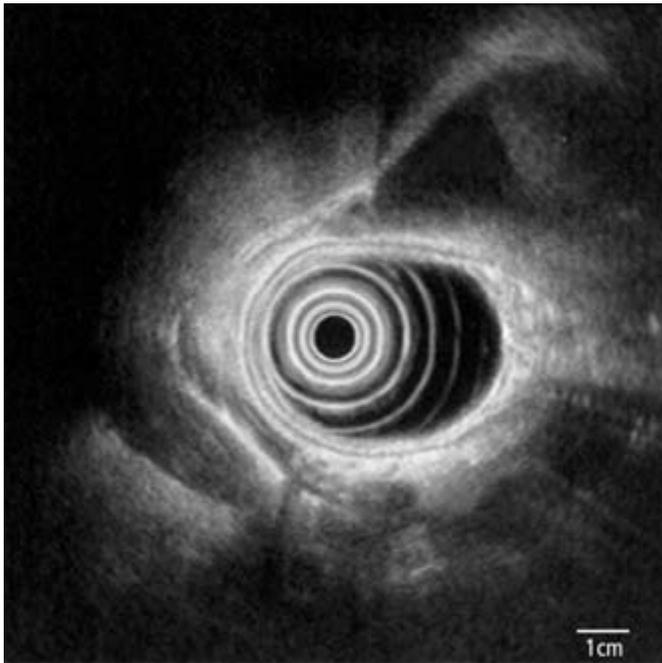


6.93

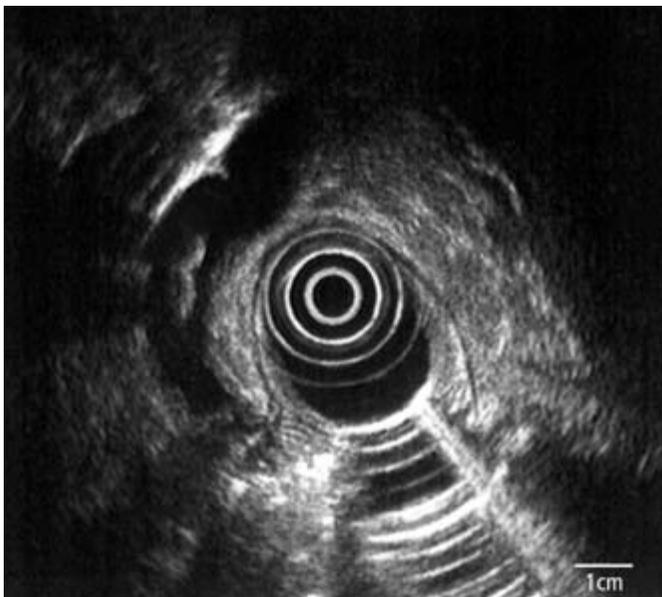
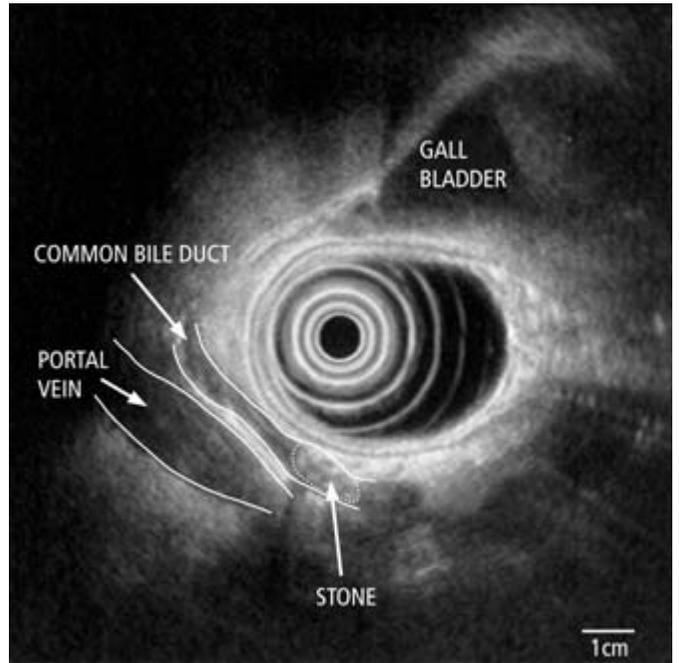


### Common bile duct stones

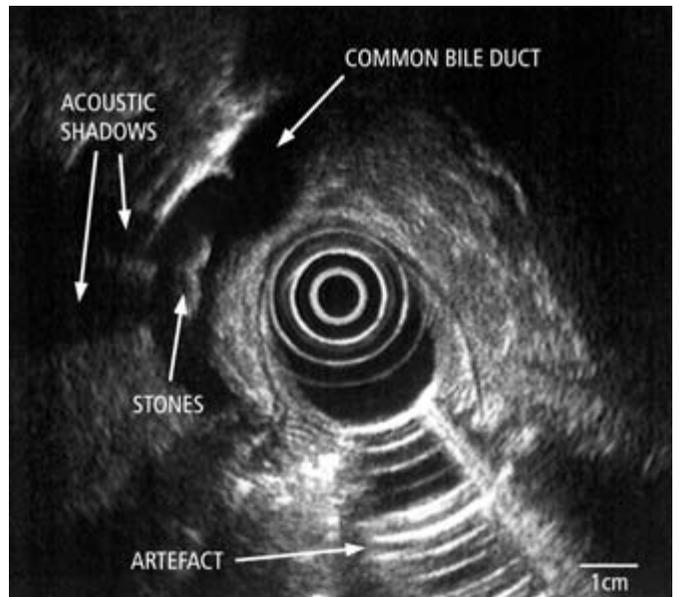
EUS is more than 90% sensitive and specific in the diagnosis of choledocholithiasis and is good at detecting stones whether large or small (Figs 6.94 and 6.95). The appearance of intraductal filling defects convex to the probe is typical of stones, as is the presence of acoustic shadows. Thus EUS may be preferable to US and ERCP in the diagnosis of CBD stones before laparoscopic cholecystectomy or in acute pancreatitis, in patients with a low or intermediate level of suspicion on clinical and laboratory grounds. Patients with a high suspicion of stones (history of jaundice) should probably go directly to ERCP. Newer imaging techniques such as magnetic resonance cholangiography (MRC) may be competitors in the future.



6.94



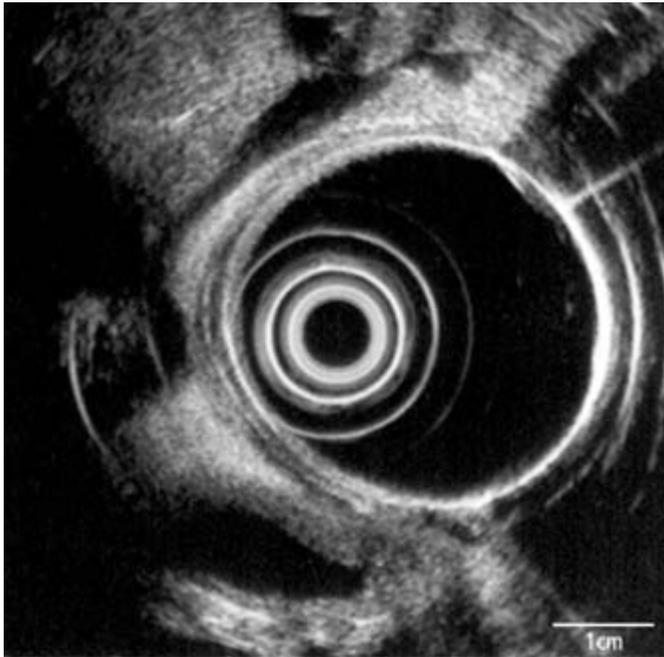
6.95



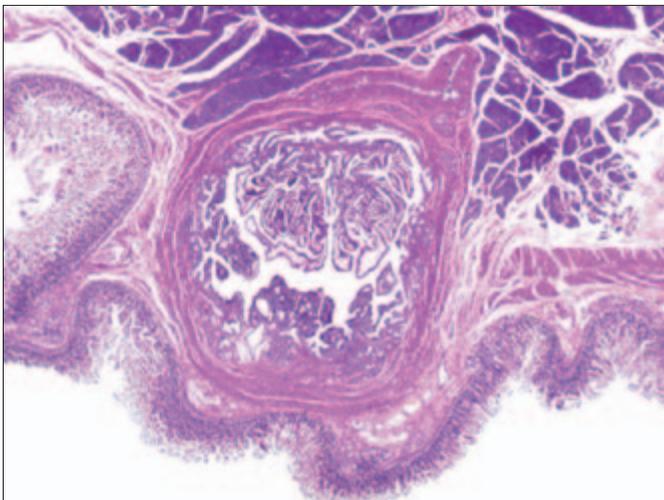
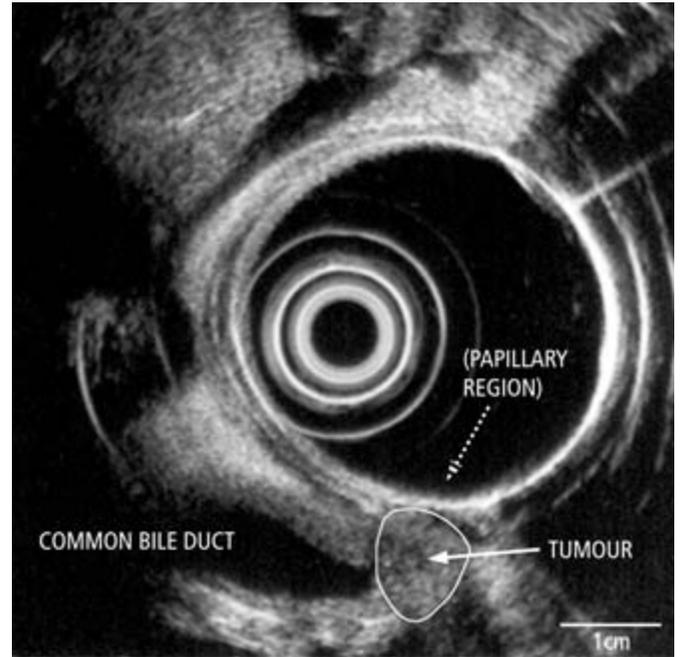
## Ampullary lesions

EUS is effective at detecting and staging ampullary lesions. Figure 6.96 shows a hypoechoic tumour in the ampullary region, and a dilated common bile duct; the tumour proved to be a biliary adenoma (Fig. 6.97).

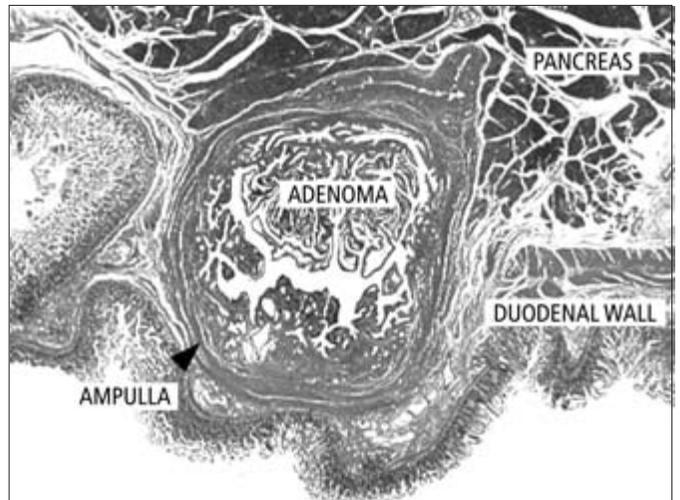
Ampullary lesions are discussed more fully in Chapter 4.



6.96



6.97



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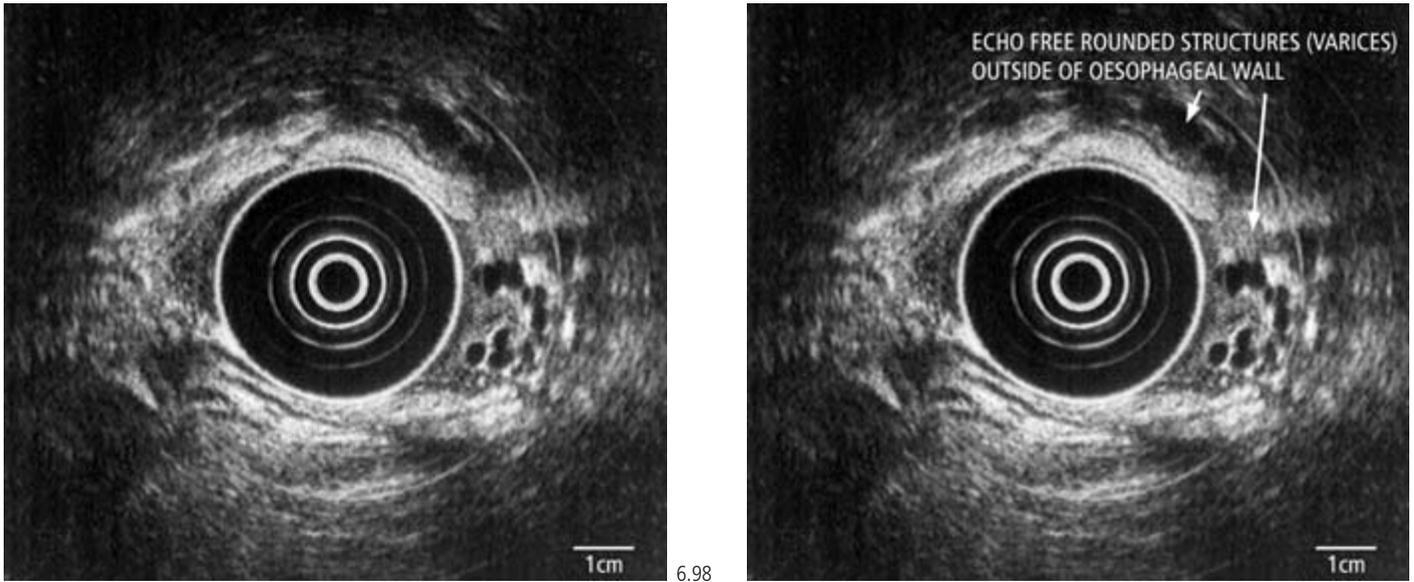
## **EUS-guided fine needle aspiration biopsy**

EUS-guided fine needle aspiration biopsy (FNAB) currently requires the use of linear EUS instruments to show the course of the needle but radial echo endoscopes may eventually be shown to be equally effective. Early results in EUS-guided FNAB show sensitivity rates ranging from 75% to 85% for mediastinal and perigastric lymph nodes, other mediastinal masses and for pancreatic tumours, whereas accuracy for submucosal tumours and enlarged gastric folds was less good. Complication rates are low, the most significant being infection of pancreatic pseudocysts. The clinical value (influence on outcome) in comparison to percutaneous ultrasound-guided or CT-guided puncture has not yet been fully evaluated.

It is thought that EUS-guided biopsy and fine-needle aspiration should pose little risk of tumour seeding as the mural structures involved can be removed at time of surgery if indicated.

**Oesophageal and perigastric varices**

EUS can detect oesophageal (Fig. 6.98) and perigastric varices but it is not yet clear whether use of EUS-guided treatment has any advantage over conventional methods.



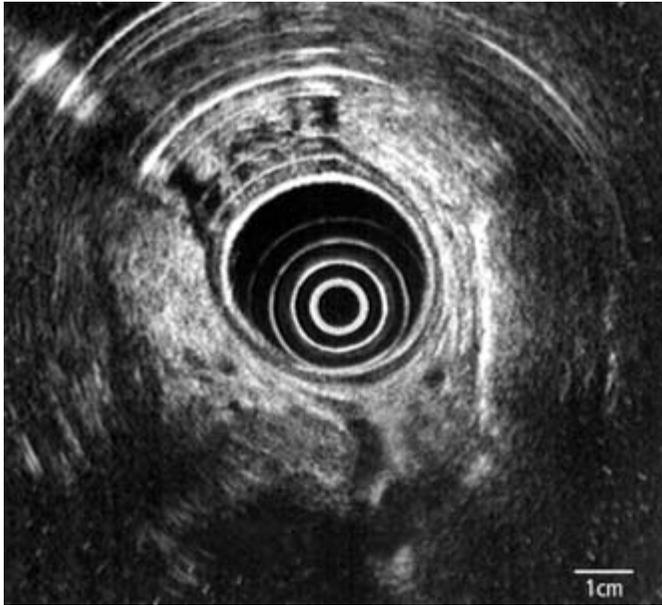
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**Healing of gastric ulcers**

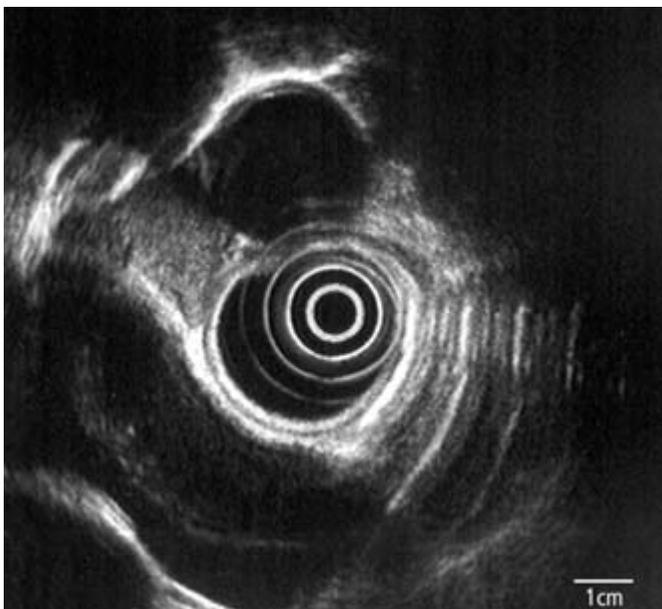
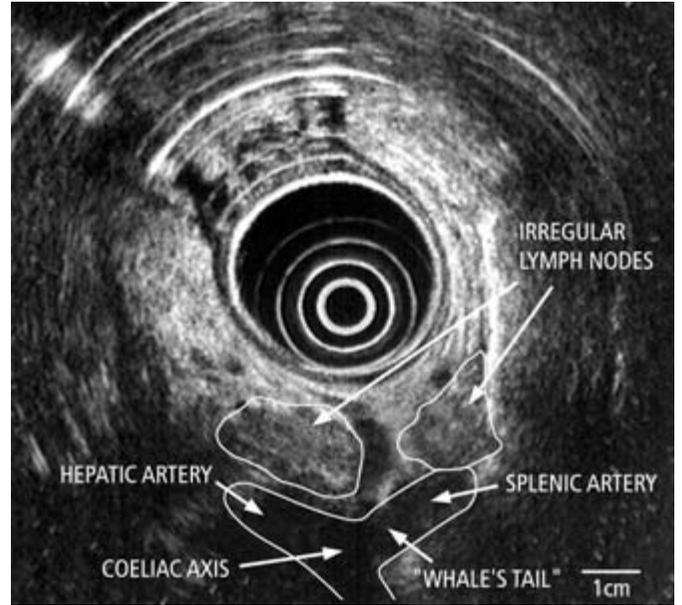
EUS has been used for assessment and monitoring of the healing of gastric ulcers. First, serial observation of an ulcerated lesion can confirm that invasion through the muscularis mucosae, as may occur with a carcinoma, is not present. Secondly, as the ulcer heals, oedematous change in the submucosa diminishes and an echo-dense fibrous scar replaces the echo-poor oedematous area.

## Enlarged lymph nodes

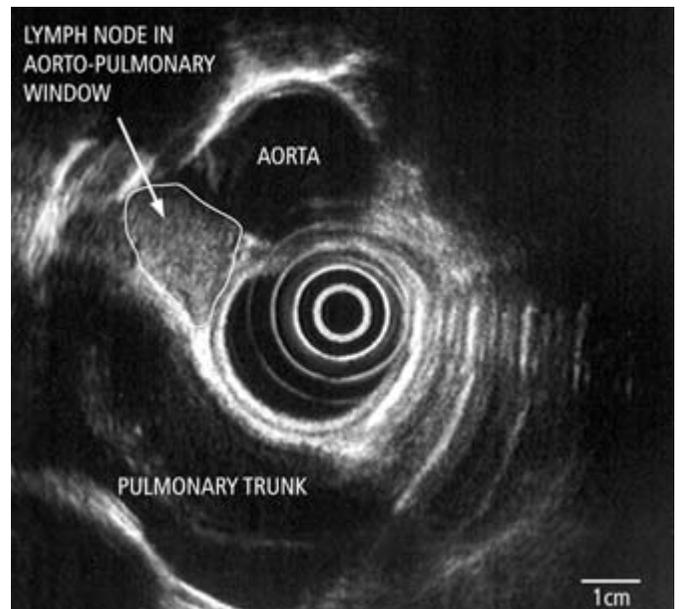
EUS is excellent at demonstrating enlarged lymph nodes adjacent to organ and vascular structures (Figs 6.99 and 6.100) but there are no characteristics which are specific in differentiation of malignant (Fig. 6.101) from inflammatory nodes (Fig. 6.102); the large irregular lymph nodes shown in Fig. 6.101 were found in a patient with chronic lymphatic leukaemia while the large but regular node appearing in Fig. 6.102 was inflammatory in nature.

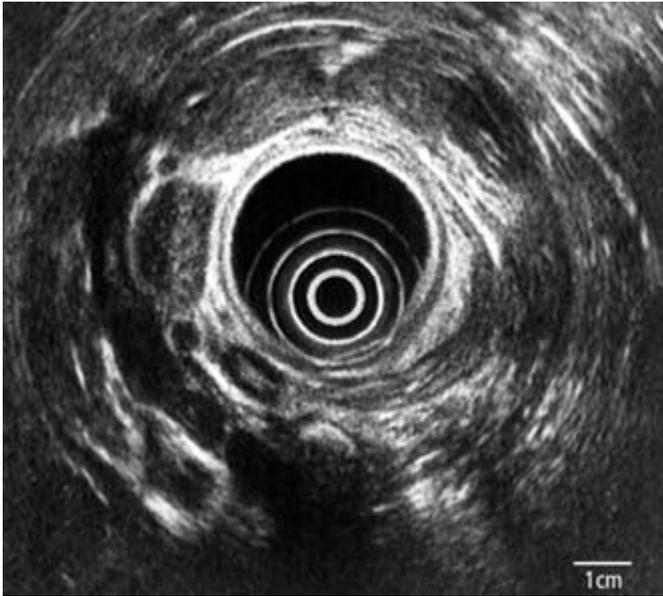


6.99

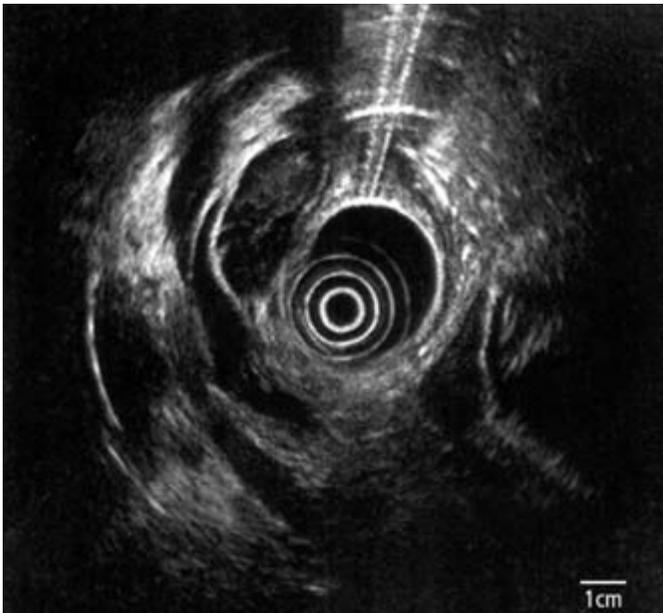
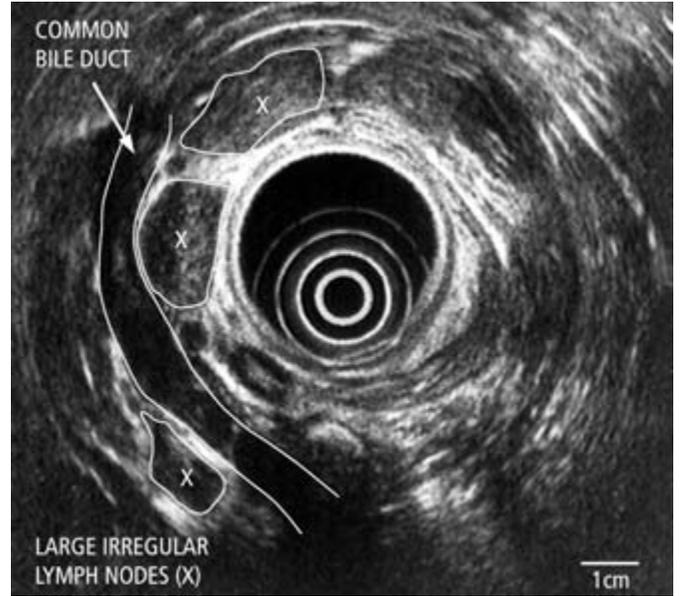


6.100

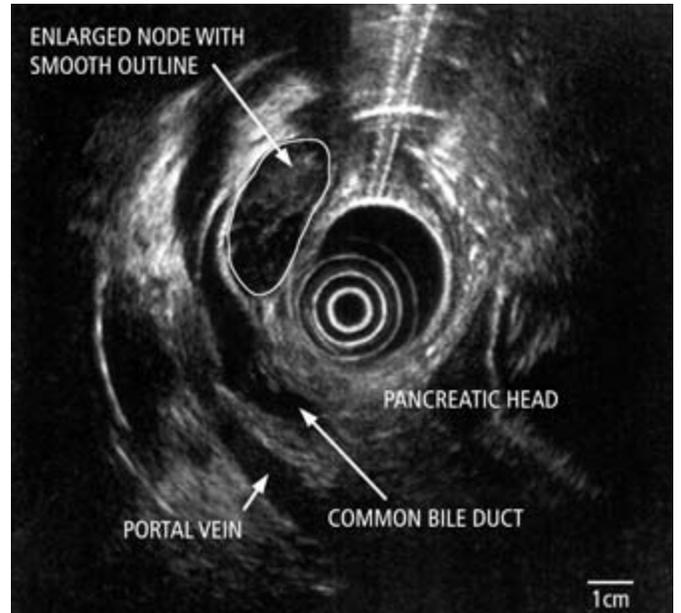




6.101



6.102



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## Future prospects

Current miniproboscopes introduced via the working channel of conventional forward- or side-viewing endoscopes suffer from limited penetration depth and insufficient durability. However it is anticipated that technical improvements will lead to the development of more stable miniproboscopes. Three-dimensional reconstruction of EUS images has been used for oesophageal and gastric lesions, but in the pancreaticobiliary area this is more difficult. A single instrument which combines both radial and linear scanning modes would be a most welcome development. The possibilities of Doppler EUS have yet to be fully evaluated.

Mechanical scanning is already giving way to solid state radial and linear probes. Both types are likely to get smaller and may have an increased range of switchable frequencies.

An exciting potential of EUS lies in therapy. The combination of optical flexible endoscopy and focused ultrasound to destroy tissue has not yet been achieved, though percutaneous US treatment has been used for liver tumours. Under EUS guidance a needle such as is used for FNAB could be employed to inject substances with therapeutic potential, for example alcohol for coeliac plexus neurolysis, botulinum toxin for achalasia, or, perhaps activated macrophages or anticancer treatments into tumour tissue.

At present, diagnostic EUS is underused and deserves wider acceptance. With therapeutic ultrasound, we are only at the beginning of an important new era.

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### **Guidelines/consensus papers**

It was our intention to include a list of guidelines and consensus papers to cover many aspects of gastrointestinal endoscopy. However due to rapid advances, changes in practice and the growth of the internet any such references would be out of date by the time of publication. It is therefore suggested instead that access to such sources should be made via the internet using some of the sites listed below.

### **Websites of major gastroenterology and endoscopy societies**

American Society for Gastrointestinal Endoscopy (ASGE)  
<http://www.asge.org/>

American Gastroenterological Association  
<http://www.gastro.org>  
British Society of Gastroenterology  
<http://www.bsg.org.uk>  
Canadian Association of Gastroenterology  
<http://www.gi.ucalgary.ca>  
European Society of Gastrointestinal Endoscopy  
<http://www.esge.com/choix.html>  
EUS-ONLINE  
<http://www.eus-online.org>  
Society of Gastroenterology Nurses and Associates, Inc.—SGNA  
<http://www.sgna.org>  
World Organization for Digestive Endoscopy  
<http://www.omed.org/>

### **Guidelines on line**

British Society of Gastroenterology  
<http://www.bsg.org.uk/guidelines.html>  
American Society for Gastrointestinal Endoscopy  
<http://www.asge.org/>

### **Online endoscopy image libraries**

Atlas of Endoscopy of Gastrointestinal Diseases  
[http://www.luz.ve/ICA/Atlas\\_med/i\\_index.html](http://www.luz.ve/ICA/Atlas_med/i_index.html)  
Atlas of Gastrointestinal Endoscopy  
<http://www.mindspring.com/~dmmmd/index.html>  
An annotated library of endoscopic images  
<http://.home.t-online.de/home/afreytag/indexe.htm>  
A weekly endoscopy quiz, and an archive of almost 200 previous images  
<http://www.cag.ucalgary.ca/endoquiz/quiz.HTM>  
An image library with both endoscopic and radiologic examples  
<http://www.gastrolab.net/welcome.htm>

### **Other useful sites**

Doctor's Guide to the Internet  
<http://www.pslgroup.com/DOCGUIDE.HTM>  
Gastroenterology Resources from Columbia University  
<http://cpmcnet.columbia.edu/dept/gi/elsewhere.html>  
Gastrointestinal Endoscopy  
<http://www1.mosby.com/scripts/om.dll/serve?action=searchDB&searchDBfor=home&id=ge>  
GastroHep an international gastroenterology resource site  
<http://www.gastrohep.com/>  
Gastrolab Home Page  
<http://www.gastrolab.net/g5endo.htm>  
Medscape's Gastroenterology MedPulse(R)  
<http://gastroenterology.medscape.com>  
PubMed—Medline on line  
<http://www.ncbi.nlm.nih.gov/PubMed/>

It must be remembered that web sites come and go, and others change their locations. Most web sites quoted have links with other relevant sites enabling easy surfing to areas of further interest.

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Page numbers refer to information in the text and relevant figures when they occur on the same page. Page numbers in *italics* refer to information that is shown only in figures. Page numbers in **bold** refer to tables. For definitions of abbreviations see p. xv.

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