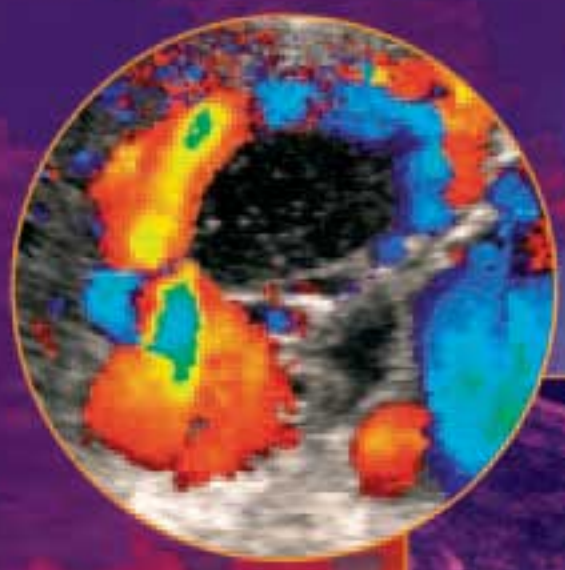
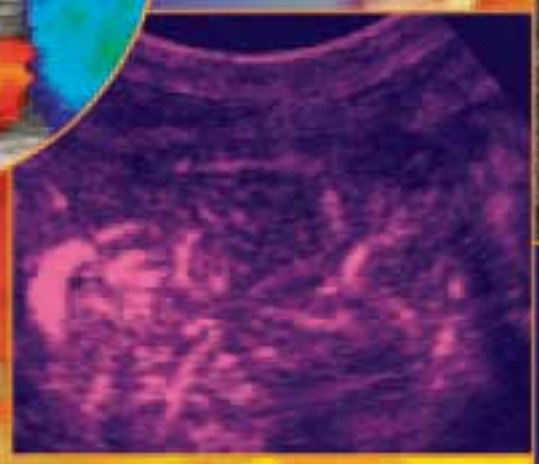
SECOND EDITION

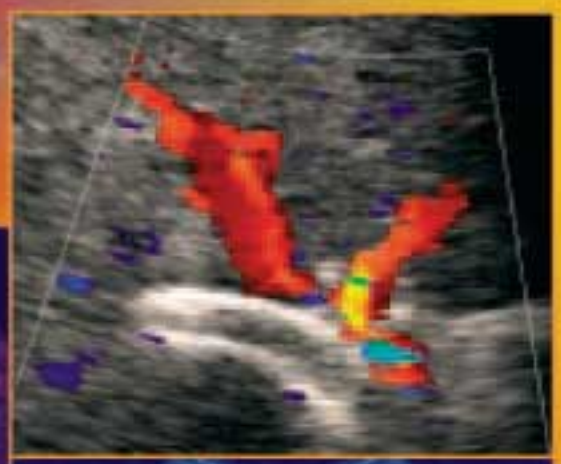
Abdominal ULTRASOUND

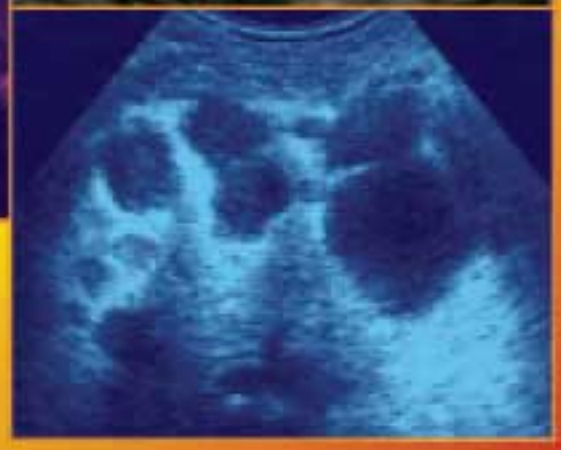
HOW, WHY AND WHEN

Jane Bates









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Abdominal Ultrasound

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SECOND EDITION

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CHURCHILL LIVINGSTONE

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Preface

Ultrasound continues to be one of the most important diagnostic tools at our disposal. It is used by a wide range of healthcare professionals across many applications. This book is intended as a practical, easily accessible guide to sonographers and those learning and developing in the field of abdominal ultrasound. The most obvious drawbacks of ultrasound diagnosis are the physical limitations of sound in tissue and its tremendous dependence upon the skill of the operator. This book seeks to enable the operator to maximize the diagnostic information and to recognize the limitations of the scan.

Where possible it presents a wider, more holistic approach to the patient, including presenting symptoms, complementary imaging procedures and further management options. It is not a comprehensive account of all the pathological processes likely to be encountered, but is intended as a springboard from which practical skills and clinical knowledge can develop further.

This book aims to increase the sonographer's awareness of the contribution of ultrasound within the general clinical picture, and introduce the sonographer to its enormous potential.

The author gratefully acknowledges the help and support of the staff of the Ultrasound Department at St James's University Hospital, Leeds.

Leeds 2004 Jane Bates

Abbreviations

ADPCDK	autosomal dominant polycystic disease of the kidney
AFP	alpha-fetoprotein
AI	acceleration index
AIDS	acquired immune deficiency syndrome
AIUM	American Institute for Ultrasound in Medicine
ALARA	as low as reasonably achievable
ALT	alanine aminotransferase
AP	anteroposterior
APKD	autosomal dominant (adult) polycystic kidney
ARPCDK	autosomal recessive polycystic disease of the kidney
AST	aspartate aminotransferase
AT	acceleration time
AV	arteriovenous
BCS	Budd–Chiari syndrome
CAPD	continuous ambulatory peritoneal dialysis
CBD	common bile duct
CD	common duct
CF	cystic fibrosis
CT	computed tomography
DIC	disseminated intravascular coagulation
DICOM	Digital Imaging and Communications in Medicine
DMSA	dimercaptosuccinic acid
DTPA	diethylene triaminepenta-acetic acid
EDF	end-diastolic flow
ERCP	endoscopic retrograde cholangiopancreatography
ESWL	extracorporeal shock wave lithotripsy
EUS	endoscopic ultrasound
FAST	focused assessment with sonography for trauma
FDA	Food and Drug Administration
FPS	frames per second
HA	hepatic artery
HCC	hepatocellular carcinoma
HELLP	haemolytic anaemia, elevated liver enzymes and low platelet count
HIDA	hepatic iminodiacetic acid
HPS	hypertrophic pyloric stenosis
HV	hepatic vein
INR	international normalized ratio
IOUS	intraoperative ultrasound
IVC	inferior vena cava
IVU	intravenous urogram
KUB	kidneys, ureters, bladder
LFT	liver function test
LPV	left portal vein
LRV	left renal vein
LS	longitudinal section
LUQ	left upper quadrant
MCKD	multicystic dysplastic kidney

MHA	middle hepatic artery
MHV	middle hepatic vein
MI	mechanical index
MPV	main portal vein
MRA	magnetic resonance angiography
MRA	main renal artery
MRCP	magnetic resonance cholangiopancreatography
MRI	magnetic resonance imaging
MRV	main renal vein
ODS	output display standard
PAC	photographic archiving and communications
PACS	photographic archiving and communications systems
PBC	primary biliary cirrhosis
PCKD	polycystic kidney disease
PCS	pelvicalyceal system
PD	pancreatic duct
PI	pulsatility index
PID	pelvic inflammatory disease
PRF	pulse repetition frequency
PSC	primary sclerosing cholangitis
PTLD	post-transplant lymphoproliferative disorder
PV	portal vein
RAS	renal artery stenosis
RCC	renal cell carcinoma
RF	radiofrequency
RHV	right hepatic vein
RI	resistance index
RIF	right iliac fossa
RK	right kidney
RPV	right portal vein
RRA	right renal artery
RRV	right renal vein
RUQ	right upper quadrant
RVT	renal vein thrombosis
SA	splenic artery
SLE	systemic lupus erythematosus
SMA	superior mesenteric artery
SV	splenic vein
TB	tuberculosis
TGC	time gain compensation
THI	tissue harmonic imaging
TI	thermal index
TIB	bone-at-focus index
TIC	cranial index
TIPS	transjugular intrahepatic portosystemic shunt
TIS	soft-tissue thermal index
TORCH	toxoplasmosis, rubella, cytomegalovirus and HIV
TS	transverse section
UTI	urinary tract infection
VUJ	vesicoureteric junction
WRMSD	work-related musculoskeletal disorders
XGP	xanthogranulomatous pyelonephritis

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Chapter 1

Optimizing the diagnostic information

CHAPTER CONTENTS

IMAGE OPTIMIZATION

Misinterpretation of ultrasound images is a significant risk in ultrasound diagnosis. Because ultrasound scanning is operator-dependent, it is imperative that the sonographer has proper training in order to achieve the expected diagnostic capabilities of the technique. The skill of effective scanning lies in the operator's ability to maximize the diagnostic information available and in being able to interpret the appearances properly. This is dependent upon:

- Clinical knowledge—knowing what to look for and why, knowing how to interpret the appearances on the image and an understanding of physiological and pathological processes.
- Technical skill—knowing how to obtain the most useful and relevant images, knowledge of artifacts and avoiding the pitfalls of scanning.
- Knowledge of the equipment being used—i.e. making the most of your machine.

The operator must use the controls to their best effect (see Box 1.1). There are numerous ways in which different manufacturers allow us to make compromises during the scanning process in order to improve image quality and enhance diagnostic information.

The quality of the image can be improved by:

- Increasing the frequency—at the expense of poorer penetration (Fig. 1.1).
- Increasing the line density—this may be achieved by reducing the frame rate and/or reducing the sector angle and/or depth of field (Fig. 1.2).

Box 1.1 Making the most of your equipment

- Use the highest frequency possible—try increasing the frequency when examining the pancreas or anterior gallbladder.
- Use the lowest frame rate and highest line density possible. Restless or breathless patients will require a higher frame rate.
- Use the smallest field practicable—sections through the liver require a relatively wide sector angle and a large depth of view, but when examining an anterior gallbladder, for example, the field can be greatly reduced, thereby improving the resolution with no loss of frame rate.
- Use the focal zone at relevant correct depth.
- Use tissue harmonic imaging to increase the signal to noise ratio and reduce artefact.
- Try different processing curves to highlight subtle abnormalities and increase contrast resolution.

- Using the focal zones correctly—focus at the level under investigation, or use multiple focal zones at the expense of a decreased frame rate (Fig. 1.3).
- Utilizing different pre- and post-processing options, which may highlight particular areas (Fig. 1.4).
- Using tissue harmonics to reduce artefact (Fig. 1.5). This technique utilizes the second harmonic rather than the fundamental frequency using either filtration or pulse inversion.[1] This results in a higher signal-to-noise ratio which demonstrates particular benefits in many difficult scanning situations, including obese or gassy abdomens.

It is far better to have a scan performed properly on a low-tech piece of equipment by a knowledgeable and well-trained operator than to have a poorly performed scan on the latest high-tech machine (Fig. 1.6). A good operator will get the best out of even the lowliest scanning device and produce a result that will promote the correct patient management. A misleading result from a top-of-the-range scanner can be highly damaging and at best delay the correct treatment or at worst promote incorrect management. The operator should know the limitations of the scan in terms of equipment capabilities, operator skills, clinical problems and patient limitations, take those limitations into account and communicate them where necessary.

THE USE OF DOPPLER

The use of Doppler ultrasound is an integral part of the examination and should not be considered as a separate entity. Many pathological processes in the abdomen affect the haemodynamics of relevant organs and the judicial use of Doppler is an essential part of the diagnostic procedure. This is discussed in more detail in subsequent chapters.

Colour Doppler is used to assess the patency and direction of flow of vessels in the abdomen,

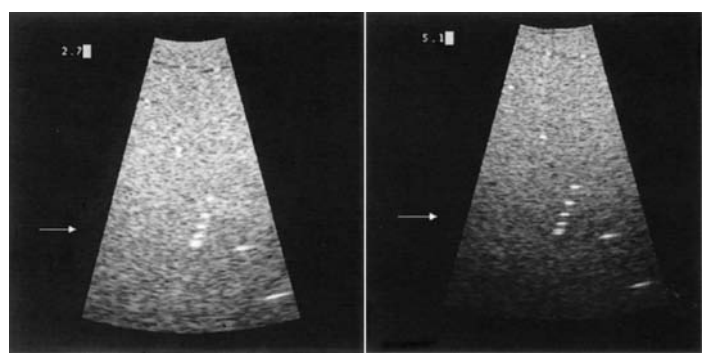

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Figure 1.1 The effect of changing frequency. (A) At 2.7 MHz the wires are poorly resolved and the background 'texture' of the test object looks coarse. (B) The same transducer is switched to a resonant frequency of 5.1 MHz. Without changing any other settings, the six wires are now resolved and the background texture appears finer.

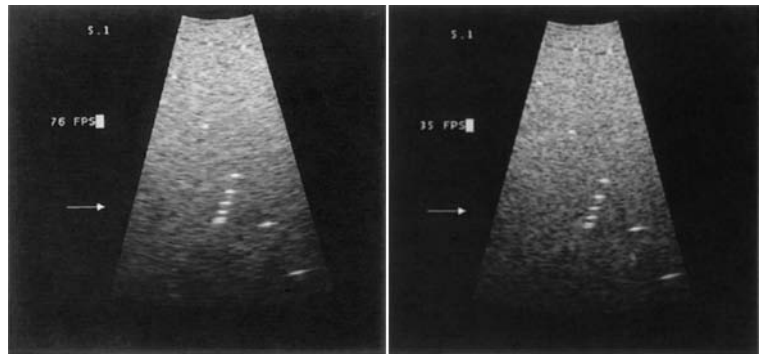

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Figure 1.2 The effect of frame rate. (A) 76 frames per second (FPS). (B) 35 FPS—the resulting higher line density improves the image, making it sharper.

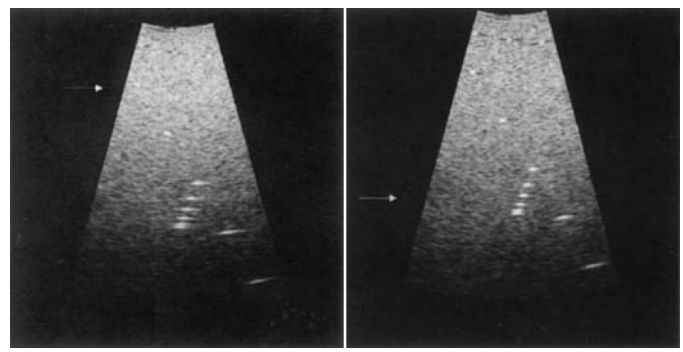

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Figure 1.3 The effect of focal zone placement. (A) With the focal zone in the near field, structures in the far field are poorly resolved. (B) Correct focal zone placement improves both axial and lateral resolution of the wires.

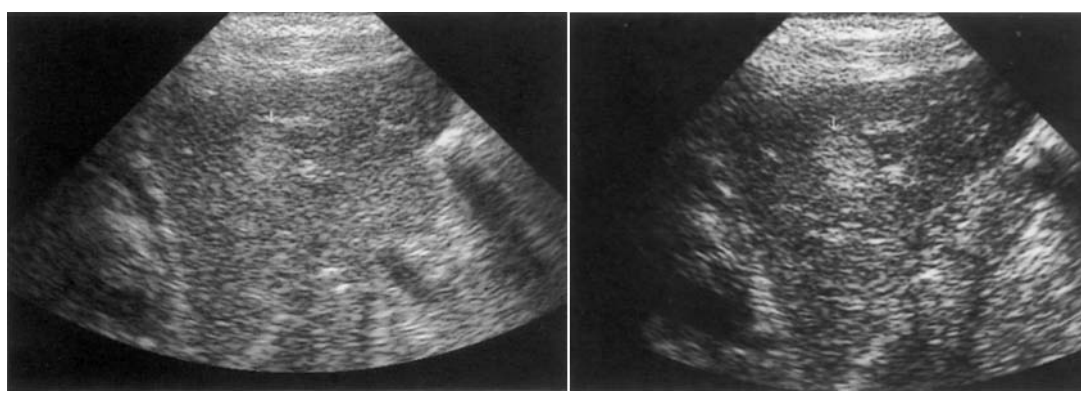

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Figure 1.4 The effect of using post-processing options. (A) A small haemangioma in the liver merges into the background and is difficult to detect. (B) A post-processing option, which allocates the range of grey shades in a non-linear manner, enhances contrast resolution and improves detection of focal lesions.

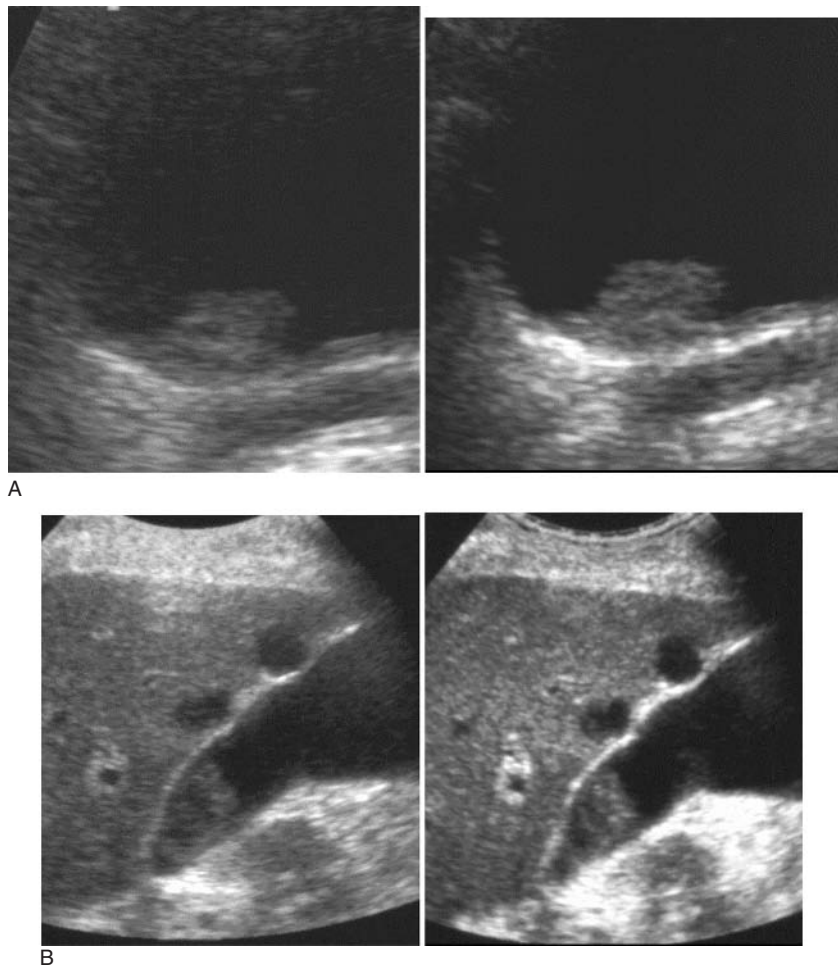

Figure 1.5 The effect of tissue harmonic imaging (THI): (A) a bladder tumour in fundamental imaging mode (left) is shown with greater definition and loss of artifact in THI (right). (B) In an obese patient, cysts near the gallbladder (left) are shown in greater detail using pulse inversion tissue harmonics (right). A small nodule is demonstrated in the lower cyst.

to establish the vascularity of masses or lesions and to identify vascular disturbances, such as stenoses. Flow information is colour-coded (usually red towards and blue away from the transducer) and superimposed on the image. This gives the operator an immediate impression of a vascular map of the area (Fig. 1.7). This Doppler information is obtained simultaneously, often from a relatively large area of the image, at the expense of the grey-scale image quality. The extra time taken to obtain the Doppler information for each line results in a reduction in frame rate and line density which worsens as the colour Doppler area is enlarged. It is advisable, therefore, to use a compact colour 'box' in order to maintain image quality.

Power Doppler also superimposes Doppler information on the grey-scale image, but without any directional information. It displays only the amount of energy (Fig. 1.8). The advantage of this is that the signal is stronger, allowing identification of smaller vessels with lower velocity flow than colour Doppler. As it is less angle-dependent than colour Doppler it is particularly useful for vessels which run perpendicular to the beam, for example the inferior vena cava (IVC).

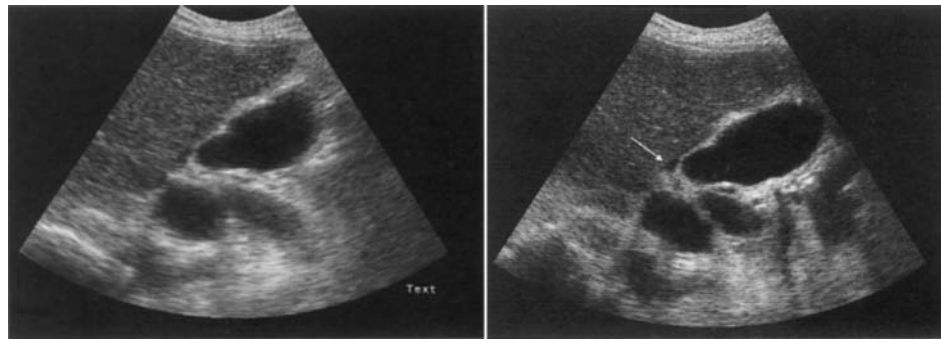

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Figure 1.6 The importance of using the equipment properly. (A) Incorrect use of equipment settings makes it difficult to appreciate the structures in the image. (B) By increasing the resonant frequency, decreasing the frame rate and adjusting the focal zone correctly, a small rim of fluid around the gallbladder is seen and the gallbladder wall and vessels posterior to the gallbladder are made clear.

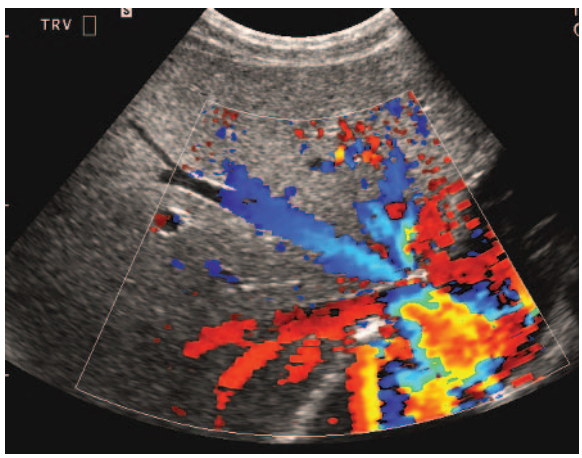

Figure 1.7 Colour Doppler of the hepatic vein confluence. The right hepatic vein appears red, as it is flowing towards the transducer. The left and middle hepatic veins are in blue, flowing away from the transducer. Note the peripheral middle hepatic vein, which appears to have no flow; this is an artifact due to the angle of that part of the vessel to the beam.

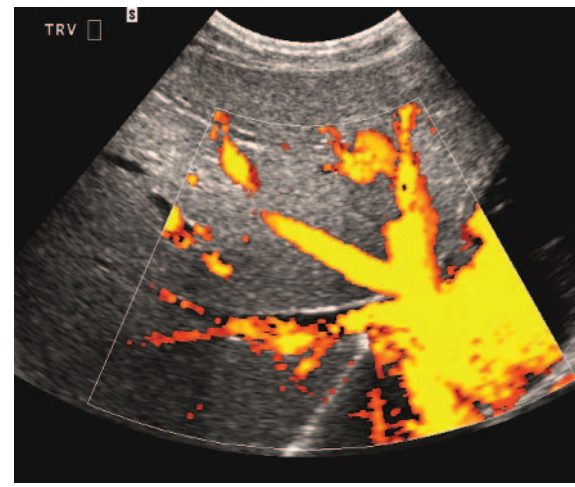

Figure 1.8 Power Doppler of the hepatic vein confluence. We have lost the directional information, but flow is demonstrated in all parts of the vessel—even those perpendicular to the beam.

Pulsed Doppler uses pulses of Doppler from individual elements or small groups of elements within the array. This allows the operator to select a specific vessel, which has been identified on the grey-scale or colour Doppler image, from which to obtain a spectrum. This gives further information regarding the flow envelope, variance, velocity and downstream resistance of the blood flow (Fig. 1.9).

Getting the best out of Doppler

Familiarity with the Doppler controls is essential in order to avoid the pitfalls and increase confidence in the results.

It is relatively straighforward to demonstrate flow in major vessels and to assess the relevant spectral waveform; most problems arise when trying to diagnose the *lack* of flow in a suspected thrombosed vessel, and in displaying low-velocity

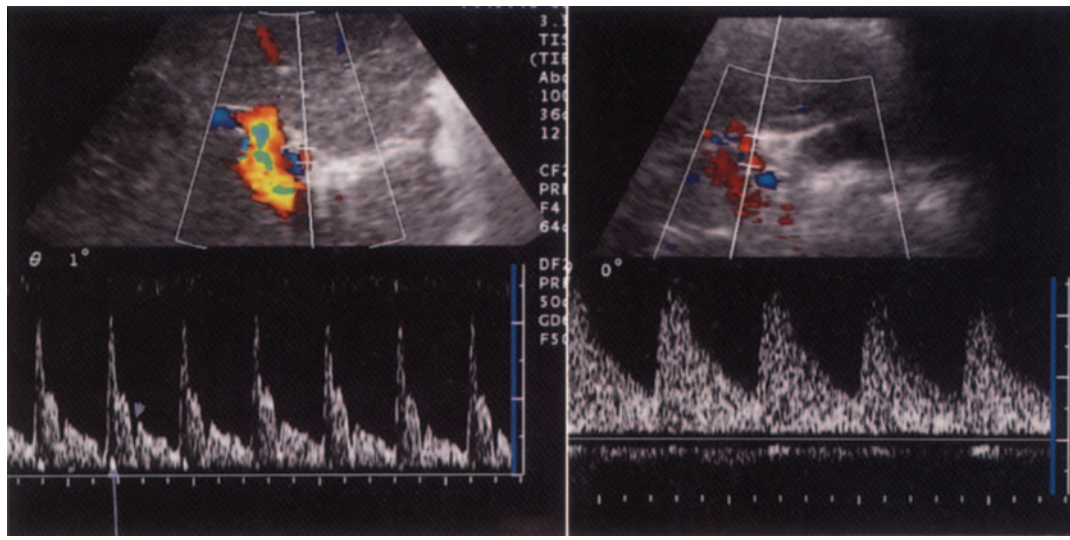

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Figure 1.9 Flow velocity waveforms of hepatic arteries. (A) High-resistance flow with low end-diastolic flow (EDF) and a dichrotic notch (arrowhead). The clear 'window' during systole (arrow) indicates little variance, with the blood flowing at the same velocity throughout the vessel. During diastole, the area under the envelope is 'filled in', indicating greater variance in flow. (B) By contrast, this hepatic artery trace indicates low-resistance flow with good EDF and no notch. Variance is apparent throughout the cycle.

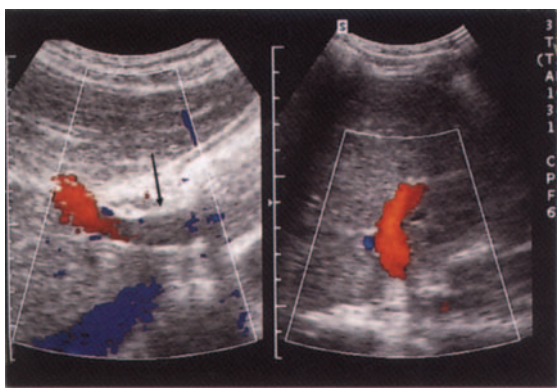

Figure 1.10 On the left, the portal vein appears to have no flow (arrow) when it lies at 90° to the beam—a possible misinterpretation for thrombosis. When scanned intercostally, the vein is almost parallel to the beam and flow is easily demonstrated.

flow in difficult-to-access vessels. Doppler is known to produce false-positive results for vessel occlusion (Fig. 1.10) and the operator must avoid the pitfalls and should ensure that the confidence levels are as high as possible (see Box 1.2).

CHOOSING A MACHINE

The ultrasound practitioner is confronted with a confusing range of equipment and choosing the right machine for the job can be a daunting task.

An informed and useful choice is more likely when the purchaser has considerable experience within the particular clinical field. Many machines, purchased in the first enthusiastic flush of setting up a new service, for example, turn out to be unsuitable two or three years later.

Mistakes are made by insufficient forward planning. A number of machines (usually at the cheaper end of the market), though initially purchased for specific, sometimes narrow, purposes, end up being expected to perform more complex and wider-ranging applications than originally planned.

Take careful stock of the range of examinations you expect your machine to perform. Future developments which may affect the type of machine you buy include:

- Increase in numbers of patients calculated from trends in previous years.

Box 1.2 Steps to take if you can't detect flow with Doppler

- Ensure the angle of insonation between the vessel and the transducer is <60°. Colour and pulsed Doppler are highly angle-dependent.
- Ensure the Doppler gain is set at the correct level. (Colour and pulsed Doppler gain settings should be just below background noise level.)
- Ensure the Doppler power/output setting is sufficient.
- Ensure the pulse repetition frequency (PRF) is set correctly. A low PRF ('range' or 'scale' setting) is required to pick up low-velocity flow.
- Ensure the wall thump filter setting is low. (If the setting is too high, real low-velocity flow is filtered out.)
- Use power Doppler, which is more sensitive and is not angle-dependent.
- Know the limitations of your machine. Machines differ in their ability to detect low-velocity flow.
- If in doubt, test it on a reference vessel you *know* should contain flow.

- Increase in range of possible applications, an impending peripheral vascular service, for example, or regional screening initiative.
- Clinical developments and changes in patient management which may require more, or different, ultrasound techniques, for example, medical therapies which require ultrasound monitoring, applications involving the use of contrast agents, surgical techniques which may require intraoperative scanning, increases or decreases in hospital beds, introduction of new services and enlargement of existing ones.
- Impending political developments by government or hospital management, resulting in changes in the services provided, the funding or the catchment area.
- Other impending ultrasound developments, such as the use of contrast media or ultrasound-guided therapies which may be required in future.

The following points are useful to bear in mind when purchasing new equipment:

Probe number and design (Fig 1.11)

Consider the footprint, shape and frequencies required: most modern transducers are broadband in design, enabling the user to access a wider range of frequencies. This is a big advantage as this limits the number of probes required for a general service. A curved array probe is suitable for most general abdominal applications, operating in the 3.5–6 MHz region. Additional higher-frequency probes are useful for paediatrics and for superficial structures. A small footprint is essential if neonatal and paediatric work is undertaken and a 5–8 MHz frequency will be required.

A biopsy attachment may be needed for invasive procedures, and, depending on the range of work to be undertaken, linear probes, endoprobes, intraoperative probes and other designs can be considered.

Image quality

There are very few applications where this is not of paramount importance and abdominal scanning requires the very best you can afford. A machine capable of producing a high-quality image is likely

Figure 1.11 Curved arrays (left and centre) suitable for abdominal scanning. A 5 MHz linear array (right) is useful for superficial structures, e.g. gallbladder and anterior abdominal wall.

to remain operational for much longer than one capable of only poor quality, which will need replacement much sooner. A poor-quality image is a false economy in abdominal scanning.

Machine capabilities and functions

The availability and ease of use of various functions differ from machine to machine. Some of the important issues to consider when buying a machine include:

- probe selection and switching process, simultaneous connection of several probes
- dynamic frequency capability
- dynamic focusing control, number and pattern of focal zones
- functions such as beam steering, sector angle adjustment, zoom, frame rate adjustment, trackerball controls
- time gain compensation and power output controls
- cine facility—operation and size of memory
- programmable presets
- tissue harmonic and/or contrast harmonic imaging
- body marker and labelling functions
- measurement packages—operation and display
- colour/power and spectral Doppler through all probes
- Doppler sensitivity
- Doppler controls—ease of use, programmable presets
- output displays
- report package option.

Ergonomics

Good ergonomics contribute considerably to the success of the service provided. The machine must be usable by various operators in all the required situations. There is a significant risk of work-related musculoskeletal disorders (WRMSD)[2] if careful consideration is not given to the scanning environment (see p. 12). When choosing and setting up a scanning service, forethought should be given not only to the design of the ultrasound machine, but also to the seating arrangements and examination couch. These should all be adjustable in order to facilitate the best scanning position for the operator.

Other considerations include:

- System dimensions and steering. The requirement for the system to be portable, for example for ward or theatre work, or mobile for transportation to remote clinics. Machines used regularly for mobile work should be robust and easy to move.
- Moveable (swivel and tilt) monitor and control panel, including height adjustment for different operators and situations.
- Keyboard design, to facilitate easy use of the required functions, without stretching or twisting.
- Hand-held portable machines are an option that may be considered.

Maintenance issues

It is useful to consider the reliability record of the chosen equipment, particularly if it is to operate in out-reach clinics, or without available backup in the case of breakdown. Contacting other users may prove useful.

Various maintenance contract options and costs are available, including options on the replacement of probes, which should be taken into account when purchasing new equipment.

Upgradeability

A machine which is potentially upgradeable has a longer, more cost-effective life and will be supported by the manufacturer over a longer period of time. Consideration should be given to future software upgrades, possible effects and costs and other available options for the future, such as additional transducers or add-on Doppler facilities.

Links to image-recording devices

Most ultrasound machines are able to link up to most types of imaging facility, whether it be a simple black and white printer or a radiology-wide photographic archiving and communications (PAC) system. There may be costs involved, however, in linking your new machine to your preferred imaging device.

Equipment manufacturers now follow the DICOM standard. **D**igital **I**maging and **Co**mmunications in **M**edicine is the industry standard for transferring medical images and related information between computers. This facilitates compatibility between different pieces of equipment from different manufacturers and potentially enables them to be linked up.

RECORDING OF IMAGES

There are no hard and fast rules about the recording of ultrasound scans and departmental practices vary. It is good practice for departments to have guidelines for taking and retaining images within individual schemes of work, outlining the minimum expected.[3]

The advantages of recording images are:

- They provide a record of the quality of the scan and how it has been conducted: the organs examined, the extent of the scan, the type and standard of equipment, the settings used and other scanning factors. This can be an invaluable tool in providing a medicolegal defence.
- They provide an invaluable teaching aid.
- They help to ensure quality control within departments: promoting the use of good technique, they can be used to ensure protocols are followed and provide an excellent audit tool.
- They can be used to obtain a second opinion on difficult or equivocal cases and provide a basis for discussion with clinical colleagues.

The disadvantages are:

- The cost of buying, running and maintaining the recording device or system.
- The quality of images in some cases may not accurately reflect that of the image on the ultrasound monitor.
- The scanning time must be slightly increased to accommodate the taking of images.
- Storage and retrieval of images may be time- and space-consuming.
- Hard copy may be mislaid or lost.
- If the examination has been *badly* performed, the hard copy may demonstrate that too!

Generally speaking the recording of images is encouraged. It reduces the operator's vulnerability to litigation and supports the ultrasound diagnosis.[4] It is only possible to record the *entire* examination by using videotape, which is rarely practical in larger departments. The operator must take the responsibility for ensuring the scan has been performed to the required standard; any images produced for subsequent discussion are only *representative* of the examination and have been chosen by the operator as an appropriate selection. If you have missed a small metastasis in the liver while scanning, or a gallstone in the gallbladder, you are unlikely to have included it on an image.

Choice of image-recording device depends on many factors. Considerations include:

- image quality—resolution, grey-scale, storage life
- capital cost of the system—including the installation together with the installation of any other necessary equipment, such as a processor
- cost of film
- processing costs if applicable—this includes the cost of chemicals, the cost of buying and maintaining a processor and possibly a chemical mixer
- maintenance costs
- reliability of the system
- storage of images in terms of available space and cost
- location and size of the imaging system
- other considerations
  —ease of use
  —mobility
  —colour capability
  —ability to produce slides/teaching aids
  —shelf life of unused film and stored images.

Numerous methods of recording images are available to suit all situations. Small printers, attached to ultrasound scanners, are easy to use, cheap to buy and run and convenient if the machine is used on wards or distant satellite units. However, systems which produce hard copy, however good, are inevitably of inferior image quality to electronic image capture.

Multi-system departments are tending towards networked systems which produce high-quality images, and can be linked to multiple machines and modalities. These are, of course, more expensive to purchase and install, but are generally reliable and produce consistent, high-quality image.

Ultimately, the goal of the filmless department is being realized in PACS (photographic archiving and communications systems). Digital imaging networks are convenient, quick and relatively easy to use. The image quality is excellent, suffering little or no degradation in capture and subsequent retrieval, and the system can potentially be linked to a conventional imager should hard copy be required.

The number of workstations in the system can be virtually unlimited, depending on the system, affording the operator the flexibility of transmitting images immediately to remote locations, for example clinical meetings, outpatient clinics, etc. It is also possible to download images from scans done with mobile equipment, remote from the main department, on to the PACS.

Digital storage and retrieval avoid loss of films and afford considerable savings in time, labour and space. Increasingly it is also possible to store moving clips—useful for dynamic studies such as those involving contrast agents and for teaching purposes.

Many systems also incorporate a patient registration and reporting package, further streamlining the ultrasound examination. Not all systems store images in colour and there are considerable differences between the facilities available on different systems. The potential purchaser is advised to plan carefully for the needs of the ultrasound service.

The capital costs for PACS are high, but these can, to a certain extent, be offset by subsequently low running costs and potential savings in film, processing materials, equipment maintenance, and manual storage and retrieval.

SAFETY OF DIAGNOSTIC ULTRASOUND

Within the field of clinical diagnostic ultrasound, it is currently accepted that there is insufficient evidence for any deleterious effects at diagnostic levels and that the benefits to patients outweigh the risks. As new techniques and technological developments come on to the market, new biophysical conditions may be introduced which require evaluation with regard to safety[5] and we cannot afford to become complacent about the possible effects. The situation remains under constant review.

Several international bodies continue to consider the safety of ultrasound in clinical use. The European Federation of Societies for Ultrasound in Medicine and Biology (EFSUMB) has confirmed the safety of diagnostic ultrasound and endorsed its 'informed' use.[6] Whilst the use of pulsed Doppler is considered inadvisable for the developing embryo during the first trimester, no such exceptions are highlighted for abdominal ultrasound.

The European Committee for Ultrasound Radiation Safety (ECURS) confirms that no deleterious effects have yet been proven in clinical medicine. It recommends, however, that equipment is used only when designed to national or international safety standards and that it is used only by competent and trained personnel.

The World Federation for Ultrasound in Medicine and Biology (WFUMB) confirms that the use of B-mode imaging is not contraindicated,[7] concluding that exposure levels and duration should be reduced to the minimum necessary to obtain the required diagnostic information.

Ultrasound intensities used in diagnostic ultrasound vary according to the mode of operation. Pulsed Doppler usually has a higher level than B-mode scanning, which operates at lower intensities, although there may be overlap with colour or power Doppler.

The American Institute for Ultrasound in Medicine (AIUM) has suggested that ultrasound is safe below 100 W/cm.[8] This figure refers to the spatial peak temporal average intensity (I_{SPTA}).

The use of intensity, however, as an indicator of safety is limited, particularly where Doppler is concerned, as Doppler intensities can be considerably greater than those in B-mode imaging. The Food and Drug Administration (FDA) sets maximum intensity levels allowed for machine output, which differ according to the application.[9]

Biological effects of ultrasound

Harmful effects from ultrasound have been documented in laboratory conditions. These include thermal effects and mechanical effects.

Thermal effects are demonstrated as a slight rise in temperature, particularly in close proximity to the transducer face, during ultrasound scanning. This local effect is usually of no significance but the operator must be aware of the phenomenon. The most significant thermal effects occur at bone/tissue interfaces and are greater with pulsed Doppler. Increases in temperature of up to 5°C have been produced. Areas at particular risk are fetal bones and the interfaces in transcranial Doppler ultrasound scans.

Pulsed Doppler has a greater potential for heating than B-mode imaging as it involves greater temporal average intensities due to high pulse repetition frequency (PRF) and because the beam is frequently held stationary over an area while obtaining the waveform. Colour and power Doppler usually involve a greater degree of scanning and transducer movement, which involves a potentially lower heating potential than with pulsed Doppler. Care must be taken to limit the use of pulsed Doppler and not to hold the transducer stationary over one area for too long.

Mechanical effects, which include cavitation and radiation pressure, are caused by stresses in the tissues and depend on the amplitude of the ultrasound pulse. These effects are greatest around gas-filled organs, such as lungs or bowel and have, in laboratory conditions, caused small surface blood vessels in the lungs to rupture. Potentially, these effects could be a hazard when using contrast agents which contain microbubbles.

Safety indices (thermal and mechanical indices)

In order to inform users about the machine conditions which may potentially be harmful, mechanical and thermal indices are now displayed as an output display standard (ODS) on all equipment manufactured after 1998. This makes operators aware of the ultrasound conditions which may exceed the limits of safety and enables them to take avoiding action, such as reducing the power or restricting the scanning time in that area.

In simple terms the mechanical index (MI) is related to amplitude and indicates how 'big' an ultrasound pulse is, giving an indication of the chances of mechanical effects occurring. It is therefore particularly relevant in the abdomen when scanning gas-filled bowel or when using microbubble contrast agents. Gas bodies introduced by contrast agents increase the probablility of cavitation.

The thermal index (TI) gives an indication of the temperature rise which might occur within the ultrasound beam, aiming to give an estimate of the reasonable worst-case temperature rise. The TI calculation alters, depending upon the application, giving rise to three indices: the soft-tissue thermal index (TIS), the bone-at-focus index (TIB) and the bone-at-surface, or cranial index (TIC). The first of these is obviously most relevant for abdominal applications. In well-perfused tissue, such as the liver and spleen, thermal effects are less likely due to the cooling effect of the blood flow.

The display of safety indices is only a general indication of the possibility of biological hazards and cannot be translated directly into real heating or cavitation potential.[10] These 'safety indices' are limited in several ways. They require the user to be educated with respect to the implications of the values shown and they do not take account of the duration of exposure, which is particularly important in assessing the risk of thermal damage.[4] In addition, the TI does not take account of the patient's temperature, and it is logical to assume that increased caution is therefore required in scanning the febrile patient.

MI and TI are also unlikely to portray the optimum safety information during the use of contrast agents, in which, theoretically, heating effects and cavitation may be enhanced.[5]

Other hazards

Whilst most attention in the literature is focused on the possible biological effects of ultrasound, there are several other safety issues which are within the control of the operator.

Electrical safety All ultrasound machines should be subject to regular quality control and should be regularly checked for any signs of electrical hazards. Loose or damaged wiring, for example, is a common problem if machines are routinely used for mobile work. Visible damage to a transducer, such as a crack in the casing, should prompt its immediate withdrawal from service until a repair or replacement is effected.

Microbiological safety It is the responsibility of the sonographer to minimize the risks of cross-infection. Most manufacturers make recommendations regarding appropriate cleaning agents for transducers, which should be carefully followed. Sterile probe covers should be used in cases where there is an increased risk of infection.

Operator safety By far the most serious hazard of all is that of the untrained or badly trained operator. Misdiagnosis is a serious risk for those not aware of the pitfalls. Apart from the implications for the patient of subsequent incorrect management, the operator risks litigation which is difficult or impossible to defend if they have had inadequate training in ultrasound.

Work-related musculoskeletal disorders

There is increasing concern about WRMSD related to ultrasound scanning, as workloads increase and it has been estimated that a significant proportion of sonographers who practise full-time ultrasound scanning may be affected.[2] One contributing factor is the ergonomic design of the ultrasound machines, together with the position adopted by the operator during scanning. While more attention is now being paid by ultrasound manufacturers to designs which limit WRMSD, there are various other contributing factors which should be taken into account when providing ultrasound services. Well-designed, adjustable seating for operators, adjustable patient couches, proper staff training for manoeuvring patients and a varied work load all contribute to minimizing the potential problems to staff.

Hand-held, portable ultrasound machines are now available. Provided they are of sufficient functionality to provide the service required, they may also potentially limit the problems encountered when manoeuvring larger scanners around hospital wards and departments.

The safe practice of ultrasound

It is fair to say that the safety of ultrasound is less of an issue in abdominal scanning than in obstetric or reproductive organ scanning. Nevertheless it is still incumbent upon the operator to minimize the ultrasound dose to the patient in any practicable way.

The use of X-rays is governed by the ALARA principle—that of keeping the radiation dose As Low As Reasonably Achievable. Although the risks associated with radiation are not present in the use of ultrasound, the general principle of keeping the acoustic exposure as low as possible is still good practice and many people still refer to ALARA in the context of diagnostic ultrasound (see Box 1.3).

MEDICOLEGAL ISSUES

Litigation in medical practice is increasing and the field of ultrasound is no exception to this. Although currently the majority of cases involve firstly obstetric and secondly gynaecological ultrasound, it is prudent for the operator to be aware of the need to minimize the risks of successful litigation in all types of scanning procedures.

Patients have higher expectations of medical care than ever before and ultrasound practitioners should be aware of the ways in which they can protect themselves should a case go to court. The

Box 1.3 Steps for minimizing the ultrasound dose

- Ensure operators are *properly trained*, preferably on recognized training programmes.
- Minimize the output (or power) level. Use amplification of the *received* echoes to manipulate the image in preference to increasing the transmitted power.
- Minimize the time taken to perform the exam.
- Don't rest the transducer on the skin surface when not scanning.
- Make sure the clinical indications for the scan are satisfactory and that a proper request has been received. Don't do unnecessary ultrasound examinations.
- Be aware of the safety indices displayed on the ultrasound machine. Limit the use of pulsed Doppler to that necessary to contribute to the diagnosis.
- Make the best use of your equipment—maximize the diagnostic information by manipulating the controls effectively.

onus is upon the defendant to prove that he or she acted responsibly and there are several helpful guidelines which should routinely be followed (see Box 1.4).[11]

The medicolegal issues surrounding ultrasound may be different according to whether the operator is medically or non-medically qualified. Depending on their profession, operators are constrained by codes of conduct of their respective colleges and/or Councils.[12] Either way, the operator is legally accountable for his or her professional actions.

If non-medically qualified personnel are to perform and report on scans (as happens in the UK, USA and Australia), this task must be properly delegated by a medically qualified practitioner, for example a radiologist in the case of abdominal scanning. As the role of sonographers continues to expand, it is noteworthy that the same standard of care is expected from medically and non-medically qualified staff alike.[13] To avoid liability, practitioners must comply with the Bolam test, in which they should be seen to be acting in accordance with practice accepted as proper by a responsible body of relevant medical people.

Box 1.4 Guidelines for defensive scanning *(adapted from Meire HB[11])*

- Ensure you are properly trained. Operators who have undergone approved training are less likely to make mistakes.
- Act with professionalism and courtesy. Good communication skills go a long way to avoiding litigation.
- Use written guidelines or schemes of work.
- Ensure a proper request for the examination has been received.
- A written report should be issued by the operator.
- Record images to support your findings.
- Clearly state any limitations of the scan which may affect the ability to make a diagnosis.
- Make sure that the equipment you use is adequate for the job.

DEPARTMENTAL GUIDELINES/SCHEMES OF WORK

It is generally considered good and safe practice to use written guidelines for ultrasound examinations.[3]

These serve several purposes:

- They may be used to support a defence against litigation (provided, of course, that the operator can prove he or she has followed such guidelines).
- They serve to impose and maintain a minimum standard, especially within departments which may have numerous operators of differing experience levels.
- They serve to inform operators of current practice.

Guidelines should ideally be:

- Written by, and have input from, those practising ultrasound in the department (usually a combination of medically and non-medically qualified personnel), taking into account the requirements of referring clinicians, available equipment and other local operational issues.
- Regularly reviewed and updated to take account of the latest literature and practices.
- Flexible, to allow the operator to tailor the scan to the patient's clinical presentation and individual requirements.

Guidelines which are too prescriptive and detailed are likely to be ignored by operators as impractical. The guidelines should be broad enough to allow operators to respond to different clinical situations in an appropriate way while ensuring that the highest possible standard of scan is always performed. In cases when it is simply not possible to adhere to departmental guidelines, the reasons should be stated on the report, for example when the pancreas cannot be demonstrated due to body habitus or overlying bowel gas.

QUALITY ASSURANCE

The principles of quality assurance affect various aspects of the ultrasound service offered. These

include staff issues (such as education and training, performance and continuing professional development), patient care, the work environment (including health and safety issues) and quality assurance of equipment. Quality assurance checks on ultrasound equipment, unlike most other aspects of an ultrasound service, involve measurable and reproducible parameters.

Equipment tests

After installation, a full range of equipment tests and safety checks should be carried out and the results recorded. This establishes a baseline performance against which comparisons may later be made. These tests should normally be carried out by qualified medical physicists.

It is useful to take a hard-copy image of a tissue-mimicking phantom, with the relevant settings marked on it. These images form a reference against which the machine's subsequent performance can be assessed. If your machine seems to be performing poorly, or the image seems to have deteriorated in some way, you will have the proof you require.

A subsequent, regular testing regime must then be set up, to ensure the standards of quality and safety are maintained. This programme can be set up in conjunction with the operators and the medical physics department and relevant records should be kept. The use of a tissue-mimicking phantom enables the sonographer to perform certain tests in a reproducible and recordable manner (Fig. 1.12).

Checks should be carried out for all probes on the machine.

Suggested equipment checks include:

- caliper accuracy
- system sensitivity and penetration
- axial and lateral resolution
- slice thickness
- grey scale
- dead zone
- checks on the various machine controls/functions
- output power
- safety checks: electrical, mechanical, biological and thermal, including a visual inspection of all probes and leads
- imaging device checks for image quality, settings, dynamic range, functionality and electrical safety

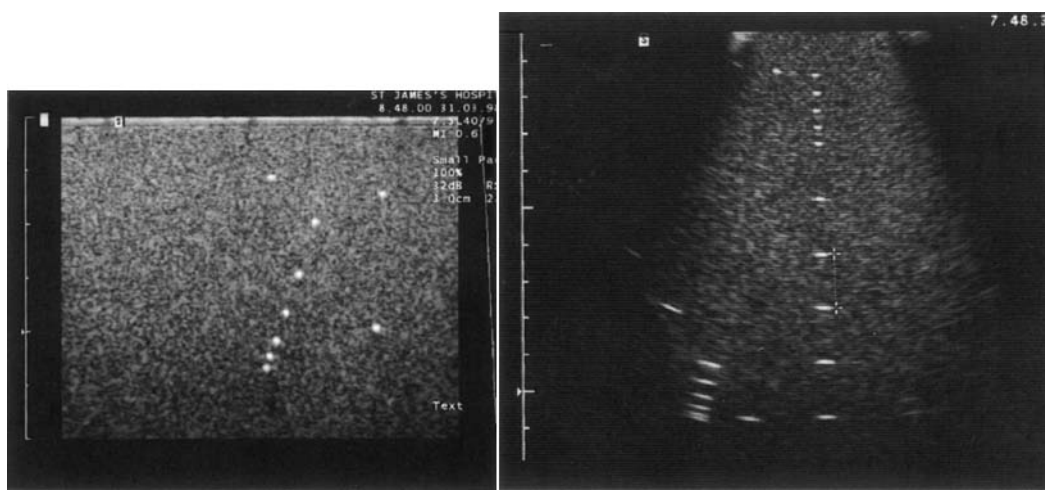

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Figure 1.12 Tissue-mimicking phantom. (A) When using a high-frequency linear array, cross-sections of the wires in the phantom are clearly demonstrated as small dots. (B) When using a curved array of a lower frequency, such as that used for abdominal scanning, the lateral resolution is seen to deteriorate in the far field as the beam diverges. The wires are displayed correctly in the near field but appear as short lines in the far field. Spacing of the wires is known, allowing caliper accuracy to be assessed.

- biopsy guide checks
- colour, power and spectral Doppler checks (complex, requiring specialized equipment).

Some of these checks can be easily and quickly carried out by users in the department on a regular basis, for example caliper checks and biopsy guide checks. Others are more complex and may be appropriately undertaken by specialist medical physicists. All equipment should undergo regular servicing and any interim faults should naturally be reported.

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Chapter 2

The normal hepatobiliary system

CHAPTER CONTENTS

INTRODUCTION

Ultrasound is the dominant first-line investigation for an enormous variety of abdominal symptoms because of its non-invasive and comparatively accessible nature. Its success, however, in terms of a diagnosis, depends upon numerous factors, the most important of which is the *skill of the operator*.

Because of their complexity and extent, the normal appearances and haemodynamics of the hepatobiliary system are dealt with in this chapter, together with some general upper-abdominal scanning issues. The normal appearances of the other abdominal organs are included in subsequent relevant chapters.

It is good practice, particularly on the patient's first attendance, to scan the whole of the upper abdomen, focusing particularly on the relevant areas, but also excluding or identifying any other significant pathology. A full abdominal survey would normally include the liver, gallbladder, biliary tree, pancreas, spleen, kidneys and retroperitoneal structures. Apart from the fact that many pathological processes can affect multiple organs, a number of significant (but clinically occult) pathological processes are discovered incidentally, for example renal carcinoma or aortic aneurysm. A thorough knowledge of anatomy is assumed at this stage, but diagrams of upper abdominal sectional anatomy are included in the appendix to this chapter for quick reference (see pp. 36–39).

It is important always to remember the operator-dependent nature of ultrasound scanning (see Chapter 1); although the dynamic nature of the scan is a huge advantage over other forms of

imaging, the operator must continuously adjust technique to obtain the maximum diagnostic information. In any abdominal ultrasound survey the operator assesses the limitations of the scan and the level of confidence with which pathology can be excluded or confirmed. The confidence limits help in determining the subsequent investigations and management of the patient.

It is important, too, to retain an open mind about the diagnosis when embarking on the scan; an operator who decides the likely diagnosis on a clinical basis may sometimes be correct but, in trying to fit the scan to match the symptoms, risks missing significant pathology.

GENERAL POINTERS ON UPPER-ABDOMINAL TECHNIQUE

Scanning technique is not something that can be learnt from a book. There is absolutely no substitute for regular practical experience under the supervision of a qualified ultrasound practitioner.

There are, however, some general approaches which help to get the best from the scanning procedure:

- Scan in a systematic way to ensure the whole of the upper abdomen has been thoroughly interrogated. The use of a worksheet, which indicates the structures to be examined, is advisable when learning.[1]
- Always scan any organ in *at least* two planes, preferably at right angles to each other. This reduces the risk of missing pathology and helps to differentiate artefact from true pathology.
- Where possible, scan in at least two patient positions. It is surprising how the available ultrasound information can be enhanced by turning your patient oblique, decubitus or erect. Inaccessible organs flop into better view and bowel moves away from the area of interest.
- Use a combination of sub- and intercostal scanning for all upper-abdominal scanning. The different angles of insonation can reveal pathology and eliminate artefact.
- Don't limit yourself to longitudinal and transverse sections. Use a variety of planes and angulations. Trace ducts and vessels along their courses. Use the transducer like a pair of eyes.
- Deep inspiration is useful in a proportion of patients, but not all. Sometimes it can make matters worse by filling the stomach with air and obscuring large areas. An intercostal approach with the patient breathing gently often has far more success.
- Positioning patients supine, particularly if elderly or very ill, can make them breathless and uncomfortable. Raise the patient's head as much as necessary; a comfortable patient is much easier to scan.
- Images are a useful record of the scan and how it has been performed, but don't make these your primary task. *Scan first*, sweeping smoothly from one aspect of the organ to the other in two planes, then take the relevant images to support your findings.
- Make the most of your equipment (see Chapter 1). Increase the confidence level of your scan by fully utilizing all the available facilities, using Doppler, tissue harmonics, changing transducers and frequencies and manipulating the machine settings and processing options.

THE LIVER

Normal appearance

The liver is a homogeneous, mid-grey organ on ultrasound. It has the same, or slightly increased echogenicity when compared to the cortex of the right kidney. Its outline is smooth, the inferior margin coming to a point anteriorly (Fig. 2.1). The liver is surrounded by a thin, hyperechoic capsule, which is difficult to see on ultrasound unless outlined by fluid (Fig. 2.2).

The smooth parenchyma is interrupted by vessels (see below) and ligaments (Figs 2.3–2.15) and the liver itself provides an excellent acoustic window on to the various organs and great vessels situated in the upper abdomen.

The ligaments are hyperechoic, linear structures; the falciform ligament, which separates the anatomical left and right lobes is situated at the

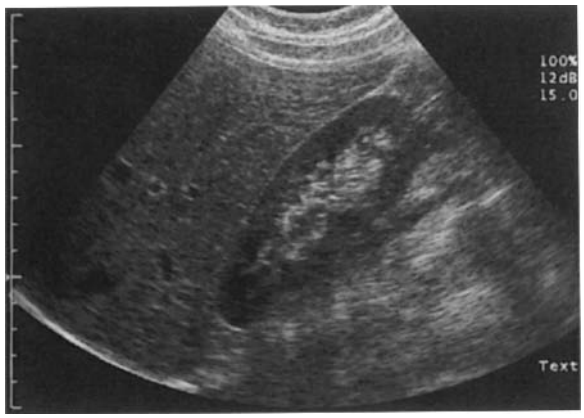

Figure 2.1 Longitudinal section (LS) through the right lobe of the liver. The renal cortex is slightly less echogenic than the liver parenchyma.

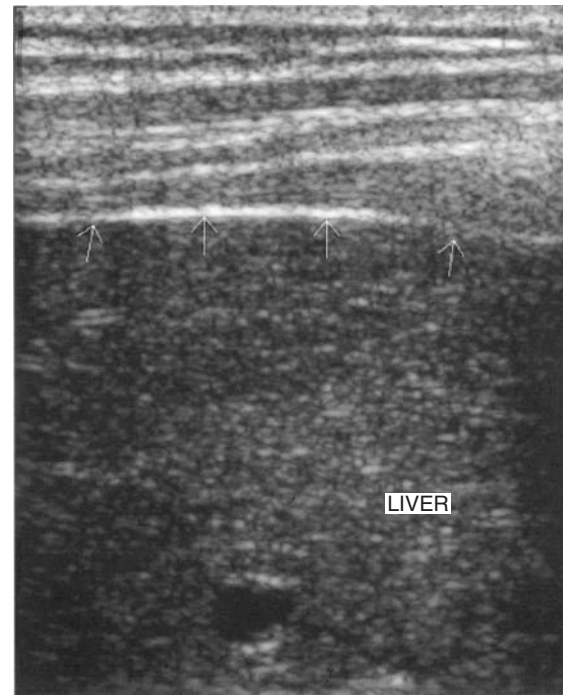

Figure 2.2 The capsule of the liver (arrows) is demonstrated with a high-frequency (7.5 MHz) probe.

superior margin of the liver and is best demonstrated when surrounded by ascitic fluid. It surrounds the left main portal vein and is known as the ligamentum teres as it descends towards the infero-anterior aspect of the liver (Figs 2.9 and 2.15). The ligamentum venosum separates the caudate lobe from the rest of the liver (Fig. 2.6).

The size of the liver is difficult to quantify, as there is such a large variation in shape between normal subjects and direct measurements are notoriously inaccurate. Size is therefore usually assessed subjectively. Look particularly at the inferior margin of the right lobe which should come to a point anterior to the lower pole of the right kidney (Fig. 2.1). A relatively common variant of this is the *Reidel's lobe*, an inferior elongation of segment VI on the right. This is an extension of the right lobe over the lower pole of the kidney, with a rounded margin (Fig. 2.16), and is worth remembering as a possible cause of a palpable right upper quadrant 'mass'.

To distinguish mild enlargement from a Reidel's lobe, look at the left lobe. If this also looks bulky, with a rounded inferior edge, the liver is enlarged. A Reidel's lobe is usually accompanied by a smaller, less accessible left lobe.

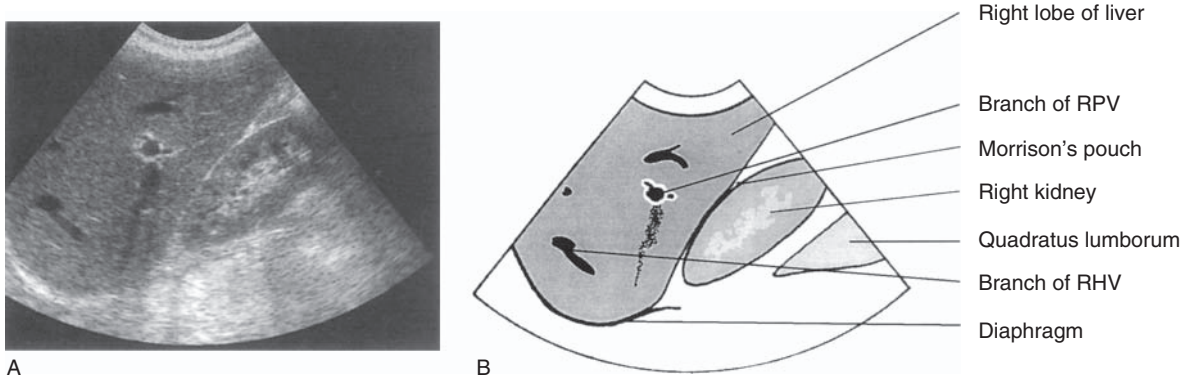

Figure 2.3 LS through the right lobe of the liver and right kidney. RPV = right portal vein; RHV = right hepatic vein.

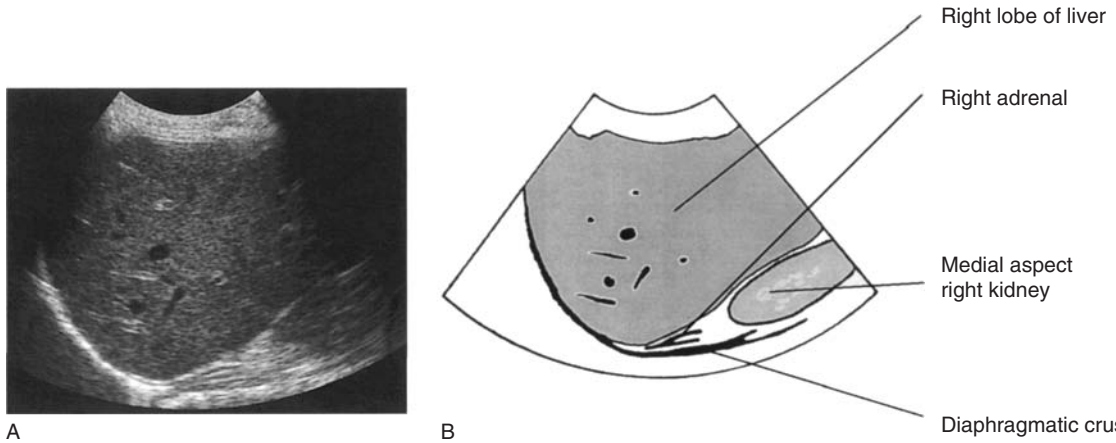

Figure 2.4 LS, right lobe, just medial to the right kidney.

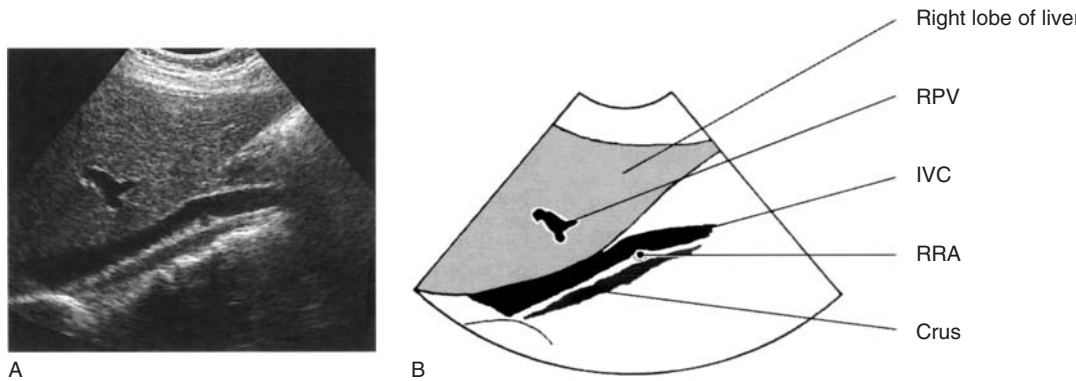

Figure 2.5 LS, right lobe, angled medially towards the inferior vena cava (IVC). RRA = right renal artery.

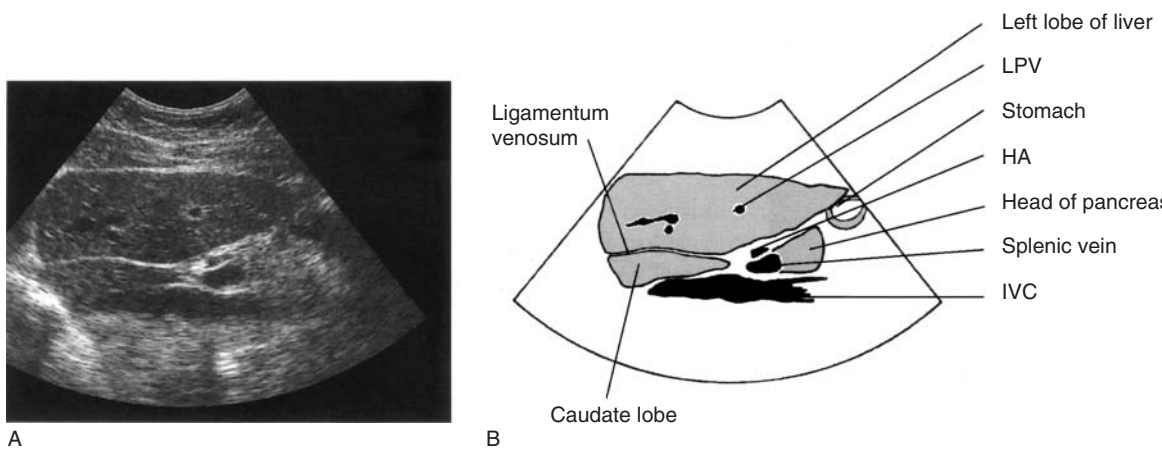

Figure 2.6 LS, midline, through the left lobe, angled right towards the IVC. LPV = left portal vein; HA = hepatic artery.

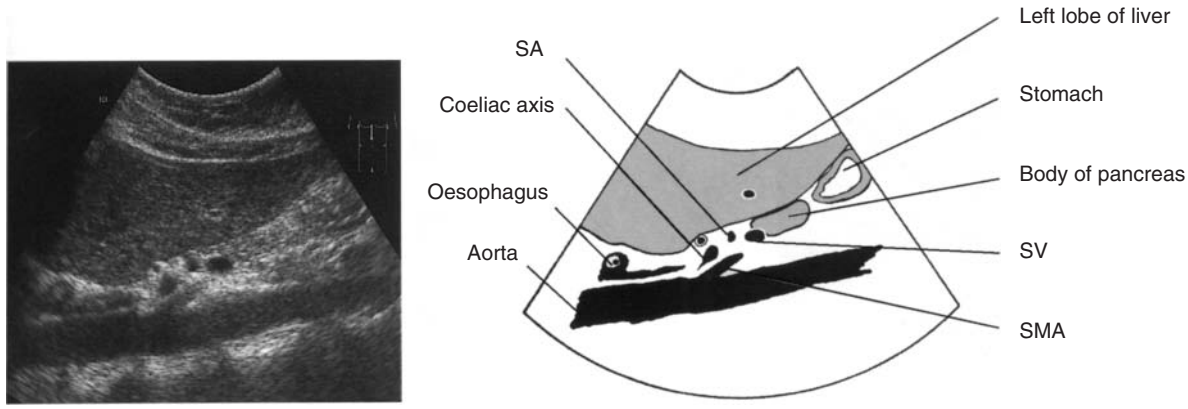

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Figure 2.7 LS through the midline. SV = splenic vein; SA = splenic artery; SMA = superior mesenteric artery.

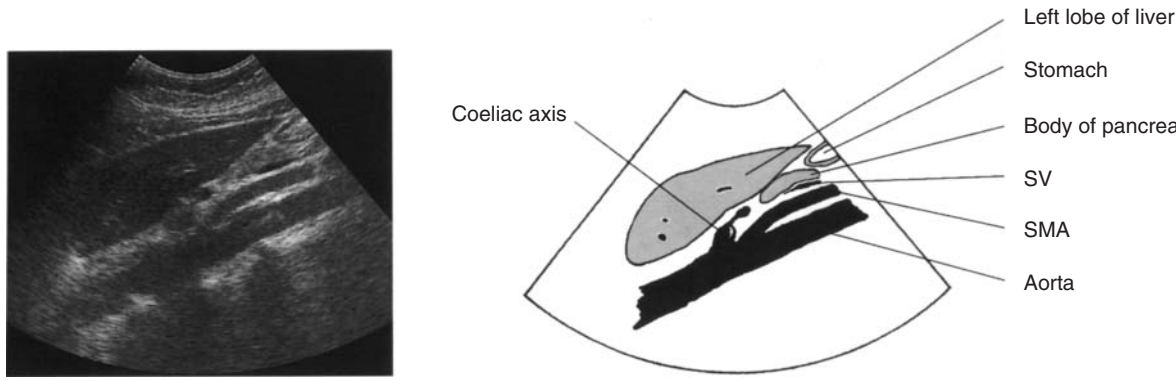

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Figure 2.8 LS just to the left of midline.

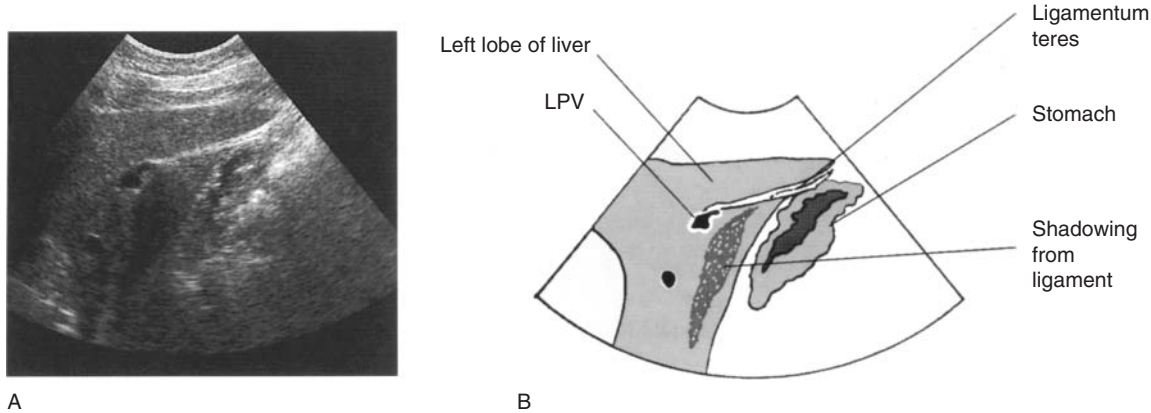

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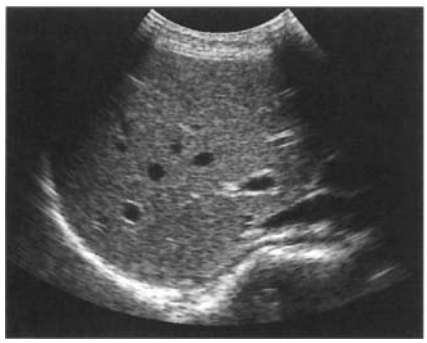
Figure 2.9 LS, left lobe of liver.



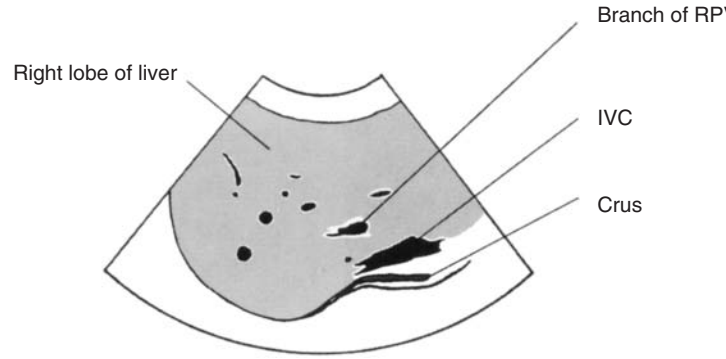


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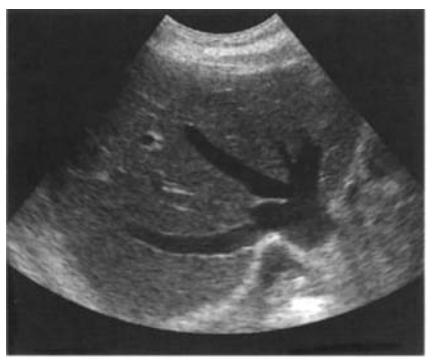
Figure 2.10 Transverse section (TS) through the liver, above the confluence of the hepatic veins.



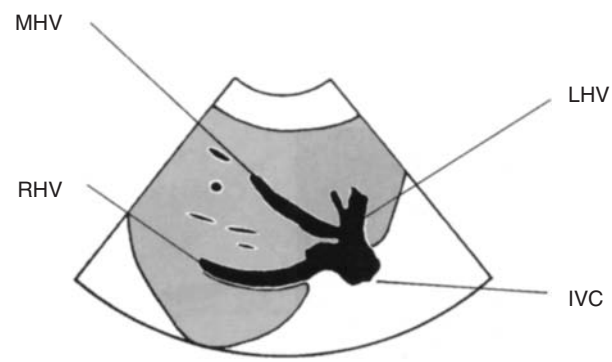


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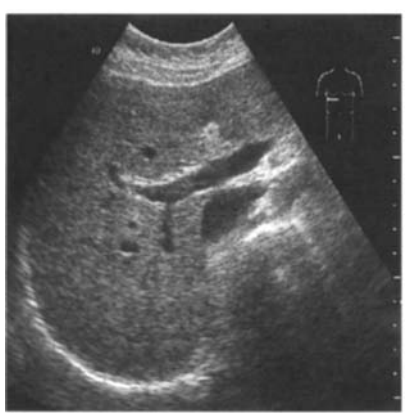
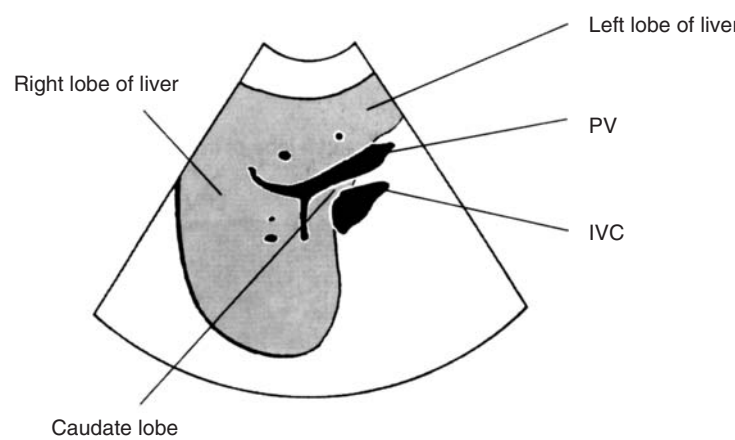
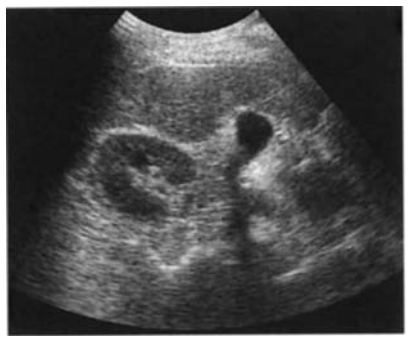
Figure 2.11 TS at the confluence of the hepatic veins (HV).

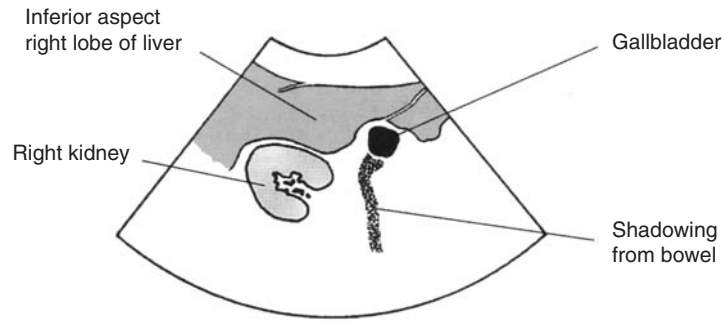




A B

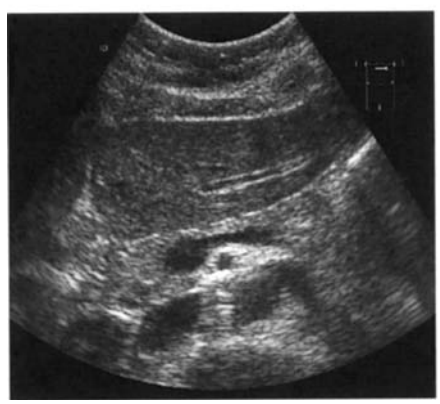
Figure 2.12 TS at the porta hepatis. PV = portal vein.

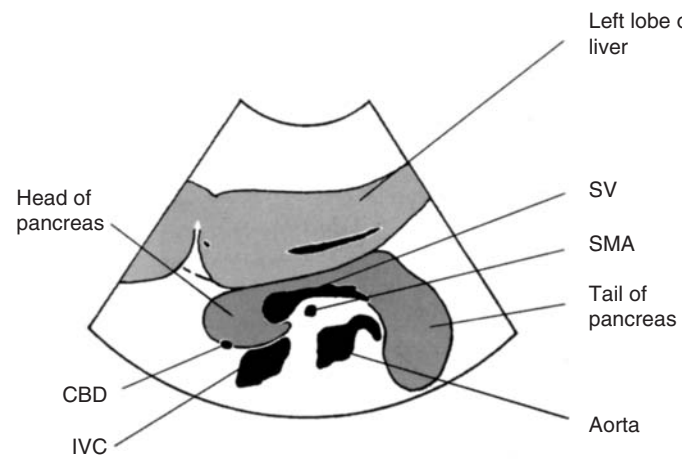




A B

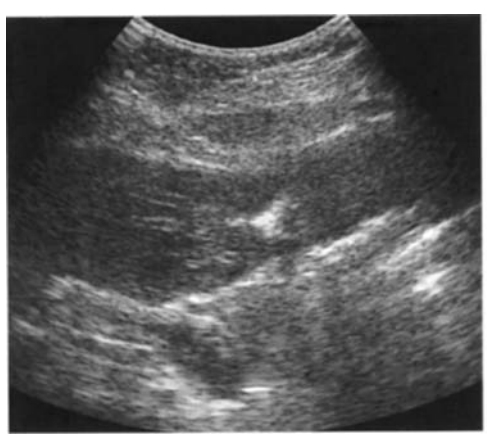
Figure 2.13 TS through the right kidney.

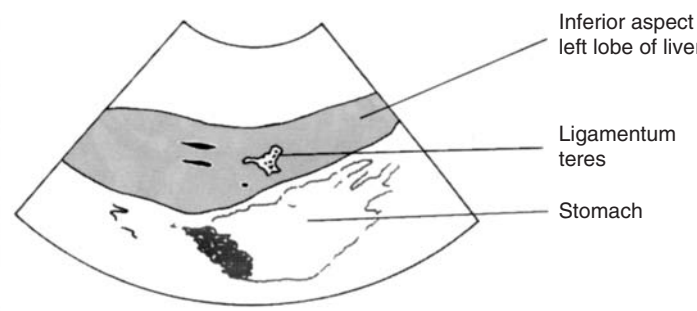




A B

Figure 2.14 TS at the epigastrium. CBD = common bile duct.





A B

Figure 2.15 TS at the inferior edge of the left lobe.

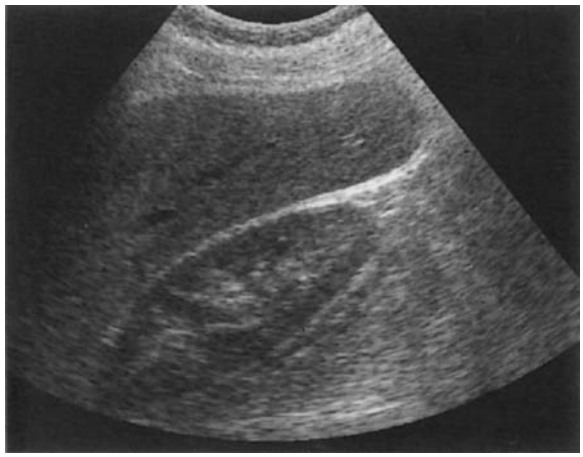


Figure 2.16 LS through the right lobe, demonstrating a Reidel's lobe extending below the right kidney. (Compare with the normal liver in Figure 2.1.)

The segments of the liver

It is often sufficient to talk about the 'right' or 'left' lobes of the liver for the purposes of many diagnoses. However, when a focal lesion is identified, especially if it may be malignant, it is useful to locate it precisely in terms of the surgical segments. This allows subsequent correlation with other imaging, such as computerized tomography (CT) or magnetic resonance imaging (MRI), and is invaluable in planning surgical procedures.

The segmental anatomy system, proposed by Couinaud in 1954,[2] divides the liver into eight segments, numbered in a clockwise direction. They are divided by the portal and hepatic veins and the system is used by surgeons today when planning surgical procedures (Fig. 2.17). This system is also used when localizing lesions with CT and MRI.

Identifying the different segments on ultrasound requires the operator to form a mental three-dimensional image of the liver. The dynamic nature of ultrasound, together with the variation in planes of scan, makes this more difficult to do than for CT or MRI. However, segmental localization of hepatic lesions by an experienced operator can be as accurate with ultrasound as with MRI.[3] Systematic scanning through the liver, in transverse section, identifies the main landmarks of the hepatic veins (Fig. 2.11) separating segments VII, VIII, IV and II in the superior part of the liver. As the transducer is moved inferiorly, the portal vein appears, and below this segments V and VI are located.

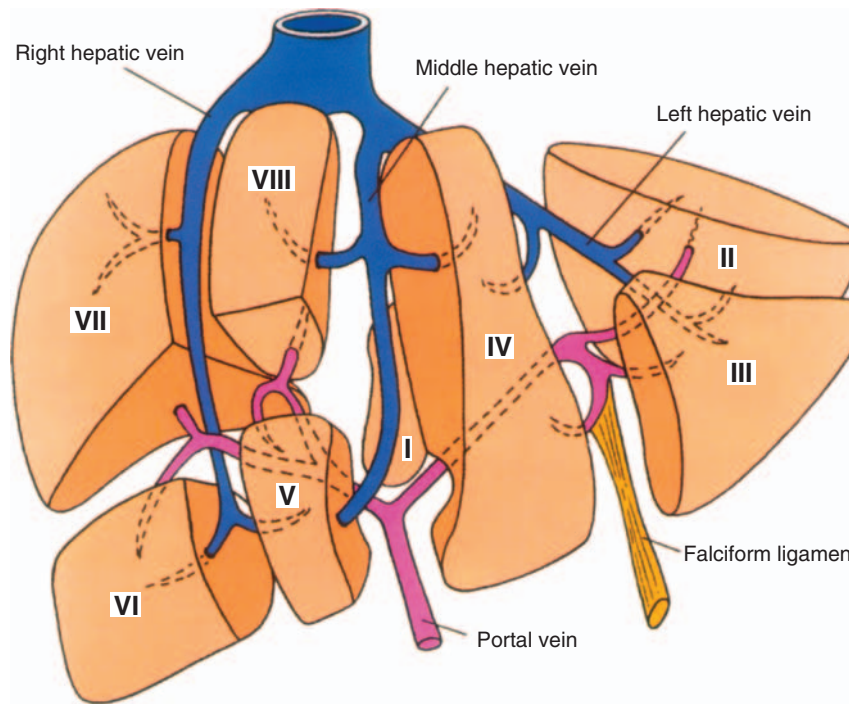


Figure 2.17 The surgical segments of the liver (after Couinaud[2]).

Hepatic vasculature

The *portal veins* radiate from the porta hepatis, where the main portal vein (MPV) enters the liver (Fig. 2.18). They are encased by the hyperechoic, fibrous walls of the portal tracts, which make them stand out from the rest of the parenchyma. Also contained in the portal tracts are a branch of the hepatic artery and a biliary duct radical. These latter vessels are too small to detect by ultrasound in the peripheral parts of the liver, but can readily be demonstrated in the larger, proximal branches (Fig. 2.19).

At the porta, the *hepatic artery* generally crosses the anterior aspect of the portal vein, with the common duct anterior to this (Fig. 2.20). In a common variation the artery lies anterior to the duct. Peripherally, the relationship between the vessels in the portal tracts is variable, (Fig. 2.21).

The three main *hepatic veins*, left, middle and right, can be traced into the inferior vena cava (IVC) at the superior margin of the liver (Fig. 2.11). Their course runs, therefore, approximately perpendicular to the portal vessels, so a section of liver with a longitudinal image of a hepatic vein is likely to contain a transverse section through a portal vein, and vice versa.

Unlike the portal tracts, the hepatic veins do not have a fibrous sheath and their walls are therefore less reflective. Maximum reflectivity of the vessel walls occurs with the beam perpendicular (Fig. 2.22).

The anatomy of the hepatic venous confluence varies. In most cases the single, main right hepatic vein (RHV) flows directly into the IVC, and the middle and left have a common trunk. In 15–35% of patients the left hepatic vein (LHV) and middle hepatic vein (MHV) are separate. This usually has no significance to the operator. However, it may be a significant factor in planning and performing hepatic surgery, especially tumour resection, as the surgeon attempts to retain as much viable hepatic tissue as possible with intact venous outflow (Fig. 2.23).[4]

Haemodynamics of the liver

Pulsed and colour Doppler to investigate the hepatic vasculature are now established aids to diagnosis in the upper abdomen. Doppler should always be used in conjunction with the real-time image and in the context of the patient's presenting symptoms. Used in isolation it can be highly misleading. Familiarity with the normal Doppler

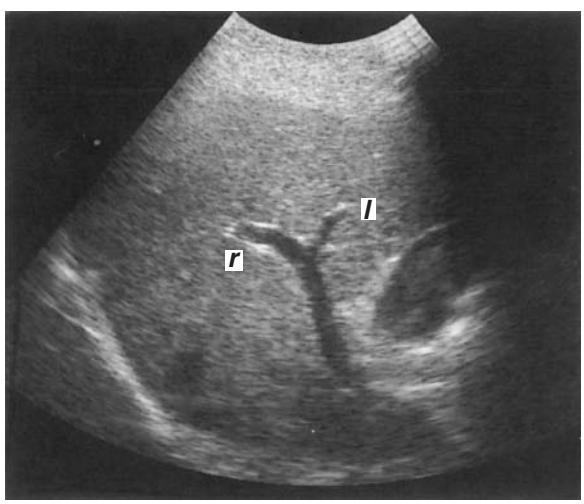


Figure 2.18 The right and left branches of the portal vein.

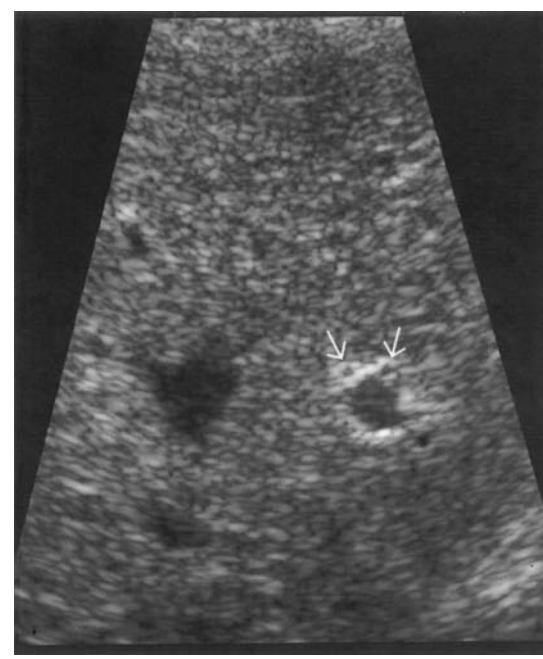
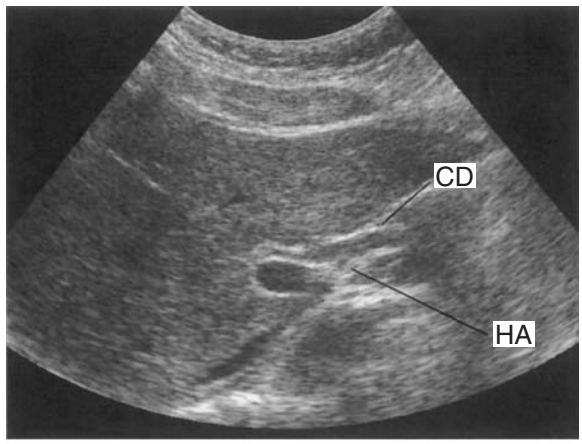
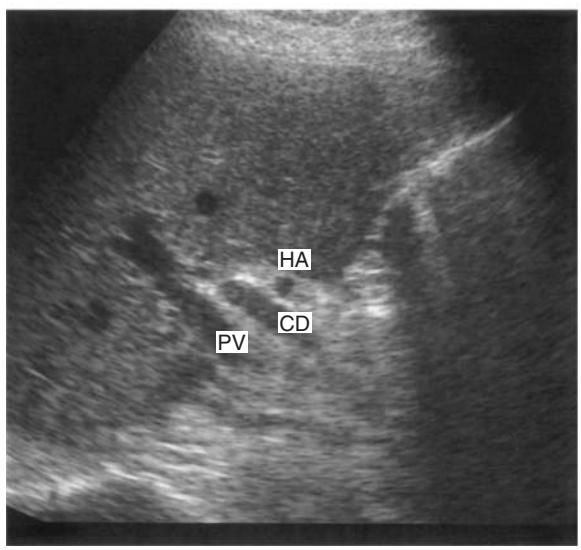


Figure 2.19 The portal vein radical is associated with a branch of the hepatic artery and a biliary duct (arrows) within the hyperechoic fibrous sheath.



A



B

Figure 2.20 (A) The porta hepatis. (B) A variant with the hepatic artery anterior to the duct. CD = common duct.

spectra is an integral part of the upper-abdominal ultrasound scan.

Doppler of the portal venous and hepatic vascular systems gives information on the patency, velocity and direction of flow. The appearance of the various spectral waveforms relates to the downstream resistance of the vascular bed (see Chapter 1).

The portal venous system

Colour Doppler is used to identify blood flow in the splenic and portal veins (Figs 2.24 and 2.25). The direction of flow is normally hepatopetal, that is towards the liver. The main, right and left portal branches can best be imaged by using a right oblique approach through the ribs, so that the course of the vessel is roughly towards the transducer, maintaining a low (< 60°) angle with the beam for the best Doppler signal.

The normal portal vein diameter is highly variable but does not usually exceed 16 mm in a resting state on quiet respiration.[5] The diameter increases with deep inspiration and also in response to food and to posture changes. An increased diameter may also be associated with portal hypertension in chronic liver disease (see Chapter 4). An absence of postprandial increase in diameter is also a sign of portal hypertension.

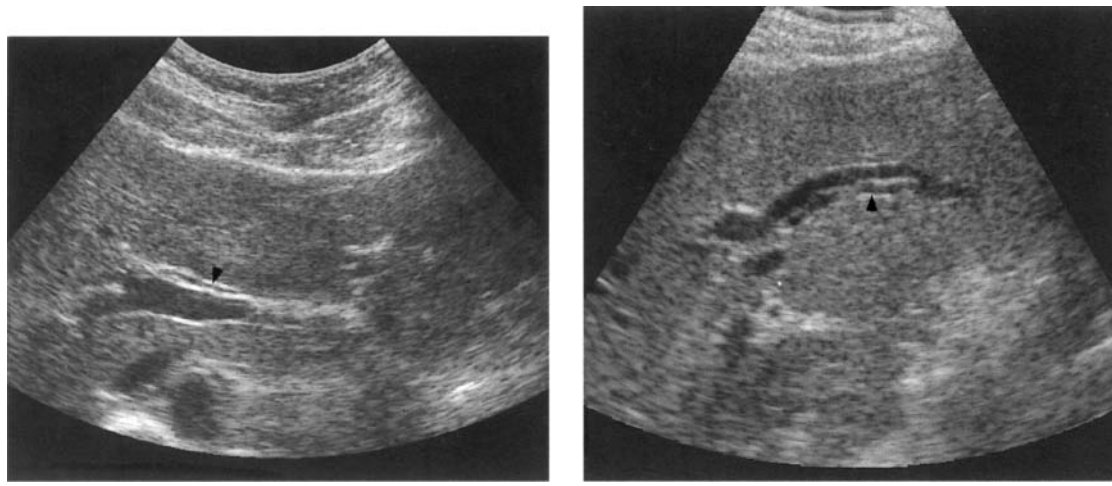
The normal portal vein (PV) waveform is monophasic (Fig. 2.26) with gentle undulations which are due to respiratory modulation and cardiac activity. This characteristic is a sign of the normal, flexible nature of the liver and may be lost in some fibrotic diseases.

The mean PV velocity is normally between 12 and 20 cm per second[6] but the normal range is wide. (A low velocity is associated with portal hypertension. High velocities are unusual, but can be due to anastomotic stenoses in transplant patients.)

The hepatic veins

The hepatic veins drain the liver into the IVC, which leads into the right atrium. Two factors shape the hepatic venous spectrum: the flexible nature of the normal liver, which can easily expand to accommodate blood flow, and the close proximity of the right atrium, which causes a brief 'kick' of blood back into the liver during atrial systole (Fig. 2.27). This causes the spectrum to be triphasic. The veins can be seen on colour Doppler to be predominantly blue with a brief red flash during atrial contraction. Various factors cause alterations to this waveform: heart conditions, liver diseases and extrahepatic conditions which compress the liver, such as ascites. Abnormalities of the hepatic vein waveform are therefore highly unspecific and should be taken in context with the clinical picture.

As you might expect, the pulsatile nature of the spectrum decreases towards the periphery of the liver, remote from the IVC.



A B

Figure 2.21 The relationship of the biliary duct to the portal vein varies as the vessels become more peripheral. In (A) the duct lies anterior to the LPV; in (B) the duct is posterior to the LPV.

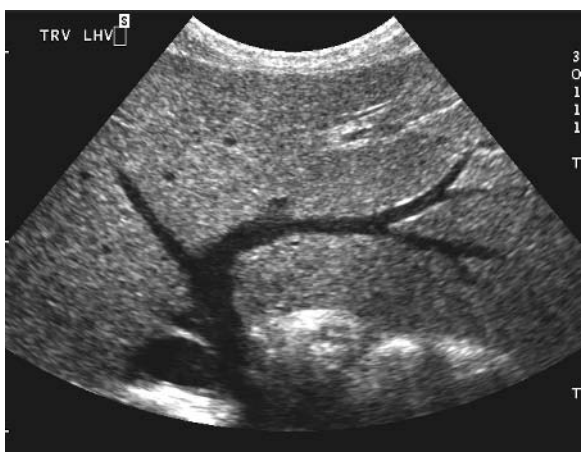


Figure 2.22 The left hepatic vein. Vessel walls are not as reflective as portal veins; however, maximum reflectivity is produced when the beam is perpendicular to the walls, as at the periphery of this vessel.

The hepatic artery

The main hepatic artery arises from the coeliac axis and carries oxygenated blood to the liver from the aorta. Its origin makes it a pulsatile vessel and the relatively low resistance of the hepatic vascular bed means that there is continuous forward flow throughout the cardiac cycle (Fig. 2.28). In a normal subject the hepatic artery may be elusive on colour Doppler due to its small diameter and tortuous course. Use the MPV as a marker, scanning from the right intercostal space to maintain a low angle with the vessel. The hepatic artery is just anterior to this and of a higher velocity (that is, it has a paler colour of red on the colour map (Fig. 2.24)).

THE GALLBLADDER

The normal gallbladder is best visualized after fasting, to distend it. It should have a hyperechoic, thin wall and contain anechoic bile (Fig. 2.29). Measure the wall thickness in a longitudinal section of the gallbladder, with the calipers perpendicular to the wall itself. (A transverse section may not be perpendicular to the wall, and can overestimate the thickness.)

After fasting for around six hours, it should be distended with bile into an elongated pear-shaped sac. The size is too variable to allow direct measurements to be of any use, but a tense, rounded shape can indicate pathological, rather than physiological dilatation.

Because the size, shape and position of the gallbladder are infinitely variable, so are the techniques required to scan it. There are, however, a number of useful pointers to maximize visualization of the gallbladder:

- Use the highest frequency possible: 5.0 MHz or higher is especially useful for anterior gallbladders.
- Use a high line density to pick up tiny stones or polyps (reduce the sector angle and the frame rate if possible). Make sure the focal

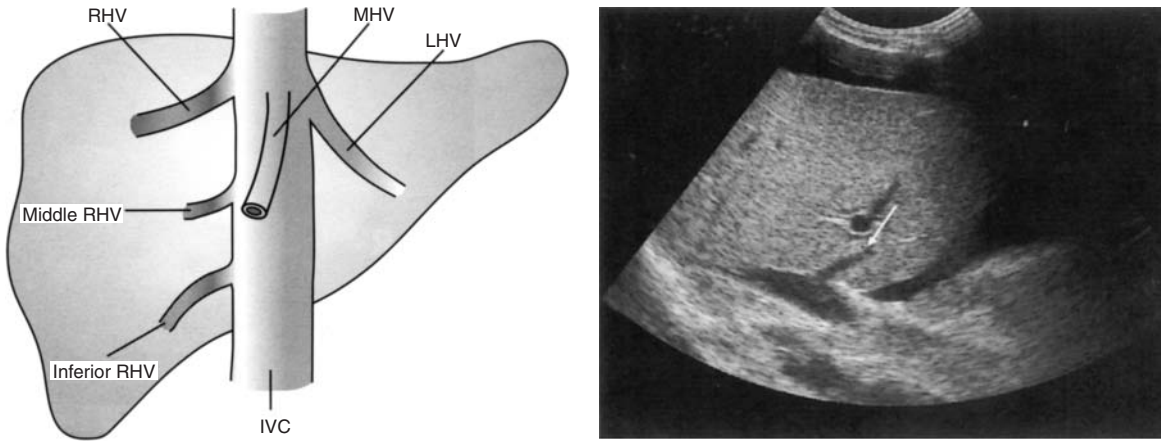


Figure 2.23 (A) Configuration of the hepatic venous system. (B) Inferior middle hepatic vein (arrow) arising from the IVC.

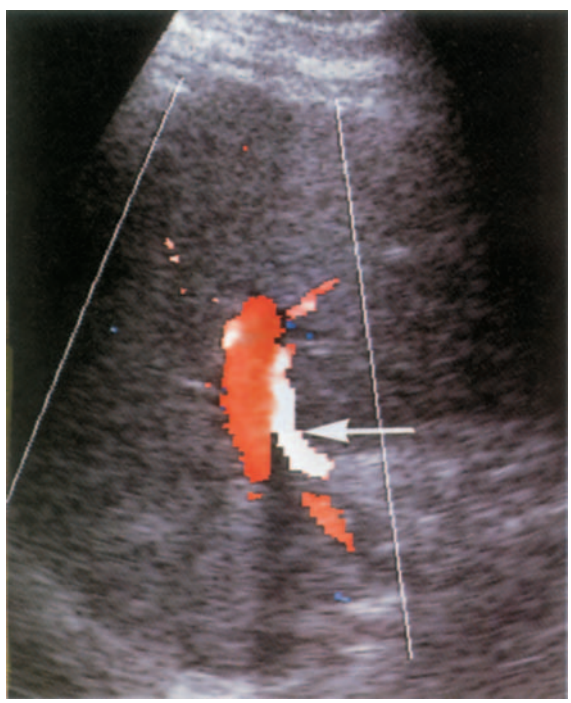


Figure 2.24 Main portal vein at the porta hepatis demonstrating hepatopetal flow. The higher velocity hepatic artery lies adjacent to the Main portal vein (arrow).

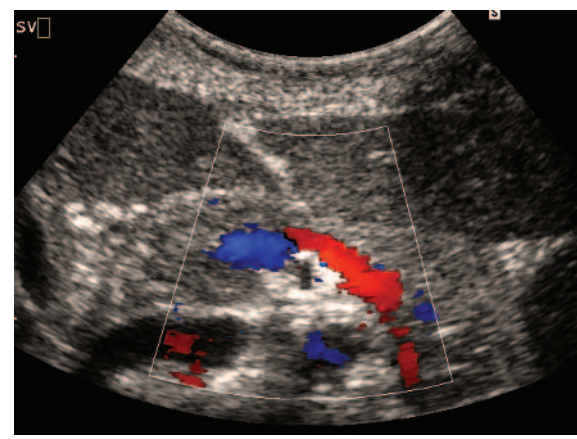


Figure 2.25 TS through the epigastrium, demonstrating the normal splenic vein with flow towards the liver. Note the change from red to blue as the vessel curves away from the transducer.

zone is set over the back wall of the gallbladder to maximize the chances of identifying small stones (see Chapters 1 and 3).

- Alter the time gain compensation (TGC) to eliminate or minimize anterior artefacts and reverberation echoes inside the gallbladder, particularly in the near field.
- Use tissue harmonic imaging to reduce artifact within the gallbladder and sharpen the image of the wall (particularly in a large abdomen).
- Always scan the gallbladder in at least two planes (find the gallbladder's long axis, incorporating the neck and fundus; sweep from side to side, then transversely from neck to fundus) and two patient positions. You will almost certainly miss pathology if you do not.

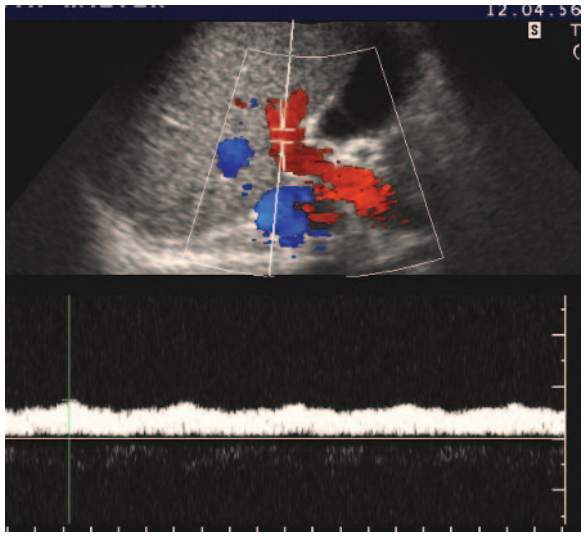


Figure 2.26 Normal portal vein waveform. Respiratory modulations are evident.

- The gallbladder may be 'folded' (the so-called Phrygian cap). To interrogate its contents fully, unfold it by turning the patient decubitus (right side raised), almost prone or erect (Fig. 2.30).
- Bowel gas over the fundus can also be moved by various patient positions.

Normal variants of the gallbladder

The mesenteric attachment of the gallbladder to the inferior surface of the liver is variable in length. This gives rise to large variations in position; at one end of the spectrum the gallbladder, attached only at the neck, may be fairly remote from the liver, even lying in the pelvis; at the other the gallbladder fossa deeply invaginates the liver and the gallbladder appears to lie 'intrahepatically' enclosed on all sides by liver tissue.

The presence of a true septum in the gallbladder is rare. A folded gallbladder frequently gives the impression of a septum but this can be distinguished by positioning the patient to unfold the gallbladder.

Occasionally a gallbladder septum completely divides the lumen into two parts. True gallbladder duplication is a rare entity (Fig. 2.31) and it is important not to mistake this for a gallbladder with a pericholecystic collection in a symptomatic patient. Occasionally the gallbladder is absent altogether.

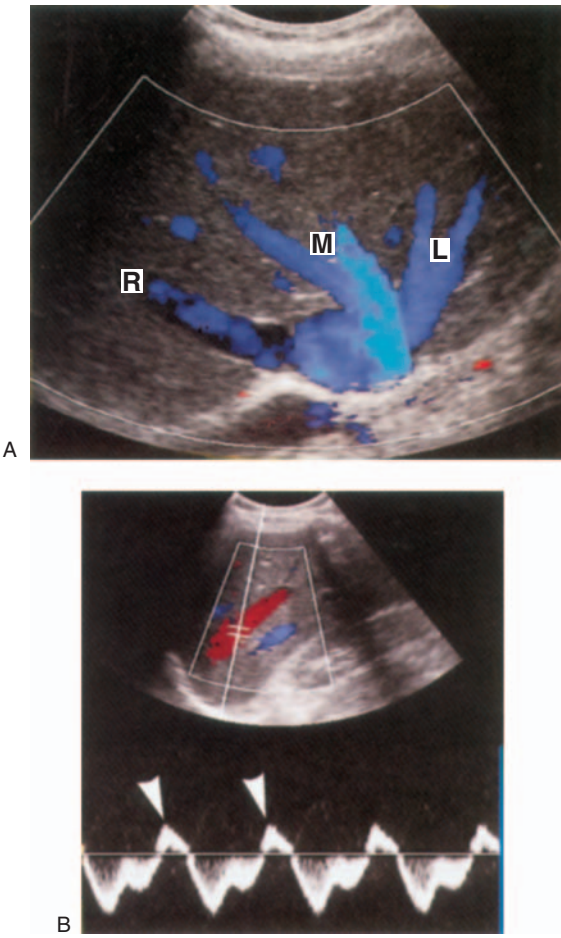
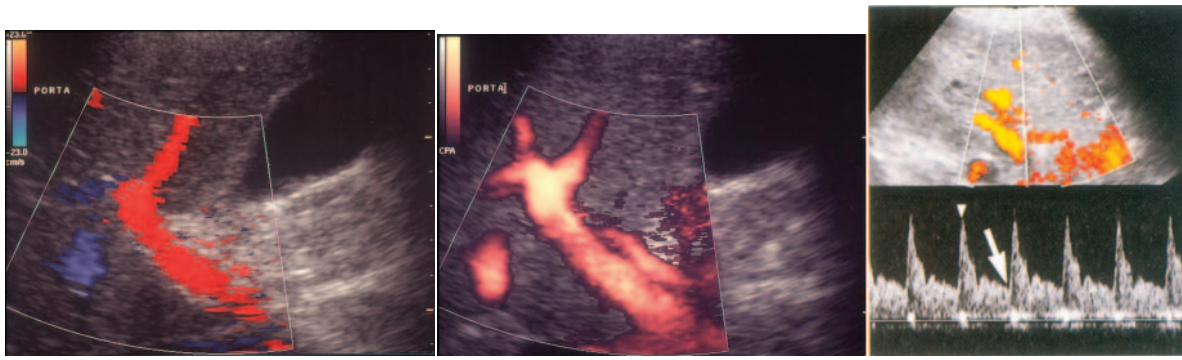


Figure 2.27 (A) The confluence of the right, middle and left hepatic veins with the IVC. (B) Normal hepatic venous waveform. The reverse flow in the vein (arrows) is due to atrial systole. Note that the image has also been frozen during atrial systole, as the hepatic vein appears red.

Pitfalls in scanning the gallbladder

If the gallbladder cannot be found

- Check for previous surgery; a cholecystectomy scar is usually obvious, but evidence of laparoscopic surgery may be difficult to see in the darkened scanning room.
- Check the patient has fasted.



A B C

Figure 2.28 (A) The hepatic artery may be difficult to locate with colour Doppler in some subjects. (B) The same patient using power Doppler; visualization is improved. (C) The normal hepatic artery waveform demonstrates a relatively high-velocity systolic peak (arrowhead) with good forward end-diastolic flow (arrow).

- Look for an ectopic gallbladder, for example positioned low in the pelvis.
- Check that a near-field artefact has not obscured an anterior gallbladder, a particular problem in very thin patients.
- Ensure the scanner frequency and settings are optimized, find the porta hepatis and scan just below it in transverse section. This is the area of the gallbladder fossa and you should see at least the anterior gallbladder wall if the gallbladder is present (Fig. 2.32).
- A contracted, stone-filled gallbladder, producing heavy shadowing, can be difficult to identify due to the lack of any contrasting fluid in the lumen.

Duodenum mimicking gallbladder pathology

- The close proximity of the duodenum to the posterior gallbladder wall often causes it to invaginate the gallbladder. Maximize your machine settings to visualize the posterior gallbladder wall separate from the duodenum and turn the patient to cause the duodenal contents to move.
- Other segments of fluid-containing gastrointestinal tract can also cause confusion (Fig. 2.33).

Stones that don't shadow

- Ensure they are stones and not polyps by standing the patient erect and watching them move with gravity. (Beware—polyps on long stalks also move around.)
- The stones may be smaller than the beam width, making the shadow difficult to display. Make sure the focal zone is set at the back of the gallbladder.
- Increase the line density, if possible, by reducing the field of view.
- Scan with the highest possible frequency to ensure the narrowest beam width.
- Reduce the TGC and/or power to make sure you have not saturated the echoes distal to the gallbladder (see Chapter 3).

Beware the folded gallbladder

- You may miss pathology if the gallbladder is folded and the fundus lies underneath bowel. Always try to unfold it by positioning the patient (Fig. 2.30).
- A fold in the gallbladder may mimic a septum. Septa are comparatively rare and have been over-reported in the past due to the presence of folding.

Pathology or artefact?

Sometimes the gallbladder may contain some echoes of doubtful significance, or be insufficiently distended to evaluate accurately. A rescan, after a meal followed by further fasting, can be useful.

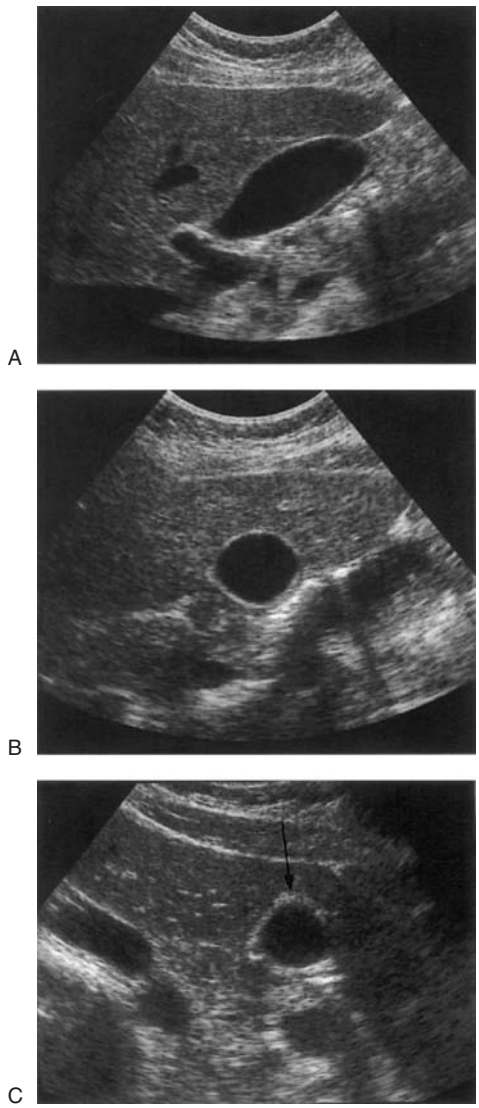


Figure 2.29 The gallbladder: (A) LS, (B) TS. (C) False appearance of wall thickening is produced (arrow) when the angle of scan is not perpendicular to the gallbladder wall in TS.

This can flush out sludge, redistending the gallbladder with clear bile. It may also help to clarify any confusing appearances of adjacent bowel loops.

BILE DUCTS

The common duct can be easily demonstrated in its intrahepatic portion just anterior and slightly to the right of the portal vein. A cross-section of the main hepatic artery can usually be seen passing between

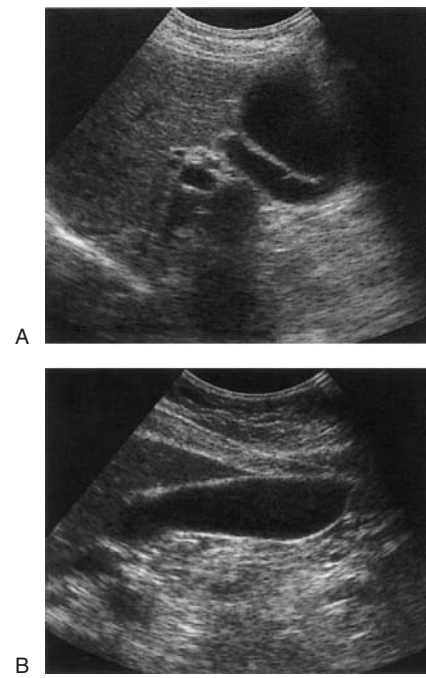


Figure 2.30 (A) A folded gallbladder is difficult to examine with the patient supine. (B) Turning the patient decubitus, right side raised, unfolds the gallbladder, enabling the lumen to be satisfactorily examined.

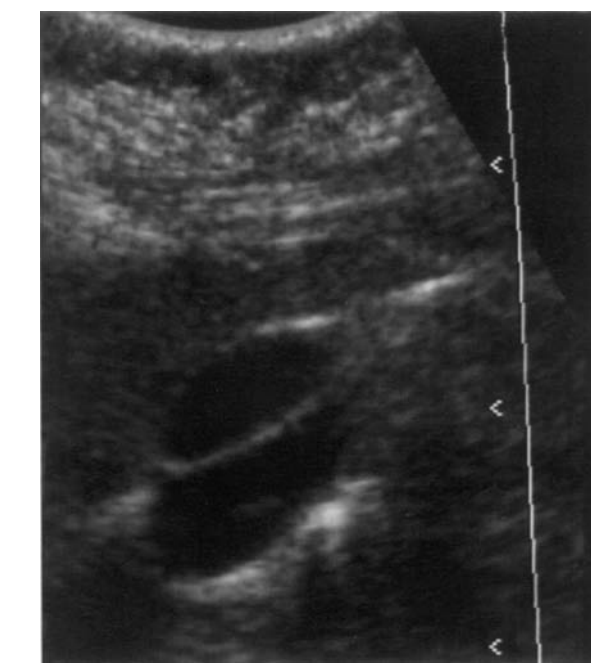


Figure 2.31 Double gallbladder—an incidental finding in a young woman.

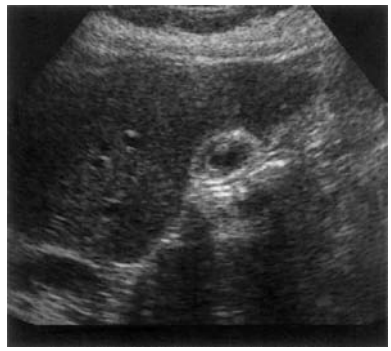


Figure 2.32 A contracted, thick-walled gallbladder located in the gallbladder fossa on TS.

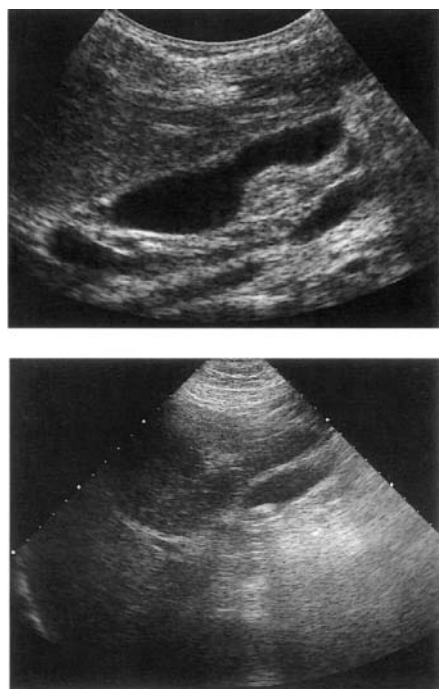


Figure 2.33 (A) The duodenum frequently invaginates the posterior wall of the gallbladder and may mimic pathology if the machine settings are not correctly manipulated. (B) Fluid-filled stomach near the gallbladder fossa mimics a gallbladder containing a stone. The real gallbladder was normal.

the vein and the duct (Figs 2.20A and 2.34), although a small proportion of hepatic arteries lie anterior to the duct (Fig. 2.20B). At this point it is usually referred to as the common duct, although it may, in fact, represent the right hepatic duct[7] rather than

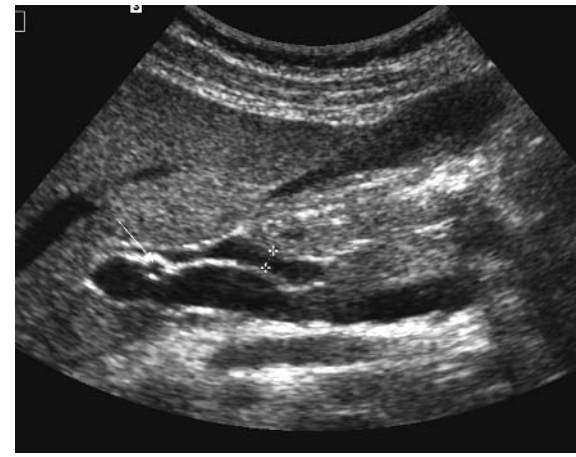


Figure 2.34 CBD at the porta hepatis. The lower end is frequently obscured by shadowing from the duodenum. The duct should be measured at its widest portion.

the common *bile* duct, because we can't tell at what point it is joined by the cystic duct.

The extrahepatic portion of the duct is less easy to see as it is often obscured by overlying duodenal gas. Good visualization of the duct usually requires perseverance on the part of the operator. It is insufficient just to visualize the intrahepatic portion of the duct, as early obstruction may be present with a normal-calibre intrahepatic duct and dilatation of the distal end. (Fig. 2.35).

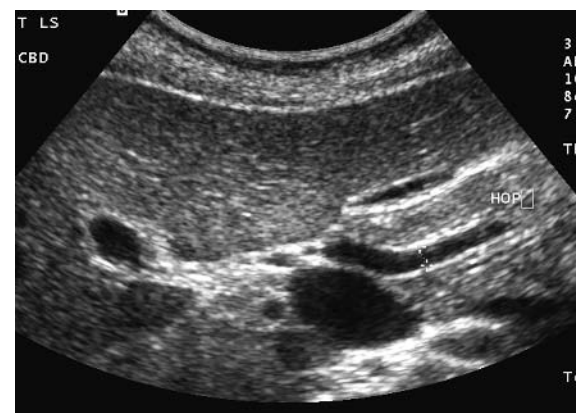


Figure 2.35 Visualization of the lower end of the duct often requires the operator to persevere with technique and patient positioning. The normal duct (calipers) is seen in the head of the pancreas.

Bile duct measurements

The internal diameter of the common duct is usually taken as 6 mm or less. It is age-dependent, however, and can be 8 or 9 mm in an elderly person, due to degeneration of the elastic fibre in the duct wall. Ensure this is not early obstruction by thoroughly examining the distal common bile duct or rescanning after a short time interval. The diameter can vary quite considerably, not only between subjects, but along an individual duct. The greatest measurement should be recorded, in longitudinal section. Never measure the duct in a transverse section (for example at the head of pancreas); it is invariably an oblique plane through the duct, which will overestimate the diameter. Intrahepatically, the duct diameter decreases. The right and left hepatic ducts are just visible, but more peripheral branches are usually too small to see.

Patients with a cholecystectomy who have had previous duct dilatation frequently also have a persistently dilated, but non-obstructed, duct (Fig. 2.36). Be suspicious of a diameter of 10 mm or more as this is associated with obstruction due to formation of stones in the duct.

Techniques

The main, right and left hepatic ducts tend to lie anterior to the portal vein branches; however as the biliary tree spreads out, the position of the duct relative to the portal branches is highly variable. Don't assume that a channel anterior to the PV branch is always a biliary duct—if in doubt, use colour Doppler to distinguish the bile duct from the portal vein or hepatic artery.

The proximal bile duct is best seen either with the patient supine, using an intercostal approach from the right, or turning the patient oblique, right side raised. This projects the duct over the portal vein, which is used as an anatomic marker.

Scanning the distal duct usually requires more effort. Right oblique or decubitus positions are useful. Gentle pressure to ease the duodenal gas away from the duct can also be successful. Sometimes, filling the stomach with water (which also helps to display the pancreas) and allowing it to trickle through the duodenum does the trick. Try also identifying the duct in the pancreatic head (Fig. 2.37) and then tracing it retrogradely towards the liver. Asking the patient to take deep breaths is occasionally successful, but may make matters worse by filling the stomach with air. It is definitely worth persevering with your technique, particularly in jaundiced patients.

SOME COMMON REFERRAL PATTERNS FOR HEPATOBILIARY ULTRASOUND

There is an almost infinite number of reasons for performing abdominal ultrasound. Some of the more common referrals are discussed below.

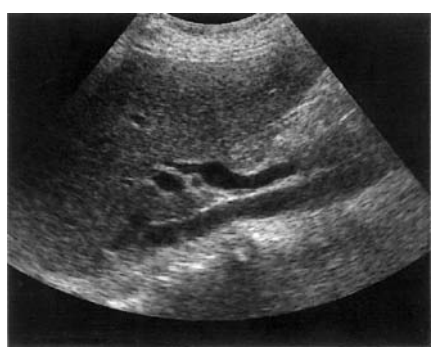


Figure 2.36 A persistently, mildly dilated duct postcholecystectomy (8.5 mm).

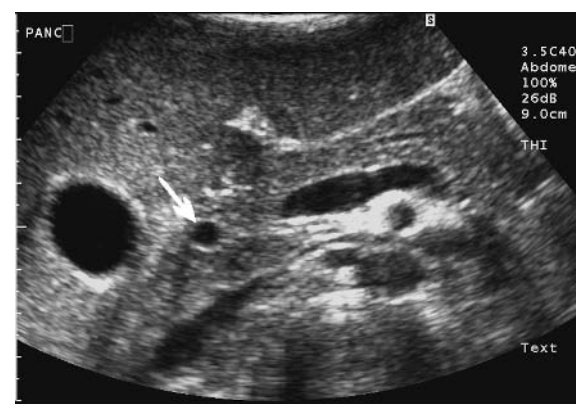


Figure 2.37 The common bile duct (arrow) seen on the head of pancreas on transverse section.

Jaundice

This symptom is a frequent cause of referral for abdominal ultrasound. It is therefore essential for the sonographer to have a basic understanding of the various mechanisms in order to maximize the diagnostic information from the ultrasound scan. The causes and ultrasound appearances of jaundice are dealt with more fully in Chapters 3 and 4; a brief overview is included here.

Jaundice, or *hyperbilirubinaemia*, is an elevated level of bilirubin in the blood. It is recognized by a characteristic yellow coloration of the skin and sclera of the eye, often accompanied by itching if prolonged.

Bilirubin is derived from the haem portion of haemoglobin. Red blood cells are broken down in the liver into haem and globin, releasing their bilirubin, which is non-soluble. This is termed *unconjugated bilirubin*. This is then taken up by the liver cells and converted to a water-soluble form, *conjugated bilirubin*, which is excreted via the biliary ducts into the duodenum to aid fat digestion.

By knowing which of these two types of bilirubin is present in the jaundiced patient, the clinician can narrow down the diagnostic possibilities. Ultrasound then further refines the diagnosis (Fig. 2.38).

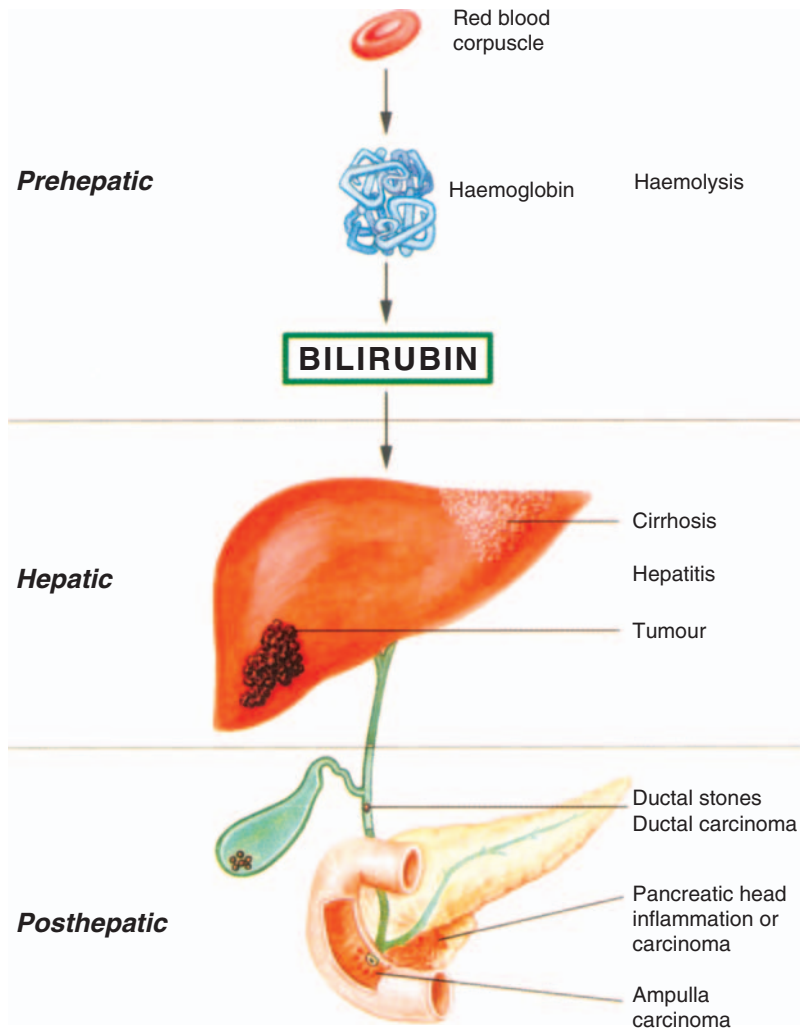


Figure 2.38 Some common causes of jaundice.

Jaundice can fall into one of two categories:

- *obstructive* (sometimes called posthepatic) in which the bile is prevented from draining out of the liver because of obstruction to the biliary duct(s)
- *non-obstructive* (prehepatic or hepatic) in which the elevated bilirubin level is due to haemolysis (the breakdown of the red blood cells) or a disturbance in the mechanism of the liver for uptake and storage of bilirubin, such as in inflammatory or metabolic liver diseases.

Naturally, the treatment of jaundice depends on its cause (Table 2.1). Ultrasound readily distinguishes obstructive jaundice, which demonstrates some degree of biliary duct dilatation, from non-obstructive, which does not.

Abnormal liver function tests

Altered or deranged liver function tests (LFTs) are another frequent cause of referral for abdominal ultrasound.

Biochemistry from a simple blood test is often a primary pointer to pathology and is invariably one of the first tests performed as it is quick and easily accessible. Most of these markers are highly unspecific, being associated with many types of diffuse and focal liver pathology. The most frequently encountered LFTs are listed in Table 2.2.

Other common reasons for referral

In some cases, the presenting symptoms may be organ-specific or even pathognomonic, simplifying the task of ultrasound diagnosis. Often, however,

Table 2.2 Common serum liver function tests

Test	Association with increased level
Bilirubin	Obstructive or non-obstructive jaundice. (Differentiation can be made between conjugated and unconjugated bilirubin)
Alkaline phosphatase (ALP) *(liver enzyme)*	Non-obstructive jaundice Metastases Other focal hepatic lesions
Alpha fetoprotein	Hepatocellular carcinoma (HCC)
Prothrombin time	Malignancy Diffuse liver disease (often with portal hypertension)
Gamma glutamyl transferase	Obstructive jaundice Alcoholic liver disease
Alanine amino-transferase (ALT)	Obstructive or non-obstructive jaundice
Aspartate amino-transferase (AST) *(liver enzymes)*	Hepatitis Viral infections Other organ failure (e.g. cardiac)
Protein (serum albumin)	Lack of protein is associated with numerous liver diseases. Low levels are associated with ascites, often due to portal hypertension

Table 2.1 Common causes of jaundice

Non-obstructive	Obstructive
Unconjugated hyperbilirubinaemia —haemolysis —haematoma —Gilbert's disease	*Conjugated hyperbilirubinaemia* —stones in the biliary duct —carcinoma of the duct, head of pancreas or ampulla —acute pancreatitis —other masses which compress the common bile duct (e.g. lymph node mass) —biliary atresia
Mixed hyperbilirubinaemia —hepatitis —alcoholic liver disease —cirrhosis of all types —multiple liver metastases —drug-induced liver disease	

(See Chapters 3 and 4 for further information.)

the symptoms are vague and non-specific, requiring the sonographer to perform a comprehensive and knowledgeable search. The non-invasive nature of ultrasound makes it ideal for the first-line investigation.

Upper abdominal pain

- Upper abdominal pain, the origin of which could be linked to any of the organs, is one of the most frequent causes of referral. The sonographer can narrow the possibilities down by taking a careful history (see Box 2.1).
- Is the pain focal? This may direct the sonographer to the relevant organ, for example a thick-walled gallbladder full of stones may be tender on gentle transducer pressure, pointing to acute or chronic cholecystitis, depending on the severity of the pain.
- Bear in mind that gallstones are a common incidental finding which may be a red herring. Always consider multiple pathologies.
- Is the pain related to any event which may give a clue? Fat intolerance might suggest a biliary cause, pain on micturition a urinary tract cause, for example.
- Is it accompanied by other symptoms such as a high temperature? This may be associated with an infective process such as an abscess.
- Could it be bowel-related? Generalized abdominal pain could be due to inflammatory or obstructive bowel conditions and knowledge of the patient's bowel habits is helpful.
- Has the patient had any previous surgery which could be significant?

Box 2.1

Always:
- take a verbal history from the patient—don't just rely on the request card
- obtain the results of any previous investigations, including previous radiology
- consider the possibility of multiple pathologies

Palpable right upper quadrant mass

A palpable right upper quadrant mass could be due to a renal, hepatobiliary, bowel-related or other cause. The sonographer should gently palpate to get an idea of the size and position of the mass and whether or not it is tender. Specifically targeting the relevant area may yield useful and unexpected results, for example a Reidel's lobe, colonic carcinoma or impacted faeces, which will help to guide the nature of further investigations.

APPENDIX: UPPER-ABDOMINAL ANATOMY

Diagrams of sectional upper-abdominal anatomy are reproduced here for quick reference. See Box 2.2 for the abbreviations used here.

Box 2.2 Abbreviations

AO	Aorta
CBD	Common bile duct
GB	Gallbladder
GDA	Gastroduodenal artery
HA	Hepatic artery
HOP	Head of pancreas
IVC	Inferior vena cava
LHV	Left hepatic vein
LL	Left lobe of liver
LPV	Left portal vein
LRV	Left renal vein
MHV	Middle hepatic vein
R Adr	Right adrenal gland
RHV	Right hepatic vein
RK	Right kidney
RL	Right lobe of liver
RPV	Right portal vein
RRA	Right renal artery
SA	Splenic artery
SMA	Superior mesenteric artery
SMV	Superior mesenteric vein
SPL	Spleen
ST	Stomach
SV	Splenic vein
TOP	Tail of pancreas

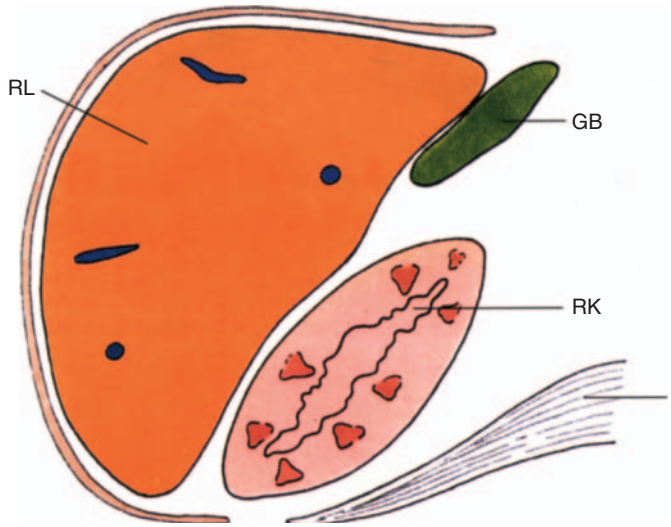


Figure 2A.1 LS through the right lobe of the liver.

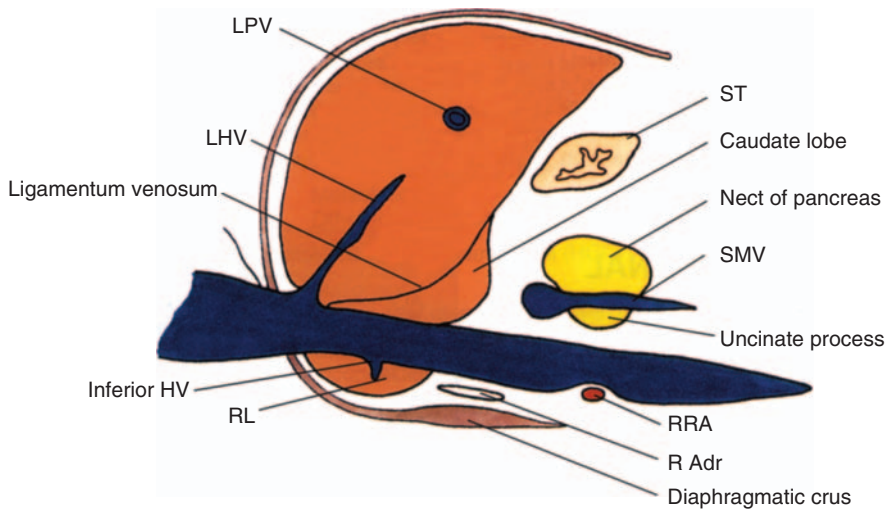


Figure 2A.2 LS through the IVC.

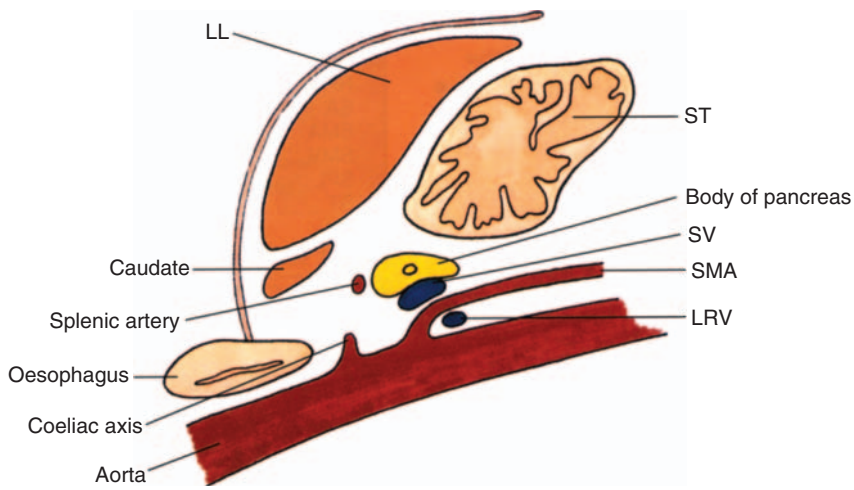


Figure 2A.3 LS through the midline, level of the aorta.

Figure 2A.4 Longitudinal oblique section through the CBD.

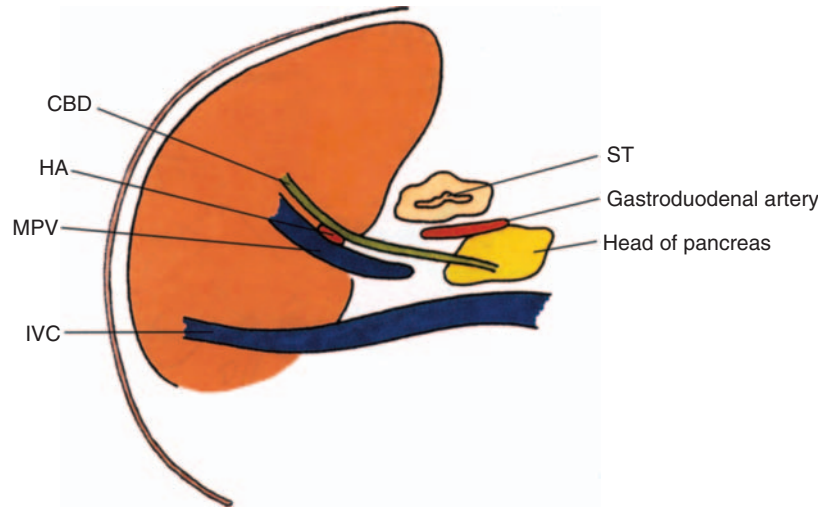
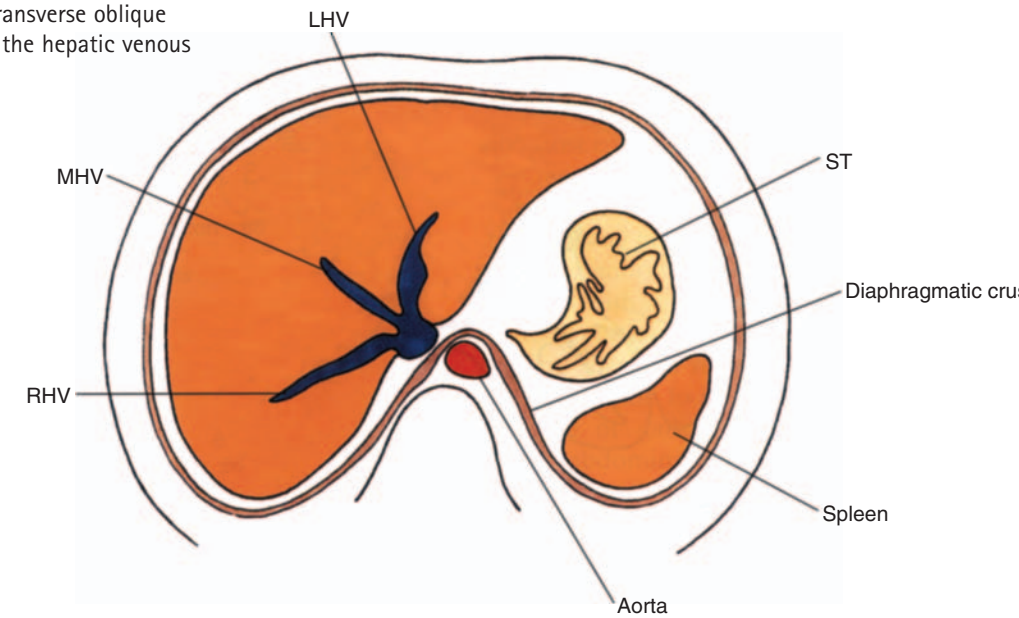


Figure 2A.5 Transverse oblique section through the hepatic venous confluence.



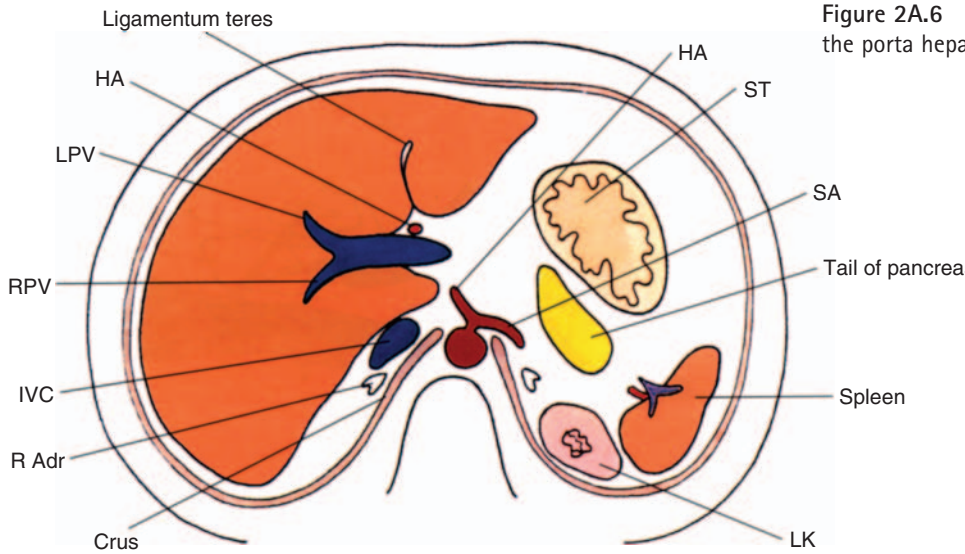


Figure 2A.6 TS through the level of the porta hepatis.

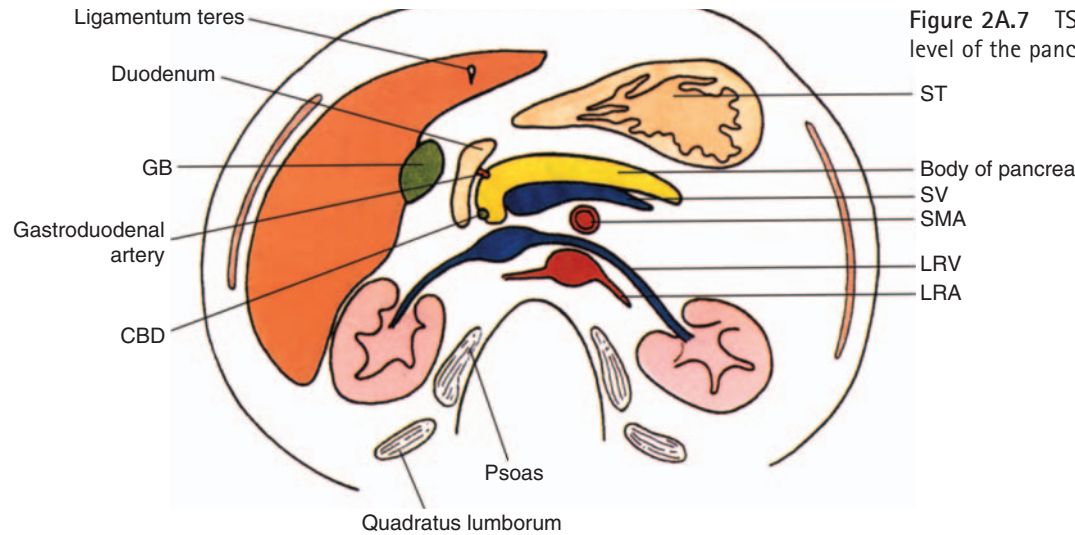


Figure 2A.7 TS at the level of the pancreas.

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Chapter 3

Pathology of the gallbladder and biliary tree

CHAPTER CONTENTS

Ultrasound is an essential first-line investigation in suspected gallbladder and biliary duct disease. It is highly sensitive, accurate and comparatively cheap and is the imaging modality of choice.[1] Gallbladder pathology is common and is asymptomatic in over 13% of the population.[2]

CHOLELITHIASIS

The most commonly and reliably identified gallbladder pathology is that of gallstones (see Table 3.1). More than 10% of the population of the UK have gallstones. Many of these are asymptomatic, which is an important point to remember. When

Table 3.1 Gallstones—clinical features

Often asymptomatic
Biliary colic—RUQ pain, fatty intolerance
+ve ultrasound Murphy's sign (if inflammation is present)
Recurring (RUQ) pain in chronic cholecystitis
Jaundice (depending on degree of obstruction)
Fluctuating fever (if infection is present)

RUQ=right upper quadrant.

scanning a patient with abdominal pain it should not automatically be assumed that, when gallstones are present, they are responsible for the pain. It is not uncommon to find further pathology in the presence of gallstones and a comprehensive upper-abdominal survey should always be carried out.

Gallstones are associated with a number of conditions. They occur when the normal ratio of components making up the bile is altered, most commonly when there is increased secretion of cholesterol in the bile. Conditions which are associated with increased cholesterol secretion, and therefore the formation of cholesterol stones, include obesity, diabetes, pregnancy and oestrogen therapy. The incidence of stones also rises with age, probably because the bile flow slows down.

An increased secretion of bilirubin in the bile, as in patients with cirrhosis for example, is associated with pigment (black or brown) stones.

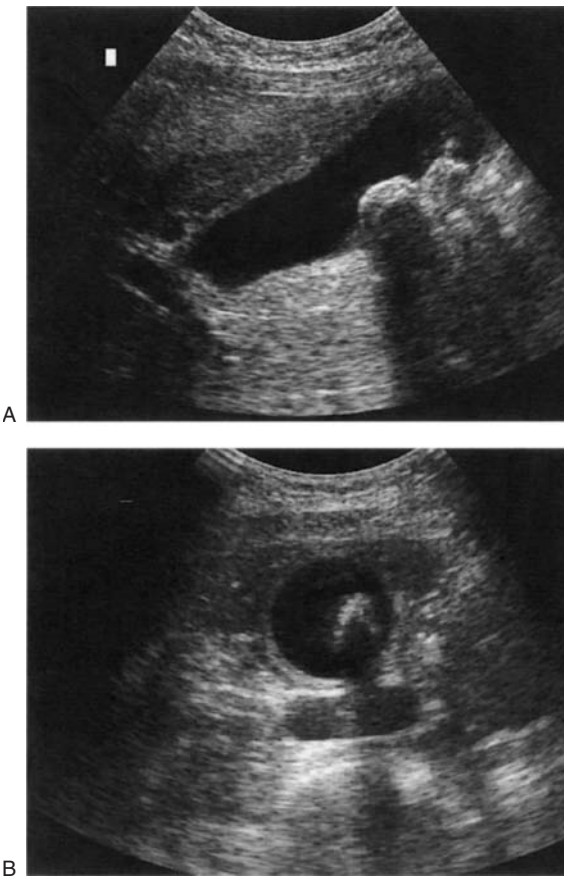


Figure 3.1 (A) Longitudinal section and (B) transverse section images of the gallbladder containing stones with strong distal acoustic shadowing. Note the thickened gallbladder wall.

Ultrasound appearances

There are three classic acoustic properties associated with stones in the gallbladder; they are *highly reflective, mobile* and cast a *distal acoustic shadow*. In the majority of cases, all these properties are demonstrated (Figs 3.1–3.3).

Shadowing

The ability to display a shadow posterior to a stone depends upon several factors:

- The reflection and absorption of sound by the stone. This is fairly consistent, regardless of the composition of the stone.
- The size of the stone in relation to the beam width. A shadow will occur when the stone

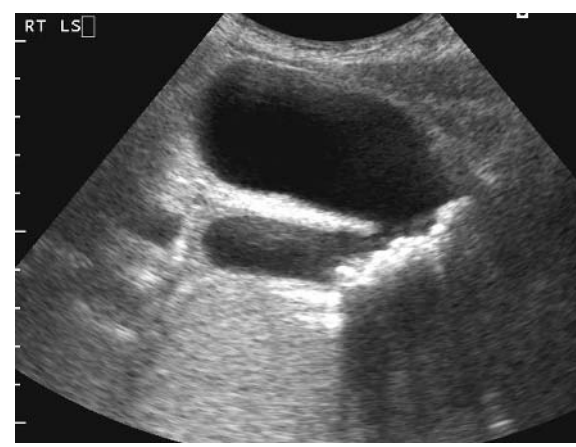


Figure 3.2 Multiple tiny stones combining to form a posterior band of shadow.

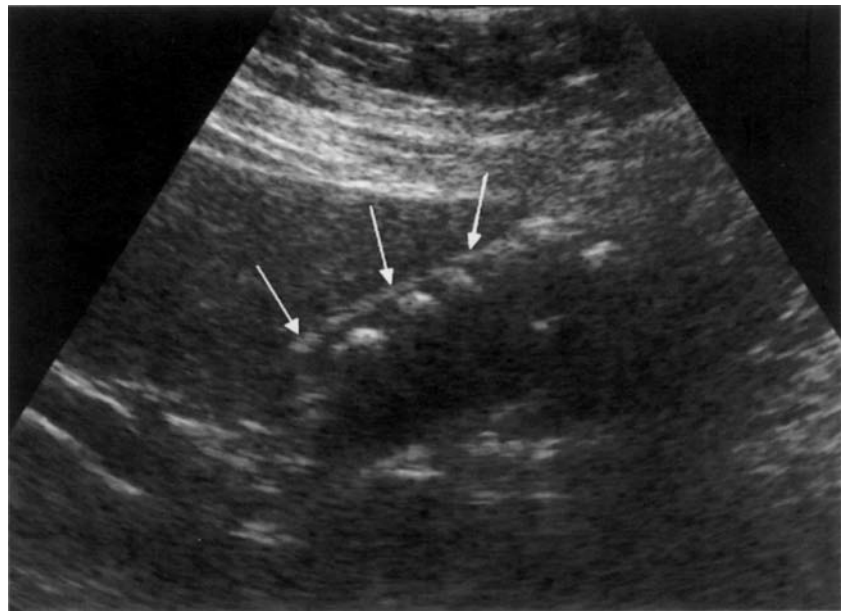


Figure 3.3 Floating stones just below the anterior gallbladder wall.

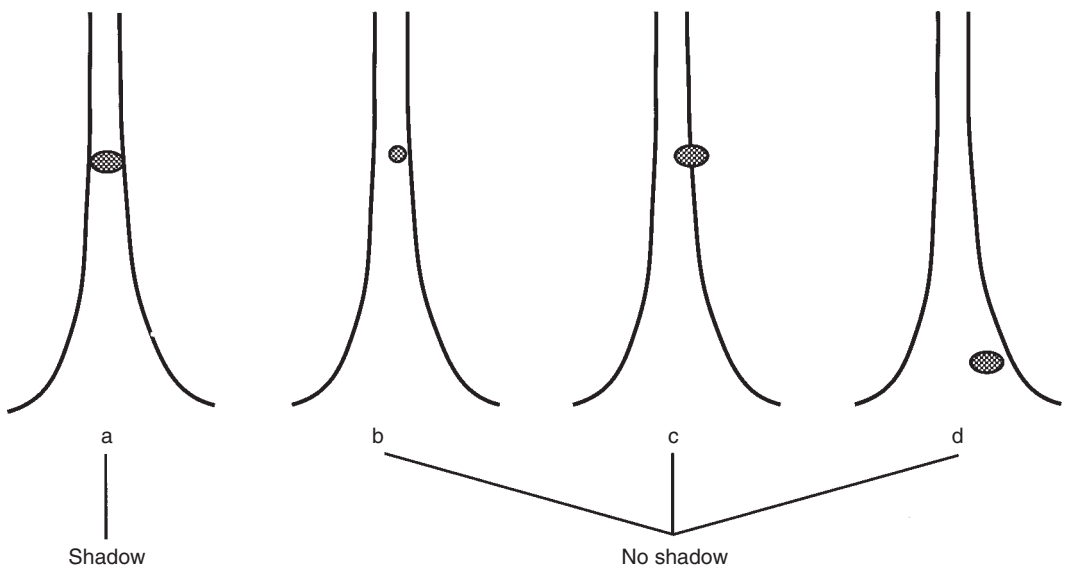


Figure 3.4 (a) A shadow will be displayed from the stone, which occupies the width of the beam. (b) The stone is smaller than the beam. (c) The stone is large, but just out of the beam. (d) The stone is large, but outside the focal zone, where the beam is wider.

fills the width of the beam (Fig. 3.4). This will happen easily with large stones, but a small stone may occupy less space than the beam, allowing sound to continue behind it, so a shadow is not seen. Small stones must therefore be within the focal zone (narrowest point) of the beam and in the centre of the beam to shadow (Fig. 3.5). Higher-frequency transducers have better resolution and are therefore more likely to display fine shadows than lower frequencies.

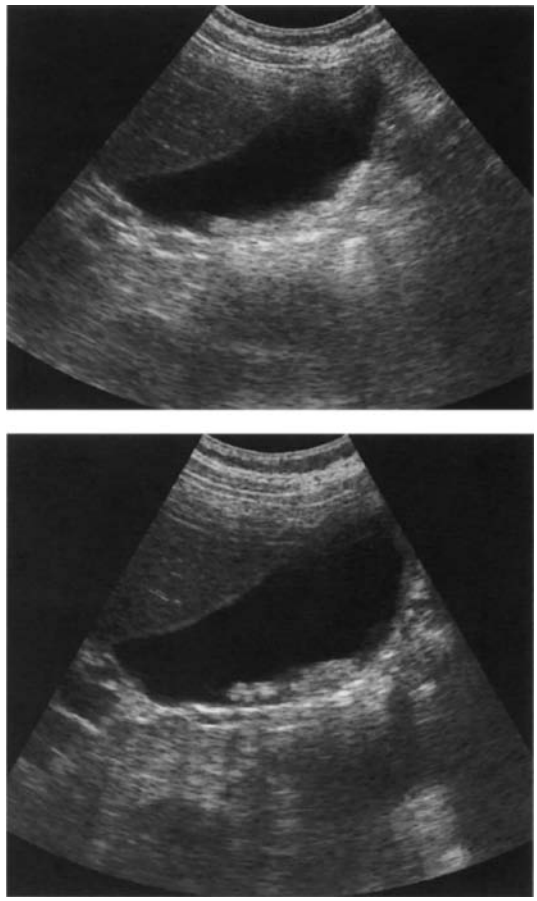


Figure 3.5 (A) The stones are outside the focal zone, and do not appear to shadow well. (B) The focal zone has been moved to the level of the stones, allowing the shadow to be displayed.

- The machine settings must be compatible with demonstrating narrow bands of shadowing. The fluid-filled gallbladder often displays posterior enhancement, or increased through-transmission. If the echoes posterior to the gallbladder are 'saturated' this will mask fine shadows. Turn the overall gain down to display this better (Fig. 3.6). Some image-processing options may reduce the contrast between the shadow and the surrounding tissue, so make sure a suitable dynamic range and image programme are used.
- Bowel posterior to the gallbladder may cast its own shadows from gas and other contents, which makes the gallstone shadow difficult to demonstrate (Fig. 3.7B). This is a particular problem with stones in the common bile duct (CBD). Try turning the patient to move the gallbladder away from the bowel. The shadow cast by gas in the duodenum, which contains reverberation, should usually be distinguishable from that cast by a gallstone, which is sharp and clean.

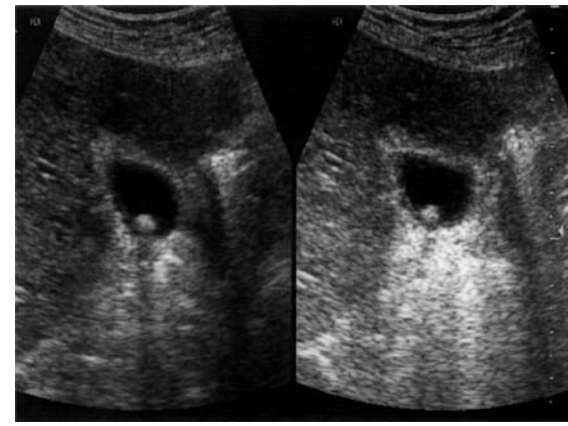


Figure 3.6 The shadow behind the gallstone (left image) is obscured if the time gain compensation is set too high behind the gallbladder (right image).

Reflectivity

The reflective nature of the stone is enhanced by its being surrounded by echo-free bile. In a contracted gallbladder the reflectivity of the stone is often not appreciated because the hyperechoic gallbladder wall is collapsed over it.

Some stones are only poorly reflective, but should still cause a distal acoustic shadow.

Mobility

Most stones are gravity-dependent and this may be demonstrated by scanning the patient in an erect position (Fig. 3.7), when a mobile calculus will drop from the neck or body of the gallbladder to lie in the fundus. Some stones will float, however, forming a reflective layer just beneath the anterior gallbladder wall with shadowing that obscures the rest of the lumen (Fig. 3.3).

When the gallbladder lumen is contracted, either due to physiological or pathological reasons,

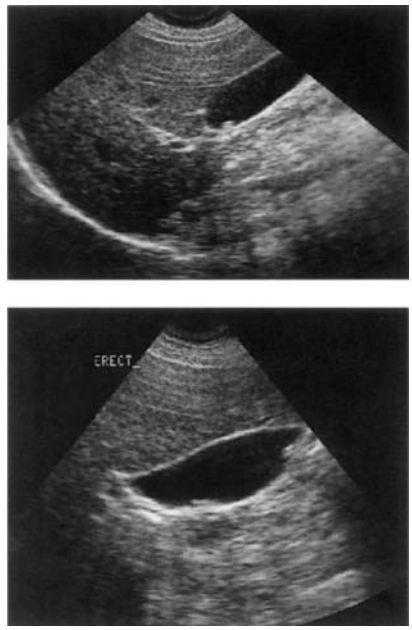


Figure 3.7 (A) Supine and (B) erect views demonstrating movement of the tiny stone into the fundus of the gallbladder. Note how duodenum posterior to the gallbladder masks the shadow in the erect state.

any stones present are unable to move and this is also the case in a gallbladder packed with stones.

Occasionally a stone may become impacted in the neck, and movement of the patient is unable to dislodge it. Stones lodged in the gallbladder neck or cystic duct may result in a permanently contracted gallbladder, a gallbladder full of fine echoes due to inspissated (thickened) bile (Fig. 3.8) or a distended gallbladder due to a mucocoele (see below).

Choledocholithiasis

Stones may pass from the gallbladder into the common duct, or may develop *de novo* within the common duct. Stones in the CBD may obstruct the drainage of bile from the liver, causing obstructive jaundice.

Due to shadowing from the duodenum, ductal stones are often not demonstrated with ultrasound without considerable effort. Usually they are accompanied by stones in the gallbladder and a degree of dilatation of the CBD. In these cases the operator can usually persevere and demonstrate the stone at the lower end of the duct. However, the duct may be dilated but empty, the stone having recently passed.

Stones may be seen to move up and down a dilated duct. This can create a ball-valve effect so that obstruction may be intermittent.

It is not unusual to demonstrate a stone in the CBD without stones in the gallbladder, a phenomenon which is also well-documented following cholecystectomy (Fig. 3.9). This may be due to a single calculus in the gallbladder having moved into the duct, or stone formation within the duct.

It is also important to remember that stones in the CBD may be present *without* duct dilatation and attempts to image the entire common duct

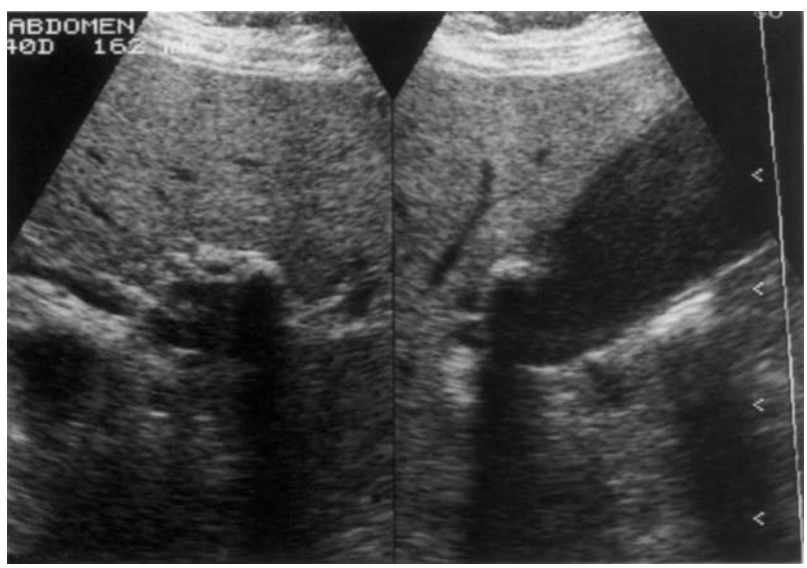
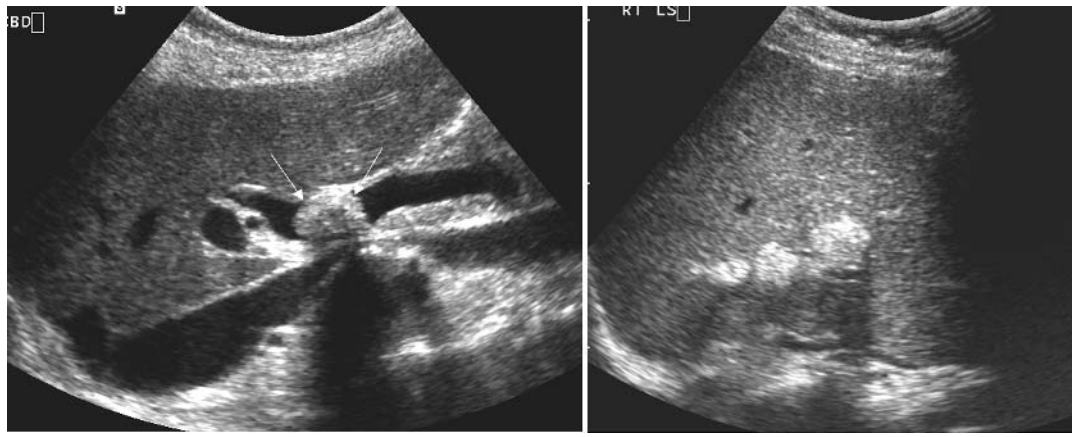
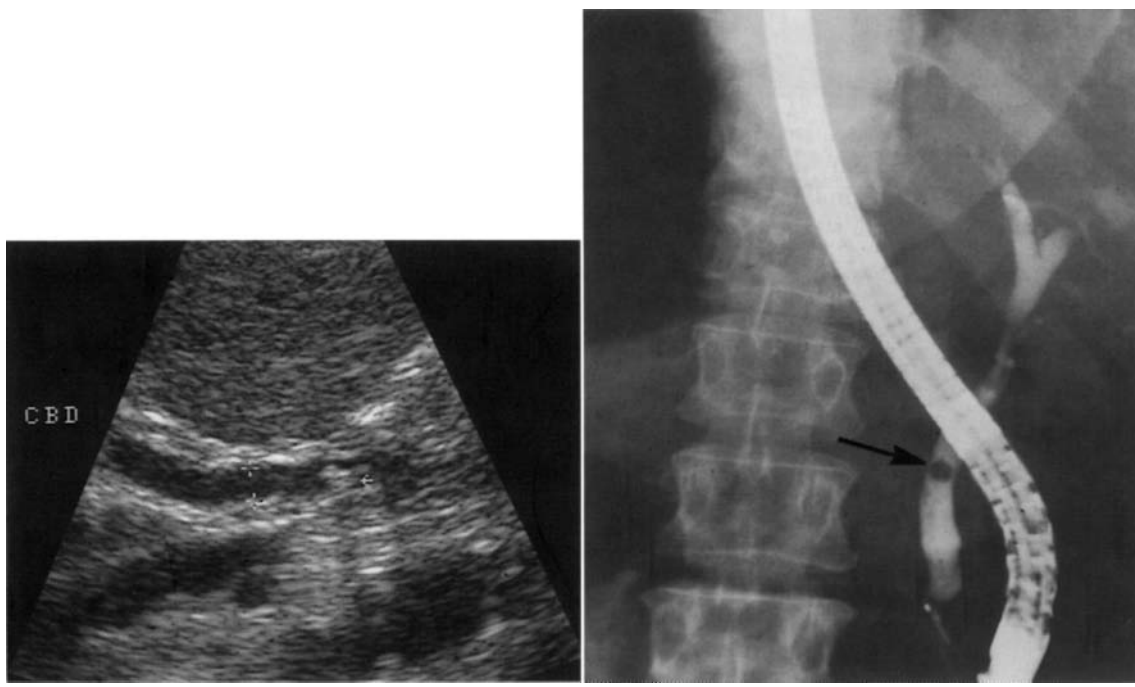


Figure 3.8 Stone impacted in the neck of the gallbladder. The left-hand image is a TS through the neck demonstrating the impacted stone. The right-hand image demonstrates the dilated gallbladder containing fine echoes from inspissated bile.



A B

Figure 3.9 (A) A stone in a dilated common bile duct (CBD) with posterior shadowing. The gallbladder was dilated but did not contain stones. (B) Stone formation in the intrahepatic ducts.



A B

Figure 3.10 (A) Small stone in the CBD causing intermittent obstruction. At the time of scanning, the CBD was normal in calibre at 5 mm. The duct walls are irregular, consistent with cholangitis. (B) Endoscopic cholangiopancreatography (ERCP) of a stone in a normal-calibre (5 mm) duct.

with ultrasound should *always* be made, even if it is of normal calibre at the porta (Fig. 3.10).

Other ultrasound signs to look for are shown in Table 3.2.

Possible complications of gallstones are outlined in Figure 3.11A. In rare cases, stones may perforate the inflamed gallbladder wall to form a fistula into the small intestine or colon. A large stone

Table 3.2 Gallstones—other ultrasound signs to look for

Acute or chronic cholecystitis
Complications of cholecystitis, e.g. pericholecystic collection
Stone impacted in the neck of gallbladder—mucocoele, hydrops
CBD stones
Biliary obstruction—dilatation of the CBD and/or intrahepatic ducts
Pancreatitis
Other causes of RUQ pain unrelated to stones

CBD = common bile duct.

passing into the small intestine may impact in the ileum, causing intestinal obstruction (Fig. 3.11B).

Biliary reflux and gallstone pancreatitis

A stone may become lodged in the distal common bile duct near the ampulla. If the main pancreatic duct joins the CBD proximal to this, bile and pancreatic fluid may reflux up the pancreatic duct, causing inflammation and severe pain.

Reflux up the common bile duct may also result in ascending cholangitis, particularly if the obstruction is prolonged or repetitive. Cholangitis may result in dilated bile ducts with mural irregularity on ultrasound, but endoscopic retrograde cholangiopancreatography (ERCP) is usually superior in demonstrating intrahepatic ductal changes of this nature.

Bile reflux is also associated with anomalous cystic duct insertion (Fig. 3.12), which is more readily recognized on ERCP than ultrasound.

Further management of gallstones

ERCP demonstrates stones in the duct with greater accuracy than ultrasound, particularly at the lower end of the CBD, which may be obscured by duodenal gas and also allows for sphincterotomy and stone removal.

Laparoscopic cholecystectomy is the preferred method of treatment for symptomatic gallbladder disease in an elective setting and has well-recognized benefits over open surgery in experienced

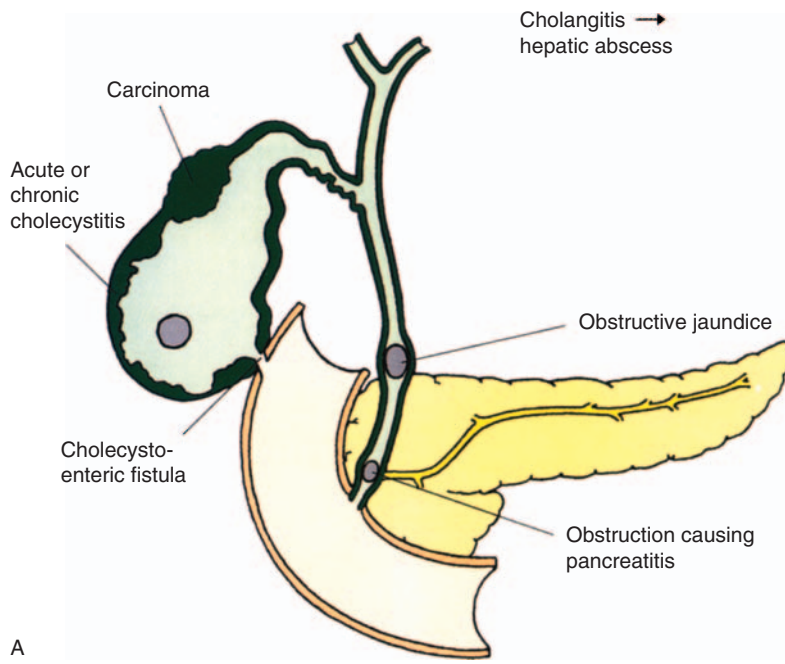
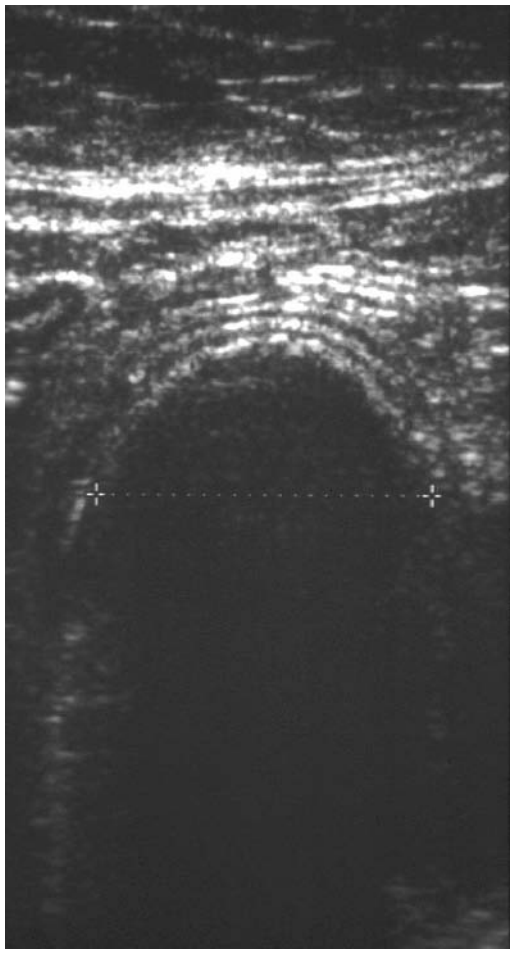


Figure 3.11 (A) The possible complication of gallstones.

(Continued)



B

Figure 3.11 cont'd (B) Gallstone Ileus.

hands. Acute cholecystitis is also increasingly managed by early laparoscopic surgery, with a slightly higher rate of conversion to open surgery than elective cases.[3] Laparoscopic ultrasound may be used as a suitable alternative to operative cholangiography to examine the common duct for residual stones during surgery.[4] Both ultrasound and cholescintigraphy are used in monitoring postoperative biliary leaks or haematoma (Fig. 3.13).

Other, less common options include dissolution therapy and extracorporeal shock wave lithotripsy (ESWL). However, these treatments are often only partially successful, require careful patient selection and also run a significant risk of stone recurrence.[5]

ENLARGEMENT OF THE GALLBLADDER

Because of the enormous variation in size and shape of the normal gallbladder, it is not possible to diagnose pathological enlargement by simply using measurements. Three-dimensional techniques may prove useful in assessing gallbladder volume[6] but this is a technique which is only likely to be clinically useful in a minority of patients with impaired gallbladder emptying.

An enlarged gallbladder is frequently referred to as *hydropic*. It may be due to obstruction of the cystic duct (see below) or associated with numerous disease processes such as diabetes, primary sclerosing cholangitis, leptospirosis or in response to some types of drug.

A pathologically dilated gallbladder, as opposed to one which is physiologically dilated, usually assumes a more rounded, tense appearance.

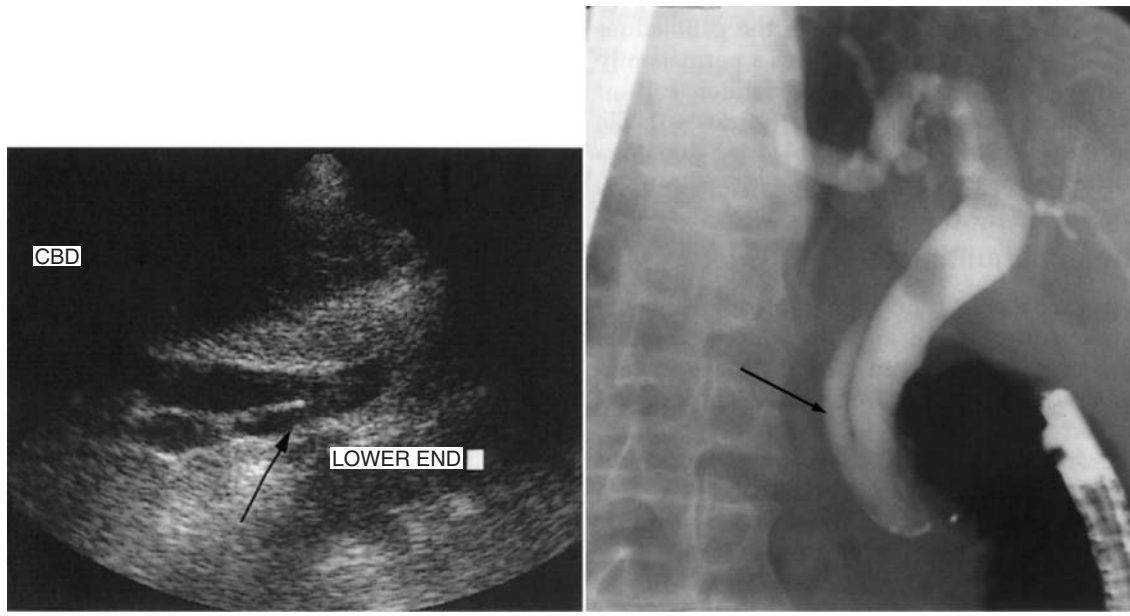
Mucocoele of the gallbladder

If the cystic duct is obstructed, usually by a stone which has failed to pass through to the CBD, the normal flow of bile from the gallbladder is interrupted. Chronic cystic duct obstruction causes the bile to be replaced by mucus secreted by the lining of the gallbladder, resulting in a mucocoele. The biliary ducts remain normal in calibre.

If the gallbladder looks dilated, make a careful search for an obstructing lesion at the neck; a stone in the cystic duct is more difficult to identify on ultrasound as it is not surrounded by echo-free bile (Fig. 3.8).

Mirizzi syndrome

Mirizzi syndrome is a rare cause of biliary obstruction in which the cystic duct is obstructed by a stone, which in combination with a surrounding inflammatory process compresses and obstructs the common hepatic duct, causing distal biliary duct dilatation. This is associated with a low insertion of the cystic duct into the common hepatic duct. Occasionally a fistula forms between the hepatic duct and the gallbladder due to erosion of the duct wall by the stone. Ultimately this may lead to gallstone ileus—small-bowel obstruction resulting from migration of a large stone through

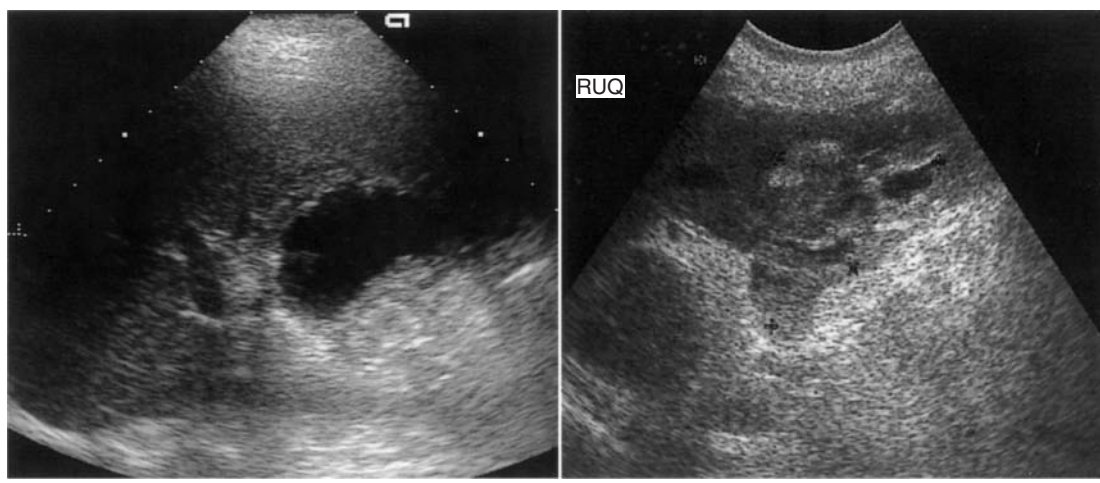


A B

Figure 3.12 (A) Anomalous insertion of the cystic duct (arrow) into the lower end of the CBD. (B) Appearances of case in (A) are confirmed on ERCP. A stone is also present in the duct.

the cholecystoenteric fistula (Fig 3.11B). If the condition is not promptly diagnosed, recurring cholangitis leading to secondary biliary cirrhosis may result.

On ultrasound the gallbladder may be either enlarged or contracted and contain debris. A stone impacted at the neck may be demonstrated together with dilatation of the intrahepatic ducts with a normal-calibre lower common duct (Fig. 3.14). The diagnosis, however, is difficult, and ERCP is generally the most successful modality. Although rare, it is an important diagnosis as cholecystectomy in these cases has a higher rate of operative and post-operative complications.[7]



A B

Figure 3.13 (A) Postoperative bile collection in the gallbladder bed. (B) Hyperechoic, irregular mass in the gallbladder bed which represents a resolving haematoma after laparoscopic cholecystectomy.

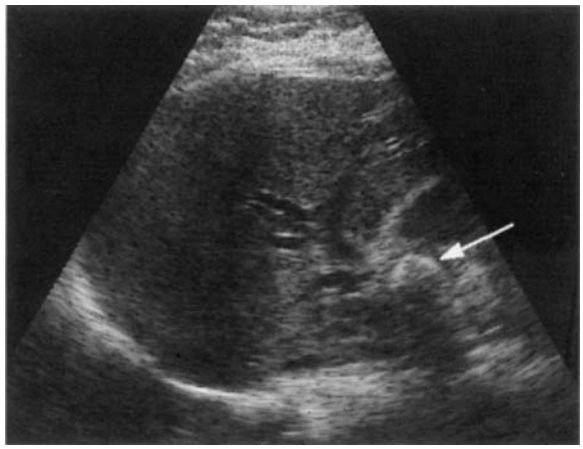


Figure 3.14 Mirizzi syndrome: a large stone in the neck of the gallbladder (arrow) is compressing the bile duct, causing intrahepatic duct dilatation. The lower end of the CBD remains normal in calibre.

THE CONTRACTED OR SMALL GALLBLADDER

Postprandial

The most likely cause is physiological and due to inadequate preparation. The normal gallbladder wall is thickened when contracted, and this must not be confused with a pathological process. Always enquire what the patient has recently eaten or drunk (Fig. 3.15).

Pathological causes of a small gallbladder

Most pathologically contracted gallbladders contain stones.

When the gallbladder cannot be identified, try scanning transversely through the gallbladder fossa, just caudal to the porta hepatis. Strong shadowing alerts the sonographer to the possibility of a contracted gallbladder full of stones. The reflective surface of the stones and distal shadowing are apparent and the anterior gallbladder wall can be demonstrated with correct focusing and good technique (Fig. 3.16).

Do not confuse the appearances of a previous cholecystectomy, when bowel in the gallbladder fossa casts a shadow, with a contracted, stone-filled gallbladder.

A less common cause of a small gallbladder is the *microgallbladder* associated with cystic fibrosis

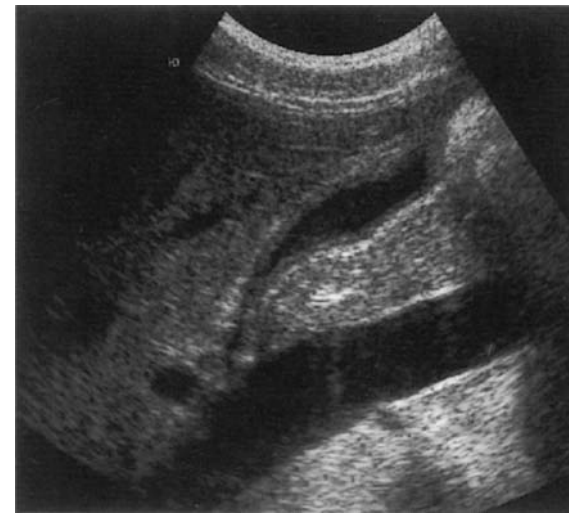


Figure 3.15 Postprandial, contracted gallbladder, with consequently thickened wall.

(Fig. 3.17). The gallbladder itself is abnormally small, rather than just contracted. Cystic fibrosis also carries an increased incidence of gallstones because of the altered composition of the bile and bile stasis and the wall might be thickened and fibrosed from cholecystitis.

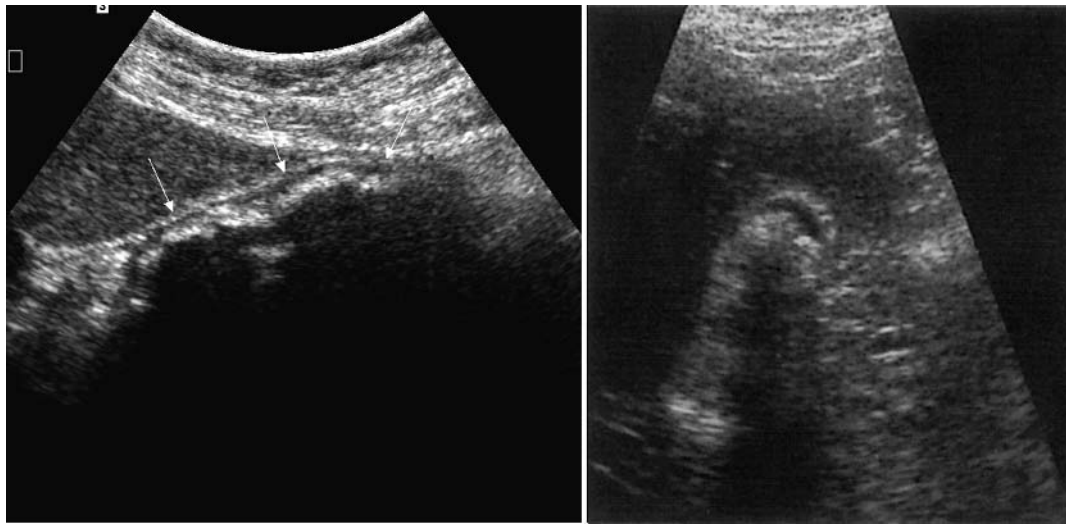
PORCELAIN GALLBLADDER

When the gallbladder wall becomes calcified the resulting appearance is of a solid reflective structure causing a distal shadow in the gallbladder fossa (Fig. 3.18). This can be distinguished from a gallbladder full of stones where the wall can usually be seen anterior to the shadowing (Fig 3.16).

A porcelain gallbladder probably results from a gallbladder mucocoele—a long-standing obstruction of the cystic duct, usually from a stone. The bile inside the non-functioning gallbladder is gradually replaced by watery fluid, the wall becomes fibrotic and thickened and ultimately calcifies.

There is an association between porcelain gallbladder and gallbladder carcinoma, so a prophylactic cholecystectomy is usually performed to pre-empt malignant development.[8]

The shadowing from the anterior gallbladder wall obscures the gallbladder contents, and can mimic bowel in the gallbladder fossa. A plain X-ray also clearly demonstrates the porcelain gallbladder.



A B

Figure 3.16 (A) The gallbladder lumen is filled with stones, causing dense shadowing in the gallbladder fossa. The thickened gallbladder wall can be demonstrated separately (arrows) from the reflective surface of the stones. (B) A small layer of bile is visible between the stones and the anterior gallbladder wall.

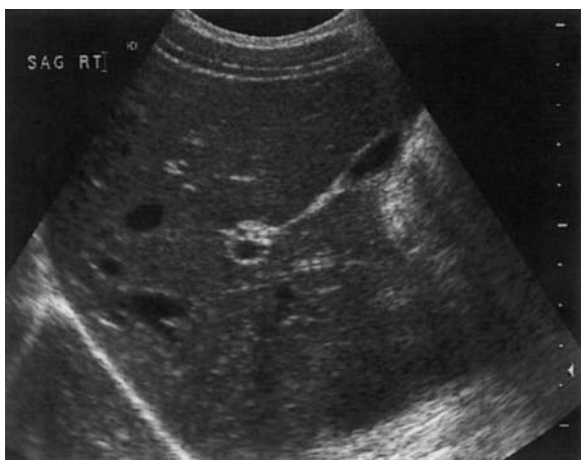


Figure 3.17 Microgallbladder in cystic fibrosis.

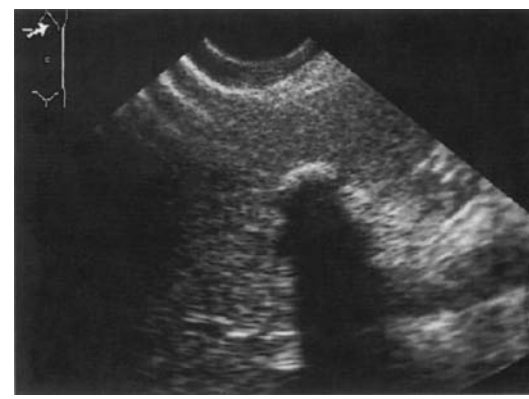


Figure 3.18 TS of a porcelain gallbladder demonstrating a calcified wall with strong acoustic shadowing.

HYPERPLASTIC CONDITIONS OF THE GALLBLADDER WALL

Adenomyomatosis

This is a non-inflammatory, hyperplastic condition which causes gallbladder wall thickening. It may be mistaken for chronic cholecystitis on ultrasound.

The epithelium which lines the gallbladder wall undergoes hyperplastic change, extending diverticula into the adjacent muscular layer of the wall. These diverticula, or sinuses (known as Rokitansky–Aschoff sinuses), are visible within the wall as fluid-filled spaces (Fig. 3.19), which can bulge eccentrically into the lumen, and may contain echogenic material or even (normally pigment) stones.

The wall thickening may be focal or diffuse, and the sinuses may be little more than hypoechoic

'spots' in the thickened wall, or may become quite large cavities in some cases.[9]

Deposits of crystals in the gallbladder wall frequently result in distinctive 'comet-tail' artefacts.

Often asymptomatic, this may present with biliary colic although it is unclear whether this is caused by co-existent stones. Its distinctive appearance allows the diagnosis to be made easily, whether or not stones are present.

Cholecystectomy is performed in symptomatic patients, usually those who also have stones. Although essentially a benign condition, a few cases of associated malignant transformation have been reported, usually in patients with asso-

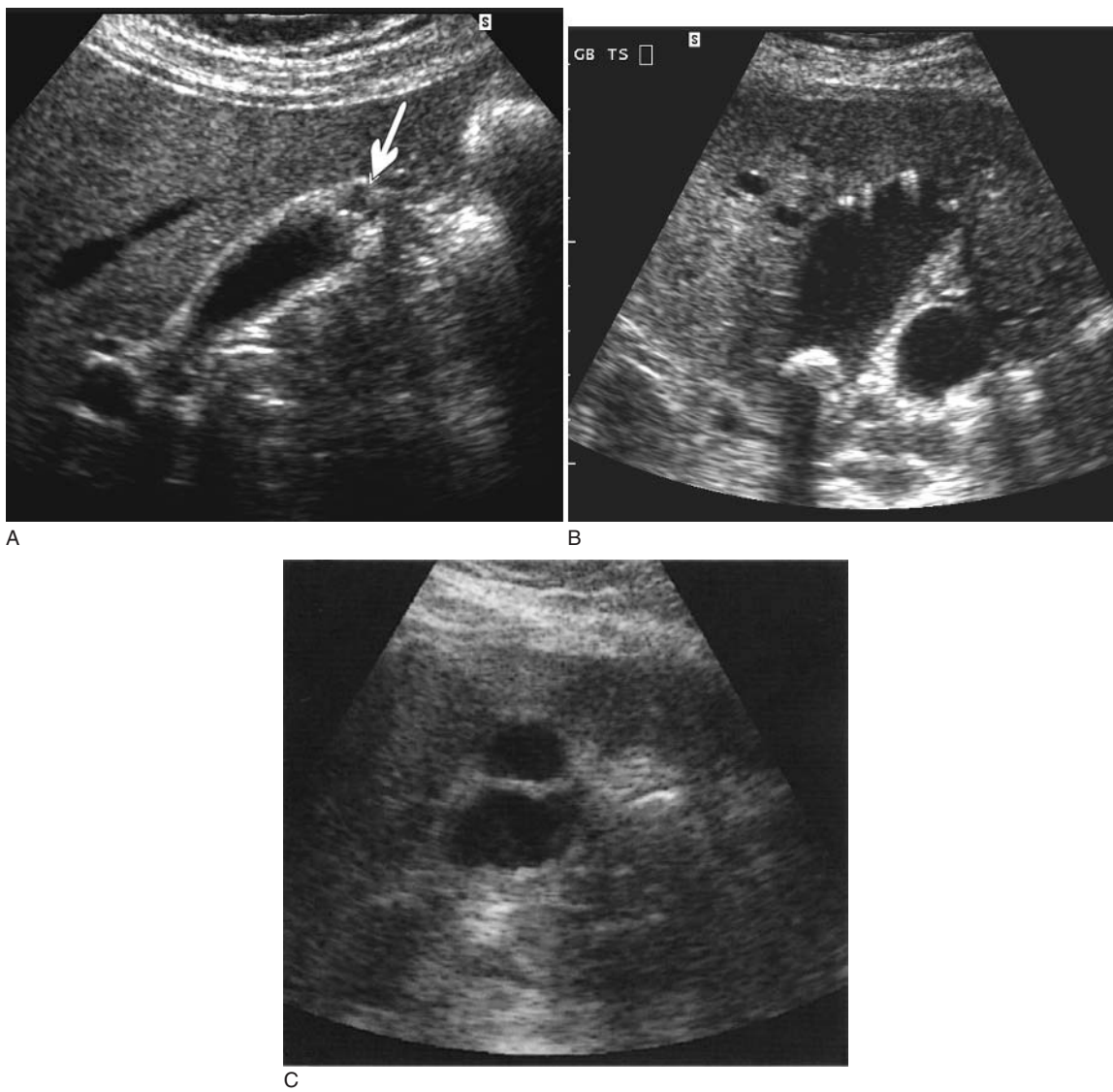


Figure 3.19 Adenomyomatosis: (A) LS demonstrating a thickened gallbladder wall with a small Rokitansky-Aschoff sinus (arrow) at the fundus. (B) TS demonstrating a stone and comet-tail artifacts from within the wall due to crystal deposits. (C) TS through a more advanced case of adenomyomatosis with a large Rokitansky–Aschoff sinus, giving the appearance of a 'double lumen'.

ciated anomalous insertion of the pancreatic duct.[10]

Polyps

Gallbladder polyps are usually asymptomatic lesions which are incidental findings in up to 5% of the population. Occasionally they are the cause of biliary colic. The most common type are cholesterol polyps. These are reflective structures which project into the gallbladder lumen but do not cast an acoustic shadow. Unless on a long stalk they will remain fixed on turning the patient and are therefore distinguishable from stones (Fig. 3.20).

There is an association between larger adenomatous gallbladder polyps and subsequent carcinoma, especially in patients over 50 years of age, so cholecystectomy is often advised (Fig. 3.20C). Smaller polyps of less than 1 cm in diameter may be safely monitored with ultrasound.[11] In particular, gallbladder polyps in patients with primary sclerosing cholangitis have a much greater likelihood of malignancy (40–60%).[12]

Cholesterolosis

Also known as the 'strawberry gallbladder', this gets its name because of the multiple tiny nodules on the surface of the gallbladder mucosal lining.

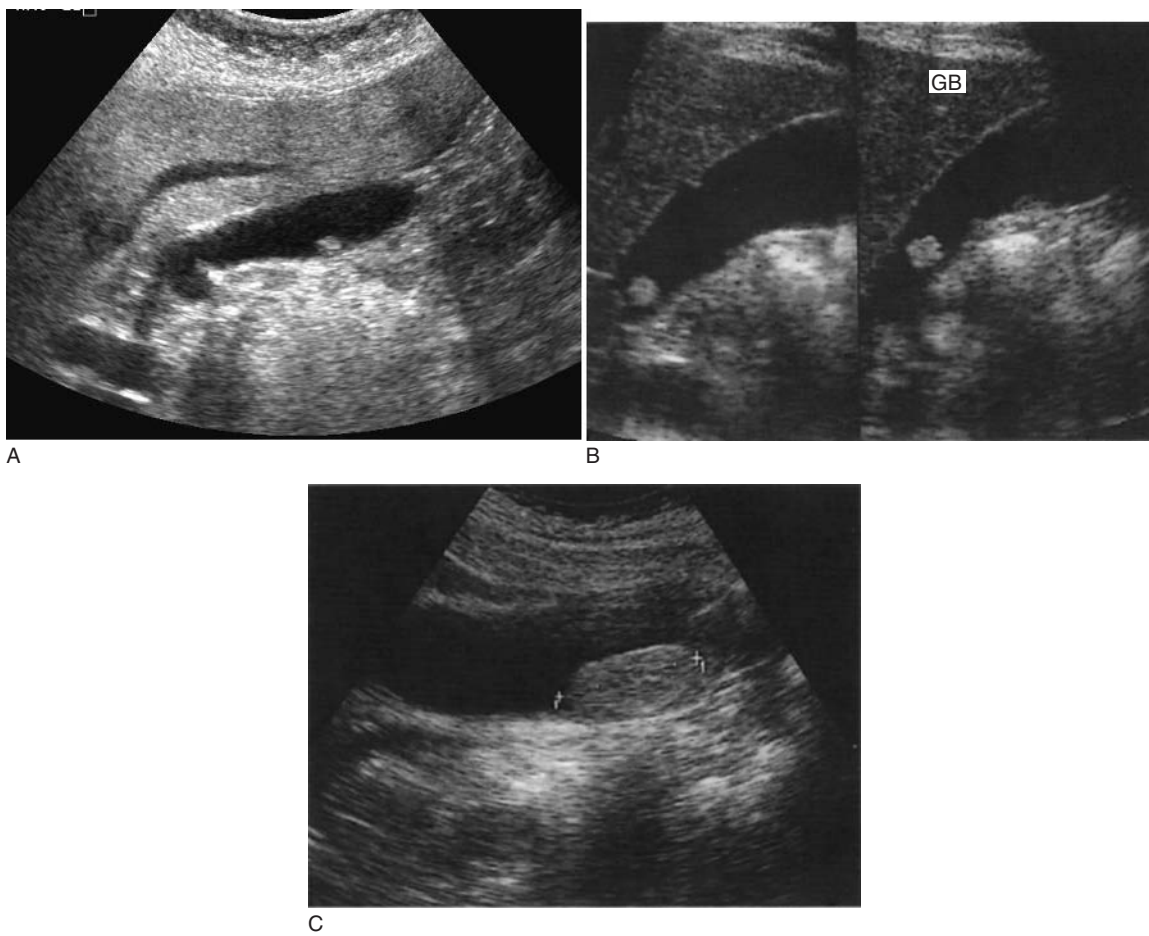


Figure 3.20 (A) Small polyp in the gallbladder lumen—no posterior shadowing is evident. (B) A gallbladder polyp on a stalk moves with different patient positions. (C) Large, fleshy gallbladder polyp.

These nodules are the result of a build-up of lipids in the gallbladder wall and are not usually visible on ultrasound. However in some cases, multiple polyps also form on the inner surface, projecting into the lumen, and are clearly visible on ultrasound (Fig. 3.21). Cholesterolosis may be asymptomatic, or may be accompanied by stones and consequently requires surgery to alleviate symptoms of biliary colic.

INFLAMMATORY GALLBLADDER DISEASE

Cholecystitis is usually associated with gallstones; the frictional action of stones on the gallbladder wall causes some degree of inflammation in almost all cases. The inner mucosa of the wall is injured, allowing the access of enteric bacteria. The inflammatory process may be long-standing and chronic, acute or a combination of acute inflammation on a chronic background.

Acute cholecystitis

Acute inflammation of the gallbladder presents with severe RUQ pain localized to the gallbladder area. The pain can be elicited by (gently!) pressing the gallbladder with the ultrasound transducer—a positive ultrasound Murphy's sign. (This sign, although a useful pointer to acute inflammation, is not specific and can frequently be elicited in other conditions, such as chronic inflammatory cases.)

On ultrasound, the gallbladder wall is thickened greater than 2 mm. This is not in itself a specific sign (see Table 3.3), but characteristically the thickening in acute cholecystitis is symmetrical, affecting the entire wall, and there is an echo-poor 'halo' around the gallbladder as a result of oedematous changes (Fig. 3.22). This is not invariable, however, and focal thickening may be present, or the wall may be uniformly hyperechoic in some cases. Pericholecystic fluid may also be present, and the inflammatory process may spread to the adjacent liver.

Colour or power Doppler can be helpful in diagnosing acute cholecystitis and in differentiating it from other causes of gallbladder wall thickening. Hyperaemia in acute cholecystitis can be demonstrated on colour Doppler around the thickened wall[13] (Fig. 3.23). In a normal gallbladder, colour Doppler flow may be seen around the gallbladder neck in the region of the cystic artery but not elsewhere in the wall. The increased sensitivity of power Doppler, as opposed to colour

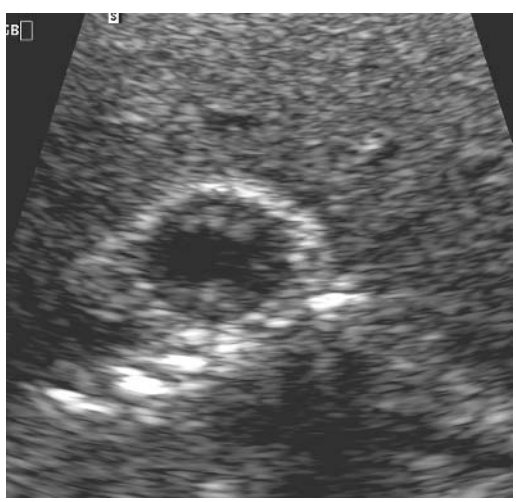


Figure 3.21 Cholesterolosis TS of the gallbladder demonstrating multiple tiny polyps in the gallbladder.

Table 3.3 Causes of a thickened gallbladder wall

Physiological
—Postprandial
Inflammatory
—Acute or chronic cholecystitis
—Sclerosing cholangitis
—Crohn's disease
—AIDS
Adjacent inflammatory causes
—Pancreatitis
—Hepatitis
—Pericholecystic abscesses
Non-inflammatory
—Adenomyomatosis
—Gallbladder carcinoma
—Focal areas of thickening due to metastases or polyps
—Leukaemia
Oedema
—Ascites from a variety of causes, including organ failure, lymphatic obstruction and portal hypertension
Varices
—Varices of the gallbladder wall in portal hypertension

Doppler, does enable the operator to demonstrate vascularity in the normal gallbladder wall and the operator should be familiar with normal appearances for the machine in use when making the diagnosis of acute cholecystitis[14] (Fig. 3.24).

Doppler can potentially distinguish acute inflammation from chronic disease.[15] However, false-positive results can be found in cases of pancreatitis and gallbladder carcinoma and the technique does not add significantly to the grey-scale image.

Complications may occur if the acute inflammation progresses (see below) due to infection, pericholecystic abscesses and peritonitis.

Further management of acute cholecystitis

In an uncomplicated acute cholecystitis, analgesia to settle the patient in the short term is followed by the removal of the gallbladder. Open surgery, which is increasingly reserved for the more

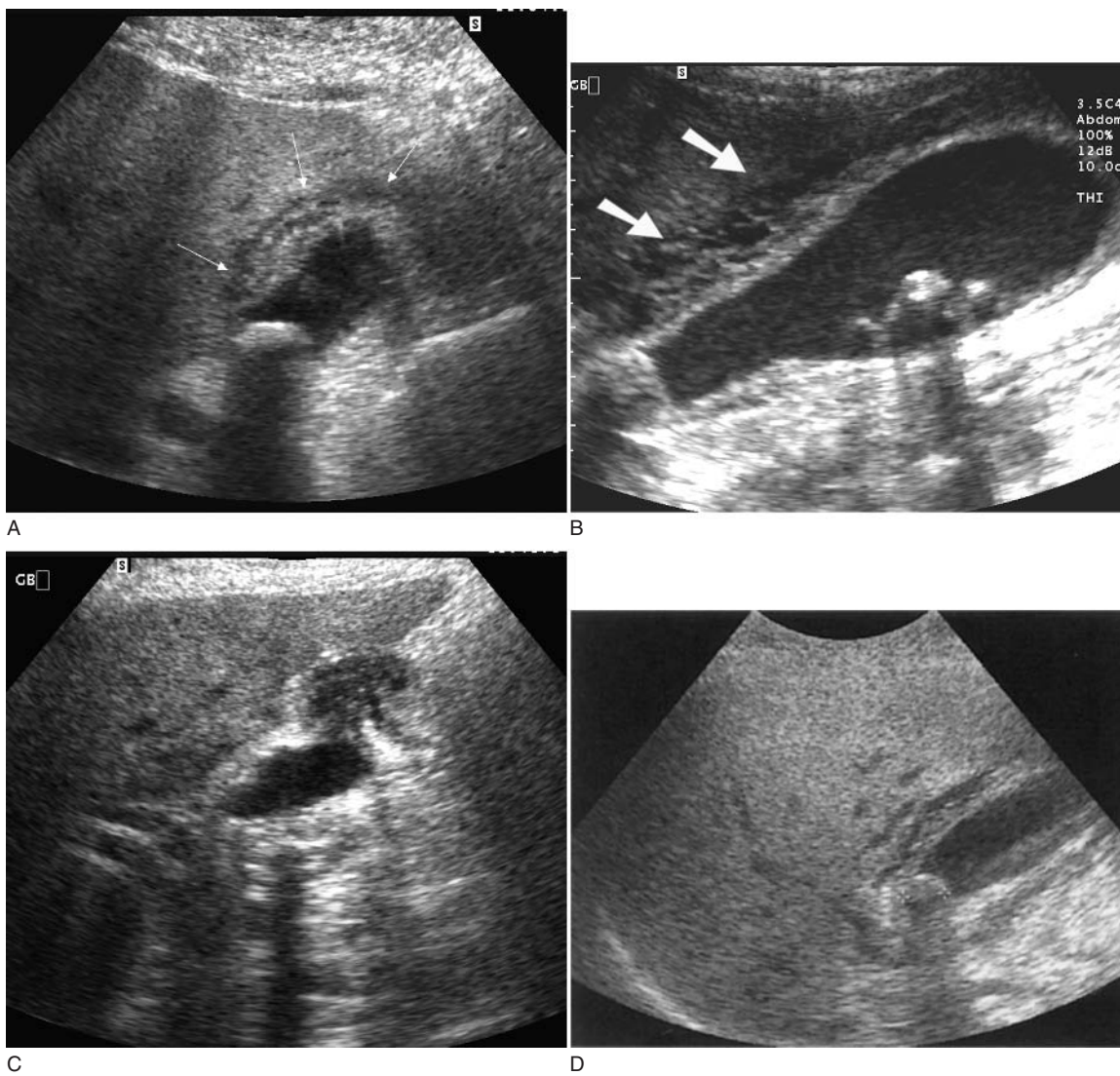


Figure 3.22 Acute cholecystitis: (A) TS of an oedematous, thickened gallbladder wall with a stone. (B) LS with a thickened wall (arrows). Stones and debris are present. (C) and (D) TS and LS demonstrating pericholecystic fluid.

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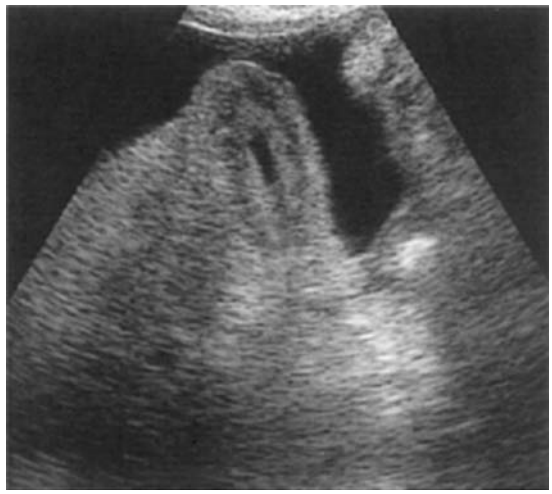


E

Figure 3.22 cont'd (E) Normal gallbladder in the presence of ascites. Oedema may cause the wall to thicken, mimicking an inflammatory process.

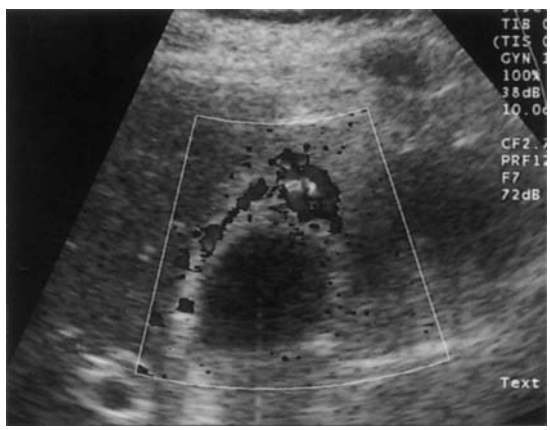


Figure 3.23 Colour Doppler demonstrates hyperaemia in the thickened gallbladder wall in acute cholecystitis.

complex cases, is giving way to the more frequent use of laparoscopic cholecystectomy.

If unsuitable for immediate surgery, for example in cases complicated by peritonitis, the patient is managed with antibiotics and/or percutaneous drainage of pericholecystic fluid or infected bile from the gallbladder, usually under ultrasound guidance. This allows the patient's symptoms to settle and reduces morbidity from the subsequent elective operation.[16]

Hepatobiliary scintigraphy has high sensitivity and specificity for evaluating patients with acute cholecystitis,[17] particularly if the ultrasound examination is technically difficult or equivocal and has the advantage of being able to demonstrate hepatobiliary drainage into the duodenum.

Plain X-ray is seldom used, but can confirm the presence of gas in the gallbladder.

Chronic cholecystitis

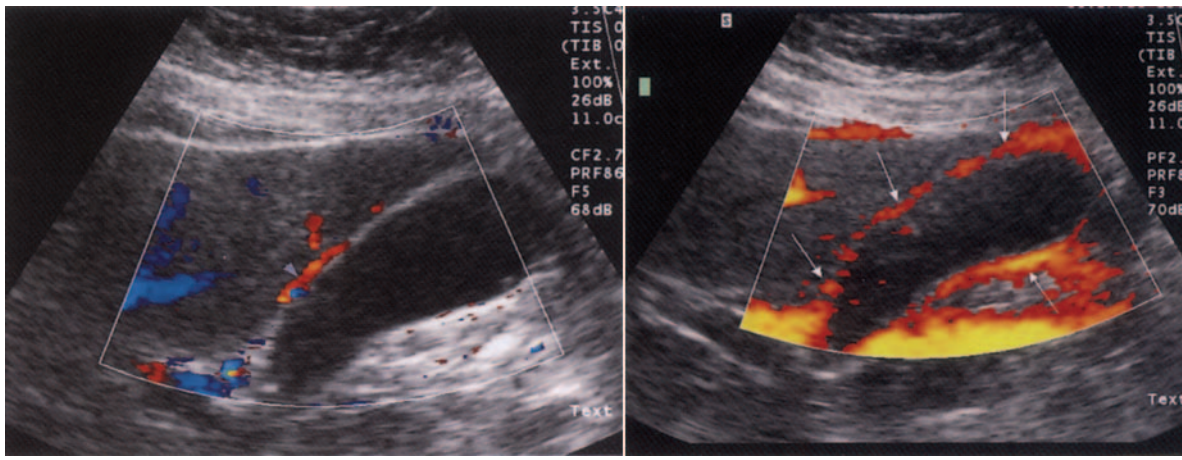
Usually associated with gallstones, chronic cholecystitis presents with lower-grade, recurring right upper quadrant pain. The action of stones on the wall causes it to become fibrosed and irregularly thickened, frequently appearing hyperechoic (Fig. 3.25). The gallbladder is often shrunken and contracted, having little or no recognizable lumen around the stones. Chronic cholecystitis may be complicated by episodes of acute inflammation on a background of the chronic condition.

Most gallbladders which contain stones show at least some histological degree of chronic cholecystitis, even if wall thickening is not apparent on ultrasound.

Acalculous cholecystitis

Inflammation of the gallbladder without stones is relatively uncommon. A thickened, tender gallbladder wall in the absence of any other obvious cause of thickening may be due to acalculous cholecystitis. This condition tends to be associated with patients who are already hospitalized and have been fasting, including post-trauma patients, those recovering from surgical procedures and diabetic patients. It is brought about by bile stasis leading to a distended gallbladder and subsequently decreased blood flow to the gallbladder. This, especially in the weakened postoperative state, can lead to infection. Because no stones are present, the diagnosis is more difficult and may be delayed. Patients with acalculous cholecystitis are therefore more likely to have severe pain and fever by the time the diagnosis is made, increasing the incidence of complications such as perforation.

The wall may appear normal on ultrasound in the early stages, but progressively thickens (Fig. 3.26). Biliary sludge is usually present and a



A B

Figure 3.24 Normal gallbladder wall vascularity. (A) In a normal gallbladder, colour Doppler can demonstrate the cystic artery (arrowhead) but does not demonstrate flow near the fundus. (B) Power Doppler is more sensitive and can demonstrate flow throughout the wall (arrows) in a normal gallbladder; this must not be mistaken for hyperaemia.

pericholecystic abscess may develop in the later stages. A positive Murphy's sign may help to focus on the diagnosis, but in unconscious patients the diagnosis is a particularly difficult one.

Because patients may already be critically ill with their presenting disease, or following surgery, there is a role for ultrasound in guiding percutaneous cholecystostomy at the bed-side to relieve the symptoms.[18]

Chronic acalculous cholecystitis implies a recurrent presentation with typical symptoms of biliary colic, but no evidence of stones on ultrasound. Patients may also demonstrate a low ejection fraction during a cholecystokinin-stimulated hepatic iminodiacetic acid (HIDA) scan. The symptoms are relieved by elective laparoscopic cholecystectomy in most patients, with similar results to those for gallstone disease[19] (although some are found to have biliary pathology at surgery, which might explain the symptoms, such as polyps, cholesterolosis or biliary crystals/tiny stones in addition to chronic inflamation).

Complications of cholecystitis

Acute-on-chronic cholecystitis

Patients with a long-standing history of chronic cholecystitis may suffer (sometimes repeated) attacks of acute inflammation. The gallbladder wall is thickened, as for chronic inflammation, and may become focally thickened with both hypo- and hyperechoic regions. Stones are usually present (Fig. 3.27).

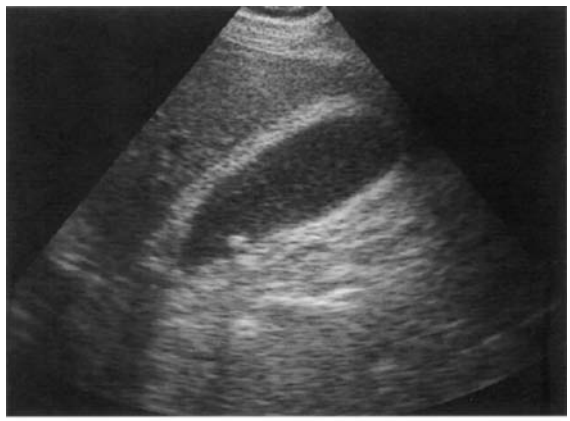
Gangrenous cholecystitis

In a small percentage of patients, acute gallbladder inflammation progresses to gangrenous cholecystitis. Areas of necrosis develop within the gallbladder wall, the wall itself may bleed and small abscesses form (Fig. 3.28). This severe complication of the inflammatory process requires immediate cholecystectomy.

The gallbladder wall is friable and may rupture, causing a pericholecystic collection and possibly peritonitis. Inflammatory spread may be seen in the adjacent liver tissue as a hypoechoic, ill-defined area. Loops of adjacent bowel may become adherent to the necrotic wall, forming a cholecystoenteric fistula.

The wall is asymmetrically thickened and areas of abscess formation may be demonstrated. The damaged inner mucosa sloughs off, forming the appearance of membranes in the gallbladder lumen. The gallbladder frequently contains infected debris

The presence of a bile leak may also be demonstrated with hepatobiliary scintigraphy, using technetium^{99}, which is useful in identifying a bile



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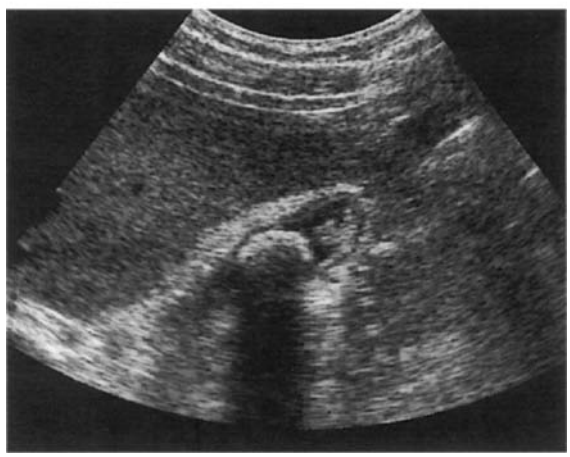


B

Figure 3.25 Chronic cholecystitis. (A) A hyperechoic, irregular, thickened wall. The gallbladder contains a small stone and thickened, echogenic bile. It was mildly tender on scanning. (B) The wall is focally thickened anteriorly, and the gallbladder contains a large stone and a polyp in the fundus.

collection which may otherwise be obscured by bowel on ultrasound.

Emphysematous cholecystitis

This is a form of acute gangrenous cholecystitis in which the inflamed gallbladder may become infected, particularly in diabetic patients, with gas-forming organisms. Both the lumen and the wall of the gallbladder may contain air, which is highly reflective, but which casts a 'noisy', less definite shadow than that from stones. Discrete gas bubbles have been reported on ultrasound within the gallbladder wall[20] and may also extend into the intrahepatic biliary ducts.[21]

The air rises to the anterior part of the gallbladder, obscuring the features behind it (Fig. 3.29). This effect may mimic air-filled bowel on ultrasound.

Emphysematous cholecystitis has traditionally had a much higher mortality rate than other forms of cholecystitis, requiring immediate cholecystectomy. However, improvements in ultrasound resolution, and in the early clinical recognition of this condition, suggest that it is now being diagnosed earlier and may be managed more conservatively. The gas in the gallbladder may be confirmed on a plain X-ray (Fig. 3.30), but ultrasound is more sensitive in demonstrating the earlier stages.

Gallbladder empyema

Empyema is a complication of cholecystitis in which the gallbladder becomes infected behind an obstructed cystic duct. Fine echoes caused by pus are present in the bile (Fig. 3.31). These patients are often very ill with a fever and acute pain. A pericholecystic gallbladder collection may result from leakage through the gallbladder wall with subsequent peritonitis. Ultrasound may be used to guide a bedside drainage in order to allow the patient's symptoms to settle before surgery is attempted.[22]

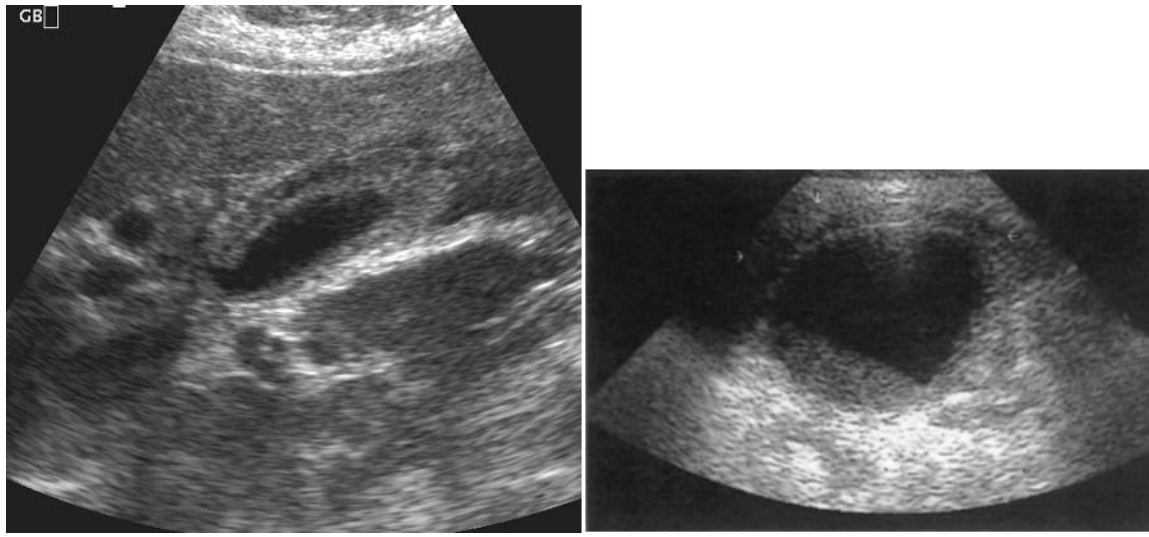
OBSTRUCTIVE JAUNDICE AND BILIARY DUCT DILATATION

Dilatation of all or part of the biliary tree is usually the result of proximal obstruction. Less commonly the biliary tree may be dilated but not obstructed (Table 3.4). The most common causes of obstruction are stones in the common duct or a neoplasm of the bile duct or head of pancreas.

The patient with obstructive jaundice may present with upper abdominal pain, abnormal liver function tests (LFTs) (see Chapter 2) and, if the obstruction is not intermittent, the sclera of the eye and the skin adopt a yellow tinge.

Assessment of the level of obstruction

It is possible for the sonographer to work out where the obstructing lesion is situated by observ-



A B

Figure 3.26 (A) Acalculous cholecystitis. The gallbladder wall is markedly thickened and tender on scanning. (B) Gravity-dependent sludge with a thick, oedematous wall. No stones were present.

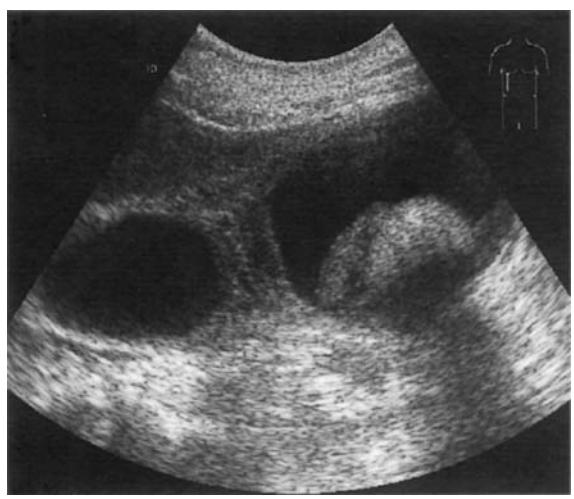


Figure 3.27 Acute on chronic cholecystitis. A patient with known gallstones and chronic cholecystitis presents with an episode of acute gallbladder pain. The wall is considerably more thickened and hyperechoic than on previous scans.

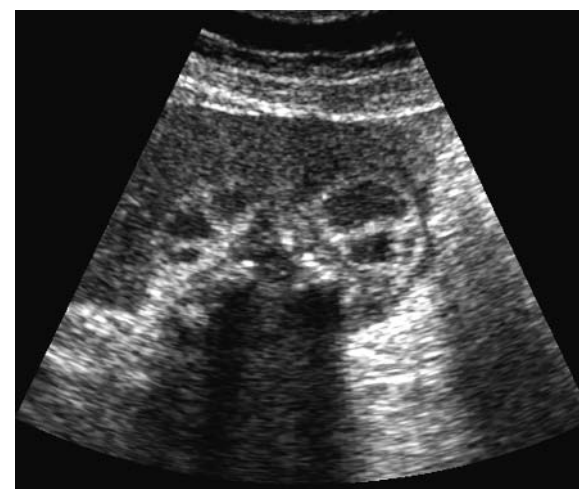


Figure 3.28 Gangrenous cholecystitis. The gallbladder wall is focally thickened and an intramural abscess has formed on the anterior aspect.

ing which parts of the biliary tree are dilated (Fig. 3.32):

- Dilatation of the common bile duct (that is, that portion of the duct below the cystic duct insertion) implies obstruction at its lower end.
- Dilatation of both biliary and pancreatic ducts implies obstruction distally, at the head of the pancreas or ampulla of Vater. This is more likely to be due to carcinoma of the head of pancreas, ampulla or acute pancreatitis than a stone. However, it is possible for a stone to be

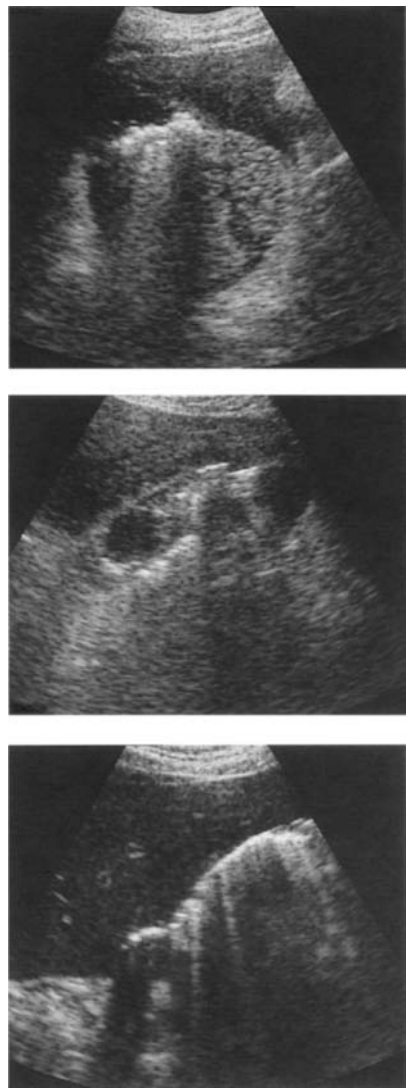


Figure 3.29 Emphysematous cholecystitis. (A) and (B) TS and LS with gas and debris in the gallbladder lumen. (C) Gas in the gallbladder lumen completely obscures the contents.

lodged just distal to the confluence of the biliary and pancreatic ducts.

- Dilatation of the gallbladder alone (that is without ductal dilatation) is usually caused by, obstruction at the neck or cystic duct (Fig. 3.8).

To assess whether the gallbladder is pathologically dilated may be difficult on ultrasound. The sonographer should look at both the size and shape; the dilated gallbladder will have a rounded, bulging shape due to the increase in pressure inside it. A gallbladder whose wall has become fibrosed from chronic cholecystitis due to stones will often lose the ability to distend, so the biliary ducts can look grossly dilated despite the gallbladder remaining 'normal' in size, or contracted.

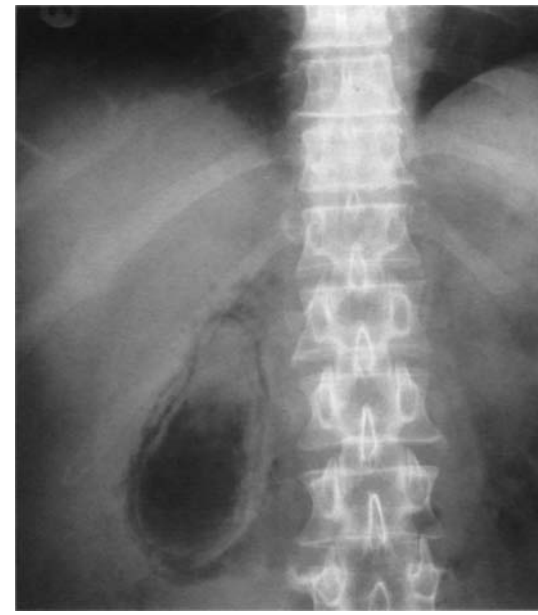


Figure 3.30 X-ray demonstrating gas in the gallbladder in emphysematous cholecystitis.

Early ductal obstruction

Beware very early common duct obstruction, before the duct becomes obviously dilated. The duct may be mildly dilated at the lower end, just proximal to a stone. Likewise intermittent obstruction by a small stone at the lower end of the duct may be non-dilated by the time the scan is performed (Fig. 3.10).

A significant ultrasound feature in the absence of any other identifiable findings is that of thickening of the wall of the bile duct. This represents an inflammatory process in the duct wall, which may be found in patients with small stones in a non-dilated duct, but is also associated with sclerosing cholangitis.[23]

It is sometimes technically difficult in some patients (particularly those with diffuse liver disease) to work out whether a tubular structure on

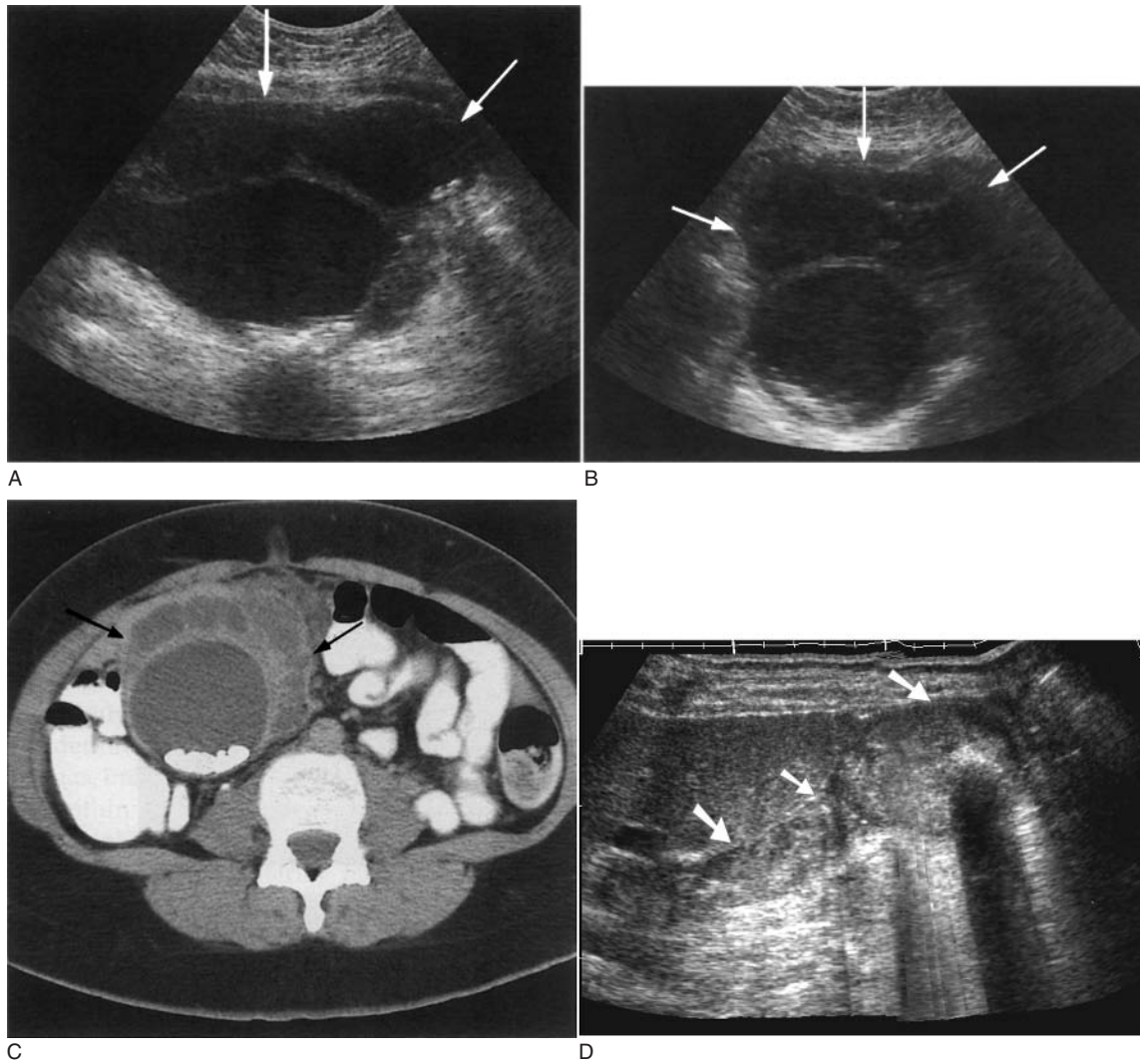


Figure 3.31 Gallbladder empyema. (A) and (B) LS and TS of the same gallbladder. The gallbladder has ruptured, forming a cholecystoenteric fistula which had resealed at surgery. The gallbladder contains pus and stones, with several anterior septations, forming pockets of infected bile which also contained stones (arrows). (C) CT scan confirming the ultrasound appearances. (D) Gallbladder empyema demonstrating a large gallbladder full of pus and stones.

ultrasound represents a dilated duct or a blood vessel. Colour Doppler will differentiate the dilated bile duct from a branch of hepatic artery or portal vein (Fig. 3.33).

Assessment of the cause of obstruction

The numerous causes of biliary dilatation are summarized in Table 3.4. Frequently, ultrasound diagnoses obstruction but does not identify the cause. This is a good case for perseverance by the operator, as the lower end of the CBD is visible in the majority of cases once overlying duodenum has been moved away (Figs 3.9, 3.10 and 33.4). However, ultrasound is not generally regarded as a reliable tool for identifying ductal stones and is frequently unable to diagnose ductal strictures, especially those from benign causes.

Table 3.4 Causes of biliary duct dilatation

Intrinsic
- —Stones
- —Carcinoma of the ampulla of Vater
- —Cholangiocarcinoma
- —Stricture (associated with chronic pancreatitis)
- —Biliary atresia/choledochal cyst
- —Post-liver-transplantation bile duct stenosis (usually anastomotic)
- —Parasites
- —Age-related or post-surgical mild CBD dilatation

Extrinsic
- —Carcinoma of the head of pancreas
- —Acute pancreatitis
- —Lymphadenopathy at the porta hepatis
- —Other masses at the porta, e.g. hepatic artery aneurysm, gastrointestinal tract mass
- —Intra-hepatic tumours (obstruct distal segments)

Diffuse hepatic conditions
- —Sclerosing cholangitis
- —Caroli's disease

ERCP, although invasive, is a more accurate method of examining the CBD and will often identify strictures or small calculi not visible on ultrasound. It has the advantage of a therapeutic role in addition to its diagnostic capabilities, by allowing the extraction of stones at the time of diagnosis. It is associated with a small risk of complication, however, and its use is therefore increasingly limited in favour of the non-invasive magnetic resonance cholangiopancreatography (MRCP) (Fig. 3.34F). MRCP has been found to be highly effective in the diagnosis of CBD stones[24] and can potentially avoid the use of purely diagnostic ERCP.[25]

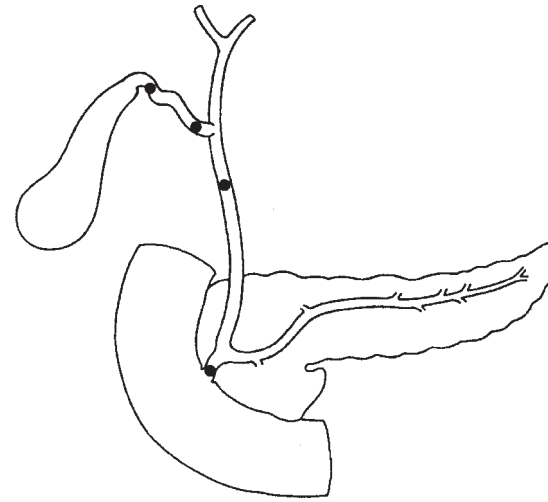


Figure 3.32 Sites of possible gallstone obstruction.

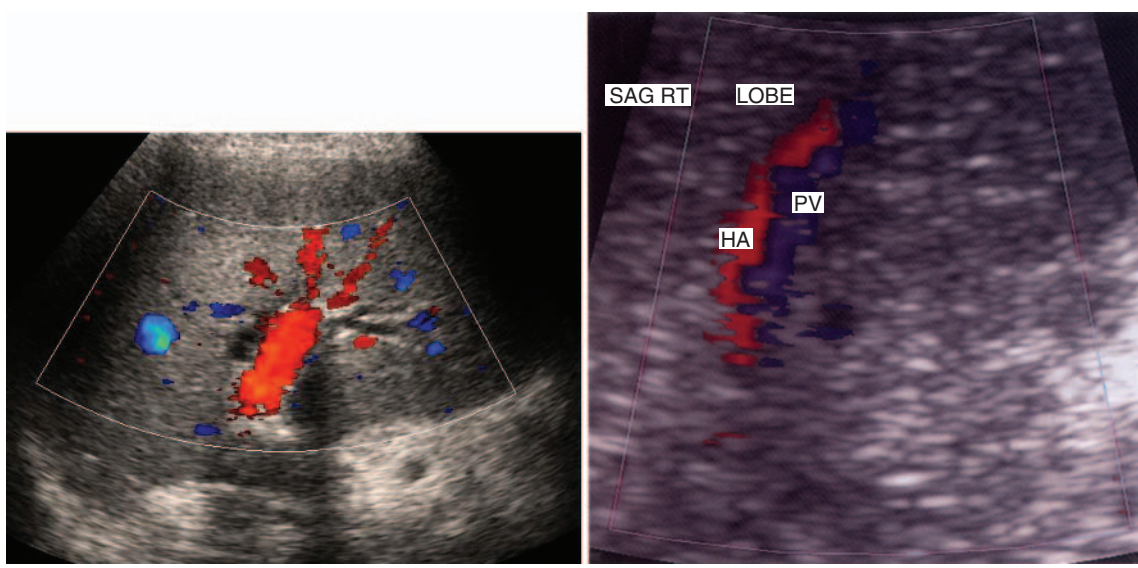


Figure 3.33 (A) Dilated biliary ducts do not demonstrate flow on colour Doppler, differentiating them from portal vessels. (B) Originally suspected as a dilated biliary tree, colour Doppler demonstrates that the 'extra tubes' are, in fact, dilated intrahepatic arteries in a patient with end-stage chronic liver disease with reversed portal venous flow.

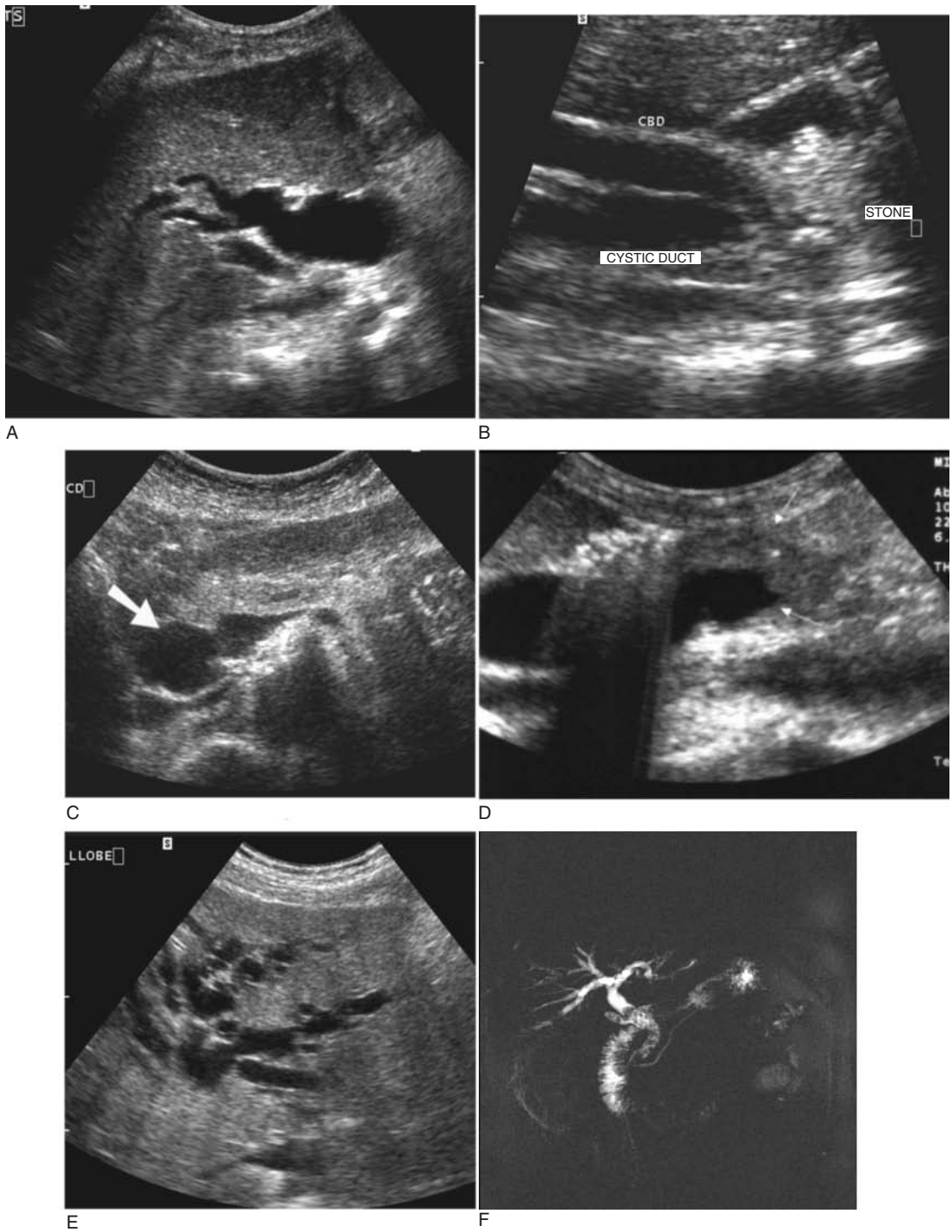


Figure 3.34 (A) Duodenal gas obscures the cause of obstruction at the lower end of this dilated CBD. (B) Patient positioning can move bowel gas away from the duct, demonstrating the cause of obstruction—a stone at the lower end. (C) TS of a dilated CBD in the head of the pancreas (arrow). (D) Dilated CBD with a hypoechoic ampullary carcinoma at the lower end (arrows). (E) Intrahepatic bile duct dilatation. (F) MRCP, post-cholecystectomy, showing stones in the CBD and cystic duct stump.

CT and MRI are useful for staging purposes if the obstructing lesion is malignant. Cholangiocarcinomas spread to the lymph nodes and to the liver and small liver deposits are particularly difficult to recognize on ultrasound if the intrahepatic biliary ducts are dilated.

In hepatobiliary scintigraphy, technetium^{99m}-labelled derivatives of iminodiacetic acid are excreted in the bile and may help to demonstrate sites of obstruction, for example in the cystic duct, or abnormal accumulations of bile, for example choledochal cysts.

Courvoisier's law, to which there are numerous exceptions, states that if the gallbladder is dilated in a jaundiced patient, then the cause is *not* due to a stone in the common duct. The reason for this is that, if stones are or had been present, then the gallbladder would have a degree of wall fibrosis from chronic cholecystitis which would prevent it from distending. In fact there are many exceptions to this 'law' which include the formation of stones in the duct, without gallbladder stones, and also obstruction by a pancreatic stone at the ampulla. Thus:

- Do not assume that obstructive jaundice in a patient with gallstones is due to a stone in the CBD. The jaundice may be attributable to other causes.
- Do not assume that obstructive jaundice cannot be due to a stone in the CBD if the gallbladder does not contain stones. A solitary stone can be passed into the duct from the gallbladder or stones can form within the duct.

Management of biliary obstruction

Management of biliary obstruction obviously depends on the cause and the severity of the condition. Removal of stones in the CBD may be performed by ERCP with sphincterotomy. Elective cholecystectomy may take place if gallstones are present in the gallbladder.

Laparoscopic ultrasound is a useful adjunct to surgical exploration of the biliary tree and its accuracy in experienced hands equals that of X-ray cholangiography. It is rapidly becoming the imaging modality of choice to examine the ducts during laparoscopic cholecystectomy.[26]

Endoscopic ultrasound can also be used to examine the CBD, avoiding the need for laparoscopic exploration of the duct when performed in the immediate preoperative stage.[27]

The treatment of malignant obstruction is determined by the stage of the disease. Accurate staging is best performed using CT and/or MRI. If surgical removal of the obstructing lesion is not a suitable option because of local or distant spread, palliative stenting may be performed endoscopically to relieve the obstruction and decompress the ducts (Fig. 3.35). The patency of the stent may be monitored with ultrasound scanning by assessing the degree of dilatation of the ducts.

Clinical suspicion of early obstruction should be raised if the serum alkaline phosphatase is elevated, (often more sensitive in the early stages than a raised serum bilirubin). In the presence of ductal dilatation on ultrasound, further imaging, such as CT or MRCP, may then refine the diagnosis.

Intrahepatic tumours causing biliary obstruction

Focal masses which cause segmental intrahepatic duct dilatation are usually intrinsic to the duct itself, for example cholangiocarcinoma.

It is also possible for a focal intrahepatic mass, whether benign or malignant, to compress an adjacent biliary duct, causing subsequent obstruction of that segment. This is not, however, a common cause of biliary dilatation and occurs most usually with hepatocellular carcinomas.[28] Most liver metastases deform rather than compress adjacent structures and biliary obstruction only occurs if the metastases are very large and/or invade the biliary tree. A hepatocellular carcinoma or metastatic deposit at the porta hepatis may obstruct the common duct by squeezing it against adjacent extrahepatic structures. Benign intrahepatic lesions rarely cause ductal dilatation, but occasionally their sheer size obstructs the biliary tree.

Choledochal cysts

Most commonly found in children, this is associated with biliary atresia, in which the distal 'blind' end of the duct dilates into a rounded, cystic mass in response to raised intrahepatic pressure.

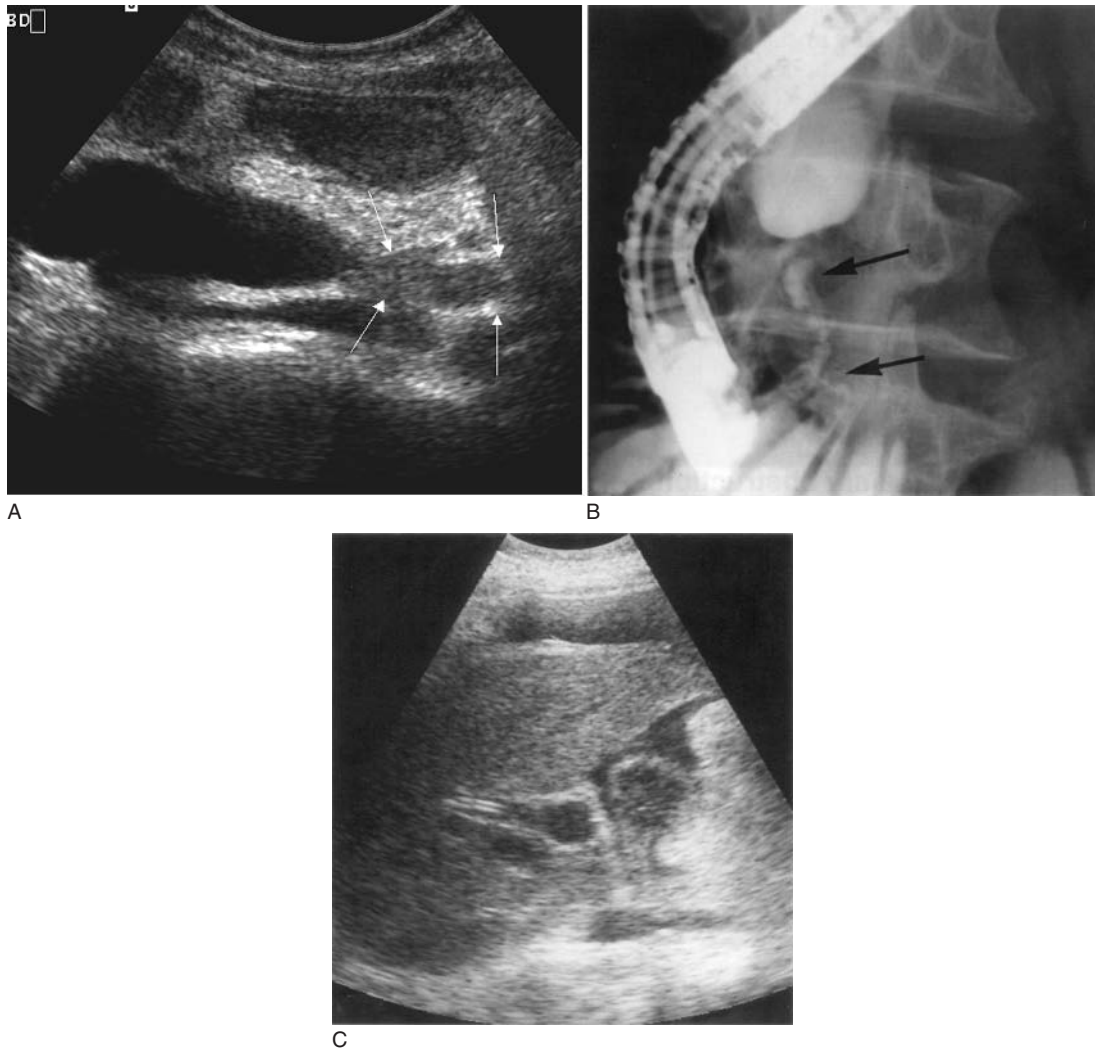


Figure 3.35 (A) This dilated CBD is obstructed by a mass (*arrows*) invading the lower end. (B) ERCP demonstrates a tight, malignant stricture, and can be used to position a palliative stent. (C) Stent in the CBD of a patient with a cholangiocarcinoma and malignant ascites. Decompression of the dilated biliary tree has been achieved, and ultrasound can be used to monitor the patency of the stent.

Choledochal cysts in adults are rare, and tend to be asymptomatic unless associated with stones or other biliary disease. They are sometimes associated with an anomalous insertion of the CBD into the pancreatic duct. The mechanism of the subsequent choledochal cyst formation is unclear, but it is thought that the common channel, which drains into the duodenum, is prone to reflux of pancreatic enzymes into the biliary duct. This can cause a biliary stricture, with subsequent proximal dilatation of the duct, forming a choledochal cyst[29] [Fig. 3.36].

Less commonly the dilatation is due to a non-obstructive cause in which the biliary ducts themselves become ectatic and can form diverticula. This may be due to a focal stricture of the duct which causes reflux and a localized enlargement of the duct proximal to the stricture. (See also *Caroli's disease*, below (Fig. 3.42.)

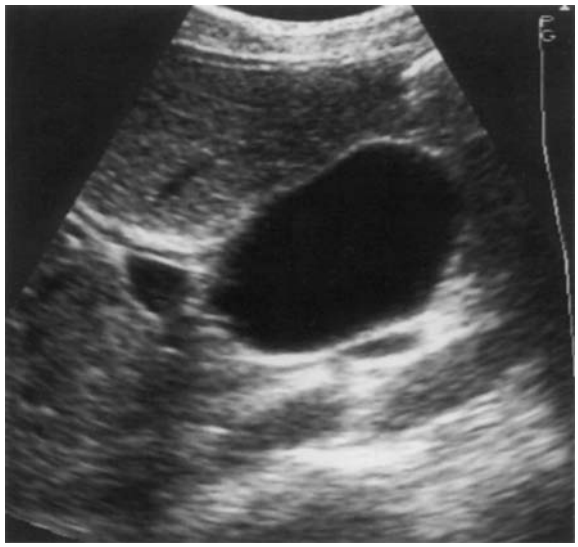


Figure 3.36 Choledochal cyst. (These can sometimes be difficult to distinguish from a gallbladder, particularly if large.)

Complications of choledochal cysts include cholangitis, formation of stones and progression of the condition to secondary biliary cirrhosis, which may be associated with portal hypertension.

It may be difficult to differentiate a choledochal cyst, particularly if solitary, from other causes of hepatic cysts. The connection between the choledochal cyst and the adjacent biliary duct may be demonstrated with careful scanning.

Cholangitis

Cholangitis is an inflammation of the biliary ducts, most commonly secondary to obstruction.

It is rarely possible to distinguish cholangitis from simple duct dilatation on ultrasound, although in severe cases the ductal walls appear irregular (Fig. 3.10A) and debris can be seen in the larger ducts (Fig. 3.37).

The walls of the ducts may appear thickened. Care should be taken to differentiate this appearance from tumour invasion and further imaging is often necessary to exclude malignancy.

Bacterial cholangitis is the most common form, due to bacterial infection which ascends the biliary tree. Bacterial cholangitis is also associated with biliary enteric anastomoses. It may be complicated by abscesses if the infection is progressive and

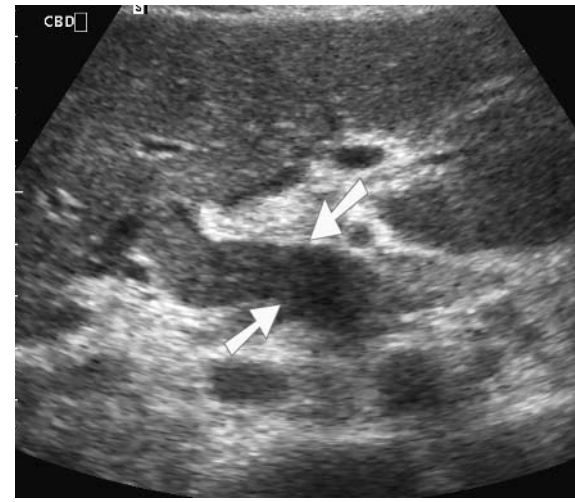


Figure 3.37 Cholangitis with debris present in the dilated CBD (*arrows*).

untreated. Small abscesses may be difficult to diagnose on ultrasound, as they are frequently isoechoic and ill-defined in the early stages and biliary dilatation makes evaluation of the hepatic parenchyma notoriously difficult.

Contrast CT will often identify small abscesses not visible on ultrasound, and MRCP or ERCP demonstrates mural changes in the ducts.

Other forms of cholangitis include:

- Primary sclerosing cholangitis, a chronic, progressive cholestatic disease, which exhibits ductal thickening, focal dilatation and strictures (see p. 67).
- AIDS-related cholangitis which causes changes similar to that of primary sclerosing cholangitis.
- Recurrent pyogenic cholangitis (Oriental cholangiohepatitis) which is endemic in Southeast Asia and is associated with parasites and malnutrition. Intrahepatic biliary stones are also a feature of this condition.

BILIARY DILATATION WITHOUT JAUNDICE

Postsurgical CBD dilatation

In patients who have had cholecystectomy associated with previous dilatation of the CBD it is common to find a persistent (but non-significant) mild dilatation of the duct postoperatively. The serum alkaline

phosphatase and bilirubin levels should be normal in the absence of pathology. Because stones may be found in the duct postoperatively, it is important to differentiate non-obstructive from truly obstructive dilatation in a symptomatic patient (Fig. 3.38). If in doubt, the patient may be rescanned at a suitable interval to assess any increase in ductal diameter.

Focal obstruction

Intrahepatic tumour, such as cholangiocarcinoma, may obstruct a segment of the biliary tree whilst the remainder of the liver and biliary tree appears normal. Focal duct dilatation should trigger the operator to examine the proximal area of dilatation for a possible mass. Such tumours may be present before jaundice is clinically apparent.

Pitfalls

Patients with cirrhosis and portal hypertension may have dilated hepatic arteries which can mimic the appearances of dilated ducts. Colour or power Doppler will readily differentiate between these, as the bile duct lacks a Doppler signal. Pneumobilia (air in the ducts) casts a distal acoustic shadow, and may therefore obscure ductal dilatation.

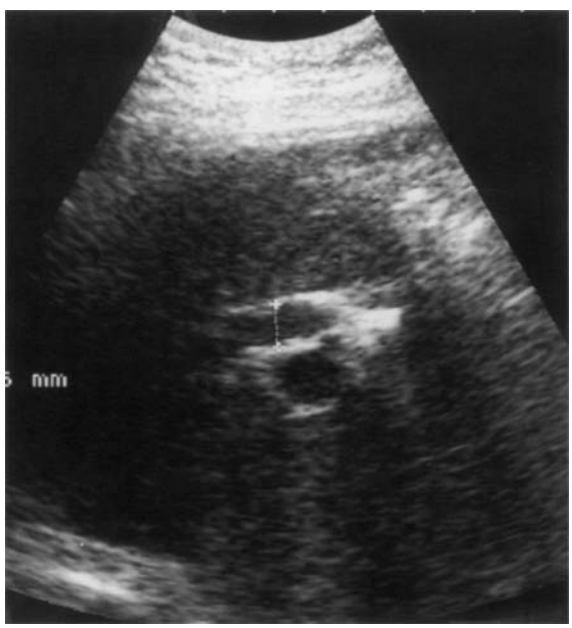


Figure 3.38 Biliary dilatation following laparoscopic cholecystectomy, due to a surgical clip across the CBD.

OBSTRUCTION WITHOUT BILIARY DILATATION

Early obstruction

It is possible to scan a patient at the time of recent onset of obstruction from a stone before the ducts have had time to dilate, leading to a false-negative diagnosis. If clinical suspicion persists, a rescan is frequently useful in these cases.

Occasionally, stones have a ball-valve effect in the duct, causing intermittent obstruction which may not demonstrate ductal dilatation on the ultrasound scan.

Fibrosis of the duct walls

There are a number of chronic pathological conditions which cause the walls of the ducts to become fibrotic and stiff. These include primary sclerosing cholangitis (see below), hepatitis and other chronic hepatic diseases leading to cirrhosis. The liver itself becomes rigid and this prevents biliary dilatation. In such cases the lack of dilated bile ducts does not necessarily imply an absence of obstruction.

OTHER BILIARY DISEASES

Primary sclerosing cholangitis (PSC)

PSC is a chronic hepatobiliary disease in which the walls of the bile ducts become inflamed, causing narrowing. It occurs predominantly in young men (with a 2:1 male to female ratio) and is characterized by multiple biliary strictures and bead-like dilatations of the ducts. The aetiology of PSC remains unclear but is associated with inflammatory bowel disorders or may be idiopathic.

Clinical features include jaundice, itching and fatigue. Some 25% of patients also have gallstones, which complicates the diagnosis. Approximately 70% of patients affected also have ulcerative colitis.

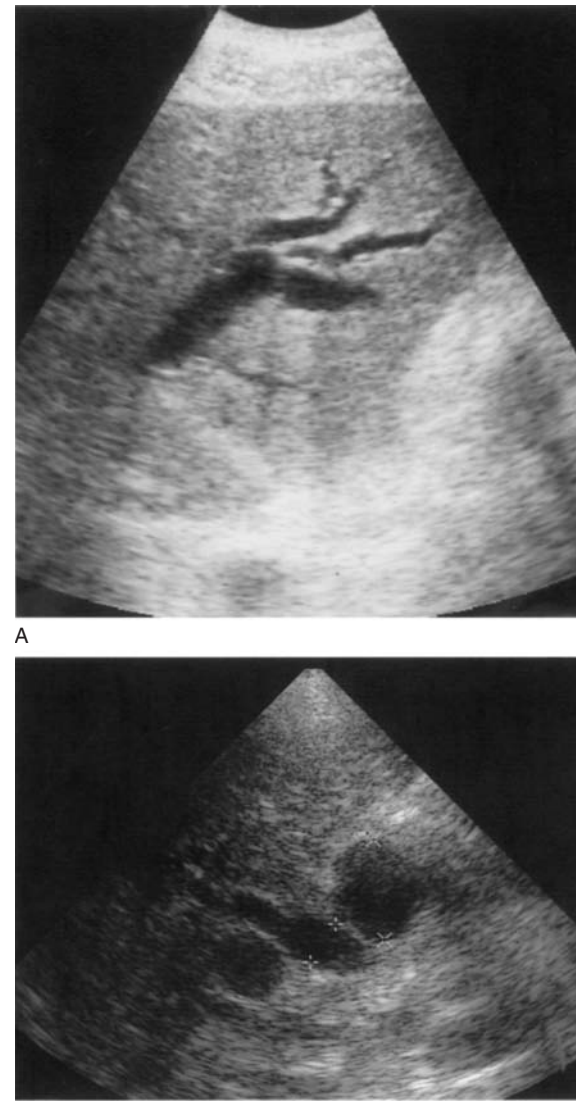
It is progressive gradual fibrosis which eventually obliterates the biliary tree. Untreated, this eventually leads to hepatic failure. PSC has a strong association with cholangiocarcinoma, and it is this, rather than hepatic failure, which may lead to death. In the absence of malignancy, however, hepatic transplant has a 70–90% 5-year survival rate.[30]

Ultrasound appearances

The ultrasound appearances in PSC may be normal or may demonstrate a coarse, hyperechoic texture throughout the liver. Ductal strictures may cause downstream dilatation in some segments (Fig. 3.39) and in some cases there is marked biliary dilatation, but in the majority of patients the biliary ducts are prevented from dilatation by the surrounding fibrosis and so appear unremarkable on ultrasound. MRCP is superior at demonstrating intrahepatic ductal strictures. Mural thickening, particularly in the CBD, may be demonstrated with careful, high-resolution scanning[31] (Fig. 3.40).

Ultrasound also demonstrates the effects of portal hypertension in advanced disease. The gallbladder may also have a thickened wall and can be dilated.[32]

Due to the association between PSC and cholangiocarcinoma, which may be multifocal, a careful search must be made for mass lesions. Because the ultrasound appearances may be those of a coarse, nodular liver texture, it is difficult to identify small cholangiocarcinomas and colour or power Doppler may be an advantage here (Fig. 3.41). This diagnosis is an important one, because the patient's prognosis and management are affected by the presence of cholangiocarcinomata. If no masses are identified, the prognosis is good and includes the endoscopic removal of stones to relieve symptoms, endoscopic stenting of main duct strictures to relieve jaundice and subsequent liver transplant to pre-empt the formation of carcinoma. However, if carcinoma is already present, 5-year survival falls to 10%.



A

B

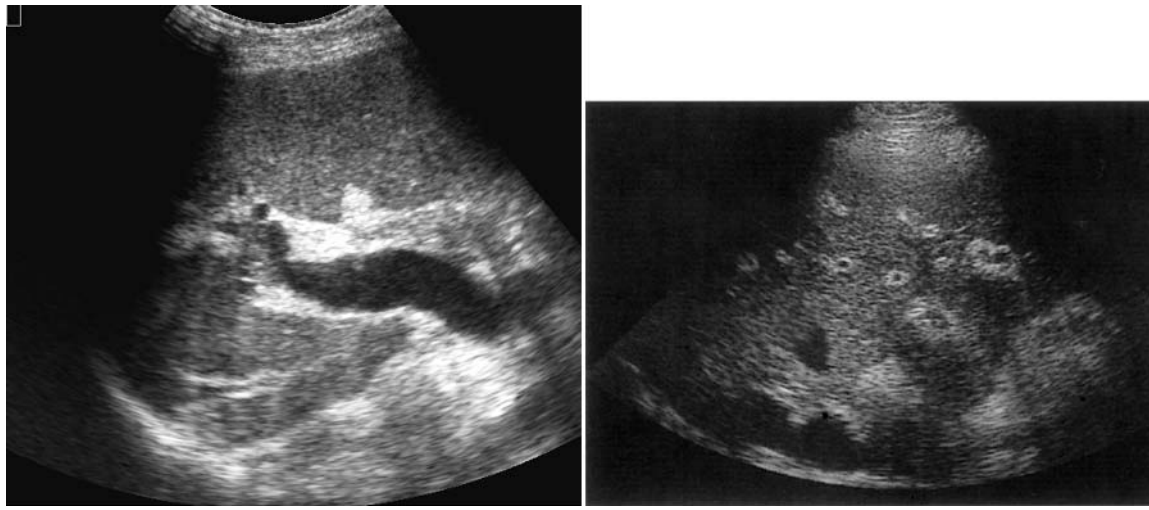
Figure 3.39 (A) Localized biliary dilatation due to a ductal stricture in a patient with primary sclerosing cholangitis (PSC). (B) Coarse-textured liver with a dilated CBD in PSC. A small choledochal cyst is present just anterior to the lower CBD.

Caroli's disease (congenital intrahepatic biliary dilatation)

This is a rare, congenital condition in which the bile ducts are irregularly dilated with diverticula-like projections. These diverticula may become infected and may separate off from the biliary duct, forming choledochal cysts (Fig. 3.42).

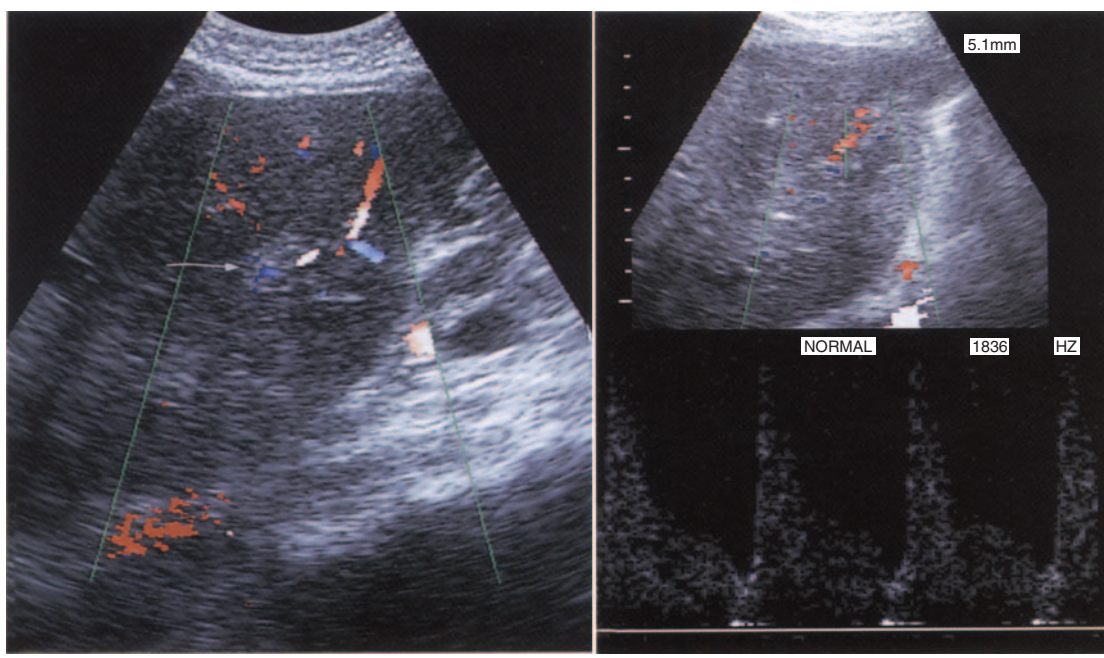
In most cases, the entire hepatobiliary system is affected to some degree. Sufferers may present in early childhood, with symptoms of portal hypertension, [33] or may remain well until adulthood, presenting with cholangitis. It is generally thought to be an autosomal recessive inherited condition and the prognosis is poor. Medical control of associated portal hypertension with varices can improve the quality of life.

In a few cases, the disease is confined to one or two segments of the liver, in which case a cure can be effected with hepatic resection.[34] The extrahepatic biliary tree is often unaffected.



A B

Figure 3.40 PSC. Hyperechoic mural thickening of the biliary tree can be seen in (A) the CBD and (B) the intrahepatic ducts.



A B

Figure 3.41 PSC. (A) A tiny, suspicious, hyperechoic focal lesion (arrow) demonstrates increased flow on colour Doppler. (B) The spectral waveform confirms vigorous arterial flow in this small cholangiocarcinoma.

The ultrasound appearances are usually of widespread intrahepatic duct dilatation, with both saccular and fusiform biliary ectasia. Because it is also associated with biliary stone formation, the diagnosis is often not clear. The dilatation is also associated with cholangitis and signs of infection may be present in the form of debris within the ducts. Sometimes, frank choledocal cysts can be located.

Advanced disease is associated with portal hypertension and, in some cases, cholangiocarcinoma.[35]

Parasites

Parasitic organisms, such as the *Ascaris* worm and liver fluke, are extremely rare in the UK. However, they are a common cause of biliary colic in Africa,

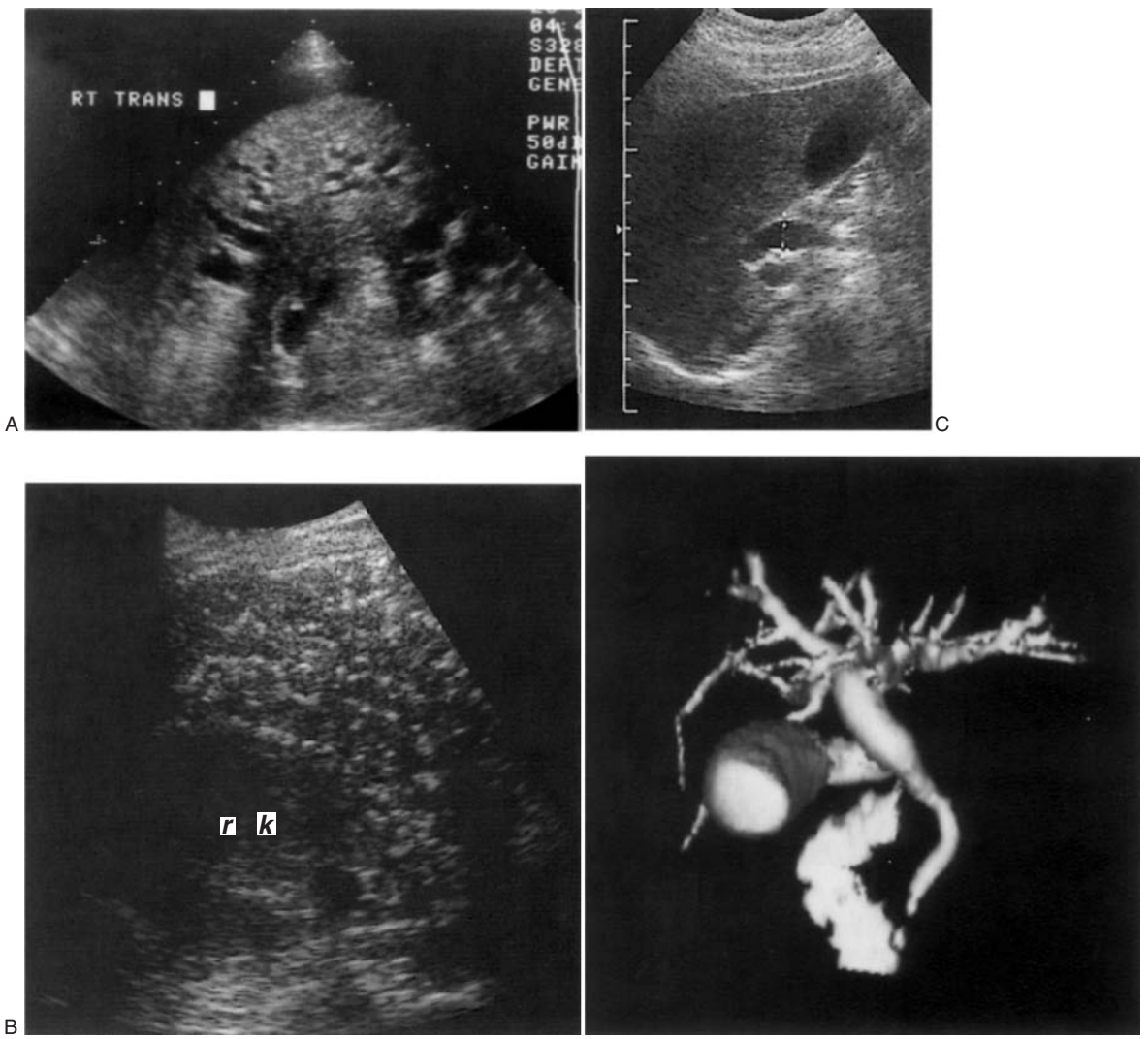


Figure 3.42 Caroli's disease. (A) Dilated biliary tree and ascites. (B) TS of a different patient with end-stage disease. The grossly abnormal liver texture contrasts with the right kidney. (C) A small section of focal CBD dilatation persisted in a symptomatic patient, with normal-calibre distal CBD. This was confirmed on ERCP and thought to be a dyskinetic segment, causing biliary reflux, but was later diagnosed as a mild form of Caroli's. (D) 3D CT reconstruction of the case in (C), confirming the ultrasound appearances. Note the tiny ectatic 'pouchings' of the intrahepatic ducts characteristic of Caroli's.

the Far East and South America. The hyperechoic linear structures in the gallbladder lumen should raise the sonographer's suspicion in patients native to, or who have visited these countries. Impacted worms in the biliary ducts may mimic other ductal masses.[36]

They are a rare cause of obstructive biliary dilatation (Fig. 3.43).

Patients may present with acute cholangitis or abdominal pain and vomiting. Endoscopic management is frequently highly effective.[37]

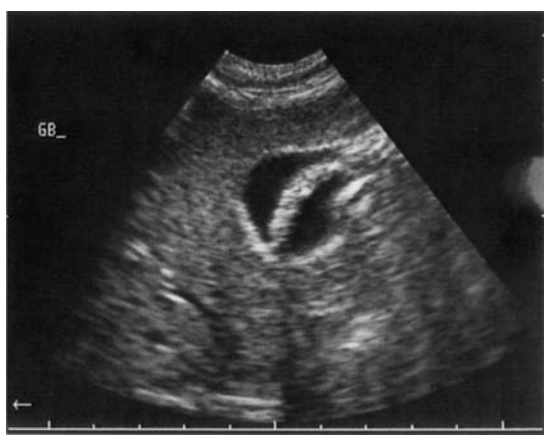


Figure 3.43 *Ascaris* worm in the gallbladder.

ECHOGENIC BILE

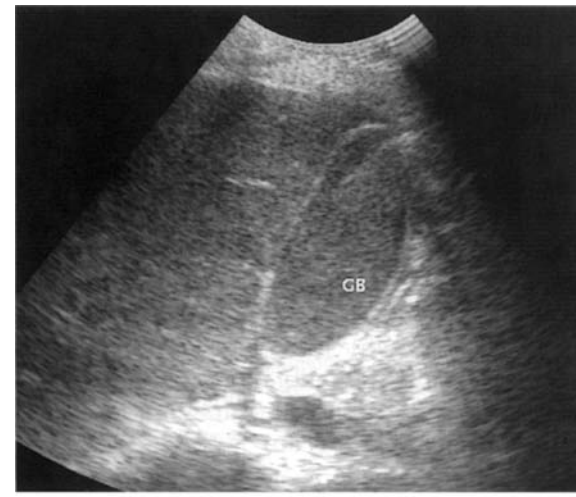
Biliary stasis

Fine echoes in the bile within the gallbladder are not uncommon on an ultrasound scan. This is commonly due to the inspissation of bile following prolonged starving, for example following surgery (Fig. 3.44). These appearances disappear after a normal diet is resumed and the gallbladder has emptied and refilled.

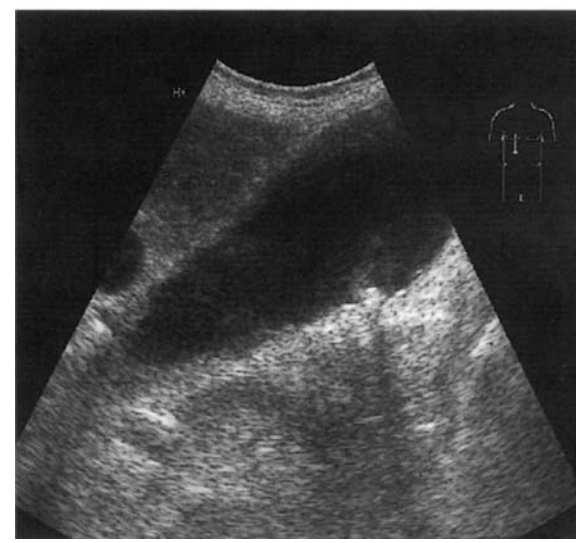
It occurs when the solutes in the bile precipitate, often due to hypomotility of the gallbladder, and can commonly be seen following bone marrow transplantation and in patients who have undergone prolonged periods (4–6 weeks) of total parenteral nutrition.[38]

Prolonged biliary stasis may lead to inflammation and/or infection, particularly in postoperative patients and those on immunosuppression (Fig. 3.44B). Its clinical course varies from complete resolution to progression to gallstones. However, following the resumption of oral feeding, the gallbladder may contract and empty the sludge into the biliary tree causing biliary colic, acute pancreatitis and/or acute cholecystitis.[39] For this reason, cholecystectomy may be considered in symptomatic patients with biliary sludge.

The fine echoes may form a gravity-dependent layer and may clump together, forming 'sludge balls'. To avoid misdiagnosing sludge balls as polyps, turn the patient to disperse the echoes or



A



B

Figure 3.44 (A) Inspissated bile in the normal gallbladder of a fasting patient. (B) Gravity-dependent biliary sludge with a small stone.

rescan after the patient has resumed a normal diet.

Biliary stasis is associated with an increased risk of stone formation.[40]

Biliary crystals

Occasionally, echogenic bile persists even with normal gallbladder function (Fig. 3.45). The significance of this is unclear. It has been suggested that there is a spectrum of biliary disease in which gallbladder dysmotility and subsequent saturation of the bile lead to the formation of crystals in the bile and also in the gallbladder wall, leading eventually to stone formation.[41] Pain and biliary colic may be present prior to stone formation and the presence of echogenic bile seems to correlate with the presence of biliary crystals.[42]

Biliary crystals, or 'microlithiasis' (usually calcium bilirubinate granules) have a strong association with acute pancreatitis[43] and its presence in patients who do not have gallstones is therefore highly significant.

Obstructive causes of biliary stasis

Pathological bile stasis in the gallbladder is due to obstruction of the cystic duct (from a stone, for example) and may be demonstrated in a normal-sized or dilated gallbladder. The bile becomes viscous and hyperechoic. The biliary ducts remain normal in calibre. Eventually the bile turns watery and appears echo-free on ultrasound; this is known as a mucocoele (see above) (Fig. 3.8).

Bile stasis within the ducts occurs either as a result of prolonged and/or repetitive obstruction or as a result of cholestatic disease such as primary biliary cirrhosis (PBC) (Chapter 4) or PSC. This can lead to cholangitis.

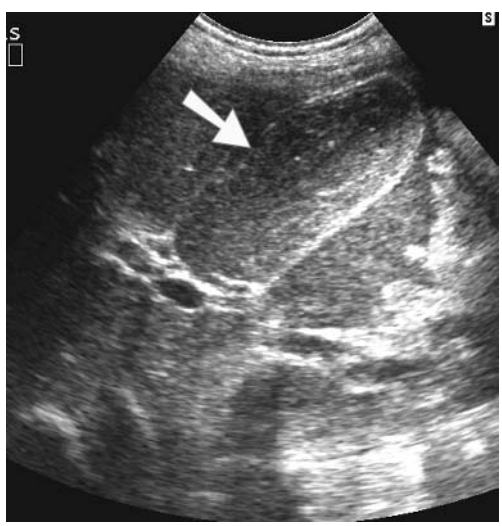


Figure 3.45 Biliary crystals.

Haemobilia

Blood in the gallbladder can be the result of gastrointestinal bleeding or other damage to the gallbladder or bile duct wall, for example iatrogenic trauma from an endoscopic procedure.

The appearances depend upon the stage of evolution of the bleeding. Fresh blood appears as fine, low-level echoes. Blood clots appear as solid, non-shadowing structures and there may be hyperechoic, linear strands.[44]

The history of trauma will allow the sonographer to differentiate from other causes of haemobilia and echogenic bile, particularly those associated with gallbladder inflammation, and there may be other evidence of abdominal trauma on ultrasound such as a haemoperitoneum.

Pneumobilia

Air in the biliary tree is usually iatrogenic and is frequently seen following procedures such as ERCP, sphincterotomy or biliary surgery. Although it does not usually persist, the air can remain in the biliary tree for months or even years and is not significant.

It is characterized by highly reflective linear echoes (Fig. 3.46), which follow the course of the biliary ducts. The air usually casts a shadow which is different from that of stones, often having reverberative artefacts and being much less well-defined or clear. This shadowing obscures the lumen of the duct and can make evaluation of the hepatic parenchyma difficult.

Pneumobilia may also be present in emphysematous cholecystitis, an uncommon complication of cholecystitis in which gas-forming bacteria are present in the gallbladder (see above), or in cases where a necrotic gallbladder has formed a cholecystoenteric fistula.

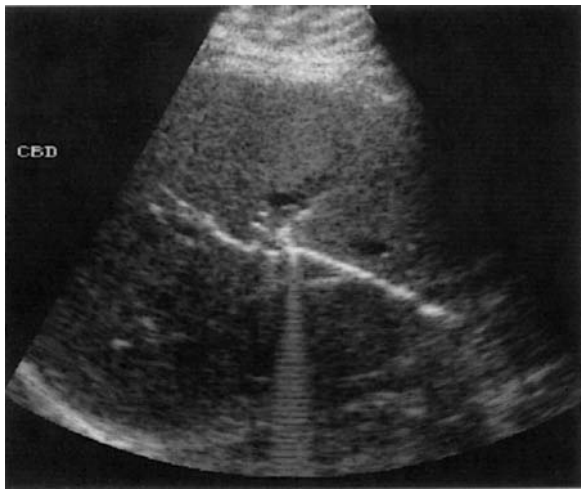


Figure 3.46 Air in the biliary tree following surgery. Note the 'reverberative' shadow.

Rarely, multiple biliary stones form within the ducts throughout the liver and can be confused with the appearances of air in the ducts.

MALIGNANT BILIARY DISEASE

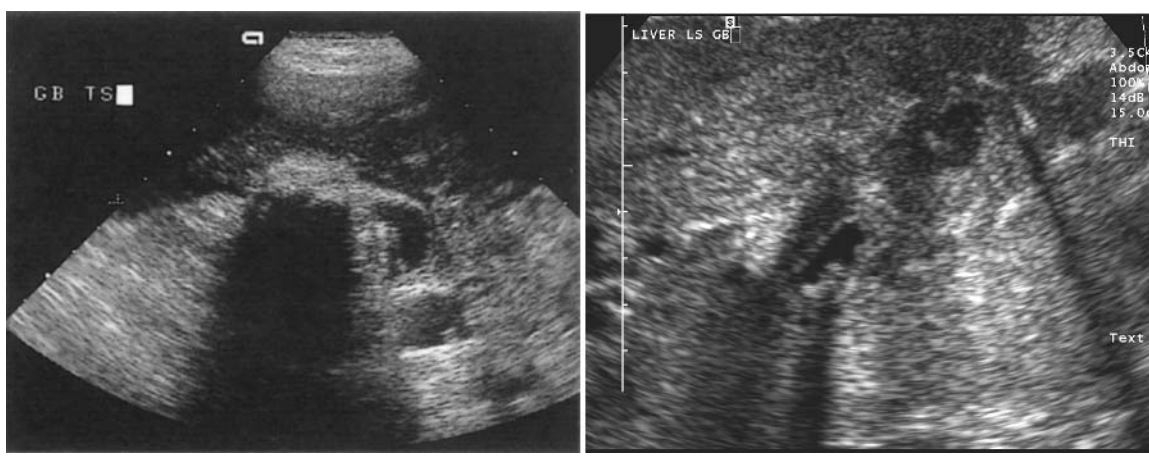
Primary gallbladder carcinoma

Cancer of the gallbladder is usually associated with gallstones and a history of cholecystitis. Most often, the gallbladder lumen is occupied by a solid mass which may have the appearance of a large polyp. The wall appears thickened and irregular and shadowing from the stones may obscure it posteriorly. A bile-filled lumen may be absent, further complicating the ultrasound diagnosis (Fig. 3.47). In a porcelain gallbladder (calcification of the gallbladder wall), which is associated with gallbladder carcinoma, the shadowing usually obscures any lesion in the lumen, making the detection of any lesion present almost impossible.

Particular risk factors for gallbladder carcinoma include large stones, polyps of over 1 cm in size, porcelain gallbladder and, occasionally, choledochal cyst due to anomalous junction of the pancreatobiliary ducts.[8]

The carcinoma itself is frequently asymptomatic in the early stages, and patients tend to present with symptoms relating to the stones. It is a highly malignant lesion which quickly metastasizes to the liver and portal nodes and has a very poor prognosis, with a curative surgical resection rate of around 15–20%.

Doppler may assist in differentiating carcinoma from other causes of gallbladder wall thickening,[45] but further staging with CT is usually necessary. Ultrasound may also demonstrate local spread into the adjacent liver.



A B

Figure 3.47 Gallbladder carcinoma. (A) TS, containing stones, debris and irregular wall thickening. (B) A different patient, demonstrating a grossly thickened hypoechoic wall with a contracted lumen.

Cholangiocarcinoma

This is a malignant lesion arising in the wall of the bile duct (Fig. 3.48). It is obviously easier to recognize from an ultrasound point of view when it occurs in and obstructs the common duct, as the subsequent dilatation outlines the proximal part of the tumour with bile. Cholangiocarcinoma may occur at any level along the biliary tree and is frequently multifocal.

A cholangiocarcinoma is referred to as a *Klatskin tumour* when it involves the confluence of the right and left hepatic ducts. These lesions are often difficult to detect on both ultrasound and CT. They are frequently isoechoic, and the only clue may be the proximal dilatation of the biliary ducts (Fig. 3.49).

Although rare, the incidence of cholangiocarcinoma seems to be increasing and it is strongly associated with PSC, a disease of the biliary ducts which predominantly affects young men (see above).

Multifocal cholangiocarcinoma may spread to the surrounding liver tissue and carries a very poor prognosis for long-term survival. In a liver whose texture is already altered by diffuse disease it may be almost impossible to identify these lesions before they become large. A pattern of dilated ducts distal to the lesion is a good clue (Figs 3.50 and 3.51).

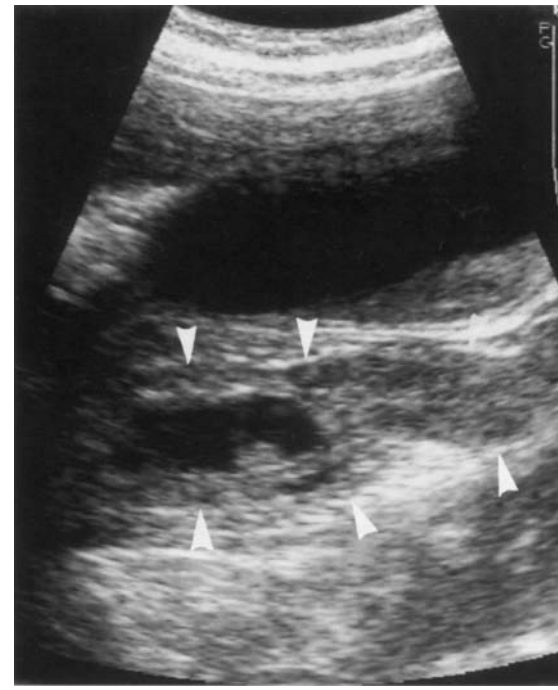
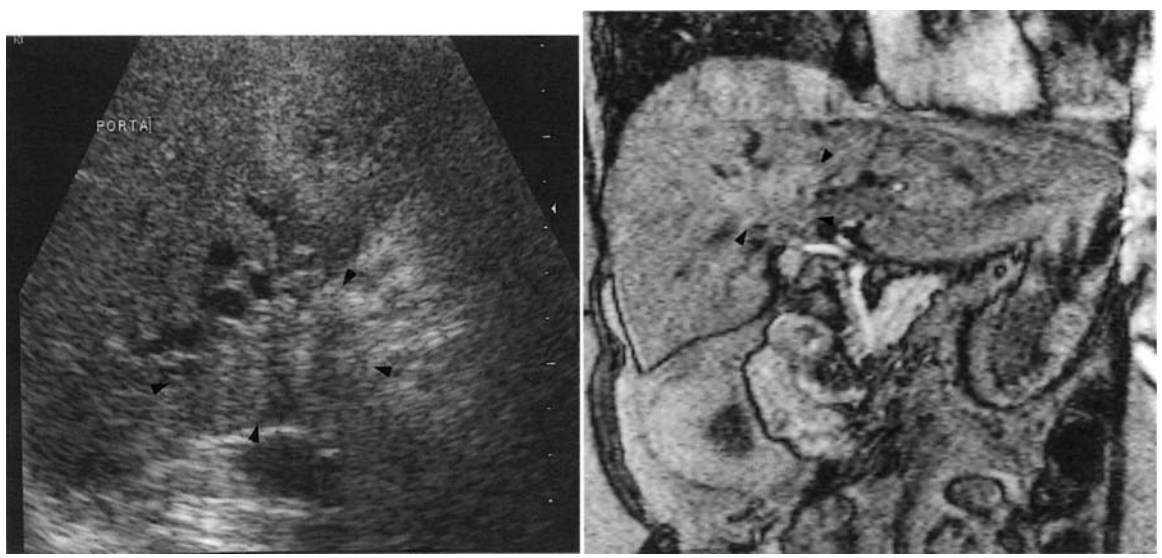


Figure 3.48 The distal CBD has a thickened wall (arrowheads), and the lumen is filled with tumour at the lower end. (Gallbladder anterior.)



A B

Figure 3.49 Cholangiocarcinoma. (A) Irregular mass at the porta, causing biliary obstruction—a Klatskin tumour. (B) MRI of the same patient, confirming the mass at the porta.

Management of the patient with cholangiocarcinoma

These patients have a poor prognosis, as the lesions usually present with jaundice due to invasion and obstruction of the duct. They spread to surrounding tissues, including the portal vein and lymph nodes, metastasize to the liver, and can be multifocal, particularly with PSC.

Staging of the disease is performed with CT or MRI. Endoscopic ultrasound can outline invasion into the biliary duct and laparoscopic ultrasound can pick up peritoneal or local spread.

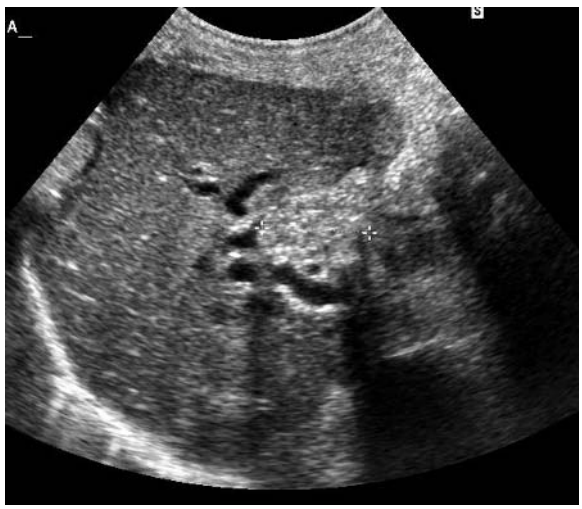


Figure 3.50 Focally dilated ducts distal to a hyperechoic cholangiocarcinoma (calipers).

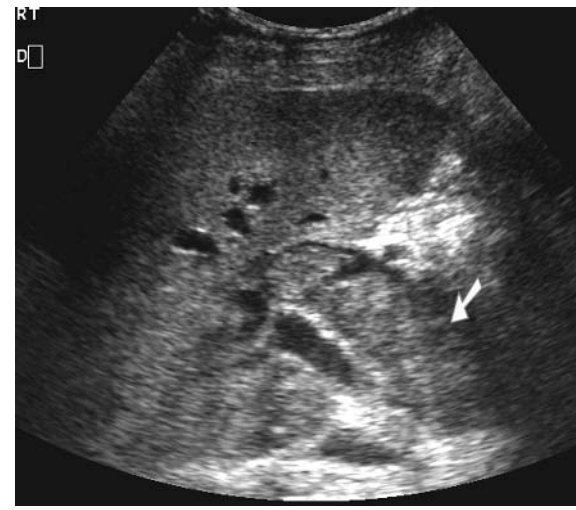
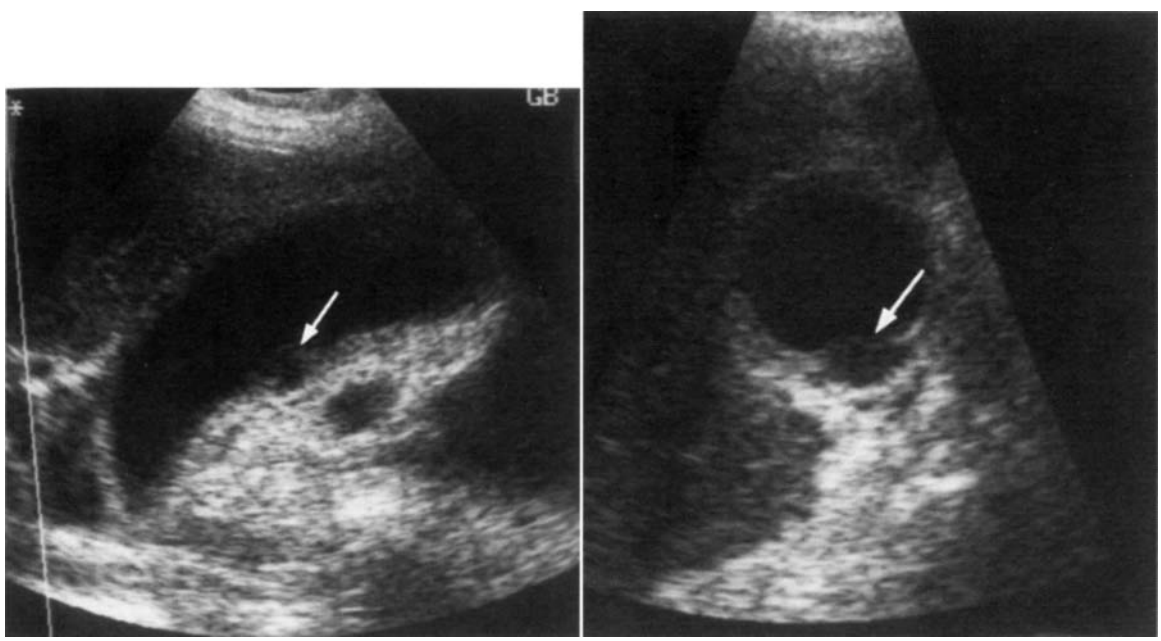


Figure 3.51 Cholangiocarcinoma invading the CBD (arrow).



A B

Figure 3.52 Metastases in the gallbladder wall (A) LS and (B) TS from advanced ovarian carcinoma.

Surgical resection of the tumour is becoming more successful in patients with single lesions.[46] Palliation is frequently the only feasible option and the insertion of a stent, either percutaneously or endoscopically, to bypass the obstructing lesion and assist drainage of the liver will relieve the symptoms and often allows the patient to return home for some months.

Other treatment options, such as chemotherapy, have limited success, although transplantation is increasingly regarded as an option in some cases. Despite improvements in treatment, only a minority of patients survive beyond twelve months after the initial diagnosis.

Gallbladder metastases

Metastases from other primaries may occasionally be deposited within the gallbladder wall (Fig. 3.52), usually as a late presentation of the disease process. Often, other metastatic deposits, for example in the liver and lymph nodes, may raise suspicion of gallbladder metastases in an irregularly thickened gallbladder wall.

The ultrasound appearances are of focal thickening and polyp-like lesions in the wall of the gallbladder. This may mimic primary gallbladder carcinoma but knowledge of a previously diagnosed primary, for example melanoma, lung or breast carcinoma, will point towards the diagnosis.

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Chapter 4

Pathology of the liver and portal venous system

CHAPTER CONTENTS

Ultrasound is often the first line of investigation for suspected liver pathology and the decision to proceed to secondary investigative procedures, such as further radiology or histology, is frequently determined by the findings of the initial ultrasound scan. Ultrasound is used in the diagnosis, staging and monitoring of liver disorders and also contributes to their treatment with ultrasound-guided invasive procedures.

Increasingly, ultrasound is also a reliable tool for more focused, complex examinations. Developing technology and techniques now result in improved diagnostic accuracy and are increasingly obviating the need for further radiology.

Intraoperative and laparoscopic ultrasound, using high-frequency, direct-contact techniques, set the standard for liver imaging in many cases.

BENIGN FOCAL LIVER LESIONS

Simple cysts

One of the most frequently seen liver lesions, the simple cyst, is either congenital (from abnormal development of a biliary radicle) or acquired (from trauma or previous infection). It is asymptomatic, unless large enough to cause a 'mass effect', compressing and displacing adjacent structures, and is

usually an incidental finding during the ultrasound scan. Frequently, small cysts are peripheral and therefore more likely to be missed on ultrasound than CT.

The simple cyst has three acoustic properties, which are pathognomonic (see Table 4.1); it is *anechoic*, has a *well-defined smooth capsule* and exhibits *posterior enhancement* (increased through-transmission of sound) (Fig. 4.1).

Although theoretically it is possible to confuse a simple cyst with a choledochal cyst (see Chapter 3), the latter's connection to the biliary tree is usually demonstrable on ultrasound. A radioisotope hepatic iminodiacetic acid (HIDA) scan will confirm the biliary connection if doubt exists.

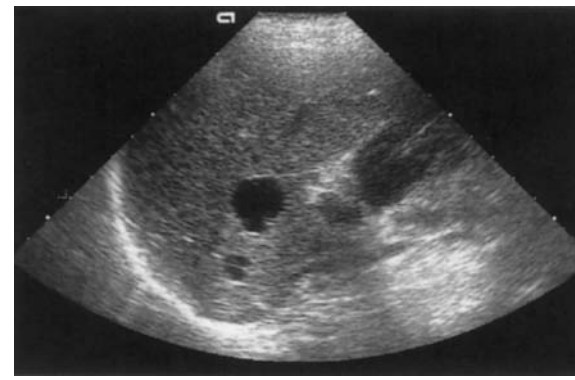


Figure 4.1 Typical simple liver cyst demonstrating a band of posterior enhancement. A smaller, bilocular cyst is seen behind it.

Complex cysts

Some cysts may contain a thin septum, which is not a significant finding. However, cysts which contain solid nodules or thickened walls should be viewed with suspicion (Fig. 4.2). Occasionally haemorrhage or infection may occur in a simple cyst, giving rise to low-level, fine echoes within it (Fig. 4.3).

These cysts are not usually actively treated; however the larger ones may be monitored with ultrasound, particularly if symptomatic. Percutaneous aspiration of larger cysts under ultrasound guidance may afford temporary decompression but is rarely performed as they invariably recur. Laparoscopic unroofing provides a more permanent solution to large, symptomatic cysts.[1]

Table 4.1 Cystic focal liver lesions—differential diagnoses

Simple cyst	
Anechoic, thin capsule, posterior enhancement (may contain thin septa)	Common finding, usually insignificant. Consider polycystic disease if multiple (Rarely an AV malformation may mimic a septated cyst—exclude by using colour Doppler)
Complex cyst	
Thin capsule + internal echoes	Haemorrhage or infection in a cyst Mucinous metastasis Cystadenoma
Capsule thickened or complex, may also contain echoes	Hydatid cyst Cystadenocarcinoma Intrahepatic pancreatic pseudocyst (rare)
Solid/cystic lesion	
Irregular margin, internal echoes + debris/solid material	Abscess Haematoma Necrotic metastasis Cavernous haemangioma

AV = arteriovenous.

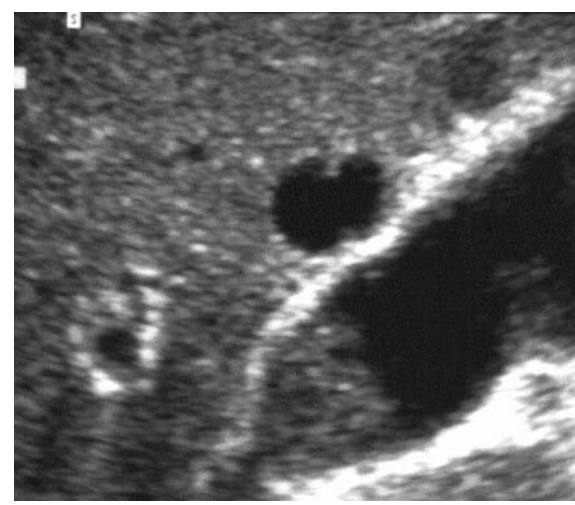


Figure 4.2 Small cyst adjacent to the gallbladder containing a nodule. This was a mucinous metastasis from an ovarian carcinoma.

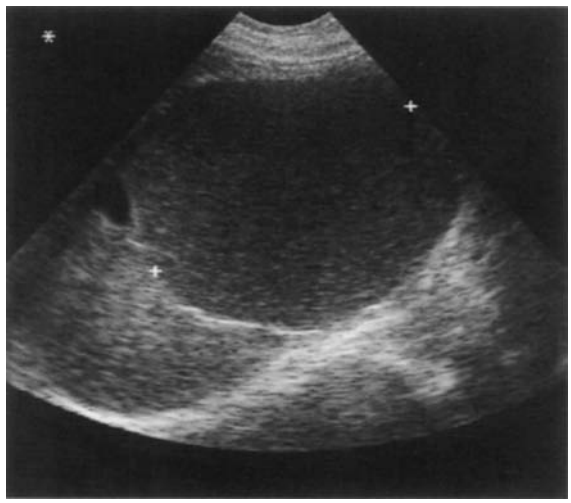


Figure 4.3 Large, infected hepatic cyst containing low-level echoes.

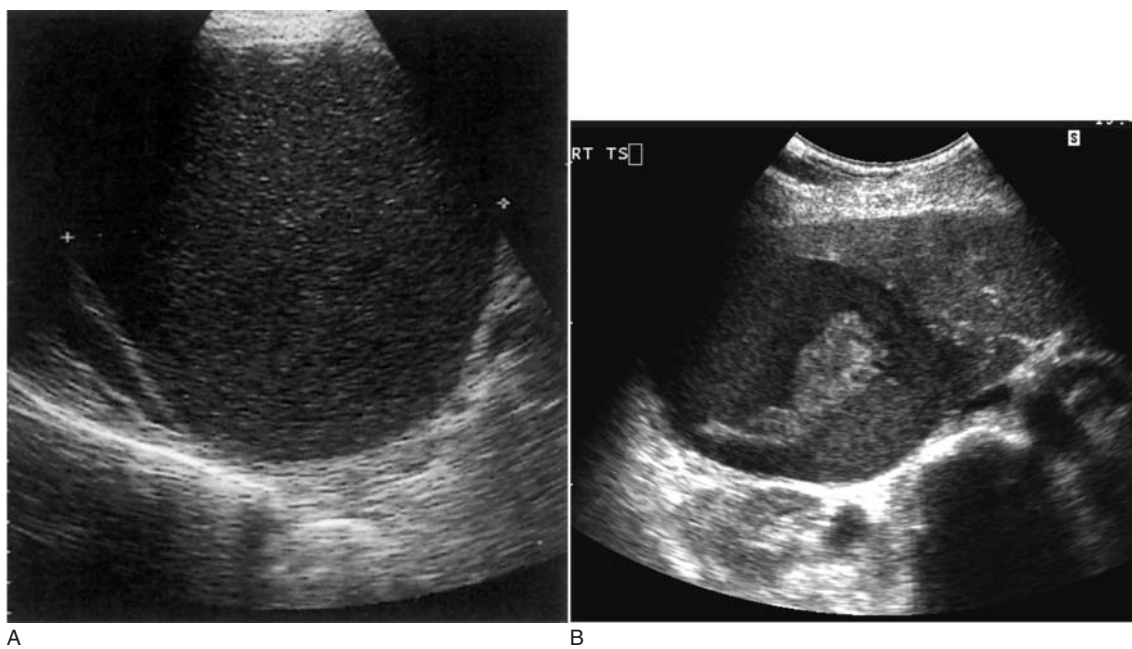
Another uncommon cause of a cystic lesion in the liver is a *cystadenoma*—a benign epithelial tumour. These have the potential to turn malignant, forming a *cystadenocarcinoma*. Close monitoring with ultrasound will demonstrate a gradual increase in size, changes in the appearances of the wall of the cyst, such as thickening or papillary projections, and internal echoes in some cases, which may arouse suspicion. A diagnostic aspiration may be performed under ultrasound guidance, and the fluid may contain elevated levels of carcinoembryonic antigen if malignant.[2] Cystadenomas are usually surgically removed due to their malignant potential (Fig. 4.4).

Rarely, cystic lesions in the liver may be due to other causes. These include pancreatic pseudocyst (within an interlobular fissure) in patients with acute pancreatitis or mucin-filled metastatic deposits in primary ovarian cancer.

An arteriovenous malformation, a rare finding in the liver, may look like a septated cystic lesion. Doppler, however, will demonstrate flow throughout the structure.

Polycystic liver

There is a fine dividing line between a liver which contains multiple simple cysts and polycystic liver



A B

Figure 4.4 (A) Large cystadenoma containing echoes and a septum. The cyst was large enough to cause obstructive jaundice—the patient's presenting symptom. The diagnosis was made by ultrasound-guided aspiration. This cyst had developed into a cystadenocarcinoma after 2 years. (B) A cystadenocarcinoma in a young woman presenting with altered liver function tests (LFTs). The cyst contains echoes and some solid material.

disease. The latter usually occurs with polycystic kidneys, a common autosomal dominant condition readily recognizable on ultrasound (see Chapter 7), but may rarely affect the liver alone (Fig. 4.5).

The appearances are of multiple, often septated cysts, of varying sizes throughout the liver. The cumulative enhancement behind the numerous cysts imparts a highly irregular echogenicity to the liver texture and may make it extremely difficult to pick up other focal lesions which may be present.

The polycystic liver is usually asymptomatic, but easily palpable, and if the kidneys are also affected the abdomen can look very distended. As with cysts in the kidneys, haemorrhage or infection in a cyst can cause localized pain. Treatment of the cysts by drainage is not successful and in rare cases hepatic transplant offers the only viable option in patients with intractable symptoms.

Hydatid (echinococcal) cyst

Hydatid disease comes from a parasite, *Echinococcus granulosus*, which is endemic in the Middle East but rare in the UK. The worm lives in the alimentary tract of infected dogs, which excrete the eggs. These may then be ingested by cattle or sheep and subsequently complete their life cycle in a human.

The parasite spreads via the bloodstream to the liver, where it lodges, causing an inflammatory reaction. The resulting cyst can be slow-growing and asymptomatic and may be single or multiple, depending on the degree of infestation.

Although the appearances are often similar to those of a simple cyst, the diagnosis can be made by looking carefully at the wall and contents; the hydatid cyst has two layers to its capsule, which may appear thickened, separated or detached on ultrasound. Daughter cysts may arise from the inner capsule—the honeycomb or cartwheel appearance (Fig. 4.6). Thirdly, a calcified rind around a cyst is usually associated with an old, inactive hydatid lesion.

The diagnosis of hydatid, as opposed to a simple cyst, is an important one as any attempted aspiration may spread the parasite further by seeding along the needle track if the operator is unaware of the diagnosis.

Management of hepatic hydatid cysts has traditionally been surgical resection. However, considerable success has now been achieved using percutaneous ultrasound-guided aspiration with sclerotherapy.[3]

Abscesses

Clinical features of an abscess

Patients present with fever, often accompanied by right upper quadrant (RUQ) pain and vomiting. Abnormal liver function tests (LFTs) and anaemia

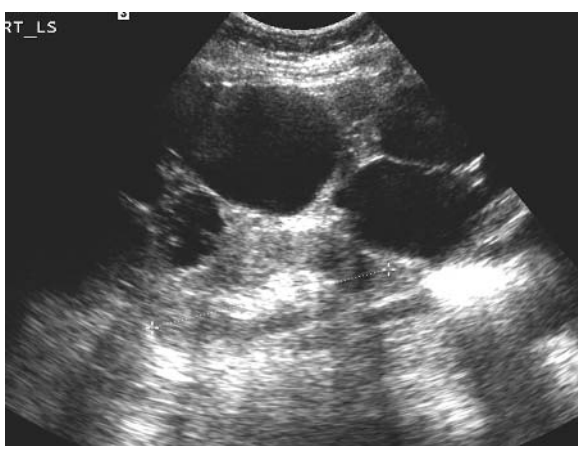


Figure 4.5 Multiple cysts in the liver. In this case the kidneys are normal. Polycystic liver is more usually associated with polycystic kidney disease.

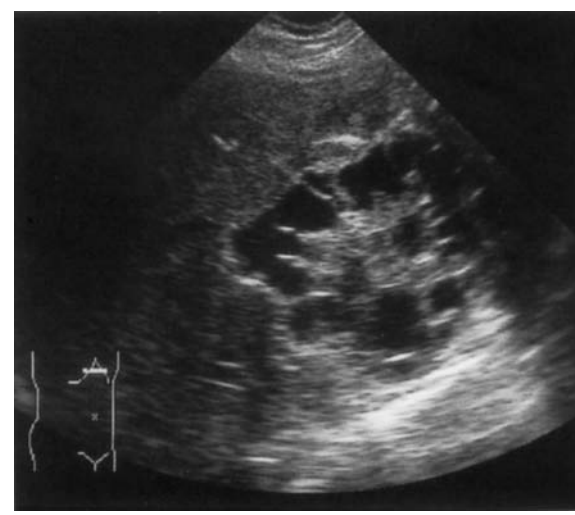


Figure 4.6 Hydatid cyst demonstrating surrounding daughter cysts.

can also be present. The clinical history helps the sonographer to establish the nature of the focal lesion and aetiology of the abscess. Abscesses of any type may be solitary or multiple.

Because the ultrasound appearances of abscesses can be similar to those of necrotic tumours or haematoma, the clinical picture is of particular importance to the sonographer.

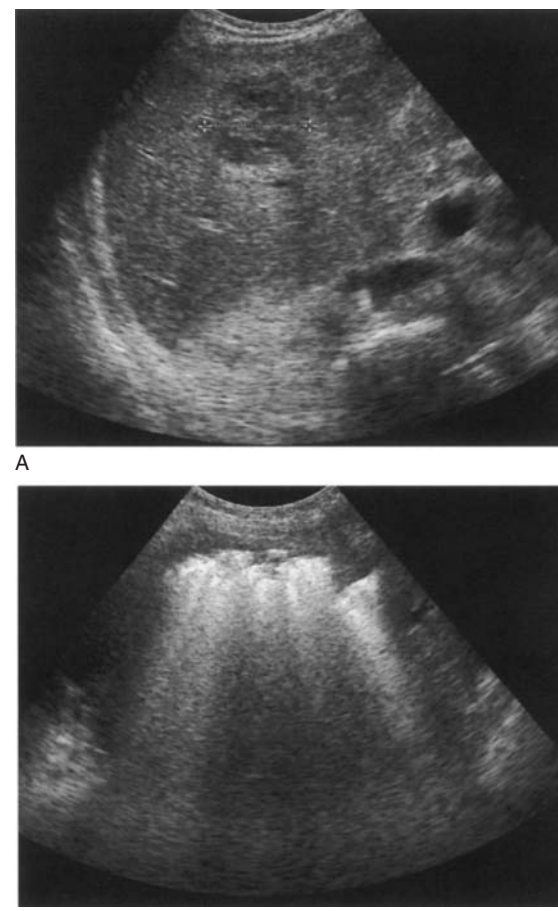
Ultrasound appearances

Hepatic abscesses may display a variety of acoustic features. Their internal appearances vary considerably. In the very early stages there is a zone of infected, oedematous liver tissue which appears on ultrasound as a hypoechoic, solid focal lesion. As the infection develops, the liver tissue becomes necrotic and liquefaction takes place. The abscess may still appear full of homogeneous echoes from pus and can be mistaken for a solid lesion, but as it progresses, the fluid content may become apparent, usually with considerable debris within it. Because they are fluid-filled, abscesses demonstrate posterior enhancement (Fig. 4.7A). The margins of an abscess are irregular and often ill-defined and frequently thickened. The inflammatory capsule of the abscess may demonstrate vascularity on colour or power Doppler but this is not invariable and depends on equipment sensitivity and size of the lesion.

Infection with gas-forming organisms may account for the presence of gas within some liver abscesses (Fig. 4.7B).

There are three main types of abscess:

- *Pyogenic abscess.* These form as a result of infection entering the liver through the portal venous system. Most commonly, appendiceal or diverticular abscesses are responsible, but intrahepatic abscesses are also seen in immunosuppressed patients and following postoperative infection. They are frequently multiple, and the patient must be closely monitored after diagnosis to prevent rapid spread. Pyogenic abscess is still considered a lethal condition, which has increased in recent years due to increasingly aggressive surgical approaches to many abdominal neoplasms.[4]
- *Amoebic abscess.* This is a parasitic infection which is rare in the UK, but found frequently in parts of Africa, India and the southern parts of the USA. Suspicion should be raised when the patient has visited these countries. It is usually contracted by drinking contaminated water and infects the colon, ulcerating the wall and subsequently being transported to the liver via the portal venous system.
- *Candidiasis abscess.* This is a fungal infection which may be seen in immunosuppressed patients. It is a rare cause of abscess formation and is usually blood-borne. The resulting



A

B

Figure 4.7 (A) Early stages of a pyogenic abscess in a transplanted liver. The lesion looks quite solid, but note the posterior enhancement. (B) The gas contained within this large abscess in the right lobe of the liver obscures the full extent of the lesion. (Large abscesses like this, which contain gas, may mimic the acoustic appearances of normal bowel.)

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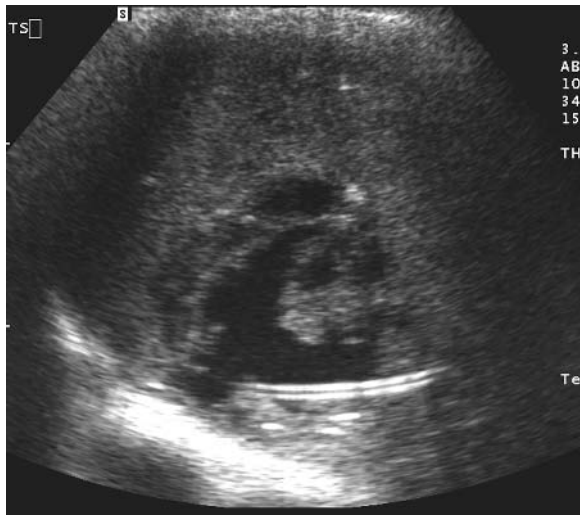


C

Figure 4.7 cont'd (C) A percutaneous drain is identified in a liver abscess.

abscesses are likely to be small but multiple on presentation. About 25% of infected patients form hepatic abscesses and the infection may spread to other sites in the abdomen.

Management of hepatic abscesses

An ultrasound-guided aspiration to obtain pus for culture is useful for identifying the responsible organism.

Aspiration combined with antibiotic therapy is usually highly successful for smaller abscesses and ultrasound is used to monitor the resolution of the abscesses in the liver.

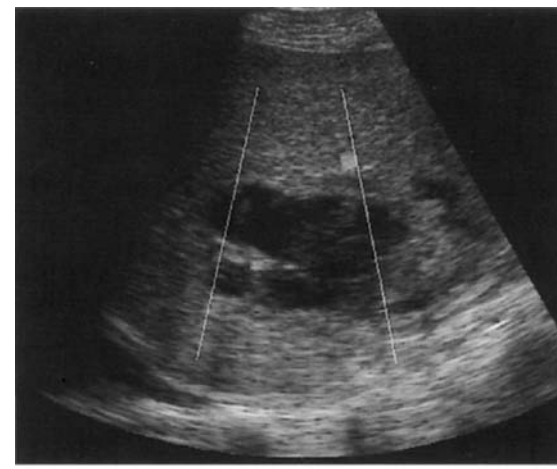
Ultrasound-guided drainage is used for large lesions, and surgical removal is rarely required.

Further radiology may be indicated to establish the underlying cause and extent, for example barium enema or CT, particularly if amoebic infection is suspected.

Haematoma

The liver haematoma may have similar acoustic appearances to those of an abscess, but does not share the same clinical features. A haematoma is the result of trauma (usually, therefore, via the Accident and Emergency department) but the trauma may also be iatrogenic, for example following a biopsy procedure (hence the value of using ultrasound guidance to avoid major vessels in the liver) or surgery (Fig. 4.8).

The acoustic appearances depend upon the timing—a fresh haematoma may appear liquid and echo-poor, but rapidly becomes more 'solid'-looking



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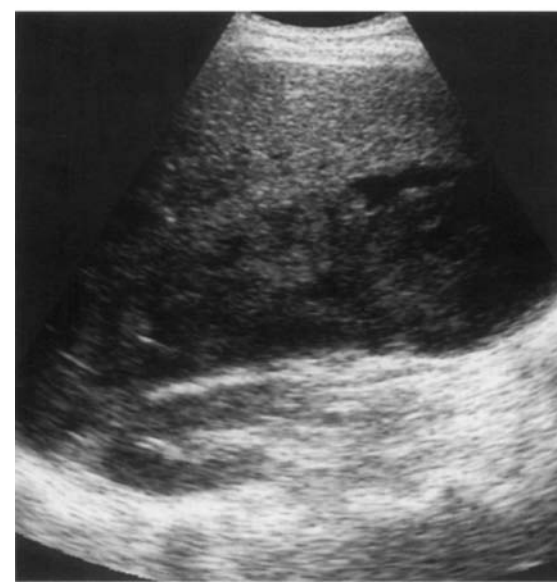


B

Figure 4.8 (A) Intrahepatic haematoma following a road traffic accident with rib fractures. The lesion is relatively fresh and contains some low-level echoes. (B) 2-day-old subcapsular haematoma. The collection became progressively smaller and hyperechoic as it resolved.

and hyperechoic, as the blood clots. As it resolves the haematoma liquefies and may contain fibrin strands. It will invariably demonstrate a band of posterior enhancement and has irregular, ill-defined walls in the early stages. Later on it may encapsulate, leaving a permanent cystic 'space' in the liver, and the capsule may calcify.

Injury to the more peripheral regions may cause a subcapsular haematoma which demonstrates the same acoustic properties. The haematoma outlines the surface of the liver and the capsule can be seen surrounding it. This may be the cause of a palpable 'enlarged' liver (Fig. 4.8B).

Intervention is rarely necessary and monitoring with ultrasound confirms eventual resolution. More serious hepatic ruptures, however, causing haemoperitoneum, usually require surgery.

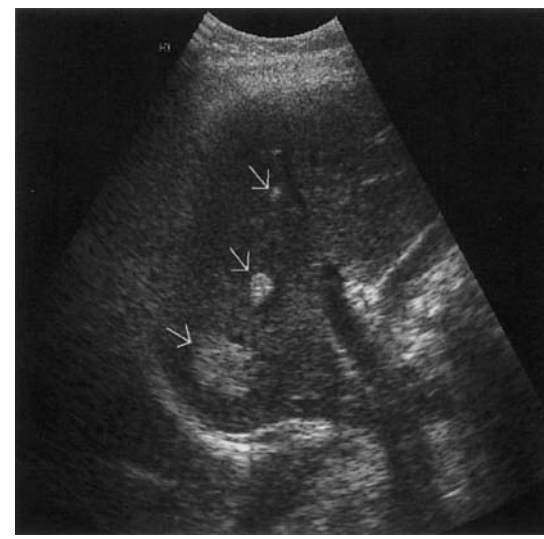
Haemangioma

These common, benign lesions are highly vascular, composed of a network of tiny blood vessels. They may be solitary or multiple. Most haemangiomas are small and found incidentally. They are rarely symptomatic but do cause diagnostic problems as they can be indistinguishable from liver metastases. Their acoustic appearances vary; the majority are hyperechoic, rounded well-defined lesions; however, atypical hypoechoic lesions or those with mixed echogenicity cause particular diagnostic dilemmas. Larger ones can demonstrate a spectrum of reflectivity depending on their composition and may demonstrate pools of blood and central areas of degeneration. They frequently exhibit slightly increased through-transmission, with posterior enhancement, particularly if large. This is probably due to the increased blood content compared with the surrounding liver parenchyma (Fig. 4.9).

Because the blood within the haemangioma is very slow-flowing, it is usually not possible to demonstrate flow with colour or power Doppler and the lesions appear avascular on ultrasound. Microbubble contrast agents demonstrate a peripheral, globular enhancement with gradual centripetal filling of the lesion, helping to characterize them and differentiate haemangioma from malignant lesions.

When found in children, haemangiomas tend to be large and do produce symptoms. These masses produce shunting of blood from the aorta via the main hepatic artery and, in extreme cases, present with resulting cardiac failure. They are often heterogeneous in appearance and larger vessels within them may be identified with Doppler. Although many regress over a period of time, others may have to be embolized with coils under radiological guidance to control the symptoms.

In patients with no cause to suspect malignancy, it may be suggested that the appearances of a small,



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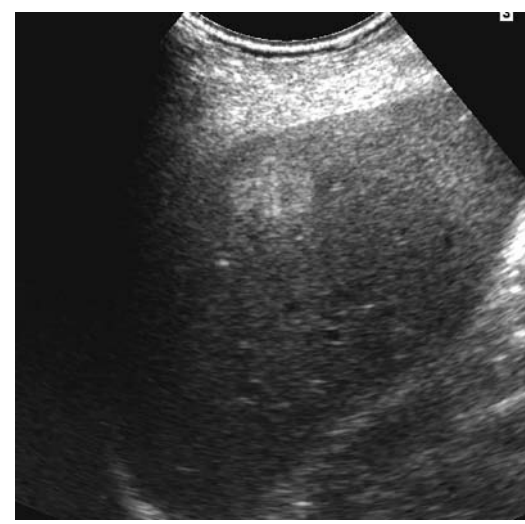
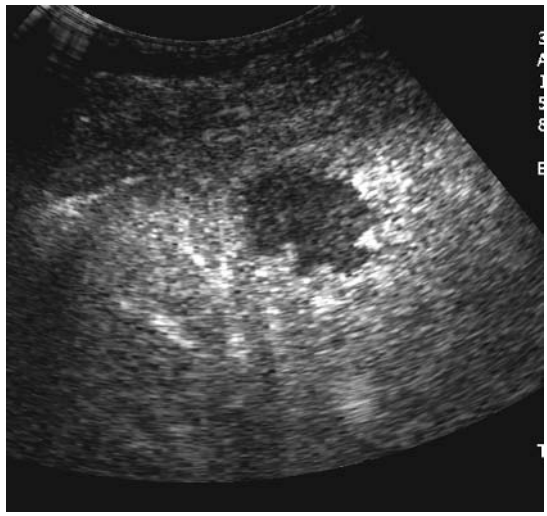


B

Figure 4.9 (A) Three small haemangiomas (arrows). (B) A haemangioma is demonstrated in the anterior part of the right lobe of the liver.

(Continued)



C

Figure 4.9 cont'd (C) On administration of microbubble contrast agent, the lesion in (B) demonstrates peripheral, globular enhancement, with gradual centripetal filling, consistent with haemangioma.

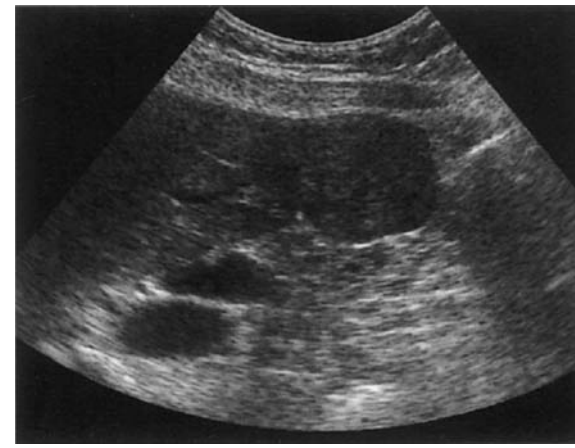
well-defined, hyperechoic mass are due to benign haemangioma. Follow-up scans will demonstrate no appreciable change over time. However, where doubt exists, it is useful to refer the patient for further imaging, such as MRI scanning, to characterize the lesion confidently.

Administration of an ultrasound contrast agent is also useful in lesion characterization and a haemangioma usually demonstrates a peripheral, nodular enhancement pattern in the arterial phase, with gradual centripetal filling (Fig. 4.9C).[5]

Adenoma

The hepatic adenoma is a benign focal lesion consisting of a cluster of atypical liver cells (Fig. 4.10). Within this, there may be pools of bile or focal areas of haemorrhage or necrosis. This gives rise to a heterogeneous, patchy echotexture. The smaller ones tend to be homogeneous with a smooth texture. They are usually less reflective than a haemangioma and may have similar reflectivity to the surrounding liver parenchyma.

Larger adenomas may contain vigorous arterial flow on Doppler, but this is not pathognomonic and does not differentiate it from a malignant lesion.



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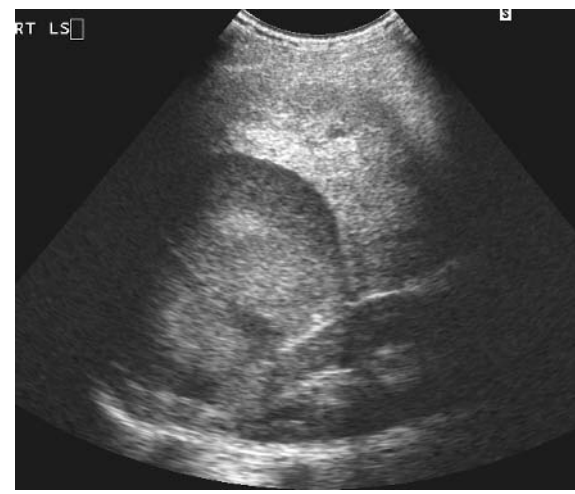


B

Figure 4.10 (A) Adenoma in segment 5 in a young woman on the oral contraceptive pill. (B) An unusual example of cystic degeneration in a large adenoma.

Clinical features

There is a particularly strong association between hepatic adenoma and use of the oral contraceptive so these masses tend to present in younger women. Adenomas are also associated with glycogen storage disease.

They may cause pain, particularly if they haemorrhage, and may be palpable. Surgical removal is the management of choice, although they occasionally regress if the oral contraceptive is discontinued.

Ultrasound is useful in monitoring patients with glycogen storage disease for changes in the charac-

teristics of their adenomas, as malignant degeneration is a possible feature.

Focal fatty change

Focal fatty infiltration

Fatty infiltration of the liver is a common occurrence which may affect the whole or part of the liver. It is associated with obesity and alcoholism, and can also occur in pregnancy, diabetes and with certain drugs.

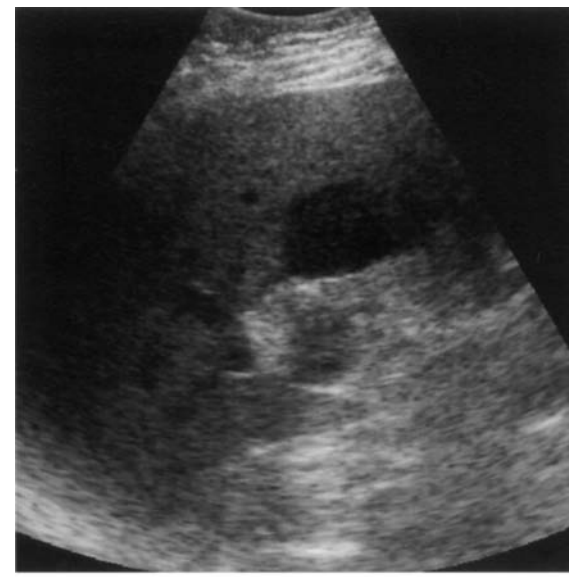
The deposition of fat confined to certain focal areas of the liver is related to the blood supply to that area. Fatty infiltration increases the reflectivity of the parenchyma, making it hyperechoic. This can simulate a focal mass, such as a metastasis. Unlike a focal lesion however, it does not display any mass effect and the course of related vessels remains constant. It has a characteristic straight-edged shape, rectangular or ovoid, corresponding to the region of local blood supply (Fig. 4.11).

Foci of fatty change may be multiple or may affect isolated liver segments. The most common sites are in segment 4 around the porta, in the caudate lobe (segment 1) and in the posterior area of the left lobe (segment 3).

Focal fatty sparing

The reverse process may also occur, in which a diffusely fatty, hyperechogenic liver has an area which has been spared from fat deposition due to its blood supply. This area is less reflective than the surrounding liver and may mimic a hypoechoic neoplastic lesion, but as with focal fatty infiltration, it has regular outlines and shape and no mass effect. The most common sites for fatty sparing are similar to those for focal fatty infiltration; segment 4 just anterior to the portal vein (Fig. 4.11B), segment 1 (the caudate lobe) and frequently there are multiple areas throughout the liver.

Unlike a true focal lesion, fatty change does not exhibit a mass effect and normal, undisplaced vasculature can be demonstrated with colour Doppler in areas both of focal fatty infiltration and fatty sparing. The administration of a contrast agent may also help to clarify the nature of the 'mass', as the area under consideration will behave exactly the same as the surrounding, normal liver in its uptake of the agent.



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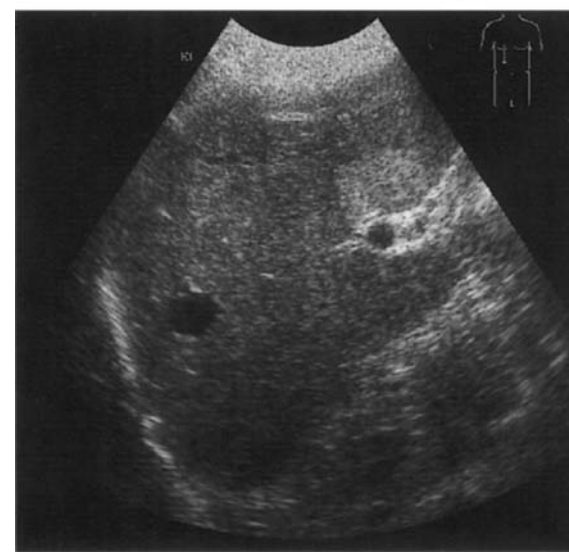
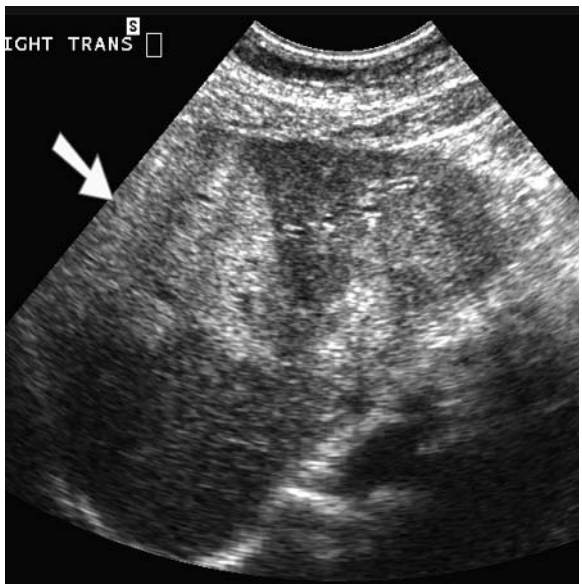


B

Figure 4.11 (A) Focal fatty sparing in the left lobe. This sharply demarcated area of normal liver contrasts with the surrounding hyperechoic fatty liver. (B) Focal fatty infiltration anterior to the main portal vein, characteristically 'square' in shape.

(Continued)



C

Figure 4.11 cont'd (C) Wedge-shaped area of fatty infiltration in the right lobe.

Lipoma

The hepatic lipoma is a relatively rare, benign hepatic tumour which is very similar in nature and acoustic appearance to focal fatty change. It differs in that it is a discrete tumour of fatty deposition rather than an infiltrative process and so can exert a mass effect on surrounding vessels if large. The fat content makes the lipoma hyperechoic compared to the surrounding liver tissue.

Focal nodular hyperplasia

This is a benign tumour made up of a proliferation of liver cells with hepatocytes, Kupffer cells and biliary and fibrous elements. It is most commonly found in young women and is usually discovered by chance, being asymptomatic. Its ultrasound characteristics vary, and it may be indistinguishable from hepatic adenoma.

It tends to affect the caudate lobe and has the appearance of a homogeneous mass often of similar echogenicity to the liver (Fig. 4.12). It presents a diagnostic difficulty both with CT and ultrasound, as its characteristics can vary.[6] Colour Doppler shows an increased arterial flow in the mass. The administration of an ultrasound contrast agent displays a characteristic 'spoked-wheel' pattern of arteries with a central scar.[7]

The diagnosis can usually be confirmed on MRI scanning (which shows a similar vascular pattern to that of ultrasound contrast scanning) but may occasionally require biopsy proof. Management of this benign mass is usually conservative, with ultrasound follow-up, once the diagnosis has been established, but surgical resection may be necessary in larger lesions.

Granuloma

Granulomata are benign liver masses which are associated with chronic inflammatory liver diseases. They are particularly associated with primary biliary cirrhosis, sarcoidosis or TB. They may be multiple and small, in which case the liver often looks coarse and hyperechoic. More often they are small discrete lesions which may be hypo- or isoechoic, sometimes with a hypoechoic rim like a target, or calcified with distal shadowing (Fig. 4.13). They can undergo central necrosis.

Differential diagnoses include metastases or regenerating nodules.

Hepatic calcification

Calcification occurs in the liver as a result of some pathological processes and may be seen following infection or parasitic infestation. It may be focal (usually the end stage of a previous abscess, haematoma or granuloma) which usually indicates that the lesion in question is no longer active. It may also be seen within some metastases.

Calcification may also be linear in nature, following the course of the portal tracts. This can be associated with old TB or other previous parasitic infestations.

Occasionally hepatic calcification is seen in children or in the fetus. This is usually not a significant finding but prenatal infection should be excluded with a TORCH (toxoplasmosis, rubella, cytomegalovirus and HIV) screen. Calcification, which casts a strong and definite shadow, should be distinguished from air in the biliary tree (Fig. 3.46), which casts a reverberative shadow and is

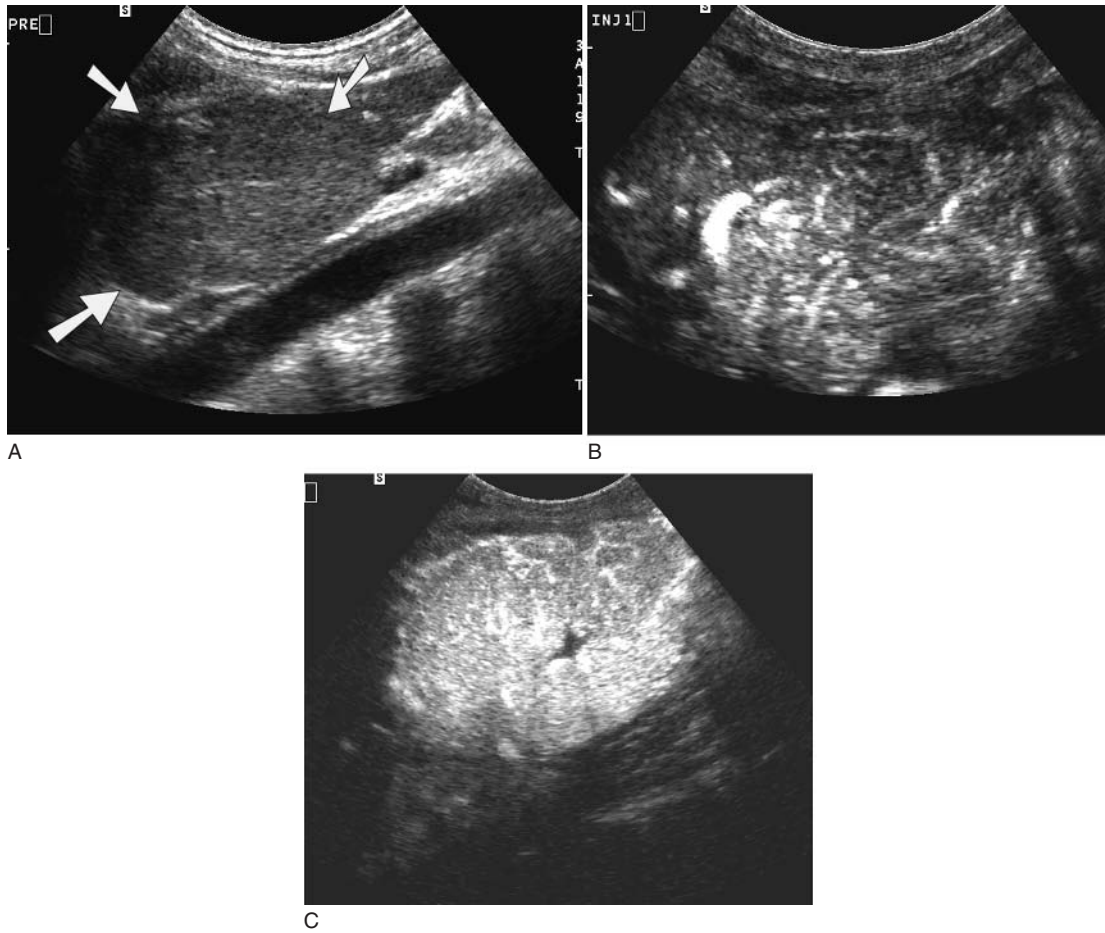


Figure 4.12 (A) Focal nodular hyperplasia in the left lobe (arrows), which is isoechoic with normal liver tissue. (B) Following administration of microbubble contrast agent, the FNH displays a 'spoked-wheel' pattern of vascular enhancement during the early arterial phase. (C) The same lesion seconds later, showing a central scar.

usually associated with previous biliary interventions, such as ERCP, sphincterotomy or stent placement (Fig. 4.14).

MALIGNANT FOCAL LIVER LESIONS

The 'mass effect'

This term describes the effect of a focal mass, whether benign or malignant, on surrounding structures and is a useful diagnostic tool. It implies the lesion's displacing or invasive nature, i.e. the displacement of vessels and/or invasion or distortion of adjacent structures and tissues as a result of the increasing bulk of a lesion. This effect differentiates a true mass from an infiltrative process such as steatosis, or an artefact.

Masses which are large and/or closely adjacent to a vessel demonstrate the effect more readily. The mass effect does not, of course, differentiate benign from malignant masses, or help in any way to characterize the mass. It is particularly useful when the mass is isoechoic compared with normal liver (Fig. 4.15). In such cases, the effect of the mass on adjacent structures may be the main clue to its presence.

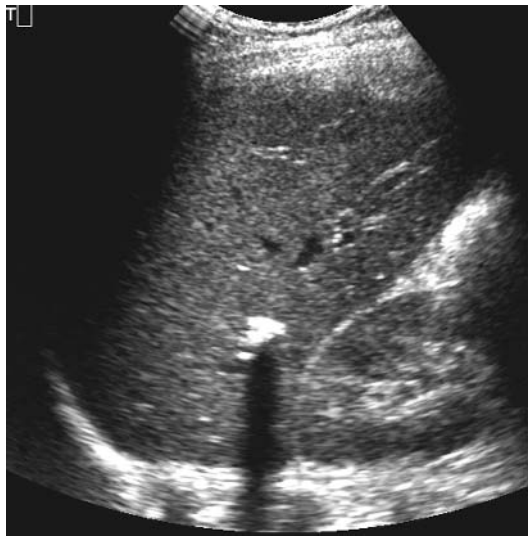


Figure 4.13 A calcified granuloma demonstrates acoustic shadowing.

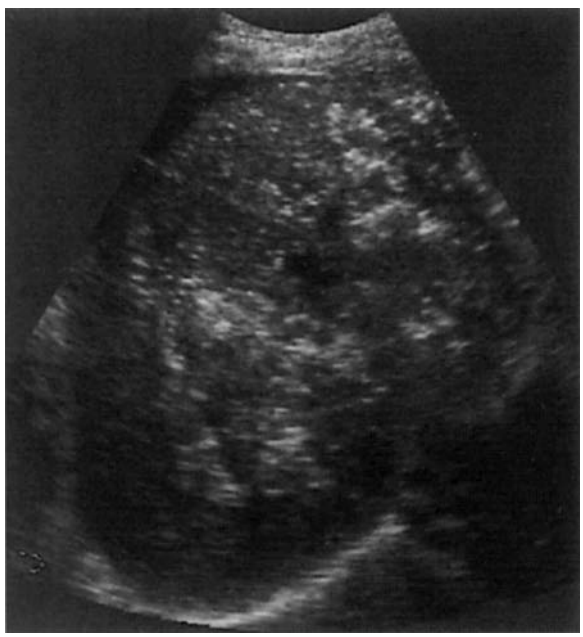


Figure 4.14 Considerable deposits of calcification are seen in the liver in this patient with nephrotic syndrome.

Metastases

The liver is one of the most common sites to which malignant tumours metastasize. Secondary deposits are usually blood-borne, spreading to the liver via the portal venous system (for example in the case of gastrointestinal malignancies), or hepatic artery (for example lung or breast primaries), or spread via the lymphatic system. Some spread along the peritoneal surfaces, for example ovarian carcinoma. This demonstrates an initial invasion of the subserosal surfaces of the liver (Fig. 4.16A), as opposed to the more central distribution seen with a haematogenous spread (Fig. 4.16B). The former, peripheral pattern is more easily missed on ultrasound because small deposits are often obscured by near-field artefact or rib shadows. It is therefore advisable for the operator to be aware of the possible pattern of spread when searching for liver metastases.

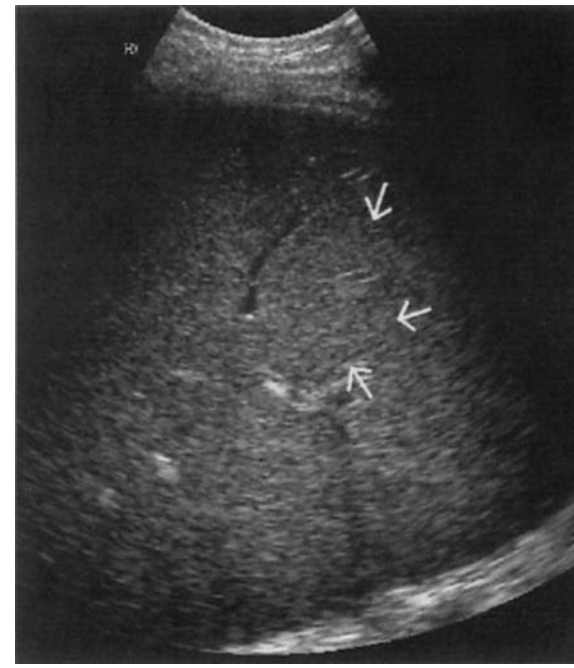


Figure 4.15 The mass effect: an isoechoic lesion (arrows), confirmed on CT, is recognized because of the adjacent deviation of the portal and hepatic venous radicles.

Ultrasound appearances

The acoustic appearances of liver secondaries are extremely variable (Fig. 4.16). When compared with normal surrounding liver parenchyma, metastases may be hyperechoic, hypoechoic, isoechoic or of mixed pattern. Sadly, it is not possible to char-

acterize the primary source by the acoustic properties of the metastases.

Metastases tend to be solid with ill-defined margins. Some metastases, particularly the larger ones, contain fluid as a result of central necrosis (Fig. 4.16E), or because they contain mucin, for example from some ovarian primaries. Occasionally, calcification is seen within a deposit, causing distal acoustic shadowing, and this may also develop following treatment with chemotherapy.

In some diseases, for example lymphoma, the metastases may be multiple but tiny, not immediately obvious to the operator as discrete focal lesions but as a coarse-textured liver (Fig. 4.16F). This type of appearance is non-specific and could be associated with a number of conditions, both benign and malignant.

Diagnosis of focal liver lesions, such as metastases, is made more difficult when the liver texture is diffusely abnormal or when there are dilated intrahepatic ducts because the altered transmission of sound through the liver masks small lesions. Other possible ultrasound features associated with metastases include a lobulated outline to the liver, hepatomegaly and ascites.

If the finding of liver metastases is unexpected, or the primary has not been identified, it is useful to complete a full examination to search for a

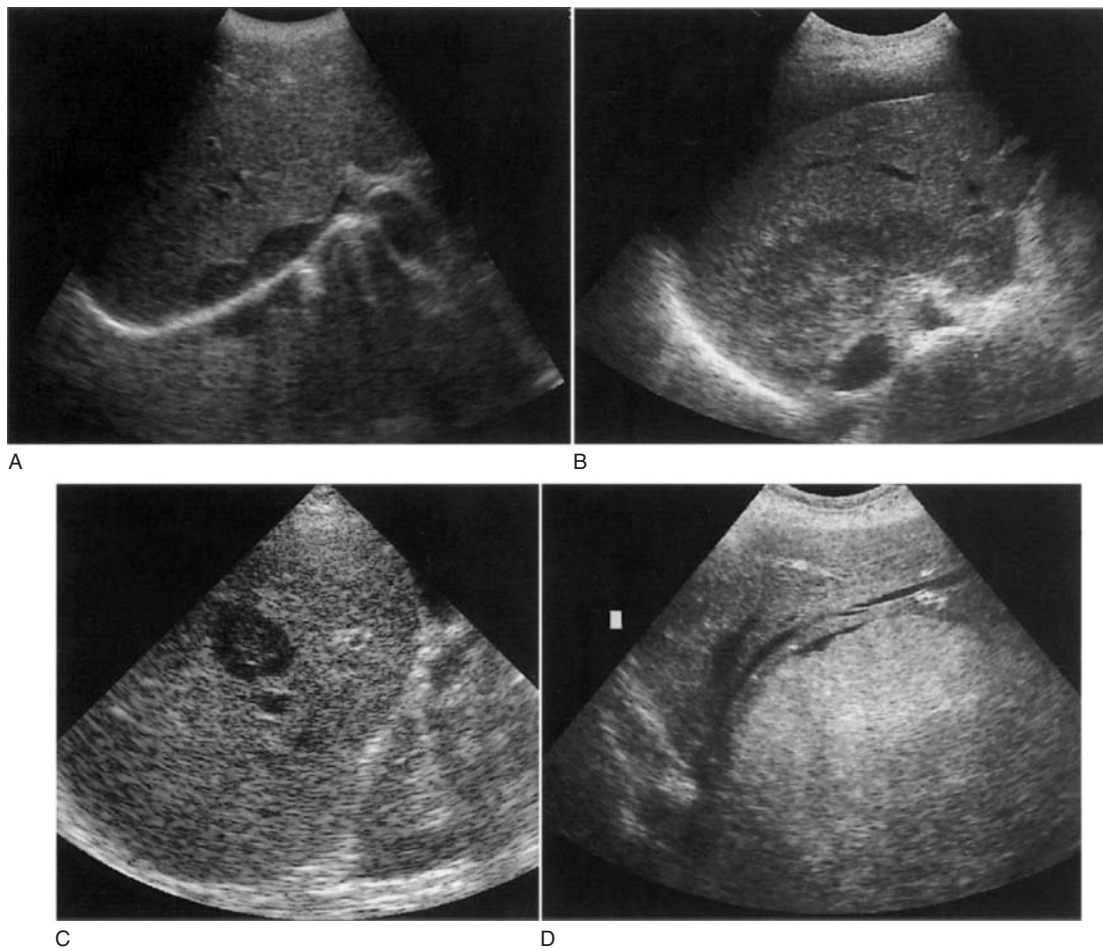


Figure 4.16 Examples of liver metastases. (A) Peripheral secondary deposits due to peritoneal spread from a primary ovarian carcinoma. (B) Blood-borne metastases from bowel carcinoma are demonstrated in the central area of the liver around the porta. (C) Solitary 'target' metastasis. (D) Large hyperechoic metastasis occupying most of the right lobe and causing an obvious mass effect.

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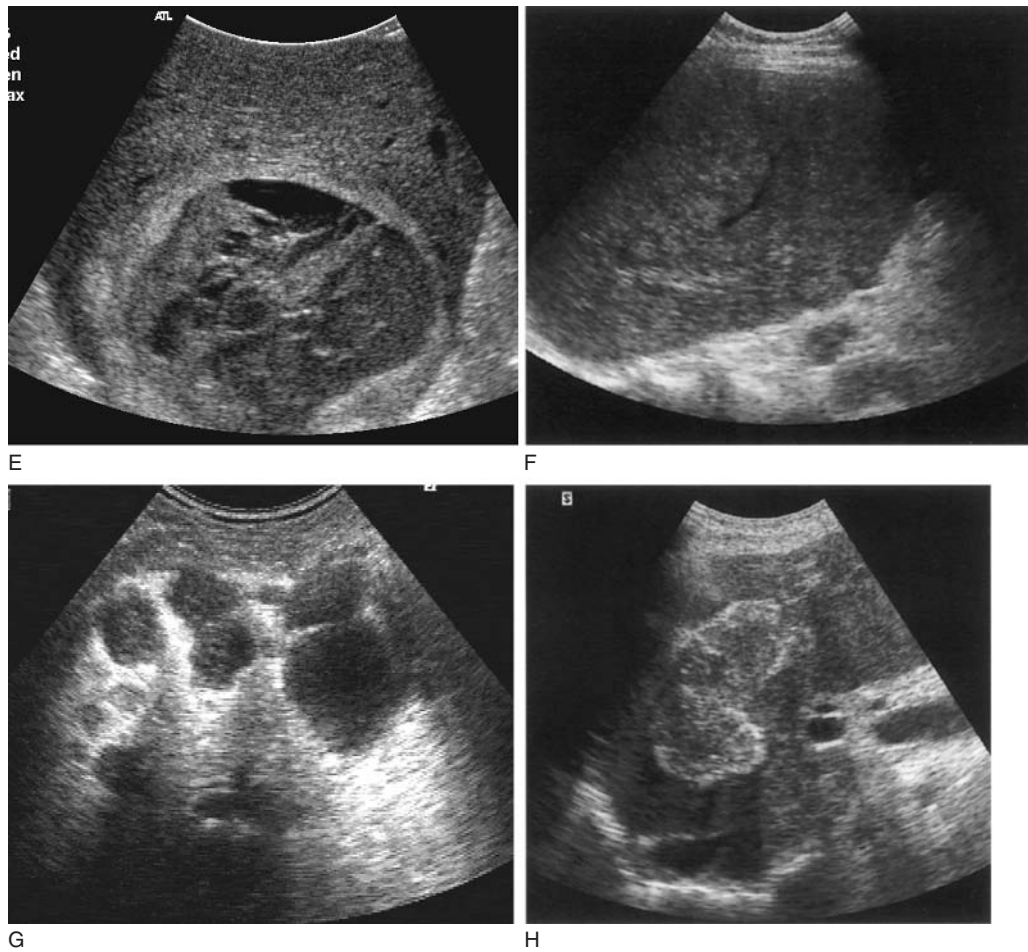

Figure 4.16 cont'd (E) Large necrotic metastasis. (F) Miliary metastases affecting the entire liver. Some larger, focal lesions are also visible. Note the hepatic enlargement and the lobulated outline of the liver. (G) Following administration of microbubble contrast agent, numerous metastases are discovered. These appear hypoechoic in the late portal venous phase, with no contrast uptake. (H) Calcified metastases from breast carcinoma.

possible primary carcinoma and to identify other sites of carcinomatous spread. Lymphadenopathy (particularly in the para-aortic, paracaval and portal regions) may be demonstrated on ultrasound, as well as invasion of adjacent blood vessels and disease in other extrahepatic sites including spleen, kidneys, omentum and peritoneum.

Doppler is unhelpful in diagnosing liver metastases, most of which appear poorly vascular or avascular. With the larger deposits, small vessels may be identified most often at the periphery of the mass.

The use of microbubble contrast agents has been shown to improve both the characterization and detection of metastatic deposits on ultrasound.[8] The injection of a bolus of contrast agent when viewed using pulse-inversion demonstrates variable vascular phase enhancement with no contrast uptake in the late phase (Fig. 4.16G).

Clinical features and management of liver metastases

Many patients present with symptoms from their liver deposits rather than the primary carcinoma. The demonstration of liver metastases on ultrasound may often prompt further radiological inves-

tigations for the primary. The symptoms of liver deposits may include non-obstructive jaundice, obstructive jaundice (which may occur if a large mass is present at the porta), hepatomegaly, right-sided pain, increasing abdominal girth from ascites and altered LFTs.

Ultrasound-guided biopsy may be useful in diagnosing the primary and complements further imaging such as X-rays and contrast bowel studies.

Accurate staging of the disease is currently best performed with CT or MRI, which have greater sensitivity for identifying small, sub-centimetre liver metastases, peritoneal deposits and lymphadenopathy and which can demonstrate more accurately any adjacent spread of primary disease.

The prognosis for most patients with liver metastases is poor, particularly if multiple, and depends to a large extent on the origin of the primary carcinoma. A regime of surgical debulking (removal of the primary carcinoma, adjacent invaded viscera, lymphadenopathy, etc.) together with chemotherapy can slow down the progress of the disease.

In an increasing number of cases, particularly those with metastases from a colorectal primary, which are less aggressive and grow more slowly, long-term survival can be achieved by resecting both the primary bowel lesion and then the liver deposits. The smaller and fewer the liver deposits, the better the prognosis. The success of this treatment has meant that tumours previously considered inoperable are now potentially curable. In such cases it is particularly useful to localize the lesions using the segmental liver anatomy prior to surgery (see Chapter 2). Intraoperative ultrasound (IOUS) is then used to confirm the preoperative appearances and examine the tumour margins to plan the line of resection (Fig. 4.17).

Other methods of treatment include chemoembolization, and radiofrequency, microwave or laser ablation often under ultrasound guidance.[9] The success of these options depends upon the number and size of the lesions, and the nature of the primary. Currently, these methods are considered palliative, rather than curative, and are an option for patients who are unsuitable candidates for hepatic resection. (See Chapter 11.)

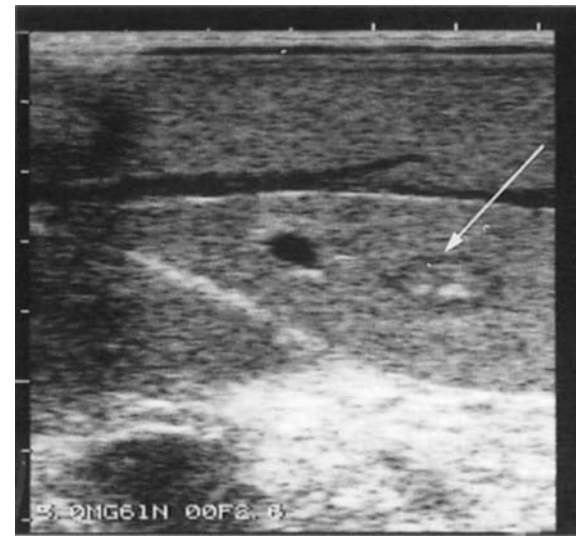

Figure 4.17 Intraoperative ultrasound scan demonstrates a small metastasis (arrow) in segment 4.

Ultrasound of other relevant areas

In suspected or confirmed malignancy, the examination of the abdomen may usefully include all the sites likely to be affected. While the liver is one of the most common sites for spread of the disease, it is also useful to examine the adrenals, spleen and kidneys, and to look for lymphadenopathy in the para-aortic, paracaval and portal regions.

If ascites is present, deposits may sometimes be demonstrated on the peritoneal or omental surfaces in patients with late-stage disease.

Hepatocellular carcinoma (HCC)

This primary carcinoma of the liver is more common in Africa and the Far East than in the UK. Most HCCs arise in diseased livers, hence the strong association with alcoholic cirrhosis and hepatitis, and one of the main reasons for ultrasound referral in these patients is to try to exclude focal liver lesions which could represent carcinoma. HCC is also associated with metabolic disorders and drug-related liver disease.

Clinically, small tumours are asymptomatic but cause a raised serum alpha-fetoprotein (AFP). The relationship between cirrhosis and HCC prompts screening of such patients with AFP and ultrasound.

The ultrasound appearances of HCC vary from hypo- to hyperechogenic or mixed echogenicity lesions (Fig. 4.18). It is often particularly difficult to locate small HCCs in a cirrhotic liver which is already coarse-textured and nodular. CT and MRI may be useful in these cases.[10,11]

These lesions may be solitary or multifocal. Colour and spectral Doppler can demonstrate vigorous flow, helping to distinguish HCCs from metastases or haemangiomas, which demonstrate little or no flow. All carcinomas demonstrate *neovascularization*: the formation of numerous new blood vessels to supply the growing lesion. The vascular characteristics of such new vessels are different from those of the normal, established vessels. The lesion usually demonstrates a knot of short, tortuous vessels with an irregular course. Because these new vessels have a paucity of smooth muscle in the intima and media, they exhibit a low resistance to blood flow, having relatively high end diastolic flow (EDF). They are able to multiply relatively quickly, causing arteriovenous shunting within the mass which may result in high velocities.

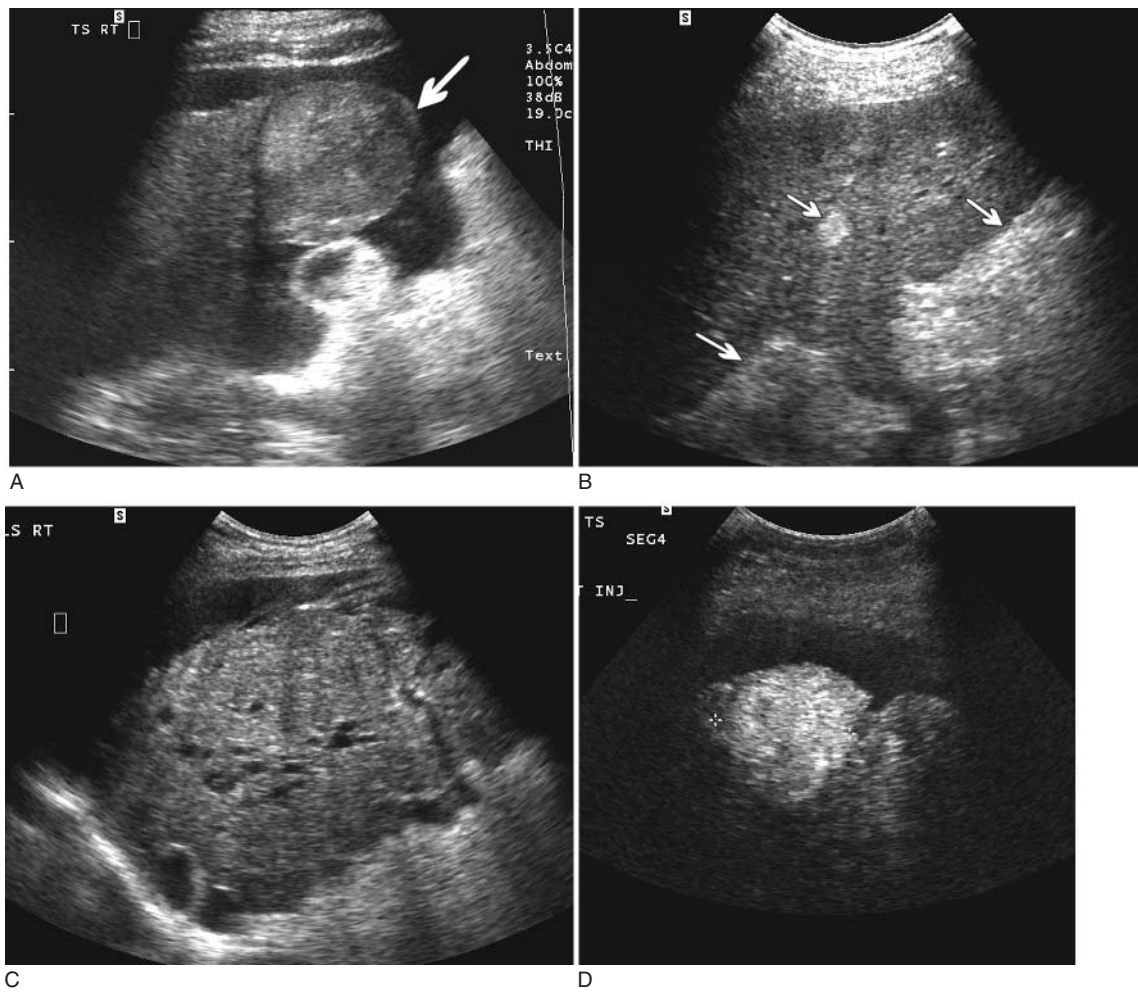

Figure 4.18 (A) Exophytic hepatocellular carcinoma (HCC) in a patient with cirrhosis. (B) Multifocal HCCs (arrows) in a cirrhotic patient. (C) A patient with chronic Budd–Chiari syndrome has a nodular liver with suspicion of a lesion near the anterior surface. (D) Administration of contrast in the same patient as (C) demonstrates increased uptake in the arterial phase, with wash-out of contrast in the late portal phase, helping to locate the lesion, and characterize it as an HCC.

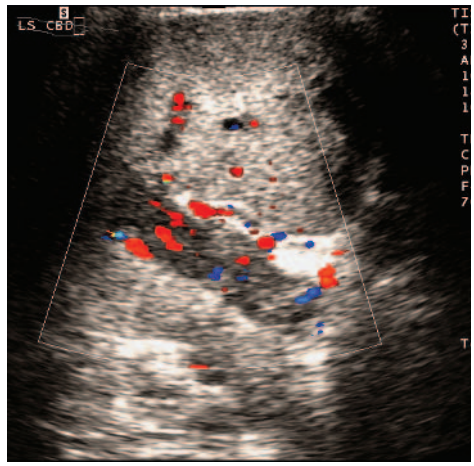

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Figure 4.18 cont'd (E) Tumour thrombus almost occluding the PV in a patient with multifocal HCC.

Increasingly, contrast ultrasound is used to detect and characterize HCCs in patients with a background of liver disease. HCCs tend to demonstrate an early enhancement of tortuous vessels, followed by a 'blush' of arterial enhancement compared to normal liver.

Cholangiocarcinoma

This primary carcinoma of the bile ducts is discussed more fully in Chapter 3. Most commonly seen affecting the main biliary ducts, it also occurs in the intrahepatic biliary tree where it infiltrates the surrounding liver parenchyma, having the appearance of a solid mass. It may be solitary or multifocal and a clue to its location is often the focal dilatation of ducts proximal to the obstructing mass.

For a summary of solid focal liver lesions, see Table 4.2.

Table 4.2 Common solid focal liver lesions: differential diagnoses

Lesion	Characteristics
Benign	
Haemangioma	Usually hyperechoic. Common incidental finding
Adenoma	Associated with oral contraceptive pill
Focal fatty change	No mass effect
Focal nodular hyperplasia	Uncommon, usually asymptomatic lesion, often found in young women
Granuloma	Associated with PBC or TB. May calcify
Regenerating nodules	Associated with cirrhosis. Multiple lesions
Abscess	May appear solid in the early stages. Look for posterior enhancement. Fever and pain
Infarct	Associated with HA thrombosis in liver transplant
Malignant	
Metastasis	Wide spectrum of possible acoustic appearances
Hepatocellular carcinoma	Associated with cirrhosis
Cholangiocarcinoma	Associated with PBC. Proximal biliary dilatation

PBC = primary biliary cirrhosis; TB = tuberculosis.

DIFFUSE LIVER CONDITIONS

Diseases which diffusely affect the liver may have very non-specific ultrasound appearances. Suspicion is usually raised following altered LFTs (see Chapter 1) and the diagnosis made histologically.

A number of diffuse liver conditions can cause hepatocellular (or non-obstructive) jaundice which is associated with increased levels of *unconjugated* bile in the blood. Many of these can be demonstrated with ultrasound, others cannot. The main role of ultrasound in the jaundiced patient is to exclude any obstructive cause (by the presence or absence of biliary duct dilatation) and to search for liver metastases or signs of a diffuse liver condition (Table 4.3).

Fatty infiltration (steatosis)

The process of accumulation of fat within the hepatic cells may be either focal (see above) or diffuse.

Related to various conditions such as alcoholism, obesity and diabetes, it is associated with any process which alters liver metabolism and it is reversible in many circumstances.

The acoustic properties of fat differ from those of normal liver tissue. The liver appears hyperechoic as the fat globules provide interfaces which are highly

Table 4.3 Causes of non-obstructive ('medical') jaundice

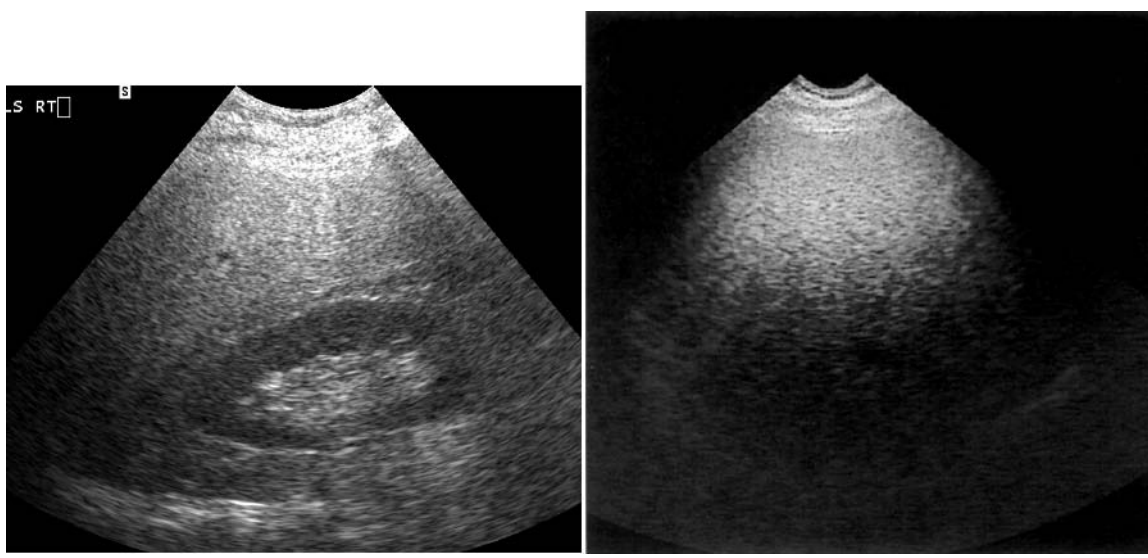
Condition	Aetiology
Haemolysis	In which red cells are destroyed, releasing the haemoglobin (from which bilirubin is derived) into the surrounding tissue
Haematoma	Haemolytic process
Gilbert's disease	A defect in the hepatic uptake of bilirubin
Viral hepatitis, cirrhosis of all types, alcoholic or drug-induced liver disease	Destruction of the liver cells by these diseases prevents the mechanism of hepatic uptake and excretion of bilirubin. Both conjugated and unconjugated bilirubin are present
Abscess, intrahepatic malignancy	Multiple and/or large lesions prevent the take-up and excretion of bilirubin by the liver cells

reflective. As the level of fat deposition increases, the level of echogenicity may reach that of the highly reflective portal tract walls. This has the effect of reducing the prominence of the portal tracts (Fig. 4.19) and making the liver appear smooth and homogeneous, with closely packed, fine echoes.

The contrast between the liver and parenchyma of the right kidney is therefore increased (a particularly useful sign confirming that the correct gain settings have been used). Hepatomegaly is also a feature, though not invariably.

Finally, the attenuation of fat is greater than that of normal liver tissue; this has the effect of reduced penetration in the far field, rather as if the time gain compensation (TGC) paddles or slope control had been incorrectly set. In severe cases of infiltration, most of the sound is reflected back to the transducer in the first few centimetres, creating a highly reflective near-field band through which the sound is unable to penetrate.

Fatty infiltration itself is not usually a significant finding; however it often occurs in conjunction with other significant diffuse processes such as cirrhosis. Its increased attenuation reduces the ability of ultrasound to exclude other disease or



A B

Figure 4.19 (A) Fatty infiltration increases the hepato-renal contrast. The portal tracts are reduced in prominence, giving a more homogeneous appearance. (B) Attenuation of the beam by fat prevents demonstration of far-field structures.

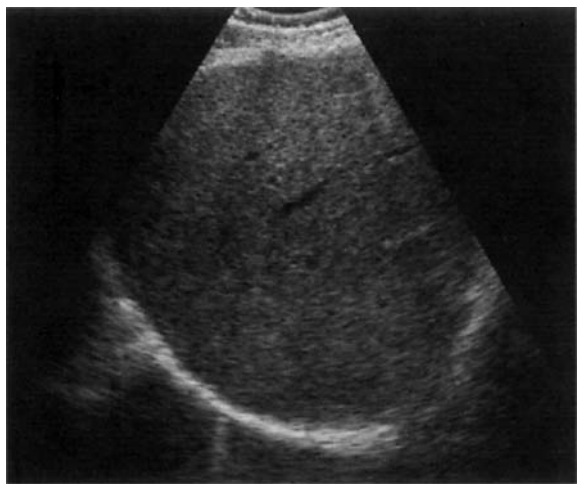
focal lesions and therefore CT is often a useful adjunct.

Cirrhosis

Cirrhosis is a process associated with end-stage chronic liver disease and is not really a disease in itself. It can result from a wide range of pathological processes including chronic hepatitis and alcoholic disease.

Ultrasound appearances of cirrhosis

In cirrhosis bands of fibrous tissue are laid down in the liver parenchyma between the hepatic lobules. This distorts and destroys the normal architecture of the liver, separating it into nodules. The process may be *micronodular*, which gives a generally coarse echotexture, or *macronodular* in which discrete nodules of 1 cm and above can be distinguished on ultrasound (Fig. 4.20).



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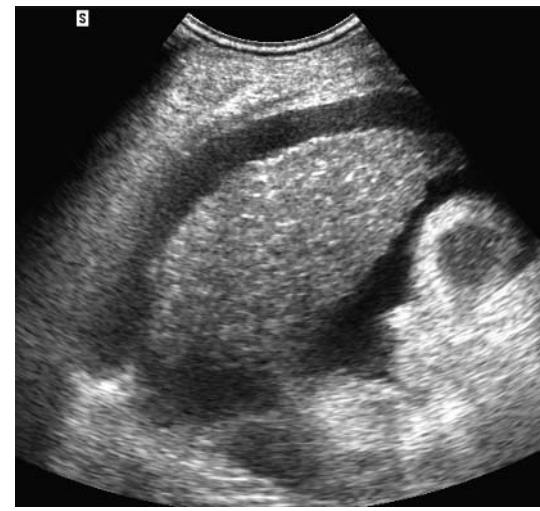

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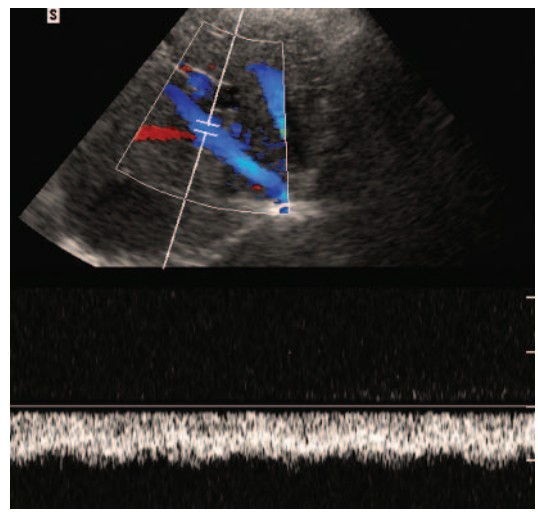

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Figure 4.20 (A) Micronodular cirrhosis in a patient with alcoholic liver disease. (B) Macronodular cirrhosis in a patient with primary biliary cirrhosis. Cirrhotic nodules are demonstrated throughout the peripheral hepatic substance with a lobulated liver outline. Ascites is also present. (C) Monophasic 'damped' flow in the hepatic veins in a patient with micronodular cirrhosis. This sign is not specific for cirrhosis and may be present under many other circumstances, including the presence of ascites.

The hepatocellular damage which causes cirrhosis gives rise to *hepatic fibrosis*, a precursor of cirrhosis. The fibrosis itself may have very little effect on the ultrasound appearances of the liver, but when advanced it is more highly reflective than normal liver tissue, giving the appearance of a 'bright' liver often with a coarse texture.[10] Unlike fatty change, which is potentially reversible, fibrosis is the result of irreversible damage to the liver cells. The picture is further complicated by the association of fibrosis with fatty change, which also increases the echogenicity. The acoustic attenuation properties of fibrosis, however, are similar to normal liver, so the ultrasound beam can penetrate to the posterior areas using normal TGC settings. Fat, on the other hand, increases both the echogenicity *and* the attenuation, preventing penetration to the far field (Fig. 4.19).

The cirrhotic liver tends to shrink as the disease progresses. However, it may be normal in size, or may undergo disproportionate changes within different lobes. In some patients the right lobe shrinks, giving rise to relative hypertrophy of the caudate and/or left lobes. This is likely to be due to the venous drainage of the different areas of the liver.

The rigid nature of the diseased liver also causes haemodynamic changes which can be demonstrated on spectral Doppler. The normally triphasic hepatic venous waveform can become flattened and monophasic (Fig. 4.20C). This is not necessarily specific to cirrhosis but is also associated with numerous types of chronic liver disease or any condition, either intra- or extrahepatic, which compresses the venous flow, such as polycystic liver disease or the presence of ascites.[12]

The portal venous flow may also be compromised due to portal hypertension (see below) and is associated with numerous changes on ultrasound showing reduced velocity, reversed flow, partial or total thrombosis.

A compensatory increase in hepatic arterial flow to the liver may also be seen as a result of portal venous compromise in portal hypertension.

Patients with cirrhosis are at increased risk of developing HCC, the detection of which is particularly difficult in an already nodular liver. Both CT and ultrasound have a low sensitivity for detecting small focal lesions in cirrhotic livers.[11] The use of Doppler, contrast CT and contrast MRI continues to improve the detection rate[13] of small lesions and many high-risk patients (i.e. those with cirrhosis) undergo regular ultrasound screening with tumour markers (AFP) as a precaution.[14] Small lesions continue to present a diagnostic challenge, and the use of ultrasound contrast agents, and the development of MRI using iron oxide, are likely to improve both detection and characterization of HCCs.[15]

Cirrhosis has numerous aetiologies:

Alcoholic cirrhosis The spectrum of alcoholic liver disease may take three forms: steatosis (alcoholic fatty liver), alcoholic hepatitis (often preceding cirrhosis) and finally cirrhosis. The later, chronic stages carry a worse prognosis, frequently associated with portal hypertension and an increased incidence of HCC (Fig. 4.18). Alcoholic liver disease may be halted or reversed in the early stages in patients who discontinue alcohol intake, with subsequent nodular regeneration of hepatic tissue (Fig. 4.20D). Nodular regeneration is not easy to distinguish from frank cirrhosis or other focal liver lesions, such as HCC, and the use of ultrasound contrast agents, or other imaging such as MRI may be required. Regenerating nodules may cause the liver to enlarge, whereas end-stage cirrhosis causes shrinkage of the liver.

Primary biliary cirrhosis (PBC) This is a progressive cholestatic liver disease of unknown aetiology which occurs predominantly in middle-aged females. The term 'cirrhosis' may be rather misleading for the early stages of this condition, which actually take the form of an inflammatory destruction of the intrahepatic bile ducts. These early stages of cholangitis are not, strictly speaking, cirrhotic. However as the destruction progresses, fibrotic bands form in a process of macronodular cirrhosis (Fig. 4.20B).

Treatment of PBC involves control of the associated symptoms of portal hypertension and pruritus, but its progression is inevitable. Liver transplantation now offers a successful therapeutic option for these patients.[16]

Although the liver frequently looks normal on ultrasound in the early stages of the disease, gallstones, splenomegaly and lymphadenopathy can be demonstrated in many patients.[17]

Secondary biliary cirrhosis This occurs as a result of long-standing biliary obstruction. Causes usually include benign strictures or chronic stone impaction in the common bile duct causing progressive, gradual obstruction over a period of time. This causes ascending cholangitis and jaundice. The bile ducts may appear only mildly dilated on ultra-

sound. It is also a recognized sequel of biliary atresia in children.

Other causes of cirrhosis Cirrhosis may be drug-induced, particularly in patients on long-term treatment or therapy.

It is also associated with many other diseases, such as hepatitis (see p. 106) diabetes, ulcerative colitis, rheumatoid arthritis or any long-term conditions, acquired or congenital, which can affect the liver.

Congenital forms of cirrhosis exist due to metabolic disorders: Wilson's disease (deposition of copper in the liver and kidneys), glycogen storage disease (inability to break down glycogen to glucose), haemochromatosis (deposition of iron in the liver and pancreas) and others.

Clinical features and management of cirrhosis

Clinical presentation depends upon the aetiology, and may involve either chronic symptoms or an acute episode.

Pruritus, fatigue and jaundice, with steatorrhoea and deranged LFTs (raised alkaline phosphatase and serum bilirubin in PBC, raised alanine aminotransferase [ALT] and aspartate aminotransferase [AST] in alcoholic disease) are generally present by the later stages. This is followed by the symptoms of portal hypertension (see below), which is a poor prognostic feature associated with late-stage cirrhosis.

The process may be reversed in alcoholics who stop drinking. However the prognosis of any cirrhotic condition is extremely poor if malignancy is present. In severe cases, the management revolves around trying to treat the symptoms of portal hypertension rather than the disease itself.

Liver transplant is now an established and highly successful treatment option for PBC when the symptoms can no longer be controlled with drugs. It is also an option for alcoholic cirrhosis, although there is currently a significant incidence of post-transplant return to alcoholism.

Portal hypertension

Portal hypertension occurs when the pressure in the portal venous system is raised. This may happen as a result of chronic liver disease, particularly in the cirrhotic stage, when the nodular and fibrosed nature of the parenchyma impedes the flow of blood into the liver. It is significant because it causes numerous deleterious effects on the patient, many of which can be recognized on ultrasound (Table 4.4).

Table 4.4 Summary of possible ultrasound appearances in cirrhosis

Normal parenchyma	May appear normal, particularly in the early stages
Changes in texture	Coarse texture (micronodular) Irregular nodular appearance (macronodular)
Changes in reflectivity	Fibrosis increases the overall echogenicity (but *not* the attenuation) May be accompanied by fatty change, which increases both echogenicity *and* attenuation giving a hyper-reflective near field with poor penetration to the posterior liver
Changes in size and outline	Small, shrunken liver Nodular, irregular surface outline Possible disproportionate hypertrophy of left or caudate lobes
Focal lesions	Increased incidence of HCC Regenerative nodules
Vascular	Signs of portal hypertension: —changes in portal vein direction and velocity —possible thrombosis —varices and collaterals —increased hepatic arterial flow —flattened, monophasic hepatic venous flow on spectral Doppler (a non-specific finding)
Other signs	Ascites Splenomegaly Lymphadenopathy

HCC = hepatocellular carcinoma.

Raised portal venous pressure is associated with several complications:

Portal vein signs Portal vein (PV) flow is influenced by numerous factors, including prandial state, patient position, exercise and cardiac output.[18] Its velocity varies considerably in both cirrhotic and healthy subjects, and it is essential to use colour and spectral Doppler to investigate the portal flow.[19]

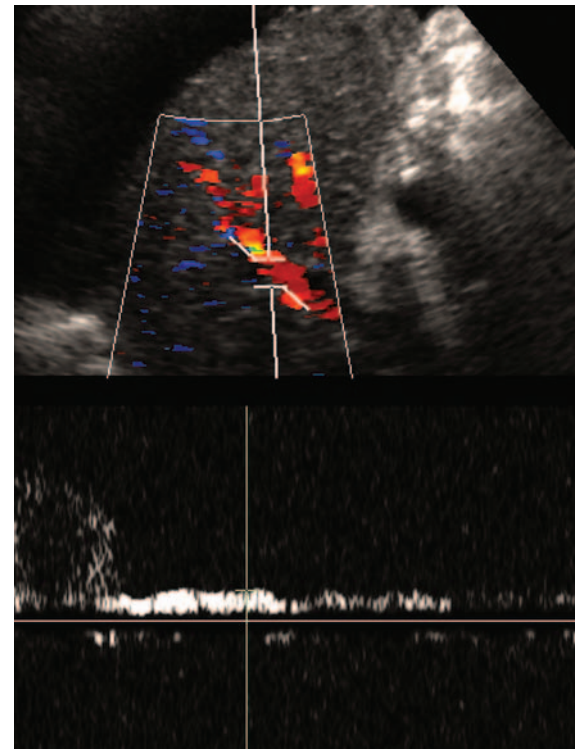
The vein may appear dilated and tortuous, but not invariably. (The normal portal vein diameter does not usually exceed 16 mm in a resting state; see Chapter 2).

Portal venous flow may be:

- *normal* in direction (hepatopetal) and velocity.[20]
- *reduced in velocity*[21] (Fig. 4.21A), < 10 cm/sec, although there is overlap with the normal range.
- *damped*, in which there is a lack of normal respiratory variation of both the calibre and the waveform of the splenic and portal veins. The normal spectrum has a 'wavy' characteristic, which may be lost.
- *reversed* (hepatofugal) (Fig. 4.21B). This indicates serious liver disease. Interestingly, patients with hepatofugal PV flow are much less likely to suffer from bleeding varices, suggesting a type of 'protective' mechanism here.
- *balanced*, in which both forward and reverse low velocity flow is present, a condition which may precede imminent thrombosis (Fig. 4.21C).
- *thrombosed* (Fig. 4.21D). Low-level echoes from the thrombus may be evident but with fresh thrombus the vein may appear anechoic, as in the normal vein. Although PV thrombosis most commonly results from portal hypertension in cirrhosis, there are many

Box 4.1 Causes of portal vein thrombosis

Chronic liver disease
—especially cirrhosis
Inflammatory
—pancreatitis
—acute cholecystitis
—necrotizing enterocolitis
Malignancy
—pancreatic tumour
—gastric tumour
Coagulation disorders
—may be associated with Budd–Chiari syndrome



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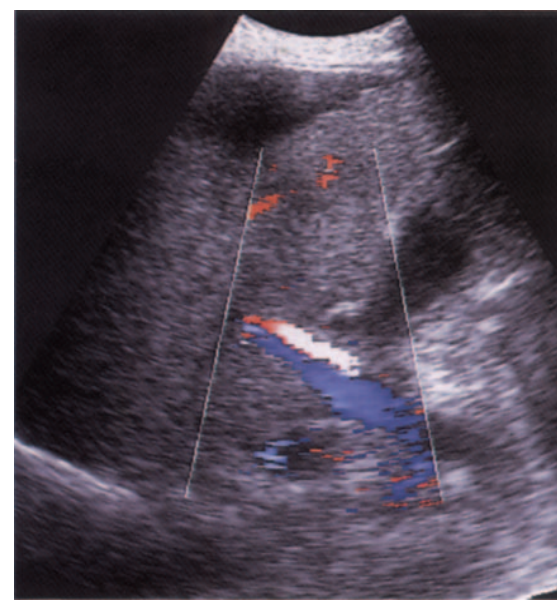
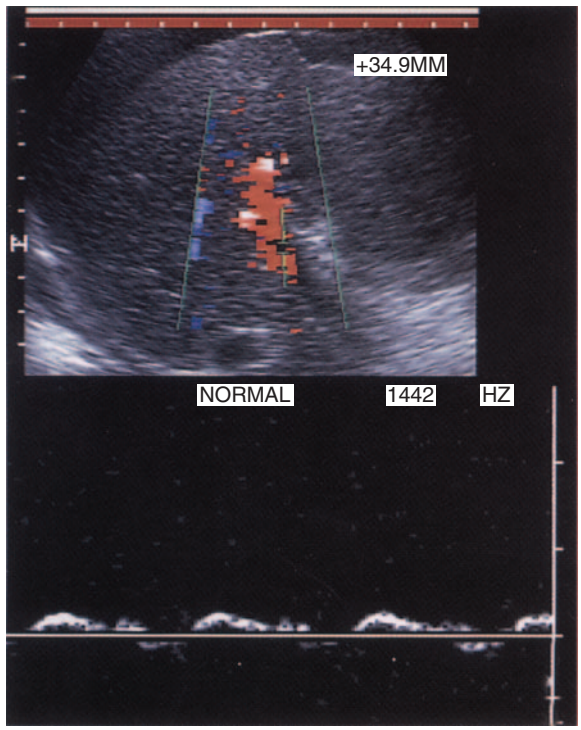


B

Figure 4.21 The MPV in portal hypertension. (A) Portal vein (PV) velocity is greatly reduced. (B) Reversed PV flow in portal hypertension. Note the increased velocity of hepatic arterial flow indicated by the light colour of red just anterior to the portal vein. The patient has macronodular cirrhosis with ascites. *(Continued)*



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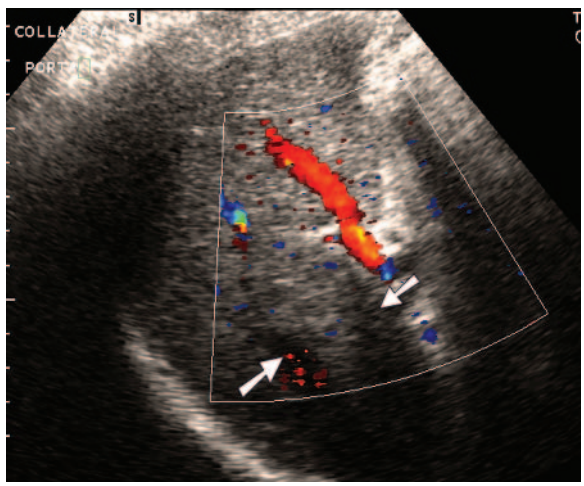

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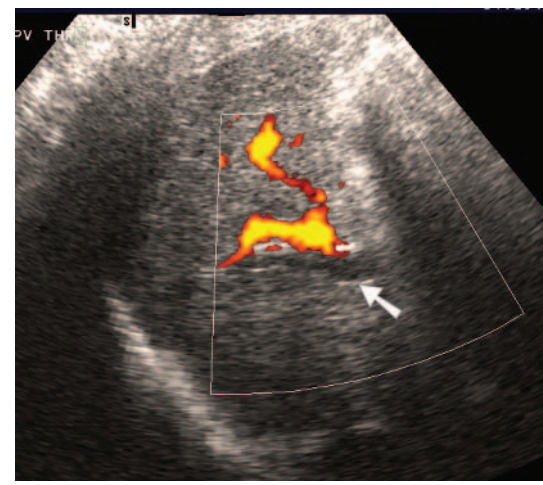

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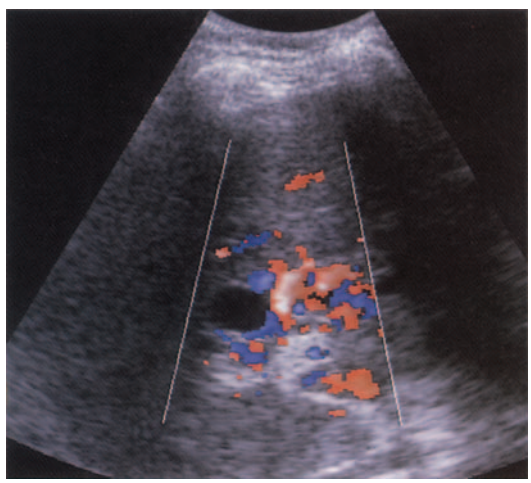
Figure 4.21 cont'd (C) Balanced PV flow. Alternate forward and reverse low-velocity flow on the Doppler spectrum. The PV colour Doppler alternates red and blue. (D) PV thrombosis. The PV is dilated (arrows) and filled with thrombus. A collateral vessel is seen anterior to this—not to be confused with the PV—as this is a source of false-negative ultrasound results. (E) Non-dilated, thrombosed PV (arrow) with collaterals demonstrated on power Doppler.

other causes, including inflammatory or malignant conditions which may surround, compress or invade the portal and/or splenic veins (Box 4.1). The thrombosis may be total or partial.

- *hepatopetal main PV flow with hepatofugal peripheral flow* may be a sign of HCC, requiring careful scanning to identify the lesion.
- *cavernous transformation*. A network of collateral vessels may form around a thrombosed main portal vein at the porta, especially if the thrombosis is due to extrahepatic causes (for example

pancreatitis) rather than diseased liver. The appearance of cavernous transformation of the PV is quite striking (Fig. 4.22A) and colour Doppler is particularly useful in its diagnosis.[22]

Make sure, before diagnosing PV thrombosis, that the vein axis is less than 60° to the transducer and that the Doppler sensitivity is set to pick up low-velocity flow. Ultrasound is known to have a false-positive rate for PV thrombosis but this is often due to inadequate technique or insensitive equipment. False-negative results, indicating that flow is present in a vein which is actually thrombosed, are due to the detection of flow within a collateral vessel at the porta, which can be mistaken for the main PV.



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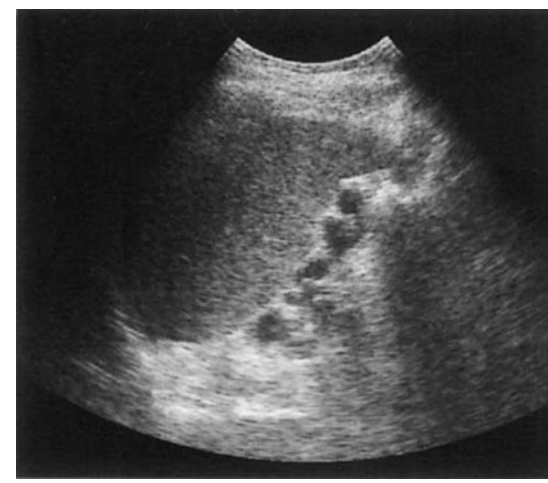

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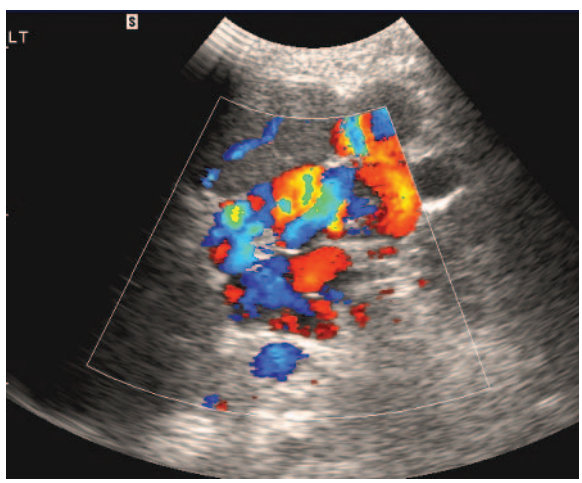

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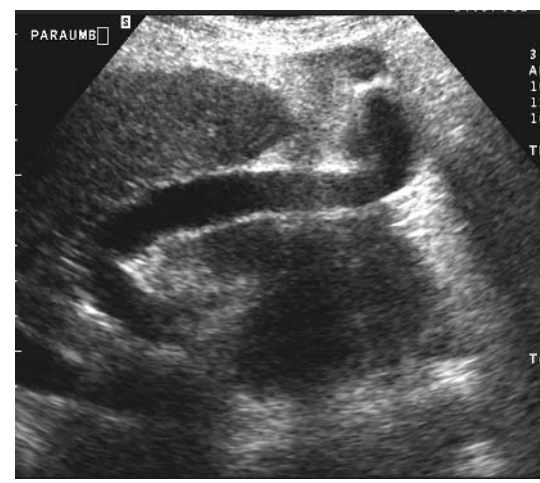

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Figure 4.22 Portal hypertension—further signs. (A) Cavernous transformation of the PV. (Note also the small cyst at the porta, which does not demonstrate flow.) (B) The tortuous vessels of a spleno-renal shunt are demonstrated along the inferior border of the spleen. (C) Colour Doppler demonstrates the tortuous vascular channel of a spleno-renal shunt. (D) Large patent para-umbilical channel running along the ligamentum teres to the anterior abdominal wall in a patient with end-stage chronic liver disease and portal hypertension.

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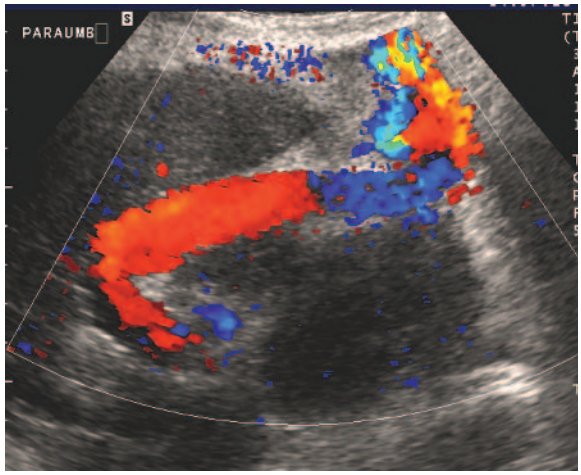

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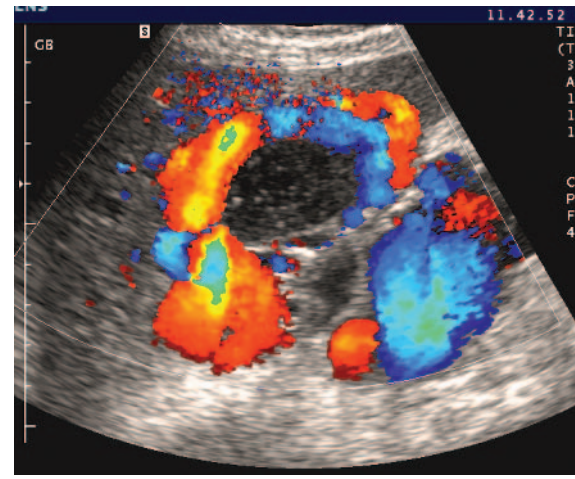

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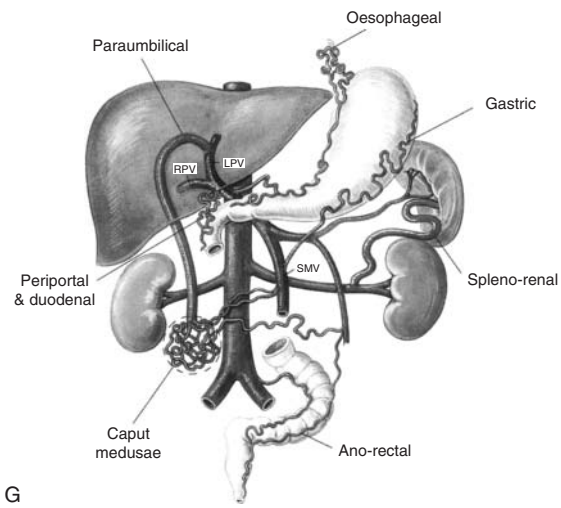

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Figure 4.22 cont'd (E) The para-umbilical vein culminates in a caput medusae just beneath the umbilicus. (F) Varices can be seen around the gallbladder wall in a case of hepatic fibrosis with portal hypertension. (G) Collaterals in portal hypertension (schematic representation).

Contrast angiography with arterioportography is considered to be the gold standard for assessing portal vein patency, but this technique is time-consuming and invasive and has similar results to carefully performed ultrasound.[23]

Ascites This is a transudate from the serosal surfaces of the gut, peritoneum and liver.

Splenomegaly This is the result of back-pressure in the portal and splenic veins. The spleen can enlarge to six times its normal size.

Varices (Fig. 4.22) These are venous anastomoses from the high-pressure portal system to the lower-pressure systemic circulation, which shunts the blood away from the portal system. These vessels have thinner walls than normal vessels, which makes them prone to bleeding.

The common sites are:

- *Gastric and lower oesophagus* Oesophageal varices are particularly prone to bleeding and this is often the patient's presenting symptom. They are difficult to see on abdominal ultrasound because of overlying stomach and are better demonstrated with endoscopic techniques. Left coronal scans may demonstrate tortuous vessels at the medial aspect of the upper pole of the spleen.
- *Spleno-renal* An anastomosis between the splenic and left renal veins which is often seen on ultrasound as a large, tortuous vessel at the lower edge of the spleen (Fig. 4.22B, C). (These anastomoses are usually very efficient at

redirecting the blood from the portal system and so these patients have a lower incidence of gastric varices and therefore a better prognosis.)

- *Periumbilical* A substantial vessel can often be seen in the liver lying in the ligamentum teres (Fig. 4.22D, E), and running down the anterior abdominal wall to a knot of vessels at the umbilicus, the so-called 'caput medusae'. (A patent para-umbilical channel may occasionally be seen in normal patients, but with a diameter of 1 or 2 mm.)
- *Porta hepatis* Varices around the main portal vein itself, especially if the latter is thrombosed (see below).
- *Gallbladder wall* Rarely, varices form around the gallbladder wall to bypass the main portal vein and feed into the intrahepatic portal branches (Fig. 4.22F).
- *Coronary vein* A vessel may be seen arising from the portal vein near the superior mesenteric vein, directing blood in a cephalic direction. (This can sometimes be seen in normal patients.)

It is fair to say that the extent of portosystemic collaterals is usually underestimated on ultrasound. However, a systematic approach which investigates all the possible sites can demonstrate up to 90% of collaterals.[20, 24] (Fig. 4.22G).

The hepatic artery This may also be another ultrasound clue to compromised portal venous flow. The main hepatic artery may demonstrate increased flow velocity, especially if the PV is thrombosed. This is a compensatory mechanism to maintain the blood flow into the liver. The main hepatic artery may appear enlarged and more obvious than usual on ultrasound, and in some cases, peripheral intrahepatic arterial flow is also easily demonstrated (Fig. 4.23).

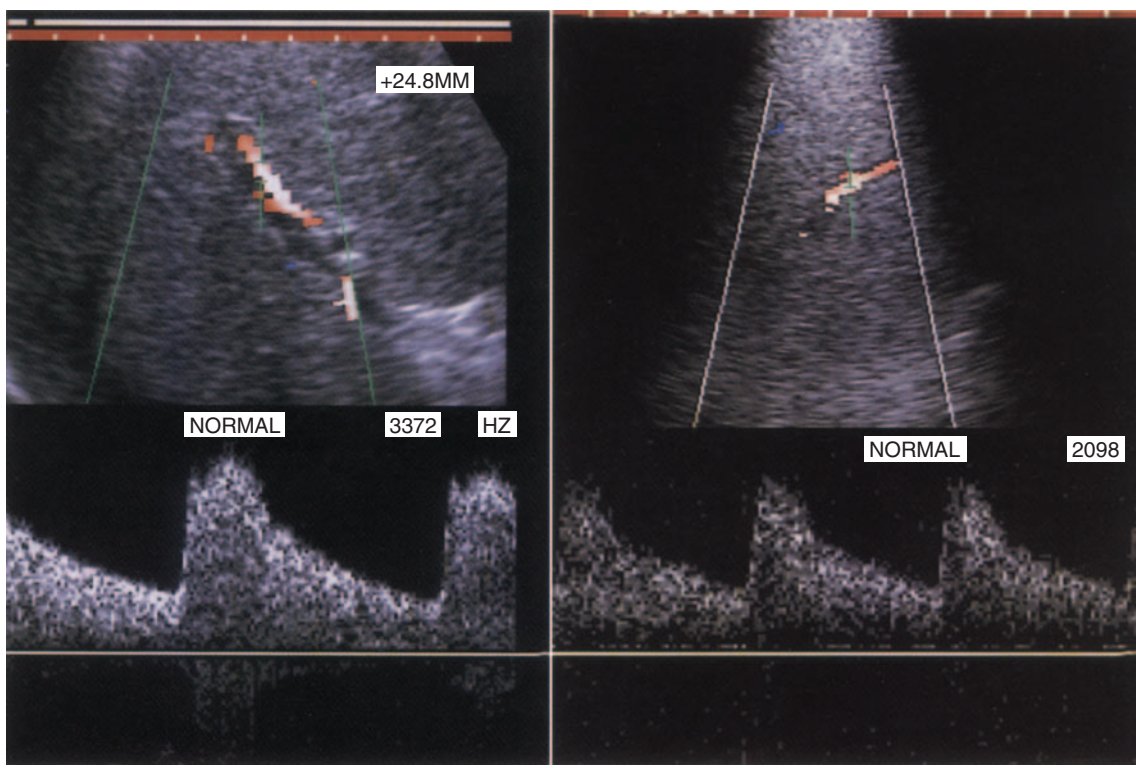


Figure 4.23 (A) Vigorous, high-velocity middle hepatic artery (MHA) flow in the presence of portal vein thrombosis. (B) Arterial flow is also readily demonstrated in the peripheral intrahepatic arteries.

Management of portal hypertension

This depends on the cause and on whether the PV is still patent or not. The most pressing problem is likely to be bleeding from varices, especially oesophageal varices, and patients may present with melaena or haematemasis. Management may involve medical means, endoscopic techniques (either injection sclerotherapy of oesophageal varices or banding, in which a ring is placed around the base of the varix causing thrombosis), compression using a Sengstaken tube with an inflated balloon, surgical or percutaneous transjugular intrahepatic portosystemic shunt (TIPS). All these methods are relatively temporary, and can relieve pressure in the portal venous system, controlling portal hypertensive complications in order to plan further management.

TIPS is a percutaneous method used to relieve the symptoms of portal hypertension in cirrhotic patients. It connects the portal vein directly to the right hepatic vein with an expandable metal shunt. A catheter and guide wire are passed, under X-ray control, through the jugular vein to the inferior vena cava (IVC) and into the hepatic vein. A pathway is then forged with a needle through the liver parenchyma to join the PV with the insertion of a shunt to keep the channel open. Portal venous blood then effectively bypasses the liver, flowing straight into the hepatic vein. This usually results in the speedy decompression of varices and improvement of other symptoms of portal hypertension.

Ultrasound may be used to monitor stent patency (Fig. 4.24). Shunt stenosis or occlusion is a common problem, particularly in long-term shunts; this can be detected with routine postprocedure ultrasound screening and treated with reintervention. The most common site for a stenosis is at the junction of the stent with the PV. The velocity of blood flow in the shunt should be between 1 and 2 m/s and this should be consistent throughout the stent. A variety of Doppler parameters can be used to detect the malfunction of the shunt. A shunt velocity of less than 50 cm/s is a sign of stenosis[25]

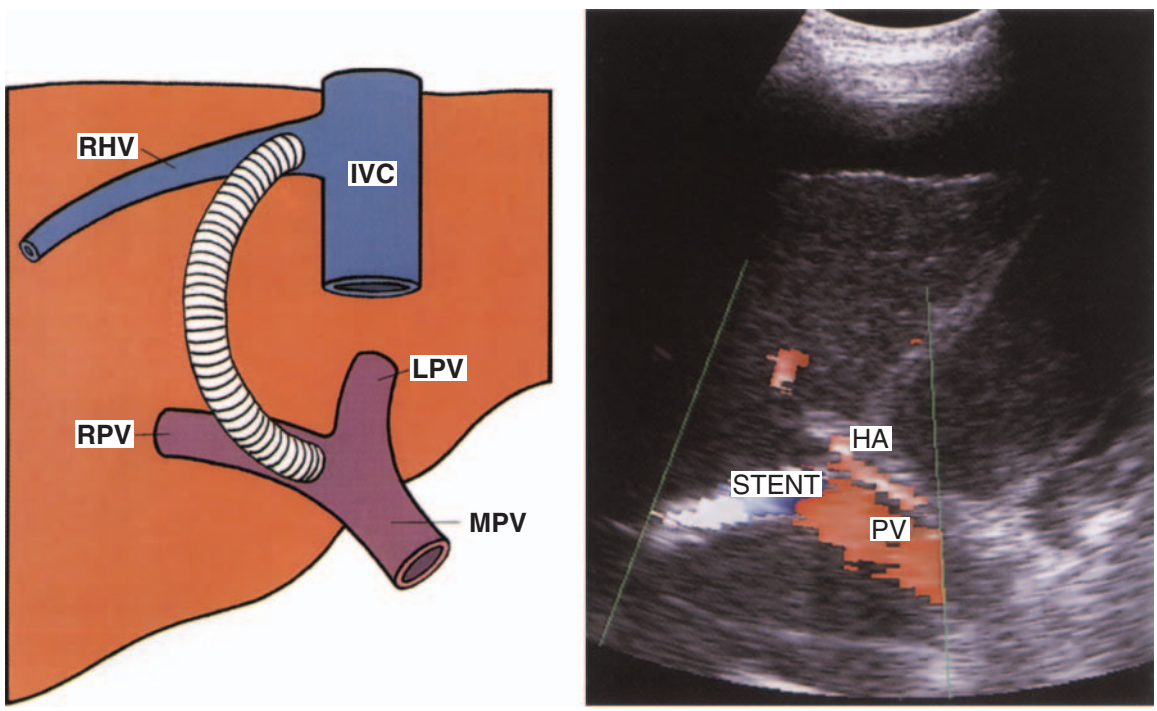
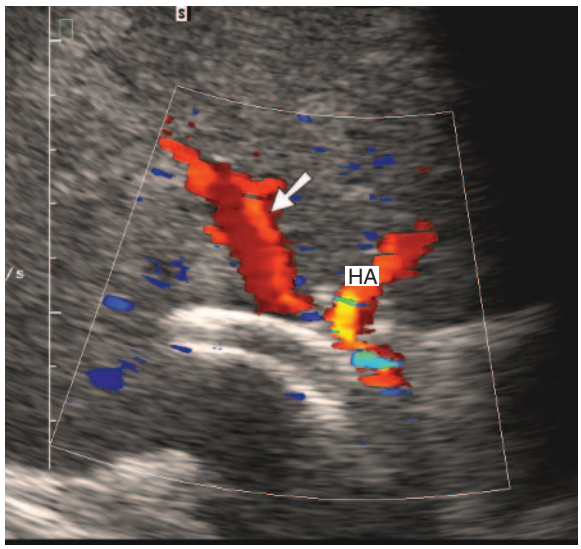


Figure 4.24 (A) Transjugular intrahepatic portosystemic shunt (TIPS). (B) TIPS shunt in a patient with severe portal hypertension. The higher-velocity MHA is seen anterior to the shunt, which demonstrates flow from right to left of the image.

(Continued)



C

Figure 4.24 cont'd (C) Thrombosed TIPS shunt. A recanalized left portal vein (LPV) (arrow) can be seen anterior to this.

but this has not been reproducible in all institutions, and other factors such as a change of 50 cm/s or more from the baseline scan, a localized elevation of velocity at the stenotic site (with an upper limit of normal of up to 220 cm/s) or an increase in the velocity gradient (as the stenotic stent exhibits an increased maximum velocity and a decreased minimum velocity) are also poor prognostic signs.[26]

TIPS is regarded as a temporary measure but can considerably improve the patient's condition pending treatment of chronic liver disease, relieving haemorrhage from varices, relieving intractable ascites and stabilizing liver function. It is increasingly used as a bridge to liver transplant. It is also used as an alternative to surgery in patients who are poor surgical risks, although the diversion of blood away from the liver can result in adversely affected liver function and eventual encephalopathy.[27]

Hepatitis

Viral hepatitis

Acute viral hepatitis may be caused by one of several viruses: A, B, C, D or E. The viruses which cause hepatitis B, C and D may also go on to chronic disease and predispose the liver to HCC in the later stages. Vaccines exist for A and B, but not yet for the others. Hepatitis A and E are transmitted via contaminated food or drink and are particularly prevalent in third-world countries. Hepatitis B, C and D are likely to be transmitted through transfusion or sexual contact.

Fulminant hepatitis, in which there is complete liver failure, is a rare complication of acute hepatitis B.

Most patients with acute hepatitis recover completely, but hepatitis B, C and D may go on to develop *chronic hepatitis*. This has two forms:

- *Chronic persistent hepatitis* is a mild form of inflammation limited to the portal tracts. It is usually of comparatively little clinical significance and does not show ultrasound changes.
- *Chronic active hepatitis* is a more serious and aggressive form of the disease which causes diffuse, persistent inflammation. This may eventually lead to cirrhosis, which can be associated with HCC.

Other causes of acute hepatitis

Acute hepatitis may also occur with many other conditions. The most common of these are alcoholic hepatitis (see alcoholic cirrhosis, above), infectious mononucleosis, herpesvirus and cytomegalovirus.

Patients with AIDS and those who are immunosuppressed are also particularly prone to hepatitis.

Clinical features of hepatitis

It may be asymptomatic (patients who have antibodies present, but who deny having had the disease, must have had subclinical disease at one time). Other signs include lethargy, nausea, vomiting and jaundice. The liver is enlarged and tender in the acute phase.

The diagnosis and classification of hepatitis must be made histologically, ideally with an ultrasound-guided biopsy.

Ultrasound appearances of hepatitis

The liver frequently appears normal on ultrasound. In the acute stage, if ultrasound changes are pres-

ent, the liver is slightly enlarged with a diffusely hypoechoic parenchyma. The normally reflective portal tracts are accentuated in contrast (Fig. 4.25A). This 'dark liver' appearance is non-specific, and may also occur in leukaemia, cardiac failure, AIDS and other conditions.

The inflammation may start at the portal tracts working outwards into the surrounding parenchyma, the so-called periportal hepatitis. In such cases, the portal tracts become less well-defined and hyperechoic. The gallbladder wall may also be thickened, and some patients demonstrate portal lymphadenopathy.

If the disease progresses to the chronic stage, the liver may reduce in size, becoming nodular and coarse in appearance (Fig. 4.25).

Primary sclerosing cholangitis (PSC)

This is a primary disease of the biliary ducts, most frequently found in young men. Like PBC, it is a cholestatic disease. It is discussed more fully in Chapter 3, but is included here for reference as it may often result in a coarse liver texture, similar to that seen in some forms of cirrhosis, and is associated with the formation of cholangiocarcinomas.

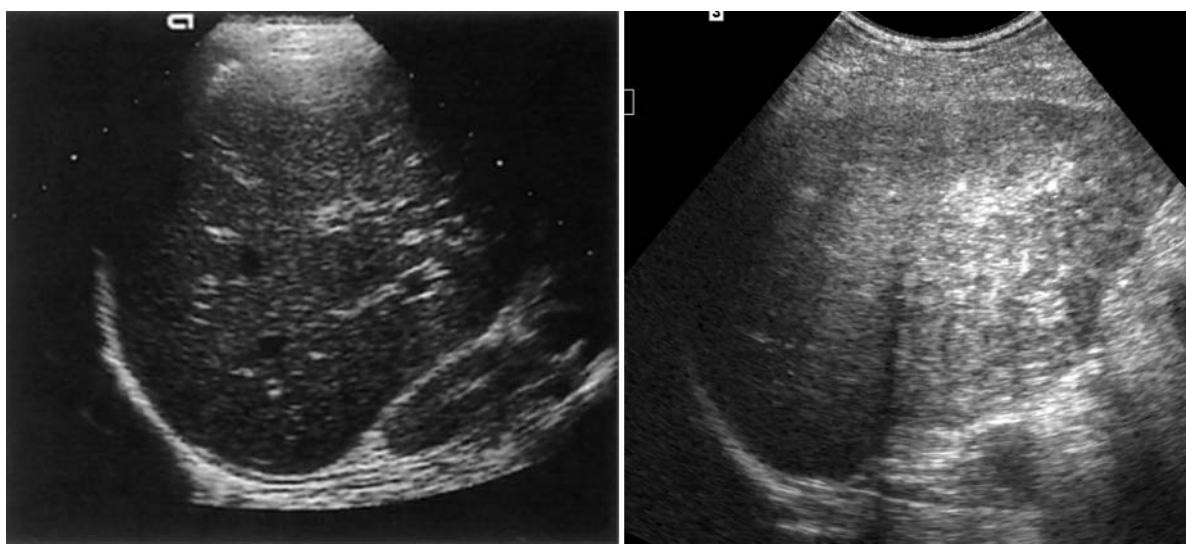
Budd–Chiari syndrome (BCS)

Budd–Chiari syndrome is the name given to the symptoms associated with partial or complete occlusion of the hepatic veins. There are numerous causes of hepatic vein occlusion, of which the main ones are:

- congenital or acquired coagulation disorders, which may affect both the hepatic and portal veins (potentially treatable by liver transplant)
- malignancy: primary or secondary liver tumour may invade the hepatic veins or may travel up the IVC (for example renal carcinoma) to occlude the hepatic vein confluence
- congenital web obstructing the IVC (surgically removable).

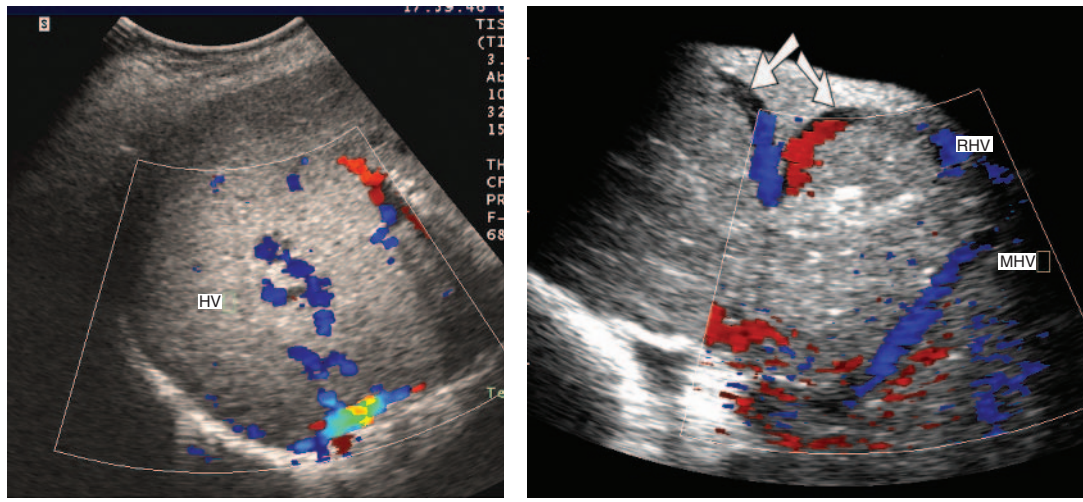
Ultrasound appearances of Budd–Chiari syndrome

In the acute stage, the liver may enlarge. As the condition progresses, compensatory hypertrophy of any 'spared' segments occurs—usually the caudate lobe, because the venous drainage from here is inferior to the main hepatic veins. The hepatic veins may be difficult or impossible to visualize (Fig. 4.26).



A B

Figure 4.25 (A) Subtle changes of oedema in acute hepatitis: the liver is hypoechoic compared with the right kidney, mildly enlarged and has prominent portal tracts. (B) Chronic hepatitis and cirrhosis, demonstrating a coarse-textured, nodular liver.



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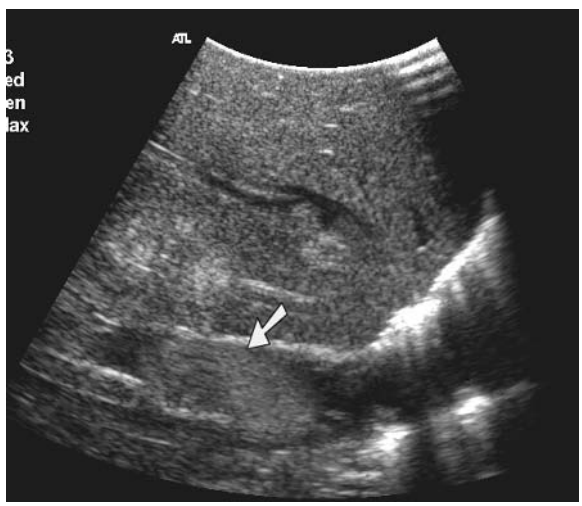


C

Figure 4.26 (A) Budd–Chiari syndrome (BCS). The MHV is tortuous and strictured, and difficult to identify on ultrasound. (B) Large collaterals are seen (arrows) near the surface of the liver in BCS. (C) Tumour thrombus from a renal carcinoma occludes the inferior vena cava (IVC), causing BCS.

Dilated serpiginous collateral veins may form to direct blood away from the liver and in some cases the portal venous flow reverses to achieve this. The spleen also progressively enlarges and, if the disease is long-standing, the liver becomes cirrhotic, acquiring a coarse texture.

Ascites may also be present, particularly if there is complete obstruction involving the IVC. The cause of IVC obstruction may be a web, which can occasionally be identified on ultrasound. If the cause of BCS is a coagulation disorder, the portal venous system may also be affected by thrombosis, causing portal hypertension.

Doppler is particularly helpful in diagnosing BCS.[21] The hepatic veins and IVC may be totally or partially occluded; if partial, the waveforms may become flattened, losing their characteristic triphasic pattern. In some cases flow may be reversed in the IVC, hepatic and/or portal veins. Ultrasound may miss partial hepatic vein occlusion, but the use of contrast agents in suspected cases of BCS may improve diagnostic accuracy.

Management of Budd–Chiari syndrome

This depends upon the cause. Both medical and surgical treatments have mixed success. Severe coagulative disorders may have to be transplanted, although there is a significant risk of recurrence. If the cause is an IVC web, this may be surgically

removed. In some patients, palliative treatment with percutaneous stent placement in the hepatic veins can relieve the symptoms of ascites and varices.[28] Ultrasound may assist in guiding the placement of stents.

Cystic fibrosis

Cystic fibrosis, one of the most common chromosomal abnormalities, has historically been associated with the paediatric population. However, increasing success in the management of this condition, particularly in specialist centres, has improved the current median survival to 40 years for a child born in the last decade.[29]

Ultrasound appearances

Progression of the disease means that changes in the ultrasound appearances of the liver are more severe in adults (Fig. 4.27) than children, in whom the liver frequently looks normal (see Chapter 9). Progressive hepatic fibrosis in adults results in a hyperechoic and enlarged liver. Ultimately the liver becomes coarse and nodular in appearance as the features of cirrhosis become apparent. Portal hypertension is a common finding at this stage with splenomegaly, varices, ascites and possibly PV thrombosis (see above). Changes of fibrosis can also be seen in the pancreas.

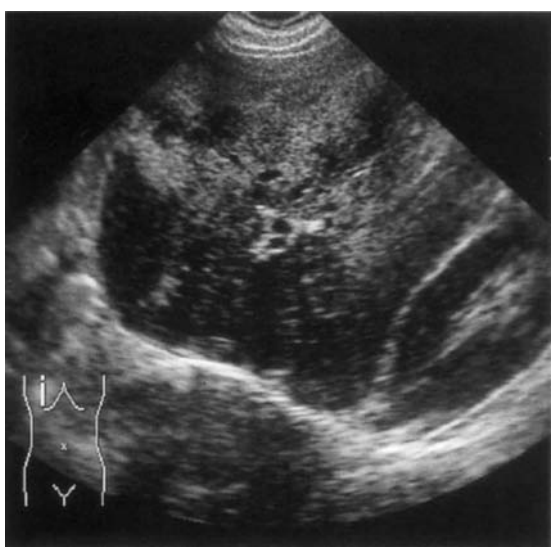


Figure 4.27 Marked changes in the liver of an adult patient with cystic fibrosis.

Congestive cardiac disease

Patients with cardiac failure frequently demonstrate dilated hepatic veins in the liver, sometimes with a dilated IVC. Although this may give the sonographer the overall impression of hypoechogenicity, due to the proliferation of large, anechoic vessels, the liver texture itself tends to be of either normal echogenicity, or, in the later stages of failure, hyperechoic.

Mitral valve disease may be the cause of altered waveforms in the hepatic veins; the usual triphasic flow becomes more pronounced, with a highly pulsatile waveform (Fig. 4.28A).

The portal venous waveform may sometimes be altered in cases of tricuspid valve regurgitation. The normally monophasic flow may become bidirectional (Fig. 4.28B). This phenomenon, associated with congestive heart failure, also occurs in cirrhosis prior to PV thrombosis. However the latter 'balanced' flow is of very low velocity (Fig. 4.21C), while that due to tricuspid regurgitation is a higher-velocity, more pulsatile waveform.

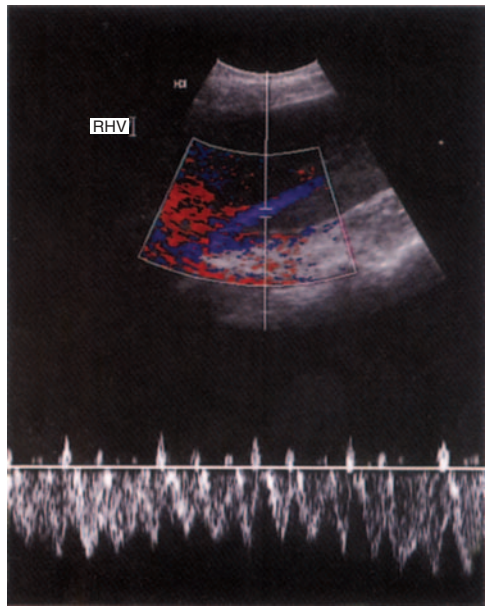
Liver conditions in pregnancy

Acute fatty liver

This rare condition occurs in the third trimester of pregnancy. Acute fatty deposition in the liver tissue can cause abdominal pain, vomiting and jaundice. The liver may appear sonographically normal or be diffusely hyperechoic, although focal areas of fatty deposition have also been reported. Acute fatty liver tends to resolve during the first month of the postpartum period, but may in rare cases progress to cause liver failure.

HELLP syndrome

The HELLP syndrome is a rare complication of pregnancy occurring in up to 20% of mothers with severe pre-eclampsia.[30] Haemolytic anaemia (H), elevated liver enzymes (EL) and low platelet count (LP) cause abdominal pain, nausea and fever.



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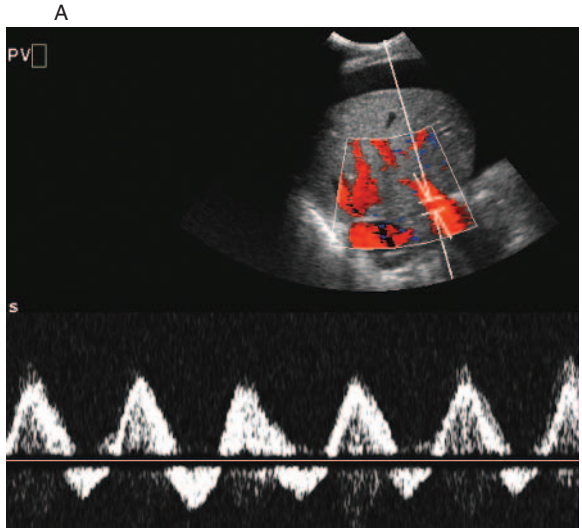


B

Figure 4.28 (A) The waveform of the hepatic vein in a patient with mitral valve disease demonstrates increased pulsatility. (B) The portal vein has an abnormal, highly pulsatile flow waveform in this patient with tricuspid regurgitation. This is quite distinct from the low-velocity 'balanced flow' of portal hypertension.

Its complications include areas of haemorrhage (either subcapsular haematoma or intraparenchymal bleeding), infarction or necrosis within the liver which can be identified with ultrasound or MRI scanning (Fig. 4.29).

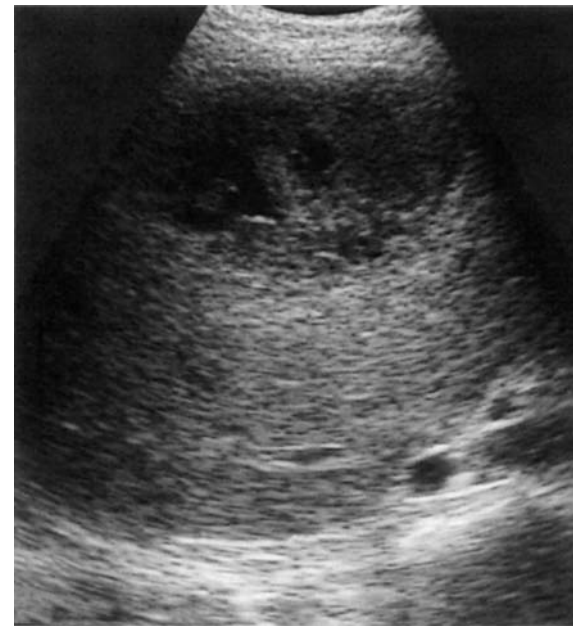


Figure 4.29 Liver infarct in pregnancy in a patient with HELLP syndrome.

The recognition and prompt diagnosis of acute fatty liver and HELLP syndrome reduce maternal morbidity by enabling emergency caesarean section to be performed.

Causes of changes in liver reflectivity are listed in Table 4.5. Causes of free intraperitoneal fluid are listed in Table 4.6.

LIVER TRANSPLANTS

Indications for transplant

Liver transplantation has now become a successful treatment for many chronic liver conditions and is also used in the treatment of fulminant hepatic failure. The range of indications has steadily increased as surgical techniques have developed and immunosuppression has improved (Table 4.7). The majority of hepatic transplants (80%) are still performed in patients with cirrhosis and primary cholestatic disease.[31]

The 5-year survival rate is between 65 and 90%.[32,33] This is highly dependent upon both the primary disease and upon the clinical state of the patient.

Currently, seven centres in the UK perform liver transplants, totalling around 700 patients per year.

Table 4.5 Causes of changes in liver reflectivity

Increased echogenicity
—fatty infiltration (also increases attenuation)
—fibrosis
—cirrhosis
—chronic hepatitis
—cystic fibrosis
Decreased echogenicity
—acute hepatitis
—AIDS
—leukaemia
—toxic shock syndrome
—can be normal, particularly in the young
Coarse or nodular texture
—cirrhosis, various aetiologies
—regenerating nodules
—metastases/diffuse metastatic infiltration
—chronic or granulomatous hepatitis
—PSC, PBC
—diffuse infective process, e.g. with AIDS or immunosuppressed patients

AIDS = acquired immunodeficiency syndrome; PSC = primary sclerosing cholangitis; PBC = primary biliary cirrhosis.

Table 4.6 Causes of free intraperitoneal fluid

Organ failure
—chronic liver disease with portal hypertension
—acute liver failure
—renal failure
—cardiac failure
Malignancy
Inflammatory
—acute pancreatitis
—acute cholecystitis
—peritonitis, TB
—Crohn's disease
Budd–Chiari syndrome
Postoperative
—blood, urine, bile or lymphatic fluid
Organ damage
—biliary perforation
—urinary tract perforation
—bowel perforation (e.g. in diverticulitis)
—trauma to liver, spleen or pancreas
CAPD fluid
—patients on peritoneal dialysis
Ruptured ectopic pregnancy
—haemoperitoneum
Gynaecological
—ruptured ovarian cyst, ovarian carcinoma, ovarian fibroma
—(Meig's syndrome), ovarian torsion, PID

TB = tuberculosis; CAPD = continuous ambulatory peritoneal disease; PID = pelvic inflammatory disease.

This figure has remained relatively stable for some time and is dependent upon the availability of donor organs.

Worldwide, the most common cause for liver transplantation is hepatitis C. The indications for transplant are now many and varied and the number of absolute contraindications continues to dwindle, including AIDS and extrahepatic malignancy.[34]

Transplantation in patients with malignant liver disease has a poorer prognosis with a lower 5-year survival. However, the presence of small HCCs in patients with chronic liver disease is not a contraindication, and tumour recurrence is uncommon in these patients. Patients with larger HCCs (> 3 cm) and those with cholangiocarcinoma have a higher rate of recurrence post-transplant, and are generally not considered for transplantation.

Preoperative assessment

The ultrasound scan is one of many investigations leading up to transplantation. The diagnosis of liver pathology often uses ultrasound scanning as a first line, augmented by histology and additional cross-sectional imaging.

The role of ultrasound includes contributing to, or confirming, the initial diagnosis, assessing the degree of severity and associated complications of the disease and providing guidance for biopsy. An important objective is also to exclude patients for whom liver transplant is not feasible, or of little benefit (Table 4.8), for example those with extrahepatic malignant disease.

The preoperative scan includes all the features of any abdominal ultrasound survey, with the emphasis on assessing the complications of the disease, depending upon the initial diagnosis.

In particular, the sonographer should look for:

- *Portal vein thrombosis*: this may be a contraindication to transplant if it is extensive,

Table 4.7 Indications for liver transplantation

Chronic cholestatic disease
—PBC, PSC
Cirrhosis
—from hepatitis, alcoholic liver disease or other causes (without malignancy)
Biliary atresia
—usually in children who have developed SBC
Malignancy
—patients with HCC associated with cirrhosis, provided the lesion is small (< 3 cm) and solitary
Budd–Chiari syndrome
—non-malignant occlusion of the hepatic veins, especially total venous occlusion and/or patients with cirrhosis resulting from BCS
Fulminant hepatic failure
—due to drug (usually paracetamol) overdose, acute hepatitis, BCS, Wilson's disease or massive hepatic trauma (an acute situation requiring immediate transplant if a suitable donor is found)
Others
—rarely, transplant is undertaken for benign lesions such as PCD, adenoma or large haemangiomas

PBC = primary biliary cirrhosis, PSC = primary sclerosing cholangitis, SBC = secondary biliary cirrhosis, BCS = Budd–Chiari syndrome, PCD = polycystic disease

Table 4.8 Contraindications to liver transplant

Absolute
—extrahepatic malignancy
—active extrahepatic sepsis
—severe cardiopulmonary disease
—AIDS
—inability to comply with regular postoperative drug treatment
Relative
—age > 65, particularly if related to poor general health
—moderate cardiopulmonary disease
—PV thrombosis
—active alcoholism or drug abuse
—previous complex hepatic surgery
—multiple or large focal hepatic malignancies (e.g. cholangiocarcinomas associated with PSC)

AIDS = aquired immunodeficiency syndrome, PV = portal vein

or unable to be effectively bypassed by the surgeon.

- Any of the features of *portal hypertension* associated with chronic liver disease (see above).
- *Focal liver lesions* which may represent malignancy. These may require the administration of ultrasound contrast agents, or further imaging to characterize, such as MRI. An HCC greater than 3 cm in diameter has an 80% chance of recurrence post-transplant. If under 2 cm and solitary, this is likely to be cured. Check the size, number and local spread of disease.
- It is useful to document the *spleen size* as a baseline for postoperative comparisons.
- *Extrahepatic malignancy*, in cases with an initial diagnosis of carcinoma.
- Degree and scope of *vascular thrombosis* in cases of BCS.
- Any *incidental pathology* which may alter the management plan.

Doppler ultrasound is, of course, essential in assessing the patency and direction of blood flow of the portal venous system, the hepatic veins, IVC and main hepatic artery. It may occasionally be possible to demonstrate arterial anomalies. While large numbers of patients are considered for transplant and undergo ultrasound assessment, the majority of these will never actually *be* transplanted. This factor has numerous implications for resources when setting up a transplant ultrasound service.

Operative procedure

Most transplants are *orthotopic*, that is the diseased liver is removed and replaced by the donor organ, as opposed to *heterotopic*, in which the donor organ is grafted in addition to the native organ (like most kidney transplants).

If the patient suffers from extensive varices, which may bleed, the removal of the diseased organ prior to transplant is particularly hazardous.

Donor livers which are too large for the recipient, for example in small children, may require cutting down to reduce the size. There is an increasing trend towards a 'split liver' technique, in which the donor liver is divided to provide for two recipients. The lack of donors has also led to the development of living-related donor transplantation for paediatrics.

The transplant requires five surgical anastomoses:

- suprahepatic vena cava
- infrahepatic vena cava
- hepatic artery (either end-to-end, or end-to-side to aorta)
- PV
- CBD (the gallbladder is removed).

IOUS is useful for assessing the size and spread of intrahepatic neoplastic growths and to assess vascular invasion in the recipient. Mapping of the hepatic vascular anatomy in living-related donors is also feasible using IOUS.

IOUS with Doppler is also useful for assessing the vascular anastomoses and establishing if portal venous and hepatic arterial flow are adequate.

Postoperative assessment

Ultrasound plays a key role in the postoperative monitoring of liver transplant patients. Numerous complications are possible (Table 4.9) and many of these can be diagnosed with ultrasound.

The operation is generally followed by ciclosporin immunosuppression. Blood levels of ciclosporin are a closely monitored balancing act; too low and the graft may reject, too high and the toxic effects of the drug may affect the kidneys.

Liver function is biochemically monitored for early signs of complications. Elevated serum bilirubin, alkaline phosphatase and/or aminotransferase levels are present with most types of graft dysfunction or complication and are investigated first with ultrasound.

Renal dysfunction is a further recognized complication following transplant. This can be due to various causes, including ciclosporin nephrotoxicity, intraoperative hypotension or preoperative renal failure.

Table 4.9 Postoperative liver transplant complications

Infection
—hepatic abscess/general abdominal infection leading to sepsis
Vascular
—anastomotic leaks → haematoma
—thrombosis or stenosis → ischaemia/infarction
Biliary
—bile duct stricture or stenosis leading to dilatation
—bile leak → biloma
Rejection
—acute episodes are common in up to 80% of patients in the first 2 weeks and are of variable severity
Other medical complications
—neurological
—renal dysfunction
Recurrence of original disease
—hepatitis
—cholangiocarcinoma or hepatocellular carcinoma
—Budd–Chiari syndrome
—PSC
Post-transplant lymphoproliferative disorder (PTLD)
—more common in children, PTLD is more usually associated with immunosuppressions, occurring within the first year of transplant

Postoperative ultrasound appearances

The vessels and vascular anastomoses

These are potential sites of complication in terms of thrombosis, stenosis, occlusion or leakage.

The **hepatic artery** is vital to graft success as it is the sole vascular supply to the biliary system. Most hepatic artery occlusions occur relatively soon after operation, before a good collateral supply is able to be established.

A blocked hepatic artery quickly results in ischaemia with resultant hepatic necrosis and is therefore treated as an emergency requiring surgical intervention and, frequently, retransplant. Taken in context with the clinical picture, the patient may proceed immediately to surgery if the ultrasound diagnosis of occlusion is confident. If doubt exists, MRI or X-ray angiography may be performed. Ensure the artery is scanned intercostally to maintain a low vessel-to-beam angle, and that the

Doppler sensitivity and filter controls are set for low velocities if arterial flow is not found.

Hepatic artery thrombosis or stenosis can lead to bile duct necrosis, causing bile leaks and abscesses, or areas of infarction within the liver tissue.

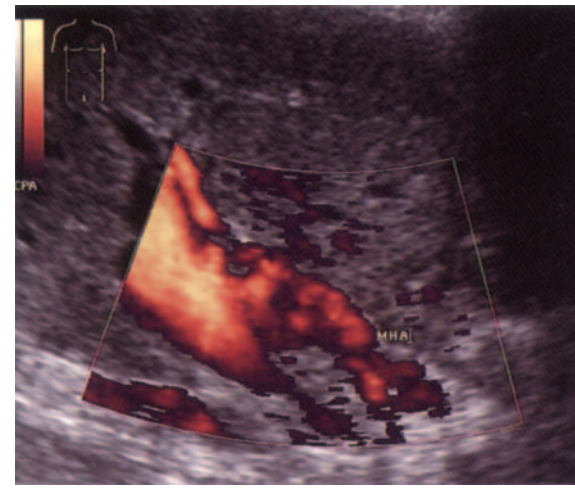
Hepatic artery stenosis/thrombosis is still a relatively common post-transplant complication in up to 12% of adult patients. Colour Doppler ultrasound detects between 50% and 86% of total occlusions[35] and angiography is still considered the gold standard although ultrasound continues to increase its clinical value here.[36] The administration of ultrasound contrast media, whilst potentially useful for detection of flow, is rarely necessary in practice.

Stenosis of the artery at the site of anastomosis is detected by examining the Doppler spectrum (Fig. 4.30). The systolic upstroke tends to be delayed ('tardus parvus' pattern) downstream of the stenosis;[37] the acceleration time is increased (over 0.08 seconds) and the resistance index decreased (less than 55) in many cases.[38] Both or either of these indices may be affected, giving a sensitivity and specificity of 81% and 60% for the diagnosis of hepatic artery stenosis with Doppler.[39]

The appearance of the hepatic artery waveform immediately postoperatively is often one of a small spike with no EDF. This is not a significant finding and will usually develop into the more familiar waveform with forward EDF by 48 hours after transplantation.[40]

The **PV** anastomosis is readily demonstrated at the porta. The waveform invariably shows turbulence associated with the anastomotic site (Fig. 4.31A), as the diameters of the donor and recipient veins invariably differ. This is not significant in itself but can indicate a clinically significant stenosis when accompanied by high velocities of greater than 100 cm/sec (Fig. 4.31B).

PV stenosis also causes a steadily increasing spleen size, which is why it is important to have a baseline measurement of the spleen. PV thrombosis should only be diagnosed using the correct Doppler settings (low pluse repetitions frequency and optimum colour gain) and at an angle as near parallel to the beam as possible. In the absence of colour flow, power Doppler may be helpful in confirming thrombosis, as it is less angle-dependent, and contrast may be used to increase the level of confidence.



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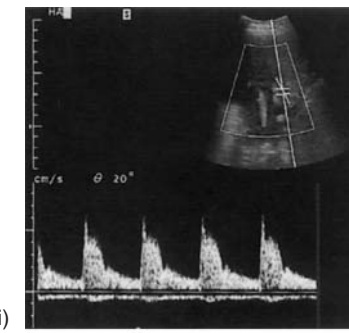
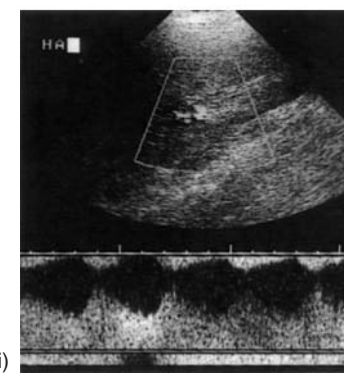


B(i)

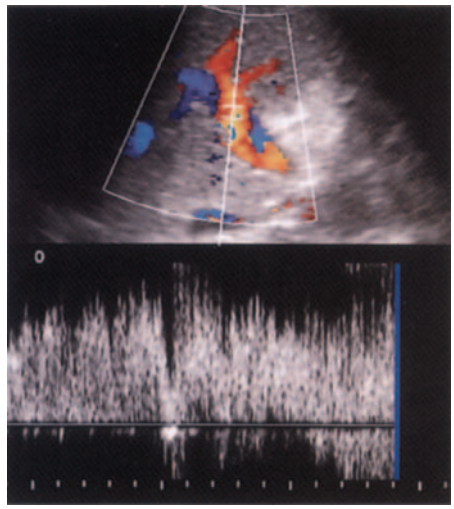


B(ii)

Figure 4.30 (A) MHA in a liver transplant demonstrated on power Doppler, lying anterior to the MPV. (B) i, Normal hepatic artery (HA) waveform post-transplant; ii, 1 month later, the systolic slope shows a tardus parvus pattern. HA stenosis was confirmed with angiography.

It is also possible to have a blocked main PV with patent intrahepatic PVs, due to collateral formation.

The **IVC** infrahepatic anastomosis is also readily seen on ultrasound (Fig. 4.32). Because of the



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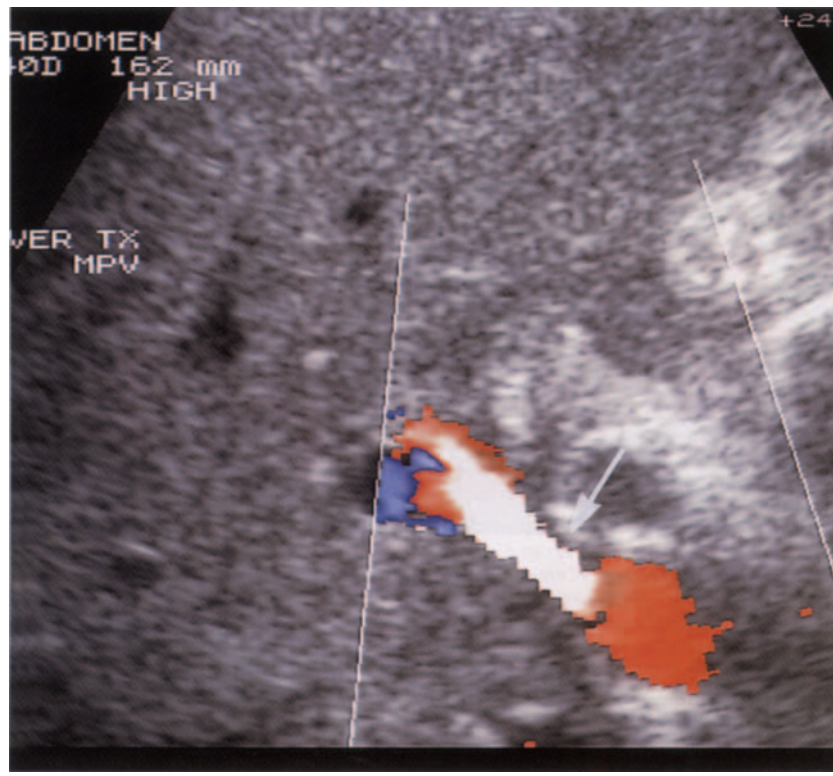

B

Figure 4.31 (A) The portal vein in a liver transplant demonstrates a very turbulent waveform because of the surgical anastomosis. This is not usually a significant finding. (B) MPV stenosis. A high-velocity jet is seen through the stenosis (arrow) at the site of the anastomosis. The spectral Doppler waveform exceeded the Nyquist limit at this point.

near-perpendicular angle of the IVC to the beam it is difficult to assess blood flow velocity in the IVC. Power Doppler is helpful in confirming patency in technically difficult cases as it is angle-independent. Thrombosis in the IVC is a relatively rare complication of transplants, accounting for fewer than 3% of patients.

If the transplant has been performed for BCS, pay particular attention to the hepatic veins, which show a tendency to re-thrombose in some patients.

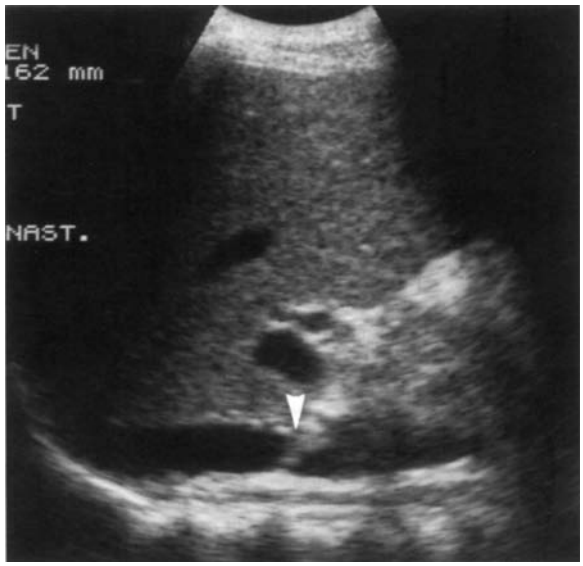

Figure 4.32 The site of anastomosis in the IVC in a liver transplant.

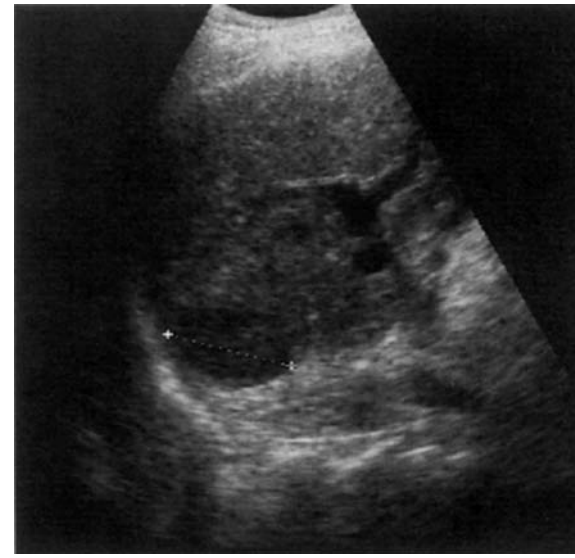

Figure 4.33 An area of infarction in a liver transplant.

The common bile duct

This should be carefully monitored postoperatively. A measurement serves as a baseline from which to detect small degrees of dilatation which may imply stenosis or obstruction. Even relatively minor dilatation can be significant in the transplant patient; cholestasis can precipitate ascending biliary infection which may subsequently form liver abscesses, a process which may be aggravated by immunosuppression.

Biliary complications occur in up to 15% of transplants and most biliary complications become evident during the first 3 months, although late stenosis can occur after this. Strictures commonly occur at the anastomosis due to scar tissue, but other, non-anastomotic strictures can result from hepatic artery insufficiency causing ischaemia. Leakage is a comparatively rare event.

Focal lesions

Focal lesions within the parenchyma of the transplant liver are usually a poor prognostic indicator. Hepatic abscesses may be multiple and are often acoustically subtle in the early stages, with echo patterns closely similar to normal liver tissue. Other causes of focal lesions in the early postoperative period may be due to infarction and are associated with interruption of the arterial supply. These can be hyper- or hypoechoic, have well-defined borders and do not exert a mass effect (Fig. 4.33).

The longer the interval between removing and transplanting the donor liver, the greater the likelihood of ischaemic patches forming.

In patients who have been transplanted following cirrhosis with malignancy, recurrence of HCC may also be a serious complication.

Post-transplant lymphoproliferative disorder may also demonstrate hypoechoic focal lesions within the liver, occasionally also involving the spleen and kidneys.

Fluid collections

These can frequently be demonstrated and monitored with ultrasound. These may represent haematoma (Fig. 4.34), seroma, loculated ascites or biloma. It is not possible to differentiate different types of collection with ultrasound alone. The appearances are taken in conjunction with the clinical features and the role of ultrasound is primarily to monitor the gradual resolution of the collection.

It is important to determine if a collection is infected in a clinically ill patient. This cannot be

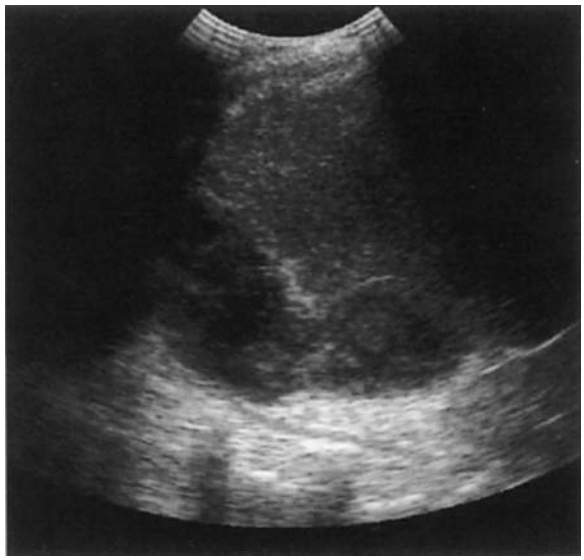

Figure 4.34 Subphrenic haematoma post-transplant.

done on the ultrasound appearances alone and guided aspiration is usually required.

Haematomas frequently resolve if left untreated. However, a large haematoma could result from an anastomotic leak requiring surgical intervention. A leaking bile duct anastomosis is potentially a serious complication which could cause peritonitis. Drainage under ultrasound guidance is a temporary option but surgical repair is invariably necessary. Recent recipients of liver transplants will often have some free intraperitoneal fluid and a right pleural effusion, which resolve spontaneously.

Rejection

Rejection episodes are common in the first 2 weeks after transplantation. Graft rejection may be acute, in which case the immunosuppression is increased, or chronic following several acute episodes. Chronic rejection can only be treated by retransplantation. Rejection does not have any specific ultrasound features on either conventional imaging or Doppler, and the diagnosis is made from a liver biopsy following clinical suspicion.

Post-transplant malignancy

Because of the immunosuppression, patients are at greater risk than normal for developing malignancy. Most of these manifest as post-transplant lymphoproliferative disorder (similar in appearance to non-Hodgkin's lymphoma) which can affect the lymphatics, gastrointestinal tract or other organs, including the transplanted liver.[41] The most commonly found ultrasound appearances include focal, hypoechoic liver lesions and lymphadenopathy.

Patients with malignant lesions pretransplant, such as HCC or cholangiocarcinoma, have a significant risk of recurrence after transplantation.

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Chapter 5

The pancreas

CHAPTER CONTENTS

THE NORMAL PANCREAS

Ultrasound techniques

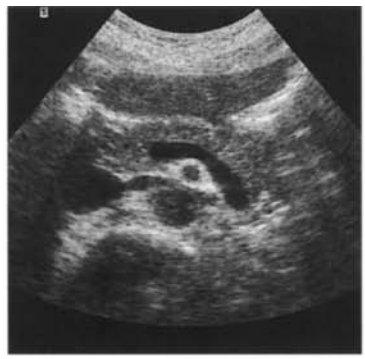
Because the pancreas lies posterior to the stomach and duodenum, a variety of techniques must usually be employed to examine it fully. Although ultrasound may still be considered the first line of investigation, CT, MRI and/or endoscopic retrograde cholangiopancreatography (ERCP) are frequently required to augment and refine the diagnosis.

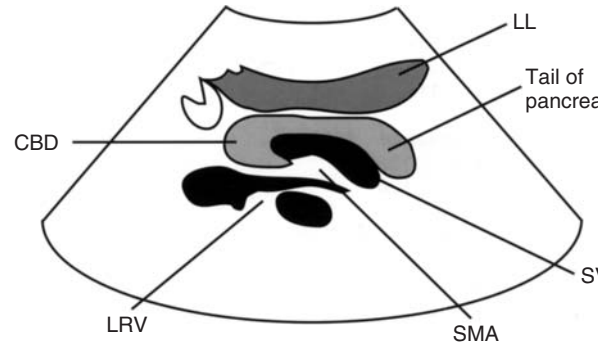
The operator must make the best use of available acoustic windows and different patient positions and techniques to investigate the pancreas fully.

The most useful technique is to start by scanning the epigastrium in transverse plane, using the left lobe of the liver as an acoustic window. Using the splenic vein as an anatomical marker, the body of the pancreas can be identified anterior to this. The tail of pancreas is slightly cephalic to the head, so the transducer should be obliqued accordingly to display the whole organ (Fig. 5.1).

Different transducer angulations display different sections of the pancreas to best effect:

- Identify the echo-free splenic vein and the superior mesenteric artery posterior to it. The latter is surrounded by an easily visible, hyperechoic fibrous sheath. The pancreas is 'draped' over the splenic vein (Fig. 5.1).
- Where possible, use the left lobe of the liver as an acoustic window to the pancreas, angling slightly caudally.
- The tail, which is often quite bulky, may require the transducer to be angled towards





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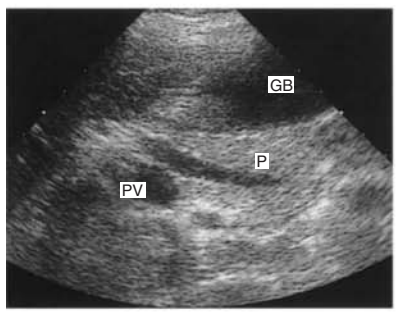

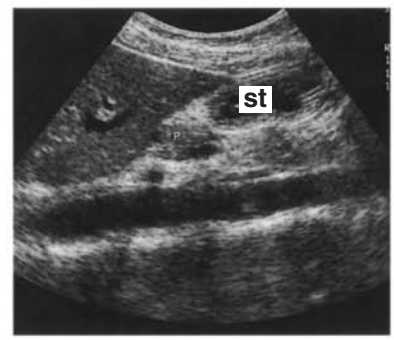

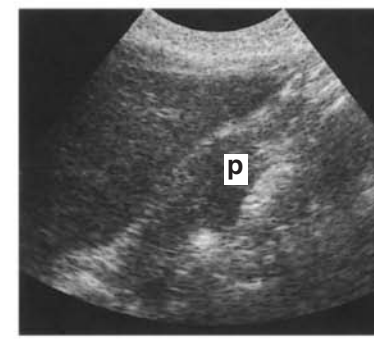

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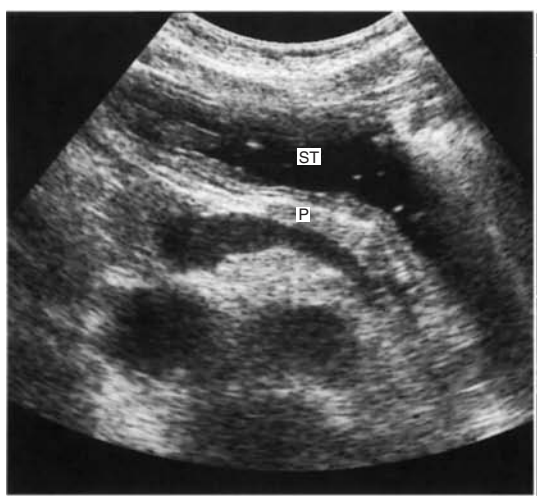

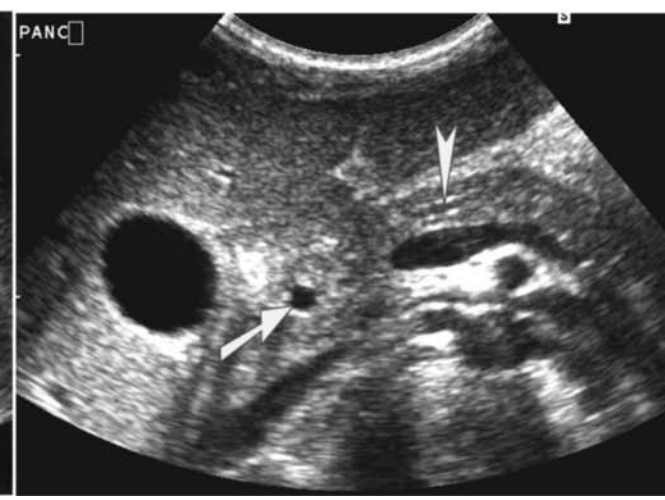

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Figure 5.1 (A) i, ii, Transverse section (TS) showing the normal pancreas. (B) Longitudinal section (LS) oblique to the right of midline, demonstrating the head of pancreas, P, with the common bile duct (CBD) running through it. (C) LS at the midline, demonstrating the body of pancreas. (D) LS angled through the left lobe of the liver towards the tail of pancreas (p). (E) Water in the stomach, ST, provides a window through which to view the pancreas. (F) The main pancreatic duct (arrow) is normally up to 2 mm in diameter (arrow = CBD).

the patient's left. The spleen also makes a good window to the tail in coronal section.

If you can't see the pancreatic head properly, turn the patient left side raised, which moves the duodenal gas up towards the tail of the pancreas. Right side raised may demonstrate the tail better.

If these manoeuvres still fail to demonstrate the organ fully, try:

—asking the patient to perform the Valsalva manoeuvre with abdominal protrusion
—scanning the patient erect
—filling the stomach with a water load to create an acoustic window through which the pancreas can be seen.

Ultrasound appearances

The texture of the pancreas is rather coarser than that of the liver. The echogenicity of the normal pancreas alters according to age. In a child or young person it may be quite bulky and relatively hypoechoic when compared to the liver. In adulthood, the pancreas is hyperechoic compared to normal liver, becoming increasingly so in the elderly, and tending to atrophy (Fig. 5.2).

The pancreas does not have a capsule and its margins can appear rather ill-defined, becoming infiltrated with fat in later life.

These age-related changes are highly significant to the sonographer; what may be considered normal in an elderly person would be abnormally *hyper*echoic in a younger one, and may represent a chronic inflammatory state. Conversely a hypoechoic pancreas in an older patient may represent acute inflammation, whereas the appearances would be normal in a young person.

The main pancreatic duct can usually be visualized in the body of pancreas, where its walls are perpendicular to the beam. The normal diameter is 2 mm or less.

The common bile duct can be seen in the lateral portion of the head and the gastroduodenal artery lies anterolaterally. The size of the uncinate process varies.

Pitfalls in scanning the pancreas

The normal stomach or duodenum can mimic pancreatic pathology if the patient is insufficiently fasted. A fluid-filled stomach can be particularly difficult when looking for pancreatic pseudocysts in patients with acute pancreatitis. Giving the patient a drink of water usually differentiates the gastrointestinal tract from a collection.

Epigastric or portal lymphadenopathy may also mimic a pancreatic mass. If careful scanning and appropriate patient positioning are unable to elucidate, CT is normally the next step.

Biochemical analysis

In many pancreatic diseases, the production of the digestive pancreatic enzymes is compromised, either by obstruction of the duct draining the pancreas or by destruction of the pancreatic cells which produce the enzymes. This can result in malabsorption of food and/or diarrhoea.

The pancreas produces digestive enzymes, amylase, lipase and peptidase, which occur in trace amounts in the blood. If the pancreas is damaged or inflamed, the resulting release of enzymes into the blood stream causes an increase in the serum amylase and lipase levels. The enzymes also pass from the blood stream into the urine and therefore urinalysis can also contribute to the diagnosis.

Congenital anomalies of the pancreas

The normal pancreas is the result of the fusion of two embryonic buds: the ventral bud arises from the CBD, forming the uncinate process and part of the head, and the dorsal arises from the posterior wall of the duodenum. Developmental anomalies of the pancreas occur as a result of a failure of the dorsal and ventral pancreatic ducts to fuse, that is *pancreas divisum*. This arrangement may cause inadequate drainage of the pancreatic duct, leading to pancreatitis. A rare developmental anomaly of the ventral bud may occur, *pancreas annulare*, in which pancreatic tissue encircles the bowel. In this latter case, patients can present with proximal small-bowel obstruction in infancy, but this may also be an incidental finding at autopsy. These relatively uncommon anomalies cannot usually be diagnosed on ultrasound. Increasingly, magnetic resonance cholangiopancreatography (MRCP) is replacing ERCP in the

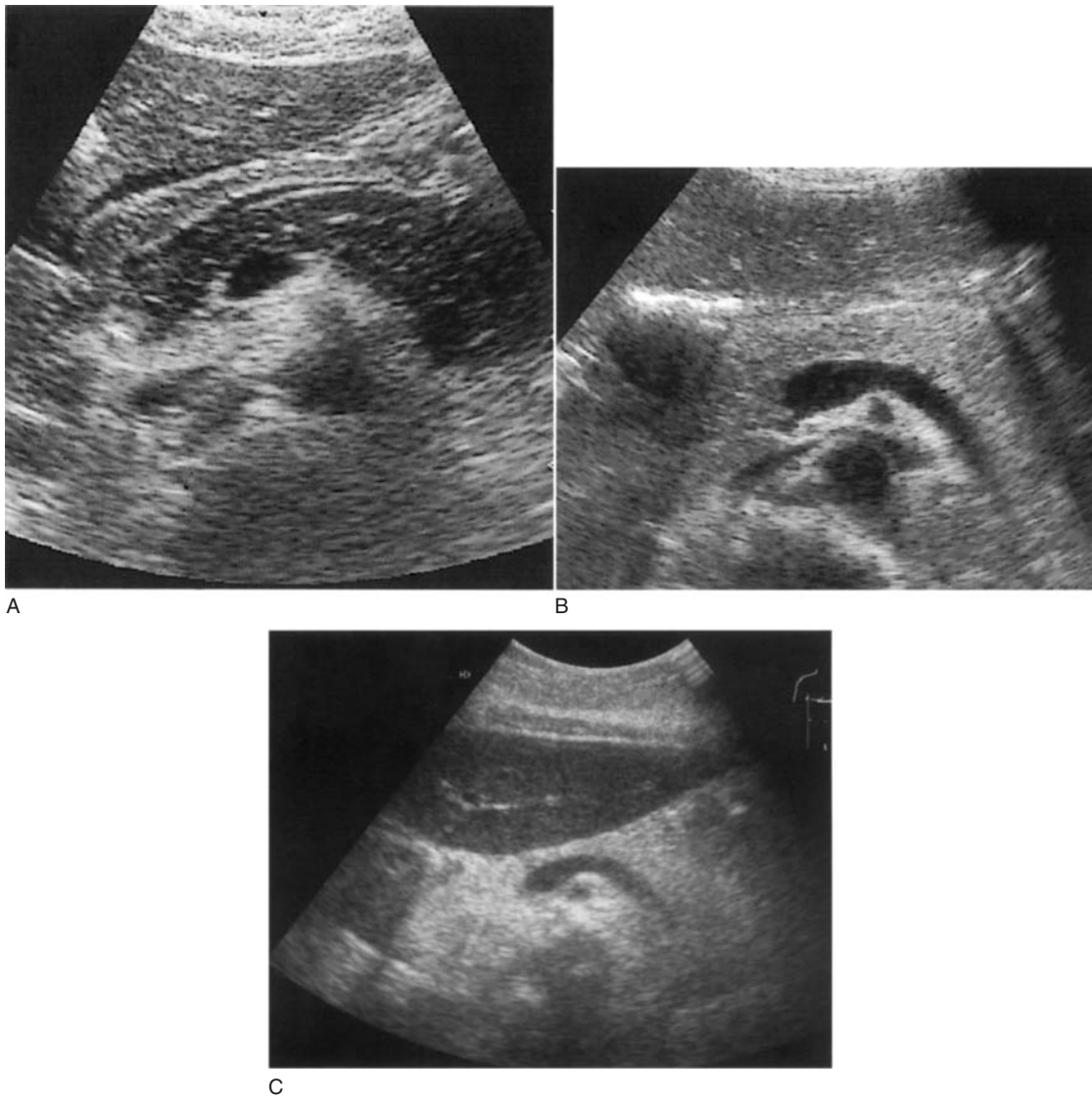


Figure 5.2 (A) Pancreas in a young person, demonstrating normal hypoechogenicity. (B) The normal adult pancreas is slightly more echogenic than the liver. (C) The pancreas becomes hyperechoic in an older patient.

evaluation of the pancreas and ductal system, due to its relative non-invasive nature and low risk compared with ERCP.[1, 2]

Agenesis of the pancreas is very rare, usually in association with other defects, and children usually die soon after birth.

PANCREATITIS

Inflammation of the pancreas may be acute or chronic and is usually a response to the destruction of pancreatic tissue by its own digestive enzymes (*autodigestion*), which have been released from damaged pancreatic cells.

Acute pancreatitis

Clinical features

Acute inflammation of the pancreas has a number of possible causes (Table 5.1), but is most commonly associated with gallstones or alcoholism.

Clinically it presents with severe epigastric pain, abdominal distension and nausea or vomiting. In milder cases, the patient may recover spontaneously. If allowed to progress untreated, peritonitis and other complications may occur.

Biochemically, raised levels of amylase and lipase (the pancreatic enzymes responsible for the digestion of starch and lipids) are present in the blood and urine. Acute inflammation causes the pancreatic tissue to become necrosed, releasing the pancreatic enzymes which can further destroy the pancreatic tissue and also the capillary walls, entering the blood stream.

Ultrasound appearances

Mild acute pancreatitis may have no demonstrable features on ultrasound, especially if the scan is performed after the acute episode has settled. In more severe cases the pancreas is enlarged and hypoechoic due to oedema. The main duct may be dilated or prominent.

As the condition progresses, digestive enzymes leak out, forming collections or *pseudocysts*. These are most frequently found in the lesser sac, near the tail of the pancreas, but can occur anywhere in the abdomen—within the pancreatic tissue itself, anywhere in the peritoneal or retroperitoneal space or even tracking up the fissures into the liver—so a full abdominal ultrasound survey is essential on each attendance (Fig. 5.3).

Pseudocysts are so called because they do not have a capsule of epithelium like most cysts, but are merely collections of fluid surrounded by adjacent tissues. A pseudocyst may appear to have a capsule on ultrasound if it lies within a fold of peritoneum.

Pseudocysts may be echo-free, but generally contain echoes from tissue debris and may be loculated.

In a small percentage of cases, a pseudocyst or necrotic area of pancreatic tissue may become infected, forming a pancreatic abscess.

Although acute pancreatitis usually affects the entire organ, it may occur focally. This presents a diagnostic dilemma for ultrasound, as the appearances are indistinguishable from tumour. The clinical history may help to differentiate; suspicion of focal pancreatitis should be raised in patients with previous history of chronic pancreatitis, a history of alcoholism and normal CA 19–9 levels[4] (a tumour marker for pancreatic carcinoma).

The enlargement of the pancreas in acute pancreatitis may have other consequences, for example the enlarged pancreatic head may obstruct the common bile duct, causing biliary dilatation.

Doppler ultrasound is useful in assessing associated vascular complications. Prolonged and repeated attacks of acute pancreatitis may cause the splenic vein to become encased and compressed, causing splenic and/or portal vein thrombosis, with all its attendant sequelae (see Chapter 4) (Fig. 5.3E).

Although ultrasound is used to assess the pancreas in cases of suspected acute pancreatitis, its main role is in demonstrating the *cause* of the pancreatitis, for example biliary calculi, in order to plan further management. The ultrasound finding of microlithiasis or sludge in the gallbladder is highly significant in cases of suspected pancreatitis,[5]

Table 5.1 Causes of acute pancreatitis

Biliary calculi—most common cause. Obstructs the main pancreatic duct/papilla of Vater and may cause reflux of bile into the pancreatic duct
Alcoholism—alcohol overstimulates pancreatic secretions causing overproduction of enzymes
Trauma/iatrogenic—damage/disruption of the pancreatic tissue, e.g. in a road traffic accident, or by surgery, biopsy or ESWL[3]
Drug-induced—a relatively uncommon cause. Some anticancer drugs can cause chemical injury
Infection—e.g. mumps. A rare cause of pancreatitis
Congenital anomaly—duodenal diverticulum, duodenal duplication, sphincter of Oddi stenosis or choledochal cyst may obstruct the pancreatic duct, giving rise to pancreatitis
Hereditary—a rare, autosomal dominant condition presenting with recurrent attacks in childhood or early adulthood

ESWL = extracorporeal shock wave lithotripsy.

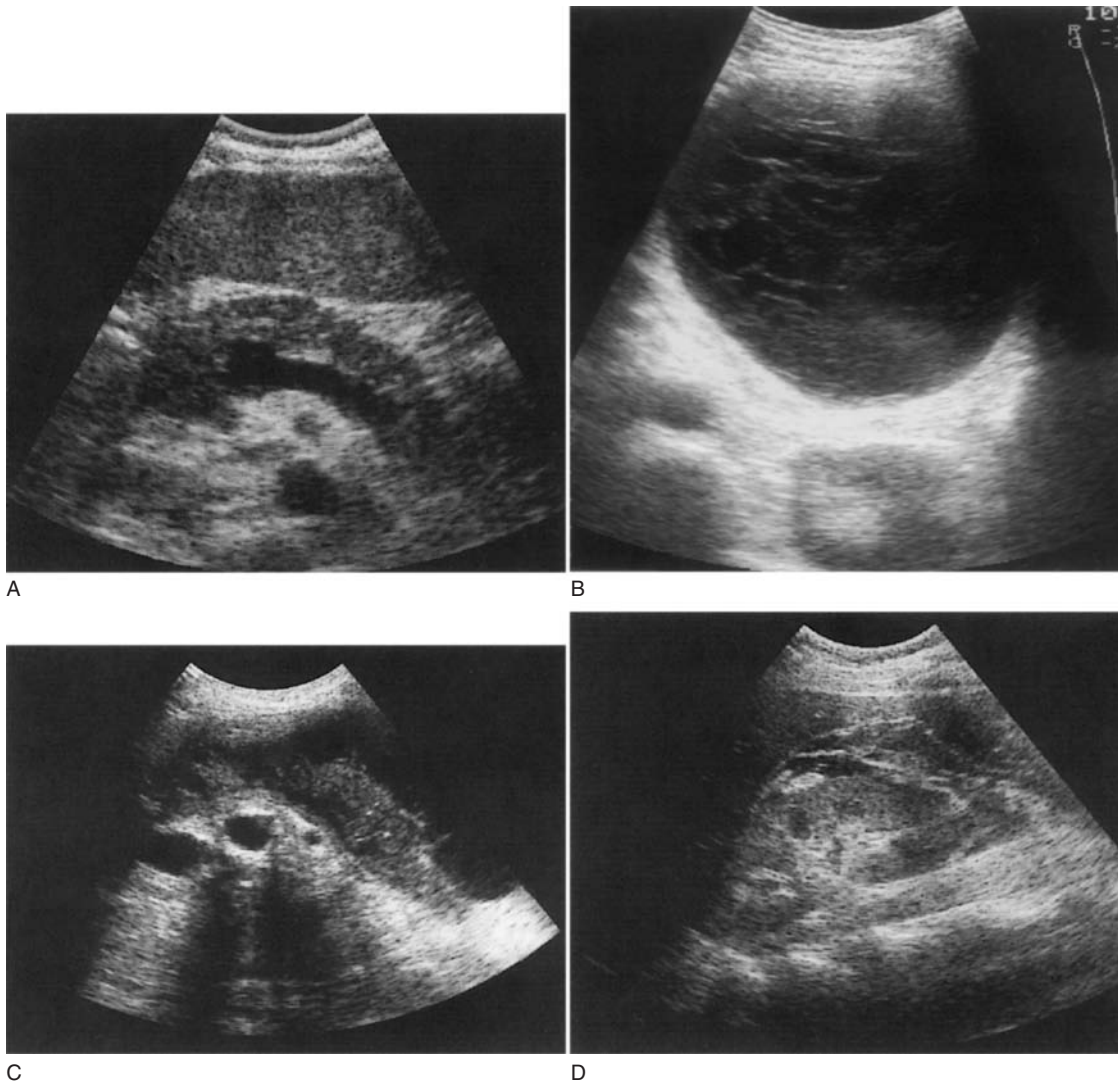


Figure 5.3 (A) Acute pancreatitis in a patient with alcoholic liver disease. The pancreas is hypoechoic and bulky with a lobulated outline. (B) Large pseudocyst near the tail of the pancreas in acute pancreatitis. (C) Necrotic tail of pancreas surrounded by exudate. (D) Inflammatory exudate is seen around the right kidney in acute pancreatitis.

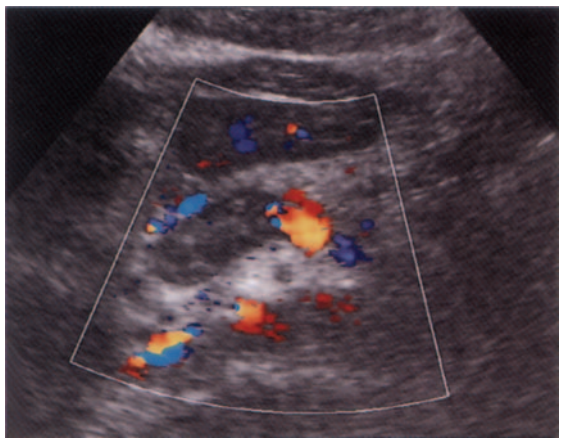
(Continued)

and has been implicated in the cause of recurrent pancreatitis.

Management of acute pancreatitis

While ultrasound is useful in demonstrating associated gallstones, biliary sludge and fluid collections, CT or MRI demonstrates the complications of acute pancreatitis with greater sensitivity and specificity. Localized areas of necrotic pancreatic tissue can be demonstrated on contrast-enhanced CT, together with vascular complications, such as thrombosis.

MRCP or CT is used to demonstrate the main pancreatic duct and its point of insertion into the common bile duct. Anomalous insertions are asso-



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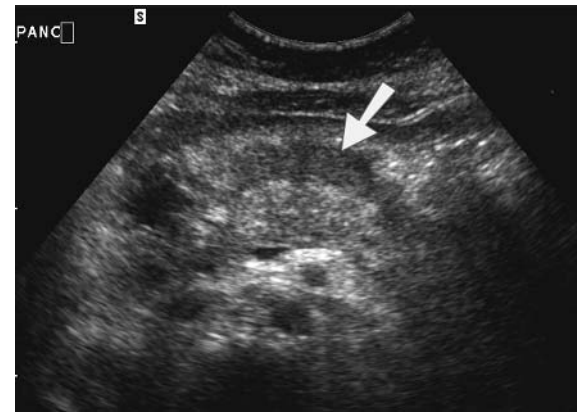

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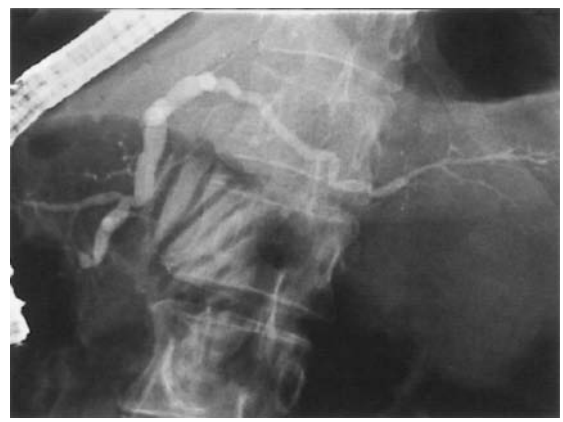

G

Figure 5.3 cont'd (E) Splenic and portal vein thrombosis is a complication of pancreatitis. (F) A dilated pancreatic duct (arrow) filled with blood in haemorrhagic pancreatitis. (G) ERCP: a patient with chronic pancreatitis has a dilated proximal pancreatic duct.

ciated with pancreatitis, due to the reflux of bile into the pancreatic duct. ERCP, which is more invasive and subject to potential complications, is generally reserved for circumstances which require the removal of stones, alleviating the need for surgery, and in the placement of stents in the case of strictures.[6]

Pancreatitis can be difficult to treat, and management consists of alleviating the symptoms and removing the cause where possible. Patients with gallstone pancreatitis do well after cholecystectomy, but if the gallbladder is not removed recurrent attacks of increasingly severe inflammation occur in up to a third of patients.

Pseudocysts which do not resolve spontaneously may be drained percutaneously under ultrasound or CT guidance, or, depending on the site of the collection, a drain may be positioned endoscopically from the cyst into the stomach.[7]

Pseudocyst formation may cause thrombosis of the splenic vein, spreading to the portal and mesenteric veins in some cases. Other vascular complications include splenic artery aneurysm, which may form as a result of damage to the artery by the pseudocyst.

Surgery to remove necrotized or haemorrhagic areas of pancreatic tissue may be undertaken in severe cases.

Chronic pancreatitis

Patients with acute pancreatitis are at risk of repeated inflammatory episodes which eventually develop into chronic inflammation. The most common cause is alcohol abuse. In other cases, chronic pancreatitis has a gradual onset which does not seem to be associated with previous acute attacks.

The normal pancreatic tissue is progressively replaced by fibrosis, which may encase the nerves in the coeliac plexus, causing abdominal pain, particularly post-prandially. The patient has fatty stools (steatorrhoea) due to malabsorption, as there is a decreased capacity to produce the digestive enzymes.

Diagnosis of chronic pancreatitis can be difficult, especially in the early stages.[8] Serum enzyme levels are less elevated than in acute disease (if at all). ERCP, which detects abnormalities of the ductal system in the early stages, is increasingly contraindicated due to the risk of aggravating the pancreatitis. MRCP is promising, but is limited in assessing the smaller side ducts. Endoscopic ultrasound is currently a sensitive and accurate modality in assessing both the ductal system and the pancreatic tissue.

Ultrasound appearances

The pancreas becomes abnormally hyperechoic (Fig. 5.4A). This should not be confused with the normal increase in echogenicity with age. The gland may be atrophied and lobulated and the main pancreatic duct is frequently dilated and ectatic,[9] with a beaded appearance.

Calcification may be identified in the pancreatic tissue, both on ultrasound and on a plain X-ray, and there may be stones in the duct. (Generally speaking, strong shadows are cast from the calcific foci, but small flecks may be too small to shadow) (Fig 5.4 B, C).

As with acute inflammation, CT is the method of choice for demonstrating the complications of chronic pancreatitis.

Obstruction of the duct can cause pseudocyst formation, and other complications include biliary obstruction and portal/splenic vein thrombosis.

MALIGNANT PANCREATIC DISEASE

Pancreatic carcinoma

Clinical features and management

Carcinoma of the pancreas is a major cause of cancer-related death. It carries a very poor prognosis with less than 5% 5 year survival,[10] related to its late presentation.

The presenting symptoms depend on the size of the lesion, its position within the pancreas and the extent of metastatic deposits. Most pancreatic carcinomas (60%) are found in the head of the pancreas,[11] and patients present with the associated symptoms of jaundice due to obstruction of the common bile duct (Fig. 5.5). Carcinomas located in the body or tail of pancreas do not cause obstructive jaundice.

The majority (80%) of pancreatic cancers are ductal adenocarcinomas, most of which are located in the head of pancreas. The rest comprise a mixed bag of less common neoplasms and endocrine tumours.

Endocrine tumours, which originate in the islet cells of the pancreas, tend to be either insulinomas (generally benign) or gastrinomas (malignant). These present with hormonal abnormalities while the tumour is still small and are more amenable to detection by intraoperative ultrasound than by conventional sonography.

Mucin-secreting tumours (Fig. 5.5E), which appear predominantly cystic on ultrasound, tend to be located in the body or tail of pancreas and follow a much less aggressive course than adenocarcinomas, metastasizing late. These tumours, though comparatively rare, have a much higher curative rate with surgery.[12]

Metastatic deposits from primary pancreatic adenocarcinoma occur early in the course of the disease, and 80% of patients already have nodal disease or distant metastases in the lungs, liver or bone by the time the diagnosis is made, which accounts for the poor prognosis.

Surgical removal of the carcinoma by partial pancreaticoduodenectomy, the Whipples procedure, is potentially curative but only 20% of patients have a tumour which is potentially resectable, and the 5-year survival rate following resection is less than

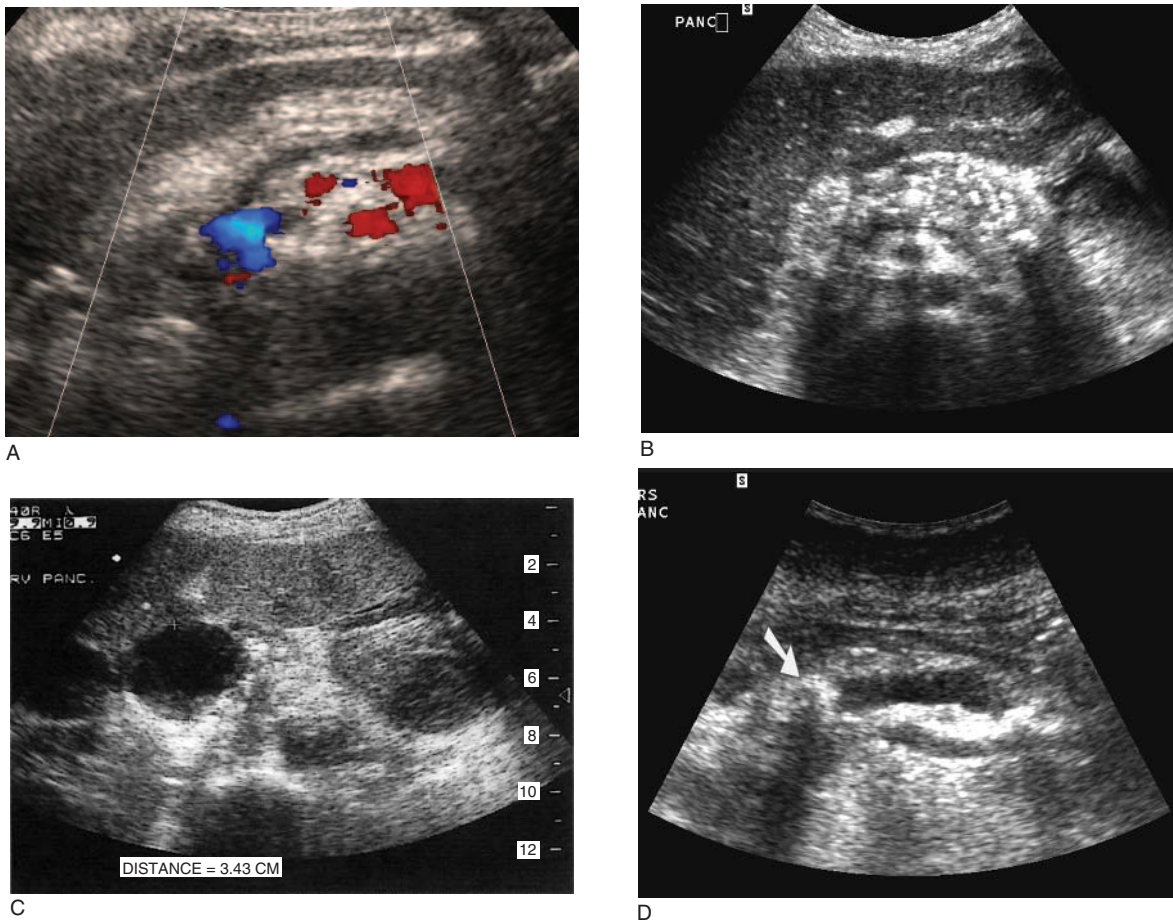

Figure 5.4 (A) Chronic pancreatitis in a patient with alcoholic cirrhosis; the pancreas is hyperechoic compared with the liver and has a heterogeneous texture with a lobulated outline. (B) Calcification of the pancreas in hereditary pancreatitis. (C) A cycle of acute on chronic pancreatitis, with pseudocysts and considerable calcification. (D) A stone (arrow) is obstructing the main pancreatic duct.

5%.[13] Over 70% of patients die from hepatic metastases within 3 years postoperatively.[14]

Differential diagnoses of pancreatic masses must always be considered (Table 5.2); focal lesions in the pancreas may represent inflammatory rather than malignant masses. An ultrasound-guided biopsy is sometimes useful in establishing the presence of adenocarcinoma if the biopsy is positive, but the sensitivity of this procedure is relatively low.[15] The value of a negative biopsy is dubious because of the inflammatory element surrounding many carcinomas.

Endosonography-guided biopsy, however, has high sensitivity and specificity for diagnosing pancreatic cancer, and is also useful in patients with a previous negative biopsy in whom malignancy is suspected.[16] ERCP may also be used to insert a palliative stent in the common bile duct, to relieve biliary obstruction.

The detection of a pancreatic carcinoma by ultrasound is usually followed by a CT scan for staging purposes as this will demonstrate invasion of peripancreatic fat, vascular involvement and lymphadenopathy.[16]

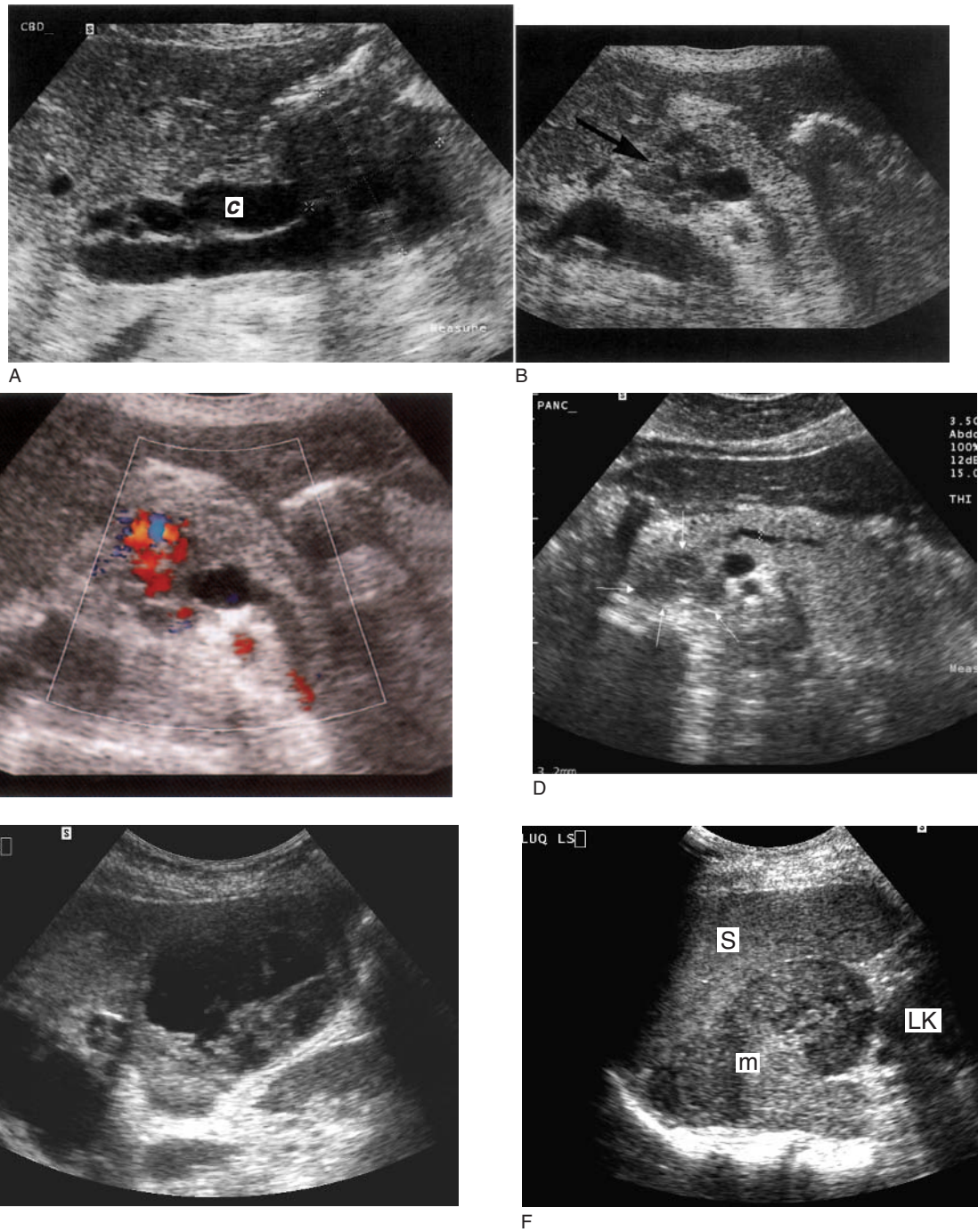

Figure 5.5 (A) The common bile duct, c, is obstructed by a large hypoechoic solid mass at its lower end (calipers), which is a carcinoma in the head of the pancreas. (B) TS through the head of the pancreas, which is swollen by a hypoechoic adenocarcinoma (arrow). (C) The tumour in (B) displays considerable vascularity on colour Doppler. (Note the colour sensitivity setting has been reduced to accommodate this, so eliminating low-velocity flow from the splenic vein.) (D) Tumour in the head of the pancreas (arrows), confirmed by CT. (E) Complex cystic mass in the head of the pancreas, confirmed as a cystadenocarcinoma. (F) A complex mass (m) between the spleen (S) and the left kidney is a large carcinoma of the tail of the pancreas.

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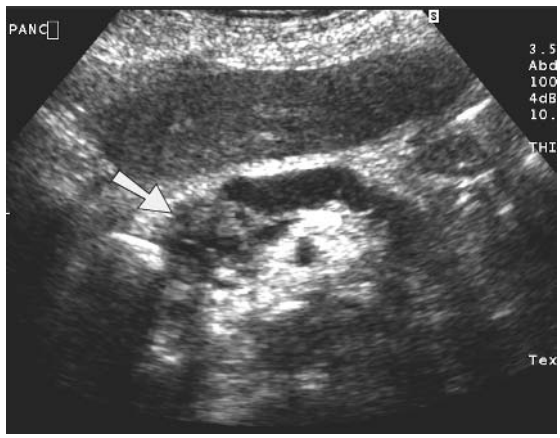

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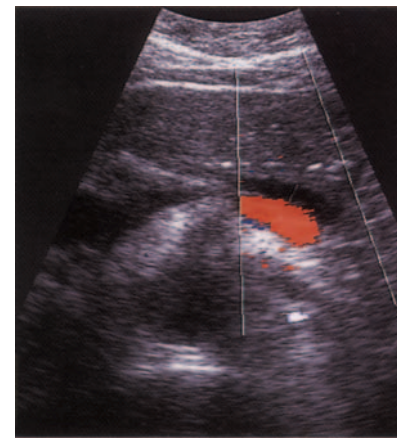

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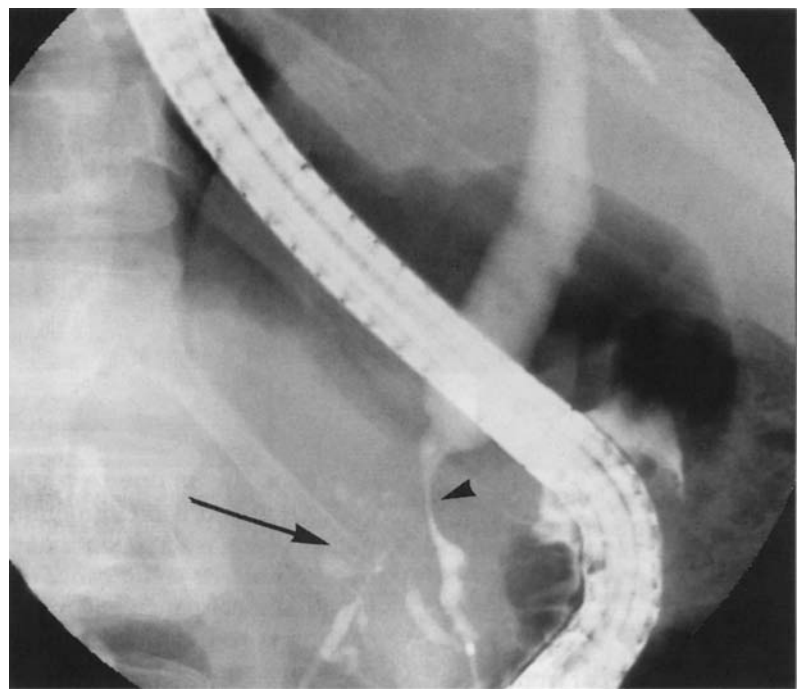

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Figure 5.5 cont'd (G) Dilated pancreatic duct due to a carcinoma in the head (arrow). (H) Colour Doppler helps to differentiate the dilated pancreatic duct (measured), which does not contain flow, from the splenic vein posterior to the duct. (I) Endoscopic retrograde cholangiopancreatography (ERCP) demonstrating a long stricture of the pancreatic duct (arrow) involving the side branches, in a large pancreatic carcinoma. The CBD is compressed (arrowhead) by nodes, causing biliary dilatation. A palliative stent was inserted.

Table 5.2 Differential diagnoses of focal pancreatic masses

Mass	Characteristics
Solid	
Adenocarcinoma	Hypoechoic, usually in the head of pancreas
Focal acute pancreatitis	Hypoechoic. Clinical history of pancreatitis
Focal chronic pancreatitis	Hyperechoic, sometimes with calcification. History of pancreatitis
Endocrine tumour	Less common. Small, hypoechoic, well-defined
Metastases	Late manifestation, widespread disease
Cystic[17]	
Pseudocyst	History of pancreatitis
Mucinous tumour	Less common than adenocarcinoma, tending to form in the body or tail of pancreas. Favourable prognosis following resection
Necrotic or haemorrhagic tumour	
Simple cyst	Rare. Exclude polycystic disease by scanning the liver and kidneys

Ultrasound appearances of pancreatic carcinoma

The adenocarcinoma, which comprises 80% of pancreatic neoplasms, is a solid tumour, usually hypoechoic or of mixed echogenicity, with an irregular border (Fig. 5.5). Because the mass is most frequently located in the head of the pancreas, which lies behind the duodenum, it may be difficult to identify at first.

Endocrine tumours, which arise from the islet cells in the pancreas, include insulinomas, which are benign, and gastrinomas, which are more often malignant. They are usually hypoechoic, well-defined and exhibit a mass effect, often with a distally dilated main pancreatic duct. They are generally smaller at presentation than adenocarcinomas, and tend to arise in the body or tail of pancreas. Up to 40% of these tumours go undetected by both transabdominal ultrasound and CT, with endoscopic ultrasound and laparoscopic ultrasound having the highest detection rates for insulinomas. Gastrinomas tend to be multiple and may also be extrapancreatic.

A small proportion of pancreatic cancers contain an obvious fluid content. Cystadenocarcinomas, which produce mucin, are similar in acoustic appearance to a pseudocyst, but unlike a pseudocyst, a mucinous neoplasm is not associated with a history of pancreatitis.

It is also possible within a lesion to see areas of haemorrhage or necrosis which look complex or fluid-filled. Calcification is also seen occasionally within pancreatic carcinomas.[18]

The adenocarcinoma is vascular and high-velocity arterial flow may be identified within it in many cases (Fig. 5.5C, F).

The pancreatic duct distal to the mass may be dilated. It may, in fact, be so dilated that it can be initially mistaken for the splenic vein. The walls of the duct, however, are usually more irregular than the smooth, continuous walls of the splenic vein. Colour Doppler is useful in confirming the lack of flow in the duct and in identifying the vein behind it (Fig. 5.5G, H).

Secondary ultrasound findings in pancreatic adenocarcinoma

The most obvious secondary feature of carcinoma of the head of pancreas is the dilated biliary system (see *Obstructive jaundice*, Chapter 3). In a recent series of 62 pancreatic cancers, biliary dilatation occurred in 69%, pancreatic duct dilatation in 37% and the *double duct sign* (pancreatic and biliary duct dilatation) in 34% of patients.[18]

Although the gallbladder is frequently dilated with no visible stones, this is not always the case; incidental gallstones may be present, causing chronic inflammation which prevents the gallbladder from dilating. For this reason it is imperative that the common duct is carefully traced down to the head of pancreas to identify the cause of obstruction.

A thorough search for lymphadenopathy and liver metastases should always be made. CT is usually the method of choice for staging purposes. If the mass is large, it is not possible to differentiate whether it arises from the ampulla of Vater or the

head of pancreas. This differentiation, however, is usually academic at this stage.

Colour Doppler can demonstrate considerable vascularity within the mass and is also important in identifying vascular invasion of the coeliac axis, superior mesenteric artery, hepatic, splenic and/or gastroduodenal arteries and of the portal and splenic veins, a factor which is particularly important in assessing the suitability of the tumour for curative resection. The recognition of involvement of peripancreatic vessels by carcinoma with colour Doppler, together with the ultrasound assessment of compression or encasement of these vessels, has been found to be highly sensitive and specific (79% and 89%) for diagnosing unresectability,[19] thus the need for further investigative procedures such as CT may be avoided, particularly in cases of large tumours.[20]

Pancreatic metastases

Pancreatic metastases may occur from breast, lung and gastrointestinal tract primary tumours. They are relatively uncommon on ultrasound (Fig. 5.6), simply because they are a late manifestation in patients who already have known, widespread disease and in whom investigations are generally considered unnecessary.

Widespread metastatic disease can be demonstrated on ultrasound, particularly in the liver, and there is often considerable epigastric lymphadenopathy, which can be confused with the appearances of pancreatic metastases on the scan.

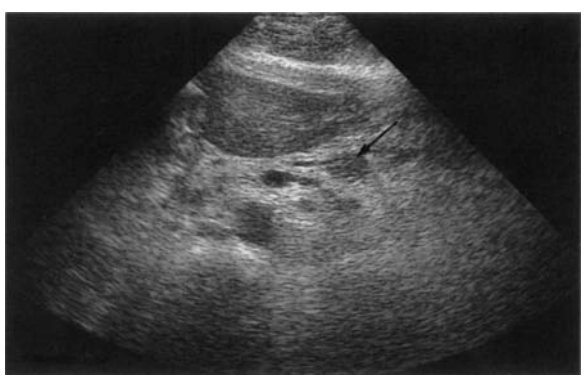

Figure 5.6 Metastatic deposit from primary breast carcinoma in the body of the pancreas (arrow).

Pathology of the pancreas, both benign and malignant, can affect the adjacent vasculature by compression, encasement or thrombosis. **Doppler** of the splenic, portal and superior mesenteric veins is useful in demonstrating the extent of vascular complication when pancreatic abnormalities are suspected.

BENIGN FOCAL PANCREATIC LESIONS

Focal fatty sparing of the pancreas

The uncinate process and ventral portion of the head of pancreas may sometimes appear hypoechoic in comparison with the rest of the gland (Fig. 5.7). This is due to a relative lack of fatty deposition and is often more noticeable in older patients, in whom the pancreas is normally hyperechoic. Its significance lies in not confusing it with a focal pancreatic mass. The area of fatty sparing is well-defined, with no enlargement or mass effect, and is regarded as a normal variation in the ultrasound appearances. If doubt exists, CT will differentiate fatty sparing from true neoplasm.[21]

Focal pancreatitis

Inflammation can affect the whole, or just part of the gland. Occasionally, areas of hypoechoic, focal acute or chronic pancreatitis are present (see *Pancreatitis,* above). These are invariably a diagnostic dilemma, as they are indistinguishable on ultrasound from focal malignant lesions (Fig. 5.8). Factors which point towards inflammation include

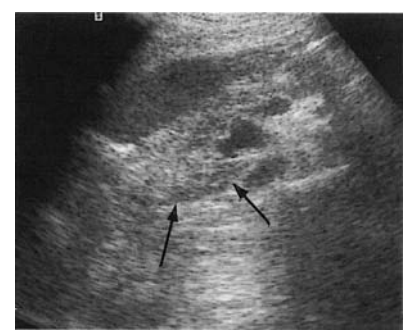

Figure 5.7 The uncinate process is relatively hypoechoic (arrows) because of fatty sparing.

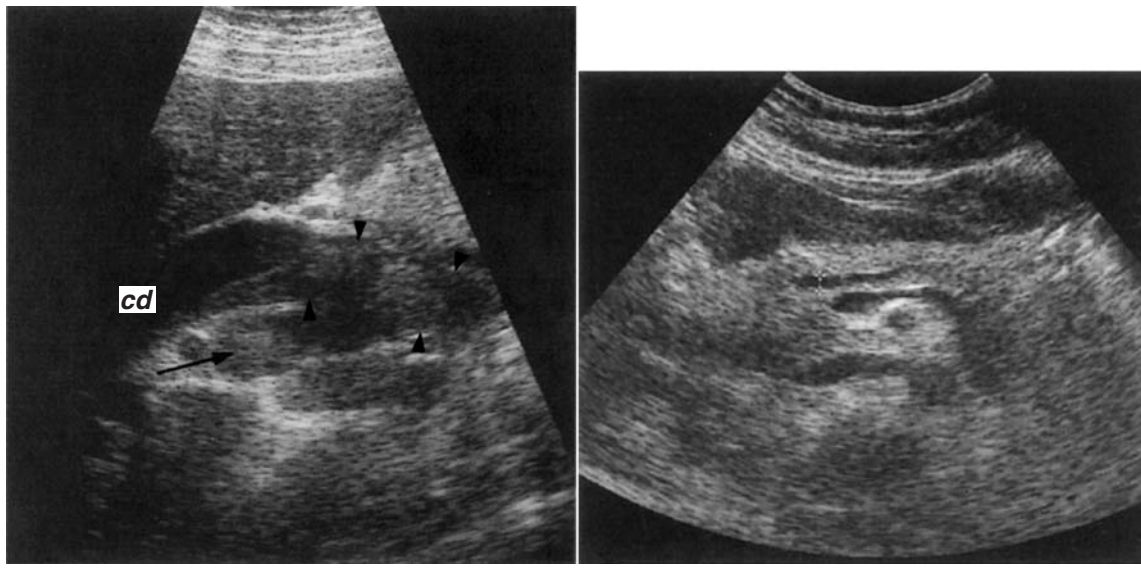

Figure 5.8 (A) Focal acute pancreatitis in the head of the pancreas. The CBD is obstructed by a hypoechoic mass in the head, with blood clots and debris within the duct. The differential diagnosis was malignancy. (B) The same patient 8 months later. The acute inflammation has resolved, the obstruction is relieved and the pancreas now appears hyperechoic with a mildly dilated duct, consistent with chronic pancreatitis.

a previous history of pancreatitis and a normal CA 19–9 tumour marker level.

Because malignant lesions are frequently surrounded by an inflammatory reaction, biopsy is also of questionable help in differentiation of focal benign and malignant lesions.

Cysts

Benign cysts in the pancreas are rare (Fig. 5.9) and tend to be associated with other conditions such as polycystic disease, cystic fibrosis or von Hippel–Lindau disease (an autosomal dominant disease characterized by pancreatic and renal cysts, renal carcinoma, phaeochromocytoma and/or haemangioblastomas in the cerebellum and spine). The presence of a cystic mass in the absence of these conditions should raise the suspicion of one of the rarer types of cystic carcinoma, or a pseudocyst associated with acute pancreatitis.

TRAUMA OF THE PANCREAS

The pancreas is particularly vulnerable to 'blunt' trauma in road traffic accidents, in which the upper abdomen is thrown against the seat belt, resulting in laceration, often at the neck of the pancreas. The duct may be ruptured, with consequent leakage of

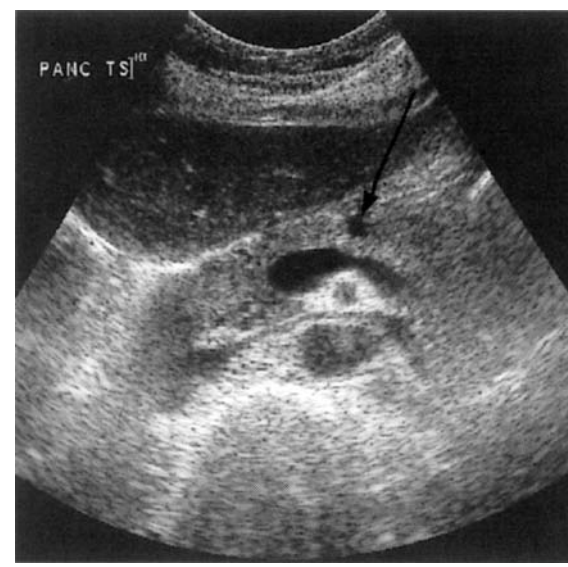

Figure 5.9 Tiny cyst in the body of the pancreas. This was confirmed on CT and remained stable over a period of 2 years.

pancreatic juice into the abdominal cavity and severe cases result in complete pancreatic transection with pancreatic ascites.

The release of pancreatic enzymes triggers pancreatitis and/or peritonitis, with the gland appearing enlarged and hypoechoic.

Ultrasound may be helpful in localizing a collection, but will not differentiate pancreatic secretions from haematoma. CT is the method of choice in cases of suspected pancreatic trauma, although even here the signs of injury can be surprisingly subtle considering the damage.[22]

PANCREATIC TRANSPLANT

In patients with insulin-dependent diabetes mellitus with end-stage renal disease, simultaneous pancreatic and kidney transplant is a successful treatment which improves the quality of life and the survival of the patients. Typically such patients also have severe complications, such as retinopathy and vascular disease, which may be stabilized, or even reversed, by transplantation.

Simultaneous pancreas and kidney transplantation now has a 1-year graft survival of almost 90% due to improved organ preservation techniques, surgical techniques and immunosuppression.[23]

The transplanted kidney is placed in the iliac fossa with the pancreas on the contralateral side. The donor kidney is transplanted in as usual, with anastomoses to the recipient iliac artery and vein. The pancreatic vessels are anastamosed to the contralateral iliac vessels.

The pancreatic secretions are primarily by enteric drainage, as the previous method of bladder drainage was associated with an increased incidence of urologic complications such as urinary tract infection, haematuria or reflux pancreatitis.[24]

Postoperative monitoring of the pancreatic transplant is difficult, on both clinical and imaging grounds. No one imaging modality has proved without limitations and a combination of ultrasound, CT, MRI, angiography and nuclear medicine may be required.[25] Postoperative complications include thrombosis, infection, inflammation, anastomotic leaks and rejection. Localized postoperative bleeding usually resolves spontaneously.

Ultrasound appearances

The donor pancreas is usually situated in the iliac fossa but can be placed more centrally, particularly if a renal transplant has also been performed.

Ultrasound is limited in its ability to assess the transplanted pancreas, even if it can be located amongst the bowel loops. The lack of an adjacent reference organ, such as the liver, makes assessment of its echogenicity subjective, and therefore subtle degrees of inflammation are difficult to detect. Fluid collections are frequently concealed beneath bowel and, when identified, their appearance is non-specific. Contrast CT is more successful in detecting anastomotic leaks and collections, and is usually used for guided aspiration.

Colour Doppler should display perfusion throughout the pancreas and the main vessels may be traced to their anastomoses, depending on overlying bowel (Fig. 5.10). Neither CT nor ultrasound is particularly helpful in evaluating rejection, and it is difficult to differentiate transplant pancreatitis from true rejection. The Doppler resistance index does not correlate with a rejection process and has not been found useful. MRI has been found to display more positive findings in pancreatic rejection than other imaging modalities.

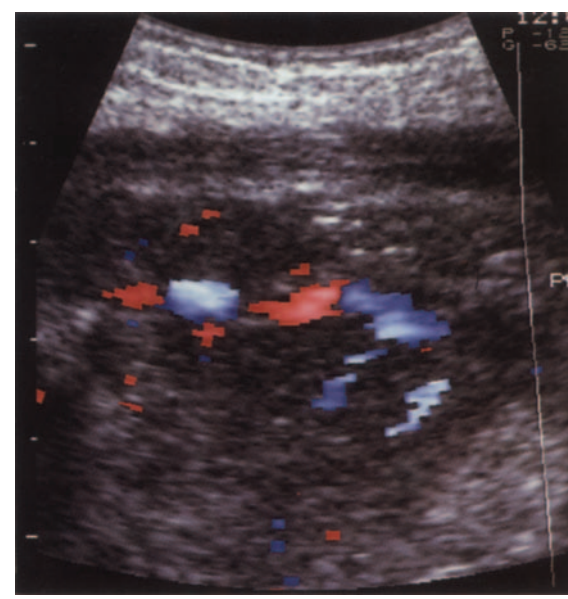

Figure 5.10 The transplanted pancreas may be difficult to identify in the iliac fossa. The main artery is seen here running through the body of the pancreas.

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Chapter 6

The spleen and lymphatic system

CHAPTER CONTENTS

THE SPLEEN—NORMAL APPEARANCES AND TECHNIQUE

The spleen normally lies posterior to the splenic flexure and stomach, making an anterior approach almost invariably unsuccessful due to overlying bowel gas. The spleen should therefore be approached from the left lateral aspect: coronal and transverse sections may be obtained with the patient supine by using an intercostal approach. Gentle respiration is frequently more successful than deep inspiration, as the latter brings the lung bases downwards and may obscure a small spleen altogether.

Lying the patient decubitus, left side raised, may also be successful but sometimes has the effect of causing the gas-filled bowel loops to rise to the left flank, once again obscuring the spleen. A slightly posterior approach may overcome this.

Ultrasound appearances

The normal spleen has a fine, homogeneous texture, with smooth margins and a pointed inferior edge. It has similar echogenicity to the liver but may be slightly hypo- or hyperechoic in some subjects.

Sound attenuation through the spleen is less than that through the liver, requiring the operator to 'flatten' the time gain compensation controls in order to maintain an even level of echoes throughout the organ. The main splenic artery and vein and their branches may be demonstrated at the splenic hilum (Fig. 6.1).

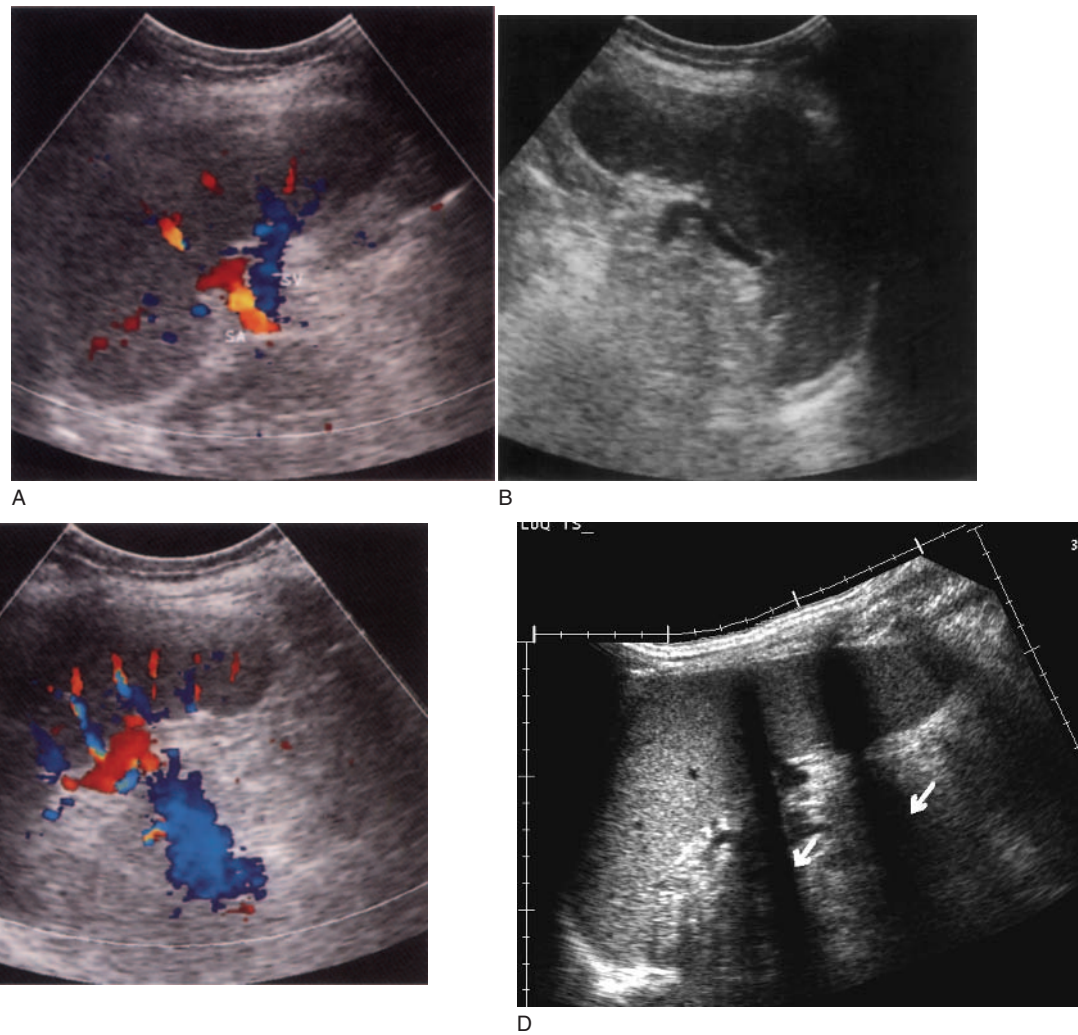


Figure 6.1 (A) Left coronal view of the normal spleen demonstrating the main splenic artery and vein at the hilum. (B) Transverse section (TS) demonstrating the splenic vein at the hilum. (C) By increasing the Doppler sensitivity, the intrasplenic perfusion can be demonstrated. (D) An elongated or enlarged spleen can be displayed more fully using an extended field of view. Shadowing from the ribs (arrows) is evident.

The spleen provides an excellent acoustic window to the upper pole of the left kidney, the left adrenal gland and the tail of the pancreas.

Splenic variants

Spleen size and shape are both highly variable, with a gradual age-related decrease in volume. A splenic length of below 12 cm is generally considered normal, although this is subject to variation in shape and the plane of measurement used.

Rarely, the diaphragmatic surface of the spleen may be lobulated, or even completely septated. This appearance may give rise to diagnostic uncertainty, and Doppler may be helpful in establishing the vascular supply, and differentiating this from other masses in the left upper quadrant (LUQ), or from scarring or infarction in the spleen.

The spleen may lie in an ectopic position, in the left flank or pelvis, or posterior to the left kidney. The ectopic (or wandering) spleen is situated on a long pedicle, allowing it to migrate within the abdomen.

The significance of this rare condition is that the pedicle may twist, causing the patient to present acutely with pain from splenic torsion. Ultrasound demonstrates the enlarged, hypoechoic organ in the abdomen, with the absence of the spleen in its normal position.

Splenomegaly

Enlargement of the spleen is a highly non-specific sign associated with numerous conditions, the most common being infection, portal hypertension, haematological disorders and neoplastic conditions (Table 6.1).

As with the liver, measurement of splenic volume is usually considered inaccurate due to variations in shape, and not reproducible. However, the length of the spleen is an adequate indicator of size for most purposes and provides a useful baseline for monitoring changes in disease status. The length of the normal adult spleen is less than 12 cm.

The spleen enlarges downwards and medially. Its inferior margin becomes rounded, rather than pointed, and may extend below the left kidney (Fig. 6.2).

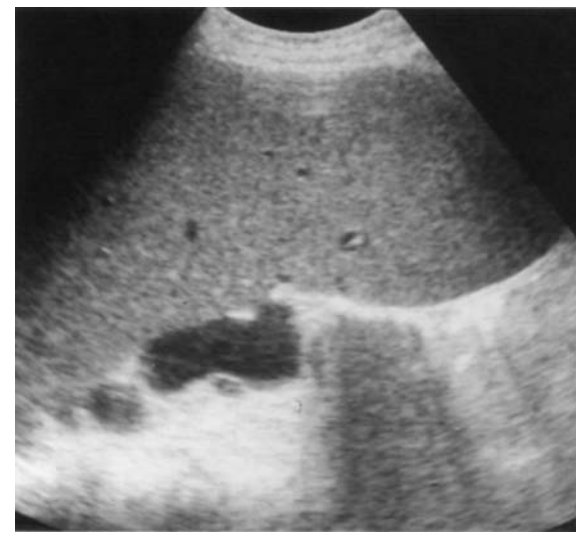
Although the aetiology of splenomegaly may not be obvious on ultrasound, the causes can be narrowed down by considering the clinical picture and by identifying other relevant appearances in the abdomen. Splenomegaly due to portal hypertension, for example, is frequently accompanied by other associated pathology such as cirrhotic liver changes, varices (Fig. 6.2B) or ascites (see Chapter 4).

Table 6.1 Examples of causes of splenomegaly

- Portal hypertension
- Acute or chronic systemic infection, e.g. hepatitis, AIDS, infectious mononucleosis, sepsis
- Haemolytic anaemia, sickle cell disease, thalassaemia, pernicious anaemia, spherocytosis
- Malignancy—leukaemia, Hodgkin's and non-Hodgkin's lymphoma, myeloproliferative disorders
- Storage disorders
- Immunological diseases

Splenunculi

In around 10% of the population, a small accessory spleen, or splenunculus, may be located at the splenic hilum. These small, well-defined ectopic



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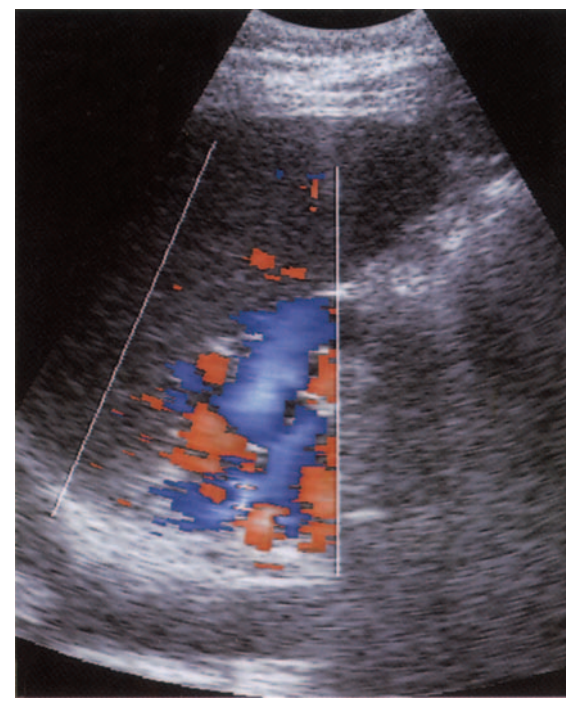

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Figure 6.2 (A) Splenomegaly in portal hypertension. The inferior splenic margin is blunted, descending below and medial to the left kidney. (B) Varices at the splenic hilum in portal hypertension.

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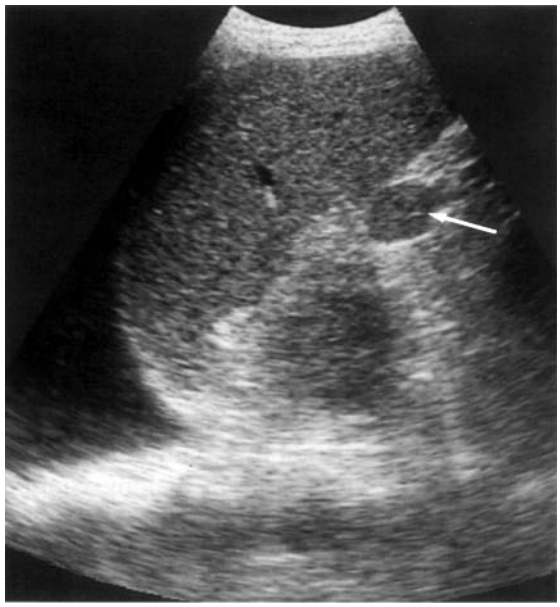

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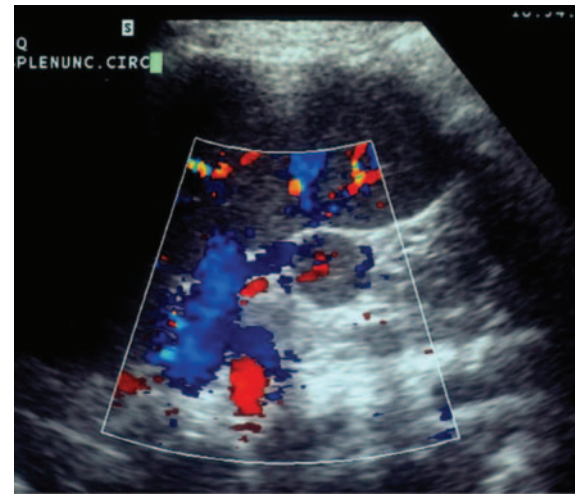

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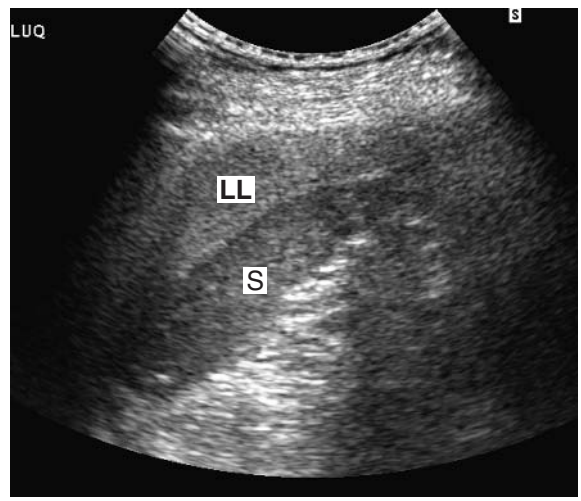

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Figure 6.2 cont'd (C) A splenunculus (arrow) at the hilum of a mildly enlarged spleen. (D) The circulation of the splenunculus derives from the main splenic artery and drains into the main splenic vein. (E) The left lobe of the liver, LL, extends across the abdomen and above the spleen, S, in hepatomegaly, giving the appearance of a well-defined splenic mass.

nodules of splenic tissue (Fig. 6.2C) rarely exceed 2 cm in diameter. Splenunculi enlarge under the same circumstances as those which cause splenomegaly and may also hypertrophy in post-splenectomy patients.

The importance of recognizing these lies in differentiating them from lymph nodes, left adrenal nodules or masses in the tail of pancreas. Colour Doppler may identify the vascular supply as being common to the main spleen[1] (Fig. 6.2D).

Pitfalls in scanning the spleen

- In hepatomegaly, the left lobe of liver may extend across the abdomen, indenting the spleen. This can give the appearance of a

homogeneous, intrasplenic 'mass' when the spleen is viewed coronally (Fig. 6.2D). A transverse scan at the epigastrium should demonstrate the extent of left hepatic enlargement and confirm its relationship to the spleen.

- Splenunculi may be mistaken for enlarged lymph nodes at the splenic hilum. Colour Doppler can confirm the vascular supply is shared by the spleen.
- The normal tail of pancreas may mimic a perisplenic mass.
- A left adrenal mass, or upper pole renal mass, may indent the spleen making it difficult to establish the origin of the mass.

MALIGNANT SPLENIC DISEASE

Lymphoma

Lymphoma is the most common malignant disease affecting the spleen. Lymphomas comprise a number of diseases, all malignant, which affect the lymphocytes. Malignant cells can infiltrate the spleen, lymph nodes, bone marrow and thymus and can also involve the liver, gastrointestinal tract, kidney and other organs. Approximately 3% of malignant diseases are lymphomas.

Splenic involvement may be found in up to 60% of lymphomas as a result of dissemination of the disease. Primary splenic lymphoma, limited to the spleen, is very rare, and accounts for less than 1% of lymphomas. There are two main groups: Hodgkin's and non-Hodgkin's lymphomas.

Clinical features and management

Patients may present with a range of non-specific symptoms which include lymph node enlargement, anaemia, general fatigue, weight loss, fever, sweating and infections associated with decreased immunity.

If the disease has spread to other organs, these may produce symptoms related to the organs in question.

Prognosis depends upon the type of the disease, which must be determined histologically, and its stage. Both ultrasound and CT may be used in staging: ultrasound demonstrates splenic involvement with greater sensitivity than CT, and CT is superior in demonstrating para-aortic and iliac lymph nodes.[2] Bone scintigraphy and MRI are further supplementary techniques in staging.

Depending upon the type of lymphoma, chemotherapy regimes may be successful and, if not curative, can cause remission for lengthy periods. High-grade types of lymphoma are particularly aggressive with a poor survival rate.

Ultrasound appearances

The range of possible ultrasound appearances in lymphoma is varied (Fig. 6.3). In many cases the spleen is not enlarged and shows no acoustic abnormality. In a study of 61 patients with Hodgkin's disease involving the spleen, the organ was usually normal in size and showed no acoustic abnormality in 46% of cases.[3]

Lymphoma may produce a diffuse splenic enlargement with normal, hypo- or hyperechogenicity. Focal lesions may be present in up to 16% of lymphomas.[4,5] They tend to be hypoechoic and may be single or multiple, and of varying sizes. In larger lesions the margins may be ill-defined and the echo contents vary from almost anechoic to heterogeneous, often with increased through-transmission. In such cases, they may be similar in appearance to cysts, however, the well-defined capsule is absent in lymphoma, which has a more indistinct margin.[6] Smaller lesions may be hyperechoic or mixed. Tiny lymphomatous foci may affect the entire spleen, making it appear coarse in texture.

Lymphadenopathy may be present elsewhere in the abdomen. If other organs, such as the kidney or liver, are affected, the appearances of mass lesions vary but are commonly echo-poor or of mixed echo pattern.

A differential diagnosis of metastases should be considered in the presence of multiple solid hypoechoic splenic lesions, but most cases are due to lymphoma.[7]

Metastases

Metastatic deposits occur in the spleen much less commonly than in the liver. Autopsy reports an incidence of around 10%, although a proportion of

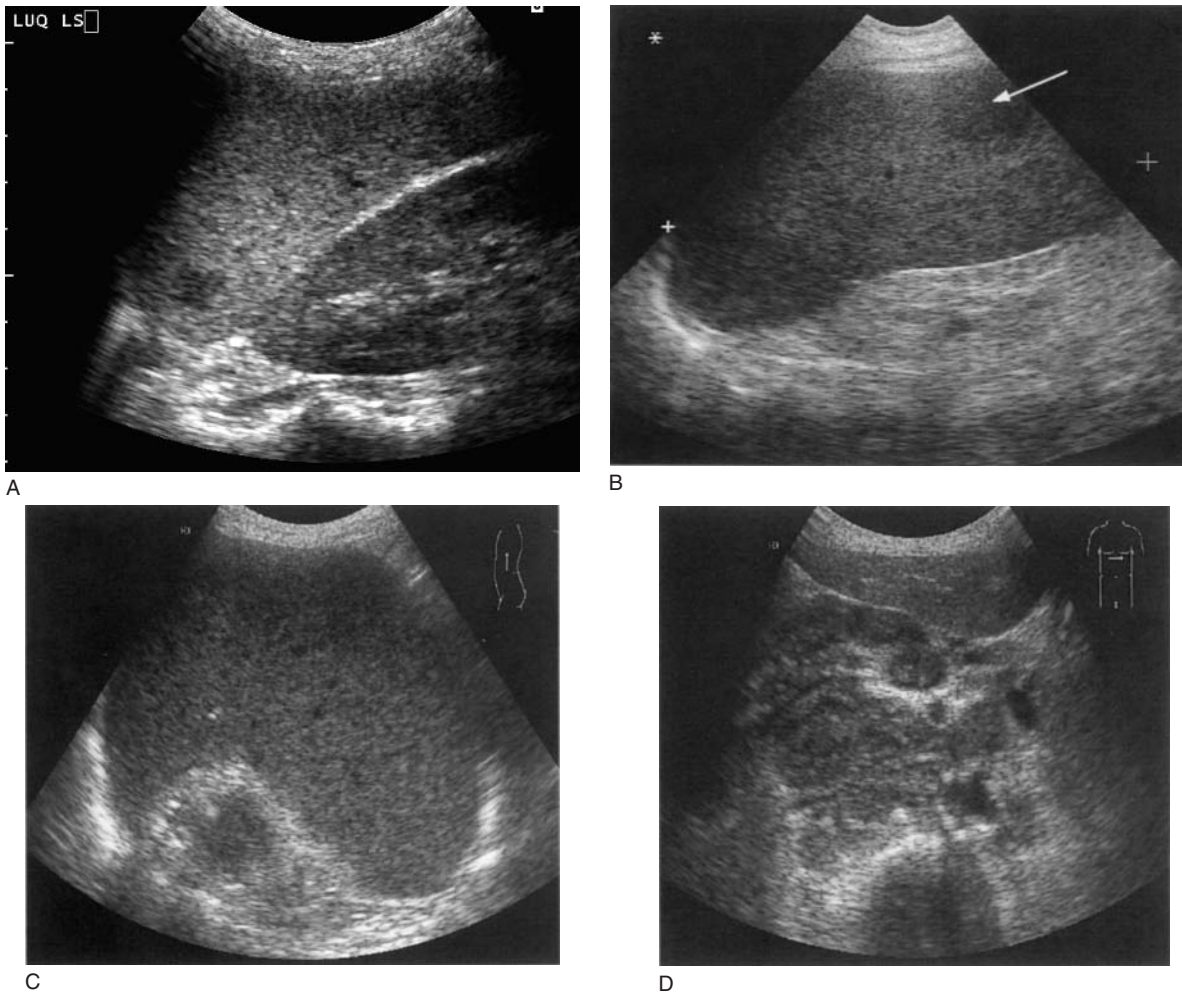

Figure 6.3 Lymphoma: (A) Small, focal lesion in a normal-sized spleen. (B) Enlarged, hyperechoic spleen with a hypoechoic focal lesion (arrow). (C) Enlarged, coarse-textured spleen containing multiple tiny lymphomatous lesions. (D) Extensive lymphadenopathy in the epigastric region.

these are microscopic and not amenable to radiological imaging.

The most commonly found splenic metastases on ultrasound are from lymphoma, but may occur with any primary cancer. Intrasplenic deposits are more likely in later-stage disease and favour melanoma, pulmonary, ovarian or breast primaries.

As with liver metastases, the ultrasound appearances vary enormously, ranging from hypo- to hyperechogenic or of mixed pattern (Fig. 6.4). They may be solitary, multiple or diffusely infiltrative, giving a coarse echo-pattern.[8]

Leukaemia

Leukaemia (literally meaning 'white blood', from the Greek) is characterized by an increased number of malignant white blood cells. Unlike lymphoma, which affects the lymphatic system, leukaemia affects the circulation.

There are two main types, myeloid and lymphoid, both of which can be either acute or chronic.

The bone marrow becomes infiltrated with malignant cells which cause the blood to have increasing levels of immature blood cells.

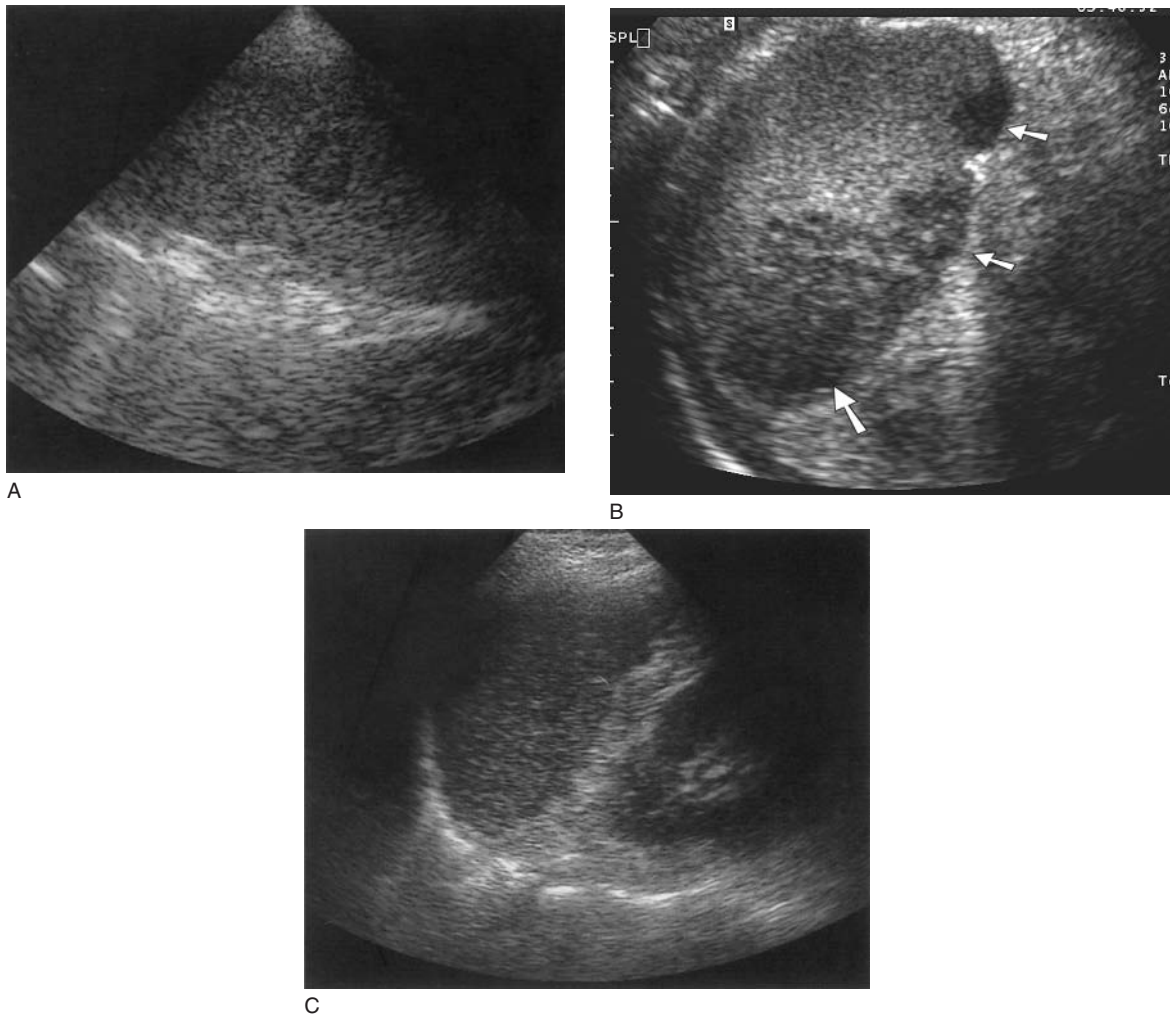

Figure 6.4 (A) Solitary hypoechoic splenic metastasis from melanoma. (B) Metastasic deposits (arrows) in a patient with gastric carcinoma. (C) Disseminated metastases from breast carcinoma affect the spleen, giving it a coarse texture and lobulated outline.

Patients present with fatigue, anaemia, recurrent infections and a tendency to bleed internally. The patient's inability to overcome infections may eventually lead to death. Chemotherapy is successful in curing acute lymphoblastic leukaemia in approximately half the patients, and may induce remission in others. The long-term prognosis is poor for other types of leukaemia, although patients may survive for 10 years or more with the slow-growing chronic lymphocytic leukaemia.

Leukaemia produces diffuse splenic enlargement, but rarely with any change in echogenicity. Abdominal lymphadenopathy may also be present.

BENIGN SPLENIC CONDITIONS

Many benign focal lesions which occur in the spleen are of similar nature and ultrasound appearances to those in the liver. Focal lesions are less common in the spleen, however.

Cysts

Splenic cysts have a relatively low incidence, but are nevertheless the most common benign mass found in the spleen. They demonstrate the usual acoustic characteristics of well-defined capsule, no internal echoes and posterior enhancement (Fig. 6.5). Splenic cysts may occasionally be associated with adult polycystic disease.

Other causes of cystic lesions in the spleen include post-traumatic cysts (liquefied haematoma), hydatid cysts (*Echinococcus granulosus* parasite) or cystic metastases (for example from primary ovarian carcinoma, which may contain mucin).

As with hepatic cysts, haemorrhage may occur, causing LUQ pain (Fig. 6.5B). Large cysts may be resected, in order to avoid rupture.

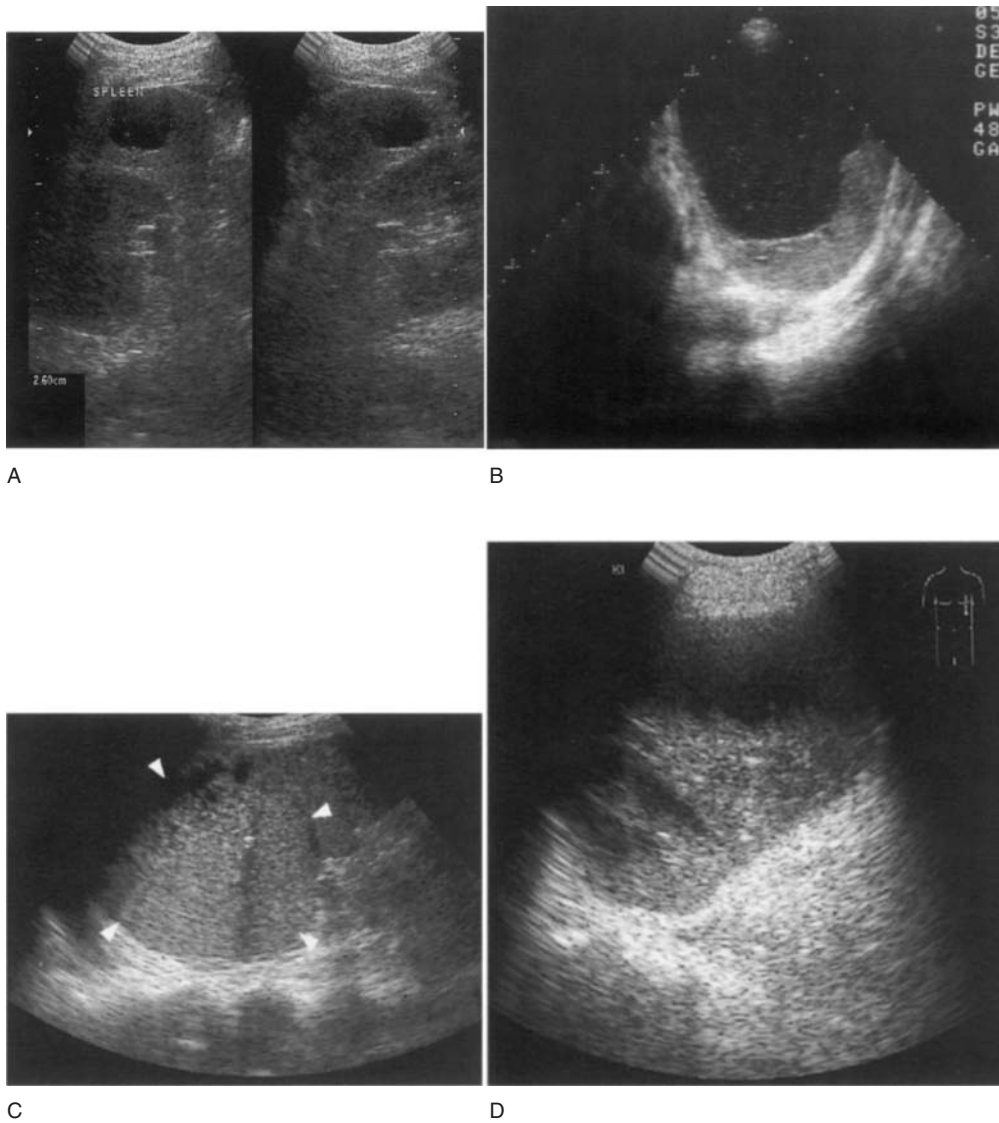

Figure 6.5 (A) Small, simple splenic cyst. (B) Haemorrhage into a splenic cyst causes low-level echoes. (C) Large splenic abscess in an immunosuppressed patient following hepatic transplantation. (D) This abscess, involving the entire spleen, followed a severe episode of empyema. The patient presented, following cholecystectomy, with a spiking temperature.

Haemangioma

The benign haemangioma occurs rarely in the spleen. As in the liver, it is usually hyperechoic and well-defined, though may, rarely, contain cystic areas.[9] Like the hepatic haemangioma, they may pose a diagnostic dilemma as characterization is difficult with ultrasound alone. In cases with a low clinical suspicion of malignancy, such lesions may be followed up with ultrasound, and tend to remain stable in size. Less commonly, haemangiomas may also be multiple.

Abscess

Splenic abscesses are relatively uncommon compared with their incidence in the liver. They usually result from blood-borne bacterial infection, but can also be due to amoebic infection, post-traumatic or fungal infection. Patients with splenomegaly resulting from typhoid fever, malaria and sickle cell disease are particularly predisposed to the formation of multiple pyogenic abscesses in the spleen.

Increasingly splenic abscesses are associated with immunosuppressed patients, patients with AIDS and those on high-dose chemotherapy. Such patients become susceptible to invasive fungal infections which can cause multifocal micro-abscesses in the liver and spleen.[10]

Patients present, as might be expected, with LUQ pain and fever.

The ultrasound appearances are similar to liver abscesses; they may be single or multiple, hyperechoic and homogeneous in the early stages, progressing to complex, fluid-filled structures with increased through-transmission (Fig. 6.5 C, D).

Splenic abscesses are frequently hypoechoic and it may not be possible to differentiate abscess from lymphoma or metastases on ultrasound appearances alone. This applies both in cases of large solitary abscesses and in multifocal micro-abscesses. They may also contain gas, posing difficulties for diagnosis as the area may be mistaken for overlying bowel.

As with liver abscesses, percutaneous drainage with antibiotic therapy is the management of choice for solitary abscesses.

Calcification

Calcification may occur in the wall of old, inactive abscess cavities, forming granulomatous deposits. Other infective processes, particularly in association with AIDS, may cause multiple small calcific foci throughout the spleen and liver (Fig. 6.6).

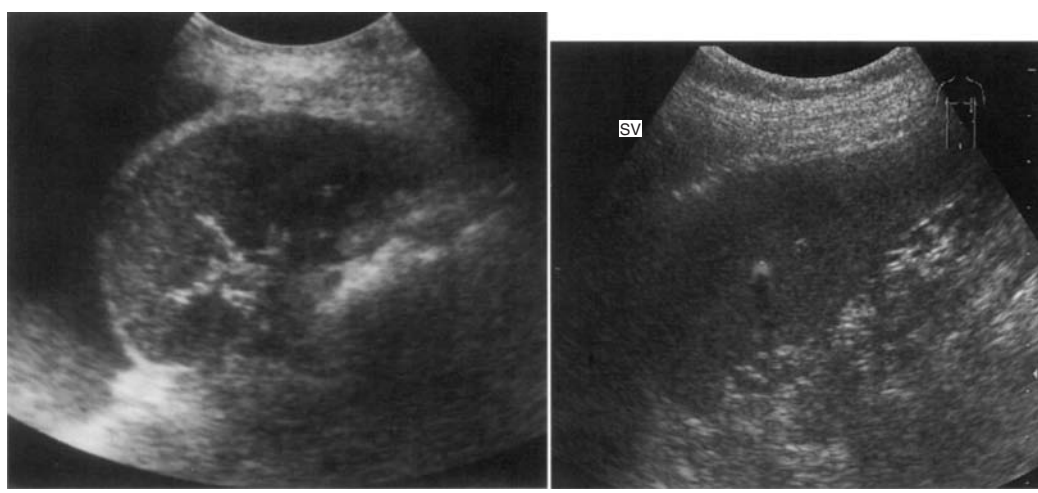

Figure 6.6 (A) Calcification in the spleen in a patient with nephrotic syndrome. Note the left pleural effusion. (B) Small calcified foci in the spleen of a patient with hepatitis.

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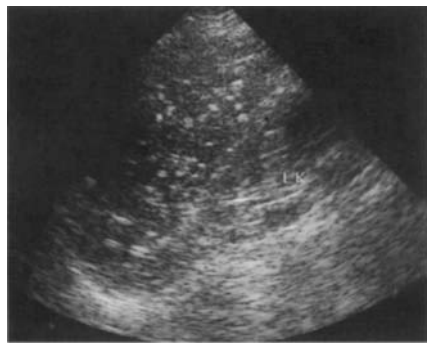

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Figure 6.6 cont'd (C) Multiple granulomata throughout the spleen.

Calcification is also associated with post-traumatic injury and may be seen around the wall of an old, resolving post-traumatic haematoma.

Conditions which predispose to the deposition of calcium in tissues, such as renal failure requiring dialysis, are also a source of splenic calcification.

Haemolytic anaemia

Increased red blood cell destruction, or *haemolysis*, occurs under two circumstances: when there is an abnormality of the red cells, as in sickle cell anaemia, thalassaemia or hereditary spherocytosis, or when a destructive process is at work, such as infection or autoimmune conditions. Fragile red cells are destroyed by the spleen, which becomes enlarged (Fig. 6.7).

Sickle-cell anaemia is most prevalent in the black American and African populations. Progression of the disease leads to repeated infarcts in various organs, including the spleen, which may eventually become shrunken and fibrosed. Patients have (non-obstructive) jaundice because the increased destruction of red blood cells (RBCs) releases excessive amounts of bilirubin into the blood.

Vascular abnormalities of the spleen

Splenic infarct

Splenic infarction is most commonly associated with endocarditis, sickle cell disease and myeloproliferative disorders[11] and also with lymphoma and cancers. It usually results from thrombosis of one or more of the splenic artery branches. Because the spleen is supplied by both the splenic and gastric arteries, infarction tends to be segmental rather than global. Patients may present with LUQ pain, but not invariably.

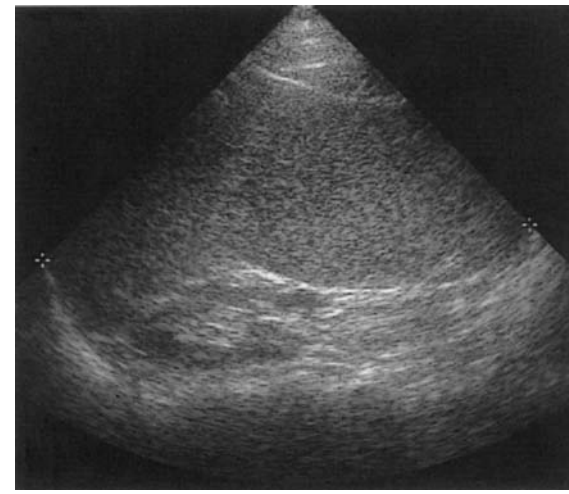


Figure 6.7 Splenomegaly in hereditary spherocytosis.

Initially the area of infarction is hypoechoic and usually wedge-shaped, solitary and extending to the periphery of the spleen (Fig. 6.8 A and B). The lesion may decrease in time, and gradually fibrose, becoming hyperechoic.

It demonstrates a lack of Doppler perfusion compared with the normal splenic tissue. In rare cases of total splenic infarction (Fig. 6.8C), due to occlusion of the proximal main splenic artery, grey-scale sonographic appearances may be normal in the early stages. Although the lack of colour Doppler flow may assist in the diagnosis, CT is the method of choice.

Occasionally infarcts may become infected or may haemorrhage. Sonography can successfully document such complications and is used to monitor their resolution serially. In patients with multiple infarcts, such as those with sickle-cell disease, the spleen may become scarred, giving rise to a patchy, heterogeneous texture.

Splenic vein thrombosis

This is frequently accompanied by portal vein thrombosis and results from the same disorders.

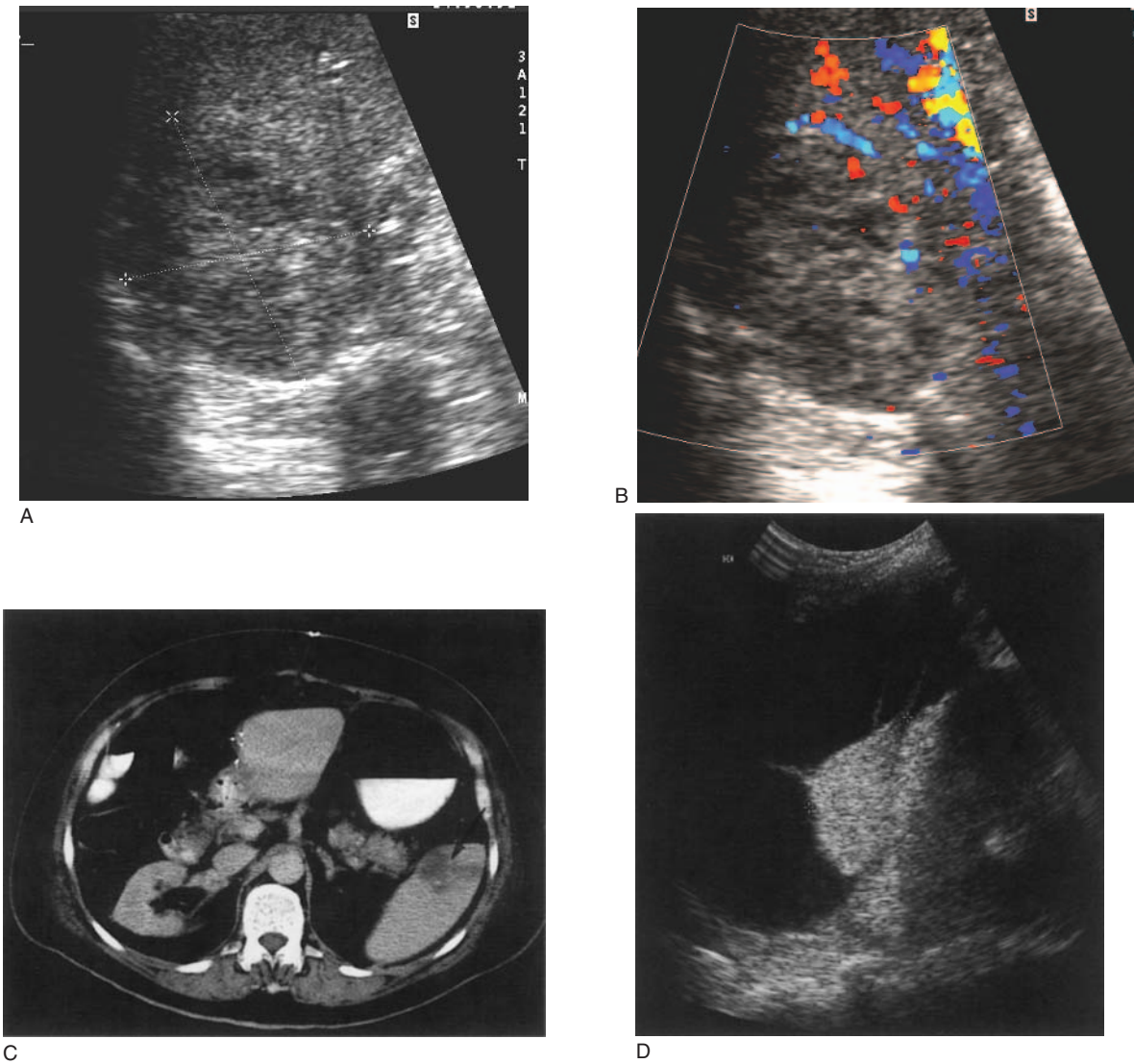


Figure 6.8 (A) Splenic infarct due to an embolus following recent liver resection. (B) Colour Doppler of the same patient demonstrates a lack of perfusion in the infarcted area. (C) CT scan of the same patient. (D) Complete splenic infarction. The spleen is small and hyperechoic. Considerable free fluid is present.

The most common of these are pancreatitis and tumour thrombus. Colour and spectral Doppler are an invaluable aid to the diagnosis, particularly when the thrombus is fresh and therefore echo-poor. Contrast agents may be administered if doubt exists over vessel patency.

Splenic vein occlusion causes splenomegaly and varices may be identified around the splenic hilum.

Splenic artery aneurysm

These are rare, although more common than hepatic artery aneurysms. They are only clinically significant if over 2 cm in diameter, when the risk of rupture and fatal haemorrhage is present.

Colour and spectral Doppler confirm arterial flow through the aneurysm and help to differentiate it

from other possible cystic masses near the splenic hilum, such as pancreatic pseudocysts.

They are usually asymptomatic and are associated with pregnancy or liver disease with portal hypertension. Surgical resection or ligation is performed to prevent rupture, although smaller aneurysms may be safely monitored with ultrasound.[12]

Pseudoaneurysm

Pseudoaneurysm in the spleen occurs in a minority of cases following splenic trauma. An echo-free or 'cystic' area may be observed, which demonstrates flow on colour Doppler.

In rare cases, pseudoaneurysm is also a complication of splenic infarct, inflitration of the spleen by malignancy, inflammatory disease such as pancreatitis, or infection[13] and usually occurs in association with non-traumatic splenic rupture.

Splenic trauma

(*See also Chapter 10.*) Splenic laceration may be particularly difficult to detect on ultrasound, particularly in the immediate post-trauma phase. The presence of free fluid in the abdomen of a trauma victim should alert the sonographer to the strong possibility of organ injury. The laceration may appear as a subtle, hyperechoic line within the spleen immediately after the injury. A frank area of haemorrhage, easily identifiable on ultrasound, may not develop until later.

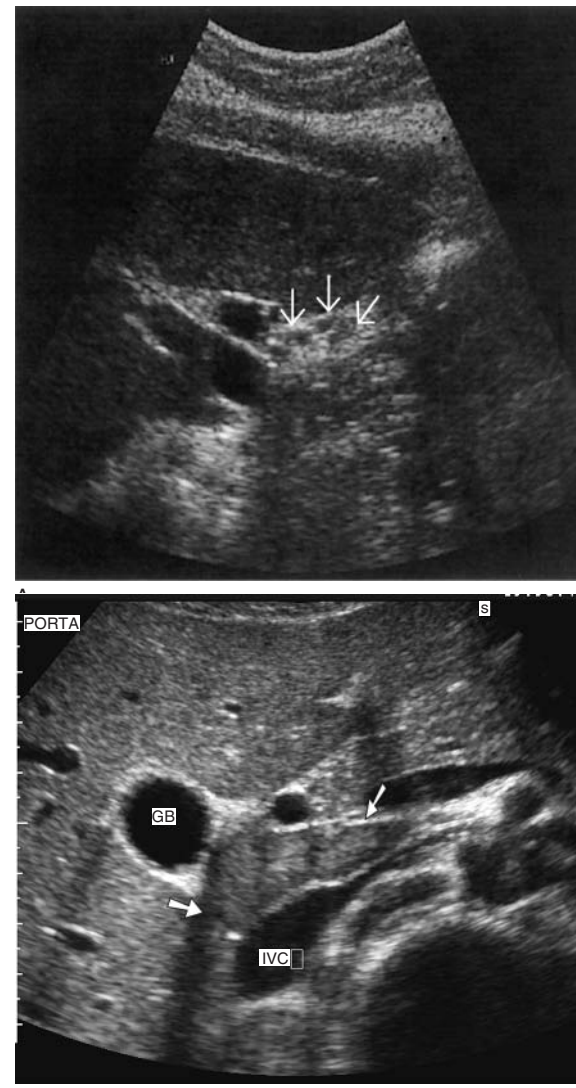
CT is normally performed following the identification of free fluid on ultrasound in order to assess the extent of organ injury. Intrasplenic pseudoaneurysm is a recognized, but rare complication of splenic trauma, which can be demonstrated on colour Doppler.

In rare cases, spontaneous splenic rupture may be encountered, most usually associated with massive splenomegaly of the sort seen in infectious mononucleosis.

LYMPHATICS

Traditionally, normal lymph nodes are difficult or impossible to demonstrate on ultrasound. However, with good-resolution equipment, and using a suitable acoustic window, such as normal liver tissue, normal lymph nodes can be demonstrated in the hepatoduodenal ligament at the porta hepatis (Fig. 6.9A), particularly in younger patients.[14]

The search for lymphadenopathy should include the para-aortic and paracaval regions, the splanchnic vessels and epigastric regions, and the renal hila (Fig. 6.9). Ultrasound has a low sensitivity for demonstrating lymphadenopathy, in the retroperi-



B

Figure 6.9 (A) Normal lymph nodes at the porta. (B) Lymphadenopathy in the epigastrium (arrows) can be seen anterior to the inferior vena cava (IVC).

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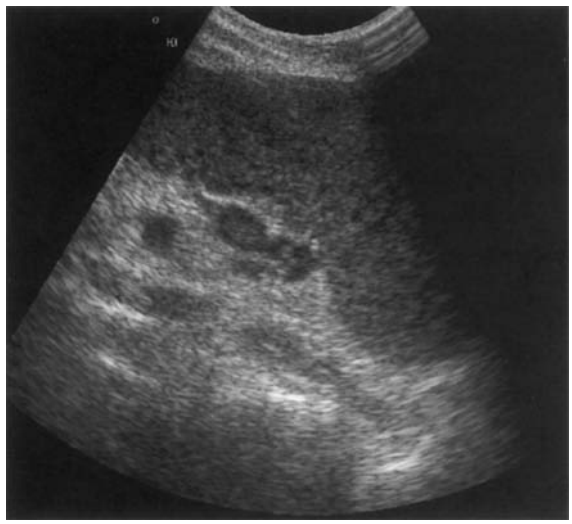

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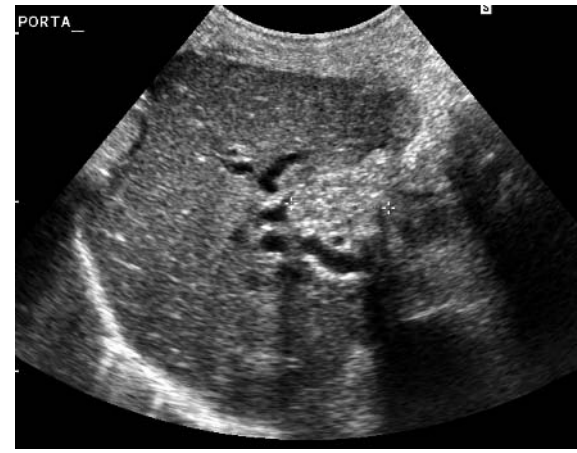

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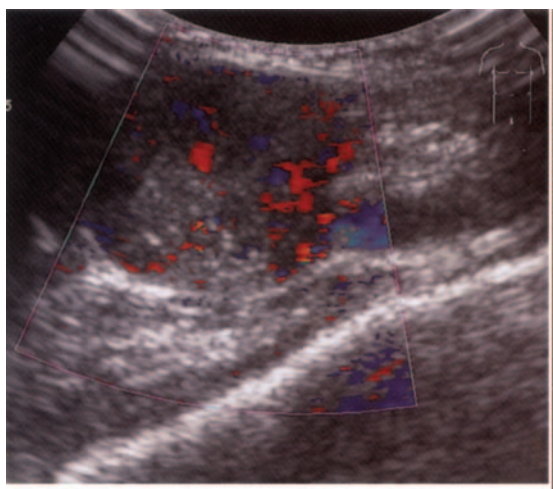

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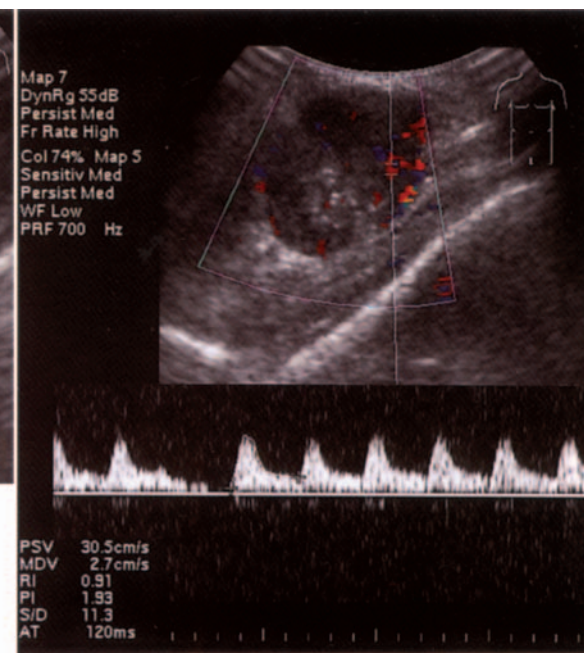

E(ii)

Figure 6.9 cont'd (C) TS through the left upper quadrant (LUQ) showing lymphadenopathy at the splenic hilum of a patient with lymphoma. (D) A large hyperechoic lymph mass at the porta hepatis, causing obstructive jaundice. (E) i, Blood flow in a malignant lymph node in a patient with renal carcinoma; ii, showing a high resistance index of 0.91.

toneum, as bowel contents frequently obscure the relevant areas.

CT or MRI is better able to define the extent of lymphadenopathy, particularly in the pelvis.

The presence of lymphadenopathy is highly non-specific, being associated with a wide range of conditions including malignancy, infections and inflammatory disorders.

Benign lymphadenopathy is commonly seen in conjunction with hepatitis and other inflammatory disorders such as pancreatitis, cholangitis and colitis.[15]

Nodes of 1.5 cm or over are generally considered pathological. Enlarged nodes are most often hypoechoic, rounded or oval in shape and well-defined. Larger nodes display colour or power Doppler flow.

Less frequently nodes are hyperechoic, or may combine to form large, lobulated masses. There is some evidence that colour Doppler may assist in differentiating benign from malignant superficial nodes (Fig. 6.9E), the latter displaying a significantly higher resistance on spectral analysis.[16,17]

Lymphadenopathy at the porta may occasionally cause obstructive jaundice due to compression of the common bile duct.

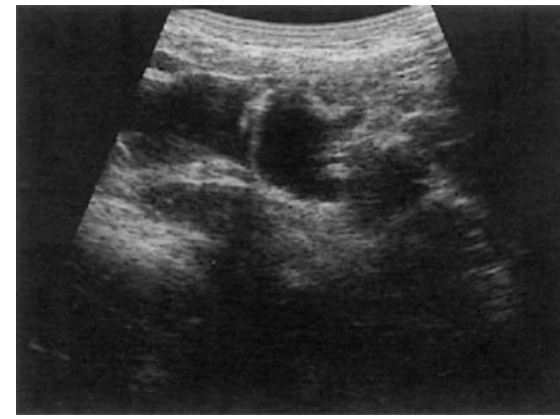

Figure 6.10 Lymphangioma. This large, septated cystic mass was present in the chest wall of this 2-year-old girl.

Lymphangioma

These are benign tumours of the lymphatic vessels, usually diagnosed in the neonatal period or on prenatal sonography. They are predominantly cystic, frequently septated, and may be large (Fig. 6.10). They can compress adjacent organs and vessels and their severity depends to a large extent upon their location. They are most common in the neck (cystic hygroma) but can be found in various locations, including the abdomen,[18] and are occasionally found in adults after a long asymptomatic period.

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Chapter 7

The renal tract

CHAPTER CONTENTS

THE NORMAL RENAL TRACT

Ultrasound technique

The right kidney is readily demonstrated through the right lobe of the liver. Generally a subcostal approach displays the (more anterior) lower pole to best effect, while an intercostal approach is best for demonstrating the upper pole (Fig. 7.1).

The left kidney is not usually demonstrable sagittally because it lies posterior to the stomach and splenic flexure. The spleen can be used as an acoustic window to the upper pole by scanning coronally, from the patient's left side, with the patient supine or decubitus (left side raised), but, unless the spleen is enlarged, the lower pole must usually be imaged from the left side posteriorly.

Coronal sections of both kidneys are particularly useful as they display the renal pelvicalyceal system (PCS) and its relationship to the renal hilum (Fig. 7.2A). This section demonstrates the main blood vessels and ureter (if dilated).

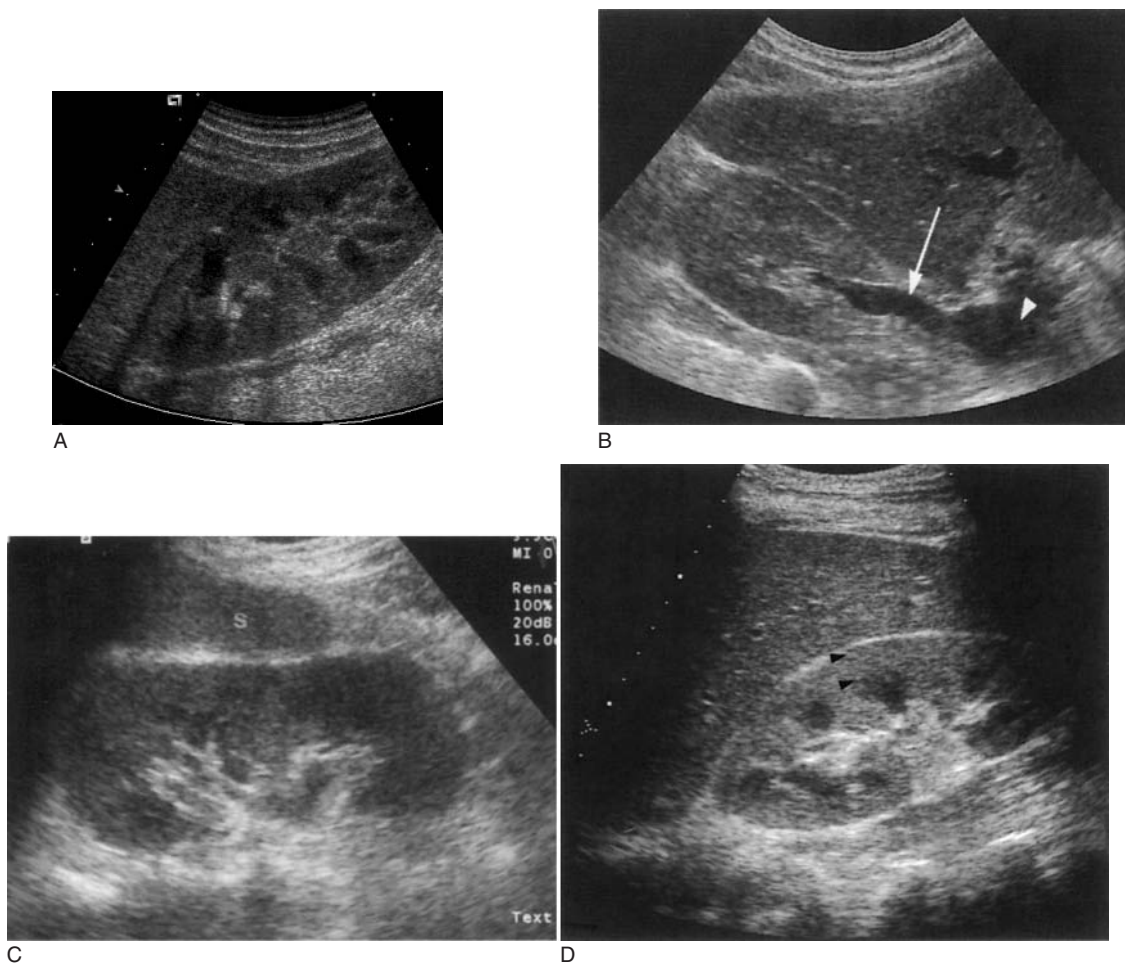


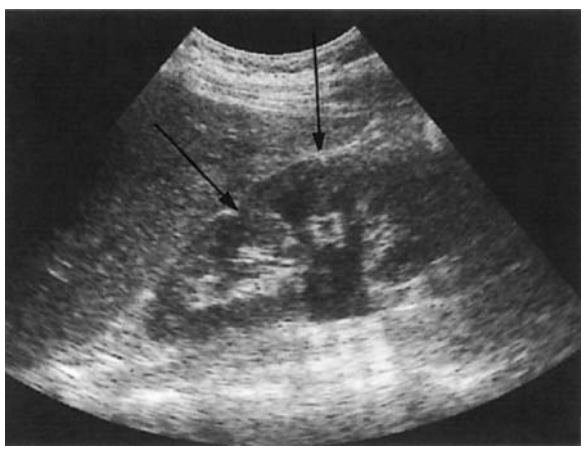
Figure 7.1 (A) Sagittal section through the normal right kidney (RK), using the liver as an acoustic window. The central echoes from the renal sinus are hyperechoic due to the fat content. The hypoechoic, triangular, medullary pyramids are demonstrated in a regular arrangement around the sinus. The cortex is of similar echogenicity to the liver. (B) TS through the hilum of the RK, demonstrating the renal vein (arrow) draining into the inferior vena cava (IVC) (arrowhead). (C) Left kidney (LK) in coronal section. The renal hilum is seen furthest from the transducer (s = spleen). (Compare this with the *sagittal* section of the RK in which cortex is seen all the way around the pelvicalyceal system.) (D) The renal cortex lies between the capsule and the lateral margin of the medullary pyramid (arrowheads).

As with any other organ, the kidneys must be examined in both longitudinal and transverse (axial) planes. This usually requires a combination of subcostal and intercostal scanning with anterior, posterior and lateral approaches. The operator must be flexible in approach to obtain the necessary results.

The bladder should be filled and examined to complete the renal tract scan. An excessively full bladder may cause mild dilatation of the PCS, which will return to normal following micturition.

Normal ultrasound appearances of the kidneys

The cortex of the normal kidney is slightly hypoechoic when compared to the adjacent liver parenchyma, although this is age-dependent. In young people it may be of similar echogenicity and in the elderly it is not unusual for it to be compara-



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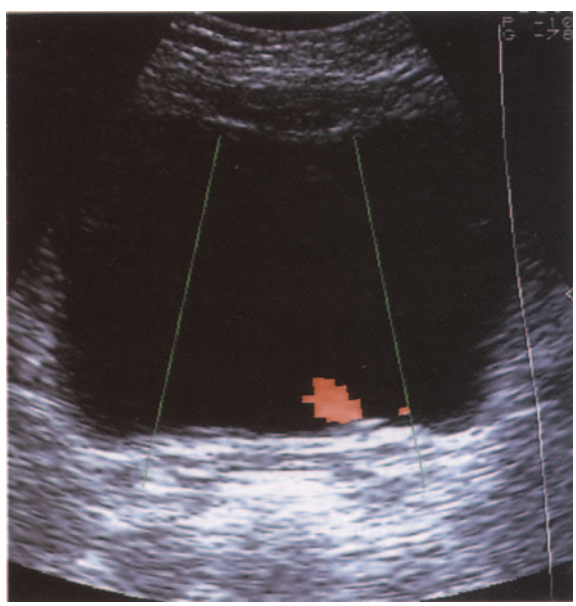

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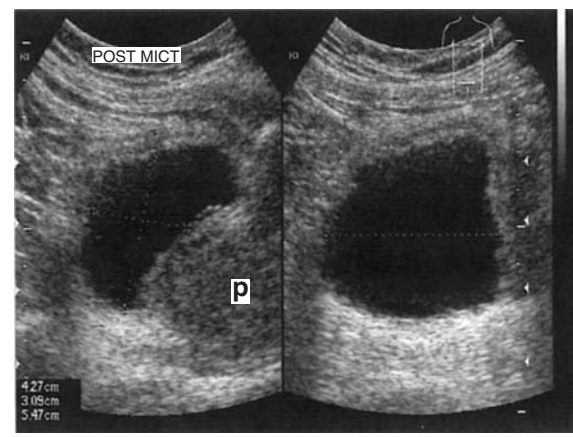

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Figure 7.2 (A) Coronal section through the RK demonstrating fetal lobulations (arrows). The pelvicalyceal system (PCS) is mildly distended due to a full bladder. (B) TS through the base of the bladder, demonstrating a left ureteric jet. (C) Longitudinal section (LS) and TS scans through the bladder after micturition demonstrating an enlarged prostate (P) and a small residual urine volume of 40 ml.

tively hyperechoic and thin. The medullary pyramids are seen as regularly spaced, echo-poor triangular structures between the cortex and the renal sinus (Fig. 7.1). The tiny reflective structures often seen at the margins of the pyramids are echoes from the arcuate arteries which branch around the pyramids.

The renal sinus containing the PCS is hyperechoic due to sinus fat which surrounds the vessels. The main artery and vein can be readily demonstrated at the renal hilum and should not be confused with a mild degree of PCS dilatation. Colour Doppler can help differentiate.

The kidney develops in the fetus from a number of lobes, which fuse. Occasionally the traces of these lobes can be seen on the surface of the kidney, forming *fetal lobulations* (Fig. 7.2A); these may persist into adulthood.

Normal ultrasound appearances of the lower renal tract

When the bladder is distended with urine, the walls are thin, regular and hyperechoic. The walls may appear thickened or trabeculated if the bladder is insufficiently distended, making it impossible to exclude a bladder lesion.

The ureteric orifices can be demonstrated in a transverse section at the bladder base. Ureteric jets can easily be demonstrated with colour Doppler at this point and normally occur between 1.5 and 12.4 times per minute (a mean of 5.4 jets per minute) from each side[1] (Fig. 7.2B).

It is useful to examine the pelvis for other masses, e.g. related to the uterus or ovaries, which could exert pressure on the ureters causing proximal dilatation. The prostate is demonstrated transabdominally by angling caudally through the full bladder (Fig. 7.2C). The investigation of choice for the prostate is transrectal ultrasound; however an approximate idea of its size can be gained from transabdominal scanning. When prostatic hypertrophy is suspected, it is useful to perform a postmicturition bladder volume measurement to determine the residual volume of urine (see *Measurements* below).

Measurements

The normal adult kidney measures between 9 and 12 cm in length. A renal length outside the normal range may be an indication of a pathological process and measurements should therefore form part of the protocol of renal scanning. The maximum renal length can often only be obtained from a section which includes rib shadowing. A subcostal section, which foreshortens the kidney, often underestimates the length and it is more accurate to measure a coronal or posterior longitudinal section.

The cortical thickness of the kidney is generally taken as the distance between the capsule and the margin of the medullary pyramid (Fig. 7.2D). This varies between individuals and within individual kidneys and tends to decrease with age.

The bladder volume (Fig. 7.2C) can be estimated for most purposes by taking the product of three perpendicular measurements and multiplying by 0.56:

$$\textit{Bladder volume (ml)} = \textit{length} \times \textit{width} \times \textit{anteroposterior diameter (cm)} \times 0.56$$

Haemodynamics

The vascular tree of the kidney can be effectively demonstrated with colour Doppler (Fig. 7.3). By manipulating the system sensitivity and using a low pulse repetition frequency (PRF), small vessels can be demonstrated at the periphery of the kidney.

Demonstration of the extrarenal main artery and vein with colour Doppler is most successful in the coronal or axial section by identifying the renal hilum and tracing the artery back to the aorta or the vein to the inferior vena cava (IVC). The best Doppler signals, that is, the highest Doppler shift frequencies, are obtained when the direction of the vessel is parallel to the beam, and taken on suspended respiration. The left renal vein is readily demonstrated between the superior mesenteric artery (SMA) and aorta by scanning just below the body of the pancreas in transverse section. The origins of the renal arteries may be seen arising from the aorta in a coronal section [Fig. 7.3D].

The normal adult renal vasculature is of low resistance with a fast, almost vertical systolic upstroke and continuous forward end diastolic flow. Resistance generally increases with age.[2] The more peripheral arteries are of lower velocity with weaker Doppler signals, and are less pulsatile than the main vessel.

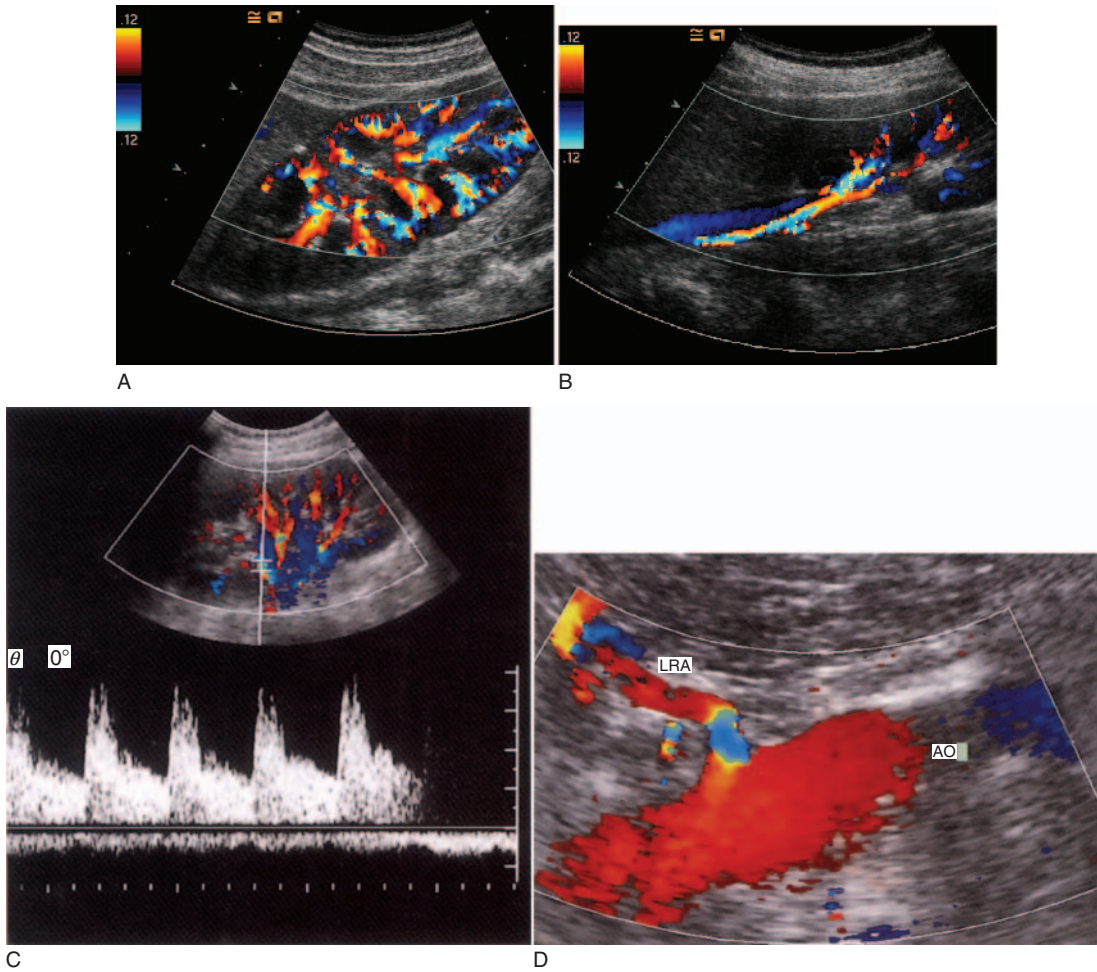


Figure 7.3 (A) Colour Doppler of the RK in coronal section demonstrates normal global intrarenal perfusion throughout the kidney. (B) TS through the LK demonstrates the main renal vein (blue) draining into the IVC. The main renal artery can be seen in red alongside. (C) The waveform from the main renal artery at the hilum of the kidney is of low resistance with good end-diastolic flow. The spectrum from the adjacent vein can be seen below the baseline. (D) Coronal section through the aorta (AO) showing the origin of the left renal artery. The blue colour in the proximal section of the artery is an aliasing artefact due to the strong Doppler signal from this part of the vessel, which is parallel to the beam. (Compare this with the aorta, which, because of its relatively perpendicular angle with the beam, has a poor Doppler signal, despite its high velocity in reality.)

Assessment of renal function

Blood and urine tests can be useful indicators of pathology. Frequently, the request to perform ultrasound is triggered by biochemical results outwith the normal range. Raised serum levels of urea and creatinine are associated with a reduction in renal function. However, any damage is usually quite severe before this becomes apparent. The creatinine clearance rate estimates the amount of creatinine excreted over 24 h, and is a guide to the glomerular filtration rate (normal glomerular filtration rate 100–120 ml/min). A poor rate of clearance (ml/min) is indicative of renal failure.

Au: is there a fig missing?

Blood in the urine is a potentially serious sign which should prompt investigation with ultrasound. Frank haematuria may be a sign of renal tract malignancy. Microscopic haematuria may reflect inflammation, infection, calculi or malignancy. The urine can be easily examined for protein, glucose, acetone and pH using chemically impregnated strips.

Radioisotope scans

Although the ultrasound scan is invaluable in assessing the morphology of the kidneys, it is not able to assess function. The administration of a radioactive tracer, however, reveals valuable information regarding renal function and an isotope scan may often be performed in addition to ultrasound.

A diethylene triaminepenta-acetic acid (DTPA) scan, in which the isotope is intravenously injected as a bolus, can assess renal perfusion, with further data reflecting renal uptake, excretion and drainage during later images.

A dimercaptosuccinic acid (DMSA) scan shows uptake of isotope which is proportional to functioning renal tissue. Relative renal function can be determined between kidneys and localized areas of poor or absent function, such as scars, are clearly demonstrated.

Normal variants

Duplex kidneys

These occur in a spectrum of degrees, from two separate organs with separate collecting systems and duplex ureters, to a mild degree of separation of the PCS at the renal hilum (Fig. 7.4A). The latter is more difficult to recognize on ultrasound, but the two moieties of the PCS are separated by a zone of normal renal cortex which invaginates the kidney, a *hypertrophied column of Bertin* (see below).

If duplex ureters are present (a difficult diagnosis to make on ultrasound unless dilatation is present) then a ureterocoele related to the upper moiety should be sought at or adjacent to the bladder. This may cause dilatation of the affected moiety.

The main renal artery and vein may also be duplicated, which can occasionally be identified using colour or power Doppler.

Ectopic kidneys

The kidney normally ascends from the pelvis into the renal fossa during its course of development. During this 'migration' it rotates inwards so that the renal hilum faces medially. A failure of this mechanism causes the kidney to fall short of its normal position, remaining in the pelvis, that is, a pelvic kidney. Usually it lies on the correct side, however occasionally it can cross to the other side, lying inferior to its normally placed partner—*crossed renal ectopia*. Frequently it may fuse with the lower pole of the other kidney, *crossed fused renal ectopia*, resulting in what appears to be a very long, unilateral organ.

Horseshoe kidneys

In the horseshoe kidney, the kidneys lie one on each side of the abdomen but their lower poles are fused by a connecting band of renal tissue, or *isthmus*, which lies anterior to the aorta and IVC (Fig. 7.4). The kidneys tend to be rotated and lie with their lower poles medially.

It may be difficult to visualize the isthmus due to bowel gas anterior to it but a horseshoe kidney should always be suspected when the operator is unable to identify the lower poles of the kidneys confidently.

When the isthmus can be seen, it is important not to confuse it with other abdominal masses, such as lymphadenopathy. CT is occasionally performed because of this but normally clarifies the findings.

Extrarenal pelvis

Not infrequently, the renal pelvis projects outside the kidney, medial to the renal sinus. This is best seen in a transverse section through the renal hilum. It is frequently 'baggy', containing anechoic urine, which is prominently demonstrated on the ultrasound scan (Fig. 7.4E).

The importance of recognizing the extrarenal pelvis lies in not confusing it with dilatation of the PCS, or with a parapelvic cyst or collection.

Hypertrophied column of Bertin

The septum of Bertin is an invagination of renal cortex down to the renal sinus. It occurs at the

junctions of original fetal lobulations and is present in duplex systems (see above), dividing the two moieties. Particularly prominent, hypertrophied columns of Bertin may mimic a renal tumour. It is usually possible to distinguish between the two as the column of Bertin does not affect the renal outline and has the same acoustic characteristics as the adjacent cortex (Fig. 7.4F).

Colour or power Doppler can be helpful in revealing the normal, regular vascular pattern (as opposed to the chaotic and increased blood flow pattern of malignant renal tumours). If doubt persists, particularly in a symptomatic patient, CT will differentiate tumour from a prominent column of Bertin; an isotope scan can also be helpful in demonstrating normally functioning renal tissue.

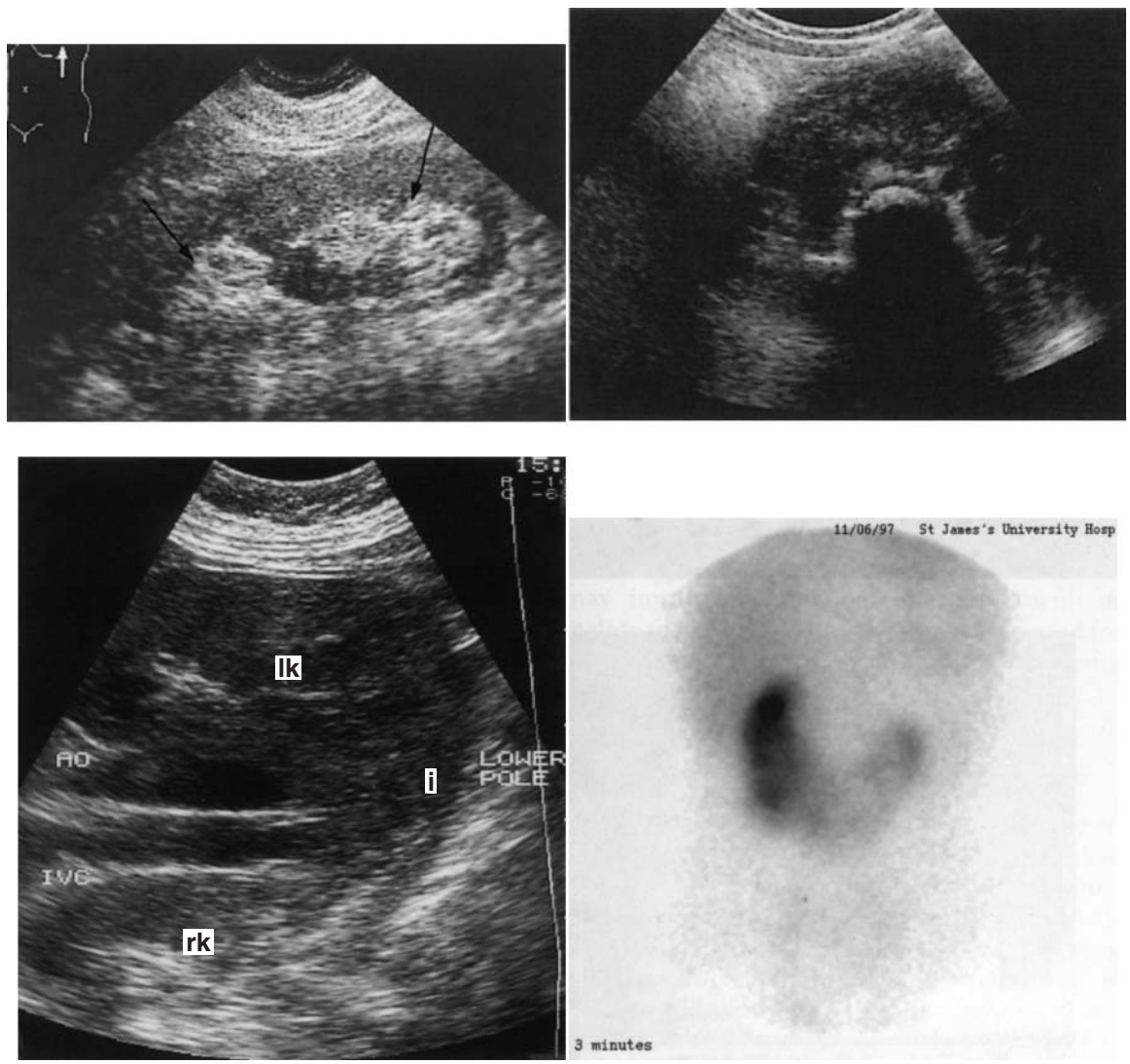
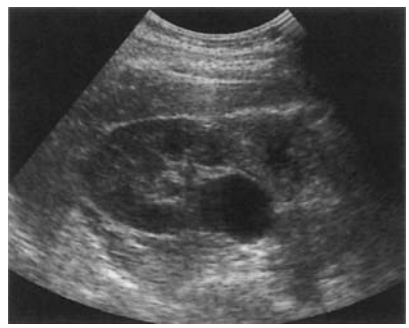


Figure 7.4 (A) Duplex kidney showing two separate intrarenal collecting systems (arrows). These drained into a single ureter on intravenous urogram (IVU). (B) TS through the abdomen demonstrating the fused lower poles of the horseshoe kidney anterior to the spine. (C) Coronal section through a horseshoe kidney with the isthmus of the kidney (i) anterior to the aorta and IVC. (D) MAG3 scan demonstrating a horseshoe kidney with a poorly functioning LK and isthmus. Differential function is 86% on the right and 14% on the left.

(Continued)



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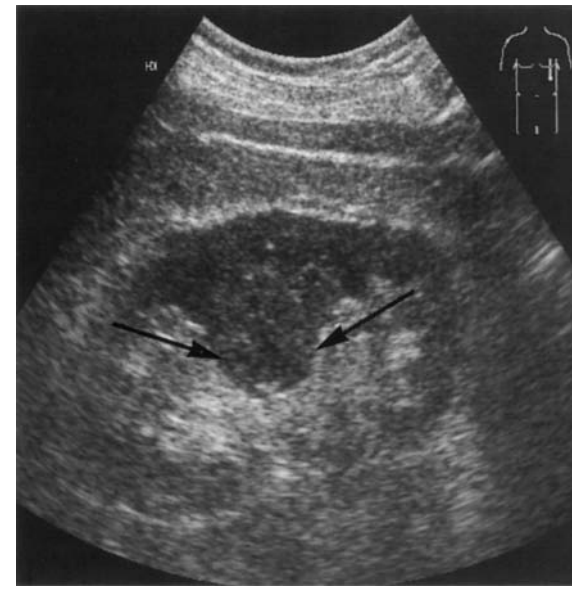

F

Figure 7.4 cont'd (E) TS through the RK demonstrating a baggy extrarenal pelvis. The PCS remains undilated, and this should not be confused with hydronephrosis. (F) Hypertrophied column of Bertin (arrows).

Renal humps

These are areas of renal cortex, which form a bulge in the renal outline. Like the hypertrophied column of Bertin, a hump may mimic a renal mass. Careful scanning can usually solve the dilemma as the cortex remains constant in thickness. The most usual manifestation is the *splenic hump* on the left kidney, which is a flattening of the upper pole with a lateral prominence just below the margin of the spleen. Humps are basically a variation in the shape of the kidney rather than an area of hypertrophied tissue.

RENAL CYSTS AND CYSTIC DISEASE

Cysts

The most common renal mass is a simple cyst which can be found in up to 50% of the population, the incidence increasing with age. Most cysts are asymptomatic and may be solitary or multiple. Generally they are peripheral but may occur within the kidney adjacent to the renal pelvis. A parapelvic cyst may be difficult to distinguish from pelvicalyceal dilatation, a calyceal diverticulum or an extrarenal pelvis and careful scanning is required to differentiate. A parapelvic cyst may be the cause of a filling defect on intravenous urogram (IVU) and CT can differentiate a cyst from a diverticulum if necessary, as the latter will fill with contrast.

Occasionally cysts can haemorrhage causing pain. Large cysts, particularly of the lower pole, may be palpable, prompting a request for an ultrasound scan.

Ultrasound appearances

Like cysts in any other organ, renal cysts display three basic characteristics: they are anechoic, have a thin, well-defined capsule and exhibit posterior enhancement. It can be difficult to appreciate the posterior enhancement if the hyperechoic perirenal fat lies distal to the cyst; scanning from a different angle (Fig. 7.5) may be helpful. Haemor-rhage or infection can give rise to low-level echoes within a cyst and in some cases the capsule may display calcification.

Whilst a solitary, simple cyst can almost certainly be ignored, cysts with more complex acoustic characteristics may require further investigation,

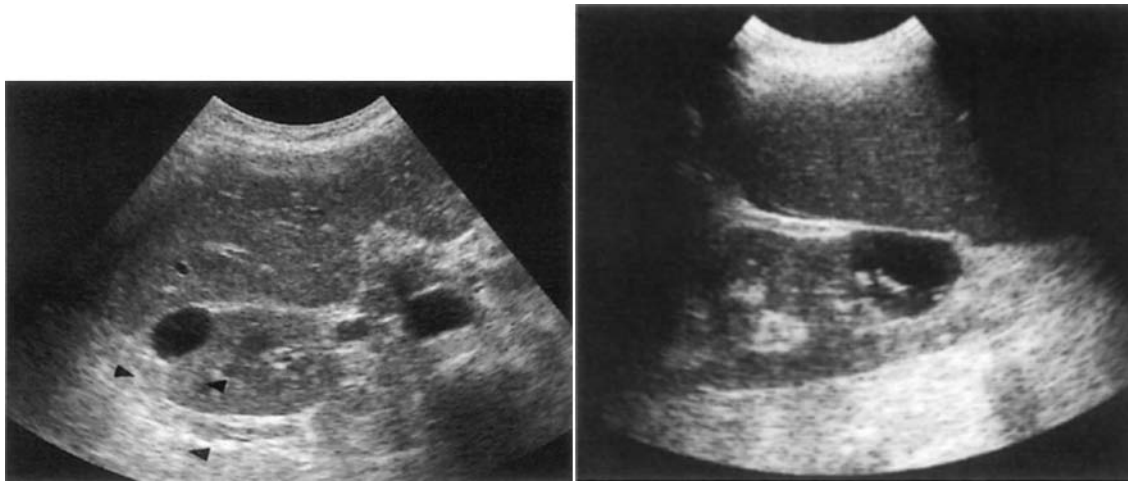

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Figure 7.5 (A) Simple renal cyst with posterior enhancement (arrowheads). (B) Small renal cyst containing calcification following episodes of infection. This remained stable on follow-up.

for example CT. A calcified wall may be associated with malignancy.

Autosomal dominant (adult) polycystic kidney disease (APKD)

This autosomal dominant disease has a wide spectrum of presentation. It is normally associated with progressive renal failure. A renal transplant offers a successful cure for many patients. Although in some cases APKD may cause renal failure in early life, it is also possible to achieve a normal life span with no appreciable symptoms.

In about 50% of cases, cysts are present in the liver; they are also found in the spleen and pancreas in a small proportion of patients.

Ultrasound screening for APKD is performed in families with a positive history, as patients may then be monitored and treated for hypertension. A negative scan does not entirely exclude disease, especially in the younger patient, and multiple examinations over years may need to be performed.

Ultrasound appearances

The disease is always bilateral, causing progressively enlarging kidneys with multiple cysts of various sizes, many having irregular margins (Fig. 7.6). There is often little or no demonstrable normal renal tissue and the kidneys may become so large that they visibly distend the abdomen.

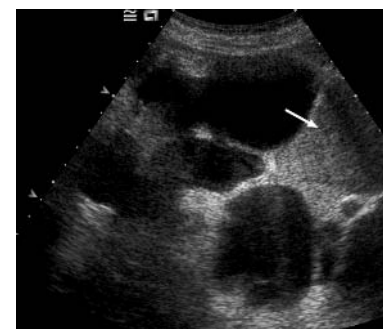


Figure 7.6 Autosomal dominant ('adult') polycystic disease. Numerous cysts of varying size are seen within the renal bed. No discernible renal architectrue is apparent. A cyst containing solid debris, i.e. haemorrhage (arrow), is seen.

APKD predisposes the patient to urinary tract infections and some of the cysts may contain low-level echoes as a result of infection or haemorrhage.

The liver, spleen and pancreas should also be examined on ultrasound for associated cysts. A small but recognized increased incidence of tumour is recorded in patients with APKD.

Autosomal recessive (infantile) polycystic kidney disease (PCKD)

This autosomal recessive condition may often be diagnosed prenatally on ultrasound. The disease carries a high mortality rate in early childhood, and is therefore rarely seen on ultrasound in children.

Tiny cysts replace both kidneys, giving them a hyperechogenic appearance due to the multiple reflections from the cyst walls and the overall increased through-transmission.

Acquired cystic disease

This condition tends to affect patients on long-term dialysis who may already have shrunken, end-stage kidneys. Its frequency increases with the duration of dialysis.

Multiple cysts form in the kidneys, which may, like adult PCKD, haemorrhage or become infected. The disease tends to be more severe the longer the patient has been on dialysis. The proliferative changes which cause acquired cystic disease also give rise to small adenomata and the ultrasound appearances may be a combination of cysts and solid, hypoechoic nodules. In particular, acquired cystic disease has the potential for malignancy[3,4] and it is therefore prudent to screen native kidneys, even after renal transplantation has been performed (Fig. 7.7).

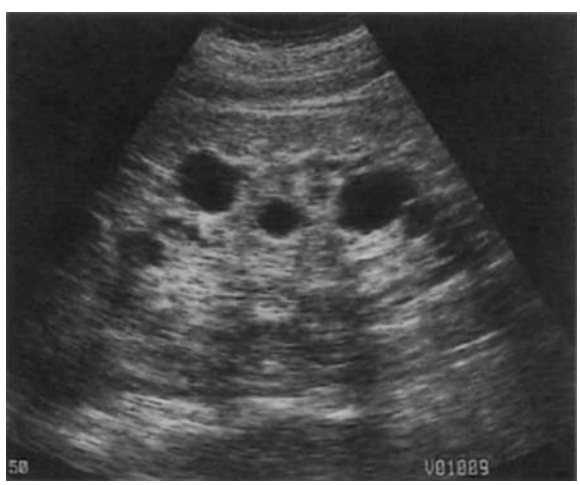


Figure 7.7 Acquired cystic disease in a patient with chronic renal failure who has been on long-term dialysis.

Multicystic dysplastic kidney (MCDK)

This is a congenital malformation of the kidney, in which the renal tissue is completely replaced by cysts. It is frequently diagnosed prenatally (although it is naturally a lethal condition if bilateral).

The MCDK may shrink with age and, by adulthood, may be so small that it is difficult to detect and may be mistaken for an absent kidney. Contralateral renal hypertrophy is often present. MCDK can be associated with contralateral pelviureteric junction obstruction, which is also frequently diagnosed in utero.

It is thought that MCDK occurs as a result of severe early renal obstruction during development in utero. Obstructed calyces become blocked off, forming numerous cysts which do not connect.

BENIGN FOCAL RENAL TUMOURS

Angiomyolipoma

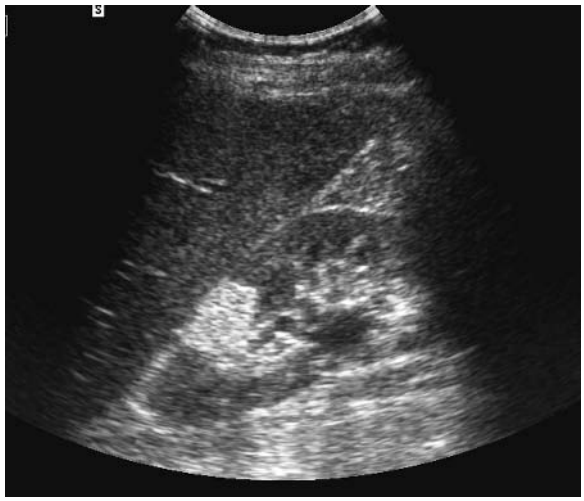
This is a homogeneous, highly echogenic, usually rounded lesion in the renal parenchyma containing blood vessels, muscle tissue and fat, as the name suggests. They are usually solitary, asymptomatic lesions, found incidentally on the scan, although the larger lesions can haemorrhage, causing haematuria and pain. Angiomyolipomas are also associated with tuberose sclerosis, when they are often multiple and bilateral (Fig. 7.8).

Because the contrast between the hypoechoic renal parenchyma and the hyperechoic angiomyolipoma is so great, very small lesions in the order of a few millimetres can easily be recognized.

It may be difficult confidently to differentiate an angiomyolipoma from a malignant renal neoplasm, particularly in a patient with haematuria. Angiomyolipomas tend to be smaller and more echogenic than renal cell carcinomas, and sometimes demonstrate shadowing, which is not normally seen in small carcinomas.[5] When doubt persists, CT is often able to differentiate in these cases by identifying the fat content of the lesion.

Adenoma

The renal adenoma is usually a small, well-defined hyperechoic lesion, similar in appearance to the



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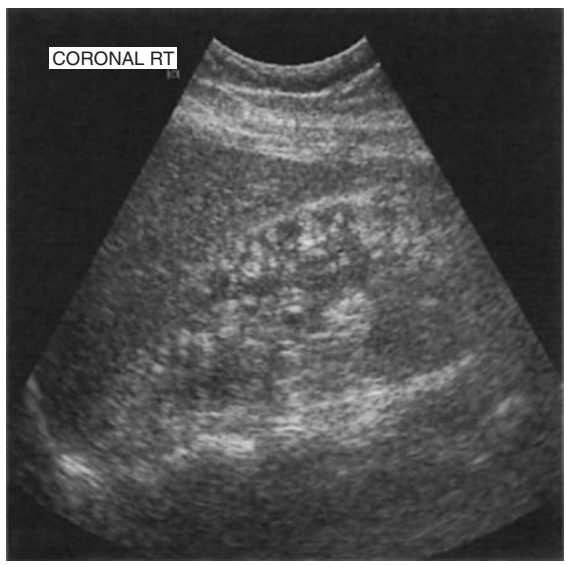

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Figure 7.8 (A) Angiomyolipoma in the RK. (B) Multiple, small angiomyolipomas in a patient with tuberose sclerosis.

angiomyolipoma. It is felt that adenomas are frequently early manifestations of renal carcinoma as distinct from a benign lesion[6] and the two may be histologically indistinguishable.

Renal adenomas are often found in association with a renal cell carcinoma in the same or contralateral kidney,[7] although these are radiologically indistinguishable from metastases.

Because of the controversy surrounding the distinction between adenomas and small renal cell carcinomas, the management of patients with these masses is uncertain. Most incidentally discovered, small (less than 3 cm), parenchymal renal masses are slow-growing and may be safely monitored with CT or ultrasound, particularly in the elderly.[8]

MALIGNANT RENAL TRACT MASSES

Imaging of malignant renal masses

Ultrasound, as one of the first-line investigations in patients with haematuria, is highly sensitive in detecting large renal masses above 2.5 cm in diameter and in differentiating them from renal cysts. Smaller masses may be missed with ultrasound however, as they are frequently isoechoic (in 86% of cases); CT is more sensitive in small lesion detection.

MRI also detects small renal masses more frequently than ultrasound but is generally reserved for patients with equivocal CT scans as it is less widely available. IVU is also known to miss small renal masses and normally requires further characterization of any detected mass with ultrasound or CT.

Renal cell carcinoma (RCC)

Adenocarcinoma is the most common type of renal malignancy (referred to as renal cell carcinoma) occurring less commonly in the bladder and ureter. RCCs are frequently large at clinical presentation; they may occasionally be identified as an incidental finding in an asymptomatic patient.

Ultrasound appearances

The RCC is a (usually) large, heterogeneous mass which enlarges and deforms the shape of the kidney (Fig. 7.9). The mass may contain areas of cystic degeneration and/or calcification. It has a predilection to spread into the ipsilateral renal vein and IVC (see also Chapter 8).

Colour Doppler usually reveals a disorganized and increased blood flow pattern within the mass with high velocities from the arterioverous shunts within the carcinoma.

Smaller RCCs can be hyperechoic and may be confused with benign angiomyolipoma. The latter

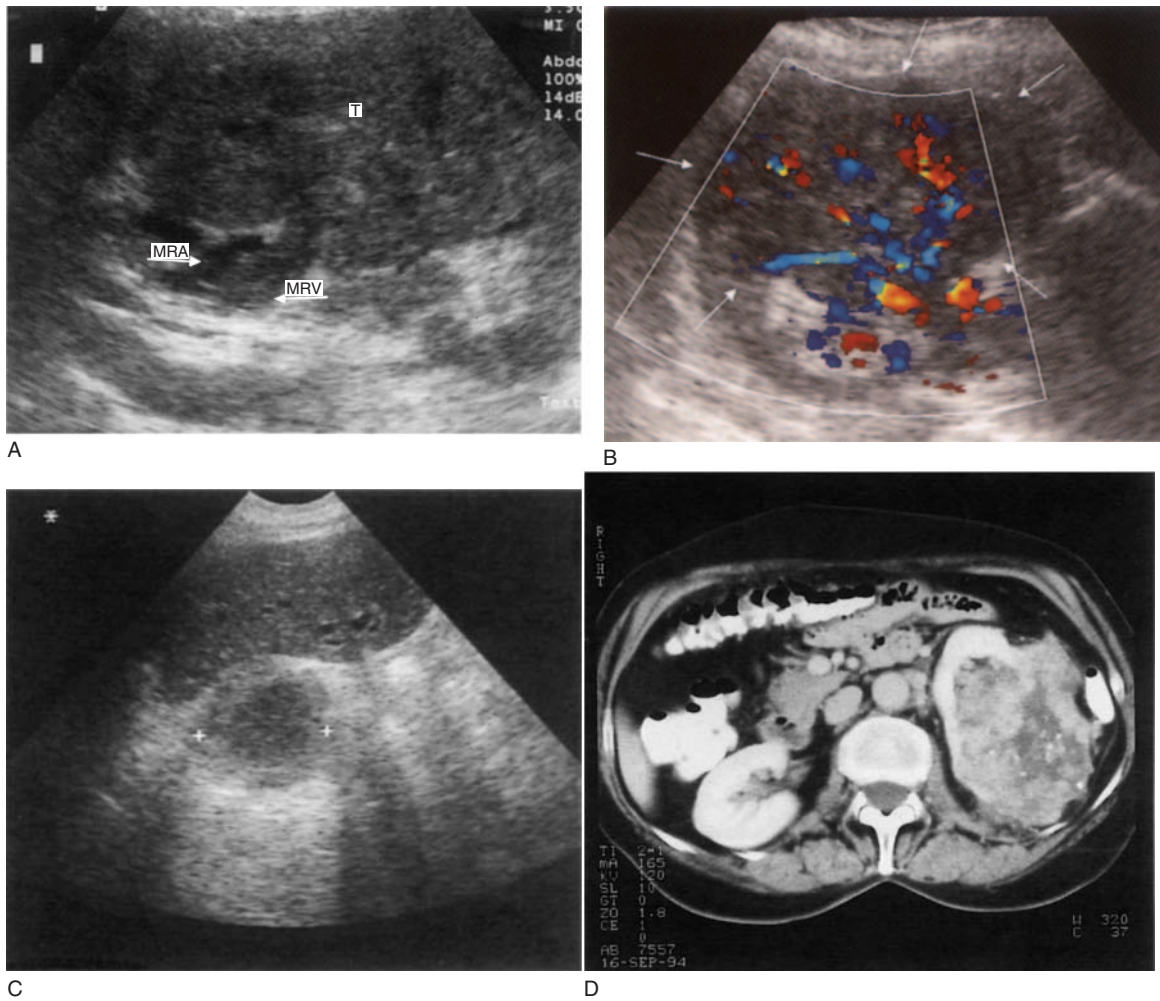


Figure 7.9 (A) The RK is almost completely replaced by a large renal carcinoma (T). The main renal vein contains tumour thrombus which has spread into the IVC. The main renal artery is seen alongside. (B) Colour Doppler of the tumour reveals vigorous, multidirectional blood flow within it. (C) Recurrence of carcinoma (between calipers) in the right renal bed of a patient following right nephrectomy for renal carcinoma. (D) CT demonstrating a large left renal carcinoma.

has well-defined borders whilst an RCC is ill-defined: differentiation may not be possible on all occasions and biopsy or interval scan may be required.

A chest X-ray and/or CT will demonstrate if metastases are present in the lungs. Liver, adrenal and lymph node metastases can be demonstrated on ultrasound but CT is used for staging purposes as ultrasound generally has a lower sensitivity for distant disease detection.

Transitional cell carcinoma

Transitional cell carcinoma is the most common bladder tumour, occurring less frequently in the collecting system of the kidney and the ureter. It usually presents with haematuria while still small. It is best diagnosed with cystoscopy. Small tumours in the collecting system are difficult to detect on ultrasound unless there is proximal dilatation. Depending on its location it may cause hydronephrosis, particularly if it is situated in the ureter (rare) or at

the vesicoureteric junction (VUJ). IVU, retrograde cystography and CT are methods of diagnosis.

Ultrasound appearances

Situated within the collecting system of the kidney, the transitional cell tumour is usually small (compared to the RCC), homogeneous and relatively hypoechoic (Fig. 7.10A). Proximal renal tract dilatation may sometimes be present. These tumours are easy to miss on ultrasound unless the kidney is scanned very carefully, and often are, unless the case is highlighted by clinical symptoms or a high clinical index of suspicion. They can mimic a hypertrophied column of Bertin (see above); CT may differentiate in cases of doubt.

Once large, they invade the surrounding renal parenchyma and become indistinguishable from RCC on ultrasound. They frequently spread to the bladder and the entire renal tract should be carefully examined.

In the bladder they are potentially easier to see as they are surrounded by urine (Fig. 7.10B). Invasion of the bladder wall can be identified on ultrasound in the larger ones but biopsy is necessary to determine formally the level of invasion.

IVU or a retrograde cystogram are the methods of choice for demonstrating a filling defect in the PCS (Fig. 7.11) or ureter; CT may be useful and is also used for staging purposes.

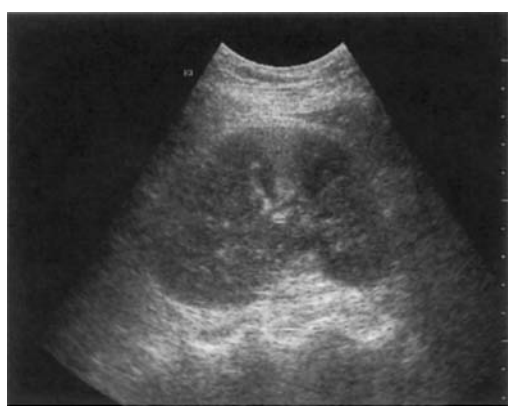
Lymphoma

Renal involvement of non-Hodgkin's or Hodgkin's lymphoma is not uncommon and depends upon the stage of the disease. The ultrasound appearances are highly variable and range from solitary to multiple masses, usually hypoechoic but sometimes anechoic, hyperechoic or mixed.

The masses may have increased through transmission of sound and may mimic complex fluid lesions such as haematoma or abscess. The clinical history should help to differentiate these cases. Occasionally diffuse enlargement may occur secondary to diffuse infiltration.

Metastases

Renal metastases from a distant primary are usually found in cases of widespread metastatic disease and are frequently multiple.



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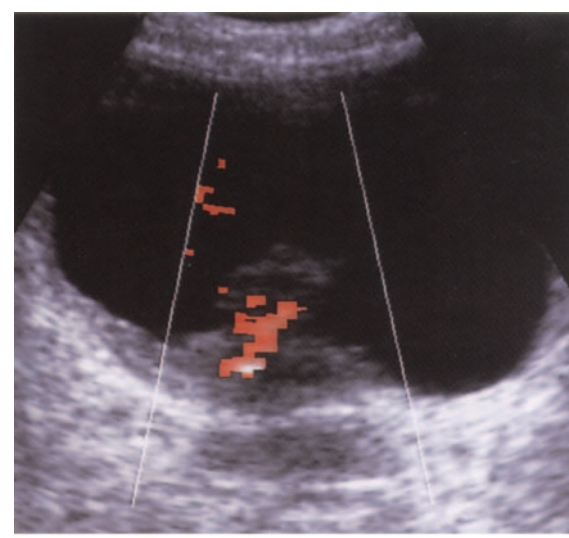

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Figure 7.10 (A) Large, transitional cell carcinoma in the upper pole of the RK. The changes are more subtle than those of renal cell carcinoma, and the renal outline remains intact. (B) Transitional cell carcinoma in the bladder at the right vesicoureteric junction. Blood flow can clearly be seen within the tumour, and right renal and ureteric dilatation was present.

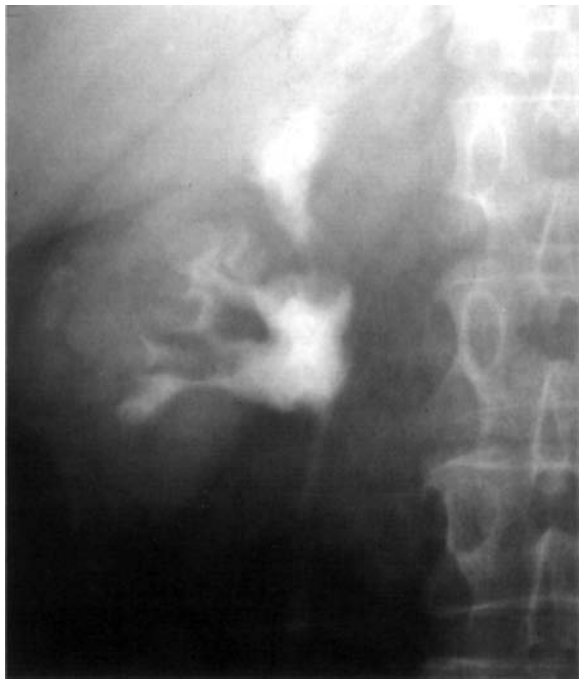

Figure 7.11 IVU with multiple filling defects in the relatively non-dilated PCS of the RK, which represent transitional cell carcinomas.

In such cases, the primary diagnosis is usually already known and other abdominal metastases, such as liver deposits and/or lymphadenopathy, are commonly seen on ultrasound.

Rarely, a single metastasis is seen in the kidney without other evidence of metastatic spread, making the diagnosis difficult (as the question arises of whether this could be a primary or secondary lesion). CT may identify the primary and frequently picks up other, smaller metastases not identified on ultrasound.[9]

PELVICALYCEAL SYSTEM DILATATION AND OBSTRUCTIVE UROPATHY

Not all PCS dilatation, i.e. hydronephrosis, is pathological, or indeed obstructive, that is, there can be dilatation without physiological obstruction. Conversely, not all obstructive uropathy necessarily results in PCS dilatation.

Physiological dilatation

Mild dilatation of the renal collecting system is a common finding, most commonly secondary to an over-distended bladder. Following micturition, the collecting system decompresses and returns to normal. An external renal pelvis (see above) is a non-obstructive 'baggy' dilatation of the pelvis and can be regarded as a normal variant. The intrarenal collecting system is normal in this situation (Fig. 7.4, D,E).

Pregnancy is another common cause of mild PCS dilatation, more frequently on the right, particularly in the second and third trimester. This is thought to be due partly to pressure on the ureters from the advancing pregnancy and partly hormonal. It is however wrong to assume that the kidney is not obstructed just because the patient is pregnant. If symptomatic, the suspicion of obstruction in a dilated system is increased, particularly if echoes are present in the PCS.

Obstructive uropathy

Renal obstruction, particularly if long-standing, can irreversibly damage the kidney or kidneys, leading eventually to renal failure. If diagnosed early enough, renal function can be preserved and therefore ultrasound plays a prominent role as one of the first-line investigations in patients with loin pain, renal colic or micturition disorders (Table 7.1).

In the vast majority of cases, urinary tract obstruction causes dilatation of the collecting system proximal to the site of obstruction (Fig. 7.12). Whether the hydronephrosis is bilateral or unilateral and whether or not it involves the ureter(s) depends on the cause and site of the obstructing lesion.

Dilatation of the collecting system may be localized. Sometimes only one moiety of the kidney may be obstructed by a stone or tumour, whilst the rest of the kidney remains normal. In a duplex kidney, dilatation of the upper pole moiety is a common occurrence due to an anomaly at the VUJ, that is, a ureterocoele.

If the obstruction is long-standing the renal cortex may atrophy, becoming thin. Normal thickness of cortex is a good prognostic indicator. Function may be assessed with a nuclear medicine (DTPA) scan prior to further management.

Table 7.1 Causes of renal tract obstruction

Source of obstruction	Characteristics
Intrinsic factors	
Stones	Accompanied by renal colic. May be situated anywhere along the renal tract
Tumour	In the bladder, PCS or ureter
Blood clot	From infection or trauma
Papillary necrosis	Sloughed papillae can travel down the ureter, causing obstruction
Infective processes	
Stricture	Caused by chronic, repeated infection
Fungal balls	Rare
Tuberculosis	
Congenital	
Idiopathic PUJ obstruction	Usually unilateral. PCS dilatation only
Posterior urethral valves	Entire renal tract dilatation. Frequently diagnosed antenatally
Ureterocoele	Unilateral hydronephrosis with hydroureter
Outflow obstruction	
Prostate enlargement	Benign or malignant
Urethral stricture	May be iatrogenic, congenital or as a result of infection. Accompanied by disturbed micturition
Extrinsic pelvic mass	
Cervical carcinoma	Proximity to the ureters causes obstruction
Endometriosis	Endometriotic lesions adhere to the peritoneal and/or ureteric surfaces, causing compression
Others: lymphadenopathy, inflammatory bowel masses, gynaecological masses	Always scan the kidneys to exclude obstruction when a pelvic mass is present
Iatrogenic	
Postsurgical procedure	Ligation of ureters in gynaecological procedures
Trauma	Can cause a stricture of the ureter or can cause the renal tract to be blocked by blood clot from damage to the kidney

PCS = pelvicalyceal system; PUJ = pelviureteric junction.

Further management of renal obstruction

In the majority of cases the exact level and cause of obstruction are difficult to identify on ultrasound. Confirmation of the cause and identification of the exact level is traditionally best established on IVU;[10] however CT IVU is becoming a rapidly universally adopted first-line investigation.[11]

A plain abdominal X-ray is useful in confirming the presence of calculi in the renal tract, but ultrasound may demonstrate stones which are non-opaque on X-ray; CT is probably the best overall test for stone detection.

It is important to assess the *function* of the obstructed side, as a chronic, longstanding obstruction with no residual function cannot be treated, but a kidney which still has function is worth saving. A DTPA scan can assess the relative functions of the obstructed and non-obstructed side.

Percutaneous nephrostomy (the placing of a tube into the PCS to drain the urine) in the case of unilateral obstruction is performed to relieve the obstruction, minimizing damage to the kidney and maintaining renal function and drainage. This may be done under either ultrasound or fluoroscopic guidance or a combination of both. The decision of whether to proceed to nephrostomy or cystoscopic stent will depend upon patient presentation and local factors and policies.

Pyonephrosis

Pyonephrosis is a urological emergency. An obstructed kidney is prone to become infected. High fever and loin pain can suggest obstructive pyonephrosis. Pus or pus cells may also be detected in the urine.

Low level echoes can be seen within the dilated PCS on ultrasound, and may represent pus. Sometimes, however, the urine may appear anechoic, despite being infected. The clinical history should help differentiate pyo- from simple hydronephrosis (Fig. 7.13A). Percutaneous drainage by ultrasound or fluoroscopically guided nephrostomy is usually necessary, partly as diagnostic confirmation and partly as a therapeutic procedure.

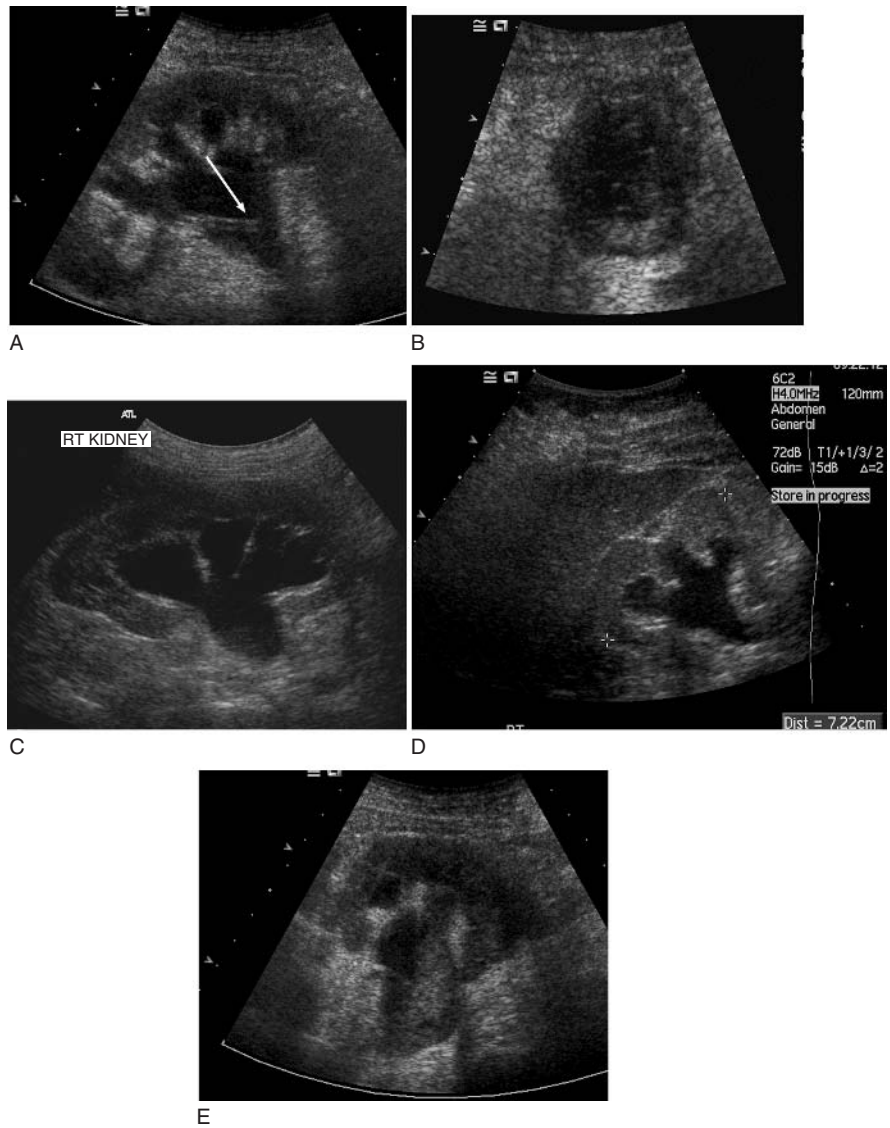

Figure 7.12 (A) Hydronephrosis of the left kidney, secondary to a large circumferential bladder tumour. (B) A ureteric stent is noted within the renal pelvis of (A) (arrow); however, a moderate degree of hydronephrosis is present and highly suggestive of partial or complete stent occlusion. (C) Moderate to marked hydronephrosis of the right kidney secondary to a pelvic lesion. The cortical thickness is normal suggesting the obstruction is relatively recent and that relief of obstruction should produce a significant improvement in renal function. (D) Hydronephrosis of the right kidney. The kidney however is small at 7.2 cm, the cortex echogenic and thinned, particularly at mid pole level. Appearances suggest this appearance is chronic. (E) TS of a left-sided hydronephrosis. Echogenic material is present within the collecting system. The patient was pyrexial. Pus was drained.

Haemo-hydronephrosis

Blood within the dilated PCS may be due to trauma or other local or semilocal pathological processes such as infection or tumour. It is not usually possible to determine whether obstruction is caused by a blood clot or whether the blood is the result of an obstructing lesion which is also causing bleeding. Renal colic as a result of obstruction by a blood clot in the absence of trauma or blood dyscrasia must naturally be thoroughly investigated to exclude an underlying lesion.

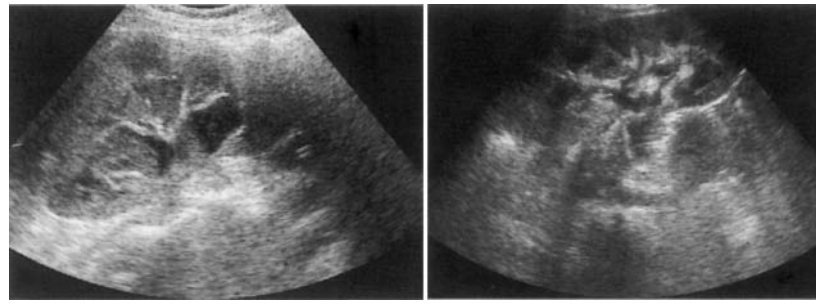

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Figure 7.13 (A) Pyonephrosis. Low-level echoes from pus can be seen in the dilated PCS. (Note that absence of echoes does not exclude pyonephrosis.) (B) A hyperechoic blood clot can be seen within the collecting system of this dilated kidney.

Like pyonephrosis, low-level echoes may be seen on ultrasound within the collecting system (Fig. 7.13B). Although ultrasonically it is not possible to differentiate pyo- from haemohydronephrosis, the clinical picture can be suggestive of one or the other.

Non-dilated renal obstruction

Obstruction may occasionally be present in the acute stages before renal dilatation is apparent: beware—*the finding of a non-dilated PCS on ultrasound does not exclude obstruction in any patient with symptoms of renal colic.*

Spectral Doppler is useful in diagnosing acute, early renal obstruction, before PCS dilatation develops, because of the associated increase in blood flow resistance in the affected kidney (Fig. 7.14). This causes an increase in the resistance and pulsatility indices (RI and PI) on the obstructed side, due to a reduction in diastolic flow. A raised RI in itself is a non-specific finding, not necessarily indicating obstruction; it is known to be age-related or can be associated with extrinsic compression of the kidney (for example by a fluid collection or mass) or with some chronic renal diseases or vascular disorders. This can be overcome by analysing Doppler spectra from both kidneys and evaluating any difference between the two sides.

A marked *difference* in the RI between the kidneys in a patient with renal colic points towards obstruction of the kidney with the higher resistance.[12,13] A difference in RI of greater than 6 is highly suspicious of obstruction in a patient with renal colic; a reduction in the RI on the affected side

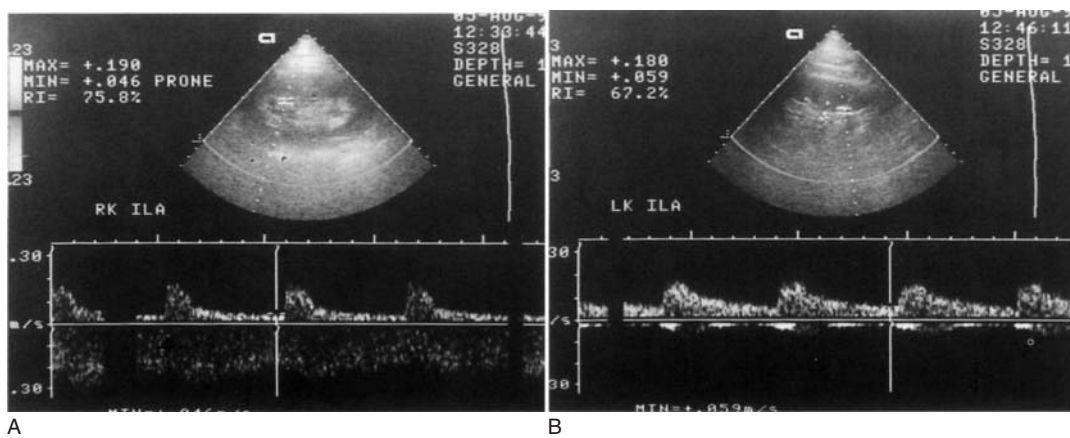

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Figure 7.14 (A) A patient with acute renal colic has a normal-looking, non-dilated RK with a raised resistance index of 75.8. IVU subsequently confirmed early obstruction of the RK. The resistance index (RI) subsequently returned to normal following relief of the obstruction. (B) In the same patient, the contralateral, normal kidney has a much lower resistance index of 67.2.

can be observed when the obstruction has been relieved or after the renal PCS has become dilated.

This effect often does not persist once the kidney dilates, presumably because the intrarenal pressure is relieved, which emphasizes the use of Doppler in acute cases, before dilatation has become established.[14] Because of the vagaries of the stage of obstruction, renal pressure, etc. the interpretation of RI should be made cautiously.

IVU will show delayed PCS opacification and is also more useful than ultrasound in assessing the level of obstruction. CT IVU, as mentioned previously, is more commonly fulfilling the role previously held by the IVU.

Vesicoureteric junction

The normal ureters may be identified on ultrasound with high-resolution equipment, as they enter the bladder. Jets of urine emerge into the bladder at these points and can be demonstrated with colour Doppler. An absent or reduced number of jets may indicate obstruction on that side; this finding again should be interpreted cautiously. Ureteric jet analysis is not routinely performed at most hospitals as a diagnostic test of renal obstruction.

Careful scanning at the VUJs can identify significant anomalies (Figs 7.12 D, E):

- Reflux can be seen to dilate the ureter intermittently (see below).
- A ureterocoele may be diagnosed as it dilates with the passage of urine; it may not be obvious until the operator has watched carefully for a few minutes.
- Stones may become lodged at the VUJ, causing proximal dilatation.

Non-obstructive hydronephrosis

Not all renal dilatation is the result of an obstructive process and the kidney may frequently be dilated for other reasons.

Reflux

This is the most common cause of non-obstructive renal dilatation, and is normally diagnosed in children. Reflux is associated with recurrent urinary tract infections and can result in reflux nephropathy, in which the renal parenchyma is irretrievably damaged.

Reflux can be distinguished from other causes of renal dilatation by observing the dilatation of the ureters at the bladder base, due to the retrograde passage of urine. For a more detailed consideration of the diagnosis of reflux, see Chapter 9. Paediatric ch?

Postobstructive dilatation

Dilatation of a once severely obstructed kidney may persist. The PCS remains baggy and dilated despite the obstruction having been relieved.

Papillary necrosis

The renal papillae, which are situated in the medulla adjacent to the calyces, are susceptible to ischaemia due to relatively low oxygenation in the region of the medullary junction. This is particularly associated with diabetic patients and those on long-term anti-inflammatory or analgesic medication.

The papillae tend to necrose and slough off, causing blunting of calyces on IVU. Sloughed-off papillae may lodge in the entrance to the calyces, causing obstruction.

Papillary necrosis is difficult to detect on ultrasound unless advanced. It appears as prominent calyces with increased corticomedullary differentiation. IVU is the imaging method of choice (Fig. 7.15).

Congenital megacalyces

This is a congenital condition in which the PCS is dilated due to poor development of the papillae. The calyces are normally markedly enlarged but the cortex is normal and the ureters are of normal calibre and not dilated.[15] Occasionally this is associated with *congenital megaureter* in which the muscular layer of the ureter is atonic.

Differential diagnoses for fluid-filled renal masses are summarized in Table 7.2.

RENAL TRACT CALCIFICATON

Calcification within the kidney usually occurs in the form of stones. Smaller foci of calcium, which

Table 7.2 Differential diagnoses for fluid-filled renal masses

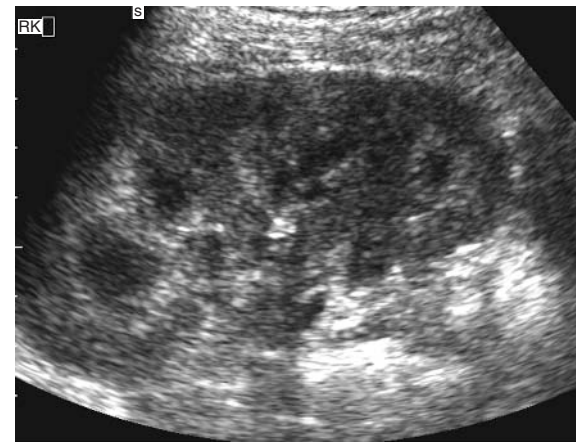
Solitary lesions	Simple cyst
	Infected or haemorrhagic cyst
	Hydatid cyst (rare)
Complex fluid lesions	Haematoma
	Abscess
	Lymphoma
	Necrotic primary or secondary tumour
	Tuberculosis
Pelvicalyceal system dilatation	Obstructive or non-obstructive causes
	Xanthogranulomatous pyelonephritis
Multiple cystic lesions	Polycystic or acquired cystic disease
	Multiple abscesses

do not shadow on ultrasound, are associated with conditions such as tuberculosis, xanthogranulomatous pyelonephritis, nephrocalcinosis or some neoplastic tumours.

Renal tract stones

Renal calculi are a common finding on ultrasound. They may be an incidental discovery in an asymptomatic patient; alternatively they may be present in patients with acute renal colic and complete or partial obstruction of the ipsilateral renal tract. They may be the cause of haematuria and can also be associated with urinary tract infections. The composition of calculi can vary. The common types include:

- *Calcium stones* are the most common type and are frequently associated with patients who have abnormal calcium metabolism.
- *Struvite (triple phosphate) stones* have a different composition of salts and are associated with urinary tract infections. They may form large, staghorn calculi (see below).
- *Uric acid stones* are rare, and tend to be associated with gout.
- *Cystine stones* are the rarest of all and result from a disorder of amino acid metabolism—cystinuria.



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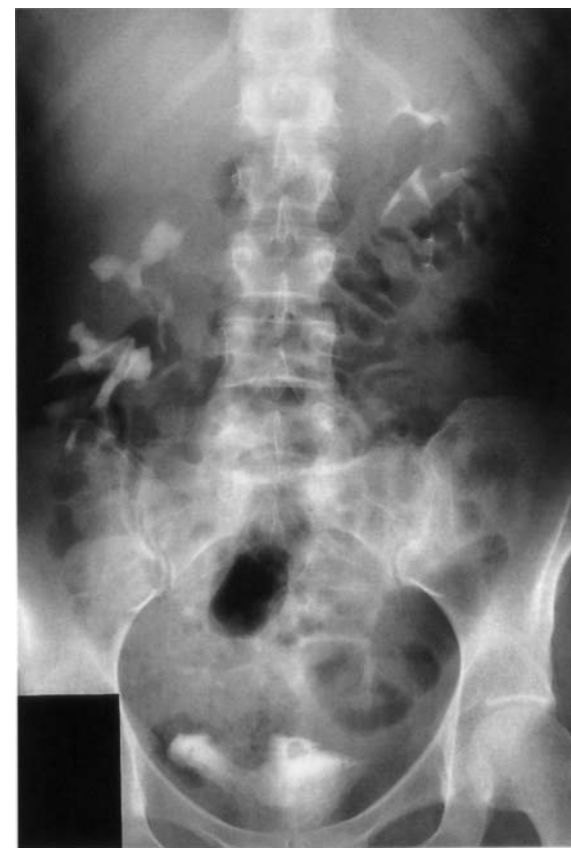

B

Figure 7.15 (A) Papillary necrosis. The calyces are mildly dilated with blunted, irregular margins and contain low-level echoes from sloughed papillae. (B) IVU of the same patient demonstrating the blunted calyces.

Ultrasound appearances

Most renal calculi are calcified foci located in the collecting system of the kidney. Careful scanning with modern equipment can identify over 90% of these.[16] Most stones are highly reflective structures which display distal shadowing (Fig. 7.16). The shadowing may, however, be difficult to demonstrate due to the proximity of hyperechoic sinus echoes distal to the stone, or due to the relatively small size of the stone compared to the beam width.

The identification of reflective foci in the kidney is complicated by the fact that the normal renal sinus echoes are of similar echogenicity. This means that small stones may be missed on ultrasound. Differentiation of stones from sinus fat and reflective vessel walls is dependent upon careful technique and optimal use of the equipment. The operator must adjust the technique to display the distal shadow by using a variety of scanning angles and approaches and by ensuring that the suspected stone lies within the (narrowest) focal zone of the beam. The higher the frequency used, the better the chances of identifying the stone.

Clearly the identification of large calculi is normally straightforward; however, for many of the reasons above, identification of small calculi can be difficult, especially in a patient with pain. Both false-positive and false-negative studies are well recognized. Although traditionally the plain film, that is kidneys, ureters, bladder (KUB), is often the first-line investigation for patients with suspected calculi, it is now being accepted that CT IVU is the best and most reliable diagnostic test for calculi detection (Fig. 7.16 C and D).

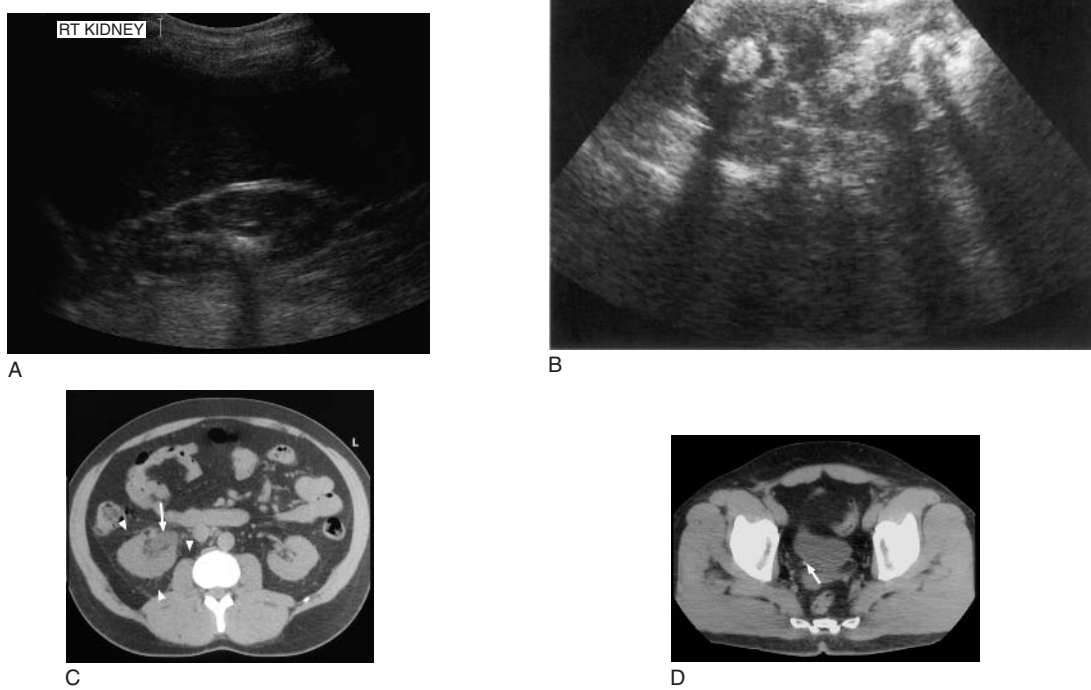

Figure 7.16 (A) A calculus within the PCS of the RK. Distal acoustic shadowing is easily seen. (B) A staghorn calculus fills the entire PCS of the kidney. A sagittal section through the lateral aspect of the kidney gives the impression of several separate stones, although this is, in fact, a single calculus. (C) CT IVU through the renal area. The right renal pelvis is mildly dilated (arrow) and a small amount of perirenal stranding is noted, suggestive of obstruction (arrowheads). (D) CT scan through the bladder showing a small calculus on the right (arrow) at the right vesicoureteric junction.

Ultrasound still has a major role, however, not just in calculus detection but in identifying the secondary effects, that is, hydronephrosis, and where necessary, guiding renal drainage. The PCS may be obstructed proximal to the stone. Obvious hydronephrosis may be present and a dilated ureter may be apparent when the stone has travelled distally. The stone can sometimes be identified in the dilated ureter, but this is unusual as the retroperitoneum is frequently obscured by overlying bowel. Plain X-ray and/or IVU are traditional essential adjuncts to investigating renal colic in these cases; however CT IVU is rapidly becoming accepted as one of the mainstream investigations.[17]

Early obstruction occurs *before* the PCS can become dilated, making the diagnosis more difficult on ultrasound. Occasionally there will be mild separation of the PCS to give a clue, but sometimes the kidney appears normal. Doppler ultrasound can help to diagnose obstruction in a non-dilated kidney, as discussed previously; however this may not always be definitive.

Staghorn calculi

These large calculi are so called because they occupy a significant proportion of the collecting system, giving the appearance of a staghorn on X-ray (Fig. 7.16B). They may be less obvious on ultrasound than on X-ray, casting a dense shadow from the PCS which may obscure any associated dilatation and can, in small, atrophied kidneys, be misinterpreted as shadowing from bowel gas. Because of the lobulated shape of the calculus it may appear as several separate calculi on ultrasound. A coronal section may therefore be more successful in confirming a staghorn calculus than a sagittal section.

Cystinuria

This rare metabolic disease causes crystals of cystine to precipitate in the kidneys and be excreted in the urine (Fig. 7.17). Cystine stones form in the kidneys and may result in obstruction.

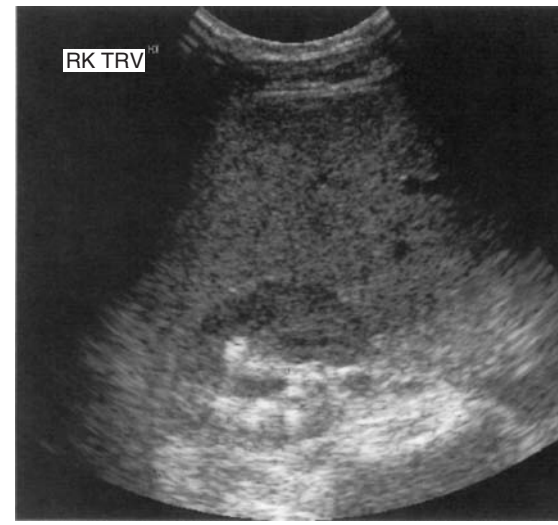

Figure 7.17 Cystinuria. TS through the RK. Small, highly reflective crystals of cystine are demonstrated.

Nephrocalcinosis

This term is used to describe the deposition of calcium in the renal parenchyma. It is most often related to the medullary pyramids and is frequently associated with medullary sponge kidney (see below). It may also be seen in papillary necrosis and in patients with disorders of calcium metabolism, e.g. hyperparathyroidism.

Ultrasound appearances

Nephrocalcinosis may affect some or all of the pyramids. A regular arrangement of hyperechoic pyramids are seen which may shadow if large calcific foci are present, but not if the foci are numerous and tiny, as they are smaller than the beam width (Fig. 7.18).

Less frequently, calcification is seen in the renal cortex.

Hyperparathyroidism

The (normally) four parathyroid glands in the neck regulate calcium metabolism in the body. Patients with *primary hyperparathyroidism* (due to an adenoma or hyperplasia of one or more of the parathyroid glands) have hypercalcaemia, which makes them prone to nephrocalcinosis or stones in the kidneys.

Secondary hyperparathyroidism is associated with chronic renal failure; hypocalcaemia, which results from the chronic renal failure, induces

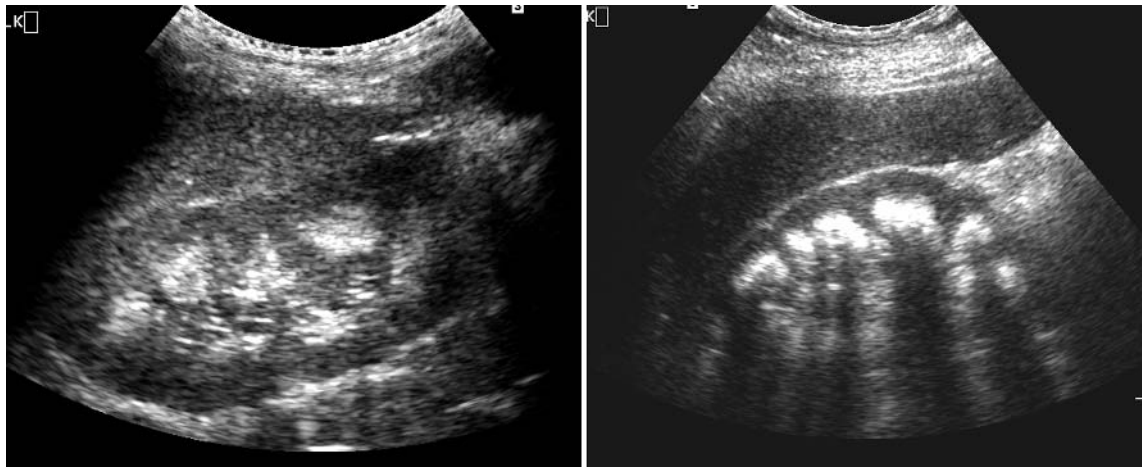

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Figure 7.18 (A) Nephrocalcinosis, demonstrating deposists of calcium within the renal pyramids which are too small to cast an acoustic shadow. (B) Calcification in the renal pyramids with strong acoustic shadowing.

compensatory hyperplasia of the parathyroid glands. There is a high incidence of hyperparathyroidism secondary to chronic renal failure in patients on dialysis; scintigraphy may demonstrate the region of increased activity and ultrasound is particularly suitable for demonstrating the enlarged parathyroid, guiding a diagnostic aspiration and, if necessary, ablating the gland with ethanol as an alternative to surgical removal. Alcohol ablation is generally reserved for those patients deemed to be a poor surgical risk.

RENAL TRACT INFLAMMATION AND INFECTION

The most common urinary tract infections are bacterial in origin, with viral and fungal infections being comparatively rare. The diagnosis is made by urinalysis after the patient presents with symptoms of dysuria, haematuria and/or suprapubic or renal angle pain. The origin of the infection may be via the blood stream (haematogenous) or the urethra (ascending). Ascending infections are more common in women due to their short urethra.

Ultrasound is often requested, particularly in children, to identify any unsuspected renal pathology which may be associated with the infection, for example a duplex collecting system, pelvic kidney.

Common conditions which may be identified on ultrasound include renal cystic diseases, calculi, obstructive uropathy, reflux and anatomical variants.

The infection may be either acute or chronic. Ultrasound signs of renal infection may be absent altogether, and this is the commonest scenario as the infective episode has often been successfully treated with antibiotics by the time the ultrasound scan is performed.

The infection may be confined to the bladder, that is *cystitis*, in which case low-level echoes and/or hyperechoic debris may be identified, or may have progressed to the kidneys. Scarring and/or cortical thinning may be present in cases of repeated infections (see *Chronic pyelonephritis* below).

Pyelonephritis

Acute pyelonephritis

Acute inflammation of the kidney rarely results in any ultrasound abnormality. Occasionally the kidney may be enlarged and hypoechoic, the contrast between the kidney and the hepatic or splenic parenchyma increasing due to oedema, but the ultrasound changes are generally subtle.

The normally clear differentiation between the cortex and the medullary pyramids may become indistinct, but again may go unrecognized.

CT is useful for detecting subtle inflammatory changes within the kidney.

Chronic pyelonephritis

This chronic inflammatory state is usually the result of frequent previous inflammatory/infective episodes.

The kidney may be small and often has focal scarring present. Scar tissue has the appearance of a hyperechoic, linear lesion which affects the smooth renal outline and crosses the renal cortex (Fig. 7.19A). (Do not confuse focal scarring with fetal lobulation: the latter is smooth, thin, continuous with the capsule and forms an indentation between the pyramids.)

The renal cortex is frequently thin in chronic pyelonephritis and may appear abnormally hyperechoic.

Bladder diverticula Repeated infections can cause the bladder wall to thicken and become trabeculated. In such cases, a bladder diverticulum may form, making treatment of subsequent infections particularly difficult. The diverticulum may harbour debris or stones and may fail to empty properly, often enlarging as the urine refluxes into it when the patient micturates (Fig. 7.19B).

Focal pyelonephritis

The presence of acute infection within the kidney may progress in focal regions of the renal parenchyma. This phenomenon is particularly associated with diabetics.

The ultrasonic changes are subtle, as in diffuse pyelonephritis, but it is possible to detect a slight change in echogenicity when it is surrounded by normal-looking parenchyma.

Focal pyelonephritis (sometimes called focal nephronia) may be either hypo- or hyperechoic compared with normal renal tissue. Depending on the size of the lesion, it may cause a mass effect, mimicking a renal tumour. The outline of the kidney is preserved, however (Fig. 7.19C).

The patient presents with fever and tenderness on the affected side and frequently has a history of urinary tract infection. A focal renal mass under these circumstances is highly suggestive of focal pyelonephritis and is also well demonstrated on CT.[18] It usually responds to antibiotic therapy and resolution of the lesion can be monitored with ultrasound scans. Focal pyelonephritis can progress to form an abscess in the kidney, which can normally be treated by percutaneous drainage and antibiotics.

Renal abscess

A renal abscess is generally a progression of focal inflammation within the kidney (see above). The area liquefies and may enlarge to form a complex mass with distal acoustic enhancement. Low-level echoes from pus may fill the abscess cavity, giving it the appearance of increased echogenicity, but it may also be hypoechoic. The margins of the abscess may be ill-defined at first but may develop a more obvious capsule as the lesion becomes established (Fig. 7.19F), this capsule often has an easily identifiable thick rim. Flow may be seen in the inflammatory capsule with colour Doppler, but not in the liquefied centre.

A renal abscess may mimic a lymphoma as both may be hypoechoic on ultrasound, and both may have either single or multiple foci.

The abscess may be intrarenal, subcapsular or perirenal. Frequently, drainage under ultrasound guidance is the preferred treatment; gradual resolution of the abscess can also be monitored with ultrasound.

Tuberculosis (TB)

Renal TB is an uncommon finding and a difficult diagnosis to make on ultrasound. The subtle inflammatory changes which affect the calyces in the early stages are best demonstrated with CT.

In the later stages ultrasound may show calcific foci and obstructed calyces as a result of thickened inflammatory calyceal walls, calcification and debris.

TB frequently spreads to other adjacent sites in the abdomen, including the psoas muscle and gastrointestinal tract.

The differential diagnosis is xanthogranulomatous pyelonephritis, which is often indistinguishable from TB on ultrasound, or a necrotic renal neoplasm.

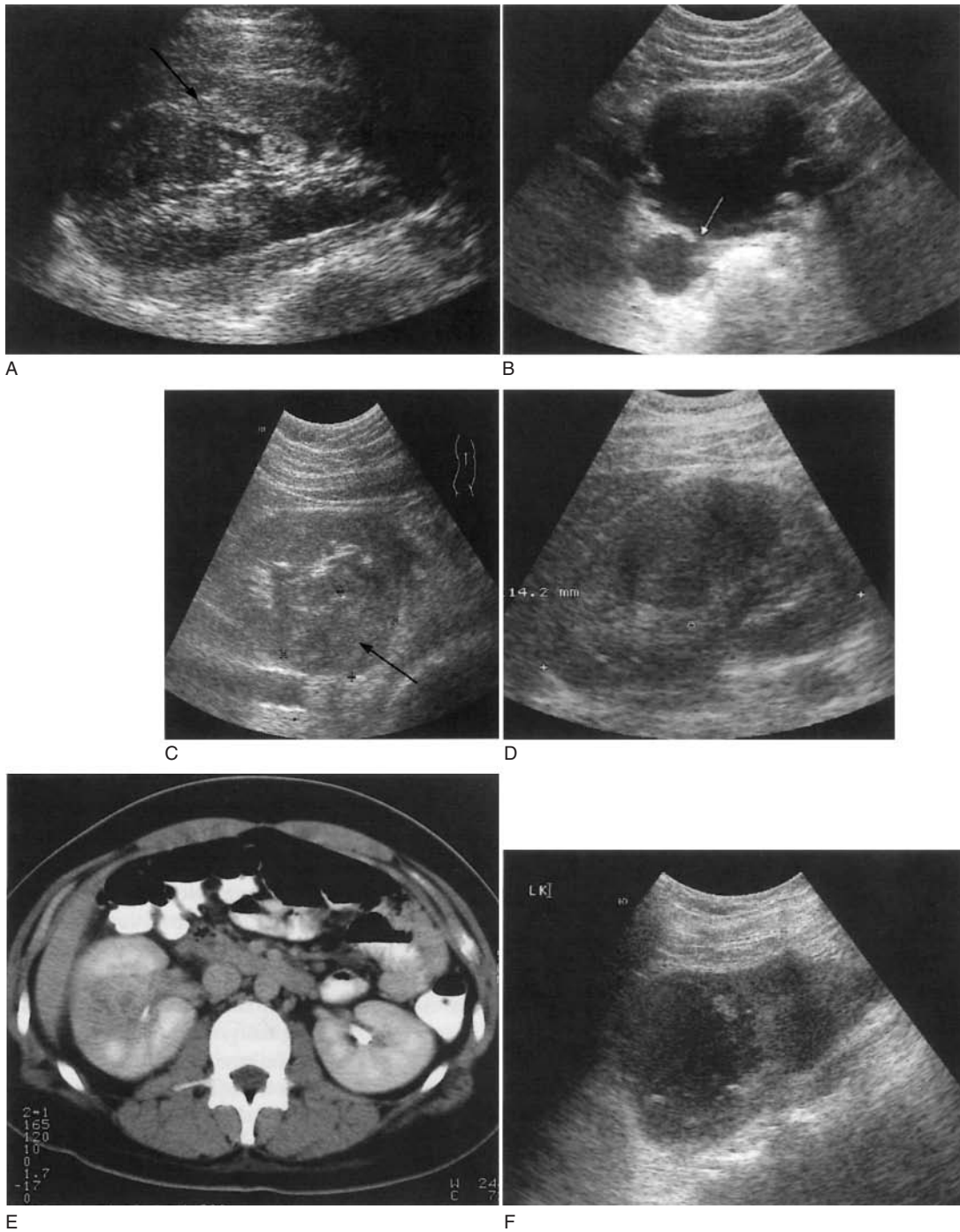


Figure 7.19 (A) Cortical scar tissue is demonstrated following repeated episodes of urinary tract infection. (B) A bladder diverticulum can be seen communicating with the bladder (arrow). The main bladder wall is trabeculated. (C) Focal pyelonephritis (arrow). This subtle area of altered echogenicity in the kidney slightly displaces the renal sinus echoes. (D) Another case of focal inflammation in an enlarged RK with an area of decreased echogenicity. (E) CT of case in (D) demonstrates the area with greater clarity. (F) Abscess in the LK containing low-level echoes from pus. The abscess capsule is irregular and thickened.

Xanthogranulomatous pyelonephritis (XGP)

This condition (which gets its name from the yellow colour of the kidney) is the result of renal obstruction by calculi in the pelvicalyceal system. Frequently, a staghorn calculus is responsible.

The kidney becomes chronically infected and the calyces enlarge and become filled with infected debris. The cortex may be eroded and thin (Fig. 7.20).

On ultrasound, these appearances are similar to TB or to a pyonephrosis. The latter is usually accompanied by a more severe, acute pain and fever whereas XGP or TB has a lower-grade, chronic pain.

CT may differentiate TB from XGP and is also more sensitive to extrarenal spread of disease.

Hydatid cysts

The *Echinococcus* parasite spends part of its life cycle in dogs. The larvae may be transmitted to humans through contact with dog faeces, finding their way to the lungs, liver and, less frequently, the kidneys.

The parasite forms a cyst which has a thickened wall, often with smaller, peripheral daughter cysts. Frequently the main cyst contains echoes.

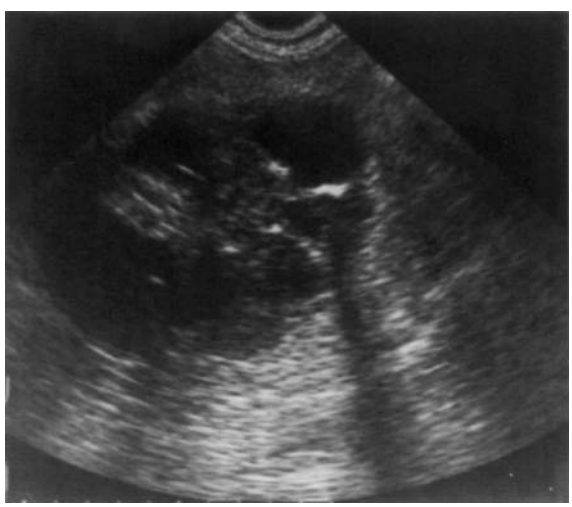


Figure 7.20 Xanthogranulomatous pyelonephritis. Numerous stones have obstructed the PCS, which contains debris. The renal cortex is thin, and the architecture of the kidney is difficult to recognize.

The condition is rare in the UK, but may be diagnosed when small, grape-like cysts are passed in the urine.

DIFFUSE RENAL DISEASE AND RENAL FAILURE

Most diffuse medical renal conditions have non-specific appearances on ultrasound, the kidneys often appearing normal in the early stages of disease. Renal failure may be acute or chronic and its causes are numerous. If acute, an increase in overall renal size may be observed and there may be a diffuse alteration in the renal echogenicity, however this can be either hypo-or hyperechoic compared with normal. Either increased or decreased corticomedullary differentiation may also be observed (Fig. 7.21). Although ultrasound is successful in detecting renal parenchymal disease, the acoustic changes are not specific and the cause must usually be diagnosed histologically,[19] ultrasound being invaluable in directing the biopsy procedure.

In chronic renal failure the kidneys shrink and the cortex thins. The end-stage kidney can be quite tiny and hyperechoic and may be difficult to differentiate from the surrounding tissues (Fig. 7.21C).

Depending on the cause, either one but generally both of the kidneys are affected.

Acute tubular necrosis

Acute tubular necrosis is the result of ischaemia, which destroys the tubules of the kidney, resulting in acute renal failure. It occurs when there is a sudden decrease in renal perfusion as a result of a severely hypotensive episode, for example, cardiac arrest, massive haemorrhage, drug toxicity or septicaemia.

Patients are treated temporarily by dialysis. Tubular damage is capable of regeneration once the blood supply and perfusion pressure return to normal, reversing the renal failure. If suspected, it is useful to perform a biopsy to determine the cause of renal failure, in order to plan further management.

On ultrasound the kidneys are normal in size or slightly enlarged. They may be completely normal in appearance, a not uncommon finding, although in some cases the echogenicity is altered,

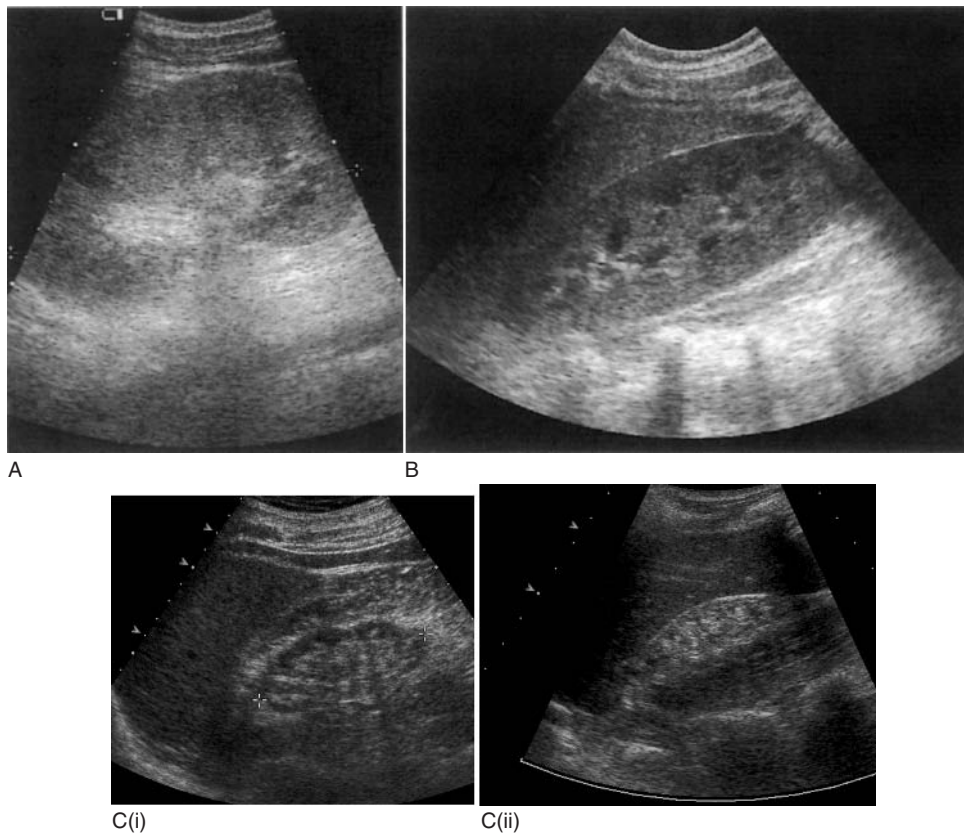


Figure 7.21 (A) Acute renal failure demonstrating an enlarged, diffusely hyperechoic kidney with loss of corticomedullary differentiation. (B) Acute renal failure in paracetamol overdose. The kidney is large (16 cm) and hyperechoic with increased corticomedullary differentiation. (C) Chronic renal failure. The kidney is shrunken with only a thin rim of cortical tissue remaining. The cortical rim may be of normal echogenicity (i) or hyperechoic (ii). The latter situation is more common.

sometimes having a hyperechoic cortex with increased corticomedullary differentiation.

Spectral Doppler can be normal or demonstrate increased arterial resistance with reduced or even reversed end diastolic flow.

Glomerulonephritis

Glomerulonephritis is an inflammatory condition which affects the glomeruli of the kidney. It may be either acute or chronic, and frequently follows prolonged infection.

Patients may present in acute renal failure, with oliguria or anuria, or with features of nephrotic syndrome such as oedema, proteinuria and hypoalbuminaemia.

Depending upon aetiology, acute renal failure may be reversible or may progress to chronic renal failure requiring dialysis.

Glomerulonephritis can be caused by numerous mechanisms:

- *Immunologic mechanisms*, for example in systemic lupus erythematosus (SLE) or acquired immune deficiency syndrome (AIDS)
- *Metabolic disorders*, for example diabetes
- *Circulatory disturbances*, for example atherosclerosis or disseminated intravascular coagulation (DIC).

As with acute tubular necrosis, the ultrasound appearances are non-specific. In the acute stages the kidneys may be slightly enlarged; changes in the echogenicity of the cortex may be observed. In the chronic stages the kidneys shrink, become hyperechoic, lose cortical thickness and have increased corticomedullary differentiation.[19]

Medullary sponge kidney

In medullary sponge kidney the distal tubules, which lie in the medullary pyramids, dilate. This may be due to a developmental anomaly but this is not certain. In itself it is usually asymptomatic and therefore rarely seen on ultrasound. However, the condition is prone to nephrocalcinosis, particularly at the outer edges of the pyramids, and stone formation (see above), which may cause pain and haematuria.

Amyloid

In amyloid disease, excess protein is deposited in the renal parenchyma, predominantly the cortex. This causes proteinuria and may progress to nephrotic syndrome (oedema, proteinuria and hypoalbuminaemia).

Amyloidosis can cause acute renal failure and is particularly associated with long-standing rheumatoid arthritis.

As with other diffuse renal diseases, the acute stage may cause renal enlargement and the parenchyma tends to be diffusely hyperechoic. By the time the chronic stage of disease has been reached, the kidneys become shrunken and hyperechoic, in keeping with all end-stage appearances.

The renal biopsy

(*See Chapter 11.*) Biopsy is rarely merited in end-stage renal failure, as the only treatment is dialysis or renal transplantation. Small kidneys, below 8 cm in length therefore, are almost never subjected to biopsy.

Histology is required when the kidney is potentially curable, such as in cases of acute disease, or when a specific knowledge of aetiology is paramount.

RENAL VASCULAR PATHOLOGY

Renal artery stenosis (RAS)

Stenosis of the renal artery is due to atreroscleroticdisease in the vast majority of patients, or to fibromuscular dysplasia of the arterial wall in the younger, generally female patient. RAS may cause hypertension and may eventually cause renal failure. It is frequently bilateral, and is responsible for up to 15% of patients who require long-term dialysis. It is associated with aortic aneurysm, neurofibromatosis or can be traumatic in origin.

Stenosis generally affects the main vessel at its origin and involves the aorta (ostial) or occurs within 1 cm of its origin (non-ostial). It can occur in both native and transplanted organs. It is frequently bilateral.

Ultrasound appearances of RAS

If the stenosis is long-standing and/or severe, the kidney is likely to be small. Loss of renal mass is associated with a stenosis of 60% or greater.[20] However, the ultrasound appearances are often normal with milder grades of RAS.

Ultrasound has traditionally had a limited role to play in the diagnosis of RAS and digital subtraction angiography is still considered the gold standard, although magnetic resonance angiography (MRA) is now regarded as the first-line imaging modality of choice. However, colour and spectral Doppler techniques have greatly enhanced the usefulness of ultrasound, particularly in experienced hands.[21]

At the site of a stenosis, an increase in peak systolic velocity may be found (greater than 1.5–1.8 m/s) with poststenotic turbulence. Although, it is often not technically possible confidently to examine and sample the whole main renal artery and thus make a definitive diagnosis, it nevertheless remains the best Doppler technique for diagnosis.

In addition, the intrarenal vessels may show changes on colour or power Doppler which are indicative of a downstream stenosis. Within the kidney, the perfusion may appear subjectively reduced in the number of vessels and velocity of flow and it may be necessary for the operator to use a low PRF value to detect blood flow. This is

very subjective and variable. The spectral waveforms of arteries distal to the stenosis also reflect changes which suggest a proximal stenosis; the normally fast systolic upstroke is replaced by a delayed *parvus tardus* pattern (Fig. 7.22), making the waveform less pulsatile with a rounded envelope.[22]

This type of waveform can be appreciated subjectively, but quantitative measurements may be used to support the diagnosis. *The acceleration time (AT)* or *acceleration index (AI)* is the most common; a normal AT is < 0.07s, and a normal AI > 3 m/s.

The actual value of these indices, however, does not reflect the severity of stenosis; unfortunately stenoses of < 70–80% narrowing do not normally demonstrate the parvus tardus effect (although these tend to be less clinically significant) and these spectral phenomena may be obscured altogether if the vessels are rigid and severely diseased[23] or if a good collateral circulation has developed. In such cases the Doppler result is falsely negative and the operator should bear this in mind when attempting to exclude RAS.

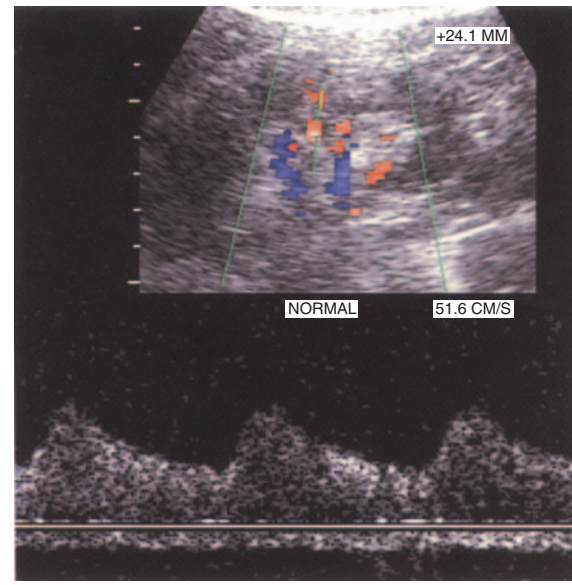
Renal artery occlusion may occur as a result of further progression of the same disease process which causes stenosis. Doppler will confirm the lack of renal perfusion. The kidney is likely to be small as a result of gradually deteriorating arterial perfusion.

Management of RAS

Stenosis of the main renal artery is amenable to percutaneous angioplasty and/or stenting, which can effect a cure or more realistically stabilize or slow disease progression. A postangioplasty ultrasound scan can confirm vessel patency, and may play a role in monitoring the patient for disease recurrence. For those with deteriorating function, for whom percutaneous techniques have failed, renal failure will ultimately necessitate dialysis. Renal transplant is a viable option, particularly for those who have been treated in the long term.

Renal vein thrombosis

This can occur when chronic renal disease is already present or in cases of a coagulation disor-



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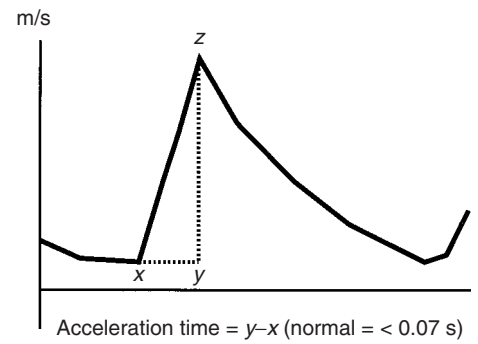
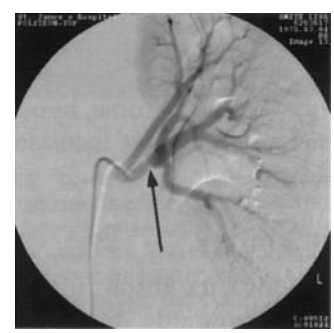


Acceleration time = $y–x$ (normal = < 0.07 s)

Acceleration index = $\frac{z–x}{y–x}$ (normal = > 3 m/s)

B



C

Figure 7.22 (A) Renal artery stenosis. The kidney is small, with subjectively reduced perfusion on colour Doppler. The spectrum displays the parvus tardus pattern. (B) Measurement of the acceleration time. (C) The arteriogram in case (A) confirms a stenosis (arrow).

der with increased tendency to thrombose, for example polycythaemia. It is frequently associated with nephrotic syndrome. Other associated factors include the oral contraceptive pill and the use of steroids.[24]

Tumour thrombus from RCC is also prone to invade the ipsilateral renal vein, and sometimes may extend into the IVC and even renal artery.

Thrombus in the renal vein, whether secondary to a malignancy or thrombocythaemia can travel up the IVC forming a source of emboli. If non-malignant, the thrombus may be successfully treated medically and the renal function can be preserved even if the vein is totally occluded.

Ultrasound appearances

It is often possible to see echo-poor thrombus within a dilated renal vein, running beside the renal artery in an axial section through the renal hilum (Fig. 7.9). Colour Doppler confirms absent venous flow.

Perfusion within the kidney itself is reduced and there may be a highly pulsatile arterial waveform with reversed diastolic flow (Fig. 7.23), although this is not commonly seen in the native kidney.

If the thrombus produces a total and sudden occlusion, the kidney becomes oedematous and swollen within the first 24 h. Eventually it will shrink and become hyperechoic.

Partially occluding thrombus is more difficult to diagnose as the changes in the kidney may not be apparent. However, a non-dilated renal vein with good colour Doppler displayed throughout has a high negative predictive value.

Incomplete thrombosis may still demonstrate venous flow within the kidney, although the arterial waveforms are of lower velocity than normal, with a marked reduction in the systolic peak.[25] Forward diastolic flow may be preserved at this stage.

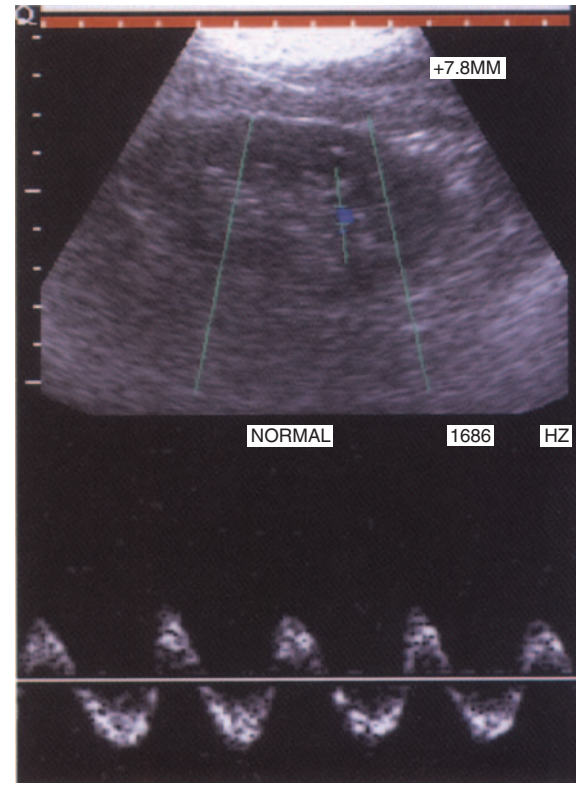


Figure 7.23 Renal vein thrombosis. Small shrunken kidney (6 cm) demonstrating hardly any perfusion, apart from a tiny interlobar artery with forward and reverse flow.

Arteriovenous fistula

These lesions can occur at the site of a biopsy and are recognized on colour and spectral Doppler by localized vessel enlargement with turbulent, sometimes high-velocity flow. A 'pool' of colour flow is often present. The vein may show a regular, pulsatile pattern and be dilated. These iatrogenic fistulae usually resolve spontaneously and are clinically insignificant. If bleeding is a clinical problem and is ongoing, recurrent and/or severe then embolization is the treatment of choice.

Ultrasound in dialysis

Patients with chronic renal failure may undergo either haemodialysis (in which a subcutaneous arteriovenous shunt is created, often in the wrist) or continuous ambulatory peritoneal dialysis (CAPD), in which a catheter is inserted through the abdominal wall. Ultrasound may be used to assess the patency of the shunt or catheter, and may identify localized areas of infection along the CAPD tract which can be drained under ultrasound guidance if necessary.

Ultrasound may also be used to diagnose acquired cystic kidney disease in long-term dialysis patients (see above).

RENAL TRAUMA

The severity of trauma to the kidney may vary significantly and therefore a range of findings can be seen with ultrasound. A direct injury can rupture the kidney. This will result in blood and/or urine leaking out into the perinephric space. The nature of the fluid can be determined by ultrasound guided aspiration. A large urinoma or haematoma may be drained percutaneously. The main renal vessels may also be damaged, causing lack of perfusion (see Chapter 10).

Causes of haematuria are listed in Table 7.3.

RENAL TRANSPLANTS

Although there are a number of treatment choices for patients with renal failure including peritoneal and haemodialysis, undoubtedly the treatment of choice is renal transplantation.

From the very early days of Carrel's experimental attempts at transplantation in the 1900s (resulting in the Nobel Prize of 1912[26]), to the un-immunosuppressed allografting of the 1950s, the more successful and encouraging outcome of twin to twin transplants,[27] a better understanding of tissue rejection and the introduction of azathioprine and steroid in 1963,[28] and more specifically ciclosporin A by Calne in the 1970s,[29] have all contributed immensely to slow but positive progress in this field. Improvements in surgical technique, newer, more effective and less toxic anti-rejection therapy, the routine use of ultrasound in the 1970s and then Doppler a decade later, and the development of interventional radiology have all combined to make this the successful operation and clinical outcome we now take so much for granted.

Although many different imaging modalities are available, ultrasound is the single most useful investigation in the postoperative monitoring of the transplant. Amongst its many roles, it is sensitive to early PCS dilatation, can be used to guide biopsy procedures and to guide the drainage of fluid collections and placement of nephrostomy tubes.

An early, baseline scan is an essential part of the postoperative management, and serial scans are to be recommended.

Table 7.3 Causes of haematuria

- Urinary tract infection
- Stones
- Neoplasm (*renal cell or transitional cell carcinoma in the kidney, ureter or bladder*)
- Prostatic pathology (*benign hypertrophy or carcinoma*)
- Renal cyst haemorrhage
- Papillary necrosis
- Glomerulonephritis
- Trauma
- Tuberculosis
- Renal infarct

Normal anatomy

Most renal transplants are *heterotopic*, that is they are placed in addition to the diseased, native kidneys, which remain in situ.

The transplanted organ is usually positioned in the iliac fossa anterior to the psoas and iliacus muscles. It lies outside the peritoneal cavity.

Within the UK the majority of transplanted kidneys are cadaveric, and are harvested with their main vessels intact, which are then anastomosed to the recipient iliac artery and vein.

Normal ultrasound appearances

The transplanted kidney is particularly amenable to ultrasonic investigation; its position relatively near to the skin surface allows a high frequency transducer (5 MHz) to be used for better detail. For visualization of the vasculature or origins of the transplant vessels a 3.5–4.0MHz probe is normally required.

The ultrasonic appearances of the transplant kidney are the same as expected for a native kidney, allowing for the higher resolution [Fig. 7.24]. The transplant kidney should be assessed in the same way as the native organ, that is in two planes. Features to be observed include:

- *Morphological appearances* This should include an assessment of the relative echogenicity of the cortex, medulla and renal sinus and corticomedullary differentiation. Focal or diffuse changes in echogenicity may be observed, but are non-specific findings associated with inflammation, infection or infarction.
- *Size* Changes in renal size may be significant in transplanted organs; it is useful to calculate the renal volume, circumference or area, rather than just relying on the length.
- *PCS dilatation* Even mild PCS dilatation may be significant, as it may represent an early obstructive process. The bladder should be empty before assessing the PCS, to eliminate physiological dilatation. Any degree of hydronephrosis should be correlated with the clinical findings and biochemistry;

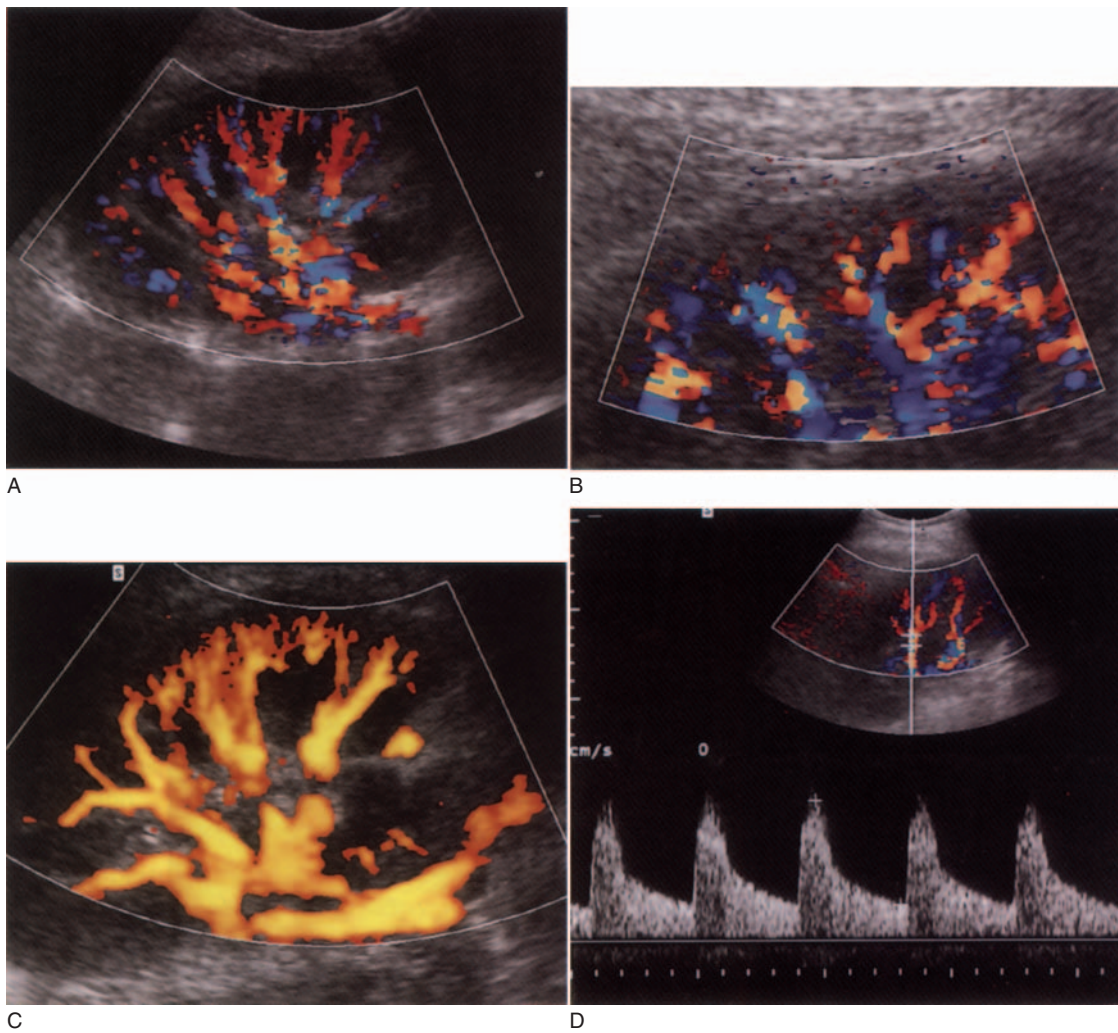
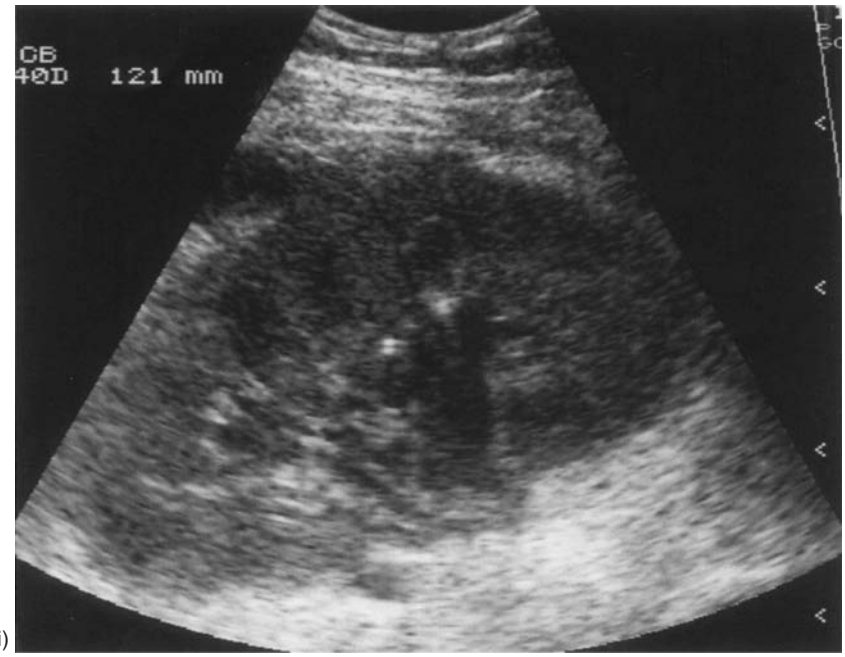


Figure 7.24 (A) Perfusion within the transplanted kidney is easily displayed. A higher frequency may be used, as the kidney is usually superficially situated in the iliac fossa. (B) Same kidney as (A). The Doppler sensitivity has been increased to demonstrate tiny arcuate vessels at the periphery of the kidney. (C) The increased sensitivity of power Doppler is valuable in demonstrating perfusion in the transplanted kidney. (D) Normal spectrum from the interlobar renal artery, demonstrating good end-diastolic flow (EDF) (low resistance) with a vertical systolic upstroke.

(Continued)

E(i)



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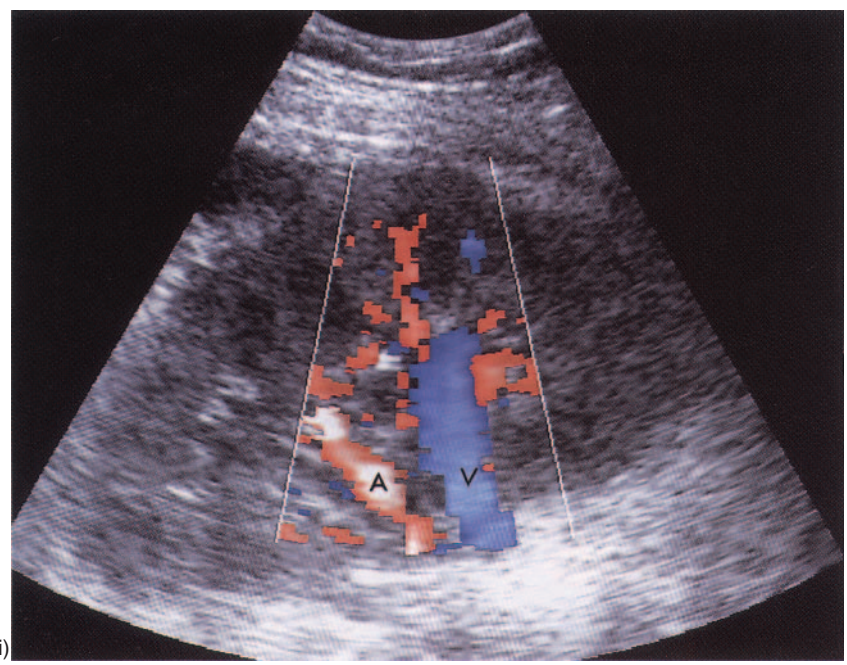


Figure 7.24 cont'd (E) i, large vessels at the hilum may mimic dilatation; ii, colour Doppler demonstrates this is the main renal vein.

hydronephrosis in isolation is not a reason for nephrostomy.

- *Vascular anatomy* The main transplant artery and vein are anastomosed to the recipient's external iliac artery and vein respectively and can normally be visualized throughout their length. Overall global perfusion can be assessed with colour Doppler and the smaller vessels at the periphery of the kidney (Fig. 7.24) should be discernible. The normal spectral Doppler waveform is a low-resistance waveform with continuous forward end diastolic flow.
- *Perirenal fluid* A small amount of free fluid is not unusual postoperatively. This usually resolves spontaneously. Fluid collections around the kidney are a common complication. They may resolve on further scanning; drainage is only peformed for good clinical reasons (see below).

Postoperative complications

Ultrasound has an essential role in assessing the transplant and makes a significant contribution towards graft survival through the early recognition of postoperative complications. Complications are varied and include acute rejection, ureteric obstruction, vascular occlusions, perirenal fluid collections, renal dysfunction (of various aetiologies) and infection. Drug toxicity from the immunosuppressive therapy can also compromise graft function. Finally, in the long term, the original disease, for which transplantation was performed, may recur.

Complications can be divided into three main categories: immediate postoperative complications, primary and secondary renal dysfunction.

- *Immediate*
  —non-perfusion, normally the result of an occluded or twisted renal artery; correction is surgical
  —haematoma
- *Primary dysfunction*
  —non-perfusion (arterial occlusion), total or lobar
  —acute tubular necrosis
  —renal vein thrombosis
  —obstruction
  —acute or accelerated acute rejection
- *Secondary dysfunction*
  —acute rejection
  —ciclosporin nephrotoxicity
  —acute tubular necrosis
  —obstruction
  —RAS
  —postbiopsy fistula
  —infection
  —chronic rejection.

Renal transplant dilatation

A mild degree of PCS dilatation is normal postoperatively, due to oedema at the site of the vesicoureteric anastomosis. This phenomenon is usually transient, and serial scans in conjunction with biochemistry (urea, creatinine) is normally all that is required. More severe dilatation may be indicative of obstruction, especially if the individual calyces are also dilated. A trend of increasing dilatation is a poor prognostic indicator. A ratio between the area of the PCS and the renal outline in two planes, the *dilatation index*, has been found to predict obstruction and differentiate obstructive from non-obstructive dilatation[30] (Fig. 7.25).

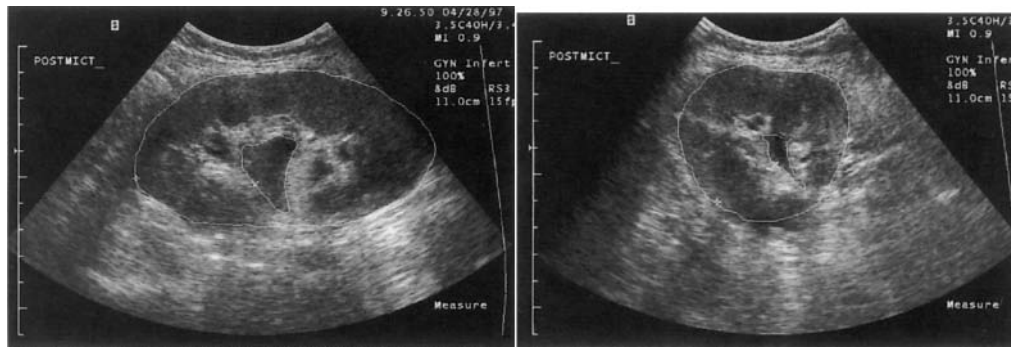
The degree of dilatation of the PCS correlates well with the severity of obstruction.

Obstruction of the transplant kidney may be due to an ischaemic related stricture at the vesicoureteric anastomosis, or may be the result of a blood clot or infected debris in the ureter. Haematoma or debris within the PCS may appear echogenic but requires to be differentiated from fungal balls.

Percutaneous nephrostomy is the method of choice to relieve obstruction.

Rejection

This can be acute or chronic. Acute rejection may be responsible for delayed graft function whereas chronic rejection is a gradual deterioration in renal function that may begin any time after 3 months of transplantation. Ongoing episodes of acute rejection should raise the possibility of non-compliance with therapy. Acute rejection cannot be differentiated on ultrasound from other causes of delayed function, particularly acute tubular necrosis, and therefore biopsy is invariably necessary.



A B

Figure 7.25 (A) LS and (B) TS of a dilated transplant kidney, showing the measurements used to calculate the ratio between the dilated PCS and the kidney. This kidney was dilated but not obstructed.

Pathologically, rejection can be either cellular (98%) or vascular (now accounting for only 2% of cases). Improved immunosuppressive therapy has greatly reduced the problems of rejection.

Ultrasound appearances of rejection

These are varied and non-specific. In the majority of cases the kidney appears normal; however, grey-scale findings include enlargement due to oedema (this change is subtle in the early stages and not a reliable ultrasonic indicator), increased cortico-medullary differentiation with prominent pyramids, infundibular thickening (thickening of the PCS walls) and decreased fat in the renal sinus[31] (Fig. 7.26). These findings are subjective, non-specific and limited in the diagnosis of rejection.

In chronic rejection there may be an overall increase in the echogenicity of the kidney with reduced corticomedullary differentiation. Eventually the kidney will shrink. The Doppler resistance indices are increased in rejection but, again, this finding is non-specific[32] (see Table 7.4) (Fig. 7.26B). In general, the higher the RI or PI, the more likely is the diagnosis of acute rejection.

The cause of renal dysfunction is established by biopsy.

Fluid collections associated with transplantation

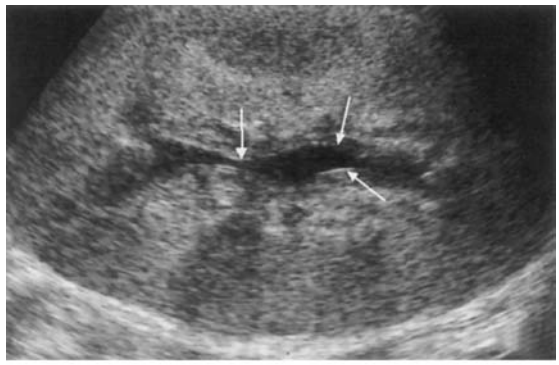
Up to 50% of renal transplants will demonstrate perirenal fluid.[33] The size of the collection should be monitored with ultrasound, as significant growth may require intervention.

Table 7.4 Causes of high-resistance Doppler (low EDF) in renal transplants

Cause	Characteristics
Acute rejection	Does not occur in the first 48 hours
ATN	Always occurs in the first 48 hours
Obstruction	Has a relatively *slight* increase in RI and is accompanied by pelvicalyceal system dilatation
Ciclosporin nephrotoxicity	Has to be prolonged and severe to affect the EDF and blood levels.
Renal vein thrombosis	A late complication of renal transplants has a characteristic reversed EDF pattern. The artery has a low-velocity systolic peak in the early stages. No venous flow identified
Perirenal fluid collections	Compression of the kidney causes an increase in intrarenal pressure

While it is not possible to classify the collection on the ultrasound appearances alone, the clinical picture, including the time interval following transplantation, can often be helpful.

- *Lymphocoele* The commonest perirenal fluid collection, lymphocoeles usually occur several weeks or months after the transplant. They may resolve spontaneously but occasionally require percutaneous drainage if large. They may compress the kidney, causing an increase in vascular resistance on spectral Doppler (Fig. 7.27). The collection is anechoic but may contain loculations or septa. If treated, then



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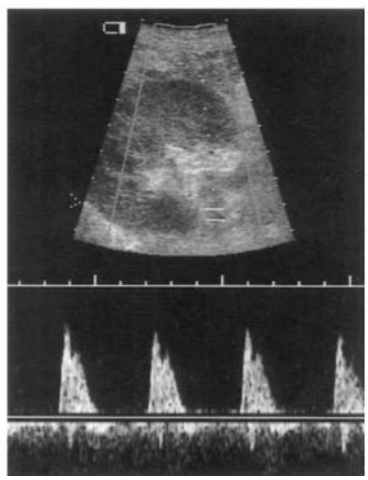


B

Figure 7.26 (A) Transplant rejection: peri-infundibular thickening (arrows) is demonstrated and the renal parenchyma looks abnormally hyperechoic with increased corticomedullary differentiation. (B) The Doppler indices are raised in this rejecting kidney, with no EDF. Loss of corticomedullary differentiation is noted in the kidney.

surgical laparoscopic marsupialization is the treatment of choice.

- *Haematoma* An immediate postoperative phenomenon which usually resolves spontaneously. If the haematoma is due to an anastomotic leak at the main artery or vein, it can compress the renal vein, causing thrombosis in rare cases. On ultrasound, the haematoma can appear hyperechoic and ill-defined in the early stages. As it resolves and liquefies, the margins become more defined and the centre becomes anechoic. Hyperechoic blood clots and strands of fibrin may be seen within the haematoma.

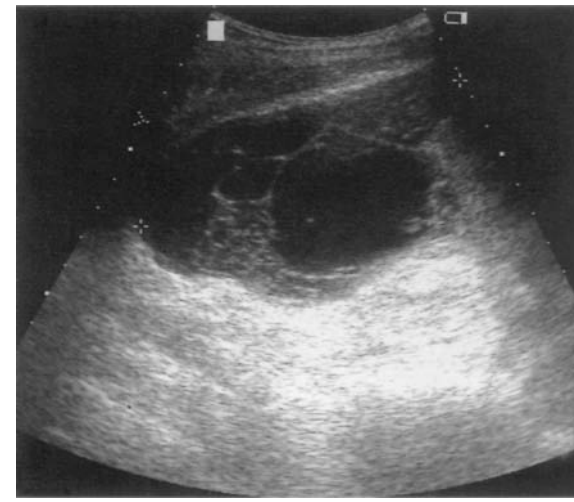


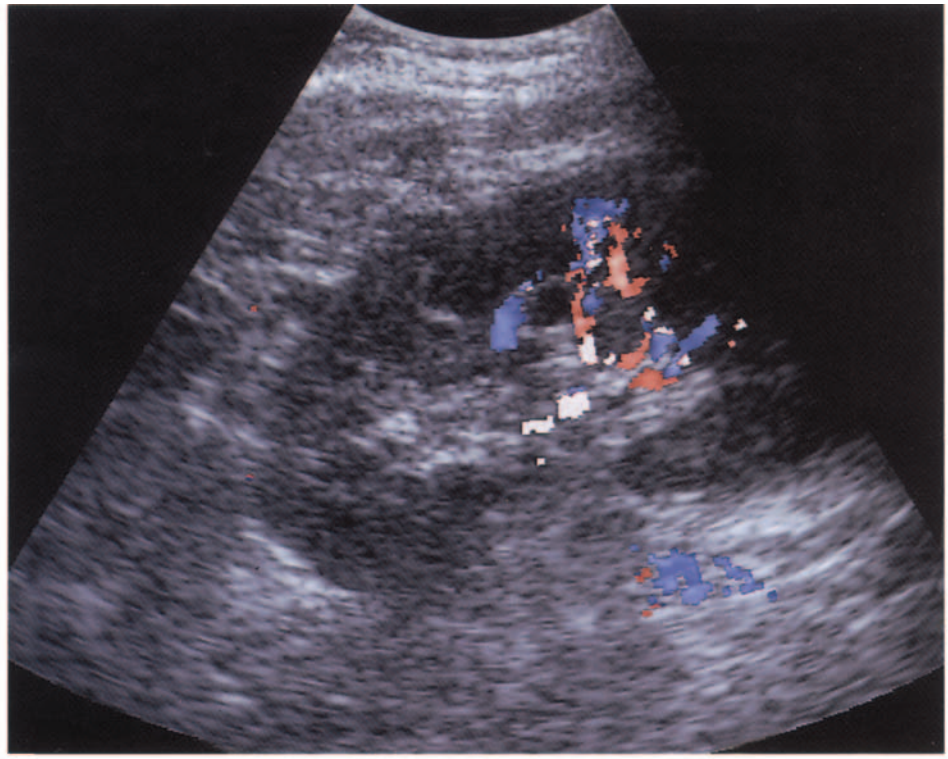
Figure 7.27 A lymphocoele adjacent to the upper pole of a transplant kidney. Ultrasound monitored the gradual resolution of this collection.

- *Urinoma* This occurs as a result of an anastomotic leak in the ureter. Urinomas are uncommon, but may progress to urinary ascites. They occur early following the surgical procedure, unlike lymphocoeles.
- *Abscess* If any of the above fluid collections becomes infected, this leads to an abscess. Hyperechoic debris can be seen in the collection and this may be treated with percutaneous drainage.

Vascular complications

Vascular occlusion

Colour and spectral Doppler are essential for the diagnosis of postoperative vascular complications. Non-perfusion may be total or lobar (Fig. 7.28). Focal areas of hypoperfusion may be due to oedema in focal infection, arteriovenous fistula or severing of an accessory artery during harvesting of the transplant or at the time of implantation. Total vascular occlusion is rare, but occurs early. Patients may be asymptomatic and non-perfusion of the transplant may be inadvertently seen on either a routine scan or isotope study. Graft nephrectomy is the most likely outcome. Conversely, the appearance of good renal perfusion throughout the kidney on colour or power Doppler does not necessarily indicate normal vascularity and severe



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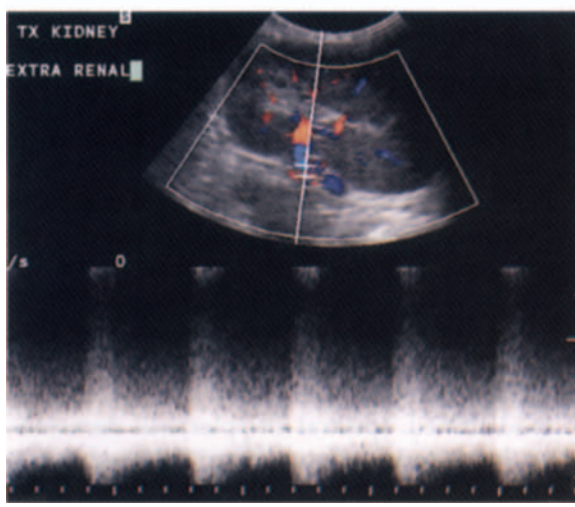

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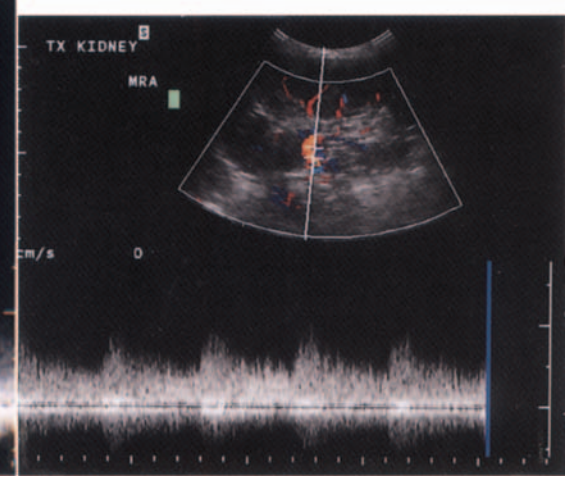


C

Figure 7.28 (A) Lack of perfusion in the upper pole due to rejection. (B) High-velocity jet at the site of a magnetic resonance angiography stenosis. This patient had had increasing, badly controlled hypertension since his transplant. (C) The same kidney, demonstrating turbulence distal to the site of stenosis.

vascular rejection or acute tubular necrosis can be present under such circumstances.[34]

Vascular complications can include arterial stenosis or thrombosis, venous stenosis or thrombosis, pseudoaneurysms and arteriovenous fistulae.[33]

Renal artery stenosis

This generally occurs at the site of the anastomosis close to the iliac artery but can also occur along the length of the artery or even affect the intrarenal branches. The patient may present with severe, difficult-to-control hypertension, graft dysfunction, or both. Alternatively the patient's renal function may deteriorate following angiotensin-converting enzyme inhibitor therapy and this is also an indication of a possible underlying RAS. Careful Doppler examination is now the accepted first-line investigation in the diagnosis of RAS.

In most cases it is possible to trace the artery back to its anastomosis with the iliac artery, using colour Doppler. If the site of the stenosis is identified, spectral Doppler will demonstrate an increase in peak systolic velocity at the lesion, followed by poststenotic turbulence (Fig. 7.28, B,C). This can be difficult to pinpoint with MRA, especially if bowel is overlying the vessel.

A delayed systolic rise (the *parvus tardus* waveform) can be identified in the intrarenal spectral Doppler waveforms, as for the native kidney (see above). The diagnosis however is primarily made on the peak systolic velocity within the renal artery. A value of < 2.5 m/s is normal while > 2.5 m/s constitutes RAS. If the stenosis is severe, it may be difficult to identify colour flow in the kidney and the waveform may be reduced in velocity with a tiny, damped trace in the main vessel.

A stenosis affecting an interlobar artery may result in focal, segmental changes in the kidney.

In general, contrast angiography is only used to grade and treat stenoses after a positive ultrasound scan, or when a high index of clinical suspicion persists, despite a negative ultrasound.[33]

Renal vein thrombosis

The occlusion may be partial or complete and the venous Doppler spectrum may therefore be absent (Fig. 7.29).

If venous thrombosis is partial, the arterial spectral waveform becomes very pulsatile, with reverse end diastolic flow; in the clinical setting of an oliguric patient with a tender graft in the early postoperative period, this is almost pathognomonic for RVT.

During the early stages, when thrombosis is incomplete, venous flow may be seen in the kidney, but the artery is of reduced velocity.[25]

The ultrasound findings of renal vein thrombosis may be indistinguishable from severe rejection; however venous flow is generally unaffected in the latter.

Thrombosis is rare, occurring typically in the immediate postoperative period.[33] It may be associated with a faulty venous anastomosis, secondary to compression of the vein, for example by a large, perivenous collection, or the patient may have an increased thrombotic tendency for a number of reasons.

Pseudoaneurysms and arteriovenous fistulae

These may sometimes form as a result of vascular damage during biopsy procedures. They are usually not significant and tend to resolve spontaneously (Fig. 7.30A).

An arteriovenous fistula shows an irregular knot of vessels on colour or power Doppler with a pulsatile venous waveform and high peak and end diastolic velocity in the feeding artery. A large draining vein may also be seen.

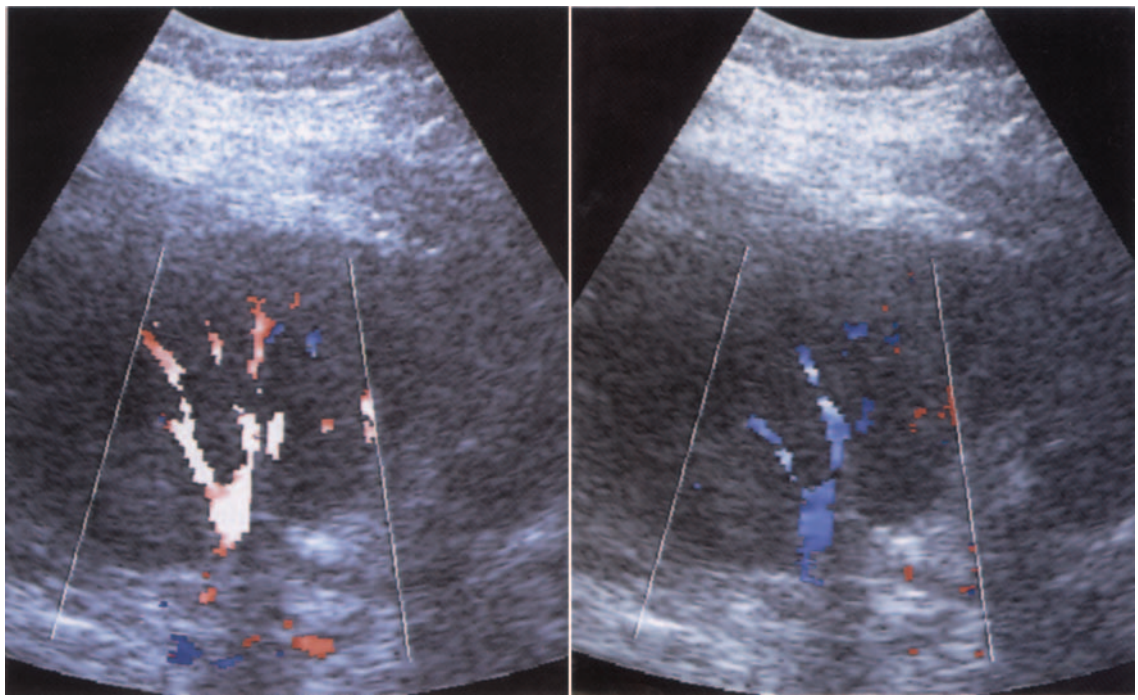
A pseudoaneurysm may appear cystic on the grey-scale image, but will demonstrate filling on colour Doppler with a pulsatile flow velocity waveform (Fig. 7.30B, C). Careful biopsy technique helps to avoid such lesions (see Chapter 11).

Infection

This is characterized by swelling of the uroepithelium, especially with fungal infections. Fungal balls may be visible as relatively hyperechoic structures within the PCS (Fig. 7.31).

Acute tubular necrosis

This may demonstrate prominent medullary pyramids on ultrasound, with low end diastolic flow. Reverse end diastolic flow is uncommon but recognized. A biopsy is required for confirmation.



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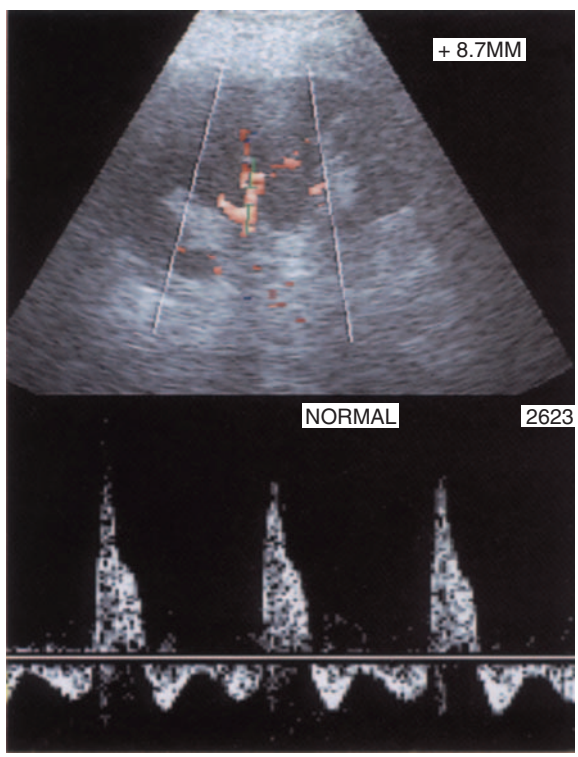
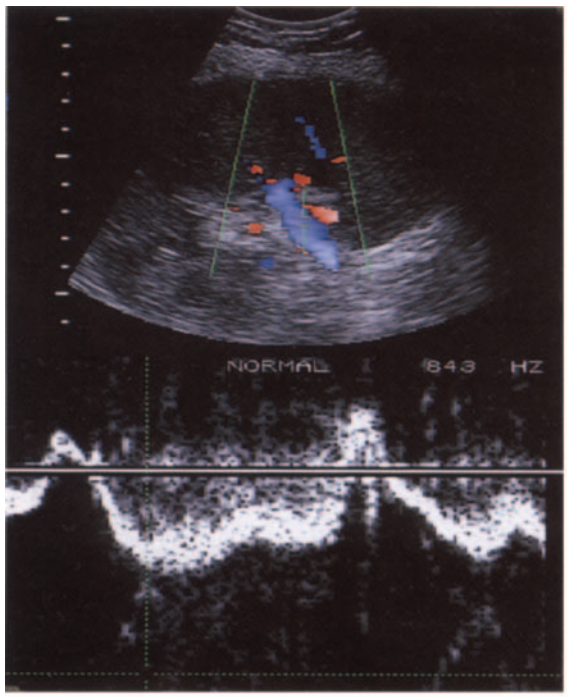


C

Figure 7.29 Renal vein thrombosis. (A) Poor transplant perfusion, with scanty demonstration of arterial flow. (B) The same vessels demonstrate reversal of flow at the end of the cardiac cycle. (C) The bidirectional flow is demonstrated on the spectral trace.



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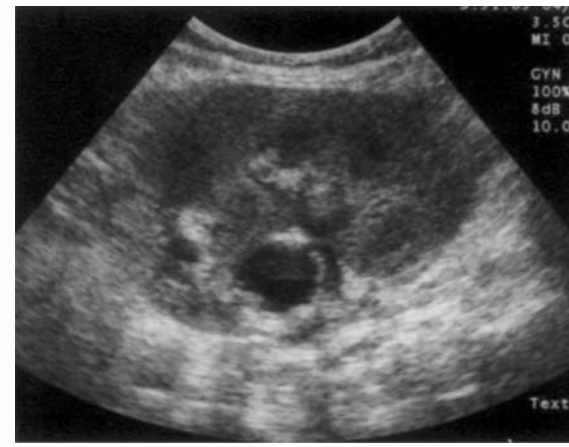

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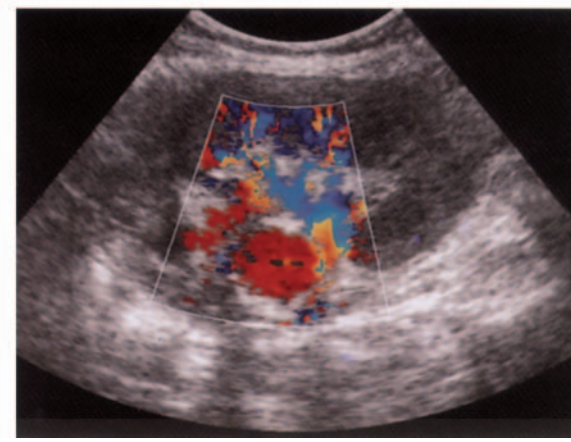

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Figure 7.30 (A) Pulsatile venous waveform is seen as a result of a small arteriovenous fistula following biopsy procedure. (B) This renal artery aneurysm appears cystic on grey-scale. (C) Colour Doppler demonstrates arterial flow.

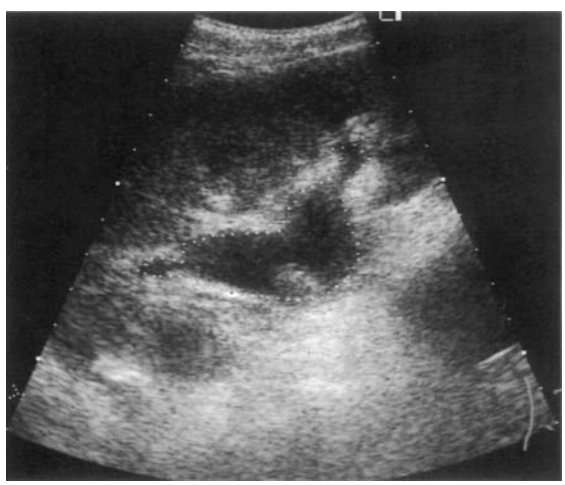

Figure 7.31 Fungal ball in the dilated PCS.

Ciclosporin nephrotoxicity

The toxic nature of the immonosuppressive regime requires the dose to be very carefully adjusted. There may be increased Doppler resistance, as for acute tubular necrosis, but normally indices remain unaffected. Histology is required to confirm the diagnosis, or a clinical improvement following reduction or withdrawal of the immunosuppressive agent.

Renal transplant dysfunction and Doppler correlation

Doppler correlation with the different types of renal graft dysfunction is not possible. However, by taking the *clinical picture* into account it *is* possible to differentiate these situations. (Table 7.4).

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Chapter 8

The retroperitoneum and gastrointestinal tract

CHAPTER CONTENTS

NORMAL ANATOMY

The peritoneum is the large sheet of serous membrane which lines the abdominal cavity and surrounds the organs. The peritoneum has several 'extensions' which bind the organs together: the mesentery, which loosely anchors the small bowel ensuring it does not twist, the transverse mesocolon, which attaches the transverse colon to the posterior abdominal wall, and the greater and lesser omentum. These projections coat the viscera and form pouches, or sacs, within the peritoneal cavity in which dependent fluid can collect.

The retroperitoneal space contains the kidneys and ureters, adrenal glands, pancreas and duodenal loop, great vessels and the ascending and descending portions of the large bowel, including the caecum (Fig. 8.1).

THE ABDOMINAL AORTA

The abdominal aorta can be visualized proximally in the midline, posterior to the left lobe of the liver. The coeliac axis and superior mesenteric artery (SMA) are easily demonstrated in longitudinal section (LS), arising from its anterior aspect (Fig. 8.2).

In transverse section (TS) the coeliac axis branches, the main hepatic and splenic arteries, may be better appreciated. Just below this level, the origin of the renal arteries is seen.

The distal abdominal aorta, which runs more anteriorly, and bifurcation are frequently obscured by bowel gas in sagittal section. A coronal

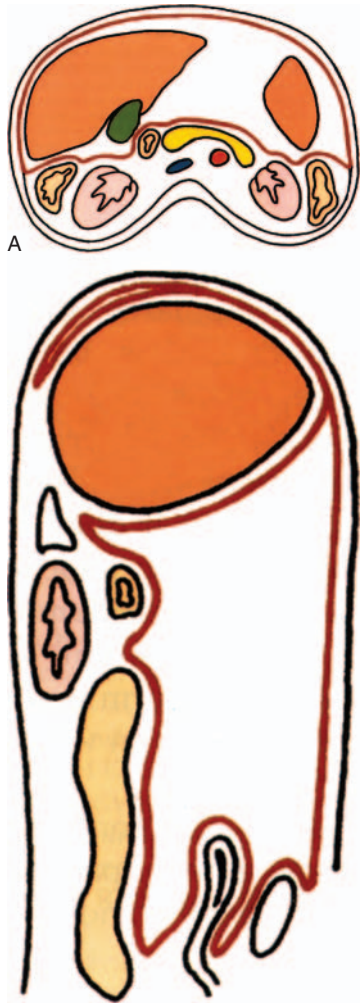

Figure 8.1 (A) Axial and (B) sagittal sections through the abdomen, showing the relationship of the abdominal viscera to the peritoneum (red).

approach from the patient's left side often overcomes this problem (Fig. 8.2D) and is also useful in displaying the origin of the renal arteries.

The aorta often becomes ectatic and tortuous with age, and it is not unusual to detect considerable calcification of the walls (Fig. 8.2G).

Aortic aneurysm

The most frequent referral for aortic scanning is to establish or monitor the presence of an aneurysm. The incidence of aortic aneurysm increases with age and patients may present with a pulsatile, mid abdominal mass. Patients most at risk are men aged 60 or over, with an incidence of up to 9% after age 75. The risk of aneurysm rupture increases with diameter, increasing dramatically when it reaches 6 cm, with a 1-year mortality of 50%.[1]

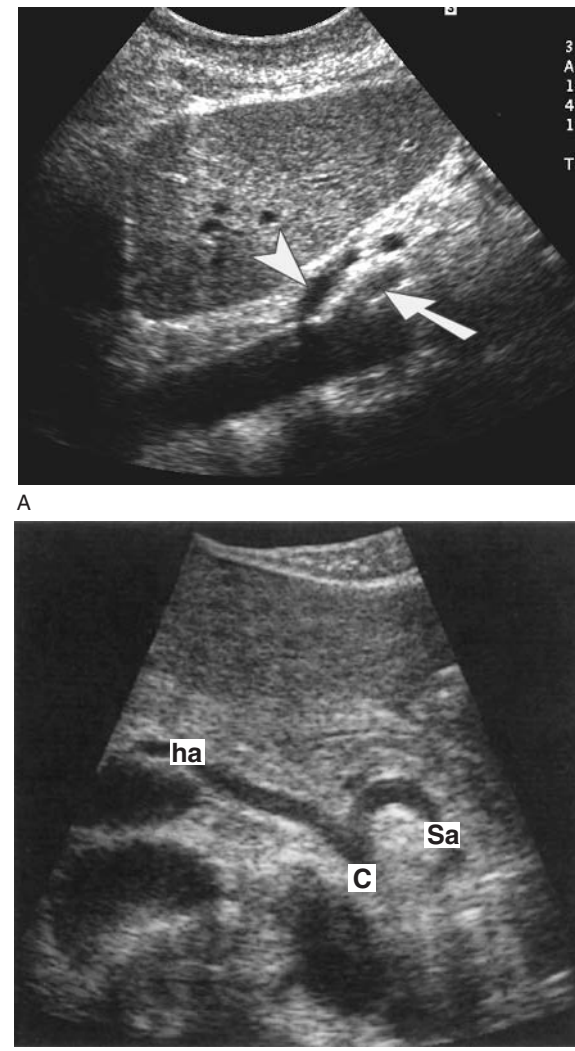

Figure 8.2 (A) Longitudinal section (LS) through the abdominal aorta, demonstrating the coeliac axis (arrowhead) and the superior mesenteric artery (SMA) (arrow). The splenic vein (SV) and body of pancreas are seen anterior to the SMA. (B) Transverse section (TS) through the proximal abdominal aorta. The coeliac axis, (C), divides into the hepatic and splenic arteries.

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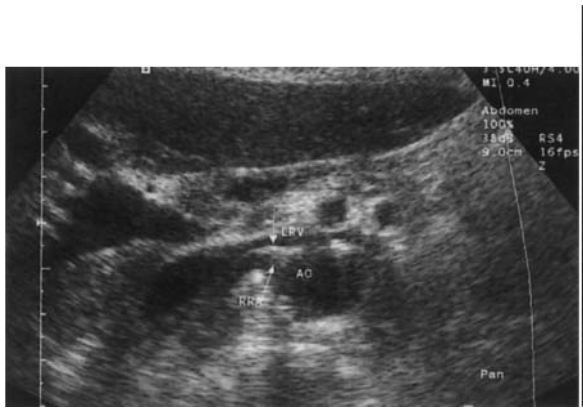

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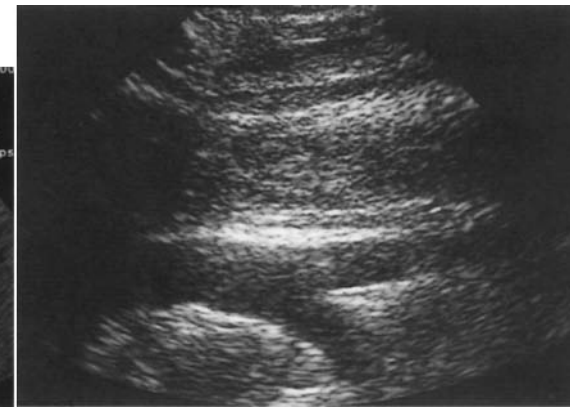

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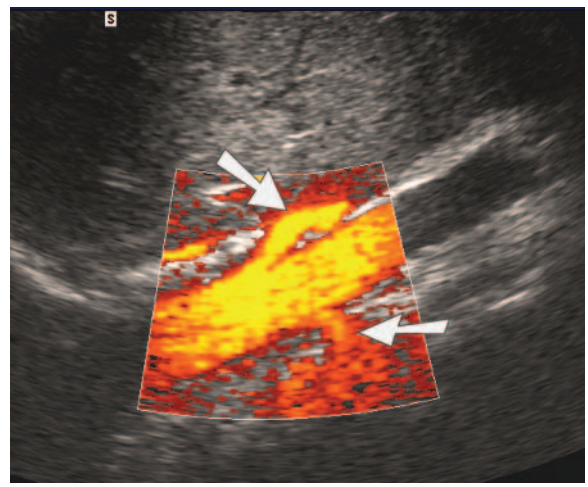

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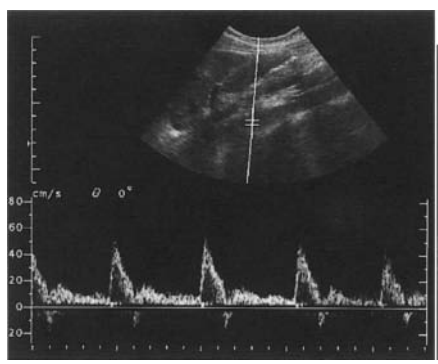

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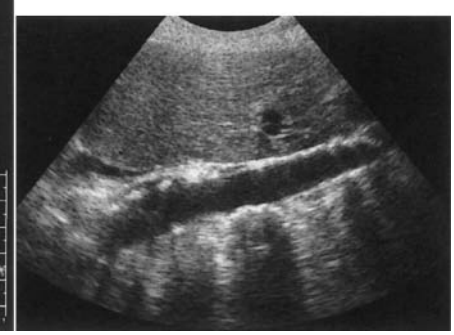


G

Figure 8.2 cont'd (C) TS slightly distal to (B). The right renal artery (RRA) is seen arising from the lateral aspect of the aorta; the left renal vein (LRV) passes anteriorly to drain into the inferior vena cava (IVC). (D) A coronal plane, from the patient's left side, demonstrates the aortic bifurcation. (E) Coronal section of the aorta at the level of the renal arteries (arrows). (F) The Doppler spectrum from the aorta demonstrates a highly pulsatile waveform with reversed flow in early diastole. (G) The aorta of an elderly patient contains calcification in the walls, which causes acoustic shadowing.

For this reason, aortic aneurysms are monitored, and a graft placed within the vessel in aneurysms over 5 cm which are increasing in size. Postoperative complications of grafts, such as infection or pseudoaneurysm, are usually monitored with CT or MRI.

Discussions of the benefits of screening programmes for selected populations are ongoing.[1,2] However, there is some evidence that, despite the reduction of mortality due to aneurysm rupture, overall mortality in men over 65 remains unaffected by screening,[3] and it has not been widely adopted into patient management.

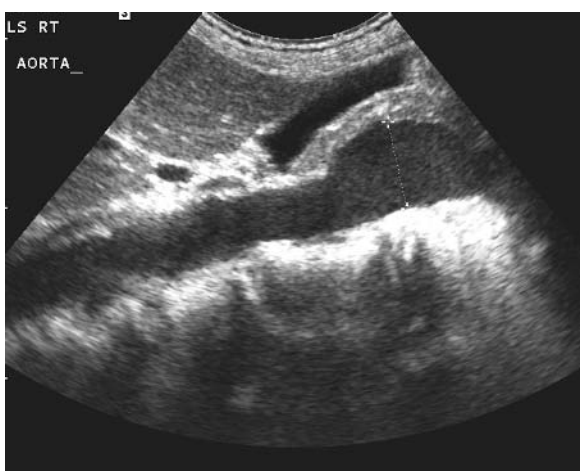
Most aneurysms are associated with atherosclerosis, which weakens the media of the wall, causing the vessel to dilate and eventually rupture.

The aneurysm may be fusiform or saccular (Fig. 8.3). Blood flow within it is turbulent, and the slow-flowing blood at the edges of the vessel tends to thrombose.

Surgery is always complicated by the involvement of the renal arteries. Fortunately, the vast majority of aneurysms are infrarenal, but it can be difficult to determine the relationship of the aneurysm to the renal artery origins on ultrasound, and CT is helpful in such cases. The use of angiography can be beneficial in this respect; however its disadvantage is that, unlike ultrasound, it displays only the lumen of the vessel and can underestimate the pathology present.

Occasionally the aneurysm affects the bifurcation and common iliac arteries, which should be examined during the scan as far as possible.

The true maximum diameter of the aneurysm should be ascertained in TS and LS. A true anteroposterior diameter is most accurately measured in LS, by ensuring the calipers lie in a plane perpendicular to the vessel axis at its widest part. To measure the lateral diameter in TS, care must be taken to keep the angle of the transducer perpendicular to the vessel axis to ensure an accurate and reproducible measurement. The ability of ultrasound to locate the



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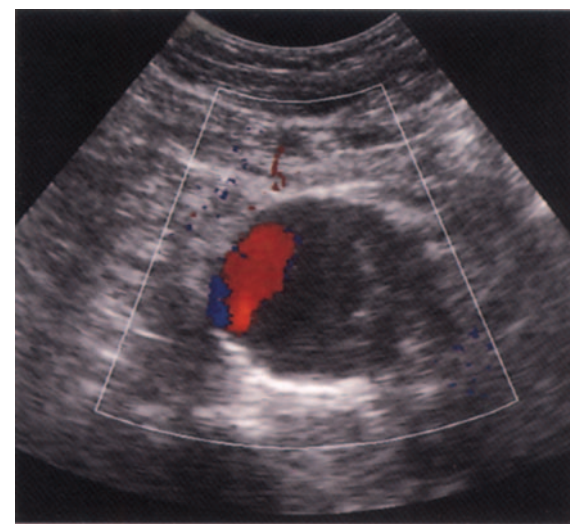
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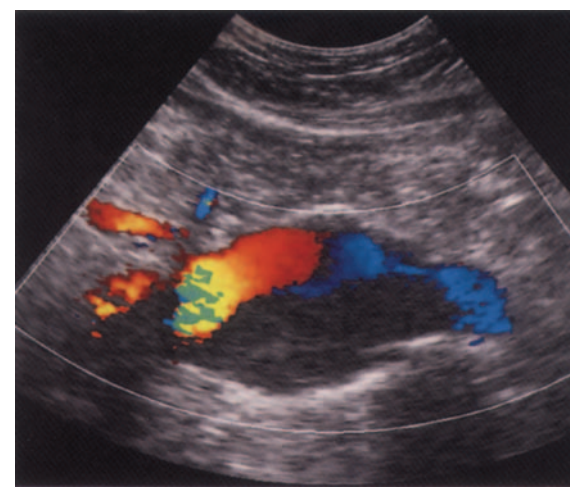
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Figure 8.3 (A) LS demonstrating an aneurysm of the lower abdominal aorta. (B) TS through the aneurysm containing thrombus with an eccentric lumen demonstrating turbulent flow. (C) LS demonstrating flow around the thrombus in the aneurysm.

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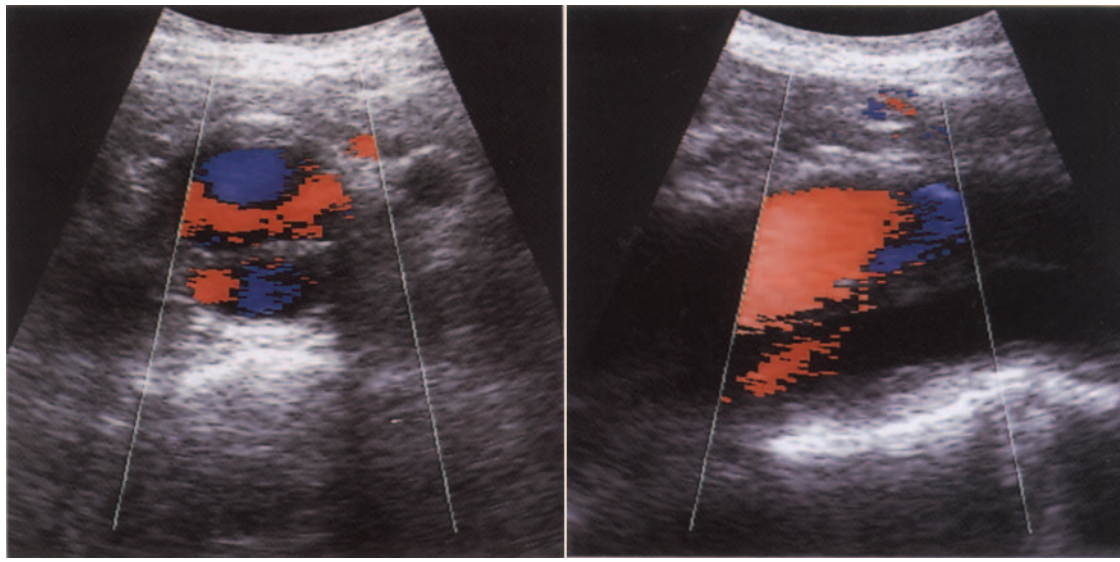

D E

Figure 8.3 cont'd (D) TS and (E) LS of a dissecting aortic aneurysm. The detached intimal flap is clearly seen within the aortic lumen.

correct plane, regardless of vessel tortuosity, is a distinct advantage over CT, which may over- or underestimate the size of the aneurysm in an axial plane.

Complications of aortic aneurysm

Dissection of the aneurysm, in which the intima becomes detached, is uncommon in the abdomen. Ultrasound may visualize the intimal flap and the false lumen created between the media and intima often contains slower, more turbulent or even reversed flow. Layers of thrombus may mimic a dissection, and colour flow Doppler is particularly useful in such cases.

Leakage of an aneurysm may cause retroperitoneal haematoma, but CT is usually more reliable in detecting leaks than ultrasound.

Rupture of an aortic aneurysm is not unknown in the ultrasound department or emergency department, and is accompanied by abdominal pain and severe hypotension. It is associated with a high mortality rate and is a surgical emergency.

Involvement of the renal arteries may cause renal artery thrombosis and subsequently small kidney(s). Always check the kidneys at the time of scanning to ensure they are of normal size and appearance.

THE INFERIOR VENA CAVA (IVC)

Ultrasound is highly successful in demonstrating the proximal IVC, by using the liver as an acoustic window, especially if the patient is turned right anterior oblique. The distal IVC may be obscured by overlying bowel gas and, unlike the aorta, is also susceptible to compression, making visualization difficult in some cases.

The normal IVC has thinner walls and a more flattened profile than the aorta, and its lumen alters with changing abdominal pressure; for example, during respiration the lumen decreases on inspiration, or with the Valsalva manoeuvre (Fig. 8.4). Its course becomes slightly anterior as it passes through the diaphragm, unlike the aorta which travels posteriorly at this point.

The main renal veins may be seen in TS, entering the IVC just below the level of the pancreas (Fig. 8.2).

Haemodynamically, the blood flow spectrum from the IVC alters according to the distance of the sample volume from the right atrium (Fig. 8.4 F, G). The blood flow through the IVC and proximal hepatic veins is pulsatile, with reverse flow during right atrial systole. Pulsatility reduces in the distal IVC.

The most common anomaly of the IVC is that of duplication. However this is infrequently picked up on ultrasound and is best demonstrated with CT or MRI. Transposition of the IVC may be seen in situs inversus.

Pathology of the IVC

Thrombus in the IVC may be due to benign causes, or the result of tumour. It is not usually possible to tell the difference on grey-scale appearances alone, but vascularity may be demonstrated on power or colour Doppler within tumour thrombus, and the clinical history is helpful. Tumour thrombus invades the renal vein and

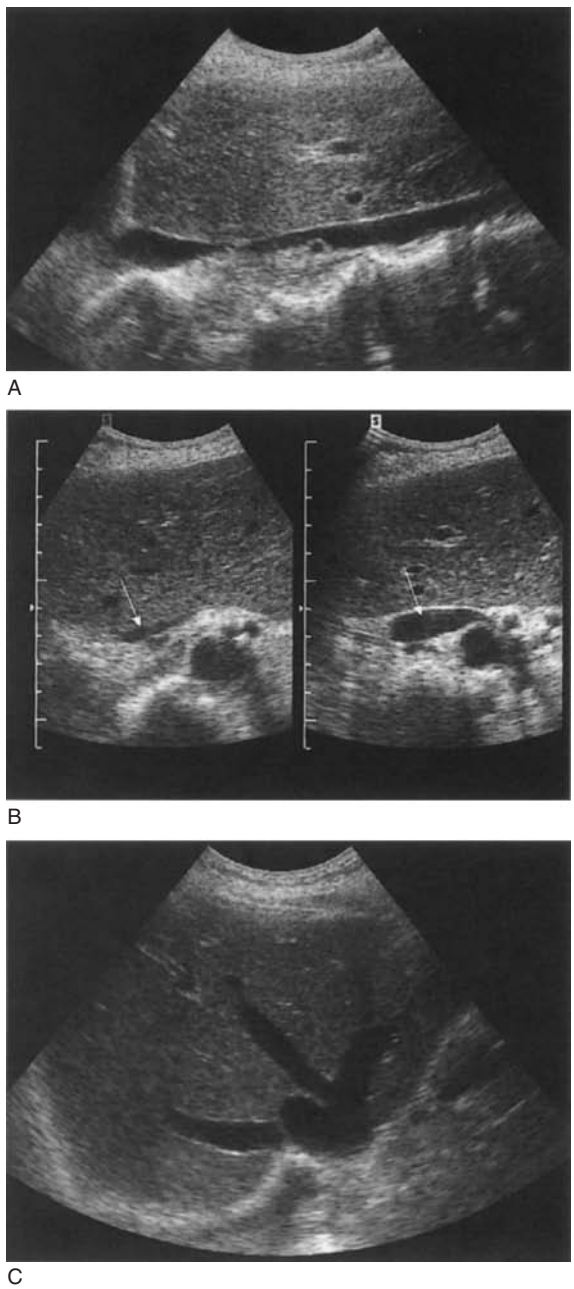

Figure 8.4 (A) LS through the IVC. The RRA is seen passing underneath the IVC. (B) TS through the IVC, demonstrating the difference in profile during the Valsalva manoeuvre (left) compared with normal expiration (right). (C) IVC at the level of the confluence of the hepatic veins, just beneath the diaphragm. (D) Power Doppler of the IVC overcomes problems associated with its perpendicular angle to the transducer. Portal vein (PV) anterior to IVC. (E) The right renal vein (RRV) (in red) is seen draining into the IVC on colour Doppler.

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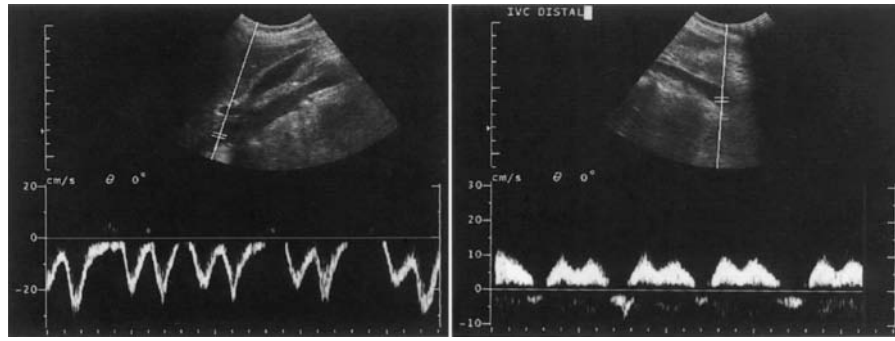


F G

Figure 8.4 cont'd (F) Normal, pulsatile spectrum from the proximal IVC is influenced by the proximity of the right atrium. (G) The waveform from the distal IVC is lower in velocity, less pulsatile and displays more variance.

enters the IVC in around 10% of renal carcinoma cases. Tumour thrombus from hepatic or adrenal masses can also invade the IVC.

Coagulation disorders, which cause Budd–Chiari syndrome (see Chapter 4) predominantly affect the hepatic veins, but may also involve the IVC (Fig. 8.5).

Patients may require the insertion of a caval filter, which is performed under X-ray guidance, but may be monitored for patency using ultrasound with Doppler.

Dilatation of the IVC is a finding commonly associated with congestive heart failure, and is frequently accompanied by hepatic vein dilatation.

Compression of the IVC by large masses is not uncommon. This may be due to retroperitoneal masses, such as a large lymph node, or liver masses such as tumour or caudate lobe hypertrophy. Colour or power Doppler is particularly useful in confirming patency of the vessel and differentiating extrinsic compression from invasion. Insertion of metallic stents may be performed under angiographic control to maintain the vessel patency, particularly if the compression is due to inoperable hepatic metastasis (Fig. 8.6).

Tumours of the IVC are rare. Leiomyosarcoma is a primary IVC tumour, appearing as a hyperechoic mass in the lumen of the vein.[4,5] This causes partial or complete obstruction of the IVC, resulting in Budd–Chiari syndrome. In partial occlusion, the hepatic veins and proximal IVC may be considerably dilated. Resection of the tumour, with repair of the IVC, is possible provided the adjacent liver is not invaded.[4]

THE ADRENAL GLANDS

Normal appearances

The normal adrenal glands can be seen on ultrasound in the vast majority of patients,[6,7] if you know where and how to look. Each adrenal gland is constructed with a central fold or ridge, which points anteromedially, from which extend two thin 'wings' of tissue—a medial and a lateral wing (Fig. 8.7).

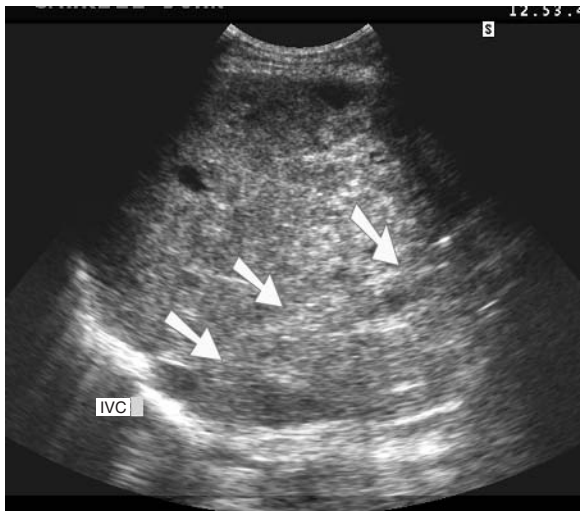
The ultrasound appearances are therefore of a < shape in LS, or a thin, linear structure as the transducer is moved medially towards the central ridge.

The wings of the gland appear hypoechoic and are no more than 2 mm in thickness.

Ultrasound technique

For the right adrenal, use the liver as an acoustic window. Scan the upper pole of the kidney intercostally through the liver, and angle slightly medially to the kidney, where the gland can be located between the liver and the diaphragmatic crus (Fig. 8.7A). Continue angling slightly medially towards the IVC and the central ridge of the gland is seen behind the IVC (Fig. 8.7B).

For the left gland the spleen must be used as a window. To avoid overlying bowel this is best achieved with the patient supine, using a coronal section. When the upper pole of the left kidney is located through the spleen, the left adrenal can be seen in the small triangular area between the spleen, kidney and diaphragmatic crus (Fig. 8.7D).[6,7]



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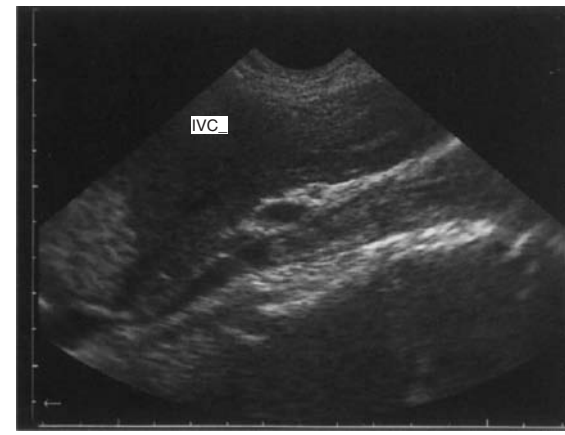

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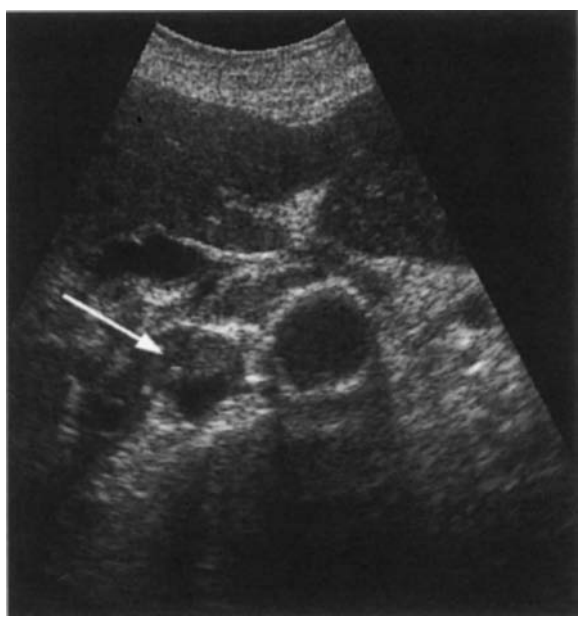

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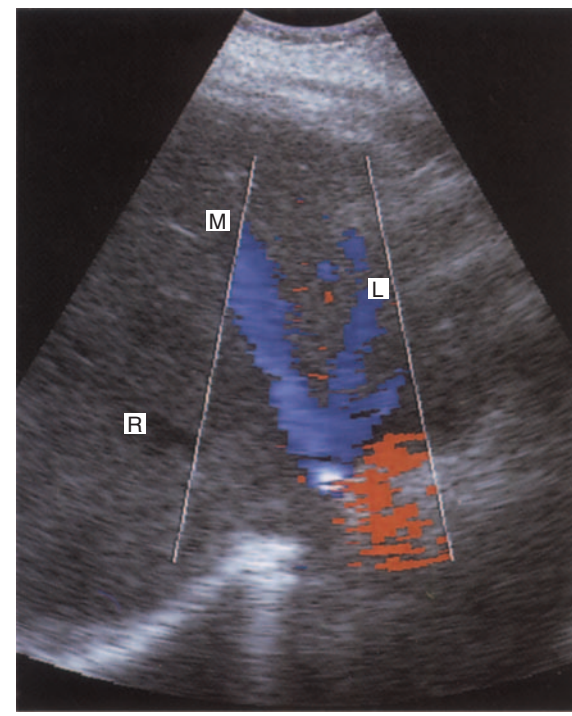

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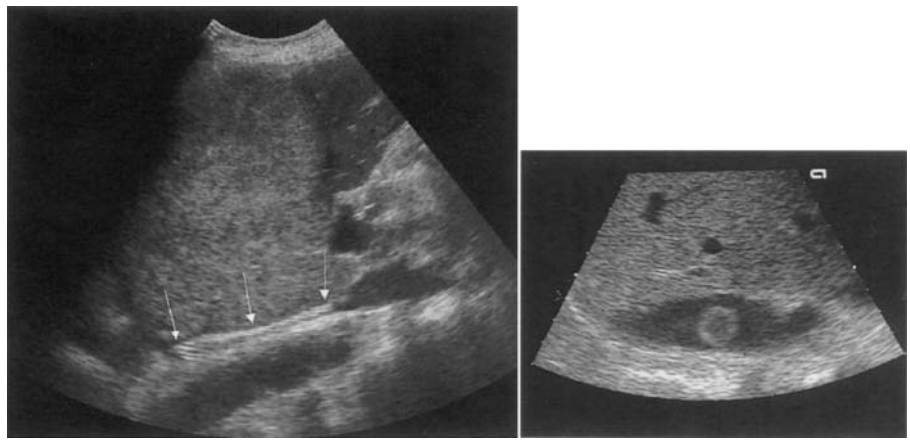
Figure 8.5 (A) Tumour thrombus from a left renal carcinoma completely occludes the IVC (arrows). Liver metastases are also present. (B) Advanced renal carcinoma. The IVC is full of tumour. (A hyperechoic liver metastasis is also seen on the left.) (C) TS through the IVC containing thrombus. This is the result of proximal compression of the IVC by a large liver abscess. (D) Tumour thrombus from a renal carcinoma has spread up the IVC and invaded the right hepatic vein (RHV), causing a partial Budd–Chiari effect.



A B

Figure 8.6 (A) Compression of the IVC by a large liver metastasis caused Budd–Chiari syndrome. This has been relieved by the insertion of a metal stent into the IVC (arrows) under angiographic control. (B) Unusual case of a small leiomyosarcoma in the IVC found incidentally. Normally they are larger, presenting with symptoms of IVC obstruction.

Pathology of the adrenal glands

Adenoma

Small (less than 3 cm) solid adrenal nodules are a common, incidental finding in non-symptomatic patients (Fig. 8.8A).

Benign, non-hyperfunctioning adenomas account for the majority of adrenal nodules, and are of no clinical significance. Their incidence increases with age and they are present in around 2% of adult autopsies.

Small nodules in asymptomatic patients generally require no further action, but endocrine function may be evaluated to rule out a functioning mass.

A hyperfunctioning adenoma (a determination made by evaluation of the endocrine function), although an essentially benign mass, usually requires surgical resection.

As a solitary abdominal finding in a patient with no relevant clinical history, it is generally safe to assume a small adrenal nodule requires no further action. However, because it is not possible to distinguish benign, incidental nodules from other forms of more serious pathology, incidental nodules of greater than 4 cm should be investigated further to confirm their benign nature.[8] Non-functioning adenomas will remain stable in size on ultrasound follow-up.

Metastasis

The adrenal glands are a common site for metastases, particularly from lung, breast and bowel cancer. Although frequently accompanied by liver metastases, they may be present in the absence of any other obvious abdominal deposits, and therefore the adrenal glands should routinely be examined when staging malignant disease.

The adrenal glands are also commonly involved in non-Hodgkin's lymphoma.

Like adenomas, they are often small, well-defined and hypoechoic on ultrasound (Fig. 8.8B). It is not possible to differentiate between benign adenoma and metastasis on the ultrasound appearances alone, but a small adrenal mass in the absence of a known primary carcinoma is likely to be benign, and will remain stable on follow-up. A solitary adrenal mass in the presence of known carcinoma requires biopsy for diagnosis.

Adrenal cysts

Simple cysts are uncommon in the adrenal gland, but are easily differentiated from solid lesions with ultrasound. Some cysts may be the sequelae of previous haemorrhage, but most are simple, epithelial cysts.

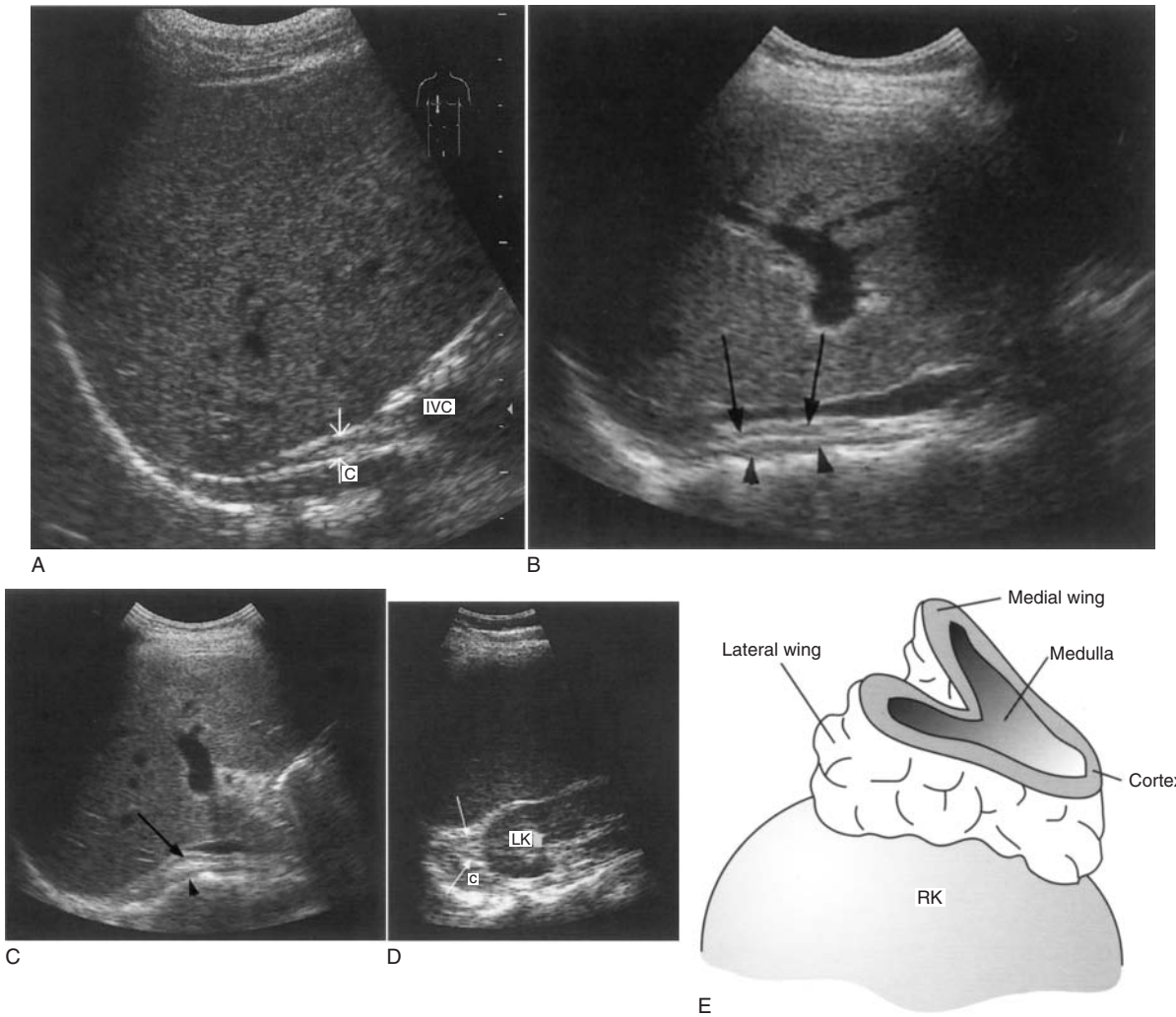


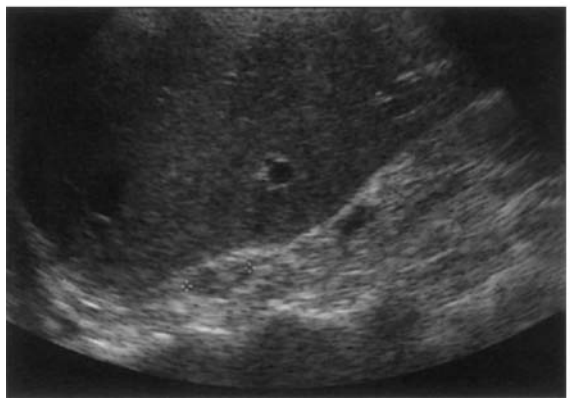
Figure 8.7 (A) Right adrenal. The medial (anterior arrow) and lateral (posterior arrow) wings of the gland lie just anterior to the crus, C. (B) The medial ridge of the right adrenal (arrows) is seen anterior to the diaphragmatic crus (arrowheads) and posterior to the IVC. (C) Transverse right adrenal (arrow) between the IVC and crus (arrowheads). (D) The two hypoechoic wings of the left adrenal (arrows) can be seen in the space between the spleen, left kidney (LK) and the crus, c. (The dynamic range has been reduced to appreciate the gland better.) (E) Section through the right adrenal gland.

Myelolipoma

The adrenal myelolipoma is found, uncommonly, as an incidental mass. It is highly echogenic and well-defined, due to its fatty content (Fig. 8.8C). These are relatively rare, require no further management, and are endocrinologically non-functioning.

Phaeochromocytoma

The phaeochromocytoma is uncommon, but may be found in up to 1% of patients with hypertension. It is a tumour arising in the chromaffin cells of the adrenal medulla (most commonly) or in autonomic nervous tissue. It may be bilateral and appears solid on ultrasound, although larger masses may have



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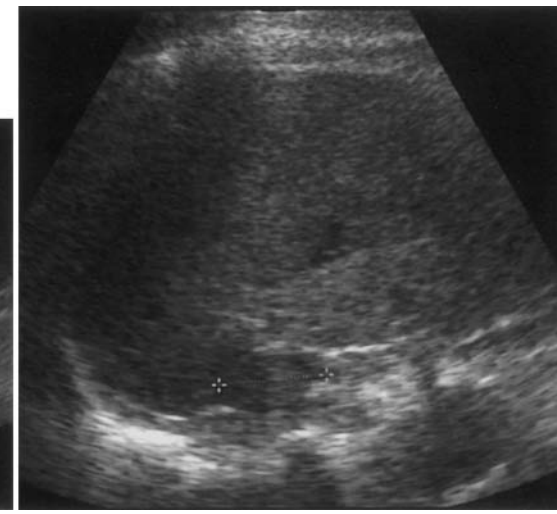

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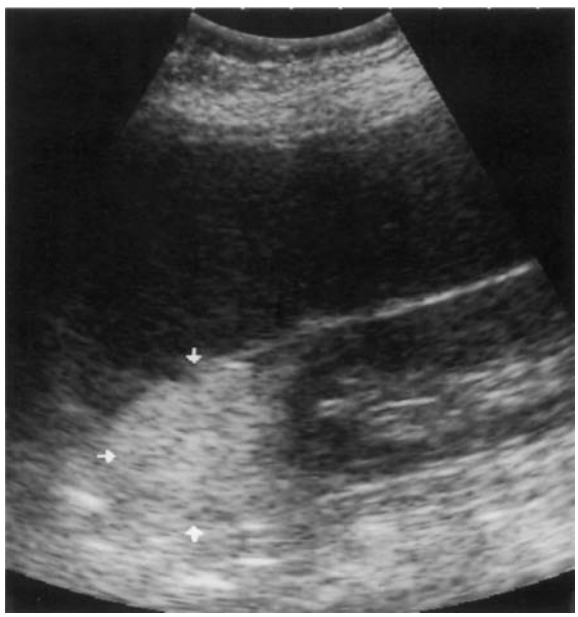

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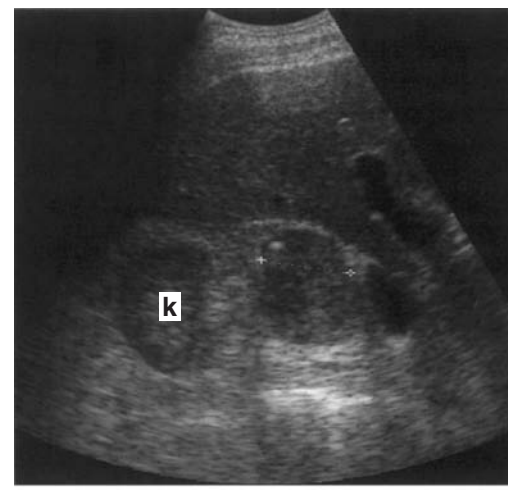

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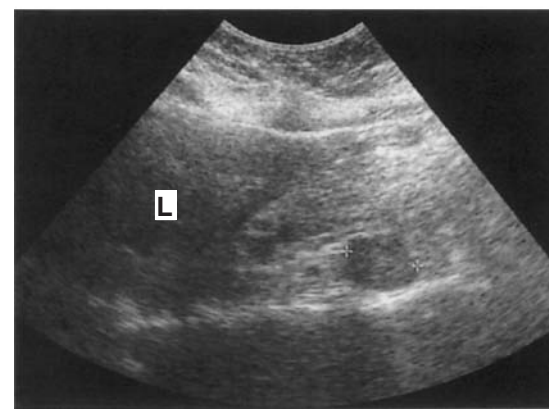


E

Figure 8.8 (A) Typical, incidental, non-functioning adrenal adenoma <2 cm in size. (B) TS through the right adrenal showing a small, 2.4 cm metastasis from a primary lung carcinoma. No liver metastases were present at the time. The lesion showed a reduction in size following chemotherapy. (C) Left adrenal myelolipoma—an incidental finding which was confirmed on CT and remained stable over a period of 3 years. Its high fatty content makes it hyperechoic. (D) Adrenal phaeochromocytoma between the upper pole of the RK and the IVC. (E) LS through the midline, between the IVC and aorta, demonstrating an extra-adrenal phaeochromocytoma. (The differential diagnosis was of lymphadenopathy.)

areas of necrosis within them. Most are benign, but 5–10% are malignant. It presents on a background of episodic, severe hypertension and the urine contains catecholamines. (Although this is also a feature of adrenal neuroblastoma, the latter is predominantly a childhood tumour.) These lesions should be treated with great care—vigorous palpation may precipitate a severe hypertensive episode and biopsy should therefore be avoided.

Although most phaeochromocytomas arise in the adrenal glands, and are therefore demonstrable on ultrasound, those arising in the sympathetic chain may be obscured by bowel gas and are not possible to exclude on ultrasound (Fig. 8.8D, E). If there remains biochemical evidence of phaeochromocytoma in the presence of normal adrenal glands, a Meta-Iodobenzylguanidine isotope scan will demonstrate increased activity in a phaeochromocytoma and CT scan can then be targeted to the appropriate area.

Phaeochromocytomas are also associated with von Hippel–Lindau syndrome.

Adrenal carcinoma

Primary adrenal carcinomas are rare in the adult. They are commonly endocrinologically inactive in adults, and therefore tend to present late when they are quite large. They may invade the IVC and metastasize to the liver. Surgical removal of tumours in the absence of liver metastases has a good prognosis[9] and, in patients with metastases, radiofrequency ablation of the adrenal mass may have some benefit in prolonging survival.[10]

GASTROINTESTINAL (GI) TRACT

Contrast radiographic investigations, including CT, are generally accepted as the methods of choice for investigating diseases of the GI tract. Although ultrasound is not considered a primary tool in the investigation of bowel lesions, as the gas-filled lumen makes visualization difficult in many cases, ultrasound is remarkably successful in diagnosing GI tract pathology in the hands of an experienced operator. GI tract ultrasound can be time-consuming, but a wealth of information can be obtained with a high-frequency linear probe in a symptomatic patient. Considerable diagnostic benefit has been shown for careful, targeted, percutaneous ultrasound of the large and small GI tract using high-frequency transducers.[11]

It is important to be aware of the variable ultrasound appearances of normal bowel, as it may be responsible for mimicking other pathology. Normal bowel is frequently difficult to examine on ultrasound as the gas-filled lumen reflects the sound, requiring careful compression techniques. Abnormal bowel is particularly accessible to ultrasound, however. A fluid-filled lumen also make easy the demonstration of valvulae conniventes of the small bowel and haustra of the large colon.

Oesophagus and stomach

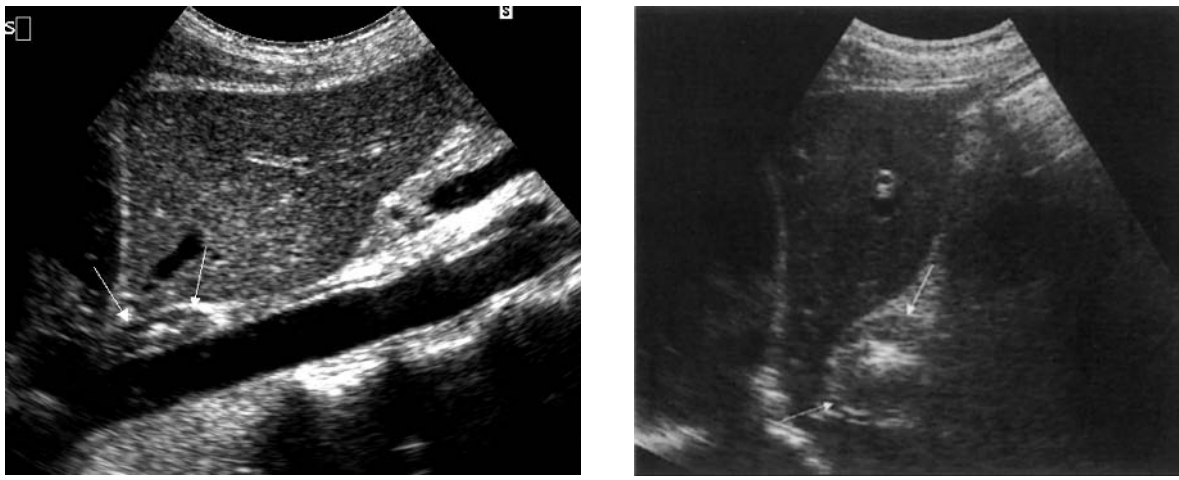
The oesophagus is not usually accessible to percutaneous ultrasound; however, the lower end can be demonstrated as it passes through the diaphragm in the midline, just anterior to the aorta (Fig. 8.9A). Its normal appearances should not be confused with a mass. Occasionally, ultrasound demonstrates the thickened wall associated with an oesophageal carcinoma involving the lower oesophagus (Fig. 8.9B).

Endoscopic ultrasound (EUS), with its high frequency and proximity to the relevant structures, is able to demonstrate the layers of the gut wall, and to demonstrate pathology and accurately stage malignant disease in both the oesophagus and stomach, and also to guide invasive procedures.[12,13]

Barium X-ray studies are still the first-line investigation of choice for many potential GI tract conditions; however, endoscopy is regarded as the gold standard for investigating the lining of the stomach and duodenum and can be combined with biopsy when necessary. Although percutaneous ultrasound has had modest success in revealing stomach masses if the stomach is filled with water,[14] it can never replace endoscopy. However, if such lesions are discovered, this helps to direct subsequent radiological management (Fig. 8.10).

Appendix

Acute appendicitis is a common diagnosis on admission to the casualty department with right lower abdominal pain. However around 15–25% of



A B

Figure 8.9 (A) Normal oesophagus between the aorta and the left lobe of the liver—thin, hypoechoic walls with a hyperechoic lumen due to the presence of air. (B) Thickened walls of the oesophagus in a carcinoma involving the lower oesophagus.

patients who undergo laparotomy turn out to have normal appendices.

The use of ultrasound in the investigation of acute abdominal pain is well established and can increase the reliability of the diagnosis of acute appendicitis when performed by an experienced operator.[15]

The normal appendix is difficult to locate. A high-frequency (7 MHz or more) linear or curved array probe is useful. Gentle, graduated compression may move overlying bowel. Raising the patient's left side may encourage bowel gas to move away from the area of interest. The normal appendix is compressible by gentle transducer pressure, which is usually well tolerated by the patient.

The ultrasound features of acute appendicitis include an enlarged, usually hypoechoic appendix greater than 6 mm in diameter. The inflamed appendix is non-compressible. Attempted compression of the acutely inflamed appendix obviously requires great care from the operator. Compression must be very slow and the release of compression must be equally as gentle. These features have a high sensitivity and specificity for acute appendicitis (74% and 94% respectively).[15] Acute appendicitis often demonstrates hypervascularity on power Doppler.

Other causes for right iliac fassa masses in patients presenting with pain include inflamed diverticula in patients with diverticulitis.[16]

Perforation of the appendix may result in a demonstrable periappendiceal fluid collection, or free fluid plus or minus dilated loops of non-peristaltic small bowel. The presence of an ill-defined fluid mass in the right iliac fossa of a symptomatic patient is highly suggestive of acute appendicitis with perforation (Fig. 8.11). This may become infected, leading to peritonitis.

Occasionally, a hyper-reflective appendicolith may be seen in the blind end of the inflamed appendix, casting an acoustic shadow.

Mesenteric ischaemia

Mesenteric ischaemia is a potentially lethal condition, associated with atherosclerosis of the mesenteric vessels, which can cause bowel necrosis and death if left untreated. It is a difficult diagnosis to make on clinical grounds because the symptoms are varied and non-specific, including acute abdominal pain following meals, diarrhoea and

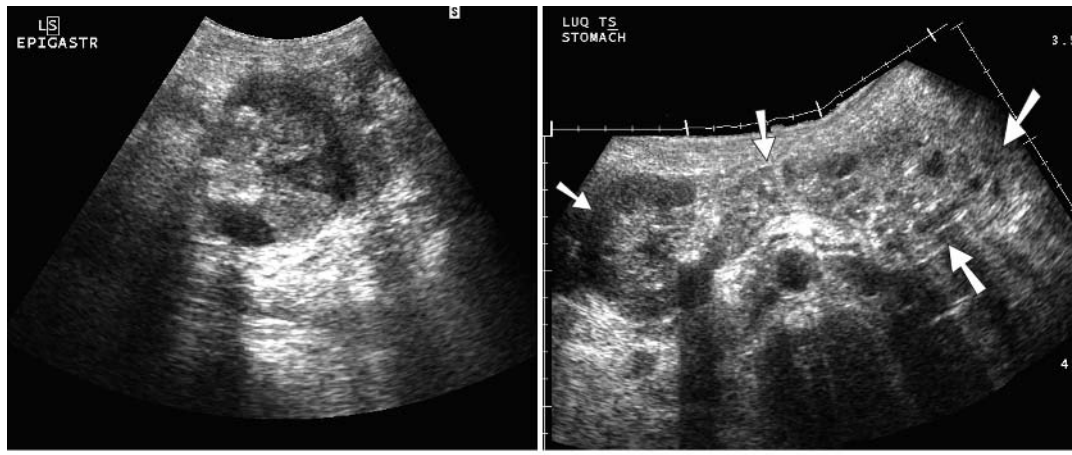


Figure 8.10 (A) LS through the epigastrium demonstrating a gastric carcinoma (arrows). (B) TS through the epigastrium demonstrating a gastric carcinoma (arrows). (C) Gastric carcinoma, presenting with altered liver function tests (LFTs). The patient had multiple liver metastases.

subsequent weight loss. Patients frequently undergo a number of comparatively invasive investigations before a diagnosis is reached, and this delay increases the mortality and morbidity of the condition.[17,18]

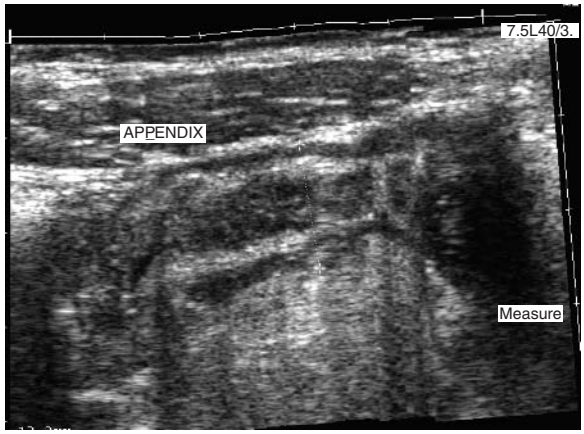
Treatment involves restoring the blood flow via angioplasty or surgery and, if necessary, resecting segments of necrosed bowel.

Atherosclerosis may be demonstrated in the SMA in a number of cases. Signs of occlusion or stenosis of the SMA may be identified with colour or power Doppler[19] as a filling defect within the lumen of the vessel. However, significant stenoses have been diagnosed with Doppler in a relatively high percentage (18%) of the asymptomatic, elderly population. The finding of a stenosis on ultrasound, therefore, is an indication for further imaging in symptomatic patients, rather than an absolute indicator of mesenteric ischaemia.[20]

In a normal patient, the response of the SMA to food can be demonstrated as an increase in end diastolic flow velocities (Fig. 8.12).

Mesenteric compromise has also been associated with an abnormal postprandial response; Doppler waveforms of the SMA have decreased peak systolic and end diastolic velocities after food.[21]

None of these ultrasound signs and appearances are specific for mesenteric ischaemia; the mesentery is supplied by three arteries which cannot all be evaluated with ultrasound and, in addition, numerous other conditions are associated with altered SMA Doppler resistance, including inflam-



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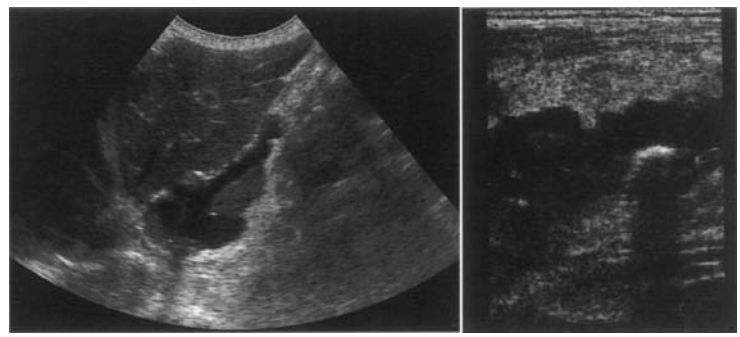


B C

Figure 8.11 (A) Acute appendicitis with a thickened, tender wall and an abscess at the distal end. (B) Perforation of the inflamed appendix has resulted in the presence of intraperitoneal fluid under the liver. (C) Inflamed, fluid-filled appendix containing an appendicolith.

matory bowel conditions, haemorrhage, elevation of venous pressure and cirrhosis.

Inflammatory bowel conditions

Both barium studies and ultrasound have a useful role to play in the management of patients with inflammatory bowel disease. Diagnosis is generally made with conventional barium X-ray studies, while ultrasound may be used to monitor disease and identify extraluminal complications of the disease.[22]

Crohn's disease is a common cause of inflammation affecting the small bowel and particularly the terminal ileum. It usually presents with pain, diarrhoea and weight loss. The terminal ileum/ileocaecal junction is involved in the majority of cases, and thickened, hypoechoic bowel wall can often be demonstrated in this area.[16] Ultrasound may be used to identify complications of Crohn's disease, screen patients at risk, and monitor patients for recurrence of disease following surgery.[23]

Crohn's disease affects the entire thickness of the bowel wall, and one of the common complications is that of intramural abscesses. These can sometimes be seen within the thickened wall as gas-containing, highly echogenic areas. When large, they may perforate, resulting in an ill-defined collection of pus, which may be drained percutaneously (Fig. 8.13).

Fistulae are another complication of Crohn's, and are easier to demonstrate with contrast radiography.

Ulcerative colitis affects the mucosa, rather than the whole wall. On ultrasound it produces a thickened, stratified hypoechoic wall, unlike Crohn's, in which the entire thickness of the wall is affected.

A wall thickness greater than 3 mm is considered abnormal. Like Crohn's, small ulcer craters within the wall of the colon in ulcerative colitis may appear as hyperechoic gas-filled foci.

Inflammatory bowel diseases increase the perfusion of the intestine, decreasing vascular resistance. Hypervascularized bowel wall has been identified

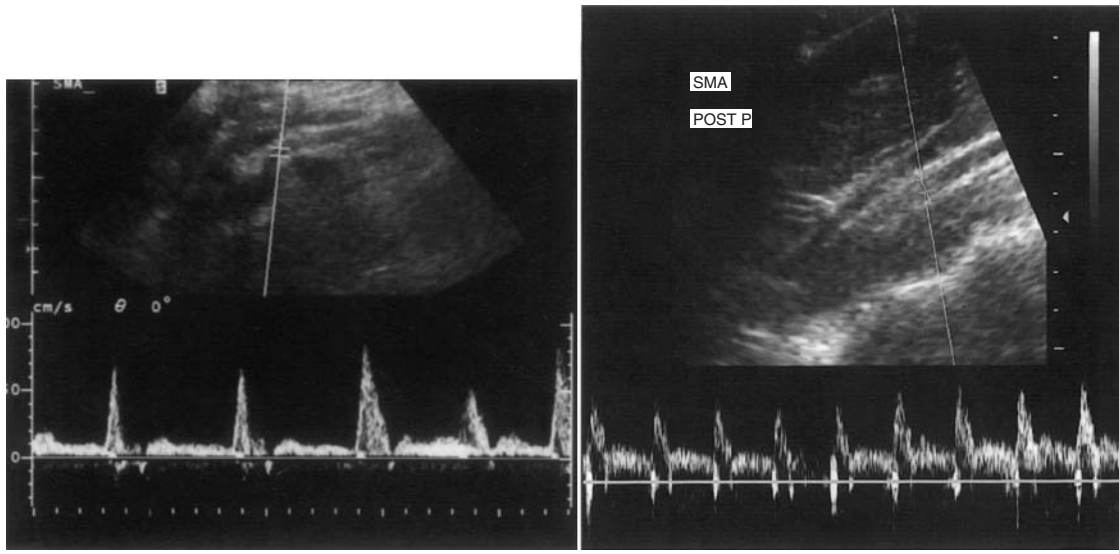


Figure 8.12 (A) Normal spectral waveform from the SMA in a fasting patient is highly pulsatile with little or no end-diastolic flow (EDF) and reverse flow in early diastole. (B) Postprandially, the waveform becomes much less pulsatile, with low resistance and good EDF.

in both Crohn's and ulcerative colitis[24] compared with normal subjects. Doppler of the SMA has revealed an increase in flow velocities (both peak systolic and end diastolic) and a decrease in resistance index in numerous types of pathological bowel, including Crohn's.[25] However the lack of specificity limits its use in clinical work.

Changes in resistance index have been found to be related to the activity of Crohn's disease,[26] which could prove valuable in monitoring patients with known disease.

Diverticulitis may also be recognized on ultrasound as outpouchings from the bowel wall, most commonly affecting the sigmoid colon[11] (Fig. 8.13C). Perforation of a diverticulum may give rise to a diverticular abscess, although the presence of air makes ultrasound limited in its evaluation of this condition.

Malignant tumours

The most common site for a bowel tumour in the adult is around the caecum. It is useful to target this area in patients with altered bowel habit in whom bowel carcinoma is suspected, although detection with ultrasound is usually incidental.

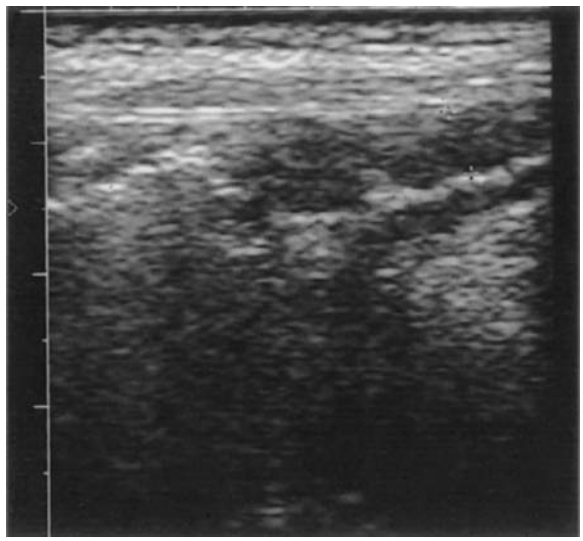
The mass tends to be hypoechoic, or of mixed echogenicity, with a small, eccentric, gas-filled lumen. This cannot be differentiated, however, from an inflammatory mass on ultrasound. Vigorous Doppler flow can usually be visualized in both inflammatory and malignant masses (Fig. 8.14).

The finding of a colonic mass would normally prompt a barium enema, to delineate the nature, extent and position of the mass, with subsequent staging by CT if malignancy is confirmed. The advantage of ultrasound over barium enema is that of displaying the tumour itself, rather than just the narrowed lumen.

The role of ultrasound in patients with known bowel carcinoma is to identify and document the presence of distant metastases, particularly in the liver, as metastases from colorectal carcinoma are particularly amenable to curative resection.

Bowel tumours should be considered in the list of differential diagnoses when the origin of a mass discovered on ultrasound is unclear.

Endosonography may be used to detect and stage rectal cancers, although it is only able to demonstrate perirectal nodes and cannot evaluate distant disease. Endosonography is ideal however,



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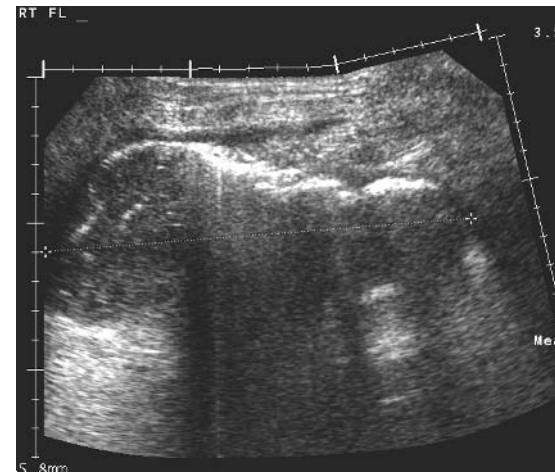

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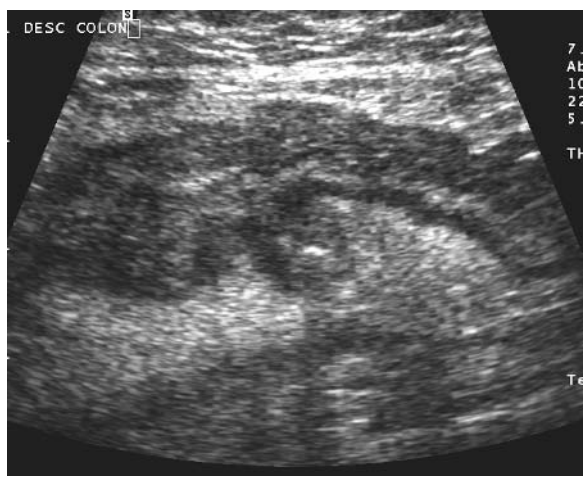

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Figure 8.13 (A) Thickened, hypoechoic bowel wall (5 mm) in Crohn's disease. (B) Large Crohn's abscess containing gas. (C) Diverticulum arising from the ascending colon.

in the follow-up of rectal cancer, and can detect early recurrence of disease.

Obstruction

Ultrasound has been found to be helpful in the investigation of acute obstruction. It can confirm obstruction, by demonstrating dilated, fluid-filled bowel loops with ineffective peristalsis (Fig. 8.15). These fluid-filled loops of bowel are highly amenable to ultrasound scanning, which has the advantage of being able to visualize peristalsis directly, unlike a plain X-ray. It is possible to trace the dilated bowel to the site of obstruction, distal to which are normal loops of collapsed bowel.[27]

The confirmation of obstruction with ultrasound has been proved to be as sensitive as and more specific than plain X-rays and can potentially reduce the need for surgery in such patients, save costs and reduce radiation dose.

However, identifying the actual site and cause of obstruction is time-consuming and frequently unsuccessful. Patients with suspected bowel obstruction, therefore, usually proceed straight to CT.

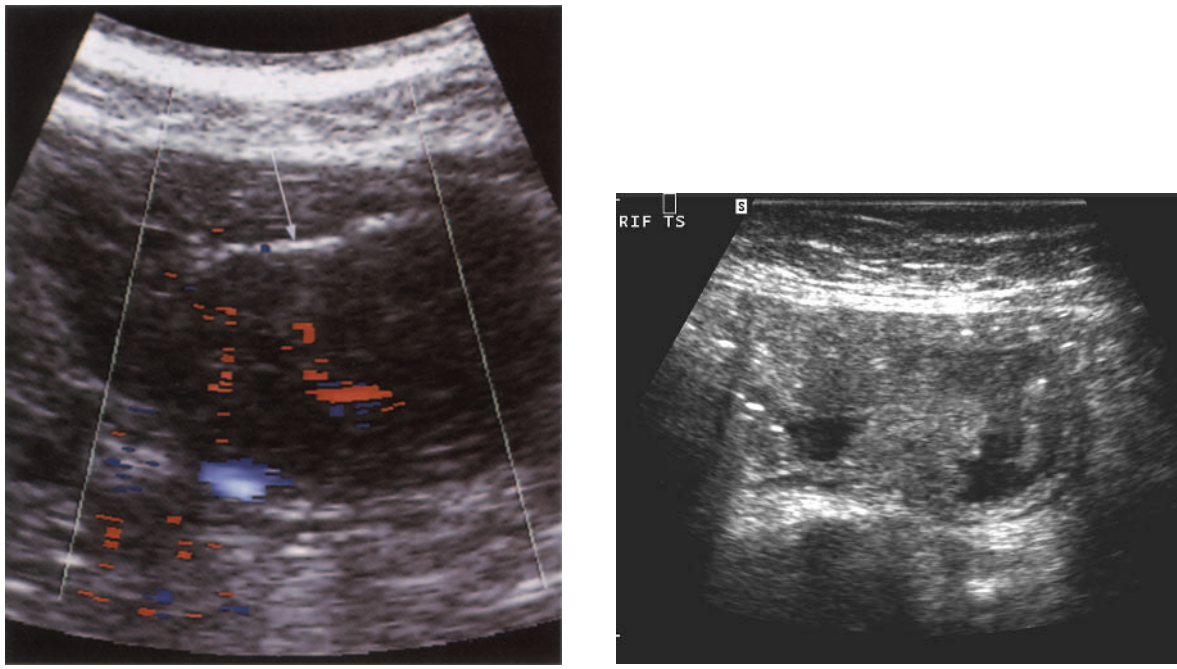

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Figure 8.14 (A) Hypoechoic, caecal carcinoma. The eccentrically thickened bowel wall is demonstrated with a narrow, hyperechoic lumen (arrow). (B) Caecal carcinoma presenting as a mass in the right iliac fossa.

OTHER RETROPERITONEAL ABNORMALITIES

Ultrasound is successful in identifying retroperitoneal masses, but CT and MRI are more effective at establishing the extent and nature of many of these masses, particularly those partly obscured by gas-filled bowel.

The majority of malignant retroperitoneal tumours are renal or adrenal in origin. Other primary tumours, apart from lymphomas, are rare, and include liposarcoma and leiomyosarcoma. These tend to be large when they present, and of variable/complex ultrasound appearance. Encasement of major vessels by tumour is a further characteristic of the retroperitoneal origin of the mass, together with anterior displacement of structures such as the pancreas, kidneys, aorta and IVC.

Ultrasound is also able to identify peritoneal and omental deposits in patients with late-stage carcinoma. These are particularly amenable to diagnosis when surrounded by ascites (Fig. 8.16) and usually arise from gynaecological or urological tumours.

Benign retroperitoneal masses identifiable on ultrasound include haematomas, psoas abscesses, lymphadenopathy (Fig. 8.17) and pancreatic pseudocysts.

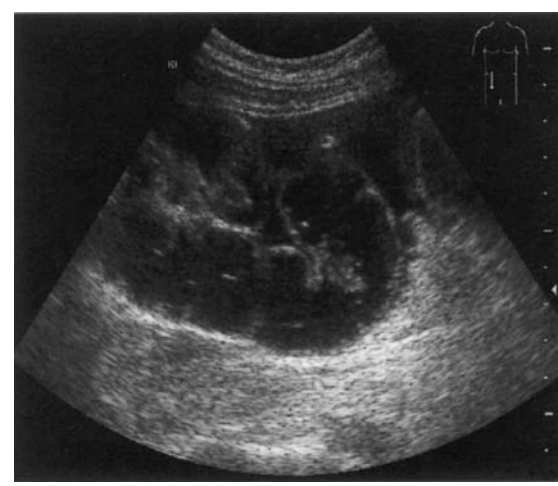
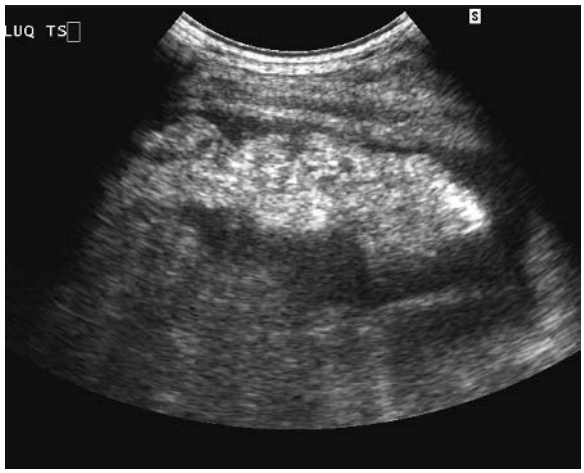


Figure 8.15 Dilated, fluid-filled loops of bowel as a result of an obstructing caecal carcinoma. Ascites is also present.



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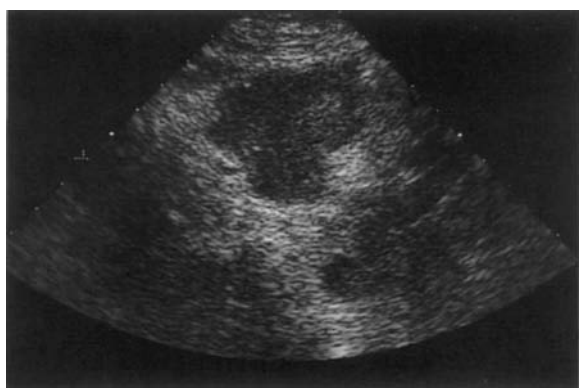

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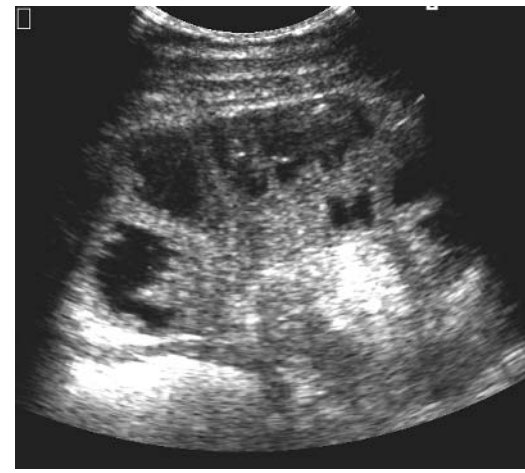
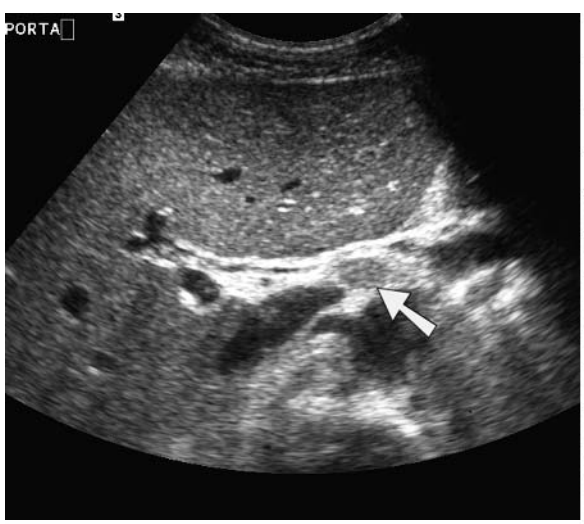


C

Figure 8.16 (A) Late-stage breast carcinoma demonstrates abdominal ascites with a hyperechoic omental cake of metastatic deposit in the left upper quadrant (LUQ). (B) A large, irregular omental deposit from ovarian carcinoma was palpable during the scan. (C) Retroperitoneal metastases from a teratoma.



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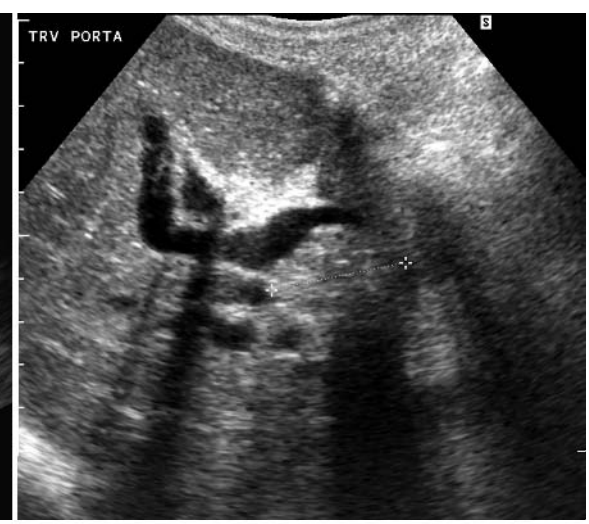

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Figure 8.17 (A) Enlarged lymph node anterior to the aorta (arrow). (B) Lymphadenopathy may be the cause of obstructive jaundice.

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Chapter 9

The paediatric abdomen

CHAPTER CONTENTS

Ultrasound of the paediatric abdomen requires different techniques and skills from those used in the adult. Although there are a few situations in which knowledge of adult pathology can be applied to the paediatric patient, the child cannot simply be considered a mini-version of an adult. The presenting symptoms and pathological processes in the child are generally quite different from those in adults and the operator must be fully aware of the special considerations of the paediatric patient in terms of both diagnosis and technique. This chapter addresses only the most common paediatric situations likely to be encountered in a general department, and further specialist paediatric reading is recommended. (*See the general reading list at the end of this chapter.*)

Techniques

The operator can minimize any distress to the child, and make the examination considerably easier and quicker, in numerous ways.

- The ultrasound environment should be as friendly as possible, with appropriate decorations and toys, and should always be kept warm (nothing is more likely to make your patient uncooperative than cold gel). Minimize the time the child spends in the scanning room by having everything ready first. Small children may benefit from seeing a video of a scan beforehand and being reassured that it will not hurt.
- Most children find it reassuring to be accompanied by their carer during the examination.

- Separate facilities, such as a dedicated children's waiting area, are preferable and more reassuring for the child.
- The equipment should incorporate a range of high-frequency (5–15 MHz) transducers with small as well as conventional footprints. A small curvilinear transducer is useful for most abdominal work and a high-frequency linear probe is essential for bowel sonography and assessment of the acute abdomen. Transducers with a dynamic frequency facility are an excellent choice, as it is easy to switch to the highest possible frequency without having to change the transducer. The use of more than one transducer, that is, both curvilinear and linear, may be necessary, particularly in the assessment of the acute abdomen.
- A cine facility on the ultrasound machine is invaluable, and cuts down scanning time. Colour Doppler is essential in the assessment of hepatobiliary problems and the examination of the acute abdomen.
- Generally speaking it is necessary to employ a fast frame rate. (The smaller field of view needed for children allows the line density to remain high, maintaining adequate resolution.)

HEPATOBILIARY PATHOLOGY

Cystic fibrosis

Cystic fibrosis (CF) is a common, autosomal recessive multisystem disease. The main organs affected are the lungs, liver and pancreas. Accumulation of mucus in the bronchi rapidly leads to respiratory problems including infections, with a predisposition to abscess formation with destruction of the terminal bronchioles developing into bronchiectasis.

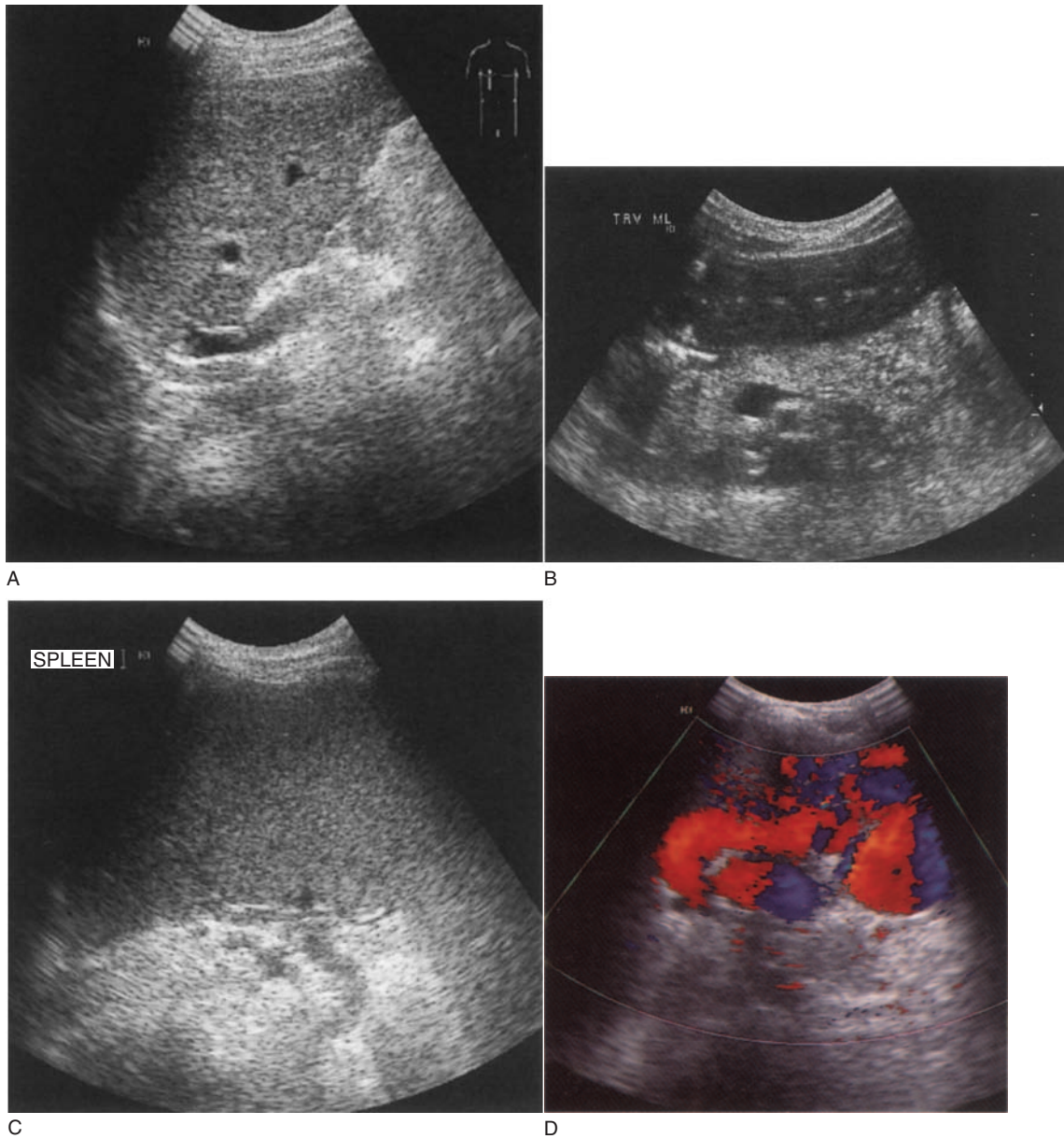
Pancreatic insufficiency, requiring enzyme supplements, is a feature of CF, with gradual fatty replacement and subsequent fibrosis of pancreatic tissue, resulting in increased echogenicity of the pancreatic parenchyma. The pancreas is generally reduced in size. Cysts, calcification and ductal dilatation may also be found.[1] Advances in the management of pulmonary problems associated with CF have led to longer survival and a subsequent increase in the prevalence of chronic liver disease. Annual ultrasound examination is recommended as sonographic changes may be identified in the absence of abnormality on biochemical assessment.[2] The liver may be hyperechoic and the texture becomes coarse and nodular as fibrosis develops (Fig. 9.1). Increased periportal echogenicity may be demonstrated. Eventually cirrhosis develops, causing portal hypertension. Assessment of the portal venous system with colour and spectral Doppler is useful, providing a baseline with which to compare progression of the disease.

The gallbladder is small in up to one-third of patients[3] (Fig. 9.1E). This microgallbladder measures less than $3 \times 1 \times 1$ cm after fasting and is filled with mucus. Up to 10% of patients with CF may have gallstones; cholecystitis and biliary strictures may occur.

Neonatal cholestasis and biliary atresia

Neonatal hepatitis and biliary atresia are the most common causes of neonatal cholestasis, presenting around the age of 4 weeks with neonatal jaundice, dark urine and pale stools. Early diagnosis of biliary atresia and differentiation from hepatitis and other causes of neonatal cholestasis is crucial to successful treatment. The aetiology of biliary atresia remains unclear but progressive inflammation, destruction and fibrosis of the biliary tree occurs, resulting in obliteration of all or part of the bile ducts and gallbladder, with the subsequent development of biliary cirrhosis.[4]

The ultrasonic features of neonatal hepatitis and biliary atresia overlap. The gallbladder is generally small and thick-walled or absent in biliary atresia, but may occasionally appear normal,[5] whereas in hepatitis the gallbladder, although often normal in size, may be difficult to visualize. The presence or absence of a gallbladder is not a reliable sign of biliary atresia. In cases of biliary atresia where only the hepatic duct is atretic (that is, with a normal gallbladder and common duct), the gallbladder can appear normal in size and contract postprandially. The liver may appear normal but in more severe cases the liver parenchyma shows increased echogenicity due to developing cirrhosis. The intra- and extrahepatic biliary tree is not dilated, although occasionally small choledochal cysts or



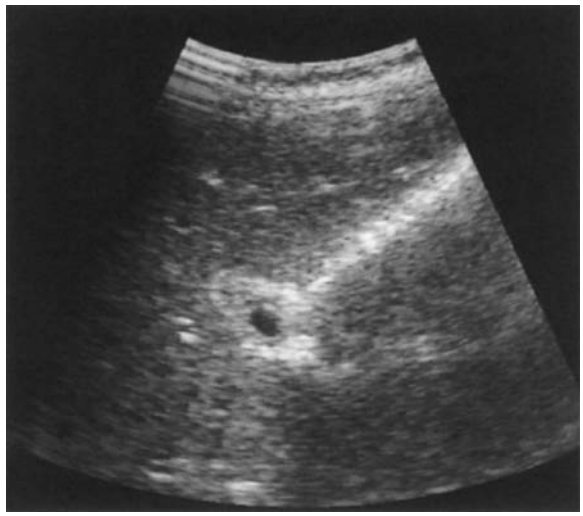
A B C D

Figure 9.1 Cystic fibrosis (CF). (A) Coarse, hyperechoic liver with a lobulated outline. At least two discrete nodules can be seen at the inferior margin. (B) Hyperechoic, coarse pancreas, typical of CF. (C) Massively enlarged spleen caused by portal hypertension in CF. (D) Varices around the lower margin of the spleen in a 12-year-old CF sufferer.

(Continued)

intrahepatic bile lakes may be seen close to the porta hepatis in infants with biliary atresia. Approximately 10–20% of infants with biliary atresia have associated congenital abnormalities, including choledochal cyst, situs inversus, polysplenia, preduodenal portal vein and interruption of the inferior vena cava (IVC) with azygous continuation, all of which may be detected on sonography.

The main role of sonography is to exclude other less frequent causes of neonatal cholestasis such as a congenital choledochal cyst and obstruction to the common bile duct due to bile inspissation where



E

Figure 9.1 cont'd (E) Microgallbladder. The gallbladder is thick-walled and small, despite fasting.

biliary tract dilatation will be noted. The diameter of the normal common bile duct should not be greater than 2 mm in the infant up to 1 year old (or 4 mm in children up to 10 years of age).[6]

Liver biopsy and radioisotope studies are used to differentiate biliary atresia from neonatal hepatitis. Excretion of radionuclide from the liver into the duodenum excludes biliary atresia although a lack of excretion into the duodenum may be seen in both atresia and severe neonatal hepatitis. In these cases laparotomy with intraoperative cholangiogram will be necessary to reach a final diagnosis, although in a few centres MR cholangiography and/or endoscopic retrograde cholangiopancreatography (ERCP) have been used to identify a patent biliary tree and thus exclude the diagnosis of biliary atresia.

Biliary atresia is usually treatable by early surgery, provided the diagnosis is made before the age of 8 weeks, at which time irreversible biliary cirrhosis may have developed. Liver transplant may eventually be required, particularly in those presenting late with established biliary cirrhosis.

Choledochal cyst

Choledochal cysts are congenital dilations of the biliary tree that may present at any age, and can be diagnosed in the fetus during routine obstetric scanning. In the neonate the main presenting feature will be cholestatic jaundice but the classic triad of pain, jaundice and a palpable mass is more likely to be seen in the young adult. A number of types of choledochal cysts have been recognized and in many cases there is an anomalous insertion of the bile duct into the pancreatic duct of Wirsung. On sonography a well-defined cyst will be identified close to the porta hepatis and in about 50% of patients there will be dilatation of the proximal bile ducts which may be seen to communicate directly with cyst (Fig. 9.2A). Sludge or calculi may be seen within the cyst. Small choledochal cysts may be seen in association with biliary atresia but in these cases there will be no associated biliary tract dilatation (Fig. 9.2B). Definitive diagnosis is made by MR cholangiography, although scintigraphy and ERCP may be useful in difficult cases.[7]

Other causes of cholestasis in children and neonates include bile duct stones (more common in girls), sclerosing cholangitis, CF, infections and Alagille's syndrome (a congenital paucity of the bile ducts). Acute cholestasis may also be caused by viral hepatitis, drugs, toxins, metabolic diseases or hypoxaemia.

Hepatoblastoma and hepatocellular carcinoma (HCC)

Primary malignant tumours of the liver are comparatively rare in children and frequently present as a large abdominal mass. Large hepatic tumours may present acutely as a result of haemorrhage. Hepatoblastoma is the commonest primary liver malignancy in childhood, generally occurring in children under 3 years of age, and may be associated with predisposing conditions such as Beckwith–Wiedemann syndrome and children infected with HIV[8] (Fig. 9.3A). HCC is more usually associated with chronic liver disease and tends to develop during the later stage of disease with peak incidences of 4–5 years and 12–14 years. Both tumours are associated with increased levels of serum alpha-fetoprotein.

On ultrasound, these tumours appear solid, heterogeneous and are often large and poorly demarcated from the adjacent liver parenchyma. Areas of necrosis or haemorrhage may be identified in the mass. Occasionally they may be multifocal. Although

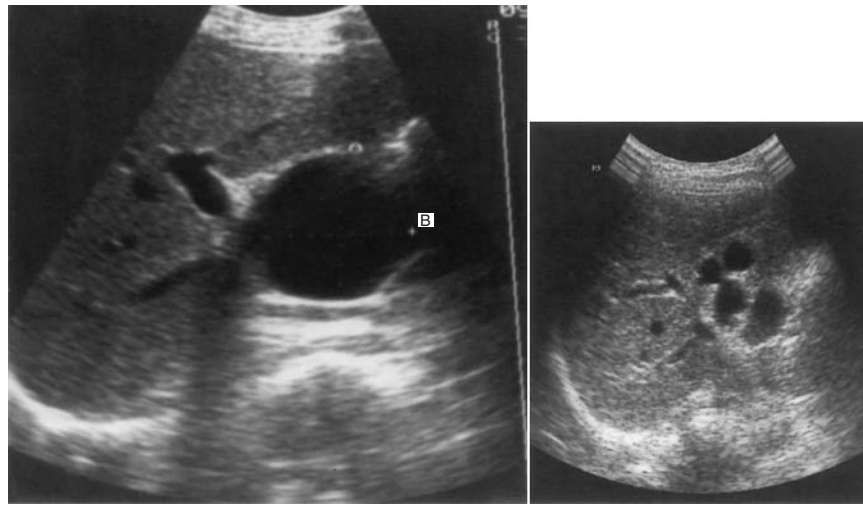


Figure 9.2 (A) Large choledochal cyst at the porta hepatis. (B) Intrahepatic choledochal cysts in biliary atresia with no proximal biliary tract dilatation.

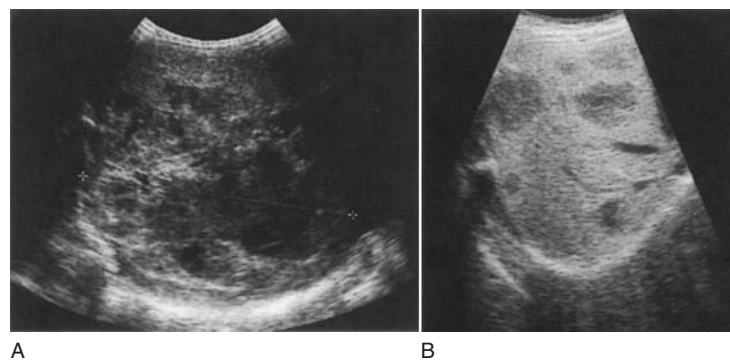


Figure 9.3 (A) A large hepatoblastoma, containing both cystic and solid areas, occupies most of the right lobe of the liver in this 18-month-old girl. (She is undergoing chemotherapy at the time of going to press.) (B) Multiple hypoechoic haemangiomata. Hyperdynamic circulation was noted in the portal vein and hepatic artery on Doppler. These lesions spontaneously regressed, leaving only a solitary haemangioma at the time of going to press.

the two types of tumour are not distinguishable on ultrasound, the clinical history may give a clue and ultrasound-guided biopsy can be used to obtain a histological diagnosis.

Ultrasound is useful in identifying the extent of the tumour and, when combined with colour flow Doppler imaging, adjacent vascular invasion can be evaluated. CT or MRI complements the ultrasound findings and is essential for staging and assessment of suitability for resection or transplantation.[9] Chemotherapy may be used to shrink the tumour prior to surgery.

Rhabdomyosarcoma is a rare tumour which may originate in the biliary ducts, causing biliary dilatation. It is indistinguishable from other liver tumours on ultrasound. Rhabdomyosarcoma originates from muscle cells and is the commonest type of soft-tissue sarcoma seen in childhood, with a

peak incidence before 5 years of age. It occurs in various sites throughout the body.

Other causes of focal liver lesions

Liver metastases may occur from most paediatric malignancies, particularly neuroblastoma, rhabdomyosarcoma and Wilms' tumour (p. 229). Leukaemia and lymphoma may also cause focal defects in the liver. Liver involvement may be manifested by hepatomegaly with normal liver texture, a non-specific sign, or by diffuse coarsened liver texture with or without hepatomegaly.

Haemangioendothelioma

Vascular tumours account for most benign liver tumours in childhood, with haemangioendotheliomas being seen more frequently than cavernous haemangiomas. Although haemangioendothelioma may be asymptomatic, infants generally present before the age of 6 months with an abdominal mass, respiratory distress, anaemia and cardiac failure, caused by the shunting of blood from the aorta through the tumour. Large tumours may bleed spontaneously, resulting in haemoperitoneum. They may present with jaundice and increased transaminase levels and 50% of children also have cutaneous haemangioma.[10]

These tumours are generally multiple, of varying echogenicity and may have a complex echotexture due to thrombus, calcifications and internal septations (Fig. 9.3B). The vascular nature of these lesions is demonstrated by a large coeliac axis and marked decrease in the size of the aorta below the origin of the coeliac axis. The main differential diagnosis of multiple haemangioendothelioma is from metastatic liver disease, particularly from disseminated neuroblastoma.

Although most asymptomatic paediatric haemangioendotheliomas regress spontaneously, those complicated by cardiac failure require active treatment. Steroids may be administered and serial ultrasound scans may be used to monitor the gradual resolution of the lesion. Angiographic embolization or surgical ligation of the major feeding vessels of the hepatic artery may be necessary in severe cases that fail to respond to steroid therapy.

PANCREAS

Normal appearances

The acoustic characteristics of the pancreas vary with age. Pancreatic echogenicity is quite variable and is occasionally hypoechoic in neonates compared with the adult gland. In older children echogenicity is equal to or slightly greater than that of the liver. The pancreas is relatively larger in young children than in adults, gradually increasing with age, reaching adult size in late teens.[11] The pancreatic duct is often visualized but should not be greater than 2 mm in width. The relative hypoechogenicity and relatively larger size of the normal pancreas in childhood should not be misinterpreted as a sign of probable pancreatitis when scanning a child with abdominal pain (Fig. 9.4).

Pathology of the pancreas

Pancreatic abnormalities are relatively uncommon in childhood. Most ultrasound abnormalities are the result of infiltrative processes associated with other syndromes or diseases (Table 9.1). Focal pancreatic lesions are rare.

Ultrasound is an ideal investigation for evaluating the paediatric pancreas, as a high-frequency

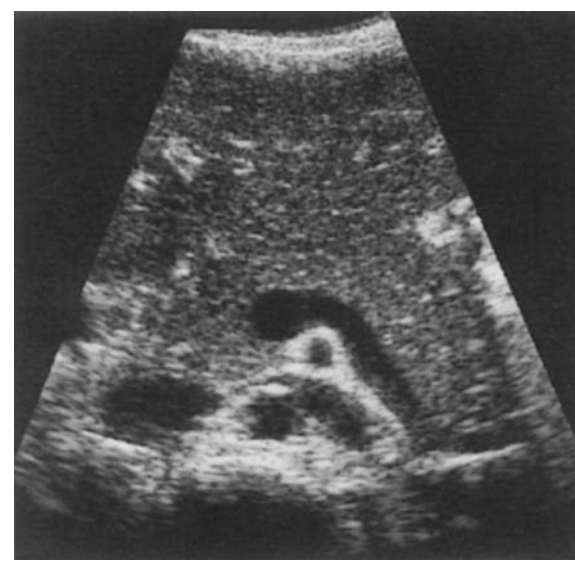


Figure 9.4 Normal pancreas in a 13-year-old girl—relatively hypoechoic and bulky in comparison with the adult gland.

Table 9.1 Paediatric pancreatic abnormalities

Increased echogenicity
Cystic fibrosis
—fatty replacement of the pancreas, calcifications, ectatic pancreatic duct, coarse texture, cysts
Pancreatitis
—hereditary
—trauma (physical abuse, road traffic accident)
—congenital anomaly, e.g. choledochal cyst
—drug toxicity
—viral and parasitic infection
Haemochromatosis
—pancreatic fibrosis, iron deposition in liver and pancreas
Focal lesions
Cysts
—isolated congenital cyst
—autosomal dominant polycystic disease
—von Hippel–Lindau disease
—Meckel–Gruber syndrome
Solid lesions
—primary pancreatic neoplasms are very rare in children

probe demonstrates excellent detail. A water-based drink may be given to provide an acoustic window. In cases of blunt injury to the abdomen with suspected pancreatic damage, CT is the imaging modality of choice in the acute situation, although sonography should be used during follow-up to detect the presence of a pseudocyst.

URINARY TRACT

Ultrasound is the first line of investigation in both antenatally detected abnormalities and in symptomatic children.

- The bladder should be scanned first, as voiding may often occur during the examination.
- Measurements of both kidneys, either length or renal volume, should be taken to highlight any difference in size and to provide a baseline for further growth comparison.
- A variety of planes can be used to view the kidneys in children. Often a posterior approach is best for obtaining an accurate bipolar length.
- Ensure that renal pelvic dilatation is not physiological, by rescanning postmicturition.
- Measure the anteroposterior diameter of any renal pelvic dilatation in transverse section through the renal hilum.
- Always scan the bladder immediately after micturition, paying attention to the ureteric orifice and looking for any ureteric or renal dilatation which may suggest reflux. Measure any residual volume.
- Colour Doppler may be helpful in identifying the ureteric orifice, by locating the jets of urine entering the bladder (Fig. 9.10D).

Normal appearances

After birth the renal cortex is relatively hyperechoic compared to the adult kidney, in strong contrast to the hypoechoic medullary pyramids. The outline of the kidney is often lobulated due to a persistent fetal lobulation. The renal pelvis is relatively hypoechoic, as the fat deposition seen in the adult is not yet present (Fig. 9.5A).

Gradually the cortex becomes less hyperechoic with age, the corticomedullary differentiation lessens and fat deposition in the renal sinus becomes more evident. The outline becomes smooth, although fetal lobulations do persist in some adult kidneys.

Normal postnatal growth of the kidneys, in terms of length and volume, is closely related to the height, weight and age of the child. Charts giving normal age- and weight-related values should routinely be referred to.[12] Errors do occur in measurements of renal length with a potential error in the order of 1 year's growth.[13] Thus follow-up measurements for renal growth should not be undertaken at intervals of less than 1 year.

Anatomical variants and pathology

The duplex system

The duplex system is one of the more common congenital anomalies, occurring in up to 9% of referrals.[14] It stems from aberrant budding of the Wolffian duct in utero, and can take a variety of forms, from complete duplication with two kidneys, each with a separate ureter, to a partial duplication involving the kidney only. Complete

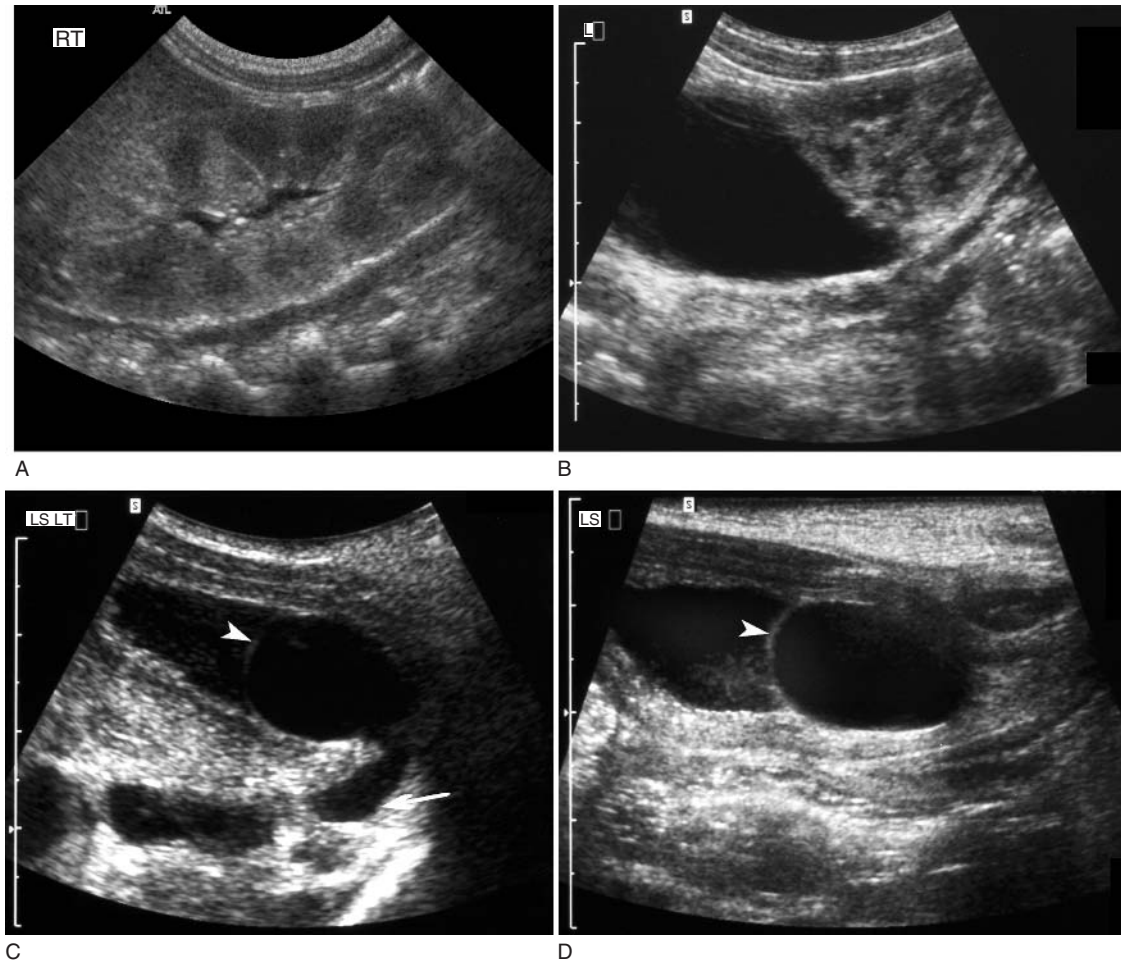


Figure 9.5 (A) Normal neonatal kidney, showing lobulated outline, hyperechoic cortex, increased corticomedullary differentiation and reduced renal sinus echoes. (B) Duplex kidney. The upper moiety is dilated with a thin cortex. The lower moiety is normal. (C) Same patient as in (B). Dilated ureter (arrow) of the upper moiety of the duplex kidney terminating in a uretercoele (arrowhead) seen in the base of the bladder at the vesicoureteric junction. (D) Ureterocoele (arrowhead) at the base of the bladder covering the urethral orifice.

duplication predisposes to reflux, particularly into the lower moiety and subsequently to infection.

The upper pole moiety of a duplex kidney is more prone to obstruction either secondary to a ureterocoele or, less commonly, with an obstructed ectopic ureter. In the former case the obstructed upper moiety may be associated with a dilated ureter which can be followed to the bladder where a ureterocoele, that is, a cystic dilatation of the distal ureter, may be seen within the bladder at the ipsilateral vesicoureteric junction (Fig. 9.5 B, C and D). The ureterocoele may extend into the urethra, causing bladder outlet obstruction which, if severe, may result in bilateral hydronephrosis.

In the absence of any dilatation, it may be difficult to demonstrate the duplex kidney on ultrasound. Generally, the kidney is longer than normal and two discrete, hyperechoic sinus echoes can be seen. Ectopic insertion of the upper pole ureter in a duplex system is a cause of urinary incontinence in girls. It may not be possible to follow an ectopic ureter to its distal end, even when dilated, but one may be able to demonstrate that the ureter is passing distally to the bladder.

When there is a strong clinical suspicion of an ectopic ureter an intravenous urogram or MR urogram will be required to identify a duplex kidney and site of ureteric insertion.

Renal fusion and ectopia

The horseshoe kidney is the most common form of renal fusion, in which the lower poles of the kidneys are fused with a central isthmus or 'bridge' across the front of the spine (Fig. 9.6). The isthmus frequently lies behind gas-filled bowel and can be difficult to detect. The sonographer should be suspicious of a horseshoe kidney when the lower poles of the kidneys cannot be clearly outlined, particularly when both kidneys look a little smaller than expected for age. Always ensure you see the outline of the lower poles clearly by turning the child prone or by scanning coronally through the side if necessary.

A dimercaptosuccinic acid (DMSA) scan may demonstrate the isthmus or bridge of renal tissue (when the ultrasound scan is equivocal) but only if it is functioning. In some cases the bridge is composed of non-functioning, fibrous tissue.

Fusion can take other forms, including an L shape, where one kidney lies horizontally across the midline; crossed ectopia, where both kidneys lie on the same side; H-shaped fusion of the hilar regions; and complete fusion to form a 'cake'-shaped solitary kidney.

Ectopic kidneys occur most frequently in the pelvis (Fig. 9.7). In rare cases the kidney may be situated in the thorax.

Ectopic and horseshoe kidneys are often associated with a degree of malrotation of the kidney. This can be associated with a degree of obstruction at the pelviureteric junction, and predispose to the development of renal calculi.

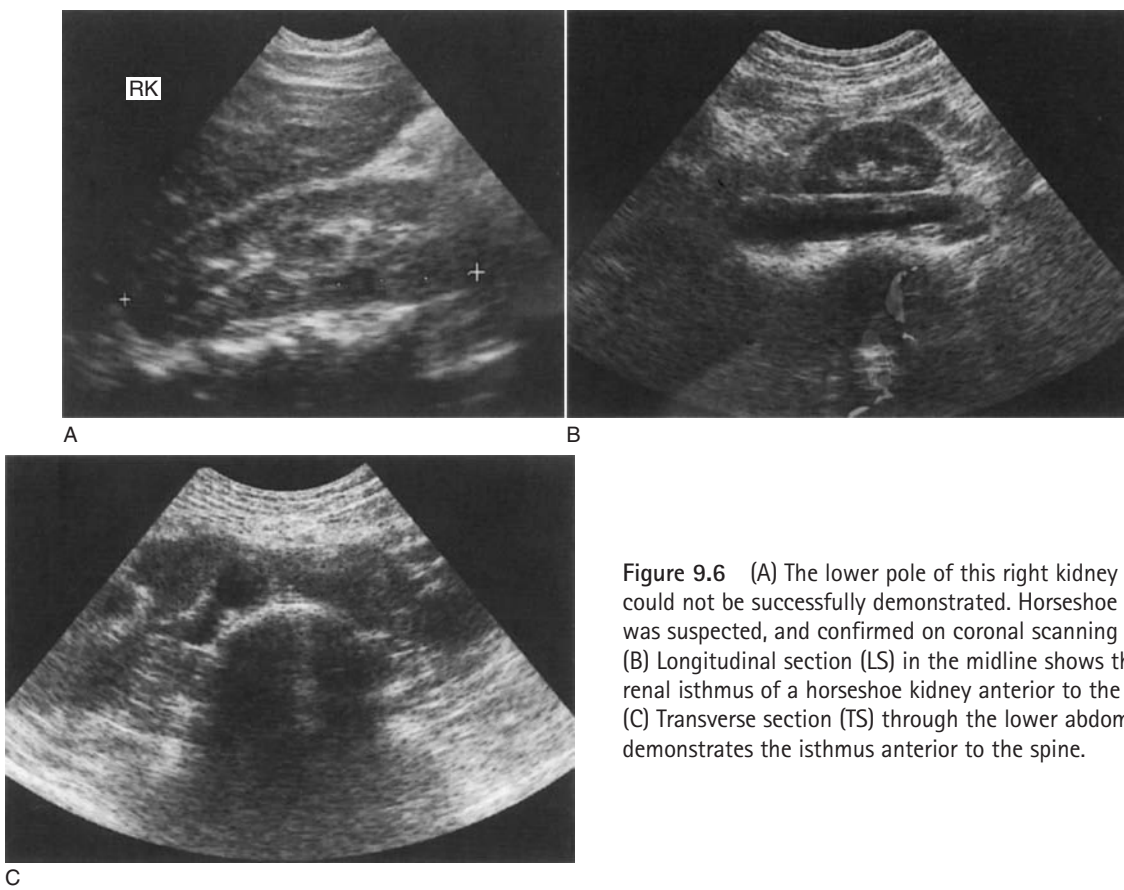


Figure 9.6 (A) The lower pole of this right kidney (RK) could not be successfully demonstrated. Horseshoe kidney was suspected, and confirmed on coronal scanning planes. (B) Longitudinal section (LS) in the midline shows the renal isthmus of a horseshoe kidney anterior to the aorta. (C) Transverse section (TS) through the lower abdomen demonstrates the isthmus anterior to the spine.

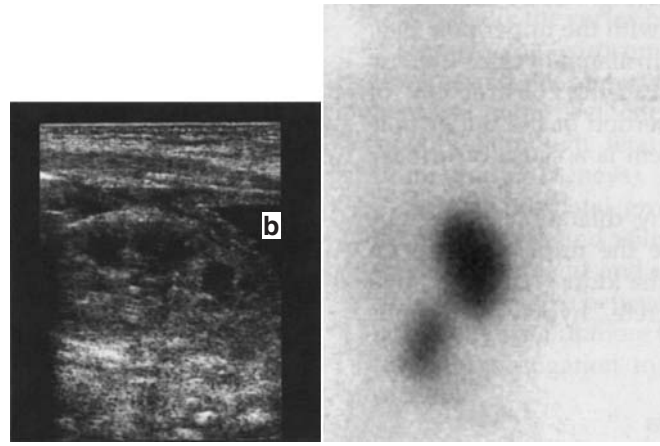


Figure 9.7 (A) Pelvic kidney (b = bladder). (B) Dimercaptosuccinic acid (DMSA) scintigraphy shows 33% function in the smaller, pelvic RK and 66% in the left kidney (LK).

Renal agenesis

The kidneys form from the ureteric bud, which arises from the pelvic area during the fifth to sixth week of gestation. The bud undergoes numerous divisions, forming the ureters, renal pelvis, calyces and renal tubules. Any interruption of this process may cause renal agenesis or ectopia.

Bilateral renal agenesis is lethal and is usually diagnosed prenatally. The incidence of unilateral renal agenesis is about 1:450 live births and is usually prenatally detected. Ultrasound is useful in confirming the prenatal diagnosis and excluding the presence of an ectopic kidney. A DMSA scan confirms the diagnosis. Renal agenesis is associated with VATER syndrome and with ipsilateral gynaecological anomalies in girls.

Multicystic dysplastic kidney (MCDK)

The MCDK is generally the result of complete, early ureteric obstruction in utero before 10 weeks, and is frequently diagnosed antenatally. The resulting kidney is non-functioning and contains cysts of varying sizes, separated by echogenic 'dysplastic' renal parenchyma. In general the cysts do not communicate but occasionally some communication can be seen, making differentiation from a severe hydronephrosis difficult.

MCDK is usually unilateral and is considered a benign condition, although there is a slight risk of malignancy and hypertension in later life. The kidney gradually involutes and often completely disappears (Fig. 9.8A, B). Surgical removal is unnecessary unless symptomatic due to its large size or is associated with repeated episodes of infection. Provided the contralateral kidney is normal, with good function, the prognosis is good. There is, however, an increased risk of associated urinary tract anomalies, such as ureterocoele, vesicoureteric reflux or contralateral pelviureteric junction obstruction, which may predispose to infection. These can be demonstrated with ultrasound and micturating cysto-urethrogram.

A DMSA scan differentiates MCDK, which is completely non-functioning, from a grossly hydronephrotic kidney, a distinction which may sometimes be difficult to make on ultrasound. Follow-up ultrasound scanning is generally advised in view of the slight increased risk of Wilms' tumour and to monitor the growth of the contralateral kidney.

Polycystic disease of the kidneys

Autosomal recessive polycystic disease of the kidney (ARPCDK: infantile) may be diagnosed prenatally. Both kidneys are abnormal, being large and hyperechoic, with loss of corticomedullary differentiation (Fig. 9.8C). There is a spectrum of severity of disease and in some cases it may present later

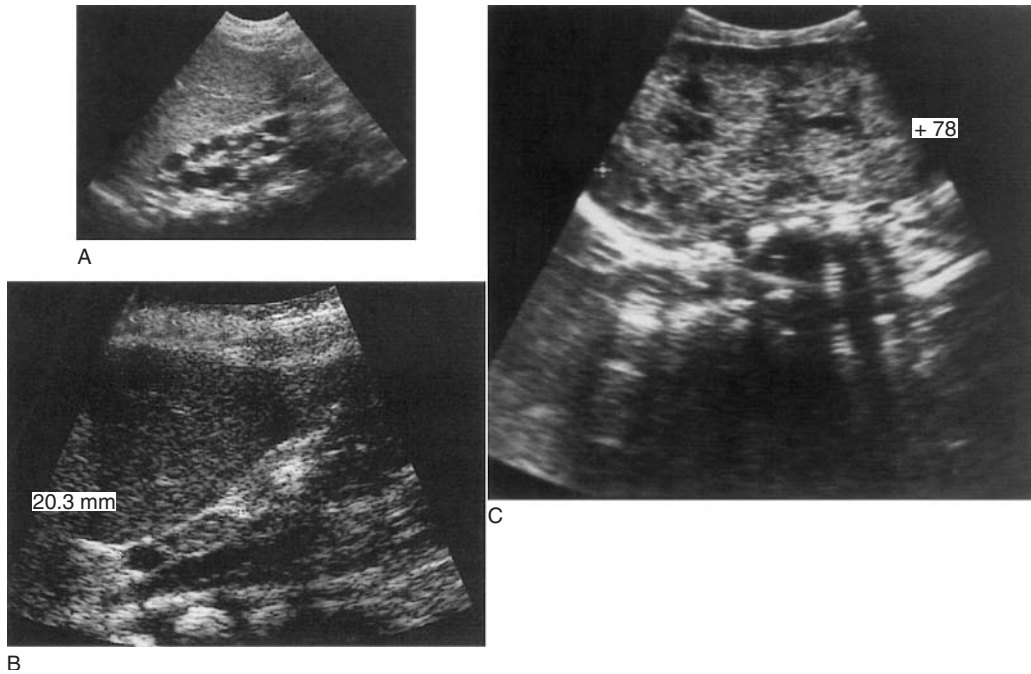


Figure 9.8 (A) Multicystic dysplastic kidney. (B) This multicystic dysplastic kidney, diagnosed antenatally, has shrunk to little over 2 cm in length by the age of 1 year. (C) Large, hyperechoic kidneys in a neonate in autosomal recessive polycystic disease.

in childhood with the milder, juvenile form of the disease. Prenatally the less severe forms appear normal on ultrasound. ARPCDK is associated with hepatic fibrosis and portal hypertension.

Autosomal dominant polycystic disease of the kidney (ADPCDK: adult) also has a wide spectrum of severity. Although it tends to present later in life, the more severe forms can present in childhood and can occasionally be diagnosed prenatally. Frank cysts can usually be demonstrated on ultrasound, but may not be detected until the second or third decade of life. The disease is also associated with cysts in the liver and pancreas, and with intracranial berry aneurysms.

Renal dilatation

Hydronephrosis is frequently detected antenatally, although the cause may be difficult to demonstrate. Dilatation is due either to obstructive uropathy, for example vesico- or pelviureteric junction obstruction, posterior urethral valves or obstructed upper moiety of a duplex kidney (Fig. 9.9), or it may be non-obstructive, for example due to reflux (Fig. 9.10).

Postnatal ultrasound scans should be performed when the infant is more than 4 days old, because there is commonly a period of dehydration immediately after birth. This may cause an obstructed or otherwise dilated kidney to appear normal for the first few days of life. If normal a follow-up scan is generally recommended at about the age of 6 weeks.

The presence of any calyceal dilatation or ureteric dilatation, as opposed to dilatation confined to the renal pelvis, is an important factor to note, indicating a greater degree of severity. A measurement of the anteroposterior diameter of the dilated intrarenal pelvis is a useful baseline from which to compare subsequent follow-up scans (Fig. 9.9D). It should be noted that slight separation of the renal pelvis is a normal finding in the newborn: an anteroposterior renal pelvis of 5 mm is the upper limit of normal.

The presence of a baggy, extrarenal pelvis, less than 10 mm, without pelvicalyceal system (PCS) dilatation is usually managed conservatively using ultrasound monitoring to demonstrate any increasing dilatation. PCS dilatation with a renal pelvic diameter of between 10 and 20 mm is more serious

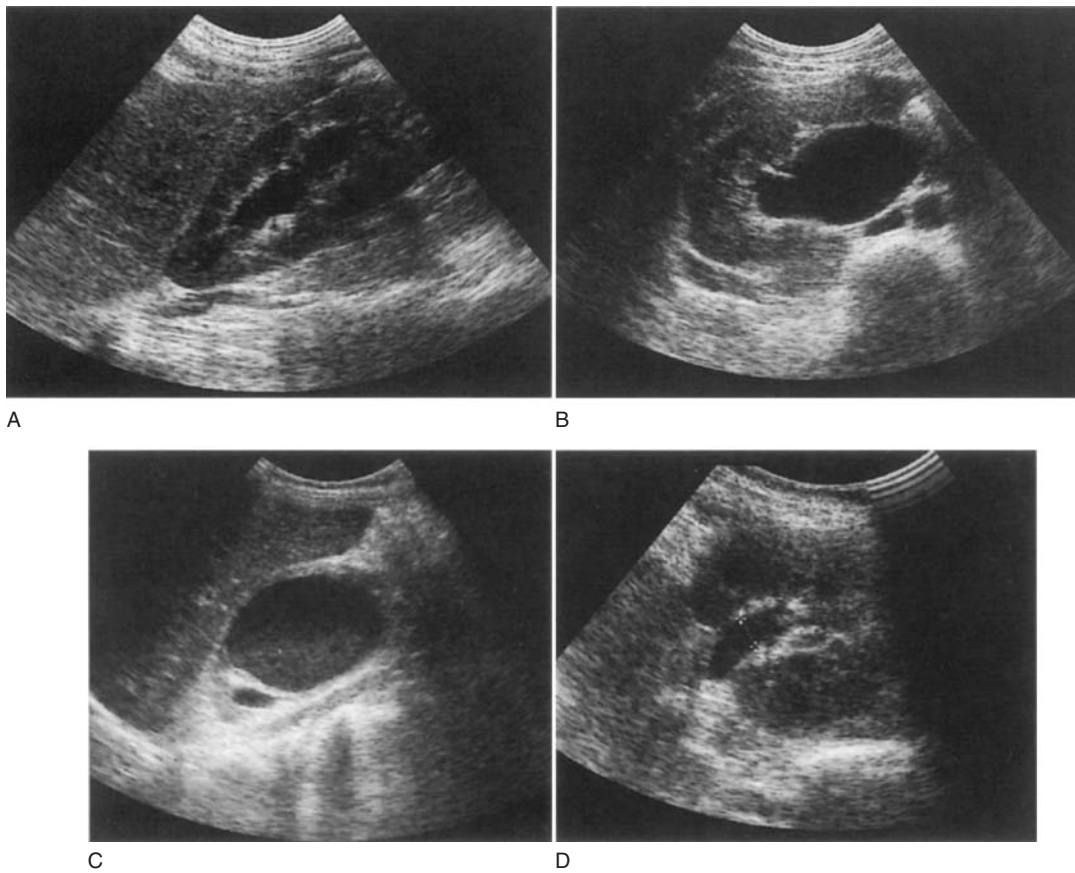


Figure 9.9 Renal dilatation. (A) Dilatation of the pelvicalyceal system (PCS) due to pelviureteric junction obstruction. (B) TS of the same kidney. The ureter was not dilated. (C) Duplex RK with gross dilatation of the lower pole moiety containing echoes due to infection. The cortex is thin. The smaller upper pole moiety is also dilated. A ureterocoele was present at the right vesioureteric junction (VUJ). (D) Mild dilatation of the LK. An anteroposterior (AP) measurement of the PCS provides a good baseline for follow-up.

and likely to require an assessment of renal function with a MAG3 renogram. Conservative treatment is possible, but surgery may be required for very poor function.

The dilated renal tract is predisposed to infection due to ascending infection in reflux or haematogenous infection in an obstructed system, where a pyonephrosis requiring percutaneous nephrostomy may develop. As a consequence antibiotic prophylaxis is frequently advised in the neonate with significant renal tract dilatation.

Bilateral renal tract dilatation in boys may be due to posterior urethral valves with secondary dilatation of the upper tracts due to the urethral obstruction. The diagnosis is confirmed by fluoroscopic micturating cystography. This diagnosis may be suspected sonographically by the association of bilateral hydronephrosis with a distended and thick-walled bladder.

Vesicoureteric reflux

Vesicoureteric reflux, the retrograde passage of urine from the bladder up the ureter and into the kidney, predisposes the child to urinary tract infection and the development of reflux nephropathy. In the first year of life only, reflux is more common in boys than in girls and is usually more

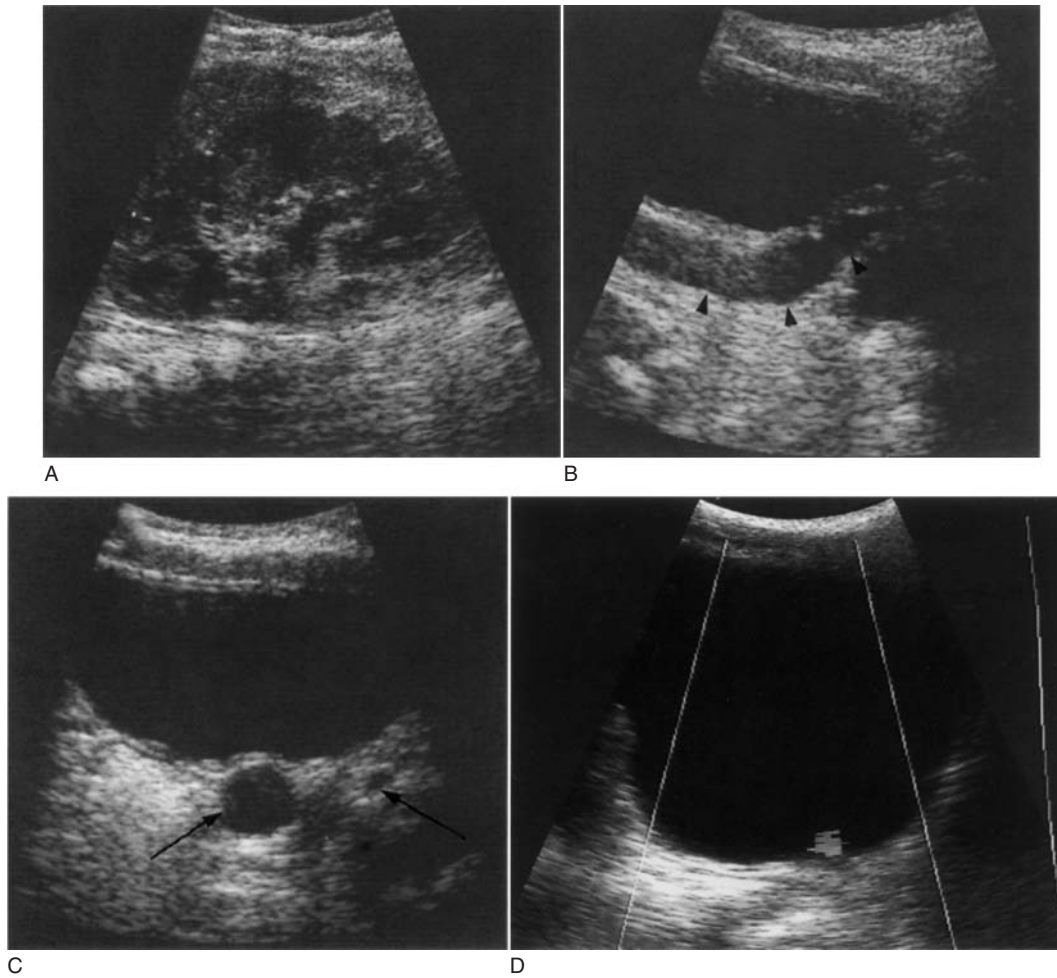


Figure 9.10 (A) Mild PCS dilatation due to reflux. (B) The right lower ureter is dilated (arrowheads) and urine was seen to reflux back up the ureter, dilating the kidney. (C) Bilateral reflux (right worse than left) is observed in a TS through the base of the bladder. Both ureters (arrows) are seen to dilate intermittently. (D) Compare (C) with a normal patient, in whom the vesicoureteric junctions can be identified by the presence of jets on colour Doppler. No ureteric dilatation can be demonstrated either before or after micturition.

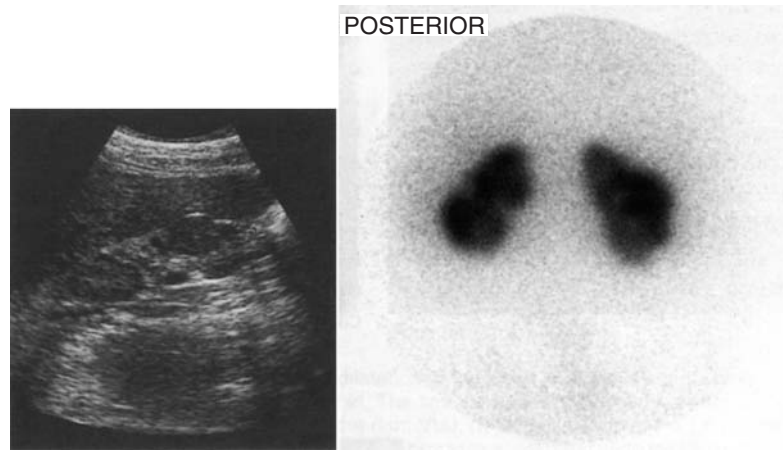
(Continued)

severe.[15] Conversely, after the first year of life reflux is more likely to present in girls and is often less severe. Vesicoureteric reflux is a common cause of hydronephrosis antenatally, accounting for up to 38% of all prenatal urinary tract dilatations, requiring ultrasound follow-up and antibiotic prophylaxis.[16,17]

Reflux may either be due to a developmental anomaly at the vesicoureteric junction, or the result of a neurogenic bladder, partial outlet obstruction or foreign bodies such as calculi and the presence of a catheter.

Children who have had one or more episodes of urinary tract infection should be investigated to search for an underlying cause and to identify evidence of reflux nephropathy (Tables 9.2 and 9.3). Approximately 2% of boys and 8% of girls will develop at least one urinary tract infection by 10 years of age, requiring investigation, and in most centres will account for a substantial proportion of the paediatric sonography performed.

Reflux itself is not reliably diagnosed by ultrasound as it is possible to have intermittent reflux in the presence of a normal ultrasound scan, with a



E F

Figure 9.10 cont'd (E) Small, scarred RK, due to reflux. (F) DMSA scan showing bilateral renal scarring due to reflux. Note in particular two wedge-shaped scars in the RK.

Table 9.2 Conditions associated with urinary tract infection (UTI)

- Vesicoureteric reflux
- Obstruction
  - —pelviureteric junction
  - —vesicoureteric junction
  - —posterior urethral valves
  - —duplex kidney with obstructed moiety/ectopic ureter
  - —ureterocoele
- Other structural anomalies
  - —duplex and/or ectopic renal anatomy
  - —multicystic dysplastic kidney
  - —prune belly syndrome
- Calculi
- Neurogenic bladder

non-dilated urinary tract.[17,18] There may be evidence of thickening of the uroepithelium of the renal pelvis due to intermittent renal pelvis distension. Uni- or bilateral dilatation may be present to a mild or severe degree and may involve the kidney and/or ureter (Fig. 9.10). It is important to scan the renal pelves and ureteric orifice immediately after micturition, when intermittent dilatation due to reflux may be demonstrated on an otherwise normal scan.

When dilatation *is* seen, the exact cause may be uncertain unless reflux is actually visualized, which is rare, and micturating cystography is required. Although most commonly performed conventionally by fluoroscopy using iodinated contrast medium, radionuclide cystography and more recently contrast sonocystography have been used as an alternative, particularly in the older child.[18]

The most common complication of reflux is infection and most children present with at least one episode of urinary tract infection. This can cause renal scarring. It is important to make the diagnosis of vesicoureteric reflux and renal scarring early in order to prescribe prophylactic antibiotics in an attempt to avoid the damaging complications caused by reflux of infected urine. The ultrasound appearances of scarring include a focal reduction in cortical thickness, irregular outline, interruption of or loss of the renal capsule echo or a disruption in the renal architecture. Colour flow and power Doppler may show triangular areas of decreased or absent blood flow (and occasionally increased flow) and can improve the detection rate of focal scarring on sonography.[19] These signs can be difficult to demonstrate in young children's kidneys, particularly when highly lobulated, and the most reliable method of scar detection is a DMSA scan (Fig. 9.10F).

Chronic reflux nephropathy leads to failure of renal growth, resulting in a shrivelled, poorly functioning kidney. Measurements of the maximum length of the kidneys should be routinely performed, and can be related to age, height and weight.[12] A difference in renal length of more than 10% between

Table 9.3 Imaging the paediatric renal tract

Ultrasound	First-line investigation in all cases. Excellent structural detail Limited sensitivity for duplex kidneys, reflux, ureteral pathology and small scars Monitoring of disease progression Monitoring of treatment
Contrast sonocystography	Alternative to X-ray or radionuclide cystography. Poor structural detail, unsuitable for the demonstration of urethral anomalies
Diuretic renogram (dynamic) Tc^{99m} MAG3	Outlines the pelvicalyceal system. Diagnosis of obstruction and relative renal function by analysis of excretion curves
Radionuclide cystography (dynamic) Direct (via catheter or suprapubic injection of isotope into the bladder) or indirect (following diuretic renogram)	Diagnosis of reflux
Cortical scintigraphy (static)—Tc^{99m} DMSA	Demonstrates uptake in the renal cortex Superior detection of renal scarring in vesicoureteric reflux and acute pyelonephritis Demonstration of congenital anomalies, e.g. ectopic or solitary kidney Analysis of *differential* renal function
Intravenous urography (IVU)	Limited use in children Assessment of level of ureteric obstruction Assessment of congenital anomalies, e.g. ectopic ureters and duplex kidney Postoperative evaluation
Micturating cystourethrogram	Accurate diagnosis of reflux, polyps, diverticula, strictures and urethral anomalies, but involving a significant radiation dose
Plain X-ray	Some calculi, mainly those in the ureter Of limited value in paediatric renal work-up May show gross spinal anomalies
CT	Reserved mainly for confirmation and staging of malignant tumours, due to significant radiation dose Renal trauma Increasingly used for ureteric calculus detection
MRI	Assessment of difficult congenital anomalies and focal masses. Staging of malignancy

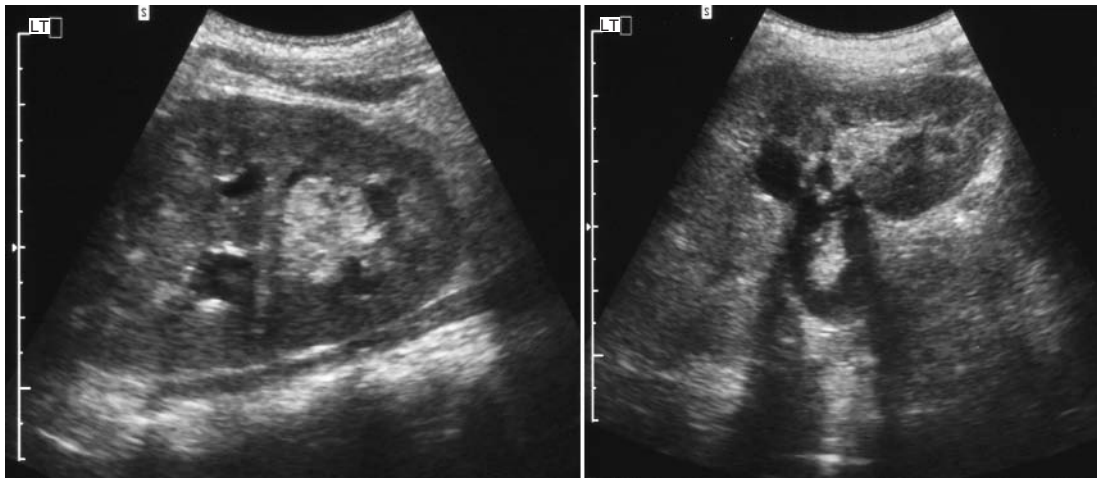
the two kidneys should prompt further investigation into renal function with a DMSA scan.

Fungal infection

Candidiasis is a fungal infection which is most commonly seen in infants who are acutely ill or immunocompromised or in sick ventilated neonates. Fungal balls dilate and may obstruct the collecting system of the kidney (Fig. 9.11). Ultrasound is particularly useful in making the diagnosis by demonstrating the hyperechoic fungal balls within the dilated collecting system. Fungal infection may also undergo haematogenous spread to the spleen and liver, where it can result in multifocal abscess formation.

Wilms' tumour

The most common paediatric renal malignancy, Wilms' tumour usually presents before the age of 3 years. Although the lesion generally occurs in previously fit individuals, there are several known predisposing conditions, including hemihypertrophy, Beckwith–Wiedemann syndrome and sporadic aniridia, with a 30–40% incidence in sporadic aniridia.



A B

Figure 9.11 (A) The dilated collecting system of this kidney is filled with a large, rounded fungal ball of candidiasis infection. (B) The fungus ball is seen to disintegrate, emptying into the renal pelvis.

The tumours are large at presentation, presenting with a palpable abdominal mass, and, less frequently, pain, haematuria and fever. About 5% of these tumours are bilateral.[20] The prognosis when unilateral is generally good.

The ultrasound appearances are of a relatively well-defined heterogeneous mass, predominantly solid but frequently with some necrotic or haemorrhagic areas, often almost completely replacing the kidney (Fig. 9.12). Small focal areas of calcification are seen very occasionally. A search should be made for tumour invasion of the renal vein and IVC which occurs in up to 10% of cases. Tumour invasion may extend into the right atrium. Occasionally a large, right-sided Wilms' tumour may compress the IVC but not invade it; colour or power Doppler may be useful in the difficult distinction between compression and invasion on ultrasound. Ultrasound also identifies associated lymphadenopathy, particularly in the para-aortic and paracaval regions, and metastatic liver disease.

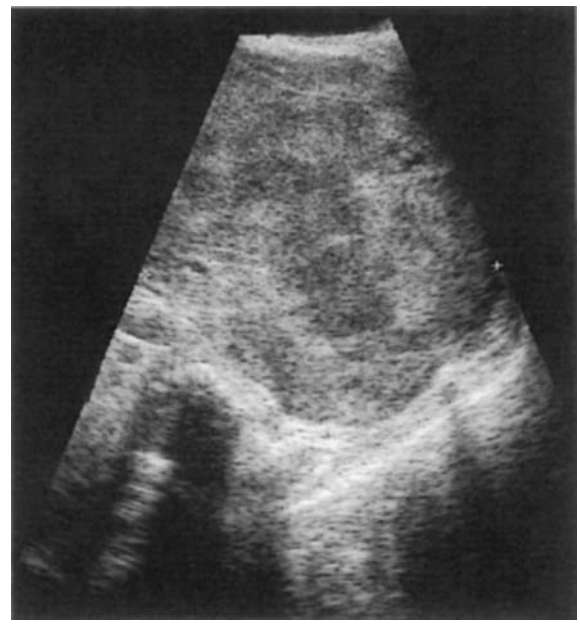
In a small percentage of cases, tumour may also be found in the contralateral kidney. This is usually much smaller than the mass on the presenting side and may be acoustically subtle. Up to 7% of contralateral tumours are missed on preoperative imaging due to their small size and the operator must be alert to the possibility of bilateral disease.[20]

Occasionally a Wilms' tumour may be found to be predominantly cystic, having the appearances of a large, multiloculated cystic mass. The main differential diagnosis would be of a mesonephric blastoma occurring during the first year of life and histology is required to establish the diagnosis.

In most cases, an ultrasound and chest radiograph are sufficient to diagnose correctly Wilms' tumour but CT of the chest and abdomen is generally used for staging, and to exclude metastatic disease in the chest and liver.[21] Percutaneous biopsy for confirmation of histological type is generally performed. CT or MRI is more sensitive than ultrasound scanning in demonstrating small tumours in the contralateral kidney.

Xanthogranulomatous pyelonephritis

Xanthogranulomatous pyelonephritis results from chronic infection in an obstructed kidney and children present with a history of general malaise, low-grade fever and flank pain and may be found to be anaemic. The finding of a palpable abdominal mass on examination often leads to an early diagnosis of a possible Wilms' tumour. On sonography the kidney is diffusely enlarged, with loss of the normal corticomedullary differentiation. The presence of calyceal dilatation with debris and calculi in the collecting system and confirmation of urinary infection in addition to the generalized involvement of the kidney helps to differentiate this condition from Wilms' tumour (Fig. 9.12B). Occasionally



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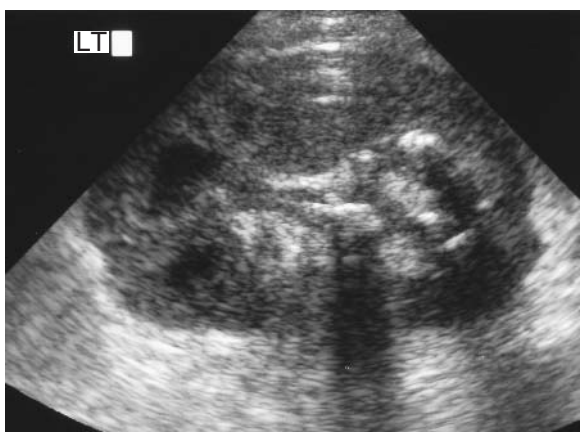

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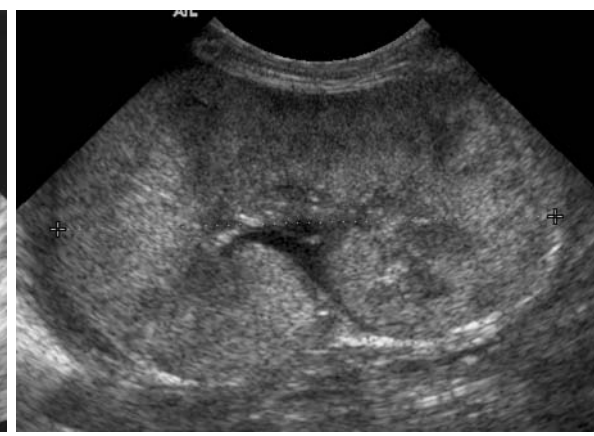


C

Figure 9.12 (A) Large Wilms' tumour arising in the left kidney and filling the left flank with a solid, heterogeneous mass. (B) Xanthogranulomatous pyelonephritis was the cause of the renal mass in this 8-year-old boy presenting with anaemia and a flank mass. (C) Renal vein thrombosis in a dehydrated neonate, showing an enlarged 'globular' kidney with loss of the normal corticomedullary differentiation.

CT scanning may also be helpful. The kidney will usually be found to be non-functioning on a DMSA scan and nephrectomy is required.

Renal vein thrombosis (RVT)

RVT primarily occurs in the neonatal period but may occur in the older child, particularly in association with renal malignancy and amyloidosis. Classically the sick neonate is noted to develop gross haematuria in association with a palpable abdominal mass. RVT is usually unilateral but may be bilateral and is associated with acute adrenal haemorrhage when left-sided. Sonographically the affected kidney is enlarged and globular and develops an inhomogeneous echogenicity of the renal parenchyma with areas of increased echogenicity due to haemorrhage (Fig. 9.12C). Thrombus may be detected in the

ipsilateral renal vein and IVC and Doppler sonography shows reduced or absent blood flow in the renal vein and loss of the normal variation in the renal vein waveform. Arterial flow is also decreased. On follow-up the kidney may completely recover due to the development of collateral blood flow or early recanalization of the renal vein, but in severe cases the kidney may atrophy and calcify.

ADRENAL GLANDS

Normal appearances

In utero and postnatally, the adrenal glands are large, about one-third the size of the kidney, and composed mainly of the bulky, hypoechoic fetal cortex which makes up about 80% of the gland. The neonatal adrenal glands are easily demonstrated on ultrasound. The bulky fetal cortex is sonographically apparent as a thick hypoechoic layer surrounding the thinner, hyperechoic adrenal medulla (Fig. 9.13A). The fetal cortex surrounds the smaller, permanent cortex and gradually starts to involute after birth. By the age of 2–4 months, the adrenal glands have attained their normal adult configuration of the thin, hypoechoic cortex with a tiny layer of hyperechoic adrenal medulla within.

Neuroblastoma

The neuroblastoma is a malignant tumour arising in the sympathetic chain, most commonly the adrenal medulla. The majority of neuroblastomas present before the age of 4 years with a palpable abdominal mass, and many already have metastases at the time of presentation to the liver, bone marrow, skin or lymph nodes. Table 9.4 lists the most frequent abdominal tumours occurring in childhood.

The tumour is usually large on presentation, displacing the kidney downwards and laterally. In some cases it may invade the adjacent kidney, becoming difficult to distinguish from a Wilms' tumour. Neuroblastoma is predominantly solid on ultrasound, having a heterogeneous texture and frequently containing calcification. The tumour margins are ill-defined and infiltrate the surrounding organs and tissues, crossing the midline and encasing vascular structures: it may be difficult to differentiate from lymphadenopathy (Fig. 9.13B, C and D). Nodes tend to surround and elevate the aorta and IVC.

MRI and CT are used for staging, particularly in assessing retroperitoneal spread.[22] Bone scintigraphy and MIBG scans are also useful in demonstrating metastases.

Adrenal haemorrhage

After birth, the bulky fetal cortex normally involutes. Adrenal haemorrhage occurs in the neonate as a result of trauma to the vulnerable fetal cortex during delivery or in association with perinatal asphyxia. Haemorrhage may occur in up to 2% of births.[23] This may be uni- or bilateral and may cause a palpable mass and abdominal pain. Ultrasound can be used to follow the resolution of the haemorrhage over a period of weeks; in the initial stages of haemorrhage the adrenal mass is hyperechoic, gradually liquefying into a well-defined mass of mixed echo pattern and becoming cystic (Fig. 9.13 E, F). This may completely resolve over a period of some weeks leaving a normal adrenal gland or the gland may become atrophic and calcify. In rare cases an adrenal haemorrhage may progress to an abscess.[24]

Adrenal calcification

Calcification of the gland in babies and infants is usually the result of previous infection or haemorrhage. Adrenal abscess cavities may calcify after successful treatment. Gross calcification in bilateral adrenal glands in association with hepatosplenomegaly in the infant indicates the likely diagnosis of Wolman's disease, an inborn error of lipid metabolism that is invariably fatal.

GASTROINTESTINAL TRACT

Bowel ultrasound in paediatrics is an established and readily accepted investigation, replacing contrast radiology in many cases. The range of potential applications continues to increase.[25] Most gastrointestinal tract scanning in paediatrics is best performed with a high-frequency (15–7.5 MHz) linear or small footprint curvilinear probe.

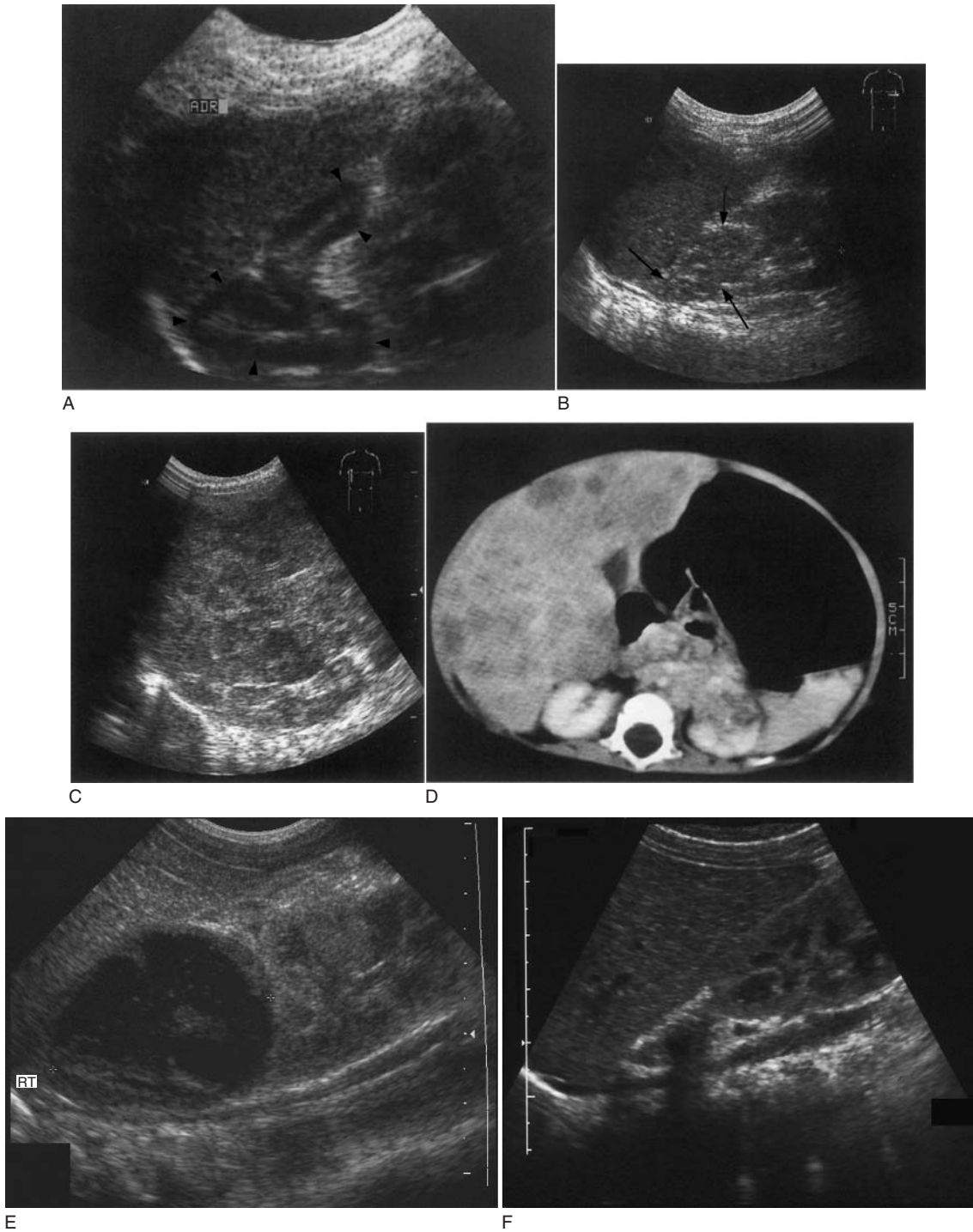


Figure 9.13 (A) Normal adrenal gland in a neonate, demonstrating the bulky, hypoechoic fetal cortex surrounding the thinner, hyperechoic medulla. (B) Left adrenal neuroblastoma. (C) Metastases were also present throughout the liver. (D) Confirmation of the left adrenal neuroblastoma and liver metastases on CT. (E) Adrenal haemorrhage in a neonate. (F) Same patient as in (E); 3 months later the haemorrhage has resolved and calcification has developed in the involuted adrenal gland.

Table 9.4 Paediatric abdominal malignancies

Wilms' tumour
Neuroblastoma
Hepatoblastoma
Hepatocellular carcinoma
Rhabdomyosarcoma
Leukaemia
Lymphoma

Hypertrophic pyloric stenosis (HPS)

HPS is a condition occurring in newborn infants commonly about 6 weeks of age when the pyloric muscle becomes hypertrophied and elongated, restricting the passage of gastric contents, causing projectile vomiting. Most infants with HPS are found to have a hypochloraemic alkalosis and, when seen in association with a palpable epigastric mass the size of an olive on test feeding, the diagnosis is clear without the need for ultrasonic imaging. However, ultrasound is very successful in demonstrating HPS in approximately 20% of infants in whom the pyloric olive cannot be palpated.[26,27]

The baby should be positioned comfortably right side down and the stomach and pylorus identified usually just to the right of the midline in the low epigastric region. A small feed, of approximately 20–30 ml of sugared water (preferable to milk as it does not contain echoes which may obscure vital detail), may be used to aid visualization of the gastric antrum if the stomach is empty. A nasogastric tube may also be used to administer clear fluid in a controlled way providing that the gastric position of the tube is confirmed prior to injection of the fluid. A small, high-frequency linear or curved linear transducer is best.

The pylorus projects into gastric lumen and is outlined by the fluid. HPS can be confirmed by the demonstration of:

- thickened and elongated pyloric muscle
- increased but ineffective peristalsis
- failure of the pylorus to relax and open

Various figures have been quoted for muscle thickness in hypertrophic pyloric stenosis ranging from 2.5 to 5 mm but 3 mm is most commonly accepted[28,29] (Table 9.5).

It must be stressed that the examination is dynamic and measurements of muscle thickness must be interpreted in conjunction with the observations of gastric peristalsis and failure of the pylorus to relax normally. Sensitivity and specificity of 97% and 99% for the diagnosis of HPS have been reported in expert hands (Fig. 9.14). If clinical suspicion persists after a negative ultrasound, a repeat examination after 1 or 2 days may be performed to exclude an evolving pyloric stenosis.

Intussusception

Intussusception is the invagination of a segment of bowel into the lumen of the adjacent bowel. It is a common paediatric emergency, especially in younger children aged 3 months to 3 years, and tends to affect the ileocaecal region.

The child presents with abdominal pain, sometimes with a palpable mass, vomiting or rectal bleeding. Intussusception can result in bowel necrosis and subsequently perforation requiring surgery.

The ultrasound appearances of bowel within bowel are characteristic. In cross-section, the bowel assumes a 'doughnut' configuration, with concentric rings of bowel wall (Fig. 9.15). Dilated loops of fluid-filled obstructed bowel may be demonstrated proximal to the intussusception.

The use of ultrasound to diagnose this condition is highly reliable,[30] reducing or eliminating the need for contrast radiology.

An air enema is most commonly used to reduce the intussusception using inflation pressures of up to 120 mmHg. Hydrostatic reduction (that is, with water/saline) under fluoroscopic or ultrasound control is also an accepted treatment.[31]

Table 9.5 Pyloric muscle dimensions

	Normal pylorus	Hypertrophic pyloric stenosis
Pyloric length (mm)	< 15	≥ 16
Pyloric width (mm)	< 11	≥ 11
Muscle thickness (mm)	< 2.5	≥ 3

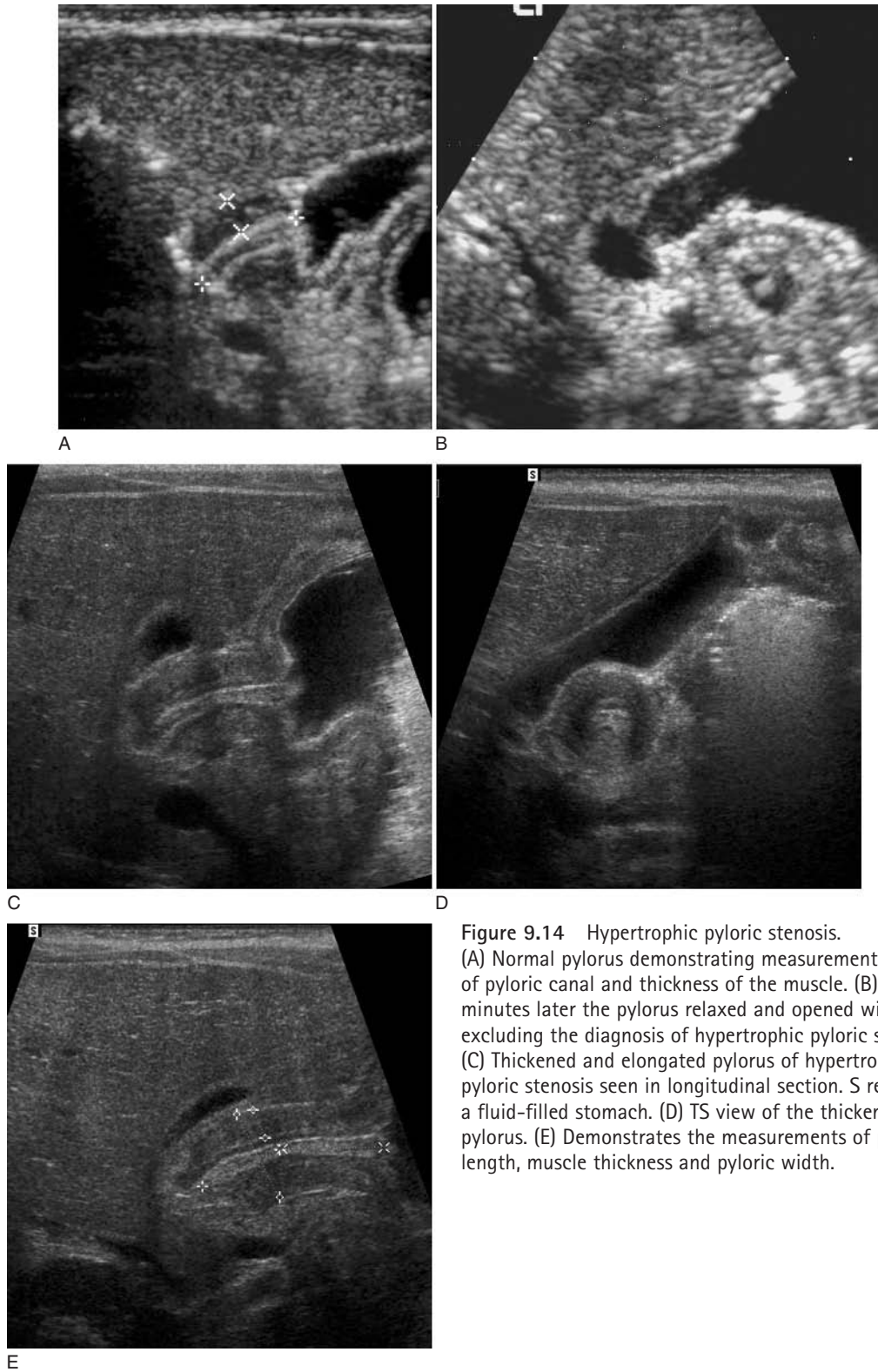
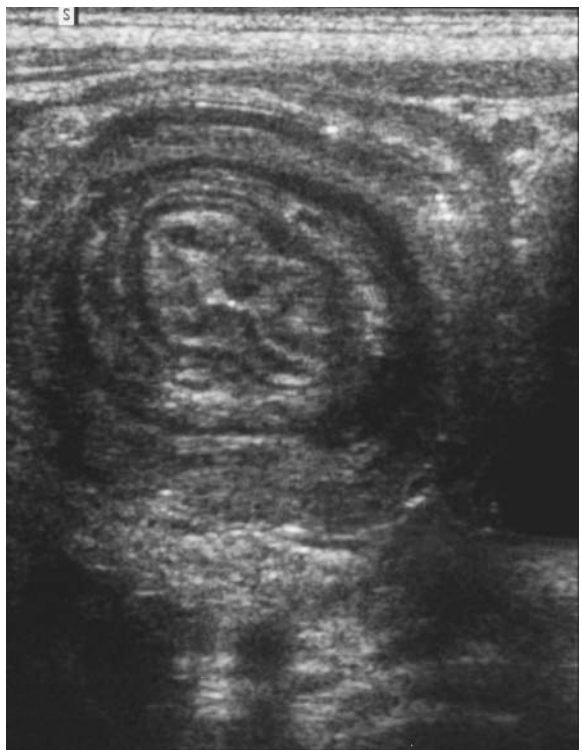


Figure 9.14 Hypertrophic pyloric stenosis. (A) Normal pylorus demonstrating measurement of length of pyloric canal and thickness of the muscle. (B) A few minutes later the pylorus relaxed and opened widely, excluding the diagnosis of hypertrophic pyloric stenosis. (C) Thickened and elongated pylorus of hypertrophic pyloric stenosis seen in longitudinal section. S represents a fluid-filled stomach. (D) TS view of the thickened pylorus. (E) Demonstrates the measurements of pyloric length, muscle thickness and pyloric width.

The main contraindications to attempting a non-surgical reduction are peritonitis and free intraperitoneal air. A number of sonographic features have been reported to be associated with a decreased success rate of non-surgical reduction, including a hypoechoic rim greater than 10 mm, absent blood flow on colour flow Doppler sonography, or a large amount of fluid trapped within the intussusception, but these findings are not contraindications to a careful attempt at non-surgical reduction.[32] Approximately 10% of cases recur whether the initial intussuception was treated surgically or non-surgically.

Midgut volvulus

Malrotation of the midgut occurs as a result of failure of normal rotation of the small bowel during intrauterine development, resulting in a shortened



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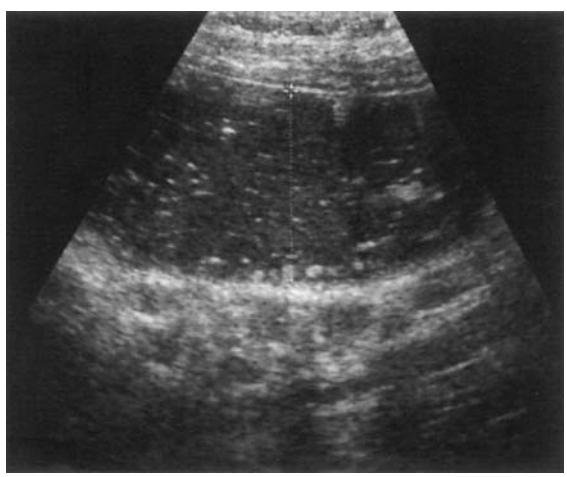

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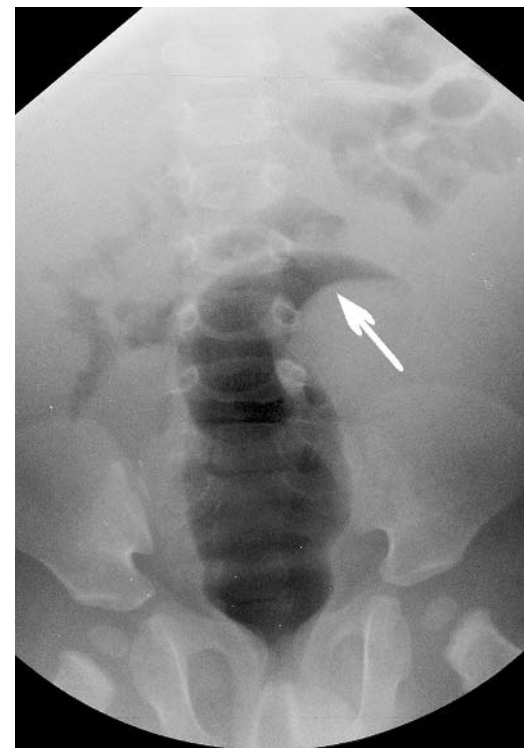

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Figure 9.15 Intussusception. (A) The characteristic appearance of bowel within bowel due to an intussusception. (B) Dilated, fluid-filled loops of obstructed bowel are seen proximal to the intussusception. (C) Air enema; the intussusceptum is seen indenting into the lumen of the air-filled sigmoid colon during a successful air enema reduction (arrow).

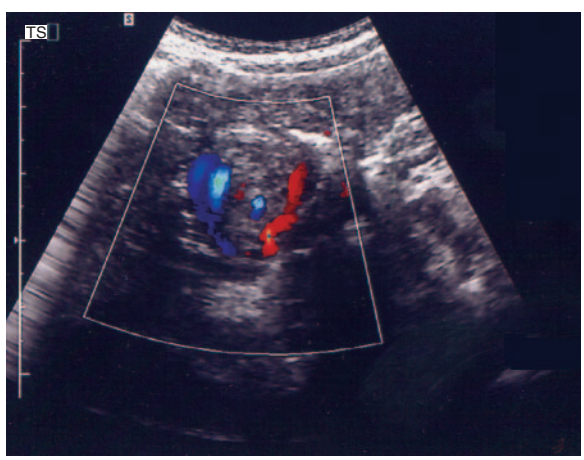
mesenteric fixation of the small bowel to the posterior abdominal wall. This predisposes the small bowel to twisting (volving) around the mesenteric vascular axis, resulting in bowel obstruction and vascular compromise with a risk of infarction of most of the small bowel if the volvulus is not treated quickly.

Following volvulus the child presents with acute pain and bile-stained vomiting. The bowel may intermittently twist and untwist, resulting in temporary alleviation of symptoms, which may make diagnosis more difficult.

The definitive diagnosis is usually made fluoroscopically during an upper gastrointestinal contrast study. In malrotation the duodenal jejunal flexure is generally found to be lower and in a more medial position than is normal and if a volvulus has occurred a corkscrew appearance of the volved small bowel may be seen (Fig. 9.16). The proximal duodenum will be dilated secondary to the duodenal obstruction.

Malrotation without volvulus may be suspected during a sonographic examination performed for intermittent abdominal pain due to the associated malposition of the mesenteric vessels and is best seen on colour Doppler sonography. The normal relationship of the superior mesenteric vein to the superior mesenteric artery is reversed, with the superior mesenteric vein lying anteriorly and/or to the left of the superior mesenteric artery.[33] However this finding is not always present and may occasionally be seen in normal individuals and therefore a contrast study is required for confirmation.

When volvulus has occurred the vessels may be noted to be spiralling around a bowel mass, that is, the 'whirlpool sign'[34] (Fig. 9.16). Other ultrasound appearances include a dilated, fluid-filled obstructed duodenum, although the obstructed duodenum may be gas-filled, obscuring visualization. This sign is not invariable, however, and a contrast study may still be needed to confirm or exclude the diagnosis of a midgut volvulus. Surgery is performed to untwist the bowel, which is then laid carefully in the correct position; attachment is usually unnecessary, as abdominal adhesions tend to stabilize the bowel.



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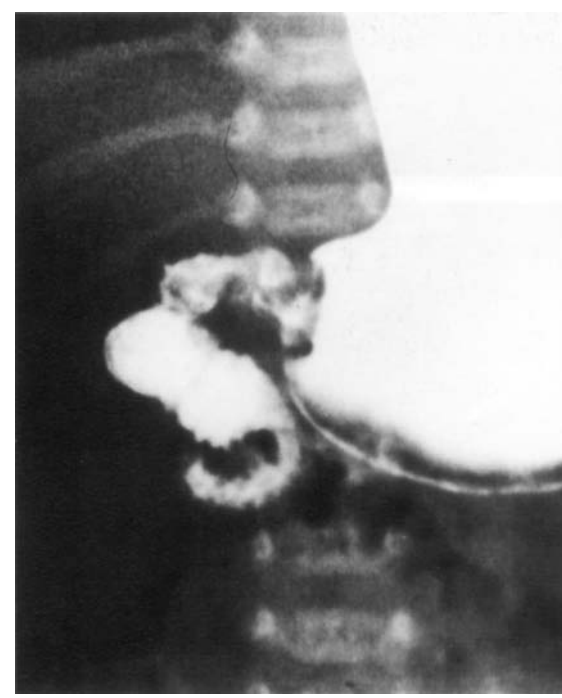

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Figure 9.16 Volvulus. (A) Mesentery and superior mesenteric vein are twisted around the superior mesenteric artery, which is seen in cross-section at the centre of the film. (B) Barium meal shows corkscrewing of the duodenum away from the midline, consistent with a malrotation and volvulus. (By kind permission of Dr Delia Martinez, Leeds.)

Gastro-oesophageal reflux

Reflux through the gastro-oesophageal hiatus is a common problem associated with neonatal vomiting, leading to oesophagitis. The diagnosis is usually made with a contrast meal, PH probe and isotope milk scan. Reflux can be observed on ultrasound as the retrograde flow of stomach contents through the hiatus and up the oesophagus.

The normal intra-abdominal segment of the oesophagus can be demonstrated through the left lobe of the liver, is usually between 2 and 3 cm long, and makes an acute angle with the gastric wall. When episodes of reflux are seen over three times in 10 minutes, this is said to be pathological.

Appendicitis

Ultrasound is the first line of investigation for the child presenting with acute abdominal pain, where the diagnosis is uncertain following clinical assessment. The position of the appendix in small children may vary—pointing upwards, downwards or to the patient's left—making the clinical diagnosis difficult, as the pain is not always confined to the right lower quadrant. Ultrasound is particularly useful in establishing the diagnosis of acute appendicitis and in diagnosing other possible causes of acute abdominal pain, such as gynaecological disorders.[35,36] It is always good practice to perform a full abdominal survey when the clinical presentation is indeterminate.

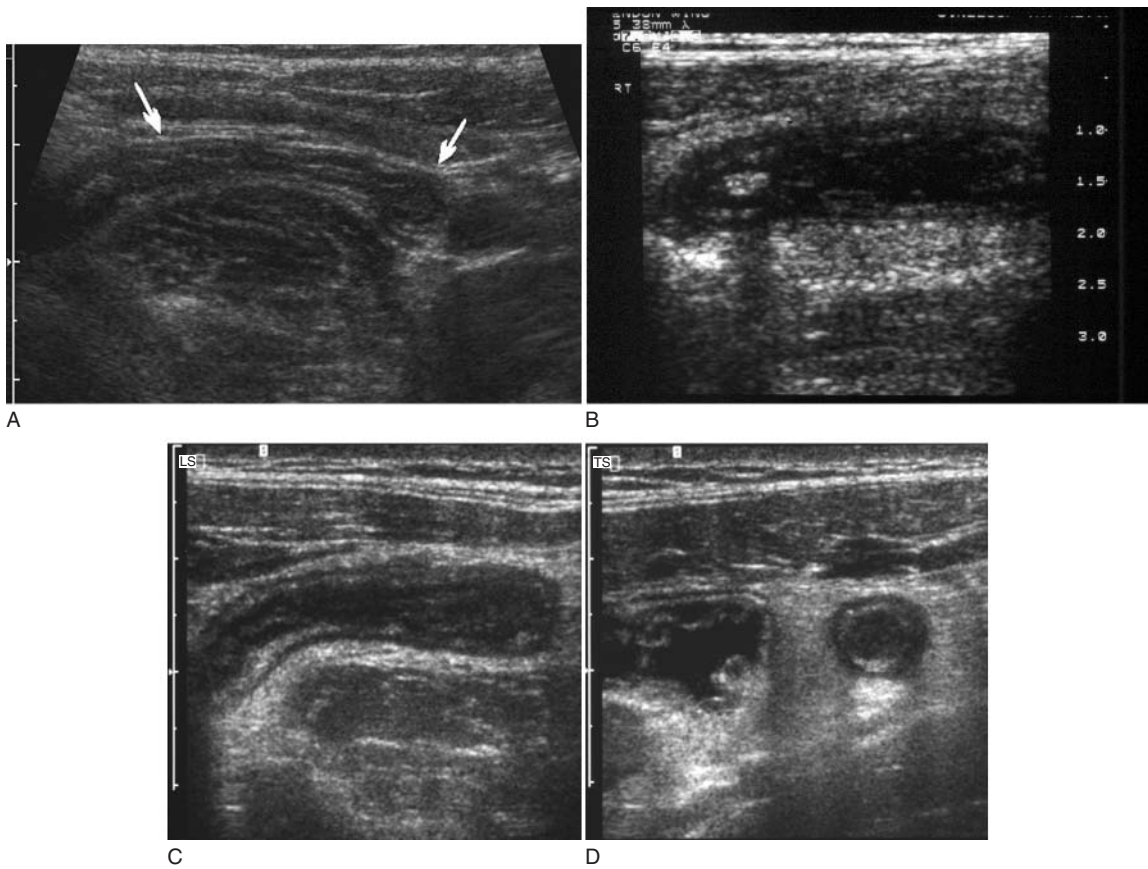
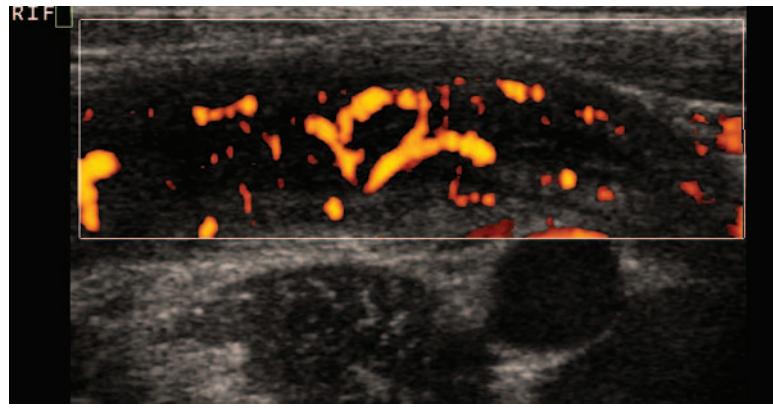


Figure 9.17 Appendicitis. (A) The normal appendix (arrows) lying transversely across the psoas muscle. (B) Longitudinal scan through dilated inflamed appendix containing appendicolith. (C) Dilated thick-walled appendix seen in LS. (D) Same patient as (C) where appendix is seen in cross-section surrounded by echogenic oedematous mesentery, with dilated fluid-filled caecum seen just laterally.

(Continued)



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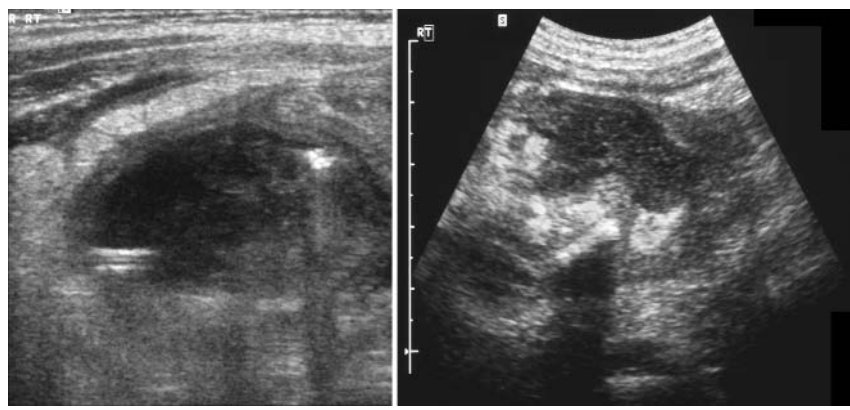


F G

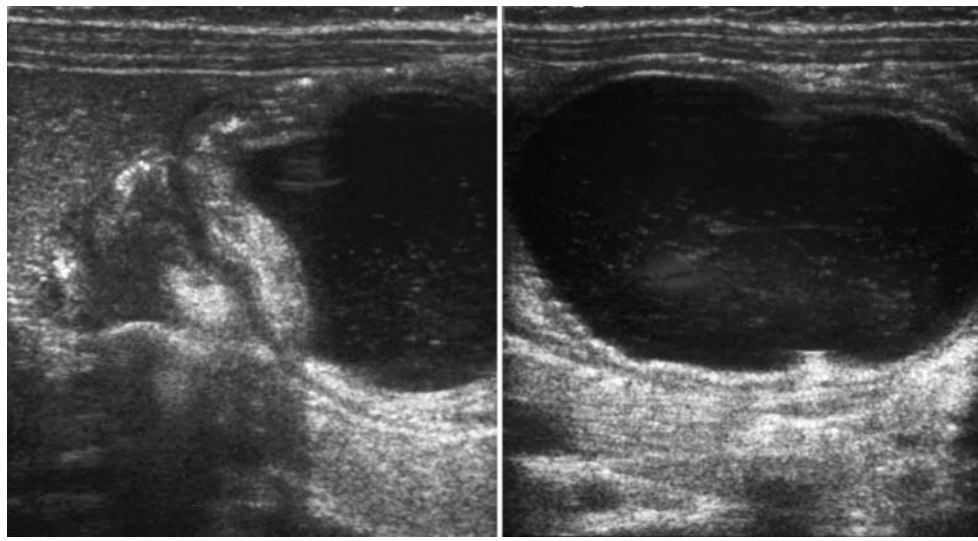
Figure 9.17 cont'd (E) Increased vascularity of inflamed appendix seen on colour flow Doppler imaging. (F) Walled off appendix abscess containing fluid and gas. (G) Complex inflammatory mass containing appendicolith.

Ultrasound demonstrates a hypoechoic, thickened appendix, > 6 mm thick, with a blind end (Fig. 9.17). Occasionally an appendicolith, with strong acoustic shadowing, is present. The inflamed appendix is not compressible on gentle, graded compression with the transducer. This should be done *very* carefully, and released very slowly to avoid rebound tenderness. Ultrasound cannot reliably exclude appendicitis, especially if the appendix is retrocaecal.

Perforation may not be easy to see with ultrasound, as fluid may disperse through the abdomen with decompression of the appendix itself. However, a frank periappendiceal fluid collection or abscess is easily demonstrable in a proportion of children and may, in some cases, be treated conservatively with antibiotics or drained percutaneously prior to surgery. The presence of free fluid, particularly if clear, in the abdomen is a non-specific finding and is not a reliable indicator of an acute abdomen. If echogenic fluid is seen, this is suggestive of intraperitoneal infection in the child with acute abdominal pain, but may be seen in other conditions, for example rupture of a haemorrhagic ovarian cyst. If ultrasound is equivocal, the clinicians may decide to observe the child but further imaging with CT scanning can be helpful in a few selective cases. Alternatively a laparoscopic examination may be performed where there is significant clinical concern[37].

Enteric duplication cysts

These comparatively rare lesions present in infancy or early childhood with nausea, gastrointestinal bleeding, intestinal obstruction and, occasionally, a palpable mass. Most are intra-abdominal but oesophageal



A B

Figure 9.18 (A) Duplication cyst with thickened wall adjacent to bowel. (B) Typical 'double' wall seen in enteric duplication cysts.

Table 9.6 Abdominal fluid-filled masses in paediatrics—differential diagnoses

Choledochal cyst
Mesenteric cyst
Duplication cyst
Hepatic cyst
Pancreatic pseudocyst
Epidermoid cyst of the spleen
Lymphangioma
Ovarian cyst
Encysted fluid associated with ventriculoperitoneal shunt tubing
Renal cyst or renal dilatation
Cystic renal tumour

duplication cysts cause a thoracic lesion with respiratory symptoms. Multiple cysts may be present.

The fluid-filled lesion may demonstrate a spectrum of ultrasonic appearances, from anechoic to hyperechoic, sometimes with gravity-dependent debris or blood.[38]

The wall is well defined and a hyperechoic inner rim of mucosa may be identified in some cases of intestinal duplication (Fig. 9.18). The cyst is closely related to the adjacent bowel and this can be appreciated on real-time scanning as the bowel peristalses. CT and MRI rarely add anything to the ultrasound information. Contrast radiography may show an extrinsic defect but communication with the cyst is rare.

There are many causes of intra-abdominal cystic masses in children. (Table 10.6). The main differential diagnosis in the infant girl is from an ovarian cyst as the ovary is generally an intra-abdominal organ at this age. Useful indicators of an ovarian origin can be detected on careful sonography, by detecting some residual ovarian tissue in the cyst wall, and the finding of a clearly seen multifollicular ovary on one side with absent visualization of a definite ovary on the other side.

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Chapter 10

The acute abdomen

CHAPTER CONTENTS

Ultrasound has an increasingly important role in the initial evaluation of the acute abdomen. Many trauma centres recognize the value of ultrasound as a first-line investigation in properly trained hands. Small portable scanners now offer bedside—even roadside—assessment that can speed the triage process, whereas higher-specification scanners enable the experienced operator to diagnose detailed pathology in the acute abdomen. CT also has an increasing role in this situation. It is readily available in most centres and is proven to be highly accurate. But CT is static, takes longer to arrange and perform and is not always possible, particularly in acutely ill and unstable patients.

There is little doubt that the accuracy of the ultrasound scan is directly attributable to the skill and experience of the operator.[1] For instance, a detailed knowledge of the anatomy, and therefore potential communications, of the peritoneal and retroperitoneal fascial spaces is essential in order to understand the significance and likely origin of an abdominal fluid collection. A left iliac fluid collection may simply be due to local causes such as a diverticular abscess, but could be the result of fluid tracking from a leaking aortic aneurysm or an acutely inflamed or ruptured pancreas.

One other significant advantage of ultrasound is that it is usually an 'interactive' process. In the acute setting, the simple question 'Where does it hurt?' will frequently direct the operator to the underlying pathology, for example in acute bowel inflammation or acute cholecystitis. Clinical signs, such as *erythema ab igne*, which results from pain relief by the patient applying a hot water bottle to

the symptomatic area, may also help to focus the examination. The operator should be alert to potential clues and be prepared to step outside standard scanning protocols, adapting to the many possible presentations of trauma or other acute abdominal conditions.

Although many of the following conditions are dealt with in other relevant chapters, together with details of the respective ultrasound appearances, there are issues that are specific to the patient who presents acutely.

TRAUMA

Blunt or penetrating trauma to the torso, frequently due to a road traffic accident (RTA) or other forms of accident or violence, is a frequent cause of referral to most accident and emergency departments, and forms the main indication for trauma ultrasound. Internal organ injury as a result of trauma is extremely difficult to assess clinically, especially as many patients are admitted unconscious or in a highly unstable condition. Such trauma patients may require emergency laparotomy and ultrasound has been shown to be an invaluable tool in the triage process.[2,3] This may be accompanied by CT, which has the advantage of being able to recognize other injuries which may be present, such as bony, spinal or retroperitoneal trauma which may or may not be accessible to ultrasound investigation.

A system of scanning known as FAST (focused assessment with sonography for trauma) has recently become widely adopted in trauma centres. This system depends upon the proper training of appropriate personnel, and a number of standardized training and accreditation programmes have been devised, notably by the American College of Emergency Physicians.[4] FAST scanning involves a minimum four-view examination, principally to detect the presence of fluid which may result from the rupture of internal organs. The four-view scan should include the right and left flanks (for hepatorenal space, perisplenic regions and spaces above and below the diaphragm), the subcostal region (to include the pericardial space) and the pelvis (retrovesical and retrouterine spaces).[5]

Free fluid is associated with numerous types of injury, which may be detected on ultrasound with varying success. These include rupture of the liver, spleen, kidney, pancreas or bowel (Fig. 10.1). A notable limitation of sonography in the trauma situation is in detecting free fluid in the pelvis, as the bladder is frequently empty or underfilled, and the use of the Trendelenburg position, if possible, helps to reduce the number of false-negative results in this respect by allowing any free fluid to collect in the pelvis under the influence of gravity. Ultrasound is more successful in detecting free fluid than in detecting organ injury directly.[6,7] One study reported a 98% sensitivity for detection of fluid, but only 41% of organ injuries could be demonstrated.[8] However, most of the published studies have concentrated only on the presence or absence of free fluid, rather than the comprehensive assessment of the abdomen by suitably qualified sonographers. The presence of free fluid on ultrasound in a trauma situation therefore infers organ injury requiring careful ultrasonic assessment, further investigation with CT or direct referral for surgery depending on the state of the patient.

Direct visualization of organ rupture is difficult unless a haematoma or other collection is seen. Laceration or contusion may be demonstrated in the liver, kidneys or spleen, but less easily in the pancreas and very infrequently in the bowel. A subtle change in texture may be observed by the experienced operator, or a fine, high-reflectivity linear band representing an organ tear. A delayed scan may demonstrate more obvious organ injury than that apparent on an immediate post-trauma examination. Small visceral lacerations not visible on ultrasound may become apparent when imaged with CT. In particular, pancreatic damage (often due to the sudden pressure of a seat belt across the abdomen during road accidents) may not be obvious immediately post-trauma on either ultrasound or CT.[9] Damage to the pancreatic duct (Fig. 10.1E) causes leakage of pancreatic fluid into the abdominal cavity, resulting in pancreatitis and possible pseudocyst formation or peritonitis.

Free fluid may be present as the result of vessel, rather than organ, rupture. A reduction or loss of blood flow to all or part of the relevant organ, for example the kidney, may be demonstrated using colour and power Doppler ultrasound. The finding of free fluid in women should prompt a detailed

scan of the pelvis where possible. Gynaecological masses may rupture or haemorrhage, presenting acutely, and in women of childbearing age, ectopic pregnancy should be included in the list of differential diagnoses.

When visceral trauma is treated conservatively, follow-up ultrasound may be used to monitor the resolution of any fluid collections or haematoma.

GASTROINTESTINAL TRACT

Most acute presentations of gastrointestinal tract pathology are due to obstruction or inflammation, and the ultrasound appearances of these conditions are discussed more fully in Chapter 8. Appendicitis, and its possible complications, is one of the most common reasons for referral (Fig. 10.2). Ultrasound

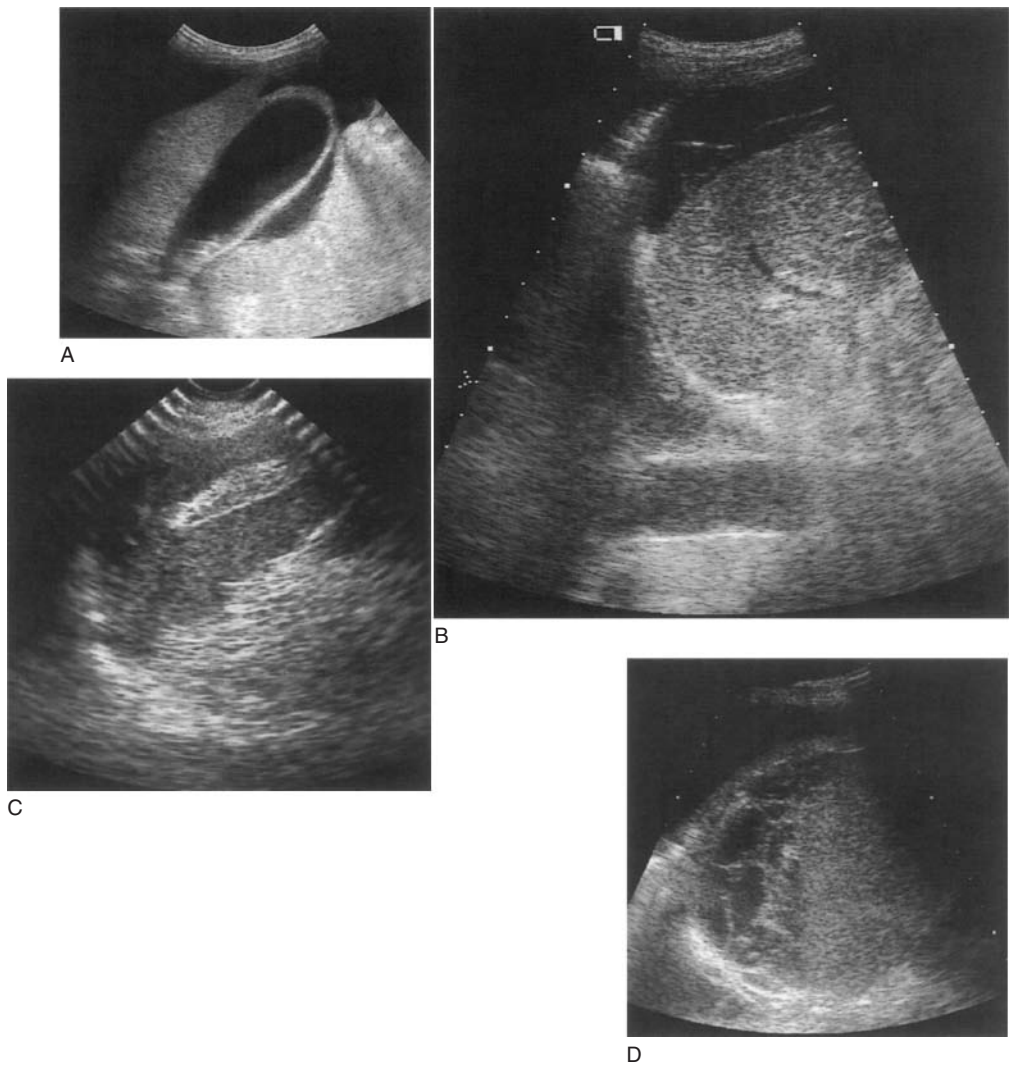

Figure 10.1 (A) The presence of free fluid in a trauma patient implies organ injury, even if this cannot be successfully demonstrated on ultrasound. CT on this patient demonstrated perforation of the bowel. (B) A patient who has been stabbed on the right side has injury to the liver causing a subcapsular haematoma. Blood is also present in the right chest. (C) Laceration of the spleen following a road traffic accident. Free fluid was also present in the abdomen. (D) Splenic lacerations are more obvious several hours after injury. This large splenic haematoma resolved following conservative treatment.

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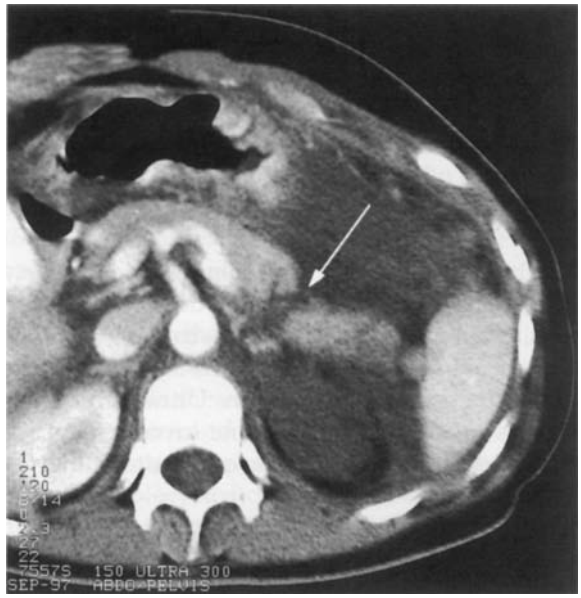

E

Figure 10.1 cont'd (E) CT demonstrating pancreatic fracture (arrow) in the tail of the pancreas following a road traffic accident. Ultrasound was not able to demonstrate the fracture but did demonstrate free fluid following the accident and also diagnosed devascularization of the left kidney (no Doppler flow within the kidney) following a severed left renal artery. This is also confirmed on CT.

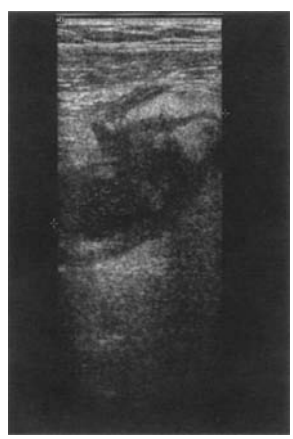

Figure 10.2 Appendicitis abscess.

has a high sensitivity for acute appendicitis, particularly in children.

Although the detailed assessment of the primary gastrointestinal pathology usually requires evaluation by an experienced operator with a high-frequency linear probe, many useful indicators can be found with the basic curvilinear or sector abdominal scan. The presence of fluid-filled bowel segments, which may also show 'overactive' peristalsis, should alert the operator to the possibility of acute intestinal obstruction. Such segments frequently lie proximal to the obstructing lesion, and so the point at which they appear to end should be the subject of detailed examination. Ultrasound is highly accurate in demonstrating obstruction. However, it is less successful in finding its cause and contrast CT or other bowel studies are usually undertaken when obstruction is diagnosed. With both intestinal obstruction and focal pain it may be necessary to examine the hernial orifices. A small but symptomatic epigastric hernia often goes unnoticed unless a detailed scan of the abdominal wall is performed.

Fluid collections such as abscesses may also point to the diseased segment, for example in Crohn's disease or acute diverticulitis. Such inflammatory bowel conditions may well present with an established history which helps the operator to focus the ultrasound examination accordingly.

Perforation of an abdominal viscus can produce small amounts of ascites. This is usually 'mucky', i.e. containing particulate or gas bubble echoes, and may be localized close to the perforation site, around the duodenum or within the lesser sac. Although gas is usually regarded as an obstacle to ultrasound diagnosis, recent studies have shown that specific patterns of gas echoes can make ultrasound more sensitive than plain radiography in the diagnosis of pneumoperitoneum.[10]

HEPATOBILIARY EMERGENCIES

Ultrasound scanning is invariably the first-line investigation for suspected biliary tract emergencies. These include inflammatory conditions causing right upper quadrant and epigastric pain, mostly acute cholecystitis or gallstone pancreatitis, and the various causes of obstructive jaundice (Fig. 10.3). If possible, interventional treatment should be delayed until a detailed imaging assessment of the cause of biliary obstruction has been made, since the presence of a biliary stent can compromise subsequent imaging by CT, MRI or endoscopic ultrasound. Similarly, biliary stents

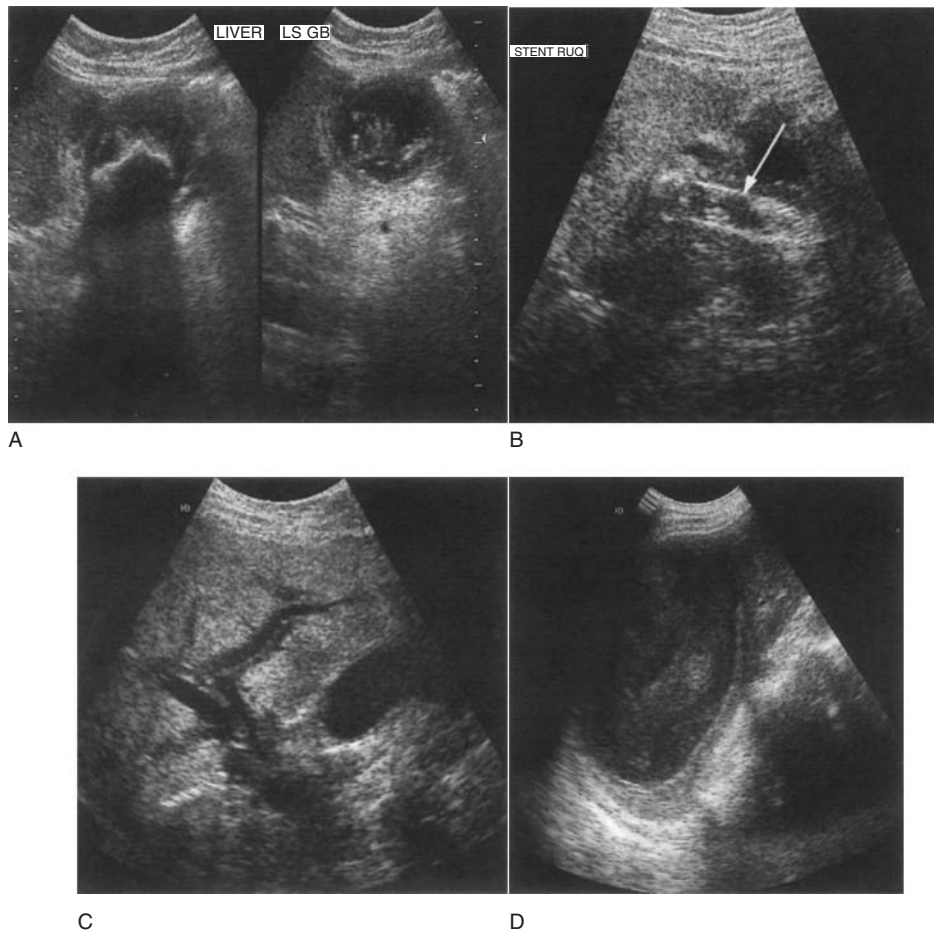

Figure 10.3 (A) An acutely tender, inflamed gallbladder containing stones and debris. (B) A patient with known carcinoma of the head of the pancreas has presented acutely with obstructive jaundice. Her stent (arrow) is blocked, causing intrahepatic biliary duct dilatation with cholangitis (C). (D) Large liver abscess in an acutely ill patient.

frequently cause bile duct wall thickening and may introduce gas into the biliary tree. These will prevent the diagnosis of cholangitis or ductal calculi with ultrasound, and may impede detailed Doppler investigation of, for example, the portal vein. If urgent biliary drainage is required, particularly when the bile is infected, this can quickly be effected by endoscopic stent placement or sphincterotomy.[11] These less invasive methods are replacing surgery as the treatment of choice in this situation, having a lower mortality rate. Endoscopic sphincterotomy and stone extraction have been found to be preferable to surgery, particularly in cases of severe gallstone pancreatitis where patients may be poor operative risks[12] and in cases of stone-related cholangitis.

Ultrasound-guided bed-side cholecystostomy may also be useful in high-risk patients with infected gallbladders and is an effective treatment for acalculous cholecystitis brought on by prolonged postoperative fasting.

The liver itself may be acutely tender in systemic venous congestion due to cardiac failure, acute hepatitis, or the presence of an intrahepatic abscess. The management of liver abscesses is determined by their size, number and cause. Ultrasound is used to guide diagnostic aspiration and drainage procedures, and most types of hepatic

abscess can be treated successfully using these techniques combined with appropriate antibiotic therapy.

THE ACUTE PANCREAS

(See also Chapter 5.) Most cases of acute pancreatitis are suspected clinically, with raised amylase levels and often a history of recurrent epigastric pain pointing to the diagnosis of acute pancreatitis (Fig. 10.4). Although pancreatitis may be due to abdominal trauma, it is more frequently due to gallstone obstruction or alcohol abuse. The pancreas often appears normal even when acutely inflamed, so ultrasound examination should focus on the possible causes (such as gallstones, biliary dilatation or evidence of alcoholic liver disease) and complications (pseudocysts, portal or splenic vein thrombosis). Many pancreatic pseudocysts are now managed successfully by endoscopic ultrasound-guided transgastric drainage.[13]

RENAL TRACT EMERGENCIES

(*See also Chapter 7.*) Ultrasound is usually the first-line investigation in the assessment of acute loin pain, which in the absence of trauma is commonly due to acute urinary tract obstruction and/or renal infection (Fig. 10.5). Less common acute presentations include renal vein thrombosis or spontaneous haemorrhage, usually from a renal tumour or cyst.

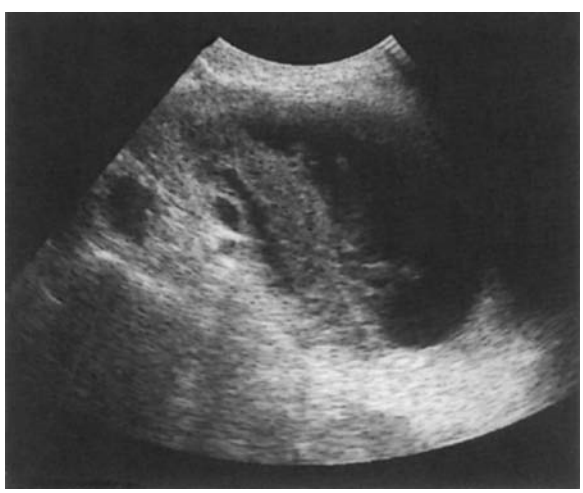

Figure 10.4 Pancreatitis with a large pseudocyst. The patient was acutely tender, and the cyst was drained under ultrasound guidance.

Until recently, ultrasound and/or intravenous urography (IVU) have been the investigations of choice in acute renal colic due to suspected ureteric calculus, and in most UK centres the IVU is currently the method of choice for demonstrating ureteric obstruction (Fig. 10.5E). Low-dose unenhanced multislice CT is increasingly being recommended as a replacement for these two modalities,[14] but even with this technique diagnostic pitfalls exist.[15] Abdominal ultrasound with or without plain radiography may still provide comparable accuracy where CT resources are limited.[16,17]

The main limitation of ultrasound in acute ureteric obstruction is that obstruction may be present in the early stages without collecting system dilatation. But the minimally dilated renal pelvis, which would normally be dismissed as unremarkable in a patient with a full bladder, should raise the operator's suspicion in the patient with acute loin pain. Doppler ultrasound of the kidneys shows a higher resistance index in the obstructed kidney than in the normal side.[18] Upper tract obstruction can be relieved via cystoscopy-guided ureteric stent placement. Ultrasound-guided percutaneous nephrostomy may be required if this is not practicable, or if there is evidence of infection.

Renal infection with parenchymal involvement (acute pyelonephritis) may be the cause of severe acute loin pain with fever, but ultrasound examination mostly shows no abnormality. Occasionally the skilled operator using high-specification equipment may be able to identify segmental areas of high reflectivity, showing *decreased* blood flow with power Doppler. The diagnosis of this condition is usually based on clinical criteria, but these segments can be demonstrated with CT if necessary.

OTHER RETROPERITONEAL EMERGENCIES

(See also Chapter 8.) Ultrasound has an established role in identifying the presence of an abdominal aortic aneurysm, but should not be used to assess subacute leakage or rupture. However, where rupture is suspected, and no previous imaging results

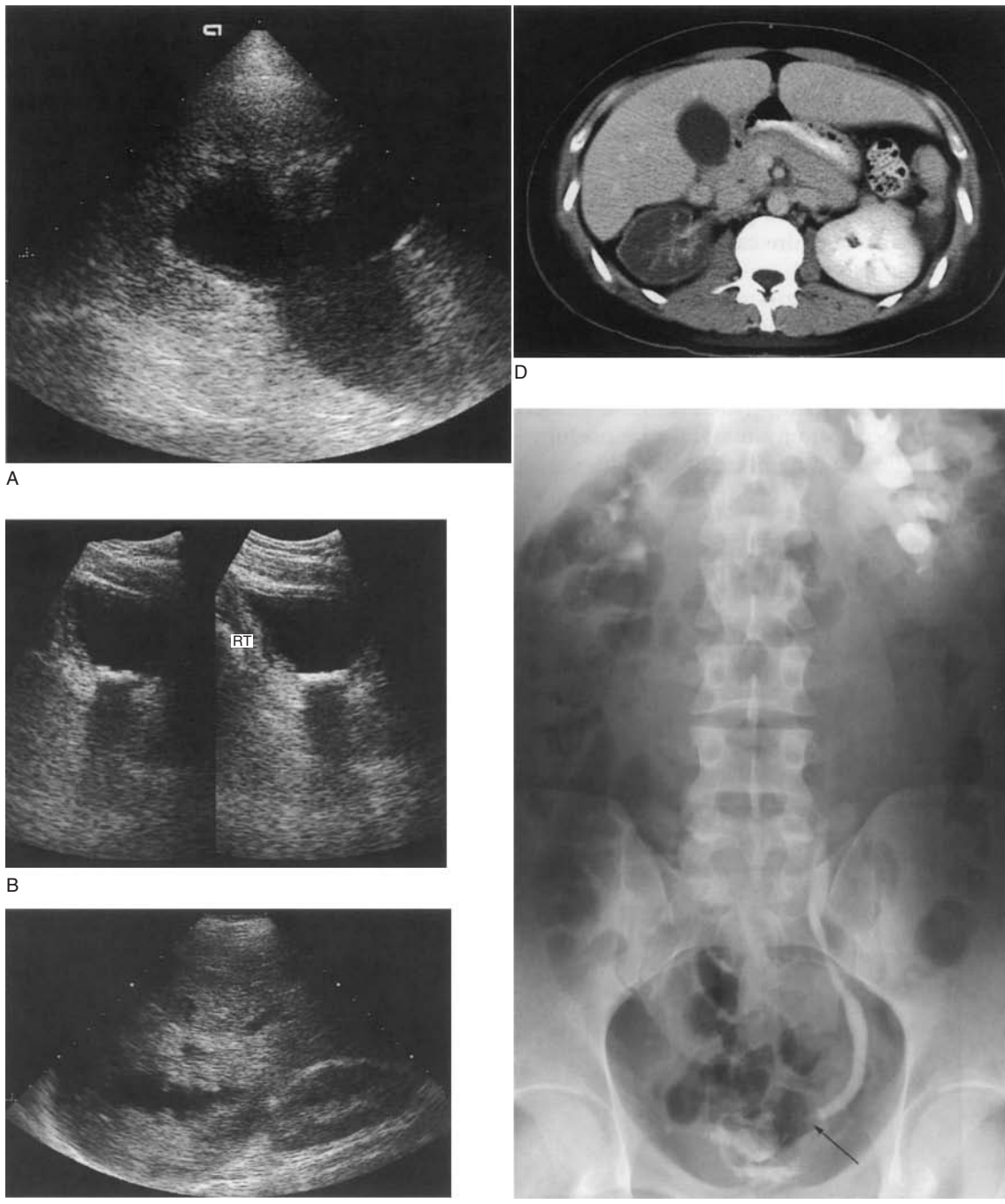

Figure 10.5 (A) Obstructed kidney with pelvicalyceal system (PCS) dilatation and a dilated upper ureter. (B) Mobile stones were demonstrated in the bladder, but the level of obstruction in the ureter could not be positively identified. Intravenous urogram (IVU) confirmed a stone in the ureter. (C) Severe laceration to the liver following a road traffic accident. (D) The same patient's CT scan confirms the liver injury and demonstrates an avascular right kidney (compared with the normal left kidney (LK)) due to laceration of the renal vessels. (E) Acute renal colic. IVU demonstrates a left hydronephrosis with a stone in the lower ureter. (This area is not usually demonstrable with ultrasound.)

are available, ultrasound can be a time-saving triage tool to exclude an aneurysm from the differential diagnosis of abdominal pain. Suitably trained emergency department clinical staff can perform this quickly and successfully.[19] Rupture of an aortic aneurysm is a catastrophic event, and although an urgent contrast-enhanced CT can be helpful, emergency surgery based on clinical findings should not be delayed by imaging investigations.

Ultrasound is also the first investigation of choice for demonstrating suspected psoas abscess or haematoma[20] (Fig. 10.6).

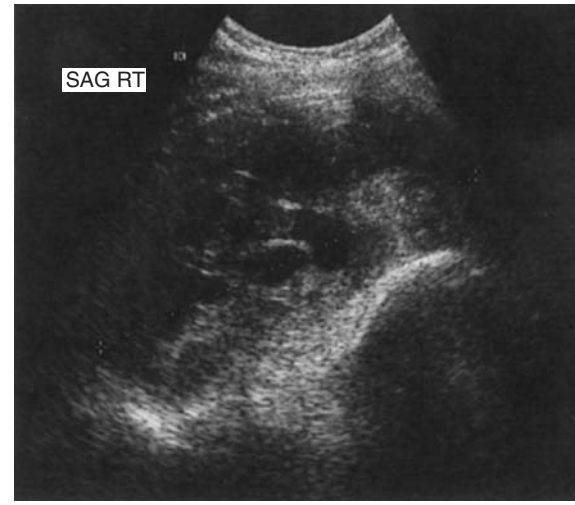

Figure 10.6 A large, right-sided psoas haematoma.

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Chapter 11

Interventional and other techniques

CHAPTER CONTENTS

The development of ultrasound-guided minimally invasive procedures, incorporating diagnostic biopsy, therapeutic drainage and treatment techniques, has developed significantly over the last decade and is now accepted practice, in many circumstances being used in isolation as a definitive treatment, and in others in conjunction with other radiological or surgical options. These minimally invasive methods are advantageous, with lower patient mortality and morbidity, increased patient acceptability, and are economically beneficial.

The relative speed and ease with which these procedures can be carried out have resulted in a reduction of the diagnostic laparotomy and more prompt and appropriate patient treatment. Whilst both ultrasound and CT may be used for many of these procedures, in general, ultrasound is often the first-line method as it is effective in the vast majority, generally more accessible, and does not carry a radiation risk. Clearly, the choice of technique will depend upon the experience of the individual, machine availability and the site and depth of the lesion.

ULTRASOUND-GUIDED BIOPSY: GENERAL CONSIDERATIONS

Percutaneous biopsy of organs, masses or focal visceral lesions is an integral part of the diagnostic process for a large number of patients. Although changes on ultrasound may confirm the suspected clinical suspicion, that is, a bright liver may indicate fatty change, a nodular liver may suggest cirrhosis or enlarged kidneys of increased echogenicity may

suggest glomerulonephritis, imaging alone is not enough and a definitive histological diagnosis is required. The advantages of using ultrasound to guide such procedures are numerous:

- The needle tip is directed, in real time, along the biopsy path and visualized within the lesion.
- Greater precision is obtained; needle guidance is essential for all small lesions and lesions at depth.
- Fewer needle passes are required to obtain the desired result.
- The best route can be utilized and vital structures, such as blood vessels, avoided.
- Postprocedure complications, such as haematoma, are minimized.
- Confidence in the biopsy result, particularly a negative one, is increased due to direct visualization of the needle tip in the lesion.
- All the advantages of ultrasound over other imaging methods apply (quick, direct vision, no radiation hazard, low cost). The limitations due to bone and air-filled structures also apply.
- The capability to perform bedside procedures for critically ill patients and to use in conjunction with other imaging techniques, for example fluoroscopy, is advantageous.

With ultrasound the biopsy procedure is quick, safe and accurate and is therefore acceptable to the patient. There are several accepted methods of performing a guided biopsy, but certain generic rules are common to the procedure, regardless of the organ under investigation:

- A written request form from a medical practitioner with the results of any previous investigations should be available. The reason for biopsy should be appropriate.
- Assessment of blood clotting status. Normally the prothrombin time should be within 3 s of the control, platelet count > 75 000/ml and international normalized ratio (INR) < 1.3.
- Identification of possible contraindications to biopsy, for example an uncooperative patient, coagulopathy.
- Careful explanation of the procedure to the patient, including risks and benefits.
- Informed, written consent for the procedure.
- Procedure should be performed in a quiet and clean environment. Appropriate measures should be taken to preserve pre-, peri- and postprocedure sterility.
- A prebiopsy scan to identify a suitable biopsy route avoiding vital structures.
- Satisfactory care of the patient both during and after the biopsy procedure with relevant observations of vital signs. A pulse oximeter and appropriate nurse cover are now recommended.
- Appropriate preparation of the specimen.
- Contraindications are relative and include the biopsy pathway, an uncooperative patient and uncorrectable coagulation and should be assessed on an individual basis.

Analgesia

For the vast majority of biopsy procedures local anaesthetic is administered following localization of the biopsy site on ultrasound. Either 1% or 2% lidocaine (lignocaine) is commonly used; the volume will depend upon patient build, depth of lesion and patient anxiety. Normally a short period of time, commonly 4–5 min, is allowed to pass so that the anaesthetic can work, after which a small scalpel incision is made in the skin to facilitate the biopsy needle's introduction, with little or no discomfort to the patient.

In cases of, for example, simple aspiration with a 22-gauge needle or smaller, local anaesthetic is normally unnecessary.

Patients who are particularly apprehensive may require preprocedure medication with a sedative such as diazepam or similar anxiolytic agent; however this is not common. Very occasionally intravenous analgesia and/or sedation may be required during the procedure; it is often a good idea to have an intravenous cannula in situ prior to biopsy.

The use of a general anaesthetic for children is common practice, to enable the procedure to be carried out quickly and accurately while the child remains still.

Methods of ultrasound guidance

There are various ways of performing ultrasound-guided procedures: organ/lesion localization ('blind biopsy'), biopsy guide or freehand technique. The choice of method depends upon the procedure in question, equipment and the experience and skill of the operator.

Blind biopsy

With this method a position on the skin surface is marked overlying the organ or lesion to be biopsied, using ultrasound to localize. This remains acceptable for diffuse disease, when only a representative sample of liver tissue is required. Nevertheless, it is good practice even in these situations to visualize the needle during the procedure, and this method of biopsy is now used less frequently.

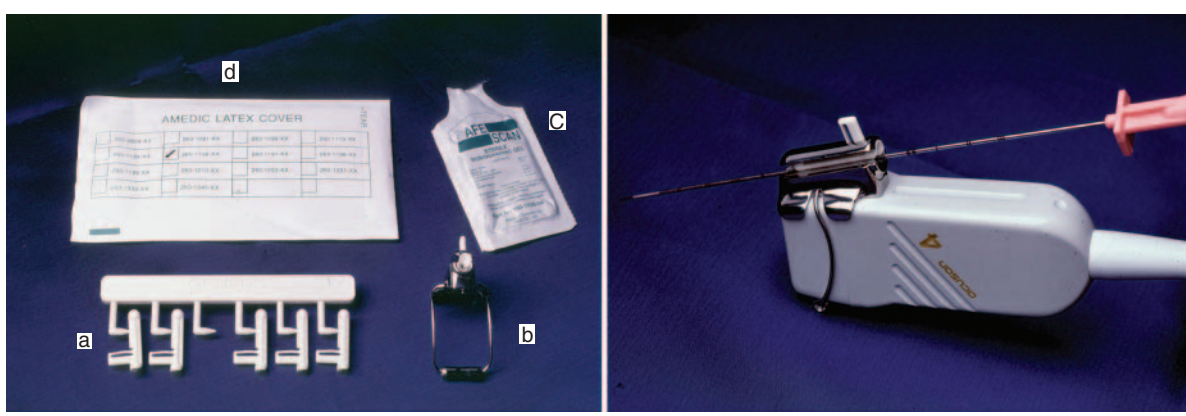
Biopsy guidance

Most manufacturers provide a biopsy guide which fits snugly on to the transducer head and provides a rigid pathway for the needle (Fig. 11.1). These are now the commonest and preferred method of biopsy. Previously adjustable angle biopsy guides were available; however these offered no specific advantages and were prone to user error. The fixed biopsy guides contain a groove for a series of plastic inserts ranging from 14G to 22G size, depending on the size of the biopsy needle. It is often preferred to use one size greater than the needle, that is a 16G insert for an 18G needle, as the needle tends to move more freely. These guides are sterilized and fitted on to the transducer, which can either be covered by a sterile sheath or thoroughly cleaned with chlorhexidine solution. The use of a sheath is highly recommended, as it maintains the sterility of the procedure, reducing the risk of infection, with no adverse effect on the image.

The needle pathway is displayed on the ultrasound monitor electronically as a line or narrow sector, through which the needle passes. The operator then scans in order to align the electronic pathway along the chosen route, the needle is inserted and the biopsy taken. These attachments should be tested regularly to ensure the needle follows the correct path (Fig. 11.2).

Freehand

A freehand approach, in which the operator scans with one hand and introduces the needle near to the transducer with the other, may be used for larger or more superficial lesions. This technique is commonly used for breast biopsy and biopsy in the head and neck. The needle is inserted from one end of the probe at right angles to the ultrasound beam; generally speaking the angle utilized is shallow in comparison with the fixed guide systems for deeper structures.



A B

Figure 11.1 (A) Necessary component parts to perform an ultrasound-guided biopsy procedure. A series of plastic inserts (A) range in size from 14 to 22G. The appropriate insert is inserted into a fixed biopsy guide (B). The procedure is performed with sterile jelly (C) and a sterile probe cover (D) if required. (B) The assembled biopsy guide.

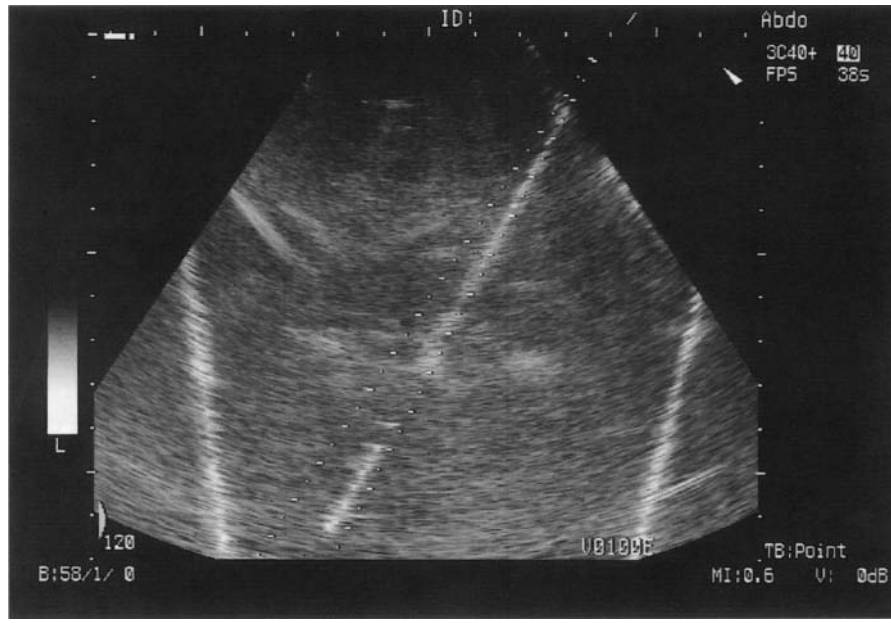


Figure 11.2 Testing the alignment of the biopsy guide. The electronic pathway is activated on the image, and the needle is scanned as it is passed into a jug of water.

Equipment and needles

The core of tissue for histological analysis is obtained with a specially designed needle consisting of an inner needle with a chamber or recess for the tissue sample and an outer, cutting needle which moves over it—the Tru-Cut needle. The biopsy is obtained in two stages: first the inner needle is advanced into the tissue, then the outer cutting sheath is advanced over it and the needle withdrawn containing the required tissue core (Fig. 11.3).

The use of a spring-loaded gun to operate these needles is now commonplace (Fig. 11.4). Such devices are designed to operate the needle with one hand; the whole needle is advanced into the tissue, just in front of the area to be biopsied. By pressing the spring-loaded control, the inner part is rapidly advanced into the lesion, followed rapidly by the cutting sheath over it. These needles can be obtained in a variety of sizes—generally 14, 16, 18 or, less commonly, 20 gauge. Most focal lesions are biopsied with a standard 18G needle. As a general principle, as the needle advances approximately 1.5–2.0 cm during biopsy, it is advisable to position the needle tip on the edge of a lesion to obtain a good histological sample as most lesion necrosis tends to be centrally located.

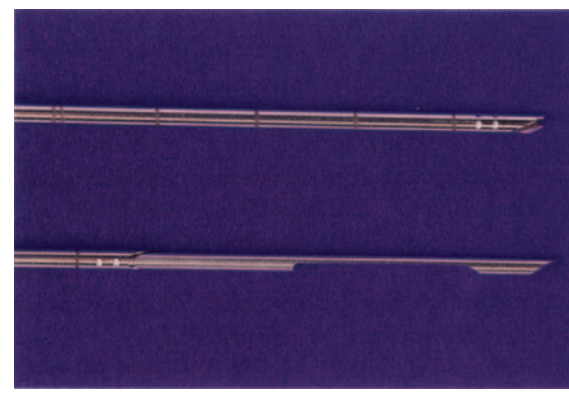


Figure 11.3 Biopsy needle closed (top) and open (bottom).

Such biopsy guns enable the operator to scan with one hand and biopsy with the other, observing the needle within the lesion, yielding a high rate of diagnosis with a single-pass technique[1] and minimizing post-biopsy complications.

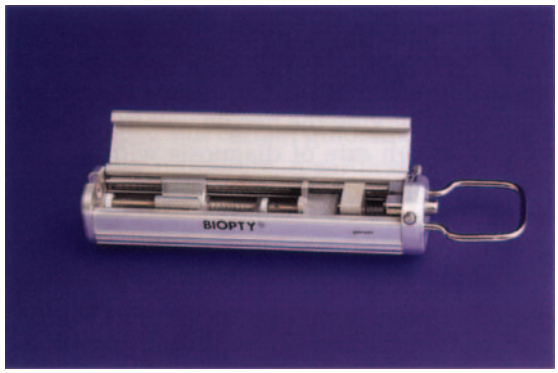


Figure 11.4 Spring-loaded gun designed to operate the cutting needle.

As an alternative to the gun/needle combination a number of 'self-fire' needles are available. This is essentially a single-use spring-loaded biopsy needle. Again these come in a variety of sizes but their advantage is that they are easier and lighter to use than the gun/needle combination, and therefore are easier employed in the CT situation. Most departments will tend to utilize a combination of both.

In cases where the clinician is not familiar with ultrasound techniques, appropriate guidance by a sonographer, while the clinician biopsies, is highly successful, quick and avoids potential complications.

Fine-needle histology, involving the use of needles of 21 gauge or less, reduces even further the possibility of postprocedure complications. These are generally not used as only small amounts of tissue are obtained for analysis and, as thin needles, they are apt to bend more easily, and are therefore more difficult to see and retain within the plane of the scan. Biopsy of deep lesions is therefore more difficult, if not impossible.

Fine-needle aspiration cytology

Cytology is the analysis of cells rather than the core of tissue obtained for histology. This is generally more difficult to interpret pathologically, as the characteristic architecture and intercellular relationships seen in a histological sample are absent. It has the advantage, however, of allowing a finer needle to be used. This can be passed through structures, for example the stomach, blood vessels, en route to the site of interest, with no adverse effects.

Fine needles for cytology are of 21 gauge or smaller. They are of a simple design with a bevelled, hollow core and no cutting mechanism.

The needle is introduced under ultrasound guidance to the required position. Fragments of tissue are removed into the needle by applying negative (sucking) pressure with a syringe to the needle, while moving the needle to and fro to loosen the tissue.

These can then be expelled on to a microscope slide and smeared. The main disadvantage of this technique is that it requires a highly trained and specialized pathologist to interpret the samples, whereas all trained pathologists can view histological specimens. In addition, for many conditions, histological diagnosis is required, although cytology remains a useful tool in the breast and thyroid.

ULTRASOUND-GUIDED BIOPSY PROCEDURES

Liver biopsy

The most common reason for ultrasound-guided biopsy is for suspected metastatic disease. The liver is one of the most common sites for metastases and histology is often required to confirm the diagnosis, or to identify the origin of an unknown primary lesion (Figs 11.5 and 11.6).

Biopsy of other focal lesions in patients with chronic liver disease (for example, cirrhosis, hepatitis B or C) in whom there may be suspected hepatocellular carcinoma and occasionally in patients with benign disease (for example, capillary haemangiomas or focal nodular hyperplasia) can also be performed, although MRI and contrast ultrasound are increasingly used to characterize lesions, without recourse to biopsy.

Focal lesion biopsy is generally safely and accurately performed with an 18G Tru-Cut needle which yields reliable tissue for histological analysis. In general an accuracy of 96% should be achievable[2] (Fig. 11.7).

In addition to focal lesion biopsy another common reason for liver biopsy is to assess the

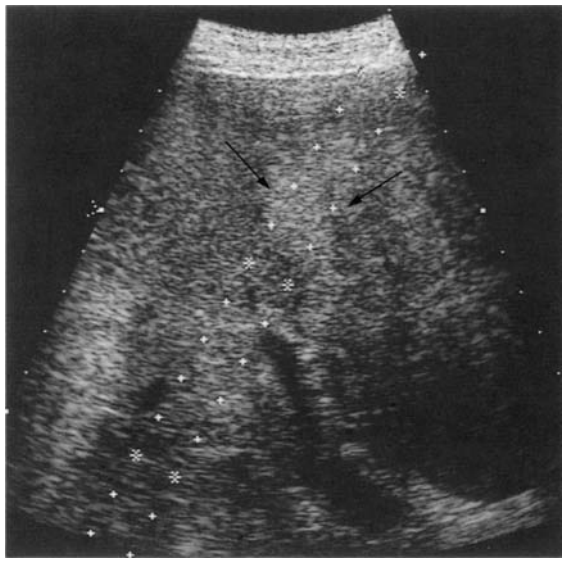


Figure 11.5 In a liver full of metastases, the electronic pathway is lined up on a hyperechoic lesion near the surface (arrows).

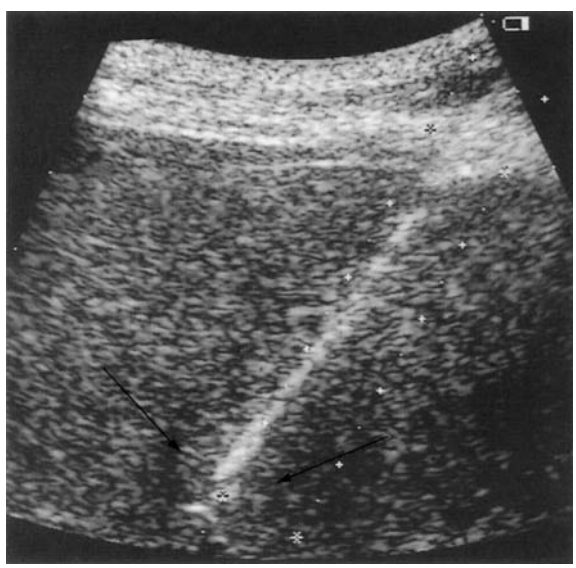


Figure 11.6 The needle is introduced into the liver, just in front of the lesion, and the gun is fired, propelling the needle tip into the chosen lesion (arrows).

presence/absence of parenchymal liver disease, severity of disease and, where appropriate, the aetiology of the disease process. This is often performed in patients with abnormal liver function tests with no evidence of biliary obstruction. The

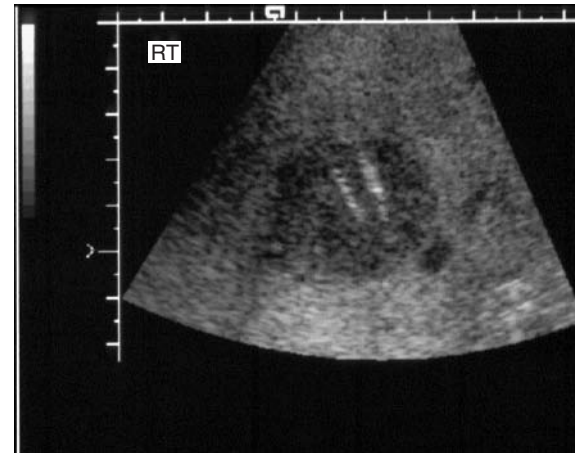


Figure 11.7 A focal liver lesion immediately post-biopsy, two passes. Residual air is noted within the lesion outlining the recent biopsy tracks. This is a very useful appearance and visually confirms that the biopsy has been taken from the correct area.

clinical history and serological analysis can be helpful in determining aetiology; however biopsy is often required. This is normally performed with a 14G or 16G Tru-Cut needle. Very often the liver is simply identified with ultrasound and a suitable mark made on the skin, often in the mid-axillary line, and the biopsy performed through the right lobe. Although this is acceptable for this type of biopsy, as no guidance is required towards a specific focal lesion, ultrasound guidance during the procedure is still preferable to the 'blind' technique in order to avoid large vessels and reduce the subsequent risk of haematoma. Biopsy may also be performed for patients with suspected rejection following hepatic transplantation.

Where coagulation profiles are not correctable (and most generally are), liver biopsy can be performed using a 'plugged' technique or, more commonly, by the transjugular route (Fig. 11.8).

Pancreatic biopsy

The commonest reason for biopsy of the pancreas is in patients presenting with obstructive jaundice due to a mass in the head of the gland. A fine-needle technique enables the mass to be accessed through the stomach and left lobe of liver without

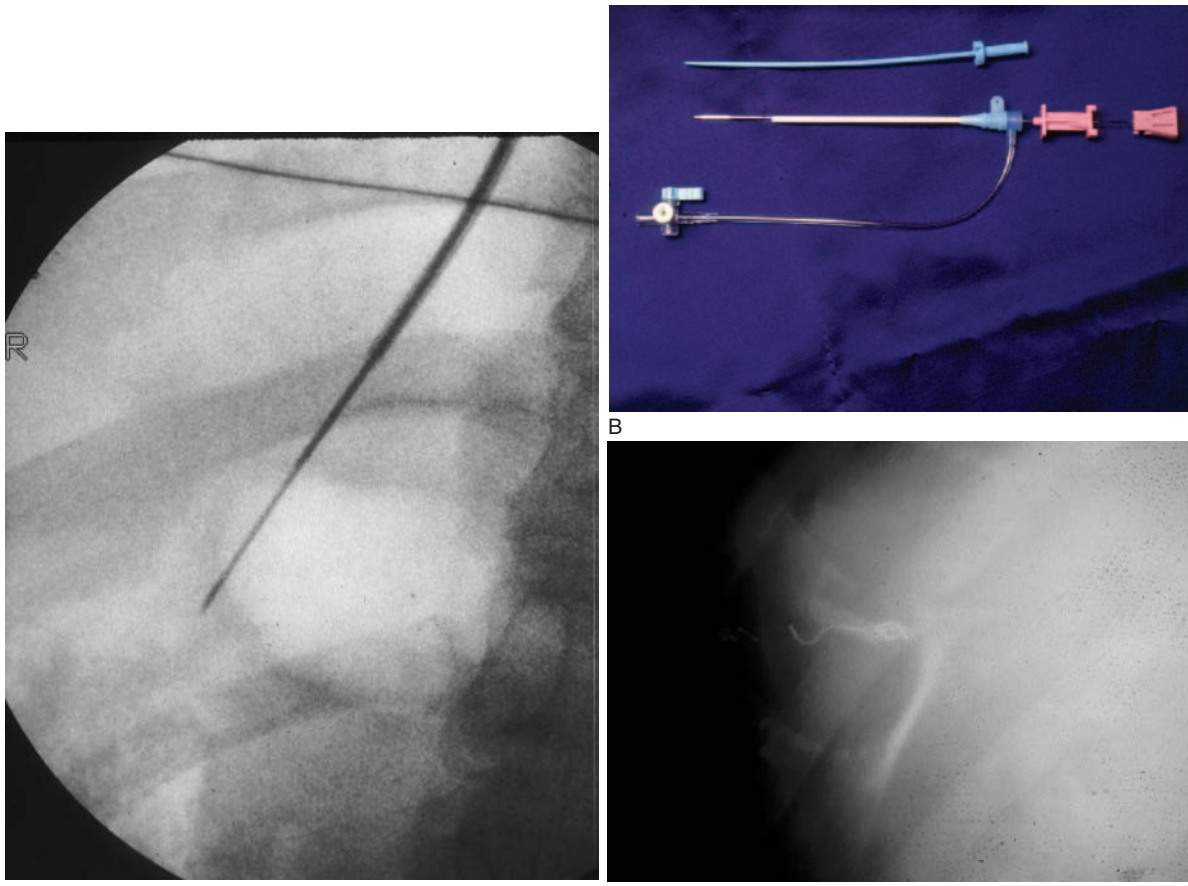


Figure 11.8 (A) Transjugular biopsy of the liver. Access is via the right internal jugular vein, through the right atrium and into the inferior vena cava and hepatic veins. Once the catheter is wedged in the hepatic vein the cutting needle is released and a biopsy is taken. (B) Plugged liver biopsy technique. This is no longer, or only rarely, used. A 4F sheath can accept an 18G biopsy needle and is inserted into the liver. Multiple biopsies can be taken: at the end of the procedure the needle is removed and the biopsy track embolized via the sheath with embolic material, e.g. sterispon and coils. (C) X-ray of the post-embolization track.

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complications. However an 18G needle biopsy is advisable to reduce false-negative results due to the well-known situation of a carcinoma being associated with an element of peripheral inflammation. Pancreatic biopsies are often better performed under CT control (Fig. 11.9), particularly when lesions are small, patients big and/or the lesion is difficult to identify with ultrasound. In those patients with negative biopsies very often interval CT scans are performed to see if the lesion is static or progressive.

Native kidney biopsy

Histology is frequently required in order to direct further management of diffuse renal disease. Biopsy of solid renal masses is rarely performed as the diagnosis of renal cell or transitional cell carcinoma is usually clear from imaging. Biopsies are still performed however in those patients who are not having surgery to confirm the diagnosis; this is often required prior to chemotherapy or new therapeutic regimes. Biopsy of the native kidney is

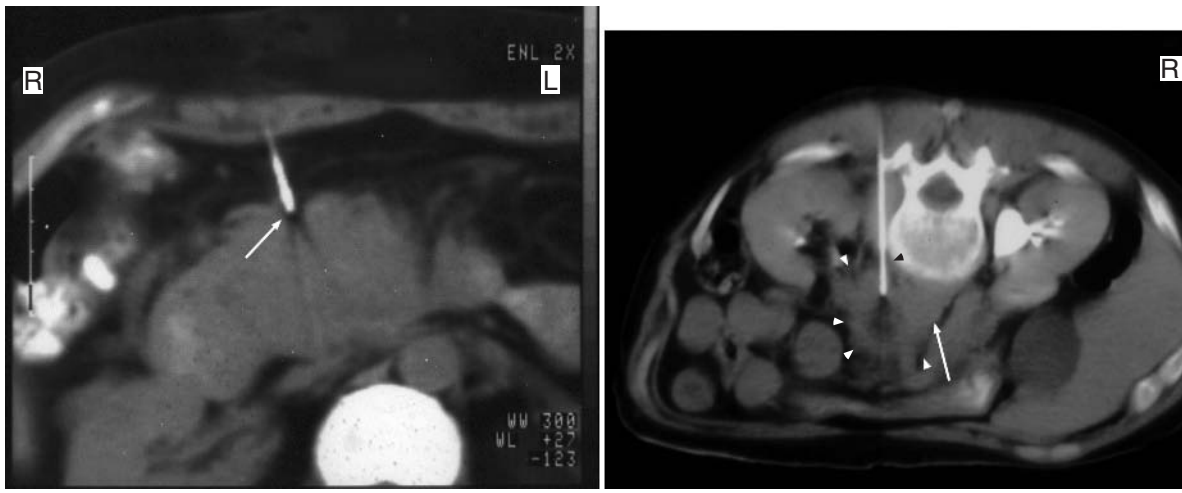

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Figure 11.9 (A) CT-guided biopsy of a pancreatic head mass. The tip of the biopsy needle (arrow) is positioned in the periphery of the lesion so that when the biopsy is taken a good core of tissue is obtained. Note the artifact from the needle tip. (B) CT-guided bioposy of a retroperitoneal lymph node mass (arrowheads). The mass lies adjacent to the aorta (arrow); however this is protected from the needle by the angle of approach and its relationship to the vertebral body. CT is the preferred biopsy method of choice for deep structures within the retroperitoneum.

performed in the majority of centres under ultrasound guidance. Contraindications to biopsy include hydronephrosis, which may be more appropriately treated with catheterization or nephrostomy, or small kidneys, that is < 8 cm longitudinal axis (these appearances being indicative of chronic renal impairment). Kidneys > 9 cm can potentially be biopsied; however other factors, including cortical thickness, age, clinical history and the requirement for definitive diagnosis will all have a bearing on whether biopsy is performed or not. Hydronephrosis and kidney size are easily assessable with a prebiopsy scan.

In most cases the biopsy is performed with the patient prone over a small bolster to maximize access to the kidney. The shortest route, avoiding adjacent structures, is selected; subcostally, traversing the cortex of the lower pole and avoiding the collecting system and major vessels is recommended. With ultrasound guidance, either kidney may be chosen and accessibility will vary between patients. The depth of penetration and angle of approach are carefully assessed. Biopsy is normally with a 16G needle.

The patient's cooperation is required with suspending respiration at the crucial moment. This avoids undue damage to the kidney as the needle is introduced through the capsule. The needle should be positioned just within the capsule prior to biopsy so that the maximum amount of cortical tissue is obtained for analysis, as the throw of the needle may be up to 2 cm.

Renal transplant biopsy

Biopsy is a valuable tool in the postoperative management of the transplant patient (Chapter 7), enabling the cause of graft dysfunction to be identified, in particular differentiating acute tubular necrosis from acute rejection. Ultrasound guidance is essential in order to reduce complications such as haematoma, vascular damage (which may result in arteriovenous fistula or pseudoaneurysm formation) and laceration of the renal collecting system. A single-pass technique, using the spring-loaded biopsy gun with a 16-gauge needle, is usually sufficient for histological purposes; however two passes are often required so that electron microscopy and immunofluorescence can also be perfomed. The procedure is well tolerated by the patient and the complication rate low, at less than 5%.[3]

A full scan of the kidney is first performed to highlight potential problems, for example perirenal fluid collections, and to establish the safest and most effective route. The transplanted kidney lies in an extraperitoneal position and the chosen route should avoid puncturing the peritoneum, to minimize the risk of infection. Unlike the native kidney, the upper pole of the transplanted kidney is usually chosen to avoid major blood vessels and the ureter, which pass close to the lower pole.

The biopsy aims to harvest glomeruli, and the chosen route should therefore target the renal cortex. An angle is chosen to include the maximum thickness of cortex and, where possible, avoid the renal hilum (Fig. 11.10).

Complications of ultrasound-guided biopsy

Postprocedure complications such as haematoma requiring blood transfusion and trauma to adjacent viscera occur very infrequently when ultrasound guidance is used. As expected, the risk of complications is less in fine-needle biopsy than with larger needles;[4] however, there is no significant difference in complication rate between a standard 18G Tru-Cut needle and a 22G Chiba needle.[5] The mortality and major complication rates vary but using a standard 18G needle these are approximately 0.018–0.038% and 0.18–0.187% respectively, mortality being due to haemorrhage in 70%. As a working figure this means the mortality is approximately 1 in 3300–5400 and morbidity 1 in 530 biopsies (Table 11.1).[4,6,7] The risk of haemorrhage is increased in patients with coexistent cirrhosis and is more likely to occur with malignant than benign lesions,[8,9] although large haemangiomas also can carry a significant risk of bleeding.

As with any procedure of this nature, there is a very small risk of infection, which can be minimized by using an aseptic technique.

Tumour seeding of the biopsy tract is an uncommon complication of biopsy and reports of tumour seeding are associated with repeated passes into the mass using large needles. Although much talked about, tumour track seeding is in fact rare, occurring in approximately 1 in 20 000 biopsies.[7,10] The best-known tumours for this are mesothelioma and hepatoma.

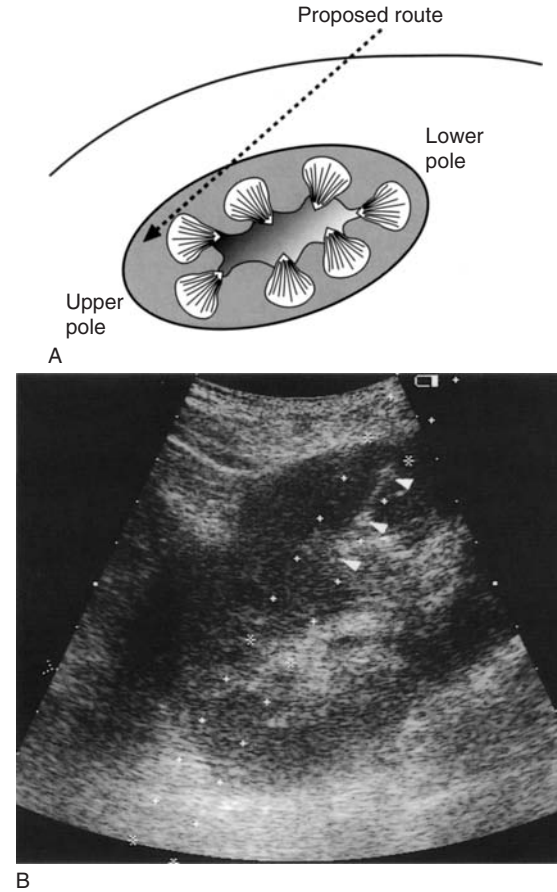


Figure 11.10 (A) The transplanted kidney lies in the iliac fossa and is biopsied with the patient supine. (B) The needle is seen entering a transplanted kidney (arrowheads).

Complications following abdominal biopsy are increased with multiple passes and are at least in part related to the skill and experience of the operator.

If the biopsy result is negative or unexpected then a number of scenarios should be considered and include sampling error, poor histological specimen, sonographic or pathological misinterpretation or indeed a true negative finding. A repeat biopsy is sometimes justified.

ULTRASOUND-GUIDED DRAINAGE

Many fluid collections are the result of surgical intervention and often cannot be differentiated on ultrasound alone. Diagnostic aspiration of

Table 11.1 Complications of ultrasound-guided biopsy

Author	Year	Number of biopsies	Mortality rate	Major complication rate
Fornari et al[4]	1989	10 800	1:5400	1:530
Nolsoe et al[6]	1990	8000	1:2700	1:540
Smith[7]	1991	16 400	1:3300	–

fluid collections is used to establish their exact nature: this may include haematoma, lymphocoele, urinoma, biloma, pseudocysts and others.

Postoperative haematomas are normally treated conservatively and tend to resolve spontaneously. Insertion of a drain into such a collection is at high risk of converting the collection into an abscess.

Abscess drainage

Ultrasound-guided drainage of abscesses is now the preferred treatment when the collection can be visualized on ultrasound and a safe route chosen. These may result from postoperative infection, inflammatory bowel conditions, such as Crohn's disease or appendicitis, or other sources of infection, particularly in immunosuppressed patients. Drains come in different sizes and generally the thicker the pus, the larger the bore of drain that is required. Whilst aspiration is initially performed to confirm the nature of the collection, very often a drain is left in situ; together with appropriate antibiotic therapy this is usually effective. At the very least it normally leads to an improvement in the overall clinical condition to allow definitive treatment and can in itself be a definitive cure.

Ultrasound is particularly useful in cases of hepatic abscesses and in draining the subphrenic, pericolic and subhepatic areas. Superficial collections, usually associated with wound sites, are also readily accessible to ultrasound. Collections obscured by bowel gas are best drained under CT guidance.

Gallbladder drainage

Gallbladder drainage under ultrasound control is a temporary, palliative procedure which tends to be reserved for particularly ill patients with septicaemia, as a method of stabilizing their condition prior to surgery. Drainage of, for example, a gallbladder empyema buys useful time, reducing the risk of perforation and subsequent peritonitis and improving clinical status prior to surgical removal. Although the portable nature of ultrasound allows a bedside procedure to be performed (which is particularly useful in patients under intensive therapy who cannot be moved), these procedures carry a high risk to the patient and full anaesthetic, nursing and medical support is required.

Nephrostomy

Renal obstruction in which the pelvicalyceal system is dilated may be alleviated by the percutaneous introduction of a nephrostomy tube under ultrasound guidance. This procedure relieves pressure in the renal collecting system and avoids potential irreversible damage to the renal parenchyma (Fig. 11.11). Although the procedure may be carried out completely under ultrasound control, it is normally performed in a screening room where a combination of ultrasound and X-ray screening can be used to maximal effect.

Cyst drainage

The percutaneous treatment of renal and hepatic cysts by simple aspiration may afford only temporary relief as they frequently recur, but a more permanent result may be achieved by injecting a sclerosant, for example absolute alcohol or tetracycline into the cyst. In addition, percutaneous treatment of hydatid liver disease (traditionally avoided because of the risk of spreading parasites along the needle track and causing further infection) has been successfully performed by injecting of a scolicidal agent,[11] avoiding the need for surgical removal.

Other applications include draining of pancreatic pseudocysts and inserting a cystogastrostomy tube with combined fluoroscopy and ultrasound guidance; the cyst is allowed to drain through this

tube into the stomach. This is now better done endoscopically.[12]

Indirect ultrasound guidance

Not infrequently drainage of fluid, for example from the pleural cavity, may be performed away from the ultrasound department in the ward or clinic. Although ideally this is done under guidance with a portable scanner, in practice excellent results are obtained for larger, non-loculated collections, particularly pleural effusions, by marking the skin surface with a felt-tip marker in the main scanning department to enable drainage to be safely carried out on the ward.

The mark should be made with the patient in the position in which drainage is to be attempted, for example sitting or decubitus right side raised,

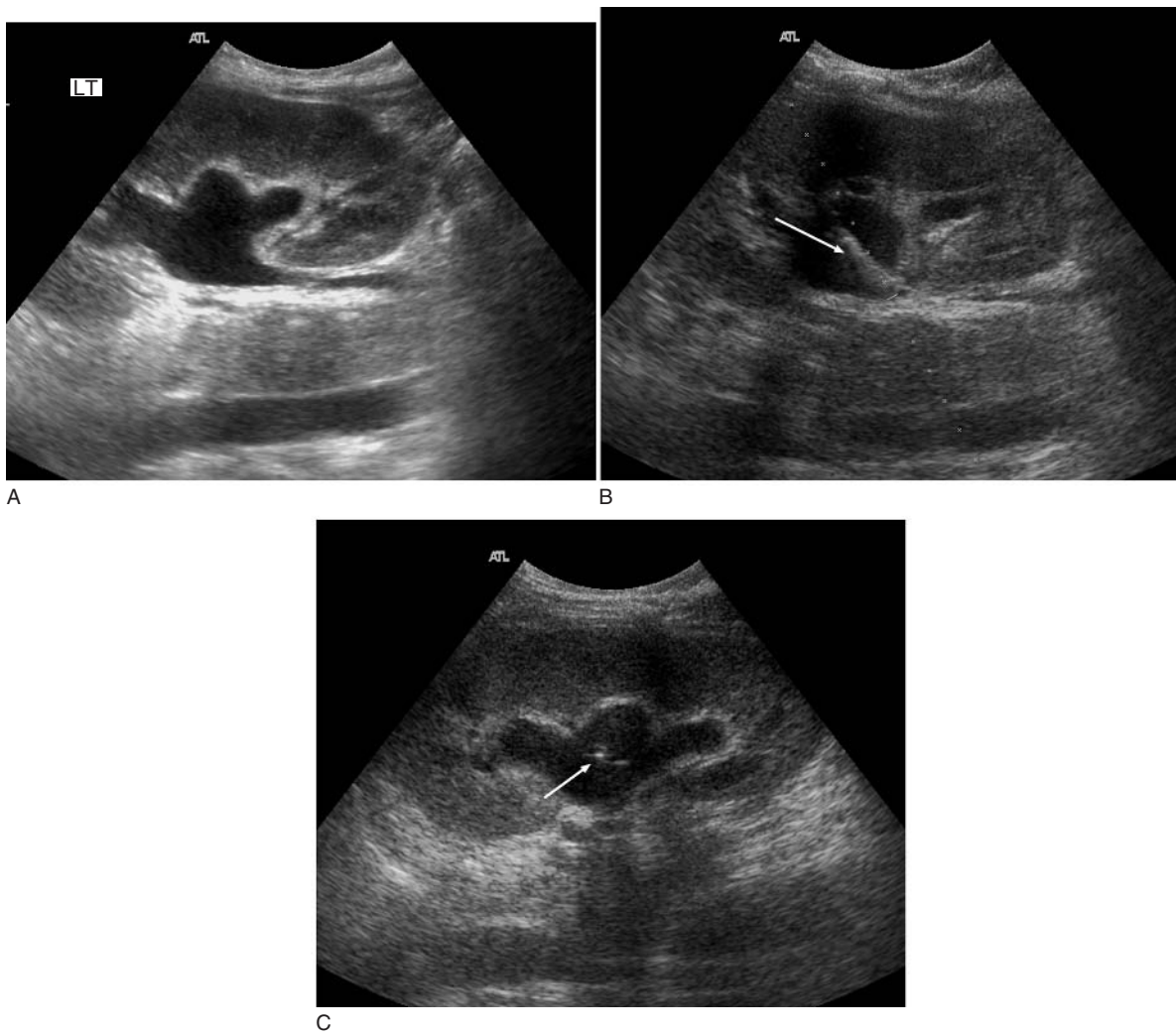


Figure 11.11 (A) Longitudinal ultrasound image of the left kidney. There is clear evidence of hydronephrosis. (B) Similar image during a nephrostomy procedure. The electronic ultrasound guide path can be easily visualized. The guide wire (arrow) can also be seen within the renal pelvis and collecting system. (C) Longitudinal ultrasound scan of the left kidney immediately following nephrostomy. The collecting system remains dilated due to injected contrast. The echogenic tips of the drainage cathether (arrow) can be visualized within the renal pelvis.

and this information communicated to the clinician performing the drainage, together with the depth from the skin surface to the fluid. The puncture site should be marked so that the route is perpendicular to the skin surface. Drainage of pleural effusions and of ascites are the two most commonly performed procedures using this method.

INTRAOPERATIVE ULTRASOUND (IOUS)

IOUS is increasingly used in the abdomen, in both the diagnosis and treatment of lesions. Its applications are varied and its dynamic nature, mobility and high resolution make it ideal for surgical work.

Hepatic IOUS

The most frequent application in the abdomen is in diagnosing and locating liver metastases prior to surgical resection. Resection of metastases, particularly from colorectal tumours, is a potential cure, but results are unsuccessful if small lesions, undetected preoperatively, are not removed at operation.

The direct contact of the IOUS probe with the liver surface, avoiding attenuative subcutaneous tissue, enables a high-frequency (7.5 MHz) probe to be used. IOUS can demonstrate lesions too small to be detected on preoperative imaging, and as a result can change operative management[13,14] in terms of altering the resection line to include more tissue, removing additional hepatic segments or even abandoning the operative procedure altogether.

A combination of surgical palpation, which detects small surface lesions, and IOUS, which detects small, deep lesions, has the highest diagnostic accuracy. IOUS is quick to perform in the hands of an experienced operator and its contribution to the success of surgery is invaluable[15] (Fig. 11.12).

IOUS is particularly useful when there has been a delay between preoperative imaging and surgery, as progression of disease may have occurred during this interval, or when preoperative imaging is equivocal (for example, differentiating tiny cystic from solid lesions). IOUS is often able to offer a definitive diagnosis and when doubt still exists guided biopsy under ultrasound control may be performed. In addition to lesion detection it is able to demonstrate vascular invasion by tumour and to demonstrate clearly, in real time, the relationship of the tumour to adjacent vascular structures; this is essential for planning a resection line. The greater the margin of normal tissue around the resected tumour, the better the long-term prognosis, and a margin of greater than 1 cm normal tissue is preferred. IOUS can also be used to locate deep lesions for ultrasound-guided biopsy or ablation.

Other applications of IOUS

There are numerous extrahepatic applications for IOUS in the abdomen, including urological, vascular and gastrointestinal tract scanning.

Ultrasound evaluation of the common duct for calculi following cholecystectomy can identify small fragments which may not be easily palpable through the duct wall. Using this technique the duct is less susceptible to injury which may be associated with direct examination or the introduction of X-ray contrast agents.

Pancreatic scanning is particularly useful in identifying small tumours of the body and tail of pancreas for curative resection[16] and in differentiating small pancreatic retention cysts from solid nodules.[17]

The treatment of tumours by percutaneous ultrasound-guided techniques, rather than surgical resection, is becoming more common. However, it may not always be possible to achieve success percutaneously and techniques have been developed to ablate tumours during open surgery. Cryotherapy,[18] in which the lesion is frozen by introducing a cryoprobe into the centre of the lesion under intraoperative ultrasound guidance, has been successfully used, but is now largely superseded by radiofrequency and microwave ablation (Fig. 11.13). These techniques have resulted in long-term survival in patients with hepatocellular carcinoma[19] and multiple liver metastases.[20] The success of such techniques depends to a large extent upon patient selection. Those with very large and/or multiple lesions tend to have a poor prognosis compared with patients with smaller, well-confined disease. However these techniques continue to develop and are likely to offer hope to many patients currently untreatable with conventional methods.

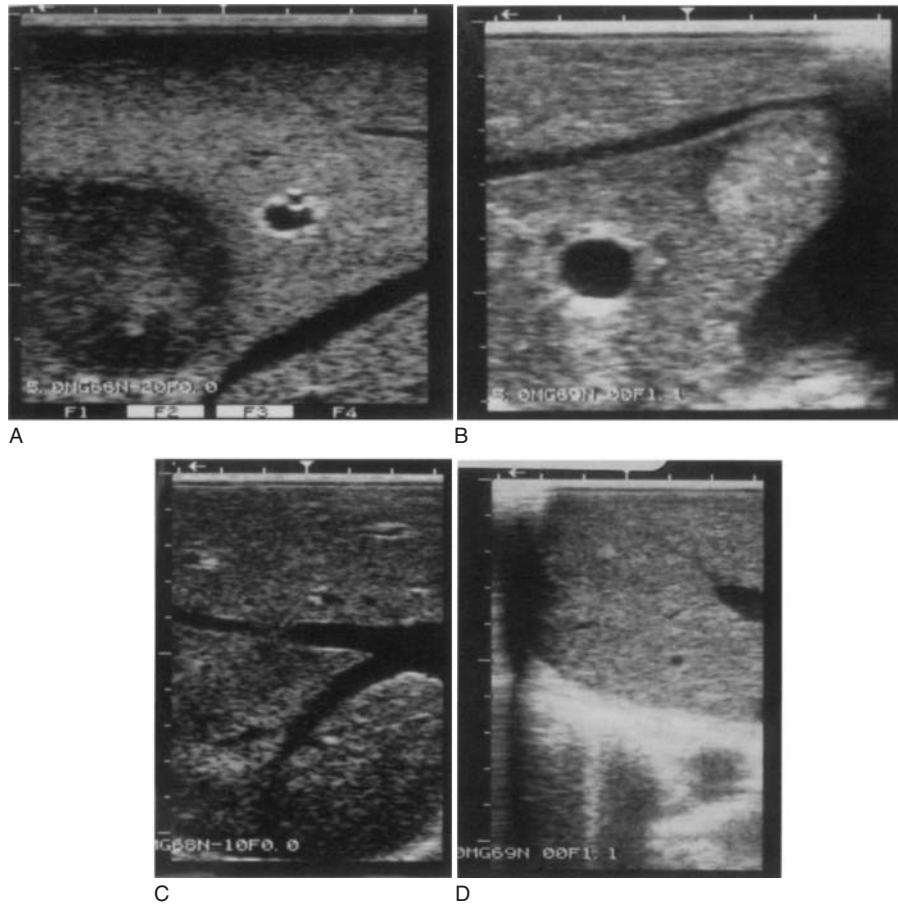


Figure 11.12 Intraoperative ultrasound (A) Demonstrating a margin of tissue of only 2 or 3 mm between the metastasis and the hepatic vein. (B) Metastasis in segment 8, at the confluence of the hepatic veins. (C) This metastasis has started to invade the hepatic vein. (D) Tiny metastasis, not diagnosed on preoperative imaging and not surgically palpable. (Differential diagnosis would be of haemangioma.)

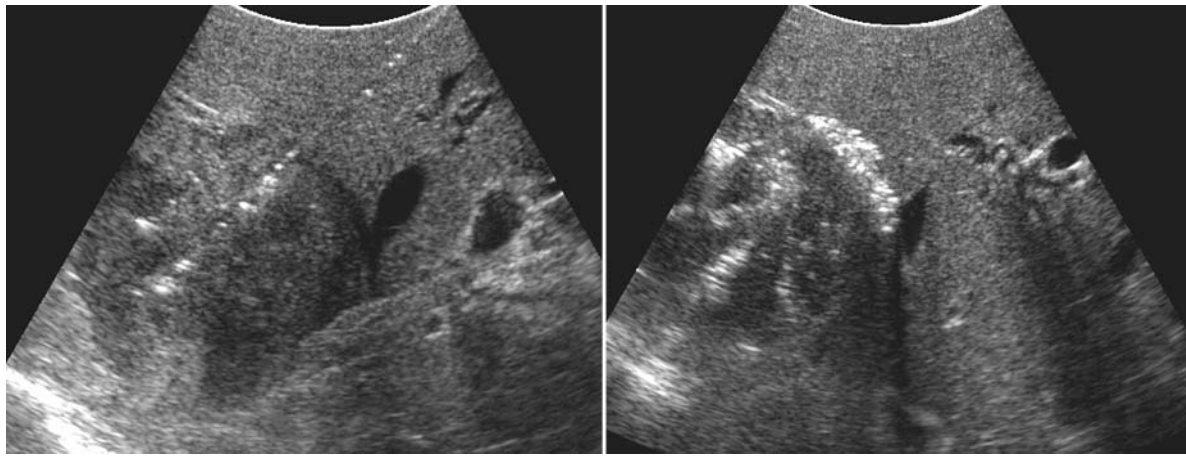
LAPAROSCOPIC ULTRASOUND

Dedicated laparoscopic ultrasound probes may be passed through the laparoscopic port during surgical procedures to investigate the liver, biliary tree, pancreas and other viscera without the need for open surgery (Fig. 11.14).

The trend towards laparoscopic rather than open cholecystectomy has increased the need for accurate laparoscopic exploration of the biliary ductal system to confirm the presence or absence of stones. Laparoscopic ultrasound is better at demonstrating stones in the duct and anatomical ductal variations than conventional intraoperative cholangiography.[21]

Laparoscopic ultrasound has also proven advantageous in staging patients with hepatic tumours for liver resection,[22] demonstrating deep tumours not visible on surgical laparoscopy, or by preoperative imaging methods and so avoiding the need to proceed to open hepatic resection in some patients.

Patients with pancreatic head and ampullary carcinomas are potentially resectable in only a minority of cases. Preoperative imaging is known to underestimate the extent of the disease, and so many patients traditionally undergo a staging laparotomy before resection is attempted. However, over one-third of patients previously considered resectable



A B

Figure 11.13 Radiofrequency ablation (RFA). (A) The RF probe is introduced into the metastasis under intraoperative ultrasound guidance. (B) The lesion is gradually ablated; the area of ablated tissue reflects the sound and can be seen to increase in size progressively during the course of the therapy.

will demonstrate occult metastases, often in the peritoneum.

Staging laparoscopy still cannot demonstrate intrahepatic metastases, and the use of laparoscopic ultrasound at this stage greatly increases the accuracy of staging and influences the surgical decision.[23]

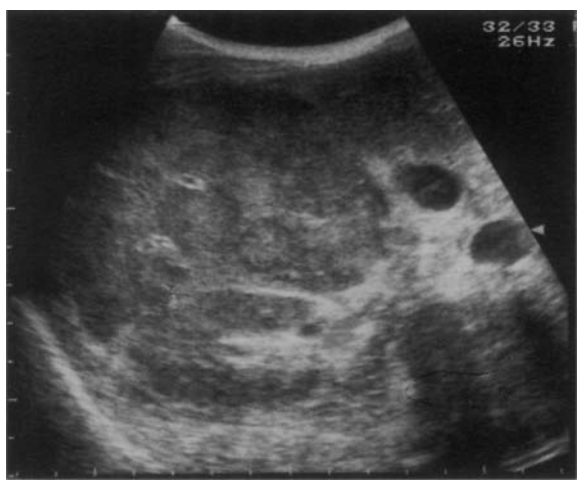


Figure 11.14 Laparoscopic ultrasound demonstrating multiple liver metastases in a patient with carcinoma in the tail of the pancreas.

Laparoscopic ultrasound is also useful in staging patients with gastric cancer[24] and colorectal cancer.[25] Curative resection of bowel cancer can be performed with either open surgery or laparoscopic resection. Laparoscopic ultrasound can be used to examine the liver to confirm the absence of metastases: this is particularly useful in a laparoscopic resection as the surgeon is unable to palpate the liver under these circumstances. This laparoscopic approach reduces patient morbidity when compared with open surgical exploration.

ULTRASOUND CONTRAST AGENTS IN THE ABDOMEN

Ultrasound contrast media have been well established for cardiac imaging since the 1980s and the first clinical use of such an agent was in 1968 and involved the injection of saline to identify echoes from the mitral valve.[26]

These early contrast agents were composed of relatively large (by today's standards) microbubbles of air in solution. They were unstable, short-lived and the bubbles were too large to pass through the capillary beds, hence their use exclusively for cardiac ultrasound.

Since those early years there have been a number of developments in the field of contrast ultrasound. Agents such as Albunex (Molecular Biosystems, San Diego, USA), consisting of albumin-coated microbubbles, were small enough to pass through the pulmonary capillaries and enter the left side of the heart; however they were too weak to withstand systolic pressure and could not therefore enter the blood pool in any appreciable quantity.

A more stable suspension was then produced, consisting of small microbubbles in the order of 2–5 μm which passed through the pulmonary capillary bed after intravenous injection, and acted as a true blood-pool agent. Called Levovist (Schering, Berlin, Germany), this is a galactose-based agent (99.9%) containing palmitic acid (0.1%) for stability, which traps air which is subsequently released when the bubbles burst. As the first stable blood-pool agent it could be used for examining the abdominal viscera and vasculature.

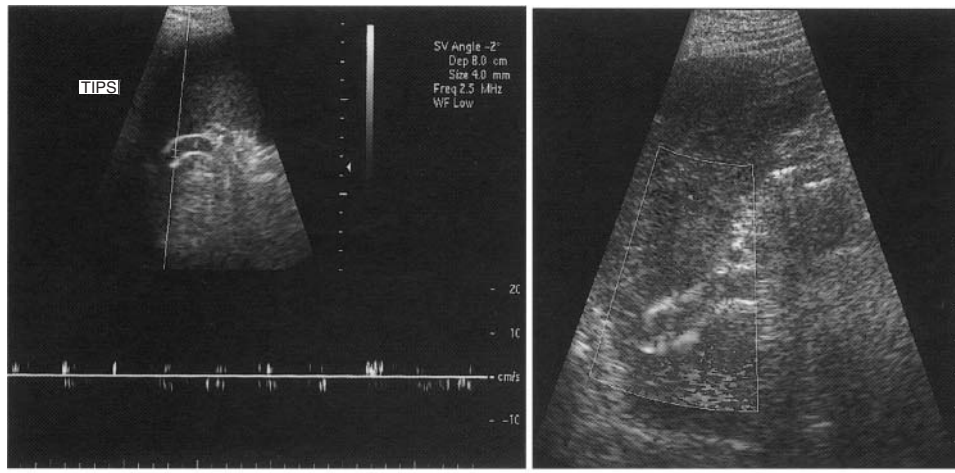
By coincidence, microbubbles of this size can pass through the pulmonary capillaries and resonate at frequencies used in clinical diagnostic ultrasound (1–20 MHz). This resonance causes a much greater capacity for scattering the beam than that from a non-resonating particle and thus a stronger signal is produced of up to 25 dB on both grey-scale and Doppler. The Doppler signal from a contrast-enhanced blood vessel is therefore much easier to identify. In addition, vessels too small to be identified on normal grey-scale or non-enhanced Doppler scans can be identified when using a microbubble agent (Fig. 11.15).

Despite the use of microbubble agents, blood flow in tiny vessels can still be difficult to detect; harmonic imaging techniques however aid detection further. When insonated with ultrasound of a certain frequency, microbubbles emit a secondary harmonic frequency twice that of the incident wave, in addition to the primary harmonic. As the second peak is a purer signal, this increases the sensitivity, enabling smaller vessels with slow flow to be successfully detected and distinguished from surrounding tissues. Many harmonic-based pulse methods are used with contrast agents and some of the more popular ones include pulse and phase inversion, for example pulse inversion is a dual pulse technique, 180° out of phase, resulting in summation of signal from non-linear scatterers (microbubbles) and cancellation of signal from linear scatterers (tissue).

Potential applications of these agents include situations in which ultrasound findings are equivocal or in which Doppler information is suboptimal. A contrast agent will enhance the Doppler ultrasound signal from the blood pool and increase diagnostic confidence. This may therefore obviate the need for other more invasive angiographic investigations.[27] These agents therefore have the potential to extend the applications of Doppler ultrasound in the abdomen.

With regard to the abdomen it can be useful in patients with chronic liver disease for the investigation of portal vein thrombosis. Increased sensitivity and specificity have been reported for examination of the portal vein,[28] avoiding the need for contrast angiography. In patients with hepatic transplantation it is helpful in confirming hepatic artery patency in the early postoperative period: this can be difficult to confirm with conventional imaging alone. Although it is felt intuitively that it may be of help in the diagnosis of renal artery stenosis, the evidence is as yet not convincing to support its routine use.

The diagnosis and characterization of hepatic tumours are also improved with contrast agents as these agents, for example, Levovist and Sonovue, are preferentially taken up by the hepatosplenic parenchyma and so focal lesions appear as filling defects in much the same way as CT or MRI (Fig. 11.16). The exact site of accumulation within the liver is unknown but may be within the reticuloendothelial system or liver sinusoids. There is growing evidence to support the differentiation of lesions within the liver, not previously possible with conventional ultrasound,[29,30] and certainly microbubble agents are helpful in the diagnosis of capillary haemangiomas, hepatomas and focal nodular hyperplasia. How are microbubble agents used currently? Generally an agent such as Sonovue, currently the most commonly used, which consists of a phospholipid membrane containing perfluorocarbon gas, is injected intravenously. Imaging, for example in the liver, can now be performed in both a hepatic arterial and portal venous phase similar to CT. Most imaging is performed with a harmonic-based technique—pulse or phase inversion—and utilizing a low mechanical index (< 0.15) to



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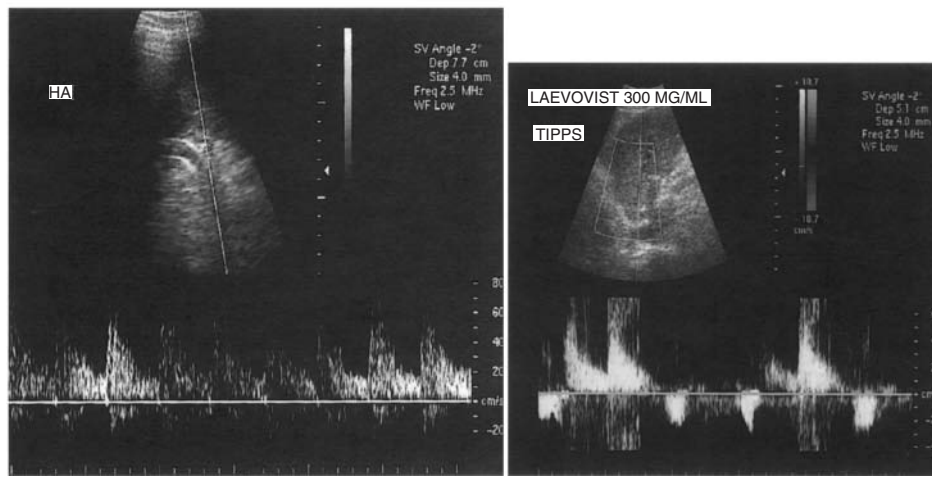


B (i) B (ii)

Figure 11.15 (A) i, Pre-injection of contrast—no flow is demonstrated in this transjugular intrahepatic portosystemic shunt; ii, post-injection, flow is still not demonstrated, confirming the shunt to be thrombosed. (B) i, the same patient—pre-injection the hepatic artery is identified adjacent to the shunt; ii, post-injection: without altering the settings, greatly enhanced arterial Doppler signals are demonstrated from even the most peripheral hepatic arteries.

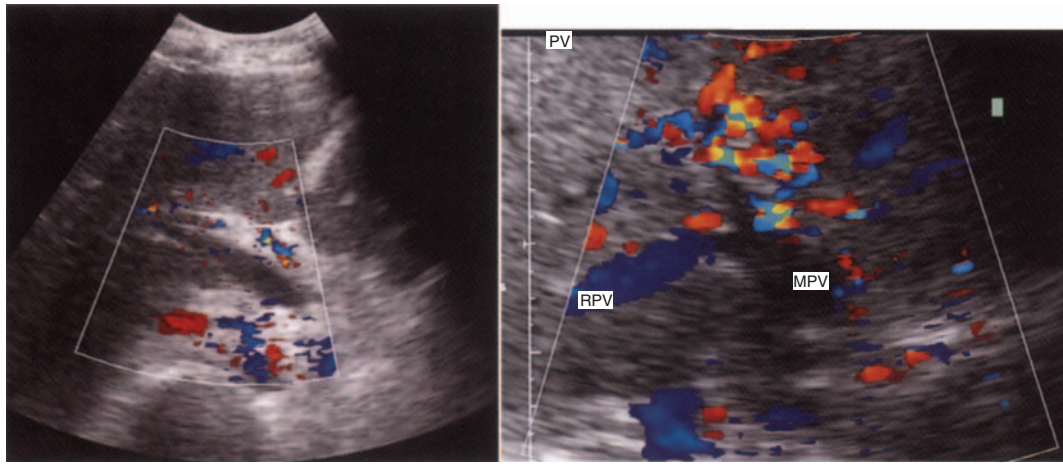
(continued)

prolong longevity of the bubbles. Although there is no definitive evidence as yet to support its routine use, it is currently undergoing a number of trials comparing it with CT and MRI in the detection of focal liver disease, and early results are favourable.[31]

Other simple substances, technically considered contrast agents, including water or saline, are used to outline the stomach (for example, to visualize the pancreas or to assess the nature of an epigastric mass) or the rectum. In the future, oral ultrasound contrast agents may be developed specifically to examine the stomach or colon and to reduce bowel gas.

THE TREATMENT OF PRIMARY AND SECONDARY HEPATIC TUMOURS BY PERCUTANEOUS METHODS

In patients with colorectal carcinoma the presence of liver metastasis is the most accurate predictor of survival. Resection of liver metastases is known to increase the lifespan of patients, with good quality



c(i) c(ii)

Figure 11.15 cont'd (C) i, Before contrast injection, portal vein thrombosis is suspected in a patient with alcoholic liver disease; ii, after injection, the main portal vein is confirmed as thrombosed. Forward flow is seen in the right portal vein (presumably due to collateral circulation), and increased hepatic arterial flow is clearly demonstrated.

of life and an overall 5-year survival of 20–45%, and up to 60% in unifocal disease. Without surgery the 5-year survival in this patient group is effectively zero. However, not all patients with liver metastases are deemed suitable for resection, being poor surgical risks or having lesions which are either too large or affect too many hepatic segments. Percutaneous ablation of liver tumours is now a viable and rapidly developing option for control of liver metastases, prolonging survival time after initial diagnosis and, in some cases, shrinking tumours to enable future curative resection. Various methods have been investigated, using ultrasound guidance.

- Alcohol injection has proved highly effective for hepatocellular carcinoma (HCC),[32,33] shrinking tumours over a period of time and causing necrosis within them, but has not proved as effective for metastatic liver disease. This is thought to reflect the fact that HCC is a 'soft' tumour and so the alcohol can be instilled effectively into the tumour whereas metastases are 'hard' lesions and often the alcohol seeps out of the lesion.
- Radiofrequency (RF) thermal ablation and laser ablation are also developing as minimally invasive percutaneous therapeutic techniques and are becoming increasingly popular.

Ablation of liver metastases using RF is a recent method of ultrasound-guided therapy for liver metastases and HCC in which RF, applied to monopolar electrodes either individually or with multiprobe arrays, is guided into the lesion to be treated. RF tissue ablation through an 18G needle uses fewer probes than laser. It is an outpatient procedure: 1–4 sessions has been reported to achieve complete necrosis of liver metastases in 67% of lesions.[34] It is a simple, safe and potentially effective treatment for liver metastases, associated with a low rate of complications (in one study only one small area of haemorrhage was observed in 75 sessions)[35] together with a significant rate of shrinking or stabilization of the metastases.

ENDOSCOPIC ULTRASOUND

Some of the limitations of conventional ultrasonography in biliary and pancreatic imaging can be overcome by the use of endoscopic probes and miniprobes. Endoscopic probes are either radial or linear arrays which are incorporated into the end of an endoscope. They have a frequency of 7.5–12 MHz and are used to image the pancreas, biliary tract, portal vein and adjacent structures

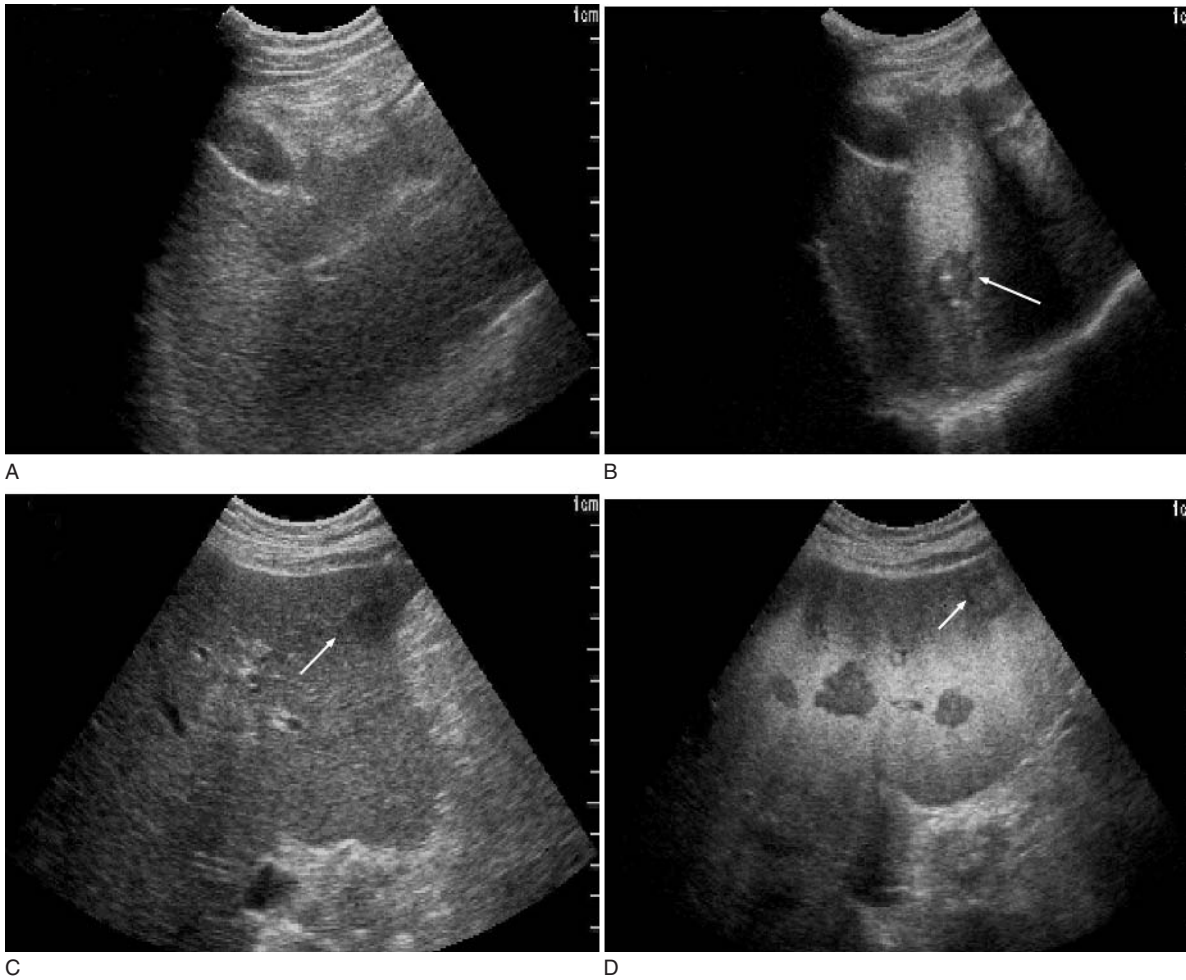


Figure 11.16 (A) Conventional ultrasound of the liver showing no abnormality. (B) Pulse inversion mode following intravenous Levovist injection showing a focal lesion (arrow) i.e. metastasis in the same patient as Fig. 16a. (C) Conventional grey-scale scan of the liver. A number of metastases were seen throughout the liver. One in the left lobe has been arrowed. (D) Pulse inversion mode with intravenous Levovist in the same patient as (C). The metastasis seen on the unenhanced grey-scale image can still be seen (arrow); however, easily discernible additional lesions are now also appreciated.

within 5–6 cm of the probe. Radial probes may be used in the preoperative staging of a number of diseases, including oesophageal, gastric, pancreatic and lung cancer, whilst linear array probes are used for interventional procedures such as fine-needle aspiration analysis of mediastinal lymph nodes, solid organ assessment, for example pancreas, occasionally liver, adrenals, pseudocyst drainage and coeliac plexus neurolysis.

Endoscopic ultrasound is more sensitive and specific than spiral CT, MRI or transabdominal ultrasound in the detection of small pancreatic masses and its diagnostic ability can be further enhanced by the use of endoscopic ultrasonically guided fine-needle aspiration cytology[36] and biopsy.

It may also detect early changes of pancreatitis which are not visible on endoscopic netrograde cholangiopancreatography (ERCP), and one of its

main uses is in staging pancreatic tumours, predicting their resectability, identifying small lymph node metastases and assessing vascular invasion.[37] It is particularly accurate in identifying small pancreatic insulinomas,[38] often difficult or impossible to identify on conventional cross-sectional imaging despite a documented biochemical abnormality, and thus guiding subsequent surgical procedures. Endoscopic ultrasound is also used in the detection of biliary calculi, particularly in the normal-calibre common bile duct, with a much higher accuracy than other imaging techniques and without the potential additional risks of ERCP.[39]

Further, less-established uses of endoscopic ultrasound include gastrointestinal examinations, in which invasion of gastric lesions into and through the wall of the stomach can be assessed,[40] anal ultrasound, which is used to visualize the sphincter muscles in cases of sphincter dysfunction, the staging of colorectal carcinomas and the demonstration of bowel wall changes in inflammatory bowel conditions.[41]

The miniprobe has a higher frequency (20–30 MHz) and may be passed down a conventional endoscope. It therefore has the advantage of a one-stage gastrointestinal tract endoscopy/ERCP, rather than requiring a separate procedure. It may be inserted into the common duct of the biliary tree to assess local tumour invasion and to clarify the extent and/or nature of small lesions already identified by other imaging methods. It shows remarkable accuracy in the detection of common bile duct tumours and other biliary tract disease when compared with other imaging modalities.[42]

It may be used in the staging of oesophageal and gastric cancer, and is especially useful when a tight oesophageal stricture prevents the passage of the endoscope.[41] The layers of the oesophageal or gastric wall and the extent of tumour invasion can be accurately assessed.

The miniprobe is also used in patients with suspected pancreatic carcinoma, for example in patients with a negative CT but who have irregularity of the pancreatic duct on contrast examination. The probe can be passed into the pancreatic duct during ERCP to detect small lesions, assess the extent of the tumour and predict resectability.[43] It is superior to conventional endoscopic ultrasound in the detection of the smaller, branch tumour nodules, and can also detect local retroperitoneal or vascular invasion in areas adjacent to the probe.

The use of endoscopic ultrasound is currently limited to a few specialist centres. A steep learning curve together with the expense of the equipment is likely to restrict its widespread use; however, as its applications expand and its value becomes proven, it is likely to become a more routine investigation at many centres.[41]

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