Chapter **4**

Pathology of the liver and portal venous system

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.

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
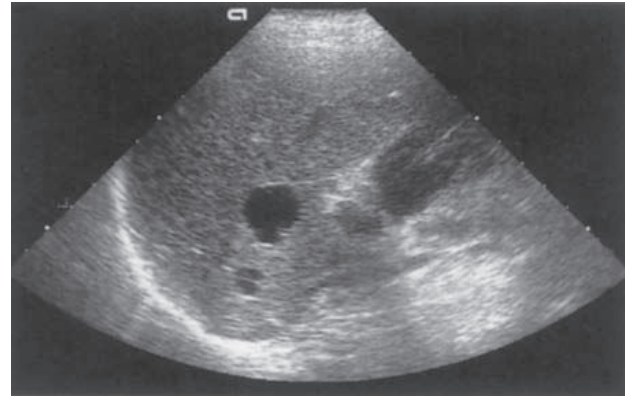

Figure 4.1 Typical simple liver cyst demonstrating a band of posterior enhancement. A smaller, bilocular cyst is seen behind it.

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.
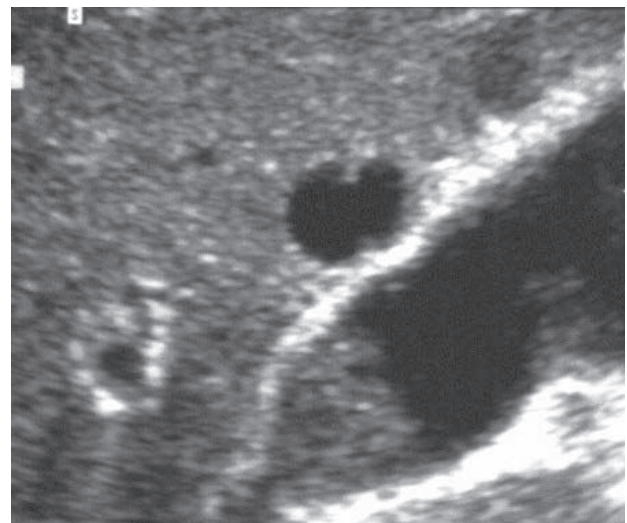
Figure 4.2 Small cyst adjacent to the gallbladder containing a nodule. This was a mucinous metastasis from an ovarian carcinoma.
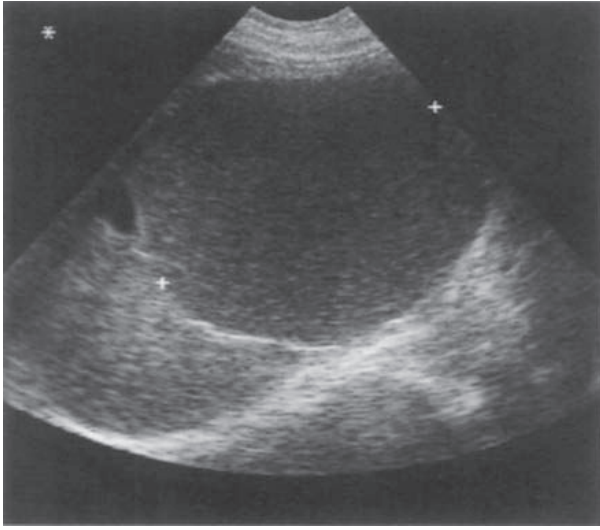
Figure 4.3 Large, infected hepatic cyst containing low-level echoes.

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 ultra-sound 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 through-out the structure.

Polycystic liver

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

A   B

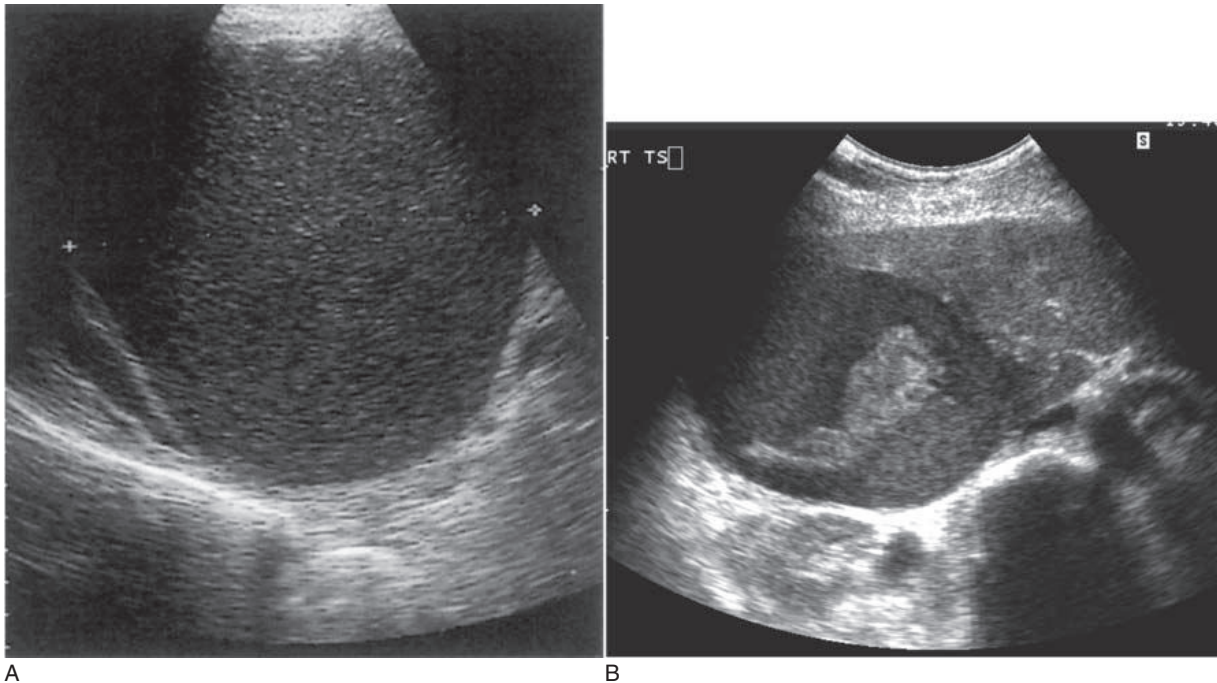**Figure 4.4** (A) Large cystadenoma containing echoes and a septum. The cyst was large enough to cause obstructive jaundice—the patient's presenting symptom. The diagnosis was made by ultrasound-guided aspiration. This cyst had developed into a cystadenocarcinoma after 2 years. (B) A cystadenocarcinoma 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
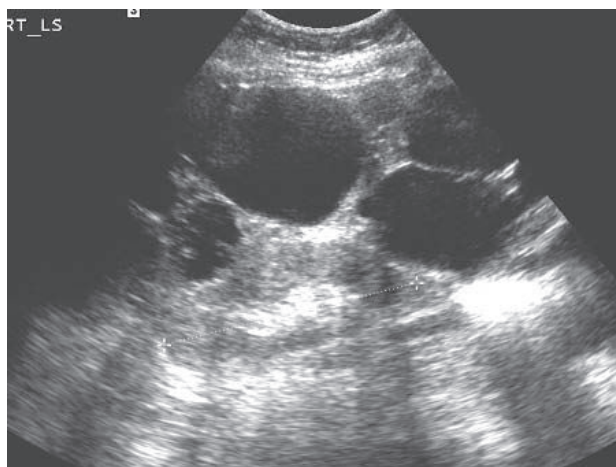

Figure 4.5 Multiple cysts in the liver. In this case the kidneys are normal. Polycystic liver is more usually associated with polycystic kidney disease.
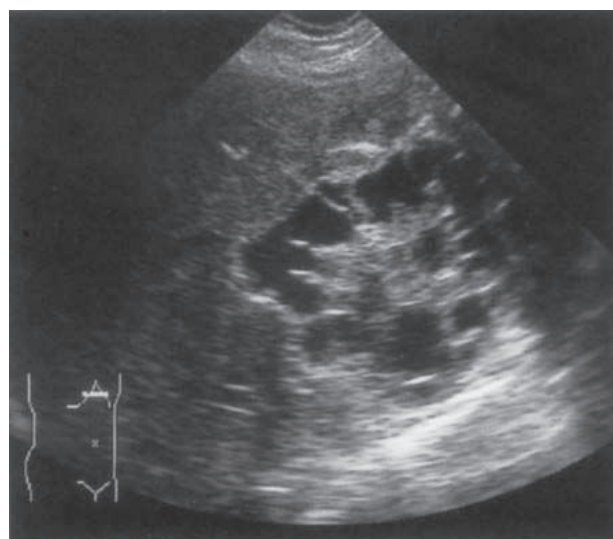

Figure 4.6 Hydatid cyst demonstrating surrounding daughter cysts.

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
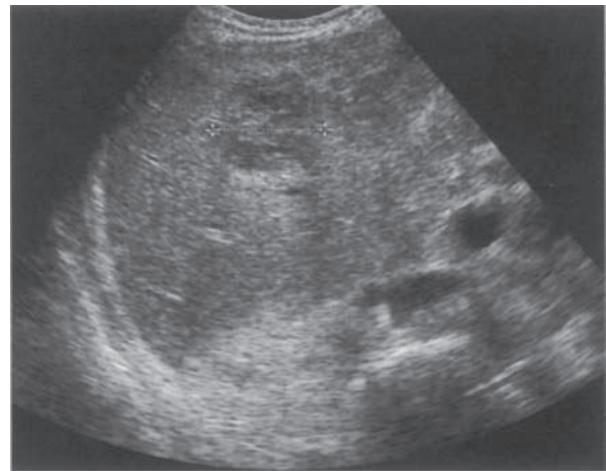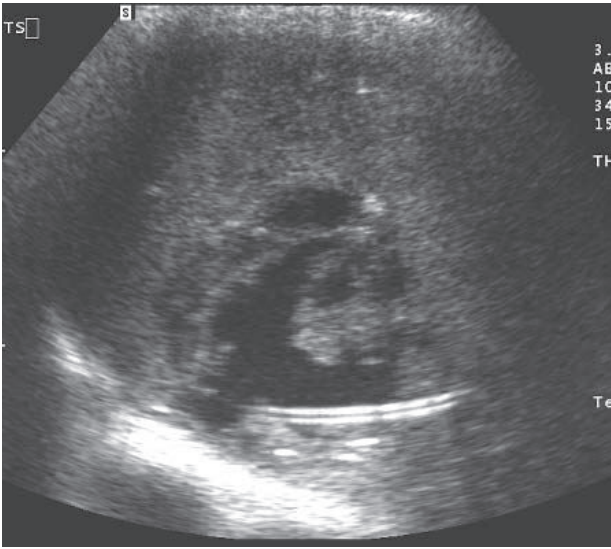

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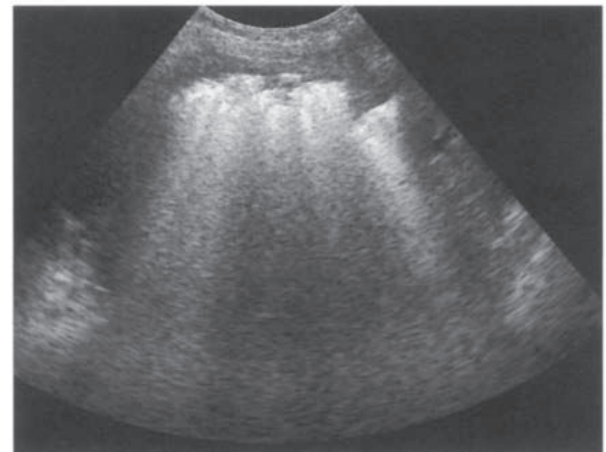
B

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.)

(Continued)

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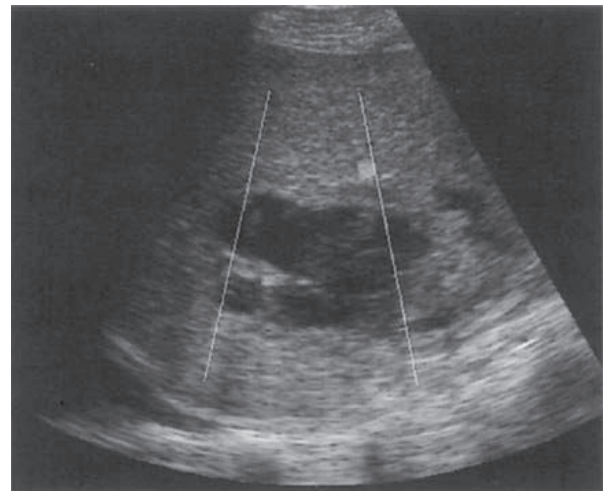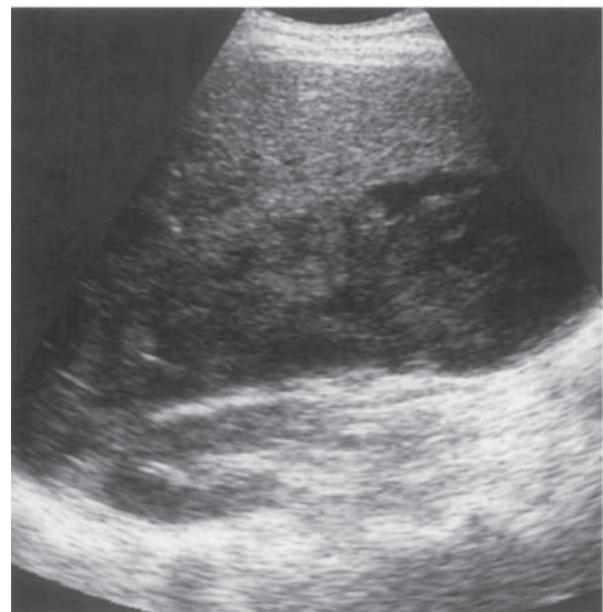
Figure 4.8 (A) Intrahepatic haematoma following a road traffic accident with rib fractures. The lesion is relatively fresh and contains some low–level echoes. (B) 2-day-old subcapsular haematoma. The collection became progressively smaller and hyperechoic as it resolved.

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.
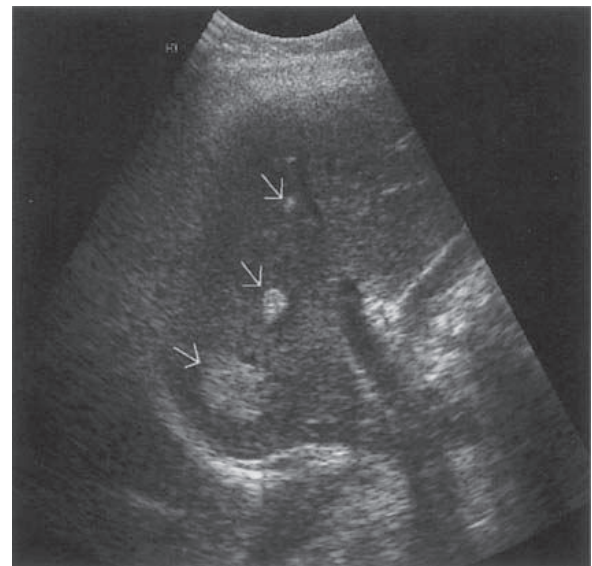
Haemangioma

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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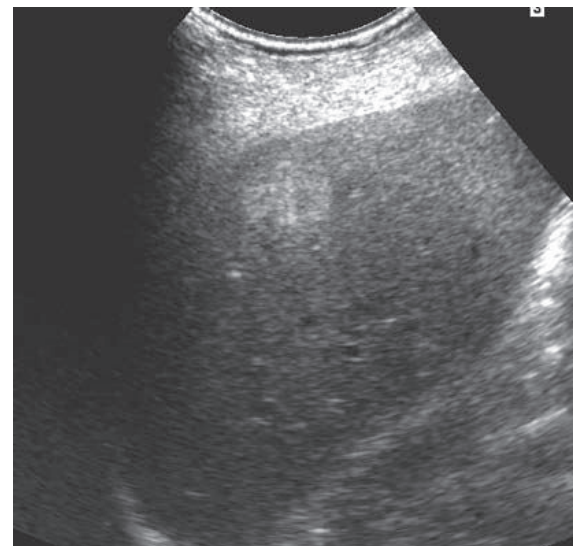
B

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.
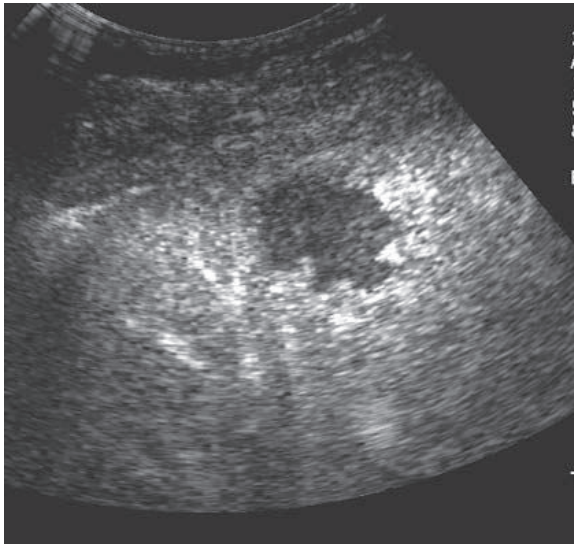

A

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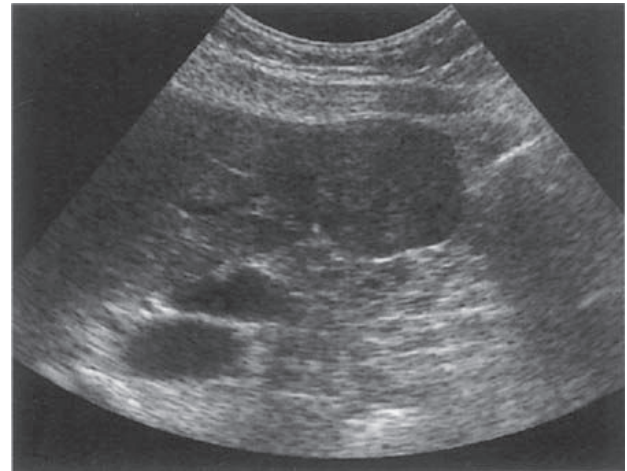
well-defined, hyperechoic mass are due to benign haemangioma. Follow-up scans will demonstrate no appreciable change over time. However, where doubt exists, it is useful to refer the patient for further imaging, such as MRI scanning, to characterize the lesion confidently.

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]
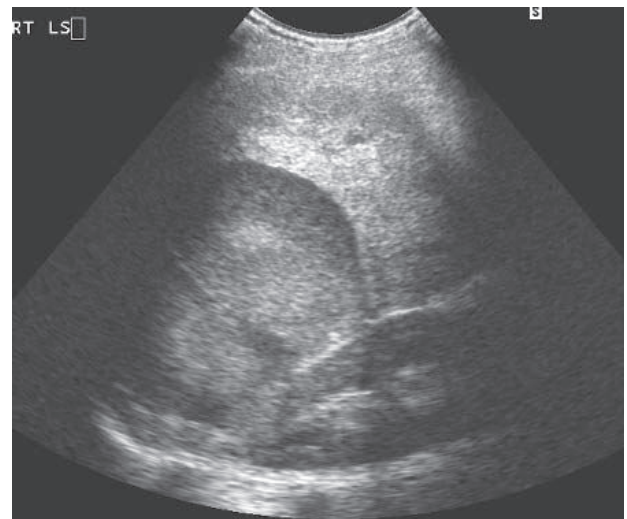
Adenoma

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.

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).
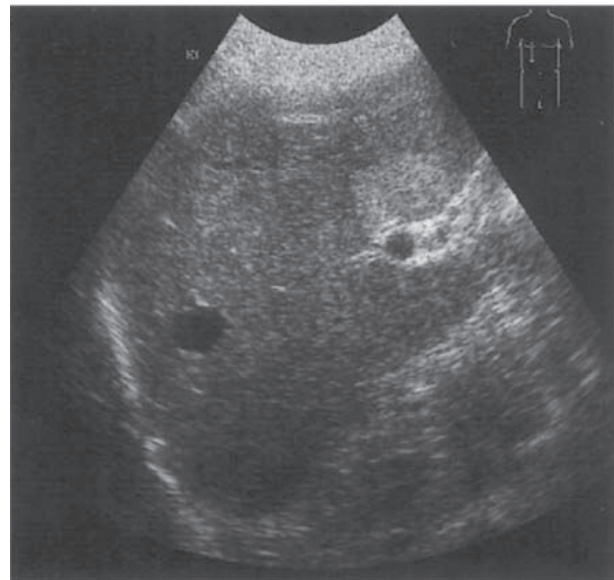
Focal fatty sparing

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.
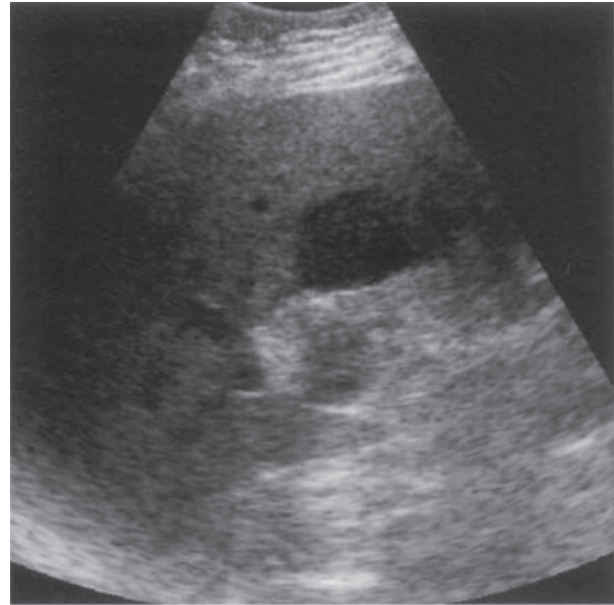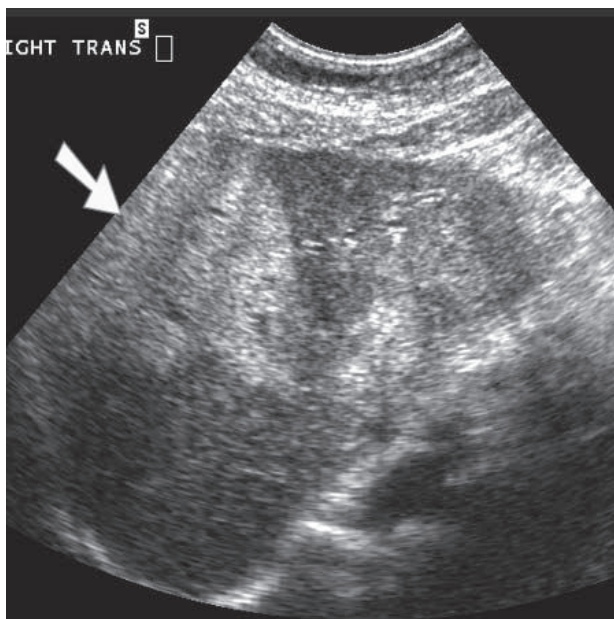
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B

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
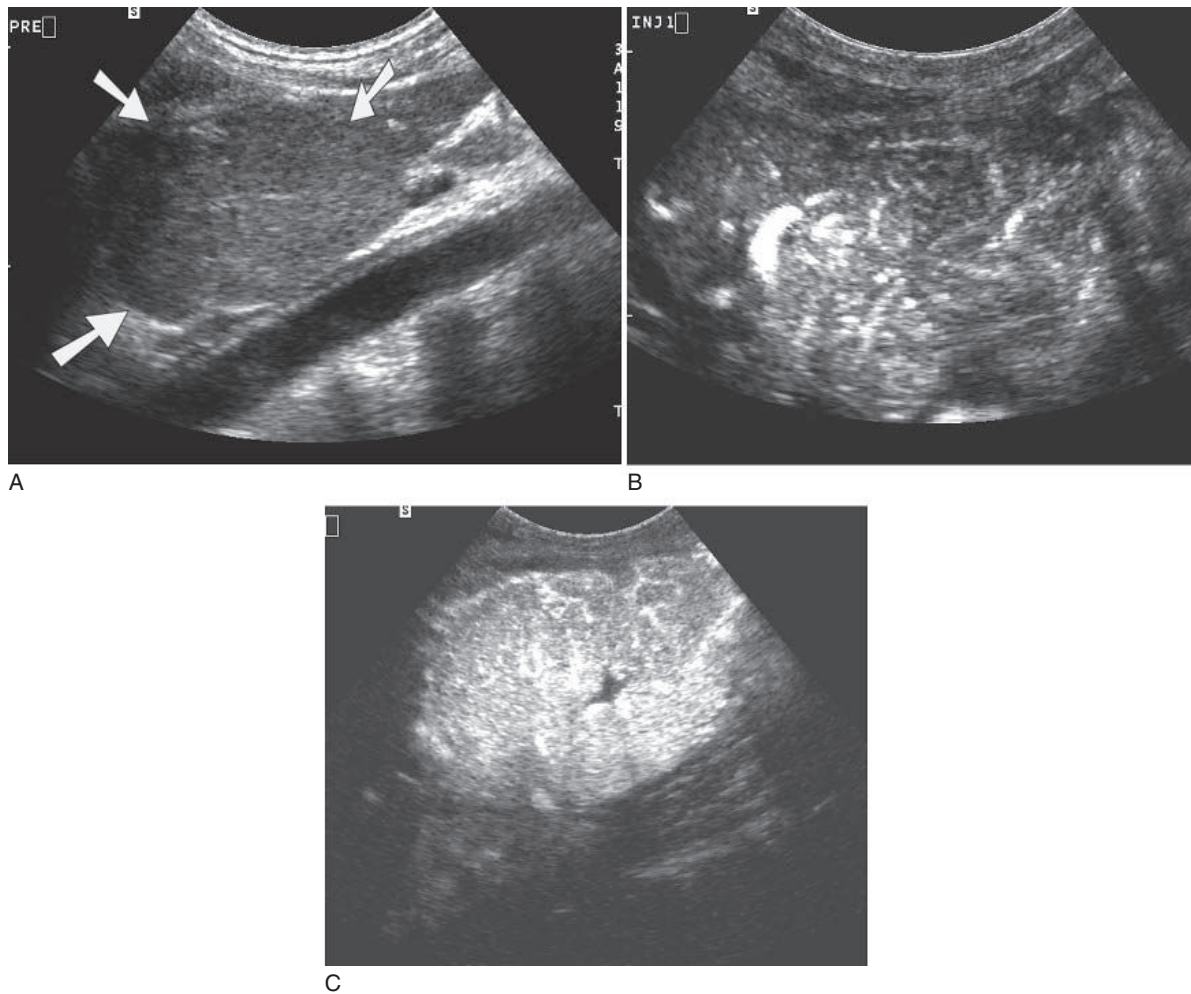
Figure 4.12 (A) Focal nodular hyperplasia in the left lobe (arrows), which is isoechoic with normal liver tissue. (B) Following administration of microbubble contrast agent, the FNH displays a 'spoked-wheel' pattern of vascular enhancement during the early arterial phase. (C) The same lesion seconds 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.
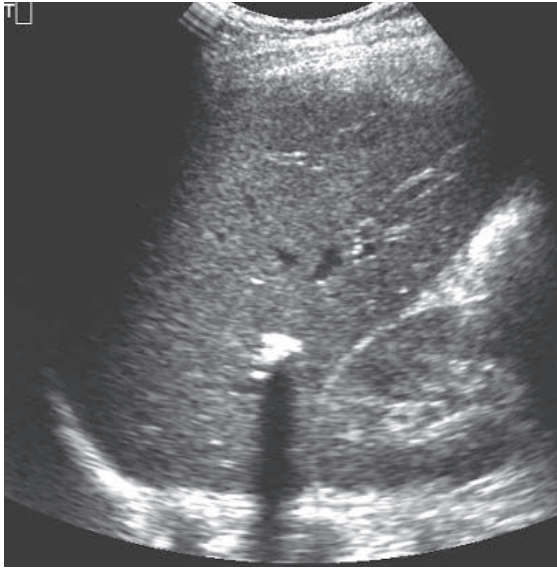
Figure 4.13 A calcified granuloma demonstrates acoustic shadowing.

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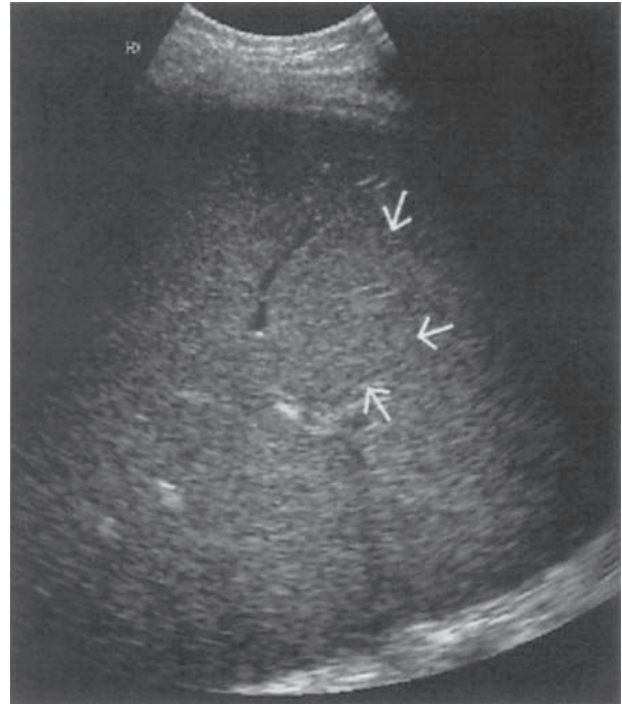

Figure 4.15 The mass effect: an isoechoic lesion (arrows), confirmed on CT, is recognized because of the adjacent deviation of the portal and hepatic venous radicles.

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.

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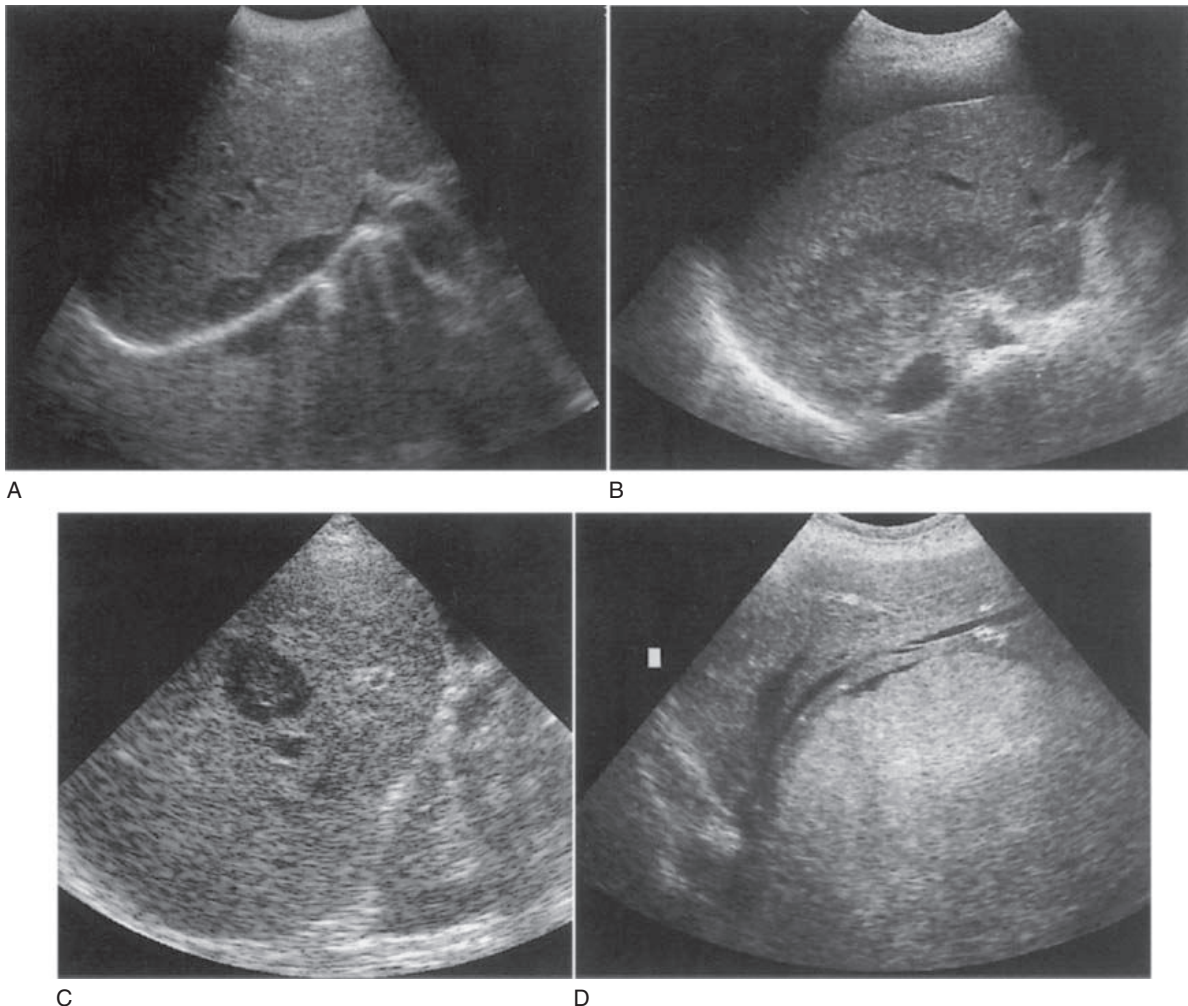
Figure 4.16 Examples of liver metastases. (A) Peripheral secondary deposits due to peritoneal spread from a primary ovarian carcinoma. (B) Blood-borne metastases from bowel carcinoma are demonstrated in the central area of the liver around the porta. (C) Solitary 'target' metastasis. (D) Large hyperechoic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

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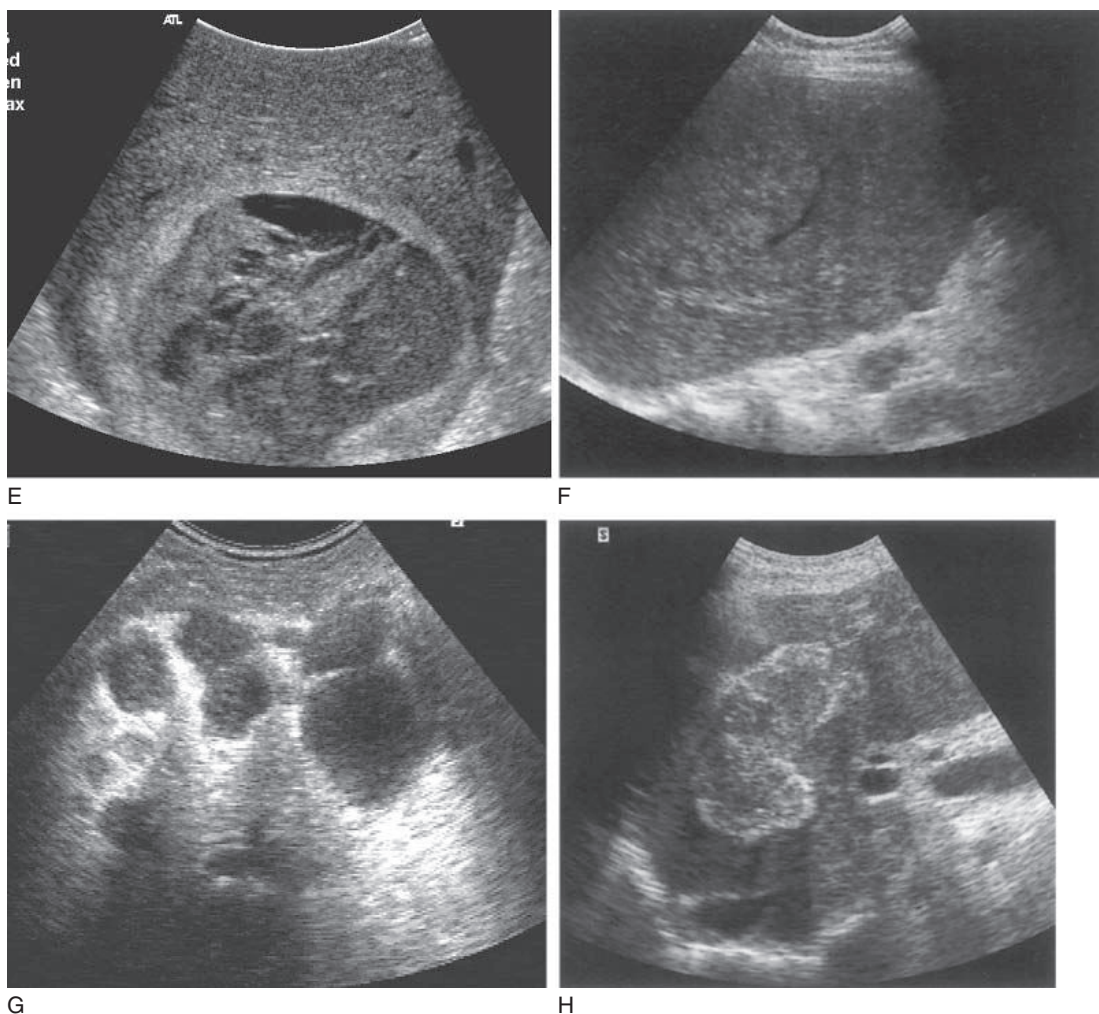
Figure 4.16 cont'd (E) Large necrotic metastasis. (F) Miliary metastases affecting the entire liver. Some larger, focal lesions are also visible. Note the hepatic enlargement and the lobulated outline of the liver. (G) Following administration of microbubble contrast agent, numerous metastases are discovered. 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.)
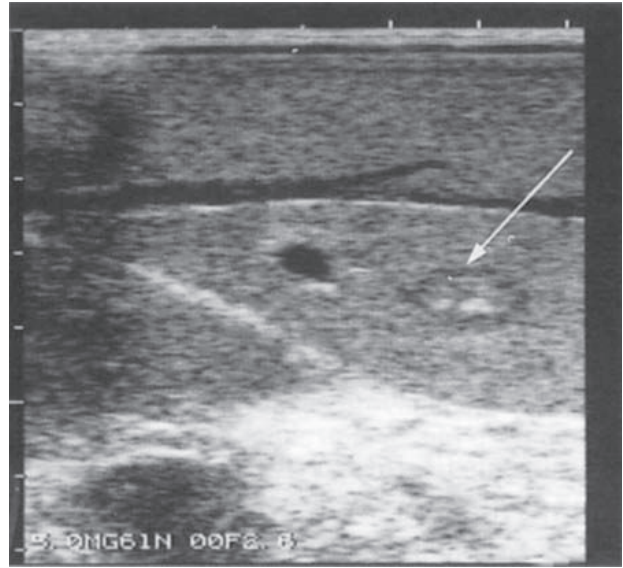

Figure 4.17 Intraoperative ultrasound scan demonstrates a small metastasis (arrow) in segment 4.

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.
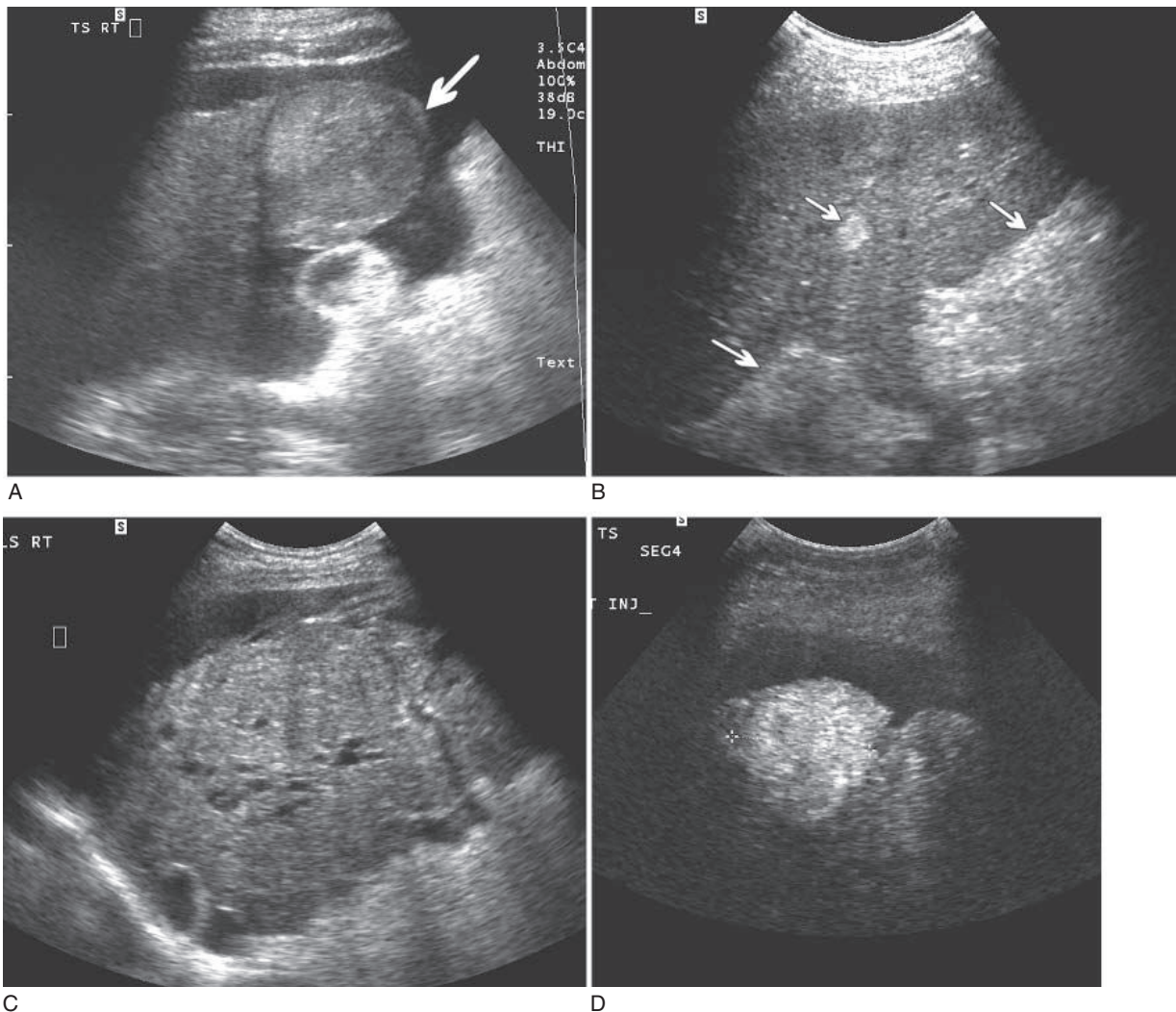
Figure 4.18 (A) Exophytic hepatocellular carcinoma (HCC) in a patient with cirrhosis. (B) Multifocal HCCs (arrows) in a cirrhotic patient. (C) A patient with chronic Budd–Chiari syndrome has a nodular liver with suspicion of a lesion near the anterior surface. (D) Administration of contrast in the same 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.
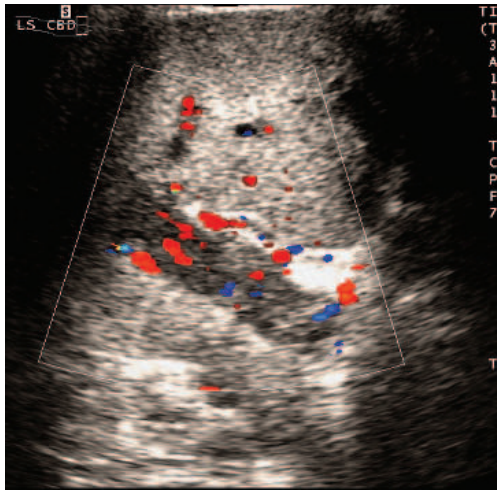
Figure 4.18 cont'd (E) Tumour thrombus almost occluding the PV in a patient with multifocal HCC.

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.

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 demon-

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.

strated 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 haemo-globin (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

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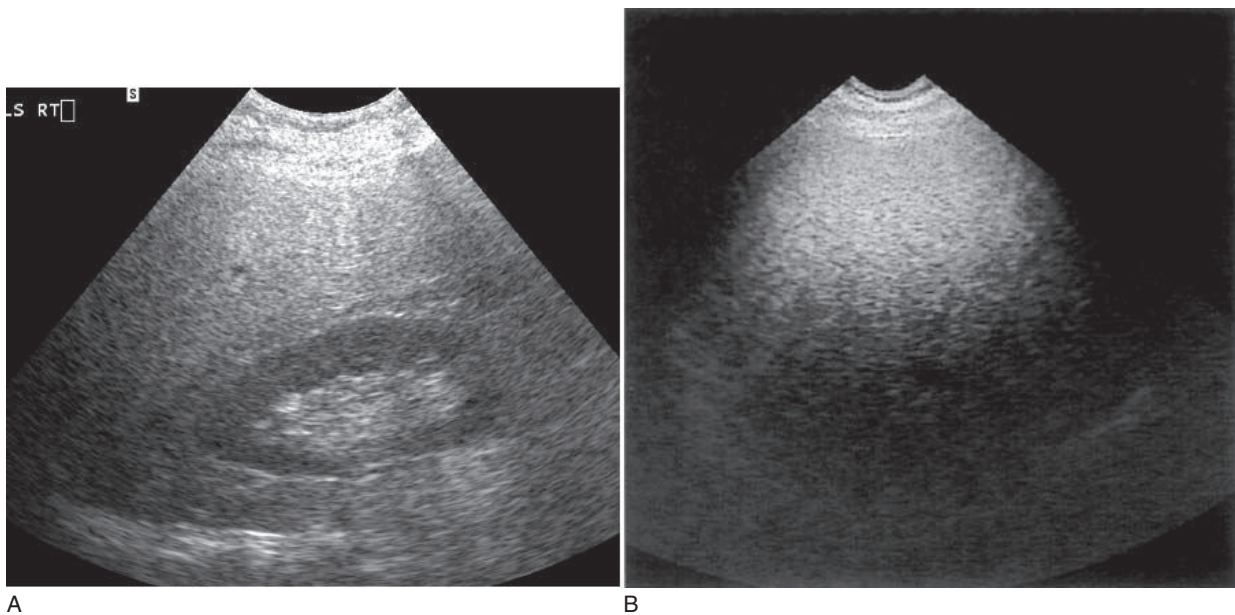
Figure 4.19 (A) Fatty infiltration increases the hepato-renal contrast. The portal tracts are reduced in prominence, giving a more homogeneous appearance. (B) Attenuation of the beam by fat prevents demonstration of far-field structures.

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).
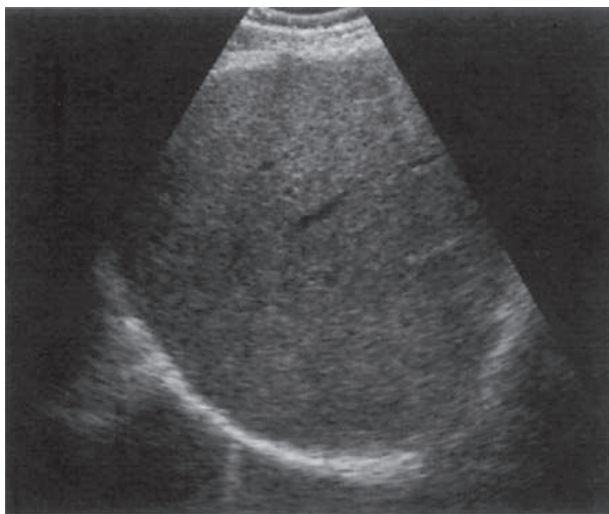
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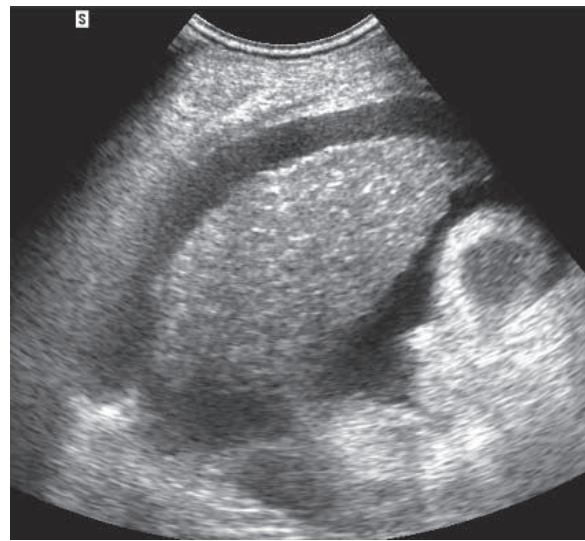
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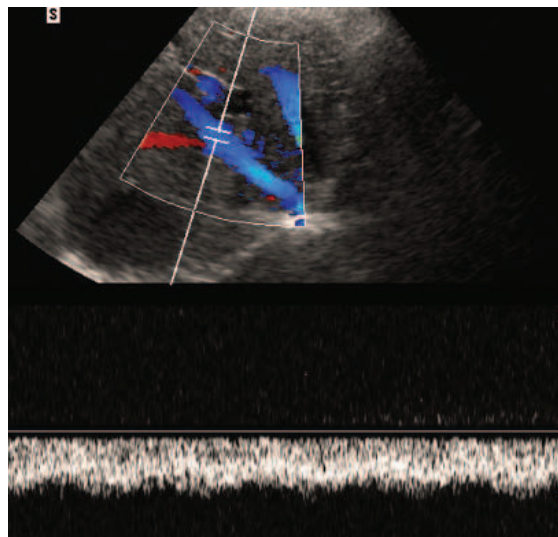
C

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

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.

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).

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

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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
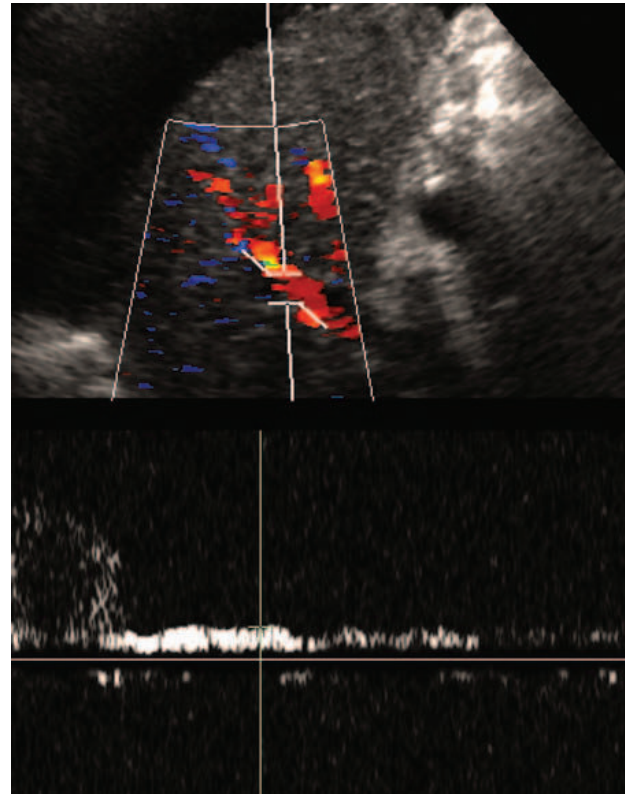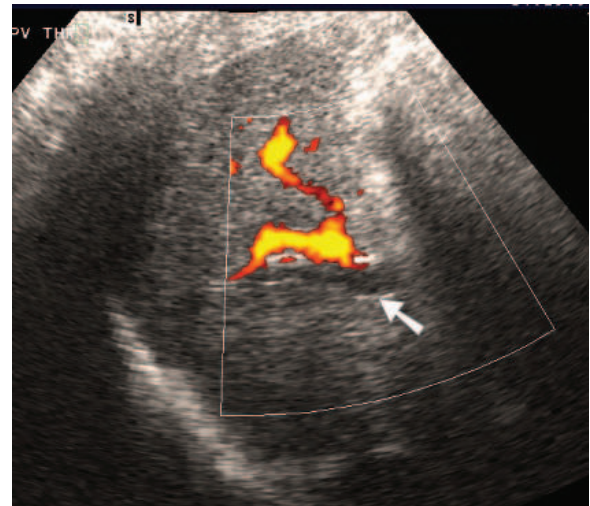

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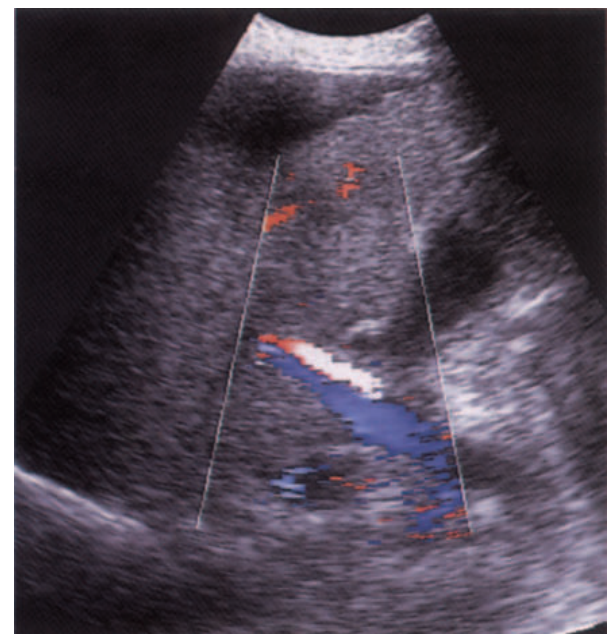
B

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)*
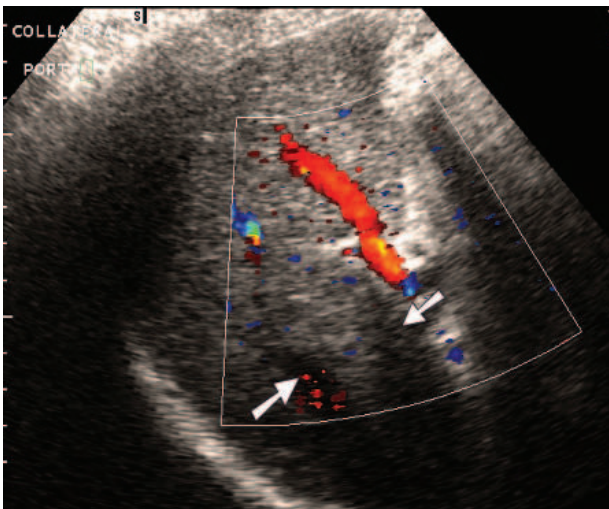
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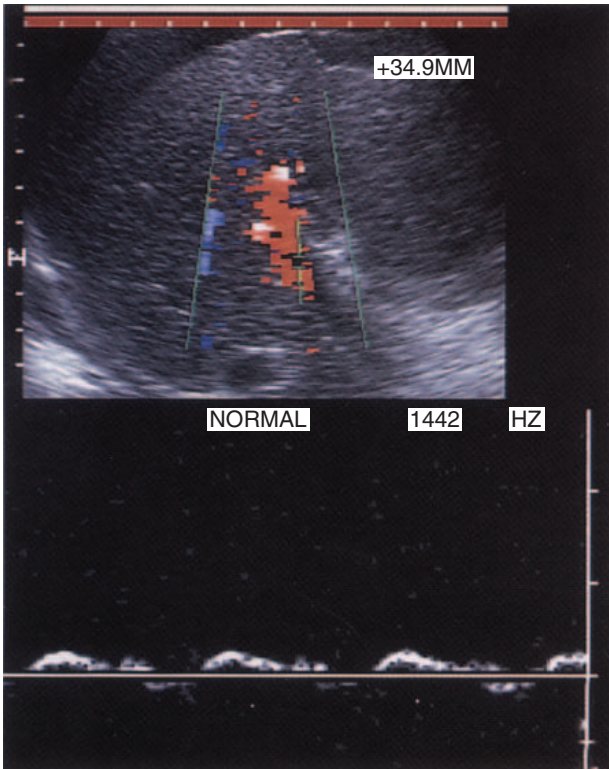
Figure 4.21 cont'd   (C) Balanced PV flow. Alternate forward and reverse low-velocity flow on the Doppler spectrum. The PV colour Doppler alternates red and blue. (D) PV thrombosis. The PV is dilated (arrows) and filled with thrombus. A collateral vessel is seen anterior to this—not to be confused with 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.
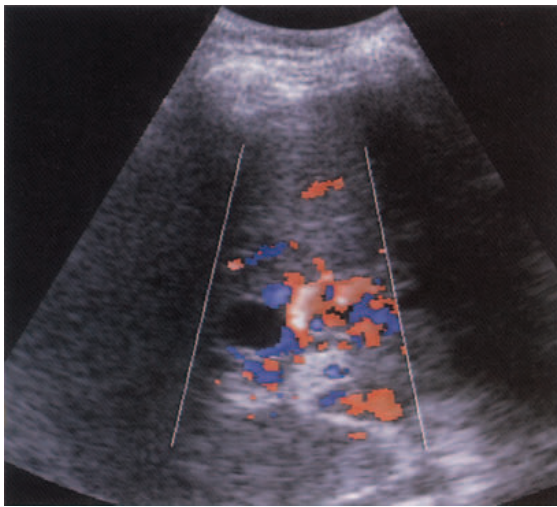
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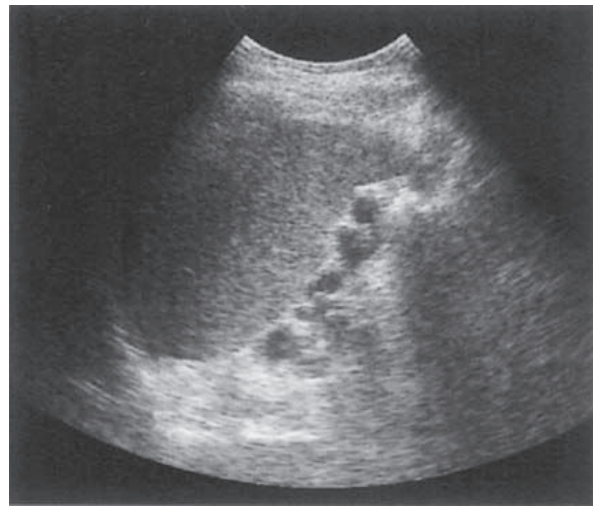
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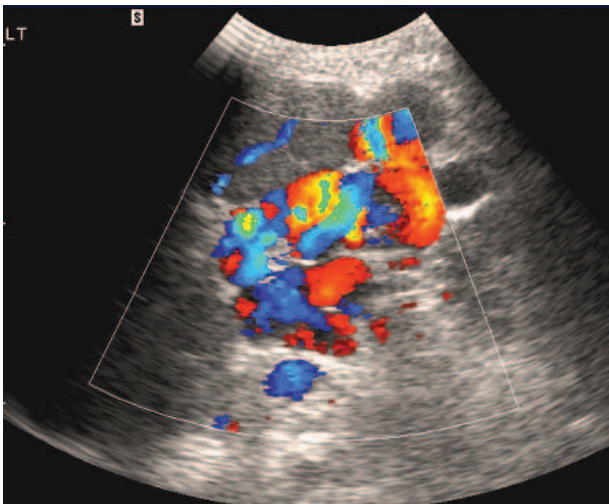
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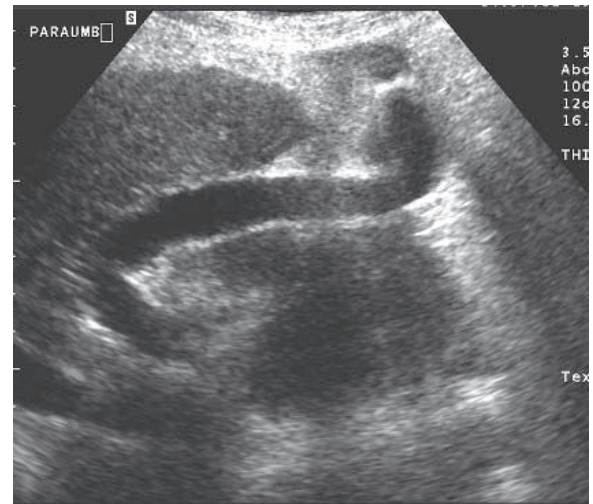
D

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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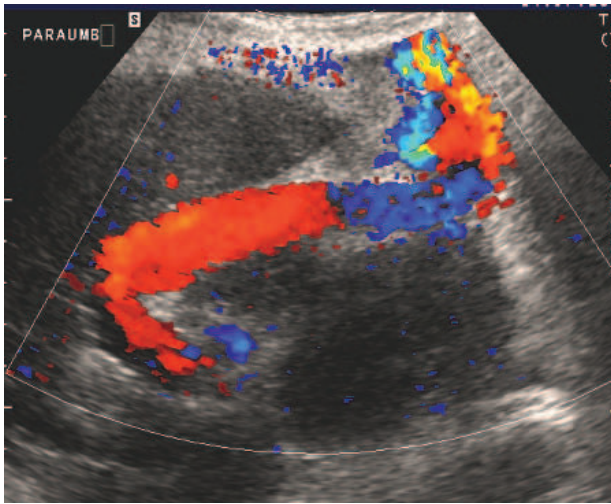
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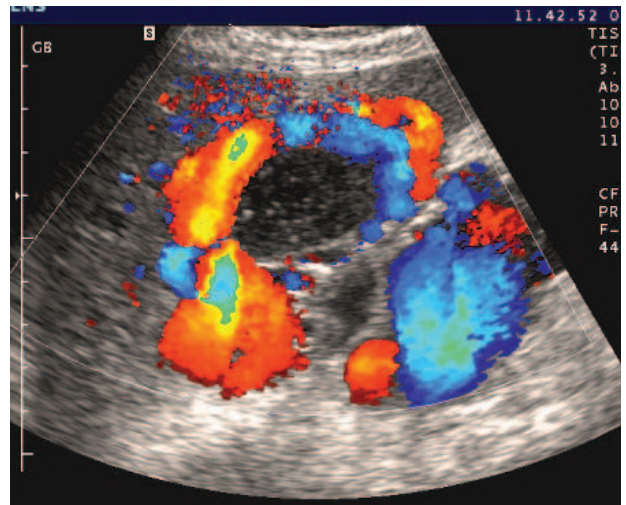
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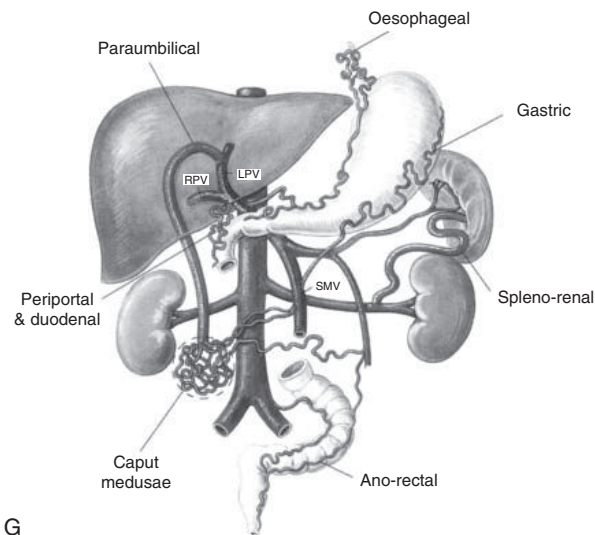
G

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).
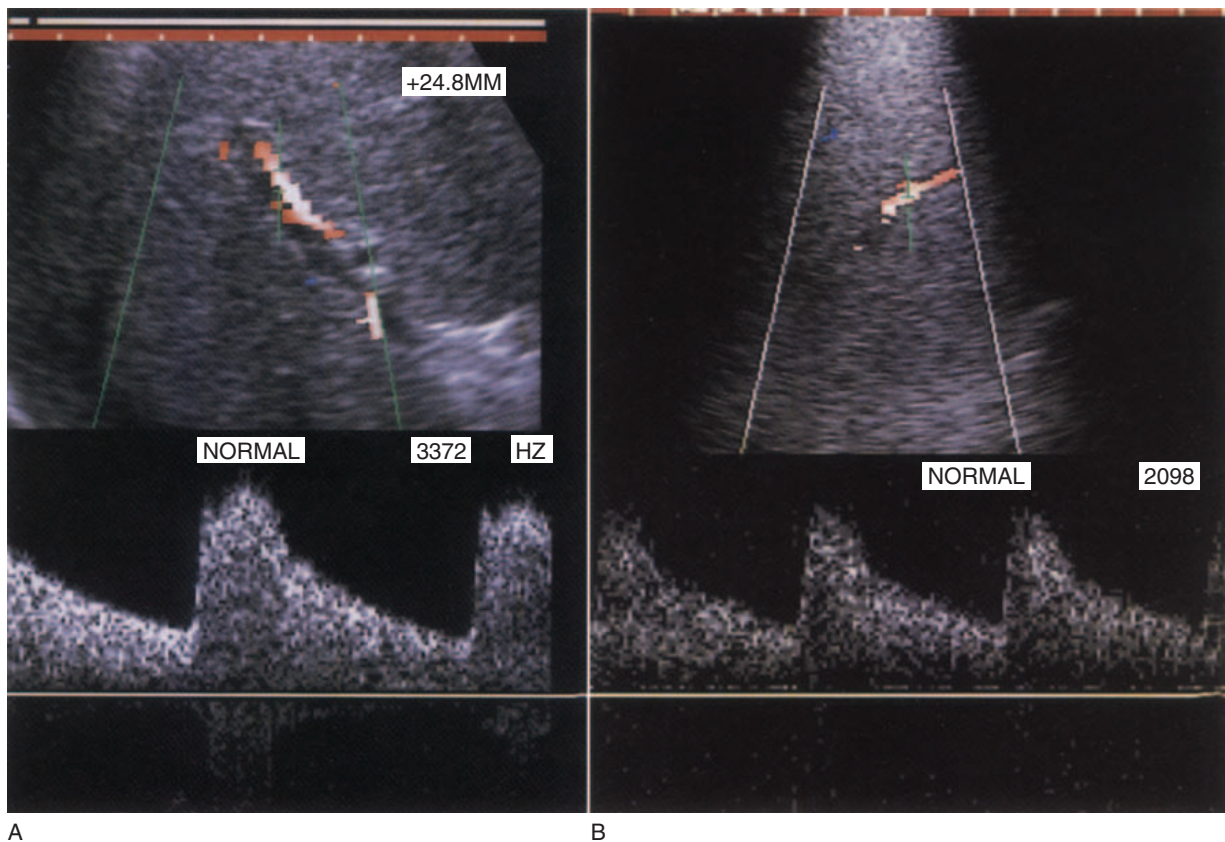

Figure 4.23 (A) Vigorous, high-velocity middle hepatic artery (MHA) flow in the presence of portal vein thrombosis. (B) Arterial flow is also readily demonstrated in the peripheral intrahepatic arteries.

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 haematemesis. 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]
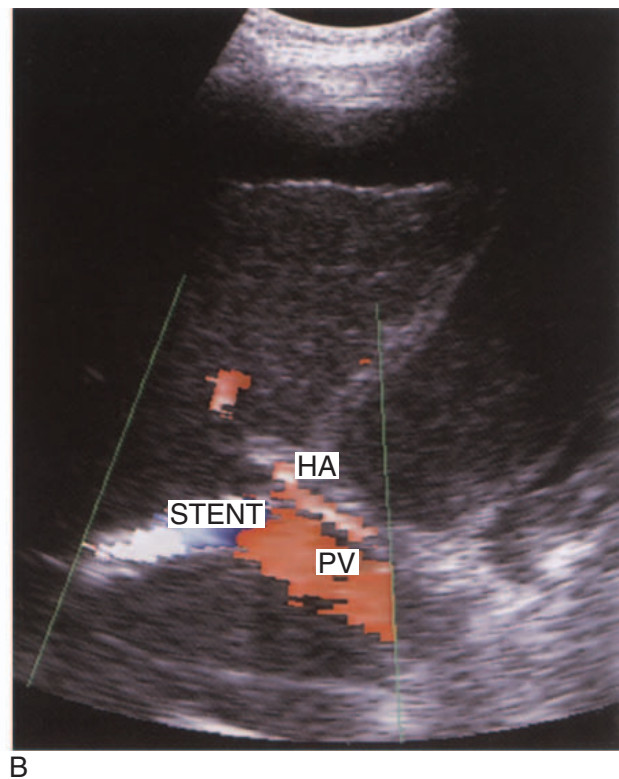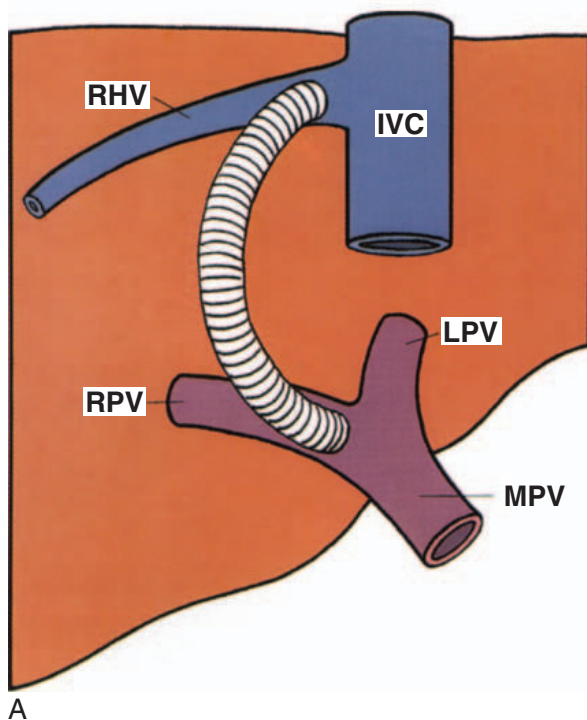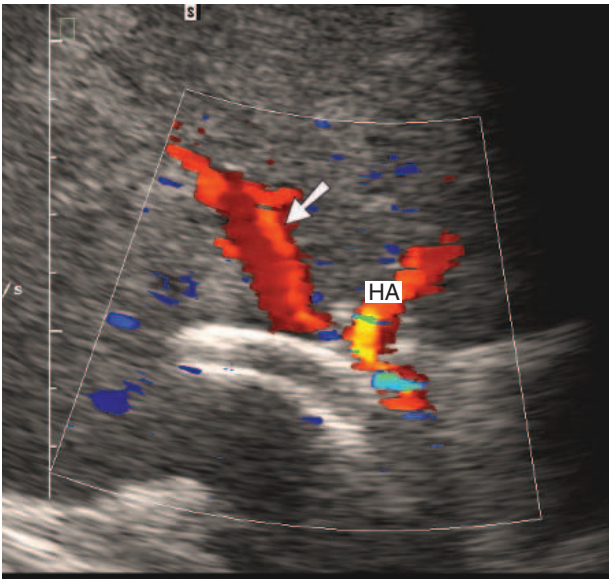

Figure 4.24   (A) Transjugular intrahepatic portosystemic shunt (TIPS). (B) TIPS shunt in a patient with severe portal hypertension. The higher-velocity MHA is seen anterior to the shunt, which demonstrates flow from right to left of the image.

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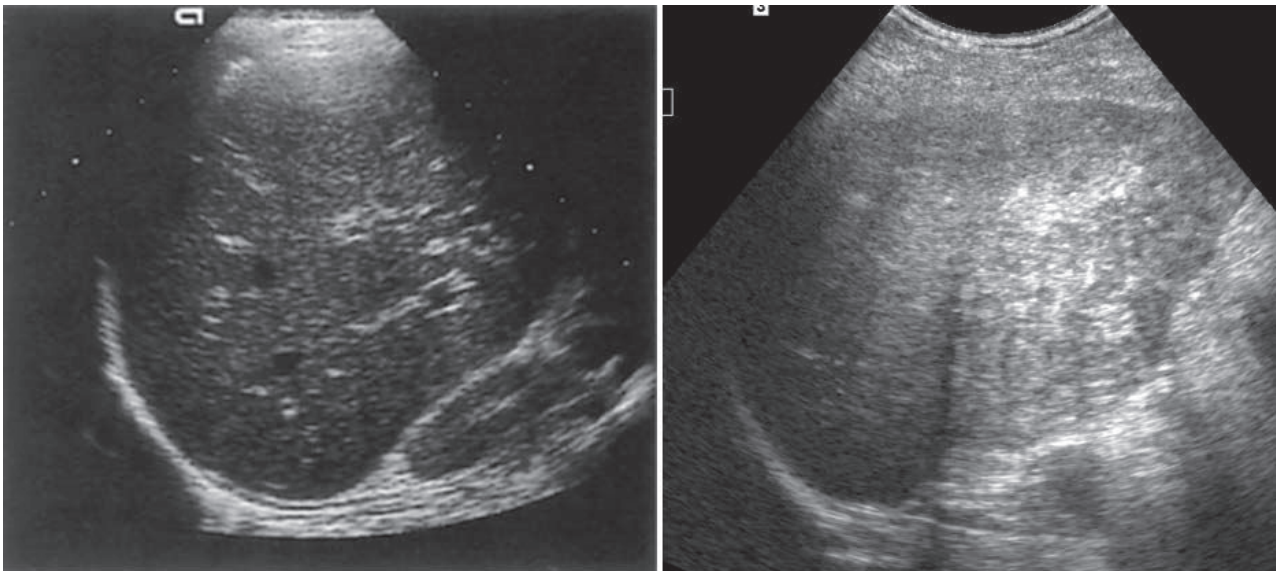
Figure 4.25 (A) Subtle changes of oedema in acute hepatitis: the liver is hypoechoic compared with the right kidney, mildly enlarged and has prominent portal tracts. (B) Chronic hepatitis and cirrhosis, demonstrating a coarse-textured, nodular liver.
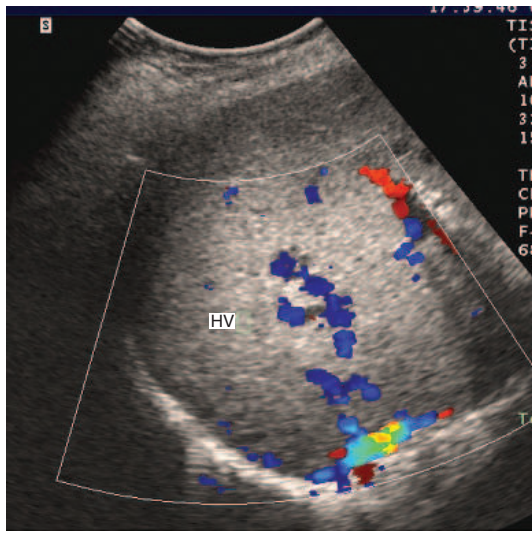
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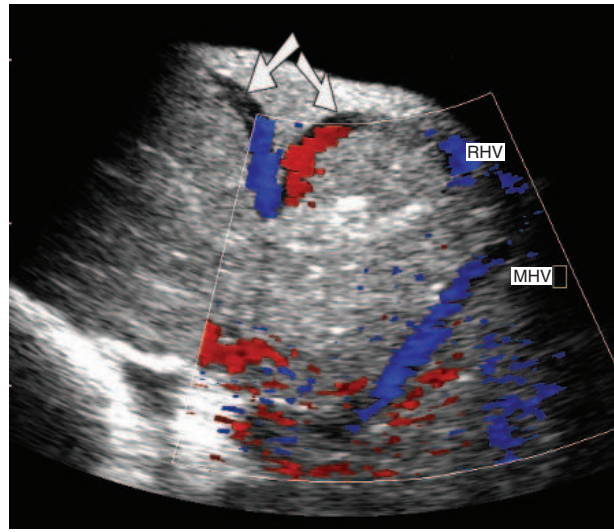
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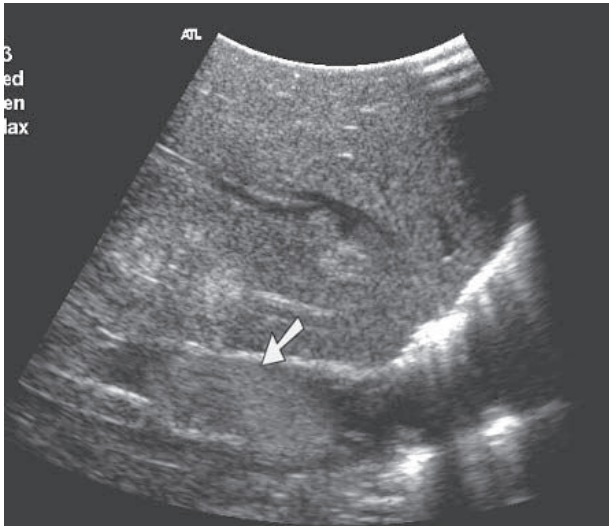
C

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
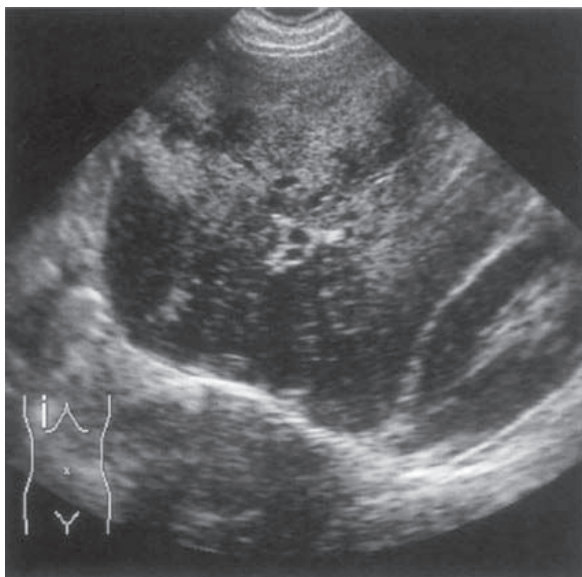

Figure 4.27   Marked changes in the liver of an adult patient with cystic fibrosis.

thrombosis (see above). Changes of fibrosis can also be seen in the pancreas.

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.
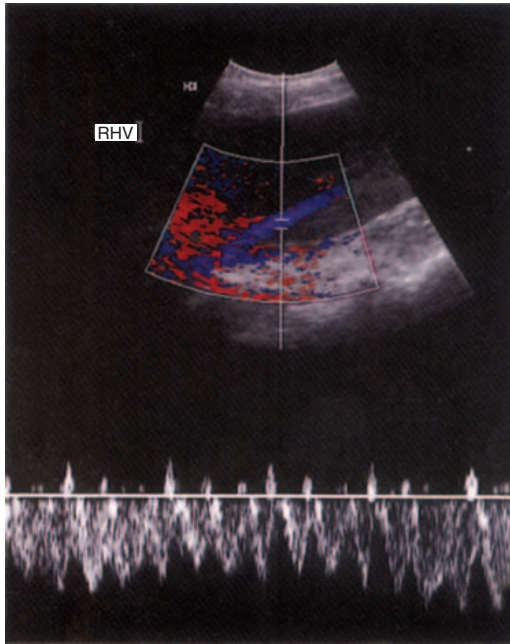
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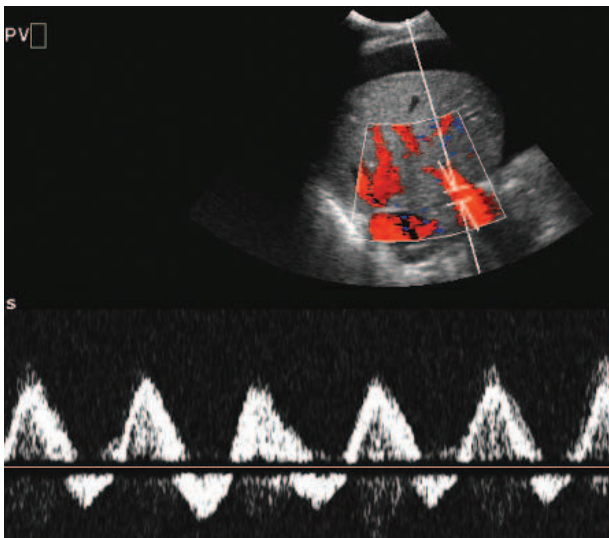
B

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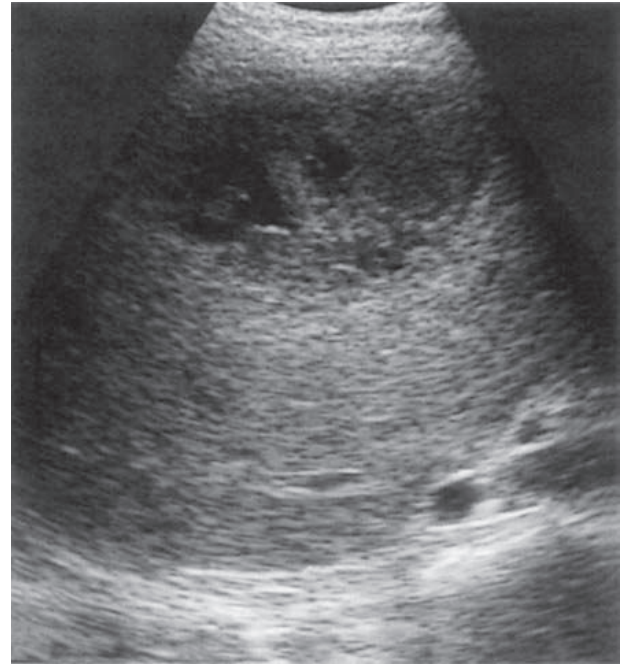
Causes of changes in liver reflectivity are listed in Table 4.5. Causes of free intraperitoneal fluid are listed in Table 4.6.

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.
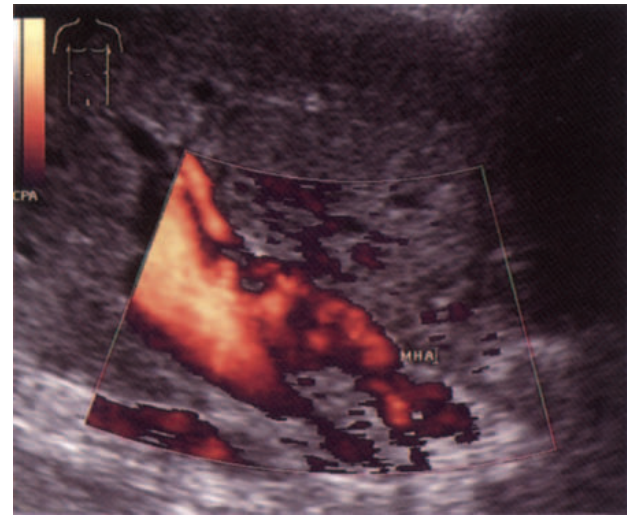
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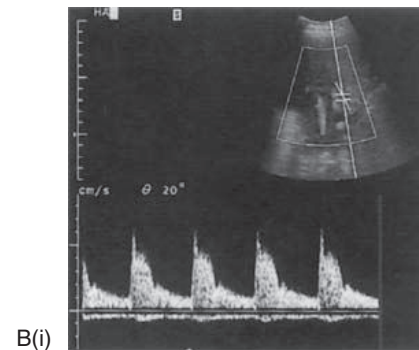
B(i)
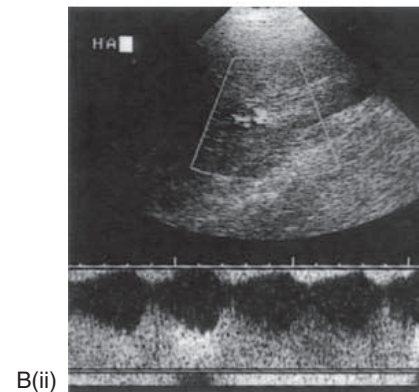
B(ii)

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
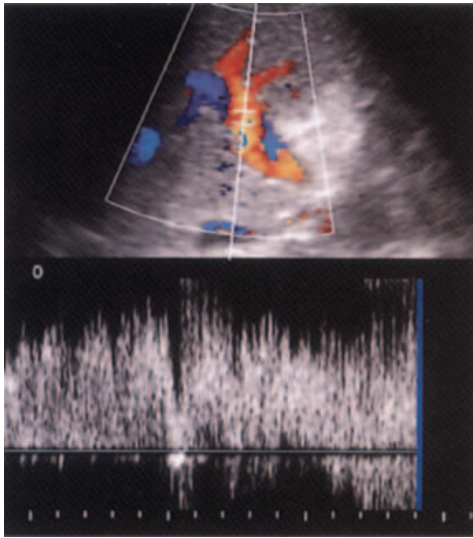
Figure 4.31   (A) The portal vein in a liver transplant demonstrates a very turbulent waveform because of the surgical anastomosis. This is not usually a significant finding. (B) MPV stenosis. A high-velocity jet is seen through the stenosis (arrow) at the site of the anastomosis. The spectral Doppler waveform exceeded the Nyquist limit at this point.
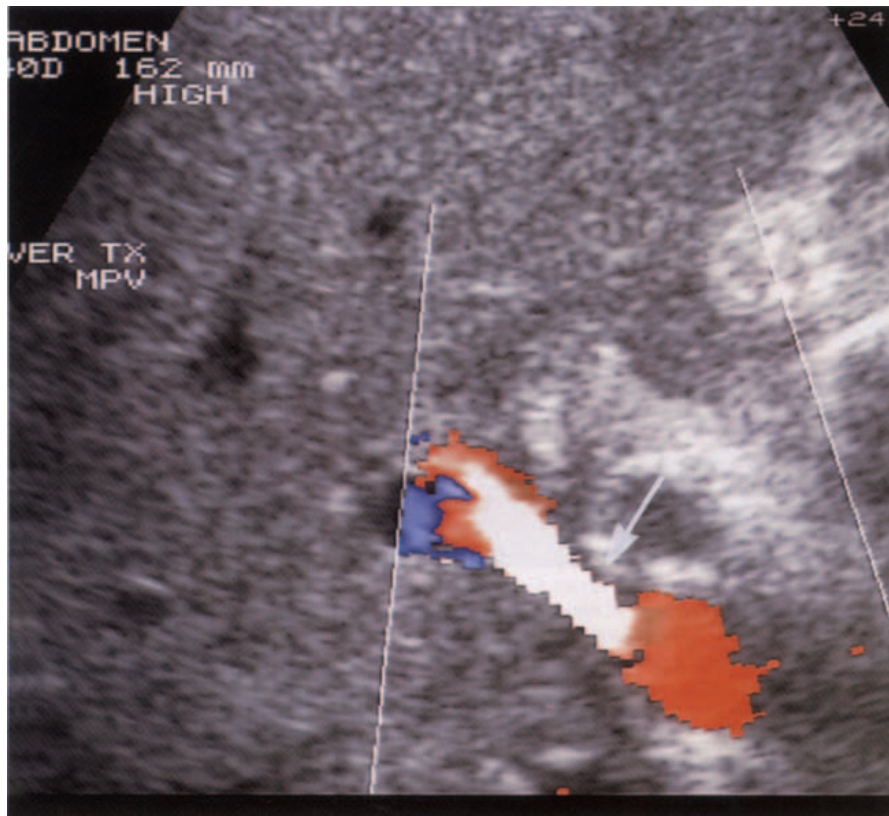
near-perpendicular angle of the IVC to the beam it is difficult to assess blood flow velocity in the IVC. Power Doppler is helpful in confirming patency in technically difficult cases as it is angle-independent. Thrombosis in the IVC is a relatively rare compli-

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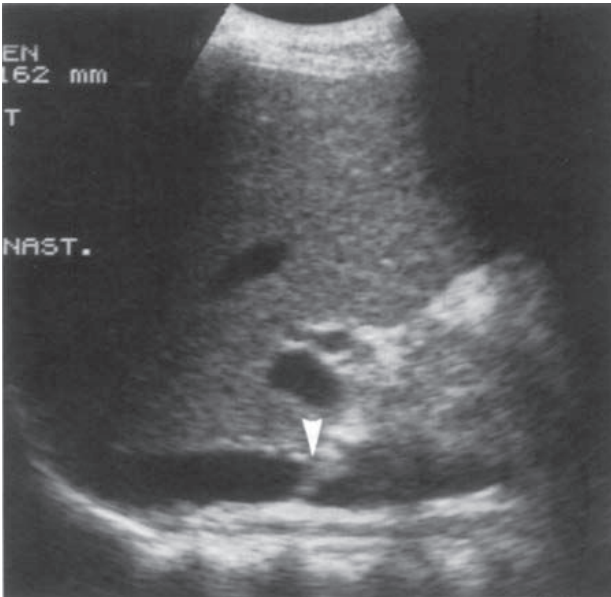
Figure 4.32 The site of anastomosis in the IVC in a liver transplant.

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 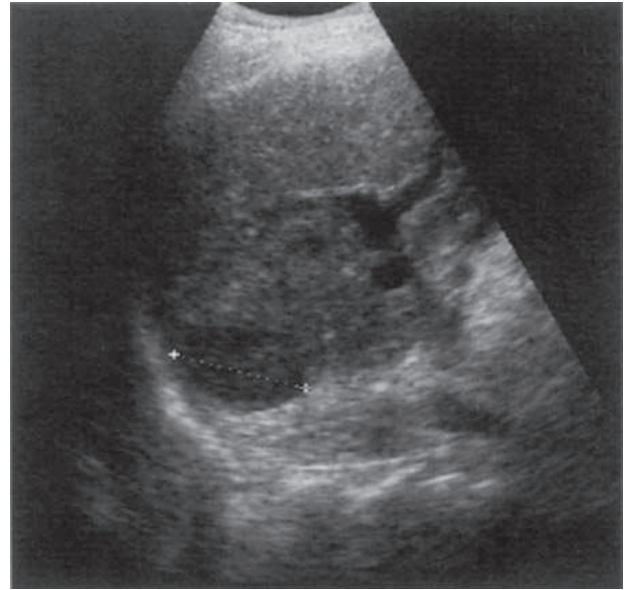tissue. Other causes of focal lesions in the early postoper-ative period may be due to infarction and are associated with interruption of the arterial supply. These can be hyper- or hypoechoic, have well-defined borders and do not exert a mass effect (Fig. 4.33).

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
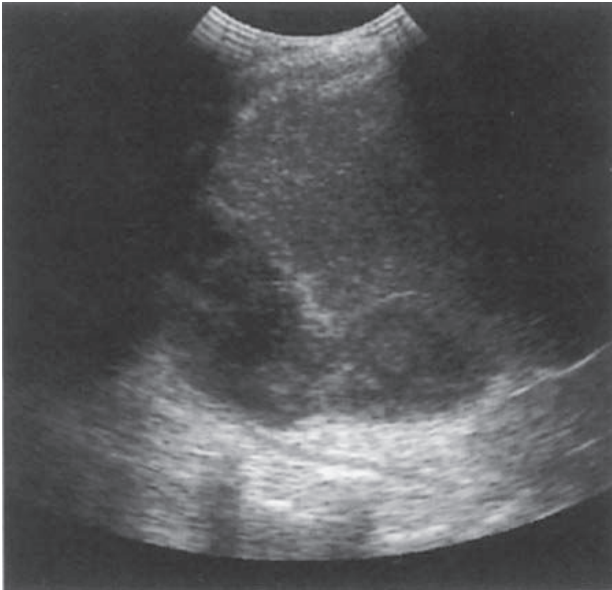
Figure 4.34   Subphrenic haematoma post-transplant.

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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