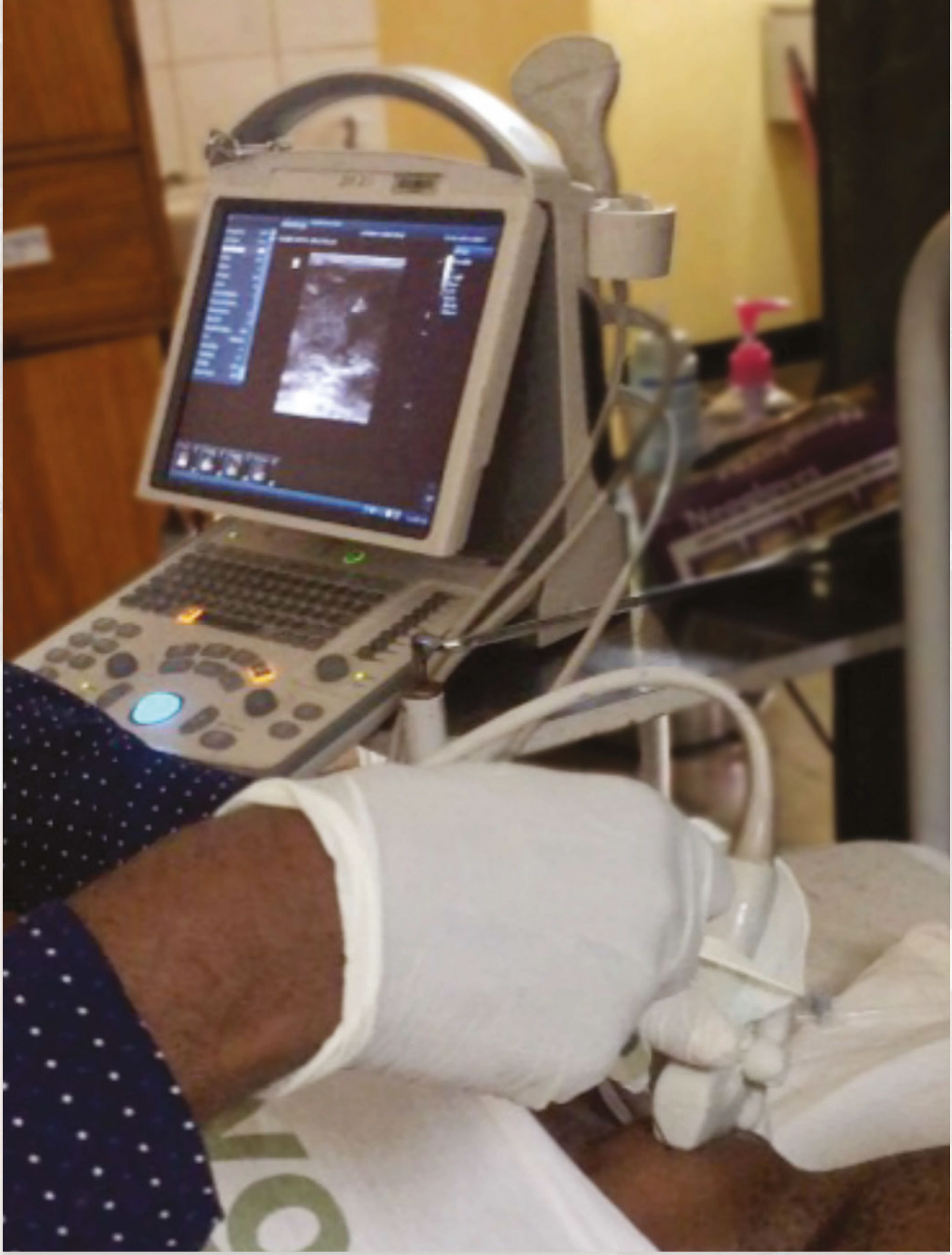


LIGHTHOUSE

ULTRASOUND MANUAL FOR HIV AND TB CLINICIANS

Tom Heller, MD

Lighthouse Ultrasound Manual for HIV and TB Clinicians

Tom Heller, MD
Specialist in Internal Medicine and Infectious Diseases (Germany)
Clinical Advisor
Lighthouse Clinic
Lilongwe, Malawi

To my brother Jan
who taught me that a book must be relevant to be read,

and

to Boniface, Crust, Davis, Eric, Joe, Kelvin, Lydia, Sunshine, Tapiwa, Zachalia, and all the other Lighthouse clinicians who made me believe that ultrasound and this book could be relevant to them.

You only see what you know.
—Johann Wolfgang von Goethe

Disclaimer
Every attempt has been made to ensure that the information in this casebook is accurate and correct. The author and publishers accept no responsibility for any loss or damage that may arise out of the reliance of any person upon any of the information provided in the book, nor is responsibility accepted for any loss or damage sustained as a result of the use of the information contained herein.

When in doubt, seek the assistance of a more senior colleague, or, when further information is required concerning drug indications or dosage, consult the National Drug Formulary, the current pharmaceutical package inserts, or the relevant pharmaceutical company.

Table of Contents

Acknowledgements

The idea to compile this manual was born out of the need at Lighthouse, which operates five large referral-level HIV/TB clinics with over 30 clinicians in different geographical areas, to have a summary and a reference for those who have embraced FASH—both as part of their routine and as part of the service they want to render to our patients. I would like to thank all those Lighthouse clinicians for their hard work, their commitment to 'save a few clients', and for their openness to embrace a new technology—even when it is certainly more work in the beginning.

The content of the manual grew over the years from the many practical ultrasound trainings we held in various 'tropical' settings in Africa, Asia, America, and Europe. These were conducted not just for clinical officers, but also for medical registrars, medical students, and radiographers. In the courses, we always had two goals: to cover clinically relevant pathologies and techniques that can make a difference to patients in the tropical setting, and to keep it simple enough to be manageable.

I have learned from so many colleagues during these courses and projects—Drs Enrico Brunetti (Pavia, Italy), Francesca Tamarozzi (Rome, Italy), Liz Joekes (Liverpool, UK), Ted Kuhn and Dan Kaminstein (Georgia, USA), Benno Kreuls (Hamburg, Germany), Sabine Belard (Berlin, Germany), Mischa Huson (Amstredam, Netherlands), and Danny Kahn (Los Angeles, USA), plus anyone I have forgotten to mention. I'd like to thank them—and all the many course participants—for their enthusiasm.

Thanks to Dr Francesco Taccari (OC Malattie Infettive, Fondazione Policlinico Universitario A. Gemelli IRCCS, Rome, Italy) for his image material on peritoneal tuberculosis, and for many interesting cases and discussions.

I'd further like to thank Dr Sagar 'Arnie' Shah (Department of Internal Medicine, UCLA Medical Center) and Drs Shoaled Rezaee and Daniel Palasz (Department of Emergency Medicine, Medical College of Georgia, Augusta University) for reading through this text, and improving and correcting language and content. The improvements are to their credit; the remaining faults are mine.

As usual, I would like to thank Kevin J. O'Conner from Seattle, with whom I have worked on various similar projects. His great work on the artwork, editing, layout, and typesetting of this manual cannot be overestimated—he did a tremendous job handling all my requests.

I would further like to thank I-TECH Malawi, in particular Country Director Dr Yusuf Babaye, for their support, and to acknowledge our funding organisation, PEPFAR through CDC Malawi.

Finally, I would like to thank my wife, Dr Claudia Wallrauch, for being my partner-traveler, my partner-physician, my partner-ultrasound trainer, and just the most important partner I have.

—Tom Heller, MD

Abbreviations

ACE	Angiotensin-converting enzyme
AFB	Acid-fast bacilli
AKI	Acute kidney injury
ALP	Alkaline phosphatase
ART	Antiretroviral therapy
CCC	Cholangiocarcinoma
CHF	Congestive heart failure
CURLS	Cardiac Ultrasound for Resource-Limited Setting (protocol)
CXR	Chest X-ray(s)
DILS	Diffuse infiltrative lymphocytosis syndrome
DST	Drug susceptibility testing
DVT	Deep vein thrombosis
EBV	Epstein-Barr virus
EPTB	Extrapulmonary tuberculosis
FASH	Focused Assessment with Sonagraphy for HIV/TB (protocol)
FAST	Focused Assessment with Sonography for Trauma (protocol)
FATE	Focused Assessment of Transthoracic Echo (protocol)
GGT	Gamma-glutamyl transferase
GUTB	Genitourinary tuberculosis
HCC	Hepatocellular carcinoma
INR	International normalised ratio
IRIS	Immune reconstitution inflammatory syndrome
IUP	Intrauterine pregnancy
IVC	Inferior vena cava
IVS	Intraventricular septum
KS	Kaposi sarcoma
LA	Left atrium
LAM	Lipoarabinomannan (antigen)
LN	Lymph node(s)
LV	Left ventricle, left ventricular
MAI	Mycobacterium avium intracellulare
MCD	Multricentric Castleman disease
MTB	Mycobacterium tuberculosis
NPV	Negative predictive value
NRTI	Nucleoside reverse transcriptase inhibitor
PCP	*Pneumocystis jiroveci* (*P. jiroveci*) pneumonia
PICD	Paracentesis-induced circulatory dysfunction
PLUS	Point-of-care limited ultrasound
POCUS	Point-of-care ultrasound
PPV	Positive predictive value
PTB	Pulmonary tuberculosis
PTLD	Post-tuberculosis lung disease
RHD	Rheumatic heart disease
RV	Right ventricle, right ventricular
TDL	Tuberculosis destroyed lung
TGC	Time gain compensation
TPT	Tuberculosis preventive therapy
US	Ultrasound
VZV	Varicella-zoster virus

How to Use This Book

This book is written for those who are just starting to use ultrasound—but it also contains additional material for those who have a bit more experience.

The running headers appearing in the outside margins of each page are colour-coded to indicate the complexity of the material:

Black
The introductory Chapter 1 gives an overview of the settings in which we use ultrasound, and gives some facts relevant to using ultrasound in an HIV/TB setting. Chapter 2 describes basic ultrasound physics and technology; Chapter 3, general abdominal anatomy.

Green
Chapters 4 and 5 are the most important chapters for the beginner who wants to learn to recognize ultrasound findings which are suggestive of tuberculosis. Chapter 6 teaches a simplified approach to cardiac ultrasound. The final basic topic, deep vein thrombosis, is dealt with in Chapter 7. (These are all fundamental topics. The beginner may want to ignore the boxes at the ends of Chapters 5 and 6, which contain additional facts and information that may otherwise be too much detail.)

Yellow
The rest of the book is for those who have some experience already and want to learn more. Chapters 8–13 all cover topics which are a bit more difficult; in the boxes at the end of these chapters (except Chapter 12), even more rare and difficult findings are tackled.

Red
Chapter 14 presents some basic facts about puncture and aspiration procedures; these should be used together with practical guidance by an experienced trainer.

Purple
For those who would like a bit of theory and want to think deeper about ultrasound, some ideas are found in the appendices. Appendix 1 looks at how we as clinicians interpret tests, and what the sensitivity and specificity of those tests mean for the decisions we make, such as when we are diagnosing TB. Appendix 2 gives some philosophical answers to a common argument against ultrasound—that it is 'too subjective.' We argue that, while ultrasound is certainly subjective and requires good skills and training, this is not much different from other methods we use in the practice of medicine.

1. Ultrasound in HIV/TB Settings

Since the mid-1990s, **point-of-care limited ultrasound (PLUS)** techniques have extended the utility of ultrasound beyond its well-established use in radiology departments. Although diagnostic ultrasound has been used for decades to help diagnose a variety of diseases, initially the technology was **predominantly used by radiologists** and imaging specialists. Now, clinicians from diverse specialties are using ultrasonography to **examine specific organs and disease** processes, and to **help perform relevant procedures.**

Clinicians use ultrasound scanners as a tool to aid in making diagnostic decisions, extend the scope of physical examinations, and facilitate therapeutic interventions. With PLUS, images are obtained instantaneously, in real time, allowing for direct comparison and correlation of findings with presenting signs and symptoms. Diagnostic ultrasound is a **quick, inexpensive procedure** that can be performed as often as needed, **without exposing the patient to radiation.**

The fundamental difference between these focused exams and conventional comprehensive radiology ultrasound exams is that with PLUS, the clinician seeks to answer simple, clinically relevant, **binary questions.** For example: *Is a pericardial effusion present, yes or no?*

Findings included in PLUS protocols usually need to fulfil **three criteria.** They must be:

- **prevalent in the setting** where the scan is done,
- **relevant to the immediate treatment** of the patient, and
- **easy enough to recognise** that health care workers without extensive training can identify them correctly.

Gradually, as their experience with this technology grows, clinicians will be able to use it to help answer more complex questions.

Emergency departments in Western countries benefit from PLUS because it can be performed without sending the patient to another department, and does not require consultation with a radiologist. Because of this, PLUS is of even further interest in resource-limited settings where **other imaging modalities (for example, CT and MRI) are scarce.**

However, ultrasound is a user-dependent technique, and there is a shortage of skilled sonographers in many African hospitals. The World Health Organization (WHO) recommends that physicians undergo three to six months of comprehensive ultrasound training, which includes performing 300 to 500 ultrasound examinations. However, this is a general guideline; some applications may require less training. Either way, training requirements must have enough flexibility to avoid hindering the ability of trainees to access the equipment.

Here again, the PLUS approach is relevant. Patients may not necessarily benefit from a complete exam, but PLUS can be used to detect (or rule out) many relevant findings. For this reason, it is more **important for users to be trained to detect relevant findings** with confidence than it is for them to detect each and every minor finding possible.

Nevertheless, we must understand that **PLUS is NOT meant to replace a full, detailed abdominal ultrasound** by a trained, certified sonographer or radiologist, when available. Rather, it is intended only as a technique to quickly assess patients for characteristic findings in situations where access to other diagnostic imaging modalities is limited. **PLUS extends the scope of the standard physical examination** performed by physicians using their hands, stethoscopes, and other tools; used in this way, it can open the eyes of clinicians and improve the care of patients.

The **Focused Assessment with Sonography for HIV/TB (FASH)** protocol, which forms the core of this book, has been developed to help diagnose the extrapulmonary and disseminated forms of tuberculosis (TB) that are frequently seen in patients who have the human immunodeficiency virus (HIV). This protocol was **developed in Sub-Saharan Africa,** where the prevalence of HIV and TB is high—but it also works in **other settings where these conditions are encountered.** Clinicians in TB-endemic regions of Southeast Asia, Central and South America, Europe, and the US have all found the protocol helpful.

Especially in Sub-Saharan Africa, the dual HIV/TB epidemic has produced strikingly unbalanced figures. Although only **12% of the world's population live in this region,** it is estimated that **68% of all HIV cases** (22 million) and **26% of all new TB cases** (2.3 million) occur here. The reasons for this correlation are both socioeconomic and biological. Increased immunosuppression due to HIV infection raises not only the risk of reactivating latent TB infection, but also of new and of disseminated TB infection. At the same time, the progression of HIV in the patient is accelerated by concomitant TB disease.

The main objectives of the FASH ultrasound exam are to detect **effusions** that may suggest **pleural, pericardial, or abdominal TB; enlarged abdominal lymph nodes;** and **focal lesions in the spleen,** which may suggest miliary or **disseminated TB** encountered in severely immunocompromised patients.

Although the role of ultrasound in the diagnostic algorithms of smear-negative TB has not been fully established, studies have shown that **additional TB cases can be identified by adding ultrasound** to such techniques as sputum GeneXpert testing and chest X-rays (CXR). It is therefore **included in some national TB guidelines** (as in Malawi) as an accepted diagnostic modality for TB. In addition to the skill and ability of the examiner, the prevalence of disseminated or abdominal TB among HIV patients will affect the impact and usefulness of FASH. This will depend on such factors as the prevalence of TB in the general patient population and the degree of immunosuppression at the time of presentation.

General points and principles governing the diagnosis and treatment of HIV and TB

The following is a summary of **general points and principles** (from WHO guidelines and other literature) regarding the diagnosis and treatment of HIV and TB.

- During the last 15 years, many countries have made enormous efforts to scale up access to antiretroviral therapy (ART). Established TB-control programmes have also continued their efforts. To cope with the dual challenge, the WHO recommends integrating HIV and TB treatment programmes; the two diseases can be considered as two sides of the same coin.

- The diagnosis of HIV is straightforward, thanks to highly sensitive, specific, and inexpensive antibody tests that can be performed at points of care. Health care providers should initiate HIV testing in the appropriate setting—and whenever TB infection is suspected or diagnosed, as the diseases frequently travel together.

- Antiretroviral therapy (ART) is highly effective. To improve survival rates, reduce morbidity, and lower the risk of HIV transmission, it should be started as soon as possible for all HIV patients (except those with contraindications, such as intracerebral infection), especially those with TB.

- Many HIV patients with advanced immunosuppression (including those with concomitant TB disease) should receive cotrimoxazole prophylaxis, and possibly other prophylactic treatment (antifungal and antibacterial drugs). If TB is ruled out, then TB preventive therapy (TPT) should be given.

- TB is primarily a pulmonary disease, but it can also be seen as a lymph node disease, as the lymph nodes are frequently involved. However, due to haematogenous spread with seeding in multiple sites, TB can affect any organ or system; it therefore has a variety of clinical presentations.

- TB transmission occurs through microdroplets coughed up by patients who have pulmonary TB. HIV patients are at particularly high risk of TB infection. Infection control measures within the health care setting are therefore of great importance during ultrasound exams.

- Extrapulmonary TB (EPTB) and smear-negative TB are particularly common in immunosuppressed patients. Diagnosis is often difficult; acid-fast bacilli (AFB) smears are frequently negative, and even GeneXpert testing may not produce positive results. Therefore, diagnosis usually relies on observation of clinical signs and symptoms.

- Sonography can detect findings suggestive of EPTB even if there are no CXR changes that suggest pulmonary disease. Nevertheless, many patients with EPTB may also have pulmonary disease. A CXR and sputum GeneXpert test can help support the diagnosis, and should be done whenever possible. Interpretation of an HIV patient's CXR is an important skill for anybody treating patients, especially in our low-resource setting. (Refer to the Lighthouse CXR manual for more information.)

- A wide variety of differential diagnoses of opportunistic infections and malignancies are seen among HIV patients in tropical countries, especially when they present with advanced HIV disease (defined as a CD4 count below 200 cells/mL). Some of these diseases present with signs and symptoms similar to those of TB; therefore, Kaposi sarcoma, lymphoma, and other systemic infections should be considered as possible differential diagnoses.

- TB is the most frequent cause of death among HIV patients in African hospitals, found in a third to half of all cases. It is disseminated in about 80% of cases, often going undiagnosed before death. Therefore, be careful about ruling out TB—when in doubt, remember that it would be better to treat one patient too many than to treat one too few!

- The established guidelines that TB treatment has followed for many years are still valid. We consider HIV patients to fall within these guidelines, so they should be treated using the same rifampicin-based regimens.

- Multidrug-resistant TB (MDR-TB) has become an increasingly prevalent problem in many parts of the world. It should be treated according to the results of genotypic or phenotypic drug susceptibility testing (DST). In patients with EPTB, biopsy material is often needed for culture and DST. This can be obtained through ultrasound-guided aspiration.

- Immune reconstitution inflammatory syndrome (IRIS) is often seen when HIV patients with TB or other opportunistic infections start on ART. Therefore, correct timing when administering medication is essential to balancing possible morbidity and mortality due to IRIS with that caused by delaying ART and the resulting prolonged immunosuppression. We must consider changes caused by IRIS when following lesions detected with ultrasound over time, as the lesions may initially increase in size with treatment.

2. Technical Aspects of Ultrasound

Physics

Basic knowledge of the physics of ultrasound will help us to understand how the image on the screen is generated. The piezoelectric **crystals** located at the core of the transducer are the most essential part of the ultrasound equipment, transforming **electrical energy** into **sound energy** (vibrations), and vice versa.

The crystals send short ultrasound pulses into the body. Sound travels through some tissues quickly and through others more slowly; at interfaces between different tissues, some of the **ultrasound energy is reflected back as echoes.** (The rest is lost through attenuation—absorption, refraction, and diffusion of the sound waves as they interact with tissue, fluids, and gas.) The crystals also detect these echoes, which are converted back into electrical current for display on the screen. The location of each image point on the screen is determined by the **time elapsed between transmission of the signal and return of the echo** (Figure 2.1); the brightness of each point is based on the intensity of the echoes received. After each pulse-echo cycle, a new cycle begins. The amount of **energy required for diagnostic ultrasound is very low;** thus, it has no side effects when used in point-of-care ultrasound (POCUS) indications.

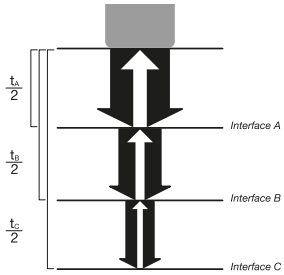


Figure 2.1. Depiction of the sound beam travelling from the transducer to the interface and back after reflection. The time until the sound returns depends on the distance to the reflecting interface—which means the travel time can be measured and converted into a distance.

Ultrasound machines can operate in different modes. The mode that produces the well-known two-dimensional, black-and-white screen image is known as **B-mode** (brightness mode). **Doppler mode** enables visualisation of blood flow using a Doppler signal; blood flowing away from the transducer appears blue, and **blood flowing towards the transducer appears red.** This is known as **colour flow mapping.** Some machines have a **power mode,** which displays any blood flow as a golden-brown colour. Although power mode cannot show the direction of blood flow, its advantage is that it can detect very minimal flow. In **M-mode** (motion mode), echoes received from different depths are displayed along one axis, with time displayed along the other. This allows the examiner to assess the motion of the interfaces over time; it is particularly helpful for heart and lung ultrasound.

To describe and interpret ultrasound images, the brightness of the anatomical or pathological structures is compared with that of surrounding tissues (such as normal liver tissue). If the structure generates a stronger echo signal (appears brighter) it is referred to as **hyperechoic** or **echogenic** (Figure 2.2); if it generates a weaker echo signal (appears darker), it is **hypoechoic** or **echo-poor** (Figure 2.3). A structure that appears completely black (meaning no echo signal was reflected back to the transducer) is described as **anechoic** or **echo-free** (Figure 2.4). A structure that is similar in brightness to the surrounding tissue is referred to as **isoechoic** (Figure 2.5). Isoechoic structures will be difficult to distinguish from the surrounding tissues, making them easy to miss.

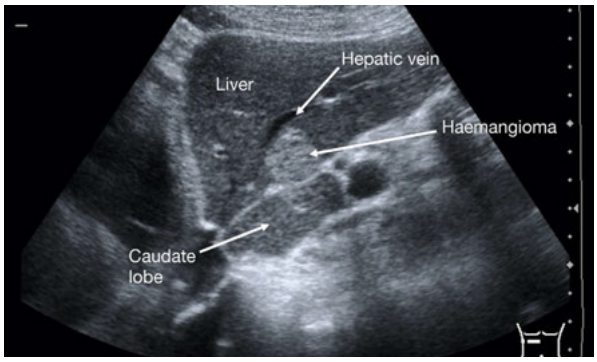


Figure 2.2. Hyperechoic lesion (haemangioma) in normal liver tissue, between the caudate lobe and a hepatic vein.

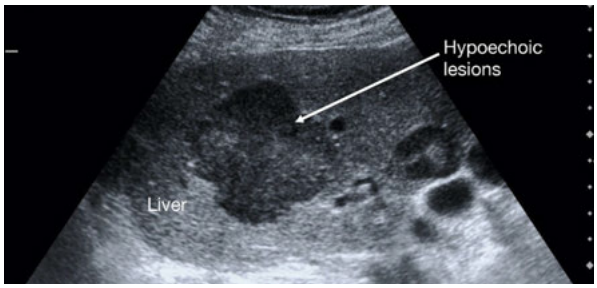


Figure 2.3. Large, well-defined hypoechoic lesion (lymphoma) in the liver.

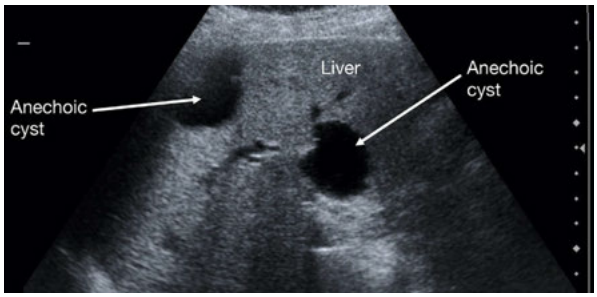


Figure 2.4. Two anechoic, well-defined lesions (cysts) in the liver. Both cysts show increased echogenicity in the tissue behind the lesion due to increased acoustic energy reaching these areas (dorsal acoustic enhancement).

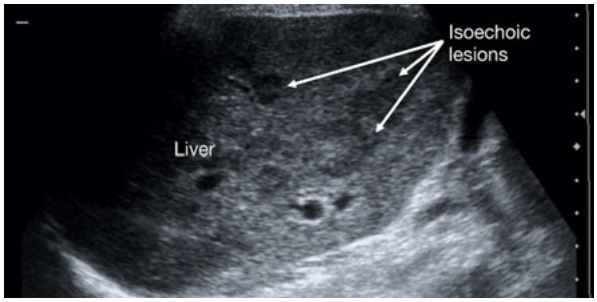


Figure 2.5. Multiple isoechoic lesions (metastases) in the liver. The lesions are minimally darker than the surrounding tissue and disturb the architecture—this is what makes them visible.

Air and **gas** in the lung or bowel and **calcified bones** will impede the transmission of sound. Consequently, the image that results from the interaction of sound waves with gas or bone interfaces will show strong artefacts. The presence of shadowing can yield important diagnostic information, but will also obscure any structures behind the gas or bone. The sonographer should be aware of the following artefacts, which are frequently encountered during ultrasound examinations:

- **Acoustic dorsal enhancement** can appear behind anechoic structures (like cysts) (Figure 2.4).

Because of the greater magnitude of acoustic energy remaining, this tissue appears hyperechoic, or brighter than the surrounding tissue.

- **Acoustic shadows** can be created by nearly complete absorption or reflection of the ultrasound beam by a structure. If most of the beam is absorbed and reverberations are absent (as with bone or calcified gallstones), the result is an **anechoic black ('clean') shadow** (Figure 2.6). If the beam is mostly reflected (as with gas), the numerous reflections and reverberations cause the area below the structure to appear irregular and greyish; this is referred to as **'dirty shadows'** (Figure 2.7).

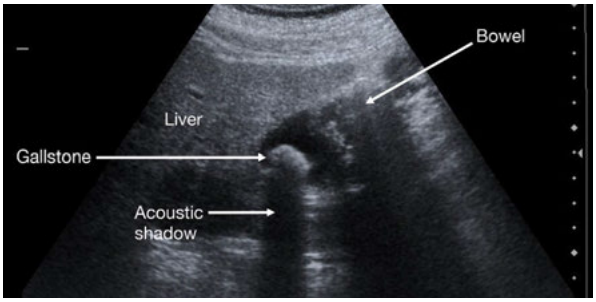


Figure 2.6. Bright echogenic gallstone with dorsal acoustic shadowing. Gas at the tip of the bowel throws a softer, 'dirty' shadow.

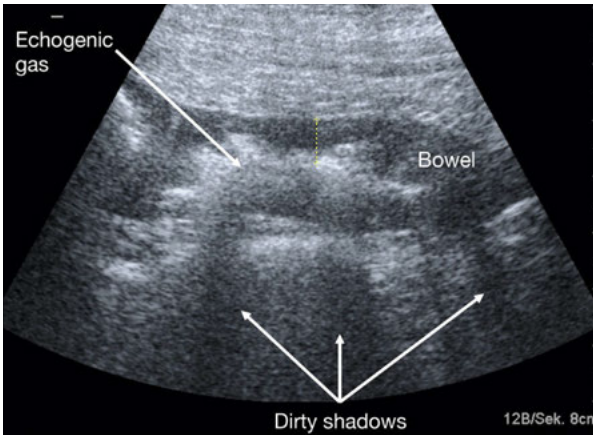


Figure 2.7. Thickened bowel with (echogenic) gas in the lumen. The gas creates a dirty shadow.

- **Edge shadowing** can appear distal to the lateral margins of fluid-filled curved structures (such as the gall bladder, bladder, cyst, kidney, or adrenal glands). The sound waves penetrating the edge of a structure may be refracted, producing a **linear or triangular anechoic zone** below the lateral edges of the structure.
- **Reverberation artefacts** (Figure 2.8) develop when echoes reflected from a tissue interface within the body are reflected back into the body again by the surface of the ultrasound transducer. The echo is then reflected for a second time at the interface of its origin. Because of the extra reflections, twice the time is needed for this signal to be recorded. This can happen several times; the computer thus generates images in multiples of the distance between the probe and the actual structure. The brightness of this artefact becomes weaker with each reflection. This happens most often with strong reflecting interfaces like the pleura; in this case, these artefacts are called **A-lines.**
- **Proximal noise** (Figure 2.9) is a result of multiple reverberation echoes in the proximal parts of

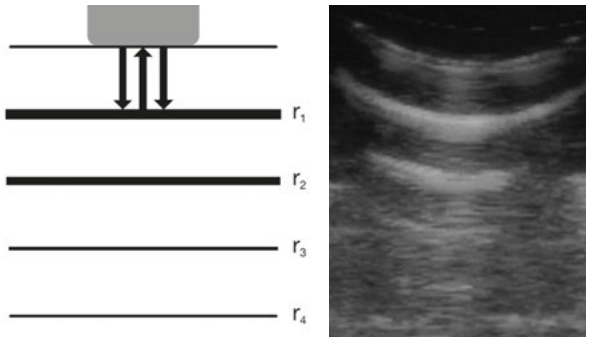


Figure 2.8. Reverberation echoes: (L) The sound beam travels between probe and strong reflector r_1. Artefact images r_2, r_3, etc. are falsely generated. (R) How this appears in an ultrasound.

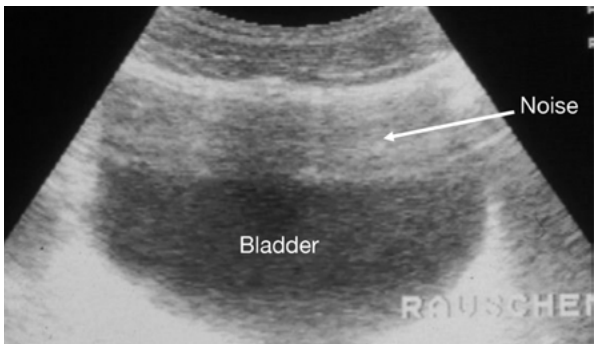


Figure 2.9. Proximal noise close to the transducer in the bladder.

cystic organs (for example, the urinary bladder and gall bladder). This artefact can be mistaken for solid tissue.

Components and controls

In recent years, the cost of ultrasound equipment has dropped substantially; affordable, robust, and portable scanners (Figure 2.10) have become readily available. High-end ultrasound machines provide numerous additional options, such as Doppler and colour flow technologies, image post-processing software solutions, and other support functions for the user. Although some of these are helpful in certain conditions, they are not essential in most POCUS applications.

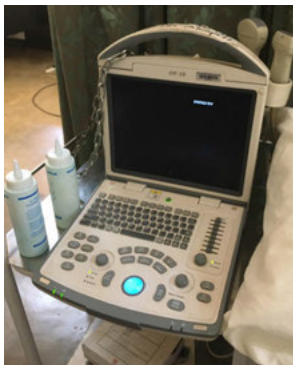


Figure 2.10. Portable ultrasound.

In principle, all ultrasound machines have the same basic functions. The three basic components are the **ultrasound probe,** which sends and receives the ultrasound signal; the **computer,** which performs the necessary calculations; and the **screen and keyboard,** which displays the image and enables the user to interact with the system.

Let's look at these in reverse order:

The **screen** (or **display**) is where the ultrasound image is displayed. The size of the image depends on the size of the screen. Some modern machines may have displays that are very small—about the size of a cell phone. Though these small screens are not very good for displaying detailed images, they nonetheless allow maximum portability—that is, you can use them in the ward, at bedside, or even outside the hospital in emergency settings. Most ultrasound displays are the size of

a normal computer screen. In addition to the ultrasound image, they often display information like patient name (if entered), date, time, and probe settings.

It is important that we optimise our view of the screen. Because the ultrasound image is dark, it is **ideal to be in a darkened room**—the darker the better—so that our eyes will optimally adapt to dark images. Bright light shining on the screen can create reflections, making it more difficult to see details. Therefore, **if you film the screen** with a smart phone, make sure the **flash is turned off.**

The **computer** (or **CPU**), where the calculations are performed, is obviously located inside the machine. Its size depends on the age of the machine; some older machines are as large as pieces of furniture, whereas modern equipment can be as small as a laptop, or even a smart phone. We interact with the computer through a **user interface;** this can be a **screen and keyboard** (sometimes with a mouse) or a **touch screen.** Either way, it is important to locate a few of the machine's relevant buttons to help us optimise and adapt the image.

Make sure you are able to find at least the following functions on your machine:

- **On/Off button** – This is essential (for obvious reasons); it turns the machine on and off.
- **Freeze** – This freezes the image so that measurements can be done, structures examined in more detail, or printouts or photos made.
- **Gain** – This controls amplification of the returning echoes; increasing the gain increases the amplification. (You can think of it like a hearing aid, which can be turned up to make it more sensitive; increasing the gain will make the ultrasound image brighter. However, as gain is increased, artefacts may also become more pronounced.) Most machines offer two or more gain level control options: **near and far field gain,** which controls the brightness of the top and bottom halves of the image, respectively; and **multilevel time gain compensation (TGC),** which allows adjustment of gain at multiple depth levels in the image (for example, to optimise visibility of pelvic organs when scanning through the bladder).
- **Depth** – This changes the maximum scan depth displayed on the screen. For example, the view of smaller structures in the near field can be enlarged by reducing the scan depth. To visualise deeper structures, scan depth can be increased.
- **Focus** – This electronically manipulates the ultrasound beam to change the area of optimal resolution of the image. The focus should be set at or just below the level of the structure being examined.
- **Measurements** – It is important to measure (or at least approximate) the actual size of structures to avoid misinterpretation of results. When image size is changed (see *Depth* above), small structures may appear large on the screen, and vice versa. Measurements are usually done using the SET button and trackball.

Ultrasound probes (Figure 2.11) differ mainly in sound frequencies generated and shape. The **probe frequencies** used for diagnostic purposes range from 2 MHz (for echocardiography) to 15 MHz (for very superficial small body parts). **Higher frequencies produce better resolution** of the structures, but the depth they can penetrate is **limited**. **Lower frequencies provide better penetration**, but at **lower resolution.** For visualisation

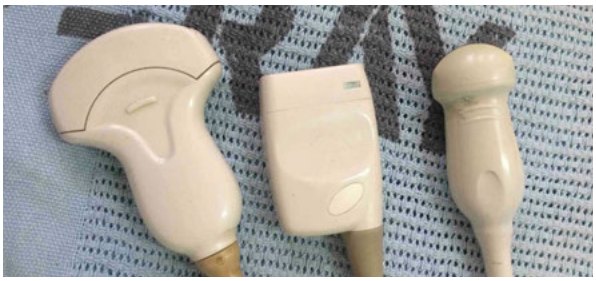


Figure 2.11. Three different probes. From left to right: Curvilinear abdominal probe, linear superficial probe, small-footprint cardiac probe.

of deeper anatomical structures (for example, the aortic lymph nodes), it is necessary to use low-frequency probes. For superficial structures (such as lymph nodes in the neck), a high-frequency transducer is preferred. Most transducers have a small marker on one side (usually a knob or ridge) to help orient the image.

The shape of the transducer used for the ultrasound procedure is determined by the structure being scanned. **Abdominal ultrasound** is usually performed using a **curvilinear probe;** for **smaller parts, linear transducers** are preferred. For **cardiac scans,** a **sector probe with a small footprint** is desired to allow the ultrasound beam to pass between the ribs. When possible, it is best to use at least two transducers to scan thoracic, abdominal, and superficial structures. Dedicated probes, such as transvaginal probes, exist for intracavitary use.

Ultrasound image orientation

Understanding the orientation of an ultrasound image and how it reflects the anatomy within the body is one of the most difficult concepts for the ultrasound novice. It is also one of the most important. Structures are commonly examined on two orthogonal planes, **transverse** and **longitudinal.**

Always ensure that the **transducer marker corresponds to the left side** of the image, and remember that in any scan plane, the left side of the image should correspond to the side of the probe with the marker.

If a **longitudinal image** (Figure 2.12 on the next page) is required, place the **transducer marker towards the patient's head.** This will project **cranial structures on the left side of the screen** and caudal structures on the right. On a correctly oriented image, the cranial parts of the liver will be projected on the left side of the screen; the caudal edge of the liver will be visible on the right.

If a **transverse image** (Figure 2.13) is required, rotate the transducer anticlockwise from the longitudinal position. In the midline, this will result in the **transducer marker pointing to the right side of the patient,** and **right-sided structures projecting on the left side of the screen.** However, in the flank (when scanning the right kidney, for example) this will result in the marker pointing to the patient's back; posterior structures will appear on the left side of the screen.

All structures located in the **near field** (close to the transducer) will appear in the **upper parts of the screen,** while deeper structures in the **far field** will appear towards the **bottom of the screen.**

Documentation and remote consultation

Findings of the ultrasound examination should always be documented. Ideally, this should be done by recording the images, but most of the time it is done by **writing a report.** Even when you do a point-of-care ultrasound, you should write a report. The name of the patient must

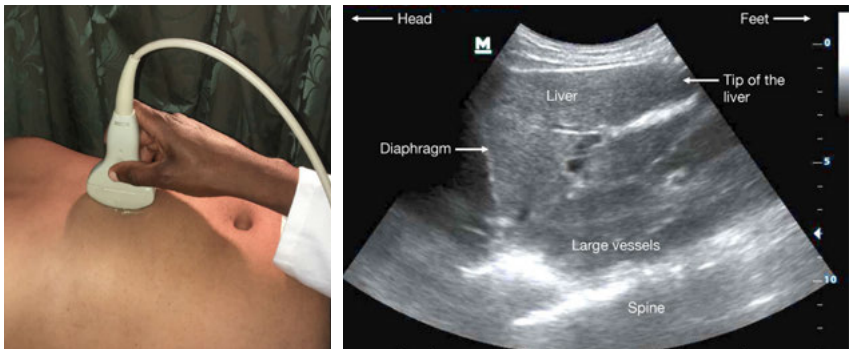


Figure 2.12.
(L) Longitudinal upper abdominal scan. (R) The liver can be seen with the tip (lower pole) of the liver pointing towards the right side of the screen. The cranial parts can be seen on the left side. In the distance the large vessels can be seen as anechoic bands and the spine as an echogenic structure. (This image can be used to calibrate your image and check that the transducer orientation is correct—thus we sometimes call it the 'position zero' of the FAST and FASH scans. If your image is unclear, twisted, or turned, it is wise—especially for the beginner—to come back to this position.)

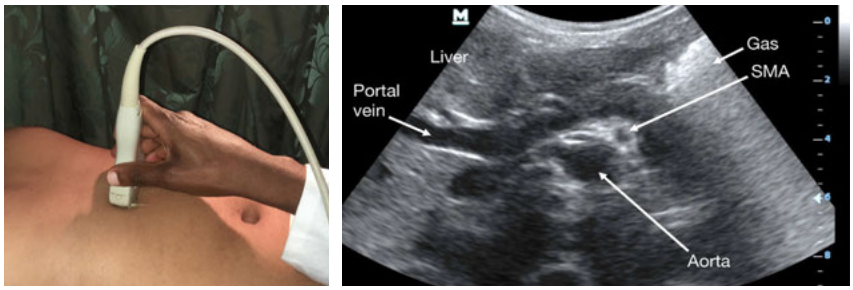


Figure 2.13.
(L) Transverse upper abdominal scan. (R) The liver (on the patient's right side) can now be seen on the left side of the screen with the portal vein. On the right side of the screen (corresponding to the patient's left side) the stomach, with gas causing dirty shadows, can be seen. The large vessels (aorta, superior mesenteric artery) are now cut perpendicular and appear as anechoic circles.

be noted, unless the report is written in the patient's health passport or file. The name of the examiner should also be included, to allow for follow-up questions. Make sure to clearly state the reason for the examination (in other words, **formulate the question that you want to answer**). **Describe what you found**—and anything you did not find due to technical reasons; this is helpful when later reading and interpreting the results. Not mentioning an organ without any explanation could be interpreted to mean that the organ was either not seen or appeared normal. So, it is important to be as precise as possible. Finally, include a summary of findings that refers to your initial clinical question.

For a **FAST/FASH exam,** the following (Figure 2.14) may suffice as a short sample report:

FASH exam. 1.1.2021:

- *No pericardial effusion – No pleural effusion.*
- *No enlarged abdominal lymph nodes visible in the epigastric region.*
- *Spleen normal size without focal lesions.*
- *No ascites visible.*

Conclusion: no sign of disseminated TB.

[Signature and name of clinician]

Figure 2.14. Example of a FAST/FASH exam summary report.

(For a more **comprehensive exam,** Figure 2.15 may serve as a template.)

Bedside ultrasound exam 1.1.2021:

- *Liver: liver normal in size and form, liver tissue smooth, no focal lesions visible, liver surface smooth, hepatic veins not dilated, portal veins branching normal, extra- and intrahepatic bile ducts not dilated.*
- *Gall bladder: thin wall, no stones visible.*
- *Spleen: size 11 x 4 cm, homogenous tissue.*
- *Pancreas: normal organ size, no tumour or enlargement visible.*
- *Kidneys: normal in size and echogenicity, no hydronephrosis.*
- *Large vessels not enlarged, no enlarged lymph nodes in periportal or para-aortic area.*
- *No pleural or pericardial effusion, no ascites.*

Conclusion: normal abdominal ultrasound.

[Signature and name of clinician]

Figure 2.15. Example of a summary report from a more comprehensive exam.

Because many different findings are possible with ultrasound, it is impossible to mention them all in introductory texts like the one you are currently reading. It is, therefore, reasonable to **seek expert advice** whenever you are not sure about a finding. Most ultrasound machines can save and store clips and images so that they can be reviewed later with another person.

An efficient alternative is to send images or **video clips** of findings to someone with more experience (Figure 2.16). In such cases, make sure that the other person knows the clinical picture and understands what you are scanning and why. It is best to **take short clips** that show the finding while the transducer is **moving slowly and steadily.** Still images are less suitable for remote assessment, as video clips give the reviewer better information on how the finding relates to surrounding structures. If you record the screen using a smartphone, it is best to have one person scanning and one person filming. The person operating the camera must ensure that the **screen is in focus,** the **image is maximised (ideally, filling the screen),** and **no reflections** that would make the clip difficult to assess are visible (and no flash!). It is better to send several short clips rather than one long clip, as data transmission may be a problem with longer clips. While filming, remember that your voice is usually being recorded as well, so you can use the audio to describe what you are doing and ask questions. WhatsApp is a popular option for sharing ultrasound clips—though image quality may deteriorate during transmission, it often facilitates timely feedback on the findings.

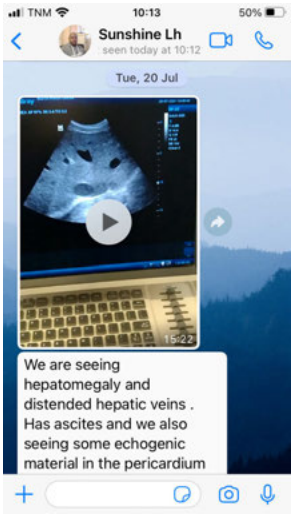


Figure 2.16. Images or better videos of the screen can be shared via WhatsApp or other messaging software to get second opinions and expert advice.

3. Basic Ultrasound Anatomy of the Abdomen

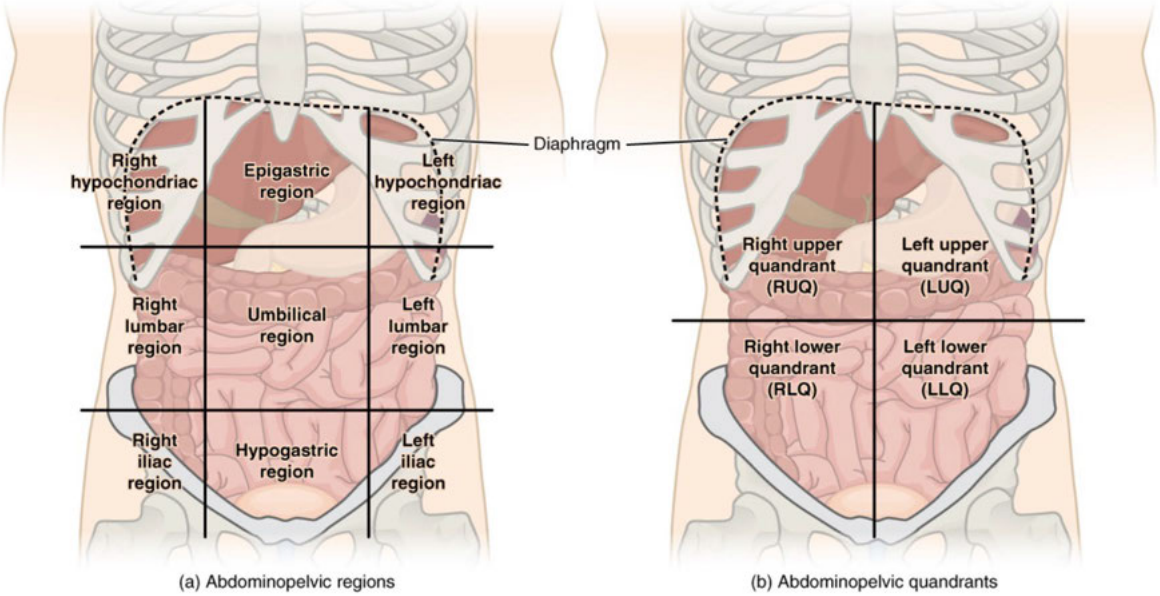
The abdomen (commonly called the belly, tummy, or stomach) is the part of the body between the thorax and the pelvis. The **abdominal cavity** is surrounded and protected by a thin membrane (the **parietal peritoneum**), a tough layer of tissue (the fascia), the abdominal and back muscles, and finally, the skin. The **diaphragm** is the upper limit of the abdomen. The **pelvic region, which forms the lower limit** of the abdomen, begins somewhere at the level of the pelvic bones.

The main part of the abdomen contains the digestive organs, including the **stomach, small and large intestines, pancreas, liver, and gall bladder.** The **kidneys** and **spleen** are located in the upper dorsal parts of the abdomen; the **bladder** and **genital organs** reside in the pelvic abdomen. All these organs are loosely held in position by connecting tissue and fat (called the **mesentery**), which allows the organs to expand and to slide against each other. Surrounding most of the organs is again a thin membrane, the **visceral peritoneum.** Many important blood vessels travel through the abdomen, including the **aorta** and its branches (the **celiac trunk** and the **superior mesenteric artery**), the **inferior vena cava**, and many smaller branches. There are also many **lymph nodes** located in the mesentery alongside these blood vessels.

For the sake of the ultrasound exam, the adjacent thoracic structures should be mentioned, as these can also be visualised. These are the **pleural cavity** and the **pericardial sac, which contains the heart.** Both are visible from the cranial part of the abdomen.

More information on the anatomy of these organs will be given in later chapters, but here we want to give an overview of which organs and structures can be found and where they are located. The abdomen can be divided into four regions (or **quadrants**) to help describe the locations of organs. These quadrants (shown in the right half of the image below) are classically described as **left upper, left lower, right upper, and right lower.** Their main use is to describe the location of pain.

The **nine regions** shown in the left half of the image below are a more helpful guide for the sonographer. In the **right hypochondriac region,** it is important to examine the right pleural cavity, the liver, the biliary system, and the gall bladder. In the **epigastric region,** the pancreas and the large vessels can be found. (Enlarged periportal and para-aortal lymph nodes can also be seen here.) The heart is visible from the epigastric angle if the transducer is tilted upwards. In the **left hypochondriac region,** we will find the spleen and the left pleura. In the **lumbar regions**, we find the kidneys. The **hypogastric region** serves as a window to the pelvis and the organs located there. In the **iliac regions** and the **umbilical region,** we will mainly find intestines—which often contain gas, making assessment of the ultrasound more difficult.



Source: http://cnx.org/contents/17e4eea8-a005-45af-b835-f756a014cd48@3

4. Ultrasound of Effusions and Free Fluid: The FAST Protocol

Introduction

Each year, injuries account for nearly one in 10 deaths worldwide. Among young adult men, road traffic injuries are the leading cause of trauma-related deaths, most of them in developing countries. All **trauma victims,** regardless of cause, may suffer from **internal bleeding,** which can lead to haemorrhagic shock and death if not diagnosed and treated in time. Both intra-abdominal and intrathoracic bleeding are concerns, as body cavities allow large-volume haemorrhaging. For these reasons, the **Focused Assessment with Sonography for Trauma (FAST)**, a rapid **point-of-care ultrasound protocol** for patients with suspected internal bleeding after trauma, was developed more than 20 years ago.

The purpose of the FAST scan is to detect free intra-abdominal, pericardial, or pleural fluid in body cavities using standardised views as a **rapid, minimal ultrasound examination.** Any free fluid detected in a trauma victim suggests internal bleeding unless a different explanation for the fluid is known. Because FAST is not time-consuming and has no side effects for the patient, it is basically indicated in every form of trauma (such as acute blunt or penetrating torso trauma, trauma in pregnancy, or paediatric trauma). Accordingly, it has become a well-established, widely used protocol around the world. The information from a FAST scan can significantly shorten time to surgical intervention. On average, it takes only two to four minutes to perform a complete FAST examination—so it is fast in the truest sense of the word.

Though free fluid suggests internal bleeding in a trauma victim, this interpretation needs to change for **patients without trauma.** Here, the cause of the fluid is less likely to be bleeding, and more likely to be an underlying **infectious disease** or **malignancy.** In recent years, POCUS has proved to be a useful tool for evaluating HIV patients suspected of co-infection with **extrapulmonary TB (EPTB). Unilateral pleural effusions** or **pericardial effusions** are suggestive of pleural or pericardial EPTB; though less specific, **ascitic fluid** may be a sign of peritoneal TB. In other words, the same FAST protocol used with trauma victims is also used in the HIV/TB setting—only the interpretation is different.

Technique and typical ultrasound findings

Technique

Six probe positions are used in the FAST protocol. The examiner assesses all of them for the presence of anechoic fluid. (Figure 4.1)

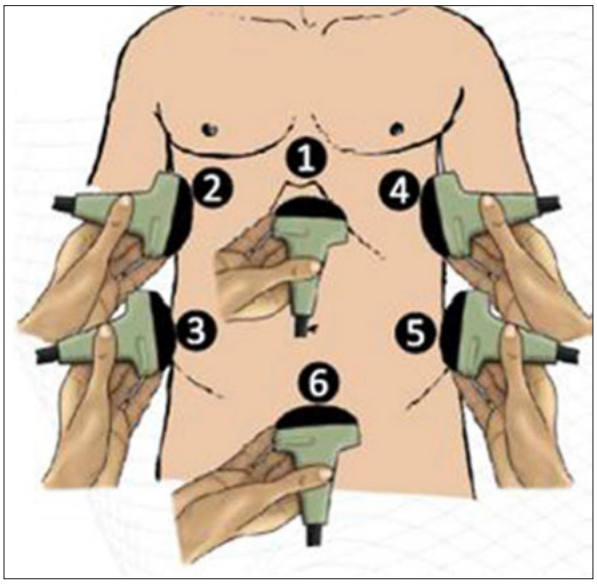


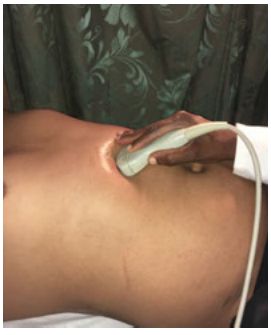
Figure 4.1. Schematic drawing of the six ultrasound probe positions used during the FAST exam. (Source: Umuhire et al. Ultrasound J (2019) 11:18. https://doi.org/10.1186/s13089-019-0133-8.)

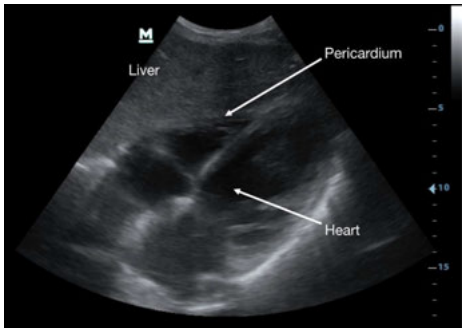
Cardiac view

The cardiac view (Figure 4.2) is obtained in a **subxiphoid position** with the convex transducer in a transverse orientation, tipped cranially and aimed towards the patient's left shoulder, using the liver as an acoustic window. The patient should try to **relax the abdominal muscles,** with arms placed beside the body; pressure is applied with the transducer parallel to the abdominal wall, while aiming the beam behind the ribs. Asking the patient to take a deep breath can help displace the heart caudally and improve visualisation.

Right flank views

The patient is asked to place their **arms behind the head** for better access to the flanks. This position also widens the space between individual ribs. The transducer is positioned on the **longitudinal plane,** posterior to the right mid-axillary line at the caudal part of the thorax, to detect **pleural fluid above the right hemidiaphragm** (Figure 4.3). The scan plane can be adjusted so that the long axis of the probe is parallel to the ribs, allowing an intercostal view. It is important to **place the transducer**





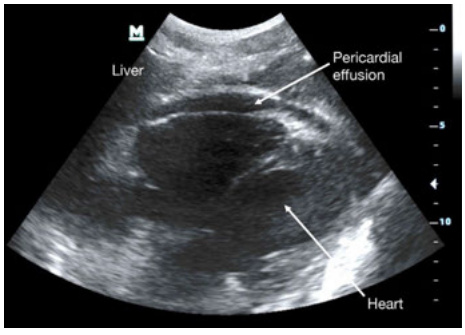
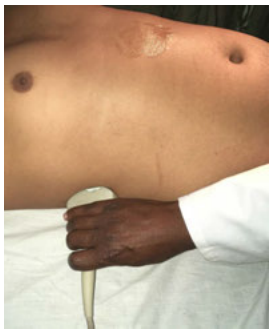
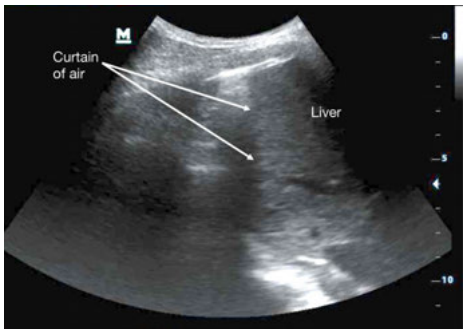


Figure 4.2. a) Position 1: Transverse upper abdominal scan, with probe tilted upwards (used to look for pericardial effusion).

b) Normal: Using the liver as an acoustic window, the heart is visible.

c) In the same probe position, an anechoic rim can be seen around the right and left ventricles.





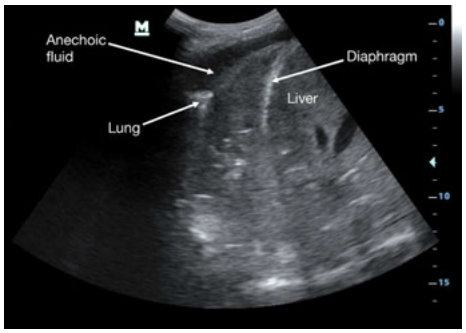
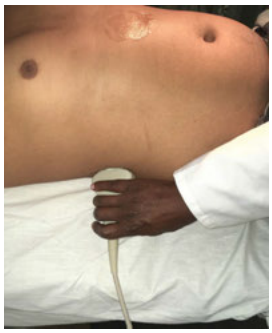
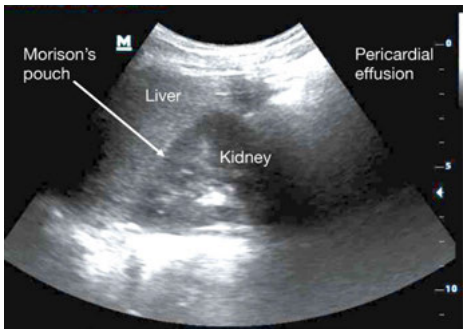


Figure 4.3. a) Position 2: Right flank cranial scan (used to look for right pleural effusion).

b) Normal: The cranial parts of the liver can be seen; a moving 'curtain' of air in the costophrenic angle is also visible.

c) The liver and the echogenic diaphragm can be seen in the cranial angle, with an anechoic rim of fluid visible next to the lung.





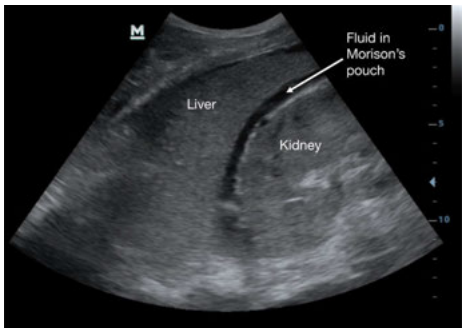
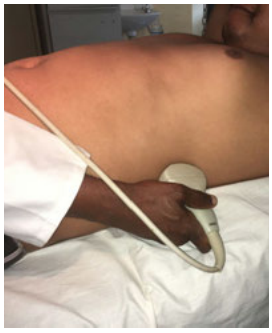
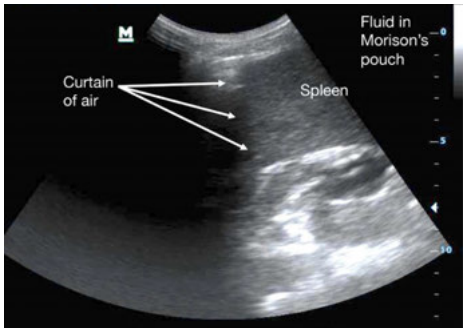


Figure 4.4. a) Position 3: Right flank caudal scan (used to look for ascites in Morison's pouch).

b) Normal: Liver and kidney can be seen adjacent to each other.

c) An anechoic rim is visible between the liver and kidney, representing fluid in the Morison's pouch. Additionally, a small amount of fluid is visible between the liver and abdominal wall.





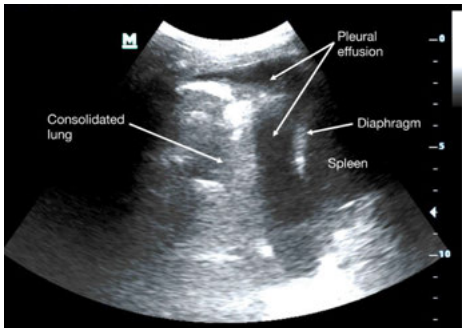
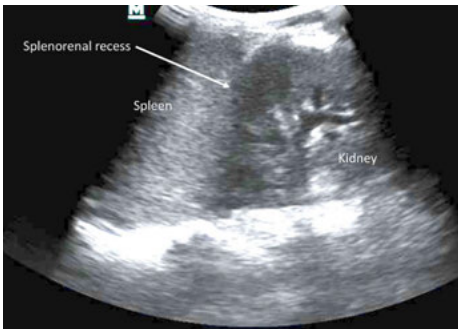


Figure 4.5. a) Position 4: Left flank cranial scan (used to look for left pleural effusion).

b) Normal: Spleen is visible; similar to the right flank, a 'curtain' of air can be seen moving up and down during respiration (note that this movement will not be visible in the still image).

c) Anechoic fluid filling the costophrenic angle is visible above the diaphragm. The partly compressed lung with echogenic air is also visible.



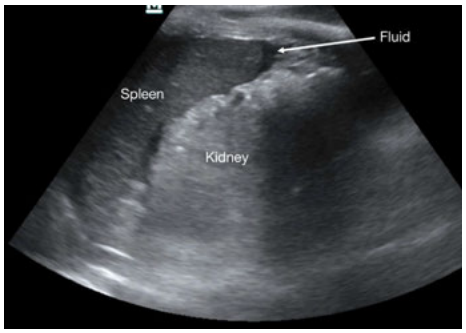


Figure 4.6. a) Position 5: Left flank cranial scan (used to look for ascites in the splenorenal recess).

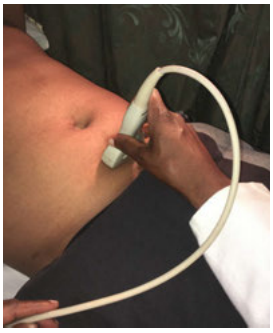
b) Normal: Spleen and kidney can be seen adjacent to each other.

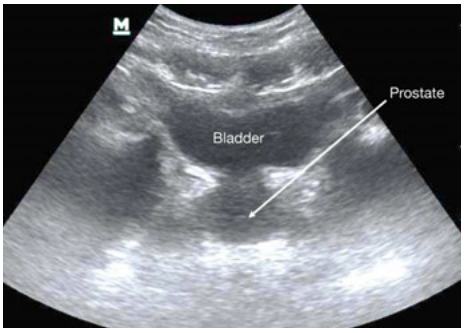
c) Around the lower pole and towards the hilum of the spleen a small anechoic rim of fluid can be seen.

as far back as possible (with the back of the technician's hand touching the examination couch—aim for 'knuckles to the bed'), as gravity causes fluid to collect in the dependent parts. Sliding further, caudal fluid in the abdomen can be detected in **Morison's pouch** (the hepatorenal recess), as this is dependent in a supine patient, and fluid follows gravity (Figure 4.4).

Left flank views

These positions are **analogous to the right flank views.** The transducer is now positioned on the longitudinal plane, posterior to the left mid-axillary line at the caudal part of the thorax. Pleural fluid can be detected above the **left hemidiaphragm** (Figure 4.5), and abdominal fluid in the dependent **splenorenal recess** (Figure 4.6).





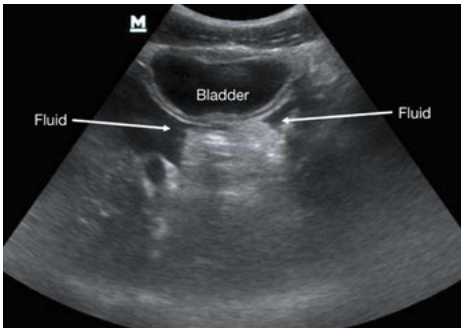
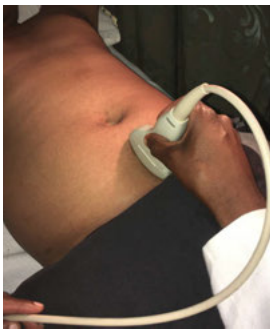
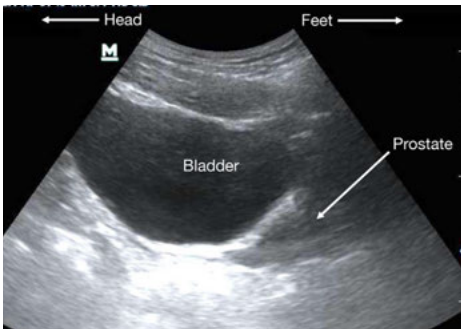


Figure 4.7. a) Position 6: Transverse pelvic scan (used to look for ascites in the pouch of Douglas).

b) Normal: When the probe is tilted further downwards, the prostate becomes visible under the bladder.

c) Fluid is visible next to and behind the bladder.





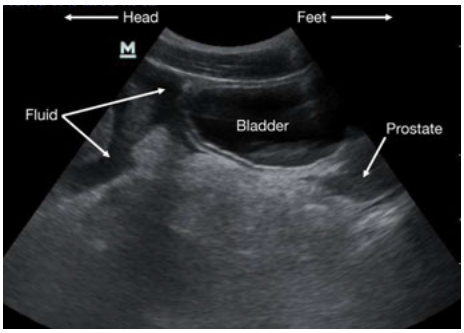


Figure 4.8. a) Position 6: Longitudinal pelvic scan (used to look for ascites in the pouch of Douglas).

b) Normal: The urine-filled bladder is visible as an anechoic area; behind the bladder, the hypoechoic seminal vesicles are seen. The rectum is visible in the distance.

c) Anechoic fluid can be seen next to the bladder.

Pelvic view

Finally, the probe is placed on the **lower abdomen,** touching the upper rim of the symphysis pubis. The pelvic region should be scanned along the **transverse axis** (Figure 4.7). For visualisation of structures deeper in the pelvis, the transducer needs to be tipped so that the ultrasound beam is directed caudally into the small pelvis. The **pouch of Douglas** (rectouterine pouch) in females and the **rectovesical pouch** in males are of particular importance if the patient was walking or sitting upright prior to the examination, as free fluid will have predominantly collected in these spaces (Figure 4.8).

Pathological findings

Pericardial fluid

Pericardial fluid appears as an **anechoic, black rim around the heart** (Figure 4.9), separating the visceral and parietal pericardia. **Small amounts of fluid** are mainly seen **next to the right atrium** and ventricle. The anechoic rim may surround the entire heart as the amount of fluid increases.

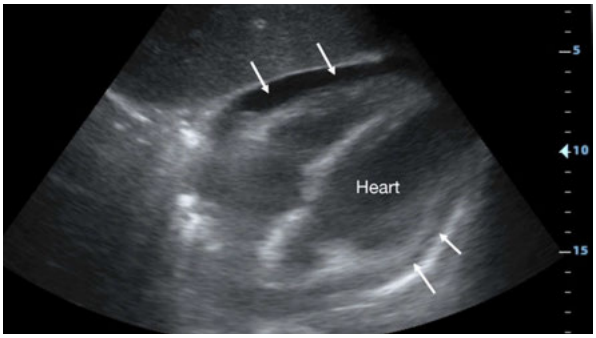


Figure 4.9. Anechoic black fluid (simple pericardial effusion) can be seen surrounding the entire heart (arrows).

In patients with **tuberculous pericarditis, echogenic fibrin streaks** (Figure 4.10) may be seen floating in the anechoic effusion. These may be attached to the pericardium and move as the heart moves. Occasionally, the whole effusion appears echogenic due to high particle content in purulent exudative effusions.

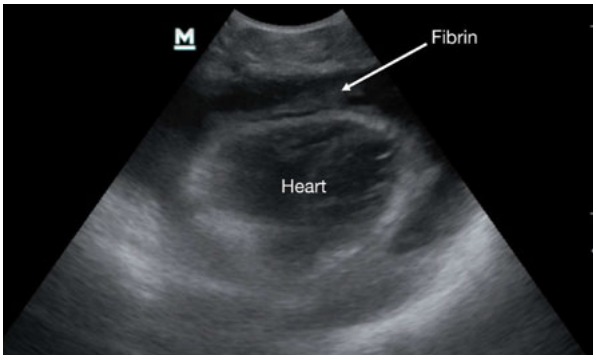


Figure 4.10. Larger pericardial fluid collection with echogenic material and fibres (complex pericardial effusion).

Once an effusion has been identified, it is important to determine whether it is haemodynamically relevant. Signs of tamponade, such as **diastolic collapse of the right atrium and ventricle,** may be seen due to increased pericardial pressure (see Chapter 6).

Pleural fluid

Anechoic fluid (Figure 4.11) may be visible in the costophrenic space. In the case of transudates or simple parapneumonic effusions, fluid may be completely echo-free. As in tuberculous pericarditis, it may contain internal echoes, which appear as strands, septa (Figure 4.12), or **turbid 'smoke'** (Figure 4.13), due to fibrinous structures or cells within the effusion.

Abdominal fluid

Ascites can be seen in the most dependent pockets of the abdominal cavity (Figure 4.14):

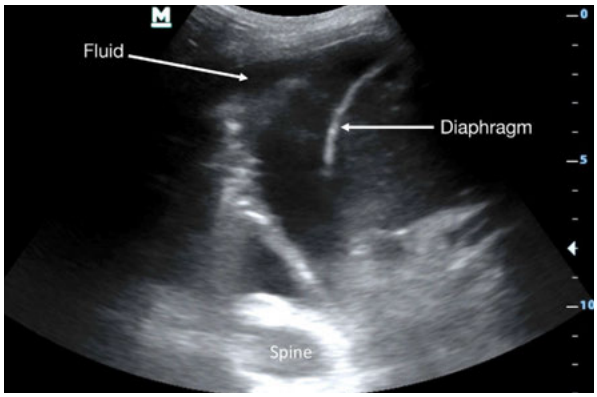


Figure 4.11. Anechoic pleural effusion above the echogenic diaphragm. In the distance, in the cranial parts of the spine are visible (spine sign).

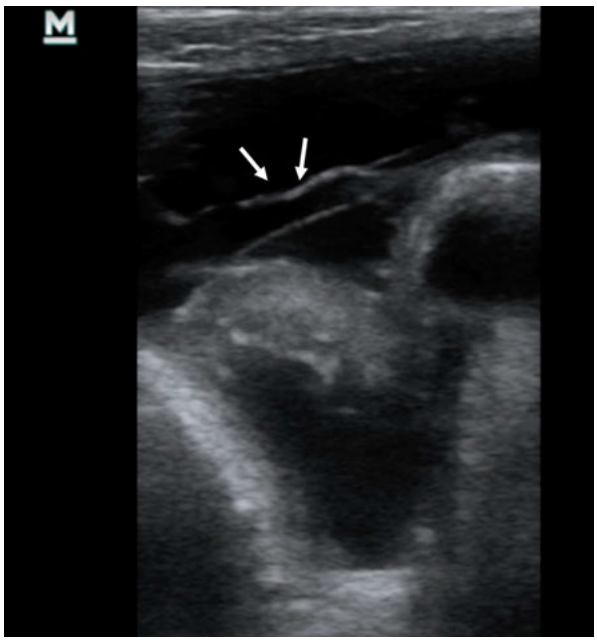


Figure 4.12. Complex pleural effusion with fibrin strands and septa.

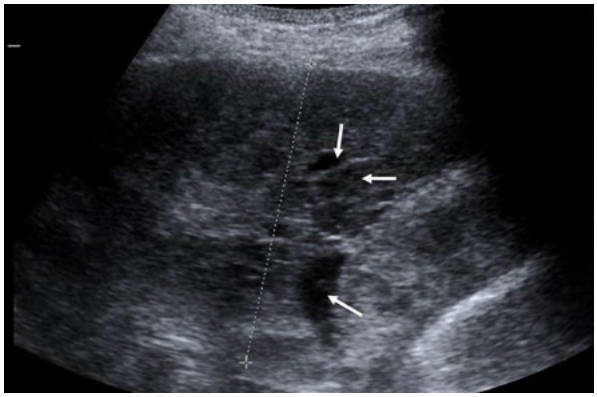


Figure 4.13. Hypoechoic ('solid') pleural effusion with anechoic areas (arrows).

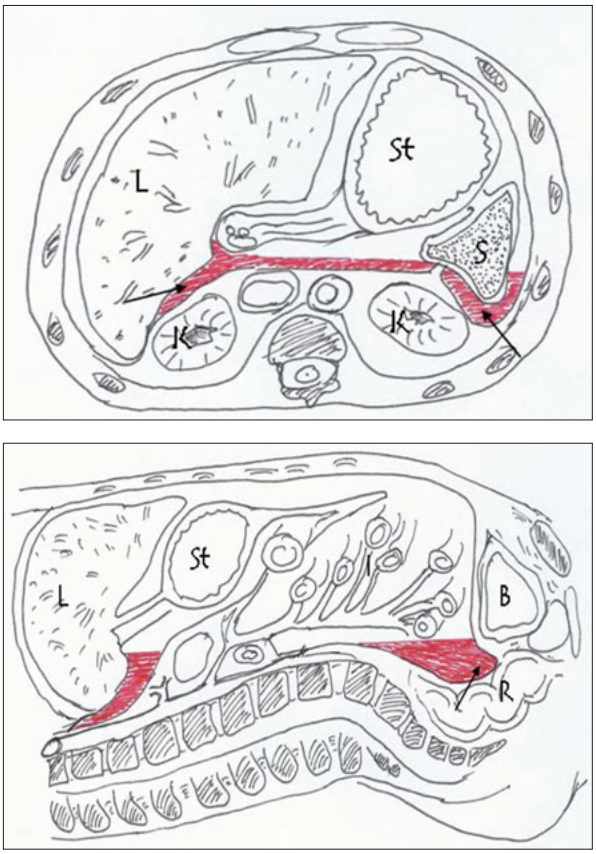


Figure 4.14. Illustrations showing transverse and longitudinal sections of the body. Effusions collect in the supine patient in the most dependent pouches of the peritoneal cavity (arrows): between liver (L) and kidney (K), between spleen (S) and kidney (K), and behind the bladder (B) close to the rectum (R). (St = Stomach, I = intestine).

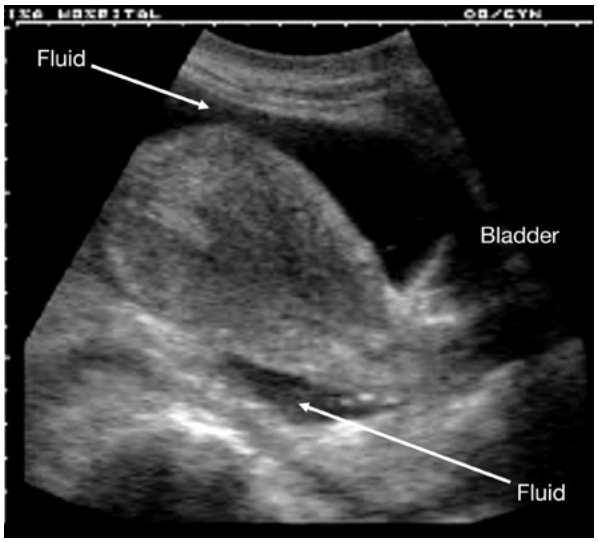


Figure 4.15. Longitudinal view of female pelvis with anechoic fluid both behind the uterus (pouch of Douglas) and surrounding it anteriorly.

- **Morison's pouch** and **splenorenal pouch –** Anechoic collections between liver and kidney, or between spleen and kidney, are signs of free abdominal fluid.
- **Pouch of Douglas –** In the pelvic view, fluid may be seen between the bladder and rectum (in male patients), or in Douglas' pouch, behind or around the uterus (in female patients) (Figure 4.15).

Since ascites due to **tuberculous peritonitis** has a high protein content (and a predominance of lymphocytes), fibrin strands, septations, and webs are commonly visible (Figure 4.16 on the next page). **Several organs** in the abdomen can **contain fluid** that may be mistaken for free fluid. In the upper abdomen, fluid within the **gall bladder** (Figure 4.17), **aorta and inferior vena cava (IVC)** (Figure 4.18), **stomach,** and (on rare occasions) the **colon** can be seen. In the pelvis, fluid in the **bladder,** large **ovarian cysts,** or the seminal vesicles can pose diagnostic challenges. Emptying the bladder before the examination may be helpful in these cases to confirm the presence of free fluid. In **premenopausal women, small amounts of free fluid** in the pouch of Douglas **may be normal.**

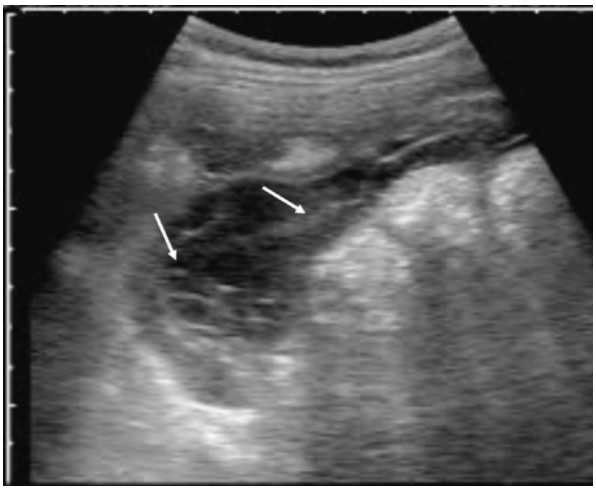


Figure 4.16. Like other effusions, ascites can have a complex appearance, with strands and septa (arrows).

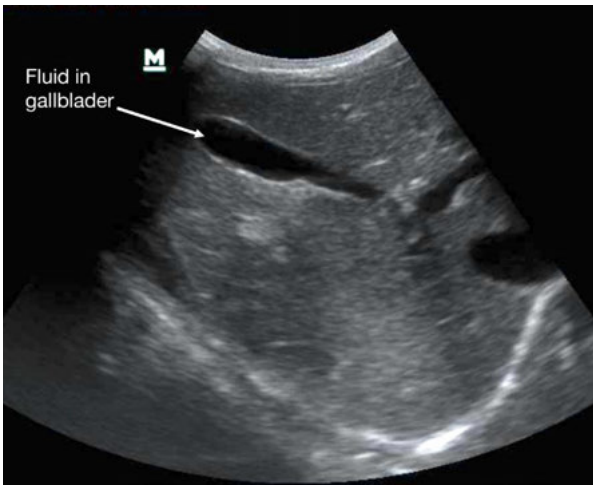


Figure 4.17. Pitfall: Subcostal view of the liver. In the left upper corner, anechoic fluid is visible in the gallbladder; this should not be confused with ascites.

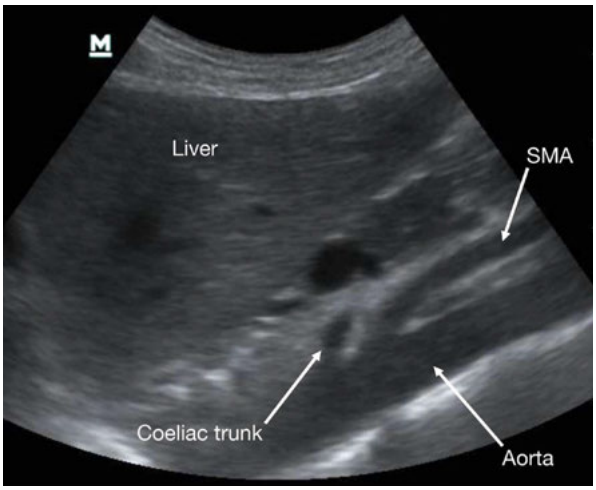


Figure 4.18. Pitfall: Longitudinal view of the upper abdomen. In the distance, the anechoic vessel (abdominal aorta) is visible, along with its branches (coeliac trunk, SMA = superior mesenteric artery).

Diagnostic and therapeutic decisions

The diagnostic and therapeutic decisions made based on fluid found on ultrasound will largely depend on the clinical context.

In an HIV patient, a **pericardial effusion** is highly suggestive of TB pericarditis. The most important **differential diagnosis** is **Kaposi sarcoma (KS),** so the patient's skin (especially on the groin and legs, but best the whole integument) and oral mucosa must be thoroughly examined for KS lesions. If none are found, **empirical anti-TB treatment** is warranted, particularly in the presence of severe immunosuppression. **Corticosteroid treatment** should also be given, especially in cases of large and hemodynamically relevant pericardial effusions. (It should be noted that malignant effusions may also partially respond to steroid treatment, but patients will tend to relapse after the steroids are tapered off.)

Further diagnostic procedures, such as **pericardiocentesis, are rarely performed** unless there are signs of tamponade requiring therapeutic drainage. When fluid is obtained, it is most commonly straw-coloured, but is blood stained in about 10% of cases. (Blood-stained fluid should make you think of KS again!) The fluid should be submitted for GeneXpert MTB/RIF Ultra testing.

A **pleural effusion** in an HIV patient, especially when it is unilateral, is also highly suggestive of TB. Still, a few other differential diagnoses should be considered:

- **Cardiac disease** – Effusions are commonly bilateral; symptoms suggestive of cardiac origin include orthopnoea (shortness of breath when lying down), nycturia (the urge to get up and pass urine more than once or twice during the night), and lower limb oedema.
- **Community-acquired pneumonia (CAP)** – CAP is usually caused by bacteria and can be unilateral. Suggestive clinical clues are a shorter history (usually days) of fever and cough symptoms. A CXR may help to differentiate.
- **Other infections** – Pleural empyema may be a consequence of bacterial lung infection.
- **Cancer** – Again KS, but also consider and look for other cancers.
- **Kidney** (renal failure with fluid overload) and **liver disease** (cirrhosis with hypoproteinaemia).

TB is the most common reason for pleural effusion in HIV patients hospitalised in Malawi, accounting for 40% of cases, followed by cancer (25%). Therefore, the threshold for empirical anti-TB treatment should be low. Among **HIV-negative patients, cardiac causes** (30%) and **CAP** (20%) are more frequent than TB (15%). Still, TB treatment may be considered if no other explanation can be found.

A **diagnostic tap of pleural fluid** can be easily done **using ultrasound guidance.** It is helpful if the **GeneXpert MTB/RIF Ultra** test result is **positive,** as this confirms the diagnosis (Gene Xpert specificity is high). A negative result, on the other hand, does not rule out the possibility that the patient will improve on TB treatment (sensitivity is low); empiric treatment may still be needed.

Ascites without other supporting findings of disseminated TB (such as enlarged lymph nodes or splenic microabscesses) may be caused by various conditions; it should be interpreted in the context of other clinical and laboratory findings.

Other common explanations that should be considered:

- **Liver disease** is the most common cause of ascites, regardless of HIV status. Cirrhosis and hepatocellular carcinoma (HCC), both of which are associated with hepatitis B infection, are frequent underlying diseases. Schistosomiasis may also need to be considered.
- **Cardiac diseases** are also common causes of ascites; look for signs and symptoms of underlying heart disease.

- **Cancer,** especially ovarian cancer in female patients, can present with ascites.
- **Kidney disease** or frank renal failure can present with fluid overload.
- **Other infections,** such as typhoid and other forms of bacterial peritonitis, are differential diagnoses that, although rare, should be considered.

TB is the cause in about a quarter of ascites cases among HIV patients hospitalised in Malawi. If no other explanation is found, and the clinical picture suggests TB is likely, empiric treatment may be started. Among HIV-negative patients, TB is found to be the cause of ascites in less than 5% of cases.

Drainage of ascites can be easily done **using ultrasound guidance.** It is helpful if the **GeneXpert MTB/RIF Ultra** test result is **positive,** as it confirms the diagnosis (specificity is high); it should at least be attempted with HIV patients. Again, a negative result does not rule out the possibility that the patient will improve on TB treatment (sensitivity is low); empiric treatment may still be required. A sample can be sent for cytology to look for cancer cells.

Pearls and pitfalls

- When pleural fluid is present, make sure that the transducer is posterior and cranial enough to allow identification of the hemidiaphragm beneath the pleural fluid.
- Do not hesitate to use a needle to obtain a sample through paracentesis or pleural tap.
- Be aware of other causes of abdominal fluid, such as ascites in hepatic cirrhosis and physiologic fluid within organs.
- Be cautious when examining ultrasounds of pregnant women; intrauterine fluid can cause confusion during evaluation for ascites.
- Know the limitations—obesity and bowel gas reduce penetration of the ultrasound beam.

5. Ultrasound of Abdominal Lymph Nodes and Spleen: The FASH Protocol

Introduction

Weight loss is a frequently encountered clinical problem in resource-limited settings, especially in populations with a high prevalence of HIV and TB. A variety of conditions can cause weight loss and wasting, ranging from nutritional deficiencies (due to malnutrition or chronic diarrhoea), metabolic diseases (diabetes mellitus), and infectious diseases (HIV, TB, parasitic infections) to malignancies.

When weight loss is accompanied by **fever, night sweats, or cough,** TB is highly likely, particularly in **HIV co-infected patients who have a substantially higher risk of TB.** For this reason, these three symptoms are used by a variety of screening algorithms to identify suspected cases of TB.

Obviously, other HIV-related pathologies can cause similar symptoms. Such malignancies as HIV-associated non-Hodgkin lymphomas and disseminated KS, and a variety of opportunistic infections, produce similar presentations. Identifying a suspected case of TB will prompt the search for further evidence to support the diagnosis of TB, which can be confirmed by a positive AFB stain from sputum, or by positive GeneXpert MTB/RIF or urine LAM test results. If these tests are negative or inconclusive, CXR can be done to check for radiological patterns suggestive of TB, including infiltrates, cavities, or a miliary pattern.

In recent years, ultrasound has proved to be a useful tool in evaluating HIV patients suspected of co-infection with EPTB. Besides the **effusions,** which we discussed in the previous section, detection of abdominal **lymphadenopathy** and **splenic microabscesses** are typical findings suggestive of abdominal TB.

A logical consequence has been the development of a focused POCUS protocol for EPTB, the **Focused Assessment with Sonography for HIV-associated TB (FASH).** The FASH exam combines the ultrasound views of FAST with additional tilting of the transducer to assist in detection of upper abdominal lymphadenopathy and splenic microabscesses. FASH should be considered for all patients who have a high clinical probability of disseminated TB and EPTB.

Technique and typical ultrasound findings

Abdominal lymph nodes

To detect enlarged upper retroperitoneal and periportal abdominal lymph nodes, the transducer is tilted to a **transverse position** at the level of the epigastric view of the heart (Figure 5.1). In this position, the **upper abdominal and periportal area** can be visualised by angling the ultrasound beam, first up through the left lobe of the liver towards the diaphragm, and then down along the aorta. The liver serves as a good ultrasound window for detecting periportal nodes in the liver hilum and around the coeliac axis. To minimise the distance between the probe and the retroperitoneal areas, the abdominal wall should be relaxed, with the patient's **arms positioned next to the abdomen.**

Normal lymph nodes are small round or oval structures whose size ranges from a few millimetres up to 1 cm; they **are usually not visible in the abdomen** unless they are enlarged. The **parietal nodes** are lo-

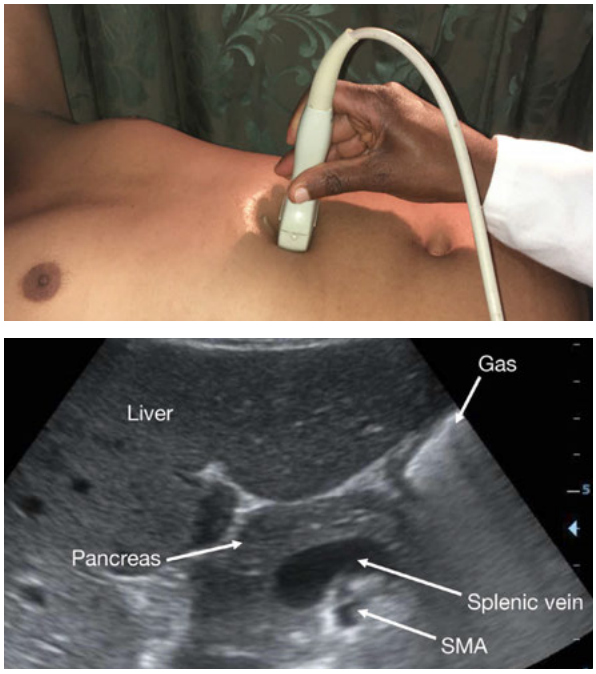


Figure 5.1. Transverse scan of upper abdomen. (Top) The transducer is placed perpendicular to the abdomen, and moved slowly from the epigastrium to the umbilicus. (Bottom) In a normal image, the liver is seen on the left, with the pancreas and vessels (splenic vein and superior mesenteric artery) visible in the centre. Towards the right, gas in the stomach may affect the image, as in this example.

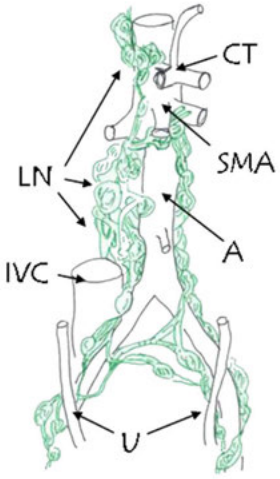


Figure 5.2. Para-aortic lymph nodes (LN) are located around the aorta (A) with its branches (CT=coeliac trunk, SMA=superior mesenteric artery). Their proximity to the inferior vena cava (IVC) and ureters (U) can be seen in this view.

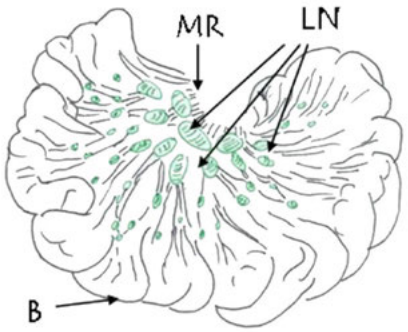


Figure 5.3. Visceral lymph nodes (LN) drain lymphatic vessels from the bowel (B); they are located towards the mesenteric root (MR).

cated in the retroperitoneum, **close to the large vessels** (Figure 5.2). These nodes are a continuation of the lymphatics, which drain from the lower half of the body. The **visceral lymph nodes** are located at the **root of the mesentery as well as in the portal area** (Figure 5.3). They drain lymphatics from the bowels, pancreas, and hepatobiliary system.

Although normal-sized abdominal lymph nodes cannot be detected through ultrasound, **superficial lymph nodes** (such as those in the axilla or the inguinal region) may be visible, especially when using high-frequency linear transducers. When visible, they appear as round, **hypoechoic structures** surrounded by a **connective tissue capsule,** and often show an **echogenic centre (hilus fat sign)** due to central fat and connective tissue (Figure 5.4).

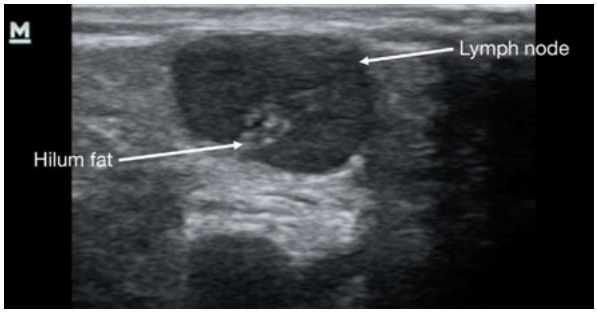


Figure 5.4. Lymph node: hypoechoic oval structure with central echogenic hilum.

Spleen

The spleen is best examined with the patient in a supine or right lateral decubitus position, with the **left arm placed above the head.** A lateral and posterior probe position is used, angling the beam anteriorly to identify the spleen. (If the probe is placed anteriorly, the spleen is usually obscured by gas in the stomach or colon.) The **transducer is turned parallel to the ribs** (Figure 5.5).

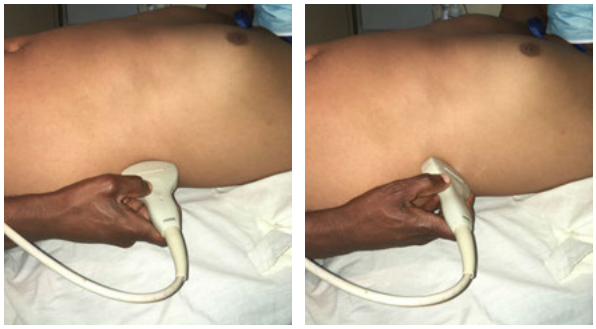


Figure 5.5. Transcostal spleen: to optimise the visibility of the spleen, rotate the transducer slightly to scan between (parallel to) the ribs. (L) From the longitudinal left flank scan position (position 5, as shown in Fig. 4.6a), (R) rotate clockwise until parallel to the ribs and scan between the ribs.

Asking the patient to breathe in is often counterproductive; the spleen usually disappears behind the expanding lung. For this reason, the spleen should first be scanned using an abdominal probe. A **linear transducer can be very helpful** here, as it enables higher-resolution capture of parenchymal changes and detection of microabscesses; it should be used whenever available.

The shape of the normal spleen is variable, but typically resembles a coffee bean (Figure 5.6). Its size and weight may differ from person to person, but **normal size** is about **11 cm x 4 cm.** In African patients, the upper end of the normal range may be larger (~13 cm), as **mild enlargement is frequently seen** in settings

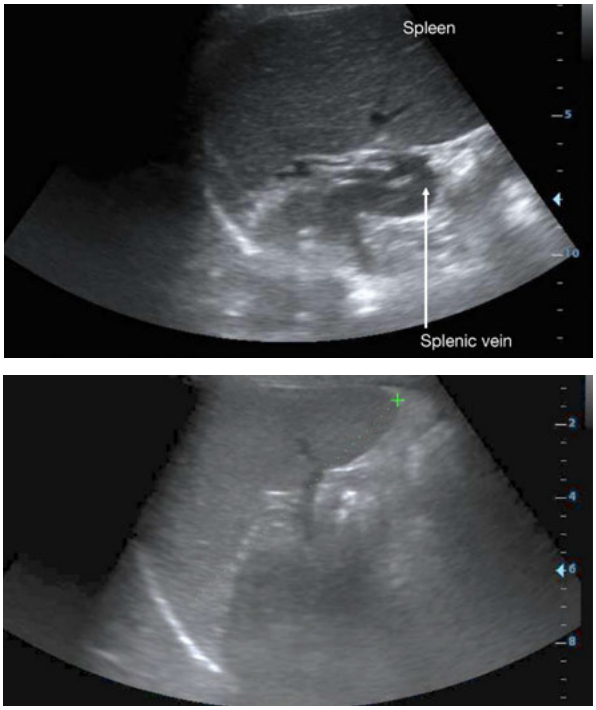


Figure 5.6. Transcostal spleen: (Top) The normal homogenous bean-shaped organ, seen with a convex abdominal probe; the vessels at the hilum are usually visible. (Bottom) When measured along the longest axis, the spleen should be smaller than 11 cm.

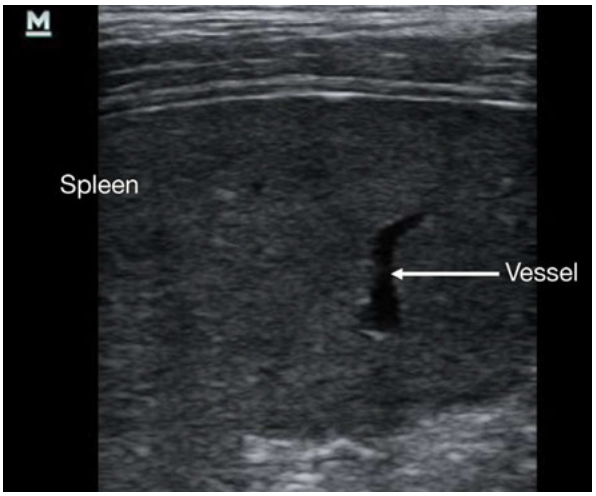


Figure 5.7. Transcostal spleen scanned with linear probe: normal vessels can be seen branching in the spleen. The tissue is homogenous.

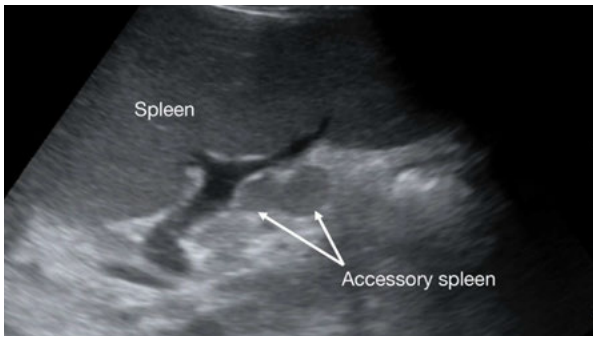


Figure 5.8. Normal spleen with two accessory spleens in the region of the hilum. Accessory spleens are usually seen in this location, and towards the caudal pole of the spleen.

where chronic parasitic infection and recurrent malaria are common. In slim patients, longer, flatter spleens may be seen. The parenchyma of the spleen is homogenous, with a **fine, velvet-like echo pattern** (Figure 5.7). It resembles the liver, but is often slightly less echogenic. In the hilar region, the splenic artery and splenic vein are

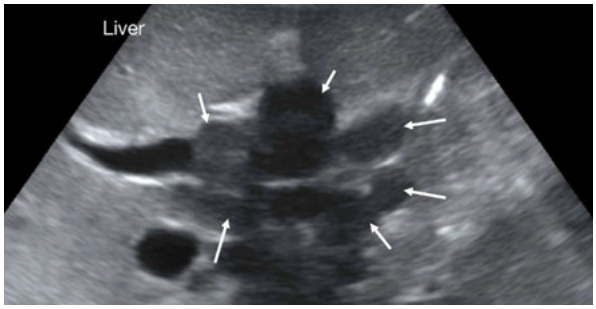


Figure 5.9. Upper abdomen transverse: Compared to the scan in Figure 5.4, a large number of roundish, hypoechoic structures (arrows) can be seen. Some of them may be vessels, but there are too many for that to be the case—many are actually enlarged lymph nodes.

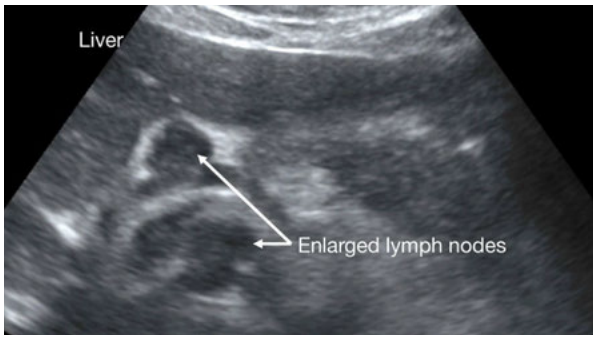


Figure 5.10. Enlarged lymph node in the portal area behind the liver.

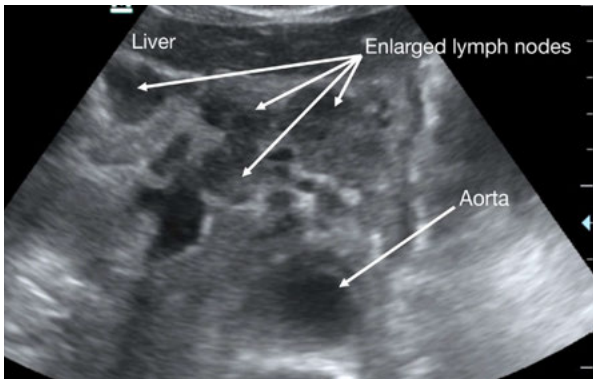


Figure 5.11. Another example of multiple enlarged lymph nodes in the upper abdomen. The nodes are slightly more echogenic than in the previous examples.

visible centrally. Lobulation with a variable outer contour of the spleen can be seen as a normal variant. **Accessory spleens** (Figure 5.8 on the previous page) are often located in the hilar region of the spleen, along the gastrosplenic ligament, or at the caudal pole of the organ. They have the same echo pattern as the spleen itself, are often round, and are usually small (< 2.5 cm).

Pathological findings

Enlarged lymph nodes

Lymph nodes larger than 1.5 to 2 cm are considered pathological in an adult with HIV. Pathological **enlarged nodes in TB** are often **hypoechoic** and **rounded** (Figures 5.9–13). The markedly low echogenicity is due to loss of internal structures caused by **caseous necrosis.** The lymphadenopathy may be discrete, or nodes might be **conglomerated into a larger mass.** On rare occasions, tuberculous lymph nodes may appear hyperechoic. Lymph node masses may cause obstruction of ureters, the pancreas, and biliary tract, or (less often) the digestive system.

Lymph nodes can be differentiated from adjacent tubular vessels by their rounded appearance, which will be noticeable as the transducer moves across the node

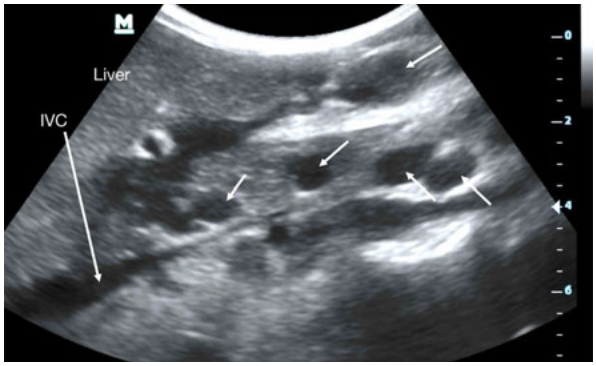


Figure 5.12. Upper abdominal longitudinal scan: The liver and the large vessels (IVC) can be seen. Multiple hypoechoic roundish lymph nodes (arrows) are also visible.

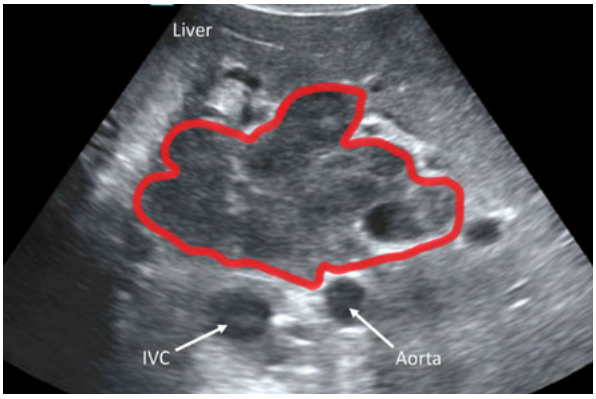


Figure 5.13. Multiple enlarged lymph nodes. The nodes seem to be matted together, forming a bulky structure. Still, the nodular structure can be seen in the outline.

and along the vessels. This gives the lymph nodes a **'blinking' appearance.** The size of a node is measured at its maximal diameter in the short axis.

Splenic lesions

In patients with disseminated TB, **splenic microabscesses** may appear as multiple small **hypoechoic lesions a few millimetres in size** (Figures 5.14–16). These microabscesses are distributed throughout the spleen and represent miliary seeding. Again, as the transducer fans through the spleen, they will appear to be blinking.

Diagnostic and therapeutic decisions

TB treatment without further confirmation is indicated in patients with enlarged abdominal lymph nodes or splenic microabscesses, especially when local TB prevalence is high. In many cases, further diagnostic workup may delay (or even prevent) treatment, thus increasing morbidity or mortality. A careful physical **examination should be done for KS** as a potential differential diagnosis.

When empiric treatment is started, **the patient should be assessed clinically after two to four weeks, and possibly again after eight weeks.** To evaluate the effectiveness of treatment, one should inquire as to whether symptoms are improving. In patients with persistent findings, **non-compliance,** an **incorrect drug regimen, drug resistance, IRIS,** or such **alternative diagnoses** as lymphoma or KS must be considered and investigated (for example, by doing an ultrasound-guided biopsy).

It is important to remember that in patients with **very low CD4 counts** (< 50 cells/mm^3), disseminated infection with ***Mycobacteria avium intercellulare*** can cause

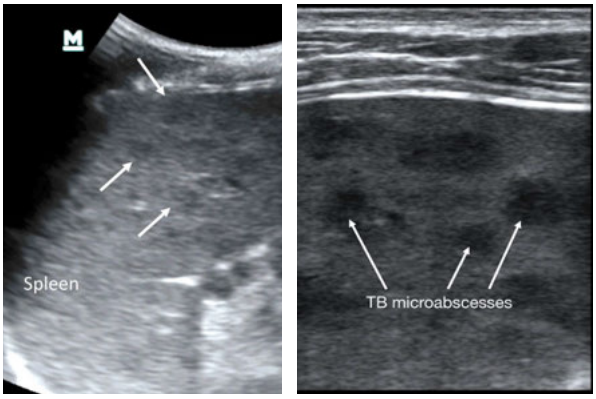


Figure 5.14. Transcostal spleen: (L) Scanned with the convex probe, irregularities are visible (arrows)—although these may be difficult for a scanning novice to see. (R) The same spleen scanned with a linear probe shows clearly visible, round, hypoechoic TB microabscesses.

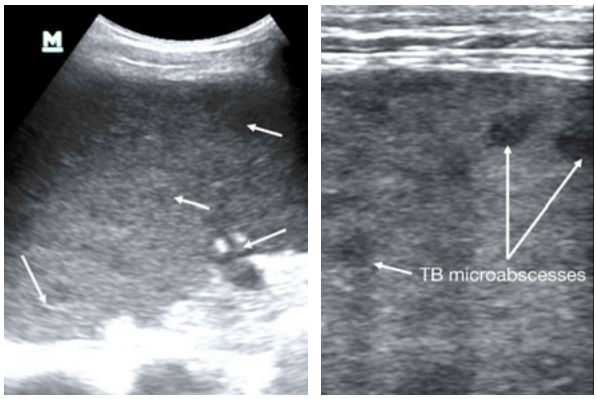


Figure 5.15. Transcostal spleen: (L) With the convex probe, the irregularities (arrows) are again easy to miss. (R) The same spleen scanned with a linear probe clearly shows the TB microabscesses.

the same sonographic changes of enlarged lymph nodes and splenic lesions. As culture is rarely available, it may be necessary to cover these organisms by adding a macrolide antibiotic to the patient's drug regimen.

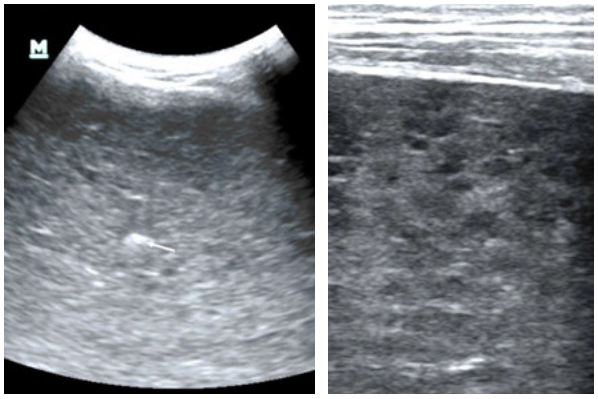


Figure 5.16. Transcostal spleen: (L) With the convex probe, the enlarged spleen shows irregularities that are easy to miss. (R) The same spleen scanned with the linear probe reveals thousands of lesions next to each other.

Pearls and pitfalls

- Usually there are multiple enlarged lymph nodes, rather than only one. They may also be matted together.
- Move the transducer slowly over the abdomen and watch for round structures, which appear small, become larger, and then smaller again (they appear to blink). The diameters of tubular structures (such as vessels), on the other hand, do not change rapidly.
- The spleen is usually more posterior and more cranial than you think. Inspiration is often not helpful, as pulmonary air obscures the spleen.
- In approximately 75% of patients, sonographic findings resolve within three months. However, sonographic findings may initially persist, or even increase during treatment, because of IRIS. This can be especially pronounced when the patient starts ART at the same time; therefore, the persistence of findings does not necessarily indicate an incorrect diagnosis.
- In a significant proportion of patients with positive FASH findings, abnormalities typical for TB are also visible on the CXR (e.g., an enlarged cardiac shadow due to pericardial effusion, pleural effusions, miliary TB in disseminated disease, or classic features of pulmonary TB). On the other hand, approximately 25% of HIV patients with positive FASH findings will have a completely normal CXR.

Lymph nodes and focal lesions in the spleen: exploring differential diagnoses beyond disseminated TB

*As we have seen, enlarged abdominal lymph nodes are one of the hallmark signs of disseminated TB, which makes them an important FASH finding. As much as enlarged lymph nodes suggest TB, a wide variety of **differential diagnoses** should be considered if the clinical picture does not fit well, or if the enlarged nodes do not respond to TB treatment. These situations are complex; therefore, this section is aimed at clinicians who have already developed advanced ultrasound and clinical skills.*

Lymph nodes

Enlarged lymph nodes must be investigated in the context of the most prevalent and probable local aetiologies. In our setting, the most common causes of lymphadenopathy in HIV patients **in addition to TB** are **lymphoma, KS** (Figure 5.17), **persistent generalised lymphadenopathy,** and **reactive lymphadenopathy due to local infections**. These causes should be thought of as the 'Big Five'.

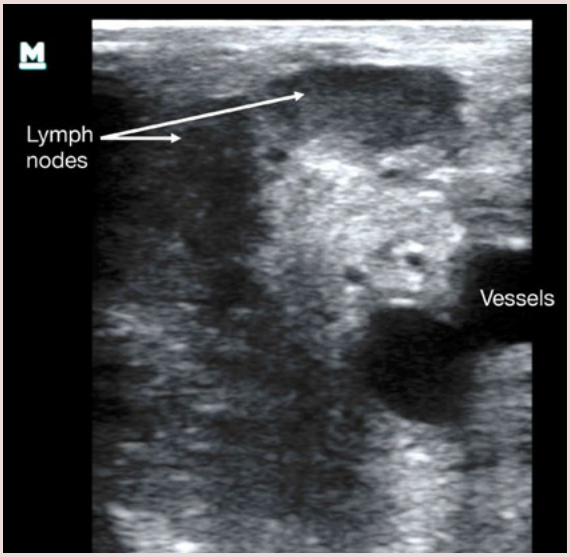


Figure 5.17. Multiple enlarged inguinal lymph nodes (arrows) due to Kaposi sarcoma.

Still, other less common causes of lymphadenopathy must be considered when the patient is HIV-negative and the clinical picture does not fit (Figure 5.18). **Multicentric Castleman disease (MCD)** is a lymphoproliferative disease associated with HHV-8 in HIV patients presenting with **fever, lymphadenopathy,** and often **anaemia.** It is also seen in patients with well-controlled viral replication after many years on ART. **Disseminated *Mycobacterium avium intracellulare* (MAI)** should especially be considered in patients presenting with fever and diarrhoea whose CD4 counts are less than 100 cells/mL. **Histoplasmosis** is a fungal infection that can cause enlarged lymph nodes; though it is more commonly seen in South America, cases have also been observed in Africa.

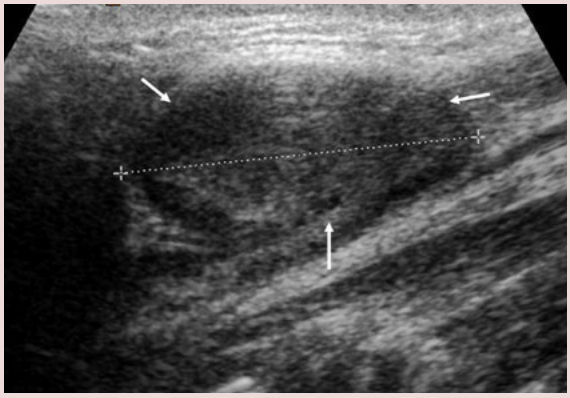


Figure 5.18. Enlarged LN (arrows) on the neck in a patient with testicular cancer metastasis. The node is slightly more echogenic, but a final diagnosis can only be reached by performing a biopsy and histological examination.

The **rare inflammatory diseases** listed in Table 1 below can realistically be **diagnosed only by biopsy** of the nodes. The apparent rarity of these diseases in Africa is probably due to the limited availability of pathology services. Fortunately, they can often be treated using steroids. The usefulness of ultrasound for the biopsy of LN is well established (see Chapter 14 for a description); when pathology services are available, biopsies should be done frequently.

Spleen

In our clinical setting, focal hypoechoic splenic lesions are also mostly the result of mycobacterial microabscesses, and thus are usually an indication for TB treatment. Still, alternative diagnoses exist and should be considered by the experienced clinician. These can be grouped into **malignant** causes, **systemic infections,** and **other causes** (Table 2).

Table 1. Differential diagnosis of lymphadenopathy

<table>
<tr><th>The 'Big Five'</th><th colspan="2">The 'Rare Zebras'</th></tr>
<tr><td rowspan="2">• Tuberculosis
• Lymphoma
• KS
• HIV-associated persistent generalised lymphadenopathy (PGL)
• Reactive lymphadenitis in local infections</td><td>Systemic infections
• Atypical mycobacteriosis (e.g., MAI)
• Infectious mononucleosis (EBV)
• Brucellosis
• Tularaemia
• Syphillis
• Bartonellosis (Bacillary angiomatosis, CSD)
• Fungal infections (e.g., histoplasmosis)
• Toxoplasmosis</td><td>Inflammatory diseases
• Kikuchi-Fujimoto disease
• Dermatopathic lymphadenitis
• Rosai-Dorfman disease
• Langerhans histiocytosis
• Sarcoidosis
• Kawasaki disease
• Sytematic lupus erythematosus</td></tr>
<tr><td>Malignant causes
• Metastatic carcinoma
• T-cell leukaemia
• Hairy-cell leukaemia
• Castleman disease
• Myeloid tumours</td><td>Other causes
• Amyloidosis
• Metabolic storage diseases</td></tr>
</table>

Table 2. Differential diagnosis of focal spleen lesions

Malignant causes	Systemic infections	Other causes
• Lymphoma (NHL) • KS • Other cancer metastases (e.g., GI tract, lung, melanoma)	• Atypical mycobacteriosis (e.g., MAI) • Infectious mononucleosis (EBV) • Meliodosis • Bartonella (Baciliary angiomatosis, CSD) • Visceral leishmaniasis • Bacterial and fungal abscesses (rare)	• Focal calcifications (e.g., following miliary TB, but also trauma) • Gamna-Gandi bodies (e.g., in portal hypertension and sickle cell anaemia) • Splenic infection (e.g., in splenomegaly) • Spleen cysts

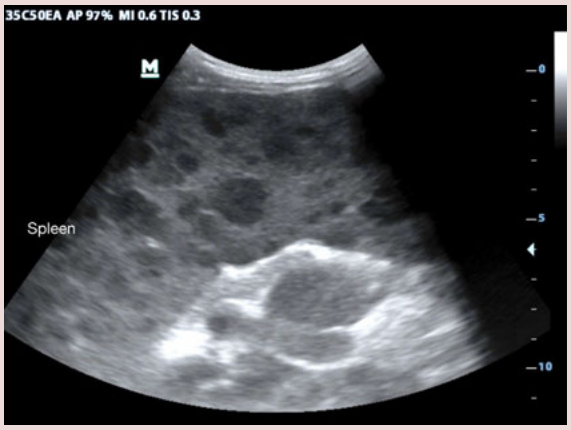


Figure 5.19. Spleen lesions are consistent with TB, but these were due to lymphoma. The slightly larger size of the lesions may suggest malignancy.

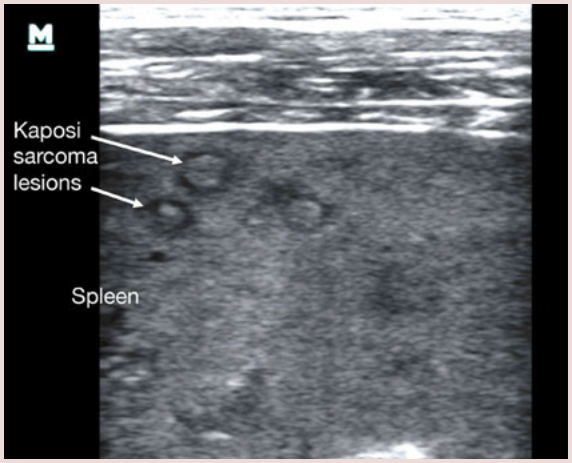


Figure 5.20. KS lesions in the spleen. Compared to TB microabscesses, these lesions are often more echogenic—or can even show a target sign of concentric circles, as in this example.

The most frequent neoplastic focal lesions of the spleen are **lymphomas** (Figure 5.19). Lymphomas are commonly hypoechoic, ill-defined lesions of varying size, and are generally larger than the lesions seen in TB. Burkitt's lymphoma lesions tend to be larger and may have a complex echo-structure.

KS can involve the spleen; these lesions tend to be echogenic (Figure 5.20), though they may also vary in size from a few millimetres to a large lesion occupying a large area of the spleen. Target lesions, with an echogenic centre and hypoechoic rim, have also been observed.

Systemic disseminated infections, such as **brucellosis, melioidosis,** and **visceral leishmaniasis,** can mimic the lesions of disseminated TB—but these infections are not prevalent in our setting. **Toxoplasmosis** presents with calcifications of a few millimetres in size, with comet-tail artefacts or a posterior acoustic shadow.

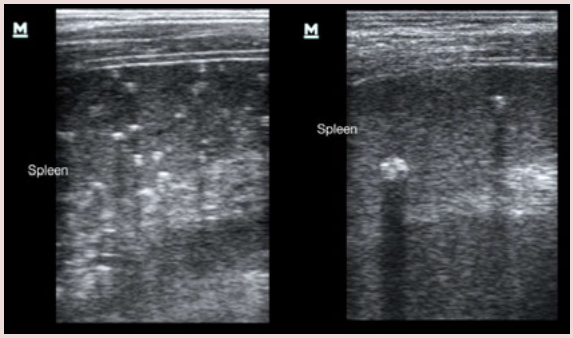


Figure 5.21. (L) Multiple echogenic calcifications in the spleen, some throwing an acoustic shadow. This is seen as a residual of past cases of miliary TB with calcified granulomas in the spleen. (R) Fewer individual calcifications in the spleen is usually a sign of inactive disease.

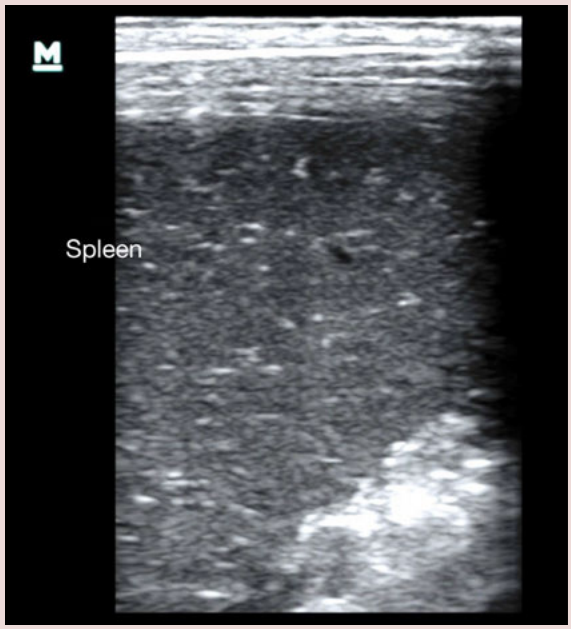


Figure 5.22. Numerous small echogenicities visible throughout the spleen. This is commonly described as a 'starry sky' or 'snowstorm' pattern. This kind of pattern can have many causes.

A similar pattern can be seen in **healed miliary TB** with multiple calcifications in the spleen (Figure 5.21).

A **snowstorm pattern** (echogenic disseminated lesions) has been associated with *Pneumocystis jirovecii* pneumonia (PCP) and other opportunistic fungal infections (Figure 5.22). **Fungal abscesses** can also present as a 'wheel-in-wheel' pattern of multiple concentric rings.

Gamna-Gandy bodies are echogenic nodules (without acoustic shadow) caused by haemosiderin; these are mainly seen in portal hypertension and sickle cell anaemia patients. The pathophysiological explanation is that microhaemorrhages result in haemosiderin and calcium deposits, and produce a fibroblastic (scarring) reaction.

Splenic infarction (Figure 5.23) often appears as a hypoechoic, wedge-shaped triangular lesion with the base towards the capsule. Infarction is seen when perfusion with oxygen-rich blood becomes insufficient to support the spleen, especially when splenomegaly (Figure 5.24) is present.

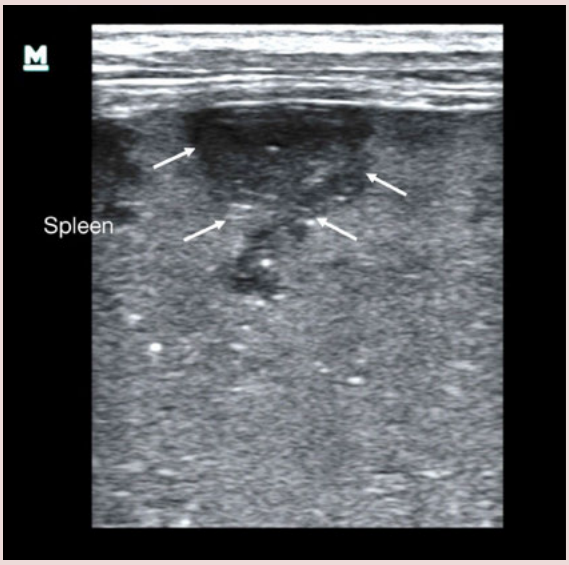


Figure 5.23. Wedge-shaped hypoechoic area in the subcapsular region of the spleen, suggestive of splenic infarction.

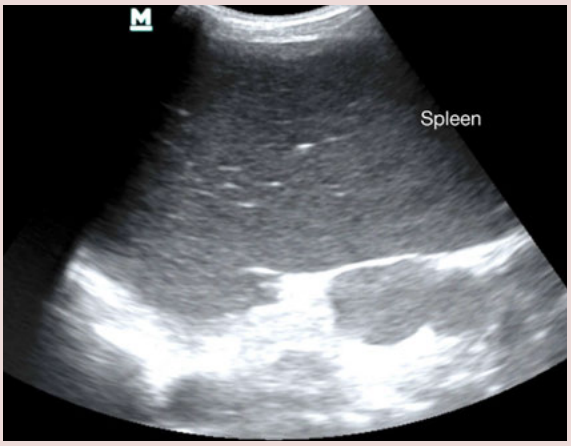


Figure 5.24. Enlarged homogenous spleen—a finding with a broad differential diagnosis.

Cysts in the spleen (Figure 5.25) are not as frequent as cysts in the liver, but when found they are very characteristic—they have sharp borders, are circular or round, and are completely anechoic. They require no further diagnostic workup or treatment.

In rare cases, tears of the capsule and **splenic rupture** can be seen (Figure 5.26). This is usually a consequence of trauma; in some cases with underlying splenomegaly, the trauma may be minor.

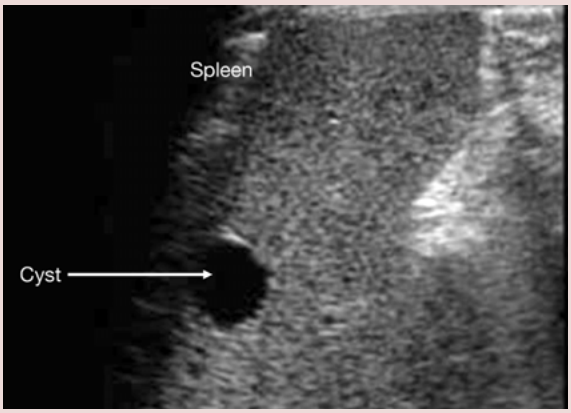


Figure 5.25. Simple, round anechoic cyst in the spleen—usually an accidental finding not suggesting pathology.

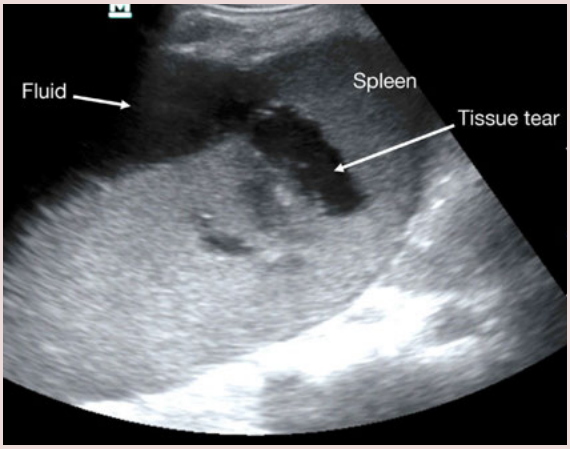


Figure 5.26. Spleen with irregular upper border and hypo- to anechoic fluid in that area. This is a ruptured spleen with blood in the subcapsular area, where the tear into the deeper tissue can be seen.

6. Ultrasound of the Heart: The CURLS Protocol

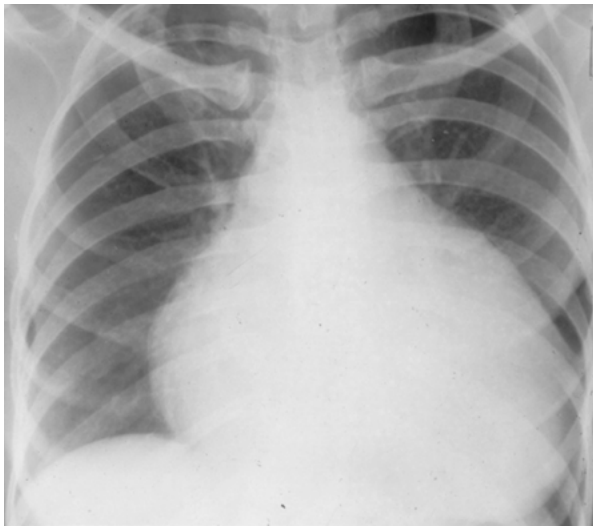


Figure 6.1. Cardiomegaly on a chest x-ray.

Introduction

Ultrasound is an excellent tool for evaluating the aetiology of **dyspnoea, peripheral oedema,** and the source of **cardiomegaly seen on a CXR** (Figure 6.1). Congestive heart failure (CHF) is probably the most common cause of these symptoms and findings; however, there are also other causes, which are treated differently.

In an HIV-prevalent tropical setting, common cardiac causes of dyspnoea and oedema are **pericardial effusion with tamponade** (most often due to TB), **dilated cardiomyopathy** (for example, peripartum or HIV-cardiomyopathy), **post-rheumatic heart disease** (causing mitral valve disease in particular), **cor pulmonale,** and **hypertensive heart disease.** The findings for these conditions were combined to form the **Cardiac Ultrasound for Resource-Limited Settings (CURLS) protocol** (Table 6.2). For lower limb oedema and ascites, further diagnoses may warrant consideration, such as hepatic cirrhosis, KS and other malignancies, kidney disease, venous thrombosis, and filariasis.

Technique and typical ultrasound findings

Echocardiographic examination of the heart is generally done using intercostal and parasternal approaches (or windows) with a phased array transducer. An overview is also possible from the abdomen, using a **3.5 MHz convex transducer.** This view is obtained using a **subxiphoid position,** with the convex transducer in a transverse orientation, tipped cranially, and aimed towards the patient's left shoulder. In this position, the **liver acts as an acoustic window.** In the subxiphoid view (Figure 6.3), the right heart is closest to the surface; the left heart is visible in the far field. The normal heart is surrounded by the **echogenic pericardium;** however, minuscule amounts of pericardial fluid can sometimes be seen as a black line next to the right atrium. Normally, the size of the left ventricle is approximately double that of the right ventricle; the atria are smaller than the ventricles. The septum between the ventricles usually has a thickness of 11 mm or less at the end of the diastole (when the cardiac cavity is largest). Finally, ultrasound of the heart should include a **view of the inferior vena cava and the liver veins** (Figure 6.4) to get a general idea of **intravascular volume status.**

In some cases, a sufficient subcostal view cannot be obtained. When this happens, it may be helpful to attempt an intercostal ultrasound of the heart. (As mentioned, though well-defined intercostal views are used

Table 6.2. The CURLS protocol

Question	Interpretation
1. Is a pericardial effusion present?	Yes: Consider cardiac tamponade. No: Consider other diagnoses.
2. Is left ventricular function reduced?	Yes: Consider causes of cardiomyopathy. No: Consider other diagnoses.
3. Is the right ventricle larger than the left ventricle?	Yes: Consider pulmonary artery hypertension or pulmonary embolism. No: Consider other diagnoses.
4. Is the left atrium larger than the left ventricle?	Yes: Consider mitral stenosis or regurgitation, possibly caused by rheumatic heart disease. No: Consider other diagnoses.
5. Is the left ventricle wall (septum) thicker than 12 mm?	Yes: Consider hypertension or aortic stenosis/regurgitation. No: Consider other diagnoses.

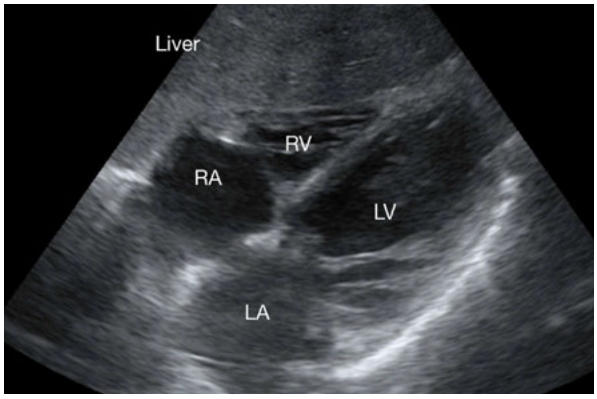


Figure 6.3. Subxiphoid view: normal heart (RV=right ventricle, LV= left ventricle, RA = right atrium, LA= left atrium).

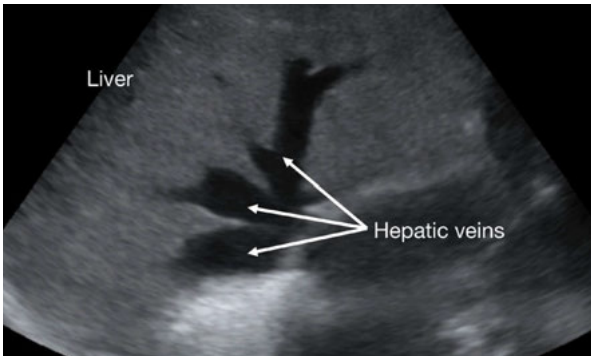


Figure 6.4. Hepatic vein confluence in transverse scan.

in normal echocardiography, these views are beyond the scope of this text. A brief description for those interested is provided in the box at the end of the chapter.)

Pathological findings

The CURLS protocol is designed to offer a targeted approach to cardiac ultrasound in resource-limited settings, like Sub-Saharan Africa. The protocol prioritises evaluation based on the **epidemiology of cardiac disease,** as well as on the **impact on treatment decisions.**

The discussion of pathological findings in this protocol follows the answers to five important questions (shown earlier in Table 6.2).

1. Is a pericardial effusion present?

Pericardial effusion is easily identified in a subxiphoid view; it usually presents as an **anechoic rim around the heart** (Figure 6.5) (also see Chapter 4). If a significant pericardial effusion is present, the heart should be evaluated for signs of tamponade. If **jugular veins are distended** and the patient has **tachycardia** and **low blood pressure,** obstructive shock and tamponade may be present. **Right atrial systolic collapse** and **right ventricle (RV) diastolic collapse** (Figures 6.6 and 6.7) suggest tamponade physiology. It is important to be aware that if the rate of fluid accumulation is rapid, even a small effusion can cause haemodynamic compromise. In a patient with cardiac tamponade, emergency pericardiocentesis can be life-saving. In resource-limited settings, an ultrasound-guided intercostal approach with a simple cannula can be used.

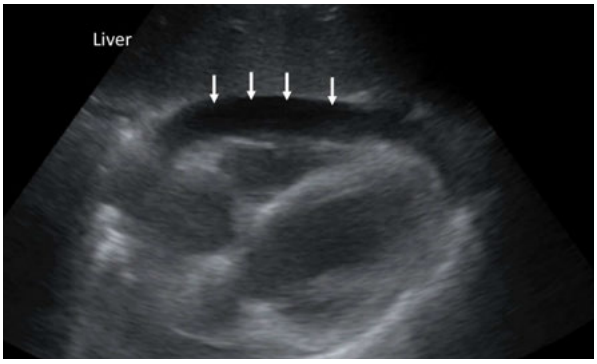


Figure 6.5. Subxiphoid view: Anechoic pericardial effusion.

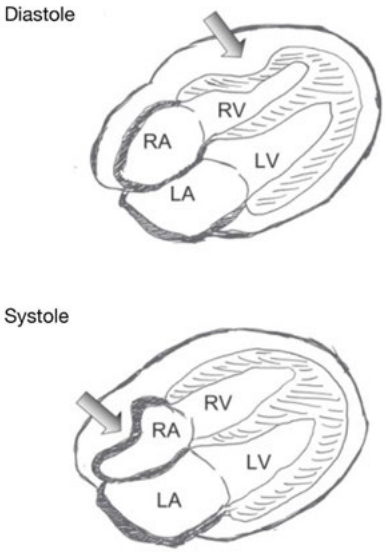


Figure 6.6. Tamponade with diastolic compression of the right ventricle (Diastole), and systolic compression of the right atrium due to increased pressure in the pericardial sac (Systole).

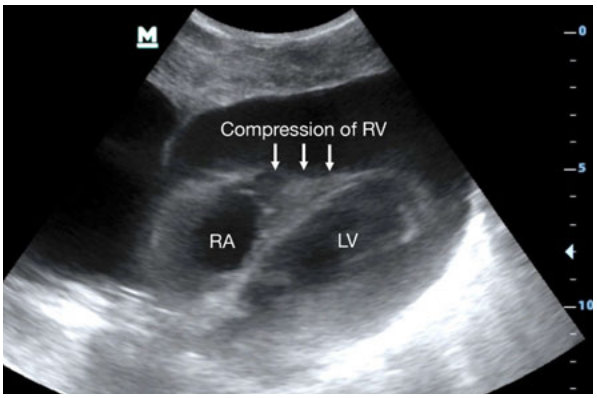


Figure 6.7. Subxiphoid view: large effusion with compression of the right ventricle.

2. Is left ventricular function reduced?

Dilated cardiomyopathy can have multiple causes, but sonographically, the end stage is similar, with **global enlargement of both atria and ventricles** (Figure 6.8), plus reduced left ventricular systolic function. Unfortunately, most symptomatic patients present in a late stage of the disease. Formal measurement of the ejection fraction and assessment of regional wall motion abnormalities is mostly limited to referral centres, as these require dedicated echocardiographic software and substantial experience. However, a broad classification of left ventricular contractility (as hyperdynamic, normal, moderately impaired, and severely impaired) can be made by **estimating whether contraction is symmetrical towards the centre,** whether the myocardium thickens as it contracts, and whether the mitral valve opens normally during diastole. In an enlarged heart, this can usually be achieved through a subxiphoid view.

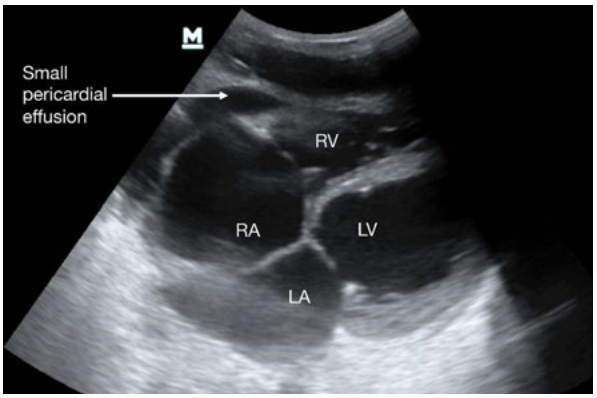


Figure 6.8. Subxiphoid view: dilated cardiac chambers and reduced contractility of the left ventricle (only visible in moving clip) due to cardiomyopathy.

3. Is the right ventricle larger than the left ventricle?

An enlarged RV can indicate **cor pulmonale** (Figure 6.9). Cardiac ultrasound can demonstrate sonographic features of cor pulmonale by evaluating the shape, size, and pressure of the right side of the heart. In the subxiphoid view, **the RV is typically half to two-thirds the size of the normal-sized left ventricle (LV);** any larger suggests RV enlargement. If the **RV is equal in size to the LV,** it is considered **moderately enlarged.** Pronounced dilatation of the RV to the extent that it is **larger than the LV** is considered **severe RV enlargement,** and cor pulmonale may be considered. In addition, movement of the intraventricular septum away from the RV indicates increased right ventricular pressure or volume, resulting in a D-shaped LV in the (optional) parasternal

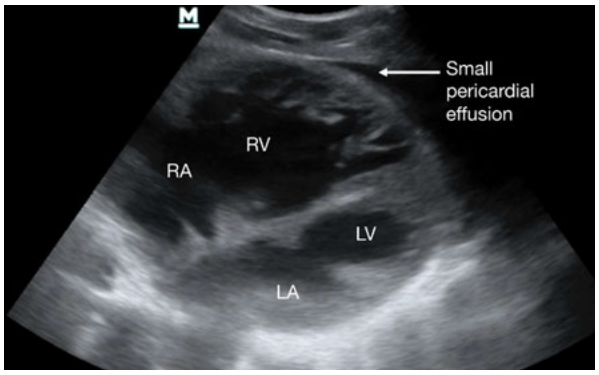


Figure 6.9. Subxiphoid view: enlarged right ventricle and atrium in cor pulmonale.

short axis view. The measurement of RV contractility and the calculation of the pressure gradient in the setting of tricuspid regurgitation can be helpful. However, because this procedure is technically more difficult and requires Doppler software, it is best performed at referral sites.

4. Is the left atrium larger than the left ventricle?
An isolated, **enlarged left atrium (LA)** is a feature of **mitral stenosis or regurgitation,** which may serve as a surrogate marker for **rheumatic heart disease (RHD)** (Figures 6.10 and 6.11). The LA often has become larger than the normal-sized LV by the time the patient presents. Additional findings include a thickened mitral valve with a **diastolic hockey stick-like appearance** or a **'doming' of the valve leaflets.** The right heart may also be enlarged due to the subsequent increase in pulmonary pressures. Post-rheumatic changes of the aortic

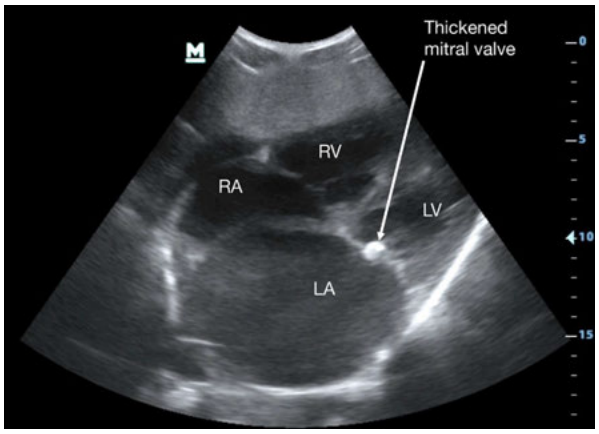


Figure 6.10. Subxiphoid view: enlarged left atrium and thickened mitral valve in post-rheumatic mitral valve disease.

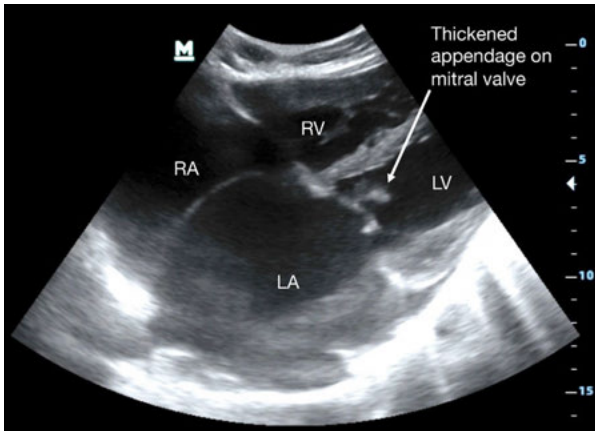


Figure 6.11. Subxiphoid view: another example of predominantly enlarged left atrium and thickened mitral valve in post-rheumatic mitral valve disease.

valve are slightly more difficult to detect, as they initially lead to left ventricular hypertrophy only, and the aortic valve is more difficult to visualise on ultrasound from a subxiphoid view. Morphological assessment of the aortic valve can be done at referral centres, as can detection of mitral and aortic regurgitation (if colour Doppler is available). For the basic CURLS exam, these questions are not addressed.

5. Is the left ventricle wall (septum) thicker than 12 mm?
Left ventricular hypertrophy (Figure 6.12) can be determined by measuring the **intraventricular septum (IVS) at end diastole.** The standard measurement of the IVS is performed in the parasternal long axis view; it should be **less than 12 mm.** Nevertheless, the septum is generally visible in the subxiphoid view. The clinician should freeze the screen and scroll back to end diastole, where the IVS is measured at the level of the mitral valve leaflet tips.

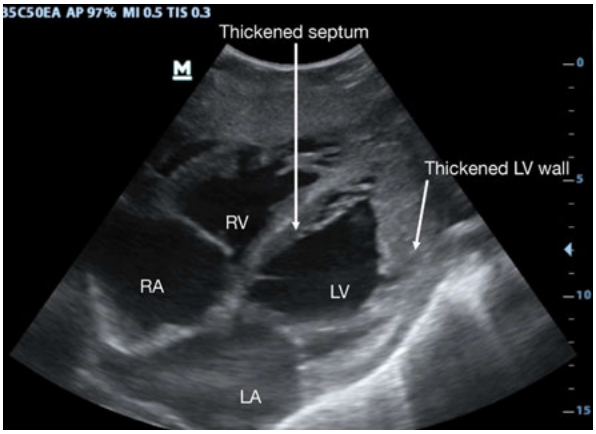


Figure 6.12. Subxiphoid view: thickened wall of the left ventricle in hypertrophic heart disease (e.g., due to hypertension or aortic valve stenosis).

Diagnostic and therapeutic implications

Pericardial fluid
Treatment should aim to address the underlying cause of pericarditis, which is usually **TB** or **malignancy** (such as KS in HIV patients). After careful examination for KS lesions on skin and palate, the presence of pericardial effusion may prompt empiric treatment for TB, especially in HIV patients with severe immune suppression. **Corticosteroids** may be given in addition to antibiotic treatment for TB pericarditis, although their impact on outcome is still a subject of debate. A recent multicentre study found that prednisolone had no significant effect on overall risk of death, cardiac tamponade requiring pericardiocentesis, or constrictive pericarditis in patients with tuberculous pericarditis. However, prednisolone therapy was associated with significant reductions in the incidence of constrictive pericarditis and hospitalisation. In our experience, patients often show faster clinical improvement with adjunctive prednisolone. We therefore prescribe **prednisolone for four to eight weeks,** especially in cases with cardiac tamponade, despite the fact that HIV patients who receive steroids may have a small but significant increase in risk of developing HIV-associated malignancies. Colchicine, while generally effective for the treatment of acute and recurrent pericarditis, seems to be of no benefit in TB pericarditis.

Congestive heart failure
Dilated cardiomyopathy is a common cause of heart

failure in Sub-Saharan Africa, accounting for approximately 20% of cases. (Often, the specific underlying disease remains unknown in the individual patient.) Causative factors include **myocarditis, HIV-associated cardiomyopathy, alcohol abuse, nutritional deficiency, thyrotoxicosis, and pregnancy.** In some areas, **iron overload** and other metabolic factors (vitamin B deficiency) may also play a role. HIV-associated cardiomyopathy is reported in 9–57% of HIV patients in Africa. Sonographically, the end stage is similar in all of the above-mentioned diseases.

Patients with impaired contractility and fluid overload will usually be started on **furosemide,** a widely available **loop-diuretic** drug. Administering **oxygen** is helpful in acute cases. Long-term management of CHF should include the addition of an **angiotensin-converting enzyme (ACE) inhibitor** and a **beta-blocker,** both of which have been shown to have a mortality benefit in cases of systolic heart failure. Additionally, **spironolactone** could be added; however, in these cases the patient's potassium must be monitored. All of these drugs are considered essential, so they are often readily available.

Cor pulmonale

The reported prevalence of cor pulmonale in African countries varies; its causes are often difficult to determine. Contributing to the problem is that right-sided heart failure can be difficult to diagnose through history and physical examination alone. Patients will often present with complaints of dyspnoea on exertion, breathlessness, and lower extremity swelling. The underlying causes of cor pulmonale differ between high- and low-resource settings. Although **chronic obstructive bronchitis or emphysema** and **pulmonary hypertension secondary to left-sided heart failure** are seen in both settings, chronic fibrocavitary TB (also known as **post-tuberculosis lung disease (PTLD)** or **tuberculosis-destroyed lung (TDL)** is a frequent underlying cause in Africa. The role of chronic pulmonary vascular diseases, including **pulmonary schistosomiasis,** as a cause of cor pulmonale depends on local epidemiology. **Pulmonary embolisms** can be a cause of acute cor pulmonale; these are more frequently seen in patients with HIV, who also seem to have more extensive pulmonary thrombi on computer tomography compared with HIV-negative patients. Finally, patients with HIV may suffer from **HIV-associated primary pulmonary artery hypertension.**

Identification of features of cor pulmonale should prompt a diagnostic workup for thrombosis (ultrasound of the veins) and related pulmonary embolism, which may in turn prompt anticoagulant treatment. In other cases, use CXR to look for underlying lung disease, and treat if possible.

Calcium channel blockers can help to reduce pulmonary pressure; these can also be tried. **Sildenafil** may have a role for selected patients with pulmonary hypertension associated with pulmonary disease. The medication seems to be well tolerated, improving exercise capacity for some patients. However, this medication is not widely available, and must be bought privately.

Rheumatic heart disease

In contrast to high-resource settings, where valvular disease is largely degenerative in origin, valvular disease in Africa is almost invariably the result of infectious disease. Infectious valvular disease can develop either directly, as with **infective endocarditis,** or indirectly, following rheumatic fever. **RHD** is often encountered in school-aged children or young females of childbearing age. The course of RHD is usually much more rapid than in degenerative disease and predisposes the patient to cardiac failure, secondary endocarditis, **atrial fibrillation,** and **stroke.** Most patients with RHD have mixed valvular lesions. Mixed mitral valve stenosis and regurgitation are frequently seen. Aortic regurgitation is seen in less than 50% of the patients; when found, it is almost always in combination with mitral valve disease. (Aortic stenosis is rare.) Ideally, patients with severe mitral RHD should be referred for **valvuloplasty or valve surgery;** unfortunately, this procedure is rarely available in resource-limited settings. Patients must be treated conservatively to mitigate symptoms and minimise progression. Diuretics are required; heart rate control (for example, with **beta blockers or digoxin**) can be attempted, especially if atrial fibrillation is present. Patients with atrial fibrillation, history of ischaemic stroke, or left atrial thrombus should receive **anticoagulants** to reduce the risk of systemic embolisation. Patients should also be given **regular long-acting penicillin** for secondary prevention of rheumatic fever.

Hypertensive heart disease

The prevalence of hypertension is high in many African settings, especially urban areas, and increases with age. Studies have shown that only 40% of people with hypertension are diagnosed, fewer than 30% are put on any drug treatment, and in even fewer cases is blood pressure adequately controlled. Hypertension is therefore a frequently identified aetiology for cardiac failure. Patients who develop **LV hypertrophy** are at increased risk for cardiovascular events. After identifying LV hypertrophy, it is often helpful to **assess kidney size; small, hyperechoic kidneys may point to either a renal cause of hypertension or to chronic kidney injury** due to long-standing high blood pressure. Conspicuous kidneys should prompt further investigation into the presence of hypertension, which can be treated according to local guidelines. Different classes of antihypertensives may be available, even in resource-limited settings. **Diuretics** (mostly hydrochlorothiazide), **calcium channel blockers** (such as amlodipine), and **ACE inhibitors** (such as enalapril) are among the WHO-designated essential drugs that may be used with these patients. Combinations of drugs are often required; given that blood pressure control requires titration of medications, follow-up is essential.

Pearls and pitfalls

- When using the subxiphoid view, increase scan depth to cover the entire outline of the heart.
- When the heart does not come into view in the subxiphoid position, flatten the probe against the abdominal wall and have the patient bend their knees to relax the abdominal wall. If the view is obstructed by the stomach, slide the probe to the right to use the liver as an acoustic window.

Transcostal cardiac views: an introduction

Echocardiography, or ultrasound of the heart with a phased array probe, generally uses four views, also called 'windows':

- *the subxiphoid view (as described above and shown in Figure 6.13 below),*
- *the left parasternal long axis view,*
- *the left parasternal short axis view, and*
- *the apical four-chamber view.*

These views require more skill and training; therefore, this section is for the clinician who already has advanced training and experience in ultrasound.

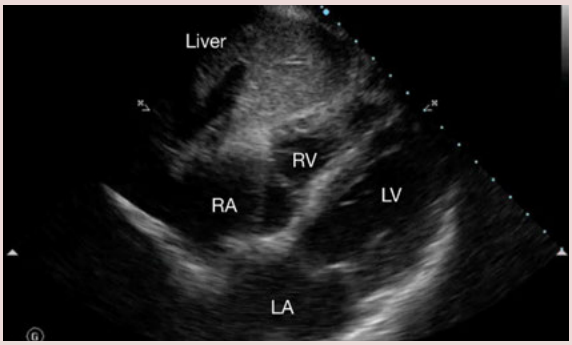


Figure 6.13. Subxiphoid view: RV=right ventricle, LV= left ventricle, RA = right atrium, LA= left atrium.

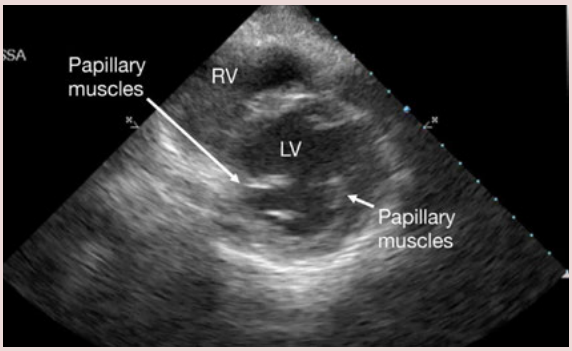


Figure 6.15. Parasternal short view: LV = left ventricle.

The **parasternal long axis view** (Figure 6.14) is found by placing the probe in the third or fourth intercostal space, directly to the left of the sternum, and turning the probe to align between the right shoulder and the left hip. The angle of the probe is then adjusted to visualise the **mitral valve, aortic valve, and if possible, the apex of the LV** in the same view. The parasternal long axis is well suited for differentiating pericardial and pleural effusions. Large pleural effusions can appear to surround the heart, but will taper to the descending aorta, whereas pericardial effusions will continue anterior to the descending aorta. Several measurements can be taken; this view can be used to assess left ventricular contractility.

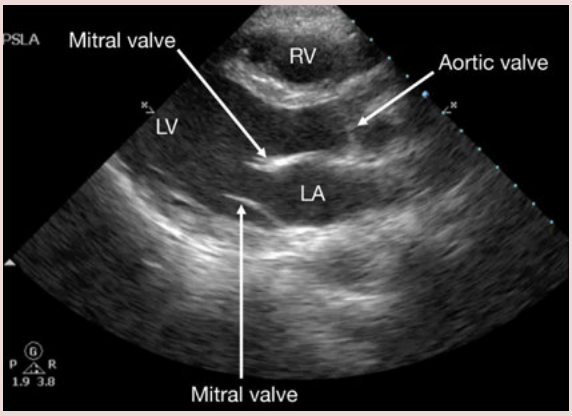


Figure 6.14. Parasternal long view: LV = left ventricle, RV = right ventricle, LA = left atrium.

From the parasternal long axis, with the mitral valve in the centre of the image, the probe is rotated 90 degrees clockwise to obtain the **parasternal short axis view** (Figure 6.15), visualising the **left chamber** and **mitral valve** in a circumferential plane, with the right chambers adjacent. This view can provide additional information about **left ventricular contractility** and right ventricular size and pressure.

The **apical four-chamber view** (Figure 6.16) is obtained at the apex of the heart—which is usually located below the nipple, in the fifth intercostal space, but can be displaced in patients with cardiomegaly. This view can also be found by sliding the probe down towards the apex from the parasternal short axis, and then tilting it towards the patient's right shoulder. This view is the best for **assessing the size and dimensions of the atria and ventricles.**

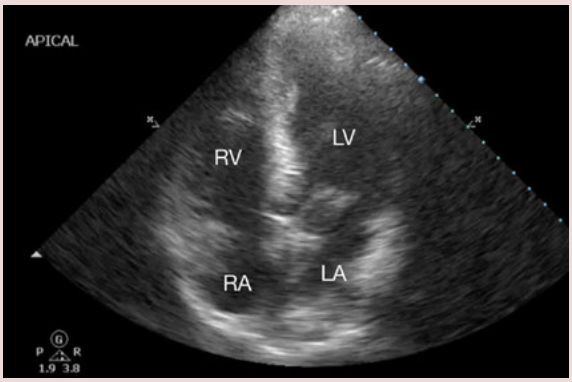


Figure 6.16. Four-chamber view: RV=right ventricle, LV= left ventricle, RA = right atrium, LA= left atrium.

Several protocols for point-of-care cardiac ultrasound have been developed in Europe and the US, including the Focused Assessment of Transthoracic Echo (FATE) protocol. A free card with the different views and pathologies can be downloaded at *https://usabcd.org/fate-card/*.

Material for this chapter adapted from:
Huson MAM, Kaminstein D, Kahn D, et al. Cardiac ultrasound in resource-limited settings (CURLS): towards a wider use of basic echo applications in Africa. *Ultrasound J. 2019 Dec 27;11(1):34. doi: 10.1186/s13089-019-0149-0.*

7. Ultrasound of the Veins: Compression Ultrasound for Deep Vein Thrombosis

Introduction

Deep vein thrombosis (DVT) is a common clinical problem amongst HIV patients. DVT has a significant mortality rate, given its potential for **pulmonary embolism.** HIV patients are up to ten times **more likely to develop venous thrombosis** than HIV-negative individuals of the same age. Advanced disease is associated with a further increase in the incidence of thrombotic events.

This higher risk could be explained by the presence of a **hypercoagulable state,** characterised by an increase in procoagulant factors, endothelial tissue factor expression, and thrombogenic microparticles. In HIV patients, microparticles originate from CD4+ lymphocytes as a direct result of HIV infection, and possibly as a reflection of CD4+ lymphocyte cell death. A decrease in anticoagulant factors, including antithrombin III and factors in the protein C pathway, may add to the pathophysiology. Several **other diseases**—including **cancer, stroke, nephrotic syndrome, and TB**—are also associated with **increased risk** of thromboembolic disease.

The signs and symptoms of DVT include **calf tenderness** and **unilateral limb swelling** (Figure 7.2). If pulmonary embolism is present, **tachycardia** and **tachypnoea** may also be seen. Bedside assessment of DVT is one of the most useful POCUS applications; it is easy to perform and has immediate therapeutic consequences.

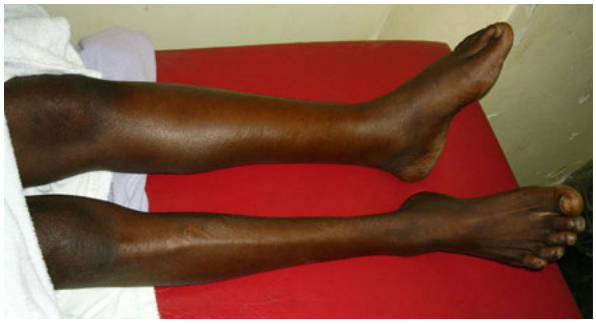


Figure 7.1. Unilateral swelling of the left leg is suggestive of DVT.

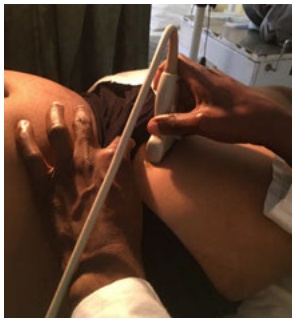


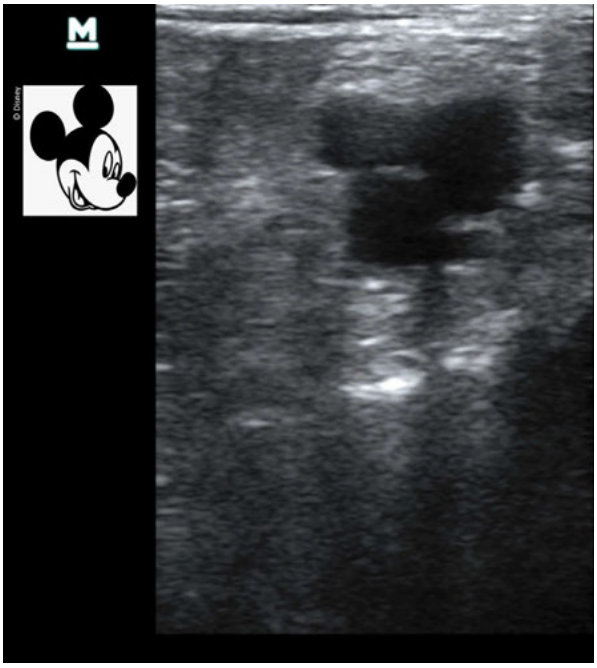
Figure 7.2. Scan of the inguinal vessels (ideally with a linear transducer). Gentle compression will make the venous vessels collapse.

Figure 7.3. Scan of the popliteal vessels. Mild flexion of the knee allows for easier compression.

Technique and ultrasound findings

A simplified protocol DVT screening has been developed by emergency physicians. It includes assessment of the **common femoral vein in the groin** (Figure 7.3), and of the **popliteal vein at the knee** (Figure 7.4). A 5 to 10 MHz transducer is used; **linear transducers** are more suitable for compression. The leg should be bent at the knee and turned outward; if possible, the end of the stretcher with the patient's feet should be lowered to make the examination easier. The **affected leg is scanned;** if necessary, it can be **compared to the other leg.** In fact, it may help to scan the other leg as well, as thrombosis is often a bilateral disease, meaning an otherwise unexpected thrombosis may be found. Clinically, however, this has little effect on therapy in our setting.

With compression ultrasound, the lumen of the proximal part of the vessel is localised in the groin using



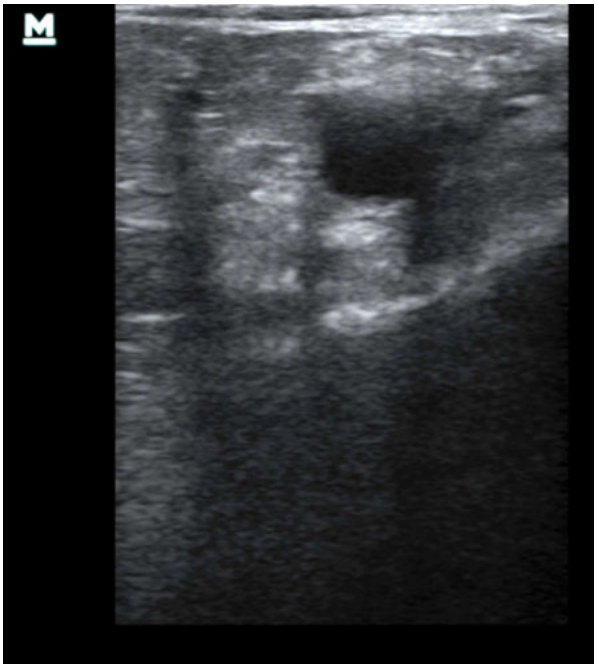
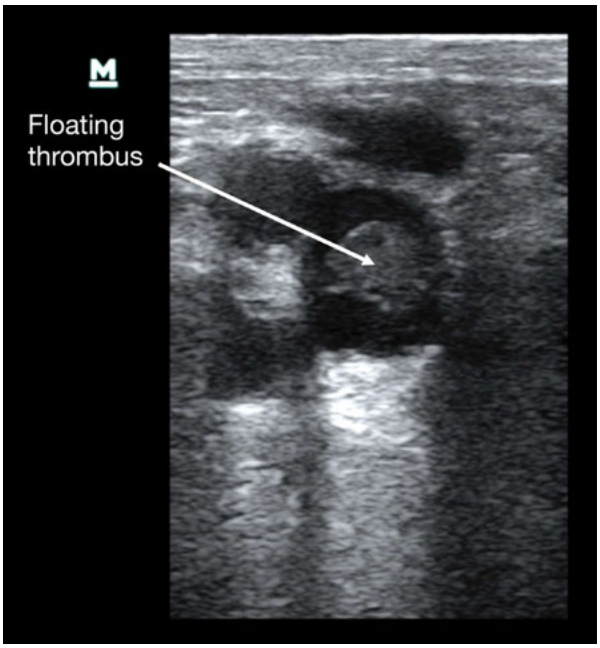


Figure 7.4. Normal inguinal vessels: (L) Without compression, we see a Mickey Mouse-like shape. (R) With compression, the veins collapse.



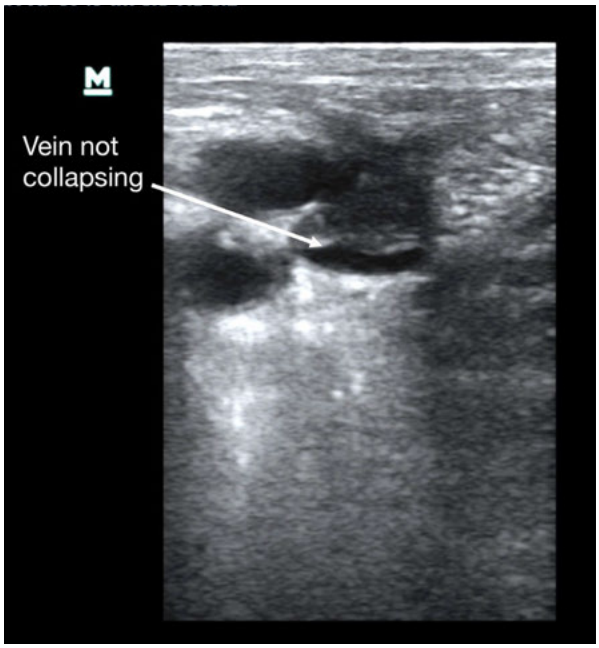


Figure 7.5. Inguinal vessels: (L) Without compression, a floating echogenic thrombus is seen in the femoral vein. (R) With compression, the lumen of the vein does not collapse completely.

minimal pressure. (Make sure to use enough gel to enable acoustic coupling!) The veins are scanned on a **transverse plane;** they will appear as anechoic tubular structures. Once visualised, **mild compression should be sufficient** to cause the **lumen of the vessel to disappear** completely, unless clotted material is present in the vessel (Figure 7.3). Pressure is then released, and the same procedure used to check the popliteal vein in the knee. Thrombosis is confirmed when the vein contains **hyperechoic clot** and is **not compressible** with the probe when applying enough pressure to deform the adjacent arterial wall (Figure 7.5).

If available, colour flow mapping may help to image the vessels and the intraluminal material. It is important to remember that **partial compression is not enough to exclude DVT.** Though the presence of a venous clot in the superficial veins (saphenous vein) is not an uncommon finding, it clearly poses a lower risk of embolism unless it is propagating to the saphenofemoral junction. Occasionally, slow flow can create the impression of 'smoke' in the lumen; this should not be mistaken for a clot. In **acute thrombosis,** the **vein is generally distended,** whereas chronic occlusion leads to narrowing of the occluded vein.

Diagnostic and therapeutic implications

The presence of DVT warrants **immediate treatment with anticoagulants** to prevent further thromboembolic complications. **Heparin injection** and (if possible) oral anticoagulation with **warfarin** should be initiated.

Treating HIV patients for DVT may prove difficult in resource-poor settings; drug interactions must be considered when treating patients for concomitant TB or HIV. **Rifampicin** induces hepatic enzymes and **may lead to suboptimal levels of anticoagulants.** Nevirapine and **efavirenz** have **unpredictable effects;** both have been reported to reduce warfarin levels, but they can also increase levels, thus increasing the risk of haemorrhage. The amount of warfarin needed by the individual patient must be determined through **international normalised ratio (INR) monitoring,** which may be difficult to coordinate.

Pearls and pitfalls

- Have the supine patient rotate their leg externally to better visualise the common femoral vein.
- To exclude DVT, make sure that the vein fully collapses, with the walls falling together. If the vein compresses only partially, repeat the manoeuvre; if partial compression persists, consider DVT as a diagnosis.
- Lymph nodes in the groin and Baker's cyst in the popliteal fossa can mimic vessels in the transverse plane; they can easily be identified by rotating the probe to a longitudinal view.
- A negative scan result in a patient who has leg swelling without an obvious alternative cause does not exclude DVT; repeating the exam in five to seven days may detect a DVT that originated in the deep calf veins and subsequently propagated to the larger veins.

8. Ultrasound for Lower Abdominal Pain in Female Patients

Introduction

When women of childbearing age present with **pelvic pain** or **vaginal bleeding** (with or without hypotension), **ectopic pregnancy** must always be excluded. Ectopic pregnancy is **more frequently seen in women with HIV**—possibly due to scarring and injury of the fallopian tubes caused by other sexually transmitted diseases. In resource-limited settings, **pelvic inflammatory disease** associated with sexually transmitted diseases is the most important risk factor for ectopic pregnancy. In African countries, hospital-based studies have reported ectopic pregnancy case mortality rates of around 1–3%, ten times the rates reported in Western countries.

The main reasons for such high mortality rates are late diagnosis, which in almost all cases leads to major complications, and delayed emergency surgical treatment. POCUS as a diagnostic test for ectopic pregnancy provides excellent sensitivity and negative predictive value; visualisation of an intrauterine pregnancy (IUP) is generally enough to rule out ectopic pregnancy. The aim of POCUS in the first trimester is thus to **rule in (confirm) IUP,** and thereby **rule out ectopic pregnancy.**

Technique and ultrasound findings

Transabdominal scans are performed using a **3.5 MHz curvilinear probe.** Ideally, the patient should have a full bladder for this scan. The probe is placed **superior to the pubic symphysis;** both longitudinal and transverse views must be obtained.

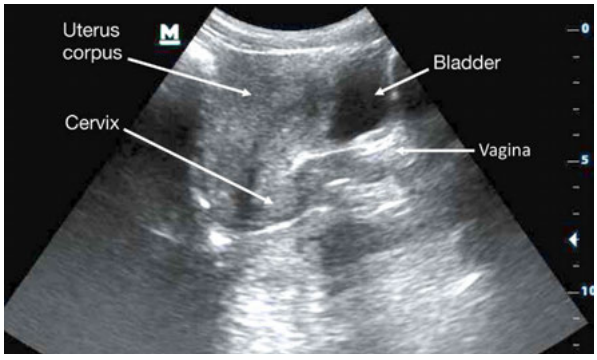


Figure 8.1. Female pelvic longitudinal: The anteflexed uterus is visible cranially of the bladder. The area of the cervix is visible.

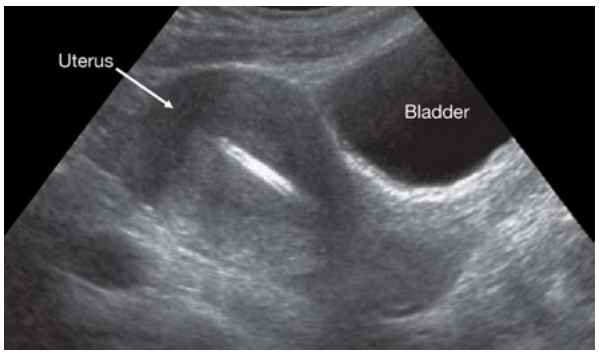


Figure 8.2. Intrauterine device (IUD): Seen as an unusual echogenic straight line in the centre of the uterus.

The **longitudinal view** (Figure 8.1) visualises the **bladder,** the **uterus** in the craniocaudal axis, and the vaginal stripe. In most patients, the uterus is anteflexed. In some patients with intrauterine anticontraceptive devices (IUDs), the IUD can be seen as an echogenic straight line in the centre of the uterus (Figure 8.2). The **transverse view** (Figure 8.3) visualises the **bladder,** the **uterus** in an axial view, and the **ovaries and adnexa** lateral to the uterus. To assess the entire uterus in the transverse view, the beam must be angled craniocaudally; the ovaries can be difficult to identify, and the fallopian tubes are rarely seen unless they are pathologically enlarged (see the box at the end of this chapter). If a transabdominal scan does not provide all the necessary information, it should be complemented by a transvaginal scan (if available).

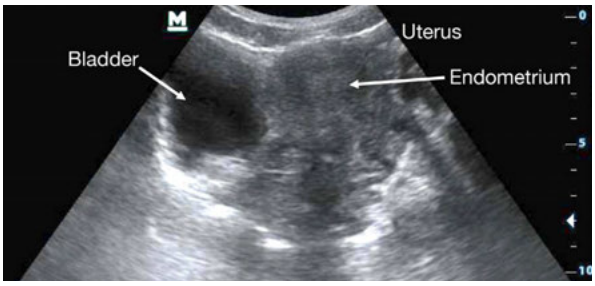


Figure 8.3. Female pelvic transverse: The uterus is seen anteflexed slightly to the right of the bladder. The slightly echogenic endometrium is visible in the centre of the uterus.

Any of the following three findings establishes the diagnosis of a definitive IUP when visualised inside the uterus:

1) Yolk sac

The **yolk sac** is a sac of 5–6 mm in diameter located within a bigger sac called the gestational sac. Both the gestational sac and the yolk sac are anechoic, round structures with an echogenic rim. The yolk sac becomes visible six to seven weeks after the last menstrual period; it then regresses and disappears by 12 weeks of gestation. To ensure that the gestational sac with its nested yolk sac is located within the uterus, the myometrial mantle should be clearly visualised.

2) Fetus

The fetal pole becomes visible by seven weeks, as an echogenic structure between the yolk sac and gestational sac (Figure 8.4).

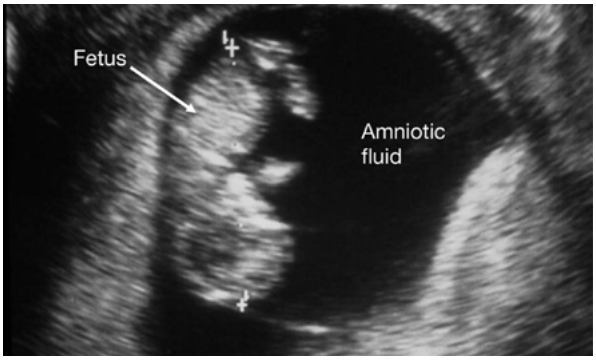


Figure 8.4. The fetus is visible in the anechoic amniotic fluid. In early pregnancy, the crown–rump length (measured using callipers) can be used to date the age of the fetus.

3) Intrauterine heartbeat

By the end of the seventh week of gestation, the embryo is 5–10 mm long, and cardiac motion should be visible.

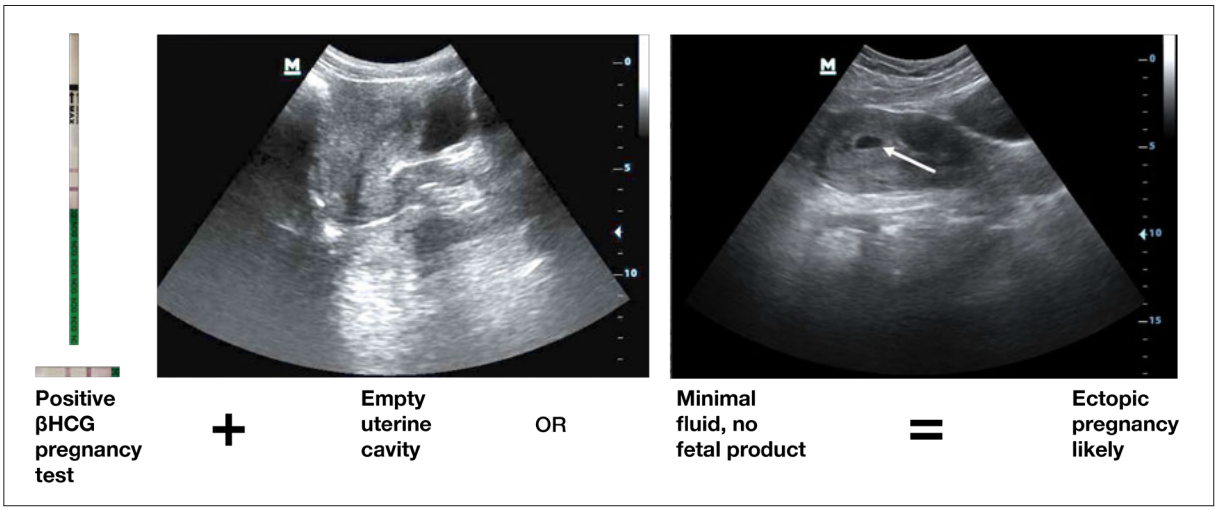


Figure 8.5. A positive ßHCG pregnancy test and a cavum uteri (see Fig. 8.2) that is either empty (L), or contains only minimal fluid (R) but no fetal product, are highly suggestive of ectopic pregnancy.

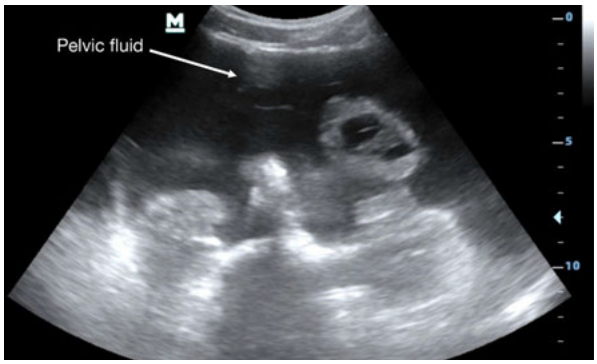
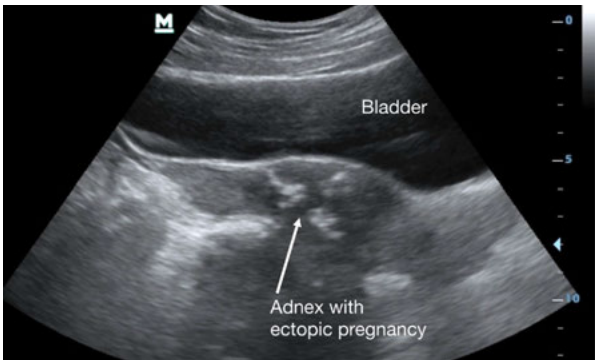


Figure 8.6. (L) Free pelvic fluid is often the only abnormal finding in ruptured ectopic pregnancy.



(R) Sometimes the ectopic pregnancy itself can be seen beside the uterus in the adnexal area.

Pathological findings on transabdominal sonography are **failure to obtain the above-mentioned findings** of IUP, **together with a positive βHCG-pregnancy test (urine strip pregnancy test)** (Figure 8.5). In cases of ectopic pregnancy, free fluid may be present in the pouch of Douglas, especially as a sign of rupture (Figure 8.6). If no IUP is seen, the abdomen should be evaluated for free fluid.

Diagnostic and therapeutic implications

Failure to identify a definitive IUP through transabdominal ultrasound in patients with a positive βHCG test is due to either an extremely early normal IUP, an abnormally developing IUP, an active or complete miscarriage, or an ectopic pregnancy. **In all of these cases, the patient should be immediately referred to gynaecology for further evaluation.** Unclear or pathologic findings warrant a formal obstetric consultation and (if available) a transvaginal scan. If the patient's condition is unstable, emergency management (IV-line placement and fluid bolus) is necessary; this also includes timely surgical intervention and possible blood transfusion.

Pearls and pitfalls

- The goal of a first-trimester POCUS in women of childbearing age with pelvic complaints is to confirm IUP, and thereby EXCLUDE ectopic pregnancy. If you cannot confirm IUP, you should suspect ectopic pregnancy.
- Pelvic structures are often not midline; scan from right to left to find the best plane.
- Identifying the gestational sac alone is not confirmation of normal IUP gestation; the sac can also be present in an ectopic pregnancy. Either a yolk sac or a fetus must be seen within the gestational sac to diagnose IUP.
- Beware of the limitations of transabdominal ultrasound in early pregnancy; transabdominal ultrasound is less sensitive than transvaginal ultrasound.
- Hypotension should increase your suspicion of a ruptured ectopic pregnancy.
- Do not only look for free fluid in the pouch of Douglas; also perform a FAST examination.

Adnexal masses

*Adnexal masses are **not easy to find,** due to unclear anatomy. When they are found, it may be difficult to determine exactly what they are. Therefore, patients with suspected pathology in this region should be referred to gynaecology. Nevertheless, some basic facts and knowledge may be helpful for the clinician with more advanced ultrasound skills.*

Bacterial abscesses

Patients typically present with a combination of **fever, elevated inflammatory markers, lower abdomino-pelvic pain, and vaginal discharge.** Fever and leucocytosis may sometimes be absent. Abscesses are often polymicrobial, with a preponderance of anaerobic organisms.

Transabdominal ultrasound findings may include **multilocular complex retro-uterine or adnexal masses** with septations and irregular walls (Figure 8.7). These masses are **commonly bilateral.** Fluid and echogenic debris may be seen in the pouch of Douglas. The initial treatment is antibiotic therapy.

TB abscesses

Women with TB involvement of the genital tract can present with **menstrual irregularity, infertility, abdominal pain,** and pelvic inflammatory disease. Ultrasound may show **tubo-ovarian abscesses;** extension of these collections to extraperitoneal areas can suggest TB. However, the **diagnosis** is often made only after aspiration and **microbiological analysis** of the material.

Ovarian cancer

Ovarian cancer is relatively common. Symptoms of ovarian cancer are usually non-specific; patients may present with a **palpable mass, abdominal pain or distension, weight loss, or vague gastrointestinal symptoms.** Presence of **ascites** is also suggestive of malignant disease; peritoneal metastasis may be a differential diagnosis for abdominal TB. Therefore, **in a female patient with ascites, the ovaries should be assessed.**

Most ovarian cancers are of epithelial type, with serous cystadenocarcinomas being the most common form. Sonographically, they present as **cystic masses in the adnexal area** (Figure 8.8). These cysts may be either **unilocular or multilocular;** in approximately 50% of serous cystadenocarcinoma cases, both ovaries are affected. Features reported to be associated with malignancy include **large size** and thick, **irregular walls and septa,** as well as nodules and **solid elements.** Any case of ascites with (cystic) masses in the ovarian region should be referred to gynaecology for further workup and treatment.

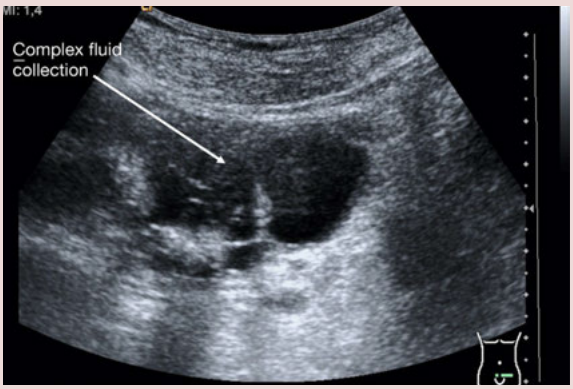


Figure 8.7. Pelvic inflammatory disease and tubo-ovarian abscesses are characterised by complex fluid collections with septa and strands in the adnexal area. These are often painful on palpation.

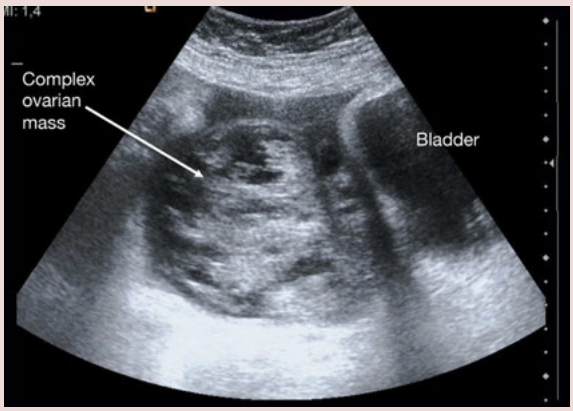


Figure 8.8. Look for ovarian masses in female patients with ascites. Ovarian cancers can be cystic, solid, or complex, and mixed in echogenicity.

9. Ultrasound of the Kidneys and Bladder

Introduction

Renal and **urinary tract disease** is common in **adults and children** throughout all continents. Numerous protocols for point-of-care renal and bladder ultrasound examination have been developed, especially for **patients with flank pain or abdominal pain and urinary symptoms.** These protocols focus on identifying hydronephrosis, bladder distension, and stone disease. The aetiologic spectrum leading to urinary tract obstruction is broad: common causes of urinary tract obstruction in resource-limited settings include **external compression** of the ureters from **lymphadenopathy** due to **malignancy, TB, or other masses; occlusion** of the ureters from **stone disease;** and **chronic infections, such as urinary schistosomiasis.** Calcification at any level along the urinary tract (including the kidneys) may be a **consequence of TB,** arising either from concomitant metabolic conditions or from the side effects of drugs, such as the **protease inhibitor atazanavir.**

Another frequent reason to perform a renal ultrasound is **elevated creatinine** or abnormal urine dipstick results (especially **proteinuria**). In this case, check for signs of chronic kidney disease or HIV-associated nephropathy (HIVAN). If neither of these is present and acute kidney injury (AKI) is likely, ultrasound can help to find signs of **post-renal AKI** (urinary tract obstruction) or **pre-renal AKI** (hypovolemia with collapsing IVC and hyperkinetic, tachycardiac heart). An algorithm you can use to assess kidney problems in patients with HIV appears at the end of this chapter.

Technique and ultrasound findings

Normal kidneys

The kidneys are **retroperitoneal organs** located below the liver on the right side and below the spleen on the left side. The basic architecture of the kidney consists of the **cortex,** the **pyramids,** and the **renal sinus/pelvis** (Figure 9.1).

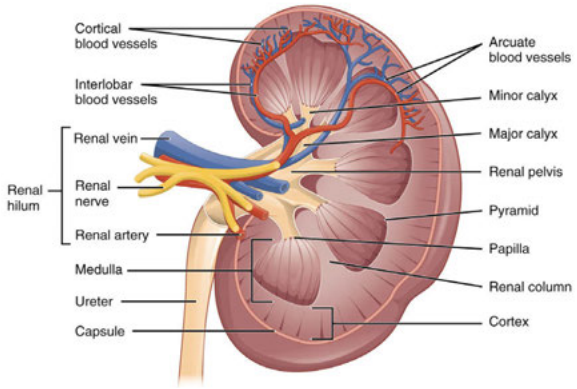


Figure 9.1. Diagram of the anatomy of the kidney. (Source: OpenStax College - Anatomy & Physiology, Connexions web site. http://cnx.org/content/col11496/1.6/, Jun 19, 2013. CC BY 3.0, https://commons.wikimedia.org/w/index.php?curid=30148537.)

Renal ultrasound is usually performed using a curvilinear probe, with the patient in the supine position. Both kidneys are evaluated in longitudinal and transverse views. The right kidney is assessed by placing the probe at the mid-axillary line in the lower intercostal spaces, using the liver as an acoustic window. The left kidney is assessed by placing the probe at the posterior axillary line in the lower intercostal spaces. To obtain the longitudinal view, visualising the **longest craniocaudal diameter** of the kidney, the probe may need to be rotated slightly to maximally lengthen the longitudinal view (Figure 9.2). **Normal kidney size is approximately 10 ± 2 cm,** with the difference between the left and right kidneys usually no more than 1.5 cm.

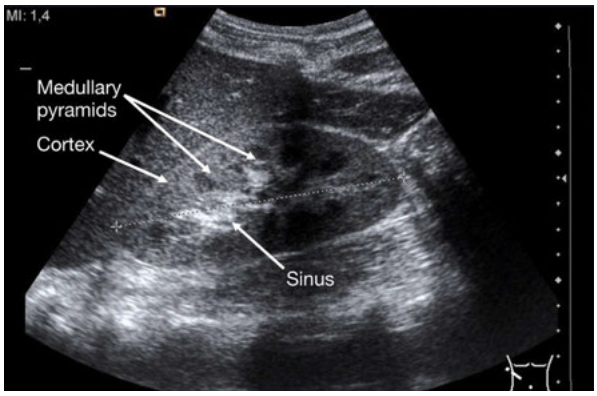


Figure 9.2. Ultrasound of the kidney. The cortex and medullary pyramids can be seen, and centrally the more echogenic sinus, which contains the collecting system, vessels, and lymphatic structures.

To obtain the transverse view, the probe is turned anticlockwise 90 degrees; the probe is then moved or angled up and down to visualise the renal hilum and assess the renal pelvis for dilatation. In a normal image, the **renal cortex and pyramids will appear hypoechoic** (similar to the liver in echogenicity). The **renal sinus** contains the collecting system, renal vessels, lymphatics, fat, and fibrous tissue. The multiple interfaces of these central structures make them **echogenic.** The collecting system surrounds the pyramids, but is not visible in a normal kidney. The proximal ureters are not visible with ultrasound unless they are distended.

Renal cysts

Renal cysts are the single **most frequent focal kidney finding.** About a third of people in their sixties have cysts, but it is a common (even normal) finding even in younger patients. Cysts are usually visible, at around 1 cm in size, and are an **unequivocal sonographic diagnosis:** the **round shape** with **clear wall demarcation** and **anechoic content** with possible acoustic enhancement behind the cyst are characteristic (Figure 9.3).

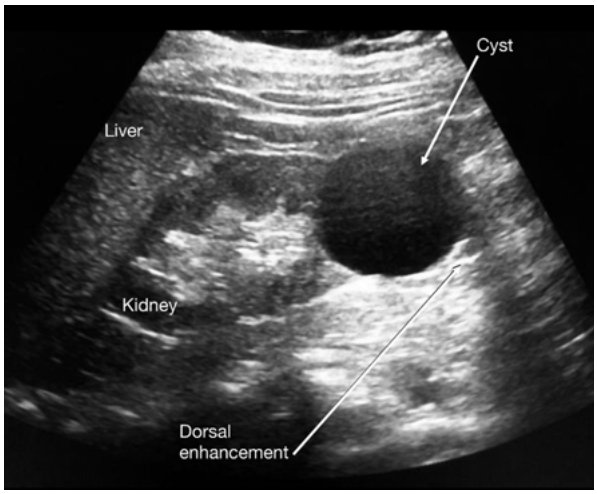


Figure 9.3. Round, anechoic renal cyst at the caudal pole of the kidney.

Hydronephrosis

In hydronephrosis, a dilated renal pelvis presents as an increased **anechoic space at the centre of the kidney,** which is continuous with dilated calyces. Classification of hydronephrosis by degree, depending on the extent of dilation, is commonly practiced (see Table 9.4 and Figures 9.5–9.8).

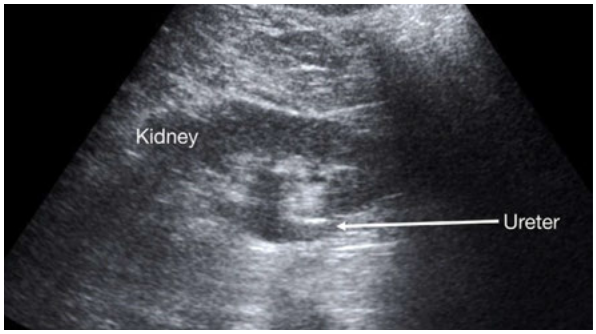


Figure 9.5. Mild hydronephrosis I°: Discrete central lucency.

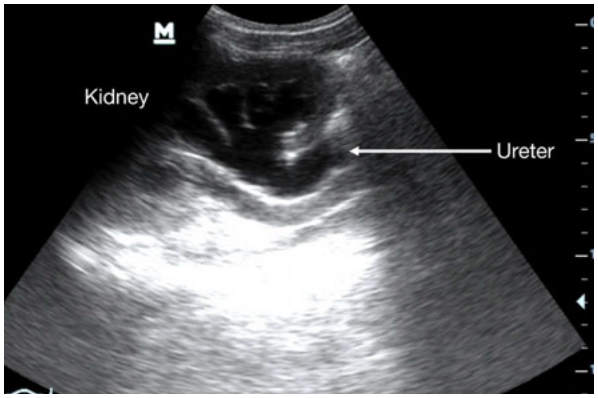


Figure 9.6. Moderate hydronephrosis II°: Calyces dilated, normal thickness of the parenchyma.

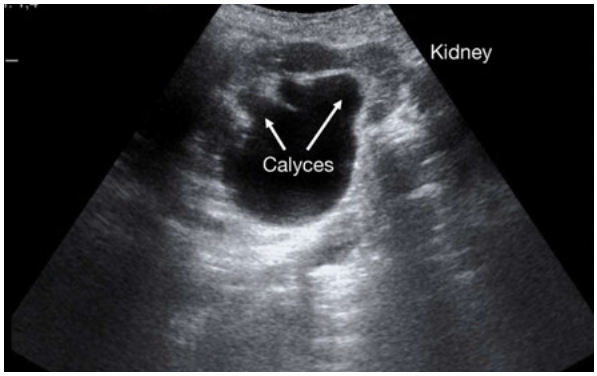


Figure 9.7. Severe hydronephrosis III°: Calyces concave and bulged outwards, with thinning of the parenchyma.

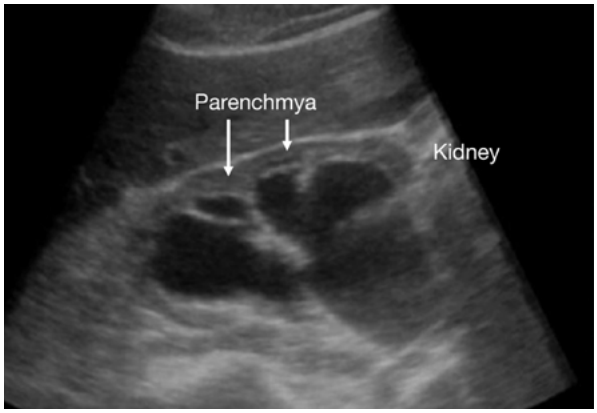


Figure 9.8. Terminal hydronephrosis IV°: Thin parenchyma around a septated fluid sac.

Table 9.4. Classification of hydronephrosis

Degree of Hydronephrosis	Ultrasound Criteria
Mild (I°) (Figure 9.5)	• Slight separation in the echogenic central region, visible as a discrete central lucency. • Normal thickness of parenchyma (cortex).
Moderate (II°) (Figure 9.6)	• Clear separation of the echogenic central region by anechoic area. • Calyces are convex. • Normal thickness of parenchyma.
Severe (III°) (Figure 9.7)	• Large anechoic area. • Calyces are concave (bulged outward). • Thinning of the parenchyma (esp. in chronic obstruction).
Terminal (IV°) (Figure 9.8)	• Only anechoic, fluid-filled structure. • Parenchyma thin or no longer present.

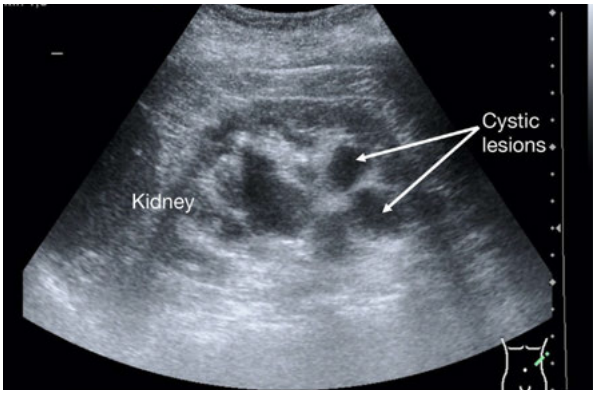


Figure 9.9. Multiple cystic lesions in the central kidney. In contrast to hydronephrosis, each cyst is separated by its own walls.

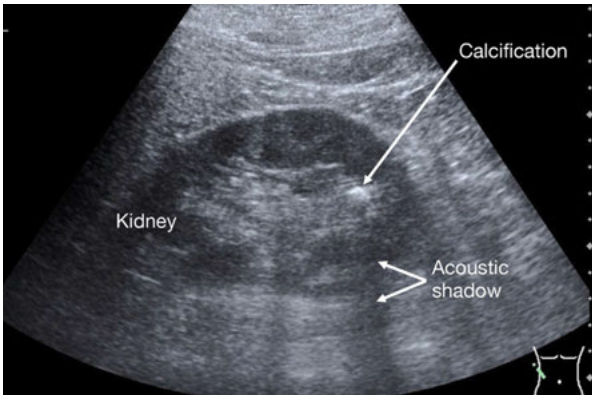


Figure 9.10. Kidney with echogenic calcification in the lower pole. The calcification throws an acoustic shadow.

In kidneys with multiple central cysts, care should be taken **not to mistake the cysts** (which have individual walls) (Figure 9.9) for a hydronephrosis (which is confluent).

Renal calcifications and kidney stones

Calcifications and stones appear as **strongly hyperechoic structures** with a posterior **acoustic shadow** (Figure 9.10). Stones at the renal-ureteric junction will often show a dilated renal pelvis with a collapsed proximal ureter. Stones within the ureter are rarely seen, but the resulting hydronephrosis can be easily detected. Stones at the **ureterovesical junction** can be seen as echogenic structures **behind the bladder** wall at the orifice of the ureters.

Small or shrunken kidneys

With chronic, longstanding renal diseases, the kidneys become **smaller,** usually shrinking by **similar amounts on both sides** (Figure 9.11). As normal kidney size is 10 ± 2 cm, **kidneys smaller than 8 cm** should suggest the possibility of chronic kidney disease, especially when the shrinkage is bilateral.

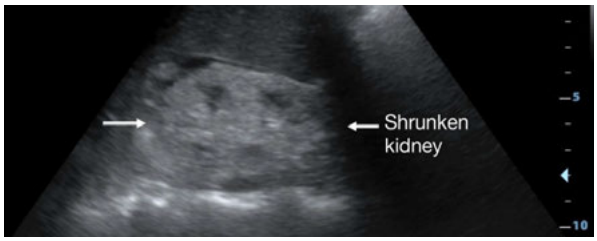


Figure 9.11. Echogenic small kidney, consistent with chronic renal disease.

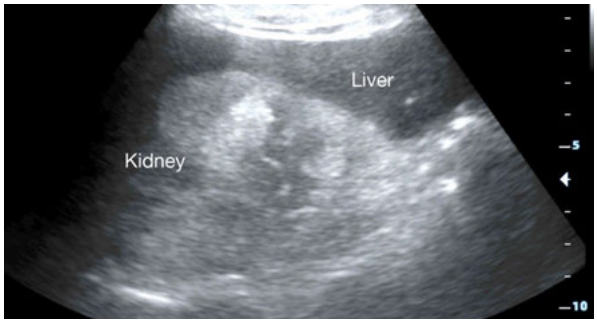


Figure 9.12. Normal-sized, echogenic kidney in HIV-associated nephropathy (HIVAN).

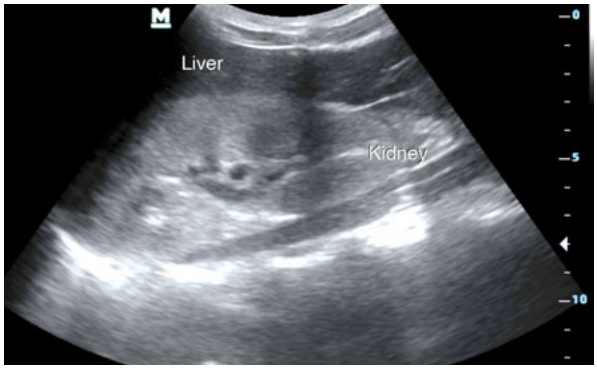


Figure 9.13. Another example of HIVAN.

HIVAN

In ultrasound examinations of patients with HIV, **normal-sized** kidneys with **hyperechoic cortex** are often seen (Figures 9.12 and 9.13). The pyramids may seem slightly hypoechoic in comparison. These changes may be found even when the results of renal function tests are normal. Focal segmental glomerulosclerosis is the most frequent underlying pathology of HIVAN. The morphology is **non-specific;** it can also be found in other renal diseases, such as **diabetic glomerulosclerosis** and other forms of chronic glomerulonephritis.

Normal bladder

The bladder is assessed in transverse and longitudinal views. Transverse sweeps through the bladder are performed to assess its shape and wall thickness, as well as the distal ureters. To obtain the transverse view, the probe is placed **horizontally above the pubic symphysis.** For the longitudinal view, the probe is turned to the midline. A normal, fully distended bladder has a regular, rectangular shape with a **wall no thicker than 5 mm.** Normal distal ureters are not visible (Figure 9.14).

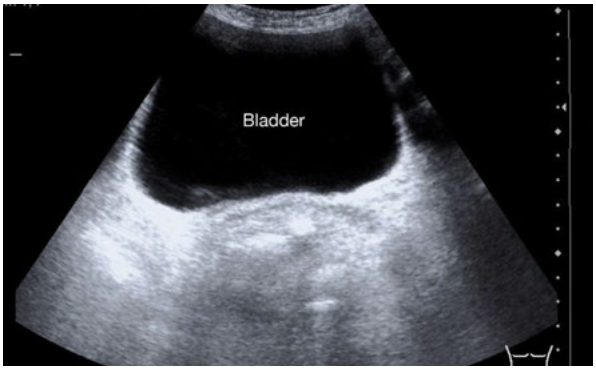


Figure 9.14. Bladder with thin regular wall, filled with anechoic urine.

Bladder volume can be measured by using the transverse and longitudinal images to obtain height, width, and depth. Once the three dimensions are measured, the formula *(H x W x D) x 0.7* is used to calculate volume. If the bladder is not fully distended, its wall cannot be reliably assessed.

Bladder distention

Bladder distention is the most common pathology of the bladder. It is characterised by an enlarged bladder, which may extend into the **mid-abdomen.** It can be secondary to bladder outlet obstruction (for example, by the prostate) or neurological disease.

Schistosomiasis

Schistosomiasis-related urinary pathologies include irregular bladder shape, **bladder wall thickening** with **diffuse or focal thickening of > 5 mm, bladder wall calcifications,** and **pseudopolyps** (Figure 9.15) or **masses** protruding into the bladder lumen. These masses can progress into **bladder cancer** (Figure 9.16) and spread beyond the wall.

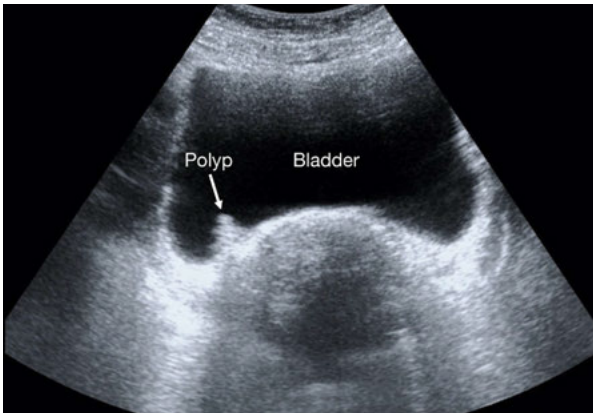


Figure 9.15. Focal bladder wall thickened (polyp) due to schistosomiasis.

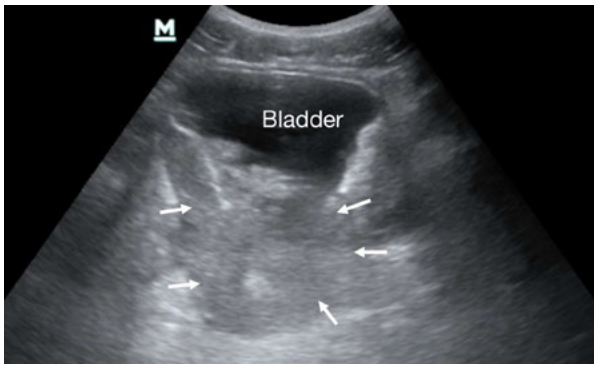


Figure 9.16. Bladder cancer (arrows) in a 24-year-old, secondary to schistosomiasis.

Diagnostic and therapeutic implications

Further diagnostic and therapeutic steps depend on the findings:

Renal cysts

- Cysts are benign findings; they do not usually cause clinical problems, and require no further investigation.
- Polycystic kidney disease, which can cause clinical problems, is a differential diagnosis characterised by hundreds or thousands of cysts (Figure 9.17). A genetic disease accompanied by reduced renal function, it is always bilateral. The unusually large number of cysts combined with the absence of normal parenchyma usually make it easy to distinguish.

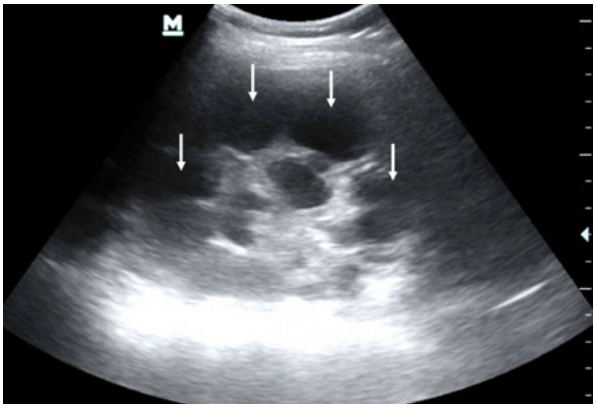


Figure 9.17. Polycystic kidney disease, charcterised by uncountable cysts and little normal cortex.

Urinary tract obstruction

- When hydronephrosis is identified, try to follow the course of the ureter, as lymph node masses in this area may be the cause of dilatation. Also consider non-HIV/TB-associated aetiologies. These include distended urinary bladder, kidney stones or stones at the ureterovesical junction in the bladder wall, bladder cancer or other bladder wall irregularities, and prostate hypertrophy (normal prostate size: < 2.5 x 4.5 cm).
- Investigations directed at the underlying downstream aetiology (such as the bladder and prostate) will guide therapeutic management. A distended bladder is easily identified and can be treated by inserting a Foley catheter. Management of stone disease often relies on analgesia and hydration.
- Though sonographic features suggestive of schistosomiasis are not pathognomonic, in high endemic areas they should prompt antihelminthic treatment to prevent further deterioration. Treatment may also lead to improvement of sonographic findings.
- If acute kidney injury with impaired renal function is clinically present, these sonographic findings suggest **post-renal AKI.**
- Evaluation by a urologist may be helpful in case urine drainage is required.

Small or shrunken kidneys

- Chronic kidney disease is likely; check for underlying disease. The most common aetiologies are chronic hypertension or chronic diabetes, which can be identified by checking blood pressure, left ventricular wall thickness, and both random and fasting blood sugars.
- Consultation with a nephrologist may be helpful in case dialysis is required.

HIVAN

- The sonographic finding should prompt an assessment for proteinuria and serum creatinine. HIVAN may lead to renal impairment with proteinuria. It is associated with a CD4 of less than 200 cells/mL. The serum albumin may be reduced.
- Though rarely performed in resource-poor settings, biopsy is required to confirm the diagnosis.
- Other than ART, there is no specific treatment indicated. In cases with proteinuria and echogenic kidneys, ACE inhibitors should be added, and co-existing hypertension well controlled.

Pearls and pitfalls

- Not all hydronephrosis is due to obstruction. Reflux is a common cause, especially in children.
- Back pressure from a distended bladder may lead to physiological dilation of the renal collecting system. If mild hydronephrosis is identified in the presence of a full bladder, a repeat scan 20 minutes after voiding is required to confirm persistent hydronephrosis.
- Dilation, especially on the right side, is commonly seen in pregnancy; it may or may not be symptomatic.
- Stones in the ureters are often overlooked as a cause of obstruction.
- The significance of the sonographic changes of HIVAN is unclear, especially in patients with normal urea and creatinine in serum.

Investigating renal problems in HIV patients in resource-limited settings: a three-layer algorithm

Layer 1

Possible Diagnosis	Presentation/Cause	Ultrasound Findings	Treatment/ Course of Action
Does it look like chronic kidney disease (CKD)?	Commonly due to poorly controlled diabetes and/or hypertension	Small or shrunken, often echogenic kidney	• Improve management of the chronic condition. • Stop smoking. • Avoid NSAIDs. • Adjust drug doses as needed.
Could it be HIVAN?	Proteinuria 2+ on dipstick, with no haematuria, normal BP, no oedema, and no rash	Normal sized, echogenic kidney	• Continue ARVs. • Consider TDF-sparing regimen. • Start enalapril.
If none of the above: Could this be acute kidney injury (AKI)?			**Go to Layer 2.**

Layer 2

Possible Diagnosis	Presentation/Cause	Ultrasound Findings	Treatment/ Course of Action
Could this be pre-renal AKI?	Usually associated with hypovolaemia and low BP (due to dehydration, diarrhoea, sepsis)	• IVC collapsing • Tachycardia • Hyperkinetic heart	• Immediately administer IV fluids. • Treat the underlying condition.
Could it be post-renal AKI?	Enlarged lymph nodes due to: • TB • Malignancy • Schistosomiasis	• Hydronephrosis? • Enlarged bladder?	Treatment will depend on underlying cause; a urologist may be needed.
If none of the above: Could this be infrarenal AKI?			**Go to Layer 3.**

Layer 3

Possible Diagnosis	Presentation/Cause	Ultrasound Findings	Treatment/ Course of Action
Could this be tubular necrosis (85%)?	TDF toxicity involves direct damage to the tubules (Fanconi syndrome); it can occur in weeks to months, presenting as elevated creatinine, glycosuria, or even oedema.	No specific signs—possibly enlarged or swollen kidney	• Stop nephrotoxic drugs. • Immediately administer IV fluids. • Treat the underlying condition.
Could it be acute interstitial nephritis (5%)?	• May occur with rash, fever, joing pain, eosinophilia, eosonophiluria. • Can look like pyelonephritis with fever and flank pain. • Recurs on re-exposure (Bactrim, rifampicine).	No specific signs—possibly enlarged or swollen kidney	• Stop the offending drug. • Administer steroids (in some cases).
Could this be acute glomerulonephritis (5%)?	• Usually presents with haematura, proteinuria, hypertension, and oedema. • Requires hospitalisation.	No specific signs—possibly enlarged or swollen kidney	**Refer patient to nephrologist.**

Genitourinary TB

Genitourinary TB (GUTB) is seen **more frequently in male patients.** One-third of patients with GUTB report a previous history of tuberculosis.

The pathogenesis involves the haematogenous spread of *M. tuberculosis* and seeding of the renal cortex; this tissue is exposed to high oxygen tension and is therefore susceptible to seeding. **Granulomas** may form; when the immune system deteriorates, these can spread into the renal medulla, causing **papillary necrosis** and **renal obstruction.** Excretion of *M. tuberculosis* in the urine can lead to **ureter and bladder involvement,** followed by **fibrosis** and reflux. The most common symptoms are frequent urination, dysuria, lumbar pain, and haematuria; only one in five patients complains of systemic symptoms like fever and malaise. Recurrent findings of pyuria with sterile urinary bacterial culture may prompt suspicion of GUTB.

Sonography may show **renal calcifications,** thickening (Figure 9.18), **papillary irregularities,** and intrarenal masses. The most frequent, and most easily recognisable, finding is urinary tract obstruction (**hydronephrosis**). The diagnosis is usually made with a urine culture; urine specimens can also be assessed through **GeneXpert MTB/RIF testing,** as recent studies have shown good sensitivity and specificity. A urine **LAM** test can also be used.

Genital TB in males (Figure 9.18) may present with a wide range of symptoms, from **painless scrotal swelling** without additional symptoms to gram-negative sepsis due to **obstruction and secondary superinfection.** Epididymo-orchitis is a relatively common manifestation. **Focal areas of decreased echogenicity** (Figure 9.20) can be demonstrated in **the testis or epididymis, with or without calcifications.** Differentiation from tumours and bacterial abscesses may be difficult, so image-guided aspiration (GeneXpert) is often needed. (For female genital TB, refer to the box at the end of Chapter 8.)

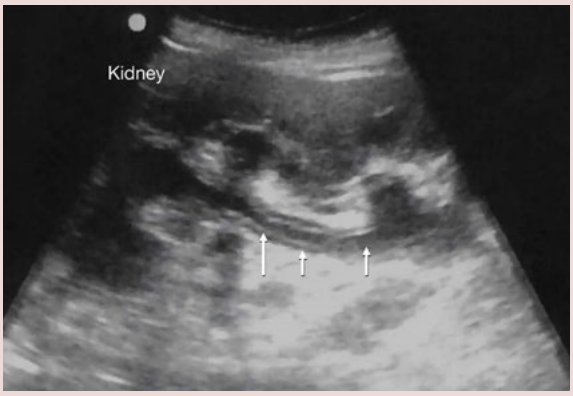


Figure 9.18. Urinary tract TB with thickening of the collecting system and ureter (arrows).

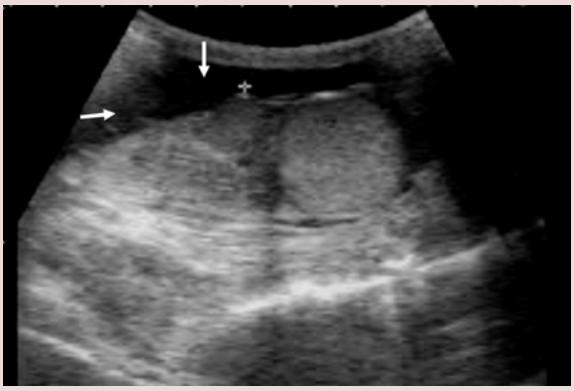


Figure 9.19. Fluid collection (arrows) with minimal separations in the scrotum due to genital TB.

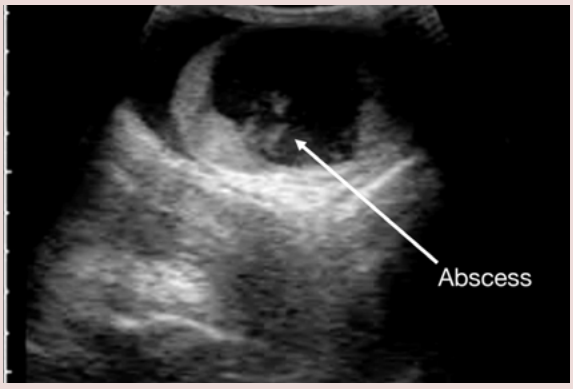
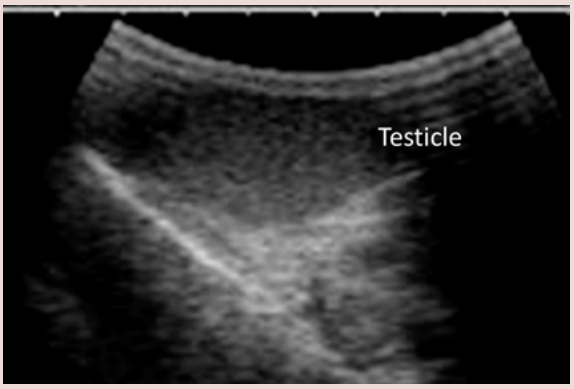


Figure 9.20. (L) Hypoechoic abscess in the testicle. The aspirate was positive for TB.



(R) Normal testicle for comparison.

10. Ultrasound of Superficial Structures: Skin Abscesses and Peripheral Lymph Nodes

Introduction

The **skin, subcutaneous tissues, lymph nodes,** and many parts of the musculoskeletal system are relatively **superficial structures.** They are thus ideal targets for ultrasound examination.

Patients with **soft tissue infection** may have fever, chills, and leucocytosis in addition to **redness, local heat,** and **swelling** at the infected sites.

Cellulitis is the most common type of soft tissue infection; it is confined to the subcutaneous compartment and **spreads diffusely through the tissue.** An **abscess** is a **collection of pus within tissue;** it develops either as the primary site of infection, or as a complication of cellulitis (Figure 10.1). A painful red mass is usually present;

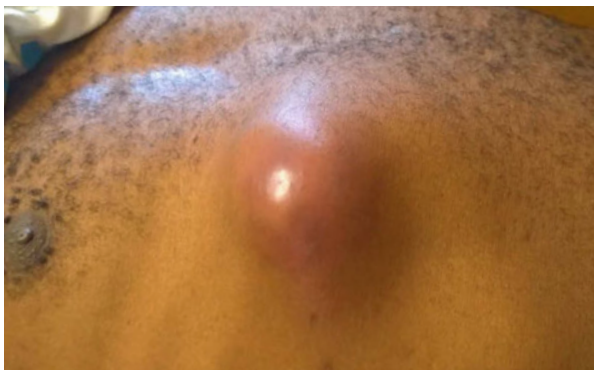


Figure 10.1. Hot abscess: subcutaneous abscess with swelling, redness, heat, and pain.

it may be tender, fluctuating, and often warm. Differentiating between an abscess and cellulitis is important, as the two conditions are managed differently; **abscesses are treated using incision and drainage,** whereas **cellulitis is treated with antibiotics.** In the absence of other imaging modalities, bedside ultrasound is a good way to differentiate between these two conditions.

Enlarged lymph nodes are a frequent presentation of **patients with TB** (Figure 10.2), but they have wide differential diagnoses, especially in HIV patients in a tropical setting. The differential diagnoses include TB, MAC, toxoplasmosis, and cytomegalovirus (CMV), as well as generalised KS and lymphoma.

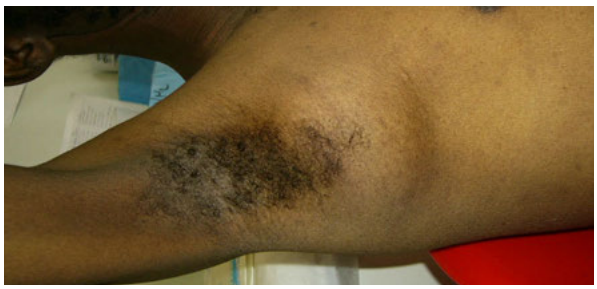


Figure 10.2. Cold abscess: axillary swelling with little pain, and absent heat and redness.

Technique and ultrasound findings

For sonographic assessment of superficial structures, a **high-frequency linear transducer** is used; assessment of deeper structures requires a lower-frequency curvilinear transducer. The location of interest should always be scanned on two planes and **compared to the healthy side** whenever possible.

The **epidermis** and dermis appear as **thin, hyperechoic layers.** The **subcutaneous fat** layer usually appears **hypoechoic** and nodular, with hyperechoic linear septa. **Striated muscles** are **hypoechoic with hyperechoic striation,** best seen in the long axis of the muscles (Figure 10.3).

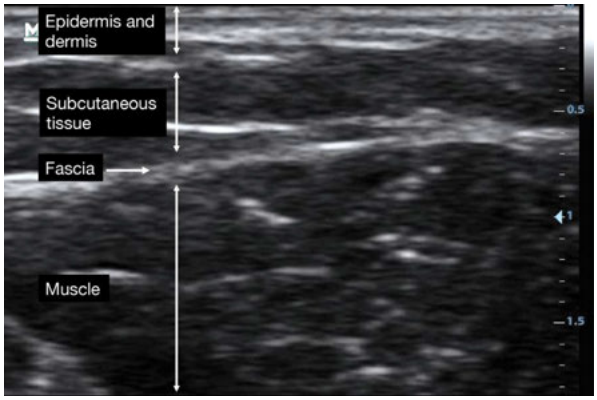


Figure 10.3. Normal skin: Epidermis and dermis (echogenic), subcutaneous tissue (hypoechoic), muscular fascia (echogenic), and muscle (hypoechoic).

With **cellulitis,** the dermis appears thick and bright, has blurred tissue margins, and may display a characteristic **cobblestone pattern** (Figure 10.4). This pattern results from hyperechoic, inflamed subcutaneous fat being intersected by hypo- to anechoic fluid tracking along the connective tissue.

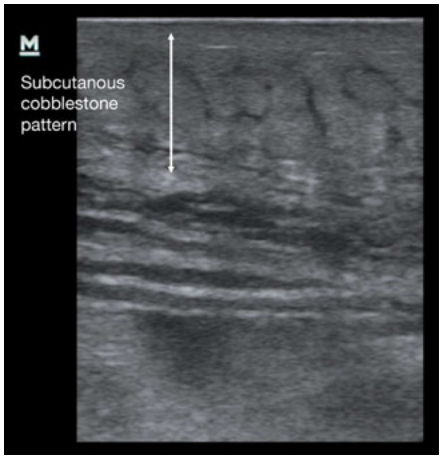


Figure 10.4. Cobblestone pattern in cellulitis (and oedema). The subcutaneous tissue shows hyperechoic fat lobules septated by hypoechoic fluid-filled areas.

Abscesses are usually **anechoic or hypoechoic focal areas** with posterior acoustic enhancement (Figure 10.5 on the following page). They may also contain **mixed internal echogenicity,** including hyperechoic foci with posterior shadowing, representing gas inclusions. The margins have an **irregular shape;** with complex abscesses, different layers may be distinguishable. The **'squish sign'** is a **movement of echogenic particles** in response to compression; this movement can be used to differentiate a liquefied abscess from a soft tissue mass.

Peripheral lymphadenopathy **most frequently** involves the **lymph nodes of the neck,** but it can also affect the axillary and inguinal nodes. On grayscale sonography, TB nodes tend to be hypoechoic, round,

and without the echogenic hilum (that is, missing the 'hilum fat sign') often seen in reactive lymph nodes (Figure 10.6). They may show **intranodal cystic necrosis** (caseous necrosis) (Figure 10.7). **Nodal matting** with adjacent echogenic soft tissue oedema is often seen. Reactive lymph nodes typically show a bright hilum, discrete borders, and no posterior acoustic enhancement on ultrasound.

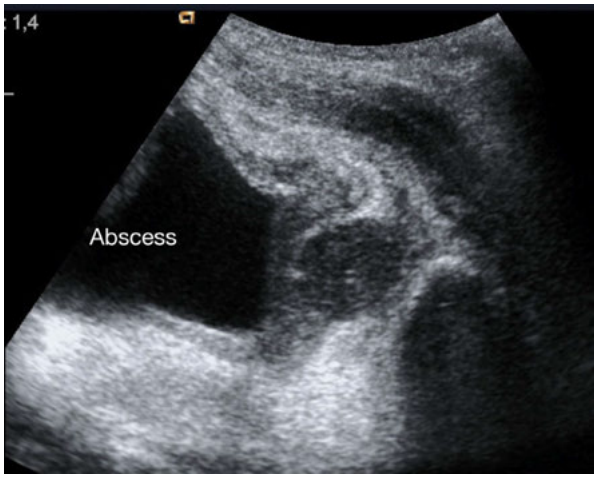


Figure 10.5. Anechoic collection in the skin, with capsule surrounding the lesion.

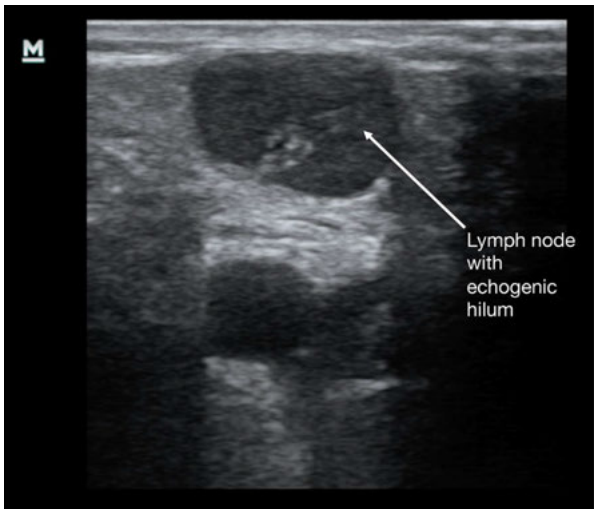


Figure 10.6. Subcutaneous normal lymph node with central echogenic hilum.

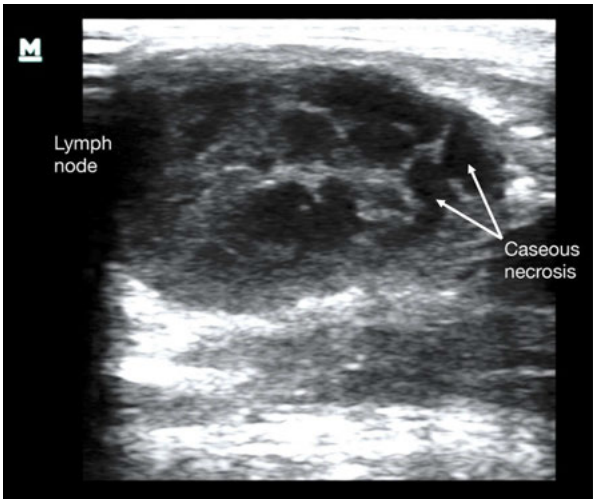


Figure 10.7. Subcutaneous tuberculous lymph node with almost anechoic areas due to caseous necrosis.

Diagnostic and therapeutic implications

Differential diagnoses of cellulitis should include **DVT** and **oedema,** which can also produce a cobblestone pattern. Differential diagnoses of abscesses in the groin should include hernias and enlarged lymph nodes. **Herniated bowel loops** may demonstrate peristalsis on ultrasound. **Necrotic** malignant or tuberculous nodes, however, are very similar in appearance to abscesses.

Uncomplicated cellulitis is treated with antibiotics. Uncomplicated and accessible abscesses are usually managed by blind incision and drainage. POCUS can be used to diagnose occult (hidden) abscesses, as well as to help determine the safest route for incision and drainage. For enlarged lymph nodes, **ultrasound-guided biopsy** is a practical way to reach a final diagnosis. Enlarged lymph nodes can be **aspirated with a fine needle;** the aspirate can be flushed into a small amount of normal saline, and the sample **submitted for GeneXpert MTB/RIF testing.** The test is easy to perform, widely available, specific, and more sensitive than smear microscopy for TB. If GeneXpert results come back negative, ultrasound-guided **core-needle biopsy,** which is frequently diagnostic (when **pathology services** are available), is a possible next step.

Pearls and pitfalls

- When in doubt, compare the area of interest to the contralateral normal area.

Parotid changes in patients with HIV

Approximately 5–10% of patients with HIV have some degree of **parotid swelling** (Figure 10.8). It is more common in cases of advanced disease, and is most often caused by **cystic lymphoepithelial lesions** of the salivary gland associated with **diffuse infiltrative lymphocytosis syndrome (DILS).** This is a Sjögren's-like syndrome with painless salivary gland swelling, peripheral CD8 lymphocytosis, and sicca symptoms of dry mouth and insufficient saliva production. DILS is more common amongst patients of African descent than in Caucasians.

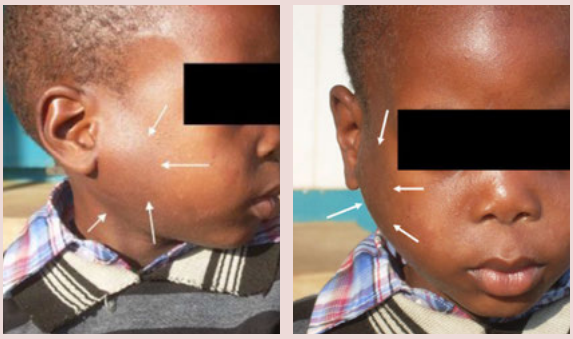


Figure 10.8. Bilateral swelling of the parotid glands, observed as a swelling of the cheeks (arrows).

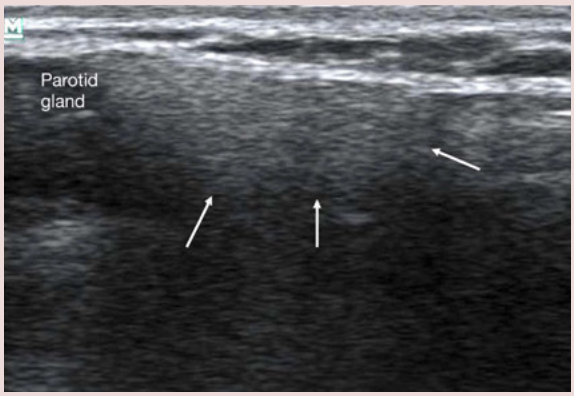


Figure 10.9. Normal parotid with homogenous tissue (arrows).

On sonographic examination, **normal salivary glands appear smooth and velvet-like,** similar to thyroid tissue (Figure 10.9). With DILS, the ultrasound shows unilaterally or bilaterally enlarged glands with **multiple cystic lesions.** The size of the cystic areas

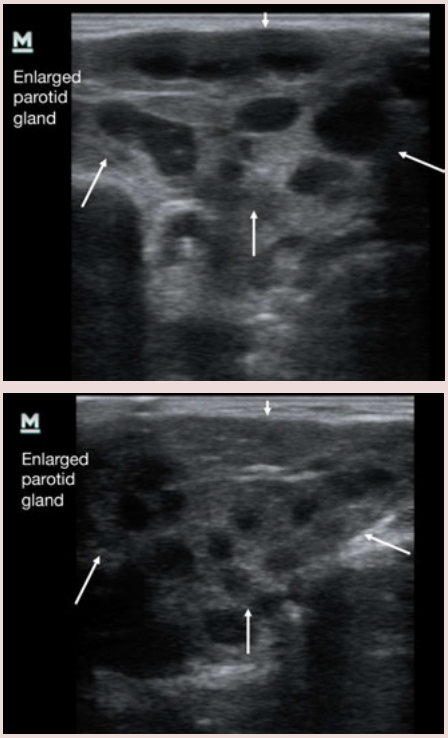


Figure 10.10. Examples of (Top) enlarged parotid with multiple hypoechoic areas (arrows), (Bottom) some almost cystic (arrows).

can vary from a few millimetres to a few centimetres; similarly, the number of cysts can vary considerably, sometimes creating a Swiss cheese-like appearance (Figure 10.10).

If necessary, the diagnosis can be confirmed through fine-needle aspiration. In cytological investigations, lymphocytes and epithelial cells are seen. When the results are unclear, histology may be required.

Treatment modalities **include ART,** which should be initiated (or continued). Additionally, **prednisolone,** in a dose of approximately 0.5 mg/kg tapered over a period of four to six weeks, is usually helpful. For **larger cysts,** simple **aspiration** and even surgical resection can be performed (although surgery is rarely indicated). Regression of swelling has been demonstrated in patients treated with ART, so this should be the mainstay of treatment.

11. Ultrasound of the Liver

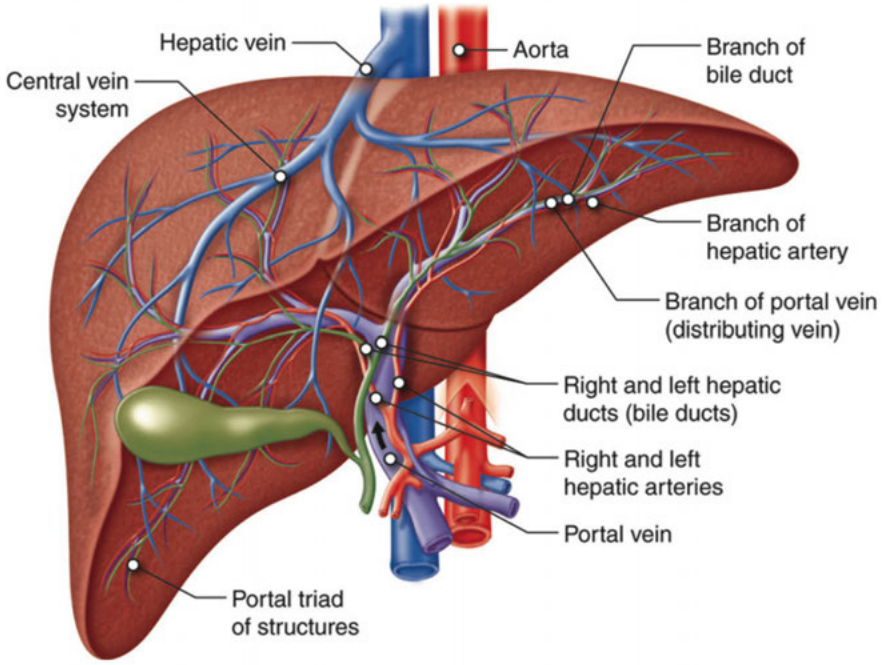


Figure 11.1. Gross anatomy of the liver. (Illustration by Cenveo. Source: https://www.openassembly.com/document/47c7a9dc-776c-4d15-b56c-8ca1bfa86b16. Used under a Creative Commons Attribution 3.0 license. http://creativecommons.org/licenses/by/3.0/us/.)

Anatomy and normal ultrasound findings

Ultrasound examination of the liver is a demanding procedure, due to the complex anatomy and large size of the organ. The liver is best examined with the patient in a supine position. Having the patient take and hold a **deep breath** helps to displace the diaphragm and liver downwards, making them more easily visible. For patients who are too short of breath to hold their breath for prolonged periods, a good alternative is to ask them to **press out a round belly,** which will also **displace the diaphragm and liver downwards.**

The **size of the liver is difficult to measure;** significant variation is seen, depending on its shape and on where and how the measurement is done. For this reason, many examiners offer general descriptions (normal/large/very large) rather than exact measurements.

When measurements are performed, a longitudinal scan at the mid-clavicular line should be used. Normal size is around 12–14 cm. The **surface of the liver** should be flat. The **caudal border** should be **acute-angled** and sharp-edged, with the angle more acute at the left side of the organ (30–45° for the left lobe, vs 45–75° for the right lobe). The **parenchyma** is **moderately echogenic,** with fine, **homogenous echoes;** when compared to the adjacent normal kidney, it should be isoechoic or slightly more echogenic than the renal parenchyma.

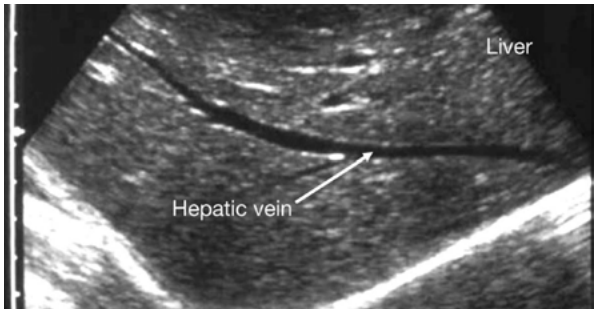


Figure 11.2. Hepatic vein in the liver: oblong, acute-angled branches with minimal connective tissue.

Vessels of the liver

It is possible to identify three types of tubular structures in the liver: the hepatic venous system, the portal venous system, and the biliary tree (bile ducts).

Hepatic veins have a rectilinear, oblong course and imperceptible margins (Figure 11.2). In this respect, they resemble a poplar tree (Figure 11.3). The hepatic veins drain into the IVC, forming a **star-like shape** when viewed from the epigastric angle (Figure 11.4). In the central parts they may fluctuate in size with the respiratory cycle. The hepatic veins mark the borders of the medial and lateral parts of the left liver lobe, both between the left and right lobes and between the anterior and posterior parts of the right lobe.

The **portal vein** has many more branches. Only short stretches are visible in each ultrasound section; the wall

Figure 11.3. Hepatic vein branching resembles poplar trees in winter.

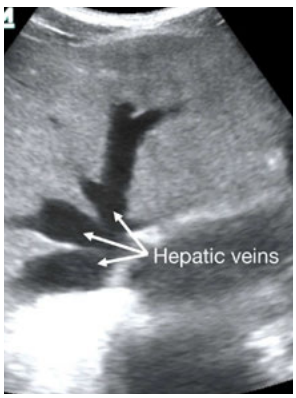


Figure 11.4. Confluence of the three hepatic veins close to the IVC leaving the liver cranially—appearing as a star-like shape in the image.

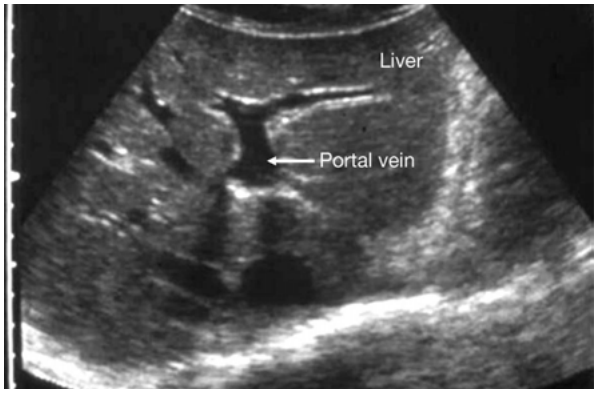


Figure 11.5. Portal vein in the liver: short segments, obtuse-angled branches with significant echogenic connective tissue.

Figure 11.6. Portal vein branching resembles a twisted willow tree in winter.

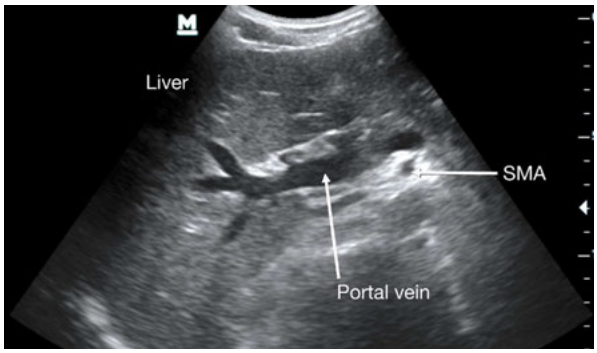


Figure 11.7. Portal vein entering the liver caudally. It is fed by the splenic vein. (SMA= superior mesenteric artery.)

is visible as an echogenic margin (an **'embankment of vessels'**) (Figure 11.5). This resembles the branching of a willow tree (Figure 11.6). The larger parts of the vessel are directed caudally towards the **porta hepatis** (Figure 11.7).

Intrahepatic bile ducts are normally not visible. Only the **common bile duct** can be seen, as an anechoic tubular structure ventral to the portal vein. (Note: It can be mistaken for the hepatic artery).

TB of the liver

Involvement of the liver in TB is common (up to 80% in autopsies of PTB). On histological analysis, multiple hepatic granulomata are often present. Clinical manifestations of this involvement are less frequently seen. Elevations of **alkaline phosphatase (ALP)** and **gamma-glutamyl transferase (GGT)** are the most frequent laboratory findings, though these are non-specific. Hypo- and hyperglobulinemia are common, although these are more indicative of chronic tuberculous disease than of hepatic synthesis dysfunction.

Two forms of liver involvement can be sonographically distinguished. In some patients, a **diffuse homogenous hepatomegaly** is seen, often with a **bright echo pattern** (Figure 11.8). This can be misdiagnosed as fatty infiltration (see p. 46), but when biopsied shows **hepatic granulomatous disease.** These findings are also described as granulomatous hepatitis, but this is a misnomer—hepatic granulomas generally do not affect liver cells.

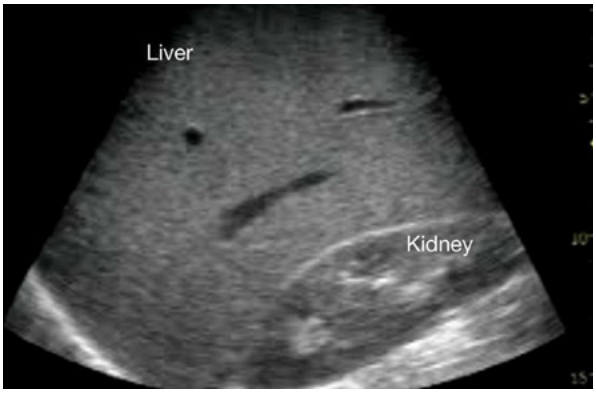


Figure 11.8. Granulomatous hepatitis due to TB: the liver is enlarged and more echogenic compared to the kidney.

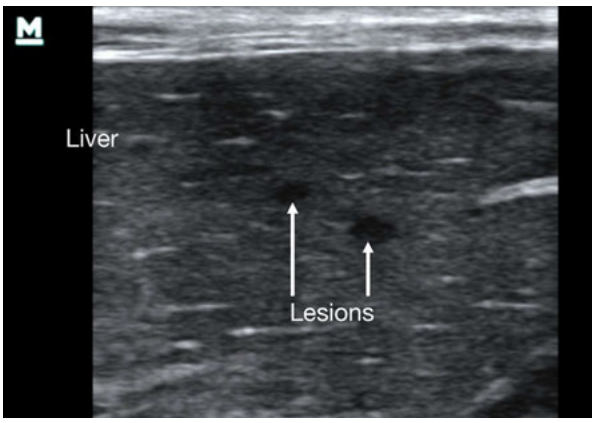


Figure 11.9. Tuberculoma in the liver: hypoechoic small lesions, which often look similar to the microabscesses in the spleen.

In other patients, **focal tuberculomas** may be visible (Figure 11.9), appearing as one or more, usually **hypoechoic, abscess-like** lesions that **vary in size** (0.5–12 cm). It should be remembered that these lesions (as with all tuberculous lesions) may initially increase in size during otherwise successful treatment, as the body's improved immunological response increases the inflammatory reaction.

Focal liver lesions in the tropical setting

Focal hepatic lesions are a difficult topic. Some may be easy to find, depending on their echogenicity and location, but then can be difficult to characterise. Others may be missed on ultrasound because their echogenicity is similar to that of the surrounding liver, meaning that changes in the echo pattern can be very subtle. At the same time, the location of the lesion has a huge impact—a central lesion may be seen right away, while a lesion under the diaphragm may easily go unnoticed.

In this text, we will present a few examples of the most frequent and important findings. (A brief list of appears in Table 11.10 on the next page. For more detailed descriptions, ultrasound textbooks should be consulted.)

Cysts of the liver (Figure 11.11) are anechoic structures characterised by dorsal acoustic enhancement and fine walls. Most are congenital, found incidentally, and typically have little or no clinical significance. They are mainly significant in that their presence may cause confusion with the other potential causes of hypoechoic lesions listed below.

Echinococcal cysts (Figure 11.12) are often located in the liver (50% of this type of cyst is found there).

Table 11.10. Differential diagnosis of focal liver lesions

Malformations	• Congenital cysts
Infections	• Abscesses • Echinococcal cysts
Sequalae of trauma	• Haematoma
Neoplasms	• Benign tumours (e.g., haemangioma) • Malignant tumours (e.g., HCC, CCC, metastases, lymphoma)
Others	• Patchy distribution of fat • Calcifications • Nodules of regeneration in cirrhosis

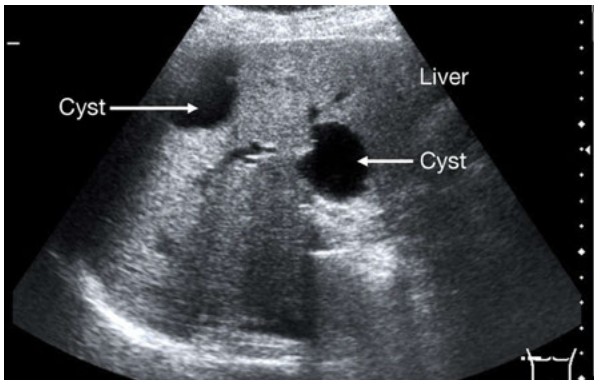


Figure 11.11. Liver cyst: Two well-defined, round anechoic lesions resembling holes punched out of the liver.

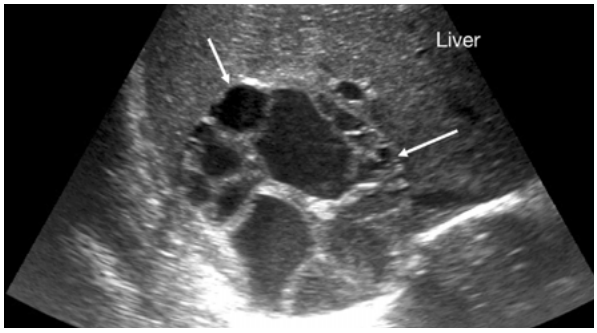


Figure 11.12. Echinococcal cyst in the liver: Well-defined, round mother cyst (arrows) with multiple daughter cysts within. Also described as a honeycomb-shaped cyst.

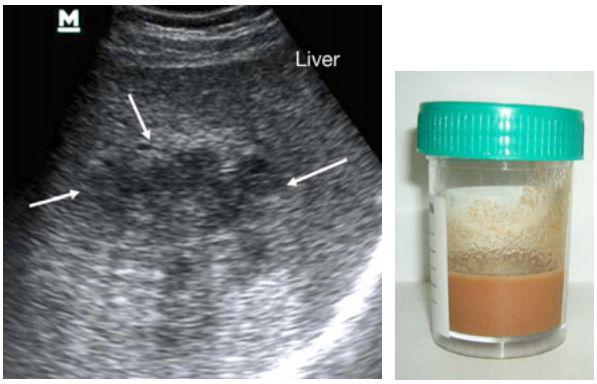


Figure 11.13. (L) Amoebic liver abscess: Ill-defined, roundish but irregular hypoechoic lesion (arrows) in the right lobe of the liver. (R) Material aspirated from a liver abscess. The anchovy sauce-like appearance suggests amoebic abscess.

Compared to congenital cysts, they have thicker walls—often with two layers visible, and sometimes calcified. They may show detached inner cysts or multiple daughter cysts within the mother cysts (honeycomb cysts).

Amoebic abscesses (Figure 11.13) are frequent findings in tropical countries. Around 8% of patients with

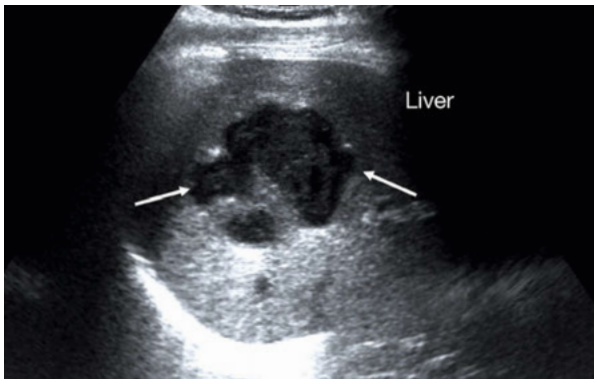


Figure 11.14. Pyogenic liver abscess: Hypoechoic, often irregular lesion (arrows) in the liver.

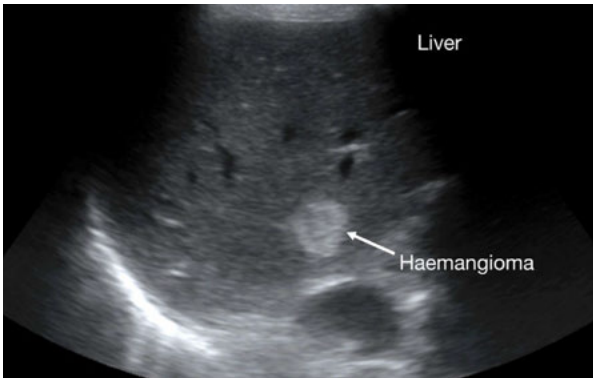


Figure 11.15. Typical liver haemangioma: Well-defined echogenic lesion, usually small.

amoebiasis develop hepatic abscesses. They are more common in adults than in children, and in males than in females. In animals, substantial necrosis is induced five to seven days after the amoebae reach the liver. This explains the abrupt onset often seen in previously healthy individuals.

Often, a hypoechoic lesion lacking significant wall echoes is seen. In more than 60% of patients, this is a single lesion, usually located in the dorsal right lobe. No gas bubbles are seen within the lesion, as it results from liquefaction necrosis of hepatocytes. When aspirated, the typical anchovy sauce-like fluid is produced. The ultrasound appearance of the lesion changes with proper therapy, first becoming more hypoechoic, and then starting to shrink. Sometimes partly calcified residual lesions are found (even after a long time); these do not require further treatment.

Hepatic pyogenic abscesses (Figure 11.14) vary greatly in shape and size. They usually have irregular walls, and the pus they contain may range from anechoic to highly echogenic. In most cases, this type of abscess is less echogenic than liver tissue. Marked echogenicity is sometimes seen due to gas bubbles within the abscess. Mainly at risk are patients who have other infections (biliary tree, diverticulitis), or who have experienced abdominal trauma, surgery, or intervention of the biliary system. However, immunocompromised patients are also at risk.

Haemangiomas (Figure 11.15) are the most common benign tumour found in the liver. In more than 10% of cases, multiple haemangiomas are visible. These are very hyperechoic and well-defined; sometimes even a feeding vessel may be visible. Typical haemangiomas can be diagnosed with ultrasound alone. However, atypical haemangiomas, which are larger (often > 3 cm) and have an inhomogeneous structure with hypoechoic areas, may need further workup, as the differential di-

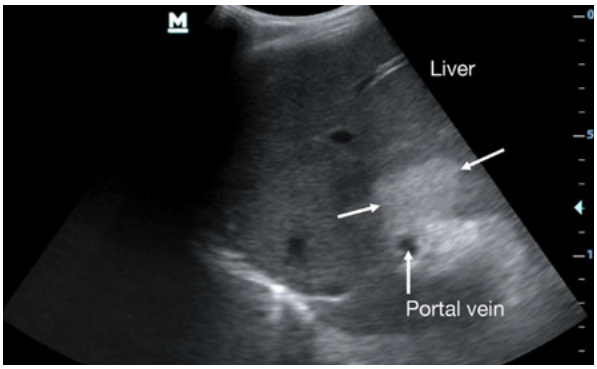


Figure 11.16. Focal patch of intrahepatic fat: Well-defined, echogenic area (arrows) with a geographic appearance. Focal fatty infiltration is often localised close to the portal vein and the gall bladder.

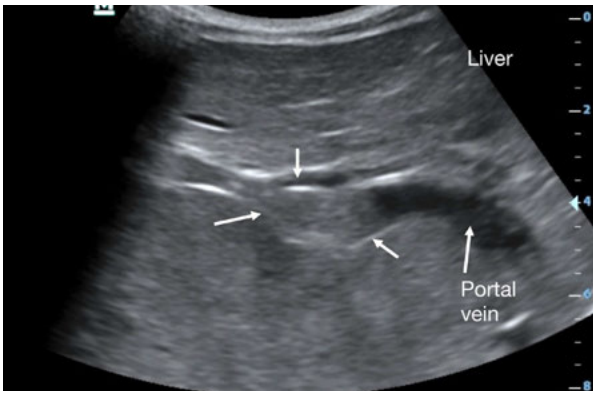


Figure 11.18. HCC tends to infiltrate the portal vein and cause thrombosis or intravascular growth (arrows).

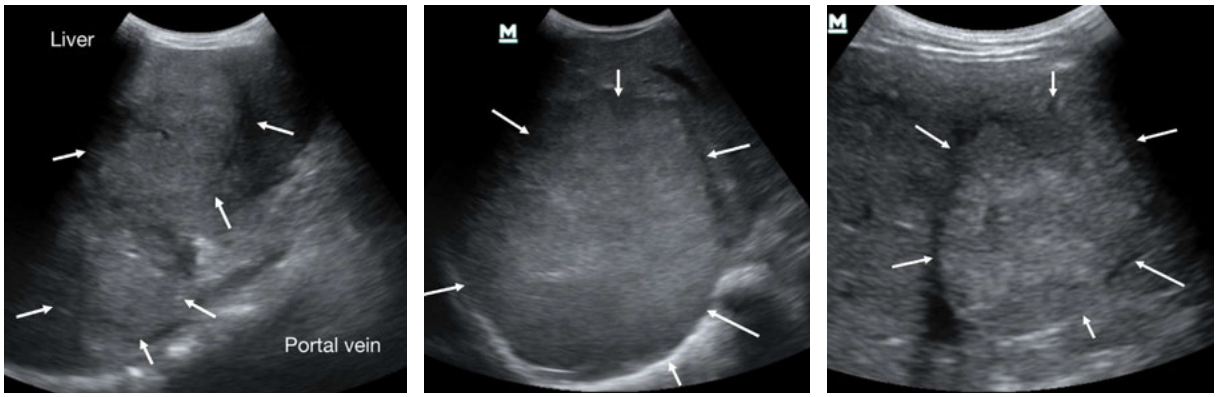


Figure 11.17. Three examples of hepatocellular carcinoma (HCC), appearing as an irregular, inhomogeneous, slightly hyperechoic area (arrows). Often associated with hepatitis B and found in cirrhotic livers, but can also show up in normal livers. HCC is often diagnosed late, by which time lesions have already grown large.

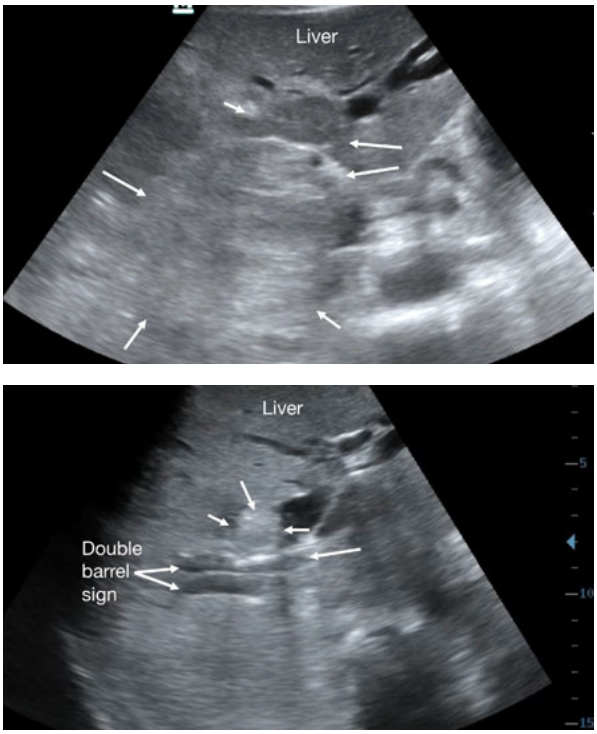


Figure 11.19. Cholangiocarcinoma (CCC): (Top) Irregular, often large tumours (arrows) causing biliary dilatation. (Bottom) Tumour growth (arrows) can sometimes be seen in the bile ducts, along with the double barrel sign (the two parallel tubular structures suggest biliary dilatation).

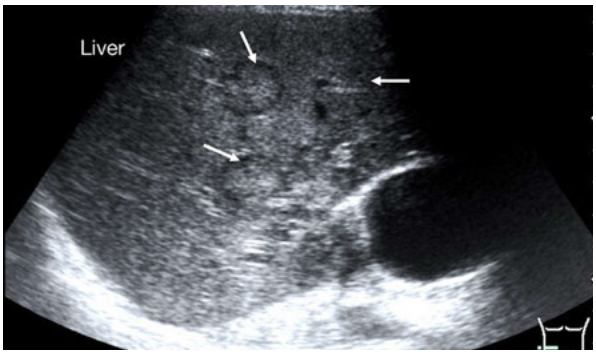


Figure 11.20. Liver metastasis: multiple focal lesions of variable echogenicity. Hypoechoic rims or haloes (arrows) can be seen around the lesions in this case.

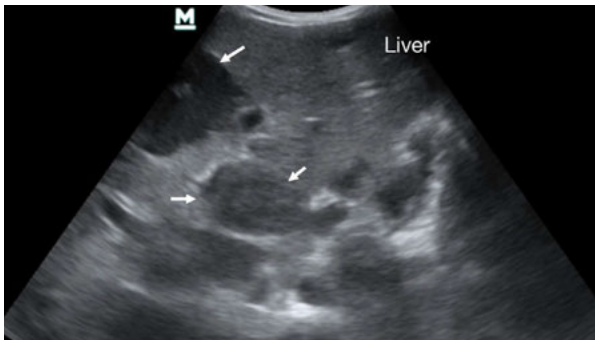
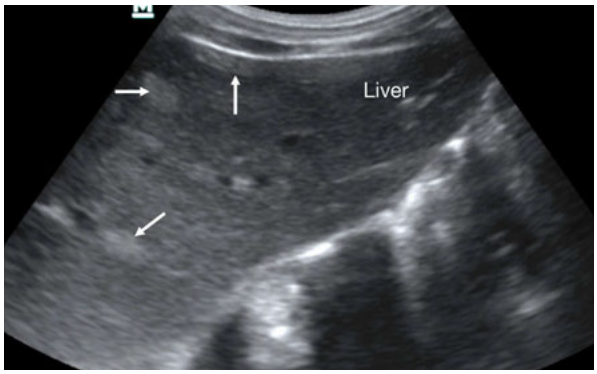


Figure 11.21. Lymphoma of the liver: Multiple, usually hypoechoic lesions (arrows) in the liver.

agnosis includes HCC and metastasis. In the event of uncertainty, a contrast-enhanced CT scan is the investigation of choice.

Focal patchy fat (Figure 11.16) is also characterised by an echo-dense, clearly demarcated lesion. It is often triangular or wing-shaped, and located near the main portal branches or the gallbladder. These findings may be caused by rapid weight reduction or cessation of alcohol intake.

Hepatocellular carcinoma (HCC) (Figures 11.17 and 11.18) occurs mainly in livers with pre-existing cirrhosis or in patients with chronic hepatitis B or C infection. Sol-



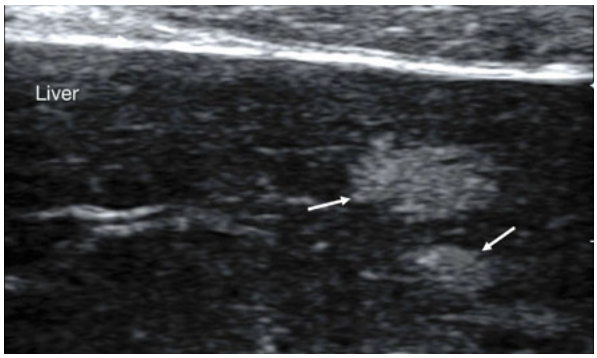
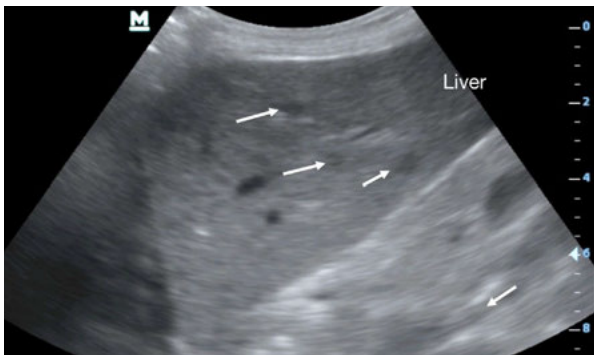


Figure 11.22. Kaposi sarcoma: Multiple ill-defined, often hyperechoic lesions (arrows) in the liver, shown using a convex transducer (top) and a linear transducer (bottom). Lesions are often more difficult to see, or are not visible at all, when the convex probe is used; to ensure that KS lesions in the liver can be seen, the linear probe should be used.



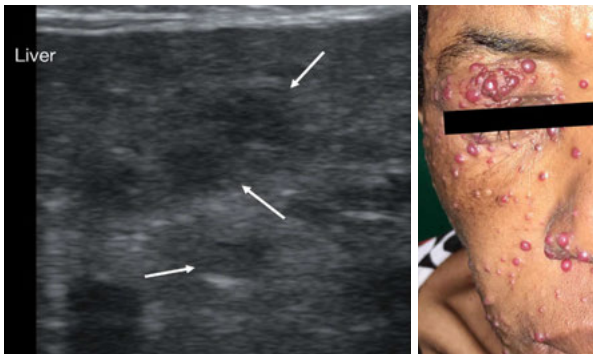


Figure 11.23. Bacillary angiomatosis: Hypoechoic lesions (arrows) in the liver (peliosis hepatis). (Top) Scanned using a convex transducer. (Bottom L) Scanned using a linear transducer. (Bottom R) Skin lesions.

itary or multiple tumours can be found. Hypoechoic and hyperechoic areas form mixed-echo patterns. In typical cases, an onion skin-like appearance of concentric bands of different degrees of darkness may be visible. The tumour tends to invade and occlude veins, as in Budd-Chiari syndrome and portal vein thrombosis.

Cholangiocarcinoma (CCC) (Figure 11.19 on the previous page) is the second most common primary hepatic cancer; it arises from the bile duct epithelium. Most CCCs occur at the bifurcation of the hepatic ducts (Klatskin tumours). Patients usually present at a late stage, with signs and symptoms suggestive of biliary obstruction, such as jaundice or deranged liver function tests. Mass-forming CCCs appear as a solid homogeneous mass of intermediate echogenicity, with a peripheral hypoechoic halo of compressed liver parenchyma. Tumours tend to be well delineated, but irregular in shape. They may be associated with either narrowing or dilatation of bile ducts. A polypoid mass in the dilated bile ducts may be seen; this is typically hyperechoic compared to the surrounding liver.

Metastases of the liver (Figure 11.20 on the previous page) appear as nodules, often in multiples. They become visible beyond 1 cm in size. Detection is easier when the nodules have a different echogenicity than normal liver tissue. Lesions may show a central lucency due to necrosis. Other possible signs are the bulging of the liver surface, displacement of vessels, and local dilatation of the bile ducts due to obstruction. The usual sites of the primary tumour include the gastrointestinal tract (particularly the colon), the breast, and the lung. Note, however, that the appearance of a tumour on an ultrasound does not necessarily point to the organ where it originated.

Lymphoma (Figure 11.21 on the previous page) can infiltrate the liver, manifesting as plain hepatomegaly or as focal infiltration. Focal infiltrates are usually hypoechoic areas which sometimes follow portal vessel structures. Diffuse infiltration patterns tend to have the worst prognosis; they are mainly diagnosed through biopsy.

Disseminated KS (Figure 11.22) may present in the liver, with hyperechoic disseminated lesions in the spleen and liver ranging from 5 to 10 mm in size. KS lesions have been reported even in the absence of visible cutaneous involvement. With larger KS lesions, a complex echo pattern with both hyper- and hypoechoic areas is observed.

In **many generalised infections,** multiple focal lesions can be seen in the liver and spleen. In cases of generalised **toxoplasmosis,** for example, multiple calcifications of a few millimetres in size, with dorsal acoustic shadow, are seen in both organs. Small hyperechoic liver lesions are also seen in disseminated **TB** and **MAI** disease. **Bacillary angiomatosis (BA)** (Figure 11.23) is characterised by cystic, blood-filled spaces in the liver; it is linked to opportunistic infection with *Bartonella henselae*. This infection also appears as multiple hyperechogenic or hypoechoic liver lesions on ultrasound examination.

The term **snowstorm pattern** is often used to describe the appearance of multiple, diffuse, small echogenic lesions in the liver or spleen on an ultrasound. Although this pattern was initially associated with disseminated ***P. jiroveci*** infections, it can be caused by other organisms, such as ***Candida*** and ***Aspergillus*****.** Comparison with histological features reveals that foci of calcification are present, but their frequency is not sufficient to explain the multiple echogenic foci. It is postulated that the interfaces caused by the fibrosis are largely responsible for the snowstorm appearance.

As we can see from this long list, there is a broad differential diagnosis for focal liver lesions. If clinically necessary to guide further treatment, and if pathology services are available, fine- or core-needle biopsy is often the only way to reach a definitive diagnosis.

Diffuse liver changes in the tropical setting

Diffuse changes of the liver are frequently overlooked or

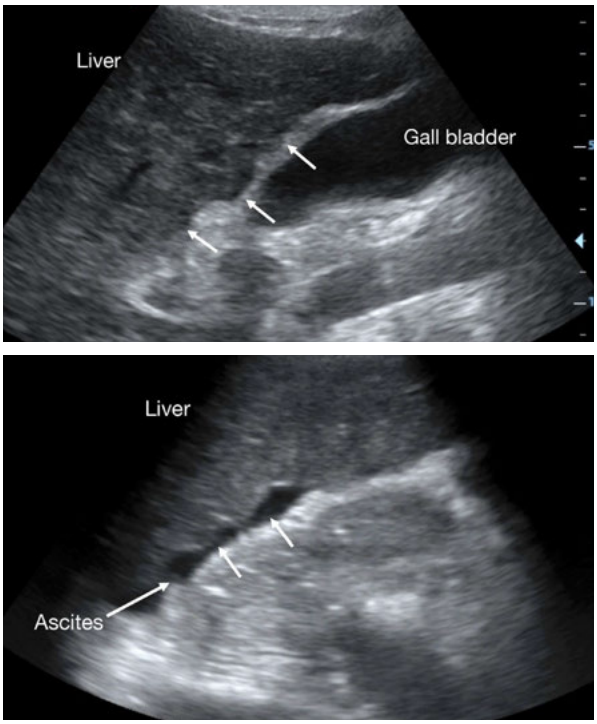


Figure 11.24. Liver cirrhosis: (Top) Irregular, nodular surface (arrows). (Bottom) This is even more obvious when ascites is present.

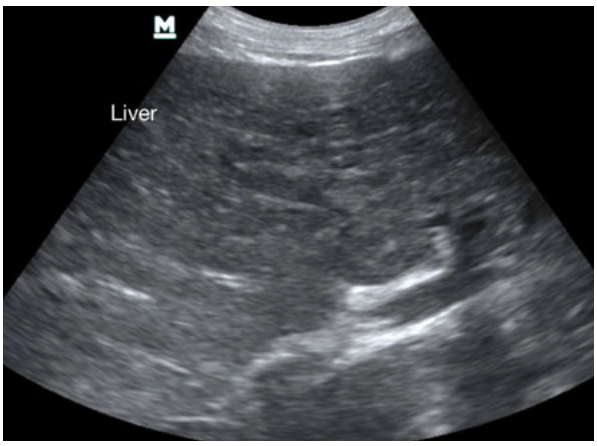


Figure 11.25. Liver cirrhosis: Coarse (salt-and-pepper) pattern of the liver parenchyma. This interpretation is often subjective, and difficult for the ultrasound beginner to make.

not recognised, as they affect the entire organ, thereby precluding comparison of heathy and diseased tissue. Only a few diffuse liver changes are relevant in our setting, the most common of which is cirrhosis. These are shown in the list below.

Differential diagnosis of diffuse liver changes

- Cirrhosis
- Schistosomiasis
- Fatty infiltration
- Granulomatous infiltration (TB)
- Venous congestion (due to cardiac failure)

Liver cirrhosis is common; it is most often associated with **hepatitis B** (hepatitis C is very rare in our setting) or toxins (such as alcohol). The diagnosis is made either at screening for cirrhosis caused by known risk factors (like hepatitis B), during evaluation of **elevated liver enzymes** or non-specific symptoms (such as **right upper quadrant pain**), or during workup for one of its complications (**ascites** or portal hypertension with **splenomegaly**).

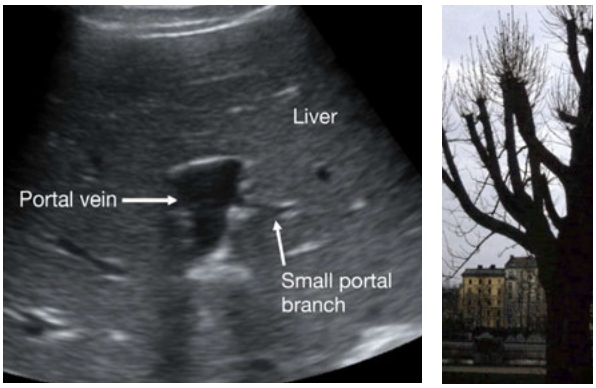
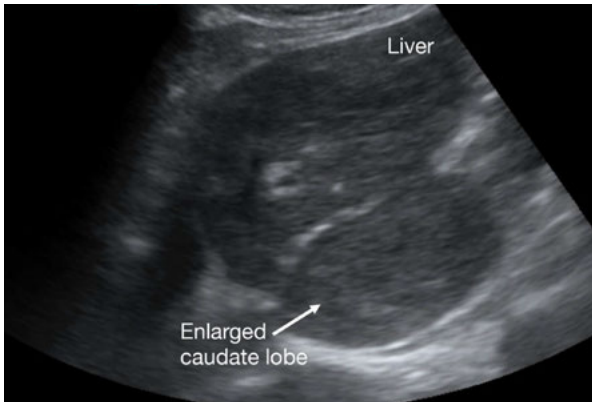


Figure 11.26. Liver cirrhosis: (L) Although the central portal vein is often enlarged in cirrhosis due to portal hypertension, the peripheral branches may not be visible; the liver as a whole will appear to have fewer vessels. (R) The pattern resembles a pruned tree with newly sprouting twigs.



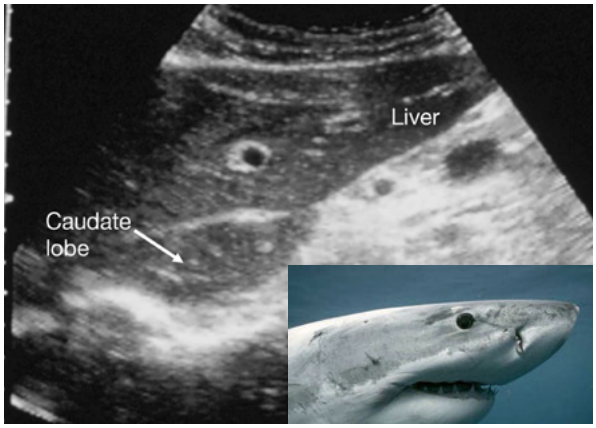


Figure 11.27. Liver cirrhosis: (Top) Enlarged caudate lobe. For comparison (bottom), an epigastric longitudinal scan with a normal caudate lobe is shown—which resembles a shark (inset).

Traditionally, cirrhosis has been classified as micro- or macronodular. However, this distinction is of limited utility, as cirrhosis usually starts out as micronodular (< 3 mm) and progresses to macronodular.

Presentation of cirrhosis varies widely; during its early stages, sonographic changes can be minimal, and thus very easy for even experienced sonographers to miss. It is worth checking for the following four signs of advancing cirrhosis:

- Nodular surface (Figure 11.24)
- Coarsened echo pattern (a 'salt and pepper' appearance) (Figure 11.25)
- Decreased vascularity in the peripheral liver (Figure 11.26)
- Caudate lobe enlargement (normal size is 2 x 5 cm) (Figure 11.27)

The **regenerative nodules** that form in cirrhosis are the reason for **surface changes;** this is probably the

easiest sign to identify. The presence of ascites makes evaluation of the liver surface even easier, because perihepatic fluid helps to delineate the edges of the liver.

The coarse, heterogenous pattern (sometimes described as a 'salt and pepper' pattern) observed throughout the liver is also due to this nodularity. However, it is a very subjective finding; one develops an understanding of normal and abnormal through experience.

Nodules and the stiffer liver tissue compress the smaller portal vein branches; this leads to **amputated portal vein,** which is large in its central parts and then abruptly narrows to a smaller diameter. The smaller branches are often barely visible at the periphery of the liver tissue; large areas of the liver may therefore seem 'vessel-free'.

Hypertrophy of the caudate lobe with concomitant atrophy of the right lobe is caused by changes in blood flow between the segments. This can sometimes be seen in the longitudinal scan of the liver in the midline.

If cirrhosis is found (or suspected), it is important to look for and describe signs of portal hypertension. These include increased diameter of the **portal vein** (> 1.4 cm), **splenomegaly** (> 14 cm) (Figure 11.28), ascites (Figure 11.29), and the presence of **collateral vessels** (Figure 11.30), especially between the spleen and left kidney.

One common mistake is the overdiagnosis of cirrhosis in the presence of massive ascites. Due to the large fluid collection around the liver, the organ appears small; as a result, one might jump to the conclusion that it is a shrunken, cirrhotic liver. The diagnosis of cirrhosis should not be made until the four characteristics mentioned above have been carefully assessed.

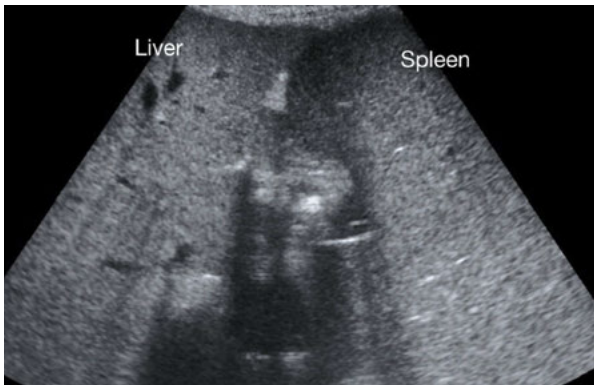


Figure 11.28. Liver cirrhosis: Splenomegaly is one finding in portal hypertension. In this epigastric transverse view, the massively enlarged spleen borders the liver directly (as if liver and spleen are kissing).

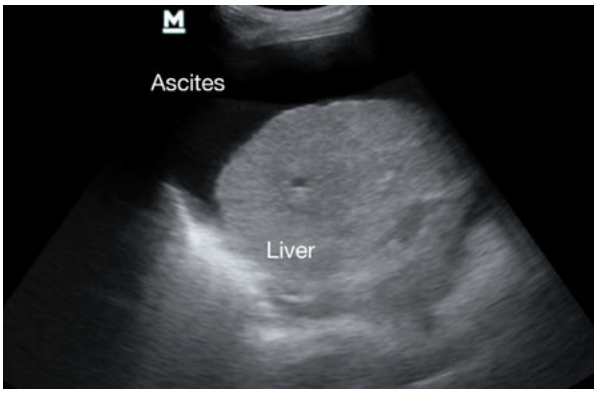


Figure 11.29. Liver cirrhosis: Ascites is frequently seen in portal hypertension due to cirrhosis.

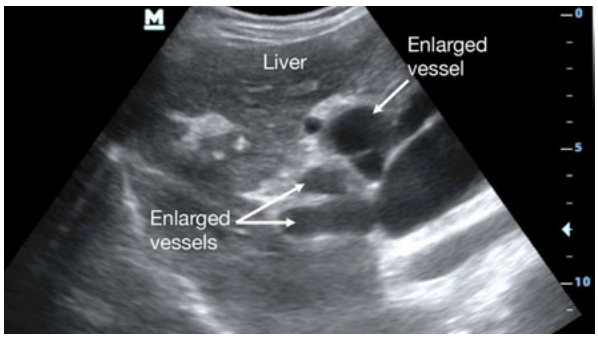


Figure 11.30. Liver cirrhosis: (L) Enlarged vessels in the portal area of the liver.

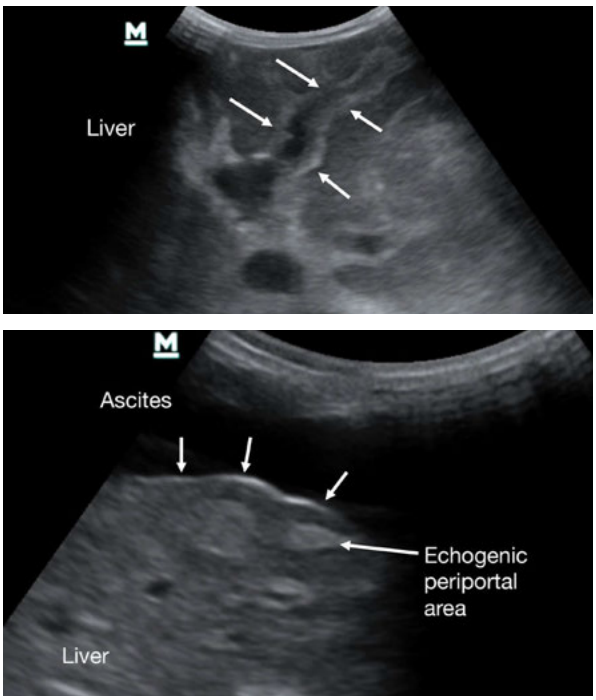
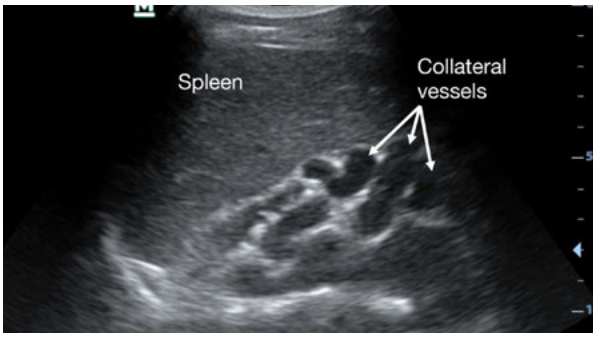


Figure 11.31. Hepatic schistosomiasis: (Top) Thickened echogenic fibrosis (arrows) around the portal vein branches (pipe stem fibrosis). (Bottom) The surface can appear bumpy (arrows), with ascites seen due to portal hypertension.

Schistosomiasis (Figure 11.31) of the liver due to *Schistosoma mansoni* is caused by the displacement of eggs into the liver from worms infecting the intestine. This induces fibrotic changes, which cause hepatomegaly. The initial signs include diffuse **echogenic foci** (a 'starry sky' appearance). Later, increasing **echogenic rings** around the portal branches are seen (equivalent to the pipe stem fibrosis seen in pathology), as well as echogenic bands extending from the main portal vein to the liver surface. As with cirrhosis, it is important to look for the same secondary changes of portal hypertension mentioned above.

Fatty Infiltration (Figure 11.32) (steatosis hepatis) is characterised by **increased organ size,** with a bulging surface and obtusely angled lower border. The main



(R) These enlarged vessels can also be seen as collaterals between the spleen and left kidney (not visible).

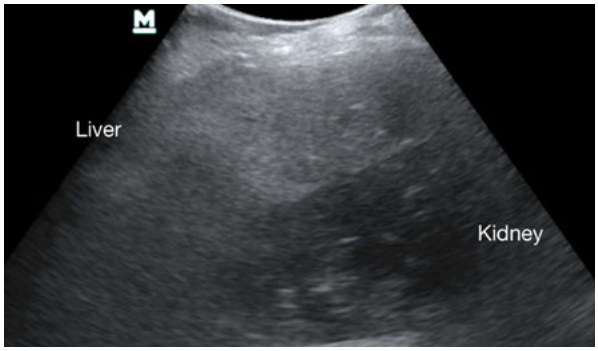


Figure 11.32. Fatty infiltration of the liver (steatotic liver): Echogenic liver, with increasingly worse image quality in the distal areas due to absorbed energy.

finding is the **brightly reflective echo pattern** known as 'white liver'. The increased echogenicity causes decreased beam penetration. Increased echoes within the liver result in decreased echoes posterior to the liver. In advanced stages of hepatic steatosis, the **veins are barely visible.** Fatty infiltration in patients with HIV is of particular interest, as it can be caused by ART. Many of the older types of ART, like stavudine (d4T), cause metabolic changes and fatty tissue in the liver. These changes can also be seen with newer NRTIs. These metabolic changes can lead to sonographically visible steatosis hepatis (also associated with lactic acidosis). As mentioned previously, **granulomatous hepatitis** in TB may yield similar sonographic changes as well.

Changes of the gall bladder and the biliary system: biliary dilitation, acalculous cholecystitis, and cholangitis

The normal **gall bladder** (Figure 11.33) is a musculomembranous sac with anechoic content. It has a variety of folds and kinks. The wall of the gall bladder is 2–3 mm thick; overall size usually does not exceed 4 x 10 cm. **Gallstones** (Figure 11.34) are echo-dense structures in the gall bladder, with a **posterior acoustic shadow;** they become visible at sizes of 2–3 mm.

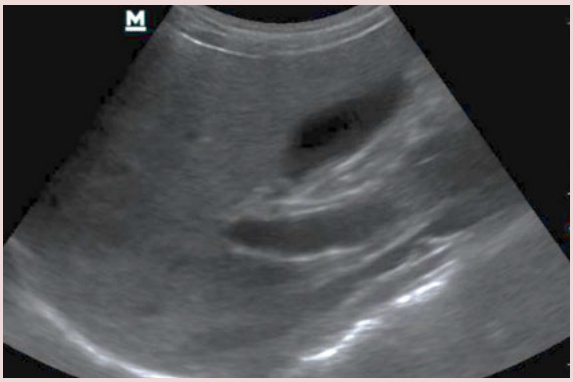


Figure 11.33. Normal-sized gall bladder with a fine wall and anechoic content.

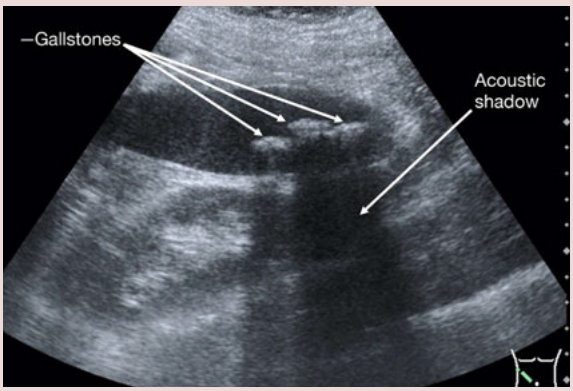


Figure 11.34. Gallstones: Multiple echogenic structures in the gall bladder, with acoustic shadow.

Gall bladder **wall thickening** is a frequent finding in patients with HIV. However, this finding must be interpreted carefully, as in most cases it is incidental, without clinical symptoms or relevance. It can be found in general oedema in cardiac failure, and ascites due to a variety of causes. It can also be seen in patients with capillary leakage syndrome (as in Dengue virus infection). Direct palpation with the ultrasound probe evokes the **sonographic Murphy sign** (in which the patient reports tenderness and pain with focal palpation). In these cases, the thick wall and distended gall bladder may point to a diagnosis of **cholecystitis** (Figure 11.35), which usually occurs only in the presence of gallstones.

In patients with HIV, cholecystitis may develop in the absence of gallstones. This condition, **acalculous cholecystitis,** is otherwise primarily seen in seriously ill ICU patients. Ultrasound-guided puncture, aspiration, and drainage of the gall bladder is a therapeutic option.

As discussed earlier, the **bile ducts** are difficult to visualise within the liver on ultrasound when they are normal and not dilated. The larger main right and left bile ducts appear as tubular structures running anterior to, and parallel with, the right and left branches

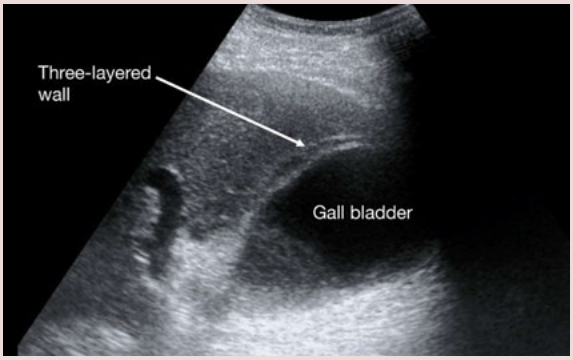


Figure 11.35. Cholecystitis: Enlarged, distended gall bladder with a three-layered wall. The gallbladder was painful on sonopalpation.

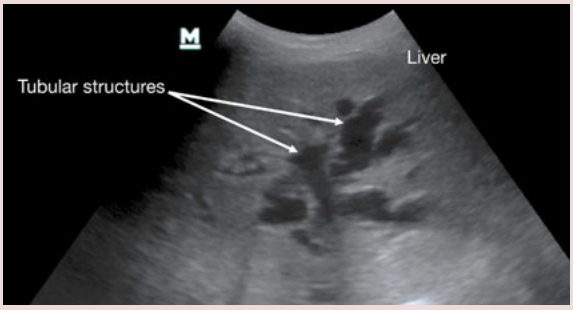


Figure 11.36. Biliary dilatation: An excess of multiple tubular structures in the central area of the liver. It is unclear which structures are portal branches, and which are dilated bile ducts.

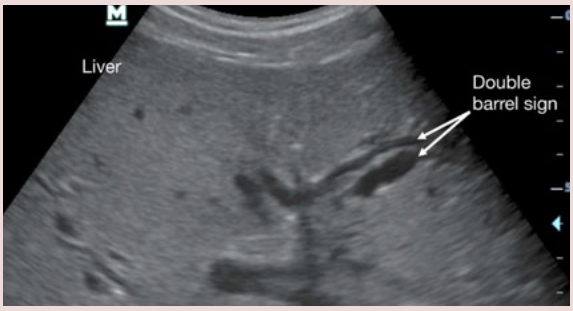


Figure 11.37. Biliary dilatation: Parallel portal vein branch and dilated bile duct (double-barrel shotgun sign).

of the portal vein. The main right and left bile ducts measure up to 2 mm in diameter. The **common bile duct** can be seen ventral to the portal vein; it appears as an anechoic tubular structure **no larger than 8 mm in diameter.**

When scanning a patient with **dilated intrahepatic ducts,** the liver often appears to have **too many tubes** (Figure 11.36). Because these 'extra' tubes do not correspond to hepatic arteries or portal veins, they must therefore be bile ducts. The ultrasound appearance of dilated ducts is often described as **'parallel channel'** or **'shotgun' signs** (Figure 11.37), because the ducts dilate to a diameter equal to or greater than that of the portal vein branch next to it. A **dilated common bile duct** can also become similar in calibre to the portal vein; here also, the two anechoic parallel structures are visible (Figure 11.38).

Whenever duct dilatation is encountered, it is important to assess the degree of **gall bladder distension** (size > 10 x 4 cm), as this may offer a clue to the level of the obstructing lesion. A distended gall bladder suggests a low common bile duct obstruc-

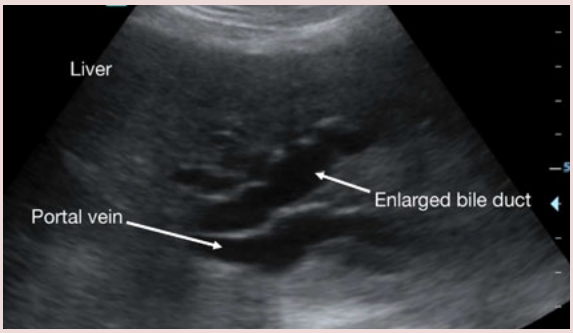


Figure 11.38. Biliary dilatation: Next to the portal vein, a second tubular structure (the common bile duct) is visible, suggesting extrahepatic obstruction.

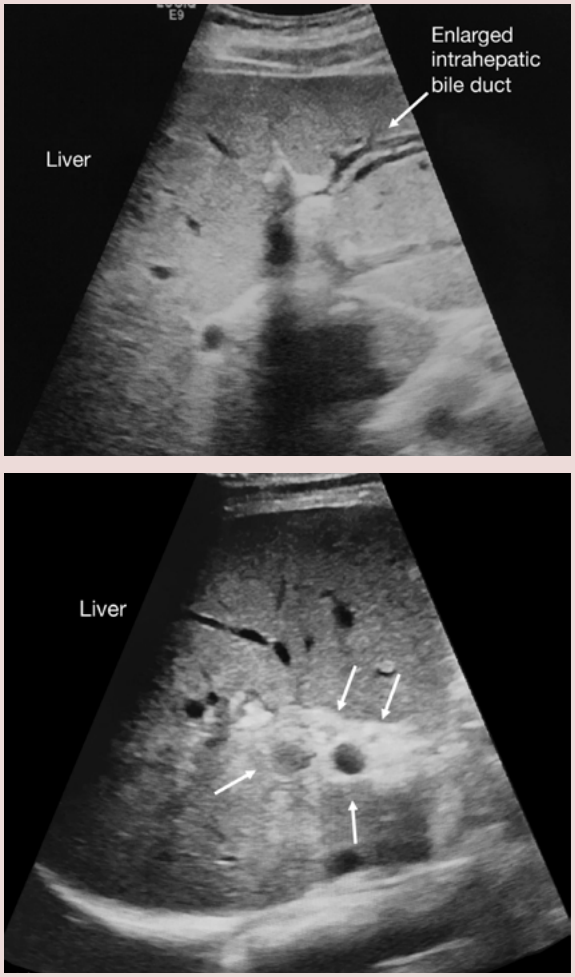


Figure 11.39. HIV cholangiopathy: (Top) Visibly enlarged intrahepatic bile ducts with echogenic surrounding. (Bottom) The central area shows increased periportal connective tissue. An enlarged common bile duct cannot be seen in this area.

tion, whereas a small gall bladder is consistent with obstruction above the level of the cystic duct. In our setting, this is also important information for the surgeon; when the gall bladder is distended (obstruction below the cystic duct), biliodigestive anastomosis (anastomosis of gall bladder to bowel) is a possible palliative procedure.

The differential diagnoses of **biliary dilatation** depend on which part of the biliary system is dilated, **intrahepatic or intrahepatic and extrahepatic.** Intrahepatic biliary dilatation alone is mainly seen as a result of cholangiocarcinoma (such as a Klatskin tumour) or recurrent cholangitis. Intra- and extrahepatic biliary dilatation are seen with a pancreatic mass (e.g., pancreatic carcinoma or pancreatitis), and as the result of external compression (e.g., lymphadenopathy). (Choledocholithiasis, a cause of biliary dilatation commonly found in Europe, is rare in our setting.)

AIDS cholangiopathy (Figure 11.39) is commonly caused by CMV virus and cryptosporidium infection. Irregular or smooth dilatation of intrahepatic bile ducts and concentric thickening of the intra- and extrahepatic biliary tree (similar to sclerosing cholangitis) may be seen. Extrahepatic strictures and papillary stenosis have also been well described.

Biliary involvement of **KS** can manifest in echogenic lesions along the intrahepatic biliary ducts adjacent to the portal branches (Figure 11.40).

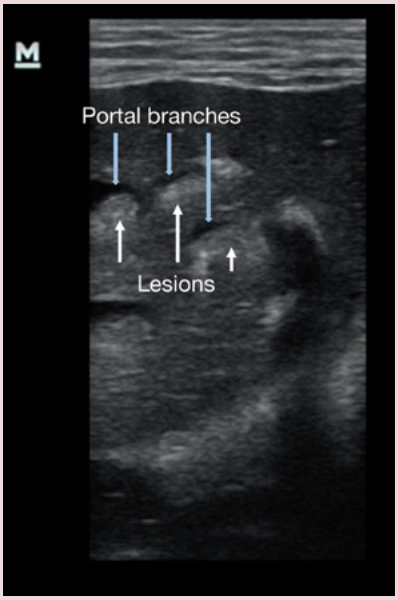


Figure 11.40. Biliary involvement in KS: Echogenic lesions along the intrahepatic biliary ducts next to the anechoic portal branches.

12. Peritoneal and Abdominal TB: More Subtle Findings

Introduction

Sonographic changes in abdominal and disseminated TB are found in the lymph nodes and spleen (see Chapter 5). However, there are other findings associated with TB in the abdominal cavity that are more subtle and thus more difficult to recognise. These findings are discussed here for clinicians who have more ultrasound experience.

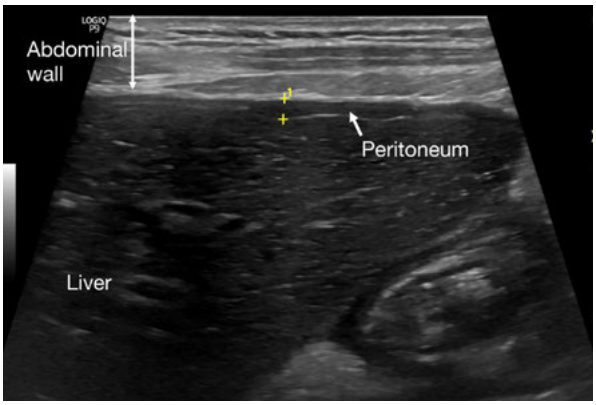
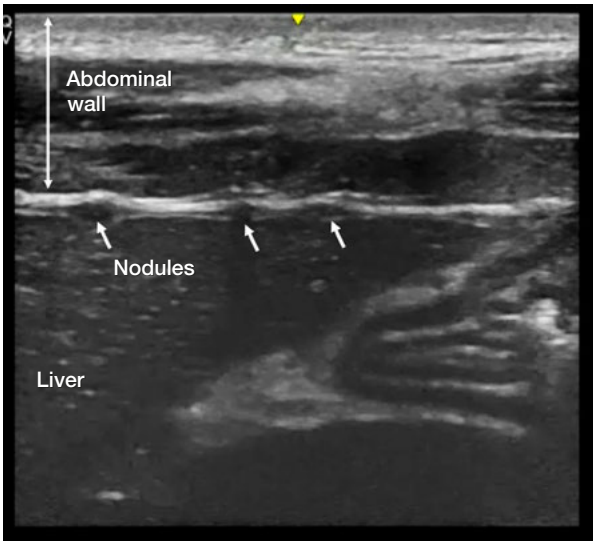


Figure 12.1. Diffuse hypoechoic thickening (~3 mm) of the peritoneum.



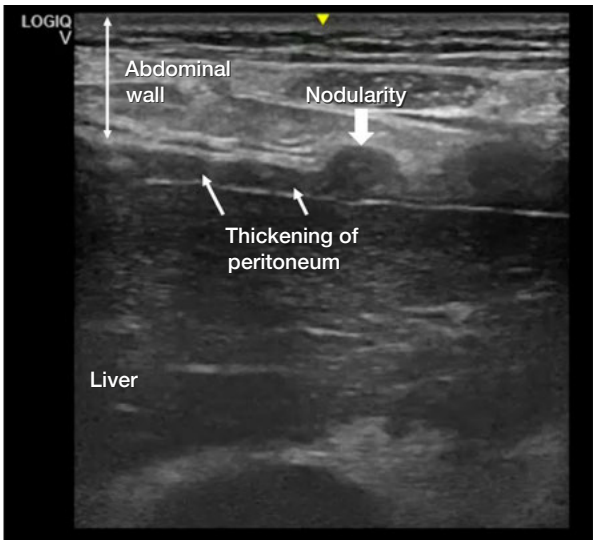


Figure 12.2. Nodular thickening of the peritoneum can be seen with the linear probe. (Top) It is especially visible between the liver and abdominal wall, with mulitple tiny nodules. (Bottom) General thickening of the peritoneum, with nodularity.

TB peritonitis

The peritoneum is the most frequent abdominal site of extrapulmonary TB (comprising about half of abdominal cases); overall, the abdomen is **third most frequent location of all extrapulmonary TB cases. TB peritonitis** is particularly prevalent in African health care settings. It is mainly caused by **haematogenous spread** and reactivation of long-latent foci or mesenteric lymph nodes, but **contiguous spread** from the bowel or fallopian tubes is also possible. The main clinical symptoms of TB peritonitis are **ascites** and **fever.** Abdominal pain, reported in about half of TB patients, is most often moderate; rarely does the pain mimic acute peritonitis.

The hallmark ultrasound finding is ascites. Ascites can be **clear or complex,** with fixed **membranes, septa, strands,** and **floating debris.** Ascites in abdominal TB has characteristics of an exudate (protein content > 2.5 g/dL); often a moderate amount of leukocytes (150–4000) will be found, with a **lymphocytic** predominance. Ascites is usually **straw-coloured,** but in some cases may be blood stained. As mentioned previously, enlarged abdominal lymph nodes is a frequent finding.

Additional findings are typically only visible using a high-frequency linear probe, with the **parietal peritoneum** a frequently affected structure. Sonographic findings include a **regular hypoechoic thickening** (Figure 12.1), which reflects chronic inflammation of the peritoneal leaflet. On closer inspection, hypoechoic **peritoneal nodules** (Figure 12.2), variable in size and sometimes confluent, can also be found within the layers of peritoneum (Figure 12.3).

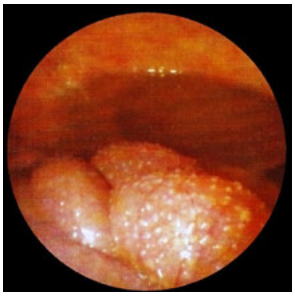


Figure 12.3. Laparoscopic appearance of peritoneal tuberculosis with multiple white granulomata on both visceral and parietal peritonea.

Involvement of the large omentum **(omental cake)** (Figure 12.4) is another finding highly suggestive of peritoneal TB. The sonographic finding is a multilayered thickening with a **hyperechoic thick central layer** corresponding to inflamed omental fat, surrounded by **thinner hypoechoic layers,** identical to the affected visceral peritoneum (Figure 12.5). Nodules in the omentum, most often hypoechoic, can be seen; these suggest enlarged lymphatic structures. When these findings are detected, TB is likely;

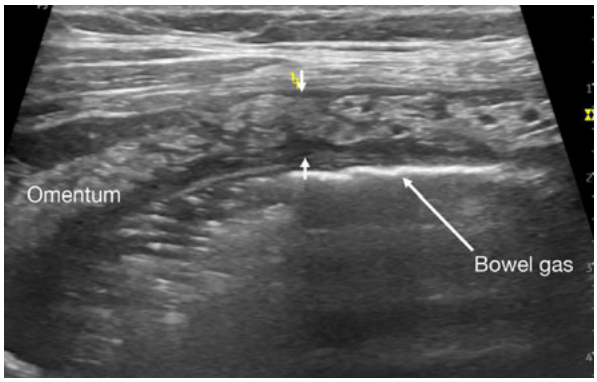


Figure 12.4. Enlarged omentum with echogenic fat due to inflammation, and hypoechoic thickening of the overlying peritoneum (small arrows).

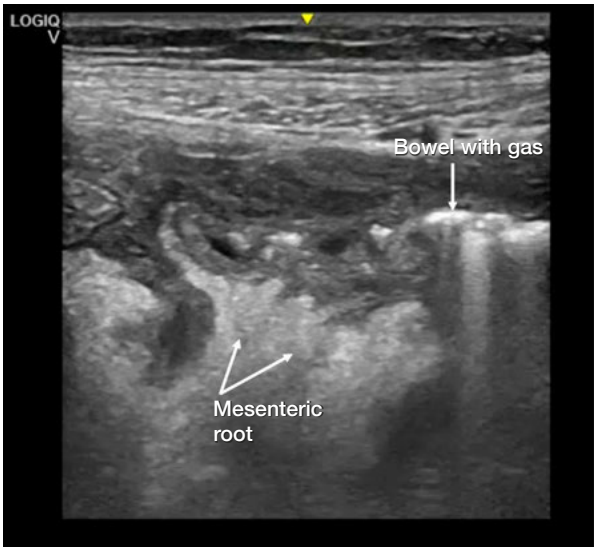
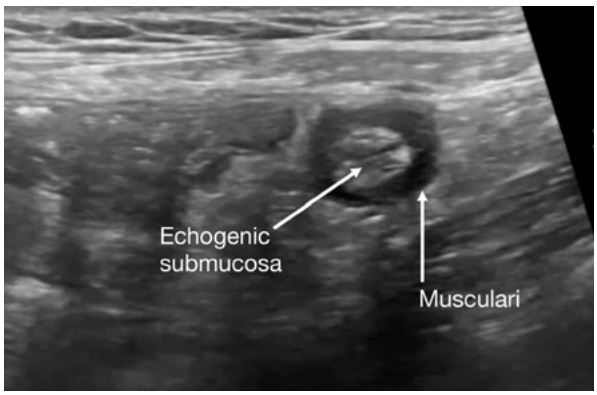


Figure 12.5. Thickening of the mesenteric root of the bowel, with high echogenicity due to inflammation.



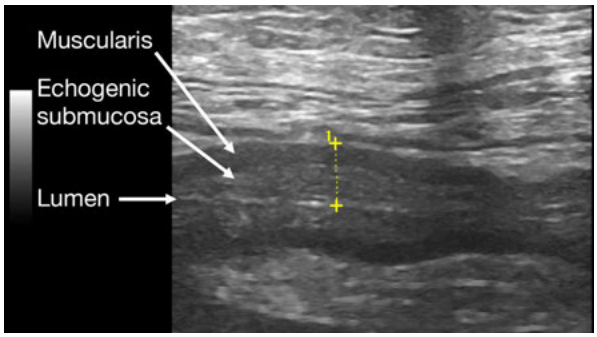


Figure 12.6. Thickened wall of a bowel loop with hypoechoic muscularis and echogenic centre (white bowel): (Top) Bowel loop cut transverse. (Bottom) Longitudinal.

nevertheless, **peritoneal carcinomatosis** remains the main differential diagnosis. If the CD4 count is very low, infection with **MAI** must also be considered.

TB of the bowel

Although tuberculosis can involve any portion of the gastrointestinal tract, there is a striking predilection for the **area of the ileocecal valve,** adjacent ileum, and ascending colon. This may be due to the abundance of lymphoid tissue located within Peyer's patches in this area. During the ultrasound exam, **concentric, hypoechoic bowel wall thickening resembling other forms of bowel inflammation** (such as inflammatory bowel disease) is seen (Figure 12.6). (A **'white bowel'** resulting from inflammation of the lymphatic vessels has been described.) Again, this finding is mainly seen when using the linear high-frequency probe; intramural abscesses and fistulae may also be seen with this transducer. In some patients,

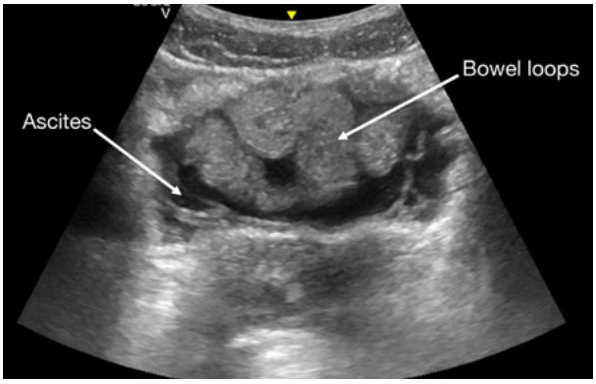
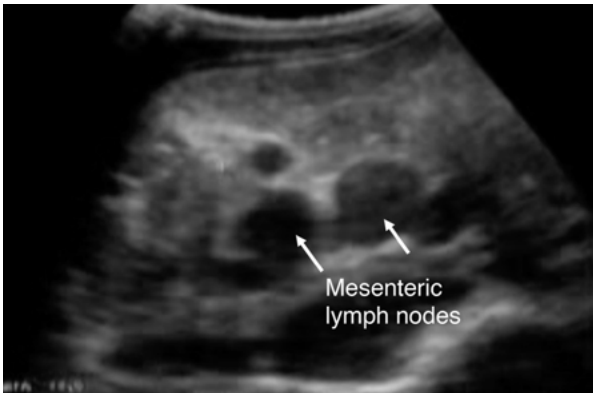


Figure 12.7. Bowel conglomerate surrounded by local ascites containing septate and strands.



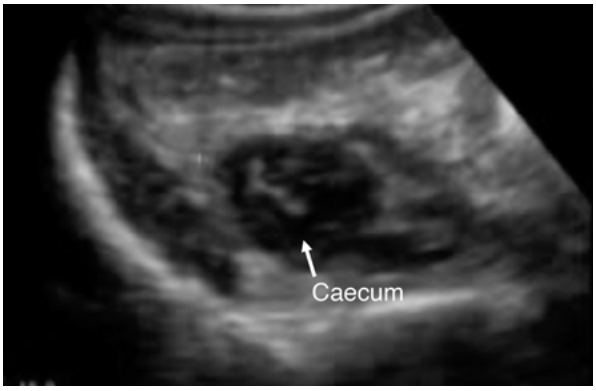


Figure 12.8. Enlarged lymph nodes in the caecal area: Hypoechoic lymph nodes in the mesenteric root (top) next to the caecum with thickened wall (echogenic center = gas) (bottom).

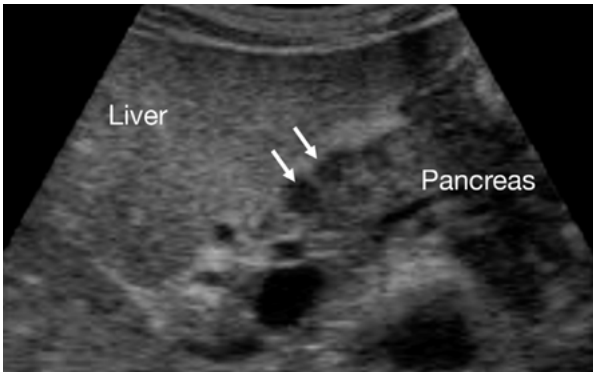


Figure 12.9. TB of the pancreas: Multiple hypoechoic nodes (arrows) in the area of the pancreas lead to a secondary pancreatitis.

conglomerate masses of thickened bowel loops are found (Figure 12.7). Extraintestinal signs, such as enlarged mesenteric lymph nodes and mesenteric thickening, are frequent accompanying findings (Figure 12.8). The differential diagnosis of thickened bowel loops in a patient with HIV includes **TB or MAI infection,** other **enteric infections, KS, and lymphoma.**

TB of the pancreas

Pancreatic involvement in tuberculosis is rare. When it does occur, in addition to the systemic symptoms of tuberculosis, it may present with **symptoms of pancreatitis.** Focal tuberculomas can obstruct the pancreatic duct, thereby causing secondary pancreatitis. **Hypoechoic lesions in the pancreas** are seen (Figure 12.9 on the previous page); radiologically (and even during laparotomy), these may resemble pancreatic carcinoma. In the appropriate epidemiological and clinical setting, TB treatment should be initiated. Steroids can be added, as they will induce a faster shrinking of inflammatory tissue, thereby alleviating the pancreatic obstruction.

13. Ultrasound of the Lung

Introduction

The **classic imaging** modality for examining pathological processes in the thorax is the **CXR.** This is also the first choice for assessing pulmonary changes in TB patients and HIV patients with respiratory symptoms. However, CXR is not immediately available in some settings—or, when it is available, the findings may be ambiguous. **Ultrasound** of the chest can be helpful in detecting and characterising intrathoracic processes, **especially those close to** or within **the chest wall.** For some indications, ultrasound may even be superior to CXR.

Presentations for which ultrasound may be helpful include **acute respiratory distress, dullness on percussion, reduced breath sounds,** and ambiguous **opacities on CXR.** In this last scenario, ultrasound can be used to differentiate suspected pleural effusions from large areas of consolidations or an elevated hemidiaphragm.

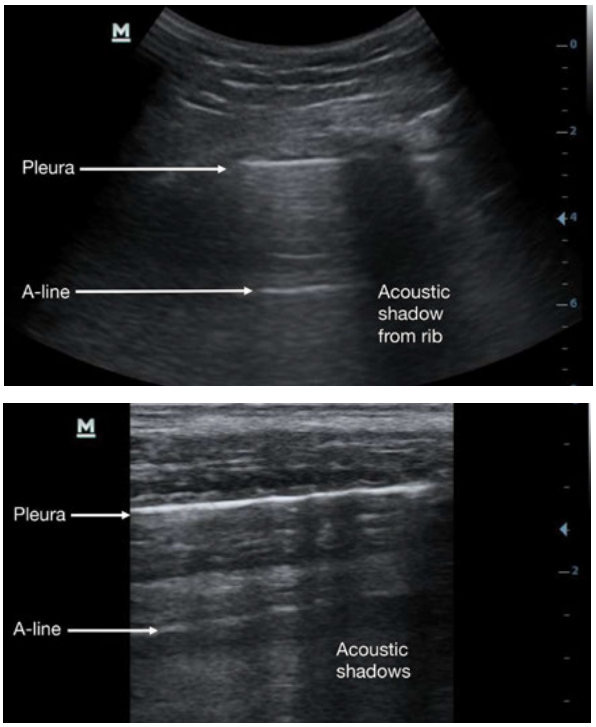


Figure 13.1. Convex (top) and linear (bottom) ultrasound scans demonstrating horizontal, parallel A-lines.

Technique and typical ultrasound findings

The scan is performed using an intercostal approach, with the patient in a **sitting or supine position.** Anterior, lateral, and posterior scanning on the longitudinal plane must be performed to detect effusions. It is important to make sure that the **hemidiaphragm** is identified above the liver and spleen to be certain that any fluid seen lies in the hemithorax rather than in the subphrenic spaces. In a **supine patient,** fluid accumulates **posteriorly** and may not be visible in the lateral views unless it is a large effusion. It is therefore important to place the probe very dorsal, close to the patient's bed (think **'knuckles to the bed'**—the back of your hand should be touching the bed to ensure that you are sufficiently dorsal). Because the pleural space is located only a few centimetres below the skin, a high-frequency linear transducer may be suitable for slim patients; it may be necessary to use a lower-frequency abdominal probe for larger patients, or to assess deeper structures.

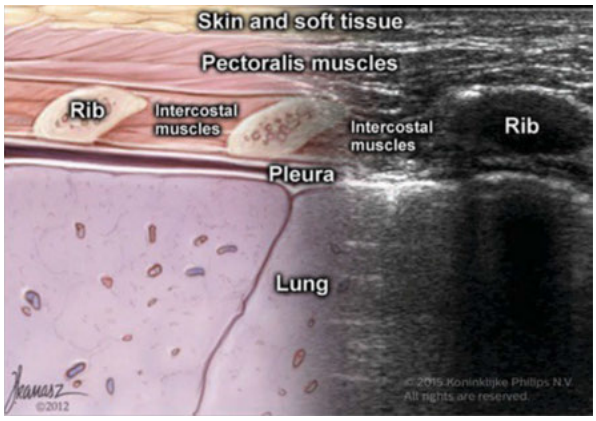


Figure 13.2. Anatomical drawing fused with a with corresponding ultrasound image demonstrating the superficial chest wall structures. (Source: Adapted from Stone MB: Point-of-care lung ultrasound. Philips tutorial. http://viewer.zmags.com/publication/1f3688e9#/1f3688e9/8.)

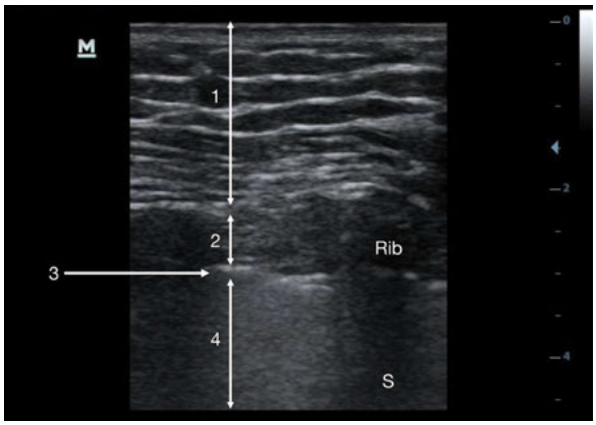


Figure 13.3. Chest wall in ultrasound: 1) Skin, fat, and muscle. 2) Ribs and intercostal muscles. 3) Pleural line. 4) Lung. S) Acoustic shadow from the rib.)

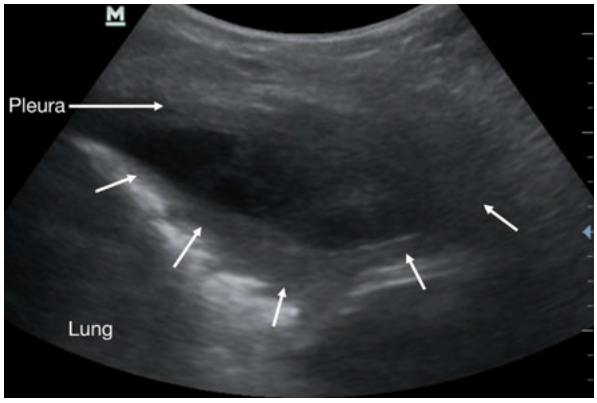


Figure 13.4. Hypo- to anechoic pleural empyema (arrows) between thoracic wall and lung

In a patient with normal anatomy, the **subcutaneous fat** is hypoechoic. The **ribs** appear as hyperechoic (ossified) structures with a clean acoustic shadow. Between the ribs, intercostal muscle is visible as a linear striation. Air in normal lung parenchyma obscures deeper views by causing massive artefacts (reverberation echoes parallel to the pleura, called **A-lines** (Figure 13.1). The **pleural line,** formed from the visceral and parietal pleura, can be identified on intercostal views behind the posterior border of the ribs. Once this view is obtained, the transducer is held still to observe **lung sliding** at the pleural interface during the respiratory cycle (Figures 13.2 and 13.3).

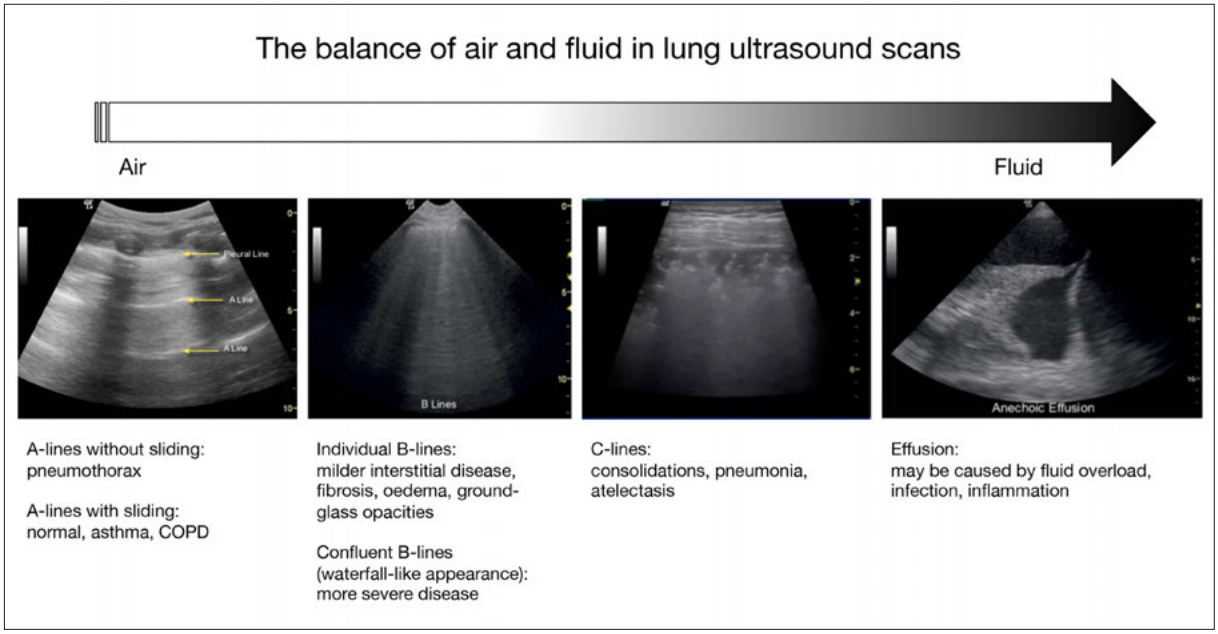


Figure 13.5. Air and fluid in lung ultrasound scans.

Pathological findings

Pathological findings can be broadly grouped according to **air-fluid ratio.** Uncomplicated **effusions contain fluid only; lung consolidations** contain **more fluid than air;** interstitial abnormalities, such as **interstitial oedema,** contain **more air** than interstitial fluid; **normal lung parenchyma contains air** with normal interstitium; and a **pneumothorax consists solely of air.** The air-fluid ratio and the distribution of fluid and air together determine which ultrasound phenomena and artefacts will appear (Figure 13.5).

Transudates and **uncomplicated parapneumonic effusions** appear as anechoic homogenous fluid (see Chapter 4). **Empyema** (Figure 13.4 on the previous page), haemothorax, and complex parapneumonic effusion are three entities that can contain dense strands and appear multiloculated. On occasion, they can be difficult to distinguish from consolidated lung.

Consolidated lung presents as hypoechoic, tissue-like replacement of normal lung without signs of lung collapse. Consolidations are sometimes called **C-lines,** although they are not really lines (Figure 13.6). A consolidation may be small, focal, and subpleural, or it may be large and surrounded by parapneumonic fluid. With pneumonia, pathologists have long used the term **hepatisation** to describe the liver-like appearance of the lung as it becomes increasingly echo-dense. This is a helpful reminder: on ultrasound examination, a consolidated lung also resembles **liver parenchyma.** Within the consolidation, the larger bronchi remain filled with air and appear as intensely echogenic bands. These are equivalent to the **air bronchogram** seen on CXR. Consolidations may be caused by infection (pneumonia) or (less often) malignancy.

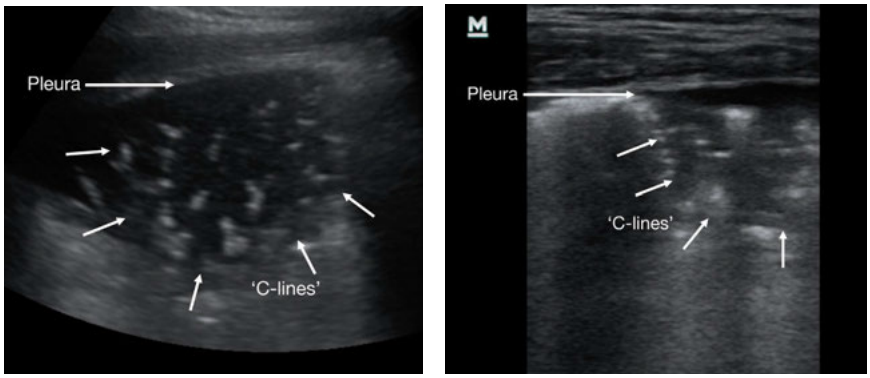


Figure 13.6. Convex (L) and linear (R) ultrasound scans demonstrating tissue-like structure in the lungs. Consolidations (sometimes called 'C-lines') may contain visibly echogenic, string-like air-bronchograms.

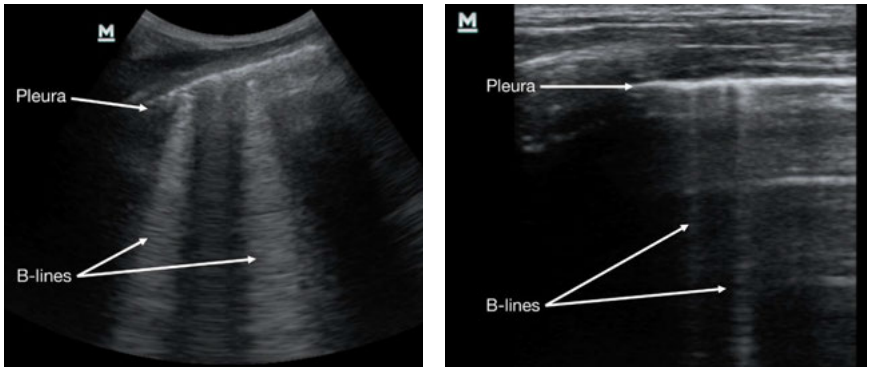


Figure 13.7. Convex (L) and linear (R) ultrasound scans demonstrating vertical, laser-like B-lines

Lung collapse (atelectasis) is difficult to differentiate from consolidation, as the two often occur together in pneumonia. However, unlike consolidation, healthy collapsed lung (as in obstruction of central airways or secondary to pleural fluid) is more hyperechoic, with the collapsed part having very little volume. With partial collapse, the tip of the lung is triangular in shape and borders normal-looking parenchyma. The lung tip may re-expand on inspiration; it can often appear to 'float' in the fluid.

Interstitial syndromes are sonographically characterised

by so-called **B-lines** (Figure 13.7). B-lines occur when sound waves encounter a mixture of air and fluid (as with interstitial oedema, where the increased fluid in the interstitium is surrounded by aerated alveoli). This creates an artefact, which appears as a laser-like **vertical hyperechoic line** that originates in the pleura and extends to the bottom of the screen without fading. The lines move synchronously with lung sliding. Because B-lines are frequently encountered in normal subjects, only the presence of **more than three B-lines in one intercostal space** in the longitudinal plane is considered pathologic. In addition to interstitial oedema, B-lines can also be seen in **interstitial pneumonias,** partial atelectasis, or with lung contusions. B-lines in multiple areas of the thorax are present in **pulmonary oedema, acute respiratory distress syndrome (ARDS), or diffuse pneumonitis.** Differentiating cardiogenic from noncardiogenic pulmonary oedema or pneumonitis is not possible. (Because the topic of interstitial lung diseases in HIV patients is a difficult one, it is discussed in more detail in the box at the end of this chapter.)

Finally, ultrasound is a very useful tool for diagnosing **pneumothorax.** In a healthy lung, **pleural sliding** is visible during the respiratory cycle. This **sliding will be absent** if the lung has collapsed due to pneumothorax. In this case, only static **A-lines** will be visible. If the patient is examined while in a supine position, air in the pleural space rises to the anterior chest wall. Therefore, it is particularly important to investigate the parasternal and midclavicular regions to exclude a significant pneumothorax. If **normal lung sliding** is observed, **pneumothorax can be excluded** at that intercostal space. However, if **no lung sliding** is observed, pneumothorax may be present. **Absent ventilation, pleural adhesions, bullae, or pleurodesis** may also cause loss of sliding. If the edge of the collapsed lung can be seen moving into and out of the scan beam (known as a **lung point sign**), this is an additional sign of pneumothorax.

Diagnostic questions and therapeutic implications

Completely white hemithorax on CXR: effusion or consolidation?

This is probably the most frequent question related to lung ultrasound. Large pleural effusions may lead to complete opacification of the entire hemithorax, usually with a mass effect on the mediastinum (shifting away from the effusion!). In some cases, large effusions may be confused with entirely consolidated lungs. In these cases, it is important to keep in mind that in a case of consolidation, associated midline shift would occur towards the consolidation.

Pleural fibrosis and pleural thickening due to previous TB infection may also appear radiologically as an opacification; this finding can be confused with an effusion. With pleural fibrosis (scarring), the pleural space becomes diffusely enmeshed in **fibrous material.** This appears as a **solid, homogenous structure** between the wall of the thorax and the lung. It usually has a hypoechoic (but not anechoic) appearance, or it may have an echogenicity similar to that of other solid structures nearby (such as intercostal muscle). Attempts to aspirate the pleural effusion will not yield fluid. Ultrasound can be used to differentiate between effusion, lung consolidation, and pleural thickening; this can be very useful in guiding drainage of fluid collections, which can provide immediate relief to the patient and aid the diagnostic process.

Diminished breath sounds: effusion or pneumothorax?

In patients with diminished breath sounds, ultrasound can help in cases where CXR is not easily accessible. The features of a pneumothorax (A-lines, absence of lung sliding) are very different from those of an effusion (anechoic fluid). Identification of a pneumothorax, especially in trauma patients, may warrant tube drainage. As discussed above, identification of effusions requires further workup.

Local swelling or pain: thoracic wall lesion?

Focal swelling of the thoracic wall, which can be seen or palpated during a physical examination, may be further characterised using ultrasound. It is possible to trace swelling to its origin point within ribs or other structures. **Metastasis** (for example, in the rib) and tumours may cause these changes. In rare cases of **empyema necessitans,** longstanding pleural TB infection dissects through the pleural lining and into the adjacent soft tissue of the chest wall. It follows the fascial planes and may track towards the retroperitoneum or the paravertebral area. It may also present as localised swelling of the chest wall. The continuity of the subcutaneous collection to the pleural space can be visualised sonographically.

Focal peripheral opacity: pleural lesion?

Masses seen on the CXR are often intrapulmonary, and therefore not accessible using ultrasound. Chest CT is the best way to examine the lesion further. In some cases, the lesions (whether caused by infection or metastasis) may have **contact with the chest wall.** In such cases, they may be seen as a hypoechoic structure adjacent to the lung. Ultrasound can be used to guide needle biopsy to obtain material for histological or microbiological investigation.

Pearls and pitfalls

- If no A-lines or B-lines are visualised initially, try sliding or angling the probe.
- Always confirm the position of the hemidiaphragm to ensure that any fluid seen lies within the chest.
- When searching for pneumonia or empyemas, remember to scan the entire hemithorax, as they may be focal and in a nondependent location.

PCP and COVID-19 interstitial lung infections

As discussed earlier, B-lines are the hallmark of interstitial lung changes. Though **multiple B-lines** are absent under normal conditions (maximum 0–3 lines per view), they are present in a **variety of alveolar-interstitial diseases.** The most common underlying condition is **pulmonary oedema** (either cardiogenic or noncardiogenic). If generalised B-lines are present and the clinical picture is suggestive of pulmonary oedema, diuretic treatment should be initiated.

Additionally, **viral pneumonias** associated with cytomegalovirus (CMV) and varicella-zoster virus (VZV) or SARS-CoV-2 show a similar interstitial pattern. **COVID-19** changes are seen predominantly in the **periphery of the lung** (Figure 13.8), rendering them amenable to ultrasound evaluation. Changes tend to have a bilateral and basilar predominance. Multiple **B-lines** (Figure 13.9) and small **subpleural consolidations** (Figure 13.10) can be seen in cases of milder disease. With severe, progressive disease, alveolar consolidations can give the lungs a tissue-like appearance (as with bacterial pneumonia). These may be associated with pleural effusions, which are practically never seen in mild cases of COVID-19. During recovery, the reappearance of bilateral A-lines can be ascribed to increasing air in the diseased lung.

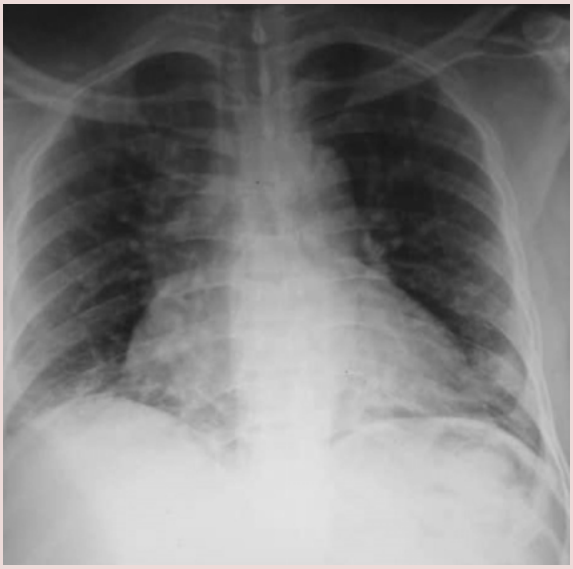


Figure 13.8. Chest X-ray of a patient with COVID-19 pneumonia, showing a ground-glass appearance, especially in the periphery of the lung.

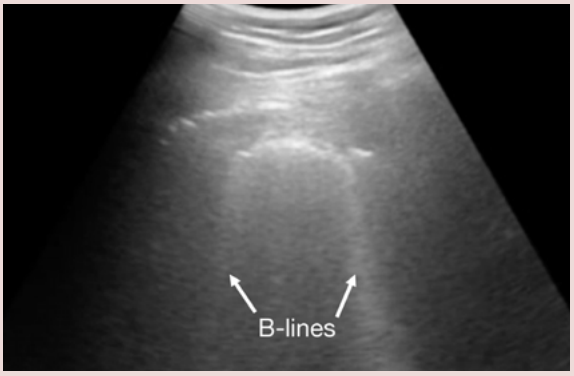


Figure 13.9. Lung ultrasound changes in mild case of COVID-19: B-lines as sign of the interstitial oedema.

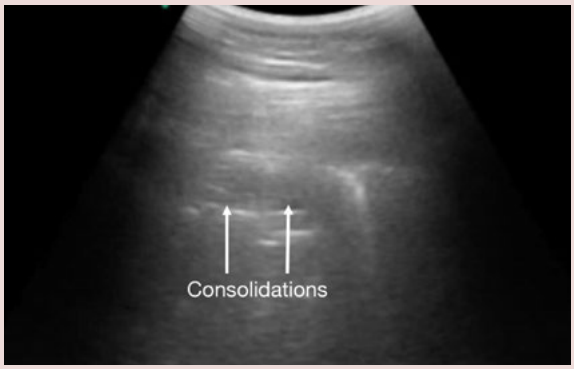


Figure 13.10. Lung ultrasound changes in a more severe case of COVID-19: Small hypoechoic consolidation in the subpleural lung area.

B-lines and subpleural consolidations are also suggestive of another interstitial infection, **pneumocystis pneumonia (PCP)** (Figures 13.11 and 13.12). In more severe cases of PCP, **consolidations with cystic changes** have been described (Figure 13.13). These are characterised by a hypoechoic consolidation (similar to all other consolidations), with widely scattered echogenic regions suggestive of air-containing cysts. Findings such as lung consolidation with linear air bronchograms or pleural effusion should prompt suspicion of other aetiologies, as these are rare in PCP.

Finally, an interstitial pattern of B-line artefacts disseminated throughout multiple lung fields in combination with subpleural granularity on ultrasound can indicate **miliary TB.** As miliary TB is defined by systemic disease, changes are usually seen in several lung zones.

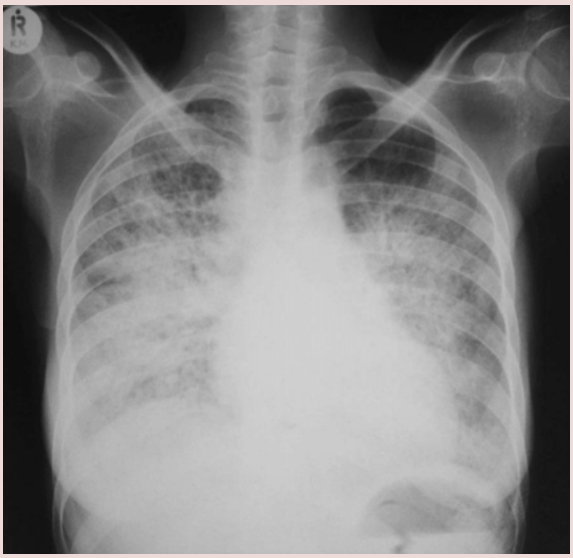


Figure 13.11. Chest X-ray of a PCP patient, showing a ground-glass appearance, especially in the central lung areas.

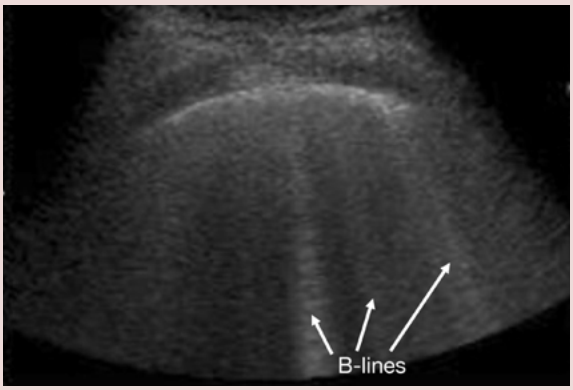


Figure 13.12. Lung ultrasound changes in a mild case of PCP: B-lines as a sign of the interstitial oedema.

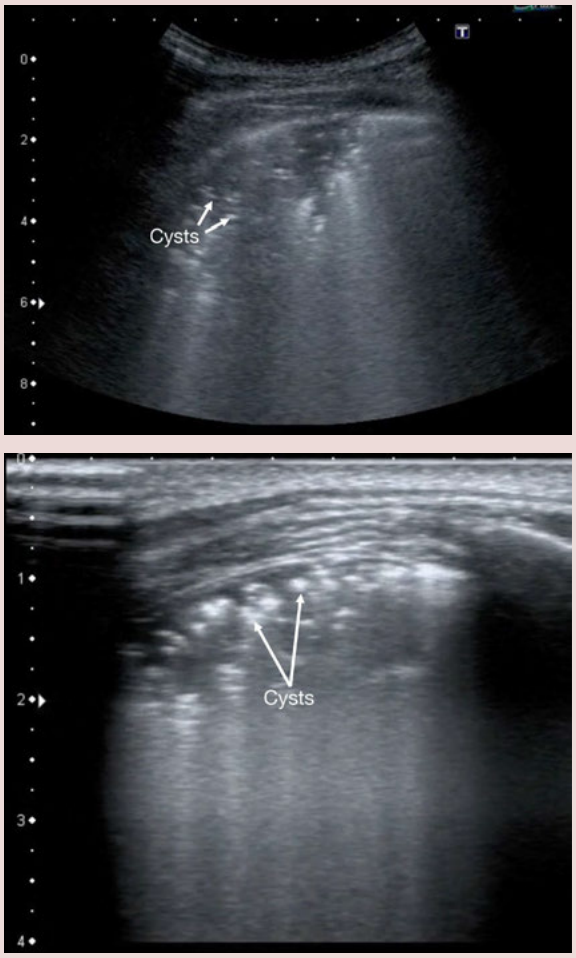


Figure 13.13. Lung ultrasound changes in a severe case of PCP: Both convex and linear scans show hypoechoic consolidation with irregularly disseminated gas-containing echogenic cysts.

14. Interventional Ultrasound: Aspiration and Biopsy

Introduction

Ultrasound-guided puncture uses sonography to both **localise targets of puncture** and identify **potentially dangerous structures to avoid.** It can help the clinician obtain samples to differentiate causes of fluid collection (intra-abdominal fluid and intrahepatic collections, such as abscesses, and collections in the soft tissue), drain collections, and facilitate placement of central venous lines.

Ultrasound can help not only to guide interventions (placement of the needle), but also to **determine whether a procedure is indicated** in the first place.

Some guiding principles to keep in mind before considering intervention:

- Ultrasound-guided diagnostic procedures should be performed only **if a diagnosis cannot be made** using less invasive (or noninvasive) methods (such as imaging).
- Laboratory or pathological diagnostic facilities must be available; **confirmation of a definitive diagnosis should be associated with treatment options.**
- Therapeutic procedures must be **more effective or less invasive** than conventional methods (such as surgery).
- Before any ultrasound-guided procedure is performed, the patient must be **informed** of its benefits and risks, **and their consent obtained.**
- Before an ultrasound-guided procedure is done, bleeding disorders should be ruled out. Ideally, **prothrombin activity** (> 50%) or the **INR** (< 1.5) and **platelet count** (> 50,000/mL) should be measured. An alternative to these measurements is **normal bleeding time** (the Duke method), in which the patient's earlobe or fingertip is pricked with a lancet after having been swabbed with alcohol. (The prick is about 3–4 mm deep.) The patient then blots the blood every 30 seconds with a filter paper. The test ends when bleeding ceases, which is usually within 2–5 minutes. Additionally, a **thorough history** of any abnormal bleeding may be helpful.

Contraindications include uncooperative patients, known or suspected coagulation or bleeding disorders, and long or dangerous puncture routes.

Aspiration of pleural, pericardial, and ascitic fluids

Pericardiocentesis, pleurocentesis, and paracentesis are procedures that frequently have both diagnostic and therapeutic utility. These are amongst the most common examples of procedures that ultrasound can help to make safer for (and thereby reduce risk to) the patient.

Pleurocentesis

Pleurocentesis, or pleural tap, is usually performed using **a blind technique,** in which ultrasound is used to localise the area of fluid collection, and then a needle is inserted at that location to withdraw the fluid. Ultrasound guidance facilitates correct needle insertion and positioning, particularly when **small fluid collections** or **septated collections** are targeted.

Generally, it is inadvisable to extract **more than 1–1.5 L** of fluid at one time; draining more than that in a single session may **increase the risk of pneumothorax—**and, more importantly, the risk of **re-expansion pulmonary oedema.** The reported incidence following drainage of a pleural effusion (and pneumothorax) is between 0% and 1%; however, it may be more prevalent, as re-expansion pulmonary oedema is often clinically mild, and thus goes undetected.

Symptoms of re-expansion pulmonary oedema include **chest discomfort, persistent cough, production of frothy sputum, and dyspnoea.** The onset of symptoms in most patients is **one to two hours** after lung re-expansion—but it can take up to 24 hours after the procedure for symptoms to appear. Risk factors include age between 20 and 40 years, duration of collapse greater than 72 hours, and rapid lung expansion with drainage of large volumes of pleural fluid. **If a patient reports vague chest pressure during pleurocentesis, this may indicate a drop in intrapleural pressure, and the procedure should be stopped.**

Pericardiocentesis

Using ultrasound guidance during pericardiocentesis reduces the risk of lung or heart injury that exists when using the blind puncture technique. Generally, the procedure has both **diagnostic and therapeutic value,** as the pressure of pericardial fluid can tamponade the heart and interfere with cardiac filling. We rarely perform pericardiocentesis for diagnostic purposes alone.

In some cases, a permanent drainage catheter is necessary; it can be placed using the Seldinger technique. In most cases, drainage of fluid followed by administration of steroids or anti-TB/anti-KS treatment is sufficient.

In addition to the subxiphoid approach, which is typically described in cardiology textbooks, ultrasound enables the use of a **transcostal approach,** similar to pleurocentesis. This is done by localising a pouch of pericardial fluid and draining it using a normal IV cannula (which is usually long enough to reach the pericardial sac). Many patients experience rapid clinical improvement despite the continued presence of residual pericardial effusion. It is worth remembering that **the aim is reduction of pressure in the pericardial space** rather than a particular volume of drainage; draining as little as 100 mL of fluid might be enough to resolve the tamponade.

Paracentesis

Complications occur in about 1% of procedures; most frequently reported are **bleeding, infection, and bowel perforation.** Ultrasound guidance ensures correct positioning of the needle, especially when the sample to be collected is small or loculated. Sonography is often used when problems with the procedure are expected. Again, the tap is usually performed using the **blind technique;** ultrasound is used only to find a good location for puncture, not to guide insertion of the needle.

Often, **the goal of paracentesis is to provide symptomatic relief** of abdominal tension for as long as possible. At the same time, complications such as **paracentesis-induced circulatory dysfunction (PICD)** should be avoided. PICD usually occurs only following large-volume paracentesis (> 5–6 L), and results in **faster reaccumulation of ascites, hyponatraemia, renal failure,** and **shorter survival.** Therefore, some guidelines suggest giving intravenous albumin when more than 5 L

of fluid is removed; however, this is costly. **One of the simplest ways to prevent PICD is to limit the volume of fluid removed to 5 or 6 L at a time.**

Needle size

In most cases, an IV cannula will be used to drain fluid from the cavities. It is therefore good to remember the **sizes** and **flow rates** of commonly available cannulas, shown here in Table 14.1.

Table 14.1. Cannula flow rates

Colour	Size (gauge)	Outer Diameter	Flow Rate
Orange	14G	2.1 mm	240 mL/min
Grey	16G	1.7 mm	180 mL/min
Green	18G	1.3 mm	80 mL/min
Pink	20G	1.1 mm	54 mL/min
Blue	22G	0.9 mm	33 mL/min

Biopsy and aspiration of focal processes

Ultrasound-guided puncture uses sonography to localise targets and dangerous intervening structures. Because the procedure is invasive and can cause harm to the patient, it is essential to receive **practical training** from a **colleague who is experienced in the procedure.**

Puncture techniques

Three different techniques can be used:

1) The target is localised, and the point of puncture marked on the skin; then the transducer is removed, and the puncture performed **'blind',** without further ultrasound guidance.

2) The target is localised using ultrasound, then the needle is inserted next to the transducer. Next, the needle, which appears on screen as an echogenic structure, is advanced under continued ultrasound guidance until it reaches the target. The path of the needle is visible on the screen (Figure 14.2); the needle must be kept on the plane of the transducer using one's **free hand**.

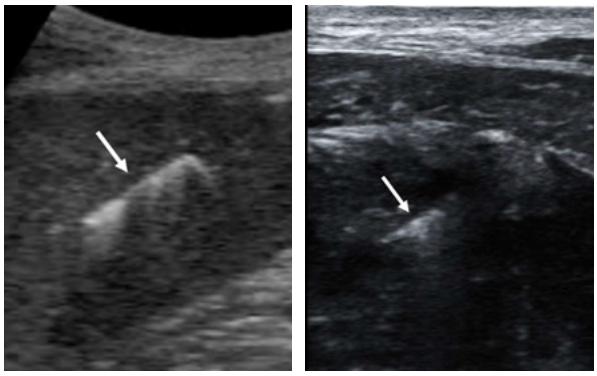


Figure 14.2. Linear echogenic structure (arrow) resulting from insertion of a needle into the tissue. (L) Convex probe, biopsy of the spleen. (R) Linear probe, biopsy of a superficial lymph node.

3) A transducer with a **needle guide** is used to determine the path of the needle.

Needle types

Which type of needle is used to sample tissue or fluid depends on the pathology. Needles (Figure 14.3) are generally categorised as **aspiration needles,** which are used for microbiology and cytological sampling, and **cutting needles or core biopsy needles,** which are used for tissue acquisition. Needles are distinguished

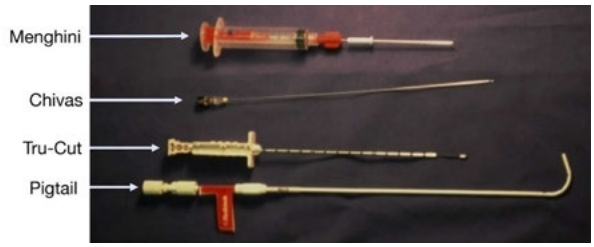


Figure 14.3. Needles used in interventional ultrasound.

according to size (diameter): **fine needles are < 1 mm** (19G and above); **coarse needles are > 1 mm** (18G and below). For tissue sampling, we typically use 18G or 16G needles.

Aspiration needles have an inner removable stylet. Examples include the **spinal needle** and the longer **Chivas needle.** A normal injection needle is often used if the target is not very deep. Cellular material is collected using aspiration after the needle is connected to a 10 ml syringe.

Cutting needles, even those of a calibre < 1 mm, have a **blunter tip** than aspiration needles. Therefore, **local anaesthetic** (for example, 2–3 mL lidocaine) is placed subcutaneously into the muscle layer after the skin has been disinfected. This is particularly necessary when larger calibre needles (18G or 16G) are used. The skin is then **pricked with a small lancet** to facilitate the entrance of the needle. The biopsy technique used is based on the type of needle.

The two main types of core needles are the **Menghini (end-cutting)** and the **Tru-Cut (side-cutting).**

The **Menghini needle** uses an inner device in the needle to avoid aspirating the tissue core into the syringe when suction is applied. The Menghini needle is introduced close to the superior margin of the target before applying suction to the syringe. The needle is rapidly advanced 2–3 cm and then retracted.

The **Tru-Cut needle** (Figure 14.4) has both an outer cutting cannula and an inner cannula with a notch (where the biopsy specimen is trapped) located close to the tip. The Tru-Cut needle is also advanced to the proximal border of the target. The internal cannula is then advanced into the target, and the tissue trapped in the notch. Finally, the outer sheath cuts the sample, which is then placed into a specimen container (Figure 14.5).

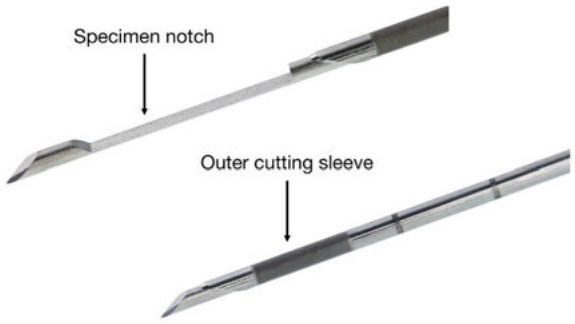


Figure 14.4. Tru-Cut needle: (Top) Step 1. The inner stylet is advanced. The groove (specimen notch) where the tissue sample will be collected can be seen in this view. (Bottom) Step 2. The outer sheath (cutting sleeve) is advanced; the excised sample is contained inside.

Figure 14.5. Cellular tissue cylinders collected from a tumour, to be sent for histology.

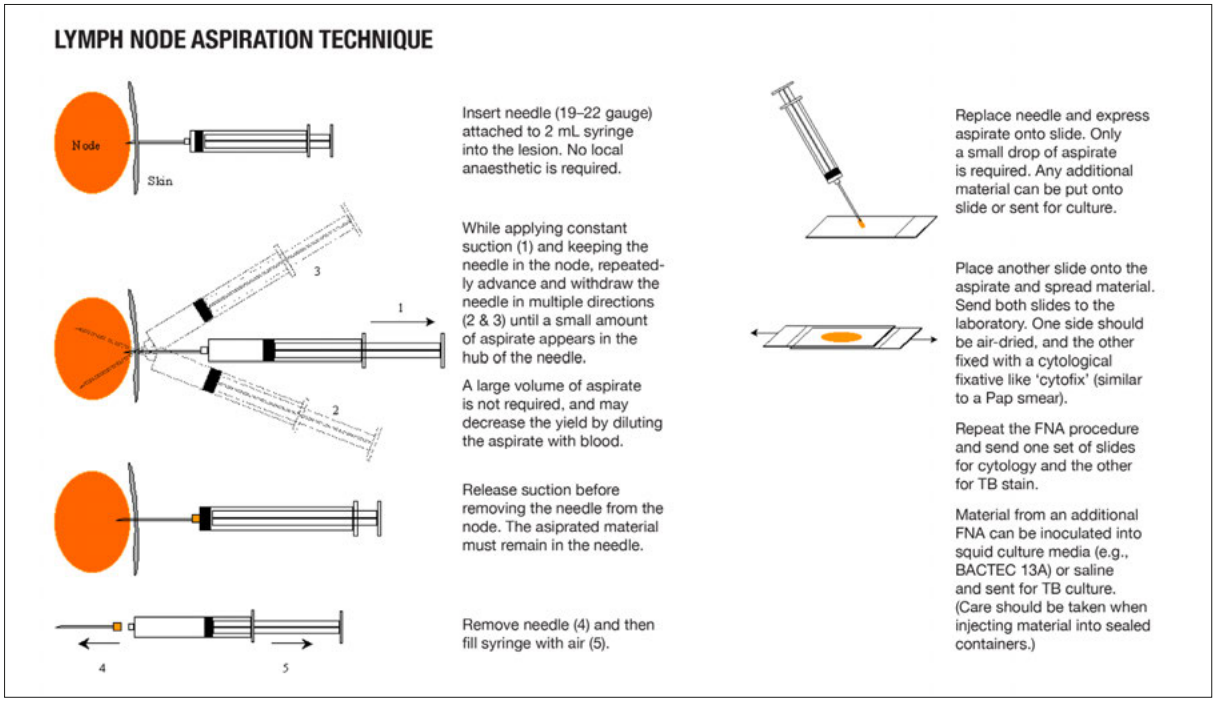


Figure 14.6. Lymph node aspiration technique.

Aspiration and biopsy of lymph nodes

Fine-needle aspiration of lymph nodes is a safe first step in the diagnostic workup of lymphadenopathy, as it yields a diagnosis in many patients. (The fine-needle LN aspiration technique is shown in Figure 14.6.) Though concern has been raised that sinus tracts can form along the needle path, recent experience with antibiotic therapy indicates that this is not the case (sinus tracts tend not to form in patients who are on anti-TB antibiotics).

The **aspirate can also be used for GeneXpert testing** (Figure 14.7). If the result does not suggest a diagnosis and the LN swelling is not responsive to TB treatment, a **core-needle biopsy** should be done. To investigate lymphoma and other malignancies, remember: 'tissue is the issue'.

Figure 14.7. Material obtained through repetitive puncture of a lymph node can be flushed in 1 cc of normal saline for GeneXpert MTB/RIF processing.

Aspiration of soft tissue and abscesses

Catheters (tubes) come in a variety of lengths and sizes. Most have a **terminal end configured in a 'pigtail'** (J-shape) to prevent accidental withdrawal. A **catheter mounted on a trocar** can be directly inserted into the collection site. Target depth should be accurately measured, and the track of the trocar carefully followed on the ultrasound screen. When the catheter is in position, the trocar is removed, and the drain stays in place.

With the **Seldinger technique,** a needle is inserted into the collection site and **a guide wire inserted through the needle.** The needle is then withdrawn, and the catheter tube is **directed over the guide wire** into place at the site. Placement of the wire is confirmed using ultrasound guidance. The trocar technique has the advantage of being relatively simple; however, the Seldinger technique enables more accurate placement of the catheter.

Placing a catheter is an elegant procedure, but care must be taken during dressing, as the catheter can become kinked. To avoid obstruction of the catheter requires intermittent saline instillation and aspiration, which is often not practical. **An alternative is to aspirate as much material as possible during the procedure** (Figure 14.8) **and withdraw the needle afterwards.** (Administering an effective antibiotic may often be enough to resolve the infection.) Even if aspiration has to be repeated once or twice in subsequent days, in many settings it may be an easier solution than catheter drainage.

Figure 14.8. Aspirated pus from the abscess should be sent for bacterial culture and GeneXpert MTB/RIF testing.

Venous cannulation and central lines

Central venous catheterisation is a common intervention in intensive and **critical care settings.** Ultrasound guidance can **shorten the duration of the procedure and reduce the number of attempts** needed to successfully catheterise the central vein; it is particularly useful for cannulation of the **internal jugular vein.** The addition of ultrasound guidance greatly **reduces the incidence of arterial puncture** (carotid artery) and subsequent formation of haematoma. Ultrasound allows for visualisation of anatomical variation prior to intervention, continual visualisation of the needle during placement, or both. The value of using ultrasound during the procedure is indisputable; all studies assessing the difference between ultrasound and landmark-based methods showed preferable outcomes with ultrasound. This has made **ultrasound guidance standard practice for central line placement.** In special cases, when no peripheral vein can be found, ultrasound using a superficial linear probe may also help to guide cannulation of the cubital veins.

Appendix 1. Diagnostic Tests and How to Interpret Them: Some Test Theory for the Curious

A physical sign, finding, or test result that is characteristic of a suspected diagnosis can be either present or absent. If it is present (a positive finding), the diagnosis becomes more likely; if absent (a negative finding), the diagnosis becomes less likely. How much these positive and negative results change the probability of a diagnosis varies for each sign, finding, or test result.

Some tests have very high **diagnostic accuracy:** when the results are positive, the disease is very, very likely to be present. (When they are negative, the disease is likely to be absent.) The best example in our setting is HIV tests, which are highly accurate.

Ultrasound findings typically have lower diagnostic accuracy, and are not as easy to interpret—which means they may not provide a definitive diagnosis. Even so, some highly specific findings, when positive, significantly improve the probability of a diagnosis. However, ultrasound's lack of sensitivity means the probability of that diagnosis changes very little when a finding is absent.

Other signs are more useful when they are absent, because the negative finding excludes the disease on a practical level. The trade-off here is that a positive finding does not affect probability very much.

To understand how positive or negative findings change the probability of a diagnosis, it is important to review and understand some basic diagnostic concepts: pre-test probability, sensitivity and specificity, and positive and negative predictive values.

Pre-test probability

Pre-test probability is the probability of disease prior to interpretation of the results of a test or a finding. It is also called the prevalence. This is the starting point for all clinical decisions. Published estimates of disease prevalence help us 'guesstimate' pre-test probability; however, they are often not available for every condition discussed in this book. Even when figures for national or regional prevalence are available, clinicians must adjust these estimates using information from their own practice.

Let's look at an example. Studies based in an emergency department in Khayelitsha (near Cape Town in South Africa) have shown that 13% of all patients presenting have TB. This means that the pre-test probability that any patient coming to that emergency department will have TB is 13%. On the other hand, the probability of TB is certainly lower in patients presenting at an emergency department in Italy; because the general prevalence there is lower, the pre-test probability would be lower. Meanwhile, the pre-test probability of TB in a patient coming to the South African emergency department may be even higher than 13% if the patient has HIV.

In fact, estimates of pre-test probability must always incorporate information from our own practice—where we are located and how underlying diseases, risks, and exposures make specific diseases more or less likely. In practice, pre-test probability is more often a feeling than an exact number.

Sensitivity and specificity

Sensitivity and specificity describe the ability of signs, findings, or tests to differentiate between the presence and absence of disease.

Sensitivity measures how often a finding is present, or a test comes back positive, in people who we already know have the disease.

Specificity is the opposite: it tells us how often the finding is absent in people who we know do NOT have the disease.

Clearly, for these definitions of sensitivity and specificity to be useful, we need some kind of **gold standard** (the truth) for determining who really has the disease we are testing for.

Sensitivity and specificity are easily calculated by constructing a **2 × 2 table,** which (as its name suggests) has two columns (one each for presence and absence of the disease) and two rows (one each for presence and absence of the finding). These rows and columns create four quadrants, typically referred to as cells A, B, C, and D:

- Cell A represents the **true positives**—disease and finding both present.
- Cell B represents the **false positives**—finding present, disease absent).
- Cell C represents the **false negatives**—finding absent, disease present).
- Cell D represents the **true negatives**—finding and disease both absent.

Let's look at a hypothetical example: A clinician working in an HIV/TB clinic knows that sonographic splenic abscesses are a sign of disseminated TB, and wonders how accurate this finding is—that is, how often the presence of a splenic abscess on ultrasound indicates a true diagnosis of TB, and how often the absence of a splenic abscess on ultrasound indicates a true absence of TB.

The clinician does a study of 100 patients presenting with suspected TB; the findings are presented in a 2 x 2 table:

		TB		
		Present	Absent	
Spleen abscesses	Present	12	2	14
	Absent	23	63	86
		35	65	

In this study, 14 patients have splenic abscesses (the sum of row 1); 86 patients do not (the sum of row 2). The sensitivity of the splenic abscesses is the proportion of patients with TB (35 patients) who have splenic abscesses (the positive finding, 12 patients); this is 12/35 = 0.34, or 34%. The specificity of splenic abscesses is the proportion of patients without TB (65 patients) who have no splenic abscesses (the negative result, 63 patients); this is 63/65 = 0.96, or 96%.

(As the table also shows, 35 of the 100 patients had TB, meaning the prevalence, or pre-test probability, was 35/100 = 0.35, or 35%—which, of course, is very high!)

When sensitivity is low, relying only on the finding will lead to many missed diagnoses. When specificity is low, over-reliance on the findings will lead to many incorrect diagnoses. In short, **poor sensitivity yields many false negatives; poor specificity yields many false positives.** We will come back to this later.

Positive and negative predictive value

These are very important concepts for the clinician, as we use the findings and tests to make medical decisions.

The **positive predictive value (PPV)** is the proportion of all patients testing positive who really have the disease. The PPV depends on the pre-test probability and the specificity (and a little on the sensitivity).

The **negative predictive value (NPV)** is the proportion of all patients testing negative who really do NOT have the disease. The NPV depends on the pre-test probability and the sensitivity (and a little on the specificity).

In our hypothetical study, the PPV and the NPV can be calculated from the 2 x 2 table. The PPV is 12/14 = 0.86, or 86%; the NPV is 63/86 = 0.73, or 73%. This means that there is an 86% probability that someone in the hypothetical population with splenic abscesses on ultrasound has disseminated TB. If that same person does NOT have spleen abscesses, there is a 73% chance the there is no TB. This leaves a 27% probability TB is present despite a negative test result.

What is the diagnostic accuracy of FASH findings, and how does this affect our interpretation?

Some studies have specifically looked at the diagnostic accuracy of FASH findings (lymph nodes, spleen abscesses, pericardial effusion, pleural effusion, and ascites) for TB. The findings can be briefly summarised as follows:

1) The sensitivity of all FASH findings is usually low.
2) The specificity of some findings (enlarged LN, spleen abscesses, and pericardial effusion) is high.
3) The specificity of other findings (pleural effusion, ascites) is not so high.

How can we explain these findings?

1) The majority of patients who have TB do not show abdominal changes; this includes all of those with pulmonary TB. Therefore, the overall sensitivity of FASH is not very good (only 63% in a meta-analysis).

2) If we look at **splenic abscesses**, sensitivity is low, at 39%. The explanation is similar to that mentioned above, but the specificity of spleen abscesses is 91%. If we look at **abdominal LN**, the test characteristics are similar: sensitivity is low, at just 43%, and varies widely, but specificity is a much better 87%. For **pericardial effusion,** a study in Cape Town found a sensitivity of 37% and a specificity of 83%.

3) **Pleural effusion and ascites** can signal the presence of TB, but they are found in many other diseases as well. For pleural effusion, we have to consider parapneumonic effusions, KS, and cardiac failure. For ascites, we need to also consider cirrhosis, malignancy, hepatitis, and cardiac failure. The presence of other causes significantly reduces the specificity of these findings.

How do we use FASH findings in light of this information?

We know now that FASH is **not useful as a screening test** for TB, because both its sensitivity and NPV are low. Many patients have disease in the lungs or elsewhere that cannot be found with the FASH scan.

FASH does work well with patients for whom the pre-test probability of TB is high—for example, those who are sick and immunocompromised. With these patients, FASH findings can be used to **confirm the TB diagnosis** if we find enlarged LN, splenic abscesses, or pericardial effusion. Because the specificity of these findings is high, they have a useful PPV that can help us secure the TB diagnosis.

If we do not find any of these, we still **CANNOT rule out TB, because many patients with TB will not have these findings present.** In these cases, we must use other tests if our clinical suspicion remains high. This is similar to how we use the **urine LAM test:** it can confirm a diagnosis, but cannot rule out a diagnosis (its sensitivity in numerous studies is around 28–69%; its specificity is 78–99%). The characteristics of the urine LAM test are comparable to the numbers for FASH findings.

For **pleural effusion and ascites,** the picture is a bit more complicated—we need to **check whether any of the other possible diagnoses** mentioned are present. If present, then the effusion can be explained by something other than TB. If none of the other diseases is present, then TB should be considered as a possible explanation, and we should consider starting the patient on presumptive TB treatment. In the end, the FASH scan needs to be regarded as one clinical piece in the bigger diagnostic puzzle of HIV-associated TB.

Appendix 2. Subjectivity and Point-of-Care Ultrasound

Tom Heller, Elisabeth Joekes

'medicine is magic, and magic is art'
—Paul Simon

Introduction: ultrasound and subjectivity

Ultrasound is one of the most widely used medical imaging techniques of our time. The positive aspects of ultrasound, which are often stressed, are that it is non-invasive, radiation-free, affordable, fast, and can be performed at the bedside as often as is needed.[1]

However, also frequently underlined are the risks and limitations of a lack of standardisation and a dependence on operator performance. A WHO paper published in 1998, long before the explosive growth in deployment of point-of-care ultrasound, pointed out that 'the skill and training of the user are often more important than the equipment used', and that 'patients may be harmed by misdiagnosis resulting from improper indications for use, poor examination technique, and errors in interpretation.'[2]

After years of experience teaching ultrasound to a wide variety of clinicians in various economic and geographic settings, we cannot help but agree on the importance of high quality, appropriate training, and operator skill. Nevertheless, in our opinion the often-negative connotation of 'subjectivity' linked to non-expert operators (that is, those not formally trained in sonography or radiology) may hinder the wider spread of a highly useful technique—to the detriment of patient care, especially in health care systems with limited resources. In this brief essay, the technique of medical ultrasound will be examined by following post-structralist Michel Foucault's philosophical analysis of the medical gaze, with the intention of highlighting the actual benefits of this 'subjectivity'.

The medical gaze and the advent of pathological anatomy

By the end of the 18th Century and the beginning of the 19th, after many centuries during which the pathophysiological theory of the humours prevailed, the location of disease was increasingly sought in the solid organs. Anatomy thus began to dominate the discourse of medical theory:

> *The anatomo-pathologists discovered in theirs a non-philosophy, an abolished philosophy, that they had conquered in learning at last to perceive: it was simply a question of a shift in the ontological foundation on which their perception was based.*[3(p.155)]

Previously, for the medical gaze, 'causes and locales did not interest…: it was interested in history, not geography.'[3(p.126)] Now, during increasingly frequent autopsies, organic lesions, tubercles, and tumours were described and their causality for diseases postulated:

> *The anatomo-clinician's gaze has to map a volume; it deals with the complexity of spatial data which for the first time in medicine are three-dimensional.*[3(p.163)]

The theoretical knowledge gained during autopsies was also translated into clinical medicine, and by various techniques physicians attempted to bring the changes from within the darkness of the body to light. Not just post-mortem, but also during life. Percussion (established by Auenbrugger) and auscultation (which Laennec refined around the same time) interrogate the depth and the volume of the body; these techniques only made sense in light of this new mode of understanding.

Thus 'the access of the medical gaze into the sick body was not the continuation of a movement of approach that had been developing in a more or less regular fashion since the day when the first doctor cast his somewhat unskilled gaze from afar on the body of the first patient; it was the result of a recasting at the level of epistemic knowledge itself.'[3(p.137)]

The image of the bones of Roentgen's hand at the end of the 19th century and the medical imaging revolution following it are based ultimately in the prior change of mode to 'see' medically. 'Since 1816, the doctor's eye has been able to confront a sick organism. The historical and concrete a priori of the modern medical gaze was finally constituted.'[3(p.192)]

The intentional gaze

When we look at an image, whether radiological or an image in general, we assume that the image has unambiguous relations to the reality that is depicted. Through this principle of reference, we generate an idea of the object, and thus an idea of the human being from which this images arise. We can work with this idea, but it nevertheless will almost always stand in only vague relation to the real object itself.

Diagnostic imaging can be separated into two phases: acquiring the image and interpreting the image. The process of obtaining a sonographic image is certainly more complicated than other medical imaging processes: the degrees of freedom; the turning, tilting, and positioning of the transducer; and the manipulation of technical parameters to enhance the image are all highly variable. Given this, we can see that other imaging methods are also subject to variability during this phase: the information in computerised tomography (CT) images depends on patient variables and the use of contrast media to enhance the view of tissues. The large number of possible sequences in magnetic resonance imaging (MRI) proves that this image also depends on the operator setting the correct parameters.

Once obtained, the X-ray or MRI image is fixed, either as a constant image on film or as a reproducible, unchangeable computer file—making it putatively more 'objective'. True diagnostic information is only gained by interpreting the image. Here the physician's gaze is directed at the image, extracting potential information and applying it to answer a clinical question. Any previous objectivity becomes relative.

As Husserl puts it, 'in every act [of perception] an "imagined" object is defined as "so and so", and as such it is the aim of changing intentions.'[4(p.57)] The intentionality of consciousness is always 'directed at' something. Thus, the information obtained always depends on the person who considers the image, and on what he or she can see and wants to see.

Like the medical gaze itself, the gaze directed at the image depends on the current pathophysiological discourse medical imaging is based on.

The only normative observer is the totality of observers: the errors produced by their individual points of view are distributed in a totality that possesses its own powers of indication. Their very divergences reveal, in this nucleus in which, after all, they intersect, the outline of undeniable identities: 'Several observers never see the same fact in an identical way, unless nature has really presented it to them in the same way.'[3(p.102)]

Although three, five, or seven radiologists can look at the same image and by consent or majority decide on the 'true diagnosis', in many cases it will still not be identical to the true diagnosis for that patient.

In reality, these 'objectively acquired' images, including those from standardised diagnostic ultrasound tests performed by trained sonographers or radiologists, will frequently be interpreted by a single individual, often one who does not have all the clinical information—or even much relevant clinical knowledge. The verbalised quintessence of the image, in the form of a descriptive report, will in turn need to be interpreted by the treating clinician.

Consequently, this information cascade seems no more objective than the information obtained directly from point-of-care ultrasound images by the treating clinician. In our interpretation of 'objective' tests, including formal ultrasound tests performed by trained technicians, the absence of the immediate medical gaze introduces a risk of error that might not otherwise arise in the point-of-care scenario.

The gaze beyond the gaze: see–hear–feel

The fact that ultrasound cannot be completely standardised can be lamented, as we need standards to communicate about results. Unfortunately, the ultrasound exam can never merge completely with any standard, because there is always a component of personal style involved in conducting the exam. During the exam the clinician palpates the patient with the transducer; the mode of palpating always remains, at least in part, individual and personal. We are questioning a living being and can immediately perceive their reactions during the exam. Thus, one learns more about the patient than the image on the screen alone will tell.

Thus armed, the medical gaze embraces more than is said by the word 'gaze' alone. It contains within a single structure different sensorial fields. The sight/touch/hearing trinity defines a perceptual configuration in which the inaccessible illness is tracked down by markers, gauged in depth, drawn to the surface, and projected virtually on the dispersed organs of the corpse.[3(p.164)]

Like 'the stethoscope […] transmits profound and invisible events along a semi-tactile, semi-auditory axis,'[3(p.164)] ultrasound transmits more than the visual information; a 'more' that the experienced clinician will include in his or her diagnostic thoughts, but which is difficult to standardise. 'Since everything, or nearly everything, in medicine is dependent on a glance or a happy instinct, certainties are to be found in the sensations of the artist himself rather than in the principles of the art.'[3(p.121)]

Ultrasound as dialogue

A further important aspect is communication—the dialogue between clinician and patient during the exam. While other technical examinations may forcibly interrupt that dialogue at least temporarily, the ultrasound exam requires a contact that facilitates conversation in the anonymous intimacy of a darkened room.

Although the technology is between the patient's body and the physician's hand, it allows a dialogue with the person being examined. It allows a conversation about the body that is not just the object of examination but is also part of the person being examined.[5]

This dialogue can increase the amount of information acquired; the patient can guide the transducer, and thereby the gaze of the clinician, to regions of pain or other importance. Without knowing what you want to see, or in which direction to look, you will see less. The dialogue can also convey something to the patient that is important in the relationship between clinician and patient. The exam, during which the physician touches the other human indirectly, approaching them carefully, '…can communicate closeness in the face of objectivity.'[5] 'Ultimately it must be recognised that ultrasound examination is not just a technical procedure but involves a certain artistry, a combination of standardisable skill and non-standardisable *plus*. This *plus* is the essence of ultrasound.'[5 (italics added)]

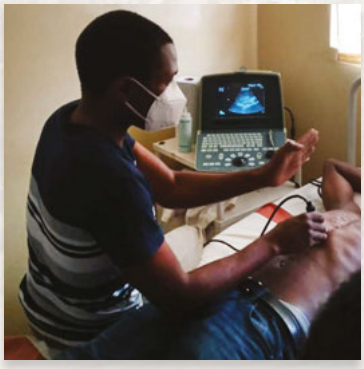
Conclusion

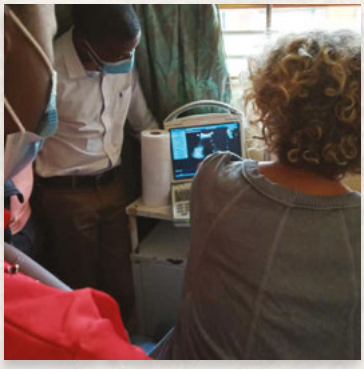
Point-of-care ultrasound, especially during image acquisition, may be more dependent on the knowledge and skill of the operator than other imaging methods. However, this does not necessarily lead to a fundamental difference in value, as all imaging methods contain an inherent element of subjectivity when it comes to interpretation. The claim that patients can be harmed by poor examination technique, incorrect indications, and errors in interpretation is no more true for ultrasound than it is for any other clinical technique. It merely emphasises the need for high-quality education and training. 'So it is not the gaze itself that has the power of analysis and synthesis, but the synthetic truth of language, which is added from the outside, as a reward for the vigilant gaze of the student.'[3(p.60)]

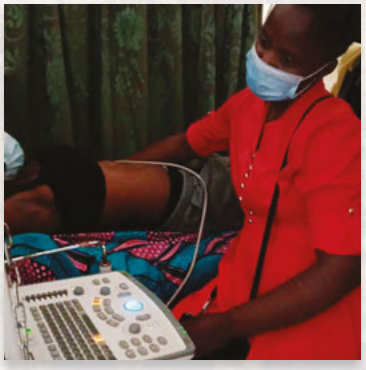
References

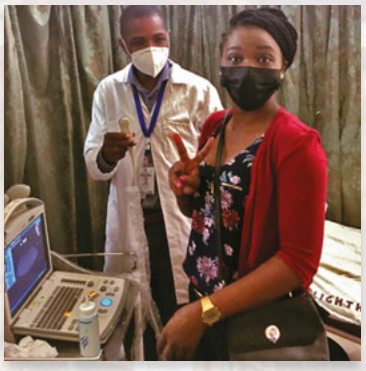
1. Brunetti E, et al. The current role of abdominal ultrasound in the clinical management of patients with AIDS. Ultraschall Med. 2006;27(1):20-33. doi:10.1055/s-2005-858649
2. World Health Organization. Training in diagnostic ultrasound: essentials, principles and standards. Report of a WHO study group. World Health Organ Tech Rep Ser. 1998;875:i-43;back cover. PMID: 9659004.
3. Foucault, Michel. The birth of the clinic: an archaeology of medical perception. Routledge; 1973.
4. Husserl, E. Logische Untersuchung (2. Aufl) [Logical investigations, 2nd edition]. Meiner Verlag; 1988.
5. Maio G. Medicine and the holistic understanding of the human being: ultrasound examination as dialog. Ultraschall Med. 2014 Apr;35(2):98-107. doi:10.1055/s-0034-1366182. Epub 2014 Apr 11.

Index









Since the mid-1990s, point-of-care limited ultrasound techniques have extended the utility of ultrasound beyond radiology departments. Initially, diagnostic ultrasound technology was predominantly used by radiologists and imaging specialists. Now, clinicians from diverse specialties are using ultrasonography to examine specific organs and disease processes, and to help perform relevant procedures.
— Tom Heller, MD

PEPFAR

I-TECH
International
Training & Education
Center for Health

LIGHTHOUSE
2022