Chapter 44
Acute Problems and Emergency Surgery: Surgery in the Tropics

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Abstract The authors of this chapter include specialists, many of whom have extensive deployment experience. The target group of this chapter, however, is not their fellow-specialists; but the "junior" doctors, trying to help them find their way in the difficulties posed by an "adverse" environment. That junior doctor will be confronted by all imaginable ailments and injuries, and should be a true generalist. As we're all aware, even in medical school nowadays there's a tendency to make students choose the direction of their future work at an ever earlier stage; the opposite of what's needed for a generalist.

Keywords Triage • Trauma and medical emergencies • Ballistic and blast injury • Infectious diseases • Climatic influences • Bites and stings • Maxillofacial problems • Head and spinal cord injuries • Abdominal complaints • Non-traumatic surgical emergencies • Soft tissues and skeleton • Surgery in the tropics • Anaesthesia and analgesia • Hostile environments • Disaster environments • Conflict environments • Catastrophe environments

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Objectives
To describe prevalent surgical conditions and procedures in the tropics due to the following conditions/diseases/injuries:

- AIDS
- Tuberculosis
- Malaria
- Tropical ulcer
- Buruli ulcer
- Ludwig's angina
- Typhoid fever
- Volvulus of the sigmoid colon
- Amoebiasis
- Schistosomiasis
- Hydatid disease
- Intestinal roundworms
- Hookworms and whipworms
- Filariasis
- Onchocerciasis
- Osteomyelitis
- Trauma
- Anaemia, malaria and HIV/AIDS during obstetric surgery
- Obstructed labour
- Vesicovaginal fistula

Introduction

It has been stated that the glory of ancient civilisations departed on account of disease and microbes than have changed the map of the Earth more often by wars. In China in the fourteenth century, 13 million died from the "Black Death". In Europe from 1347 to 1350, 25 million people died from the same disease, and in the eighteenth century, 60 million people died from smallpox in Europe alone. For centuries men have sought the causes of yellow fever, plague, malaria and other diseases, and some of the guesses were near the mark. Egyptians made the cat a sacred animal because where it flourished there was no plague [1]. Infectious diseases are still by far the most important agents of disease in the tropics today, and millions of children suffer and die from malaria, respiratory infections and diarrhoeal disease; those concomitantly infected with HIV have little chance of recovery.

This chapter is written based on my personal experience of tropical conditions encountered whilst working with Medecins Sans Frontieres in East, Central and West Africa. All my missions have been outside teaching or capital city hospitals, and the majority of the diagnoses have been made based on clinical features alone.

HIV/AIDS

The AIDS pandemic has radically changed the practice of surgery, and in many tropical countries many conditions are as a direct result of HIV infection. As a surgeon it is very important to take particular care and to double and triple glove and wear eye protection especially in dealing with bone fragments in fractures and ensure that you wear plastic aprons if theatre gowns are not waterproof.

HIV and AIDS more heavily affect sub-Saharan Africa than any other region of the world. By the end of 2009, there were an estimated 22.5 million people living with HIV in the region, around two-thirds of the global total. In that year around 1.3 million people died from AIDS, and 1.8 million people became infected with HIV [2]. HIV gradually destroys the human immune system, increasing vulnerability to other infections and cancers, such as pneumonia, Kaposi sarcoma and tuberculosis (TB).

The virus consists of two copies of the RNA genome and two copies of the enzyme reverse transcriptase contained in a viral core surrounded by an envelope composed of lipids. The viral envelope contains 20 glycoproteins, and most diagnostic tests detect the antibodies to these glycoproteins. HIV uses the CD4 receptor to attach to lymphocytes, and once inside the cell viral RNA is transcribed to host DNA using the enzyme reverse transcriptase, and it is this enzyme which is targeted by compounds, which inhibit HIV replication, the antiretroviral drugs. HIV causes disease in humans primarily by causing destruction of cells of the immune system, thus making the HIV-infected individual increasingly susceptible to other pathogens as the course of HIV disease progresses. CD4+ lymphocytes are the main target cells infected by HIV, and loss of CD4+ lymphocytes is a key feature of HIV-related immunosuppression. The function of the cellular immune system is best measured using the CD4+ lymphocyte count, which can also be used to both stage the disease and predict treatment response. Immunoassay tests for antibodies to HIV are highly sensitive and commercially available at US$1 per test. But still millions of people at risk in Africa are not tested or avoid testing, and of those tested positive only 37 % receive antiretroviral therapy [3].

The clinical diagnosis of HIV infection is difficult because most of the characteristic symptoms and signs of HIV infection are neither sensitive nor specific. A particular problem is that the clinical presentation of advanced HIV disease can be indistinguishable from tuberculosis in an HIV-negative individual. However, with clinical assessment, the significant stages of HIV disease can be recognised, and the patient's probable immune status assessed [4]:

Clinical stage 0	Serologically HIV positive and no signs or symptoms
Clinical stage 1	Generalised lymphadenopathy
Clinical stage 2	Weight loss, cutaneous lesions and recurrent upper respiratory tract infection
Clinical stage 3	Severe weight loss, diarrhoea, fever, oral candidiasis, pulmonary TB and severe bacterial infection
Clinical stage 4	Full-blown AIDS wasting, severe opportunistic infections, extrapulmonary TB, lymphoma, Kaposi sarcoma and encephalopathy

From a surgical point of view, patients with HIV tend to develop post-operative wound infections, and in fact one study showed that the rate of post-operative wound infections doubled in stage 0 and were treble in those in stages 1–4 [5].

Some time ago, I treated a patient in the Congo who I felt was clinical stage 3 with fever, weight loss and diarrhoea for several months who was admitted with signs very suggestive of a perforated duodenal ulcer. At laparotomy, there was large perforation in the terminal ileum, which was walled off with little abdominal contamination. I elected to perform a right hemicolectomy. Sadly the patient died of an overwhelming sepsis 7 days later. From a clinical point of view, I suspect the patient had a cytomegalovirus (CMV) perforation, as it is the most common cause of emergency abdominal surgery in AIDS patients. CMV causes a vasculitis in the arterioles of the GI tract, which results in focal or diffuse ulcerations, most commonly of the terminal ileum and colon where they can cause bleeding and spontaneous perforation [6].

The commonest cancer in HIV-infected people in Africa is Kaposi sarcoma, a tumour of the endothelial cells of the lymphatics. Surgery is occasionally indicated to remove single lesions; multiple lesions need systemic treatment such as vincristine and other cytotoxics. In the limbs multiple lesions cause oedema and may break through the skin and extend from the foot to the midthigh. The patient shown in Fig. 44.1 had experienced such severe pain that they requested an amputation.

Occasionally plaques of Kaposi occur in the small bowel and give rise to intussusception and bowel obstruction [7]. However, bowel obstruction in patients with

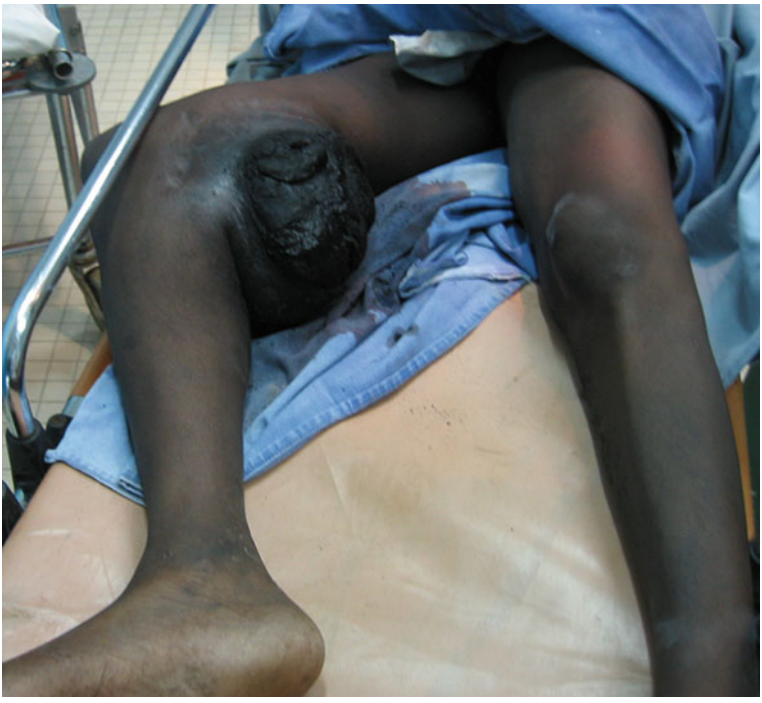

Fig. 44.1 Karposi sarcoma

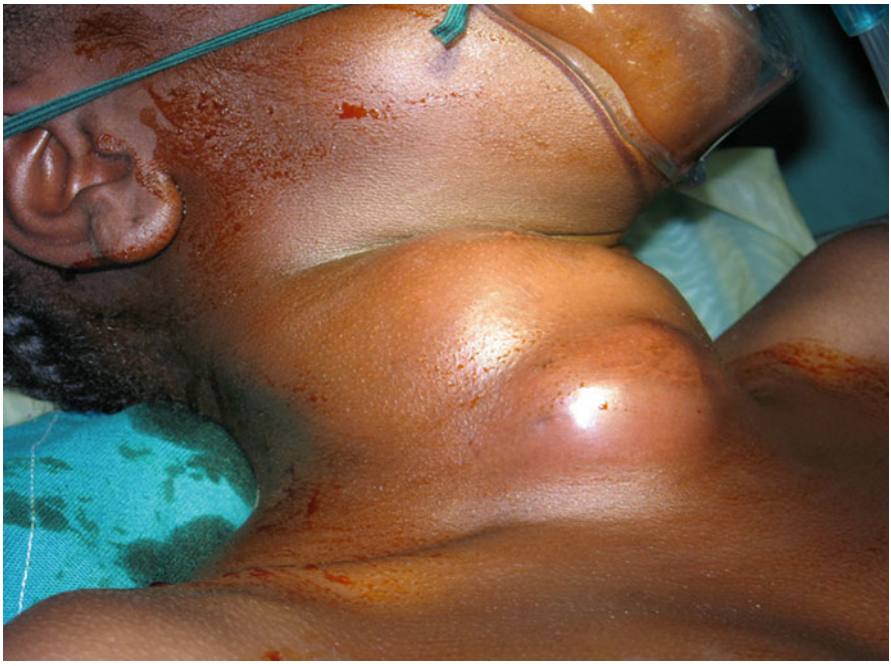

Fig. 44.2 Thyroid gland abscess

AIDS is commonly due to non-Hodgkin's lymphoma. Although peripheral lymphadenopathy is often absent, bulky retroperitoneal and mesenteric lymphadenopathy is common in AIDS patients and can cause abdominal pain, nausea and vomiting. In addition to obstruction, gastric haemorrhage and intestinal perforation with peritonitis have also been reported [8].

Surgical sepsis in HIV affects mainly the female genital tract; the pleural cavity, causing empyema; the large joints; and the anorectal area. A few patients have multiple abscesses or infection in unusual sites such as the thyroid gland (Fig. 44.2).

Patients with HIV are also at risk of developing necrotising fasciitis (Fig. 44.3), which is a rapidly progressive inflammation and necrosis of subcutaneous tissues and the deep layer of superficial fascia with sparing of the deep fascia and muscle. Aerobic pathogens are usually the primary tissue invaders, for instance, beta-haemolytic streptococci and staphylococci. They destroy tissues and create an anaerobic environment conducive for anaerobic or microaerophilic organisms, which are secondary invaders such as gram-negative organisms such as *Bacteroides fragilis*, peptostreptococci, *Proteus* sp., *Pseudomonas* sp. or *Enterobacter* spp. [9]. The surgical treatment in all cases involves fasciotomy and wide debridement of all dead tissue supplemented by combinations of penicillins, aminoglycosides and metronidazole [10, 11].

In patients with HIV, necrotising fasciitis can complicate such surgical procedures as colostomy, appendicectomy, herniotomy, laparotomy or dental extraction; or it can follow infections such as chickenpox, gingivitis, boil or perineal abscess [12].

Today, the orofacial form of necrotising fasciitis is called cancrum oris (noma), and the perineal form is called Fournier's gangrene [13]. Noma (Fig. 44.4) is typically found in the tropical sub-Sahara and affects children under the age of 6 and is the result of a combination of factors. It begins as a polymicrobial infection caused by anaerobic organisms that produce enzymes, as well as pro-inflammatory mediators, that degrade the intracellular matrix. Acute necrotising gingivitis is a risk factor for developing the disease. It may start as a simple ulcer and, without nutritional support and antibiotics, can rapidly progress. Children often have systemic comorbidities, such as leukaemia, HIV/AIDS, diarrhoea, malaria or a recent case of

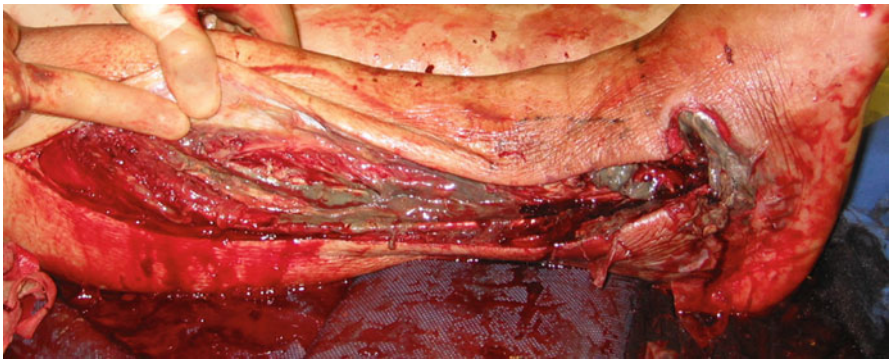

Fig. 44.3 Necrotising fasciitis

Fig. 44.4 Child with noma

measles. Every year, WHO estimates more than 100,000 cases of noma occur worldwide, and only 10 % survive. The lack of an adequate diet lowers the immune system's defences in children, and these illnesses only aggravate the situation. Without appropriate treatment the child will perish from complication such as pneumonia, septicaemia or diarrhoea. Those who survive usually suffer from severe facial disfigurement and/or trismus and will need difficult staged surgical reconstructions. Initial treatment is with penicillin or broad-spectrum antibiotics together with rehydration correction of electrolyte imbalances and supplemental nutrition as soon as erythema or oedema develops [14].

Pyomyositis (myositis tropicans) is a disease strongly associated with HIV infection in young adults (Fig. 44.5). Usually skeletal muscles are immune from infections; however, this condition causes a severe infection of the skeletal muscle usually the powerful muscles of the trunk and extremity. Intramuscular abscesses occur without any penetrating trauma or adjacent septic focus. *Staphylococcus aureus* is still the usual organism although a variety of other organisms have been found in HIV-positive patients [15]. The treatment invariably requires drainage of the abscess and combination treatment using penicillins, metronidazole and gentamicin.

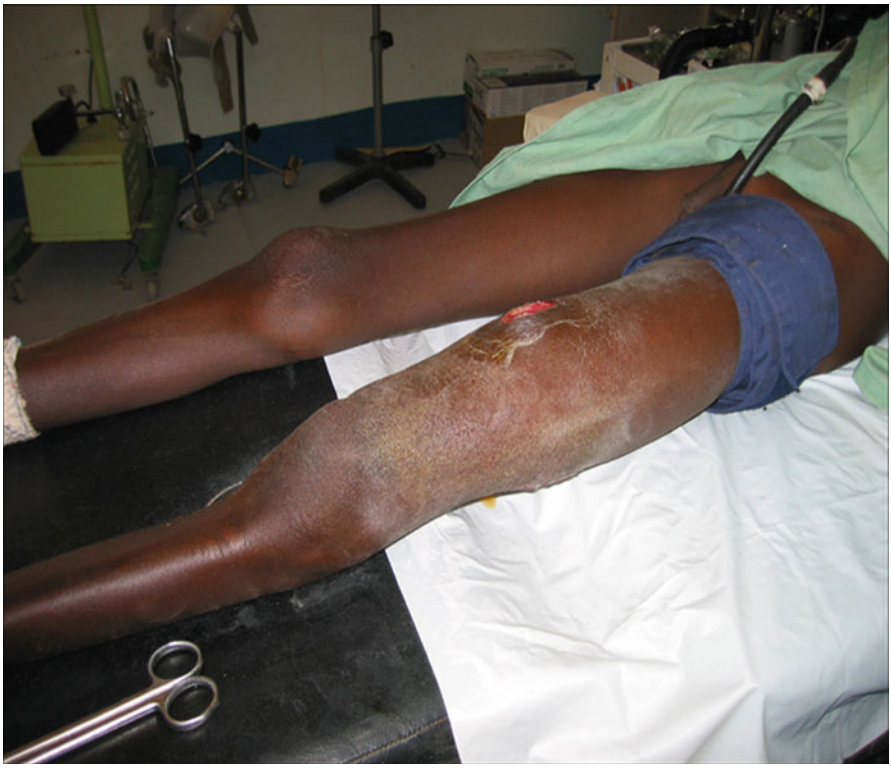

Fig. 44.5 Polymyositis involving the quadriceps muscle

Tuberculosis

One-third of the world's population is thought to be infected with *M. tuberculosis* [16]. A person infected with both TB and HIV is 30–50 times more likely to develop the active form of TB than someone infected with TB alone [17]. In some countries such as Malawi, Zambia and Botswana, 70 % of tuberculosis patients are HIV positive [18]. When tuberculosis becomes active, 75 % of cases involve infection in the lungs (pulmonary TB), and the other 25 % affect extrapulmonary sites. Pulmonary symptoms include chest pain, haemoptysis and a productive, prolonged cough for more than 3 weeks. Systemic symptoms include fever, chills, night sweats, appetite loss, weight loss, pallor and fatigue. Extrapulmonary infection causes tuberculous pleurisy, tuberculous meningitis, scrofula of the neck (cervical tuberculous lymphadenitis), urogenital tuberculosis and infection of the bones, joints and spine (Potts disease), and tuberculosis of the breast can mimic breast cancer [19].

Abdominal Tuberculosis

Tuberculosis can involve any part of the gastrointestinal tract and is the sixth most frequent site of extrapulmonary involvement. Tuberculous bacteria reach the gastrointestinal tract via haematogenous spread, ingestion of infected sputum or direct spread from infected contiguous lymph nodes and fallopian tubes. The gross pathology is characterised by transverse ulcers, fibrosis, thickening and stricturing of the bowel wall, enlarged and matted mesenteric lymph nodes, omental thickening and peritoneal tubercles.

Peritoneal tuberculosis occurs in three forms, the wet type with ascites, the dry type with adhesions and the fibrotic type with omental thickening and loculated ascites. The most common site of involvement of the gastrointestinal tuberculosis is the ileocaecal region (Fig. 44.6). Like Crohn's disease any part of the gastrointestinal tract can be affected, and pathologically it may be either an ulcerative form with caseation, perforation and internal fistulae or the hypertrophic form with fibroblastic reaction leading to intestinal stricture and obstruction. Ileocaecal and small bowel tuberculosis presents with a palpable mass in the right lower quadrant and/or complications of obstruction, but also examination of the abdomen may reveal a vague lump, which may be rolled-up omentum, enlarged lymph nodes or matted loops of bowel with omentum. In Africa therefore be aware that a lump in the right iliac fossa may well be due to abdominal tuberculosis than acute appendicitis. Always be aware that TB can mimic lots of western diseases and can include dysphagia, odynophagia and a mid-oesophageal ulcer due to oesophageal tuberculosis and dyspepsia and gastric outlet obstruction due to gastroduodenal tuberculosis. In the majority, abdominal TB presents as abdominal pain, and ascites is the most frequent physical finding followed by hepatomegaly, splenomegaly, abdominal

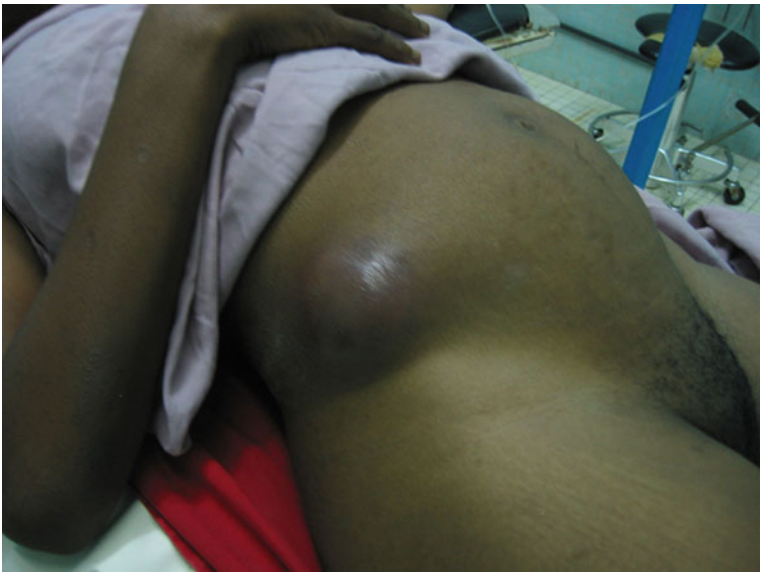

Fig. 44.6 Tuberculous abscess in the right iliac fossa

mass, fistula and acute abdomen if perforation of the bowel has occurred. Most of these patients are significantly anaemic [20].

Management is with conventional anti-tuberculous therapy for at least 6 months. In the tropics keep tuberculosis at the forefront of the differential diagnoses because to operate is fraught with complications and is only reserved for life-saving instances such as perforation [21, 22].

Malaria

Unexplained persistent pyrexia and general deterioration are presenting features following a surgical procedure in patients infected by malaria. Indeed malaria is a common cause of hyperpyrexia, unexplained post-operative confusion progressing to coma, shock and acute renal failure (blackwater fever). Contaminated fresh blood transfusion may cause profound coma and severe shock with hyperpyrexia if infected with malaria. For planned surgery, especially in children, a haemoglobin check and a paracheck should always be performed before surgery [23]. I have seen a young boy die post-operatively having been taken to surgery for appendicectomy when a simple 5-min paracheck would have made the diagnosis of malaria.

The spleen is a major site of antigen presentation and acts as a reservoir. Splenic macrophages remove damaged or old red cells. The spleen enlarges as a result of this haematological overactivity or the antigenic process. The classic causes of

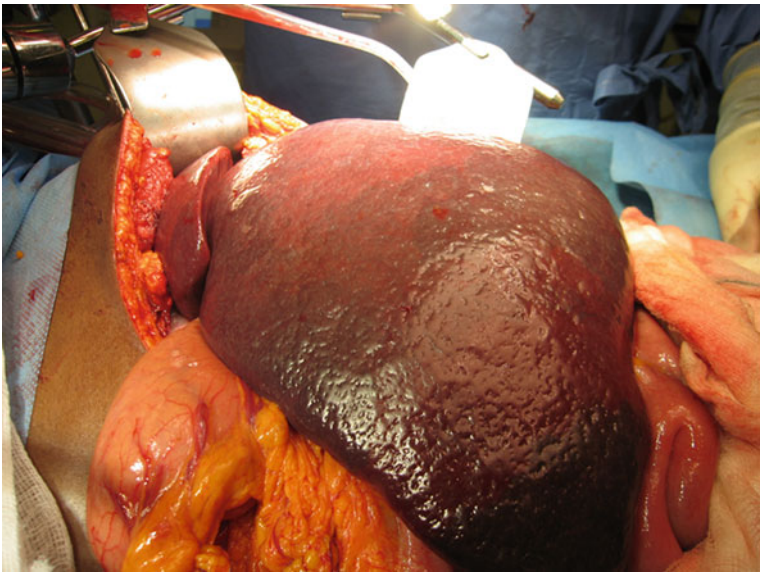

Fig. 44.7 Massive spleen due to malaria

massive tropical splenomegaly are hyperreactive malarial splenomegaly (HMS), schistosomiasis (see discussion to come) and visceral leishmaniasis. Almost all patients with massive spleens have blood pooling and are therefore anaemic which may contribute to the high mortality associated with HMS [24].

Those in whom malaria parasites are found should have a course of antimalarial treatment using chloroquine, and surgery if required should be delayed several days. However, this may not be possible in trauma. The spleen in HMS is enormous (Fig. 44.7) and if ruptured is commonly fatal. Apart from the initial anaemia procedure, the surrounding perisplenitis makes it adhere to the diaphragm and stomach, and it is a relatively anchored organ. Following removal of the spleen, a large bed is left and the surgeon has to be extremely vigilant with haemostasis in this situation. In an emergency situation such as removal of a malarial spleen in cases of trauma, antimalarial drugs should be taken soon after the procedure.

It is unlikely that a splenectomy would be performed for anything other than trauma outside a major teaching establishment in Africa. It is very important to recognise the causes of massive spleens if encountered during a laparotomy and the potential morbidity and mortality when having to perform a splenectomy for trauma.

Tropical Ulcer

Skin infection and breakdown is common in tropical ulcer (Fig. 44.8). This is because bactericidal and fungicidal compounds containing fatty acids produced by sebaceous glands in the skin are defective due to malnourishment, and the skin may

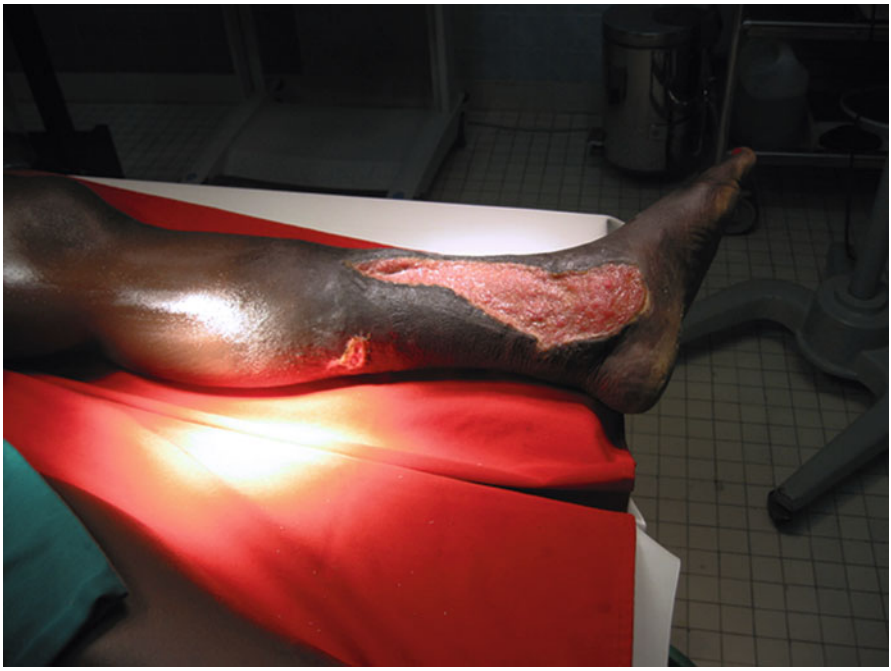

Fig. 44.8 Tropical ulcer

be thin. Skin infections are much more common in the wet, humid times of the year. Bacteria and fungi are able to multiply easily at these times, possibly because bactericidal secretions are diluted by sweat. Many patients will be seen with superficial skin ulceration secondary to insect bites and scratches become infected with *Streptococcus pyogenes* or *Staphylococcus aureus* together with *Treponema vincentii*. These are usually simple to treat with antibiotics but become more difficult as these tropical ulcers normally have a necrotic base and are colonised by anaerobic *Fusobacteria*.

Buruli Ulcer

Buruli ulcer (Fig. 44.9) is caused by *Mycobacterium ulcerans* and is endemic in Ghana and estimated to affect 150–180 per 100,000 population and often leaves children with large disfiguring scars and occasional joint deformities with massive loss of skin if not treated. Bacteria enter the skin from scratches from vegetation in swamps. The mycobacteria causes disease by producing a potent tissue-destructive toxin called mycolactone [25]. Treatment involves removing infected tissue and skin grafting, but recently more effective medical treatment using rifampicin and streptomycin for 12 weeks proves to be a better alternative to radicle debridement [26].

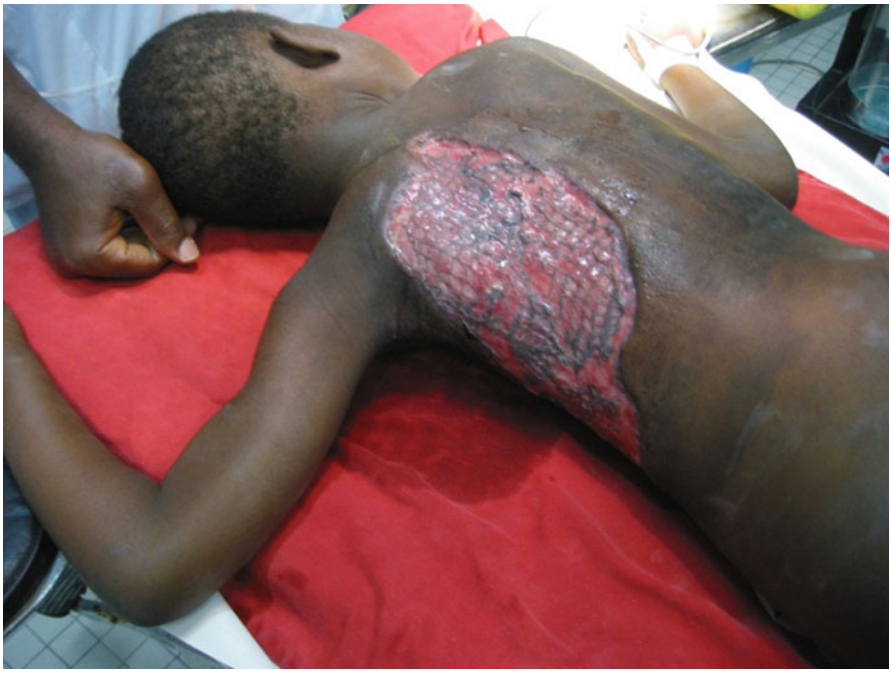

Fig. 44.9 Buruli ulcer

Ludwig's Angina

The cause is usually an infection from the lower molars or the gums surrounding the third molar tooth. It can occur in healthy individuals but more often in immune-compromised patients. Symptoms include swelling, pain, fever, dysphagia and in severe cases stridor and difficulty in breathing due to the pressure pushing up the tongue against the soft palate. The word angina comes from the Greek meaning strangling. The patient shown in Figs. 44.10, 44.11 and 44.12 had developed sudden airway obstruction and was becoming hypoxic. A decision was made to thrust a knife and scissors into the floor of the mouth to evacuate the abscess. Following that intervention the patient was able to breathe almost immediately. The alternative was to perform an urgent tracheostomy, but this can be difficult if the tissues of the neck of firm and oedematous.

Tropical Conditions Affecting the Abdomen

Typhoid Fever

Typhoid fever is caused by *Salmonella typhi* that includes bacteraemic phase with fever and chills during the first week, sustained high temperature, a rose spot rash and abdominal pain during the second week and ulceration of the Peyer's patches of the

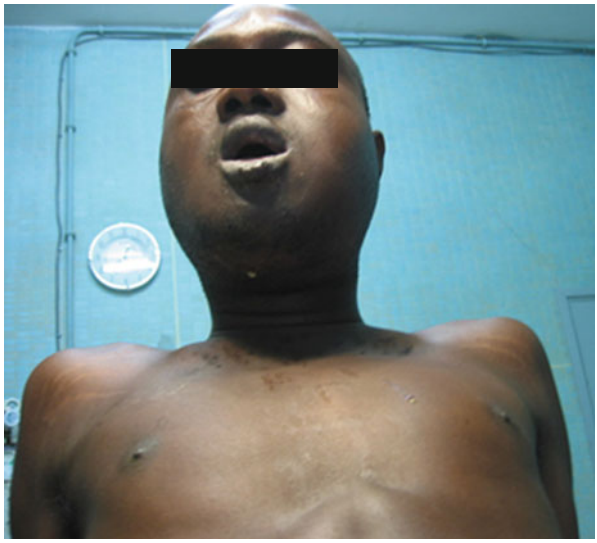

Fig. 44.10 Acute dyspnea due to Ludwig's angina (note the drop of pus below the mouth)

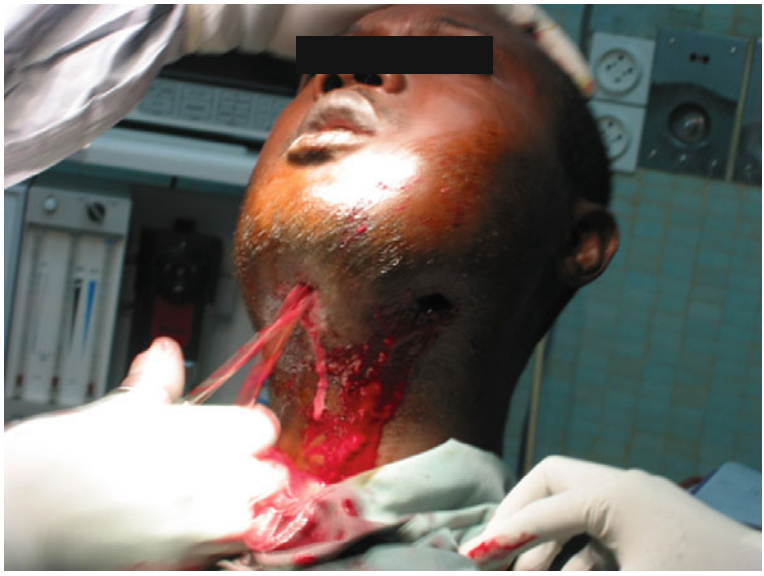

Fig. 44.11 Urgent decompression of the floor of the mouth in patient in Fig. 44.10

small bowel with intestinal bleeding and perforation of the patch during the third week usually on the antimesenteric border situated within 45 cm of the ileocaecal valve [27].

Intestinal perforation (Fig. 44.13) is the most common complication of typhoid fever and without surgery is uniformly fatal [28]. The current surgical options include copious washout of the abdomen, freshening of the ulcer followed by primary doubled layered closure, segmental resection and end-to-end anastomosis and primary ileostomy (Fig. 44.14). Medical treatment includes chloramphenicol, amoxicillin and cotrimaxazole.

Fig. 44.12 Patient from Figs. 44.10 and 44.11 the following day

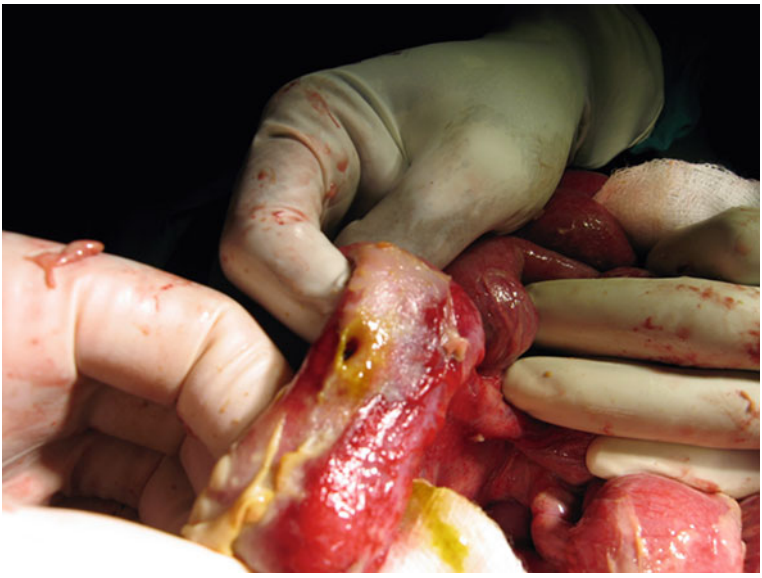

Fig. 44.13 Typhoid perforation

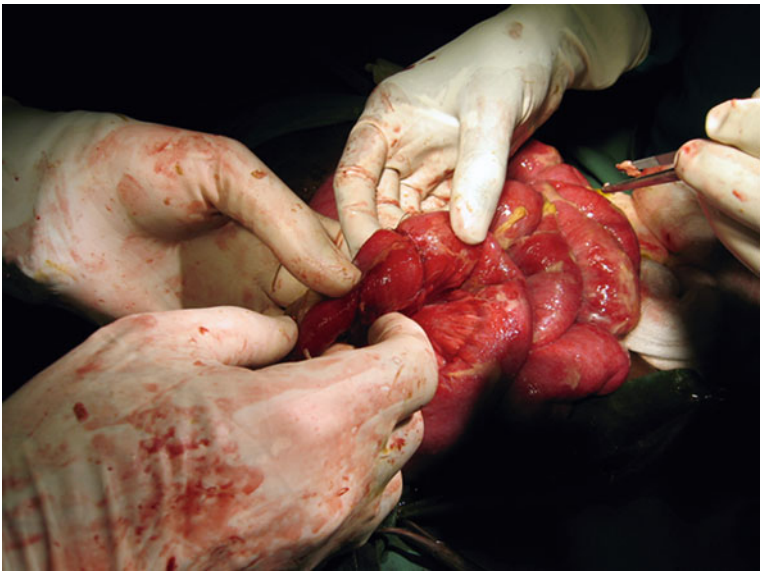

Fig. 44.14 Typhoid perforation closed by 2-layered anastomosis

Volvulus of the Sigmoid Colon

A high-fibre diet has many advantages, which are said to include the low incidence of appendicitis, and a much lower incidence of carcinoma and diverticula of the colon. But it may have at least one disadvantage. A large sigmoid colon distended with gas due to a high-fibre diet is more liable to twist on its mesentery and is the commonest cause of large bowel obstruction, particularly in Africa. The main danger of deflating a patient with a sigmoidoscope is that gangrene may be missed which could be lethal (Fig. 44.15). In Africa if there is any doubt, perform a laparotomy. It is the best investigation.

Amoebiasis

Caused by the protozoan parasite *Entamoeba histolytica* and has a worldwide distribution. It is estimated that 40–50 million cases of amoebic liver abscesses occur annually with 40,000 deaths per year [29]. Transmission is via the faecal-oral route following ingestion of infective cysts in food or drinks contaminated by faeces or possibly transported by insects such as flies and cockroaches. The cysts survive gastric acidity, but once in the large bowel they release trophozoites in the colonic mucosa causing amoebic colitis, and further spread within the portal vein may lead

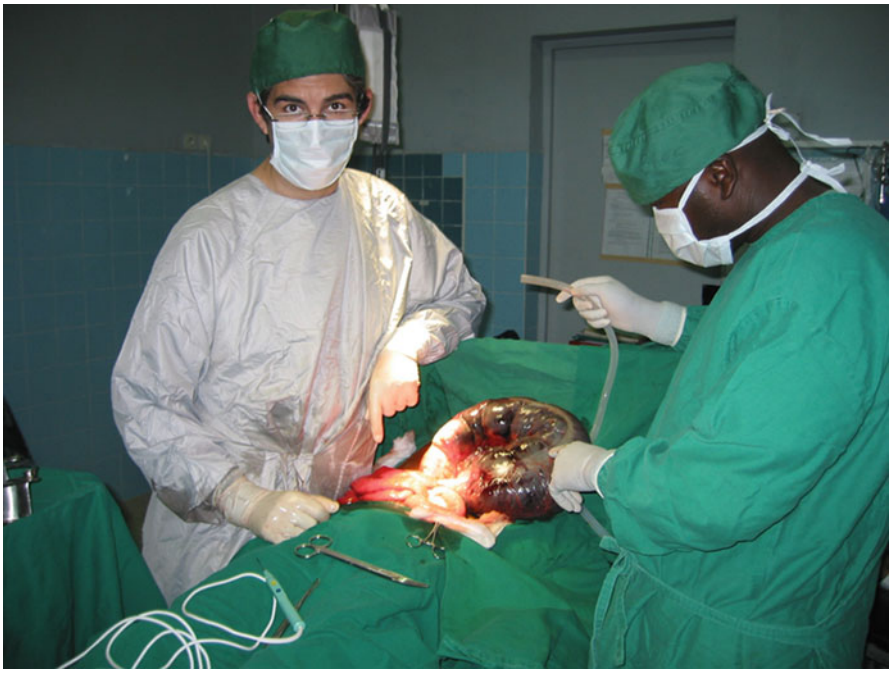

Fig. 44.15 Gangrene of the sigmoid colon due to volvulus

to liver abscesses or abscesses at other sites. A colitis develops gradually with loose stools with blood and mucus and colicky abdominal pain usually in the lower abdomen. The infection can continue for weeks or months and tends to involve the caecum and ascending colon, which may give right abdominal tenderness. The appendix is rarely affected, but it's important to take a good history because weight loss predominates with incessant diarrhoea and although, presenting with right iliac fossa pain, appendicectomy may be perilous as perforation of the colon may ensue. Other differential diagnoses are ileocaecal tuberculosis, schistosomal colitis and Trichuris infection. In severe cases the whole colon may be distended giving an acute toxic megacolon (Figs. 44.16 and 44.17).

Abscesses usually occur in the right lobe of liver and may eventually perforate into the pleural cavity, peritoneal cavity or, if in the left lobe of the liver, into the pericardial cavity with the risk of cardiac tamponade. The patient may present with fever and sweating and pain in the right hypochondrium aggravated by inspiration, which may radiate to the right shoulder. Symptoms of dry cough and difficulty breathing occur if there is a pleural effusion or emphysema or brown haemoptysis if the abscess ruptures into a bronchus. It is now recommended not to aspirate the amoebic liver abscess (classical anchovy sauce) as medical treatment such as metronidazole and tinidazole will give rapid improvement of the clinical condition.

Fig. 44.16 Toxic megacolon in amoebic dysentery in a 7-year-old

Schistosomiasis

Schistosomiasis is the generic name given to diseases caused by parasitic flukes of the genus *Schistosoma*. An older name, still widely used in Africa, is Bilharzia after Theodor Bilharz who discovered the condition in 1851. Although exact figures are hard to obtain, it is estimated that out of 600 million people at risk of schistosome infection, 200 million infected live in Africa and 20 million suffer from severe sequalae [30]. Of the three main species that commonly infect humans, two occur predominantly in Africa. *Schistosoma mansoni* is the cause of intestinal schistosomiasis, and *Schistosoma haematobium* produces urinary schistosomiasis. The adult flukes causing human schistosomiasis are wormlike creatures 1–2 cm long, which have a groove (schist), which gives them their name. The life cycle is digenetic with an asexual phase in freshwater snails and a sexual phase in the human. The eggs hatch in freshwater to release free-swimming miracidium larvae, which penetrate freshwater snails. The larval form, the cercaria, breaks out of the snail and penetrates human skin. Following migration to the liver via the portal venous system (*S. haematobium*) or to the venous plexus of the bladder (*S. haematobium*), they enter the sexual phase over the next 20 or 30 years producing eggs which lead to granuloma formation causing fibrotic changes and cirrhosis. Patients with *S. haematobium*

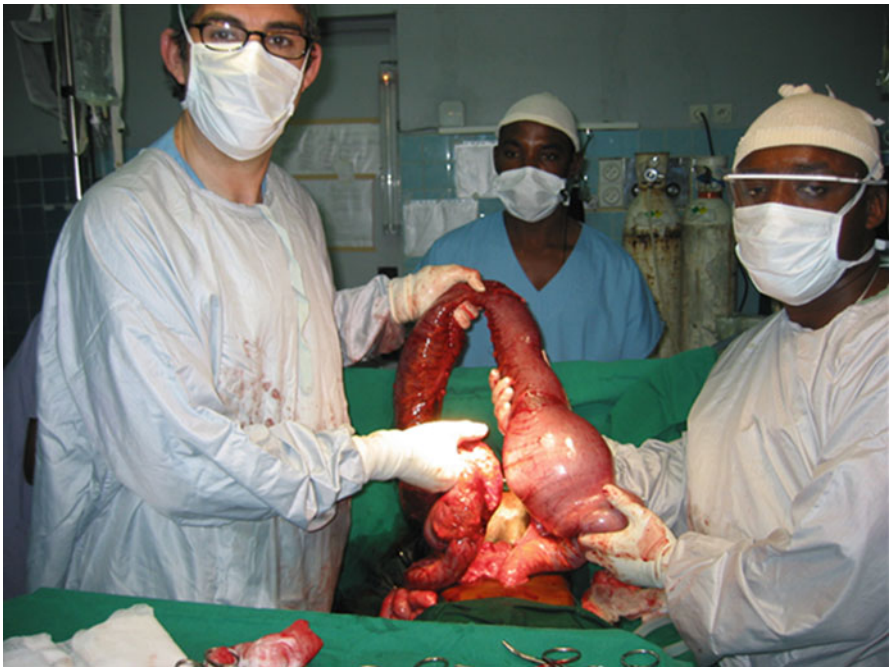

Fig. 44.17 Laparotomy specimen of toxic megacolon

present with symptoms of urinary tract infection with suprapubic pain frequency, dysuria and haematuria, the haematuria classically coming at the end of micturition. *S. mansoni* often presents with massive haematemesis with an enlarged liver and spleen with ascites due portal hypertension. Giant urticaria can occur with acute toxaemic reaction with fever, chills and hepatosplenomegaly (Katayama fever, Fig. 44.18).

Currently, the drug used in treatment is praziquantel; however, it only is effective against adult worms and does not affect eggs or immature worms. Treatment with this drug is simple, and its dose is based on the patient's weight with two doses given on 1 day. The drug causes rapid disintegration of the worm, which in turn allows the human immune system to attack the parasite.

Hydatid Disease

I have seen this condition many times; in Africa it is rife in northwest Kenya in the Turkana region. In this tribe of 200,000, there are approximately 200–300 new cases of hydatid disease each year. A local custom was to use nurse dogs to clean infants.

Humans are infected by hand-to-mouth transfer of all ova picked up by stroking a dog or drinking water or eating food contaminated with dog faeces. The adult stage

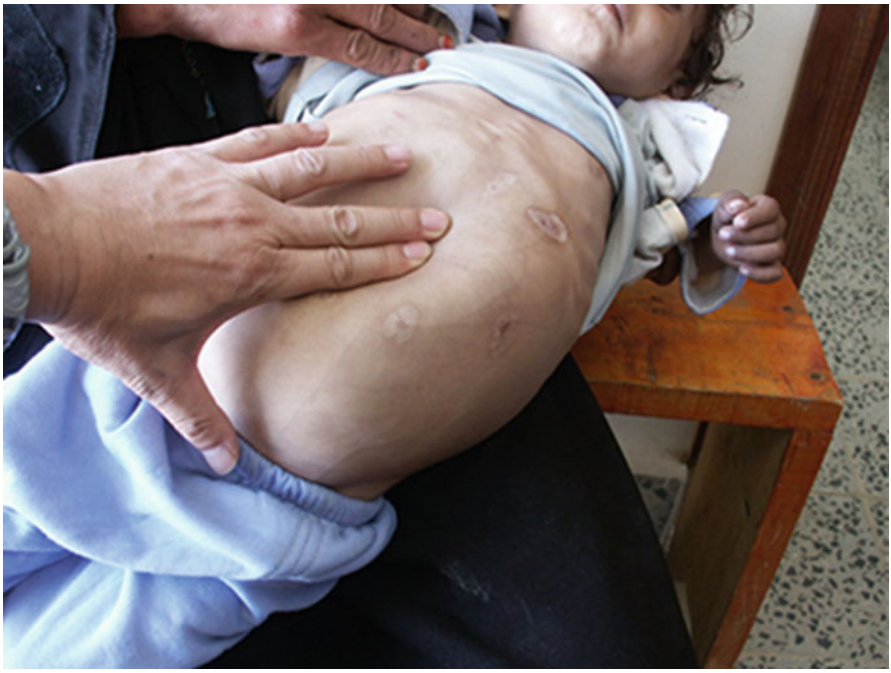

Fig. 44.18 Katayama fever in a child from Yemen

is the tapeworm *Echinococcus granulosus* about 5 mm long, which is found in enormous numbers in the small bowel of the dog. Once ingested by the human, larvae penetrate the gut wall, which travel to various sites to form cysts. A cyst contains capsules full of tapeworm heads. Seventy percent occur in the liver and about 20 % in the lungs and there may be single or multiple. Symptoms are caused by the mass effect of the growing cyst, by secondary bacterial infection or leakage of the cyst. Spillage or leakage of the cyst fluid can produce intense hypersensitivity reaction ranging from urticaria, a pruritic fever, to fatal anaphylaxis. The patient in Fig. 44.19 was a man who fell from a height onto his right side of the abdomen and burst one of these cysts. He was admitted hypotensive and in severe shock; the preliminary diagnosis of ruptured spleen was made, but in fact it was bright red due to the significant hypersensitivity reaction, and the shock was due to anaphylaxis. At laparotomy a huge cyst in the right lobe of the liver had burst. The cyst was completely removed from the liver bed. He was treated post-operatively with albendazole.

Intestinal Roundworms

These are soil-transmitted helminths, and their life cycle depends on the period of development outside the human host, typically in moist warm soil. Most important

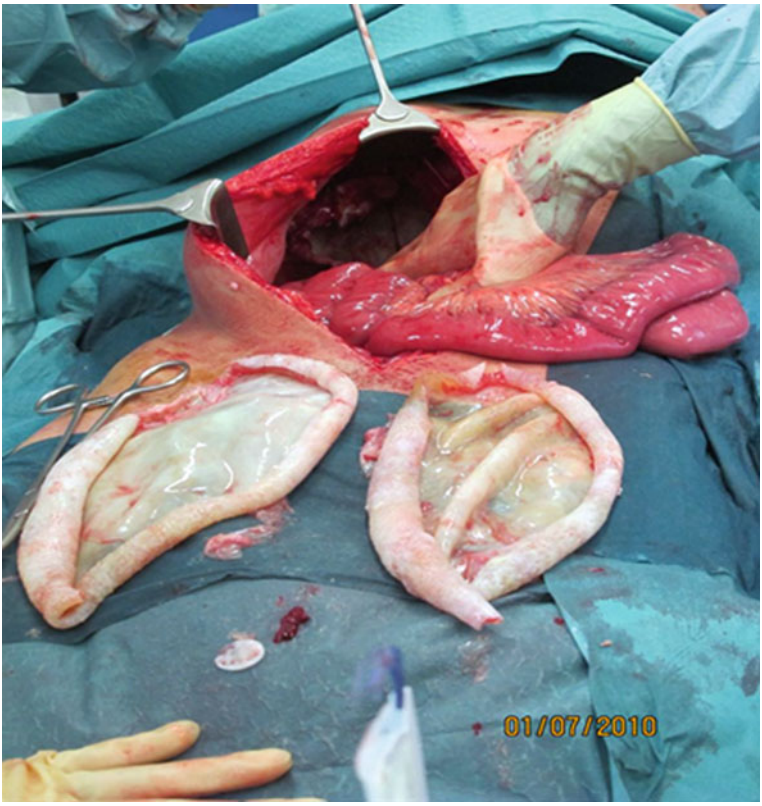

Fig. 44.19 Hydatid disease of the right lobe of the liver

of these globally are roundworms (*Ascaris lumbricoides*), whipworms (*Trichuris trichiura*) and hookworms (*Ancylostoma duodenale*).

Ascaris lumbricoides (Figs. 44.20 and 44.21) affects over 600 million people worldwide. They gain access to the human via the faecal-oral route. The *Ascaris* eggs liberate larvae as they pass through the stomach and small intestine, which penetrate the intestinal mucosa to enter the bloodstream and lymphatics and reach the lung about 2 weeks later. They then penetrate the alveoli and migrate via the respiratory tract to be coughed up and swallowed finally living in the small bowel where they obtain nourishment. Adults are large, cream-coloured worms between 15 and 40 cm in length and can produce up to 200,000 eggs per day. They are excreted in faeces and there remain viable for years. From a surgical point of view, worms may form a bolus in heavy infections causing intestinal obstruction, perforation and peritonitis or can migrate to the common bile duct causing cholangitis, pancreatitis and liver abscess; they can block nasogastric tubes and have been seen inside endotracheal tubes. On several occasions, I have seen them free swimming in the abdomen following abdominal gunshot wounds. A particularly unpleasant sight! They have also been known to escape through the suture line in single-layer bowel anastomoses, and therefore in Africa if performing bowel anastomoses, I routinely

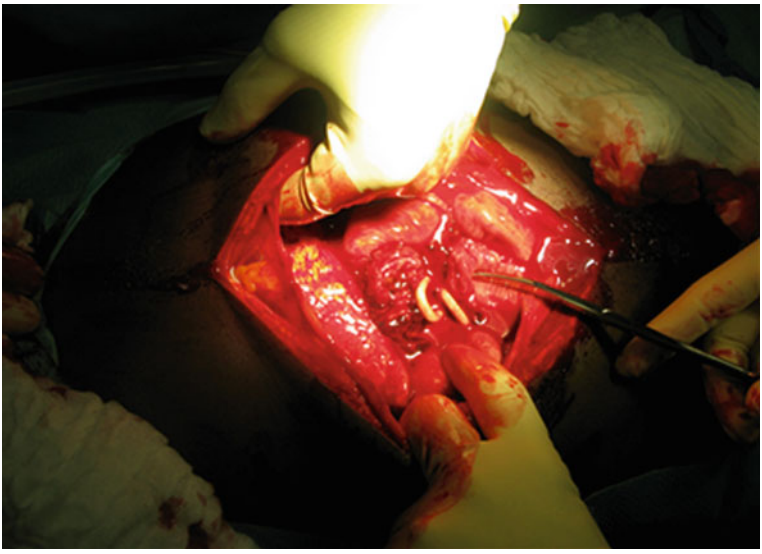

Fig. 44.20 *Ascaris lumbricoides* from the small bowel due to gunshot wound to the abdomen

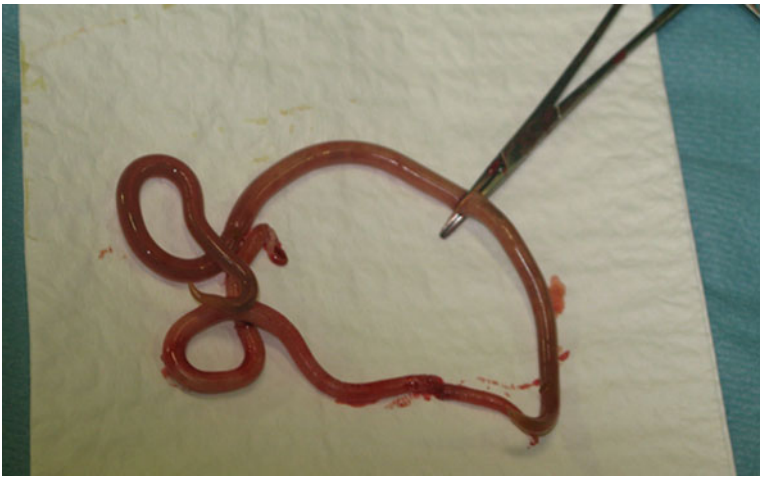

Fig. 44.21 *Ascaris lumbricoides*

and regularly perform double- or triple-layer anastomoses, followed by a course of albendazole if worms are detected.

*Hookworms (*Ancylostoma duodenale*)*

Hookworms normally penetrate the feet where they migrate in the veins to the right side of the heart and lungs and penetrate the alveoli and then into the trachea coughed up and swallowed. These worms are about 1 cm in length and live in the

small bowel sucking blood from intestinal mucosa causing severe iron deficiency anaemia. Again the treatment is albendazole; it is mentioned purely because it is a cause of severe anaemia and should be part of the differential diagnosis.

Whipworms (Trichuriasis)

Whipworm eggs when ingested hatch in the small intestine, and the larvae live in the caecum and sometimes rectum. The adult worms are 3–5 cm in length and cause infants to have severe chronic bloody diarrhoea, which is so intense that it causes a rectal prolapse (Fig. 44.22) . Treatment is with albendazole.

Filariasis

The WHO considers lymphatic filariasis to be the fourth leading cause of permanent disability worldwide. Larvae of *Wuchereria bancrofti* are ingested by female mosquitoes and undergo further development within the mosquito. Larvae are then injected into humans by the mosquito and develop further eventually migrating into lymphatic system. The primary pathological process is local damage to the lymphatics almost certainly as a result of local inflammatory response. The initial presentation is fever

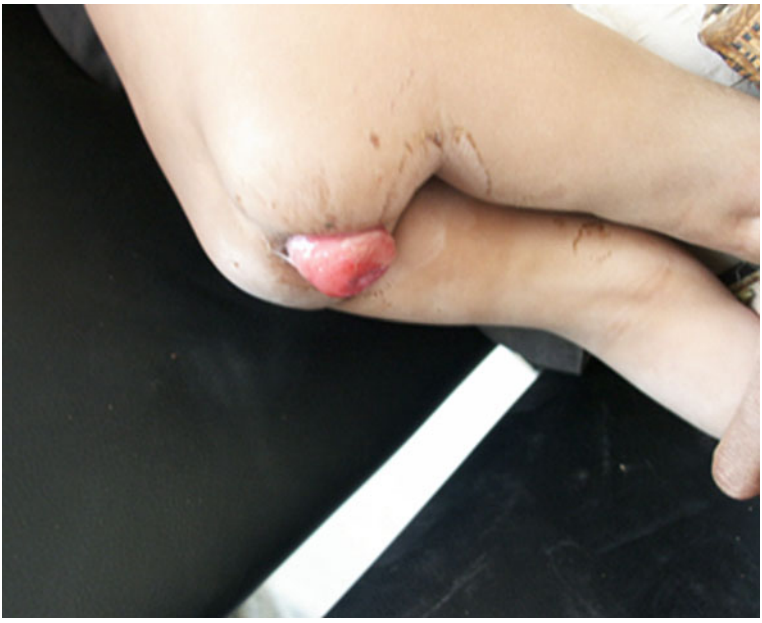

Fig. 44.22 Whipworm infection causing rectal prolapse in a baby

with painful lymphadenopathy and lymphangitis followed by gross swelling of the leg and/or the upper arm and breast in women and external genitalia in men. There are some oddities associated with this condition. I remember being shown the urine of a man who was allegedly passing milk; the diagnosis was made after removal of his gown revealed gross elephantiasis of one leg. Most likely the dilated lymph vessels in his pelvis had ruptured and discharged lymphatic fluid into the bladder thus producing the milky appearance known as chyluria. The surgical management of this condition is beyond the scope of this chapter, and the medical management is also changing from the classical diethylcarbamazine (DEC) to combination treatments [31].

Onchocerciasis

Onchocerciasis or river blindness is caused by the filarial worm *Onchocerca volvulus* transmitted by the bloodsucking Simulium black fly, breeding near fast rivers. Approximately 270,000 people in 500,000 have significant visual loss making it the world's fourth most common cause of blindness [32]. Apart from causing blindness the adult filariae become encapsulated in fibrous tissue, which forms nodules in subcutaneous tissues to the extent that the inguinal lymph nodes can become so large that they can be misdiagnosed as incarcerated inguinal hernia (Fig. 44.23), which I have seen on occasions. But there are other telltale signs before one operates

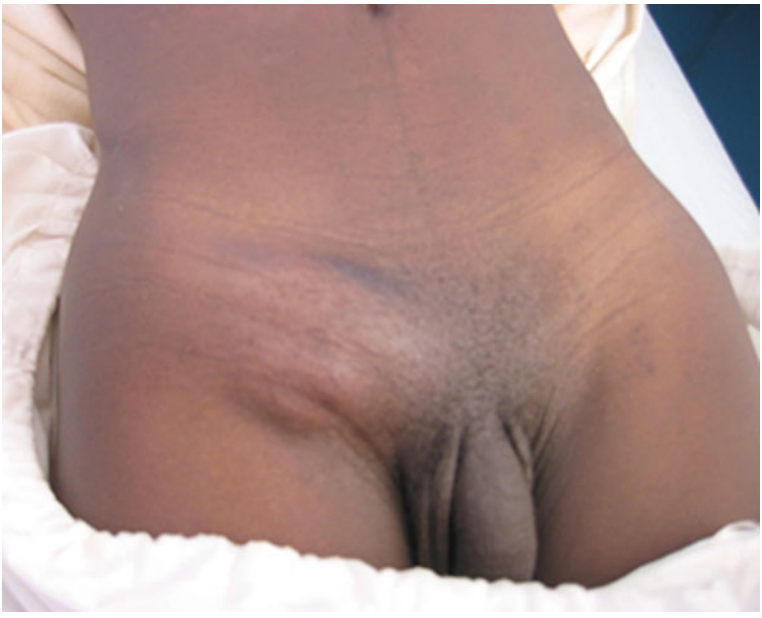

Fig. 44.23 Lymph node enlargement mimicking incarcerated hernia in a patient with onchocerciasis

that is to look at other nodules and the pre-tibial deep pigmentation sometimes known as leopard skin where atrophy of the skin can also occur resulting in a tissue paper appearance. The mainstay of this treatment is medical notably ivermectin.

Osteomyelitis

This is a particularly tragic disease, which has almost disappeared from the industrialised world, but is very common in Africa. There are three kinds of osteomyelitis. Acute haematogenous osteomyelitis mainly affects the metaphysis of the tibia and femur. Pyogenic staphylococci remain the commonest causative organisms although some yield *Salmonella typhi*, *Escherichia coli* and other exotic bacteria. The second cause is traumatic osteomyelitis following road accidents and is usually as a result of inadequate wound toilet. Thirdly and perhaps most importantly from a clinical point of view is osteomyelitis following orthopaedic procedures particularly internal fixation of femur fractures with intramedullary nails and metal plates and screws for tibial fractures. This is the main reason why patients in the tropics should be treated by immobilisation using combinations of plaster of Paris, traction and external fixation.

In tropical areas, acute haematogenous osteomyelitis (Fig. 44.24) is a common severe affliction with high morbidity and occasional deaths that affects infants and children. Unless treatment is early, most patients progress to chronic osteomyelitis. At onset, there is pain in the affected bone (commonly the tibia, femur or humerus), and often there is a history of recent trauma. Soon the pain becomes severe and high fever develops. The child refuses to use the limb, and the bone is tender to gentle percussion. Infants respond to infection with lethargy, failure to feed and subnormal temperature. Pseudoparalysis of the affected limb is an important sign but is more likely to be noticed by the mother than by medical staff. Concomitant septic arthritis (Fig. 44.25) is the norm, because the blood vessels crossing the epiphyseal plates of the infant provide a pathway for the spread of sepsis. If diagnosis is delayed, joint destruction occurs and loss of the femoral head is particularly common. Appropriate antibiotic should be given intravenously, then the joint drain thoroughly washed; the hip should be held in abduction to prevent septic dislocation and other affected joints sprinted in a functional position. A combination of ampicillin and cloxacillin of the broad-spectrum antibiotics is also indicated.

If the infection progresses areas of periosteal reaction are separated by a smooth area of unreactive cortex, which becomes nonviable, and subsequently becomes known as the sequestrum. In sub-Saharan Africa, sickle cell disease is common. Children who are homozygotes for the sickle cell gene develop a chronic haemolytic anaemia with splenomegaly and intermittent jaundice. They have periodic sickling crises, which may include painful bone infarction, and they also frequently develop acute haematogenous osteomyelitis due to staphylococcus and salmonella.

The treatment of post acute osteomyelitis is with analgesics and antibiotics and by draining the extra osseous abscess.

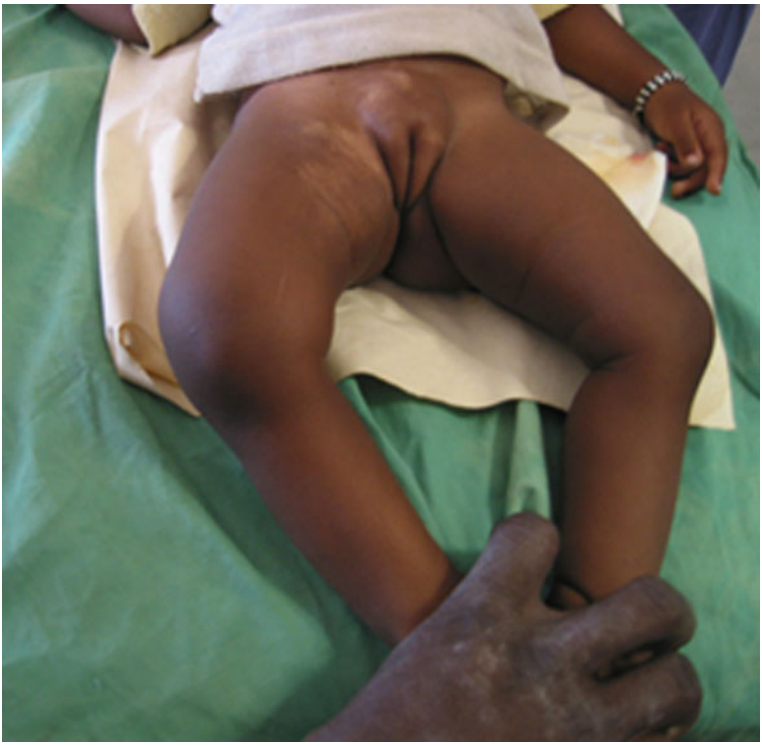

Fig. 44.24 Acute haematogenous osteomyelitis

Chronic Haematogenous Osteomyelitis

If the disease becomes chronic, then this is characterised by a swollen limb with sinuses discharging pus and palpably thickened bone (Fig. 44.26). Adjacent joints are often already stiffened by the time of first presentation. The stripped periosteum begins to form new bone overlying the dead cortex and is called the involucrum. This forms to maintain the integrity and length of the bone; however, the dead cortical bone, the sequestrum, continues to cause sinuses and accumulation of pus. Radiographs as in Fig. 44.27 show a characteristic pattern of bone destruction, sequestrum, and new bone (involucrum) surrounding the sequestrum (Fig. 44.28). There is a spectrum of severity ranging from severe infections with huge diaphyseal sequestrum and inadequate involucrum that fails to bridge the remnants of missing bone to small sequestrum surrounded by sealed cavity of pus or surrounded hypertrophic cortical bone. The latter situation is often described as a Brodie's abscess, which gives rise to intermittent pain, fever and swelling. The bacteriology of chronic osteomyelitis is mixed. The variety of organisms isolated increases with the number of swabs taken and usually include staphylococcal, bowel flora and

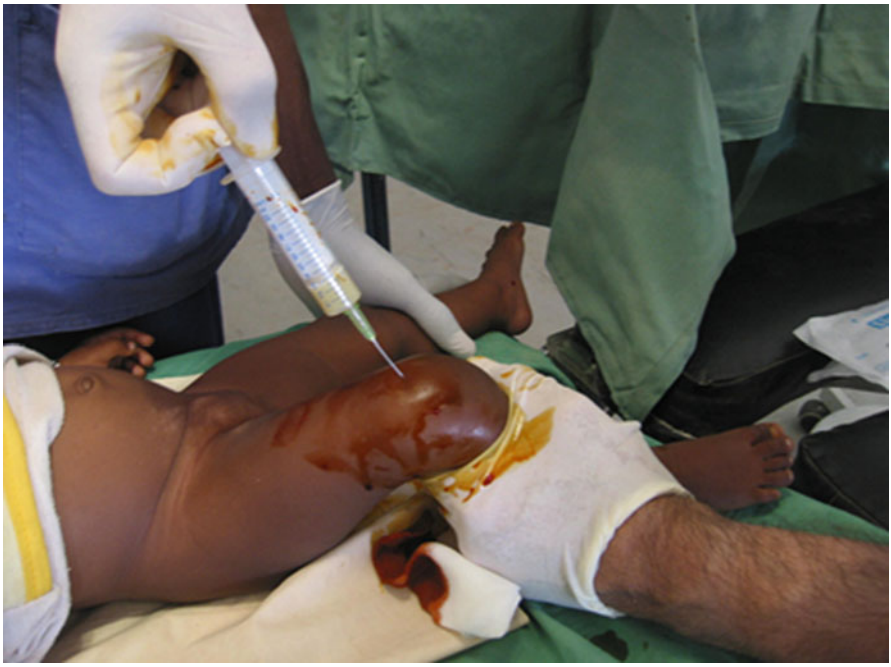

Fig. 44.25 Aspiration of pus from septic arthritis

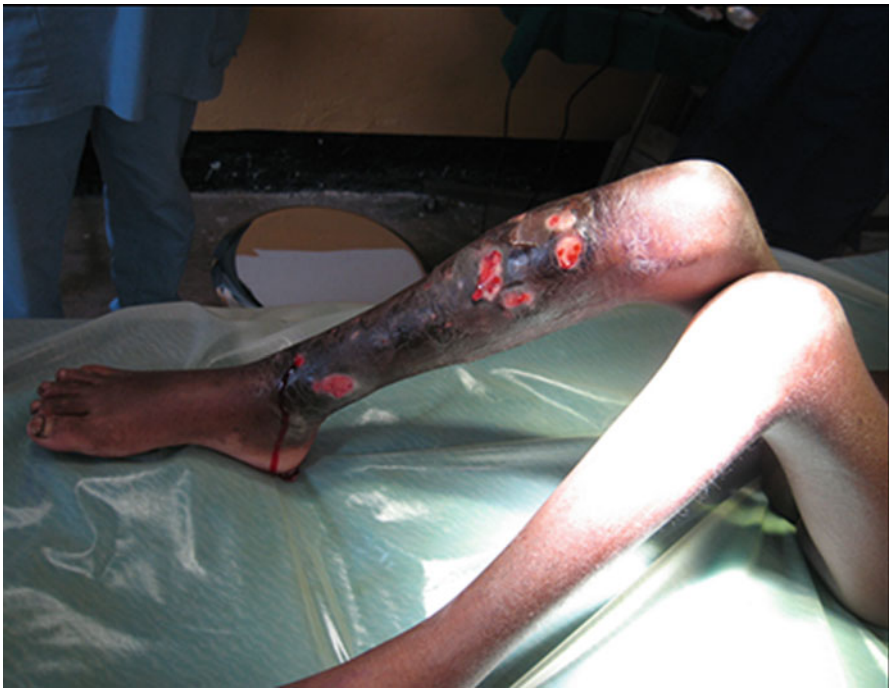

Fig. 44.26 Chronic osteomyelitis with sinuses

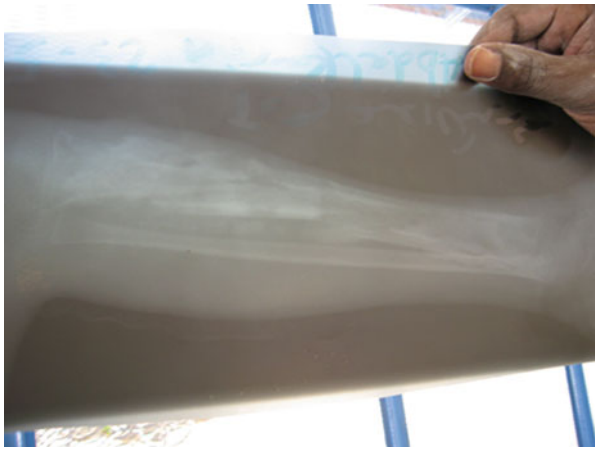

Fig. 44.27 Osteomyelitis showing new bone (involucrum and dead bone the sequestrum)

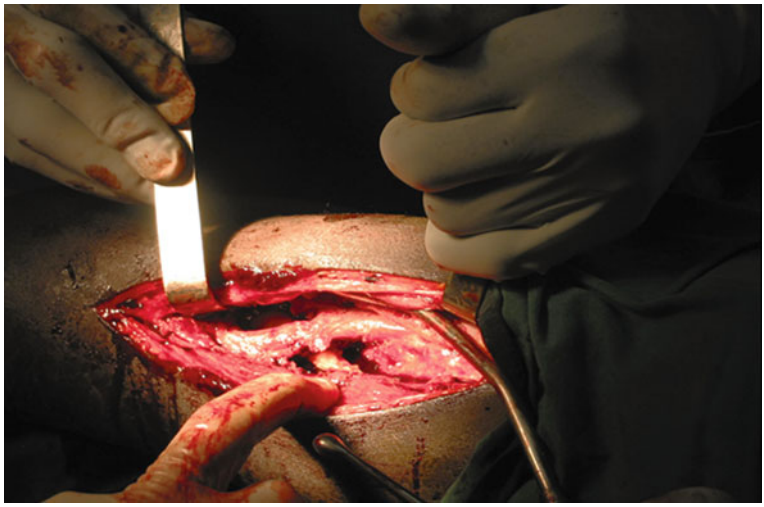

Fig. 44.28 Sequestrum and involucrum

anaerobic organisms. Antibiotic should be given immediately before surgery is undertaken in a few days post-operatively to prevent septicaemia, but the treatment of chronic osteomyelitis is essentially surgical. The limb should not be used, but protected in a brace to allow the involucrum to bridge the dead bone. Sequestrectomy can then be done (Fig. 44.29). If the involucrum has failed to progress within 3 months, then sequestrectomy should be performed to bring infection under control, whilst providing stability and maintaining limb length with external fixation. When small sequestra lie in a cavity surrounded by thick new cortical bone, the cavity must be widely opened (saucerised), cleared and opened into the adjacent medullary cavity proximally and distally and the wound left open for daily saline dressings.

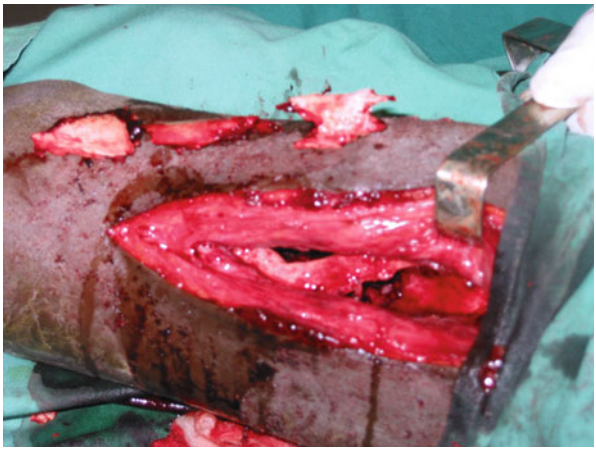

Fig. 44.29 Sequestrectomy

Trauma in the Tropics

Often patients arrive having had long and difficult transportation with no prehospital care. There is limited emergency room competence with the staff not being trained enough to understand the concept of emergency care and certainly not within the bounds of ATLS. There is often no x-ray machine and little diagnostic capability. Once treated, the patient may discharge himself or through cultural reasons refuse treatment or if treated may never return for follow-up. From the trauma point of view, most injuries will have self-selected themselves, and very unusually you may have to treat patients within the golden hour which mainly involves control of haemorrhage. More likely patients will be seen several hours or days following the accident. The patients who suffer trauma and blood loss may have also been anaemic prior to the accident and are severely anaemic on presentation. Usually if blood for crossmatch is available, then a target of 8 g/l is usually satisfactory. Consideration for fluid management is a high priority, and the patient may be significantly dehydrated because of the length of time taken getting to the hospital and the ambient temperature. A target of urine output of 30mls per hour is mandatory prior to surgery as a guide for resuscitation. The mainstay of orthopaedic management for fractures will be reduction and stabilisation usually with traction and plaster of Paris. External fixation is mainly used for soft tissue management during open fractures, septic non-union and malunion of fractures. Usually the external fixator remains in place for between 2 and 6 months. The techniques are not difficult to perform but should not be used without consideration of simpler methods of fracture fixation. Internal fixation should never be used in this sort of environment for the reasons described previously. To assess whether or not the fracture has healed in this environment, after a period 3 months or so, loosening the connection struts and allowing the patient to walk and if without pain, then you can assume the fracture has healed and the pins can then be taken out. Obviously a further few weeks of gentle weight bearing before full weight bearing is necessary. Figure 44.30 shows a fractured femur treated with traction and physiotherapy.

Fig. 44.30 Fractured femur treated by traction and physiotherapy

Obstetrics in the Tropics

Between 5 and 10 % of babies die in the perinatal period from 28 weeks of pregnancy to 7 days postpartum. Intrauterine deaths are often unexplained, but preventable causes include malaria, syphilis and obstructed labour. Most perinatal deaths in Africa are usually of normally formed, normal-weight babies who die avoidably from trauma, asphyxia or infection. Many neonatal deaths occur in babies whose low birth weight is due to prematurity or intrauterine growth retardation, the deaths of both mothers and babies mostly due to the maternal and social conditions under which they live.

Anaemia, Malaria and HIV/AIDS During Obstetric Surgery

Major causes of anaemia in pregnancy are malaria, iron deficiency due to worms, folate deficiency and haemoglobinopathies and AIDS. Anaemia is often multifactorial, with the different causes interacting in a vicious cycle of depressed immunity, infection and malnutrition.

Anaemia progresses through three stages:

(a) Compensation, with breathlessness on exertion only
(b) Decompensation, with breathlessness at rest and haemoglobin (Hb) below 7 g/ dl
(c) Cardiac failure, with Hb below about 4 g/dl

Surviving infants have low birth weights, immune deficiency and poor reserves of iron and folate. They have entered already the vicious cycle of infection, malnutrition and impaired immunity. Treatment with blood transfusions is even more hazardous because of HIV and should be limited to saving the life of the mother. Treatment of malaria is complex as chloroquine-resistant strains are now common. Prevention remains relatively easy with proguanil and supplements of iron and folic acid and is highly cost-effective in the improvement of maternal and infant health; it is more important than ever as it avoids the unnecessary exposure of women and infants to HIV transmitted through blood transfusion.

Patients arriving after long journeys from distant villages in obstructed labour are often dehydrated, ketotic, shocked, anaemic or infected. Performing an emergency Caesarean section through infected tissues may be complicated by peritonitis, which antibiotics may fail to control. When recovered, memories of a frightening operation followed by a difficult puerperium may make the mother deliberately not seek hospital care when she becomes pregnant again. If the baby dies, the family may blame the hospital and decide to have the next one at home. Unfortunately, Caesarean section seldomly removes the factor, which caused the obstructed labour that is of a narrow pelvis. In addition to that, the scar in the uterus following caesarian section is now its weakest part, and therefore the chances of the uterus rupturing on the during the next birth are very high.

Obstructed Labour

Obstruction is “the failure of the presenting part to descend in spite of uterine contractions”. What really distinguishes delay from obstruction are the secondary signs and complications such as severe moulding and caput, fetal distress, stretched lower segment, bloody urine, fistulae and rupture of the uterus. Whereas delayed labour is usually inevitable and readily treatable and is comparatively harmless, obstructed labour is none of these things. It should never happen where care is adequate. Obstruction may be due to (a) a small contracted and underdeveloped pelvis; (b) an abnormality in the baby, e.g. hydrocephaly; and (c) an abnormality in the birth process. This can be either an abnormal lie or presentation such as a breach, a brow, a face, a shoulder presentation or a prolapsed arm in a transverse lie or an unfortunate coincidence of the relative sizes of the head and the pelvis such as cephalopelvic disproportion (CPD). Rarer causes such as stenosis of the vagina, locked twins or pelvic tumour, particular fibroids or an ovarian cyst. CPD is the most important

cause (two-thirds of these cases), and an impacted transverse lie is second most common cause.

In a labour that is going to obstruct, the first stage is often prolonged, but it can be normal or even short. The membranes rupture and liquor escapes, and the uterus contracts and retracts and forces the baby into its lower segment, which gradually becomes overstretched. Obstruction prevents the baby's escape, so the lower segment moulds closely around and thins. The contractions of the uterus become hypotonic and relaxation between them poor. The placenta becomes poorly perfused and fetal distress occurs and unfortunately the baby dies. Obstructed labour has two main dangers: (a) the vagina, bladder and rectum are trapped between the head and pelvic symphysis so that they become necrotic, slough and develop fistulae, and (b) the uterus ruptures. Primips usually develop fistulae, and multips usually rupture, but both can do either and rupture and fistulae can occur in the same patient. If the mother and baby do not die from the process and the dead baby is delivered either by caesarian or by symphysiotomy, then the likelihood of developing a chronic vesicovaginal fistula is very high.

Vesicovaginal Fistula

One of the most satisfying operations a surgeon can perform is that of vesicovaginal fistula repair. These patients are normally rejected by their husbands and families and often develop flexion contractures of their knees and hips because they have taken to lying and squatting in a darkened room because of the smell of urine. Following vaginal examination, a fistulous connection from the anterior vagina wall to the bladder is easily confirmed; however, the procedure to repair it is usually straightforward but can be difficult. I usually perform the operation with the patient lying in the prone position (Fig. 44.31) and carefully separate the anterior vagina wall from the bladder and separately suture the bladder and vagina with a 2/0 Vicryl stitch. A catheter is then placed in the bladder for several days to allow healing (Fig. 44.32). Figure 44.33 shows a repaired fistula.

Conclusion

Surgery in the tropics is an exciting area of medicine to explore. The lack of investigations and equipment brings medicine back to basics. Clinical acumen needs to be fine-tuned, and a knowledge of tropical medicine is a must before one can understand the diseases that may influence decision-making. For a western-trained surgeon, embarking on a first mission to Africa takes a good supply of information in the form of books on a USB stick because your educational needs will be high.

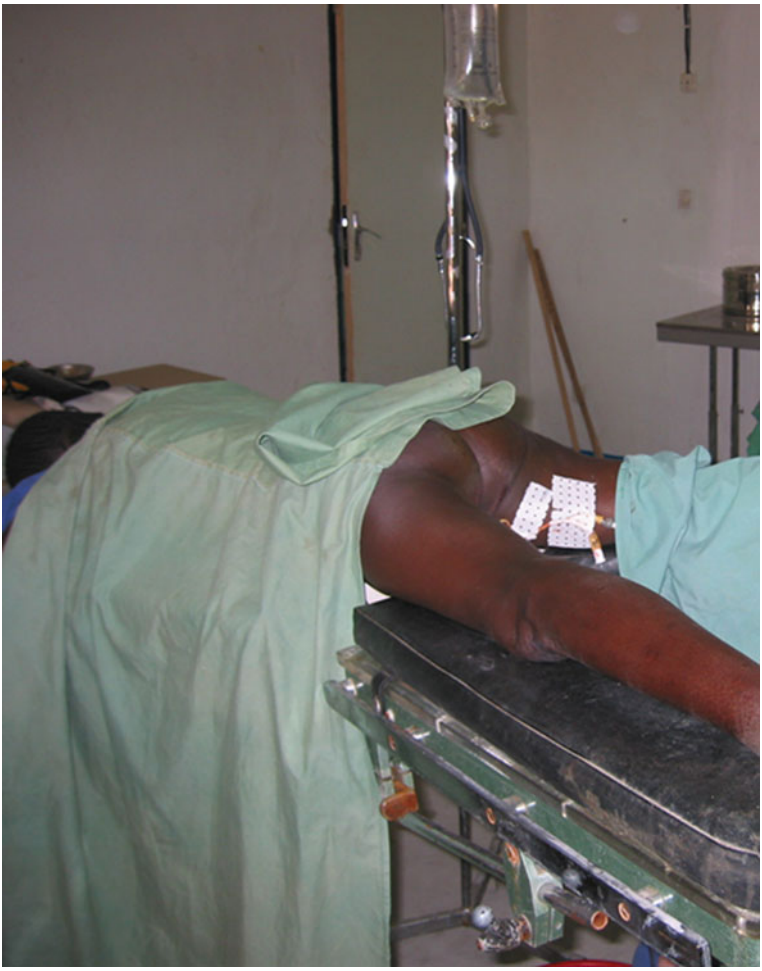

Fig. 44.31 Position of the patient for repair of vesicovaginal fistula

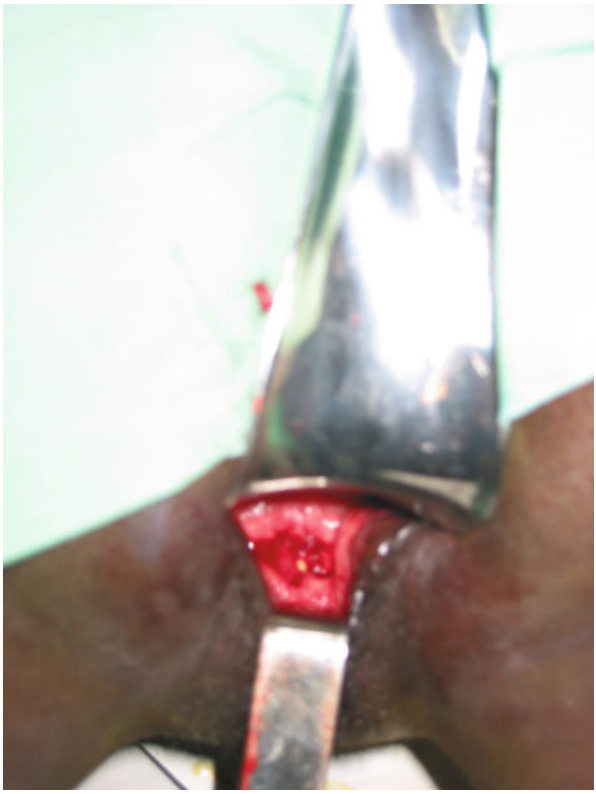

Fig. 44.32 Vesicovaginal fistula with catheter in the bladder

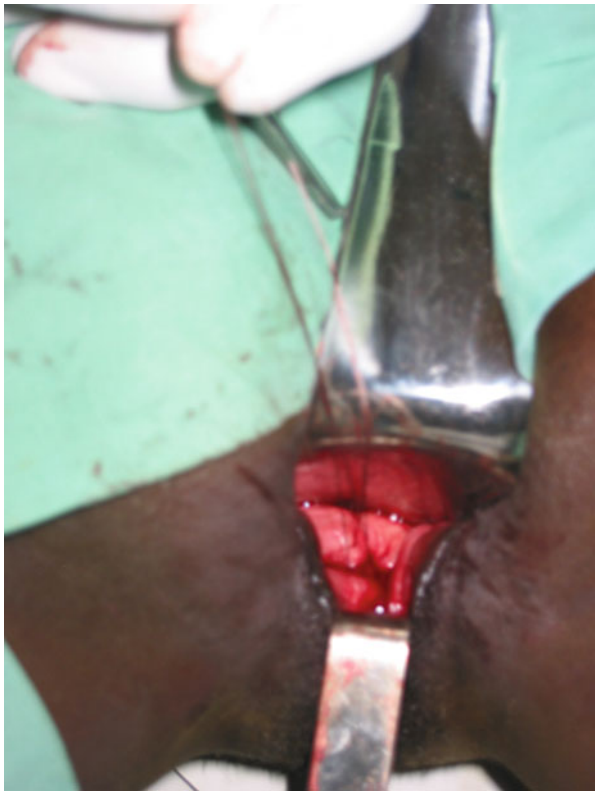

Fig. 44.33 Repaired fistula

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