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Tropical Medicine and Anaesthesia; part 2

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Learning objectives

By reading this article, you should be able to:

1. Identify which tropical diseases may require surgery or complicate anaesthesia.
2. Explain the interactions between specific conditions, their pathophysiological sequelae and management, and how these combine to affect the approach to anaesthesia.
3. Discuss the risks and benefits associated with blood product use in resource-poor settings and explain the different considerations compared to high-income environments.

Key points

1. Tropical diseases may represent both an indication for surgery and be a complication for anaesthetic management.
2. The anaesthetist must consider the effects of the underlying condition, its sequelae, and its treatment as all may affect management.
3. Comorbidity and polypharmacy are common in resource poor-settings, often with an unpredictable effect on anaesthesia.
4. Blood product transfusion has particular risks in areas with endemic blood-borne infection.

Introduction

'Tropical Medicine and Anaesthesia 1' (*BJA Education* 2018; 18,2: 35–40) focused on those common tropical diseases that can affect anaesthetic practice in any setting. This second article is concerned with the practice of anaesthesia in tropical locations, and explores further those tropical diseases that may require surgical intervention, and the medications that may interact with anaesthetic agents. Some of the issues around blood product administration in the tropics are also discussed.

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Tropical conditions requiring surgical intervention

Typhoid

Typhoid, or enteric fever, has an estimated 21.7 million cases annually worldwide.[1] It commonly presents with non-specific symptoms of headache, fever, cough, and abdominal pain; constipation or diarrhoea also occur frequently. Caused by *Salmonella enterica*, with various *Salmonella typhi* or *Salmonella paratyphi* serovars, typhoid is usually diagnosed clinically although a definitive diagnosis can be made by isolating the

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bacteria from specimens such as blood, bone marrow or bile. The Widal agglutination test, commonly used to diagnose typhoid fever, should be regarded as an unreliable diagnostic test because of its low sensitivity, specificity, and positive predictive value.[2]

Complications occur in 10–15% of patients, of which intestinal perforation is the most serious.[3] Perforation, which may be at multiple sites, occurs due to necrosis of Peyer's patches and may present with an acute abdomen, or worsening of pre-existing abdominal pain. Mortality from perforation can be up to 30% despite surgery, rising to 70% in those unable to access timely surgical care. Patients are likely to be critically ill, with aggressive fluid resuscitation and high-dependency postoperative care indicated. Gastrointestinal bleeding may also occur as a complication of typhoid, but is usually self-limiting and surgical intervention is only rarely required.

Hydatid disease

Human hydatid disease, or cystic echinococcus, is caused by infection with the larval stage of the canine tapeworm *Echinococcus granulosus*. Cysts typically develop in the liver, most commonly the right lobe, but approximately 20% are found in the lungs[4] (Fig. 1). Rarer sites include the eye, brain, peritoneum, spleen and bone. Cysts are usually asymptomatic until they grow large enough to produce a mass effect, become infected, or start leaking fluid. If hydatid antigens enter the circulation, hypersensitivity reactions or even fatal anaphylaxis can occur. Rupture may occur spontaneously but is more likely to complicate surgical or percutaneous cyst removal.[5]

Surgery, previously the mainstay of treatment, is now reserved for complicated cysts such as those that have caused biliary fistula, have ruptured, bled, or are infected.[6] Surgery may be open or laparoscopic, and hepatic resection may be necessary. Simple cysts are treated by percutaneous aspiration, usually under ultrasound guidance, injection with a scolecidal agent such as hypertonic saline or alcohol and then reaspiration (the puncture, aspiration, injection, reaspiration, or PAIR technique).

Adrenaline, antihistamines, and corticosteroids need to be available in anticipation of anaphylaxis. Surgical patients require a course of antihelminthic treatment, usually with albendazole with or without praziquantel, to reduce cyst dissemination and recurrence.[4]

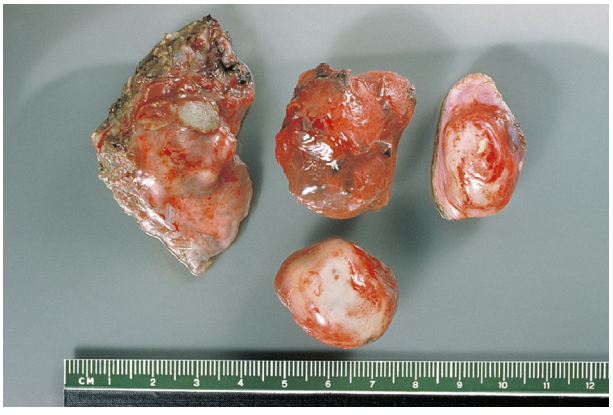

Fig 1 Gross pathology photograph of the membrane and hydatid daughter cysts excised from a human lung. Content provided by Centers for Disease Control and Prevention/Dr Kagan.

Amoebiasis

Amoebiasis caused by the parasite *Entamoeba histolytica* is found throughout the world, but is particularly prevalent in conditions of overcrowding and poor hygiene.[3] Symptoms may develop several years after the initial infection which usually presents as dysentery with crampy abdominal pain and bloody diarrhoea. Complications include abscess formation, strictures, haemorrhage, postdysenteric ulcerative colitis, peritonitis, and amoeboma formation: a chronic inflammatory mass that may cause intestinal obstruction or intussusception.[4]

Surgery may be necessary for acute colonic perforation, although aggressive resection of the bowel in cases of severe colitis is associated with high mortality.[7] Obstruction or intussusception caused by an amoeboma may also require surgical treatment. Liver abscesses usually respond to a course of metronidazole, but aspiration and drainage may be required, especially if infected or rupture appears imminent.[4]

Amoebic liver abscesses occur when the parasites enter the liver via the portal veins and destroy hepatocytes, causing multiple small abscesses that amalgamate.[4] Patients may present with fever and a dull right upper quadrant pain. There may be cough and right-sided pleuritic pain, but jaundice is uncommon.[3] Rupture of a liver abscess occurs in about 20% of patients and typically perforates through the diaphragm to cause an empyema, with hepatobronchial fistula a rarer complication.[3] Occasionally the abscess may rupture into the pericardium causing a cardiac tamponade, associated with significant mortality. Acute rupture into the peritoneum characteristically presents with peritonitis.

Schistosomiasis

Schistosomiasis, also known as bilharzia, affects around 200 million people worldwide, approximately 97% of whom live in Africa.[8] Caused by the blood fluke *Schistosoma*, there are three main types that infect humans and cause problems mainly in the urinary and intestinal tracts. These small worm-like creatures penetrate the skin and enter the circulation, migrating via the lungs to the liver where they mature.[4] Their final destination may be the mesenteric veins or vesical plexus, where they deposit eggs (Fig. 2).

Found in fresh water, schistosomiasis typically affects school-aged children as they have greatest exposure, but also occurs in those whose occupation puts them at risk. Acute schistosomiasis (sometimes called Katayama fever) typically causes a mild illness, with fever, myalgia, skin rashes, malaise, dry cough, nausea, vomiting, or diarrhoea. However severe disease occurs later in life.

Urinary schistosomiasis may occur due to the passage of eggs through the bladder. Terminal haematuria is the main symptom, but perforations in the bladder wall may also develop causing microalbuminuria.[4] Fibrotic changes and bladder calcification can cause bladder contraction and ureteric strictures resulting in hydronephrosis.[7] There is also an association between chronic schistosomiasis and squamous cell bladder cancer. Eggs may be deposited throughout the urogenital system, with inflammatory lesions occurring

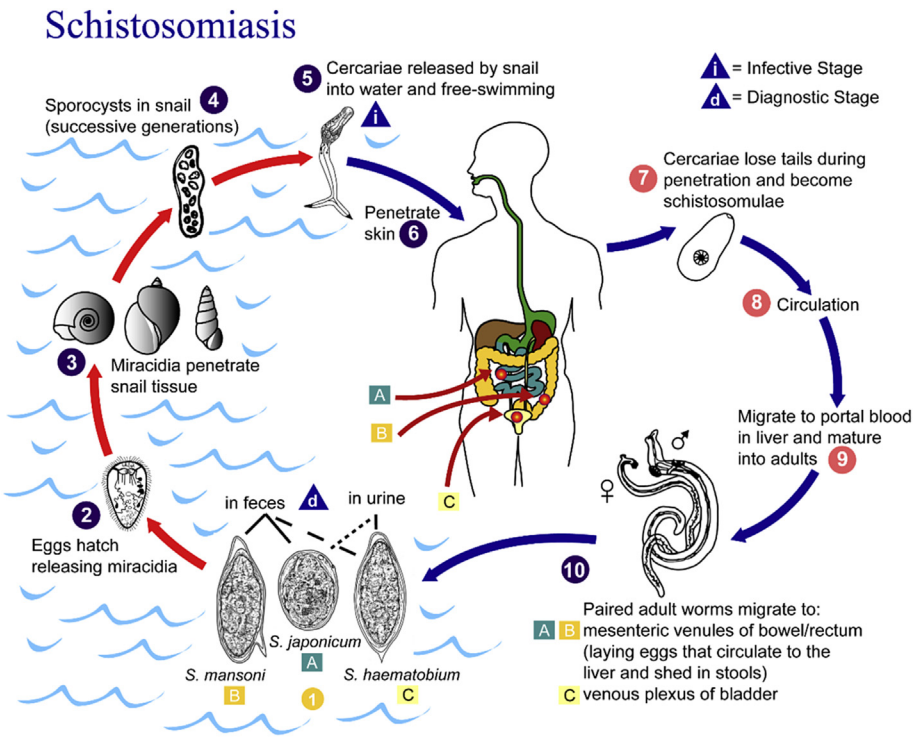

Fig 2 An illustration of the life cycle of the parasitic agents responsible for causing schistosomiasis. Content provided by Centers for Disease Control and Prevention/ Alexander J. da Silva, PhD/Melanie Moser.

around any deposited eggs. This may cause haematospermia in men, and may even cause sterility in women.

Eggs escaping the portal system may reach the lungs, with approximately 1% of those with chronic schistosomiasis developing pulmonary hypertension and, if severe, cor pulmonale. Chronic schistosomiasis also causes hepatic fibrosis caused by a periportal granulomatous response to eggs that have escaped from the lower mesenteric veins, leading to portal hypertension. Oesophageal varices and gastrointestinal bleeding may result, but hepatic function tends to be preserved, and ascites is absent until late in the disease.[4] Portal hypertension may lead to massive splenomegaly requiring splenectomy for symptom control or because of spontaneous splenic rupture.

Diagnosis of schistosomiasis is most effective by directly observing viable eggs. Eggs from the bladder are typically voided around midday and can be detected by sedimentation or filtration.[4] Those in the gut require examination of a stool sample or biopsy of rectal mucosa. Immunodiagnosis may detect circulating antischistosomal antibodies, but does not become positive until 3 months after infection and cannot distinguish between past and active infection.

Schistosomiasis is effectively treated with a single oral dose of praziquantel, which has very few serious side effects and is ideal for individual treatment as well as for large-scale population treatment.

Lymphatic filariasis

Endemic in >80 countries, one fifth of the world's population is at risk of lymphatic filariasis.[9] Caused by filarial worms transmitted by mosquitoes, *Wurchereria bancrofti* accounts for over 90% of cases. Infection usually occurs in childhood, but the long-term consequences such as elephantiasis tend not to manifest until adulthood.

The most common chronic complication is filarial hydrocele, a condition that affects approximately 27 million men worldwide and develops because of mechanical blockage of the lymphatic vessels by adult worms.[9] The resulting accumulation of fluid may itself be problematic, but it also lead to further complications including lymph scrotum, recurrent infections, and inguinal adenitis.

Diagnosis is through identification of filarial parasites in blood or hydrocele fluid, however in advanced stages of the disease, patients may be amicrofilaric.[9] Newer immunological techniques may make the diagnosis easier, but such is the strong association between hydrocele and filariasis in endemic countries that the World Health Organization recommends that all hydroceles are presumed to be caused by filariasis unless proved otherwise.

Treatment for lymphatic filariasis traditionally consisted of ivermectin, albendazole, and diethylcarbamazine. This treatment does not kill adult worms and therefore is not effective in reducing the hydrocele. However, the discovery that tetracyclines such as doxycycline can kill the parasites by targeting *Wolbachia*—the symbiotic bacteria living within the worms—has improved management of the condition. Treatment with doxycycline for 6 weeks has been shown to eliminate worm nests from the scrotum of men with filarial hydroceles.[10]

Hydrocelectomy may be required for those that do not respond to drug therapy and may be performed under general, spinal, or local anaesthesia with sedation.[11] Local anaesthesia has advantages in reducing the risks and costs associated with general anaesthesia, and spermatic cord block with local anaesthetic infiltration has been shown to be successful.

Lymphoedema of the limbs may make cannulation and blood pressure monitoring problematic in patients with filariasis.

Brucellosis

Brucellosis is a zoonosis caused by *Brucellae*: Gram-negative coccobacilli, of which three types can infect humans and may be hosted by cattle, pigs, sheep, goats, or camels.[4] The infection is usually acquired by drinking the milk or eating the dairy products of infected animals. Human to human transmission may also occur sexually, via blood transfusions, or breast feeding.

Brucellosis is characterized by recurrent pyrexia and other non-specific symptoms including fatigue, headache, and myalgia. Brucella endocarditis is a rare but potentially fatal complication that occurs in approximately 1% of patients.[12] The infection predominantly affects the aortic valve and may subsequently require valve replacement. The mitral valve may also be affected, particularly if there is pre-existing rheumatic heart disease. If congestive cardiac failure occurs as a result of endocarditis or if the patient is in cardiogenic shock, emergency cardiac surgery may be required, which is associated with significant mortality.

Other subacute localized disease may also occur, most commonly bone or joint involvement causing sacroiliitis or spondylitis, but also central nervous system involvement causing meningitis or spinal abscess. Respiratory involvement is less common but effusions, lung abscesses, and bronchitis may all occur and complicate anaesthesia.[8]

Culture of *Brucella* from blood, pus or other body fluid confirms the diagnosis. Serological tests, such as the serum agglutination test may also detect immunoglobulin G and M levels.[12] Acute brucellosis requires 6 weeks of treatment; however, for those with focal or chronic disease, this extends to 3 months.[4] Single antibiotic therapy is associated with a one third relapse rate, so combination therapy is required. Doxycycline with streptomycin is the traditional treatment of choice, but gentamicin is an alternative.

Ascariasis

Ascaris lumbricoides (roundworms) affects over 800 million people worldwide, most commonly children aged between 3 and 8 years.[4] Ingestion of contaminated vegetables or soil with *Ascaris* eggs leads to release of larvae, which pass through the intestinal mucosa. These enter the bloodstream and lymphatics and pass through the lungs around 4–16 days after the initial infection.

Ascaris pneumonitis causes fever and urticaria as well as wheeze, cough, and dyspnoea. In severe infection, haemoptysis, cyanosis, and chest pain may occur. Loeffler's syndrome occurs when the pneumonitis is associated with eosinophilia. *Ascaris* infection may occasionally cause upper airway obstruction, as the stress of anaesthesia causes migration of worms, and physical obstruction of tracheal tubes or laryngeal mask airways by the worms has been reported.[13]

Intestinal worms do not themselves damage the mucosa nor cause blood loss, but heavy infestations can form a bolus causing obstruction, volvulus or perforation with subsequent peritonitis. Intestinal obstruction is more common in children, whereas biliary obstruction more common in adults, where it may lead to biliary colic, pancreatitis, or liver abscesses.

Treatment is with a single dose of albendazole or a 3-day course of mebendazole, although longer treatment may be required for a heavy infection. Surgery is occasionally required for intestinal obstruction, though it is usually treated conservatively. *Ascaris* pneumonitis is treated symptomatically with bronchodilators and steroids.

Buruli ulcers

Buruli ulcer, caused by *Mycobacterium ulcerans*, is a necrotizing and ulcerating infection of the skin. Mycobacteria are found in soil or stagnant water and are thought to enter the body after a penetrating injury.[4] Ulcers are most commonly seen in children and predominantly affect legs, arms, and other areas exposed to minor trauma, but may affect any area of the body including eyes. The ulcer usually starts as a non-ulcerative lesion, however eventually the skin breaks down causing a painless ulcer with a necrotic centre often with associated satellite lesions. Approximately a quarter of those with a Buruli ulcer develop complications, such as contractures, amputation, or loss of sight.[4]

Culturing a sample from the ulcer is difficult, but DNA identification using the polymerase chain reaction is increasingly being used in endemic areas with sensitivities >90%.[8] Antituberculosis and antileprosy medication is very effective at treating the ulcer, and current guidelines suggest a combination of streptomycin, and rifampicin for 8 weeks. Previously guidelines suggested excising all affected tissue, although this is no longer recommended.[8] Surgery may be required to remove necrotic tissue, or more commonly to repair large defects with skin grafts. Patients should have a minimum of 4 weeks of antibiotic therapy prior to surgery. Surgery and physiotherapy may also be required to help treat contractures.

Noma

Noma, or cancrum oris, is an infectious gangrene of the mouth. It often occurs after an acute infection such as measles or malaria, particularly in malnourished children.[8] It usually starts as a periodontitis, developing into an ulcerative stomatitis, which rapidly progresses into gangrene and necrosis of the bone. If untreated, the condition may rapidly be fatal.

Chlorhexidine mouthwashes can prevent progression of the periodontitis, while high doses of penicillin may be required to treat established infection. However, the functional and cosmetic deformities that remain can be significant and reconstructive surgery may be required. There can be significant airway abnormalities including complete trismus, with consequent difficulties in airway management and intubation.[14] Noma may be considered a disease of the malnourished, and this carries attendant perioperative issues discussed previously in '*Tropical Medicine and Anaesthesia 1*'.

Potential drug interactions

Malnutrition complicates many tropical diseases in resource-poor environments, and the effects of this on drug metabolism are dealt with in '*Tropical Medicine and Anaesthesia 1*'. Therapeutic agents used to treat tropical diseases may also interact with anaesthetic medications, both through induction/inhibition of the cytochrome p450 enzyme family, and through alternative pathways.

Tuberculosis chemotherapy

Rifampicin, a standard tuberculosis (TB) therapeutic agent, is a potent inducer of CYP3A4, which is responsible for the metabolism of alfentanil, fentanyl, midazolam, and lidocaine.[15] The increased metabolism may result in a reduced therapeutic effect or increased generation of potentially toxic metabolites. Isoniazid, conversely, is a CYP3A4 inhibitor although these effects do not predictably cancel each other out. As CYP3A4 may be found in the small intestine, oral drugs tend to be more affected by these drugs than those given intravenously.[15] Generally, all opioid levels are reduced by rifampicin, meaning additional analgesia may be required. Lower plasma concentrations of diclofenac are seen in patients also taking rifampicin, but no such effect is seen with ibuprofen, which may be a preferred option.[16]

Drug-induced hepatitis may occur with isoniazid, rifampicin, or pyrazinamide. Hepatotoxicity usually occurs within the first 2 months of therapy, and is more common in malnourished patients, or those with human immunodeficiency virus (HIV) or viral hepatitis coinfection. Symptomatic hepatitis is associated with mortality of around 5% and elective surgery should be avoided. Isoniazid also induces CYP2E1, which metabolizes halothane, rendering patients on antituberculous therapy at increased risk of halothane hepatitis.[15]

Streptomycin may potentiate the effects of non-depolarizing neuromuscular blockers; however, enzyme induction may increase the metabolism of rocuronium and vecuronium.[16] Careful titration of drugs and monitoring of neuromuscular function is essential.

Some of the adverse effects of the drugs used in TB may also have an impact upon anaesthesia. Rifampicin may cause thrombocytopenia, which may be significant enough to cause spontaneous bleeding, or may limit the choice of anaesthetic technique. Isoniazid may also cause thrombocytopenia, but more commonly causes anaemia or agranulocytosis. Rarely, disseminated intravascular coagulation may occur due to hypersensitivity reactions. Isoniazid may cause a peripheral neuropathy, which may have significance to those undergoing regional anaesthesia.

Antiretrovirals

Protease inhibitors are used to treat HIV as they prevent viral replication. However, they also have an effect on cytochrome CYP3A4. Although all the same class of drug, each protease inhibitor has a different effect on the cytochrome isoenzymes. Ritonavir is the most potent inhibitor of CYP3A4 and shows clinically significant interactions with several anaesthetic drugs.[17] It will reduce the metabolism of fentanyl, pethidine, midazolam, and possibly other opioids.[18] Dexamethasone and thiopentone can reduce protease inhibitor concentration.[19]

In contrast to the protease inhibitors, nevirapine, a non-nucleoside reverse transcriptase inhibitor used combination antiretroviral therapy, induces cytochrome P450 enzymes. Patients on combination therapy may therefore be taking drugs that both inhibit and induce hepatic enzymes leading to an unpredictable response to co-administered anaesthetic agents.

Tenofovir, a nucleotide analogue reverse transcriptase inhibitor, does not interact with drugs through the P450 cytochrome system; however, it is associated with a small but significant risk of kidney injury.[19] Nephrotoxic drugs such as gentamycin or non-steroidal anti-inflammatory drugs should consequently be avoided in the perioperative period.

Antimalarials

In general, antimalarial agents do not complicate anaesthesia to the extent of TB and anti-retroviral medications. However, of note to the anaesthetist, quinine use is associated with cardiac arrhythmias, hypoglycaemia and prolongation of neuromuscular blockade.[20]

Blood transfusions

Most blood in resource-limited tropical settings is transfused as whole blood, and recipients are at increased risk of febrile non-haemolytic transfusion reactions due to the activity of donor leucocytes.[8] Whole blood is also associated with a higher incidence of graft *vs* host disease, transfusion-associated lung injury, and transfusion-associated immunosuppression, which may lead to disease progression in patients with HIV infection.[8]

Many tropical diseases can be transmitted in the transfusion of blood, including viral, parasitic and bacterial infections. This risk is related to the prevalence of the infection in the local blood donating population, and the local blood screening policies and practices.[8] The World Health Organization have issued recommendations on the screening of both potential donors and donated blood for transfusion-transmissible infections.[21] Donated blood should be screened for HIV-1, HIV-2, hepatitis B and C, and syphilis. Screening for other infections such as malaria and Chagas' disease, may be recommended depending on local epidemiology.

However, while blood transfusion carries a risk of infection, this must be set against the risk of untreated end-organ hypoperfusion. In many resource-poor settings the greatest risk to the surgical patient is a lack of access to blood or blood products, due to a complex interplay of factors: cultural reticence to donate, poor storage and supply chains, and poor clinical utilization may all play a role.

Viral infections

When HIV-contaminated blood is transfused the seroconversion rate is approximately 96%, much higher than any other route of transmission. Prior to widespread screening, 10% of all new HIV infections in sub-Saharan Africa were thought to be a result of blood transfusions.[8]

Hepatitis B and C can both be transmitted via transfused blood. Transfusion of unscreened hepatitis B positive blood results in infection of the hepatocytes, with chronic infection leading to cirrhosis and hepatocellular carcinoma. Screening may include testing of serological markers such as surface antigen or core antigen, and viral DNA may also be detected.[21]

Human T-cell lymphotropic virus type 1 (HTLV-1) is a retrovirus able to be transmitted by transfusion with a high seroconversion rate. Infection is associated with adult T-cell leukaemia; however, rarely, it may also cause HTLV-1 associated myelopathy, formerly known as tropical spastic paraparesis. This slowly progressive weakness and spasticity of the lower limbs affects <2% of those infected with HTLV-1. It is possible to screen for specific HTLV-1 antibodies, although may not be routinely undertaken in all endemic countries.

Other viral diseases able to be transmitted through transfused blood include dengue, yellow fever, West Nile virus, chikungunya, and Zika virus. Natural infection with these viruses may be subclinical in the immunocompetent host, but may have significant consequences in an immunocompromised recipient. Chikungunya is a viral infection usually transmitted by mosquitoes that causes fever, arthralgia, and rash. West Nile virus causes a similar clinical picture, but may also cause encephalitis. Yellow fever is usually a mild illness, but in severe disease, jaundice, fulminant hepatic failure, and gastrointestinal bleeding may be seen.[4] Similarly, dengue normally causes a non-specific febrile illness, but can also precipitate a haemorrhagic fever associated with increased vascular permeability, thrombocytopenia, bleeding, and cardiovascular collapse. Other viral infections may have the potential to be transmitted through blood transfusion, but data are scarce and it can be difficult to differentiate between naturally-acquired and transfusion-related infection.[22]

Parasitic infections

Malaria may be transmitted through blood transfusion although, as with viral transmission, it is difficult to distinguish cases from natural infection via a mosquito bite.[8] Screening is advocated, but microscopy will not always detect low level parasitaemia and rapid diagnostic tests using immunochromatographic dipsticks may be more effective. Malaria prophylaxis should be given to all those receiving a blood transfusion or deemed at high risk.[21] In non-endemic countries, donor selection will eliminate all those with a history of malaria or recent travel to a malarial area.

Chagas' disease may be spread through either blood or solid organ donation, as the parasite can survive for at least 10 days in refrigerated citrated blood.[23] It is endemic in areas of Central and South America, and parasitaemic donors may be asymptomatic.[21] In endemic countries, all donated blood should be screened with a highly-sensitive antibody enzyme immunoassay. Elsewhere, donor selection should exclude anyone with a personal history of or risk factors for Chagas disease.

Bacterial infections

Syphilis is caused by *Treponema pallidum*, which may be transmitted through transfusion. It is endemic in most tropical countries and should be screened for, however storage of blood below 20°C for >72 h usually damages the bacteria so that it is no longer infectious.[21]

Brucellosis also has the potential to be transmitted through blood transfusion. Donors in endemic areas may have subclinical *Brucella* bacteraemia, and screening blood donations for this condition is rarely performed.

In many tropical countries, the most common transfusion-related infection is that caused by bacterial contamination of blood products during collection or processing, with contamination rates in Africa up to 2500 times those seen in higher income settings.[22] Gram-negative bacteria tend to be found in whole blood, whereas Gram-positive organisms tend to be found in platelets, whose storage at room temperature promotes the growth of skin flora.

Summary

This series of articles, *Tropical Medicine & Anaesthesia* 1 and 2, has aimed to demonstrate the impact that tropical illness can have on the perioperative patient in terms of pathophysiological complications, surgical presentation, and medication interaction. While all anaesthetists should have an awareness of the impact that endemic tropical illnesses may have on their practice, these articles are also intended to provide an introduction for the anaesthetist who is interested in practising in tropical settings and does not have clinical experience in this area. It should be recognized that the experts in providing anaesthesia to patients with tropical illness are the army of physician and non-physician anaesthetists who live and practice in those geographic locations where these conditions are endemic, often in under-resourced settings. The review of the literature presented needs to be complemented by practical experience learning from those who manage these conditions regularly in their anaesthetic patients.

Declaration of interests

None declared.

MCQs

The associated MCQs (to support CME/CPD activity) can be accessed at www.bjaed.org/cme/home by subscribers to *BJA Education*.

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