

# MEDICAL CONDITIONS REQUIRING INTENSIVE CARE

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## Abstract

Patients who require critical care for internal medical conditions make up a small but significant proportion of those requiring evacuation to the Royal Centre for Defence Medicine in Birmingham, UK. Infectious, autoimmune, neurologic, cardiac and respiratory conditions are all represented. Conditions which preclude military service and which one would not necessarily expect to see in a military hospital are still prevalent in civilian contractors and host nation personnel. With some 250,000 British military personnel based in the UK and overseas individual presentations of rare conditions occur regularly. This article discusses the ITU management of some key conditions. Whilst trauma makes up the majority of the workload in a field Intensive Care Unit, medical admissions happen not infrequently. This article describes some of the most common medical causes for admission and treatment is considered.

## Introduction

The full range of internal medical conditions that may require intensive care admission is too extensive to cover in a brief article. Most conditions are amenable to standard critical care management regimens. Drug regimens are mostly covered by the British National Formulary [1]. The main internal medical conditions that require intensive care support in a deployed military setting do so because of respiratory failure, renal failure, neurological compromise or failure of thermoregulation.

This article covers conditions that are particularly relevant to caring for the deployed population, including neurological, cardiac, respiratory and environmental illnesses. Although renal failure is frequently seen in sepsis, and may occur in other conditions such as following crush injury or burns, it is uncommon for isolated renal failure to present late enough to require intensive care in young adults, and renal failure is therefore not covered further in this article. Sepsis is covered in a separate article. Key references are given at the end of the article.

## Meningitis and encephalitis

These two diseases have a significant incidence and prevalence in developed and developing nations. They carry significant morbidity and mortality [2].

Meningitis and encephalitis are inflammatory conditions affecting the central nervous system. They may be caused by viral or bacterial infection, but also have other causes including vasculitis, granulomatous diseases and other rare aetiologies (Table 1). Because of significant clinical overlap the recognition and work-up may involve investigating broadly for these causes.

## Clinical presentation

Meningitis is characterized by the acute onset of intense headache, fever, nausea, vomiting, photophobia, and neck stiffness. Neurological signs may include neck stiffness, lethargy, delirium, convulsions or coma. Most adult patients have an altered mental state, clinical signs of meningism (e.g., Kernig's sign, Brudzinski's sign), and fever. Elderly patients are prone to less clear presentations with an altered mental state and a prolonged course with fever. Neck stiffness may be absent in infants and disease progression can be more insidious.

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### BACTERIAL CAUSES

Meningococcal (*Neisseria meningitidis*) - type B predominating in UK following introduction of vaccine against Meningitis C  
Pneumococcal (*Streptococcus pneumoniae*)  
Haemophilus Influenzae B (less common following vaccine introduction)  
Listeria monocytogenes - in very young or very old patients  
Mycobacterium tuberculosis - atypical presentations or in immunocompromised patients  
Group B Streptococcus - neonates

### Other organisms

Rickettsial: Rocky Mountain spotted fever, endemic typhus, epidemic typhus, Q fever, human monocytic ehrlichiosis.  
Fungal: cryptococcosis, coccidiomycosis, histoplasmosis, North American blastomycosis, candidiasis.  
Parasitic: African trypanosomiasis, toxoplasmosis, echinococcus, schistosomiasis, primary amoebic meningoencephalitis

### VIRAL CAUSES

Enteroviruses (coxsackie A, B, echovirus)  
Herpes Simplex Virus (HSV) 1 and 2  
Epstein Barr Virus (EBV)  
Varicella (chickenpox)  
Human Herpes Virus 6 - uncommon, similar to EBV clinically  
Measles  
Mumps

### Rare viral causes

Adenovirus, norovirus, rubella, rabies, arenavirus, west nile virus, viral haemorrhagic fevers, dengue.

Table 1. Aetiological agents in meningitis and encephalitis.

Encephalitis is typically marked by the acute onset of a febrile illness. Signs and symptoms of leptomeningeal irritation (e.g. headache, fever, neck stiffness) occur, along with focal neurological signs. In some patients seizures may be the presenting feature. Others present with alteration of consciousness, starting with lethargy and progressing to confusion, stupor, and coma. Behavioural and speech disturbances are common.

The history is crucial in identifying potential viral factors including a thorough travel history and attention to the way the disease has progressed, including which parts of the nervous system have been affected. A list of rarer differential diagnoses is given in Table 2.

- Cerebral Venous Thrombosis, Basilar Artery Thrombosis, Cavernous Sinus Thrombosis.
- Epileptic Encephalopathies.
- Febrile convulsions.
- Primary Human Immunodeficiency Virus Infection.
- Opportunistic infections in the Acquired Immunodeficiency Syndrome (AIDS).
- Confusional states and acute memory disorders.
- Status epilepticus - convulsive or non-convulsive.
- Intracranial haemorrhage.
- Intracranial abscess.
- Aseptic Meningitis.
- Hepatic encephalopathy.
- Acute disseminated encephalomyelitis.
- Subacute sclerosing panencephalitis (SSPE) following measles infection.

Table 2. Differential diagnosis in meningitis and encephalitis - less common causes.

### Diagnosis

The diagnosis of meningitis and encephalitis is made by interpreting the clinical picture in conjunction with laboratory results. Lumbar puncture (LP) is the main diagnostic tool in meningitis and encephalitis. There are numerous anecdotal cases of coning following LP. This risk seems to be greatest in patients who have neurological signs or altered conscious level, and in this setting LP should not be done until after Computed Tomography scanning of the brain (CT). Where CT is not available Lumbar Puncture may be performed in patients who do not have the signs listed in Table 3. The absence of these signs makes an abnormal CT very unlikely [3,4]. Delay in performing an LP should not delay antibiotic/antiviral therapy. Identifying an organism helps to tailor specific treatment, but in many patients either no organism will be identified, or the results will not be available for several days. In such cases general supportive measures and “best guess” antimicrobials are indicated.

- Signs of raised intracranial pressure with changing level of consciousness.
- Focal neurological signs, abnormal posturing, abnormal pupil reactions, gaze deviation, or papilloedema.
- Respiratory or cardiovascular compromise with impaired peripheral perfusion or hypotension, or hypertension with bradycardia.
- Thrombocytopenia or coagulopathy.
- Significant skin lesions or purpura over site of LP.
- Recent seizure, or prolonged seizures.
- Inability to correctly answer two consecutive questions or follow two consecutive commands.

Table 3. Contraindications to Lumbar Puncture (LP).

Cerebrospinal fluid is sent for microbiology tests including microscopy, gram stain, culture and sensitivities. CSF can also be sent for protein and glucose, along with a contemporaneous blood glucose (Table 4). Where practicable, CSF should also be sent for Polymerase Chain Reaction (PCR) investigations to

complement any blood PCR specimens. PCR may identify viral DNA in the CSF, aiding identification of the causative organism. Unfortunately, even in the UK PCR must be done in a regional reference lab, and takes a significant period of time to be done. CSF for PCR must either be frozen to -20°C or kept at 4°C during transport to the reference lab. This may be impractical in a deployed field hospital. Cytologic analysis of CSF is standard in developed countries, and may reveal neoplasia. In the deployed setting a manual differential white cell count may be available on the CSF.

### Results

Opening pressure for CSF can be raised >14 cm H<sub>2</sub>O in meningitis. Glucose interpretation requires paired CSF and plasma glucose measurement. A low CSF to blood glucose ratio is < 0.31.

	Bacterial	Viral
Appearance	Turbid	Clear
Cells (mm <sup>3</sup> )	5-2000	5-500
Main type	Neutrophil	Lymphocyte
Glucose (mM)	Very low	Normal
Protein (g/l)	>1	0.5-0.9

Table 4. CSF findings in Meningitis.

When CT scanning of the brain is available it may demonstrate low density lesions in the temporal lobes, and increase confidence in a clinical diagnosis of encephalitis. It does not allow reliable discrimination between different viral aetiologies. Important complications such as haemorrhage, oedema and herniation are clearly demonstrated on CT and may guide neurosurgical intervention. CT later in the disease course may identify post-infectious complications such as secondary haemorrhage, abscess or hydrocephalus.

Magnetic Resonance Imaging (MRI) and electroencephalography can aid diagnosis but are not available in the Field Hospital, and may not show changes early in the disease process.

### Management

Management of meningitis follows the familiar ABC paradigm. Airway management with intubation and ventilation in the obtunded patient takes priority over other interventions. Oxygen should be administered, and measures taken to prevent hypoxia and hypotension, which are potent causes of secondary brain injury. Patients who have only confusion or mild reductions in conscious level may deteriorate quickly, and should be managed in a high dependency setting.

Early antibiotic therapy is the cornerstone of treatment for meningitis, and should be given immediately by the first doctor to suspect the disease, in the prehospital setting if necessary.

Early antibiotic administration saves lives in meningitis!

The choice of antibiotics depends on local resistance patterns, but typically benzylpenicillin (IM) is given prehospital and is available at role 1. Cefotaxime is the drug of choice in hospital. In the very young or old, and where atypical organisms such as listeria are suspected, antibiotic therapy may differ, and should be guided by the BNF. Antiviral treatment is added where a viral etiology is suspected. This will typically be aciclovir, although there may be a role for ribavirin in arenavirus infection (i.e. Lassa fever).

Steroid administration reduces the incidence of death and of deafness. Dexamethasone should be given with or before the first dose of antibiotic, unless the patient has overt sepsis. The complications of meningitis are listed in Table 5.

Coning.  
Epilepsy.  
Cerebral venous sinus thrombosis.  
Cerebral abscess.  
Infection at other sites, including skin, joints or lung.  
Deafness.  
Communicating Hydrocephalus.  
Subacute Sclerosing Panencephalitis (following measles infection).

Table 5. Complications of Meningitis that may occur in up to 30% of survivors.

### Contact tracing and treatment

Chemoprophylaxis is often offered to close contacts of the patient. Health workers who have had droplet exposure are also offered treatment. Usually either ciprofloxacin or rifampicin is given, dependent on local policy. The Army Public Health department should be informed of any case of suspected meningitis. Soldiers living in close proximity, for example at forward operating bases should be considered for prophylaxis. This also applies to soldiers sharing the same accommodation or who have recently travelled within the same armoured vehicle.

### Other Neurological Conditions

There is an almost unending list of conditions that may cause altered sensorium or neurological compromise in previously young fit adults. Intracranial haemorrhage may occur from spontaneous rupture of arteriovenous malformations or aneurysms. Cerebral venous sinus thrombosis affects previously well patients, usually women with antiphospholipid antibodies, Factor V Leiden, or other thrombophilias. Vasculitides such as Systemic Lupus Erythematosus, may affect the brain. The mainstay of management of these conditions is protection of cerebral oxygenation with ventilatory support if necessary, maintenance of cerebral perfusion and early evacuation. Conditions such as norovirus gastroenteritis which would not be expected to produce neurologic compromise in the UK may do so in the harsh environment in which military operations occur.

Patients with Guillan-Barré syndrome (GBS) is the most common acute polyradiculopathy. It presents with progressive motor weakness and areflexia (often following a prodromal illness), and may rapidly progress to autonomic failure and respiratory failure requiring intubation. Miller-Fisher Syndrome is a GBS variant that affects the cranial nerves, with ophthalmoplegia, loss of reflexes and ataxia. It should be noted that these patients often need ventilating for weeks, so a plan for early evacuation to an alternate facility needs to be made to maintain the operational efficiency of the Field Hospital ICU.

For patients whose neurologic condition progresses to the point where raised intracranial pressure presents a risk to their life it is unclear whether neurosurgical intervention in the form of decompressive hemicraniectomy is effective. Anecdotal cases of successful neurosurgical management of such patients exist in the literature, and the authors have recently seen a civilian patient with cerebral venous sinus thrombosis who had persistently raised ICP, and made a good recovery after decompressive craniectomy. Early evacuation to a neurosurgeon is vital.

### Acute Ischaemic Stroke

Fortunately acute ischaemic stroke is rare in young adults. However, when a patient presents to the field hospital with symptoms and signs consistent with acute ischaemic stroke consideration should be given to thrombolysis. Thrombolysis is of most benefit in patients with more severe strokes. Clinical assessment and neuroimaging must be completed prior to

administration of thrombolysis, and within three hours of onset of symptoms.

Aspirin should be given to all stroke patients once CT has excluded intracranial haemorrhage. Early evacuation should be arranged.

Note that the differential diagnosis of ischaemic stroke includes conditions as diverse as migrainous hemiplegia, factitious weakness, and cerebral venous sinus thrombosis. Early assessment by a clinician with expertise in internal medicine or emergency medicine is warranted.

## Myocardial Infarction and Cardiac Arrest

### Clinical Presentation

The clinical presentation of MI is classically of crushing central chest pain which radiates to the neck or arm. Sweating, dyspnoea, or nausea are common. Some patients, especially women, may present with back or abdominal pain.

### Diagnosis

Diagnosis is based on a history of chest pain consistent with myocardial infarction, along with appropriate ECG findings and raised cardiac enzymes or Troponin, which is now available in the field.

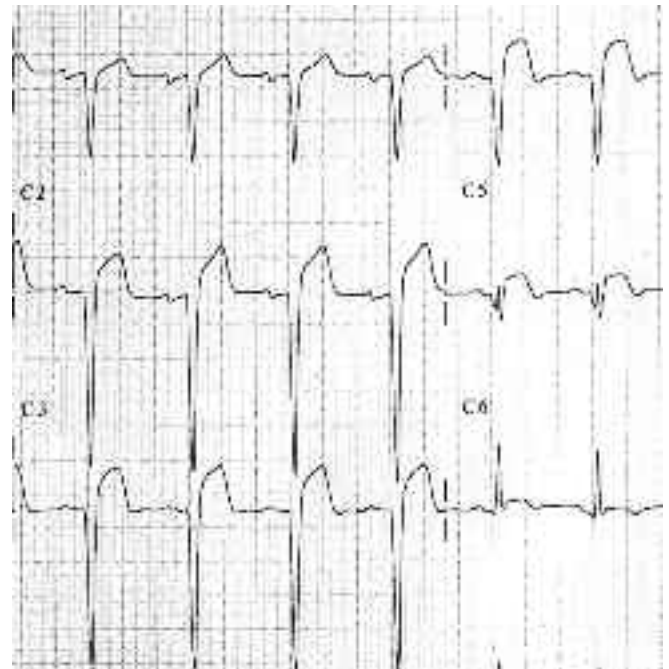


Figure 1. Anterior MI with ST-elevation in anteroseptal leads.

In the patient with a cardiac arrest who presents following successful resuscitation but does not regain consciousness the ECG may indicate acute myocardial infarction. It should be remembered that neurological conditions such as subarachnoid haemorrhage may cause ECG changes that mimic MI, so neuroimaging is required if the patient remains unconscious to rule out an intracranial catastrophe [5].

### Management

The ideal treatment for MI is angioplasty. This is not available in the field hospital. Thrombolysis is a good alternative to angioplasty, as long as it is given as soon as possible. Thrombolysis is indicated in patients with myocardial infarction and ST-elevation or new left bundle-branch block where no contraindications exist. Antiplatelet therapy with aspirin and clopidogrel should be instituted, and consideration given to administering an intravenous beta-blocker and an oral statin. This may be all that is required, and the patient may then be admitted to ITU (in the absence of a Coronary Care Unit).

Evacuation should probably be delayed for at least 24 hours until the risk of cardiac arrhythmia has begun to decline. The exception to this is where thrombolysis is unsuccessful, or only partially successful. Cardiogenic shock requires urgent revascularisation, the only treatment which significantly improves survival. Where possible patients should be urgently evacuated to the nearest facility with the ability to perform percutaneous coronary intervention and coronary artery bypass grafting. Milder forms of left ventricular failure may respond to mask CPAP, which is not yet available in the field.

### Management after cardiac arrest

Patients arriving in the ED with primary cardiac arrest in asystole are extremely unlikely to survive if the duration of asystole exceeds 8 minutes. The exception to this rule is that patients with hypothermia or drowning may occasionally survive.

Patients who survive an out of hospital cardiac arrest and do not immediately regain consciousness should be managed with therapeutic hypothermia. This intervention improves outcome with a number needed to treat of 6 to produce one extra survivor. The patient should be cooled to 32-34°C as quickly as possible after arrival in the ED. Cooling should be maintained for 12-24 hours. The best method of cooling is not yet determined, and civilian hospitals use various modalities, from packing the patient's axillae, groins, and neck with bags of ice, placing wet sheets over them and blowing a fan over to increase evaporative cooling, to using sophisticated circulating fluid devices either externally or intravascularly using modified central venous catheters. Cooling is not easy to do successfully and post arrest patients often develop hyperpyrexia, by unknown mechanisms. Shivering frequently occurs in these patients, and neuromuscular blocking agents may be used to prevent shivering. Therefore it should be carried out on ICUs with the proper equipment, using patient feed back loops as part of the machine's monitoring.

Prognostication, i.e. predicting outcome, in patients who have an out of hospital cardiac arrest is difficult and is not accurate before the 24 hour point. Poor prognostic indicators at 24 hours include absent corneal or pupillary responses; absent withdrawal response to pain or absent motor response [6].

Where there is a reasonable expectation of recovery these patients should be evacuated to an appropriate facility.

## Severe Acute Asthma

### Presentation

It is extremely unusual for acute severe asthma to present without a preexisting history of asthma. Because asthma is a bar to military entry, severe forms of the disease are relatively uncommon in the deployed military setting. However there are a small number of patients who develop asthma after joining the military. These patients, who typically have well controlled asthma, may develop exacerbations spontaneously, due to viral infection, or in response to environmental conditions.

### Diagnosis

Diagnostic features of severe acute asthma and life threatening asthma are given in Tables 6a & 6b.

Inability to complete a sentence with one breath.  
Tachypnoea > 25 breaths per minute.  
Tachycardia > 110 beats per minute.  
SpO<sub>2</sub> < 92%  
Peak Expiratory Flow Rate 33% -50% of predicted or best.

Table 6a. Features of Severe Acute Asthma.

Silent chest, feeble respiratory effort or cyanosis.

Hypotension, bradycardia, dysrhythmia, exhaustion, agitation, confusion, altered conscious level.

SpO<sub>2</sub> < 92%

PEFR < 33% predicted or best.

Table 6b. Life Threatening Asthma.

Recognition of the life-threatening nature of this condition is very important. Arterial blood gases should be taken. Hypoxia or an inappropriately normal or high PCO<sub>2</sub> may indicate impending respiratory arrest. A chest radiograph should be obtained to look for some of the conditions which may mimic severe asthma, such as cardiogenic or non-cardiogenic pulmonary oedema, or pneumonia, and other conditions such as pulmonary embolism and vocal cord dysfunction should also be considered.

### Management

Repeated nebulised salbutamol should be given along with 100% oxygen by a non-rebreathing reservoir mask. Nebulised ipratropium bromide should also be given. Intravenous steroids in the form of hydrocortisone 100 mg 6 hourly should be given. Intravenous magnesium does not reduce mortality or the need for ITU admission in trials, but it does improve spirometry in the acute severe setting. Long experience with magnesium sulphate in treating other conditions suggests it is safe, so magnesium sulphate 2 grams should be given over 20 minutes to avoid the main side effects of hypotension or flushing. Intravenous aminophylline has been shown to be ineffective in adult patients, and probably adds little but the risk of nausea and vomiting or arrhythmias, so should be avoided. Likewise intravenous salbutamol has not been shown to be any more effective than nebulised in adults, although it is helpful in children.

All attempts to manage the patient non invasively should be made, as invasive ventilation in asthmatic patients is notoriously difficult. However a third or so of patients will require ventilation, here a strategy that utilises a short inspiratory and long expiratory time is required. As the major resistance is to the passive expiratory gas flow, this should reduce the amount of auto-PEEP or dynamic hyperinflation that the patient develops. A plateau pressure of less than 30 cm H<sub>2</sub>O should also be targeted. It may therefore be necessary to allow a degree of permissive hypercapnia. Where a raised PCO<sub>2</sub> develops gradually, it is usually well tolerated by the patient [7]. Bicarbonate may be administered if required to reduce the degree of acidosis. Other approaches such as Ketamine infusions and inhaled volatile anaesthetics are anecdotally effective in reducing bronchospasm, but have not been demonstrated to be effective in randomised clinical trials. Where possible prolonged use of neuromuscular blocking agents (in a group of patients receiving high doses of steroids) should be avoided to minimise the risk of critical illness myopathy/neuropathy.

## Pneumonia including Influenza/Viral (Chickenpox)

### Presentation

Patients with pneumonia may present with the gradual onset of a febrile illness, or may develop sudden onset pleuritic chest pain associated with cough. Dyspnoea may develop gradually, or may be rapidly progressive. There may be a dry cough, for example in atypical and viral pneumonia, or the more usual productive cough with green or bloodstained sputum. It should be noted that some patients will present with non-specific symptoms such as vomiting or headache. Patients may progress rapidly to overt sepsis with respiratory failure.

## Diagnosis

By necessity pneumonia is often diagnosed clinically, despite the fact that clinical diagnosis of pneumonia is inaccurate. Where crackles are present on auscultation there may well be consolidation, however clinical signs of consolidation are only present in about a third of patients with radiological consolidation. The diagnosis of pneumonia requires evidence of consolidation on the chest radiograph (Figure 2). In atypical pneumonias consolidation may lag behind the clinical presentation by a few days.

Scoring systems such as CURB-65 (Table 7) have been validated in civilian populations with mean ages in the 70s, well above that of a military cohort. Accordingly their utility in military populations is unknown, and it may be that young, fit patients develop significant physiological derangement at a lower score than a civilian population.

Confusion - new mental confusion.

Urea greater than 7.

Respiratory Rate > 30 per minute.

Blood Pressure less than 90 mmHg systolic or 60 mmHg diastolic.

65 - age greater than 65.

Patients with a CURB-65 score of 0 or 1 are at a low risk of death, and in the civilian setting may be managed as outpatients. A score of 2 denotes a raised risk of death, and a score of 3 a high risk of death, informing management decisions.

Table 7. CURB-65 score for pneumonia. It predicts risk of poor outcome - score one point for each feature present.

## Management

Patients with pneumonia who require intensive care typically do so due to respiratory failure or sepsis. The mainstay of treatment is early antibiotic therapy and adequate fluid resuscitation, as well as an awareness of the early complications that may accompany pneumonia. These include failure of initial antibiotic therapy, infection with a resistant organism, cavitation, metastatic infection and empyema.

It should be remembered that viral pneumonia may occur including following influenza infection. Some viral pneumonias are relatively mild, but others, such as varicella (chickenpox) (Figure 3) may be severe, even in immunocompetent adults, and may require ventilatory support.



Figure 2. Viral pneumonia secondary to varicella infection in an immunocompetent adult with no comorbidities.



Figure 3. Varicella infection.

Patients with very high FIO<sub>2</sub> and ventilatory support requirements comprise a particular problem for aeromedical evacuation. A clinical decision on whether they are likely to improve in the operational environment or whether they should be immediately repatriated before they deteriorate further is required. Specialist ventilatory modalities such as high frequency oscillatory ventilation (HFOV) and extracorporeal membrane oxygenation (ECMO) are not available in the field hospital. HFOV has been shown to be non-inferior to conventional ventilation, and as yet unpublished results from the CAESAR trial of ECMO suggest it may be effective, however their superiority to conventional ventilation is still not proven.

## Heat Stroke

### Presentation

Heat stroke may be exertional or non-exertional, and typically presents on a spectrum from confusion, through prostration, to status epilepticus in a patient who has been exposed to a hot environment. It may also occur when the patient is exposed to heat stress, for example carrying out vigorous exercise while fully clothed in an environment with a moderate ambient temperature. It is believed that some of the variation in susceptibility to heat stroke is genetically determined.

### Diagnosis

Heat stroke is diagnosed when a patient presents with a combination of a core temperature greater than 40°C and encephalopathy. It is a condition characterised by the systemic inflammatory response syndrome (SIRS). An elevated heart rate and respiratory rate are found. Many patients develop severe rhabdomyolysis. As the condition progresses disseminated intravascular coagulation, along with dysfunction of numerous organs occurs. Even with treatment this may progress to death [8].

## Management

The mainstay of initial treatment is rapid cooling, which may be accomplished by placing ice packs in the patient's axillae and groins. Alternatively tepid sponging or the application of wet sheets and the use of a fan can help to control the hyperthermia. Other features such as respiratory failure and seizures are managed as for other conditions. Where there is a possibility of rhabdomyolysis adequate fluid administration to drive a diuresis is imperative.

## Hypothermia

### Presentation

Hypothermia occurs in patients who are exposed to the elements when the ambient temperature is low. Severe hypothermia may occur rapidly in patients who have been immersed in water or suffered a near drowning. High altitude environments with hypobaric hypoxic conditions seem to predispose to hypothermia.

### Diagnosis

Hypothermia should be considered in any patient with a relevant exposure, particularly if there has been immersion in water. Core temperature should be measured using a low reading rectal thermometer, or other appropriate device.

### Management

Patients with hypothermia and a core temperature of less than 27°C are at risk of cardiac arrhythmia and arrest. In a large UK teaching hospital such as our institution such patients would be considered for arteriovenous cardiopulmonary bypass to rapidly rewarm them. In military practice these patients will require active warming using forced air blankets, warmed fluids and possibly warm bladder lavage. It is possible to lavage other body cavities such as the pleural cavity, using two intercostal drains, but in UK practice this is almost never carried out. Remember that careful handling of hypothermic patients is required. The sudden jolt of transfer from the ambulance stretcher to a trolley or bed can precipitate cardiac arrhythmia and arrest.

Patients who have suffered a cardiac arrest whilst hypothermic may occasionally make a complete or near complete neurologic recovery. This applies particularly to children, but may also occur in fit adults [9]. However standard Advanced Life Support guidelines need a degree of modification: early tracheal intubation is advocated as the resuscitation attempt is likely to be prolonged, and the interval between giving drugs is doubled as metabolism is slowed. It is also thought that defibrillation is ineffective if the core temperature is under 28 degrees, therefore shocks should be limited until the temperature has risen above this.

## Methanol and Ethylene Glycol Poisoning

### Presentation

Methanol poisoning typically occurs in host nation patients, who ingest methanol for its intoxicating effects, methanol poisoning may present as visual disturbance, abdominal pain and gastrointestinal distress. Ethylene Glycol is found in antifreeze

and is poisonous if ingested. It has similar clinical effects to methanol but also causes kidney failure.

### Diagnosis

Metabolic acidosis with a raised anion gap, raised osmolar gap and raised lactate are the hallmarks of methanol poisoning. Blindness, respiratory failure, seizures, and eventually death may occur.

Ethylene Glycol is converted to various compounds, including oxalate crystals. These are toxic to various organs. A high anion-gap metabolic acidosis and high osmolar gap are characteristic. Hypocalcaemia occurs as body calcium combines with oxalate. Weakness, myoclonic jerks and tetany may occur. Renal failure and pulmonary oedema are also features of this poisoning. Urine microscopy may show oxalate crystals, and the urine may fluoresce under UV light if the antifreeze contains fluorescent dye.

### Management

Treatment is supportive for the neurological and renal manifestations. The alcohol dehydrogenase enzyme system converts methanol to formaldehyde, which gives the toxic effects. Administration of ethanol saturates this system, allowing metabolism and excretion of methanol by other routes. As a gold standard the amount of ethanol given should be guided by frequent blood level estimations, which can prove difficult, however a novel antidote now exists in the form of fomepizole. Fomepizole inhibits alcohol dehydrogenase and is effective both in the treatment of methanol poisoning and in ethanol glycol poisoning [10].

### Conclusions

The medical conditions seen in the Field Hospital Intensive Care Units are not dissimilar to those seen in UK practice. However as the patients tend to be younger and fitter we can expect to see a higher survival. Some treatment regimes need slight modification in view of the logistics of being on operations.

### Further Reading

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