The anaesthetist plays an important role in the multidisciplinary team management of patients with major burns which should occur in specialised regional units. All anaesthetists need to be familiar with the principles of anaesthesia for these patients as they may be required to care for a major burn in an emergency or to provide care for minor burns outside the regional centres. Initial resuscitation and stabilisation has already been covered previously in this journal (see key references). This article deals with the preparation of burns patients for theatre and peri-operative anaesthetic management.

Pathophysiological changes

Airway

The upper airway may be compromised. Acutely, thermal injury leads to progressive swelling of the soft tissues and potential airway obstruction. This swelling may persist for several days and may be complicated by scarring and contractures. The lower airway is rarely burned by exposure to heat unless substances with a very high specific heat capacity are inhaled, such as superheated steam. The lower airway is injured by inhalation of smoke, leading to inflammation, mucosal sloughing, airway irritability and activation of the systemic inflammatory response syndrome (SIRS). The acute consequences of this in severe smoke inhalation include development of excess pulmonary secretions, bronchospasm and the acute respiratory distress syndrome (ARDS).

The constituents of the smoke also determine the degree of airway injury and consequent hypoxaemia. For example, products of combustion of certain household commodities such as PVC, Teflon and polyurethane are particularly toxic as they contain chemicals such as hydrogen chloride, phosgene, hydrogen cyanide and isocyanate. In addition to the hypoxaemia resulting from airway damage, many of these compounds have toxic effects on the cellular respiratory chain, further aggravating the hypoxaemia. Even when the effects have resolved, increased airway reactivity may persist for several months after the injury.

Circulation

Burns have a direct local effect on blood vessel integrity and function; there are also widespread effects on capillary permeability and blood flow. In the area of full thickness burns, vessels will be thrombosed or destroyed. There is a localised increase in tissue capillary permeability as a direct result of thermal injury. In addition, activation of the systemic inflammatory response causes a widespread increase in vascular permeability with generalised oedema. Mediators involved in the vascular response to burn injury include histamine, prostaglandins PGE2 and PGI2, leukotrienes LB4 and LD2, thromboxane A2, interleukin-6, catecholamines, oxygen free radicals, platelet aggregation factor, angiotensin II and vasopressin. Although bradykinin and serotonin levels are increased in the immediate post-burn period, their antagonists have not been demonstrated to reduce consequent oedema. Inhalation injury, hypo-albuminaemia and sepsis exacerbate this increase in vascular permeability. Patients require fluid resuscitation to allow for these losses but invariably, in the early stages after the burn, patients have relative hypovolaemia and decreased tissue perfusion. Later, the patient develops a cardiovascular picture of high cardiac output and vasodilatation due to sepsis or SIRS. As a result of the cardiovascular changes, there may be impaired renal perfusion and function.

Muscle

The muscle underlying any burn may be damaged, but this is much more likely following electrocution which may cause myoglobin release and further renal damage. More widespread
muscle conformational changes gradually develop after injury with proliferation of acetylcholine receptors which has consequences for neuromuscular junction function.

Myocardial depression occurs as a result of circulating factors which have been shown to have direct negative inotropic effects on in vitro myocardial preparations. The exact mechanism of action is unknown, but free radical scavengers such as super-oxide dismutase have been shown to increase myocardial contractility following burn injury, suggesting oxygen free radicals may play a role. Following an electrical burn, cardiac muscle may be damaged, increasing the risk of myocardial dysfunction and dysrhythmias.

Pharmacology

Large fluid shifts, changes in compartment sizes and increase in metabolic rate alter the pharmacokinetics of many drugs. Low albumin leads to an increased free fraction of acidic drugs such as sulphonylureas or anticonvulsants. Raised fibrinogen and α1-acid glycoprotein will reduce the free fraction of basic drugs (e.g. local anaesthetics, propranolol and muscle relaxants). As most laboratories measure total drug concentrations rather than free fractions, serum concentrations may be misleading. These effects, in combination with altered receptor populations and pharmacodynamics, significantly alter the dose requirements and effects of many anaesthetic drugs.

Requirements for anaesthesia

The anaesthetist has a number of skills that may be required in the care of the burn patient. In the initial resuscitation, airway assessment, intubation, vascular access, fluid resuscitation, administration of analgesia and intensive care may all be required. General anaesthesia may be required for intubation, emergency tracheotomy, escharotomy and urgent surgery for other injuries.

There is an increasing trend towards early definitive surgery to the burn wound, both as a means of improving cosmetic result and as a way of removing necrotic tissue, thereby reducing the ongoing stimulus for SIRS. As a consequence, patients may require anaesthesia as soon as they are fully resuscitated. From this point, the patient may require multiple procedures over many days and weeks. Subsequently, reconstructive surgery may be performed over several years.

Choice of agents

Induction and maintenance of anaesthesia

Ketamine 1–2 mg kg⁻¹ has traditionally been advocated as the induction anaesthetic of choice. The combination of its analgesic effects, sympathetic stimulation and maintenance of airway reflex-
acetylcholine receptors. The earliest described post-burn hyperkalaemic response is at 9 days and the earliest cardiac arrest at 21 days. The exaggerated response persists for up to 10 weeks, though some argue that succinylcholine should be avoided for 1 year post-injury.

Burns patients also demonstrate a resistance to non-depolarising muscle blockade with the ED50 of vecuronium in one study being 3 times that of controls. This resistance develops by 1 week and usually persists for 8 weeks; though resistance to metocurine has been described 463 days post-burn. It can only be partially explained by pharmacokinetic mechanisms; acetylcholine receptor proliferation may be responsible for some of this resistance.

Other drugs

Epinephrine solutions ranging in concentration from 1:1000 to 1:500,000 are applied topically or infiltrated subcutaneously to reduce blood loss at excision and donor sites. Resting plasma catecholamine concentrations are elevated post-burn, but the systemic absorption of epinephrine does not seem to be associated with significant cardiovascular side effects.

Local anaesthetics applied topically are used for burn excision and donor site analgesia. The use of EMLA alone has been described for graft harvest in debilitated patients with burns of < 10% total body surface area. Lidocaine 2% sprayed onto the harvest site has been shown to reduce opioid requirements in the 24 h post-surgery when compared with a placebo or 0.5% bupivacaine group. Toxic concentrations were not seen in either the lidocaine or bupivacaine group. Increased concentrations of α1-acid glycoprotein seen post-burn would be protective.

Burns patients are particularly prone to infection due to the loss of the skin’s barrier function. Randomised controlled trials have failed to show any benefit of prophylactic antibiotics and their use merely promotes resistant strains of bacteria. Dose requirements of aminoglycosides, cephalosporins and β-lactams are altered due to their increased clearance. Plasma concentrations should be monitored and doses adjusted accordingly.

Monitoring

Monitoring should routinely include ECG, arterial haemoglobin oxygen saturation, respiratory gas analysis, central temperature, urine output and blood pressure. Some of the difficulties relating to monitoring are summarised in Table 1.

Pre-operative preparation and conduct of anaesthesia

Fasting

Burns patients are hypermetabolic. Repeated fasting required for surgical procedures may interfere with nutritional goals with associated increase in wound infection rate and muscle catabolism. It has been shown that patients only receive 15% of their nutritional needs on the day of surgery with a pre-operative fast of 4 h and 30% if the fasting time is reduced to 1 h. No aspiration was seen in either group suggesting that fasting guidelines should be modified in burns patients. Intubated patients can be fed intra-operatively.

Transport to theatre

There is considerable debate regarding the location of the theatre dealing with burns. A theatre in the burns unit minimizes patient transport distances, but has the risks associated with an isolated site. On the other hand, if surgery is taking place in the main theatre suite, there may be logistical issues concerning the
transport of very unstable patients. Patients receiving intensive care should be transferred with full mobile facilities irrespective of the location of the operating theatre.

Airway

Concerns about airway patency have been referred to above and should have been addressed at the initial resuscitation phase. However, oedema may be present even in the absence of overt airway injury. The anaesthetist should, therefore, be mindful of any airway compromise and, if there is any doubt, an awake fibre-optic intubation or gaseous induction should be carried out. Theoretically, because of the increased cardiac output occurring post-burn, a gas induction should take longer than usual. However, in practice, this is not seen. Intubation secures the airway and allows bronchoscopic evaluation of airway injury. Patients already intubated should have the tube position checked and secured prior to the commencement of surgery. Concerns about airway patency in the later stages of burn injury are related to fibrosis and contractures making airway manipulation and intubation difficult or impossible. Fibre-optic intubation may be required. The successful use of the laryngeal mask airway has been described in both adults and children.

Patients with significant airway injury or a large burn will need artificial ventilation in theatre. They require a greater than normal minute volume as the basal metabolic rate is elevated and gas exchange may be compromised. Because of increased dead-space, end-tidal CO₂ measurements may not reflect arterial CO₂ tension and arterial gas analysis may be indicated. An intensive care ventilator may be required to deliver adequate minute ventilation and provide PEEP and advanced ventilatory modes. Following major burns, smoke inhalation and the subsequent development of ARDS, these patients may be very dependent on PEEP. If there has been extensive blood loss and replacement, delayed extubation may be preferred.

Fluid and temperature homeostasis

A patient should only be taken to theatre when fluid resuscitation is adequate (as evidenced by haemodynamic parameters and urine output) and in the absence of hypothermia. Wound debridement involves significant blood loss. It has been estimated that for every 1% body surface excised, 3–4% of the circulating blood volume may be lost. There should be adequate availability of red cells and clotting factors. Due to damage of the natural skin barrier, evaporative losses are raised, further increasing fluid requirements. Good wide bore i.v. access is essential. Depending on the size and position of the burn, traditional i.v. access sites may be unavailable and unusual peripheral venous sites or central venous access are frequently required. Care of the available veins is a priority and all i.v. access should be performed by experienced personnel.

The combination of large fluid requirements and extensive patient exposure predisposes to intra-operative hypothermia which has undesirable effects on coagulation, cardiorespiratory function and drug handling. It has been shown to be associated with worse outcome in burns patients and should, therefore, be avoided. Central temperature should be measured and maintained and there should be adequate provision of warmed rapid infusion systems. If possible, forced warm air blankets should be used but these may be of limited efficacy due to the degree of patient exposure required. It is important that the theatre environment is at a thermoneutral temperature (about 30°C for these patients), although this may be uncomfortable for theatre staff.

In the reconstructive phase of surgery, the viability of skin grafts and tissue flaps is improved if the patient is warm and hyperdynamic. Careful attention should be paid to fluid status and maintenance of temperature using the measures outlined above.

Patient position

In both the acute and reconstructive phase of burn surgery, meticulous attention should be given to patient positioning as many procedures are prolonged. Patients are often required to be placed in the prone position or moved intra-operatively.

Regional anaesthesia

Despite being of use in minor burns, regional anaesthesia is not beneficial in major burns. The reasons for this are: (i) it can be difficult to block a sufficient area; (ii) vasodilatation associated with epidural blockade accentuates the hypotensive effects of blood loss during surgery; and (iii) the loss of the normal skin barrier leads to transient intra-operative bacteraemia which may colonise an epidural catheter.

Key references


Yowler CJ. Recent advances in burn care. Curr Opin Anaesth 2001; 14: 251–5

See multiple choice questions 111–113.