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Review

The Burning Truth(s)

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Abstract

A severe burn is arguably the most significant injury that the human body can sustain. No other form of trauma causes the massive physiological changes seen in a patient with a major burn injury. This is true in the first few weeks as well as up to two years post-burn, due to the persistence of the hypermetabolic response.[1] Both the profound alterations in physiology as well as numerous anatomical changes make anaesthesia of the severely burned patient complex and challenging.

Introduction

Burns surgery has traditionally been somewhat of a "Cinderella" subspecialty, with the burn surgeon regularly being faced with significant physical and emotional demands. In addition, this branch of surgery has neither complicated surgical procedures nor a plethora of technological equipment to pique the interest of surgical registrars. Surgical procedures in acute burns can be broadly divided into four groups: ablative (tangential or fascial excision of slough; wound debridement; amputations), reconstructive (skin grafting; flaps), decompressive (fasciotomy; laparotomy for abdominal compartment syndrome), and supportive (tracheostomy; gastrostomy). Often overlooked is that fact that in order to ensure good outcome for a severely burned patient, meticulous attention to detail is essential. The same is true for burns anaesthesia.

Pathophysiology of burns

Skin is the largest organ in the body and has multiple vital functions. It provides thermoregulation, protection from the environment, homeostasis of fluid and electrolytes; plays a significant role in metabolism as well as in the immune system; and is the sensory and psychosocial interface between the human organism and the outside world.[2] All these functions are negatively affected by a major burn wound. Anatomically, the skin is comprised of the epidermis and dermis, both of which are injured to a greater or lesser degree depending on the temperature and duration of contact of the heat source.

The pathophysiology caused by a severe burn is complicated and involves changes in all organ systems. A good understanding of these changes is crucial when anaesthetising a severely burned patient.[3] An overview of some of these changes follows, but for completeness the reader is advised to refer to textbooks and articles that discuss this topic more extensively.

Burn shock

At the core of burn pathophysiology is the concept of "burn shock" - a state of dysfunction of the heart and lungs, large blood vessels, and micro-circulation which is not reversed by adequate fluid resuscitation.[4] This dysfunctional circulatory state is the product of burn-induced local tissue trauma and systemic hypovolaemia causing inflammatory mediators to be produced and released both locally and into the systemic circulation. The well-known, Landis-Starling equation, describes the physical forces that regulate fluid movement across the walls of the microvasculature. In a severe burn, all the variables in the equation change markedly to increase fluid movement from the vascular to extravascular compartment. The interstitial pressure and intravascular oncotic pressures decrease while the microvascular permeability, capillary hydrostatic pressure and interstitial oncotic pressures all increase.

Intravascular depletion

The netto effect is intravascular depletion with significant tissue oedema due to extravasation of plasma into the interstitium. Fluid replacement will worsen the oedema, but is essential to maintain intravascular volume. Fluid requirements in the acute resuscitation phase (first 24 hours) are traditionally calculated by using the Parkland's formula: 2-4 ml x body weight x total body surface area (TBSA) burned. Half of this total is given in the first 8 hours post-burn and half in the following 16 hours.[5] This formula should be used as a guide to fluid replacement rather than a prescriptive specification. More important than the predicted volume requirement is the individual patient's metabolic response to resuscitation - urine output, serum lactate and central venous oxygen saturation are useful indicators. Fluid resuscitation should be adjusted on an hourly basis according to these parameters during the first 24 hours following the burn injury.

Haemodynamic changes

Burn shock causes haemodynamic changes mimicking those due to acute haemorrhage, with the notable exceptions of haemoglobin and haematocrit levels. Both of these increase in burn shock. As in haemorrhagic shock; plasma volume, cardiac and urinary output will decrease and systemic vascular resistance increase. Depression of cardiac output is thought to be due to a combination of reduced venous return and increased afterload as well as circulating chemical mediators causing myocardial depression.[2]

Burns anaesthesia

As mentioned previously, a severely burned patient poses numerous challenges to the anaesthetist. Many of these are not exclusive to burns but the combination of difficulties is not often found outside of the burns theatre. Challenges include airway distortion, pulmonary dysfunction, difficult vascular access, rapid blood loss, problematic monitoring and positioning, impaired temperature regulation, altered drug pharmacokinetics and pharmacodynamics, renal dysfunction and sepsis.[2] To be able to effectively deal with these challenges a thorough pre-operative assessment with attention to burn-specific parameters is necessary. It is important to know the extent (TBSA, depth, distribution) and mechanism of the burn injury, the time since the injury occurred, whether there are any associated non-burn injuries, and the surgical plan.

Securing the airway

In the operating theatre, the most immediate challenge is that of securing the airway. In a burns patient the possibility of a difficult airway should always be considered. The airway can be distorted due to oedema, narrowed due to injury that has healed with stricturing, or difficult to position due to facial and neck burns or contractures. Once intubated the challenge is to secure the endotracheal tube (ETT) so that it will remain in place during intra-operative manipulation of the patient's head. This is particularly problematic in the patient with a burned face. The ETT can be firmly secured by looping a nasogastric tube around the hard palate and securing it to the ETT (the "Gray-Rode technique").[6] If the patient has been intubated via the nasal route the nasotracheal (NTT) tube can be secured by looping umbilical tape around the bony nasal septum and tying it to the NTT.[2]

Inhalation injury can affect the upper airway (larynx and above) or the lower airway (below the larynx). The upper airway is directly damaged by hot gases - air or steam, whereas the lower airway is damaged by toxic products of combustion. The tracheobronchial tree develops a large sustained increase in blood flow as well as shedding of the bronchial epithelium and copious secretions. These solidify to form casts which obstruct various sized airways, potentially creating areas of increased shunt as well as barotrauma to non-occluded lung.[2]

Intravenous fluid administration

Once the airway is secured and the patient being mechanically ventilated, the next challenge intra-operatively is that of intravenous fluid administration - which fluids to give and when to give them. Although patients with severe burns will all require blood transfusion at some point, it is important to bear in mind the associated risks and apply restrictive strategies to blood products. Apart from the small but obvious risk of transmission of infectious diseases, blood transfusion causes significant immune suppression leading to increased rates of post-operative infections. There is also the risk of transfusion related lung injury (TRALI) as well as ABO incompatibility and transfusion errors.

Due to these risks as well as differences in individual patient physiology, the concept of a single universal transfusion trigger is being replaced by that of a physiological transfusion trigger.[7] Instead of transfusing based on the haemoglobin level, the decision to transfuse blood products should be tailored to the individual's blood volume status; acuity of blood loss and tissue perfusion. Thus instead of a single value dictating blood transfusion, clinical signs and symptoms as well as biochemical markers (e.g. lactate, central venous oxygen saturation) should be informing the decision to transfuse. Blood loss can be predicted according to the TBSA being debrided as well as how soon after burn the surgical procedure is taking place. Strategies to minimise intra-operative blood loss include subdermal clysis with an adrenaline-containing solution; applying adrenaline-soaked swabs to excised areas; using tourniquets where appropriate and maintaining euthermia by aggressive warming of the operating theatre and patient.

Thermoregulation

Maintaining euthermia also avoids enhanced catecholamine release, tissue catabolism and further up-regulation of an already hypermetabolic state. It is important to remember that euthermia in a severely burned patient is higher than "normal" patients. The normal threshold temperature (the set temperature range that triggers a response to change in temperature) is set in the hypothalamus. Due to the hypermetabolic state and inflammatory mediators, severe burn injury causes the hypothalamus to increase the threshold set-point by 0.03 degrees Celsius per %TBSA.[2] Thus it is important to avoid relative hypothermia as this will cause a rapid increase in the metabolic rate in an attempt to return the core temperature to the higher set-point. Burn patients are unable to thermoregulate peripherally due to loss of cutaneous vasoconstriction and evaporation control in the injured skin.

Pharmacological considerations

Drug administration is also profoundly affected by the changes in physiology in a patient with severe burns. Pharmacokinetics and pharmacodynamics change according to the post-burn phase. The initial 48 hours (acute / resuscitation phase) is associated with decreased cardiac output, decreased renal and hepatic flow (and thus drug clearance) and decreased tissue perfusion. Thereafter the patient enters the hypermetabolic/hyperdynamic phase and will have increased cardiac output and oxygen consumption as well as increased renal and hepatic flow.[2] This phase continues for up to 2 years post-burn. Also influencing drug action is the fact that many drugs are protein-bound and thus their efficacy is affected by the changes in protein levels post-burn. Albumin (whose levels decrease with burn injury) binds mostly acidic

and pH neutral drugs, and alpha-acid glycoprotein (AAG, whose levels may double due to it being an acute phase protein) binds mostly pH basic drugs.

Nutrition

Feeds should be continued up until time of surgery in the intubated patient who is not undergoing any form of airway manipulation in the operating theatre. This is to ensure optimization of nutrition - the hypermetabolic state demands high caloric intake. Patients with major burns will undergo repeated surgical procedures and the cumulative loss of nutrition is unacceptably high if feeds are stopped pre-operatively for every procedure. All unintubated patients, but in particular paediatric patients, should be allowed clear fluids up until 2 hours pre-operatively.

In conclusion

Finally, the importance of communication within the multidisciplinary team cannot be over-emphasised. The management of a severely burned patient is unique in many ways, and good outcome can only be achieved through dedicated multidisciplinary involvement and co-operation.

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