

ANAESTHESIA FOR BURNS

Dr Grant Waters

Middlemore Hospital
Auckland

Burns is a ubiquitous disease treated in varying ways throughout the world, based on workload and facilities available. There are many interventions that anaesthetists perform although currently there is little high quality evidence in support of these. This presentation is about some of the principles involved with burns care, and some of the methods we find useful.

Continuum of Care

Anaesthetic input into management of the burns patient is part of a continuum of care, and our role changes through the patient's hospital admission.

- Many burns are associated with a *traumatic event*, so we may be involved with perioperative management of the burns patient having surgery for a non burn related injury.
- In the first week we're involved in *repetitive* trips to theatre to *debride* and remove the dead tissue. The dressings used vary largely on the depth of the wound, and in most places, what is available.
- Once the dead tissue is removed, there are multiple trips to theatre for *dressing changes* and the gradual replacing of biological dressings with the patient's own skin.
- Interspersed with trips to theatre are the dressing changes and wound wash downs. These procedures involve input from minor sedation in the burns unit through to general anaesthesia.
- Throughout the patient's course we're involved with *pain management*, which can be extremely complex and contributes directly to patient outcome.
- Patients return repetitively throughout their lives for management of *scar contractions*. They become very institutionalised, prone to depression and post traumatic stress disorder if poorly managed.

As interventions are numerous, varied and "made up" on the spur of the moment based on what the patient looks like at a point in time, there needs to be good communication between all members of the team caring for the patient.

Pathophysiology

Despite huge variability in care throughout the world, burns survival has continued to increase, due to improvements in care in many areas, and the gradual increase in understanding of the pathophysiology of burn injury. To understand where treatment is heading, we need some understanding of the pathophysiology of the burn injury. Tissue injury following a major burn can be divided into the following zones –

1. Zone of necrosis. This is the area of burn injury that is non viable. The dead tissue needs to be debrided to allow healing and prevent sepsis.
2. Zone of stasis. Here there is local tissue hypoxia with inflammatory mediator generation and superoxide generation with reperfusion. If improperly treated, the zone of stasis can convert to a zone of necrosis.
3. Zone of hyperaemia. In this area, there are leaky capillaries with fluid shifts resulting in intravascular hypovolaemia and oedema formation. This in turn leads to organ dysfunction, increased burn depth and reduced graft viability. Where burn injury exceeds 30% BSA there is a systemic "zone of hyperaemia" which produces ARDS (even in the absence of an inhalational injury) and gut oedema. This in turn leads to a breakdown of gut barrier function with increased bacterial translocation and multi organ dysfunction.

These changes lead to the massive fluid volumes required for initial resuscitation of the burns patient. Many of the improvements in patient outcome relate to management of the inflammatory response related to burn injury.



Resuscitation

This is the foundation of a successful outcome in burns. The aim is to restore oxygen delivery to the tissue and most centres use formula based fluid resuscitation. However significant numbers of ED doctors have poor knowledge of resuscitation formula (4% in UK, 33% in US). For ISBI (International Society for Burn Injury) burns centres, 90% start with a formula, but 50% go off on their own variation within 24 hours. Most (70%) use Parklands, but as many as eight formulae are used with 13 different fluids. This variation suggests significant unsolved issues –

- Cannot always get euvolaemia – zone of hyperaemia.
- Local tissue hypoxia occurs despite adequate global parameters: zone of stasis.
- In many situations formulae underestimate the resuscitation volume required and inadequate end points are used, eg urine output used exclusively in 95%, but adequate urine output doesn't reflect adequate renal flow, volume status or the lack of local tissue hypoxia.

The net result is administration of 6 +/- 2.3ml/kg/% burn (cf Parkland 3-4ml/kg/% burn) with a high incidence of abdominal compartment syndrome. This has a very high (70-100%) mortality.

Possible Solutions

- Control inflammatory mediators
 - Early resus
 - Early excision
 - Early enteral nutrition
 - Aggressive sepsis control
- ??? Antioxidants
- Start with a formula and use the minimum required to achieve adequate organ perfusion

Early Debridement

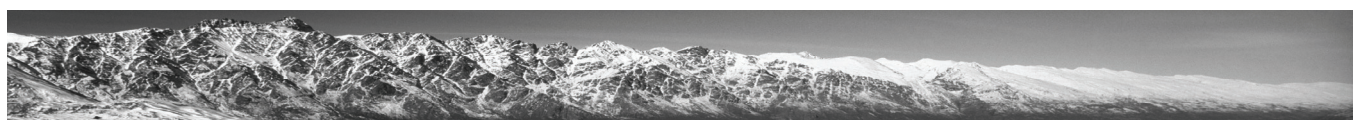
This is probably the most significant change in practice contributing to a reduction in mortality for major burns. Burns are not static and wounds evolve over about 72 hours, so complete debridement may require multiple trips to theatre. Complete excision and closure of the burn wound within 2-3 days of injury improves the hypermetabolic response to the burn, removes the source generating the inflammatory cytokines and oxidants. Early debridement also decreases wound contamination, infection, and blood loss associated with debridement. In addition, pain control is improved.

Middlemore Hospital – Practical Approach to Burn Debridement

- Two specialist anaesthetists
- Secure ETT via maxillary screw
- Monitoring – ECG dots stapled to defibrillator pads
- Blood reduction
 - One surgical team
 - Tumescence – 1:500,000 adrenaline
- Warming
 - Warmed tumescence fluid
 - Blanketrol

Metabolism

The hypermetabolic response to burn injury is the primary contributor to morbidity and mortality and increases linearly with total burn size. This metabolic state lasts from five days to one year post burn. It is mediated through catabolic hormones and inflammatory mediators and can only be modulated, not curtailed. It also contributes to poor wound healing and death.



Factors modulating metabolic response to burns –

- Surgical
 - Early excision
 - Infection control
- Anaesthetic
 - Maintenance of body temperature
 - Analgesic management
 - Anxiolysis
- Pharmacological
 - Oxandrolone
 - Insulin
 - β -blocker
- Nutritional
 - Early enteral nutrition

Enteral Nutrition

Patients with burns greater than 30% BSA need nutritional augmentation, and the earlier it's started, the less likely to fail. Post pyloric feeding is more reliable due to a high incidence of gastric stasis. Enteral nutrition –

- Modifies inflammatory and hypermetabolic responses
- Increases insulin secretion
- Improves gut integrity and gut oxygen delivery in early phases
- Decreases bacterial translocation and sepsis
- Decreases LBM loss
- Improves wound healing
- Improves survival
- Decreases length of stay
- Improves immune competence

Enforcing a six hour pre-operative starvation generates calorie deficit. There are many approaches to feeding peri-operatively –

- Enforced six hour starve
- Two hour starve
- Switched off at the door to OR
- Continued NJ feeds through theatre

The Middlemore Hospital approach is to continue feeding. There is no evidence that it increases risk, and we have had no episodes of significant aspiration. There is a potential problem with enteral feeding resulting in increased gut oxygen requirement in the septic phase of burns causing gut necrosis and death. There may be a place for suspending enteral nutrition during periods of shock or high inotrope requirement.

Pain Management

Most patients with burns are guaranteed pain. The management or lack thereof is directly linked to onset of depression, post traumatic stress disorder, speed of representing for scar revision, and lack of rehabilitation back into the community.

Treatment of burns pain is made more complex due to altered pharmacology due to –

- Changed blood flow to organs of metabolism and excretion
- Changed plasma protein / protein binding
- Altered Vd from fluid shifts
- Prolonged use causing tolerance



Patient specific analgesic requirements may change from day to day and all analgesia needs to be titrated to effect.

Burns pain is divided into –

- Background – rest and movement
- Breakthrough – surges through day
- Procedural – may be worse than pain of original injury
- Chronic pain

The backbone of treatment is still opiates, however one does see opiate tolerance and opiate induced hyperalgesia, so drugs to opiate spare and opiate rotation become standard practice.

References

1. Steven E Wolf. The year in burns 2008 – Review. Burns 35 (2009) 1057-1070
2. Steven E Wolf. The year in Burns 2007 – Review. Burns 34 (2008) 1059-1071
3. Ricardo Alvarado et al. Burn Resuscitation – Review. Burns 35 (2009) 4-14
4. Stephen Tricklebank. Modern trends in fluid therapy for burns – Review. Burns 35 (2009) 757-767
5. Joachim Boldt et al. Fluid management in burn patients: Results from a European survey – More questions than answers. Burns 34 (2008) 328-338
6. Ernest A Azzopardi et al. Fluid resuscitation in adults with severe burns at risk of secondary abdominal compartment syndrome – An evidence based systematic review. Burns 35 (2009) 911-920
7. P. Richardson et al. The management of pain in the burns unit – Review. Burns 35 (2009) 921-936
8. Nida H Corry et al. Post Traumatic stress disorder and pain impact functioning and disability after major burn injury. Journal of Burn Care and Research Vol 31 No1 Jan/Feb 2010. 13-25
9. Nguyen Nhu Lam et al. Early enteral feeding for burned patients – An effective method which should be encouraged in developing countries. Burns 34 (2008) 192-196
10. Kathy Prelack et al. Practical guidelines for nutritional management of burn injury and recovery – Review. Burns 33 (2007) 14 -24

