Anaesthetic considerations for major thermal injury

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Summary

Extensive heat-induced thermal injury occurs with alarming regularity in the North American continent. Approximately two million major burns occur annually in the United States, of which about 12,000 are fatal.[1] The Canadian experience is probably proportionately similar. Most anaesthetists will on occasion be asked to assist in the initial assessment, resuscitation and stabilization of these patients and subsequently provide anaesthesia for the multitude of surgical procedures most will require. Many major burn patients will have to be admitted to intensive care units, allowing additional involvement of the anaesthetist in their care.

A brief review of the classification of severity, extent and causative factors of major thermal injury is in order. Burns can be inflicted by radiation, caustic chemicals, electricity or heat.[2] Heat burns, with which this paper concerns itself, can be due to scalding, contact with hot objects or direct flame injury.

A first degree burn involves only the epithelial layer. Though often very painful, the erythema that occurs resolves with no residual scarring. A second degree burn involves epithelium and, to a varying degree, also the dermis. Re-epithelialization will occur due to sparing of dermal appendages such as hair follicles. Pain and scarring vary according to the depth of the dermal injury. A third degree burn involves the entire skin thickness. It is usually painless and will lead to scarring.

It is useful for initial management and prognostic purposes to estimate roughly the proportion of body surface injured by a burn. In this regard, the familiar "rule of nines" has proved reasonably accurate and easy to apply.[2] In the adult, the anterior or posterior surface of an upper extremity comprises approximately 4.5 per cent of body surface area (BSA); nine per cent applies to the anterior or posterior surface of a lower extremity. The anterior or posterior trunk each accounts for 18 per cent, while the anterior or posterior surface of the head and neck comprise 4.5 per cent. A modified "rule of nine" for a child accounts for a disproportionately larger head and smaller lower extremities.[2]

A "major" thermal burn has been defined to include a second degree burn in adults of 25 per cent of BSA or greater, a third degree burn of ten per cent of BSA or greater and all smoke inhalation injuries.[3]

Pathophysiology of major burn injury

Clinical as well as basic research demonstrate the devastating effect on total body homeostasis in-

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duced by extensive thermal injury. Major body systems, physically distant from the actual site of burning, are profoundly affected. A sound knowledge of these important pathophysiological changes, particularly as they relate to cardiovascular and respiratory function, is vital to safe, optimal patient care in the immediate post-burn phase and thereafter.

Cardiovascular effects

Profound changes occur in the circulation following a major burn. These relate specifically to cardiac output, volumes of vascular compartments and vascular permeability.

Animal studies demonstrate a marked immediate increase of vascular permeability in the area of burn. Though most prominent here, this increase in vessel "leakiness" becomes generalized in burns comprising greater than 30 per cent of BSA.[4] The destruction of the semi-permeable membrane qualities of the vascular tree leads to a tremendous transvascular loss of fluid, electrolytes and proteins. A serious blood volume deficit thus results. Interstitial fluid volume also decreases, often to a greater extent than the intravascular loss because of transfer into the cells of injured tissue. This redistribution of body fluids, the visual manifestation of which is tissue swelling, is essentially complete within 24 hours, the greater proportion having occurred during the first 12 hours.[4] These events have important implications regarding the appropriate type and quantity of fluid administered in the post-burn period.

A significant reduction of cardiac output would be the expected result of a reduced plasma volume. This change indeed does occur but to a far greater extent than can be attributed to this cause alone. Cardiac output can be abruptly reduced to as little as 50 per cent of normal before a demonstrable reduction in plasma volume.[4] A low cardiac output in the presence of reasonable cardiac filling pressures usually suggests reduced myocardial contractility. A circulating myocardial depressant factor or factors have been found in cases of major burn injury.[5] Human sera derived from burned patients contain a low molecular weight protein of about 8,000 daltons which in rabbits produces ECG changes suggestive of myocardial ischaemia and, *in vitro*, depression of smooth muscle contractility.[6] Additionally, papillary muscle *in vitro* exposed to serum from burned patients soon ceases to contract.[4] The significance of this substance or substances in the burned patient, at least as it relates to overall survival, remains speculative. In any event, cardiac output returns to normal within 36 hours, assuming of course that adequate volume resuscitation has occurred.

Respiratory effects

Derangement of the oxygen delivery systems of the body is a major cause of mortality and morbidity during and following thermal injury. This is usually a consequence of inhalation of the various products of combustion. Alternatively, pulmonary dysfunction can occur without actual inhalation, representing an end-organ response to a generalized systemic insult.

Carbon monoxide (CO) poisoning is a major cause of early mortality following exposure to fire.[7] This odourless and non-irritating gas is produced by the incomplete combustion of carbon-containing materials. Because its affinity for reversible binding to the haemoglobin molecule is more than 200 times that of oxygen, very small concentrations in inhaled air can be lethal. For example, the concentration of oxyhaemoglobin is reduced to 50 per cent of normal by prolonged inhalation of air containing 0.1 volume per cent CO.[8] Carbon monoxide also inhibits oxygen delivery by shifting the oxygen dissociation curve to the left. The lethal potential of this gas is demonstrated by the fact that 50 to 60 per cent of all fire deaths are related to inhalation injury, with the vast majority of these being due to carbon monoxide. Many of these fatalities occur at the scene of the fire.

Direct thermal injury to both upper and lower airway can occur. A combination of efficient dissipation of heat in the upper airway[9] and probable reflex laryngeal closure[10] account for a very low incidence of heat injury below the larynx.[11] Intraoral structures, including the larynx, however, can quickly become massively oedematous leading to obstruction and asphyxia.

Injury to tissue below the vocal cords can occur with the inhalation of superheated soot particles and/or a variety of noxious substances produced by fire. These include aldehydes, oxides of sulphur and nitrogen, hydrogen cyanide, hydrochloric acid, sulphuric acid and the halogens.[7] Inhaled smoke causes mucosal oedema and an

abrupt decrease in ciliary activity. Histologically, epithelial damage resembling a tracheobronchitis occurs.[12] Surfactant activity decreases, resulting in an immediate tendency to small airway collapse. Bronchoconstriction with wheezing might result from a combination of irritant-receptor stimulation by smoke particles and decreased luminal diameter from oedema.

Frank parenchymal lung injury results from the travel distally of inhaled toxins. The combination of a direct injurious action of these and the generalized increase in capillary permeability induced by extensive burn injury can cause extensive alveolar and capillary epithelial damage. The concomitant loss of semi-permeable membrane function of capillaries can culiminate within 12 to 48 hours of injury[12] in a picture typical of the adult respiratory distress syndrome. This can progress after several days to mucosal sloughing, atelectasis and intrapulmonary haemorrhage. The adverse effects on pulmonary function are predictable. Increased airway resistance and reduced lung compliance combine to increase the work of breathing significantly.[8] Arterial hypoxaemia, often combined with hypocarbia, is the usual finding on blood gas analysis. These changes, most marked for the 10–14 days following burns, usually resolve within one month.[12] Bacterial superimposed infection is a frequent occurrence within a few days of inhalation injury, occurring in 15 per cent of patients.[13] Bronchopneumonia, caused by airborne organisms, has displaced in frequency miliary pneumonias associated with haematogenous spread from the infected burn-wound.[4]

Metabolic effects

The burn victim quickly develops a marked increase in catabolic activity with its associated nitrogen losses,[14] and that to a greater extent than occurs in any other catastrophic illness. This stress response to injury is manifested by a persistent hyperpyrexia, tachycardia, hyperpnoea and body wasting. A proportionate increase in oxygen consumption occurs. This metabolic effort, mounted to heal extensively damaged tissue, appears to be directed by neurohumoral mechanisms. Prolonged serum epinephrine and norepinephrine elevations have been noted,[13] in addition to the usual biochemical milieu typical of a stress response. The overall result is heightened catabolic activity which provides the substrate necessary for restoration of injured tissue. Elements of this process include elevated free fatty acids, glycogen mobilization, gluconeogenesis and relative insulin resistance associated with glucose intolerance.[3]

Appropriate care of the burn patient must take into account this high level of metabolic activity. Early start of high-caloric feedings, enterically if possible, is a top priority following initial stabilization. Additionally, evidence exists that nursing patients with burns greater than 50 per cent surface area in a warm environment will decrease the tendency towards a negative nitrogen balance.[15] Presumably the body needs less tissue breakdown for the production of body heat in the presence of a high ambient temperature. The most appropriate temperature is considered to be approximately 34° C.[4]

Sepsis

Before the clinical use of effective topical therapy, burn-wound sepsis accounted for 75 per cent of deaths occurring from burns.[16] A wide choice of effective topical agents has changed this. Topical agents, however, are not free of side-effects. These are pertinent to the anaesthetist and will be discussed later.

Acute management

Immediate care of the burn patient must take into account the previously discussed major organ pathophysiologic changes. This can be logically effected by appropriate application of the ABC's of resuscitation appropriate to any acutely critically ill patient.

Airway and breathing

Early assessment of a fire victim must assume a variable degree of carbon monoxide poisoning. This is particularly the case if any mental disturbance is apparent. High concentrations of oxygen by mask should be immediately administered without waiting for or relying upon arterial blood gas results. The half-life of carboxy-haemoglobin in the blood is reduced from four hours to about one hour by breathing 100 per cent oxygen compared to room air.[17] Carbon monoxide poisoning can be confirmed by direct measurement of blood carboxy-

haemoglobin. Burned patients who manage to reach the hospital rarely have a carboxyhaemoglobin saturation greater than 15 per cent. Colour of the skin is not particularly helpful in assessing carbon monoxide poisoning, for the typical cherry red colour might be mased by true cyanosis secondary to hypoxaemia. Arterial PO_2 can be entirely normal during significant carbon monoxide poisoning. A valuable diagnostic clue is a measured oxyhaemoglobin saturation much lower than expected in relation to PaO_2.

Careful assessment of patency of the upper airway is vital. Fire need not necessarily occur in a closed space for major damage to occur. Any singeing of hair or burn above the upper thorax should arouse suspicion. Stridor or hoarseness must suggest critical encroachment of some level of the airway with potential obstruction and asphyxia. It is remarkable how quickly orofacial oedema, even in the absence of laryngeal injury itself, can seal off the airway. If any doubt exists, the airway should be secured immediately with a tracheal tube. The use of a muscle relaxant in such a situation is clearly contraindicated. An awake intubation will usually be possible. Fibreoptic laryngoscopy and intubation would only be feasible if the operator has a wealth of previous experience with this technique, considering this situation of a deformed, oedematous secretion-filled airway. A tracheostomy occasionally is the only viable option, with the recognition that patient mortality and morbidity might be significantly increased, especially if the incision is made through burned tissue.[18] After the airway has been secured, a more thorough and leisurely assessment of intrapulmonary damage can follow, if necessary. Abnormalities on chest *x*-ray might lag several hours behind the onset of clinical respiratory difficulty and arterial hypoxaemia. Fibreoptic bronchoscopy is often helpful for a direct visual estimate of major airway damage.[19] Treatment at this stage, in addition to high concentrations of oxygen may include controlled ventilation and bronchodilators for audible wheezing. The addition of PEEP, or of CPAP if spontaneous respiration is feasible, will often improve gas exchange.

Circulation

Preservation of effective major organ perfusion becomes increasingly compromised following major thermal injury. This is probably primarily because of massive loss of intravascular volume, mostly into the region of the burn but also into more distant tissues due to the generalized increase of vascular permeability. Cardiac filling pressures are therefore reduced. This occurs in the presence of reduced cardiac function which is likely toxin-induced. Immediate monitoring of the circulation must therefore be established. This should include an ECG, measurement of urine output by means of an indwelling bladder catheter, insertion of arterial and CVP lines, and arterial blood gas determinations. The CVP, as is the case in other critical illness where ventricular function might be compromised, can give misleading and unreliable estimations of circulatory volume. A pulmonary artery catheter can be more useful for more accurate assessment and appropriate haemodynamic manipulations.[20]

The appropriate type of fluid replacement, i.e., colloid vs. crystalloid, has been controversial. A consensus, however, appears to be emerging. The generalized vascular permeability defect corrects itself within 24 hours after the burn. During that time, therefore, it is probably reasonable to resuscitate only with crystalloids.[4] The loss of the vascular semipermeable membrane qualities would negate the usual positive effect on intravascular fluid retention of protein-containing colloid solutions. Guidelines for the appropriate quantity of fluid to infuse have been suggested by several formulae, including Brooke's, Moore's and the Parkland Hospital. These regimens attempt to account for fluid loss related to evaporation, oozing from raw surfaces and fluid shifts. Whatever the formula, it is apparent that fluid administration rates of 10 $ml \cdot kg^{-1} \cdot hr^{-1}$ during the first 12 hours followed by 5 $ml \cdot kg^{-1} \cdot hr^{-1}$ for the subsequent 12 hours will often be necessary.[3] Regardless of fluid type or quantity administered, a diuresis of at least 1 $ml \cdot kg^{-1} \cdot hr^{-1}$ provides reassuring evidence of adequate organ perfusion. Colloid solutions such as albumin are best introduced following the initial 24 period after the burn.

Occasionally cardiac output and resulting organ perfusion will be inadequate in the presence of satisfactory cardiac filling pressures. This suggests depressed myocardial contractility, perhaps related to a myocardial depressant factor or factors previously discussed. Treatment with an inotropic

agent, such as dopamine, perhaps in combination with an after load reducer, such as sodium nitroprusside, might be necessary.

Concomitant factors

Assessment and care of burn patients can be complicated by other factors common to many severely traumatized patients. There is usually a paucity of information on pertinent medical problems including current medications or significant chronic disease such as diabetes or hypertension. "Medic-Alert" bracelets are particularly helpful in this regard. The acutely burned patient must be assumed to have a full stomach. Because gastric emptying time can be greatly reduced following any trauma, the potential for bronchopulmonary aspiration of gastric contents probably correlates better with the period of fasting prior to the burn rather than after. Many patients will be inebriated,[21] with resultant depression of the respiratory, cardiovascular and central nervous systems. Major burns resulting from motor vehicle or aircraft accidents are often accompanied by head, thoracic, abdominal and extremity injuries. Emergency assessment must be rapid but nevertheless thorough in order to detect these coexisting injuries.

Acute anaesthetic considerations

Many patients with major thermal injury will not require anaesthesia in the immediate post-burn period. Some, however, will have associated life-threatening injuries which require prompt surgical intervention or rapidly deteriorating ventilatory or vascular function necessitating emergency escharotomy. Appropriate preoperative work-up, if not already obtained, would include a haemoglobin, platelets, PT, PTT, serum electrolytes, blood glucose, urinalysis, arterial blood gases, chest *x*-ray and ECG. The major considerations for safe anaesthesia in the immediate post-burn period include the degree of completeness of volume resuscitation, the safety of the airway and the appreciation of a potentially rapid deterioration in pulmonary function secondary to manifestation of the true extent of pulmonary injury.

Any suggestion of an upper airway burn is a contraindication to the use of muscle relaxants. Tracheal intubation is best accomplished with the patient awake, with or without use of fibreoptic laryngoscopy, or following an inhalation induction. Burns remote from the face might allow for the safe use of muscle relaxants. Although succinylcholine is said to be safe in the immediate post-burn period, pancuronium given in a higher than usual dose ($0.15\ mg \cdot kg^{-1}$) is just as effective and rapidly-acting in a rapid sequence induction.[22] A short period of possible postoperative ventilatory support is a small price to pay for what is perhaps a safer option.

Circulatory resuscitation is unlikely to be complete before any emergency operation. Appropriate monitoring should be established preoperatively if possible. Induction of anaesthesia must assume only partially corrected intravascular volume. Thiopentone in reduced dosage ($1–2\ mg \cdot kg^{-1}$) or ketamine ($1–2\ mg \cdot kg^{-1}$) might be reasonable choices. Maintenance of anaesthesia at this stage should probably not include agents with direct myocardial depressant effects, such as the inhalational agents in high concentration.

The full extent of pulmonary injury is often not apparent during the first few hours after a burn. Adequacy of intraoperative arterial oxygenation should therefore be assured by serial blood gas analyses. Increasing end-inspiratory pressures might signify decreasing pulmonary compliance and suggest the use of PEEP if PaO_2 is simultaneously decreasing. Extubation after the operation requires careful consideration and is best delayed in some instances.

Later anaesthetic considerations

The seriously burned patient, following successful initial stabilization and resuscitation, faces the psychological and physiological insults of a multitude of debridement and reconstructive procedures during the ensuing weeks and months. Initial management has become increasingly aggressive, for recent experience has shown a positive correlation between survival and the rapidity of burn-wound closure.[23] The latter is accomplished by many closely spaced major debridement procedures during the first few weeks.

The anaesthetist will play an important and vital role during the patient's long road to recovery. The pertinent ongoing areas of relevance for safe, effective anaesthesia in this more chronic, stabilized situation are now discussed.

Transport to the operating room

Transfer of the patient to a stretcher in the burn unit,

movement through often long and rough corridors, and countless manipulations in the operating room before induction of anaesthesia can be the most unpleasant and traumatic aspect of the entire perioperative period. This applies particularly to children. Premedication consisting of a narcotic analgesic in combination with a benzodiazepine is a humane approach to this problem. The patient will appreciate induction of anaesthesia in bed or on a stretcher if this can be safely accomplished.

Intravenous lines are usually scarce, and their continued functioning should be closely guarded. Body temperature should of course be maintained en route. Respiratory management, including supplementary oxygen and occasionally ventilatory support through an artificial airway, should be duplicated as closely as feasible during transport.

Monitoring

Suitable available sites for intravenous fluid therapy and placement of monitoring equipment are often at a premium. For major burn debridement, two large-bore intravenous lines, one of which is a central line, should be established. Access to vessels through burned tissue is sometimes necessary, although a cut-down should be avoided in these areas.

Certain aspects of monitoring may require modification. Even with all extremities burned, it is usually possible to place a blood pressure cuff somewhere and obtain a reasonable reading. An intra-arterial line can be justified for major debridement procedures where large blood loss is anticipated. Early removal postoperatively should avoid any significant morbidity. An oesophageal stethoscope might be more practical than the usual precordial variety. Skin for placement of ECG patches is often unavailable in the usual locations. An acceptable recording for rhythm analysis can usually be obtained from locations quite distant from the standard ones. The use of needle electrodes will occasionally be warranted, bearing in mind the increased electrical hazard to the patient. The oesophageal ECG,[24] which may be introduced into clinical practice soon, will likely prove popular in these patients.

Temperature control

The combination of cold operating theatres, large exposed areas of body surface with compromised heat-retaining qualities, administration of large volumes of fluids and lengthy procedures can promote marked intraoperative hypothermia. Already under a marked metabolic stress, some patients, particularly the elderly, do not tolerate hypothermia well with its resulting postoperative shivering and attendant increase in oxygen consumption.

The operating room should be warm, above 25° C. This is uncomfortable for the staff, but serves as a solid inducement for physicians and other personnel to work with dispatch. A radiant heater in the vicinity of the patient will increase the effectiveness of a warm room. Body surfaces which are not being worked on should of course be kept covered. Ventilation of the lungs with warmed humidified gases has been shown to be a great help in maintaining body temperature.[25] Prep solutions should be warmed before use. Blood and intravenous solutions should be run through a warming device.

Blood requirements

Few surgical procedures are as bloody as major burn debridement. Not uncommonly replacement of greater than one blood volume is necessary.[26] Many patients will be anaemic before coming to the operating theatre. It is prudent, therefore, not only to ensure that an adequate volume of cross-matched blood is available in the operating area before the operation starts but also, in selected patients, to start transfusing following induction. A Foley catheter will be helpful in monitoring intravascular volume and renal perfusion.

The usual problems associated with massive blood transfusion require attention. In addition, recovering burn patients have recently been recognized to exhibit persistently low ionized calcium levels.[27] Unexplained hypotension in the presence of an adequate central venous pressure during rapid transfusion of citrate-containing blood should suggest hypocalcaemia-induced myocardial depression. ECG changes typical of hypocalcaemia are not necessarily apparent. Calcium chloride should be administered intravenously in this circumstance.

Topical medications

The importance of topical therapy in burn management lies in the demonstration that fatal burn-wound sepsis can occur when the organisms involved have not significantly spread beyond the

areas of burn.[4] Currently employed topical treatments present some interesting implications for the anaesthetist.

Silver nitrate 0.5 per cent has enjoyed extensive use. An effective prophylactic agent for burns comprising less than 50 per cent of body surface area, it owes its antibacterial activity to the silver ion. Although systemic absorption of silver is minimal, absorption of large quantities of distilled water and leaching into the open wound of significant amounts of plasma sodium, potassium and other electrolytes can occur.[28] Sodium loss can amount to 350 mEq per square metre daily, requiring replacement either orally or parenterally. Conversion of the nitrite to nitrate by specific organisms in the wound occasionally causes methaemoglobin formation. This is suggested by a brownish appearance of the blood or slate-grey appearance of the wound. A blood methaemoglobin level determination is confirmatory.

Mefanide, available as a ten per cent cream, originally was used prophylactically but is presently employed more specifically for treatment of invasive burn sepsis. Its high osmolality causes pain on application, sometimes leading to a request for anaesthesia services in the burn unit. Mefanide is readily absorbed systemically. Its metabolic product, a paracarboxy compound, is very acidic. The drug is an inhibitor of the enzyme carbonic anhydrase. The resulting renal loss of bicarbonate, in conjunction with the acid metabolite, promotes a state of metabolic acidosis.[28] Although ordinarily compensated for by enhanced ventilation, the decreased ventilatory reserve associated with the hypermetabolic response to burn injury and perhaps an associated inhalational injury can result in respiratory failure. The agent should be used for as brief a period as possible.

The topical use of povidone-iodine ointment also is not hazard-free. Renal toxicity resulting from systemic absorption has caused fatal metabolic acidosis.[29] The high osmolality of this substance (> 1000 mosmol$\cdot L^{-1}$) creates a strong osmotic gradient between medicated areas and healthy tissue. Free water is lost into the wound, leading to intravascular dehydration and hypernatraemia.[30]

Metabolic alterations

Perioperative management of these patients must account for a significantly increased metabolic rate. Manifestations of this include moderate pyrexia and tachycardia. Although nutrition administered by mouth should be stopped several hours preoperatively to promote an empty stomach, the NPO period should be kept as short as possible commensurate with safety. The usual precautions related to parenteral feeding apply. The risk of hypoglycaemia following abrupt discontinuance of TPN solutions can be countered simply by continuing the infusion at the usual rate. Ventilatory settings must compensate for increased CO_2 production, with fine tuning provided by arterial blood gas measurements.

Sepsis management

These patients are already struggling to maintain asepsis. No additional insult is necessary. The anaesthetist, frequently a flagrant disregarder of standard protocol pertaining to care of intravenous lines, etc., must discipline himself in these cases. Any equipment designed for patient contact, such as blood pressure cuffs, should be sterilized beforehand. The wearing of plastic gloves by all those involved with the patient seems reasonable.

Anaesthetic techniques

A wide spectrum of anaesthetic agents and techniques can be safely employed provided that some basic precautions are taken.

Induction of anaesthesia must be cognizant of potential difficulties in airway management. Many patients with burns of the upper thorax, neck and face will quickly develop flexion contractures of the neck. The oral opening may be compromised by scarring. Unthinking use of muscle relaxants can lead to untreatable loss of airway control and therefore could be rapidly fatal. Tracheal intubation in the awake but sedated patient, either orally or nasally, is much safer. This might be impossible, whether attempted directly or blindly. Before causing extensive trauma to the airway as well as demoralization of the patient, fibreoptic bronchoscopy, in which every anaesthetist should become expert, ought to be considered.

Thiopentone is a valuable induction agent in burn patients and can be used safely following careful assessment of the airway and the adequacy of intravascular volume. Ketamine is also useful and is probably underutilized. Its most appropriate place has been the provision of unconsciousness and

profound analgesia for short painful procedures such as dressing changes. Airway obstruction or aspiration of gastric contents occasionally occurs with this agent. The anaesthetist must therefore have formulated a plan of action to deal with this possibility. The cerebral effects of this agent can be attenuated by small preoperative doses of droperidol or diazepam.

The inhalational anaesthetics have enjoyed considerable use. Excessive morbidity following multiple halothane administrations has not been demonstrated,[31] nor will this likely occur with enflurane or isoflurane.

The use of muscle relaxants poses unique problems in burn patients. Marked hyperkalaemia culminating in cardiac arrest occurring within minutes of a paralyzing dose of succinylcholine is well documented.[32] Precurarization can attenuate but not abolish the response.[33] The period of risk is considered to start from the fifth to the fifteenth day post-burn and last for two to three months by which time skin closure has usually occurred.[33] Because inadvertent exchange of even labelled syringes can occasionally occur, it is prudent to remove from the working surface of the anaesthetic cart any succinylcholine-containing syringes before anaesthetizing a burn patient.

The cause of this hyperkalaemic response in thermally-injured patients is still speculative.[33] The mechanism might be similar to that seen in neurologic disease, where there occurs increased chemosensitivity of the muscle membrane due to the development of new receptor sites in extrajunctional areas. The release from muscle of potassium after succinylcholine, rarely causing more than a 0.5 m Eq/L increase of serum concentration in the normal situation, can result in a lethal serum level in the presence of increased membrane sensitivity.

Thermal injury greater than 25 per cent of body surface area is followed by an altered response to non-depolarizing muscle relaxants.[34] Serum levels of d-tubocurarine necessary to achieve a given level of twitch depression are consistently in the range of five times normal. Reversal with neostigmine is easily accomplished with the usual reversing dose, but at a serum level of dTc associated with complete paralysis in normal individuals. This hyposensitization to dTc, apparently an alteration in pharmacodynamics rather than pharmacokinetics, is not likely explainable by the decreased levels of serum calcium present after major burns,[34] and can be attributed only minimally to the increased plasma protein binding of dTc known to occur.[35] This relative resistance to non-depolarizing relaxants may persist long after skin closure has occurred.[36]

Summary

Extensive thermal injury represents a major insult to body homeostasis. The anaesthetist, while providing anaesthesia for a multitude of debridement and reconstructive procedures, is also likely to assist in initial resuscitation and stabilization and subsequent intensive care management. A thorough understanding of the major systemic and end-organ effects after a major burn allow for a better appreciation of the many pertinent considerations for anaesthesia during the immediate post-burn phase as well as the later period of reconstruction and rehabilitation.

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