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The Anesthetic Management of the Severely Burned Patient

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Abstract. The anesthetic management of patients with major burns must be based on pathophysiological knowledge of the disease.

In the immediate post-burn period hemodynamic changes are of major importance. Because in severe cases any of the determinants of cardiac output can be implicated in these changes, precise physiological measurements are required. Arterial pressure, urinary output, central venous pressure and right heart catheterization can help in choosing the appropriate intervention. The metabolic response to the injury is initially protective, providing enough substrate, but later will lead to extreme levels of catabolism which can impair wound healing and immunological response. The anesthesiologist can decrease that response by providing calories, adequate room temperature, a reduction of the NPO period to the minimum necessary, and avoiding stress situations.

Respiratory injury can either affect the upper airway or produce the picture of ARF, which may require special treatment before, during and after surgery.

Several technical problems are usually present in the anesthetic management of these patients: 1) difficult airway, 2) scarce venous access, 3) no places available for monitoring, 4) drug dependency, 5) multiple anesthetics, 6) tendency to hypothermia, 7) inaccurate estimation of blood loss, 8) hyperkalemia after succinylcholine administration, and 9) systemic effect of topical medications.

Key words: Anaesthesia – Severe burns

The management of patients with major burns must be accomplished by a multidisciplinary team. The anesthesiologist plays a double role; in the operating room, providing adequate surgical conditions, and in intensive care units, contributing to the general management of the patient. The severity of the injury, with the corresponding metabolic and hemodynamic changes, as well as the time course of these changes, do not allow room for interruption(s) of total optimal care. The therapy must continue during surgery. Therefore, anesthesiologists must be familiar with the general pathophysiology of this type of injury.

A major burn includes: 1) a second-degree burn of 25% of body surface area or greater, in adults; 2) all third degree burns of 10% BSA or greater; 3) all smoke inhalation injuries; 4) electrical burns; and 5) any other complicated burn injury [41]. It has been estimated that 2,000,000 major burns occur per year in the United States, with a mortality of about 12,000 a year [26]. The significance for anesthesiological services demands can be appreciated if we consider the multiple surgical procedures each of these patients will require.

The three most common reasons for surgical intervention in burn patients are 1) wound debridement, 2) skin grafting, 3) correction of contractures [9].

Pathophysiology: Basis for Treatment

The course of the severely burned patient has been divided into an early, immediate post-burn period (24 – 48 hrs) when the resuscitation is the major aspect of treatment and second period extending up to the time when the wounds are healed, when wound care and nutritional balance are the prominent aspects of therapy [35]. However, metabolic alterations and other severe complications threaten the life of the patient during the whole course of the disease. The pathophysiological understanding of the injury justifies striving for the continuity of care during surgery and the immediate post-operative period.

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Hemodynamic Changes – Fluid Requirements

The hemodynamic changes observed in the hours following the injury are often due to changes in the preload and also the contractility of the heart. If they are not treated properly, hypoperfusion, hypotension, metabolic acidosis and renal failure may take place. The decrease in preload is mainly caused by sequestration of fluid in the burned area, with the corresponding decrease of the functional extracellular fluid [6]. During the first 6 to 12 hours after injury, the decrease in plasma volume requires electrolyte containing solutions, as large as 10 ml/kg/hour; it then improves and can be maintained with less than half that volume, becoming similar to the preburn values between 24 and 48 hours post-injury [6, 34]. Hemodynamic studies performed between the 7th and 22nd post-burn day showed an increase in blood flow directed primarily to the burn wound. The increase in cardiac output is a function of the body surface injury, probably associated with the formation of a hypervascular granulation tissue [47].

The red cell mass decreases from the time of injury at a rate of 8 to 8.5% of the initial value per day, probably secondary to entrapment and destruction of erythrocytes [16]. In patients with large burns, elevated values of erythropoietin were found during the initial stage (hypoperfusion hypoxia) up to the third post-burn day. Thereafter, erythropoietin fell to subnormal levels, persisting until wound healing was complete. Although significant aregenerative anemias were present in all cases, along with low erythropoietin [37], their cause is unknown.

Another source of fluid depletion is the increase in evaporated water which is about 1.2 ml/kg/% burn/24 hr in patients with more than 40% SBA burns [21].

In the early phase of major burn patients, the correction of preload is not the only factor to improve cardiac output. The release of a myocardial depressing factor (MDF) has been implicated as the cause of impairment of myocardial contractility [7]. Hemodynamically the low contractile state is manifested by a low left ventricular stroke work in the presence of an adequate or even elevated filling pressure (↓ LVSW/↑ PCWP). In this case, the administration of inotropic agents is indicated ($CaCl_2$ 1 to 2 g in 30 min; digoxin; dopamine $\leqslant$ 20 µg/kg/min).

In a very selected and small group of patients an increase in afterload and its favorable response to vasodilator treatment was reported [34]. However, the inherent dangers of using vasodilators in acute burn patients are indeed serious, and further experience with this technique is necessary before it is considered in the protocol.

The fluid requirements in the hours following the injury are related to the extension of the burn as well as to patient's weight. Several formulas are used to estimate the amount of fluid replacements (Evan's; Brooke's; Baxter's; Moore's; etc.). All these formulae are an approximation of the fluid requirements, and only with accurate monitoring can they be adjusted to each patient's needs. The administration of fluids without adequate monitoring can lead to serious error in management.

A fluid regimen can be considered effective if the following goals are obtained:

1. Normalization of blood volume and functional extracellular fluid space
2. Adequate peripheral perfusion
3. Avoid extensive and unnecessary fluid administration that can lead to extreme edema, congestion, and ischemia

The hemodynamic parameters usually measured and their limitations are the following:

Arterial Blood Pressure. In severe cases it is advisable to measure this directly (radial, dorsalis pedis, temporal, femoral, axillary, etc.). Since arterial pressure is an approximate index of perfusion pressure, it must be maintained above certain levels. Cerebral and coronary circulation are not usually affected until systolic pressure falls below 80 torr; renal perfusion is affected below 70 torr. It should be kept in mind, however, that arterial pressure is not an indicator of flow and therefore peripheral blood perfusion and cardiac output can decrease or increase without changes in arterial pressure, as indicated.

$$\text{Cardiac Output } (\downarrow\uparrow) \simeq \frac{\text{Arterial Pressure}}{\text{Systemic Vascular Resistance } (\uparrow\downarrow)}$$

Central Venous Pressure (CVP). In young and healthy patients with minor burns, the estimation of central venous pressure can be a valuable indirect index of blood volume and cardiac performance. Nevertheless, in cases of major burns or in patients with pre-existing heart and/or lung disease, CVP is not reliable. Biventricular disparity present in such patients makes the estimation of left ventricular function mandatory [15, 29].

Swan-Ganz Catheter. Since the introduction of a flow directed, balloon-tipped thermodilution pulmonary artery catheter into critical medicine [42], the measurement of all determinants of cardiac output

Table 1. Hemodynamic and respiratory information

Data to be entered	Calculations
1. Systolic arterial pressure	1. Cardiac Index
2. Diastolic arterial pressure	2. Stoke volume
3. Mean arterial pressure	3. Stroke index
4. Systolic pulmonary artery pressure	4. Left ventricular stroke work
5. Diastolic pulmonary artery pressure	5. Slope of left venticular function curve
6. Mean pulmonary artery pressure	6. Right ventricular stroke work
7. Pulmonary capillary wedge pressure	7. Slope of right ventricular function curve
8. Central venous pressure	8. Systemic vascular resistance
9. Heart rate	9. Systemic vascular resistance index
10. Cardiac output	10. Pulmonary vascular resistance
11. Body surface area	11. Pulmonary vascular resistance index
12. Hemoglobin	12. Pulmonary capillary oxygen content
13. Inspired oxygen concentration (%)	13. Arterial oxygen content
14. Arterial oxygen tension	14. Venous oxygen content
15. Mixed venous oxygen tension	15. Shunt fraction
16. Arterial saturation	16. Arterial-venous oxygen content difference
17. Mixed venous saturation	17. Oxygen delivery
	18. O_2 consumption
	19. O_2 utilization
	20. Shunt flow
	21. Pulmonary capillary flow

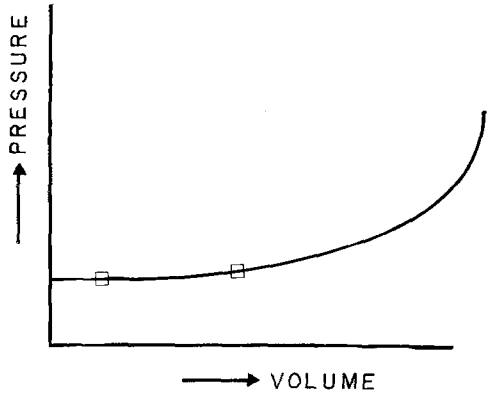

Fig. 1. Volume-pressure relationship of the left ventricle (schematic)

has been possible at the bedside. With the aid of an inexpensive programable digital calculator[1], a number of calculations can be made in about one minute (Table 1), both in intensive care and in the operating room, if surgery is necessary during this unstable period.

[1] Texas Instrument TI-59 with printer

The isolated estimation of pulmonary artery (PAP) and capillary wedge (PCWP) pressures can lead to erroneous fluid management, with extensive administration of fluids being more likely. The preload and output of the heart are related through the Starling law: if the ventricular fiber length increases (volume), the next contraction will be more forceful. Therefore what is important is a change in volume. The wedge pressure is an approximate estimation of end-diastolic volume, though it is not the same. Pressure and volume are related through the compliance of the ventricle. The normal heart possesses a compliance as depicted in Figure 1. Important changes in volume (and therefore output) can occur with minimal changes in pressure in normal hearts. During the fluid replacement phase, excessive amounts of fluid can be administered if "low wedge" is the only parameter observed. Our hemodynamic approach is the following: if the arterial pressure is adequate for cerebral and coronary perfusion (systolic > 80 torr), the cardiac output, and oxygen delivery are adequate (CI > 3 liters per minute; O_2 delivery 1000) and the arterial venous content difference is low (<5 vol%), the preload does not need to be increased further, despite a "low wedge."

Summing up, right heart catheterization allows us to measure the three determinants of stroke volume: preload, contractility, and afterload, and to suggest the necessary therapeutic intervention as well as determine the effectiveness of the therapy.

Bladder Catheterization is essential, with hourly or half hourly measurements of urinary output. A urine output of 1 ml/kg/hour or higher in the absence of osmotic effect (glucose, etc.) or diuretics is a favorable index of organ perfusion.

Metabolic Response

Glucose, protein and lipids are the sources of energy. In the normal individual 60–70% of the cellular requirements are covered by the metabolism of glucose. In general, the tissues can utilize any source of energy with the exception of the brain, red blood cells, white blood cells, and possibly new granulation tissue, which are glucose dependent [23]. When glucose is not available, it can be generated from proteins (gluconeogenesis) but not from fat.

In patients with major burns the following metabolic alterations occur: intake decreases; there is a marked increase in metabolic rate and in heat production; and several catabolic hormones are released.

The initial response after injury is catabolic. Glycogen is mobilized and glucose is released to cover the energy needs of the specific tissues. Therefore, glycogen is rapidly depleted and the proteins become the major source of glucose [23]. Fat, on the other hand, provides the rest of the energy needs (80 – 90%).

The signal which directs this metabolic response is hormone-mediated; in this process, insulin plays an important role. The activity of insulin is diminished because of antagonism [17] (with increase of catecholamines and glucagon) and also development of resistance [45] which leads to lipolysis, proteolysis and hyperglycemia, protecting mechanisms which increase the availability of the substrate.

The metabolic rate is elevated in direct proportion to the size of the burned area, thus it has been estimated that in burns of more than 40% of body surface area, the metabolic rate rises to 2 to 2.5 times normal [33]. The oxygen consumption and caloric expenditure have been measured at 1.5 to 2 times normal [5]. On the other hand, the catabolism present can be further increased by stressful situations which activate the hormone response, such as sepsis, light anesthesia, cold temperature. Changes in ambient temperature (25 – 33 °C) do not affect metabolism in a predictable manner in burns of less than 50% of body surface area [48]; however, in more extensive burns, a warm temperature (33 °C) decreases caloric demands and can be an important factor in the maintenance of energy balance [33].

The prolongation of the catabolic stage leads to tissue wasting, impairs the immune response [8, 18, 28], and alters wound healing. The goal of therapy is to raise caloric intake and lower demands. During anesthesia that goal can be accomplished by 1) continuing caloric intake; 2) reducing the period of fasting to the minimum necessary; 3) controlling room temperature; 4) avoiding stress situations (shivering, light anesthesia); 5) following strict aseptic conditions. As a whole, these needs can best be expressed by Allison's statement: "it should be a rule that all fluid administered should have at least some caloric or protein content" [2].

Respiratory Tract Injury

Respiratory injuries can result from upper airway obstruction due to thermal burns of the upper airway caused by direct effect of the heat (hot air; flames) and/or from acute respiratory failure (ARF) due to inhalation of suspended particles (smoke), and the toxic products of incomplete combustion [1, 3, 49] and perhaps a decreased activity in the clearance mechanism of blood microaggregates present in the early post-burn stages [36]. During the whole post-burn period ARF can also result from the most common causes seen in severely ill patients (sepsis, embolism, atelectasis, etc.) Pulmonary complications contribute to or cause the demise of most patients who die after burns. The mortality rate of patients who develop major pulmonary complications is as high as 80% [1, 22].

When the upper airway is involved, facial burns are frequently observed. If the edema reduces the airway passage to a critical level, symptoms of airway obstruction will appear. The inflammatory response is usually maximal at 48 hours and then begins to subside [3]. Indirect or direct laryngoscopy can provide the diagnosis; however, if anesthesia is required during this early post-burn period and severe edema is present (even in the absence of symptoms of obstruction) the anesthesiologist should consider the possibility of leaving the patient intubated until the edema subsides (using small diameter tubes with low pressure cuffs). Parenteral administration of steroids and spray of racemic epinephrine may reduce the edema, but their effects are not always predictable.

The diagnosis of ARF secondary to smoke inhalation can be suspected when: 1) facial burns are present, 2) the fire has been in a closed space, 3) there was open flame, or 4) high carbon monoxide levels [32, 49] were present. Arterial blood gases and chest roentgenograms will confirm the diagnosis. Because not uncommonly there is a discrepancy between the results of blood gases and the radiological examination, treatment must be initiated even with a normal chest x-ray. The increase in intrapulmonary shunt (Q_s/Q_t) is one of the earliest signs of ARF manifested by a PaO_2/FIO_2 lower than 300 torr (sea level) and/or a Q_s/Q_t greater than 15% (mixed venous blood gases obtained from pulmonary artery). In the early post-burn period other factors such as low cardiac output can decrease the arterial oxygen tension, therefore estimation of the PaO_2/FIO_2 alone can be misleading. Early bronchoscopy and ^{133}xenon scintillography have been reported to be helpful in the diagnosis of respiratory burns [30].

The therapy of ARF is based on the increase of transpulmonary pressure with the aim of increasing functional residual capacity above the closing volume, avoiding alveolar collapse and improving intrapulmonary shunt. The introduction of positive end-expiratory pressure (PEEP) by Ashbaugh and Petty [4] in the treatment of adult ARF along with cardiovascular monitoring and the use of intermittent mandatory ventilators (IMV) [13, 24] have allowed the use of a wide range level of PEEP [25] required to improve the intrapulmonary shunt and the functional

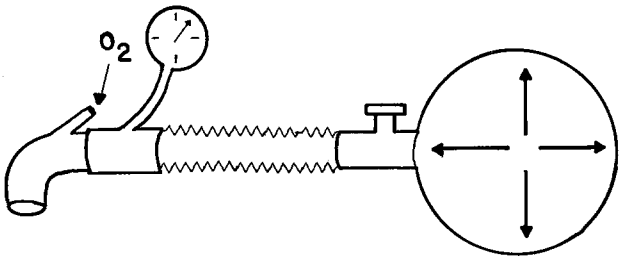

Fig. 2. Mapleson D system with a manometer

residual capacity to near normal values, thus drastically reducing mortality [14].

Anesthetic Considerations

The patients with major burns that will require surgery might present several anesthesiological problems:

Transport

Heat loss should be kept to a minimum, covering the patients with warm blankets if necessary.

If ARF is present and high levels of ventilatory support (IMV/PEEP) are required, the use of a self-inflated bag for transport may be dangerous because no PEEP can be administered. In some cases, the patients have to be transported with a ventilator and portable O_2/air tanks; however, in the majority of cases a Mapleson D system with a manometer can deliver the ventilatory support required (Fig. 2).

Difficult Airway

In the early post-burn period a patient with facial burns presents two major problems: first inadequate mask fitting and second the possibility of upper airway burns. Upper respiratory tract injury can lead to obstruction and asphyxia during induction. Besides, a swollen tongue can make the visualization of the larynx very difficult. In the late post-burn period scars and retractions can limit neck motion and also mouth opening.

The anesthesiologist must take all the necessary precautions to prevent these difficulties. Awake intubation, without paralysis, and the aid of a fiberoptic bronchoscope can be in difficult cases the clue of a successful anesthetic management.

Endotracheal intubation is mandatory in 1) severe burns of face and neck; 2) when upper airway lesions are present; 3) when patients are going to be placed in the prone position; 4) extensive and prolonged procedures; 5) in the presence of a full stomach.

In short procedures, mask anesthesia with inhalation agents or ketamine IV [27] can safely be administered and avoid the trauma of intubation in patients who will require a considerable number of anesthetics. However, gentle intubation with small and low pressure cuff tubes produces little or no trauma to the upper airway.

In cases of facial burn, it is sometimes very difficult to secure the endotracheal tube. An umbilical tape around the neck, tied to the tube, has been useful in these instances.

Difficulty with Intravenous Ports of Entry

In this case a major continuous venoclysis is procured through either subclavian, internal jugular, or femoral veins. Very seldom do we perform cutdowns.

Early administrations of blood in patients scheduled to have extensive debridement avoid the need for massive and fast blood replacement late in the case. This is an important consideration when intravenous sites are limited.

No Available Places for Monitoring

In extensive burns involving the four extremities this is a usual problem; however, that does not justify the avoidance of monitoring. If a precordial stethoscope cannot be placed (precordial, suprasternal, etc.) an esophageal stethoscope can be inserted through the nose or mouth before induction. EKG electrodes can be applied to almost any place of intact skin (forehead, neck, back, etc.). The same can be done with sterile needle electrodes, the only other alternative to obtain an acceptable electrocardiographic tracing.

A blood pressure cuff can be placed on any of the extremities either proximal or distal (forearm or leg). The concomitant use of the doppler ultrasound or the finger plethysmograph, allows accurate estimation of systolic blood pressure. In difficult cases an indwelling arterial cannula in the radial, dorsalis pedis, femoral, axillary, or temporal arteries is the only way to monitor arterial pressure. A small cannula, made of teflon and left only for the minimum time necessary, decreases the risk of complications.

Drug Dependency

These patients might require high doses of narcotics, tranquilizers, or barbiturates to obtain the desired

effect. Whether this is due to drug adaptation, tachyphylaxis or plain addiction, the dose requirements are certainly increased!

Multiple Anesthetics

Recommendation has been made to avoid repeated use of halothane within short intervals because of the possibility of liver injury [27]; however, Gronert et al. [19] showed that after administration of up to 22 halothane anesthetics per patient to burn patients, undue liver dysfunction was not present. Other inhalation agents such as enflurane can also be used, as well as neuroleptanalgesia or ketamine.

Ketamine has become very popular for anesthesia in burn patients [12]; nevertheless, it should be emphasized that it is an anesthetic which should be used with the same precautions as other general anesthetics. The airway is not protected, and to use ketamine when there is risk of aspiration is risky [11, 31]. It is also not suitable for long procedures as the only anesthetic agent, since prolonged recovery will then ensue. Emergence from anesthesia can be associated with psychological disturbances and restlessness. Nevertheless, it is a very useful agent for burns in children, for short debridements or dressing changes. The concomitant administration of diazepam or droperidol can avoid in part some of the untoward effects.

Tendency to Hypothermia

Monitoring of core temperature with an esophageal or rectal probe is mandatory under anesthesia, even for short periods of time. Several precautions can be taken to avoid hypothermia: 1) warming of intravenous fluids; 2) warming blankets used on the operating table; 3) room temperature adjusted to 26 °C and at least 50% humidity; 4) radiation heat sources around the operating table; 5) maintain covered the areas not affected by the procedure.

Estimation of Blood Loss

Massive blood loss often seen in extensive debridement and the inaccuracy of the methods to estimate blood loss justify early blood replacement (as soon as the patient is induced). Hemodynamic monitors (CVP and Swan-Ganz catheters) can provide an accurate estimation of blood and fluid volume replacement if they are available.

Succinylcholine and Thermal Injury

Cardiac arrest has been associated with the administration of succinylcholine in burned patients [10]. Tomie et al. [43] pointed out the massive release of potassium as the mechanism of cardiac arrest, occurring between the 10th and 60th days post-burn. The response is partially dose-related and is higher with more extensive burns [38]. The hyperkalemic peak is seen three to five minutes after the administration of the drug [38].

The mechanism of hyperkalemia has been extensively reviewed [20], indicating that after succinylcholine there is an increased permeability (K^+ leaves and Na^- enters the cell) at the endplate following depolarization. In the normal muscle, the increase in permeability occurs only at the receptor site. When the muscle is denervated, the chemosensitive area spreads to the whole muscle membrane. During this period of hypersensitivity, the administration of succinylcholine would produce an increase in permeability in the extremely chemosensitive area, with massive release of intracellular potassium [38]. However, the delayed development (20 days) of hyperkalemia in burned patients questions the theory that the muscle cell dysfunction plays an important role in its production [40]. Small doses of non-depolarizing muscle relaxant drugs can decrease the hyperkalemic response; however, their effect is unpredictable and only a full paralyzing dose can completely block their effect [20].

If cardiac arrest develops resuscitation is usually effective because the rise of serum K^+ is transient (10 minutes). Calcium chloride can be a valuable drug because it antagonizes the effects of potassium in the heart; cardiopulmonary resuscitation should be instituted immediately.

Systemic Effect of Topical Medication

Several of the topical medications used in burn areas may produce systemic effects after their absorption.

Mafenide and its degradation compound (p-carboxybenzensulfonamide) are strong carbonic anhydrase inhibitors. Carbonic anhydrase is present in various tissues including kidney, red cell and brain. Its inhibition at the kidney level leads to alkaline urine and metabolic acidosis very often compensated for by hyperventilation. An increase in the alveolar-arterial gradient of CO_2 (normally both are almost identical) suggests inhibition of the red cell carbonic anhydrase; this effect is also compensated by a rise in alveolar ventilation [46]. Persistent increases of minute ventilation, or in arterial carbon dioxide content or

metabolic acidosis are indications for discontinuing the use of mafenide [35].

Silver nitrate is another major wound antiseptic to be used with care, since some bacteria can reduce nitrate to nitrite. If systemic absorption is excessive, it can produce methemoglobinemia [44]; however, this is not often seen.

Providone-iodine ointment is also used in burn wound care, having an osmolarity in excess of 1000 μosmol/l. Under certain circumstances it can produce a major osmotic gradient resulting in intracellular dehydration and hypernatremia [39].

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