

The Importance of Different Parameters in Detection of Adequate Early Fluid Resuscitation in Burn Patients

Essay

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أهمية المؤشرات المختلفة لمعرفة كفاءة الإنعاش المبكر بالمحاليل الوريدية لمرضى الحروق

رسالة

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Summery and Conclusion

The patient suffered from a major burn has undergone one of the most severe forms of trauma. This injury results in loss of the essential function of the skin, which is the largest organ in the body that protects it from heat loss, evaporative losses of fluids and microbial invasion.

The treatment of patients with extensive burns remains a major challenge due to the lack of available autologous skin. Even with advances in burn care over recent decades, rates of mortality remain high among severely burned patients. Recent U.S. data indicate a 69% mortality rate among patients with burns over 70% of the total body surface area (TBSA).

Burn shock is a complex process of circulatory and microcirculatory dysfunction, not easily nor fully repaired by fluid resuscitation. Severe burn injury results in significant hypovolemic shock and substantial tissue trauma, both of which cause the formation and release of many local and systemic mediators. These mediators released from damaged tissues and cause increased capillary permeability.

The increased capillary permeability is a part of the edema process, especially in full thickness burns, where most of the capillary bed is occluded soon after injury. The protein rapidly leaves the plasma space through the “leaky” capillaries into the burn. Therefore, the effect of plasma colloid osmotic pressure is markedly diminished.

Early fluid resuscitation of burn shock proved to be effective in restoring normal tissue oxygenation to minimize complications and improve outcome. When resuscitation is delayed, recovery can be complicated by sepsis and multiple organ failure secondary to prolonged tissue hypoxia and reperfusion injury.

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List of Abbreviations

ACS	Abdominal compartment syndrome
AP	Arterial pressure
ARF	Acute renal failure
BD	base deficit
BL	Blood lactate
BUN	Blood urea nitrogen
C(p)	Compliance factor
CO	Cardiac output
CRF	Corticotropin releasing factors
CVP	Central Venous Pressure
DO2	Oxygen delivery
EVLW	Extravascular Lung water
Fe2	Ferrous ion
g	Grams
GEDV	Global end diastolic volume
H2O2	Hydrogen peroxide
HR	Heart rate
HSS	Hypertonic Saline
IAP	Intra-abdominal pressure
ICU	Intensive care unit
ITBV	Intrathoracic blood volume
ITTV	Intrathoracic thermal volume
kf	Filtration coefficient
kg	Kilogram
LR	Lactated Ringer
LV	Left ventricle
MAP	mean arterial pressure
MAP	Mean Arterial Pressure
mEq	Milli-equivalent
ml	Milliliter

mmHg	Millimeter mercury
MOF	Multiple organ failure
Na	Serum Sodium

List of Abbreviations (Cont.)

NIRS	near infra red spectroscopy
NO	Nitric oxide
O₂	Superoxide anion
OH	Hydroxyl ion
ONOC	Peroxy nitrite
PA	Pulmonary Artery
PAC	Pulmonary artery catheter
PAF	Platelet aggregation factor
PBV	Pulmonary blood volume
P_c	Capillary hydrostatic pressure
PCWP	Pulmonary capillary wedge pressure
PGI₂	Prostacyclin
P_i	Interstitial hydrostatic pressure
PiCCO	Pulse induced contour cardiac output
P_{if}	Interstitial hydrostatic pressure
[pi]p	Plasma colloid osmotic pressure
PTV	Pulmonary thermal volume
Q	Fluid filtration rate
Sigma	Capillary permeability
SV	Stroke volume
SVI	Stroke volume index
SVR	System Vascular resistance
SVV	Stroke volume variation
TBSA	Total body surface area
TBV	Total circulating blood volume
TxA₂	Thromboxane A ₂
USA	United States of America
VO₂	Oxygen consumption

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Introduction

The patient suffered from a major burn has undergone one of the most severe forms of trauma. This injury results in loss of the essential function of the skin, which is the largest organ in the body that protects it from heat loss, evaporative losses of fluids and microbial invasion (*Settle, 1996*).

The treatment of patients with extensive burns remains a major challenge due to the lack of available autologous skin. Even with advances in burn care over recent decades, rates of mortality remain high among severely burned patients. Recent U.S. data indicate a 69% mortality rate among patients with burns over 70% of the total body surface area (TBSA) (*Wang et al., 2010*).

A major focus of research during the 1960s and 1970s was the investigation of fluid shifts during the first 24 hours after burn injury. There has not been such an effort since that time. One of the key figures in burn resuscitation was Charles Baxter, who was important in developing the Parkland Formula, which today is the most frequently used resuscitation formula (*Baxter and Shires, 1968*).

Baxter and his colleagues understood that fluid requirements should be dictated by the urine output of the burn patients. They determined that patients required 4 ml/kg/% TBSA burn in the first 24 hours (*Baxter, 1974*).

Several resuscitation formulae for calculation of the fluid requirement are used worldwide. The most known formulae are: Evans, Brooke, modified Brooke, Monafo and Parkland (*Barrow et al., 2000*).

Early fluid resuscitation of burn shock proved to be effective in restoring normal tissue oxygenation to minimize complications and improve outcome. When resuscitation is

delayed, recovery can be complicated by sepsis and multiple organ failure secondary to prolonged tissue hypoxia and reperfusion injury (*Xia et al., 1991*).

Burn shock is a complex process of circulatory and microcirculatory dysfunction, not easily nor fully repaired by fluid resuscitation. Severe burn injury results in significant hypovolemic shock and substantial tissue trauma, both of which cause the formation and release of many local and systemic mediators (*Shimzu et al., 2003*).

The increased capillary permeability is a part of the edema process, especially in full thickness burns, where most of the capillary bed is occluded soon after injury. The protein rapidly leaves the plasma space through the “leaky” capillaries into the burn. Therefore, the effect of plasma colloid osmotic pressure is markedly diminished (*Rawlingson et al., 2001*).

Fluid resuscitation is aimed at supporting the patient throughout the initial 24 to 48-hours period of hypovolemia. The primary goal of therapy is to replace the fluid sequestered as a result of thermal injury. The critical concept in burn shock is that massive fluids shifts can occur even though total body water remains unchanged (*O'Mara et al., 2005*).

The resuscitation fluids should be minimized to decrease iatrogenic complication such as abdominal compartment syndrome. The concept of permissive hypovolemia was introduced in the practice of burn resuscitation through many publications and trials of alternate fluid types like hypertonic solutions, colloids and dextran, as original resuscitation efforts frequently lead to over-resuscitation (*Oda et al., 2006*).

Monitoring of burn shock resuscitation had traditionally relied on clinical assessment of cardiovascular and renal systems and biochemical parameters as indicators of vital organ perfusion. Heart rate, blood pressure, and

electrocardiographic recordings are the primary modalities for monitoring the cardiovascular status in any patient (**Huang et al., 1995**).

In major burns, the traditional clinical parameters (urine output and mean arterial blood pressure) provide insufficient information to serve as a guide for resuscitation. Other semi invasive or invasive methods have some potential hazards and need expensive equipments. The Pulmonary Artery Catheter (PAC), Central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP) and blood lactate level has been suggested as resuscitative parameters (**Dries and Waxman, 1991**).

Ideal endpoints in major burn fluid resuscitation are still a matter of controversial discussion in the literature. Several formulas have been developed to estimate the required fluid amount. Fluid administration should be adjusted to the individual needs of the patient. Hence, it is important to identify treatment-related factors influencing survival of patients with severe burns (**Holm et al., 2004**).

Aim of The Work

The aim of this study is to review the different parameters in detection of adequacy of early fluid resuscitation in burn patients and show the importance of each parameter.

Pathophysiology of Burn Shock

Burns are one of the most devastating conditions encountered in medicine. The injury represents an assault on all aspects of the patient, from the physical to the psychological aspects. It affects all ages, from babies to elderly people, and is a problem in both the developed and developing world. Burn injuries represent a diverse and varied challenge to medical and paramedical staff. Correct management requires a skilled multidisciplinary approach that addresses all the problems facing a burn patient (*Wilkinson, 1998*).

Understanding the pathophysiology of the burn injury is important for effective management. In addition, different causes lead to different injury patterns, which require different management. It is therefore important to understand how burn was caused and what kind of physiological response it will induce (*Kao and Garner, 2000*).

Burn injuries result in both local and systemic responses.

Local response

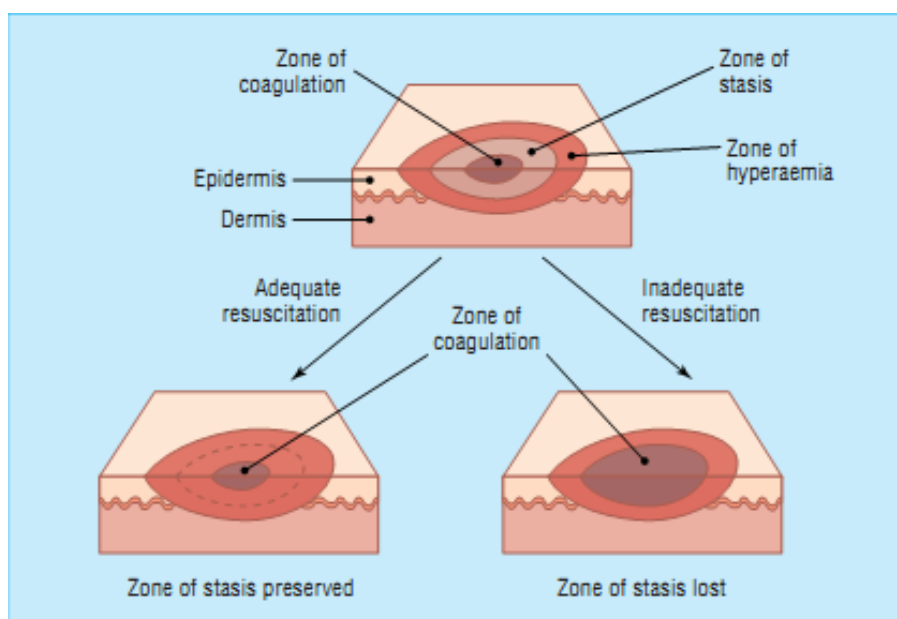
The three zones of a burn were described by Jackson in 1947.

Zone of coagulation: occurs at the point of maximum damage. In this zone there is irreversible tissue loss due to coagulation of the constituent proteins.

Zone of stasis: characterized by decreased tissue perfusion. The tissue in this zone is potentially salvageable. The main aim of burns resuscitation is to increase tissue perfusion here and prevent any damage becoming irreversible.

Zone of hyperaemia: In this outermost zone tissue perfusion is increased. The tissue here will invariably recover

unless there is severe sepsis or prolonged hypoperfusion (*Kramer et al., 2007*).



Fig(1): Jackson's burns zones and the effects of adequate and inadequate resuscitation (*Kramer et al., 2007*).

Systemic response

Burn injury causes extravasation of plasma into the burn wound and the surrounding tissues. Burn shock is hypovolemic in nature and characterized by the hemodynamic changes similar to those that occur after hemorrhage, including decreased plasma volume, cardiac output, urine output, and an increased systemic vascular resistance with resultant reduced peripheral blood flow (*Demling, 1987*).

Burn shock :

Burn is a major cause of morbidity and mortality. If left untreated cutaneous thermal injury greater than one-third of the total body surface area (TBSA) invariably results in the

severe and unique derangement of the cardiovascular function called burn shock (shock is defined as an abnormal physiologic state in which the flow of blood is insufficient to maintain adequate nutritive perfusion to meet cellular needs (*Kinsky et al., 1998*).

The critical concept in burn shock is the massive fluid shifts. So, total body water remains minimally unchanged. What actually changes is the volume of each fluid compartment, intracellular and interstitial volumes are increasing on the expense of intravascular volume (*Bartolani et al., 1996*).

In 1923, *Frank Underhill et al.*, demonstrated that unresuscitated burn shock correlates with greatly increased hematocrit values in burned patients, secondary to fluid and electrolyte loss after burn injury. The increased hematocrit values occurring shortly after severe burn were interpreted as a plasma volume deficit.

Initially, burn shock is hypovolemic in nature and is characterized by the hemodynamic changes similar to those that occur after hemorrhage, including decreased plasma volume, cardiac output, urine output, and an increased systemic vascular resistance with resultant reduced peripheral blood flow. Hypovolemia also results in hemoconcentration (*Demling, 1987*).

Burn edema

Massive tissue edema after thermal injury is a well-recognized entity; edema is the abnormal accumulation of liquid in cells, tissues, or cavities of the body with the expansion of the interstitial liquid volume (*Kinsky et al., 1998*).