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Anesthetic Management of Pediatric Burned Patients

Easy

Submitted for Partial Fulfillment of the Master Degree in
Anesthesia

By

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List of Abbreviations

ABCDE	Airway, breathing, circulation, disability, exposure.
ADH	Antidiuretic hormone.
ALI	Acute lung injury.
ALT	Alanine transferase.
ARDS	Acute respiratory distress syndrome.
AST	Aspartat transferase.
ATP	Adenosine triphosphate.
ATPase	Adenosine triphosphatase.
BDC's	Burn dressing changes.
BEE	Based energy expenditure.
BSA	Body surface area.
BUN	Blood urea nitrogen.
C.V.P	Central venous pressure.
Ca⁺⁺	Calcium.
CBC	Complete blood picture.
CHEOPS	Children Hospital of Eastern Ontario pain scale.
CHO	Carbohydrate.
CO	Carbon monoxide.
CoHb	Carboxy hemoglobin.
COP	Cardiac output.
CRF	Cortico trophin-releasing factor.
CT	Computed tomography.
D5	Dextrose 5%.
DIC	Disseminated intravascular co-agulation.
DSW	Dextrose 5% in water.
ECG	Electrocardiogram.
EFF	Enteral feeding formulation.
ETT	Endotracheal tube.
FFB	Fresh frozen plasma.
FIO₂	Fraction of insprated oxygen.
H	Height (cm).

H₂O₂	Hydrogen peroxide.
HES	Hydroxy ethyl starch.
HTS	Hypertonic saline.
IBW	Ideal body weight.
ICU	Intensive care unit.
IGF-1	Insulin like growth factor type I.
IL-1	Interlukin-1.
IL-6	Interleukin- 6.
IL-8	Interlukin-8.
IM	Intra muscular.
iNOS	Inducible nitric oxide synthase.
IV	Intravenous.
Kcal	Kilo calorie.
L-R	Lactated ringer.
LT	Leukotriens.
LTV	Low-tidal volume ventilation.
MAC	Minimum alveolar concentration.
MCT	Medium chain triglycerides.
N	Nitrogen.
Na⁺	Sodium.
NDMR	Non-depolarizing muscle relaxants.
NF	Nuclear factor.
NMDA	N-methyl-D-aspartate.
nNos	Neuronal nitric oxide synthase.
NO	Nitric oxide.
NPC	Nonprotein calorie.
NRS	Numerical rating score.
NSAIDs	Non-steroidal anti-inflammatory drugs.
O⁻²	Super oxide anion.
OH⁻	Hydroxyl ion.
OR	Operating room.
PA	Pulmonary artery.
PAF	Platelet aggregation (or activating) factor.

PaO₂	Arterial partial pressure of oxygen.
PARP	Poly (ADP ribose) polymerase.
PCA	Patient-controlled analgesia.
PCWP	Pulmonary capillary wedge pressure.
PEEP	Positive end expiratory pressure.
PGE2	Prostaglandin E2.
PGI2	Prostacyclin.
PN	Parenteral nutrition.
PPN	Partial parenteral nutrition.
RBC	Red blood cell.
RDA	Recommended dietary allowance.
RNS	Reactive nitrogen species.
ROS	Reactive oxygen species.
RQ	Respiratory quotient.
SIR	Systemic inflammatory response.
SIRS	Systemic inflammatory response syndrome.
SPI	Soy protein isolates.
SPS	Soy polysacharid.
SVC	Superior vena cava.
SVR	Systemic vascular resistance.
TBSA	Total body surface area.
TEE	Total energy expenditure.
TIVA	Total intravenous anesthesia
TLA	Tumescent local anaesthesia.
TNF-α	Tumour necrosis factor alpha.
TPN	Total parenteral nutrition.
TXA2	Thromboxan A2.
TXB2	Thromboxane B2.
UUN	Urinary urea nitrogen.
VAS	Visual analogue scale.
$\dot{V}CO_2$	Carbon dioxide production.
$\dot{V}O_2$	Oxygen Consumption.
W	Weight (Kg).



INTRODUCTION AND AIM OF THE WORK

Introduction

Severe burn injury is a leading cause of life-threatening pediatric trauma. The anesthesiologist will be a member of the interdisciplinary team caring for these patients at the site of the accident, during transport, in the emergency room, or in a specialized burn unit. The anesthesiologist's tasks include resuscitation in the initial phase, providing safe anesthesia even for unstable patients during surgical procedures, and analgesic treatment during the rehabilitative period. (*Beushausen and Mucke, 1997*).

A burn is defined as any destruction of skin or body tissues resulting from heat, chemicals, radiation or electricity. Inhalation injury contributes to increased morbidity and mortality from burn injury in pediatric patients. Children with inhalation injury should be treated aggressively with supportive measures and pulmonary toilet (*Fidkowski et al, 2009*).

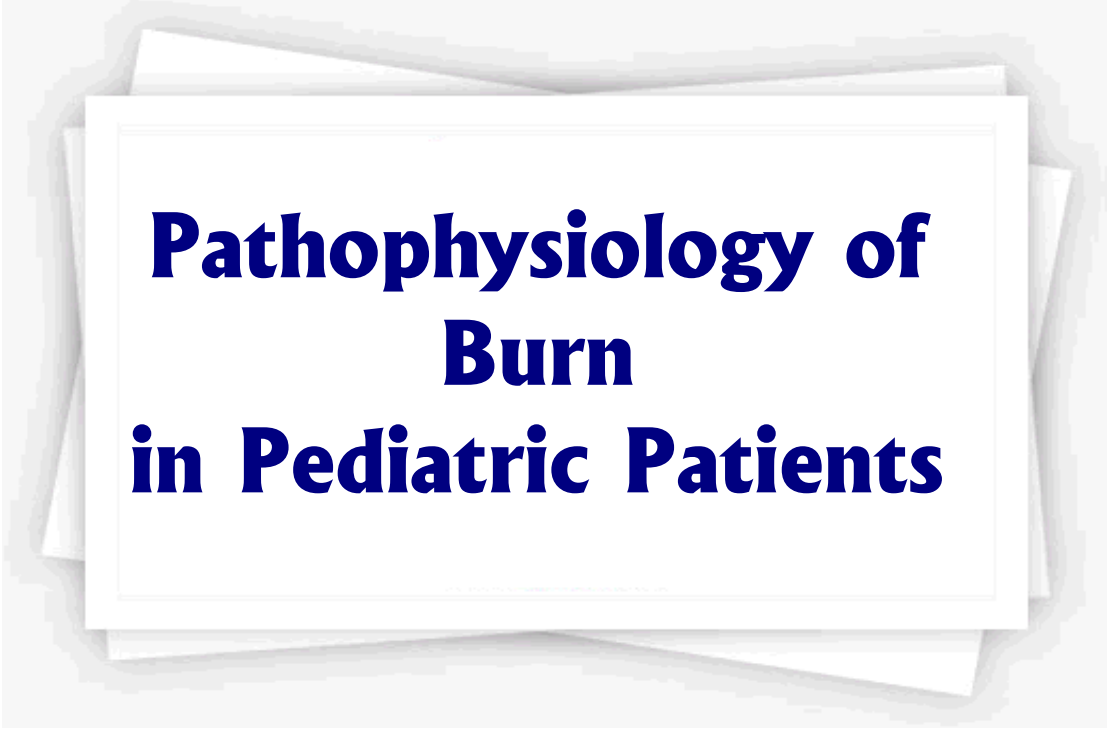
Major advances have been made in early resuscitation, respiratory care, treatment of inhalation injury, control of infection, modulation of the hypermetabolic response, and nutritional support. The biggest impact on survival, however, has been the change in the anesthetic approach and burn wound treatment (*Michael et al, 2005*).

Burns in pediatric patients create many diagnostic and therapeutic problems not seen in adults. These include correct estimation of burn size and depth, fluid resuscitation and fluid maintenance, vascular access, airway management, nutritional support, and prevention of sepsis. A child is not just a small adult, but a person who is even more devastated by burn injury, and who is less able to respond to it (*Purdue et al., 2002*).

Aim of the Work

The aim of this work is to review the current medical literature addressing the anesthetic management of pediatric burn patients, including the pathophysiological aspects of burns, initial management of burned children and perioperative management of burned children subjected to surgical interventions.

Chapter I

A graphic of a stack of papers with a white card on top. The card has a thin black border and contains the chapter title in a bold, dark blue serif font. The papers behind the card are slightly offset, creating a sense of depth.

Pathophysiology of Burn in Pediatric Patients

Chapter (1)

Pathophysiology of Burn in Pediatric Patients

Skin is the largest organ in the body, forms 0.8% of the total body mass, and its surface area varies with height and weight. Its thickness ranges from 1.5- 4.0 mm; these variations reflect maturation, ageing, and regional specializations (*Desanti, 2005*).

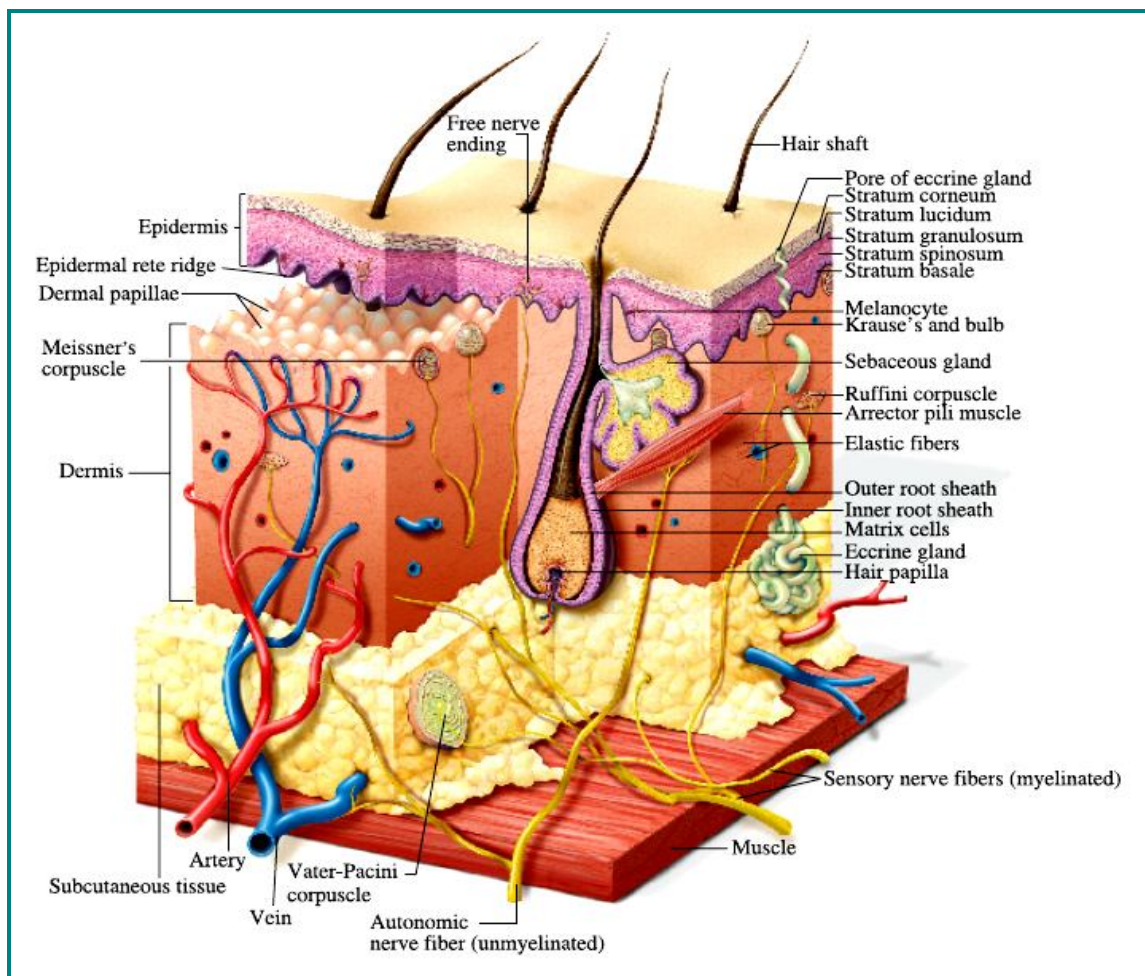


Fig. (1): Anatomy of normal skin. From Desanti (2005).

Skin Function:

Understanding a burn injury requires recognition of the anatomy and physiology of the skin. The skin is a bilayer organ (Figure 1) with many protective functions essential for survival (Table 1). The outer epidermal layer provides critical barrier functions and is composed of an outer layer of dead cells and keratin, which presents a barrier to bacterial and environmental toxins. The basal epidermal cells supply the source of new epidermal cells. The undulating surface of the epidermis, called rete pegs, increases adherence of the epidermis to the dermis via the basement membrane (*Desanti, 2005*).

Table (1): Skin functions

Epidermis
<ul style="list-style-type: none">▪ Protection from desiccation▪ Protection from bacterial entry▪ Protection from toxins▪ Fluid balance (preventing excess evaporative loss)▪ Neurosensory▪ Social-interactive
Dermis
<ul style="list-style-type: none">▪ Protection from trauma (due to elasticity and durability properties of the dermis)▪ Fluid balance through regulation of skin blood flow▪ Thermoregulation through control of skin blood flow▪ Growth factors for epidermal replication and dermal repair

Adapted from Desanti (2005)

The inner dermal layer has a number of essential functions, including continued restoration of the epidermis. The dermis is divided into the papillary dermis and the reticular dermis. The former is extremely bioactive, the latter less bioactive. This difference in bioactivity within the dermis is the reason that superficial partial-thickness burns generally heal faster than deeper partial-thickness burns; the papillary component is lost in the deeper burns. Loss of the normal skin barrier function causes the common complications of burn injury. These include infection, loss of body heat, increased evaporative water loss, and change in key interactive functions such as touch and appearance (Table 2) (*Desanti, 2005*).

Table (2): Complications of burn-induced skin barrier loss

- | |
|---|
| <ul style="list-style-type: none">▪ Increased heat loss (hypothermia)▪ Increased infection risk▪ Wound desiccation▪ Increased evaporative water loss▪ Loss of sensation or hyperalgesia▪ Loss of skin elasticity |
|---|

Adapted from Desanti (2005)