

Anaesthetic management of patients with severe sepsis

An Essay

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Anaesthesiology*

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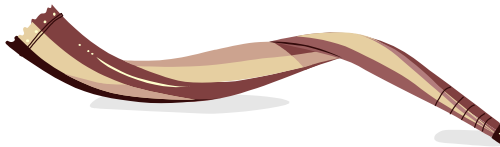
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Mary Meshmesh Samy

List of Abbreviations

ABC	Airway, breathing, and circulation.
ABG	Arterial blood gases.
ACEIs	Angiotensin-converting enzyme inhibitors.
ACTH	Adrenocorticotrophic Hormone.
ADAMTS-13	Thrombospondin type 1 domain 13
ALI	Acute lung injury.
ALT	Alanine aminotransferase.
APACHE II	Acute Physiology and Chronic Health Evaluation II
APC	Activated protein c
aPTT	Activated partial thromboplastin time
ARBs	Angiotensin receptor blockers.
ARDS	Acute Respiratory Distress Syndrome.
ARF	Acute renal failure
AST	Aspartate aminotransferase.
AT-III	Antithrombin-III
ATP	Adenosine triphosphate
AVP	Arginine vasopressin.
BK	Bradykinin.
BP	Blood Pressure.
BUN	Blood Urea Nitrogen.
C ⁻ vO ₂	Mixed venous oxygen content.
CaO ₂	Arterial oxygen content.
CD	Cluster of differentiation.
CECs	Circulating ECs
cGMP	Cyclic guanosine monophosphate
CNB	Central neuraxial block.

CNS	Central nervous system.
CO	cardiac output
CRF	Chronic Renal Failure.
CRP	C-reactive protein.
CRRT	Continuous renal replacement therapy.
CRRT	Continuous renal replacement therapy.
CSF	Cerebrospinal fluid.
CT	Computed tomography.
CVC	Central venous catheter.
CVO	Circumventricular organs.
CVP	Central venous pressure.
DIC	Disseminated Intravascular Coagulation.
DNA	Deoxyribonucleic acid.
DO ₂	Oxygen delivery
E. coli	Escherichia coli
EC	Endothelial cells
ECG	Electrocardiography.
ECHO	Echocardiography.
EDRF	Endothelium Derived Relaxing Factor.
EPCR	Endothelial PC receptor
ER	Emergency room
ERCP	Endoscopic retrograde cholangiopancreatography.
ESBL	Extended-spectrum beta-lactamase.
ET-1	Endothelin-1
FiO ₂	Fraction of inspired oxygen
FIX	Factor 9
FIXa	Active factor 9
FVII(a)	Active factor 7
FX	Factor 10

FXIIIa	Active factor 13
GAGs	Glycosaminoglycans
GI	Gastrointestinal.
HBO	Hyperbaric oxygen.
HES	Hydroxyethyl starch.
Hgb	Hemoglobin concentration.
HR	Heart rate.
HSPGs	HS polysaccharides
ICP	Intracranial pressure.
ICU	Intensive care unit
IHD	Intermittent haemodialysis.
IL-1ra	IL-1 receptor antagonist
ILs	Interleukins.
INR	International normalized ratio
IRRT	Intermittent renal replacement therapy.
IV	Intravenous.
LBP	LPS-binding protein
LP	Lumbar puncture.
LPS	Lipopolysaccharide.
LPS	Lipoproteins
MAC	Minimum alveolar concentration.
MAO	Monoamine oxidase.
MAP	Mean arterial pressure.
MAP	Mean arterial blood pressure.
MB	Methylene blue.
MIF	Macrophage migration inhibitory factor
MODS	Multi Organ Dysfunction Syndrome.
MPs	Microparticles
MRI	Magnetic resonance imaging.
MRSA	Methicillin-resistant Staph. aureus.

MVO ₂	Mixed venous oxygen saturation.
NAC	N-acetylcysteine.
NF-κB	Nuclear factor-κB.
NO	Nitric oxide
NSAIDs	Non steroidal anti-inflammatory drugs.
OR	Operating room.
OR	Odds ratio
PaCO ₂	Arterial partial pressure of CO ₂
PAI-1	Plasminogen activator inhibitor-1
PaO ₂	Partial pressure of arterial O ₂ .
PAOP	Pulmonary artery occlusion pressure.
PARs	Protease-activated receptors
PBMC	Peripheral blood mononuclear cells.
PC	Protein C
PCWP	Pulmonary Capillary Wedge Pressure.
PEEP	Positive end-expiratory pressure.
PG	Prostaglandins
pGE ₂	Prostaglandin E ₂ .
PMNs	Polymorph-nuclear neutrophils
PT	Prothrombin time.
RAS	Renin-angiotensin system.
rhAPC	Recombinant human activated protein C.
ROS	Reactive oxygen species
RR	Relative risk.
S ⁻ vO ₂	Mixed venous oxygen saturation
SaO ₂	Arterial oxygen saturation.
SaO ₂	Oxygen saturation of arterial blood.
SBP	Systolic blood pressure.
SBP	Systolic blood pressure
ScvO ₂	Central venous oxygen saturation

SD	Standard deviation
SIRS	Systemic inflammatory response syndrome
SSRI	Selective serotonin reuptake inhibitor.
SvO ₂	Venous oxygen saturation.
Svo ₂	Venous oxygen saturation
TAFI	Thrombin-activatable fibrinolysis inhibitor
TF	Tissue factor
TFPI	TF pathway inhibitor
TLRs	Toll-like receptors.
TM	Thrombomodulin
TNFR	TNF receptors
TNF- α	Tumor Necrosis Factor-alpha.
t-PA	Tissue plasminogen activator
TPN	Total parenteral nutrition.
TX	Thromboxane
TXA ₂	Thromboxane A ₂
uPA	Urokinase-type plasminogen activator
UTI	Urinary tract infection.
Va	Active factor 5
VALI	Ventilator-associated lung injury.
VIIIa	Active factor 8
Vo ₂	Oxygen consumption.
VS	Vasoplegic syndrome.
vs	Versus.
vWF	von Wellbrand factor.
WBC	White blood cell count.

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Introduction

Despite recent advances in the management of severe sepsis and septic shock, this condition continues to be a leading cause of death worldwide. Some experts usually consider sepsis as one of the most challenging syndromes because of its multiple presentations and the variety of its complications (*Ricardo, 2012*).

The American college of Chest Physicians and The Society for Critical Care Medicine defined sepsis as an infection syndrome involving two or more manifestations of systemic inflammatory response syndrome which include temperature more than 38 degrees or less than 36 degrees , heart rate more than 90 beats/min, respiratory rate more than 20 breaths/min. or PaCO₂ less than 32 mmHg and white blood cell count more than 12000/micro liter , less than 4000/micro liter or more than 10 % immature (band) forms. Septic shock is an increasingly severe sequela of sepsis involving hypotension despite adequate fluid resuscitation as well as the presence of perfusion abnormalities or organ dysfunction (*Clive et al., 2008*).

Anaesthetists play a central role in the multidisciplinary management of patients with severe sepsis from their initial deterioration at ward level, transfer to the diagnostic imaging suite, and intra-operative management for emergency surgery. Preoperative resuscitation, aimed at optimizing major organ

perfusion, is based on judicious use of fluids, vasopressors, and inotropes. Intra-operative anaesthesia management requires careful induction and maintenance of anaesthesia, optimizing intravascular volume status, avoidance of lung injury during mechanical ventilation, and ongoing monitoring of arterial blood gases, lactate concentration, haematological and renal indices, and electrolyte levels. Postoperative care overlaps with ongoing management of the severe sepsis syndrome patient in the intensive care unit (*Eissa et al., 2010*).

These patients are by definition, high risk, already requiring multiple supports, and require experienced and skillful decision-making to optimize their chances of a favorable outcome. Similar to acute myocardial infarction, stroke, or acute trauma, the initial hours (golden hours) of clinical management of severe sepsis represent an important opportunity to reduce morbidity and mortality. Rapid clinical assessment, resuscitation and surgical management by a focused multidisciplinary team, and early effective antimicrobial therapy are the key components to improved patient outcome (*Johnson et al., 2011*).

Aim of the work

The aim of the work is to highlight the importance of anaesthetic assessing and managing the patients with severe sepsis undergoing surgical operation.

Definitions and epidemiology of Septic shock

Because varying definitions of sepsis and septic shock were used in the past, standardized definitions were produced by The American College of Chest Physicians and the Society for Critical Care Medicine Consensus Conference on Standardized Definitions of Sepsis in 1992. Sepsis is defined as an infection-induced syndrome involving 2 or more manifestations of systemic inflammatory response syndrome: (1) temperature > 38 degrees or < 36 degrees; (2) heart rate > 90 beats/min; (3) respiratory rate > 20 breaths/min or $\text{PaCO}_2 < 32$ mmHg; and (4) white blood cell count $> 12000/\text{microliter}$, $< 4000/\text{microliter}$ or $> 10\%$ immature (band) forms (*Clive et al., 2008*).

Septic shock is an increasingly severe sequela of sepsis involving hypotension despite adequate fluid resuscitation as well as the presence of perfusion abnormalities or organ dysfunction. The latter are evident in resultant lactic acidosis, oliguria, obtundation, and so forth (*Clive et al., 2008*).

This circulatory shock represents an imbalance between the body's oxygen demands and the available oxygen supply, and is principally of the distributive type, although cardiogenic and hypovolemic components may also be involved. Classically, septic shock is associated with a normal or high cardiac output and a low systemic vascular resistance, generalized changes in capillary permeability result in edema formation with 'third

spacing' and altered tissue function. While traditionally associated with bacterial infection, fungi or even viruses can represent the infecting pathogen (*James et al., 2008*).

Diagnostic criteria for sepsis

Documented or suspected infection with some of the following clinical signs or laboratory data

1. Infection: documented or suspected infection

2. Signs of systemic inflammation

(a) General parameters

- Fever (core temp. $>38.8^{\circ}\text{C}$).
- Hypothermia (core temp. $<36^{\circ}\text{C}$).
- Tachycardia (>90 beats\ min or >2 SD above the normal value for age).
- Tachypnea (>20 breaths \ min).
- Altered mental status.
- Significant edema or positive fluid balance (>20 ml\ kg over 24 h).
- Hyperglycemia (plasma glucose > 120 mg\dl or 7.7 mmol \L) in non-diabetic patients.

(b) Inflammatory parameters

- Leukocytosis (WBC count $> 12,000/\text{mm}^3$).
- Leukopenia (WBC count $< 4,000/\text{mm}^3$).
- Normal WBC count with $> 10\%$ immature forms.