

# COLLABORATION



THE OFFICIAL NEWSLETTER OF  
INTERNATIONAL PAN-ARAB CRITICAL CARE MEDICINE SOCIETY  
VOL. 1 ISSUE 1 APRIL 2011

## WELCOME TO ECCC-DUBAI 2011



Dr. Hussain Al-Rahma  
President - IPACCMS

### President Message:

On behalf of International Pan Arab Critical Care Medicine Society (IPACCMS) and Emirates Intensive Care Society (EICS) it gives us immense pleasure to invite you all to Emirates Critical Care Conference - Dubai. ECCC-Dubai2011 will be held in conjunction with the 3rd Asia Africa Conference of the World Federation of Societies of Intensive and Critical Care Medicine (WFSICCM), the 7th International Pan Arab Critical Care Medicine Society (IPACCMS) congress and the Dubai Neurocritical Care Conference. Emirates Critical Care Conference-Dubai 2011 focuses on leading-evidence

base science through informative thematic lectures, Pro-Con debates, workshops and an interactive meeting with the experts, poster presentations, and interactive exhibition. It is one of the most significant teambuilding events in critical care community in the Middle East, with more than 1400 attendees, 70 International faculty, and more than 120 sessions over 3 days. The Conference is where key leaders in the field shape the future of critical care medicine in Asia and Africa.



Khalid Shukri, MD  
Secretary-General  
IPACCMS

### The IPACCMS Journey continues

It's been 7 years since the foundation of IPACCMS, today we are in the 7th congress. our board of directors has increased to 32, our partnership and network with different societies around the globe has strengthened the Asia-Africa WFSICCM event has become a reality in its 3rd year. the Dubai event will be special as we meet leadership from WFSICCM, ESICM, SCCM, GSA, NCS with regional societies leadership as our theme this year: towards a new era of collaboration.



Prof. Stephen Mayer  
President Neuro Critical Care Society

### Neurocritical care society participates in IPACCMS Congress for the Second Time

Prof. Stephen Mayer, President Neurocritical care society will be joining the 7th International Pan Arab Critical care medicine in Dubai 2011., this is his second visit to the region since the 4th congress in Kuwait in 2008. his sessions include: Subarachnoid Hemorrhage :new concept, illuminating the comatose human brain, Neurological emergencies :time-brain and Electrical Rhythms in Brain Death. Members of Neurocritical Care society in the IPACCMS congress include: Katja Wartenberg-Germany, Tamer Abdelhak-USA, Khalid Shukri-KSA. The Neurocritical Care Society is a rapidly growing international organization composed of multiprofessional healthcare providers that are dedicated to improve the care and outcomes of patients with life-threatening neurological illnesses by promoting quality patient care, professional collaboration, research, training and advocacy. We hope that you take a moment to get to know the Neurocritical care society web pages:  
[www.neurocriticalcare.org](http://www.neurocriticalcare.org)

### World Federation of Societies of Intensive and Critical Care Medicine

The World Federation of Societies of Intensive and Critical Care Medicine (WFSICCM) was established in 1977 and is a membership organization comprised of National Societies of Intensive and Critical Care Medicine. The principle objective of the World Federation is to promote the highest standards of Intensive and Critical Care Medicine for all mankind, without discrimination. The WFSICCM now has a membership of over 55 Societies with a combined individual membership of over 55,000 intensive and critical care practitioners throughout the world.

The World Federation is governed by an elected Council of 16 members each of whom serve a term of office of 8 years. The leadership of the organisation is provided by an Executive Committee comprised of the President, Secretary General and Treasurer. The organisation is headquartered near London, UK

In seeking to promote the highest standards of Intensive and Critical Care Medicine for all mankind without discrimination, there is an increasing recognition that we have a growing responsibility to foster international collaboration

in intensive and critical medicine because of the growing international focus of disease. Disease knows no international boundaries. We want to be the global organization that can facilitate and enable collaborative efforts in research, training and education to raise standards of care and improve outcomes for patients

International Pan-Arab Critical Care Medicine society joined WFSICCM as full member in (2005)

Egyptian society critical care & emergency medicine (2009), Saudi critical care society (2009), Emirate intensive care society (2009)

Expected new societies from the arab region to enroll as full members in WFSICCM are: Lebanon, Oman, Kuwait and Tunisia.

WFSICCM has played a major part in promoting Critical care in Asia and Africa

Special thanks to Prof. Edgar Jimenez, President WFSICCM and the rest of the Council for their support

For more information on WFSICCM go to:  
<http://www.world-critical-care.org>



Edgar Jimenez, MD  
President - WFSICCM



**GSA**  
GLOBAL SEPSIS ALLIANCE



**Prof. Konrad Rienhart**  
Chairman Global Sepsis Alliance

## Why we need the Global Sepsis Alliance?

The Global Sepsis Alliance (GSA) is urging healthcare providers, patients and policy-makers worldwide to treat sepsis as a medical emergency. Tens of millions of people die from sepsis each year, making it the likely leading cause of death worldwide. Sepsis kills regardless of age, ethnicity, location and access to care it's imperative that we come together as a global community to address this enormous public health problem.

The GSA, which represents approximately 250,000 intensive and critical care physicians around the world, announced this call to action at the conclusion of the Merinoff Symposium, an international sepsis conference sponsored by the Feinstein Institute for Medical Research. Sepsis, the body's life-threatening response to infection, afflicts approximately 750,000 Americans each year and costs the health care system in the U.S. nearly \$17 billion. It causes more deaths per year than prostate cancer, breast cancer and HIV/AIDS combined. Globally, an estimated 18 million cases of sepsis occurs each year. In fact, experts in the field believe sepsis is actually responsible for the majority of the mortality associated with HIV/AIDS, malaria, tuberculosis, pneumonia and other infections acquired in the community, in health care settings, or by traumatic injury.

The GSA urges the medical community to recognize sepsis as a medical emergency requiring the administration of fluids, antibiotics and appropriate anti-infectives within one hour of suspicion of sepsis. We call for new studies to generate data demonstrating the efficacy of this approach and to generate data confirming sepsis as the common pathway to death and the leading cause of death worldwide. The GSA agreed on a public definition to help lay people and policymakers to better understand sepsis

*"Sepsis is a life threatening condition that arises when the body's response to an infection injures its own tissues and organs. Sepsis may lead to shock, multiple organ failure and death especially if not recognized early and treated promptly. Sepsis remains the primary cause of death from infection despite advances in modern medicine, including vaccines, antibiotics and acute care. Millions of people die of sepsis every year worldwide."*

Multiple surveys show that as high as 50-60 percent of individuals around the world are unfamiliar with sepsis. More specifically, a recent Feinstein Institute-sponsored survey of 1,000 Americans showed that 60 percent of respondents were not familiar with the term, and the lack of familiarity was greatest among seniors, men and African-Americans, all of whom are at increased risk of sepsis. A major aim of the GSA is to overcome the lack of awareness and understanding of sepsis as one of the major challenges we face in health care today.

Sepsis occurs more frequently in the young and the elderly, and in many hospitals, sepsis is the leading cause of death in non-coronary intensive care units. In addition, anti-cancer drugs frequently render oncology patients susceptible to infection, and sepsis is a major cause of death in this population. Focus on sepsis as an emergency is even more critical in the developing world where there are so few ICUs and sepsis is the cause of death for many as 7 out of 10 children worldwide. That is why it is so important that the World Federation of Pediatric Intensive and Critical Care is one of the founding organizations of the GSA.

Sepsis is under-recognized and poorly understood as a leading cause of death in the world due to confusion about its definition among patients and health care providers, lack of documentation of sepsis as a cause of death on death certificates, inadequate diagnostic tools, and inconsistent application of standardized clinical guidelines to treat sepsis. There is hope, that the GSA with its steadily increasing member organizations help to improve the availability of interventions (e.g., fluids, antibiotics and appropriate anti-infectives) that can dramatically alter the course of sepsis and improve survival if administered within the first hour of suspicion of sepsis.

For further information please view: [www.globalsepsisalliance.org](http://www.globalsepsisalliance.org)  
<http://my.molmed.org/merinoffvideo>

# GUIDELINES FOR MANAGEMENT OF SEVERE SEPSIS AND SEPTIC SHOCK



**Surviving Sepsis Campaign** This is a summary of the Surviving Sepsis Campaign *International Guidelines for Management of Severe Sepsis and Septic Shock: 2008*, condensed from Dellinger RP, Levy MM, Carlet JM, et al: *Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock. Critical Care Medicine (2008) 34:17-60 and Crit Care Med 2008; 36(1) 296-327.*

This version does not contain the rationale or appendices contained in the primary publication. The SSC guidelines do not cover every aspect of managing critically ill patients, and their application should be supplemented by generic best practice and specific treatment as required. Please refer to the guidelines for additional information at [www.survivingsepsis.org](http://www.survivingsepsis.org)

Strength of recommendation and quality of evidence have been assessed using the GRADE criteria, presented in brackets after each guideline.

For added clarity:

- ◆ Indicates a strong recommendation or "we recommend"
- ◇ Indicates a weak recommendation or "we suggest"

The Surviving Sepsis Campaign is a collaboration of the European Society of Intensive Care Medicine, the International Sepsis Forum, and the Society of Critical Care Medicine.



January 2008

SSC Guidelines have been endorsed by

American Association of Critical-Care Nurses  
American College of Chest Physicians  
American College of Emergency Physicians  
Canadian Critical Care Society  
European Society of Clinical Microbiology and Infectious Diseases  
European Society of Intensive Care Medicine  
European Respiratory Society  
Indian Society of Critical Care Medicine  
International Sepsis Forum  
Japanese Association for Acute Medicine  
Japanese Society of Intensive Care Medicine  
Society of Critical Care Medicine  
Society of Hospital Medicine  
Surgical Infection Society  
World Federation of Critical Care Nurses  
World Federation of Societies of Intensive and Critical Care Medicine.  
Participation and endorsement by German Sepsis Society and Latin American Sepsis Institute.

## Initial resuscitation (first 6 hours)

- ◆ Begin resuscitation immediately in patients with hypotension or elevated serum lactate  $\geq 4$  mmol/L; do not delay pending ICU admission. (1C)
- ◆ Resuscitation goals: (1C)
  - Central venous pressure (CVP) 8–12 mm Hg\*
  - Mean arterial pressure  $\geq 65$  mm Hg
  - Urine output  $\geq 0.5$  mL.kg<sup>-1</sup>.hr<sup>-1</sup>
  - Central venous (superior vena cava) oxygen saturation  $\geq 70\%$ , or mixed venous  $\geq 65\%$
- ◇ If venous O<sub>2</sub> saturation target not achieved: (2C)
  - consider further fluid
  - transfuse packed red blood cells if required to hematocrit of  $\geq 30\%$  and/or
  - dobutamine infusion max 20  $\mu\text{g.kg}^{-1}.\text{min}^{-1}$\* A higher target CVP of 12–15 mmHg is recommended in the presence of mechanical ventilation or pre-existing decreased ventricular compliance.

## Diagnosis

- ◆ Obtain appropriate cultures before starting antibiotics provided this does not significantly delay antimicrobial administration. (1C)
  - Obtain two or more blood cultures (BCs)
  - One or more BCs should be percutaneous
  - One BC from each vascular access device in place  $\geq 48$  hours
  - Culture other sites as clinically indicated
- ◆ Perform imaging studies promptly in order to confirm and sample any source of infection if safe to do so. (1C)

## Antibiotic therapy

- ◆ Begin intravenous antibiotics as early as possible, and always within the first hour of recognizing severe sepsis (1D) and septic shock. (1B)
- ◆ Broad-spectrum: one or more agents active against likely bacterial/fungal pathogens and with good penetration into presumed source. (1B)
- ◆ Reassess antimicrobial regimen daily to optimize efficacy, prevent resistance, avoid toxicity, & minimize costs. (1C)
- ◇ Consider combination therapy in Pseudomonas infections. (2D)
- ◇ Consider combination empiric therapy in neutropenic patients. (2D)
- ◇ Combination therapy no more than 3–5 days and de-escalation following susceptibilities. (2D)
- ◆ Duration of therapy typically limited to 7–10 days; longer if response slow, undrainable foci of infection, or immunologic deficiencies. (1D)
- ◆ Stop antimicrobial therapy if cause is found to be non-infectious. (1D)

## Source identification and control

- ◆ A specific anatomic site of infection should be established as rapidly as possible (1C) and within the first 6 hours of presentation. (1D)
- ◆ Formally evaluate patient for a focus of infection amenable to source control measures (eg: abscess drainage, tissue debridement). (1C)
- ◆ Implement source control measures as soon as possible following successful initial resuscitation. (1C)
  - ◇ Exception: infected pancreatic necrosis, where surgical intervention best delayed. (2B)
- ◆ Choose source control measure with maximum efficacy and minimal physiologic upset. (1D)
- ◆ Remove intravascular access devices if potentially infected. (1C)

## Fluid therapy

- ◆ Fluid-resuscitate using crystalloids or colloids. (1B)
- ◆ Target a CVP of  $\geq 8$  mmHg ( $\geq 12$  mmHg if mechanically ventilated). (1C)
- ◆ Use a fluid challenge technique while associated with a hemodynamic improvement. (1D)
- ◆ Give fluid challenges of 1000 mL of crystalloids or 300–500 mL of colloids over 30 minutes. More rapid and larger volumes may be required in sepsis-induced tissue hypoperfusion. (1D)
- ◆ Rate of fluid administration should be reduced if cardiac filling pressures increase without concurrent hemodynamic improvement. (1D)

## Vasopressors

- ◆ Maintain MAP  $\geq 65$  mmHg. (1C)
- ◆ Norepinephrine or dopamine centrally administered are the initial vasopressors of choice. (1C)

- ◆ Epinephrine, phenylephrine, or vasopressin should not be administered as the initial vasopressor in septic shock. (2C)
  - Vasopressin 0.03 units/min may be subsequently added to norepinephrine with anticipation of an effect equivalent to norepinephrine alone.
- ◇ Use epinephrine as the first alternative agent in septic shock when blood pressure is poorly responsive to norepinephrine or dopamine. (2B)
- ◆ Do not use low-dose dopamine for renal protection. (1A)
- ◆ In patients requiring vasopressors, insert an arterial catheter as soon as practical. (1D)

## Inotropic therapy

- ◆ Use dobutamine in patients with myocardial dysfunction as supported by elevated cardiac filling pressures and low cardiac output. (1C)
- ◆ Do not increase cardiac index to predetermined supranormal levels. (1B)

## Steroids

- ◆ Consider intravenous hydrocortisone for adult septic shock when hypotension responds poorly to adequate fluid resuscitation and vasopressors. (2C)
- ◇ ACTH stimulation test is not recommended to identify the subset of adults with septic shock who should receive hydrocortisone. (2B)
- ◇ Hydrocortisone is preferred to dexamethasone. (2B)
- ◇ Fludrocortisone (50  $\mu\text{g}$  orally once a day) may be included if an alternative to hydrocortisone is being used which lacks significant mineralocorticoid activity. Fludrocortisone is optional if hydrocortisone is used. (2C)
- ◇ Steroid therapy may be weaned once vasopressors are no longer required. (2D)
- ◆ Hydrocortisone dose should be  $\geq 300$  mg/day. (1A)
- ◆ Do not use corticosteroids to treat sepsis in the absence of shock unless the patient's endocrine or corticosteroid history warrants it. (1D)

## Recombinant human activated protein C (rhAPC)

- ◆ Consider rhAPC in adult patients with sepsis-induced organ dysfunction with clinical assessment of high risk of death (typically APACHE II  $\geq 25$  or multiple organ failure) if there are no contraindications. (2B; 2C for post-operative patients)
- ◆ Adult patients with severe sepsis and low risk of death (eg: APACHE II  $\leq 20$  or one organ failure) should not receive rhAPC. (1A)

## Blood product administration

- ◆ Give red blood cells when hemoglobin decreases to  $\leq 7.0$  g/dL ( $\leq 7.0$  g/L) to target a hemoglobin of 7.0–9.0 g/dL in adults. (1B)  
*A higher hemoglobin level may be required in special circumstances (eg: myocardial ischemia, severe hypoxemia, acute hemorrhage, cyanotic heart disease, or lactic acidosis)*
- ◆ Do not use erythropoietin to treat sepsis-related anemia. Erythropoietin may be used for other accepted reasons. (1B)
- ◇ Do not use fresh frozen plasma to correct laboratory clotting abnormalities unless there is bleeding or planned invasive procedures. (2D)
- ◆ Do not use antithrombin therapy. (1B)
- ◇ Administer platelets when: (2D)
  - counts are  $<50,000/\text{mm}^3$  ( $5 \times 10^9/\text{L}$ ) regardless of bleeding.
  - counts are  $50,000$  to  $30,000/\text{mm}^3$  ( $5$ – $30 \times 10^9/\text{L}$ ) and there is significant bleeding risk.
  - Higher platelet counts  $\geq 50,000/\text{mm}^3$  ( $50 \times 10^9/\text{L}$ ) are typically required for surgery or invasive procedures.

## Mechanical ventilation of sepsis-induced acute lung injury (ALI)/ARDS

- ◆ Target a tidal volume of 6 mL/kg (predicted) body weight in patients with ALI/ARDS. (1B)
- ◆ Target an initial upper limit plateau pressure  $\leq 30$  cmH<sub>2</sub>O. Consider chest wall compliance when assessing plateau pressure. (1C)
- ◆ Allow PaCO<sub>2</sub> to increase above normal, if needed, to minimize plateau pressures and tidal volumes. (1C)
- ◆ Positive end expiratory pressure (PEEP) should be set to avoid extensive lung collapse at end expiration. (1C)
- ◇ Consider using the prone position for ARDS patients requiring potentially injurious levels of FIO<sub>2</sub> or plateau pressure, provided they are not put at risk from positional changes. (2C)
- ◆ Maintain mechanically ventilated patients in a semi-recumbent position unless contraindicated. (1B)
  - ◇ Suggested target elevation 30–45 degrees. (2C)

- ◇ Noninvasive ventilation may be considered in the minority of ALI/ARDS patients with mild-moderate hypoxemic respiratory failure. The patients need to be hemodynamically stable, comfortable, easily arousable, able to protect/clear their airway, and expected to recover rapidly. (2B)
- ◆ Use a weaning protocol and a spontaneous breathing trial (SBT) regularly to evaluate the potential for discontinuing mechanical ventilation. (1A)
  - SBT options include a low level of pressure support with continuous positive airway pressure 5 cm H<sub>2</sub>O or a T-piece.
  - Before the SBT, patients should:
    - be arousable
    - be hemodynamically stable without vasopressors
    - have no new potentially serious conditions
    - have low ventilatory and end-expiratory pressure requirement
    - require FIO<sub>2</sub> levels that can be safely delivered with a face mask or nasal cannula
- ◆ Do not use a pulmonary artery catheter for the routine monitoring of patients with ALI/ARDS. (1A)
- ◆ Use a conservative fluid strategy for patients with established ALI who do not have evidence of tissue hypoperfusion. (1C)

## Sedation, analgesia, and neuromuscular blockade in sepsis

- ◆ Use sedation protocols with a sedation goal for critically ill mechanically ventilated patients. (1B)
- ◆ Use either intermittent bolus sedation or continuous infusion sedation to predetermined end points (sedation scales), with daily interruption/ lightening to produce awakening. Re-titrate if necessary. (1B)
- ◆ Avoid neuromuscular blockers where possible. Monitor depth of block with train-of-four when using continuous infusions. (1B)

## Glucose control

- ◆ Use IV insulin to control hyperglycemia in patients with severe sepsis following stabilization in the ICU. (1B)
- ◇ Aim to keep blood glucose  $\leq 8.3$  mmol/L (150 mg/dL) using a validated protocol for insulin dose adjustment. (2C)
- ◆ Provide a glucose calorie source and monitor blood glucose values every 1–2 hours (4 hours when stable) in patients receiving intravenous insulin. (1C)
- ◆ Interpret with caution low glucose levels obtained with point of care testing, as these techniques may overestimate arterial blood or plasma glucose values. (1B)

## Renal replacement

- ◇ Intermittent hemodialysis and continuous veno-venous hemofiltration (CVVH) are considered equivalent. (2B)
- ◇ CVVH offers easier management in hemodynamically unstable patients. (2D)

## Bicarbonate therapy

- ◆ Do not use bicarbonate therapy for the purpose of improving hemodynamics or reducing vasopressor requirements when treating hypoperfusion-induced lactic acidemia with pH  $\geq 7.15$ . (1B)

## Deep vein thrombosis (DVT) prophylaxis

- ◆ Use either low-dose unfractionated heparin (UFH) or low-molecular weight heparin (LMWH), unless contraindicated. (1A)
- ◆ Use a mechanical prophylactic device, such as compression stockings or an intermittent compression device, when heparin is contraindicated. (1A)
- ◇ Use a combination of pharmacologic and mechanical therapy for patients who are at very high risk for DVT. (2C)
- ◇ In patients at very high risk LMWH should be used rather than UFH. (2C)

## Stress ulcer prophylaxis

- ◆ Provide stress ulcer prophylaxis using H<sub>2</sub> blocker (1A) or proton pump inhibitor (1B). Benefits of prevention of upper GI bleed must be weighed against the potential for development of ventilator-acquired pneumonia.

## Consideration for limitation of support

- ◆ Discuss advance care planning with patients and families. Describe likely outcomes and set realistic expectations. (1D)

# INTRA-ABDOMINAL HYPERTENSION (IAH) ASSESSMENT ALGORITHM

- Patients should be screened for IAH/ACS risk factors upon ICU admission and with new or progressive organ failure.
- If two or more risk factors are present, a baseline IAP measurement should be obtained.
- If IAH is present, serial IAP measurements should be performed throughout the patient's critical illness.

Patient has TWO or more risk factors for IAH/ACS upon either ICU admission or in the presence of new or progressive organ failure

## Measure patient's IAP to establish baseline pressure

IAP measurements should be:

1. Expressed in mmHg (1 mmHg = 1.36 cm H<sub>2</sub>O)
2. Measured at end-expiration
3. Performed in the supine position
4. Zeroed at the iliac crest in the mid-axillary line
5. Performed with an instillation volume of no greater than 25 mL of saline [1 mL/kg for children up to 20 kg] (for bladder technique)
6. Measured 30-60 seconds after instillation to allow for bladder detrusor muscle relaxation (for bladder technique)
7. Measured in the absence of active abdominal muscle contractions

Sustained IAP  $\geq$  12 mmHg?

YES

NO

Patient has IAH

Patient does not have IAH

Notify patient's doctor of elevated IAP.  
Proceed to IAH / ACS management algorithm.

Observe patient.  
Recheck IAP if patient deteriorates clinically.

## Risk Factors for IAH / ACS

1. Diminished abdominal wall compliance
  - Acute respiratory failure, especially with elevated intrathoracic pressure
  - Abdominal surgery with primary fascial or tight closure
  - Major trauma / burns
  - Prone positioning, head of bed > 30 degrees
  - High body mass index (BMI), central obesity
2. Increased intra-luminal contents
  - Gastroparesis
  - Ileus
  - Colonic pseudo-obstruction
3. Increased abdominal contents
  - Hemoperitoneum / pneumoperitoneum
  - Ascites / liver dysfunction
4. Capillary leak / fluid resuscitation
  - Acidosis (pH < 7.2)
  - Hypotension
  - Hypothermia (core temperature < 33°C)
  - Polytransfusion (>10 units of blood / 24 hrs)
  - Coagulopathy (platelets < 55000 / mm<sup>3</sup> OR prothrombin time (PT) > 15 seconds OR partial thromboplastin time (PTT) > 2 times normal OR international standardised ratio (INR) > 1.5)
  - Massive fluid resuscitation (> 5 L / 24 hours)
  - Pancreatitis
  - Oliguria
  - Sepsis
  - Major trauma / burns
  - Damage control laparotomy

## IAH Grading

Grade I	IAP 12-15 mmHg
Grade II	IAP 16-20 mmHg
Grade III	IAP 21-25 mmHg
Grade IV	IAP $\geq$ 25 mmHg

## Abbreviations

IAH - intra-abdominal hypertension  
ACS - abdominal compartment syndrome  
IAP - intra-abdominal pressure

Adapted from *Intensive Care Medicine* 2006;32(11):1722-1732 & 2007;33(6):951-962  
© 2007 World Society of the Abdominal Compartment Syndrome. All rights reserved.



**World Society of the Abdominal Compartment Syndrome (WSACS)**

ZNA Stuivenberg, Lange Beeldekenstraat 267, B-2060 Antwerpen 6, Belgium  
Tel: +32 3 2177092 Fax: +32 3 2177279 e-mail: info@wsacs.org  
Website: <http://www.wsacs.org>