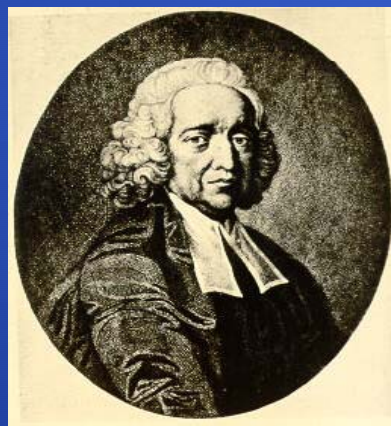


CVP Pro

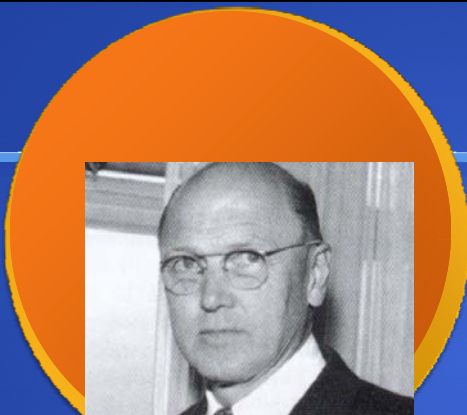
Fahad Bafaqeeh, MD, FCCP, SBIM
Deputy Director of Intensive Care Department
Riyadh Military Hospital



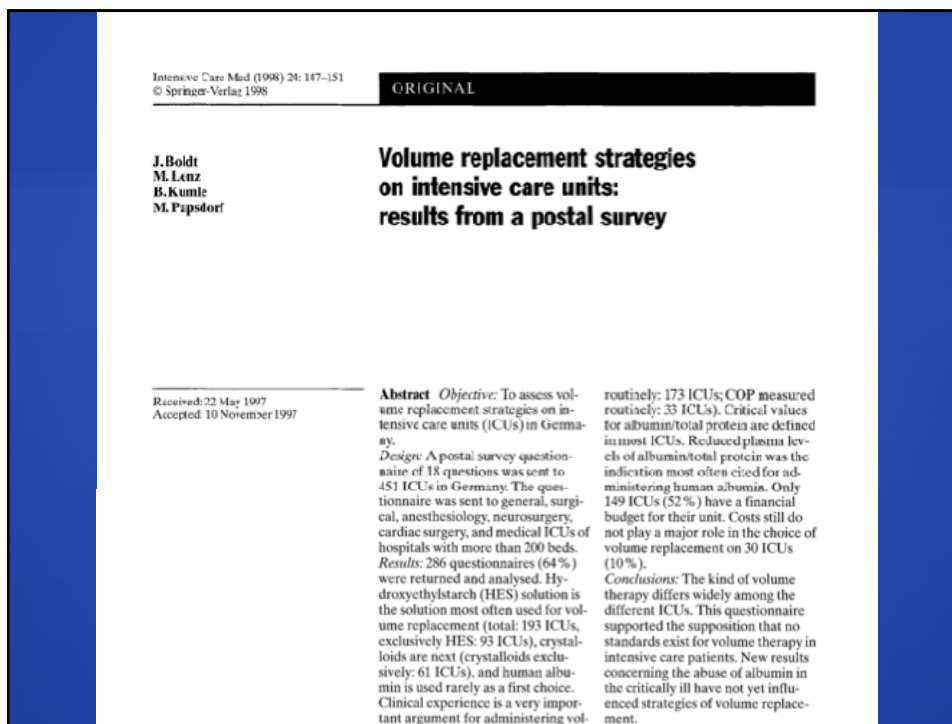
History



29/04/2010



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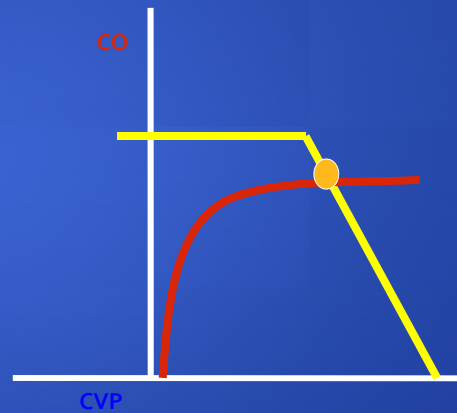


- CVP monitoring is used in more than 90% of the cases as an indicator of fluid therapy.
- CVP was the most frequent parameter used to assess fluid need.
- Boldt J, Lenz M, Kumle B, Papsdorf M. Volume replacement strategies on intensive care units: results from a postal survey. Intensive Care Med. 1998;24:147-151.

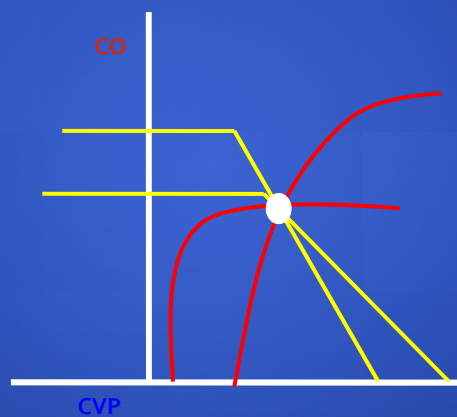
Physiology



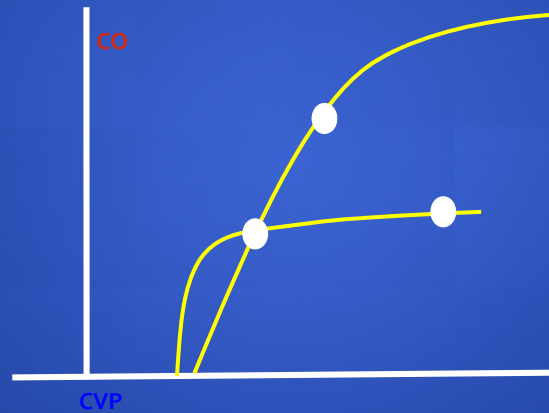
- CVP is determined by the interaction of cardiac function and return function and a change in either can alter the CVP



A single CVP measurement



Two is better than one



Can we use these observations to predict fluid responsiveness

ORIGINAL RESEARCH

The Clinical Role of Central Venous Pressure Measurements

Sheldon Magder, MD¹
Fahad Bafaqeeh, MD²

Central venous pressure (CVP) is commonly measured, but its clinical use is still not clear. We argue that the interpretation of the CVP needs to be considered in conjunction with an assessment of cardiac output. The objective of this study was to define an elevated CVP as one in which there is a low probability for cardiac output to increase with a volume infusion through a Starling mechanism by relating the initial CVP (measured relative to a reference point 5 cm below the sternal angle) to the response in cardiac output with volume infusion. The authors studied consecutive patients who had pulmonary artery catheters in place and who had a volume challenge as part of routine management as ordered by the treating physician. To ensure an adequate test of the Starling mechanism, data were included only if the volume infusion increased CVP by ≥ 2 mm Hg. Responders were defined a priori as those with an increase in cardiac index ≥ 300 and nonresponders as <300 mL/min/m². Patients failed to respond to volume infusion at all CVP values, and even 25% of those with CVP ≤ 5 mm Hg were nonresponders. However, when CVP was >10 mm Hg, physicians prescribed less fluid challenges, and when they did, a positive response was much less likely. Change in blood pressure or changes in urine output with volume infusion correlated poorly with change in cardiac index. A CVP of >10 mm Hg should be considered high, and the probability of an increase in cardiac output with volume infusion is low. This value is a reasonable upper limit for algorithms for empiric fluid challenges.

Key words: volume challenge, cardiac output, right atrial pressure

preload for the purpose of improving cardiac output and tissue perfusion. However, there is still debate about the best clinical indicators for this optimization. Central venous pressure (CVP) or right atrial pressure (Pra) have long been used for this purpose. The terms CVP and Pra are essentially equivalent as long as there is no vena caval obstruction, and we will use the term CVP throughout this article. CVP is readily available in any patient with a central venous catheter and, even without a central venous catheter, in most patients, CVP can be assessed easily by simple inspection of the jugular veins. Indeed, this is one of the basic bedside skills taught to all medical students [1]. A postal survey of German intensivists found that 93% of them used the CVP as an indicator for volume replacement, and CVP was by far the major indicator used [2]. Yet the value of this simple clinical measurement is frequently criticized. A specific value of CVP is a poor predictor of changes in cardiac output with volume infusion [3-5], and the CVP does not accurately reflect pulmonary artery occlusion pressure, which is seen by many to be the true determinant of cardiac preload [6,7]. We argue that this failure to find a useful clinical utility for the CVP is due to a failure to consider the physiological determinants of the CVP.

CVP is determined by the interaction of cardiac function and the function that determines the return of blood to the heart [8-10]. Thus, the CVP by itself



- 83 patients were enrolled in this study.
- Their hemodynamic data were collected before and after any fluid challenge.
- 118 fluid challenges attempt were done and were successful in increasing the CVP by more than one

Magder S, Bafaqeeh F. The clinical role of central venous pressure measurements. J Intensive Care Med. 2007;22:44-51.

Results



Table 2. Hemodynamic Profile Prior to Volume Infusion of Responders and Nonresponders

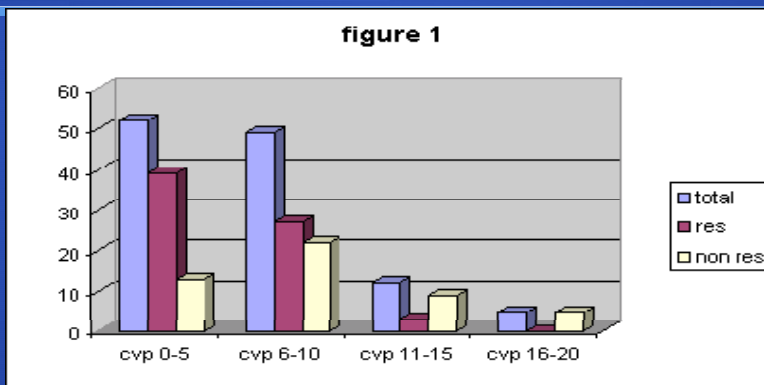
| | Total | Responders | Nonresponders | P Value |
|--|-------------|-------------|---------------|---------|
| Central venous pressure, mm Hg | 7.0 ± 1.7 | 5.9 ± 2.7 | 8.7 ± 4.2 | <.001 |
| Pulmonary artery occlusion pressure, mm Hg | 10.0 ± 2.4 | 9.7 ± 3.1 | 12.1 ± 4.5 | <.001 |
| Heart rate, beats/min | 86 ± 16 | 85 ± 15 | 90 ± 17 | |
| Systolic blood pressure, mm Hg | 109 ± 12 | 110 ± 12 | 107 ± 12 | |
| Diastolic blood pressure, mm Hg | 55 ± 10 | 54 ± 9 | 55 ± 11 | |
| Mean arterial pressure, mm Hg | 73 ± 9 | 73 ± 8 | 73 ± 10 | |
| Systolic pulmonary artery pressure, mm Hg | 31 ± 10 | 30 ± 10 | 32 ± 10 | |
| Diastolic pulmonary artery pressure, mm Hg | 14 ± 14 | 13 ± 4 | 15.14 ± 4.32 | <.001 |
| Mean pulmonary artery pressure, mm Hg | 20 ± 6 | 19 ± 6 | 20.95 ± 5.66 | <.05 |
| Cardiac index, L/min/m ² | 2.59 ± 0.82 | 2.42 ± 0.75 | 2.83 ± 0.86 | <.001 |
| Urine output, mL/h | 104 ± 148 | 119 ± 176 | 82 ± 92 | |
| Ejection fraction, % | 48.9 ± 15.7 | 51.3 ± 13.7 | 45.2 ± 17.8 | <.05 |
| Ventilated ^a | 90 (76) | 51 (74) | 39 (80) | |
| Vasopressors ^b | 80 (68) | 46 (67) | 34 (69) | |

Magder S, Bafaqeh F. The clinical role of central venous pressure measurements. J Intensive Care Med. 2007;22:44-51.

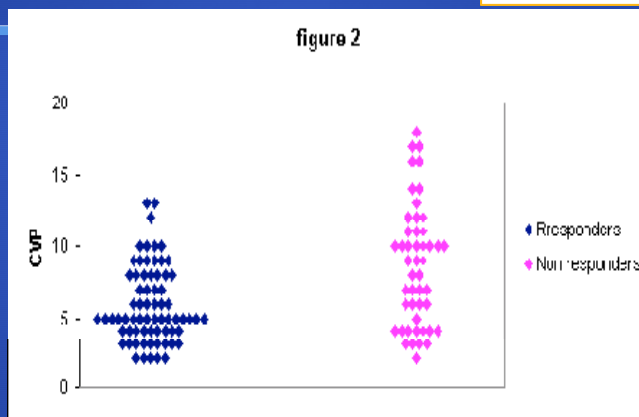
Results



figure 1



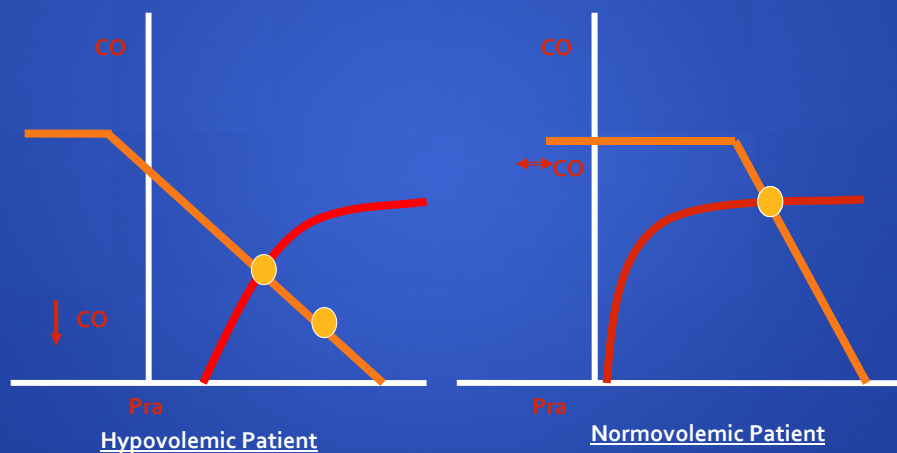
Magder S, Bafaqeh F. The clinical role of central venous pressure measurements. J Intensive Care Med. 2007;22:44-51.



Magder S, Bafaqeh F. The clinical role of central venous pressure measurements. J Intensive Care Med. 2007;22:44-51.

Heart-Lung Interaction

Effect of Rise of ITP on CO/VR Curves



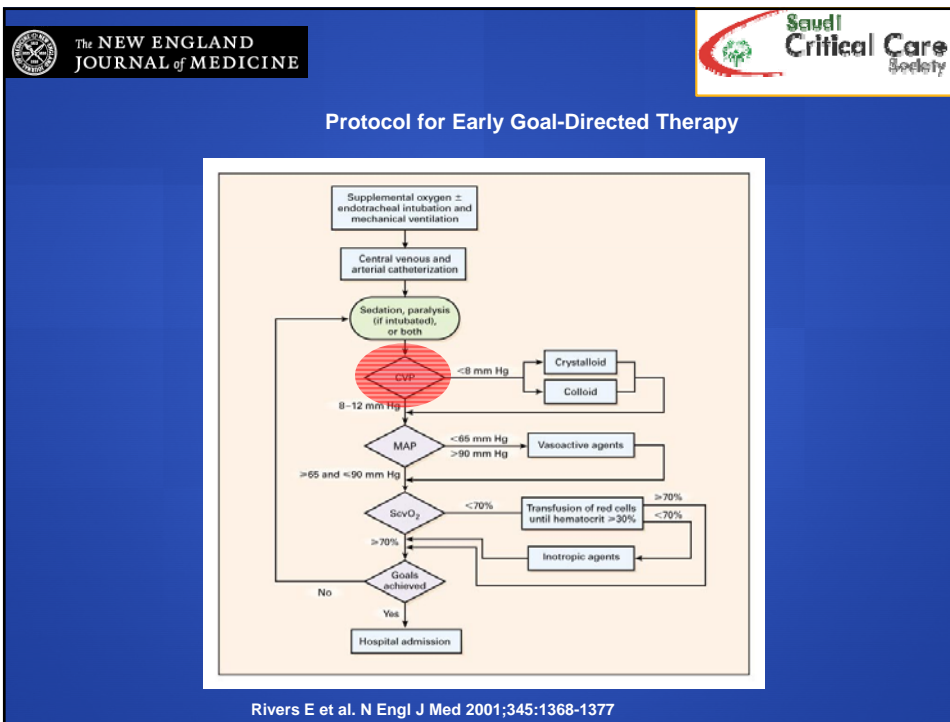


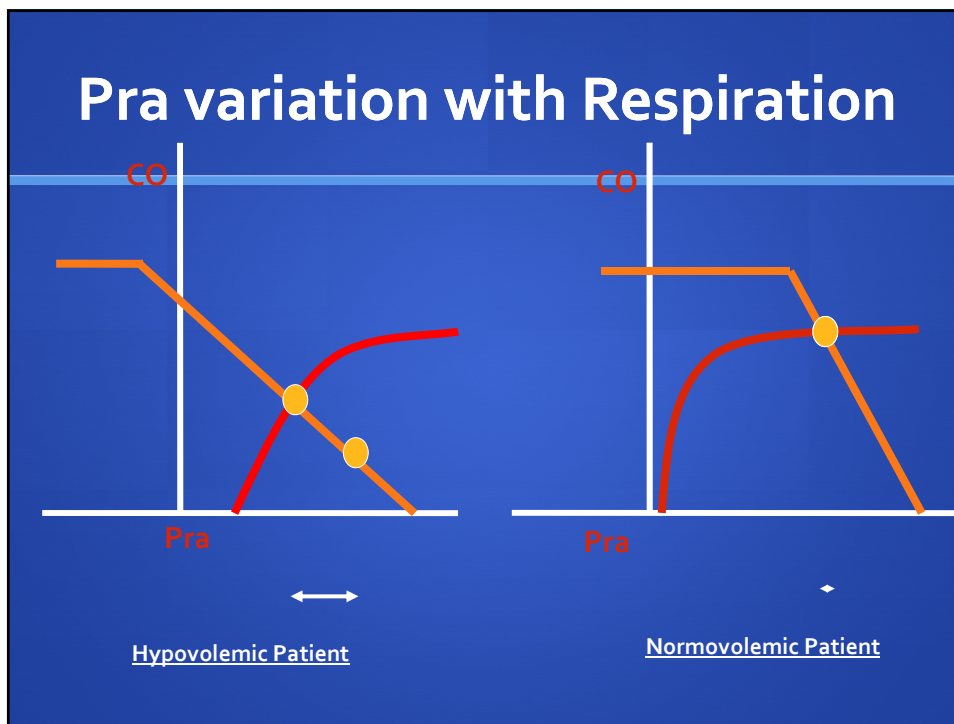
TABLE 3. KAPLAN-MEIER ESTIMATES OF MORTALITY AND CAUSES OF IN-HOSPITAL DEATH.*

| VARIABLE | STANDARD THERAPY (N=133) | EARLY GOAL-DIRECTED THERAPY (N=130) | RELATIVE RISK (95% CI) | P VALUE |
|--------------------------------|--------------------------|-------------------------------------|------------------------|---------|
| | n.c. (%) | | | |
| In-hospital mortality† | | | | |
| All patients | 59 (46.5) | 38 (30.5) | 0.58 (0.38–0.87) | 0.009 |
| Patients with severe sepsis | 19 (30.0) | 9 (14.9) | 0.46 (0.21–1.03) | 0.06 |
| Patients with septic shock | 40 (56.8) | 29 (42.3) | 0.60 (0.36–0.98) | 0.04 |
| Patients with sepsis syndrome | 44 (45.4) | 35 (35.1) | 0.66 (0.42–1.04) | 0.07 |
| 28-Day mortality† | 61 (49.2) | 40 (33.3) | 0.58 (0.39–0.87) | 0.01 |
| 60-Day mortality† | 70 (56.9) | 50 (44.3) | 0.67 (0.46–0.96) | 0.03 |
| Causes of in-hospital death‡ | | | | |
| Sudden cardiovascular collapse | 25/119 (21.0) | 12/117 (10.3) | — | 0.02 |
| Multiorgan failure | 26/119 (21.8) | 19/117 (16.2) | — | 0.27 |

*CI denotes confidence interval. Dashes indicate that the relative risk is not applicable.

†Percentages were calculated by the Kaplan-Meier product limit method.

‡The denominators indicate the numbers of patients in each group who completed the initial six-hour study period.



Pra variation with Respiration

- 35 spontaneously breathing patients were prospectively observed for Pra variation with respiration. Then they were given a bolus of fluids.

J Critical Care 1992, 7: 76

Magder SA, Georgiadis G, Cheong T. Respiratory variations in right atrial pressure predict response to fluid challenge. J Crit Care 1992; 20:29-42

| CVP Fluctuation | Total | Responders | Non-Responders |
|-------------------|-------|------------|----------------|
| CVP Fluctuate | 19 | 16 | 3 |
| CVP not Fluctuate | 14 | 1 | 13 |

THE NEW ENGLAND JOURNAL OF MEDICINE

ORIGINAL ARTICLE

Comparison of Two Fluid-Management Strategies in Acute Lung Injury

The National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network*

ABSTRACT

BACKGROUND: Optimal fluid management in patients with acute lung injury is unknown. Diuresis or fluid restriction may improve lung function but could jeopardize extrapulmonary-organ perfusion.

METHODS: In a randomized study, we compared a conservative and a liberal strategy of fluid management using explicit protocols applied for seven days in 1000 patients with acute lung injury. The primary end point was death at 60 days. Secondary end points included the number of ventilator-free days and organ-failure-free days and measures of lung physiology.

RESULTS: The rate of death at 60 days was 25.5 percent in the conservative-strategy group and 28.4 percent in the liberal-strategy group (P=0.30; 95 percent confidence interval for the difference, -2.6 to 8.4 percent). The mean (±SE) cumulative fluid balance during the first seven days was -136±491 ml in the conservative-strategy group and 692±502 ml in the liberal-strategy group (P<0.001). As compared with the liberal strategy, the conservative strategy improved the oxygenation index (mean airway pressure × the ratio of the fraction of inspired oxygen to the partial pressure of arterial oxygen) × 100 and the lung injury score and increased the number of ventilator-free days (14.6±0.5 vs. 12.1±0.5, P<0.001) and days not spent in the intensive care unit (13.4±0.4 vs. 11.2±0.4, P<0.001) during the first 28 days but did not increase the incidence or prevalence of shock during the study or the use of dialysis during

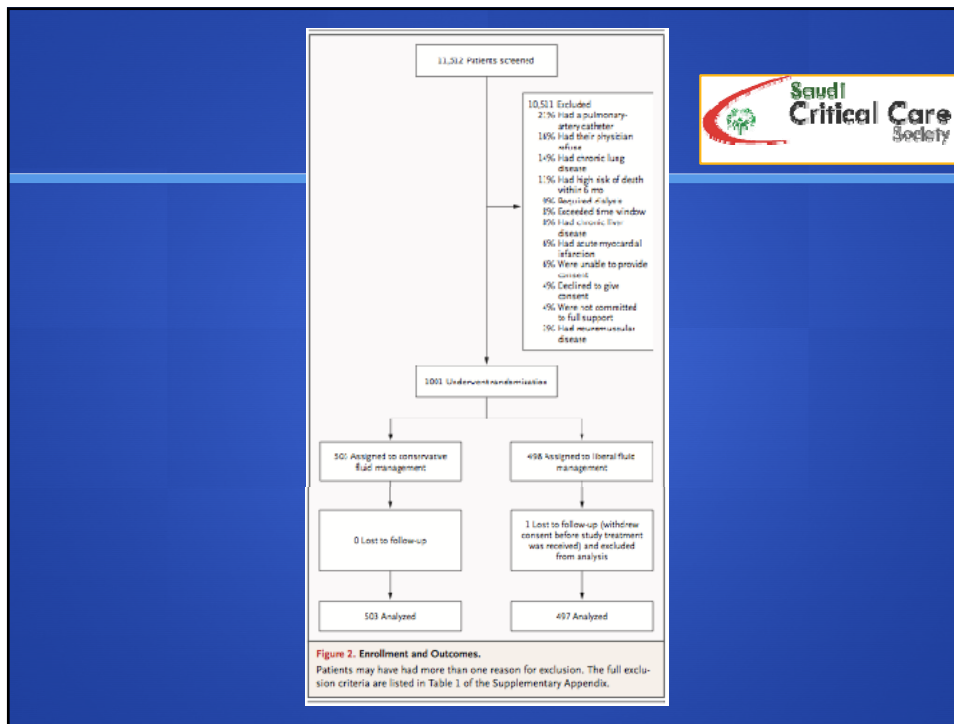
The members of the Writing Committee (Herbert P. Wiedemann, M.D., Cleveland Clinic; Cleveland, Arthur P. Wheeler, M.D., and Gordon R. Bernard, M.D., Vanderbilt University; Nashville; B. Taylor Thompson, M.D., and Douglas Hayden, M.A., Massachusetts General Hospital, Boston; Ben deBorja, M.D., Louisiana State University Health Sciences Center, New Orleans; Alfred F. Conners, Jr., M.D., Case Western Reserve University at MetroHealth Medical Center, Cleveland; R. Duncan Hite, M.D., Wake Forest University Health Sciences Center, Winston-Salem, N.C.; and Andrei L. Harabin, Ph.D., National Institutes of Health, National Heart, Lung, and Blood Institute, Bethesda, Md.) assume responsibility for the integrity of the article. Address reprint requests to Dr. Wiedemann at the Department of Pulmonary, Allergy, and Critical Care Medicine, Cleveland Clinic, 9500 Euclid Ave., Desk A-90, Cleveland, OH 44195, or at wiedemh@ccf.org.

*Participants in the ARDS Clinical Trials Network are listed in the Appendix.

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Study Protocol

| Measured intravascular pressure (mm Hg) | | | | MAP <60 mm Hg or a need for any vasopressor (except dopamine <5 µg/kg/min); consider conservative measures if shock first | MAP ≥60 mm Hg without vasopressors (except dopamine ≤5 µg/kg/min) | | | |
|---|------------------|-----------------------|------------------|--|--|--|--|--|
| CVP | | PcOP ^o | | | Average urinary output <0.5 ml/kg/hr | | Average urinary output ≥0.5 ml/kg/hr | |
| Conservative strategy | Liberal strategy | Conservative strategy | Liberal strategy | Ineffective Circulation Cardiac index <2.5 liters/min/m ² or cold, mottled skin with capillary-refilling time >2 sec | Effective Circulation Cardiac index ≥2.5 liters/min/m ² or absence of criteria for ineffective circulation | Ineffective Circulation Cardiac index <2.5 liters/min/m ² or cold, mottled skin with capillary-refilling time >2 sec | Effective Circulation Cardiac index ≥2.5 liters/min/m ² or absence of criteria for ineffective circulation | |
| Range 1 | | | | 1 Vasopressor ^f Fluid bolus ^g | 3 KVO IV Dobutamine ^h Furosemide ^{h,12,4} | 7 KVO IV Furosemide ^{h,12,4} | 11 KVO IV Dobutamine ^h Furosemide ^{h,13,4} | 15 KVO IV Furosemide ^{h,13,4} |
| >13 | >18 | >18 | >24 | | 4 KVO IV Dobutamine ^h | 8 KVO IV Furosemide ^{h,13,4} | 12 KVO IV Dobutamine ^h | 16 KVO IV Furosemide ^{h,13,4} |
| Range 2 | | | | 2 Fluid bolus ^g Vasopressor ^f | 5 Fluid bolus ^c | 9 Fluid bolus ^c | 13 Fluid bolus ^c | 17 Liberal KVO IV |
| 9-13 | 13-18 | 13-18 | 19-24 | | 6 Fluid bolus ^c | 10 Fluid bolus ^c | 14 Fluid bolus ^c | 18 Conservative Furosemide ^{h,13,4} |
| 6-9 | 10-14 | 8-12 | 14-18 | | | | | 19 Liberal Fluid bolus |
| <6 | <10 | <8 | <14 | | | | | 20 Conservative KVO IV |



Outcome

Table 3. Main Outcome Variables.*

| Outcome | Conservative Strategy | Liberal Strategy | P Value |
|--|-----------------------|------------------|---------|
| Death at 60 days (%) | 25.5 | 28.4 | 0.30 |
| Ventilator-free days from day 1 to day 28† | 14.6±0.5 | 12.1±0.5 | <0.001 |
| ICU-free days‡ | | | |
| Days 1 to 7 | 0.9±0.1 | 0.6±0.1 | <0.001 |
| Days 1 to 28 | 13.4±0.4 | 11.2±0.4 | <0.001 |
| Organ-failure-free days†† | | | |
| Days 1 to 7 | | | |
| Cardiovascular failure | 3.9±0.1 | 4.2±0.1 | 0.04 |
| CNS failure | 3.4±0.2 | 2.9±0.2 | 0.02 |
| Renal failure | 5.2±0.1 | 5.6±0.1 | 0.45 |
| Hepatic failure | 5.7±0.1 | 5.5±0.1 | 0.12 |
| Coagulation abnormalities | 5.6±0.1 | 5.4±0.1 | 0.23 |
| Days 1 to 28 | | | |
| Cardiovascular failure | 19.0±0.5 | 19.1±0.4 | 0.85 |
| CNS failure | 18.8±0.5 | 17.2±0.5 | 0.03 |
| Renal failure | 21.3±0.5 | 21.2±0.5 | 0.59 |
| Hepatic failure | 22.0±0.4 | 21.2±0.5 | 0.18 |
| Coagulation abnormalities | 22.0±0.4 | 21.5±0.4 | 0.37 |

CVP reading as a predictor of responsiveness

| Reference | Responder | Non-Responder | P value |
|---|-----------|---------------|----------------|
| Pra | | | |
| Calvin JE: Surgery 1981, 90:61–76. | 5 ± 1 | 5 ± 2 | NS |
| Pinsky: Chest 1990, 98:1450–1454. | 9 ± 4 | 8 ± 4 | NS |
| Teboul JL: Chest 1997, 111:351–358. | 7 ± 2 | 6 ± 3 | NS |
| Michard F: Am J Respir Crit Care Med 2000, 162:134–138. | 9 ± 3 | 9 ± 4 | NS |
| Bafaqeeh F, Magder S: J Critical Care 2007 | 6 | 9 | 0.002 |
| Pwp | | | |
| Calvin JE: Surgery 1981, 90:61–76. | 8 ± 1 | 7 ± 2 | NS |
| Pinsky: Chest 1990, 98:1450–1454. | 10 ± 4 | 10 ± 3 | NS |
| Diebel L: J Trauma 1994, 37:950–955. | 16 ± 6 | 15 ± 5 | NS |
| Tavernier B: Anesthesiology 1998, 89:1313–1321. | 10 ± 4 | 12 ± 3 | NS |
| Toussignant CP: Anesth Analg 2000, 90:351–355. | 12 ± 2 | 16 ± 3 | less than 0.01 |
| Michard F: Am J Respir Crit Care Med 2000, 162:134–138. | 10 ± 3 | 11 ± 2 | NS |
| Bafaqeeh F, Magder S: J Critical Care 2007 | 10 | 12 | 0.01 |



critical care review

Predicting Fluid Responsiveness in ICU Patients*

A Critical Analysis of the Evidence

Frédéric Michard, MD, PhD; and Jean-Louis Teboul, MD, PhD



Table 5—Positive and Negative Predictive Values of Dynamic Parameters

| Source | Patients, No. | Parameters Tested | Best Threshold Value | Positive Predictive Value, % | Negative Predictive Value, % |
|------------------------------------|---------------|-------------------|----------------------|------------------------------|------------------------------|
| Magder et al ¹ | 33 | ΔBAP | 1 mm Hg | 84 | 83 |
| Tavernier et al ² | 35 | Δdown | 5 mm Hg | 93 | 83 |
| Magder and Laguardie ¹⁰ | 29 | ΔBAP | 1 mm Hg | 77 | 81 |
| Michard et al ¹¹ | 40 | ΔPP | 13% | 94 | 96 |
| Feissel et al ¹³ | 19 | ΔVpeak | 12% | 91 | 100 |

Special Article



Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

R. Phillip Dellinger, MD; Mitchell M. Levy, MD; Jean M. Carlet, MD; Julian Bion, MD; Margaret M. Parker, MD; Román Jaeschke, MD; Konrad Reinhart, MD; Derek C. Angus, MD, MPH; Christian Brun-Buisson, MD; Richard Beale, MD; Thierry Calandra, MD, PhD; Jean-François Dhahaut, MD; Herwig Goebel, MD; Maureen Harvey, RN; John J. Marini, MD; John Marshall, MD; Marco Ranieri, MD; Graham Ramsey, MD; Jonathan Sevransky, MD; B. Taylor Thompson, MD; Sean Townsend, MD; Jeffrey S. Vender, MD; Janice L. Zimmerman, MD; Jean-Louis Vincent, MD, PhD; for the International Surviving Sepsis Campaign Guidelines Committee

Objective: To provide an update to the original Surviving Sepsis Campaign clinical management guidelines, "Surviving Sepsis Campaign Guidelines for Management of Severe Sepsis and Septic Shock," published in 2004.

Design: Modified Delphi method with a consensus conference of 55 international experts, several subsequent meetings of subgroups and key individuals, teleconferences, and electronic-based discussion among subgroups and among the entire committee. This process was conducted independently of any industry funding.

Methods: We used the Grades of Recommendation, Assessment, Development and Evaluation (GRADE) system to grade assessment of quality of evidence from high (A) to very low (D) and to determine the strength of recommendations. A strong recommendation (1) indicates that an intervention's desirable effects clearly outweigh its undesirable effects (risk, burden, cost) or clearly do not. Weak recommendations (2) indicate that the benefits/burden/risks and undesirable effects are less clear. The grade of strong or weak is considered of greater clinical importance than a difference in letter-level of quality of evidence. In areas without complete agreement, a formal process of resolution was developed and applied. Recommendations are grouped into those directly targeting severe sepsis, recommendations targeting general care of the critically ill patient that are considered high priority in severe sepsis, and pediatric considerations.

Results: Key recommendations, listed by category, include early goal-directed resuscitation of the septic patient during the first 6 hrs after recognition (1C); blood cultures before antibiotic therapy (1C); imaging studies performed promptly to confirm potential source of infection (1C); administration of broad-spectrum antibiotic therapy within 1 hr of diagnosis of septic shock (1B) and severe sepsis without septic shock (1D); reassessment of antibiotic therapy with microbiology and clinical data to narrow coverage, when appropriate (1C); a usual 7–10 days of antibiotic therapy guided by clinical response (1D); source control with attention to the balance of risks and benefits of the chosen method (1C); administration of either crystalloid or colloid fluid resuscitation (1B); fluid challenge to restore mean circulatory filling pressure (1C); reduction in rate of fluid administration with rising filling pressures and no improvement in tissue perfusion (1D); vasopressor preference for norepinephrine or dopamine to maintain an initial target of mean arterial pressure ≥ 65 mm Hg (1C); dobutamine inotropic therapy when cardiac output remains low despite fluid resuscitation and combined inotropic/vasopres-

sure is identified to be poorly responsive to fluid and vasopressor therapy (2C); recombinant activated protein C in patients with severe sepsis and clinical assessment of high risk for death (2D except 2D for postoperative patients). In the absence of tissue hypoperfusion, coronary artery disease, or acute hemorrhage, target a hemoglobin of 7–9 g/dL (1B); a low tidal volume (1B) and limitation of inspiratory plateau pressure stability (1C) for acute lung injury (ALI)/acute respiratory distress syndrome (ARDS); application of at least a minimal amount of positive end-expiratory pressure in acute lung injury (1C); head of bed elevation in mechanically ventilated patients unless contraindicated (1B); avoiding routine use of pulmonary artery catheters in ALI/ARDS (1A); to decrease days of mechanical ventilation and ICU length of stay, a conservative fluid strategy for patients with established ALI/ARDS who are not in shock (1C); protocols for sedation and analgesia (1B); using either intermittent bolus sedation or continuous infusion sedation with daily interruptions or lightening (1B); avoidance of neuromuscular blockers, if at all possible (1B); institution of glycemic control (1B), targeting a blood glucose < 168 mg/dL after initial stabilization (2C); equivalence of continuous versus intermittent heparin infusion or intermittent heparin bolus (2B); prophylaxis for deep vein thrombosis (1A); use of stress ulcer prophylaxis to prevent upper gastrointestinal bleeding using H2 blockers (1A) or proton pump inhibitors (1B); and consideration of limitation of support where appropriate (1D). Recommendations specific to pediatric severe sepsis include greater use of physical examination therapeutic end points (2C); dopamine as the first drug of choice for hypotension (2C); steroids only in children with suspected or proven adrenal insufficiency (2C); and a recommendation against the use of recombinant activated protein C in children (1B).

Conclusions: There was strong agreement among a large cohort of international experts regarding many level 1 recommendations for the best current care of patients with severe sepsis. Evidence-based recommendations regarding the acute management of sepsis and septic shock are the first step toward improved outcomes for this important group of critically ill patients. (Crit Care Med 2008; 36:296–327)

Key Words: sepsis; severe sepsis; septic shock; sepsis syndrome; infection; Grades of Recommendation, Assessment, Development and Evaluation criteria; GRADE; guidelines; evidence-based medicine; Surviving Sepsis Campaign; sepsis

Special Article

Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

Table 3. Initial resuscitation and infection issues

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

- Indicates a strong recommendation, or "we recommend"
- Indicates a weak recommendation, or "we suggest"

Initial resuscitation (first 6 hrs)

- Begin resuscitation immediately in patients with hypotension or elevated serum lactate > 4 mmol/L; do not delay pending ICU admission (1C)
- Resuscitation goals (1C)
 - CVP 8–12 mm Hg
 - Mean arterial pressure ≥ 65 mm Hg
 - Urine output ≥ 0.5 mL·kg⁻¹·hr⁻¹
 - Central venous (superior vena cava) oxygen saturation $\geq 70\%$ or mixed venous $\geq 65\%$

Special Article

Surviving Sepsis Campaign: International guidelines for management of severe sepsis and septic shock: 2008

Table 4. Hemodynamic support and adjunctive therapy

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

● Indicates a strong recommendation, or "we recommend"

○ Indicates a weak recommendation, or "we suggest"

Fluid therapy

- Fluid resuscitate using crystalloids or colloids (1B)
- Target a CVP of ≥ 8 mm Hg (≥ 12 mm Hg 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 mins. 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)

