Rapid Assessment of the High Acuity Patient

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Objectives: Rapid Assessment of the High Acuity Patient

1. Outline an approach to assessing the severity and etiology of disease in the high acuity patient
2. Understanding the methods available to determine the response to therapy and to guide subsequent interventions
3. Review examples of recent studies that highlight the challenges of clinical assessment and the benefit of therapeutic interventions in the high acuity patient

Conflict of Interest:

• None

Outline: Rapid assessment of the high acuity patient

1. Physical assessment of the high acuity patient
2. Secondary assessment approaches
   – Oxygen transport balance
   – Respiratory muscle performance
   – Hemodynamic performance markers
   – Central nervous system dysfunction
3. Recent studies of therapies for the high acuity pediatric patient

Reasons to monitor with the goal to correct physiologic variables in the high acuity patient

1. It can work *
2. Traditionally, physiology has been the basis for assessment and treatment in critically ill patients
3. The degree of physiologic variability from normal values is a marker of severity of illness
4. The response of physiologic variables to therapeutic interventions is a marker of prognosis.
   * There are many exceptions to this rule.

Physical Assessment of the High Acuity Patient

• Looks good, looks bad (the Hazinski rule)
• Components of the 15 second assessment
   – Vital signs
   – Mental status
   – Work of breathing
   – Peripheral perfusion
   – Gross abnormalities
Secondary Assessment of the Physiologic Variables

- Oxygen transport balance
- Respiratory muscle performance
- Hemodynamic performance markers
- Central nervous system dysfunction

Oxygen Transport Balance

Oxygen Delivery (DO₂)

\[ \text{DO}_2 = \text{CO} \times \text{CaO}_2 \]

\[ \text{HR} \times \text{SV} \]

\[ \text{Hb} \times \text{SaO}_2 \times 1.34 \]

Preload  Contractility  Afterload

Oxygen Delivery (DO₂)

\[ \text{DO}_2 = \text{CO} \times \text{CaO}_2 \]

\[ \text{VO}_2 = \text{CO} \times (\text{CaO}_2 - \text{CvO}_2) \]

\[ = \text{CO} \times 1.34 \times \text{Hb} (\text{SaO}_2 - \text{SvO}_2) \]

\[ \text{O}_2\text{ER} = \frac{\text{VO}_2}{\text{DO}_2} = \frac{\text{SaO}_2 - \text{SvO}_2}{\text{SaO}_2} \]

- Normal O₂ ER = 25 – 30%
- Critical O₂ ER = 50-60%

Therapeutic Intervention

- Preload
- Vasoactive Agents
- PPV
- RBC
- Sedation
- Hypothermia
- Mechanical ventilation

Venous O₂ Saturation

Changes in blood pressure and arterial saturation are minimal as progressive circulatory compromise is detected by severely falling SvO2 (A and C). The device guided successful (B, E, G, H) and unsuccessful (D, F) interventions. Oscillations in SvO2 and arterial pressure (I) suggest significant changes in vascular resistance.

Early Goal Directed Therapy

The History of Near Infrared Spectroscopy

- NIRS to assess tissue oxygenation: 1977
- FDA approval of NIRS cerebral oximeter for clinical use in adults: 1996
- Approved for use in children: 2000
- Used in 80-90% of pediatric cardiac centers during CPB
- Increasingly being used in PICU & NICU


How the NIRS System can Help

The INOS System uses two depths of light penetration to subtract out surface data, resulting in a regional oxygenation value for deeper tissues.
OxyAlert™ NIRSensor

Pediatric, Adult  Infant/Neonate

NIRS Oximetry

NIRS Monitoring: rSO₂
- rSO₂ index represents the balance of site specific O₂ delivery and consumption
- rSO₂ measures both venous (75%) and arterial blood (25%)
- rSO₂ indicates adequacy of site-specific tissue perfusion in real time
- rSO₂ correlates positively with SvO₂

Accuracy of Cerebral Oximetry


Cerebral Oxygen Transport Balance
- Cerebral saturation correlates inversely with measurements of brain lactate concentration

Reversal of Shock
Underlying data and case notes on file ISC-10001.
Penetration of NIRS Light

The human skull is easily penetrated by near-infrared light. Longer wavelength infrared light penetrates better than visible light.

Cerebral Oxygen Transport Balance

Relationship between NIRS-derived cerebral O₂ saturations & brain tissue lactate concentration in piglets as cerebral DO₂ is manipulated.


NIRS Oximetry: Benefits

- Instant, noninvasive, continuous real time data
- Site-specific (regional) measurement
- Excellent indicator of regional oxygen changes associated with shock
- Early warning indicator
- Immediately reflects the impact of interventions
- May be deployed in virtually any setting

NIRS Oximetry: Limitations

- Cerebral O₂ saturation approximates SmvO₂
- Sensitive to acute changes in PaCO₂
- May be affected by skull thickness and Hb concentration

Respiratory Muscle Performance: Assessment in the high acuity patient

Case Presentation

A 4 y.o. female with acute lymphocytic leukemia is transferred to the Pediatric Intensive Care Unit 10 days after hematopoietic stem cell transplantation with fever, respiratory distress, and hypotension. She has been resuscitated with 60 cc/kg crystalloid and is administered vancomycin and cefipime after blood cultures are obtained.

At the time of transfer pertinent laboratories are as follows:
- Absolute neutrophil count O, Hemoglobin 8.9 gm/dl, Platelet count 10.k
- SaO₂ 89% (10 LPM O₂ face mask)
Case Presentation: Cont.

Chest – tachypnea with labored respiratory effort, decreased air entry bilaterally
Cardiac – Tachycardia, diminished peripheral pulses, 5 second capillary refill
Abdomen – Paradoxical movement of the abdomen on inspiration
Neurologic – agitated, no focal abnormalities

Venous pH 7.18 PvCO2 55mmHg ScO2 55%
Lactate 4.2 mmol/L
BNP 1726 ng/L
Chest X-ray- diffuse bilateral alveolar infiltrates

Case Presentation: Cont.

Physical exam at PICU admission:
Wt 20 kg, T 39° C, HR 130 bpm (sinus rhythm), RR 48 bpm, BP 85/60, SaO2 95% on 15 LPM non rebreathing mask

Another normal saline bolus of 20 cc/kg is administered and dopamine at 10 mcg/kg/min is started for blood pressure support.
Soon after admission to the PICU, you note that the patient is having episodes of 5 second apnea with a decrease in SaO2 to 75%.
A radial arterial line is placed: pH 7.10, PaCO2 70 mmHg, PaO2 50 mmHg, Lactate 5.6 mmol/L

Hypercapnic respiratory failure in this patient is most likely due to:

1. Intrapulmonary shunt
2. Increased physiologic dead space
3. Blunted central respiratory drive
4. Diaphragmatic muscle fatigue
5. Excessive CO2 production

The oxygen cost of breathing (VO2 resp) is determined by which of the following factors?

1. Minute ventilation
2. Lung compliance and airways resistance
3. Respiratory muscle blood flow
4. Oxygen extraction of the respiratory muscles
5. All of the above
When low cardiac output limits respiratory muscle blood flow in a patient with increased respiratory demand, respiratory muscle fatigue is a likely consequence.

Shock and Diaphragmatic Fatigue

Mechanical Ventilation: Friend and Foe to the Diaphragm

The clinical manifestations of diaphragmatic muscle fatigue include all of the following except:

1. Hypercapnia
2. Respiratory alternans
3. Wheezing
4. Paradoxical abdominal motion
5. Apnea

Diaphragm and Abdominal Motion
Hemodynamic Performance Markers in the high acuity patient

Physical Exam:
**ALWAYS necessary** but what else is out there?
1. Anion gap metabolic acidosis
2. Capillary or venous blood gas
3. Lactate
4. B-Type Natriuretic Peptide (BNP)
5. Bedside ECHO
6. Near Infrared Spectroscopy (NIRS)

Metabolic Acidosis
- Presence of metabolic acidosis or base deficit:
  1. Nonspecific
  2. Poor correlation w/ elevated lactate
- Calculate anion gap:
  - Difference b/t “unmeasured” cations & anions
  - \( \text{Na}^+ - (\text{Cl}^- + \text{HCO}_3^-) \)
  - Normal anion gap 10 ± 2 mEq/L

Anion gap acidosis
- Etiology:
  1. Decreased unmeasured cations (\( \text{K}^+ , \text{Ca}^{2+} , \text{Mg}^{2+} \))
  2. Increased unmeasured anions
    - Lactic acidosis (shock states)
    - Ketoacidosis (diabetic ketoacidosis)
    - Accumulation of renally excreted organic & inorganic anions (uremia)

Lactate - Clinical Use
- Measure severity of shock and risk of mortality
  - Easily determined with arterial venous sample
  - More specific than base deficit
- Triage illness severity (useful for ED patients)

Lactate - Studies in Children
- Most studies in cardiac surgery patients
  - ↑ Lactate correlated w/ postoperative death and MSOF
- General Peds ICU patients
  - ↑ Lactate = predictor of mortality and poor outcome
Myocardial dysfunction in sepsis: Troponin I

• Etiology of myocardial dysfunction in sepsis
  – Myocardial depressant factor
  • IL, TNFα, endotoxin, lipopolysaccharide
  – Cytotoxic process
  • Elevated troponin levels as marker of myocardial cell injury in septic shock
    – Troponin I ↑ in 57% of patients
    – ↑ Troponin assoc with decreased EF
    – Not associated w inotrope requirement

Brain (B Type) Natriuretic Peptide (BNP)

• Hormone secreted by ventricular myocardium in response to
  – Increase LV filling pressures
  – Myocardial wall stretch
  – Pressure load

BNP

• Actions of BNP include:
  – Diuresis
  – Natriuresis
  – Vascular smooth muscle dilation
  • Balanced arterial and venous
    – Antagonist renin-angiotensin-aldosterone system

Studied in many conditions
  – Elevated in:
    • Congestive heart failure
      – Adult “cut-off” are 100 pg/ml
    • Pulmonary hypertension
    • Children w/ various congenital heart defects
    – Used to differentiate cardiac vs. non-cardiac causes of dyspnea
    – Used to guide therapy for CHF
    – Marker in patients with septic shock
    – Fluid restriction in ARDS

Bedside ECHO

• Non-cardiologist MD’s and nurses can be trained to accurately visualize and make reliable assessments of certain trans-thoracic echocardiography (TTE) features

TTE features:
  – Left ventricular function (LVEF)
    • Agreement between ED MD & formal echo
      – 92% when normal LVEF
      – 70% when poor LVEF
  – Pericardial Effusion (presence or absence)
    – 95% agreement
  – CVP (IVC diameter)
    • Agreement between ED MD & formal echo
      – 67% when high CVP
      – 20% when low CVP
Negative Intrathoracic Pressure and Cardiac Output: Clinical Clues

1. High index of suspicion
2. Presence of a pulsus paradoxus
3. Reduced amplitude of pulse oximetry or arterial wave form
4. Alteration (downward trend) in Near Infrared Spectroscopy (NIRS)

Pulsus Paradoxus

- Result of marked inspiratory ↓ in left-sided cardiac output, caused by ↓ left atrial return from ↑ capacitance of pulmonary vascular bed and ↑ left ventricular afterload from negative intrapleural pressures

End-tidal C02 Monitoring

- How to interpret the value:
  A. ↑ ETCO₂ and ↑ PaCO₂
  ↓ VE
  B. ↓ ETCO₂ and ↓ PaCO₂
  ↑ VE
  C. ↓ PETCO₂ and ↑ PaCO₂
  ↑ VD/VT = \( \frac{\text{PaCO}_2 - \text{PETCO}_2}{\text{PaCO}_2} \)

Physiologic dead space

- \( \frac{V}{Q} = \infty \)
- Lung ventilated but not perfused
- Consider causes of decreased pulmonary blood flow

Which of the following clinical conditions is associated with decreased pulmonary blood flow?

A. Pulmonary hypertension
B. Asystole
C. Pulmonary Embolus
D. Excessive PEEP
E. All of the above
Capnogram in Patient Undergoing Chest Compressions

Fig. 9. Capnogram from a patient undergoing chest compressions. Note that the capnogram curve changes at the point at which a "fresh" (less tired) clinician (assistant) took over the chest compressions. (Adapted from Reference 42, with permission.)

Capnogram and Endotracheal Intubation

Fig. 5. Adult capnograms of tracheal intubation (upper) and esophageal capnogram (below). (Adapted from Reference 31, with permission.)

Central Nervous System Dysfunction: Assessment in the high acuity patient

- Assessment in the high acuity patient
- Biomarkers of brain injury
- Portable CT scanner
- Bedside video EEG with telemetry
- Near infrared spectroscopy
- Telemedicine

Recent Physiologic Studies

1. Goal directed therapy for septic shock
2. Tight glycemic control
3. Oxygenation in response to inhaled nitric oxide or patient proning
4. Red blood cell transfusions in PICU patients

Summary:

It remains fundamentally true that rapid assessment and directed therapy in the high acuity patient increases the likelihood of a favorable outcome. Physical and biologic variables is the responsibility of the critical care practitioner. Continuing education is required to be in a position to employ the variety of tools that are increasingly available.

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