Recognizing the Critically Ill Patient

Andrew Wormsbecker
PGY5 Critical Care Medicine
Disclosures

- none
Objectives

- Use a systematic assessment approach to identify underlying physiologic problem(s) and their causes to direct initial treatment of seriously ill patients
- Elicit patient features on history that indicate increased risk for deterioration
- Perform initial examination and interpret vitals and other findings as they relate to identification of organ dysfunction or limited reserves
- Choose and interpret high yield initial investigations
- Plan follow up assessment (what will be done, who will do it, and when) based on your expected response to treatment
- Distinguish acute from chronic disease presentation
- Identify various healthcare providers you can call upon to help in the care of your patient.
Additional Objectives

- Approach to shock
- Lactic Acidosis
Intro

- Many patients demonstrate concerning historical symptoms or physiologic signs hours before cardiopulmonary arrest
- Young, fit patients have large compensatory reserves
- Immunosuppressed patients may not mount robust responses which may mask signs of illness
“How sick is this patient?”

- “Spidy sense” and clinical experience will help guide you.
- You have a senior, and a staff with more of this experience.
- An organized approach will help you build this experience and pattern recognition.
- The CA system has taken away a lot of the “on-call emergencies” at VGH and SPH.
  - Try and tag along if you aren’t slammed.
Case

- 54M, HIV+ not on treatment presents with SOB, mild pleuritic chest pain and a new RLL infiltrate
- Sr “triages” patient and then gets called away so sends you to see as the IM Jr because patient looks pretty sick
• All: none
• Meds: ventolin, flovent, aspirin
• PHx: 1 intubation for asthma in past, HIV for 5 years from past IVDU, no treatment, CD4 50
• 2 days of chills, “fever”, SOB, productive cough of green sputum, worse today, hard to catch breath, no immunizations
OE:

- 120/70, 125, 26, 89% on HFFM, 38.5
- Dissheveled, looks “sick”
- Mildly drowsy, easily roused, GCS 13 (E3, V4, M6)
- CVS: JVP @ sternal angle, flow murmur, normal S1S2
- Resp: increased acc muscles, tracheal tugging, increased tactile fremitus RLL, expiratory wheeze throughout, RLL coarse crackles and bronchial breath sounds
- Abdo: unremarkable
- Skin: mottling of knees
- Fingers: mild acrocyanosis
• Inv:
  • WBC 13 with N 12, Hgb 125, Plt 120
  • Electrolytes normal
  • Cr 125 (baseline 50)
  • HCO3 16
  • Lactate 4
  • ABG: 7.27/31/70/17/-6 on ~40% FiO2
  • CXR: RLL opacity
  • ECG: sinus tachycardia
Can you write-down what indicates critical illness
Approach to the Sick Patient

- “ABC, MOVIE”
  - Airway, Breathing, Circulation
  - Monitor, Oxygen, Vital Signs, IV, Exposure/draping/lighting

- Should be first words out of your mouth on any OSCE acute medicine physical exam or oral scenario

- Akin to “Primary survey” from trauma
- THIS COMES BEFORE YOUR HISTORY DESPITE WHAT THE BOOK SAYS
What does this actually mean?

- **Airway**
  - is the patient protecting their away
  - Hint: if they can talk to you, they are protecting and you can move on
  - If unsure, ask a question
  - If not: Ask for help, assess for airway obstruction (foreign body, signs of stridor), noisy breathing, grunting, cyanosis
  - level of consciousness (GCS – “less than 8=intubate”)
## Glasgow coma scale

<table>
<thead>
<tr>
<th>Eye opening</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>Response to verbal command</td>
<td>3</td>
</tr>
<tr>
<td>Response to pain</td>
<td>2</td>
</tr>
<tr>
<td>No eye opening</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Best verbal response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Oriented</td>
<td>5</td>
</tr>
<tr>
<td>Confused</td>
<td>4</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>3</td>
</tr>
<tr>
<td>Incomprehensible sounds</td>
<td>2</td>
</tr>
<tr>
<td>No verbal response</td>
<td>1</td>
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<table>
<thead>
<tr>
<th>Best motor response</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Obeys commands</td>
<td>6</td>
</tr>
<tr>
<td>Localizing response to pain</td>
<td>5</td>
</tr>
<tr>
<td>Withdrawal response to pain</td>
<td>4</td>
</tr>
<tr>
<td>Flexion to pain</td>
<td>3</td>
</tr>
<tr>
<td>Extension to pain</td>
<td>2</td>
</tr>
<tr>
<td>No motor response</td>
<td>1</td>
</tr>
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</table>

**Total**

The GCS is scored between 3 and 15, 3 being the worst, and 15 the best. It is composed of three parameters: best eye response (E), best verbal response (V), and best motor response (M). The components of the GCS should be recorded individually; for example, E2V3M4 results in a GCS score of 9. A score of 13 or higher correlates with mild brain injury; a score of 9 to 12 correlates with moderate injury; and a score of 8 or less represents severe brain injury.
• Breathing (not your complete resp exam!)
  • -look, listen, feel
  • If they are talking, probably not a huge problem
  • Look at symmetry, pattern, rate, accessory muscle use
  • Many potential locations of disorder
    • CNS- decreased drive
    • Cardiac- ischemia, valvulopathy, rhythm, pump failure
    • Bellows- neuromuscular, chest wall
    • Airway
    • Parenchyma
    • Vascular
    • Pleura
    • Systemic conditions – anemia, sepsis, liver failure, drugs
Breathing Patterns

- **Bradypnea**: sedative-hypnotics (Benzos, EtOH), narcotics, encephalopathy
- **Tachypnea**: acidosis, sepsis, sympathomimetics, ASA
- **Biot**: ataxic respirations
  - Severe brain injury/medullary injury
- **Cheyne-Stokes**: apneas followed by hyperpneas that then decrease to apnea
  - Altered “loop-gain” response to CO2
  - Think CHF, bihemispheric or brainstem injury
- **Kussmaul**: deep, labored, gasping breaths that typically follow initial tachypnea
  - Compensatory hyperventilation for severe acidosis
  - *think DKA, lactic acidosis*
Circulation:

- HR, BP, orthostatics
- *skin warmth, mottling
- Assess pulses for rate, volume, regularity, symmetry
- Can actually assess pulsus by palpation (but should do BP if concerned)

- May relate to primary cardiovascular problem or secondary to metabolic issues, sepsis, hypoxemia, drugs
Historical Features

- Classically >90% diagnosis made on history
- In critically ill, patient may not give history!
  - Collateral: nurses, care aides, family, friends, EHS, other consult notes, chart, etc
Historical Features

- Rapid History:
  - Signs/symptoms
  - Allergies
  - Medications
  - Past Hx
  - Last meal
  - Events surrounding

- OPQRST of issue
• Symptoms will usually direct toward organ-specific history and approach – ie chest pain, dyspnea, altered mental status
  •  -separate talks

• Attempt to distinguish acute from chronic dysfunction, assess for reversible causes
  • Eg: COPD patient with high CO2 – look at the compensation to see if acute or chronic
    • Look at previous notes, dictations, dig for collateral
    • Focus on what has changed from their baseline
People to worry about:

- Emergency admission - limited info
- Advanced age – comorbidities, limited reserve
- Severe coexisting illnesses – juggling problems, limited reserve
- Severe physiologic abnormalities – limited reserve, refractory to therapy
- Need/recent major surgery
- Severe bleed, need for massive transfusion
- Deterioration on repeat assessment/fail to respond to treatment
- Immunodeficiency
- Combinations of above
- SIRS response
Other people I worry about

- Asthmatics/COPDer's with past ICU, intubation
- Submassive PE's
- Anyone who’s pregnant, especially with respiratory illness
- Young septic patients
- Polysubstance overdose, long-acting drugs (like methadone!)
- Severe valvular abnormalities or pulmonary hypertension
- Elevated lactate with no great explanation
- DKA
- Severe hyponatremia
- The pneumonia patient on BiPap
- Unexplained metabolic acidosis
Examination

- After your screening ABC’s and vitals move on to “complete focused physical exam” focusing on the suspected organ system involved
  - “secondary survey”
  - Neuro: CN, screening motor and sensory, reflexes
  - Cardiac: IPPA, peripheral pulses
  - Resp: IPPA
  - Abdo: IPPA, always lay hand on abdomen
  - Don’t neglect the skin/integument
    - Dry, wet, think, thick, edematous, bruised, cyanosis, rashes
    - Nails: clubbing, splinters
    - Eyes: icterus, pale conjuctiva
Investigations

- CBC with dif, lytes, urea, Cr
  - WBC with left shift may be sign of infection
  - Metabolic acidosis, AG may be early sign of critical illness
    - Pay attention to unexplained low HCO3
- ABG
- Trop, CK, LACTATE
- CXR
- ECG
Put it all together

- Recognize patterns from history, physical, investigations
SIRS

- Systemic inflammatory response syndrome
- May be a clue of sepsis
- At least 2 of:
  - HR > 90
  - Temp < 36 or > 38
  - RR > 20 or PaCO2 < 32
  - WBC < 4, > 12, or > 10% bands
Management

- Depends on etiology
- Ensure large bore IV access (>18G x 2)
- Early aggressive fluids in septic and hypovolemic patients
- Early antibiotics in patients with SIRS/sepsis
  - 8%/hr decreased mortality after onset of low BP
- Supplemental O2
- Call for help!
Reassess

- Critically ill patient will require much more time
  - Not a consult you can bang out then see with staff in the morning
  - Frequent reassessment
  - After each intervention assess the response
  - If not respond ASK FOR HELP
Who can I call?!

1) your Senior

2) your staff – yes you can call them! It doesn’t matter if they are sleeping

3) Resp Therapist- can assist with high flow O2, BiPap, ABG’s

4) VGH: CCOT – can actually be nurse triggered as well
   *THIS IS WHAT CCOT IS FOR, THERE IS AN INTENSIVIST ON THE TEAM DURING THE DAY
4) SPH and RCH: no CCOT at present

5) HAU/ICU Consult
   - **if your patient is sick enough that they need critical care
     your staff needs to know about it
   - You are operating under their name, they are ultimately
     responsible and are supposed to supervise you
# Who needs ICU?

<table>
<thead>
<tr>
<th>Clinical indicator</th>
<th>Feature of the clinical indicator</th>
<th>Rationale for inclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potentially threatened airway</td>
<td>Evidence of stridor or noisy breathing. Swelling and bleeding of the airway</td>
<td>Such patients cannot be adequately monitored on the ward, and an urgent artificial airway may be required</td>
</tr>
<tr>
<td>Sustained tachypnoea</td>
<td>Respiratory rate &gt;26 breaths/min and/or increased work of breathing</td>
<td>Such patients may require non-invasive or invasive mechanical ventilation or frequent physiotherapy</td>
</tr>
<tr>
<td>Cyanosis despite FiO₂ &gt;0.4</td>
<td>Central cyanosis and/or SpO₂ &lt;90 % on pulse oximetry</td>
<td>This implies a PaO₂ &lt;60 mmHg (equating to a P:F ratio of approximately 150)</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>Heart rate &gt;120 bpm</td>
<td>This may suggest ensuing sepsis, hypovolaemia or bleeding</td>
</tr>
<tr>
<td>Systolic blood pressure</td>
<td>Systolic blood pressure measured as less than 100 mmHg</td>
<td>This suggests that shock may be developing, and that the patient may need insertion of invasive monitoring and commencement of inotropes</td>
</tr>
<tr>
<td>Altered skin colour</td>
<td>Cool periphery, mottled skin, prolonged capillary refill</td>
<td>This suggests a low CO state or microvascular insufficiency which may be due to cardiac failure, hypovolaemia or cardiodepression due to sepsis</td>
</tr>
<tr>
<td>Altered conscious state</td>
<td>Reduced GCS, delirium (particularly in a young person), focal neurology, new onset of a focal neurological deficit</td>
<td>The patient may not be able to protect their own airway or have intracranial pathology that requires post-procedural support. A delirious patient may have a serious underlying disease or require closer nursing observations</td>
</tr>
<tr>
<td>Frequent seizures</td>
<td>Seizures which are protracted (&gt;5 min) or recurrent (e.g. more than 2 in a 12-h period)</td>
<td>Patient may require endotracheal intubation and infusions of benzodiazepines, propofol or barbiturates</td>
</tr>
<tr>
<td>Increasing creatinine</td>
<td>Cr level which is rising, even if in the normal range</td>
<td>A rising creatinine can suggest developing renal failure and is an important marker of shock</td>
</tr>
<tr>
<td>Rising lactate level</td>
<td>Serum lactate &gt;3 mmol/L</td>
<td></td>
</tr>
</tbody>
</table>

Jones et al. Intensive Care Med. 2015
Shock

OMG!
Definition

- “state of cellular and tissue hypoxia due to reduced oxygen delivery and/or increased oxygen consumption or inadequate oxygen utilization”
- DOES NOT necessarily = hypotension
- “cryptic shock” or compensated shock
  - Compensatory tachycardia, vasoconstriction
- “normal BP” may be hypotension for that individual who has shifted their auto-regulation to the right

Shoemaker. New Horiz. 1996
Gaieski. Uptodate. 2015.
ICU Equation

- $DO_2 = CO \times O_2$ carry capacity
- $DO_2 = CO \times (Hgb \times 1.34 \times SaO_2 + 0.0031PaO_2)$
  - $CO = HR \times SV$
    - SV depends on PL, AL, contractility
- $DO_2 \sim VO_2$
  - When $DO_2$ drops below critical value (anaerobic threshold) shift from TCA cycle to anaerobic metabolism -> lactate production

Marino. The ICU Book. 2014
ATP Theoretical Yield

- Glucose → 2 ATP → Glycolysis → 2 NADH → 4 ATP
- Pyruvate → 2 NADH → 6 ATP
- Acetyl-CoA → 6 NADH → 18 ATP
- 2 FADH₂ → 4 ATP

Total net ATP yield = 36 ATP
Types of Shock

- 3 main types:
  - Distributive/Vasodilatory
  - Hypovolemic
  - Cardiogenic/Obstructive

- Can also think of as “warm” – vasodilatory problem, and “cold” – cardiac output problem
- In reality, often mixed
<table>
<thead>
<tr>
<th>Shock Type</th>
<th>Preload (CVP/JVP)*</th>
<th>SVR (warm/cold)</th>
<th>CO</th>
<th>PCWP/CXR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Distributive</td>
<td>low</td>
<td>Low/warm</td>
<td>high</td>
<td>Low/clear</td>
</tr>
<tr>
<td>Hypovolemic</td>
<td>low</td>
<td>High/cold</td>
<td>Low/normal</td>
<td>Low/clear</td>
</tr>
<tr>
<td>Cardiogenic</td>
<td>high</td>
<td>High/cold</td>
<td>low</td>
<td>High/wet</td>
</tr>
<tr>
<td>Obstructive</td>
<td>high</td>
<td>High/cold</td>
<td>Low</td>
<td>*PE-low/clear</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*TPTX-low/clear</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>*tamponade-high/wet</td>
</tr>
</tbody>
</table>

* TPTX: Tamponade Perforation Thromboembolism
Hypotension

- It’s all math!- sort of

- BP = CO X SVR
  - CO= HR X SV
    - SV determined by PL, AL, Contractility
4 questions

- 1) Warm or Cold? – feel leg
- 2) Empty or full? – JVP/CVP/Ultrasound IVC
- 3) Lungs wet or dry?
- 4) From above answers which subtype -> differential for that subtype
Distributive/Vasodilatory

- SEPSIS – another talk
- Adrenal Insufficiency, thyroid storm
- Neurogenic (esp T1-T5 injuries)
- Reperfusion injury/post-resuscitation/post cardiac arrest
- Anaphylaxis
- Fat, air, amniotic fluid embolus
- Liver failure
- SIRS syndromes- pancreatitis, idiopathic capillary leak
- Burns, trauma, crush injury
- Drugs – cyanide, long acting opiates, serotonin syndrome, TCA’s
Hypovolemic

- Hemorrhage
- GI losses - Severe diarrhea, vomiting
- Skin – toxic shock, TENS, burns, etc
- Renal – hypoaldo, salt wasting, osmotic diuresis (DKA, HHS, etc)
- Third spacing- burns, pancreatitis, bowel obstruction, post-op
Cardiogenic

- Heart is simple! It’s a pump!
  - Wiring – rhythm problem (brady/tachy)
  - Plumbing – CAD – MI, vasospasm, cocaine
  - Mechanical – valve failure – MR, MS, AI, AS, HOCM, septal rupture
  - Pump- CHF, cardiomyopathy, constriction, diastolic dysfunction, HTN, myocarditis
Obstructive

- Low cardiac output state from obstruction to filling right or left heart
  - Tension Pneumothorax
  - Pulmonary Embolism
  - Cardiac Tamponade
  - Constrictive Pericarditis
  - Severe Pulm HTN
  - Right Heart Failure
  - Abdo Compartment Syndrome
  - IVC tumor
Unusual Causes when things don’t make sense

- I keep this list on my phone
  - Extreme hyper/hypo thyroid
  - CO
  - Cyanide poisoning (usually from fires, not poisonings)
  - HLH
  - Mastocytosis
  - Scromboid
  - BeriBeri
  - Sheehan’s/Pit failure
  - Pheo
  - Tetanus
  - Serotonin Sx
  - GBS
  - Toxic shock syndrome- staph or strep
  - Drugs
  - Profound electrolyte abnormalities- hypoCa, hypoPO4
  - Severe acidosis – catecholamine unresponsiveness
  - Anatomical defect
Shock with relative bradycardia

- Cardiac ischemia (RCA/LCx lesions)
- Salmonella sepsis, viral infections, CNS infections
- Drugs: 2B’s, 3C’s and a D
  - BB, Barbiturates
  - CCB, Clonidine, cholinergics
  - Dig
Markers of shock

- Vitals: hypotension (and look at pulse pressure, DBP), tachycardia (rarely bradycardia), tachypnea (to compensate for acidosis), fever (infectious etiology)

- Exam: altered LOC, JVP/CVP, heart failure, skin mottling, urine output

- Labs: Lactate, elevated coags, low platelets, elevated bili, elevated ALT/AST, elevated Cr
Mottling
• Classic marker of microcirculatory dysfunction

• One study of 60 septic shock patients showed that the size of knee mottling, urine output and lactate were associated with mortality whereas traditional resuscitation guides (CVP, MAP, CO) were not

Ait-Oufella et al. Intensive Care Med. 2011
Lactic Acidosis

- 3 types

- Type A: hypoperfusion causing anaerobic metabolism

- Type B: non-hypoperfusion, redox state alterations, decreased clearance, etc

- Type D: D-lactate (not measured by our labs) – may see unexplained metabolic acidosis in patient with previous short gut/jejenoileal bypass/small bowel resection
Lactate Production

- Byproduct of glycolysis
- Pyruvate + NADH → Lactate + NAD+
- In states of anaerobic metabolism pyruvate cannot enter TCA cycle and thus produces lactate
- Altered “Redox” state drives pyruvate to lactate
  - Eg: chronic EtOH – alcohol dehydrogenase uses NAD+ as cofactor resulting in NAD+ deficient state
Type A Lactic Acidosis

- Hypoperfusion
- O2 demands exceed delivery
- Microcirculatory > macrocirculatory dysfunction
- Find cause: hypovolemia, sepsis, CHF, post arrest, etc
Type B

- Altered Redox State
  - Chronic alcohol/AKA
  - DKA
  - Epinephrine/catecholamines
  - Hypothermic shivering
  - Strenuous exercise
  - Post seizure
  - Mitochondrial dysfunction (drugs, sepsis, toxins, genetic like MELAS)
- Impaired pyruvate usage
  - Reye’s syndrome
  - Diabetes
  - congenital

- Increased pyruvate production
  - Glycogen storage diseases
  - Resp Alkalosis
  - Beta Agonists
• Malignancy – esp hematologic or large tumor burden
  • Increased metabolism, rapid growth

• Decreased clearance
  • LIVER FAILURE – 70% lactate clearance
  • Renal failure - <20% of lactate clearance
Drugs
- Metformin
- Stavudine and zidovudine
- Propofol
- Ethylene Glycol
- Cyanide, toluene, arsenic
- linezolid

McCullough. UpToDate 2015
De Backer. Intensive Care Med 2003
Lactate Clearance

- Lactate clearance >10% at 2 hours is a marker of organ perfusion and improving circulation in sepsis

- Failure to clear lactate associated with increased mortality

- As good as mixed venous in a 300 person study
  - *3 recent trials do not support mixed venous monitoring
  - You will hear more about this in your sepsis talk

Jones. JAMA 2010
Back to our case
What are concerning?

- All: none
- Meds: ventolin, flovent, aspirin
- PHx: 1 intubation for asthma in past, HIV for 5 years from past IVDU, no treatment, CD4 50
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Case Resolution

- You start patient on CAP antibiotics, PJP coverage
- BP decreases, you give 2L crystalloid with improved BP, lactate clearance
- Oxygenation gets worse, starts desatting
- ICU called, come intubate patient, place on pressors and take to unit