A to Z of Critical Care

Marianne Michaels ARNP, ACNP-BC, MSN, CCRN
ACIDOSIS

* Normal arterial pH 7.35 - 7.45; pH < 7.35 = acidosis (uncompensated; respiratory or metabolic)

* Serum CO2 (HCO3) low = metabolic acidosis (normal 22-30mEq/L)

* Based on Stewart approach, 3 determinants of pH:
  * PCO2 (respiratory) - normal 35-45mmHg; > 45 = acidosis
  * SID (Strong Ion Difference) (metabolic)
  * Weak acids (metabolic) - albumin, phosphate

* H+ and HCO3- are dependent variables without direct effect on pH
Strong ion difference (SID)

* SID (Strong Ion Difference) - difference between strong cations & anions
  
  \[ \text{SID} = (\text{Na} + \text{K} + \text{Ca} + \text{Mg}) - (\text{Cl} + \text{(other/unmeasured anions)}) \]

* Abbreviated formula Na - Cl; normal = 38 mEq/L

* SID < 38 = abnormal - “hyperchloremic metabolic acidosis” (Na deficiency or Cl excess, RTA, diarrhea)

* SID not low: lactate; ketones, renal, toxin

* SIG (Strong Ion Gap) = (Base deficit) + (SID - 38) + 2.5(4.2 - Albumin) - lactate; SIG > 2 = renal, keto, toxin, D-lactate; SIG < 2 = Hyper Ca, Mg, K
Stepwise approach

- ABG: pH low
- ABG: PCO2
- Lactate
- SID low: hyperchloremic (Na low or Cl high), RTA, diarrhea
- SID not low: renal, ketoacidosis (ETOH, DKA, starvation)
- Toxin - Osmolar gap
METABOLIC ACIDOSIS
Anion Gap

* Anion Gap (AG) is a calculated variable used to evaluate metabolic acidosis; Na - (Cl + CO2); normal 5-15mmol/L; > 30 reliable; 20-29 not as reliable & 1/3 will not have metabolic acidosis

* Normal AG metabolic acidosis (NAGMA), AKA reduced SID, causes: chloride excess - particularly IV NS, GI losses, RTA

* High AG metabolic acidosis (HAGMA), AKA normal SID, causes: lactate, renal, ketones (DKA, alcohol, starvation); toxin (ethylene glycol, propylene glycol, methanol, salicylate); if ? Toxin calculate osmolar gap
Examples

* ABG normal: pH 7.40 - PCO2 40 - PO2 85
* VBG normal: pH 7.37 - PCO2 48 - PO2 40
* ABG: pH 7.32 - PCO2 28
* CMP: Na 149 - Cl 119 - CO2 18; ABG pH 7.49 - PCO2 25 (on MVS)
* CMP: Na 132 - Cl 99 - CO2 13 - AG 31
ACIDOSIS
Manipulating the variables

* Respiratory - increase MV (minute ventilation) with NIV or MVS, or increase respiratory drive (i.e. Narcan)
* Lactic: identify & treat the cause
* Hyperchloremic - common in ICU; adjust IVF; administer Na (NaHCO3) if RTA or diarrhea
* Renal – depends on cause
* DKA - IVF + insulin; NaHCO3 rarely indicated
* Toxin - variable - antidote, HD
Bicarbonate

- ABG: HCO3 is a calculated variable based on pH + PCO2
- Serum CO2 (on BMP or CMP) is a measured variable; primarily a measurement of HCO3 (95% is HCO3; other 5% dissolved CO2 & carbonic acid)
Sodium bicarbonate
NaHCO₃

• 8.4% NaHCO₃ = equal parts Na & HCO₃; very hypertonic (osmolality 2000 mOsm/kg - about 7x serum osmolality)

* **Chloride-free Na bolus** - in the body it breaks down into: sodium carbonate, water, carbon dioxide

* **Side effects:**
  * Hypernatremia
  * Hyperosmolality
  * Hypokalemia
  * Hypercapnia
  * Oxyhemoglobin curve left shift: decrease O₂ delivery to tissues
  * Tissue necrosis if extravasation
Sodium bicarbonate
When do we give it?

* Generally, **treat the underlying cause of acidosis first**

* Severe hyperkalemia, salicylate toxicity, RTA, severe diarrhea; can also consider lieu of hypertonic NS i.e. severe hyponatremia w/ seizure, cerebral edema

* Severe acidosis - arterial pH < 7.1; risk of hemodynamic collapse d/t peripheral arterial dilatation, myocardial depression, catecholamine resistance

* Exception: DKA - pH 6.8

* Caution: CHF (Na load), Hypercapnic respiratory failure (CO2), Hypokalemia
Base excess (BE)

* BE: calculated using pH, PCO2, Hgb (or normal Hgb assumed)

* Dose of acid or alkali needed to return pH to 7.40 (at normal body temp & considering normal PCO2 40)

* Normal -3 to +3

* If abnormal recommend treat the underlying cause; may or may not need NaHCO3
Coffee

* Essential ingredient for a successful ICU and a happy Intensivist service

* Studies have shown that coffee may have health benefits, including protecting against Parkinson's disease, type 2 diabetes and liver disease, including liver cancer. It also appears to improve cognitive function and decrease the risk of depression.
D-dimer

- D-dimer is fibrin degradation product (FDP), reflective of ongoing activation of the hemostatic system/ fibrinolytic activity

- Highly sensitive (close to 100%) but non-specific (around 46%); a negative result is useful but a positive result can mean many things

- Can be useful to rule out VTE when the pre-test probability of VTE is low (Wells’ criteria)

- Elevated D-dimer levels associated with advanced age, smoking, post-operative status, trauma, pregnancy, malignancy, infection, DIC, Afib, ACS, GI bleed
Wells’ criteria for PE

- Clinical s/s DVT
- PE is #1 diagnosis or equally likely
- HR > 100
- Immobilization or recent surgery
- Prior PE or DVT
- Hemoptysis
- Malignancy
Extubation assessment

* Vent weaning & extubation - 2 different things

* Provided pt passes SAT (spontaneous awakening trial) & SBT (spontaneous breathing trial) - can we extubate?

* Appx 15% of patients fail extubation in ICU; average 5-10%
My pt passed SBT - can we extubate?
Factors to consider

- Adequate oxygenation / gas exchange on FIO2 < 50 & no excessive secretions
- CXR stable
- Hemodynamics stable
- Adequate mentation & neuromuscular strength (lift head, protect airway) - combination of inadequate cough, excessive secretions & impaired mental status = high failure rate
- Acceptable acid-base status
- Acceptable volume status
- Disease processes stabilized; Apache score < 13
- Avoid nocturnal extubation
Fever in the ICU

- Fever: 38.3 C (101 F)
- Fever 38.3 C (101 F) - 38.8 C (101.9 F) - infectious or noninfectious
- Fever 38.9 C (102 F) - 41 C (105.8 F) - assume infectious
- Fever > 41.1 C (106 F) - usually non-infectious (drug fever, transfusion reaction, heat stroke, AI, thyroid storm, neuroleptic malignant syndrome, malignant hyperthermia)
Infection

* Most common sources in ICU pts: VAP, line related, catheter related, surgical site infection, sinusitis, CDT

* Clinically obvious source of infection - order appropriate diagnostic tests/cultures & add empiric Abx

* If no obvious source: remove & culture lines, remove NGT, imaging (CT sinuses, chest, abdomen/pelvis); consider empiric Abx
Non-infectious causes of fever

- Alcohol/drug withdrawal
- Post op
- Cerebral infarct/hemorrhage
- Hematoma
- Malignancy
- Drug fever (anticonvulsants, Abx, allopurinol)
- ARDS
- DVT/PE
- Mesenteric ischemia
- Acute pancreatitis or acalculous cholecystitis
- Adrenal crisis
- Thyrotoxicosis
- Acute hemolytic transfusion reaction
Group collaboration

“Choosing wisely”

* Critical Care Societies Collaborative (CCSC) established in 2000 as a partnership among the four major critical care societies:
  * AACN (American Association of Critical Care Nurses)
  * ACCP (American College of Chest Physicians)
  * ATS (American Thoracic Society)
  * SCCM (Society of Critical Care Medicine)
Five things Physicians & Patients should question

1. Don’t order diagnostic tests at regular intervals (such as every day), but rather in response to specific clinical questions

2. Don’t transfuse red blood cells in hemodynamically stable, non-bleeding ICU patients with a Hgb > 7 g/dl

3. Don’t use parenteral nutrition in adequately nourished critically ill patients within the first 7 days of an ICU stay

4. Don’t deeply sedate mechanically ventilated patients without a specific indication and without daily attempts to lighten sedation

5. Don’t continue life support for patients at high risk of death or severely impaired functional recovery without offering (pt/family) the alternative of care focused entirely on comfort.
Hemoptysis

* Mild-moderate-massive; massive = 500mL/24 hrs or > 100mL/ hour

* Common causes: bronchitis, bronchogenic carcinoma, bronchiectasis

* Others: infection (TB, mycetoma, lung abscess, other PNA), iatrogenic (ie lung biopsy), pulmonary embolism, coagulopathy, autoimmune disorders, trauma or foreign body, pulmonary AVM or fistula, cocaine, pulmonary venous hypertension (LV failure, mitral stenosis)

* Pseudohemoptysis - ENT or GI source; fabrication
Hemoptysis evaluation

- Associated symptoms i.e. fever, night sweats, weight loss, cough, SOB, CP, joint aches or fatigue, hematuria
- CBC, coags, type & screen
- CTA Chest
- Bronchoscopy
- Arteriogram
- Echo
Massive hemoptysis

- Correctly position patient
- Secure airway
- Hemodynamic support including blood products
- Anticoagulation reversal if applicable
- Intervention: bronchoscopy, embolization, surgery
IVC Filter

- Wire basket device placed via femoral approach into the IVC
- Traps blood clots from pelvic/leg veins to prevent travel to the lungs (PE)
- Retrievable or nonretrievable
- Does not treat blood clots
IVC Filter indications

- **ACCP 2016 guidelines:** “In pts with acute DVT or PE who are treated with anticoagulants we recommend against the use of an IVC filter” (Grade 1B)

- The only two clear cut indications for IVC filter placement are contraindication to anticoagulation (i.e. active bleeding or high risk of bleeding) or failure of anticoagulation (pt develops VTE while on appropriate anticoagulation) in patients for which anticoagulation for VTE indicated.

- At discretion of provider: prophylactically (surgery), extensive clot burden

- Complications are infrequent but do occur - bleeding or infection at puncture site, malposition, migration, thrombosis, fracture; lawsuits

- Retrievable filters should be removed but often patients are lost to follow up; 10-20% unable to retrieve
Ketamine

- Dissociative anesthetic & analgesic; pain relief, amnesia, hypnosis
- Pt may appear awake
- Hemodynamically friendly (elevates HR & BP) & limited respiratory depression
- IVP or infusion
- Combination of Ketamine & Propofol favorable, particularly for procedural sedation
- Adverse effects: emergence delirium/hallucinations, salivation, N/V; reduced seizure threshold; elevated ICP (newer data negative)
Lactate

* Normally produced in the body; increased production occurs during cellular metabolism when insufficient O2
* All tissues can produce & consume
* Normal < 1.25mmol/L; hyperlactatemia >2
  * 2-4 mild
  * 4-8 moderate
  * > 8 severe
Lactic acidosis

- Type A - Hypoperfusion
  - Hypotension, cyanosis, cool/clammy skin, mottling, oliguria-anuria, altered mental status

- Type B - No hypoperfusion
  - Systemic disease, drugs, altered metabolism
Lactic acidosis causes

- **Type A**: (hypoperfusion/decreased oxygen delivery): shock, cardiac arrest, sepsis, severe hypoxemia, limb or mesenteric ischemia, CO

- **Type B**:
  - Severe Asthma / B-agonists
  - Seizure, strenuous exercise or shivering
  - Drugs (HAART, propofol, linezolid, metformin)
  - Malignancy
  - Alcoholism/liver disease
  - Mitochondrial dysfunction

- **D-lactate** (short bowel syndrome, propylene glycol (ingestion or lorazepam/diazepam), DKA); special assay to measure
Why does it matter?

- Multiple studies have demonstrated hyperlactatemia & prolonged/poor lactate clearance is associated with increased mortality
- Key is to fix the underlying cause
Midodrine

- Alpha-1 adrenergic agonist (same as phenylephrine) - increases peripheral vascular resistance/afterload; NO increased HR or contractility
- Onset 45-90min
- Start: 10mg q 8 hours
- May facilitate weaning off IV vasopressors in appropriate pts & facilitate transfer out of ICU, ie septic shock (persistent vasodilatation may take few days to resolve)
- Black box warning: may cause marked elevation of supine blood pressure; can also cause reflex bradycardia, UA retention
- Hold parameter (SBP > 110-120mmHg); stop date 2 days
Norepinephrine

- Vasopressor: strong Beta-1 & Alpha adrenergic effects & mild Beta-2 effect; causes increased contractility & HR along with vasoconstriction; increases systemic BP & coronary blood flow

- Vasopressor of choice for septic shock (numerous trials)

- Also data to support use in cardiogenic shock (in comparison to dopamine - trend toward higher rate of death at 28 days w/ dopamine + significantly more arrhythmias; De Backer, D, et al. 2010. Comparison of dopamine and norepinephrine in the treatment of shock. New England Journal of Medicine 362.9, 779-789.

- Caveat: Afib RVR

- Dosing: mcg/min @ Flagler (others mcg/kg/min); start 3-5 mcg/min; no EB maximum however a reasonable juncture to add 2nd agent: 30 mcg/min; probably lack of efficacy at doses > 2 mcg/kg/min
Norepinephrine peripheral infusion

- Although there are incidents of tissue necrosis after extravasation (and this is listed as caution by manufacturer), there is also some data showing safe usage, particularly at lower dosing; no specific guideline

- One recommendation: OK for short time (1-2 hours) at low dose in a new, functioning INT while awaiting central access; would not allow hypotension/hypoperfusion while awaiting central access

Peripheral noradrenaline; Scancrit.com; June 10 2015
Osmolar gap
(unexplained metabolic acidosis)

* Metabolic acidosis w/ normal SID & you have ruled out lactic, renal or keto (including ETOH) acidosis, check gap

* Simple: difference between the measured serum osmolality (normally 285-295mOsm/kg) & calculated osmolarity

* Calculated: (2 x Na) + glucose/18 + BUN/2.8 + ethanol/3.7; or utilize App

* Normal = < 10

* > 10 consider toxin (i.e. ethylene glycol, propylene glycol, methanol, isopropyl alcohol)
Pulmonary Embolism (PE)

* Thrombus within pulmonary artery; typically originates from another location, frequently lower extremity DVT
* AHA classification: Massive, Submassive or Low risk; based on clinical parameters not thrombus extent or location
* Anticoagulation is TOC, unless contraindication; duration depends on pt.
* 30% mortality if untreated
Massive PE
Size isn’t everything

(*AHA*) - Hemodynamically unstable w/ shock (SBP < 90mmHg for at least 15 minutes or requiring inotropic support, not d/t a cause other than PE), pulselessness, profound bradycardia (HR < 40 with s/s shock). “Saddle embolus” does not equate to Massive PE.

* Fibrinolysis (i.e. TPA/alteplase or TNKase) unless contraindicated

* Catheter or surgical embolectomy if contraindication to fibrinolysis or if remains unstable after fibrinolysis

* RV failure - adequate but not excessive volume, early vasopressor support (NE), NO or Flolan, cautious intubation if necessary - high risk hemodynamic collapse upon induction

* Coding pt - consider TPA 1/2 dose IVP bolus & Epinephrine
Submassive PE

- PE without systemic hypotension but with either RV dysfunction or myocardial necrosis; *varying severity effects treatment algorithm*
  - **RV dysfunction**: RV dilatation by Echo or CT, elevated BNP, EKG changes (i.e. RBBB, anteroseptal ST elevation/depression or T wave inversion)
  - **Myocardial necrosis**: elevated troponin > 0.1
  - **Respiratory failure**

- Fibrinolysis may be considered in pts judged to have clinical evidence of adverse prognosis & low risk of bleeding complications; otherwise catheter or surgical embolectomy; PESI score
Low risk PE

- Normotensive, normal biomarkers, no RV dysfunction by imaging, normal oxygenation
- Best prognosis/ lowest short term mortality
- Anticoagulation; no other modality indicated
- For Low risk or Submassive (lower severity) - early ambulation should be encouraged once pt. is definitively treated
QT prolongation

* Acquired or congenital

* Causes: hypokalemia, hypomagnesemia, SA or AV node dysfunction, drugs

* Torsades de pointes or syncope

* TdP - unsynchronized CV if hemodynamically unstable; magnesium is first line treatment, overdrive pacing, isoproterenol; avoid bradycardia - keep HR 100 bpm
### Drugs & Risk Factors

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Very Probable</th>
<th>Possible in High Risk Patients</th>
<th>Improbable</th>
</tr>
</thead>
<tbody>
<tr>
<td>Antiarrhythmic</td>
<td>Amiodarone, Disopyramide, Procainamide, Sotalol, Quinidine</td>
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<tr>
<td>Antipsychotics</td>
<td>Thioridazine</td>
<td>Chlorpromazine, Haloperidol, Olanzapine, Risperidone</td>
<td></td>
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<tr>
<td>Antidepressants</td>
<td>*</td>
<td>Amitriptyline, Imipramine, Sertraline, Venlafaxine</td>
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<tr>
<td>Anti-infective</td>
<td>*</td>
<td>*</td>
<td>Fluconazole, Levofloxacin, Trimethoprim-Sulfamethaxazole</td>
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MCRT (221 pts) showed NIPPV (BIPAP) for pts who develop respiratory distress within 48 hrs after extubation does not prevent reintubation (and in fact increased risk of death); however preemptive NIPPV (>8 hours) for pts with risk factors for reintubation does appear to prevent reintubation. Nava, S., et al (2005) “Noninvasive ventilation to prevent respiratory failure after extubation in high risk patients” Crit Care Med 33:2465-2470

More emerging data suggesting HFNC (40-50L) immediately following extubation reduces risk of reintubation

FLORALI study (NEJM 2015) - in pts w/ HRF (82% PNA) HFNC (50L): reduction in mortality & days spent on MVS; possibly BIPAP (FFM) inferior d/t interference w/ secretion clearance
Respiratory Failure
Pulmonary edema

- BIPAP isn’t best for everyone


Syncope

- Life threatening conditions:
  - Cardiac (ischemia, arrhythmia, valvular abnormalities, tamponade, PPM dysfunction
  - Pulmonary embolism (massive)
  - Blood loss
  - Subarachnoid hemorrhage
Syncope

- Other less serious causes
  - Vasovagal (autonomic dysfunction)
  - Orthostasis
  - Medications (particularly those lowering HR or BP, or prolonging QT)
  - Neurogenic (TIA, migraine)
  - Metabolic disturbance (hypoglycemia)
  - Hypoxia
  - Anxiety
Syncope evaluation

- Consider associated symptoms i.e. chest pain, SOB, headache; also PMH
- Rule out life threatening conditions - CT Brain, CTA Chest, Echo, EKG, Labs (CBC, chemistry, cardiac enzyme, coags, possibly D-dimer)
- Consultants may include Cardiology, Neurology
- Other studies: carotid US, EEG, Holter monitor
Tracheostomy

- Indications: need for prolonged artificial airway (14 days or longer), poor airway protection/secretions, upper airway obstruction

- Benefits: less need for deeper sedation (several RCT’s), improved pt comfort (uncontrolled reports & clinical opinion), shorter weaning time (1 RCT), shorter ICU & hospital stay (several meta-analyses)

- When to perform? Consider between 2-10 days of intubation, when a projected need for 14 days of intubation - difficult to predict; systematic review early vs late - no difference in mortality or risk of PNA but significant difference in duration of MVS & ICU stay


Ventilation

- **Modes:** AC (TV) or PCV (pressure) - full support, SIMV - partial support, CPAP/PS - no prescribed rate

- **Lung protective ventilation:** current standard of care for MVS; lower TV w/ increased RR; need IBW in Kg; N/A in PS mode
Ventilator issues

* **Auto-peep**: incomplete emptying of alveolar gas at end of exhalation; consequences - decreased preload & increased WOB; treatment - increase expiratory time (decrease MV, increase I-flow rate); severe – pt. hypotensive, tachycardic, high airway pressures - disconnect from Vent

  * Normal MV (minute ventilation) = 10L/min (TV x RR)

  * Normal I:E ratio 1:2

* **Dyssynchrony**: many reasons - check pt. for restlessness, cough, hiccups - may need increased sedation; RT troubleshoot Vent for triggering issues, setting or flow modification; manual BVM
Withdrawal

- Alcohol:
  - Minor vs DT’s (tachycardia, HTN, agitation, hallucinations, diaphoresis, hyperthermia; 5% mortality)
  - Delirium tremens - benzos - symptom triggered therapy preferred (EBM) - often high dose required
  - Withdrawal seizures - no evidence to support anticonvulsants for most cases; if status epilepticus (or refractory DT’s) consider phenobarbital or propofol w/ MVS
  - Antipsychotics (i.e. Haldol) lower seizure threshold & should be avoided
  - Dexmedetomidine (Precedex) - no RCT’s showing prevention of seizures or DT; should not be first line therapy
CXR’s

- ETT placement: 2-5cm above carina (bifurcation)
- Aortic knob - 45 degree angle to locate carina
CXR’s

- Central line tip in SVC
CXR’s

- Left pneumothorax (total)
- Trachea deviates away from the collapsed lung & may have mediastinal shift
CXR’s

- R lung atelectasis (total)
- Trachea deviates toward the atelectatic lung
CXR’s

- R pleural effusion (large)
Thank You!
Additional references

- Life in the Fast Lane (lifeinthefastlane.com)
- EmCrit.org
- Uptodate.com