10 Things You Must Consider in

The Crashing Patient

(Beyond A-B-C and ACLS)

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OBJECTIVES
At the conclusion of this presentation, each participant should be able to…

1. Describe some pitfalls associated with commonly-used resuscitation techniques.
2. Identify how bedside ultrasonography can help in the diagnosis and treatment of moribund patients.
3. Discuss some of the new concepts in the recent AHA guidelines that improve outcomes in cardiac arrest patients.
**Aortic Disasters**

- Consider thoracic aortic dissection (TAD) and abdominal aortic aneurysm (AAA) in crashing/arresting patients, **regardless of presence or absence of prior “typical” symptoms**…prompt diagnosis can save lives!
  - Evaluated patients from a cardiac arrest registry (Austria) that died of either TAD or AAA
  - Atypical presentations were common
    - Patients with AAA: abdominal pain and/or flank pain in only 52% and 32%
    - Patients with TAD: chest pain in only 48%
  - 70% had PEA as the initial cardiac rhythm (most common presenting rhythm)
  - Early routine ECHO/ultrasound in all crashing/arresting patients!
    - TAD ➔ look for pericardial effusion
    - AAA ➔ look for large aorta (> 3 cm diameter)
- Tsai TT, et al. (*Am J Cardiol*, 2005)
  - Hypotension was noted in 29% of TAD patients

**Acidosis**

- Primary metabolic acidosis is associated with compensatory respiratory alkalosis ➔ tachypnea, hyperventilation, Kussmaul’s breathing
  - Concurrent primary respiratory alkalosis (e.g. sepsis, salicylate toxicity) will produce even more profound tachypnea
- **Beware paralysis and intubation!** But if you must, then remember to set respiratory rate high!
  - “Normal respiratory rate” (e.g. 12-16/minute) will cause precipitous fall in systemic pH ➔ arrest
  - If the patient is hypovolemic and you can’t hyperventilate (see below), **consider giving IV sodium bicarbonate** before intubation

**Bagging/Breathing**

  - Professional rescuers (both pre-hospital and in-hospital) often excessively ventilate patients during cardiopulmonary resuscitation (CPR)
  - Resuscitation guidelines recommend a delivery of only *8-10 breaths per minute* during CPR…in other words, one breath every 6 seconds
  - The elevated intrathoracic pressure from hyperventilation produces decreases in preload, cardiac output, coronary perfusion, and cerebral blood flow
  - Animal studies confirm that hyperventilation produces decreased coronary perfusion and decreased survival rates
  - American Heart Association and international guidelines also deemphasize importance of bagging/rescue breathing
Often is too fast, compromises circulation and limits chest compressions

**Baby?**
- Consider ruptured ectopic pregnancy in the crashing/arresting female patient
  - Paradoxical relative bradycardia → common source of confusion in diagnosis
  - Early routine ultrasound in crashing/arresting patients!
- Dysrhythmias
  - Avoid amiodarone in pregnancy
    - Is the only class D antidysrhythmic
    - Risk of fetal hypothyroidism, intrauterine growth retardation, fetal bradycardia, prematurity
    - Only recommended if other drugs fail
    - For ventricular dysrhythmias, use procainamide or lidocaine first
  - Cardioversion/defibrillation is considered safe
    - Fetus has high fibrillation threshold
    - Amount of current reaching uterus is small
    - Be certain to remove fetal and uterine monitors before shocks!
  - Temporary or permanent pacing and implanted defibrillators are considered safe as well
- Positioning during resuscitation
  - International Guidelines recommend…
    - Compressions higher on sternum to adjust for diaphragm and abdominal contents
    - Resuscitation in gravid in partial left lateral tilt position to improve venous return and improve cardiac output (up to 30%)
      - In left lateral tilt position, only 80% of the external compression forces of CPR are transmitted
      - Best compromise for CPR and optimal venous return is in supine position with manual displacement of the uterus to the left
      - i.e. 3-person CPR is optimal
- Perimortem C-section
  - Even if the fetus is not viable emergency Cesarean-section is required in order to improve venous return and consequently the cardiac output during CPR
  - Following delivery of fetus cardiac output in mother can increase up to 80%
  - Even if estimated gestational age is 20-23 weeks!
  - Maternal brain damage is likely after 4 min of cardiac arrest, irreversible after 6 min → perimortem cesarean-section should be considered at the fourth minute of cardiac arrest!

**Compressions**
• Background: AHA guidelines recommend chest compression rate of 100/min in adults
• Valenzuela TD, et al. (Circulation, 2005)
  o Reviewed 61 out-of-hospital cardiac arrests
    ▪ Chest compressions were performed during only 43% of the total time the patients were pulseless!
      • Interruptions were caused by excessive bagging, pulse checks, drug administrations, intubation attempts
• Abella BS, et al. (Circulation, 2005)
  o Evaluated compression rates in 97 cardiac arrests
    ▪ Rate was < 80 in 37%, < 70 in 22%
    ▪ Higher compression rates are significantly correlated with initial return of spontaneous circulation (ROSC)
    ▪ Compression rate does make a difference (and it’s cheap!)
• Hostler D, et al. (Resuscitation, 2005)
  o Using the currently recommended compression:ventilation (C:V) ratio of 15:2, there were only 60 compressions per minute and 26 seconds of “hands-off” time per minute (patient not getting compressions)
  o Best results with 30:2 or greater compression:ventilation ratios (in terms of approaching the recommended 100/min rate
  o New recommendation for 1- or 2-person CPR for adults is 30:2 compression:ventilation ratio
• AHA motto: “Push hard, push fast!”
  o Emphasis on optimizing basic techniques:
    ▪ Proper compression rate (100/min)
    ▪ Minimize interruptions: it is recommended that compressions never be interrupted by more than 15 seconds at a time when doing pulse/rhythm checks…unless, of course, a pulse has returned.
    • Interruptions of compressions should also be minimized or avoided for airway measures, drug administrations, defibrillations, etc.
    ▪ Avoid hyperventilation (bagging rate 12/min)
    ▪ Rapid single defibrillation (see below)
• Cardiocerebral resuscitation (CCR): the future is CCR rather than CPR!
    ▪ Significant de-emphasis on early ventilations in “typical” cardiac arrest (doesn’t apply to pulmonary arrest, e.g. drowning, opiate OD, crashing asthma/COPD, pediatric arrests, etc.)
    ▪ Passive oxygenation, good compressions, early defibrillation when needed and early EPI
    ▪ No positive pressure ventilation/intubation for the first 8-12 minutes!
    ▪ Increased survival and neurological outcome!

Cooling
• International Liaison Committee on Resuscitation (ILCOR) recommendations:
Unconscious adults with spontaneous out-of-hospital cardiac arrest and an initial rhythm of ventricular fibrillation should be cooled to 32-34°C (~90-93°F) for 12-24 hours.

Based on two studies (Austrian study, 275 patients; Australian study, 77 patients), cooling may also be beneficial for other rhythms or for in-hospital cardiac arrest.

- **Methods of cooling**
  - Cooling blankets
  - Ice packs to groin, neck, and axillae
  - Wet towels and fanning
  - Cooling “helmet”
  - Cool intravenous fluids to 4°C
  - Internal methods generally considered too invasive for routine use

- **Prevent shivering with paralytics and sedatives**
  - **Warnings**
    - Increased incidence of dysrhythmias, infection, coagulopathy if < 32°C
    - Uncertain in pediatrics
    - Not intended for cardiogenic shock, pregnancy

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**“Decline position” (TrenDelenburg)**

  - Maneuver fails to increase blood pressure and/or cardiac output in most patients, does not improve tissue oxygenation, results in displacement of only 1.8% of total blood volume, and actually decreases cardiac output in the hypotensive patients.
  - Produces right ventricular stress and deterioration of pulmonary function.
  - Also of limited benefit in placing central venous catheters (subclavian, internal jugular)

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**Defibrillation**

- Monophasic vs. biphasic defibrillators?? → “...no specific waveform (either monophasic or biphasic) is consistently associated with a greater incidence of ROSC or survival to hospital discharge rates after cardiac arrest than any other specific waveform. Research indicates, however, that when doses equivalent to or lower than monophasic doses are used, biphasic waveform shocks are safe and effective for termination of VF.” (*Circulation*, 2005;112(24):page IV-37)

- If unknown down-time or collapse > 4-5 minutes ago in ventricular fibrillation (VF) arrest, new recommendation → **2 minutes of chest compressions before first shock**

- Repeated sequence of initial shocks and escalating dosages of current no longer recommended
  - Modern biphasic defibrillators reportedly have a 90% first-shock efficacy at terminating (at least temporarily) VF
Even with monophasic defibrillators, if the first shock is ineffective, it is unlikely that the subsequent 2 shocks will be effective.

Data does not support the efficacy of escalating dosages of current for either monophasic or biphasic defibrillators, therefore…

Therefore, the current recommendation is that only a single shock be given for VF at a time. Biphasic defibrillators should be set at 120 J – 200 J; monophasic defibrillators should be set at 360 J (you start with 360 J).

- Immediately after the shock is delivered, 2 minutes of CPR should be initiated before the pulse check and before the rhythm check. In other words, shock the patient, do 2 minutes of CPR, then check the rhythm and pulse. This applies not just to the first shock, but to every shock given. This again is meant to minimize delays in compressions.

**Effusion (pericardial)**

- Common cause of unexplained dyspnea
  - Dyspnea and tachypnea caused by pericardial fluid compression bronchial structures
  - Alveolar oxygen exchange should be unaffected → oxygen saturation usually normal
- Marked cardiomegaly
- ECG triad: low voltage (very common), tachycardia (very common), electrical alternans (< 30%)
- Patients are preload dependent → IVF often helps BP
  - Beware intubation, positive pressure ventilation!
    - Decreases venous return → decreases cardiac output and BP
    - Think of this when you see sudden deterioration immediately after intubation; treat with IVF
- Early routine ECHO in crashing/arresting patients!

**Embolus (pulmonary)**

- Beware overzealous IV hydration…may see BP start falling with IVF!
  - Use vasopressors earlier, e.g. dobutamine, dopamine, norepinephrine
- Hypoxia often present with massive PEs, intubation is a better choice
  - May actually help BP by decreasing venous return and RV overload
- ECG: new T-wave inversions common in cases of massive PE, especially in anteroseptal and inferior leads
  - Other ECG clues: rightward axis, tall R-wave in lead V1
- Early routine ECHO in crashing/arresting patients!
- Hemodynamically unstable patient with suspected PE (RV distention or dysfunction) is reasonable indication for **empiric thrombolytics**

Amal Mattu, MD
The Crashing Patient
Forget About It!
A. High Dose Epinephrine (HDE)
- HDE may be associated with an increase in return of spontaneous circulation (ROSC), but is not associated with an increase in hospital discharge or neurologic recovery; may be associated with a decrease in neurologic recovery.
  - Lipman, et al. (Anaest Intensive Care, 1993)
  - Rivers, et al. (Chest, 1994)
  - Sherman, et al. (Pharmacotherapy, 1997)

B. Amiodarone
- Pharmacology
  - Class III antidysrhythmic (potassium channel blockade)
  - Also has some class I (sodium channel blockade), class II (beta blockade), and class IV (calcium channel blockade) activity
  - As effective as procainamide or bretylium in ventricular tachycardia (VT)
  - 60–80% “effective” in refractory VT (outcome??)
  - Drawbacks — hypotension (may require vasopressors), bradycardia (may require pacing), expensive

  - Manufacturer-supported study (Wyeth Ayerst)
  - Seattle prehospital study of patients with shock-resistant VT/VF
  - Patients received three shocks, intubation, epinephrine (EPI) 1 mg
  - Then received either 300 mg amiodarone (246 patients) or placebo (258 patients)
  - Subsequent treatment at the discretion of medics, physicians—followed general Advanced Cardiac Life Support (ACLS) guidelines
  - Results
    - Amiodarone patients more likely to survive to hospital admission than placebo patients (44% vs. 34%; p=0.03)
    - No significant difference between amiodarone and placebo groups for survival to hospital discharge (13.4% vs. 13.2%) or good neurologic outcome (7.3% vs. 6.6%)

  - Manufacturer-supported study (Wyeth Ayerst)
  - Canadian prehospital study of 348 patients with shock-resistant VT/VF
  - IV lidocaine vs. amiodarone
  - Patients received three shocks, epinephrine, repeat shock
  - Then received either 1.5 mg/kg lidocaine or 5 mg/kg amiodarone
    - Then repeat shock, EPI, then half-dose of the study drug
  - Preliminary results presented March 2001
- Amiodarone patients more likely to survive to hospital admission than lidocaine patients (22.7% vs. 11%)
  - Survival to discharge “data still being analyzed…”
    - Final results presented November 2001, AHA Conference
    - No significant difference in survival to hospital discharge

C. Vasopressin

- Pharmacology
  - Naturally occurring antidiuretic hormone
  - V1 receptors in vascular smooth muscle; V2 receptors in renal collecting duct
  - In high doses, acts as a potent non-adrenergic peripheral vasoconstrictor
    - Increases diastolic aortic blood pressure and coronary perfusion pressure
  - Effect is similar to epinephrine’s alpha effect but without the beta effect
  - Beta effect is associated with increased myocardial oxygen consumption
    - Increased incidence of post-resuscitation myocardial infarction

- Pig studies of cardiac arrest
  - Improved coronary perfusion pressure
  - Improved vital organ blood flow
  - Improved cerebral oxygen delivery
  - No increase in myocardial oxygen demand
  - Improved ROSC

  - Eight cases of hospitalized patients with VF resistant to standard ACLS treatment
    - Vasopressin 40 units given; all patients had ROSC
    - Three patients survived to hospital discharge

- Lindner, et al. (Lancet, 1997)
  - Vasopressin vs. EPI; randomized, double-blind trial
  - Forty out-of-hospital patients with VF resistant to initial CPR and defibrillation received either EPI 1 mg or vasopressin 40 units, then further standard ACLS measures
    - ROSC: 11/20 EPI, 16/20 vasopressin
    - Survival to hospital admission: 7/20 EPI, 14/20 vasopressin
    - Survival > 24 hours: 4/20 EPI, 12/20 vasopressin
    - Survival to hospital discharge: 3/20 EPI, 8/20 vasopressin
      - Survival to hospital discharge not statistically different (small study)

- Stiell, et al. (Lancet, 2001)
  - Vasopressin vs. EPI; randomized triple-blind trial
  - 200 in-hospital cardiac arrest (undifferentiated) patients requiring drug therapy received either EPI 1 mg or vasopressin 40 units, then EPI if “rescue” medications required
Survival one hour: 35% EPI vs. 39% vasopressin (not statistically different)
Survival to hospital discharge: 14% EPI vs. 12% vasopressin (not statistically different)
No difference in neurologic outcomes
Conclusion: “We cannot recommend the routine use of vasopressin for in-hospital cardiac arrest patients, and disagree with American Heart Association guidelines, which recommend vasopressin as alternative therapy for cardiac arrest.”

- Vasopressin (VP) vs. EPI; randomized pre-hospital trial
- 1186 patients randomized
  - Either received 40 IU VP (x 2) or 1 mg EPI (x 2)
  - Then received additional EPI as needed
- Ventricular fibrillation: no signif. difference
- Pulseless electrical activity (PEA): no significant difference
- Asystole:
  - VP group had higher survival to hospital admission
    - 29.0% vs. 20.3%
  - VP group had higher survival to hospital discharge
    - 4.7% vs. 1.5%
- 732 patients in whom spontaneous circulation was not restored with the first 2 injections, then received further EPI
  - VP patients: 25.7% survived to admission, 6.2% survived to discharge
  - EPI patients: 16.4% survived to admission, 1.7% survived to discharge
- Conclusions
  - VP and EPI are similar for ventricular fibrillation and PEA
  - VP is better than EPI for asystole
  - VP followed by EPI is better than EPI alone in the treatment of refractory cardiac arrest
- All patients: identical survival, but VP associated with worse neurologic outcome
  - Survival to hospital discharge
    - 9.9% in both groups
  - Cerebral performance VP vs. EPI
    - Good cerebral performance 32.6% vs. 34.8%
    - Moderate disability 15.2% vs. 26.1%
    - Severe disability 19.6% vs. 15.2%
    - Coma/vegetative state 32.6% vs. 23.9%

Summary for drugs in cardiac arrest:
- Antiarrhythmics: “There is no evidence that any antiarrhythmic drug given routinely during human cardiac arrest increases survival to hospital discharge.”
- Vasopressors: “To date no placebo-controlled trials have shown that administration of any vasopressor agent at any stage during management of pulseless VT, VT, PEA, or asystole increases the rate of neurologically intact survival to hospital discharge.”
Final Points

- Post-resuscitation care is now receiving greater emphasis as well.
  - Avoid hyperthermia (e.g. fever), hyperventilation, hyperglycemia, and further cardiac ischemia post-resuscitation.
  - Closely monitor vital organ function (e.g. renal function) post-resuscitation. End-organ failure will obviously contribute to worse outcomes.
  - Early cardiac catheterization for survivors of cardiac arrest (definitely if STEMI…possibly even if no evidence of STEMI!)

The “4 Cs” of cardiac arrest: Cardioversion, Compressions, Cooling, Catheterization!

**Recommendation for Further Reading**
If you enjoy reading about and learning from “Pitfalls,” check out the following text:

Questions? Contact me:
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