Periop Emergencies

1. Emergency Assessment for 2014

1.1 Periop Emergencies

1.2 Crisis Resource Management

What?
- A set of common principles to apply to a variety of stressful situations
1.3 Crisis Resource Management

Crisis Resource Management

1. **Know the environment** - how to locate and operate your equipment, trouble shooting, alternatives
2. **Anticipate/Plan** - some patients are at greater risk than others
3. **Call for help** - self-explanatory
4. **Designate leadership** - someone must step back from procedures to guide others, otherwise you may focus on one aspect while more important steps are missed
5. **Role clarity** - each person has their job
6. **Distribute the work** - one person can’t do it all

2. **Use cognitive aids (checklists)** - improve adherence to guidelines
3. **Mobilize resources** - initiate other teams as necessary (cath lab, OR, ICU, IR)
4. **Allocate attention/use all information** - do not fixate on one problem, one diagnosis

1.4 Untitled Slide

BLS SAVES LIVES!
(The rest is fluff)

Pay attention to good chest compressions (CC) and early defibrillation
1.7 Untitled Slide

- The difference between this and the previous graphic is the two pathways for Shock or “No shock advised”
- Shock for VF/VT
- “No shock advised” is Pulseless Electrical Activity (PEA) or Asystole
- Drugs
  - Epinephrine 1 mg IV/IO every 3-5 mins and for Vasopressin 40 units IV/IO once increase peripheral vascular resistance to (hopefully) perfuse the head and heart
  - Amodarone 300 mg IV/IO is main antiarrhythmic for shock resistant VF/VT
  - Other drugs may be considered but, are not necessarily part of this algorithm

1.8 Key concepts to ACLS

Key Concepts to ACLS

- Call for HELP!
- Focus on chest compressions and appropriate early defib
- Emphasize chest compressions (CC) and de-emphasize ventilation
  - Why?
    - Normally, venous blood returns to right heart and is pumped to the lungs where ventilation brings in O₂ and eliminates CO₂
    - BUT, without cardiac output, there is no pumping blood to the lungs, no pulmonary blood flow, so no O₂ is brought to the lungs and no CO₂ is added to the blood
    - Therefore, in settings of low or no cardiac output, any ventilation is deadspace (ventilation without pulmonary blood flow/cardiac output) and therefore not useful
    - Further, ventilation in this setting increases intrathoracic pressure and further reduces venous return
    - And, securing around with ventilation and advanced airway devices interferes with CC
- Resume CC after shock
- Don’t check rhythm, don’t feel for a pulse. Even if there is a return of organized conduction, the heart is unlikely pumping effectively for the first several minutes
### Key Concepts to ACLS

**Big changes in 2010:**
- Monitor end-tidal CO₂ (ET CO₂) whenever CC are ongoing and if ET CO₂ is <10mmHg, improve CC
  - **WHY?**
    - Venous blood brings CO₂ back to the body. That venous, CO₂-rich blood is pumped out of the right ventricle to the lungs where it diffuses into the alveoli and then is exhaled.
    - So, if there is no right heart output, there is no pulmonary blood flow, there is no CO₂ in exhaled breath.
    - In cardiac arrest, there is no cardiac output, no pulmonary blood flow, no CO₂ in exhaled breath unless there is good CC. Put another way, CC are creating whatever cardiac output there is. In this setting, if there is little to no ET CO₂, the CC are ineffective.
  - So, ET CO₂ provides a rough estimate of cardiac output.

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### Key concepts to ACLS

**Big changes in 2010:**
- Biphasic (120-200J) or monophasic (360J) shocks - no meaningful difference btw these two devices, use the device specific recommended energy or the maximum energy available on that device
- Drugs for cardiac arrest: epinephrine 1mg IV, vasopressin 40units IV or amiodarone 300mg IV (for shock resistant VF or VT)
- Ventilation is not that important during adult cardiac arrest (at least until cardiac output is restored), therefore intubation or placement of other advanced airways are not stressed.
1.11 Near-arrest situations

Near-arrest Situations

- Remember, the name of the game is deliver oxygen to vital organs.
  - $O_2$ delivery $\sim O_2$ content in blood x cardiac output
- Inadequate oxygen delivery manifests as shortness of breath, dizziness, mental status changes, increasing lactate, signs of heart failure, etc

1.12 How is blood pressure related to cardiac output (oxygen delivery)?

How is blood pressure related to cardiac output (oxygen delivery)?

- Cardiac Output (CO) = Heart rate (HR) x stroke volume (SV)
- Also, remember Ohm's law, flow = change in pressure divided by resistance (I=V/R)
- So, whole body blood flow (also known as cardiac output) is approximately (MAP-CVP)/SVR
- So, BP can be high even when CO is low (if SVR is high) and BP can be low when CO is high (if SVR is low)
  - This is why in a failing heart, we strive for afterload reduction, which promotes forward flow and oxygen delivery
  - And, just because the BP is “ok”, that doesn’t mean oxygen delivery is ok
Back to近-arrest situatins

**1.13 Back to near-arrest situations**

Back to Near-arrest Situations

- What is too high for a HR?
  - Rule of thumb: 220-age is max HR
  - Higher HR than max may compromise diastolic filling
- So, check out the AHA/ACC algorithm for tachycardia

**1.14 Untitled Slide**

[Diagram of Adult Tachycardia (With Pulse)]

<table>
<thead>
<tr>
<th>Persistent tachycardia (no shock)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes: Synchronized cardioversion</td>
</tr>
<tr>
<td>No: Consider adenosine (or calcium channel blocker)</td>
</tr>
</tbody>
</table>

**Brachycardia**

- Synchronized cardioversion
  - 150-200 J
  - Adenosine 6 mg IV push (2 mg bolus)
  -/or 10 mg IV push (2 mg bolus)

**Synchronized Cardioversion** (No shock)

- Consider adenosine only if regular and nonhemodynamically significant

**Other hemodynamic or electrolyte abnormalities**

- Consider expert consultation

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1.15 Key points to tachycardia

Key Points to Tachycardia

- O₂ is rarely harmful for adults (so, use it)
- If there is inadequate O₂ delivery, rapidly move to shock
  - Whenever there is a recognizable pattern in the ECG, use synchronized shocks
    - Sync tries to deliver the shock not on the T-wave
    - If the shock occurs during the T-wave, you may decompensate (go to V-fib), known as the “R-on-T” phenomenon
  - Narrow complex tachycardias come from at or above the AV node
    - Therefore, they require less electricity since there is less atrial muscle mass to depolarize, to put the refractory phase

1.16 Key points to tachycardia

Key Points to Tachycardia

- While preparing to cardiovert, you may consider sedation, antiarrhythmics, etc
- What conditions may put a patient at risk for tachydysrhythmia?
  - Low K, low Mg, mostly
  - Sympathetic excess (like pain)
  - Myocardial ischemia
  - Volume overload
1.17 What causes myocardial ischemia?

Myocardial ischemia either arises from . . . .

- An imbalance between myocardial oxygen supply and demand

OR

- PLAQUE RUPTURE/CORONARY THROMBOSIS

1.18 What causes myocardial ischemia?

- Myocardial ischemia arises from an imbalance btw myocardial O₂ delivery vs O₂ consumption
- So, what is myocardial O₂ delivery?
  - Remember O₂ delivery is determined by blood flow and blood oxygen content
  - For the heart specifically, we’re talking about coronary blood flow (CBF)
  - CBF ~ (diastolic BP - left ventricular end-diastolic pressure)/coronary vascular resistance
1.19 What causes myocardial ischemia?

What causes myocardial ischemia?

- Why diastolic BP?
  - The pressure inside the ventricle during systole must be greater than the pressure in the aorta to eject blood.
  - The pressure in the wall of the ventricle, where the coronary arteries perforate, is about the same as that in the ventricle. So, there is little to no CBF during systole (to the left ventricle, that is).
  - During diastole, the aortic valve closes (as indicated by C in the figure) and the pressure in the aorta stays high (dBP) while the pressure inside the ventricular wall falls (isosystolic relaxation and diastolic filling).
  - So, most coronary blood flow occurs during diastole and dBP is the pushing pressure.

- Why LV end-diastolic pressure (LVEDP)?
  - During diastole, the ventricle relaxes and the pressure falls to less than the pressure in the left atrium and the mitral valve opens (as indicated by D in the figure).
  - So, the pressure in the LV wall is about the same as the pressure in the ventricle and the coronary arteries pass through the wall, so that LVEDP is the back-end pressure the blood is flowing against.

1.20 What causes myocardial ischemia?

What causes myocardial ischemia?

- What about coronary vascular resistance?
  - Many organs ‘autoregulate’ their blood flow to maintain a constant supply by constricting or dilating blood vessels.
  - For the heart, all the things that are by-products of ischemia dilate coronary arteries (lactate, H+, adenosine, K+, etc.).
  - So, when someone is experiencing myocardial ischemia, there is little we can do to further medically dilate the coronaries.
1.21 What causes myocardial ischemia?

What causes myocardial ischemia?

- In addition to coronary blood flow, there must be O₂ carried by that blood
- The arterial O₂ content (CaO₂) = how much O₂ hemoglobin is carrying plus the amount of O₂ that is dissolved in blood
  - \[ CaO₂ = 1.34 \text{ml O}_2/\text{gHgb} \times Hgb \text{ (g/dL of blood)} \times \text{saturation (SpO₂)} + \text{PaO}_2 \times 0.003 \text{ mL O}_2/\text{mmHg} \]
- So, give supplemental O₂ and consider transfusion (but, don’t forget about the countless ways transfusion can harm)

1.22 What causes myocardial ischemia?

What causes myocardial ischemia?

- So, what about myocardial O₂ demand?
  - The heart is a muscle; like any muscle it consumes O₂ when it does work
  - So, how hard is the heart working? \( (HR \times P \times r)/2H \)
    - \( HR = \) Heart rate, the number of times the heart contracts in a minute
    - \( P = \) Afterload, the pressure the heart must generate (or weight the muscle must lift)
    - \( r = \) radius or the Preload, the distance the heart moves
    - \( H = \) wall thickness, the bigger the muscle, the easier it is to lift a weight
    - Unfortunately for wall thickness, the thicker the muscle, potentially the higher the LVEDP
1.23 Untitled Slide

Acute Coronary Syndromes

Symptoms suggestive of ischemia or infarction

EMS assessment and care and hospital preparation:
- Monitor, support ABCs. Be prepared to provide CPR and defibrillation
- Administer aspirin and consider oxygen, nitroglycerin, and morphine if needed
- Obtain 12-lead ECG; if ST elevation:
  - Notify receiving hospital with transmission or interpretation; note time of onset and first medical contact
  - Notify hospital should mobilize hospital resources to respond to STEMI
- If considering prehospital fibrinolysis, use fibrinolytic checklist

Immediate ED general treatment
- If O2 sat <91%, start oxygen at 4 L/min, titrate
- Aspirin 160 to 325 mg (if not given by EMS)
- Nitroglycerin sublingual or spray
- Morphine IV if discomfort not relieved by nitroglycerin

Concurrent ED assessment (<10 minutes)
- Check vital signs; evaluate oxygen saturation
- Establish IV access
- Perform brief, targeted history, physical exam
- Review/complete fibrinolytic checklist (Figure 2);
  - check contraindications (Table 5)
- Obtain initial cardiac marker levels, initial electrolyte and coagulation studies
- Obtain portable chest x-ray (<30 minutes)

1.24 Untitled Slide

ST elevation or new or presumably new LBBB, strongly suspicious for injury
- Elevation in V6/V2/V4

ST depression or dynamic
- T-wave inversion, strongly suspicious for ischemia
- High-risk chest pain (angina or T wave)<1.5mm

Reperfusion goals
- Thrombolysis:
  - EPA guidelines
  - Hemorrhagic stroke (H- or LWM)
  - Care provider decision

Reperfusion therapy:
- Thrombolysis
  - Primary percutaneous transluminal coronary angioplasty (PTCA)

Consider administration to ED chest pain unit or to appropriate bed and follow:
- Serial cardiac markers (including troponin)
- Repeat ECG/continuous monitoring
- Hemodynamic monitoring

Consider noninvasive diagnostic test

Yes

Clinical high-risk features
- Dynamic ECG changes consistent with ischemia
- Troponin elevated

If no evidence of ischemia or interaction by testing, can discharge with follow-up
1.25 How do we optimize myocardial O2 supply vs O2 demand?

How do we optimize myocardial O₂ supply vs O₂ demand?

- Don’t get too vasodilated (too low DBP - decreases O₂ supply)
- Don’t get too volume overloaded (too high preload - increases O₂ demand)
- Don’t get too hypertensive (too high afterload - increases O₂ demand)
- Don’t get too tachycardic (increases O₂ demand)
- Don’t get too hypoxic (decreases O₂ supply)
- Don’t get too anemic (decreases O₂ supply)

- However, most of the time a coronary thrombus (or two) is causing the ischemia
- Optimizing supply and demand won’t fix that
- Antiplatelet (aspirin, clopidogrel, glycoprotein Ilb/IIIa inhibitors) and antithrombotic (UFH/LMWH) meds are needed and potentially percutaneous coronary intervention