RECOGNITION AND TREATMENT OF CARDIAC DYSRHYTHMIAS IN THE OPERATING ROOM AND BEYOND

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CARDIAC RHYTHM OUTLINE
- Cardiac physiology
- Cardiac Electrical Activity
- Arrhythmia Recognition
- Arrhythmia Treatment
- Case Study in Treatment

CARDIAC ELECTRICAL TRANSMISSION
- Need to understand timing and "spread" of depolarization
- Effect it has on cardiac cycle
- "Where" does the "signal" eminate from?
- How does it effect function and rhythm?
- Can I treat or block the rhythm?
- "Fixed" or "Non-Fixed" Defect
- Underlying defect? Electrolytes?
- Chemical vs. Electrical Treatment
CARDIAC CONDUCTION

Electrical System of the Heart

CARDIAC PRESSURE VOLUME CYCLES

Cardiac Contraction Cycle
ARRHYTHMIAS

ATRIAL

- Atrial Fibrillation
- Atrial Flutter
- Atrial Tachycardia

ATRIAL FIBRILLATION

- Chaotic Rhythm
- No Organized Atrial Activity
- 30-40% loss of Cardiac Output
- "Chronic" or "Acute"
- Stability??
- May need immediate Treatment

ATRIAL FIBRILLATION

CAUSES

- Ischemia
- Electrolytes
- Acidosis
- Toxicity
- Temperature
- Volume Overload
- Advanced Age
- Trauma
- Endocrine
- Sepsis
ATRIAL FIBRILLATION

- Determine stability
- Chronic vs. Acute
- Electrolytes
- Medication Induced
- Treatment Driven

ATRIAL FLUTTER

- Hypertension
- Hypothyroidism
- Pulmonary Edema
- Drugs and substances
- Alcohol (beer, hash, or hard liquor)
- Stimulants such as narcotics, diazepam, and even caffeine.

TREATMENT ATRIAL FLUTTER

- Intravenous adenosine: This drug, administered as an intravenous push followed by an intravenous bolus with flush, can also be helpful in making the diagnosis of atrial flutter by transiently blocking the AV node.
TREATMENT ATRIAL FLUTTER

- Restoration of sinus rhythm

After determining the patient's needs for anticoagulation and ventricular rate control, the issue of restoration of the sinus rhythm can be safely addressed.

- Radiofrequency ablation
  - Radiofrequency ablation is often used as first-line therapy to permanently restore sinus rhythm. This procedure is often performed electively, rather than in the acute setting, to restore sinus rhythm.

- Electrical cardioversion
  - The success rate of electrical cardioversion is higher than 95%.

- Pharmacological cardioversion
  - Flecainide is only effective in approximately 10% of patients.

Prevention of complications:

- Thromboembolic
  - Patients with atrial flutter are at increased risk of thromboembolic complications compared with the general population. The anticoagulation strategy used for atrial fibrillation is also recommended for atrial flutter.

ATRIAL TACHYCARDIA

- Atrial tachycardia arises from a small area (focus) of tissue, anywhere in the atria of the heart (see diagram). This focus starts to fire and drive the heart, more rapidly than the heart's natural pacemaker.

- It can continue for days or even persist for months at a time. In some patients (especially the elderly or those with other significant heart disease) there is more than one abnormal focus.

ATRIAL TACHYCARDIA CAUSES

- Atrial tachycardia can occur for a number of reasons:
  - Birth defects
  - Valve problems
  - Damaged or weakened heart muscle (e.g., from a prior heart attack or inflammation of the heart)
  - Drug/alcohol intoxication
  - Metabolic disturbances such as an overactive thyroid or adrenal gland

However, in most patients no particular cause is found. Atrial tachycardia's are so common that they can be considered normal.
ATRIAL TACHYCARDIA
TREATMENT
Because atrial tachycardia is not generally dangerous, treatment is only required if it is causing you symptoms. Often the rhythm disturbance is a chance intermittent finding on an ECG and no treatment is needed. However, if the patient experiences unpleasant symptoms or a permanently increased heart rate is risking heart enlargement, your doctor may recommend treatment with medication or catheter ablation.

SUPRAVENTRICULAR TACHYCARDIA
Tachycardia is the term describing an abnormally rapid heart rate of more than 100 beats per minute.

Paroxysmal, or sporadic, supraventricular tachycardia usually occurs without other symptoms. However, it may be associated with a number of medical conditions, such as the following:

- Certain drugs and social habits
- Cocaine abuse
- Alcohol abuse
- Smoking
- Drinking too much caffeine in coffee, tea, or soft drinks
- Emotional stress
- Heart failure
- Throat disease
- Chronic lung disease
- Pancreatitis
- Pulmonary embolus, or blood clots that enter the lung through damaged tissue in the body
- Pancreatitis
TREATMENT OF SVT

ARRHYTHMIAS

VENTRICULAR

- Ventricular Tachycardia
- Ventricular Fibrillation
- Torsades de Pointes

Ventricular Tachycardia

Normal Sinus Rhythm

Ventricular Tachycardia
Ventricular Tachycardia

Description

- **Ventricular tachycardia** is a condition where the heart undergoes a series of rapid heartbeat rhythms.
- Diagnosis of this condition may sometimes be difficult as classifying irregular heart rhythms can be difficult and may be confused for other conditions such as supraventricular tachycardia. But VT is defined in conditions where there are at least 3 beats coming from either ventricle of the heart at rates over 100 beats per minute. This heart rate may span from 120 to 300 beats per minute.
- The rhythm may be felt in short bursts or as a sustained rhythm.
- VT can be life-threatening as it may lead to ventricular fibrillation or the rapid irregular trembling and beating of the heart which can cause cardiac arrest, then eventually death. Rarely though, some cases of VT may be benign, especially when experienced in younger patients.
- Pulse vs. Pulseless

Ventricular Tachycardia Causes

- The most common setting for VT is **ischemic heart disease**, in which myocardial scar is the substrate for electrical reentry.
- Nonischemic cardiomyopathies
- Ion channel abnormalities and other conditions in which cardiac function and structure are normal.
- Electrolyte abnormalities

Monomorphic V-Tach

- Monomorphic ventricular tachycardia
  
  When the ventricular activation sequence is constant, the electrocardiographic pattern remains the same, and the rhythm is called monomorphic VT.
  
  Monomorphic VT is most commonly seen in patients with underlying structural heart disease. It's typically seen in cases of heart disease, most commonly due to damage from coronary artery disease. In some cases, ion channel abnormalities or electrolyte imbalances can also cause monomorphic VT. Understanding the mechanism of monomorphic VT involves understanding the reentrant circuit. The reentrant circuit involves an electrical wavefront traveling through the same path, causing the rapid, irregular heartbeat.
  
  Monomorphic VT can be treated with medications, devices, or surgery to interrupt the reentrant circuit. It's important to monitor and manage any underlying heart disease to prevent recurrence.
Polymorphic VT and torsades are also observed in drug-free, structurally normal hearts when patients have genetic abnormalities affecting performance or intracellular processing of cardiac ion channels. Examples include long QT syndrome or short QT syndrome.

Broad Treatment of V-tach

- If unstable may proceed directly to 
- If not, treat according to morphology:
  - Monomorphic VT
    - EF normal, one or more of the following:
      - procainamide (2a)
      - sotalol (2a)
      - amiodarone (2b)
      - lidocaine (2b)
    - EF poor
      - amiodarone 150 mg IV over 10 min OR lidocaine 0.5–0.75 mg/kg IV push
      - Synchromed
  - Polymorphic VT
    1. Baseline QT Normal
      - Possible ischemia, electrolyte (esp. low K), Mg; abnormality (correct)
      - EF normal: betablocker, lidocaine, amiodarone, procainamide, or sotalol
    1. Prolonged QT baseline (torsades)
      - Synchromed, magnesium, isoproterenol
      - Treatment option: magnesium, overdrive pacing, isoproterenol

Ventricular Fibrillation
VENTRICULAR FIBRILLATION CAUSES

The most common cause of ventricular fibrillation is inadequate blood flow to the heart muscle due to coronary artery disease, as occurs during a heart attack. Other causes include the following:

- Shock (very low blood pressure) which can result from coronary artery disease and other disorders
- Electrical shock
- Drowning
- Very low levels of potassium in the blood (hypokalemia)
- Drugs that affect electrical currents in the heart (such as sodium or potassium channel blockers)

VENTRICULAR FIBRILLATION ACLS ALGORITHM

- Remember: initial stacked shocks are part of the algorithm. Implement the algorithm after your stacked shocks.
- Meds: drug-shock-drug-shock pattern. Continue CPR while giving med, and shock (360J or 150J if biphasic) within 30-60 seconds. Evaluate rhythm and shock for pulse immediately after shocking.
- Epinephrine or vasopressin big drugs (may give either one as first choice).
- If VF/VFVT persists, may move on to antiarrhythmics and sodium bicarb.
- Max out one antiarrhythmic before proceeding to the next in order to limit pro-arrhythmic drug-drug interactions.

"Think Shock Shock Shock, Everybody Shocks: Anna Nicole Shocks, Lydia Possner Shocks, Madeleine Cox Shocks, Pamela Anderson Shocks, Bridget Hall Shocks"... this one needs some work. I couldn't think of enough names, so did a quick search for "models" and found a list - I recognized only a few names; choose your own favorites (this page happens to have only females, I think)

VENTRICULAR FIBRILLATION ACLS ALGORITHM

- AED
  - Use paddle shock as needed, shock immediately
  - Do not default to shock if patient is bradycardic and no emergency
- Medications
  - Epinephrine 1 mg IV q 3-5 min
  - High dose epinephrine is no longer recommended
  - Vasopressin 40 U IV; one time dose (wait 5-10 minutes before starting epi)
Ventricular Fibrillation
ACLS ALGORITHM

- Block NSC
- Amiodarone
  - 300mg IV push
  - May repeat once at 150mg in 3-5 min
  - max cumulative dose = 2.2g IV/24hrs

- Lidocaine (Class Inderterminate)
  - 1.0-1.5 mg/kg IV q 3-5 min
  - max 3 mg/kg

- Magnesium Sulfate
  - 1-2 g IV (over 2 min) for suspected hypomagnesemia or torsades de pointes (polymorphic VT)

- Procainamide
  - 30 mg/min or 100 mg boluses q 5 min, up to 17 mg/kg.
  - Besides having a pro-arrhythmic drug-drug interaction with amiodarone, procainamide is of limited value in an arrest situation due to lengthy administration time.
  - Note: bretylium acceptable but no longer recommended in ACLS

- Bicarbonate
  - 1 mEq/kg IV for reasons below:
    - hyperkalemia
    - bicarbonate-responsive acidosis, toxic OD, ts, midepsines, but not for aspirin OD
    - prolonged arrest
  - Not for hypercarbia-related acidosis, nor for routine use in cardiac arrest

* Or equivalent biphasic shocks (150J-150J-150J).

Ventricular Fibrillation
Definitive

- Automatic Implantable Defibrillator
- Risk during placement
- External Defibrillator
- Arterial Line
- General Anesthetic
- Understand underlying causes
- High Risk
- Pre-op

Electrolyte Arrhythmias
Potassium

- Hypokalemia
- Hyperkalemia

<table>
<thead>
<tr>
<th>Electrolyte Level</th>
<th>Arrhythmia</th>
</tr>
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<tbody>
<tr>
<td>3.5-4.5 mEq/L</td>
<td>Normal</td>
</tr>
<tr>
<td>&lt;3.5 mEq/L</td>
<td>Hypokalemia</td>
</tr>
<tr>
<td>&gt;5.0 mEq/L</td>
<td>Hyperkalemia</td>
</tr>
</tbody>
</table>
CAUSES OF HYPOKALEMIA

HYPOKALEMIA

Hypokalemia is serum K concentration < 3.5 mEq/L caused by a deficit in total body K stores or abnormal movement of K into cells. The most common causes are excess losses from the kidneys or GI tract. Clinical features include muscle weakness and polyuria; cardiac hyperexcitability may occur with severe hypokalemia. Treatment is administration of K and addressing the cause.

- Hypokalemia can be caused by any cause of K loss, such as dietary K restriction or loss of K through the GI tract resulting in a negative K balance.

- Vomiting, diarrhea, or use of diuretics can cause hypokalemia.

- GI losses can be compounded by concomitant renal K losses due to metabolic alkalosis and stimulation of aldosterone.

- Intracellular shift can be caused by stimulation of the sympathetic nervous system, particularly with β2-agonists.

- Drugs: Diuretics are by far the most commonly used drugs that cause hypokalemia. K-wasting diuretics that block Na reabsorption proximal to the distal nephron include thiazides and loop diuretics.

CAUSES OF HYPERKALEMIA

CAUSES

- Ineffective elimination from the body due to resistance, such as with familial hyperkalemia.
- Defective modulators of salt transporters, including the 
- Excess release from cells due to excessive intake or lean muscle mass.
- Excessive release due to death, such as from lethal injection.

- Pseudohyperkalemia is a rise in the amount of potassium that occurs due to excessive leakage of potassium from cells, during or after blood is drawn. It is a laboratory artifact rather than a biological abnormality and can be misleading to caregivers.

- Excessive release from cells due to death, such as from lethal injection.

- Pseudohyperkalemia is typically caused by excessive leakage of potassium from cells, during or after blood is drawn. It is a laboratory artifact rather than a biological abnormality and can be misleading to caregivers.

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ACUTE MANAGEMENT OF HYPERKALEMIA

Lowering K temporarily

Several medical treatments shift potassium from the bloodstream into the cellular compartment, thereby reducing the risk of complications. The effect of these therapies tends to be temporary, but they are often used in conjunction with other therapies to achieve permanent lowering.

- Insulin 10-15 Units can add Glucose
- HCO3
- β-agonists

Severe cases require hemodialysis or hemofiltration, which use the renal replacement methods of dialysis and ultrafiltration to remove excess potassium. These are especially useful if non-obedient patients cannot be controlled safely while temporary measures are instituted or if there is no response to these measures.

- Dialysis solutions can be modified to reduce potassium levels in the dialysate.

- Long-term prevention

- Preventing recurrence of hyperkalemia involves reduction of dietary potassium, removal of an offending medication, and replacement of oral bicarbonate.

- Kayexalate is occasionally used on an ongoing basis to maintain lower serum levels of potassium. Concerns regarding its use are noted in the previous section.
PULSELESS ELECTRICAL ACTIVITY

Causes Pulseless Electrical Activity (PEA)

Pulseless Electrical Activity (PEA)

1. Treat the cause first!

Causes: Remember 5 "H's" and 5 "T's"

- Hypoxia
- Hypothermia
- Hypovolemia
- Hyper/hypokalemia
- Hypocalcemia
- Tension Pneumothorax
- Tachycardia (cardiac)
- Tamponade (cardiac)
- Thrombosis, coronary (ACS)
- Thrombosis, pulmonary (embolism)
- Tablets (drug overdose)

Most common causes:

Algorithm "P-E-A":

- Possible causes: always give 500 cc bolus of fluid since hypovolemia is common cause.
- Epinephrine 1 mg IV every 3-5 minutes.
- Atropine 1 mg IV every 3-5 minutes.
- Consider inotropic support.
- Defibrillation: after rhythm and pulse returns to treat BP.

ARRYHTMIA CASE PRESENTATION

Patient is a 48 year old white male with history of hypertension, diabetes and sleep apnea. Routine induction and intubation for a laparoscopic cholecystectomy.

Thirty minutes into case patient has sudden change in rhythm:
TREATMENT
CARDIOVERT
RHYTHM CONTROL
PRESSURE SUPPORT

DISPOSITION
PCU
CHRONIC VS. ACUTE
RULE OUT MI

Patient is a 39 year old black female with moderate hypertension, diabetes, CVA admitted with intraabdominal sepsis and volume depletion.
Severely acidic (Base excess is Negative 16)
Emergently being rushed to OR
Labs no good (hemolysis)
ARRYTHMIA CASE PRESENTATION

ECG

ARRYTHMIA CASE PRESENTATION

Treatment
Stability for Induction
Monitoring
Disposition

ARRYTHMIA CONCLUSION

The failure to recognize, treat and potentially define an intraoperative arrhythmia can lead to serious and potentially lethal consequences.

Be prepared, think conduction pathway and understand the potential for cardiac instability.
Questions???

Answers???

Doubts???

Comments???