

Ventricular Arrhythmias

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What are arrhythmias?

- Any condition where the heart beats with an irregular rhythm
- Many have electrical conduction abnormalities where conduction doesn't follow the physiologic pathways to depolarize the atria and ventricles
 - Which makes it not a sinus rhythm
- Supraventricular arrhythmias – arrhythmias that originate in the atria
- Ventricular arrhythmias – arrhythmias that originate in the ventricle
- Tachyarrhythmias – abnormal rhythm with rates > 100 beats per min (bpm)
- Bradyarrhythmias – abnormal rhythm rates < 60 bpm

What can ultimately happen if arrhythmias go untreated?

- The potential for harm depends on what type of arrhythmia the patient has
- It can vary from all ends of the spectrum
 - Sinus bradycardia or tachycardia is typically benign and transient
 - Atrial arrhythmias can cause hemodynamic instability and ischemic stroke
 - Ventricular arrhythmias can require defibrillation or even cause death
- Some arrhythmias may be benign and harmless, others can cause long-term damage and even death if left untreated

Are certain patients at higher risk for arrhythmias than others?

- Patients with certain cardiac diseases in their past medical history are at higher risk including:
 - Hypertension, congenital heart disease, heart failure
 - Coronary artery disease is another major risk factor
- Other risk factors include:
 - Hypo-/hyperthyroidism
 - Electrolyte imbalances
 - Obstructive sleep apnea
- Lifestyle risks include:
 - Obesity and smoking

- Caffeine/alcohol intake

What are the most common atrial arrhythmias?

- The most common sustained atrial arrhythmia is atrial fibrillation
- Atrial flutter is more transient in nature
- Other atrial arrhythmias such as sinus tachycardia are common but usually not sustained

What are common overarching principles to keep in mind when treating arrhythmias, regardless of their origin?

- Focus on the most common arrhythmia: atrial fibrillation
- Three treatment principles
 - Stroke prevention
 - 2019 AHA guidelines recommend DOACs over warfarin for treatment
 - Very few patients, outside of those with mechanical heart valves, are on warfarin today
 - Rate control
 - Rhythm control
 - The focus for both rate/rhythm treatment is heart rate control
 - AFFIRM trial found no mortality difference between rate/rhythm control but increased hospitalizations and side effects in patients in the rhythm control treatment arm
 - Most algorithms start with rate control and use rhythm control in select patients

What type of patients would you consider using rhythm control drugs in?

- Typically use in select patients
 - Intolerable symptoms from AF
 - Difficult to control their HR with rate control
 - New-onset AF
 - Rhythm control with cardioversion/ablation has a higher success rate
 - Previous cardioembolic stroke
 - Convert to normal sinus rhythm (NSR) to decrease risk of thrombus formation

Adenosine is the first-line treatment for paroxysmal supraventricular tachycardia (SVT). Should we be using adenosine in the critically ill? Are there pearls for adenosine administration/monitoring?

- Adenosine stops cardiac conduction and can be used in critically ill patients
 - Can break a fast rhythm or slow it down to help diagnose the underlying rhythm which you can then identify and treat
- Adenosine has greater effectiveness in terminating re-entrant pathway arrhythmias
 - It's not as effective in converting atrial fibrillation/atrial flutter
- Most common use is going to be in patients with regular narrow complex SVT
 - QRS less than 120 msec (or less than 3 little boxes on EKG)
- The half-life of adenosine is very short, approximately 5-10 seconds
- Give it via central line or a peripheral line that is closest to the heart
 - Flush with at least 20 mL NS to ensure the drug gets to the heart
- Patients feel horrible, sometimes it's described as a "feeling of impending doom"
 - Counseling the patients about what they may experience can be helpful
 - The half-life is so short that the side effects are transient

Does adenosine have any utilization for ventricular arrhythmias?

- Won't be a therapeutic treatment for ventricular arrhythmias
- Can be used in regular wide complex tachycardia
 - These are typically ventricular tachycardia but could be SVT with aberrancy
 - Adenosine can help differentiate which arrhythmia is occurring

For patients who won't or can't receive adenosine, what is their treatment?

- Try vagal maneuvers prior to giving adenosine
 - Valsalva maneuver or "bearing down" can help slow down SVT
- No absolute contraindications to using in adenosine in patients with SVT
 - Doesn't mean that some rhythms won't worsen after adenosine administration, such as worsening heart block or atrial fibrillation
- Can also attempt cardioversion in these patients

What is the clinical significance of non-sustained ventricular tachycardia or premature ventricular complexes (PVCs)?

- PVCs are ectopic heart beats that originate in the ventricles
- Non-sustained VT (NSVT) are 3+ consecutive PVCs that last for less than 30 seconds
 - This is completely normal and up to 50% of people have PVCs, many are asymptomatic
 - Frequent PVCs (>30/hr) can increase the risk of mortality
- NSVT depends on the underlying presentation of the patient
 - For patients without any CV disease, non-sustained VT likely isn't an accurate predictor of future events
 - In patients with heart failure or CAD, NSVT can be a predictor for future ventricular arrhythmias and increased risk of sudden cardiac death

Do we ever treat PVCs or NSVT?

- For the most part, no therapy is indicated for occasional PVCs
- CAST trial demonstrated that attempting to suppress PVC with sodium channel blocking anti-arrhythmic agents (e.g. flecainide) actually increased mortality
- If the symptoms from PVC are intolerable can use BB or non-DHP CCB
- Also optimize electrolytes to prevent potentiating further arrhythmias
- In patients with extreme PVC burden, could consider doing an ablation

What are the differences between monomorphic and polymorphic ventricular tachycardia (VT)?

- We differentiate monomorphic and polymorphic VT via EKG rhythm strip
 - Monomorphic VT wave forms have similar QRS complexes with each beat
 - Polymorphic VT contains multiple QRS waveform morphology throughout
- Sustained VT, regardless of mono- or polymorphic, have similar symptoms ranging from benign to pulseless hemodynamic collapse
 - This can depend on the rate, patient's underlying LV function, or if the patient has pre-existing heart disease (e.g. HFrEF or atherosclerosis)
- Polymorphic VT occurs more frequently in an acute event
- Monomorphic VT typically occurs due to scar tissue from an old infarct that creates a re-entry pathway

- Both types of VT can progress to ventricular fibrillation (VF)
 - Outcomes are worse with polymorphic VT as they occur more commonly in an acute cardiac event
 - Polymorphic VT that occurs without acute ischemia may be related to QT prolongation
- Prognosis depends on the underlying heart disease and ability to re-vascularize
- Management of VT (both mono- and polymorphic) depends on the patient's symptoms, the acute condition, and the patient's underlying disease
 - Asymptomatic patients with monomorphic VT can be managed with anti-arrhythmic agents:
 - Amiodarone, sotalol, or procainamide
 - Symptomatic patients (with a pulse) may require cardioversion and anti-arrhythmic drugs
 - Pulseless patients will require emergent treatment and defibrillation/CPR per ACLS protocols
 - Long-term therapy typically involves fixing the underlying problem
 - Then a combination of ICD, ablation, and anti-arrhythmic drugs
 - Monomorphic VT can be ablated to a better extent than polymorphic VT because there is an easier target to map out

How and why should we be administering Magnesium for the treatment of Torsades de Pointes?

- Magnesium (Mg) isn't recommended for routine use in all VT or VF
 - It is reserved for the treatment of torsades or polymorphic VT with a prolonged QT
- Magnesium helps to suppress torsades by blocking early depolarizations by decreasing calcium influx
- Administer 1-2g Mg IV push over a few minutes
 - Repeat a bolus dosing if needed
 - Some patients also receive concomitant Mg infusions, especially if previously hypomagnesemic
- Will still give Magnesium if the patient had a normal Mg level
 - We aren't worried about toxicity from magnesium until we get really, really elevated levels

What treatment options are there if Magnesium doesn't terminate Torsades de Pointes?

- If Mg doesn't terminate the torsades and the patient is symptomatic, next step is:
 - Cardiovert if unstable
 - If rhythm is too unstable may have to use cardioversion
 - Defibrillate in patients without a pulse

When should we be worried about QTc prolongation? At what point is the risk of Torsades de Pointes significantly increased due to a prolonged QTc?

- Remember that QT interval is just one piece of the puzzle when thinking about the risk of torsades and QT prolongation
- Take that information and evaluate it as a whole, instead of just looking at one number
- QT prolongation, typically above 500 msec, put patients at an increased risk of Torsades de Pointes
 - But it is also dependent on other factors that are present
- There is also variation in calculating the QTc interval, and it is commonly done by a machine leading to inaccuracies
- Evaluating other risk factors that may be present in a patient may help guide how you change or choose therapy for certain patients
 - Balance the risk of torsades with potential negative effects a patient could have by delaying/avoiding specific treatments
- Don't look just at the QTc, look at all risk factors a patient has for torsades

When will you simply watch the QT interval? Is there anything we can do to help prevent torsades?

- We should always be watching
 - Have those multidisciplinary discussions about patients, discussing all risk factors, and make the ultimate decision on their therapy
- In terms of prevention, be stewards of medications
 - Stop medications without a need
 - Correct electrolytes
 - Use guideline-directed medical therapy
 - Monitor EKGs in patients who have started or continued on therapies
 - Use alternate therapies when appropriate

Do you have a favorite reference/website to see a medication's true risk of QT prolongation and subsequently Torsades de Pointes?

- Crediblemeds.org for everything you want to know about QT prolonging medications
 - Will need to create a free account to access
 - Good medication information, medication-specific risk of torsades, and patient risk factors associated with QT prolongation and torsades
 - Also have a smart phone application you can download

Is management the same for patients if they have either ventricular tachycardia or ventricular fibrillation?

- VT can range in presentations from mild symptoms to pulseless
- VF is a completely unorganized, non-perfusible rhythm
 - Management will be similar to pulseless VT

What are the two primary pharmacologic agents used for pulseless VT/VF in ACLS? Is our dosing the same as when it's used outside of ACLS?

- 2015 ACLS guidelines recommend the use of epinephrine and amiodarone
- The 2018 guideline update also recommend lidocaine as a first-line anti-arrhythmic medication
- Epinephrine – can be used in shockable/non-shockable rhythms
 - Give 1mg IVP every 3-5 minutes to help improve ROSC
- Amiodarone – can be used in VT/VF arrest that is refractory to defibrillation
 - First dose: 300mg IVP
 - Second dose: 150mg IVP
 - Patients who have a pulse receive 150mg amiodarone as an IVPB bolus administered over 10 minutes
- Lidocaine – first-line agent used in VT/VF arrest that is refractory to defibrillation
 - Dose: 1-1.5 mg/kg

Is there better evidence for defibrillation or pharmacologic agents when treating pulseless VT/VF?

- Two therapies most associated with favorable long-term outcomes:
 - High-quality CPR
 - Defibrillation of a shockable rhythm as early as possible

- Drug therapies may help to improve ROSC, but haven't been linked to improved long-term survival with good neurologic recovery
 - ROC-ALPS looked at out-of-hospital cardiac arrest and found anti-arrhythmic drugs (amiodarone/lidocaine) improved survival to the hospital but no difference in overall survival or favorable neurologic outcomes compared to placebo

Amiodarone

- Wide array of indications in its use for ventricular arrhythmias
 - Use in refractory pulseless VT/VF, which is defined as >3 shocks
 - Less data on the use for switching between amiodarone/lidocaine
 - Use in monomorphic or polymorphic VT
 - Secondary prevention of ventricular arrhythmias
- Important note: the first sentence in the ventricular arrhythmia guidelines states “no anti-arrhythmic drugs have been shown to improve survival as therapy for primary/secondary prevention of ventricular arrhythmias.”
 - No data that amiodarone improves survival for primary or secondary prevention of ventricular arrhythmias
 - Anti-arrhythmic drugs are typically part of a multi-modal treatment approach that includes devices (e.g. ICDs) and electrical therapy (e.g. ablation)
 - Drugs can help control symptoms and decrease events
 - But these can also cause arrhythmias
- Amiodarone has a class 1B recommendation for use to suppress recurrent ventricular arrhythmias in combination with ICDs and beta blockers
 - Monomorphic VT, Polymorphic VT, and VF
- Before classifying someone as an amiodarone “non-responder” ensure they received enough so they are at a pseudo-steady state
 - 8-10g total amiodarone load which will take more than just IV therapy
- Questions to ask:
 - Does amiodarone help break the rhythm?
 - Does amiodarone increase the arrhythmia burden?

Lidocaine

- Typical starting rate is 1mg/min (DI references recommend 1-4 mg/min)
 - This is typically after administering a bolus dose
- If patients are having recurrent arrhythmias, will re-bolus at a smaller dose (0.25-0.5 mg/kg) or increase the rate by 0.5-1 mg/min
- Try to monitor lidocaine levels every day, especially in patients with renal or hepatic dysfunction because lidocaine can accumulate with extended infusions
 - Lidocaine has a narrow therapeutic range and toxicities that should be closely monitored
- Typical CNS side effects that are first seen include numbness/tingling, dizziness, blurred vision/speech which can worsen into AMS and seizures
 - Patients can also have hemodynamic changes (low BP/HR)
- When you can't get lidocaine levels back in a timely fashion, be really cognizant of monitoring for side effects as well as renal/hepatic function
 - If these adverse events are present in patients, typically treat it as lidocaine toxicity until proven otherwise
- Mexiletine is the oral version of lidocaine
 - Patients who respond to lidocaine are often transitioned from IV lidocaine to PO mexiletine
 - Administer mexiletine as you stop the IV lidocaine infusion
- May also see mexiletine used in patients with genetic conditions such as long QT syndrome

If patients respond to the lidocaine/amiodarone initial IV bolus, should we always start an IV infusion afterward?

- Minimal to no evidence looking at continuing amiodarone/lidocaine post-ROSC in pulseless VT/VF
 - Commonly in practice, an infusion is started to help prevent recurrent events
 - Also allows the team to create the plan moving forward (devices, procedure, etc)
- If the arrhythmia was triggered by a “reversible” cause (e.g. hypokalemia), will likely not continue

Sotalol

- IV sotalol was re-introduced to the market a few years ago
- Sotalol can be used in refractory ventricular arrhythmias
 - Sustained monomorphic VT to help convert and also suppress refractory disease (Same 1B recommendation as amiodarone) as long-term oral therapy
- IV dosing differs slightly from oral dosing (based on bioavailability) and may be used in acute patients without enteral access or poor oral absorption
- Typically, don't use sotalol in patients with EF < 20-25% due to its negative inotropic effects
 - If the EF between 25-40% it's a gray area
 - Usually try amiodarone in these patients, because there is less risk in patients with heart failure

Sodium Channel Blockers

- Don't routinely use these agents, excluding lidocaine/mexiletine
- Two agents that may get used:
- Quinidine – Brugada syndrome
 - Can help suppress arrhythmias
- Procainamide – Sustained monomorphic VT
 - More of an acute therapy to terminate the rhythm, not a long-term suppression agent
- Avoid using both of these agents in patients with prolonged QT and use caution in patients with CHF

Isoproterenol

- β_1/β_2 agonist which increases heart rate and causes peripheral vasodilation
- Primarily used during bradyarrhythmias such as complete heart block
 - Typically used for heart block unresponsive to atropine or pacing
 - Also used in cases of recurrent torsades to help increase the HR and suppress the arrhythmia
- Start at a low dose and titrate to desired effect
 - Fairly short half-life of ~5 minutes
 - Can titrate fairly quickly

Rate Control (Beta Blockers/Calcium Channel Blockers)

- Will use beta blockers (BB) more than calcium channel blockers (CCB) for the treatment of ventricular arrhythmias
 - BB are effective in treating ventricular arrhythmias as well as for secondary prevention
 - Decrease sympathetic system activation and electrical excitability which can lead to ventricular arrhythmias
 - Used for the treatment of PVCs, VT, VF and reduce sudden cardiac death in CHF patients post-MI
- Improved outcomes when using BB and anti-arrhythmics, compared to anti-arrhythmic monotherapy
- IV propranolol can be used to suppress VT during recurrent episodes or VT storm
- Dose to symptom control, no target dose for the treatment of ventricular arrhythmias

- Calcium channel blockers have a minimal role in the treatment of ventricular arrhythmias due to their negative inotropic effects
 - CCB have a Class III recommendation (Harm) when used in patients with wide complex tachycardia of *unknown origin*
- In patients without structural heart disease, verapamil can be used to help terminate idiopathic VT

Does our management change for long-term control or acute management for patients with a history of ventricular arrhythmias?

- Therapies discussed above are technically secondary prevention strategies
 - Treat the acute arrhythmia and attempt to decrease the chance it happens again with treatment
- There is good evidence showing the effectiveness of ICD compared to anti-arrhythmic medications for secondary prevention of VT/VF
 - Many patients will receive a device in combination with an anti-arrhythmic agent
- Main difference in patients with a history of ventricular arrhythmias would be attempting second- or third-line agents that typically aren't used in the majority of patients

Do ventricular arrhythmias carry the same thrombotic risk as atrial arrhythmias?

- Overall, they do not because the clot risk with atrial arrhythmias is due to the venous stasis occurring in the left atrial appendage
 - Additionally, atrial fibrillation/atrial flutter, are long-term conditions allowing time for clots to form
 - Ventricular arrhythmias are typically more acute
- Patients with heart failure and hypokinesia are at risk for LV thrombus, but related to the ventricular arrhythmias
- Unsure if we should anticoagulate patients post-VT ablation, there is no consensus
 - May depend on other risk factors for clots

Is there anything we can do while these patients are in the ICU to prevent ventricular arrhythmias?

- Yes, there are multiple things
- Manage risk factors that can potentiate ventricular arrhythmias
 - Medication-induced, infection, or electrolyte abnormalities
- Ensure they are on their appropriate medications
 - Resume guideline-directed medical therapy as soon as it is safe

For those of us who want to improve our knowledge and interpretation of EKGs, where would you recommend, we turn to?

- Takes time and effort to improve the ability to read them
- Try EKG textbooks that contain EKG cases/answers to help learn
- Pay attention on rounds and look at real EKGs to interpret
 - Remember to interpret the EKG with the patient's clinical condition

When you think about pharmacologic management of VT/VF, do you have a stepwise approach or algorithm in your head you use for most cases?

- Evaluate and interpret a rhythm
- Assess patient's symptoms and risk factors to make treatment decisions
- VT/VF happen acutely and may not have been a part of the initial treatment process
 - But can help monitor therapy and modify when appropriate

- Evaluate drugs, assess for contraindications, monitor for adverse effects, and therapeutic drug monitoring when able
 - Know whether the patient has structural heart disease, will guide therapy
- Pulseless VT/VF – ACLS

What are the most important principles to keep in mind when treating ventricular arrhythmias?

- Due to lack of efficacy and evidence, anti-arrhythmic medication monotherapy treatment has been replaced with a combination of devices (ICD +/- ablation) AND anti-arrhythmic treatment
- Not too many medications still used for ventricular arrhythmias
 - Know a few clinical pearls with each medication
- Understand the QTc and medications that prolong the QT
 - Other patient risk factors that put patients at risk for torsades is important
 - Evaluate the whole picture, not just the number