Overview of approach to ventricular tachycardia

EKG Features of VT
- If patient is hemodynamically stable, take a deep breath and get a 12-lead EKG!
- Leads of choice to analyze: V1-2 and 6 for bundle branch morphology; I and F for axis
- EKG findings suggestive of VT:
  1) AV dissociation/VA block with Wenckebach: “cherchez le p” and try to march them out using the “index card” approach
  2) Fusion/capture beats
  3) Concordance V1-6: can also be seen in WPW however
  4) V1: slurred downstroke; RBBB morphology Rs'; monophasic or biphasic
  5) V6: LBBB morphology monophasic
  6) NW axis: negative I, aVF
  7) QRS > 0.14

Approach to differential diagnosis of VT

Step 1: Look at EKG morphology
Monomorphic or polymorphic?
If monomorphic, try to identify location of VT focus:
- If VT has an LBBB pattern (i.e. positive in I, V6, negative in V1), it is likely coming from the RIGHT ventricle (particularly if patient is young and/or ostensible has no structural heart disease)
- If VT has an RBBB pattern (i.e. isoelectric in I, positive in V1), it is likely coming from the LEFT ventricle
- If VT has a superior axis (i.e. positive in I, negative in F), it is likely coming from the region of the ventricular apex or diaphragmatic region
- If VT has an inferior axis (i.e. negative in I, positive in F), it is likely coming from the region of the outflow tract
- To help remember the above, just think OPPOSITE bundle, OPPOSITE axis EKG morphology from anatomic source

Step 2: Is the heart structurally normal?
Monomorphic VT with
- Structurally abnormal heart:
  - **CAD**: Ischemia induced v. Scar induced
  - **Cardiomyopathy** (dilated CM, hypertrophic CM, valvular CM)
  - **Infiltrative disease** (amyloidosis, sarcoidosis)
  - **Valvular disease**: mitral valve prolapse association
  - **Bundle branch reentry**: associated with DCM, valve surgery and myotonic dystrophy
  - **Arrhythmogenic right ventricular dysplasia** (ARVD)
    - Seen primarily in young patients; marked replacement of right ventricular myocardium by fibrofatty tissue with development of RV cardiomyopathy
    - ECG during VT: LBBB morphology with inferior (pulmonary infundibular source) or superior axis (apical source)
    - ECG during NSR: T wave inversions beyond V1 (50%), incomplete RBBB (18%), epsilon waves (postexcitation activation of right ventricular fibers) are seen in 30% cases: defined as potential in V1-3 (can look like pseudo R') that is >25 msec beyond end of QRS in V6 (or QRS > 110 ms V1-V3)
  - **DX**: MRI, right ventriculography (infundibular aneurysm, hypertrophic trabeculae, TVP)
  - **TX**: ICD, antiarrhythmics, RFA (usually adjunctive), surgical isolation, cardiac transplant

Structurally normal heart:
- **RV outflow tract tachycardia** (adenosine-sensitive VT)
  - Two types: repetitive monomorphic VT (RMVT) and exercise-induced VT
  - LBBB morphology with inferior axis (usually originates from RVOT)
• Mechanism: adenosine sensitivity with catecholamine-mediated triggered activity and DADs (cAMP stimulation of oscillatory calcium channel release, giving rise to DAD (due to I_t))
• TX: RFA (definitive), beta blockers, CCB, class I agents

**NOTE:** variants include LVOT source (10%) with either RBBB pattern in V1 or LBBB with early precordial transition in V2 (aortic sinus cusp source; Ouyang et al., JACC 2002)

• Need to rule out ARVD

**Idiopathic LV tachycardia** (verapamil-sensitive fascicular VT)
• Usually RBBB morphology with relatively narrow QRS and superior axis
• Mechanism: reentry; left posterior fascicular origin (inferoposterior origin with short retrograde VH interval), EP study will reveal location of retrograde Purkinje potentials (also known as mid or late diastolic potentials) that reflect depolarization of the slow conduction portion of reentrant circuit
• TX: RFA (definitive), CCB

**Automatic (propanolol sensitive) VT** (see Familial polymorphic VT syndrome below)
• Younger patients, precipitated by exercise
• May be polymorphic or monomorphic

**Inflammatory LV microaneurysms:** RBBB morphology, seen in cases of myocarditis with generally good prognosis

**Polymorphic VT with:**

**Normal QT**
• MYOCARDIAL ISCHEMIA (MOST COMMON CAUSE)
• Brugada syndrome:
  • Association of VT/VF with characteristic ECG pattern: RBBB morphology with ST elevation in V1-3 (unrelated to ischemia, electrolyte abnormalities or structural heart disease)
  • Mechanism: some forms linked to SCN5A gene mutation (also in LQT3 form of long QT); impaired Na channel function leads to unopposed I_{to} current during phase 1 of the action potential leading to early repolarization (this occurs primarily in the epicardium leading to a transmural voltage gradient that is manifest by J point elevation) \( \rightarrow \) dispersion of repolarization \( \rightarrow \) phase 2 reentry \( \rightarrow \) extrasystole \( \rightarrow \) circus movement reentry – VT/VF
• TX: ICDs primarily given high mortality rate

**Familial polymorphic VT syndrome** (also known as catecholaminergic VT) – now thought to be a major subset of automatic (propranolol sensitive) VT
• Classically presents with stress-induced bidirectional VT; look for strong FH of SCD
• Autosomal dominant with association with RyR2 mutation (ryanodine receptor/calcium release channel); RyR sits on sarcoplasmic reticulum (SR) membrane and is activated by Ca influx through L-type calcium channels; RyR in turn activates calcium-induced calcium release channels of the SR

**Long QT**
• Acquired long QT:
  • Class IA, class IC, and class III antiarrhythmic agents; Haldol, erythromycin, Seldane/Cisapride (pulled off market), Reglan, TCAs
  • Mutations in HERG and MiRP1 genes have been associated with drug-induced torsades

**Congenital long QT**
• LQT1-6 (linked to variety of sodium channel and potassium channel mutations- KVLQT1, minK, HERG, MiRP1, SCN5A)
• See Torsades de Pointes section for details
Acute treatment of ventricular tachycardia

Unstable VT/VF arrest

- See ACLS protocol and Resuscitation (in this curriculum) section for details
- DC cardioversion (UN-synchronized): monophasic shocks 200J – 300J – 360J (biphasic shocks require less energy and have been shown to result in higher rates of successful defibrillation with 1st shock)
- Epinephrine 1 mg or Vasopressin 40U IV
- Amiodarone 300 mg bolus (can repeat 150 mg) IV; can then infuse at 1 mg/min initially
- Its use has been supported by the ARREST trial (amiodarone v. placebo in out-of-hospital VF/pulseless VT arrests) and the ALIVE trial (amiodarone v. lidocaine in VF/pulseless VT arrests)
- Lidocaine 1.5 mg/kg bolus (can repeat 0.5-0.75 mg/kg) IV; can then infuse at 2-4 mg/h
  - Although lidocaine is generally safe, its effectiveness in terminating VT/VF has been called into question by various studies comparing it to placebo and other antiarrhythmics
- Procainamide 30 mg/min (17 mg/kg max) load IV → stop if hypotension, QRS prolonged > 50%, or QT prolongation → can then infuse at 1-6 mg/min initially (check NAPA/procainamide levels after 24hs if renal failure or if infusion rate>= 2 mg/min)
  - While procainamide has been shown to be effective in terminating stable VT, its use is generally NOT recommended during arrests given the long time required for its infusion and its hypotensive effects
- Magnesium 1-2 mg IV push should be given if torsade de pointes or hypomagnesemic states are suspected
- REMEMBER TO CONTINUE TO ADMINISTER DEFIBRILLATION SHOCKS WITH EACH MEDICATION ADMINISTRATION OR AFTER 1 MINUTE OF CPR WHILE STILL IN VT/VF

Stable VT

- DC cardioversion (synchronized)
- Pharmacological therapy
  - If low EF, consider:
    - Amiodarone 150 mg/min bolus IV over 10 min then 1 mg/min x 6hs then 0.5 mg/min x 18hs initially
    - Lidocaine 1 mg/kg load IV then 1-4 mg/min maintenance
  - If normal EF consider
    - Procainamide 100 mg IV q10 min or infuse at 20 mg/min to total 17 mg/kg (renal elim)
    - Amiodarone
    - Lidocaine
  - Other pharmacological agents can be considered which should be tailored to specific etiologies of the arrhythmia (e.g. verapamil in idiopathic left ventricular tachycardia)

Pearls

- Immediate post-MI ectopy (within 48ds) such as PVCs or asymptomatic NSVT does NOT need to be treated
- Post MI AIVR (rate 50-120) is NOT treated because 1) it is usually transient, benign and a sign of reperfusion and 2) are at times an escape rhythm due to intrinsic pacemaker failure
Chronic treatment of sustained VT: ICD Therapy

Key questions to answer when considering ICD therapy
1) Did VT/VF occur in the setting of ACUTE ischemia? (answer with cardiac enzymes, cardiac catheterization)
2) Does the patient have significant coronary disease? (answer with stress testing, cath)
3) Does the patient have a structurally normal heart? (answer with echo +/- cardiac MRI)
4) Does the patient have a family history of sudden cardiac death?

Primary prevention of SCD
If low EF (i.e. < 30-40%) AND ischemic cardiomyopathy
• If patient has NSVT, perform EP study; if inducible, place ICD → this has been supported by the MADIT-I (EF 35% cutoff) and MUSTT (EF 40% cut off) trials
• However, MADIT-II data (EF 30% cut off) currently supports ICD placement regardless of presence of ectopy

If low EF AND nonischemic cardiomyopathy
• There is poor data at this point to guide the use of ICD therapy
• Patients with NICM, low EF and history of syncope are at high risk of SCD and ICDs are often indicated
• It is unclear what to do with patients with NICM, low EF and NSVT
  • EP studies are of limited utility in these situations (it would allow detection of bundle branch reentry which is an ablable rhythm)
  • GESICA and subgroup analysis of CHF STAT trials would support the use of amiodarone although this is controversial
  • Several ongoing studies including DEFINITE and SCD-HeFT trial should answer some of these questions

If normal EF and apparently structurally normal heart
• Consider ICD and/or pharmacological therapy if etiology of SCD is understood
• Examples:
  • ARVD
  • Brugada syndrome: consider ICD therapy given high risk of SCD
  • Long QT syndrome

Secondary prevention of SCD
• The use of ICDs have been supported by the AVID study (to a greater extent than the CIDS and CASH studies) in patients with one of the following:
  • History of resuscitation from VF/pulseless VT arrest
  • Sustained VT with EF < 40% resulting in hemodynamic compromise (i.e. near syncope, CHF and angina
  • In the above cases, would need to rule out acute myocardial infarction as cause of VF/VT episode before considering ICD implantation as coronary revascularization would be treatment of choice in such cases

REFERENCES
ALIVE Dorian et al., Amiodarone as compared with lidocaine for shock-resistant ventricular defibrillation. NEJM 2002; 346: 884-90.


Klein et al. New primary prevention trials of sudden cardiac death in patients with left ventricular dysfunction: SCD-HEFT and MADIT-II. *Am J Cardiol* 199; 83: 19D-97D.


