- R-on-T phenomenon.
- Chest pain, SOB, palpitations, presyncope, altered mental status.

Overview

- VTach is caused by high-frequency electrical depolarization from a ventricular myocardial focus and is characterized by a widened QRS (>0.12 sec), high rate (>120 bpm) and variable morphology (MVT or PVT) and duration (sustained vs. nonsustained).
- Atrioventricular dissociation may be present, where p waves may be seen with or without capture/fusion beats. This implies VTach rather than SVT with aberrant conduction.
- MVT has a single QRS morphology and can evolve into PVT. Often reentrant etiology post-MI.
- Torsade de pointes: Atypical PVT with beat-to-beat variation, prolonged QT, changing/twisting QRS axis around baseline.
- VFIB: Nonperfusing broad complexes (fast, chaotic, irregular, and disorganized).
- Ventricular ectopic beats can sometimes precede VTach.

Etiology

- Structural and ischemic heart disease (MI, CAD, CHF, valvular disease, cardiomyopathies, myocarditis).
- VEs are provoked by dental procedures and anal stretch, particularly in combination with halothane, raised CO_2, and light anesthesia.
- TdP: Severe nonuniform delay in repolarization (QT prolongation). Familial, idiopathic, or acquired secondary to hypokalemia, hypocalcemia, hypomagnesemia.
- QT-prolonging drugs include class 1 and 3 antiarrhythmics, antihistamines, TCAs, lithium, antipsychotics, certain analgesics, ondansetron, and droperidol (see http://www.sads.org.uk/drugs-to-avoid/).
- Short-QT syndrome
- MVT/PVT secondary to excessive endogenous or exogenous catecholamines (stress, exercise, cocaine, etc.).
- Brugada syndrome: RBB-like conduction and ST-segment elevation in precordial leads without prolonged QT or structural heart disease (heritable).
- VFIB: Recent MI, ischemic heart disease, hypokalemia/hyperkalemia, excessive catecholamine levels (endogenous or exogenous), myocardial irritation (e.g., from CVC guidewire or mechanical ventilation).
- Hypoxia, hypothermia, hypercarbia, hypokalemia, hypomagnesemia, acidosis, thromboembolism, tamponade, tension pneumothorax.

Usual Treatment

- Assess vital signs (ABCs).
- Depends on stability of patient. Unstable (systolic BP <90 mm Hg, HR >150 bpm, heart failure, evidence of myocardial ischemia) with pulse then synchronized DC cardioversion. Pulseless VTach equals cardiac arrest, so ALS/ ACLS protocols are needed (CPR, DC cardioversion, epinephrine/vasopressin).
- VFIB is not associated with palpable cardiac output and should be treated with CPR or ALS/ACLS protocols.
- In either VFIB or pulseless VTach in the cardiac cath lab or immediately after cardiac surgery, three consecutive "stacked" shocks may be used.
- Hemodynamically stable MVT/PVT: Amiodarone 300 mg IV over 1 h via central access (or large-bore peripheral access; risk of extravasation) followed by 900 mg over 24 h. Alternative drugs include lidocaine (100 mg bolus, 4 mg/min for 30 min, 2 mg/min for 2 h, 1 mg/min for 4 h) or sotalol 100 mg/procainamide 100 mg. Consider adenosine if there has been earlier SVT with aberrant conduction. Electrical cardioversion as above.
- Correct electrolyte and acid-base disturbances (including magnesium), hypoxia, hypovolemia, hypothermia, and hypercarbia. Look for and treat tension pneumothorax, cardiac tamponade, and thromboembolic causes.
- TdP $MgSO_4$ 1–2 g over 1–2 min, replete K+, consider atropine, isoprenol, asynchronous DC countershocks (avoid epinephrine as it may precipitate VFIB).
- If all fails, then cardiology for overdrive pacing.
- Review medications and remove possible precipitants.
- Chronic VTach: ICD, RF ablation of aberrant pathways, regular antidysrhythmic medications. Stellate ganglion blockade has been used in long-QT syndrome.
- VEs may be treated with beta blockade. If slow, they could be ventricular escape beats calling for anticholinergics (e.g., atropine/glycopyrrolate) or >30 sec (VTach).

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Ischemia, MI, decreased cerebral perfusion, decreased cardiac output	Angina, palpitations, anxiety, lightheadedness, syncope	Pallor, diaphoresis, heart murmur, tachycardia, JVD, cannon a waves, displaced PMI, S_3 gallop	12-lead ECG, TTE, TEE, Holter monitor, cardiac CT and cardiac MRI, cardiac cath (right or left heart), cardiac enzymes
RESP	Increased pulm venous pressure, pulm edema secondary to HF	Dyspnea, tachypnea, sleep apnea	Wheezing, course breath sounds, crackles	CXR, CT chest, PFTs, ABG

Key References: Thompson A, Balser JR: Perioperative cardiac arrhythmias, *Br J Anaesth* 93(1):86–94, 2004; Roberts-Thompson K, Lau D, Sanders P: The diagnosis and management of ventricular arrhythmias, *Nat Rev Cardiol* 8:311–321, 2011.

Perioperative Implications

Preinduction/Induction/Maintenance

- Pt history of VFIB/VTach, CAD, CHF. Is cardiac disease optimized medically?
- Congenital/acquired long-QT syndrome; avoid and/or stop causative drugs.
- Minimize sympathetic stress response; adequate depth of anesthesia/analgesia; avoid sympathomimetics/sensitizers; be careful with catecholaminergics.
- Ask: Is there a problem with the anesthetic? Modify depth of anesthesia, look for drug interactions or error.
- Ask: Is there a problem with the surgery? Avoid anal stretch, ocular traction, peritoneal traction. Care with Moffats solution in ENT surgery. Air or fat embolism? Unexpected blood loss? Mediastinal manipulation?
- Ensure that acid-base, lytes, hypoxia/hypercarbia are assessed and treated

Monitoring

- Routine monitoring including ST-segment trending and recording.
- Arterial +/– cardiac output monitoring for pts with a history of cardiac disease, history of VTach, or undergoing high-risk procedures.

General Anesthesia

- Unclear evidence on usage of volatile anesthetic agents in long-QT syndrome; both prolongation and shortening have been reported. Consider use of TIVA in at-risk population. Halothane in particular is implicated in ventricular dysrhythmias.
- Care with CVC placement: Check position if there are new ventricular arrhythmias.

Regional Anesthesia

- Meticulous avoidance of intravascular injection
- Avoid hypoperfusion secondary to vasodilatation

Postoperative Period

- Adequate analgesia
- Further workup including echocardiography, 12-lead ECG, and cardiology consult.
- Consider increased care environment and ongoing antiarrhythmic therapy.

Ventricular Tachycardia

Arvind Rajagopal | Kenneth J. Tuman

Risk

- Structural heart disease (most commonly a chronic phase of MI); predictor of sudden cardiac death after MI.
- Most common cause of mortality with CHF.
- Cardiomyopathies, both hypertrophic and dilated, are assoc with VTach.
- Seen in genetic syndromes such as long QT syndrome, Brugada syndrome, and arrhythmogenic right ventricular dysplasia.

Perioperative Risks

- Endogenous or exogenous catecholamines trigger VTach in susceptible pts.
- Central venous, pulm artery cath and intubation can trigger VTach.
- Hyperventilation may decrease serum K^+.
- Precipitation of polymorphic VTach with agents that alter QT interval.

Worry About

- Decreased vital organ perfusion related to low cardiac output
- Possible effect of antiarrhythmics on cardiac and pulm function
- Periop ventricular dysfunction and/or ischemia
- Progression of VTach to VFIB
- Reduction of left ventricular function due to IV antiarrhythmic

Overview

- Defined as 3 or more consecutive ventricular beats (usually at a rate >100 bpm).
- Sustained VTach persists for >30 sec or requires an intervention for termination.
- VTach storm is 3 or more separate episodes of sustained VTach within 24 h requiring intervention.
- Nonsustained VTach is ≤6 consecutive beats terminating spontaneously within 30 sec.
- Possible signs of VTach include a wide QRS (>140 ms), presence of fusion beat, AV dissociation, and LBBB morphology.
- Must rule out SVT with aberrant conduction or preexisting bundle branch block.
- Torsades de pointes refers to VTach characterized by polymorphic QRS complexes that undulate in a regular fashion about baseline. Often associated with prolonged QT interval.

Etiology

- CAD: Acute myocardial ischemia or MI or old MI with left ventricular scar or aneurysm
- Cardiomyopathies, especially with ventricular dilation/enlargement
- Myocarditis
- Mechanical irritation (cath)
- Metabolic (hypokalemia, hypomagnesemia)
- Hypertrophic cardiomyopathy or mitral valve prolapse may present with VTach.
- Acquired polymorphic VTach (torsades) may result from electrolyte imbalances (K^+, Mg^{2+}) or drugs that prolong repolarization (phenothiazines, tricyclic antidepressants, class Ia antiarrhythmics, erythromycin, pentamidine, terfenadine, astemizole).
- Congenital QT prolongation may be assoc with left-sided cardiac sympathetic dominance.
- Rare association with right radical neck dissection.

Usual Treatment

- Removal or manipulation of intracardiac cath if pt hemodynamically stable.
- Chronic PO therapy: Ia: quinidine, procainamide, disopyramide; Ib: mexiletine, tocainide; Ic: propafenone; II: beta-blockers; III: amiodarone, sotalol.
- IV therapy includes amiodarone, procainamide, phenytoin, lidocaine, and bretylium (less commonly quinidine) as well as Mg^{2+} and/or K^+ when necessary. Amiodarone is superior to other agents.
- Digoxin antibodies for digitalis-induced VTach.
- Class I antiarrhythmics are generally contraindicated in presence of polymorphic VTach (torsades de pointes).
- Electrical cardioversion for VTach with hemodynamic instability.
- Nonpharmacologic management includes ablative techniques, myocardial revascularization, implantable cardioverter-defibrillators (recommended for recurrent VTach and structural heart disease with poor ventricular function), and left ventricular assist devices.
- IABP may be used to improve myocardial perfusion and hemodynamics.
- Treatment of torsades includes withdrawal of offending agent, correction of electrolyte abn (K^+, Mg^{2+}), and/or electrical defibrillation to terminate episode. Accelerating HR with isoproterenol or cardiac pacing may terminate rhythm. Empirical Mg^{2+} treatment may be lifesaving.
- Treatment of congenital QT prolongation, including beta-blockade to blunt sympathetic activity, Mg^{2+}, and/or left cervicothoracic sympathectomy.
- Treatment of VTach storm has involved sympathetic blockade with a thoracic epidural or a stellate ganglion block.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Myocardial ischemia Hypotension Cardiac arrest	Angina/anginal equivalent (syncope, SOB, palpitations, and exercise intolerance) CHF	Cardiomegaly, JVD Cannon A waves; S_3, S_4	ECG, CXR Electrophysiologic studies Ambulatory ECG
RESP	Pulm edema Amiodarone effects (fibrosis)	SOB	Rales (wet or dry)	CXR, PFTs (A-a)O_2 gradient
CNS	Syncope	Dizziness or LOC		

Key References: Amar D: Strategies for perioperative arrhythmias. *Best Pract Res Clin Anaesthesiol* 18(4):565–577, 2004; Mittnacht AJ, Dukkipati S, Mahajan A: Ventricular tachycardia ablation: a comprehensive review for anesthesiologists. *Anesth Analg* 120(4):737–748, 2015.

Perioperative Implications

Preoperative Preparation

- Ascertain etiology of VTach and associated problems.
- Evaluate for Hx of palpitations, SOB, VTach, dizziness, syncope, chest pain.
- Evaluate ECG for morphology of PVCs, QT interval, underlying BBB (important for Dx and therapy of wide complex tachycardia).
- Review electrophysiologic studies to determine optimal treatment of VTach.
- Assess K^+ and Mg^{2+} levels and digoxin level if indicated.
- Pulm and thyroid function tests may be indicated for chronic amiodarone therapy.
- Continue PO antiarrhythmic therapy.
- Have defibrillator immediately available (nearby) whenever inserting central venous cath.
- May need to have AICD deactivated for surgery to prevent firing with electrocautery use.

Monitoring

- ECG for ischemia or QT prolongation.
- Consider invasive hemodynamic monitor if suspicion of serious concomitant cardiac disease and major anesthetic/surgical intervention.

Anesthetic Considerations for VTach Ablation

- Typically occurs in non–operating room settings with limited support.
- The type of anesthetic may impact ability to induce VTach, especially catecholamine sensitive VTach. Sedation is preferred for shorter procedures.
- Paralysis may need to be avoided for phrenic nerve monitoring during procedure.
- Prolonged complex ablation procedures in pts with structural heart disease often are associated with significant volume expansion, electrolyte disturbances, lactate accumulation, and acute exacerbation of heart failure.

Induction/Maintenance

- Avoid myocardial ischemia (maintain O_2 supply and minimize O_2 demand).
- Minimize surgical stimulus response and subsequent catecholamine release.
- Avoid sympathomimetics, which may aggravate ventricular dysrhythmias.
- Avoid hypokalemia and excessive hyperventilation.

Postoperative Period

- Consider continuous arrhythmia monitoring.
- Continue parenteral antiarrhythmics until able to resume PO.
- Treat Mg^{2+} and K^+ deficits (common postop, especially after major surgical procedures).

Vitamin B_{12}/Folate Deficiency

Sharmil S. Gohil | Onyi Onuoha

Risk

- 5–10% of adults over the age of 65 have vitamin B_{12} or folate deficiency.
- Vitamin B_{12} (cobalamin) deficiency is associated with a strict vegan diet, pernicious anemia, gastrectomy procedures, exposure to nitrous oxide, HIV infection, *H. pylori* infection, certain medications, and ileal resections.
- Folate deficiency is associated with chronic alcoholism and malnutrition.

Perioperative Risks

- Intraop:
  - Increased risk of vitamin B_{12} deficiency after the exposure to nitrous oxide anesthesia due to the irreversible inhibition of vitamin B_{12} activity.
  - Homocysteine levels can be elevated after the use of nitrous oxide. The risk of coronary artery and cerebrovascular complications are increased in patients with high total plasma homocysteine levels.
- Postop:
  - Increased risk of postop MI.
  - Risk of neurologic symptoms including peripheral neuropathy, paresthesias, and subacute combined degeneration of spinal cord following nitrous oxide anesthesia.

Worry About

- Limited oxygen carrying capacity due to megaloblastic anemia caused by vitamin B_{12} and folate deficiency.