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

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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.

- Delayed onset of hematologic and neurologic abnormalities seen after nitrous oxide exposure; several wk may elapse before symptoms develop.

Overview

- Vitamin B_{12} and folate have interdependent and essential roles in DNA synthesis.
- Vitamin B_{12} is needed for
  - Synthesis of methionine from homocysteine via methionine synthase.
  - Conversion of methylmalonyl coenzyme A to succinyl coenzyme A via methylmalonyl-CoA mutase.
  - Development and myelination of the CNS and its maintenance.
- Folate has multiple metabolic roles including purine synthesis and amino acid metabolism.
- Deficiencies of vitamin B_{12} and folate lead to increased serum homocysteine levels, which is associated with cardiovascular disease.

Etiology

- Vitamin B_{12} deficiency is normally associated with inadequate absorption from the GI tract, as seen with
  - Pernicious anemia due to antibodies to gastric cells and the lack of intrinsic factor.
  - Gastrectomy and gastritis causing decreased gastric acid and the inability to liberate cobalamin from food.
  - Intestinal disorders and resections leading to malabsorption.
  - Medications such as proton pump inhibitors and H_2 receptor antagonists, which decrease gastric acid secretion and lead to an inability to liberate cobalamin from food.
- Folate deficiency is commonly associated with poor nutrition, alcoholism, goat's milk, and medications such as methotrexate and phenytoin.

Usual Treatment

- Daily oral supplements of folate and/or weekly IM injections of vitamin B_{12}. Vitamin B_{12} can also be given orally, sublingually, or via a nasal spray/gel; however, due to erratic absorption, these therapies should be considered after levels have normalized with parental vitamin B_{12} first.
- Folate treatment alone in individuals who are vitamin B_{12} deficient may produce partial hematologic remission but can result in irreversible neurologic symptoms. Therefore, if emergent therapy is indicated without a diagnosis, both folate and vitamin B_{12} should be supplemented simultaneously.
- Deficiencies associated with nitrous oxide exposure have been successfully treated with IM injections of vitamin B_{12}, IV administration of folinic acid, and oral methionine.

Assessment Points

System	Effect	Test
HEENT	Glossitis and painful tongue (infrequent)	
CV	Angina and DOE secondary to anemia Coronary artery disease secondary to increased homocysteine levels	ECG
GI	Anorexia, diarrhea	Schilling test for malabsorption of vitamin B_{12}
HEME	Megaloblastic anemia Thrombosis	Serum levels of vitamin B_{12} and folate. RBC folate considered better indicator of tissue folate levels than serum folate. ↑ Urinary levels of methylmalonic acid in vitamin B_{12} deficiency. Hematologic variables may be normal or abnormal. Anemia, increased mean corpuscular volume Increased serum levels of plasma homocysteine Hypersegmented neutrophils may be present Marked hyperhomocysteinemia
GU	Impotence	
CNS	Subacute combined degeneration of spinal cord Gait ataxia Romberg sign, memory deficits, psychosis	
PNS	Diminished vibratory sense, proprioception, and sensation; paresthesias, loss of deep tendon reflexes	

Key References: Badner NH, Freeman D, Spence JD: Preoperative oral B vitamins prevent nitrous oxide-induced postoperative plasma homocysteine increases. *Anesth Analg* 93(6):1507–1510, 2001; Nagele P, Zeugswetter B, Wiener C, et al: Influence of methylenetetrahydrofolate reductase gene polymorphisms on homocysteine concentrations after nitrous oxide anesthesia. *Anesthesiology* 109(1):36–43, 2008.

Perioperative Implications

Preoperative Preparation

- If elective procedure, postpone to correct vitamin deficiencies and hematologic and/or neurologic abnormalities.
- Preop vitamin therapy has been shown to prevent an increase in homocysteine levels after nitrous oxide anesthesia.

Monitoring

- Myocardial ischemia may occur with anemia and is associated with increased homocysteine levels.
- Basic monitoring should include measuring ECG (lead II and V5) for ischemia, pulse rate, pulse oximetry, and BP by noninvasive method. Consider direct intra-arterial BP monitoring in unstable pts.

Induction/Airway

- Large and painful tongue may be present. Hence, with the need for general anesthesia:
  - Tongue swelling may complicate optimal mask ventilation and intubation. An oral airway should be immediately available.
  - With the anticipation of a difficult intubation, consider the use of airway adjuncts (e.g., video laryngoscopy) or an awake/anesthetized fiberoptic endotracheal tube placement. A difficult airway cart should always be in close proximity.

Maintenance

- Avoid nitrous oxide if pt is known to be vitamin B_{12}/folate deficient and has hematologic and/or neurologic abnormalities.

Adjuvants

- Regional: Documentation of preexisting neurologic deficits is required before proceeding with regional anesthesia.

Postoperative Period

- Worsening of hematologic and neurologic abnormalities may not occur until several wk after nitrous oxide exposure.
- Monitor for postop myocardial infarction.

Anticipated Problems/Concerns

- Anemia may result in impaired oxygenation of tissues and be associated with myocardial ischemia.
- CNS and PNS symptoms may exist.
- Nitrous oxide may exacerbate preexisting hematologic/neurologic symptoms associated with vitamin B_{12} and/or folate deficiency.