Ventricular Septal Rupture (Defect), Postmyocardial Infarction

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Risk

- Historically seen in 1–3% of MIs prior to era of acute revascularization.
- Incidence is 0.2% in current era of acute percutaneous intervention.
- Most occur within 1 wk of MI; 20–30% occur in first 24 h post-MI.
- Rarely occurs >2 wk post-MI.
- Medical management alone results in a mortality >90%.

Perioperative Risks

- Accounts for 5% of MI-related deaths,
- Without surgical therapy, survival is less than 10% at 1 mo.
- Surgical short-term survival 40–81%.
- Increased mortality seen in the setting of urgent repair (due to tissue fragility), posterior VSD, preop dialysis, mitral regurgitation, and redo cardiac surgery.
- Improvements in surgical techniques have enabled earlier surgery prior to hemodynamic deterioration, with associated increase in survival.
- Percutaneous device closure with GA and TEE has similar mortality.

Worry About

- Associated papillary muscle rupture
- Poor systemic perfusion and end-organ dysfunction
- Pulm congestion with massive L-to-R shunt

Overview

- Sudden onset of holosystolic murmur with thrill and hemodynamic deterioration (hypotension and pulm congestion).
- Despite advances in periop management, expect increased morbidity and mortality.
- Expect a complicated postop course with prolonged ICU stay.

Usual Treatment

- Repair of new VSD with hemodynamic deterioration using pericardial or prosthetic patch material.
- Support preop with inotropic agents/intra-aortic balloon counterpulsation.
- Percutaneous device closure as an alternative to surgery.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Low forward cardiac output due to massive L-to-R shunt	Sudden onset of hypotension and shock	Loud holosystolic murmur and thrill	ECHO, cardiac cath
RESP	Congestion/edema	Respiratory distress	Rales	CXR
RENAL/HEPATIC	Dysfunction due to cardiogenic shock	Anuria		ABGs, Foley cath

Key References: Arnaoutakis GJ, Zhao Y, George TJ, et al.: Surgical repair of ventricular septal defect after myocardial infarction: outcomes from the Society of Thoracic Surgeons National Database, *Ann Thorac Surg* 94(2):436–443, 2012; Jeppsson A, Liden H, Johnsson P, et al.: Surgical repair of post infarction ventricular septal defects: A national experience, *Eur J Cardiothorac Surg* 27:216–221, 2005.

Perioperative Implications

Preoperative Preparation

- Consider elective tracheal intubation and PEEP.
- Support cardiac output using inotropic agents.
- Lower resistance to forward cardiac output using afterload reduction, including intra-aortic balloon counterpulsation.
- Obtain coronary angiogram. Concurrent revascularization can potentially improve outcome, although recent studies have not found this.

Anesthetic Technique

- High-dose opioid/muscle relaxant technique common
- Prior to CPB, use minimal FIO_2 and PEEP (maximizes PVR) to decrease L-to-R shunt across VSD.

Monitoring

- Intra-arterial line.
- Most use PA cath owing to pulm Htn and for shunt quantitation; step up saturation between right atrium and PA to measure degree of shunting.
- Thermodilution cardiac output may be falsely elevated.
- TEE to define anatomy, diagnose assoc papillary muscle rupture, monitor ventricular function (including stroke volume), and assess adequacy of surgical repair.

Airway

- High airway pressures and frequent suctioning in the setting of pulm edema.

Induction

- High-dose opioid technique to maintain hemodynamic stability. Avoid vasodilation assoc with volatile anesthetics.

Maintenance

- If pt is hypertensive, titrate low doses of volatile agent or benzodiazepines.

Surgical Stages

- Pre-CPB:
  - Median sternotomy with aortic and biatrial cannulation.
  - Vein or internal mammary artery harvest may be required for concomitant myocardial revascularization.
  - Lowest FIO_2 consistent with adequate oxygenation.
- CPB:
  - Maintain Hct using hemofiltration and transfusion.
- Post-CPB:
  - Inotropic support almost universally required for LV failure.
  - RV failure common.
  - Assess ventricular repair using TEE or right atrial-to-pulm O_2 sat ratio.
  - FIO_2: 1.00 to minimize PVR.
  - May require ventricular assist devices.
  - Rule out residual shunting by TEE.
  - Emergent surgery is associated with residual shunt.
- Blood loss/volume concerns:
  - Antifibrinolytic therapy (beginning pre-CPB).
  - Transfuse coagulation factors based on results obtained from point-of-care testing (TEG, platelet function analyzers).

Postoperative Considerations

- Postop renal/hepatic/neurologic dysfunction
- Postop LV, RV, or biventricular failure

Anticipated Problems/Concerns

- Cardiogenic shock with MODS
- Prolonged ventilatory dependency and ICU stay
- Course not dramatically improved with percutaneous device closure

Ventricular Tachyarrhythmias

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Risk

- VTach/VFIB are uncommon but potentially fatal dysrhythmias requiring urgent diagnosis and management.
- Risk increases with age owing to the higher incidence of structural and ischemic heart disease and cardiac failure.
- Primary cause of sudden death and accounts for 75–80% of sudden cardiac death. Incidence in USA is about 300,000/y and similar in other developed nations.
- Males at greater risk (46% vs. 34%).
- Pts under 30 with HOCM, myocarditis, RV dysplasia, or long-QT syndrome are at higher risk for VTach/VFIB.

Perioperative Risks

- Cardiac and vascular surgery (up to 50% incidence) does not influence late mortality if LV function is preserved.
- Low cardiac output after CABG (requiring pressors) predicts life-threatening VTach/VFIB within 72 h postop.
- Cardiac ischemia.
- Uncorrected electrolyte and/or acid-base disturbances, hypoxia, hypercarbia, hypothermia.
- Use of class 1 and 3 antiarrhythmics, sympathomimetics, QT-prolonging drugs.
- Placement of central venous catheters.

Worry About

- Electrolyte imbalance (particularly hypokalemia and hypomagnesemia), acid-base disturbances, hypoxia, hypotension, fluid overload, ongoing myocardial ischemia, and metabolic disturbances.
- Use of IV epinephrine and other catecholamines/sympathomimetics.
- Drugs that prolong QT (organophosphates, antipsychotics, tricyclics) may precipitate PVT, particularly in Brugada and other long-QT syndromes.
- Poor cardiac function.
- Modulation of neuroendocrine stress responses.

- 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

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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