Overview

- Severity of disease varies widely based on the clinical and diagnostic criteria used to establish the diagnosis.
- MVP is defined by echocardiography as isolated prolapse of the mitral valve leaflets ≥2 mm beyond the mitral valve annular plane into the LA during systole. The myxomatous degeneration causing MVP is characterized by leaflet thickening, leaflet redundancy, chordal elongation, or chordal rupture. MRI may demonstrate scarring or fibrosis of the papillary muscle or inferobasal segments of the LV
- Structural changes lead to weakness and deformity of the valve apparatus. Annular dilation, stretching of leaflets and chordal elongation impair leaflet coaptation causing progression of MR.
- Fibrosis or scarring of the valve apparatus may increase the risk of ventricular arrhythmias or sudden cardiac death.
- Rupture of weakened chordae results in a flail leaflet and produces acute severe MR.
- Chronic MR causes progressive atrial dilation, eccentric LV hypertrophy, HF, and AF.
- MVP syndrome is MVP associated with a spectrum of nonspecific symptoms, including atypical chest pain, palpitations, exertional dyspnea, exercise intolerance, syncope, anxiety, lean body habitus, electrocardiographic repolarization abnormalities, and sudden cardiac death. A pathophysiologic link between genetics and the molecular biology of disease expression for MVP and its syndromes has yet to be fully defined.
- Onset of HF symptoms, LV dysfunction (ejection fraction <50% or end-systolic diameter >40 mm) and pulm Htn worsen pt prognosis. AF, left atrial enlargement, leaflet thickening (>5 mm), flail segments, and age >50 y is associated with worsening disease.

Etiology

- Familial (autosomal dominant, genetics not completely defined) or sporadic occurrence.
- Inherited connective tissue disorders.
- Myxomatous degeneration caused by dysregulation of collagen and elastin matrix protein synthesis and degradation.

Usual Treatment

- No treatment if asymptomatic or in pts with MVP syndrome without severe MR.
- Medical therapy with ACE inhibitors, beta-blockers, angiotensin receptor antagonists, aldosterone antagonists, and diuretics in pts with significant MR or HF (see Mitral Regurgitation or Heart Failure).
- Antiarrhythmic and anticoagulation therapy in pts with AF (see Atrial Fibrillation).
- Mitral valve repair recommended in pts with symptomatic severe MR or if asymptomatic with moderate LV dysfunction. Valve repair is reasonable in asymptomatic pts with preserved LV size and systolic function if expected mortality rate is <1% and likelihood of successful repair >95%. Mitral valve repair preferable to mitral valve replacement when a successful and durable repair can be achieved

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	MVP	Atypical chest pain	Mid- and late-apical nonejection systolic clicks	ECHO
	MR	DOE	Mid- to late-apical systolic murmur	ECHO
	AFIB	CHF	Irregular pulse	CXR
	Infectious endocarditis	NYHA class	Embolic phenomena	ECG
		Palpitations		TEE, blood culture
		Fever, chills		Cardiac MRI
CNS	Stroke	Neurologic deficits	Focal neurologic signs	Head CT scan
		TIAs		TEE
MS	Connective tissue disorders		Pectus excavatum	
			Scoliosis	
			Lean stature	

Key References: Delling FN, Vasan RS: Epidemiology and pathophysiology of mitral valve prolapse. New insights into disease progression, genetics, and molecular basis, *Circulation* 129(21):2158–2170, 2014; Frogel J, Galusca D: Anesthetic considerations for patients with advanced valvular heart disease undergoing noncardiac surgery, *Anesthesiol Clin* 28(1):67–85, 2010.

Perioperative Implications

Preoperative Preparation

- Assess presence and severity of MR.
- Assess for signs and symptoms of HF.
- Prophylactic antibiotics for endocarditis only indicated in pts with prior episode of IE.

Monitoring

- Routine.
- Consider invasive hemodynamic monitoring for major operations in pts with symptoms, severe MR, and/or LV dysfunction.

Preinduction/Induction/Maintenance

- Avoid Htn and acute increases in sympathetic tone.
- Consider regional anesthesia.

Adjuvants

- Interventions that increase BP, myocardial contractility, preload, or sympathetic tone may increase severity of MVP, MR, or the risk of chordal rupture.
- Antihypertensives, afterload reducing agents, and positive inotropic drugs are effective for increasing cardiac output in pts with significant MR.

Extubation and Postoperative Period

- Avoid Htn and acute increases in sympathetic tone.

Anticipated Problems/Concerns

- Htn and intravascular volume expansion may increase severity of MVP, worsen the degree of MR, and increase the risk of pulm edema and acute exacerbation of HF.
- Presence of severe MR, LV dysfunction, or associated connective tissue disease may alter routine management of pts with isolated MVP (see Mitral Regurgitation).
- Risk of sudden cardiac death among predominantly young females with MVP who have frequent ECG repolarization abnormalities.

Mobitz I (Second-Degree Atrioventricular Block)

James R. Zaidan

Risk

- Occurs after inferior MI or occasionally in trained athletes or in normal, sleeping people.
- Incidence varies based on etiology.

Perioperative Risks

- Without associated heart disease and without symptoms, should not present undue risk during anesthesia (e.g., in trained athletes).
- If occurs secondary to inferior MI, periop risk depends on extent of ischemic area.

Worry About

- Advancing to a higher-degree block if ischemic zone extends to anterior wall.
- Papillary muscle dysfunction may occur.

Overview

- Found usually in presence of CAD.
- Block generally occurs in AV node, resulting in normal QRS complexes.
- ECG reveals progressive lengthening PR intervals at decreasing increments and progressively shortening RR intervals leading to regular atrial rhythm and irregular ventricular rhythm.
- Bradycardia usually responds to atropine.

Etiology

- Acquired, usually with MI
- Increased resting parasympathetic tone relative to resting sympathetic tone

Usual Treatment

- Specific therapy in absence of heart disease not necessary unless pt is symptomatic.
- Treatment of an infarction-related Mobitz I block includes observation and medical therapy with atropine.
- Temporary pacing is necessary only if a medically unresponsive pt is symptomatic.
- Permanent pacing seldom required and considered only in persistently blocked, symptomatic pts.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Commonly no Sx Bradycardia on occasion	Exercise tolerance Angina SOB	Signs of CHF and decreased perfusion	ECG CXR
RENAL	Likely normal			Renal function testing?
CNS	No effect or decreased perfusion of CNS	No Sx or only mild Sx: Fainting, dizziness	Normal Bruits	PE Carotid US

Key References: Epstein AE, DiMarco JP, Ellenbogen KA, et al.: 2012 ACC/AHA/HRS focused update incorporated into the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society, *J Am Coll Cardiol* 61(3):e6–e75, 2013; Coumbe AG, Haksuh N, Newell MC, et al.: Long-term follow-up of older patients with Mobitz type 1 second-degree atrioventricular block, *Heart* 99(5):334–338, 2013.

Perioperative Implications

Preoperative Preparation
- Consider availability of transcutaneous pacing.

Monitoring
- Based on coexisting disease.
- Observe for and prepare to treat tertiary block when positioning PA cath in pt with Mobitz I block.

Airway
- None

Induction and Maintenance
- Regional or general.
- No contraindications to any standard anesthetic drugs.
- Intraoperative processes and drugs that increase atrial rate could decrease ventricular rate.

Extubation
- None

Adjuvants
- Cautious use of drugs that slow AV conduction

Anticipated Problems/Concerns
- Extension of infarcted area with higher-degree block and CHF

Mobitz II (Second-Degree Atrioventricular Block)

James R. Zaidan

Risk
- Occurs after anterior infarction and can quickly proceed to third-degree heart block

Perioperative Risks
- Risk of developing third-degree block

Worry About
- Rapid development into third-degree block, which requires temporary transvenous pacing

Overview
- Unlike Mobitz I block, Mobitz II block is located in bundle of His or bundle branches, resulting in lengthening QRS duration.
- PP and RR intervals are constant, and PR intervals are constant prior to the dropped QRS complex.

Etiology
- Acquired; usually associated with MI

Usual Treatment
- Temporary pacemaker insertion should be considered soon after onset of this block because third-degree block commonly occurs.
- Pacing does not improve survival.
- Atropine usually does not improve conduction.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Bradycardia	Exercise tolerance Angina SOB	Signs of CHF and decreased perfusion	ECG CXR Other tests as indicated
GU	Likely normal			Renal function testing?
CNS	Decreased perfusion of CNS	Fainting, dizziness	Normal? Bruits	PE Carotid US

Key References: Epstein AE, DiMarco JP, Ellenbogen KA, et al.: 2012 ACC/AHA/HRS focused update incorporate dinto the ACCF/AHA/HRS 2008 guidelines for device-based therapy of cardiac rhythm abnormalities: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Heart Rhythm Society, *J Am Coll Cardiol* 61(3):e6–e75, 2013; Arias MA, Sánchez AM: Obstructive sleep apnea and its relationship to cardiac arrhythmias, *J Cardiovasc Electrophysiol* 18(9):1006–1014, 2007.

Perioperative Implications

Preoperative Preparation
- Evaluation of CAD important.
- Likely a transvenous pacemaker will be in place.
- Transcutaneous pacing should be available if temporary transvenous pacing was not established prior to induction of anesthesia.

Monitoring
- Based on severity of heart disease and extent of infarcted area.
- Prepare to treat third-degree block when positioning a PA cath.

Airway
- None

Induction and Maintenance
- No contraindications to any standard anesthetic drugs.
- Any intraop process or drug increasing atrial rate could worsen block and decrease ventricular rate.

Adjuvants
- Cautiously use drugs that slow conduction through AV node unless they also slow SA nodal rate and allow 1:1 AV conduction and increased ventricular rate.
- First-degree AV block will persist if 1:1 conduction occurs.