Maintenance
- Tachycardia or hypovolemia and Hct <28 can precipitate ischemia.
- No agent with demonstrated outcome superiority.
- Intensively normalize hemodynamics and HR.

Extubation
- In nonstressful fashion for pt without compromising supply of O_2 to myocardium
- Aggressive stepped pain therapy recommended; alpha$_2$-adrenergic agonist recommended by some

Adjuvants
- CHF decreases liver blood flow and clearance of drugs requiring hepatic metabolism (such as lidocaine).
- β-adrenergic receptor antagonists and nitrates can be associated with profound hemodynamic disturbances if there are drug interactions or sudden preload, afterload, or contractility perturbations (such as rapid onset of spinal anesthesia).

Anticipated Problems/Concerns
- Preop and postop periods at least as great a cause of morbidity as intraop period.
- Restart antianginal and antiplaque therapies (i.e., statins, CO Q10, aspirin, DHA) and physical activity rehab program as soon as possible postop if D/C preop.
- Consider compassionate anxiety-relieving yet aggressive preop consultation and intensive stepped pain prophylaxis consultations postop.

Myocarditis

Ronak Shah

Risk
- Incidence of idiopathic or viral myocarditis in the general population is unknown.
- Infectious and noninfectious causes; viruses are the most common.
- Pts with autoimmune diseases are at increased risk.

Perioperative Risks
- If pt develops DCM: EF <35% increased risk of MACE.
- Atrial arrhythmia if significant mitral regurgitation is present.
- Postop respiratory failure secondary to pulm edema.

Worry About
- Acute cardiovascular decompensation.
- New-onset atrial or ventricular arrhythmias, complete heart block, or an acute MI–like syndrome.
- Worsening of chronic HF.
- Chest pain in acute myocarditis can result from an associated pericarditis or occasionally from coronary artery spasm.
- Sudden death occurs in ~12%.

Overview
- Inflammatory infiltrative process targeting the myocardium.
- Usually due to viral infection and/or a postviral immune-mediated response.
- Virus or infectious agent enters myocytes.
- Viral replication and cell necrosis initiate a response from host's immune system.
- Immune response declines with elimination of virus and ventricular function recovers.
- However, the autoimmune processes persist independently of detection of the virus genome in the myocardium, leading to the chronic phase, characterized by myocardial remodeling and development of DCM.
- DCM is enlargement of RV and LV with hypertrophied muscle fibers but no increase in size of the free wall of the septum; this gives the heart a spherical shape.
- The heart is 2–3 times larger than normal and systolic function is impaired.

Etiology
- Endomyocardial biopsies have implicated multiple viruses, such as coxsackievirus B, adenovirus, parvovirus B19, and even HCV.
- Bacterial causes include *Chlamydia trachomatis*, *Corynebacterium diphtheriae*, *Legionella pneumophila*, *Mycobacterium tuberculosis*, *Mycoplasma pneumoniae*, *Staphylococcus aureus*, *Streptococcus aureus*, and *Streptococcus pneumoniae*.
- Other noninfectious causes include hypersensitivity and toxic reactions to medications.
- Autoimmune diseases such as giant cell myocarditis, hypereosinophilic syndrome, Kawasaki disease, lupus erythematosus, lymphofollicular myocarditis, rheumatoid arthritis, sarcoidosis, scleroderma, and ulcerative colitis place pts at higher risk.

Usual Treatment
- For acute DCM, treatment focus is supportive therapy for LV dysfunction.
- Most pts will improve with a standard HF regimen that includes ACEIs pr ARBs, beta blockers, and diuretics.
- Complete heart block and bradycardia are treated with a temporary pacemaker.
- If etiology is autoimmune-related, treatment includes immunosuppression therapy specific for the disease.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
HEENT	Lymphadenopathy if caused by viral/bacterial infection and sarcoidosis	Hx of fever, chills, upper respiratory tract infections	Enlarged cervical lymph nodes if progressed to DCM, JVD	Blood and sputum cultures, immunologic assays for viral infections
RESP	Pulm edema if HF develops	Dyspnea, frothy sputum Exercise intolerance and fatigue	Tachypnea, rales on auscultation	CXR, ABG analysis
CV	LV dysfunction, with LV dilatation and subsequent RV overload with chronic HF Atrial and ventricular arrhythmias	Orthopnea, chest pain, peripheral edema, fatigue, palpitations, and hepatomegaly	Tachycardia or irregularly irregular S_3 gallop Distant heart sounds Cardiomegaly (broad and displaced point of maximal impulse, RV heave)	Labs: Cardiac enzymes (troponin I or T) indicators for cardiac myonecrosis Viral antibody titers Rheumatologic screening ECHO to exclude other causes of HF and assess extent of cardiac dysfunction Coronary cath: rule out ischemic causes ECG: ST, QRS/QT prolongation, diffuse T-wave inversions, AV conduction defects and ventricular arrhythmias. Cardiac MRI Endomyocardial biopsy

Key References: Kaur H, Khetarpal R, Aggarwal S: Dilated cardiomyopathy: an anaesthetic challenge, *J Clin Diagn Res* 7(6):1174–1176, 2013; Daabiss MA, Hasanin A: Perioperative anesthetic management of a case with severe dilated cardiomyopathy, *Oman Med J* 25, 2010, doi:10.5001/omj.2010.20.

Perioperative Implications

Preoperative Preparation
- If acute cardiac decompensation presents with myocarditis, consider delaying surgery.
- Continue and optimize HF regimen (except for ACE inhibitors).
- Consider ECHO/CXR if there is a change in functional clinical status.
- If present, cardiac pacemaker should be evaluated.

Monitoring
- Consider invasive monitoring such as arterial line and pulm artery catheter, depending on type of surgery and condition of pt.
- Consider intraop TEE if significant hemodynamic changes occur.

Airway
- HF pts can present with frothy secretions resulting from pulm edema.

Preinduction/Induction
- Pts with DCM are extremely sensitive to cardiodepressants.
- Narcotic-based technique (EF <30%) is preferred to minimize cardiac depression.
- Also consider ketamine (<0.5 mg/kg) and etomidate.

Maintenance
- Conducted under general anesthesia.
- Fluid balance regulated to avoid hypervolemia and hypovolemia.
- Acute LV failure is more sensitive to the depressant effects of volatile agents.
- Afterload reduction in DCM is key as it will improve regional and global indices of ventricular relaxation and EF during anesthesia when myocardial depression is significant.

Adjuvants
- Hemodynamic instability can be treated with low-dose inotrope and vasodilator.

Anticipated Problems/Concerns
- Prolonged intubation secondary to pulm edema/hemodynamic instability.
- If LV function worsens despite optimal medical management, consider mechanical circulatory support, such as ventricular assist devices or ECMO, as a bridge to transplantation or recovery.