Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Pulm Htn Cor pulmonale	Dyspnea Exercise tolerance Leg swelling	S_3 Peripheral edema Distended neck veins	ECG CXR TTE
RESP	Pulm fibrosis Bulla/bleb formation	Cough Sputum production Dyspnea Exercise tolerance	Rales, rhonchi, Wheezing Cyanosis Use of accessory respiratory muscles RR	CXR ABG PFTs Inspiratory force Diffusing capacity Lung biopsy/lavage fluid microscopy
GI/ENDO	Weight loss Hyperglycemia (in chronic steroid treatment)			Body weight monitoring Blood glucose
MS	Generalized weakness			
RENAL	Renal insufficiency		Hypertension Peripheral edema Oliguria	Serum creatinine, BUN, potassium Creatinine clearance
IMMUNE	Hilar adenopathy (eggshell calcification) Increased susceptibility to infection, especially pulm	Cough Fever Sputum production		CXR Sputum culture and sensitivity

Key References: Rose C: Silicosis. In King TE Jr, Hollingsworth H, editors: *UpToDate*, Waltham, MA. www.uptodate.com/contents/silicosis. (Accessed 09.06.16); Stafford M, Cappa A, Weyant M, et al.: Treatment of acute silicoproteinosis by whole-lung lavage, *Semin Cardiothorac Vasc Anesth* 17(2):152–159, 2013.

Perioperative Implications

Preoperative Preparation

- Lung condition optimization: Treat bronchospasm (if present), bronchitis, and other pulmonary infections; possible lung lavage.
- Consider steroids (short course).
- Stop smoking at least 24 h before surgery.

Monitoring

- Preop and postop: Consider repetitive ABGs, lung mechanics (RR, TV, MV, FVC, NIF, etc.).
- Intraop: Arterial line; CVP is controversial. Consider PA catheter if pulm Htn is present and/or significant fluid shifts are expected.

Pre-induction/Induction

- Caution with IV agents that depress ventilation and regional techniques that affect accessory muscles of respiration (e.g., high epidural and interscalene blocks).
- Maintain adequate preload, and optimize cardiac output. Avoid hypoxemia, hypercapnia, and acidosis (both respiratory and metabolic), as these may increase PA pressures and worsen cor pulmonale.

Airway

- In case of difficult airways, consider techniques with spontaneous respiration preservation (e.g., awake FOI).

Maintenance

- Consider pressure-controlled mode of ventilation, for poor lung compliance may require increased airway pressures to reach the adequate TV. Observe for spontaneous pneumothorax, especially in severe disease.
- Optimize volume status, while avoiding crystalloids overload; rather, use colloids. If possible, minimize blood products use to avoid lung injury.
- Avoid hypotension. Treatment may include low doses of vasopressin, which decreases PA pressures while maintain systemic BP, rather than norepinephrine, which increases PAP and promotes acidosis; phenylephrine, while safe at low rates (0.2–0.6 mcg/kg/min), may exacerbate pulm Htn in higher rates.
- For severe metabolic acidosis treatment, consider THAM solution. Bicarbonate should be avoided because of excessive CO_2 production and hypernatremia.
- Consider use of remifentanil. Caution with long-acting opioids.
- For muscle relaxation, short-acting agents titrated to effect may be preferred.
- Any of inhalational agents are adequate options.

Extubation

- Consider temporary postop mechanical ventilation, especially for upper abdominal and thoracic surgery, until stringent criteria are met.

Postoperative Period

- Pain management is critical for adequate respiration and to avoid worsening pulm Htn.

Adjuvants

- Bronchodilators, supplemental O_2, incentive spirometry may improve ability to wean.

Anticipated Problems/Concerns

- Increased risk of respiratory failure and complications, especially after upper abdominal and thoracic surgery.
- Pts with pulm Htn with or without cor pulmonale are at increased risk of cardiac complications.

Single (Including Common) Ventricle

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Risk

- HLHS is the most common SV congenital cardiac malformation.
- HLHS accounts for 7.5% of newborns with CHD.
- Male predominance for HLHS.

Perioperative Risks

- Paradoxical emboli.
- Complications of chronic hypoxemia: Hyperviscosity, decreased coagulation factors and platelets
- Surgical shunts (narrowing of vessels anastomosed, obstructed shunts)
- Hypovolemia-induced poor pulm blood flow or shunt occlusion.
- Additional risks specific to anatomy and planned procedure.

Worry About

- Effect of changes in PVR, SVR, and cardiac function on blood flow, cardiac output, and O_2 saturation.
- Diastolic pressure and coronary perfusion.
- AV valve regurgitation.
- Systolic and diastolic dysfunction.
- Associated anomalies.
- Increasingly common to care for CHD and SV pts having noncardiac surgery who may be at various stages in the palliation repair process and may have comorbidities including protein losing enteropathies, plastic bronchitis, ventricular dysfunction, and arrhythmias.

Overview

- A wide variety of lesions are usually associated with atresia of the ipsilateral AV or semilunar valve resulting in SV physiology:
  - TA is the prototypic single left ventricle (see Tricuspid Atresia).
  - HLHS with mitral and aortic stenosis/atresia is the prototypic single right ventricle.
- Other anatomies include unbalanced AV canal, some double inlet or double outlet ventricles, and some heterotaxies.
- Initial lesion requires mixing of systemic and pulm venous return at ASD or VSD level. The SV output is divided between pulm and systemic circulations.
- SV anatomy may be associated with hypoplasia of a great vessel (pulm artery or aorta) and prior to initial palliation; systemic or pulm blood flow may be dependent on ductus arteriosus patency.
- Balance of blood flow in each Qp:Qs is governed by the relative resistance to flow as determined by both anatomic and vascular resistance considerations.
- Goal throughout all stages is to balance the Qp:Qs at 1:1.
  - With complete mixing, Qp:Qs at 1:1 results in sat of 75–80% at FiO_2 0.21.
- FiO_2, CO_2, and pH management can be used to manipulate the Qp:Qs.
- Qp:Qs > > 1 results in pulm overcirculation/pulm vascular congestion and potentially hypoperfusion to end organs.
- Qp:Qs < < 1 results in hypoxemia.

Etiology

- Incompletely understood and likely multifactorial. Has been associated with several genes (connexin protein 43, lesion at 11q23.3, cardiac homeobox transcription factor NKX2) and chromosomal abnormalities (Jacobsen syndrome, Turner syndrome, trisomy 18, trisomy 13).

Usual Treatment

- Series of palliative procedures with the goal of creating reliable systemic and pulm blood flow.
- Stage a connection of systemic venous return directly to pulm artery, dedicating the SV to systemic circulation.
- First, stable blood flow to systemic and pulm circulations are established and balanced.
  - For TA, a BT shunt is placed.
  - For HLHS, a stage I Norwood procedure is performed.
- For other SV lesions, BT shunt or PA banding as dictated by anatomy.
- Complete intracardiac mixing of blood is imperative.
- Stage I Norwood:
  - A neoaorta is created from hypoplastic aortic arch and native PA tissue, connecting the SV to systemic circulation.
  - A BT shunt provides pulm blood flow, connecting branch of neoaorta to ipsilateral pulm artery.
  - An atrial septectomy is performed to ensure complete intracardiac mixing of systemic and pulm venous blood.
- At completion of the stage I Norwood, the SV provides cardiac output to the systemic circulation via the neoaorta and the pulm circulation via the BT shunt.
- The second stage is the first of two procedures to direct systemic venous return to the pulm artery.
  - The SVC is connected to the ipsilateral PA, which remains connected to the PA confluence.
  - This procedure is referred to as a cavopulmonary connection, Bidirectional Glenn or hemi-Fontan, and is commonly performed around 6 mo of age.
  - Low PVR is necessary to promote pulm blood flow, which is passive.
- The final stage, Fontan completion, is typically done 18 mo-5 y.
  - The IVC blood is directed to the ipsilateral PA, either intracardiac via lateral tunnel or extracardiac via graft.
  - This effectively separates the circulations and reduces volume workload on SV; systemic venous return now flows passively to the PA without interposed pumping chamber.
  - A small fenestration from the IVC-PA conduit to the atrium is sometimes created. The fenestration ensures preload to the systemic circulation even when PA pressures fluctuate, maintaining cardiac output but at the expense of decreased O_2 sat via right-left shunt.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	CHF	Dyspnea, tachypnea, feeding difficulties	S_3, rales, wheeze, enlarged liver, metabolic acidosis	CXR, pulse oximetry, ABG ECHO
	Hypoxia Arrhythmia	Dyspnea, tachypnea, feeding difficulties cyanosis	Cyanosis	CXR, pulse oximetry, ABG ECG
HEME	Polycythemia	See above	See above	Hgb, Hct

Key References: Barron DJ, Kilby MD, Davies B, et al.: Hypoplastic left heart syndrome, *Lancet* 374(9689):551–564, 2009; Yuki K Casta A, Uezono S: Anesthetic management of noncardiac surgery for patients with single ventricle physiology, *J Anesth* 25(2):247–256, 2011.

Perioperative Implications

Preoperative Preparation

- Depending on the stage of the palliative process (Norwood stage I, Glenn/hemi-Fontan, completion Fontan), optimize hemodynamics.
- Cardiac catheterization is typically performed prior to Glenn/hemi-Fontan to measure PVR and coil any collateral venous vessels.
- Higher O_2 sat can decrease O_2 delivery to the tissues by facilitating overcirculation to the lungs, particularly when pulm blood flow is via BT shunt.

Monitoring

- Arterial BP.
- CVP monitoring via IJ is controversial due to SVC thrombosis risk and implications for subsequent staging, which requires patency of these vessels.
- Consider TEE.

Preinduction/Induction

- Dependent on exact anatomy and stage of palliation.
- Induction technique should consider impact of PVR and SVR changes on myocardial, systemic, and pulm blood flow.

Airway

- ET intubation and PPV.
- Minimize intrathoracic pressures where possible to encourage pulm blood flow.

Maintenance

- IV or inhalational agents are acceptable.
- Body temperature as dictated by potential use of cardiopulmonary bypass.

Extubation

- Following stage I Norwood, pt requires mechanical ventilation for >2 d.
- Early extubation is recommended to facilitate pulm blood flow after stage II (Glenn or hemi-Fontan) or the completion Fontan. High intrathoracic pressure from PPV impedes venous flow to the pulm circulation, while negative intrathoracic pressure (spontaneous respiration) enhances flow.

Anticipated Problems/Concerns

- Overcirculation.
- Hypoxemia.
- New anatomy postprocedure will necessitate a reassessment of desired PVR and SVR to optimize flow to both circulations.
- Postop low cardiac output syndrome.

Sleep Apnea, Central and Mixed

Michael F. Roizen | Andreas M. Ostermeier

Risk

- Incidence USA is 3–12% of middle-aged adults (which has increased fourfold in last 15 y, presumably due to increase in obesity). The M:F ratio is 2–2.5:1; obstructive or mixed.
- Risk increases with male sex, upper middle age (55–64 y), obesity, and Hx of snoring with impaired daytime performance.
- In elderly, risk is 2× higher for African Americans.

Perioperative Risks

- Increased risk of central and mixed (central and obstructive) apnea. In mixed SAS, obstructive apnea component can mask central apnea.
- Risk for respiratory depression also in intubated, tracheotomized, and awake pts.
- Increased risk with sedative-hypnotic narcotics, postop with any form of pain relief.

Worry About

- See medical records for previous problems.
- Look for related medical disorders (e.g., cor pulmonale, cardiac arrhythmias, erythrocytosis, disordered cognition, daytime somnolence).
- Apnea possible even several h postop, especially after epidural anesthesia.
- When administering O_2, think of possible dependence of ventilation on hypoxic drive.

Overview

- Central sleep apnea implies failure of respiratory rhythmogenesis. In SAS pts, at least 30 periods of apnea, defined as cessation of airflow for ≥10 sec, are found during normal nocturnal sleep.
- Obstructive sleep apnea relates to a failed or inadequate respiratory activation of upper airway muscles, resulting in lack of airflow.
- In central apnea, hypoventilation persists despite relief of obstruction.
- Central apnea is unaccompanied by any respiratory effort, in contrast to obstructive sleep apnea.
- Related to central alveolar hypoventilation syndrome, also known as Ondine curse.

Etiology

- Central: Familial basis is evident in some cases; possible relation to neurologic disorders (e.g., encephalitis in childhood, damaged respiratory centers, autonomic neuropathy in diabetes)