

Postoperative Period

- Continue invasive BP monitoring if necessary.
- Monitor volume status, respiratory mechanics, and temp closely.
- Abnormal lytes can affect BP and response to medications.

- Restart home dysautonomia medications as soon as possible postop.

Anticipated Problems/Concerns

- Autonomic dysfunction with CV collapse
- Aspiration risk

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Sick Sinus Syndrome

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Risk

- Highest incidence occurs in pts >60 y.
- Common in pts who have had congenital heart defect repair surgery.
- Sinus node dysfunction is a common indication for pacemaker implantation.

Perioperative Risks

- Syncope, symptomatic bradycardia, and asystole.

Worry About

- Sinus bradycardia can be poorly responsive to atropine and require a pacer intraop.
- Termination of a tachy-brady event where an atrial tachycardia occurs can lead to a prolonged bradycardia or asystole.
- A tachy-brady event can lead to demand myocardial ischemia and can precipitate heart failure in pts with related comorbidities.

Overview

- A significant portion of elderly pts with SSS frequently have other cardiac comorbidities, such as CAD.

- SSS includes several ECG abnormalities: Non-iatrogenic persistent spontaneous sinus bradycardia that is inappropriate for physiologic circumstance, sinus arrest or exit block, or alterations of paroxysmal atrial tachyarrhythmia (often fibrillation or flutter) and period of bradycardia—tachy-brady syndrome.
- Class I indications for pacemaker insertion in SSS include SND with documented symptomatic bradycardia, or sinus pauses; SND as a result of essential long-term therapy; or SND with symptoms of chronotropic incompetence.
- A large portion of pts with chronotropic incompetence (defined as the inability to achieve 80% of age predicted HR during physiologic stimulus) also have SND.

Etiology

- Causes are numerous and not clearly defined.
- In the elderly, likely due to fibrosis of SA node and hypersensitivity to autonomic changes.
- In adults with congenital heart disease (especially with ASD repair, or extensive atrial reconstruction), likely due to surgical (direct or inflammatory) trauma to the SA node.

Usual Treatment

- When SSS remains undiagnosed until an episode of bradycardia in the OR, administer atropine (0.5 mg–3 mg IVP) and epinephrine (10 µg IVP with rapid up-titration based on HR), along with pacing (external or transvenous). Pts with SSS often have a poor response to atropine.
- When SSS is diagnosed preop (i.e., ECG changes with symptoms), suggest a pacemaker (often DDD or AAI). A pacemaker does not control tachyarrhythmia, but instead it allows antiarrhythmic therapy for tachycardia by pacing during bradycardia caused by the therapy.
- Anticoagulation with warfarin is used if continuous ECG monitor detects paroxysms of atrial tachyarrhythmia, since this subset of pts has an increased thromboembolic risk.

Assessment Points

System	Effect	Assessment by Hx	PE	Test
CV	Low cardiac output Tachy-brady event	Syncope, presyncope, lightheadedness, decreased exercise capacity, dyspnea on exertion, fatigue, confusion, memory loss, CVA (especially in elderly) Palpitation, angina, CHF symptoms	Bradycardia, tachycardia	ECG Continuous ECG monitor with symptom diary
RENAL	Accentuate SSS			Potassium (hypokalemia)

Key References: Semelka M, Gera J, Usman S: Sick sinus syndrome: a review, *Am Fam Physician* 87(10):691–696, 2013; Staikou C, Chondrogiannis K, Mani A: Perioperative management of hereditary arrhythmic syndromes, *Br J Anaesth* 108(5):730–744, 2012.

Perioperative Implications

Preinduction/Induction

- Factors that alter autonomic balance to favor a parasympathetic state can produce sinus bradycardia: Eye surgery, increased ICP, severe hypoxia, and cervical/mediastinal tumors.
- Optimize extrinsic factors that can decrease heart rate: hypoxia, hypothermia, ICH, and hypothyroidism.
- Volatile anesthetics, propofol, and vecuronium all decrease sinus node activity in a dose-dependent fashion. Consider using NMBA with vagolytic properties (e.g., rocuronium).
- Standard monitors (pulse oximetry, ECG).
- In pt population that is dependent on atrial systole for sufficient cardiac output (such as those with ischemic cardiomyopathy, aortic stenosis, or diastolic dysfunction), an atrial tachyarrhythmia (as part of the tachy-brady phenomenon) can lead to hypotension.

- Consider placing transcutaneous pacing pads on pt prior to induction, or placing a central venous introducer in preparation for a transvenous pacing wire if needed.
- In cases of asystole or bradycardia, attempt therapeutic medications: Atropine 0.5–1 mg IV q 3–5 min up to 0.04 mg/kg (max 3 mg), or ephedrine 5–5 mg IV bolus, or infusions of dopamine 5–20 µg/kg per min IV, or epinephrine 2–10 µg/min IV, or isoproterenol 2–10 µg/min. Electrical pacing is most likely to succeed.
- If an episode of tachycardia occurs in pt with pacemaker, use normal agents for control of arrhythmia, as the pacemaker should rescue bradycardia. If no pacemaker, use short acting agents for rate control such as esmolol.

Regional Anesthesia

- Case reports demonstrate that SSS may manifest from several types of blocks, including a regional block that produces sympathectomy, stellate

ganglion blockade, thoracic epidural, and spinal anesthesia.

- Standard monitoring is recommended.
- If history is strongly suggestive of SSS, consider having pacing pads in the room.

Postoperative Period

- If SSS manifests in a pt without pacemaker intraop, consult cardiology to further evaluate for permanent pacemaker placement.

Anticipated Problems/Concerns

- An episode of atrial tachyarrhythmia that terminates in asystole or significant bradycardia.
- Atrial tachyarrhythmia in a susceptible population (aortic stenosis, diastolic dysfunction) may itself compromise cardiac output.
- Symptomatic sinus bradycardia may not respond robustly (or at all) to atropine. It may need a pacing if a second agent (isoproterenol, epinephrine, ephedrine, or dopamine) does not work.