Table 7. Common Potentially Reversible or Treatable Causes of SND[S5.3.1-1]

Acute myocardial ischemia or infarction[S5.3.1-2–S5.3.1-4]
Athletic training[S5.3.1-5]
Atrial fibrillation[S5.3.1-6]
Cardiac surgery
Valve replacement,[S5.3.1-7,S5.3.1-8] maze procedure,[S5.3.1-7] coronary artery bypass graft[S5.3.1-9,S5.3.1-10]
Drugs or toxins*
Toluene, organophosphates, tetrodotoxin, cocaine[S5.3.1-11]
Electrolyte abnormality
Hyperkalemia,[S5.3.1-12] hypokalemia,[S5.3.1-13] hypoglycemia[S5.3.1-14]
Heart transplant:[S5.3.1-15] Acute rejection, chronic rejection, remodeling[S5.3.1-16,S5.3.1-17]
Hypervagotonia[S5.3.1-18,S5.3.1-19]
Hypothermia
Therapeutic (post-cardiac arrest cooling[S5.3.1-20]) or environmental exposure[S5.3.1-21]
Hypothyroidism[S5.3.1-22]
Hypovolemic shock[S5.3.1-23]
Hypoxemia, hypercarbia, acidosis[S5.3.1-24]
Sleep apnea, respiratory insufficiency (suffocation, drowning,[S5.3.1-25] stroke,[S5.3.1-26] drug overdose)
Infection[S5.3.1-27]
Lyme disease,[S5.3.1-28] legionella, psittacosis, typhoid fever, typhus, listeria,[S5.3.1-29] malaria, leptospirosis, Dengue fever, viral hemorrhagic fevers, Guillain-Barre[S5.3.1-30]
Medications*
Beta blockers, non-dihydropyridine calcium channel blockers, digoxin,[S5.3.1-31] antiarrhythmic drugs, lithium,[S5.3.1-32] methyldopa, risperidone, cisplatin, interferon

*Partial list.

Recommendation-Specific Supportive Text

1. Because patients are typically stable and minimally symptomatic on presentation with SND, no acute therapy is usually required, and evaluation of SND and assessment for potentially reversible causes can be performed in an outpatient setting.[S5.3.1-6,S5.3.1-33–S5.3.1-53] In some cases, although evaluation of reversible causes for SND should be undertaken, treatment may not be necessary (eg, stopping a beta blocker in an asymptomatic patient with sinus bradycardia after ST-elevation MI).[S5.3.1-54] Notably, some patients with tachy-brady syndrome may have improvement of sinoatrial node function after treatment aimed at maintaining sinus rhythm.[S5.3.1-6]

5.3.2. Acute Medical Therapy for Bradycardia

5.3.2.1. Atropine and Beta Agonists for Bradycardia Attributable to SND

Recommendations for Atropine and Beta Agonists for Bradycardia Attributable to SND

Referenced studies that support recommendations are summarized in Online Data Supplements 8, 9, 10, and 11.

COR	LOE	Recommendations
IIa	C-LD	1. In patients with SND associated with symptoms or hemodynamic compromise, atropine is reasonable to increase sinus rate.[S5.3.2.1-1–S5.3.2.1-4]
IIb	C-LD	2. In patients with SND associated with symptoms or hemodynamic compromise who are at low likelihood of coronary ischemia, isoproterenol, dopamine, dobutamine, or epinephrine may be considered to increase heart rate and improve symptoms.[S5.3.2.1-5–S5.3.2.1-11]
III: Harm	C-LD	3. In patients who have undergone heart transplant without evidence for autonomic reinnervation, atropine should not be used to treat sinus bradycardia.[S5.3.2.1-12,S5.3.2.1-13]

Synopsis

Several drugs can be used for the acute treatment of bradycardia (Table 8). Atropine is a parasympatholytic drug that blocks the muscarinic acetylcholine receptor. In the sinus node, it facilitates sinoatrial conduction and increases sinus node automaticity at doses of approximately 0.5 to 2 mg with a half-life of approximately 2 hours.[S5.3.2.1-14,S5.3.2.1-15]

Isoproterenol is a nonselective beta agonist with both chronotropic and inotropic effects on cardiac myocytes, enhancing sinus and atrioventricular nodal function without exerting a vasopressor effect.[S5.3.2.1-16,S5.3.2.1-17] In patients with SND undergoing isoproterenol infusion in the electrophysiology laboratory setting, heart rate increases similar to normal controls are described, although some patients do not demonstrate a robust heart rate response, may require higher dosages, or may have a vasodilatory effect.[S5.3.2.1-7,S5.3.2.1-8,S5.3.2.1-10]

Dopamine is a catecholamine with mixed alpha-adrenergic, beta-adrenergic, and dopaminergic effects that depend on dosage, distribution, and metabolism.[S5.3.2.1-18] At lower doses of 1 to 2 mcg/kg/min, the effect is predominantly vasodilatory, while at doses of 5 to 20 mcg/kg/min, enhanced chronotropy and inotropy predominate. Higher doses may be required for a chronotropic response but must be used judiciously because of the association with profound vasoconstriction and proarrhythmias.[S5.3.2.1-19] Epinephrine is a catecholamine with strong alpha-adrenergic