The Pulse: An Essential Vital Sign

Paul E. Marik

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Leaning Objectives

The objectives of this chapter are to review the pathophysiology, causes, and approach to the treatment of both sinus tachycardia and sinus bradycardia in ICU patients.

7.1 Introduction

It is by no accident that the four vital signs, namely, blood pressure, heart rate, respiratory rate, and temperature, are called *vital signs*. Yet, many clinicians do not appreciate the importance of these *vital* signs nor how to interpret them. The blood pressure (mean arterial pressure—MAP) and heart rate are the most important of the vital signs, while the temperature is the "least vital" of all the vital signs. Any patient with an abnormal vital sign is at an increased risk of death. The risk of death is compounded by derangements of multiple vital signs [1]. In addition, the trends in the vital signs are vitally important in tracking a patient's progress.

7.2 Physiology of Heart Rate

All cardiac myocytes in the embryonic heart have pacemaker properties. Some myocytes synthesize large amounts of contractile proteins to become "working" myocardium. Others retain pacemaking ability and generate impulses spontaneously; the mammalian heart region that ordinarily generates impulses at the greatest frequency is the sinoatrial (SA) node; it is the natural pacemaker of the heart. The SA node is the phylogenetic remnant of the sinus venosus of lower vertebrate hearts. In humans it is about 8 mm long and 2 mm thick. It lies in the groove where the superior vena cava joins the right atrium.

The autonomic nervous system controls various aspects of cardiac function, including the frequency at which the heart beats. However, cardiac function does not require intact nervous pathways as a completely denervated heart (a cardiac transplant recipient) can adapt well to stressful situations. Ordinarily, the frequency of pacemaker firing is controlled by the activity of both divisions of the autonomic nervous system. Increased sympathetic nervous activity, through the release of norepinephrine, raises the heart rate principally by increasing the slope of the pacemaker potential. This mechanism of increasing heart rate operates during physical exertion, anxiety, and certain illnesses, such as febrile infectious diseases. Increased vagal activity, through the release of acetylcholine, diminishes the heart rate by hyperpolarizing the pacemaker cell membrane and by reducing the slope of the pacemaker potential.

7.3 Tachycardia

Cardiac output (CO) is a function of heart rate (HR) and stroke volume (SV); Cardiac output = heart rate × stroke volume, with an increase in heart rate being the most important mechanism of increasing cardiac output. Tachycardia, defined as a heart rate > 100/min, therefore occurs in situations of increased oxygen demand with the need for an increased cardiac output or in conditions associated with decreased SV. Tachycardia also occurs in situations of increased sympathetic tone, i.e., anxiety and fight-flight response. Stroke volume may be reduced due to decreased preload (volume depletion) or impaired

systolic heart function. Since tachycardia reduces diastolic time during which ventricular filling occurs, stroke volume may decrease at high heart rates; this however only becomes clinically significant in patients with diastolic dysfunction who have impaired diastolic filling. This implies that unless the patient has predominant diastolic dysfunction, slowing the heart rate (e.g., with a beta-blocker) will reduce cardiac output and oxygen delivery.

There is decreased responsiveness to beta-adrenergic receptor stimulation and decreased reactivity to baroreceptors and chemoreceptors with aging. Fibrosis and calcification of the fibrous skeleton of the heart, composed of the annular rings and fibrous trigones, together with calcification of the bases of the aortic cusps develop. These changes contribute to the high incidence of sick sinus syndrome, atrial arrhythmias and bundle branch blocks. In younger persons, cardiac output is increased predominantly by increasing heart rate in response to beta-adrenergic stimulation. With aging there is a relative "hyposympathetic state" in which the heart becomes less responsive to sympathetic stimulation, possible secondary to declining receptor function. The aging heart, therefore, increases cardiac output predominantly by increasing ventricular filling (preload) and stroke volume rather by an increase in heart rate.

Sinus tachycardia is always an ominous sign, and its cause must always be determined. A presenting heart rate >105/min and a sustained heart rate >90/min in patients with hemodynamic compromise are associated with an increased risk of death [2, 3]. The higher the heart rate, the more life-threatening the situation, and a tachycardia >110/min in an elderly patient is a very worrying sign. Tachycardia is most commonly due to a low stroke volume and/or a hypermetabolic state with an increased oxygen demand and in most instances represents an appropriate compensatory response. However, the clinical context and derangements of the other vital signs are important in assessing the implications of a tachycardia. Tachycardia in combination with hypotension (SBP < 110 or MAP < 75 mmHg) and a high respiratory rate (>20/min) is a deadly trio [1]. A tachycardia in the setting of left- (systolic heart failure) or right-sided heart failure (e.g., pulmonary embolism) [4] is a particularly foreboding sign being indicative of severely diminished stroke volume. It is important to emphasize that in almost all circumstance, one should treat the underlying cause (if possible) of the tachycardia and not the tachycardia itself. Always determine the cause of a sinus tachycardia (echocardiogram, stroke volume determination, etc.) and never treat an unexplained sinus tachycardia with a beta-blocker. The higher the heart rate, the more life-threatening the situation, and a tachycardia >110/min in an elderly patient is a very ominous sign. ICU patients with cardiac risk factors and a persistent tachycardia (HR > 95/min) are at an increased risk of having an acute cardiac event [5].

The role of a short-acting cardioselective beta-blocker (esmolol) in resuscitated septic shock patients who remain tachycardiac is controversial. While Morelli et al. demonstrated a benefit from this approach [6], this study has a number of limitations, including the fact that all patients required high inotropic support with levosimendan and that the overall mortality was very high, which may have concealed a potential detrimental impact of beta-blockade. This approach may be of benefit in patients with demonstrated diastolic dysfunction; however, this therapy is best attempted under continuous cardiac output monitoring.

Inappropriate sinus tachycardia is defined as a sinus heart rate >100 bpm at rest (with a mean 24-h heart rate >90/min not due to primary causes) [7, 8]. Patients are primarily young women, and clinical symptoms range from intermittent palpitations to general multisystem complaints.

The more common causes of sinus tachycardia in the ICU setting include:

- Hypovolemia
- Blood loss (hemorrhagic shock)
- Myocardial dysfunction
- Sepsis
- Fever
- Hypoxemia
- Anxiety/delirium/agitation
- Substance withdrawal; alcohol, opiates, etc.
- Alcohol intoxication
- Thyrotoxicosis
- Pulmonary embolism
- Severe anemia
- Drug induced; dopamine, epinephrine, etc.
- Drug toxicity with sympathomimetic agents (cocaine, amphetamines), synthetic cannabinoids, etc.

7.4 Bradycardia

Sinus bradycardia is defined as a heart rate less than 60 beats/min. Patients with a sinus bradycardia usually have a rate between 45 and 59 beats/min, but on rare occasion it may be as slow as 35 beats/min. Sinus bradycardia is often benign and does not necessarily indicate sinus node dysfunction. In the ICU, sinus bradycardia is most commonly due to a drug reaction, but it may occur in patients with intrinsic disease of the conducting tissues of the heart. Bradycardia may also occur with hypothermia, hypothyroidism, and raised intracranial pressure. The most commonly implicated drugs include beta-blockers, calcium channel blockers, dexmedetomidine, propofol, clonidine, and digoxin. Dexmedetomidine, an alpha-2 receptor agonist, decreases the production and response to catecholamines and leads to bradycardia from these sympatholytic effects. Propofol induces bradycardia by blocking calcium and potassium channels in cardiac cells. While propofol and dexmedetomidine alone have a relatively low incidence of bradycardia when combined with other AV nodal blocking medications, the risk of bradycardia increases substantially [9].

7.4.1 Treatment

Asymptomatic bradyarrhythmias do not carry a poor prognosis, and in general no therapy is indicated. Recommended initial therapy for bradycardia inducing end organ perfusion problems is atropine. Atropine is an anticholinergic medication with parasympatholytic properties leading to enhanced SA node automaticity and AV node conduction. The initial intravenous dose of atropine is 0.5–1.0 mg, which can be repeated every 5 min to a total dose of 0.04 mg/kg (3 mg for the average adult). Dopamine and isoproterenol are alternative agents in patients who have responded poorly to atropine. Dopamine is the preferred catecholamine for symptomatic bradycardia refractory to atropine. Glucagon

may be beneficial in the treatment of bradycardia associated with β-blocker or calcium channel blocker toxicity. An initial intravenous dose of 0.05–0.15 mg/kg is recommended. Emergency cardiac pacing is indicated for patients with hemodynamically unstable bradycardia, especially for patients who have failed medical therapy. The presence of syncope, heart failure, or other symptoms accompanying bradycardias is an indication for pacemaker implantation.

Take-Home Messages

- Sinus tachycardia, defined as a heart rate >100/min is an ominous prognostic sign in the critically ill and injured patient.
- The underlying cause of the tachycardia must be determined in all cases, with treatment directed at the underlying cause. Treatment with a beta-blocker may be a hazardous complication.
- Sinus bradycardia, defined as a heart rate <60/min, is usually a benign rhythm that occurs most commonly due to an adverse drug reaction.

Conflicts of Interest None of the authors have any real or potential conflicts of interest with regards to this manuscript.

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