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Wolters Kluwer

Sinus tachycardia: Evaluation and management

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INTRODUCTION

Sinus tachycardia is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is elevated. Sinus tachycardia is most often a normal and physiologic response, for example during exercise. However, sinus tachycardia can in some instances be inappropriate or pathologic. It is one of the most commonly encountered (and often overlooked) heart rhythms that may portend an adverse prognosis, particularly in patients with cardiovascular disease.

The etiology, clinical presentation, evaluation, and management of sinus tachycardia, including inappropriate sinus tachycardia, will be reviewed here. Other supraventricular tachycardias, including sinoatrial reentry supraventricular tachycardia (which involves tissue from the SA node), are discussed elsewhere. (See "[Overview of the acute management of tachyarrhythmias](#)", [section on 'Narrow QRS complex tachyarrhythmias'](#) and "[Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation](#)" and "[Sinoatrial nodal reentrant tachycardia \(SANRT\)](#)".)

DEFINITION AND ECG FEATURES

Normal sinus rhythm (NSR) is the characteristic rhythm of the healthy human heart. NSR is considered to be present in adults if the heart rate is between 60 and 100 beats per minute, the P wave vector on the electrocardiogram (ECG) is normal, and the rate is largely regular

([waveform 1](#)). Inspection of the P-wave vector is often overlooked but is important. The normal sinus P wave demonstrates right followed by left atrial depolarization giving rise to an upright P wave in leads I, II and aVL, and a negative P wave in lead aVR.

By conventional definition, a tachycardia requires the heart rate to be greater than 100 beats per minute. As such, sinus tachycardia can be thought of as a sinus-driven rhythm (normal-appearing P wave axis on the surface ECG) which is occurring at a rate of greater than 100 beats per minute ([waveform 2](#)).

However, the "normal" heart rate is, in part, the result of the complex interplay between the sympathetic and parasympathetic nervous systems. It is affected by numerous factors and varies in part with age ([table 1](#)) [1-3]. The heart rate is usually between 110 and 150 beats per minute in infants, with gradual slowing over the next six years. The resting sinus rate in older children and adults is approximately 65 to 85 beats per minute, with slowing in older age [4-6]. There is also considerable variation based upon level of fitness and underlying medical comorbidities. (See "[Normal sinus rhythm and sinus arrhythmia](#)".)

ETIOLOGY AND CLINICAL SYNDROMES

Sinus tachycardia as a physiologic response — In the majority of patients, sinus tachycardia is a physiologic response to a demand for greater cardiac output, increased sympathomimetic state, or vagal/parasympathetic withdrawal. Sinus tachycardia is an important mechanism for increasing cardiac output in the setting of infection or volume depletion or other stressors/illnesses.

Sinus tachycardia is a normal physiologic response to exercise and conditions in which catecholamine release is physiologically enhanced or, less commonly, in situations where the parasympathetic nervous system is withdrawn. A long list of other factors may be responsible in selected cases, including:

- Fever
- Volume depletion
- Hypotension and shock
- Sepsis
- Anemia
- Hypoxia
- Pulmonary embolism
- Acute coronary ischemia and myocardial infarction

- Pain
- Anxiety
- Sleep deprivation
- Pheochromocytoma
- Hyperthyroidism
- Decompensated heart failure
- Chronic pulmonary disease
- Exposure to stimulants (nicotine, caffeine, amphetamines), anticholinergic drugs, beta blocker withdrawal, or illicit drugs
- Abrupt withdrawal of medications such as beta blockers

Postural orthostatic tachycardia syndrome — Postural orthostatic tachycardia syndrome (POTS) is a condition that occurs predominantly in young women in the absence of structural heart disease. Characteristically, patients develop symptoms upon assuming the standing position. Symptoms may include palpitations, fatigue, lightheadedness, or exercise intolerance. The 2015 Heart Rhythm Consensus statement defined POTS as a heart rate rise of ≥ 30 beats per minute (≥ 40 beats per minute in individuals 12 to 19 years of age) in the absence of orthostatic hypotension (≥ 20 mmHg systolic blood pressure drop) [7]. Sinus tachycardia is only one component of this condition, which is a disorder of autonomic dysregulation [8]. (See "[Postural tachycardia syndrome](#)".)

Inappropriate sinus tachycardia — Inappropriate sinus tachycardia, also called chronic nonparoxysmal sinus tachycardia, is an unusual condition that occurs in individuals without apparent heart disease or other cause for sinus tachycardia, such as hyperthyroidism or fever, and is generally considered a diagnosis of exclusion [9-12]. Inappropriate sinus tachycardia is defined as a resting heart rate >100 beats per minute (with a mean heart rate >90 beats per minute over 24 hours) associated with highly symptomatic palpitations [7,13]. Commonly used criteria to define inappropriate sinus tachycardia include [14]:

- P wave axis and morphology similar or identical to sinus rhythm.
- Resting heart rate of 100 beats per minute or greater (with a mean heart rate >90 beats per minute over 24 hours) or with activity heart rates of 100 beats per minute or greater but in excess of what one would expect for the amount of exertion. Additionally, patients with inappropriate sinus tachycardia classically experience a drop in heart rate during sleep.
- Palpitations, presyncope, or both related to the tachycardia. Very rarely do patients experience syncope.

- Exclusion of identifiable causes of sinus tachycardia.
- Exclusion of atrial tachycardia.

Most of these patients are young and female. Among a single-center cohort of 305 patients with inappropriate sinus tachycardia seen between 1998 and 2018, 92 percent were female, with an average age of 33 years [15]. Affected patients have an elevated resting heart rate and/or an exaggerated heart rate response to exercise that is out of proportion to the body's physiological needs; many patients have both.

Patients are invariably symptomatic; the presence of symptoms is an essential component of the definition. In contrast to sinus tachycardia occurring as a physiologic response, inappropriate sinus tachycardia can continue for months or years and may produce troublesome symptoms, most commonly with palpitations, but other common symptoms include chest discomfort, fatigue, dizziness/presyncope/syncope, and shortness of breath [15]. Most patients have resting heart rates of greater than 100 beats per minute and average heart rates on a 24-hour Holter greater than 90 beats per minute with no clear physiologic, pathologic, or pharmacologic trigger [12]. Characteristic of this disorder, the mean heart rate drops during sleep. Absence of nocturnal abatement of tachycardia should prompt consideration of a diagnosis other than inappropriate sinus tachycardia. The diurnal variations in heart rate seen in inappropriate sinus tachycardia may explain the low incidence of developing tachycardia-associated cardiomyopathy in this disease [12,16]. However, patients may rarely develop cardiomyopathy secondary to a sustained rapid heart rate. (See ["Arrhythmia-induced cardiomyopathy"](#).)

The pathophysiologic mechanism behind this disease is poorly understood and is thought to consist of intrinsic sinus node hyperactivity coupled with autonomic perturbations modulated by neurohormonal influences [12]. One study suggested that this tachycardia is related to a primary sinus node abnormality, characterized by a high intrinsic heart rate, depressed efferent cardiovagal reflex, and beta-adrenergic hypersensitivity [10,17]. Both [isoproterenol](#) and enhanced vagal tone shift the pacemaker site along the crista terminalis, while [adenosine](#) slows the rate but has little effect on the pacemaker focus and on activation sequence [18,19]. The shifting site of the SA pacemaker within the large epicardial SA nodal complex can be problematic when ablation is considered as a therapy [18].

CLINICAL PRESENTATION

Most patients with physiologic sinus tachycardia do not have symptoms directly attributable to the tachycardia itself but present with signs or symptoms related to the associated condition (eg, pain, fever, shortness of breath, etc). However, a patient with a greater awareness of his or her heartbeat may report palpitations (subjective awareness of a rapid or forceful heartbeat).

In contrast, patients with postural orthostatic tachycardia syndrome or inappropriate sinus tachycardia are commonly symptomatic, as discussed above. (See ['Postural orthostatic tachycardia syndrome'](#) above and ['Inappropriate sinus tachycardia'](#) above.)

As with any tachycardia, sinus tachycardia can indirectly lead to symptoms due to the impact of the tachycardia in the presence of underlying heart disease. Tachycardia may result in:

- Decreased cardiac output due to shortened ventricular filling time
- Increased myocardial oxygen consumption
- Reduced coronary blood flow

The above physiologic changes induced by tachycardia may result in symptoms of angina or dyspnea, the severity of which will depend upon how rapidly the heart is beating and the extent of the underlying cardiac comorbidities. In rare cases, sustained inappropriate sinus tachycardia can lead to tachycardia-induced cardiomyopathy [20,21]. (See ["Arrhythmia-induced cardiomyopathy"](#).)

Examination of precordial pulsation and the arterial pulse as means of assessing heart rate and rhythm is discussed separately. (See ["Examination of the precordial pulsation"](#) and ["Examination of the arterial pulse"](#).)

DIAGNOSTIC EVALUATION

Confirm sinus tachycardia — Sinus tachycardia is generally confirmed by ECG after a rapid pulse is identified on physical examination, with the diagnosis usually being easy to establish from the surface ECG. An upright P wave in leads I and II indicates a sinus origin of the tachycardia [1]. However, the P waves may be difficult to identify at heart rates above 140 beats per minute, since they are often superimposed on the preceding T wave ([waveform 2](#)). As a result, sinus tachycardia can be confused with another supraventricular tachycardia.

Vagal maneuvers (eg, carotid sinus massage, Valsalva maneuver) or intravenous AV nodal blocking agents (eg, [adenosine](#), [verapamil](#)) may help in the differentiation of sinus tachycardia from another supraventricular tachycardia by inducing one or more of the following (see ["Vagal maneuvers"](#)):

- Slowing the sinus rate to allow definitive identification of the sinus P waves.
- Causing transient AV nodal block to make atrial flutter with 2:1 block ([waveform 3](#)) or atrial tachycardia apparent.

Termination of a paroxysmal supraventricular tachycardia (atrioventricular nodal reentrant tachycardia or atrioventricular reentrant tachycardia). (See "[Narrow QRS complex tachycardias: Clinical manifestations, diagnosis, and evaluation](#)".)

Differential diagnosis — Sinus tachycardia should be distinguished from atrial tachycardias, which may appear similar on the ECG. Focal atrial tachycardia is a regular atrial rhythm at a constant rate of >100 beats per minute originating outside of the sinus node. If the focus of the atrial tachycardia is close to the sinoatrial (SA) node, such as in the case of atrial tachycardia emanating from the superior crista terminalis, the P wave may be similar in appearance to a sinus P wave, giving the impression of a sinus tachycardia. The sudden and inappropriate onset of tachycardia on an ECG monitor can help differentiate one from the other.

Sinus tachycardia also must be distinguished from sinoatrial nodal reentrant tachycardia. Sinus node reentrant tachycardia is a reentrant arrhythmia that is paroxysmal with a discrete onset and offset (unlike sinus tachycardia) [1]. Distinguishing focal atrial tachycardia or sinoatrial nodal reentrant tachycardia from sinus tachycardia can often only be accomplished in the EP laboratory. (See "[Focal atrial tachycardia](#)" and "[Sinoatrial nodal reentrant tachycardia \(SANRT\)](#)".)

The primary distinction between sinus tachycardia and an atrial tachycardia is made based on the clinical situation and the onset and termination of the tachycardia. Sinus tachycardia is an appropriate response to a physiologic, pathologic, or pharmacologic trigger. Its onset and resolution are gradual. In contrast, atrial tachycardias are paroxysmal in nature with abrupt onset and termination. Atrial tachycardias can generally be distinguished by their abrupt onset and termination, in contrast to the slow ramping up and slowing down of the heart rate in sinus tachycardia [22].

Further evaluation — For the majority of patients with sinus tachycardia, the underlying cause is determined from history and physical examination. Important features to elicit in a history and examination include exposure to stimulants and drugs, pain, and anxiety; and measurement of a full set of vital signs, including temperature and pulse oximetry [13].

Clues derived from the history and physical examination will direct subsequent evaluation, which can range from simple reassurance to admission and extensive testing depending on the situation. As examples:

- Patients with pain or anxiety will often require only simple reassurance, with return of the sinus rate to the normal range.
- Patients with a fever or other signs of infection should have a complete blood count drawn along with workup focused at the source of infection.
- Patients with signs or symptoms of volume depletion should be rehydrated; if the suspected cause of the volume depletion is anemia, a complete blood count should be performed.
- Patients with signs or symptoms of hyperthyroidism (TSH), pheochromocytoma (24-hour urinary catecholamines and metanephrines), or another systemic illness should have an evaluation focused on the suspected disorder.

The most important component of evaluation in sinus tachycardia is determining if the sinus tachycardia is due to a physiologic insult such as infection, pulmonary embolus, or blood loss. Patients with sinus tachycardia who have hypotension or signs of shock related to suspected volume depletion, signs of sepsis related to infection, or acute clinical deterioration related to another suspected medical condition (eg, hypoxia, myocardial ischemia, heart failure, etc) should be admitted for evaluation and treatment.

The postural orthostatic tachycardia syndrome, of which sinus tachycardia is one component, is diagnosed using a set of diagnostic criteria that incorporates exaggerated postural changes in heart rate elicited by standing, in the absence of orthostatic hypotension. The diagnosis of inappropriate sinus tachycardia is challenging and is typically made in a patient with persistent tachycardia in whom other clinical entities have been excluded. (See ['Inappropriate sinus tachycardia'](#) above.)

MANAGEMENT

Treat the underlying cause — In most settings, sinus tachycardia will improve or resolve following treatment directed at the underlying etiology. Rarely, patients with sinus tachycardia who have hypotension or signs of shock related to suspected volume depletion, signs of sepsis related to infection, or acute clinical deterioration related to another suspected medical condition (eg, hypoxia, myocardial ischemia, heart failure, etc) should be admitted for evaluation and treatment. Management is generally driven by the underlying causation, as therapy that is indicated for sinus tachycardia in certain conditions (eg, beta blockers for acute myocardial ischemia) may be contraindicated in other conditions (eg, hypovolemia or sepsis).

Management in patients with acute coronary syndrome — Sinus tachycardia occurs in one-third or more of patients with acute coronary syndromes [23,24]. The reasons for sinus tachycardia in acute coronary syndromes can vary significantly from a reaction to pain, associated anxiety, hypoxia, or even impending cardiogenic shock.

The heart rate usually declines over time to a level that reflects the degree of activation of the sympathetic nervous system. Patients with persistent sinus tachycardia usually have larger infarcts that are more often anterior and a marked impairment in left ventricular function, which is associated with substantial morbidity and increased early and 30-day mortality [24-27]. In addition, sinus tachycardia may increase the size of ischemic injury and infarction due to increased myocardial oxygen demand.

In view of the prognostic importance of sinus tachycardia, it is important to exclude other causes of this arrhythmia. These include fear, anxiety, fever, pericarditis, and medications. Heart failure, hypoxia, recurrent ischemia, and hypotension should be aggressively treated. In the absence of identifiable triggers, sinus tachycardia in acute myocardial infarction can be treated with cautious beta blockade. However, most patients will receive such therapy independent of the tachycardia since early beta blocker administration is part of routine management of acute myocardial infarction. (See "[Acute myocardial infarction: Role of beta blocker therapy](#)".)

Inappropriate sinus tachycardia — Treatment of symptomatic inappropriate sinus tachycardia is frequently challenging, often with suboptimal results [7,13,28]. Prior to beginning treatment, it is important to exclude other etiologies of sinus tachycardia, notably postural orthostatic tachycardia syndrome (POTS), and continue withdrawing any medications that may be contributing to tachycardia (eg, stimulants). (See '[Catheter ablation](#)' below.)

The pharmacologic management of inappropriate sinus tachycardia continues to evolve as new treatments are being developed. Once other etiologies of sinus tachycardia have been excluded, our approach to therapy is as follows:

- For patients with symptomatic inappropriate sinus tachycardia, we suggest a trial of beta blockade as the initial medical therapy.
- For patients with persistently symptomatic inappropriate sinus tachycardia after a trial of beta blockers, we suggest using [ivabradine](#) (5 mg to 7.5 mg twice daily).
- Radiofrequency catheter ablation to modify the sinus node may be a treatment of last resort for patients with refractory symptoms. However, symptomatic recurrence after sinus node modification is frequent, and repeated procedures often result in pacemaker implantation.

Beta blockers — While usually considered a first line of treatment, beta blockers have been poorly tolerated (usually due to the higher doses required for adequate heart rate control) and/or minimally effective for patients with inappropriate sinus tachycardia [13,28]. When attempting beta blocker therapy, we typically start long-acting [metoprolol](#) 50 mg daily, with upward titration for adequate heart rate and symptom control. The results with beta blockers are often disappointing, however, since even if ventricular rates can be controlled, the symptoms will often persist [12,13]. Beta blocker therapy may be effective if the cause of inappropriate sinus tachycardia is overactivity of the sympathetic nervous system [29]. However, control of the heart rate is difficult if the sinus tachycardia results from depressed vagal activity.

Ivabradine — For patients with persistently symptomatic inappropriate sinus tachycardia, we suggest using [ivabradine](#) (5 mg to 7.5 mg twice daily) with or without a beta adrenergic receptor blocker. Although ivabradine is not available in all countries, and its use for inappropriate sinus tachycardia would be considered an off-label use in the United States, various professional society guidelines for the treatment of supraventricular tachycardia both support the use of ivabradine alone or, preferably, in conjunction with beta blockers for inappropriate sinus tachycardia [7,13,28].

[Ivabradine](#) is labeled by the FDA for use in patients with systolic heart failure (ejection fraction <35 percent) with a resting heart rate above 70 beats per minute. It is a selective blocker of the sodium channel I_f , one of the channels that regulates sinus node automaticity [30-32]. Ivabradine decreases the depolarizing I_f current in the sinoatrial node, thereby decreasing the heart rate [32,33]. A large randomized study of ivabradine versus placebo in patients with coronary artery disease and left ventricular dysfunction supports the safety and efficacy of ivabradine for lowering heart rates, although these patients did not have inappropriate sinus tachycardia and there are limited data on long-term safety and efficacy [34]. Additionally, ivabradine appears to be an effective treatment option for patients with inappropriate sinus tachycardia [35-38].

- In a systematic review and pooled analysis that included 145 patients (70 percent female) from nine studies (one randomized trial and eight observational studies), all studies reported a reduction in heart rate following [ivabradine](#) (averaging between 10 and 20 percent reduction in mean resting heart rate) [39]. The majority of patients also reported improvement in symptoms following ivabradine.
- In a double-blind study of 21 patients with inappropriate sinus tachycardia, patients received either [ivabradine](#) (5 mg twice daily) or placebo for six weeks, followed by a washout period and cross over to the other treatment for six additional weeks [36]. Symptom evaluation and heart rate assessment using supine, standing, and exercise

electrocardiography were performed at the beginning and end of each phase of the study. Ivabradine use resulted in the following outcomes:

- Improvement in symptoms in all patients, with 14 patients (67 percent) reporting elimination of >70 percent of symptoms and 9 patients (43 percent) reporting complete resolution of symptoms while on [ivabradine](#).
- Significant reductions in resting heart rate (from 88 to 76 beats per minute), standing heart rate (from 108 to 92 beats per minute), and heart rate during effort (176 to 158 beats per minute).
- Significant increases in exercise time (7.2 to 8.9 minutes).

The long-term safety and efficacy of [ivabradine](#) is unknown. In early studies, it was shown to increase the risk for atrial fibrillation and development of phosphenes (enhanced visual brightness). Patients who develop atrial fibrillation should stop the medication. Continued use of ivabradine in the presence of phosphenes is reasonable [34]. (See "[Approach to the patient with visual hallucinations](#)".)

Catheter ablation — For patients with persistent symptomatic inappropriate sinus tachycardia despite optimal pharmacologic therapy, radiofrequency catheter ablation can be attempted, although ablation is performed very rarely and only after all other therapeutic options have been exhausted. The results of catheter ablation of the sinus node have been mixed. The goal is to modify the sinus node without ablating it completely to avoid the need for permanent pacemaker implantation. However, if the tachycardia is a reflex response related to POTS, ablation may lead to worsening symptoms; hence the importance of excluding POTS before attempting ablation of the sinus node. In fact, the 2015 consensus statement relegates catheter ablation/modification of the sinus node for inappropriate sinus tachycardia and for POTS as ineffective and probably harmful (class III) [7]. (See "[Postural tachycardia syndrome](#)", [section on 'Management'](#)".)

Total sinus node ablation and sinus node modification have been attempted in small numbers of patients with inappropriate sinus tachycardia; both are technically difficult because the sinus node is a sizable (and largely epicardial) complex of cells lying along the lateral right atrial wall, not a tiny discrete focus [40]. Total sinus node ablation leaves the patient with a junctional rhythm, which usually necessitates permanent pacemaker implantation. Sinus node modification involves an initial ablation at the superior aspect of the sinoatrial nodal complex. Ablation then proceeds inferiorly until the resting heart rate and the heart rate in response to [isoproterenol](#) infusion decrease markedly. In patients who do not respond adequately to

medical therapy or endocardial ablation, an alternative approach utilizing epicardial (via the pericardial space) ablation has been described [41,42].

Although studies are limited, both in number and in duration of follow-up, ablation for inappropriate sinus tachycardia may be modestly effective in highly selected patients with expert electrophysiologists performing the procedure.

- A 2017 systematic review of the literature identified 153 patients (mean age 35 years, 91 percent female, mean heart rate 105 beats per minute by 24-hour ambulatory recording) who underwent catheter ablation after failing to adequately respond to maximal medical therapy (mean 3.5 drugs) for inappropriate sinus tachycardia [40]. Acute procedural success (not uniformly defined) was reported in 89 percent of patients, with 86 percent of patients having a successful outcome at mean follow-up of 28 months, although 20 percent of patients reported recurrent symptoms. Severe procedure complications (eg, pericardial tamponade, superior vena cava syndrome, phrenic nerve paralysis) occurred in 13 patients (9 percent), and 15 patients (10 percent) required permanent pacemaker implantation.
- In a prospective registry of patients undergoing catheter ablation, 40 patients underwent ablation for inappropriate sinus tachycardia; acute success was achieved in 71.4 percent, but 10 percent had recurrent symptoms [43]. Other data suggest a much higher overall recurrence rate between 27 and 45 percent, in addition to the risks of right phrenic nerve injury, the future need for a pacemaker, and the risk of superior vena cava syndrome as a result of occlusion/thrombosis [13].

Sinus node ablation, however, is not effective in patients with inappropriate sinus tachycardia who have features of POTS; although the sinus rate is effectively slowed, there is no significant improvement in clinical symptoms [44]. In fact, ablation of the sinus node in POTS could lead to significant exacerbation of symptoms [12]. Given the young age of patients with inappropriate sinus tachycardia, the potential need for a pacemaker, the possible complications of the procedure (eg, phrenic nerve paralysis, superior vena cava syndrome), mixed results, and high recurrence rates, professional society guidelines do not support **routine** treatment with catheter ablation or modification of the sinus node for inappropriate sinus tachycardia, although it may be useful in very specific patients [7,13,28].

SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Supraventricular arrhythmias"](#).)

INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, "The Basics" and "Beyond the Basics." The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on "patient info" and the keyword(s) of interest.)

- Basics topics (see ["Patient education: Tachycardia \(The Basics\)"](#))
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SUMMARY AND RECOMMENDATIONS

- **Definition** – Sinus tachycardia is a rhythm in which the rate of impulses arising from the sinoatrial (SA) node is elevated. The normal heart rate has been considered historically to range from 60 to 100 beats per minute, with sinus tachycardia being defined as a sinus rhythm with a rate exceeding 100 beats per minute. However, the "normal" heart rate varies in part with age as well as level of fitness and underlying medical comorbidities ([table 1](#)). (See ['Definition and ECG features'](#) above.)
- **Causes and syndromes** – The most common cause of sinus tachycardia is a physiologic response to exercise and other conditions with catecholamine release, such as fever, volume depletion, hypoxia, pain, and anxiety. Other clinical syndromes associated with sinus tachycardia include autonomic dysregulation (eg, postural orthostatic tachycardia syndrome [POTS]) and inappropriate sinus tachycardia (cause unknown). (See ['Etiology and clinical syndromes'](#) above.)

- **Symptoms** – In the vast majority of patients, physiologic sinus tachycardia itself does not directly cause symptoms, although a patient with a greater awareness of his or her heartbeat may report palpitations (manifest as the sensation of a rapid heartbeat). However, as with any tachycardia, sinus tachycardia can indirectly lead to other symptoms due to the impact of the tachycardia in the presence of underlying heart disease. (See ['Clinical presentation'](#) above.)

In contrast, patients with POTS or inappropriate sinus tachycardia are generally symptomatic. (See ['Postural orthostatic tachycardia syndrome'](#) above and ['Inappropriate sinus tachycardia'](#) above.)

- **Diagnosis** – Sinus tachycardia is generally confirmed by ECG after a rapid pulse is identified on physical examination, with the diagnosis usually being easy to establish from the surface ECG. Since the tachycardia arises from the SA node, the P waves should have a normal or near-normal appearance on ECG and should occur in a regular fashion ([waveform 2](#)). (See ['Confirm sinus tachycardia'](#) above.)

- **Management**

- **For physiologic sinus tachycardia** – In most settings, sinus tachycardia will improve or resolve following treatment directed at the underlying etiology. Patients with sinus tachycardia who have hypotension or signs of shock related to suspected volume depletion, signs of sepsis, or acute clinical deterioration related to another suspected medical condition (eg, hypoxia, myocardial ischemia, heart failure, etc) require admission for evaluation and treatment. (See ['Management'](#) above.)
- **For acute coronary syndrome** – Because persistent tachycardia in a patient with acute coronary syndrome can result in larger infarcts and a more marked impairment in left ventricular function, treatment of sinus tachycardia with beta blockers is appropriate in most patients. (See ['Management in patients with acute coronary syndrome'](#) above and ['Acute myocardial infarction: Role of beta blocker therapy'](#).)
- **For inappropriate sinus tachycardia** – Treatment of symptomatic inappropriate sinus tachycardia is challenging, often with suboptimal results. Before embarking on treatment, exclusion of secondary causes of sinus tachycardia is imperative.

For patients with symptomatic inappropriate sinus tachycardia, we suggest a trial of beta blockade as the initial medical therapy (**Grade 2C**). We typically start long-acting [metoprolol](#) 25 to 50 mg daily, with upward titration for adequate heart rate and symptom control. Results are often disappointing. (See ['Beta blockers'](#) above.)

For patients with persistently symptomatic inappropriate sinus tachycardia, we suggest using [ivabradine](#) (5 mg to 7.5 mg twice daily) (**Grade 2C**). If symptomatic sinus tachycardia persists or the response is suboptimal, we will then add a beta blocker. Ivabradine is not available in all countries and its use for inappropriate sinus tachycardia would be considered an off-label use in the United States. (See '[Ivabradine](#)' above.)

For patients with persistent symptomatic inappropriate sinus tachycardia despite optimal pharmacologic therapy, radiofrequency catheter ablation may be attempted. Postural orthostatic tachycardia syndrome must be excluded first, since ablation may worsen symptoms in these patients. (See '[Catheter ablation](#)' above.)

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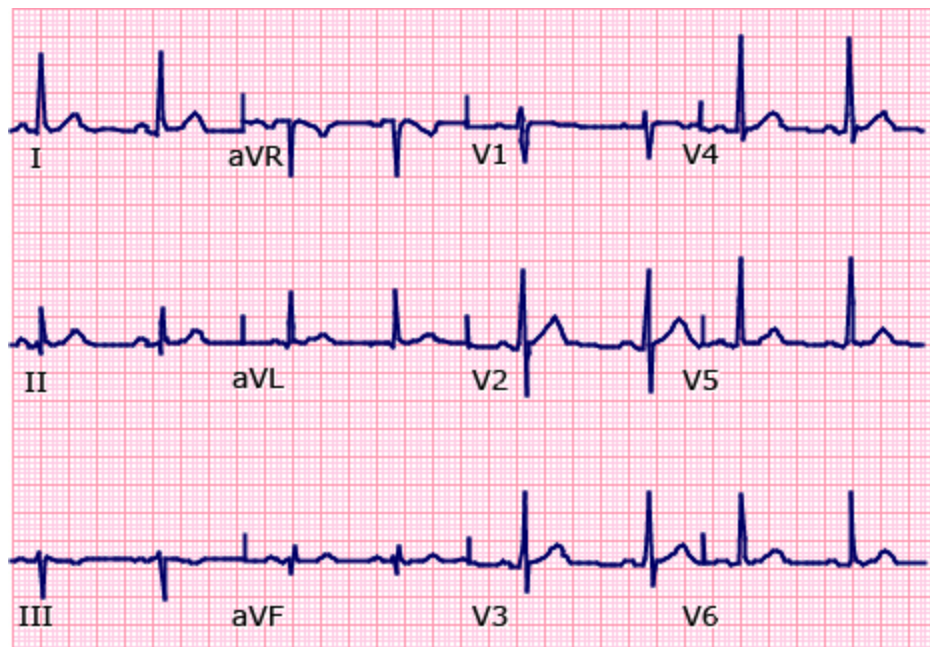
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Topic 1074 Version 41.0

GRAPHICS

Normal ECG in sinus rhythm



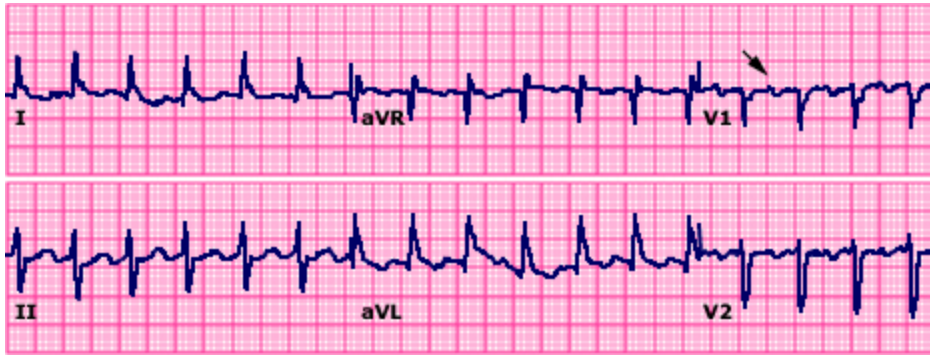
Normal sinus rhythm at a rate of 71 beats/minute, a P wave axis of 45°, and a PR interval of 0.15 seconds.

ECG: electrocardiogram.

Courtesy of Morton Arnsdorf, MD.

Graphic 58149 Version 6.0

Electrocardiogram (ECG) showing sinus tachycardia at a rate of 150 beats/min



Note the difficulty in separating the P waves from the T waves in the standard leads. The P waves are most evident in lead V1 (arrow) where the terminal negativity suggests left atrial enlargement.

Courtesy of Morton Arnsdorf, MD.

Graphic 78938 Version 3.0

Normal heart rates in adults based on age and sex

Age (years)	HR (beats per minute)								
	All			Male			Female		
	N	Mean	1%-99%	N	Mean	1%-99%	N	Mean	1%-99%
20-29	6086	67	43-98	3127	64	42-99	2959	69	46-99
30-39	9569	69	46-100	4605	67	44-99	4964	70	48-100
40-49	15,392	69	46-101	7104	68	45-101	8288	70	48-102
50-59	18,578	68	46-102	9936	68	45-102	8642	69	47-102
60-69	16,585	67	44-102	9457	65	42-102	7128	68	46-101
70-79	8432	65	43-101	4509	64	42-102	3923	67	44-101
80-89	2259	65	44-101	1001	63	41-98	1258	67	46-102
90-99	119	70	43-146	58	64	43-95	81	72	44-147

Normal heart rate values (with range from 1st to 99th percentile) for heart rate (beats/minute) in 77,276 healthy adults according to age and gender.

%; percent; HR: heart rate.

Data from: Mason JW, Ramseth DJ, Chanter DO, et al. Electrocardiographic reference ranges derived from 79,743 ambulatory subjects. *J Electrocardiol* 2007; 40:228.

Graphic 77746 Version 4.0

Atrial flutter at a rate of 250 beats/minute with 2:1 AV conduction in the presence of left bundle branch block



Although every other flutter wave can be seen at the end of the T wave in the first part of the tracing (arrows), a sinus mechanism cannot be excluded. The flutter waves become clearly apparent after carotid sinus massage is applied to slow conduction through the AV node, thereby increasing the degree of AV block.

AV: atrioventricular

Courtesy of Morton Arnsdorf, MD.

Graphic 76876 Version 3.0

Normal rhythm strip



Normal rhythm strip in lead II. The PR interval is 0.15 sec and the QRS duration is 0.08 sec. Both the P and T waves are upright.

Courtesy of Morton F Arnsdorf, MD.

Graphic 59022 Version 3.0

