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Chapter 24

Modern Diagnostic Modalities in Sinus Node Dysfunction

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Abstract

Sinus node dysfunction (SND) is a group of rhythm abnormalities caused by impaired function of the heart's intrinsic pacemaker, the sinoatrial node.

There are following types of sinus node dysfunction: "persistent" sinus bradycardia, chronotropic incompetence, sinoatrial blocks, conversion pauses, tachy-brady syndrome. Chronotropic incompetence is defined as failure to achieve 85% of the age-predicted maximum heart rate. Sinoatrial block is classified as first-degree SA block (undetectable on the surface ECG), type I second-degree SA block (progressive shortening of PP interval prior to the dropped P wave), type II second-degree SA block (intermittent dropped out P waves and QRS complexes, while subsequent P waves and QRS complexes arrive "on time"), third-degree SA-block (pauses lasting more than twice of the subsequent P-P interval). Conversion pauses referred to pauses just after termination of atrial fibrillation/flutter or supraventricular tachycardia. Tachy-brady syndrome is alternating between sinus bradycardia and paroxysmal atrial fibrillation/flutter or supraventricular tachycardia.

Diagnostic methods include conventional (surface) ECG, ambulatory ECG monitoring, implantable loop recorder, exercise testing, tilt table test, carotid sinus massage, electrophysiological study.

Only symptomatic individuals without any transient cause of SND require permanent pacing. DDD pacemaker is generally preferred over AAI pacemaker, except for young age and limited venous access. In the case of chronotropic incompetence, pacemaker with rate-responsive pacing is preferred. In the case of conversion pauses, pacemaker is often implanted, although ablation of atrial fibrillation/flutter or supraventricular tachycardia may be an option.

Keywords: Sick sinus syndrome, Sinus node dysfunction, Sinus arrest, Sinus exit block, Conversion pauses, Chronotropic incompetence.

Introduction

The sinoatrial node (SN) is located subepicardially at the anterolateral aspect of the junction of the superior vena cava and the right atrium [1]. It is a predominant cardiac pacemaker. Unlike atrial and ventricular cells, the pacemaker cells of the SN are characterized by their automaticity, arising from spontaneous diastolic depolarization [2]. The SN is composed of P cells (pacemaker cells that generate impulses and initiate cardiac contraction), T cells (transitional cells), and Purkinje-like cells [3]. Injury of any of these cell types may lead to sinoatrial node dysfunction (SND), also called sick sinus syndrome.

The incidence of SND is 0.8 per 1000 person-years [4]. Aging and cardiovascular risk factors significantly contribute to SND [5,6]

SND may result from abnormalities of either automaticity (P-cell dysfunction) or conduction (dysfunction of T cells and Purkinje-like cells). The most common causes of SND include SN fibrosis [7], myocardial ischemia [8,9], amyloidosis [10,11], sarcoidosis [12],

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myocarditis [13,14], and mutation in the *SCN5A* gene [15]. Also, beta-blockers, non-dihydropyridine calcium channel blockers, ivabradine, other antiarrhythmic medications, digoxin, and acetylcholinesterase inhibitors may depress the sinus node function leading to reversible SND [16]. Hypothermia [17], hypothyroidism [18], and obstructive sleep apnea [19] may be associated with SND.

Patients with SND typically complain of lightheadedness, fatigue, presyncope, and syncope [16]. Also, patients may complain of palpitations if they have alternating tachycardia and bradycardia, referred to as tachy-brady syndrome [20]. Patients with chronotropic incompetence may have reduced exercise capacity [21].

Diagnostic methods

Conventional ECG

Conventional 12-lead ECG should be performed in all patients presenting with symptoms consistent with SND. However, signs of SND may be found on conventional ECG in asymptomatic patients. Conventional 12-lead ECG has limited value in diagnosing some types of SND. It may be useful only if there is “persistent” sinus bradycardia (fig. 1) or frequent episodes of sinus exit block (fig. 2) and sinus arrest. Because of short registration time, infrequent sinus pauses are usually missed.

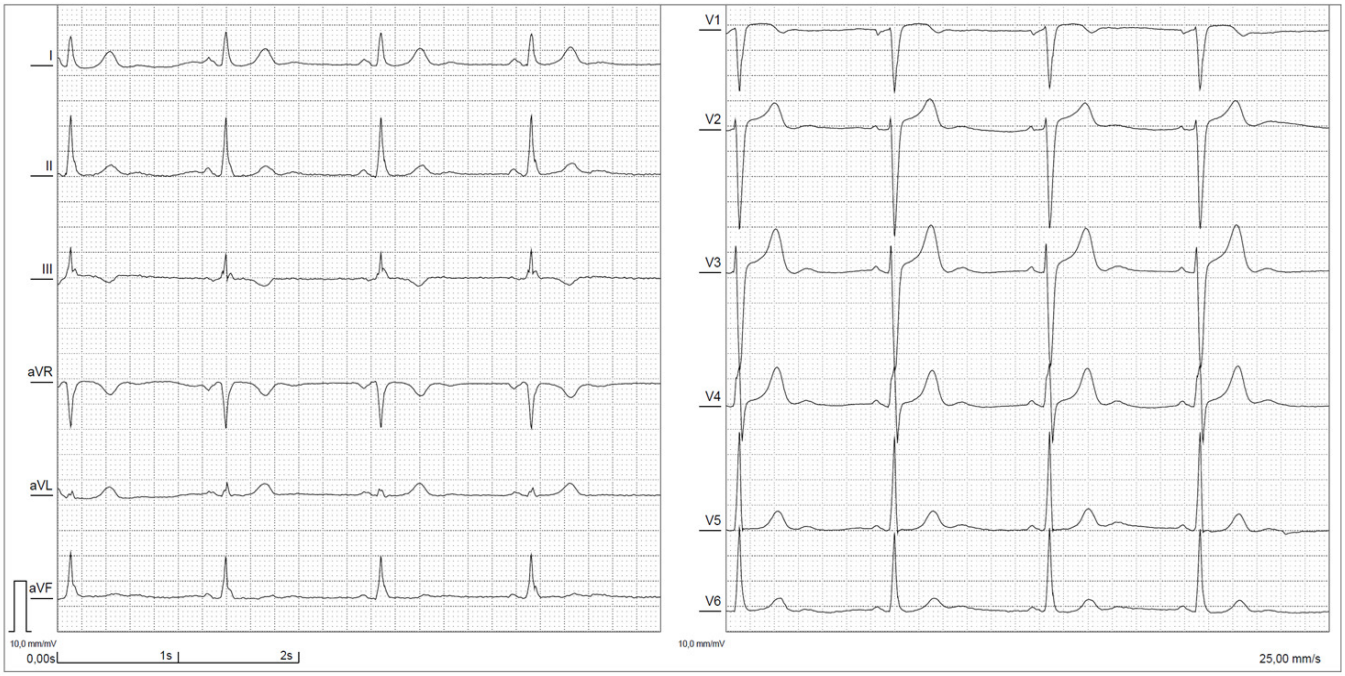

Figure 1: Sinus bradycardia with a heart rate of 47 bpm

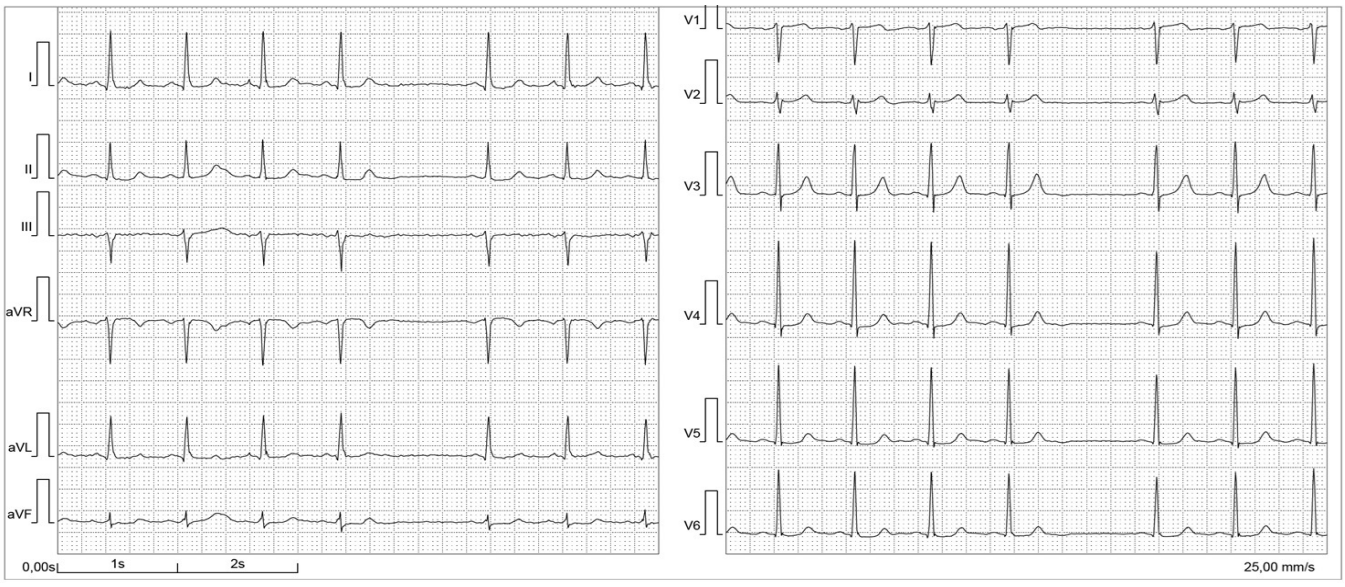

Figure 2: Type II sinus exit block

Ambulatory ECG monitoring

Ambulatory ECG monitoring is the most important diagnostic tool for the diagnosis of SND. Because of the prolonged monitoring duration (up to 14 days), signs of SND occurring rarely can be detected. If symptoms are prolonged and there is no syncope a handheld ECG recorder may be used. Nowadays, an implantable loop recorder is also an option.

There are the following types of SND:

1. *"Persistent" sinus bradycardia* (sinus bradycardia lasting for most monitoring time, the heart rate increases only upon exertions; figs. 3–5). If the rate of sinoatrial node discharge falls to the level below atrioventricular node discharge or Purkinje fiber discharge, junctional (fig. 6, 7) or idioventricular (fig. 8) escape rhythms occur. This results from dysfunction of P cells of the SA node.
2. *Chronotropic incompetence* (the heart's inability to appropriately increase its rate in response to heightened physical activity; fig. 5). It is usually confirmed with exercise testing, but heart rate trend allows to suggest chronotropic incompetence. This condition is due to P-cell dysfunction.
3. *Sinoatrial (SA) blocks* (T-cell dysfunction):
    - First-degree SA block is undetectable on the surface ECG; it can be diagnosed with electrophysiological study if sinoatrial conduction time (SACT) is prolonged.
    - Type I second-degree SA block (type 1 sinus exit block) is characterized by progressive shortening of PP interval prior to the dropped P wave (fig. 9).
    - Type II second-degree SA block (type 2 sinus exit block) is characterized by intermittent dropped out P waves (and QRS complexes), while subsequent P waves (and QRS complexes) arrive "on time". The pause surrounding the dropped P wave is equal to twice the subsequent P-P interval (fig. 10).
    - Third-degree SA-block is characterized by pauses lasting more than twice of the subsequent P-P interval (figs. 11, 12). Third-degree SA-block (dysfunction of T cells) cannot be distinguished from sinus arrest (dysfunction of P cells) on surface ECG and this condition is often referred to as sinus arrest. Sinus arrest is often terminated by junctional or ventricular escape complex (fig. 13).
4. *Conversion pauses* (pauses just after termination of atrial fibrillation/flutter or supraventricular tachycardia; figs. 14, 15). More time is needed for SN to restore its function. This is caused by P-cell dysfunction.
5. *Tachycardia-bradycardia (tachy-brady) syndrome* (alternating between sinus bradycardia and paroxysmal atrial fibrillation/flutter or supraventricular tachycardia; fig. 16).

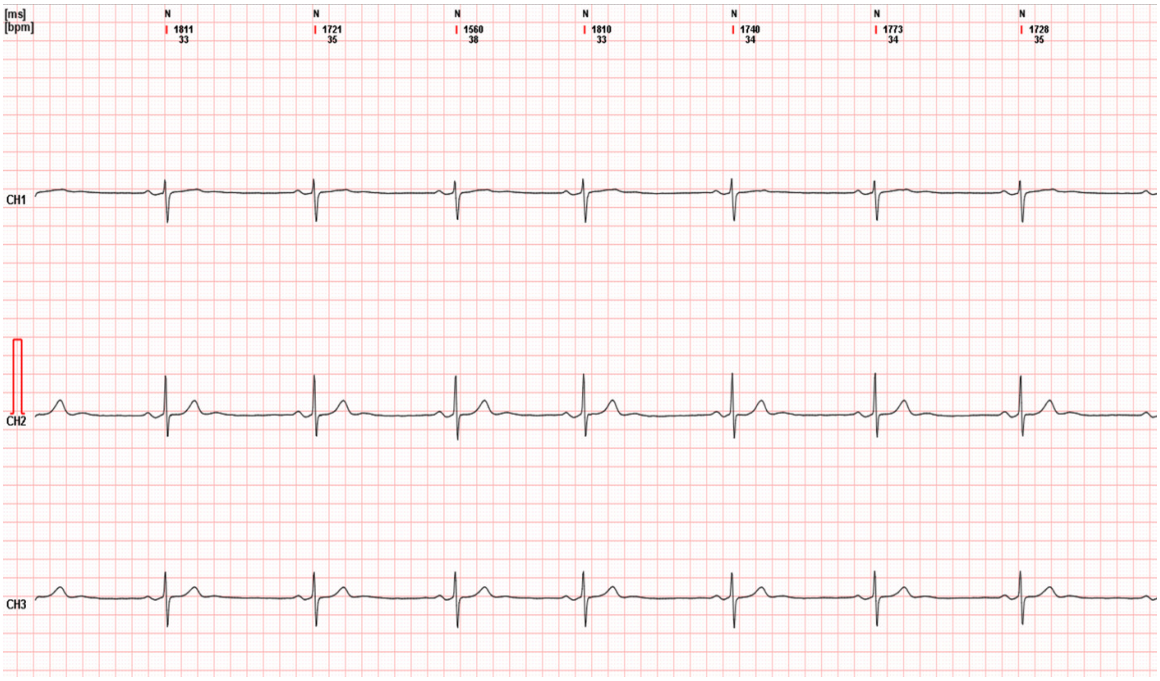

Figure 3: Sinus bradycardia with a heart rate of 35 bpm

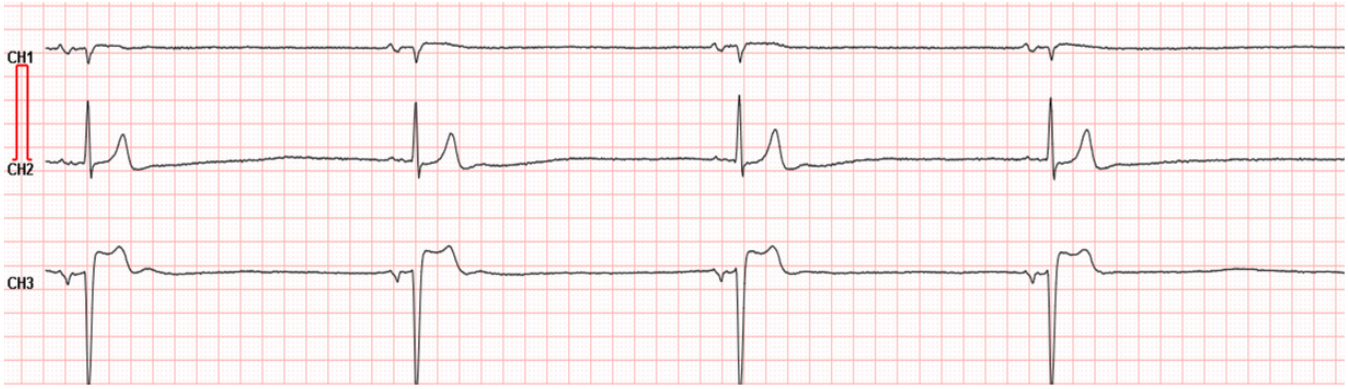

Figure 4: Profound sinus bradycardia with a heart rate of 20 bpm

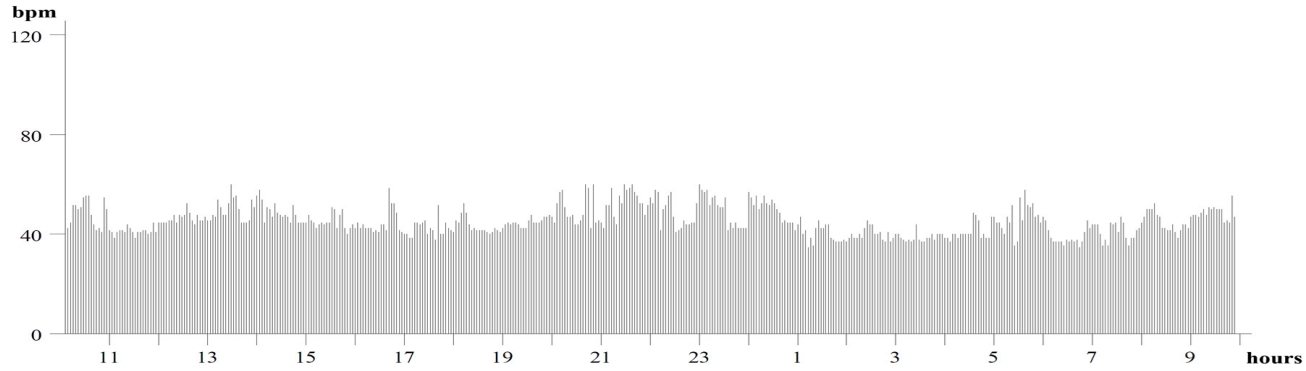

Figure 5: Heart rate trend in a patient with "persistent" sinus bradycardia and chronotropic incompetence. Minimum heart rate was 34 bpm. Mean heart rate was 43 bpm. Maximum heart rate was 62 bpm

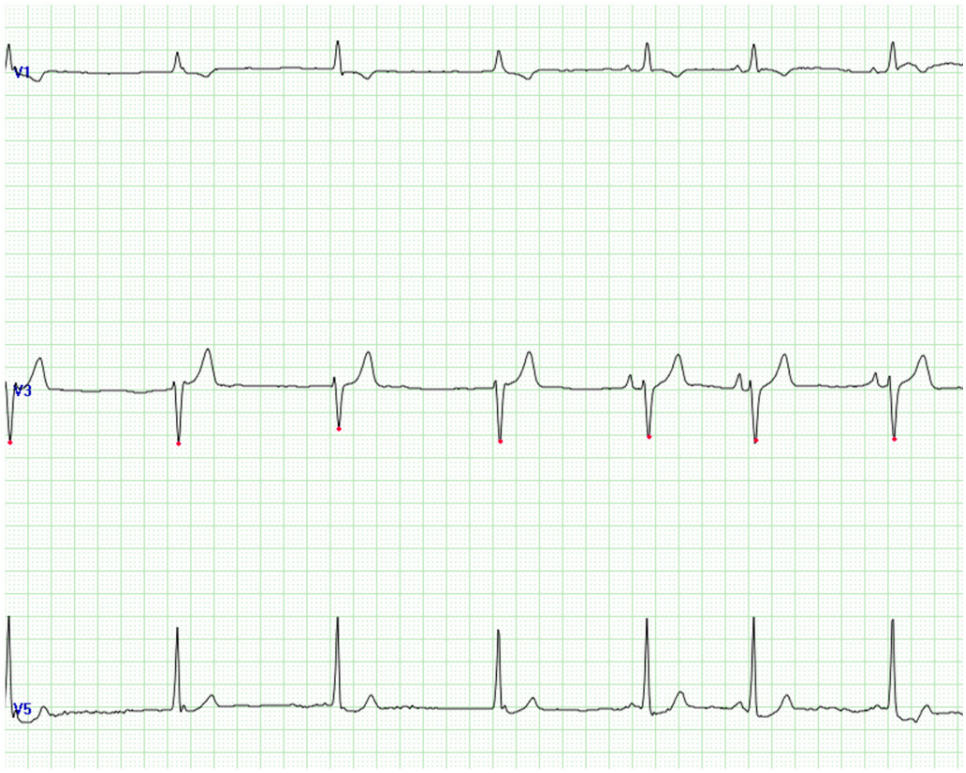

Figure 6: Transition from junctional escape rhythm (heart rate 42 bpm) to sinus rhythm

Note: The last three complexes are sinus complexes (each QRS complex is preceded by a P wave), whereas the preceding complexes are junctional (no P wave is visible before the QRS complex)

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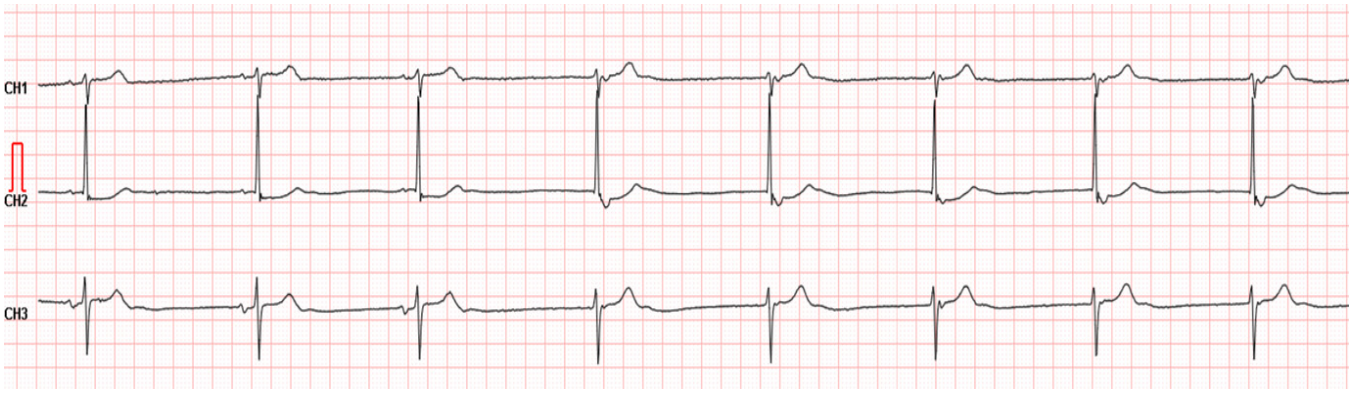

Figure 7: Transition from sinus rhythm to junctional escape rhythm (heart rate 35 bpm)

Note: The first three complexes are sinus complexes (each QRS complex is preceded by a P wave). The 4th to 8th complexes are junctional, with retrograde P waves visible immediately after the QRS complexes

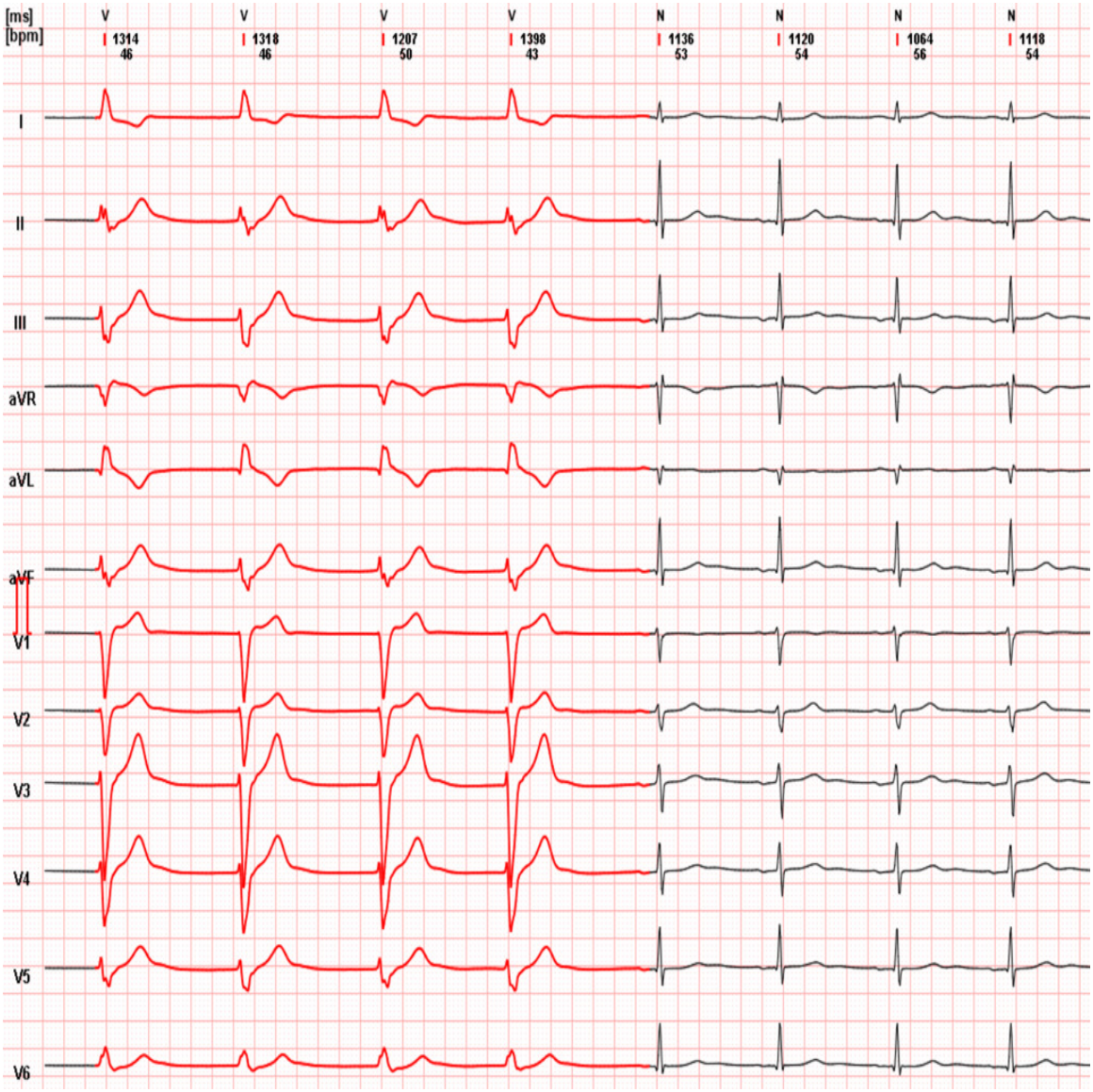

Figure 8: Transition from idioventricular escape rhythm to sinus rhythm

Note: The first four complexes (red) are wide and are not preceded by P waves, indicating their ventricular origin.

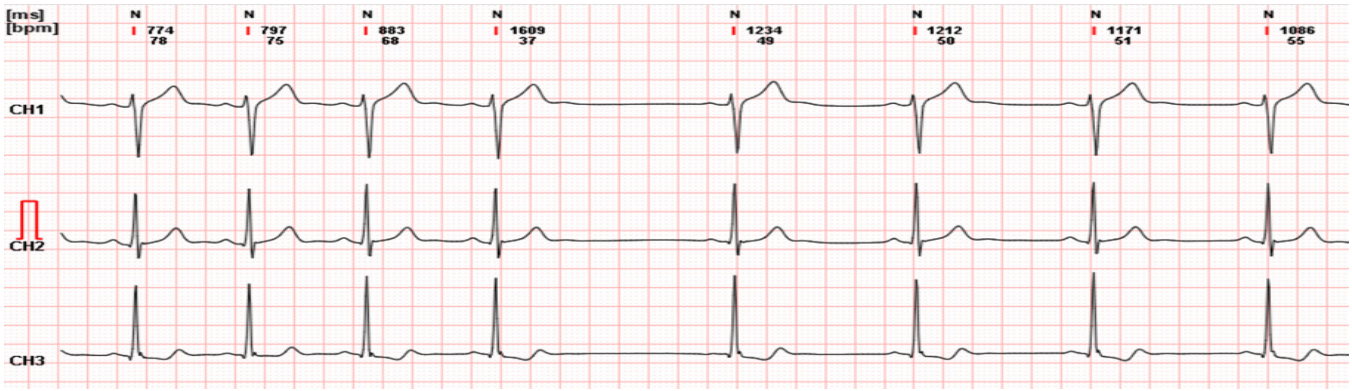

Figure 9: Type I sinus exit block

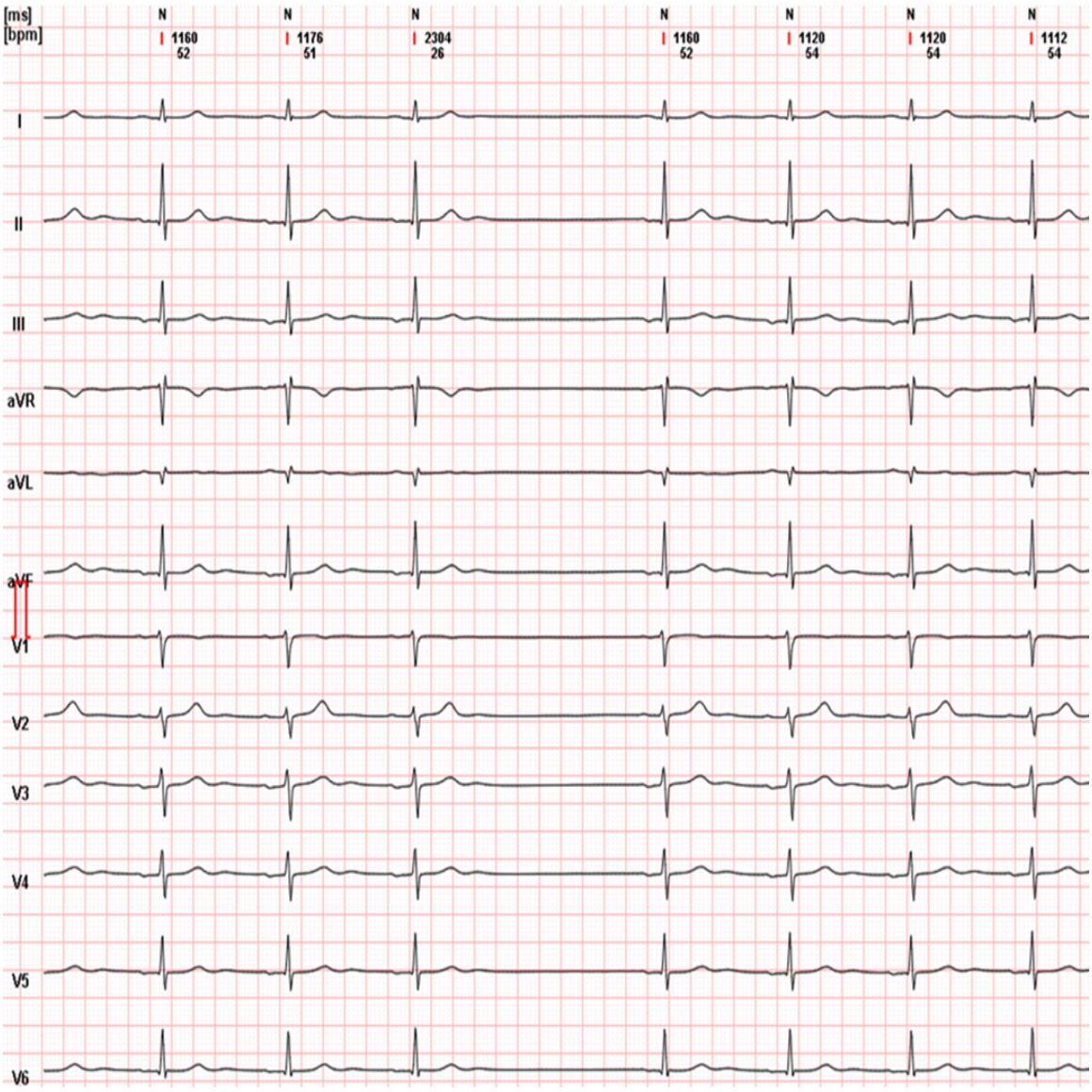

Figure 10: Type II sinus exit block

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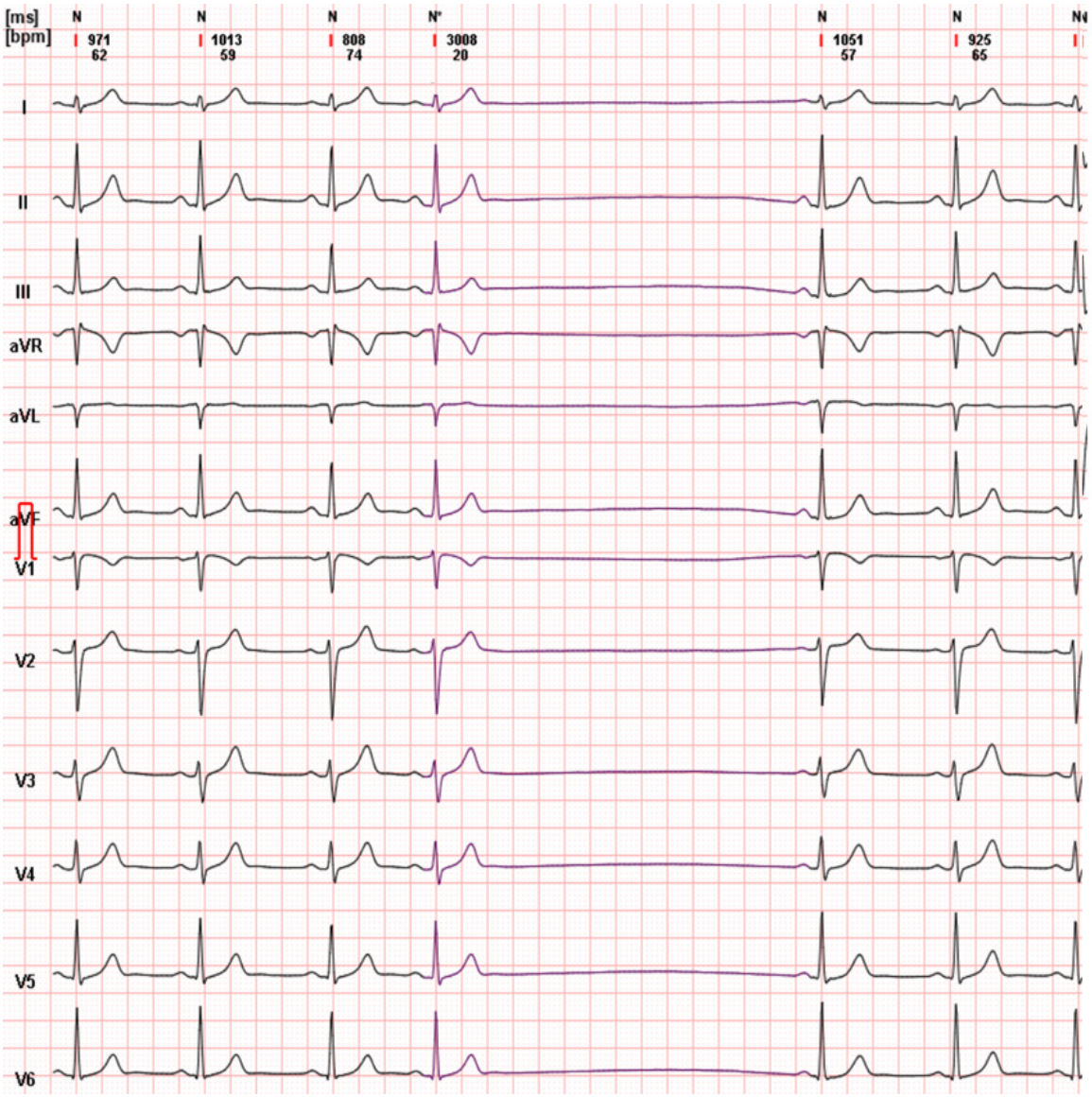

Figure 11: Sinus arrest

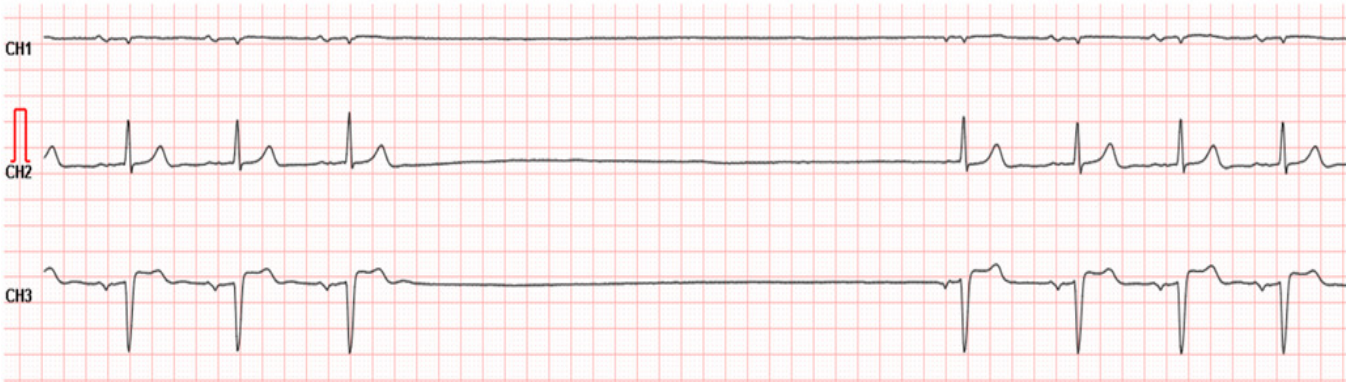

Figure 12: Sinus arrest with a pause lasting 5560 ms

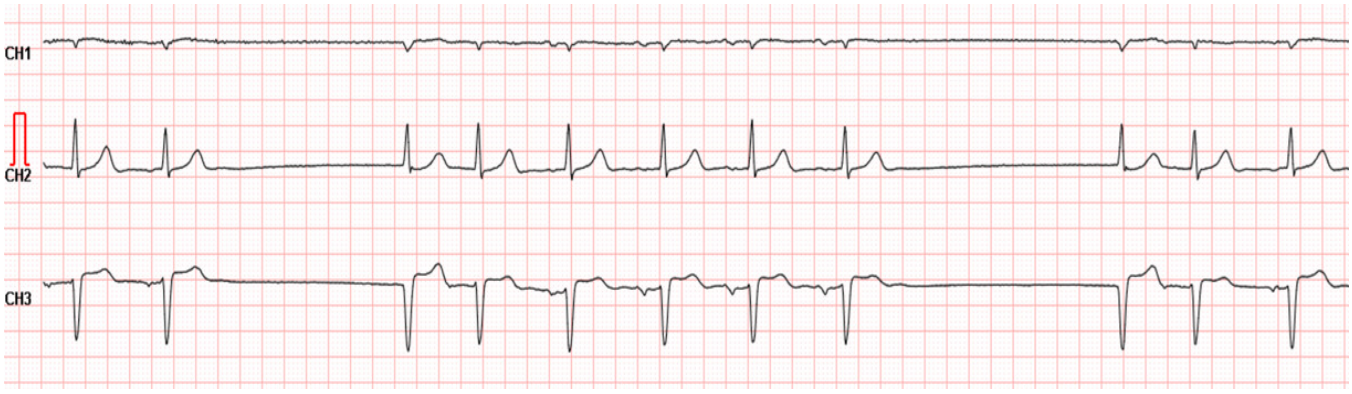

Figure 13: Two episodes of sinus arrest terminated by junctional escape beats

Note: The first two QRS complexes after each pause are not preceded by P waves, indicating a junctional origin.

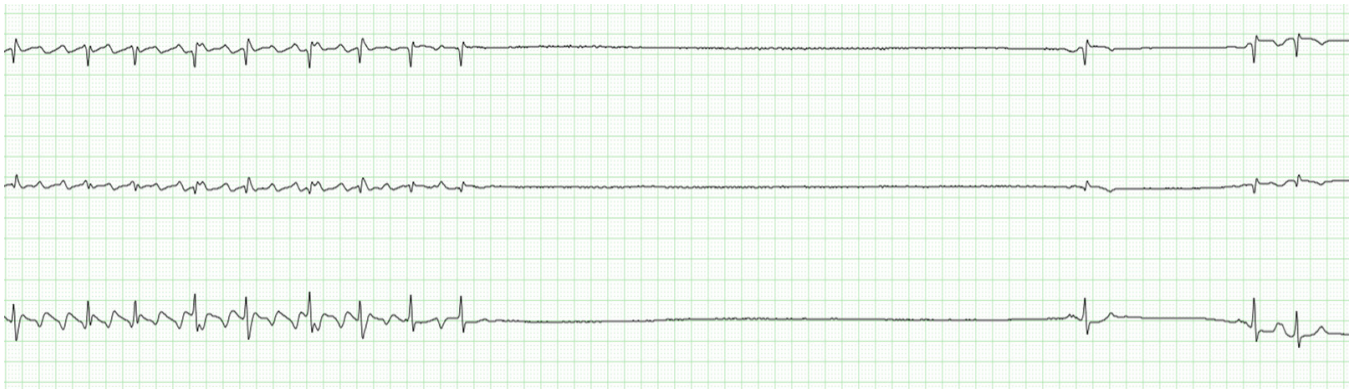

Figure 14: Conversion pause (7.5 s) after termination of atrial flutter, followed by sinus bradycardia and a premature atrial complex

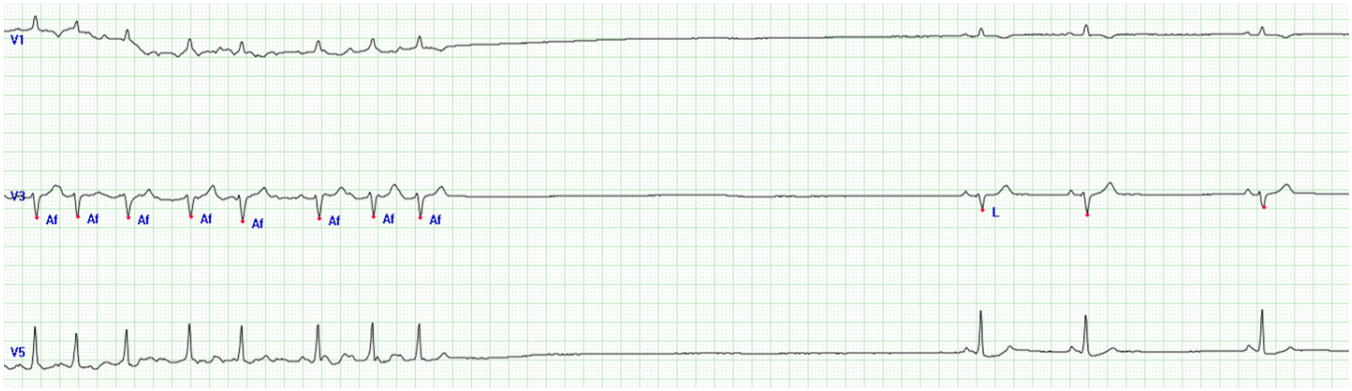

Figure 15: Conversion pause (6.0 s) after termination of atrial fibrillation, followed by sinus bradycardia

Exercise testing

Exercise testing is the most important diagnostic tool for diagnosing chronotropic incompetence. Chronotropic incompetence is defined as failure to achieve 85% of the age-predicted maximum heart rate or by a low chronotropic index (a heart rate response measure that accounts for effects of age and resting heart rate) [22]. Moreover, exercise testing is useful for myocardial ischemia diagnosis as a potential cause of SND.

Tilt table test

Sometimes syncope occurs very rarely and ECG during the episodes cannot be fixed by ambulatory ECG monitoring. Tilt table test enables to detect signs of SND that occurs rarely. It is the most important diagnostic method for vasovagal syncope. Except fall in blood pressure and temporally suppressed atrioventricular conduction as a cause of syncope, tilt table test allows to diagnose vagally mediated SN suppression. Positive (cardioinhibitory) response is defined as bradycardia $<$40 bpm lasting $>$10 s or asystole $>$3 s [23]. Sinus arrest, severe sinus

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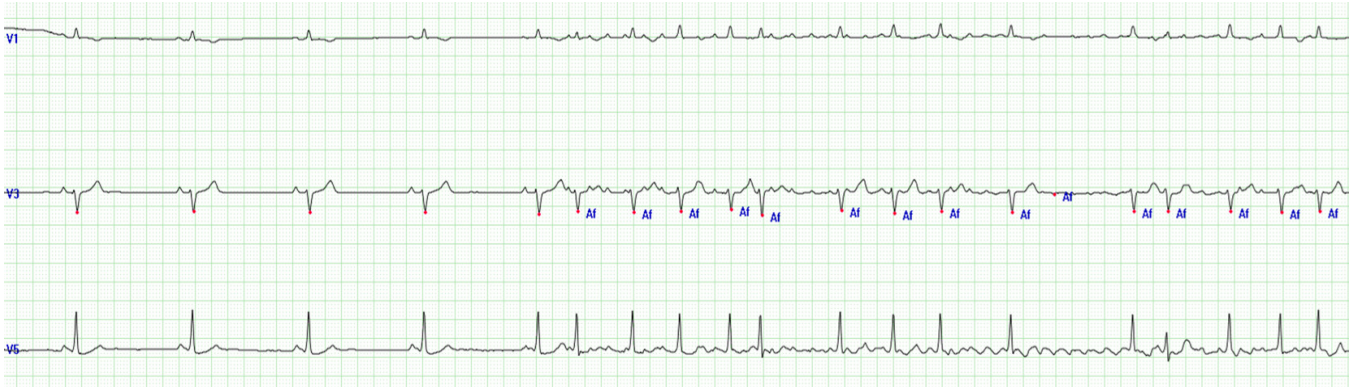

Figure 16: Onset of atrial fibrillation in the setting of sinus bradycardia with a heart rate of 42 bpm

bradycardia or junctional/idioventricular escape rhythms (fig. 17) are detected. Carotid sinus massage is used to test for carotid sinus syndrome. Its results are interpreted similarly to tilt table test.

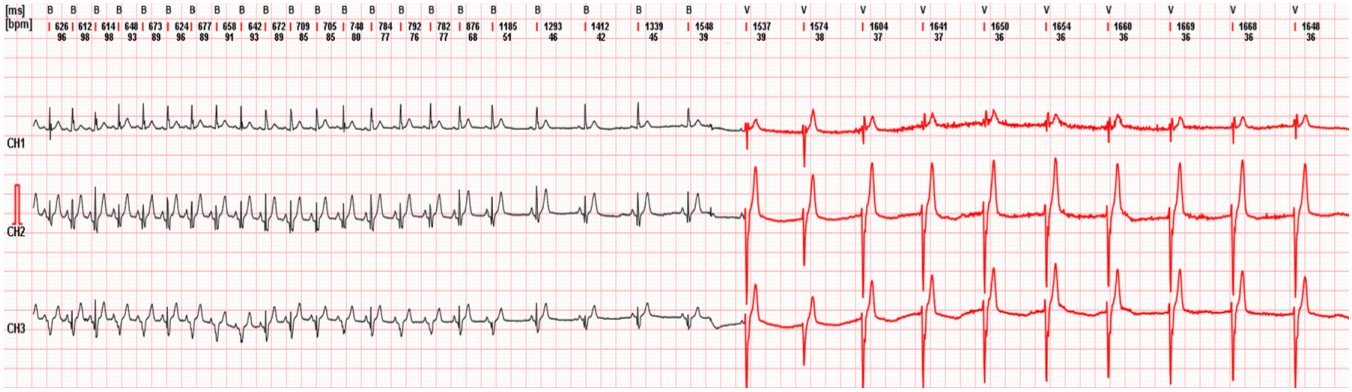

Figure 17: Transition to sinus bradycardia followed by an idioventricular escape rhythm with a heart rate of 37 bpm during a tilt table test

Note: There was a sudden decrease in heart rate. Initially, normal sinus rhythm transitioned to sinus bradycardia. As the bradycardia worsened and the heart rate decreased below 39 bpm, an idioventricular escape rhythm (red complexes) emerged.

Electrophysiological study

Measurement of SACT enables to find early abnormalities in conduction from the sinus node to the atrial myocardium. Prolonged SACT, also referred to as first-degree SA block, confirms SND. Moreover, electrophysiological study enables to measure sinus node recovery time (SNRT) which is defined as the interval from the last paced atrial beat to the first spontaneous sinus beat after rapid atrial pacing. Corrected SNRT (cSNRT) defined as SNRT minus the intrinsic sinus cycle length is considered more accurate in SND diagnosis. SACT >200 ms, SNRT >1500 ms, cSNRT >525 ms are considered to be abnormal [24,25]. Additionally, sinus pauses >3 s after atrial pacing is generally considered abnormal.

Other diagnostic methods

In patients with clinical suspicion for potential underlying causes of reversible bradycardia (potassium, magnesium, calcium levels, digitalis level, thyroid function tests, Lyme titers, and pH) are recommended [23]. In patients with suspected specific aetiologies (myocarditis, amyloidosis, sarcoidosis) associated with conduction abnormalities, cardiovascular magnetic resonance may be performed. Genetic testing for common genes associated with progressive cardiac conduction disease (SCN5A and TRPM4) may be performed in young individuals.

Management

Asymptomatic sinus bradycardia and sinus pauses <2.5 s may occur in healthy individuals, especially athletes, and do not require treatment. Transient causes of SND should be corrected and prevented. If there is no transient cause of symptomatic SND, pacing is recommended. DDD pacemaker is preferred over AAI pacemaker, except for young age and limited venous access [23]. In the case of chronotropic incompetence, pacemaker with rate-responsive pacing is preferred. In the case of conversion pauses, pacemaker are often implanted, although ablation of atrial fibrillation/flutter or supraventricular tachycardia may be an option [23].

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