Sinus Node Dysfunction

Sinus node dysfunction refers to a number of conditions causing physiologically inappropriate atrial rates. Symptoms may be minimal or include weakness, palpitations, and syncope. Diagnosis is by ECG. Symptomatic patients require a pacemaker.

Sinus node dysfunction includes inappropriate sinus bradycardia, alternating bradycardia and atrial tachyarrhythmias (bradycardia-tachycardia syndrome), sinus pause or arrest, and sinoatrial (SA) exit block. Sinus node dysfunction affects mainly the elderly, especially those with another cardiac disorder or diabetes.

Aetiology

- Primary SA node dysfunction
  - In elderly persons - ischaemic heart disease, scleroderma
  - In younger persons - cardiomyopathy, myocarditis, amyloidosis, collagen disease, surgery for congenital heart disease.
- Secondary SA node dysfunction
  - Drugs: Digitalis, beta-blockers, quinidine, procainamide, amiodarone, sotalal, diltiazem, verapamil, centrally acting sympatholytic antihypertensives, lithium

Pathology

In patients with primary SA node dysfunction, rest of the conduction system of heart e.g. intra-atrial conduction, A-V node, HIS bundle, bundle branches, fascicles and purkinjee system are usually simultaneously involved.

ECG- manifestations of sinus node dysfunction

**Failure of impulse generation**

Sinus bradycardia

P-wave rate of less than 60/minute is called sinus bradycardia (Fig. 1a,b,c). However, a rate of less than 40/minute is certainly abnormal and needs further evaluation and correlation with symptoms. Sinus bradycardia may be persistent or episodic.

Wandering pacemaker

Because of inability of SA node to maintain adequate rate, the pacemaker site gradually shifts from SA node, across atrium to AV junction and gradually back to SA node (Fig. 2).

Fig. 1(a): Lead II showing sinus bradycardia
(b): Lead V5 showing sinus bradycardia with low voltage of QRS and T wave in a case of myxoedema
(c): Lead III showing sinus bradycardia in a case of inferior myocardial infarction

Fig. 2: Lead II showing sinus bradycardia with wandering pacemaker. First two beats show sinus rhythm. 3rd, 4th and 5th beats are junctional escape beats (EB) with short PR interval because of delayed P wave. Sixth, 7th and 8th beats are again sinus beats

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**Chronotopic incompetence**
Inadequate increase in sinus rate during conditions like fever, injection of atropine and exercise (Fig. 3)

**Sinus pause**
Irregular pauses or absence of P wave which are longer than regular P-P interval but are not exactly multiple of regular sinus cycle length (P-P interval) (Fig. 4a, 4b). This results in sinus arrhythmia in presence of sinus bradycardia. This type of sinus arrhythmia is not related to respiration (non-phasic sinus arrhythmia). Long sinus pauses can cause syncope. Sinus pause needs to be differentiated from pause produced by blocked atrial ectopic (Fig. 4c). Preceding T-wave should be analysed carefully for deformation by the blocked atrial ectopic. A sinus pause of more than three seconds needs careful evaluation and correlation with symptoms.

- Increased sensitivity to suppression of impulse formation from drugs mentioned above (Fig. 5)
- Long pause before appearance of sinus rhythm following
  - Supraventricular ectopic (Fig. 6a)
  - AV-nodal or ventricular ectopics with retrograde depolarisation of SA node (Fig. 6b)
  - Electric cardioversion for atrial flutter or fibrillation
  - Termination of atrio-ventricular nodal reentrant tachycardia by verapamil/diltiazem (Fig. 6c)

**Failure of impulse transmission to atria (SA exit block)**
SA-node consists of impulse generating cells in the centre, surrounded by conducting cells which conduct the impulse out to atrial muscle and internodal pathways. Involvement of these cells results in SA exit block. (Fig. 7) Like AV-block, SA-block can also be first degree, second degree or complete SA-block.

First degree and complete SA exit block can not be

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**Fig. 3:** Stress test recording showing failure of increase in heart rate from rest to stage-3

**Fig. 4(a):** Ladder diagram showing genesis of ECG in sinus pause
(b) Lead II showing long sinus pause. It also shows prolonged PR interval due to concomitant AV nodal disease and broad QRS due to intraventricular conduction defect (Panconductance defect)
(c): Lead V1 showing a sharp deflection in the T wave of second beat due to a blocked atrial ectopic (AE) followed by a pause. This pause should not be confused with a sinus pause

**Fig. 5:** Lead II showing marked sinus bradycardia following 10mg TDS of propranolol for control of anxiety
diagnosed by surface ECG (Fig. 7) Second degree SA-block with Wenckebach phenomenon produces progressive shortening of PP interval followed by absence of one P wave. The pause is less than double of previous P-P cycle (Fig 8a). Like AV-block, Wenckebach type SA-block can also produce bigeminy (Fig. 8b).

Second degree exit block (Mobitz type II) results in regular PP-interval with absence of one or more P-waves. The pause so produced is exactly multiple of regular PP-cycle (Fig 8c)

In patient with concomitant sinus arrhythmia, differentiation between sinus pause and sino-atrial exit block may be difficult.

- **Concomitant intra-atrial conduction defect**
  - Abnormal P-wave configuration- wide and notched or deformed P-wave. Shape of P-wave may change because of varying intra-atrial conduction (Fig 9a)
  - No or slow atrial escape rhythm during significant sinus bradyarrhythmia
  - Persistent atrial-stand-still; there is no spontaneous atrial activity. ECG shows junctional bradycardia without any atrial activity(Fig 9b). Such patients have high risk of thromboembolism. It may be difficult to differentiate atrial-stand-still from fine atrial fibrillation.

Fig. 8(a): Lead II showing type I second degree SA exit block. There is progressive shortening of PP-interval followed by a pause which is less than twice of preceding PP cycle
(b): Lead II showing bigeminy produced by type I second degree SA exit block. It also shows left anterior hemiblock
(c): Lead II showing type 2-second degree SA exit block. The pause produced by the dropped P wave is double of preceding PP cycle
- Persistent atrial fibrillation, atrial flutter or atrial tachycardia with slow ventricular rate due to concomitant AV nodal disease (Fig 9d,e).

- Inadequate subsidiary pacemaker activity
  Normally a sinus pause of 1 to 2 seconds results in escape beat from a lower site capable of impulse formation. These escape beats can be atrial, AV junctional or ventricular. Failure of timely generation of escape beats from these subsidiary pacemaker sites occur due to simultaneous involvement of the rest of the conduction system of heart. Pause produced by failure of the lower pacemaker sites to produce escape beats can result in prolonged asystole with syncope.

- Escape-capture bigeminy: Escape beat may be followed by a sinus beat if next P-wave falls after T-wave of escape beat. Depending on sinus rate and rhythm the sinus beat and escape beat may alternate producing escape-capture bigeminy. (Fig 10)

- Concomitant AV block: This may be first degree, second degree or third degree. AV block may be permanent or may manifest following a premature beat (Fig. 4b, 6a).

- Concomitant infra-HIS conduction defects (Fig. 4b, 6a, 8b)
  - Left anterior hemiblock
  - Right bundle branch block
  - Left bundle branch block

- Repolarisation changes
  - Bradycardia may be accompanied by prolonged QT (Fig. 11) and susceptibility to bradycardia dependent ventricular tachyarrhythmia.

References