ECG Quiz 50

CASSA
CARDIAC ARRHYTHMIA SOCIETY OF SOUTHERN AFRICA
A SPECIAL INTEREST GROUP OF SA HEART ASSOCIATION

ANSWER

OVERVIEW OF THE ECG

The rate is slow (54bpm) and the QRS complexes are slightly irregular. The QRS complexes are narrow (90 - 100ms).

MORE DETAILED ANALYSIS OF THE ECG

There is a P wave before each QRS complex with a fixed short PR interval of 80 - 90ms. The P wave axis is +60 degrees, compatible with a sinus node origin. An ectopic atrial bradycardia is therefore unlikely. The P waves are slightly irregular (sinus arrhythmia). All the P waves are conducted to the ventricles with a fixed short PR interval i.e. AV association is present – sinus bradycardia with isorhythmic AV dissociation and a junctional escape is excluded.

Closer inspection of the QRS morphology reveals a narrow QRS complex (90 - 100ms) with no delta wave (pre-excitation). Subtle degrees of pre-excitation can sometimes be seen in patients with a left sided accessory pathway because atrial activation reaches the AV node before the accessory pathway and activation of the ventricles occurs predominantly via the His Purkinje system. In this ECG, subtle pre-excitation is not seen in any of the leads which excludes a WPW pattern. In addition, shortening of the PR interval to this extent as a result of pre-excitation would result in significant widening of the QRS.

Atrio-fascicular bypass tracts are long accessory tracts that connect the right atrial free wall with the distal part of the right bundle branch. The tissue of these tracts has AV nodal properties and therefore they have conduction delays and display decremental conduction to increased atrial pacing. The PR interval is therefore normal in duration with no evidence of pre-excitation. Sinus arrhythmia with an atrio-fascicular pathway (Mahaim pathway) is excluded.

The best description of this ECG is therefore 4) Sinus arrhythmia with a short PR interval (Lown-Ganong-Levine pattern).

DISCUSSION

A normal PR interval is 120 - 200ms. This interval reflects conduction from the high right atrium (onset of P wave) to the point of earliest ventricular depolarisation (onset of the QRS).

When catheters are placed in the heart, different intervals that comprise the PR interval on an ECG can be measured (Figure 1):

- PA interval (i.e. onset of P wave to the atrial signal in the His catheter) or intra-atrial conduction time. This interval is constant (20 - 60ms).
- AH interval (i.e. atrial signal in the His catheter to the His bundle signal) or AV nodal conduction time. This interval is highly variable, depending on autonomic tone, and has a wide range of 50 - 120ms.
- HV interval (i.e. His bundle signal to the onset of QRS) or time from the His bundle to the ventricular myocardium. This interval is constant (35 - 55ms).

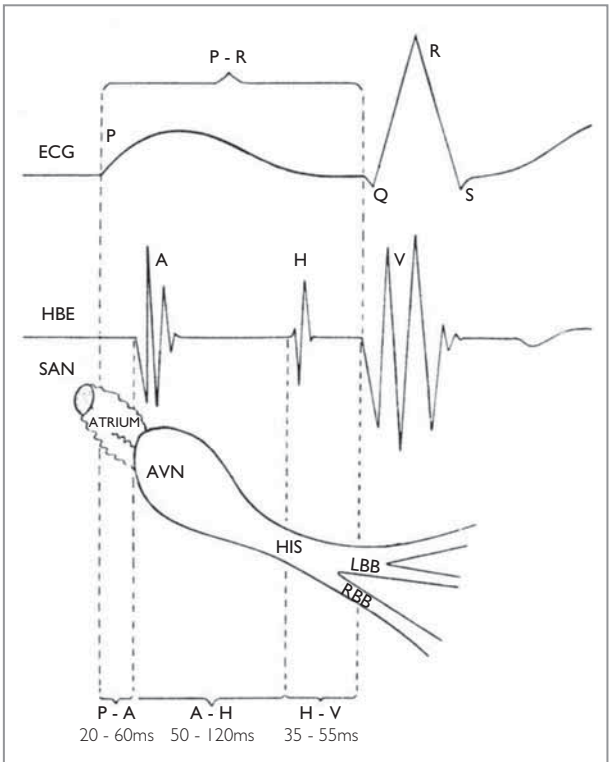

FIGURE 1: Schematic diagram showing a PR interval comprises PA, AH and HV intervals (see text for details).

HBE = His bundle electrogram, A = Atrial electrogram, H = His bundle electrogram, V = Ventricular electrogram, SAN = Sinoatrial node, AVN= AV node, LBB = Left bundle branch, RBB= Right bundle branch.

A short PR interval (<120ms) can have different mechanisms:

- Enhanced AV nodal conduction due to either a small AV node, enhanced physiological AV nodal conduction or enhanced AV nodal conduction due to specialised intra-nodal, atrio-nodal or atrio-Hisian fibres. Features include a short PR interval (short AH interval) with a normal narrow QRS complex. This pattern has become known as the Lown-Ganong-Levine pattern.
- Ectopic atrial rhythm arising near the AV node. Features include an abnormal P wave axis with a short PR interval (short PA interval).
- Isorhythmic AV dissociation (note: P waves are not conducted to the ventricles because a junctional rhythm has depolarised the His-Purkinje system before the P wave was allowed to conduct). Features include variation in the PR intervals with a regular, junctional rhythm.
- Atrioventricular bypass tract (WPW pattern). Features include a short PR interval (short HV interval which sometimes can be negative) with a delta wave and a wide QRS complex.

In 1952, Bernard Lown, William Ganong and Samuel Levine reported a series of patients with short PR intervals and normal QRS complexes with 11% of the patients presenting with a supraventricular tachycardia which became known as the Lown-Ganong-Levine syndrome.[1] These patients were mostly female, without structural heart disease and with snapping first heart sounds (due to the short PR). This pattern of a short PR interval and a normal QRS complex was purely descriptive. Since that ECG description, anatomical and physiological theories have been put forward to explain possible electrophysiological mechanisms of enhanced AV nodal conduction:

- Enhanced AV nodal conduction may be physiological ("slick AV node") and may represent one end of a normal spectrum of normal AV nodal function. This can be seen in patients with anatomically small AV nodes.
- Intra-nodal or atrio-nodal fibres (James fibres). Such fibres bypass the transitional portion of the AV node which is largely responsible for AV conduction delay (Figure 2).
- Atrio-Hisian fibres (Breckenmacher fibres). Such fibres can bypass the AV node completely (Figure 2).

While the first 2 mechanisms may have overlapping electrophysiological features and cannot be distinguished on the 12 lead ECG, features that may suggest enhanced "physiological" AV nodal conduction at EP study include: (1) rapid 1:1 AV conduction with atrial pacing <300ms and (2) AH prolongation is smooth and blunted with a maximal increase in the AH interval <=100ms during atrial pacing at 300ms when compared to sinus rhythm.[2] (This distinction is of interest only as distinguishing the 2 entities is clinically unimportant as management remains conservative for both conditions).

Intra-nodal and atrio-nodal fibres are not directly able to sustain re-entry tachycardias. AV nodal re-entry tachycardia (AVNRT) and orthodromic atrioventricular re-entry tachycardia (AVRT) have indirectly been associated with these patients and, interestingly, the incidence of these arrhythmias is higher compared to patients with a normal PR interval.

Atrio-Hisian fibres are very rare and can be distinguished by a constant pacing spike to QRS complex interval during atrial extra-stimuli because the AV node is bypassed and decremental conduction cannot occur with decremental atrial pacing. Patients with atrio-Hisian fibres are at risk of sudden death as

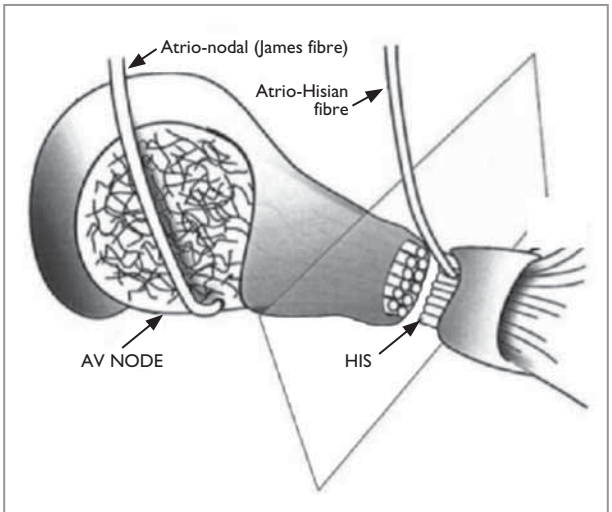

FIGURE 2: Schematic diagram of the AV node depicting an atrio-nodal fibre (connecting the atrium to the distal AV node) and an atrio-Hisian fibre (connecting the atrium to the His bundle).
Adapted from CJ Brechenmacher.[3]

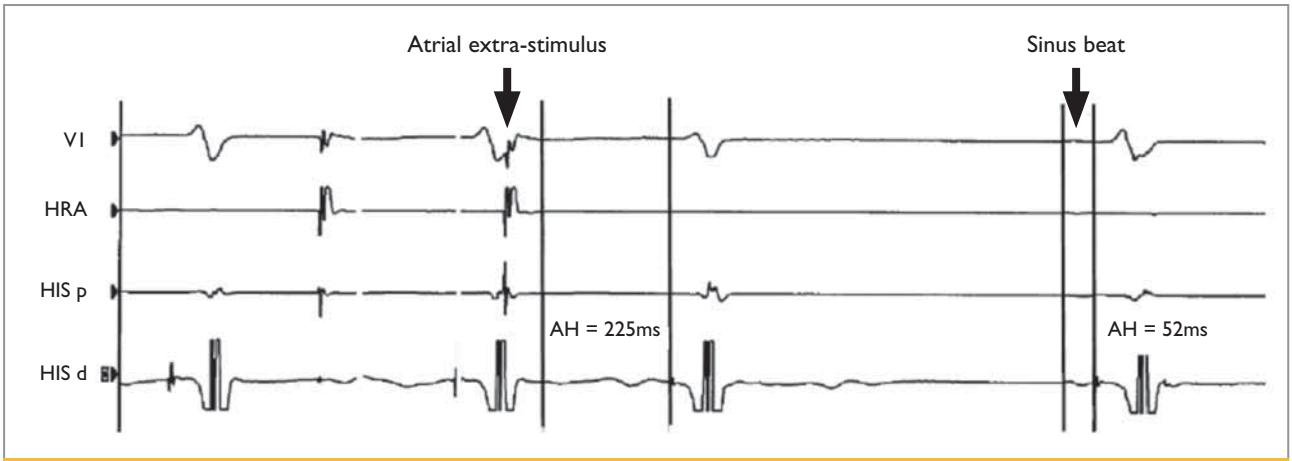

FIGURE 3: Intracardiac electrocardiograms showing HRA (high right atrium), HIS p (proximal electrode) and His d (distal electrode) electrocardiograms with V1 ECG lead. The sinus beat has a very short AH interval of 52ms. Single atrial extrastimulus results in marked prolongation of the AH interval (225ms). This excludes an atrio-Hisian fibre. This is compatible with enhanced physiological AV nodal conduction.

rapid ventricular rates can occur during atrial fibrillation or atrial flutter (analogous to a rapidly conducting AV accessory pathway). These high risk patients may require His bundle ablation and insertion of a pacemaker.[3]

An electrophysiological study was performed in view of her palpitations and to exclude an Atrio-Hisian fribre. She had a very short AH interval of 52ms and a normal HV interval of 35ms. Single atrial extra-stimuli resulted in prolongation of the AH interval (to 225ms) which excluded the presence of an atrio-Hisian fibre (Figure 3). She had 1:1 AV conduction with atrial pacing at 300ms with a maximal increase in the AH time of <=100ms (not shown). This suggests a diagnosis of enhanced physiological AV nodal conduction. She had no inducible arrhythmias and no ablations were performed.

Conflict of interest: none declared.

REFERENCES

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3. Brechenmacher CJ. Atrio-hisian fibers anatomy and electrophysiology. Pacing Clin Electrophysiol. 2013;36(2):137-41.

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