Another AV Block?
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Keywords: AV dissociation; Hisian extrasystoles; Phase 4 block

A 66 year old woman with history of phenytoin-controlled seizures and hypothyroidism was admitted after a syncopal attack. Whilst monitored as an inpatient, she suffered another syncopal episode. Telemetry revealed this to be associated with AV dissociation (Figure 1) that lasted for 20 seconds. Thus dual chamber pacemaker was implanted and the patient had no further symptoms at 6 months follow up.

What is the mechanism of the paroxysmal AV dissociation?

Close inspection of the monitor strip allows speculation as to the possible mechanisms:

(i) The P-P interval prior to the pause remains constant with no progressive prolongation, thus making a vagal reaction unlikely; (ii) the PR interval preceding the pause is constant; (iii) preceding the pause, there is a premature atrial contraction (Figure 1, black arrow). The negative P-wave in lead II suggests caudal-cranial atrial activation, thus it is possible that this beat penetrates the AV node making it refractory to the next sinus beat. But where does this beat originate from? One speculation is that a hisian extrasystole was able to depolarize retrogradely both the AV node and the atrium [1], and that subsequent concealed hisian extrasystoles were unable to activate the atrium but maintained the AV node refractoriness preventing conduction of the subsequent sinus beats resulting in the 20s episode of AV dissociation. This mechanism is known as interference AV dissociation [2]. Other possible origins for this beat include the ostium of the coronary sinus and the low atrium region. An alternative explanation for this phenomenon is Phase 4 Block due to the atrial premature beat. The P-P interval (and thus the input interval to the distal conduction system) is longer post-premature atrial beat, allowing Phase 4 block to occur. However, Phase 3 and 4 pathophysiological mechanisms as the cause of paroxysmal AV block have not been validated using Purkinje fiber models [3].

References

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Received June 06, 2013; Accepted June 20, 2013; Published June 24, 2013


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