What Is the Mechanism of Group Beating?

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Case report

An eighty-year-old male was referred to our center because of dizziness and palpitation for the previous two weeks. He had no history of syncope and the drug history was unremarkable. Figure 1 shows the resting ECG, interpreted as complete heart block by the referring physician. A transthoracic echocardiography was done and showed a left ventricular ejection fraction of 35% and a severely calcified mitral valve with an estimated mitral valve orifice area of 1.4 cm^2.

Group beating was observed on the ECG, and a closer look at the ECG suggested the diagnosis of sino-atrial node Wenckebach (SANW) exit block. Premature atrial beat trigeminy could have also explained the group beating of the P-waves. However, the similarity of the P-wave morphologies and the confirmation of the diagnosis of sick sinus syndrome by electrophysiology study made this diagnosis unlikely and suggested the SANW as the cause of P-wave group beating.

Apparently there seems to be a conduction defect at the atrio-ventricular nodal (AVN) level. The PR interval of the first conducted P-wave is longer (first black arrow) than the subsequent PR interval, which is then followed by a non-conducted P-wave. Nevertheless, a close look at the ECG reveals the real underlying mechanism of apparent AV block. Due to SANW, the P-P intervals gradually shorten and the third P-wave is blocked as it encounters the *physiologic* refractory period of the AVN. After the third P-wave, there is a pause due to SA exit block (4^{th} P-wave, blocked at the SAN level) followed by a conducted P-wave with a long PR interval. This phenomenon could be best explained by phase-four (bradycardia-dependent) conduction blocks.

Electrocardiographic changes in this patient could also be explained by the effect of cycle-length duration on infra-Hissian conduction. Thus, PR interval prolongation can be due to His-Purkinje refractoriness in the longer cycle length. The shorter PR interval in the next beat may be due to a decrease in the HV interval in the shorter cycle length. In this scenario, the last P-wave would be blocked in the AV node ***(Iranian Heart Journal 2006; 7 (2):42-43)***.

Disussion

Bradycardia-dependent conduction block at AVN level usually occurs when the heart rate falls below a critical level. It may be caused by abnormal phase-four depolarization of cells in the AVN, hence the activation occurs at less negative resting membrane potentials, leading to

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conduction delay and/or block.[1-4]
The phase-four conduction block is usually seen in patients with preexisting conduction system disease, like our case who had preexisting left bundle branch block (Fig. 1). Finally, it is worth mentioning that in cases of periodic group beating and/or conduction block, respiration-dependent vagal tone modulation and its effect on the conduction properties of SA and AV nodes should be considered as a possible underlying mechanism of apparent conduction delay and/or block. [3]

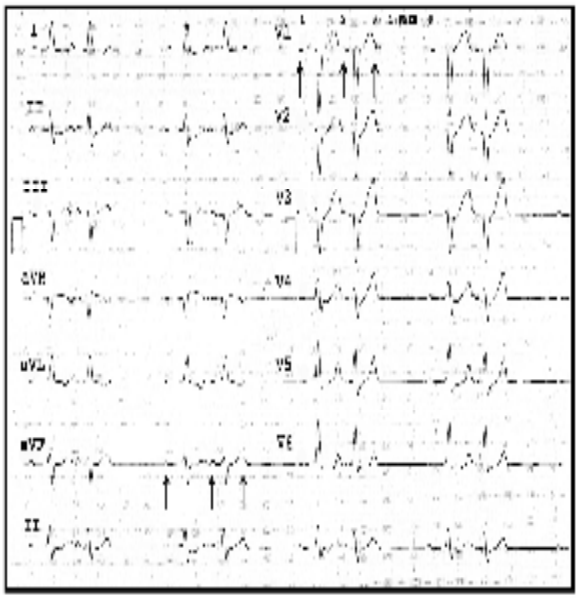

Fig. 1. 12-lead surface electrocardiogram of the patient. Note the P-waves marked by black arrows. SAEB: Sino-atrial nodal exit block.

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