INTERESTING ELECTROCARDIOGRAM

COMPLEX CASE STUDY

Unexpected Recovery of 1:1 Atrioventricular Conduction in Advanced AV block: What’s the Mechanism?

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ABSTRACT. In this report we discuss a case of unexpected 1:1 conduction in a patient with high-degree atrioventricular block. The concept of supernormal conduction as a possible mechanism is reviewed.

KEYWORDS. infranhisian block, supernormal conduction.

Introduction

Unexpected paroxysmal recovery of 1:1 conduction in patients with advanced atrioventricular (AV) block can occur under specific circumstances. In this article we report a case of advanced infranhisian AV block with such recovery of 1:1 AV propagation during high-rate atrial pacing which was followed by repetitive 3:1 and 4:1 blocks on continued pacing. What’s the mechanism?

Case report

A 76-year-old male was referred to our center with dyspnea and AV block with wide QRS complexes. His medical history included smoking and arterial hypertension, which was being treated with losartan and amlodipine. Echocardiogram revealed moderate left atrial dilatation, normal left ventricular systolic function and dimensions, a calcified posterior mitral annulus with normal valve function, and calcified aortic valve with moderate aortic stenosis. His baseline electrocardiogram (ECG) showed advanced AV block with wide QRS complexes. The irregular RR intervals occur due to the presence of a junctional rhythm with intermittent delayed ventricular captures, both showing right bundle branch block and left anterior fascicular block morphology (Figure 1). This reveals a delayed recovery of excitability in the AV conduction system.

During the intravenous isoproterenol infusion (4 μg/min), 3:2 and 4:3 infranhisian Mobitz II type AV block were observed (Figure 2a,b) with the His–Purkinje refractory period estimated at 535–545 ms. Despite this, rapid atrial pacing at a cycle length of 360 ms paradoxically resulted in transient restoration of 1:1 AV conduction followed by 3:1 and 4:1 infranhisian AV block (Figure 3a–c). This phenomenon was reproducible. These unexpected changes may be explained by supernormal conduction (SNC) in the damaged left posterior fascicle that until then was maintaining the intermittent AV propagation. Interestingly, during ventricular pacing there was 1:1 ventriculoatrial conduction at a cycle length of 450 ms (not shown). A dual-chamber pacemaker was implanted in this patient.

Comments

SNC has been defined as the condition under which impulse propagation improves in relation to a supernormal...
Figure 1: High-degree atrioventricular block with late ventricular captures. The shortest conducted RP interval measures 1125 ms (RR interval: 1245 ms) and the longest non-conducted RP interval measures 1015 ms (fourth RR interval). This denotes a delayed recovery of excitability in the AV conduction system. Note that the conducted beats show right bundle branch block plus left anterior fascicular block morphology.

Figure 2: (a) 3:2 and 4:3 Mobitz II type AV block during intravenous isoproterenol infusion. The PP intervals measure between 535 and 545 ms. (b) Simultaneous recording of leads I, II, III, V1, and V6 and the high-right atrial (HRA) and His bundle (HBE) electrograms during 3:2 and 4:3 periodicities shown in Figure 1a. Arrows point to the His bundle potentials.
Unrefractoriness can be excluded. For instance, a “gap” phenomenon, due to either AV nodal or intrahisian refractoriness can be discarded as a premature ventricular depolarization did not occur prior to the recovery of 1:1 AV conduction. The 1:1 AV conduction persisted only for a few seconds before 3:1 and 4:1 AV block ensued. The 3:1 AV block may be explained by partial penetration of the left posteroirinferior fascicle by the SNC phase impulse in the first blocked P wave, which increased the refractory period leading to total block of the second P wave. This allowed total recovery of excitability with AV propagation of the next P wave. A similar sequence, but with concealed conduction of two successive P waves and total block of the third P wave could account for the 4:1 AV block.

One-to-one impulse propagation attributable to SNC is not often identified, as this necessitates a specific search to be performed and a particular set of conditions to occur. In this way the SNC phase behaves as an elusive, dynamic target that varies as the atrial cycle length changes. Normally, when the atrial cycle length equals that of the SNC phase, impulse propagation follows a 2:1 conduction pattern. For 1:1 SNC to occur, prior adrenergic stimulation must precede the appropriate cycle length shortening.

In the case reported here, the His–Purkinje system was severely damaged with Mobitz II type block being unmasked by isoproterenol infusion. Under these conditions, the periodical infrahisian block (cycle lengths between 535 and 545 ms) denoted a clear prolongation of refractoriness in the posterior division of the left bundle branch. However, 1:1 AV propagation was observed at a much shorter cycle length (360 ms) induced by atrial pacing, which may be explained by the presence of a SNC phase in the ventricular fascicle maintaining the AV conduction. Alternative mechanisms that could simulate SNC such as “gap” phenomenon or “peel back” refractoriness can be excluded. For instance, a “gap” phenomenon, due to either AV nodal or intrahisian conduction delay during rapid atrial pacing, would require extremely slow propagation of the impulses so that they reach the zone of intermittent block in the posterior division of the left bundle branch after the end of its prolonged refractory period. By necessity this would involve the longer RR intervals of 535–545 ms rather than those of 360 ms observed during atrial pacing. Similarly, “peel back” refractoriness can be discarded as a premature ventricular depolarization did not occur prior to the recovery of 1:1 AV conduction.

The peculiar behavior of conduction described in this patient is certainly uncommon, but not exceptional. However, it requires the presence of SNC to be appropriately investigated with careful scanning of the cardiac cycle and the accurate exclusion of pseudo-SNC. Indeed, Tavazzi et al. uncovered SNC by programmed atrial pacing in 25.6% of intermittent bundle branch block (n=15) and Halpern et al. found SNC in 45 of 47 cases of Mobitz II AV block.

References

Figure 3: (a) Surface 12-lead electrocardiogram. Transient 1:1 atrioventricular (AV) conduction followed by repetitive 3:1 and 4:1 AV block during atrial pacing (AP) at a cycle length (CL) of 360 ms. (b) Surface electrocardiogram and His bundle recording (HBE) during initiation of 1:1 AV conduction induced by rapid atrial pacing. The first paced beat, with a short HH interval that should be blocked (according to the preceding conditions) can propagate to the ventricles and the same occurs in the subsequent paced beats in spite of the brief cycle length (360 ms). Arrows point to His bundle potentials. (c) 1:1 AV conduction shown in (b) was followed by 3:1 and 4:1 infrahisian AV block without any change in atrial cycle length and HH intervals. The different AV ratios may be explained by concealed conduction in the posteroinferior fascicle of the left bundle branch. Arrows point to His bundle potentials.
