Unexpected Atrioventricular Conduction in High-Grade Atrioventricular Block

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A 90-year-old man with a history of high blood pressure, a cerebrovascular accident without focal residua, dementia, and stage 3 chronic kidney disease went to the emergency department because of dizziness and near syncope. His medications were aspirin 81 mg qd, clopidogrel 75 mg qod, escitalopram oxalate 10 mg qd, Seroquel 25 mg qd, and memantine hydrochloride 10 mg qd. He had orthostatic hypotension with supine blood pressure of 173/77 mm Hg falling to 116/68 on standing, while pulse increased from 66 to 84 beats/min. He received IV fluid and returned home. Two days later he saw his primary care physician because of episodes of dizziness and confusion. The figure shows an electrocardiogram recorded during that visit.

Figure: Outpatient electrocardiogram recorded in a 90-year-old man with episodes of dizziness and confusion. See text for explication.

What is your diagnosis?

Explication is on p. 98
ECG of the Month
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DIAGNOSIS: Sinus rhythm; high-grade second degree atrioventricular block with a junctional escape rhythm and three capture complexes, each with right bundle branch block aberration; possible septal myocardial infarct of indeterminate age; ST-T and U wave changes suggesting hypokalemia.

In most cases of second degree atrioventricular block, the longer the R-P interval, the more likely is the P wave to be conducted to the ventricles. Thus, in this ECG the 2nd, 6th, 10th, and 12th P waves, with R-P intervals of 0.67 to 0.81 seconds, would be the most likely ones to be conducted, and the 4th and 8th P waves, with R-P intervals of 0.42 to 0.50 seconds, also would have a good chance to be conducted. None of them is. Instead, the 3rd, 7th, and 13th P waves with much shorter R-P intervals, 0.19 to 0.23 seconds, are conducted. The 1st, 5th, 9th, and 11th P waves either occur simultaneously with a junction-initiated QRS or 0.08 seconds after one and are not conducted. Thus, there appears to be only a short window of time soon after junction-initiated QRs when atrioventricular conduction can occur. This seeming paradox requires explanation.

In times past, supernormality often has been invoked to explain unexpected excitability or conductivity in cardiac tissue. Pick et al defined the supernormal phase as “…a short, early, and limited period of the cardiac cycle during which a stimulus elicits either a totally unexpected response or one that is less abnormal than expected considering the state of recovery from the preceding impulse.” They then stated that they had collected 18 cases demonstrating a supernormal phase of atrioventricular conduction in the preceding 5 years in addition to 10 cases that they had described previously. All were cases of advanced atrioventricular block, and the authors concluded, “…supernormal AV conduction is an abnormal phenomenon encountered only in the presence of AV block—in other words, only when the absolute or relative refractory period of the conduction system is unduly prolonged may a phase of supernormality become manifest.” In reviewing the previous literature, including the 10 cases they reported earlier, but none of those in their current publication, they decided “…that a supernormal phase was definitely in action in 32 instances...In 14 additional ones alternative interpretation appears possible…” Alternative explanations included vagal effects, second degree AV block with escape complexes, dual AV nodal pathways, and “…a prolonged rest period in the upper AV junction following retrograde conduction.” Their final conclusion stated, “The application of the concept of supernormal conduction in conjunction with that of concealed conduction and of unidirectional block permits a satisfactory interpretation of some otherwise inexplicable features of AV block encountered in clinical electrocardiography.”

Six years after the paper of Pick et al, Moe and colleagues wrote a seminal paper demonstrating that in most, if not all, published cases of supernormality of AV conduction alternative mechanisms could explain the observed phenomena. They divided the alternative mechanisms into three main classes: “…(1) occult 2:1 A-V block in which an idioventricular beat “retracts” an otherwise refractory bar- rier within the A-V node; (2) alternation between dissociated intranodal transmission pathways; and (3) “ventriculo- phasic” (vagal) depression of nodal conductivity.”

Although there is now general agreement that true supernormal conduction does not occur in the AV node, supernormal conduction has been demonstrated in the bundle branch-Purkinje system of the dog. Furthermore, Spear and Moore have demonstrated supernormal conduction in the distal bundle of His and proximal bundle branches, but they could not demonstrate it in the proximal portion of the bundle of His. They ascribed this difference to the relatively low transmembrane resting potential of the proximal bundle of His, similar to that in the AV node, whereas the electrophysiological properties of the distal bundle of His are similar to those in bundle branches.

 Supernormal AV conduction allowing transmission of P waves with R-P intervals of 0.19 to 0.23 s, but not those with shorter or longer R-P intervals, would explain the unexpected AV conduction in our patient. If, as described above, supernormality does not occur in the AV node, could supernormality in the distal His bundle following His-initiated complexes of the escape rhythm explain the unexpected conduction? In fact, Moe et al theorize such a possibility in section F of their paper.

Whatever the fine points of the patient’s atrioventricular block, it was successfully treated with an electronic AV sequential pacemaker that captured 100 percent of the time, usually in the P-synchronous mode. None of the patient’s other ECG’s before the pacemaker implantation suggested a septal myocardial infarct as the one in the Figure did. Furthermore, his echocardiogram showed normal left ventricular function, but also showed moderate aortic stenosis.

REFERENCES

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