Ventricular Parasystole following Implantation of an Electronic Pacemaker Unit

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Since the introduction of electronic pacemakers, a number of previously unencountered arrhythmias are now seen frequently.1,2 Among those more commonly observed is artificial ventricular parasystole which occurs when normal A-V conduction is resumed in patients with fixed rate pacemaker units.3 Spontaneous ventricular parasystole is occasionally encountered in patients with supraventricular mechanisms.4 The subject of this report is a patient who demonstrated a spontaneously active left ventricular focus (ventricular parasystole) after insertion of a permanent transvenous demand pacemaker unit.

CASE REPORT

A previously healthy 64-year-old white man was admitted to a nearby hospital following the onset of dyspnea and dizziness. An electrocardiogram revealed complete heart block and a slow idioventricular rhythm with a rate of less than 20 beats per minute. After pharmacologic therapy, a heart rate of 40 beats per minute was achieved with clinical improvement. Serum enzyme studies did not indicate recent myocardial infarction. After six days, the patient was transferred to this facility where an electrocardiogram disclosed persistence of complete heart block (Fig 1). A temporary transvenous pacemaker was inserted. Observation over a two-week period of artificial pacing failed to disclose a return of normal A-V conduction. A permanent demand type transvenous pacemaker was then inserted and the rate adjusted to 68 per minute. On the sixth postoperative day, the pulse was noted to be irregular. An electrocardiogram revealed recurrent beats from an ectopic ventricular focus (ventricular parasystole) after insertion of a permanent transvenous demand pacemaker unit.

DISCUSSION

The electrocardiographic diagnosis of parasystole requires the demonstration of beats originating from two foci which initiate impulses at different rates independently of one another. Usually the dominant rhythm stems from the SA node while an ectopic focus in either the atria, ventricles or junctional tissue, independently initiates impulses, being protected by a uni-directional entrance block. To substantiate a parasystolic focus, it is necessary to demonstrate that the ectopic beats have an inconstant relationship to the dominant rhythm and a predictable relationship to one another.

In the present case these criteria are fulfilled. The dominant rhythm is the artificial pacemaker stimulus in the right ventricle while the ectopic foci have the same QRS configuration as the previous idioventricular rhythm of left ventricular origin. The R-R interval of least common denomination, representing the fundamental rate of the parasystolic focus, varies from 1.47 sec to 1.58 sec and is similar to that of the previous idioventricular rhythm.

A second possible explanation of the arrhythmia is atrioventricular nodal parasystole5 with aberrant ventricular conduction. Against this explanation is the slow fundamental rate of the parasystolic focus and the absence of retrograde atrial depolarization as evidenced by the consistent P-P interval of 0.86 sec.

The arrhythmia is further complicated by the demand function of the pacemaker unit, which allows a 0.84 sec pause following each beat from the parasystolic focus. In effect the dominant rhythm is not protected by entrance block as is the parasystolic focus.

FIGURE 1. Electrocardiographic tracing from lead II. There is complete heart block with an idioventricular rhythm. The average R-R interval is 1.49 sec.
Not all ectopic impulses are conducted to the remainder of the heart (exit block). Although the interval between pacemaker impulses is 0.91 sec, only those impulses that occur from 0.46 sec to 0.69 sec after the preceding pacemaker impulse are effective in depolarizing the heart. This can be explained by a phase of super-normal excitability following the pacemaker beats which has been well demonstrated in artificial pacing of the heart. Since occasional impulses calculated to fall in this period are not conducted, there may exist a beat to beat variation in the phase of super normal excitability or an intermittent exit block. Of more significance, however, is the repeated failure of conduction after the super normal period.

REFERENCES

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