a mass visible in roentgenograms. In this case the surgical and radiologic observations indicate that the lobe receiving a major dual arterial supply was normal. The involved systemic artery arose from the lower descending aorta and was considered to be other than a standard bronchial artery.

Under the circumstances of our case with regular and assumed normality of the bronchopulmonary connections, one might consider that the anomalous artery was part of a bronchial arteriovenous fistula. There was no evidence for such a process. This leaves our case as a most unusual one in which an otherwise normal pulmonary lobe received a major dual arterial supply. The data at our disposal do not allow determination as to whether the termination of the systemic artery was into the pulmonary arterial or pulmonary capillary beds.

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Figure 4. Anomalous artery removed at operation. a. Low power (elastic tissue stain, original magnification × 15). b. Higher magnification shows evenly distributed layers of elastic tissue in media characteristic of elastic artery (elastic tissue stain original magnification × 210).

Rate-Dependent Premature Beats in Man

Herman O. Klein, M.D.

Ventricular premature beats (VPBs) appeared in a patient after pacemaker insertion for complete heart block secondary to acute myocardial infarction. Contrary to expectations, the frequency of VPBs was directly related to the basic pacemaker rate. The VPBs are either reentrant beats or represent VPBs arising from pacemaker cells with "slow-response" characteristics, which have been shown to become more automatic with increasing rates of electrical stimulation. This case documents the phenomenon of rate dependency of VPBs in man and discusses its practical importance.

Overdrive suppression1,2 is a well-known method of treatment for ventricular arrhythmias. It is distinctly unusual to observe instances in which ventricular premature beats (VPBs) are induced by a moderately rapid rate of pacing and are decreased or eliminated by a reduction in the rate of the pacemaker. This seemingly paradoxical response to electrical stimulation was first observed experimentally in digitalis toxicity3 and subsequently in isolated canine Purkinje fibers.4,5 This report illustrates its occurrence in man, demonstrating that it is of more than theoretic interest.

CASE REPORT

Transvenous endocardial pacing was instituted in a 50-year-old man with complete heart block following acute myocardial infarction. The ventricular response was 33 beats per minute; an atrioventricular junctional rhythm had been abolished. Two days after installation of the transvenous endocardial electrodes, the pacemaker was discontinued, and the patient was discharged with an atrial pacemaker and an atrioventricular junctional rhythm of 40 beats per minute. Two months later, while off all medication, a 24-hour EKG demonstrated a rate of 40 beats per minute with occasional VPBs. The VPBs were associated with occasional premature atrial contractions and paroxysmal atrial tachycardia. A month later, while on digitalis and parenteral fluids, VPBs were found to be a normal part of the 40-beat-per-minute ventricular response. The VPBs were abolished by a decrease in the pacemaker rate from 100 to 60 beats per minute. Soon after pacemaker rate reduction, digitalis was discontinued, and the patient was discharged with a ventricular response of 50 beats per minute and no VPBs. Overdrive suppression was instituted on readmission to the hospital. Several hours after the pacemaker rate was increased from 60 to 100 beats per minute, the patient had an unexplained sudden cardiac arrest. XP acardiac rhythm was restored by external cardiac massage and defibrillation. The atrial and ventricular responses were 80 beats per minute, and the VPBs had disappeared. No VPBs were observed while the pacemaker rate was increased or decreased. The patient was discharged with a ventricular response of 80 beats per minute and no VPBs.

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dial infarction. Previous electrocardiograms had shown left bundle-branch block.

The VPBs appeared after insertion of the pacemaker and increased in frequency with any increase in the pacemaker rate. When the basic stimulation (R-R) interval was 0.62 second (rate, 97/min), single unifocal VPBs occurred after each fourth to fifth paced beat, with a constant coupling interval varying by no more than 0.04 second. Decreasing the R-R interval to 0.59 second (rate, 102/min) only increased the frequency of the VPBs, which now occurred after each second to third paced beat. Increasing the R-R interval to 0.70 second (rate, 85/min) completely abolished the VPBs.

To prove that this unusual response to rate was not merely a chance happening, the same maneuvers were performed repeatedly over the course of one-half hour. During the entire period, slight variations in the rate of stimulation repeatedly produced a change in the frequency of VPBs, as illustrated in Figures 1 and 2. In Figure 1B, at a stimulating interval of 0.58 second (rate, 103/min), every third beat is followed by a VPB. At an interval in Figure 1A of 0.56 second (rate, 107/min), every second beat is now followed by a VPB. As the stimulating interval is increased to 0.63 second (rate, 96/min) and 0.64 second (rate, 94/min) in Figure 1C and 1D, respectively, the VPBs decrease in frequency. At an interval of 0.72 second (rate, 83/min) in Figure 1E, the VPBs are abolished.

The observations are plotted in a time graph in Figure 2. Increasing the R-R interval to 0.67 second initially and to 0.70 second or beyond subsequently always resulted in the elimination of the VPBs (Figure 2, points A, C to F, and H to...
VPBs. Thus, after a change in pacing rate, returning to the original rate did not necessarily result in a return to the same frequency of VPBs.

At two points (Fig 2B and G), the frequency of VPBs decreased when the stimulating rate was increased. At one of these points (Fig 2C), the VPBs actually ceased completely.

The pacing interval was maintained at 0.72 second (rate, 83/min), and pacing was accomplished without any further occurrence of VPBs over several days of observation. Thereafter, block disappeared, and pacing was discontinued. There were no further arrhythmias.

**DISCUSSION**

This report presents an apparently paradoxical response of VPBs to pacing. Overdrive suppression usually abolishes VPBs, decreases their number, or has little effect. Here, VPBs appeared when the pacing interval was within a critical range (0.52 to 0.66 second) and were eliminated when the pacing interval was increased to 0.70 second or more (early in the course of observations, the cutoff point was at 0.67 second). At one point the VPBs also ceased to appear when the pacing interval was shortened to 0.56 second (Fig 2, point G).

Two explanations can be advanced for the paradoxical effect of pacing on the VPBs. The first is that the VPBs represent reentrant beats. The same pathophysiologic events which produced left bundle-branch block and complete heart block in this patient are probably also able to produce localized areas of conduction delay. The normal impulses (in this case, the paced beats) thus linger in some part of the heart long enough to reexcite other parts after finally emerging from the area in which they were temporarily delayed.4-8 Wit et al.4 demonstrated in Purkinje fibers that, up to a point, increasing the rate of pacing resulted in increasing frequencies of reentrant beats. The same phenomenon had previously been observed in dogs with digitalis toxicity.8

An explanation for this rate dependency which is applicable to our patient is the following: At a moderately fast rate of pacing, the impulses arise too quickly in succession for all to be transmitted successfully through a depressed area. Each successive beat passing through the depressed area experiences gradually increasing delay (Wenckebach phenomenon). Eventually the delay is such that the exiting impulse finds other parts of the myocardium once again excitable, resulting in a VPB every third to fifth beat at an R-R interval of 0.62 second. As the R-R interval is decreased to 0.59 second, the rate of development of the Wenckebach phenomenon is even more rapid, and each second to third impulse now lingers long enough in the depressed segment to find the ventricle excitable, thus increasing the frequency of VPBs. As the pacing rate is decreased, on the other hand (Fig 2, points A, C to F, and H to J), the impulses pass rapidly enough through the depressed area to find, as they reach its other end, the ventricle still refractory.

Two points in Figure 2 (B and G) suggest that increasing the rate of pacing above a critical rate may actually decrease and even abolish VPBs. Such a phenomenon was also observed by Wit et al.4 in isolated Purkinje fibers and was explained as being due to either a total abolition of conduction into the depressed area or to an improvement of conduction through the depressed fiber.

The second explanation which can be advanced for the rate dependency of the VPBs is that these represent true automaticity arising in pacemaker cells of the "slow-response" type.9 These cells, often seen in an infarct,10 have been found to become more automatic with increasing rates of stimulation, in contrast to cells of the ordinary "fast-response" type. Overdrive may, therefore, be not only ineffective but actually detrimental in attempts at suppressing these automatic cells.

The incidence in man of such a relationship as was described here between VPBs and the basic rate after myocardial infarction remains to be clarified. Wellens11 mentioned a patient in whom pacing at a rate of 110/min could not control a ventricular tachycardia, whereas pacing at a frequency of 85/min suppressed it. Zipes and Knoebel12 reported two patients in whom increasing the ventricular rate with atropine administration increased the number of VPBs. It also remains to be confirmed whether increasing the rate of stimulation beyond a critical point would again reduce the number of VPBs, as anticipated by Wit et al.4 and Cranefield et al.13 and as suggested by points B and G in Figure 2. Certainly an obvious practical point to be learned from this patient and from the studies mentioned is that if overdrive suppression does not seem to have the anticipated effect on VPBs, decreasing the pacing rate may be the next maneuver to try.

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