have to face the fact of the existence of a significant intraventricular conduction defect which increases little the QRS duration and which changes the QRS configuration in an entirely noncharacteristic fashion. A similar QRS pattern is commonly observed in normal people of slender body build, in pulmonary emphysema, and in some cases of right ventricular hypertrophy; and this is indeed the main reason why LPH went unrecognized or was neglected for so many years. It is then not unlikely that many more cases of LPH exist than we are able to recognize; but it remains true that "LPH cannot be a pure electrocardiographic diagnosis; it must necessarily be a clinical-electrocardiographic diagnosis."

The right axis deviation in our case, although not impressive by itself, was more significant because of occurring in an obese person in whom such an electrical axis orientation was not to be expected. This illustrates perhaps the best possible approach to the diagnosis of pure LPH.9 The presence of an unexpected right axis deviation (in regard to the body build, shape of the chest, or particular heart condition), even if the AQRS does not go beyond +90°, and provided there is some form of left ventricular disease, may suggest the existence of LPH. From our experience dealing with the association of RBBB with LPH,1 4 10 it may also be said that the taller the R waves in leads II and III, the greater the likelihood of LPH.

Summarizing the information gathered up to this moment, the following criteria for the diagnosis of pure LPH may be suggested: Pure, uncomplicated LPH should be diagnosed when the AQRS is oriented around +120°, with an Si Q3 pattern, with relatively tall R waves in leads II and III, provided a vertical heart or right ventricular hypertrophy can be excluded, and provided some form of left ventricular disease is present. At this time, it would seem unsound to suggest the diagnosis in the absence of the last requisite. A lateral infarction, when it causes right axis deviation, should also be excluded. This is usually not difficult. The lateral infarct tends to be readily apparent, an Si Q3 pattern is not necessarily present, and the voltage in the standard leads is usually small. Smaller degrees of LPH may be expected to produce both a lesser axis shift and a smaller QRS voltage. In such cases, the diagnosis of LPH may still be considered as a possibility, but only uncommonly as a definite diagnosis, unless conduction is intermittent, as in the case presented in this paper.

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Suppression by a Prominent T-Wave: An Unusual Cause of Malfunction of a Transvenous Demand Pacemaker*

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An unusual mechanism of malfunction of a transvenous demand pacemaker is reported. This is caused by intermittent suppression of the pacemaker by a prominent T-wave resulting from too close a contact of the intracardiac electrode with the ventricular endocardium. The importance of its detection and method of prevention are discussed.

Demand pacemakers have been used with increasing frequency during the past few years so much so that many cardiologists now recommend this type for all patients with heart block.1 The demand pacemaker of the ventricular-inhibited type continues to release an electrical stimulus at a preset pacing frequency unless a spontaneous or ectopic ventricular beat occurs before the end of the preset time interval. When this happens, it resets the timing circuit of the pulse generator and suppresses its output for one complete cycle. In addition this type of pacemaker also may be suppressed by external electrical fields or rarely by a tall T-wave. It is of

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paramount importance to realize that an unusual configuration of T-wave resulting from direct contact of the intracardiac electrode with the endocardium, a prerequisite for reliable transvenous demand pacing, also may defeat the very same purpose by suppressing the pacemaker and causing a reduced pacing rate. Such a situation is reported here.

CASE REPORT

The patient, a 69-year-old man, was admitted because of exertional dyspnea, history of syncope and a slow heart rate. There was a vague history of intermittent digitalis ingestion. Electrocardiogram on admission revealed sinus rhythm alternating with atrial flutter and complete atrioventricular block. Because of the possibility of this being the result of digitalis toxicity, temporary instead of permanent pacing was instituted initially. Transvenous demand pacing, instituted percutaneously by the femoral approach,\textsuperscript{2,3} was carried out by means of an external demand pacemaker (Medtronic, model 5480) connected to a bipolar electrode catheter (Goetz, USCI) placed inside the right ventricle. Demand pacing was easily accomplished at a low stimulation threshold of less than one milliampere and a rate of 75 per minute. However, it was noted later that the interval between the pacemate complexes changed frequently and abruptly between 800 milliseconds (corresponding to a preset rate of 75 pulses per minute) and 1160 ± 20 msec (Fig 1 A). The position of the sensing electrode was in excellent contact with the endocardium of the right ventricle as judged by fluoroscopic examination and confirmed by recording of a contact current from the tip electrode (Fig 1 B). This erratic response persisted in spite of decreasing pacemaker sensitivity. Twelve hours later regular pacing at 75 per minute was again observed (Fig 1 C). An endocardial lead taken at this time revealed that there was no longer any contact current recorded from the tip electrode (Fig 1 D). Ten days later, since the heart block persisted and since it was clearly ascertained from the referring physician that no digitalis preparation was prescribed within the past six weeks, a permanent transvenous demand pacemaker (Medtronic) was installed with resultant effective pacing at a preset rate of 60 per minute. The postoperative course was uneventful.

DISCUSSION

With the increasing popularity of demand pacemaker over the fixed-rate unit and the universal acceptance of transvenous method in the management of patients with heart block,\textsuperscript{1-3} it is most important that the physician should be aware of all the different causes of pacemaker malfunction as remedy depends on their prompt recogni-
tion and correct interpretation.

In our patient there seemed to be two groups of R-R intervals between the paced beats, the shorter ones at 800 msec (in accord with the preset rate of 75 per minute) and the longer ones at 1,160 msec. The difference between the two is 360 msec, which is the interval between the pacing spike before each QRS complex and the peak or nadir of the ensuing T-wave (Fig 1A). This constant delay would indicate that the demand generator was intermittently recycling off a prominent T-wave. The latter was unusually sharp and of large amplitude, especially in the unipolar endocardial electrogram recorded from the tip electrode where it was associated with a contact current and the distance between the beginning of the QRS complex and the inflection point of ST-T junction measured 360 msec (Fig 1B). When pacing became again regular at 75 per minute 12 hours later (Figure 1 C), the endocardial electrogram no longer depicted a contact current (Fig 1 D). Obviously the electrode must have gradually moved away from its original juxta-endocardial position during the ensuing 12 hours, as evidenced by a significant difference in the configurations of the R and T waves between the two electrocardiograms (Fig 1 A and 1 C).

The most important consideration in producing effective demand pacing by transvenous route is intimate endocardial contact by the electrode. When the contact is poor, the demand pacemaker fails to sense the R-wave and thus will not function properly. On the other hand, if the contact is too close between the electrode and the ventricular endocardium, too large a contact current and too sharp a T-wave will trigger the inhibitory circuitry and thus cause, as in our case, a slower discharge rate of the pacemaker than it was set for.

Prevention and treatment of this unusual cause of pacemaker malfunction is, first, to advance the transvenous electrode to the apex of the right ventricle till a contact current is recorded from the distal electrode and then to back off till there is no longer or very little ST elevation on the unipolar intracardiac electrogram. If marked variation of pacing rate persists and cannot be overcome by either increasing the pacer rate or decreasing the pacemaker sensitivity, then either a new endocardial site must be chosen or the electrode system be converted to a unipolar configuration.

This maneuver of electrode placement is of far greater importance when the electrode is passed "blindly" than when under fluoroscopic control. Correct positioning of the float-in pacemaker-electrode is determined by recording a distinct contact current on the intraventricular electrogram from the tip electrode. On the other hand, since it is difficult to ascertain without fluoroscopic control the relative position of the electrode with reference to the endocardial surface in the absence of a contact current on the intracardiac electrogram, placement of pacing electrode by the float-in technique is far less exact and reliable than under direct fluoroscopic control.

It is the purpose of this communication to report this unusual cause of pacemaker irregularity, not only because it may stimulate further reports from other workers in this field so as to assess its frequency of occurrence, but because once recognized its "cure" is a relatively simple matter.

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Calcification in Chickenpox Pneumonia*

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The relationship between pulmonary calcifications and chickenpox pneumonia is reviewed. A case is presented in which pulmonary calcification developed over a seven-year interval following Varicella pneumonia in a patient who had a previously negative chest radiograph. After accidental death, an autopsy was performed and the pulmonary findings are presented. It is felt that an etiologic relationship has been established between the patient's Varicella pneumonia and her subsequent pulmonary calcifications.

During the past ten years many papers have appeared describing calcification following chickenpox pneumonia. The occurrence of this type of calcification in one of my colleagues and her death and the autopsy results were felt worthy of report. Negative films prior to the acute infection were available as well as annual radiographs during the subsequent seven years while calcification developed. Since there was no evidence of histoplasmosis or tuberculosis this seemed to quality as the ultimate proved case which prior authors have envisioned.

REVIEW OF LITERATURE

Much of the credit for making the profession aware of focal calcifications in the chest as a result of chickenpox pneumonia goes to authors from Australia and New Zealand. Of these Mackay and Cairney are generally credited with the first description of this entity in 1960. Review of the