nonspecific depletion of lymph nodes due to chronic lymphocyte loss. Lymph nodes obtained from the sigmoideectomy specimen showed no abnormality and no other nodes were obtained for study. None of the lymph nodes seen on lymphography showed evidence of lymphoblastic or other malignancy.

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Failure of Demand Pacemaker Sensing Due to Electrode Fracture*

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Defects in temporary pacemaker electrode catheters should be considered in the differential diagnosis of inappropriate pacing by ventricular inhibited demand units. In two of three patients, sensing failure was the only manifestation of electrode fracture. Fractures often occur near the junction of the steel pin terminal and electrode and, if intermittent, may be overlooked if flexion maneuvers are omitted from the routine of catheter testing. Unless this complication is appreciated and corrected it may progress to further disruption of the circuit with complete loss of pacing.

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Electrode fracture has not been reported previously as a cause of malfunction of the sensing circuit of temporary demand pacemakers. This paper describes inappropriate fixed rate pacing resulting from such a defect and the means by which this complication can be diagnosed and treated.

In properly functioning ventricular inhibited demand pacing systems, spontaneous QRS complexes are detected by endocardial electrodes and transmitted to the pacemaker unit. When afferent impulses exceed a preset rate, artificial pacing is cancelled. When the intrinsic rate is slower than the preset rate, inhibition does not occur and pacing is initiated.

Malfunction of the sensing circuit of demand pacemakers has been ascribed to low voltage endocardial QRS complexes in patients with acute myocardial infarction and to malposition of the catheter in relatively electrically silent areas. The following case reports demonstrate that electrode defects should also be considered in the differential diagnosis of inappropriate demand pacemaker function.

MATERIALS AND METHODS

Each patient in this report had a No 6 bipolar catheter inserted by cutdown into the right external jugular vein and passed under image intensifier fluoroscopic control to the apex of the right ventricle. External demand pacemakers were employed with the sensitivity set at maximum and electrical output two to three times the stimulation threshold. Continuity testing of the catheters was performed just prior to insertion. Catheter position was checked immediately after placement and subsequently by x-ray, threshold measurement, and unipolar and bipolar endocardial electrograms. Electrode resistance was measured by a Heathkit condenser checker, model C-3. At the time of these studies, the catheter was directly connected to the external pacemaker which in turn was strapped around the patient's upper arm.

CASE REPORTS

Case 1

Intermittent inappropriate demand pacemaker firing was first noted four hours after the establishment of pacing in an 80-year-old man with acute subendocardial infarction, bilateral bundle branch block and sinus bradycardia (Fig 1). Good position of the catheter was indicated by chest x-ray film, pacing threshold of 0.7 ma., and large QRS complexes and ST segment elevations on ring and tip endocardial electrograms. The QRS amplitude of the bipolar electrogram was 7 mv, well above the 1.5 to 2.0 mv required to activate the sensing circuit of the pacemaker. In order to exclude component failure, the pulse generator was replaced by a similar model. However, this failed to eliminate the problem and since the clinical situation had improved, the pacemaker was turned off. Repeated evaluation during the next five days showed a gradual increase of the stimulation threshold to 1.3 ma, and periods of inappropriate fixed-rate pacing of varying duration. On the seventh day, pacemaker function appeared to be normal. At that time there was no change in either the threshold or the endocardial QRS-T contour. The unit seemed to function satisfactorily until the time of its removal.

**Electrode No. 5651, U.S. Catheter and Instrument Co.
*External Demand Pacemaker, Model 5840, Medtronic, Inc.

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on the fifteenth hospital day. 

Comment: The intermittent nature of the malfunction was consistent with electrode fracture, but this diagnosis was rejected at the time because of the absence of baseline interference of endocardial electrograms which is characteristic of this complication.\(^5\) It was not until the pacing catheter was sterilized and reused in the second patient that the nature of the defect became evident. 

CASE 2

Temporary pacing was instituted in an 81-year-old man with an acute diaphragmatic myocardial infarction complicated by sinus bradycardia and ventricular and supraventricular tachyarrhythmias. Inappropriate fixed-rate pacing was noted on the third day after the onset of pacing (Fig 2). Chest x-ray examination and endocardial electrograms showed that catheter position, endocardial contact and QRS amplitude were adequate. Nevertheless, malfunction of the sensing circuit occurred with increasing frequency in the ensuing days. On two occasions malfunction coincided with the patient’s movements in bed and the stimulation threshold fluctuated from 1.3 to 3.0 ma with head movement. The pacemaker continued to perform in an erratic fashion until it was removed on the 13th day. Endocardial electrograms were consistently normal with stable baselines and pacemaker

**Figure 1.** Malfunction of the pacemaker sensing circuit (Case 1) despite large QRS complexes on the endocardial electrogram. A, monitor lead demonstrating a single prematurely paced beat. B, lead II showing inappropriate fixed rate stimulation. C, bipolar endocardial electrogram recorded immediately after B. 1 mv = 2.5 mm.

**Figure 2.** Intermittent failure of the pacemaker sensing circuit due to electrode fracture (Case 2). A and B, continuous electrocardiogram. C and D, normal unipolar endocardial electrograms. 1 mv = 2.5 mm. Fracture seemed unlikely because of the absence of interference (see text).
impulses during nonrefractory periods always caused ventricular depolarization.

Comment: In this patient the variability of threshold and malfunction of the sensing circuit coincident with changes of head and body position suggested either slipping of the electrode tip in the right ventricle, apparently excluded by unchanging electrograms, or an intermittent electrode defect. As in the first case, electrode fracture seemed unlikely because of the consistent absence of interference on the electrograms.

After the catheter was removed, it was subjected to further testing. Visual inspection revealed intact insulation and no kinking. Ohmeter analysis of the circuits from tip and ring to their respective proximal terminals disclosed a resistance of less than 5 ohms. Agitation of the proximal limb of the tip electrode, however, caused fluctuations in resistance from 0 to 20,000 ohms. This was not observed with the ring circuit, nor with flexion of other parts of the catheter, nor with similar catheters. This established the diagnosis of a fracture in the region of the terminal steel pin and proximal limb of the tip electrode.

Case 3
Temporary demand pacing was initiated in a 70-year-old woman with complete heart block and Stokes-Adams attacks. The unit functioned in the fixed-rate mode because of the patient's slow spontaneous ventricular rate. During the next two weeks there was a progressive increase in the excitation threshold to a level of 3.0 ma. On the 16th day an electrocardiogram demonstrated intermittent failure of pacing with absence of pacemaker stimuli (Fig 3). Increasing the pulse generator output to maximum (25 ma) failed to reestablish effective pacing. Endocardial electrograms were obtained and appeared to be normal. Flexion of the proximal limb of the tip electrode, however, caused marked electrogram interference. There was no baseline variation during manipulation of the ring electrode. The pacing system was unpolarized utilizing the intact ring electrode and a subcutaneous anode, thereby restoring pacing. Occasional spontaneous premature systoles inhibited the pacer, showing that demand function was also normal. Later a permanent pacemaker was implanted.

Comment: In this patient failure of pacing indicated an electrode defect that could not be overcome even with maximal pacemaker output. The likelihood of proximal electrode fracture was appreciated and electrograms performed during flexion of this area demonstrated characteristic interference.

Discussion
The diagnosis of electrode fracture can be established by demonstrating characteristic interference on an endocardial electrogram. The misleadingly normal recordings in patients 1 and 2 emphasize the difficulty in diagnosing intermittent fractures. In retrospect, manipulation
of the involved proximal catheter during electrogram recording probably reestablished continuity.

While both the sensing and pacing functions of the system utilize the same circuit, the sensing mechanism generally fails first when the resistance rises. An intact electrode normally has a resistance of less than 5 ohms while a fractured electrode with intact insulation has a resistance of 10,000-20,000 ohms (Medtronic, Inc., technical information). The Medtronic 5840 pulse generator provides a variable voltage up to 30 volts to deliver a fixed current into a variable resistance. From Ohm’s law 30 volts/20,000 ohms = 1.5 milliamperes, a current usually sufficient to depolarize the heart. On the other hand, the comparatively small electrical output of the heart is insufficient to overcome the augmented resistance of a fractured electrode. In our first two patients this caused demand pacer entrance block and inappropriate fixed rate pacing. In the third patient even maximal pacemaker output was insufficient to bridge the gap so pacing and sensing functions were lost. Therefore, when the sensing function of a demand pacemaker fails, it is important to rule out electrode fracture since further disruption of the circuit may lead to complete loss of pacing.

With catheters of the type used in our patients, most fractures occur at the junction of the terminal steel pin and the proximal catheter limb. Tension at this junction is minimal when the diameter of the loop of the external portion of the catheter exceeds eight inches, whereas smaller loops or acute angulation increase the likelihood of fracture. Motion at this vulnerable location can be reduced by employing a patient cable or an adapter plug instead of a direct connection between the catheter terminal and the pacemaker. When an electrode fracture occurs in one wire of a bipolar catheter, normal demand pacing can often be restored by utilizing the intact wire as the cathode in a unipolar system.

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Conversion of Atrial Flutter to Normal Sinus Rhythm after Intravenous Administration of Edrophonium Chloride*

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Carotid sinus stimulation or the administration of cholinergic drugs are known to convert paroxysmal atrial tachycardia to normal sinus rhythm. In atrial flutter usually only a slowing of the ventricular response, due to decreased atrioventricular conduction, is obtained, while the atrial flutter mechanism persists. A patient is described in whom the intravenous administration of 10 mg edrophonium chloride (Tension) resulted in the conversion of atrial flutter to normal sinus rhythm.

INTRODUCTION

Edrophonium chloride, a cholinergic drug, has been used as a diagnostic agent for myasthenia gravis and employed as an antagonist of curare in anesthesia. The drug combines low toxicity with a short duration of action and a rapid maximal effect. Recently, it has been reported to be effective in terminating paroxysmal atrial tachycardias because of its parasympathomimetic action.1,2

The objective of this communication is to report our observation of a patient with atrial flutter in whom the intravenous administration of edrophonium chloride resulted in restoration of normal sinus rhythm. As far as we know, the results of this drug in atrial flutter have not been previously reported.

CASE REPORT

A 65-year-old white man was admitted to the medical ward of the Philadelphia General Hospital because of an episode of rapid heart action which failed to respond to carotid sinus stimulation. The patient presented a long history of recurrent attacks of “paroxysmal tachycardia” which could usually be abolished by Valsalva maneuvers or carotid sinus pressure. The past history revealed rheumatic heart disease with severe aortic stenosis and insufficiency, documented myocardial infarction and angina. In 1965 the aortic valve was replaced by a Starr-Edwards prosthesis. The patient’s medication included furosemide, 80 mg daily, potassium supplement and coumadin.

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