False Inhibition of a Demand Pacemaker by Inactive Myocardial Electrodes

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SUMMARY

A 14-year-old girl has been implanted a cardiac pacemaker with a myocardial electrode since 8 years old. As the first myocardial electrode fractured near its tip, the second one was added. Thus, patient had two pairs of myocardial electrode systems with one demand pacemaker implanted. When she was readmitted to hospital because of light-headedness, ECG monitor revealed the frequent suppression of pacemaker emission on transcutaneous waving of pacemaker unit. Application of the magnet over the generator resulted in no inhibition even on moving the unit. The pacemaker pocket was reopened. Waving the active myocardial electrode or pacemaker unit did not inhibit the demand pacemaker, but manipulation of the inactive lead induced suppression of pacemaker emission. Such manipulation produced high interference waves, which were created probably by motion of the cut end of inactive leads against patient's abdominal muscle. The exchange of the demand pulse generator to a fixed mode was followed by complete disappearance of light-headedness in this case.

Additional Indexing Words:
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ALTHOUGH the demand cardiac pacemaker offers a degree of safety from electrical competition not available with the continuous rate pacemaker, it has created a new set of pacemaker problems originating from the mechanism that detects ventricular depolarization. A new type of interference has been documented in our experience, presumably electromechanical in origin.

In a patient with two pairs of myocardial electrodes, transcutaneous manipulation of inactive leads induced suppression of demand pacemaker emission. There were no signs of pacemaker malfunction. Electrode motion
produced interference waves, which were sufficient to inhibit a demand pacemaker. Similar complication has been reported by Widmann and associates,\textsuperscript{1)} which was observed in cases with two endocardial leads. Present report is the first one in that myocardial lead systems are concerned with such complication.

**Case Report**

A 14-year-old girl has had complete A-V block following surgical repair of persistent common atrioventricular defect at her age of 5. When frequent episodes of Stokes-Adams attack occurred at her age of 8, she had a Medtronic\textsuperscript{*} model 5862 pacemaker, with an automatic rate of 88/min and the feature of fixed type. A pair of electrodes were sutured on the anterior right ventricular myocardium following median sternotomy. Generator was implanted in the abdominal muscle. Patient became asymptomatic. When she was 10 years old (April 1973), only the pulse generator was exchanged because of battery exhaustion.

In March 1975, both tips of myocardial electrodes were found fractured on

![Fig. 1. X-ray film of antero-posterior view of abdomen. Pacemaker unit is implanted in abdominal subcutaneous pocket. Active (new) and inactive (old) leads crossed at upper abdominal area. Proximal ends of inactive leads are cut without any caps.](image)

* Medtronic, Inc., Minneapolis, Minn.
Fig. 2. X-ray film of lateral view of thoraco-abdominal region. Distal ends of inactive lead were sutured on right ventricular myocardium. Tip of inactive lead is fractured, shown by the arrow. Active lead is sutured on the left ventricular wall.

X-ray films though ECG showed no signs of pacing failure. She did not have any intrinsic rhythm. In order to prevent occurrence of Stokes-Adams attack, new implant of myocardial electrode in addition to pulse generator exchange were performed on July 21st, 1975. Cathode and anode myocardial electrodes were sutured separately on the left ventricular myocardium following left side thoracotomy. Two electrodes employed were CPI* model UM. Previous fractured leads were left unremoved, proximal ends of which were cut off and buried in the abdominal muscle. Cut ends were not covered by non-conductive materials. The patient's pulse generator, implanted on the right abdominal wall, was CPI model 401 BD with an automatic rate of 74.6/min and the mode of demand type (Figs. 1 and 2).

Approximately 9 months following this implantation, she began to feel light-headedness without any particular reasons. It usually occurred on exercise. This dizziness gradually increased in frequency as well as in severity, despite of the absence of syncopal episodes. When patient was readmitted for evaluation on December 17th 1976, she felt dizziness about 3 times a week. She had grade 3 systolic murmur over apex. No diastolic murmur was audible. Second heart sound over pulmonic area was splitted without any accentuation. On X-ray films
of chest, cardiothoracic ratio was 62%. Blood pressure was 122/72 mmHg. Pulse rate was 74/min regular. ECG showed only pacing rhythm. Neurological examinations were normal. Electroencephalogram showed nothing abnormal. ECG monitor recorded suppression of the demand unit on waving of the implanted generator, and on pressing the upper abdominal wall. No pacing failure was noted. No escape beat was found. Even slight pressure on the upper abdominal wall resulted in variable suppression of pacemaker emission (Fig. 3). The basic escape interval of this pacemaker was 813 msec. The irregular beats exhibited R-R intervals from 1,100 to 3,000 msec, none of which were even multiples of the escape rate.

Deep respiration produced changes of QRS configuration, but did not show any suppression of pacemaker emission. The suppression occurred only when patient moved or when abdominal wall around the generator was manipulated. There was no evidence of pacemaker malfunction. Both pacing interval and pulse width (0.96 msec) had been stationary since implant.

Application of the magnet over the generator resulted in absolutely regular ventricular pacing with an automatic interval of 670 msec (Fig. 4). Patient was monitored with magnet for 3 days. There was no loss of capture. No variation of rate during generator manipulation was detected. Voltage change of pacing spike was not found either. It was evident that patient’s demand pacemaker sensed some signals produced by manipulation.

On December 22nd, 1976, operation was carried out. Prior to reopening of the pacemaker pocket, a temporary transvenous pacing electrode was inserted.
under fluoroscopic control into the apex of the right ventricle. The external pacing was set at the rate of 50/min with demand mode. During operative procedures, external pacing worked efficiently whenever patient's demand pacemaker was inhibited by manipulation. Following removal of the CPI unit from pacemaker pocket, pacing electrodes were carefully examined. Leads were properly insulated and firmly connected to the generator. The suppression of the pacemaker emission was not noted by motion of active pacemaker electrodes, or of pacemaker unit. Flexion or extention of electrodes did not suppress the demand circuit either. Suppression occurred easily by manipulation of inactive electrode. The inactive lead was bipolar system as shown in Figs. 1 and 2. The distal end was sutured on the right ventricle, and the proximal end was in the abdominal rectal muscle. Distance between old (inactive) lead and new (active) lead was approximately several millimeters at the cross point. Two pairs of myocardial leads did not have any direct contact. Active electrodes were disconnected from the generator, and attached to the temporary external pacemaker for the measurement of stimulation threshold. It was 2.0 mA, tested by Medtronic model 5880A, when the lower myocardial lead

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**Fig. 4.** Simultaneous recording of ECG following application of the magnet. R-R interval was 670 msec. No pacing failure was noted. QRS configuration changed by respiration.
Fig. 5. Lead I, epicardial and lead III ECGs were simultaneously recorded during operation. Patient was paced at the rate of 50/min by the intravenous temporary electrode. Epicardial ECG was recorded through active (new) leads following removal of pulse generator. Left panels were control. Right panels were recorded during manipulation of inactive electrodes. Abnormal interference waves appeared on epicardial recordings though both leads I and III did not show any such waves.

External sources of electrical interference with demand pacemaker function include automobile ignition systems, electrocautery units, radiofrequency transmitters, radar transmitters, physiotherapy diathermy units, electric...
razors, and microwave ovens.

None of the aforementioned devices was a factor in our present case. This directed attention to the electrode systems themselves. In our case, passive motion of inactive leads against abdominal tissue produced electrical potentials. In a condition that a piece of metal contacts with vital tissue, the motion of the former creates electrical potentials on its contact surface, which can be 50 to 500 mV and generally called "vital electricity." There is even an idea to use this "vital electricity" for cardiac pacing instead of conventional chemical battery.

Electromechanical pacemaker interference of our present case is similar in mechanism to that described by Widmann and associates. The cut (or proximal) end of inactive leads was uncovered in our case, and so the bare electrode surface contacts with patient's abdominal muscle, creating vital electricity on its motion. From this experience, the authors consider when inactive pacing leads are left in patient's body, the cut end should be capped by some non-conductive material.

Prior to reopening of the pacemaker pocket in present case, the authors assumed muscle potentials, partial breakage of leads and a loose electrode connection as well. Suppression due to muscle potentials, however, could be denied because our case had a bipolar system. The inhibition due to muscle potentials is always encountered in cases with unipolar lead system. Partial breakage of leads or a loose electrode connection was highly suspected. Sudden changes of resistance in the electrode-lead system produce a potential change at the pacemaker terminals, which is of sufficient magnitude to be interpreted as spontaneous ventricular depolarization and inhibit the demand pacemaker. This complication, however, is usually associated not only with false inhibition but also with pacing failure. Even if pacing failure is not noted, intermittent variations of pacing spike voltage are found on electrode manipulation. Our case presented neither pacing failure nor variations of pacing spike voltage. Therefore, partial breakage of leads were also excluded from possible causes.

Recent pacemaker models are designed to switch into an asynchronous mode in the presence of intense electromagnetic radiation. Interference potential in our case seemed to be too weak to switch into the asynchronous mode.

Finally authors agree to the opinion that the use of a fixed type pacemaker should be much more recommended by physicians. When no spontaneous ventricular activity exists, implantation of asynchronous pacemaker seems to be much safer and more reasonable.
REFERENCES


