Phrenic Nerve Stimulation as a Complication of the Implantable Cardiac Pacemaker

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The implantable cardiac pacemaker has rapidly become an essential modality in the clinical management of medically refractory Stokes-Adams disease. In spite of increasing refinement in both the myocardial electrodes and the pacemaker, significant complications still occur and are discussed in representative papers by Chardack and Zoll. The unusual complication of phrenic nerve stimulation was recently encountered after insertion of a Chardack pacemaker. Since this proved to be troublesome and has not been previously reported, it seemed to be worth recording.

Case Summary
J.C.S., a 36-year-old white man, was admitted to the Veterans Administration Hospital, Oteen, North Carolina, June 1959, with symptoms of increasing exertional dyspnea, precordial pain, and fullness in the head dating back to January 1959. He was found to have idiopathic atrioventricular block varying from second degree to complete dissociation, unresponsive to medical treatment. In February 1960, while driving a truck, he suffered his first Stokes-Adams attack. A compression fracture of the sixth dorsal vertebra and dislocation of the left acromioclavicular joint were sustained. Syncope occurred February, April, and July 1961. On the day of admission, September 25, 1961, three Stokes-Adams attacks occurred. Physical findings included pulse rate of 48 per minute and blood pressure in the arm of 130/70. The cardiac size was normal to percussion. There was a grade-II apical systolic murmur and no evidence of congestive heart failure. Admission electrocardiogram showed atrioventricular block varying between 2 to 1 and 3 to 1. The atrial rate was 96 per minute and ventricular rates were 32 to 48 per minute.

On September 28, 1961, a Chardack internal pacemaker and electrodes were implanted. The pericardium was opened anterior to the phrenic nerve and the electrodes were inserted into the myocardium near the base of the left ventricle and sutured in standard fashion. Two hours postoperatively, contractions of the left diaphragm synchronous with the artificial cardiac rate were observed. Because it was thought that this would not be well tolerated by the patient, re-exploration of the chest was carried out promptly. The electrodes were still well seated on the base of the left ventricle. The left diaphragm was contracting simultaneously with the stimulated cardiac impulse. Without disturbance of the electrodes buried in the myocardium, the lead wires were transplanted anteriorly, farther from the phrenic nerve, and distal phreniclasia was performed. Teflon felt was interposed between the electrodes and pericardium. Postoperatively no further phrenic stimulation occurred and a heart rate of 52 per minute was maintained. Transient pericardial effusion was noted, which produced no significant elevation of venous pressure and which resolved spontaneously. Atelectasis of the left lower lobe developed. This cleared with bronchoscopy, antibiotics, and expectorants.

Nine months later, diaphragmatic contractions synchronous with the paced cardiac rate recurred. These were more noticeable in the supine position and decidedly interfered with sleep. On August 14, 1962, a left cervical phreniclasia was performed under local anesthesia without immediate benefit. On the fifth postoperative day, however, abnormal diaphragmatic contractions ceased and temporarily recurred on the seventh postoperative day. The left hemidiaphragm was seen on fluoroscopy to be elevated on the forty-second postoperative day and no spasmotic contractions were observed. There have been no further abnormal diaphragmatic contractions to date (4½ months postoperatively). No further Stokes-Adams episodes occurred until 4 months later, when the pacemaker ceased to function. Upon exposure of the “tail” of the device, it was determined that the cardiac electrodes were still able to conduct electric impulses from a substitute pacemaker. Therefore, a new pacemaker with variable amplitude and frequency controls was implanted under local anesthesia on December 3, 1962.

Discussion
In order to observe this phenomenon di-
rectly, the intact pericardium adjacent to the phrenic nerve was stimulated in three mongrel dogs with use of the Chardack electrodes. When one electrode was placed 8 mm. from the phrenic nerve, diaphragmatic contractions were produced with the second electrode at distances of 4 to 10 mm. from the phrenic nerve and in various orientations.

Since afferent impulses of pericardial origin have been demonstrated in the phrenic nerve of cats synchronous with heart-beat and with mechanical stimulation of the parietal pericardium, the possibility of an afferent effect was investigated in another dog. The phrenic nerve was isolated intact with a flap of pericardium. While the pericardial flap bearing the phrenic nerve was in contact with epicardium, stimulation of the epicardium produced diaphragmatic contractions, both before and after division of the phrenic nerve high in the thorax. This suggested that efferent pathways only were involved. When the proximally divided nerve was elevated into the wound, repeated epicardial and pericardial stimulation failed to produce diaphragmatic response. This demonstrated that the stimulus was mediated only through the phrenic nerve and by spread of the electrical impulse through the epicardium to the overlying phrenic nerve.

In one human patient the intact pericardium adjacent to the phrenic nerve was stimulated with Chardack electrodes, and diaphragmatic response similar to that in the dog was observed.

Since the complication under study has not been previously reported, further cases were gathered through personal communications.

Thomson reported two cases, both following surgery for congenital cardiac anomalies. In the first case, atrioventricular block was induced following successful repair of multiple defects, and Hunter-Roth electrodes and Chardack pacemaker were implanted. Post-operatively, left diaphragmatic contractions synchronous with the paced cardiac beat were present for 48 hours and then spontaneously disappeared. Subsequently, the patient had several operative procedures for replacement of the Hunter-Roth electrodes by Chardack electrodes, and later also for replacement of the pacemaker itself. Immediately after this, abnormal left diaphragmatic contractions were again noted. The chest was re-explored and one electrode, which was very close to the left phrenic nerve, was found to be seated tangentially in the myocardium. A portion of the uninsulated electrode wire, therefore, was in contact with epicardium. Minor alterations of the position of the electrode produced phrenic nerve stimulation. Therefore, the electrodes were reinserted more anteriorly in the left ventricle, and the difficulty was immediately corrected. The second case was one of congenital atrioventricular canal with surgically induced block. The Chardack pacemaker and electrodes were utilized, and diaphragmatic contractions synchronous with the paced cardiac beat of only a few hours’ duration resulted. No further treatment was required in this case.

Wolcott implanted one of the earlier Chardack pacemakers in a 60-year-old man with complete heart block. Postoperatively, the patient developed abnormal contractions of the left chest related to turning on the side. This continued for 1 month, then intermittently for 2½ months, and spontaneously subsided. Eighteen months later the pacemaker was replaced and a break in the plastic at the point of electrode entry was found. The thoracic contractions were considered to be a result of intercostal nerve irritation and may not represent phrenic nerve stimulation.

Kraeft implanted a General Electric pacemaker and noted stimulated diaphragmatic contractions prior to closure of the chest. Distal phreniclasia was performed and the difficulty was corrected. Five months later, stimulated diaphragmatic contractions recurred that interfered with sleep. A left cervical phrenicotomy was performed and 48 hours later contractions ceased. This case most closely resembled our own.

Zoll has seen this complication two or three times following insertion of the Electrodyne
Pacemaker-Electrode unit. This has been corrected during the initial implantation by moving the terminal cut end of the electrode or interposing gelfoam between the electrode wire and the pericardium. The complication is now prevented by turning the cut end of the electrode wire downward into the myocardium and maintaining its position by suture. Diaphragmatic stimulation has not been a problem for longer than a few days in Zoll's experience.

Weirich described a case in which the right diaphragm was stimulated with an endocardial electrode in place. Conceivably this could have been the result of a leak in the electrode wire insulation along the superior vena cava, in an area near the phrenic nerve.

In review, it is apparent that in some cases aberrant diaphragmatic contractions have been transient and of little moment. This complication may be very detrimental, however, especially in the patient with heart block recuperating from thoracotomy. We would consider the following factors important in management.

An anterolateral or anterior thoracotomy incision allows for adequate exposure of the anterior aspect of the heart.

The electrodes should be placed well anteriorly on the left ventricle as far as possible from the position that the phrenic nerve will occupy when the pericardium is reapproximated.

The electrodes should be inserted into the myocardium accurately to avoid exposure of any uninsulated portion.

Insertion of insulating material such as Teflon felt between the electrodes and the pericardium may be advisable.

One should observe the diaphragm prior to closure, when it is reasonably certain that the effect of muscle-paralytic drugs has passed. If diaphragmatic contractions are present in spite of all the above precautions, distal phreniclasia will interrupt the abnormal contractions.

If the syndrome is delayed in onset and persists, cervical phreniclasia under local anesthesia, which has been effective in two cases, may be efficacious.

Summary and Conclusions

The unusual complication of phrenic nerve stimulation following implantation of a cardiac pacemaker is documented. From experimental observations in the dog, it was concluded that the stimulus was transmitted to the phrenic nerve independent of pericardial nervous connections; that diaphragmatic response could only have been mediated through the phrenic nerve; and that efferent impulses only were involved in the phrenic nerve mechanism. This complication has occurred with various electrode designs. Symptoms may be transient and of little consequence, or persistent and troublesome, requiring further treatment. Suggestions for the prevention and management of future cases are presented.

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References

7. ZOLL, P. M.: Personal communication, 1962.

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