Sound Caused by Diaphragmatic Contraction Resulting from Transvenous Cardiac Pacemaker*

Joseph Schluger, M.D.** and Robert E. Wolf, M.D., F.C.C.P.†

A presystolic "click" beginning 0.1 second before the first heart sound occurred in a patient with a permanent transvenous cardiac pacemaker. Simultaneous fluoroscopy, phonocardiography and electrocardiography demonstrated that the "click" was generated by contraction of the left hemidiaphragm which was stimulated by the electrical discharge from the tip of the electrode catheter located in a tributary of the coronary sinus.

Extraneous sounds and auscultatory "clicks" caused by electronic cardiac pacemakers have been recently reported.1-3 These extra sounds have been attributed to contractions of intercostal muscles stimulated by electrical discharge of the pacemaker. An instance is reported in which the extra sound was caused by contraction of the diaphragm stimulated by the electrical discharge of a transvenous cardiac pacemaker electrode catheter which was in an abnormal location, ie, a tributary of the coronary sinus.

CASE REPORT

A 76-year-old white man was hospitalized on August 23, 1967 because of chest pain, dyspnea and dizziness. An electrocardiogram revealed atrial flutter and complete heart block with a ventricular rate of 36 per minute. During temporary transvenous cardiac pacing, the electrocardiographic pattern was that of a left bundle branch block. A Medtronic permanent bipolar transvenous catheter, model No. 5816, was inserted via the right external jugular vein and was positioned in what was thought to be the apex of the right ventricle. Because a stable pacing site with a low threshold could not be located, a Medtronic permanent pacemaker, model No. 5807C, with a rate of 68 per minute and output of 11.2 ma was connected to the transvenous electrode even though the pacing threshold was 3 ma. The electrocardiogram indicated a right bundle branch block.

The patient was readmitted to the hospital on June 1, 1968 because of bone pain due to metastatic carcinoma. He stated that for one to two hours a day since discharge, he had felt a "jumping or pulsation" in the anterolateral aspect of the left chest. There was no relationship to activity, position, time of day or meals. Visible and palpable sequential pulsations of the lower intercostal spaces in the anterolateral aspect of the left hemithorax were noted at the pacemaker rate. Over the left hemithorax, an audible "click" was heard closely preceding the first heart sound and loudest at the anterior axillary line. During normal breathing, the "click" was loudest in expiration and disappeared during deep, held inspiration as did the pulsations of the chest wall. With deep, held expiration, the "click" was initially very loud but faded gradually and became inaudible by the fourth or fifth cardiac cycle. Although the "click" and chest wall movement were present during the examination, the patient was unaware of any discharge of the transvenous cardiac pacemaker electrode catheter which was in an abnormal location, ie, a tributary of the coronary sinus.

Reprint requests: Dr. Wolf, Cardiac Laboratory, Long Island College Hospital, Brooklyn nickel 11201.

*From the Cardiac Catheterization Laboratory and the Department of Medicine of The Long Island College Hospital and The State University of New York, Downstate Medical Center, Brooklyn.

**Director, Cardiac Catheterization Laboratory; Attending Physician, The Long Island College Hospital; Clinical Associate Professor of Medicine, State University of New York, Downstate Medical Center, Brooklyn.

†Associate Attending Physician, The Long Island College Hospital; Clinical Instructor of Medicine, State University of New York, Downstate Medical Center, Brooklyn.

Figures 1 and 2. Posteroanterior left and lateral right chest roentgenograms. The tip of the electrode catheter is within the cardiac silhouette and not in proximity to the chest wall.
subjective "jumping or pulsation" of the chest wall. The electrocardiogram was unchanged from the time of implantation.

Routine chest roentgenography, image-intensification fluoroscopy and cineroentgenography were performed. The catheter tip was within the cardiac silhouette but was in an abnormal position close to the posterior border of the heart and unchanged from the time of implantation (Fig 1 and 2). Rhythmic contractions of the left hemidiaphragm, at the pacemaker rate, were noted when the diaphragm appeared to be in contact with the catheter tip in deep expiration and during quiet breathing (Fig 3). Diaphragmatic contraction occurred just prior to ventricular systole. During attempted deep, held expiration, vigorous diaphragmatic contractions occurred initially but progressively decreased until no contractions were seen by the fourth or fifth cardiac cycle. With deep inspiration (Fig 4), no diaphragmatic contractions occurred at all.

On phonocardiography (electronics for Medicine DR-8 Recorder with a multiple band phono amplifier, model TPD, and a PS-1B microphone), the auscultatory "click" was recorded best in the low frequency range (50 to 100 cps) over the anterolateral aspect of the left hemithorax. The initial component of the "click" occurred almost simultaneously with the pacemaker stimulus and 0.1 second before the initial component of the first heart sound. It had a duration of 0.08 second (Fig 5). The phonocardiogram confirmed the respiratory variations noted by physical examination (Fig 6). A totally satisfactory apexcardiogram could not be obtained but there was no early outward movement prior to systole.

**DISCUSSION**

Extra "heart sounds" or auscultatory "clicks" occur in some patients with permanent electronic cardiac pacemakers. The extraneous "heart sound" is a discrete presystolic "click" which occurs 0.08 to 0.12 second before the first heart sound and may be mistaken for the first heart sound. The "click" may be present only with expiration or may decrease in loudness with inspiration and is usually heard best in the mitral or tricuspid areas. There may be associated twitching of the thoracic musculature in the region of the fifth intercostal space in the mid-clavicular line. On routine phonocardiography, the "click" appears to occur simultaneously with the pacemaker stimulus but by high speed phonocardiography, the sound is shown to occur 6 milliseconds after the pacemaker stimulus. Apexcar-
diography usually shows an early outward movement of the chest wall coinciding with the ausculatory “click.” 1-7

Nager and co-workers 1 believe that the sound is cardiac in origin caused by premature contraction of the myocardium directly under the electrode catheter. Harris 2 has pointed out that such a view is not consistent with the “all or none law” of Bowditch or with the known 21 millisecond electromechanical interval for heart muscle. The persistence of the sound even when the electronic pacemaker stimulus falls in the refractory period of the cardiac cycle strongly mitigates against a cardiac origin of the “click.” 2-4 Harris 2 demonstrated that the “click” could be completely abolished when an anesthetized patient was given a neuromuscular blocking agent. These observations rather conclusively eliminate a cardiac etiology for the extraneous “heart sounds.”

Although the diaphragm was at first considered to be the muscle producing the “click,” abnormal diaphragmatic contractions were not seen fluoroscopically 1-2 except in one patient, in whom decrease in the pacemaker output eliminated the diaphragmatic contraction without eliminating the extra sound. 3 Misra, Korn, Ghahramani and Same 1 stated that contraction of the diaphragm can cause a pacemaker “click” and refer to an article by Morris Jr and associates 10 for documentation, but Morris Jr. and associates 10 make no mention of a diaphragmatic pacemaker sound occurring in their six cases of pacemaker-induced diaphragmatic contraction. The example cited by Misra and co-workers 10 was not clearly documented since there was no mention of the presence of fluoroscopically visible diaphragmatic contractions and the low frequency epigastric movement could have been due to chest or abdominal wall muscle contraction rather than diaphragmatic contraction. Massumi 11 recorded a presystolic “sound” in the 40 to 200 cps range associated with diaphragmatic contraction, in an example of cardiac pacing from the coronary sinus, but failed to mention if the “sound” was audible with the stethoscope. Currently, contraction of intercostal muscles stimulated by the cardiac catheter electrode is believed to be the cause of the extra sound. 2-5, 7, 8 Important differences between previously reported cases and the patient reported herein do exist, however, and the question of diaphragmatic contraction in the genesis of some of the auscultatory “clicks” is again raised.

In the present patient, the tip of the electrode did not approximate the chest wall (Fig 1 and 2) but was very close to the left hemidiaphragm (Fig 3). The “click” was not heard at all times and disappeared during deep inspiration. It was demonstrated that the left hemidiaphragm contracted synchronously with the pacemaker discharge and was associated with the extra sound. The loudness of the “click” varied directly with the vigor of the abnormal diaphragmatic contraction. The gradual decrease in loudness of the “click” and vigor of the paced diaphragmatic contractions noted during attempted held deep expiration may have been due to inability of the patient to maintain the deep expiratory position. Movement of the intercostal spaces was noted only when the extra sounds were present but unlike the twitching of intercostal contraction was wave-like as described by Litten 12 and attributable to peeling of the diaphragm from the thorax. There was no presystolic outward movement of the chest wall demonstrated by apexcardiography.

Diaphragmatic contraction due to phrenic nerve stimulation by endocardial and epicardial cardiac pacemaker catheters, but without the production of an auscultatory “click,” has been reported. 16, 13-16 Diaphragmatic stimulation by the electrode catheter may occur with or without perforation 10, 18 and may be the first sign of perforation. This possibility was considered in all previously reported cases of pacemaker “clicks” but was dismissed for lack of substantiation, except in one case, where perforation was proved by angiocardiography.

Although right bundle branch block configuration should arouse suspicion of perforation of the free wall of the ventricle or of the interventricular septum with endocardial pacing of the left ventricle, a right bundle branch block pattern may be present without perforation. 11, 17-21

The radiographic appearance of the position of the electrode catheter, the right bundle branch block pattern of the electrocardiogram, the high current required from the very outset to produce consistent cardiac pacing and the absence of definite evidence of cardiac perforation lead us to conclude that the electrode catheter, in the present case, did not perforate the heart but is abnormally located within a tributary of the coronary sinus.

Mowrer and co-workers 10 postulated that pacemaker “clicks” may indicate current leakage at the connection of the pacemaker generator and catheter with resultant conversion of the bipolar to a unipolar system leading to excessive current drain and premature battery exhaustion.

Because of the possible significance of the pacemaker-induced extraneous sound, in relation to perforation of the right ventricle, abnormal catheter position or premature battery exhaustion, all patients with implanted pacemakers should have careful inspection and auscultation. Phonocardiography will confirm the pacemaker “click” and differentiate it from the first heart sound. Patients with pacemaker “clicks” should be examined roentgenologically to determine the presence of cardiac perforation or abnormal catheter position. The catheter should be repositioned unless the patient is not uncomfortable and pacing is satisfactory. Korn and associates 11 found no evidence of pacemaker malfunction in their seven cases despite malposition of the catheter tip. If the electrode catheter is in its normal position, the patient with a pacemaker “click” should be observed carefully for premature battery failure.

ACKNOWLEDGMENT: We gratefully acknowledge the secretarial assistance of Mrs. Maryann Brown in the preparation of the manuscript.

REFERENCES

1 Nager F, Buhlmann A, Schaub F, et al: Auskultatorische und Kardiographische Befunde bei Patienten mit Im-
Endobronchial Hodgkin's Disease and Bronchoesophageal Fistula

Gildo Renzi, M.D., F.C.C.P.,* and Robert Lesage, M.D.,**

A patient with Hodgkin's disease complicated in the terminal stage by endobronchial involvement and bronchoesophageal fistula is reported. The patient died of recurrent bronchopneumonia secondary to the fistula. Even though mecloretamine (Mustargen) intravenously and radiotherapy locally were given, no improvement was noted.

In the chest, malignant lymphoma may involve lymph nodes, pulmonary parenchyma and the tracheobronchial tree. Endobronchial involvement is rare and may be diagnosed only by bronchoscopic examination. We present a case of Hodgkin's disease with endobronchial involvement and bronchoesophageal fistula formation.

**Case Report**

A 25-year-old white woman followed and treated for Hodgkin's disease over the past four years was admitted to Notre Dame Hospital with the following symptoms: over the past two weeks she had noted a progressive productive cough. She also noted attacks of fever and dysphagia and dyspnea which were intermittent and secondary to the ingestion of solids or liquids and followed by vomiting and regurgitation. She also complained of generalized pruritus and a significant weight loss. Her past history was noteworthy in that, four years previously, she had a biopsy of a subclavicular node which revealed Hodgkin's disease. At that time, a chest x-ray film showed marked mediastinal adenopathies and the patient was treated by radiotherapy, 2,000 rads, on the mediastinal region. Following this, she had recurrences in the inguinal region and also the splenic angle of the colon and both these responded to radiotherapy. On her last admission, physical examination revealed a patient who was dyspeptic, cyanotic and who presented an almost continuous cough. Blood pressure was 120 over 70 mm Hg, temperature was 101°F, pulse rate was 92 per minute and regular, respiration rate was 30 per minute. Palpable lymph nodes were noted in the cervical area. The trachea was in the midline and the neck veins were not distended. Chest examination revealed no dullness but crepitant rales were heard bilaterally. Examination of the abdomen did not reveal any abnormality. Results of routine laboratory tests were normal. Chest x-ray picture revealed probable aspiration bronchopneumonia but no mediastinal adenopathy. A barium swallow showed a fistula between the left main stem bronchus and the esophagus (Fig 1). Esophagoscopy revealed the fistula to be 26 cm on the anterolateral wall. A biopsy specimen was taken but was negative. During esophagoscopy, as the patient coughed, bubbles of air could be seen in the fistula. Bronchoscopy revealed granular tissue partially obstructing the posterior wall of the left main stem bronchus.

*Active Physician, Department of Medicine, Notre Dame Hospital; Clinical Professor, University of Montreal, Montreal, Quebec.

**Active Physician, Department of Pathology, Notre Dame Hospital; Professor, Department of Pathology, University of Montreal.