have the benefit of a lung biopsy, a hypersensitivity reaction to CS must be considered as a possible cause of our patient's condition. This case emphasizes the need for close and continued monitoring of patients receiving this compound.

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Elimination of Diaphragmatic Contractions from Chronic Pacing Catheter Perforation of the Heart by Conversion to a Unipolar System*

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This report describes how simple conversion from a bipolar to unipolar pacing system eliminated bothersome diaphragmatic stimulation in a patient with chronic catheter perforation of the heart. This approach, when feasible, appears preferable to the insertion of a second pacing catheter.

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M yocardial perforation by endocardial electrodes remains a relatively uncommon but important complication of the percutaneous technique of cardiac pacing. Although perforation usually results in pacemaker failure or remains clinically unapparent, it may occasionally cause bothersome diaphragmatic contraction in the absence of pacemaker failure. This brief report describes how simple conversion from a bipolar to unipolar pacing system eliminated unrelenting diaphragmatic stimulation in a patient with chronic catheter perforation of the heart.

CASE REPORT

In 1967, a 71-year-old woman with Stokes-Adams attacks received a permanent transvenous bipolar pacemaker at another hospital. Twenty-four hours after implantation she developed uncomfortable “twisting” of the left upper abdominal area synchronous with the heart beat. Syncope did not recur but the annoying abdominal jerking continued, often awakening her at night. The pulse generator was replaced electively two and one-half years later.

We first saw the patient in July, 1971 when her primary concern was abdominal jerking. On examination, there was prominent left upper abdominal jerking synchronous with the pulse. Changes in posture produced no effect. Localized contraction of the intercostal muscles in the anterior axillary line in the sixth left intercostal space was clearly visible, and auscultation revealed a loud pacemaker sound² confirmed by phonocardiography (Fig 1). The electrocardiogram showed regular ventricular capture, the paced beats exhibiting a left bundle branch block pattern of depolarization, with

![Figure 1](http://journal.publications.chestnet.org/pdffaasdownload.ashx?url=data/journals/chest/20953/)

**Figure 1. (Upper Panel)** Phonocardiogram showing recording of pacemaker sound (PS), carotid pulse and electrocardiogram. (Lower Panel) Recording of chest wall muscle contraction. Onset of pulsation is coincident with pacemaker sound and spike.
left axis deviation in the frontal plane. The chest x-ray film findings suggested perforation (Fig 2). Fluoroscopy confirmed contraction of the left hemidiaphragm synchronous with cardiac pulsation.

At operation, the electrode could not be withdrawn. Unipolar and bipolar ventricular electrograms were recorded (Fig 3). An external pacemaker (Medtronic model 5880) was then connected to the permanent electrode. During bipolar ventricular pacing, diaphragmatic contractions ceased (confirmed by fluoroscopy) only when the output was below 3.5 mA, although the localized chest wall muscle contraction and pacemaker sound persisted at that setting. The threshold for bipolar pacing was 3 mA. When the tip electrode became the cathode of a unipolar system, the pacing threshold was 10 mA, with persistence of diaphragmatic and intercostal contractions. When the proximal electrode became the cathode of a unipolar system, the pacing threshold decreased to 3 mA: such a unipolar system failed to produce diaphragmatic or intercostal muscle stimulation at current settings of 15 mA. Consequently, a Medtronic 5943 unipolar demand pulse generator was connected to the proximal electrode.

The patient subsequently remained well, free of abdominal twitching for over two years. The diaphragmatic and intercostal muscle contractions as well as the pacemaker sound have disappeared. The unipolar pulse generator was replaced after two years. At that time, the chest x-ray film findings, the pacing threshold and ventricular electrograms (Fig 3) were unchanged since the initial unipolarization.

COMMENT

Longstanding diaphragmatic contractions associated with cardiac pacing are often poorly tolerated. This problem may be corrected by several methods such as the use of low output pulse generators, repositioning of the catheter, phrenic nerve crush or conversion to an epicardial system. Unipolarization of an epicardial system has been described for this complication, but unipolarization of an endocardial system has not been reported before. Our case illustrates how the simple conversion of bipolar to unipolar pacing, when feasible, may eliminate diaphragmatic contraction in chronic perforation with stable catheter position. This approach appears preferable to the insertion of a second catheter. The longterm risk of thrombosis associated with two permanent pacing catheters in the heart is unknown, but the intermittent contact of active and inactive electrodes lying side-by-side may create false signals that may inhibit a demand pulse generator.

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Subacute Bacterial Endocarditis in a Patient with Isolated, Nonejection Systolic Click but without a Murmur*

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An 18-year-old college girl with an isolated, late systolic, nonejection click developed subacute bacterial endocarditis. Her course was uncomplicated except for the development of a late systolic murmur following the click. The importance of prophylaxis against endocarditis in patients with nonejection, systolic clicks, even in the absence of a murmur is emphasized.

The syndrome of a nonejection systolic click and late systolic murmur sometimes associated with abnormal T waves, occurring primarily in young women is well known.1-5 Subacute bacterial endocarditis has occasionally been associated with this syndrome,6-7 but only in one previous report8 has endocarditis developed in a patient with an isolated nonejection systolic click, ie, without the murmur.

We have recently observed a young woman with an isolated systolic click in whom subacute bacterial endocarditis developed.

CASE REPORT

An 18-year-old college girl was admitted to University Hospitals of Cleveland for evaluation of erythema and swelling of the right ankle of two days' duration associated with fever and chills. Malaise had been present for a few weeks. She had visited her dentist two and one-half months previously for a routine examination, including vigorous cleaning. No prophylactic antibiotics were given. Her father had died after his third myocardial infarction at 54 years of age, but her mother and three siblings were without cardiac disease. Routine examinations during childhood including cardiac auscultation never revealed cardiac abnormalities.

On admission she was febrile at 38.8°C and had a 3-4 cm area of erythema, swelling and tenderness below the right lateral malleolus. Examination of the heart showed the point of maximal impulse to be inside the midsclavicular line in the fourth intercostal space; the first and second heart sounds were normal; there was a distinct late systolic click heard best at the lower left sternal border and apex; no murmurs were heard by a number of observers.

Pertinent laboratory data included a white blood cell count of 7,200 with a normal differential count, erythrocyte sedimentation rate of 43 corrected to 38, hematocrit value of 39, negative urinalysis, C-reactive protein level of 1+, and an antistreptolysin 0 titer of 125. Three of six blood cultures drawn on three separate occasions grew Streptococcus viridans. X-ray film findings of chest and ankle were normal. The electrocardiogram was normal. An apical phonocardiogram before vasoactive maneuvers (Fig 1A) showed the prominent late systolic click but no murmurs. The apical phonocardiogram after intravenous administration of phenylephrine (Neo-Synephrine) (Fig 1B) showed an accentuated late systolic click with a short, late-systolic murmur beginning with the click.

The late systolic murmur again became inaudible after the phenylephrine effect had dissipated but could be precipitated by isometric exercise. On standing after amyl nitrite inhalation, the click disappeared but a prominent pansystolic murmur was heard at the apex.

The patient was treated with a four-week course of intravenously administered penicillin, resulting in prompt disappearance of her ankle pain and slight malaise. After two weeks of treatment with penicillin the click became less audible, but a late systolic murmur could then be heard at

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Figure 1A. Simultaneous recording of apical phonocardiogram (top), lead 2 electrocardiogram (middle), and carotid pulse tracing (bottom), before administration of phenylephrine. Prominent late systolic click is evident. No murmurs are present. B. Simultaneous recording of apical phonocardiogram (top) and electrocardiogram (bottom), after administration of phenylephrine. First and third cardiac cycles show accentuated late systolic click with short late systolic murmur. Second cardiac cycle shows click almost superimposed on second sound without murmur.