This case illustrates the value of coronary arteriography in patients with aortic valvular disease.\textsuperscript{6,7}

ACKNOWLEDGMENT: We thank Alexander Nedwich, M.D., who reviewed the pathologic specimen.

REFERENCES

An Unusual Precordial Pulse and Sound Associated with Large Pericardial Effusion*

Anthony J. Bonner, Jr., M.D.; Carlos M. Estevez, M.D.; James C. Dillon, M.D.; R. Joe Noble, M.D.; and Morton E. Tavel, M.D., F.C.C.P.

An unusual, high-pitched, early diastolic sound coinciding with a prominent, sharp precordial pulse was observed in a patient with a large chronic pericardial effusion. The pulse and sound coincided exactly with the anterior excursion of the heart within the fluid-filled pericardial sac, suggesting that the sound and pulse result from the ballistic effect of the heart striking the anterior pericardium and chest wall. This finding may be specific for large pericardial effusion with a “swinging heart.”

Physical findings associated with large pericardial effusion are often sparse and nonspecific.\textsuperscript{1-4} We have recently observed a patient with a large pericardial effusion who demonstrated an unusual early diastolic precordial pulsation associated with a high-pitched

\*From the Department of Medicine, Indiana University School of Medicine, and the Krannert Institute of Cardiology, Indianapolis.

Supported in part by the Herman C. Krannert Fund and by grants HL-06308, HL-05363, and HL-05749 from the National Heart and Lung Institute of the National Institutes of Health, Public Health Service.

This study was carried out during the tenures of Dr. Bonner and Dr. Estevez as Public Health Service Trainees in Cardiology.

Reprint requests: Dr. Tavel, Indiana University, 1130 West Michigan, Indianapolis 46202

Figure 1. Resected aortic valve from base to tip shows thickening due to hyalinization. Distal end is extremely cellular, with proliferation of young spindle-type cells. Subvalvular fibrous bump described by Bulpkey and Roberts\textsuperscript{3} is present.

The patient was readmitted to Hahnemann Hospital in April 1972, when he underwent aortic valve replacement with a No. 27 Bjork-Shiley aortic valve prosthesis; triple aortocoronary vein grafts were done to the right coronary artery, the left anterior descending artery, and the left circumflex coronary artery. His postoperative course was uncomplicated, and the patient was discharged on May 6, 1972 in good condition.

On Sept 21, 1973, the patient’s pacemaker failed, and he had to be readmitted for replacement of his pacemaker generator. During the time when the pacemaker was not functioning, sinus rhythm was present, with first-degree heart block.

Comment

The combination of complete heart block and aortic insufficiency has been regarded as rare in patients with rheumatoid spondylitis.\textsuperscript{1} However, subsequent reports\textsuperscript{3} have emphasized that this combination may not be as rare as previously thought. Our patient has an additional cardiac lesion, that is, coronary arteriosclerosis. Innumerable articles have been written describing the surgical approach to coronary arteriosclerosis by aorto-coronary bypass graft; but, so far as we know, this is the first case in which such surgery has been performed in a patient with rheumatoid spondylitis. Three patients in another series\textsuperscript{4} had angina pectoris; however, they had just undergone aortic valve replacement but did not undergo bypass grafting. Aortic valve replacement in rheumatoid spondylitis has been described.\textsuperscript{5} In the case of our patient, the aortic valve was replaced in addition to the bypass graft. One year after operation, the patient is asymptomatic.

Chest, 68: 6, December, 1975

Unusual Precordial Pulse 829
sound. These findings have been reported once previously; but the mechanism was obscure. Findings in the present case allowed us to make additional observations on this phenomenon and to suggest a mechanism. Although such a sound is probably rare, it may be specific for large pericardial effusion with a "swinging heart."

**CASE REPORT**

A 79-year-old white woman was referred to the Indiana University Medical Center for evaluation of a pericardial effusion. The patient gave a history of a "bad heart" of approximately 15 years' duration, having received treatment with digitalis preparations and diuretics for approximately the same period of time. Mild hypertension had persisted for an undetermined period of time. Cardiomegaly had been demonstrated by chest x-ray film for at least four years. The patient denied orthopnea, paroxysmal nocturnal dyspnea, chest pains, or severe dyspnea on exertion. There was no history of rheumatic fever, weight loss, or pulmonary tuberculosis. The accuracy of the historic information was limited because of senile dementia.

Physical examination revealed an elderly lady in no acute distress. She was uncooperative, had poor recall for recent events, and had rambling speech. Pertinent physical findings were limited to the cardiovascular system. The blood pressure was 150/60 mm Hg, and the pulse rate was 88 beats per minute and regular. The carotid pulses were full and equal bilaterally with a rapid upstroke. No bruits were audible. No abnormal jugular pulsations were noted. The chest was clear. Cardiac examination revealed a discrete point of maximal impulse located at the fifth intercostal space lateral to the midclavicular line. A prominent apical thrust was palpated immediately following the second sound. This was accentuated during expiration and with the patient in the left lateral decubitus position. The impulse was well localized and had a sharp tapping quality. The first heart sound was normal, and the second sound was physiologically split at the pulmonic area. A fourth heart-sound gallop was audible at the apex. A grade 2/6 harsh systolic ejection murmur which peaked in midsystole was audible at the aortic area and radiated to the carotids, left sternal border, and the apex. A grade 2/6 diastolic blowing murmur was heard along the left sternal border. Immediately following the second sound, a sharp high-pitched sound was heard best at the apex. This sound coincided with the apical thrust.

A chest x-ray film (Fig 1) showed considerable enlargement of the cardiac silhouette with normal pulmonary vascu-

![Figure 1](image1.png)

Figure 1. Chest x-ray film showing marked enlargement of cardiac silhouette without pulmonary venous engorgement.

![Figure 2](image2.png)

Figure 2. Sound recordings from pulmonic area (PA) and apical area (APEX) with simultaneous carotid pulse. With inspiration, diastolic sound (X) moves closer to S2. Aortic and pulmonic components (A2 and P2) can be identified in PA tracing during early inspiration. SM, Systolic murmur.

830 BONNER ET AL

CHEST, 68: 6, DECEMBER, 1975
larity. The electrocardiogram demonstrated nonspecific S-T segment changes and no electrical alternans. Intermediate-strength purified protein derivative of tuberculin and histoplasmin skin tests were negative.

Because the patient was most uncooperative, a diagnostic pericardiocentesis was not attempted. In view of her advanced age, the lack of demonstrable hemodynamic impairment, and the presence of a stable cardiac silhouette for at least four years, it was elected to manage the patient medically with the presumptive diagnoses of chronic idiopathic pericardial effusion, mild aortic insufficiency of undetermined cause, and senile dementia.

Phonocardiogram

Phonocardiography and external pulse recording showed an early systolic murmur, normal respiratory splitting of S2, and an intense high-pitched sound (X) at a variable interval after S2 (Fig 2). With the patient lying supine, the A2-X interval varied widely (0 to 0.08 second). In the left decubitus position, it was generally more widely split (0.07 to 0.09 second) than A2. The A2-X interval diminished slightly (0.02 second) with inspiration. The apex cardiogram recorded in both the left decubitus and supine positions (Fig 3) registered a large, peaked, early diastolic vibration whose onset was timed exactly with the onset of the diastolic sound. An exaggerated a wave coincident with an S4 sound was also present. The time constant for the apex cardiogram system was 1.8 seconds. The jugular pulse had moderately prominent a waves and an A2-V interval of 0.10 second (normal, 0.05 to 0.15 second), with a normal y descent.

Echocardiogram

An M-mode scan (Fig 4) confirmed the presence of a large anterior and posterior pericardial effusion with a "swinging heart," suggesting that perhaps the early diastolic pulse and sound could be caused by the apex of the heart swinging up to strike the anterior pericardium and chest wall. Simultaneous phonocardiographic, apex cardiographic, and echocardiographic recordings were obtained; the apex cardiogram and echocardiogram were recorded while positioning the transducers as near as possible to each other (Fig 5). The pulse and sound coincided exactly with the point at which the apex of the heart made contact with the chest wall. This point varied with respiration coincident with the sound and pulse.

Discussion

Several physical findings have been described in patients with large pericardial effusions. Extension of dullness significantly beyond the apex impulse, diminution of the apex impulse, reduction of the intensity of the heart sounds, and an alteration in intensity of heart sounds with position have all been reported. Many, if not most, patients with this problem show no specific findings.

![Figure 3](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20974/)

Figure 3. Sound recordings from apex and simultaneous apex cardiogram (ACG) showing effect of position on diastolic pulse and sound. Tracings were obtained serially within a minute of each other. SM, Systolic murmur.

![Figure 4](http://journal.publications.chestnet.org/pdfaccess.ashx?url=/data/journals/chest/20974/)

Figure 4. M-mode echocardiographic scan recorded from left sternal border location. Both anterior and posterior pericardial fluid (PF) is present. Note that heart swings anteriorly at end of each systole and apex appears to strike chest wall. LA, Left atrium.
Sakamoto et al. first reported this unusual early diastolic sound and pulse in a patient with massive pericardial effusion. They postulated that the pulse and sound were due to early diastolic filling analogous to the pulse and sound of pericardial constriction, even though at times these events were also coincident with the second heart sound in their patient. The early diastolic knock sound associated with constrictive pericarditis is split an average of 0.10 second from \( A_2 \) and has not been reported to coincide with \( A_2 \). Since diastole must follow \( A_2 \) by the isovolumetric relaxation interval, it seems most unlikely that early diastolic filling could have generated this unusual sound and pulsation.

Several authors have examined cardiac motion echocardiographically in patients with large pericardial effusions and have shown that the heart, attached to a pedicle formed by the great vessels and left atrium, swings rather freely within the pericardial sac. The heart may swing anteriorly on every beat (as in our patient), on every other beat, or in an irregular pattern.

Finding an exact time relationship between the sound, the pulse, and the anterior excursion of the apex of the heart on the echocardiogram leads us to believe that the sound and pulse are indeed caused by the ballistic effect of the apex of the heart swinging up and striking the anterior pericardium and chest wall. We believe that these signs, though rare, are probably specific for large pericardial effusion with a "swinging heart."

We are unsure exactly why this event is so rare. Ostensibly, the phenomenon occurs more commonly but is misinterpreted at the bedside to represent a pericardial knock. However, it would appear that in most very large effusions the heart does not swing up to meet the anterior pericardial sac, or at least does not do so with enough force to be palpable and audible. We examined echocardiographic records of 14 patients with large pericardial effusions with "swinging hearts" and were able to find only one in which the anterior wall of the heart may have contacted the anterior pericardium. The aortic insufficiency in our patient may have caused her heart to swing more vigorously and, thus, cause it to become palpable. We were unable to assess her aortic insufficiency echocardiographically because measurement of left ventricular dimensions in the presence of a "swinging heart" is believed to be hazardous.

It is possible that with careful recording of precordial impulses and sounds in more patients, we will be able to identify these phenomena in some who possess less impressive clinical signs.

ACKNOWLEDGMENT: We wish to express our appreciation to Dr. Douglas P. Zipes for allowing us to study the patient described in this report and to Mrs. Ronald Stewart for performing the graphic studies.

REFERENCES

2. Harvey WP: Auscultatory findings in diseases of the pericardium. Am J Cardiol 7:15, 1961

832 BONNER ET AL

CHEST, 68, 6, DECEMBER, 1975

![Figure 5. Simultaneous sound and apical pulse recordings and echocardiogram recorded with transducers close together. Diastolic sound and pulse coincide with point of contact of heart with chest wall, as demonstrated by echocardiogram. ACG, Apex cardiogram; SM, systolic murmur; PF, pleural fluid.](http://journal.publications.chestnet.org/pdф access.ashx?url=/data/journals/chest/20974/ on 04/15/2017)