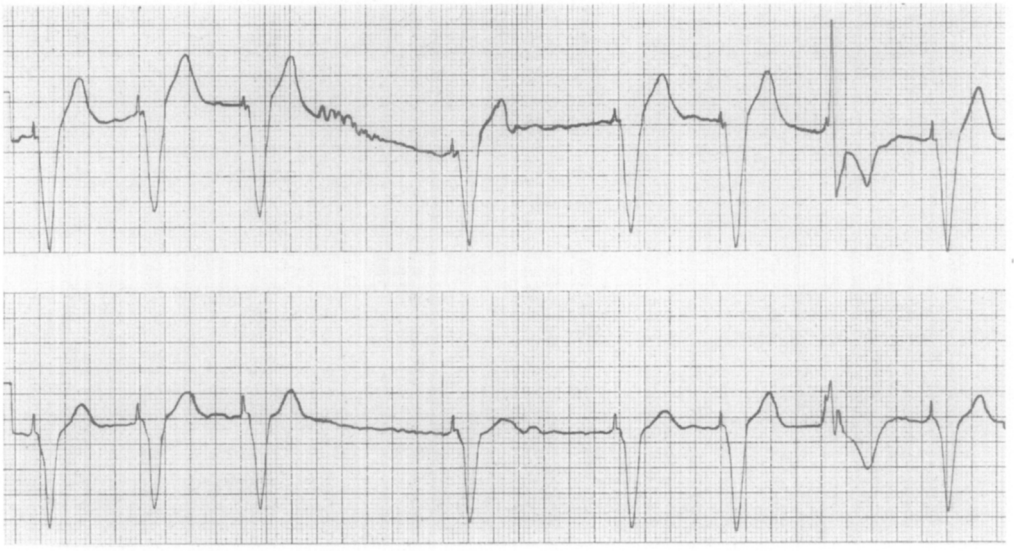

Fig. 1. Strip from Holter monitor (two-lead system). Pacemaker inhibition is associated with a myopotential "artifact." This is best seen in the upper lead, after the third paced beat.

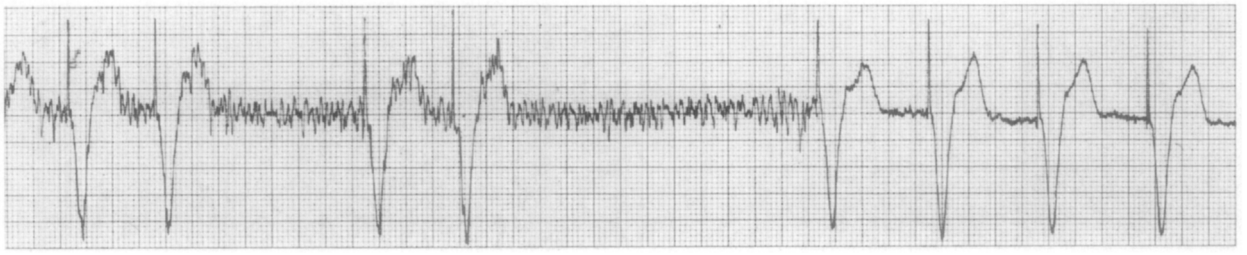

Fig. 2. Myopotentials and pacemaker inhibition recorded during isometric exercise, under direct electrocardiographic monitoring.

asymptomatic after the unipolar lead was changed to a bipolar system.

Monitoring artificial pacemaker function is an important application of ambulatory electrocardiography. Of 48 patients with suspected pacemaker malfunction reported by Iyengar et al., 24-hour monitoring detected failure in nine in whom routine examination, standard electrocardiograms, and pacemaker evaluation clinics failed to detect the malfunction.[2] One type of malfunction to which unipolar demand ventricular pacemakers are vulnerable is inhibition by skeletal muscle potentials. This phenomenon, if suspected, can be provoked by isometric arm exercise in up to 85% of patients with unipolar lead systems.[3-5] The recording of myopotential inhibition by ambulatory electrocardiography, however, has not been previously documented. We appreciated this cause of pacemaker failure in our patient, only after we more carefully inspected a Holter monitor "artifact," originally thought to be insignificant.

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Pericardial effusion swinging heart phenomenon by dynamic radionuclide ventriculography

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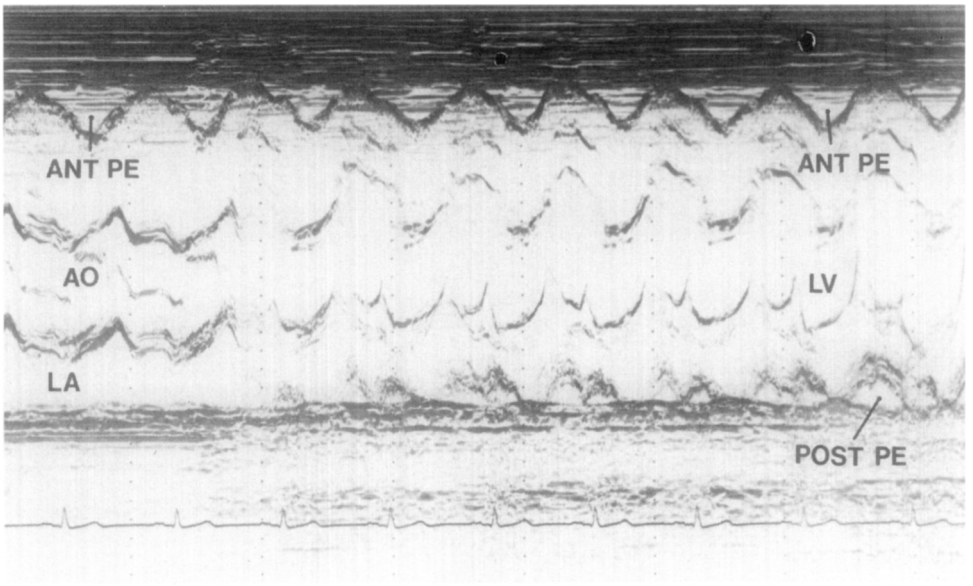


Fig. 1. M-mode echocardiogram demonstrating anterior and posterior pericardial effusion *(PE)*. The effusion is not visualized behind the left atrium *(LA)*. *LV* = left ventricle; *AO* = aorta.

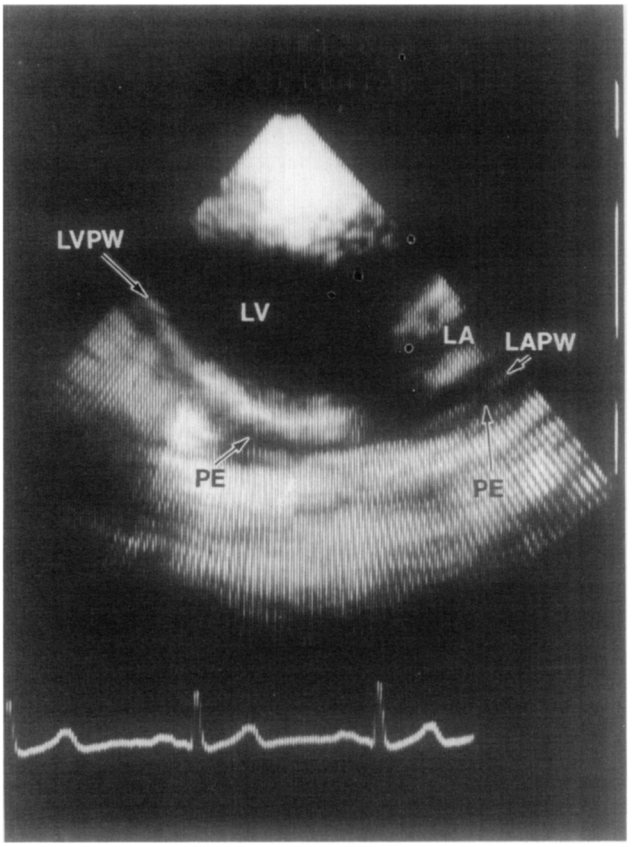
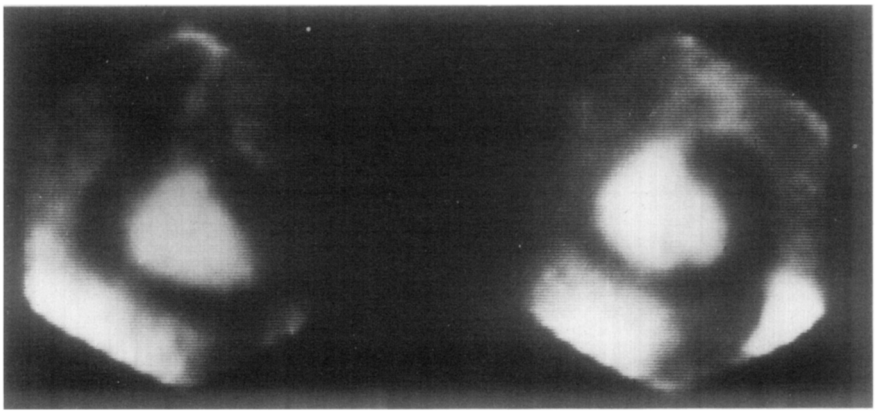


Fig. 2. Extension of pericardial effusion *(PE)* behind the left atrium *(LA)* is demonstrated by two-dimensional echocardiography. *LV* = left ventricle; *LAPW* = left atrial posterior wall; *LVPW* = left ventricular posterior wall.

Pericardial effusion was first detected by static radionuclide blood pool imaging in 1952 by Rejali et al.[1] Since that time there have been several studies describing its characteristics by nongated radionuclide techniques. The phenomenon of swinging heart in patients with pericardial effusion has not been previously reported with radionuclide ventriculography.

This 24-year-old man was in good health until December, 1979, when he noted a painless right cervical lump. He was otherwise asymptomatic. Physical examination revealed generalized lymphadenopathy. Blood pressure measured by cuff was normal without paradoxic pulse. There was no jugular venous distention. Palpation and percussion of the chest revealed decreased precordial activity and increased dullness which extended laterally to the anterior axillary line. Heart sounds were normal. Following cervical lymph node biopsy, the diagnosis of diffuse lymphoblastic lymphoma was made. Chest x-ray showed clear lung fields with marked cardiomegaly. M-mode echocardiography (echo) as shown in Fig. 1 revealed a large pericardial effusion with an exaggerated and abnormal motion of the cardiac chambers. The posterior echo-free space did not extend behind the left atrium. Two-dimensional echo (Fig. 2) confirmed the presence of pericardial effusion with excessive cardiac motion, and showed extension of the posterior echo-free space behind the left atrium.

Equilibrium radionuclide ventriculography (Fig. 3) revealed a wide photon deficient area around the blood pool image, extending superiorly to the atrioventricular valve plane. Septal thickness was normal. A distinct swinging motion of the cardiac blood pool could be seen on dynamic display. During ventricular systole, the cardiac chambers moved inferiorly and slightly posterolaterally.



RAO LAO

Fig. 3. Right anterior oblique *(RAO)* and left anterior oblique *(LAO)* views of radionuclide ventriculogram at end-diastole. Markedly increased photon deficiency due to pericardial effusion is visualized in both views around the blood pool image.

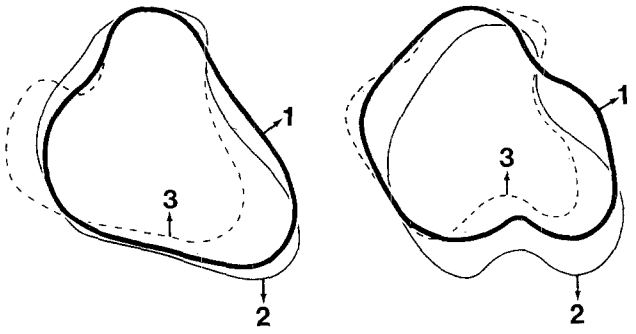


Fig. 4A. This drawing illustrates the end-diastolic *(heavy line–1),* end-systolic *(thin line–2),* and early diastolic *(broken line–3)* cardiac position in swinging heart phenomenon. *RAO* = right anterior oblique; *LAO* = left anterior oblique.

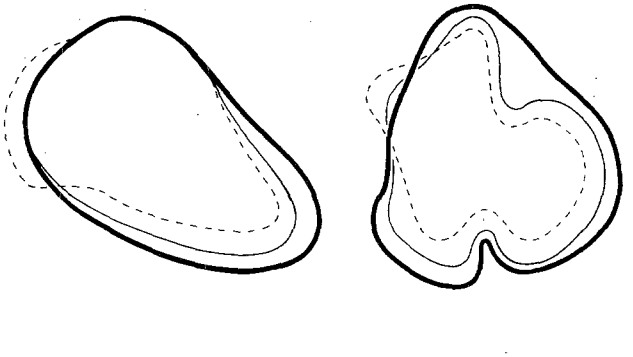


Fig. 4B. Normal end-diastolic *(heavy line),* early systolic *(thin line),* and end-systolic *(broken line)* cardiac motion. Abbreviations as in Fig. 4A.

At the beginning of ventricular diastole, this motion had reversed with the cardiac chambers moving superiorly and anteromedially (Fig. 4*A*). During the last third of ventricular diastole, the heart gradually returned to its base line end-diastolic position. (Fig. 4*B* illustrates the normal cardiac motion for comparison). The resting left ventricular ejection fraction was 0.66.

The presence of pericardial effusion on static imaging is characterized by (1) a small cardiac blood pool, (2) a discrepancy between cardiac size on x-ray and blood pool size on scintiscan, (3) a zone of photon deficiency between the heart and lungs, and (4) a separation of the cardiac blood pool from the hepatic vasculature.[2] These signs are not specific for pericardial effusion, however, and may also be seen with pericardial thickening, intrapericardial masses, ventricular hypertrophy, and intracardiac blood clots. The characteristic appearance of the swinging heart phenomenon on dynamic display in this patient is unique and should be added to the radionuclide criteria for pericardial effusion. In contrast to previously mentioned signs, this phenomenon although not frequent is specific for the presence of intrapericardial fluid and should be carefully searched for whenever the presence of pericardial effusion is suspected on gated radionuclide ventriculography. In adults the swinging heart is seen mostly in patients with malignant, large pericardial effusion. This phenomenon has been attributed to loss of the restraining effect of the pericardium associated with a minimum of adhesions, chronic fluid accumulation, and/or the presence of sanguinous effusion.[3] In conclusion, determination

of cardiac motion should be part of the radionuclide evaluation of patients with suspected pericardial effusion in order to detect the swinging heart phenomenon.

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Right ventricular outflow obstruction in acute myocardial infarction

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The differential diagnosis of a new systolic murmur with acute myocardial infarction includes perforation of the interventricular septum, papillary muscle dysfunction or rupture, or left ventricular dilatation with resultant mitral regurgitation. This report describes circumstantial evidence for a potential new cause of systolic murmur apparently due to the development of partial obstruction across the right ventricular outflow tract associated with the infarction process.

A 63-year-old hypertensive white male presented with an acute anteroseptal wall myocardial infarction, complicated initially by pulmonary congestion. He improved with diuretics, but on the second hospital day developed a new grade 2/6 systolic ejection murmur at the mid-left sternal border. Swan-Ganz catheterization under fluoroscopy revealed an 18 mm Hg gradient within the right ventricle (Fig. 1) with the balloon inflated avoiding catheter entrapment. The wedge pressure was 9 mm Hg without prominent V wave or right-heart oxygen step-up. An M-mode echocardiogram showed paradoxical septal motion and a small right ventricle. The visualized portions of the aortic and mitral valves appeared normal. The patient improved with resolution of symptoms of heart failure, but the systolic murmur persisted. The patient died suddenly at home 4 weeks following admission without postmortem examination.

Right ventricular gradients can occur in subpulmonic stenosis which may be infundibular (discrete) or subinfundibular due to anomalous muscle bundles. Hypertrophic cardiomyopathy may have infundibular right ventricular outflow gradients in 15% of cases[1] or more with dynamic maneuvers,[2] but only rarely in the absence of left ventricular outflow obstruction.[3] Aortic valve disease may have a gradient between the body of the right ventricle and pulmonic valve. Spontaneous closure of congenital ventricular septal defects (VSD) may be associated with septal aneurysm formation[4] and a case with right ventricular obstruction with abnormal septal motion has been reported.[5] Abnormal septal motion also has been described in anterior wall aneurysms following acute myocardial infarction (AMI). We hypothesize a new septal aneurysm in our patient. The absence of prior murmur and absence of ECG evidence of either right or left ventricular hypertrophy supports this as being related to the patient's infarction, rather than other acquired or congenital heart disease. The evaluation of a new murmur following infarction should, therefore, include right ventricular obstruction as a possibility. Two-dimensional echocardiography may help determine the occurrence and frequency of acquired septal aneurysm without VSD due to AMI.

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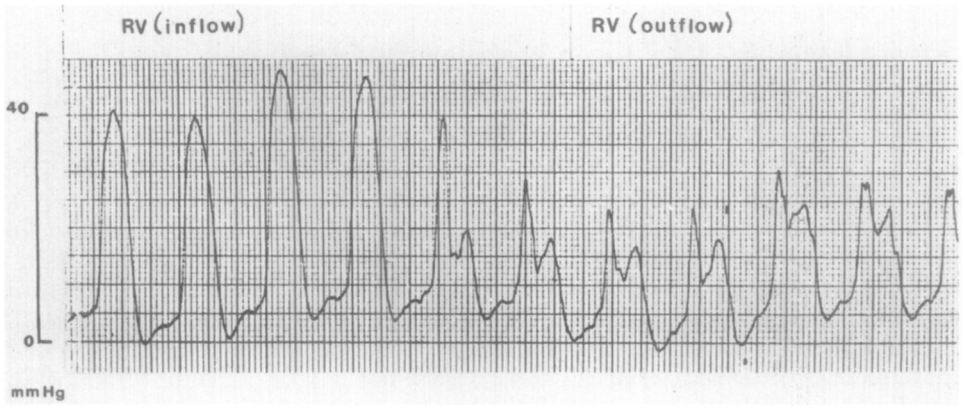

Fig. 1. Continuous pressure recording from right ventricle *(rv)* inflow tract (body) to outflow tract.

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