adds more information about the lesion showing the site of rupture through the detection in that zone of a continuous gradient between the aorta and inside the aneurysm. These findings were confirmed by aortography and surgery.

TRA has a very bad short-term prognosis and for this reason, in view of clear clinical or roentgenographic signs, urgent aortography must be performed. However, in those cases in which signs are not evident, or even in all cases of severe thoracic contusion in which aortography is not indicated, a Doppler echocardiographic study could help to detect those patients in whom the disorder may otherwise be undiagnosed, with all the therapeutic implications this involves.

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Transient Mitral Regurgitation Due to Mitral Valve Prolapse Accompanied by Systolic Anterior Motion of the Mitral Valve*

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A grade 4/6 systolic murmur, systolic anterior motion of the mitral valve (SAM), and severe mitral regurgitation (MR) documented by two-dimensional Doppler echocardiography developed suddenly on the structurally normal heart of a patient with idiopathic portal hypertension. The patient did not have signs of congestive heart failure and the aforementioned phenomenon disappeared completely when the patient was in hepatic failure. This could be explained by a change in circulating blood volume either by gastrointestinal hemorrhage or hepatic failure.

(Chest 1990; 98:1017-19)

HOCM = hypertrophic obstructive cardiomyopathy

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Systolic anterior motion of the mitral valve is not specific for hypertrophic obstructive cardiomyopathy but sometimes documented in hypovolemia or pheochromocytoma. We experienced a case of transient SAM and severe MR documented by two-dimensional echocardiography and two-dimensional Doppler echocardiography.

**CASE RECORD**

The patient was a 66-year-old woman who was admitted to this hospital for the evaluation of idiopathic portal hypertension. She had no history of heart disease. During hospitalization, owing to a grade 4/6 systolic murmur, which had not been seen before but developed suddenly accompanied by palpitation, the patient was referred to our department. She was well nourished and appeared well. On physical examination, the blood pressure was 100/70 mm Hg and the pulse rate was 100 beats per minute. A carotid upstroke was not bisferiens and a jugular vein was visible with prominent A wave. A grade 4/6 pansystolic murmur was heard at the apex and was transmitted to the back and axilla. No S$_2$ or S$_3$ was heard. A large spleen was palpable but liver was not felt. Chest x-ray film was unremarkable, and an ECG showed nonspecific ST and T wave changes.

A hypercontractile nonhypertrophied small left ventricle and SAM with posterior mitral valve prolapse were seen by two-dimensional echocardiography. Severe MR and a mosaic signal in the left ventricular outflow tract were shown by two-dimensional Doppler echocardiography (Fig 1). As the general condition was stable and there were no signs of congestive heart failure, no specific therapy was introduced. The intensity of the systolic murmur varied from one day to another and disappeared completely in two weeks. At that time, all findings seen by echocardiography disappeared completely. The left ventricular internal diastolic dimension and the percent fractional shortening were 39 mm and 46 percent, respectively, when a grade 4/6 systolic murmur was heard, and 43 mm and 34 percent, respectively, when the murmur disappeared completely (Fig 2). The wall thickness of the left ventricle was within normal limits.

The plasma catecholamine value was normal on three occasions. There was no correlation with the intensity of the murmur and hematocrit value. When the systolic murmur disappeared completely, the patient's condition followed sclerotherapy for esophageal varices, and she suffered hepatic failure and developed generalized edema and ascites. At that time, amyl nitrate administration could induce a grade 3/6 systolic murmur with SAM but there was no mitral valve prolapse and MR. The blood velocity of the left ventricular outflow tract was 1.3 meter/s and of a late peaking type.

**DISCUSSION**

This patient was characterized as follows: first, the sudden onset of severe systolic murmur accompanied by SAM, posterior mitral valve prolapse, and MR was seen. Second, the intensity of the murmur varied from one day to another and when the murmur disappeared completely, the above findings seen by echocardiography also disappeared. Cardiac hypertrophy was not present. Third, the underlying disorder of this patient was idiopathic portal hypertension.

One of the possible causes of SAM and septal contact is the result of the Venturi effect due to rapid ejection in HOCM. The SAM is not specific for HOCM but occasionally was reported in cases of hypovolemia and pheochromocytoma. Experimentally, amyl nitrate or catecholamine induces a pressure gradient between the aorta and left ventricle. So, SAM may occur when the left ventricle is small and hyperkinetic, even if there is no cardiac hypertrophy.

In this case, the left ventricular cavity was small and hypercontractile when systolic murmur was heard, which supports the above hypothesis. The cause of mitral regur-
The Effect of Prolonged Hypothermia on Cardiac Function in a Young Patient with Accidental Hypothermia

Yoram Maaravi, M.D.; and A. Teddy Weiss, M.D.

A 20-year-old man had accidental, prolonged, and severe hypothermia. Serial radionuclide ventriculography disclosed reduced myocardial contractility during hypothermia that resolved after warming. The effects of hypothermia on cardiac function are discussed. (Chest 1990; 98:1019-20)

Accidental hypothermia, defined as a core body temperature of less than 35°C, is a well-known complication of exposure associated with a variety of disorders with complex pathophysiology. This condition has profound effects on multiple body systems, including the cardiovascular, respiratory, central nervous, metabolic, and endocrine systems. Since accidental hypothermia carries a high mortality rate due to circulatory failure, it is important to recognize, very accurately, the cardiovascular state of this condition. Unfortunately, the paucity of information on cardiac function during hypothermia, especially in the human, is mostly contradictory. The introduction of radionuclide ventriculography into clinical medicine has provided us with one of the most accurate noninvasive techniques of assessing global regional wall motion and cardiac function in critical circulatory disorders, as well as in accidental hypothermia.

We describe below a young man with accidental hypothermia in whom serial bedside radionuclide ventriculography disclosed reduced global and regional myocardial contractility during hypothermia, that improved promptly after rewarming.

CASE REPORT

A 20-year-old man was admitted to the hospital during the winter months because of accidental hypothermia. He was well until the day of admission, when he was found stuporous in an open field after apparently lying there for at least 12 hours. At the time of hospital admission he appeared somnolent. Rectal temperature was 28°C, blood pressure was 144/76 mm Hg, pulse rate was 45 beats per minute and regular, and respirations were 24/min. Pupils were dilated, equal, and reactive. Neurologic examination revealed disorientation of time and place. Results of the rest of the physical examination were normal. Hemogram and coagulation test results were normal. Serum sodium level was 138 mEq/L, potassium was 3.7 mEq/L, and urea nitrogen was 32.2 mg/dL. The glucose level,

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