two stated that good dental hygiene was present on examination. In five cases, authors mention the possibility of dogs being the source. However, in 1966 Page and King⁴ refuted dogs as the source, stating that organisms referred to them from dog bites had never been \textit{Haphrophilus} and that the majority had been \textit{Pasteurella multocida}, which resembles \textit{Haphrophilus} in being a gram-negative coccobacillus with bipolar staining, but has differing cultural characteristics.¹²

In a study by Kraut et al,¹³ \textit{Haphrophilus} was isolated from the oral flora of 35.5 percent of 45 healthy adult volunteers. Mesko⁶ and Page and King⁴ reported cases of human bites leading to soft-tissue infections by \textit{Haphrophilus}. In our patient, the organism was not isolated from gingival scrapings but was grown from dental plaque, despite four weeks of antibiotic therapy. This suggested persistence of the organism in an avascular focus (plaque), which we believe made corrective periodontal surgery imperative, a view supported by periodontists.¹⁴

With the prevalence of \textit{Haphrophilus} in human oral flora, the frequency of periodontal disease or dental manipulation in reported cases of endocarditis due to this organism and the demonstration of a primary focus in the dental plaque of our patient, we believe that an oral source should be assumed in cases of endocarditis due to \textit{Haphrophilus}. Therefore, management of these cases should be similar to that for \textit{Streptococcus viridans} endocarditis, with a deliberate search for a correctable primary oral focus of infection.

**References**


**Left Ventricular Outflow Obstruction Induced by Tamponade in Hypertrophic Cardiomyopathy**

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Echocardiographic abnormalities of valvular movement described in patients with pericardial effusions have included systolic anterior motion (SAM) of the mitral valve. Published illustrations have shown, however, "pseudo-SAM" rather than true SAM. We report a patient with asymmetric septal hypertrophy whose echocardiogram during tamponade showed true SAM, which was no longer apparent and could not be provoked following resolution of tamponade. Two prior cardiac catheterizations revealed no intraventricular pressure gradients in either normal or postextrasystolic beats. Tamponade was the only stimulus that provoked signs of obstruction in this patient with asymmetric septal hypertrophy.

Movement abnormalities of cardiac valves have been recognized echocardiographically in patients with large pericardial effusions.¹² These have included mitral valve prolapse, premature aortic valve closure, mid-systolic pulmonic valve notching, changes in mitral valve DE amplitude and EF slope,¹ and systolic anterior movement (SAM) of the mitral valve.³

Nanda et al described four patients, without asymmetric septal hypertrophy, who had SAM of the mitral valve in association with pericardial effusion. The velocity of early anterior systolic mitral valve movement, however, was less than the corresponding velocity of posterior wall movement, suggesting a pseudo-SAM rather than true SAM. Pseudo-SAM, which represents an exaggeration of the normal anterior systolic movement of the mitral valve apparatus,⁴ is not indicative

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of left ventricular outflow obstruction. In contrast, true SAM, characterized by an abrupt anterior movement out of proportion to posterior wall movement, has been correlated with dynamic left ventricular outflow tract obstruction.4.5

We describe a patient with a large pericardial effusion and tamponade who showed true SAM and premature aortic valve closure, characteristics of dynamic left ventricular outflow obstruction. Following resolution of the tamponade, but not of the effusion, SAM and premature aortic valve closure were no longer present. Subsequent echocardiographic studies, including during inhalation of amyl nitrite and performance of the Valsalva maneuver, revealed asymmetric septal hypertrophy but no findings suggestive of dynamic left ventricular outflow tract obstruction.

CASE REPORT

A 59-year-old man with a history of prior anteroseptal myocardial infarction and prior catheterization—documented two-vessel coronary artery disease was admitted to Beth Israel Hospital with unstable angina. Cardiac examination revealed blood pressure of 145/90 mm Hg and no heart murmur. Repeated coronary angiography was unchanged. No intraventricular or transaortic pressure gradients were present in normal or postextrasystolic beats, and Brockenbrough's sign4 was absent on pressure tracings from both cardiac catheterizations. The left ventriculograms did not suggest dynamic left ventricular outflow tract obstruction.

Coronary bypass surgery was performed. The chest was closed in a standard fashion, leaving the pericardium opened. The initial postoperative course was stable. On the seventh postoperative day, the patient noted chest pain, fatigue, and shortness of breath. An enlarging cardiac silhouette was present on chest x-ray film on the 14th postoperative day. Coumadin therapy, begun on the second postoperative day, was discontinued. Physical examination revealed a blood pressure of 120 mm Hg jugular venous distention, and a grade 2/6 ejection-quality murmur. On ECG, voltage was decreased in all leads. An echocardiogram performed with the patient receiving no pressor agents revealed a large pericardial effusion, with an anterior echo-free space and a posterior echo-free space measuring 5 cm (Fig 1A). There was no apparent swinging movement of the heart. True SAM of the mitral valve apparatus to abut the interventricular septum and mid-systolic aortic valve closure, followed by reopening, were present (Fig 1A and 1B). The interventricular septum could not be adequately visualized below the tips of the mitral valve leaflets, but was not hypertrophied relative to the posterior wall in the region seen.

A Swan-Ganz catheter was placed in the pulmonary artery, and pericardiocentesis was performed, with removal of 775 ml of sanguinous fluid. Systolic arterial pressure immediately increased to 140 mm Hg, pulmonary arterial diastolic and pulmonary capillary wedge pressures fell from 33 to 15 mm Hg, and the previously elevated central venous pressure of greater than 18 mm Hg fell to 9 mm Hg. The patient's symptoms resolved and his murmur disappeared. Subsequent echocardiography (Fig 2) showed residual posterior pericardial effusion, but SAM of the mitral valve and mid-systolic aortic valve closure were no longer apparent.

Repeated examinations were performed seven months after surgery. No cardiac murmur was detectable at rest, during performance of the Valsalva maneuver or following inhalation of amyl nitrite. On carotid pulse tracing, the ejection time index, corrected for heart rate according to the method of Weisler et al.,7 was 0.41 sec and did not change following inhalation of amyl nitrite. No pericardial effusion was demonstrable by echocardiogram. SAM of the mitral valve and mid-systolic aortic valve closure were absent during the Valsalva maneuver and following inhalation of amyl nitrite. The septum was hypertrophied (measuring 24 mm) relative to the left ventricular posterior wall (11

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**Figure 1.** Echocardiograms recorded during tamponade. Mid-systolic aortic valve closure (black arrows, Fig 1A) and systolic anterior motion of mitral valve (black arrows, Fig 1B) in setting of large posterior pericardial effusion. Abbreviations: ECG = electrocardiogram; AV = aortic valve; LA = left atrium; MV = mitral valve; PE = pericardial effusion; SAM = systolic anterior motion of mitral valve.
Ficuna 2. M Mode sweep from base toward apex of heart performed following pericardiocentesis; patient monitored by Swan-Ganz catheter. Residual pericardial effusion, but mitral valve in area of left ventricular outflow tract shows no systolic anterior motion. Reverberations from Swan-Ganz catheter located in right ventricular outflow tract are seen in the left atrium. Aortic valve not optimally seen in this scan. When best visualized, no mid-systolic aortic valve closure was apparent. Abbreviations: AV = aortic valve; ECG = electrocardiogram; LA = left atrium; MV = mitral valve; PE = pericardial effusion; SG = Swan-Ganz catheter.

mm) in the dimensional area (Fig 3). The septum was thinner at the base of the heart. The appearance of an asymmetrically hypertrophied septum, with maximal septal thickness toward the apex, was confirmed by cross-sectional echocardiography (Fig 4).

Discussion

In prior reports, echocardiographically detected mitral valve movement abnormalities associated with large pericardial effusions have been attributed to

Figure 3. Left ventricular echocardiogram performed with inferolateral transducer angulation seven months after discharge. Asymmetric septal hypertrophy (2.2:1 septal to LV posterior free wall ratio). Pericardial effusion disappeared. Abbreviations: ECG = electrocardiogram; Endo = endocardium; IVS = interventricular septum; LV = left ventricle; Peri = pericardium; RV = right ventricle.

Figure 4. Cross-sectional echocardiogram performed 14 months postoperatively. Long-axis view: disproportionate lower septal hypertrophy. Triangular shape of septum with thicker portion lying inferiorly. Abbreviations: AO = aortic root; LA = left atrium; LV = left ventricular cavity; MV = mitral valve; SEP = septum.

112 SCHULMAN ET AL

CHEST, 80: 1, JULY, 1981
swinging and rotary motion of the heart within a fluid-filled pericardial sac.\textsuperscript{5,6,8} This explanation could account for pseudo-SAM, but not for true SAM, in which the velocity of early systolic movement exceeds that of the posterior left ventricular wall.

True SAM of the mitral valve to abut the septum and mid-systolic aortic valve closure correlate with dynamic left ventricular outflow tract obstruction.\textsuperscript{5,5,8} Their presence during tamponade and disappearance following relief of tamponade, but not of the large pericardial effusion, in our patient suggests tamponade-induced left ventricular outflow tract obstruction. Subsequently, neither a murmur nor echocardiographic changes of dynamic obstruction could be induced by the Valsalva maneuver or by inhalation of amyl nitrite. In addition, the left ventricular ejection time did not change following inhalation of amyl nitrite, in contrast to the expected increase in patients with dynamic obstruction.\textsuperscript{10} Finally, the potent inotropic effect of postextrasystolic potentiation, shown to be a sensitive provocative test for idiopathic hypertrophic subaortic stenosis,\textsuperscript{11} had failed to elicit obstructive physiology in our patient during two prior cardiac catheterizations. The effect of tamponade seems to have been a more sensitive provocative maneuver than postextrasystolic potentiation, inhalation of amyl nitrite, or performance of the Valsalva maneuver.

It is possible that the difficulty in inducing dynamic left ventricular outflow tract obstruction in our patient with hypertrophic cardiomyopathy may be related to the configuration of the interventricular septum. Autopsy\textsuperscript{12,13} and cross-sectional echocardiographic studies\textsuperscript{13,14} from patients with idiopathic hypertrophic subaortic stenosis have demonstrated a variety of patterns of asymmetric septal hypertrophy. Signs of dynamic outflow obstruction, such as SAM of the mitral valve, appear to be more frequent in patients with predominant subaortic asymmetric septal hypertrophy\textsuperscript{14} and less common, as in our patient, in those patients in whom the apical septum is greatly hypertrophied.

Two major mechanisms may influence the development of obstructive physiology in tamponade. First, increased sympathetic activity may increase myocardial contractility and, therefore, outflow obstruction, a mechanism that has been postulated to be responsible for the SAM that may be observed in patients with hypercontractile cardiac stases independent of asymmetric septal hypertrophy.\textsuperscript{15,16} Second, decreased left ventricular filling resulting in decreased left ventricular end-diastolic volume may further promote outflow obstruction in the setting of increased myocardial contractility.

While therapy for cardiac tamponade is rapid evacuation of pericardial fluid or open drainage, isoproterenol,\textsuperscript{17} nitroprusside, and hydralazine\textsuperscript{18} infusions have been advocated as potential temporizing measures. In a patient with asymmetric septal hypertrophy, it is possible that those agents could result in clinical deterioration by worsening dynamic outflow obstruction. Although pseudo-SAM in a patient with pericardial effusion may be an artifact of altered cardiac motion in a fluid-filled pericardium, true systolic anterior motion should alert the examiner to the possibility of dynamic left ventricular outflow obstruction.

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CHEST, 80: 1, JULY, 1981

LEFT VENTRICULAR OUTFLOW OBSTRUCTION 113