diagnosis" of Aschoff bodies at necropsy, using loose criteria which may include nonspecific inflammatory lesions of the heart.

Our patient had a classic congenitally bicuspid aortic valve and numerous classic Aschoff bodies in the heart at necropsy. To our knowledge, this combination has neither been clearly documented nor illustrated previously.

REFERENCES
7 Vermani R, Roberts WC: Incidence and significance of Aschoff bodies in atrial appendages removed at operation: an analysis of 800 patients. (In preparation)

Persistence of the Third Heart Sound after Resection of the Native Mitral and Tricuspid Valves

Evidence Against the Valvular Theory of Third Sound Origin

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A patient is described with severe rheumatic mitral and tricuspid insufficiency in whom both atrioventricular valves with their chordae tendineae and papillary muscles were resected and replaced with Hancock porcine grafts. This would appear to be the second such patient reported and the first described in detail in whom the third heart sound persisted postoperation. This occurrence documents the fact that a third heart sound can occur in the absence of native atrioventricular valve leaflets and the major portions of the subvalvular supporting apparatus, and argues against the theory that the third heart sound is generated by either the valvular leaflets or the subvalvular apparatus.

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For many years, a controversy has existed concerning the mechanism of production of the third heart sound or ventricular gallop. Findings of the patient reported herein are of considerable interest regarding that question.

Case Report

A 23-year-old Korean woman with a past history of rheumatic fever was admitted to Cook County Hospital because of class 3 symptoms of left ventricular failure. Physical examination revealed a diffuse hyperdynamic apex beat in the sixth left intercostal space, anterior axillary line. A prominent parasternal heave was present. The first heart sound was diminished at the apex. The pulmonary closure sound was markedly increased. A very loud third heart sound was heard and recorded at the apex of the left ventricle. A grade 3/6 holosystolic blowing murmur was heard at the apex and radiated to the axilla and left sternal border. A short diastolic rumble followed the third heart sound. There was no jugular venous distention, but prominent V waves were seen. Arterial pulses were normal. An electrocardiogram revealed atrial fibrillation and left ventricular hypertrophy.

Cardiac catheterization and left ventricular angiography confirmed severe mitral regurgitation with pulmonary hypertension (Table 1). Trivial aortic insufficiency was noted on retrograde ascending aortography.

On February 18, 1975 the patient underwent elective mitral valve replacement. Severe tricuspid regurgitation found at surgery necessitated additional tricuspid valve replacement. The chordae tendineae and papillary muscles were resected along with the valve leaflets in both ventricles. The mitral and tricuspid valves were replaced with stented Hancock porcine heterograft valves with 31 and 33 mm diameter rings, respectively.

Two months after surgery, on April 16, 1975, with the patient still in atrial fibrillation, phonocardiography was repeated and auscultation of the heart was carefully performed by three cardiologists. At that time again a left ventricular third heart sound was easily audible as was a short diastolic rumble initiated by the third heart sound. A holosystolic murmur was audible at the apex of the left ventricle. As shown in Figure 1, the third heart sound, rapid filling wave of the apexcardiogram, holosystolic murmur and mid-diastolic rumble were all diminished in intensity compared to the preoperative state. Figure 2 shows the postoperative chest film with both valve stents in place. The patient’s heart size was noted to have diminished considerably postoperation and her symptoms had markedly improved. For this reason, a decision was made to follow-up the patient carefully without repeating cardiac catheterization. This decision was also influenced by a desire to avoid passing a catheter across the heterograft valve in the early postoperative period. At the time of this writing the patient continues to do well.

Discussion

Three theories have been suggested to explain the genesis of the third heart sound. The oldest theory

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Table 1—Preoperation Catheterisation Data

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrium</td>
<td>10 (V = 16)</td>
</tr>
<tr>
<td>Right ventricle</td>
<td>87/7</td>
</tr>
<tr>
<td>Pulmonary artery</td>
<td>87/34 (50)</td>
</tr>
<tr>
<td>Pulmonary arterial wedge</td>
<td>25 (V = 53)</td>
</tr>
<tr>
<td>Left ventricle</td>
<td>103/16</td>
</tr>
<tr>
<td>Aorta</td>
<td>103/60 80</td>
</tr>
<tr>
<td>Cardiac index (Fick)</td>
<td>3.3L/min/M²</td>
</tr>
</tbody>
</table>

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Figure 1. Electrocardiogram (ECG), phonocardiogram (PCG) and apexcardiogram (ACG) showing holosystolic murmur (HSM) and a loud third heart sound (3) coinciding with rapid filling wave of apexcardiogram. Postoperative tracing to the right shows continued presence of a prominent third heart sound and marked decrease in the systolic murmur. HF = high frequency; LF = low frequency.
suggested first by Potain\textsuperscript{1} in 1900 and subsequently supported by several workers including Kuo\textsuperscript{2} and colleagues in 1957 and Crevasse et al\textsuperscript{3} in 1962, is that the sound is generated in a ventricle at the conclusion of rapid passive filling. The second theory, proposed by Gibson\textsuperscript{4} in 1907 and Thayer\textsuperscript{5} in 1909 is that mitral valve closure in diastole produces the sound. This has subsequently been challenged by many authors and was rejected by Fleming.\textsuperscript{6} The third theory proposed by Dock and associates\textsuperscript{7} in 1955 and supported by Nixon\textsuperscript{8} in 1961 and Fleming\textsuperscript{6} in 1969 is that the sound is generated by the stretching taut of the subvalvular apparatus-papillary muscles and chordae tendineae.

The study of patients who have had an atroventricular valve replaced would be expected to shed light on this problem. Early observation of patients with mitral ball valve prostheses in place showed that even in the presence of severe volume overload, a third heart sound was rarely if ever heard, favoring a subvalvular or valvular mechanism for the production of a third heart sound.\textsuperscript{8,9,10,11} Subsequent studies\textsuperscript{12} have, however, shown that a third heart sound is not uncommonly present in a patient whose mitral valve has been replaced with a homograft or heterograft valve and who has severe mitral and/or aortic insufficiency. In addition, third heart sounds have also been noted occasionally with ball valve prostheses\textsuperscript{13} in the presence of severe paravalvular leak. The reason for the more common occurrence of a third heart sound with homograft or heterograft valves than with caged prostheses has been postulated to be the fact that the prostheses cause a mild to moderate obstruction to inflow to the ventricle and rapid passive filling cannot take place at a rate sufficient to generate the third heart sound.\textsuperscript{13}

Since a third heart sound can be generated from either ventricle, proof that such a sound can occur in the absence of the subvalvular apparatus and the native atrioventricular valve leaflets would require demonstration of such a sound in a patient whose atrioventricular valve leaflets, papillary muscles and chordae tendineae have been resected on both sides of the heart. The patient we describe meets these criteria.

In a review of the literature, we were unable to find another such case described in detail. Stimmel et al\textsuperscript{12} described a series of patients in whom the mitral valve had been replaced with a heterograft and a third sound was heard postoperation. Case 2 in their series, described in table form only, had additionally tricuspid and aortic valve replacement. This patient was described in the table as having a third heart sound 0.13 sec after aortic closure. The phonocardiogram of this patient shows this sound to be intermittent and faint and the only reference tracings are the electrocardiogram and arterial pulse tracing. With a timing of 0.13 second after aortic closure, an apexcardiogram would be necessary to prove that this transient is a third sound rather than an opening sound or pericardial knock.

**References**