Heart Sounds in Patients with Homograft Replacement of the Mitral Valve

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SUMMARY
Phonocardiographic, hemodynamic, angiographic, and cardiac echographic studies have been performed on 11 patients whose diseased mitral valves have been replaced by a supported aortic valve homograft. The chordae tendineae and papillary muscles were removed in all patients during the operation. Apical first heart sounds of normal intensity were present in all patients after surgery. Nine patients exhibited splitting of the first sound. The initial component occurred when bulging of the homograft leaflets into the atrium was suddenly checked after the onset of ventricular systole. The second component occurred at the onset of rapid annular motion toward the base. Both sounds thus appear to originate in the homograft valve. Four patients had third heart sounds of left ventricular origin. The homograft leaflets were open and demonstrated no abrupt motion at the time of the third sound. It is evident that the third heart sound may occur in the absence of chordae tendineae and papillary muscles.

Additional Indexing Words:
First heart sound Third heart sound Cardiac echography
Mitral valve replacement First heart sound, splitting of

tenses the chordae.1 If this were the sole mechanism responsible for the third heart sound, such sounds would not be expected to occur in patients whose mitral valves have been replaced by an SAVH. Third heart sounds have not been observed in patients who have had replacement of their mitral valves by an artificial prosthesis such as the Starr-Edwards caged-ball valve.2, 3 Such prostheses, however, produce moderate mitral orifice obstruction.4 The resulting slow left ventricular filling rate could be the reason for absence of third heart sounds under these circumstances. An SAVH in the mitral orifice produces less resistance to flow.5 In addition, moderate valvular incompetence may result in a more rapid rate of early diastolic filling, and the hemodynamic features usually associated with a third heart sound are more likely to occur. For these reasons a study has been made of patients whose mitral valves have been replaced with

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# Table 1

**Clinical and Hemodynamic Data before and after Mitral Valve Replacement**

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<th>LAm</th>
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*Severity of mitral regurgitation indicated by 0 to 4+.

Abbreviations: MR = mitral regurgitation; TR = tricuspid regurgitation; AR = aortic regurgitation; MS = mitral stenosis; AS = aortic stenosis; T = tricuspid valve; M = mitral valve; A = aortic valve; NSR = normal sinus rhythm; AF = atrial fibrillation; AV diss. = atrioventricular dissociation; A flutter = atrial flutter; PAw = pulmonary artery wedge pressure; LAm = mean left atrial pressure; C. index = cardiac index.
HOMOGRAFT REPLACEMENT OF MITRAL VALVE

an SAVH with particular reference to the first and third heart sounds.

Methods

The SAVH has previously been described. A fresh human aortic valve is removed under sterile precautions at autopsy and preserved in cold Hank's base tissue culture solution. It is trimmed and mounted with sterile technic on a Teflon-covered pre-shaped titanium ring and stored in tissue culture antibiotic solution until use. Before the SAVH is placed in the mitral annulus, the diseased mitral valve is removed with its chordae. The papillary muscles are transected near the base. The outside diameter of the support ring varies from 22 mm to 35 mm. The inside diameter of the preparation with the valve leaflets in the open position varies from 16 mm to 30 mm.

Eleven patients were studied. Prior to surgery, phonocardiograms were recorded in all and cardiac catheterization studies were performed in nine. Mitral valve replacement with an SAVH was performed in all 11 patients. In five the aortic valve was also replaced with a nonsupported fresh aortic homograft. In one of these patients the tricuspid valve was also replaced with an SAVH.

Preliminary studies of eight of these patients have been previously reported. Late follow-up studies have demonstrated an excellent clinical result in eight and a good result in three. Only a small pressure gradient was present across the SAVH at rest during diastole (mean 3.6 mm, range 1.0 mm to 8.0 mm). Three patients with less than an excellent result had a moderate degree of regurgitation, but this has not increased during subsequent follow-up visits. Clinical and hemodynamic details are summarized in Table 1.

Phonocardiograms were recorded using a Sanborn twin-beam oscillograph at a paper speed of 75 mm/sec. In three patients heart sounds were recorded using a Hewlett-Packard model 4564B 4-channel photographic recording system. A series 350 preamplifier with a chart speed of 75 mm/sec was employed. A standard crystal microphone was used. Stethoscopic and logarithmic tracings were recorded during held expiration. Recordings were made of apex motion and carotid pulses using a standard pulse pickup. Records were made from 1 day to 1 year after surgery, and most patients had several phonocardiograms recorded on different postoperative clinic visits. Measurements of time intervals were made from the onset of the Q wave to the onset of the major deflections of the first heart sound recorded at the apex and lower left sternal edge. Ten consecutive cycles were measured and the mean value was reported. The amplitude of the components of the first heart sound was measured in millimeters during held expiration using a stethoscopic trace. The length of the R-R interval preceding the first sound was recorded in seconds.

Left ventriculograms, with cinetrace, were done in six patients. The contrast material was 76% Renograin, and 50 cc was injected with a force of 300 pounds/square inch. Patients were positioned in the right anterior oblique or anteroposterior projections, and cineangiography was done at 48 frames/sec. A simultaneous ECG reference trace was recorded. In one patient a catheter-tip manometer was advanced via the femoral artery into the left ventricle. Pressures were recorded from the left atrium using a Ross needle passed across the atrial septum. A simultaneous electrocardiogram and the two pressures were recorded at a paper speed of 200 mm/sec.

Cardiac echograms were done in seven patients. The commercially available ultrasonoscope was used. This instrument emits impulses at the rate of 1,000/sec, and it has a 2.24 megaHertz transducer (0.75 inch diameter). The duration of the impulse is 1 µsec, and the transducer acts as a sound receiver when no impulse is being emitted. The echoes returning from the tissues are displayed as waves on the face of an oscilloscope. The movement of the echo is plotted against time, and a record of the oscilloscope trace is made with a Polaroid camera. The patients were examined in the recumbent position. The transducer was placed in the fourth intercostal space 1 to 4 cm to the left of the left sternal border, and it was directed posteriorly and medially until the echo of the anterior leaflet of the valve in the mitral orifice was identified.

Results

Auscultatory Findings

The first heart sound was easily heard in all patients with maximal loudness at the apex. The sound was of normal intensity in seven patients and faint in four. In nine patients distinct close splitting of the sound at the apex could be identified. When atrial fibrillation was present, the first sound did not vary predictably in intensity in the usual manner with variable diastolic intervals. In three patients the loudness of the first sound varied paradoxically with the duration of the preceding filling period; in other words, faint sounds followed short filling periods and sounds of normal intensity followed long filling periods. Very short diastolic intervals were not fol-
lowed by accentuated sounds. The first sound was not louder in patients with sinus rhythm compared to patients with atrial fibrillation.

In one patient (case 2) a prominent third heart sound was easily heard with maximal loudness at the apex. The sound had the usual dull, thudding quality of a gallop sound; it was not clicking or snapping. There was a moderate respiratory variation in loudness with an increase in intensity during expiration. The sound was only faintly heard at the lower sternal edge and base of the heart. In three other patients a faint third sound was heard.

In three patients a soft, full-length, apical systolic murmur of moderate intensity (grade II-III) was present. The murmur had the usual blowing quality of a mitral regurgitant murmur.

Second sound splitting was normal in all patients. No opening snaps were heard. In the four patients who had aortic homograft replacement, in addition to mitral valve replacement, an aortic diastolic murmur was present in one patient.

**Phonocardiography**

**First Heart Sound**

In all patients careful study of phonocardiograms from the apex or lower sternal edge revealed two distinct major components of the first heart sound. The initial component began an average 0.07 sec after the QRS onset (range 0.06 to 0.08 sec). The second component occurred 0.10 sec (range 0.075 to 0.12 sec) after the QRS onset. Both components were usually of approximately equal loudness and clearly formed the loudest portion of the first heart sound at the apex (figs. 1 and 2).

In all patients faint low-frequency vibrations preceded the major component of the first sound but followed the QRS onset by 0.028 sec (range 0.02 to 0.04 sec). These vibrations were similar in appearance and timing to early low-frequency components of the first sound in preoperative phonocardiograms (figs. 1 and 2).

In nine patients late vibrations followed the first heart sound and were compatible in timing and appearance with ejection sounds. These sounds began 0.14 sec (0.12 to 0.16 sec)
HOMOGRRAFT REPLACEMENT OF MITRAL VALVE

Figure 2

Phonocardiograms from a patient with an SAVH in the mitral position (patient 5). (A) Note split first sound preceded by a low frequency component. (B) Note late ejection sound prominent at aortic area. (C) Relation of split first sound to apex impulse tracing. (D) Note late ejection component occurring at the time of carotid pulse rise. Aortic closure precedes pulmonic closure.

Table 2

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Range

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Abbreviations: Q-M = Q to onset of initial low-frequency "muscular" component of first sound; Q-1 = Q to first loud component of first sound; Q-2 = Q to second loud component of first sound; Q-ES = Q to late or ejection component of first sound; Ar-P2 = interval from aortic to pulmonic closure.

after the QRS onset (fig. 1). The pertinent data regarding these observations are summarized in table 2.

A study was made of the loudness and time of occurrence of the major components of the first sound in patients with atrial fibrillation and an irregular ventricular rhythm. The loudness of the first sound did not vary predictably in relation to the preceding diastolic filling period. In three patients very short filling periods were followed by faint sounds. In addition, the onset of the first sound in these patients was delayed 0.03 sec after short filling periods compared to normal filling periods.

In order to determine if splitting of the first sound was due to asynchronous closure of the mitral and tricuspid valves, the relative loudness of the two components was determined in phonocardiograms obtained from the
cardiac apex and from the lower left sternal edge at the fourth intercostal space. No consistent difference in loudness was observed between these two areas. In most patients both major components of the first sound were present in apex phonocardiograms and were of approximately equal loudness (figs. 1 and 2).

The relation of the sounds to the apex impulse trace was examined. The initial low-frequency component occurred simultaneously with the onset of apical precordial motion. The first loud component occurred about 0.03 sec after the onset of precordial motion and always before but sometimes close to the E point. The second loud component occurred at or just after the E point. Ejection sounds occurred later on the descent of the tracing from the E point. These time relationships are illustrated in figures 1 to 3.

The second sound appeared normal in all patients including five patients who also had aortic valve replacement with a homograft. Pulmonic closure followed aortic closure in all patients, and abnormal splitting was not observed. No opening snaps were recorded.

An important observation was the presence of a third heart sound in four patients. In one patient the sound was very prominent and was louder than the second heart sound at the
apex (fig. 4). The A2-third sound interval was 0.16 sec. The interval did not vary with respiration. The sound occurred at the end of a small rapid filling wave in the apex impulse tracing. Following the sound, an inward apex sound was observed. The sound was louder during expiration. The patient had a grade III full-length systolic murmur compatible with mitral regurgitation. Postoperative hemodynamic and angiographic studies confirmed the presence of moderate mitral regurgitation. A later follow-up examination 24 mo after surgery revealed similar phonographic findings. Auscultation revealed a grade IV full-length, rough apical systolic murmur with radiation to the axilla. A grade II blowing diastolic murmur of slight aortic regurgitation was present along the upper left sternal border. Prior to surgery, this patient had a low, grade IV, apical systolic murmur of mitral regurgitation and a faint third heart sound occurring 0.14 sec after aortic valve closure. Three other patients had third heart sounds of lesser intensity occurring 0.11, 0.13, and 0.16 sec after aortic valve closure (table 1).

In one patient a fourth heart sound was recorded 1 day after surgery (case 10). The sound was loudest at the apex. Prior to operation, severe mitral stenosis, mitral regurgitation, and aortic regurgitation had been present. In two other patients a fourth sound was present 4 mo after surgery. One year later the fourth sound was absent in one patient and a loud third sound was present (case 2).

**Echocardiographic Studies**

The movements of the anterior leaflet of the SAVH were analyzed by means of cardiac echography. As would be expected, there was a marked difference between the movements of a normal mitral valve and those of the SAVH, because the leaflets of the former are considerably larger than those of an aortic valve. Figure 5 shows a comparison of the movements in situ of the normal aortic valve and the SAVH. The leaflet producing an echo has a similar total excursion and wave form of motion in either aortic or mitral location.

**Figure 5**

Cardiac echograms of the orthotopic aortic valve (A) and the homograft valve in the mitral position (B) are shown. The diagram in the center illustrates the heart in diastole and the path of the sound beam which produced the echogram designated by the corresponding letter. The horizontal dotted lines link the valve leaflets with their echoes. Two of the orthotopic aortic valve leaflets are seen to separate during systole and fuse between the aortic wall echoes during diastole. The leaflets have a characteristic modified "square wave" motion. The motion of only one leaflet of the homograft valve in the mitral position is recorded. This heterotopic valve also shows a "square wave" motion with similar amplitude to that of the orthotopic valve leaflets. The difference in slope after the aortic valve opens in systole and after the homograft valve opens in diastole is related to volume changes of the heart altering the distance between the transducer and the valve.
Simultaneously recorded phonocardiogram and echogram of the homograft valve ("square wave" in the lower portion of the figure) with lead II of the electrocardiogram on both records from patient R.C. The premature ventricular beat seen on these records allowed alignment of the separate phonocardiogram and echogram in this patient with atrial fibrillation. The second component of the first heart sound occurs approximately 0.08 sec after the onset of each QRS complex, and coincides with the trough of the homograft valve closure wave in each supraventricular beat. The second component of the first heart sound occurs 0.12 sec after the onset of the premature beat but again coincides with the trough of the homograft valve closure wave.

Figure 5 shows the actual separation of two of the SAVH leaflets at the onset of ejection and coaption of these leaflets at the end of systole. Only one of the homograft leaflets is seen, and it opens (moves toward the transducer) at the onset of diastole and closes at the onset of systole.

A comparison was made of the time of closure of the homograft valve, as indicated by abrupt cessation of the rapid posterior movement of the leaflet, and the time of appearance of the first heart sound. Figure 6 shows that the first major component of the first heart sound coincides with the trough of the mitral closure wave.

An analysis of the cardiac echogram and the phonocardiogram in the patient with a loud third sound indicated that at the time of the third sound the SAVH leaflets were open, and there was no evidence of leaflet motion at the instant of the onset of the sound (fig. 7).

Cineangiographic Study

The cineangiocardiograms clearly showed the movements of the mitral valve ring. In two cases the valve leaflets could also be well seen. During isometric contraction of the ventricle, the valve ring moved toward the base, and the valve leaflets coapted as they bulged suddenly into the atrium. Figure 8 shows photographs of two frames taken from the cineangiogram of one patient. On the left, the left ventricle is shown during late diastole, and the initial portion of the QRS complex is just visible. Four frames, or 0.084 sec later, the ventricle is seen in isometric contraction, and the leaflets of the SAVH valve are bulging into the atrium. The phonocardiogram of this patient showed that the interval between the Q wave
of the electrocardiogram and the first major component of the first heart sound was 0.08 sec. Therefore, sudden checking of the protrusion of the mitral valve into the atrium appeared to be simultaneous with the occurrence of the first component of the split first heart sound.

With the onset of ejection the mitral valve ring moved suddenly toward the base carrying the bulging SAVH leaflets with it. The contour of the domed leaflets did not change at this time or during the remainder of systole (fig. 8).

Careful study of the angiogram in the patient with a loud third sound (case 2) confirmed that the SAVH leaflets were in an open position at the time of occurrence of the sound, as seen in the echogram. At this instant no abrupt motion of the leaflets was observed. Study of the contour of the left ventricular
chamber and motion of the left ventricular wall revealed no abrupt change in contour or interruption of the smooth outward diastolic filling motion at the time of the third sound. There was no evidence that left ventricular filling was limited by pericardial disease.

High-speed pressure tracings with the electrocardiogram in one patient revealed the presence of three sharp deflections in the ascending limb of the left ventricular pressure trace. The first occurred 0.06 sec after the QRS onset and 0.02 sec after the LV-LA crossover point. The second occurred 0.09 sec after the QRS onset. The third occurred higher on the ascending limb 0.14 sec after the QRS onset and 0.02 sec after the LV-aortic crossover point. These deflections probably reflect left ventricular sounds and correspond approximately in their time relationships to the externally recorded sounds (table 2). A study of the movements of the SAVH by angiography in this patient using a simultaneously recorded reference electrocardiogram, indicated that the first deflection occurred at the time the doming of the leaflets of the SAVH was suddenly checked early in systole. The second deflection occurred at the time the domed leaflets and the support ring were accelerated suddenly toward the apex, and the third deflection occurred just after the onset of ejection into the aorta.

Discussion

The present study has demonstrated the presence of four components to the first heart sound in patients whose mitral valves have been replaced by an SAVH.

Muscular Component

These vibrations are present prior to surgery and probably represent a muscular or non-valvar sound. The sound was present in patients with atrial fibrillation, hence an atrial origin can be excluded. Similar vibrations have been observed following tricuspid and mitral replacement with a Starr-Edwards prosthesis, so a valvar origin seems highly unlikely.

Valvular Components

This sound of usually normal intensity is easily heard at the apex. Careful auscultation will detect close splitting in most patients. Cardiac echograms and left ventricular angiograms suggest that the initial component of the split sound is of valvular origin and occurs when the early systolic bulging of the SAVH leaflets is suddenly checked. This occurs a brief instant after the left ventricular pressure exceeds the left atrial pressure.

The origin of the second component is not clear. It is not consistently louder at the lower sternal edge; hence a tricuspid origin seems unlikely. The high incidence of demonstrable splitting and the fairly constant degree of splitting (range 0.015 to 0.04 sec) suggest that the sound may also arise in the SAVH. Cardiac echography and angiography indicate that at the onset of ejection, the bulged leaflets of the SAVH are suddenly jerked toward the base. This sudden acceleration of a mass of blood behind tensed valve leaflets could produce a sound.

Additional facts support the possible SAVH origin of the split first sound. Both components usually produce a deflection of the apex impulse tracing in proportion to the intensity of the sound. Both components are usually present in apex phonocardiograms. Deflections of the ascending limb of the direct LV pressure recorded with a catheter manometer correspond in timing to the externally recorded sounds. The time relationships and the nearly equal magnitude of the sound deflections on the pressure tracing from the left ventricle also suggest a left ventricular origin of the corresponding external sounds. If this concept is correct, it lends indirect support to the view that splitting of the first heart sound is not due to asynchronous closure of the A-V valves but that both components of the split first sound arise in the left ventricle. Luisada and his co-workers have published data that suggest the left ventricular origin of both components of the split first sound in man and dog. It is unlikely that splitting of the apical first sound is due to an ejection sound. The second component of the split...
sound occurs earlier than the onset of the carotid pulse rise. Aortic valve disease was not present in patients with first sound splitting. Pressure tracings from the left ventricle and cineangiograms in one patient demonstrated the onset of the second component of the split sound to occur before the onset of ejection. In patients with ejection sounds of abrupt onset, clear separation between the second loud component of the first sound and the ejection sound could be observed.

**Ejection Component**

Vibrations following the major components of the first sound correspond in timing and character to ejection sounds. The presence of these sounds at the apex and the absence of pulmonary hypertension in the patients suggest that these sounds are aortic in origin.

The occurrence of a third heart sound in the absence of chordae tendineae and papillary muscles indicates that these structures cannot be involved in the mechanism of the third sound in the patients described in this report. The sharp deflection in the apex impulse trace at the time of the third sound has been attributed by Nixon to the jerk of the chordae at the time of rapid ventricular filling. The presence of this deflection in patient 2 (M.B.) would appear to exclude the possibility that the chordae are involved in this phenomenon.

However, these conclusions should be accepted with some reservation. In excision of the mitral valve, all of the chordae may not be removed. Short chordae may arise directly from the ventricular myocardium and insert into the posterior leaflet of the mitral valve close to the annulus. Lam and his group found such chordae in 31 of 50 hearts. It is possible that sudden tension of such short chordae could produce a third heart sound and the characteristic deflection of the apex tracing. Two observations have recently been made in our laboratory which refute this concept. A second operation was performed on patient 2 (M.B.) because of progressive mitral and aortic regurgitation. At surgery two basal chordae were observed extending from the myocardium to the annular remnant of the posterior mitral valve leaflet. The longest measured 2 mm in diameter and 35 mm in length. These chordae were removed and the aortic and mitral valves were replaced with supported fresh homografts. Seven days postoperatively a phonocardiogram was recorded. The P-R interval was prolonged to 0.29 sec and a sinus tachycardia was present. A prominent summation gallop sound occurred 0.16 sec after aortic valve closure. A second patient has also been observed who had a third heart sound prior to replacement of an incompetent SAVH in the mitral position. One basal chorda was observed at surgery and resected prior to insertion of another supported homograft. Ten days postoperatively a phonocardiogram revealed a third sound 0.20 sec after aortic closure occurring at the peak of the rapid filling wave in the apex impulse tracing.

The data do not exclude the possibility that the third heart sound arises in the valve leaflets. However, the fact that the leaflets were in the open position and exhibited no sudden motion at the time of the third sound suggests that early diastolic closure of the SAVH leaflets cannot account for the sound. If valve tension is occurring, it is not evident by the technics of study employed.

Using a similar technic of echocardiography, Fleming has recently demonstrated that in patients with mitral regurgitation, the mitral valve is open at the time of the third heart sound. His tracings also show no abrupt motion at the time of the third heart sound. Studies in our laboratory have confirmed these observations. In addition, in eight patients with Starr-Edwards mitral prostheses and significant regurgitation through a periprosthetic leak, no third sounds were present and no third sound deflections in apex traces were recorded despite steep filling angles. Crevasse and his co-workers have previously shown that mitral valve closure does not occur at the time of the third sound since atrial pressure is higher than left ventricular pressure at this time.

The possibility that the third heart sound arises in the left ventricular wall cannot be
answered by the present study. The angiographic demonstration that no abrupt checking of the diastolic filling motion of the left ventricular chamber occurred at the time of the third sound makes a muscular origin of the sound somewhat unlikely. Third heart sounds or "pericardial knocks" occurring in constrictive pericarditis, for example, are clearly associated with a sudden checking of the diastolic filling motion of the left ventricle.\(^{14,15}\) These sounds occur earlier in diastole than the third heart sound.

The incidence of third and fourth heart sounds in patients with SAVH replacement of the mitral valve appears to be low. A survey in our laboratory of 40 patients, including the present group, revealed five with third heart sounds. One was loud (reported in this paper) and three were faint. Two patients had faint fourth heart sounds.

Several observers have noted that third heart sounds are absent in patients whose mitral valves have been replaced by a Starr-Edwards prosthesis.\(^2,3\) This has been ascribed to the loss of chordae tendineae and papillary muscles, and the observations have been used as indirect support for the concept that the third heart sound results from chordal tension. Since the present study indicates that the chordae are not essential for third heart sound production, one might inquire why third heart sounds do not occur following replacement of the mitral valve with an artificial prosthetic valve. Two possibilities exist: (1) The degree of mitral orifice obstruction by the prosthetic valve reduced the rate of left ventricular filling below that required for the production of a third heart sound. This problem might be surmounted by a search for third heart sounds in patients who have a leak around the Starr-Edwards prosthesis which permits rapid ventricular filling to occur through the leak and the valve orifice. (2) Valve leaflets in the mitral position are necessary to produce a third heart sound. The latter possibility is supported by the present study and by Marshall and Gibson\(^6\) who have observed a third sound in 11 patients with homograft replacement of the mitral valve. Early clicking sounds corresponding in their timing to third heart sounds have been observed in patients who have mitral valve replacement with a Starr-Edwards prosthesis.\(^3\) Thus, it would appear that early diastolic forces are present that are capable of producing sounds at the A-V valve. If a prosthesis is present, a click occurs and if a homograft is present, a valvular sound occurs. These pertinent questions can only be answered by additional phonocardiographic studies on patients who have had mitral valve replacement either by homograft or artificial valves.

It is evident that studies of heart sounds in patients with prosthetic or homograft valves will continue to yield important information regarding the mechanism of production of normal heart sounds.

**References**


_Circulation, Volume XLII, August 1970_
HOMOGRAFT REPLACEMENT OF MITRAL VALVE

12. HULTGREN H: Unpublished data
15. HARVEY W: Auscultatory findings in diseases of the pericardium. Amer J Cardiol 7: 15, 1961

75 Years Ago
Coronary Disease and Myocardial Infarction
Dock’s Early Report (1896)

Case IV. Angina pectoris; dyspnoea; double hydrothorax; sudden death; atheroma and obstruction of coronaries; infarction of the heart.

Mr. B., lumber dealer, sixty-four years old, a man of large frame, was never sick until about three months before death. He then began to notice shortness of breath, especially when walking up hill. A week before death severe pain in the heart-region began. . . .

The diagnosis was myomalacia following coronary sclerosis, with secondary pericarditis. This was based on the history of increasing dyspnoea and heart pain, without evidence of disease in lungs or kidneys, or other (valvular) disease of the heart, the history of the acute attack indicating infarction, and the acute onset of pericarditis without other cause. . . .

Just below the orifice the left coronary artery became extremely atheromatous. . . .

The descending branch was narrowed, calcified, and about the middle of the anterior wall was obstructed by a red thrombus. . . .

The circumflex branch was nodular, but its lumen was free as far as the first branch, 2.5 cm. from its origin. Here it was completely obstructed by nodular arteritis for a distance of 3 mm. Beyond this, the lumen of the circumflex proper was free, but the next large descending branch was also totally obstructed. The wall of the left ventricle from this point, i.e., from the anterior papillary muscle to the septum, the posterior part of which was involved, and from near the ring to the apex, was the seat of a recent infarction. Only a thin layer, under the epicardium, from one to two mm. in thickness, was not necrosed, and it was red, swollen, the fibres cloudy and granular. . . .

In this case the relation of the coronary sclerosis to the gradually developing dyspnoea, and of the infarction to the acute attack a week before death is clear.

*Name as printed in the journal of 1896.