SYMPOSIUM ON CORONARY HEART DISEASE

Auscultatory Findings in Myocardial Infarction

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ON THE subject of auscultation in acute myocardial infarction James B. Herrick\(^1\) wrote in 1912 "The heart tones have been feeble—indeed, often startlingly feeble. Feeble contraction of the weakened, anemic heart muscle accounts for the weak pulse and the weak tones." A review of current texts on heart disease\(^2-4\) gives much the same information. Little investigative work has been done on this subject, which is not particularly surprising when one realizes that electrocardiography and not auscultation is the more precise method of diagnosis in acute myocardial infarction.

Other reasons for the lack of precise knowledge are the technical difficulties of making phonocardiograms or tape recordings and in doing hemodynamic studies during the acute phase of myocardial infarction. These difficulties include the critical condition of the patient, environmental noise, and the lack of any standardization technique for phonocardiography that allows accurate comparison of intensity and frequency of the heart sounds from patient to patient.

Bean\(^5\) noted that the heart sounds were described as weak, distant, or muffled in about 80 per cent of 160 cases of acute myocardial infarction and the loudness of the sounds was inversely related to the degree of shock in most cases. A phonographic study of heart sounds in acute myocardial infarction was done by Parsonnet and Hyman\(^6\) in 1931, but because of lack of accurate recording equipment at that time it is difficult to evaluate their results in light of present techniques. In 1942 Masters and Friedman,\(^7\) using better equipment, recorded phonocardiograms in 100 normal people for controls and then a series of patients with acute myocardial infarction. They reported that the first heart sound was absolutely diminished in amplitude in 24 per cent of the cases of infarction and was relatively reduced in relation to the second heart sound in 54 per cent. They also noted that an atrial or fourth sound was recorded in 83 per cent of cases with infarction but in only 38 per cent of the controls and was practically always associated with cardiac failure. Likewise, a third heart sound was present in 47 per cent of cases with infarction, but only in 12 per cent of the controls and was invariably associated with cardiac failure. Another observation was that shock was much less common in those cases with an unimpaired first heart sound.

Normal Intensity of Heart Sounds

Many factors affect the intensity of the first and second heart sounds.\(^8\) The sounds are accentuated when the heart is close to the precordium as in children and thin adults, and, conversely, they are diminished in obese individuals and in emphysema because of the distance of the heart from the precordium and the poor conductive qualities of fat and lung tissue.

It should be pointed out that because of the wide variation in intensity of heart sounds normally, it is extremely difficult to evaluate changes unless each individual acts as his own control by having phonocardiograms or tape recordings made before the occurrence of a disease process. Even the change from a sitting or reclining position to a supine position frequently alters the intensity of the heart sounds rather markedly and is

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only one of several factors that must be controlled. Also, as far as we are aware, no one has carefully studied the intensity of the heart sounds in normal individuals confined to bed for a prolonged period of time.

Our knowledge of the changing intensity of heart sounds due to pathologic processes is fragmentary, since adequate hemodynamic studies have not been done under acute conditions. It would seem, however, that the intensity of the first heart sound (which results from the vibrations of the blood initiated by the closure of the mitral and tricuspid valves) must be related to diastolic filling and ventricular ejection time.

Since we have so little data on this subject, we can only rely on clinical observations that have shown that the heart sounds tend to be diminished in such conditions as shock, pericardial effusions, cardiac decompensation, and terminal states. Thus, when myocardial infarction is accompanied by some degree of shock, the heart sounds commonly seem to be diminished in intensity. On the other hand, we have examined many cases of acute myocardial infarction without shock and we have not been impressed with any marked changes in the intensity or character of the heart sounds. But to confuse the issue further we must confess that we have occasionally, in cases of acute myocardial infarction, noted changes in the intensity of the heart sounds from time to time in the same patient even though no objective signs (changes in blood pressure, heart rate, etc.) have occurred to indicate a change in clinical status.

Additional Sounds

When the left ventricle is dilated as the result of myocardial disease of either acute or chronic nature, a gallop sound is frequently present, which is generally accepted to be an accentuation of a normal third heart sound or a normal atrial sound. Thus, a gallop may be protodiastolic if due to accentuation of the third sound or presystolic if due to accentuation of the atrial sound. At times, both may be present as separate sounds or may be fused into a single loud diastolic sound.

Other Auscultatory Findings

Most of the abnormal auscultatory findings in acute myocardial infarction and chronic arteriosclerotic heart disease are the result of complications. These findings are often of definite diagnostic aid, since they may give information that is not readily obtainable from such tests as the electrocardiogram.

Pericarditis

While figures vary widely, about 20 per cent of cases of acute myocardial infarction are said to show pericardial friction rubs at some time. This figure might be higher if continuous recordings were to be made, since some pericardial involvement has been noted in as many as 80 per cent of autopsied cases of myocardial infarction. It is difficult, however, to know how much involvement is necessary to produce rubs; moreover autopsied cases are a small percentage of the total number of cases of myocardial infarction.

The pericardial friction rubs resulting from myocardial infarction have no characteristics that differentiate them from other types of pericarditis producing rubs. Rubs are probably most commonly heard 2 to 4 days following the infarction.

Perforation of the Interventricular Septum

This is an uncommon situation probably occurring in only 1 to 2 per cent of deaths from myocardial infarction but the diagnosis is relatively simple. The sudden occurrence of a loud systolic murmur, usually maximal in the fourth left intercostal space with or without a thrill is sufficient evidence. Loud pericardial rubs, however, may at times be confusing. The patient usually develops increasing venous pressure and left ventricular failure and the course is generally rapidly deteriorating although a few cases have been known to survive for several months or even years.12

Ruptured Papillary Muscle

This may also be confused with perforation of the interventricular septum but the murmurs tend to be more bizarre, sometimes have been reported to be diastolic in time and are
Figure 1

Systolic murmurs recorded from the cardiac apex of a 54-year-old man with previous myocardial infarction in whom this murmur developed suddenly. The figures at the left of each stethogram indicate the number of days following the onset of the murmur at which the recordings were made. Note the high-frequency musical characteristics of the murmur at the time of the first 3 recordings. The heart sounds can scarcely be identified because the murmur was so loud (grade VI) that the recording volume had to be markedly reduced. In time the murmur decreased in intensity and lost its musical character and in the last 3 recordings the heart sounds can be identified. Time intervals are 0.04 second.

usually better heard at the apex. A thrill is generally absent in distinction to its rather common occurrence in interventricular septal rupture.

Figure 1 illustrates the bizarre and changing murmur encountered in a 54-year-old man who had had a previous myocardial infarction. He was awakened from sleep, and although he noted no precordial pain, he felt weak and perspired profusely. His wife could hear a peculiar sound and upon placing her hand on his chest, felt a “crawling” sensation. When examined, the patient had a grade-VI systolic murmur at the apex accompanied by a thrill. The electrocardiogram showed evidence of subendocardial infarction. The patient survived this episode but died suddenly in his sleep 9 months later. Although the diagnosis of ruptured papillary muscle cannot be proved because an autopsy was not obtained, the figure is consistent with the diagnosis and at least illustrates the type of murmur with sudden onset that should arouse the suspicion of a ruptured papillary muscle or perforation of the interventricular septum.

Rupture of the Ventricle

This complication usually occurs between the second and twelfth day after infarction. Again the murmur is loud and systolic in time and accompanied by a thrill. Cardiac tamponade and rapid deterioration are the consequence.

Ventricular Aneurysm

Although various murmurs have been reported in cases of ventricular aneurysm following myocardial infarction, in our experience most of these cases have no unusual auscultatory findings and the diagnosis is best made by electrocardiography and cardiac fluoroscopy.

Other Murmurs Encountered in Arteriosclerotic Heart Disease

There are other murmurs that may at times be most confusing in a patient with arteriosclerotic heart disease with or without acute myocardial infarction. One of these (fig. 2) is a diastolic murmur, often with presystolic accentuation, which is very similar to the murmur of mitral stenosis. The figure illustrates the stethogram from the cardiac apex of a 48-year-old woman with previous authenticated myocardial infarction who showed progressive cardiac enlargement and cardiac failure but gave no history of angina pectoris. At autopsy there was left ventricular hypertrophy and dilatation and severe coronary atherosclerosis with an old myocardial infarction. The valves were essentially normal, and it is presumed that the murmur resulted from relative mitral stenosis due to dilatation of the left ventricle. It will be noted that there is no opening snap, which would be common in mitral stenosis. Instead there is a low-frequency sound which would be consistent with a third heart sound, which is commonly seen.
Stethogram recorded from the apex of a 48-year-old woman with a history of previous myocardial infarction, increasing cardiac enlargement, and cardiac decompensation but without angina pectoris. The first and second heart sounds are identified. Note the vibrations occurring during diastole including a low-frequency sound, which is probably a third heart sound. This murmur sounded like the low-pitched rumble of mitral stenosis. Time intervals are 0.04 second.

with left ventricular dilatation and in the presence of cardiac failure is interpreted as a gallop sound. When this patient was first seen, the diastolic murmur was wrongly interpreted as evidence of mitral stenosis.

Rheumatic valvular lesions in the older age groups may frequently pre-exist in patients with myocardial infarction and cause great confusion if they have not previously been known. A particularly common variety is aortic stenosis, which is a frequent precursor of angina pectoris and myocardial infarction. We recently had this brought to our attention when we saw a 58-year-old man for the first time who gave a history of severe precordial pain of several hours' duration and in whom the electrocardiogram showed S-T segment changes presumed to be the result of acute subendocardial myocardial ischemia or infarction. While he did have a faint systolic murmur at the aortic area, no great significance was attached to it until he had been hospitalized for a few days. Then it became apparent from the lack of clinical and laboratory signs of acute myocardial infarction and a stable electrocardiogram that the condition was in reality one of previously undiagnosed aortic stenosis with an anginal syndrome. The carotid pulse curve was typical of hemodynamically significant aortic stenosis, and the electrocardiographic pattern was evidently the result of left ventricular hypertrophy.

One could cite many examples of this sort but the variability is extreme and taxes the ingenuity of the physician.

Summary

The auscultatory findings in acute myocardial infarction and arteriosclerotic heart disease have been briefly reviewed. It has been pointed out that there are no auscultatory findings that can equal the efficiency of the electrocardiogram in the diagnosis of acute myocardial infarction. The signs that do occur are probably the result of variable factors such as diastolic filling and ventricular ejection time and are not necessarily peculiar to myocardial infarction. On the other hand, certain complications that may occur as the result of myocardial infarction and certain conditions that may simulate myocardial infarction may produce auscultatory findings that are of definite diagnostic and prognostic significance.

Summario in Interlingua

Es presente un breve revista del constatationes auscultatorii in infarimento myocardial acute e morbo cardiae arteriosclerosis. Es signalate que il existe nulle constatationes auscultatorii que es equal in lor efficacia diagnostic al electrocardiogramma in casos de acute infarimento myocardial. Le signos que es notate in le auscultation es probablemente le resultato de varie factores, como replemamento diastolico e tempore de ejection ventricular, e non es necessarimente peculiar a infarimento myocardial. Del altere latere, certe complicationes que potte occurrer como le resultato de infarimento myocardial e de cete conditions enpace a simular infarimento myocardial, produce in cete casos constatationes auscultatorii che es definitemente de signification diagnostic e prognostic.

References


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