Alterations in Intensity of Heart Sounds After Myocardial Infarction

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A reduction in the intensity of the heart sounds is a familiar sequel to myocardial infarction. Sounds are sometimes described as muffled, weakened, or distant, and when the first sound alone is affected so that both sounds become of equal loudness a characteristic rhythm, sometimes known as "tick-tack rhythm", is produced. This change in intensity of the heart sounds has been attributed to alterations in heart rate, lengthening of the P-R interval, or to a lowering of systemic blood pressure, but it may be the only physical sign detectable, and not infrequently it is observed for the first time several days after the acute episode when the patient is making an otherwise uneventful recovery.

In this paper the results of measurement of intensity of the sounds after myocardial infarction are described, and an attempt is made to relate them to other effects of myocardial infarction.

SUBJECTS AND METHODS

The series included 75 male patients with a history of myocardial infarction within the week preceding the first examination and with confirmatory electrocardiographic abnormalities and transaminase levels.

The control group comprised 58 male patients admitted to a general medical ward for reasons other than cardio-respiratory disease. In order to cover a wide range of blood pressure, patients with hypertension but normal electrocardiograms and patients with hypotension due to gastro-intestinal haemorrhage or self poisoning were included in this group.

Apparatus and Procedure. Heart sound intensity was measured by a method similar to that described by Rushmer, Bark, and Ellis (1952). A piezo crystal microphone applied in the region of the cardiac apex at

the point at which the sounds could be heard best with a stethoscope was linked to an audio frequency spectrometer (type 2111 Bruel and Kjaer) and a sound level recorder (type 2301). Tracings were recorded on calibrated paper. In an initial trial the heart sound levels were found to be in the range 30 decibel (db) to 60 db so a weighting network, DIN 2 (A weighting) was employed. These limits were found to be satisfactory for all the sound levels encountered throughout the study. The human ear is less sensitive to sounds of low and high frequency than to those with the middle frequencies. The weighting network makes appropriate adjustments to the sensitivity of the sound level recorder so that the sound level measured describes as closely as possible the strength of a sound as perceived by the human ear. In the remainder of this paper intensity, loudness, and sound level will all be used to refer to the strength of the sound as measured in this way. Recording was carried out with the patient recumbent. Ventricular rate and blood pressure were recorded at the same time, and skinfold thickness was measured at the site of application of the microphone. The site of the infarct and the P-R interval were recorded from the electrocardiogram. Pulse rate, blood pressure, and skinfold thickness were also measured in control subjects, but as electrocardiograms were recorded on only a few no data are available on the P-R intervals.

RESULTS

A typical tracing of heart sound level is shown in Fig. 1. Some fluctuation in the amplitude of the heart sounds was observed, but this was minimized by taking records with the breath held in inspiration. Each recorded value is the mean of 10 consecutive first and second heart sounds. For computation these values were grouped at intervals of 2.5 db. The results are summarized in Table I and in Fig. 2. The distribution of first heart sound levels in the patients with myocardial infarction is bimodal, and in Fig. 3 the peak of lower level is shown to be due to posterior infarcts. Anterior infarcts account for the peak close to that of the "normal" distribution.

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FIG. 1.—Tracing of heart sound levels in a normal subject. dbA = sound level in decibels employing "A weighting" network. (Baseline = 30 db threshold; vertical calibration in 5 db intervals.)

TABLE I
MEAN VALUES AND STANDARD DEVIATIONS OF
HEART SOUND LEVELS IN 75 PATIENTS WITH
MYOCARDIAL INFARCTS AND IN 58 CONTROLS

	"Controls"	Myocardial infarcts	Value for p
Number	58	75	
Sound level { 1st sound	45.8 ± 3.2	43.2 ± 3.9	<0.001
dbA { 2nd sound	41.1 ± 3.1	39.4 ± 2.7	<0.003
Age (yr.)	54.0 ± 14	57.4 ± 10	<0.1 > 0.05
Heart rate/min.	81.5 ± 18.6	80 ± 14.4	<0.3 > 0.2
Mean blood pressure (mm. Hg.)	102 ± 25.6	97 ± 13.0	<0.1 > 0.05
Skinfold thickness (mm. × 100 log. 10*)	201 ± 32.4	212 ± 22.9	<0.05 > 0.02

dbA = Decibels employing "a weighting"

*Edwards *et al.*, 1955.

Table II shows a comparison of results in these two groups.

It is clear that the first and second heart sounds of patients suffering from myocardial infarction are

significantly quieter than those of the control subjects. Both heart sounds are also significantly quieter in patients with posterior infarctions than in patients with anterior infarctions.

Skinfold Thickness. A slight correlation existed between the intensity of the first heart sound and the skinfold thickness for both the controls ($r = -0.36$; $p = 0.02$) and the cardiac patients ($r = 0.232$; $p = 0.05$). No such correlation could be demonstrated for the intensity of the second heart sound.

The mean skinfold thickness of patients with myocardial infarction was greater than that of the controls ($p = 0.05$), but there was no difference between the skinfold thickness of those with anterior infarction and of those with posterior infarction.

Heart Rate and Systemic Blood Pressure. No significant correlation was demonstrated between the intensity of heart sounds and the heart rate or the systemic blood pressure (systolic or mean) among either the controls or patients with myocardial

TABLE II
MEAN VALUES AND STANDARD DEVIATIONS OF HEART SOUND LEVELS IN 40 PATIENTS WITH ANTERIOR
AND 35 WITH POSTERIOR MYOCARDIAL INFARCTION

	Anterior Infarcts	Posterior Infarcts	Value for p
Number	40	35	
Sound level { 1st sound	44.35 ± 3.6	41.85 ± 3.9	<0.005 > 0.001
dbA { 2nd sound	40.06 ± 2.3	38.72 ± 3.25	<0.02 > 0.01
Age (yr.)	57.2 ± 8	57.8 ± 6	<0.8 > 0.7
Heart rate/min.	82.2 ± 13.9	77.0 ± 15.6	<0.2 > 0.1
Mean blood pressure (mm. Hg.)	98.1 ± 14.3	95.1 ± 11.7	<0.2 > 0.1
Skinfold thickness (mm. × 100 log. 10*)	215.8 ± 20.0	219.4 ± 25.8	<0.4 > 0.3
P-R interval (sec.)	0.146 ± 0.022 (n=35)	0.145 ± 0.026 (n=28)	>0.9

*Edwards *et al.*, 1955.

FIG. 2.—Distribution of heart sound levels in 75 patients with myocardial infarction and in 58 controls.

infarction. There was also no significant difference in the systolic blood pressure, mean blood pressure, and heart rate of controls and patients.

P-R Interval. Only one patient had a prolonged P-R interval. Four patients had atrial fibrillation, two had ventricular tachycardia, and in six patients the P-R interval was recorded as normal but was not measured. In the remaining patients no correlation could be shown between the length of the P-R interval and the intensity of the heart sounds. The duration of the P-R interval did not differ significantly with the position of the infarct.

Necropsy Studies. During the course of the study six patients whose heart sounds had been recorded died while still in hospital. Three had anterior myocardial infarcts, and in these three patients the intensity of the heart sounds had been close to the mean "normal" value. A fourth patient had a posterior myocardial infarct with an anterior extension, and in this patient also the heart sound intensity had been close to the mean "normal" value. The remaining two patients had posterior infarcts, and both had values for heart sound intensities which were well below the normal mean and also below the mean for posterior infarcts. Both of

FIG. 3.—Distribution of heart sound levels in 40 patients with anterior and 35 with posterior myocardial infarction.

these patients, unlike the others, had mural thrombus between the posterior wall and the papillary muscles, the bases of which were involved in the necrosis.

DISCUSSION

The intensity of heart sounds recorded in the region of the cardiac apex was reduced in patients suffering from recent myocardial infarction. This was not due to the age of the patients, to a fall in blood pressure, to change in heart rate, or to prolongation of the P-R interval. The mean skinfold thickness of the patients was 3 mm. greater than that of the controls, but it can be calculated from the regression coefficient (0.032) that a difference of 25 mm. would be required to produce this difference in the mean heart sound intensities.

An unexpected and significant finding in this series was the very much greater reduction in sound intensity in the group of patients whose myocardial infarcts involved the "posterior" wall of the heart. There was no difference in age, blood pressure, heart rate, duration of P-R interval, or skin thickness between these patients and those whose infarct was anterior.

Of 6 patients who died there were 2 whose heart sounds during life had been unequivocally reduced in intensity. These were also the only 2 of the 6 in whom the recent infarct was posterior, and in both of these the infarct had involved the papillary

muscles. To account for the relation between the degree of attenuation of the heart sounds and the position of the infarct, it seems to us reasonable to suggest that it is due to damage to the papillary muscles, which as they arise from the posterior wall, the apex, and the lateral wall of the left ventricle, with little or no origin from the septum and anterior wall, are more likely to be involved by posterior infarction than by anterior infarcts. The first sound is related to the closure of the AV valves, and it is generally believed that tensing of the valve curtain by the contraction of the papillary muscles is responsible for producing the sound. Infarction of the papillary muscles, or of the underlying myocardium, could slacken the chordae tendineae and the valve curtain, and so reduce the intensity of the sound produced when these valves close. The second sound occurs with the closure of the semilunar valves and is believed to be due to tensing of the valve cusps by the local back flow of blood after ventricular ejection (McKusick, 1958). The origin of two cusps of the aortic valve is closely associated with the base of the anterior cusp of the mitral valve, and when viewed from within the ventricular cavity they appear to be one continuous structure. It is possible therefore that infarction in the region of the papillary muscles also influences the loudness of the second sound.

We are aware that many of the factors that influence the production and transmission of heart

sounds have not been assessed in this study. Measurements of pressure transients (Rushmer, 1955), and assessment of the loss of sound by absorption in compressible tissues between the sound source and the chest wall (Dock, 1959), cannot be conveniently undertaken without discomfort and risk to the patient. Further, it should be remembered that the A-weighted instrument used in this study would not detect sound frequencies that are outside the range of human audibility, and that a change in sound quality could account for an apparent change in sound level. It may be that additional information might be obtained by measuring sound levels at all frequencies, and relating these to the electrocardiographic findings and to the extent and position of infarcts at necropsy.

SUMMARY

The intensity of the heart sounds after myocardial infarction was found to be significantly reduced.

Attenuation of the heart sounds could not be accounted for by the age of the patients, by alteration in heart rate, by a fall in systemic blood pressure, or by prolongation of the P-R interval.

The reduction in intensity was much more ob-

vious when the infarction was posterior than when it was anterior.

The findings are discussed, and it is suggested that involvement of the origins of the papillary muscles is an important factor contributing to the considerable reduction in intensity which follows posterior myocardial infarction.

Necropsy findings in two patients support this view.

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