The Quality of Resonance of the First Heart Sound After Myocardial Infarction: Clinical Significance

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SUMMARY  Frequency analyses of the first heart sound (S₁) were performed in 80 normal subjects and 80 postinfarction patients. A readily recognizable frequency pattern characterized by a quality of resonance ≥2, as measured by the Q factor at 3 db down, was noted in 78 of the 80 apparently normal subjects. An aberrant pattern with a Q <2, often accompanied by a lowering of the frequency content, was found in 78 of 80 postinfarction patients. We propose that the quality of resonance of S₁ is a measure of the degree to which the structural homogeneity of the left ventricle as a compliant contractile unit has been preserved after myocardial infarction.

IN 1961, RUSHMER suggested that the first heart sound (S₁) results from the vibrations of the entire blood-filled heart as a dynamically coupled system.¹ In the intervening years, this “cardiohemic” concept has won increasing acceptance.² ³ In 1970, Adolph et al. tested Rushmer’s hypothesis by performing frequency analyses of S₁ during isovolumic contraction.⁴ They found a consistent and reproducible pattern in 74 normal subjects and an aberrant pattern characterized by a greater voltage output at 30 Hz than at 40 Hz in 21 of 24 patients with acute myocardial infarction. A similar pattern was found in patients with healed infarcts, cardiomyopathy, and in highly trained athletes. Normal patterns were found in patients with rheumatic valvular disease, including four patients with Starr-Edwards prosthetic valves. They reasoned that the variations between normal and abnormal patterns could be explained in terms of myocardial


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elasticity and mass (left ventricular muscle mass and the volume of blood in the left ventricle at end-
diastole), and that their results supported the "car-
diohemic" hypothesis of the origin of $S_1$.

We performed frequency analyses in 80 normal sub-
jects and 80 patients who had had a transmural
myocardial infarction. We confirmed the finding of
Adolph et al. that the normal subject has a readily
recognizable frequency pattern with peak voltage out-
put usually at 40 Hz. A lowering of the frequency con-
tent to 30 Hz was commonly found in the post-
infarction patients and in some normal, physically
active young adults. When the quality of resonance as
measured by the Q factor was determined, we found
that the normal subjects had a $Q \geq 2$ and that the post-
infarction patients had a $Q < 2$. Although the deter-
mination of the Q factor proved to be useful because it
provided an objective measurement, abnormal
patterns could generally be distinguished from normal
patterns at a glance (figs. 1–3). The degree of distor-
tion of the normal frequency pattern tended to cor-
relate with the clinical impression of the severity of the
myocardial damage.

**Methods**

The control group of 80 subjects was selected on the
basis of a normal history, physical examination, and
ECG. These subjects were 7–86 years old. The 80
postinfarction patients had a well-documented history
of myocardial infarction and an abnormal Q in the
ECG. One was 3 days postinfarction; the others had
had their infarctions from 4 weeks to several years
previously.

An electronic analyzer was used to identify the fre-
quency composition of the $S_1$. The output of a
stethoscope was detected by an electret microphone
and recorded on a standard Sony TC-280 tape deck
(Superscope Inc., Sun Valley, California) with a fre-
quency response of 20–20,000 Hz. The output of the
tape deck, after amplification, was scanned using a
variable frequency analog filter (Applied Research
Associates, Boston, Massachusetts) designed and con-
structed for the study. A high-fidelity speaker was
used to monitor the auscultatory quality of the input
to the filter/analyzer. The range of the frequency scan
was 20–100 Hz, and we paid particular attention to
the 20–60-Hz range, which includes the fundamental
and first harmonic output of $S_1$. All instrumentation
had excellent low-frequency response capabilities. (A
more complete description of the equipment may be
found in reference 6.) The sounds were recorded with
the patient in the left lateral recumbent position. As
the observer listened to the $S_1$, he recorded the relative
voltage output at frequencies from 20–100 Hz at in-
crements of 10 and plotted the results on a graph (fig.
1). The quality of resonance, Q factor, was determined
from the equation

$$Q = \frac{F_r}{F_2 - F_1}$$

where $F_r$ is the resonant frequency and $F_2$ and $F_1$ are
the two frequencies above and below resonance at
which the average power has dropped to one-half its
resonance value, i.e., 3 db down (fig. 1). The quantity
Q is a measure of the sharpness of resonance or fre-
cquency selectivity of a resonant vibratory system hav-
ing a single degree of freedom, either mechanical or
electrical. The value of the Q factor as determined in
this study is related to the response characteristics and

![Figure 1](quc311f1.jpg)
bandpass of the frequency analyzer and might vary slightly in a study with different equipment.

Results

The results of the frequency analyses of the 80 controls and the 80 postinfarction patients are given in table 1.

![Figure 2. The prototype of the normal frequency pattern, ABC, with a peak voltage at 40 Hz is shown, as well as two normal variants: MNO with peak voltage at 50 Hz and XYZ with peak voltage at 30 Hz. All three patterns have a Q factor \( \geq 2 \) (2.5, 2.7 and 2.9). The secondary peak at 60–70 Hz is a common finding.]

The normal frequency pattern (figs. 1 and 2) is characterized by a peak voltage output at 40 Hz and a Q factor \( \geq 2 \) (43 of 80 subjects). The results were not age-related in the group with peak voltage at 40 Hz. The oldest subject was 86 years old and had a normal ECG and a normal frequency pattern. A common variant pattern had a peak voltage at 50 Hz (24 subjects). The 13 subjects with a peak voltage at 30 Hz

![Figure 3. Three abnormal patterns are shown. In two, ABC and MNO, the initial peak is at 30 Hz. In XYZ, the output at 20 Hz is as high as at 30 Hz, making this a very abnormal pattern. In all three patterns the Q factor is <2.]

were all young and physically active, although only a few could be considered trained athletes. The two apparently normal subjects with a Q < 2 were encountered early in the series when experience with the technique was being acquired. It is doubtful that the normal heart produces a frequency pattern with a Q factor < 2.

The abnormal pattern is characterized by a Q factor < 2 (78 of 80 patients) and by a strong tendency to a lowering of the frequency content to 30 Hz (fig. 3). The two patients with a Q factor ≥ 2 had had relatively mild infarctions clinically, had made good recoveries, and were asymptomatic when their patterns were recorded.

**Discussion**

The resonant frequency of a vibrating system is inversely proportional to the square root of mass (weight, volume) and compliance (softness, elasticity); therefore, the frequency varies directly with stiffness (lack of flexibility). The left ventricle may vibrate at a lower frequency because of increased mass, as in the trained athlete, or because of increased compliance caused by the loss of contractile muscle elements, as in the patient with acute myocardial infarction. In the latter case, when the soft necrotic myocardium is replaced by fibrous tissue, the ventricle will become stiffer and the frequency will tend to rise. Also, after infarction the combined mass of the left ventricle may be changed. A smaller mass times a greater compliance in the correct proportions will give rise to the same frequency as a greater mass times a smaller compliance. In this study, in which frequency analyses were carried out at widely varying periods after infarction, it is not surprising that there was a considerable variation in the frequency at which the peak voltage developed. Nevertheless, a validity study of the data in table 1 gives a probability percentage of 5%, suggesting that the reduction in frequency in the post-infarction patients compared with control subjects is probably significant.

The Q factor is defined as a measure of the sharpness of resonance of a mechanical or electrical system, and is not directly related to frequency. The determinant of the value of Q is the ratio of mass or compliance to system effective resistance. In the normal heart the Q is high even though the frequency may be low because the structural homogeneity of the myocardium of the left ventricle provides low resistance to movement. In the heart that has sustained a myocardial infarction, the value of Q is related to alterations in the structure of the myocardium which increase its resistance to movement. An asynergic area of myocardium will dampen the vibrations of the left ventricle and lower the Q. The Q factor may be viewed as an indicator of the degree to which the left ventricle vibrates as a structurally homogeneous unit.

The potential clinical value of frequency analysis is twofold: 1) as an aid in the diagnosis of myocardial infarction, and 2) as an indicator of the degree of residual damage to the left ventricle as a compliant contractile unit.

Clinical situations are commonly encountered in which it is difficult to be sure whether infarction, especially remote infarction, has occurred. Q waves may be questionably pathological. The ECG in pulmonary embolism and in the Wolff-Parkinson-White syndrome may show changes suggestive of myocardial infarction. In five cases of pulmonary embolism in the study of Adolph et al. and in one case of Wolff-Parkinson-White syndrome in our study, the frequency patterns were normal. Although cases of subendocardial infarction were intentionally excluded from the study, an extensive subendocardial infarction would probably give rise to a frequency pattern similar to that seen after transmural infarction, since the structural homogeneity of the myocardium would be similarly disturbed in both types of infarction.

Because we did not perform hemodynamic studies of left ventricular function, we do not know to what extent distortion of the frequency pattern and the lowering of the Q factor correlate with measurements of impaired ventricular function. However, we observed that when there was extensive loss of R waves in the precordial leads, severe angina, or persistent congestive failure after infarction, the Q factor was usually close to 1. The one patient in the study in whom a ventricular aneurysm was demonstrated after multiple episodes of cardiac arrest had an extremely abnormal frequency pattern.

**Conclusions**

1) The normal heart generates the S1 with a frequency pattern characterized by a Q factor ≥ 2 and a peak voltage output at 40 Hz. Normal variants are patterns with the peak voltage at 50 Hz and with peak voltage at 30 Hz, the latter seen in young, physically active people and in trained athletes.

2) Myocardial infarction causes an aberrant pattern characterized by a Q factor < 2 and a tendency to a lowering of the frequency content to 30 Hz.

3) The degree of distortion of the normal frequency pattern after myocardial infarction tends to correlate with the clinical impression of the severity of the myocardial damage.

4) We hypothesize that the quality of resonance of S1 after myocardial infarction provides a measure of the degree to which the structural homogeneity of the left ventricle as a compliant contractile unit has been preserved.
The Effect of Delay in Propranolol Administration on Reduction of Myocardial Infarct Size After Experimental Coronary Artery Occlusion in Dogs

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SUMMARY The effects of intravenous propranolol (2 mg/kg) on myocardial ischemic injury in relation to the influence of delay in therapy on gross infarct size (GIS) were determined in 39 closed-chest anesthetized dogs in which the left anterior descending coronary artery (LAD) was occluded at a fixed distance from its origin by a balloon catheter. Precordial ECG maps, hemodynamic variables and serum CK levels were monitored for 24 hours. After 24 hours, we estimated GIS from the measured areas of ischemic discoloration in serial sections of the left ventricle (LV). In nine dogs, propranolol administration was started before LAD occlusion, in another nine 3 hours and in 10 others 6 hours after occlusion; the remainder (n = 11) served as controls. In the dogs pretreated with propranolol, the GIS (14.0 ± 4.0 g or 10.0 ± 2.0% of LV weight) was 53% smaller (p < 0.01) than in the controls (29.0 ± 2.0 g or 22.0 ± 1.0% of LV weight); those given propranolol 3 hours after occlusion had 28% smaller (p < 0.05) GIS (19.0 ± 2.0 g or 15.0 ± 2.0% LV weight) than the controls. However, GIS in the dogs receiving propranolol 6 hours after occlusion (24.0 ± 3.0 g or 19.0 ± 3.0% of LV weight) was not significantly different from that in the controls. The beneficial effect of propranolol on GIS was accompanied by corresponding directional changes in the precordial ST-segment elevation and in the rate of decline of the R-wave amplitude of the ECG. Propranolol reduced the heart rate and cardiac output for 5–6 hours in pretreated dogs; in dogs given propranolol 3 and 6 hours after occlusion, heart rate was reduced for 3–4 hours, but the cardiac output remained low for the remainder of the 24 hours. The data in these studies indicate that the beneficial effect of propranolol on GIS varies inversely with the delay in drug administration after LAD occlusion, and that no effect is apparent when propranolol infusion is begun 6 hours after occlusion.

HOSPITAL MORTALITY, severity of hemodynamic impairment and the incidence of ventricular arrhythmias in patients with acute myocardial infarction have all been shown to be related to the enzymatically estimated infarct size as well as to that determined at autopsy. Therefore, if infarct size can be contained, the overall prognosis after acute infarction may be improved. Studies in experimental animals have suggested that ischemic injury after coronary occlusion can indeed be altered by different pharmacologic, metabolic or mechanical interventions. Most of these reports have been based on studies in which the test therapy was initiated either before or within the first 30 minutes after coronary artery occlusion. However, there are data based on electrocardiographic and histochemical criteria which have indicated that salvage of ischemic myocardium may still be possible by certain interventions 5 hours or longer after coronary occlusion in the experimental animal, although the effect of such a delay in therapy on anatomic infarct size has not been determined. From effects on the electrocardiographic criteria of ischemic injury, there have also been

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