Q-Second Sound Interval in Acute Myocardial Infarction

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In clinical practice an objective measurement of cardiac function is desirable in following patients with acute myocardial infarction. Ježek (1963) and Weissler, Harris, and Schoenfeld (1968) have suggested that the Q-S₂ time interval expressed as a fraction of the cardiac cycle length may be of value in the assessment of cardiac function. We have measured this interval in a series of normal patients and in those who have had a myocardial infarction. The purpose of this paper is to evaluate the practical value of this measurement.

SUBJECTS AND METHODS

One hundred normal subjects and 20 patients who had had acute myocardial infarction were studied. The normals consisted of 69 men and 31 women between the ages of 31 and 70. Clinical examination, electrocardiogram, and chest x-ray were done in each case. A group of 3 normal controls was studied daily for 10 days before and after exercise to note any deviation of Q-S₂ interval from the normal range.

The patients with acute myocardial infarction were included if they showed the typical signs of acute myocardial infarction with chest pain, diagnostic changes in electrocardiogram, and elevation of serum lactic dehydrogenase. Patients with bundle-branch block on the electrocardiogram were excluded from this study.

The patients were grouped clinically according to absence (Group I) or presence (Group II) of cardiac abnormalities, including the presence of a third heart sound, cardiomegaly, and pulmonary congestion. The maximum level of serum lactic dehydrogenase rise was also recorded.

The electrocardiogram and phonocardiogram were recorded simultaneously on 3-channel Elema Schonander Mingograph recorder at a paper speed of 100 mm./sec. Two electrocardiographic leads were used to

determine the onset of ventricular depolarization as one lead only could be misleading, as illustrated in Fig. 1. The crystal microphone was placed in the position which clearly showed the onset of initial high frequency vibrations of the first and second heart sounds. The Q-S₂ interval was measured from the onset of Q wave to the onset of aortic component of the second heart sound, which was taken as the beginning of first high frequency vibrations. Five consecutive cycles were measured in each tracing and averaged. The measurements were made during held quiet expiration in the supine position. The records in which the onset of ventricular depolarization or onset of second heart sound was not clear were discarded.

The patients were studied daily at a fixed time from

FIG. 1.—Simultaneous phonocardiogram and two leads of electrocardiogram, showing the value of recording two leads of electrocardiogram in measurement of Q-S₂ interval.

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FIG. 2.—The linear relation between Q-S₂ interval with heart rate in 100 normal subjects. Each dot represents Q-S₂ interval in a normal subject. ($R = -0.943$; $p = < 1 \times 10^{-7}$; regression equation for Q-S₂ interval $= 393.20 - 2.13$ (heart rate 71.0)).

the day of admission, after onset of symptoms, until the day of discharge.

The regression equation relating heart rate to Q-S₂ interval in normal subjects was calculated.

RESULTS

Q-S₂ Interval in Normal Subjects. Fig. 2 shows

the linear relation between Q-S₂ interval with heart rate in 100 normal subjects. No significant difference in Q-S₂ interval was found between various age-groups or between men and women.

Fig. 3 shows the Q-S₂ interval in 1 of the 3 normal subjects measured daily for 10 days before and after exercise. This showed no deviation from the

FIG. 3.—The daily measurements of Q-S₂ interval for 9 days before and after exercise in one normal subject.

TABLE
RELATION OF MAXIMAL PERCENTAGE DECREASE IN
Q-S₂ INTERVAL IN GROUP I AND II PATIENTS WITH
ACUTE MYOCARDIAL INFARCTION

Groups	No. of patients	Maximal per cent decrease in Q-S ₂ interval as compared to normal mean expected value at same heart rate		
		5-11 per cent	11-20 per cent	> 20 per cent
I	6	5	1	—
II	14	1	6	7

normal range from day to day and before or after exercise.

Q-S₂ Interval in Patients after Acute Myocardial Infarction. The Q-S₂ interval was shortened in all 20 patients with acute myocardial infarction. This shortening tended to return towards normal range during the 3-week recovery period. The patients were divided into the following groups, according to the percentage shortening of Q-S₂ interval as compared to the normal expected value at the same heart rate, namely, 5 to 11 per cent, 11 to 20 per cent, and more than 20 per cent. A comparison between these and the clinical groups I and II is shown in the Table. The relation between the peak level of lactic dehydrogenase and the maximum shortening of the Q-S₂ interval is shown in Fig. 4.

This figure shows that there is a correlation between the maximum peak level of serum lactic dehydrogenase and the maximum percentage shortening of Q-S₂ interval.

The following cases illustrate the patterns of change in the Q-S₂ interval in acute myocardial infarction.

Case 13. A 32-year-old man had severe retrosternal pain of 4 hours' duration, pulse rate was 110/min. regular, blood pressure 90/60 mm. Hg, third heart sound present, electrocardiogram showed extensive acute inferior myocardial infarction with lateral extension, chest x-ray showed moderate cardiomegaly with evidence of pulmonary congestion, lactic dehydrogenase rose to 1000 I.U. (65% heat stable). The changes in Q-S₂ interval are shown in Fig. 5. The return towards normal values was associated with clinical improvement and decrease in lactic dehydrogenase level.

Case 11. A 63-year-old man was admitted with severe retrosternal pain of 3 hours' duration. On admission pulse rate was 64/min. regular, blood pressure was 150/90 mm. Hg, no third heart sound present, no clinical evidence of cardiac failure, and electrocardiogram showed recent inferior myocardial infarction. Chest x-ray showed moderate cardiomegaly with clear lung fields, lactic dehydrogenase rose to 520 I.U. (45% heat stable). On admission the Q-S₂ interval was minimally decreased. On the 6th hospital day he suddenly

FIG. 4.—The relation between peak level of serum lactic dehydrogenase and maximum percentage shortening of Q-S₂ interval, as compared to normal expected value. ($R=0.504$; $p < 0.002$.)

FIG. 5.—Daily percentage decrease in Q-S₂ interval in Case 13, and progressive return towards normal range with recovery.

developed cardiac arrest, with ventricular fibrillation which was restored to sinus rhythm with defibrillation. Later his heart rate rose to 110/min., blood pressure 70/50 mm. Hg, and electrocardiogram showed fresh ischaemia in anterior wall. After this, he gradually improved. The changes in Q-S₂ interval are shown in Fig. 6, with maximum decrease after cardiac arrest.

Case 18. A 64-year old man, who had a previous antero-lateral acute infarction in April 1968, was readmitted in July with a praecordial pain of 5 days' duration; pulse rate was 80/min. regular, blood pressure 100/60 mm. Hg, pericardial friction rub present, moderate cardiomegaly, delayed grade I apical systolic murmur and third heart sound at apex, congestive cardiac failure with jugular venous pressure of 4 cm., bilateral basal râles, and pleural effusion left base. Lactic dehydro-

genase was 1000 I.U. (60% heat stable). Electrocardiogram showed old antero-lateral infarction. Two days later he developed attacks of recurrent praecordial pain and frequent ventricular extrasystoles. Electrocardiogram showed fresh T wave changes suggestive of ischaemia. He was treated with digoxin and diuretics, with uneventful recovery. Fig. 7 shows the percentage shortening of Q-S₂ interval, with progressive return to normal with recovery.

Of the 2 patients who died, one (Case 20) died suddenly of cardiac asystole on the 11th hospital day. Necropsy revealed rupture of the left ventricle. Q-S₂ interval changes are shown in Fig. 8. The other patient (Case 19) died in extreme circulatory failure and shock on the 13th hospital day,

FIG. 6.—Daily percentage decrease in Q-S₂ interval in Case 11. This graph illustrates maximum shortening of Q-S₂ interval after ventricular fibrillation and cardiac arrest on 6th hospital day.

FIG. 7.—Percentage decrease in Q-S₂ interval in Case 18, with return to normal with recovery.

with a marked decrease of Q-S₂ interval on the day before death as shown in Fig. 9.

No correlation was found between the site of infarction, anterior, inferior, and posterior, and the degree of Q-S₂ shortening.

DISCUSSION

The regression line relating Q-S₂ interval to heart rate in our series of normal subjects is similar to that of Weissler *et al.* (1968). The Q-S₂ interval varies inversely with heart rate, and no significant

difference was found for various age-groups or sex. In addition, this study shows that there is no day-to-day variation before or after exercise in healthy normal subjects. In normal subjects (Weissler *et al.*, 1965, 1968) and in experimental animals (Wallace *et al.*, 1963) the inverse relation between the Q-S₂ interval to heart rate is due primarily to shortening of left ventricular ejection.

The mechanism of the shortening of the Q-S₂ interval after acute myocardial infarction is not clear. It may be due to a diminished stroke volume

FIG. 8.—Daily percentage decrease in Q-S₂ interval in Case 20. The patient died suddenly of rupture of the left ventricle on the 11th hospital day.

FIG. 9.—Conspicuous decrease in Q-S₂ interval on day before death in a patient with cardiogenic shock (Case 19).

and ejection time due to failure of the myocardium *per se*; on the other hand it may be associated with the increase in catecholamine excretion found in these patients (Nuzum and Bischoff, 1953; Gazes, Richardson, and Woods, 1959). Valori, Thomas, and Shillingford (1967) and Wallace (1968) have shown there to be a considerable increase in catecholamine excretion in the urine in patients seriously ill after myocardial infarction, and that this increase bears a relation to the severity of the cardiac failure. This theory would also explain the return to normal over a period of a few days, as was also shown by Ježek (1963). Further work is necessary to correlate these factors, but the Q-S₂ interval seems to be a useful empirical additional objective guide in assessing disturbance of cardiac function after myocardial infarction. It has the advantage of simplicity and does not disturb the patient in any way.

SUMMARY

The Q-S₂ interval has been studied in 100 normal subjects and 20 patients with acute myocardial infarction. All the patients with acute myocardial infarction showed shortening of the Q-S₂ interval in the acute stage of the illness, with a tendency to return towards normal with clinical recovery. The degree of shortening correlates with the clinical state and serum lactic dehydrogenase levels. The significance of these findings is discussed.

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