EXERCISE PHONOCARDIOGRAM:
WITH SPECIAL REFERENCE TO Q-II_A INTERVAL ON EXERCISE

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In the past, the Q-II_A interval (from the beginning of the QRS complex to the beginning of the aortic component of the second heart sound) has indicated as an index of the mechanical duration of the left ventricular systole. It was reported that the II_A was closely associated with the incisura of the aortic pressure! A high degree of linear correlation between Q-II_A and the duration of the left ventricular systole was reported by Shahr and Slodki2. A paradoxical splitting of the second heart sound is a very important sign of the left ventricular dysfunction3-10. The great prolongation of Q-II_A may cause the paradoxical splitting of the second heart sound, but in mild dysfunction of the left ventricle elicted by systemic hypertension or ischemic heart disease, paradoxical splitting of the second heart sound is not always found.

A latent delay of the aortic closure without paradoxical splitting has not been estimated as a clinical diagnosis of the left ventricular dysfunction. In order to estimate the delay of the aortic closure, a relationship between Q-II_A and cycle length (R-R) was studied in normal subjects, and a formula for calculating the expected Q-II_A interval for a given cycle length was obtained. Using this formula in the phonocardiographic exercise test, we have found significant results in the changes in percent Q-II_A corrected by cycle length (Q-II_C index) after exercise in patients with systemic hypertension, arteriosclerosis, and ischemic and/or hypertensive heart disease. We believe that the changes in Q-II_C index after exercise may help for evaluating the left ventricular reserve capacity.

MATERIALS AND METHODS

The materials used in the present study were 170 patients, aged ranging from 18 to 71. They were divided into four groups: 87 normal subjects, 35 hypertensive subjects, 29 arteriosclerotic subjects, and 19 subjects with ischemic and/or hypertensive heart disease, all of which were selected by using the criteria in Table 1.

In all subjects, a direct-writing high-frequency

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<th>TABLE 1 CRITERIA FOR DIAGNOSIS</th>
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<td>Ischemic and/or hypertensive heart disease</td>
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<td>Arteriosclerosis</td>
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<td>Hypertension</td>
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Key Words: Exercise phonocardiogram
The left ventricular mechanical systole
Ischemic heart disease

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Japanese Circulation Journal Vol. 35, December 1971 1551
(120–500 cps) phonocardiogram with a microphone placed at the pulmonic area and electrocardiogram using lead II were taken simultaneously before and one, three, and six minutes after Master’s double two-step test. Phonocardiographic microphone was placed in the same position as accurately as possible before and after exercise.

R-R and Q-II A in the first two or three end-expiratory cycles were measured and averaged.

RESULTS

1. End-expiratory Q-II A and R-R intervals in normal subjects

A linear relationship between Q-II A and the square root of R-R was demonstrated by statistical analysis. The correlation coefficient between the two was 0.82 (p<0.005). The results on 101 normal subjects, aged ranging from 18 to 66, are presented in Fig.1. The predicted Q-II A interval for a given R-R interval can be expressed as:

\[
Q - II_A = 8.7 \sqrt{R-R} + 105.8 \text{ msec}
\]

\[
(p<0.005, r = 0.82)
\]

Therefore, Q-II C index (percentage of the measured Q-II A to the predicted Q-II A) can be expressed as:

\[
Q - II_C \index = \frac{\text{Measured } Q - II_A}{\text{Predicted } Q - II_A} \times 100% = \frac{\text{Measured } Q - II_A}{8.7 \sqrt{R-R} + 105.8} \times 100%
\]

In 90% of normal subjects, Q-II C index was ranged from 90 to 110%. 

2. Changes in Q-II C index before and after exercise

A. Normal group: The changes in Q-II C index before and one, three and six minutes after Master’s double two-step test in 87 normal subjects were presented in Fig.2. Before exercise, Q-II C index ranged from 88.7 to 116.2% with an average of 101.0%. In 60 elderly subjects with their age of 30 years or more, the averaged value for Q-II C index was 99.6% and in 27 younger subjects with 29 years or less, it tended to be greater than in elderly subjects and averaged 104.2%. The difference was statistically significant (p<0.005).

One minute after exercise, Q-II C was decreased in 78 cases (90% of all normal subjects), and the averaged value (94.6%) was significantly less in all normal subjects than that of pre-exercise value (p<0.005). In younger subjects, the average decrease in Q-II C index was 9.8%. While in elderly subjects it was 4.9%, and the difference was statistically significant (p<0.005). Therefore, Q-II C index one minute after exercise in the younger subjects was almost identical value as in the elderly subjects. Three and six minutes after exercise, Q-II C index returned to the pre-exercise level but the averaged value was not greater than the control.

Fig.1. End-expiratory Q-II A intervals vs. \(\sqrt{R-R}\) in 101 normal subjects.

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B. Hypertensive group: Fig. 3 shows the changes in Q-II\textsubscript{C} index before and after exercise in 35 hypertensive subjects. Before exercise, Q-II\textsubscript{C} index was greater than in elderly normal subjects (p<0.005). It ranged from 93.5 to 112.1% with an average of 104.1%.

One minute after exercise, Q-II\textsubscript{C} index was decreased in 32 cases (86%). The average value was 98.3%, and it was significantly greater than that of normal subjects (p<0.005). The average decrease was 5.8% which was as the same value as in normal subjects.

Three and six minutes after exercise Q-II\textsubscript{C} index returned to the pre-exercise level in the same fashion as in the normal subjects.

C. Arteriosclerotic group: Fig. 4 shows the changes in Q-II\textsubscript{C} index before and after exercise in 29 arteriosclerotic cases. Before exercise Q-II\textsubscript{C} index ranged from 91.1 to 109.9% with an average of 101.8% which was almost the same as normal.

One minute after exercise, Q-II\textsubscript{C} index was decreased in 22 cases (82%). The average value for Q-II\textsubscript{C} index was 98.8% and it was significantly greater than in the normal subjects (p<0.005). The average decrease was 3.0% which was less than in the normal and hypertensive subjects (p<0.1).

Three and six minutes after exercise Q-II\textsubscript{C} index was tended to be greater than the pre-exercise level, and the average value for Q-II\textsubscript{C} index was 101.1 and 104.1%, respectively.

D. Ischemic and/or hypertensive heart diseased group: Fig. 5 shows the changes in Q-II\textsubscript{C} index in 19 cases of ischemic and/or hypertensive heart disease before and after exercise. In all subjects the exercise test was completed without severe complaints such as chest pain, arrhythmia or dyspnea. In 2 cases (dotted lines in Fig. 5) phonocardiogram was not recorded one minute after exercise. Before exercise, Q-II\textsubscript{C} index was ranged from 96.1 to 117.0% with an average of

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**Fig. 2.** Changes in Q-II\textsubscript{C} index before and after exercise in normal group. In this and following figures, unit on the ordinate is the deviation of Q-II\textsubscript{C} index from 100%.

**Fig. 3.** Changes in Q-II\textsubscript{C} index before and after exercise in hypertensive group.
105.8% which was 6.2% greater than in the elderly normal group (p<0.005). In 5 cases (26%) of them, Q-II C index was more than 110%, while in the normal subjects Q-II C index over 110% was found only in 5 cases (6%) out of 87 subjects.

One minute after exercise, Q-II C index was increased above the pre-exercise level in 7 cases (41%) out of 17 subjects. The average value of Q-II C index was 103.6% which was significantly greater than in the normal subjects (p<0.005) and the average decrease was only 2.2% which was the smallest among the four groups.

In the cases in which Q-II C index showed a decrease one minute after exercise, Q-II C index returned to the pre-exercise level three or six minutes after exercise, while in the cases in which Q-II C index was increased one minute after exercise, Q-II C index tended to persist at high level. The averaged Q-II C index three and six minutes after exercise was 106.1 and 107.5%, respectively.

The changes in the averaged Q-II C index before and after exercise are summarized in Fig.6. Before exercise, the averaged Q-II C index was greatest in the subjects with ischemic and/or hypertensive heart disease. The averaged Q-II C index in the hypertensive group and the arteriosclerotic group was greater than in the normal group, but there was no significant difference between the normals and the arteriosclerotics, and between the young normals and the hypertensives.

One minute after exercise, the averaged Q-II C index was decreased most remarkably in the normal group, especially in the younger normal group. The difference in Q-II C index between the normal and the other three groups was more evident after exercise.

**DISCUSSION**

1. Evaluation of Q-II A

There is another type of paradoxical splitting which is independent to the left ventricular function and is seen in WPW syndrome and tricuspid insufficiency. This condition gives a paradoxic split due to early occurrence of the pulmonary component of the second sound. Therefore, if a delay of the aortic component is evaluated only by presence of paradoxic split of the second sound the evaluation is considered to be insufficient.

In order to estimate the left ventricular mechanical systole, there have been some investigations dealing with Q-II_A/Q-T and the interval between the end of T-wave and aortic closure sound. There are some disadvantages on this kind of studies which pays special attention to the T-wave on ECG, because the T-wave tends to be easily changeable in shape in the presence of myocardial damage and the end point of the T-wave is sometimes difficult to be pointed out. Many authors have tried to estimate Q-II_A or ejection period during angina pectoris and after exercise, which are also not much valuable, because they are considerably influenced with the variation of the heart rate.

On the basis of correlation between Q-II_A and heart rate, there have appeared Q-II_A/√R-R, the difference between the measured Q-II_A and the predicted Q-T interval using Heghlin-Holmzmann’s formula (Q-T = a√R-R) and Q-II_A/√R-R. These parameters listed here, however, were assumed as Q-II_A was proportionate to √R-R or R-R, but they were not fully proven. Several reports have been published, which included Q-II = -a X heart rate + b, Q-II_A = a√R-R + b and Q-II_A = a√R-R.

At the result of preliminary analysis on 101 normal subjects, we have found that Q-II_A vs. √R-R could be demonstrated as a linear distribution in a range from 50 to 120/min of the heart rate, and we obtained a formula where Q-II_A = 8.7 X √R-R + 105.8 msec (p<0.005, r=0.82). It is, therefore, worthwhile for us to use the final formula: Q-II_C index = (Measured Q-II_A)/(8.7 X √R-R + 105.8) X 100% for evaluation of Q-II_A.

Thus, by using this formula, we were able to find out the delayed II_A in cases having ischemic and/or hypertensive heart disease, arteriosclerosis and hypertension. The result obtained from our study showed a similarity as results from a prolongation of Q-II_A in the case of hypertension and in the premature newborn and it would be a helpful procedure which is able to evaluate a relationship quantitatively between pressure and...
volume load of the heart and the heart muscle contractility.

2. Changes in the II_A component before and after exercise

Several authors describe that Q-II_A becomes shortened after exercise. In the majority of these literatures dealing with Q-II_A or ejection time no significant differences have been observed between normal and diseased subjects. These data, however, may not be exactly evaluated on a variation of the II_A component, because, as described before, Q-II_A has a strong dependance to the heart rate. It is reported that after physical exercise the ejection rate is hardly increased or is decreased in coronary heart disease and in congestive heart failure and that the paradoxical split of the second sound is observed only after exercise in myocardial infarction. Our result revealed that by exercise the shortening of Q-II_C index was most remarkable in younger normal people and was less remarkable in the elderly healthy persons, hypertensives, arteriosclerotics and ischemic and/or hypertensive heart disease patients in this order.

According to Heglin the shortening of Q-II_A is divided into two groups. One is hyperdynamic and the other is hypodynamic shortening. In general, exercise acts as a positive chronotropic effect and also positive inotropic effect to the heart. Therefore, the shortening of Q-II_C index is considered to correspond a hyperdynamic shortening due to positive inotropic action. After exercise, in this study, inotropic effect which corresponded to chronotropic effect might not be fully demonstrated in ischemic and/or hypertensive heart disease group, while this might be completely demonstrated in younger healthy group. These speculations should be allowed from the different behavior of Q-II_C index one minute after exercise between these two studied groups. Fig.7 shows the changes in the mean square root of the R-R interval and the mean measured Q-II_A interval before and one minute after exercise. The gradient of the line, showing the changes in \( \sqrt R-R \) and Q-II_A, was most steep in the young normals and most gentle in the ischemic and/or hypertensive heart disease group. Therefore, the normal group accomodated to the exercise chiefly by shortening mechanical systole and diseased groups did it by increasing their heart rate.

No one revealed definite paradoxical splitting in our studied subjects either before or after exercise, but it is noteworthy that there was some difference in post-exercise variation of Q-II_C index between the normal and the other groups. In some cases with ischemic and/or hypertensive heart disease, Q-II_C index exceeded the resting level at 3 and 6 minutes after exercise, which is suspected to have a further depression of the myocardial reserve by exercise in diseased individuals.

3. Significance of Q-II_A

There are a number of reports explaining paradoxical splitting of the second heart sound or prolongation of Q-II_A in left ventricular dysfunction. On the other hand, some authors have postulated a clinical significance of the shortened Q-II_A in heart failure especially in the patient with Heglin's syndrome. In coronary patients, only 1 out of 41 cases showed Heglin's syndrome at resting condition and 3 showed post-exercise appearance of this phenomenon. Shortening of Q-II_A in patients

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*Figure 7. Changes in Q-II_A vs. \( \sqrt R-R \) before and 1 minute after exercise in the young normal and elderly normal groups and the diseased groups.*

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with myocardial dysfunction may be related to the decrease in stroke volume. In our present study, however, no patient with ischemic heart disease demonstrated the shortened Q-II C index of less than 90% either in the pre- or post-exercise period, and no one had a sign of overt left ventricular failure. Therefore, it is reasonable that in case of myocardial dysfunction without a reduction of stroke volume, Q-II C index discloses its prolongation, and a failure to decrease in Q-II C index following stress upon the heart may happen. Q-II C index may be shortened in clinical conditions of hyperdynamic status such as physical effort, thyrotoxicosis and sympathicotonia. It may be prolonged and may fail to decrease on physical effort in mild myocardial damage, and if failure becomes advanced and is accompanied with a reduction of stroke volume, it may elicit its shortening again.

**SUMMARY**

The changes in the Q-II A interval were analysed using direct-writing phonocardiograph and Master's double two-step test for the purpose of early detection of the left ventricular dysfunction in cases of systemic hypertension, arteriosclerosis and ischemic and/or hypertensive heart disease.

A linear relationship between the Q-II A interval and the square root of the R-R interval was demonstrated by statistical analysis in 101 normal subjects aged ranging from 18 to 66. Using this relationship, the Q-II A interval was corrected by cycle length and Q-II C index was obtained.

The changes in Q-II C index before and after exercise were studied. A significant prolongation of Q-II C index before exercise and a failure to decrease in Q-II C index 1 minute after exercise was demonstrated in the diseased groups.

From the result of this study, the phonocardiographic analysis of the Q-II A interval before and after exercise is considered as a useful diagnostic procedure for evaluation of the left ventricular dysfunction.

**Acknowledgement**

The author wishes to express his gratitude to Dr. Isao Niki and Dr. Toshitami Sawayama for their helpful advice and excellent guidance.