P WAVE CHANGES IN BODY SURFACE POTENTIAL MAPS DUE TO INCREASING HEART RATE DURING EXERCISE IN NORMALS

YOSHIHIKO WATANABE, M.D., YOSHIHARU KOHGAME, M.D., HIROSHI NAKANO, M.D.
YASUHIRO ABO, M.D., AND YASUSHI MIZUNO, M.D.

Exercise-induced changes in P maps were investigated in normal subjects (n = 20) in order to clarify the basic changes in P maps caused by exercise in patients with ischemic heart disease or by exercise-induced dysfunction of the left ventricle in patients with various heart diseases.

Maps were obtained using an 87 lead-point system (HPM-6500, Chunichi-Denshi Corporation) and were recorded at rest and at heart rates up to 140/min in 20/min steps under graded bicycle ergometric exercise.

P waves were divided into 3 phases (initial, middle and terminal) by locating the maximum. Maps were characterized by the distribution pattern of the positive and negative potentials in each frame.

Peak voltages increased proportionally to the increase in heart rate. We observed a decrease in P wave duration concomitant with the increase in heart rate. Time from P onset to peak voltage increased slightly. We believe these findings to be due to the acceleration of sympathetic nerve tone accompanying exercise. We observed 2 patterns: type A showed the relatively short middle and terminal phases, and type B a prolongation of the middle phase and a shortening of the terminal phase. We consider the differences between types A and B to be partly due to individual differences in the degree of increase in pulmonary air volume and sympathetic nerve tone influence on the atria.

In evaluating the exercise-induced P map changes, special consideration should be paid to the changes due to increase in heart rate.

BODY surface potential maps (maps) are constructed from unipolar electrocardiograms (ECGs) recorded from multiple sites on the torso, and are apparently superior to the standard 12-lead electrocardiograms for the assessment of fine regional variations caused by the myocardial electrical excitation sequence.

Many reports have already been published on the QRS changes in maps in various heart diseases, map changes in response to exercise in both normal subjects and cardiac patients have

been described extensively, especially the ST-segment and R-wave.¹⁻⁶ However, low level potential events such as P waves have not yet been clinically investigated using maps. A few reports have been published on P waves in normals (Taccardi,⁷ Mirvis⁴), in dogs (Spach),^{8,9} in normal subjects and those with valvular disease and ectopic beats (Kawano),^{10,11} and in patients with pneumoconiosis (Kohgame),¹² Moreover, a precise investigation had not yet been performed on the variations or changes induced by exercise.

P maps under exercise are thought to be highly sensitive for the diagnosis of atrial performance on cardiac function in patients with

Key words:

Body surface potential mapping
P wave
Ergometric exercise testing

(Received September 18, 1987; accepted November 7, 1987)

Department of Internal Medicine, Fujita-Gakuen Health University School of Medicine, Toyoake, Aichi, Japan

Mailing address: Yoshihiko Watanabe, M.D., Department of Internal Medicine, Fujita-Gakuen Health University School of Medicine, 1-98 Kutsukake-cho, Toyoake, Aichi 470-11, Japan

Fig.1. Definition of initial, middle and terminal phases of P maps.

Initial phase: the period in which the maximum is located at the mid-sternal portion.

Middle phase: the period in which the maximum moves from the mid-sternal portion to the left axillary line.

Terminal phase: the period in which the maximum migrates from the left axillary line to the back.

Fig.2. Changes in P wave duration in relation to heart rate increase.

P duration decreased along with the increase in heart rate, from 89.8 ± 9.6 at rest to 84.4 ± 12.7 at +60 over resting heart rate.

various heart diseases. In this study, we investigated the kinds of P map variations that were induced in normal subjects in relation to heart

rate changes during bicycle ergometric exercise as a means of evaluating exercise P maps.

Fig.3. Changes in peak P voltage in relation to heart rate increase.

Peak P voltage increased from 0.081 ± 0.017 mV at rest to 0.11 ± 0.058 mV at +40 over resting heart rate ($p < 0.05$).

METHODS

Subjects consisted of 20 healthy male volunteers aged 20 to 30 years (mean 21 years). Each subject provided a detailed medical history and underwent a physical examination by an internist and a standard 12-lead electrocardiographic study. All subjects were normotensive, none had a history of chest pain or cardiac dysfunction and physical findings in all cases were normal.

Body surface potential mapping was performed using an HPM-6500 system¹³ (Chunichi-Denshi Corporation), equipped with 96 amplifiers, a 12-bit A-D converter and an 8-bit micro-computer. Fifty-nine leads were located on the anterior chest, 28 on the back and an additional 9 were used for standard 12-lead electrocardiograms and vectorcardiograms.

Simultaneous data were obtained from all 87 leads in digital form, at a sampling rate of 500 or 1000 samples/sec/channel, for 8 seconds using the Wilson central terminal potential as reference. Sequential beats were averaged for an 8 second period after time alignment to improve signal to noise ratio and eliminate a baseline shift due to respiration or body movement.

The exercise test included the use of a bicycle ergometer (Monark Co. Ltd) in a supine position. Maps were recorded at rest and at 20 beats/min increments in heart rate up to 140 beats/min during exercise. The work load started at 25 watt/sec. Cuff blood pressure was measured at the time of map recording.

Body surface maps were analyzed as follows.

The onset and offset of P waves were determined visually by Frank vectorcardiographic X, Y, Z leads. Baseline drift is one of the practi-

Fig.4. Changes in time from P onset to peak voltage in relation to heart rate. Peak time increased from 38.0 ± 9.5 msec at rest to 44.4 ± 13.0 msec at +60 over resting heart rate.

cal problems which must be considered in the interpretation of exercise ECGs. We used the U-P interval as voltage reference for P wave measurements. Because U and P waves merge and the offset voltages are difficult to determine at heart rates of more than 140 beats/min, only data heart rates of up to 140 beats/min were used in this study.

(1) The voltages and locations of the maximum and the minimum of each map frame, and the time to peak voltage, were studied with heart rate changes sequentially from P onset.

(2) The following 3 phases were determined according to the location of the maximum, i.e. an initial phase (from the onset of the P wave to the time when the maximum migrates and stays at the mid-sternal line of the fifth intercostal space), a middle phase (from the end of the initial phase to the time when the maximum migrates to the mid-axillary line at the level of the 6th or 7th intercostal space), and a terminal phase (from the end of the middle phase to the time when the maximum shifts to the left upper back through the left lateral chest) (Fig. 1).

All data are expressed as mean $\pm$ SD.

Statistical analysis was performed using a paired t-test.

RESULTS

The pressure rate product (PRP) at rest was 8160 ± 789 (mmHg·beat/min), during exercise PRP was 11668 ± 930 (mmHg·beat/min) at +20 delta heart rate above resting heart rate (DHR), 16565 ± 1714 (mmHg·beat/min) at +40 DHR, and 22358 ± 2132 (mmHg·beat/min) at +60 DHR. The PRP ratio of exercise to rest was 1.43 at +20 DHR, 2.03 at +40 DHR and 2.74 at +60 DHR.

(1) Changes in P wave duration

P wave duration was 89.8 ± 9.6 msec at rest, 89.9 ± 12.5 msec at +20 DHR, 87.6 ± 14.9 msec at +40 DHR, and 84.4 ± 12.7 msec at +60 DHR. Thus decrease in P wave duration was observed concomitant with heart rate increase. (Fig. 2)

(2) Changes in peak P voltage

The peak P voltages were 0.081 ± 0.017 mV at rest, 0.090 ± 0.026 mV at +20 DHR, $0.110 \pm$

Fig.5. Changes in duration of initial, middle and terminal phases in relation to heart rate. In the middle phase, duration increased along with an increase in heart rate, especially in type B ($p < 0.05$).

0.058 mV at +40 DHR, and 0.108 ± 0.046 mV at +60 DHR. The peak P voltage increased with increasing heart rate. There was a statistically significant difference ($p < 0.05$) between the mean peak P voltage at rest and that at +40 DHR. (Fig. 3)

(3) Changes in time from P wave onset to peak P voltage (peak time). (Fig. 4)

The peak time was 38.0 ± 9.5 msec at rest, 42.2 ± 11.6 msec at +20 DHR, 42.8 ± 11.9 msec at +40 DHR, and 44.4 ± 13.0 msec at +60 DHR. There was a tendency to prolongation of peak time with heart rate increase.

(4) Changes in the duration of initial, middle and terminal phases induced by exercise

Generally speaking, the initial and middle phases were prolonged with increasing heart rate in contrast to a decrease in the terminal phase.

Especially at +60 DHR, the terminal phase was significantly shortened, as compared with

that at rest. (Fig. 5)

(5) Types observed in P maps during exercise

Twelve subjects were classified as type A and 6 as type B according to difference in duration of the middle phase.

The maps were classified into 2 types (A and B).

(a) Characteristics of type A

This type showed a relative shortening of the middle and terminal phases; in other words, a quick activation conduction in the right atrium and relatively early initiation of left atrium activation. (Fig. 6a)

(b) Characteristics of type B

This type showed a prolongation of the middle and shortening of the terminal phase. The maximum was located to the right inferiorly, and the minimum stayed at the same position during exercise. The maximum was therefore located beneath the minimum for most of the atrial

TYPE A

Fig.6A. A typical P map showing type A.

Type A shows the maximum moving to the back relatively quickly.

TYPE B

Fig.6B. A typical P map showing type B.

Type B shows the maximum staying at the right lower chest almost during the whole of atrial activation.

activation. (Fig. 6b)

The PRPs of type A were 8288 ± 713 (mmHg·beat/min) at rest, 11603 ± 995 (mmHg·beat/min) at +20 DHR, 16244 ± 1906 (mmHg·beat/min) at +40 DHR and 21880 ± 1823 (mmHg·beat/min) at +60 DHR. The PRPs of type B were $7620 \pm$

912 (mmHg·beat/min) at rest, 11506 ± 762 (mmHg·beat/min) at +20 DHR, 15926 ± 1448 (mmHg·beat/min) at +40 DHR and 22403 ± 1905 (mmHg·beat/min) at +60 DHR. There were no significant differences in PRPs between the two groups.

DISCUSSION

Exercise testing is a popular clinical method for the assessment of cardiac function in patients with ischemic and various other cardiac diseases. Until recent years, attention was focused mainly on ST-T and R amplitude changes induced by exercise. In this study, P waves were selected for the purpose of early detection of heart diseases, i.e., detection of atrium overload caused by ventricular dysfunction. For basic evaluation of exercise-induced atrium overload, the responses recorded in P maps during exercise on normal subjects were studied in relation to heart rate increment.

In our results, we noted several changes in P waves using body surface potential maps in normal subjects during exercise.

- (1) Decrease in P duration
- (2) Increase in peak P voltage
- (3) Prolongation of time from P onset to peak voltage
- (4) Prolongation of the middle and shortening of the terminal phase
- (5) Two typical patterns, A and B

These characteristics were observed in relation to heart rate changes induced by exercise. The main or essential factors for these changes might be explained as follows: the conduction velocity over the atria is accelerated¹⁴ in accordance with increasing heart rate due to exercise, because of a decrease in parasympathetic tone and an increase in sympathetic tone. These effects result in a decrease in P wave duration and an increase in P amplitude.

A considerable influence of the increase in pulmonary volume was noted on lead vectors or transfer impedance from the heart to lead points on the body surface during exercise. These lead vector changes are thought to be due to an increase in the vertical and decrease in the horizontal components (as reported by Nagata et al), causing a vertical shift in the location of maximum and minimum through atrial activation.

Moreover, an inevitable overlap of U and P wave was observed with rapid heart rates. This might cause a baseline shift and changes in the potential distribution pattern. Hence, in this study, data for heart rates of more than 140 were excluded. The slight reduction of the peak P amplitude at +60 DHR over HR at rest was probably due to such a distortion.

Two different patterns were observed during exercise in apparently healthy subjects. Type A

showed only a slight shortening of the initial, middle and terminal phases, and hence a quick backward shift of the maximum. Type B showed a remarkable prolongation of the middle phase and a considerable shortening of the terminal phase.

In type A, a synchronization of the right and left atria was more noticeable than in type B. In type A, therefore, the maximum moved to the back more quickly than in type B, indicating a relatively early depolarization of the appendage of the left atrium.^{15,16}

In type B, there is a prolonged duration of the middle phase probably caused by an increased amplitude in the right atrium and a relatively late activation of the left.

These pattern changes might be caused by the difference in increasing pulmonary air volume and/or sympathetic nerve tone influence on the atria.

Cardiovascular responses to exercise are complex and involve changes in myocardial contractility, ventricular volume, heart rate, and arterial and venous tone. As reported by Sorensen,¹⁷ hemodynamic changes during exercise occurred as follows: the left ventricular ejection fraction increased by approximately 10% and stroke volume by 30%. Contractility also increased along with increasing heart rate, while end-diastolic volume and mean pulmonary wedge pressure did not change significantly. The increase in peak P amplitude could not be explained merely by the changes in hemodynamic parameters.

We conclude that the evaluation of P maps in exercise testing should be carefully conducted, bearing in mind the effects induced by changes in heart rate and autonomic nerve tone.

REFERENCES

1. HIGGINBOTHAM MB, MORRIS KG, COLEMAN E, COBB FR: Sex-related differences in the normal cardiac response to upright exercise. *Circulation* 70: 357, 1984
2. McPHERSON DD, HORACEK BM, SUTHERLAND DJ, ARMSTRONG CS, SPENCER CA, MONTAGUE TJ: Exercise electrocardiographic mapping in normal subjects. *J Electrocardiol* 18: 351, 1985
3. KUBOTA I, WATANABE Y, HARADA M, KAMINISHI T, TSUIKI K, YASUI S: Treadmill stress testing using body surface mapping in coronary artery disease - the clinical significance of ST depression. *Jpn Circ J* 46: 8, 1982
4. MIRVIS D, KELLER FW, COX JW, ZETTERGREN DG, DOWDIE RF, IDEKER RE: Left

- precordial isopotential mapping during supine exercise. *Circulation* 56: 245, 1977
5. MILLER WT, SPACH MS, WARREN RB: Total body surface potential mapping during exercise: QRS-T wave changes in normal young adults. *Circulation* 62: 632, 1980
 6. MIRVIS DM: Body surface distribution of exercise-induced QRS changes in normal subjects. *Am J Cardiol* 46: 988, 1980
 7. TACCARDI B: Body surface distribution of equipotential lines during atrial depolarization and ventricular repolarization. *Circulation Res* 14: 865, 1966
 8. SPACH MS, KING TD, BARR RC, BOAZ DE, MARROW MN, HERMAN-GIDDENS S: Electrical potential distribution surrounding the atria during depolarization in the dog. *Circulation Res* 24: 857, 1967
 9. KING TD, BARR RC, HERMAN-GIDDENS S, BODZ DE, SPACH MS: Isopotential body surface maps and their relationship to atrial potential in the dog. *Circulation Res* 30: 393, 1972
 10. KAWANO S, YAMAMOTO M, SAWANOBORI T, HIRAOKA M, SAKAMOTO Y: The body surface isopotential distributions during atrial excitation in atrial disorders. *Jpn Circ J* 45: 915, 1981
 11. KAWANO S, SAWANOBORI T, HIRAOKA M: Human body surface mapping during atrial depolarization in normal and diseased subjects. *J Electrocardiol* 16 (2): 151-160, 1983
 12. KOHGAME Y: Body surface potential mapping in patients with pneumoconiosis. *J of Fujita Gakuen Medical Association* 12: 29, 1983 (in Japanese)
 13. WATANABE T, TOYAMA J, TOYOSHIMA H, OGURI H, OHNO M, OHTA T, OKAJIMA M, NAITO Y, YAMADA K: A practical micro-computer-based mapping system for body surface, precordium and epicardium. *Comput Biomed Res* 14: 341, 1981
 14. ATTERHOG JH, LOOGNA E: P-R interval in relation to posture and autonomic tone. *J Electrocardiol* 10: 331, 1977
 15. IRISAWA H, SEYAMA I: The configuration of the P-wave during mild exercise. *Am Heart J* 71: 467, 1966
 16. RAUTAHARJU PM, PUNSAR S, BLACKBURN H, WARREN J, MENOTTI A: Waveform patterns in Frank-lead rest and exercise electrocardiograms of healthy elderly men. *Circulation* 48: 541, 1972
 17. SORENSEN SG, RITCHIE JL, CALDWELL JH, HAMILTON GW, KENNEDY JW: Serial exercise radionuclide angiography validation of count-derived changes in cardiac output and quantitation of maximal exercise ventricular volume changes after nitroglycerin and propranolol in normal men. *Circulation* 61: 600, 1980