HYPOXEMIA VECTORCARDIOGRAPHY

JUN-ICHI YOSHIDA

Loading electrocardiography was performed by using the following stresses: (1) Drugs (epinephrine, pitressin), (2) hypoxemia, (3) exercise and (4) atrial pacing.

In the 1930s, epinephrine and pitressin were used for diagnosis of coronary insufficiency, but these drugs are not recommended because of slow recovery of the induced coronary insufficiency.

In the 1940s, the hypoxemia test was established by Levy et al. and has been examined by many investigators from different points of view. But the test was not as popular as the exercise test because of the particular instrument used.

In the 1950s, the ‘two-step test’ as an exercise test was introduced by Master, and this is still the most frequently and widely used because of its simplicity. However, nowadays, the treadmill or bicycle ergometer tests as exercise tests are increasingly used.

Recently ‘atrial pacing’, i.e., the increase of heart rate by electrical atrial stimulation with an electrode in the right atrium, has been added to the electrocardiographic stress tests. The influence of this stress is immediately terminated with cessation of the stimulation, in contrast to other stress tests. However, the need of catheterization for the test limits wider application.

What is the ideal stress to get the best loading vectorcardiogram (VCG) convenient to observe the electrical activity of the heart as faithfully as possible both quantitatively and qualitatively?

Simple anesthesia apparatuses having been popularized in recent years, it is possible to perform the hypoxemia test simply, exactly and safely. Thus the hypoxic effect to VCG was analysed in controls and patients in this study.

Examinees

The examinees, as shown in Table 1, consisted of 104 cases, 71 males and 33 females, who were admitted or ambulatory in the 2nd Department of Internal Medicine. The age ranged from 16 to 77 years.

The subjects were divided into the following 2 groups:
(1) The control group consisted of 47 subjects, 32 males and 15 females, who had no history of cardiovascular disease and no evidence of cardiopulmonary abnormalities by physical, roentgenological and electrocardiographic examinations.
(2) The patient group consisted of 57

<table>
<thead>
<tr>
<th>TABLE 1</th>
<th>EXAMINEES BY AGE GROUP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>No. of Cases</td>
</tr>
<tr>
<td>16 – 77 yr.</td>
<td></td>
</tr>
<tr>
<td>16 – 77 yr.</td>
<td>40 – 59</td>
</tr>
<tr>
<td>16 – 77 yr.</td>
<td>60</td>
</tr>
<tr>
<td>Mean or Total</td>
<td>40.1 yr.</td>
</tr>
<tr>
<td>16 – 77 yr.</td>
<td>50 – 65</td>
</tr>
<tr>
<td>16 – 77 yr.</td>
<td>60</td>
</tr>
<tr>
<td>Mean or Total</td>
<td>48.2 yr.</td>
</tr>
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</tbody>
</table>

Key Words:
Hypoxemia test
Ecg oximeter
Vectorcardiography
X-, Y- and Z-lead electrocardiograms
QRS-T angle

(Received on April 25, 1974; Accepted for publication: December 19, 1974)
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Japanese Circulation Journal Vol. 39, June 1975 665
subjects, 36 had ischemic heart disease (20 without typical anginal pain, 6 with anginal pain and 10 with old myocardial infarction), 16 had hypertension, and 5 had pulmonary disease.

Hypertension was diagnosed according to the criterion of WHO. Patients with valvular, congenital or arrhythmic heart disease were excluded from the study.

**METHOD**

1) Hypoxemia Test

The hypoxemia test used most widely is that of Levy and co-workers, in which a mixed gas of 10% oxygen and 90% nitrogen is administered for 20 minutes through a non-rebreathing mask by connecting a tank of the mixed gas and another tank of pure oxygen to a simple and routine anesthesia machine.

Arterial oxygen saturation was checked by an ear oximeter.

**TABLE II**

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Patient Group</th>
<th></th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td></td>
<td>Mean S.D.</td>
<td>Mean S.D.</td>
<td></td>
<td>Mean S.D.</td>
<td>Mean S.D.</td>
</tr>
<tr>
<td>QRS (mV)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Frontal plane</td>
<td>1.56 ± 0.67</td>
<td>1.38 ± 0.64</td>
<td>−</td>
<td>1.48 ± 0.40</td>
<td>1.35 ± 0.42</td>
</tr>
<tr>
<td>Horizontal plane</td>
<td>1.15 ± 0.39</td>
<td>1.06 ± 0.39</td>
<td>−</td>
<td>1.28 ± 0.46</td>
<td>1.19 ± 0.48</td>
</tr>
<tr>
<td>Sagittal plane</td>
<td>1.15 ± 0.65</td>
<td>1.06 ± 0.59</td>
<td>−</td>
<td>1.11 ± 0.45</td>
<td>1.06 ± 0.47</td>
</tr>
<tr>
<td>X - lead</td>
<td>1.07 ± 0.41</td>
<td>0.96 ± 0.41</td>
<td>−</td>
<td>1.09 ± 0.44</td>
<td>0.97 ± 0.46</td>
</tr>
<tr>
<td>Y - lead</td>
<td>1.05 ± 0.68</td>
<td>0.93 ± 0.62</td>
<td>−</td>
<td>0.88 ± 0.45</td>
<td>0.81 ± 0.46</td>
</tr>
<tr>
<td>Z - lead</td>
<td>−0.15 ± 0.40</td>
<td>−0.18 ± 0.41</td>
<td>−</td>
<td>−0.28 ± 0.68</td>
<td>−0.33 ± 0.62</td>
</tr>
<tr>
<td>Space</td>
<td>1.62 ± 0.66</td>
<td>1.46 ± 0.63</td>
<td>−</td>
<td>1.62 ± 0.46</td>
<td>1.50 ± 0.49</td>
</tr>
</tbody>
</table>

Mean and Standard Deviation

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were recorded in the frontal, horizontal and left sagittal planes. Chest electrodes were attached on the level of the fifth intercostal space at the left sternal border. The three-planar projections of the VCG were photographed as loops through the oscilloscope screen with a 35 mm camera.

Four channels of a six-channel electromagnetic oscillograph recorder were employed for simultaneous recording of three scalar electrocardiograms and a pneumogram at paper speed of 25 and 100 mm/sec. An impedance pickup was attached on the chest wall for recording the pneumogram which showed respiratory rate and depth. These recordings were made with the patients in the supine position. VCG was taken before and every 5 minutes after the beginning of the hypoxemia test, for 20 minutes.

A block diagram of the data processing system is shown in Fig. 1.

The following 16 parameters were analysed:

1) Voltage of maximum QRS vector in each plane.

2) Voltage of maximum T vector in each plane.

3) QRS-T angle, namely, absolute value of the angle between maximum QRS vector and maximum T vector in each plane.

4) T/QRS ratio, namely, voltage ratio of the maximum T vector to the maximum QRS vector in each plane.

5) Voltage of QRS in each of X-, Y- and Z-leads.

QRS voltage consists of the sum of Q, Y- and S voltages.

6) Voltage of T in each of X-, Y- and Z-leads.

If the T wave was diphasic, the voltage consisted of the sum of each deflection.

7) Voltage of J (corresponding to ST junction in ECG) in each of X-, Y- and Z-leads.

8) Voltage of JT/2, namely, the voltage at the midpoint between J and the top of T in each of X-, Y- and Z-leads, as shown in Fig. 2.

9) Interval of QTc, namely, corrected QT interval in the clearest one among X-, Y- and Z-leads, which was calculated by dividing the estimated QT interval with V/RR.

The following 4 spatial data were calculated using the scalar waves:

10) Voltage of spatial QRS vector.

11) Voltage of spatial T vector.

12) Spatial QRS-T angle.

13) Spatial T/QRS ratio.

Fig. 3. The reference frame and each quadrant (Q) in this study.

saturation, immediately after pure O₂ was administered through a non-rebreathing mask for 3 minutes. And after removing the mask, normal atmospheric breathing was carried out for at least 5 minutes to stabilize the grade of arterial oxygen saturation.

Thus it was possible to inspect and record accurately the effect of hypoxemia test which followed.

The examinee then received a mixture of 10% oxygen and 90% nitrogen from the hypoxemia circuit for 20 minutes. But the hypoxemia test was interrupted if the examinee showed any significant distress, any evident changes in VCG, or a falling of arterial oxygen saturation below 50%.

2) Vectorcardiogram and Pneumogram

Using a three-channel vectorcardiograph, vectorcardiograms (VCGs) of Frank lead system

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TABLE III  EFFECTS OF HYPOXEMIA TEST ON VOLTAGES OF MAXIMUM T VECTOR IN EACH
PLANE, OF T IN EACH SAAGAR WAVE AND OF SPATIAL T VECTOR

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Patient Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>T (mV)</td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>Frontal plane</td>
<td>0.52 ± 0.28</td>
<td>0.39 ± 0.23</td>
</tr>
<tr>
<td>Horizontal plane</td>
<td>0.48 ± 0.20</td>
<td>0.39 ± 0.20</td>
</tr>
<tr>
<td>Sagittal plane</td>
<td>0.48 ± 0.27</td>
<td>0.37 ± 0.24</td>
</tr>
<tr>
<td>X - lead</td>
<td>0.35 ± 0.19</td>
<td>0.27 ± 0.15</td>
</tr>
<tr>
<td>Y - lead</td>
<td>0.35 ± 0.25</td>
<td>0.26 ± 0.20</td>
</tr>
<tr>
<td>Z - lead</td>
<td>0.27 ± 0.20</td>
<td>0.23 ± 0.20</td>
</tr>
<tr>
<td>Space</td>
<td>0.61 ± 0.29</td>
<td>0.48 ± 0.25</td>
</tr>
</tbody>
</table>

-Mean and Standard Deviation-

TABLE IV  EFFECTS OF HYPOXEMIA TEST ON VOLTAGES OF J AND JT/2 AND ON QTc INTERVAL

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Patient Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>J, JT/2, QTc</td>
<td>Mean</td>
<td>S.D.</td>
</tr>
<tr>
<td>X - lead</td>
<td>0.01 ± 0.04</td>
<td>0.01 ± 0.03</td>
</tr>
<tr>
<td>J (mV)</td>
<td>0.02 ± 0.07</td>
<td>0.01 ± 0.07</td>
</tr>
<tr>
<td>Z - lead</td>
<td>0.01 ± 0.05</td>
<td>0.01 ± 0.09</td>
</tr>
<tr>
<td>JT/2 (mV)</td>
<td>0.06 ± 0.06</td>
<td>0.04 ± 0.05</td>
</tr>
<tr>
<td>QTc (sec)</td>
<td>0.43 ± 0.04</td>
<td>0.43 ± 0.03</td>
</tr>
</tbody>
</table>

-Mean and Standard Deviation-

The following 3 parameters were estimated and referred for study.

(14) Heart rate.
(15) Respiratory rate.
(16) Arterial oxygen saturation.

The reference frame was named, as shown in Fig. 3, according to the recommendation of the American Heart Association in the following way: Left, inferior and anterior sides were named positive (+) in each axis. The frame of reference for angular measurement of each planar projection was expressed by \(0^\circ \sim +180^\circ\) in a clockwise direction and by \(0^\circ \sim -180^\circ\) in a counterclockwise direction. The 1st, 2nd, 3rd and 4th quadrants lay every ninety degrees clockwise.

The polarity in X-, Y- and Z-leads was regarded as positive in the upper part of the baseline, as shown in Fig. 2.

RESULTS

In the following tables, each numeral shows the mean and standard deviation, and the significant difference between the two values before and after the hypoxemia test are shown by 'P' value.

1) QRS voltage (Shown in Table II)

In the control group, no significant change

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TABLE V  EFFECTS OF HYPOXEMIA TEST ON QRS-T ANGLE

<table>
<thead>
<tr>
<th></th>
<th>Control Group</th>
<th>Patient Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>QRS-T angle</td>
<td>Mean ± S.D.</td>
<td>Mean ± S.D.</td>
</tr>
<tr>
<td>Frontal plane</td>
<td>12.2 ± 12.0</td>
<td>14.6 ± 11.9</td>
</tr>
<tr>
<td>Horizontal plane</td>
<td>48.9 ± 30.3</td>
<td>53.0 ± 32.8</td>
</tr>
<tr>
<td>Sagittal plane</td>
<td>50.4 ± 32.5</td>
<td>57.3 ± 34.2</td>
</tr>
<tr>
<td>Space</td>
<td>39.4 ± 22.6</td>
<td>45.1 ± 24.4</td>
</tr>
</tbody>
</table>

—Mean and Standard Deviation—

TABLE VI  DIRECTIONS OF MAXIMUM QRS VECTOR AND MAXIMUM T VECTOR IN EACH PLANE BEFORE THE HYPOXEMIA TEST

<table>
<thead>
<tr>
<th>Direction of Maximum QRS Vector (Degree)</th>
<th>Plane</th>
<th>Mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>42.4 ± 25.4</td>
<td></td>
</tr>
<tr>
<td>Horizontal</td>
<td>—18.3 ± 14.5</td>
<td></td>
</tr>
<tr>
<td>Sagittal</td>
<td>32.6 ± 49.1</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Direction of Maximum T Vector (Degree)</th>
<th>Plane</th>
<th>Mean ± S.D.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frontal</td>
<td>46.5 ± 15.6</td>
<td></td>
</tr>
<tr>
<td>Horizontal</td>
<td>35.8 ± 25.7</td>
<td></td>
</tr>
<tr>
<td>Sagittal</td>
<td>123.5 ± 48.5</td>
<td></td>
</tr>
</tbody>
</table>

—Control Group—

TABLE VII  EFFECT OF HYPOXEMIA TEST ON T/QRS RATIO

<table>
<thead>
<tr>
<th>T/QRS ratio</th>
<th>Control Group</th>
<th>Patient Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td></td>
<td>Mean ± S.D.</td>
<td>Mean ± S.D.</td>
</tr>
<tr>
<td>Frontal plane</td>
<td>0.35 ± 0.17</td>
<td>0.30 ± 0.15</td>
</tr>
<tr>
<td>Horizontal plane</td>
<td>0.43 ± 0.15</td>
<td>0.38 ± 0.17</td>
</tr>
<tr>
<td>Sagittal plane</td>
<td>0.46 ± 0.30</td>
<td>0.42 ± 0.29</td>
</tr>
<tr>
<td>Space</td>
<td>0.39 ± 0.15</td>
<td>0.34 ± 0.15</td>
</tr>
</tbody>
</table>

—Mean and Standard Deviation—

was provoked by the hypoxemia test in the voltages of maximum QRS vectors in each plane, of the QRS in each scalar wave and of the spatial QRS vector.

In the patient group, the voltage of maximum QRS vector decreased slightly in frontal plane and the voltage of QRS in X-lead did also after the hypoxemia test.

2) T voltage (Shown in Table III)

In the control group, all the T parameters except T voltage in the scalar wave of Z-lead decreased significantly after the hypoxemia test. Among them, the spatial T vector decreased the most remarkably, form 0.61 mV to 0.48 mV.

In the patient group, the results were the same as in the control group. The decrease was the
most remarkable in the following 3 parameters:

a) Maximum T vector in frontal plane decreased form 0.42 mV to 0.33 mV.

b) Maximum T vector in sagittal plane decreased from 0.40 mV to 0.31 mV.

c) Spatial T vector decreased from 0.52 mV to 0.41 mV.

3) J voltage (Shown in Table IV)

In both groups, J voltage decreased slightly in Z-lead after the hypoxemia test, though it did not decrease in other leads.

4) JT/2 voltage (Shown in Table IV)

In the control group, voltage of JT/2 decreased slightly in X-lead after the hypoxemia test, though it did not decrease in other leads.

In the patient group, voltage of JT/2 decreased distinctly in Y-lead and slightly in X- and Z-leads after the hypoxemia test.

5) QTc interval (Shown in Table IV)

In both groups, there was no significant difference between QTc interval before the hypoxemia test and that after the test.

6) QRS-T angle (Shown in Table V)

In the control group, there was no significant difference between QRS-T angle before the hypoxemia test and that after the test in each plane and space.

In the patient group, the increase of QRS-T angle was provoked by hypoxemia, and was distinct in the sagittal plane and space, and slight in the frontal and horizontal planes.

7) Directions of maximum QRS vector and maximum T vector in control group

These directions were estimated in VCGs in 3 planes recorded before the hypoxemia test in control group. The results, as shown in Table VI, are in good agreement with those of other investigators. Thus, it is concluded that the control examinees are not extraordinary but common.

8) Displacement of T vector by the hypoxemia test

On the basis of the location of T vector before the hypoxemia test and the direction of
displacement in T vector by the hypoxemia test, the displacement of T vector in horizontal plane was classified into the following 6 types.

(1) T vector in the 1st quadrant shifted clockwise.
This type included 22 control cases and 16 patients. In some of the patients, T vector shifted from the 1st quadrant to the 2nd quadrant.
(2) T vector in the 1st quadrant shifted counterclockwise.
This type included 16 control cases and 14 patients. Two of this type showed remarkable increase of QRS-T angle and 3 showed decrease of the angle.
(3) T vector in the 2nd quadrant shifted clockwise.
This type included 11 patients, and none of control cases.
(4) T vector in the 3rd quadrant shifted counterclockwise.
This type included 3 patients, and none of control cases.
(5) T vector in the 4th quadrant shifted clockwise.
This type included one control case and one patient. In the latter case T vector shifted from the 4th quadrant to the 2nd quadrant.
(6) T vector in the 4th quadrant shifted counterclockwise.
This type included 4 control cases and 5 patients. In 3 of the 5 patients, T vector shifted from the 4th quadrant to the 3rd quadrant and QRS-T angle increased remarkably. Four control cases and 7 patients besides the above mentioned persons had no evident displacement of T vector.

9) T/QRS ratio
As shown in Table VII, T/QRS ratio decreased slightly in horizontal plane and space after the hypoxemia test in the control group, and decreased distinctly in space, and only slightly in frontal and horizontal planes in the patient group.

10) Heart rate
As shown in Table VIII, heart rate increased remarkably from 66.64/min to 81.21/min in the control group and from 73.70/min to 84.46/min in the patient group by the hypoxemia test.

11) Respiratory rate and amplitude of pneumogram
As shown in Table VIII, there was no significant change by the hypoxemia test in respiratory rate in both groups. But the amplitude of pneumogram increased as arterial oxygen saturation decreased, in almost all persons.

12) Arterial oxygen saturation
As shown in Table VIII, arterial oxygen saturation decreased remarkably at the end of the hypoxemia test from 95.23% to 64.55% in the control group and from 95.92% to 64.40% in the patient group by the hypoxemia test.

Arterial oxygen saturation monitored during the course of the experiment was shown in Fig. 4. The saturation decreased distinctly from 5 minutes to 15 minutes after the beginning of hypoxemia loading, and held an almost fixed value from 15 minutes to 20 minutes.

SUMMARY AND DISCUSSION

The hypoxemia test is considered to be contraindicated for coronary insufficiency, as is the exercise test. The author excluded congenital and rheumatic heart diseases, pregnancy, myxedema, epilepsy and anemia from his examinees, such diseases being regarded by Stewart et al. as contraindicating the hypoxemia test.

A few cases may complain of such central vagal stimulation symptoms as bradycardia, hypotension, sweating, paleness and unconsciousness in the hypoxemia test. Its incidence was 5% according to Burnett et al. and Biörck. Nylin et al. reported that such vasovagal attacks took place in only one out of 1,130 hypoxemia tests. Several of the author's examinees complained of slight dull headache and/or lassitude, but none of them complained of distinct vasovagal attacks and of precordial pain.

Arterial oxygen saturation was continuously observed throughout the experiment, which was interrupted halfway when any risk was suggested in the hypoxemia test. This may be the reason why there were no distinct complications in the experiment.

The vector loop, namely Lissajous' figure of VCG, is suitable for observing gross qualitative changes of VCG, but unsuitable for doing quantitative changes. Also the scalar wave of VCG is useful for its qualitative interpretation. And so, both Lissajous' figure and the scalar wave were simultaneously recorded in the experiment.

The voltages, QRS-T angle and T/QRS ratio were estimated on QRS loop and T loop in frontal, horizontal and left sagittal planes, and the voltages of QRS, T, J and JT/2, and QTc were estimated on X, Y, Z scalar waves. Also the spatial QRS loop and T loop were calculated from the above values of scalar waves.
1) QRS voltage
Slight grade changes of QRS voltage were provoked by hypoxemia in the patient group in frontal plane and X scalar wave, though there was little change in the control group. Likewise, Levy et al. report a decrease under 0.4 mV of QRS voltage provoked by hypoxemia. They do not discuss the meaning of the QRS voltage change.

There were cases in which respiration became deep by the hypoxemia test, resulting in the amplitude of the scalar wave being a little bigger in the inspiratory phase than in the expiratory phase. In order to avoid the respiratory effect on QRS amplitude, a pneumogram was recorded simultaneously, and the mean of the 3-phase QRS waves (inspiratory, expiratory and middle phases) was calculated and regarded as representative. The same was the case with T, J or JT/2.

2) T voltage
T voltage was decreased by the hypoxemia test in both patient group and control group. In the former even the inverted T was observed, though not in the latter. And so, the T inversion has a clinical meaning.

The decrease or inversion of T voltage by hypoxemia has been reported by Levy et al., and many investigators but in all these reports there was a deficiency, being failure to demonstrate electromotive forces of the heart faithfully because their electrocardiograms were recorded mainly by limb leads.

The author's result is more reliable because he kept a concept of vector in mind by using vectorcardiogram representing electromotive forces of the heart as vector quantities.

3) J voltage
Fall of J voltage was provoked by the hypoxemia, though very small in both patient group and control group. Many investigators regard a fall of J voltage over 0.1 mV and a negative inversion of T as abnormal and as of the same clinical significance. But, according to the author's result, the J change had little clinical significance.

4) JT/2 voltage
JT/2 voltage was estimated for reference in evaluating the fall of J voltage. JT/2 voltage was unchanged by the hypoxemia in Y- and Z-lead ECGs in the control group, and decreased in the patient group. JT/2 voltage may be better than J voltage as an indicator, and may be dependent rather on T voltage.

A further study is to be performed.

5) QTc of ECG
Though QTc was prolonged by hypoxemia test in patients with angina in the experiment of Roehn et al., it was not done in the author's experiment.

6) QRS-T angle
QRS-T angle was apparently increased by hypoxemia in frontal, horizontal and left sagittal planes, and in space in patient group, though it was only apt to be increased in control group. Therefore, QRS-T angle is one of the most reliable clinical indicators. In 1954 Gürler et al. reported that QRS-T angle was increased by hypoxemia in the Levy-positives.

QRS voltages in X-, Y-, Z-lead ECGs were scarcely changed by hypoxemia. Only a few cases showed some change, but it was of almost the same degree among the 3 ECGs; and so, the direction of QRS vector was to be determined by the 3 voltages of QRS was not displaced as a whole.

The direction of T vector was already abnormal in the patient group before the hypoxemia test. Also the T vector was greatly changed by hypoxemia, and its change was classified into 6 patterns.

The change of T voltage provoked by hypoxemia was not the same among the 3 ECGs, contrary to QRS voltage; the displacement arose in the direction of T vector.

It is concluded that the change of QRS-T angle by hypoxemia is caused by the displacement of the direction of T vector because the direction of QRS vector is not displaced.

7) T/QRS ratio
It seems to be reasonable to evaluate the change of T vector in reference to the change of QRS vector, namely as its relative quantity. T/QRS ratio had a tendency to be decreased by hypoxemia in either patient group or control group, and so it was not useful for demonstrating the hypoxemia effect.

8) Physiological changes other than VCG
In order to understand the grade of hypoxemia, such indicators as pulse rate, respiratory rate and arterial oxygen saturation were observed throughout the experiment, with the same results as reported by Simonsen and Stewart et al. Therefore, the author's experiment was regarded as correctly performed by Levy's original method.

CONCLUSIONS

The hypoxemia test as a loading method is
useful for clinical practice of vectorcardiography. QRS-T angle, T vector and QRS vector, especially the first one, are good keys for interpreting the effect of hypoxemia on the heart.

The scalar electrocardiogram is more helpful for the quantitative analysis of hypoxemia effect than the vector loop is.

The hypoxemia test is performed exactly and safely by steady observation of arterial oxygen saturation and electrocardiogram.

REFERENCES