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The Diagnosis of Slight Changes in the Electrocardiogram

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SOME ELECTROCARDIOGRAPHIC findings are considered to be abnormal, but to represent no serious disorder. Notching or slurring of the QRS deflection, the deep S wave in the left precordial leads, the ST elevation in the right precordial leads, shift of the transitional zone, the inverted T wave in the lead III alone, the augmentation of the height of the T wave, the transient change in the T wave, the abnormal QT interval, depression of the ST_r segment, the low voltage of the QRS deflection, the small deflections immediately preceding or following the QRS deflection, etc. are included in this category of the electrocardiographic findings. They are observed often in the apparently healthy persons and perplex us in making diagnosis.

In this paper the following four subjects were taken up and discussed. The electrocardiographs employed were the Sanborn Visocardiette Model 52, the Fukuda Cardiorite RS-13 and the Chiba Medic Cardiograph CF 6000, 6100 and 8000.

1. THE ST ELEVATION IN THE CHEST LEADS, ESPECIALLY IN THE RIGHT PRECORDIAL LEADS

Not infrequently one observes the ST elevation in V₁ or V₂ leads in the healthy subjects, and especially when this finding is associated with a small R deflection, it is often misjudged as representing anteroseptal infarction.

According to the concept of the ventricular gradient¹⁾, the vector T is the sum of the vector of the ventricular gradient

(vector G) and the vector, which is oriented opposite to the vector QRS. In this paper the latter vector will be called as the vector E. Both process G and E are considered to start before the end of the S wave. Since the angle between these two vectors is not so large during the period of the ST segment, their projection cancels each other and the ST segment lies on the zeroline, showing no ST deviation. If the heart is located in some peculiar position, however, the projection of the vectors on some lead line may diverge and the ST segment may deviate from the zeroline.

From this viewpoint, the relation of the ST elevation in the precordial leads to the heart position was investigated in nearly one thousand apparently healthy cases. The position of the heart was judged principally with the Criteria of Ashman²⁾, employing the QRS configuration of the standard limb leads.

Fig. 1. shows the cardiac position in the cases showing the ST elevation in the precordial leads V₂ through V₅ above 0.1mV. 69.7 % of the cases had the forward displacement of the apex, while very few had the back-ward displacement. Especially in the cases showing the ST elevation in lead V₄ above 0.15 mV., no backward displacement of the apex was observed. Of total cases, 35 % showed the forward displacement. On the contrary, in the cases showing the ST elevation in the right precordial leads alone, the backward displacement of the apex was observed relatively frequently,

Fig. 1. The cardiac position of the cases showing the ST elevation in the leads V_2 through V_5 .

vert.: vertical position; intermed.: intermediate position; hor.: horizontal position; counterclock.: counterclockwise rotation; clockwise: clockwise rotation; apex intermed.: apex intermediate position.

as indicated in Fig. 2.

The mechanism of such ST elevation in the precordial leads is interpreted with the concept of ventricular gradient as following. The forward downward rotation of the axis of the ventricular gradient in the apex forward position must result in a greater influence on lead V_4 than in the normal position and this probably causes the ST segment to be elevated.

On the other hand, the influence of G on lead V_2 is diminished in this position. Since, however, lead V_2 faces the electro-

motive force at the opposite side, the ST segment is elevated also in this lead. Relatively frequent incidence of the backward displacement of the apex in the cases showing the ST elevation in the right precordial leads alone is explained by the fact that the direction of the vector QRS or E points to the neighbourhood of the lead point of V_2 and overbalances the effect of G, so that the ST segment is elevated.

If the change in the heart position is one of the causes of the ST elevation, the transitional zones of the QRS and T waves may have some relation to the ST elevation. As shown in Fig. 3, the ST elevation was observed relatively frequently when the transitional zone of the QRS deflection is in lead V_2 and that of the T wave in lead V_3R .

In conclusion, it is probable that the changes in the cardiac position cause the ST elevation in the apparently healthy subjects. It is needless to say, however, that other various factors affect the ST segment.

Fig. 2. The cardiac position in the cases showing the ST-elevation in the right precordial leads alone.

$V_1, V_2, V_3 > 0.1$ mv., one of which > 0.2 mV. $V_3, V_4, V_5 < 0.1$ mV.

Fig. 3. The location of the transitional zones in the cases showing the ST elevation in the lead V_2 .

2. LOW VOLTAGE OF THE QRS DEFLECTION IN THE APPARENTLY HEALTHY SUBJECTS

The physico-mathematical analysis of the potential field of the limited volume conductor shows that two potential maxima, positive and negative, exist asymmetrically on the surface of the conductor, when the electromotive force lies eccentrically and in some given direction within it. The absolute values of these potential maxima are

larger when the electromotive force lies near the surface than when it is in the vicinity of the center of the conductor. Any point on the surface has the potential value between these two maxima. In the human body, the location as well as the direction of the electromotive force change from instant to instant, as excitation wave propagates. In consequence, the shape and the value of the potential field on the body surface also change. In this situation, any

point on the body surface shows the fluctuation of the potential in some range. This range of fluctuation is assumed to be larger when the heart is located near the precordium than when it locates near the center of the thorax.

With this assumption, the relation of the voltage of the QRS deflection to the position of the heart was investigated in the same subjects as in the foregoing paragraph. The voltage was measured from the top of the R wave to the bottom of the Q or S waves.

Fig. 4. shows the results. Of the cases showing the QRS deflection lower than 0.1 mV. in the standard limb leads, 38 % showed the backward displacement of the apex and

10 % the forward displacement. The relation was reverse in the total cases. The same finding was seen in the cases showing the QRS voltage lower than 1.5 mV. in the precordial leads. Forward displacement of the apex was observed only in 4.4 %.

Further investigation was made concerning the cases showing the low voltage both in the standard limb leads (lower than 0.7 mV.) and in the precordial leads (low than 2.0 mV.), including various patients (excluding the myocardial infarction, the bundle branch block and the pericarditis). No cases were judged as having their heart in apex forward position.

Although the heart with forward displaced apex is not always located near the

Fig. 4. The cardiac position in the cases showing the low voltage
 upper column: Total cases
 middle column: The cases showing the low voltage in the limb leads (smaller than 1.0 mV.)
 lower column: The cases showing the low voltage in the chest leads (smaller than 1.5 mV.)

precordial wall and the heart with the backward displaced apex does not always lie in the vicinity of the center of the thorax, such relations may be assumed to exist to a first approximation in no small number of cases. That this is true was verified by the observed low incidence of the forward displacement and the relatively high incidence of the backward displacement of the apex in the cases showing low QRS voltage in both standard limb and precordial leads. In other words, the low voltage both in the standard limb and the precordial leads can be attributed to the backward displacement of the whole heart.

In this connection, the meaning of the high voltage both in the limb and the precordial leads was also investigated. In the apex forward position, approximately half of the cases showing higher voltages than 4.0 mV. in the chest leads had also higher voltages than 2.0 mV. in the standard limb leads. The QRS voltage of this magnitude was not observed in any case with the backward displacement of the apex.

The high voltage of the QRS deflection is one of the characteristics of the hypertrophy pattern. Its mechanism has been interpreted in various ways. The view introduced by the author some years ago³⁾, that the approach of the apex to the precordial chest wall as a result of the ventricular hypertrophy is one of the causes of the high voltage, falls on the same line of thought.

In conclusion, the low voltage both in the limb and the precordial leads can be produced by the backward displacement of the whole heart within the thorax, although this is not the sole cause.

3. SMALL DEFLECTION FOLLOWING THE QRS DEFLECTION AND THE BOW-SHAPED RS-T JUNCTION

It is often observed that the small deflection follows the QRS deflection, as seen in leads V_5 and V_6 of the electrocardiogram taken on Feb. 17, 1960 in Fig. 5. The bow-shaped RS-T junction is also the frequently observed finding. Since these two

findings appear often in the same case and even in the same lead, they may be discussed as a single group.

As pointed out by the author previously⁴⁾, digitalis or glycine solution produced the small deflection after the QRS deflection, when they were applied on a circumscribed area of the toad's ventricular surface and the electrocardiogram was lead directly from that area. Recently, it was discovered that the small wave resembling this deflection appears transiently during the hypothermia⁵⁾.

In view of this recent development, it became necessary to re-examine the subject. No special relationship, however, was observed to exist to the age or the blood pressure. There was no high incidence of these findings in the coronary disease group. Accordingly, these findings may be concluded to have no special significance in the clinical practice. But the case shown in Fig. 5. gave suggestive evidences that these findings would not completely be neglected, because they appeared spontaneously or after exercise.

In conclusion, the clinical significance of the small deflection after the QRS deflection is uncertain, although it can be produced in some experimental condition.

4. TRANSIENT CHANGES IN THE T WAVE, ESPECIALLY IN CASES WITH HYPOTENSION

It is known that the T wave is lowered or inverted in the upright position in some patients, especially in cases with hypotension or neurocirculatory asthenia. Various hypotheses have been presented to interpret this finding. The most interesting is Goldberger's suggestion⁶⁾ that the lowering of the serum potassium level, resulting from the increase of the sympathetic tone in the upright position, is the cause of the T wave change. Nevertheless, he did not prove it experimentally.

Fig. 6. shows my observation. The potassium level was not always reduced, when the T wave became lower. But a significant, though not high, correlation was demonstrated between the potassium level and the height of the T wave in the upright position, as indicated in Fig. 7. In

Fig. 5. A case in which the small deflection following the QRS appears spontaneously or after exercise.

this respect, the suggestion of Goldberger seems to have some validity.

Another interesting fact was observed concerning the relation of the hypotension to the potassium level. As indicated in Fig. 8, some of the hypotensive patients have the potassium level below 4.0 mEq/L. This fact was already pointed out by Kato, et al⁷⁾. Such hypotensive patients with low potassium level often have the ST-T changes even in the recumbent position. Fig. 9. shows that the potassium level is significantly lower in the patients with ST-T changes in the recumbent position than in those without ST-T changes.

It is likely that some relation exists

between the hypotension, the low potassium level and the ST-T changes. The low potassium level per se, however, may not be the direct cause of the ST-T changes, because its level ranged from 3.4 to 3.8 mEq/L, a range of level where electrocardiographic changes are usually not produced. The diminution of the coronary blood flow due to hypotension cannot be accepted as the cause of the ST-T changes, too, because, as indicated in the animal experiments in our previous report⁸⁾, the coronary blood flow does not fall in proportion with the lowering of the blood pressure and the ST-T changes appear only when the coronary blood flow falls below the level of one third of

Fig. 6. Changes in the height of the T wave in lead II and the serum potassium level before and after standing.

Fig. 7. The relationship of the height of the T wave to the serum potassium level in the upright position.

Fig. 8. Serum potassium level in various diseases.

Fig. 9. The ST-T changes and the serum potassium level in the hypotensive patients.

the normal.

In conclusion, among the cases whose conditions are diagnosed as essential hypotension, cardiac neurosis or neurocirculatory asthenia, etc. there are certain cases who have a relatively low potassium level and the ST-T changes even in the recumbent position, in addition to having a low blood pressure. It seems adequate to consider these findings in combination as a specific syndrome.

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