ELECTROCARDIOGRAPHIC CHANGES DURING BRIEF ATTACKS OF ANGINA PECTORIS

BY

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Large numbers of electrocardiograms of attacks of cardiac infarction have been published, but there are relatively few of the brief attacks of angina pectoris. This is due, no doubt, to the infrequent opportunities which arise, but the records are of importance in throwing light on the mechanism of production of the pain.

Levine, Ernstene, and Jacobson¹ published a series of electrocardiograms taken during attacks of angina produced by injections of epinephrine. They did not find any specific changes, the only modification being a differ-

knowledge of the effect of the drug itself on the electrocardiograms during the attack.

CASE RECORDS

Case 1.—A female, aged 62, first seen in June, 1932, when she complained of precordial pain radiating to the left arm. She had had the pain for two years, but it had become worse recently. It was induced by exertion or excitement, and was always relieved by rest. The heart was not enlarged, and the sounds were normal. Blood pressure: 160/80. Prolonged rest benefited her, but in March, 1933, she became gradually worse, and during an examination of the heart she had an attack which she described as being quite typical. The pain was relieved by ten minutes' rest, and with adequate rest she has since remained fairly well. The electrocardiograms are reproduced in Fig. 1. (A) was taken immediately after the commencement of the attack, and (B) some time later, when the patient was free from pain. (A) shows a depression of the RT interval in Leads 1 and 2, and an elevation in Lead 3. The amplitude of QRS is slightly increased. (All electrocardiograms are standardized 0.2 sec. $\times$ 0.2 cm.)

ence in size of the waves without any change of direction. These attacks were artificially produced, and it would be unwise to apply their conclusions to a "naturally" produced attack. Parkinson and Bedford² summarized the literature to date, and reported five cases of brief attacks of angina in which electrocardiograms had been taken during and after the paroxysm. There was a depression of the RT interval and a diminution or an inversion in T waves in one or more leads. They did not suggest that these changes were present in all cases. Wood and Wolferth³ reported thirty cases, fifteen of which showed changes in the T wave during the attack; seven of these were severe and eight were mild attacks. The changes consisted of an alteration in height of the T wave, or a change from positive to negative, and the appearance of a depression in the RT interval. In the electrocardiograms reproduced Charts 8 and 12 showed the depression of the RT interval. The authors mentioned this as occurring in five other cases, but no charts were given. Fifteen cases showed no change. They also completed a series of animal experiments, and their conclusions were that the majority of the attacks of angina were associated with a localized circulatory disturbance in the heart. The possibility of other mechanism was not definitely ruled out.

The records of four cases are given below; all the patients had an attack in the consulting room. The attacks were probably induced by excitement, as there was no preceding exertion, and were relieved by rest, the longest time for complete cessation of pain being twenty minutes. Amyl nitrite was not used, as there is no

Case 2.—A female, aged 72, first seen in October, 1931, when she complained of substernal pain radiating in both arms, but worse on the left side; duration four to five months. The pain was always induced by exertion or excitement, and relieved by rest. There was some dyspnoea at night, which was relieved by sitting up. The heart was slightly enlarged, and was half an inch beyond the mid-clavicular line. There were no murmurs; aortic second sound increased. Blood pressure: 250/140. While the examination was in progress the patient complained that the pain was beginning. It developed into a typical attack, which was relieved by ten minutes' rest. Following a prolonged rest she has since remained well. The electrocardiograms are reproduced in Fig. 2. (A) was taken at the height of the attack, and (B) half an hour later, when the patient was free from pain. (A) shows a depression of the RT interval in Leads 2 and 3. The amplitude of QRS is increased. A further electrocardiogram taken in November, 1931, was identical with (B).

Case 3.—A female, aged 53, first seen in March, 1932, when she complained of a dull, aching precordial pain radiating to the left arm, induced by exertion and relieved by rest; duration eighteen months. Less exertion was required to produce the pain than a year previously. The left border of the heart was one inch beyond the mid-clavicular line; there were no murmurs. Blood pressure: 175/95. During the examination the patient had a typical attack, which was relieved by rest in a very short time (about five minutes). A prolonged rest has given her freedom from pain. The electrocardiograms are reproduced in Fig. 3. (A) was taken during the attack, and (B) following it. (A) shows a depression of the RT interval in Leads 1 and 2. The amplitude of QRS is slightly increased. (B) shows no depression in Lead 1, but a slight depression in Lead 2.

Case 4.—A man, aged 62, first seen in January, 1932; he complained of substernal pain radiating to both arms, of six years' duration. The pain was much more easily produced in the last year; moderate exercise, such as light digging, brought it on, and it lasted for two to three minutes. It was always relieved by rest. The left border of the heart was in the mid-clavicular line; there were no murmurs. Blood pressure: 180/90. During the examination the patient had an attack, which he described as being rather worse than usual. It was relieved by twenty minutes' rest. He now keeps well with adequate rest. The electrocardiograms are reproduced in Fig. 4. (A) was taken during the attack and (B) following it. (A) shows a depression of the RT interval in all leads, particularly in Leads 2 and 3.

DISCUSSION

In these four cases definite changes in the ventricular complexes of the electrocardiograms are demonstrated. These changes resemble those produced by coronary thrombosis. It is agreed that they are not always present, due no doubt to "silent areas" in the myocardium, changes in which do not affect the electrocardiogram. The positive evidence adduced in the above cases and in those previously published points very strongly towards the coronary origin of anginal pain.

REFERENCES

- ¹ Levine, Ernestene, and Jacobson: *Arch. Int. Med.*, February, 1930, p. 191.
- ² Parkinson and Bedford: *Lancet*, 1931, i, 15.
- ³ Wood and Wollerth: *Arch. Int. Med.*, March, 1931, p. 339.

The Italian Fascist National Federation Against Tuberculosis has again placed at the disposal of the International Union Against Tuberculosis six scholarships at the Benito Mussolini Institute in Rome. The conditions submitted are as follows: These competitive scholarships, of a value of 6,000 lire respectively, plus board and lodging, are intended to enable foreign medical practitioners to stay at the Benito Mussolini Institute in Rome for the purpose of following a course of studies. This stage of eight months will correspond with the academic year (from November 15th to July 15th), including the usual holiday periods. The scholarships will preferably be awarded to young physicians already familiar with tuberculosis problems who wish to improve their knowledge of this branch of medicine. The kind of work undertaken at the Institute will be subject to an agreement between the director and the candidate. The publication expenses resulting from this work may be defrayed partly or entirely by the Institute. The scholarships will be awarded at the summer session of the Executive Committee of the International Union, which meets this September. No candidature will be considered unless it has been forwarded by an association belonging to the International Union. The body representing Great Britain and Northern Ireland is the National Association for the Prevention of Tuberculosis, Tavistock House North, Tavistock Square, London, W.C.1, to whom applications should be sent not later than July 1st, giving particulars as to age, qualifications, and professional experience.

MAGNESIUM DEFICIENCY IN THE RAT

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The intensive study of calcium metabolism, for which the investigations of rickets and tetany carried out in recent years have been chiefly responsible, has not stimulated significantly inquiry into the part played in the organism by magnesium, which so closely resembles calcium in its chemical properties. Whereas the concentration of magnesium in serum is of the order of 2 mg. per 100 c.cm. as compared with 10 mg. of calcium, its concentration in muscle, which accounts for so large a part of the body weight, is four times as great as that of calcium—that is, 20 mg. as compared with 5 mg. The distribution of the two constituents in the tissues suggests that magnesium plays a part in physiological processes not less important than that played by calcium. Nevertheless, it is only comparatively recently that magnesium has been shown to be necessary to life.

Leroy¹ found that when mice were fed on a diet containing 1.03 mg. of magnesium in 100 grams, death occurred in twenty-four to thirty-five days, and his work has been considerably amplified by Kruse, Orent, and McCollum,² who, using a diet which contained only 0.18 mg. of magnesium in 100 grams, have studied the symptoms and some of the biochemical changes which result when rats and puppies are deprived of magnesium. Cramer,³ by placing young rats on a diet in which the amount of magnesium was reduced to 0.632 mg. per 100 grams, showed that degenerative changes occur in the kidneys as a result of magnesium deficiency. I have been engaged on a biochemical and pathological study of magnesium deprivation, and have utilized for this purpose a small group of rats. The findings are of interest in that while the convulsive phenomena described by Kruse, Orent, and McCollum have been confirmed, the cutaneous manifestations, which are regarded by these workers as one of the distinctive features of magnesium deprivation, have been entirely absent. Moreover, histological examination of the organs has revealed marked pathological changes in the liver, as well as changes in the kidneys somewhat similar to those reported by Cramer.

EXPERIMENTAL

A diet composed of a mixture of casein, starch, butter-fat, vitamins, and salts, freed from magnesium by the methods employed by Kruse, Orent, and McCollum, was given to eight young black-and-white rats weighing from 65 to 107 grams. Four other rats, which served as controls, received the same diet with the addition of magnesium sulphate. The animals took the diet well and increased in weight for a short period. Later the weights of both test and control rats tended to remain stationary or to fall slightly. On the twenty-second day of the experiment Rat 1, which had been quiet for some days, was found dead in its cage, and two days later Rat 3 was found in a moribund condition. On the twenty-seventh day the first convulsions were observed in Rats 7 and 8, which had previously appeared entirely normal. After going round in circles for a short period Rat 8 dived under its feeding dish and went into generalized convulsions, a tonic being followed by a clonic stage. A second slighter convulsion occurred after the first. The convulsive attack of Rat 7 was milder, but otherwise similar. Both animals recovered completely