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SOME ELECTROCARDIOGRAPHIC PROBLEMS.

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THE electrocardiograph is now considered to be an important accessory in the investigation of cardiovascular conditions. As to the interpretation of the various waves of the electrocardiogram, our knowledge has gradually increased, so that we are now able to give an accurate opinion on many problems of cardio-analysis whose diagnosis formerly could only be surmised on clinical grounds.

The scientific researches of Sir Thomas Lewis in this field have done more than anything else to place our knowledge on a surer foundation. From the American side the work of men such as Pardee and Willius has done much to correlate laboratory findings with the clinical picture.

Nowadays, most of the salient features of the electrocardiogram record can be interpreted easily, but I am of the opinion that the majority of workers are inclined to dissociate the clinical from the electrocardiographic findings, and it is with the object of bridging this gap that I am writing this paper.

Mitral stenosis causes increased work on the left auricle, resulting in hypertrophy

of the muscle. This is shown by enlargement of the P wave. As the degree of stenosis increases there is often asynchronism of the contraction of the auricles. This is shown electrocardiographically by a bifid or flat topped P wave, whose amplitude is smaller than it was prior to the occurrence of asynchronism.

In a well-developed mitral stenosis with regular rhythm, a small P wave suggests auricular fatigue and foreshadows the occurrence of auricular fibrillation.

Mitral stenosis also causes increased work on the pulmonary circuit and on the right heart. The electrocardiogram, therefore, will tend to show a right-sided preponderance. If the preponderance is absent or left-sided we may expect to find one of the following associated conditions :—

- (1) Mitral regurgitation.
- (2) Aortic regurgitation.
- (3) Adherent pericardium.
- (4) Hypertropia.

Mitral regurgitation causes enlargement of the left ventricle and increased work on the right heart. If the lesion is well compensated there will be relatively more hypertrophy than dilatation of the muscle, and the electrocardiogram will show a left-sided preponderance. In time, as compensation gets less, dilatation of the left ventricle will increase at the expense of its hypertrophy. The effect on the right ventricle will also become more marked, and this will result in hypertrophy of that chamber. The preponderance will now be right-sided.

Aortic regurgitation causes enlargement of the left ventricle. Until the late stages hypertrophy is relatively greater than the dilatation; one would expect therefore a left-sided preponderance. If preponderance is absent or right-sided the condition cannot be a simple case of aortic regurgitation. One of the following associated conditions must be looked for :—

- (1) Mitral stenosis.
- (2) Double mitral lesion with right heart load.

(3) Pulmonary congestion, e.g., chronic bronchitis, pulmonary atheroma.

(4) Failing left ventricular muscle, e.g., myocardial degeneration, cardiovascular syphilis.

Premature contractions are of frequent occurrence and may arise from foci in the auricles, ventricles or from the auriculo-ventricular node itself.

Usually the impulse arises in the sinus node, which is the most irritable part of the heart, but in certain conditions other parts of the heart become hyper-irritable, and the impulse arises ectopically.

Premature or ectopic beats may be caused by conditions in the heart itself, i.e., intrinsic, or through neighbouring organs affecting some part of the heart, and inducing hyper-irritability, i.e., extrinsic.

Right ventricular premature beats are the most common variety. They may be physiological and of no moment. Such are seen not uncommonly in young persons. Right ventricular premature contractions are usually of extrinsic origin, and are caused by increased intra-abdominal pressure on the right ventricle. They may therefore be noted in the majority of women during the later months of pregnancy. Other extrinsic causes are flatulent distension, ascites and abdominal tumours.

Intrinsic right ventricular premature beats are found in the following conditions:—

- (1) Acute myocarditis, e.g., acute fevers.
- (2) Toxic myocarditis, e.g., post-influenzal, tobacco, digitalis.
- (3) Myocardial degeneration.
- (4) Failing right heart.
- (5) Heart block.

The effect of nicotine poisoning on the heart is shown by the occurrence of frequent right ventricular premature beats, e.g., tachycardia, and symptomatically by palpitation, shortness of breath and præcordial discomfort. Some hearts are much more susceptible than others.

Digitalis poisoning causes coupling of the heart beats due to the occurrence of right

or left ventricular premature beats alternating with normal ones. This effect is best seen in patients with auricular fibrillation. A problem therefore arises as to the advisability of giving digitalis in cases of failing compensation where premature beats are present. It is important first of all to find out whether the patient has been taking digitalis and, if so, the amount. If from the history one can exclude digitalis as the cause of these beats, there is no contra-indication to its use.

In myocardial degeneration the occurrence of premature beats is often of significance, and is an early indication of myocardial failure.

Right ventricular premature contractions are common in cases of failing right heart, and if auricular fibrillation is not already present indicates its probable occurrence.

In complete heart block the occurrence of premature beats is usual where the ventricular rate is below 30, and in this condition is beneficial.

Left ventricular premature beats are always intrinsic, and are found in toxic myocarditis and myocardial failure. Their occurrence in elderly men with cardiovascular sclerosis is of serious import.

Auricular premature beats are usually intrinsic, and are most frequently seen in mitral stenosis. They indicate auricular fatigue and foreshadow the occurrence of fibrillation.

Nodal premature beats are mostly intrinsic and are seen chiefly in toxic myocarditis and in myocardial degeneration. In view of their position at the A—V node their presence must be considered of some gravity.

The T wave is considered to represent the alterations in electrical potential due to the contraction of the ventricular muscle, principally in the region of the base. Its amplitude depends upon the state of the ventricular muscle. A T wave of high amplitude is found in the hearts of athletes, whereas its size tends to diminish with age,

and in elderly people it is usually of low amplitude. Cardiac hypertrophy from whatever cause, provided the muscle is healthy, will show a large well formed T wave in leads 1 and 2. T_1 is considered by certain cardiologists to refer to the right ventricular muscle and T_2 to the left. Up to the present this theory is little more than a surmise and a great deal more work will have to be done, both from the scientific and clinical aspects, before our knowledge of the T wave becomes firmly established. Certainly cases of simple mitral stenosis without evidence of failure show a large P wave and right ventricular preponderance in association with a large T_1 .

Again, in the case of athletes, although T_1 is well formed, it is T_2 which shows the typical enlargement.

Thus, if we elaborate this theory, we should expect cases of general cardiac hypertrophy to show large T_1 and T_2 , those with right ventricular hypertrophy to show a large T_1 and those with left a large T_2 . If the T wave does not conform to our expectations we can presume that there is damage or disease of the muscle. For example, a case of developed mitral stenosis with a poorly formed or negative T_1 , should have a worse prognosis than one with a well formed T_1 , and we may anticipate the serious effect of any pulmonary complications.

Again, a case of aortic reflux with a poorly formed T_2 will suggest a damaged left ventricular muscle, perhaps the result of deficient blood supply from a narrowed coronary orifice, and the prognosis will be entirely different from a similar case with a normal electrocardiogram.

Inversion of T_1 and T_2 is considered more serious than mere flattening, and Pardee has shown that the mortality of such cases is extremely high.

T_3 is still an unknown phenomenon ; it is found in about 30 per cent. of normal hearts.

I have, however, noted certain cases whose improvement has coincided with a

change of T_3 from negative to positive. A negative T_3 in these cases may therefore indicate a lack of tone in the ventricular muscle.

The following records of cases demonstrate some of the points raised :—

Case 1.—M. M., female, aged 27. Flatulent dyspepsia with heart burn. Heart normal.

E (fig. 1), physiological record.

FIG. 1.

Case 2.—J. B., male, aged 23. Rowing man in good training.

E (fig. 2), large, well-formed T waves, especially in lead 2.

FIG. 2.

Case 3.—B. C., male, aged 70. Seen in routine examination. Heart normal,

E (fig. 3), slight left-sided preponderance, T waves subphysiological. Typical record of a senile heart.

FIG. 3.

Case 4.—L. G., male, aged 70. Myocardial degeneration with commencing failure. Heart moderately enlarged, no valvular disease. B.P. 122/76.

FIG. 4.

E (fig. 4), left-sided preponderance, small T waves and left ventricular premature beats. Death nine months afterwards from syncopal attack.

Case 5.—J. H., male, aged 66. Myocardial degeneration with associated chronic bronchitis. Heart moderately enlarged, no valvular disease. B.P. 170/90.

E (fig. 5). Marked left-sided preponderance, poorly formed T waves and auricular premature beats tending to alternate with

FIG. 5.

the normal beats. Two years after, going along satisfactorily under medical supervision and very limited activities.

FIG. 6.

Case 6.—J. W. F., male, aged 57. Syphilitic mesaortitis with aortic reflux. Heart large with heaving impulse. Free aortic regur-

gitation. B.P. 166/64. X-ray showed dilatation of ascending aorta.

E (fig. 6), absence of expected left-sided preponderance and poorly formed T waves. P waves in leads 2 and 3 large.

Death in six months from angina.

We much regret that there was a printer's error in the authorship of the paper in our last issue on "The Stools in Infancy." The author is Dr. Maizels, and not as was printed. The paper was founded on the work that Dr. Maizels has done over a long period at the Infants' Hospital, Vincent Square.

POST-GRADUATE NEWS.

A FEW remarks concerning the special courses for March may be helpful to intending entrants.

From March 10 to March 15 an all-day course at the Hospital for Consumption, Brompton, will be available. This course will comprise instruction by lecture, by demonstration and by operation. Fee £3 3s.

Beginning also on March 10 but continuing for two weeks, the Staff of the Royal National Orthopædic Hospital will hold an all-day course. Lecture-demonstrations will be given each day, and the clinical practice of the hospital will be open to entrants. As this course is only held subject to an entry of ten, early application is requested. Fee £3 3s.

For those post-graduates who have but the late afternoon at their disposal, a course of clinical demonstrations upon selected cases will be given in the Out-Patient Department (including one or two demonstrations at the In-Patient Department) of the West End Hospital for Nervous Diseases, at 5 p.m., from March 17 to April 11 inclusive. A minimum entry of ten is insisted upon, so early application is requested. Fee £2 2s.

A post-graduate course in Venereal Diseases will be held at the London Lock Hospital, from March 17 to April 12. Afternoons and evenings will be occupied, and the fee for the course is £2 2s.

From March 24 to April 12, a course in Medicine, Surgery and Gynæcology will take place at the Royal Waterloo Hospital for Children and Women. Afternoons and some mornings will be occupied. Fee £3 3s.

On the last day of the month (March 31) a one week's all-day course in Gastroenterology will be held at the North East London Post-Graduate College (Prince of Wales's Hospital), Tottenham, N.15. Instruction will be given from the medical as well as the surgical aspect. Fee £4 4s.

We would draw attention to the fact that *members* of the Fellowship of Medicine and Post-Graduate Medical Association are invited to attend the Annual General Meeting of the Association on Monday, March 31, at 5.30 p.m., to be held at the house of the Royal Society of Medicine, 1, Wimpole Street, W.1.

Detailed information relating to all courses (including the "General" consisting of the clinical practice of the hospitals associated with the Fellowship of Medicine) as well as the programmes of lectures and clinical demonstrations, can be obtained from the Secretary of the Fellowship of Medicine.

M.R.C.P. SPECIAL COURSE.

The dates of the next course arranged by the Fellowship of Medicine have been fixed for May 13 to July 4, and will consist of a series of lectures given by masters in their own particular branch of medicine. The lectures will be delivered (unless otherwise stated), at the Medical Society of London, 11, Chandos Street, Cavendish Square, W.1, at 8.30 p.m. The fee for the course is £6 6s., or 10s. 6d. per lecture. It is hoped to issue the detailed syllabus shortly.