is not blue he tends to call yellow, as that is the brightest part in the not-blue region of the spectrum.

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SOME CARDIOLOGICAL FALLACIES

BY

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Even to-day unfounded beliefs regarding cardiovascular disease appear to be so widespread in the profession that we think it worth while to discuss some of the commonest of them in a short paper.

Fatty Heart

For a number of reasons, into which it would take too much time to enter, all obese people tend to become short of breath on exertion. On this account alone a diagnosis of "fatty heart" is often made. In post-mortem examinations of the obese—and not only of the obese—it is not unusual to find fat stored in and around the heart, but during life its presence can so rarely be proved to give rise to cardiac insufficiency that it is never wise to make the diagnosis. When definite symptoms of cardiac insufficiency are present in an obese patient another cause for them should be sought. The commonest by far is hyperpiesis, but there are, of course, others.

Physical Strain

The belief that a healthy heart can be injured by physical strain still unfortunately persists, in spite of the immense amount of work that has been done on the subject during the last thirty years. The truth of the matter has never been better put than by Lewis (1937) when he says: "It is reasonable to conclude that the burdens imposed by physiological acts upon the normal heart, however heavy these burdens may be, never injure the heart and fibres, never produce injurious dilatation, and never exhaust the heart's reserve." That the heart tends to be a little larger and rounder than the average in persons who habitually do hard physical work is not denied, but it is a normal and healthy reaction of the

muscle to exercise and no more to be accounted pathological than the hypertrophy of, say, their biceps.

There is a corollary to this: Every now and then one sees a patient in consultation whose heart, according to his medical attendant, has recently dilated enormously within the space of a few minutes or, at most, a few hours, usually, though not always, as a result of effort, and has returned to its normal size, either with rest or through his treatment, just as rapidly. Such acute dilatation of the heart, in our experience, never occurs. In this connexion it is interesting to observe that in a prolonged attack of "paroxysmal tachycardia," in which the heart beats continuously at a rate of over 200 a minute, appreciable enlargement—when it develops—is a matter of several days.

Screening the Heart

Even in a patient neither emphysematous nor obese it is impossible to estimate the transverse diameter of a heart with any accuracy by percussion—one has only to screen a normal chest in the lateral or oblique position to be persuaded of this. And, supposing it were, no one has ever suggested that the depth of a heart—a very important matter in cases of mitral stenosis, congenital morbus cordis, etc.—can be estimated by this means. Yet not only can both of these points be elucidated by screening, but a reasonably accurate estimation of the size of each individual chamber of the heart—surely a consummation earnestly to be desired—is as a rule easily made by this means.

Displacement of the apex beat to the left, where this cannot be attributed to pulmonary causes, though very suggestive of an enlarged heart, is not absolutely diagnostic. Scoliosis, even of so slight a degree as to be only recognizable by radiography, is a fruitful cause of such displacement. Again, a high diaphragm—the constant accompaniment of obesity—by causing the lie of the heart to become more transverse, tends to produce it. Through ignorance of these facts on the part of medical men, multitudes of healthy people are accounted victims of heart disease who, were their hearts screened by anyone conversant with the technique, could be safely assured as to the integrity of that organ.

Enlargement and Dilatation

The heart is generally believed to increase in size with age, as a normal process. There is no evidence for this. It may even diminish in size. When it is found to be enlarged in old age another cause should be looked for—for example, hyperpiesis, bundle-branch block, etc.

It is almost an article of faith in the profession that in all toxæmic states of any severity there is necessarily some dilatation of the heart through flabbiness of its musculature. It is forgotten that in cases of very advanced phthisis where there has been the severest degree of toxæmia for months or years the heart in life usually appears smaller than normal under the screen, and at necropsy is found to be wasted, perhaps, but not dilated. Again, in a recent radiological study of 119 hearts in pneumonia by Davies, Hodgson, and Whitby (1935), definite enlargement was witnessed in no more than three.

It is, we believe, practically only when auricular fibrillation, auricular flutter, or heart-block develops in the course of toxæmia that the heart dilates. Circulatory failure, when it occurs in toxæmia, is in our opinion almost always due to loss of contractility of the muscular coat of the peripheral arterioles and not to myocardial weakness.

Contrary to common belief, in cases of congestive failure the size of the heart does not materially decrease as the signs of failure diminish and disappear. A generation ago Mackenzie, estimating the size of the heart by clinical methods, came to this conclusion. Latterly Palmer (1937) has established the truth of it by radiological methods.

Oedema which is not accompanied by enlargement of the heart is practically never cardiac in origin. It must be borne in mind, however, that such enlargement may not be obvious clinically and that radiological examination of the heart may be required before it can be detected—for example, in emphysema and obesity.

Heart Sounds and Extrasystoles

A heart is often unjustly condemned because in the opinion of the examiner the sounds are "weak," "toneless," "of poor quality," or the like. The *character* of heart sounds, whether first or second, is on occasions of great help in the diagnosis of cardiac conditions, but the idea that one can gauge the functional efficiency of a heart by the *loudness* of the sounds is altogether fallacious. Dulling of heart sounds is far more often due to obesity or emphysema, or to both, than to a feeble myocardium. In addition chests possess very different acoustic properties, and listeners differ greatly, not only in the acuity of their hearing but also in their standards of normality.

Even at the present time many practitioners regard extrasystoles as an indication of organic disease of the heart—to the great hurt of their patients. As Mackenzie (1918) pointed out, extrasystoles are in themselves of no clinical significance, and seldom, if ever, are they harbingers of auricular fibrillation or any other grave cardiac condition. After middle age they may be considered almost a normal phenomenon. When present in a case of organic disease of the heart—as they often are—they may be safely ignored in assessing the seriousness of the condition and in considering the prognosis.

Rheumatism

It is well known that acute rheumatism is comparatively rare in adult life. It is not so well known that a first attack which develops after the age of 20 seldom causes permanent damage to the heart and that, below this age, the nearer a patient is to it the less likely is his heart to be affected.

One is often asked during or at the end of a first attack of acute rheumatism whether the heart of the patient is affected or not. It is frankly impossible to answer this question. The presence of a systolic bruit at the apex is of little help. A transient systolic bruit is a commonplace in any severe toxæmia, and a systolic bruit in a first attack of acute rheumatism may be no more than this; moreover, a patient in whom at the end of the attack no bruit of any kind whatever is audible may a year or two later exhibit definite signs of valvular disease.

The scarring of the valves which follows their inflammation, and to which mitral stenosis, aortic regurgitation, and aortic stenosis are due, is a slow process, and it is almost certainly six months at least, and generally much longer, before it has advanced sufficiently to produce recognizable signs of these conditions. When, during the course of a supposedly first attack of acute rheumatism, definite signs of valvular disease are discovered, it can be laid down with certainty that the patient has had a previous attack, even if it has gone unnoticed.

Systolic Murmurs

Mitral regurgitation, unaccompanied by mitral stenosis, seldom, if ever, occurs as a result of rheumatic endocarditis. Contrary to current teaching, a *systolic* murmur at the mitral area is the commonest murmur in mitral stenosis, not a presystolic. This fact has been firmly established in recent years by radiological examination of the heart, although Graham Steell drew attention to it as long ago as 1906. It is the long, blowing, apical systolic murmur in a patient who gives a history of previous acute rheumatism which is especially suspect. "Functional" apical systolic murmurs are typically short and non-blowing in character. In no branch of cardiology is fluoroscopy more useful than in determining the significance of systolic murmurs.

Dizziness, Faintness, and Syncope

Dizziness, faintness, and syncope are not *cardinal* symptoms of heart disease. That they can occasionally result from it is not denied—unconsciousness, for example, occurs in some, though by no means all, cases of high-grade heart-block and, more rarely, at the onset of an attack of auricular fibrillation, auricular flutter, or coronary thrombosis—but far more often they are symptomatic of a vasomotor disturbance of psychoneurotic origin. Neither is exhaustion or a feeling of tiredness a cardinal symptom of heart disease.

It cannot be laid down too strongly that the cardinal symptoms of heart disease are two, and two only: (i) shortness of breath on exertion, and (ii) pain, usually retrosternal, though sometimes in such situations as we describe elsewhere in this paper.

Pain

The pain of angina pectoris is typically retrosternal, not precordial, as is commonly believed. It tends to radiate across the front of the chest, often to the left shoulder and axilla, in many cases down the left arm as far as the elbow or even the fingers, sometimes down both arms or down the right arm only, and upwards to the neck on one or both sides. It may involve the jaw, it may spread to the back, it may have other situations and distributions, yet hardly ever is it situated below the left breast. Wherever it is or whatever its spread, it cannot be laid down too strongly that it is almost invariably brought on by exercise and relieved by rest.

The pain in an attack of angina pectoris may vary in different patients or in the same patient at different times from a dull ache to an agonizing feeling of constriction involving the whole thorax. It is never "stabbing" in nature.

Left submammary pain is very rarely due to heart disease, even though it may sometimes extend to the left scapula and, by way of the left supramammary region and left shoulder, to the left arm. Such pain is persistent, aching in character, and not induced, though it may be increased, by exercise. It is usually associated with a group of symptoms indicative of physical and nervous exhaustion which have no organic basis (Baker, 1930). Such symptoms do, of course, also occur in patients with cardiovascular disease—for example, mitral stenosis or aortic incompetence—for it is only natural that such patients should tend to develop anxiety states.

Coronary Thrombosis

One has often found a practitioner doubtful if a coronary thrombosis has occurred in a patient who has

experienced all the typical symptoms, because, on clinical examination of the heart, he can find no changes of any kind whatever. It may be interesting for such a one to know that, though a coronary thrombosis can give rise to any of the known arrhythmias, in the majority of cases the rhythm remains regular, no bruits are produced, and no increase in the size of the heart can be demonstrated even by radiological methods.

It is commonly believed that an electrocardiogram taken after a coronary thrombosis *invariably* shows characteristic abnormalities. This is not so. Very occasionally it is normal in every particular. Again, a reversed T wave in Leads I and II is not *invariably* a sign of coronary disease. It occurs sometimes in severe grades of aortic stenosis and hyperpiesis.

Coronary thrombosis very seldom occurs as a result of syphilitic aortitis, in spite of the fact that in this latter condition narrowing of the orifices of the coronary arteries is almost the rule.

Some Further Points

That a patient's systolic blood pressure reckoned in millimetres of mercury should equal 100 plus his age in years is still an article of faith in many quarters. Mass statistics now available prove that this is not the case. The blood pressure should normally rise only slightly with increasing age. This is evidenced by the fact that in normal people the increase between the ages of 20 and 60 is no more than 15 mm.

Atheroma is never a cause of high blood pressure, though there is little doubt but that the latter ultimately tends to lead to atheromatous degeneration of the arteries.

Syphilis never causes a *general* arteriosclerosis. The arterial involvement in syphilis is invariably focal.

It should always be borne in mind that aneurysm of the thoracic aorta scarcely ever causes dysphagia with regurgitation of food, although it may occasionally be accompanied by some discomfort in swallowing.

Auricular fibrillation is, curiously, very rarely associated with subacute bacterial endocarditis or syphilitic disease of the heart.

We would acknowledge our indebtedness for many of the points we have stressed to the late Sir James Mackenzie, with whom one of us was associated for a considerable time, and to Dr. John Parkinson, with whom both of us have been closely associated in recent years in the cardiac department of the London Hospital.

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As a result of this year's collection the Metropolitan Hospital Sunday Fund has a sum of £51,000 to distribute. The money will be allocated to hospitals, convalescent homes, dispensaries, and nursing associations in the London area. At a meeting of the council of the Fund the report of the Distribution Committee was adopted. The larger allocations include £3,400 to the London Hospital; £2,010 to St. Thomas's; £1,950 to the Royal Free; £1,850 to the Middlesex; £1,800 to University College Hospital; £1,540 to Guy's; £1,400 to St. George's; £1,360 to St. Mary's; £1,200 to Charing Cross; and £1,190 to King's College Hospital.

THE "RESTROPIC" ACTIVITY OF BLOOD

BY

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In previous publications (Wetzler-Ligeti and Wiesner, 1937, 1938a, 1938b) we have described the effects of various pituitary extracts upon the reticulo-endothelial system (RES) of the rabbit. Congo-red solution was injected intravenously and the rate at which the dye disappeared from the circulation was determined; this rate has been taken, in accordance with the view of previous authors (Adler and Reimann, 1925; Moncorps and Günther, 1933; Dieryck, 1929; Stern and Willheim, 1935), as an index of the functional activity of the RES. It was found that certain anterior lobe extracts raised the Congo-red index by a considerable amount. The active substance—termed the positive RES-tropic (restropic) factor—is not identical with either gonadotropic or thyrotropic hormone. In addition to positive restropic extracts, the anterior lobe yields a substance that rapidly, though only temporarily, lowers the index and thus presumably inhibits the function of the RES. This negative restropic factor is closely associated, or identical, with the thyrotropic hormone.

These experiments suggested that the RES was subject to endocrine control. Since other hormones, such as the oestrogenic and gonadotropic substances, can be demonstrated in the circulation, the experiments have been extended to the examination of blood. Some of the results are presented here, in extension of preliminary notes on the subject published elsewhere (Wetzler-Ligeti and Wiesner, 1938c; Czato, Wetzler-Ligeti, and Wiesner, 1938).

Method of Extraction

Fresh blood—obtained from the veins of rabbits or human subjects, or from the heart of the horse—was defibrinated by shaking it with beads. An equal volume of a 20 per cent. aqueous solution of sulphosalicylic acid was added to the defibrinated blood and the mixture was shaken vigorously. The precipitate was collected in a centrifuge and taken up in 50 ml. of 6 per cent. aqueous ammonia. The mixture was allowed to stand for twelve hours at a temperature of 37° C. It was then centrifuged and the precipitate was discarded. The supernatant liquid was adjusted to pH 5 by means of acetic acid, and an equal volume of 90 per cent. alcohol was added. The resulting precipitate was discarded. An equal volume of 95 per cent. alcohol was added to the supernatant liquid. The precipitate was made up with distilled water to a volume the same as the initial volume of blood. The precipitate did not wholly dissolve, and the solution was clarified by centrifuging. The whole process was carried to completion within twenty-four hours of collecting the blood sample. The extracts were stored at 4° C., as the activity is lost at room temperature.

The general properties (solubility, stability, etc.) of the active fraction agree with those of the pituitary extracts obtained by similar methods.

Determination of Congo-red Index

Stern and Willheim's modification (1935) of the Congo-red method (Adler and Reimann, 1925) was again used. Virgin rabbits obtained from a commercial breeder were employed in these experiments. Uniformity of stock and age is desirable; for although the Congo-red index tends only to vary slightly in untreated rabbits (± 5 per cent.),