Observations

ON

THE SIMULATION OF HEART DISEASE*

BY

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The imitations of heart disease are as variable and frequent as actual heart diseases. Many individuals who experience supposedly cardiac symptoms believe erroneously that they are the victims of heart disease, and their belief is not uncommonly confirmed by the non-committal diagnosis of "weak heart" or "myocarditis." Such patients may suffer from all grades of ill-health, from the state of chronic ailing and malaise to the serious degrees of bed-ridden invalidism. Moreover, it must be the universal experience that a very large bulk of disability in all walks of life arises from those disorders of the circulatory mechanism which produce the symptoms of heart disease but not the signs. Because a disorder of the nervous mechanism underlying the action of the heart and the circulatory system is generally held to be the basis of the conditions simulating heart disease, the term "neuro-circulatory asthenia" is best adopted to include all of them.

Breathlessness is recognized as the cardinal symptom of heart disease. It may likewise be the presenting symptom in neuro-circulatory asthenia, but it is not usually difficult clinically to differentiate between true and spurious heart disease. Still, some patients with dyspnoea actually of cardiac origin have very few, if any, supporting signs of heart disease, and these occasionally may be difficult to assess correctly. Further, the subject of organic heart disease may suffer also from some degree of neuro-circulatory asthenia, while it occasionally happens that a patient possessing a syndrome characteristic of one or other form of neuro-circulatory asthenia is ultimately declared, possibly by the electrocardiograph, possibly by the turn of events, as the possessor of serious organic heart disease. It must be admitted, therefore, that much difficulty may uncommonly attend the determination of the true state of affairs. Obviously, it is of fundamental importance to make the correct diagnosis; this, in general, may be an assertion that the patient is suffering from heart disease, or that no heart disease is present, or that neurotic symptoms are complicating organic disease of the heart.

How these disorders of the neuro-circulatory mechanism arise is largely a matter for conjecture. Willius¹ believes that their origin is primarily psychological, depending upon the universal conception of the heart as the hub of physical and spiritual activity. He refers to the Arts generally as constituting a "synthetic emotionalism" vividly bringing the heart to the attention of many persons. Boas² admits the existence of a psychological basis for many neurogenic disorders of the heart, and holds that they are frequently precipitated by increased activity of the reflex arcs as a result of excessive or exaggerated stimuli, or by increased irritability of the visceral nervous system of unknown localization. He further cites the common association of peripheral vasomotor disorders and neuro-circulatory asthenia in support of his view that neuropathic symptoms may owe their origin to altered blood distribution caused by local neurogenic vasomotor anomalies. Laubry³ considers that hyperexcitability of the nervous centres may be constitutional or acquired, and that the factors concerned in the production of the

latter group are intoxications, repeated emotional upsets, and general infections.

There are three principal syndromes which may simulate heart disease. They may be classified as follows:

1. The effort syndrome.
2. Left mammary pain: (a) chronic, (b) paroxysmal.
3. Retrosternal pain of muscular origin.

In general, the recognition of these syndromes depends partly upon the context of the patient's complaint, and partly upon the absence of signs of cardio-vascular disease. The symptoms are usually numerous, and their recital may present evidence of exaggeration; although any one of these syndromes may be isolated and clear-cut in a given case, all grades, variations, and combinations may occur. Essentially, the attitude of the physician should be to believe the heart healthy unless there are signs of disease or other indications of serious significance.

THE EFFORT SYNDROME AND INVALIDISM

The patient afflicted with the effort syndrome complains usually of breathlessness, palpitation, dizziness, faintness, and tightness or pain in the chest. These symptoms are provoked by effort—often by trivial degrees of effort. They may alternatively or in addition be produced by excitement. Further, the power of a given exertion or of a certain emotional factor to produce these symptoms tends to vary very much from time to time. Conversely, patients suffering from cardio-vascular disease complain predominantly and perhaps exclusively of dyspnoea which is either related to exertion or occasionally nocturnal. Excitement in them does not typically produce dyspnoea, and the degree of breathlessness is generally more or less closely related to the degree of effort, and some measure of cyanosis nearly always coexists.

Other important indications in differential diagnosis may be derived from the history. On what account is exertion limited? If the patient complains that the "legs go weak," or that "exhaustion" compels him to rest or sit down, he is in all probability suffering from functional disorder and not from disease. Mackenzie⁴ has expressed this important distinction as follows: "When a patient, for instance, states that on exertion, as in walking, particularly upon the level, he is pulled up by pain in the chest or breathlessness, then the probabilities are that it is the heart which is producing his limitation. If, on the other hand, he becomes tired and exhausted after walking a relatively short distance, that is, a distance much shorter than he was able to undertake with comfort in good health, then it is always certain that the limitations are due to conditions other than the heart." Mackenzie further made the important observation that in the case of organic heart disease the patient feels quite well between the bouts of dyspnoea or pain, while the patient whose symptoms are not truly cardiac has a background of other "vague and indefinite sensations by which he knows that he is not well."

The underlying causes of the effort syndrome have been classified by Lewis,⁵ who investigated 588 soldier patients. He found that the symptoms dated from an infectious disease in one-third of the cases, while such diseases were considered to play a major part in the production of the syndrome more often than not. White⁶ has attempted an etiological classification of the effort syndrome among civilians. He discounts the role of infection, and holds that fully nine-tenths of these patients are the subjects of fatigue or introspection neuroses, the remainder having anxiety or substitution neuroses. Infection as a factor in the production of this form of neuro-circulatory asthenia may be regarded in the following way. If a patient complaining of the typical syndrome has it as a result of infection, then such infection is generally revealed either

* Based on a post-graduate lecture delivered at the Charing Cross Hospital Medical School on October 4th, 1931.

in the history or on physical examination. If no such infective cause is evident from these sources, it becomes unlikely that infection is at the root of the trouble.

Just as the diagnosis of angina pectoris implies liability to sudden death, so a recognition of the effort syndrome should imply the danger of invalidism. Confinement to bed should therefore be no part of the treatment of this disorder. Lewis⁸ aptly summarized this point when he wrote: "The bed treatment of cases showing the effort syndrome is deplorable. The patients deteriorate in health; they become undisciplined and hospitalized; it becomes ever more difficult to disabuse them of the notion that their hearts are diseased. The inevitable tendency of bed treatment is to exaggerate the complaints and to render them more persistent." The following case serves as an illustration of this danger:

A. M., aged 33, was a young woman living with her husband and her mother-in-law. There was a history of rheumatic fever in childhood, but there had been no other serious illnesses. She complained of palpitation and breathlessness, sometimes together, sometimes separately, feelings of dizziness and faintness, and occasional pains across the left breast. These symptoms were related usually to effort or excitement. She had been twice in hospital during the past three years, and the disorder had been looked upon as due to cardiac disease. Courses of vaccine treatment, directed against oral sepsis and infection of the colon, had been given, without improvement. Courses of acetylcholine, nitroglycerin, and digitalis had likewise been tried without avail, and the patient had been bed-ridden for nine months when she first came under observation.

At this time she was pale, with moist skin, variable tachycardia at 130, and bouts of tachypnoea and precordial distress. She was admitted to Charing Cross Hospital, where complete investigation of the cardio-vascular system revealed no evidence of disease. The patient was put on a system of graduated effort, first sitting out of bed, then standing, and walking, and, lastly, climbing the stairs. The increasing stages were insisted upon regardless of complaints of palpitation, faintness, etc., and the patient then returned home, where, under the relentless observation of her local doctor, she ultimately became able to resume indoor and outdoor household duties.

The psychological basis of this case is interesting. Before the onset of her illness the patient had been obliged to overwork on account of her mother-in-law's illness. It became evident that the resort to bed was in part, and subconsciously, a measure of retaliation, and a ruse to transfer the burden of duties upon the mother-in-law.

LEFT MAMMARY PAIN

A syndrome of which dull aching or gnawing pain under the left breast is the principal factor is generally known as left inframammary pain. It has been studied of late years by Parkinson,⁷ who has stressed the common association of this pain and left subscapular hyperalgesia. He found that patients complaining of this symptom were as likely to be free from any disease as afflicted with cardio-vascular disease. Studying a larger consecutive series of 332 patients attending a cardiac clinic, Baker⁸ observed that 42.3 per cent. of patients presenting no evidence of cardio-vascular disease complained of this symptom, whereas among those afflicted with organic disease it appeared in only 22 per cent. of patients. Further statistical evidence was forthcoming from this study to show that a patient suffering this type of pain is more likely to be normal than the subject of hypertension or mitral stenosis—the two most common cardio-vascular diseases.

In assessing the significance of this symptom of left inframammary pain it must be realized that pain in this situation very often occurs in association with heart failure, especially if fibrillation has supervened. The

writer⁹ has shown that such pain was experienced by 21 patients out of 27 in whom pain or discomfort attended congestive failure. Of 16 consecutive patients with auricular fibrillation, 13 complained of left inframammary pain. The presence of such pain in heart disease does not necessarily mean that the disease is complicated by a superadded form of neuro-circulatory asthenia, since there may be complete absence of all other elements of the characteristic syndrome. Further, the presence of chronic left inframammary pain in association, say, with hypertensive disease, scarcely modifies either prognosis or treatment.

The subject of chronic left inframammary pain often suffers from respiratory troubles. These may include breathlessness on exertion, but the complaint is more usually of inability to take a deep breath, or of a stifling or choking sensation, or it may be noticed that the patient takes at intervals deep sighing respirations. All these manifestations occur independently of effort, often being relieved by it. Thus, in these patients the respiratory symptoms commonly show an inverse relationship to effort, being less rather than more noticeable with moderate effort. Herz,¹⁰ describing the association of this symptom with palpitation and mammary pain, termed it "phrenocardie," and remarked that it was particularly apt to occur in women with genital disorders or sexual neuroses. Gallavardin¹¹ has shown by radiography that these sighing or yawning respirations are associated with a restricted action of the diaphragm, from weakness or spasm.

A host of collateral symptoms frequently accompany left inframammary pain. Exhaustion may be a salient complaint, the capacity for effort being much greater on some days than on others. Very often the patient states that her nerves are bad, and she may further be troubled by pains all over, by fainting attacks, trembling fits, and insomnia. While these manifestations are principally subjective, they are sometimes far from trivial, and occasion great physical distress and mental agitation.

Left inframammary pain in the absence of cardio-vascular disease seldom afflicts men, and, when it does, it is generally the sequel to over-smoking. When tobacco is the cause, the pain is apt to be paroxysmal, and this syndrome will be referred to below. Etiological factors among women are principally a highly strung temperament, hyperthyroidism, and menopausal or other genital disturbances. It has been seen above how phrenocardia is specially frequent in those syndromes accompanying sexual disorder. The following case serves to illustrate the relation of menstrual function to this group of disorders.

A widow of 35 had no ill-health until eighteen months ago, when she began to experience attacks of palpitation, with throbbing in the neck and vague pains about the left breast. These symptoms have persisted until the present time, but they have become more continuous, and in addition she now has exhaustion and variable dyspnoea on effort. Deep sighing respirations are taken at intervals, and the patient feels that she "never has enough air." All the symptoms become more severe and distressing during the ten days preceding menstruation, and also during the five days that this lasts. Maximal relief is experienced in the intermenstrual period. Physical examination has revealed no evidences of heart disease, and the x ray and electrocardiogram are normal. Ventricular extrasystoles are frequent at times. A fine tremor of the fingers is present, but there are no other signs of hyperthyroidism. During the last two months, in which an exacerbation of the symptoms has occurred, the periods have been noticeably scanty.

The relationship of the severity of symptoms to menstruation, the recent suggestive diminution of the menses, and the phrenocardia all point in this case to endocrine imbalance of pelvic origin as the basis of the functional malady.

PAROXYSMAL LEFT MAMMARY PAIN

Left mammary pain may be of all grades, from the slight, constant, nagging discomfort below the left breast to paroxysms of intense pain accompanied by loss of consciousness. While these extremes presumably represent grades of the same malady, it is probably advantageous to class the group of paroxysmal pain separately. This syndrome was termed by Mackenzie⁴ "secondary angina," and he stated that he could not recall a single instance of death during the attack. Gallavardin¹¹ refers to this group as "angor névrosique," and reserves this denomination for those manifestations which, on account of their intensity, general symptomatology, and paroxysmal nature, resemble angina pectoris. "Paroxysmal left mammary pain" is here applied to those syndromes which simulate, more or less closely, angina pectoris. The benign nature of such attacks renders paradoxical the use in connexion with them of so ominous a term as "angina," however qualified.

The salient symptom is paroxysmal pain of great intensity and lightning onset in the region of the left breast. This may be without obvious cause, or it may appear to be consequent upon some trivial effort. It spreads frequently into the left arm, but rarely elsewhere. Faintness or actual syncope may follow the abrupt onset. Restlessness, palpitation, and throbbing in the neck are characteristic sequelae, while feelings of heat and cold and tingling and numbness in the limbs are also common. While this syndrome is highly suggestive of a functional malady, it is not every patient that presents a picture so concise. For example, the onset may be related to strenuous effort, the pain not involving the usual left inframammary site, but described as shooting across the chest; again, the patient may be the subject of underlying organic heart disease, rendering him more liable to true angina pectoris, while it may be that none of the collateral phenomena usually associated with paroxysmal left mammary pain are observed. It is in cases of this type that the physician may be obliged to reserve judgement, at the same time continuing the pursuit of semiological clues or objective evidence either pointing in the direction of a true coronary syndrome or leading to the inference that the syndrome is not of organic but of functional origin—paroxysmal left mammary pain. The following case illustrates the syndrome now under consideration:

An obese female patient at the climacteric, aged 48, sought advice at the out-patient department on account of general exhaustion, breathlessness, palpitation, and aching in the region of the left breast. A mild degree of hypertension was present, and there was a simple tachycardia varying between 120 to 150 beats per minute. There were no other signs of cardio-vascular abnormality. On treatment by luminal (1 grain *omne nocte*) there was progressive subjective improvement, until one day she was sent into hospital urgently on account of supposedly anginal seizures. In an attack witnessed in hospital it was observed that the onset was with palpitation, succeeded immediately by intense pain at the left breast, spreading down the arm. The patient lay back on pillows, restless, rather pale, breathing hurriedly, and with a heart rate of 160. She complained of trembling, and "pins and needles" in the legs, and there was marked hypersensitiveness at and below the left breast. The blood pressure was unaltered during the attack, which lasted about half an hour from the onset, until by degrees the pain and other symptoms subsided. Amyl nitrite gave no relief. In such attacks at her home great alarm had naturally been aroused, and relatives had gathered at the bedside awaiting the fatal outcome. By rest in bed, reassurance, and the regular administration of bromide and chloral and luminal the attacks became much less frequent and severe, while the pulse rate fell to the region of 90. A measure of disregard of the attacks expressed by the attendants, both in hospital and at home, also seemed to curtail the paroxysms.

A reflex mechanism is believed to underlie the phenomena of paroxysmal left mammary pain. As a result of some unknown toxic, endocrine, or metabolic agent the afferent neural paths from the heart become so hyper-sensitive that a trivial stimulus, such as momentary acceleration or palpitation, is greatly exaggerated, and therefore misinterpreted, by the medulla. Widespread discharges along the efferent paths from the medulla produce, in the case of the heart, acceleration and violent palpitation, in the case of the respiratory apparatus, tachypnoea and disturbance of diaphragmatic action, in the peripheral parts pain and hyperaesthesia of the left mammary and arm regions. Sweating, flushing, and paraesthesiae are all explicable on the same basis. It is clear that the same neural paths subserve the production of pain in angina pectoris; but here the afferent paths may be little, if at all, hyperexcitable, while on the other hand the stimulus, whether it arises in the heart, myocardium, coronary arteries, or aorta, is gross. An intense afferent stimulus excites the medulla, by which the pain is referred to the chest and arm, fewer parallel sensory phenomena occurring. In contradistinction to the syndrome of paroxysmal mammary pain, motor discharges to the intercostal musculature commonly occur in angina pectoris, with the production of a characteristic sensation of constriction.

Differential diagnosis between the syndrome of hyperexcitability and angina pectoris is usually not difficult, although any of the collateral manifestations of paroxysmal mammary pain may occur in angina. If it is remembered that a wealth of subjective and objective phenomena speaks against angina, that the functional attacks mostly afflict women at or before middle life, that amyl nitrite will relieve anginal but not neuropathic pain, and that the pain of angina is usually constrictive and sternal, a mistaken diagnosis should rarely be made.

RETROSTERNAL PAIN OF MUSCULAR ORIGIN

It has been suggested by Briscoe¹² that retrosternal pain during effort, or at night, may arise from exhaustion of the respiratory muscles. He has shown that in some patients over-fatigued muscles may give rise to referred pain in distant areas. Further, pressure over the triangularis sterni may produce pain which is recognized by the patient as identical with the pain of his attacks, and which may also be referred to those areas commonly affected in angina pectoris. Briscoe has demonstrated that the supine position is one which is specially apt to induce exhaustion of the respiratory muscles. He believes that this mechanism underlies the production of nocturnal sternal pain which has hitherto been regarded as of vascular origin, and, further, that the phenomena of angina pectoris can be as well accounted for on a respiratory hypothesis as on a vascular one.

While it may well be that muscular exhaustion can give rise to sternal or radiating pain, there are many facts which render acceptance of Briscoe's general theory difficult. If muscular exhaustion is the cause of anginal pain, how is it that amyl nitrite gives such immediate relief? And why is not anginal pain common in states of intense dyspnoea of extra-cardiac origin? Again, the subjects of nocturnal anginal attacks usually show other evidence of cardiac disease, and are liable to death in the attack. A crucial proof of the myocardial origin of anginal pain is furnished by the work of Parkinson and Bedford,¹³ who have demonstrated transient electrocardiographic changes in the course of attacks of angina pectoris.

It must be concluded, therefore, that paroxysmal sternal pain occurring either during effort or at night is generally of cardiac origin, and consequently of serious significance. It is possible that some nocturnal attacks

may wholly or in part be attributable to muscular exhaustion, and this may be suspected if it is found that thoracic respiration preponderates over abdominal in the supine position, and if excessive tone or tenderness is found in the muscles in the neighbourhood of the sternum. At the same time, all such patients should receive full investigation, since it is less dangerous to mistake muscular exhaustion for angina pectoris than to make the converse error in diagnosis.

TREATMENT

Two factors commonly militate against successful treatment of these conditions which counterfeit cardio-vascular disease. The first is the indecision as to diagnosis frequently felt (and often betrayed) by the physician. The second is the lack of confidence of the physician (usually sensed by the patient) in any line of treatment undertaken. Since moral support and reassurance are the fundamental bases of treatment, and since these must generally be derived from knowledge and experience, patients with these maladies are apt to drift upon a sea of medicaments and half-hearted exhortation, finally to come to grief on the rocks of disability and invalidism.

In general, the patient who can readily be convinced that his trouble is not one that need lead to incapacity or shorten his life derives great benefit from this assurance alone, especially if an introspection neurosis has played any part in the production or aggravation of his symptoms. Sympathetic inquiry should be made into the home and economic conditions of the patient, in an attempt to remedy such influences as worry and anxiety. A brief removal to a totally different environment is often of great benefit. In all cases a search should be made for sources of infection with the aim of removing them. In the effort syndrome group the pursuit of normal healthy exercise must be encouraged, although the patient who has once suffered from a more or less pronounced effort syndrome should be warned that he is not constitutionally fitted for intense or prolonged effort. Certain drugs are found empirically to be of benefit when taken over strictly limited periods; the sufferer from faintness, giddiness, and syncope is often greatly relieved by iron and strychnine, the extrasystole group by quinine and strychnine, and the ultra-nervous group by bromide and valerian.

The general measures already mentioned are applicable in the treatment of chronic left inframammary pain. Obesity, if present, must be dealt with, and is only properly corrected by readjustment of quality and quantity of food intake and energy output. If thyroid extract is employed at all, it should be given in strictly moderate doses and with careful supervision. The writer has recently seen an example of angina pectoris precipitated by the injudicious administration of thyroid. The syndrome dated from three years ago, when the patient was given by his doctor thyroid extract, 15 grains daily, for reduction of his weight. Local applications for left mammary pain only serve to focus the attention of the neurotic upon her pain. Bromide and valerian, or bromide and chloral, may give considerable relief.

Both chronic and paroxysmal mammary pain may be very resistant to treatment. The diagnosis of a benign disorder often does much for the patient, not only directly, but also indirectly, by establishing a cheerful and healthy attitude among relatives. If restlessness and tachycardia are pronounced, luminal (1 grain omne nocte) may lessen the frequency and severity of the attacks, while bromide and chloral may be given in a mixture twice daily in addition. No special treatment is indicated during attacks.

In all of these conditions which simulate heart disease it is of great importance for the patient not to be treated

as an invalid. Frequent consultations and long-continued medication are alike harmful, while admission to hospital is only justifiable for investigation or observation, or when an underlying cardiopathy or general condition is patent or suspected.

REFERENCES

- ¹ Willis, F. A.: *Minnesota Med.*, 1928, xi, 102.
- ² Boas, E. P.: *Amer. Journ. Med. Sci.*, 1928, clxxvi, 789.
- ³ Laubry, C.: *Sémiologie cardio-vasculaire: troubles fonctionnels*. Paris, 1924, p. 69.
- ⁴ Mackenzie, J.: *Diseases of the Heart*. Fourth edition, London, 1925, pp. 353 and 349.
- ⁵ Lewis, T.: *The Soldier's Heart and the Effort Syndrome*. London, 1918, p. 33.
- ⁶ White, P. D.: *Nelson's Loose-leaf Living Medicine*. Revision 1931, iv, p. 635.
- ⁷ Parkinson, J.: *Lancet*, 1919, i, 550.
- ⁸ Baker, D. M.: *Ibid.*, 1930, i, 1280.
- ⁹ Smith, K. Shirley: *Ibid.*, 1929, i, 1080.
- ¹⁰ Herz, M.: *St. Petersburg, med. Woch.*, 1910, xxxv, 72.
- ¹¹ Gallavardin, L.: *Les Angines de Poitrine*. Paris, 1925, p. 100.
- ¹² Briscoe, J. Charlton: *Lancet*, 1927, i, 857; 1921, ii, 1257.
- ¹³ Parkinson, J., and Bedford, D. E.: *Ibid.*, 1931, i, 15.

OCCUPATION AND NEUROSIS *

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"Blessed is he who has found his work; let him ask no other blessedness." So wrote Carlyle before the days of women in industry and in the professions; but as "work" surely includes the rearing of a family, the epigram holds good for both sexes equally.

The reason why the subject of choosing a career is included in a course of lectures under the auspices of the National Council for Mental Hygiene is that the happiness and therefore the mental health, of an individual is largely dependent on a right choice, a wrong choice being often a considerable factor in nervous or mental breakdown. Nervous illness of the most usual kind, as the majority of us now realize, is a manifestation of failure to adapt oneself to the circumstances of life. Among the most important of these are the conditions of one's work. A simple instance will help to make this clear.

The other day a young man came to Guy's Hospital complaining of exhaustion and of slowness at everything. His occupation was, shall we say, that of a shoemaker. He had lost his last job because his employer had found him so slow; and other jobs had been lost for the same reason. He was now 31, unmarried, and had worked at his trade since leaving school at 15. He had very few friends, but had become accustomed to his loneliness. His hobbies were drawing and painting, for which he had displayed definite talent at school—more so than for anything else. I asked him why he had so few friends, and he explained that he had not been at home for some years, having been forbidden the house by his father unless he would eat humble pie and apologize for daring to quarrel with him some years ago. It was difficult to see by what serious offence this rather mild young man could have raised such wrath; he did not drink, or use foul language, nor did he even permit himself to smoke. The father, on the other hand, was one of those "strong" characters, the son being his complementary opposite, probably as a direct result of the life-long paternal domination. His trade was not his own fancy, but his father's. The latter had insisted on his being a shoemaker; he "would see the wisdom of it later on," and a shoemaker's apprentice, willy-nilly, this boy of 15 had become. Thus he was forced into an occupation for which he had neither liking nor aptitude, and had thereby perpetuated his unhappiness and also his inefficiency. One

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