THE HEART, THE PHYSICIAN AND THE PSYCHIATRIST*

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The heart draws the physician and psychiatrist together in a number of clinical situations, and an attempt is made in this paper to review them, with special reference to their psychogenesis, and to indicate what help the psychiatrist can bring to the relief of the clinical conditions under examination.

I. CARDIAC DISEASE AND PSYCHOLOGICAL REACTIONS

The psychological symptoms resulting from organic cardiac disease will depend on whether the cardiac condition comes on precipitately or insidiously, whether the condition is transitory and, if not, the degree of compensation evoked, and finally the pre-morbid personality. The psychological symptoms can be divided into (a) personality disturbance, and (b) symptoms resulting from physiological disturbance of the brain.

CORONARY THROMBOSIS

Generally speaking, the psychological reactions to a recent coronary thrombosis, and the symptoms they embrace, fall under the following headings: (1) anxiety reaction, (2) depression, (3) disturbance of body image, and (4) cerebral symptoms.

Anxiety Reaction

It is understandable that a degree of anxiety should accompany the occurrence of a coronary thrombosis. This psychological aspect of the condition may escape notice in the concentration on the physical condition. When the critical phase is over, and depending on the personality of the patient and on the way he is being managed by his doctor or doctors, a morbid anxiety may set in. The individual worries beyond cause and reason about his business, his finances, his family responsibilities, with the unspoken fear that he has little time if any to set his house in order. He becomes generally apprehensive, is afraid to be left alone, afraid of company, afraid to sleep. He lacks application, and feels restless and may become agitated. Somatic manifestations of anxiety occur—bouts of diarrhoea, excessive sweating, palpitations, giddiness, etc.

The development of an anxiety state in the course of a coronary thrombosis has an adverse influence on the cardiac condition. In a recent case I saw, the patient (Mr. K., aged 53) was so agitated that when every means of sedating him failed, ECT (electro-convulsive treatment) had to be applied. Despite the physician's warnings, he insisted on being motored 30 miles to his farm to reassure himself that the cattle were being properly tended. He was never reassured by these visits, however; and there can be little doubt that the anxiety about the cattle was essentially a displacement of anxiety about himself (see below). He was so afraid to go to sleep that he stayed awake far into the night, keeping the household awake with him, placed himself in an armchair for the night's sleep, waking almost hourly and rousing his wife to keep him company.

This patient kept on going into failure, and I have little doubt that the physician will attribute it to the excessive activity of the patient, on the one hand, subjecting the weakened heart to needless demands on it, and to the physical effect of anxiety on the other, mediated through the autonomic system. The ECT helped to allay the patient's agitation and at no time during this therapy—he had 10 treatments—did he go into failure. In the presence of myocardial infarction, both physician and psychiatrist are chary of employing ECT, but if the alternatives presented are a patient exhausting his cardiac reserve through anxiety and agitation or the physician exposing his patient to the risk of a catastrophe induced by ECT, then the latter is probably the lesser risk, unless the heart is in a very grave condition. I have never had a fatality in the use of ECT in damaged hearts. That is not to say, however, that there is no risk to heart and life. Where there is a clear psychiatric indication for its use, the contra-indication should, in my opinion, be only such a degree of myocardial damage that life is in the balance; and the clearest indication for ECT in coronary thrombosis is agitation. ECT is more often given for depression, and with the depressive reaction I shall deal later, but agitation may be present without any marked degree of depression, and is a bigger threat to the heart than depression.

In advising ECT to an anxious patient, the first response is usually a hostile one, born out of his anxiety; but as one remedy after another fails to give him rest and peace of mind, he is, sooner or later, likely to be won over to the idea. If proof were wanted that anxiety is harmful to the heart and helps to precipitate cardiac failure in the damaged heart, it is available in the observation that when the patient relapses mentally, after cessation of ECT, a physical relapse follows shortly after.

I have mentioned that Mr. K. was afraid to go to sleep. This is, of course, an inevitable sequel of disturbed sleep, and is unfailingly present with severe anxiety. The anxious preoccupations of the day are magnified at night and inflamed by the anxieties buried within the unconscious. The result is that the patient keeps waking, feeling uneasy, worried or panicky, and this further aggravates the nocturnal tendency to cardiac asthma. The effect of these nightly experiences is to aggravate the fear of death, already aroused by the heart condition, and makes the patient fight with and chase away sleep, as if he were fighting for his very life. The patient in such a plight demands stronger and stronger hypnotics with little benefit or satisfaction. The danger of introducing habit-forming drugs to such

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a patient must be obvious to all, and the better remedy, I suggest, is to give ECT. The human capacity for enduring anguish has its limitations. When the physical integrity of the ego is threatened objective appraisal and contemplation of the threat is, as a rule, rendered well-nigh impossible by various devices which are unconsciously stimulated. One of these is displacement of anxiety (see above). The anxiety aroused by the recognition that life is threatened is repressed and unconsciously displaced onto other causes of anxiety, real or imagined, but external to the ego. Mr. K. worried about his cattle. A medical man I saw recently began to worry about the arrangement he had concluded with his assistant to carry on his practice during his illness. When one questions the subject of a recent coronary thrombosis about his anxieties, the one he does not mention, because he is not in fact fully conscious of it, is his anxiety about himself. He confines, however, other anxieties which preoccupy him and are, to a greater or lesser extent, absorbing the primary anxiety about his condition.

Some of the somatic symptoms complained of by patients are probably not directly related to the cardiac lesion, but are referable to the physiological disturbance consequent on the anxiety, as, for example, peripheral paraesthesiae and abdominal paraesthesiae, such as emptiness or coldness in the abdomen.

**Disturbance of Body Image**

There is a disturbance of the body image in cases of coronary thrombosis which, I imagine, few physicians will have failed to observe; and that is the total denial or refusal to recognize that the heart has suffered damage. Either the patient insists he is all right or wants to carry on his life as if nothing has happened. I had one patient who wanted to carry on with a previously planned trip overseas, and another who could not be persuaded to give up his mountaineering, not to mention other patients who insist on returning to active business almost immediately. Nielsen (1948) has referred to this non-recognition of a pathological state as a disturbance of the physiological body-image.

The converse to the above picture is presented by the patient with hypochondriacal preoccupation. Here the disturbed body-image forms the nidus for a number of mechanisms operating within the psyche. One is the tendency to hypochondriacal rumination. Some individuals are more disposed than others to keep track of their bodily sensations and are, of course, more likely to be come absorbed in them when they undergo pathological change. One has heard it alleged that the injudicious address of the medical attendant to his patient and his fussy management of the case may be responsible for his patient's hypochondriasis. I believe the fault lies essentially with the patient's premorbid personality. This does not, of course, refer to iatrogenic cardiac neurosis, with which I shall deal later.

When the critical phase is over, and as the period of convalescence lengthens, the hypochondriacal individual becomes progressively more preoccupied with his condition and its daily state. He becomes obsessive in his scrupulous observation of the restrictions imposed on him by his doctors, exceeds them and becomes panicky when he is pressed to relax them. He wants reassurance and has no faith in it, and slowly, or perhaps not so slowly, settles down to the life of an invalid, refusing to test his cardiac capacity, narrowing down his interests progressively until his waking hours are spent in keeping an inventory of his aches and pains and incapacities, and taking medicines because it is part of the day's ritual and not because he has belief in them.

A modification of the above is where there is less obsessiveness and more anxiety accompanying the hypochondriacal preoccupation.

Another mechanism is that of delusion formation, where the affected organ becomes the subject of hypochondriacal delusions. I cannot say that I have seen this type of psychotic reaction in cases of coronary thrombosis, though I have seen coronary thrombosis occurring in patients with hypochondriacal delusions which embrace the heart as well as other organs.

**Depression**

A depressive reaction occurs not uncommonly in association with coronary thrombosis, with the usual features of hopelessness, purposelessness, worthlessness and retardation. Where the depressive reaction has succeeded a period of anxiety and agitation the condition fluctuates between intervals of anxiety and agitation and depression, and in the latter it arouses suicidal reflections and impulses. Delusions of financial ruin and guilt are expressed. The more the depression reflects the precipitation of an endogenous depression the more abundant and bizarre the delusions, and the hypochondriacal delusions especially point to a severe degree of depression which is likely to be endogenous in character.

Early-morning wakefulness in a depressed mood which is at its worst at that time is particularly significant of the transition from an anxiety reaction to a depressive reaction.

I have mentioned the use of ECT in cases of anxiety and agitation; the indication for its employment is even clearer in cases of depression associated with coronary thrombosis, and the benefit is likely to be greater and more lasting to the extent that the depressive reaction is unassociated with anxiety. Unless one bears this in mind, disappointment may be felt that the relief obtained from ECT is not fully sustained. As anxiety is present in most of the psychiatric reactions to coronary thrombosis, the aid to be obtained from ECT may be found to be not fully lasting; but it can be life-saving, and in any case even temporary relief is welcome to the patient. The relapse which may follow the cessation of treatment, after a variable interval, does not, in any case, bring back the patient to the clinical level which existed before treatment.

**Cerebral Symptoms**

The reactions mentioned so far are essentially psychical in character. That is not to say that the cerebral anaemia which results from coronary thrombosis has no part in precipitating or sustaining these reactions, but that they are basically affective responses to an ego-threat. There are, in addition, symptoms which directly
reflect the cerebral anaemia consequent on coronary thrombosis, such as the loss of consciousness or epileptiform convulsions that may accompany the occurrence of coronary thrombosis, or the confusion and intellectual impairment which may be present for a variable interval after the event.

**Aetiology of Coronary Thrombosis**

In the search for the aetiology of coronary thrombosis the personality of its victims has come under suspicion. Generally speaking, they are people who take their responsibilities very seriously, and set themselves high standards of achievement. Their make-up compels them to react to circumstances to establish this disposition. The effect is that they are continually driving themselves, fearful and eventually unable to relax, and anxious with the least obstacle or threat to their self-set programmes. Their interest and energies have one focus and goal, success and security, with the result that they live in the narrow groove of effort, tense and earnest and often humourless. Their mission is a closely guarded secret and they are equally reticent about their anxieties. At what point the coronary attack occurs, and its relationship to the special stress of circumstances, it is difficult to dogmatize about. Physical factors no doubt play their part, very probably the major part, in predisposing the individual to an attack, and in precipitating it. Nevertheless, it is a common observation that financial stress and catastrophe march hand in hand with coronary thrombosis.

**OTHER CARDIAC LESIONS**

The psychiatric reactions that are associated with other cardiac lesions are divisible, like those connected with coronary thrombosis, into two classes; the essentially psychical reactions and essentially organic reactions. To the extent that a cardiac lesion reduces the cerebral blood-supply the resulting psychiatric symptoms are more likely to be of the organic-reaction type than of the non-organic type. That is not to say that cerebral anaemia is not an operative mechanism even when the reaction appears to be essentially psychical, but as the symptoms come to take on more of the appearance of an organic reaction the casual responsibility must lie more with cerebral anaemia.

The organic symptoms are of 3 degrees—acute, subacute and chronic—depending on whether the cardiac lesion has arisen suddenly or insidiously. In addition, and this applies especially to the more chronic cardiac lesions, the symptoms derive in part from the causal lesion and in part from the resulting lesion; as, for example, hypertension and the ensuing cardiac failure. In yet another class of case, that of arteriosclerosis where the arterial changes affect both the coronary and cerebral vessels, the symptoms are based on a cerebral anaemia for which both heart and cerebral vessels are responsible. In arteriosclerotic dementia the heart hardly ever escapes from the arteriosclerotic process.

Marchand (1955) recently made an interesting observation on the occurrence of myocardial infarction in psychotic patients. He said that pain was absent in 82.5% of cases, in sharp contrast to the much lower frequency of painless myocardial infarction in the general population. He gave it as his opinion that the explanation lay in the fact that the meaning of pain was lost to the majority of patients with psychoses, both organic and functional.

Peskin (1955) has recorded a case, which I had the opportunity of examining with him, which cardiologically presented a combination of mitral stenosis and a 3:1 flutter and, from the psychiatric aspect, ‘blackouts’ and depression. As happens so frequently in psychiatric conditions, there were a number of aetiological factors in operation, and so little was the heart condition suspected of responsibility for the ‘blackouts’ and the depression that the former was suspected of being hysterical and the latter endogenous. He was in fact referred to Tara Hospital for shock treatment. A valvotomy did little to change the psychiatric picture, but after non-convulsive cerebral electro-stimulation a normal cardiac rhythm was re-established, and with it the ‘blackouts’ and depression disappeared. This case well illustrates the direct relationship between cardiac lesions, cerebral anaemia and psychiatric symptoms. It also brings to light a new form of treatment for cardiac arrhythmias. Lewis, Richardson and Gahagan (1955), on the other hand, found that nearly 80% of the series of 21 patients with pathological cardiovascular conditions that they treated with electric stimulation developed cardiac arrhythmias of various degrees of severity. Because of the risks involved in the treatment, they gave it under continuous electrocardiography and, in addition to the usual precautionary measures of pre-treatment fast of 3 hours, rapid and short-acting intravenous anaesthesia and scoline, they gave intravenous atropine to block the cardiac portion of the vagus nerve, and either quinidine or chlorpromazine to reduce the treatment-induced extravagal arrhythmias.

With heart failure coming on insidiously the patient may complain of lack of concentration, undue mental fatigability, insomnia, emotional lability, impoverishment of memory. Anxiety, agitation, depressive and paranoid reactions may occur, and dementia may develop. Mayer-Gross, Slater and Roth (1954) mention that hypomanic pictures and delirious states may occur.

**Prognosis**

The prognosis in the psychiatric reactions to cardiac lesions depends on two considerations in the main. First, to the extent that it is based on cardiac pathology, the condition will run parallel to it. Secondly, the endogenous component provoked by the cardiac lesion will run a course that is determined largely by the dynamics that are inherent in it. Roughly speaking, therefore, the more profound the psychiatric disturbance aroused by the cardiac lesion the longer is it likely to endure beyond the time-span of the cardiac lesion itself. Caution is required in predicting the likely progress of a psychiatric reaction to a cardiac lesion that does not quickly clear up, both because it is not easy to assess the unconscious difficulties that have been unleashed nor, I suppose, for the physician, to estimate the danger of recurrence of the cardiac lesion.
An acute confusion developing in the course of chronic heart failure is likely to be a terminal phenomenon.

**Diagnosis**

The diagnosis of the psychiatric reaction, as a descriptive entity, presents little difficulty. The aetiological diagnosis can only be made, with confidence, with the efflux of time and the progress of the cardiac lesion.

Two special circumstances should put the psychiatrist on his guard when he comes to making a diagnosis, and bid him keep in mind the possibility of a coronary thrombosis. One is met with in dealing with an unconscious patient, of middle age and beyond, when what may have the appearance of a cerebral apoplexy may be coronary infarction. The other, which is not infrequently met with in mental hospitals, is a sudden change in the behaviour of a psychotic patient, as, for instance, in the case of the patient who has slavishly and energetically applied himself to certain routine jobs in the hospital and who suddenly prefers to stay in bed, or to remain in the ward instead of going into the farmlands; electrocardiography in such instances would reveal not a few cases of coronary thrombosis (Marchand 1955).

II. **CARDIAC SYMPTOMS IN A NEUROTIC SETTING IN THE ABSENCE OF CARDIAC DISEASE**

**Effort Syndrome**

Symptoms referable to the cardiovascular system are present at some time or another in most cases of anxiety neurosis; yet in civilian life the psychiatrist very uncommonly refers to such cases as effort syndrome. In World War I, however, about 60,000 cases with such symptoms had been diagnosed as effort syndrome in the British Army by the summer of 1918 (Lewis 1940). It was Thomas Lewis who separated this condition from organic cardiovascular disorder, and though he appreciated that there was a psychological factor in operation he believed the condition to be a definite clinical entity. At the outbreak of World War II, in anticipation of the recurrence of the syndrome in war time, an effort-syndrome unit was established within the E.M.S. (emergency medical service) in England, and in the Middle East the British Army set up two effort-syndrome centres. It was the work done in Mill Hill, London, that demonstrated that neither aetio logically nor symptomatically was there any valid ground for applying a special label to symptoms which were psychiatric in origin and were found in a number of different psychiatric conditions. Jones and Lewis (1941) found 100 successive admissions of effort syndrome to be classifiable as follows: anxiety state, acute and chronic, 31%; depression, 12%; psychopathic personality, 18%; hysteria, 11%; post-infec tive, 11%; hyperventilation, 11%; organic disease, 11%; high-grade mental defect, 4%; schizophrenia, 1%. Early in the war High Authority requested that the label ‘effort syndrome’ should be avoided, with the result that the two effort syndrome centres in the Middle East faded out, and effort syndrome became a relic of the past.

The elimination of effort syndrome as a label may not have made any real difference to the number of casualties in the war. Nevertheless, it prevented harmful hospitalization and the creation of neuroticism by suggestion.

In World War I 5/6ths of the sick cases classified as cardiovascular were effort syndrome. In civilian life, according to Paul Wood (1950), approximately 1 in 3 or 1 in 4 patients who present themselves at cardiovascular clinics are suffering from psychoneurosis without organic disease of the heart. It is possible therefore that the elimination of the label ‘effort syndrome’ may have obscured an important fact, namely, that in wartime more symptoms presented by the psychoneurotic patients are referred to the cardiovascular system than in peace time. The inference to be drawn from this is that the special circumstances of war provoke a psychoneurotic response which obtains most dramatic expression via cardiac symptoms. In other words, it is in special measure a war neurosis, and it may be that the Mill Hill team failed to see this angle, when they sought and produced support, in clinical observations and figures, for their view that there was no warrant for the use of the term ‘effort syndrome’. Effort syndrome, it certainly was not, because the reaction could obtain when the subject was at rest and, again, the affected soldier could engage in some energetic activity in which he was interested without getting the symptoms. On the other hand, it was a reaction which obtained prominence during war time and, though there was no constant causal environmental factor to be found in the cases, they all had this in common, that they occurred against the background of war and its multitudinous stresses of fear and of assault on established islets, conventions and dignities. I do not, however, want to labour this question of terminology. What I am anxious to do is to see what light the wartime cases of effort syndrome can throw on the subject of cardiac neurosis, and in particular on the questions: (a) What types of personality develops cardiac neurosis, (b) the circumstances that precipitate it, and (c) why the heart is chosen as a setting for staging the drama of the battle between the psyche and the external world.

There appear to be 3 predisposing factors, viz. (a) a family history of neurosis, psychopathy, insanity and epilepsy, (b) physical inferiority, and (c) a particular kind of psychological make-up.

**Family History.** Wittkower and Rodger (1941) found that 15 of their series of 50 gave evidence of lifelong physical inferiority.

**Physical Inferiority.** Wittkower and Rodger (1941) investigated, he found a positive family-history in about 56%; and a cardiac neurosis was present in about 50% of these. These figures indicate how the individual is prepared by home training for his later maladjusted reactions.

**Psychical Make-up.** Wittkower and Rodger (1941) make the following comments: ‘None of the patients suffering from effort syndrome was found to be emotionally well-adjusted. The vast majority conformed to the obsessional type, possessing an over-keen sense of duty and over-rigid morality’. The effort-syndrome patient is preoccupied with problems of morality, self-regard,
religiosity, patriotism, duty, responsibility and self-sacrifice’. Needless to say, this make-up had made itself manifest in various ways long before the illness of the effort syndrome. Thus Jones and Lewis (1941) found that 50% of their patients ‘had had, often for the greater part of their lives, symptoms of vegetative disorder (such as breathlessness, palpitation, excessive sweating or diarrhoea) on slight provocation of an emotional sort’, 40% of them had not played any games, in many instances, because it upset them'; half had shown neurotic traits, like stammering, bed-wetting, sleep-walking; 'nearly 2/3rds had shown more or less pronounced anomaly of personality—shy, tense, hypochondriacal, sensitive or delinquent'. '67% of the patients had had symptoms of effort syndrome before the war', in many dating from their early school days. Paul Wood (1950) found that 'the most constant characteristic is timidity, which results in lack of confidence, a sense of inferiority and undue apprehension. On such a soil an anxiety state takes root very easily'.

There are two clinical aspects to effort syndrome: the psychic and the psychosomatic. As just indicated, there are a number of psychiatric conditions found associated with the somatic symptoms of effort syndrome. There would thus appear to be two levels of personality predisposition, the one predisposing the individual to a personality disturbance with its specific features, and the other determining reactively for the individual concerned, in both the aetiological and psycho-mechanistic sense, the genesis of psychosomatic symptoms in response to a stressful or traumatic situation.

What determines the somatic manifestations of the effort syndrome? It was mentioned earlier that in 50% of cases there was a family history of cardiac neurosis. This means that the body image borne by these individuals includes a sub-image of dysfunction in certain circumstances. This sub-image is supplemented by further sub-images that are based on the fears and anticipations of difficulty and defeat aroused by any physical defects or inadequacy that may be present or imagined. Because of timidity, for instance, the individual may feel that he is physically unequal to a particular test of physical strength or endurance, and when he is confronted with such a test he reacts with the dysfunction previously rehearsed mentally.

At a deeper level it is probable that complexes are represented in the body image by physiological correlates. Thus the breathlessness of the effort syndrome might be a reflection of the fear of suffocation that lies at the heart of claustrophobia (Wittkowski and Rodger 1941).

In addition to the symptoms that are latent in the body image, others derive from the stimulation or inhibition of the vegetative system by the emotion aroused in a particular situation as, for example, anxiety or depression. How far the body image determines the vegetative expression of emotion and how it is reflexly stimulated by peripheral somatic experience, is a subject that would take me too far afield.

Predisposition, divided into factors, creates the appearance of something formal and static residing within the mind, or perhaps a spring set for action but inactive until it is touched off. But it is more than that. It is also something dynamic, a ferment of conflicts and strivings which keeps changing the formal predisposition and the susceptibilities it imposes on the individual, and shapes the reaction that results from the clash with the environment. The precordial pain of the effort syndrome case may, in the first instance, reflect and represent guilt over aggressive drives and the self-punishment attendant on it, and secondarily, may be unconsciously sustained by the necessity to escape from the painful conflict.

Predisposition, psychical and psycho-physiological, is a pathological mechanism that is evolved under the impact of various influences, from early life, to preserve the infantile ego and, when circumstances arise that challenge this narcissistic ego, the effort syndrome, amongst other reactions, emerges or erupts, according to the violence of the challenge. It is worthy of note that the effort syndrome occurred in relatively few cases of battle exhaustion (1-2%), whereas in non-combat psychoneurosis it occurred in about 20% of cases.

Clinical Features. According to Paul Wood (1950) the chief symptoms are breathlessness, palpitations, fatigue, left inframammary pain, dizziness, excessive sweating and headache; and the most important signs are nervousness and depression, tremor, frequent sighs and hyperventilation, clammy cold hands, tachycardia, raised casual blood-pressure around 160/90 mm. Hg, and inability to hold the breath for longer than 20 seconds.

Diagnosis. The combination of psychiatric and psychosomatic symptoms as a reaction of maladjustment to a situation points unmistakably to the diagnosis. The recognition that it was a psycho-reactive entity led to the abolition of the term 'effort syndrome' in World War II and its replacement by labels descriptive of the psychiatric presentation. In peacetime the effort-syndrome case is apt to be labelled hyperventilation syndrome by the physician and anxiety state or anxiety-hysteria by the psychiatrist.

Psycho-physiological Mechanism. According to Paul Wood (1941) it was Soley and Shock (1938) who first claimed that hyperventilation was responsible for the symptoms of effort syndrome. He reviewed the evidence for the claim and gave it as his considered opinion that when present, and it was present in only 21-5% of his series of 200 cases, hyperventilation 'must aggravae certain of the symptoms. In any case, spontaneous hyperventilation is not a diagnosis, but apart from rare organic causes, merely one mechanism by which psychosomatic manifestations are produced'. There are, of course, other physiological mechanisms for translating emotion into somatic symptoms, which operate more universally. I refer to the action of the pituitary and adrenals, and possibly other endocrine glands, which affect the soma directly or via the vegetative system or the central nervous system, and the action of the thalamus and hypothalamus, which affect the soma via the central nervous system or the autonomic system or the pituitary.

Whatever the route, there is evidence (Dunbar quoting Wolf, 1948) that psychical influences can produce alterations in the rate and size of the heart and, in the
electrocardiogram, that anxiety can produce coronary spasm. It is also believed (ibid.) that coronary spasm or occlusion produces anxiety as a direct response, in the same way that a peptic ulcer, for example, produces pain. The suggestion therefore is that anxiety can produce coronary disease and coronary disease anxiety. Nevertheless, though anxiety can produce coronary disease, there are instances as, for example, in effort syndrome, where anxiety produces symptoms that stimulate coronary disease and yet do not provoke any overt organic change in the coronaries. What is the factor or factors that determine whether anxiety is to produce a cardiac neurosis or a coronary disease? It may be based on a physical factor or factors which are unevenly distributed in the population, or on a personality factor. It is my belief that the predisposed individual who can verbalize his anxiety is likely to develop a cardiac neurosis, whilst the person whose capacity for emotional expression is more inhibited is likely to develop a coronary disease.

Prognosis. The patient with praecordial pain defies persuasion that it has nothing to do with his heart. Today, with the prevalence of coronary thrombosis, his greatest fear is represented by that condition. I have never come across a case of effort syndrome that has developed coronary thrombosis. Grant (1925) in a follow-up study of 601 men who had been discharged from the army in World War I with the diagnosis of effort syndrome, did not find a single case of coronary disease at the end of 5 years after discharge, and not a single case of death on account of coronary disease in the 52 deaths that had occurred.

In Paul Wood's (1941) series of 182 cases 31·1% returned to full duty, 36·3% returned to light duty, and 32·3% were discharged. In Grant's (1925) follow-up series, after 5 years 15·3% had recovered, 17·8% had improved and 56·2% were unchanged. The poor prognosis of cases discharged on account of effort syndrome is in line with the pronounced predisposition that operates in these cases.

III. IATROGENIC CARDIAC NEUROSIS

The number of people who have been led to believe by a medical man that they have a diseased heart, through a misinterpretation of some innocent clinical sign as, for example, a soft systolic murmur at the apex, or by an injudicious comment to a patient regarding his heart, must be legion. The longer they live with that belief and order their daily lives to conform with it, the more difficult it is to shake it. It is extraordinary how often a person accepts the diagnosis of a diseased heart, and the shackles that go with it, without overmuch questioning or resistance. It rather suggests that the person unconsciously desires and seeks a defence through incapacity. However that may be, he certainly resists giving it up. It will be readily appreciated that such a subject is a candidate for the effort syndrome. The more integrated the personality the easier it will be to reassure the individual and persuade him to cast aside the illness that his medical attendant has unwittingly and unintentionally brought to him.

SUMMARY

Two psychiatric situations are associated with the heart, the one connected with organic heart disease and the other occurring in its absence. In the former the symptoms, which may be psychoneurotic or psychotic, are precipitated by the anxiety aroused or by cerebral anaemia, or both. Predisposition in the pre-morbid personality is a contributory factor. ECT is advocated for agitation and depression associated with coronary thrombosis; it can be life-saving.

Anxiety may cause coronary spasm and ultimately disease, but the effort syndrome case never goes on to coronary disease. Apart from physical factors the psychical make-up may determine whether anxiety ends in coronary disease or effort syndrome.

REFERENCES

Grant, R. J. (1925): Heart, 12, 121.

UNION DEPARTMENT OF HEALTH BULLETIN

Plague: Nil.
Smallpox, Transvaal: One (1) European case in the Nelspruit district.
Typhus Fever, Natal: Two (2) European cases in the Port Shepstone municipal area. Diagnosis confirmed by laboratory tests. Orange Free State: One (1) Native case in the Thaba Nchu district. Diagnosis confirmed by laboratory tests. Cape Province: No further cases have been reported from the Glen Grey district since the notification of 1 December 1955. This area may now be regarded as free from infection.
Epidemic Diseases in Other Countries.
Plague: Nil.
Cholera in Chalna, Dacca (Pakistan).
Smallpox in Herat, Kabul, Kandahar (Afghanistan); Rangoon (Burma); Phnom-Penh (Cambodia); Ahmedabad, Allahabad, Delhi (India); Dacca, Lahore (Pakistan).
Typhus Fever in Cairo (Egypt).