reasons for this view, apart from the lack of alternative facilities, and they may be summarized in the statement that some patients have no home, many have no wish to leave, and all in greater or less degree show the mark of the inadequate resources to which they have had access. These facts do not reflect on the hospitals themselves, for too many patients, too few staff, and hopelessly inadequate facilities were not problems which could be solved at the level of the individual hospital. For these reasons we must plan chiefly for future admissions.

Without attempting to give precise figures, we may safely conclude that a considerable proportion of patients of the type now cared for in hospital could in future be accommodated elsewhere if the necessary services were available, and it is evidently the intention of recent Acts of Parliament that this shall be the responsibility of local authorities. To what extent it can be met by complementary home services cannot be known without an examination of the circumstances of patients waiting for admission to hospital, followed by a period of experience in the organization and accountancy of such services. It is at least fairly certain that for some time to come institutions will be needed which add simple nursing and occasional medical attention to the usual resources of the municipal home for the aged.

If relieved in this way of all cases which need never enter hospital, and if permitted to discharge their patients at the earliest moment after completion of treatment, regional hospital boards can re-examine more realistically the proposal that they should in time care for the chronic sick in wards of general hospitals. Alarming as this suggestion is if 2-2.5 beds are needed for every thousand of the population—that is, about one-third of the beds in general hospitals—the situation is quite different if, as our investigation suggests, not more than one-tenth of the total accommodation would suffice. (Separate arrangements would of course be needed for the considerable number of patients who require supervision because of their abnormal mental state.) The advantages to the elderly patient of being received in the acute ward, where modern resources in treatment are available, have always been obvious; the advantages to hospital authorities in easing the most thorny of their staffing problems are hardly less important.

Summary

In Birmingham approximately twelve persons in every thousand aged 65 and over are in hospitals for the chronic sick.

On the assumptions that only patients in need of frequent medical attention or skilled nursing should be in general hospitals, and that patients in need of supervision because of their mental state should be in some form of mental institution, disposal indicated by current requirements of 1,005 patients in Western Road Infirmary, a hospital for the chronic sick, is as follows:—Under regional hospital boards: (a) one-fifth in general hospitals, (b) one-fifth in some form of mental institution; under local authorities: (c) three-fifths divided between (i) their own homes, and (ii) other institutions offering domestic services, simple nursing, and occasional medical attention.

The adequacy of the services received by patients in Western Road Infirmary was assessed in relation to the question of admission, investigation, continence, mobility, and mental state: (a) three-quarters of the patients were admitted direct from their homes, and two-thirds had not been investigated before admission; (b) about one-third of the patients were incontinent—in many cases the incontinence was not explained by the diagnosis or the mental state of the patient; (c) 58% of patients were bedfast, and a further 17% could be moved to a chair at the side of the bed; (d) about one-third of all patients were abnormal mentally, but only about one-fifth to a degree necessitating constant personal supervision.

Improved services will no doubt reduce the number of patients incontinent or immobile, but will be unlikely to alter substantially the proportion requiring supervision because of their mental state.

We have pleasure in acknowledging our indebtedness to Professor A. P. Thomson, who has contributed to this survey in many ways. The medical records were completed by Dr. Charmian Elkes, Dr. S. E. Hallissy, and Dr. P. M. Meynell, and the social records by Mrs. L. Leaver and Mrs. H. Pringle. The inquiry owes much to their work and to the generous co-operation of Dr. L. Nagley and his staff at Western Road Infirmary. We are also indebted to Miss Frew and to those members of the Almoner's Department of the Birmingham United Hospitals who assisted in a preliminary pilot survey. Miss Catherine Wall prepared the diagrams in this paper.

REFERENCES

- Affleck, J. W. (1947). *Lancet*, **1**, 355.
 Amulree, Lord, and Sturdee, E. L. (1946). *British Medical Journal*, **1**, 617.
 Cosin, L. (1948). *Proc. R. Soc. Med.*, **41**, 333.
 Greenwood, J. M. (1949). *Lancet*, **2**, 1047.
 Howell, T. H. (1948). *British Medical Journal*, **2**, 1029.
 Lowe, C. R., and McKeown, T. (1949). *Brit. J. soc. Med.*, **3**, 110.
 — (1950). *Ibid.* In press.
 Ministry of Health (1945). *Hospital Survey for the London and Surrounding Areas*. H.M.S.O., London.
 — (1945-6). *Hospital Surveys*. H.M.S.O., London.
 Thomson, A. P. (1949). *British Medical Journal*, **2**, 243, 300.
 Warren, M. W. (1948). *Geriatrics*, **3**, 42.

THE SIMULATION OF HEART DISEASE*

BY

A. RAE GILCHRIST, M.D., F.R.C.P.

Physician, Royal Infirmary, Edinburgh
 Reader in Clinical Cardiology, Edinburgh University

"The heart is deceitful above all things . . . who can know it?"—Jeremiah xvii. 9

The manifestations of organic heart disease are numerous and widespread. When symptoms are confusing and diagnosis is difficult we must not too readily assume that ankle oedema is necessarily of cardiac origin or purpuric spots embolic, any more than that a precordial pain is necessarily of coronary origin or a systolic murmur the product of endocarditis. Symptoms and signs, including radiological and cardiographic, may each in turn mislead.

Ability in differential diagnosis depends on a sound working knowledge and a familiarity with related possibilities, not merely gained in the seclusion of the hospital ward and academic library but reinforced with advantage by personal endeavour and responsibility won in the hard school of private practice. The course of events, if followed with accuracy even for a short space of time, will often provide a diagnostic clue denied to the isolated observations of a single opportunity. Time, in other words, simplifies the problem while it lasts.

Our subject requires to be defined and discussion limited to certain aspects of differential diagnosis. A simulation being a pretence of what is not, consideration must therefore be restricted to those conditions which mimic or are confused with organic heart disease when, in fact, the heart is healthy. There are many conditions which fulfil these requirements. A dissimulation, according to the dictionary, is a concealment of what is. However tempting it may be to include this aspect of diagnosis—namely, the concealment of organic heart disease by symptoms or signs arising elsewhere, as, for instance, left ventricular failure in the

*Read in opening a discussion in the Section of Cardiology at the Annual Meeting of the British Medical Association, Harrogate, 1949.

course of acute Bright's disease—we must keep closely to our title and avoid confusing two distinct entities. Our subject is therefore restricted to those conditions in which, for one reason or another, organic heart disease is assumed to be present when in reality it is not.

Misinterpretation of Symptoms

Difficulties arise in various ways. Perhaps the commonest error is a misinterpretation of symptoms. Most heart disease is imaginary (Cabot, 1926), and unless close attention is given to the character of the complaint, its development and course, the functional can readily be confused with the organic. Both may intermingle in varying proportions.

In forming a judgment from a history the precise description must be extracted from the sufferer by the doctor himself. The personal interview conveys an understanding which no written words can describe. This is always important, but never more so than in cardiology, where an estimation of the efficiency of the heart is assessed on the patient's account of his capabilities, and where on occasions the diagnosis—for example, angina pectoris—is established by this means alone. The doctor must free his mind of prejudice and preconceived diagnostic notions and set about giving attention to the details of the history of the present illness, which only the sufferer can describe. It is a good rule, occasionally forgotten, to base a differential diagnosis on the patient's leading symptom.

Pain

As an example, we may consider pain as a symptom likely to give rise to confusion. The pain of organic heart disease has certain well-known characteristics which are commonly mimicked by a variety of lesions above and below the diaphragm. It is, for instance, notorious how gall-bladder pathology can lead to incrimination of a healthy heart. Sternal pain, sudden in onset and followed promptly by breathlessness and shock, is too readily assumed to be of a coronary origin. When the attack has occurred some time in the past, even a few weeks previously, the diagnosis may be more difficult on account of the patient's inability to supply important details.

If, for instance, the attack has been preceded for some months by a sense of abdominal distension after meals and is accompanied or followed by a shiver, by pain in the tip of the right shoulder, or by moisture at the base of the right lung, a gall-bladder lesion is more likely than a coronary occlusion. The absence of jaundice does not help in the differential diagnosis, as it is the cystic-duct stone which gives rise to most difficulty in separating these two conditions. Recurrent attacks, similar in pattern, without a sustained fall in blood pressure, without an electrocardiographic sequence, and even without positive cholecystograms, will favour a cause other than coronary, and most commonly a cholesterol stone in the neck of the gall-bladder.

Even after removal of the gall-bladder recurrent precordial pain does *not* exclude a biliary cause. If attacks of sternal pain in these circumstances occur at longish intervals, a hint of jaundice appears sooner or later as a result of oedema of the common duct, in which the surgeon will find inspissated bile, gritty material, or even a stone that may have lain overlooked since the previous exploration. A history misinterpreted as a result of the omission of essential details causes great confusion. To witness the attack of pain is of course more helpful, but mistakes are bound to occur, particularly as both conditions occasionally coexist.

Of the acute conditions causing confusion a perforated peptic ulcer is well known. The suddenness of the attack, the severity of the pain, its midline distribution, and the subsequent development of shock with some degree of upper abdominal rigidity are features common to both conditions. Unnecessary exploration of the abdomen is now less frequent than formerly: in fact, the tendency is perhaps to err in the opposite direction. Duodenal ulceration and coronary disease can coexist, and when acute symptoms arise it can be excessively difficult to separate one from the other: electrocardiograms may establish the latter diagnosis; radiology of the abdomen, by conclusively demonstrating a cap of gas below the dome of each diaphragm, may confirm the former diagnosis.

Massive Pulmonary Embolism

With acute conditions in mind it is necessary to refer briefly to pulmonary embolism, as this condition is commonly mistaken for a coronary thrombosis. It is true that the right side of the heart takes the burden in a massive embolization of the lung, but acute cor pulmonale is a temporary derangement of function, and, if the patient survives, it is satisfactory to have the justification of assuring him that irreversible cardiac damage has not necessarily been sustained.

The distinction between a massive pulmonary embolism and an acute coronary occlusion can often be made immediately at the bedside without instrumental aid. Contrasting clinical features are shown in the accompanying Table, but

Table Showing Contrasting Symptomatology in Massive Pulmonary Embolism (Acute Cor Pulmonale) and Acute Coronary Thrombosis

	Massive Pulmonary Embolism	Acute Coronary Occlusion
Preceding state . .	Convalescent; commonly confined to bed; often post-operative	Ambulant; active
Onset	Abrupt and maximum	Developing rapidly to climax, often in waves
Character of pain	Oppressive, suffocating, overwhelming, occasionally pleuritic	Crushing, tearing, constrictive, vice-like
Site	Widespread, across front of chest, occasionally lateral	Central, retrosternal, occasionally epigastric
Radiation . .	Not characteristic; occasionally to shoulders	Upwards to neck and jaws; downwards to arms and hands
Dyspnoea . .	Sudden, intense, gasping	Generally mild
Attitude . .	Often orthopnoeic, cyanosed, sweating	Restless, less cyanosis, rolling about
Shock	Immediate	Develops later
Local phlebitis .	Often tender calf or palpable vein	None

the suddenness of the attack, the degree of shock, the fall in pressure, the leucocytosis, and the increased sedimentation rate are common to both conditions (Roth, 1948).

Pulmonary infarction is readily confused with a myocardial infarct involving the posterior and diaphragmatic wall of the heart as is shown in the electrocardiograms. These must be interpreted with care. The most striking difference is the transient distortion of pattern associated with the pulmonary infarct and the rapid restoration of more normal curves in the course of two or three days, whereas the changes observed after a myocardial infarct pass through successive stages and may persist for months or years.

Further difficulties arise if pericardial friction is detected. After a lung embolism friction over the base of the heart has been attributed by White (1944) to the pressure exerted by the stretched and bulging pulmonary artery against the pericardium underlying the sternum. The detection of pericardial friction over the front of the chest is not a feature of the isolated and uncomplicated posterior myocardial infarct.

When it does occur in such circumstances it suggests either an extension of the infarct to involve the anterior wall of the heart or a revision of the diagnosis in the light of the possibility of a major pulmonary infarct. The

pericarditis of an anterior myocardial infarct is commonly first detected towards the apex, in contrast to the pericardial friction of the pulmonary embolism, which is occasionally heard over the base of the heart.

My justification for emphasizing the distinction between these two similar conditions is experience of the relief it has been to certain patients to know that they have not sustained the cardiac damage at first suspected.

As the diagnosis of angina of effort depends primarily on a study of painful experiences, mistakes can readily occur. If the patient can take a brisk walk in cold weather without experiencing pain or distress in the chest, it is most unlikely that he suffers from

angina. On the other hand, when discomfort is experienced only in the elbow, a referred pain arising from an arthritis in the left shoulder-joint, irritated by movement of the arm on walking, is a more likely explanation than coronary disease. Discomfort in both hands and in the epigastrium experienced during or after a meal, perhaps aggravated by exertion, is also not so uncommon in the presence of a peptic ulcer of the lower end of the oesophagus—a condition recently discussed by Allison (1948).

Anxiety States

Difficulties also arise as a result of a failure to sift the functional from the organic manifestations of heart disease. Careful attention to the patient's description of his symptoms is most important. The apical pain, the emphasis on fatigue, the obvious nervous instability, the multiplicity of his complaints, his demeanour and obvious apprehension, all go to make a picture in sharp contrast with that which we associate with the more apathetic attitude of the genuine sufferer from organic heart disease. It is, alas, only too true that a large proportion—upwards of 50%—of neurotic cardiac patients are "doctor-made" (Hill and Dewar, 1945; Hill, 1946), dating their disability to injudicious advice or restrictions imposed after an inadequate and unconvincing medical examination.

The situation is complicated further by the fact that the organic and the functional may coexist. The differentiation between those symptoms which are the direct outcome of the organic disease, such as the exertional dyspnoea, and those which are merely the expression of the associated emotional upheaval, such as the periapical, axillary, and even brachial discomfort, requires the utmost skill and judgment on the part of the physician, particularly in relation to treatment. It is necessary to reassure, restrain, and at the same time steer a middle course.

It is also well to recognize that, as pointed out by Weiss (1932), temporary or sustained emotional tension, not unlike physical strain, represents a burden on the cardiovascular system, as a result of which pathological processes may develop or become unmasked. After years of semi-invalidism the cardiac neurotic gets a coronary thrombosis which may well confuse and mislead his doctor. I have been impressed by the frequency with which organic vascular disease, coronary thrombosis in particular, may follow on protracted emotional disturbances in the younger age groups.

Misinterpretation of Physical Signs

Just as a symptom may mislead, so also certain physical signs, notably the heart sounds, are readily misinterpreted. The systolic murmur has long given rise to difficulty. Its importance as a manifestation of organic heart disease has undoubtedly been overemphasized in the past. At all events most men are now on guard when faced with the systolic murmur as a solitary finding and rightly hesitate to incriminate the heart. On the other hand, the significant varieties of gallop or triple rhythm are less easily appreciated by those who continue to concentrate solely on the presence or absence of murmurs and not on the equally important character of the heart sounds. Great help is to be had in making a diagnosis of heart disease by consideration of the nature of the sounds themselves, and particularly with regard to the analysis offered by Evans (1948) of the different types of triple rhythm.

Mediastinal Emphysema.—Mediastinal emphysema, originally described by Hamman (1937), is a curiosity which gives rise to crunching, clicking sounds, synchronous with the heart beat and sometimes loud enough to be heard across the room. The fault is attributable to the tracking of air from a ruptured

Electrocardiograms recorded within a few hours of the onset of a massive pulmonary embolism (A), and a posterior myocardial infarct (B). From above downwards are depicted the three standard limb leads and the serial precordial leads from across the chest, beginning with V1 (taken at fourth right intercostal space close to the sternum) and V6 (fifth space in mid-axillary line). The Q waves and inverted T waves in Lead III are similar features, but the presence of S1 and T inversion in the first three chest leads, overlying the right ventricle, is characteristic of pulmonary infarction and serves to distinguish the two conditions. Furthermore, there is notable depression of the S-T segments of V3, V4, and V5 in the records taken after the myocardial infarct.

lung bulla along the pericardial folds and into the mediastinum. It is an innocent condition unaccompanied by cardiac damage. Similar systolic clicking sounds in the presence of a shallow left-sided pneumothorax have been described by Scadding and Wood (1939). All my five cases were males between 22 and 33 years of age. None had organic heart lesions, although each was suspected to be a victim of serious cardiac disease.

Hypotension.—The clinical features of coronary thrombosis are now so well known, and so much emphasis has been put on the subsequent development of a fall in blood pressure, that the detection of hypotension in an obscure malady, characterized perhaps by exhaustion and bodily weakness, may arouse in the mind of the doctor a suspicion that a "silent" coronary thrombosis may perhaps account for the illness. Influenzal debility, low-grade sepsis, and any protracted infection are much more likely causes for hypotension in such circumstances than a coronary occlusion. This is particularly true if the heart is slow, the voice is strong, and the lung bases are free of moisture. Low blood pressure, with systolic and diastolic readings in proportion, is not a sign of organic heart disease. On the other hand, a low systolic pressure with a relatively high diastolic pressure may well be an indication of left ventricular weakness. In this connexion it is equally justifiable to emphasize that high blood pressure does not necessarily imply organic heart disease.

Oedema.—Ankle or sacral oedema should not necessarily incriminate the heart unless a proportionate degree of dyspnoea is also present. A patient, for instance, may complain of fatigue and exhaustion and show on examination a trace of oedema about the feet and ankles. If he is not also breathless, or rendered breathless by lying flat, the oedema is not directly of cardiac origin and nephritis, varicose veins, phlebitis, and anaemia must be considered. Knight (1940) described a form of soft pitting oedema, extending sometimes from the feet to the thighs, in individuals, commonly women, who have spent a night in a deck-chair in an air-raid shelter. The oedema was attributed in part to temporary pressure on the thighs by the wooden cross-piece of the chair obstructing the venous return and in part to local thrombosis. A similar form of oedema is seen during convalescence from various debilitating illnesses and also in healthy people who have been obliged to adopt a sitting posture for long hours at a time, as in long journeys by car or plane.

Less common and more difficult to understand is that variety of oedema which takes a premenstrual form and which may be confined to the feet and ankles, though the hands and eyelids are occasionally involved (Sweeney, 1934). It is usually associated with salt retention, and is commonly accompanied by fatigue and irritability and occasionally by neurotic symptoms. A genuine cardiac oedema does not display a cyclic rhythm.

In general, it may be said that when the nature of the illness is obscure a single physical sign in the circulatory system seldom justifies in itself a cardiological diagnosis. Heart disease of organic origin almost invariably conforms to well-defined patterns and, with the exception of angina, is accompanied by multiple signs dovetailing to make a distinct entity. When these are absent it is rash to incriminate the heart. Similarly, when cardiac signs which cannot be reconciled with the patient's most prominent symptoms are present, it is unlikely that heart disease is the immediate cause of the presenting illness.

Avoidance of Errors.—As already emphasized, an adequate history of the development and course of the illness is the first essential. Breathlessness on exertion is the almost invariable symptom of a diseased heart. Hesitate therefore to condemn a heart unless this symptom has been experienced on an amount of exertion customarily undertaken without distress. To obtain a measure of its severity it is useful to have the patient compare his present or recent capacity for exercise as restricted by breathlessness with that of a month or a year previously. Thus is determined the rate of decline in efficiency of the heart. Even in angina pectoris exertional dyspnoea is usual, if not in the actual attacks of pain, then on minor amounts of exertion between the acute phases, and even often before the more

severe handicap first developed. It is true that other symptoms—palpitation, exhaustion, faintness, or actual syncope—may occur in organic heart disease, but without effort dyspnoea, retrosternal pain, or distinctive physical signs they count for little.

Personality Factors.—Errors will be avoided by taking into account the nervous temperament and emotional outlook of the individual. Almost automatically the doctor comes to assess his patient in broad terms of nervous stability and personality. By so doing it is often possible to gauge with greater accuracy the severity of the organic handicap. Allowance must be made for the patient's reliability as a witness, his tendency to minimize, exaggerate, or unintentionally distort his symptoms. We must therefore be prepared to take into account his emotional make-up, mental balance, and susceptibility to pain and distress when we consider his general physical state, constitution, and body build. By so doing the error of confusing extraneous factors with inherent organic cardiac damage will be more readily avoided.

General Physical State.—The weight of the patient is of great importance. It can be measured and compared with standard tables. Before condemning a heart as unsound it is wiser to explain that a man is too heavy and, in simple phraseology, "overloading the engine" rather than to burden his mind with such a mischievous and spurious diagnosis as a fatty heart. The mechanical handicap of obesity must be assessed before assuming that the heart itself is diseased. In other words, the significance of exertional dyspnoea is all the greater if body weight is less than the standard for sex, age, and height.

Anaemia is a constant source of difficulty. As in the case of obesity, fewer errors will be made when haemoglobin readings are determined and the defect recorded quantitatively. Breathlessness, fatigue, and retrosternal pain on exertion, indistinguishable from angina of coronary disease, are well-recognized symptoms of anaemia, and such signs as malleolar oedema, cardiac enlargement, tachycardia, and murmurs, systolic and occasionally diastolic, can all add to the confusion. Haemoglobin estimations can be made in a couple of minutes, the appropriate treatment ordered, weeks or months of invalidism avoided, and a heart acclaimed as healthy. If the blood is not examined any form of anaemia, including leukaemia, can present as angina of effort and the heart be condemned unjustifiably.

Pyrexia often consorts with anaemia and can give rise to difficulty. It is wise not to condemn the heart as the site of the trouble until other possible causes for pyrexia are excluded. Positive blood cultures are not synonymous with bacterial endocarditis. If there is a doubt regarding the presence of a valvular or congenital flaw, bacterial endocarditis is most unlikely, as it occurs in a heart already damaged.

Organic Signs.—Errors will also be avoided by accepting as conclusive evidence of cardiac damage only well-recognized signs of organic heart disease. Admittedly, the diagnosis of angina pectoris is often perforce made on symptoms without a demonstrable structural flaw, but for the most part heart disease is accompanied by precise signs in the cardiovascular system. An enlarged heart is a diseased heart. Its size can be determined by palpation. The feel of the heart in action, the accurate location of the apex beat, its thrusting character in left ventricular enlargement, the tapping quality of the enlarged right ventricle, thrills and rough systolic murmurs, or a soft diastolic murmur at the fourth left interspace should leave no doubt in the mind of the clinician. Certain irregularities—extrasystoles and sinus arrhythmias—can be discarded as diagnostic trash, but fibrillation, paroxysms of tachycardia, and heart-block point to structural flaws. A gallop rhythm, muffled sounds, friction, and pulsus alternans, recognized by auscultation in the course of the determination of blood pressure, are clues to organic damage not readily dismissed.

Aetiological Factors.—Finally, it is a good principle to hesitate to condemn the heart as the site of a disease process without a well-established aetiological factor appropriate to the age group. Congenital flaws, rheumatic infection, luetic disease,

thyrotoxicosis, hypertension, vitamin deficiencies, and degenerative changes are distinct entities. Imaginary toxæmias from teeth and tonsils and from bowel and appendix can now be less readily accepted, as aetiological standards become more exacting and the pattern and course of organic heart disease are appreciated with greater precision. When a critical survey fails to solve the problem, resort must be made to supplementary methods of investigation. Unfortunately, radiology, basal metabolism, blood chemistry, blood pictures, sedimentation rates, and electrocardiograms are often equivocal when their help is most needed, and the final diagnosis then depends on a review of the whole situation helped by the clearer pattern which becomes apparent with the passage of time.

It is therefore reasonable to conclude with Parkinson (1924) that if we get no history of an illness which can produce heart disease, no characteristic symptoms, no age changes, and no signs of the kind ordinarily found on examination, the likelihood of a primary cardiac cause is remote and we are well on the way to the diagnosis of a healthy heart.

Cardiovascular Borderland

It must be admitted that from time to time hard-and-fast decisions are difficult to reach, particularly when the illness consists of one or more isolated episodes, sometimes interspersed at long and irregular intervals in an otherwise healthy subject.

Syncopal Attacks

Faints and fits of various kinds are the outward manifestations of the close linkage between the heart and the brain—a union reinforced by the autonomic nervous system, whose regulator mechanisms are easily disturbed.

The simple syncopal attack, related almost invariably to the erect posture and precipitated by some disagreeable experience, the syncope of a hypersensitive carotid sinus, reproduced by artificial stimulation of the bifurcation of one or other carotid artery, and syncope resulting from postural hypotension are not primarily of cardiac origin. In each instance the attack develops gradually with a train of symptoms leading to loss of consciousness, and recovery ensues gradually from the moment cerebral blood flow is restored by the correction of the gravitational disturbance. These attacks are unassociated with an acute sense of rotation, which points to a vestibular disturbance.

On the other hand, the syncope of the Stokes-Adams seizure, a genuine cardiac cause of loss of consciousness, is abrupt in its onset and as a rule almost equally abrupt in recovery. A diagnostic point of some consequence is the facial flush which accompanies the restoration of ventricular beating. The total arrest of the circulation, the absent pulse and silent heart during the attack, and the facial flush which heralds recovery are not witnessed in other forms of syncope. Hesitate to concur in a diagnosis of senile epilepsy, hypertensive encephalopathy, or even hysteria in the middle-aged or elderly without attention to these points. Furthermore, the Stokes-Adams seizures are not confined to complete heart-block, as normal rhythm may be present in the intervals between the seizures (Gilchrist, 1934, 1937).

Gowers's Syndrome

The term "heart attack" covers a multitude of sins, some of which are easily unravelled, but one condition worthy of exclusion from the realm of organic heart disease is that described by Gowers (1907) and attributed by Ryle (1934) to a "vaso-medullary storm." The attack lasts for 10 to 30 minutes and is dominated by a sense of impending death, associated with utter powerlessness, minor precordial aches, a trifling degree of breathlessness or choking, with tingling in the hands or feet. Facial pallor,

cold perspiration, and the appearances of grave illness suggest superficially a serious situation, but the heart is undisturbed, consciousness is never lost, and recovery is gradual without convulsions and is complete in half an hour or so. It is usually possible to justify an optimistic outlook and exclude any organic cardiac cause.

The Heart in Pregnancy

The borderland between the pathological and the physiological is well illustrated by a study of the heart in pregnancy. Here the gain in body weight, the augmented blood volume, the fistulous network at the placental site, and the demands of the foetus result in an increased volume output of the heart, a greater respiratory ventilation, and an augmented oxygen consumption. A burden is thus imposed on the healthy heart, which reaches a maximum about the thirty-sixth week.

When it is appreciated that in her circulatory functions a healthy pregnant woman at rest behaves similarly to a non-pregnant woman engaged in moderate work (Widlund, 1945) it can be understood how breathlessness on effort may be noted during the later months. Some perfectly healthy women during pregnancy are a trifle orthopnoeic and may develop a few scattered rales towards the lung bases; a few may even show a more complete picture of congestive heart failure, with oedema of the lumbar region (a sign more common with hydramnios) and of the feet and ankles. A systolic apical murmur adds to the difficulties. In these circumstances radiology does not readily exclude mitral disease: in fact, the prominent left auricle common in pregnancy may suggest mitral stenosis when in reality no valvular flaw exists (Hollander and Crawford, 1943).

If conclusive auscultatory signs of valvular disease are consistently lacking, if the oedema by its extent is not supported by a proportionate degree of exertional dyspnoea, if the moisture at the lung bases clears on deeper breathing, and if the venous pressure in the neck veins is not obviously increased, a primary cardiac cause for the apparent congestive failure is unlikely.

Gammeltoft's (1928) suspicion that the circulatory adjustments in a healthy pregnant woman could so mimic heart disease as to cause confusion has been confirmed by personal experience at a cardiac clinic in an obstetric hospital where 37 of 352 consecutive pregnant women with alleged signs of cardiac disease were found some years after their delivery to present no clinical or radiological evidence of organic heart disease (Gilchrist and Haig, 1949), and this despite the fact that 18 had shown while pregnant peripheral oedema with dyspnoea sufficient to justify rest in bed for several weeks at a time. In two women the "congestive failure" had warranted caesarean section with sterilization. On subsequent examination some years later both women were found entirely free of organic heart disease and both were in perfect health.

In these particular circumstances it was probably wise to err on the safe side, but it was even more satisfactory to be able to reassure these women that the circulatory disturbances of pregnancy had been temporary and that organic heart disease did not in fact exist.

Summary

There are many conditions which mimic or are confused with organic heart disease when, in fact, the heart is healthy.

Difficulties in diagnosis commonly arise through a misinterpretation of symptoms, pain being a frequent source of confusion. Close attention to the patient's own account of the details of his difficulties is most important if his heart is to be exonerated.

The distinction of gall-bladder lesions from cardiac damage may be exceedingly difficult, particularly in the first attack. On account of the comfort and reassurance which come from the knowledge that irreversible cardiac damage has not been sustained, if the acute phase of a massive pulmonary embolism is survived it is always worth while to endeavour clearly to distinguish this condition from an acute coronary occlusion, which it can on occasions mimic closely.

Difficulties also arise through the misinterpretation of certain physical signs. Apart from those arising locally in the heart, such as systolic murmurs and extraneous noises, hypotension and oedema can give rise to confusion, particularly when the exact nature of the illness is obscure.

Errors in diagnosis can be lessened by assessing the symptoms with care, checking the body weight against standard tables, estimating the haemoglobin level, considering the personality of the patient, and accepting as evidence of organic cardiac damage only well-recognized and indisputable physical signs.

There is, however, a cardiovascular borderland where physiological adaptations and pathological change overlap. The heart in pregnancy is an example. Without close scrutiny of the heart and careful assessment of the degree of exertional dyspnoea in relation to the degree of peripheral oedema, both of which are common in the later months of pregnancy, the ranks of those women known to suffer from rheumatic heart disease may commonly be increased by at least a fictitious 10% in whom the heart is fundamentally sound. On occasions it may be necessary to wait until after the confinement before a final opinion can be expressed regarding the state of the heart, so closely can the temporary adaptations of pregnancy mimic rheumatic heart disease.

It is a good rule never to condemn a heart without ample and conclusive proof.

I am indebted to my colleague, Dr. I. G. W. Hill, for the set of electrocardiograms recorded after a massive pulmonary infarct.

REFERENCES

- Allison, P. R. (1948). *Thorax*, 3, 20.
 Cabot, R. C. (1926). *Facts on the Heart*. Philadelphia.
 Evans, W. (1948). *Cardiology*. London.
 Gammeltoft, S. A. (1928). *Surg. Gynec. Obstet.*, 46, 382.
 Gilchrist, A. R. (1934). *British Medical Journal*, 1, 610.
 — (1937). *Ibid.*, 1, 203.
 — and Haig, D. C. (1949). *Edinb. med. J.* In the press.
 Gowers, W. (1907). *The Borderland of Epilepsy*. London.
 Hamman, L. (1937). *Trans. Ass. Amer. Phys.*, 52, 311.
 Hill, I. G. W. (1946). *Edinb. med. J.*, 53, 485.
 — and Dewar, H. A. (1945). *Lancet*, 2, 161.
 Hollander, A. G., and Crawford, J. H. (1943). *Amer. Heart J.*, 26, 364.
 Knight, B. W. (1940). *British Medical Journal*, 2, 610.
 Parkinson, J. (1924). *Canad. med. Ass. J.*, 14, 221.
 Roth, I. R. (1948). *Amer. J. Med.*, 4, 493.
 Ryle, J. A. (1934). *British Medical Journal*, 1, 89.
 Scadding, J. G., and Wood, P. (1939). *Lancet*, 2, 1208.
 Sweeney, J. S. (1934). *J. Amer. med. Ass.*, 103, 234.
 Weiss, S. (1932). In *Emanuel Libman Anniversary Volumes*, 3, 1181.
 White, P. D. (1944). *Heart Disease*, 3rd ed. New York.
 Widlund, G. (1945). *The Cardio-pulmonal Function in Pregnancy*. Uppsala.

A scheme for smallpox treatment is suggested by Johnstone and Fluker in the *Monthly Bulletin of the Ministry of Health and the Public Health Laboratory Service* (1949, 8, 188). They suggest that in confluent and haemorrhagic cases penicillin, in doses of not less than 0.5 mega unit daily for four days, should be given within 24 to 48 hours of the appearance of the focal eruption. When the vesicles become umbilicated, intramuscular streptomycin should be given in a daily dose of 1 g., and the daily amount of penicillin should, if necessary, be increased to 2–5 mega units. If the case is severe streptomycin should be continued for about a fortnight, or until the lesions have become well dried. The penicillin should be administered until most of the scabs have separated, though there can be no precise rule on this point. This scheme was put into operation by the authors for three cases. The effect on the skin lesions was remarkable, and far less scarring resulted than would have been expected.

PAIN IN THE CHEST WALL SIMULATING HEART DISEASE

BY

D. RHODES ALLISON, M.D., M.R.C.P.

Consulting Physician, Withington Hospital, Manchester

Pain in the chest offers a wide field in differential diagnosis, and is particularly interesting to the clinician because in the majority of cases simple clinical methods are sufficient to establish its origin and nature. In spite of this, mistakes are frequent, and 10% of patients referred to hospital with a diagnosis of angina are found in fact to be suffering from pain arising in the chest wall only, with no evidence whatever of underlying cardiac disease. If the patients sent for electrocardiography on the suspicion of cardiac disease are included this percentage is very much higher. Many of these have been induced to give up their employment or been relegated to a life of invalidism, and in all of them the diagnosis has caused unnecessary anxiety.

The frequency with which such patients are seen in routine out-patient work emphasizes the need for a reorientation towards pain in the chest, and suggests that in clinical teaching pride of place is too often given to angina pectoris in explanation of the pain and too little regard is paid to local structural causes.

When the pain is specifically related to exertion it too frequently breeds in the mind of the patient or his doctor the thought of cardiac disease, particularly in over-anxious individuals and in middle-aged men and women who have some other symptom such as breathlessness to act as a pointer in this direction. In a proportion of such cases organic disease of the cardiovascular system can be excluded by a sufficiently detailed history and a careful examination designed not only to establish the normality of the heart but also to discover some local cause to account for the symptoms.

This paper is based on a follow-up of 50 consecutive cases to which a diagnosis of coronary disease had been wrongfully applied, and in each case the cause of the pain was found to be in the structures of the chest wall. It takes no account of the many cases in which coronary disease is simulated by visceral disease elsewhere or is an expression of an underlying anxiety neurosis.

In almost all the cases the mistake in diagnosis had been due to the readiness with which precordial pain on effort had been accepted as indicating heart disease without realizing the need for the closest questioning on the time relationship between the effort and the pain and the exact nature of the effort involved.

The cases under review were classified as follows: (1) muscle strain affecting the chest wall and attachments of the diaphragm, 16 cases; (2) fibrositis of the chest wall, 20 cases; (3) kyphoscoliosis and osteoarthritis of spine, 14 cases.

Cases of disease of the lung and p'euira invading the chest wall were not included, though they are well recognized as an occasional cause of difficulty in diagnosis. No instance of a diaphragmatic hernia causing pain simulating coronary disease occurred in the present series, but such a condition might be borne in mind in assessing a difficult case. The salient points of a few selected cases are given for illustrative purposes.