miscellaneous disorders, is a frequent finding in diabetes mellitus, and a still more frequent occurrence in hyperthyroidism and in the non-megalocytic "hypochromic" anaemia above described. In the latter disease it seems to have causal relationship, direct or indirect, but in the light of recent work on pernicious anaemia even this surmised causal relationship may ultimately prove to be incorrect. At any rate, this form of anaemia is of frequent occurrence, and its adequate treatment with iron in the ferrous form, by mouth, gives satisfactory results.

It gives me great pleasure to acknowledge, in this study, the cordial co-operation of Dr. W. R. O'Farrell, pathologist to the hospital, and of Drs. L. K. Malley and M. A. Moriarty while they were my house-physicians.

The original lecture was illustrated by more extensive tables and details, which cannot be reproduced here owing to lack of space.

REFERENCES

- ¹ Castle, W. B.: *Amer. Journ. Med. Sci.*, 1929, clxxviii, 748.
- ² Castle, W. B., and Townsend, W. C.: *Ibid.*, 1929, clxxviii, 764.
- ³ Castle, W. B., Townsend, W. C., and Health, C. W.: *Ibid.*, 1930, clxxx, 305.
- ⁴ Barnett, C. W.: *Ibid.*, 1931, clxxxii, 170.
- ⁵ Gompertz, L. M., and Cohen, W.: *Ibid.*, 1929, clxxvii, 59.
- ⁶ Bennett, T. I., and Ryle, J. A.: *Guy's Hospital Reports*, 1921, lxxi, 286.
- ⁷ Baird, M. McC., Campbell, J. H. M., and Hern, J. R.: *Ibid.*, 1924, lxxiv, 339.
- ⁸ Connor, H. M.: *Journ. Amer. Med. Assoc.*, 1930, xciv, 606.
- ⁹ Vinson, P. P.: *Minnesota Med.*, 1922, v, 107.
- ¹⁰ Mettler, S. R., and Minot, G. R.: *Amer. Journ. Med. Sci.*, 1931, clxxxi, 25.
- ¹¹ Kelly, A. B.: *Journ. Laryngol. and Otol.*, 1919, xxxiv, 289.
- ¹² Witts, L. J.: *Practitioner*, 1930, cxxiv, 348.
- ¹³ *Idem*: *Guy's Hospital Reports*, July, 1930, 253.
- ¹⁴ Moore, H.: *Irish Journ. Med. Sci.*, March and December, 1930.
- ¹⁵ *Idem*: *British Medical Journal*, 1930, i, 698.
- ¹⁶ Davies, D. T.: *Lancet*, 1931, ii, 385.
- ¹⁷ Faber, K.: *Berl. klin. Woch.*, 1913, i, 598.
- ¹⁸ Joslin, E. P.: *Nelson Loose-leaf Medicine*, November, 1929.
- ¹⁹ Bowen, B. D., and Aaron, A. H.: *Arch. Int. Med.*, 1926, xxxvii, 674.
- ²⁰ Rabinowitch, I. M., Fowler, A. F., and Watson, B. A.: *Arch. Int. Med.*, 1931, xlvii, 384.
- ²¹ Joslin, E. P.: *Nelson's Loose-leaf Medicine*, May, 1931.
- ²² Faber, K.: *Amer. Journ. Med. Sci.*, 1926, clxii, 1.
- ²³ Wilkinson, J. F., and Oliver, T. H.: *Lancet*, 1931, i, 66.
- ²⁴ Faber, K.: *Ibid.*, 1927, ii, 901.

SOME CLINICAL ASPECTS OF CORONARY DISEASE *

BY

THOMAS F. COTTON, M.D., C.M., F.R.C.P.

PHYSICIAN TO OUT-PATIENTS, NATIONAL HOSPITAL FOR DISEASES OF THE HEART

Clinical observations of practical value in the treatment of patients with disease of the coronary arteries have in recent years been rapidly accumulated, and some of the gaps in our knowledge of the clinical manifestations of this malady have been filled in. For many years the general conceptions of coronary disease, and the events associated with it, were influenced by studies which often lacked the observational precision shown by those great physicians, Heberden, Parry, and Jenner, in their classic records of a hundred and fifty years ago. The theories advanced were based on insufficient knowledge of the anatomy and physiology of this region, and progress was relatively slow. The observation that nitrites, introduced by Lauder Brunton, controlled the pain of angina pectoris by means of vaso-dilatation has undoubtedly increased our knowledge of the clinical pathology of coronary artery disease. Head and Mackenzie made important observations regarding the distribution of pain in anginal subjects, and Mackenzie revived the theory of myocardial exhaustion, and its relation to a deficient coronary circulation, as the cause of anginal pain and heart failure. Allbutt viewed angina as a symptom of

aortic disease, and Wenckebach supported him in opposing those who accepted the coronary explanation of the anginal syndrome; others have maintained that anginal pain may be a symptom either of coronary or of aortic disease. It has long been known that pain with an anginal distribution may be due to other causes than heart disease; the term "secondary angina" has been employed to distinguish such cases from primary angina subjects with structural disease of the heart. In 1912 Herrick in America described symptoms and signs associated with acute obstruction of a coronary vessel, and stated that these patients might recover from the attack. His description of the coronary occlusion syndrome was a valuable contribution to clinical medicine, and it has helped clinicians to diagnose obstruction of a coronary vessel at the bedside, and to offer a fair prospect of recovery to the patient. To American physicians is due the credit of describing the clinical picture of this disease as we now recognize it, and their success in this field of medicine is a tribute to observational methods of research.

Shortly after the war, Pardee and others observed characteristic changes in the electrocardiogram of patients with coronary artery occlusion, which are of the greatest value in the diagnosis of this disease. Some years later Parkinson and Bedford in this country, in a series of accurately recorded cases, confirmed by post-mortem evidence, described electrocardiographic signs which have added to our knowledge of coronary infarction and thrombosis. These clinical observations, by distinguishing the coronary occlusion syndrome from angina pectoris, have established on a sound basis our present conception of some of the clinical phenomena of coronary artery disease.

Within the last three or four years observations relating to the mechanism controlling the peripheral circulation have been made by Lewis and his co-workers. By employing precise observational and experimental methods, they have brought forward evidence which explains how pain is produced in contracting visceral muscle under conditions of circulatory arrest. Their studies support the generally accepted view that ischaemia of the heart muscle, from a defective coronary circulation, is responsible for the pain in anginal subjects. Lewis has now isolated from the general group of anginal patients a distinct type with aortic regurgitation, having a high blood pressure and rapid heart rate during the paroxysms of pain. These signs suggest that widespread vaso-constrictor influences, involving the coronary circulation, are an essential feature, and they emphasize the importance of recognizing, in addition to coronary disease, other contributory factors associated with the anginal seizure. It is beyond the scope of this paper to discuss the pathology of arteriosclerosis, which is the cause of coronary disease, or the class and sex incidence of anginal pain, except to point out that vaso-constrictor influences may be a determinant factor.

There are certain characteristic symptoms arising out of an insufficient blood supply to the heart muscle from structural changes in the coronary vessels which enable us to distinguish two types of cases presenting distinct clinical phenomena: in one, angina pectoris; in the other, the syndrome of coronary occlusion. The differential diagnosis is extremely important, for upon it depends the treatment—which is quite different in the two maladies—and not infrequently the life of the patient.

ANGINA PECTORIS

The distinctive character of the pain of angina pectoris is that it can always be provoked by effort; it is paroxysmal, usually felt in the front of the chest, under the sternum, and down the left arm, and is relieved by rest and nitrites. An emotional upset, a heavy meal, exposure to cold, may initiate an attack or favour the

* A lecture given to the Leicester and Rutland Division, British Medical Association, on December 4th, 1931.

onset of pain after exercise. An attack can, however, occur when the patient is at rest, and not be related to any known exciting cause. The pain is of short duration, but may recur at frequent intervals and completely incapacitate the patient. Pallor, sweating, the look of anguish, and the fear of death are well-known features of this malady. There are signs of structural disease of the cardio-vascular system in the majority of cases—enlargement of the heart, aortic disease, high blood pressure, and electrocardiographic changes. In not a few, perhaps 25 per cent. of all cases, there is no clinical evidence of a structurally damaged heart, and the diagnosis of angina pectoris is based entirely on the character and the position of the pain. Signs of disease are of diagnostic value as confirmatory evidence in doubtful cases, particularly in women and young men who are much less likely to have angina than middle-aged and elderly men. It is particularly important, in cases showing no signs of structural disease of the heart, to look for structural or functional changes in other organs of the body, for such changes may be the cause of a pain closely resembling the true anginal syndrome. An unstable vasomotor system, with a predominant vaso-constrictor element, a toxic state from an infected gall-bladder, an inflamed appendix, a toxic goitre, or some other seat of focal sepsis, may play an important part in producing the pain of what has been called "secondary angina." In some primary anginal subjects the contributing causes of secondary angina may influence the course of the disease, and to assess their relative significance, although in many cases a difficult task, is important from the point of view both of prognosis and of treatment.

SYNDROME OF CORONARY OCCLUSION

The coronary thrombosis syndrome is a definite clinical picture, easy to recognize and quite distinct from angina pectoris. The pain is anginal in character and distribution, and lasts for hours or days; it is sudden in its onset, gradually fades away, usually occurs when the patient is at rest, and is often accompanied by a considerable degree of respiratory distress. Amyl nitrite inhalations do not control this pain. Occasionally breathlessness is a conspicuous symptom and pain is absent; in these rare cases an electrocardiogram with characteristic changes is usually required to establish a diagnosis. Vomiting is a commonly associated symptom, and when accompanied by abdominal pain may lead to a mistaken diagnosis of an acute surgical condition. Symptoms of shock, with a rapid pulse, low blood pressure, and weak heart sounds, may be a striking feature of the illness. There is generally a rise of temperature for a few days or, in exceptional cases, for several weeks; a leucocytosis is the rule, and occasionally a pericardial friction rub can be heard. Signs of embolism arising from the necrosis of the infarct, which, in the majority of cases, is in the descending branch of the left coronary artery, may be expected. Death can occur within a few minutes of the onset of the occlusion. Of those who recover, probably 50 per cent. of all cases, some may lose all their symptoms within a few days; others develop signs of congestive failure or symptoms of angina pectoris, and many have exhaustion symptoms and a low blood pressure for weeks or months. With the onset of the attack, there is sometimes a disturbance of the heart's rhythm; all types of cardiac irregularities have been recorded. Coronary infarction can develop without causing symptoms; there is post-mortem evidence of this in patients who have died without presenting any manifestations of heart disease during life.

There are certain electrocardiographic signs which may be regarded as reliable evidence of coronary thrombosis. Characteristic changes occur in the QRS and T complexes

which form that part of the curve lying between the initial and the terminal ventricular deflections. The most important of these are the coronary T wave changes described by Pardee. Although electrocardiographic signs are of the greatest value in diagnosing coronary thrombosis, abnormalities in the electrocardiogram are not always present in this disease.

A large number of these patients are hypertension subjects, and many have had anginal pain with coronary occlusion the terminal event. Prognosis is more favourable in the group with angina pectoris than in those with coronary occlusion; the average anginal patient is likely to die within five years from the onset of symptoms, and generally the coronary occlusion case will not survive the attack for a longer period than two or three years. Although in both types the malady is serious and the prognosis must be a guarded one, there are records of patients living for many years. If there are signs of gross structural disease, with enlargement of the heart or valve disease, the outlook is more serious than when such signs are absent. Within the first three weeks of the onset of occlusion, whether the symptoms are severe or mild, no definite prognosis can be given with any degree of certainty. Beyond this period, if there are no symptoms or signs of heart failure, and there has been an improvement in the general condition, the patient may be expected to recover from the attack, and possibly regain normal health and be able to lead an active life; others are crippled with anginal pain or develop congestive failure symptoms and die. In the anginal group, frequent seizures occurring at rest and not controlled by treatment are a grave prognostic omen; sudden death can occur at any time, life is a misery, and the end is a happy release from a precarious existence. When pain is felt only after effort and is easily avoided by restricting activities the patient may have an unexpectedly long and useful life.

TREATMENT

The indications for treatment of these two maladies are essentially different. Coronary thrombosis is an acute illness, sudden in its onset, and there may be rapidly developing symptoms of a grave heart affection. Morphine should be given in doses large enough to relieve the pain, which is an early and conspicuous feature of the occlusion syndrome. Rest in bed for at least a month is essential, preferably for six weeks; and every unnecessary movement should be avoided for two weeks, or longer if there are symptoms of shock, signs of embolism, or congestive failure. Good nursing and mild sedatives are more important than circulatory stimulants: mild purgatives and enemas are usually required if morphine is continued for any length of time. Digitalis is indicated when there are signs of congestive failure and the auricles are fibrillating or in flutter; adrenaline should be reserved for patients with signs of heart-block, and quinidine restricted to rare cases of paroxysmal tachycardia of unusual length. A convalescence of two months is always necessary before allowing the patient gradually to resume a part- or full-time active life.

Angina pectoris is a chronic malady and the treatment ambulatory in most cases. When the attacks recur at frequent intervals, or reach a stage of status anginosus, and the pain is not easily controlled by the nitrites, a long rest in bed is indicated. Such patients are sometimes helped by chloral, or morphine given in sufficient doses to raise their pain threshold and keep them free from pain for a week or two. The therapeutic measures upon which we must rely are based on restricting their activities, both physical and mental, and regulating their lives so that the extracardiac contributory factors aggravating the anginal syndrome may be eliminated or controlled;

a suitable diet, a change of environment, and the treatment of focal sepsis, form part of this regimen. It is doubtful whether drugs which are supposed to have a dilating action on the coronary vessels materially alter the course of the disease. For patients with cardio-aortic syphilis, iodides and mercury should always be prescribed; intravenous injections of arsenic are indicated in the early stages of the disease, and can be given under conditions of rest in moderately large doses.

As regards the narrowing or obstruction of the coronary arteries in relation to breathlessness and congestive heart failure, it is a well-known clinical experience to see congestive failure develop rapidly as a result of a sudden occlusion from infarction of a coronary vessel, or to have dyspnoea a striking symptom of arrest of the coronary circulation. When there is an insufficient blood supply to the myocardium from a narrowing of the coronary arteries as part of a general or local arterio-sclerotic process, the function of the heart is impaired and there is undue breathlessness on exertion. This is a common complaint in elderly people with cardio-vascular changes of a senile type, and is often observed in middle-aged hypertension subjects. With a gradual or rapid failure of the heart to maintain the general circulation of the body, the heart increases in size, and signs of congestive heart failure dominate the clinical picture. Progressive heart failure of this nature is likely to follow the onset of auricular fibrillation, although it may take place with a normal rhythm. The treatment of breathlessness in this large and ill-defined group of patients is that of heart failure in all its grades. With suitable therapeutic measures a recovery of the functional efficiency of the heart which can be maintained for a long period may be expected in some cases; in others, heart failure cannot be arrested, and with a rapidly increasing disability death is not long postponed.

THE DIAGNOSIS OF NEUROTIC CONDITIONS IN GENERAL PRACTICE*

BY

DAVID FORSYTH, M.D., D.Sc., F.R.C.P.

SENIOR PHYSICIAN TO CHARING CROSS HOSPITAL; CONSULTING PHYSICIAN
TO THE EVELINA HOSPITAL FOR CHILDREN; LATE SENIOR PHYSICIAN,
PSYCHOLOGICAL CLINIC, MINISTRY OF PENSIONS

During the last quarter of a century medical psychology has been revolutionized. The neuroses have been made to yield the secret of their origin and nature, and for the first time the kinship has been traced between all the chief types of human personality—normal, eccentric, neurotic, psychotic, sexually perverted, and criminal. But as yet little of this accession to our knowledge has reached the body of medical practitioners, at any rate to the extent to being utilized by them in daily practice. For one thing, this new work has been mainly contributed by psycho-analysis, and Freud's discoveries have been looked at even more askance by the medical profession than by the laity—perhaps for the reason that doctors have received no training in psychology (thanks to the medical curriculum), and both in their student days and later their preoccupation is with organic diseases. They have yet to realize that the importance of Freud's discoveries is no longer disputed among psychologists, and that many of these discoveries are directly applicable to their neurotic patients. Further, now that the acceptance of psycho-analysis is general among the laity, the difficulty has increased of satisfying patients or their relatives by merely pushing the subject aside as an unpleasant one.

* A post-graduate lecture delivered at Charing Cross Hospital Medical School.

Not that a general practitioner has need of any technical training in analysis, but he should be able to recognize a neurotic case as surely as he diagnoses organic diseases, and to overlook a case of either kind is equally serious a technical error. Even after a full medical examination has excluded organic disease, the diagnosis of a neurosis is to be made only on psychological evidence obtained by a full psychological examination. It is not permissible to make this diagnosis negatively, merely for lack of evidence of organic disease. We know how often physical signs are absent in the earlier stages of numerous organic diseases, including cancers, tuberculosis, anaemias, cerebral tumours, and general paralysis. Remember, also, how organic and neurotic diseases may coexist in the same subject; and that many patients with organic disease develop a "halo" of psychogenic symptoms which may easily obscure the organic condition.

I

As a first step towards this psychological diagnosis the practitioner will probably require to revise some ideas which have guided him hitherto. The very term "functional" (which, after all, is more misleading than informative) is best replaced by "psychogenic," that is, mentally produced. Little or no diagnostic value now attaches to the so-called "stigmata" of hysteria, to areas of anaesthesia or hyperaesthesia, or to the state of the tendon reflexes. The methods, clinical and laboratory, of investigating organic disease are inapplicable to cases of neurosis, a totally different examination being needed. In approaching a case of this latter kind forget for the time being that your patient is anatomically composed of different organs—your concern with him is of another sort. Discard equally the supposition of a neurosis of a viscus—a "gastric" neurosis or a "cardiac" neurosis—which implies a distortion and falsification of the facts, inasmuch as psychogenic symptoms referable to a viscus are never more than a detail of a neurosis of which more widespread evidence can be found.

A positive diagnosis requires that two primary conditions must be satisfied. The first is that a *neurosis* is *essentially a disorder of temperament*; the manifestations of it, therefore, are to be looked for in the patient's emotions and behaviour. It never consists of a single symptom, localized perhaps to one part of the body, and rarely of a few symptoms, but of many, which are to be sought over the whole range of the patient's temperament. With experience one comes to recognize a fairly typical case of neurosis as readily as one does a straightforward case of organic disease.

The second condition to be satisfied before diagnosing a neurosis is that *every neurosis begins in childhood*. The diagnosis, therefore, is not to be made without a psychopathic history leading back to nursery years. As a rule, the earlier in childhood that the psychological development went wrong the graver is the result, so that, for example, disturbances within the first year or two are responsible for setting up psychoses, while those between the third and sixth lead to nothing more serious than a neurosis; and healthy development over the entire first five or six years excludes a neurosis in adult life.

The investigation of a case of neurosis proceeds by allowing the patient to talk freely, and so disclose his disposition and its peculiarities. The very loquaciousness of most neurotics, which to the psychologically untrained can be frankly boring, becomes to the medical psychologist as significant and illuminating as is a detailed physical examination to the clinician. Here a warning must be given. Though neurotics are usually tiresome, difficult, and exacting, a doctor must not be provoked into showing any impatience or annoyance at their failings. He needs