REFRESHER COURSE FOR GENERAL PRACTITIONERS

CORONARY ARTERY DISEASE—I. THE CLINICAL SYNDROMES

BY

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Sooner or later the hearts of most adults are affected by arteriosclerotic changes in the coronary arteries; fortunately, in only a minority are the obliterative changes so advanced as to impair seriously the myocardial blood supply and thus give rise to symptoms. Myocardial ischaemia may be circumscribed or diffuse, temporary or permanent, acute or gradual in onset, depending on a number of factors. Hence, when blood flow is disturbed by obliterative disease in the coronary tree a variety of overlapping syndromes may result.

Diagnosis of Coronary Sclerosis

The diagnosis of arteriosclerotic heart disease is based primarily on symptomatology. In contradistinction to other forms of heart disease, chest pain is a very characteristic feature. It dominates the clinical picture of coronary sclerosis.

In the absence of a history of pain the diagnosis of coronary sclerosis is less certain but may be presumed when heart failure in an acute or chronic form is encountered in the absence of other provoking cause. Cardiac asthma and acute pulmonary oedema should always suggest coronary disease as readily as the gradual development of congestive failure in an elderly person.

Disturbances of rhythm can give a clue to the diagnosis. A bout of auricular fibrillation may be the first evidence of arteriosclerotic heart disease. Curiously it is seldom associated with angina. Complete heart-block, for instance, with or without its characteristic syncopal attacks, has for its origin an arteriosclerotic basis more often than any other cause. Block of one or other bundle-branch is more common than block of the main stem of the bundle. Branch-block is not in itself a cause of symptoms, but it is a common manifestation of coronary damage.

Coronary sclerosis can be productive of symptoms without there being a change in the size of the heart. Except in the presence of congestive failure, radiological examination seldom shows more than minimal enlargement. It is worth emphasizing that coronary heart disease occurs independently of thickened radial, retinal, or other arteries, and that it is not a mere product of age. Youth does not exclude the diagnosis. The myocardial infarct is its hallmark.

As clinical examination may not elicit any physical signs, even in the presence of serious coronary disease, the diagnosis often depends on an assessment of symptoms. It is therefore imperative to hear the patient's own account of his difficulties and his personal description of their nature and development. This applies particularly to the differentiation of the causes of precordial pain.

Angina Pectoris

The basis of cardiac pain is ischaemia, the result of a discrepancy between the oxygen supply and the oxygen needs of the myocardium. The quantity and quality of

the blood delivered to the active muscle depend largely on the calibre of the vessels, the blood pressure, the heart rate, and the haemoglobin content. The demands of the heart for oxygen are determined primarily by the amount of work which it is called upon to perform. In health considerable reserves permit ready adjustments between the supply and demand, but in the presence of thickened and rigid coronary arteries with narrowing of their lumen the delivery of adequate amounts of blood falls short of the local metabolic needs. Pain is then apt to occur and to persist until either the debt is made good or local necrosis develops.

The quality of the discomfort varies greatly from patient to patient. Some deny that they have pain, and emphasize a sense of heat, weight, oppression or tightness in the chest, or a choking in the throat induced by exercise or emotion and equally promptly relieved by rest or peace of mind. Upward radiation of retrosternal pain, with numbness and tingling in the jaws and gums, or downward radiation of similar sensations in one or both arms to the elbows, wrists, and hands are very characteristic features. Distress more readily provoked after than before a meal, and earlier in onset in cold than in warm surroundings, is commonly noted.

The "second-wind" phenomenon can be a misleading feature of organic coronary disease. The patient then observes that an attack, often quite severe, is readily provoked when exercise is first undertaken. Having rested a minute or two and recovered from the initial pain, he finds, on resuming his activities, that his capacity for effort thereafter quickly increases. He can walk at a similar or faster pace, perhaps for hours, with freedom from distress. It is commonly the first hole of a round of golf which presents the greatest difficulties to the anginal patient. A reactive hyperaemia, which makes good the initial coronary insufficiency and which is thereafter maintained by gentle exercise, has been advanced as the explanation for this common feature of coronary disease.

Accounts of other chest pains—arthritic, muscular or pleural, submammary and functional, peptic, biliary, or oesophageal in origin—can all be misleading, particularly when the patient is liable to two types of chest pain—the short-lived constrictive pain or transient ischaemia, and a more prolonged pectoral or upper abdominal ache, the product of fatigue, anxiety, or other cause. The dominant oppressive quality of genuine coronary pain, its wide radiations, and its precise relation to activity are its typical features. It is a good rule that if a man can take a brisk walk on a cold day without interruption necessitated by substernal pain he is not suffering from angina of effort. Occasionally, in those of less active habits, the pain is experienced only under conditions of emotional distress.

Instrumental Proof.—When doubt exists about the nature of the attacks an exercise test may be employed with advantage. A true anginal attack can usually be

induced within a few minutes by making the patient run up a flight of stairs, step up and down on a stool, or undertake standing-running exercise at as brisk a pace as he can manage; it can as promptly be relieved by rest on a couch. In a large proportion of sufferers from angina the electrocardiograms show, for a few minutes immediately after exercise vigorous enough to cause even minor distress, transient alterations in the pattern of the RST segment and T waves (see Fig. 1). This is

FIG. 1.—The attack of angina pectoris temporarily disturbs the pattern of the electrocardiogram in the majority of sufferers—a finding of value when the diagnosis is in doubt, as thereby the cardiac origin of the pain may be substantiated. Column A from above downwards records in each of the three standard limb leads and one chest lead (from the region of the apex beat) a single heart cycle under resting conditions. The patient, a man of 57, with a five-year history of angina, performed an exercise test sufficient to induce a mild attack of pain with well-marked dyspnoea. Column B presents similar leads from the same patient recorded consecutively from above downwards at intervals of a few seconds up to two minutes as he lay on a couch after completion of the test. The alteration of the form of the ST intervals, most evident in the chest lead (IVF), is evidence of ischaemia and confirms the cardiac origin of the pain. Column C shows that similar alterations in pattern present 5 to 6½ minutes after exercise in this man. More than 15 minutes elapsed after this induced anginal attack before the electrocardiograms regained the form existing before the exercise test was undertaken.

an important test of coronary efficiency, as in doubtful cases it may provide the only objective proof of the cardiac origin of the pain. Casual electrocardiograms recorded in the intervals between attacks may be of normal form or show only evidence of inert myocardial damage. They are of much less value than those displaying a resolving pattern of ischaemia in serial records after exercise. A simpler diagnostic method, but less reliable on account of the allowance which must be made for the "second-wind" phenomenon and for psychic influences, is the employment of nitroglycerin as a therapeutic test.

Coronary Thrombosis with Myocardial Infarction

The features of the major attack of coronary thrombosis are well known. The formation of the circumscribed infarct commonly gives rise to prolonged sub-sternal pain of great severity which persists for hours. Profound shock develops rapidly. Restlessness, pallor, sweating, nausea, and vomiting may be followed in severer cases by respiratory distress and signs of left ventricular failure with evidence of congestion along the margins of the lungs, in the axillae, and at the bases posteriorly. The blood pressure falls progressively for a few days.

Examination may show tachycardia, a feeble apical impulse, poor quality heart sounds, and a triple rhythm, and within a day or two local pericardial friction may occasionally clinch the diagnosis.

Tissue necrosis accounts for the prompt leucocytosis, early febrile reaction, long-sustained disturbance of the sedimentation rate—a sign which, persisting for a month or more, is often helpful in establishing the

FIG. 2.—The sustained elevation of the sedimentation rate after a myocardial infarct is often a diagnostic sign of value. The disturbance is slow to develop, reaches a maximum in the second or third week, and reverts to the normal range within a month or two as a rule.

diagnosis in retrospect (Fig. 2)—and the sequence of changes observed in serial electrocardiograms. An increased clotting tendency of the blood, usually maximum at the end of the second week, explains the proneness of these patients to thrombo-embolic complications and the hazard of early death when the worst of the acute illness is apparently past.

Milder attacks of coronary thrombosis are common and are readily overlooked. In taking a history from a patient whose account suggests angina pectoris, inquiry should always be directed to the possibility of one or more attacks of infarction. A history of a minor occlusion, perhaps with acute symptoms of no more than 10 to 20 minutes' duration, may be obtained by having him describe his longest, his most severe, or any unexpected or unprovoked attack of pain. Suspicions will be strengthened by any admission of unusual exhaustion accompanying the attack or a less prompt restoration of bodily well-being thereafter.

Instrumental Proof.—The nature of the attack may be established by multiple-lead electrocardiograms,

which are capable of locating the site of the myocardial infarct with accuracy in most instances. By special techniques, using multiple chest and unipolar limb leads, anterior, antero-septal, antero-lateral, posterior, postero-lateral, high lateral, and certain septal infarcts, which would have escaped detection some years ago, can now be recognized and the diagnosis established even when months or years have elapsed since the acute episode. Exceptionally, even serial electrocardiograms repeated at intervals of a few days after the acute infarction fail to confirm the diagnosis. This is true particularly of

certain septal infarcts. The method, like other laboratory procedures, therefore has its limitations, but refinements in technique are steadily reducing the likelihood of error.

Coronary Insufficiency

In a physiological sense all attacks of angina pectoris and the various grades of myocardial infarcts are examples of coronary insufficiency. In recent years this particular term has, however,

been applied by Master and his colleagues to a fairly distinctive syndrome intermediate between the milder attack of infarction and the severer anginal seizure. Occasionally ischaemic attacks are asymptomatic, but more often they are characterized by spontaneous cardiac pain at rest, longer and severer than the usual attack of effort angina and yet not as severe as the clear-cut episode of coronary thrombosis. These attacks may proceed in the more prolonged instances to tissue asphyxia, with the development of a patchy speckled subendocardial necrosis evident in the papillary muscles or in the deepest muscular layers of the left ventricle. Coronary sclerosis, moderate to severe in degree, is the rule.

The attacks are not the result of coronary occlusion but develop under conditions of increased cardiac work, diminution of blood flow, or defective oxygenation of the blood. Hence, in the presence of coronary sclerosis, severe and prolonged physical exertion, intense emotional distress, a paroxysm of tachycardia, an exacerbation of hypertension, or, on the other hand, prolonged hypotension or the sudden anaemia of acute blood loss, as for instance a profuse haematemesis, may result in myocardial ischaemia severe enough to lead to tissue necrosis.

The most ominous of these attacks are those lengthening bouts of severe angina experienced at rest at increasingly shorter intervals for no apparent reason, so that ultimately a "status anginosus" may be described. Such a sequence often heralds a major coronary thrombosis with massive infarction.

Instrumental Proof.—As might be anticipated, acute coronary insufficiency is accompanied by depression of the S-T intervals with flattening or inversion of the T wave in two or more leads—a pattern very similar to that observed in induced attacks of angina. In the latter condition the characteristic deformities seldom persist for more than 15 to 20 minutes. In coronary insufficiency of the type now under consideration the ischaemic pattern wears off in hours or at most in a few days, its long persistence denoting diffuse subendocardial necrosis of some severity. In contrast, the electrocardiographic pattern of the circumscribed myocardial infarct of coronary thrombosis undergoes a more gradual transformation, the process commonly extending over months or years. An example of the electrocardiograms of coronary insufficiency is shown in Fig. 3.

Other Syndromes of Coronary Sclerosis

The diagnosis of myocardial infarction is so important, and so many cases are missed on account of the absence of pain, that it should be a rule to consider this possibility under a variety of conditions. Among these, attacks of nocturnal dyspnoea are predominant. Every attack of cardiac asthma should be regarded as the product of a myocardial infarct unless other demonstrable cause exists. Similarly, a sudden pulmonary oedema, so characteristic of acute left ventricular failure, may mask other symptoms and be revealed only as the product of an infarct, when the subsequent behaviour of the blood pressure, the sedimentation rate, and serial electrocardiograms supplement the diagnosis.

Sudden prostration or collapse, syncopal attacks, or giddiness, with residual exhaustion and general weakness, are sometimes explicable on a similar basis. Less clear-cut syndromes occur in the elderly. Nocturnal

FIG. 3.—Coronary insufficiency produces a temporary disturbance in the electrocardiogram, the pattern reverting to normal within a day or two. Column A (taken on December 19) displays from above downwards electrocardiograms each of a single cardiac cycle in the three standard limb leads and in three chest leads. These were recorded 48 hours after the onset of a severe anginal attack of seven hours' duration, provoked by intense emotional distress but unaccompanied by shock or by signs of tissue necrosis. The patient was a man of 30, whose attack developed while under cross-examination by the police. He was admitted to hospital with probable coronary thrombosis. Column B (taken on December 22) presents similar electrocardiograms from the same patient after two further days in bed. The pattern has reverted to normal form. Comparison indicates that the inversion of the T waves in Leads I, II, and III present on the first occasion has been corrected and the T waves in chest Leads V2, V4, and V5 have increased in amplitude.

restlessness with insomnia and perhaps a minor sense of dyspnoea followed by a transient auricular fibrillation may provide a clue which would be otherwise missed were a search not made for evidence of tissue necrosis. The occurrence of systemic and commonly fatal embolism a week or two after the restless night too often clarifies the diagnosis when, alas, the opportunity for effective therapy is past.

Finally, there are those incipient cases of congestive heart failure of gradual onset in which by a process of exclusion the aetiology is assumed in the absence of other cause. The electrocardiogram may then provide a further link in the diagnostic chain.

MEDICAL AUXILIARIES IN NATIONAL HEALTH SERVICE

PROPOSED STATUTORY REGISTRATION

"Medical auxiliaries are persons who assist medical practitioners (otherwise than as nurses) in the investigation and treatment of disease by virtue of some special skill acquired through a recognized course of training." With this definition—which did not go unchallenged by some of the medical auxiliaries concerned—eight committees set up by the Minister of Health and the Secretary of State for Scotland have been considering the supply and demand, the training and qualifications, of certain of the classes of medical auxiliaries employed in the National Health Service—namely, almoners, chiropodists, dietitians, laboratory technicians, occupational therapists, physiotherapists and remedial gymnasts, radiographers, and speech therapists. Mr. V. Zachary Cope was chairman of all the eight committees, and one other medical and one lay member were common to them all. For the rest, each committee consisted of two or three—in one case four—medical members and usually two members representative of the category concerned. This eightfold report has just been published as a White Paper,¹ the first part being devoted to problems common to all these auxiliary services, and the second to the assessment of each service separately.

The committees, in the course of two years, have held more than 100 meetings and received a vast amount of evidence. Evidence from the British Medical Association and from the present Board of Registration of Medical Auxiliaries was tendered to all eight committees. Mr. Zachary Cope had a difficult team to drive, due, not to personalities, but to the diversity of the interests represented. The one common factor is that the auxiliaries (with the possible exception of the chiropodists) work under the direct or indirect supervision of a doctor. But in the various schemes of training the points of emphasis are different, and the desirable standards of attainment are not the same in all the categories. Certain basic sciences, such as anatomy and physiology, may be necessary in most cases, but obviously the radiographer and the physiotherapist require a much more comprehensive knowledge in this field than the chiropodist or the speech therapist. These latter two need to have a certain amount of detailed anatomical knowledge, but relating to two different parts of the body. Again, the occupational therapist has a specialized knowledge and skill which can be acquired only outside the field of medicine, and the almoner is primarily a social worker, bringing into the Health Service knowledge and experience gained in the social science departments of universities and in fields of social survey other than health.

Indeed, a minority report by the almoners and the occupational therapists disputes the term "medical auxiliary," which in their contention denotes an untrained or partially trained person—e.g., a nursing auxiliary. They prefer the term, "non-medical professional and technical staff," like the

"non-professorial teaching staff" in universities. It is an indication of the difficulty of this inquiry that there are three minority reports which are signed by all the medical auxiliary representatives of four of the eight committees, though their reservations are only to portions of what is recommended. And one minority report falls foul of the report of another minority.

The Present Position

At the end of 1949 the number of persons in England and Wales and in Scotland working in the National Health Services as auxiliaries was as follows:

Almoners	975	Occupational therapists ..	980
Chiropodists	388	Physiotherapists ..	4,019
Dietitians	177	Radiographers ..	2,444
Laboratory technicians ..		Remedial gymnasts ..	146
(including students) ..	3,195	Speech therapists ..	121

About 84% of these were employed whole-time. Only among chiropodists and speech therapists was there a majority of part-time workers.

It will be remembered that as a result of the efforts of the British Medical Association and the Society of Apothecaries of London a Board of Registration of Medical Auxiliaries was formed in 1936. This board is on a voluntary basis and as such is subject to certain handicaps. Of the qualifying bodies now under consideration, two only—the Society of Chiropodists and the College of Speech Therapists—insist upon their members registering with the board. In some of the categories only a small proportion have applied for registration.

What is now proposed by the eight committees is the setting up of a council for Medical Auxiliary Services in the National Health Service to maintain statutory registers of persons qualified for these employments, to recognize schools, courses, and examinations, to keep under review the relation of supply to demand over the whole range of the services, and, among other functions, to ensure that the training in the medical auxiliary services keeps pace with the advances in medical thought and practice. While the diversity of the services is recognized, it is felt nevertheless that a single council is preferable to independent group councils as being more likely to command respect among the auxiliaries themselves, as well as in the medical profession and among the general public. Registration by the council would be an essential qualification for work in the National Health Service.

Central Council and Professional Committees

In setting up this central council the pattern of the General Medical Council and similar bodies would be followed. It is proposed that it should consist of four members appointed by the Privy Council, under whose aegis it would function, three by the Minister of Health and the Secretary of State for Scotland, seven by the Royal Colleges and Corporations, and one by the General Medical Council. There would be six representatives of the medical auxiliaries themselves. It may be asked why the figure is limited to six when the inquiry has necessitated eight committees to cover the different categories. It is suggested that the function of these medical auxiliary representatives on the council would not be to put forward the views of a particular category, but to supply the point of view of the medical auxiliary services as a whole. Thus it will not be necessary to have every one of the medical auxiliary services represented, unless matters pertaining to it alone are brought forward, when special representation may be arranged.

In addition to the council there will be a series of professional committees, the majority of whose members will be experts in a particular field of auxiliary service. The chairmen of these committees will be appointed by the council, but the vice-chairmen will be appointed by the committees themselves, and these vice-chairmen will form a kind of electoral college for the appointment of the six representatives of the auxiliaries on the council. The number of these professional committees is not laid down. As a rule, each

¹ Reports of the Committees on Medical Auxiliaries. Cmd. 8188. H.M.S.O. 5s. net.