NOTES ON ISCHAEMIC HEART DISEASE

D. G. FRENCH, M.D.
Kidsgrove

THIS paper is based on a review of 370 electrocardiograms done in my practice in the past five years for purely clinical purposes. An unexpected proportion of these (79 per cent) is concerned with the various manifestations of ischaemic heart disease; there are 91 ECGs of infarction, and 202 were done for the purpose of investigation or diagnosis and follow-up of possible or actual ischaemic heart disease other than infarction.

During this period, 78 cardiac infarctions have been studied, that is, 15.5 per year, but this does not represent the whole incidence in the practice as some infarctions occurred without warning, became coroner's cases, and were never investigated by me. The practice population is 5,700, so the incidence of cardiac infarction is about three per 1,000 per year.

Pain

Angina of effort and the pain of cardiac infarction are usually readily recognized. There is nearly always chest pain, which patients describe in a variety of ways. It is commonly felt across the chest about the level of the 3rd or 4th costal cartilage and may be described as a tight pain or a constricting pain, or as a sensation of pressure on the chest. The pain may be more localized and central, and I have had it described as feeling as though the chest were full of hot sand, or like having a stake driven into the chest. The pain may go through to the back, and sometimes the pain in the back so completely overshadows the anterior pain that the patient does not mention it. Then, the distribution of severe pain in the back, between the shoulders and going up to the back of the neck is most misleading unless the history is carefully taken.

The chest pain is usually accompanied by spread to other parts. Most commonly there is pain in the left upper limb; it may be confined to the shoulder or the arm or forearm, or the whole arm and hand may be affected; less commonly it is limited to the fingers or the ulnar side of the hand or the wrist or thenar eminence. The

right arm is not often affected alone, but commonly there is pain in both arms. There may be pain in the left side of the face and neck, or in the lower jaw which the patient may describe as toothache, even if edentulous.

The pain may be described as ' like indigestion ' and I have had a cardiac infarct present with epigastric pain and vomiting. In these circumstances the diagnosis is liable to be missed, but if the history is carefully taken it is usually possible to establish the relationship to effort during the few preceding weeks, and probably also that there has been pain of more typical anginal distribution, not mentioned by the patient because it has been the less severe of the two. Few of us can say that we have never missed the diagnosis in the middle-aged or elderly patient whose presenting complaint was indigestion.

On reviewing the cases of unexpected sudden death from coronary artery occlusion which have occurred in the practice during the past few years, I find that on questioning the relatives afterwards for a history suggesting angina, they have frequently answered ' he had no pain whatever', but in answer to a direct question have admitted that he had had ' indigestion ' for a week or two before death.

These different distributions of pain cannot be related with any degree of certainty to the position of the infarct as determined by the electrocardiograph, though symptoms of indigestion have been more frequently associated with anterior infarcts in this series.

Duration of pain. Angina is almost always related to exercise or effort and passes off in a few minutes with rest. Angina which lasts 20 minutes or more with rest is almost certainly due to infarction. Other than that, the commonest cause of angina at rest is an attack of paroxysmal tachycardia; this type of angina is not relieved by nitroglycerine but is prevented by quinidine. A test of some diagnostic value, irrespective of tachycardia, is massage of the right carotid sinus which usually relieves the pain of angina at once.

The ECG *in ischaemic heart disease*

The importance of history and clinical examination cannot be overstressed, but the ECG is usually most helpful. The commonest ECG evidence of ischaemia is depression of the ST segment in the left praecordial leads, and the characteristic of this depression is that the take-off point from the S wave is depressed. Digitalis produces ST segment depression but in this case, the take-off point of the ST segment is iso-electric. When there is little or no ST segment depression at rest, it may be provoked by means of the two-step exercise.

The application of vectorcardiography to the 12-lead ECG is a rapid way of recognizing two other signs of ischaemia; the first is a wide angle, more than 90 degrees, between the frontal plane QRS and T

vectors; the second is an angle of more than 90 degrees between the vectors for the initial and terminal 0.04 second of the QRS complex.

The essential sign of infarction on an electrocardiogram is the presence of Q waves in the limb or praecordial leads, and to be diagnostic these waves must be at least 0.04 second in duration. Small short sharp Q waves of less than 0.04 second duration in the limb leads are usually physiological and can never be regarded as diagnostic of infarction. Q waves usually persist; other changes, shift of the ST segment and inversion of T waves which usually accompany the acute stages of infarction, are temporary.

Peri-infarction block, shown by wide separation (100 degrees or more) of the initial and terminal 0.04 second vectors of the QRS complex is, by definition, another ECG sign of infarction. Anterior peri-infarction block is accompanied by left axis deviation and Q waves in some of the praecordial leads and there is no difficulty about diagnosis. Posterior peri-infarction block is not so easy, and unless a previously normal ECG is available, can only be relied upon in the presence of strong supporting history and clinical examination.

Infarction

Angina of recent origin. All new cases of angina should be examined electrocardiographically. In the majority, the ECG shows only minor changes, but occasionally there is marked ST segment depression (more than 1 mm., and sometimes more than 2 mm.) in the left praecordial leads without exercise. These patients usually infarct within a few days of the examination and therefore must be treated urgently. Admission to hospital is indicated. It may be difficult to convey this sense of urgency not only to the patient but also to the hospital. I have had six cases of angina which have shown this ECG pattern in the past five years and all infarcted.

Infarction without ECG *changes.* On three occasions a clinical diagnosis of infarction has been made and the ECG has been normal. In two of these, the ECG was done within 12 hours of the infarction. In the first, I waited a further two days and repeated the ECG; as it was still normal, a cardiologist was called in and he admitted the patient to hospital, where, in the course of a few days, an ECG confirming the diagnosis of infarction was obtained. The second patient was sent straight into hospital and, again, confirmatory evidence of infarction was obtained after an interval of one or two days. The third patient had an ECG the day following his infarction and it was normal; blood was taken and sent to hospital for transaminase estimation. On the fourth day after the infarct, the ECG was repeated and was still normal. At this stage he was sent into hospital where he subsequently developed an ECG pattern of posterior infarc-

tion. Two days after his admission I received the report from the pathology department showing SGOT 116 units.

Cases of this sort are rare but they show that a normal ECG must sometimes be disregarded in favour of a tentative clinical diagnosis. This just makes one wonder about those doubtful attacks of chest pain in which an ECG, done for diagnostic purposes, is normal; but I must add that for some time I have made a practice of repeating the ECG after an interval of a week in all suspicious cases and, apart from the three cases described, have not come across a missed infarct.

Infarction with equivocal ECG *changes.* The commonest difficulty here is the ECG which shows left bundle branch block, because this completely obscures the signs of infarction. One must then rely on ordinary bedside diagnosis though this can be supported by an SGOT estimation later. If a previously normal ECG is available, the development of left bundle branch block coinciding with an attack which suggests infarction clinically is presumptive evidence that an infarct has in fact taken place. Similarly, the ECG pattern of posterior peri-infarction block may be difficult to interpret in the absence of a previous normal ECG for the same patient. No ECG done in general practice is ever wasted effort and they should all be filed for future reference.

Infarction without previous history. In the past five years, I have found four cases of infarction which were quite unexpected. Three were patients aged between 65 and 70 years who presented with signs of heart failure and on investigation were found to have had an infarct at some previous date. No amount of questioning could elicit a history of the acute episode. The fourth was found during a survey in the practice; in this case, very close questioning of the man and his wife elicited the history that, three months previously, he had had a bad attack of indigestion and had missed one day's work. It is of some interest that of these four patients, two were diabetics. So called ' silent infarct ' is a very real thing and should be considered when one is confronted with an unexplained heart failure.

Associated conditions

In this series, other conditions most commonly associated with ischaemic heart disease have been diabetes mellitus, gall stones, pneumoconiosis, hypertension, obesity and aortic disease. So far as possible, these conditions require treatment, but control of hypertension and obesity are the only ones likely to have any long-term effects.

Angina and anaemia. Angina of effort due to anaemia is exactly like angina due to coronary artery disease, but disappears when the

anaemia is effectively treated. These patients are usually suffering from temporary heart failure of which there are other manifestations —dyspnoea, palpitation and symmetrical dependent oedema. This is surprisingly common and one must always keep in mind the prevalence of anaemia in patients past middle age. In this practice, a recent survey of 500 successive patients over the age of 50 showed that 13 per cent of women had haemoglobin levels of 80 per cent or less; anaemia in men was less common. It is a salutary experience to check the haemoglobin levels of those patients who return for repeat prescriptions for nitroglycerine. The anaemia itself may require investigation in case there is some serious underlying cause.

'*Strokes*'. In the absence of previous infarction, a stroke is probably due to an unrelated cerebrovascular accident. Sudden death occurring in a patient who has had treatment for angina is usually attributed by most of us, for purposes of death certification, to cardiac infarction, but some of these cases come into the province of the coroner and necropsies are performed. Not infrequently cerebral haemorrhage or ruptured dissecting aneurysm is shown to be the cause of death.

After a cardiac infarction, a stroke is much more probably due to cerebral embolism due to a fragment of left ventricular mural clot. In fact, embolism may occur anywhere in the systemic circulation and must be considered in the diagnosis of any sudden pain occurring during convalescence from cardiac infarction.

'*Black-outs*'. This vague term is used to describe everything from a temporary and convenient loss of memory to total loss of consciousness. Transient loss of consciousness, without much in the way of residual signs in the central nervous system, is frequently due to vertebrobasilar ischaemia in the middle aged and elderly, but in the period under review there have been four cases of this type in my practice, in which the diagnosis has been complete heart block with Stokes-Adams attacks, suggested by finding a pulse rate of 40 or less. It is sometimes possible to see canon waves in the veins of the neck, due to the occasional synchronous contraction of atria and ventricles. The diagnosis of heart block was confirmed by electrocardiography. All four patients were aged over 70, and one was a diabetic.

Hiatus hernia and oesophagitis. These conditions can produce chest pains which resemble angina sufficiently closely to cause worrying doubt. An ECG which is normal before and after exercise and remains normal when repeated a week later is very helpful, but the true exclusion of angina depends upon the establishment of correct diagnosis by full x-ray investigation.

Other conditions associated with chest pain. Angina due to

anaemia has already been mentioned. Sudden haemorrhage into the bowel, severe enough to cause collapse, produced a clinical picture resembling infarction, but malaena the following day established the diagnosis.

Dyspnoea and chest pain may be due to pulmonary conditions; one elderly man whose history suggested angina of effort was found to have pneumoconiosis and tuberculosis; and a middle-aged man had apical pneumonia.

An elderly man had pain resembling angina and it was most difficult to get definite answers to questions on the relationship to effort. After two normal ECGs he was sent to hospital for investigation and was found to have osteoporosis of the thoracic spine.

There is a steady stream of patients who have recently lost a near relative with a 'coronary' and who promptly develop chest pains and come for reassurance.

The electrocardiograph in general practice

The ECG is a most useful addition to the armamentarium of the general practitioner who has a special interest in cardiology, and it completely alters the management of disorders of the heart. One would advise, however, that the subject should either be regarded as a special time-absorbing study or left entirely alone; casual electrocardiography is full of traps and pitfalls.

Whooping-Cough: Difficulties in Diagnosis and Ineffectiveness of Immunization. ALLEN T. WILSON *et al. Brit. med. J.* 1965. **2**, 623.

Four general practitioners studied an epidemic of whooping-cough occurring in a Peeblesshire village during 1964. The first case was noted in a village school and the epidemic was traced through the schoolchildren to their families. In all 408 children under 15 were involved in the study, of whom 184 had cough (not necessarily all whooping-cough).

Quarantine regulations were not imposed because of the good immunization state of the children and this probably contributed to the spread of the infection. The protective effect of previous immunization against whooping-cough proved disappointing. Atropine methonitrate was found effective in controlling paroxysms but the effect of tetracyclines, given mainly to younger children, was less impressive clinically.

Pernasal swabs were positive in only 14 per cent of cases and the authors conclude that " All present clinical criteria and laboratory methods are quite inadequate for diagnostic purposes ".