deficiency in the body, the resistance to infection of this type is greatly diminished. It is obviously necessary that this work¹³ should be repeated on a larger scale and extended to the treatment of other infective conditions, for its practical importance, if established, cannot be exaggerated.

What value these results, if established, may have to medical men in Africa is for you to determine. They may point to a method of reducing the high mortality rate in pneumonia among the natives as well as among Europeans. If it does act curatively in this condition, the probability is greater that the inclusion in the normal diet of sufficient quantities of vitamin A will prevent the disease as well as other infections of a similar bacterial nature. Rich sources of vitamin A are egg yolk, mammalian liver, cod-liver oil, and there is some in carrots, green vegetables, milk, butter, and suet. It may be well to add that vitamin A is a very unstable substance and is apt to disappear rapidly, especially in high concentrations.

I have endeavoured to supply you with evidence, both experimental and clinical, which shows how important is this subject of the relation of diet to disease. In particular, I have tried to demonstrate that food includes both harmful and protective constituents. In so far as the harmful substances mentioned are in the food itself, it appears that they can be neutralized in their action by the protective agencies. The protective agencies are of the nature of vitamins, and the two I have specially dealt with are vitamins A and D. The harmful factors are mostly associated with cereals and cereal products so far as present knowledge goes.

I wish especially to emphasize that this subject is only in its infancy, and that many of the facts I have dealt with here have still to be investigated more closely, partly because it is impossible as yet to decide what is

their true clinical significance, and partly because there is no knowledge of the methods by which these substances exert their effect. So far as you in Kenya are concerned, it seems to me that these dietetic problems, especially those concerning the toxic action of cereals, may have special significance, for I cannot believe that your native population with its high cereal diet completely escapes those effects. It is true that the sunshine to which the African native exposes himself must tend to neutralize the anticalcifying effect of maize by supplying vitamin D, but I should expect the native who wears European clothing, especially in childhood, and remains on his maize diet to suffer accordingly. Similarly it seems to me probable that a deficiency of vitamin A might be present in many native diets, especially in the towns, and that pathological results would follow. I shall be interested to hear of any investigations which may be made in the future to see whether these hypotheses have a bearing on any of the problems of disease in Africa.

REFERENCES.

- ¹ Eijkman: *Arch. f. path. Anat. (etc.)*, 1897, 149, 197.
- ² Holst and Frölich: *Journ. of Hyg.*, 1907, 7, 634.
- ³ Mellanby, M.: *Brit. Dental Journ.*, 1927, 48, 1481.
- ⁴ Mellanby, M., and Pattison, C. L.: *British Medical Journal*, 1928, ii, 1079.
- ⁵ Acton: *Indian Med. Gaz.*, 1922, 57, 241, 331, and 412.
- ⁶ Anderson, Howard, and Simonsen: *Indian Journ. of Med. Research*, 1924-25, 12, 613.
- ⁷ Stockman: (a) *Edinburgh Med. Journ.*, 1917, 19, 277 and 297; (b) *Journ. of Pharm. and Exper. Ther.*, 1929, 37, 43.
- ⁸ Goldberger, Wheeler, Lillie, and Rogers: *Pub. Health Rep.*, Wash., D.C., 1926, 41, 297.
- ⁹ Mellanby E.: *Lancet*, 1919, i, 407.
- ¹⁰ Idem: *British Medical Journal*, 1926, i, 515.
- ¹¹ Mori: *Bull. Johns Hopkins Hosp.*, 1922, 33, 357.
- ¹² Green, H. N., and Mellanby, E.: *British Medical Journal*, 1928, ii, 691.
- ¹³ Idem: *Ibid.*, 1929, i, 984.
- ¹⁴ Mellanby, E.: *Journ. of Physiol.*, Proceedings, 1926, vol. lxi.
- ¹⁵ Ungley, C. C., and Suzman, M. M.: *Brain*, 1929, 52, 271.

A British Medical Association Lecture

ON

CORONARY OCCLUSION.

DELIVERED BEFORE THE HARROGATE DIVISION,
NOVEMBER 30TH, 1929,

BY

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It is only recently that the subject of coronary occlusion has attracted the attention of clinicians in this country. Until a few years ago it had generally been regarded as of purely pathological interest, as one of the possible causes of sudden death; but we now know that many of these patients survive, and that the symptom complex is often so clearly defined that the diagnosis during life presents no difficulty.

INCIDENCE.

The condition is by no means rare. During the past two years I have seen 23 cases in private practice. This series,* though small, will, I think, suffice to illustrate most of the clinical features to which I wish to refer. I shall also include one other case (Case i) which presents certain points of special interest. Both the sex and the age incidence in my series agree fairly closely with that reported by other workers.

The preponderance of males over females suggests that mental and physical strain may be important etiological factors. The age incidence coincides with that of the degenerative period of life, and this is what might be expected, since neither syphilis nor rheumatic infection appears to play any significant part in the etiology. It is curious that syphilis is not a more common cause of coronary occlusion, since the first part of the aorta is so frequently involved.

* This series is one of several groups of cardiac cases the after-histories of which are being followed up on behalf of the Medical Research Council.

TABLE I.—Age and Sex Incidence.

	Bramwell.	Levine. ⁸	Parkinson and Bedford. ⁴
<i>Age.</i>			
Over 70	3	14	10
50-69	17	99	68
Under 50	4	22	22
<i>Sex.</i>			
Male	18	111	93
Female	6	34	7
Total	24	145	100

CLINICAL FEATURES.

Let me begin by recounting the clinical findings in two typical cases.

CASE I.

A man, aged 53, who had always lived an exceptionally healthy and abstemious outdoor life, after a hurried lunch went out to play golf. When walking downhill to the sixth green he experienced a sense of fullness and discomfort in the chest. At the next hole, which was an uphill slope, his discomfort increased, and he had to give up and walk home. The pain spread down the left arm to the little finger, and continued to increase in severity in spite of his going to bed. His doctor saw him at 4 p.m. and gave amyl and ethyl nitrite and a quarter of a grain of morphine. This, however, failed to bring any appreciable relief. The pain persisted. During the night it began to involve the other arm, and it was not until the following afternoon that it became easier and gradually passed off.

When I saw him, ten days later, although he had had no recurrence of the pain since the day following the attack, he still looked extremely ill and careworn. He walked and behaved like an old man, obviously afraid that the least exertion might precipitate another attack. On physical examination the heart was slightly enlarged. The second sound was unduly resonant in the aortic area. The retinal arteries showed atheromatous changes. The blood pressure was raised (systolic 180, diastolic 105). The urine contained

albumin. The electro-cardiogram (Fig. 1)* showed the characteristic features associated with coronary thrombosis; but an x-ray examination, beyond confirming the presence of cardiac enlargement, revealed nothing abnormal.

Nearly three years have now elapsed since his attack. During this period he has had no recurrence. Nevertheless he has been compelled to take things quietly, for if he attempts to walk briskly uphill he gets what he describes as a "warning shadow" of the pain in his chest. His blood pressure, when I saw him two months after the attack, had risen to 190/115. It is now 220/130.

In this case I wish to call attention more particularly to two points. First, to the fact that the patient was a man of exceptionally good physique who had lived a careful life, and believed himself to be in perfect health up to the moment that his pain commenced. Except for an operation for gall-stones three years previously he had never had any serious illness. Levine,³ in his comprehensive survey of the subject, goes so far as to say that

"The typical patient is a well-set person, somewhat overweight, often of considerable physical strength, who enjoyed unusually good health. Such patients often have indulged in vigorous physical efforts either in the form of sport or in their ordinary work."

Secondly, I wish to dwell on the character of the pain itself. Pain such as I have described in this case differs from the pain of an ordinary anginal attack. Instead of passing off in the course of a few minutes, it lasted for many hours. The pain was not relieved by rest; in fact, for a time after the patient got home it actually increased, both in intensity and in the extent of its distribution. The pain was not relieved by nitrites, and only partially by morphine. Unlike angina, the pain of coronary thrombosis is just as liable to come on when the patient is resting. In several of my cases the attack occurred when the patient was lying quietly in bed at night, or before getting up in the morning. This is in striking contrast to the typical anginal attack, which is brought on by exertion or emotion, and relieved by rest. Further, instead of remaining absolutely motionless and afraid to move during the attack, the patient often tosses or walks about, for he discovers that rest does not relieve the pain.

Most frequently, in my series, the pain started either at the level of the lower end of the sternum, or in the epigastrium, though in one case it was first felt in the jaw, and in another in the region of the larynx. It often spread right across the chest. It varied greatly in intensity, and in the milder cases was sometimes described as a feeling of tightness and discomfort which prevented the patient taking a deep breath. The pain often spreads down the left arm, and in six of my cases the right arm was also involved.

CASE II.

A gardener, aged 70, who had never had occasion to consult a doctor, though he had been somewhat short of breath for about twelve months, was walking to his work, a distance of half a mile, one Thursday morning, when he suddenly experienced a sharp pain, strictly localized to a small area in the middle line at the level of the lower third of the sternum. The pain passed through to the back, but did not radiate to other parts of the chest nor to the arms. The day was windy, and he was walking uphill when the pain came on. He rested and returned home, having to stop at intervals on the way to obtain relief. On reaching home he vomited. After resting the pain gradually passed off. He had some lunch at noon, and felt sufficiently well to go back to work. On the way he experienced slight discomfort in the same position as before. When he had almost reached his destination the severe pain came on again, and again he vomited. He took things quietly

for the next couple of hours, and the pain "mended" a little. About 3 o'clock, however, whilst he was potting some plants, it became worse, and he went home to bed at 6.30. His doctor saw him shortly afterwards, and found him collapsed and vomiting watery fluid. The liver was enlarged and very tender. The pain and vomiting continued all night. On Friday the pain was easier, and by midday it had quite passed off. On Friday and Saturday his temperature was slightly above normal; and on Sunday morning he coughed up a little blood.

When first I saw him, at noon on Sunday, his pulse was barely palpable, and he had auricular fibrillation, with a heart rate of 160. The blood pressure was about 110/90. The liver was still slightly enlarged and very tender, and there was some general rigidity in the abdominal muscles. He was lying flat in bed, and there was no dyspnoea.

On the following Thursday—a week after the attack—he was admitted to hospital. In the interval there had been no recurrence of the pain, and his general condition was much improved. The pulse was now quite strong and regular, the heart rate being 96 and the blood pressure 135/95. The electro-cardiogram showed ventricular deflexions of low voltage with a large negative T wave in Lead III. Examination of the blood showed a leucocytosis of just over 10,000, with excess of polymorphs.

On Sunday, when his wife came to visit him, she remarked how well and cheerful he was. About 6.30 the following morning, however, when the nurse went to make his bed, she thought at first he was asleep, but found that he was unconscious and cyanosed, with Cheyne-Stokes respiration. The radial pulse was not palpable. She administered a stimulant, and his condition had already improved when the house-physician arrived a few minutes later. His pulse was then quite good, 70 and regular, though he had not regained consciousness. When doing his morning round at 10.30 the house-

physician noticed that he had again collapsed, never having completely regained consciousness in the interval. He died a few minutes later. Post-mortem examination showed that death was due to rupture of the ventricle at the site of the infarct.

This case serves to illustrate certain other features which are highly characteristic of coronary occlusion. The attack is frequently accompanied by intense shock and prostration, due to a sudden fall in blood pressure. This is obviously a very different state of affairs from the ordinary anginal attack, in which, if the

blood pressure changes at all, it tends to rise. In an anginal attack the rate and rhythm of the pulse remain unchanged, whereas after a coronary thrombosis the patient may be pulseless, or, if the pulse be palpable, it is often very rapid or irregular. There is frequently a combination of pallor and cyanosis, which gives to the face a peculiar appearance.

Quite apart from the character of the pain and the shock and collapse associated with it, however, there are other features which serve to differentiate coronary occlusion from true angina. As in this case, vomiting often accompanies the pain. It is a symptom which is apt to be misleading, for when the pain is confined to the epigastrium, as is not infrequently the case, the condition closely simulates an acute abdominal emergency. The presence of abdominal rigidity helps to reinforce this impression. The abdomen has often been opened in error in cases of coronary occlusion. This is perhaps hardly surprising, for without an electro-cardiogram the differential diagnosis may occasionally be very difficult. The occurrence of dyspnoea should arouse suspicion, for when epigastric pain, vomiting, collapse, and abdominal rigidity are associated with dyspnoea, in a patient past middle age, the possibility of coronary occlusion should always be considered.

Coronary thrombosis not infrequently occurs in patients who have previously suffered from ordinary paroxysmal angina, though, as in Case i, it may come "like a bolt from the blue" to a man who had apparently been in perfect health up to the moment of the seizure. When, however, a patient who has previously suffered from angina

FIG. 1.—Two records taken from Case i on March 28th, 1927 (a), and May 16th, 1927 (b), showing change in form of ventricular complex.

* All the electro-cardiograms in this paper were taken with a string sensitivity of 2 cm.=3 mv.

develops coronary occlusion, he generally realizes at once that this attack is something quite different from what he has previously experienced. In 8 of the 24 cases in my series the cardiac infarction had been preceded by ordinary anginal symptoms.

Another sign of diagnostic value is transient pericardial friction, which may be heard over the site of the infarct. I met with this sign in three only of the cases in the present series, but it is probable that the true incidence is really considerably higher, for friction may disappear in a few hours, and often I myself only had the opportunity of examining the patient once, and that not until several days after the attack.

ATYPICAL CASES.

When pain is not a prominent symptom the diagnosis is apt to present more difficulty. In the two following cases in my own series I should have been in doubt as to the diagnosis had it not been for the electro-cardiographic findings.

CASE III.

An unmarried woman, aged 50, was sent to see me by one of my gynaecological colleagues whom she had consulted on account of amenorrhoea, under the impression that her symptoms might be due to the menopause. She gave a peculiar history, that for precisely one week she had suffered from tightness in the chest and dyspnoea on exertion. It was difficult to account for the sudden onset of these symptoms, and no cross-examination would shake the patient as to the date of their appearance.

On examination the heart was considerably enlarged. The blood pressure was 250/130; the resting heart rate was 120, with typical gallop rhythm. The electro-cardiogram afforded the explanation of the unusual history, the features being characteristic of coronary occlusion.

This patient kept surprisingly well for eight months after I saw her. Subsequently, however, she became extremely emotional, and spent a large part of her time weeping, a mental state which I have noted in two other cases, and am inclined to attribute to the associated cerebral arterio-sclerosis.

CASE IV.

A commercial traveller, 40 years of age, who to the best of his knowledge had previously been in perfect health, was endeavouring to start his motor car one cold morning. He struggled with it for nearly half an hour, and then had to give up on account of dyspnoea and some discomfort in the chest. After resting a quarter of an hour he felt better, and tried again. This time he stuck it for about a quarter of an hour and again collapsed exhausted, the pain being more severe. His doctor, who saw him shortly afterwards, thought that he was going to die. He was intensely cyanosed and shocked. The pulse was barely palpable. All the following day the heart was extremely irregular, but normal rhythm was restored forty-eight hours later, and it was not till then that the pain passed off.

When I saw him four days later the slightest movement provoked respiratory distress, and he still complained of a good deal of pain in the left chest, passing through to the angle of the scapula. His heart rate was then 54 per minute, and the blood pressure only 85/50. He was admitted to hospital five days later, and the electro-cardiogram shown in Fig. 2a was taken.

In this case it was difficult to account for the sudden onset of heart failure in a man who, up to the time of his attack, had apparently been in good health.

DIFFERENTIAL DIAGNOSIS.

Coronary occlusion is apt to be mistaken for various other conditions. To two of these I have already referred in some detail—namely, ordinary angina and the acute abdominal crises such as perforated peptic ulcer, acute cholecystitis, and acute pancreatitis.

All erroneous diagnosis of diabetic ketosis is not unknown. This is perhaps less surprising than might appear at first

sight, for not only is transient glycosuria a common manifestation after an attack, but coronary occlusion not infrequently develops in patients suffering from true diabetes. Levine noted the presence of glycosuria or actual diabetes in 34 of the cases in his series, and two of my patients were diabetics. Lastly, those patients who develop pulmonary oedema with moist sounds at the bases of the lungs are sometimes thought to be suffering from pneumonia, and it is important to remember that in some cases dyspnoea, not pain, is the principal symptom.

BLOOD PRESSURE.

When one comes to analyse the blood pressure readings in these cases, one cannot fail to be struck by the figures obtained.

TABLE II.—Blood Pressure.

	Cases.	Died.
Normal values	3	1
High systolic (180 or over) after attack ...	7	4
High systolic (200 or over) before attack ...	3	0
Low systolic (110 or under)	7	3
High pulse pressure (both had aortic incompetence)	2	1
Low pulse pressure (150/115 and 140/110) ...	2	2
Total	24	11

It is interesting to follow up the after-histories of patients who had hypotension when I first saw them. Of the seven, three have died. In the remaining four cases the blood pressure subsequently attained the normal level, though in one case (Case iv) it took eight weeks to do so. It is noteworthy that the three cases with the lowest readings all did well. They were the three youngest patients in my series.

A considerable fall in pressure at the time of the infarction is the rule, and in some of the cases, where prior to the attack the pressure was high, it never again attains its former level. This may be advantageous to the patient, for, as I have already mentioned, coronary occlusion is apt to

supervene in patients who have previously suffered from angina, and as the result of the fall in pressure these patients may remain free from anginal attacks after the coronary thrombosis. On the other hand, there is a less fortunate group—patients who prior to the thrombosis had enjoyed good health, and who after the attack suffer from paroxysms of ordinary angina.

TABLE III.

Blood Pressure when first seen.	Age.	Sex.	Subsequent Readings.	Remarks.
105/65	50	F.	135/95	Fairly well; 20 months.
110/90	70	M.	135/95	Died, ruptured ventricle (Case ii).
110/90	58	M.	—	Died three months after attack.
110/90	71	F.	—	Died in attack.
85/50	40	M.	100/65, 120/75	Well; working 7 months (Case iv).
80/60	37	M.	120/75, 140/90	Well; working, 12 months (Case vi).
Not obtainable	39	M.	135/95	Convalescent; 4 months (Case v).

ELECTRO-CARDIOGRAPHIC FINDINGS.

I have already referred to the fact that certain changes occur in the electro-cardiogram in many, though by no means in all, cases of coronary thrombosis. Let me now amplify this point.

FIG. 2.—Records taken from Case iv on May 15th, 1929 (a), and January 7th, 1930 (b), showing change in T wave.

In 18 cases in my series I was able to obtain an electro-cardiogram. One case had right branch bundle-block, and another incomplete bundle-block. Eleven exhibited certain special features which are of peculiar interest and importance, for it has been shown by Smith³ that they can be produced experimentally in the laboratory by ligation of one of the coronary arteries. In health the form of the electro-cardiogram remains remarkably constant in any one individual. After myocardial infarction, however, striking changes occur. The T wave starts before the initial rapid ventricular deflexion has returned to its base line. It "takes off" at an abnormally high level. Or perhaps it would be more accurate to say that the R-T interval becomes convex. In its extreme form this type of ventricular complex is absolutely pathognomonic of myocardial infarction, though minor degrees of variation from the normal, in which the T wave takes off slightly above the base line, have been met with in other conditions. Levine³ has published records from cases of pneumonia and uraemia which exhibit this peculiarity, and Hoogerwerf, Ellis, and I² obtained similar records from athletes who were competing in the Olympic Games at Amsterdam. As time goes on the record again changes, the T wave becoming negative (Fig. 1).

Another curious feature in many of these records is seen in Lead III, where the initial ventricular deflexion is of large amplitude and of negative sign (Figs. 1 and 3). This

FIG. 3.—Records taken from two cases in present series showing characteristic form of ventricular complex with large negative T waves and initial negative deflexion in Lead III.

feature is not peculiar to coronary thrombosis; I have seen it occasionally in records from patients with old-standing high blood pressure.

PROGNOSIS.

There is perhaps no disease in which an accurate prognosis is more difficult than in coronary thrombosis. Eleven of my 24 patients have died, 3 from the effects of shock in their first attack, and 3 others in subsequent attacks. Those patients who survive their first attack are liable to have further attacks, any one of which may prove fatal. Hence the possibility, if not the probability, of sudden death must always be borne in mind in framing the prognosis. Sudden death in these cases is probably due to ventricular fibrillation. In patients who escape this fatality, the profound shock associated with the infarction is the most grave immediate danger. One should not, however, despair of recovery even in those cases which are most profoundly shocked. The most extreme example I have seen was the following.

CASE V.

A mechanic, aged 39, who had previously been in good health, rushed out one evening after a hurried meal to play in a tennis tournament. During the game he became dizzy, collapsed, and cyanosed. He managed to struggle home. When his doctor saw him his temperature was 95°, and he was intensely shocked and collapsed, and complaining of pain across the lower part of the chest. His pulse was 140. He vomited at the commencement, and several times subsequently. The following morning he developed a loud pericardial rub, and when I saw him, eighteen hours after the onset of his illness, he was still clammy and collapsed. No pulse could be felt in the radial or in the brachial arteries. The heart rate was 228, and its rhythm regular. Pericardial friction was audible all over the precordium. He appeared to be on the verge of death. For the next twenty-four hours his condition

seemed hopeless, but on the following morning his heart rate suddenly dropped to 70, and he began to show signs of recovery. He then gradually improved, though it was not until ten days after his attack that he became fully conscious of his surroundings. Three months later he was sufficiently well to be brought up to see me for re-examination.

Since seeing this patient I have hesitated ever to give an absolutely hopeless prognosis in coronary occlusion, no matter how desperate the condition of the patient might appear. In those patients who get over the initial stage of shock there still remains the danger of rupture of the heart. This complication may arise most unexpectedly, as in Case ii in my series. Though rupture generally occurs within a fortnight of the infarction, it is probable that cicatrization of the scar is not complete for another three or four weeks. For this reason alone the importance of complete and absolute rest during the early weeks of the illness is apparent. In a considerable percentage of cases death results from progressive heart failure of the congestive type, with or without auricular fibrillation. The other four deaths in my series were due to this cause.

Finally, there is one other accident which merits attention—namely, embolism. When infarction has taken place, a mural thrombus forms on the infarcted area. Portions of this thrombus are liable to separate off and give rise to embolism in the brain or elsewhere.

During and immediately after the attack prognosis is exceedingly difficult. There does not appear to be any one symptom or sign which affords a reliable guide. The degree of the initial shock and the extent of the fall in blood pressure might be expected to indicate the ultimate prospect, but, as I have already pointed out, this is not invariably true; some of the most desperate cases make a truly surprising recovery. There is, however, in this connexion one finding which in my experience is of grave significance—namely, the presence of a high diastolic associated with a low pulse pressure. As I have pointed out elsewhere,¹ I regard the systolic pressure as a measure of the heart's capacity to meet its liabilities. A high diastolic pressure greatly impairs the elasticity of the arteries. It increases the heart's liabilities, and necessitates a considerable elevation in the systolic pressure if the cardiac output is to be maintained. In these circumstances a fall in systolic pressure means a diminished output and a failing heart muscle.

On the optimistic aspect I am inclined to think that the age of the patient should be regarded as a factor of considerable importance. My three youngest patients are interesting. They were all very severe cases, and all three have done unexpectedly well. To two of them (Cases iv and v) I have already referred. The history of the third is as follows.

CASE VI.

An estate agent, aged 37, was suddenly seized with severe substernal pain while still in bed one morning. His doctor found him intensely collapsed, ashen in colour, and pulseless. He vomited. The pain spread to the region of the cardiac impulse and down the inner side of the left arm. By 11 a.m. it had become less severe, but was still present on the two following days, in spite of repeated injections of morphine. Nearly a fortnight later pericardial friction was noticed for the first time, to the right of the sternum. It was only present for about twenty-four hours and was accompanied by a slight rise in temperature. On the day of the attack the systolic blood pressure was 80, but when I saw him a fortnight later it had risen to 120, with a diastolic pressure of 75, though the first heart sound was still barely audible at the apex. The superficial vessels were not thickened, but his retinal arteries were unduly tortuous. An electro-cardiogram taken three months later showed a P-R interval of more than 0.2 second with a small wave in the ventricular complexes immediately preceding the rapid deflexions. The heart's rhythm remained normal throughout.

The patient's doctor now informs me that he has had no recurrence of the pain since his original attack, twelve months ago, and is able to attend to most of his office work, and to drive his car. In fact, he is remarkably fit.

This patient was a very heavy smoker, and admitted to fourteen ounces of tobacco a week. It seems possible that excessive smoking may have played some part in producing his arterial degenerative changes. On the other hands there was a familial tendency to arterial disease, for an uncle died of angina at the age of 30, and his father died

of a heart attack in the forties. The patient himself, though otherwise healthy, had always been somewhat short of breath on going uphill.

TREATMENT.

I know of no drug but morphine which will relieve the pain of coronary thrombosis, and it must be given freely. A quarter of a grain is generally insufficient, and my own practice, in severe cases, is to give an initial dose of half a grain hypodermically. When the blood pressure is low active stimulation is indicated, and apart from the relief of pain the treatment resolves itself into that of "shock"—keeping the patient warm, raising the foot of the bed, giving brandy or other stimulants by mouth, and camphor subcutaneously. I have tried ephedrine in some cases, but am not fully convinced of its value. Measures such as these may tide over the danger period. Let me repeat that, if there is one group of drugs more strongly contraindicated than any other in these cases, it is the nitrites. Continuous oxygen inhalation may be helpful when oedema of the lungs is present, though it cannot be expected to do good when the cyanosis is attributable solely to a diminished cardiac output.

The rapid heart action associated with auricular fibrillation, or paroxysmal tachycardia, which sometimes complicates coronary thrombosis, may in itself endanger life. In the former digitalis given by the massive dose method of Eggleston or intravenous strophanthin is called for; while the only drug which seems to have any effect in arresting the paroxysms of tachycardia is quinidine; six grains may safely be given, and repeated, if necessary, at two-hourly intervals for five doses.

To the importance of complete rest I have already referred. After a severe attack of coronary thrombosis the patient should not be allowed even to sit up in bed for two or three weeks.

CONCLUSION.

In the short time at my disposal I have only attempted to deal with the more important practical points in connexion with coronary occlusion. I have endeavoured to show that the study of coronary thrombosis is not confined to the post-mortem room. It is a definite clinical entity, which, in a large percentage of cases, can be recognized with certainty during life. Its recognition is important, because many of the patients survive, and a few are sufficiently restored to health to resume their former occupations. Further, a correct diagnosis is of vital importance, not merely in framing the prognosis, but also in determining the treatment. An acute coronary occlusion may closely simulate an abdominal crisis, and if unrecognized may be submitted to laparotomy, which, in view of the cardiac lesion, is almost certain to prove fatal. Other cases are mistaken for ordinary angina and treated with nitrites—a dangerous procedure in view of the low blood pressure.

The crescendo character of the pain, its duration, the fact that it is not relieved by rest and that the attack frequently occurs when the patient is resting and is in no way attributable to exertion, are important. The fall in blood pressure, the shock, prostration, and vomiting which generally accompany the pain, serve to distinguish it from true angina, as do the feeble first heart sound, the gallop rhythm, the rapid pulse, and the pericardial friction which are sometimes present. I need hardly say that one must not expect to find all these signs in every case, but there is generally ample evidence, even without the electro-cardiograph, which, if it be available, will often clinch the diagnosis.

The aspect of prognosis is so difficult that I almost hesitate to refer to it, though my own experience would lead me to believe that even the most desperate cases may recover, especially when the patient is under 50 years of age.

REFERENCES.

- 1 Bramwell, J. C.: *Quart. Journ. of Med.*, 1924, xvii, 225.
- 2 Hoogerwerf, S.: *Sportärztliche Untersuchungen bei der IX Olympiade*, 118, Berlin, Julius Springer, 1929.
- 3 Levine, S. A.: *Medicine*, 1929, viii, 245.
- 4 Parkinson, J., and Bedford, D. Evan: *Lancet*, 1928, i, 4.
- 5 Smith, F. M.: *Arch. Int. Med.*, 1918, xxii, 8.

THE CORONARY ARTERIES IN A CASE OF FAMILIAL LIABILITY TO SUDDEN DEATH.*

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WE publish the following record in the hope of eliciting from others, and especially from family practitioners of long experience, notes of similar cases.

In 1924 Carey Coombs and J. J. S. Lucas described to the Association of Physicians a family of whom the father and two sons had died with great suddenness. They had examined the heart of one of these sons, a man aged 32, who had been taken ill while playing football and had died within an hour or so; it was found to present macroscopical changes apparently of an atheromatous type in the coronary arteries. These changes had not been histologically examined as the specimen had been lost. They also gave particulars of a third son who had been seen by them for symptoms suggesting angina pectoris.

This man was first seen in 1923. The history was that in 1918, during exercise, he complained of an uncomfortable tight feeling across the chest. This passed off, and it was not until 1923 that

FIG. 1.

the attacks became at all frequent. He then stated that for the previous ten months the attacks had been getting more frequent, and were brought on more and more readily, till in the latter part of the year he could not run ten yards without experiencing a tight gripping sensation in the chest. He never became dyspnoeic because the feeling came on before he had time to get out of breath. It never occurred at night, but was more easily brought on after a meal.

In May, 1925, one of us (C. E. K. H.) was asked to take an electro-cardiogram. (See Fig. 1.) The following is a genealogical tree of the family.

Heavy type indicates affected members.

In October, 1927, the patient was sent to see one of us (C. E. K. H.). He stated that the attacks were now associated with pain. A sudden tight feeling over the sternum was associated with an acute pain in the same region which was quite stationary,

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