CLINICAL LECTURE
on
A CASE OF CORONARY THROMBOSIS.

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The case I wish to bring before you to-day is that of a male, single, aged 58, who was admitted to the General Hospital, Johannesburg, at 6.45 p.m. on the 4th June, 1928.

He was born in England, came to South Africa in 1897, and worked as a horticulturist, pruning trees being his special line. He stated that while riding in a bus he was suddenly seized with an intese burning pain on the left side of the chest in front. He got out of the bus and lay on the ground, groaning and rolling about in agony. He had difficulty in drawing his breath because of the pain, which radiated up the chest and down both arms, but chiefly the left, which also felt weak and numb. He was taken to a chemist and then to the hospital, still in great pain. The pain remained intense for three and a half days, though eased temporarily to some extent by hypodermic injections of morphia. The impression he had given was that he was not as ill as he claimed to be, and, in fact, he was regarded as playing up about the pain and was receiving very little sympathy from the staff. In the past six months he had been having slight attacks of pain in the chest while walking. Recently he had been troubled with slight giddy turns. He stated also that he had become easily fatigued lately, both mentally and physically, and was aware of palpitation at times.

He gave a history of a great deal of malaria and blackwater fever. He had nephritis during the Anglo-Boer War.

As far as his habits were concerned he chewed tobacco considerably, had been a heavy drinker, and had led a strenuous life.

For the first day or two of his pain his face shewed anxiety and was ashen in hue, and he was sweating though the weather was cold. Thereafter his colour returned, and he looked a well-nourished, healthy-looking, man.

His temperature on admission and for twenty-four hours after was sub-normal. Next day it rose to 100.2° F., and for six days he ran an intermittent temperature varying between 97° and 100°. The pulse rate, which was about 80 on admission, quickened, but not beyond 116 per minute. There was an occasional intermission of the pulse which, on auscultation of the heart sounds, seemed to be associated with a premature beat, probably ventricular in origin. Nothing abnormal otherwise could be detected in the examination of the heart except that on percussion the left border seemed just outside the mid-clavicular line. The blood pressure was 105 Systolic and 70 Diastolic. On the 16th July, 1928, the blood pressure was 80 Systolic 50 Diastolic, on 18th July, 1928, 90 Systolic 55 Diastolic. The Radial Arteries were a little thickened and tortuous. The Lungs, Abdomen, and Nervous System showed nothing abnormal. The urine was normal except for a few pus cells.

A Blood Count showed:—

Haemoglobin, 84%.
Colour Index, 1.
Red cells per c.mm., 5,020,000.
Leucocytes per c.mm., 26,200.
Polymorphonuclears, 88%.
Lymphocytes, 12%.

Smears for Malarial parasites were repeatedly negative.
The Wassermann was negative.
Blood urea 29 mgs. per 100 c.c. blood.
Urea concentration :—

Specimen 1 — 3.1% Urea.
Specimen 2 — 1.7% ,,
Specimen 3 — 3.6% ,,

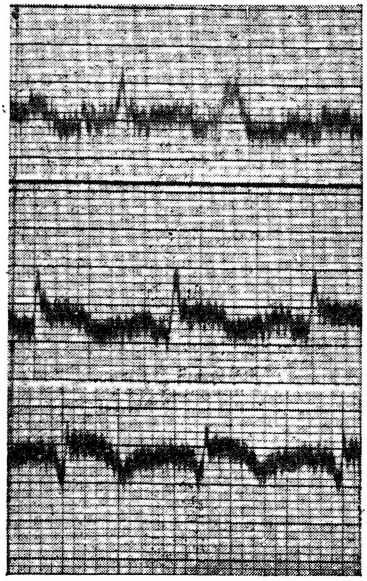
An X-ray of the chest showed some uniform enlargement of the Aorta, but no definite evidence of Aneurysm.

On the 11th June, the appearance of the patient was excellent. He was still running a slight temperature, and that day for the first time pericardial friction was detected over a limited area, the size of a five-shilling bit, internal to the left nipple. Next day this could not be detected. Nor has there been any since.

From the character of the pain, its site, radiation and persistence, the presence of the signs of shock at the beginning, i.e., the ashen colour of the patient, the lowered temperature, the cold sweats, the low blood pressure; from the pain requiring morphia injections repeatedly for its relief, succeeded presently by the reaction stage with the improved appearance of the patient, the temperature and the polymorphonuclear leucocytosis, a diagnosis of Coronary Thrombosis, or Cardiac Infarction, was made.

On the 12th June, an electrocardiographic record was made. This shows the plateau type of curve between the R. and T. with inversion of the T-wave in the 2nd and 3rd leads. The T-wave is deeper in the 3rd than in the 2nd lead. Electrocardiographic records have been taken in this case a month and two months later. They show this marked inversion of the T-wave in the 2nd and 3rd leads, still deeper in the 3rd and very pointed, but the plateau portion has disappeared. These changes are exactly as described by Parkinson and Bedford recently (1).

12/6/28.



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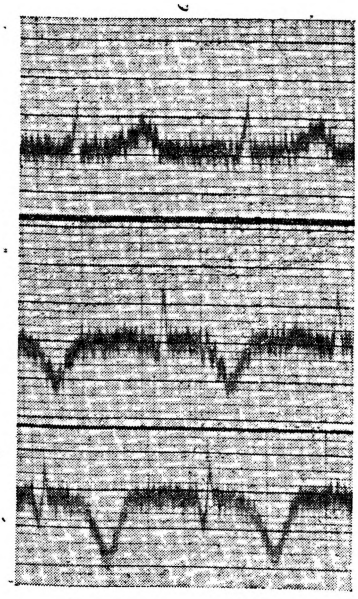

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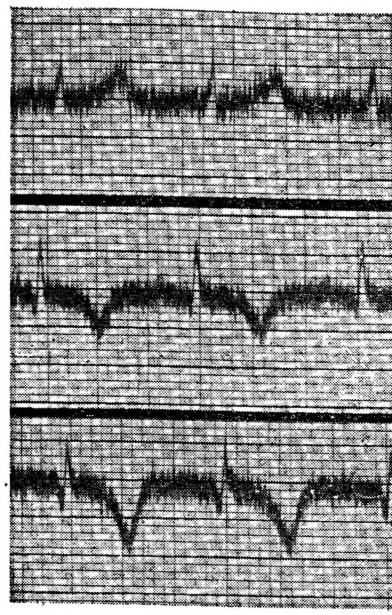

These two men deal with the condition of Coronary Thrombosis from the historical, pathological and clinical point of view. They bring out the point that it was only in 1910 that the distinction between Coronary Thrombosis as a clinical entity and Angina Pectoris was made for the first time. The credit for this was given to Obratzow and Strachesko, though apparently the first case of Coronary Thrombosis to be diagnosed correctly during life was by Hammer in 1878. The pathology of cardiac infarction they associate with atheroma of the coronary arteries, the final occlusion of the vessel being usually due to thrombosis at the site of the constriction. Only eight of their eighty-three cases examined pathologically were associated with syphilis. In fifty-one of their cases of recent infarction old infarcts were present in addition. The left coronary artery was more frequently involved than the right and oftenest the anterior descending branch of the former. The infarcted area in that case involves the apex, anterior wall of the left ventricle, anterior part of the inter-ventricular system, and possibly an adjacent strip of the right ventricle.

They bring out the point that the infarcted area is more extensive on the endocardial aspect than on the pericardial. The latter may escape, and this may account for the absence of pericardial friction in many cases. In fourteen of their eighty-seven cases examined post mortem, there was intra cardiac thrombosis over the area of necrosed muscle. In their 100 clinical cases this complication occurred only in eight, or rather evidence of it in emboli was obtained only in that number. Of their 187 cases recorded, 90% were males. The maximum age-incidence was between 50 and 70 years, though also common after 40. Clinically they divide their cases into three groups:—

1. Sudden death at onset.
2. Prolonged anginal pain with shock.
3. Dysopnoea and failure with severe pain.

Dr. Sutherland-Strachan, the pathologist at the General Hospital here, informs me that he has rarely seen examples of coronary thrombosis in the post mortem room in Johannesburg. The reason for this is, no doubt, that in all cases of sudden death, where the practitioner is unwilling to grant a death certificate, the body is taken to the Government Mortuary for the District Surgeon to deal with. It would be interesting to know the incidence of cardiac infarction discovered there.

Our case under discussion belongs to Parkinson's second group and may be taken as a fairly typical example of this syndrome. The branch affected is probably the anterior descending branch of the left coronary, if the site of the pericardial friction is of any significance. The occurrence of friction may be indicative of a fairly extensive infarction, and this is borne out by the patient's markedly diminished response to effort to-day. Breathlessness and pain still pull him up readily.

In the last four years I have seen ten other cases of clinical Coronary Thrombosis. Nine were males. Two are dead, dying within a month or two of the onset of the symptoms. Of two I have lost trace. Three are carrying on work in a more restricted manner, one as a farmer, another as a chemist and the third as a general practitioner; the two former for nearly four years, now, and without recurrence of symptoms, beyond the shortness of breath and constriction in the precordia, associated with exertion beyond a certain limit. In these three cases the only evidence of sepsis was oral. In two the repeated seiures of pain disappeared only after the teeth had been extracted.

The present case seems another such, as he had a high dgree of oral sepsis, without evidence of any other focal infection. Examination for Malaria was repeatedly negative. But, on the other hand, he chewed tobacco considerably and had drunk alcohol excessively for years. Tobacco angina has to be considered. Moschcowitz (2) deals with four cases suggesting tobacco angina. He states that the pain ceased only when they no longer smoked, and that the pains are more intense and of longer duration in tobacco angina than in true angina. In this respect, of course, it resembles the pain in Coronary Thrombosis, but he expressly states that there is no change in facial colour. The change of colour from that of health to an ashen hue during the pain and shock, and then in some hours or a day or two to normal again, is a striking feature in the syndrome of Coronary Thrombosis. In Tobacco Angina, uncomplicated by infection or Coronary Thrombosis, there is no rise of temperature and leucocytosis, nor electrocardiographic evidence of a myocardial lesion, all of which are present in this case,

Ordinary Angina Pectoris has to be considered in the differential diagnosis. In this patient's case the onset of pain was during rest, in ordinary Angina it is associated with exertion as a rule and lasts only a few seconds or minutes, while in infaretion it often lasts hours or days, as in this case. Ordinary Angina is not associated with shock as this case was. There is no fever in ordinary Angina; here fever followed on the second day. The blood pressure fell in this case; in ordinary Angina it may remain normal or rise. Finally the electrocardiogram was characteristic of cardiac infarction.

The treatment was absolute rest in bed. We kept him in bed a month. Morphia grain ¼ repeated whenever the severity of the pain justified it. In this case it was necessary for the best part of a week. In one case, four years ago, the pain was so agonising and continuous that even ½ grain of Morphia Hyd. gave little or no relief, and on several occasions the inhalation of chloroform sprinkled on a mask was resorted to by his doctor, with success. But as a rule morphia is sufficient. In this case there were no signs of cardiac failure with the patient at rest

the rate remained moderate and no abnormal rhythm developed, beyond an occasional premature ventricular beat. Digitalis was not indicated.

As the only indication of infection in his case was the oral sepsis and it was possibly streptococcal, he was given injections of cacodylate of sodium, and at the end of the month his teeth were removed under ether and oxygen, an operation which he stood very well. He is now convalescent and has pain only when he walks hurriedly. It would be unwise for him to return to work for some months.

Prognosis :—This is doubtful. He may die from cardiac failure any time. He may get another infarction. He may live for an indefinite number of years if he leads a restrained life. One case of Parkinson's lived 11½ years after his attack.

P. D. White (3) states that patients often survive for years in good or fair condition. The average duration of life in his 62 cases, half of whom were still alive when he wrote, was close to two years. Sex, age, and hypertension seem to make little difference to the prognosis, while poor heart sounds and signs of congestive failure add to the gravity of the case. White states that the electrocardiogram did not help in prognosis. Complete heart block was a bad sign. Intra ventricular block was more common in the survivors in his cases.

References.

(1) Parkinson and Bedford.—The Lancet, Jan. 7, 1928. P. 4.

(2) Moschcowitz.—J.A.M.A., March 10, 1928. P. 736.

(3) White, P. D.—J.A.M.A., Nov. 6, 1926. P. 1525.

PYORRHŒA.

Marcus Berman, L.D.S., R.C.S. (Eng.).

Pyorrhœa is a disease found in the oral cavity and associated with the teeth and its adjacent tissues. Literally, pyorrhœa means "pus flowing" and consequently our modern day practitioners interpret or diagnose any condition of the teeth where pus is present as pyorrhœa. One may even go so far as to say that many ascribe the name Pyorrhœa to any reasonably severe inflammatory condition of the gums.

The dental surgeon would define it as a disease which essentially involves the periosteum of the tooth root (called the periodontal membrane) and its adjacent tissues—the gum and alveolus. Later, more extensive areas of the oral mucous membrane may or may not be involved. These tissues may, however, become affected by other causes and bearing this in mind the author offers this effort in the hope that a more exact and ready diagnosis may be made.

In view of the unfortunate discord between the word and its meaning attempts have been made to reconcile the two and various terms have been evolved to denote it. The British school refer to it as Generae Chronic Suppurative Periodontitis, whereas the American school favour Periodotaclasia. In addition, others claim that Pyorrhœa Gingival appropriately describes the disease in that it is said to commence in the Gingival Trough. But none of these have proved happy synonyms and the word most current is still Pyorrhœa. One must bear in mind, however, that Pyorrhœa (pus flowing), is a symptom of General Chronic Suppurative Periodontitis and is not in itself a disease entity, though custom and use have made it so.

Anatomy.—A brief description of a tooth and its anatomical relations is necessary to follow this paper more clearly. (See Fig.) The tooth shown is an erupting single-rooted tooth in situ.