

pleurisy, tuberculosis of tracheo-bronchial lymph glands, bacillary dysentery, arterio-sclerosis of the aorta and coronary arteries, and rupture of the aorta due to arterio-sclerosis to produce a dissecting aneurysm of the last part of the transverse and of the descending aorta.

#### Comment

According to Osgood, Gourley and Baker (*Ann. Int. Med.*, IX, 1398; April 1936), although over 400 cases of dissecting aneurysm have been recorded in the literature, only 11 of these have been diagnosed during life. The same authors report Shennan as finding only 6 acceptable cases diagnosed during life until 1933. Five more cases have been reported and these three also reported 2 cases; ours bringing the total to 14 cases diagnosed during life. The following brief résumé of this condition has been taken from the above-mentioned report:—

The aetiology is usually hypertensive cardiovascular disease although some few cases have occurred with coarctation of the aorta and one with a basophile adenoma of the pituitary. The onset is usually determined by some sudden rise in the already existing hypertension. The essential pathology is a rupture of the intima with a splitting of the media. In most instances a secondary rupture occurs externally but in a few cases back into the lumen of the aorta. This secondary rupture is the cause of death in 95 per cent cases. The initial rupture is in the ascending aorta in most cases, about 20 per cent occur in the transverse aorta and 10 per cent in the descending aorta. The length of the dissection varies from a few centimetres to the full length of the vessel.

Clinically the onset is usually with a sudden, severe, tearing pain in the chest coming on after some excitement or strain and in a patient who has had previously evidences of hypertension or coarctation of the aorta. The pain usually centres under the upper sternum and radiates to the back, abdomen, shoulders or arms. It is severe, not readily controlled and is accompanied by extreme restlessness. There may be sudden exacerbations. Evidences of the previous existing hypertension or coarctation will be present and also there may be signs of obstruction of one of the aortic branches (coma, hemiplegia, anæmia, gangrene, paralytic ileus or localized swellings). Sudden death supervenes within a few minutes in 65 per cent cases and in the remainder from a few days to several months. The most suggestive laboratory change is an increase in the icteric index. The roentgenographic features are a diffuse widening of the aortic arch with diminished pulsation, an enlarged heart, displacement of the œsophagus and trachea to the right and shadows of the dissection along the aorta. There may also be fluid at the left base. Diagnosis should not be difficult if the condition is kept in mind and it is based on the history, the pain, and the

laboratory and x-ray findings. The condition usually confused is coronary occlusion. The icteric index, the x-ray and the character of the pain with absence of pericarditis and typical electrocardiographic findings will help to differentiate. The prognosis is grave and the treatment symptomatic.

In the case reported no history of pain was obtained. The condition had probably been pre-existent before admission and the betterment of the circulation attendant upon the use of digitalis in the congestive heart failure probably increased the extent of the dissection. Death was due to an intercurrent bacillary dysentery.

#### Summary

A case of dissecting aneurysm of the aorta, diagnosed during life by the roentgenographic findings and confirmed by autopsy is reported.

### HÆMATEMESIS IN A CASE OF MALARIA\*

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K., aged 32 years, Hindu male, was admitted into the Sambhunath Pundit Hospital on 25th April, 1937, with the history of having vomited blood three times during the previous night and passed blood per rectum.

*Condition on admission.*—Temperature—98.2°F., respiration 24 and pulse 82 per minute. Volume—not good. Extremities—cold. Malæna once in my presence. Abdomen moving with respiration. Slight tenderness in the upper part of the abdomen. Spleen and liver not palpable. History of indigestion with pain in the abdomen for some time. Nothing abnormal detected in the lungs. No history of fever previous to admission. No difficulty in micturition, urine clear.

On the following day malæna continued, and the condition did not improve in spite of the administration of hæmostatics. Pulse was feeble and rapid. Temperature went up to 100.2°F.

Blood examination on the 27th, while the patient had slight fever, showed presence of many benign tertian malaria parasites. Hæmoglobin was 50 per cent and leucocytes 11,250 per c.mm.

*Treatment.*—25th April. Injections of glucose, normal saline, horse serum and calcium gluconate. Foot end of the bed raised. Ice bag to the pit of the stomach.

26th April. Injections of calcium gluconate, congo-red solution and camphor in ether. Foot end of the bed raised.

27th April. Injection of quinine bihydrochloride gr. x intramuscularly.

28th April. Repeated the quinine injection. General condition of the patient improved and there was no more hæmorrhage.

29th April. Alkaline and quinine mixture.

Later, the patient developed parotitis which subsided with local application of belladonna.

*Point of interest.*—The history, signs and symptoms of the patient on admission were suggestive of hæmorrhage from gastro-duodenal ulcer but a diagnosis was not made till the blood was examined.

[*Note.*—It would be of interest to investigate the case further to exclude the presence of peptic ulcer as well.—Editor, I. M. G.]