

casks of great weight, and at the same time, after the manner of his kind, to consume large quantities of beer—for the last three years he had taken from five to eight quarts daily. In October, 1892, previous to which time he had enjoyed excellent health, he was seized with dyspnoea when at work, succeeded by palpitation and great weakness. He stopped off work for a week, and, on resuming, the symptoms had disappeared completely. About December 15th, again while at work, a similar sudden attack of dyspnoea came on, and again he was obliged to discontinue. For two days he had almost continuous dyspnoea, and then palpitation and prostration supervened, with some oedema of the lower extremities, and he was forced to remain in bed. There he remained until January 26th, when feeling somewhat stronger he got up for the first time and walked to the General Hospital, where he was admitted under Dr. Stewart. There was a good family history, and the personal history revealed no previous rheumatism, syphilis, chorea or other disease predisposing to cardiac lesions. There was no complaint of palpitation while the patient was in the hospital; the pulse varied from 68 to 100, and was irregular in volume and in rhythm; the arteries were thickened and not easily compressed. The apex beat was in the sixth interspace $5\frac{3}{4}$ inches from midsternum, and the vertical dullness began at the lower border of the third rib, the transverse began at the right edge of the sternum and extended $6\frac{3}{4}$ inches to the left. There were no murmurs to be heard. The urine contained no albumen on admission, but before death was loaded with it.

A diagnosis of myocarditis was made, and the patient appeared to be improving slowly for a time, but for two weeks before death the dyspnoea became more severe and more frequent, finally becoming Cheyne-Stokes in character, and upon the morning of February 24th the patient died suddenly.

At the autopsy performed upon the following day there was found some anasarca of the lower extremities and slight oedema below the eyes. The lungs were greatly congested and oedematous, so that they only just floated; there were, as so frequently is the case in Montreal, evidences of old pleurisy. The liver was enlarged, fatty and congested; the mucous membrane of the stomach was also moderately congested; there were evidences of old peritonitis in the shape of adhesions. The kidneys were congested, the cortex enlarged and fatty, the capsules peeled off with some difficulty, and the surface of the organs showed well-marked granular change; in both kidneys were several white infarcts surrounded by inflammatory zones.

The heart, however, showed the greatest departure from the normal. Upon opening the thorax it could be seen to be of great size. The apex lay $\frac{3}{4}$ inch outside the left nipple line and

2 inches below; the left lung was pushed upwards and outwards. The pericardial cavity contained more than 250 c.c. of fluid, having a very faintly reddish tinge, but there was no recognizable sign of recent pericarditis, though there was a slight old and loose adhesion close to the apex of the left ventricle. All the cavities were greatly dilated, those of the right side contained fairly solid clot, those of the left a softer, more tarry, coagulum. The heart weighed 690 grm., or just about three times the normal. It was a true "cor bovinum." There was no acute and but little evidence of chronic valvular disease. The pulmonary orifice measured 8.4 centimetres in circumference, the aortic 7.5, and just above the orifice there was a little early fatty degeneration of the intima of the aorta; the segments of both pulmonary and aortic valves were normal. The tricuspid orifice admitted the tips of four fingers, the mitral those of three; and in connection with this last valve the papillary muscles were greatly hypertrophied, the chordæ somewhat short and thick, as were also the edges of the cusps. The endocardium of the ventricles presented no inflammatory change recognizable by the naked eye. The walls of all the cavities were hypertrophied; at the junction of the upper and middle third the myocardium of the left ventricle was 2.3 cm. across. The muscle substance could not be described as other than firm, but here and there it was perhaps a little paler than normal. Upon dissecting up the coronaries no endarteritis was found, but in one branch of the right coronary passing over the hinder wall of the left ventricle, there was at the commencement of the lower third of the organ a red clot about half an inch long, and beneath this the myocardium was red and suffused with blood.

Unfortunately by mischance the heart and kidneys, having been taken from the post-mortem room to demonstrate to Dr. Stewart's clinic, did not reach the laboratory until twenty-four hours later, and then were in a condition far from satisfactory for study of finer details, so that I am unable to make any further statement than that the myocardium around the seat of the lesion of this coronary vessel was necrosed, and here and there were evidences of fatty degeneration in the heart muscle and that the fibres of the left ventricle were in general thin and smaller than normal. Some of the finer branches of the right coronary coursing on the surface of the left ventricle showed evidences of both acute and chronic endarteritis. There was no marked interstitial fibrosis, nor could any small celled infiltration be recognized.

This case, while presenting features which if relatively uncommon are capable of explanation, is beset with several difficulties. It is easy to find in the hæmorrhagic infarct of the left ventricle the cause of the sudden death. But what brought about the condition of this