expressed himself as feeling stronger and his temperature had been subnormal for several days, but on the evening of June 14th, while the dressings were being changed, he suddenly collapsed, became cyanosed, struggled violently, and finally sank into complete unconsciousness. On the following morning right hemiplegia was demonstrated, and cerebral abscess or embolus was suggested as the cause. His temperature assumed a spiky character, his pulse became weaker, and eventually he died on June 20th, exactly four weeks after admission. I felt fairly certain that we should find an ulcerative endocarditis, although a cardiac murmur could never be detected during life. In this I was wrong, as I also was in my surmise as to the cause of the right hemiplegia. As you will see in a moment, there was embolic softening in the brain, but it was in the right side and not in the motor path.

Necropsy.

I now demonstrate to you the organs which have been preserved and at the same time I give you the result of the *post-mortem* examination, which was made on the morning after death by Dr. William Boxwell, our Assistant Pathologist.

The pleural cavity on the right side contained a small quantity of pus, and the surface of the lung, which was to a great extent collapsed, showed a thick coating of fibrin. The base of the left lung also showed signs of a localized acute pleurisy, and at one spot the diaphragmatic pleura was firmly adherent. At this point, on attempt at separation, the lung substance gave way and disclosed a small cavity about the size of a walnut from which pus flowed freely. The abscess cavity was surrounded by a patch of unresolved pneumonia. At the exactly corresponding abdominal surface the spleen was found adherent, and further examination disclosed a collection of blood-stained pus, corresponding to a large septic infarction which had undergone partial disintegration. Beyond a large double infarction in the right kidney, which also showed signs of softening, there was no other noticeable abdominal lesion. The liver was large, congested, but otherwise normal.

Heart and Pericardium.—In the pericardial sac was a small quantity of clear serous transudate. No evidence of acute pericarditis. The heart itself was not markedly enlarged, but globular in shape and very soft. The myocardium was dark and of a "doughy" consistency. There was not the slightest evidence of endocarditis anywhere. The cavities of the auricle and ventricle were partially filled with dark clots.

Brain.—When exposed the pia arachnoid was oedematous all over and the vessels greatly congested. The convolutions seemed widely divaricated, but there was no noticeable suppurative lepto-meningitis. On section, two days after immersion in a solution of salts and formalin, a patch of softening was found just beneath the cortex on the right side, corresponding to the superior temporal convolution; it was behind the "insula." The patch measured about 1 in. each way and was brownish-yellow in colour. The thrombosed vessel leading into it was easily demonstrated. On the left side nothing abnormal could be seen, and serial sections downwards through the corona, capsule, crus, and pons failed to show any clear reason for the right-sided paralysis. The left middle cerebral admitted a flexible probe without resistance, and no evidence of *ante-mortem* clotting on this side could be found on dissection.

This case is of great interest, because of the far-reaching effects produced by the pneumococcus. I regret that I am unable to enter at any length into the ravages which the pneumococcus is capable of producing throughout the body. Apart from any involvement of lung tissue, this organism may produce a general pyaemia with pus formation in the skin, pleurae, kidneys, spleen, joints, etc. It may cause meningitis, endocarditis, pleuritis, and empyema, and the infection is an extremely fatal one.

A CASE OF BRADYCARDIA WITH EPILEPTOID ATTACKS.

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FOLLOWING your account of the fatal illness of Sir William Gairdner (BRITISH MEDICAL JOURNAL, July 6th, p. 53) and Dr. Fred. Hugh Clarke's address (p. 308), a brief note of the following typical case of bradycardia with epileptoid seizures may prove interesting:

History.

Mrs. W., aged 74 years, has a good family history, free from epilepsy and heart disease. Of her eleven children, eight are alive and healthy, two died in infancy from intestinal troubles, and one was killed in an accident. Ten years ago, after a medical examination, she was accepted as a first-class life by an insurance company. Except for frequent headaches, which ceased at the age of 40, she has enjoyed good health until

between two and three years ago, when she noticed that she was getting thinner and began to suffer from increasing attacks of breathlessness with a "sinking sensation like hunger" in the epigastrium. As these got worse they were accompanied by "faint feelings all over." At Christmas, 1905, she was medically attended, and for the first time learned that her heart was affected. In the following March (1906) she had an attack of bronchitis, and since that time she has been subject to "fainting fits."

Six months ago she came under my care for a slight attack of bronchitis. I found she had bradycardia, and advised her to remain under observation and treatment.

Epileptiform Seizures.

About a year ago, after a restless night, during which she several times felt faint, she was surprised to find that she had unconsciously passed urine into her bed. This possibly occurred during an epileptiform attack.

On June 3rd last, while sitting sewing alone in her house, she suddenly felt more than usually faint and lost consciousness. On regaining consciousness she was on the floor, but managed to pull herself into a chair. Here she was found by her daughter with her face bruised and bleeding. Half an hour later, while her bowels were being moved, she became unconscious a second time, and had what her daughter thought was an epileptic fit. She was put to bed. When I saw her, about an hour after the first attack, she was conscious; the face was slightly blue, with abrasions on right side of forehead and cheek; the pulse was 28 per minute, and very weak. While feebly trying to tell me what had occurred she suddenly stopped, made a few restless movements, during which her face became deeply cyanosed, and then, after giving a slight moan, the eyes turned upwards, and the whole body became rigid. In about twenty seconds slight, slow twitchings commenced in the face, and were quickly followed by convulsive movements of the limbs. As these clonic spasms passed off she regained consciousness, and complained of faintness and pain in the epigastrium. This was her third fit within an hour and a quarter, and all were probably similar, though the first was not observed. Her daughter saw the second fit and said it was more severe than the one I witnessed. No biting of the tongue occurred, but she passed urine involuntarily during one or more of the attacks. She was kept in bed and given bromides, with ammonia and strophanthus. She has had no more epileptiform seizures, though she still has frequent attacks of faintness.

Present Condition.

The patient is a well-built woman, and not obviously wasted. Her face is covered with venous stigmata. There are many small, purple, naevoid spots on the face, and several larger and brighter on the body, all having developed during the past two years. She has repeated small subconjunctival ecchymoses. The cardiac impulse cannot be seen or palpated; by auscultation it is in the fifth left intercostal space, $4\frac{1}{2}$ in. to the left of the middle line. Deep cardiac dullness in fourth spaces extends from $\frac{1}{2}$ in. to the right of the right costal margin to $4\frac{1}{2}$ in. to the left of the middle line. The upper limit of superficial cardiac dullness is at the junction of the third left costal cartilage with the sternum. The cardiac sounds are feeble, being loudest at the apex and nearly inaudible in the pulmonary area. The first sound is short, abrupt, and quickly followed by a sharp and slightly louder second sound, which is sometimes reduplicated. Pulse: Rate varies from 26 to 33, and is generally 30 per minute; it is feeble, of small volume, and low tension; vessels are not thickened. All heart-beats are transmitted to radial pulse. A few coarse crepitations are heard all over the lungs. The abdomen is soft and slightly tender, especially over the liver, the lower border of which is 1 in. below the costal margin in the right nipple line; the upper border of absolute liver dullness is at the sixth rib in the same vertical line. Physical examination is difficult, as any but the briefest examination causes her to become blue and faint. The urine is of low specific gravity, and contains a little albumen and a few granular casts.

The bradycardia in this case is probably due to cardiac degeneration. The veins of the neck do not show auricular pulsations of greater frequency than the heart-beat, such as are described as proofs of heart-block in Stokes-Adams disease. The thinness of the vessels, in spite of the kidney condition, is probably due to the absence of high tension. The fact that cyanosis preceded the tonic epileptiform stage supports the view that such epileptiform seizures are due to disturbed cerebral circulation.

THE Professors of the medical faculty of the University of Rome have at last succeeded in inducing the Italian Parliament to give heed to their representations as to the inadequacy of salaries. The average amount of the salaries attached to the medical chairs is only £280, while the deputy professors and assistants are of course paid on a still lower scale. A Bill has been introduced into the Chamber of Deputies providing for such an increase as will enable the professors to devote themselves wholly to scientific work;