A CASE OF ANGINAL SPASM OF EXCEPTIONAL DURATION.

BY

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WE are so accustomed to look upon angina pectoris as a disease in which the patient either dies or recovers from the spasm in a very short time—certainly under an hour—that a case in which the spasm lasted for a whole night is of sufficient interest to place on record.

The patient was a retired gentleman in his 80th year. Some twenty years ago he had a severe attack of diphtheria which left no evil effects in the way of paralysis, etc. From that time he had no illness worth mentioning, and in latter years especially his health was excellent—in fact, after he was 70 he visited friends both in South Africa and in the United States, and only the year before his death he had a motor run of 100 miles, returning by train the same day, without experiencing the slightest undue fatigue.

His first attack of angina was on June 29th, 1911; other attacks followed on July 3rd, August 16th, September 8th, 9th, and 10th. The last attack was the prolonged one lasting from 9.20 p.m. to 6.25 a.m. Several of the earlier ones were fairly severe, but in each case the spasm soon yielded (less than half an hour).

No signs of cardiac disease were to be detected, the sounds were good and clear, the apex not displaced outwards, and the right heart boundaries normal. The arteries were quite soft and the arterial tension slightly below the average. Urine was normal, with no trace of albumen. There was no respiratory trouble of any kind. He had a hearty appetite, but took a fair amount of exercise, being a good walker.

The last attack began at 9.20 p.m., after he had retired for the night. As it did not yield to amyl nitrite inhalations, I was sent for. I found him in great distress; he was gasping for breath and complained of great tightness in the upper part of the chest. The pain was most severe; it was situated near the upper part of the sternum, radiating up both sides of the neck to the ears, hardly any passing down the left arm. He was propped up in bed, his face slightly flushed, but by no means cyanosed; the pulse very slightly affected in either wrist.

When I saw him he had already inhaled the contents of two amyl nitrite capsules (5 minims each) and taken a dose of carminative medicine, which had previously been of service in relieving flatulence. I gave him a hypodermic injection of $\frac{1}{10}$ grain nitroglycerine, and, as this produced no effect, I gave him an injection of scopolamine and morphine ($\frac{1}{32}$ and $\frac{1}{4}$). Again I was disappointed, no improvement following. I then administered chloroform on a Skinner's mask, and he became easier, the spasm passed off completely, and the breathing was deep and peaceful. In about twenty minutes I stopped the chloroform, thinking that the spasm would not now return, but, with recovery of consciousness, it slowly reappeared, and was eventually as severe as before. The only way to give the patient any relief was to resort to chloroform, and this I continued to administer at intervals until 6 a.m., when the spasm relaxed, passing off altogether at 6.25 a.m.; the patient then had two hours' natural sleep. During the night I used nearly a pound bottle of D. and F. chloroform. Except when under the influence of chloroform, there was no relaxation of the spasm from 9.20 p.m. to 6.25 a.m. The sense of impending death was markedly present. The pulse was never markedly affected, at times a little irregular. It gradually increased in rate and became smaller in volume.

The next day he expectorated some plugs of dark-coloured mucus, suggestive of pulmonary embolism; but the most careful examination of the chest revealed no signs of one. A few days later (September 13th) I found a small spot on the right base which was dull on percussion, and where some moist sounds could be heard (the following night he had a slight rigor), but these signs soon cleared up, and when examined in consultation a few days later no traces were found. At the most there were barely six pieces of blood-stained mucus expectorated.

He had no other attacks, and in the next week slowly gained strength, his appetite markedly improved, and he felt altogether better; but the improvement did not continue, he became gradually weaker, and died quite peacefully in his sleep ten days after the prolonged attack. All this time his pulse had been rapid (90 to 100), frequently irregular and intermittent, and always very weak, especially so the last few days. The heart sounds were distinctly heard, but very feeble—especially the first. The temperature kept low, usually 96° to 97°, except on the night of the 13th, when there was a slight rigor, the temperature rising to 101.6°, but falling to normal by morning. The day before his death it went up to 99°, but only for a few hours.

It may be suggested that this was a case of pulmonary embolism, and not angina pectoris. But on reviewing the whole case I do not think this to be the correct diagnosis. Against this view we have the absence of any obvious source of the embolus; the previous undoubted anginal attacks unaccompanied by any pulmonary symptoms, the

absence of cyanosis or of cough after the attack, as would assuredly have followed an infarction sufficiently large to have given rise to such severe symptoms. I take it that the course of events was as follows—an obstruction in some of the smaller branches of the coronary artery, insufficient in size to cause stoppage of the heart, but which obstruction was to some extent permanent, as evidenced by the subsequent cardiac degeneration. The time taken by the heart in accommodating itself to its reduced blood supply, accounted for the length of the attack. Probably owing to the obstructed coronary arterial flow, there was some stasis in the coronary veins where a small embolus formed, which accounted for the pulmonary symptoms. The coronary circulation probably never became completely re-established, but gradually more interrupted, and so caused the cardiac degeneration which finally brought about death.

RELIABILITY OF THE RESULTS OBTAINED BY THE WASSERMANN TEST ON SERUMS AND CEREBRO-SPINAL FLUIDS OBTAINED POST MORTEM.

BY

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THE object of this paper is to record our experiences of the Wassermann reaction as applied to blood serums and cerebro-spinal fluids obtained *post mortem* from cases of insanity at the London County Asylum of Claybury.

We have been engaged upon this work during the past year, and, as our results do not accord with some recent publications on the same subject, Dr. Mott has suggested that we should record the results of our own observations.

In his Hunterian Lecture¹ Major French quoted the findings of Ponfik and Winckler to the effect that, of 101 specimens of serums from cadavera, 59 (that is, 60 per cent.) gave a positive reaction. Further, Luksch² states that of 309 *post-mortem* cases a positive result was obtained with the serums in 46 per cent., and he did not from this fact infer that all these 46 per cent. were cases of syphilis. Luksch believes that the occurrence of the Wassermann reaction shortly before or after death does not prove anything, because, in his opinion, serious diseases produce changes in the blood serum which may cause it to yield the reaction in question. This might take place a long time before the close of life. During the course of sepsis, uraemia, or pneumonia such changes were observed very often, even if the patient recovered completely. He considered that the Wassermann reaction was not pathognomonic of syphilis, because under certain conditions, both during life and after death, it might occur in persons who were free from syphilis. For this reason a pathologist was not entitled to diagnose syphilitic changes if the Wassermann reaction was present in the cadaver, and in cases of serious illness the physician must be careful not to rely too much on the reaction. More satisfactory results have been obtained by Nauwerck and Weichert, Schlimpert, Löhlein, and Veszprémi, but these observers have excluded *post-mortem* serums which were not obtained in a fresh condition.

Routine Method adopted at Claybury.

In all cases of death the bodies are placed in the cold chamber and the autopsy is made generally within twenty-four hours. The fluids are withdrawn at the time of autopsy, and if not tested on that day are placed in the ice chamber until the test is made (generally within a day or so), when the serum which has separated is pipetted off and inactivated in the usual manner.

The technique adopted in this research was that of the original Wassermann method described by one of us (J. P. C.).³

Results of the Investigation.

A total number of 112 cases have been examined, of which the cerebro-spinal fluid and the blood serum have been examined in 92 cases, the cerebro-spinal fluid alone in 20 cases. With regard to the cerebro-spinal fluid, it will thus be seen that 112 samples were examined, of which 25 were found to give a positive result. Each of these 25 cases