A CASE OF ANGINAL SPASM OF EXCEPTIONAL DURATION.

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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

was shown by macroscopic and microscopic examination to be an instance of general paralysis, and in 17 of these cases where the serum also was examined a positive result was obtained. In 8 of these the cerebro-spinal fluid had been examined during the life of the patient with a positive result.

In 3 cases out of the remaining 87 the result of the Wassermann test was not in accordance with the clinical evidence and *post-mortem* appearances.

CASE I.—F. A case of general paralysis. Male, aged 42, dying of general paralysis of the insane and hypostatic congestion of lungs, gave a negative result both with the serum and cerebro-spinal fluid. (This fluid had become turbid from decomposition before the test was made.)

CASE II.—M. A case of general paralysis, gave a marked positive result with the blood serum, but a negative with the cerebro-spinal fluid. This case had given a positive result with the cerebro-spinal fluid during life. Death had been accelerated in this case by an acute meningitis. The fluid was purulent when withdrawn, and was crowded with streptococci and polymorphonuclear leucocytes.

CASE III.—J. A case of insanity (male, aged 68) other than general paralysis. The fluid showed some degree of complement deviation, which, however, was not sufficient to be accepted as a definite positive result. Decomposition had commenced in this fluid at the time of examination. Cause of death, bronchopneumonia.

We will explain later to what we consider these anomalous reactions to be due.

Examination of Blood Serum.

With regard to the 92 cases in which the serum was examined, as already stated, 20 of these were cases of general paralysis, and in only one (Case F.) was a negative result obtained. Of the remaining 72 cases, a positive result was obtained in only 10. Two of these cases died from rupture of a thoracic aneurysm, and there was undoubted evidence of previous syphilitic infection; in both these cases the cerebro-spinal fluid gave a negative result. In 2 of the cases there was found a marked nodular fibrosis of the aorta, which in Dr. Mott's opinion was undoubtedly syphilitic in character. In the remaining 6 cases there was no definite history or signs of previous syphilitic infection. They were as follows:

1. Female, aged 28. Acute melancholia. Cause of death: Pyaemia secondary to stomatitis and gangrenous tonsillitis. Serum: Positive. Cerebro-spinal fluid: Negative.
2. Female, aged 39. Melancholia. Cause of death: Acute ulcerative dysentery. Serum: Positive. Cerebro-spinal fluid: Negative.
3. Female, aged 41. Dementia. Cause of death: Carcinoma of cervix uteri with vesico-vaginal fistula; purulent cystitis and septicaemia. Serum: Positive. Cerebro-spinal fluid: Negative.
4. Female, aged 47. Mania. Cause of death: Tumour cerebri and pulmonary tuberculosis. Serum: Positive. Cerebro-spinal fluid: Negative.
5. Male, aged 57. Senile dementia with epilepsy. Cause of death: Bronchopneumonia. Serum: Positive. Cerebro-spinal fluid: Negative.
6. Female, aged 37. Melancholia. Cause of death: Pulmonary and intestinal tuberculosis. Serum: Positive. Cerebro-spinal fluid: Negative. The brain of this patient showed some evidence of congenital imbecility; and in the light of H. P. Dean's observations⁴ our result on the serum may be correct.

In the remaining 62 cases a negative reaction was obtained on the blood serum. We have investigated the histories of these cases as fully as possible, but without finding any notes which would lead us to think that these results are incorrect, except in one case of marked tabes dorsalis, in which the cerebro-spinal fluid two years before death and also *post mortem* gave a negative reaction and the blood serum *post mortem* was likewise negative. In this case there was a history of syphilis contracted twenty years previously.

SUMMARY OF RESULTS.

Cerebro-spinal Fluid (Number of Cases examined, 112).

Number of cases of general paralysis giving a positive result ...	25
Number of cases of general paralysis giving a negative result...	2*
Number of cases other than of general paralysis giving a negative result ...	85
Number of cases other than of general paralysis giving a positive result ...	—

* Cases F. and M.

Therefore out of 112 cases in which the cerebro-spinal fluid was examined *post mortem* a correct result was obtained in 110 instances (98 per cent.). Of the 85 cases other than general paralysis only one case gave the slightest evidence of retardation of haemolysis, not sufficient, however, to justify a positive reaction.

Serums (Number of Cases examined, 92).

Number of cases of general paralysis giving a positive result ...	19
Number of cases of general paralysis giving a negative result...	1
Number of cases other than general paralysis examined ...	72
Number of cases other than general paralysis examined giving positive result ...	10 (14%)
Number of cases other than general paralysis examined giving positive result (but excluding 4 cases of undoubted syphilis) ...	6 (8.3%)

If our figures are contrasted with the 60 per cent. and 46 per cent. of positive results obtained by other observers on serums from cadavera, it will be seen that, after deducting the cases of general paralysis from our figures, we only obtained a percentage incidence of 14 per cent., and after deducting from these the undoubted syphilitic cases, we only obtained an apparently anomalous reaction in 8.3 per cent. We are therefore inclined to the opinion that the test, as applied to *post-mortem* material, is a very helpful one—at any rate, as far as the diagnosis of general paralysis is concerned, as well as for evidence of previous syphilitic infection. It is, however, not so reliable as the fluid withdrawn during life. There are limitations to its use, and to these we desire to draw attention.

Luk's objection as to its reliability is based on the fact that such a large percentage of cases after death react positively to the Wassermann test, although apparently not syphilitic. We have not met with that difficulty in our asylum cases to anything like the same extent, though we have obtained a positive result in 8.3 per cent. of cases where a negative result was expected; on the other hand, we have obtained a negative where a positive result was expected. Our attention was first drawn to one of the factors responsible for the unreliability of the reaction in *post mortem* cases by noting that a serum which had at first given a completely negative result and which was being kept as a control became, after a short time, positive to the test. With this alteration in the reaction there had occurred some turbidity of the fluid and growth of micro-organisms. Similar results were found to occur in the case of a cerebro-spinal fluid.

This phenomenon was investigated more carefully by allowing serum and cerebro-spinal fluids, whose reactions to the Wassermann test had been recorded, to decompose at room temperature. We then found that anomalous reactions were frequently obtained; a positive reaction would become a negative and a negative tend to give a positive result. In the case of a perfectly clean and sterile inactivated serum or a cerebro-spinal fluid to which 0.5 per cent. phenol had been added, the reaction would remain unaltered even after the lapse of a considerable period. In one case of ruptured thoracic aneurysm the blood serum removed *post mortem* and inactivated was used periodically as a control for over three months, and at the end of that time showed the same power of complement deviation as when first obtained, and cerebro-spinal fluids from cases of general paralysis and other forms of insanity which were removed *post mortem* and carbolized five months ago are still in use as positive and negative controls.

In order to show that bacteria and their products can materially alter the reaction, we have lately taken known positive and negative cerebro-spinal fluids and inoculated them with laboratory strains of *Staphylococcus aureus*, *Bacillus coli communis*, and *Bacillus pyocyaneus*. At the end of twenty-four hours' incubation the test was again applied, and the strain of *Bacillus pyocyaneus* used was found to have caused a positive fluid to become negative, and a negative to have become positive.

These investigations are as yet quite incomplete, but taken in conjunction with the changes in reaction which have been observed in fluids which have been allowed to decompose, they suggest that certain varieties of saprophytic and pathogenic bacteria or their products may have the power of altering the reaction.

It is to the action of decomposition products that we attribute the anomalous reactions obtained with the cerebro-spinal fluids of Case F. and Case J. already referred to, for the cerebro spinal fluids of both these cases were turbid and full of bacteria when they were examined.

In Case M., however, we are inclined to attribute the failure to obtain a positive reaction to some changes set up

by the streptococcal infection of the meninges which had obviously occurred before death. The cerebro-spinal fluid of this patient had given a positive reaction before death. A known positive cerebro-spinal fluid was inoculated from a culture of this streptococcus, with the result that a considerable degree of haemolysis was obtained.

With regard to the serums giving positive reactions in cases that were not apparently specific, we would explain the anomalous reactions as the result of decomposition of the serum, for we have found that as soon as the serum commences to show any turbidity the reactions will tend to become positive. In some of these cases decomposition had obviously commenced even when the blood was removed from the body.

CONCLUSIONS.

While, therefore, our observations lead us to consider that the reactions on the blood and cerebro-spinal fluid removed from the cadaver before decomposition has commenced will give reliable results, we agree with Luksch that decomposition is liable to alter the reaction, as may also a terminal microbial infection. This change, however, is not confined to the alteration of a negative to a positive result, but may alter a positive into a negative; and we would point out that decomposition may similarly influence the result of the test on fluids and serums removed during life.

To ensure satisfactory results on *post-mortem* material it is, therefore, necessary to remove the cerebro-spinal fluid and blood as soon as possible after death. The serum should be separated and inactivated at once, and the cerebro-spinal fluid should be carbolized, unless the test can be applied the same day. The same remarks apply with regard to the preservation of material removed during life.

REFERENCES.

¹ *Verhandl. der deutsch. patholog. Gesellschaft.*, 1910, xiv, p. 249. ² *Lancet*, 1910, vol. ii, p. 204. ³ *Ibid.*, November 11th, 1911. ⁴ *Proc. Roy. Soc. Med.*, July, 1910. For complete bibliography see *Syphilis from the Modern Standpoint*, McIntosh and Fildes (Arnold, 1911).

AN ACCURATE METHOD OF ESTIMATING THE VIBRATORY SENSE.

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EGGER in 1899 called attention to the sense of vibration felt when the foot of a vibrating tuning-fork is placed over subcutaneous bony points. The investigations of Bing showed that the sensation is probably conducted upwards in the posterior column of the white matter by fibres which do not decussate. In this country Williamson has shown that the vibratory sense is often lost when other forms of sensation are present. In peripheral neuritis, due to diphtheria, influenza, diabetes and alcohol, it may be lost at an early stage, and it may be an early sign in tabes.

The ordinary method of investigating the vibratory sense does not show the abnormal states which precede its complete or almost complete disappearance. In my investigations I have used the tuning-fork introduced and made by Edelmann of Munich for use in Schwabach's aural test for the absolute bone conduction of hearing. Its vibration frequency is 108.75 a second. Two pieces of steel are attached to the upper portion of the fork, arranged in such a manner that when the arms of the fork are vibrating a small window is seen between them; this disappears when the vibrations of the fork reach a definite amplitude. The moment that the window disappears I apply the fork to the subcutaneous bony point and start a stop-watch. The patient is instructed to give a signal at the moment when he ceases to appreciate the vibration; the watch is then stopped and the time recorded.

By this method on thirty normal individuals I found what period elapses before the sensation disappears at each bony point. The average periods, together with the maximum and minimum, are shown in the accompanying table. In the list there given it is noticeable that the average times over the sacrum and anterior superior spines are considerably less than the time for the other bony points. This is of interest, as Mott and others have found that the vibratory sense may be absent over the sacrum in cases of tabes

	Average.	Maximum.	Minimum.
	Seconds.	Seconds.	Seconds.
Internal malleolus ...	27	35	25
External malleolus ...	28	38	25
Tibia (middle of shaft, inner aspect)	29	37	25
Anterior superior spine ...	21	25	16
Sacrum ...	20	23	15
Sternum ...	30	36	24
Radius (lower end) ...	33	40	28
Ulna (lower end) ...	34	42	28

when still present at the other bony points. This would follow a general depression of the sensation, since it is appreciated over these points less readily than in the other situations.

When this test is applied to patients suffering from impairment of the vibratory sensation a numerical ratio to the average normal is obtained which accurately represents the degree of impairment. I have used a graphic method to demonstrate these changes; the height in the accompanying curves represents the time during which the sensation was appreciated. Superimposed in most cases is a normal average curve for comparison.

CASE I.—Diabetic Neuritis.

Male, aged 54, suffering from diabetes. Pain and tenderness along the right sciatic nerve was the only evidence of neuritis, both tendo Achillis jerks being present. In spite of this the

Fig. 1.—Case I. A, Internal malleolus; B, external malleolus; C, tibia; D, anterior superior spine; E, sacrum; F, sternum; G, radius; H, ulna. — — — Average normal; — — — right side; — — — left side.

vibratory sense was one-half the normal over the tibia and external and internal malleoli of both sides, the right being only slightly more affected than the left.

CASE II.—Plumbic Neuritis.

Male, aged 32, suffering from lead poisoning. The right musculo-spiral nerve was markedly affected, the left very slightly.

Fig. 2.—Case II. A, Internal malleolus; B, external malleolus; C, tibia; D, anterior superior spine; E, sacrum; F, sternum; G, radius; H, ulna. — — — Average normal; — — — right side; — — — left side.