shortly afterwards. Since then I have been able to investigate the fluid in about a dozen other cases either shortly before or shortly after death had occurred. In every case the approach or occurrence of death led to a large increase in the nitrogenous contents of the cerebro-spinal fluid. For example, Case No. 20 had been suffering from general paralysis for fourteen months and his nitrogen number had gradually increased from 25 to 39. He died eleven days after this, and his fluid, withdrawn by lumbar puncture thirty minutes after death, gave NN 109. In another case (No. 3), diagnosed as confusional insanity, the nitrogen number had been found to be 24 and 27 on two occasions during life. One hour after death it was 38, and twenty hours after death it was 88.

Results similar to these two instances quoted were obtained in every case examined, and they leave no room for doubt that a very remarkable change in the composition of the cerebro-spinal fluid begins to take place shortly before death and continues after it. Whether this is directly due to excessive katabolism in the brain tissue, or whether it is due to a partial breakdown of the cells secreting the cerebro-spinal fluid, is a matter of speculation, but it is a matter of fact that it does occur and that in consequence any analyses of fluids obtained *post mortem* are valueless, for they bear no relation to the composition of the fluid during life, which is the only thing that matters. It is unfortunate that this objection renders valueless a great deal of the work on cerebro-spinal fluid recorded in the literature.

The Nitrogen Number of Cases other than General Paralysis.

It is well known that although every effort has been made to discover a physical basis for mania, melancholia, and other commonly occurring forms of mental disorder, no such basis has ever been found. These forms of mental disorder have been investigated histologically, bacteriologically, chemically, and in many other directions, but in all cases with negative results. It remains to be seen whether the study of the cerebro-spinal fluid during life may solve the problem, but the results exhibited in Diagram 2 show that the nitrogen method by itself brings no help in the matter.

The scheme of graphic representation employed is the same as that in Diagram 1, a circle indicating a case of the disease found to have the nitrogen number opposite which it is placed. It will be seen that in all the ten types of mental disorder included in the table the circles are pretty uniformly distributed between about 15 and 22, and that this is also the range of the cases not insane in column 11. There is therefore no differentiation between the different clinical types recorded or even between them and the cases not insane.

Variations in Progressive Disease.

In a progressive disease such as general paralysis it might be expected that the nitrogen number would mirror to some extent the course of the disease: that when the patient was going downhill the nitrogen number would increase, and that a stationary state or a definite remission would be accompanied by a stationary or a reduced nitrogen number. I have records of fourteen cases of general paralysis in which samples of the fluid had been obtained from time to time, and in some of these the expected parallelism is to be noticed. As examples I may cite the following cases:

Case 20.—Aged 41. Admitted January 2nd, 1911. Had seizure in December, 1911. Died February 27th, 1914.

December 10th, 1912: Nitrogen number 25. Patient in stationary state, considerably demented.

January 22nd, 1913: NN 27. Condition unchanged.

April 9th, 1913: Condition unchanged; in partial remission, no seizures. NN 27.

On June 16th, 1913, the nitrogen number was 29, and on September 29th it was 25.

December 15th, 1913: Disease progressing rapidly; several seizures. Nitrogen number 30.

February 16th, 1914: Nitrogen number 39.

February 27th, 1914: Fluid taken thirty minutes after death. Nitrogen number 109.

Case 222.—Male, aged 45. Admitted about February, 1921. On March 9th, 1921, the nitrogen number was 40; disease in active phase. On April 16th and April 30th, 1921, the disease was still in active phase (worse). The nitrogen number on both dates was 36.

From mid June until October, 1921, he had a quiet interval. October 6th: Growing more excited daily. Nitrogen number 30. October 21st: Still excited. Nitrogen number 29.

In remission from about November 19th, 1921. The patient had received an intensive course of treatment with salvarsanized serum, and, his remission continuing, was released on trial in January, 1922.

In other cases, however, the figures for the nitrogen number do not fit the condition of the patient so well.

Insanity with Gross Brain Lesion.

It has already been pointed out that the nitrogen number reveals abnormal breakdown of tissue without indicating the cause of that breakdown. The following cases illustrate this:

Case 101.—Syphilitic meningitis. On November 10th, 1913, the nitrogen number was 57; on March 2nd, 1914, it was 31; and on March 30th, 1914, it was 27. The patient was discharged relieved on July 30th, 1914.

Case 137.—Semi-recent cerebral haemorrhage, verified *post mortem*. On June 8th, 1914, the nitrogen number was 32. Patient died eight days later (June 16th).

Case 204.—The patient died from brain tumour on September 26th, 1919. The nitrogen number on September 24th was 273.

Case 250.—The patient died from brain tumour on the day of puncture (December 1st, 1921). Nitrogen number 44.

Senile Dementia and Senility.

Reference to Diagram 2, column 9, will show that in cases of secondary or terminal dementia fairly high nitrogen numbers, ranging up to 30, are frequently found. As such cases would not in any event be confused with anything else, the matter is of no importance from the point of view of diagnosis; but a very interesting observation was made recently which gives the subject a certain importance from another aspect. Case No. 257, not insane, was operated on for senile gangrene, he being aged 78. His nitrogen number was, however, only 21. I have records in two other cases, aged 68 and 72 respectively, of patients not insane and having practically normal nitrogen numbers (21 in each case).

It would seem, therefore, that there is a difference between secondary dementia and mere senility, and this leads to the conclusion that the acute mental condition which preceded the dementia must have had some physical basis in spite of the fact that neither by the method described in this paper nor by any other has that physical basis yet been discovered.

I am under obligation to my colleagues, the medical superintendent and the present and former assistant medical officers of the hospital, for the clinical information and for the collection of specimens. I have at all times had the advantage of their willing co-operation, without which, indeed, the work could not have been carried out.

REFERENCES.

- ¹ *Lancet*, December, 1914.
- ² Compare Goodall, *BRITISH MEDICAL JOURNAL*, August 16th, 1919, p. 199.
- ³ Kauffmann, *Ber.* 1910, 43, 2735.
- ⁴ *Zeit. f. Physiolog. Chemie*, vol. 86, 1913, 219-233.
- ⁵ Reports from the Chemical Laboratory, Cardiff City Mental Hospital, No. 2.

POISONING BY BUTTER OF ANTIMONY.

BY

WILLIAM BELL, M.D., M.R.C.P. EDIN.,

BRADFORD.

COMMERCIAL antimony chloride, commonly called butter of antimony, contains an excess of acid, which gives it a power of corrosion additional to that effected by the antimony ions, which are readily separated. The effect on the bare fingers of handling the stomach in the case here recorded was similar to that produced by a moderately strong solution of hydrochloric acid—tingling and a sensation of intolerable heat.

On September 16th, 1918, I saw at his house a farmer, who was said to have "fallen into a dyke." I found him, a man aged 46, in bed upstairs at 9 a.m. He had been found lying in a flooded stream about a hundred yards away, conscious, with his head above water. Urged and assisted by his wife, he had walked home and been put to bed. He was shivering; his skin was very cold and livid all over; his pulse was rapid and full. He lay with eyes closed, but he opened them and put out his tongue when ordered. The former were congested, and the latter coated heavily with a harsh white fur. There were no marks about the lips. He was groaning and attempting to vomit. Hot bottles were obtained, but his great restlessness made it difficult to cover him warmly. His appearance at first was simply that due to exposure to cold, but the retching suggested something more, and this impression was deepened by his inability to swallow some warm milk, which he coughed back, clutching at his throat as if the attempt hurt him greatly. Violent retching ensued. Further inquiries elicited the following:

He was seen by his wife about 6.20 a.m., but not again till 8 a.m., when she found him at the stream. About his face was dark brown and slimy vomit, which his wife wiped away with her apron. He walked home and vomited some yellow-stained mucus on to a towel, which, with the apron, was shown to me. He had had no food before leaving the house. Near to a railing, which he had crossed a few yards from the stream, was found a bottle labelled "Butter of Antimony." The bottle and its contents were identified by a chemist, who said the patient had been in the habit

of purchasing two ounces of butter of antimony at a time for treating a horse's foot. The bottle contained 10 fluid drachms.

By 11 a.m. the patient was much worse: the pulse much weaker, the skin still cold and livid, the stomach the seat of burning pain. He died at 3 p.m., seven hours after he had been found, and probably a little over eight hours after he had swallowed the poison.

An autopsy was performed at 2 p.m. the next day. Rigor mortis was extreme. The body generally and the various organs appeared quite healthy except for changes produced by an irritant or corrosive poison acting rapidly. Therefore reference will here be made only to the parts on which the stress of the injury fell. On the abdomen being opened the stomach bulged forward, a striking object deep red in colour. A few coils of small intestine were injected a bright red on their anterior surfaces. There was a small bright red stain on the great omentum adjoining the greater curvature and another stain at the duodenal junction, which looked bruised. The whole outer surface of the stomach was plum-red. Its contents were 20 ounces of dark, almost black, fluid, like charcoal powder and water thoroughly mixed. After being washed almost the whole inner surface was black. It could be scraped away, as could moist dense cardboard, with considerable force. It was not rough and there was no perforation. The mucous coat had been transformed by charring into friable material and its surface shed.

As to the quantity of the poison taken, from the statement of the chemist it would appear that it could not have been more than 6 drachms. Recoveries from 4 or 5 drachms (by a boy aged 12 years) and from 1 fluid ounce have been recorded. In the present case, being empty at the time of administration, the stomach was vulnerable. The subsequent ill effects were intensified by the man's action in lying in a cold stream, probably for a considerable time. One may imagine that, planned or impulsive, his procedure was to rush to the stream to slake his burning in the cool water. By so doing he would provide a rapid diffusion of a strong irritant over the interior surface of the stomach and also liberate free acid.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

DIFFERENTIAL DIAGNOSIS BETWEEN CEREBRAL HAEMORRHAGE AND DIABETIC COMA.

The following case is worth recording as it illustrates how easily an incorrect diagnosis may be made between cerebral haemorrhage and coma from diabetes. The case is "complete," for it was followed from the onset to a complete *post mortem* examination.

A woman, aged 72, was admitted to hospital at 6 p.m. on November 29th, 1921, in deep coma. Her son stated that she had always had good health, and in fact had never had a doctor as far as he knew. He returned home at 5 p.m. on November 29th and found his mother lying on the kitchen floor: there was a strong smell of gas in the room, and the tea things were laid, the lid of the kettle being on the floor. A doctor was called in and he sent the patient to hospital at once.

On admission the patient had a temperature of 97°, pulse rate 88, respiration rate 28. She was in a deep coma; the breathing was stertorous, the cheeks puffing in and out. Both pupils were pin-point and equal; there was very slight reaction to bright light. A small cataract was present in the left eye. The breath had a faintly sweetish smell. The heart and lungs were normal; the blood pressure was 155 (systolic) and 130 (diastolic). The arms and legs were very rigid; all reflexes more brisk than normal, particularly the knee-jerks; there was incontinence of urine. The differential diagnosis at this stage appeared to be between carbon monoxide poisoning, cerebral haemorrhage, and diabetic or uraemic coma. A catheter was passed and ten ounces of urine withdrawn and examined. The urine was acid, specific gravity 1005; sugar ++ (Fehling and Benedict, immediate reduction); acetone and diacetic acid ++ (iron perchloride and Rothera test). A diagnosis of diabetic coma was made, and the patient was treated for twenty-four hours with soda bicarbonate and subcutaneous salines.

On November 30th her condition seemed the same; the urine contained no sugar, but acetone was present in a considerable quantity. On December 1st there was a marked change: the rigidity was replaced by flaccidity in the arms and legs; the reflexes of right arm (biceps, triceps) were very brisk; all other reflexes were absent; the urine still contained acetone, but no sugar. The diagnosis was therefore changed to cerebral haemorrhage. On December 2nd the patient died; the pupils were equal and pin-point; the temperature rose to 102°.

On *post-mortem* examination of the brain blood-stained fluid oozed out of the stalk of the infundibulum. There was an enormous haemorrhage into the basal ganglia and internal capsule of the left side. There was so much destruction of brain tissue that it was impossible to state the exact site of the original haemorrhage; a large clot was present in the distended left lateral ventricle. The third ventricle and right lateral ventricle both contained blood-stained fluid, but no clot. They were not distended. The fourth ventricle contained no blood and was not distended.

The thoracic and abdominal aorta showed considerable arterio-sclerotic changes, with many calcareous plaques of varying size. The kidneys were arterio-sclerotic; the right weighed 3½ oz. and the left 2½ oz.

There are records of acetone being found in cases of cerebral haemorrhage, but I can find no cases in which sugar in considerable quantity was present. Puncture of or damage to the floor of the fourth ventricle should give rise to glycosuria, but there was no evidence of this at the autopsy.

E. D. SPACKMAN, M.A., M.B., B.Ch. Cantab.

Wolverhampton.

THE ANTE-NATAL TREATMENT OF CONGENITAL SYPHILIS WITH SALVARSAN AND MERCURY.

The following results were obtained at the Royal Infirmary, Sunderland, in the years 1920 and 1921:

Year.	Mothers treated for Syphilis.	Babies born Alive.	Result of Repeated Wassermann Tests.		Babies Dying of Syphilis.	Deaths of Foetus.
			Positive.	Negative.		
1920	10	10	3	7	0	0
1921	7	7	1	6	0	0

These cases were all treated as out-patients. Most of them were confined in their own homes, and brought their babies for examination about a month after birth. A Wassermann test was done on the babies about the first, third, and sixth months, but occasionally the periods varied, one case, for instance, having been tested for the first time one year after birth.

Dr. Findlay's results¹ encourage one to hope that not only will the "negative" baby remain negative, but that the mothers will continue to bear non-syphilitic children.

NOEL F. ROWSTRON, M.D.,

Honorary Physician, Royal Infirmary, Sunderland.

THE TREATMENT OF EARLY CASES OF CHOLERA WITH VOLATILE OILS.

I wish to record a form of treatment which in the course of many years I have found not merely beneficial, but certainly curative in the early stages of cholera. Many years ago, placed in the midst of a severe and widespread epidemic of cholera, I met a gentleman who told me of what he called a "certain cure" for cholera, consisting of grated nutmeg in rum. I had opportunities of watching the effect of this in a good many cases; the early ones were cured immediately, the disease being apparently aborted. On reflection I was convinced that the beneficial effect of the nutmeg was due to its volatile oil, which probably had a bactericidal effect on the cholera vibrios in the duodenum and small intestine. I then found in the camp hospitals a "pro diarrhoea mixture" supplied by the medical stores which contained approximately 28 per cent. of the volatile oils of cajuput, aniseed, and juniper in equal parts, tincture of cinnamon 50 per cent., and ether alcohol and aromatic sulphuric acid about 22 per cent. I tried this for cases of cholera in 40-minim doses, containing about 12 or 15 minims of the mixed volatile oils, at half-hour intervals, and the reports I got on the effect of this mixture were most favourable. Since then I have had opportunities of watching the effects of the treatment of cases of cholera with volatile oils in three different years, and in early but typical cases of cholera, whether oil of cloves, or cinnamon oil, or the mixture of four oils previously mentioned was given, the disease was invariably aborted.

In two cases that I can call to mind cholera vibrios were isolated in the central laboratory at Baghdad. These cases were given one or two doses (12 minims) of oil of cloves before being transferred to the isolation hospital; they were walking about the following day, and saline infusions were not found necessary on their arrival.

Very recently nine cases having the clinical appearance of cholera have been brought to the military cholera hospital here during the existence of a mild epidemic in the vicinity. In some of these a bacillus resembling the cholera bacillus was found, but not giving the correct cultural reactions. The first case, treated on ordinary lines with saline infusion, etc., died. One other died ten minutes after admission. The other seven, treated with oil of cinnamon in 12-minim doses, supplemented, in several cases that were collapsed, with saline infusions, recovered.

A fact that tends to convince me of the benefit derived from these volatile oils in early cases is that the patients say immediate relief has been obtained. Assuming, then, that certain volatile oils known to have antiseptic properties are curative

¹ BRITISH MEDICAL JOURNAL, 1921, ii, p. 887.