know. As you see, we start with twenty-seven cases and rapidly drop month by month until at the end of seven months there are only five survivors. Patients tire of being prodded five times a day, and they get positively rebellious when their clothes won't meet in front. I'm afraid my practice is to promise: "This nuisance shall stop when you have gained another 7, 9, 12—or whatever it is—pounds." More often than not I have to keep my word when this is accomplished, and have to be satisfied if they retain that weight subsequently. Of course, those who proceed to lose it—and they never lose it as fast as they gained it—are coaxed to undergo another course. Realizing the possible significance of loss of weight, they are not reluctant.

Incidentally, there was one other respect in which the treated patients differed from the controls: none of them had extra milk. They simply had no time to fit it in.

Summary

The relation between variation in body weight and progress in cases of pulmonary tuberculosis is discussed.

The technique of administering insulin with a high carbohydrate diet is fully described.

Analysis of the results of this regime is found to compare favourably with controls regarding (a) increase of body weight and (b) improvement in the sedimentin index.

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LEAD ENCEPHALOPATHY SIMULATING CEREBRAL TUMOUR

BY

A. M. NUSSEY, M.D., M.R.C.P.

Physician to Selly Oak Hospital

AND

I. DRYBROUGH-SMITH, M.R.C.S., L.R.C.P.

Acting Physician to the Hospital

The present expansion in employment is likely to lead to some increase of industrial hazards to the workers. A case of lead poisoning exemplifying this came recently under our care, and because it simulated closely a cerebral tumour we thought it worth while recording.

Case History

A man aged 41 was for the last two and a half years employed in an arms factory as a range cleaner. His work involved collecting spent bullets from lead dust and sand into which they had been fired during practice. The amount of lead collected varied, but was usually as much as half a ton every night. The atmosphere would become thick with lead dust, and the only precaution against this was a gauze mask which fitted over his mouth on a wire frame. This by no means prevented the lead dust from reaching his mouth, as when he had finished work he would have a thick black layer on his lips and tongue. He used no gloves while working, but always washed his hands carefully before meals, and also made a habit of washing out his mouth. His firm supplied him with one pint of milk daily, which he consumed on the premises. He apparently remained fairly well until July, 1939, when he had a fit and became unconscious for a quarter of an hour. He was subsequently ill and had to stay away from work for a week, the condition being labelled by his panel doctor as one of influenza. It was noticed afterwards that he became irritable and that his memory was failing; he began to have severe frontal headaches and

diplopia on looking to the right. He never experienced abdominal pain, but was troubled with a good deal of cough and expectoration. On September 22 he had another fit, following which he remained somewhat comatose until his admission to Selly Oak Hospital on October 6. The patient has no clear recollection of that period, but remembers that his left arm and leg were twitching and that all this time he had severe headaches and profuse vomiting.

The range cleaners at the factory work in pairs, and we have since heard that the man who helped our patient was taken ill with abdominal pain a week after admission of his workmate.

Condition on Admission.—The patient was very restless and confused, and was vomiting continually. He was emaciated, and had a well-marked blue line on the gum margins. Examination of the nervous system showed the following: palsy of the right external rectus muscle and marked bilateral blurring of the disks. There was slight left facial weakness and disinclination to use the left arm and leg, but these limbs were not actually paralysed. It was difficult to decide whether this was due to some increase of tone on that side or to actual weakness. All deep reflexes were obtained with the exception of the left knee-jerk. Both sets of abdominal reflexes were present and equal, and the plantars were flexor. Astereognosis, wrist-drop, or foot-drop was not detected. Nothing abnormal was found in the heart, lungs, abdomen, and ears. The blood pressure was 120/70; neither blood nor albumin was present in the urine.

PROGRESS AND FINDINGS

Following admission he became even worse, being very restless, mentally confused, and abusive. He had delusions of being robbed and poisoned, and cried without any reason. On October 10 another fit occurred, in which convulsions were generalized, and he was unconscious for a short time. There was no incontinence, and he did not bite his tongue. A lumbar puncture was done on that day, and the C.S.F. pressure was found to be 350 mm. of water. Twelve c.cm. was drawn off, and gave the following analysis: protein, 160 mg. per 100 c.cm.; globulin +; chlorides, 714 mg. per 100 c.cm.; and a definite trace of lead. A blood count on October 9 showed haemoglobin, 70%; R.B.C., 3,660,000; and leucocytes, 11,400. Marked punctate basophilia was present. The twenty-four-hour urine on October 11 contained 1.16 mg. of lead, which is nearly ten times the highest accepted normal level. The blood urea was 60 mg. per 100 c.cm. The Wassermann reaction was negative.

The patient was kept on a milk diet and given sedatives. During the first few days he had three intravenous injections of 10 c.cm. of 10% calcium gluconate. After two weeks there was a decided improvement, and a twenty-four-hour specimen of urine was found to contain only 0.27 mg. of lead. Ten days later the urinary excretion of lead had dropped to the normal figure of 0.12 mg. Ammonium chloride, 15 grammes thrice daily, was then tentatively tried for a fortnight, but no rise in the excretion of lead was obtained, possibly owing to the fact that the dose was not adequate enough to render the urine acid. At no time did he show constipation or complain of abdominal pain. A radiograph of the skull and the long bones revealed no appreciable deviation from normal. The analysis of an extracted tooth yielded eighty-eight parts per million of lead, which, according to available statistics, is within the normal range (Roche Lynch *et al.*, 1934). It is fair to add, however, that the number of teeth so examined is very small.

The patient stayed in hospital for six weeks and made good progress. Towards the end of this time his visual acuity improved so much that he could read small print, and his behaviour, though still somewhat facile, became otherwise quite reasonable. The blurring of the disks had largely subsided, and the other neurological signs disappeared, but the blue line on the gums was still well marked. The C.S.F. pressure returned to normal, and on November 15 the fluid showed 85 mg. of protein per 100 c.cm., with a trace of globulin. The blood urea dropped to 33 mg. per 100 c.cm., and the urea clearance was 74% of normal. We saw him two months after his discharge and found the optic disks and general condition normal.

Discussion

It seemed quite clear that the case was one of lead encephalopathy, but owing to the presence of left-sided weakness and ocular changes, and marked increase in C.S.F. pressure, a space-occupying lesion in the cranium had to be considered. The history and chemical findings here led us to adopt an expectant policy, and the subsequent progress of the patient justified this fully. There are records of similar cases, especially in children, in which cerebral decompression and exploration for tumour have been carried out. Thus Bucy and Buchanan (1935) report three cases of encephalopathy in children in which similarity to cerebral tumour was so great that two of them were submitted to suboccipital decompression before the correct diagnosis was reached. Both of these, as well as a third case which was treated medically, ended in recovery. Machle (1935) described the occurrence in tetra-ethyl lead workers of delirious mania and confusion which resembled schizophrenia. Similarity of encephalopathy to cerebral tumour was observed by Eden (1935), whilst McKhann and Vogt (1933) noted that in children lead poisoning may resemble acute encephalitis. They mention as symptoms projectile vomiting, visual disturbance, stupor, coma, convulsions, elevation of blood pressure, choked disks, and, in extreme cases, separation of cranial suture. They believe that all this can be attributed to increased intracranial tension due to intense cerebral oedema, which if prolonged leads to degenerative processes owing to impairment of circulation.

We have not been able to discover records of lead content in cerebrospinal fluid in cases of lead encephalopathy, and although only a trace of it was found in the fluid of our patient, it is difficult to assess the exact significance of it because Rabinowitch and his co-workers (1933) reported the presence of lead in the cerebrospinal fluid of every individual in a small series of cases of lead poisoning. On the other hand, we recently had a patient suffering from severe lead colic whose blood lead was 0.335 mg. per 100 c.cm. (normal is *nil* to 0.1 mg.), whose twenty-four-hour excretion of lead in the urine was 0.89 mg. per 100 c.cm. (normal, *nil* to 0.11 mg.), and whose cerebrospinal fluid contained no lead.

It is interesting to note also that no gross abnormality was found in the urine of our patient, although renal function appeared impaired, and his urea clearance was still at the lowest limit of normal even after he had improved.

Considering the possible changes in the brain, which include liquefaction of the nerve cells (Lehmann *et al.*, 1926), extensive chromatolysis, and some glial proliferation (Cadwalader, 1912), our patient appears to have escaped lightly, from the point of view both of mental and of eye changes.

Summary

The findings of a patient with lead encephalopathy are described.

Superficial similarity to cerebral tumour is pointed out.

Full recovery on expectant treatment is outlined.

We should like to record our gratitude to Mr. F. Morton, the hospital biochemist, for carrying out the analyses and for his helpful criticism.

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SULPHANILAMIDE AS A PRESERVATIVE
IN STORED BLOOD

BY

R. F. HUNWICKE, B.Sc., F.I.C.

(From the Bacteriological Department, Glaxo Laboratories Ltd.)

Milan Novak (1939) describes experiments which indicate that the addition of small amounts of sulphanilamide to stored blood may be expected to prevent the development of contaminating saprophytes at low temperatures. These contaminants are often present in blood that has been stored for transfusion purposes, and they develop at refrigerator temperature, making the blood unfit for use for transfusions. The same phenomenon has been observed at times in this laboratory with horse blood for culture media: blood which is apparently sterile immediately after withdrawal from the horse begins to show the development of organisms after storage for a certain period in the refrigerator, with resultant wastage. The possibility of adding a harmless substance which will prevent the development of organisms during storage is of some importance. The following experiments are confirmatory of Novak's conclusions.

Experiment 1

A rabbit was bled from the ear into citrate solution. To the blood a mixture of organisms isolated from contaminated human blood was added so as to give approximately 100 per c.cm. These organisms were mostly staphylococci and streptococci, with a few common sporing bacilli of the *subtilis-mycoides* type. To one portion of blood sulphanilamide 1:5,000 was added. The other portion was a control without any addition. Both samples were stored in the refrigerator at 2° C. and plated at intervals, the plates being incubated for three days at 37° C. The results are shown in Table I.

TABLE I

	Initial	3 Days	7 Days	14 Days	20 Days
+ Sulph. 1 : 5,000...	150	200	400	22	26
Control	100	2,000	Uncountable	Uncountable	Uncountable

Experiment 2

240 c.cm. of oxalated rabbits' blood freshly drawn was divided into twelve equal portions. Sulphanilamide was added to give three portions containing 1:1,250, three with 1:2,500, and three with 1:5,000. The remaining three portions had none. To each dilution and each control a suspension of (a) *B. subtilis*, (b) *B. pyocyaneus*, (c) staphylococcus was added to give 10 to 100 organisms per c.cm. The samples were kept in the refrigerator and plated at intervals. The temperatures of refrigeration and incubation of the plates were the same as in Experiment 1. The results are shown in Table II.

TABLE II

	Initial	3 Days	6 Days	14 Days	22 Days	29 Days
<i>B. subtilis</i> :						
1 : 1,250	28	17	7	11	8	11
1 : 2,500	29	33	34	7	14	4
1 : 5,000	24	28	13	6	22	42
Control	56	19	48	14	800	1,000
<i>B. pyocyaneus</i> :						
1 : 1,250	35	20	7	13	0	7
1 : 2,500	26	33	22	16	2	0
1 : 5,000	21	22	38	42	2	0
Control	99	26	60	40	29	2,000
Staphylococcus :						
1 : 1,250	22	21	24	7	10	10
1 : 2,500	16	34	20	12	21	16
1 : 5,000	39	31	26	27	13	2
Control	48	37	49	49	20	13