the fluid had a definitely increased globulin; the cells were increased to 16 lymphocytes, and the mastic test now showed a paretic type of reaction, *i.e.*, 5553221000000.

Diagnosis.—Hypoglycæmia was proved but the cause had not been determined. There was no evidence of Addison's disease. Her basal metabolism was plus 20 per cent, and there was no suggestion of pituitary or thyroid disease. Thus the only other possibilities were hyperinsulinism and a possible cerebral lesion involving the sugar centre in the pons. This latter highly theoretical possibility would not have been entertained if it had not been for the suggestive findings in the ventriculogram taken several months previously, the marked cerebral symptoms in the attacks, and the peculiar cerebrospinal fluid findings. However it was felt that if a cerebral lesion had existed there would have been localizing signs after several months. There is no record in the literature of similar findings in hypoglycæmic cases, but it is significant that in two cases of McLenahan and Norris that came to autopsy there were marked central nervous findings, including extensive Nissl's degeneration in the ganglionic cells and marked swelling of the axon sheaths. It is not difficult to imagine that the spinal fluid of these cases might well have reflected these organic changes, with findings similar to or even more marked than those in the present case. Further it was thought that the numerous violent cerebral episodes in this case immediately preceding the spinal fluid findings might easily explain them. Accordingly a tentative diagnosis of hyperinsulinism was made and an exploration of the pancreas decided on.

Operation.—On March 10, 1936, a laparotomy was performed, under general anæsthesia, by Dr. J. A. Gunn, and a small adenoma was found on the posterior surface of the pancreas near the tail. It measured one centimetre in diameter and was almost spherical. It was firm and encapsulated and weighed 0.553 grams. The cut surface was moist, bulging, and yellow with white bands. Microscopically it was an adenoma of the islets of Langerhans. (The pathological description is taken from the report of the pathologist, Dr. William A. Boyd.)

Progress of the case.—The patient made an uneventful recovery. There have been no further attacks and the blood sugar has not gone lower than 80 mg. per 100 c.c. on any examination. The basal metabolic rate dropped to minus 26 per cent four weeks after the operation and rose to minus 9 per cent on April 20th. The cerebrospinal fluid obtained six weeks after the operation showed a normal globulin content, 3 lymphocytes, and the mastic test showed a faint first zone reaction.

SUMMARY

1. A case of hyperinsulinism, associated with severe nervous manifestations, due to an adenoma of the pancreas, is presented.

2. An earlier diagnosis of a possible intracranial lesion is discussed because it emphasizes the frequent difficulties in diagnosis of the severe cases, and because of the misleading, though interesting findings, in the ventriculogram and cerebrospinal fluid.

3. Interesting basal metabolic rates are recorded.

4. Interesting cerebrospinal fluid findings are recorded, with suggestions as to their cause.

We wish to acknowledge our indebtedness to Dr. J. A. Gunn who performed the operation, to Dr. F. A. L. Mathewson who did the preliminary investigation in the case, and to Prof. A. T. Cameron, of the Department of Biochemistry.

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A CASE OF TURPENTINE POISONING

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The following report of a death due to the injudicious use of turpentine in the treatment of an infant is presented in order to draw attention to the danger attending the internal use of this drug. The present-day therapeutic use of turpentine is practically limited to external medication such as stupes, various liniments, inhalations, and its common use is in enemata. From its ease of purchase and the instructions on the ready-printed label one may surmise that this drug is a common household remedy. As such, it is frequently given internally for threadworms and for a hundred and one other complaints not serious enough to require a physician's immediate attention.

The victim was a female infant, eleven months old. She was seen for the first time at home on December 24, 1934, and was sent to hospital at once. (Hôtel-Dieu Hospital No. K. 535). She was admitted at 3.15 p.m.

The history given by the mother was that the baby had had cramps on the night of December 23rd, and cried almost continually. On the morning of the 24th she had an emesis. "Baby's Own Tablets" were given but the infant continued to cry. At about 2.30 p.m. the child was unconscious and in a convulsion, so a doctor was called.

On admission to hospital the infant was in coma. Her eyes were glassy, rotated upwards and the pupils were dilated. There was marked cyanosis. The temperature was 107° (rectal); respiration was irregular and shallow, the rate varying between 80 and 100 per minute; the heart rate was between 250 and 300 per minute.

Alternate hot and cold baths were given for almost an hour before the cyanosis disappeared and breathing became more regular. Ten minims of adrenalin were injected during resuscitation. A soap suds enema returned clear, excepting for much mucus. Catheterization gave only one dram of urine. An ice cap was applied. The temperature at 4 p.m. was 103° (rectal). The cyanosis had disappeared and the general condition was slightly improved, but the pulse was thready and the respirations rapid. Consciousness was never regained and the infant died at 6.15 p.m.

Summary of post-mortem report.—The body was that of a well developed child. The skin was pale. No odour of chemicals came from the mouth. The thymus was large and there were numerous hæmorrhages, small and scattered. The heart showed some dilatation of the right side. The bronchi contained a considerable amount of muco-purulent exudate and the mucous membrane was congested. The stomach was distended with gas and on opening a definite smell of turpentine was obtained. There was little in the way of content; what there was was coffee-ground in appearance. The mucosa

was slightly congested. The intestines showed no abnormality. The mesenteric glands were enlarged and pink in colour. The lymphoid tissue at the lower end of the ileum was well developed. The kidneys and liver were pale; spleen enlarged and mottled. The bladder contained no urine. The brain showed congestion, but no other abnormality.

Conclusion: (1) turpentine poisoning; (2) acute bronchitis; (3) enlarged thymus; (4) general lymphoid hyperplasia.

On further questioning, the infant's grandmother revealed that she had given two teaspoonfuls of spirits of turpentine to the infant on the morning of December 24th. She thought that the child had worms.

Bruce and Dilling[1] give the dosage of spirits of turpentine by mouth as 2 to 10 minims, and the anthelmintic dose for an adult, 3 to 4 drams. They discuss the usage of turpentine internally as a carminative, hæmostatic and powerful anthelmintic for tapeworm. The drug is said to have a sedative effect on the cerebral and spinal centres. It is useless as an antidote in phosphorus poisoning. Cohen and Githens[2] state that turpentine is not very toxic; it takes 150 minims, in subcutaneous injection, to kill a rabbit; a dog will be killed by one ounce by mouth. The same writers claim that half an ounce is sufficient to kill a child of 14 months, although a child of the same age is said to have survived after taking four ounces by mouth. Adults have survived after one ounce, and even after three to four ounces, but in both instances there was coma for a long time. On the other hand intoxication has been reported from doses as small as 20 drops of a 0.1 per cent solution, repeated several times. Some people exhibit a decided idiosyncrasy toward turpentine. In these 30 minims will cause vomiting and diarrhœa. In larger doses there is an acute enteritis, vomiting of mucus, which is often bloody, diarrhœa, and passage of blood-stained mucus. Large doses have a marked effect on the kidneys, causing albuminuria, hæmaturia and even complete suppression.

A search of the available literature reveals little if anything definite on the subject of turpentine poisoning, and one is forced to conclude that the condition is rare and a fatal case even more so. As to treatment, it is that of any irritant poison, evacuation of the stomach and bowels, morphia for pain, and demulcent drinks. It is self-evident that the use of this drug as a household remedy should be checked.

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Clinical and Laboratory Notes

THE TYPES OF MYCOBACTERIUM TUBERCULOSIS PREVAILING IN MONTREAL*

(AN INTERIM REPORT)

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This work was undertaken for the Shriner's Hospital for Crippled Children in Montreal, to determine the incidence of infection in man by the bovine type of *M. tuberculosis* in and about Montreal. The specimens were obtained mainly from the Shriner's Hospital and the Children's Memorial Hospital; occasional specimens were received from other Montreal institutions. The majority of the patients were children under fourteen years of age who lived in Montreal and its vicinity. A detailed classification of the cases will be made on completion of the investigation.

The procedure used in the isolation of the tubercle bacilli was the one in use in the laboratory. Both guinea-pigs and cultural methods were employed if the specimen was suitable; otherwise the material was either injected into animals or inoculated on media as circumstances indicated.

In cases which yielded tubercle bicilli a pure culture was obtained on Löwenstein's medium, with and without glycerine. The procedure used to determine the type of organism differed only from that used in previous investigations by Griffiths (1911-1928); Price (1932) and other workers in that inspissated serum, which has been used largely to provide organisms for animal inoculations and for seeding on media, was replaced by Löwenstein's medium. This, it seemed, gave a more rapid and copious growth and was more easily emulsified. It was rarely possible to use Löwenstein's media made with-

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