THE ASCHHEIM-ZONDEK TEST IN THE PUERPERIUM

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There is no general agreement as to when, following uneventful pregnancy and normal parturition, the Aschheim-Zondek test first gives a definite and permanent negative result. Aschheim and Zondek¹ state that "as little as 0.05 c.cm. of urine may give a positive reaction at the end of pregnancy. The reaction may disappear as early as five days after childbirth, and regularly ceases eight days after." Mazer and Edeiken² express the view that "the source of prolan is the living chorionic epithelium, which should not persist longer than two weeks after the termination of a normal pregnancy." According to Hamburger³ "immediately after parturition there is a very great and abrupt fall in the hormone concentration (within twenty-four hours down to about 1/20), and then in the next dozen days the hormone content in the urine falls off below any demonstrable amount." Mack and Agnew⁴ refer to four cases in which the test was carried out two to seven days following parturition, giving positive results. Robson,⁵ quoting Zondek,⁶ says that the test remains positive not only so long as living products of conception are present, but for some days after that period ("about a week to nine days as a rule").

In connexion with the work of the Pregnancy Diagnosis Laboratory it was found desirable to examine this question. Through the eager co-operation of the honorary and nursing staff of the Royal Simpson Memorial Hospital, Edinburgh, specimens from a series of fifty cases were made available for the purposes of this study. From each case a specimen of urine was obtained as soon as possible after parturition, and thereafter at twenty-four-hour intervals. The results obtained are given in the following table:

		Positive	Negative
1st specimen ...	2-7 hours after parturition	45	5
2nd	24 hours later	26	24
3rd	48 ..	15	35
4th	72 ..	2	48
5th	96 ..	0	50
6th	120 ..	0	50
7th	144 ..	0	50

Of the five cases in which the first specimen gave a negative result one gave a weak positive (one blood spot in one mouse) at the second test and negatives thereafter: the other four continued to give negatives consistently. In two cases in which the first specimen gave a positive and the second a negative result, the forty-eight-hour specimens evoked weak positive reactions, whilst the seventy-two-hour and ninety-six-hour specimens were negative again. The two seventy-two-hour positives were both weak. In those cases in which a series of positives was succeeded by a negative, the last of the positives was, as a rule, a weak reaction.

It may be concluded that under the conditions which obtain in this laboratory, the Aschheim-Zondek test invariably yields a definite and (presumably) permanent negative result ninety-six hours after normal and complete parturition. The results obtained suggest that the Aschheim-Zondek test on the fifth day of the puerperium might well be used as a routine procedure for the diagnosis of retained living products of conception.

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Clinical Memoranda

Strychnine Poisoning Treated Successfully

A strong, healthy young man, aged 22, bought 6 grains of strychnine hydrochloride for the purpose of suicide. He obtained the poison by stating that he wished to poison rats, went home, dissolved it in water, and drank it. While doing so he remarked to a member of his family that he was taking poison and would be dead in half an hour. The family doctor was summoned immediately, and the patient was rushed off to hospital within twenty minutes of swallowing the draught.

CASE REPORT

On admission at 1 p.m. twitching had commenced. Apomorphine was not obtainable, so zinc sulphate 30 grains was administered. By the time this had been done all the signs of severe strychnine poisoning were in evidence, with exceptional opisthotonos and pronounced cyanosis and asphyxia.

Chloroform anaesthesia was administered, and as this did not entirely control the intense spasms, at 1.30 p.m. he was given a hypodermic injection of morphine sulphate 1/2 grain. In spite of this treatment the patient became so deeply cyanosed that amyl nitrite pearls were resorted to, and this rapidly relieved the asphyxia. The stomach was washed out with about a gallon of potassium permanganate solution, and at least half a pint was left in the stomach.

FURTHER DRUG THERAPY

At 2 p.m. the convulsions were still violent, and another hypodermic injection of morphine 1/2 grain, with atropine 1/50 grain, was given.

At 3 p.m. chloral hydrate 30 grains and potassium bromide 60 grains were given per rectum, and at 3.45 p.m. a third hypodermic injection of morphine sulphate 1/4 grain and atropine 1/100 grain was administered. At this time the pulse was 142, the spasms, though less frequent, were still severe, and the degree of cyanosis was alarming. Chloroform anaesthesia was maintained with the occasional administration of amyl nitrite to relieve asphyxia and cyanosis.

At 7 p.m. chloral hydrate 20 grains and potassium bromide 15 grains were given again per rectum.

At 9.30 p.m. the patient appeared to be in a very critical condition: his respirations had assumed a Cheyne-Stokes character, and there was grave cyanosis. He was now given atropine 1/100 grain hypodermically.

At 9.45 p.m. carbon dioxide and oxygen were administered until 10.15 p.m., when the spasms diminished, cyanosis became less and less, and the respirations assumed a more normal character and rhythm. He awakened, asked for water, and then fell off into a deep sleep, which lasted until 6.15 o'clock the next morning. At 7.55 a.m. he vomited and complained of severe headache, but in other respects he felt well. He did not seem any the worse for his ordeal, and was discharged the following day.

COMMENTARY

There is no doubt that the time factor operated in helping to save this patient, for there was little or no delay in getting him to hospital. Deep chloroform anaesthesia, even combined with large doses of morphine, did not relieve the severe asphyxia resulting from the spasms of the respiratory attacks, but when combined with amyl nitrite the effect was magical.

Spinal puncture and the injection of a 25 per cent. solution of magnesium sulphate was considered, as also

was the intravenous injection of sodium evipan, but both were abandoned as being too risky, especially in a case of this sort when the spasms were so frequent and severe; so we relied on our old friends and won through.

Our thanks are due to the other members of the staff who rendered valuable assistance and are in great measure responsible for the successful result.

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Cerebral Cysticercosis Simulating Epilepsy

A short note of this case may be of interest at this time, as it comes so recently after the annotation on "Cysticercosis and Epilepsy" in the *Journal* (December 21st, 1935, p. 1214) and the correspondence which followed (*Journal*, January 4th, p. 32). It emphasizes, too, the advice quoted there, from Dixon and Smithers, to suspect cysticercosis in a patient previously healthy who has lived abroad if he develops fits or anomalous nervous or mental symptoms.

CASE REPORT

The patient was a man, 50 years of age, who had served in India twelve to fourteen years ago: he gave a history of being subject to malaria since this service. Before and after being in the Army he was a caulker in a shipyard. From 1923 to 1933 he led an apparently normal life. In 1933 he began to suffer from epileptiform seizures, and one year later was admitted to an epileptic colony, where he remained until shortly before his death. He was subject to fits of depression, occasionally with confusion, and at one time he had an outburst of religious mania. He had indefinite seizures in which his temperature rose to over 101° F., perspiration became profuse, and incontinence and exhaustion followed, though consciousness was not lost. These signs were not indicative of true epilepsy, and other investigations resulted as follows. The urine and faeces were normal; there was abundant foul sputum, but examination for tubercle bacilli was negative, and x-ray examination of his chest showed no abnormality. His reflexes were normal, and the Wassermann reaction was negative; a blood film taken in August, 1935, showed a 6 per cent. eosinophilia.

In September, 1935, the patient began to suffer from cerebral vomiting; lumbar puncture showed that the cerebro-spinal fluid was under slightly increased pressure. The vomiting increased in frequency, and a severe and persistent headache developed over the occipital region. The patient's condition became rapidly worse, and he died on December 3rd, five hours after admission to hospital.

POST-MORTEM FINDINGS

A post-mortem examination was performed on December 4th. There were no points of interest in the organs of the chest and abdomen. In the brain there was evidence of increased intracranial pressure: the convolutions were flattened, and there was little cerebro-spinal fluid over the vertex; the lateral and third ventricles were slightly dilated. In the brain substance there were about thirty rounded cyst-like structures, varying in size from 0.5 cm. to 2 cm. in diameter. In a horizontal section at the level of the basal nuclei two were present in the left and one in the right occipital lobes, one at the posterior end of the left island of Reil, and one in the left frontal lobe; these were all subcortical. Others were seen in the basal nuclei—one in the left caudate nucleus, three in the right thalamus, and one in the right lenticular nucleus; one lay at the tip of the right caudate nucleus. Another one was present in the membranes of the right Rolandic fissure. Some of them had thin translucent walls with clear fluid inside, and contained small solid structures attached by pedicles to the walls; in others the walls were thicker and the solid bodies inside were more opaque; others were smaller and denser, and contained calcified material.

Microscopically these rounded structures were seen to be parasitic cysts, each containing a scolex with four suckers

and a ring of hooklets, the denser ones showing calcification in parts. A specimen was submitted to Professor R. T. Leiper, professor of helminthology of the London University and the London School of Hygiene and Tropical Medicine, who kindly supplied the information that they were *Cysticerci cellulosae*, larvae of the *Taenia solium*.

This patient's history before 1934 was very indefinite, but there was no evidence of his having passed segments of a worm, and no adult tapeworm was found post mortem; so he does not seem to have infected himself. This case corresponds, then, to the description of cases given by Colonel W. P. MacArthur in his article on "Cysticercosis as Seen in the British Army, with Special Reference to the Production of Epilepsy,"¹ when cysticerci were confined to the brain.

I wish to acknowledge my indebtedness to the doctor who provided the clinical notes of this case, and to Professor Leiper.

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Obesity in Pulmonary Tuberculosis

A pronounced gain in weight in cases of active pulmonary tuberculosis under institutional treatment is a frequent occurrence, but for a patient to double her weight in a period of eight months must be exceptionally rare.

A dressmaker, aged 25, was first seen in October, 1933, at the Tuberculosis Dispensary at the Royal Northern Hospital. She complained of cough with expectoration, lassitude, and loss of weight over a period of six weeks, but had continued her work until two weeks previously. On inspection she was obviously pale, wasted, and ill, pulse 108, and temperature 100.2° F. Examination of the chest showed impaired resonance over both upper lobes, while there was marked dullness and obvious cavitation at the left base, coarse rales being audible in this area. A skiagram showed coarse tuberculous mottling at the left base and finer mottling in the right upper lobe. The sputum was positive. Her weight was 6 st. 12 lb. (clothed), her normal weight being 7½ st. In view of the patient's general condition and the well-marked basal location of the lung lesion a rather gloomy view of the prognosis was taken. Sanatorium was advised, but the patient and her parents were very strongly against this course, and, accordingly, she was advised to rest thoroughly at home under the care of her doctor. This she did, and was waited on assiduously by her mother while she remained conscientiously in bed for seven months. During this period her cough and sputum gradually disappeared.

In July, 1934, she reattended the Royal Northern Hospital, and I was astonished to find that her weight was now 13 st. 8 lb. She was obviously grossly obese and almost top-heavy. There was well-marked tachycardia and dyspnoea, associated, I think, almost entirely with the obesity. The lung signs had much improved. The left basal cavities seemed diminished in size and drier, and an x-ray showed considerable clearing-up of the previous shadows. The patient, who was naturally sensitive about her changed appearance, was advised to diet and gradually increase the amount of exercise taken. This, however, has only resulted in a loss of 5 lb. to 13 st. 3 lb. at the present time. She has no symptoms referable to the lungs.

This isolated case is reported as a pathological curiosity from which no conclusions can be drawn. One cannot help wondering, however, whether an initial period of two months in bed in every case of pulmonary tuberculosis, even if afebrile, would not give better results than the method adopted in most English sanatoria of getting a patient on to his feet almost as soon as the temperature has fallen to normal.

I am indebted to Dr. MacWhirter for permission to report this case, occurring in his panel practice.

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¹ *Journ. of R.A.M.C.*, 1934, lxii, No. 4.