believe she was pregnant. Reckoning, somewhat academically be it admitted in view of this particular patient's irregular menstruation, on a 28-day cycle and a missed period, the date of onset coincided with the probable time of ovulation in the new cycle.

Examination under anaesthesia was carried out. Nothing definite could be felt, but an impression was received of a vague soft swelling in the left fornix. Needling of the posterior fornix was contemplated but not undertaken, as the finding of blood in such a case would not have further clarified the diagnosis.

The abdomen was opened under curarine and cyclopropane anaesthesia. A moderate amount of free blood was found in the pelvis. The uterus appeared normal in size and consistency. The left ovary was found to have a ruptured follicle which appeared to have been about the size of a marble. A half-handful of blood clot was adherent to this. Immediately adjacent was a dark swelling occupying approximately the base of what had been the follicle. A light prick of the scalpel showed the yellow convolutions of a recent corpus luteum. It seemed impossible to say whether one was dealing with one or two structures—a cystic corpus luteum of pregnancy or a ruptured Graafian follicle adjacent to a recent corpus luteum. The other ovary was normal in appearance. Apart from removing the blood clot and making sure that bleeding had ceased, nothing was done and the abdomen was closed.

Next morning a specimen of urine was taken for an Aschheim-Zondek test. On the third day the patient announced that she was menstruating. She had a painless loss, which ceased after four days. The Aschheim-Zondek test was reported weakly positive. Such a result can be obtained immediately before the onset of menstruation. In view of the events outlined it was thus interpreted at first. Two days later bleeding per vaginam was resumed and was followed by slight brownish discharge which continued. Finally on Aug. 8 curettage was undertaken and fragments of an early ovum were found in process of extrusion. Histological examination confirmed the presence of chorionic villi. Recovery thereafter was uneventful.

The events subsequent to laparotomy make it clear that the emergency had been due to rupture of the corpus luteum of pregnancy. Whether or not this had been cystic it is impossible to say, as the normal size is known to have wide variations. It seems likely, though not necessarily conclusive, that the abortion was attributable to the impairment of the integrity of the corpus luteum from traumatic rupture.

W. R. SLOAN, M.D., F.R.C.S.Ed., M.R.C.O.G.

Unusual Case of Meningococcal Septicaemia

Meningococcal septicaemia is usually considered to be either an acute fulminating condition associated with the Waterhouse-Friderichsen syndrome or as a more chronic form characterized by intermittent fever, a rash which is frequently purpuric, and arthritis. The following case corresponds with neither of the accepted varieties and presented some other unusual features.

CASE RECORD

A boy aged 9 was admitted to hospital on Dec. 28, 1946, with a history of having developed a feverish cold and a rash on the hands and trunk on Dec. 21. Medical advice was not sought till the day of admission, when the patient developed severe pain in the chest and abdomen.

On admission his general condition was poor: temperature 101.6° F. (38.7° C.); pulse 110; respirations 48. There was well-marked cyanosis and a generalized haemorrhagic rash varying from areas two inches (5 cm.) in diameter to pin-point petechiae. He had a well-marked pericardial effusion with dullness three inches (7.5 cm.) outside the nipple line. The liver was enlarged four fingers' breadth below the costal margin, evidently resulting from heart failure. Moist sounds were generalized throughout the chest, but in addition there was evidence of consolidation of the left lower lobe.

There was no spinal retraction, headache, neck rigidity, or impairment of consciousness, but Kernig's sign was positive and there was a bilateral extensor plantar response. Turbid fluid was obtained both on lumbar puncture and on aspiration of the pericardium. The turbidity of these fluids was mainly due to polymorphs—3,500 per c.mm. in the case of the cerebrospinal fluid. Glucose broth cultures of the blood and C.S.F. gave negative results, but meningococci were grown from the pericardial effusion.

Treatment was instituted as follows. *Sulphonamides*: soluble sulphathiazole 1 g. intravenously followed by sulphadiazine by mouth 1 g. four-hourly. *Penicillin*: 25,000 units in 5 ml. saline intrathecally after the removal of 20 ml. C.S.F. followed by injections of 100,000 units intramuscularly, giving the first two doses at three-hourly and subsequent doses at six-hourly intervals. Oxygen was given continuously and 20 ml. of fluid was aspirated from the pericardium.

Marked improvement followed except for a temporary extension of the consolidation of the left lung. By Dec. 30 the liver edge

was only one finger's breadth below the costal margin and the cardiac dullness was one inch (2.5 cm.) outside the nipple line. A loud pleural rub was present over the consolidated lung tissue. By Jan. 1 the pericardial effusion was seen to have rapidly re-formed; 70 ml. was aspirated and 50,000 units of penicillin injected into the pericardium. The aspirated fluid was now sterile. The signs of effusion were replaced by a loud pericardial rub. Intramuscular penicillin was discontinued on Jan. 5 after it had been reported that meningococci had been isolated. Sulphadiazine was reduced to 0.5 g. four-hourly on Jan. 1 and discontinued on Jan. 5. From Jan. 2 the patient had been completely afebrile and on the following day further improvement permitted the discontinuance of oxygen. By this time the rash had also faded.

Except for persisting consolidation of the left base improvement was maintained till Jan. 7, when the temperature rose to 99° F. (37.2° C.) and the pericardial effusion again re-formed; 50 ml. of blood-stained fluid was aspirated and again proved to be sterile. The effusion did not re-form again and the patient was afebrile from Jan. 9 onwards. Nevertheless from Jan. 7 congestive heart failure became progressively more acute, as evidenced by increasing enlargement of the liver-dyspnoea, and the development of ascites. Treatment with oxygen, digitalis, and subsequently with calcium failed to arrest the condition and the patient died on Jan. 16.

Necropsy on the same day showed the following:

Heart.—The fluid had absorbed and the pericardium was widely adherent. The condition was one of healing fibropurulent pericarditis. The myocardium showed cloudy swelling with vacuolation of some cells suggesting fatty degeneration. The heart valves were apparently healthy.

Lungs.—Septic infarcts with evidence of early organization. Areas of collapse, engorgement, and bronchiectasis.

Spleen.—Firm consistency; congested but otherwise healthy.

Kidneys.—Cloudy swelling of the tubular epithelium.

Liver.—Congestion atrophy and fatty degeneration of the nutmeg pattern.

Adrenals.—Congestion of the zona reticularis; otherwise healthy.

Meninges.—Subpial fibroblastic reaction in the region of the optic chiasma indicating previous inflammations; otherwise normal.

COMMENT

The unexpected features of this case were: (1) localization of the meningeal reaction resulting in turbid C.S.F. without clinical meningitis; and (2) death from carditis rather than from adrenal apoplexy—more commonly associated with meningococcal septicaemia—occurring after the infection had been eliminated.

I have not been able to find any reported case of a similar localized meningitis. In an article on meningococcal infection in the *Annals of Internal Medicine* (April, 1946) Goldbloom, Nickman, and Leidman noted a few cases in which there were C.S.F. changes without clinical meningitis, but these changes were considered as precursors of a general meningitis rather than as a localized meningeal infection.

Pericarditis is usually listed as one of the rarer complications of meningococcal infection. Hartwell (1939) reported a fatal case of meningococcal myocarditis and was able to discover twelve other cases in the literature. Saphir (1936) stated that meningococcal myocarditis was common in those who succumbed to the infection, and commented on the possible confusion with the Waterhouse-Friderichsen syndrome. More recently Holman and Angevine (1946) reported two cases in detail. One patient had a fatal heart attack on the 33rd day of illness after having been allowed to get up; severe myocardial degeneration was confirmed at necropsy. In the other case the diagnosis was based on clinical and electrocardiographic evidence. They note that toxæmia and severe systemic infection are features of recorded cases and consider meningococcal infection a notable omission from the listed causes of myocarditis. In my own patient it would appear that the embarrassment of a recently and extensively adherent pericardium added to the effect of myocardial degeneration to produce fatal heart failure.

I wish to thank Dr. W. H. Hughes and Dr. W. D. Linsell, who performed the post-mortem examination, and Dr. D. M. Pryce, who carried out the histological examinations. I am grateful to Dr. R. Leader, M.O.H. Ealing Borough, for permission to publish this case.

J. J. LINEHAN, M.D., D.P.H., D.C.H.

REFERENCES

- Hartwell, R. M. (1939). *Amer. J. Dis. Child.*, **55**, 823.
Holman, D. V., and Angevine, D. M. (1946). *Amer. J. med. Sci.*, **211**, 129.
Saphir, O. (1936). *Amer. J. Path.*, **12**, 677.