were felt and no foetal heart was heard. The uterus was contracting every 10 minutes. On vaginal examination the cervix was high up and admitted two fingers. The membranes were tense and bulging, and a foetal head was ballotted without difficulty.

The patient was admitted to the ward and given pethidine hydrochloride, 100 mg. intramuscularly. After routine preparation she was taken to the theatre and the membranes ruptured. Nine pints (5 litres) of fluid was removed, the symptoms being almost immediately relieved. On examination the abdomen was still slightly tender, the uterus had contracted down on to the foetus, and a foetal heart could then be heard. An abdominal binder was applied, but the patient objected to this and it was removed after an hour. She went into labour and was delivered 12 hours later quite normally. There was no post-partum haemorrhage. The patient felt so well that it was with difficulty that she was persuaded to stay in hospital for three days. When she left her condition was good and there was no albuminuria or oedema. I have not seen her since.

The foetus was a stillborn male, and, as in most of these cases, it was abnormal. It was estimated to be about eight weeks premature (the patient gave a history of seven months' amenorrhoea, but this discrepancy is common in our practice here), it had a rather cretinous appearance, it had no ears except for small rudimentary knobs of skin, the external auditory meatus was absent on both sides, and the right foot was clubbed. No other congenital abnormalities were detected, and the oesophagus was patent.

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Fatal Case of Agranulocytosis following Phenylbutazone

The following case is of interest because of: (1) the small dosage and duration of phenylbutazone administration (14 g. over 27 days); (2) the appearance of a rash six days after cessation of treatment; (3) the long interval between the cessation of the drug and the recovery of marrow function (19 days); and (4) the tragic consequence of the administration (even though every precaution was taken) of a toxic drug, instead of an inert one, as a placebo to a patient whose symptomatology was largely functional.

CASE REPORT

A woman aged 49 was admitted to hospital on June 22, 1953. She had complained of "arthritis" for many years, and for the preceding two years had been under treatment by the general practitioner who had sent her to hospital. During the two years' treatment he had tried most of the common symptomatic drugs—phenobarbitone, aspirin, codein. co., etc., and her husband had spent many hours massaging her various aching areas, chiefly back, lower chest, and thighs. Periodically she had complained of pain and stiffness and swelling of her fingers, knuckle-joints, and the small joints of her feet. She had begged for the "new treatment" of which she had heard. In view of reports of toxic effects her doctor had given way with misgivings, and after a blood examination on May 14 (haemoglobin, 95%; white cells, 8,400; differential count, "within normal limits"; E.S.R. (Westergren), 18 mm. in one hour, phenylbutazone ("butazolidin"), 200 mg. three times a day, was started on May 19, and she was advised to stop taking her sleeping-tablets—sodium amytal, 3 gr. (0.2 g.) nocte.

On May 28 her haemoglobin was 96%, and the white-cell count 5,200, the differential count being "within normal limits." By June 4 she had noticed no improvement, although she had not taken the tablets regularly. She was

advised to continue with them a little longer, and then to stop if they did not help. On June 14, for that reason and because she felt somewhat nauseated, she stopped taking them. On the 18th sodium amytal was again taken because she had not been sleeping well, and on the 20th she was covered with a rash. This was reported to her doctor the next day (Sunday), when he made sure that she had stopped the tablets, and arranged for a blood test next morning. This revealed haemoglobin, 81%; white-cell count, 1,600 (polymorphs 2%, small lymphocytes 92%, large lymphocytes 4%, monocytes 1%, Türk cells 1%); E.S.R., 20 mm. in one hour (Westergren). She was immediately sent to hospital.

Examination showed her to be an anxious alert woman, with a temperature of 101° F. (38.3° C.), a generalized maculo-papular rash, a slightly injected throat, and no clinical evidence of arthritis. Crystalline penicillin, 1,000,000 units intramuscularly three-hourly; pyridoxine, 50 mg. intramuscularly daily; liver extract ("livogen"), 2 dr. (7 ml.) three times daily; and vitamin-B complex, one tablet three times daily orally, were given. Sodium amytal, 3 gr. (0.2 g.) nightly, was prescribed as a sedative, to be taken as desired.

Clinically she was progressing well on June 26, the rash had nearly faded, and the white-cell count was 4,000 (polymorphs 1%). Next day the white-cell count was 4,200 (polymorphs 2%, small lymphocytes 93%, large lymphocytes 5%); platelets 150,000. On the 28th her temperature, which had been averaging 100° F. (37.8° C.), was elevated to 104° F. (40° C.). She had a cough and complained of pain in the left side of her chest; there were signs of consolidation at the left base. Oxytetracycline ("terramycin"), 750 mg. six-hourly, and oral vitamin K, 10 mg. twice daily, were added to the previous treatment. The white-cell count was 1,600 (small lymphocytes 98%, large lymphocytes 2%, no polymorphs). On June 30 she was delirious and irrational. The white-cell count was 1,000 (small lymphocytes 96%, large lymphocytes 4%, no polymorphs). Next day there was scattered consolidation over both lungs; she was dehydrated, and it was necessary to begin intravenous therapy cautiously.

A myelogram revealed: Neutrophils—polymorphs 0, metamyelocytes 6%, myelocytes 43%, promyelocytes 3%; blast cells, 2%; lymphocytes, 25%; monocytes, 2%; megakaryocytes, 1%; nucleated red cells and normoblasts, early 2%, intermediate 15%, late 1%. The myeloid-erythroid ratio was 1.5 : 1 (normal 8 : 1 to 2 : 1), the leuco-erythrogenic ratio was 1.5 : 1 (normal 1.5 : 1 to 2.5 : 1). *Interpretation:* This is a cellular marrow in which there is evidence of active erythropoiesis and leucopoiesis. The granular cells are all immature.

The white blood cell count was 800; no polymorphs seen. On July 2 the white-cell count was 600 (polymorphs 1%). Next day her temperature, which for the previous five days had remained between 103 and 104° F. (39.4 and 40° C.), had dropped to 100° F. (37.8° C.), and the white-cell count was 4,300 (polymorphs 67%, small lymphocytes 28%, large lymphocytes 1%, monocytes 1%, eosinophils 1%, metamyelocytes 2%). However, she was still very confused mentally, and was cyanotic; there was evidence of diffuse bronchopneumonia.

On July 4 the white-cell count was 11,200 (polymorphs 74%, metamyelocytes 4%) and her temperature was within normal limits, but she died early the next morning.

Post-mortem Examination.—The chief findings were:—Respiratory: some adhesions in the left pleural sac; some frothy mucus, slightly purulent, in the air passages, chiefly the right bronchus. Lungs: multiple small abscesses and hypostatic oedema. Spleen: enlarged (230 g.), soft, diffuent. Liver: a few small abscesses. In other words, findings of septicaemia.

I am indebted to Dr. J. Fine for his pathological reports, and to Dr. E. Grahame Jones, under whose care the patient was admitted, for permission to publish the case.

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