incision in the vessel was sutured with arterial-silk atraumatic needles. All the instruments and the wound were washed with sodium citrate.

Post-operatively he was given 15,000 units of heparin through a polythene tube six-hourly. At 2 a.m., five hours after the operation, I was called to see him because of persistent oozing. This was not severe, and not unexpected in view of the spinal anaesthetic. It was noted at this time, however, that he had made a complete recovery from the paralysis and could actively dorsiflex and plantar-flex his foot. Both his lower-limb pulses had returned. Transfusion was considered because of loss of blood during and after operation and because of the necessity for an adequate blood pressure to maintain a good popliteal circulation, but in view of the recent hemiplegia it was thought inadvisable at that stage.

On April 9 much oedema was present from the popliteal fossa downwards, but there was no evidence of aneurysm. Next day an electrocardiogram showed atrial infarction. This no doubt accounted for the source of his emboli. On the 11th his leg suddenly went blue about half an hour after an injection of heparin. As this was thought to be due to haemorrhage with compression of the vessel, 15 ml. of protamine sulphate was given intravenously. It was decided that in order to try to save his limb a blood drip should be set up.

On April 24 the swelling had gone down, but he had complete sensory-nerve loss from the knee downward. His red cells numbered 3,750,000 and the haemoglobin was 68%. The condition of his leg gradually improved. First protopathic sensation and then epicritic sensation returned.

A final examination on July 4 showed some wasting of the calf muscles and a slight equinus deformity due to fibrosis. He was able to walk fairly well with a raised heel and a toe spring.

I am greatly indebted to Mr. J. M. Turner and Mr. H. J. McCurich for their permission to publish this case.

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Acute Pethidine Poisoning

In a search of the literature only one case has been found where pethidine has been taken with suicidal intent. The satisfactory response to treatment also makes the following case interesting.

CASE REPORT

A married woman aged 43 had suffered from tuberculosis of the bladder for 20 years. She had had a left nephrectomy 20 years ago. Because of the pain and frequency, admission to hospital was arranged for July 22, 1950. She had previously refused to come into hospital, but eventually agreed, stipulating a Saturday. Owing to the shortage of beds it was necessary to cancel this carefully arranged admission at the last minute. The same day she was found unconscious at home. She was brought to hospital accompanied by her mother. The mother's account and the patient's appearance indicated that she was suffering from a narcotic poison. The mother had not brought the remaining tablets, so was sent to fetch them, having identified a tablet of phenobarbitone, 1 gr. (65 mg.), as being similar to those in the box.

The patient was found to be deeply comatose, and there was no response to pressure on the supra-orbital nerves nor to any other stimuli. There was extreme respiratory depression, the rate being 16 per minute and very shallow; pulse rate 90 per minute; temperature 97° F. (36.1° C.); blood pressure 100/65; no sweating. The pupils were equal and contracted, with no response to light, and no tendon jerks were present. There were no plantar reflexes, and no other relevant findings.

TREATMENT

It was clear that whatever tablets had been taken they were not aspirin. Phenobarbitone is the commonest agent

in attempted suicide by drugs, and the pupils were contracted. (Opinions vary on the state of the pupils in barbitone poisoning, but from personal observation the pupils are more often contracted than dilated.) The patient's condition called for immediate treatment, and, as her mother had not returned with the tablets for identification, treatment had to be empirical. She was given picrotoxin, 6 mg. intravenously, with no effect. This was followed two minutes later by amphetamine sulphate, 60 mg. intravenously, with no effect. Three minutes later nikethamide, 6 ml., was given intravenously. In thirty seconds the patient became conscious. Although a little confused she recognized her surroundings, and when questioned immediately replied that she had taken 30 tablets of pethidine (1,500 mg.), as she was "fed up." Her mother then arrived with the box of tablets, which were confirmed as pethidine; 20 of the original 50 remained. The patient was now very emotional and would not co-operate in having her stomach washed out. No further treatment was given, but there was no sign of her relapsing back into coma. At 8.30 p.m. she was sweating and shocked. She had a disturbed night (doubtless due to the amphetamine sulphate), but the next morning she was normal again.

Pethidine is rapidly absorbed, quickly metabolized, and partially excreted unchanged in the urine. Elimination of the ingested pethidine was studied by analyses of urine collections, using the method of Lehman and Aitken (1943), with the following results: 6.7 mg. (49th to 73rd hour after ingestion), 2.8 mg. (81st to 105th hour), 0.6 mg. (105th to 129th hour).

DISCUSSION

The absence of vomiting and sweating in the case reported here is noteworthy. The pupils were small, which is unexpected, as in therapeutic doses pethidine causes mydriasis. Recovery was complete within 24 hours. Way (1946) has pointed out that pethidine may stimulate or depress the central nervous system. With overdoses it is the stimulatory action that is usually seen (for example, tremors, uncoordinated muscular movements, and, experimentally, epileptiform seizures), but in certain individuals (as in this case) the depressant action may predominate.

Cases of acute poisoning by pethidine are few. Experimentally, death results from respiratory failure (Gruber *et al.*, 1941). Polonio (1947) mentions two patients with pethidine addiction who had several crises of respiratory failure which responded well to nikethamide. In two deaths which he observed, necropsies could not be made but "the history and post-mortem appearance of cyanosis and engorged veins alike indicated respiratory failure." The only case found of pethidine being taken with suicidal intent is reported by Cohen (1950).

Nikethamide counteracts the effect of depressants of the central nervous system, but how it does so is obscure. From personal experience it is useful in interrupting pentothal narcosis, the treatment of phenobarbitone poisoning, and acute alcoholism. In this case it was dramatically effective in terminating the coma and respiratory depression, and, although one might have expected a relapse into coma, no further injection was necessary. Perhaps the amphetamine sulphate contributed to the sustained recovery of consciousness. As nikethamide is effective in correcting depression of the central nervous system from such different causes, this suggests that, when confronted with a coma from suspected but unknown poisoning, an empirical trial, as in this case, is well worth while.

I am grateful to Dr. H. L. Milles, under whose care the patient was, and to Mr. H. B. Salt, biochemist, for his help with the urinary estimations.

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