tolerating an oro-tracheal airway during this period, the tube being necessary to prevent a valvular obstruction of his larynx by a mobile epiglottis.

His post-operation pulse rate rose to between 130 and 140 for the subsequent two days. There was no post-anaesthetic vomiting and he made an uninterrupted recovery.

COMMENT

The significance of the fact that this patient had been treated with oral chlorpromazine, 25 mg. three times a day, for the fourteen months previous to his admission was not appreciated at the time, and consequently he was given a normal anaesthetic for his age and weight, with the somewhat anaesthetically distressing results. The post-operation tachycardia and absence of vomiting are a well-known feature of chlorpromazine premedication. This case suggests that care should be exercised in giving normal doses of anaesthetic drugs to patients taking chlorpromazine.

My thanks are due to Dr. H. M. Bird for permission to publish this case.

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Prolonged Coma in Simmonds's Disease

Patients with Simmonds's disease often die in coma, which may be induced by various factors, such as the following.

Hypoglycaemia.—A low fasting blood sugar, sensitivity to insulin, and repeated hypoglycaemic attacks are often features of Simmonds's disease (Sheehan and Summers, 1949, 1952; Alexander, 1953). While in mild cases recovery may be spontaneous or may follow administration of glucose, prolonged hypoglycaemia may result in damage to cerebral tissue which is irreversible and fails to respond to glucose. Although the length of hypoglycaemic coma which prevents a full degree of physical and mental recovery must vary from case to case, Lawrence *et al.* (1942) suggest that "coma of one to three hours is usually associated with complete recovery, while longer periods are dangerous." Convulsions in hypoglycaemia have been mentioned as an important factor in preventing recovery from that state (Grayzel, 1934).

Hypothermia.—The profound lowering of the body's metabolic rate that occurs both in Simmonds's disease and in myxoedema may lead to a reduction of temperature sufficient to promote coma. Sheehan and Summers (1952) have shown that in a patient in coma with a very low temperature rapid recovery took place following immersion in a hot bath.

Low Blood Pressure and Loss of Electrolytes.—The adrenals are commonly atrophied in Simmonds's disease, and in some cases coma may be ushered in by marked hypotension and salt depletion, suggesting Addisonian crisis (Sheehan and Summers, 1949; Wolff, 1950).

The case here described makes it apparent that deep coma of 40 hours' duration, associated with hypoglycaemia and convulsions, can be followed by full mental and physical recovery.

CASE REPORT

A woman of 41, who had been unconscious with convulsions since about 1 a.m., was admitted at 2.30 p.m. on June 22, 1953. She was lying deeply comatose, slightly sweating, and with shallow respirations, in a pool of her own excreta. Her limbs were spastic, her trunk in opisthotonos, while every few minutes she was seized with generalized clonic and tonic convulsions associated with coarse grunting noises. Her temperature was 101.6° F. (38.7° C.), pulse 88, B.P. 120/70. Plantar reflexes were extensor, corneal reflexes absent.

The patient was moderately well nourished, with pendulous breasts; her skin was waxy pale, axillary hair was

absent, and pubic very scanty. Following 50 g. of glucose intravenously, the convulsions became less severe, and on receipt of the blood-sugar report as 32 mg. per 100 ml. half an hour later, a further 100 mg. of glucose was administered intravenously with 100 mg. of cortisone acetate and 1 mega unit of penicillin intramuscularly. An intravenous 5% glucose-saline drip was set up. By 6 p.m. the blood sugar had risen to 86 mg. per 100 ml. and, although severe convulsions had ceased, she remained in deep coma, with extensor plantar reflexes. Cortisone, 100 mg. eight-hourly, was continued.

The next morning she was still deeply unconscious, and was incontinent of urine and faeces. The blood sugar was 140 mg. per 100 ml. During the day there were signs of improvement, and by 5 p.m. she had begun to stir and open her eyes. This was 40 hours after the known onset and 26 hours after the institution of treatment, during which she had been given 400 mg. of cortisone intramuscularly and about 260 g. of glucose intravenously.

From then on, progress was uneventful. On a regime of cortisone, 50 mg. daily by mouth, thyroid, 1 gr. (65 mg.), and methyltestosterone, 15 mg., she asserted she had "never felt better."

The electrocardiogram on admission showed low-voltage and flat T waves, later reverting to normal. The electroencephalogram after recovery showed no convulsive organic abnormality.

The history, obtained subsequently, revealed that the patient had had three children, there having been serious post-partum haemorrhage after the last confinement 16 years previously, and subsequent total amenorrhoea. Simmonds's disease was diagnosed elsewhere in November, 1952, and she had been on testosterone and thyroid since then. She had never had fits, but had had one previous short coma, without convulsions, in January, 1953.

COMMENT

Of the known factors which can induce coma in Simmonds's disease, hypothermia and Addisonian crisis can be excluded in this patient, and the question then arises to what extent the hypoglycaemia was responsible for the prolonged coma. In fact, during the latter 24 hours of coma the blood-sugar levels were within normal range, but, as is known in insulin overdosage, the return of the blood sugar to normal may prevent neither permanent mental damage nor a fatal outcome. We have been unable to find a case recorded in which full recovery occurred after hypoglycaemia so deep and prolonged, and it is possible that in our case other factors were partly responsible for maintaining the comatose state.

While cortisone plays an important part in the maintenance therapy of Simmonds's disease, treatment of the coma with cortisone has not been universally successful. Thus Sheehan and Summers (1952) found that intravenous cortisone produced no appreciable effect in two cases of hypopituitary coma, although it is not clear to what extent hypoglycaemia was a factor in those cases. We feel that administration of cortisone as well as glucose to our patient was largely responsible for the successful outcome. Cortisone has been used to combat hypoglycaemia of various causes, such as pancreatic islet tumour and Addison's disease, and we feel encouraged to use it in prolonged hypoglycaemia of whatever aetiology.

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