of the instability of the chest wall or only because of inadequate ventilation (Case 2), but once it is started the paradoxical movements are abolished, this sign is no longer present, and the chances of early diagnosis are lost.

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Two Cases of Ergotamine Poisoning in Infants

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Ergot poisoning is rare. No death has been reported in the British literature after accidental overdose of an ergot-containing preparation. We report two cases of ergotamine poisoning in infants, one being fatal.

CASE 1

Christopher, aged 14 months, was admitted to hospital on 11 March 1964 at 12.30 p.m., and died 14 hours later. At 11.30 a.m. that morning he had swallowed 12 Cafergot tablets (1 mg. of ergotamine tartrate and 100 mg. of caffeine citrate per tablet), a total dose of 12 mg. of ergotamine tartrate. Vomiting was induced immediately. The child became cold and clammy with laboured breathing.

On admission the child was semicomatose with sinus tachycardia (E.C.G.) 180 per minute, shallow respirations at 50 per minute, and cold, blue extremities. The respiratory rate was 70 per minute, temperature 39.5° C. The child was coughing in spasms, and there were crepitations at the right base and middle lobe. The pupils were pin-point, did not react to light, and the cranial nerves and fundi were normal. He was hypotonic, all reflexes being present but sluggish. The stomach was washed out immediately and penicillin given intramuscularly.

After initial improvement he suddenly had a rigor and vomited blood-stained material, became pale and sweating, with a blood-pressure of 90/40 mm. Hg, and a pulse rate of 140 per minute. Intramuscular mephentermine sulphate 30 mg. was given and an intravenous infusion set up of half normal saline containing 200 mg. hydrocortisone per litre. Large amounts of blood-stained fluid were obtained by gastric suction. The child became comatose, and mechanical intermittent positive-pressure ventilation was started via a cuffed endotracheal tube. The pupils were dilated, and the blood-pressure unrecordable. Triple rhythm and papilloedema appeared. At 2.45 a.m. the child died.

The relevant findings at necropsy were cerebral oedema, haemorrhagic gastritis, submucous haemorrhages at the level of the larynx, and petechiae of the visceral pericardium.

The organs could not be assayed for ergot, but caffeine was present as follows: gastric fluid, 42 mg./100 ml.; urine, 2.2 mg./100 ml.; whole blood, 4.4 mg./100 ml.; and cerebrospinal fluid, 4.7 mg./100 ml. The brain contained 14 mg./100 g. wet weight and the kidney 21 mg. Histological examination showed early anoxic changes in the cerebellum and Ammon's horn. The cause of death was "cerebral oedema due to Cafergot poisoning."

CASE 2

Maria, aged 13 months, was admitted on 4 October 1964. Five hours before admission her mother could not rouse her. A sibling then confessed to having given the child about 50 Bellergal tablets (phenobarbitone 20 mg., ergotamine 0.3 mg., and belladonna 0.1 mg.). The estimated amount of ergotamine taken was 15 mg.

On admission the child was unconscious but reacted sluggishly to painful stimuli. The pupils were dilated and reacting to light. Tendon reflexes were all present, sluggish, and equal on both sides. The respiratory rate was 32 per minute and the chest clinically clear. The pulse rate was 120 per minute, regular, and of fair volume,

and the systolic blood-pressure 50 mm. Hg. There was marked peripheral cyanosis and the hands and feet were cold and puffy. A blood level of more than 5 mg./100 ml. of barbiturate was found. The cerebrospinal fluid was normal. The stomach was washed out immediately. During the two subsequent hours the respiratory rate fell to 20 per minute, the child became more deeply unconscious, and peripheral cyanosis increased.

Peritoneal dialysis was performed with Baxter dialysing fluid (1.5% dextrose). Forty millilitres of 10% mannitol solution was injected in two doses of 20 ml. into the saphenous vein. Two hundred millilitres was dialysed every half-hour. The total amount of fluid infused was about 7.1 litres and the total amount drained about 7.9 litres. The total amount of intravenous fluid was 1.6 litres and the urine output was 542 ml.

Nine hours later the child was fully conscious. After a further nine hours there was marked improvement in peripheral circulation and the dialysis was discontinued. The child made rapid progress and was discharged on 23 October 1964.

COMMENT

The total amount of ergot taken by the two children (12 mg. and 15 mg.) was similar. Treatment of an overdose is generally directed towards restriction of further absorption of the drug and elimination of the drug already absorbed. These measures proved unsuccessful in the first child; as the condition of the second child deteriorated, peritoneal dialysis and forced diuresis were undertaken. Routine adult equipment and technique were used, only the amounts of fluid being modified. The first 200 ml. of the dialysate was analysed in the laboratories of Sandoz Ltd. and contained 17.1 mg. of phenobarbitone and 90 µg. of ergotamine (present as ergotaminine). A substance appeared on the chromatogram which was probably a metabolite of ergotamine. The dialysis therefore helped to remove the poison. Elimination was aided by infusion of a 10% solution of mannitol. The urine was not assayed for ergot.

Case 1 was unusual, with severe dyspnoea, pyrexia, haemorrhagic gastritis, rapid onset of cerebral oedema, and death. Case 2, though complicated by a high blood level of barbiturate, recovered after peritoneal dialysis and mannitol diuresis. Both had cold, blue extremities.

Preparations containing ergot are widely used in the treatment of migraine. Poisoning produces cerebral oedema and peripheral circulatory failure. It is important, therefore, to instruct the public about the dangers to children. Peritoneal dialysis and mannitol diuresis are effective treatment.

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