of the latter to the administration of experimental drugs designed to lower pulmonary vascular resistance. There may also be a temptation to increase the peak inspiratory pressures in such a situation. In the case described, any of the above measures might have had dire consequences.

We have seen hypoxia relieved by stopping IPPV on previous occasions, but have been unable to document the event. We have also seen patients with bronchopulmonary dysplasia whose early neonatal course was similar to that described above.

The indications for IPPV were obscure and this case serves as a reminder that IPPV should not be undertaken without good reason. It also emphasises the importance of proper assessment of a patient after transfer from another hospital.

References

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Eucalyptus oil poisoning

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SUMMARY Accidental ingestion of eucalyptus oil by a 3-year-old boy caused profound central nervous system depression within 30 minutes, but he recovered rapidly after gastric lavage. The extreme toxicity of eucalyptus oil is emphasised.

Eucalyptus oil is a traditional remedy for a variety of common ailments, particularly of the respiratory tract. It is cheap, freely available, and found in many households. However, its extreme toxicity is not generally appreciated and reports of poisonings are rare. With this in mind we report a recent ingestion of eucalyptus oil that was nearly fatal.

Case report

A 3-year-old Muslim boy was seen within 30 minutes of ingesting about 10 ml eucalyptus oil. On examination he was deeply comatose and his breath smelt strongly of eucalyptus. The pupils were constricted, muscle tone was markedly reduced, and his tendon reflexes could not be elicited. His respirations were shallow and irregular at a rate of 10/min. The pulse rate was 70 beats/min and the blood pressure 75/40 mmHg. Biochemical investigations on admission included a serum urea of 6·3 mmol/l (38 mg/100 ml), with normal electrolytes. Initial treatment included the insertion of a cuffed endotracheal tube (which produced no gag reflex), and gastric lavage with sodium bicarbonate solution. Sodium sulphate 100 ml was left in the stomach as a cathartic.

By 2 hours after admission his pulse, blood pressure, and respiration rate had gradually returned to normal. After 5 hours consciousness had gradually been regained, and by 24 hours physical examination was normal apart from a faint smell of eucalyptus on the breath. Urinary output remained satisfactory throughout. He was discharged home 48 hours after admission.
Factor X deficiency in the neonatal period

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SUMMARY An infant with a severe deficiency of factor X presented in the neonatal period with uncontrollable bleeding from heel prick sites, spontaneous bruising, and haematoma. The deficiency was controlled by infusions of dried human factors II, IX, and X concentrate; the half-life of the infused factor X material is only 18 hours. Despite prophylactic weekly infusions of factor X concentrate, the child developed a fatal intracerebral haemorrhage when only 4 months old. Coagulation studies on both parents and the elder sister showed no obvious coagulation abnormality.

Factor X deficiency is a rare bleeding disorder inherited as an autosomal incompletely recessive trait. Usually the deficient state is clinically relatively slight, and for effective haemostasis a factor X level >10% is thought to be adequate.1

Case report

An Indian girl was born after an uneventful pregnancy which had progressed to term. She was clinically well and delivery had been normal. A prophylactic intramuscular injection of 1 mg vitamin K1 was given one hour after birth into the