hours. No bronchospasm occurred. Skin tests[1] showed a definite reaction to CT 1341 and propanidid (1/10 with saline) but not to saline alone.

Case 4.—A healthy 15-year-old girl had a fractured tibia manipulated under general anaesthesia using propanidid 500 mg intravenously and maintained on nitrous oxide, oxygen, and halothane. Thirteen days later the procedure was repeated, but CT 1341 5 ml was used for induction, after which the patient started coughing and ventilation was inadequate. Despite intubation using suxamethonium 100 mg inflation was difficult owing to bronchospasm. Facial oedema, tachycardia, and hypotension (systolic blood pressure 50 mm Hg) developed. Hydrocortisone 200 mg plus another 500 mg and aminophylline 250 mg were given intravenously and a rapid intravenous infusion was begun. The bronchospasm subsided rapidly and blood pressure became normal over the next hour, during which a petechial rash developed over the neck and shoulders. This and the facial oedema disappeared after three days. No skin tests were performed.

Discussion

In August 1974 the Committee on Safety of Medicines had received 62 reports on CT 1341 Althesin, 21 about allergic reactions. Dundee[2] reported 13 such reactions with thiopentone, and 23 with propanidid. The incidence of CT 1341 sensitivity was estimated as less than 1 in 25 000. Tammisto *et al.*[3] suggested that hypersensitivity might be due to Cremophor EL; such a response had been shown in dogs.[4] Hypotensive reactions to Cremophor EL have been reported in humans[4] but not with clinical doses. Histamine release has been shown in response to CT 1341 and propanidid (both contain Cremophor EL) but not to Cremophor EL alone.[5] CT 1341 was withdrawn from routine use at this hospital.

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Perforation of Small Bowel Due to Slow Release Potassium Chloride (Slow-K)

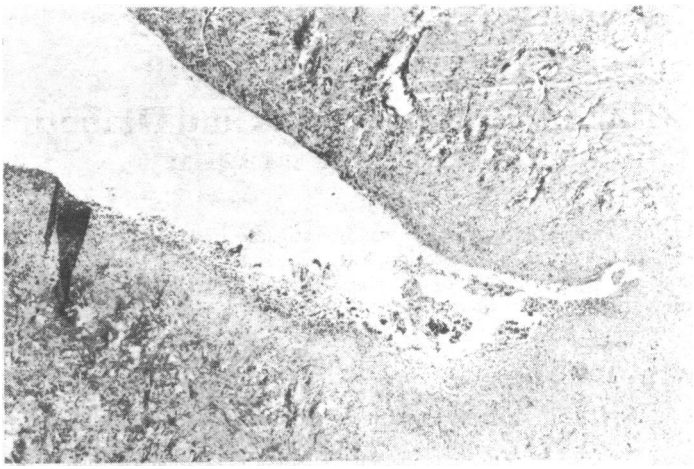
Perforation of the small bowel is a recognized complication of enteric-coated potassium chloride preparations. Slow release preparations of potassium chloride were thought to be free of this risk, but we report here a case of perforation due to Slow-K.

Case Report

A 57-year-old man with a long history of hypertension and multiple strokes was admitted from a long-stay hospital as a surgical emergency. He had the symptoms and signs of a perforated abdominal viscus. In the six months before admission he had received two tablets (2 × 8 mmol) of Slow-K daily, bendrofluazide (Aprinox) 5 mg each morning, and nitrazepam (Mogadon) 5 mg each night. He had been fed a low-volume, high-calorie liquid diet for some months before his transfer as he was otherwise difficult to feed.

At laparotomy a fibrous stricture was found about half way along the small bowel. This was about 0·5 cm long and had narrowed the lumen to about 0·3 cm. A perforation was present at this point. The lesion was resected and end-to-end anastomosis performed. After recovery his gastrointestinal transit time was estimated twice using oral carmine dye and was eight days, mouth to anus.

Histological sections showed superficial acute mucosal ulceration, one area showing acute deep ulceration and perforation (see fig.). There was underlying destruction of the muscularis mucosa in the ulcerated area and acute inflammatory infiltration through the muscularis propria. The picture



Area of small bowel showing acute deep mucosal ulceration and perforation.

was consistent with a perforated, and almost certainly iatrogenic, ulcer with no evidence of malignancy.

Discussion

The thiazide diuretics are potent and safe but need potassium supplementation. Supplements were available as enteric-coated tablets of potassium chloride, either separately or compounded with the diuretic itself. The enteric coating was necessary because potassium chloride irritates the stomach. Experience showed that it also irritated the small bowel when the enteric coating breaks down and releases the potassium chloride. By 1965 over 300 cases of small bowel ulceration and perforation had been reported.[1] The important factor in damage to the small bowel is the high local concentration of potassium chloride, which causes oedema, haemorrhage, erosion, and cicatrizing stenosis in turn; the lesion is essentially a haemorrhagic infarct with venous thrombosis.[1]

Other potassium compounds—for example, bicarbonate—while safer, are metabolically unacceptable, the chloride ion being essential.[2] Slow-release tablets consisting of potassium chloride embedded in a wax matrix from which it slowly dissolves became available, the rationale being that these would give a slow and sustained release over a length of small bowel, thereby preventing a high local concentration of potassium chloride. Slow-K in particular was widely recommended as the drug of choice, as it was supposed to be free of the risk of haemorrhage or ulceration.[3] In all published cases of ulceration due to Slow-K there has been local stasis in the oesophagus due to cardiomegaly.[4]

Our patient, whose drug treatment was accurately known (the other drugs he was receiving were blameless[5]), had delayed intestinal transit, which abolished the "protective" effect of slow-release potassium. The stricture he developed led to further stasis and then perforation.

Patients likely to have delayed intestinal transit—the elderly, immobile, or those taking a low-volume diet—should be given any necessary potassium supplementation in a well-diluted liquid form with or after food.

We thank Mr. M. Golby for allowing us to describe a patient under his care, and Drs. R. A. Caldwell, and E. Seveides for the histological analysis.

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