—brain levels of HCP varied between 0.442 and 1.204 nmol/g (0.18 and 0.49 µg/g).⁴ So myelinopathy in case 2 was probably attributable to HCP, despite the absence of clinical signs of toxicity. The trace level in case 1 suggests that myelinopathy was unrelated to HCP, for other causes of infantile spongiiform myelinopathy are known, though none has been identified in small premature infants. Nevertheless, case 1 may have lived long enough (24 days) for HCP to have been cleared from the brain. In rats HCP myelinopathy takes months to resolve, whereas the half-life of a dose of HCP is 19 hours.

Until now, the only direct evidence that HCP may cause myelinopathy in premature babies has been the correlation between spongy change and the extent of exposure to HCP.⁵ The amount of the compound recovered from our second case indicates that myelinopathy may result from the normal clinical use of a 3% solution of HCP on very immature infants.

¹ Lockhart, J. D., *Journal of Clinical Pharmacology*, 1973, 13, 445.

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³ Browning, R. S., Grego, J., and Warrington, H. P., *Journal of Pharmaceutical Sciences*, 1968, 57, 2165.

⁴ Kimbrough, R. D., *Pediatrics*, 1973, 51, 391.

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Slow Release Potassium Chloride Treatment

Preventing potassium depletion due to diuretic treatment is important. Kassirer *et al.* emphasized that potassium supplements should be as the chloride, as other salts tend to intensify any alkalosis caused by chloride loss and so perpetuate the loss of potassium.¹ After the recognition that potassium chloride tablets may cause ulceration and stenosis of the small intestine² Ciba claimed to have virtually overcome this problem by the introduction of Slow-K. Unfortunately, this has not removed the risk of life-threatening or even fatal adverse effects such as hyperkalaemia,³ oesophageal ulceration, and stricture. Here we draw attention to the last complication and suggest that Slow-K is sometimes contraindicated.

Case Report

A 42-year-old housewife with a history of rheumatic fever was first seen at the outpatient clinic in 1963 when aged 33. She had atrial fibrillation and signs of mitral and aortic incompetence. Cardiac screening showed enlargement of the left atrium. Digoxin treatment was begun and she was not seen again until 1971, when she was admitted to hospital in congestive cardiac failure. After being given frusemide and Slow-K 1.2 g, thrice daily, her failure resolved and cardiac catheterization was carried out in January 1972. The findings were consistent with aortic incompetence, mitral stenosis and incompetence, pulmonary hypertension, and tricuspid incompetence. In July she underwent tricuspid valve annuloplasty and mitral valve replacement with an aortic homograft. The postoperative course was satisfactory and she was discharged from hospital in August. She had continued treatment with digoxin, a diuretic, and Slow-K.

A few days after returning home she had difficulty in swallowing and this steadily led to considerable dysphagia. Discontinuation of digoxin did not bring relief and regurgitation of fluid and vomiting necessitated her re-admission to hospital. Barium swallow examination showed almost complete hold-up of barium just below the carina. The oesophagus was slightly dilated above this with a rounded lower margin changing to a very narrow track. Oesophagoscopy in late September 1972 confirmed the stricture in the mid-oesophagus and biopsy showed no evidence of malignancy. Successful bouginage was performed one week later, when a further biopsy again excluded malignant change. The patient was taught to pass bougies and subsequently she was able to eat solid food again.

Slow-K tablet lodged in mid-oesophageal region demonstrated by barium swallow.

Discussion

Dysphagia occurs only rarely in mitral valve disease despite the presence of even considerable enlargement of the left atrium. Chesshyre and Braimbridge⁴ described a patient with this complication after mitral Starr valve replacement, showing that the operation had resulted in a movement of the oesophagus to the left of its normal position. They concluded that this, in association with left atrial enlargement, caused sufficient compression of the oesophagus to hold up Slow-K and food producing irritation and stenosis. Pemberton reported oesophageal obstruction developing 10 days after mitral valve replacement, and oesophagoscopy showed mucosal ulceration of the posterior wall of the lower third of the oesophagus.⁵ This area was filled with white powder considered to be potassium chloride as the patient was taking Slow-K. We know of a similar case in a patient who complained of persistent pain on swallowing two weeks after operation for complete correction of Fallot's tetralogy. Barium swallow showed a Slow-K tablet lodged in the mid-oesophageal region (see figure).

The reports and the two cases, with quite different cardiac lesions indicate that Slow-K may be held up in the oesophagus with resultant dysphagia or even ulceration and stricture formation. Hence there is sufficient evidence to contraindicate the use of slow-release potassium chloride after cardiac surgery. It may also be advisable to avoid this preparation when cardiac or other disease, requires treatment involving a period of recumbency.

¹ Kassirer, J. P., et al., *American Journal of Medicine*, 1965, 38, 172.

² Baker, D. R., et al., *Journal of the American Medical Association*, 1964, 190, 586.

³ Lawson, D. H., *Quarterly Journal of Medicine, New Series XIII*, 1974, 171, 433.

⁴ Chesshyre, H. M., and Braimbridge, M. V., *British Heart Journal*, 1971, 33, 798.

⁵ Pemberton, J., *British Heart Journal*, 1970, 32, 267.

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