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CORRESPONDENCE

Correspondents are urged to write briefly so that readers may be offered as wide a selection of letters as possible. So many are being received that the omission of some is inevitable. Letters should be signed personally by all their authors.

Salt overdosage

SIR,—Having very recently published an account of two fatalities due to saline emetics,[1] we read Dr R C M McGouran's report of a further case with partial recovery of brain damage due to hypernatraemia with interest (15 November, p 386). Other examples of recovery have been recorded by Capper[2] and by Schatz.[3]

Now that this serious complication is proved, the administration of salt emetics should always be followed by early and repeated estimation of the plasma electrolytes so that appropriate treatment of hypernatremia may be instituted in time to avert brain damage. First-aid manuals and medical textbooks should reconsider the advisability of recommending salt emesis without warning about the dangers of overdosage.[4] We suspect that in the past neurological deterioration and death have mistakenly been attributed to the ingested poison for which the salt was administered when hypernatremia was the true cause.

The close resemblance between some hypernatremic brains and anoxic-ischaemic brain damage has doubtless contributed to failure to recognise this important syndrome at necropsy. From our experience we would not subscribe fully to Dr McGouran's rather mechanistic theory of the pathology of the brain damage. Bleeding is not an invariable feature and the changes are more likely to be explicable on a molecular basis than attributable solely to simple shearing stresses upon the vessels.

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1 Goodbody, R A, Middleton, J E, and Gamlen, T R, *Medicine, Science, and the Law*, 1975, **15**, 261.
2 Capper, J, *St Bartholomew's Hospital Journal*, 1975, **79**, 365.
3 Schatz, W J, *Medical Record*, 1937, **145**, 487.
4 *Black's Medical Dictionary*, 29th edn, p 309. London, Black, 1971.

SIR,—In his case report (15 November, p 386) of the management of an adult with salt overdosage Dr R C M McGouran states that his patient was treated with "frusemide and dextrose given as fast as possible by intravenous infusion." In recent years we have learnt much about the safest way to treat hypernatremia in paediatric practise. Several studies have shown that slow rehydration with a polyionic solution (usually dextrose in 0·18% saline) is associated with a far lower incidence of convulsions during treatment, irrespective of the severity of the hypernatremia.[1 2] I cannot see how the situation can be so different in an adult and suggest that the generalised convulsions which began during treatment and were uncontrollable in Dr McGouran's patient may have resulted from cerebral oedema due to too rapid reduction in serum sodium.

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1 Banister, A, Matin-Siddiqi, S A, and Hatcher, G W, *Archives of Disease in Childhood*, 1975, **50**, 179.
2 Bruck, E, Abal, G, and Aceto, T, *American Journal of Diseases of Children*, 1968, **115**, 281.

Biopsy of nasopharynx as staging procedure in Hodgkin's disease

SIR,—The remarkable progress that has been achieved during the past decade in the treatment of Hodgkin's disease is partly due to the introduction of new methods for charting the extent of the disease before the beginning of treatment.

Most patients with Hodgkin's disease present with cervical lymphadenopathy. The nodes in the neck drain, among other structures, the nasopharynx. But the occurrence of nasopharyngeal involvement in Hodgkin's disease has been reported only in individual cases.

During the past six years 76 new patients with Hodgkin's disease underwent a detailed prospective ear, nose, and throat examination, which in 45 cases included a biopsy of the nasopharyngeal mucosa. The main reason for not performing a biopsy in 31 was a normal finding at the ENT examination. Seven out of the 45 biopsied patients proved to have abnormal microscopic findings in the nasopharynx, four having clearcut Hodgkin's disease and three abnormal changes sug-