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Hyponatraemia and death or permanent brain damage in healthy children.

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OBJECTIVE--To determine if hyponatraemia causes permanent brain d in healthy children and, if so, if the disorder is primarily limited to fema occurs in adults. DESIGN--Prospective clinical case study of 16 affecte children and a review of 24,412 consecutive surgical admissions at one medical centre. PATIENTS--16 children (nine male, seven female; age ' 5) years) with generally minor illness were electively hospitalised for pr care. Consultation was obtained for the combination of respiratory arres symptomatic hyponatraemia (serum sodium concentration less than or e 128 mmol/l). MAIN OUTCOME MEASURES--Presence, gender distril and classification of permanent brain damage in children with symptom hyponatraemia in both prospective and retrospective studies. RESULTS retrospective evaluation the incidence of postoperative hyponatraemia a 24,412 patients was 0.34% (83 cases) and mortality of those afflicted w 8.4% (seven deaths). In the prospective population the serum sodium concentration on admission was 138 (SD 2) mmol/l. From three to 120 inpatient hours after hypotonic fluid administration patients developed progressive lethargy, headache, nausea, and emesis with an explosive or respiratory arrest. At the time serum sodium concentration was 115 (7) 1 and arterial oxygen tension 6 (1.5) kPa. The hyponatraemia was primari caused by extrarenal loss of electrolytes with replacement by hypotonic All 16 patients had cerebral oedema detected at either radiological or postmortem examination. All 15 patients not treated for their hyponatra in a timely manner either died or were permanently incapacitated by bra damage. The only patient treated in a timely manner was alive but ment retarded. CONCLUSIONS--Symptomatic hyponatraemia can result in h morbidity in children of both genders, which is due in large part to inad brain adaptation and lack of timely treatment.

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