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## Papers

# Childhood deaths from acute appendicitis in England and Wales 1963-97: observational population based study

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The number of children under 15 years of age certified as dying of acute appendicitis each year in England and Wales decreased from the 1930s to the 1980s. Factors contributing to death have been analysed in audits in 1963-7 and 1980-4. [1 2] We undertook an audit of children dying in 1993-7, compared these results with those of previous audits, examined hospital administrative statistics,[3] and identified possible reasons for the fall in the number of deaths.

## Methods and results



We used similar methods to previous audits to examine hospital and coroner's reports of all 12 childhood deaths in 1993-7. [1 2] The median age of the children who died was 10.6 (range 3.0-14.2) years. Six children died at home or on arrival at hospital, and six died postoperatively in hospital from multiorgan failure (four in district general hospitals, two in children's hospitals). Median duration of symptoms before hospital admission or death was 3 (2-6) days. Difficulty or delay in diagnosis was the main factor contributing to death in six cases.

The number of deaths from acute appendicitis in hospital fell from an annual average of 36.2 in 1963-7 to 1.8 in 1993-7. The case fatality rate in hospital has fallen from 1.06 to 0.16 per 1000 discharges with acute appendicitis, a fall of 85% (table). If children who were moribund on arrival at hospital are excluded the decrease is 97%. If the 1963-7 age specific death rates had applied to children admitted with acute appendicitis in 1993-7 the expected number of deaths would have been 50 rather than the nine recorded. Thus 41 of 172 (24%) fewer deaths in 1993-97 may be attributed to a fall in the hospital case fatality rate, the greatest improvement being in the youngest age group.

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| **View this table:** [in this window] [in a new window] | Death rates per 1000 discharges (actual number of deaths) in hospital from acute appendicitis in England and Wales |

The number of children with a discharge diagnosis of "acute appendicitis" fell from an annual average of 34 000 in 1963-7 to 11 500 in 1993-7; the discharge rate fell from 3117 to 1153 per million children (63% fall). The discharge rate for children with "operations on the appendix" decreased by 60%, and the rate for children with a discharge diagnosis of "abdominal pain" increased by 88%. The population of children aged 0-14 years decreased by 8.5%.

## Comment

Top
Methods and results
Comment
References

Three quarters of the dramatic fall in the number of children dying of acute appendicitis during the past 30 years is due to a decrease in the incidence of appendicitis in a slightly smaller child population. A quarter of the fall is due to a marked decline in the hospital case fatality rate, probably reflecting improved medical care. In the 1960s the main factors contributing to death were inadequate fluid replacement, anaesthetic complications, hyperpyrexia, convulsions, sepsis, and difficulty or delay in diagnosis. Difficulty or delay in diagnosis is now the main factor contributing to the small number of deaths.

The recorded decline in discharges for acute appendicitis has been noted in other studies, and the parallel rise in discharges for abdominal pain suggests that some of this fall is due to improved diagnosis. The combined impact of computer aided diagnosis, clinical scoring systems, ultrasonography, laparoscopy, and cross sectional imaging techniques remains uncertain. Clinical assessment and "active observation" still have a vital role in the diagnosis of acute appendicitis.[4]

The reason for the declining incidence of acute appendicitis is unknown. It has been attributed to improved hygiene and changing patterns of childhood infection resulting in less lymphoid hyperplasia in the gut.[5] It is intriguing that such a dramatic reduction in the number of cases of a serious and worrying illness has come about for reasons that are not clear and without any national strategy.

## Acknowledgements

We thank the consultants and coroners who kindly responded to our requests for information and Professor Richard Thomson for helpful advice.

Contributors: GP conceived the study. MDS undertook the clinical audit, and GP did the epidemiological studies. Both authors contributed to interpreting the data and writing the paper, and both are the guarantors.

## Footnotes

Funding: Charges for obtaining death certificates from the Office for National Statistics were paid with funds donated for paediatric surgical research.

Competing interests: None declared.

## References


- Top
- Methods and results
- Comment
- References



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**Decrease in incidence of Appendicitis**

Gordon Caruana-Dingli, Surgeon , *St Luke's University Teaching Hospital, Malta.*

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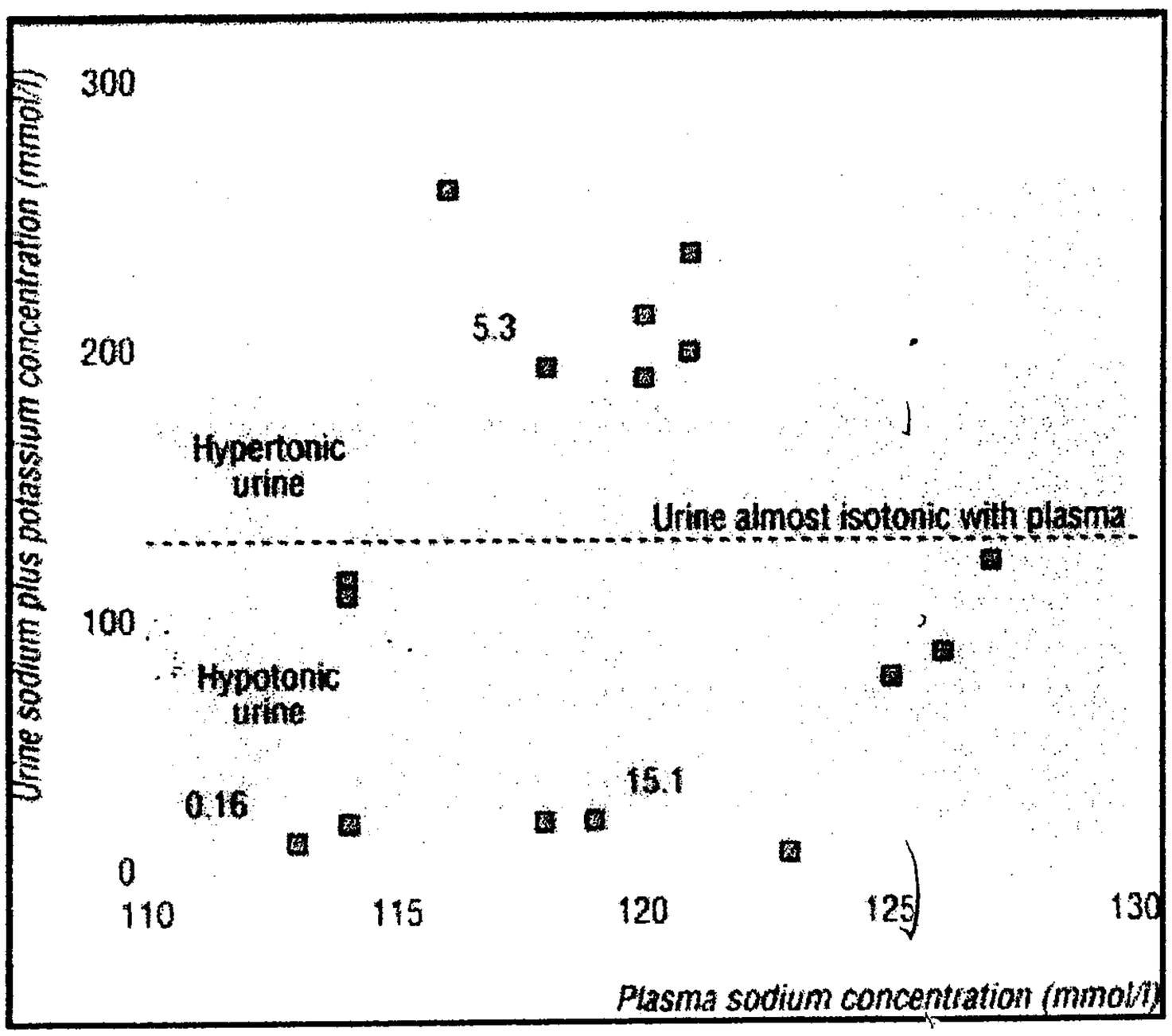


**Fig. 1.** Concentration of sodium and potassium in urine at nadir observed for plasma sodium concentration in 17 patients (numbers represent rate of urine flow)

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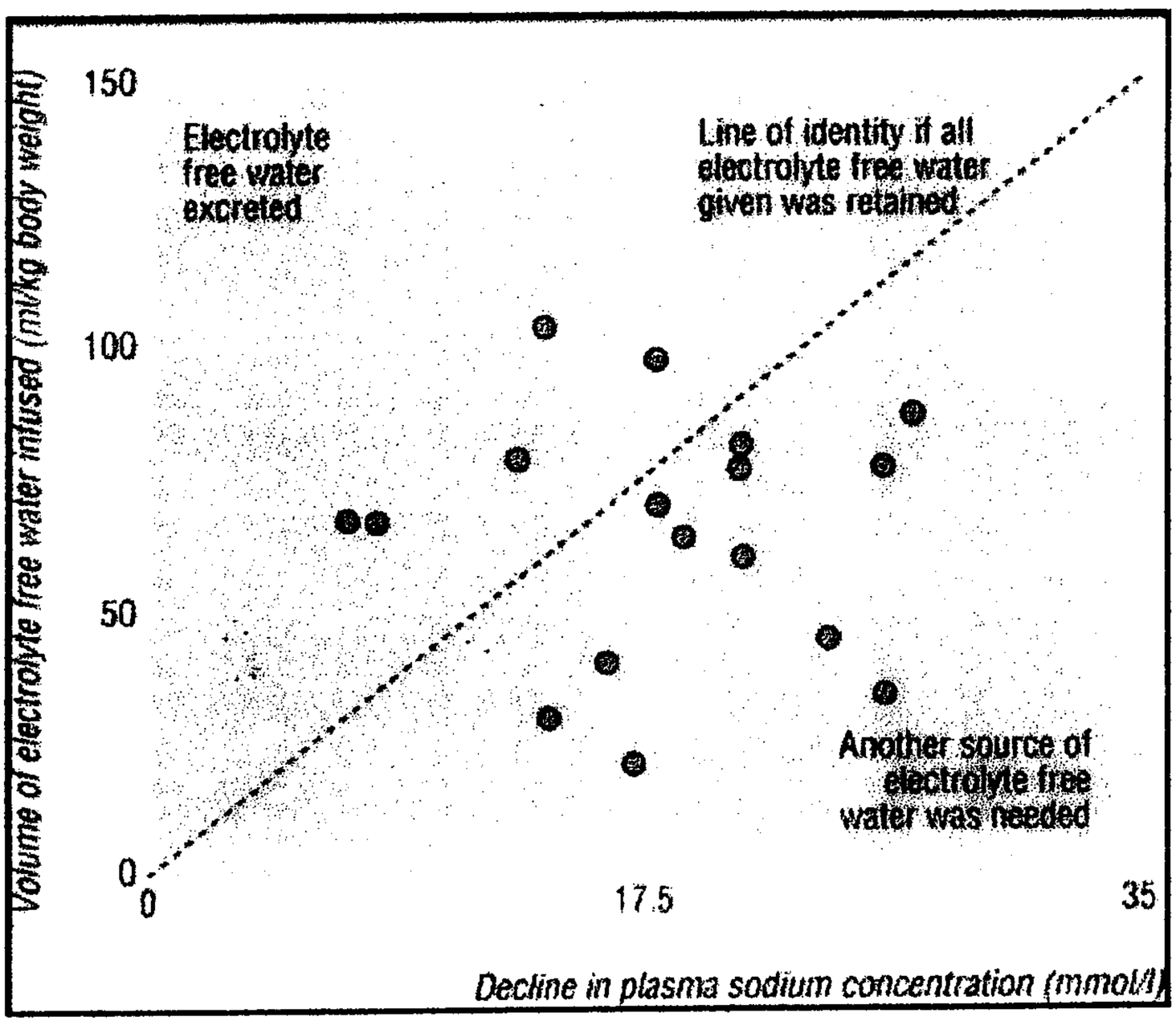


**Fig. 2.** Comparison of decline in plasma sodium concentration with amount of electrolyte free water given in 17 patients. Difference in sodium concentration was between initial value and that at its nadir

[Return to article]

Acute hyponatraemia leading to brain damage is largely preventable but only if we learn to understand that normal physiology is frequently perturbed perioperatively. We need to recognise the prodromal symptoms and be aware that any postoperative patient is potentially at risk. Finally, the use of hypotonic saline should be prohibited perioperatively and reserved only for situations in which the administration of electrolyte free water is genuinely needed.

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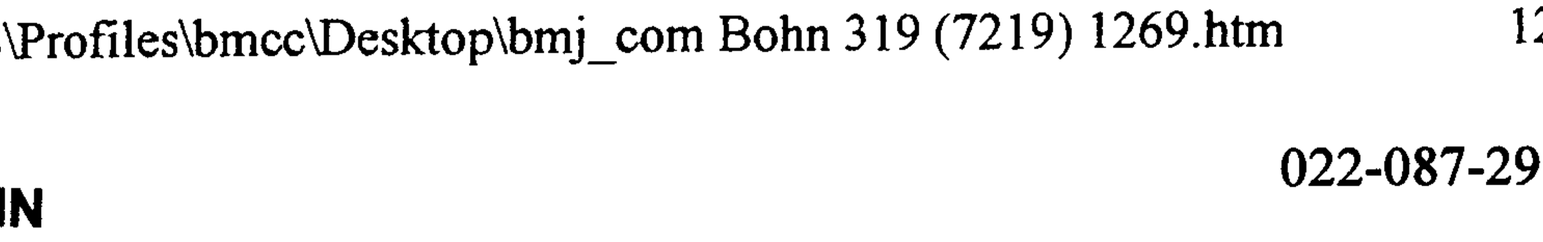

*BMJ* 1999;319:1269 ( 6 November )



## Letters

# Children are another group at risk of hyponatraemia perioperatively

EDITOR—Our poor understanding and practice of giving fluid and electrolytes perioperatively spans all age groups. The editorial by Lane and Allen highlighted elderly postmenopausal women as being particularly at risk of hyponatraemia postoperatively,[1] as has been emphasised in several case series, [2 3] and the electronic responses to the editorial also concentrated on this age group (www.bmj.com/cgi/content/full/318/7195/1363#responses).

The use of hypotonic saline is standard practice for the paediatric age group. This practice is based on calculations of normal physiological requirements for salt and water homoeostasis. These principles were laid down over 40 years ago and have never been challenged, although every year otherwise healthy children suffer brain damage associated with acute perioperative hyponatraemia.[4] Isotonic solutions are not used in children because it is assumed that their kidneys cannot handle a salt load.

The basic flaw that prevents us understanding the cause of these catastrophes is our assumption that normal physiological rules apply. They don't, and we are frequently undone by the subtleties of secretion of antidiuretic hormone. Practically everything that happens to a patient in the perioperative period is guaranteed to turn on secretion of antidiuretic hormone and inhibit the excretion of electrolyte free water when the situation demands.

Even when an isotonic or near isotonic solution is given surgical patients can become hyponatraemic. In a study by Steele et al 21 out of 22 patients who received only these types of fluid showed a fall in serum sodium concentration from a mean of 140 to 136 mmol/l.[5] The explanation became evident from the urine sodium measurements, which showed that most patients were excreting a hypertonic urine (sodium concentration 150 mmol/l) and were in positive water balance. This implied that antidiuretic hormone was acting, a combination of events referred to by the authors as "desalination."

In a case series of 23 children with acute hyponatraemia presenting with convulsions that I have collected seven of them were excreting a hypertonic urine at presentation despite having a serum sodium concentration of <125 mmol/l (data submitted for publication). Much can be learnt from measuring urine sodium concentrations and calculating not only fluid but tonicity balances (sodium plus potassium).

Acute hyponatraemia leading to brain damage is largely preventable but only if we learn to understand that normal physiology is frequently perturbed perioperatively. We need to recognise the prodromal symptoms and be aware that any postoperative patient is potentially at risk. Finally, the use of hypotonic saline should be prohibited perioperatively and reserved only for situations in which the administration of electrolyte free water is genuinely needed.

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