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Correspondence

Blood glucose concentration profile after 10 mg dexamethasone in non-diabetic and type 2 diabetic patients

Editor—With interest, we have read the article by Hans and colleagues[1] entitled 'Blood glucose concentration profile after 10 mg dexamethasone in non-diabetic and type 2 diabetic patients undergoing abdominal surgery'. In recent years, an increasing amount of attention has been directed at perioperative and critical illness hyperglycaemia, as hyperglycaemia has been associated with higher morbidity and mortality rates.[2 3] Several risk factors have been identified that increase the risk of hyperglycaemia in the perioperative period, including abnormal glucose homeostasis, obesity, duration of fasting and type of surgery.[4] In addition, administration of dexamethasone may affect normal glucose homeostasis resulting in hyperglycaemia.

To date the effect of the low dose of dexamethasone that is commonly used for PONV prophylaxis on blood glucose concentration is not known. Therefore, the conclusion of this study that 10 mg dexamethasone may increase blood glucose concentration in non-diabetic and type 2 diabetic patients is potentially very relevant.

However, we feel that Hans' conclusion is not entirely justified by the data that are presented. Besides the possible effect of dexamethasone, several other factors may have contributed to the increase in blood glucose concentration, of which surgical stress is probably the most important. That blood glucose concentration has previously been shown to increase significantly over the course of surgery in patients who did not receive dexamethasone may by itself explain the results, whether patients had diabetes or not.[5 6] Therefore, the lack of a control group of patients that did not receive dexamethasone or preferably a control group receiving a placebo precludes any conclusion about the effect of dexamethasone on blood glucose concentration.

Hence, the hyperglycaemia found could have been caused by other factors than the low dose dexamethasone. This is supported by others showing that in normal subjects, high dose dexamethasone indeed increased insulin resistance, but did not increase blood glucose concentration.[7] Once more, this illustrates the importance of including a control group in the design of the study. Without such a control group, interpretation of the data of this study remains difficult.

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Editor—We thank Drs Kooij and Kal for their interest and comments regarding our paper on blood glucose concentration profile after dexamethasone administration in diabetic and non-diabetic patients undergoing abdominal surgery. They first noted that several risk factors including abnormal glucose homeostasis, obesity, duration of fasting and type of surgery, may be responsible for hyperglycaemia in the perioperative period. We completely agree with that comment for two reasons: (i) as mentioned in their letter, this particular point has already been highlighted and published; and (ii) we demonstrated and confirmed in our study that severe obesity is a determinant factor of glucose increase, regardless of the metabolic status of patients. Their second point is that they consider 10 mg of dexamethasone as a low dose. However, there is still some controversy regarding the optimal dose of dexamethasone to be used for PONV prophylaxis and a 10 mg injection is probably in the upper part of the prophylactic range. They also noted that surgical stress is another very important contributing factor to the increase in blood glucose concentration. Once again, we agree with that point which was clearly mentioned and discussed in our paper. In our study, we addressed this issue by looking at the CRP level measured in all patients the day after surgery and failed to find any difference between groups. However, given the disparity of surgery in our patients, we think that surgical stress cannot be considered as a minor risk factor and agree that it should be evaluated specifically and in a different way in a further study.

Finally, the last and most important comment questions the role of dexamethasone as the primary cause of hyperglycaemia and stresses the importance of including a control group in the design of the study. As mentioned in the paper, our study was not designed to investigate the effect of dexamethasone on blood glucose level. This question has already been answered in the literature, even if dexamethasone is certainly not the only factor that should be incriminated. We did not conclude that dexamethasone was responsible for hyperglycaemia. We actually demonstrated that after dexamethasone administration, blood glucose profile significantly differs in non-diabetic and type 2 diabetic patients undergoing elective abdominal surgery early in the morning. In addition, the main message the reader should keep in mind is that in the course of dexamethasone injection, severe obesity and poor-controlled diabetes rather than diabetes *per se* are determinant factors of hyperglycaemia and should incite to a close monitoring of blood glucose level.

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Hyponatraemia after postoperative fluid management in children

Editor—Most postoperative paediatric surgical patients will be nil by mouth from a few hours to at least a few days or even weeks after the operation and the importance of postoperative fluid and electrolyte balance cannot be stressed strongly enough. We present an audit project for which the local audit committee gave approval. The aims of this study were: (i) to check what types of fluids are being used after operation; (ii) to determine whether electrolyte levels are being checked after operation; and (iii) to determine whether the commonly used fluids cause any fluids and electrolytes derangement.

A retrospective study of 104 patients who underwent appendectomy at Royal Manchester Children's Hospital from September 2004 to March 2005 found that 51 patients (49%) had their electrolytes monitored 24 h after operation, 23 patients (22%) had their electrolytes monitored 3 days after operation and the remainder (34) had no record of electrolytes after operation. The postoperative fluids used were: in 91 patients (87%) 0.45% saline and 5% dextrose, 7 patients (6.7%) Hartmann's solution in addition to the above, and 1 patient (0.96%) total parenteral nutrition in addition to the above fluid. Data were incomplete on three patients.

Of the 51 patients who had their electrolytes checked on the first postoperative day, 16 (32%) had hyponatraemia ranging from 127 to 133 mmol litre^{-1}, 7 (14%) of them <130 mmol litre^{-1}; and 32% of the patients who had their electrolytes checked within a day of surgery had hyponatraemia, 14% severe <130 mmol litre^{-1}.

Between 70% and 80% of a child's body is made up of water which is divided into extracellular fluid (ECF) and intracellular (ICF). The ECF is subdivided into interstitial fluid (ISF) and intravascular fluid (IVF). Although osmolarity of these fluids are similar (ranging 290–320 mosmol litre^{-1}), the electrolyte contents are very different. The ECF contains a high concentration of sodium and the ICF has a high concentration of potassium and magnesium. Fluid moves from one component to another depending on various physiological pressure and osmotic gradients. In illness and injury these fluid shifts may be rapid, with significant clinical consequences.

The ECF volume is controlled by manipulation of its major cation: sodium. The sensors are carotid baroreceptors, atrial stretch receptors and juxtaglomerular apparatus. A reduction in ECF volume causes ADH release, release of atrial natriuretic peptide and activation of renin–angiotensin–aldosterone system. Control of osmolarity is by varying water intake and excretion. A rise in ECF osmolarity triggers sensation of thirst and causes release of ADH. Very sick children may not be able to respond to the sensation of thirst and most of them are kept nil by mouth. The secretion and levels of antidiuretic hormone may be raised in sick children, worsening hyponatraemia.

Dr Cunliffe's editorial on this subject contributes an interesting viewpoint, but as far as we can see it is not evidence based.[1] Alder Hey uses a fluid saline 0.18% and dextrose 2.5% which does not have a majority following according to the questionnaire survey[2] in the *British Journal of Anaesthesia*, and we know of no published audit of results of its use. Readers will recognize that this is a recurring issue: a surfeit of opinions without evidence and surveys of what anaesthetists might do or say they do. Our audit stands out as a record of what people did, and not a paper about what they said they could have done.

The Department of Health has also recently renewed an interest in hyponatraemia occurring after operation in children in hospital. In 2003 there was advice from the Royal College of Anaesthetists[3] warning of problems of four-and-a-fifth saline (dextrose 4% and saline 0.18%), at which point this hospital switched to half normal saline (dextrose 5% and saline 0.45%) which was the fluid used in our review. The issue is under current review and renewed consultation in 2006. The use of four-and-a fifth saline was described as 'dangerous and harmful and should never be used'[4] in a GMC case. However, we have found one of the recommended solutions for its replacement is also associated with significant hyponatraemia. Doctors expect that if they are told not to use one solution but to use others by outside agencies, there is an evidence base that it is safer. At the present time, this is not the case, nonetheless the authors note that tendentious GMC cases on such things as hyponatraemia will become much easier to prove against doctors when the standard of proof goes down to the civil level of balance of probabilities.