Perioperative fluid therapy

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Intravenous fluid therapy is an important aspect of perioperative care, but doctors often prescribe fluid with limited knowledge of its benefits and risks. This article provides an evidence based summary of current best practice in the prescription of fluid for patients undergoing major non-cardiac surgery.

Patient outcomes after major non-cardiac surgery can be improved considerably through more effective perioperative care.1 Factors such as advancing age, comorbidity, and complex surgical procedures can result in postoperative morbidity and mortality rates similar to those found with common acute medical emergencies.2,3 Patients who survive postoperative complications experience functional limitation and reduced long term survival.1,3 Doctors often prescribe intravenous fluid with limited knowledge of the benefits and risks of this treatment. Doctors in training commonly express frustration at the lack of clear guidance on the optimal approach to fluid therapy. The debate that followed recent UK guidelines aimed at standardising best practice highlights the uncertainty in this area, even among experienced practitioners.4 5

We review the evidence from clinical studies, systematic reviews, and practice guidelines to provide a summary of current best practice in the prescription of fluid for patients undergoing major non-cardiac surgery.

What are the principles behind fluid therapy?

In health, 60% of total body mass consists of water. Most water resides within the intracellular compartment, separated from extracellular water, which comprises the interstitial and plasma volumes. The neuroendocrine response to surgery results in retention of sodium and water with a reduction in maintenance requirements. Conversely, absolute hypovolaemia (blood loss) and relative hypovolaemia (such as epidural or inflammation mediated vasodilatation) commonly result in a fluid deficit. For most patients fluid losses are replaced during surgery and oral intake of fluid is rapidly resumed after surgery. However, for some procedures (such as gastrointestinal surgery, proximal femoral fracture repair), the preoperative deficit and losses during surgery vary widely and may not be adequately replaced.

Inadequate fluid replacement leads to reduced cardiac output and oxygen delivery to injured tissues, which is associated with an excess of postoperative complications. Excessive fluid administration may also have adverse effects, including acidosis, coagulation defects, and oedema of both lungs and peripheral tissues (fig 1).6 It is also worth noting that postoperative adverse events may be attributed to fluid prescribing when associated factors are to blame. The tissue injury of surgery results in a systemic inflammatory response associated with both tissue oedema and hypovolaemia. Negative fluid balance after surgery is associated with reduced mortality (odds ratio 0.50 (95% confidence interval 0.28 to 0.89)),7 although this may reflect the degree of inflammatory response as well as excessive fluid administration.8 Fluid restriction and diuresis may decrease oedema in patients with poor ventricular function but also increase the incidence of acute kidney injury.

How should we select the dose of intravenous fluid?

Perioperative maintenance fluid

The normal daily dietary requirements for water and electrolytes are listed in table 1. However, retention of sodium and water after surgery reduces their requirements. Additional amounts should be given only to correct deficit or continuing losses. Monitoring should include clinical examination, fluid balance charts, regular weighing, and biochemical analyses (urea, electrolytes, creatinine, bicarbonate).
It is often helpful to calculate the quantity of water, sodium, and potassium prescribed in a given fluid regimen (table 2). The optimal daily dose of water and electrolytes cannot be provided by a single crystalloid solution, and a suitable daily prescription for maintenance fluid will normally include more than one formulation. Many fluid solutions contain large quantities of sodium and chloride, which may result in nausea and vomiting, metabolic acidosis, and impaired renal blood flow. However, although hypotonic solutions are important sources of water, they may cause severe hypernatremia and neurological impairment when used as replacement fluids. Analysis of 38 randomised trials involving 1589 patients undergoing major abdominal aortic surgery failed to identify specific superior fluid regimens. Although crystalloid solutions that contain bicarbonate may limit the deleterious effects of chloride, the clinical and metabolic consequences of limiting chloride-rich fluids remain unclear.

Replacement of perioperative fluid deficit

The most important information required to assess intravascular volume is provided by the clinical scenario; is it likely that the patient is hypovolaemic? Estimates of fluid deficit based on traditional physiological parameters such as heart rate, blood pressure, and central venous pressure are not reliable. A systematic review of 24 studies showed that central venous pressure is a poor measure of fluid deficit (pooled correlation between central venous pressure and change in cardiac output 0.11 (95% confidence interval 0.02 to 0.21)). However, although hypotonic solutions are important sources of water, they may cause severe hypernatremia and neurological impairment when used as replacement fluids. Analysis of 38 randomised trials involving 1589 patients undergoing major abdominal aortic surgery failed to identify specific superior fluid regimens. Although crystalloid solutions that contain bicarbonate may limit the deleterious effects of chloride, the clinical and metabolic consequences of limiting chloride-rich fluids remain unclear.

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administration may be avoided in patients with a fluid deficit. Thus both volume, but only in patients returning and hence stroke result in an increase in venous pressure. A physiological "challenge" with a small bolus of intravenous fluid will result in an increase in venous return and hence stroke volume, but only in patients with a fluid deficit. Thus both inadequate and excessive fluid administration may be avoided.

When should we transfuse blood to a surgical patient?
Increased mortality among patients undergoing non-cardiac surgery is associated with both preoperative anaemia (odds ratio 1·42 (1·31 to 1·54)) and perioperative haemorrhage. However, the benefits of blood transfusion are uncertain, and further research is required in this area. The adverse effects associated with transfusion are well described, and, for surgical patients, include increased risks of postoperative infection and increased recurrence after cancer surgery. Current guidelines recommend transfusion when haemoglobin values fall below 70 g/L during the perioperative period but do not recommend transfusion to achieve values >100 g/L. A recent large randomised trial in patients undergoing proximal femoral fracture repair, which randomised patients to transfusion triggers of 100 g/L versus 80 g/L, did not show any differences in functional outcome (odds ratio 1·01 (0·84 to 1·22)). Consideration should also be given to the use of techniques which reduce transfusion requirements, including iron and erythropoietin, antifibrinolytic therapy, and red cell salvage devices. Systematic reviews indicate that use of autologous transfusion of the patient’s own blood (donated before surgery) is not associated with benefit and may increase transfusion rates.

How should we optimise perioperative nutrition?
Surgical patients should be screened for nutritional deficit and managed according to published guidelines for perioperative nutritional support. In the small proportion of patients who present for surgery with a severe nutritional deficit, caution should be taken when re-feeding after surgery either by enteral or parenteral routes. The advice of a specialist dietician should be sought. Most patients without disorders of gastric emptying who are undergoing elective surgery should be fasted for six hours for solids (including milk), but clear non-particulate oral fluids should not be withheld for more than two hours before induction of anaesthesia. Preoperative administration of carbohydrate-rich drinks two to three hours before induction of anaesthesia may enhance recovery from surgery. These approaches should be considered for most patients in the preparation for elective surgery. Meanwhile, routine use of preoperative mechanical bowel preparation is not beneficial and should be avoided, unless indicated surgically. Most patients do not require intravenous fluid once surgery is complete and can rapidly resume oral fluids and light diet.

Conclusions
Perioperative fluid therapy may have important beneficial effects on outcome after surgery. Inappropriate fluid management is likely to harm patients, but fluid prescribing practice still varies widely. Forthcoming guidelines from NICE will encourage a standardised approach to fluid prescription and management. Such initiatives are welcome and should be widely implemented to ensure the highest standards of patient care.

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EASILY MISSED?

Late onset type 1 diabetes
Daniel Lasserson,1 Robin Fox,2 Andrew Farmer1

A 41 year old man from an Indian family whose father had type 2 diabetes presented to his general practitioner with a four week history of increasing thirst and polyuria. He had not noticed any weight loss. Blood tests were arranged to confirm the diagnosis of diabetes. One week later, after having to push his car home, he began to feel exhausted and developed intermittent vomiting, which he attributed to exertion. Over the next two days he became more unwell, and the out of hours primary care service was contacted. He was reviewed urgently and admitted with diabetic ketoacidosis.

What is late onset type 1 diabetes?
A spectrum of autoimmune diabetes presents in adulthood, with type 1 diabetes characterised by the requirement of insulin at diagnosis to control glycaemia and prevent ketogenesis. Latent autoimmune diabetes of adulthood (LADA) also occurs but with much slower progression to requiring insulin after initial diagnosis.

KEY POINTS
Delay in diagnosis of adult onset type 1 diabetes can result in progression to ketoacidosis Some families have a mix of type 1 and type 2 diabetes in their pedigrees, so family history is an unreliable guide for classifying type of diabetes Routinely test for ketonuria on suspicion of diabetes in adults even if there is convincing evidence for type 2 diabetes When making a diagnosis of type 2 diabetes in a young adult in primary care, ensure that the patient is aware of the symptoms of progression to ketoacidosis to prompt early healthcare contact for reassessment

HOW COMMON IS LATE ONSET TYPE 1 DIABETES?
In the 30–50 year age group, type 1 diabetes accounts for 13% of all new cases of diabetes Annual incidence is 15/100 000 in the 15–34 year age group, increasing by 2.8% annually,2 and is 7/100 000 in the 30–50 year age group1

Why is it missed?
Similar rates of ketoacidosis are seen in patients with type 1 diabetes at diagnosis in adulthood and childhood,1 and diagnostic delay is thought to account for many presentations with ketoacidosis in children. Although the classic symptoms produced by hyperglycaemia are unlikely to be missed, there may be a delay in accurately identifying patients requiring insulin at diagnosis if there is a clinical resemblance to type 2 diabetes at initial consultation. Family history is an important component of diagnostic reasoning in primary care, and, although type 2 diabetes has a strong familial link, the incidence of type 1 diabetes is also increased in relatives of patients with type 2 diabetes.5

Why does this matter?
Delay in diagnosis of type 1 diabetes can result in the development of ketoacidosis, requiring intensive management in secondary care. Patients may also be unnecessarily exposed to the risks from dehydration, acidosis, potassium shifts, and renal failure. Large cohort studies of patients with type 1 diabetes show that, although long
How is type 1 diabetes diagnosed in adults?

Clinical features

A small cohort study reported that the classic symptoms of hyperglycaemia (thirst and polyuria) were observed at diagnosis of both adult and childhood onset type 1 diabetes, but the time course of symptoms was longer in adults (five weeks compared with two weeks) with less weight loss and few preceding superficial infections in the prodromal stage. 1 In type 2 diabetes the prodrome of symptoms of hyperglycaemia can be longer, with 20% of patients symptomatic for up to six months before diagnosis. 2 The symptoms and time course of type 1 diabetes can therefore overlap with those of type 2 diabetes, and there are no reliable clinical predictors from history and physical examination that will distinguish type 1 from type 2 diabetes in young adults. However, the National Institute for Health and Clinical Excellence (NICE) recommends that type 1 diabetes should be suspected if there is weight loss or lack of features of the metabolic syndrome in someone with newly diagnosed diabetes. 3

If diabetic ketoacidosis has developed, common features are lethargy and vomiting with an increased respiratory rate and tachycardia, and in severe presentations, reduced level of consciousness, deep sighing respiration, and collapse.

Investigations

With new onset diabetes in young adults, the traditional categories of type 1 (insulin requiring) and type 2 (insulin resistant), which are oversimplifications, are not easily discriminated at initial presentation. 4,5 The diagnostic criteria for diabetes, irrespective of type, are more straightforward, with random plasma glucose concentration ≥11.1 mmol/L and fasting plasma glucose concentration ≥7.0 mmol/L (in the presence of symptoms only one of these criteria is required). 6 Diagnosis by means of HbA1c is not recommended for type 1 diabetes.

Although a large prospective cohort study showed that type 1 and type 2 diabetes can be differentiated using laboratory markers for aetiology and level of insulin secretion (autoantibodies and C peptide levels), 7,8 they are not yet recommended for routine use in supporting diagnosis.

NICE recommends initial assessment for ketonuria as a marker of the need for exogenous insulin as other routinely ordered tests are unlikely to alert the clinician to the possibility of type 1 diabetes. Cohort studies based in secondary care show that up to 20% of patients with adult onset type 1 diabetes do not have ketonuria at presentation. 9 However, there are no community based studies of late onset type 1 diabetes, so the prevalence of ketonuria at initial presentation in primary care is not known. Nevertheless, continuing review and reassessment may be needed when the initial diagnosis is unclear, particularly if there is rapid clinical deterioration. 10 Making all patients with newly diagnosed diabetes in primary care aware of the symptoms of progression to ketoacidosis may prompt earlier healthcare contact for the minority who have type 1 diabetes that is initially misclassified as type 2.

How is late onset type 1 diabetes managed?

Initiate insulin at diagnosis to control blood glucose levels and suppress ketogenesis. Most patients will require acute assessment in secondary care for renal function, acid-base balance, and ketone body levels, as these will guide the need for intravenous therapy with insulin, fluid, and electrolytes. Detect and manage any underlying precipitants such as infection.

After initial stabilisation, a number of different insulin regimens may be suitable depending on agreed goals of therapy, although optimal glucose control requires long acting insulin to suppress fasting glucose levels and short acting insulin to reduce postprandial levels. Offer structured education encompassing dietary and lifestyle advice, self monitoring of blood glucose with adjustment of insulin doses, insulin treatment during periods of illness, and complications of diabetes. Undertake vascular risk assessment to identify and control risk factors with additional treatment. Coordination between primary and secondary care is essential to provide effective long term support.

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