

British Consensus Guidelines on Intravenous Fluid Therapy for Adult Surgical Patients

GIFTASUP

Jeremy Powell-Tuck (chair)¹, Peter Gosling², Dileep N Lobo^{1,3}, Simon P Allison¹, Gordon L Carlson^{3,4}, Marcus Gore³, Andrew J Lewington⁵, Rupert M Pearse⁶, Monty G Mythen⁶

On behalf of ¹BAPEN Medical, ²the Association for Clinical Biochemistry, ³the Association of Surgeons of Great Britain and Ireland, ⁴the Society of Academic and Research Surgery, ⁵the Renal Association and ⁶the Intensive Care Society.



With the involvement of and endorsement by:

BAPEN , the Association for Clinical Biochemistry, the Association of Surgeons of Great Britain and Ireland and Society of Academic and Research Surgery, the Renal Association and the Intensive Care Society.

And the following individuals who have participated in the consensus process:

Ravalia Abdulsatar, William Alazawi, Nawaf Al-Subaie, Charlotte Anderson, Jana Bellin, Matthew Biggart, Harsha Boralessa, Tim Bowling, Christopher Chan, Michael Colley, Euan Dickson, Simon Eaton, MarinosElia, John Feehally, Ian Fellows, Simon Gabe, David Gore, Emma Greig, Richard Griffiths, Arpan Guhu, Mark Hamilton, Sura Hamoudi, Richard Hartopp, Anne Holdoway, Alan Jackson, Shaman Jhanji, Suren Kanagasundaram, Liza Keating, Andrew Kilner, Fiona Leitch, RazMahroof, Jo Manson, Michael Margaron, Peter Mathieson, Peter McQuillan, Telet Mumtaz, Thomas Mphanza, Ruth McKee, Penny Neild, Nicholas Levy, Maria O'Donnell, Tim Palfreman, Kanthi Raveendran, Platon Raziz, Kalpana Krishna Reddy, Saxon Ridley, Brian Rowlands, G. Samra, Sophia Sanibdeen, Charles Schmulian, Jon Shaffer, Debbie Shawcross, Nicola Simmonds, Mike Stroud, Clare Soulsby, Mo Thavasoathy, Roger Townsend, Tim Trebble, Bill Tunnicliffe, Martin Tweeddale, Howard Wakeling, Jagadeesh Waran, Tim Webb, David Welsh, Chris Willars, P. Vadgama, Kumaresh Venkatesan

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Summary and recommendations

From October 2006 the Association of Surgeons of Great Britain and Ireland, SARS, BAPEN Medical, the Intensive Care Society, the Association for Clinical Biochemistry and the Renal Association nominated core members of a steering committee who came together to establish consensus for good perioperative fluid prescribing. Concern arose from a high incidence of postoperative sodium and water overload, and evidence to suggest that preventing or treating this, by more accurate fluid therapy, would improve outcome.

The following recommendations are extracted from the complete document below.

Members of the steering committee used the definitions of the Oxford Centre for Evidence-based Medicine Levels of Evidence (May 2001) accessed from <http://www.cebm.net/index.aspx?o=1025> to assign levels of evidence each of which, after debate, was accepted unanimously.

Distinction is made (a) between fluid and electrolytes required for normal existence (daily *maintenance*) and (b) for *resuscitation or replacement* of abnormal losses. No intravenous infusion should be continued simply because it is a “routine” component of clinical care. Food and fluids should be provided orally or enterally and intravenous infusions discontinued as soon as possible.

Prescribers need to understand the effects of surgical and metabolic stress on the renin-angiotensin-aldosterone system and on vasopressin elaboration. They must take care to assess the patient’s sodium, chloride, potassium, and water requirements from a knowledge of the stress response and any current deficit or excess; they must take into consideration normal maintenance requirements and the expected composition of intestinal or other losses. Requirements thus calculated are to be met based on a quantitative knowledge of the sodium, chloride and potassium contents of the fluids prescribed. Prescription should not be made without such knowledge and no intravenous fluid should be regarded as intrinsically safe.

Nutrition should be assessed and cautiously maintained. The oedematous patient should be managed with particular care, in order to achieve successful negative sodium and water balance.

Recommendation 1

Because of the risk of inducing hyperchloraemic acidosis in routine practice, when crystalloid resuscitation or replacement is indicated, balanced salt solutions e.g. Ringer’s lactate/acetate or Hartmann’s solution should replace 0.9% saline, except in cases of hypochloraemia e.g. from vomiting or gastric drainage.

Evidence level 1b¹⁻⁷

Recommendation 2

Solutions such as 4%/0.18% dextrose/saline and 5% dextrose are important sources of free water for maintenance, but should be used with caution as excessive amounts may cause dangerous hyponatraemia, especially in children and the elderly. These solutions are not appropriate for resuscitation or replacement therapy except in conditions of significant free water deficit e.g. diabetes insipidus.

Evidence level 1b⁸⁻¹¹

Recommendation 3

To meet maintenance requirements, adult patients should receive sodium 50-100 mmol/day, potassium 40-80 mmol/day in 1.5-2.5 litres of water by the oral, enteral or parenteral route (or a combination of routes). Additional amounts should only be given to correct deficit or continuing losses.

Careful monitoring should be undertaken using clinical examination, fluid balance charts, and regular weighing when possible.

Evidence level 5¹²

Preoperative fluid management

Recommendation 4

In patients without disorders of gastric emptying undergoing elective surgery clear non-particulate oral fluids should not be withheld for more than two hours prior to the induction of anaesthesia.

Evidence level 1a¹³⁻¹⁵

Recommendation 5

In the absence of disorders of gastric emptying or diabetes, preoperative administration of carbohydrate rich beverages 2-3 h before induction of anaesthesia may improve patient well being and facilitate recovery from surgery. It should be considered in the routine preoperative preparation for elective surgery.

Evidence Level 2a¹⁶⁻²⁰

Recommendation 6

Routine use of preoperative mechanical bowel preparation is not beneficial and may complicate intra and postoperative management of fluid and electrolyte balance. Its use should therefore be avoided whenever possible.

Evidence level 1a²¹⁻²³

Recommendation 7

Where mechanical bowel preparation is used, fluid and electrolyte derangements commonly occur and should be corrected by simultaneous intravenous fluid therapy with Hartmann's or Ringer-Lactate/acetate type solutions.

Evidence level 5

Recommendation 8

Excessive losses from gastric aspiration/vomiting should be treated preoperatively with an appropriate crystalloid solution which includes an appropriate potassium supplement. Hypochloraemia is an indication for the use of 0.9% saline, with sufficient additions of potassium and care not to produce sodium overload. Losses from diarrhoea/ileostomy/small bowel fistula/ileus/obstruction should be replaced volume for volume with Hartmann's or Ringer-Lactate/acetate type solutions. "Saline depletion," for example due to excessive diuretic exposure, is best managed with a balanced electrolyte solution such as Hartmann's.

[Evidence level 5 Consensus for the electrolyte content of secretions \(Table III\) based on Geigy Scientific Tables²⁴](#)

[Evidence level 2a for Hartmann's versus 0.9% saline.¹⁻⁷](#)

Recommendation 9

In high risk surgical patients preoperative treatment with intravenous fluid and inotropes should be aimed at achieving predetermined goals for cardiac output and oxygen delivery as this may improve survival.

[Evidence level 1b²⁵⁻²⁸](#)

Recommendation 10

Although currently logistically difficult in many centres, preoperative or operative hypovolaemia should be diagnosed by flow-based measurements wherever possible. The clinical context should also be taken into account as this will provide an important indication of whether hypovolaemia is possible or likely. When direct flow measurements are not possible, hypovolaemia will be diagnosed clinically on the basis of pulse, peripheral perfusion and capillary refill, venous (JVP/CVP) pressure and Glasgow Coma Scale together with acid-base and lactate measurements. A low urine output can be misleading and needs to be interpreted in the context of the patient's cardiovascular parameters above.

[Diagnosis of hypovolaemia: Evidence level 1b²⁹⁻³⁶](#)

Recommendation 11

Hypovolaemia due predominantly to blood loss should be treated with either a balanced crystalloid solution or a suitable colloid until packed red cells are available. Hypovolaemia due to severe inflammation such as infection, peritonitis, pancreatitis or burns should be treated with either a suitable colloid or a balanced crystalloid. In either clinical scenario, care must be taken to administer sufficient balanced crystalloid and colloid to normalise haemodynamic parameters and minimise overload. The ability of critically ill patients to excrete excess sodium and water is compromised, placing them at risk of severe interstitial oedema. The administration of large volumes of colloid without sufficient free water (e.g. 5% dextrose) may precipitate a hyperoncotic state.

[Suitable colloid or crystalloid for hypovolaemia: Evidence level 1b³⁷⁻⁴⁸](#)

[Administration of sufficient water: Manufacturers recommendations^{49,50}: Evidence level 5](#)

Recommendation 12

When the diagnosis of hypovolaemia is in doubt and the central venous pressure is not raised, the response to a bolus infusion of 200 ml of a suitable colloid or crystalloid should be tested. The response should be assessed using the patient's cardiac output and stroke volume measured by flow-based technology if available. Alternatively, the clinical response may be monitored by measurement/estimation of the pulse, capillary refill, CVP and blood pressure before and 15 minutes after receiving the infusion. This procedure should be repeated until there is no further increase in stroke volume and improvement in the clinical parameters.

Evidence level for flow-based measurements: 1b⁵¹

For bolus infusion: Evidence level 1b⁵²

Volume to be given: Evidence level 5 (consensus)

For suitable colloid: Evidence level 1b³⁷

Intra operative fluid management

Recommendation 13

In patients undergoing some forms of orthopaedic and abdominal surgery, intra-operative treatment with intravenous fluid to achieve an optimal value of stroke volume should be used where possible as this may reduce postoperative complication rates and duration of hospital stay.

Orthopaedic surgery: Evidence level 1b^{29,34}

Abdominal surgery: Evidence level 1a^{31-33,35,52-54}

Recommendation 14

Patients undergoing non-elective major abdominal or orthopaedic surgery should receive intravenous fluid to achieve an optimal value of stroke volume during and for the first eight hours after surgery. This may be supplemented by a low dose dopexamine infusion.

Evidence level 1b^{25-28,54,55}

Postoperative fluid, and nutritional management.

Recommendation 15

Details of fluids administered must be clearly recorded and easily accessible

Evidence level 5

Recommendation 16

When patients leave theatre for the ward, HDU or ICU their volume status should be assessed. The volume and type of fluids given perioperatively should be reviewed and compared with fluid losses in theatre including urine and insensible losses.

Recommendation 17

In patients who are euvolaemic and haemodynamically stable a return to oral fluid administration should be achieved as soon as possible.

Recommendation 18

In patients requiring continuing i.v. maintenance fluids, these should be sodium poor and of low enough volume until the patient has returned their sodium and fluid balance over the perioperative period to zero. When this has been achieved the i.v. fluid volume and content should be those required for daily maintenance and replacement of any on-going additional losses.

Recommendation 19

The haemodynamic and fluid status of those patients who fail to excrete their perioperative sodium load, and especially whose urine sodium concentration is <20mmol/L, should be reviewed.

Evidence levels for recommendations 16,17,18 & 19: 1b^{2,8,56-63}

Recommendation 20

In high risk patients undergoing major abdominal surgery, postoperative treatment with intravenous fluid and low dose dopexamine should be considered, in order to achieve a predetermined value for systemic oxygen delivery, as this may reduce postoperative complication rates and duration of hospital stay.

Evidence level 1b^{35,55,64-66}

Recommendation 21

In patients who are oedematous, hypovolaemia if present must be treated (as in Section 6g), followed by a gradual persistent negative sodium and water balance based on urine sodium concentration or excretion. Plasma potassium concentration should be monitored and where necessary potassium intake adjusted.

Evidence level 1b^{2,8,57-63,67}

Recommendation 22

Nutritionally depleted patients need cautious refeeding orally, enterally or parenterally, with feeds supplemented in potassium, phosphate and thiamine. Generally, and particularly if oedema is present, these feeds should be reduced in water and sodium. Though refeeding syndrome is a risk, improved nutrition will help to restore normal partitioning of sodium, potassium and water between intra- and extra-cellular spaces.

Evidence level 5

Recommendation 23

Surgical patients should be nutritionally screened, and NICE guidelines for perioperative nutritional support adhered to. Care should be taken to mitigate risks of the refeeding syndrome.

Evidence level 5: (NICE guidelines)⁶⁸

Fluid management in acute kidney injury(AKI)

Recommendation 24

Based on current evidence, higher molecular weight hydroxyethyl starch (hetastarch and pentastarch MW \geq 200 kDa) should be avoided in patients with severe sepsis due to an increased risk of AKI.⁶⁹⁻⁷¹

Evidence level 1b

Recommendation 25

Higher molecular weight hydroxyethyl starch(hetastarch and pentastarch MW \geq 200 kDa) should be avoided in brain-dead kidney donors due to reports of osmotic-nephrosis-like lesions.⁶⁸

Evidence level 2b

Recommendation 26

Balanced electrolyte solutions containing potassium can be used cautiously in patients with AKI closely monitored on HDU or ICU in preference to 0.9% saline. If free water is required 5% dextrose or dextrose saline should be used. Patients developing hyperkalaemia or progressive AKI should be switched to non potassium containing crystalloid solutions such as 0.45% saline or 4%/0.18 dextrose/saline

[Ringer's lactate versus 0.9% saline for patients with AKI¹](#)

Evidence level 1b

Recommendation 27

In patients with AKI fluid balance must be closely observed and fluid overload avoided. In patients who show signs of refractory fluid overload, renal replacement therapy should be considered early to mobilize interstitial oedema and correct extracellular electrolyte and acid base abnormalities.

Evidence level 5⁷²

Recommendation 28

Patients at risk of developing AKI secondary to rhabdomyolysis must receive aggressive fluid resuscitation with an isotonic crystalloid solution to correct hypovolaemia. There is insufficient evidence to recommend the specific composition of the crystalloid.

Evidence level 5⁷³

1 Introduction

Intravenous salt solutions were first used in the 1830s for the treatment of fluid loss due to cholera.^{74,75} and intravenous saline was administered to surgical patients in the late 19th century. As early as 1911 Evans⁷⁶ warned of the dangers of excessive saline administration, a warning repeated in the mid 20th century by both Collier⁷⁷ and Le Quesne.⁷⁸

In 1959 Francis Moore coined the terms 'sodium retention phase' to describe the changes which accompany the flow phase of injury and 'the sodium diuresis phase' to describe the return of the normal ability to excrete sodium chloride and water, heralding recovery and convalescence.⁷⁹ These observations emphasised how the pathophysiology of the response to injury increased the vulnerability of surgical patients to errors in fluid and electrolyte administration, and the importance of prescribing fluids with a clear understanding of this response. Despite all this previous work, a UK study in 1997 showed that postoperative patients were frequently in positive fluid balance of 7 litres or more, with a positive sodium load of 700 mmol in the first few postoperative days.⁸⁰

Whilst the problem of salt and water overload is not new, the magnitude of the problem is recent. Although avoidance of perioperative hypovolaemia remains an essential requirement and preoperative intravascular optimization improves outcome,^{25,29,30,81-83} excessive fluid infusion leading to sodium, chloride and water overload is now becoming recognized as a major cause of postoperative morbidity and a contributory factor to length of hospital stay, organ failure and mortality.⁸⁴ In a review of US practice, Arieff reviewed 13 patients who died of postoperative pulmonary oedema; their mean postoperative fluid retention was 7 litres with a positive fluid balance greater than 67 ml/kg/day within the first 36 postoperative hours.⁶⁰ In the USA if all of the other comorbidities which might be associated with pulmonary oedema were subtracted there would be 8315 patients who died each year from pulmonary oedema in the absence of causes other than excessive fluid administration.

A telephone survey,⁶² two prospective audits,^{56,63} and a postal survey of 710 consultant surgeons⁶¹ have confirmed that preregistration house officers (Foundation Year 1 doctors) are commonly made responsible for fluid prescription, but that less than half of them or their SHOs know even the sodium content of "normal" saline. Too few check fluid balance charts regularly, or appreciate for example the need for fluid replacement if bowel preparation is employed. Some consultants recognize the problem and believe that better education is needed, although they lack consensus on optimal approaches to fluid and electrolyte replacement.⁶¹ As well as drawing attention to the contribution of errors in fluid and electrolyte management to perioperative morbidity and mortality, the 1999 Report of the National Confidential Enquiry into Perioperative Deaths ascribed many of the errors to inadequate knowledge and training.⁸⁵

In this consensus document our objective is to provide a basis for good practice in adult patients and a resource for appropriate education which could be rolled out through our respective medical and surgical societies.

2 The Consensus Process

In October 2006 the Association of Surgeons of Great Britain and Ireland, SARS, BAPEN Medical, the Intensive Care Society, the Association for Clinical Biochemistry and the Renal Association nominated core members of a steering committee who came together to try to establish consensus for good perioperative fluid prescribing.

After an International Round Table meeting held at the Intensive Care Society Headquarters 27/28th February 2007, a national meeting was held in March 2007 which brought together over fifty delegates expressing interest from the named societies who were pre-circulated with the basis of the core presentations. At this meeting delegates worked through a structured agenda of presentations, large and small group discussion, and graffiti exercises. The steering committee then drafted an initial document which was circulated to all delegates who attended in March and a number of others who expressed an interest subsequently. A total of 71 people were included in this process. Comments were collated to produce a further draft which was circulated to the steering committee who met again in September 2007. Presentations relating to the document were made at very well attended conference meetings of the Association of Surgeons and SARS, BAPEN Medical, and The Intensive Care Society during 2007 which further generated discussion and feedback. A penultimate draft was produced and circulated to the wider group for final comment prior before these comments were considered at a final meeting of the steering committee in March 2008 and incorporated into the final version.

Members of the steering committee used the definitions of the Oxford Centre for Evidence-based Medicine Levels of Evidence (May 2001) accessed from <http://www.cebm.net/index.aspx?o=1025> to assign levels of evidence each of which, after debate, was accepted unanimously.

3 Overview of normal sodium and water homeostasis

Fluid and electrolyte balance should not only be considered as the external balance between the body and its environment, but also the internal balance between the extracellular compartment and between the intravascular and interstitial compartments of the extracellular fluid (Fig. 1).

The sodium intake of an adult varies with diet, but the UK Reference Nutrient Intake suitable for maintenance in normal adult males and females is 70 mmol/24 hours, which should be accompanied by about 1.5 to 2.5 litres (25 to 35 mL/kg/24h) of water.¹² Despite a wide variation in salt and water intake, in normal subjects the kidneys are able to maintain the extracellular fluid sodium concentration and osmolality within a narrow range. This is mainly achieved via the osmoreceptors and appropriate changes in vasopressin secretion affecting urinary concentration and free water clearance. In the presence of salt depletion the renin-angiotensin-aldosterone system (RAAS) is activated with consequent reduction in urinary sodium to 5 mmol/L or less. Perhaps, due to the fact that our physiology has evolved in an environment with wide variations in water availability but a relative paucity of salt, the response to changes in water intake or to a low sodium intake is both rapid and efficient. In contrast, we have not been exposed during evolution to excessive salt intake or infusion until recent times, so that the response to sodium excess is sluggish and even normal subjects are slow to excrete an excess sodium load.^{2,4,86} The excretion of excess sodium appears to be dependent on the passive and permissive suppression of the RAAS rather than any positive action of natriuretic hormone^{87,88} and is, therefore, slow. In addition studies have revealed that chloride ions cause renal vasoconstriction and reduce glomerular filtration rate resulting in sodium retention.⁸⁹

4 Overview of fluid and electrolyte therapy in injury, illness and starvation.

It is important to distinguish between fluid and electrolytes required for normal existence (daily maintenance) and for resuscitation or replacement of abnormal losses.

Crystalloid solutions contain low molecular weight salts or sugars which dissolve completely in water and pass freely between the intravascular and interstitial compartments. Colloid solutions contain larger molecular weight substances that do not dissolve completely and, depending on their molecular size, structure and the permeability of the capillaries of the patient, remain for a longer period in the vascular compartment than crystalloid solutions. Several times more crystalloid than colloid is required to achieve the same degree of vascular filling, and because crystalloid solutions move rapidly into the interstitial compartment, a side effect of crystalloid resuscitation is more interstitial oedema than in colloid treated patients.

There have been many studies attempting to compare outcomes using 'colloids' or 'crystalloids' for volume replacement therapy.⁹⁰ Unfortunately there is widespread ignorance among doctors of the content and clinical properties of different colloids and crystalloids and it is incorrect to assume that all crystalloids and all colloids have similar properties.⁹¹ Colloids in common use are gelatines, albumin and hydroxyethyl starches and all have a significant sodium and chloride content (Table 1). In general gelatines have a low molecular weight and are rapidly excreted through the kidneys giving short term volume expansion. In health, 40% of albumin is in the intravascular space and leaks through the capillary pores at a rate of 5%/h, being returned to the circulation via the lymphatic system. This flux increases in inflammatory conditions including sepsis and surgery which, together with the cost, has led to a decreased use of albumin as a volume expander for resuscitation. Hydroxyethyl starch preparations have widely differing properties depending on their average molecular weight, the degree of hydroxyethyl group substitution of the starch polymer and the C2 to C6 substitution ratio.^{92,93} There are well recognized and documented differences in the pharmacokinetic properties of these colloids.⁹³

Because the choice of colloid is controversial, where recommendation is made in these guidelines for a 'suitable colloid,' the choice is left to the practitioner based on their own judgement and experience. However, it must be emphasized that because of the widely differing properties of colloids, care must be taken to ensure that sufficient water is given to avoid hyperoncotic states which may lead to acute kidney injury.⁴⁶ No intravenous solution is without risk, and it is essential that the practitioner reads and follows the product instructions carefully and understands its limitations.

As can be seen in Table 1, with the exception of 5% dextrose solutions, almost all intravenous solutions contain sodium and chloride – some in near

physiological concentrations of 140 mmol/L for sodium and 95 mmol/L for chloride, and others in supranormal amounts e.g. 154 mmol/L Na and 154 mmol/L Cl in 0.9% (so-called “normal”) saline. Several studies have demonstrated that, in comparison with more physiological solutions such as Hartmann’s, even healthy subjects find it difficult to excrete solutions with a high chloride content such as 0.9% saline, which can cause hyperchloraemic acidosis and reduced glomerular filtration rate (GFR).^{2,4,94-96}

For the injured or surgical patient it is even more difficult to excrete a salt and water load and to maintain normal serum osmolarity for several reasons.

1. The stress response to the injury or surgery causes anti-diuresis and oliguria mediated by vasopressin, catecholamines and the RAAS. Water and salt are therefore retained even in the presence of overload. The role of natriuretic peptides in this situation is unclear.
2. Following surgery, even when the serum osmolarity is reduced by administration of hypotonic fluid, the ability to excrete free water is limited^{97,98} because the capacity of the kidney to dilute, as well as to concentrate the urine, is impaired (see below). Thus excess free water infusion risks dilutional hyponatraemia.
3. If saline is infused, chloride overload accompanies sodium overload, and hyperchloraemia causes renal vasoconstriction and reduced GFR,^{89,99} further compromising the ability of the kidney to excrete sodium and water.
4. In more seriously ill surgical catabolic patients with significant comorbidities and increased urea production, there is a reduced ability to concentrate urine.¹⁰⁰ As a consequence it requires two or more times the normal volume of urine to excrete a sodium and chloride load given in the perioperative period. Sodium and chloride excretion competes with excretion of nitrogen mobilized by the inflammatory response to surgery; a large proportion of the administered sodium, chloride and water is therefore retained as interstitial oedema.¹⁰¹
5. Potassium depletion, due both to RAAS activity and the cellular loss of potassium which accompanies protein catabolism, reduces the ability to excrete a sodium load.¹⁰²
6. Acute kidney injury may occur due to abdominal compartment syndrome compressing the kidney externally¹⁰³⁻¹⁰⁵ and increased intra-capsular pressure due to oedematous renal tissue.¹⁰⁶
7. A sustained increase in systemic capillary permeability allows albumin and its attendant fluid (18 ml for every gram of albumin) to leak into the interstitial space^{81,107,108} and thereby worsens interstitial oedema. This also causes intravascular hypovolaemia and further sodium and water retention by activation of the RAAS and secretion of vasopressin.
8. Intracellular sequestration of sodium and fluid due to lack of intracellular energy and failure of the cellular Na/K ATPase pump¹⁰⁹⁻¹¹¹ may occur in trauma, shock¹¹² and fasting/malnutrition.¹¹³ In severe cases this gives rise to the so-called sick cell syndrome.^{109,110}

In recent years there has been major concern expressed in the context of paediatric practice about the risks of dilutional hyponatraemia when hypotonic solutions are infused,^{114,115} but even in children where the risks of hyponatraemia appear to be greatest, it seems best to stress that intravenous fluids should be used with care and knowledge, rather than denounce any particular solution entirely and risk over-infusion of sodium.^{10,116-118} In postoperative adults hyponatraemia can still occur when near-isotonic solutions are used.¹¹

In the absence of complications, oliguria occurring soon after operation is usually a normal physiological response to surgery. However, at the bedside a falling urine output is commonly interpreted as indicating hypovolaemia and prompts infusion of yet more sodium-containing fluids. This not only expands the blood volume (often unnecessarily) but also over-expands the interstitial fluid volume, causing oedema and weight gain, as well as causing haemodilution, resulting in reduced serum albumin concentration and reduced haematocrit.^{2,67,86} The response to injury impairs the patient's ability to excrete the additional saline load, making interstitial oedema worse, compromising organ function and increasing the risk of morbidity and mortality. Confusion may also arise in the common context of dilutional hypoalbuminaemia and dilutional or euvolaemic hyponatraemia, both of which are erroneously taken as indications for more saline.

The key question is whether or not the oliguric patient has significant intravascular hypovolaemia which needs treatment. That can usually be decided on clinical grounds, but in more severe cases, and particularly intra-operatively, it may necessitate more invasive monitoring (see below – section 4). Clinical signs reflecting intravascular volume include capillary refill, jugular (central) venous pressure, and the trend in pulse and blood pressure. Urine output should be interpreted in the light of these clinical signs, bearing in mind the normal short term physiological effects of surgery on urine output.

Recommendation 1

Because of the risk of inducing hyperchloraemic acidosis in routine practice, when crystalloid resuscitation or replacement is indicated, balanced salt solutions e.g. Ringer's lactate/acetate or Hartmann's solution should replace 0.9% saline, except in cases of hypochloraemia e.g. from vomiting or gastric drainage.

Evidence level 1b¹⁻⁷

Recommendation 2

Solutions such as 4%/0.18% dextrose/saline and 5% dextrose are important sources of free water for maintenance, but should be used with caution as excessive amounts may cause dangerous hyponatraemia, especially in the elderly. These solutions are not appropriate for resuscitation or replacement therapy except in conditions of significant free water deficit e.g. diabetes insipidus.

Evidence level 1b⁸⁻¹¹

5 Assessment of fluid requirements

There follow guidelines for managing the common haemodynamic and fluid and electrolyte problems associated with the pre-, peri- and post-operative periods. For many surgical procedures, the assessment of fluid requirements will be straightforward, relying on the usual clinical parameters. Although the gold standard for volume replacement is invasive cardiac monitoring (see below), especially in high dependency patients, in most cases fluid requirements have to be assessed and monitored using the usual clinical approach of history, clinical examination and investigations¹¹⁹ (Table 2). No symptom or sign is pathognomonic in isolation so that a proper assessment can only be made using a combination of different variables interpreted in the light of an understanding of the underlying pathophysiology. Changes over time in any variable or the response to an appropriate fluid challenge are usually more significant than any isolated measurement. Table 2 outlines some of these variables and their possible significance.

Before any intravenous fluid is prescribed, whether for resuscitation, replacement of ongoing losses, or just maintenance, the following should be considered:

- a. Clinical assessment of the patient's fluid status, i.e. is there a deficit requiring replacement or does the patient need maintenance fluids only.
- b. Where a fluid deficit is identified (e.g. haemorrhage or vasodilatation, diarrhoea or vomitus, insensible or renal losses), the nature of the fluid deficit must be identified.
- c. The type of fluid which will best treat the deficit or maintain euvolaemia.
- d. The appropriate rate of fluid administration guided by clinical assessment and safety limits.
- e. The proposed clinical endpoint.
- f. Continued monitoring of fluid and electrolyte status.

Flow guided fluid therapy

Historically, intravenous fluid administration to treat hypovolaemia has been guided by measurements of pulse rate, arterial pressure and central venous pressure. However, this approach seems to lack sensitivity and specificity in identifying volume deficit, leading to both inadequate and excessive fluid administration (See section 6). Thus while the pattern of change in heart rate, arterial pressure and central venous pressure remain helpful markers of the effects of fluid administration on vascular filling they are influenced by other factors and do not have a linear relationship with cardiac output or tissue perfusion. The absolute values of these parameters may therefore fail to provide a reliable indication of the need for intravenous fluid administration.

Because of the Frank-Starling relationship between cardiac filling pressure and stroke volume, the latter more reliably reflects vascular filling and hence fluid requirement. With the availability of minimally invasive techniques for measurement of stroke volume and cardiac output, using trans-oesophageal

Doppler or pulse contour analysis, it has been possible to tailor fluid requirements more precisely to the needs of the individual patient. Clinical trials have demonstrated that fluid therapy guided by measurements of stroke volume and cardiac index result in significantly better clinical outcomes than those associated with traditional intraoperative monitoring. The beneficial effect is likely to relate to the early tailoring of fluid administration to the requirements of the individual patient rather than the blanket administration of extra fluid.

Maintenance requirements

In the adult, daily maintenance requirements are usually the reference nutrient intake (RNI) 70mmol sodium, and 1500-2500 ml water. In the absence of kidney disease or hyperkalaemia potassium needs to be provided in amounts close to the RNI for adults (40-80 mmol/day) – bearing in mind that in the unfed, low insulin state potassium may equilibrate more slowly with the intracellular space than when insulin or nutritionally significant amounts of carbohydrate are being administered.

Recommendation 3

To meet maintenance requirements, patients should receive sodium 50-100 mmol/day, potassium 40-80 mmol/day in 1.5-2.5 litres of water by the oral, enteral or parenteral route (or a combination of routes). Additional amounts should be given to correct deficit or continuing losses. Careful monitoring should be undertaken using clinical examination, fluid balance charts and regular weighing, when possible.

Evidence level 5¹²

6 Recommendations for preoperative fluid management

a) Euvolaemia - maintenance

Although most patients undergoing elective surgery will derive their preoperative fluids from normal oral intake, those undergoing emergency surgery may require fluid and electrolytes parenterally for both replacement and maintenance. Judgements in practice will be based on clinical parameters such as, jugular (central) venous pressure, pulse, blood pressure, capillary refill, the presence of oedema – and on fluid balance charting.

b) Preoperative fasting

Intra-and postoperative complications of fluid and electrolyte balance often have their origins in the preparation of patients for theatre. It is important that, where possible, patients are brought to theatre in a state of normal and stable fluid and electrolyte balance. Where appropriate and possible, fluid retention caused by cardiac, renal or hepatocellular disease should be corrected prior to surgery.

Patients should be screened nutritionally preoperatively and if malnourished consideration should be given to perioperative nutritional support (see page 29). It is also important that patients are adequately hydrated prior to surgery. The traditional axiom of the need to withhold all oral diet and fluids overnight prior to induction of anaesthesia has been reviewed and expert guidance, issued both in North America¹³ and in the UK¹⁵ now considers it unnecessary and undesirable to restrict access to clear, non-particulate fluids for more than two hours prior to induction of anaesthesia. These guidelines have since been supported by a Cochrane collaboration review¹⁴ reporting the results of 22 trials in which a relaxation of the period of preoperative fasting, as indicated above, failed to alter significantly the volume or pH of gastric secretions (although aspiration and regurgitation were seldom reported and studies excluded patients at particular risk of morbidity related to aspiration).

Recommendation 4

In patients without disorders of gastric emptying undergoing elective surgery clear non-particulate oral fluids should not be withheld for more than two hours prior to the induction of anaesthesia.

Evidence level 1a¹³⁻¹⁵

c) Preoperative administration of carbohydrate-rich beverages

Relaxation in the required duration of preoperative fasting has led to studies of the effect of nutrients upon patient well being and outcome after surgery. Preoperative oral administration of solutions of carbohydrate oligomers has been shown in several trials to attenuate preoperative thirst, anxiety and postoperative nausea and vomiting. It also substantially reduces postoperative insulin resistance, thereby improving the efficacy of postoperative nutritional support.¹⁶⁻²⁰

Recommendation 5

In the absence of disorders of gastric emptying or diabetes, preoperative administration of carbohydrate rich beverages 2-3 h before induction of anaesthesia may improve patient well being and facilitate recovery from surgery. It should be considered in the routine preoperative preparation for elective surgery.

Evidence level 2a¹⁶⁻²⁰

d) Bowel preparation

Many patients undergoing colonic surgery receive agents designed to empty the colon of solid faeces. Although mechanical bowel preparation was previously considered the cornerstone of safe colorectal surgery,²¹ the use of powerful laxative agents to empty the colon has significant adverse effects on perioperative fluid balance¹²⁰ and the benefits may be less clear than previously thought. Although surveys have shown that the majority of colorectal surgeons in the UK, USA and Scandinavia continue to use mechanical bowel preparation,^{121,122} this results in dehydration and derangement of plasma electrolyte concentrations, even in previously healthy patients.²³ Unless corrected preoperatively, these fluid and electrolyte derangements may complicate intra- and postoperative fluid management. This may exacerbate hypovolaemia after induction of anaesthesia, necessitating intraoperative over-replacement and causing retention of fluid in the extravascular space and postoperative oedema. Since mechanical bowel preparation may actually increase anastomotic leak rates and has failed to show any benefit in terms of reducing postoperative complication rates,²² consideration should be given to avoiding mechanical bowel preparation in routine colorectal surgery. In those cases in which mechanical bowel preparation is deemed appropriate, simultaneous intravenous fluid therapy should be administered to minimize the resulting fluid and electrolyte derangement.

Recommendation 6

Routine use of preoperative mechanical bowel preparation is not beneficial and may complicate intra and postoperative management of fluid and electrolyte balance. Its use should therefore be avoided whenever possible.

Evidence level 1a²¹⁻²³

Recommendation 7

Where mechanical bowel preparation is used, fluid and electrolyte derangements commonly occur and should be corrected by simultaneous intravenous fluid therapy with Hartmann's or Ringer-Lactate/acetate type solutions.

Evidence level 5

e) Replacement of Fluid Losses

Fluid replacement should be appropriate to the fluid deficit (e.g. pure dehydration, lack of salt and water, or intravascular hypovolaemia). Replacement is also needed in salt losing renal, or endocrine disease. Increased evaporation, for

example from hyperventilation, non-humidified face masks, open wounds or excessive sweating, results in proportionately greater need for free water replacement.

Table 3 gives the approximate electrolyte content of secreted body fluids, allowing an estimate of replacement needs, over and above maintenance requirements, when these are lost.²⁴ Thus, after correction of any current intravascular hypovolaemia (see below), the patient's daily infusion should comprise:

- i) Maintenance requirements for water and electrolytes
- ii) Replacement of water and electrolytes to correct external losses of body fluids from gastric aspirate, vomitus, diarrhoea, intestinal stoma output or enterocutaneous fistulae; or internally e.g. from pooling of fluid in the gut from ileus or obstruction.
- iii) Correction of any continuing intravascular fluid loss, e.g. from serous losses into wounds or increased albumin escape rate due to inflammation.

It is sometimes forgotten that, in the presence of hypovolaemia, the efficient mechanism for maintaining serum osmolality in normal subjects is superseded by the need to preserve volume, so that the kidney retains whatever volume or composition of fluid is infused.⁹⁸ If that fluid is hypotonic there is a risk, particularly in the elderly, of causing hyponatraemia and its clinical consequences.¹²³

Recommendation 8

Excessive losses from gastric aspiration/vomiting should be treated preoperatively with an appropriate crystalloid solution which includes an appropriate potassium supplement. Hypochloraemia is an indication for the use of 0.9% saline, with appropriate additions of potassium and care not to produce sodium overload.

Losses from diarrhoea/ileostomy/small bowel fistula/ileus/obstruction should be replaced volume for volume with Hartmann's or Ringer-Lactate/acetate type solutions.

"Saline depletion," for example due to excessive diuretic exposure, is best managed with a balanced electrolyte solution such as Hartmann's.

[Evidence level 5 Consensus, based on Geigy Scientific Tables²⁴ for body fluid composition](#)

[Evidence level 2a for Hartmann's versus 0.9% saline.¹⁻⁷](#)

f) High risk surgical patients

It is now well recognized that around 15% of patients undergoing in-patient surgical procedures are at particular risk of complications and death.^{124,125}

Factors which place such patients at increased risk relate both to the patient and to the surgical procedure and include advanced age, the presence of co-morbid

disease, major and emergency procedures. At present only a minority of such patients are admitted to critical care in the perioperative period.^{124,126} Optimal perioperative fluid management is of great importance in improving outcomes for the high-risk surgical patient. There is also a growing body of evidence to suggest that the use of low dose inotropic therapy may also be of benefit in selected cases.⁵⁵

Preoperative 'goal directed haemodynamic therapy' using protocols incorporating fluid and inotropic therapy to achieve predetermined goals for cardiac output and systemic oxygen delivery in very high-risk surgical patients has been shown to improve outcome.²⁵⁻²⁸ However, such an approach in high risk surgical patients has not been widely implemented, mainly because of the problems associated with arranging preoperative admission to intensive care. In the UK current practice focuses on the optimal fluid management of patients during surgery. In addition, for high-risk patients the use of early postoperative Goal Directed Haemodynamic Therapy may also offer additional benefit.

Recommendation 9

In high risk surgical patients preoperative treatment with intravenous fluid and inotropes should be aimed at achieving predetermined goals for cardiac output and oxygen delivery as this may improve survival.

Evidence level 1b²⁵⁻²⁸

g) Fluid resuscitation prior to urgent or emergency surgery

Patients requiring urgent or emergency surgery may present with a widely differing pattern of fluid and electrolyte deficit or redistribution e.g. from haemorrhage, sequestration of fluid within the gut and widespread capillary leak due to sepsis. Although the primary aim of preoperative preparation is to ensure adequate tissue perfusion and oxygenation, there may be little time for detailed assessment and fluid resuscitation.

The initial fluid and blood product requirement may have to be based on clinical criteria alone (Table 2). These include pulse rate, respiratory rate, arterial pressure, urine output, conscious level (Glasgow coma score), capillary refill time and the presence of peripheral cyanosis. Under these circumstances, clinical measures of end-organ function such as urine output and Glasgow coma score are particularly important and although less sensitive than flow based assessments of vascular filling, should be carefully assessed in the context of the individual patient. Arterial (or venous) blood gas analysis allows confirmation of a clinical impression of hypoperfusion as evidenced by an increasing base deficit or increased plasma lactate concentration. These biochemical markers are easily assessed and of great prognostic value.¹²⁷

It is important to make regular adjustments based on changes in the clinical parameters which first indicated hypovolaemia.

Infusion of boluses of 250 ml of hypertonic crystalloid or colloid has been shown to have some benefit in trauma patients, especially those with head injuries.¹²⁸⁻¹³⁰

Recommendation 10

Although currently logistically difficult in many centres, wherever possible preoperative or operative hypovolaemia should be diagnosed by flow based measurements. The clinical context should also be taken into account as this will provide an important indication of whether hypovolaemia is possible or likely. When direct flow measurements are not possible, hypovolaemia will be diagnosed clinically on the basis of pulse, peripheral perfusion and capillary refill, venous (JVP/CVP) pressure and GCS together with acid-base and lactate measurements. A low urine output can be misleading and needs to be interpreted in the context of the patient's cardiovascular parameters above.

Diagnosis of hypovolaemia. Evidence level 1b^{29-36,51}

Recommendation 11

Hypovolaemia due predominantly to blood loss should be treated with either a balanced crystalloid solution or a suitable colloid until packed red cells are available. Hypovolaemia due to severe inflammation such as infection, peritonitis, pancreatitis or burns should be treated with either a suitable colloid or a balanced crystalloid. In either clinical scenario, care must be taken to administer sufficient balanced crystalloid and colloid to normalise haemodynamic parameters and minimise overload. The ability of critically ill patients to excrete excess sodium and water is compromised, placing them at risk of severe interstitial oedema. The administration of large volumes of colloid without sufficient free water (e.g. 5% dextrose) may precipitate a hyperoncotic state.

Suitable colloid or crystalloid for hypovolaemia: Evidence level 1b³⁷⁻⁴⁸

Administration of sufficient water: Manufacturers recommendations^{49,50}: Evidence level 5

Recommendation 12

When the diagnosis of hypovolaemia is in doubt and the central venous pressure is not raised, the response to a bolus infusion of 200 ml of a suitable colloid or crystalloid should be tested. The response should be assessed using the patient's cardiac output and stroke volume measured by flow-based technology if available. Alternatively, the clinical response may be monitored by measurement/estimation of the pulse, capillary refill, CVP and blood pressure before and 15 minutes after receiving the infusion. This procedure should be repeated until there is no further increase in stroke volume and improvement in the clinical parameters.

For flow based monitoring: Evidence level 1b⁵¹

For bolus infusion Evidence level 1b⁵²

Volume to be given: Evidence level 5

For suitable colloid: Evidence level 1b³⁷

7 Recommendations for intra operative fluid management

The basic principle of maintaining adequate tissue perfusion must continue to be followed during surgery. However, this is influenced by a number of factors including the vasodilatory effects of anaesthesia, blood loss, the hormonal response to surgery, increased capillary permeability and albumin escape rate, and increased insensible losses.

A number of single centre trials have evaluated flow guided intra-operative fluid therapy for patients undergoing orthopaedic^{29,34} and abdominal surgery.^{31-33,35,52} Recent meta-analyses suggest this approach is associated with reductions in the duration of postoperative hospital stay and complication rates, although not mortality.^{53,54} Those studies of patients undergoing abdominal surgery have identified an earlier return to enteral feeding associated with flow directed fluid therapy.^{32,33,53,54} This observation may be explained by a reduction in mesenteric hypoperfusion and therefore postoperative ileus.¹³¹

Recommendation 13

In patients undergoing some forms of orthopaedic and abdominal surgery, intra-operative treatment with intravenous fluid to achieve an optimal value of stroke volume should be used where possible, as this is likely to reduce postoperative complication rates and duration of hospital stay.

Orthopaedic surgery: Evidence level 1b^{29,34}

Abdominal surgery: Evidence level 1a^{31-33,35,52-54}

Very few published data are available to guide fluid management of patients undergoing urgent and emergency surgery. However, in some studies of goal directed haemodynamic therapy a subgroup of patients recruited underwent emergency surgery and appeared to benefit from this approach.^{31-33,52,55} This is also supported by related research which describes significant improvements in survival when goal directed haemodynamic therapy is utilised early in the management of medical and surgical patients presenting to hospital with severe sepsis and septic shock.¹³² Further research is required to confirm this impression.

Recommendation 14

Patients undergoing non-elective major abdominal or orthopaedic surgery should receive intravenous fluid to achieve an optimal value of stroke volume during and for the first eight hours after surgery. This may be supplemented by a low dose dopexamine infusion.

Evidence level 1b^{25-28,55}

8 Recommendations for postoperative fluid management

It is essential that the haemodynamic state of the patient is assessed when they arrive on the ward, HDU or ICU. Before deciding the postoperative fluid regimen, adjustment must be made for the volume and content of fluids given pre operatively and perioperatively together with perioperative fluid losses. A frequent mistake is to implement a standard postoperative fluid regimen instead of tailoring it to the individual patient's needs. This is partly because details of fluid balance are often poorly recorded on several different documents. It is important to recognize the point where adequate replacement or resuscitation has been achieved and the goal changes to fluid and sodium mobilisation. Overloading with fluids is frequently caused by continuing a replacement regime for longer than necessary.

Postoperatively almost all patients will be in positive sodium and fluid balance often reflected by interstitial oedema, and the aim should be to restore the patient to their normal weight and extracellular volume status. Provided the patient is euvolaemic, the aim should be to allow The postoperative fluid regime should be considered in relation to the current balance due to prior fluid treatment pre- and intra-operatively. Adjustment should also be made for perioperative fluids losses and the haemodynamic state of the patient when they arrive on the ward, HDU or ICU.

Recommendation 15

Details of fluids administered must be clearly recorded and easily accessible.

[Evidence level 5](#)

Recommendation 16

When patients leave theatre for the ward, HDU or ICU their volume status should be assessed. The volume and type of fluids given perioperatively should be reviewed and compared with fluid losses in theatre including urine and insensible losses.

[Evidence level: see below](#)

Recommendation 17

In patients who are euvolaemic and haemodynamically stable a return to oral fluid administration should be achieved as soon as possible.

[Evidence level: see below](#)

Recommendation 18

In patients requiring i.v. maintenance fluids, these should be sodium poor and of low enough volume until the patient has returned their sodium and fluid balance over the peri operative period to zero. When this has been achieved the i.v. fluid volume and content should be those required for daily maintenance and replacement of any on-going additional losses.

[Evidence level: see below](#)

Recommendation 19

The haemodynamic and fluid status of those patients who fail to excrete their peri operative sodium load, and especially whose urine sodium concentration is <20mmol/L, should be reviewed.

Evidence level: see below

Evidence levels for recommendations 16, 17, 18 and 19: 1b^{2,8,56-63}

The difficulties associated with preoperative admission to intensive care have resulted in a recent change in focus for goal directed haemodynamic therapy. The findings of a recent single centre trial suggest that a postoperative Goal Directed Haemodynamic Therapy protocol designed to achieve a predetermined goal for systemic oxygen delivery for the first eight hours after surgery may significantly reduce complication rates for high-risk surgical patients.⁵⁵ These findings are supported by those of two similar trials in cardiac surgical patients^{35,66} as well as a currently unpublished meta-regression analysis of perioperative low dose dopexamine infusion.⁵⁵ The protocol utilised cardiac output monitoring to achieve an optimal value of stroke volume with intravenous fluid and in addition, for those patients who did not achieve the oxygen delivery goal, low dose dopexamine was added. Importantly, the use of low dose inotropic therapy is not associated with any increase in the risk of myocardial ischaemia⁶⁵ which has been a concern with some of the previous Goal Directed Haemodynamic Therapy protocols.

Recommendation 20

In high risk patients undergoing major abdominal surgery, postoperative treatment with intravenous fluid and low dose dopexamine should be considered, in order to achieve a predetermined value for systemic oxygen delivery, as this has been shown to reduce postoperative complication rates and duration of hospital stay.

Evidence level 1b^{35,55,64-66}

(a) Treatment of postoperative oedema

Postoperative oedema reflects accumulation of salt and water in the interstitial space and is associated with positive fluid balance and increase in weight. Potassium depletion and hyperchloraemia make mobilization of oedema more difficult. In the critically ill, oedema due to overloading of the interstitial space with salt and water can coexist with intravascular hypovolaemia. The approach to treatment is to treat intravascular hypovolaemia (section 4g) and then aim for a negative overall fluid and sodium balance. In severe cases the latter can be assessed by comparing intake with estimated losses based on measurement of sodium urine concentration and excretion: excretion should exceed intake. Diuretics should be avoided altogether or used with great caution in order to avoid a sudden reduction in the circulating blood volume. Daily weighing of the patient, when possible, allows the efficacy of oedema mobilization to be assessed.

Starving and undernourished patients are intolerant of excess salt and water. In starvation with significant weight loss, the extracellular fluid volume occupies a higher percentage of the body composition than in normal subjects i.e it is expanded relative to lean and fat mass. Though refeeding of such individuals is associated initially with additional salt and water retention as part of the refeeding syndrome, improved nutrition is necessary in order to restore to normal the intra-cellular/extracellular partitioning of sodium, potassium and water.¹³³

Recommendation 21

In patients who are oedematous, hypovolaemia if present must be treated, (as in Section 6g) followed by a gradual persistent negative sodium and water balance based on urine sodium concentration or excretion. Plasma potassium concentrations should be monitored and where necessary potassium intake adjusted.

Evidence level 1b^{2,8,57-63,67}

Recommendation 22

Nutritionally depleted patients need cautious refeeding orally, enterally or parenterally, with feeds supplemented in potassium, phosphate and thiamine. Generally, and particularly if oedema is present, these feeds should be reduced in water and sodium. Though refeeding syndrome is a risk, improved nutrition will help to restore normal partitioning of sodium, potassium and water between intra- and extra-cellular spaces.

Evidence level 5

b) Nutrition:

Nutritional aspects of care are relevant to the correct management of water and electrolytes. Modern surgical techniques are increasingly emphasizing the early postoperative re-introduction of food when possible, particularly following colorectal surgery.¹³⁴ In most patients who are eating and drinking enough, intravenous fluids should be discontinued, unless required for a specific purpose. Nutritional support should be by the simplest, safest, most cost-effective approach acceptable to the patient. Oral feeds should be favoured over enteral tube feeds, which should be preferred to parenteral nutrition.

We accept the NICE guidelines on perioperative nutritional support,⁶⁸ and summarise our position as follows:

- Patients who are significantly malnourished and are due to undergo major abdominal surgery should be considered for preoperative nutritional support.
- Oral intake should be resumed as soon as possible after surgery. Following colorectal and some other abdominal surgery, oral intake is usually possible within 24 hours. The patient should be monitored carefully for any signs of nausea or vomiting.
- Postoperatively, only malnourished patients should receive enteral tube feeding within 48 hours of surgery.

- Parenteral nutrition should be reserved for malnourished patients who cannot be fed via the intestine preoperatively, and for patients who remain unable to eat or receive enteral feeds 5 days after abdominal surgery.

The consensus group concludes that critical illness is best managed with early (first 24 hours) nutritional support preferably by the enteral route,^{135,136} or if this is not possible, parenterally.¹³⁷

Nutritional management of more complicated cases needs to take into consideration (a) the effects of malnutrition, and (b), the effects of refeeding, on water and electrolyte partitioning. Except during the early stages of fasting in obesity when there is a brief natriuresis, sodium tends to be conserved during starvation relative to the urinary excretion of potassium. Patients with severe weight (and therefore total body potassium) loss are intolerant of excess salt and water. In starvation with significant weight loss, the extracellular fluid volume is expanded relative to lean and fat mass. Refeeding of such individuals is associated with additional salt and water retention as part of the refeeding syndrome.

Potassium is the dominant intracellular and sodium the dominant extracellular cation, but because in severe illness or malnutrition there is impairment of the Na/K ATPase pump, sodium tends to move into the cells and K out, a process which is related to surgical mortality.¹³⁸ Conversely, refeeding the depleted patient is associated with rapid cellular uptake of potassium and phosphate and exhaustion of limited thiamine stores leading to the refeeding syndrome unless appropriate supplements of potassium, phosphate and thiamine are given.¹³⁹ NICE criteria⁶⁸ for identifying patients at risk of refeeding syndrome are summarized in Table 4.

The prescription for people at high risk of developing refeeding problems should consider the following taken from NICE Guidelines, Nutrition Support in Adults.⁶⁸

- Starting nutrition support at a maximum of 10 kcal/kg/day, increasing levels slowly to meet or exceed full needs by 4–7 days.
- Possibly (*our addition because such a cautious approach may need to be balanced against the timescale of surgical priorities*) using only 5 kcal/kg/day in extreme cases (for example, BMI less than 14 kg/m² or negligible intake for more than 15 days) and monitoring cardiac rhythm continually in these people and any others who already have or develop any cardiac arrhythmias.
- Restoring circulatory volume and monitoring fluid balance and overall clinical status closely
- Providing immediately before and during the first 10 days of feeding: oral thiamine 200–300 mg daily, vitamin B co strong 1 or 2 tablets, three times a day (or full dose daily intravenous vitamin B preparation, if necessary) and a balanced multivitamin/trace element supplement once daily

- Providing oral, enteral or intravenous supplements of potassium (likely requirement 2–4 mmol/kg/day), phosphate (likely requirement 0.3–0.6 mmol/kg/day) and magnesium (likely requirement 0.2 mmol/kg/day intravenous, 0.4 mmol/kg/day oral) unless pre-feeding plasma levels are high. Pre-feeding correction of low plasma levels is unnecessary.

Recommendation 23

Patients should be nutritionally screened prior to surgery, and NICE guidelines for perioperative nutritional support adhered to. Care should be taken to mitigate risks of the refeeding syndrome.

[Evidence level 5: NICE guidelines](#)⁶⁸

9. Fluid management and acute kidney injury

Identifying the optimal fluid prescription for surgical patients remains an ongoing challenge for medical practitioners. Failure to prescribe adequate intravenous fluid can place a patient at risk of developing acute kidney injury (AKI) secondary to hypovolaemia. AKI occurring after surgery has been demonstrated to be associated with a significant increase in patient morbidity and mortality.^{140,141}

Acute kidney injury is best considered as a rapid reduction in kidney function resulting in a rise in serum creatinine $\geq 25 \mu\text{mol/L}$ or a 1.5 fold increase from the baseline value. Any patient meeting these criteria should have a thorough clinical evaluation to exclude a cause of pre- or post- kidney AKI. The prescription chart should be reviewed to identify any potentially nephrotoxic drugs.

Urine output can be difficult to interpret following surgery for the first 24 hours due to the physiological response to preserve salt and water. Oliguria is recognized as a urine output $< 0.5 \text{ ml/kg/hr}$. Isolated oliguria soon after surgery does not necessarily reflect hypovolaemia and should be evaluated in the context of the patient's volume status.

Treatment of hypovolaemia in surgical patients with AKI should follow similar principles to those outlined for patients with normal renal function. However, there are some provisos; it must be recognised that patients developing pre-kidney AKI secondary to hypovolaemia will try to conserve salt and water resulting in a reduced urine output. In this setting there will be reduced capacity to excrete fluid and electrolytes. Excessive administration of salt and water will result in interstitial oedema and a greater risk of developing hyperkalaemia. Early referral to the renal team is recommended to help with clinical evaluation of the patient's volume status and appropriate fluid management.

Traditionally there has been concern in prescribing fluids containing potassium to patients with AKI due to the risks of precipitating hyperkalaemia. Ringer's lactate has been demonstrated to be safe for plasma volume expansion in patients undergoing renal transplantation.¹ In this small study patients receiving 0.9% saline had an increased incidence of metabolic acidosis and hyperkalaemia compared with patients receiving Ringer's lactate.

The question has been raised as to whether different colloid solutions may pose a risk of AKI. It is certainly important to prescribe adequate crystalloid when administering colloid solutions to avoid inducing a hyperoncotic state. However, there are data that have demonstrated higher molecular weight hydroxyethyl starch (hetastarch and pentastarch $\text{MW} \geq 200 \text{ kDa}$), is associated with an increased risk of AKI in critically ill patients with sepsis.^{71,73} A further study has reported that kidneys retrieved from brain-dead organ donors who received hydroxyethyl starch ($\text{MW} 200 \text{ kDa}$), developed osmotic-nephrosis-like lesions and had impaired immediate kidney function.⁶⁸ In contrast a large multicentre observational study of critically ill patients showed administration of HES had no

influence on renal function or the need for renal replacement therapy in the ICU,¹⁴² and there is evidence of improved renal function with low molecular weight HES (6% 130/0.4) compared with gelatine in vascular surgery.⁴²

Rhabdomyolysis (Crush Syndrome) has many causes including direct muscle trauma and compartment syndrome. Cell lysis results in release of myoglobin which is freely filtered by the kidneys and in the setting of hypovolaemia and acidosis can cause AKI. Effective management requires aggressive fluid resuscitation with a crystalloid solution.⁷³ There is limited clinical evidence to support the common practice of alkalinising the urine through the administration of sodium bicarbonate solution.

Recommendation 24

Based on current evidence higher molecular weight hydroxyethyl starch (hetastarch and pentastarch MW \geq 200 kDa) should be avoided in patients with severe sepsis due to an increased risk of AKI.⁶⁹⁻⁷¹

[Evidence level 1b](#)

Recommendation 25

Higher molecular weight hydroxyethyl starch (hetastarch and pentastarch MW \geq 200 kDa) should be avoided in brain-dead kidney donors due to reports of osmotic-nephrosis-like lesions.⁶⁸

[Evidence level 2b](#)

Recommendation 26

Balanced electrolyte solutions containing potassium can be used cautiously in patients with AKI closely monitored on HDU or ICU in preference to 0.9% saline. If free water is required 5% dextrose or dextrose saline should be used.

Patients developing hyperkalaemia or progressive AKI should be switched to non potassium containing crystalloid solutions such as 0.45% saline or 4%/0.18 dextrose/saline

[Ringer's lactate versus 0.9% saline for patients with AKI¹: Evidence level 1b](#)

Recommendation 27

In patients with AKI fluid balance must be closely observed and fluid overload avoided. In patients who show signs of refractory fluid overload, renal replacement therapy should be considered early to mobilize interstitial oedema and correct extracellular electrolyte and acid base abnormalities.

[Evidence level 5⁷²](#)

Recommendation 28

Patients at risk of developing AKI secondary to rhabdomyolysis must receive aggressive fluid resuscitation with an isotonic crystalloid solution to correct hypovolaemia. There is insufficient evidence to recommend the specific composition of the crystalloid.

[Evidence level 5⁷³](#)

References

1. O'Malley CM, Frumento RJ, Hardy MA, Benvenisty AI, Brentjens TE, Mercer JS, Bennett-Guerrero E. A randomized, double-blind comparison of lactated Ringer's solution and 0.9% NaCl during renal transplantation. *Anesth Analg* 2005;100:1518-1524.
2. Reid F, Lobo DN, Williams RN, Rowlands BJ, Allison SP. (Ab)normal saline and physiological Hartmann's solution: a randomized double-blind crossover study. *Clin Sci (Lond)* 2003;104:17-24.
3. Wilkes NJ, Woolf R, Mutch M, Mallett SV, Peachey T, Stephens R, Mythen MG. The effects of balanced versus saline-based hetastarch and crystalloid solutions on acid-base and electrolyte status and gastric mucosal perfusion in elderly surgical patients. *Anesth Analg* 2001;93:811-816.
4. Williams EL, Hildebrand KL, McCormick SA, Bedel MJ. The effect of intravenous lactated Ringer's solution versus 0.9% sodium chloride solution on serum osmolality in human volunteers [see comments]. *Anesth Analg* 1999;88:999-1003.
5. Ho AM, Karmakar MK, Contardi LH, Ng SS, Hewson JR. Excessive use of normal saline in managing traumatized patients in shock: a preventable contributor to acidosis. *J Trauma* 2001;51:173-177.
6. Waters JH, Gottlieb A, Schoenwald P, Popovich MJ, Sprung J, Nelson DR. Normal saline versus lactated Ringer's solution for intraoperative fluid management in patients undergoing abdominal aortic aneurysm repair: an outcome study. *Anesth Analg* 2001;93:817-822.
7. Boldt J, Haisch G, Suttner S, Kumle B, Schellhase F. Are lactated Ringer's solution and normal saline solution equal with regard to coagulation? *Anesth Analg* 2002;94:378-384.
8. Lobo DN, Bostock KA, Neal KR, Perkins AC, Rowlands BJ, Allison SP. Effect of salt and water balance on recovery of gastrointestinal function after elective colonic resection: a randomised controlled trial. *Lancet* 2002;359:1812-1818.
9. MacKay G, Fearon K, McConnachie A, Serpell MG, Molloy RG, O'Dwyer PJ. Randomized clinical trial of the effect of postoperative intravenous fluid restriction on recovery after elective colorectal surgery. *Br J Surg* 2006;93:1469-1474.
10. Beck CE. Hypotonic versus isotonic maintenance intravenous fluid therapy in hospitalized children: a systematic review. *Clin Pediatr (Phila)* 2007;46:764-770.
11. Steele A, Gowrishankar M, Abrahamson S, Mazer CD, Feldman RD, Halperin ML. Postoperative hyponatremia despite near-isotonic saline infusion: a phenomenon of desalination. *Ann Intern Med* 1997;126:20-25.
12. Dietary Reference Values for Food Energy and Nutrients for the United Kingdom. COMA Report on Health and Social Subjects No. 41. London: HMSO, 1991.

13. Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures: a report by the American Society of Anesthesiologist Task Force on Preoperative Fasting. *Anesthesiology* 1999;90:896-905.
14. Brady M, Kinn S, Stuart P. Preoperative fasting for adults to prevent perioperative complications. *Cochrane Database Syst Rev* 2003;CD004423.
15. Pre-operative Assessment: The Role of the Anaesthetist. London: The Association of Anaesthetists of Great Britain and Ireland, 2001.
16. Hausel J, Nygren J, Lagerkranser M, Hellstrom PM, Hammarqvist F, Almstrom C, Lindh A, Thorell A, Ljungqvist O. A carbohydrate-rich drink reduces preoperative discomfort in elective surgery patients. *Anesth Analg* 2001;93:1344-1350.
17. Hausel J, Nygren J, Thorell A, Lagerkranser M, Ljungqvist O. Randomized clinical trial of the effects of oral preoperative carbohydrates on postoperative nausea and vomiting after laparoscopic cholecystectomy. *Br J Surg* 2005;92:415-421.
18. Noblett SE, Watson DS, Huong H, Davison B, Hainsworth PJ, Horgan AF. Pre-operative oral carbohydrate loading in colorectal surgery: a randomized controlled trial. *Colorectal Dis* 2006;8:563-569.
19. Soop M, Carlson GL, Hopkinson J, Clarke S, Thorell A, Nygren J, Ljungqvist O. Randomized clinical trial of the effects of immediate enteral nutrition on metabolic responses to major colorectal surgery in an enhanced recovery protocol. *Br J Surg* 2004;91:1138-1145.
20. Soop M, Nygren J, Myrenfors P, Thorell A, Ljungqvist O. Preoperative oral carbohydrate treatment attenuates immediate postoperative insulin resistance. *Am J Physiol Endocrinol Metab* 2001;280:E576-583.
21. Chung RS, Gurll NJ, Berglund EM. A controlled clinical trial of whole gut lavage as a method of bowel preparation for colonic operations. *Am J Surg* 1979;137:75-81.
22. Jung B, Pahlman L, Nystrom PO, Nilsson E. Multicentre randomized clinical trial of mechanical bowel preparation in elective colonic resection. *Br J Surg* 2007;94:689-695.
23. Wille-Jorgensen P, Guenaga KF, Matos D, Castro AA. Pre-operative mechanical bowel cleansing or not? an updated meta-analysis. *Colorectal Dis* 2005;7:304-310.
24. Lentner C, ed. Geigy Scientific Tables. Vol. 1. Units of Measurement, Body Fluids, Composition of the Body, Nutrition. 8th ed. Basle: Ciba-Geigy Ltd., 1981.
25. Wilson J, Woods I, Fawcett J, Whall R, Dibb W, Morris C, McManus E. Reducing the risk of major elective surgery: randomised controlled trial of preoperative optimisation of oxygen delivery. *BMJ* 1999;318:1099-1103.
26. Shoemaker WC, Appel PL, Kram HB, Waxman K, Lee TS. Prospective trial of supranormal values of survivors as therapeutic goals in high-risk surgical patients. *Chest* 1988;94:1176-1186.

27. Boyd O, Grounds RM, Bennett ED. A randomized clinical trial of the effect of deliberate perioperative increase of oxygen delivery on mortality in high-risk surgical patients. *JAMA* 1993;270:2699-2707.
28. Lobo SM, Salgado PF, Castillo VG, Borim AA, Polachini CA, Palchetti JC, Brienzi SL, de Oliveira GG. Effects of maximizing oxygen delivery on morbidity and mortality in high-risk surgical patients. *Crit Care Med* 2000;28:3396-3404.
29. Sinclair S, James S, Singer M. Intraoperative intravascular volume optimisation and length of hospital stay after repair of proximal femoral fracture: randomised controlled trial. *BMJ* 1997;315:909-912.
30. Mythen MG, Webb AR. Perioperative plasma volume expansion reduces the incidence of gut mucosal hypoperfusion during cardiac surgery. *Arch Surg* 1995;130:423-429.
31. Gan TJ, Soppitt A, Maroof M, el-Moalem H, Robertson KM, Moretti E, Dwane P, Glass PS. Goal-directed intraoperative fluid administration reduces length of hospital stay after major surgery. *Anesthesiology* 2002;97:820-826.
32. Noblett SE, Snowden CP, Shenton BK, Horgan AF. Randomized clinical trial assessing the effect of Doppler-optimized fluid management on outcome after elective colorectal resection. *Br J Surg* 2006;93:1069-1076.
33. Wakeling HG, McFall MR, Jenkins CS, Woods WG, Miles WF, Barclay GR, Fleming SC. Intraoperative oesophageal Doppler guided fluid management shortens postoperative hospital stay after major bowel surgery. *Br J Anaesth* 2005;95:634-642.
34. Venn R, Steele A, Richardson P, Poloniecki J, Grounds M, Newman P. Randomized controlled trial to investigate influence of the fluid challenge on duration of hospital stay and perioperative morbidity in patients with hip fractures. *Br J Anaesth* 2002;88:65-71.
35. McKendry M, McGloin H, Saberi D, Caudwell L, Brady AR, Singer M. Randomised controlled trial assessing the impact of a nurse delivered, flow monitored protocol for optimisation of circulatory status after cardiac surgery. *BMJ* 2004;329:258.
36. Kolev N, Brase R, Swanevelder J, Oppizzi M, Riesgo MJ, van der Maaten JM, Abiad MG, Guarracino F, Zimpfer M. The influence of transoesophageal echocardiography on intra-operative decision making. A European multicentre study. European Perioperative TOE Research Group. *Anaesthesia* 1998;53:767-773.
37. Allison KP, Gosling P, Jones S, Pallister I, Porter KM. Randomized trial of hydroxyethyl starch versus gelatine for trauma resuscitation. *J Trauma* 1999;47:1114-1121.
38. Rittoo D, Gosling P, Bonnici C, Burnley S, Millns P, Simms MH, Smith SR, Vohra RK. Splanchnic oxygenation in patients undergoing abdominal aortic aneurysm repair and volume expansion with eloHAES. *Cardiovasc Surg* 2002;10:128-133.
39. Rittoo D, Gosling P, Burnley S, Bonnici C, Millns P, Simms MH, Smith SR, Vohra RK. Randomized study comparing the effects of hydroxyethyl starch

- solution with Gelofusine on pulmonary function in patients undergoing abdominal aortic aneurysm surgery. *Br J Anaesth* 2004;92:61-66.
40. Younes RN, Yin KC, Amino CJ, Itinoshe M, Rocha e Silva M, Birolini D. Use of pentastarch solution in the treatment of patients with hemorrhagic hypovolemia: randomized phase II study in the emergency room. *World J Surg* 1998;22:2-5.
 41. Boldt J, Scholhorn T, Mayer J, Piper S, Suttner S. The value of an albumin-based intravascular volume replacement strategy in elderly patients undergoing major abdominal surgery. *Anesth Analg* 2006;103:191-199.
 42. Mahmood A, Gosling P, Vohra RK. Randomized clinical trial comparing the effects on renal function of hydroxyethyl starch or gelatine during aortic aneurysm surgery. *Br J Surg* 2007;94:427-433.
 43. Matejovic M, Krouzecky A, Rokyta R, Jr., Novak I. Fluid challenge in patients at risk for fluid loading-induced pulmonary edema. *Acta Anaesthesiol Scand* 2004;48:69-73.
 44. Lang K, Suttner S, Boldt J, Kumle B, Nagel D. Volume replacement with HES 130/0.4 may reduce the inflammatory response in patients undergoing major abdominal surgery. *Can J Anaesth* 2003;50:1009-1016.
 45. Boldt J, Dücke M, Kumle B, Papsdorf M, Zurmeyer EL. Influence of different volume replacement strategies on inflammation and endothelial activation in the elderly undergoing major abdominal surgery. *Intensive Care Med* 2004;30:416-422.
 46. Gosling P, Rittoo D, Manji M, Mahmood A, Vohra R. Hydroxyethylstarch as a risk factor for acute renal failure in severe sepsis. *Lancet* 2001;358:581.
 47. Rittoo D, Gosling P, Simms MH, Smith SR, Vohra RK. The effects of hydroxyethyl starch compared with gelofusine on activated endothelium and the systemic inflammatory response following aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2005;30:520-524.
 48. Finfer S, Bellomo R, Boyce N, French J, Myburgh J, Norton R. A comparison of albumin and saline for fluid resuscitation in the intensive care unit. *N Engl J Med* 2004;350:2247-2256.
 49. Product Information: Voluven® Fresenius 6% Solution for Infusion. Bad Homburg: Fresenius Kabi, 2004.
 50. Tetraspan® Balanced Volume Therapy: Basic scientific Information. Sheffield: B Braun, 2007.
 51. Evidence review: Oesophageal Doppler monitoring in patients undergoing high-risk surgery and in critically ill patients. CEP08012: NHS Purchasing and Supply Agency, 2008. (Also available from: <http://www.pasa.nhs.uk/PASAWeb/NHSprocurement/CEP>, accessed 20 July 2008)
 52. Conway DH, Mayall R, Abdul-Latif MS, Gilligan S, Tackaberry C. Randomised controlled trial investigating the influence of intravenous fluid titration using oesophageal Doppler monitoring during bowel surgery. *Anaesthesia* 2002;57:845-849.
 53. Abbas SM, Hill AG. Systematic review of the literature for the use of oesophageal Doppler monitor for fluid replacement in major abdominal surgery. *Anaesthesia* 2008;63:44-51.

54. Walsh SR, Tang T, Bass S, Gaunt ME. Doppler-guided intra-operative fluid management during major abdominal surgery: systematic review and meta-analysis. *Int J Clin Pract* 2008;62:466-470.
55. Pearse RM, Belsey J, Cole J, Bennet ED. Effect of dopexamine infusion on mortality following major surgery: Individual patient data meta-regression analysis of published clinical trials. *Crit Care Med* 2008;36:1323-1329.
56. Walsh SR, Cook EJ, Bentley R, Farooq N, Gardner-Thorpe J, Tang T, Gaunt ME, Coveney EC. Perioperative fluid management: prospective audit. *Int J Clin Pract* 2008;62:492-497.
57. Brandstrup B, Tonnesen H, Beier-Holgersen R, Hjortso E, Ording H, Lindorff-Larsen K, Rasmussen MS, Lannig C, Wallin L, Iversen LH, Gramkow CS, Okholm M, Blemmer T, Svendsen PE, Rottensten HH, Thage B, Riis J, Jeppesen IS, Teilmann D, Christensen AM, Graungaard B, Pott F. Effects of intravenous fluid restriction on postoperative complications: comparison of two perioperative fluid regimens: a randomized assessor-blinded multicenter trial. *Ann Surg* 2003;238:641-648.
58. Kita T, Mammoto T, Kishi Y. Fluid management and postoperative respiratory disturbances in patients with transthoracic esophagectomy for carcinoma. *J Clin Anesth* 2002;14:252-256.
59. Nisanevich V, Felsenstein I, Almogy G, Weissman C, Einav S, Matot I. Effect of intraoperative fluid management on outcome after intraabdominal surgery. *Anesthesiology* 2005;103:25-32.
60. Arieff AI. Fatal postoperative pulmonary edema: pathogenesis and literature review. *Chest* 1999;115:1371-1377.
61. Lobo DN, Dube MG, Neal KR, Allison SP, Rowlands BJ. Peri-operative fluid and electrolyte management: a survey of consultant surgeons in the UK. *Ann R Coll Surg Engl* 2002;84:156-160.
62. Lobo DN, Dube MG, Neal KR, Simpson J, Rowlands BJ, Allison SP. Problems with solutions: drowning in the brine of an inadequate knowledge base. *Clin Nutr* 2001;20:125-130.
63. Walsh SR, Walsh CJ. Intravenous fluid-associated morbidity in postoperative patients. *Ann R Coll Surg Engl* 2005;87:126-130.
64. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomised, controlled trial [ISRCTN38797445]. *Crit Care* 2005;9:R687-693.
65. Pearse RM, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett D. The incidence of myocardial injury following post-operative Goal Directed Therapy. *BMC Cardiovasc Disord* 2007;7:10.
66. Polonen P, Ruokonen E, Hippelainen M, Poyhonen M, Takala J. A prospective, randomized study of goal-oriented hemodynamic therapy in cardiac surgical patients. *Anesth Analg* 2000;90:1052-1059.
67. Lobo DN, Bjarnason K, Field J, Rowlands BJ, Allison SP. Changes in weight, fluid balance and serum albumin in patients referred for nutritional support. *Clin Nutr* 1999;18:197-201.

68. National Collaborating Centre for Acute Care. Nutrition support in adults Oral nutrition support, enteral tube feeding and parenteral nutrition. London: National Collaborating Centre for Acute Care, 2006. (Also available from: http://www.rcseng.ac.uk/publications/docs/nutrition_support_guidelines.html/view?searchterm=Nutrition%20support%20in%20adults, accessed 20 July 2008).
69. Brunkhorst FM, Engel C, Bloos F, Meier-Hellmann A, Ragaller M, Weiler N, Moerer O, Gruending M, Oppert M, Grond S, Olthoff D, Jaschinski U, John S, Rossaint R, Welte T, Schaefer M, Kern P, Kuhnt E, Kiehntopf M, Hartog C, Natanson C, Loeffler M, Reinhart K. Intensive insulin therapy and pentastarch resuscitation in severe sepsis. *N Engl J Med* 2008;358:125-139.
70. Cittanova ML, Leblanc I, Legendre C, Mouquet C, Riou B, Coriat P. Effect of hydroxyethylstarch in brain-dead kidney donors on renal function in kidney-transplant recipients. *Lancet* 1996;348:1620-1622.
71. Schortgen F, Lacherade JC, Bruneel F, Cattaneo I, Hemery F, Lemaire F, Brochard L. Effects of hydroxyethylstarch and gelatin on renal function in severe sepsis: a multicentre randomised study. *Lancet* 2001;357:911-916.
72. Palevsky PM. Clinical review: Timing and dose of continuous renal replacement therapy in acute kidney injury. *Crit Care* 2007;11:232.
73. Sever MS, Vanholder R, Lameire N. Management of crush-related injuries after disasters. *N Engl J Med* 2006;354:1052-1063.
74. Latta T. Malignant cholera. Documents communicated by the Central Board of Health, London, relative to the treatment of cholera by copious injection of aqueous and saline fluids into the veins. *Lancet* 1832;ii:274-277.
75. Awad S, Allison SP, Lobo DN. The history of 0.9% saline. *Clin Nutr* 2008;27:179-188.
76. Evans GH. The abuse of normal salt solution. *JAMA* 1911;57:2126-2127.
77. Coller FA, Campbell KN, Vaughan HH, Iob LV, Moyer CA. Postoperative salt intolerance. *Ann Surg* 1944;119:533-541.
78. Le Quesne LP, Lewis AAG. Postoperative water and sodium retention. *Lancet* 1953;1:153-158.
79. Moore FD. *Metabolic Care of the Surgical Patient*. Philadelphia: W. B. Saunders, 1959.
80. Stoneham MD, Hill EL. Variability in post-operative fluid and electrolyte prescription. *Br J Clin Pract* 1997;51:82-84.
81. Zikria BA, Bascom JU. Mechanisms of multiple organ failure. In: Zikria BA, Oz MC, Carlson RW, eds. *Reperfusion injuries and clinical capillary leak syndrome*. Armonk, NY: Futura, 1994.
82. Toraman F, Evrenkaya S, Yuce M, Turek O, Aksoy N, Karabulut H, Demirhisar O, Alhan C. Highly positive intraoperative fluid balance during cardiac surgery is associated with adverse outcome. *Perfusion* 2004;19:85-91.
83. Bellomo R, Raman J, Ronco C. Intensive care unit management of the critically ill patient with fluid overload after open heart surgery. *Cardiology* 2001;96:169-176.

84. Lobo DN, Macafee DA, Allison SP. How perioperative fluid balance influences postoperative outcomes. *Best Pract Res Clin Anaesthesiol* 2006;20:439-455.
85. Callum KG, Gray AJG, Hoile RW, Ingram GS, Martin IC, Sherry KM, Whimster F. Extremes of Age: The 1999 Report of the National Confidential Enquiry into Perioperative Deaths. London: National Confidential Enquiry into Perioperative Deaths, 1999.
86. Lobo DN, Stanga Z, Simpson JAD, Anderson JA, Rowlands BJ, Allison SP. Dilution and redistribution effects of rapid 2-litre infusions of 0.9% (w/v) saline and 5% (w/v) dextrose on haematological parameters and serum biochemistry in normal subjects: a double-blind crossover study. *Clin Sci (Lond)* 2001;101:173-179.
87. Maack T. Role of atrial natriuretic factor in volume control. *Kidney Int* 1996;49:1732-1737.
88. Lobo DN, Myhill DJ, Stanga Z, Broughton Pipkin F, Allison SP. The effect of volume loading with 1 litre intravenous infusions of 0.9% saline and 5% dextrose on the renin angiotensin system (RAS) and volume controlling hormones: a randomised, double blind, crossover study [abstract]. *Clin Nutr* 2002;21 (S1):9-10.
89. Wilcox CS. Regulation of renal blood flow by plasma chloride. *J Clin Invest* 1983;71:726-735.
90. Roberts I, Alderson P, Bunn F, Chinnock P, Ker K, Schierhout G. Colloids versus crystalloids for fluid resuscitation in critically ill patients. *Cochrane Database Syst Rev* 2004:CD000567.
91. Traylor RJ, Pearl RG. Crystalloid versus colloid versus colloid: all colloids are not created equal. *Anesth Analg* 1996;83:209-212.
92. Boldt J. Volume replacement in the surgical patient - does the type of solution make a difference? *Br J Anaesth* 2000;84:783-793.
93. Boldt J, Suttner S. Plasma substitutes. *Minerva Anesthesiol* 2005;71:741-758.
94. Healey MA, Davis RE, Liu FC, Loomis W, Hoyt DB. Lactated Ringer's is superior to normal saline in a model of massive hemorrhage and resuscitation. *J Trauma* 1998;45:894-898; discussion 898-899.
95. Waters JH, Bernstein CA. Dilutional acidosis following hetastarch or albumin in healthy volunteers. *Anesthesiology* 2000;93:1184-1187.
96. Skellett S, Mayer A, Durward A, Tibby SM, Murdoch IA. Chasing the base deficit: hyperchloraemic acidosis following 0.9% saline fluid resuscitation. *Arch Dis Child* 2000;83:514-516.
97. Allison SP. Metabolic aspects of intensive care. *Br J Hosp Med* 1974;11:860-871.
98. Allison S. Fluid, electrolytes and nutrition. *Clin Med* 2004;4:573-578.
99. Veech RL. The toxic impact of parenteral solutions on the metabolism of cells: a hypothesis for physiological parenteral therapy. *Am J Clin Nutr* 1986;44:519-551.
100. Bowyer MW. Fluid therapy. In: Parsons PE, Wiener-Kronish JP, eds. *Critical Care Secrets*. St. Louis: Mosby Year Book, 1992: 24-26.

101. Gosling P. Salt of the earth or a drop in the ocean? A pathophysiological approach to fluid resuscitation. *Emerg Med J* 2003;20:306-315.
102. Krishna GG, Chusid P, Hoeldtke RD. Mild potassium depletion provokes renal sodium retention. *J Lab Clin Med* 1987;109:724-730.
103. Kucuk HF, Cevik A, Kurt N, Bildik N, Gulmen M. The effects of abdominal compartment syndrome on the serum urea and creatinine levels. *Ulus Travma Derg* 2002;8:11-15.
104. de Cleva R, Silva FP, Zilberstein B, Machado DJ. Acute renal failure due to abdominal compartment syndrome: report on four cases and literature review. *Rev Hosp Clin Fac Med Sao Paulo* 2001;56:123-130.
105. Ma YM, Qian C, Xie F, Zhou FH, Pan L, Song Q. Acute renal failure due to abdominal compartment syndrome. *Zhonghua Yi Xue Za Zhi* 2005;85:2218-2220.
106. Stone HH, Fulenwider JT. Renal decapsulation in the prevention of post ischemia oliguria. *Ann Surg* 1977;186:343-355.
107. Fleck A, Raines G, Hawker F, Trotter J, Wallace PI, Ledingham IM, Calman KC. Increased vascular permeability: a major cause of hypoalbuminaemia in disease and injury. *Lancet* 1985;1:781-784.
108. McDonald DM, Thurston G, Baluk P. Endothelial gaps as sites for plasma leakage in inflammation. *Microcirculation* 1999;6:7-22.
109. Flear CT, Singh CM. Hyponatraemia and sick cells. *Br J Anaesth* 1973;45:976-994.
110. Flear CT, Singh CM. The sick cell concept and hyponatremia in congestive heart failure and liver disease. *Lancet* 1982;2:101-102.
111. Campbell IT, Green CJ, Jackson MJ. Muscle glycogen and electrolyte concentrations in multiple organ failure [abstract]. *Proc Nutr Soc* 1998;57:111A.
112. Cunningham JN, Jr., Shires GT, Wagner Y. Changes in intracellular sodium and potassium content of red blood cells in trauma and shock. *Am J Surg* 1971;122:650-654.
113. Waterlow JC. Protein-energy malnutrition. London: Smith-Gordon and Company, 2006.
114. Moritz ML, Ayus JC. Hospital-acquired hyponatremia--why are hypotonic parenteral fluids still being used? *Nat Clin Pract Nephrol* 2007;3:374-382.
115. Yung M, Keeley S. Randomised controlled trial of intravenous maintenance fluids. *J Paediatr Child Health* 2007.
116. Choong K, Kho ME, Menon K, Bohn D. Hypotonic versus isotonic saline in hospitalised children: a systematic review. *Arch Dis Child* 2006;91:828-835.
117. Hoorn EJ, Lindemans J, Zietse R. Development of severe hyponatraemia in hospitalized patients: treatment-related risk factors and inadequate management. *Nephrol Dial Transplant* 2006;21:70-76.
118. Soupart A, Decaux G. Therapeutic recommendations for management of severe hyponatremia: current concepts on pathogenesis and prevention of neurologic complications. *Clin Nephrol* 1996;46:149-169.

119. Lobo DN, Allison SP. Fluid, electrolyte and nutrient replacement. In: Burnand KG, Young AE, Lucas J, Rowlands BJ, Scholefield J, eds. *The New Aird's Companion in Surgical Studies*. 3rd ed. London: Churchill Livingstone, 2005: 20-41.
120. Sanders G, Mercer SJ, Saeb-Parsey K, Akhavan MA, Hosie KB, Lambert AW. Randomized clinical trial of intravenous fluid replacement during bowel preparation for surgery. *Br J Surg* 2001;88:1363-1365.
121. Zmora O, Wexner SD, Hajjar L, Park T, Efron JE, Nogueras JJ, Weiss EG. Trends in preparation for colorectal surgery: survey of the members of the American Society of Colon and Rectal Surgeons. *Am Surg* 2003;69:150-154.
122. Lassen K, Hannemann P, Ljungqvist O, Fearon K, Dejong CH, von Meyenfeldt MF, Hausel J, Nygren J, Andersen J, Revhaug A. Patterns in current perioperative practice: survey of colorectal surgeons in five northern European countries. *BMJ* 2005;330:1420-1421.
123. Allison SP, Lobo DN. Fluid and electrolytes in the elderly. *Curr Opin Clin Nutr Metab Care* 2004;7:27-33.
124. Pearse RM, Harrison DA, James P, Watson D, Hinds C, Rhodes A, Grounds RM, Bennett ED. Identification and characterisation of the high-risk surgical population in the United Kingdom. *Crit Care* 2006;10:R81.
125. Cullinane M, Gray AJG, Hargraves CMK, Lansdown M, Martin IC, Schubert M. The 2003 Report of the National Confidential Enquiry into Perioperative Deaths. London: National Confidential Enquiry into Perioperative Deaths, 2003.
126. Jhanji S, Dawson J, Pearse RM. Cardiac output monitoring: basic science and clinical application. *Anaesthesia* 2008;63:172-181.
127. Kaplan LJ, Frangos S. Clinical review: Acid-base abnormalities in the intensive care unit -- part II. *Crit Care* 2005;9:198-203.
128. Wade CE, Grady JJ, Kramer GC, Younes RN, Gehlsen K, Holcroft JW. Individual patient cohort analysis of the efficacy of hypertonic saline/dextran in patients with traumatic brain injury and hypotension. *J Trauma* 1997;42:S61-65.
129. Battison C, Andrews PJ, Graham C, Petty T. Randomized, controlled trial on the effect of a 20% mannitol solution and a 7.5% saline/6% dextran solution on increased intracranial pressure after brain injury. *Crit Care Med* 2005;33:196-202; discussion 257-198.
130. Wade CE, Kramer GC, Grady JJ, Fabian TC, Younes RN. Efficacy of hypertonic 7.5% saline and 6% dextran-70 in treating trauma: a meta-analysis of controlled clinical studies. *Surgery* 1997;122:609-616.
131. Mythen MG, Webb AR. Intra-operative gut mucosal hypoperfusion is associated with increased post-operative complications and cost. *Intensive Care Med* 1994;20:99-104.
132. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001;345:1368-1377.

133. Solomon SM, Kirby DF. The refeeding syndrome: a review. *JPEN J Parenter Enteral Nutr* 1990;14:90-97.
134. Wind J, Polle SW, Fung Kon Jin PH, Dejong CH, von Meyenfeldt MF, Ubbink DT, Gouma DJ, Bemelman WA. Systematic review of enhanced recovery programmes in colonic surgery. *Br J Surg* 2006;93:800-809.
135. Heyland DK, Dhaliwal R, Drover JW, Gramlich L, Dodek P. Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. *JPEN J Parenter Enteral Nutr* 2003;27:355-373.
136. Martin CM, Doig GS, Heyland DK, Morrison T, Sibbald WJ. Multicentre, cluster-randomized clinical trial of algorithms for critical-care enteral and parenteral therapy (ACCEPT). *CMAJ* 2004;170:197-204.
137. Simpson F, Doig GS. Parenteral vs. enteral nutrition in the critically ill patient: a meta-analysis of trials using the intention to treat principle. *Intensive Care Med* 2005;31:12-23.
138. Tellado JM, Garcia-Sabrido JL, Hanley JA, Shizgal HM, Christou NV. Predicting mortality based on body composition analysis. *Ann Surg* 1989;209:81-87.
139. Stanga Z, Brunner A, Leuenberger M, Grimble RF, Shenkin A, Allison SP, Lobo DN. Nutrition in clinical practice-the refeeding syndrome: illustrative cases and guidelines for prevention and treatment. *Eur J Clin Nutr* 2008;62:687-694.
140. Chertow GM, Levy EM, Hammermeister KE, Grover F, Daley J. Independent association between acute renal failure and mortality following cardiac surgery. *Am J Med* 1998;104:343-348.
141. Praught ML, Shlipak MG. Are small changes in serum creatinine an important risk factor? *Curr Opin Nephrol Hypertens* 2005;14:265-270.
142. Sakr Y, Payen D, Reinhart K, Sipmann FS, Zavala E, Bewley J, Marx G, Vincent JL. Effects of hydroxyethyl starch administration on renal function in critically ill patients. *Br J Anaesth* 2007;98:216-224.

Tables

Table 1: Typical properties of commonly used intravenous solutions

Type of Fluid*	Sodium mmol/L	Potassium mmol/L	Chloride mmol/L	Osmolarity mosm/L	Weight average Mol Wt kD	Plasma volume expansion duration hrs [†]
Plasma	136-145	3.5-5.0	98-105	280-300	-	-
5% Dextrose	0	0	0	278	-	-
Dextrose 4% saline 0.18%	30	0	30	283		
0.9% "normal" saline	154	0	154	308	-	0.2
0.45% "half normal" saline	77	0	77	154	-	
Ringer's Lactate	130	4	109	273	-	0.2
Hartmann's	131	5	111	275	-	0.2
Gelatine 4%	145	0	145	290	30,000	1-2
5% albumin	150	0	150	300	68,000	2-4
20% albumin	-	-	-	-	68,000	2-4
HES 6% 130/0.4	154	0	154	308	130,000	4-8
HES 10% 200/0.5	154	0	154	308	200,000	6-12
HES 6% 450/0.6	154	0	154	308	450,000	24-36

* The properties of fluids may vary depending on the manufacturer.

[†] These are approximations only. The duration of clinically effective volume expansion will vary depending on several factors including, how volume expansion is defined, the rate of in vivo degradation and excretion of the fluid and the systemic capillary permeability of the individual patient.

Note: most hospitals are trying to reduce or eliminate ward-based additions to intravenous infusions, including potassium. 5% Dextrose, dextrose-saline and 0.9% normal saline solutions are readily available with added potassium and may be required for this purpose. Care must always be taken to balance sodium needs (maintenance and replacement) with the sodium load infused.

Table 2: Assessment and monitoring of fluid balance¹¹⁹

Parameter	Significance
History	Alerts to likelihood of fluid deficit (e. g. vomiting/diarrhoea/haemorrhage) or excess (e. g. from intraoperative fluids)
Weighing	24-h change in weight (performed under similar conditions) – best measure of change in water balance. Simple to carry out by bedside.
Fluid balance charts	Inherent inaccuracies in measurement and recording. Does not measure insensible loss. Large cumulative error over several days. Good measure of changes in urine output, fistula loss, gastric aspirate, etc.
Urine output	<30 ml/h is commonly used as indication for fluid infusion, but in the absence of other features of intravascular hypovolaemia is usually due to the normal oliguric response to surgery. Urine quality (e. g. urine:plasma urea or osmolality ratio) is just as important, particularly in the complicated patient.
Blood pressure	Cuff measurements may not always correlate with intraarterial monitoring. Does not necessarily correlate with flow. Affected by drugs, etc. Nonetheless, a fall is compatible with intravascular hypovolaemia, particularly when it correlates with other parameters such as pulse rate, urine output, etc.
Capillary refill	Slow refill compatible with, but not diagnostic of volume deficit. Can be influenced by temperature and peripheral vascular disease.
Autonomic responses	Pallor and sweating, particularly when combined with tachycardia, hypotension and oliguria are suggestive of intravascular volume deficit, but can also be caused by other complications, e.g. pulmonary embolus or myocardial infarction.
Skin turgor	Diminished in salt and water depletion, but also caused by ageing, cold and wasting.
Dry mouth	Usually due to mouth breathing, but compatible with salt and water depletion.
Sunken facies	May be due to starvation or wasting from disease, but compatible with salt and water depletion.
Serum biochemistry	Indicates ratio of electrolytes to water in the extracellular fluid and is a poor indicator of whole body sodium status. Hyponatraemia most commonly caused by water excess. If change in water balance over 24 h is known, then change in serum sodium concentration can guide sodium balance. Hypokalaemia nearly always indicates the need for potassium supplementation. Blood bicarbonate and chloride concentrations measured on point of care blood gas machines are useful in patients with acid-base problems including iatrogenic hyperchloraemia.
Urine biochemistry	Urine sodium concentration reflects renal perfusion and a low value (< 20 mmol/L) indicates renal hypoperfusion. Measurement of urine sodium allows assessment of postoperative sodium mobilisation (see text) Urine potassium measurement is helpful in assessing the cause of refractory hypokalaemia. Urine urea excretion increases several fold in catabolic states (e.g. sepsis) and is an indication for provision of additional free water to avoid hypernatraemia and uraemia.

Table 3: Composition of some bodily secretions

Body Secretion	Na ⁺ mmol/L	K ⁺ mmol/L	Cl ⁻ mmol/L	HCO ₃ ⁻ mmol/L	Volume L/24 hr
Saliva	2-85	0-20	16-23	14	0.5-1.5
Gastric Juice	20-60	14	140	0 – 15	2 – 3
Pancreatic Juice	125-138	8	56	85	0.7-2.5
Bile	145	5	105	30	0.6
Jejunal Juice	140	5	135	8	-
Ileal Juice	140	5	125	30	-
Ileostomy (adapted)	50	4	25	-	0.5
Colostomy	60	15	40	-	0.1 – 0.2
Diarrhoea	30 – 140	30 – 70	-	20 – 80	Variable
Normal Stool	20 – 40	30	-		0.1-0.25
Sweat (pilocarpine)	47-60	9	30-40	0-35	0.5 + variable
Visible Sweat	58	10	45	-	0.5

Note: Composition of body fluids, especially urine, varies widely, for example with flow or with food/fluid input. Furthermore gastric contents for example can represent a mixture of parietal and oxyntic secretions, saliva and bile –clinically intestinal secretions are seldom pure. These data provide a clinical guide only and are based on those body fluid compositions given in Geigy Scientific Tables,²⁴ where more detailed information can be found. If there is doubt, or greater precision is required, biochemical measurement of a patient's secretions may be advantageous.

Table 4. NICE criteria for nutritional support.⁶⁶ Nutritional support should be by the safest, simplest, most cost effective approach acceptable to the patient. Favour oral over enteral and enteral over parenteral feeding.

- Nutrition support should be considered in people who are malnourished, as defined by any of the following:
- A BMI of less than 18.5kg/m².
- Unintentional weight loss greater than 10% within the last 3-6 months
- A BMI of less than 20kg/m² and unintentional weight loss greater than 5% within the last 3-6 months.

Nutrition support should be considered in people at risk of malnutrition who, as defined by any of the following

- Have eaten little or nothing for more than 5 days and/or are likely to eat little or nothing for the next 5 days or longer
- Have a poor absorptive capacity, and/or have high nutrient losses and/or increased nutritional needs from causes such as catabolism.

Table 5: Criteria for identifying patients at high risk of developing refeeding problems⁶⁸

Patient has one or more of the following:

- BMI less than 16 kg/m²
- unintentional weight loss greater than 15% within the last 3–6 months
- little or no nutritional intake for more than 10 days
- low levels of potassium, phosphate or magnesium prior to feeding

Or patient has two or more of the following:

- BMI less than 18.5 kg/m²
- unintentional weight loss greater than 10% within the last 3–6 months
- little or no nutritional intake for more than 5 days a history of alcohol abuse or drugs including insulin, chemotherapy, antacids or diuretics

VI. Figures:

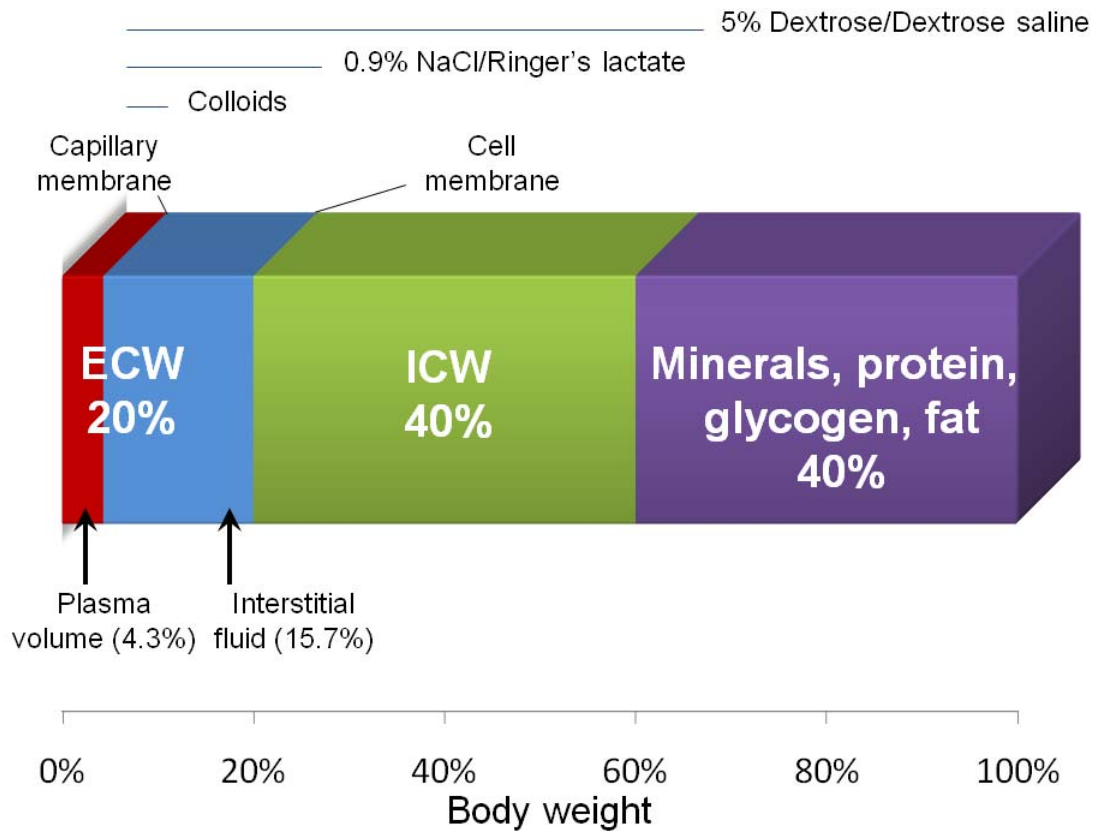


Fig 1: Body water compartments. The ability of a solution to expand the plasma volume is dependent on the volume of distribution of the solute, so that while colloids are mainly distributed in the intravascular compartment, dextrose containing solutions are distributed throughout the total body water and hence have a limited and transient volume expanding capacity. Isotonic sodium-containing crystalloids are distributed throughout the extracellular space and in practice the efficiency of these solutions to expand the plasma volume is only 20-25%, the remainder being sequestered in the interstitial space.

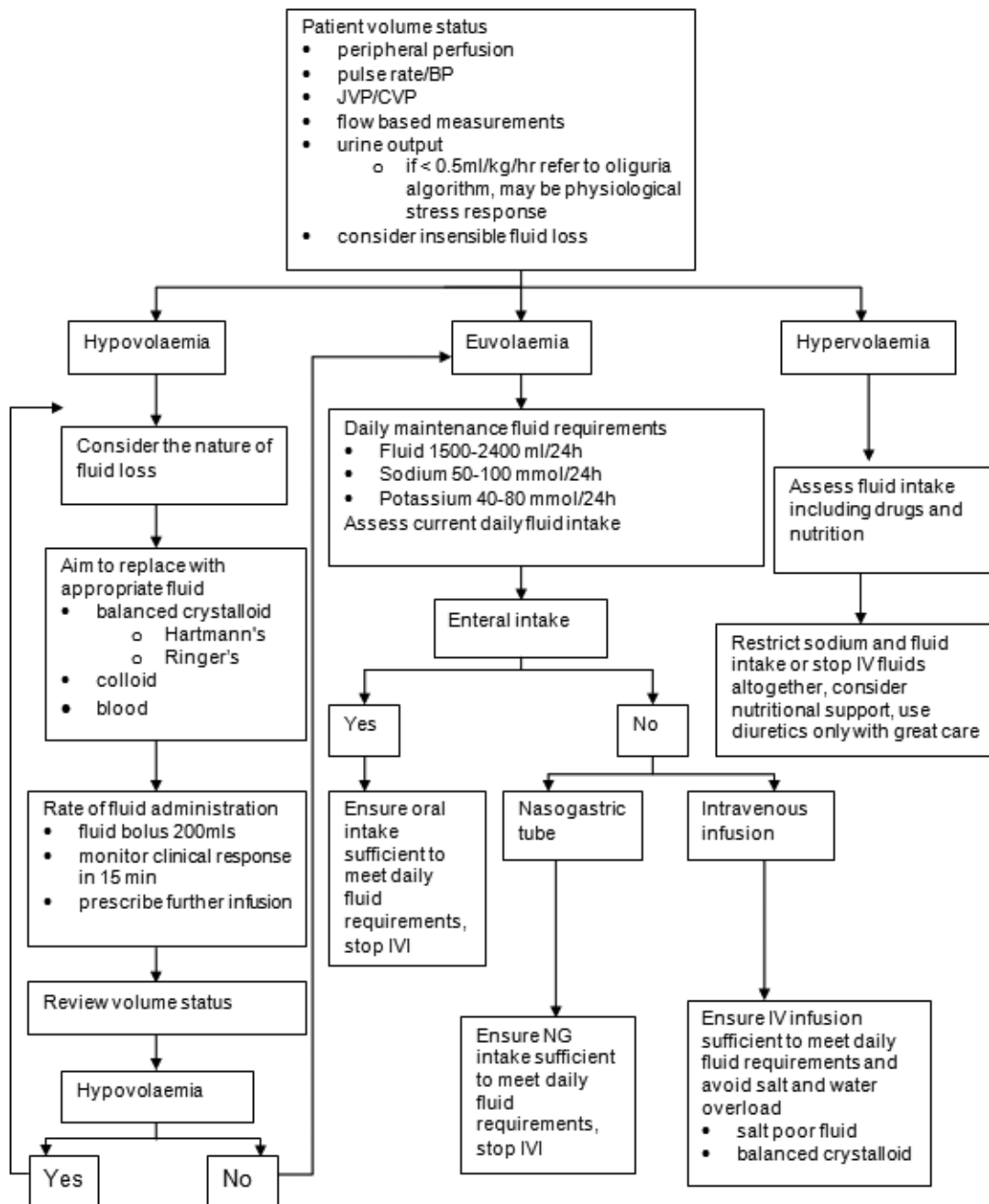


Fig. 3: Guidelines for fluid therapy

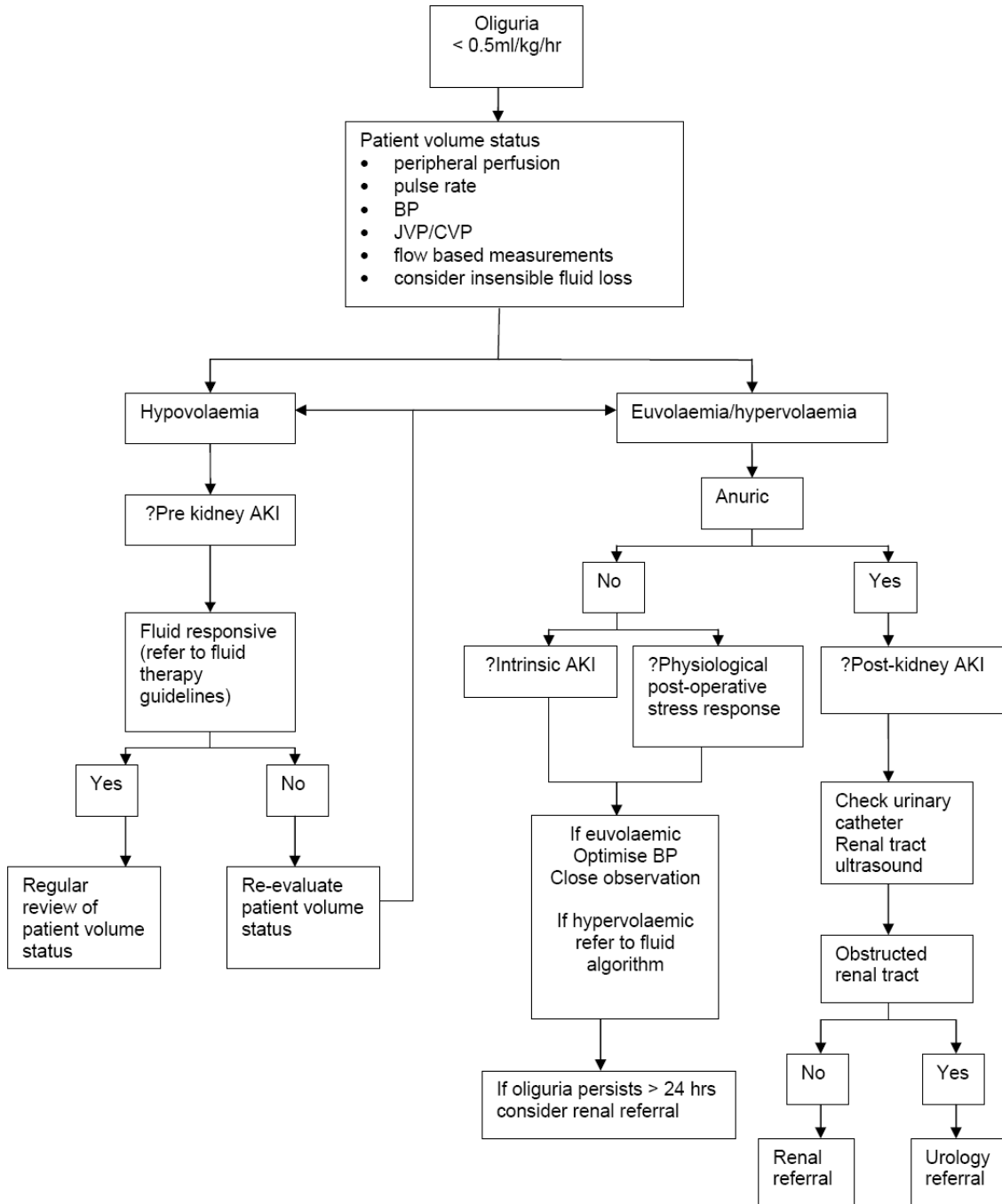


Fig. 3: Assessment of oliguria

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