venous pressure (CVP) is essential. Such a line lies in the superior vena cava and fluctuates easily with respiration. Rehydration is commenced with half normal saline. Potassium solutions are not used.

3. On receipt of the acid-base, electrolyte and haematocrit readings, an admixture of bicarbonate (44 mEq in an ampoule or 150 mEq in a bottle) and potassium (usually 40 mEq/h) may be necessary. Normal saline may be used for patients with severe hyponatraemia. This correction is made on the basis of an educated guess and is not a final or a scientific calculation.

4. The following vital functions are monitored: general appearance and mental state, blood pressure, pulse, peripheral perfusion (nailbed vasoconstriction), and urinary output. In the severely depleted or problematic patient, careful monitoring of the CVP, the ECG, and the hourly urine output (a catheter specimen measuring specific gravity and, if necessary, electrolytes and osmolality) is essential. Ideally, the patient should be admitted directly to an intensive care unit for this sort of monitoring.

5. Complex problems, e.g. arrhythmia, renal or respiratory failure, should receive early specialist attention.

6. In the urgent pre-operative situation, fluids are given rapidly (within 2 - 4 hours), titrating the absence of fluid overload, indicated by clarity of the lung bases, against the return of normal perfusion, a CVP of about 4 - 6 cm $H_2O$ and a urine output of 40 ml/h. The CVP is not an absolute measurement; instead, all observations should be considered conjointly. With careful monitoring, fluid may be given at a rate of 1 litre every half hour.

7. When the vital functions have returned to normal, or after 2 - 3 hours' therapy, the electrolyte and acid-base status should be rechecked; in this way further corrections can be made before the patient is submitted to operation.

8. Every attempt should be made to restore the patient to normovolaemia. Compensatory vasoconstriction, giving a normal blood pressure, will be abolished by anaesthesia and compounded by intra-operative fluid loss, thus leading to a disastrous fall in blood pressure.

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# Intravenous Fluid Therapy during Prolonged Surgery

## P. A. FOSTER

The regimen advocated here is for the handling of the basically healthy patient coming to surgery in order to prevent the development of shock. This is not intended as a discussion of the handling of traumatic or surgical shock.

The anaesthetist's problem is to maintain the integrity of the intracellular environment during surgery. Our access to this space is indirect in all ways (Fig. 1). Normally the route is through the vascular space via the extracellular space into the intracellular space. Therefore, our ability to monitor is indirect. The immediate dangers during surgery are either cellular hypoxia, or poor tissue perfusion, or cellular trauma, all of which cause cell swelling and expansion of the intracellular space.

Our regimen is based on the acceptance of three major premises. Firstly the work of Shires *et al.*[1,2] is accepted and it is believed that trauma produces a lesion of the interstitial extracellular space and particularly the intra-

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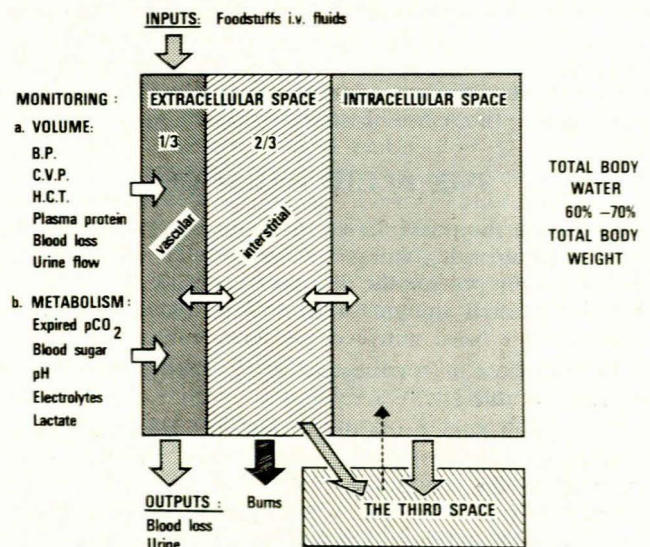
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Fig. 1. Diagrammatic representation of the different fluid spaces of the body. The 'third space' is seen as an extension of intracellular space which results from cell swelling.

cellular space, such that mainly fluid and electrolytes, possibly also protein and red cells, become sequestered from the normally exchanging pools of the body into a 'third space' from which they begin to be slowly released about 48 hours after surgery. Since the vascular and inter-stitial compartments of the extracellular space rapidly exchange water and electrolytes, the result is seen as a loss of circulating plasma volume and hypovolaemia.

The second premise is that when we restore lost blood volume, which contains both red blood corpuscles and colloids, it is necessary to consider not only the oxygen-carrying capacity, but also the colloid osmotic pressure differential between intravascular and interstitial space.[3] We do not agree with the replacement of massive blood loss with large volumes of crystalloids which some over-enthusiastic misinterpreters of Shires' concepts practise and thereby produce pulmonary oedema.

The third premise is that the trauma of surgery pro-duces a metabolic lesion, characterised chiefly by a diabetic-like state which reduces the patient's capacity to handle and utilise carbohydrate.[4] Anaesthesia may contribute to this, and the halogenated hydrocarbons such as halothane interfere with the liver's ability to handle the lactate that is produced in this insulin-resistant state.[5-8]

Our two main aims with our surgical patients are thus: firstly, to stabilise the volume of the extracellular fluid compartments which are jeopardised by: (a) pre-operative starvation; (b) blood loss, and the extracellular fluid loss that accompanies severe haemorrhage; (c) the seques-tration of fluids into non-exchanging third space created by the surgical trauma; (d) the vasodilating effects of anaesthetic drugs that may influence the ratio of blood volume to extracellular fluid volume, produce relative hypovolaemia with inadequate tissue perfusion; (e) the continuing pure water loss via the wound and the respira-tory tract.

The second aim is to provide for the nutrition of the patient. This is influenced by: (a) pre-operative starvation and dehydration; (b) the anti-insulin effects of trauma; (c) the possible hyperglycaemic or hypoglycaemic effects of certain anaesthetic drugs; (d) the change-over during long-term surgery to the use of other forms of energy substrate.

## THE FLUID BALANCE

In accepting the principles of Shires et al.,[1,2] we use poly-ionic balanced salt solutions in our patients as a routine: these not only provide the necessary variety of electrolytes, but also a small amount of free water and carbohydrate necessary for basal nutrition and evaporative loss.

The solution in routine use at Tygerberg Hospital is Hidroliet (Table I).

The rate of administration of this fluid is based on two considerations: Firstly, the accumulated negative fluid balance and the continuing hourly need for fluid — from 1,5 ml/kg/h in adults to 4 ml/kg/h in children.[9] Loss from exposed gut may amount to 250 ml/h and from breathing dry gases to 500 ml/24 h in an adult. The second factor is the additional requirement of the third space, which depends on the degree of trauma rather than

### TABLE I. COMPARISON OF SOLUTIONS

| | Hidroliet (mmol/l) | Lactated Ringer's (mmol/l) | Plasmalyte-B (mmol/l) |
|---|---|---|---|
| Na | 130 | 131 | 130 |
| K | 5,4 | 5 | 4 |
| Ca | 1,8 | 4 | — |
| Mg | 1,5 | 0 | 1,5 |
| Cl | 108 | 111 | 100 |
| Acetate | 29 | — | |
| Lactate | — | 29 | |
| Invert sugar | 5% | — | HCO₃ 28 |

It would be desirable to replace the invert sugar with glucose.

on time, but which continues to expand into the post-operative period.

In practice, intravenous crystalloid infusion starts be-fore surgery to inhibit antidiuretic hormone (ADH) secre-tion at the initial rate of 10 ml/kg/h suggested by Shires et al. However, since the patient coming to surgery is starved, dehydrated and has decreased carbohydrate re-serves, there may be benefit from a short period of rapid infusion of such a fluid before anaesthesia starts as a preload. One of our guidelines here has been the use of droperidol, a potent tranquilliser and vasodilating drug, in the pre-operative premedication in a dose of 0,15 mg/kg or 10 mg/70-kg person. Should this result in a significant reduction in the blood pressure from pre-operative values, it is assumed to indicate that the patient has a reduced interstitial fluid volume which must be immediately ex-panded, before anaesthesia is started, with crystalloids given rapidly — about 500 - 1 000 ml over a period of 5 to 10 minutes.

The adopted infusion regimen is generous, in the belief that once urine flow has been initiated at the start of an operation, and ADH and aldosterone secretion are sup-pressed, the kidney will continue to excrete excess fluid and electrolytes according to the changing needs imposed by surgery.[10] If one could monitor the needs accurately, this would be unnecessary. Since this is not possible, one aims at giving a little too much, with the reasonable expectation that the kidneys can handle such an overload.

Obviously, fluid needs vary according to the degree of trauma induced, sequestration produced, and on tissue exposure. However, if guidelines are necessary, there are 3:

1. The urine flow, which should be kept at about 1,5 ml/min — this requires the patient to be catheterised for surgery.

2. Use of a central venous pressure line in which figures of somewhere between 12 and 15 cm $H_2O$ are regarded as the upper limit, the higher figure being acceptable with intermittent positive pressure breathing.

3. The volume of crystalloid administered should be approximately 50% of the blood volume (75 ml/kg) when urine excretion cannot be monitored and third space sequestration is uncertain. This figure is based upon the fact that crystalloids are distributed through-out the interstitial space, with only one-third of the in-fused volume retained in the vascular space. The

ability of the vascular space to handle overload or loss through changing volumes of the capacitance vessels is accepted to vary 15% in either direction. In other words, 50% of the blood volume of crystalloid expands the vascular volume by about 15%.

## ANAESTHETIC TECHNIQUE

This is important and one should try throughout to maintain a reasonable degree of peripheral vasodilatation and tissue perfusion. This can be monitored by various peripheral pulse monitors which are regarded as being far more important than the ECG monitor. Good tissue perfusion at all times maintains the interstitial fluid volume, allows changes in this to be rapidly reflected as changes in blood pressure, and assures cell nutrition. Vasoconstriction can lead to metabolic acidosis and uptake of tissue fluid into the circulating blood volume.

In three categories we restrict fluids: incipient or treated cardiac failure, major neurosurgery, and pulmonary surgery when one wishes to limit organ oedema.

## BLOOD TRANSFUSION

In the presence of a normal pre-operative haemoglobin, blood transfusion is started after blood loss reaches 15% of the blood volume (calculated as ± 75 ml/kg body weight). Significant losses below this figure are treated with combined crystalloid and colloid infusions. The colloid of choice at present is the gelatine preparation Haemaccel. It appears that extracellular fluid loss along with blood is minimal below 15%, which is within the range of compensation by capacitance vessels. Therefore, provided our rate of blood replacement keeps pace with loss within 15%, extracellular fluid loss will be minimal, and one problem is simplified.

If shock develops after severe haemorrhage, extra crystalloid is given with blood.

Red cell concentrates have advantages in the replacement of blood loss, since normal bank blood is 20% diluted with crystalloid anticoagulant solution. However, more than 4 units of packed cells are never given without supplementary colloid infusion. Our present preference, human albumin being unavailable, is plasma or the colloid Haemaccel. Blood transfusions in excess of 25% of the total estimated blood volume are given through 20-$\mu$m filters to prevent respiratory symptoms postoperatively. Blood is warmed before infusion.

## THE METABOLIC LESION

The most important factor here is the liberation of adrenaline along with sympathetic activity leading to profound changes in the normal pattern of carbohydrate metabolism. Adrenaline not only suppresses the secretion of insulin, but also leads to a general overproduction with under-utilisation of glucose. There is a switch-over to significant utilisation of amino acids and fatty acids thereafter. Potentiating the adrenaline effect is glucagon and glucocorticoid secretion. There are 3 practical guidelines that may be adopted in handling the situation:

1. To give extra exogenous insulin to counteract the adrenaline effect.
2. The use of large doses of suitable drugs, as in neuroleptic analgesia techniques, to reduce adrenaline secretion and the sympathetic response. Spinal and epidural anaesthesia, by blocking sympathetic nerves, have the same effect.
3. To provide fatty acids from the start to maintain essential contracting muscle, notably the heart, at optimum efficiency from the beginning of surgery. Such fatty acids are preferentially utilised in the presence of the halogenated aliphatic inhalation anaesthetics such as halothane. Fat administration can also diminish tissue protein breakdown.

Possibly the single most important and simplest nutritional contribution one can make to the patient undergoing major surgery is to supply carbohydrate at the induction of anaesthesia to raise the hypoglycaemia of starvation before any anti-insulin effects of adrenaline appear, and when an appropriate endogenous insulin response is still possible. Initially, this carbohydrate is supplied along with the 'balanced' salt solution as 5% invert sugar, the concentration of which is low enough not to produce a notable diuretic effect. Provided the fructose dose is kept below 0,5 g/kg body weight/h (which is not a problem with the 2,5% present in 5% invert sugar), minimal side-effects need be anticipated in the normal patient.[9]

In most long cases we do not hesitate at the start of surgery to set up a special intravenous infusion of 10% invert sugar (but here preferably dextrose) with insulin, potassium and vitamin B complex constituents (KCl 20 - 60 mEq/h, insulin 10 - 20 mEq/h). Over many years of clinical observation this has been proved to stabilise the myocardium and blood pressure and to lead to : greater resistance to the development of shock, particularly in old and poor-risk patients. In the patient with poor pre-operative cardiac performance, or with a digitalised heart, or where diuretics have been used pre-operatively, we regard such an infusion as mandatory.

It is difficult to suggest the normal adult requirement for insulin during major trauma. Recent evidence suggests that about 1 unit/2 g glucose is necessary. This drops after 1 to 2 weeks to about 1 unit/6 - 7 g.[11] Our experience in a large number of open-heart cases seems to indicate that such patients can tolerate at least 10 units intravenously per hour over many hours with the heavy carbohydrate load we give. The large quantities of potassium are always run in with ECG control. The absence of any hyperkalaemic effect leads us to believe that this technique is quite safe.

Whatever the particular regimen may be, we believe it is essential to give continuous carbohydrate during prolonged surgery at least to prevent keto-acidosis after fat utilisation. The amount should be at least 10 g/h. Glucose alone in the starving individual controls keto-acidosis, but in the starving and traumatised person, insulin is needed to burn fats in the carbohydrate flame.

The use of intravenous fat emulsion during major surgery obviously has an important place in the regimen. The role of this fat emulsion is seen as supplying an immediate

need for suitable substrate for the myocardium, vascular smooth muscle, and possibly the brain, as well as preventing protein catabolism. However, it is difficult to suggest what an appropriate calorie intake should be, since the anaesthetised patient frequently has a much lowered metabolic rate in his basal condition which is further lowered by the use of muscle relaxants. Against this must be weighed the metabolic stimulating effect of adrenaline. Intralipid (500 ml) is given in a 10% solution over a 4 - 6-hour period. Indications include the malnourished patient coming for surgery, major cardiac and vascular surgery, major bowel surgery with subsequent prolonged starvation, and following severe trauma (with care after major bone fractures).

One should also remember that the effects of trauma not only commence during the period of surgery, but continue into the postoperative period. For this reason, it is necessary to continue with the use of balanced salt solutions for several hours into the postoperative period as the third space expands, and during this time also the full adrenaline metabolic effect may still develop.

## MONITORING THE PATIENT

Monitoring lines for fluid therapy and nutrition must be through the blood and these may be divided into monitoring of fluid balance and monitoring of the metabolic response.

### Fluid Balance Monitoring

In all long surgical cases, it is regarded as mandatory to use an indwelling urinary catheter and to measure urine output at half-hourly intervals. This output, as discussed previously, is maintained at least at 1,5 ml/min, which may be high for the patient in another situation. However, values two or three times in excess of this are quite acceptable during the period of surgery. One should be careful that blood pressure is adequate for renal function.

Central venous pressure lines are set up and an optimal figure of between 6 and 12 cm $H_2O$ is accepted.

Peripheral blood flow is routinely monitored — the

onset of constriction in the presence of adequate blood pressure is a sign of possible extracellular fluid decrease.

Blood pressure and blood loss are routinely measured.

Changes in the haematocrit are also significant — a haematocrit which falls to 30% is completely acceptable, but levels below 25% are to be avoided. A rising haematocrit would indicate excessive extracellular fluid loss; a falling level is associated with blood loss or fluid overload.

### Monitoring the Metabolic Response

At present there are few parameters to monitor, and here there is a place for research. One valuable parameter which is not always checked is the end-tidal $pCO_2$. Falls to the level of about 25 mmHg activate phosphofructokinase and lead to excess lactic acid production. Levels over 50 mmHg intensify adrenaline release.

Other parameters that should be monitored include: the serum electrolyte concentration; the acid-base status; the blood sugar; blood ketone bodies (difficult to determine) and blood lactate (now easily measured); and osmolality (valuable).

In conclusion, one looks with interest at the actions of somatostatin in controlling glucagon and growth hormone secretion, in reducing keto-acidosis and in controlling diabetes mellitus without insulin.[12] This hormone will doubtlessly have an important role to play during major surgery and anaesthesia.

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