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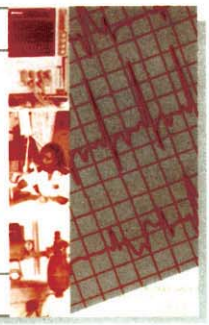
Fluid and electrolyte therapy — an approach in critically ill paediatric patients



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Introduction

'Probably, the proper use of water and electrolyte solution is responsible for saving more lives of seriously ill patients than the use of any other group of substances' D C Darrow and E L Pratt, Yale University, 1950.[1]

Fluid in the form of water is essential to maintaining a healthy human body. Electrolytes such as sodium, potassium and chloride, in addition to other nutrients, are necessary for normal growth in children.[1] Disorders of water, sodium, potassium and chloride homeostasis are common in critically ill paediatric patients. A diagnostic approach must be considered for each patient as this will facilitate the provision of safe and effective treatment strategies.

This review article will address the following aspects of fluid and electrolyte therapy in paediatric patients:

1. Basic water and electrolyte physiology
   - Body composition and compartments
   - Requirements for growth
2. Disturbances of fluid and electrolyte homeostasis and disease processes
   - Fluid (including blood)
   - pH — anion gap, acidosis, alkalosis
   - Sodium — hypernatraemia, hyponatraemia
   - Potassium — hyperkalaemia, hypokalaemia
3. A practical management approach
   - Maintenance fluid and electrolytes
   - Fluid and electrolyte deficit replacement
   - Approach to shock — an algorithm.

It is hoped that at the end of the discussion, the reader will be able to diagnose common disturbances of fluid and electrolytes in paediatric patients, and implement optimal management strategies to correct these abnormalities.

Paediatric fluid physiology

Body composition

James Gamble and Dan Darrow were pioneers in introducing the concepts of body fluid physiology and fluid therapy in paediatrics.[2] Today we know that water is the body's major constituent, comprising 60% of the weight of a lean adult. Children differ from adults in terms of body composition (Table I), water comprising over 60% of body weight in the neonate/young infant through to about 6 - 8 years of age. With the exception of newborn babies, most of this fluid is located within the intracellular fluid space (ICF). In addition, these young children have a large body surface area, a high basal metabolic rate (BMR) and immature renal mechanisms (concentrating ability) for conserving both water and electrolytes. These factors combine to predispose children to increased normal water losses (insensible and renal losses).

Children have a circulating blood volume of between 75 and 85 ml/kg, which accounts for 20 - 30% of

Table I. Approximate distribution of body water (% of total body weight)

Age	TBW	ICW	ECW
Birth	80	35	45
3 mo.	72	42	30
1 - 9 yrs	60	35	25
≥ 10 yrs	60	40	20

TBW = total body water; ICW = intracellular water; ECW = extracellular water.

extracellular fluid. Volume losses that may be inconsequential in adults therefore have the potential to seriously compromise both cardiac output and oxygen delivery in infants and children. The situation is further compounded by the fact that infants and younger children may not exhibit the normal responses to water loss, e.g. thirst, as they are unable to communicate this or may even refuse to drink fluids offered when they are seriously ill.

Normal requirements

The term 'maintenance' is used to describe the amount of fluid and electrolytes required to replace normal daily losses from the body.[2-6] The commonly used calculation of these requirements is based on predicted caloric expenditure estimated using the child's body weight.

Holliday and Seger[4] described a decrease in caloric expenditure with increasing body weight, implying that caloric expenditure per kilogram of body weight is much higher in infants and children than in adults. Accompanied by a higher BMR, this is thought also to imply a greater daily fluid requirement per kilogram of body weight (Table II).

The estimates by Holliday and Seger assume that the child is receiving a caloric intake of 100 cal/kg/day, with insensible losses of around 30 - 35 ml/kg/day and urine output of 2 - 3 ml/kg/day. Sedated and/or paralysed critically ill children require only half this amount of fluid. Calculations based on this method should be recognised as rough estimates and should be used as a guideline, titrated on the basis of clinical response.

Standard maintenance electrolyte replacement mainly involves sodium and potassium. The recommended daily requirements (Table III) are determined by growth needs. The intake of these electrolytes depends on the type of food ingested to meet caloric needs. Despite wide variations in diet, the concentrations of the electrolytes remain stable owing to renal mechanisms that alter excretion thresholds to maintain a balance. However, in disease states this situation changes and abnormalities in electrolyte levels occur.

Table III. Electrolyte requirements

Sodium
Infants 2 - 4 mEq/kg/d
Older children 1 - 3 mEq/kg/d

Potassium
Infants 4 - 6 mEq/kg/d
Older children 1 - 3 mEq/kg/d

Chemical homeostasis

Although this review addresses fluids and electrolytes, it is worth briefly mentioning mechanisms that govern plasma pH control as abnormalities in this area often accompany fluid and electrolyte disturbances.

The serum hydrogen ion concentration is maintained within tight control to ensure a pH range of 7.35 - 7.45. This is achieved through both chemical and physiological buffering mechanisms including primary buffers such as HCO_3, protein (mainly albumin which is negatively charged) and inorganic phosphate.

Fluid disturbances

Fluid loss

Fluid loss may occur as a result of acute haemorrhage (following trauma), dehydration (following gastro-enteritis), increased insensible losses with inadequate replacement (e.g. in hot climates), or where increased capillary leak leads to volume loss from the intravascular fluid space (e.g. 'third spacing' in severe sepsis).

Dehydration is a common finding in infants and children with acute gastro-enteritis. Severe dehydration is also seen in patients with diabetic keto-acidosis (DKA), but this subject is beyond the scope of this article.

Intravascular fluid loss leads to hypovolaemia, while fluid loss from the extravascular space will result in dehydration.

Table II. Estimated maintenance fluid requirements and caloric expenditure according to body weight

Body weight	Fluid amount	Caloric expenditure
1 - 10 kg	100 ml/kg/d	100 kcal/kg/d
10 - 20 kg	1 000 ml + 50 ml/kg over 10 kg/d	1 000 kcal + 50 kcal/kg over 10 kg/d
> 20 kg	1 500 ml + 20 ml/kg over 20 kg/d	1 500 kcal + 20 kcal/kg over 20 kg/d

The degree of dehydration can be assessed using a combination of clinical signs. It is important to do this assessment, as it will guide in estimating how much fluid needs to be replaced and what the preferred route of fluid administration will be. Some clinical signs, such as the turgor and feel of the skin, may also provide valuable information on the possible presence of hypernatraemia, as may changes in mental status (Table IV).

Fluid overload

Fluid overload is not a common occurrence in children, and when it does occur it implies the presence of an underlying disease process or iatrogenic cause. Some of the disease processes that may cause fluid overload are congestive heart failure leading to pulmonary oedema, and the syndrome of inappropriate antidiuretic hormone (ADH) secretion (SIADH), which can complicate central nervous system disorders (e.g. infection, trauma, tumours) and pulmonary disorders.

Iatrogenic fluid overload can occur in hospitalised children, particularly neonates and young infants, who are treated by inexperienced personnel without an adequate understanding of paediatric physiology and prescribed inappropriately high volumes of maintenance fluids for prolonged periods of time. The same principle would apply in patients with renal failure given normal maintenance fluid volumes.

Disturbance of pH

Metabolic acidosis and anion gap

This is the commonest disturbance in pH homeostasis encountered in infants and children. It is encountered in various disease processes, some of which are mentioned below in the section discussing the anion gap. The physiological response to a metabolic acidosis is hyperventilation. This compensatory decrease in partial arterial carbon dioxide pressure ($PaCO_2$) attempts to attenuate the pH drop ($pH \propto [HCO_3]/PaCO_2$). The increase in ventilation is called Kussmaul respiration and will decrease the $PaCO_2$ to 10 - 15 mmHg at most. On average, for every 1 mmol/l decrease in HCO_3 the $PaCO_2$ decreases by 1.2 mmHg. A $PaCO_2$ greater or lesser than expected indicates the presence of a mixed acid base disorder. In summary, the criteria for defining the acid base disturbance is as follows:

1. Is the pH low?

2. Is the mechanism acid load (look for presence of corresponding anion, e.g. lactate) or HCO_3 loss (no corresponding anion i.e. normal anion gap (AG))?

3. Is the compensation adequate? (for adequate compensation in metabolic acidosis a 1 mmol/l decrease in HCO_3 must be matched by a decrease of 1 mmHg in $PaCO_2$).

Table IV. Clinical assessment of dehydration in infants and children

Degree of severity	% of total body weight loss	Clinical signs	Fluid replacement guide
Mild	< 5%	Thirst Dry mucous membranes Normal to slightly increased pulse Perfusion slightly decreased Blood pressure maintained	50 ml/kg over 4 - 6 hours (if no hypernatraemia) Add maintenance fluid
Moderate	5 - 10%	Depressed fontanelle Decreased skin turgor, sunken eyes Tachycardia BP normal to decreased Delayed capillary refill time Oliguria	100 ml/kg over 4 - 6 hours (if no hypernatraemia) Add maintenance fluid
Severe	> 10%	All of the above Agitation or decreased mental status Signs of shock — BP now decreasing	100 + ml/kg over 4 - 6 h (if no hypernatraemia) Add maintenance fluid
Type dehydration	**Sodium level**	**Clinical signs**	
Isotonic	130 - 145	Skin turgor decreased, dry Mental status normal to lethargic	
Hyponatraemic	≤ 130	Skin turgor markedly decreased, clammy Coma/seizures	
Hypernatraemic	≥ 150	Skin turgor fair, doughy feel Irritability/seizures	

A similar approach can be applied for the diagnosis of metabolic alkalosis.

Anion gap[7]

The AG is calculated by subtracting the negative ions in plasma from the major positive ions, i.e. AG = (Na^+ + K^+) – (Cl^- + HCO_3). The term is really a misnomer as there is no gap, since electric neutrality is always maintained. A better term would be unmeasured anions. In health these unmeasured anions are protein (albumin), PO_4, SO_4 and some organic acids. The milli-equivalent total ranges from 7 to 17. Durward *et al.*[8] used 18 as their threshold. They also corrected for the negative charge carried by albumin. The corrected anion gap (CAG) equals (=) AG + 0.25 (normal serum albumin minus actual measured albumin). This formula has also been validated for children by Durward *et al.*[8] Hatherill *et al.*[9] showed that the CAG was more sensitive at detecting unmeasured anions (87% v. 48% using the AG). This is easily understood as a decrease in albumin results in a compensatory increase in HCO_3, leaving the net result of the AG unchanged. We believe that in severely malnourished children a similar correction for a low PO_4 may increase the sensitivity of the CAG further.

When dealing with an acidotic patient, an AG abnormality may help in evaluating the cause of the acidosis.

An elevated AG would indicate excess acid within the system:

- Increased acid production, e.g. lactate, or as may occur with DKA, ketosis, toxin ingestion (alcohol, salicylates and iron), and acute renal failure with a glomerular filtration rate of < 20 ml/min.

A normal AG in the presence of acidosis may be consistent with excess base loss from the system, as may occur in:

- Renal tubular acidosis with increased HCO_3 loss (type II)
- Decreased acid elimination as might occur in renal disease (type I or distal renal tubular acidosis)
- Diarrhoea with increased gastro-intestinal HCO_3 losses as occurs commonly in acute gastro-enteritis
- Early stages of renal failure.

Another way of evaluating the metabolic component is using the Stewart approach of strong ions. The strong ion difference (SID) gives some insight into the cause of the metabolic problem. The SID is the difference between the two strong ions Na^+ and Cl^-. The usual difference is 30 and reflects the presence of a buffer base (BB) which includes HCO_3^-, PO_4^- and albumin. Therefore electro-neutrality, which is essential, is always maintained. H^+ and OH^- act crudely as charge buffers.

An increase in the SID (> 30) indicates an increase in the BB (often HCO_3^-), which results in a decrease in Cl^- (to maintain electrical neutrality), and manifests as an alkalosis.

Similarly a decrease in SID (< 30) indicates a decrease in the BB (often HCO_3^-), which results in an increase in Cl^- (to maintain electrical neutrality), and manifests as an acidosis.

Metabolic alkalosis

This is less common than metabolic acidosis.The pathophysiology of metabolic alkalosis includes a process that generates the alkalosis and one that maintains it. Generation can be due to H^+ loss, transcellular shift of H^+, HCO_3^- retention and contraction alkalosis. Maintenance is usually due to HCO_3^- reabsorption.

Common causes are:

H^+ loss:
Renal
- Mineralocorticoid excess
- Hypoparathyroidism.

Gastro-intestinal
- Vomiting
- Congenital chloridorrhoea.

Transcellular shift of H^+ due to hypokalaemia.
HCO_3^- retention:
- Administration of sodium bicarbonate
- Massive blood transfusions (citrate).

A metabolic alkalosis is diagnosed as follows:
- A raised pH.
- There is a primary HCO_3^- excess.
- This is followed by a compensatory increase in the partial pressure of arterial carbon dioxide (PCO_2) (0.6 mmol/l for every 1 mmol/l ↑ in HCO_3^-) — respiratory compensation.

Management of metabolic alkalosis

This can be viewed in terms of the cause.

Saline responsive (volume depletion).
- Metabolic alkalosis mainly due to vomiting, nasogastric suction and diuretics results in a decreased extracellular fluid (ECF) volume. This increases Na^+ reabsorption and hence H^+ secretion (HCO_3^- reabsorption).
- Re-expansion of the intravascular volume with isotonic saline decreases Na^+ reabsorption and hence HCO_3^- reabsorption.

Saline resistant (normal volume but K^+ depleted)

- A low ECF[K^+] results in K^+ moving out of the cell in order to increase the ECK[K^+]. To maintain electroneutrality H^+ moves into the cell. In the renal proximal tubular cells this results in ↑H^+ secretion and subsequent HCO_3^- regeneration.

- Treatment here would include KCl administration to correct the hypokalaemia.

Electrolyte disturbances

Most disease processes that involve fluid loss from the body are complicated by disturbances of two main electrolytes, sodium and potassium.

Hypernatraemia (serum sodium > 150 mmol/l) occurs when there is an excess loss of water relative to electrolytes, i.e hypertonic dehydration, or as a result of diabetes insipidus or excessive sodium intake (i.e. incorrectly mixed home-made oral rehydration solution).

Hyponatraemia (serum sodium < 130 mmol/l) can occur in the setting of hypo-, hyper- and iso-osmotic plasma. Measurement of plasma osmolality excludes pseudohyponatraemia (iso-osmolar plasma) and differentiates between hypo- and hyper-osmolar hyponatraemia, thereby aiding management.

Potassium disturbances also reflect either a low serum potassium (sK) (< 3.5 mmol/l) or hyperkalaemia (> 5.5 mmol/l). Important to note is that sK is only ~2% of total body K (TBK). In fact a decrease of 0.3 mmol/l may reflect a total loss of ~ 100 mmol/l. Evaluating the mechanism of hypokalaemia (trans-cellular shift v. loss) aids in interpreting the sK.

Management of these disturbances will be discussed below.

Management approach

General principles

Emergency management principles require a quick primary survey of the patient to assess the degree of compromise and to what extent the condition is life-threatening.[10] This is followed by application of basic life support intervention if necessary. Once the patient has been assessed and stabilised, active treatment must be prioritised and commenced. Continuous evaluation and titration of therapy is important to assess ongoing needs or whether physiological endpoints have been achieved. Treatment must be directed towards correcting abnormal physiology. Before one can recognise deviation from normal, one needs to familiarise oneself with accepted normal physiological limits for paediatric patients. Table V gives normal heart rates and blood pressures for different age groups.

Some developmental physiological factors make paediatric patients different from adults.

- Cardiac physiology. Infants and young children have fewer, immature myocytes with a different expression of myocardial protein composition. There is a paucity of sarcoplasmic reticulum and calcium channels. They have a decreased number of beta- and alpha-receptors that are functionally desensitised to catecholamines. Ventricular interdependence may be an important factor, particularly in younger infants with high right-sided pressures as there is impairment of left ventricular (LV) ejection secondary to the interventricular septum bulging into the left ventricle.

- Young infants and children do not have the capacity to vary their stroke volume as much as adults.[10]

- The systemic vascular resistance tends to rise in response to volume loss in paediatric patients, so blood pressure may be maintained at near normal even in early stages of shock. This makes blood pressure on its own a poor assessment of circulatory compromise.[11-13]

- Delayed volume repletion leads to prolonged tissue ischaemic-hypoxic damage which is associated with significant ischaemia-reperfusion injury and an increased morbidity and mortality after resuscitation.

Once the above factors are borne in mind, it becomes clear that early recognition of seriousness of circulatory compromise and aggressive volume repletion with continuous re-evaluation and referral to the next level of care are of critical importance when managing fluid loss in infants and children.[14-18]

Replacement of deficits and treatment of shock

Table IV illustrates the recommended fluid volumes to be used in replacing existing fluid losses. Isotonic crystalloids (e.g. normal saline) remain the mainstay in volume resuscitation for the paediatric patient.[15] The use of colloid has been shown to be effective in certain paediatric disease processes presenting with shock,[16,17] but analysis of adult studies by the Cochrane review group suggests an increased mortality in adult patients with sepsis when albumin was used.[19] Colloids do tend to be expensive, and with the exception of blood replacement, most acute volume deficits can be optimally addressed using isotonic crystalloids.

The treatment approach to shock is illustrated in Fig. 1. It is important to note that the times between each of the steps must be as short as possible, to minimise delays in correcting circulatory compromise. There is some evidence to suggest that the use of corticosteroids is beneficial in septic shock com-

Table V. Normal physiological parameters

Heat rate at rest (/min)

	Lower limit		Average		Upper limt	
Newborn	70		125		190	
1 - 11 mo.	80		120		160	
2 yrs	80		110		130	
4 yrs	80		100		120	
6 yrs	75		100		115	
8 yrs	70		90		110	
10 yrs	70		90		110	
	Boys	**Girls**	**Boys**	**Girls**	**Boys**	**Girls**
12 yrs	65	70	85	90	105	110
14 yrs	60	65	80	85	100	105
16 yrs	55	60	75	80	95	100
18 yrs	50	55	70	75	90	95

Blood pressure values (mmHg)

	Mean systolic	Mean diastolic
Newborn	75	40
6 mo. - 1 yr	89	60
2 yrs	99	64
4 yrs	99	65
5 - 6 yrs	94	55
7 - 8 yrs	102	56
9 - 10 yrs	107	57
11 - 12 yrs	113	59
13 - 14 yrs	118	60

plicated by intractable hypotension in paediatric patients. A number of studies have shown good evidence that this modality of treatment is valuable.[20]

Managing sodium disturbances

Hyponatraemia

Hyponatraemia is defined as a serum sodium level < 130 mmol/l. Beware of treating pseudo-hyponatraemia caused by high protein or high lipid concentrations. Here the plasma osmolality is normal in the face of a low plasma Na, which is due to a technical aspect of Na measurement and does not in fact reflect a low Na.

In the case of hyperglycaemia the plasma osmolality is high due to the presence of glucose. Calculate the corrected Na as follows:

Corrected plasma Na = plasma Na + (plasma glucose – 5.5/5.5) × 1.5.

This corrects for the osmotic effect of glucose which is responsible for drawing water into the vascular compartment and diluting the Na. It may also cause an osmotic diuresis resulting in the loss of water and Na.

Sodium deficit replacement is definitely necessary for levels below 120 mmol/l. Treatment may also be required at higher levels in symptomatic cases (e.g. patient with seizures) and in cases where the decrease in Na is acute. First, the required replacement values are calculated using the following formula:

Required sodium (in mmol) = (desired sodium — actual sodium) × weight × 0.6

The required amount is then administered with maintenance fluids over 24 hours. Care must be taken not to raise the serum sodium too quickly (not more than 0.5 - 1 (mmol/h) as this might be accompanied by central nervous system complications.

In situations where hyponatraemia is due to SIADH (i.e. in patients with respiratory or central nervous system infections) or iatrogenic fluid overload, management consists of maintenance fluid restriction with or without a loop diuretic. SIADH occurs in the setting of a low Na, low plasma osmolality and euvolaemic clinical picture. This is in contrast to a fluid overloaded patient with a low plasma Na. Note also that in SIADH the urinary Na (> 20 mmol/l) is inappropriately elevated, reflecting renal wasting of Na in the face of a low plasma Na and plasma osmolality. Other exclusions to the diagnosis of SIADH are renal failure, pituitary disease, hypothyroidism, adrenal hypofunction and diuretics.

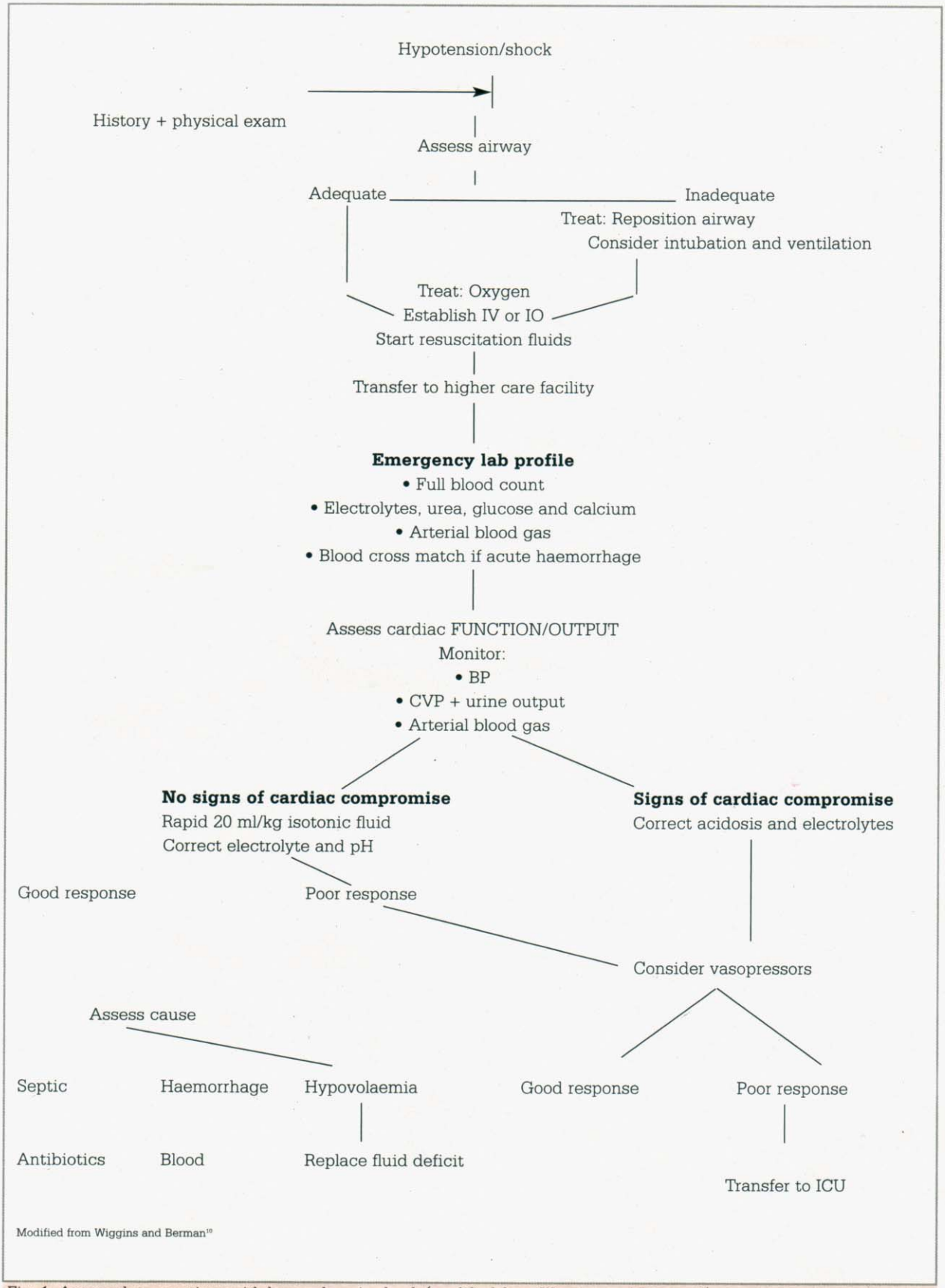

Fig. 1. Approach to a patient with hypovolaemic shock (modified from Wiggins and Berman[10])

Hypernatraemia

Hypernatraemia is defined as a serum sodium level of > 150 mmol/l. It can be euvolaemic or hypovolaemic.

The treatment of hypovolaemic hypernatraemia involves slow rehydration (over 36 - 48 hours) with isotonic fluids and the administration of daily

Table VI. Types of fluids available for paediatric patients

	Non K	Neonatalyte	1/2DD	Paediatric maintenance fluid (PMS)	10% Maintelyte	0.9% N/S	Ringer's
Glucose	10%	10%	5%	5%	10%	-	-
Na^+	33	20	61	35	35	151	131
K^+	-	15	17	12	25	-	5.4
Cl^-	33	21	51	47	65	151	108
Ca^{++}	5	2.5	-	-	-	-	2
Mg^{++}	0.5	-	-	-	2.5	-	-
$PO4^-$	-	3.75	-	-	-	-	-
Lactate	-	20	27	-	-	-	29
HCO_3	-	-	-	-	-	-	-
Osmolality	629	670	434	372	683	308	273

Glucose in %, electrolytes in mmol/l.

be directed at restoring normal circulating volume, cardiac output and organ perfusion.

1. Darrow DC, Pratt EL. Fluid therapy. Relation to tissue composition and the expenditure of water and electrolyte. *JAMA* 1950; **143:** 365.
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$PO4^-$	-	3.75	-	-	-	-	-
Lactate	-	20	27	-	-	-	29
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