Preoperative Evaluation of Electrolyte Status

J. SEDLACEK and N. AZZOPARDI

Summary

The metabolism of Na^+, K^+ and Cl^- and its disturbances described. The importance of monitoring the balance of thse electrolytes stressed. When treating the patient with electrolyte infusions calculation of the amounts given with respect to osmotic state, acid-base balance and the patient's metabolic situation have to be performed.

Key Words
Preoperative evaluation;
Electrolyte status.

It is the anaesthetist's task to prepare the patient for the operation under the best conditions. Preoperative preparation may include many important investigations, but in this paper we look only at the electrolytes Na , K and Cl. . Estimation of these ions is a part of routine examination. We want briefly to draw the attention to some aspects of their evaluation.

One may be misled in trying to evaluate electrolyte concentration if the total solvent volume is not considered:

Total amount of ions = vol. x conc.

Because the total body Na^+, K^+ and Cl^- are difficult to estimate, we normally have to rely on their levels in the plasma.

	Na^+	K^+	Cl
Plasma concentration	138 ± 7mmol/l	4.5 ± 0.7 mmol/l	101 ± 5 mmol/l
Intracellular conc.	3-35 mmol/l	110-150 mmol/l	appr. 30 mmol/l
Total amount in EC fluid	2000 mmol	60 mmol	1400 mmol
Total amount in IC fluid	1200 mmol	3200 mmol	1000 mmol
Daily turn over	140-260 mmol/ 24hrs	50-100 mmol/ 24hrs	140-260 mmol/ 24hrs
Renal excretion	120-240 mmol/ 24hrs	45-90 mmol/ 24hrs	120-240 mmol/ 24hrs

To enable estimation of concentrations and total amounts of these electrolytes we have to measure the intake from diet and/or infusion therapy and the output in the urine and from other body fluids. The normal composition of some of the body fluids is shown in table 1.

The electrolyte levels before the operation should be normalised as much as possible. Special note should be made of an existing catabolic state which may sometimes be present for a long time before operation and may persist after the operation for at least 3 - 7 days. During catabolism 300g of muscle tissue on average is broken down daily. But following injury, operation etc., the loss of tissue can amount to 20 - 30g of nitrogen, or a loss of 1000g of body tissue daily. Even if the weight of the patient is checked daily, it may not be possible to discover this loss immediately because the kidneys are not able to eliminate sufficiently rapidly the overwhelming amount of catabolites. The local oedema due to local hyperosmolality e.g. in the operating field area may mask the muscle tissue break down. The oxidation of broken down tissues leads to the formation of water and this increases its normal daily production from 330 - 380 cc to 600 - 1000cc. From 300g of tissue another 200cc of water is released which had been previously bound in intracellular structures. There is an increased excretion of urea if renal function is normal, but sometimes the urea level in plasma increases in spite of this.

The destruction of cellular proteins leads to release and loss of K from the intracellular space. For every gram of nitrogen lost there is a loss of 2,0 - 3,0 mmol K^+ but this increases fivefold during the postoperative period. However if the urine output is higher than 300cc/24 hrs no dangerous increase in potassium level of the plasma occurs.

The energy consumption of a healthy man is 1500-6000Kcal/24hrs (6300-25100KJ/24hrs). The requirements in operated patients are about 2000-8000Kcal/24hrs (8000-33500KJ/24hrs). This need for energy cannot be satisfied by the infusions of 5% Dextrose frequently used postoperatively! This impaired energy supply situation has a great influence

Dr. J. Sedlacek, MD, Sen. Assistant, Dr. N. Azzopardi, MD, FFRCS, Consultant,
Department of Anaesthesia, St. Luke's Hospital, G'Mangia, Malta.

on the metabolism of electrolytes, especially on potassium.

Sodium is the most important cation in plasma as it regulates the osmotic pressure. The normal osmolality in adults is 285 ± 10 mOsm /l. We can calculate the osmolality approximately from the relation:

Plasma Osmolality in mOsm/l = 1.86 (Na + K conc. in mmol/l) + glucose conc. + urea conc. in mmol/l

But patients in poor physical condition show large discrepancies between their real and estimated values for plasma osmolality.

Hypernatraemia of more than 145 mmol/l can appear if there is: 1. An absolute lack of water (decreased volume of total body fluid). In this situation one simultaneously finds increased haemoglobin and total protein values. The weight of the patient falls and urine hyperosmolality follows. Na^+ values higher than 155mmol/l result in encephalopathy.
2. Relative lack of water: Here one finds a normal haemoglobin level, normal total protein and body weight. Signs of extracellular fluid retention (oedemas) are usually present.
3. Absolute and relative lack of water, where the loss of water is higher than that of sodium.

Increased sodium stores lead to an increase of total body water. This happens after excessive salt intake (NaCl, NaHCO3) or after therapy with sodium salts of some antibiotics (2) following decreased elimination from the kidneys (3) in pituitary hyperfunction (due to ACTH), in hyperaldosteronism, after steroid treatment, diseases leading to oedemas and eclampsia.

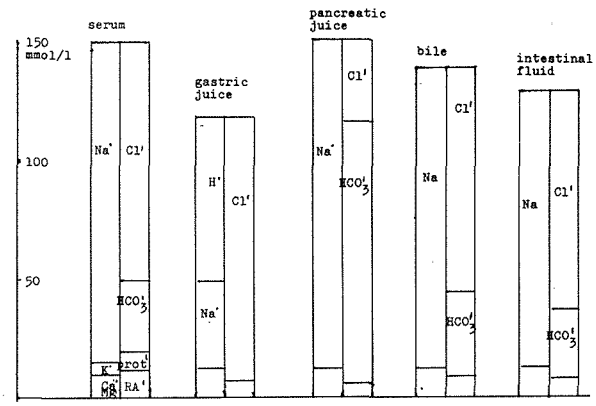
A hyponatraemia lower than 130 mmol/l is usually accompanied by hypoosmolality if there is no simultaneous hyper-glycaemia and/or uraemia. Water intoxication can occur and is manifested by cramps if the sodium decrease happens rapidly. In chronic diseases the sodium falls slowly, parallel to the fall in protein resulting from reduced intake. The body becomes accustomed to the new lower osmolality and any increase in intake of sodium can cause oedema, an increased extracellular space and may lead to circulation overload. Therefore the catabolic state has to be corrected before sodium replacement.

Decreased stores of sodium lead to decreased amount of total body fluid and it can be seen mostly in old cachectic patients. The severe state of these patients is often in contrast with their "normal" values of Na, total protein and haemoglobin. When therapy is begun all these values initially fall and this may falsely give a picture of overloading, but is in fact the result of dilution and the opening of tissue spaces. The loss of Na^+ is accompanied with a loss of anions. Their relation in plasma is:

Na:Cl = 138:101 = 1.37

If the concentration of Na^+ and Cl^- in the urine is in the ranges 1,28 - 1,42 the acid-base balance is not influenced. If the chloride output is lower the body fluids become acidotic and if higher alkalotic.

● table 1.



The relationship $Na^+ : K^+$ concentration in serum is 138 : 4,5 = 30 : 1. In urine it is usually 2 : 1. The renal clearance of Na or K manifest the intensity of the electrolyte elimination by the kidneys. The value of "excretion fraction" (EF) is the amount of the eliminated ions in percentage of its filtrated amount. The renal clearance of Na or K can be calculated from the formula:

$$C_x = \frac{U_x \times V}{P_x}$$

C = clearance, U = concentration in urine in mmol/l, V = amount of urine during 1 min., P = concentration in plasma in mmol/l X = Na or K ion
the value of C_{Na} is 0.5 - 1,0 ml/min. and C_K is 5-15 ml/min.
Excretion fraction of sodium can be calculated from the formula:

$$EF_{Na} = \frac{C_{Na} \times 100}{C_{Cr}}$$

C_{Cr} = clearance of creatinine in ml/min. EF_{Na} is usually in ranges 0,40 - 1,25% and EF_K is 4,0 - 19%
The resorption fraction RF is:

$$RF_X = 100 - EF_X \qquad \text{where X = Na or K}$$

RF_{Na} is 98,75 - 99,6%, RF_K is 81 - 96% normally

From the formula we can see that the value for the excretion fraction can be calculated by means of the concentration indices of electrolyte and endogenous creatinine even without the knowledge of urine output:

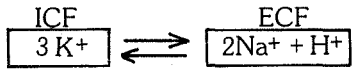
$$EF_{Na} = \frac{\frac{U_{Na}}{P_{Na}} \times 100}{\frac{U_{Cr}}{P_{Cr}}}$$

U_{Cr} = urinary level of creatnine
P_{Cr} = plasma level of creatinine

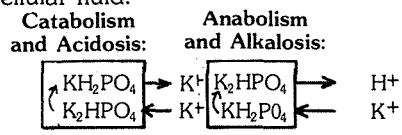
The values of resorption fractions give us the effective resorption of these electrolytes as a percentage of their filtered amounts. Monitoring of these values enables us to check very quickly the elimination of both electrolytes and renal function.

A check of sodium balance may demonstrate sodium retention which leads to an increase of the extracellular space, which can be followed by circulation overload. On the other hand an increased sodium loss leads to a decrease in extra-cellular fluid volume which can be followed by cirulatory and renal insufficiency. Large swings in sodium levels can lead to disturbances in acid-base balance. An evaluation of the ratio of sodium to protein concentration (especially albumin) in plasma gives an indication of the osmotic and oncotic pressures in blood capillaries.

Potassium is the main intracellular cation and is important in maintaining intra cellular osmotic pressure. Its presence in the cells is directly related to the metabolic status of the cell. During catabolism potassium is released from the cells and the cells let in passively sodium and protons:



The decrease of K^+ in the cells could be estimated easily from erythrocytes studies. However the potassium content in these cells is lower than in other tissues. Diabetes, injuries and cachexia are the most common examples of decreased cell potassium levels. The binding capacity for K^+ is dependent especially on hydrogen and phosphate ions. During acidosis there is a shift K_2PO_4 to KH_2PO_4. Therefore in acidosis potassium is released from the cells into the extracellular fluid:



In a similar way potassium is released from the protein bonds. Protein exchanges K^+ for H^+, the K^+ being freed to pass into the ECF.

In the first phase of catabolism there is an increased potassium elimination in the urine from the raised plasma potassium. The plasma potassium level is decreased when the supplies of K^+ in the cells become significantly lowered. On the contrary, anabolism increases not only glycogen supplies but also potassium levels inside the cells. The high concentration gradient for potassium across the cell membrane helps the transpot of K^+ to the extracellular fluid. Vice versa passage of K^+ against the concentration gradients i.e. into the cell, needs energy (sodium pump). This means therefore that the administration of K^+ ions only, by infusion cannot solve a severe decrease of K^+ in the cells as it is only eliminated from the extracellular fluid in urine without passing into the cells. For the good function of the sodium pump which is clearing Na out of the cells and transporting K^+ into the cells the following functions are needed: 1. anabolism 2. energy intake (Dextrose), 3. its utilisation (insulin), 4. potassium intake and 5. increased protein intake. The function of the sodium pump is dependent on the intact cell membrane, on the functioning enzymatic system and on the correct hormonal regulation (hypothalamus, pituitary gland and adrenals).

Hyporkalemia means values of K^+ higher than 5,2 mmol/l. The level of potassium concentration depends on the plasma pH:

pH	level of plasma K conc.
7,1	6,0 ± 0,5 mmol/l
7,3	5,2 ± 0,5 mmol/l
7,4	4,5 ± 0,7 mmol/l
7,5	3,8 ± 0,6 mmol/l

The potassium values obtained while an infusion is still running are often incorrect and therefore such studies are useful only for checking relative changes in the plasma level. The correct value of K^+ can only be obtained 30 min. after the drip has finished.

Hyporkalemia means plasma K^+ lower than 3,8 mmol/l.

The K^+ plasma fall can be very quick and dangerous especially 1. on dextrose intake with insulin during treatment of diabetic ketoacidosis, 2. rapid improvement of chronic hypoxia after inhalation of oxygen, 3. external losses (bleeding, burns) 4. high fluid intake without K^+, etc.

Potassium stores are decreased by catabolism and diuretics. Low potassium level and therefore impaired K^+ gradient across the cell membrane leads to weakness, tiredness, apathy and loss of appetite and paralytic ileus. The cardiac output is decreased and tachycardia with ectopics, atrial fibrilation and impaired conductivity occur. If hypokalaemia is prolonged the microscopic picture shows myocardial necrosis. Digoxin treatment is not effective and makes the arrythmia worse. Therefore during changes from catabolism to anabolism the patients have to receive infusions containing K^+ with frequent checks of their K^+ plasma concentration and the K^+ balance. As long as the K^+ intake is higher than the output (i.e. retention of K^+) the K^+ treatment has to be continued.

The amount of K which should be given to the patient can approximately be calculated from the formula:

mmol K = extracellular volume x (4,5 - plasma level) x 3 + potassium loss during 24 hours

When the improvement of the patients condition is apparent and the urinary output of K^+ is rapidly increasing lower doses can be given.

Potassium treatment is contraindicated during renal insufficiency. The highest concentration in the infusion should be 40 mmol/l, i.e. 3g/1 litre and the maximum rate the infusion should run is 20 mmol/l hr. The potassium losses should be checked especially at the beginning of the treatment because even relatively large deficits (e.g. 30%), the K^+ excretion may not differ too much from the normal valued and plasma K^+ levels have thus to be checked.

Chloride is the main extracellular anion maintaining the acid/base balance and osmotic pressure. It is present in high concentration in gastric juice and less in intestinal fluid. Chlorides are antagonised by bicarbonate ions. Severe vomiting leads usually to alkalosis which decreases the activity of the respiratory centre. The pCO_2 rises and the hypoventilation makes the hypoxia worse. Combined Cl^-, Na^+ and K^+ losses can lead to paralytic ileus. If the chloride level in serum decreases below 96 mmol/l we have to check the output in urine and it is usually very low in these cases and replecement is called for.

Physiological relation is: $(Na + K) : Cl = 1{,}41$

Sometimes we have to monitor also the so called anion gap:

$$(Na + K) - (Cl + HCO^3) = 10 - 18 \text{ mmol/l}$$

Pathological increase of this anion gap occurs during uraemia, diabetic ketoacidosis, fasting, severe dehydration, after diuresis and steroid therapy.

Decreased anion gap may occur during unbalanced i.v. infusion. Monitoring of the anion gap is a useful paramenter in esixtent combined chloropenic metabolic alkalosis with metabolic acidosis due to fasting or hypoxia. This combination of disturbances cannot be detected otherwise.

Hyperchloraemia is present in similar conditions as in hypernatraemia and include severe dehydration after 36 - 38 hours duration (while in children after 12 24 hours), increased chloride intake in renal diseases (sodium is eliminated sufficiently, but chlorides which are in a relatively higher amount of Na^+ are not. Also production of NH_4 and H^+ ions in renal tubuli is decreased), in hypochloraemic acidosis after brain injury (stimulation or damage of hypothalamus), (after a high dose of steroids (by higher losses of Na^+ relative to Cl^+, (severe diarrhoea or intestinal fistula, excessive reabsorption of Cl^-, after ureterocolic anastomisis and (respiratory alkalosis).

Increased stores of chloride are found when the volume of extracellular fluid is increased or in chronic catabolic states. The correction of this hyperchloraemia is harmful. During disturbances of renal excretion, hypothalamic disturbance and after excessive treatment with isotonic saline solution, the stores of Cl^- are increased too.

Decreased stores of chlorides are present after treatment with diuretics, after vomiting, sever sweating, in extracellular fluid deficit, in severe catabolism and in adrenal insufficiency.

Acknowledgements

We would like to express our thanks to Dr. V. Holecek CSc for his assistance and his permission to use the tables from his book, and to Dr. M. Falzon for his help in writing this script.

Literature:

1. Holecek V., Bielik E., Starka L.: Biochemie, Prague 1983, AVICENUM
2. Nejedlý B.: Nvitrni prostredi, Prague 1982, AVICENUM.