RES MEDICA
Journal of the Royal Medical Society

Electrolyte Imbalance

H. A. F. Dudley
Senior Lecturer in Surgery, University of Aberdeen

Abstract
The subject of this paper is Electrolyte Imbalance. Unqualified, such a title may sound like an overambitious attempt to embrace within the span of forty-five minutes the whole gamut of the disordered physiology of the anions and cations. Mindful of the penalty of such vaulting ambition which would, I think, without doubt "fall on the other" its content will be limited largely to some of the more acute and dramatic examples of disturbances of body fluid that are commonly encountered in surgical patients. Surgery deals with the sort of patients of whom John Donne might well have been thinking when he said "this minute I was well and I am ill this minute " and consequently the pattern of electrolyte imbalance encountered by the surgeon is usually drawn in starker and bolder lines than is the more subtle, long drawn out problem with which the physician is often confronted. This makes life considerably easier for the surgeon who is not uncommonly a simple man of action rather than a profound thinker; an extrovert rather than an introvert, a Roman rather than a Greek. Therefore, this account is mainly from a surgeon's point of view with an occasional digression.

ISSN: 2051-7580 (Online) ISSN: 0482-3206 (Print)
Res Medica is published by the Royal Medical Society, 5/5 Bristo Square, Edinburgh, EH8 9AL

Res Medica, Summer 1959, 1(4): 37-44
doi:10.2218/resmedica.v1i4.305

ELECTROLYTE IMBALANCE

Based on a talk given to the Royal Medical Society on Friday, 25th October 1957.

By H. A. F. DUDLEY
Senior Lecturer in Surgery, University of Aberdeen

The subject of this paper is Electrolyte Imbalance. Unqualified, such a title may sound like an overambitious attempt to embrace within the span of forty-five minutes the whole gamut of the disordered physiology of the anions and cations. Mindful of the penalty of such vaulting ambition which would, I think, without doubt " fall on the other " its content will be limited largely to some of the more acute and dramatic examples of disturbances of body fluid that are commonly encountered in surgical patients. Surgery deals with the sort of patients of whom John Donne might well have been thinking when he said " this minute I was well and I am ill this minute " and consequently the pattern of electrolyte imbalance encountered by the surgeon is usually drawn in starker and bolder lines than is the more subtle, long drawn out problem with which the physician is often confronted. This makes life considerably easier for the surgeon who is not uncommonly a simple man of action rather than a profound thinker; an extrovert rather than an introvert, a Roman rather than a Greek. Therefore, this account is mainly from a surgeon's point of view with an occasional digression.

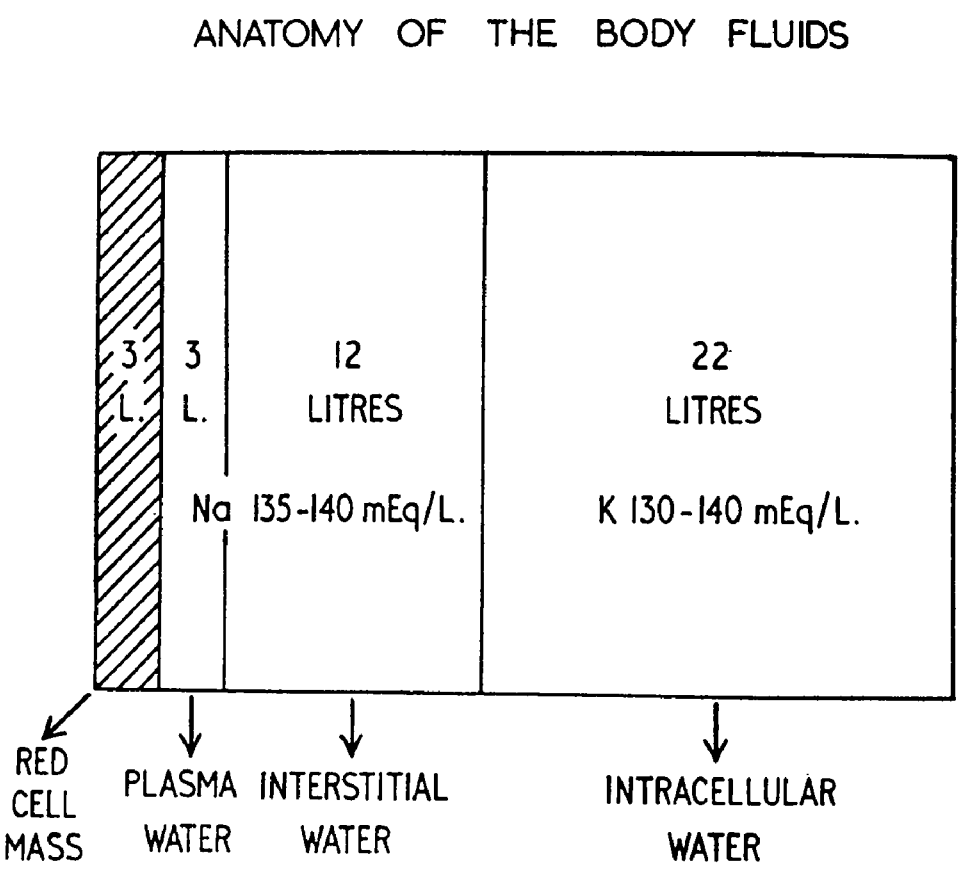
CLINICAL PHYSIOLOGY

Before the subject of electrolyte imbalance in its practical aspects can be considered it is necessary to review, in the words of Humbert Wolfe, our knowledge of " the incessant molecules that bind you," because without some appreciation of the normal anatomy of the body fluids it is difficult to understand in a rational manner the abnormalities which may occur. It is here that the first stumbling block to the study of electrolyte balance is encountered for to many who are neither chemically nor mathematically inclined the nomenclature of fluid and electrolyte anatomy may at first encounter be somewhat disheartening. It should be said at once that milliequivalents represent convenient units of combination and are of great value in the summation of acid and base factors in biological systems. However, to the clinician they are merely a method of expressing results to which too much mystical symbolism need not be attached.

Knowledge of amounts is incomplete without some information on distribution and turnover. In the former case—distribution—we encounter at once one of the great enigmata of cellular physiology—how and why is an apparently normally semipermeable membrance capable of holding apart the two ions sodium and potassium so that the greater part of sodium is outside the cell and the greater part of potassium is within it. Many theories have been developed to explain this phenomenon but none is as yet satisfactory. However, all have in common the concept of the expenditure of energy: indeed it is probably the maintenance of this ionic imbalance at the cell membrane that accounts for the large part of the energy expenditure of the so-called " resting " cell: it is one of the properties of the cell inseparable from life and disappears when the cell dies (Robinson and McCance, 1952).

With this knowledge the body fluids can be arbitrarily divided into extracellular phases (Fig. 1). Water, but not ions, can move freely across the cell

membrane. Ions do cross in small or sometimes in quite large quantities but the factors governing their mass exchange are largely unknown. The extracellular fluid includes, of course, the blood plasma and provides the vehicle by which materials can be transported to and from the cell. The extracellular phase is in direct communication with the gastro-intestinal juices and these and all the other external secretions are largely composed of it. The integrity of the extracellular fluid is dependent upon a proper restriction of sodium and water to this phase within the body; by contrast the intracellular fluid is



built upon a scaffold of potassium ions. However, intracellular fluid is certainly more complex in its structure than the extracellular fluid and the potassium ions are enmeshed—metabolically speaking—with protein and with glycogen. Indeed the interior of the cell is probably not properly a fluid at all, but a thixotropic gel which can change its physiochemical properties as a result of a variety of stimuli.

Finally in this brief review of body fluid metabolism, what of control? All body constituents are in a dynamic state, some coming, some going, none static: even bone apparently so stable, is a vast, active chemical factory. Sodium, potassium and water are constantly entering and leaving the body. For all three, the only usual route of entry is the mouth. Losses of water are about equally divided between insensible pathways (the sweat and the lungs) and the urine. Sodium and potassium losses are predominantly by the urine. The *rates* of urinary excretion are controlled by a complex system of neuro-endocrine servo-mechanisms* based upon the pituitary and the adrenal cortex. For example, cessation of the intake of water or excessive loss stimulates

*A device which maintains a steady state but is susceptible to alteration by outside stimuli.

the production of antidiuretic horomone by the posterior pituitary and so reduces urinary output. Deprivation of sodium stimulates the production of an adrenocortical steroid, aldosterone, and reduces the renal elimination of sodium. At the same time and because of the renal biochemistry involved, the output of potassium tends to rise. However, unlike sodium, diminution of the intake of potassium is not accompanied by much change in the renal elimination of this ion, a fact of considerable importance in management, because if starvation is long-continued, then a severe loss of potassium from the body may occur.

The predominant routes for the exchange of water and electrolytes are the mouth and the kidney. Christopher Wren, by placing in man's hands a hollow needle, has encouraged attempts to bypass these routes by the intravenous administration of all sorts of solutions and although this had undoubtedly saved countless lives since its introduction by a certain Doctor Latta in the city of Leith during a cholera epidemic more than a hundred years ago (Latta, 1832), it enables the physician to flood the body at will and not always wisely with a variety of substances of a nature and in a quantity which may prove embarrassing. The body may treat itself in the same way by bypassing the normal routes of elimination and causing large losses of liquid and ions by the so-called extra-renal routes which are summarised in Table I.

CLINICAL DISORDERS

Simple Deprivation of water

This is probably the most common form of electrolyte imbalance, although not necessarily the commonest in surgical practice. Deprivation of water is the scourge of shipwrecks and life rafts, of desert travel and mountain climbing, of the ark and of the submarine. The early attempts to climb Everest were probably failures not only because of technical problems but also from an inadequate appreciation that man must drink to live and that the gasping respiration of oxygen lack promoted large insensible losses of water from the lungs (Hunt, 1953). Wherever water is in limited supply, water insufficiency may develop. If Alaine Bombard (1953) is to be believed, 50,000 people a year are lost at sea, many because of water deprivation. It only remains to add that a great many people suffer a like fate in hospitals where they are either unable or unwilling to drink or the medical and nursing staff have insufficient time to provide them with water.

When water is withdrawn or lost in excessive amounts, in the sweat for example, then the urine volume falls to a very low level, but insensible water loss continues and the total body water contracts. As this happens, the concentration of electrolytes within the body rises until it reaches a level at which life is no longer possible, and the patient dies of generalised hypertonicity of the body fluids—stewed, as it were, in his own juices. Before this occurrence there is insufficient water to provide for the production of natural secretions and therefore mucous surfaces become cracked like the bed of a dry swamp, and infection with ulceration is inevitable and may contribute to or cause death.

How much loss of water is necessary for this sorry state of affairs? Francis Moore (1952) has pointed out that loss of water is unique in that it requires only a 5 per cent. change in total body water to produce severe effects. At a rate of loss of 2 litres a day, which might be expected in a thirsting man or a patient in a temperate climate, this would take about four days which is, of course, in agreement with all our knowledge of disaster and shipwreck since biblical times. How does the victim look at the end of this time? Not as in the classical description—hollow-eyed and with lax skin. In fact surprisingly

normal and it is only by his restless mien and constant pleading for water and by the dryness of his mucous membranes that it is possible to tell that he is in imminent danger of death. Consciousness is unimpaired and there is usually no alteration in the blood pressure or in the pulse rate. Why is this? The explanation lies in the small percentage loss of water involved—no single body compartment has suffered enough to produce gross haemodynamic or other visible changes. Four to five per cent. of total body water is an insignificant amount quantitatively in terms of changes in the extracellular or plasma volume. If the victim is unconscious or for some other reason unable to complain, then this degree of loss of water may go undetected, a fact that has certainly accounted for the death of many patients in hospital, comatose for reasons such as a head injury. Another striking observation on exsiccated patients is that all the symptoms of thirst and dryness can be relieved very rapidly by the intake of an amount of water far less in total volume than the known deficit (Black *et al.*, 1944). Why it should be necessary only to " prime the pump " is unknown and I commend it to you as a subject for further study.

The prevention of exsiccation rests on the provision of, in temperate climates, an intake of approximately 2-2.5 litres of water daily. In surgical patients less may suffice because oxidation of fat and the destruction of lean tissue may provide a considerable quantity of " endogenous " water for the body's uses, water that is kept within the body by the restriction on urinary excretion that always follows surgical operation and which is the result of prolonged intense secretion of antidiuretic hormone (LeQuesne and Lewis, 1953; Dudley *et al.*, 1954). If under any circumstances water cannot be assimilated by mouth or through a naso-gastric tube then it should be provided by a carefully controlled intravenous infusion of 6 per cent. dextrose which should not under any circumstances exceed the amount of water that is known to have been lost. If excess water is added to the body of surgical patients they are usually quite unable to get rid of it. Patients with too much pure water in the body do not become bloated or oedematous, indeed they have as few signs as the exsiccated patients until quite suddenly the concentration of electrolytes in the body falls too low for normal cellular function and they become comatose, may have convulsions and die—another not infrequent happening when intravenous therapy was not well understood (Zimmermann and Wangensteen, 1952; Wynn and Rob, 1954; LeQuesne, 1954).

Extracellular volume deficit

It has already been remarked that the gastro-intestinal secretions are largely drawn from the extracellular space. Under normal circumstances, these secretions are constantly formed from and returned to the extracellular fluid so that although the total volume of turnover of this fluid is high in any given period, actual net reduction is small at any single instant.

This delicate equilibrium is lost if for any reason such as intestinal obstruction, a fistulous opening, or diarrhoea, gastro-intestinal secretions escape to the exterior. In such circumstances there develops an acute drain upon the extracellular volume, a drain that is qualitative as well as quantitative. Were it merely a matter of loss of water this could be partly made good by transfer across the cellular membrane; but in extracellular deficit it is a fluid rich in sodium that is lost and there is no change in extracellular osmotic pressure to entice water out of the cell. The extracellular volume shrinks and the patient does also. His skin loses its normal turgor, the eyes become shrunken into their sockets, the patient takes on an anxious haggard look. Latta's description of " the sharpened features and sunken eyes and fallen jaw pale and cold bearing the manifest imprint of death's signal " has

never been surpassed. As the extracellular fluid falls so does the plasma volume, and if this latter deficiency becomes acute, circulatory failure or shock may develop. Often, however, it is an operation or some added stress —even standing the patient upright—that unmasks a state of circulatory embarrassment, so that a patient to all intents and purposes well, collapses dramatically and sometimes irreversibly. Such effects develop quickly in a patient losing a litre of stool, gastric aspirate or fistula daily and four days of such loss unreplaced or unchecked may lead to death. It is of some interest to speculate as did the late Hans Zinnser (1936) that this mode of death has probably done more to alter the face of history than all the treaties, pacts and successful and unsuccessful wars for this is the way that patients with typhoid, cholera and bacillary dysentery often succumb. It was extracellular volume deficiency which stopped Napoleon in his Russian campaign—dysentery and cholera with their acute reductions in extracellular volume decimated his forces in six months and reduced them from 500,000 to 80,000 men (Table II). A similar physiological derangement was also rife in the Crimea.

Treatment rests upon two principles, the first of which is the more important. If the body is losing fluid very rapidly it is far better to stop the loss rather than to try to go on replacing it for any length of time. A leak in the bottom of a ship should be repaired rather than reliance placed on the pumps. However, in the emergency, before the loss can be stopped, it is wise, as Andrew Wilkinson first showed, to concentrate on the replacement of circulating plasma volume, particularly if the patient is already in, or verging upon, shock. Such resuscitation is best carried out with blood, plasma or a plasma expander and in acute extracellular volume depletion there is little indication for any other fluid.

Acid-base imbalance

This is a subject to which text-books of physiology give a great deal of attention and space. It was probably, as Marriott has remarked (1947), an unfortunate historical accident that these acid-base considerations were thoroughly investigated before any detailed study of cationic—sodium and potassium—movements proved possible. There is a fruitful field for investigation of the effects of such accidents in the timing of discovery: A. N. Whitehead has touched upon the subject in his considerations of climates of intellectual opinion necessary for the acceptance of ideas, but there is still much to be learnt in organising future research from the more positive aspects of the effect of the acceptance of an idea ultimately proved to be false. Be this as it may, temporal primacy has invested changes in pH and U acid and alkali reserve with a physiological importance which in clinical surgical practice at least they do not possess. Unless some gross metabolic disturbance is present—the commonest is diabetes—alkalosis and acidosis are more usually the secondary result of changes in the intake and output of cations such as sodium and potassium. The occurrence of alkalosis for example in patients who are deficient in potassium probably partly represents the effects of such a cation depletion on the acid base balance at the cell membrane. Attempts to " tailor " the serium electrolytes by the administration of acid ions—the usual thing is ammonium chloride—are unlikely to succeed but energetic potassium therapy alone is not often a failure. The therapeutic corollary is that in clinical acid-base imbalance an underlying cause in either metabolic disorder or in cation loss should first be sought for; unreasoned, meddlesome interference with the serium electrolyte values should be avoided.

Chronic potassium depletion

This is depletion of *intracellular electrolytes* and the circumstance in which

this condition is encountered is in chronic starvation when lean tissue and therefore intracellular electrolytes are lost over a long period of time. The surgeon sees this chiefly in outflow obstruction to the stomach—pyloric stenosis. However, it is characteristic of any state of starvation; had more been known about potassium losses it is possible that many of the concentration camp victims might have been saved because it was undoubtedly the gastro-intestinal atomy of potassium deficiency that rendered them unable to take the food that was proferred them by their liberators. The symptoms of chronic starvation and of potassium depletion are thus almost identical—an apathetic, lisless, weak patient who drops off into sleep or semicoma if his attention is not constantly held and who reacts badly to any form of surgical procedure and often even to attempts to restore a normal intake of food. Such patients require two things—early restoration of a normal diet and rapid repletion of total body potassium which is often reduced to half its normal value. These two principles accepted, they do not often require complex electrolyte therapy, but in the case of surgical patients an expeditious operation and a well-balanced diet. The management of such patients with pyloric stenosis forms a doubly interesting chapter in surgical history. First it indicates that for many years—and indeed still to-day—there can exist confusion of thought about a simple problem, and secondly it demonstrates how therapeutic measures have run through a cycle which has brought the physician back to practices first advocated at the beginning of the century—that is to surgery as the mainstay of treatment. First, the misconception: a patient with pyloric stenosis vomits and his vomit is often dramatically large —1½, 2, 2½ litres in the day. In his own words he " vomits everything doctor." It has long and quite incorrectly been assumed that this represents a net loss of this quantity of gastric juice in the body—that is a loss of extracellular fluid. However, an extracellular fluid loss at this rate is scarcely compatible with life for more than a few days and is certainly not the picture of the patient with stenosis who may go on vomiting for months before coming to hospital in an advanced state of starvation. Before it was appreciated that the patient was in fact drinking to excess in order to absorb enough water across the gastric wall to survive and that the daily reject was in fact part of his daily intake, energetic attempts were made to replace this supposed loss by the continuous intravenous administration of saline, a procedure first cautiously and intelligently introduced in the 1920's by Rudolf Matas (1924). This, because starving patients are for a variety of reasons intolerant of salt water, not uncommonly drowned a patient who might have been saved by a judicious operation. As knowledge increased it came to be realised that this was not a good thing, and that potassium and energy were the chief needs. By far the best way to administer both is by a good mixed diet and this is possible in the patient with pyloric stenosis only if the mechanical defect is corrected. Moynihan and others had advocated this many years before and practised it with success. To-day surgery is once more the mainstay of treatment although on a more rational basis.

This last example may be utilised as an illustration of a general text in clinical surgery or indeed in clinical medicine as a whole. Physiological disorders such as those of electrolyte imbalance develop in the vast majority of cases either because of dysfunction of organs or because of mechanical derangements of some of the intricate control mechanisms of the body. Although it is frequently possible by a sort of inspired biochemical juggling, temporarily to restore normality to the system and such empirical alchemy may be more permanently successful, quite often a lasting result cannot be achieved until a malfunctioning organ works normaly again, a blocked conduit is cleared or an inflamed surface resolves. Fascination with the physico-chemical systems that make up our body fluids should not divert our atten-

tion from the organs which subserve our life processes. Claude Bernard's *milieu intérieur* is as much the liver as the extracellular fluid, the heart as the blood.

Further, in the management of problems of electrolyte imbalance it should be impressed upon all that, if a famous definition of conservation may be freely adapted, " when it is not necessary to interfere it is necessary not to interfere." Some of the disturbances of electrolytes and of water are the result of over-energetic or misguided treatment: the best doctors are those who are not for ever sticking needles into people out of idle curiosity or out of a frenzied desire for therapeutic effort. The hardest thinking in clinical physiological problems must, as Wilkinson has emphasised, be done at the bedside before complex chemical or other analyses are made and the result of such investigations can usually be only confirmatory and rarely of themselves diagnostic.

REFERENCES

Black, D. A. K. McCance, R.A. and Young, W.F. (1944). *J. Physiol., 102,* 406.

Bombard, Alaine (1953). Naufrage Voluntaire. Paris: Editions de Paris.

Dudley, H.A.F., Boling, E.A., LeQuesne, L.P. and Moore, F.D. (1954). *Ann. Surg., 140,* 787.

Elkington, J. R. and Taffel, M. (1942). *J. clin. Invest., 21,* 787.

Hunt, J. (1953). The Ascent of Everest. Appendix VII by Pugh, G. and Ward, M. London: Hodder and Stoughton.

Latta, T. (1832). *Lancet, 2,* 274.

LeQuesne, L. P. (1954). *Lancet, 1,* 172.

LeQuesne, L. P. and Lewis, A. A. G. (1953). *Lancet, 1,* 153.

Marriot, H. L. (1947). American Lecture Series No. 32. Springfield: Charles C. Thomas.

Matas, A. (1924). *Ann. Surg. 79,* 643.

Moore, F. D., Haley, H. B., Bering, E. B. Jr., Brooks, L. and Edelman, I. S. (1952). *Surg. Gynec. Obstet., 95,* 155.

Robinson, J. R. and McCance, R. A. (1952). *Ann. Rev. Physiol., 14,* 115.

Wynn, V. and Rob, C. G. (1954). *Lancet, 1,* 587.

Zimmermann, B. and Wangensteen, O. H. (1952). *Surgery, 31,* 654.

Zinnser, H. (1936). Rats Lice and History. London:

TABLE I

Extrarenal Losses of Fluid and Electrolyte

Nature of Loss	Nature of Fluid	Electrolyte Content
Vomitus	Gastric and small intestinal juices	Weak solution of sodium and water. Only small quantities of potassium
Intestinal fistulae	Small bowel, pancreatic and biliary	High concentration of sodium. Moderate to large potassium content
Diarrhoea	Small and large bowel secretions and exudates	Moderate sodium. High potassium. Exudates rich in potassium
Sweat	Insensible Sensible	Pure water. Weak solution of sodium

TABLE II

Russian Campaign of 1812

Initial strength (June)	450,000
After Battle of Ostrovo (July) Men on sick list	80,000
Beginning of retreat from Moscow (October) Men fit for duty	85,000
Vilna (December) Men fit for duty	20,000

(From de Kerhove and Zinnser)