Hamilton Bailey's

EMERGENCY SURGERY

TENTH EDITION

Edited by

HUGH A. F. DUDLEY

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PREFACE

It is difficult today to capture the exact flavour which has made for the success of Hamilton Bailey's books. Predominantly it was the result of the direct, clinical, pragmatic attack on preoperative and operative problems backed by a vivid style and first class illustrations; also, let it be admitted, there was a touch of the heroic approach to surgery—particularly urgent surgery—which has largely gone from our age. Increasing scientific knowledge attenuates dramatic immediacy and in consequence detracts from the impact of what was originally written primarily for a reader who my editorial predecessor described as 'less experienced, working often alone under conditions which may be far from ideal and who is expected to possess a far wider knowledge of medicine than is the modern specialist'. This book has always tried to regard the interests of such a generalist as paramount, yet we must come to terms with the fact that increasingly the craft of surgery will benefit from advances in the fields of physiology and pathology. In this edition I have endeavoured to blend practice with its underlying theory, but the former does, I hope, predominate and I have tried, as in the past, only to include surgical biological knowledge which is strictly relevant. The radical revision begun by Mr. McNair, which relates mainly to the interaction of surgical science with practice, has been continued, I hope with discrimination, into this edition.

Bailey's books have been traditionally without references to the literature, other than an occasional acknowledgement of a name as a courtesy to the originator of a technique, or a relatively casual reference to the history of an unusual or important event. Probably the time is past that this is appropriate, but we should recognize that references can be grossly overdone, mainly to demonstrate the erudition of the writer. In this edition I have continued the practice of not citing original sources, but all new material is derived either from the personal experiences of the contributors or from proved rather than tentative work in the current surgical literature. Should this text continue to prosper, key references may make their appearance in future editions.

Some re-arrangement of the text was inevitable and the number of chapters has fallen considerably though the size of the volume remains almost unchanged. In addition, I have tried to broaden the base of experience by increasing the amount of material derived from countries and climates other than those in which Hamilton Bailey practised. We have all much to learn from those who work with a different spectrum of disease; this is never more true than now, when the possibility of wide travel has shown many of us how diverse, yet how unified by principle, is the practice of surgery. Finally, in spite of my attempts to increase the range of the work, it does not aim to be encyclopaedic. I believe there is a place for such a catalogue of reference (particularly for abdominal surgery), but Bailey's aim, which Mr McNair pursued, was to distil wisdom from particular example. In so far as this is possible I have continued this course. Every reader will find omissions which he may deplore. Some I have already identified—alas too late. I would appreciate comments on any such gaps.

The book has become a co-operative venture. So radical has the surgery been on the text for this edition that I have felt it proper to ascribe each section to its surgeon. This is not to deny the debt owed to predecessors. I take the opportunity to thank friends and colleagues who have joined with me to produce another edition of a work which, despite its generality in an age of specialization, will, we believe, still have a place.

Wives of surgeons always have a difficult if not an impossible role, but this can be made even more intolerable when editorship is undertaken. In previous editions of this book they have consistently provided secretarial, photographic and other help. My case is no exception and, as with Hamilton Bailey and Tom McNair, I am indebted to my wife for patience, perseverance and encouragement. Miss S. Lind-Jackson has also played the role that any perfect secretary will, by co-ordinating the complex administrative aspects and typing a significant amount of the manuscript.

This edition, as was the previous one, is dedicated to Hamilton Bailey, the original author and editor who did so much to disseminate surgical knowledge to all corners of the world; also now to Tom McNair, who continued in a like way; and, as always, to the surgeons, mostly but not exclusively young, who get up at night, when confidence may be at a low ebb, in order to care for the sick and the hurt.

March 1977

H. A. F. DUDLEY

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Chapter 1

GENERAL BODILY RESPONSES IN INJURY AND ACUTE ILLNESS

D. R. BEVAN AND H. A. F. DUDLEY

The emergency surgeon deals with a patient already beset by general physiological disturbance because of the condition that calls for treatment. Aggravating this, but usually not different in nature, may be the operation that must be performed to relieve the problem. A general understanding of the physiological responses to injury and acute illness is therefore vital to the prosecution of successful emergency surgery, particularly in frail or elderly patients. This is especially so now that much can be offered to the sufferer from a surgical emergency. The emergency surgeon must have the technical skill to meet the occasion, but if he is to be the 'absolute Captain' that Montaigne described, he is also required to have constantly in his mind the hidden changes taking place in bodily economy. Fascination with and facility in technique are not enough; both must be supplemented by physiological understanding.

The neuro-endocrine response to acute illness and injury is now well understood in its broad essentials and particularly in relation to the management of therapy. The first and most acute events—secretion of catecholamines and of antidiuretic hormone (ADH)—have circulatory effects. Both produce vasoconstriction, but the latter also increases the removal of water in the distal tubule and collecting duct, so resulting in a urine of small volume and high concentration—an oliguria that is refractory to the administration of water. The output of catecholamines and ADH is increased in all injury and disease states. Even when the blood volume is not reduced the vasoconstriction may be enough occasionally to raise blood pressure slightly and to produce the pallor one associates with the sick. Increased ADH secretion persists for 24–48 hours and for longer if complications occur, a fact which must be taken into consideration in prescribing fluid replacement. If there is a threat to circulatory stability by reduction in blood volume, the output of catecholamines and ADH is markedly increased with effects which will be considered in more detail when the syndrome of shock is discussed (*see* p. 11).

The mechanisms just described are an immediate consequence of injury. Triggered also at the same time, but of more sluggish response and extending over the first 3 days with a maximum on the second day, is an increased adrenocortical secretion of both cortisol and aldosterone. Once again the effect will be prolonged if recovery is delayed. The main effect of this is to reduce the renal excretion of sodium and increase that of potassium; again, as with water excretion, the restriction is not completely reversible by sodium loading.

Finally, in acute illness and injury of any considerable degree there are changes in endogenous metabolism. Alterations in the secretion of anabolic hormones (insulin, growth hormone) and catabolic hormones (corticosteroids, catecholamines and glucagon) result in a net shift towards catabolism and a diabetic type of carbohydrate and fat metabolism and an increased rate of nitrogen breakdown. Much of the latter is more apparent than real in that it represents the temporary increased nitrogen loss in the urine that occurs in the early stages of acute starvation, but in situations where fever raises the overall level of metabolism or when there is a large mass of nitrogen-containing tissue, such as haematoma, to be broken down large nitrogen losses (of the order of 100 g of protein a day) may occur. Of much greater importance than the increased catabolism is the near-total starvation that tends to accompany

illness and injury. Although man can survive long periods of this (provided that he is healthy to start with), it is not the best way to cope with acute disturbances, and if recovery is delayed by complications then it may rapidly become of importance in its own right. Starvation is the most insidious of surgical complications; seeing the patient from day to day the surgeon may fail to recognize that the flesh is falling from the bones; he may blame the patient's listlessness and low morale on sepsis or psychological disturbance, when all that is needed are two square meals a day, by any route (p. 40).

DISTRIBUTION OF BODY WATER

In a fit, 70-kg male adult approximately 50–60 per cent body weight is water. Two-thirds is intracellular and one-third extracellular. Three litres of the latter is plasma. Increase of body fat results in lower total body water with reference to weight in females (45–55 per cent) and

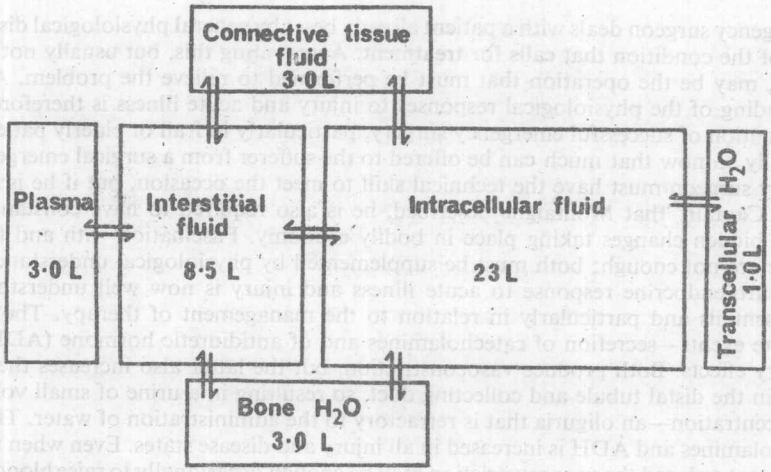

Fig. 1. The distribution of body water.

Table I. Normal values for a 70-kg man

	% *Body wt.*	% *Body water*	*Volume/l*
Plasma	4·5	7·5	3·0
Interstitial fluid	12·0	20·0	8·5
Connective tissue fluid	4·5	7·5	3·0
'Functional' extracellular fluid	21·0	35·0	14·5
Bone water	4·5	7·5	3·0
Transcellular water	1·5	2·5	1·0
Total extracellular fluid	27·0	45·0	18·5
Intracellular fluid	33·0	55·0	23·0
Total body water	60·0	100·0	41·5

the lower fat content of neonates in a higher water content (75 per cent body weight). Plasma volume and total body water can be measured fairly easily. Study of further volumes of distribution depend upon the access of the diluent to various spaces. As access to plasma, interstitial fluid and connective tissue fluid is rapid, this has been called the 'functional' extracellular fluid (ECF). Access to bone water and transcellular fluid (alimentary and glandular secretions, etc.) is slower and more erratic (*Fig.* 1 and *Table I*).

CHANGES IN 'FUNCTIONAL' ECF

In 1961 Shires and his colleagues showed that major surgery was associated with an apparent fall in the 'functional' ECF volume. As a consequence both mineralocorticoid and ADH secretion would be increased and this could then account for the oliguria and sodium retention associated with surgery.

This concept has exercised a significant influence on replacement therapy in the past 15 years. It became fashionable to prescribe large quantities of extracellular-like fluid (normal saline, Ringer lactate) to patients with large injuries or requiring major surgery. Regimens were worked out suggesting certain volumes should be given by the clock or matched to specific amounts of blood loss and replacement. It is not surprising that such practices increased urine volume and sodium excretion over the operative period. However, balance studies show that this is only achieved at the cost of fluid overload which persists for several days. Approximately one-third of this extra fluid is excreted in the urine. It is somewhat surprising that such regimens became so popular because more rigorous measurement of the presumed deficits has not revealed them to exist to anything like the degree already postulated, nor has there been any objective evidence that massive infusion of ECF-type liquids does any good. Furthermore, they may do harm, especially in the elderly, frail and those with cardiopulmonary disease who can be precipitated into pulmonary oedema with some ease.

Nevertheless, it is important to remember that in major trauma large areas of damaged tissue act as a sponge. The ischaemic cells expand because of inability of the cell membrane to sustain the extracellular–intracellular ionic concentrates. These damaged cells are surrounded by oedema fluid. In these situations, then, additional fluid of ECF-like composition should be administered in addition to replacement of blood loss and normal maintenance requirements. During major abdominal surgery Ringer lactate infused at a rate of 5 ml/kg/hour will replace these losses if given in addition to replacement of blood loss.

THERAPEUTICS OF MAINTENANCE

Before he considers water, electrolyte and metabolic disturbances caused by specific lesions, the surgeon must provide for normal requirements against a background of the changes induced by injury which have just been described. Maintenance needs may be summarized as follows.

Water. 1. Insensible losses, lungs and skin: basic 1 litre in the adult, but is increased by fever, environmental heat and low humidity. Those who work in tropical centres know that the normal inhabitants develop considerable adaptation which seems to limit their water losses by comparison with the unacclimatized. However, their resistance to losses is *not* increased. The therapeutic corollary is that insensible losses are more often over- than underestimated except in low-humidity, air-conditioned environments. Insensible loss from the respiratory tract is eliminated in patients receiving ventilator therapy with adequate humidification. Greatly increased losses occur through the denuded areas of burns, for skin is a most effective biological vapour barrier. This matter is considered in Chapter 7.

2. Urine: if injury has occurred the volume of urine is reduced. Solute diuresis may raise it and where large loads of sodium-containing fluids are used or a big load of urea is to be excreted a considerable volume of urine may still be passed. But normally, and provided the kidneys are healthy so that concentration is possible, 500–750 ml per 24 hours is an adequate urine volume to aim for. Attempts in the first 2 days to exceed this by water loading will only reduce the osmotic pressure of the body.

Sodium and Potassium. The recent controversy over effective extracellular volume has already been mentioned (*see above*). Putting this to one side for a moment, the sodium turnover of a man on a normal diet varies between 50 and 100 mmol/day. Injury halves this on the first day and again on the second, so that by the third post-injury day only 10–20 mmol are being excreted. Replacement in excess of this amount will result in the elimination of less than the load, so leading to a positive balance. Small increases in body sodium are of no great

moment, but a significant positive balance can accrue over some days of parenteral therapy and must be guarded against. Therefore, replacement in the healthy person post-injury is limited to 50 mmol/day.

Increased sodium losses via the renal route take place when the kidney is unable to conserve sodium because of disease or old age. A high urine volume after injury is an indication to assess urinary sodium losses by direct measurement, often a more useful manœuvre than measuring serum concentration. Apart from this, all other losses of sodium are extrarenal and abnormal (*see* Acute ECF Deficit, p. 5).

An increased amount of potassium is lost after injury partly because of liberation of the ion from damaged cells, and partly because of the competition between it and sodium for the ion-exchange mechanism in the renal tubule. From a normal loss of 75–100 mmol daily on a mixed diet, the post-injury patient will show a transient peak of 120–140 mmol for a day, followed by a gradual fall back to levels of 40–50 mmol if he is starving or on a minimal carbohydrate intake. For a few days such losses are of no concern, but continued for more than a week they result in the leaching out of 400–500 mmol of potassium and the gradual development of incipient hypokalaemia.

On the basis of the above information the standard water and electrolyte therapy *for maintenance* in the injured or ill adult should be as shown in *Table II*.

Table II. Maintenance water and electrolyte solutions

Solution	*Volume (ml)*	*Sodium (mmol)*	*Potassium (mmol)*	*Calories*
5% dextrose	2000	—	—	400
0·9% sodium chloride (normal saline)	500	75	—	—
Add 6 g potassium chloride*	—	—	80	—
or				
0·18% sodium chloride in 4·3% dextrose	2500	75	—	400
Add 6 g potassium chloride*	—	—	80	—
or				
5% dextrose	2000	—	—	400
Compound sodium lactate (Hartmann's solution)	500	65	2·5	—
Add 6 g potassium chloride*	—	—	80	—

* Omit on first 2 post-injury days.

In planning maintenance therapy it remains to consider calorie/nitrogen requirements. Man at rest expends about 1500–2500 cal/day, an amount that rises by no more than 10 per cent after injury and then only in rare instances (*see* Chapter 4). For a few days a total calorie deficit can be borne without difficulty and, in addition, immediately post-injury the 'set' of metabolic events is catabolic so that attempts to maintain normal intake of foodstuffs, while not necessarily unsuccessful, will be hampered. Thus, in the first 3 days of acute illness or injury, calorie replacement can be limited to that obtained from parenteral water replacement. This is about equal to the amount needed in most circumstances to reduce endogeneous nitrogen catabolism to a minimum. Beyond 3–5 days every effort should be made to restore calorie–nitrogen balance to normal (in calculating this time the surgeon must include not only the period postoperation, but also in emergency situations the days for which the patient has been ill before treatment). Often this will require only the resumption of normal oral intake. However, in the unconscious patient, in obstruction to the upper gastro-intestinal tract, in persistent paralytic intestinal states or in the presence of gastro-intestinal fistulae, other means will have to be sought.

The therapeutics of nutritional maintenance and repletion are considered in detail on p. 40.

ACUTE WATER AND ELECTROLYTE SYNDROMES COMMONLY ENCOUNTERED IN EMERGENCY SURGERY

Water Lack. Withdrawal of water leads to maximum conservation of renal excretion, but insensible losses cannot be reduced and gradually the body dries out until a point is reached where intracellular electrolyte concentration rises too high for normal enzyme function. Death takes place from malfunction of the most sensitive tissue—the brain—usually with the picture of restlessness, coma and hyperpyrexia. If measured, the serum sodium concentration is variable but usually high; renal concentration is shown by the small volume of dark urine of high specific gravity and osmolality. Otherwise the condition is characterized by its lack of signs and should be suspected largely on the analysis of a patient's history of water deprivation. Management is relatively simple—water is replaced by mouth or intravenously in amounts directed towards remoistening mucous membranes, re-establishing urine flow and reducing its concentration to levels of about 500 mmol/kg (SG 1015–1020). It is difficult, though not impossible, to administer too much water, but care must be exercised in post-operative patients whose ability to excrete water is blocked by antidiuretic hormone secretion. Signs of overinfusion of water alone are not easy to detect; water distributes itself over the body water as a whole and does *not* produce crepitations at the lung bases or a raised venous pressure. The features are the reverse of water lack: lassitude, hypothermia, then coma and possibly convulsions.

Acute Extracellular Volume Deficit. This is the syndrome usually called 'dehydration' although the literal meaning of that word is 'water lack'. The pathophysiological events are loss of sodium and water at approximately the concentration that occurs in the ECF so that this, including its intravascular component the plasma volume, shrinks. The route of loss is through some extrarenal portal: vomiting of mixed gastro-intestinal secretions, an intestinal fistula, diarrhoea (*but see* Potassium Deficiency *below*), massive sweating in hot humid environments. Very occasionally the same disorder can be produced in a patient with damaged renal tubules who maintains his ECF economy only by turning over large quantities of sodium a day. Deprived of this intake by the disciplines of a hospital or the rigours of an operation, he may rapidly empty his ECF into the drainage system: patients with obstructive uropathy who undergo emergency decompression are particularly prone to develop this trouble (*see* p. 625).

Table III. Extracellular volume replacement

	Sodium	*Potassium*	*Chloride*	*Calcium*	*Lactate*
Normal saline	150	0	150	—	—
Compound sodium lactate (Hartmann's solution–Ringer lactate)	131	5	111	4	29

(All values in mmol/l.)

All are familiar with the clinical features of acute ECF deficiency. The patient with intestinal obstruction is a typical example: dry tongue (often implying a concomitant water lack) sunken eyes, loose inelastic skin, thready pulse, low blood pressure and scanty urine (the consequence in this instance of poor renal perfusion) are the cardinal features. Examination of the blood will reveal haemoconcentration because of plasma volume shrinkage, but usually a normal or slightly low serum sodium level. If a central venous pressure cannula (p. 30) is in position it will show a low reading.

Therapy. Replacement with saline or modified saline solutions such as Ringer lactate (*see Table III*) is indicated. Usually in acute deficiencies this will be undertaken by the intravenous route. The amount to be given is calculated provisionally from: (1) History of illness; (2) Clinical state of the patient: circulatory instability and oliguria imply a loss of 10 per cent of ECF volume or 2·5 litres in an adult.