THE CONTROL OF WATER AND ELECTROLYTE BALANCE AFTER SURGERY*

BY

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The great majority of patients who undergo a surgical operation are able, within a very short while, to return to a normal intake of food and drink, so that, with regard to fluid balance, they present no problems which demand more than common sense in their management. However, in the minority of patients, particularly those submitted to major surgery on the alimentary tract, care is required if they are to be maintained in fluid and electrolyte balance in the days immediately following operation. The need for this care arises from two causes: first, because such patients are liable to develop complications such as paralytic ileus which can in themselves easily lead to severe electrolyte disturbance; secondly, because of the metabolic changes which follow surgery. This metabolic response to surgery occurs after all operations, indeed, after all forms of trauma. In general its magnitude varies in direct proportion to the magnitude of the trauma, with the result that, whilst it can be neglected after operations of average severity, an understanding and recognition of its effects is vital in the care of those submitted to major operations.

This post-operative response affects many of the metabolic processes of the body, indeed probably more than we are yet aware of, but its most important effects are on the handling by the body of water and electrolytes. Speaking in general terms these effects can be described as an impairment of the kidneys' normal ability to excrete from the body excess loads of water and salt. As a result, if too much fluid is given, it accumulates in the body, leading to salt and/or water retention. Such a retention is, of course, the end result of these metabolic changes but it is by observation of the details of such retentions that we derive our knowledge of the changes themselves. During the past few years my colleague Dr. Lewis and I have carried out a series of balance studies before and after operation which have enabled us to get a reasonably clear picture of these changes. Our results have been published elsewhere (Le Quesne and Lewis, 1953), and the description that follows will largely be based on our observations, which I should point out revealed little that was new. I shall not take up your time now with technical details, but before describing our results one or two points need emphasis. The essential basis of our observations was the

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use of a technique by which we could maintain a constant intake of water and electrolytes for several days before and after operation, the only variation being that after operation, when fluids were given intravenously, the patients received no protein and only 600 cals. per 24 hours. In most cases these patients were given 4 l. of water and 170 m.eq. of Na, that is 10 g. of salt, per 24 hours, and it must be emphasized that this intake was chosen not because it was considered optimal, but to make readily apparent any changes that might occur in water and salt excretion. The majority of the patients studied were undergoing subtotal gastrectomy for benign peptic ulcer. Balance studies were carried out in the usual way and in addition the patients were weighed daily:

it is important to emphasize that on the calorie intake given after operation we would, under normal circumstances, expect these patients to lose weight at the rate of about $\frac{1}{2}$–1 lb. (0.2–0.5 kg.) per 24 hours, so that a stationary or rising weight in these circumstances is highly significant. The metabolic data obtained by these studies has been charted in the usual way, and for those unfamiliar with this type of charting figure 1 shows the principle of the method.

The most immediately striking change found in the patients studied was the marked oliguria occurring in the first 24 hours after operation. This is illustrated by figure 2, which shows the urine output in six cases for three consecutive 24-hour periods, with the operation taking place at the start of the second of these periods.
During each of these 24-hour periods all these patients received 4 l. of fluid, and the profound diminution in urine output following operation is clearly shown. With an intake of 4 l., this diminution in urine volume must lead to a retention of water in the body, and this is confirmed by the finding that all these patients showed a marked gain in weight during this 24-hour period, despite the fact that their calorie intake on this day was quite inadequate. Figure 3 records again the urine output from one of these patients, together with the body weight, which shows a clear-cut gain on the day of operation, followed on the next day, on which a diuresis occurred, by a similar fall. Also shown in this figure is the sodium balance, and it can be seen that, simultaneously with the water retention, on the day of operation there is a distinct retention of sodium, amounting to some 100 m.eq., most of which is excreted on the following day.

In view of the simultaneous occurrence of this sodium and water retention it would be easy to assume that the latter is dependent upon and caused by the former. But in fact this is not so, as it can be clearly shown that this water retention occurs whether or not salt (i.e. sodium) is given on that day. Figure 4 shows the results obtained in two cases who received no sodium on the day of operation, except for that contained in a pint of citrated blood. It can be seen that both were clearly in negative sodium balance on that day, yet both showed the weight gain of water retention, which must therefore be an independent, primary phenomenon. The figure also shows that in both these two cases there was a distinct rise in urine specific gravity on the day of operation, and this rise was one of the most characteristic features of the episode of primary water retention. In all the cases we observed, the diminution of urine flow on the day of operation was accompanied

**Fig. 3**

Sodium balance, urine output and body weight for three consecutive 24-hour periods in a patient undergoing subtotal gastrectomy, and receiving 4 l. of water per 24 hours. The greatly diminished urine flow on the day of operation led to water retention, which is reflected by a weight rise of over 2 kg (4.4 lb.), despite an inadequate calorie intake. Simultaneous with the water retention, there is a considerable retention of sodium.
Data from two cases who received no sodium on the day of operation, except for that in 500 ml. of citrated blood. Both patients were clearly in negative sodium balance during the 24 hours following operation, yet both showed a weight gain due to water retention. Note the rise in urine specific gravity during the 24 hours after operation.

by a marked rise in the urine specific gravity and by a similar rise in the urinary concentration of sodium and chloride, all three of which usually fell to pre-operative levels during the subsequent 24-hour period. These changes in urine composition are well seen in figure 5, which shows the relevant data from the case previously illustrated.

Now these observations based on the analysis of 24-hour pooled specimens of urine necessarily give but a crude idea of the nature and duration of a change such as this. However, more detailed information can be obtained if studies are made on 4-hourly urine specimens obtained from patients requiring an indwelling urethral catheter after operation. These studies, of which figure 6 is a typical example, show that coincident with the start of the operation there is an immediate fall in the rate of urine flow, together with a rise in urine specific gravity and chloride concentration. After an operation of moderate severity, like a sigmoid colectomy, these changes last some 24–36 hours, after which there is a diuresis, and return of specific gravity to normal levels. As has already been shown, this change is independent of the intake of salt, and under
suitable circumstances can lead to considerable retention of water.

This alteration in the excretion of water, this episode which we have called "primary water retention," is but one facet, certainly a very important one, but only one facet of the post-operative metabolic response. The tendency to retain sodium on the day of operation has already been mentioned and there is a further, more prolonged, period of interference with sodium excretion, as shown in figure 7. The retention of sodium on the day of operation is clearly shown, and in all our cases there was a clear-cut retention on this day. However, the predominant phenomenon during this 24-hour period appears to be the episode of "primary water retention" just described, and it is the characteristics of this latter that we see in the urine, with the paradoxical result that this incident of sodium retention is accompanied by a high urine sodium concentration, though low sodium excretion. But on the second, third, and fourth days after operation there is a further period of diminished urinary excretion of sodium, leading to sodium retention and this time accompanied by a lowered concentration of sodium in the urine: at the same time there is a further retention of water, as is shown by the further rise in body weight despite the continuing inadequate calorie intake. This second, more prolonged episode of simultaneous water and sodium retention differs fundamentally from the independent episodes of water and salt retention occurring on the day of operation, in that during this latter period the two are integral parts of one phenomenon, the water retention being caused by the salt retention. Figure 8 shows the results obtained in one of the cases who received no salt after operation, except for that in a bottle of blood during the operation. In this case, in negative sodium balance for 4 days after operation, a second rise in weight did not occur until eventually salt was given, showing conclusively that during the later episode of retention the water retention is a secondary event, dependent upon and integrated

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**FIG. 6**

Results obtained by examination of 4-hourly urine specimens from an indwelling, urethral catheter, in a patient undergoing sigmoid colectomy. Operation (marked by the arrow) is followed by a profound fall in the rate of urine flow, which lasts for 20 hours. This diminution in urine flow is accompanied by a simultaneous rise in urinary specific gravity and chloride concentration.
with the episode of late sodium retention. In further contrast to the events on the day of operation itself this phenomenon of late sodium retention is distinctly variable, in its magnitude, in its duration, which is largely dependent on the severity of the operation, and in the time of its onset. In many of our cases late sodium

![Graph](image)

**Fig. 7**

Balance data from a case undergoing subtotal gastrectomy. On the day of operation there are simultaneous but separable episodes of salt (sodium) and water retention, the effects of the latter being reflected in the weight gain and urine changes. On the second, third, and fourth days after operation there is a further, more prolonged, period of salt (sodium) and water retention, this time accompanied by a lowered urine sodium concentration.
Data from a case receiving no sodium after operation, except for that in 500 ml. of citrated blood. The body weight was not recorded on the morning after surgery, but thereafter there was a steady fall until sodium (salt) was given, showing that during this second period the water retention is dependent upon salt retention.

Retention developed earlier than in the case illustrated, with the result that it tended to merge to a greater or lesser degree with the sodium retention on the day of operation. As is shown in figure 9 this led to varying patterns of sodium retention, which are reflected not only in the sodium balances but also in the urine sodium concentration figures and in the weight curves. It is to be noted that in many cases this leads to a continuous period of salt and water retention lasting for 4–5 days after surgery. During the first 24–36 hours after operation the interference with the excretion of water is the dominant feature, but thereafter the equally important interference with sodium excretion becomes prominent, causing a combined retention of sodium and water. It is the fusion of these phenomena which gives rise to the complex disturbance of fluid balance after operation and to the varying patterns of response which may occur.

So far the only aspects of the post-operative metabolic disturbance to be considered have been the changes in the handling of water and salt, but mention must be made of the increased urinary loss of nitrogen and potassium which occur after operation. For up to 3 days after operation there is a markedly increased loss of nitrogen in the urine: even if an adequate intake of protein is given during these days this loss cannot be reversed and necessarily leads to a negative nitrogen balance. Similarly, during this time there is an increased urinary excretion of potassium, which is relatively in excess of the nitrogen excretion and so must be indicative of a mobilization of intracellular potassium. Again, this cannot be prevented by giving potassium during these days, and this increased urinary loss of potassium during the 24–48 hours immediately following surgery is one of the most important causes of post-operative potassium depletion.

Turning to the aetiology of these metabolic changes after operation it is now apparent that more than one mechanism is at work. It can be said straight away that the changes occurring after operation are not the same as those found in starva-
Data from three cases undergoing subtotal gastrectomy (balances blocked in), showing the varying patterns of sodium retention. In the left-hand case the second, prolonged period of sodium retention is clearly separated from that on the day of operation; in the middle case, whilst not clearly separated, the two episodes are still distinct, but in the third case they have completely coalesced. This variation in time of onset of the late sodium retention is reflected in the curves of urinary sodium concentration, which show their maximum fall on the third, second, and first days after operation respectively. The weight curves also reflect the timing of the later retention.

tion, so that they cannot be explained on the simple grounds of an inadequate calorie and protein intake. It is now equally clear that after operation there is an increased liberation of adrenocortical hormones, which is in great part responsible for these changes (Moore and Ball, 1952). The increased excretion of nitrogen and potassium, the retention of sodium and other changes such as the fall in the eosinophil count are all highly characteristic of an adrenocortical release, and there is overwhelming evidence that they are in fact due to this cause. However, two distinct difficulties arise in accepting the adrenal cortex as the sole cause of the whole post-operative disturbance. First, the sodium retention appears to occur in two distinct waves: there are a number of possible explanations for this, of which the most likely seems to be that the retention on the day of operation is at least in part due to renal haemo-dynamic changes. Secondly, and of far more importance, is the fact that the features of primary water retention in no way resemble the action of
any known adrenocortical hormone: but these features, that is a diminished rate of urine flow with a urine of high specific gravity and chloride concentration, are precisely those produced by secretion of the antidiuretic hormone of the posterior pituitary gland. Recently Dr. Lewis and his colleague Dr. Eisen (Eisen and Lewis, 1954) have shown by bio-assay, in alcohol-anaesthetized rats, that the urine during the first 24 hours after operation contains a strongly antidiuretic substance, which, whilst not definitely identified as A.D.H., has properties which strongly support this hypothesis, and we believe that the interference with water excretion immediately after operation is in fact due to this hormone. There is no evidence that the usual stimulus of hypertonicity is the cause of the post-operative release of antidiuretic hormone, but it is known that pain, emotion, trauma and drugs such as pethidine and morphia can cause such a release and it is probable that a summation of these stimuli accounts for the prolonged secretion occurring after operation. To sum up then, we may say that whilst the adrenal cortex is responsible for many of the metabolic changes after operation, it appears probable that the antidiuretic hormone also plays a significant part, and it may well be that other factors contribute in producing the final, complex pattern of disturbance that has been outlined.

Whilst the exact details of their manifestations and aetiology are complicated and still partly obscure, the clinical significance of these post-operative metabolic changes is clear. In normal circumstances the kidneys are remarkably flexible in action and are capable of excreting from the body, in a short time, excess loads of water and salt. After operation this flexibility is replaced by a temporary rigidity of function, so that excess loads of water and salt cannot be eliminated and must accumulate in the body. I have already mentioned that in the cases we observed the intake was deliberately arranged so that definite retention of salt and water occurred, and I have described the metabolic changes in terms of these episodes of retention. But, of course, such retention after operation is not inevitable. What are inevitable are the metabolic changes themselves, which necessarily follow all operations, and whether or not a retention of salt or water occurs depends entirely on the intake of these substances. Not only is a retention of salt or water after operation not inevitable, but all the evidence suggests that it is undesirable. If only moderate excesses of salt or water are retained little harm can occur, but it is quite easily possible during the first few days after operation to produce serious, even fatal, clinical syndromes by overloading with either salt or water. As has already been described, the impairment of salt excretion lasts for 3 or 4 days after an operation of average severity, and throughout the whole of this time there is a danger of overloading. If such overloading is gross, a well recognized clinical picture develops with oedema formation, waterlogging of the lung bases and even a rise in the jugular venous pressure, but lesser degrees of overloading with salt can equally cause serious trouble owing to oedema formation in wounds, in intestinal suture lines and at the lung bases, all of which can lead to highly undesirable complications. This danger of giving too much salt (saline) has been recognized for many
years, but the risks of excessive administration of water are less well appreciated. During the first 36 hours after operation, when its excretion is impaired, water, usually given intravenously in the form of 5 per cent dextrose, is just as potentially dangerous as saline, and during this period it is quite easily possible to produce acute water intoxication (Le Quesne, 1954). As described recently by Wynn and Rob (1954) more chronic states of water intoxication can occur later in the post-operative period for reasons which are as yet obscure, but undoubtedly the main danger of precipitating this condition occurs during the first 36 hours after operation. Acute water intoxication presents usually with extreme restlessness, disorientation and behaviour disturbances, going on to convulsions, coma and death. In these cases examination of the fluid balance chart will always show a gross excess of intake over output, and as would be expected there is hypotonicity, with a marked fall in the plasma electrolyte concentrations. Gross manifestations of water retention such as this are not common, but, just as with salt, lesser degrees of water retention can be harmful, and probably much post-operative nausea and vomiting is in great part due to mild water overloading.

The problem, then, in the first few days after operation is to provide a basic intake of water and electrolytes which whilst not so small as to cause any depletion, is yet not so great as to allow of any sizeable retention. In those patients who can take fluids by mouth within a few hours of operation, the problem scarcely arises, because their own tastes are usually a safe and sufficient guide. But it cannot be stressed too strongly that no route of administration is immune to the dangers of overloading, and in this respect fluids given per rectum are just as potentially dangerous as those given intravenously. But for obvious reasons the problem demands most serious consideration when fluids are being given intravenously. Bearing in mind that the whole post-operative metabolic response is made up of several separable phenomena, it would be possible to design an intake which varied to meet the changing dangers, but experience shows that this is unnecessarily complicated and only leads to confusion. As will be explained, the markedly impaired ability to excrete water during the first 24–36 hours after operation imposes special restrictions, but otherwise a uniform intake can be given.

Whilst in no way minimizing the dangers of overloading, it should be borne in mind that during the first few days after operation, even in uncomplicated cases, there are likely to be increased losses from the body, both from increase in the cutaneous loss and, in abdominal cases, as a result of some pooling in the intestines from mild, transient ileus. Accordingly, if the basic intake is fixed too low, constant additions may be necessary, thus destroying the essential value of a basic intake which should in itself suffice except in complicated cases. Whilst undoubtedly the main danger after operation is that of overloading, we should not allow this to make us fall over backwards and give too little.

Considering first the water intake, there seems no reason to believe that 2.5–3.0 l. per 24 hours is too much, after the period of so-called "primary water retention" has passed: this allows an adequate margin for the cutaneous loss, and at the same
time provides a generous urine output. However, during the period of primary water retention, that is for the first 24–36 hours, such an intake is undoubtedly too high and will lead to retention, so that during this time the water intake should be restricted to 2 l. per 24 hours. Experiences show it to be safe to increase the intake from 2 l. to 3 l. after 24 hours, and for a while we invariably did this. However, bearing in mind that one of the most characteristic features of primary water retention is a raised urinary specific gravity, we now use this as our guide, and directly the gravity falls below 1010 increase the intake up to 3 l. per 24 hours. In practice there is probably little to choose between these two methods: the former is undoubtedly the simpler and the latter the more elegant, but the important point is that the intake should not exceed 2 l. in the first 24 hours, but thereafter it can be increased up to 3 l. per 24 hours.

Turning to the salt intake, the amount contained in 500 ml. of normal saline, that is 4.5 g. of NaCl or 80 m.eq., constitutes a satisfactory daily intake. Whilst providing a slight margin for any surplus losses there is no reason to believe that this amount can cause any dangerous retention, and it is, in addition, practically a most convenient amount to administer. Considering the first 24 hours after operation, the intake of 2 l. of water and 4.5 g. NaCl is provided by giving three \( \frac{1}{3} \)-l. bottles of 5 per cent. glucose and one of normal saline, and thereafter this can be increased by the addition of two more bottles of 5 per cent glucose, bringing the total intake to 3 l. per day, that is three of the standard \( \frac{3}{4} \)-l. bottles per 12 hours. It is to be noted that in many hospitals the standard bottles of intravenous fluids contain not \( \frac{1}{2} \) l. but one pint, but as the difference between these two is only some 60 ml. it can be neglected. If it is preferred, the salt can be given as 1/5 or 1/3 N saline, but it must be emphasized that such a practice in itself in no way minimizes the risks of excessive administration, and in my experience is more liable to lead to confusion.

Whilst this intake is perfectly adequate in most cases, there is much to be said in favour of adding to it some potassium. Perhaps the most important factor in the development of post-operative potassium deficiency is the increased urinary loss after operation occurring in the absence of a potassium intake. It seems illogical to let this deficit go unchecked and then later in some cases have to treat an established deficiency. There is no certain evidence as to what constitutes the optimal and entirely safe potassium intake in these circumstances, but we have been giving 6g. KCl, that is 80 m.eq. K, per 24 hours without any untoward effects. Owing to the oliguria immediately following operation, potassium should not be given during this period, and it has been our practice to add the potassium at the time the water intake is increased from 2 l. to 3 l. per 24 hours. Rather than give the patient some normal saline followed by a potassium chloride solution, it is simpler to give them together as a potassium-saline mixture. We have been using a mixture containing 2.25 g. NaCl and 3.0 g. KCl in a litre of 5 per cent dextrose, giving a potassium solution of 40 m.eq. per litre, which is the maximum strength which should be used for intravenous administration. Using this mixture, the daily ration of 3 litres of
water, 4.5 g. of salt and 6.0 g. of KCl is provided quite simply by giving each 12 hours two \( \frac{1}{2} \)-l. bottles of the mixture and one \( \frac{1}{2} \)-l. bottle of dextrose, except, as already stated, during the first 24 hours when the potassium is omitted and the water restricted to 2 litres. During the past two years in the Department of Surgical Studies we have now used this basic intake as a routine, and found it to give excellent results in a wide variety of cases. Experience shows that not only is it safe, but that it in fact constitutes a satisfactory basic intake which rarely needs alteration; we have found no complications from the routine use of potassium, except for occasional discomfort, and in our experience this practice virtually abolishes the occurrence of post-operative hypokalaemia. In order to assess its exact results, balance studies have been carried out on some of these patients, and these show that on this intake no significant retention of salt or water takes place, and that the potassium intake likewise does not give rise to danger of intoxication (see figs. 10 and 11).

Although this intake just described does not allow any significant retention to take place, it must be remembered that the post-operative metabolic changes themselves still take place and will lead to certain alterations in the urine which must be recognized for what they are if serious mistakes are to be avoided. For the first 24–36 hours after operation there will necessarily be a diminished urine output, of raised specific gravity: as explained, this is simply a manifestation of the kidneys’ temporary inability to excrete water normally and must not be interpreted as being significant, as it usually is, of water shortage, and so as an indication to increase the water intake. Similarly, during the second to fifth days after operation the urine chloride concentration will be below normal, but again this is simply a manifestation of impaired ability to excrete salt and must not be taken \textit{per se} as an indication to increase the salt intake. The intake described is a fully adequate basic intake and should only be added to if there is definite evidence of abnormal losses, as by vomiting or intestinal suction, and, indeed, in any case of doubt it is probably wiser to give less rather than more fluid.

However, this intake is only designed as a basic intake to replace losses by normal routes, and further problems are raised if abnormal losses do occur. By far the commonest cause of such losses after operation is the development of in-

![Fig. 10](image-url) Sodium balance and weight curve from a patient undergoing gastrectomy, who received the standard intake recommended in the text. The steady fall in weight after operation shows that no significant water retention occurs and the sodium retention is also insignificant.
intestinal obstruction, paralytic ileus, or some form of fistula, such as a biliary, duodenal, or ileal fistula. All these conditions lead to loss from the body of intestinal juices, and a consideration of two simple facts concerning these fluids yields the key to the solution of the problem of the management of these losses. The normal volume of intestinal juices secreted in 24 hours amounts in all to over 8 l. that is more than twice the normal plasma volume (fig. 12). When it is remembered that in cases such as intestinal obstruction an increased quantity of intestinal fluids may be secreted, it is at once apparent that very large quantities of fluid can be lost rapidly from the alimentary tract and its source. 

<table>
<thead>
<tr>
<th>Source</th>
<th>Volume</th>
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<tbody>
<tr>
<td>Saliva</td>
<td>1500 ml</td>
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<tr>
<td>Gastric Secretion</td>
<td>2500 ml</td>
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<tr>
<td>Bile</td>
<td>500 ml</td>
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<tr>
<td>Pancreatic Juice</td>
<td>700 ml</td>
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<tr>
<td>Secretion of Intestinal Mucosa</td>
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<tr>
<td><strong>Total</strong></td>
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<tr>
<td><strong>Normal Plasma Volume</strong></td>
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Fig. 12.
The normal volume of intestinal juices secreted per 24 hours by an adult (from Gamble, 1950). Three obvious criticisms of this simple scheme come to mind, and must be considered, first, that the losses consist, not associated glands, emphasizing the paramount importance of accurate measurement of these losses so that accurate, volume-for-volume replacement can be carried out. Figure 13 shows the composition of these juices and it will be seen that whilst their exact constituents vary, all of these fluids are essentially isotonic, so that they must be replaced not with glucose but with saline. The cardinal rule, then, in the treatment of observed, abnormal losses is first their accurate measurement, and then exact replacement, volume-for-volume with normal saline, this fluid being given in addition to the basic intake.
Fig. 13
Stick-graphs showing the composition of the various intestinal secretions (from Gamble, 1950).

of normal saline, but of fluids containing varying quantities of sodium and chloride; secondly, that in most cases the fluid lost is to some extent diluted by ingested water and therefore is not isotonic but somewhat hypotonic; and thirdly, that the fluids lost contain potassium. In the great majority of cases the fact that the fluids lost contain an excess of either sodium or chloride, and so cause a tendency to either acidosis or alkalosis can be neglected; if the losses are replaced by normal saline the kidneys will excrete whichever ion is in excess, and it is only rarely necessary to use special solutions such as sodium lactate or ammonium chloride to correct an acid-base disturbance. However, the kidneys can perform this function of adjusting the acid-base balance only if they are given adequate water in which to excrete the ions in excess, and this emphasizes the necessity both of replacing the losses accurately and of maintaining carefully the basic intake. If this is not done and only small quantities of urine are formed, then indeed acid-base disturbances may develop. As regards the tonicity of the fluids lost, there is no doubt that in many cases they are definitely hypotonic, but in my experience this is of no significance unless the losses are large, when it becomes necessary to take this into account and to modify not the quantity but the type of fluid given. A useful working rule is that if in any 24-hour period the losses exceed 2 l., then the fifth ½-l. bottle of
replacement fluid should consist of glucose not saline, so as to prevent giving excessive quantities of salt. Turning to the point about potassium, most of the intestinal fluids contain potassium in a concentration two or three times greater than that of plasma, so that losses of these fluids can lead to a deficit of potassium, particularly when these losses are superimposed upon the increased urinary loss of potassium which occurs after operation. As a result, when abnormal losses are large or long continued, it is wise to add potassium salts to the replacement fluid, unless the basic intake contains this ion. Again, a useful working rule is that potassium should be added if the losses in any given 24-hour period exceed 2 l. or if the losses continue for more than 48 hours. In giving such potassium the usual precautions governing the intravenous administration of this ion must, of course, be observed. These may be summarized as follows: the total daily intake should not exceed 80 m.eq.K, that is the amount contained in 6 g. KCl, it should not be given in a concentration in excess of 40 m.eq./l., or at a rate in excess of 20 m.eq./hr. Further, potassium should not be given intravenously unless there is a reasonable urine output and good kidney function, and until considerable experience in its use has been obtained, it is wise to ascertain the serum K concentration before starting administration, and at daily intervals whilst it is being given. To sum up these considerations, we may say that observed losses must be replaced volume-for-volume with normal saline, except when these are severe, when the type not the quantity of the replacement fluid must be altered.

Whilst the replacement of abnormal losses is probably the most frequent problem to be faced after operation, another not uncommon situation demanding equally careful management is provided by the patient with a very low or even absent urine output. Provided the patient was not depleted of salt or water before operation, there are four likely causes of this happening and it is important that in any given case the cause should be accurately diagnosed.

Some months ago I was asked to see a woman who, 28 hours previously, had undergone a Caesarean section. Since the operation she had passed no urine and a provisional diagnosis had been made of acute tubular necrosis. Examination showed an apparently healthy young woman and inquiry showed that in the time since operation her intake had been only 200 ml., and a satisfactory urine flow was rapidly restored by giving a suitable intake. An inadequate intake is the most obvious cause of oliguria after operation and in such cases a careful examination of the intake chart over the preceding 12–24 hours will enable the diagnosis to be made. The obvious treatment in such cases is to increase the fluid intake, and if this results in an adequate urine output the diagnosis is confirmed. However, in many cases the intake has been quite adequate and then other causes must be considered. The most likely reason in these circumstances for an absent or inadequate urine output is that the patient has developed abnormal losses which have not been replaced, and again this diagnosis can usually be reached by a simple clinical examination of the patient, and an inquiry into his progress over the preceding 12–24 hours. If such an inquiry does reveal that
abnormal losses have occurred, then these should be replaced in the manner just described, and once again a return of urine flow will confirm the diagnosis. The problem becomes more difficult when inquiry shows that not only has the intake been adequate, but also that there have been no abnormal losses. This situation usually arises during the first one or two days after operation, and the first possibility to be considered is that the oliguria is but an extreme manifestation of the normal post-operative metabolic response, and that the impairment of the kidney’s ability to excrete water is unusually severe or prolonged. In such cases clinical examination will probably show nothing abnormal, unless sufficient retention has occurred to cause the early signs of acute water intoxication; examination of the urine will show a persistently high specific gravity, probably above 1020, with a high chloride concentration but no albumen or red cells. Once this diagnosis has been made no attempt must be made to force the urine output by increasing the intake, as this can only lead to water retention and possibly intoxication; instead, the intake should be restricted to about 1 l. per 24 hours, sufficient to cover the pulmonary and cutaneous loss, until a diuresis shows that the episode of primary water retention has passed, and a normal intake can again be given.

There remains for consideration the fourth and last common cause of oliguria after operation, namely acute tubular necrosis. This condition is generally considered to cause anuria but it is important to remember that in many cases it may only lead to severe oliguria. In these cases inquiry will probably show that the intake has been adequate and that there have been no abnormal losses, but of greater importance, it will in nearly every case reveal the aetiological incident, which is usually a profound fall in blood pressure during or after operation, or may unhappily be a mis-matched transfusion. The diagnosis will certainly be suggested by a history such as this, and is confirmed by finding in the urine albumen, casts, red cells or blood pigment. The treatment of this serious condition is beyond the scope of this lecture, but it must be emphasized that again no attempt should be made to force a urine flow by increasing the intake. Instead, the intake should immediately be restricted to 1000 ml. per 24 hours and treatment along the lines of the Bull régime instituted.

It is not for me to remind this audience that after operation many other problems can arise in respect of water and electrolyte balance, in particular, the problem of potassium deficiency in patients whose losses have otherwise been adequately replaced. But without a doubt these two problems, namely those of the management of abnormal losses and of an unexpectedly low urine output, constitute the two commonest and most important disturbances of fluid balance which occur in the post-operative period. I hope I have been able to show that in essence the treatment of both these conditions is reasonably simple, though actually at the bedside the circumstances of the case may make their successful control quite difficult, owing to the nature of the patient’s original disease, or to severe shock or other complications and distractions. And always their control is rendered somewhat complex in that it must be carried out against the back-
ground of the metabolic changes occurring after surgery.

In describing the methods by which these two disturbances may be treated, quite deliberately little mention has been made of biochemical investigations of the blood or urine, and emphasis has been laid on simple clinical observations augmented by careful measurement of intake and output. Experience shows that these simple measures, applied with discretion, can be relied upon, and indeed they must form the main supports in treatment during the first few days after operation. Normally when a deficit of water or salt is being replaced, these measures can be checked by simple biochemical tests on the blood and especially the urine. But during the post-operative period the constituents of the urine are powerfully affected by the inevitable post-operative metabolic changes and so will be of little or no guide in the correction of other disturbances. Thus, normally the finding of the urine specific gravity to be above 1020 is, in the absence of glycosuria, practically diagnostic of water deficiency: but in the first 24 hours the urine specific gravity is always, as emphasized previously, at about this level, so that in these circumstances it gives us no guide as to whether the patient is short of water or has an excess in his body. Similarly the urine chloride figure will be of little help for the first three or four days after surgery. This point is well illustrated by figure 14 showing the sodium balance of a patient who developed severe paralytic ileus after a gut resection for strangulated hernia. This ileus led to large losses of fluid by suction from an indwelling gastric tube and these were replaced much in the way just described. It can be seen that on the third and fourth days the urine chloride concentration was nearly zero: at first glance this suggests that the patient was short of salt and that the replacement was inadequate; but in fact we can see that the replacement was excessive and that whilst the urine chloride concentration was at its lowest the patient was in strongly positive sodium balance. In other words,

![Figure 14](Image)

Data from a case developing severe paralytic ileus after operation for strangulated femoral hernia. Operation took place on the day before the balance studies were started. Losses were replaced on a volume-for-volume basis: as the basic intake in this case was too high, this led to excessive administration of salt with considerable retention. Note that on the day of maximum retention the urine chloride concentration was nearly zero. In these circumstances this is not indicative of a shortage of salt, but is a manifestation of the metabolic disturbance following surgery.
in these circumstances the urine chloride concentration is a reflection not of the patient’s state of salt balance, but of the impairment of the kidneys’ ability to excrete salt which always occurs after operation.

This temporary restriction in the methods available for the control of water and electrolyte disturbances emphasizes the necessity for two steps which together form the essential basis of the successful management of fluid problems after operation. First, if at all possible, any pre-existing electrolyte disturbance must be corrected before operation. In any circumstances the replacement of pre-existing losses is always more difficult than that of observed losses, and the problem becomes doubly difficult in the post-operative period when some of the essential guides to this process are rendered valueless. Secondly, all fluids given to the patient and all fluids lost by the patient must be accurately charted: the information thus provided, together with an assessment of the patient’s insensible loss, will always enable a reasonable estimate to be made of the patient’s state of fluid balance. No single step in the care of the patient is more important than the accurate prescribing and administration of the intake, and the careful measurement and recording of the output. Many of the complications which occur after surgical operations could be avoided by this simple step, which also provides the essential information required in the control of unavoidable complications. I must apologize for ending this lecture by stressing such a mundane point as the scrupulous keeping of fluid balance charts, but it is perhaps as well in that it reminds us that despite all the apparent complexities of the subject we are in fact only dealing with comparatively simple volume problems.

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