PRE- AND POST-OPERATIVE MANAGEMENT OF SURGICAL CASES.*

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Lecture I.

For many years surgeons have concentrated on making surgery safe for the patient and at the present time I think we can claim to have attained this end so far as the majority of patients is concerned. If at operation anatomical details are respected, all bleeding is arrested, infection is excluded, the tissues are handled gently and prevented from drying, surgery entails very little risk for most patients. It is quite different in the case of patients, who may be dangerously upset in their biochemical and metabolic arrangements by fatigue, undernourishment, chronic anæmia and in the later stages of most of the acute surgical emergencies.

It is to the pre-operative preparation of this type of case I intend devoting my attention for the most part, that is to say, to making the patient safe for surgery.

PHYSIOLOGICAL CONSIDERATIONS.

In order to understand the reasons for the therapeutic remedies to be discussed later it is necessary, in the first place, to say something briefly and broadly about the internal environment. The body temperature and the fluid content of the body must be kept within reasonably narrow, optimal limits, as marked or prolonged deviation in the constancy of either inhibits metabolism and ultimately causes cellular death.

Herein lies the essential principle underlying pre-operative preparation of the type of patient I am considering, because several factors may be present tending to alter this environment, the chief of which are dehydration, anæmia, fever and defects in the organs of elimination. However skilful a surgeon may be in operative technique he will not save his patient, if he does not appreciate and rectify these disturbing agents before operation.

Fluid Balance.

The body contains 60 per cent. of water, most of which is in the body cells. It is possible to exist for long periods without food, but deprivation of all water is followed by signs of dehydration in two or three days and soon terminates in death.

Fluid loss. Water is being lost continuously day and night by three routes:—

1. *Lungs—some 500 c.c.*
2. *Insensible loss from the skin—around 1,000 c.c.*

These two constitute an important avenue of loss for two reasons. First, they play a very essential part in temperature regulation and second, as the loss is not seen it is easily overlooked in a clinical estimation of water loss. Nearly half the water is lost in these two ways and therefore if forgotten a serious omission is committed.

3. *Kidneys—roughly 1,500 c.c.*

Some 200 c.c. of water are lost in the fæces, but this is a negligible factor.

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Fluid intake. Normally all fluid is taken in by mouth as food or drink, and is, therefore, easily estimated. An additional supply of about 500 c.c. is derived from the oxidation of carbohydrates and fats.

In quiet conditions fluid balance is thus maintained, but during muscular exertion, excitement, high external temperature and abnormal states of fever much larger quantities of fluid will be lost by sweating and increased respiratory excursions. In these circumstances the kidneys excrete a concentrated urine in order to conserve water for the important temperature regulating mechanism. Naturally the call for water is remedied by increased intake invoked by thirst and balance becomes restored.

It should be remembered that sweat contains 0.2 per cent. sodium chloride and during heavy sweating quite a big loss may occur. The importance of this lies in the fact that sodium plays the chief part in maintaining extra-cellular osmotic pressure. Chloride does not as it permeates the cell membrane quite readily; however, excessive loss upsets the acid-base balance in the direction of alkalæmia since sodium unites with bicarbonate if there is not sufficient chloride.

Acid-base Balance.

This is an exceedingly complex subject, but of great importance in surgery and it is necessary to understand the broader aspects. The normal hydrogen-ion concentration (pH 7.4) is maintained by the constant relationship of the bicarbonate ion to the carbonic acid. When acid is added to blood it goes into combination as carbonate salts; the more carbonate formed in this way the less is available for combination with carbonic acid as bicarbonate; consequently the more acid the blood becomes. The normal limits set to this plasma carbon-dioxide combining power are 53 to 75 c.c. of carbon-dioxide gas to 100 c.c. of blood plasma. Beyond these limits an uncompensated position may arise and if not quickly rectified, especially at the acid end, death will ensue. There is a fairly easy method of estimating this combining power.

The concentration of free carbonic acid is controlled by the respiratory centre. This is a very efficient control, so that dislocations in the direction of acidosis or alkalosis are almost always due to changes in the bicarbonate ion, either excess or deficit. Primary alkali excess causes an increase in blood bicarbonate and hydrogen-ion concentration and ultimately tetany may develop. It may arise from alkali retention or acid loss. In the first case it may be due to intravenous alkali injections, such as may be given in an attempt to counteract pre-existing acidosis, or it may be due to the intensive alkali treatment of gastric and duodenal ulcers.

When due to acid loss, it may be brought about by the vomiting of high obstruction such as occurs with pyloric obstruction, or even by prolonged and frequent gastric lavage. The chloride removed from the blood is replaced by bicarbonate, and a high and sometimes acute alkalosis develops.

The most important clinical variety of acid-base abnormality is due to primary alkali deficit, which comes about through alkali loss or acid retention. Acidosis resulting from decrease in bicarbonates may be due to excessive elimination of these by the kidneys or bowels. Since conditions associated with diarrhoea and intestinal fistulae carry off much of the alkali secreted into the upper part of the intestine which would normally be reabsorbed, depletion of blood alkali may become severe. In certain degenerative conditions of the kidneys chlorides may be retained, as they are also in the depressed function caused by blood concentration. Perhaps the most serious cases from the surgical point of view, are those in which alkali deficit

is caused by retention of ketone acids derived from fats and due to lack of carbohydrate or inadequate oxidation owing to insufficient insulin.

Carbohydrate is essential for the complete combustion of fats, and if there is a shortage in the diet the body sacrifices protein, from which sugar can be obtained. There is, therefore, an increase of nitrogenous waste products in the blood as well as displacement of base from combination with carbonic acid, thus producing acidosis. Carbohydrate may be insufficient for three reasons: shortage in oxogenous supply; deficient endogenous stores; and impairment or loss of ability to oxidise sugar. We shall see the practical importance of this later. The constant symptoms of alkali deficit are hyperpnœa, dehydration and tachycardia. In the earlier stages hyperpnœa may be overlooked since it is deep rather than rapid breathing. Later headache, nausea, vomiting and dyspnœa appear, and at this time the alkali reserve is seriously depleted. The kidneys respond by a greatly increased excretion of water, if such is available, to carry off the acids, and by producing ammonia; they can only do so if they are functioning well.

Tissue Respiration.

To utilise the nourishment brought to them the cells need oxygen; this, and the gaseous waste after combustion, is transported by the blood and across the extracellular spaces. The site of interchange between blood and extracellular fluid is in the capillary bed, and can only continue when the peripheral circulation is efficiently conducted. I cannot enter into details of blood pressure and other factors which are responsible for circulatory efficiency, but will direct your attention to two aspects of the matter from the point of view of pre-operative preparation, viz. blood volume and composition of the blood.

Diminution in *blood volume* would, in the absence of adjustments, mean a corresponding fall in blood pressure. The compensatory mechanisms involved are increased heart rate to make up for the decreased output per beat, peripheral vasoconstriction to accommodate the vessels to their contents, together with a rapid flow of fluid from the extra-cellular, and ultimately from the intra-cellular reservoirs.

If the cause of lowered blood volume be hæmorrhage the vessels become filled for the time being with watery blood, when the volume may be quickly restored but with a low hæmoglobin content. If the cause be loss of fluid other than whole blood the hæmoglobin content may be raised, and the flow of fluid into the vessels ineffective. This occurs in shock, where there is actual concentration of blood, and volume remains diminished. Here vaso-constriction increases in a further attempt at accommodation with the result that peripheral circulation becomes more and more difficult. In the case of hæmorrhage, tissue respiration suffers from shortage of corpuscular elements; in the case of loss of fluid other than whole blood, interference with tissue respiration occurs because of stagnation of the viscid, concentrated blood in the capillary bed. In hæmorrhage if no more than 25 per cent. of the total blood be lost and the bleeding has ceased, the reparative processes are quite able to completely restore the pre-hæmorrhage state; on the other hand, shock is apt to pass into a position in which a whole set of factors are tending to prevent the return to normal. These factors are falling blood pressure associated with increasing vaso-constriction which may extend to the veins causing further stagnation and concentration of blood in the capillaries; the capillaries are more permeable in shock for some reason and are more damaged as time goes on in this respect by the increasing acidity of accumulating metabolites; plasma and blood proteins may escape and still further enhance the flow of fluid away from the blood.

Dehydration is increased by copious sweating so commonly seen in shock. Once these processes have become established there is little chance of rectifying things, the patient dying in peripheral circulatory failure. Acute secondary shock is not infrequently seen in surgical practice, but I want to emphasise that chronic debilitating influences such as anæmia, toxæmia, undernourishment, etc., ultimately produce the same effect in a partially compensated form owing to the slower onset, which the trauma of operation may readily precipitate into the acute stage.

This becomes clear when we consider what are the effects produced at and after operation. For several hours before operation the patient's intake of food is stopped and his fluids restricted, and he may have been purged. During operation he loses a certain amount of blood, and frequently especially under ether anæsthesia, a large amount of fluid by sweating and deep breathing; moreover evaporation from exposed viscera leads to some loss of water. After operation the patient is made warm in bed and may sweat freely; he may vomit and possibly has a tube inserted carrying off much protein and fluid where there is suppuration, or a large quantity of fluid, e.g. as much as 1,500 c.c. in 24 hours with a tube in the common bile duct. To some extent the kidneys conserve fluid, but if carried too far this results in the retention of nitrogen in the blood. Again for some hours after operation fluids perhaps cannot be taken or may be prohibited and should there be any rise in temperature water is used to aid in dissipating the heat. It is quite usual for a patient to have his fluid content depleted in these various ways by as much as 1,500 c.c. and in less common circumstances by a great deal more.

There is, therefore, a threat of dehydration after any operation with all its evil consequences. Ordinarily this is not serious, but in a patient already suffering from water loss it is impossible to exaggerate its seriousness. It is a great deal easier to prevent such things happening than to put them right when they have become well established.

Causes of Abnormal Fluid Loss.

1. *Hæmorrhage.*
 2. *Shock.*
 3. *Loss of fluid by the intestinal tract.*
- } These have been considered already.

It should be kept in mind that a huge amount of fluid—amounting to some 10 litres—is poured out into the intestinal tract as secretions every day which is reabsorbed by the cæcum and proximal half of the colon except for about 100 c.c. which escapes with the fæces. With occlusion of the outlet of the stomach, as occurs in congenital pyloric stenosis, stenosis following on pyloric ulceration or malignant disease, most of the gastric secretion may be vomited. The result is the body loses a large quantity of water and chloride and a smaller quantity of sodium and dehydration with alkalosis develops. Duodenal fistula allows of the loss of highly alkaline pancreatic juice, mainly base-bicarbonate and water; and dehydration and acidosis follows.

Bile is only mildly alkaline, and it is clinically evident that all the bile may be drained to the surface for long periods without serious effects, provided the patient takes plenty of water. All the same, much water is lost through the tube and nearly 90 per cent. of fat is passed undigested in the stools.

Fistulæ in the ileum cause dehydration, but may cause little or no disturbance of acid-base balance owing to neutralisation of the intestinal contents; the same is true of severe diarrhœa. On the other hand acidosis or alkalosis may develop.

Acute intestinal obstruction, especially of the small intestine, produces dehydration, and depending on the position of the obstruction, some acid-base imbalance. Even in the absence of vomiting there is loss of water to the system, since the fluid accumulates in the gut because the vascular derangements caused by the obstruction prevent reabsorption. The same applies to paralytic ileus.

4. *Fluid may be lost through metabolic disorders.*

In surgical practice diabetes mellitus is a good example. Large quantities of water pass out *via* the kidneys to carry off the salts formed by the union of ketones and base. There are probably other reasons which we do not understand, which help to account for the dehydration associated with diabetes, a feature of the disease of the highest importance from the surgical point of view.

Protein loss in burns and long continued suppuration derange water balance. In the first, shock is often associated and therefore dehydration appears; in the latter, water may be retained in the tissues, causing oedema; this is because the normal protein of the plasma of about 7 per cent. is reduced maybe by half, and the osmotic influence is largely lost.

It is worth noting that conditions apparently so unlike as hæmorrhage, shock, acute intestinal obstruction, pyloric stenosis, obstructive jaundice and diabetic gangrene with coma, have fundamentally the same constitutional effect, and require the same general treatment before the very different specific operative surgery can be safely instituted. In surgical practice I feel sure dehydration is the really important factor in initiating the bio-chemical disturbances we have been considering, and it is our business as surgeons to restore fluid balance, so far as possible, before operation. It is essential, therefore, that we should recognise the position, and be able to form some idea as to the degree of water loss.

The advanced stages of dehydration are easily recognised—dry tongue, dry furred teeth, sunken eyeballs, dry inelastic skin, thirst and scanty urine; but serious water loss may be present long before these signs are apparent. The flow of water occurs between the blood, intra and extra-cellular spaces, and though, as dehydration continues, the intracellular water is less readily yielded up, nevertheless it is safe to assume that in most cases they are all depleted roughly to the same extent. It is impossible to determine the water content of the cells and extracellular spaces, but that of the blood is not so difficult, and measurement here may be used as a rough guide as to what is happening in the other places mentioned.

Blood Indications.

Total blood volume can be measured, but for practical purposes it is too long and difficult a procedure. I have already said dehydration causes concentration of the blood, so that a fair index of the patient's state may be gathered from an estimation of the rise in total cell count and the hæmoglobin content. In hæmorrhage, of course, there is a fall.

The plasma chloride content is depleted in excessive vomiting; normally it should range around 350 mgms. per cent.; if the fall reaches 250 mgms. or less the condition should be considered critical.

The measurement of the carbon-dioxide combining power of the plasma helps in estimating acid-base balance and indirectly indicates water disturbance, if it differs much from the normal 60 c.c. per cent. with rather wide limits especially on the alkaline side. Very low figures may be found in diabetics indicating serious acidosis.

A simple method of determining dehydration is to estimate the output of the urine and its specific gravity. Dehydration is not serious if the urine totals 1,000 c.c. in 24 hours and has a specific gravity of 1.020 to 1.030. 600 c.c. or less of urine of high specific gravity, in the absence of kidney disease, indicates water shortage and in such a case nitrogen retention will occur and be demonstrable as a rise in the blood from the normal of 20 to 30 mgms. per cent.; a rise to 40 or 60 mgms. or more indicates dehydration.

Daily estimations of urinary output and specific gravity together with daily estimations of blood urea and hæmoglobin values will give reasonably reliable information concerning the degree of dehydration and concerning its improvement or otherwise under treatment.

PRACTICAL APPLICATIONS.

Believing, as I do, that the problem confronting us is mainly one of fluid requirement, we have to determine how much fluid to give, in what form, and the best method of introducing it. As to the amount we have to take into consideration two groups of patients.—

1. Those who show no particular evidence of water shortage before operation, but in whom there will be certainly some loss at and after operation for reasons already stated.
2. Those dehydrated by their disease, and who will also lose water later for the same reasons as those in group 1.

According to F. A. Coller and W. G. Maddock (1933) patients in group one will lose some 1,000 c.c. at operation and in the ensuing 24 hours after an operation of average severity. They will, therefore need this amount of fluid in addition to their maintenance allowance of around 3,500 c.c. in the first 24 hours. Good kidneys can manage on less than the 1,500 c.c. of water allowed in this figure, but it is as well to budget for this amount, since the daily waste products of about 35 grms. cannot be eliminated with less if the kidneys happen to be functioning inefficiently and in any case there will be a rise over the normal waste product figure as a result of the operation.

Generally speaking patients in group one can look after their own water requirements, if allowed to do so, by means of the sensation of thirst. But nausea and vomiting may interfere with intake by the natural channel; then fluid must be given parenterally and the proper amount can be given only if the daily loss is approximately known. If the patient is taking some water by mouth, the parenterally administered fluid should be 4,500 c.c. less this amount; where abnormal loss is occurring, such loss should be added to the total requirement.

In group 2 the amount of fluid sufficient to replace the ordinary losses is not enough; they require as much more as is necessary to make up for what has been lost previously. It is not easy to assess this, though a rough estimation can be made by using the data previously described. According to Coller & Maddock, if a patient shows definite signs of dehydration, he will have lost in water some 6 per cent. of his body weight. If he weighs 10 st. he would, therefore, need some 3,500 c.c. over and above his maintenance requirement of 3,500 c.c., that is a total of 7,000 c.c. This is a lot of water, and it is desirable, especially in elderly patients, to give it slowly, allowing several days for the process of rehydration.

It must be kept in mind in treating this kind of patient that there is competition, so to speak, for water between the tissues and the kidneys. It is an unequal competition inasmuch as the kidneys only get what the temperature regulating mechanism allows them, and since every gramme of waste needs so much water for its removal by the kidneys, as dehydration continues retention products eventually occur in the blood. If this state of affairs continues long the kidneys become seriously damaged as is shown by the presence of albumen, casts and red cells in the urine.

It is for this reason so much more fluid than normal maintenance quantity is needed by this type of patient, and it is also why so much fluid can be given in the first place without any effect on the urinary output. Once urinary excretion rises to 800 c.c. or more we can be sure the fluid reservoirs are being satisfactorily filled and the fluid intake can be gradually reduced.

THE FORM IN WHICH FLUID SHOULD BE GIVEN.

This hardly arises when nourishment is being taken by mouth, but when administered parenterally whatever fluid is given should, generally speaking, be iso-tonic. If it is less it will for the most part be excreted, since the body will not retain water without salt; moreover, intravenously, distilled water might lake the red cells. If it is hypertonic it will intensify dehydration by osmosis. For dehydrated patients, therefore, this type of fluid is inadmissible.

The fluids usually employed, when given parenterally, are normal saline and/or 5 per cent. glucose, Ringer-Locke's solution and Hartman's solution. I have tried all these and cannot convince myself that the last two have any advantage over the others. Theoretically they should, because of the additional salts they contain, but in practice I think not, and probably for the reason that it is water which is really needed. One must remember that in giving large quantities of normal saline a great deal more salt is administered than is required whereby much harm may be done. The average daily intake of NaCl is about 15 grms.; much of this is excreted by the kidneys. Infusions of 4,000 c.c. (not an unusual amount) would mean giving twice as much salt as this and would seriously embarrass the kidneys which may already be working at their maximum capacity; the body can actually manage with as little as 5 grms. or less in 24 hours.

Where a litre or two is administered normal saline is quite satisfactory, but for large amounts the fluid should be changed. I used to give all normal saline and frequently saw cedema develop with great and rapid increase in weight. Cedema appears in the limbs and lungs, the latter being a serious embarrassment to the patient and may prove fatal. Now I give saline with glucose in the proportion of 1 part normal saline to 4 parts 5 per cent. glucose—or normal saline up to 2 litres and thereafter 5 per cent. glucose only. I think on the whole the former is the better method.

Of course, if salt has been lost through excessive vomiting, sweating or severe intestinal loss more saline should be given and the extent to which it is needed checked by estimating the plasma chlorides.

Hypertonic solutions induce dehydration; this is because water is drawn from the tissues in order to dilute them for kidney excretion. These solutions, therefore, are useful in conditions such as traumatic cerebral cedema and post-operative pulmonary cedema. If they are to be of use in these conditions they have to be repeated and the effect carefully watched.

Another use for hypertonic saline is in paralytic ileus, where the osmotic effect tends to take water out of the intestine thereby producing some rectification of dehydration and giving relief to the gut hampered by its excessive contents.

Water without salt usually causes marked diuresis; the patient loses some of the salt from his tissues at the same time which eventually upsets his body salt-water ratio and dehydration results. However for some reason which is difficult to understand, a patient given large quantities of water without salt may develop water intoxication, of which twitching, convulsions and unconsciousness are the chief manifestations. These symptoms are undoubtedly due to cerebral oedema. but it is not clear why water intoxication should occur in some cases and not in others. Possibly it has something to do with hypersecretion of posterior pituitary extract, which, as in diabetes insipidus, causes a marked reabsorption of water by the kidney tubules.

Glucose.

In iso-tonic strength, viz., 5 per cent., glucose can always be given parenterally with advantage to the kind of patient we are considering. In fact it should always be added to whatever fluid the patient is having. It is readily oxidised by the tissues and prevents or rectifies ketosis. When given intravenously it will provoke diuresis, because as the sugar is used up distilled water remains, which cannot be entirely retained. Alone, therefore, it will tend to dehydrate and should be used in the later stages of rehabilitation after rehydration has been achieved; at this time a copious urinary excretion is very beneficial.

Glucose should not be given faster or in greater quantity than it can be utilised. The minimum daily intake of carbohydrate which can be allowed with safety is about 240 grms. of glucose; at the rate of 1 drop per second of 5 per cent. glucose the patient would be getting about 250 grms. in 24 hours. According to Woodyatt, Sansun & Wilder (1915) a normal man can utilise 1,260 c.c. per hour of 5 per cent. glucose given intravenously, which is rather more than 1,000 grms. of glucose in 24 hours. This allows a big margin, and in certain circumstances one can take advantage of this to give glucose in greater strength than 5 per cent. All the same, unless the solution be given very slowly, even at 5 per cent. strength glycosuria may occur.

Nowadays glucose is almost invariably used in parenterally administered fluid, and for very good reasons. It is absorbed as such and is completely metabolised to CO₂ and water in the presence of insulin and this puts no strain on the excretory organs; it is an essential foodstuff and, unlike other foods, can be given easily and safely into the blood stream. When given in sufficient quantities it is stored as glycogen in the liver, and thereby protects this organ against hepatic irritants. It is a protein saver, and necessary for the complete combustion of fat, thus preventing ketosis. It promotes diuresis, and possibly through its effect on the liver inhibits the tendency towards hæmorrhage in patients with obstructive jaundice, and it is also said to have a good influence on the cardiac musculature.

For dehydrated patients, therefore, the following measures are generally effective:—

1. Water to rectify pre-existing dehydration, and to anticipate a threatened further dehydration.
2. Sodium chloride to replace salt loss—especially the chloride factor, and to promote retention of water.

3. Glucose to prevent or rectify ketosis, and to induce diuresis. Sometimes a further measure is necessary, viz., blood transfusion especially for hæmorrhage, which has occurred or is likely to occur, shock and protein loss. Not only does it directly replace the lost constituents, but overcomes the damage caused by anoxia of the tissues generally.

METHODS OF ADMINISTERING FLUIDS.

By mouth. This is, of course, the route which should be chosen when vomiting or some other reason does not prevent it. Beside being the normal channel, and so best able to deal with absorption and so on, it has the advantage that all types of nourishment can be administered and whatever medicines which may be required; also problems of excessive fluid and complications associated with parenteral introduction do not arise.

However, circumstances may compel the use of other channels. Of these there are three—intravenous, rectal and subcutaneous. I have discarded the latter except occasionally in small children, because it is painful, especially when glucose is given; it is difficult, and it may be impossible to give large quantities and absorption is uncertain.

By rectum. All the substances used for dehydration with the exception of blood, can be absorbed by this route and it is still used quite often. It may be entirely satisfactory, but sometimes after several hours in which one thought the patient was absorbing fluid a large quantity is returned, when it becomes exceedingly difficult to keep anything like a reliable record of water balance. This is the chief disadvantage, but there are others—e.g., strong solutions of glucose cause irritation of the mucosa and interfere with further absorption; patients are apt to become uncomfortable from flatulent distension and the technique, though apparently simple, is, in fact, not so, if the entire amount of fluid given is to be retained and flatus got rid of. I generally limit its use to cases in which I feel the intake of fluid parenterally is not a vital feature in the treatment.

Intravenous. Practically speaking I use this route almost always and by the continuous drip method. Fluid is delivered with certainty into the circulation and can be kept up for days without interruption at a speed of 40 to 60 drops per minute; this is the usual rate of flow, but can be varied by merely turning a tap. It is independent of the patient's co-operation and usually is quite painless. If glucose is being given a sclerosing endothelial inflammation may begin in about three days and cause quite severe pain and swelling, so that for lengthy administration it is usually necessary to change to another vein. Occasionally a reaction is seen in the first few hours with a rigor; this, I think, is the only serious objection which can be raised against this route, and it has already lost much of its point, since careful preparation of apparatus and of the water used has to a great extent excluded the likelihood of trouble in this respect.

The apparatus is simple and as the fluid is run in very slowly the temperature at which it is delivered is of no account, though it can be warmed, if desired, by exercising a little ingenuity. One of the really important features of intravenous therapy is slow administration even when calculation shows the patient needs very large quantities of fluid; rehabilitation should therefore take several days.

60 drops a minute means roughly 9 pints in 24 hours, which is generally quite as much as is necessary for badly dehydrated cases per day, for in such cases two days' treatment would almost completely restore lost water and a reduced flow of 30 to 40 drops subsequently would be enough to provide a sufficient daily maintenance.

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