SPECIAL NUMBER ON
"FLUID THERAPY"

(*For the use only of Registered Medical Practitioner or Hospital or Laboratory*)

THE MADRAS CLINICAL JOURNAL

JOURNAL OF THE MADRAS STATE BRANCH INDIAN MEDICAL ASSOCIATION

(With which is incorporated the "Miscellany")

Vol. XXV MAY 1959 No. 11

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The above "Symposium" was held at the Old Boys Annual Meeting of the Stanley Medical College, Madras.

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" FLUID THERAPY "

By

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Introduction :

Fluid therapy as such is very ancient. Bathing and drinking of the waters of rivers and springs was practised by ancient man, no less than his counter-part of the present day who resorts to spas and other health resorts for the treatment of Arthritis, Gout, Paralysis and other chronic ailments. What is comparatively new is the newer routes of administering fluid; rectally, sub-cutaneously, intravenously, intraperitoneally, into the bone marrow and so on and the nature of fluid such as saline, blood plasma etc.

70 per cent of the body weight consists of water. This was confirmed by the investigations of Hardy and Drabkin who used heavy water (H_2O_2) in their investigations to determine the percentage of water in the body. This water is however, present in 3 distinct compartments – the Vascular compartment, the intracellular compartment and the intercellular compartment. These compartments are separated by semipermeable membranes, permeable to a varying degree of water and electrolytes, and we are now in a position to alter the composition of the vascular compartments.

It is described that the Chinese used to practise a method for killing unwanted prisoners by forcing them to drink water to a point where they developed oedema, water intoxication and died without leaving any trace or incriminating evidence of poison or toxin at postmortem. Whether this is true or not, it serves to emphasize that excessive haemodilution could lead to death. I am mentioning this because of the frequency with which we disturb the water and electrolyte metabolism of the body when we administer large amounts of fluids intravenously, which results in haemo-dilution.

Vomiting, Diarrhoea, De-hydration & shock.

We administer 5% glucose saline liberally in cases of vomiting, diarrhoea, shock and dehydration. When large quantities are given, we are introducing a large number of sodium ions into the vascular compartments. Theoretically this could lead to hypo-potassemia, which may result in death, We use drugs like Cortisone or ACTH which may lead to sodium retention and serious changes in the osmotic relationship in the vascular compartments.

Therefore a note of caution is urged when large quantities of fluid are administered intravenously.

A proper understanding of the water and electrolyte balance will help us to so alter the internal environment of the body as to correct the changes brought about by disease or toxins.

Hypoproteinaemia, alkalosis & Acidosis.

Serious osmotic deficiency in hypoproteinaemia can be corrected by giving a calculated dose of plasma intravenously ; a calculated quantity of sodium lactate or bicarbonate and a calculated amount of water is given to combat acidosis or ketosis of Diabetic coma ; dehydration is combated by administering a calculated amount of normal saline. Electrolyte deficiencies are corrected by administering the equivalent amount of electrolytes.

I may mention here about a hospital in the West where the Biochemist was called in to interpret the findings in a case of Uraemia and after correlating the biochemical findings with the patient's clinical condition, he proceeded to suggest a formula which would restore the patient's biochemical balance — so much of potassium, so much of sodium, so much of calcium, so much of water — just like you would write a prescription. We are far from that state of affairs and do not always get so much help from our Biochemist.

Toxaemia

I would like to say a few words about Intravenous fluids in Toxaemia. I believe that 2 to 3 pints of isotonic glucose saline in cases of toxaemia is definitely beneficial to the patient. It will certainly dilute the circulating toxins. It will promote diuresis and soluble toxins may be excreted. Glucose certainly provides ready calories which the patient may not be able to consume orally. In toxaemias of typhoid, I have found this to be a life saving measure.

Nutrition

This leads you to another point. The use of intravenous fluid therapy to provide alimentation to a patient who is not able to take oral nourishment. It is possible today to supply carbohydrates and amino acids and vitamins in the course of intravenous drip transfusions and to supply him with the water Nitrogen, and the calories he needs. Theoretically it is possible to keep the patient alive without oral feeding for an indefinite period of time. Fats cannot be given intravenously but I expect that further research may reveal methods whereby even fats can be given by this route.

Drugs

Drugs are introduced by intravenous drip transfusions—for example Nitrogen Mustard in the treatment of Leukaemia and Hodgkin's disease and ACTH in the treatment of Status Asthmaticus.

Conclusion

The Internal environment of the body cell is kept remarkably constant by self regulating mechanisms in the body. To understand these mechanisms and to use proper fluids to correct this internal environment when it is deranged by disease or metabolic disorders is intelligent therapy. On the other hand to use fluid therapy intravenously without proper understanding of the mechanisms may lead to serious changes in the internal environment of the body cell and may do more harm to the patient. We should remember that we are interfering with the fluid and electrolyte balance when large quantities of saline are given without much thought or indiscriminately ; also when drugs which interfere with electrolyte balance like Cortisone derivatives are freely prescribed ; or when prescribed in large doses or over a long period of time, serious disturbances of the mineral balance may ensue. Modern methods of introducing large quantities of fluids and electrolytes quickly into the circulation are therefore a double-edged weapon-intelligent use may save a life and failure to understand the principles may cost a life.

FLUID THERAPHY IN INFANCY AND CHILDHOOD

By

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THE term fluid therapy means correction of changes in water and Electrolyte concentration which occur in the body as a result of disease. The principles governing fluid therapy in children are similar to those in adults. There are however some important difference which are of great practical importance.

1. The water content of the body in infancy is higher (75 to 83%) than in adults (53% to 70%).

2. The rate of obligatory exchange of water and electrolytes is higher in infants. Therefore alterations in water and electrolyte concentration will have greater effect in infants.

3. In adults the kidneys are able to adjust limited changes in water and electrolyte concentration. In infants the kidneys are immature and are unable to cope up with these changes to the same extent as in adults.

4. An infant is easily liable to suffer from conditions which give rise to changes in water and electrolyte concentration in its body. Eg : Respiratory infections, Gastro enteritis etc.

A fact which is often over-looked, is that fat healthy looking babies are more liable to develop dehydration than lean babies. This is because adipose tissue contains hardly any water, so that fat babies have less water in their bodies proportionate to their weight than lean ones.

The correct proportions of water and electrolytes should be present in the body for a proper functioning of the various tissues. The various substances which are important in this respect and diseases which commonly alter them in childhood are as follows :—

I. Water : Deficiency as well as excess can be harmful. Deficiency is more common, and is the more dangerous of the two. It occurs in the following conditions.

1. Gastro Enteritis accompanied by Diarrhoea and Vomiting.

2. Surgical conditions of the bowel, especially obstruction, in which there is always excessive vomiting.

3. Other conditions in which vomiting is an important feature such as diseases of the central nervous system like meningitis.

4. Fever is very common in childhood, High metabolic rate in fever causes increase in the rate of respiration as well as the amount of water lost in the expired air. There is additional loss of water in sweat, which is also increased in all febrile conditions.

II. Acid Base-Balance : This may be disturbed in several ways :—

1. *Metabolic acidosis* : This is the commonest type of upset which can occur in the acid base balance. It is characterised by a decrease in the carbondioxide combining power of the blood. Conditions which commonly give rise to this defect are :—

(*a*) Diarrhoea due to any cause. Acidosis is caused by the loss of relatively

greater amounts of anions in the diarrhoeal fluid.

(*b*) Starvation due to anorexia is a feature in many diseases in childhood. This leads to ketosis and acidosis.

(*c*) Excessive intake of acid producing substances. One of the commonest is salicylate poisoning.

(*d*) Moribund state due to severe infections or any other cause. Here several factors play a part in producing acidosis.

(*e*) Renal disease is common in childhood and many of them give rise to acidosis because of the inability of the kidneys to carry out physiological adjustments essential for maintaining acid base balance.

(*f*) Certain rare diseases of metabolism like Diabetes and Renal Acidosis.

Metabolic alkalosis : This is less common. It is characterised by an increase in the carbondioxide combining power of the blood. This usually occurs in Children in the following conditions :—

(*a*) The commonest disorders giving rise to this condition in childhood are those accompanied by prolonged vomiting. Especially those associated with obstruction of the bowel eg : Congenital Hypertrophic Pyloric stenosis.

The Mechanism in these cases, is loss of hydrochloric acid in the vomited material.

(*b*) Excessive ingestion of alkalies.

3. Respiratory acidosis. This is caused by increase in blood carbonic acid. It is usually caused by retention of carbondioxide in the blood. This may result from any condition which gives rise to suppression of respiration. Common conditions in childhood which give rise to this are :—

(*a*) Diseases of the central nervous system like meningitis and concephalitis causing depression of the respiratory centres.

(*b*) Poisons like morphine which also depress the respiratory centres.

(*c*) Diseases of the lung such as Broncho-pneumonia, in which exchange of gases in the alveoli is prevented.

4. *Respiratory alkalosis :* This is caused by a diminution in blood carbonic acid. This occurs in conditions like hysterical hyper-ventilation which is uncommon in children.

III. **Sodium** : Both deficiency as well as excess are harmful.

1. *Deficiency of sodium* : This occurs in most of the conditions in which water deficiency occurs. These have already been enumerated.

2. *Excess sodium* : This is usually the result of injudicious treatment of dehydration. This can also occur in certain rare metabolic disorders.

IV. **Potassium**, deficiency as well as excess are both dangerous.

1. Potassium deficiency

(*a*) Limitation of intake which occurs in all conditions where there is anorexia.

(*b*) Rare metabolic and hormonal conditions.

Excess potassium : This is always a result of injudicious treatment. This condition is less common than potassium deficiency. But it is more dangerous

because it can cause sudden stoppage of the heart. Thus, death can occur without any warning. Because of this, one should never give potassium parenterally unless the potassium concentration in plasma has been estimated.

Management of Dehydration.

The term dehydration means abnormal losses of water and electrolytes from the body. This may be accompanied by changes in the acid base balance in the body.

There are two important types of dehydration. They are :

1. Hypo-tonic dehydration in which loss of electrolytes exceeds loss of water. This is less common in children and so will not be further considered.

2. *Hypertonic dehydration :* In this loss of water from the body exceeds loss of electrolytes. This is more common and occurs usually in the following conditions.

(*a*) All cases of diarrhoea.

(*b*) All conditions accompanied by vomiting Eg : Surgical conditions of the bowel, meningitis, etc.

(*c*) Renal diseases.

(*d*) Injudicious treatment causing over-dosage of electrolytes.

Signs and symptoms of this condition are marked thirst, dryness of mucous mumbrances, oliguria, weakness, fever and loss of weight. In severe cases it leads to a state of shock. The principal laboratory finding is elevation in the concentration of electrolytes in the blood. In these cases, one must look for signs of associated conditions. The important ones and their symptoms are :

1. *Acidosis :* This is usually accompanied by rapid respiration and acid urine.

2. *Alkalosis :* The symptoms are suppression of respirations and alkaline urine.

3. *Hypo-Kalaemia :* The symptoms are loss of tone in the skeletal muscles as well as distension of the abdomen due to loss of tone in the intestinal muscles.

Management of dehydration : The type and quantity of fluid to be given and the route of administration will depend to a large extent on the degree of dehydration as well as associated deficiencies.

As diarrhoea is the commonest cause for dehydration in infants and children we shall consider mainly this condition in discussing the management of dehydration.

For practical purposes dehydration can be considered to occur in four degrees of severity.

1. *Thirst :* This increases with the degree of dehydration.

2. *Central nervous system :* In mild and moderate degrees, there is irritability. In severe cases, apathy, and in very severe cases, coma.

3. *Urine :* This is diminished in mild and moderate cases and there is complete suppression in severe and very severe cases.

4. (*a*) *Colour of skin :* This is normal in mild and moderate cases. In severe cases there is cyanosis and in very severe cases, pallor.

(*b*) There is loss of elasticity of the skin, again the degree of which is proportionate to the degree of severity of dehydration.

5. *Dryness of the tongue :* This increases with the severity of the condition.

6. In infants there is depression of the fontanelle, the degree of which will depend upon the degree of dehydration.

Mode of administration of fluid.

1. *Mild cases :* Fluids can be given by mouth.

2. *Moderate cases :* Fluid can be given by mouth or by the subcutaneous route.

3. *Severe cases :* Intravenous drip is essential.

4. *Very Severe cases :* They are in a state of shock. Initially they are given a portion of the calculated quantity of the fluid very rapidly by intravenous injection. Plasma may, with advantage, be substituted for electrolyte solutions, for this purpose. This is then followed by an intravenous drip.

Warning : Rectal administration of fluids are worse than useless because it lulls one into a sense of false security.

Quantity of fluid to be given : This is calculated on the basis of body weight. There are two factors to be considered :–

(*a*) The quantity necessary for maintenance. This depends on age and is roughly calculated as $2\frac{1}{2}$ ozs. per Lb. body weight per diem for an infant upto 6 months of age.

(*b*) The quantity necessary for replacement of water already lost. This is calculated on the basis of body weight and varies with severity of the dehydration. For practical purposes the quantity lost in each of the degrees is taken as follows :

i. *Mild cases :* $2\frac{1}{2}\%$ of body weight.

ii. *Moderate cases :* 5% of body weight.

iii. *Severe cases :* 10% of body weight.

iv. *Very severe cases :* 15% of body weight.

Thus a child weighing 10 kg. with severe dehydration (Grade III) would have lost 10% of 10 kg. is equal to $10{,}000 \times \frac{10}{100}$ = to 1000 gm. to replace this loss 1000 gms. of fluid (1000 c. c.) must be given.

For maintenance, this child should get $2\frac{1}{2}$ gz. per pound body weight which is equal to 75 c.c. per pound which is equal to 150 c.c. per kg. of body weight. The 10 kg. baby should therefore get $150 \times 10 = 1500$ c c.-1500 c.c. $= 2500$ c.c. in 24 hours. Thus the fluid requirement of a child with dehydration can easily be calculated.

The reason for calculating the quantity necessary for rehydration is because excessive fluids may prove as dangerous as inadequacy.

(*c*) The various fluids which are given in different conditions are as follows.

1. Ringer's Solution (U.S.P.).

Ringer's solution or " triple chloride " solution contains sodium chloride, potassium chloride and calcium chloride. These are present in the following concentrations :

Sodium Chloride (NaCl)
146 mEq. per litre
0·3 gm. per litre.

Potassium Chloride (KCl)
4 mEq. per litre
0·3 gm. per litre.

Calcium Chloride ($CaCl_2$)
5·4 mEq. per litre
0.3 gm. per litre.

2. Lactated-Ringer's Solution (Modified Hartmann's Solution) U.S.P.

A litre of lactated-Ringer's solution may be prepared by dissolving sodium chloride, potassium chloride, calcium chloride and sodium lactate in sufficient distilled water to make 1000 ml. These substances are present in the following concentrations.

Sodium Chlorlde (NaCl)
103 mEq. per litre
6·0 gm. per litre.

Calcium Chloride ($CaCl_2$)
3·6 mEq. per litre
0·2 gm. per litre.

Potassium Chloride (KCl).

Sodium Lactate *
25 mEq. per litre
3·1 gm. per litre.

* 25 ml. molar lactate solution may be substituted for sodium lactate.

3. Potassium-lactate Solution (Darrow's Solution).

This solution, developed by Dr. Daniel C. Darrow, contains sodium chloride, sodium lactate and potassium chloride. Sodium and chloride are present in the same ratio as in interstitial fluid. The concentration of potassium is such that toxicity is unlikely to occur if the solution is administered with certain precautions. "K-lactate," as it is called, contains 4 gm. of sodium chloride, 2·7 gm. of potassium chloride and 52 ml. of molar sodium lactate per liter. This is equal to 122 mEq. of sodium, 104 mEq of chloride, 52 mEq. of lactate and 35 mEq. of potassium.

4. Normal Saline.

5. 5% Glucose solution.

It is a dictum that in infancy and childhood, normal solutions should as a rule be avoided. This is because they cannot deal with excessive quantity of electrolytes owing to the inefficiency of their kitneys. One uses either strength or 1/3 strength normal solutions.

The fluid of choice in cases of diarrhoea is half strength lactated ringor's solution containing 2½ % glucose which can be prepared by mixing equal quantities of 2 and 5. This should be given either intravenously as a drip or by mouth. When giving intramuscular of subcutaneous transfusions glucose should not be added to the solution.

When signs of potassium difficiency are present Darrow's solution may be given by mouth. This should not be given parenterally unless potassium content of blood has been estimated and found to be low.

In cases of dehydration with alkalosis the fluid of choice is normal saline solution.

In all cases of dehydration fluid therapy is a llfe saving measure and unless the proper fluid is given in the right quantities life may still be in danger. Hence the importance of calculating accurately the quantity of fluid necessary and assessing the electrolyte which should be given along with water for correcting the condition.

FLUID THERAPY

By

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THIS symposium being a short one, we will confine our interest to the management of cases requiring parenteral administration of simple solutions. A clinico-biochemical approach is necessary for assessing the degree and type of dehydration so that appropriate solutions may be employed to secure the most favourable conditions in the cells for the action of a specific medicinal agent or for surgery.

The initiation of fluid therapy is a matter for clinical judgment. When rehydration occurs, the newly induced biochemical changes do not promptly cause tell-tale symptoms and in this situation, laboratory aids prove most helpful in confirming the suitability or otherwise of the type of fluid initially chosen for administration.

In the present day when we bypass the Gastrointestinal tract and put in massive amounts of fluids right into the circulation, we certainly embark on a mild adventure. We know that about 70% of the body weight is made up of water and that the body water of 50 litres in the adult is distributed in compartments or spaces – 35 litres inside the cells as intracellular fluid, 12 litres in the interstices of cells as interstitial fluid, 3 litres as plasma water in the vascular channels. For practical purposes, the intravascular and interstitial fluids should be considered to form one unit – the extracellular fluid (ECF), The volume and composition of the fluids in the spaces are kept constant through the activity of semi-permeable membranes which show selective preference for the maintenance of certain gradients. Thus ions are kept confined to places, water is regulated by the kidney and A. D. H, and waste products are in continual clearance by the kidneys and the lungs so as to take the load away from the buffers and keep them trim for the task of maintaining the DH of blood constant at 7·4. The physiological data at our command are descriptive of a state where the integrity of the cells and membranes are unchallenged.

As water is the medium for the carriage of solutes, it might be realised that fluid losses through abnormal channels would produce far reaching effects. Anatomical lesions at cetain levels of the GI tract are attended with loss of fluid of distinct ionic make-up and a persistent loss cannot fail to leave deep imprint on the volume and ionic strength of body compartments.

In health the composition of blood is notable for its constancy and the chemical components are expressed as so many milligrams in 100 ml. In discussions on electrolyte balance, we specially seek to present the figures in a form which highlights the capacity of the basic and acidic radicals to keep the blood neutral by their concerted effort. You might recall that in the case of solutions for titrimetric work we employ the concept of chemical equivalence. While this concept serves also our present purpose, we take account of the low concentrations of radicles present in plasma and employ as unit onethousandth of the equivalent weight of the radical for calculating how many such units go to make up the weight of the radical in one litre of plasma. When presented as milliequivalents per litre (m. equiv/L), it becomes clear how the acidic and the basic parts balance each other nicely.

Normal values in plasma

Substance		Mgs. %	Divide by	to get m. equiv/L
Na	...	330	2·3	140
K	...	18	3·9	4
Ca	...	10	2·0	5
CO_2 vols %	...	54	2·2	20
Cl	...	350	3·5	100

Note :—Chlorides are often expressed as sodium chloride (normal range 600 - 660). It is therefore necessary to convert the figures in terms of Cl

Having talked about radicals which keep the neutrality of plasma, we pause to consider particles which contribute to osmotic pressure. For Na, K and Cl, the m.equiv. values and Osmolar values are identical. For Ca and Mg, the osmolar value is just half of the m. equiv. value. Since the number of particles per gram of a substance is inversely proportional to the molecular weight, a gram of NaCl (M. wt 59) will produce a vastly greater pressure than a gram of protein whose M. wt. is 300,000 or thereabouts.

Intimately mixed up with acid-base studies is the pH of blood. Though great deviations from the normal are incompatible with life, symptoms appear when there is a tendency to raise or lower the pH. Evidence will be on hand to indicate whether the abnormal trend is being resisted by a compensatory mechanism. In this connection, the equation of Henderson-Hasselbalch lays down that the blood pH equals 6·1 plus log ($BHCO_3/H_2CO_3$). The CO_2 combining power expressed in m. equiv/L gives the value for $BHCO_3$c in the equation; the estimation of carbonic acid (H_2CO_3) does not lend itself for routine work in the laboratory. Hence it is appropriate we look for a clinical finding to substitute for the denominator (i.e. H_2CO_3) in the equation. Fortunately we get dependable information from the character of the breathing and the reaction of the urine because respiration is regulated by the level of the H_2CO_3 relative to the existing bicarbonate and the reaction of the urine usually tends to portray the pH change in the blood.

Fluid therapy can be set on rational lines if we have an insight into the extent of changes in volume, osmolar concentration and distribution of ions in each compartment. We have no ready access to the intracellular compartment and the happenings there are mainly inferred from the data on blood, urine, ejecta and the water-balance sheet kept at the bed side. In the acutely sick patient, the integrity of cell membrances is gravely undermined by the forced reversal of fluid movements. With physiological boundaries broken and with a weird migration of ions, there is a new pattern of equilibrium and the patient becomes a law unto himself. In this mess, it is only by a close study of the symptoms that we can give a meaning and set a mooring to our laboratory data. In the management of a severe case with its changing picture, rapid therapeutic orientation is an utter necessity and happy results can be ensured only by the closest cooperation between the clinician and the Biochemist.

In simple cases of dehydration where gross changes in acid-base balance do not exist, replacement of volume lost by means of isotonic solution controlled by simple laboratory methods would be feasible. A fluid-balance sheet kept by a

painstaking nurse can be of the greatest aid. The collection of ejecta in transparent graduated containers and preservation with toluene would go a long way to mitigate the hardships of the nurse and help in the maintenance of an accurate record of fluid loss for comparison with normal fluid requirements.

Normal water allowance per day

Infant (2-10 Kg) : 750 ml ; Child (10-40 Kg) : 1500 ml ; adult (70 Kg) : 3000 ml.

Sodium chloride required per day for normal unsweating person

Infant :
1G Nacl or 125 ml normal saline.

Child :
3G Nacl or 350 ml normal saline.

Adult :
6G Nacl or 700 ml normal saline.

When dehydration is obvious, the deficit would be about one-fifteenth of the body weight. This would mean that a 70Kg. man would need at least 4 litres of water for replacement. If the patient is facing a chronic rather than an acute dehydration, one half of his deficit might be presumed to have occurred in the intracellular fluid. That is why at least one-half of the 4 litres of water required for replenishment should be in the shape of normal saline. This working rule enables therapy to be commenced while laboratory studies are in progress.

The size of the volume deficit can be assessed by a clinical survey taken in conjunction with the figures for urine specific gravity, Haemoglobin and blood urea. Difficult cases may need other tests. At some stage during replacement the clinical state and biochemic figures may revert to normal while the urine chloride may keep persistently low even if the patient be passing a litre of urine per day. Serial determinations of urine chlorides may therefore help decide how long saline administration is to be kept up.

In hospital practice one often meets with advanced stages of dehydration with its grossly disturbed volume, osmolarity and acid-base balance. It would seem best to have a scheme for routine investigation so that laboratory confirmation of clinical impressions can be secured and the most desirable hypertonic, isotonic or hypotonic solution substituted early in therapy. The scheme would probably run as follows :

(*a*) The presence and extent of ECF deficit is assessed clinically. Any sequestration of fluids brought about of burns, crushed injuries etc. is taken into account.

(*b*) While the urine is being tested for acetone, a request is made for the estimation of chloride, bicarbonate, Na and K of blood. The figures would show whether changes in osmolarity have occurred and this will help decide whether the solution to be given is to be isotonic or hypertonic.

Na and K determinations are made quickly over the Flame Photometer but the instrument in its present design requires so much attention for maintenance in our hot and humid climate it is better not to count upon its availability for routine use. In this circumstance, we try to wring out as much as we can from the figures for blood chloride and bicarbonate. A rough estimate of the osmolar concentration of the base sodium can be had by adding the m. equiv. values of chloride and bicarbonate. This is usually 100 plus 20. If in the patient this is less than 120 and if there is no acetone in the urine, it may be taken to mean diminished osmolar concentration of sodium. A sum above 135 would

point to increased osmolar concentration provided other physiologic indications of disturbed osmolarity are found.

This dodge of judging total base in plasma would fail if a great excess of other anions are present in plasma, as for example excess lactic acid in starvation, acetone bodies in diabetic acidosis, phosphoric and other acids renal insufficiency.

Sodium values determine the osmolarity of this key ion. However much we wish it, it is too much to expect this value to give an indication of the volume of the ECF. In fact, Sodium concentration may be normal in a contracted ECF or its concentration may be below normal in an abnormally expanded ECF. We should rest content to interpret Na values with clinical manifestations of a contracted or expanded ECF for the mere reason that volume is one thing and osmolarity is another.

As is to be expected, blood chloride values alter without a parallel change in the ECF volume. Being stuffing ions, chloride ions are liable to be thrown in or thrown out readily by other agencies. As chloride varies reciprocally with the concentration change of bicarbonate and organic acid ions, we observe its diminution in the primary CO_2 retention of emphysema or in morphine poisoning; chlorides here get thrown out in the urine to accommodate more bicarbonate ions in the blood, leading to a state where low chloride is made to co-exist with a normal ECF volume. On the other hand, chloride may rise while the ECF volume falls when alkaline fluids containing $NaHCO_3$ are heavily lost through diarrhoea or draining fistulae. Yet again, the obligatory water retention in the post-operative period following a major operation, or in the pre-diuretic phase of lower nephron nephrosis may lead to a low chloride value in the presence of an abnormally expanded ECF. These examples illustrate how a blind reliance on chloride value as an indicator of fluid volume will lead one to withhold saline when it is sorely needed and to give salt when it is not required. If so much could be said of blood chloride levels, what great illumination can we get out of urine chloride values?

(*c*) A record should be kept of the state of breathing. When this is considered in conjunction with the reaction of urine, the existence of an alkalosis or acidosis can be suspected. The information assists in the clinical solution to the Henderson-Hasselbalch equation and points at once to the correct choice of a replacement fluid. If there is uncompensated bicarbonate excess and deficiency of chloride ions, then chloride must be given. If in addition K is deficient, it must be made good.

In very severe uncompensated acidosis, the respiratory centre becomes unresponsive and deep breathing does. not take place. In fact respiration may appear normal in depth and frequency. When patients with severe acidosis and 'normal' breathing are treated with sodium lactate or sodium bicarbonate infusions, the very partial correction of acidity tends to wake up the benumbed respiratory centre and a hyperpnoea promptly supervenes. It is well to remember the respiratory response to very severe acidosis when evaluating the meaning of carbondioxide combining capacity.

Infants present special problems. The daily turnover of fluid constitutes one-half of his ECF in contrast to the adult in whom it works out to one-sixth of the ECF. Among the factors contributing to the increased proportion of water output in infancy and childhood are the following: (*a*) heavy insensible loss due to increase in surface area relative to volume; (*b*) increased B.M.R. leading to greater output of nitrogenous products

which require water for elimination in urine ; (*c*) the kidney of the child cannot concentrate urine as highly as the kidney of the adult. The operation of these factors would explain why GI tract disturbances are particularly dangerous in infants. Acid-base troubles tend to crop up very readily. When considering replacement, the limited ability of the immature kidney to deal with ions should be borne in mind and infusions made up of disposable ions (sodium lactate, ammonium chloride) should be preferred as opposed to saline which usually suffices in the adult. And as infants do not stand starvation, the nitrogen and calorie requirements should receive attention early in therapy.

In this brief survey I have to pass over very many items of interest to the clinician. Since the aim of fluid therapy is to assist the patient to swim to safety, the rewards are indeed rich to the clinician who tempers clinical judgment with laboratory knowledge.

'FLUID THERAPY'

REMARKS OF DR. K. S. SANJIVI, M. D.,

Chairman.

WHEN even abnormal amounts of water, sodium, chloride and potassium are presented to the kidneys of a normal person, fluid and ions will be retained in ratios appropriate for the restoration of the normal volume and composition of body fluids. The kidney can be depended upon to maintain homeostasis with regard to the water, electrolyses and the acid-base balance of the body.

The anti-diuretic hormone of the posterior pituitary and the adrenal cortical steroids are the more important hormonal factors controlling the kidney's efficiency. In retaining sodium and water, the sensations of taste and thrist play important roles in regulating salt intake and fluid intake.

While in a healthy person all the complicated endocrine and biochemical influences result in protecting a patient from water logging or dehydration, in diseased states, the controls get easily upset. When such upset occurs, the physiological and biochemical aspects of fluid therapy are by no means simple. Although loss of body fluid is usually described by the term 'dehydration', it is not a comprehensive term in as much as it does not indicate the accompanied losses of electrolyte. Water loss is always accompanied by a loss of electrolyte and conversely when an electrolyte is withdrawn, an accompanying removal of water is also effected.

Ideally all the necessary biochemical estimations must precede the appropriate institution of therapy in the clinical conditions to be mentioned. A flame photameter which is quite essential for the estimation of sodium and potassium is not universally available even in the teaching institutions. Actually more than the availability of the equipment, there is full scope for yet another kind of specialist in fiuid therapy. He will have a through comprehension of the rather baffling relationships between sodium and potassium, acid and base, extracellular and intracellular fluid compartments, and osmotic pressures at different sites and will be able to carry out the delicate and varying control of patients in several emergent situations.

However, the purpose of this symposium is not to voice the need for such a specialist but rather to remind the general practitioner about the common situations demanding fluid therapy and the methods of dealing with those situations.

High fever followed by excessive sweating, tropical conditions adding to the severity of sweating, is perhaps the commonest circumstance requiring the administration of large amounts of water and some salt. Patients and relatives must be properly instructed with regard to the fluid intake. Buttermilk with salt should be added to the conjees overcoming the usual superstition prevalent in South India that buttermilk is injurious in the presence of fever.

Vomiting and diarrhoea whether produced by cholera or a salamonella infection may produce severe states of dehydration. As a salamonella infection is quite commonly met with, it is essential that when the patient has severe diarrhoea, a doctor should see the patient frequently and record the blood pressure at every visit. I have known of cases where the doctor saw a patient with a severe infection, prescribed a totally inadequate dose of Pthalyl - Sulphathiazole and omitted to watch the patient who had got into a severe state of dehydration.

Apart from surgical shock, medical conditions such as coronary thrombosis and status asthmaticus may be associated with a state of shock. A sudden fall of blood pressure in coronary thrombosis may lead later on to thrombosis elsewhere as in the cerebral vessels, or to renal tubular failure and extra-renal uraemia. Gastro duodenal bleeding is another medical emergency requiring urgent and appropriate infusions.

In typhoid toxaemia, now fortunately becoming less common, there is no measure equal to the passing of a Ryles tube into the stomach and keeping up a continuous drip of 10% glucose which is interrupted every two hours for the feed prescribed.

In diabetic coma as well as in ketosis exhibited more frequently by children than adults, the ketone acid must be given space in the electrolyte structure of the plasma and this space is provided at the expense of a concentration of bicarbonate ion. This requires the infusion of Sod bicarb solution.

The route of administration of the fluid may be oral or parenteral depending on the absence or presence of vomiting and the urgency for the fluid replacement.

Reference has been made to the administration of fluids per rectum. In the olden days all kinds of nutritious material were administered per rectum in the hope that they will be absorbed but more recent work has shown that only saline and that too diluted with tap water is absorbed per rectum. The practice of rectal infusions is therefore being given up.

The choice of fluid may be glucose, saline, potassium solutions, soda bicarb, sodium lactate, or sodium potassium lactate depending on the immediate needs of the individual patient. Diabetic coma is a situation wherein all these fluids may have to be employed though not in the above order and may prove life-saving. Plasma, plasma expandus and blood are also to be considered ' fluids ', I suppose.

Finally, the institution of I. V. infusions is so frequently required and should so urgently be carried out that a competent general practitioner should keep an infusion set sterilised and ready much in the same way as he now keeps his 5 or 10 cc. syringe.

CASE NOTES

A Case of Polyarteritis Nodosa

Name : P. Admitted : 20–5–57.

Age : 18. Expired : 6–7–57.

Admitted for irregular fever for 10 days. History of having had a complete abortion 1 month ago. No history of having taken any sulpha group of drugs.

Patient is a well nourished young woman. Low grade fever. Occasionally it rises to 102° to 103°.

A transient type of rash generalised all over disappearing for a few days and reappearing. No itching.

C. V. S. No heart lesions but persistent tachycardia. Respiratory system : Nil abnormal. X ray shows heart within normal limits and lung fields normal. B. P. 120/70. Joints not swollen. No effusion but painful.

Fundus : Slightly tortuous vessels with an occasional spot of haemorrhage.

Urine : Albumin. No sugar. Few hyaline and granular casts.

Blood : Secondary anaemia.

R.B.C. 3·8 mill H.B. 55%

E.S.R. 95/1st hour. 125/2nd hour.

T.L.C. 11,000/. D.C. Eosinophil 20%.

P. V. O. S. closed. Complete abortion.

Serum negative for Widal, Brucella Abortus, negative culture and negative for Khan. The patient was put on Achromyecin parenterally with no effect on temperature.

The rash was appearing occasionally. The albuminuria continued. T.B. specialist heard a few basal rales and in view of the temperature suggested streptomycin and I.N.H. which was given a trial of 10 to 15 days with no avail.

A tentative diagnosis of Collagen disease was made and she was put on Cortisone (Deltacortril). Within a few days, the temperature touched normal and kept normal. The patient's outlook was pleasant. The Deltacortril was withdrawn and the next day, the temperature shot up to 102 degrees. But the Albuminuria was not touched by the cortisone.

While under Deltacortril, the patient was kept under an antibiotic cover of penicillin. Deltacortril was reintroduced with the same result as above E.S.R. was always high.

Muscle Biopsy was done :—

Report :—Showed suspicion of Polyarteritis Nodosa. Some perivascular infiltration with lymphocytes, fibrinoid degeneration and a suggestion of arterial blocks. Not definite.

The patient was continued on cortisone and kept normal for two weeks when she had a relapse. Liver was palpable and spleen two fingers. Temperature reappeared in spite of cortisone and penicillin. The cortisone was stopped and only antibiotics were continued. The albuminuria continued till the end and patient died of pulmonary oedema. P.M. was not allowed.

Discussion :—First described by Kaussmal in 1866 and was supposed to be of infective origin. But later the allied nature to serum sickness and other hypersensitivity to drugs like sulpha led to an aetiology of hypersensitivity and anaphylactic state.

It is a generalised vasculitis and all organs show the pathology. It is a disease of all ages, has a febrile onset, with tachycardia out of proportion to the fever. An elevated E.S.R. and Eosinophilia of 20% or so, asthmatic type of attacks

(Harkavy's Syndrome), anaemia, albuminuria, haematuria and hypertension.

Erythematous or Urticarial lesions or even subcutaneous nodes. There may be signs of peripheral neuritis due to the affection of the vaso nervorum and cranial nerve palsies, hemiplegia etc. Fundal changes may be prominent.

The pathology being necrotising pan arteritis with fibrinoid necrosis of all coats of small arteries.

The diagnosis is to be made by the poly-pathology and symptoms and by muscle biopsy.

Thus, from the above, it could be seen that the disease could simulate most other diseases — anaemia, kidney diseases, hypertension, asthma, pyrexia of unknown origin, sub acute bacterial endocarditis, Kochs infection and many others.

The treatment is not satisfactory but with the advent of A C T H and cortisone recoveries are being reported.

" VIRUS DISEASE OF THE GENITALIA "

by

DR. R. V. RAJAM, M.S., F.R.C.D.

(*Abstracted*)

While many infectious diseases are being controlled by modern sanitary and public health measures, there is evidence of new diseases springing up, as well as the pattern of old diseases are changing with the changing ecology of the host and parasite. The wide use of antibiotic and other modern methods in control of disease, their indiscriminate uses are changing the pattern of pathogens and saprophytes.

Herpes Simplex is a disease with muliple names but with an identical aetiology. It has a capacity to persist from generation to generation without any further importation. It has been definitely proved that the causative pathogen in this disease is a virus and the primary infection occurs in childhood and leaves a carrier state. Later remissions and recrudesences occur periodically depending on various stimuli.

In Herpes Simplex, antibody formation is at its height. The virus of this disease freely passes from cell to cell directly or by the cytoplasmic contiguity or by transfer to the descendents of the cell on mitosis.

Herpes Simplex is a common, trivial, self limited one with tendency to heal and recur. There may be an initial constitutional upset, or may be varicelliform, or show off as herpetic kerato-conjunctivitis, or rarely as hepato, adrenal necrosis, or meningetic or meningoencephalitic in nature, or as this lecture's theme dwells as *Herpes progenitalis*.

Transmission of Herpes genitalis may be by sexual contact but generally it is a local manifestation of the genitalia from a primary original focus elsewhere, and its pure venereal origin has not been proved. Here a note of emphasis is made on the carrier state especially among the females.

Paucity in the diagnosis is due to the unsuspicious mind – the local infection is trivial, may be mistaken for chancroids.

The speaker (formulated many reasons for the increase in the disease, like the antibiotic era, and stressed on the social and psychological sequelae of the disease.

In appearance it is a vesicular eruption on an erythematous base. There may be regional adenitis. The lesions are on

the mucous memberance of the genitalia. The lesions have a tendency to heal but may get secondarily infected and lose its originality. Recurrance may be due to many trigger mechanisms like fever, trauma of all sorts, heat and cold, menstruation and antibiotic exhibition.

This virus measures about 100-150 millimicrons, can be grown on tissue cultures and C. A. membrane of egg and antibody can be demonstrated in patients by neutralisation and C. F. T. tests. Geimsa stain shows off gaint cells with intranuclear feulgen negative virus as amorphous bodies.

Diagnosis is from the appearence of the grouped vesicles, spontaneous healing and the Geimsa stained smears of scrapings and the demonstration of the two serological tests mentioned.

Treatment Ordinarily self limited easily healing disease. Antibiotics should *not* be used. Auto-haemotherapy, auto-vaccination, heat killed virus injections, superficial X-Ray etc are tried. But the cow pox lymph in repeated doses has done some good. The speaker from an analysis of 38 patients with Herpes progenitalis has arrived at the following.

Age has no bearing on the disease, the duration of the recurrent types may last to 12 years. That the patients are mostly from the II and III strata of society. The treatment given was fresh vaccine lymph by scairfications in the forearm ranging once a week and generally four in number. The contacts and partners were also similarly treated to prevent a back infection. The results of this treatment with *pox* vaccinia was invariably good.

ASSOCIATION NEWS

ANAMALAI BRANCH

The monthly clinical meeting was held on 17—1—59 with Dr. M. K. R. Jayachandran in the Chair. Mr. M. D. Dissawala gave an interesting talk on Do's and Dont's in Obstetrics.

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A meeting was held on 28th February 1959 with Dr M. K. R. Jayachandran presiding. Dr. Jayachandran addressed "Amoebiasis with special reference to Amoebomas and Hepatitis."

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A clinical meeting was held on 21—3—1959 under the Chairmanship of Dr. M. K. R. Jayachandran, Dr. Subbanna gave an interesting talk on Appendicitis.

CHETTINAD BRANCH

The monthly meeting of the Association was held on 21—2—1959 at the S. M. S. High School. Dr. S. Subramaniam presided.

Dr. L. Sylvester, District Medical Officer, Ramnad spoke on 'Hazards of Antibiotic therapy and their cautious use'.

ERODE BRANCH

A meeting of the Association was held on 6–2–1959 with Dr. E. S. Venkatraman in the Chair. Dr. M. V. Krishnamurthy, B. A., M. B. B. S; M. R. C. P. (England) D. T. M. & H., District Medical Officer, Coimbatore, spoke on "Nepritis in Children".

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Miss Georgine Boyd, B. Hsc., Theosophist from New Zealand spoke on "Diet and Health" in a meeting held on 9–3—1959. The learned lecturer put in a strong plea to use hand-pounded rice, unrefined sugar and groundnuts.

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Dr. Y. P. Vasudevan, Vice-President, Indian Medical Association (Central) addressed the association on "Activities and Achievements of the Indian Medical Association". In the lucid speech lasting for more than an hour, Dr. Vasudevan traced the history of the Association and the fight it had to put up to defend the rights and privileges of medical practitioners.

COIMBATORE BRANCH

A monthly meeting of the Association was held on 3rd December 1958 in the Association premises. Dr. T. V. Sivanandam, MBBS., MLC., President presided.

Dr. S. C. Sen, Ex-President of I. M. A., addressed the members on 'Problems of Euthanasia' & 'Problems of Artificial Insemination.'

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A combined meeting of the Coimbatore District Medical Association and the Clinical Association of the Headquarters Hospital, Coimbatore was held on Saturday the 31st Jauuary 1959 in the Nurses' Headquarters Hospital, Coimbatore. Dr. M. V. Krishnamoorthy, MRCP., DTM,, Dt. Medical Officer, Headquarters Hospital, Coimbatore, President of the Clinical Association presided.

There were demonstrations of interesting cases by the Medical Officers of the various departments of the Hospital. Members had lively discussions over the cases presented and in the end the President Dr. M. V. Krishnamoorthy, stressed the value of the demonstrations

of cases instead of merely giving lectures and also wished that more such meetings could be arranged for the benefit of the Medical Practitioners in future.

MADRAS CITY BRANCH

The 23rd Annual General Body Meeting was held on 8–2–59 at Hotel Dasaprakash with Dr. P. Alagasingari Naidu in the chair.

After adopting the Annual report and passing the accounts the following office bearers have elected for the ensuing year.

President :

Dr. Capt. G. Sriramulu.

Vice-Presidents :

Dr. C. R. Ramachandra Pillai,
Dr. K. Rama Rao.

Hony. Secretaries :

Dr. A. Pattabi,
Dr. D. R. Varman.

Hony. Treasurer :

Dr. K. V. Swamy.

A committee of twelve, members to the central eouncil and state council were also elected.

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On 18–2–1959 at the Madras Medical College, Dr. Libeno Ajello, Scientific Director, Mycology Unit, Communicable disease center, Atlanta, U. S. A. delivered a lecture on " Moniliasis and Allied diseases ".

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A clinical meeting was held at Madras Medical College, on 21—3—1959 when Dr. M. Viswanathan, M. D. Hony. Physician Govt. Stanley Hospital, Madras delivered a lecture on " Tropical Eosinophilia ".

MADURAI BRANCH

At the 31st Annual Conference of the Association held in the Association premises on 28th February 1959, the following have been elected as the Office-Bearers, for the year 1959—60.

President :

Dr. K. Gopal.

Vice-President :

Dr. K. Ramachandran,

Hon. Treasurer :

Dr. A. Arunachalam.

Hon. Secretary :

Dr. T. Thirugnanam.

Governing Body Members :

Dr. Abdul Sathar.
Dr. A. S. Annamalai.
Dr. K. Balakrishnan.
Dr. S N. Ganapathy.
Dr. G. A. Kennet.
Dr. K. S. Krishnan.
Dr. G. A. Naidu.
Dr. C. K. P. Menon.
Dr. M. Subramaniam.
Dr. P. Vadamalayan.
Dr. M. Venkatachalam Chetty.

Provincial Council Members :

Dr. G. A. Naidu.
Dr. S. Subbanarayanan.
Dr. K. G. Ramabadran.
Dr. K. Gopal.
Dr. S. N. Ganapathy.
Dr. A. Arunachalam.
Dr. Abdul Sathar.
Dr. P. Vadamalayan.

Central Council Members :

Dr. N. Suriyanarayanan.
Dr. Abdul Sathar.

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NILGIRI BRANCH

The monthly meeting of the Association was held on Saturday 21—3—1959 at Pasteur Institute Coonoor.

Major S. Ramaiah, A. M. C., Military Hospital, Wellington, addressed the members on " Infections of the Hand ".

The monthly meeting of the Association was held on 18th April 1959 at Lawley Institute, Ootacamund when Dr. Y. P. Vasudevan, M. B., B. S., B. S. SC., Vice-President, Indian Medical Association addressed the members on "The I. M. A's Activities and Achievements".

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The monthly meeting of the Association was held on Friday 15—5—1959 at the Lawley Institute, Ootacamund to meet Director of Medical Services, Madras & District Medical Officers in Madras State who attended a Conference at Ootacamund.

Dr. J. K. G. Webb, M. A., B. M., B. Ch., (Oxon), M. R. C. P., Professor of Paediatrics, Christian Medical College, Vellore addressed the members on "Encephalitis".

NAGAPATTINAM BRANCH

An ordinary meeting of the Association was held on 2—5—1959 at Dr. T. S. Varadachariar's Clinic. Dr. A. Thiyagarajan who presided over the meeting spoke on the importance of holding frequent scientific meetings and also requested the members to attend the meetings without fail.

SOUTH ARCOT BRANCH

The monthly meeting of the Association was held on Saturday the 24th January, 1959 in the Government Head Quarters Hospital premises, Cuddalore. Dr. P. M. Palani, M D., Hony. Physician, General Hospital Madras gave a talk on "Modern Trends in Medicine".

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The monthly meeting of the Association was held on Saturday the 21st March, 1959 in the Women Civil Assistant Surgeon's Quarters, Villupuram. Dr. AL. Annamalai, M.R.C.P. (Lond.), DTM & H, Physician General Hospital, Madras, spoke on "Cough, its significance and Management". Dr. M. Natarajan, MBBS., ZDV. (Vien.), FDS. (Lond.) Dermotologist Stanley Hospital, Madras, spoke on "Common Skin Diseases and their Management".

SALEM BRANCH

The Annual Meeting was held on 29th March 1959, and the following office-bearers for the year 1959—60 have been elected.

President :

Dr. S. Sundaram.

Vice-President :

Dr. S. Kannan.

Hony. Secretary :

Dr. P. K. Chandrapal.

Hony. Jt. Secretary :

Dr. J. Sugavanan.

Hony Treasurer :

Dr. K. V. Dhanakoti Nayudu.

Members of the Executive Committee :

1. Dr. V. Raghavendra Rao.
2. Dr. S. Krishnamurthi Rao.
3. Dr. U. B. Lakshimi.
4. Dr. S. Arthanari.
5. Dr. K. Rajagopal Naidu.

Representative to Central Council :

Dr. K. R. Hariharan.

Alternate Member to the Central Council :

Dr. T. S. Shanmugasundaram.

Representative to the State Council :

1. Dr. S. Valeeswaran.
2. Dr. K. Govindarajulu.
3. Dr. K. Jayaramachandran.

Dr. M. Sivaram, B. A., M. B., B. S., Bangalore addressed the members on "Research in General Practice".

Dr. N. R. Narayan, M.B., B S, F.R.C.S. (Edin) F.R.C.S. (Eng). Hony. Surgeon, Bowring and Lady Curzon Hospital, addressed the members on "Modern Trends in Surgery".

Dr. Y. P. Vasudevan, Vice-President, I. M. A. addressed the members on "The I. M. A's. activities and achievements".