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MODERN TRENDS IN OBSTE

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IN

OBSTETRICS

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Edited by

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FOREWORD

The first and second series of *Modern Trends in Obstetrics and Gynaecology* were edited by Kenneth Bowes, whose untimely death was a great loss to British Obstetrics. His great erudition and his wide personal knowledge of his obstetric colleagues and their work made him an ideal editor.

The publishers have decided to change the format of the book so that it will be somewhat smaller in size than the previous series. It is hoped that a new series will be published every two years or so. It has also been thought wiser to publish the series in Obstetrics and Gynaecology in separate volumes.

It has not been an easy task to select the subject matter for this volume as, for reasons of size, all modern trends could not be included. I wish to thank all the contributors to this volume for the great trouble they have taken with the subjects allotted to them and on which they are all acknowledged authorities, and to the staff of Butterworths for help in the production of this book.

ROBERT KELLAR

Edinburgh,
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CHAPTER 1

WATER AND ELECTROLYTE BALANCE IN PREGNANCY AND THE ACTION OF DIURETICS

J. C. McCLURE BROWNE

WATER BALANCE

It has long been known that of the total body weight of a woman about two-thirds is due to water. Two-thirds of this water is intracellular and one-third extracellular, the latter being partly intravascular and partly interstitial. The intracellular water with its dissolved substances constitutes the internal environment of the cells and is of paramount importance. The main, if not indeed the only, function of the interstitial and intravascular fluid being to help in its preservation by removing metabolites and bringing nutrients and the oxygen to enable them to be utilized. Metabolites such as urea are excreted by the cell in watery solution into the interstitial fluid, whence they are conveyed to the intravascular fluid and excreted by the kidney still in solution. In this way, 1,500 ml of water are lost every 24 hours. Under normal conditions, a further 1,000 ml are lost each day through the skin and in the expired air, and a further 100 ml in the faeces, making a total daily loss of 2,600 ml.

This loss is regularly made good by water ingested as such, by water contained in food, and by water produced as a result of metabolic processes. When extra water is lost as a result of sweating or increased respiration the antidiuretic hormone (ADH) of the posterior pituitary causes more water to be absorbed by the kidney tubules, the urine becoming smaller in volume and more concentrated, conserving water until ingestion makes good the deficiency. The volume of urine and the amount of water ingested varies, therefore, according to circumstances. In severe dehydration from whatever cause the volume of urine excreted daily may be extremely small. When normal water balance is restored the volume of urine excreted increases again to normal levels. Thus, when an adequate supply of water is available and renal function is normal, the body can accommodate a wide range of climatic and other conditions.

An important function of water in the body, however, is as a vehicle for dissolved substances. Thus, intracellular water has a high potassium and phosphate content, while extracellular water, on the other hand, has a high sodium and chloride content and a low potassium content. The passage of these substances into and out of the cell is governed by the cell membrane, freely permeable to water, but, under hormonal influence, highly selective in regard to sodium, chloride, potassium and other ions. There is thus a constant movement of water from one compartment to another and from within the vessels to the interstitial space, carrying with it a wide variety of substances in solution. Intravascular pressure tends to drive fluids out of the vessels while, on the other hand, the osmotic pressure of the plasma proteins tends to hold them in.

ELECTROLYTE BALANCE

Apart from the maintenance of plasma volume the chief function of water is as a solvent or vehicle for electrolytes.

Electrolytes may be defined as chemical substances which in watery solution undergo ionization, positively charged ions being known as cations and negatively charged ions being known as anions. Undoubtedly the most important cation is sodium, with potassium ranking second. Calcium and magnesium seem to be less important. The principal anion is chloride, though bicarbonate and phosphate are important, while sulphate and organic acids are of lesser significance. Inside the cells potassium is the main cation, but outside the cells, that is in the interstitial fluid and the plasma, sodium predominates.

Sodium Balance

The total amount of sodium in a woman weighing 60 kg is 53·6 g. Of this 20 g is in bone and is virtually immobile, taking little or no part in the daily exchanges of sodium throughout the body. The fatty tissues of the body contain almost no sodium and each 100 g of fat-free body weight contains 0·109 g of sodium. The intake of sodium varies widely according to habit and climatic and working conditions with a range of 5–15 g per day. In health the daily loss of sodium exactly matches the intake so that a steady state is preserved. In climatic or working conditions where sweating is excessive a considerable amount of sodium may be lost through the skin, and the urinary loss of sodium is correspondingly reduced, though under these conditions the amount of sodium conserved in this way may be

insufficient to maintain the *status quo*, and heat exhaustion or miners' cramp may result, unless steps are taken to increase the intake appropriately.

In health the urinary output of sodium is 4–6 g per day and random specimens of urine should contain not less than 3 g/l. When excessive sodium is lost, for whatever reason, its intravascular concentration is at first maintained by withdrawal of sodium from the extravascular interstitial spaces into the vascular pool. If sodium depletion continues the total sodium content of the plasma is reduced and the plasma osmotic pressure falls. As a result water escapes from the plasma into the interstitial spaces so that a contraction in plasma volume follows which may ultimately be so severe as to cause circulatory collapse.

Chloride, the anion chiefly associated with sodium, follows more or less the same pattern. When sodium is lost chloride is lost too in equivalent amounts. Depletion of body sodium, therefore, in practice implies depletion of sodium chloride. It has often been supposed that sodium retention is at least partly responsible for water retention, so as to retain the isotonicity of the body fluids. De Alvarez and Smith (1956), however, reported a case in which pre-eclamptic toxaemia with retention of water developed although the woman was kept in negative sodium balance. It may be that in such cases the sodium necessary for water retention is mobilized from sodium stores not normally taking part in electrolyte balance; stores such as are found in bone.

Potassium Balance

Potassium, the main intracellular cation, is present in a concentration of 0·265 g per 100 g of lean body weight. A woman weighing 60 kg contains 130 g of potassium, the total amount of potassium available for exchange being 102 g, as some is fixed. The average intake of potassium is 2–6 g per day and the output is approximately the same. Four-fifths of the normal daily potassium loss occurs in the urine and one-fifth through the faeces. Vomiting or diarrhoea may cause serious potassium loss resulting in weakness of muscular action, this is particularly evident in the cardiac muscle. Potassium deficiency may readily be recognized by a characteristic change in the electrocardiogram as well as in the serum levels.

Little is known of the balance of other electrolytes but it is probable that they are not of such importance as sodium, potassium and chloride.

KIDNEY FUNCTION

It will be clear that the kidney is the main regulator of water and electrolyte balance in the body. The renal glomeruli produce a filtrate which is identical in composition with the plasma, except that proteins are absent. The amount of glomerular filtrate produced depends on the blood pressure and rate of supply to the kidney, and the filtrate is modified by absorption of water and of sodium chloride and other electrolytes in accordance with the needs of the body.

Hormonal Control of Fluid and Electrolyte Balance

Kidney function as a whole is closely controlled by hormonal action.

Absorption of water through the tubules is regulated not only by the blood supply to the tubules, and the action of antidiuretic hormone, but also by the concurrent reabsorption of sodium chloride, as this must be absorbed in solution. Tubular reabsorption of sodium is governed by the action of aldosterone formed by the adrenal cortex. When the plasma sodium falls aldosterone secretion is increased causing greater reabsorption of sodium from the renal tubules.

The ovarian hormones, too, probably play some part in water and electrolyte retention.

The clinical occurrence of premenstrual oedema is well recognized. Preedy and Aitken (1956) have shown that injection of oestradiol monobenzoate into dogs caused a transitory increase in plasma sodium, an increase also found when the animals had been adrenalectomized before administration of ovarian hormone. Oestrogen therefore seems to have a direct effect on the renal tubules. However, oestrogens are so readily metabolized by the healthy liver that their real function in water conservation must be unimportant.

There is some evidence that aldosterone affects the movement of electrolytes across cell membranes so that, for example, sodium can move rapidly out of the cells into the interstitial fluid (Kumar, Feltham and Gornall, 1959). Thus, water and sodium balance in the non-pregnant woman is accomplished by a complex regulation of renal function by variation in the renal glomerular filtration rate under the influence of circulatory changes and by aldosterone acting on cell membranes, and aldosterone, the antidiuretic hormone, and possibly the ovarian hormones acting on the renal tubules.

CHANGES IN NORMAL PREGNANCY

Water Balance

In pregnancy there is an average weight gain of 24 lb. (11 kg), 60 per cent of which (6·5 kg) is due to extra water. This is distributed in the following way.

Areas of distribution	*Amount*
Plasma	1,300 ml
Foetus, placenta and liquor amnii	2,000 ml
Uterus and breasts	700 ml
Extragenital interstitial fluid	2,500 ml
Total:	6,500 ml

This extra fluid is held in the body as a consequence of sodium retention due to the action of aldosterone, which is greatly increased in amount during pregnancy. Though oestrogens and progesterone are produced by the placenta in such large amounts in the pregnant woman their role in water retention in pregnancy is probably not great, because in toxaemia, where the circulating amounts of these hormones are much the same as in normal pregnancy, there seems to be an increase in the amount of fluid retained.

It is probable that during pregnancy the water content of the cells increases appreciably (Hawkins and Nixon, 1961), the rest of the excess water being distributed in blood plasma, foetus, placenta and liquor, the extragenital interstitial fluid and in the new tissue formed by the growth of the uterus and breasts.

The increase in water content of the plasma produces a corresponding increase in blood volume, but the total amount of plasma protein does not increase, so that the concentration of the plasma proteins, which averages 8·18 g per 100 ml of plasma in the non-pregnant woman, falls steadily throughout pregnancy until just before term, when it has a value of 7·50 g per 100 ml, a drop of about 6 per cent.

The fall in plasma protein during pregnancy diminishes osmotic pressure in the vessels so that there is a tendency to oedema, more marked as the pregnancy progresses and greatest in the lower limbs, where in the erect position hydrostatic pressure is greatest, as fluid is forced out from the vessels into the interstitial spaces.

In normal pregnancy as in the healthy non-pregnant state, if excess water is taken in it is rapidly eliminated by the kidney, though the efficiency of renal function in the pregnant woman is affected by posture, being not at its most efficient in the erect position but maximal when the woman is recumbent (Assali, 1962).

Electrolyte Balance

Sodium

During pregnancy there is a considerable retention of sodium, about 20 g in all (Plentl and Gray, 1959; Davey, O'Sullivan and Browne, 1961; Mahran, 1961), though MacGillivray and Buchanan (1958) gave a somewhat lower figure (17·5 g). This is distributed in much the same proportion as is the extra water retained in pregnancy. The sodium is retained at a steady rate of about 0·5 g per week and is probably due to the action of aldosterone.

Potassium

Like sodium, there is retention of potassium in pregnancy in excess of the normal non-pregnant balance. Approximately 10 g are retained, though MacGillivray and Buchanan (1958) gave a figure of 6·7 g. It is probable that, like sodium, all the extra potassium so retained is utilized in the formation of new tissues in connection with the pregnancy.

Renal Changes

There is a progressive increase in renal plasma flow and glomerular filtration rate throughout the course of pregnancy, reaching a maximum during the third trimester and falling to non-pregnant levels 6–8 weeks after delivery. This is probably an expression of the need to facilitate elimination of metabolites such as urea, the production of which is increased.

Hormonal Changes

Oestrogens and Progesterone

The placenta produces large amounts of these hormones; it has been noted that they may cause a transient sodium retention in experimental animals. Their rapid metabolism by the liver, however, renders it unlikely that they play any major role in the altered fluid and electrolyte balance of normal pregnancy, though it will be seen later (page 9) that when there is hepatic impairment

such as occurs in pre-eclamptic toxaemia their role may be more important.

Adrenocortical Hormones

There is no doubt that the production of adrenocortical hormones is greatly increased in pregnancy (Venning and colleagues, 1957; Bayliss and colleagues, 1955; Rinsler and Rigby, 1957; Kumar, Feltham and Gornall, 1959). It is probable that the most important cortical hormone from the standpoint of fluid and electrolyte balance is aldosterone. The healthy non-pregnant woman excretes 2–10 μg per 24 hours, while in normal pregnancy up to 120 μg may be excreted in the same period. Kumar, Feltham and Gornall (1959) found that the average urinary output was between 4 and 11 μg per day in the non-pregnant woman rising to 50 μg in late pregnancy. A rise in aldosterone production causes sodium retention and could provide the mechanism whereby the necessary sodium is retained in pregnancy. As will be seen later, however, aldosterone production is impaired in toxaemia when most workers consider that there is excessive sodium retention. Aldosterone excretion returns to normal within a week after delivery (Stark, 1960).

Gonadotrophins

The urinary excretion of gonadotrophins rises sharply during the first 8 weeks of pregnancy when it reaches a peak, falling rapidly thereafter until a more or less steady low level is reached at 20 weeks which persists until delivery. This peak in no way coincides with the fluid and electrolyte changes in pregnancy and it is reasonable to suppose that the gonadotrophins have no significant role to play in this respect.

Growth Hormone

Administration of human growth hormone causes nitrogen, sodium and potassium retention although the serum levels of the electrolytes are not noticeably changed. The Medical Research Council Panel (1959), reporting on this, found that the amount of potassium, magnesium and phosphate retained was of the order which would be expected for protein synthesis calculated from the retention of nitrogen. The amount of sodium retained, however, was greatly in excess of this amount. This is exactly the pattern seen in normal pregnancy, and it is tempting to suppose that growth hormone may be responsible for these changes.

PRE-ECLAMPTIC TOXAEMIA

Pre-eclamptic toxaemia is a disease of the latter half of pregnancy characterized by the development of hypertension, oedema and albuminuria, and usually preceded by excessive gain in weight (this is denied by Rhodes (1962)), though some of the severest fulminating cases may develop gross oedema so rapidly that there may have been no fluid intake whatsoever between the onset of signs and the occurrence of marked swelling. Mukherjee and Govan (1950) have shown that in toxaemia the protein content of oedema fluid is the same as that of normal tissue fluid, so that the oedema cannot be due to increased protein osmotic pressure in the interstitial fluid.

Sodium

There is general agreement that sodium retention in cases of pre-eclamptic toxaemia is excessive (McCartney, Pottinger and Harrod, 1959; Mahran, 1961). A recent investigation by Davey, O'Sullivan and Browne (1961), however, showed that although excessive sodium retention occurred, the extra sodium was retained weeks before clinical toxaemia developed and was in large part eliminated by that time. The mechanism involved in excessive sodium retention is obscure. Associated with the sodium retention is a corresponding retention of water which can explain the excessive weight gain usually observed. MacGillivray and Buchanan (1958), using an isotope dilution method, calculated that the total exchangeable sodium in pre-eclamptic toxaemia was, if anything, less than in normal women in late pregnancy. The figures obtained by Davey, O'Sullivan and Browne (1961) are compatible with this view, as on their findings MacGillivray and Buchanan's toxaemic women should have excreted most of the retained sodium by the time toxaemia developed.

Potassium

Little work has been done on potassium balance in toxaemia. MacGillivray and Buchanan (1958) found that the total exchangeable potassium in toxaemia was less than that in normal late pregnancy.

Renal Changes in Toxaemia

The ability of the kidney to excrete water is impaired. Thus, while a normal pregnant woman made to drink a litre of water after

12 hours of fluid deprivation will excrete most of the water load within 1½ hours, reaching a peak diuresis about half an hour after drinking, the toxaemic woman takes much longer (2½ hours) to excrete the water load, and the peak of diuresis is much lower and is attained much later than in the normal woman. The glomerular filtration rate in toxaemia is considerably reduced.

Renal biopsy (Pollak and Nettles, 1960) in toxaemia shows that there is oedema of the glomerular tuft involving the endothelial and epithelial cells of the glomerular capillaries. Even in mild cases there is marked ischaemia of the tuft, and in some cases oedema of the walls of the small arteries and arterioles. The more severe the renal lesion the more severe are the clinical signs of the disease. Except in the most severe cases, when anuria may develop, these changes seem to be completely reversible once delivery has been effected.

Hormonal Factors

As has already been noted (page 6), in normal pregnancy oestrogens and progesterone are probably of minor importance in procuring the necessary alterations in fluid and electrolyte balance.

In toxaemia, although there seems little doubt that there is placental ischaemia (Browne and Veall, 1953) and therefore the likelihood of impairment of oestrogen production, the Edinburgh workers (Kellar and colleagues, 1959) have found no evidence of any fall in blood oestrogen levels, indeed they may be high, though Louros, Miras and Frangiadakis (1960) reported that urinary excretion of oestrogens and of pregnanediol is diminished. This apparent discrepancy may possibly be explained by concurrent failure of oestrogen and progesterone metabolism due to hepatic ischaemia (Hoshino, 1959). Hoshino showed that the oxygen consumption of the liver per minute per square metre is reduced to about two-thirds of that in normal pregnancy, so that the metabolism of these hormones is probably impaired. A rise in blood oestrogen level could cause salt and fluid retention, the oestrogens acting directly on the renal tubules (Preedy and Aitken, 1956).

Aldosterone

That altered adrenal cortex activity might precipitate toxaemia by causing salt and water retention seemed at first likely, but the amounts of 17-ketogenic steroids and of deoxycorticosterone are unchanged in toxaemia (Aarstrand, 1958) while the amount of circulating aldosterone is diminished (Louros, Miras and Frangiadakis, 1960; De Bruïne, 1960). Moreover, there are numerous