ELECTROLYTE METABOLISM IN NORMAL AND ABNORMAL LABOUR

by

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In recent years a growing interest has been taken in the ionic composition of the body in various physiological and pathological states. Considerable amount of literature has accumulated on the blood electrolyte changes during pregnancy, but there is singular lack of knowledge and paucity of literature on the electrolyte composition during labour, which can be considered as a form of "Stress" or tissue trauma even in the physiological state.

The pattern of recovery from injury or trauma had been recognized from very early times. Bauer (1872) observed that an increased elimination of nitrogen followed upon haemorrhage, and Hawk and Gies (1904) showed that the actual operation of venesection without withdrawal of blood was enough to cause increased urinary output of nitrogen and sulphur. Since that time many observers have noted this increased nitrogen catabolism as a result of injury associated with increased potassium loss (Cuthbertson *et al.*, 1939; Cuthbertson, 1942; Wilkinson *et al.*, 1949). The observations of Cuthbertson *et al.*, (1939) on urinary excretion of potassium and nitrogen led them to believe that the increased excretion of these substances was due to muscle catabolism following operative injury, but they noted that potassium was lost in greater proportion than nitrogen when compared to their relative concentrations in muscle protoplasm. Wilkinson *et al.*, (1949) had drawn attention to the marked retention of sodium in the post-operative period occurring at the same time as the phase of increased potassium loss. These metabolic changes have conclusively been proved to be due to mineralo-corticoid activity of the adrenal cortex. These changes in pattern, namely, sodium and potassium balance following operation or trauma can be explained by the relative permeability of the cell membrane which is altered so that potassium is liberated from the cell and any tendency to a rise in extra-cellular potassium is prevented by the rapid excretion of potassium in the urine. To replace this cell base, sodium appears to enter the cell from the surrounding extra-cellular fluid resulting in intracellular sodium retention and reduced serum sodium concentration. The effect of this stress is maximum within 48 hours of the operation.

Comparable to this state of stress and adrenocortical activity, produced by trauma of an operation, may be

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considered the trauma of an operative delivery. Trauma of childbirth, which may be considered as physiological in cases of normal labour with normal delivery, may reach pathological levels in cases of abnormal or prolonged labour especially if terminated by an operative delivery.

We can, therefore, assume that labour, especially if prolonged, can cause a state of stress which can be compared to the state of stress caused by operation and will result in increased adreno-cortical activity. It can be presumed that normal labour by itself brings about a chain of electrolyte changes under the influence of adreno-cortical hormones. But these changes are transient and mild and they do not bring about any appreciable disturbance in electrolyte composition. However, with prolonged labour, the tissue trauma, due to prolonged uterine contractions with the increase in the catabolic processes of the muscle, will bring about such changes which will disturb the metabolic process profoundly; such electrolyte balance will result in producing clinical features such as bladder and bowel distension, increase in uterine inertia and occasionally paralytic ileus.

This work was taken up with the idea that if it is established that some types of tissue reactions and electrolyte changes occur in prolonged or normal labour, as happens in post-operative cases with or without complications respectively, then those cases who go into prolonged labour should be considered as post-operative cases with complications and dealt with similarly with respect to the sodium and potassium changes.

Material and Method

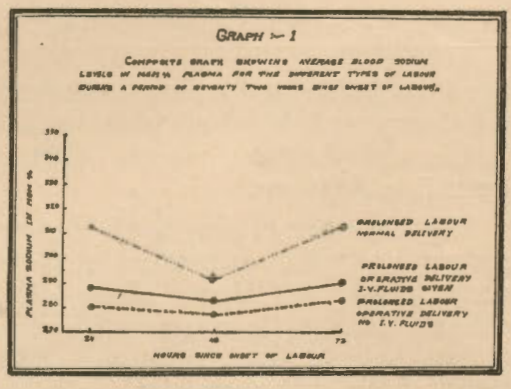
Ten cases of normal labour and 50 cases of prolonged labour, who were admitted in the Hospital for Women, were selected for analysis.

As MacPhee (1953) had shown that due to involvement of the renal excretion mechanisms the urine sodium level is not an indication of serum sodium levels and that serum potassium is not altered though urine potassium excretion is increased. Therefore, the estimations of sodium and potassium were restricted to serum sodium and urine potassium only. The estimations of these electrolytes were done on specimens collected on three consecutive days for each case, the first after twenty-four hours of the onset of labour, the second forty-eight hours and the third seventy-two hours after the onset of labour. The time of onset of labour was taken as the time when painful uterine contractions begin. Cases considered as normal labour were those who delivered within twenty-four hours of the onset of painful uterine contractions, the time of delivery being taken as that of the birth of the baby, and thus the time taken for the third stage of labour was not taken into account. Cases considered as prolonged labour were those who had been in labour for over twenty-four hours. The blood specimens were collected in oxalate tubes, 2 cc. being obtained by intravenous puncture for each specimen. After half to one hour the blood was centrifuged and the plasma sodium detemined in mgm./100 cc. Urine specimens were collected in ordinary sterile tubes; 2 cc. being obtained from the twenty-four hours' sample

of urine. The potassium level was determined in mgm./100 cc. of urine and then the total excretion for the twenty-four hours calculated in gms./day.

Result

Graphs I, II and Table I show the features of electrolyte changes in the plasma and the urine in normal and prolonged labour. In both types there was reduction in plasma sodium and increase in potassium excretion.



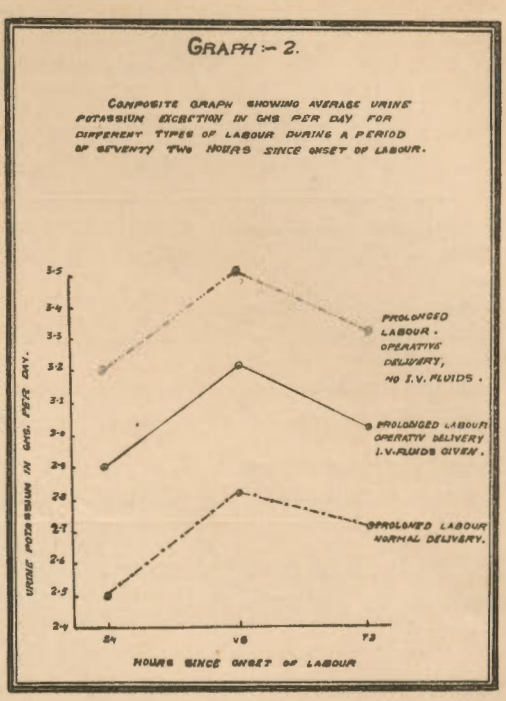


Table II shows the serum sodium and urinary potassium values in cases showing clinical evidence of potassium deficiency.

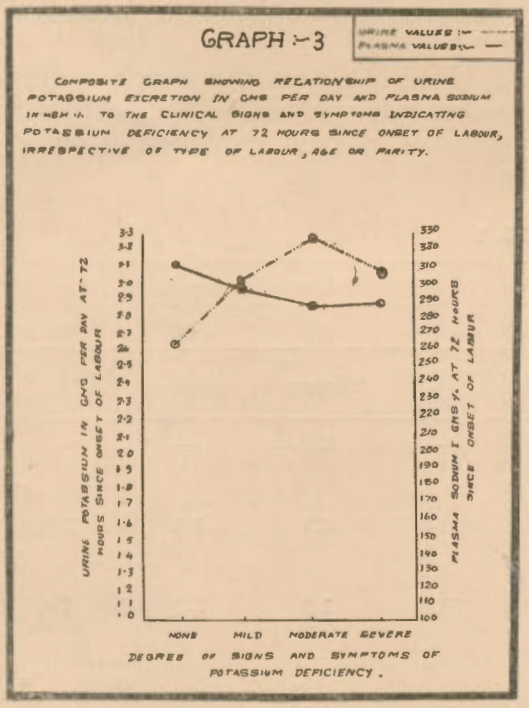
TABLE I

Types of labour	Plasma sodium in mgm% at			Urine potassium in gms/day at		
	24 hours	48 since	72 onset of labour	24 hours	48 since	72 onset of labour
Normal labour (normal deliveries)	330.2	—	—	2.4	—	—
Prolonged labour (normal deliveries)	312.8	294.0	312.8	2.5	2.8	2.7
Prolonged labour (operative deliveries No I.V. fluid)	288.0	283.2	288.2	2.9	3.2	3.0
Prolonged labour (operative deliveries with I.V. fluid)	280.0	276.2	280.8	3.3	3.5	3.3

TABLE II

Potassium deficiency	Plasma sodium in mgm% at			Urine potassium in gms/day at		
	24 hours	48 since	72 onset of labour	24 hours	48 since	72 onset of labour
No evidence of potassium deficiency ..	309.6	295.5	312.0	2.7	2:8	2.65
Mild evidence of potassium deficiency ..	297.6	283.6	297.1	2.88	3.14	3.02
Moderate evidence of potassium deficiency	284.4	279.7	286.0	3.04	3.43	3.26
Severe evidence of potassium deficiency	290.0	282.2	288.4	2.94	3.26	3.0

Graph III shows the correlation of electrolyte balance with the clinical features.



Tables III and IV show the electrolyte levels in cases who clinically show moderate and severe potassium deficiency and who had blood transfusion or dextrose saline during operative delivery.

The changes in levels of plasma sodium and urine potassium in these sixty cases in labour show that definite changes do occur in the electrolyte balance during labour. The trend of the changes found is similar to that found by other workers postoperatively namely, a decreased plasma sodium level occurring at the same time with the increased potassium excretion in the urine as demonstrated by Cuthbertson (1939).

MacPhee (1953) had obtained values of plasma sodium in postoperative patients that were on the average higher than the values obtained in this work. In his series, 48 hours after operation when maximum changes occurred, the values of plasma sodium varied between 310 mgm.% to 325 mgm.%, while in this work the average plasma sodium at 48 hours after the onset of labour, for all the types of labour and de-

TABLE III

Sl. No.	Type of intravenous fluids given	Plasma sodium in mgm% at			Urine potassium in gms/day at		
		24 hours	48 since	72 onset of labour	24 hours	48 since	72 onset of labour
1.	Blood—300 c.c. 5% G/S—1000 c.c.	294	282	293	2.8	3.4	3.0
2.	5% G/S—1000 c.c.	286	288	290	2.9	2.9	2.9
3.	Blood—150 c.c. 5% G/S—500 c.c.	285	282	286	3.0	3.2	2.9
4.	None	282	275	281	3.2	3.2	3.1
5.	None	286	279	286	3.1	3.6	3.5
6.	None	283	282	285	3.2	3.5	3.5
7.	None	275	270	276	3.1	3.8	3.5

TABLE IV

Sl. No.	Type of intravenous fluids given	Plasma sodium in mgm% at			Urine potassium in gms/day at		
		24 hours	48 since	72 onset of labour	24 hours	48 since	72 onset of labour
1.	Blood—150 c.c.	294	284	295	2.9	3.0	3.0
2.	Blood—300 c.c. 5% G/S—1000 c.c.	296	280	294	2.8	3.2	3.1
3.	Blood—150 c.c. 5% G/S—500 c.c.	288	284	284	3.0	3.2	3.1
4.	5% G/S—500 c.c.	288	283	284	3.1	3.5	3.1
5.	5% G/S—2000 c.c.	284	280	284	2.9	3.4	3.0

livery varied between 280 mgm.% to 325 mgm.% (Table I). Streeton (1952) found that marked signs and symptoms indicative of potassium deficiency were found when the urine potassium excretion exceeded 2.5 gms. per day. In this series the potassium excretion varied from 2.7 gms. to 3.26 gms. per day. MacPhee (1953) found exretion of 3 gms. of potassium at 24 hours, 48 and 72 hours after operation. In the cases of prolonged labour potassium excretion was much higher than in the post-operative cases. This high excretion of potassium, seen in cases of labour, may be explained by the fact that greater muscle catabolism is found after labour than after an operation, due to the activity of the uterine muscles in the process of contraction and retraction and sub-

sequent involution.

The importance of potassium in the serum and extracellular fluids has been well recognized ever since the work of Ringer (1883). In recent years great interest has been focused on the loss of potassium in various acute medical and surgical conditions. Brown *et al.*, (1944) described low levels of serum potassium in chronic nephritis associated with muscular weakness and paralysis. It is, however, largely due to the work of Darrow and his associates that clinical potassium deficiency has become widely recognized (Govan and Darrow, 1946; Darrow, 1948; Darrow and Pratt, 1950).

The increased potassium excretion in the urine found as a result of the stress of labour will lead to a state of potassium depletion. As potassium is contained mainly in muscle cells it is these which show the marked changes evidenced clinically as potassium deficiency. This supports the observation of Winkler (1935) who analysed specimens of uterus in uterine inertia and found a reduction in the potassium content. The recent observation of Cort and Cort (1957) supports this view, that there is loss of cellular potassium in prolonged labour. Such changes were also noticed clinically in the form of bowel and bladder paresis and paralysis, changes in electrocardiograms, bradycardia and skeletal muscle weakness. At present work is being carried out to correlate the electrocardiographic changes in labour with potassium excretion values.

In cases of normal labour which ended in a normal delivery the changes in the electrolyte balance were found to be slight and transient. Thus the stress phenomenon in such cases was within physiological limits.

In cases of prolonged labour the changes were more marked. The greatest changes occurred within 48 hours after the onset of labour with a sharp drop in the plasma sodium level and a similar concomitant sharp rise in the urine potassium excretion. If the prolonged labour ended in a normal delivery the levels returned to nearly normal limits within seventy-two hours. But in cases which terminated in an operative delivery the changes in the plasma sodium levels and the urine potassium levels persisted till the 4th day of the post-operative period. Thus prolonged labour, especially when terminating in an operative delivery, subjects the patient to a state of pathological stress as evidenced by the pathological changes both in extent and duration of the electrolyte balance of the body fluids.

In cases of operative delivery, when intravenous fluids were given, the clinical manifestations of electrolyte changes were less marked as compared to the cases in which no intravenous fluids were given. When blood was given, though the electrolyte changes persisted, clinical features were quickly stabilised (Table III and IV).

Conclusion

With the help of the above results it can be presumed that labour whether normal or abnormal can bring about a state of stress by increased mineralo-corticoid activity with resultant changes in the electrolyte balance, thus leading to patho-

logical changes in the function of the cellular unit which can be appreciated clinically. Normal labour can be regarded as cause of stress within physiological limits. But abnormal labour and abnormal delivery can be regarded as causes of stress causing pathological changes.

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