ELECTROLYTE DYNAMICS AFTER INTRA-UTERINE INJECTION OF HYPERTONIC SALINE FOR M.T.P. IN SECOND TRIMESTER

by

DAMAYANTI SHARMA,* M.D.

USHA VERMA** M.D.

and

A. S. SAINIA,*** Ph.D.

Introduction

Intrauterine hypertonic saline for M.T.P. in second trimester was originally described by Aburel (1937). Due to its effectiveness (success rate of 93%), and simplicity this technique became popular in Japan. This led to its use by non-specialists under unfavourable conditions resulting in more morbidity and mortality (1946-52). Since then this method has largely been banned in Japan.

It was again revived in 1960's in America and became popular all over the world. Berger *et al* (1974) surveyed maternal mortality associated with legal abortions in New York. In this study 49,474 abortions were induced by saline. Total of 11 deaths i.e. 19.5 per 100,000 occurred. 50% of deaths were due to sepsis and one third were due to complications of pre-existing diseases like renal, cardiac and pulmonary. One death was due to intravascular injection of saline. Cameron and Dayon (1966) described the pathophysiology of 2 deaths from intrauterine saline as due to brain oedema and haemorrhagic softening characteristically seen in severe hypernatraemia. Only few studies have been done on the development of hypernatraemia because of electrolyte disturbances after saline. Hence this study was undertaken at Medical College Hospital, Rohtak in 1975. The object of this study was to determine whether any electrolyte changes occur in blood or urine during intra-uterine saline.

*Lecturer, Department of Obstetrics and Gynaecology.

**Professor and Head of the Department of Obstetrics and Gynaecology.

***Professor of Biochemistry.

Medical College and Hospital, Rohtak.

Accepted for publication on 21-3-79.

Material and Methods

Twenty-five patients booked for second trimester termination by intra-amniotic hypertonic saline were studied for their serum and urinary electrolyte changes, after a detailed initial general and pelvic examination to rule out any cardiac, pulmonary or renal pathology. Special investigations undertaken were the estimation of serum sodium and potassium, before instillation, half an hour and 8 hours after the instillation of the saline. The chemical estimations were done by Flame photometric method. The intra-aminiotic saline injection was done after due precautions and the patients were observed for B.P., pulse, vomiting, dizziness and other symptoms in the post instillation period, till they aborted. Follow up of the cases was at 1 week or earlier, if they had any complaints.

Observation and Discussion

All patients except one aborted within

24 hours of instillation. There was one injection failure in a patient whose pregnancy was 14 weeks. The amount of 20% saline used varied from 50-200 mls.

Table I shows that there is an increase in serum sodium level half an hour after the instillation from 5 to 7.5 mEq/L. The levels did not exceed the maximum normal limit i.e. 170 mEq/L as reported by John *et al* (1966) and Visthsitpriza (1965), beyond which symptoms of hypernatraemia invariably result. Eight hours after the injection the serum sodium level returned either to preinjection value or almost near to it. Serum potassium did not alter significantly during the period of observation. The excretion of urinary sodium rose 3 times after half an hour (from 53.6 to 155 mEq/L) and eight hours later the excretion was still high viz. 142 mEq/L. Urinary potassium level fell from 73.4 mEq/L to 29.4 mEq/L after half an hour. It was still 42 mEq/L 8 hours later.

These values are in accordance with those reported in the literature. King

TABLE I

Serum Electrolytes in mEq/L

	Before injection		½ an hr. after injection		8 hrs. after injection	
	Sodium	Potassium	Sodium	Potassium	Sodium	Potassium
Mean	135.6	4.3	144.7	3.2-5.4	138.0	4.1
Range	130-147	3.5-6.5	135-155	4.4	130-145	3.4-4.8
S.D.	±5.5	±0.65	±5.10	±.55	±4.73	±.42
			URINARY ELECTROLYTES IN mEq/L			
Mean	53.66	73.4	155	29.4	142	42
Range	30.88	31-105	132-212	15-60	75-198	20-90
S.D.	±25.4	±23.0	±22.17	±13.26	±38.0	±19.24

Serum Sodium:
- Before injection/½ hr. after injection : H.S. ($p<0.001$)
- Before injection/8 hrs. after injection : N.S. ($p>0.05$)
- ½ hr. after injection/8 hrs. after injection : H.S. ($p<0.001$)

Serum Potassium:
- Before injection/½ hr. after injection : N.S. ($p>0.005$)
- Before injection/8 hrs. after injection : N.S. ($p>0.05$)
- ½ hr. after injection/8 hrs. after injection : N.S. (>0.05)

Urine Sodium:
- Before injection/½ hr. after injection : H.S. ($p<0.001$)
- Before injection/8 hrs. after injection : H.S. ($p<0.001$)
- ½ hr. after injection/8 hrs. after injection : H.S. ($p<0.001$)

Urine Potassium:
- Before injection/½ hr. after injection : H.S. ($p<0.001$)
- Before injection/8 hrs. after injection : H.S. ($p<0.001$)
- ½ hr. after injection/8 hrs. after injection : S. (p 0.005)

et al (1960) used 20% saline with radio labelled sodium and found that the sodium began to appear in serum within 30 minutes and reached equillibrium in blood and urine 8 hours after the instillation. There was no significant change in serum sodium concentration throughout the period of observation. Weingold *et al* (1965) estimated serum and potassium levels after saline. They found no significant change in urinary and serum sodium and potassium level 24 hours after the injection. However, they did not study the values in earlier phases of the post injection period. Anderson and Turnbull (1968) studied 7 patients of 20% saline injection and found biphasic rise in serum sodium levels to 147 mEq/L and 160 mEq/L, 2-3 hours after injection. The levels returned to normal 4-8 hours later. 60-70 per cent of the sodium injected was excreted in urine by the time of abortion. Pathak (1968) estimated blood electrolytes every 2 hours for the first 8 hours and every 4 hours for the next 16 hours in patients of saline injection. He found no appreciable changes. Urinary sodium rose while potassium showed a steady fall. Frederic *et al* (1971) estimated serum sodium levels in 11 patients of 20% saline abortion and observed that the serum level ranged from 135 mEq/L to 145 mEq/L 30 minutes after the injection. In his series there was no consistent pattern of changes in serum sodium levels. However, in urine there was a three-fold increase in sodium excretion after first 8 hours and a two fold increase in next 8 hours.

Mechanism of sodium excretion after saline may be that sodium causes an increased osmolar pressure in aminiotic fluid which causes a shift to the aminiotic cavity with resulting hypovolumia. This raises osmotic pressure of plasma in the mother resulting in antidiuresis. This preliminary hypovolumia changes into hypervolumia which prevents the development of hypernatraemia. This change in blood volume and sodium ions can be handled with ease by normal cardio-renal system. Apparently even though the compensatory system can be overloaded with subsequent rise in serum sodium to above normal or even critically high levels (170 mEq/L), invariably permanent damage may follow. That is why minimum effective dose of saline i.e. 40 G of sodium chloride should be used with all precautions, to ensure slow injection to avoid intravascular injection and to withhold if there is appearance of any sign or symptoms suggestive of hypernatraemia, after careful selection of the patients. Heart disease, chronic renal disease pulmonary disease and diabetes are contraindications for its use. It should be avoided in patients with toxaemia who cannot exhibit normal natriuresis after a sodium load (Menzies and Hawkins, 1968). In the present study there was no maternal death. None of the patients developed signs or symptoms suggestive of hypernatraemia; there was no accidental intravascular injection of saline. Complications of hypernatraemia can be avoided when this method is used with due precautions.

Summary and Conclusion

There was no significant increase in serum sodium level during the period of observation, while the urinary sodium rose 3 times half an hour after instillation and it was still high after 8 hours. The potassium was retained during sodium excretion.

References

1. Aburel, E.: Quoted by Fuchs, F. Advances in Obstetrics and Gynaecology, page 258 Edited by Marcus and Marcus

2. Anderson, A. B. M. and Turnbull, A. C.: Acta Obstet. Gynec. Second. 47: 1, 1968.
3. Cameron, J. M. and Dayon, A. D.: Brit. Med. J. 1: 1010, 1966.
4. Frederic, D., Frigoletto, M. D. and Thomas, B., Pokaly, M. D.: Obstet. Gynec. 38: 637, 1971.
5. John, W. C., Johnson, Cushner, I. M. and Stephen, N. L.: Am. J. Obstet. Gynec. 94: 225, 1966.
6. King, T. M., Friedman, J.C.M. (1964). Quoted by Berkowetz, R. L. Clin. Obstet Gynec. 14: 166, 1971.
7. Manzies, D. M. and Hawkins, D. P.: J Brit. C'wlth. 75: 215, 1968.
8. Pathak, J. N.: Am. J. Obstet. Gynec 100: 513, 1968.
9. Sitprija, V. and Sitprija, S.: Obstet Gynec. 26: 799, 1965.
10. Weingold, A. B., Seigal, S. and Stone, M.: Obstet. Gynec. 26: 622, 1965.