Indian J. Psychiat. (1982), **24**(2), 169—175

SERUM POTASSIUM CHANGES WITH E. C. T.

G. D. SHUKLA[1],
S. L. SRIVASTAVA[2],
A. KAUR[3],
D. D. VARMA[4]
L. D. DOSHI[5]

SUMMARY

Serum potassium changes with direct E.C.T. and with modified E.C.T., given under three anaesthetic (thiopentone, diazepam and propanidid) and relaxant (suxamethonium) combinations were studied in 25 patients. Ten patients each, undergoing minor surgical procedures, acted as controls for these three combinations.

Modified E.C.T. caused a rise in serum potassium, which, being highest at 3 minutes, was sustained well beyond 10 minutes. Direct E.C.T. caused maximum rise within one minute which, however, came down rapidly. The rise following modified E.C.T. was one and half times more compared to that occurring in controls given only drugs but not E.C.T., in all the three anaesthetic-relaxant combinations. The rise from pre-induction level was maximum following diazepam and minimum with propanidid, thiopentone falling in between. The rise from pre-relaxant level, on the other hand, was maximum with thiopentone and minimum with propanidid.

Serum potassium changes occurring during and after E.C.T. have recieved attention only recently (Haw, 1972; Valentin et al., 1973; Bali, 1975; Mehta, 1977 and Mulay et al., 1979). The results of these studies vary considerably, probably due to varied methodology with respect to case selection, method of induction of anaesthesia and the timings of blood samples. Theoretical and clinical significance of the subject is plenty but the literature is scanty, varied and far from conclusive. It was therefore thought worthwhile to study the serum potassium changes during and after E.C.T., given under three anaesthetic agents, viz., thiopentone, diazepam and propanidid; using suxamethonium as the muscle relaxant.

MATERIAL AND METHODS

Selection of cases and E.C.T. procedures:

Study group: The work was carried out in the psychiatric clinic of M.L.B. medical College, Jhansi (U.P.), India. All the cases subjected to E.C.T., after a thorough physical examination and appropriate laboratory investigations to determine their suitability for the procedure, during a period of 8 months were included in the study. However, only 25 of these were finally analysed as the rest did not have treatments under all the anaesthetic-relaxant combinations planned or the requisite number of blood samples could not be collected.

The patients received E.C.T. thrice a week, in the forenoon, by the bitemporal electrode placement technique, giving the current at 90-110 Volts for 0.5 to 1 second. In the direct E.C.T. they were treated without any premedication or subsequent oxygenation. In the modified technique, the patients were put under anaesthesia using any one of the three intravenous inducing agents-thiopentone (5 mg/kg), diazepam (0.5 mg/Kg) or propanidid (7 mg/Kg). Atropine sulphate (0.6 mg/Kg) was given

[1]Lecturer in Psychiatry
[2]Reader in Anaesthesiology
[3]Resident in Anaesthesiology
[4]Lecturer in Anaesthesiology
[5]Reader in Bio-chemistry

M.L.B. Medical College, Jhansi (U.P.)

mixed/alongwith the inducing agent. Suxamethonium (1 mg/Kg) was given through the same needle but from a separate syringe. All the cases were subjected to: (1) direct E.C.T., (2) modified E.C.T. under thiopentone + atropine + suxamethonium, (3) modified E.C.T. under diazepam + atropine + suxamethonium, and (4) modified E.C.T. under propanidid + atropine + suxamethonium on subsequent E.C.T. days.

Control group: For all the anaesthetic-atropine suxamethonium combinations, control groups of 10 patients each, undergoing minor surgical procedure were studied. These patients were induced with one of these combinations and all the blood samples were collected before the commencement of the operation.

Collection of blood samples and estimation of serum potassium:

With direct E.C.T., blood samples were collected before and 1, 3, 5 and 10 minutes after the procedure. With modified E.C.T., these were taken just before induction, just before relaxant and 1, 3, 5 and 10 minutes after E.C.T. In the controls, samples were collected just before induction, just before relaxant and 1, 3, 5 and 10 minutes after relaxant.

Drugs, if administered, were injected in the ante-cubital vein on one side while the blood samples were collected by a dry and sterile syringe, using a 18 gauge needle, from the ante-cubital vein of the other side. Care was taken to avoid exercise of the patient and/or his arm prior to the collection of blood. Minimal occlusion of the vein was used in order to prevent a rise in serum potassium on this account (Brown et al., 1970). Five millilitres of blood were drawn and transferred to plain and sterile vial and the estimations were carried out by flame photometry (Wootton, 1964) within two hours.

RESULTS

Serum potassium changes in the control group following induction and relaxant: Serum potassium concentrations decreased following induction, both with thiopentone and diazepam, though the fall was less marked with the latter. Propanidid, on the other hand, caused a slight rise. Following suxamethonium, the levels increased following induction with all the three anaesthetic agents-reaching a maximum three minutes after its administration. However, the rise was statistically significant only in cases induced with thiopentone ($p < 0.05$). Thereafter, there was a decline in serum potassium levels in all the cases, though it could not reach the baseline even after 10 minutes with any of the three inducing agents (Table I).

TABLE I—*Mean serum potassium levels (m Eq/L) in control group following thiopentone/diazepam/propanidid and suxamethonium.*

Inducing agent	Pre-induction	Pre-relaxant	Post-relaxant 1 min.	3 min.	5 min.	10 min.
Thiopentone	4.02 ±0.70	3.73 ±0.61	3.93 ±0.74	4.46 ±0.71	4.09 ±0.67	4.04 ±0.69
	t_1	1.32	0.09	1.39	0.23	0.10
	t_2	..	0.87	2.47*	1.29	1.10
Diazepam	3.78 ±0.67	3.70 ±0.73	3.82 ±0.69	4.24 ±0.61	3.97 ±0.64	3.94 ±0.61
	t_1	0.32	0.13	1.61	0.63	0.55
	t_2	..	0.55	1.80	0.87	0.80
Propanidid	3.99 ±0.47	4.09 ±0.52	4.13 ±0.53	4.39 ±0.53	4.16 ±0.54	4.09 ±0.52
	t_1	0.45	0.64	1.79	0.77	0.45
	t_2	..	0.15	1.28	0.29	0.00

*Significant ($p < 0.05$)

t_1 = t value of the difference from pre-induction level.

t_2 = t value of the difference from pre-relaxant level.

Serum potassiam changes with modified E.C.T.: Here also, there was a fall in serum potassium levels after induction with thiopentone and diazepam, significantly so with the former ($p < 0.05$). Propanidid caused a slight rise immediately after its administration. Following suxamethonium, the serum potassium levels rose with all the three drug combinations, though they still remained less than the pre-induction levels with thiopentone and diazepam. Following E.C.T. there was a marked rise in serum potassium in all the drug combinations. The maximum rise was seen in the three minute sample after which the levels declined though they still remained higher than the baseline even after 10 minutes following E.C.T. (Table II).

At three minutes following E.C.T., the serum potassium levels were significantly raised ($p < 0.05$), compared to both pre-induction and pre-relaxant levels, in all the three drug combinations. After this, however, the difference remained significant only with thiopentone where the potassium levels continued to be significantly higher compared to the pre-relaxant levels.

From a close scrutiny of the table III, it becomes clear that the percentage of peak rise in serum potassium from pre-induction level was nearly one and half times more in the study group (receiving E.C.T.) compared to controls (not receiving E C T.) in all the three drug combinations. Further, the rise from pre-induction level was maximum following diazepam both in patients with and without E.C.T. (18.76% and 12.16% for study and control groups respectively) and minimum with propanidid (15.25% and 10.03%), thiopentone falling in between (17.11% and 10.94%). Lastly, the degree of peak rise was inversely proportional to the pre-relaxant levels of potassium, the inducing agent causing lowest mean pre-relaxant level being followed by maximum percentage rise. Thus, the peak rise from the pre-relaxant level was maximum with thiopentone (19.89% and

TABLE II—*Mean serum potassium levels (m Eq/L) following modified E.C.T. under thiopentone/diazepam/propanidid and suxamethonium.*

Inducing agent	Pre-induction	Pre-relaxant	Post-relaxant	After E.C.T.			
				1 min.	3 min.	5 min.	10 min.
Thiopentone	3.41 ±0.51	3.12 ±0.67	3.34 ±0.89	3.42 ±0.64	3.99 ±0.67	3.54 ±0.59	3.47 ±0.56
	t_1	2.09*	0.35	0.06	3.49*	0.31	0.75
	t_2	—	1.57	1.53	4.61*	2.33*	2.07*
Diazepam	3.41 ±0.51	3.28 ±0.91	3.32 ±0.90	3.46 ±0.84	4.05 ±0.49	3.62 ±0.56	3.51 ±0.52
	t_1	0.70	0.50	0.50	4.54*	1.33	0.60
	t_2	—	0.20	0.75	3.90*	0.62	1.15
Propanidid	3.41 ±0.51	3.50 ±0.54	3.52 ±0.54	3.56 ±0.54	3.93 ±0.49	3.55 ±0.51	3.52 ±0.56
	t_1	0.60	0.80	1.00	3.68*	1.00	0.73
	t_2	—	0.20	0.33	2.96*	0.33	0.13

* Significant (<0.05).

TABLE III—*Comparison of mean peak rise of serum potassium concentration (m Eq/L) from pre-induction and pre-relaxant values in study and control groups, i.e. with and without E.C.T.*

Inducing agents	Groups	Rise from pre-induction conc. (m Eq/L)				Rise from pre-relaxant conc. (m Eq/L)			
		Pre-ind. conc.	Peak conc.	Mean rise	Mean %rise	Pre-relax conc.	Peak rise	Mean rise	Mean %rise
Thiopentone	A	4.02	4.46	0.44	10.94	3.72	4.46	0.74	19.89
	B	3.41	3.99	0.58	17.11	3.12	3.99	0.87	27.88
Diazepam	A	3.78	4.24	0.46	12.16	3.70	4.24	0.54	14.59
	B	3.41	4.05	0.64	18.76	3.28	4.05	0.77	23.47
Propanidid	A	3.99	4.39	0.40	10.03	4.09	4.39	0.30	7.33
	B	3.41	3.93	0.52	15.24	3.50	3.93	0.43	12.28

A Control group; B Study group

27.86% for control and study groups respectively) and minimum with propanidid (7.33% and 12.28%), diazepam falling in between (15.59% and 23.47%). Here again the changes were much more pronounced in patients given E.C.T.

Serum potassium changes with direct E.C.T.: Following direct E.C.T. there was a markedly significant rise in serum potassium levels ($p<0.01$). The rise was maximum in the sample taken one minute thereafter the levels fell rapidly so much so that it became less than the baseline value within 5 minutes. This declining trend was maintained in the 10 minute sample as well (Table IV). The mean peak rise, occurring after one minute was to the tune of 21.43%.

TABLE IV—*Mean serum potassium levels (m Eq/L) following direct E.C.T.*

Before E.C.T.	After E.C.T.			
	1 min.	3 min.	5 min.	10 min.
3.41	4.14	3.52	3.37	3.36
±0.51	±0.75	±0.73	±0.55	±0.54
	4.11*	0.65	0.44	0.42

*Significant ($p<0.01$).

DISCUSSION

In contrast to numerous studies on the other aspects of E.C.T., nicely reviewed by Fink (1979) and Weiner (1979), serum potassium changes have received scarce attention as is evidenced by the paucity of literature on the subject (Haw, 1972; Valentin et al., 1973; Bali, 1975; Mehta, 1977 and Mulay et al., 1979). There have been marked variations in the methodology adopted by these workers. The timings of blood samples varied widely in different studies. Haw (1972) took samples before induction, after suxamethonium and after re-establishment of spontaneous breathing following E.C.T. Valentin et al. (1973) carried out estimations 3 and 7 minutes after suxamethonium. This delay would give time for the redistribution of the initial potassium raising effect of the relaxant thus giving rise to misleading results. Bali (1975) and Mulay et al. (1979) took blood samples before induction, before relaxant and 1, 3, 5 and 10 minutes after cessation of suxamethonium fasciculations. Since the serum potassium changes are short lived and show rapid variations, even a slight difference in the timing of the sample would give rise to major difference in the ultimate results.

Further, all these workers took separate groups of patients for studying the serum potassium changes following modified E.C.T. given under different anaesthetic agents, thus making it difficult to rule out the inter-group differences in serum levels. In the present study the same cases (study group) were subjected to direct and modified E.C.T. under the three anaesthetic agents on consecutive turns. Since inducing agents (Stevenson, 1960; List, 1967; Anand et al., 1972; Gal and Malit, 1972; Bali et al., 1973; Bali and Dundee, 1974 and Phadke et al., 1978) and suxamethonium (Paton, 1959; List, 1967; Anand et al., 1972; Bali and Dundee, 1974 and Jassal et al., 1976), on their own, are known to produce changes in serum potassium, the three drug combinations were given to three different age and sex matched control groups of cases undergoing minor surgical procedures.

Serum potassium changes following induction: The results of the present study, with respect to thiopentone and diazepam, are similar to those of others who too found a fall in serum potassium following thiopentone (List, 1967; Gal and Malit, 1972; Haw, 1972; Bali et al., 1973; Bali and Dundee, 1974 and Phadke et al., 1978) and diazepam (Bali and Dundee, 1974 and Phadke et al., 1978). However, Anand et al. (1972) found no such change with thiopentone. The fall with thiopentone was to the extent of 0.3 m Eq/L which was almost same as that reported by Bali and Dundee (1974). Gal and Malit (1972) reported a somewhat lesser decline. The rise in potassium after propanidid, observed by us, is in line with the findings of Bali et al. (1973), Bali and Dundee (1974) and Phadke et al. (1978).

Serum potassium changes following suxamethonium: Suxamethonium has been found to cause a significant rise in serum potassium levels (Paton, 1959; Stevenson, 1960; Galindo and Davis, 1962; Belin and Karleen, 1966; List, 1967; Tolmie et al., 1967; Striker and Morrow, 1968; Evers et al., 1969; Roth and Wuthrich 1969; Weintraub et al., 1969; Cooperman et al., 1970; Anand et al., 1972; Basu et al., 1973; Bali and Dundee, 1974; Jassal et al., 1976 and Mulay et al., 1979). The peak rise in the present series occurred after 3 minutes while most of the other workers found it to occur later than this. Basu et al. (1973) and Jassal et al. (1976) found peak rise to occur after 14 and 5 minutes respectively. The rise was maximum when thiopentone had been used as the inducing agent and minimum with propanidid, diazepam falling in between. This observation of ours is in sharp contrast to those of others who found the rise to be less with thiopentone and more with propanidid (Roth and Wuthrich, 1969; Weintraube et al., 1969; Haw, 1972; Bali and Dundee, 1974; Bali, 1975 and Mulay et al., 1979).

The peak rise of serum potassium in our series (between 0.40 and 0.44 m Eq/L from the pre-induction level and between 0.30 and 0.74 m Eq/L from the pre-relaxant level) was within the range reported by others who observed variations ranging from as little as 0.1 m Eq/L (Anand et al., 1972 and Bali and Dundee, 1974) to as high as 2.5 m Eq/L (Weintraub et al., 1969), most of them reporting variations to the tune of around 0.5 m Eq/L (Paton, 1959; Basu et al., 1973 and Jassal et al., 1976). Suxamethonium induced hyperkalaemia has profound influence upon cardio-vascular stability, the incidence of cardiac arrhythymias including ventricular fibrillation being 20% higher following suxamethonium than that following the use of other relaxants (Davis et al., 1964); Belin and Karleen, 1966; Lowenstein, 1966; List, 1967; Roth and Wuthrich, 1969 and Cooperman et al., 1970).

Serum potassium changes following modified E.C.T.: Like our findings, all the workers have found a significantly

greater rise in serum potassium following modified E C.T. compared to the rise seen after the administration of inducing agent and suxamethonium but no E.C.T. (Bali, 1975 and Mulay et al., 1979). Further, all these workers have found the rise from the pre-relaxant levels to be more marked than that from the pre-induction levels when thiopentone or methohexitone was used as the anaesthetic agent. However, with propanidid, the rise was more from the pre-induction levels than from pre-relaxant levels (Table V). No report is available to compare the changes occurring following E.C.T. under propanidid.

TABLE V—*Mean peak rise (m Eq/L) from pre-induction and pre-relaxant levels following modified E C.T in different studies.*

Authors	Inducing agent	Mean peak rise in serum potassium	
		From pre-induction level	From pre-relaxant level
Bali and Dundee (1974)	Thiopentone ..	0.35	0.50
	Methohexitone	0.36	0.50
Mehta (1977) ..	Thiopentone ..	0.61	0.71
	Diazepam ..	0.75	0.81
Mulay *et al.* (1979)	Thiopantone	0.26	0.42
Present study (1982) ..	Thiopentone	0.44	0.74
	Diazepam ..	0.46	0.54
	Propanidid ..	0.40	0.30

Like the present study, Bali (1975), Mehta (1977) and Mulay et al. (1979) found modified E.C.T. to cause a higher rise in serum potassium than that observed in patients undergoing minor surgical procedures under the same anaesthetic-relaxant combination. As regards the mechanism behind this difference, one would have to agree with the suggestion of Bali (1975) that when a patient receives E.C.T., the synchronous contractions of all the voluntary muscles cause the muscle venous blood-rich in potassium following suxamethonium administration-to be pumped out into the circulation, thus causing a greater rise in the serum potassium levels.

However, contrary to the observations of Bali (1975) and Mehta (1977) who found lesser rise in serum potassium following E C.T. given under methohexitone and diazepam respectively, in the present study, the rise was more pronounced and sustained following E.C.T. under thiopentone as compared to that seen when the procedure was carried out under diazepam or propanidid. This they attributed to the 'protective' effect of thiopentone against the suxamethonium induced potassium rise. Our results could be better explained by the observation of Haw (1972) that the extent of rise in serum potassium was invariably related inversely to the pre-relaxant level. Thus under thiopentone, with minimal pre-relaxant level, the rise following E.C.T. was maximum. While using propanidid, with a maximum pre-relaxant level, the rise following E.C.T. was minimal. Under diazepam, with intermediate pre-relaxant level, E.C.T. produced an intermediate rise in serum level.

Serum potassium changes following direct E.C.T.: Our finding that direct E.C.T. caused a quick rise, followed by a quick fall in serum potassium is in conformity with those reported by other workers (Bali, 1975; Mehta, 1977 and Mulay et al., 1979). The extent of rise too, in the present study, fell well within the range of values reported by these workers.

Thus the changes in serum potassium levels following direct and modified E.C.T., observed by us, support the conclusions of Bali (1975) and Mulay et al. (1979) that

direct E.C.T. caused a quick rise followed by a quick fall in serum potassium levels while modified E.C.T. caused a slow but relatively sustained rise.

REFERENCES

ANAND, J. S., KOTWAL, S., CHUGH, D., MEHTA, J. H. AND PRADHAN, A. G. (1972). Serum potassium changes following succinylcholine with different inducing techniques. Indian J. Anaesth., 20, 109.

BALI, I. M., DUNDEE, J. W. AND ASSAF, R. A. E. (1973). Immediate changes in plasma potassium induced by intravenous inducing agents. Brit. J. Anaesth., 45, 1238.

BALI, I. M. AND DUNDEE, J. W. (1974). Immediate changes in plasma potassium, sodium and chloride induced by intravenous inducing agents. Brit. J. Anaesth., 46, 803.

BALI, I. M. AND DUNDEE, J. W. (1974). Sources of potassium. Brit. J. Anaesth., 46, 803.

BALI, I. M. (1975). The effects of modified E.C.T. on plasma potassium. Brit. J. Anaesth., 47, 398.

BASU, P. C., AGGARWAL, K. C. AND BHATNAGAR, N. S. (1973). Suxamethonium induced hyperkalaemia in burns and trauma. Indian J. Anaesth., 21, 259.

BELIN, R. P. AND KARLEAN, C. I. (1966). Cardiac arrest in burned patient following suxamathonium. Anaesthesiology, 27, 516.

BROWN, J. J., CHINN, R. H., DAVIES D. L., FRASER, R., LEVER, A. F., RAE, R. J. AND ROBERTSON, J. I. S. (1970). Falsely high plasma potassium values in patients with hyperaldesteronism. Brit. Med. J., 2, 18.

COOPERMAN, L. H., STROBEL, G. E. AND KENNELL, E. M. (1970). Massive hyperkalaemia after succinylcholine. Anaesthesiology, 732, 161.

DAVIS, L. D., HELMER, P. R. AND MURPHY, Q. R. (1964). Role of potassium in cyclo-propaine-epinephrine ventricular tachycardia. Anaesthesiology, 25, 54.

EVERS, W., RACZ, W. B., AND DOBKIN, A. B. (1969). A study of plasma potassium changes after a dose of suxamethonium. Canad. Anaesth. Soc. J., 16, 273.

FINK, M. (1979). Convulsive Therapy. New York : Raven Press.

GAL, T. J. AND MALIT, L. A. (1972). The influence of ketamine induction on potassium changes and fasciculation after suxamethonium. Brit. J. Anaesth., 44, 1077.

GALINDO, A. H. AND DAVIS, T. B. (1962). Succinylcholine and cardiac excitability. Anaesthesiology, 23, 32.

HAW, M. E. (1972). Variations in serum potassium during E.C.T. in patients anaesthetised with thiopentone and methohexitone. Brit. J. Anaesth., 44, 707.

JASSAL, D. S., SINGH, B. AND SINGH, P. (1976). Electrolyte changes in blood after single dose of suxamethonium. Indian J. Anaesth., 24, 41.

LIST, W. F. (1967). Serum potassium changes during induction of anaesthesia. Brit. J. Anaesth., 39, 480.

LOWENSTEIN, E. (1966). Succinylcholine administration in the burn patient. Anaesthesiology, 27, 494.

MEHTA, R. K. (1977). The Effect of Modified E.C.T on Serum Potassium. M. D. Thesis, Submitted to Kanpur University (Unpublished).

MULAY, .V P., PANDYA, U. AND DESHPANDE, A. M. (1979). Changes in plasma potassium after E. C. T. Indian J. Anaesth., 27, 119.

PATON, W. D. M. (1959). The effects of muscle relaxants other than muscular relaxation. Anaesthesiology, 20, 453.

PHADKE, S. N., DESHPANDE, M. S. AND KATARIA, P. S. (1978). Effect of various intravenous inducing agents on serum electrolytes and blood sugar. Indian J. Anaesth., 26, 217.

ROTH, F. AND WUTHRICH, H. (1969). The clinical importance of hyperkalaemia following suxamethonium. Brit. J. Anaesth., 41, 311.

STEVENSON, D. E. (1960). Changes in blood electrolytes of anaesthetised dogs caused by suxamethonium. Brit. J. Anaesth., 32, 364.

STRICKER, T. W. AND MORROW, A G. (1968). The effect of succinylcholine on the level of serum potassium in man. Anaesthesiology, 29, 214.

TOLMIE, J. D.; JOYCE, T. H. AND MITCHELL, G. D. (1967). Succinylcholine danger in burned patients. Anaesthesiology, 28, 467.

VALENTIN, N., SKOVSTED, P. AND DANIELSEN, B. (1973). Plasma potassium following suxamethonium and E.C.T. Acta Anaesth. Scand., 17, 197.

WEINER, R. D. (1979). The psychiatric use of electrically induced seizures. Amer. J. Psychiat., 136, 1507.

WEINTRAUBE, H. D., HEISTERKAMP, D. V. AND COOPERMAN, L. H. (1969). Changes in plasma potassium concentration after depolarising blockers in anaesthetised man. Brit. J. Anaesth., 41, 1048.

WOOTTON, I. D. P. (1964). Microanalysis in Medical Biochemistry. London : J. and A. Churchill.