DIURESIS DURING FLUID INFUSION : BUFFER NERVE AND SPINAL INFLUENCES ON IT

T.P. SURESH AND K.N. SHARMA*

Department of Physiology, Veterinary College, University of Agricultural Sciences, Hebbal, Bangalore-560 024

and

Department of Physiology and Medical Electronics, St. John's Medical College, Bangalore-560 034

Summary: The rate and cumulative volume of diuresis were measured sequentially for each incremental infusion dose of 5 *ml/kg* body weight till a 100 *ml/kg* or more dose was reached. Normal saline (NS), Ringer-Locke (RL) and tender coconut water (TCW) were infused in three groups each of paraldehyde (PLD), and chloralose and urethane (C & U) anaesthetised dogs. The slow infusion rate of about 0.5 *ml/kg/min* was used. The RL infusion was repeated in vagotomised and/or carotid sinus (CS) denervated dogs and spinal dogs with or without intact vagi.

During the NS and RL infusion schedules in PLD anaesthetised dogs produced much less urine than C & U groups. The order of minimum to maximum diuretic effect caused by these fluids were RL,NS and TCW in PLD groups and NS, TCW and RL in C & U groups. The study indicates that the type of anaesthesia and the composition of infusion fluid determines the rate of infusion induced diuresis. PLD anaesthesia has antidiuretic effect, which is not overcome by vagotomy. In C & U anaesthetised dogs the vagotomy and CS denervation performed separately greatly increased the rate of infusion induced diuresis but the diuresis largely decreased when combined surgery was performed. The diuresis in spinal dogs was very low, though in the vagotomised-spinal dogs, the rate of diuresis was more than in the spinal dogs.

Key words: fluid infusion, diuresis, buffer nerve, spinal cord, tender coconut water, anaesthetised dogs

INTRODUCTION

Cardiovascular changes during intravenous fluid infusion have been well documented (18,19,31,32). Studies on diuresis during fluid infusion and the possible mechanism of such infusion induced diuresis have also been reported (14,19,31). However, the methods used in the above studies had no relevance to clinical practice regarding the rate, volume and type of the infusion fluid used as also the effects of anaesthesias. To fill up this lacuna, the effect of normal saline (NS), Ringer-Locke solution (RL) and tender coconut water (TCW) (29,30) infusion on the rate and volume of diuresis was studied in paraldehyde (PLD), and chloralose and urethane (C & U) anaesthetised dogs receiving upto 100 *ml/kg*

*Present address: Department of Physiology, University College of Medical Sciences, Ring Road, New Delhi-110016.

body weight or more of the above fluids at a slow rate. Many workers (8,13) have suggested that buffer nerves may play a role in diuretic response in dogs. To evaluate the possible role of vagi, carotid sinus (CS) nerves and spinal cord in infusion induced diuresis, the RL infusion was repeated in vagotomised and/or CS denervated and spinal dogs.

MATERIALS AND METHODS

Seventy-eight mongrel dogs (7-20 *kg*) of both sexes in 12 groups as described in detail by Suresh (32) were used. Dogs were kept on *ad libitum* food and water schedules before use. The dogs were offered water before anaesthetic induction. PLD was injected into thigh muscles at 2.2 *ml/kg* body weight. C & U anaesthesia was given one hour after premedication with morphine hydrochloride (1 *mg/kg* body weight). The dogs were anaesthetised to Plane 3 of surgical anaesthesia (7) and maintained at that level by infusing 1 to 2 *ml* of anaesthetic mixture whenever the blinking reflex reappeared. A total of 0.068 to 0.102 g chloralose and 1.02 to 1.52 g of urethane were used per *kg* body weight in various dogs including supplementary doses.

The basal preparations in intact groups (G1 to G6) included intratracheal intubation, femoral artery and vein cannulation. Urinary bladder was catheterised through urethra with a lubricated flexible polyethylene tube (2 mm diameter for male and 5 mm diameter for female). The experimental surgery groups (G7 to G12) in addition underwent surgeries as described in detail by Suresh (32). Appropriate checks were conducted to find out the efficacy of the surgeries. Only young dogs of 1-2 years of age were used for spinal transections as the preliminary studies indicated that aged dogs were susceptible to spinal shock.

The urinary bladder was emptied before the start of the infusion. Urine drops were recorded using drop recorder (E & M Instruments, Houston, Texas, USA, 92-100-70) or a recorder designed and fabricated in our laboratory. The urine collected during each incremental infusion dose (5 *ml/kg* body weight) was measured till 100 *ml/kg* stage or more was reached. The bladder was palpated regularly to ensure that no urine was retained. The cumulative urine volume was calculated by adding together the successive urine volumes produced at each infusion stage. Fresh NS, RL (pH 7.4) and TCW were infused at room temperature via femoral vein at a constant rate (0.5 to 0.9 *ml/kg/min*) as indicated in Table I of Suresh (32). Samples of TCW infused were analysed for pH and electrolytes.

Analysis : The regression curves were fitted to mean rate and mean cumulative urine volume produced at each infusion stage by using orthogonal polynomials. The intergroup comparison of net rate and net cumulative urine volume (net = mean taken across the entire infusion schedule) were done by Bartlett's test as described in Snedecor and Cochran (28).

RESULTS

The rate and cumulative volume of urine produced at selected infusion stages in intact and experimental surgery groups are presented in Tables I, and II. Table III gives net rate and volume of urine produced, R-values (multiple correlation coefficient) of fitted regression curves and intergroup comparison of net rate and volume of diuresis. Figs. 1, 2 and 3 give the regression curves of rate and volume of diuresis. Generally, PLD groups

TABLE I : Rate of urine formation (*ml*) between two successive infusion stages.

G. No.		*Infusion stages (ml/kg body weight)*									
		10	20	30	40	50	60	70	80	90	100
G1	M	0.5	2.7	3.8	8.0	7.8	2.8	2.9	1.8	3.0	2.2
	s	1.0	3.8	4.9	11.9	13.3	4.6	3.1	1.9	3.0	2.3
G2	M	1.0	0.5	0.6	0.4	1.4	2.4	1.6	2.3	12.0	3.9
	s	1.4	0.6	0.5	0.5	1.1	3.1	2.3	2.2	11.8	2.2
G3	M	4.0	7.0	7.8	8.8	13.3	19.8	14.0	13.0	17.3	13.0
	s	3.7	4.4	2.2	5.5	6.9	13.4	4.7	4.1	9.7	9.9
G4	M	1.7	3.8	6.8	10.3	14.0	10.0	10.2	6.5	10.5	12.7
	s	1.2	2.5	5.1	13.9	17.7	10.0	12.5	3.6	13.5	10.7
G5	M	4.0	3.3	5.8	17.2	12.8	22.3	15.8	13.8	17.0	17.2
	s	3.2	1.8	6.6	12.0	9.8	13.7	10.2	8.6	11.0	9.1
G6	M	2.0	4.3	13.0	10.5	14.8	12.8	11.0	9.7	8.3	12.8
	s	4.0	5.5	11.4	9.1	15.0	11.2	10.2	7.0	4.7	4.7
G7	M	1.6	1.1	1.6	1.4	2.3	3.3	4.7	5.0	5.8	8.3
	s	3.4	1.1	1.5	1.3	2.4	4.0	5.5	5.5	5.7	4.4
G8	M	5.5	7.0	12.3	19.2	16.7	25.8	22.7	25.7	22.8	24.3
	s	4.7	6.4	9.2	22.0	14.5	22.8	18.6	19.6	15.1	16.9
G9	M	5.0	8.8	13.0	24.8	27.0	17.0	21.0	17.2	22.8	31.3
	s	4.0	10.0	12.6	15.4	20.8	11.4	17.2	16.8	14.0	21.5
G10	M	1.2	3.4	5.0	6.8	8.0	8.2	5.6	6.0	9.4	10.2
	s	1.6	3.1	5.2	5.5	7.8	6.3	4.2	5.8	11.9	9.8
G11	M	0.5	2.2	0.8	3.5	1.2	2.8	5.0	4.0	2.5	1.2
	s	0.5	1.7	1.0	3.9	1.2	3.5	5.2	6.9	4.3	2.9
G12	M	0.0	0.0	0.0	2.7	4.2	6.2	10.0	5.3	5.3	12.5
	s	0.0	0.0	0.0	4.6	3.5	4.9	11.4	5.9	6.5	9.8

G. No. = Group number; M = Group mean; s = Standard deviation.
For Other abbreviations, see Fig. 1.

July-September 1979
Ind. J. Physiol. Pharmac.

(G1 and G2) produced less urine than C & U groups (G4, G5 and G6) except during TCW infusion (G3, Table I). R-value of the fitted regression curves was high indicating good fit (Table III). Individual variation in diuretic response was high in all the groups.

TABLE II : Cumulative urine production (*ml*) at various stages of infusion.

G. No.		BU	UP DES	Infusion stages (ml/kg body weight)									
				10	20	30	40	50	60	70	80	90	100
G1	M	—	—	1	6	14	30	46	51	57	61	67	7
	s	—	—	2	10	15	40	66	75	60	81	87	88
G2	M	70	—	2	3	4	5	8	13	15	20	45	53
	s	115	—	3	4	5	5	6	9	12	11	30	34
G3	M	33	—	4	15	27	47	70	100	130	155	190	244
	s	33	—	4	4	8	17	22	31	37	35	52	34
G4	M	62	—	3	10	23	42	69	91	113	132	151	173
	s	59	—	2	3	10	31	66	93	117	141	163	182
G5	M	105	—	8	15	27	55	82	119	152	183	217	255
	s	89	—	4	6	16	34	46	60	77	95	112	130
G6	M	58	—	3	10	32	55	82	107	137	156	176	198
	s	63	—	4	13	28	44	62	69	89	102	114	122
G7	M	48	3	3	7	11	15	22	30	41	54	71	88
	s	58	4	4	7	9	8	11	17	27	39	52	63
G8	M	70	19	13	24	48	81	118	167	213	261	304	352
	s	92	14	12	20	34	61	86	130	166	197	226	246
G9	M	78	35	11	27	55	96	150	190	229	263	302	353
	s	62	30	6	18	36	58	85	106	130	150	171	190
G10	M	53	7	3	8	17	29	45	62	73	91	106	130
	s	44	3	3	7	13	22	34	44	49	67	76	97
G11	M	56	11	1	5	6	17	21	28	39	48	55	63
	s	55	12	1	2	1	5	6	8	15	25	32	33
G12	M	39	8	0	0	2	6	16	29	49	60	70	91
	s	32	8	0	0	2	6	8	15	33	37	40	40

G.No. = Group number; — = Not recorded; M = Group mean;
s = Standard deviation; BU = Volume of urine in bladder when bladder was emptied;
UP DES = Total urine production during experimental surgery.
for other abbreviations, see Fig. 1.

Intact groups :

In PLD groups, NS, RL and TCW infusion (G1, G2 and G3) caused 3.6, 2.7 and 10.4 *ml* net urine flow rate and 40.4, 16.8 and 90.8 *ml* net cumulative urine volumes, respectively. Among the three infused, the TCW caused highest diuresis and RL the least. Individual variation was large during NS infusion but small during RL and TCW infusion. The net rate of diuresis duri g NS and RL infusion was similar to the rates seen in spinal dogs (G11 and G12, Table III).

NS and RL infusion to C & U groups (G4 and G5) caused distinctly larger diuresis than in PLD groups (Table III). Their net flow rate and net cumulative urine volume were 8.7, 12.7 and 76.8, 104.9 *ml*, respectively. However, TCW infusion (G6) caused same diuresis as in PLD group (G3, Table III). In contrast to PLD group, the RL caused highest and the NS least diuresis. The rate of diuresis showed a tendency to decrease in

TABLE III : Diuretic response to infusion schedule.

		Intact groups						*Experimental surgery groups*					
		G1	*G2*	*G3*	*G4*	*G5*	*G6*	*G7*	*G8*	*G9*	*G10*	*G11*	*G12*
(i)		3.6	2.7	10.4	8.7	12.7	9.9	3.5	17.5	17.6	6.5	2.3	3.8
(ii)		0.63*	0.67*	0.90	0.85	0.88	0.83	0.95	0.94	0.73	0.79	0.70	0.77
(iii)		40.4	16.8	90.8	76.8	104.9	90.8	32.1	149.0	158.2	53.2	26.6	29.5
(iv)		0.98	0.98	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.99	0.98
(v)	UC G	G11	G2	G7	G1	G12	G10	G4	G6	G3	G5	G8	G9
	M	2.3	2.7	3.5	3.6	3.8	6.5	8.7	9.9	10.4	12.7	17.6	17.6
	UC G	G2	G11	G12	G7	G1	G10	G4	G3	G6	G5	G8	G9
	M	16.8	26.6	29.5	32.1	40.4	53.2	76.8	90.8	90.8	104.9	149.0	158.2

(i) Net urine flow, *ml*/each infusion stage.

(ii) R value of the regression curve representing rate of urine formation during infusion schedule. *Significant at $P<0.05$, others at $P<0.01$.

(iii) Net cumulative urine volume (*ml*) produced during entire infusion schedule.

(iv) R values of the regression curve representing the cumulative volume of urine produced during infusion schedule, *Significant at $P<0.05$, others $P<0.01$.

(v) Intergroup comparison of urine volume; (a) values have been arranged in ascending order, (b) values connected by the same line are similar ($P>0.05$).

G = Group, M = Net volume during the entire infusion schedule.
UR = Net urine flow (*ml*), UC = Net cumulative urine volume (*ml*).
For other obbreviations, see Fig. 1.

later half of the infusion schedule during NS and TCW infusion but showed a plateau during RL infusion (Fig. 1, G4, G5 and G6).

Experimental surgery groups :

In vagotomised dogs under PLD (G7), RL infusion did not cause any diuresis in two dogs and they were not included for calcu ation. In the remaining seven dogs the

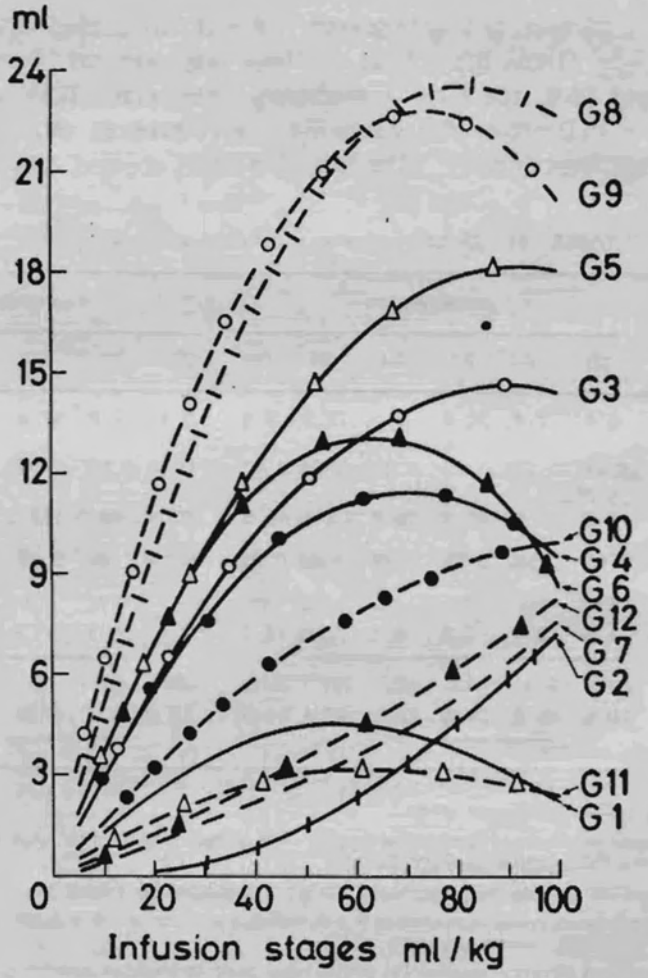

Fig. 1 : Regression lines of rate of diuresis (*ml* per infusion stage) during 0 to 100 *ml* stages of infusion schedule. G1 to G3 and G4 to G6 are intact PLD and C & U anaesthetised dogs receiving NS, RL and TCW infusion respectively; G7 to G12 experimental surgery group receiving RL infusion; G7 and G8 — vagotomised dogs ur.der PLD and C&U anaesthesia, G9, G10, G11 and G12 CS denervated, vagotomised-CS denervated, spinal and vagotomised spinal dogs, respectively.

net urine flow rate and net cumulative urine volume were 3.5 *ml* and 32.1 *ml* respectively which was slightly more than the intact dogs (G2) but was similar to vagotomised spinal dogs (G12, Table III). Vagotomy in C & U groups (G8) in contrast largely increased the diuretic response and caused second highest diuresis among the 12 infusion schedules studies. The urine flow rate and net cumulative urine volume were 17.5 and 149.0 *ml* respectively which was very much higher than the intact group (G5).

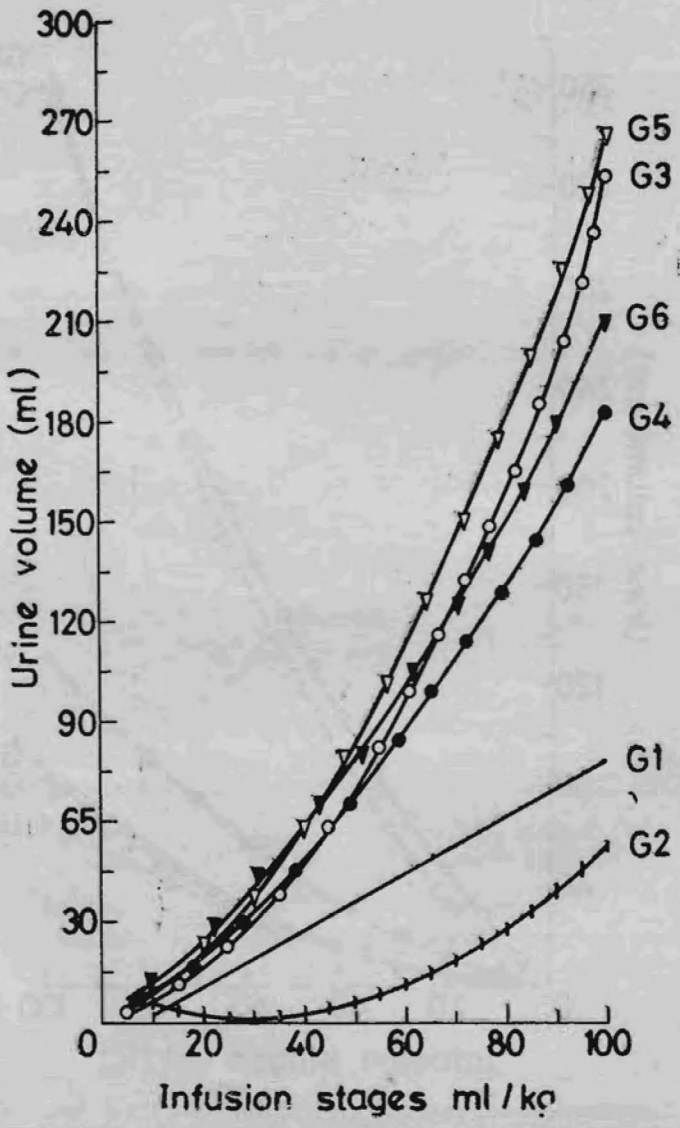

Fig. 2 : Cumulative volume of urine produced (*ml*) during 0 to 100 *ml* stages of infusion schedule in intact dogs. See Fig. 1 for other abbreviations.

CS denervation alone (G9) increased the diuretic response to RL infusion and caused the highest net urine flow rate of 17.6 *ml* and net cumulative urine volume of 158.2 *ml*. The urine flow rate decreased after 70 *ml* infusion stage (Fig. 1). Individual variation was less in this group. CS denervation along with vagotomy (G10) decreased the diuretic response to RL infusion and caused a net urine flow rate of 6.5 *ml*.

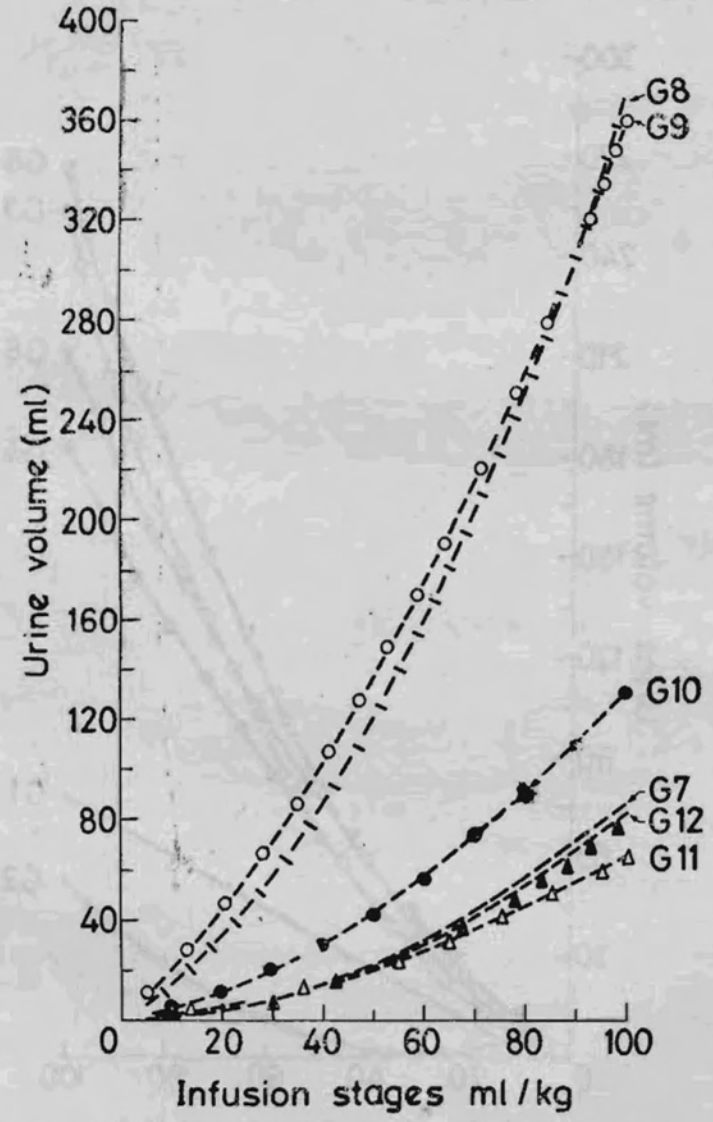

Fig. 3 : Cumulative volume of urine produced (*ml*) during 0 to 100 *ml* stages of infusion schedule in experimental surgery group of dogs. See Fig. 1 for other abbreviations.

Spinal transection either alone (G11) or along with vagotomy (G12) decreased the diuretic response to RL infusion. Two dogs died due to spinal shock and five others died during various stages of infusion. These were excluded from analysis. The net urine flow rate and net cumulative urine volume were 2.3 and 3.8, and 26.6 and 29.5 *ml*, respectively which is very much lower than 12.7 and 104.9 *ml* seen in intact dogs (G5, Table III). Here the diuresis was similar to intact PLD groups (G1 and G2). Vagotomised spinal dogs (G12) produced more urine than the spinal dogs (G11).

DISCUSSION

From the Tables I and III, it is clear that PLD groups (G1, G2, G3 and G7) produced less urine than C & U groups. The net urine flow rate and net urine volume (Table III) during the NS, RL and TCW infusions to PLD and C & U groups indicate that composition of infusion fluid can influence the extent of diuresis. This is in contrast to the same type of renal response reported by Papper *et al.* (18) and Raisz (19) for various types of fluids infused. In C & U groups (G4 and G5) though the NS and RL were infused at the same rate, the RL caused significantly more diuresis, a finding which does not support the report of Jagger *et al.* (6) that the rate of infusion is an important determinant of infusion induced diuresis but supports the above conclusion on the role of the composition of infused fluids. Tables I and III further show that PLD groups (G1 and G2) though received infusion at faster rate than C & U groups (G4 and G5) produced less urine and suggests that anaesthetic agents also determine the extent of diuresis. The mean urine flow rate (observed rate divided by 10 to express as *ml/min* at 50 *ml/kg* infusion stage in intact groups approximately 500 *ml* total dose) was very much less than the rate of 5.2 *ml/min* at 60 min after 500 *ml* saline infusion reported by Levinsky and Lalone (14).

Low rate of diuresis in PLD group (G1, G2 and G7) than in C & U group (Table I) indicates that the PLD suppresses the diuretic response. Vagotomy does not abolish this antidiuretic effect of PLD (G7, Table II) indicating that this effect is not mediated through vagi. Cause of this antidiuretic effect of PLD is not clear from the available literature. Stimulation of left atrial receptors by distension causes tachycardia and diuresis during saline infusion and this is abolished by vagotomy (8-13). PLD depresses the cholinergic transmission (17) leading to tachycardia (31). Suresh (31) has failed to observe post vagotomy tachycardia in PLD anaesthetised dogs. These evidences indicate that the depression of diuresis in PLD group is not due to vagolytic activity of PLD occurring at the peripheral nerve ending, but probably due to a central effect. This conclusion requires further validation.

The diuresis during TCW infusion was high in both PLD and C & U groups (G3 and G6). In PLD group, TCW caused significantly more diuresis than NS or RL. Excretion of only 198 and 244 *ml* urine by PLD and C & U groups during 100 *ml/kg* body weight

TCW infusion dose indicates that TCW is well retained in the body as suggested by Suresh and Hegde (30). Comparison of diuresis during NS, RL and TCW infusion in C & U groups (Table I) suggest that the retainability of TCW in these dogs was higher than RL but less than NS.

In PLD group, the TCW caused significantly more diuresis than NS and RL indicating that the TCW causes diuresis by a mechanism other than mere volume expansion. From the literature available (3,29,31), it would be seen that much of the osmolarity of the TCW is contributed by a high concentration of reducing sugars resembling hydration fluids used in clinical practice. Hence, after TCW infusion the reducing sugars get metabolised fast, decreasing the TCW osmolarity which may in turn reduce plasma osmolarity causing increased urine flow. This suggestion may need further validation for confirmation. Surprisingly this increased diuretic effect of TCW in PLD group is not seen in C & U group where RL produced maximal diuresis. Thus, the explanation put forward for PLD group will not fit. Selkurt (21) reported that acidosis increased renal vascular resistance. Suresh (31) has reported increased peripheral blood flow in G3 and a fall in G6 showing the evidence of vasoconstriction in G6. These facts suggest that the osmodilutary effect of TCW infusion was probably counteracted by the vasoconstriction resulting from acidosis caused by TCW infusion (pH 5.1 ± 0.2, Suresh, 31). Chloralose increases the chemoreceptor sensitivity and depresses the baroreceptor sensitivity (15,16) which may further increase the acidosis induced vasoconstriction in G6.

Experimental surgery groups :

Vagotomy in PLD group (G7) had no effect on RL induced diuresis but largely increased the diuresis in C & U group (G8, Table II). This failure of vagotomy to increase the diuresis in PLD group indicates that the antidiuretic effect of PLD is not mediated by vagal pathways.

Vagotomy (G8) or CS denervation (G9) in C & U group greatly increased diuresis of RL infusion (Table III). This effect is difficult to explain because it is generally believed that atrial stretch receptors having their afferents in vagi are essential for the inhibition of antidiuretic hormone (ADH) production and to cause diuresis, which disappears after vagotomy (5,8-11,23,26). Vagotomy, occlusion of common carotids and occlusion of CS in the presence of vagotomy increases ADH secretion (22-25). Kappagoda *et al.* (11) has reported that diuresis resulting from atrial stretch is not due to fall in ADH concentration. Others have also shown that saline induced diuresis is not affected by ADH and aldosterone concentration or their administration (1,33). Thus, it would appear from the available literature that the exact mechanism of saline induced diuresis is far from clear.

Combined vagotomy and CS denervation (G10) decreased diuretic response to infusion even below the intact (G5), vagotomised (G8) or CS denervated (G9) groups.

This fall in diuretic response might be due to an increased ADH production in the absence of both vagal and CS afferent inhibition (22-25). Hence, it appears that even if one of these two pathways are intact as in G8 or G9, the ADH secretion in response to infusion of RL is less. But when both types of influences are absent its secretion is more. Second possibility of the decreased diuresis in G10 is perhaps due to extreme vasoconstriction (31) resulting after removal of vagal and CS inhibitory impulse to sympathetic vasomotor tone which is high in renal vessels.

The urine flow rate of spinal dogs (G11 and G12) was almost equal to intact PLD groups (G1 and G2, Table III). Vagotomised spinal dogs (G12) had significantly more diuresis than spinal dogs (G11). In both groups, appreciable urine flow rate was seen after 30 to 35 *ml* infusion stages when their reported blood pressure (BP) was 50 to 60 mm Hg (31) and cont nued till the end of infusion schedule indicating that the renal functions in spinal dogs was re-established at 50 to 60 mm Hg. In spinal dogs (G11), there was anuria at 30 min after the completion of the infusion schedule when their reported BP was 35 mm Hg (31) indicating the renal failure. Renal blood flow and glomerular filtration falls during haemorrhagic hypotension (2,4) and extreme oliguria and anuria occur at 40 to 60 mm Hg BP (20) and urine flow begin at about 60 mm Hg of renal perfusion pressure (27). The present observation supports these data but the regression pattern of diuresis could not be compared as there are no published reports on this.

ACKNOWLEDGEMENTS

The authors gratefully acknowledge the physical facilities provided by the authorities of the St. John's Medical College, Bangalore and the Veterinary College, Bangalore to carry out this work, and that of Dr. N. Sunderraj and Mr. E. Vasantha Kumar of the University of Agricultural Sciences, Bangalore, for their kind help in the statistical analysis.

REFERENCES

1. Cortney, M.A., M. Mylle, W.E. Lassiter and W.W. Gottschalk. Renal tubular transport of water, solute and PAH in rats loaded with isotonic saline. *Am. J. Physiol.*, **209** : 1199-1205, 1965.
2. Goodyer, A.V.N. and C.A. Jaeger. Renal response to non-shocking haemorrhage: Role of autonomic nervous system and of the renal circulation. *Am. J. Physiol.*, **180** : 69-74, 1955.
3. Grove, D., Uber die Eigenschaften des Kokosrubwassers. *Tropenlandwrit*, **68** : 37-57, 1967.
4. Heinemann, H.O., C.M. Smythe and P.A. Marks. Effect of haemorrhage on estimated hepatic blood flow and renal blood flow in dogs. *Am. J. Physiol.*, **174** : 352-356, 1953.
5. Henry, J.P. and J.W. Pearce. The possible role of cardiac atrial stretch receptors in the induction of change in urine flow. *J. Physiol.*, **131** : 572-585, 1956.
6. Jagger, P.I., D.P. Lauler and G.W. Thorn. Saline-induced natriuresis. *Fed. Proc.*, **24** : 404, 1965.
7. Jones, L.M. *Veterinary Pharmacology and Therapeutics.* 2nd Edn. Ames, Iowa, The Iowa St. Univ. Press. pp. 127-130. 1957.
8. Kappagoda, C.T., R.J. Linden and H.M. Snow. A reflex increase in heart rate from distension of the junction between the superior venacava and the right atrium. *J. Physiol.*, **220** : 177-197, 1972.
9. Kappagoda, C.T., R.J. Linden and H.M. Snow. The effect of distending the atrial appendages on urine flow in the dog. *J. Physiol.*, **227** : 233-242, 1972.

10. Kappagoda, C.T., R.J. Linden and H.M. Snow. The effect of stretching the superior venacaval - right atrial junction on right atrial receptor in the dog. *J. Physiol.*, **227** : 875-887, 1972.

11. Kappagoda, C.T., R.J. Linden, E.M. Scott and H.M. Snow. The efferent pathway of the reflex increase in heart rate produced by stimulation of left atrial receptors. *J. Physiol.*, **242** : 79, 1974.

12. Kidd, C., J.R. Ledsome and R.J. Linden. Atrial receptors and the heart rate. *J. Physiol.*, **185** : 78-79P, 1966.

13. Ledsome, J.R., R.J. Linden and W.J. O'Conner. The mechanism by which distension of left atrium produces diuresis in anaesthetised dogs. *J. Physiol.*, **159** : 87-100, 1961.

14. Levinsky, N.G. and R.C. Lalone. The mechanism of sodium diuresis after saline infusion in the dog. *J. Clin. Invest.*, **42** : 1261-1276, 1963.

15. Neil, E., C.R.M. Redwood and A. Schweitzer. Pressor responses to electrical stimulation of the carotid sinus nerve in cats. *J. Physiol.*, **109** : 259-271, 1949.

16. Neil, E., C.R.M. Redwood and A. Schweitzer. Effect of Electrical stimulation of aortic nerve on blood pressure and respiration in cats and rabbits under chloralose and nembutal anaesthesia. *J. Physiol.*, **109** : 392, 1949.

17. Nicholls, J.G. and J.P. Quilliam. The mechanism of action of paraldehyde and methylpentynol on neuromuscular transmission. *Brit. J. Pharmacol. Chemotherap.*, **11** : 151-155, 1956.

18. Papper, S., L. Saxon, J.P. Rosenbaum and H.W. Cohen. Effects of isotonic and hypertonic salt solutions on the renal excretion of sodium. *J. Lab. Clin. Med.*, **47** : 776-782, 1956.

19. Raisz, L.G. Dextran and oxypolygelatin as plasma volume expanders: renal excretion and effects on renal function. *J. Lab. Clin. Med.*, **40** : 880-890, 1952.

20. Selkurt, E.E. Renal blood flow and renal clearance during haemorrhagic shock. *Am. J. Physiol.*, **145** : 699-709, 1946.

21. Selkurt, E.E. The renal circulation. In: *Handbook of Physiology.* Section II. Circulation, Vol. II. *Am. Physiol. Soc., Washington, D.C.*, pp. 1457-1516, 1963.

22. Share, L. and M.N. Levy. Cardiovascular receptors and blood titer of the antidiuretic hormone. *Am. J. Physiol.*, **203** : 425-428, 1962.

23. Share, L. Effect of carotid sinus occlusion on left atrial distension and plasma vasopressin. *Am. J. Physiol.*, **208** : 219-223, 1965.

24. Share, L. and M.N. Levy. Effect of carotid chemoreceptor stimulation on plasma antidiuretic hormone titer. *Am. J. Physiol.*, **210** : 157-161, 1966.

25. Share, L. and M.N. Levy. Carotid sinus pulse pressure, a determinant of plasma antiduretic hormone concentration. *Am. J. Physiol.*, **211** : 721-724, 1966.

26. Share, L. Role of peripheral receptors in the increased release of vasopressin on response to haemorrhage. *Endocrinology*, **81** : 1140-1146, 1967.

27. Shipley, R.E. and R.S. Study. Changes in renal blood flow, extraction of inulin, glomerular filtration rate, tissue pressure and urine flow with acute alterations of renal artery blood pressure. *Am. J. Physiol.*, **167** : 676-688, 1951.

28. Snedecor, G.W. and W.G. Cochran. *Statistical Methods.* Edn. 6. Calcutta, Oxford and IBH Publ. Co., pp 94, 100, 115, 271, 296-298 and 460-464, 1968.

29. Suresh, T.P., V. Ravivarma Hegde, S.V.S. Shetty and T.R.S. Rangachar. Fluid therapy by tender coconut water in veterinary practice. *Indian Vet. J.*, **48** : 829-837, 1968.

30. Suresh T.P. and V. Ravivarma Hedge. Tender coconut water as a plasma volume expander: II. Studies on physiological responses. *Mysore J. Agric. Sci.*, **5** : 410-422, 1971.

31. Suresh, T.P. Neural control of cardiovascular functions: Effect of volume change. Ph.D. Thesis, Bangalore University, Bangalore, 1975.

32. Suresh, T.P. Regression pattern of amplitude of electrocardiographic waves during intravenous infusion in intact, buffer nerve denervated and spinal dogs. *Ind. J. Physiol. Pharmac.*, **22** : 249-262, 1978.

33. Watson, J.F. Effect of saline loading on sodium reabsorption in the dog proximal tubule. *Am. J. Physiol.*, **210** : 781-785, 1966.