results (Table II) indicate that asphyxia or hypoxia did not contribtue to the overall effects of epinephrine secretion due to cigarette smoke in this present study. Only moderate increases in peripheral epinephrine levels were observed and these were seen only after severe asphyxia and hypoxia. In all smoking experiments there was a free exchange of respiratory gases; smoking always stimulated respiration and there were no indications of either asphyxia or hypoxia in the smoking experiments.

Summary. Inhalation of cigarette smoke causes a significant increase in epinephrine levels of the adrenal vein, vena cava, peripheral arterial and peripheral venous blood in the dog, the degree of increase being proportionally less in each area respectively.

1. Mansfield, G., *Orv. Hetil.*, 1958, v52, 241, cited in Larson, P. S., Haag, H. B., Silvette, H., *Tobacco Experimental and Clinical Studies*: A Comprehensive Account of the World Literature, Williams and Wilkins Co., Baltimore, 1961.

2. Cannon, W. B., Hoskins, R. G., *Am. J. Physiol.*, 1911-12, v29, 274.

3. Stewart, G. N., Rogoff, J. M., *J. Pharmacol. Exp. Ther.*, 1919, v13, 183.

4. Segawa, T., *Fol. Pharm., Japan*, 1954, v50, 42.

5. Silvette, H., Larson, P. S., Haag, H. B., *Rev. intern. Med.*, 1961, v107, 915.

6. Westfall, T. C., Watts, D. T., *Fed. Proc.*, 1962, v21, 193.

7. Satake, Y., *Tohoku J. Exp. Med.*, 1954, v60, suppl. 11.

8. Gaddum, J. H., Lembeck, F., *Brit. J. Pharmacol.*, 1949, v4, 401.

9. Franko, B. V., Bragg, A. D., Watts, D. T., *Arch. int. Pharmacodyn.*, 1957, v111, 123.

10. Hazard, R., Beavallet, M., Larno, S., *J. Physiol.* (Paris), 1957, v49, 196.

11. De Schaepdryver, A. F., *Arch. int. Pharmacodyn.*, 1958, v117, 475.

12. Woods, E. F., Richardson, J. A., *J. Pharmacol. Exp. Ther.*, 1955, v114, 445.

13. Woods, E. F., Richardson, J. A., Richardson, A. K., Bozeman, R. F., Jr., *ibid.*, 1956, v116, 64.

14. De Schaepdryver, A. F., Preziosi, P., Van Der Stricht, J., *Arch. int. Pharmacodyn.*, 1959, v121, 468.

15. Euler, U. S. von, Lishajko, F., *Acta physiol. scand.*, 1959, v45, 122.

16. Ruhl, A., Lin, P., *Deut. Med. Wochschr.*, 1936, v62, 493.

17. Hanson, H. B., Hastings, A. B., *J. Am. Med. Assn.*, 1933, v100, 1481.

18. Exley, K. A., *Brit. J. Pharmacol.*, 1954, v9, 170.

19. Armstrong, G. G., Jr., Porter, H., Jr., Langston, J. B., *Am. J. Physiol.*, 1961, v201, 897.

20. Koppermann, E., Brendel, W., Thauer, R., *Pflug. Arch. ges. Physiol.*, 1955, v260, 239.

21. Greever, C. J., Watts, D. T., *Circulation Res.*, 1959, v7, 192.

22. Watts, D. T., Meador, J. C., Arnett, E. L., *Pharmacologist*, 1959, v1, 57.

23. Walker, W. F., Zilele, M. S., Reutter, F. W., Shoemaker, W. G., Friend, D., Moore, F. D., *Am. J. Physiol.*, 1959, v197, 773.

24. Humer, D. M., *Surgical Forum*, 1958, v8, 11.

25. Fowler, N. O., Shabetal, R., Holmes, J. C., *Circulation Res.*, 1961, v9, 427.

26. Toyooka, E. T., Blake, W. D., *Am. J. Physiol.*, 1961, v201, 448.

Received November 16, 1962. P.S.E.B.M., 1963, v112.

Alterations of Plasma Volume, Electrolytes and Volume of Distribution of Sodium Thiocyanate Induced by Electroshock.* (28186)

W. W. SWINGLE AND A. J. SWINGLE

Biological Laboratory, Princeton University, Princeton, N. J.

Electroconvulsive shock induced by passage of an electric current through the brain serves as a powerful stimulus to the central nervous system producing many and varied reactions. Thus the dog is rendered unconscious; violent muscle spasms occur necessitating restraint; respiration may be held in abeyance; profuse salivation is present; involuntary urination and defecation occur. Aside from these gross manifestations, numer-

* Acknowledgement is made for financial and other valuable aid: Ciba Pharmaceutical Products Co., Upjohn Co., National Science Foundation, Nat. Inst. Arthritis and Metabolic Diseases, USPHS.

TABLE I. Alterations of Plasma Volume, Plasma Electrolytes and Hemoconcentration Induced By Electroshock in Unanesthetized Dogs.

Time after shock		Body wt, kg	Hb, g %	Hmct, %	RBC 10^6 mm^3	Blood sugar, mg %	Plasma volume cc/kg	Plasma volume % change	Plasma electrolytes, meq/l Na	Cl	K
Dog 1											
Control	N	14.74	14.38	38.8	5.69	71	48.7		144.5	114.2	3.16
3 min	S	14.97	15.78	40.3	5.89	141	34.4	−29.3	154.5	118.9	3.51
1 hr	R		14.20	38.0	5.70	126			145.5	116.9	2.91
24 hr	N_1	14.42	14.03	37.3	5.69	81	47.8	+38.9	144.2	115.4	3.25
Dog 2											
Control	N	14.85	14.38	38.5	5.69	71	49.0		142.0	114.2	3.06
4 min	S	14.85	15.25	39.5	5.75	145	38.8	−20.8	152.0	116.2	3.65
1 hr	R		12.51	36.8	5.30	120			143.8	114.2	2.98
24 hr	N_1	14.51	12.11	35.0	5.00	96	50.8	+30.9	142.5	112.8	3.73
Dog 3											
Control	N	20.00	15.60	40.8	5.78	80	50.0		146.0	119.0	3.47
3 min	S	19.77	16.90	47.5	6.25	106	31.5	−37.0	158.5	121.0	3.65
1 hr	R		15.70	44.1	5.89	89			143.5	116.2	3.00
14 hr	N_1	19.77	13.72	38.7	5.44	90	48.4	+53.6	147.0	115.4	3.33
Dog 4											
Control	N	15.76	16.01	44.8	5.97	80	52.0		146.5	113.5	3.67
3 min	S	15.65	17.36	49.7	6.59	96	40.0	−23.0	160.0	115.3	3.49
1 hr	R	15.65	11.71	34.9	5.34	90	53.3	+33.2	149.5	114.2	3.16

N = Normal—24 hr before shock; S = Shock; R = Recovery; N_1 = Normal at various times after application of current.

ous changes appear in the body fluids and their constituents. The blood pH and bicarbonate fall indicating acidosis; plasma lactate and Na rise to high levels; plasma volume falls and a moderate degree of hemoconcentration is present. These responses to brain stimulation by the electric current are evanescent and quickly disappear in rats(1); psychotic patients(2,3) and normal dogs with intact adrenal cortices(4). The severe decline in plasma volume and rise in Na pose the twin problems of the whereabouts of the blood fluid which so rapidly passes out of active circulation and the source of the increased plasma Na which in some dogs may rise 10-14 meq/l even before the convulsions have subsided. The following experiments were designed to study this problem in more detail.

Methods. Unanesthetized dogs were trained to remain quiet and relaxed during i.v. injections and removal of blood samples over periods of 40 minutes. Several of the methods employed have been referred to in earlier publications from this laboratory *e.g.*, electroconvulsions(4); plasma volume, Na, Cl, K and hematocrit(5). Muscle and plasma lactate were determined according to Barker and Summerson(6). Plasma volume and volume of distribution of sodium thiocyanate were measured simultaneously using the method of Gregersen and Stewart(7) and Chien and Gregersen(8) with minor modifications. A mixture of 5% NaSCN and 0.4% T-1824 dye was measured by the gravimetric technique, each dog receiving approximately 15 mg/kg of thiocyanate in 5 cc of water. The 5 cc blood samples were withdrawn through an indwelling needle in the jugular vein at 15, 20, 25, and 30 minute intervals and the syringe thoroughly rinsed at each bleeding by drawing blood in and out of the barrel. The analyses were made using 1 cc of plasma, a Beckman B spectrophotometer with 10 mm cuvettes and read at 480 mμ. No food was given for 24 hours preceding initiation of the experiments and both food and water were withheld thereafter. The dogs used for muscle lactic acid determinations were anesthetized by i.v. injections of pentobarbital immediately convulsions ceased; a portion of the gastrocnemius muscle was frozen *in situ* with a mixture of ether and pulverized CO_2, removed and weighed. An average of 4 minutes elapsed between cessation of convulsions and removal of the muscle sample.

TABLE II. Changes in Volume of Distribution of Thiocyanate Induced by Electroshock in the Normal Dog.

Time after shock	Body wt, kg	Hb, g %	Hmct, %	RBC 10^6 mm^3	Blood sugar, mg %	Plasma volume cc/kg	Plasma volume % change	Plasma electrolytes, meq/l Na	Cl	K
Dog 5										
Control N	19.88	14.90	40.8		81	65.0		142.5	110.6	3.33
3 min S	19.88	18.04	45.9		94	48.8	−26.4	153.6	117.9	3.74
24 hr N_1	19.80	15.50	38.5		85	62.5	+28.0	141.0	111.5	3.92
Volume of distribution of thiocyanate in Dog 5										
Control N		305 cc/kg								
3 min S		214 cc/kg		Decrease of 1809 cc						
24 hr N_1		301 cc/kg		Increase of 1723 cc						

N = Normal—24 hr before shock; S = Shock; N_1 = Normal at various times after application of current.

Results. I. *Plasma volume, plasma electrolytes and hemoconcentration changes induced by electroshock.* Data obtained from 5 of the 6 dogs used are shown in Tables I and II. Control levels of the plasma volume and various blood constituents were established 24 hr before shocking the animals; the following day sampling was repeated at intervals thereafter. Plasma volume declined extremely rapidly accompanied by a rise in plasma Na (Table I, Dogs 1-4). Na increase averaged 11.5 meq/l whereas plasma volume fall averaged 14.2 cc/kg. Plasma Cl changed much less, averaging 3.5 meq/l. Plasma K rose 0.35 meq/l but decreased below preshock levels within an hour. Hemoconcentration was present 3-4 minutes after applying the current but was soon followed by blood dilution as plasma volume increased. The blood was usually more dilute 24 hours after electroconvulsions than it was before. Blood urea nitrogen exhibited no significant change hence is not tabulated. Blood sugar attained high levels and remained above control readings when the experiment ended. The mean blood pressure usually dropped sharply during the first few minutes after the convulsion but soon regained its original height.

II. *Volume of distribution of sodium thiocyanate.* The sudden, marked fall in plasma volume which apparently occurs almost instantaneously with application of the current, led to detailed study of the fluid changes in the extracellular compartment. The fluid available for dilution of thiocyanate was utilized as a measure for gauging increases or decreases of extracellular fluid volume. In the representative example shown in Table II (Dog 5), the control volume of fluid available for dilution of thiocyanate was 305 cc/kg whereas 3 minutes after cessation of convulsions it had fallen to 214 cc/kg, a decrease of 1809 cc. Twenty-four hours later the volume of distribution of SCN had increased to 301 cc/kg or a total of 1723 cc despite withholding food and water. In numerous experiments, including those in which pentylenetetrazol (Metrazol, Knoll Co.), shock was employed as a stressor, the contraction and expansion of the thiocyanate space invariably paralleled the fall and rise of the plasma volume.

III. *Muscle and plasma lactate following electroconvulsions and during recovery.* The severe reactions of the unanesthetized dog subjected to electroconvulsions led to test of the possibility that perhaps increase in muscle cell lactate might be of sufficient magnitude to raise the cellular osmolar concentration of osmotically active substances to the point where shift of fluid from the extracellular compartment into cells would occur. Pertinent data are shown in Fig. 1. Both muscle and plasma lactate increased approximately 7 times their control values within a few minutes after convulsions, then slowly fell over the ensuing 4 hours. However, Fig. 1 shows that owing to rapid diffusion of lactate from cells to plasma no osmotic gradient was present at the time samples were taken and it is doubtful if any substantial amount of fluid was shifted out of the extracellular space.

TABLE III. Prevention of Plasma Volume and Electrolyte Changes by Sodium Pentobarbital Anesthesia in Dogs Subjected to Electroshock.

Time after shock	Body wt, kg	Hb, g %	Hmct, %	RBC 10^6 mm^3	Blood sugar, mg %	Plasma volume cc/kg	Plasma volume % change	Plasma electrolytes, meq/l Na	Cl	K
Dog 6										
Control N	14.97	14.38	41.2		85	54.0		151.5	114.0	3.50
3 min N*	14.97	13.96	39.5		118	53.2	1.4	151.5	118.3	2.86
1 hr N_1	14.97	13.05	35.4		99	50.3	5.4	152.5	113.5	3.40
Dog 7										
Control N	15.85	14.90	43.0		81	54.9		152.0	116.2	3.51
4 min N*	15.76	14.20	37.0		97	56.0	+2.0	146.5	111.5	2.51
1 hr N_1	15.74	12.77	36.3		95	54.4	2.8	148.5	110.1	2.91
Dog 8										
Control N	22.50	14.03	39.8		86	46.2		152.5	113.5	3.51
3 min N*	22.50	14.38	40.0		86	46.5	+0.6	152.0	114.9	3.16
1 hr N_1	22.50	13.05	38.0		94	45.8	−1.5	152.0	114.0	3.81
Dog 9										
Control N	18.63	12.40	36.8		76	50.8		151.5	113.5	3.75
3 min N_1*	18.63	13.19	37.4		70	52.7	+3.7	151.0	112.5	3.65

* 180 milliamperes delivered but reactions were slight.

N = Normal—24 hr before shock; N_1 = Normal at various times after application of current.

IV. *Prevention of plasma volume and electrolyte changes by pentobarbital anesthesia in dogs subjected to electroshock.* Attention has been called to the tranquilizing effect of certain barbiturates on the reactions produced by electroshock in rats(9,10). Because of these and other reports, 6 dogs were deeply anesthetized by i.v. injections of 30 mg/kg of sodium pentobarbital (Nembutal, Abbott) and 30-60 minutes later an electric current of the same intensity as used in the other experiments was applied. The usual strong reaction did not materialize, the animals remained relaxed and flaccid, responding to th stimulus merely by a single body jerk. Sig nificant changes in plasma volume and elec trolytes failed to appear although decrease in hemoglobin and hematocrit one hour late indicated that some sequestration of erythro cytes had taken place (Table III). Severa authors have reported that pentobarbita amytal, pentothal, etc., may increase th plasma volume 8-10% (11-13). These e periments were repeated using 4 dogs ane thetized with pentobarbital and sampled 3(60 minutes later. The results are shown i

TABLE IV. Effect of Sodium Pentobarbital Anesthesia on Non-Shocked Dogs.

Time after shock	Body wt, kg	Hb, g %	Hmct, %	RBC 10^6 mm^3	Blood sugar, mg %	Plasma volume cc/kg	Plasma volume % change	Plasma electrolytes, meq/l Na	Cl	K
Dog 10										
Control	15.31	15.43	41.0		81	48.0		151.5	116.2	3.18
Anesthesia	15.68	12.41	34.9		85	52.6	+9.5	151.5	116.9	3.35
Dog 11										
Control	22.72	18.04	49.1		78	50.0		150.0	112.1	3.21
Anesthesia	22.61	15.60	37.9		89	54.0	+8.0	148.5	111.4	3.21
Dog 12										
Control	18.72	15.45	45.0		80	41.0		152.5	117.6	3.81
Anesthesia	18.72	12.77	36.0		85	44.0	+7.3	153.0	118.3	3.41
Dog 13										
Control	25.80	16.20	43.6		78	50.0		148.0	114.8	3.67
Anesthesia	25.80	12.77	33.1		90	54.0	+8.0	148.0	113.5	3.58

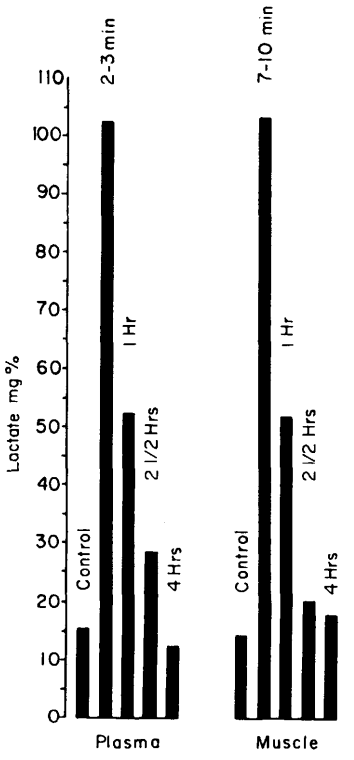

FIG. 1. Plasma and muscle lactate of electroshocked dogs. Each bar represents an avg of 8 determinations. Plasma was obtained 2-3 min and muscle samples 4 min after shock; dogs were anesthetized with pentobarbital before removing muscle.

Table IV. The plasma volume increased an average of 8.2% and was associated with decreased hemoglobin and hematocrit but the plasma Na did not change appreciably. The hematocrit-poor blood is probably not entirely accounted for by change in plasma volume since barbiturates are known to induce dilation of the spleen associated with sequestration of red cells in this organ(14). Pentobarbital apparently also has a potent effect on vasomotor tone and is reported to greatly increase blood flow under certain experimental conditions(15).

Discussion. Hematocrit and hemoglobin values of the post-shocked dogs indicate the fall in plasma volume is probably not due to shift of water from the extra to the intracellular fluid compartment. Decline in plasma volume of the degree shown to occur in Dogs 1-5, Tables I and II (average of −27.3%) should result in much higher values for hematocrit and hemoglobin than the mild increases observed. Since the volume of distribution of thiocyanate also fell a comparable percentage, the 'lost' fluid evidently was not in the extravascular-extracellular space and therefore must be held immobilized and out of circulation somewhere within the vascular tree—presumably at the periphery. It seems reasonable to assume that application of a strong electric current to the brain would induce other reactions besides those enumerated earlier. Among these probably would be a severely aggravated hyperexcitability of the vasomotor system giving rise to an intense vaso constriction and thereby closing off large areas of the peripheral vasculature resulting in sequestration and trapping of blood in the small vessels of the circulatory tree. This pooling of blood should greatly reduce the volume of fluid in actual circulation since substantial segments of the vascular tree would be denied free access to the dye T-1824 and SCN, resulting in the low post shock values recorded in Table I. The severe vasoconstriction presumably would be relieved after withdrawal of the brain stimulation; the closed vascular channels would reopen and free blood flow at the periphery be reestablished with disappearance of shock and return to normal values for plasma volume and thiocyanate space.

Low plasma volumes associated with little or no change in hematocrit are not peculiar to electroshocked dogs for several investigators report similar findings in hypothermic shock, a condition in which there is sequestration of large volumes of blood in the peripheral circulation. Thus hypothermic chicks and rabbits exhibit a 30% decrease in plasma volume without important changes in the hematocrit(16). Dogs subjected to hypothermia show markedly reduced plasma volumes with unchanged hematocrits(17,18). There was no evidence for a shift of water into cells in these dogs(17). Phenoxybenzamine (Dibenzyline) administered to normothermic chicks produces a sharp increase in the plasma volume and prevents most of the reduction in plasma volume which occurs during induced hypothermia. Calculated red cell mass

remains unaffected(19). The authors suggest the results indicate sequestration of plasma and blood cells in different vascular beds perhaps *via* neurogenic mechanisms. These results together with those recorded in Tables I and II, apparently are due to closing and opening of certain peripheral circulatory beds under the influence of central nervous system stimulation.

The striking elevation of plasma Na which accompanies the decline in plasma volume of the shocked dog does not represent increased concentration of the cation due to sudden shift of fluid into cells, for it is improbable that such a shift occurs. The rise in Na in these and similar cases reported by clinical investigators following brain injuries have no known causal relationship to plasma volume changes. Thus extreme hypernatremia and hyperchloremia not associated with dehydration or renal disease are known to occur in man and are associated with certain types of cerebral lesions. Cooper and Crevier(20) recently reviewed 14 cases plus 4 of their own patients. Three of the 4 exhibiting high serum Na and Cl had hematocrits ranging from 41-46%. In none was there evidence of insufficient fluid intake or excess water loss. The hypernatremia and hyperchloremia were considered due to neurogenic causes. The elevation of plasma Na of the dogs occurred practically instantaneously with application of the electric current and was fleeting in character. Welt, Orloff *et al.*(3) state that the high serum Na of their electroshocked patients fell to control levels within 3-4 minutes. It is unlikely that intake or output of fluid plays any role in the hypernatremia of electroshock in dogs. The data in Table I do reveal a mild hemoconcentration possibly indicating strong splenic contraction due to the sudden brain stimulus and release of red cells into the circulation. Electroshock is a violent central nervous system stressor hence it is perhaps not surprising, in view of the reports on brain injured patients, that such a stimulus should be associated with a hypernatremia of brief duration in dogs. The present experiments do not solve the problem of the source of the high plasma Na and the neurogenic mechanisms inducing its elevation.

Summary. Severely electroshocked dogs exhibit a striking fall in plasma volume, a sharp rise in plasma Na, blood sugar, muscle and plasma lactate but the hematocrit and hemoglobin show only moderate increases. The hypernatremia is fleeting in character and is largely, if not completely, independent of the decline in plasma volume; there is no evidence that water is shifted into cells causing shrinkage of the extracellular compartment. The increased concentration of the cation apparently results directly from electrostimulation of unknown neurogenic, Na regulating mechanisms in the central nervous system. The evanescent fall in plasma volume and simultaneous contraction of thiocyanate space in immediate post shocked dogs is not reflected by comparable changes in hematocrit and hemoglobin and is attributed to intense vasoconstriction due to sudden, violent shock to the brain and sympathetic nervous system with sequestration and trapping of large volumes of blood in the minute vessels at the vascular periphery. Closing off of substantial areas of the circulation by shock prevents free access of the dye T-1824 and SCN to the periphery of the vascular tree and thus accounts for the low post shock values of the plasma volume and SCN space compared to their controls.

1. Ward, J. R., Call, L. S., PROC. SOC. EXP. BIOL. AND MED., 1949, v70, 381.

2. Altschule, M. D., Tillotson, K. J., *Am. J. Psychiat.*, 1949, v105, 829.

3. Welt, L. G., Orloff, J., Kydd, D. M., Oltman, J. E., *J. Clin. Invest.*, 1950, v29, 935.

4. Swingle, W. W., Da Vanzo, J. P., Glenister, D., Wagle, G., Osborn, M., Rowen, R., PROC. SOC. EXP. BIOL. AND MED., 1960, v104, 184.

5. Swingle, W. W., Da Vanzo, J., Fedor, E., Crossfield, H. C., Glenister, D., Osborn, M., Wagle, G., *ibid.*, 1958, v97, 416.

6. Barker, S. B., Summerson, W. H., *J. Biol. Chem.*, 1941, v138, 535.

7. Stewart, J., Gregersen, M., *Am. J. Physiol.*, 1939, v125, 142.

8. Chien, Shu, Gregersen, M., *Physical Techniques in Biological Research* Vol. IV, Academic Press, New York, 1962.

9. Goodman, L., Toman, J., *Fed. Proc.*, 1945, v4, 120.

10. Toman, J., Swinyard, E., Goodman, L., *J.*

Neurophysiol., 1946, v9, 231.

11. Bollman, J. L., Svirbelly, J., Mann, F. C., *Surgery*, 1938, v4, 881.

12. Hamlin, E., Gregersen, M. I., *Am. J. Physiol.*, 1939, v125, 713.

13. Bonnycastle, D. D., *J. Pharmacol. and Exp. Therap.*, 1942, v75, 18.

14. Hausner, E., Essex, H. C., Mann, F. C., *Am. J. Physiol.*, 1938, v121, 387.

15. Imig, C. J., Roberson, M. J., Hines, H. M., *ibid.*, 1956, v186, 35.

16. Rodbard, S., Saiki, H., Malin, A., Young, C., *ibid.*, 1951, v167, 485.

17. D'Amato, H. E., Hegnauer, A. H., *ibid.*, 1953, v173, 100.

18. Sircar, P., PROC. SOC. EXP. BIOL. AND MED., 1954, v87, 194.

19. Williams, F. L., Rodbard, S., *Am. J. Physiol.*, 1960, v198, 169.

20. Cooper, I. S., Crevier, P. H., *J. Clin. Endocrinol. and Metab.*, 1952, v12, pt. 2, 821. Extensive bibliography listed.

Received November 30, 1962. P.S.E.B.M., 1963, v112.

Determination of Minimal Maintenance Levels of Adrenal Steroids in the Bilaterally Adrenalectomized Goat.* (28187)

R. M. JOHNSON AND E. E. FLAMBOE (Introduced by F. X. Gassner)
Department of Physiology, Colorado State University, Fort Collins

Few reports appear in the literature on maintenance therapy in ruminants following bilateral adrenalectomy. Differences in hormone administration routes, criteria, species and environmental conditions probably account for the different dose levels that are reported. Cowie and Stewart(1) and Cowie and Tindal(2) reported that daily absorption from implanted tablets of 6-12 mg cortisone and 2.5 mg desoxycorticosterone acetate (DCA) is sufficient for maintenance of lactation in the adrenalectomized goat. Goding and Denton(3) were able to maintain adrenalectomized sheep on daily intramuscular injections of 25 mg cortisone and 5-10 mg DCA. Estergreen and Van Demark(4) reported that 25 mg of cortisone and 5 mg DCA per 100 lb body weight per day would successfully maintain adrenalectomized calves.

Materials and methods. To evaluate properly the minimal maintenance levels of adrenal steroids required in the bilaterally adrenalectomized animal, environmental and physiological stress should be reduced to a minimum.

The animals used in this study were 3 year old female goats of a mixed dairy type breed. Adrenalectomized and intact control animals were housed in separate pens in a controlled environment chamber. A temperature of 25°C ± 0.5°C, relative humidity of 50 ± 2% and alternating 12 hours of light and darkness were maintained throughout the study, thus minimizing environmental stress.

Bilateral adrenalectomy was performed in two stages. The first stage was removal of the right adrenal gland. After a 3 week interval the second stage, removal of the left adrenal, was performed.

Adrenalectomy in the goat is complicated by the absence of adrenal veins. The right adrenal is securely attached to the vena cava and the left adrenal gland is firmly attached to the renal vein near its junction with the vena cava. Previous investigators(1,2) reported the use of special clamps which were applied between the gland and the vessel wall. This procedure involved removal of a portion of the vena caval wall and suturing the vessel. Estergreen and Van Demark(4) performed successful adrenalectomies in calves using a Merrick kidney forceps applied between the gland and the vena caval wall. Following removal of the adrenal gland the forceps were left in place for approximately 5 minutes. This eliminated the need for suturing the caval wall. The technique developed for the present study consisted of freeing the gland from surrounding adipose tissue by blunt dissection. No ligation was needed to control

* This work was sponsored by Aerospace Medical Research Laboratories, Aerospace Medical Division, Air Force Systems Command, U. S. Air Force.