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The pH of Systemic Blood in Normal and Hypertensive Dogs, Determined by Means of a Syringe Type Glass Electrode.*

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This experiment was performed in order to determine whether any pH changes of the systemic blood of dogs accompany the development of hypertension produced by partial constriction of the renal artery.[1, 2] Thirteen dogs were used; 4 were hypertensive at the beginning of the experiment; in 4 others hypertension was produced during the period of observation; the remainder served as controls. Over a period of 90 days blood was taken frequently from the saphenous vein directly into a special syringe-type glass electrode. The pH was then measured with a Beckman pH meter.

This glass electrode was a modification of the type developed by MacInnes and Belcher[3] with a capacity of 0.5 cc; it was so constructed that a sterile hypodermic needle could be attached to its tip.† The venous pressure was usually sufficient to force the blood through the straight inner tube of the electrode; occasionally slight stasis was necessary. Constant temperature of the electrode was insured by hanging it in a saturated potassium chloride solution which was kept at 37.5°C by circulating water. This potassium chloride solution served further as a liquid junction between the glass electrode and the reference half-cell. The electrode was calibrated with buffer solutions of known pH and was checked between each sample of blood. The accuracy of the measurements was 0.03 pH units.

Although only 0.5 cc of blood was necessary to fill the glass electrode completely, usually another 0.5 cc of blood was passed through it to make sure that the blood was of the same composition as that in the vein. Before taking a sample, the electrode was rinsed with a solution of heparin (obtained from Connaught Laboratories, University of Toronto, Canada, or Hynson, Westcott and Dunning, Inc.,

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1 Goldblatt, H., Lynch, J., Hanzal, R. F., and Summerville, W. W., *J. Exp. Med.*, 1934, **59**, 347.

2 Glenn, F., and Child, C. G., *Arch. Surg.*, 1938, **36**, 373.

3 MacInnes, D. A., and Belcher, D., *Ind. Eng. Chem. Anal. Ed.*, 1933, **5**, 199.

† Details of the glass electrode will be described elsewhere.

Baltimore, Md.) in 0.9% sodium chloride. Most of this was washed out by the stream of blood, but enough remained to prevent clotting. The blood pH in the presence of heparin did not differ from that in its absence in agreement with the observations of others.[4, 5] The measurements were completed in less than one minute from the time the blood was drawn so that changes in the blood pH due to glycolysis[5] could not have occurred. Blood samples taken repeatedly from the same dog during an hour had identical pH values, demonstrating the reproducibility of the method. From day to day, however, the blood pH of individual dogs varied considerably, which may have been due largely to different degrees of excitement and activity, or to variations in the diet. In Fig. 1 the arterial blood pressures and the pH values of the systemic blood are compared for one dog in each of the 3 groups. The blood pressures were measured by means of Van Leersum carotid loops.[6, 7] It may be seen from Fig. 1 that the variations in pH and arterial pressure do not go in parallel.

The results obtained are summarized in Tables I and II. If the means are calculated for Table I we find pH 7.32 corresponding to an

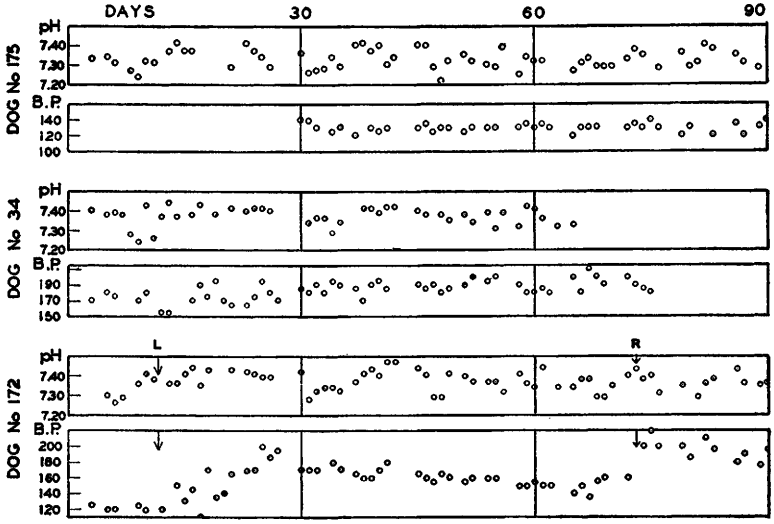

FIG. 1.

B.P.—Arterial pressure in mm mercury.
L—Application of clamp to left renal artery.
R—Application of clamp to right renal artery.

4 Yoshimura, H., *J. Biochem. Tokyo*, 1936, **23**, 335.

5 Haugaard, G., and Lundsteen, E., *Biochem. Z.*, 1936, **285**, 270.

6 Van Leersum, E. C., *Arch. f. d. ges. Physiol.*, 1911, **142**, 377.

7 Child, C. G., and Glenn, F., *Arch. Surg.*, 1938, **36**, 381.

TABLE I.

	Normal					Hypertensive			
Dog No.	3ay	4ay	175	176	402	34	135	161	173
Sex	F	F	F	M	M	M	M	M	M
Previous B.P. mm Hg.						195	170	180	160
Hypertensive for						30 mo.	7 mo.	6 mo.	1 mo.
Avg B.P. mm Hg.									
1st 30 days	160 (18)*	125 (18)				175 (19)	170 (18)	180 (20)	165 (18)
2nd 30 days	145 (20)	140 (20)	130 (20)			190 (22)	180 (21)	170 (22)	160 (19)
3rd 30 days	140 (19)	140 (19)	130 (18)	125 (13)		195 (19)	175 (21)	170 (19)	155 (19)
Avg pH									
1st 30 days	7.37 (15)	7.35 (15)	7.33 (16)	7.35 (15)	7.31 (15)	7.38 (18)	7.35 (16)	7.36 (15)	7.37 (21)
2nd 30 days	7.34 (24)	7.31 (22)	7.33 (25)	7.32 (22)	7.28 (24)	7.37 (21)	7.33 (25)	7.35 (25)	7.34 (21)
3rd 30 days	7.34 (7)	7.31 (5)	7.32 (19)	7.29 (12)	7.27 (22)	7.36 (4)	7.34 (5)	7.33 (7)	7.32 (9)
Total average B.P./pH	150/7.35	135/7.32	130/7.33	125/7.32	7.28	190/7.37	175/7.34	175/7.35	160/7.35

*The figures in parentheses indicate the number of observations.

TABLE II.

Dog No. / Sex		159 F	167 F	172 M	174 F
Avg B.P.	before first clamp	140 (4)*	130 (5)	120 (6)	135 (30)
" "	after " "	200 (4)	170 (39)	160 (43)	160 (20)
" "	" second "	—	205 (7)	195 (11)	—
" pH	before first "	7.30 (6)	7.34 (7)	7.33 (6)	7.38 (30)
" "	after " "	7.26 (3)	7.32 (39)	7.38 (45)	7.37 (22)
" "	" second "	—	7.31 (6)	7.37 (13)	—

*The figures in parentheses indicate the number of observations.

arterial blood pressure of 135 mm of mercury for the normal dogs, and a pH of 7.35 corresponding to an arterial pressure of 175 mm of mercury in the hypertensive dogs. This difference is within the experimental error of our method of measurement; however, since it is based on the average of many values, we feel that it may be significant enough to indicate the direction of pH changes in the blood due to clamping of the renal arteries. Further experiments are in progress to determine the pH changes in each renal vein.

With respect to Table II it may be mentioned that dogs 159 and 167 which had a sharp rise in arterial pressure with a change in blood pH towards more acid values, died before the experiment was finished due to uremia. The other hypertensive dogs with the more alkaline blood have a normal blood urea nitrogen content.

It may be concluded from this experiment that the buffering capacity of the systemic blood is sufficient to offset the introduction of an alkaline or acid substance if such is produced by the kidney as the result of ischemia.

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Bacteriostatic Actions of Three Thiazol Derivatives of Sulfanilamide upon Bacteria in Broth Cultures.

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On the basis of *in vitro* studies 3 members of a new group of compounds, namely 2-sulfanilamidothiazol (sulfathiazol), 2-sulfanilamido-4-methylthiazol (sulfamethylthiazol), both of which were recently described by Fosbinder and Walter,[1] and 2-sulfanilamido-4-

[1] Fosbinder, R. J., and Walter, L. A., *J. Am. Chem. Soc.*, 1939, **61**, 2032.