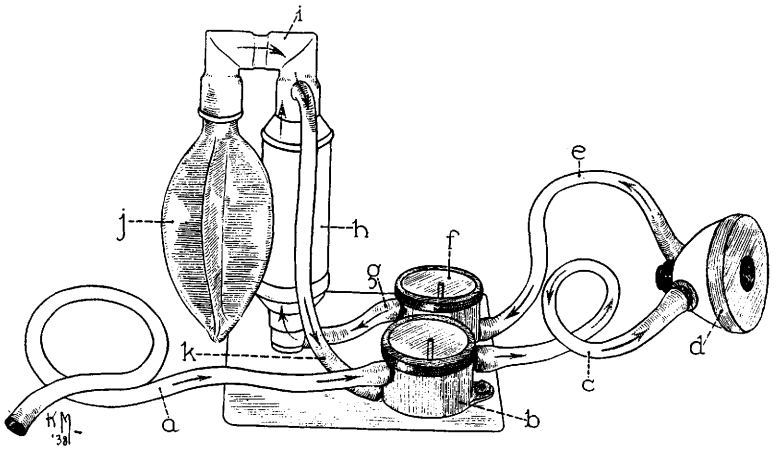

FIG. 1.
Gas machine for anesthetizing small laboratory animals.
(A) Intake tubing
(B) Intake flutter valve.
(D) Face mask.
(F) Exhaling flutter valve.
(H) Soda-lime cannister.
(J) Rebreathing bag.
(K) Inlet for gases from rebreathing bag.
(The arrows mark the course of the gases in the rebreathing circuit.)

water pressure, thus allowing the animal to breathe with a minimum of exertion. The use of the apparatus in experiments on rabbits proved highly satisfactory and with slight modification of the face mask, the apparatus can be used on any other small laboratory animal. We believe a similar machine can be satisfactorily adapted to the anesthetization of very small children.

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Blood Pressures in Experimental Hydronephrosis.

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The studies of numerous investigators[1, 2, 3] have suggested that reduction in the amount of functioning renal tissue in dogs is ac-

1 Passler, H., and Heineke, D., *Verhandl. d. deutsch. path. Gesell.*, 1905, **9**, 99.
2 Mark, R. E., *Z. f. Exp. Med.*, 1925, **46**, 1; 1928, **59**, 601.
3 Friedman, L., and Wachsmith, W., *Arch. f. Exp. Path.*, 1930, **101**, 173.

companied by a moderate rise in blood pressure. A similar rise has also been obtained by permanent obstruction of the ureter.[4, 5] The present study indicates, however, that when the reduction in functioning renal tissue is accomplished by means of experimental chronic hydronephrosis, produced by the partial constriction of the ureter, no rise in blood pressure occurs.

The experimental hydronephrosis in dogs was produced in one kidney by the method previously described,[6] followed by removal of the opposite kidney. The animals were roughly divided into 2 groups on the basis of physical symptoms, pyelographic findings, gross and microscopic pathology of the hydronephrotic kidneys, and blood findings: (1) The *chronic* group included animals in which intravenous pyelograms showed the disappearance of the calices of the kidney without further progression over a period of months, while no physical symptoms developed, the blood serum showed no significant deviation from normal, and a considerable amount of functioning renal tissue remained; these animals were in a non-uremic stage of hydronephrosis. (2) The *progressive* group included animals which developed signs and symptoms of severe renal involvement; the greater part of the renal tissue was destroyed—therefore excretory urographic shadows could not be obtained—and the animals progressed into the uremic stage of hydronephrosis, with marked acidosis and retention of non-protein nitrogen and inorganic phosphorus.

The mean blood pressures of all dogs were recorded without anesthesia by the connection of a cannula from a gauge 18 needle in the femoral artery directly to a mercury manometer. The readings (always taken in the morning) were made every 2 or 3 days for a period of 3 weeks before operation, to establish control blood pressure values. The experimental hydronephrosis was produced in the trained animals; blood pressure readings were then taken at bimonthly intervals as the animals developed chronic or progressive hydronephrosis. Ten animals from the chronic group were observed over a period of ±6 months, and 10 from the progressive group over a period of 2 to 6 months, or until death from acute uremia.

Table I shows blood pressures for 4 dogs in the chronic non-uremic group (those not included did not differ essentially from those reported). These pressures, it will be noted, differed only within experimental limits of error from the control pressures.

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4 Hartwick, A., *Z. f. d. ges. Exp. Med.*, 1930, **69**, 462.

5 Harrison, T. R., Mason, M. F., Resnik, H., and Rainey, J., *Trans. Assn. Am. Phys.*, 1936, **101**, 281.

6 Eichelberger, L., *J. Urology*, 1938, **40**, 366.

TABLE I.
Blood Pressures in Hydronephrotic Dogs.

	Mean Blood Pressure—mm. Hg														
	Range of 10 control readings	2 weeks after partial constriction	Weeks After Partial Constriction Plus Opposite Nephrectomy												
			2	3	4	6	8	10	12	14	16	18	20	22	24
			Chronic Hydronephrosis												
Dog F	120-130	130	130		132		130		125	130	125	125	125		
Dog J	118-125	125	122		118		125		122	125	125	120	120		125
Dog B	120-135	135	130		139		132		130	132	135	135	130		135
Dog D	120-130	125	122		128		125		127	127	125	120	128		122
			Progressive Hydronephrosis												
Dog O	130-140	130	128		140	138	140	138 140* 170† 175‡							
Dog C	120-130	130	150	140	130	172P	128		130	130	160P	135	130	130* 150† 180‡	
Dog E	125-135	140	130		140	140	172P		135	135	140	125	165P	137	135
Dog T	120-130	130	135		135	135	130 135† 180‡								

P following a reading indicates the animal was in pain.
* Three days before death.
† Two days before death.
‡ Day of death; N.P.N., mg%—Dog O, 200; Dog C, 250; Dog T, 205.

Table I also shows the blood pressures for 4 representative animals from the progressive group. In this group also, the mean blood pressures did not differ from the control pressures, except in the following instances, which cannot be considered as direct results of the decrease in amount of functioning renal tissue *per se:* (1) If, during the progress of the hydronephrosis, blood pressure readings were taken at a time when the sac of the hydronephrotic kidney was distended and the animal was sensitive to handling and pressure, a moderate elevation of blood pressure might be found. This was interpreted as the result of pain, since when readings were taken at a time when the sac was less full, and the animal was entirely comfortable, control values were found. (2) In a few animals, during the last few days of life a rise in blood pressure was found to accompany the severe uremia (non-protein nitrogen in excess of 200 mg %; serum phosphate 4.0 mM; marked acidosis); this was considered comparable to the rise found clinically in previously non-hypertensive patients when in acute uremia.

These results would indicate that dogs with only one kidney, in which a considerable portion of the renal tissue has been destroyed by intraureteral pressure, maintain a normal mean blood pressure.

Presumably in these hydronephrotic kidneys the increased pressure occurring in the kidney pelvis may not be great enough to reduce the blood flow in the kidney sufficiently to produce hypertension on the basis of ischemia. Hence, these findings are quite compatible with Goldblatt's conclusive demonstration that reduction of the blood flow to the functioning components of the kidney causes the development of hypertension.[7] It should be noted, too, that infection was absent, or present to only a minor degree, in the hydronephrotic kidneys. Renal vascular inflammatory changes were, therefore, not a factor, in contrast to their rôle in human pathology.

Summary. 1. Mean blood pressures were recorded on dogs at bimonthly intervals as they developed chronic or progressive hydronephrosis. 2. The data indicate that dogs with only one kidney, in which a considerable portion of the renal tissue has been destroyed by intraureteral pressure resulting from partial constriction of the ureter, maintain a mean blood pressure within normal limits.

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