TABLE I.

Animal No.	1	2	3	4	5	6	7	8
Initial B.P.	106	106	106	106	106	106	106	106
Progesterone	95	90	125	*	80	*	90	94
Nembutal	89	95	92	80	*	92	*	106

*Blood pressure recordings were not obtained at this point.

initially as well as the average of readings taken during the course of full surgical anesthesia which occurred within 20 minutes of injection and lasted one to two hours. Blood pressure is expressed as mm of mercury.

In no case did progesterone depress the blood pressure to any greater degree than nembutal, and in one case anesthesia occurred despite a rise in pressure. With both agents a brief preliminary rise of 10 mm Hg was noted immediately following injection. In the case of progesterone, a sustained rise of 15 to 25 mm was frequently noted before anesthesia, especially in those animals in which the steroid was given in divided doses. Such pressor effects with progesterone were interesting since Grollman, *et al.*,[3] claimed to have obtained a rise in blood pressure almost to hypertensive levels with chronic injections of this steroid.

Summary. Progesterone in anesthetic doses given intraperitoneally does not lower the blood pressure to any greater degree than does nembutal. Anesthesia with progesterone occurs without relation to pressor or depressor effects elicited by this steroid.

11937 P

Tensile Strength of Tibiae of Healed Rachitic and Normal Rats.*

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The early observation of Clark and Mrgudich[1] that rachitic and healed rachitic rat tibiae had lost the preferred longitudinal orientation of the crystal micelles of the inorganic material prompted

3 Grollman, Arthur E., Harrison, T. R., and Williams, J. R., Jr., *J. Pharm. and Exp. Ther.*, 1940, **69**, 149.

* Part of the expenses of this investigation were borne by a grant from the Nutrition Research Laboratories.

1 Clark, G. L., and Mrgudich, J. N., *Am. J. Physiol.*, 1934, **108**, 74.

investigation of the tensile strength of healed rachitic bone as compared with normal controls. Albino rats from the stock colony were used. At weaning, 5 litters, comprising 30 rats were divided equally into control and experimental groups. The control animals were kept on the stock diet, while the experimental group was placed on the Steenbock-Black rachitogenic diet. After 41 days the experimental animals were severely rachitic, as shown by X-ray photographs. They were then given 250 units of calciferol by mouth and placed on the stock diet. Prompt healing of the rickets resulted. The animals were continued on the stock diet for 6 weeks after the calciferol was fed, and at the end of this time no roentgenological difference could be demonstrated between the experimental animals and their littermate controls. The rats were killed by a blow on the head and the tibiae immediately removed and dissected free from soft tissues for determination of tensile strength.

The apparatus used is a modification of a standard engineering machine for testing structural material.† The tibia is supported on 2 blunt wedges 10 mm apart, and a blunted knife edge is placed on the bone from above to apply pressure. By means of a motor and suitable reducing gear a heavy weight is slowly pulled across the apparatus so as to increase the pressure on the bone through the knife edge. A writing point records the excursion of the weight, so that the total load on the bone at any instant may be calculated. Simultaneously a record of the elasticity of the bone is obtained from a gauge graduated in 0.001 inch attached to the knife edge. From such records accurate load deflection diagrams of each bone may be made by triangulation.

Preliminary results obtained from the group of 30 rats thus far studied are summarized in Table I.

The difference in breaking weight between the healed rachitic and normal bones is, in the case of the male animals, 3.51 times the

TABLE I.
Breaking Weights and Deflections of Rat Tibiæ.

	No. of bones	Sex	Mean breaking wt, g	Std. dev.	Mean def. 0.001 in.	Breaking wt / Deflections*
Healed Rachitic	10	M	9345	1142	15.1	619
	20	F	8309	896	15.0	854
Normal	8	M	13359	977	13.6	982
	22	F	11527	869	10.7	1086

*This figure represents an approximate measure of the elasticity of the bones.

† The machine was designed by Professor H. F. Moore of the College of Engineering of the University of Illinois.

standard deviation, and in the case of the females 3.59 times the standard deviation. Preliminary measurements of the cross section areas of a small number of the bones show that there is no significant difference between the healed rachitic and normal bones. While the array of results forms a somewhat skewed distribution curve, it is nevertheless probable that the differences in tensile strength between the roentgenologically healed bones and the controls are significant. Further studies are in progress.

11938

The Effect of Renin on the Cardiac Output.

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Since the original description of a pressor substance (renin) in the kidney by Tigerstedt and Bergmann there has been considerable speculation on the possible rôle that renin may play in the production and maintenance of experimental hypertension. As the pressor effect of renin can be obtained in perfused surviving organs and it has no effect on the isolated perfused heart,[1, 2, 3] it has been generally assumed that an increase in the cardiac output plays little or no part in the elevation of the blood pressure. Hessel[4] states that renin produces no increase in the cardiac output in dogs but gives no experimental data. The present study was carried out to determine what, if any, effect renin has on the cardiac output. Several determinations were made with tyramine hydrochloride, a drug which gives a pressor response similar to that of renin.

Method. The cardiac output was determined in one female and five male dogs by the use of the Fick principle following the method of Marshall.[5] All experiments were carried out in the morning under basal conditions. With the exception of three preliminary experiments the dogs received from 2.5 to 3.5 mg of morphine sul-

* Work done during tenure of the Charles Klingenstein Fellowship.

1 Tigerstedt, R., and Bergman, P. G., *Skand. Arch. Physiol.*, 1898, **8**, 223.

2 Hill, W. H. Phillip, and Andrus, Cowles E., PROC. SOC. EXP. BIOL. AND MED., 1940, **44**, 213.

3 Friedman, B., Abramson, D. I., and Marx, W., *Am. J. Physiol.*, 1938, **124**, 285.

4 Hessel, G., *Klin. Wohnschn.*, 1938, **17**, 843.

5 Marshall, E. K., *Am. J. Physiol.*, 1926, **77**, 459.