Hypertensive disease and sclerosis do not, of themselves, give rise to involution of the ovarian spiral arteries.

The significant fact may be mentioned in conclusion that a similar trophic action of estrogen on the endometrial spiraled arterioles is well known from the work of Okkels and Engle[7] (see [8], also). Hence it appears that the action of estrogen upon the vasculature of the genital tract includes not only its generally recognized effects within the uterus,[7] but it includes the ovaries as well. The hormone has, moreover, a particular predilection for spiraled arterial structures in these tissues.

7 Okkels, H., and Engle, E. T., *Acta path. et microbiol. Scandinav.*, 1938, **15**, 150.

8 Reynolds, S. R. M., *J. A. M. A.*, 1947, **135**, 552.

16404

Effect of Vagal Stimulation on Blood Glucose.

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There are conflicting reports in the literature concerning the influence of the vagus nerves on insulin secretion. Some reports[1-4] support the idea that vagal stimulation increases the pancreatic output of insulin. Other work[5-7] fails to substantiate this point of view.

Even with the marked increase in sensitivity to insulin due to hypophysectomy, Keller[8] was unable to produce hypoglycemia in the dog by faradic stimulation of the distal end of the vagi sectioned in the upper abdomen.

These conflicting reports and the studies of Portis and Zitman[9] who attempt to explain the clinical syndromes of weakness and fatigue in psychoneurotic patients on the basis of a vagal induced hyperinsulinism led us to make the following experiments.

Under sodium pentobarbital anesthesia one or both vagus nerves were isolated in the neck. The right vagus was stimulated alone in some dogs; the right and left vagi were stimulated consecutively in some dogs; and the right and left vagi were stimulated simultaneously in some dogs with the tetanizing faradic current. Stimulation was of sufficient strength to cause a marked slowing of cardiac rate as a criterion of vagal effects. Stimulation was continued from 40 minutes to 5¾ hours. Vagal stimulation was continuous except when asystole, cyanosis, apnea, retching or restlessness necessitated either decreasing the current or stopping the stimulation for periods not exceeding 2 minutes in any dog.

Insulin was given subcutaneously to 2 dogs, as indicated in Table I, to be sure that neither the anesthetic nor the vagal stimulation prevented insulin hypoglycemia.

The detailed results are recorded in the table.

Results. When insulin was given subcutaneously to Dog 8 during pentobarbital anesthesia, the usual hypoglycemia resulted. Similarly, when insulin was given subcutaneously to Dog 7 after 3 hours of vagal stimulation, and during continuation of the stimulation, the usual hypoglycemia resulted. This indicates that neither sodium pentobarbital nor vagal stimulation interferes with production of insulin hypoglycemia.

Dog 2 showed a blood glucose of 49 mg % 30 minutes after cessation of vagal stimula-

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5 Phillips, R. A., *Am. J. Physiol.*, 1933, **105**, 257.

6 Long, C. N. H., and Fry, E. G., *Am. J. M. Sc.*, 1933, **185**, 884.

7 Houssay, B. A., Lewis, J. T., and Foglia, V. G., *C. R. Soc. de Biol.*, 1929, **101**, 239.

8 Keller, Allen, personal communication.

9 Portis, S. A., and Zitman, I. H., *J. A. M. A.*, 1943, **121**, 569.

TABLE I.
The Effect of Vagal Stimulation on Blood Glucose in the Normal Anesthetized Dog.

Dog No.		1		1		2		3		4		5		6		7		8		8		9	
Vagal stimulation		(0)		(L then R)		(R)		(R)		(R)		(L and R)		(L and R)		(R)		(0)		(L and R)		(L and R)	
	Time interval after Faradic stimulation	Blood sugar*	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate	Blood sugar	Pulse rate
Control	—	125	—	82	180	109	204	87	180	76	150	87	180	93	160	75	210	81	—	93	204	112	174
	15	112	—	133	150	100	116	60	96	—	—	—	150‡	—	108	—	82	¶	—	—	84	110	64
	30	125	—	120	168	100	120	113	90§	76	132	97	180	98	120	75	144	—	—	—	—	—	36
	45	112‡	—	89†	180	75	208§	85	180	73	66	—	180	—‡	116	—	70	76	—	93	66	171	96
(1 hr)	60	123	—	121	72§	85‡	—	85	—	78	78	111	168‡	98	110	75‡	90	—	—	—	—	—	—
	75	123	—	92	—	49	—	79‡	—	78	122	—	—	—	104	—	96	48	—	—	—	182	72
	90	126‡	—	120	—	—‡	—	79	—	79	150	122	180‡	94	122	118	125	—	—	100‡	210	—	—
	105	80	—	140	—	79	—	—	—	84	145	—	180	—	108	—	130	—‡	—	—	—	160	84
(2 ")	120	88	—			—	—	—	—	85	72	122	180	—	90	—	110	—	—	—	84	—	—
	135					—	—	—	—	84	108§	—	180	90‡	72	107‡	174	31‡	—	98	84	—	—
	150					—	—	—	—	86	106	120	180	96‡	84	—	108	—	—	101	72§	—	—
	165					—	—	106	—	84	—	—	180	—	—§	—	—	27	—			148	84§
(3 ")	180					85	—	—	—			112	180	99‡	176	104	72	—	—				
	195							—	—			—	—‡	—	—	‖	—	25	—				
	210							—	—			—	—	—	—	94	100						
	225							—	—			127	180§	90	—	64	80						
(4 ")	240							—	—					—	—	—	—						
	255							—	—					—	—	42	174						
	270							—	—					—	—	—	—						
	285							—	—					85	—	36	86§						
(5 ")	300							—	—					—		—	—						
	315							128	—					—		40	198						
	330													—									
	345 (min.)													85									
Duration of exper.				61 (min.)		48		30		166		106		203		184				147		167	
Duration of vagal stimulation				40		48		30		166		66		179		184				127		151	
Period of no stimulation				21		0		0		0		40		24		0				20		16	

* Blood sugar values in mg %. † Left vagal stimulation ended, right vagal stimulation began. ‡2Additional doses of 5% sodium pentobarbital, approximately 1 cc amounts. § Vagal stimulation stopped. ‖ 10 U regular insulin given subcutaneously 3½ hr after beginning experiment. ¶ 18 U regular insulin given subcutaneously 15 min. after beginning experiment.

Note: Each dog received 0.5 cc of 5% sodium pentobarbital per kg body wt to produce anesthesia.

Dogs 2, 3, and 4 were fed Purina Chow 5 hr prior to experiment. Dogs 1 and 5 were non-fasting. Dogs 6 to 9 were fasting.

tion. With the exception of this isolated instance in one animal, there is no evidence, from our data on 8 other dogs, that faradic stimulation of either or both vagus nerves produces a hypoglycemia. From this we conclude that it is unlikely that vagal function influences insulin production.

The results were the same whether the animal was fasted 5 hours, 15 hours or fed just prior to the experiment.

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A Shaking Device for Multiple Containers.

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The need for simultaneous extraction of a series of fluids in separating funnels occasionally presents itself to the biochemist. In this department, for example, it has been necessary to carry out a number of separations by the Craig[1] counter-current method under conditions in which, either because of volume or tendency to emulsion, the stainless steel device used by Craig was inapplicable.

A piece of equipment has therefore been designed and constructed in the department workshop for the specific purpose of mixing the contents in 250-ml separatory funnels. In view of the comparative simplicity and low material cost of this shaker, as well as the interest it has aroused in workers from other departments, construction details are presented. Obviously, its design may be readily adapted to the use of containers other than separatory funnels.

This shaker (Fig. 1) has been built on a wooden table 24x47 inches, the legs of which are set on casters. Basically, the shaker consists of a frame bolted to a shaft which is turned by a small electric motor. The frame is designed to accommodate 12 separatory funnels in 2 rows, one on each side of the shaft. All structural material is ply-wood except for the half-inch shaft, its bearings, and bearing supports. A simple sheet metal cover over the motor switch has a nail soldered inside to act as a pin which passes through a hole in the shaft. By this device, the frame is held in an upright position when not in use, and accidental throwing of the switch is prevented.

The frame (Fig. 2) consists of 3 ply-wood pieces 9x34 inches. The top piece is removable while the lower 2 are fixed together and fastened to the shaft. The upper and lower pieces are cut from half-inch ply-wood, the center piece from quarter-inch ply-wood.

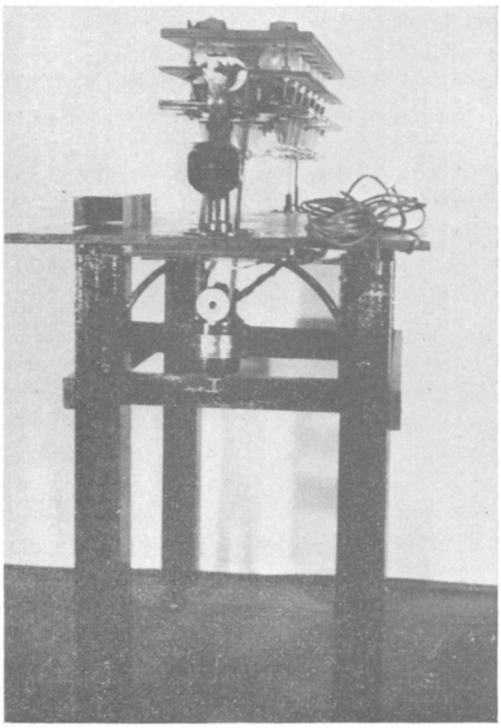

FIG. 1.

[1] Graig, L. C., *J. Biol. Chem.*, 1944, **155**, 519.