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Dependence of Ovarian Spiral Arteries on the Trophic Action of Estrogens.*

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Patterns of spiral arteries in rabbit[1-3] and human ovaries[4,5] have been described. Although there are differences in the arrangement of these vessels in the two species, homologous parts may be discerned. In the human, the ovarian artery passes along the hilus of the ovary, inosculating with its uterine branch. This may be called the basic branch and compares with the same vessel in the rabbit which lies wholly outside the ovary. There are numerous short tortuous vessels arising primarily from this basic vessel in the human and these primary branches compare with the single (or sometimes double[2]) ramus ovaricus in the rabbit. In the human, each primary branch normally gives rise to a number of still smaller helical blood vessels that pass in the folds of the posterior duplicature of the broad ligament as this envelopes the ovary. These secondary vessels compare with the ovarian spiral arteries originally described in the rabbit.[1,2] They give rise in turn to clusters of still smaller tertiary spiral arteries.

In the course of injecting the blood vessels in a series of about 60 pairs of human ovaries,[5] observations have been made on the distribution and character of the secondary and tertiary blood vessels in the human ovary. It is clear that, in the absence of estrogen these vessels undergo progressive involution and eventually disappear. They are highly developed, dense and numerous only in the presence of estrogen. The evidence for this is as follows:

a) In 4 hypertensive subjects past the menopause, the ovaries were sclerotic, one endometrium atrophic, and there were few or even no secondary and tertiary ovarian spiral arteries.

b) One normotensive subject with a recent menopause had a uterus with an actively proliferating endometrium. Her ovaries were sclerotic, but the secondary and tertiary ovarian arteries were profuse and well developed although the vessels were more widely spaced than in menstruating women.

c) Two hypertensive subjects, not yet past the menopause, possessed non-sclerotic ovaries. The ovarian spiral arteries were luxuriant and profuse.

d) Three normotensive premenopausal women possessed sclerotic ovaries and either proliferative, or early secretory endometria. The secondary and tertiary ovarian spiral arteries were profuse and highly developed.

e) Preparations from 6 fetal and neonatal ovaries showed a moderate to a profuse degree of spiraling of the minute ovarian blood vessels only in full term infants, at a time when, or soon after maternal hormones are known to exert a trophic action on the genital tract of the newborn female.[6]

It is clear from the above, therefore, that only when estrogen is demonstrably present are the ovarian spiral arteries of women well developed. In its absence they are atrophied.

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1 Reynolds, S. R. M., *Am. J. Obst. and Gynecol.*, 1947, **53**, 221.

2 Reynolds, S. R. M., *Endocrinology*, 1947, **40**, 381.

3 Reynolds, S. R. M., *Endocrinology*, 1947, **40**, 388.

4 Delson, B., Lubin, S., and Reynolds, S. R. M., *Endocrinology*, 1948, **42**, 124.

5 Delson, B., Lubin, S., and Reynolds, S. R. M., *Am. J. Obst. and Gynecol.*, in press.

6 Scammon, R. E., PROC. SOC. EXP. BIOL. AND MED., 1926, **23**, 687.

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.

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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-

1 Britton, S. W., *Am. J. Physiol.*, 1925, **74**, 291.

2 Deitrich, S., *Arch. f. exp. Path. u. Pharmakol.*, 1927, **125**, 336.

3 Clark, G. A., *J. Physiol.*, 1925, **59**, 446.

4 LaBarre, J., *Am. J. Physiol.*, 1930, **94**, 13.

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.