at the tip of the wire and its base at the surface of the tissue; thus this can be used for superficial or surface coagulation.

In use, the tip of the electrode is placed at the desired point within the embryo, and as the foot switch is intermittently depressed the control dial is gradually advanced until the desired coagulation results. For chick embryos we used a dial setting of about 17 to obtain an area of coagulation about 40 μ in diameter, which was what we needed to interfere with the formation of the sixth aortic arch.

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Glycogen Storage in the Heart in Experimental Renal Hypertension in the Rat

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Because of the rapidity with which glycogen disappears from the heart after death, there are no data on the normal glycogen content of the human heart and, therefore, none on the effect of clinical hypertension on cardiac glycogen stores. One report (*1*) on the total carbohydrate (including lactate acid) content of human hearts obtained many hours after death from various causes is suggestive. Increase in total carbohydrate was found in the hearts of subjects that had died of cardiac diseases. We have seen no report on the effect of experimental hypertension on the storage of glycogen in the heart, though a recent report by Olsen (*2*) describes certain other chemical changes in the heart in this condition.

Nine adult rats of the piebald Evans-McCollum strain made chronically hypertensive by Grollman's (*3*)[1] method constituted the experimental group. Systolic blood pressures as measured by the tail plethysmographic method ranged between 160 and 200 mm of mercury. The animals were fasted for 24 hr and sacrificed under sodium pentobarbital anesthesia. Samples of heart, liver, and skeletal muscle were obtained and analyzed for glycogen as described in an earlier communication (*4*). Blood ketones were determined by the method of Heilesen (*5*), modified for use of a tungstic acid filtrate. Ten normal rats subjected to the same regimen and sacrificed under like conditions were used as controls.

The analytical data are summarized in Table 1. There is no significant difference in the liver glycogen or the skeletal muscle glycogen in the experimental and control groups. The cardiac glycogen is significantly higher in the experimental group.

The function of glycogen in the myocardium is not known with certainty. Its concentration does not vary widely under physiological conditions, and changes that do occur may not be paralleled by changes in the glycogen content of the skeletal muscles or the liver. Indeed, in certain conditions such as fasting (*6*), pancreatic diabetes (*4, 7*), phlorizin poisoning (*8*), and use of ketogenic diets (*6*) the cardiac glycogen stores increase, whereas the glycogen content of skeletal muscle and liver may decrease or remain unchanged. Evans (*9*) showed that in rats caused to swim to exhaustion there is no decrease in cardiac glycogen, though the glycogen of the gastrocnemii is markedly depleted. Shelley, Code, and Visscher (*10*) found that rats subjected to daily bouts of exhaustive exercise show increased cardiac glycogen, with myocardial hypertrophy. Exposure to low oxygen tension results in greater depletion of cardiac glycogen than of skeletal muscle glycogen, and the suggestion has been repeatedly made that cardiac glycogen is a reserve to be called upon in hypoxemic states. Glycolosis is believed to be the principal if not the sole source of energy in the anaerobically beating heart (*11*).

Lackey (*4, 6, 12*) and co-workers have shown that in a variety of conditions in which a ketonemia is produced there is a positive correlation between blood ketone levels and cardiac glycogen storage. In the present experimental series, no increase in blood ketones was found and, therefore, no evidence that the increased storage of glycogen by the heart was related to a generalized disturbance in carbohydrate utilization. It is not clear whether an increased storage of cardiac glycogen should be regarded as a reserve against emergencies or as an indication of a lessened capacity for its utilization. However, in a number of conditions in which there is an increase in the cardiac glycogen storage, the disappearance of glycogen from the heart after death is as rapid as in the hearts obtained from normal animals (*13*).

TABLE 1

TISSUE GLYCOGEN AND BLOOD KETONE LEVELS IN HYPERTENSIVE AND NORMAL RATS FASTED FOR 24 HR

Series	No. animals	Glycogen as glucose (mg/100 g tissue)			Blood ketones (mg/100 ml as hydroxybutyric acid)
		Liver	Heart	Muscle	
Normal	10	303 ± 180*	576 ± 37*	318 ± 36*	19.1 ± 6*
Hypertensive	9	363 ± 129*	761 ± 21*	410 ± 57*	15.3 ± 3*

* Standard error of mean.

[1] I wish to thank Arthur Grollman for supplying the hypertensive animals for these observations.

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The Direction of Flow in the Blood Vessels of the Infundibular Stalk

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Recent work, especially that of G. W. Harris (*1*), has drawn attention to a possible neurohumoral relay between the hypothalamus and the anterior lobe of the hypophysis. The neural link in the chain consists of nerve fibers from the hypothalamus to the median eminence and infundibular stem, where the humoral substance originates and is transmitted to the adenohypophysis via the hypophyseal-portal blood vessels.

The direction of blood flow in the vessels of the stalk forms an important part of the theory. Wislocki (*2*), as well as Green and Harris (*3*), suggested that the direction of flow was from the median eminence and infundibular stem to the adenohypophysis. This is in contradistinction to the report of Popa and Fielding (*4*), in the original description of the hypophyseal-portal vessels, stating the blood flow was from the anterior hypophysis up the stalk to the hypothalamus.

During recent investigations into the anatomy and physiology of the pituitary gland and stalk (*5*), a surgical procedure was developed to expose completely the stalk and rostral portion of the pituitary gland to direct vision. The operative approach was parapharyngeal, and the blood vessels supplying the hypophysis were not disturbed. It occurred to us that with this exposure the direction of flow in the blood vessels of the infundibular stalk could be visualized.

The procedures were carried out on adult albino rats. The region of the infundibular stalk was exposed surgically with the animals under ether anesthesia. By this means the blood vessels of the infundibular stalk were brought into view. The chest was then opened, and a fine glass cannula was introduced into he proximal aorta through the wall of the left ventricle. Less than 5 ml of a 50% aqueous suspension of Higgins waterproof India ink was injected slowly while the infundibular stem blood vessels were viewed with a binocular dissecting microscope. India ink usually appeared in several vessels within a few seconds of the beginning of injection. This occurred before any other vessels or tissues in the operative field became injected. With a just appreciable lag, involving perhaps no more than a second, additional vessels, usually 3 in number, became filled. By varying the quantity of ink injected, as well as the injection pressure, it was possible to fill selectively the first-mentioned vascular channels, or to fill all of them (an average of 6). It was noted repeatedly that the flow of India ink in all these vessels was invariably from the stalk to the body of the hypophysis. The vessels extended from the anterior part of the stalk posteriorly to the pituitary gland, where they ramified. They had the approximate width of a very fine silk thread. One or two of these vessels entered the posterior lobe. The pars distalis did not become colored until the India ink passed down the vessels of the stalk to the gland, nor did the distalis become colored if the stalk was severed just prior to injection. We have therefore been able to substantiate the anatomical observations of Wislocki (*2*) and Green and Harris (*3*) by direct visualization of the flow of India ink in the infundibular stalk vessels.

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A Universal Line Graph for Estimating Percentage Potency in Multidose Assays

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In 1947 the author (*1*) presented in these columns simple formulas for calculating percentage potency in 3- and 4-dose assay procedures, when the log dose-response curves of the unknown and standard materials are both linear and parallel. Later Harte (*2*) demonstrated that each formula was reducible to a single line which, under the conditions of that test, could be used for a rapid graphic determination of the percentage potency. This was a distinct improvement over the use of radial lines employed by Knudsen (*3*), but still left much to be desired, since the position of the line varied with the two parameters: C, the log ratio of the concentration of the unknown to that of the standard, and d, the log interval between the successive doses of both the unknown and standard. Harte, however, avoided the second parameter by the use of a fixed d and established a