

sensori-motor area. In the monkey the cortical effect was more restricted and appeared in the precentral motor cortex (area 4 and 6) and, to a lesser extent, in the adjacent part of the postcentral sensory cortex. When proprioceptive impulses were elicited in a hindleg of a monkey the area of greatest change of potentials was in a hindleg point of the contralateral motor cortex. Lesser degrees of ex-

citation were seen in arm and face area and also in the ipsilateral leg area.

It is suggested that proprioceptive impulses to area 4 are involved in voluntary movements. Their absence accounts for the loss of voluntary movements of the deafferented limb in the monkey although no change occurs in its responsiveness to electrical stimulation of the motor cortex.

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Effect of Curare on Autonomic Reflexes.*

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The current trend to utilize curare for muscular relaxation during surgical anesthesia has led to observations disclosing a depressor effect of curare for certain autonomic reflexes.

a. Celiac plexus reflex. Pressure stimulation at the celiac plexus during surgical manipulation may result in reflexogenic changes consisting of a fall in the systolic blood pressure, a marked diminution in pulse pressure, little or no change in pulse rate, and rigidity of abdominal muscles.^{1,2} Administration of therapeutic doses of d-tubocurarine to 12 patients who presented such a syndrome during upper abdominal surgery, not only provided adequate muscular relaxation but also resulted in a return in the arterial blood pressure and in the pulse pressure to normal which were then sustained.

Experimental investigations in a series of 8 dogs corroborated the clinical observations.

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¹ Burstein, C. L., and Rovenstine, E. A., *Arch. Surg.*, 1939, **35**, 599.

² Burstein, C. L., and Rovenstine, E. A., *Curr. Res. Anesth. and Analg.*, 1938, **17**, 134.

The arterial hypotension with marked decrease in pulse pressure produced by compression of the celiac ganglion were not obtained after the intravenous administration of d-tubocurarine (2 units per kg).

b. Carotid sinus reflex. The results observed following the intravenous injection of d-tubocurarine during carotid sinus stimulation showed similar effects.³

Two patients having radical neck dissection manifested characteristic carotid sinus reflex activity with severe arterial hypotension and significant bradycardia. The intravenous administration of 40 units of d-tubocurarine resulted in a restitution of the arterial blood pressure and the pulse rate within 3 minutes.

Experimental investigations in a series of 8 dogs showed similar consistent effects. Upon pulling a tape applied about the carotid bulb during pentothal sodium anesthesia there followed arterial hypotension and bradycardia. After the intravenous administration of d-tubocurarine (2 units per kg) repeated stimulation of the carotid sinus failed to elicit any cardiocirculatory disturbance.

³ Heymans, C., Bouekaret, J. J., and Regniers, P., *Le sinus Carotidien*, G. Doin et Cie, Paris, 1933.

c. *Vagal reflex.* In 1857 Claude Bernard showed that the bradycardia or asystole produced by stimulation of the vagus nerve could no longer be obtained after a dog had been curarized.⁴ Present clinical and experimental studies have corroborated this long neglected observation.

Faradic stimulation of the vagus nerve low in the neck (10 dogs) during general anesthesia with ether or pentothal sodium caused arterial hypotension as well as bradycardia. The same stimulation after the intravenous

injection of d-tubocurarine (2 units per kg) did not cause hypotension or bradycardia.

Clinically, these reflexes of circulatory depression during intrathoracic surgery due to vagal stimulation have been corrected by administration of d-tubocurarine.

Conclusion. The intravenous administration of a dose of d-tubocurarine sufficient to produce intercostal paralysis may abolish or alleviate certain autonomic reflexes that may be encountered during stimulation of autonomic pathways. Cardiocirculatory disturbances incident to stimulation of the celiac plexus, the carotid sinus, and the vagus nerve, may be so treated.

⁴ Bernard, Claude, *Leçons Sur les Effets des Substances Toxiques et Médicamenteuses* (May 30, 1856), J. B. Baillière et Fils, Paris, 1883, 2nd edition, p. 348.

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Influence of Estradiol on Alkaline Phosphatase Activity in the Genital Tract of the Rat.

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That alkaline phosphatase may play some integral part in the physiological changes occurring in the genital tract during the normal reproductive cycle and during pregnancy was first suggested by the work of Wislocki and Dempsey.¹ More recently the influence of steroids on alkaline phosphatase distribution in the genital tract of the mouse has been studied by Atkinson and Elftman,² and by Jenner,³ who found that estradiol caused increased phosphatase activity in the circular musculature and in the epithelium of the glands and of the lining of the uterus and vagina of castrated animals.

The work reported here concerns the effect of estradiol on the alkaline phosphatase activity in the genital tract of the rat with special emphasis on its activity in the endo-

metrium of the cervix.

Methods and results. Forty-two albino rats were used in these experiments. Estradiol dissolved in sesame oil, was injected subcutaneously in doses ranging from a single injection of 0.2 μ g to 7 daily injections of 0.4 μ g. At autopsy, the genital tract, including the vagina, cervix, and uterus, was removed and prepared after the method of Gomori⁴ for histochemical determination of alkaline phosphatase. Table I summarizes experimental observations.

Notes on table. A series of 8 female rats, given 2 to 3 weeks rest after removal of ovaries, served as controls. The least amount of phosphatase activity found in any one place in the genital tract of these animals has been assigned a value of one. The amount of enzymic activity found in other locations in the same animals and in treated animals is expressed in multiple factors of this unit.

¹ Wislocki, G. B., and Dempsey, E. W., *Am. J. Anat.*, 1945, **42**, 23.

² Atkinson, W. B., and Elftman, H., *Proc. Soc. Exp. Biol. and Med.*, 1946, **62**, 148.

³ Jenner, R., *Nature*, 1947, **159**, 578.

⁴ Gomori, G. J., *Cell. and Comp. Physiol.*, 1941, **17**, 71.