

In none of the animals here reported were convulsions observed. Consistent results with various fixed periods of cephalic vascular stasis have not been previously reported.^{2, 3} Our results are to a large extent predictable because we have accomplished complete stasis and eliminated anesthetic agents.

Histologic studies are in progress. These already indicate that the first cells to suffer from circulatory stasis are the Purkinje cells of the cerebellum, which correlates with the ataxic symptoms observed in our dogs.

10492 P

Influence of Asphyxia on Reciprocal Innervation.

A. VAN HARREVELD.

From the William G. Kerckhoff Laboratories of the Biological Sciences, California Institute of Technology, Pasadena.

In cats whose spinal cords had been asphyxiated for various periods of time, van Harreveld and Marmont¹ found that after recovery the hind legs could show an exaggerated extensor tone which usually stayed until death (in some experiments 3 weeks in total). Considering that after this time the acute effect of asphyxia will have disappeared, it was concluded that the high extensor tone is caused by a more or less selective damage of an inhibiting system present in the cord which normally keeps the tone in check.

A further study of these phenomena was made. A period of asphyxia for 30 minutes was usually followed within a few hours by the development of high extensor tone without abolishment of the flexor reflex. However, the effect of pinching the foot was not the same in all animals; sometimes it caused a regular flexor reflex with flexion in ankle and knee; in other experiments it caused, after the flexion, an extension of the leg, and in a few cases this stimulus caused instead of flexion an increase of the extensor tone.

When the contractions of the *M. tibialis anterior* and the triceps group were recorded simultaneously, it was often seen that stimulation of the *N. peroneus superficialis* caused a contraction in both of these antagonists. In other animals it was observed that the stimula-

² Pike, F. H., Guthrie, C. C., and Stewart, G. N., *J. Exp. Med.*, 1908, **10**, 490.

³ Gildea, E. F., and Cobb, S., *Arch. Neurol. and Psychiat.*, 1930, **23**, 876.

¹ Harreveld, A. van, and Marmont, G., *J. Neurophysiol.*, 1939, **2**, 101.

tion of the sciatic nerve caused a contraction in the quadriceps muscle of a Sherrington preparation instead of inhibiting the tone in this muscle.

By Sherrington's scheme for reciprocal innervation these observations can be best explained. Since, according to this scheme, the extensor motor neurones are inhibited during the flexor reflex, the simultaneous stimulation of afferent nerve fibers for extensor reflexes (*e.g.*, for the extensor thrust) running in the stimulated branch of the sciatic nerve, normally remains without effect. If the internuncial neurones transporting the impulses inhibiting the extensor motor neurones were damaged, it must be expected that the flexor and the extensor reflexes would occur at the same time. Thus the abolition of reciprocal innervation by asphyxia strongly supports the assumption that asphyxia damages the inhibitory neurones more severely than the excitatory ones.

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Effect of Heptyl Aldehyde on a Spindle-Cell Sarcoma.

WILLIAM G. CLARK. (Introduced by Eaton M. MacKay.)

From The Scripps Metabolic Clinic, La Jolla, California.

Strong¹ recently reported an inhibitory effect of heptyl aldehyde on spontaneous mammary tumors in mice and later² in dogs. He reported an inhibitory effect on the growth-rate of the mouse mammary tumors with complete regressions, gross and histological alterations, especially liquefaction of the tumors and high percentages of regressions in dog mammary tumors. In the mice he administered the heptyl aldehyde in the stock diet but directly injected small amounts of the undiluted aldehyde into the dog tumors. An attempt was made to extend Strong's results using a transplantable sarcoma in the rat.

The tumor used was a spontaneous sarcoma found in the liver of a female rat. It was highly malignant, of the spindle-cell type and retained its original vigor through the tenth passage in our particular strain of rats.³ Tumor passage was afforded either by subcutaneous injection through a trochar of a single piece or a macerate or intraperitoneally by injection of a fine macerate in Tyrode's

¹ Strong, L. C., *Science*, 1938, **87**, 144; *Am. J. Cancer*, 1938, **32**, 227.

² Strong, L. C., and Whitney, L. F., *Science*, 1938, **88**, 111.

³ MacKay, L. L., and MacKay, E. M., *Am. J. Physiol.*, 1927, **83**, 179.