valves was somewhat greater at the smaller stroke volume. Similar results were obtained in second subject with a larger leak who was also studied on different occasions when the cardiac output differed markedly. This suggests that the percentage of leak is not independent of the total cardiac output but that the leak is relatively smaller at the higher level of output. Such a finding is in agreement with physiological expectations. The heart rate is generally faster when the stroke volume is increased and hence the duration of diastole, in which regurgitation occurs, is shorter. These studies are being continued on a larger scale, but it already appears that this method provides an acceptable quantitative measure of aortic or mitral regurgitation in man.

10491 P

Behavior of Dogs after Complete Temporary Arrest of the Cephalic Circulation.*

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The authors have previously reported a method of decerebration of the dog by means of cephalic vascular stasis.[1] The emphasis at that time was upon the use of the procedure as a method for preparing decerebrate animals. Since that time our interest has shifted to the evidence that could be gained concerning the resistance of various cells in the brain to temporary cessation of blood flow and the correlation of these changes with changes in the behavior of the animal.

The technic has been modified to the following form: Two days after laminectomy at the second cervical level, the animal is atropinized, and a metal tracheal tube is inserted orally. A blood pressure cuff is wrapped about the neck, and the pressure is raised quickly to 700 mm Hg, at which level it is maintained as long as vascular arrest is desired, artificial respiration being administered through the tracheal tube. The completeness of circulatory

* Assistance in the preparation of these materials was furnished by the personnel of Works Progress Administration Official Project No. 665-71-3-69.

1 Kabat, Herman, and Dennis, Clarence, PROC. SOC. EXP. BIOL. AND MED., 1938, **38**, 864.

arrest in the head is determined by ophthalmoscopic examination of the retinal vessels. With this technic, consciousness is lost in a very few seconds, the corneal reflex disappears in from 20 to 40 seconds, and spontaneous respiration ceases in from 40 to 90 seconds.

In 6 adult animals there was complete cessation of cephalic blood flow for periods of from 2 to 10 minutes. In the dogs subjected to 8 minutes or less of compression the lid wink returned in 5 to 8 minutes; in 2 animals treated for 10 minutes it did not return till later. The first gasp occurred in 1 to 5 minutes and satisfactory spontaneous respiration returned in 2 to 11 minutes, the slowest return being after the longest compression. Intermittent spontaneous, high-pitched vocalization, associated with vigorous running movements of all 4 limbs, with the dog lying on his side, often occurred during the first few hours. The 2-minute dog recovered consciousness within an hour, the 4- and 8-minute animals within 48 hours, and the 10-minute dogs failed to recover consciousness, even though surviving 4 and 6 days, respectively. Thus, as far as consciousness was concerned, the critical period of stoppage of cephalic blood flow in these dogs was between 8 and 10 minutes.

Among the 4 animals recovering consciousness, the dominant symptom was ataxia, the severity of which bore a fair relationship to the duration of compression. Early after compression these animals were comatose, later conscious but apathetic and dull. Either one or 2 days later, the first conscious movements were displayed in crawling in extremely ataxic fashion on the belly, the weight frequently being carried in the case of the fore paws on the dorsum instead of the pad of the foot. These animals showed a paucity of spontaneous movement for several days, but by the 10th day normal activity had returned, leaving only the ataxia as a residual symptom. Most of these dogs had been trained to sit up, shake hands, etc., prior to arrest of circulation; and all recovered these behavior patterns.

The dogs surviving 10 minutes of cerebral vascular stasis did not respond to visual, auditory, or olfactory stimuli. The animal would lie on its side, showing only slight extensor rigidity and occasional intermittent running movements of all 4 limbs often associated with vocalization. Postural and righting reflexes were absent. Simpler spinal and brain stem reflexes were normally responsive. If the dog's mouth were placed in milk, it would lap mechanically, slowing gradually and stopping after an ounce or two had been ingested.

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.

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In cats whose spinal cords had been asphyxiated for various periods of time, van Harreveld and Marmont[1] 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.