10042 P

Decerebration in the Dog by Complete Temporary Anemia of the Brain.

HERMAN KABAT AND CLARENCE DENNIS. (Introduced by M. B. Visscher.)

From the Department of Physiology, University of Minnesota, Minneapolis.

In many investigations, it is desirable to perform experiments upon animals in the absence of anesthetics. The usual procedure is that of Sherrington, namely, to decerebrate surgically under ether, to stop the anesthesia, and to proceed at once with the experiment.[1] Under such conditions, profound shock and the depression due to the residual anesthetic agent throw some doubt on the reliability of the results. A more delicate method of decerebration has been devised for the cat by Pollock and Davis.[2] In this method, an anemic decerebration is produced by ligation of the basilar and carotid arteries under ether anesthesia. In the dog, the inaccessibility of the basilar artery makes this a difficult method.

It has been shown that the nerve cells of the brain stem are much more resistant to anemia than are those of the cerebral hemispheres.[3] It is therefore possible to produce extensive cerebral damage by a period of anemia which will allow the cells of the brain stem to recover. Guthrie, Pike and Stewart[4] and Gildea and Cobb[5] studied the effects of temporary occlusion of the 4 chief cerebral vessels in the neck under ether anesthesia, and were unable to predict the severity of the destruction produced by a given period of such occlusion. The variability in their results was due primarily to their failure to interrupt the flow in the spinal arteries, which is not constant from animal to animal. A further uncertainty must have resulted from variations in the depth of anesthesia just prior to the occlusion of the cerebral vessels.

We have devised a method of decerebration depending upon complete temporary anemia of the brain of the dog in the absence of anesthetics. A preliminary surgical procedure is used, consisting in removal of both laminæ and spine of the second cervical vertebra

1 Liddell, E. G. T., and Sherrington, C., *Mammalian Physiology*, Clarendon Press, Oxford, 1929.

2 Pollock, L. J., and Davis, L., *Arch. Neurol. and Psychiat.*, 1923, **10**, 319.

3 Gomez, L., and Pike, F. H., *J. Exp. Med.*, 1909, **11**, 257.

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

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

and ligation of both vertebral arteries. After an interval of one to 2 weeks, the dog is given one to 2 mg of atropin sulfate to prevent vagal cardiac inhibition, and placed, back down, on the table. A large blood-pressure cuff is wrapped around the neck, and the trachea intubated orally. By means of compressed air, the cuff is rapidly inflated to a pressure of 350 mm Hg and maintained at this pressure. This procedure causes sudden complete anemia of the head since not only the carotids and the cervical branches of the subclavians are occluded, but also the spinal vessels, by virtue of compression of the spinal cord.

Sudden complete anemia of the head is followed by unconsciousness. There is a period of asphyxial activity and panting for a short time. After several minutes, respiration stops, the animal becomes flaccid, and the lid wink reflex disappears. As soon as respiration stops, rhythmic artificial respiration is begun through the tracheal cannula. The heart usually beats rapidly and vigorously throughout the procedure, but must be followed closely for signs of vagal inhibition. After a period of anemia of 15 to 20 minutes, the pressure is released. Artificial respiration is continued until the dog begins to breathe spontaneously. The lid wink returns some minutes later. It is necessary to keep the animal in a constant-temperature box at 29°C.

We have kept a dog alive for 9 days after 19 minutes of complete anemia. He appeared comatose throughout, could not stand, but lay on whichever side he was placed, showing occasional rhythmic running movements of all 4 limbs together with high-pitched vocalization. The extensor tonus was only moderately increased. Temperature regulation was markedly impaired, and no change resulted from an injection of foreign protein which caused marked fever and panting in a normal control dog. There was no evidence of the presence of sensations of smell, vision, taste, hearing, or pain. The dog was unable to eat, and had to be fed by stomach tube. Such brain-stem reflexes as the light reflex, lid wink, swallowing, and vomiting could be elicited. The carotid sinus and aortic arch reflexes were readily obtained, and demonstrated a hyperactivity of the cardiac vagus. Arterial blood pressure was 130 mm Hg. There was a normal respiratory response to administration of 2.5% to 10% CO_2 in oxygen. No change was noted in the cerebral activity of this dog during the 9 days of observation.

The brains of several such animals are being studied histologically and subsequent reports will be made on this and other aspects of the investigation.