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Carotid Receptors Essential in the Gasping of the Isolated Rat Head.

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Selle, Hiestand and others[1-4] have demonstrated gasping phenomena in the isolated ischemic rat head and determined the variations with age and drug administration prior to decapitation. In duplicating these experiments a single edge razor blade held vertically in a blade holder was used,[4] and the section made from the dorsal surface as far cephalad as possible. Subsequent examination of the heads revealed that the section had passed caudal to the carotid sinus-body region leaving this mechanism intact. To evaluate the possible role of this structure in the gasping phenomena, male rats were anesthetized with chloralose (0.4 g per kg body weight), previously shown to increase the survival time and total number of gasps more than 250% in rats of a younger age group,[1] and the carotid sinus body located bilaterally through a midline incision. Decapitation was then performed in one group of 10 animals caudal to these structures and in another group of 10 above them, the surgical technic being as identical as possible in all cases. The rats weighed from 50 g to 64 g, a range previously shown to have a characteristic and fairly constant gasping response. The results shown in Table I indicate that only when the carotid mechanism remains connected to the brain does gasping occur.

Since rats of this weight group show both an initial aerobic series of gasps and a later anaerobic series[4] it must be concluded that the carotid drive is essential to both.

In 2 dogs (7.6 and 11.3 kg) under nembutal anesthesia gasping (depression of the mandible) was produced by occluding the trachea which was subsequently released and artificial respiration instituted to restore the animals when respiratory efforts had ceased. After removal of the carotid sinus-body region by resecting this area of the common carotid artery and its branches (ext. carotid, int. carotid and occipital artery), tracheal occlusion caused marked diaphragmatic and intercostal hyperpneic efforts, but no mandibular movements occurred. Removal of the carotid reflex mechanism caused, of itself, no change in respiratory rate or depth prior to tracheal occlusion.

TABLE I.
Effect of Removal of Carotid Sinus-body on Gasping in Isolated Rat Head.

	No. of rats	Avg gasping time	Avg No. of gasps
Carotid mechanism intact	10	83 sec	18
Carotid mechanism removed	10	0	0

On the basis of the above findings, Hiestand and Thoms reinvestigated the absence of gasping in mice. They have shown (private communication) that in this species the carotid body lies at the level of the pectoral girdle and that the section must be caudal to the forelegs before any gasping will occur.

Thus the primary drive to the respiratory center producing mandibular movements in the ischemic rat head, at least under chloralose anesthesia, is a reflex from the carotid receptors. This explains in part why some narcotic drugs decrease survival time and number of gasps in the ischemic head (ether, chloroform, nembutal) whereas others (chloralose) greatly increase these factors. The latter, although diminishing the response of the center to direct chemical influence, increases the sensitivity and effectiveness of the carotid chemoreflex system.[5] Further in-

1 Selle, W. A., PROC. SOC. EXP. BIOL. AND MED., 1942, **51**, 50.

2 Selle, W. A., PROC. SOC. EXP. BIOL. AND MED., 1945, **58**, 24.

3 Tschirgi, R. D., and Hiestand, Wm. A., *Anat. Rec.*, 1944, **89**, 543.

4 Hiestand, Wm. A., Tschirgi, R. D., and Miller, H. R., *Am. J. Physiol.*, 1944, **142**, 153.

5 Schmidt, C. F., *Anesthesia and Analgesia*, 1940, **19**, 261.

dication that the essential drive producing gasping phenomena is of chemoreflex origin lies in the nature of these structures. Their extreme ruggedness enables them to activate the central neurones under conditions which render these latter unable to respond to autogenous chemical stimuli. The ability of the carotid body to function during severe anoxia provides the organism with a last line of respiratory defense, Schmidt's ultimum moriens of respiratory control.[6]

[6] Bard, P., *MacLeod's Physiology in Modern Medicine*, C. V. Mosby Co., St. Louis, 1941, p. 593.

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Mutual Depression of Reabsorption and Excretory Maxima in Renal Tubules.

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It is generally understood that the capacity of the renal tubule cells to transport a substance in one direction is not influenced by the simultaneous transport of another substance in the opposite direction. Thus Smith *et al.*[1] report that simultaneous determinations of the maximal tubular reabsorption of glucose (Tm_G) and the maximal tubular excretion of *p*-aminohippuric acid (Tm_{PAH}) yield the same values as separate determinations, and elsewhere Smith[2] illustrates a set of simultaneous measurements of these 2 functions. This is also presumably true for the dog.[3,4] However, when individual experiments are examined, there is, in some instances, evidence of depression of Tm_G by PAH and of Tm_{PAH} by glucose. We have therefore reexamined this question of mutual depression of the transport of glucose and PAH in the dog.

Experimental. Tm_G and Tm_{PAH} were determined in 3 trained, unanesthetized female dogs ranging in body weight from 17 to 24 kg. Tm_G alone was determined during 3 consecutive urine collection periods of approximately 10 minutes each. After a priming dose of PAH, this substance was added to the infusion fluid and Tm_{PAH} and Tm_G were determined simultaneously during 3 additional periods. On another day this procedure was repeated in the same dog but the substances were reversed, *i.e.*, Tm_{PAH} alone was measured first. The creatinine clearance was taken as a measure of the glomerular filtration rate. The dogs were hydrated with 50 cc of water per kg of body weight by stomach tube 45 minutes before U_O. Plasma levels of creatinine, glucose and PAH were obtained by suitable priming injections into the jugular vein 10 minutes before U_O and maintained by constant intravenous infusion into a leg vein by a gravity mercury-drip pressure apparatus. In all experiments the load/Tm ratio for both glucose and PAH was well above 1.5. Urine was collected by an indwelling bladder catheter and blood was withdrawn periodically from the femoral artery through a 19-gauge retention needle.

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[1] Smith, H. W., Goldring, W., Chasis, H., Ranges, H. A., and Bradley, S. E., *J. Mt. Sinai Hosp.*, 1943, **10**, 59.

[2] Smith, H. W., *Porter Lectures*, Series 9, 1939, Univ. Extension Division, Univ. of Kansas, Lawrence, Kansas. Revised 1943.

[3] Selkurt, E. E., *Am. J. Physiol.*, 1944, **142**, 182.

[4] Eiler, J. J., Althausen, T. L., and Stockholm, M., *Am. J. Physiol.*, 1944, **140**, 699.