9880 P

Influence of Oxygen Deficiency on Reflex Dilatation of the Pupil.

BORIS URY AND ERNST GELLHORN.

From the Department of Physiology, College of Medicine, University of Illinois, Chicago.

The purpose of the studies reported in this paper is twofold: 1. To extend the systematic observations of Gellhorn[1] and collaborators on the effect of oxygen deficiency on the central nervous system to the autonomic nervous system. 2. To choose a reaction which is determined exclusively or to a large extent by a central inhibitory process in order to study the influence of oxygen deficiency on such processes.

Although the literature on the problem of reflex dilatation is still controversial a number of authors have shown that this reaction is largely due to an inhibition of the parasympathetic tonus of the Edinger-Westphal nucleus. This is particularly clear from the work of Bain, Irving and McSwiney,[2] who find that the stimulation of the central end of the splanchnic fails to elicit pupillary dilatation after sectioning of the third nerve, although "section of the cervical sympathetic nerves has not been found to alter either the rate or the degree of the reaction to stimulation of somatic or visceral afferent fibers."

The experiments were carried out repeatedly on 12 rabbits, half of which had the cervical sympathetic cut on one side. The sciatic was exposed under light urethane anesthesia and 2-3 hours later the experiment was begun. The threshold reaction for pupillary dilatation was determined with faradic stimulation (Harvard inductorium, shielded electrode) while the animals inhaled air or a known O_2-N_2-mixture from Douglas bags.

Very weak currents (coil distance 11-12 cm. or at an angle) were used for 1-3 seconds and the pupillary reaction was measured with a telescope.

The results were uniform and showed that under the influence of 6-8% O_2 the threshold for pupillary dilatation was raised. This reaction was completely reversible. The same concentration of

1 Gellhorn, E., and Spiesman, I., *Am. J. Physiol.*, 1935, **112**, 519, 620, 662; Gellhorn, E., *Am. J. Physiol.*, 1936, **115**, 679; 1936, **117**, 75; Gellhorn, E., and Joslyn, A., *J. Psychol.*, 1936, **3**, 161; Gellhorn, E., *Am. J. Psychiatry*, 1937, **93**, 1413; Gellhorn, E., and Kraines, S., *Arch. Neur. a. Psych.*, 1937, **38**, 491; Gellhorn, E., and Storm, L. F. M., *Acta Oto-Laryngol.*, 1938, in press.

2 Bain, W. A., Irving, J. T., and McSwiney, B. A., *J. Physiol.*, 1935, **84**, 323.

oxygen inhaled in the presence of 5% CO_2 did not produce any significant changes in threshold. Control experiments with inhalation of 5% CO_2 in air showed no change in threshold. These experiments are in agreement with the earlier work of Gellhorn and collaborators on the effect of oxygen deficiency and CO_2 on cortical and subcortical reflexes.

The pupillary reaction on the normal and the sympathectomized side was observed independently and simultaneously by 2 observers. The threshold was the same on both sides. The humoral excitation of the pupil occurs only with far stronger and more prolonged stimulation and is distinguished from the quick reaction observed in our experiments by its longer latent period and duration. Therefore it may be concluded that the pupillary reaction under the conditions studied is due to an inhibition of the parasympathetic center of the third nerve. The experiments show that excitatory and inhibitory processes are altered in a similar way under oxygen deficiency.

9881 P

Rôle of Afferent Nerves in Response of Vasomotor Center to Oxygen Deficiency.*

E. LAMBERT AND E. GELLHORN.†

From the Department of Physiology, College of Medicine, University of Illinois, Chicago.

Through numerous investigations it is known that the increased respiratory response to CO_2 is due less to its effect on peripheral receptors than on the respiratory center itself, whereas a similar response to oxygen deficiency occurs only when the carotid sinus nerves are intact. The removal of the afferent fibers by elimination of all known "buffer nerves" results in a failure of respiration during O_2 deficiency (Selladurai and Wright,[1] Schmidt,[2] v. Euler and

* This work will be published in detail in the series of Medical Monographs published by the College of Medicine of the University of Illinois.

† Aided by a grant from the Graduate School of the University of Illinois to E. Gellhorn.

1 Selladurai, S., and Wright, S., *Quart. J. Exp. Physiol.*, 1932, **22**, 233.

2 Schmidt, C. F., *Am. J. Physiol.*, 1932, **102**, 94.