to make a similar comparison of the effects of intravenous injections of sodium sulphide on reflex response and respiratory movements.

The anterior tibialis muscle was isolated, its insertion severed and secured by a thread to a writing lever. A large copper indifferent electrode was fastened to the shaven side of the neck, and a sharp point electrode thrust into the plantar surface of the foot. The trachea was connected to a rebreathing tank for a recording respiration.

An increase in reflex response always followed an injection if it were large enough. Forty-four injections of varying amounts were given to 17 dogs. Two injections caused an increase before hyperpnea had begun, 8 increases began with hyperpnea, and ended with it, 10 began during hyperpnea and lasted into apnea, 12 began during hyperpnea and lasted through apnea and 4 began after hyperpnea and lasted through apnea. Eighteen injections were followed by no apnea. Fatal injections always caused a pronounced increase in reflex response.

Since hyperpnea leads to increased elimination of carbon dioxide it was essential to determine whether the increased reflex response was of this origin. This was accomplished by repeating the above experiments during constant artificial ventilation after the establishment of pneumothorax. The results of 21 injections on 10 animals were comparable to those obtained in the intact animal with ventilation under normal control.

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II. Low Alveolar Oxygen and Response of Anterior Tibialis Muscle to Reflex and Motor Nerve Stimulation.

HAROLD H. GAY. (Introduced by Robert Gesell.)

From the Department of Physiology, University of Michigan.

Glazer and Winkler[1, 2] have shown that the administration of low percentages of oxygen in nitrogen under normal conditions of respiration caused an associated increase in reflex response and respiratory movements. The augmented reflex response might be due to the blowing off of carbon dioxide by the augmented respiration. To investigate this possibility a group of 6 experiments were done in which low percentages of oxygen (4% to 7%) in nitrogen were

[1] Glazer, W., *Am. J. Physiol.*, 1929, lxxxviii, 562.

[2] Winkler, A., *Am. J. Physiol.*, in press.

administered under conditions of constant ventilation as well as normal variable ventilation.

It was found that low alveolar oxygen during constant pulmonary ventilation augmented the reflex response of the anterior tibialis muscle similar to that during normally augmented ventilation.

To determine the site of action of low oxygen pressure, both anterior tibialis muscles were isolated and stimulated, one reflexly as above and the other through its severed motor nerve. In the 4 experiments that were done it was found that the response to the direct stimulation of the motor nerve was the same as the reflex response. Whether the change in reflex response is due entirely to the action of low oxygen pressure on the motor end of the reflex arc remains to be determined.

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III. Varying Degrees of Ventilation and Response of Anterior Tibialis Muscle to Reflex and Motor Nerve Stimulation.

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Seventeen experiments were performed in which carbon dioxide mixtures were administered by artificial ventilation. In 11 of these the carbon dioxide percentage was successively increased from 5 to 25%. In 15, ventilation with pure oxygen was varied from under ventilation to over ventilation, thus permitting large fluctuations in alveolar carbon dioxide pressure without undue lowering of alveolar oxygen pressure.

Carbon dioxide reduced the reflex response of the anterior tibialis muscle in all but one experiment. It reduced nerve-muscle response in all but 5 of the 17 experiments. In some experiments even high carbon dioxide mixtures did not depress the motor nerve response, whereas the reflex response was decreased.

Under ventilation in oxygen reduced reflexes in all but 2 experiments. It reduced nerve-muscle responses in all but 8 of the 17 experiments. Pure oxygen never failed to restore reflexes after carbon dioxide in pure oxygen had reduced them. Over ventilation always restored reflexes after they had been depressed by under ventilation in pure oxygen. Pure oxygen occasionally caused hypertonus, particularly in the muscle with the nerve intact, and on 3 occasions in the other muscle as well. Where pure oxygen was administered after room air the reflex response frequently fell off somewhat.