Inhibition of the Masseteric Reflex by Vagal Afferents

We recently reported that EEG synchronization and desynchronization could be induced by the excitation of specific afferent fiber groups within the cervical vagus nerve[1,2]. Since EEG patterns are often correlated with ongoing behavior, it was of interest to determine the influence which these vagal afferents might exert upon a fundamental component of behavior, the somatic reflex. In order to minimize the influence of systemic factors induced by vagal stimulation, it was necessary to utilize the encéphale isolé preparation and to monitor a brain stem reflex. Previous studies have indicated that an interaction exists between the vagus nerve and the trigeminal system[3,4]. The masseteric monosynaptic reflex was therefore employed as the somatic test response[5,6].

In these experiments 20 adult cats were used. Operative procedures were performed while the animals were under ether and sodium methohexital (Brevital) anesthesia. The masseteric nerve was exposed peripherally, the spinal cord sectioned at the C-1 level, and both vagal and sympathetic trunks transected bilaterally in the cervical region. The central end of the right vagus nerve was separated from the sympathetic trunk and placed on a stimulating electrode. A recording electrode was placed on the vagus nerve between the point of stimulation and the brain. Another recording electrode was placed along the exposed right masseter nerve. The masseteric reflex and the vagal neurogram were monitored oscilloscopically. Respiration was artificially administered and the experiment continued in immobilized preparations (Flaxedil). All wound edges were repeatedly infiltrated with a local anesthetic (Xylocaine).

The masseteric reflex was evoked by delivering an electrical pulse to the right mesencephalic nucleus of the fifth nerve at the rate of 1/sec. The amplitude of the reflex was analyzed in conjunction with stimulation of the cut central end of the right cervical vagus nerve. 2 patterns of vagal stimulation were employed. The first pattern was one in which a single vagal pulse or a short train of vagal pulses (3 pulses, 200/sec) preceded the elicitation of the reflex. The second pattern was the repetitive excitation of the vagus nerve at frequencies ranging from 5–200 cps. The vagus was stimulated with pulses which were suprathreshold for all fibre groups[2].

When a single vagal pulse preceded the masseteric reflex, complete inhibition of the reflex was observed

[1] M. H. CHASE, M. B. STERMAN and C. D. CLEMENTE, Expl Neurol. *16*, 36 (1966).
[2] M. H. CHASE, Y. NAKAMURA, C. D. CLEMENTE and M. B. STERMAN, Brain Res. *5*, 236 (1967).
[3] J. D. GREEN, J. DE GROOT and J. SUTIN, Am. J. Physiol. *189*, 384 (1957).
[4] I. STOICA, H. M. GUILLEUX and P. DELL, J. Physiol., Paris *57*, 703 (1965).
[5] J. SZENTAGOTHAI, J. Neurophysiol *11*, 445 (1948).
[6] A. HUGELIN and M. BONVALLET, J. Physiol., Paris *49*, 210 (1957).

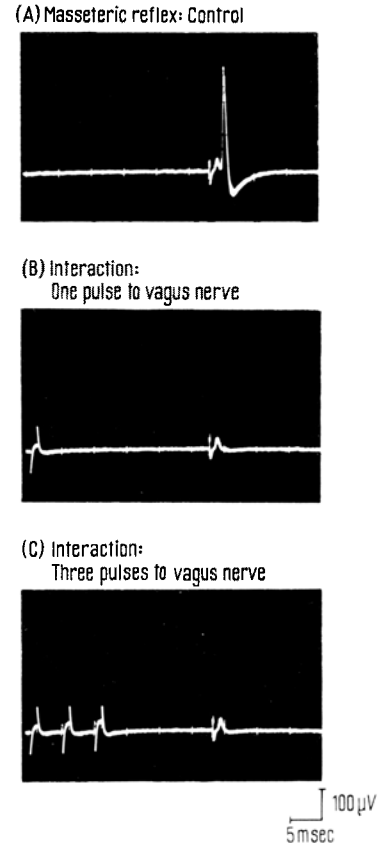

Fig. 1. Inhibition of the masseteric monosynaptic reflex by afferent vagal stimulation. In A, the control reflex was elicited by stimulation of the mesencephalic nucleus of the fifth nerve. The reflex is completely abolished when preceded by either single (B) or multiple (C) vagal pulses. The antidromic potential persists unchanged. (3 superimposed traces – Mes. Nuc. fifth: 4 V, 0.2 msec, 1/sec – Vagus: 10 V, 1 msec.)

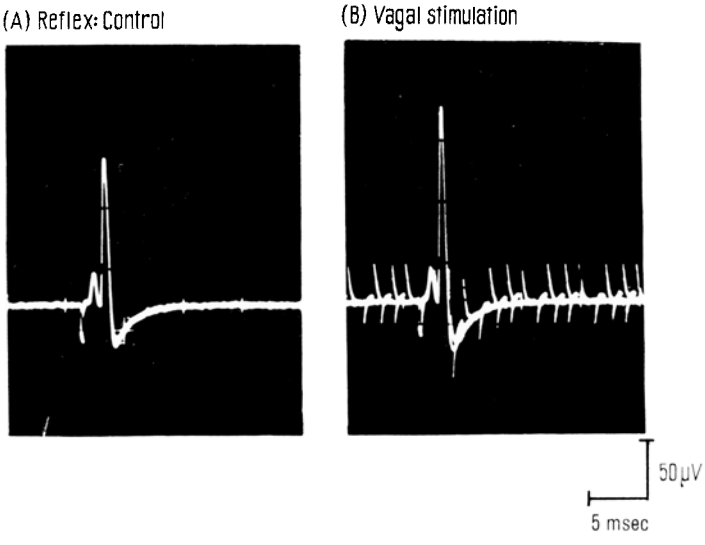

Fig. 2. Slight increase in the amplitude of the masseteric reflex is depicted during repetitive afferent vagal stimulation. (3 superimposed traces – Mes. Nuc. fifth: 6 V, 0.2 msec, 1/sec – Vagus: 10 V, 1 msec, 200 cps.)

(Figure 1 A and B). This inhibition was predominant from 10–30 msec after the first vagal pulse. With the stimulation pattern in which a short train of vagal pulses preceded reflex elicitation, inhibition of the masseteric reflex also resulted (Figure 1C). The time course of this inhibition was similar to that obtained for a single vagal pulse.

We observed no change in the amplitude of the masseteric reflex in conjunction with repetitive vagal stimulation in a few of our animals. In most experiments a slight facilitation was noted (Figure 2). This facilitation was augmented as the frequency of vagal stimulation was increased.

It was concluded that afferent vagal stimulation is capable of inhibiting the masseteric monosynaptic reflex. Although a few reports have indicated that visceral afferent activity may modify somatic reflexes, the participation of systemic factors in this interaction have thus far not been eliminated[4,7,8]. The short latency of the masseteric inhibition indicates that systemic humoral factors are not involved and that direct neural influences acting either through brain stem or forebrain structures may be responsible for its genesis. It is interesting to consider the significance of this visceral information received by the brain. Perhaps these afferent impulses which bring about inhibition of the masseteric reflex are important in the regulation of somatic musculature of the head and neck responsible for mastication and deglutition. It is probable that vagal afferent activity may affect a variety of behaviors while simultaneously modifying cortical and subcortical EEG patterns of activity[9,10].

Zusammenfassung. Es wurde an der kurarisierten Katze bei «encéphyle isolé» der Einfluss sensibler Vagusfasern auf den monosynaptischen Reflex des Musculus massetericus untersucht (für alle Fasergruppen des Vagus überschwellige Reize). Vollständige Hemmung des Masseter-Reflexes wurde erzielt bei Reizung des proximalen Endes des Halsvagus mit Einzelstromstössen oder kurzen Serien.

M. H. Chase and Y. Nakamura

Department of Anatomy, School of Medicine, University of California, Los Angeles (California 90024, USA),
7 May 1968.

7 C. A. Johnson and A. B. Luckhardt, Am. J. Physiol. *83*, 642 (1927).

8 A. Schweitzer and S. Wright, J. Physiol. *88*, 459 (1937).

9 This research was supported by a grant from the U.S.P.H.S. (No. MH-10083). This work has received bibliographic aid from the UCLA Brain Information Service which is a part of the National Information Network of the NINDB and supported under Contract No. PH-43-66-59.

10 Dr. Nakamura is a Research Fellow from the Department of Neurophysiology, Institute of Brain Research, University of Tokyo (Japan).

Secretory Potentials, Potassium Transport and Secretion in the Cat Submandibular Gland During Perfusion with Sulphate Locke's Solution

Lundberg[1–4] proposed that the type I secretory potential, i.e. the hyperpolarization of the contraluminal acinar cell membrane, occurring during stimulation of the gland, was caused by an active inward transport of chloride ions forming the acinar primary secretion. Another hypothesis has been proposed by Yoshimura and Imai[5], who suggested that the secretory potential was due to a passive efflux of potassium from the acinar cells to the extracellular fluid. The present study was undertaken to test these 2 hypotheses. The results which were obtained are in opposition to both hypotheses.

Methods. Cats (1.5–5 kg) anaesthetized with chloralose (70–80 mg/kg i.p.) were used. The preparation of the submandibular gland for artificial perfusion and the measurement of transmembrane potentials have been described previously[6]. In some experiments the potassium concentration in the venous outflow from the gland was measured together with the perfusion fluid flow. The flame photometric method used has been described previously[7]. The glands were stimulated with close intraarterial injections of 5–10 μg acetylcholine (ACh). Each time a secretory potential had been recorded after ACh injection the number of drops of saliva secreted was counted. In all experiments, both a control and a sulphate Locke's solution were used. The control Locke's solution contained (mM): 140 NaCl, 4.0 KCl, 2.4 Na_2HPO_4, 0.6 NaH_2PO_4, 1.5 $Ca(NO_3)_2$, 1.0 $MgCl_2$, 5.5 glucose. The sulphate Locke's solution contained (mM): 70 Na_2SO_4, 2.0 K_2SO_4, 2.4 Na_2HPO_4, 0.6 NaH_2PO_4, 6.0 $Ca(NO_3)_2$, 1.0 $MgSO_4$, 59 sucrose, 5.5 glucose. The perfusion fluids were equilibrated with pure oxygen.

Results. In Figure 1 it is seen that after the perfusion fluid had been changed from control to sulphate Locke's solution the secretory response to ACh was rapidly abolished while normal secretory potentials could still be recorded. In Figure 2 examples are seen of secretory potentials recorded during perfusion with control and sulphate Locke's solutions, respectively. In Figure 3 it is seen that the loss of potassium from the gland to the perfusion fluid, which normally follows after an injection of ACh, was severely reduced as was the secretion during perfusion with sulphate Locke's solution. There seemed to be full restitution of both the potassium loss and the secretion evoked by ACh after returning to control Locke's solution.

Discussion. The fact that the secretory potential is normal during perfusion with a solution which is chloride-

1 A. Lundberg, Acta physiol. scand. *35*, 1 (1955).

2 A. Lundberg, Acta physiol. scand. *40*, 21 (1957).

3 A. Lundberg, Acta physiol. scand. *40*, 35 (1957).

4 A. Lundberg, Acta physiol. scand. *40*, 101 (1957).

5 H. Yoshimura and Y. Imai, Jap. J. Physiol. *17*, 280 (1967).

6 O. H. Petersen and J. H. Poulsen, Acta physiol. scand. *70*, 293 (1967).

7 O. H. Petersen and J. H. Poulsen, Acta physiol. scand. *70*, 158 (1967).