(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 masseter 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),
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[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-

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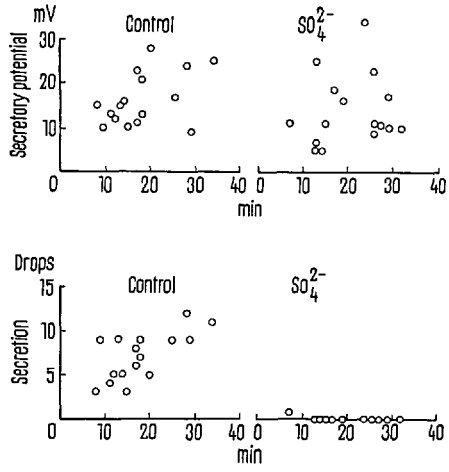

Fig. 1. Size of secretory potentials and secretion as a function of time after start of perfusion with control and sulphate Locke's solutions, respectively. For each secretory potential the corresponding secretion evoked by the same injection of ACh is shown. The data have been pooled from 6 experiments on 6 cats.

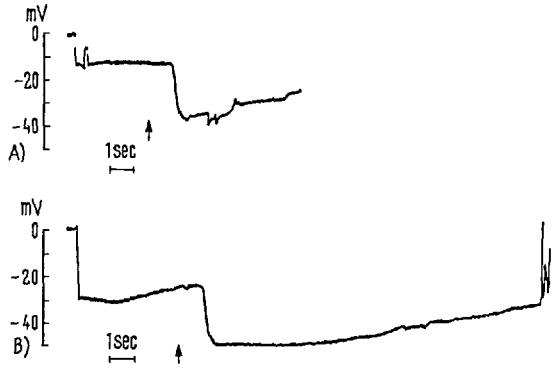

Fig. 2. (A) Resting and secretory potential during perfusion with control Locke's solution. The secretory response to the injection of ACh (↑) was 12 drops of saliva. (B) Resting and secretory potential from the same experiment after 13 min of sulphate perfusion. The secretory response to the injection of ACh which resulted in the secretory potential was zero.

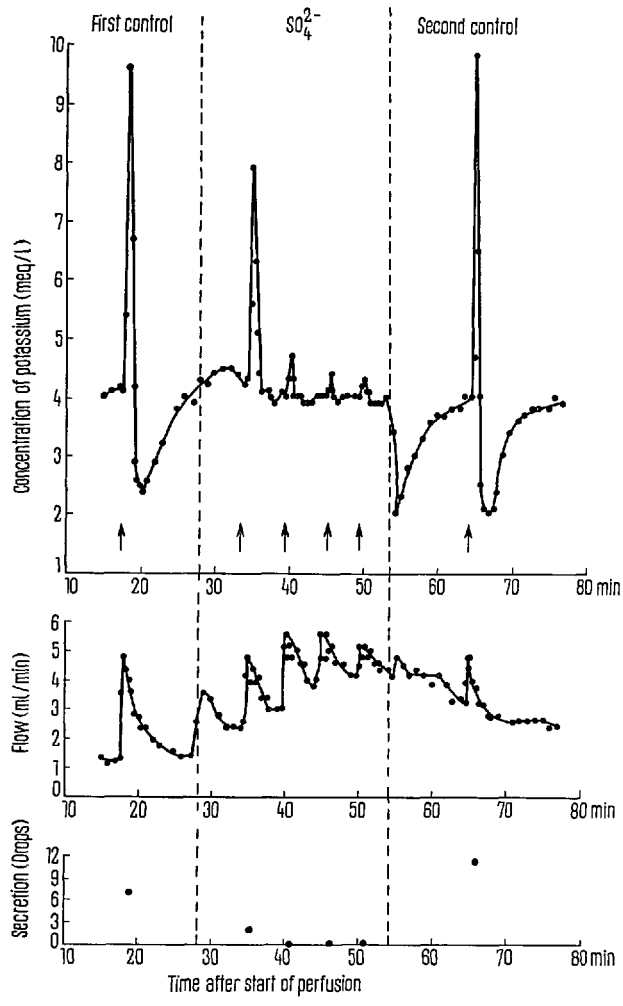

Fig. 3. Concentration of potassium in the venous perfusion fluid, glandular perfusion fluid flow and secretion during perfusion with control and sulphate Locke's solutions. The gland was stimulated by injections of ACh (10 μg) (↑).

and bicarbonate-free and in which the only anion represented in large amounts is the bulky divalent sulphate ion, seems to be incompatible with LUNDBERG's[1-4] hypothesis. Earlier, YOSHIMURA and IMAI[5] have shown a single example of a relatively normal secretory potential recorded during perfusion with sulphate Locke's solution. It is at least evident that it cannot be the ion transport forming the secretory potential that also forms the primary secretion due to the fact that the secretion is abolished during perfusion with sulphate Locke's solution.

The finding that the loss of potassium from the gland to the perfusion fluid after stimulation, is severely reduced during perfusion with sulphate Locke's solution, while the secretory potential is normal, seems to be in opposition to the hypothesis of YOSHIMURA and IMAI.

As it seems very likely that the secretory potential is due to an active ion transport[3,8,9], and as the present results make it unlikely that this transport is an anion transport, it may be suggested that the secretory potential is due to an active outward transport of sodium ions. It is probable that the function of the secretory potential is to initiate the ionic transport mechanisms forming the acinar primary secretion.

Zusammenfassung. Es wird gezeigt, dass während der Perfusion der Submandibularis-Drüse der Katze mit chloridfreier Sulfatlösung die sekretorischen Potentialdifferenzen der Azinuszellmembran unverändert bleiben und dass inzwischen die Sekretion und der Kaliumtransport von der Drüse zur Perfusionsflüssigkeit völlig aufhört.

O. H. PETERSEN and J. H. POULSEN

Institute of Medical Physiology C, University of Copenhagen (Denmark), 14 May 1968.

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