CLINICO-PHYSIOLOGICAL STUDY OF INNERVATION OF FACIAL SWEAT GLANDS

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It is believed by the majority of physiologists that sweat glands receive their innervation exclusively from the sympathetic system, the fibers of which are cholinergic.

Physiologists and pharmacologists are agreed that the locus of action of pilocarpine in stimulating sweat production is the gland itself, i. e., its receptive substance. This view is based on the experiments of Langley and Anderson [11], who showed that section of the sciatic nerve of cats did not abolish secretion of sweat following introduction of pilocarpine into the affected limb; the hypohidrosis found was ascribed by these authors to vasomotor paralysis.

Clinical observations are at variance with the results of Langley and Anderson. After section with subsequent degeneration of a spinal nerve it has been found that sweat production completely disappears in the zone of anesthesia [9, 10, and others]; a similar result follows extracranial section of branches of the trigeminal nerve [7, 12, and others]. Numerous observations have shown that the sweat glands of skin grafts do not react to pilocarpine or to any other stimulant until they have acquired a sensory nerve supply [5 and others]. Highly conflicting reports are to be found in the literature regarding the effect of pilocarpine on sweat secretion after section of the superior cervical sympathetic ganglion; in general, it appears that pilocarpine continues to stimulate sweating on the operated side for a considerable time after the operation, although to a diminished extent. It is in connection with this observation that most clinicians differ from physiologists and pharmacologists in believing that pilocarpine acts on nerve endings, and not on the glands themselves, and that the sweat glands are supplied with secretomotor nerves from both the sympathetic and the parasympathetic systems [3, 4, and others]. With regard to the facial sweat glands this view is strengthened by the following considerations: (a) it is often possible to observe sweating on the side on which the superior cervical sympathetic ganglion was resected, in response to taste stimuli, (b) in many cases it has been observed that extirpation of the cervical sympathetic ganglion does not affect secretion of sweat in the temporal region, in the so-called auriculotemporal syndrome.

The view has been expressed that two sudormotor neurons are to be found in the bulbopontine parasympathetic centers. The axons of one of them reach the sweat glands of the face via the facial nerve, and of the other via the trigeminal nerve [9]. This hypothesis has not, however, been confirmed by further study of the problem [7], which remains unresolved, as is shown by the mutually contradictory conclusions reached during the past 10-20 years [1, 12, 13, and others].

We have studied sudormotor function in three groups of patients. One group consisted of four patients suffering from paralytic lagophthalmos, the site of the lesion to the facial nerve being proximal to the geniculate ganglion in one of them, and distal in the other three. Minor's test showed that sweating of the face was unsymmetrical in all cases. One patient (facial nerve palsy following a radical operation on the ear) exhibited hypohidrosis on the affected side, in contrast to slight hyperhidrosis in the other three patients. These effects were the same in each patient, irrespective of whether sweating was provoked by warmth or by pilocarpine. *

*Thermal stimulation was effected by immersing the patient to the waist in water at 39°. Pilocarpine (0.5-1 ml of 1% solution) was injected subcutaneously into the shoulder. Gustatory sweating was provoked by application of slightly diluted vinegar or citric acid solution to the tongue.

Since thermoregulatory sweating is effected through the agency of the sympathetic system only, it is logical to conclude that these anomalies were due to secondary factors, such as repercussive effects, and also possibly to local vascular disturbances caused by paralysis of the facial muscles. The second group consisted of two patients in whom the sensory branch of the trigeminal nerve had been cut, for treatment of trigeminal neuralgia. In one of them secretion of sweat in response to warmth and to pilocarpine was approximately equal on both sides of the face, while the other exhibited slight hyperhidrosis on the side on which the nerve had been cut.

It is evident that these data do not afford confirmation of the hypothesis that the trigeminal and facial nerves contain parasympathetic sudormotor fibers.

The third group consisted of five patients who had been operated for removal of the superior cervical ganglion, one because of lagophthalmos, and the other three because of optic nerve atrophy. Tests of sudorific function were performed on all these patients, by observation of secretion of sweat in response to warmth, pilocarpine, and gustatory stimuli. The patients responded to warmth, anhidrosis being found on the affected side. One of them responded to pilocarpine earlier and more profusely on the operated than on the other side (operation performed 3 years earlier); the remaining patients exhibited hypohidrosis on the operated side. This was slight in one case (8 months after operation), more pronounced in another (6 weeks after operation), and bordered on anhidrosis in the remaining two (2 1/2 and 3 years after operation). The response to taste stimuli was absent on the operated side in the latter two cases, but was positive in the other ones.

We present two case reports:

Patient K., female aged 40, reported to the Eye Clinic with lagophthalmos, complicated by loss of lacrymatory function on the affected side. The case history revealed that she had sustained a blow on the head during a fall 8 months back, since then she had exhibited total loss of function of the right facial and auditory nerves. Superior cervical ganglionectomy was performed for the relief of lagophthalmos. Six weeks later Minor's test showed the following: (i) anhidrosis on the operated side, in response to warmth, (ii) in response to pilocarpine, secretion of sweat on the unoperated side, in the frontal and temporal areas and in the nasolabial fold, upper lip, and chin, and on the operated side scanty secretion in the frontal and temporal areas and the nasolabial fold only, (iii) in response to gustatory stimuli scattered groups of stained points, mostly in the frontal and parietal areas of the operated side.

Patient B., female aged 23, had undergone left cervical ganglionectomy. Minor's test, performed 10 weeks later, showed the following: (i) anhidrosis on the operated side, in response to warmth, (ii) sweating on the intact side, in response to pilocarpine, in the temporal area, bridge of the nose, nasolabial fold, and under the chin, whereas on the operated side only two groups of stained points were seen, along the upper border of the eyebrow, (iii) absence of response to gustatory stimuli.

If the view held by most clinicians that the facial sweat glands are innervated by both sections of the autonomic nervous system is to be accepted, how are we to explain the observation that in some cases the response to pilocarpine after superior cervical ganglionectomy is not only a relative hypohidrosis but also a drastic reduction of the area in which sweating is observed on the operated side? How are we to explain the fact that in some cases the response to pilocarpine is virtually absent on the operated side, while in other cases it is of equal or even greater intensity than on the unoperated side? It is known, moreover, that thermoregulatory sweating is effected through a central reflex, and that it is abolished by interrupting the sympathetic paths of the reflex arc; this is what we found in our patients. What is the mechanism of sweating in response to gustatory stimuli? If, as many clinicians suppose, this is essentially a bulbar reflex, which nerve is the efferent limb of this reflex arc? The cervical sympathetic nerve is excluded, nor can it be the facial nerve, as is shown by our present observations, or the trigeminal nerve, since section of its sensory branch not only does not inhibit response to pilocarpine, but, moreover, when combined with superior cervical sympathectomy it does not prevent sweating in response to taste stimuli [Wilson, 13].

Research workers have taken for granted that all the sympathetic effector neurones responsible for innervation of the head are concentrated in the superior cervical ganglion. It appears, however, from the work of B. I. Lavrentyev, published in 1939, that sympathetic cells sending out axons of the second order are most often to be found outside the limits of the ganglia of the cervical and thoracic sympathetic trunks, that their distribution in different individuals of a given species is highly variable, and that some nerve trunks are liberally supplied with such cells. It has been shown in experiments on cats by B. I. Lavrentyev's co-workers that on the

side on which the superior cervical ganglion was removed the terminal aborizations in the nictitation membrane [8] and in the tunica media of the arteries of the buccal mucosa [2] do not all undergo degeneration in most animals (giving a "mosaic" picture), and in a minority of animals they are unaffected by the operation. If, however, sympathetic fibers are cut cranial to the superior cervical ganglion, which is also removed, full degeneration of the terminations is observed in all animals.

The response of the sweat glands to pilocarpine, following superior cervical ganglionectomy, is, as we have seen, also erratic. It is most often weakened, sometimes equal in strength to that of the unoperated side, and sometimes virtually absent. A survey of the literature shows that following extirpation of the superior cervical ganglion secretion of sweat in the auriculotemporal syndrome may be diminished, abolished, or unaffected. The variable result of the operation, and the variations in response to pilocarpine, depend on individual differences in distribution of sympathetic neurons distal to the superior cervical ganglion.

As has been pointed out above, the response to pilocarpine is sometimes stronger on the operated side. This effect might be explained as being due to a combination of two factors, viz., exclusion of central inhibitory influences, and the wide distribution of sympathetic effector neurons distal to the extirpated ganglion.

Gustatory stimulation of sweat secretion after superior cervical ganglionectomy appears to be effected through the medium of a local reflex. Impulses arising from stimulation of taste receptors are transmitted to surviving sympathetic neurons through anastomoses of the taste nerves with the internal carotid plexus and the Gasserian ganglion. The manifestation of the reflex is facilitated by the connections of the sympathetic neurons with autonomic nerve centers.

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