BRITISH MEDICAL JOURNAL

LONDON SATURDAY APRIL 19 1952

THE FIFTH AND SEVENTH CRANIAL NERVES IN RELATION TO THE NERVOUS MECHANISM OF TASTE SENSATION*

A NEW APPROACH

BY

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Since the days of Magendie and Claude Bernard, over a hundred years ago, many and varied opinions have been held or suggested concerning the nerves supplying the sense of taste to the front and back of the tongue. Magendie, in 1822, declared that after intracranial section of the trigeminal nerve in dogs taste is completely lost, even for sour and bitter. Bernard, in 1831, following Bellingeri in 1818, showed that the chorda tympani running with the facial nerve conducted taste from the tongue. Yet apparently he agreed with Magendie's statement regarding the gustatory function of the fifth nerve, for he never denied it or referred to it in his writings. Lussana, in 1869, described two cases: one of destruction of the fifth nerve, with complete anaesthesia of one side of the face and tongue, in which no impairment of taste was found; in the other case the facial nerve had been divided accidentally by a charlatan, resulting in complete loss of taste on the anterior part of half the tongue, which Lussana found on examining the man two years later. Many cases have since been published of loss of taste on the tongue associated with gross disease involving the fifth nerve at the base of the skull.

Whether the fifth nerve or the chorda tympani was the conducting nerve of taste sensations, the ultimate peripheral receptors of gustatory sensation have for many years been associated with taste buds mainly on the front and back of the tongue, those on the back of the tongue being innervated by the glossopharyngeal nerve, and those on the front, especially the sides and the tip, by the chorda tympani, the dorsum being relatively insensitive to taste. The four modalities of taste perception—sweet, bitter, salt, and sour—have been recognized by physiologists as distinct, and mainly represented in certain areas of the tongue, sweet and salt especially near the tip, sour laterally, and bitter especially at the back. More recently these four modalities are believed to depend on different receptors, probably arranged zonally (Patton and Ruch, 1949), and supplied by chorda tympani or glossopharyngeal fibres respectively. In favour of this view is the known effect of certain drugs applied to the tongue in temporarily abolishing one or other of the four primary taste sensations. Thus, the chewing of leaves of *Gymnema sylvestre* abolishes taste

for sweet and bitter, and phenylthiocarbamide, which tastes bitter to most people, is tasteless in about 20% of those tested. According to Wiggers (1949) taste blindness, in which one or more of the primary sensations may be weak or absent, is not uncommon, and is hereditary as a Mendelian recessive. Sodium benzoate, a common food preservative, is tasteless for the majority, but may be called sweet or bitter by 25% of persons tested.

The 130-year-old controversy whether the fifth nerve or the chorda tympani is the central conducting path for taste from the tongue has now for many years been considered settled in favour of the chorda tympani. The object of this paper is to bring fresh proof of the importance of the fifth nerve in taste perception, not as the central continuing path from the tongue, but as furnishing in the large majority of people a necessary blending of fifth-nerve sensibility with the primary gustatory function of the chorda tympani and its receptors. The Diagram overleaf shows the course of the taste fibres and of the intrapontine connexions of the fifth nerve. In a small minority of persons the fifth-nerve sensibility is of very slight and temporary importance; in a large number its importance is great, taste being lost for weeks, months, or years after fifth-nerve lesions; and in a small minority, at the other end of the scale, this loss appears to be permanent.

Further, I wish to draw attention to the gustatory function of the great superficial petrosal (Vidian) nerve in supplying the palate, comparable to that of the chorda tympani on the tongue, in less degree, and to point out that palatal loss of taste usually occurs in lesions of the maxillary nerve or of the Gasserian ganglion which cause palatal anaesthesia to touch and prick, and, moreover, that geniculate ganglion or pars intermedia lesions, with or without accompanying facial palsy, produce palatal loss of taste in these cases without palatal anaesthesia.

Many writers have produced statistics intended to prove that taste is not lost in fifth-nerve lesions. Lewis and Dandy (1930), as the result of tests after fifth-nerve-root section for trigeminal tic, concluded that the chorda tympani is the only conducting path for taste sensations on the front of the tongue, and that the fifth nerve has no connexion with taste. Schwartz and Weddell (1938),

*A Clinical Lecture delivered at St. Mary's Hospital, November 30, 1951.

dealing with similar root-section operations at St. Louis, mostly by Sachs, quote four cases with no taste loss examined a few days after operation, and others in which loss of taste was stated to be due to damage to the Vidian nerve, with or without facial palsy. They quote loss of taste found on the tongue five days after root section, with deliberate section of the Vidian nerve but without facial palsy, as evidence of conduction of taste by the Vidian nerve from the tongue, and not by the chorda tympani. They completely ignore the tactile and pain analgesia of the tongue as a factor in the loss of taste, and conclude that in certain individuals the Vidian nerve forms part of the pathway for taste from the front of the tongue, though they cannot explain how these taste sensations reach the Vidian nerve. Carmichael and Woollard (1933) examined 17 cases of total fifth-nerve anaesthesia after Gasserian alcohol injection. They found that 10 cases of the 17 had complete ageusia on the anterior anaesthetic half of the tongue, but were unable to give any explanation of the variations found.

The Views of Gowers

Gowers held the strong belief that taste sensation was ultimately conveyed to the pons by the fifth nerve, though he agreed that the peripheral pathway was via the fibres of the chorda tympani, lingual, and facial nerves to the geniculate ganglion. He stated definitely that no taste fibres accompanied the facial nerve within the skull, but that they must leave the geniculate ganglion by the great superficial petrosal (Vidian), passing to Meckel's ganglion and thence by the second division of the fifth nerve to the Gasserian ganglion and pons. Gowers was a most careful clinical observer, and, although his statements of loss of taste in numerous cases of fifth-nerve lesions date back 60 years or more, that is no reason to disbelieve them *in toto*. He writes in 1893 (p. 226): "In almost every case I have seen

in which there were symptoms of disease of the root of the fifth nerve I have found taste abolished on the corresponding side, not only on the front of the tongue but also on the back of the tongue, and on the arches of the palate. On the other hand, cases occur in which there are symptoms of disease of the fifth nerve without loss of taste." These variations, to my knowledge, he did not attempt to explain.

When I was Gowers's house-physician at the National Hospital in 1896 I had the opportunity to examine a man he

sent into the hospital for one of his Wednesday lectures, a groom, aged 46, who ten months previously had suffered with pain for six weeks in all his teeth on the left side, followed by numbness of the left side of the face and mouth. He had right-sided cerebral facial weakness, complete left fifth-nerve anaesthesia and motor palsy, with total loss of taste on the anterior portion of the tongue on the left side, and left frontal herpes. The lesion was no doubt a gumma on the roots of the left fifth nerve, pressing on the side of the pons. He soon recovered completely after a course of mercury and iodide of potassium. This case I published, with photograph (Harris, 1926, p. 243). I have also found notes of another case of Dr. Gowers's.

Mrs. Clara O'Leary, aged 50, was admitted to the National Hospital on August 13, 1896. This is obviously the case that Gowers referred to in a footnote to page 226 of his

textbook, which he had seen on July 5, 1880, and which he published in the following year (Gowers, 1881). She was then 33, and woke up during the night of April 14 with a burning sensation in the right side of the face, and found at once she had no feeling there. On the 17th her doctor found complete loss of sensation in all parts supplied by the right fifth nerve. Nearly three months later Gowers observed that the muscles of mastication were very weak and wasted, and that taste was absolutely lost to salt, sugar, and quinine on the right half of the tongue, front and back, and on the anterior palatine arch, and in the region of the tonsil, where common sensibility was normal. This absence

Diagram of the course of the taste fibres (thick lines) and of the intrapontine connexions of the fifth nerve. (The lower half is represented in profile, and the upper in plan. For convenience the gustatory nucleus and substantia gelatinosa are represented in the pons instead of in the medulla.)

- T.f.a.: Trigeminal fillet (tactile and pressure).
- T.f.b.: Trigeminal fillet (pain and temperature).
- G.f.: Gustatory fillet.
- III: Third cranial nerve.
- VI: Sixth nerve.
- V.mes.n.: Fifth mesencephalic nucleus.
- V.m.n.: Fifth motor nucleus.
- G.n.: Gustatory nucleus.
- F.S.: Fasciculus solitarius.
- V.s.n.: Fifth sensory nucleus (tactile and pressure).
- S.G.: Substantia gelatinosa (nucleus of fifth spinal root).
- VII: Roots of the seventh cranial nerve (facial).
- V.sp.: Fifth nerve, spinal root.
- V.s.: Fifth nerve, main sensory root.
- V.m.: Fifth nerve, motor root.
- V.1.: Ophthalmic branch, fifth nerve.
- V.2.: Maxillary nerve.
- V.3.: Mandibular nerve.
- N.J.: Nerve of Jacobson.
- P.I.W.: Pars intermedia wrisbergi.

The chorda tympani and great superficial petrosal (Vidian) nerves carrying taste impressions from the front of the tongue and palate respectively are shown uniting in the pars intermedia wrisbergi. (See Harris, 1926, p. 252.) The glossopharyngeal carries taste impressions from the back of the tongue to the fasciculus solitarius.

of taste on front and back of tongue Gowers (1897) states was still unaltered seven years later. Taste was acute on the left corresponding areas. There was some diplopia due to the third and sixth nerves. His comment on the case was that this patient had but one nerve of taste, the fifth.

When I examined her, 16 years later, she stated that she had never recovered any sensation on the face, and I found that she still had complete anaesthesia to touch and prick

over the whole area of the right fifth nerve to the mid-line, including the hard palate, and complete atrophy of all the muscles of mastication on the right side. Taste was also completely lost on the front two-thirds of the tongue on the right side, but she could recognize sweet, salt, and bitter on the right back of the tongue, though much less than on the left side. Taste had evidently partly returned on the right back of the tongue in the intervening 16 years, which corresponds entirely

with the partial recovery of taste I have often observed on the front half of the tongue after Gasserian ganglion injections. She had also developed right sixth-nerve palsy, which is shown in the photograph here reproduced.

The explanation of the partial return of taste on the back of the tongue, while taste was still completely absent on the front portion, would appear to be that this area was shared between the glossopharyngeal and fifth-nerve common sensibility, and not, as Gowers thought at first, that the glossopharyngeal had no taste supply in her case. Nearly a year afterwards, in May, 1897, her right facial nerve became completely paralysed. Whatever the pathological diagnosis may be here, the special interest of the case is the total loss of taste on the right front and back of the tongue, associated with total fifth-nerve paralysis, vouched for 16 years before by Gowers, and again reported on after careful revision in 1896.

Loss of Taste in Various Fifth-nerve Lesions

Loss of taste on the front half of the tongue I have also seen in two of Victor Horsley's gasserectomies, and in one operated on in 1929 by Mr. Cairns, in which I found complete loss of taste on the front half of the tongue 14 days after the operation. In a woman, aged 57, under my care in October, 1942, for acoustic neuroma, which was successfully operated on a month later, numbness of the right side of the face, with pain, had been present for ten months, with total trigeminal anaesthesia and complete loss of taste on that side of the front of the tongue. Cushing found temporary loss of taste on the anaesthetic half of the tongue in a few of his Gasserian cases, but discounted its importance by explaining it as due to descending degeneration of the lingual nerve causing pressure on the chorda fibres which run with it. This explanation must be fallacious, for I have dozens of times demonstrated total loss of taste on half of the tongue immediately after alcohol injection of the third division of the fifth nerve, or after Gasserian injections, usually done under a local analgesic only.

In 1926 I published notes of 26 cases of complete loss of taste on the front half of the tongue, out of 80 observed cases of such loss after alcohol injection (Harris, 1926, p. 265), and in 1940 I referred to 167 cases of loss of taste I had found after similar injections. This number has now increased to well over 200, and among these are several in which I have observed that the loss still existed many months and even years after injection.

A woman aged 40, whom I saw first in 1908 for neuralgia of six years' duration of the left second division, had an injection of the second division in front of the foramen rotundum in February, 1909; this gave complete relief, with dense anaesthesia for three years. In 1915 I injected

her left Gasserian ganglion, producing total left trigeminal anaesthesia, with complete loss of taste on the left front half of the tongue. This condition persisted unchanged for 24 years; when I saw her in 1939 I found that the anaesthesia and the loss of taste were still complete as before, though taste was acute on the right side.

A man aged 33, whom I saw first in November, 1914, for severe neuralgia of the right second and third divisions for the past three years, had a Gasserian injection which produced dense right trigeminal anaesthesia, with loss of all taste on the right front half of the tongue. Pain recurred after five years' relief, and reinjection was followed by a further eight years' relief. In 1929 I again injected the ganglion, this time with permanent success. I saw him on May 2, 1951, and found the trigeminal anaesthesia still complete, and the loss of taste as before, 22 years after the last injection.

Other records I have, of four cases showing loss of taste still existing, with anaesthesia of the front half of the tongue, 7, 11, 13, and 15 years after Gasserian injection, and of numerous other cases in which it was present for periods up to two and three years. I have also seen taste return to the front half of the tongue five years after Gasserian injection, though it was still absent after two years.

Bilateral trigeminal tic I have met with in 108 cases, roughly 4-5% of all cases. Usually a considerable interval occurs before the second side is affected—in one case 67 years, the initial attack on the right side occurring at the age of 12 and that on the left side at 79. On more than one occasion less than a week had elapsed before the attacks became bilateral, the severity being about equal in intensity. In others, one side, usually but not invariably the second to be involved, may not be affected severely enough to require injection or operation. Bilateral ganglion injection may be necessary, and I have several times seen total loss of taste on both sides of the front of the tongue, while taste remained acute over the glossopharyngeal area.

Loss of Taste on Back of Tongue, with Fifth-nerve Anaesthesia

Complete hemi-ageusia on the front and back of the tongue must, I think, be rarer than Gowers has suggested in fifth-nerve lesions. I have found this total loss in a few of my Gasserian injections, and have notes of eight such cases. One recent case illustrates this point well.

An intelligent woman secretary aged 43 was injected for trigeminal tic, though the history did not accord entirely with the classical type, as eating and talking did not start the paroxysms, nor had she trigger zones. Relief was only temporary for two weeks, though the anaesthesia seemed complete at first, and I repeated the injection on February 20, 1950, after which the anaesthesia remained apparently complete and taste was entirely lost on both the front and back of the tongue, palate, and pharynx on the left side. Not only was taste lost, but there was also complete anaesthesia over the left side of the pharynx and tonsillar region, as exploration with a hatpin proved conclusively. Gowers (1893, p. 216) also states: "It is certain that disease of the fifth nerve may cause anaesthesia in the root of the tongue and palate."

As the neuralgia again returned within two to three weeks of the second injection, I became suspicious of basal tumour pressing on the root of the fifth nerve, and I persuaded Sir Hugh Cairns and Mr. Pennybacker to investigate her at Oxford, which was done most carefully by encephalogram, C.S.F. examination, and vertebral angiogram, so that they felt confident there could be no basal or other cerebral tumour, which I had begun to suspect. They agreed that the trigeminal anaesthesia was complete, but as a last resort Mr. Pennybacker resected the left fifth sensory root in January, 1951, with complete success. I saw her and examined her again in July, when she told me that as soon as she began to come round from the anaesthetic she knew that her pain had disappeared, and there has been no recur-

rence since. The anaesthesia seemed on testing to be the same, and the taste loss on the front and back of the tongue and on the soft palate and pharynx on the left side still remained complete, six months after the operation. Some few nerve cells and fibres must have escaped alcohol destruction, though undetected on most careful sensory testing before the operation.

What is certain is that in a few cases of trigeminal anaesthesia loss of taste includes the back of the tongue as well as the front half, and when this is the case the loss of taste is more likely to be of long duration. I am convinced from my experience that taste is lost at first in nearly every case when anaesthesia of the front half of the tongue is complete, but that it is regained at varying intervals, maybe of hours only, or days, weeks, months, or years. An explanation of this fact is discussed below.

Palatal Anaesthesia, with Loss of Taste

In all the records of testing for taste that I have read, the palate is scarcely, if ever, mentioned. It is true that taste buds are much more sparsely found in the palate than on the front or back of the tongue, yet taste may be quite acute on the soft palate in many people. When testing, however, considerable care is required to avoid touching the tongue with the testing material. A broad electrically illuminated spatula should be used to keep the tongue down while a camel-hair brush soaked in syrup, salt solution, quassia, or quinine is used to paint the solution on the front half of the soft palate. The patient is not allowed to speak, but must be instructed beforehand to point to one of four cards on which are clearly written "sweet," "salt," "sour," "bitter." If no taste is perceived after the lapse of ten seconds, then the test is repeated on the other side. It may be that neither side is responsive enough to make the test valid; in that event, after the mouth has been washed out with warm water, the tests are repeated with a different substance. Some people are quite unable to distinguish different tastes on the palate, but may definitely taste something on the normal side when no sign of any taste may be perceived on the anaesthetic side. In others the perception and differentiation of taste may be quite acute and immediate on the normal side when it is completely absent or uncertain on the anaesthetic side.

When the second trigeminal division is involved with the third in a Gasserian injection taste is usually lost in the early days on both tongue and palate. Such palatal loss of taste cannot be concerned with the chorda tympani, but must be due either to second division anaesthesia or to loss of gustatory conduction by the great superficial petrosal nerve. I have injected the inner half only of the Gasserian ganglion in numerous cases of migrainous and ciliary neuralgias, and in some cases of trigeminal tic, when the pain is limited to the first or second division, or both. This is not particularly difficult to do, and complete escape of the third division from anaesthesia is a great advantage to the patient in eating and drinking; the absence of loss of taste on the tongue is also of benefit. I have seen similar loss of taste with palatal anaesthesia in cases in which I have injected the second division alone in front of the foramen rotundum, also in a case of temporo-sphenoidal gumma with complete anaesthesia of the upper jaw, cheek, and palate, as well as in a case of acute herpes zoster of the second trigeminal division only, with total anaesthesia of upper jaw and cheek. In these cases there can be no lesion of the great superficial petrosal nerve—which is, in my belief, the afferent gustatory nerve from the palate, via Meckel's ganglion to the geniculate ganglion and pars intermedia—for the lesion in the maxillary nerve is well away from this area.

Palatal Loss of Taste in Facial Paralysis

I will now return to facial paralysis, and the common accompanying loss of taste on the tongue, in those cases of facial damage between the geniculate ganglion and the exit of the chorda tympani in the middle ear to join the lingual. Peripheral lesions of the facial nerve at or below the stylo-mastoid foramen are not accompanied by any loss

of taste, unless inflammation spreads upwards along the nerve to include the chorda fibres in the aqueduct of Fallopius. On testing the palate in many cases of facial paralysis I have found that loss of taste is present on the palate as well as on the tongue. In these cases, also, the chorda tympani is not directly concerned with the palatal loss; but this is easily explained if the lesion causing the facial palsy is at or above the geniculate ganglion, where the chorda is joined by the great superficial petrosal to form the pars intermedia Wrisbergi. The lesion thus interrupts not only the chorda but the great superficial petrosal, which is almost certainly the supplier of taste fibres to the palate.

The course of the stimuli is therefore via the main palatal nerve, Meckel's ganglion, and Vidian nerve (great superficial petrosal) to the geniculate ganglion, and not in the reverse direction, as Gowers believed. A. F. Dixon (1897) wrote: "The great superficial petrosal is formed very early, and is developed as an outgrowth of the cells in the geniculate ganglion, proving that it conducts towards the geniculate ganglion. The same argument applies also to the chorda tympani, and to the nerve of Jacobson, which is an outgrowth of the petrous ganglion on the glossopharyngeal nerve, and therefore conducts towards the glossopharyngeal, and not to the trigeminus, as Gowers suggested."

Indeed, the presence or absence of loss of taste on the palate may be regarded as a clue to the position of the lesion in cases of facial paralysis. Any lesion causing facial palsy, at or above the geniculate ganglion, before the pars intermedia leaves the facial motor nerve to enter the medulla, should cause loss of taste on the palate as well as on the tongue. Gowers (1893, p. 227) stated: "Disease of the facial nerve within the skull does not impair taste"; but this is disproved by modern evidence. Dandy (Lewis and Dandy, 1930) found loss of taste on the tongue in cases in which he divided the facial within the skull, and I have seen taste loss produced on both the tongue and the palate in two cases, without any anaesthesia of the tongue, when I injected the inner half of the Gasserian ganglion. Some leakage of alcohol took place into the cave of Meckel, and must have spread backwards to the internal auditory meatus, setting up the vestibular syndrome, severe vertigo, coarse nystagmus to the opposite side, nausea and vomiting, faintness and pallor, and, if more than two or three drops of alcohol have leaked, nerve deafness and facial palsy. The facial palsy is likely to last for several weeks, but in my experience it has resolved. Permanent deafness I have seen occur only once, when a surgeon injected 2 ml. of alcohol into the Gasserian ganglion under a general anaesthetic.

Some Statistics Concerning Trigeminal Tic

I have notes of 2,083 cases of trigeminal tic, and have seen a great many more in various hospitals, especially St. Mary's, of which I have kept no notes. Of these 2,083 cases, 1,382 were women and 701 men, nearly a 2 to 1 ratio. Of this total 68 also suffered from chronic spastic paraplegia, of which the large majority were undoubtedly cases of disseminated sclerosis. One case was certainly a syphilitic paraplegia, and one other possibly amyotrophic lateral sclerosis, though the disease was very chronic. Therefore 3.1% of all the 2,083 cases of tic were complicated by disseminated sclerosis. I have also seen trigeminal tic complicating other diseases: three cases of tabes, two being bilateral tic, one each of Friedreich's disease, peroneal atrophy, Parkinson's disease, achondroplasia, and acromegaly. The average age of onset of tic in these 2,083 cases was 51.87 years—being 51.34 for women and 52.93 for men. Glossopharyngeal tic is very much rarer in my experience, with 20 cases (11 female and 9 male), one case being bilateral and one also suffering from trigeminal tic. The comparative frequency is probably in the neighbourhood of 1 to 200. I have never met with a case of disseminated sclerosis complicated by glossopharyngeal neuralgia, but Dr. Purdon Martin tells me he has seen one such case.

It is not very uncommon in the early stages of disseminated sclerosis for temporary numbness of one side of the face, with trigeminal anaesthesia, to develop rapidly without any pain then or subsequently. The numbness usually passes off in about two months. I have seen four such cases, of which I have notes that there was complete loss of taste accompanying the anaesthesia of the front half of the tongue, which the patient was aware of before being tested.

As to the age of onset in trigeminal tic, three-fifths of all the cases begin between 40 and 60. It is comparatively rare for the disease to begin after 80, but I have seen bilateral tic in a woman of 90, in whom I had to inject both Gasserian ganglia on succeeding days. This of course produced temporary jaw-drop and some difficulty in feeding for about three months, much to the indignation of the patient and her daughter; but the motor palsy always resolves, owing to the motor nucleus lying in the pons, out of harm's way. The daughter wrote to me when her mother was 93, saying that she was in good health and taking her food fairly well.

Many cases of trigeminal tic begin in the twenties, but only a few in the second decade. I reported the remarkable case of a small girl in whom trigeminal tic of the left third division certainly started as early as 16 months (Harris, 1943). The pain was at first naturally ascribed to teething, though the sudden short attacks of screaming were typical of tic. Her teething was late, beginning at 12 months, and was not complete until she was 3 years old. From that age there could be no doubt, from the mother's account, that the pain was limited to the left side, especially the lower jaw. I did not see the child until she was 4 years and 5 months old, when she was brought to me in the out-patient department of Maida Vale Hospital, on July 3, 1942. There could be no question about the diagnosis; I witnessed several attacks, in which she would moan and grizzle, with her hand to her left cheek for a few seconds, and then be immediately merry and bright until the next attack. X-ray films, reported on by Harrison Orton, of St. Mary's Hospital, showed that the teeth and mandible were normal for her age. Medicines had no effect, many spasms recurring daily, so on August 21 I tried to inject V.3 under general anaesthesia. It was not until my fifth attempt, on December 9, that I succeeded in injecting the left Gasserian ganglion with 0.5 ml. of alcohol. This produced total trigeminal anaesthesia, and with the exception of transient slight keratitis, which quickly yielded to treatment, she has had no recurrence of neuralgia. She takes no special precautions, and when last seen two years ago was quite well.

The Path of the Taste Fibres to their Cerebral Terminations

There can be little if any doubt now that the gustatory fibres of the chorda tympani and the great superficial petrosal (Vidian) nerves pass centrally to the geniculate ganglion, and thence by the pars intermedia to the medulla and pons. Their course and central terminations were carefully worked out 45 years ago by Nageotte (1906), physician and pathologist to the Bicêtre Hospital in Paris. He followed the Marchi degeneration of the fibres of the pars intermedia in the medulla. A patient had died from secondary carcinoma, with a recent unilateral facial palsy, and a small seed-like secondary growth was found in the chorda tympani just below the geniculate ganglion. No Marchi degeneration, of course, was found in the pars intermedia on the central side of the ganglion until the fibres pierced the side of the medulla, after shedding their sheaths of Schwann. He traced them into a nucleus continuous below with the substantia gelatinosa surrounding the fasciculus solitarius, or descending branch of the glossopharyngeal nerve. This nucleus reaches as high as 2 mm. below the convolutio trigemini, and is 14 mm. in length. He names it the gustatory nucleus, the pars fibres occupying the median 8 mm. and joining with the glossopharyngeal fibres below, and above with numerous trigeminal fibres which he traced

into the upper portion of the nucleus. From this nucleus doubtless secondary fibres decussate to run upwards on the other side of the medulla and pons, close to the middle line, which may be called the gustatory fillet, which runs separately on the inner side of the median sensory fillet and its trigeminal portion. Evidence of its almost median situation is afforded by clinical cases of pontine thrombosis and pontine glioma, and by a published case (Harris and Newcombe, 1926) of syringobulbia with unilateral lesions of cranial nerve nuclei, but with bilateral agusia. Nageotte thought the fifth-nerve fibres which he described as entering the rostral portion of the gustatory nucleus were true taste fibres, but this view should now be discarded.

Before accepting the view that the fifth nerve contains true gustatory fibres it must be demonstrated that taste persists or returns on the tongue after destruction of the chorda tympani, which would be comparable to the frequent preservation or return of taste after total destruction of the fifth nerve.

The thalamic termination of the gustatory path is believed to be the postero-ventral nucleus, whence taste sensations were formerly stated to be relayed to the hippocampal gyrus, near the olfactory centre, partly because of the frequent association of taste and smell in recognizing and distinguishing certain flavours. Börnstein (1940) pointed out that taste appears to be even more closely related to somatic sensation of the mouth and tongue than to olfaction, a view which harmonizes completely with my assumption of a welding of fifth-nerve tactile and pain sensibility with the gustatory sensations initiated by the chorda tympani in the front of the tongue and by the Vidian in the palate, the glossopharyngeal common sensibility with its gustatory fibres supplying the back of the tongue. Penfield and Erickson (1941) locate the cortical centre in the inferior portion of the post-central area, which Börnstein (1941) defines as the foot of the post-central gyrus in the parietal lobe just behind the motor chewing centre in the precentral gyrus.

The Share of the Fifth Nerve in Taste Perception

The apparent conflict of views on the gustatory function of the chorda tympani and great superficial petrosal (Vidian) nerves on the one hand, and of the fifth nerve on the other, is to be explained by assuming that there is a correlation or welding of the sensibility of the tongue and palate supplied by the fifth nerve with the true gustatory sensations from the chorda and petrosal nerves. In normal people taste is therefore perceived through the dual function of normal sensibility of the fifth nerve and the gustatory stimuli supplied by the chorda and Vidian nerves. In all cases of total lesion of the chorda and Vidian nerves, as may occur in geniculate herpes, or in facial palsy due to a lesion at or above the geniculate ganglion, taste will be permanently lost on the front half of the tongue and palate, the normal sensibility of the fifth-nerve supply to those parts being quite unable alone to provide a taste sensation.

In the great majority of persons a complete lesion of the mandibular and of the maxillary nerves will abolish the tactile, pain, and thermal perceptions on the tongue and palate, and the chorda or petrosal nerve alone may be insufficient at first to arouse the sensation of taste. In some persons this welding of the tactile and pain stimuli on to the gustatory function of the chorda and Vidian nerves is so important that taste is never regained, as is shown in many of my records, as well as in numbers of cases published by Gowers and others of permanent loss of taste in fifth-nerve lesions. In most people taste is gradually regained after fifth-nerve lesions, after varying intervals of hours or days to weeks, months, or even years, according to the extent of dependence on the tactile, pain, and thermal stimuli of the fifth nerve in the combined gustatory sensation of the individual. In a small minority—I have met with between 30 and 40 such cases—no loss of taste is found a week or two after total anaesthesia to

touch and prick has been produced by interruption of the third division of the fifth nerve, or by complete Gasserian destruction, or by sensory root resection.

As I have shown, a total second division lesion or an inner ganglion injection produces loss of taste on the palate without any anaesthesia or loss of taste on the tongue. I have no record of return of taste on the palate in such cases, but I have no doubt that it would occur in many of the cases, just as taste returns in the majority of third division or Gasserian cases.

Reaction Time

This varying dependence in different individuals on the share in taste perception of the normal sensibility of the tongue and palate is shown by the fact that many normal persons, when tested with the tongue kept protruded, are unable to name the variety of taste, whether sweet, salt, sour, or bitter; when, however, the tongue is withdrawn and moved around in the mouth, the correct answer is given after a few seconds. Others, again, are quick in indicating their recognition of the variety of taste within one or two seconds of the testing solution being painted on the side of the front of the tongue. Others are so slow in recognizing varieties of taste that they may make mistakes even after withdrawing the tongue within the mouth. This variation in speed and accuracy of reply is even more marked when testing the half-palate. I have, however, in many cases been able to obtain definite results in testing the palate, especially in persons of quick intelligence, though much less so in old people of poor education. It has been stated that two-thirds of the taste buds may be atrophied in old age.

The speed of reaction times in the full recognition of taste may advantageously be compared with the reaction time to a touch or prick on the tongue, or of the three other special senses of sight, hearing, and smell. A flash of light, the sound of a bell struck near by, or a pin-prick on the tongue is practically instantaneously recognized; even a sense of smell, whether pleasant or unpleasant, is almost as rapidly recognized, though inhalation is necessary and the varieties of scent will take longer to differentiate.

If we take four small bottles containing water, salt, sugar, and bitter solutions at the same room temperature, and with a medium-sized camel-hair brush sweep a brushful of water across the front of the tongue of a normal adult assistant, nothing except the touch is noticed. But when, after drying the protruded tongue with a clean handkerchief or white blotting-paper, a similar brushful of one of the three other solutions is used, an immediate marked difference in sensation is felt, often as a chilly or slightly smarting sensation, but not as a definite taste at first, though "sweet," "salt," or "bitter" may be pointed to—often wrongly—and it is not as a rule for several seconds that the correct taste is fully appreciated. The time of reaction varies much in different persons, probably owing to the different degree in which the individual is dependent on fifth-nerve sensibilities; yet the reaction time is far longer than that to pin-prick on the tongue, or to a flash of light, or to a sudden clang of a bell. This delayed reaction to taste may be understood when we consider the different method of appreciation of taste, in the welding of fifth-nerve or glossopharyngeal tactile, pain, and thermal sensibility with the gustatory stimuli of the chorda, Vidian, or glossopharyngeal nerves, which almost certainly takes place in the cortical centre for taste in the post-central gyrus.

This physiological view I am now putting forward explains the difficulty surgeons and physiologists have had in accepting the idea that the fifth nerve has any share in taste production. Because of the variability of results concerning loss of taste in fifth-nerve lesions the tendency has been to ignore reported cases of loss of taste in fifth-nerve lesions as due to faulty observation.

In view of my results of loss of taste in large numbers of fifth-nerve total lesions, the scheme I have presented seems more than probable.

Conclusions

The large number of cases in which I have tested taste at different intervals of time, after lesions of the fifth nerve have been produced by disease or by alcohol injections of the Gasserian ganglion or its branches, afford definite proof that:

1. Tactile sensibility, including pain and thermal fibres, in the tongue and palate has, in the great majority of persons, an important share in the recognition of taste sensations.
2. In a small minority taste is permanently lost or much diminished in intensity after total fifth-nerve lesions.
3. In the large majority taste is lost on the front of the tongue immediately after such lesions, but returns after intervals varying from a matter of hours, days, weeks, months, or even years, according to the individual degree of dependence on fifth-nerve sensibility.
4. The above three facts prove that the gustatory stimuli provided by the chorda tympani and petrosal nerves are insufficient alone, except in a small minority of persons, to provide full gustatory sensation immediately after a fifth-nerve lesion, but that a dual function of fifth-nerve sensibility together with chorda and petrosal gustatory stimuli is generally required.
5. For the back of the tongue, in the majority of people, taste perception is served by the combined common sensibility and gustatory function of the glossopharyngeal nerve only. In a small minority the tactile and pain sensibility of the back of the tongue is provided, partially or wholly, by the fifth nerve, and taste may be abolished for years in total fifth-nerve lesions.
6. In cases of facial paralysis, if the lesion is at or above the geniculate ganglion, taste is lost not only on the tongue but also on the palate.
7. The palatal loss of taste in these cases of facial palsy is proof that the great superficial petrosal (Vidian) nerve supplies gustatory fibres to the palate, and that their stimuli reach the geniculate ganglion and the pars intermedia via the main palatal nerve and Meckel's ganglion.
8. Taste stimuli reach the medulla and pons via the pars intermedia wrisbergi and the gustatory nucleus, as described by Nageotte, and thence pass by a nerve relay decussating to the opposite side, forming the gustatory fillet, running close to the midline through the pons to the crus, and terminating in the postero-ventral nucleus of the thalamus. Thence the gustatory stimuli are relayed to the lower part of the post-central gyrus in the parietal lobe, immediately behind the motor chewing centre in the pre-central gyrus.

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