ANATOMIC PATHWAYS RELATED TO PAIN IN FACE AND NECK*

JAMES A. TAREN, M.D., AND EDGAR A. KAHN, M.D.
Section of Neurosurgery, Department of Surgery, University of Michigan Hospital, Ann Arbor, Michigan

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The remarkable ability of the higher portions of the central nervous system to readjust to surgical lesions presupposes the existence of alternate pathways. This hypothesis has been tested with regard to a specific localized sensory input, pain from the face.

Our method has been to study the degeneration of nerve fibers by Weil and Marchi techniques following various surgical lesions in man and monkey. The lesions have consisted of total and selective retrogasserian rhizotomy in 3 humans and 3 monkeys, medullary tractotomy in 2 humans and 2 monkeys, and extirpation of the cervical portion of the nucleus of the descending tract of V in 2 monkeys. The findings were duplicated in all.

Degeneration in the descending tract of V following selective retrogasserian rhizotomy in man and monkey confirms that portions of all 3 divisions of the trigeminal nerve are represented as far caudally as C4 and that the mandibular, the maxillary, and the ophthalmic divisions have an essentially dorsal to ventral arrangement, respectively.1,4,6 Fig. 1 is a diagram of the first-order neuron for pain and temperature from the face, the descending tract of V, and shows that the most central areas of the face terminate highest on the pars caudalis of the nucleus of the descending tract of V while the most peripheral areas of the face end lowest. Fig. 2 depicts the descending tract of V at various levels. Fig. 3 shows that mandibular

V is most dorsal in the tract. Fig. 4 is a diagram of the medulla 6 mm. below the obex which we believe to be the optimal level for tractotomy. The operation is done with the patient in the sitting position to facilitate exposure. The medullary incision, 4–5 mm. in depth, extends from the bulbar accessory rootlet to a line extrapolated from the posterior rootlets of the 2nd cervical nerve root. An adequate incision results in complete analgesia of all 3 divisions as well as analgesia in the distribution of VII, IX, and X except for sparing of the vermilion border of the lips (Fig. 5). A degree of ataxia of the ipsilateral upper extremity usually accompanies effective tractotomy and is caused by compromise of the cuneate and the spinocerebellar systems. Too anterior an incision into the already crossed lateral spinothalamic and ventral secondary ascending tract of V may produce analgesia on the contralateral body and face. Our failure in the past to eliminate

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Fig. 1. Diagram of the origins and terminations of the descending tract of V. The fibers shown are those carrying impulses of pain and temperature.
mandibular-division pain was the result of an incision that was not dorsal enough in extent. As further evidence for this latter point we cite a case in which medullary tractotomy 6 mm. below the obex spared the entire mandibular division. Revision of the tractotomy by extending the incision 1–2 mm. dorsally resulted in analgesia of all 3 divisions of V except for sparing of the vermilion border of the lips.

Fig. 6 is a diagram of the usual secondary pathways mediating impulses from pain stimulation of the face or of the cervical region to the posterior ventral thalamic nucleus. The ventral secondary ascending tract of V subserves the former and the lateral spinothalamic tract the latter. The nucleus of the spinal tract of V is overlapped by the dorsal-horn gray; consequently the cell bodies of origin of the second-order neurons of these two tracts are in proximity. Each tract has a specific termination in the thalamus where synapses occur and third-order neurons ascend to a specific area of the somesthetic cortex.5,8

Degeneration in the ventral secondary ascending tract of V produced by incision into, or excision of, the cervical part of the nucleus of the spinal tract of V shows that this ascending tract swings forward to cross chiefly at medullary levels rather than at the level of origin of the tract. Fig. 7 from a human medullary tractotomy shows the
fibers of the ventral secondary ascending tract of V. These bundles of fibers are crossing as they ascend and actually are from a portion of the nucleus of the spinal tract of V which is more caudad than this section. It is important to note that the ventral secondary ascending tract of V does not cross the midline directly as do the fibers carrying pain impulses from the cervical region that cross into the lateral spinothalamic tract.

Fig. 8 is a diagram of the region of overlap in the upper cervical cord between the spinal nucleus of the descending tract of V and the dorsal-horn gray. This diagram suggests cross synapses in this area. Fibers carrying pain impulses from the face may cross synapse with the fiber entering the lateral spinothalamic tract from the upper cervical area, thus allowing pain impulses from the face to ascend into the upper cervical region of the thalamus and the “upper cervical

Fig. 5. Photograph following left medullary tractotomy in a patient with intractable pain in the face from carcinoma of the tongue which is shown. The patient is free from pain. Except for sparing of the left upper lip, which is slightly more than usual, there is analgesia of entire left face including faucial tonsil and tympanic membrane.

Fig. 6. Diagram of the second- and third-order neurons mediating facial and cervical pain.
field" of the cortex. The converse appears equally probable anatomically; pain impulses from the neck may cross synapse into the ventral secondary ascending tract of V thus gaining access to positions ordinarily thought of as belonging functionally to pain impulses from the face. Of the existence of the two pathways there is no doubt; of the close, anatomic relation of incoming pain fibers from face and neck in the upper cervical cord there is also no doubt; whether cross synapse actually occurs remains to be proved. It is perhaps suggested by such curious clinical phenomena as the relief of tic douloureux by occipital nerve block, or that high cervical cordotomies without bilateral posterior rhizotomy of C2, C3 and C4 may fail to produce high levels of complete analgesia or to relieve pain in cervical areas. This permits us to hypothesize that perhaps the pain impulses from cervical areas may circumvent a surgical lesion in the lateral spinothalamic tract by detouring by way of the ventral secondary ascending tract of V.

We conclude that impulses resulting from pain stimuli to face (or to cervical regions) possess two possible anatomic pathways to the thalamus—the lateral spinothalamic tract and the ventral secondary ascending tract of V, each ending in definite portions of the posterior ventral thalamic nucleus. This also suggests a double representation for facial (or cervical) sensibility on the contralateral cortex. Thus it would appear that cortical sensory patterns are determined by the sensory input from the periphery and that these patterns therefore must shift functionally whenever the peripheral pattern is shifted.

REFERENCES
DISCUSSION

Dr. Robert B. King: This has been a delightful presentation with many detailed considerations of a complex pain-relay mechanism.

The postulate that we have heard this afternoon might be corroborated further by the common observation that subcapsular pain associated with retro-orbital pain frequently may be relieved, albeit for a transient period of time, by an occipital-nerve resection. Still in our experience the pain recurs frequently at a later date.

It has been noted also that a delayed potential, presumably analogous to the dorsal-root reflex, may be recorded in cat in the dorsal roots of C2 and C1 by stimulation of the trigeminal system. Stimulation of the dorsal roots of the upper cervical region may elicit, even under Flaxedil, an evoked potential in the trigeminal system. Whether or not this requires an intermediary neuron, as we have heard suggested today, has not been determined.

[Slide] In the cat, using the Nauta technique, we have used a similar variety of lesions to study distribution of degeneration in the trigeminal and cervical regions. Here you see the region of degeneration following trigeminal posterior rhizotomy in the cat. Circles indicate fibers in transit cut in a transverse direction; dotted lines those traversing the section under study, and the circle-X, preterminal degeneration.

This section is at the level of the pyramid decussation. After the trigeminal posterior root section degeneration extends down into the upper cervical region [slide] overlapping, in the upper cervical region, the entry zone of the root for the cervical fibers. They do not extend more centrally into the gray matter.

[Slide] The distribution of degenerating fibers following a lesion limited to a portion of spinal V subnucleus caudalis indicates degeneration extending into the region of the contralateral spinal thalamic tract (the so-called quintothalamic tract), and extending also bilaterally into the reticular formation.

[Slide] Section of the C2 dorsal root shows degeneration overlapping widely in this area with terminals in a zone common to both trigeminal and C2 rhizotomies.

[Slide] This degeneration, medial to the subnucleus caudalis crossing into the medial lemniscus ipsilaterally, followed a lesion of subnucleus caudalis as did also [slide] degeneration in the contralateral medial lemniscus.

[Slide] One of our most striking observations related to marked degeneration ascending in the spinal V complex after lesions of subnucleus caudalis. These degenerating preterminals ascend as far as the mesencephalic nucleus. Many show preterminal degeneration in the main sensory nucleus after small lesions in subnucleus caudalis.

[Slide] This summary of findings in 9 animals indicates in blue degeneration following posterior-root section, and in red, ascending in spinal V, the distribution of degeneration following lesions in subnucleus caudalis.

I should like to ask the authors whether or not they have determined any quantitative tactile alternation in the face following spinal V tractotomy? This was recorded by Walker using von Frey's hairs. May not the sparing of the vermilion border relate either to concentration of end organs or the examiner's capacity to effectively stimulate end organs in this region (in a manner analogous to that in which the cuticles of the nail may be the only region spared after a high cervical cordotomy)?

Does the analgesia maintain a permanent level after these lesions, and, if it becomes limited to a more restricted area, does it not resume its original pattern with the administration of a low concentration of nitrous oxide? And one last question: May not ipsilateral ascending pathways in spinal V or the reticular formation be in part responsible for the altered distribution of pain following C2 dorsal rhizotomy or spinal V tractotomy in addition to the crossing pathways that the authors have suggested?

Dr. Elizabeth C. Crosby: Dr. Taren's very interesting account of two distinct paths from upper cervical-cord levels to higher centers probably explains various clinical and anatomical facts that have been puzzling.

The double projection of impulses set up by painful and thermal stimuli from the peripheral projection of C2-C4 spinal nerves to higher centers has some clinical documentation in certain cases of combined syringomyelia and syringobulbia. In syringomyelia the anterior white commissure of the cord, carrying crossing fascicles for the lateral spinothalamic tracts, is affected. Bilateral loss of pain and temperature for slightly less than the levels involved follows. When syringomyelia continues forward to become syringobulbia, the spinal tract and spinal nucleus of V, situated away from the midline, are involved.

Sometimes when syringomyelia and syringobulbia are both present, between the areas on the face and the body insensitive to painful stimuli, a collar is left over the region of distribution of C2-C4 nerves, from which responses to painful stimuli are elicitable. These are the levels with double projection to higher centers. It is suggested that where such a collar sensitive to pain remains, impulses are still passing forward from C2-C4 levels over a relatively uninjured ventral secondary ascending tract of V.

That the "onion skin" pattern of Déjerine is projected not only to the spinal cord but that it is often—and perhaps always—carried forward in the ventral secondary ascending tract of V is suggested in some cases of posterior inferior cerebellar thrombosis. In such a thrombosis, together with other generally recognized signs, there is a loss of pain and temperature ipsilaterally on the face and contralaterally on the body. Occasionally there is also a partial loss of pain and temperature on the face on the side contralateral to the thrombosis. When the contralateral loss on the face is slight it almost invariably falls within the outer zone of Déjerine's onion-skin pattern. This is the portion of the face area projected by appropriate fibers of the three peripheral divisions of V to C3 and C4 cord gray and relayed to higher centers by those fascicles of the ventral secondary tract of V which cross most caudally and lie most laterally in the medulla. This suggests, as do Dr. Taren's diagrams, that the Déjerine pattern, rather than one established by the peripheral divisions of V, is projected to thalamus and then to cortex.

By the method of evoked potentials, two distinct areas on the contralateral sensory cortex of an experimental mammal were located (Woolsey and his associates, 1942, 1954) from which impulses could be recorded following cutaneous stimulation of the face.
One of these regions was the generally recognized "face" area of the sensory cortex. The other region, situated much higher on the sensory cortex, was in the area from which impulses could also be picked up following stimulation of upper cervical levels. The existence of this latter common region of reception of impulses from cervical and some facial regions favors the projection of impulses from both cervical levels and facial areas over the lateral spinothalamic tract.

Thus lesions at the transition from the cord to the medulla and those along the ascending paths, as well as cortical patterns, tend to document Dr. Taren's conclusions.

DR. FREDERICK W. L. KERR: I hope that these brief remarks won't be too disjointed, as it is an unexpected privilege to discuss this paper.

At the Mayo Clinic, for the past year, we have been working quite intensively on the problem of hemi-craniar pain. This problem I shall summarize briefly from the standpoint of anatomy and neurophysiology and then give the reasons that took us into the study of it.

We have been interested particularly in the problem that the first discusser of this paper mentioned, the overlap between the cervical and trigeminal systems at C1 and C2. Consequently, we have studied this by means of degeneration techniques, using the Nauta stain in the brain stem and cervical cord following trigeminal rhizotomy and rhizotomies of the dorsal cervical roots C1, C2, C3 and C4 as far as C6.

When one compares identical levels of the dorsal horn in the 1st and 2nd cervical segments, one finds that anatomically there is not only a zone of vertical overlap, but in fact there is an overlap of the afferent degenerating fibers into the same nuclear groups.

Of course this might mean that there are individual neurons for each one of these systems, that the trigeminal system has its own neurons, the cervical system has its neurons, and none of these neurons receives synapses from any but its own specific afferent fibers.

To clarify this problem further, we have carried out and completed a microelectrode study of this area. This has shown that there are neurons that receive convergent fibers, both from the trigeminal and cervical systems. The same neuron can be triggered from stimuli delivered to the peripheral branches of the trigeminal and the peripheral branches of the cervical roots.

Their number cannot be estimated from microelectrode studies. However, by using evoked potential techniques, we have found that there is evidence of occlusion; from the magnitude of this occlusion it is estimated on a preliminary basis that somewhere between 25 and 30 per cent of these neurons receive fibers from the two systems.

Now, this offers an alternative hypothesis or approach to the problem. Our studies indicate that the convergence of the pain pathways occurs at the level of the first interneuron in the dorsal horn at C1 and C2.

So much for the degeneration, evoked potential and microelectrode aspects of this problem.

The next aspect is really where we started from some 2 1/2 years ago. This came as a result of the work of Semmes, the Raney's, Hunter, Mayfield and many others on the importance of the cervical dorsal roots in pain in the head and face. We found in stimulating patients under local anesthesia that the 1st cervical dorsal root is present very frequently. It is usually stated to be the contrary. However, Larsell and most of the basic textbooks of anatomy note that most frequently it is present but it may be rudimentary. If one stimulates this 1st cervical dorsal root, referred pain to the back of the eye, to the forehead and pain up to the vertex is elicited commonly; rarely pain is evoked in the back of the head. Stimulation of the 2nd cervical root will give pain radiating into the face on occasion.

I will conclude by saying that this gives us significant possibilities for interpreting many of the atypical syndromes of pain in the face and head that heretofore have had no explanation. At the Clinic we have seen a large number of patients with atypical pain in the head, thanks to the interest and cooperation of the neurologists. The mechanism of convergence appears to account very satisfactorily in many instances for the somewhat bizarre syndromes complained of, but it is very premature to say anything more; certainly in some cases a psychogenic basis must be considered, but these cases probably constitute a much smaller group than is usually stated.

DR. JAMES GREENWOOD, JR.: One question that is not entirely facetious: Dr. Taren, would you anticipate that in the near future we neurosurgeons would be operating upon cervical disks for atypical facial pain, and, if so, at what level?

DR. JAMES A. TAREN: I wish to thank our discussers. While this work suggests a provocative hypothesis, we are not prepared to draw any firm conclusions other than that the anatomic pathways are present.

In response to Dr. King's questions, we believe that the most specific and most highly discriminatory tactile fibers end highest in the descending tract of V, above our level of incision, and therefore we would expect only the grossest of tactile sensation to be absent after medullary tractotomy.

We have not tested with von Frey's hairs. The suggestion that the density of the pain endings in the vermilion border might be responsible for the sparing of the lips is not in accord with the fact that the density of pain endings is not great in the lips; it is tactile endings that are more dense here.

Since we are dealing with malignancy and short-term survivals, a judgment concerning the permanence of our tractotomies is probably not valid.

Our techniques did not demonstrate ipsilateral pathways, but then we would not expect these techniques to demonstrate these pathways. There is evidence, of course, for multisynaptic ipsilateral pathways.

Dr. Kahn and I wish to express our appreciation to Dr. Crosby, since without her this work would not have been possible.