Speculative dialogue pervades many reports on the etiology of tic douloureux. This summary is similarly affected. Furthermore, we shall at times attempt to collate data and opinions which, in their own right, were not intended to be used in this presumptive fashion.

Tic douloureux, in its florid form, has long since prompted physicians, frustrated in its management, to conceptualize its etiology from within the limits of their clinical observations. These, at least, were valiant attempts but frequently attained only the limited dignity of presumed understanding. Few of us have had the good sense to patiently state, "what, therefore, I had to offer upon the nature of this disease is rather submitted for your consideration as matter of further inquiry than as opinions sufficiently established."

The occasional association of tic douloureux and multiple sclerosis in the same patient has long been argued as a circumstance in which the central nervous system might be examined for confirmatory evidence of the central origin of tic douloureux. The incidence of these associated diseases is variously reported as none in over 2,000 cases by Frazier, to 2 to 4% by Harris, and 7% by Chakravorty. Rushton and Olafson indicated that 2% of patients with tic douloureux had multiple sclerosis and 1% with multiple sclerosis developed tic douloureux. The implied criteria they used for establishing either diagnosis may seem less than satisfactory to some. Suffice it to say, seven of their 15 patients had painless paresthesia of the face for 4 to 21 years before the onset of facial pain, and in only five was it on the same side. In one, the paresthesia was on the right with tic douloureux on the left. Two patients were thought to have had tic douloureux as the first symptom of multiple sclerosis, but at least one of these had objective neurologic deficit when first examined and presumably for an unknown time before that.

Chakravorty noted one patient with paresthesia in the same divisional distribution as that in which tic douloureux developed 17 years later. In most of these reports, a full description of the neurologic examination and serial sections of the entire brain stem and thalamus were not undertaken. After reviewing reports of the pathological examinations, Olafson, et al., concluded paradoxically that there were plaques of demyelination on the side of pain at the trigeminal-root entry zone in four cases; that bilateral pain occurred with only one root entry-zone plaque in two cases; and that "in all cases plaques were found to involve the descending root of the trigeminal nerve bilaterally," irrespective of whether the pain was unilateral or bilateral. In most instances, little attention had been given to detailed sensory examination of these patients or to their total neurologic disability.

A recent case report by Olafson, et al., however, is more complete in this respect than most. They described a 42-year-old patient who experienced the onset of multiple sclerosis in 1937, a remission until 1947, followed by slow progressive spasticity in the lower extremities with paresthesia. In 1951, the patient developed right-sided facial pain, more in the distribution of the second division than in the third, and without trigger points. Pain was precipitated by chewing and talking. In 1953, at the time of examination, the patient had a wide-based spastic, ataxic gait; bilateral nystagmus of the out-turned eye; universally increased deep tendon reflexes and bilateral extensor toe signs; diminished coordination in the arms and legs; and spastic arms and legs, more severe on the left. There was no mention made of a sensory examination. The authors indicated that there were multiple sclerotic plaques throughout the white matter of the occipital lobe and the lateral angles of the lateral ventricles. The cerebellum and pons had scattered lesions, and there were numerous plaques scattered indiscriminately in the cord. There was a plaque at the entry zone of the right trigeminal root, where it penetrated the arachnoid. Serial sections were taken through the root entry zone but were
not continued through lower segments of the trigeminal system or midbrain, except as "multiple sections" were obtained. There were, in addition, plaques at the penetrating fibers of the mesencephalic root and the beginning of the descending portion of the fifth nerve on the left. There were perivascular infiltrations in the lower medulla "not immediately related" to the trigeminal nuclei. I have given this case in some detail as it was presented in the report, so that I may refer to it in later remarks. For the same purpose, let me mention another case reported earlier by the same authors, in which the patient noted that each time she had a period with increased pain in her face, she had a concomitant marked increase in the weakness and spasticity of her legs.

It has been disconcerting in this context that there has been no reported incident of multiple sclerosis associated with glossopharyngeal tic or vice versa.

For many years, Knight\textsuperscript{101} has been impressed with the high incidence of herpes simplex in patients with tic douloureux. He has drawn particular attention to the specific and unusual circumstances in which tic douloureux repeatedly precedes the outbreak of herpes simplex in identical divisions of the trigeminal nerve, repeatedly accompanies the onset of herpes simplex in the same division, and occurs for the first time, or changes sides, after a herpes simplex vesicular outbreak in the trigeminal distribution. He noted that 60\% of his patients had herpes simplex vesicles evident to the naked eye before any operative procedure and quoted Carton and Kilbourne\textsuperscript{15} as noting an incidence of greater than 90\% when a magnifying glass was used to look for the vesicles. He was further impressed with the limitation of the herpes simplex to the division in which the pain had previously been present, following a total posterior root section. He considered that the herpes simplex virus may first be introduced to the central nervous system before the outbreak of cutaneous vesicles. All of his patients had high antibody titres for herpes simplex, but no control group unaffected with tic douloureux was similarly studied.

"Activation" of herpes simplex in the skin after denervation has been thought by many to be secondary to altered metabolic processes in the peripheral nerve terminals following denervation procedures. Little support has been established for this thesis over the years, partly because of the high incidence of herpes simplex antibody titres and recoverable virus in the total population, the lethal nature of herpes simplex encephalitis, and the absence of preoperative vesicles in the view of most observers.

The occasional incidence of recurrent cases of tic douloureux in succeeding or similar generations of specific families\textsuperscript{70} would appear to occur too infrequently to justify serious consideration of establishing a familial or hereditary trait with or without a central mechanism. Nevertheless, when such a single family occurs, the suggestion is consistently reiterated and may yet prove to be of more than coincidental interest.

In 1938, Lewy and Grant\textsuperscript{111} presented a unique set of observations. They noted, with the use of graduated hairs and thorns, that 25\% of their 50 patients had decreased numbers of touch and pain points with higher thresholds for perception of these stimuli. They noted summation, radiation, persistent after-effect, hyperpathia, space-time amalgamation, and transmutation as the various reactions of their patients to stimuli. They contended, of course, that the continuance of sensation beyond the duration of the stimulus for seconds or minutes implied a delayed decline of an after-discharge and a persistent after-discharge in an augmented central excitatory state. In 32\% of their patients, the sensory responses which they elicited were not only from the face and the division affected by the tic, but from wide somatotropic areas. They indicated that 40 of 50 patients had signs of pyramidal or extrapyramidal disease. They broadened the classic description of tic douloureux by noting that their patients had other forms of pain patterns which they described as neuralgic, referable to the brachial, intercostal, and sciatic regions.

They drew many other correlations: a high incidence of hypertension; a high incidence of evident vascular disease; 50\% incidence of cardiomegaly, heart murmurs, and angina; 60\% incidence of renal dysfunction; marked vasolability in younger patients; the transient occurrence of tic douloureux in early life during periods of vasomotor instability and its subsequent recurrence 10 to 33 years later at a time when the patient developed high blood pressure. They also noted a high incidence of parental Bright's disease; 6\% incidence of
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handed familial tic douloureux; over 20% incidence of familial and personal migraine; 14% incidence of Parkinson's disease; and characteristic constitutional factors, including body build and manner.

From these data they concluded that tic douloureux was a manifestation of the thalamic syndrome, limited in general to the face and associated with other somatic complaints. They were reinforced in this view by a case of Dr. Frazier's with severe thalamic pain of the face, arm, and leg. The patient was relieved of the face pain by a posterior rhizotomy and of the arm and leg pain by cordotomy, presumably by reduction of total somatosensory input to the nervous system. They agreed that thalamic pain does not commonly affect the face, but indicated that only rarely were the secondary trigeminal fibers to the arcuate nucleus or centrum medianum involved, and then only with extremely large vascular lesions. They reported pathological material from five patients with cortical atrophy, ventricular enlargement, and multiple lesions in the thalamus. Although other physicians have never confirmed the observation, they implied a central vascular ischemic basis for this form of thalamic pain, noting further that the clinical response to cobra venom may be due to its capacity as a vasodilator in the central nervous system.

Karl, et al., made remarkable observations on seven patients with predictable triggers to whom they gave histamine, amyl nitrite, 10% carbon dioxide by inhalation, and nicotinic acid alternated with placebos. The vasodilators were effective in reducing or blocking the pain induced by repetitive stimulation of the trigger points. Placebos were without effect. Unlike Lewy and Grant, who implied that the vascular abnormalities were within the central nervous system, these authors considered it likely that an ischemic factor was important in the mechanism somewhere in the peripheral elements of the trigeminal system. They found it difficult to conceive of a central locus for this effect without any ischemic effects on other elements of the brain stem. They noted the relatively poor vascular bed for the Gasserian ganglion and suggested that reflex vasoconstriction in the peripheral trigeminal structures might be responsible for the episodic attacks of pain.

Subsequently, however, they reported in their studies of cerebral blood vessels that there was a clear parallel between the vascular bed of neural elements and the synaptic density in these regions, rather than with the size or number of neurons. Campbell also found that the distribution of oxidase granules within both the cell bodies and the intercellular spaces was presumably related to the dense synapse patterns as suggested by Marinesco. Because these synaptic structures in the Gasserian ganglion have not been demonstrated, we should consider further those areas of the trigeminal relays showing the greatest synaptic density. These areas, of course, have not been established, although it is true that by electron microscopy at least, axo-axonal synapses can be readily found in the substantia gelatinosa of nucleus caudalis of the fifth spinal nerve in a very dense pattern. Some synapses can also be seen in the interpolaris and oralis nuclei and within the principal sensory nucleus of the fifth nerve, but they are particularly well demonstrated in the nucleus caudalis. The test drugs used by Wolff are known to have a vasodilating effect on vessels of the central nervous system, but their effect on patients with tic douloureux has not yet been established.

There has been one other major approach recurrently considered in attempts to devise a more certain understanding of the basis of this painful state. Pennybacker emphasized the analogy with paroxysmal affections and the high incidence of migraine and, indeed, of epilepsy among patients with tic douloureux and their families. He noted patients in whom migraine stopped with the onset of tic douloureux and, perhaps of more significance, a patient in whom epilepsy ceased at the onset of tic douloureux. He noted that, in general, seizures recur only intermittently with the presence of a known tumor or scar and made a further analogy between tic douloureux and a specialized form of reflex epilepsy. If such were the case, it must be very special indeed, for only in the rarest of circumstances does one find reference to pain of any form other than headache as an initiate symptom for paroxysmal disorders. Foerster, Gowers, Jackson, and Penfield, et al., each reported one such case. In no instance was the pain described as paroxysmal or lancinating. An increased incidence of dysrhythmic electroencephalograms in patients with tic douloureux has been reported. Wilson noted, however, that it is simpler
to conceive of the neuralgic paroxysms of tic douloureux as sensory epileptiform discharges, a rather neat semantic play on the usual use of these terms. He said, "If lesions exist, they should be sought rather in some efferent sensory inhibitory mechanism, which a variety of reasons suggests it is needful to postulate." He pointed out that no constant lesion or sensory deficit had ever been demonstrated. He then suggested that an efferent sensory inhibitory mechanism might suffice in the absence of any suggestion of an afferent excitatory mechanism. He was aware that such systems had been at least partially demonstrated in special sensory systems.

Norman Dott stated that the origins of tic douloureux are "in the brain stem," causing "something analogous to a short circuit, so that... touch or movement are registered in consciousness as severe pain." He assumed that the pathologic agent is usually vascular in the aged, but in the young is usually a neurodegenerative or neurotropic virus infective lesion. This suggestion becomes particularly interesting in view of recent studies of slow (latent viral) infections in mammals.

In 1959, Kugelberg and Lindblom undertook a semiquantitative study of the relation between the attack of pain and stimuli applied to the skin trigger zones. Their purpose was to identify a basic pattern of the trigger mechanism. A light tactile stimulus or hair displacement was the most constantly effective stimulus. In susceptible patients, a vibratory stimulus causing rapid distortion of the skin was effective. Electrical stimulation of an infraorbital nerve with a trigger area in its peripheral distribution was ineffective, as were a number of other stimuli which they chose, after unsuccessful trials, to exclude.

Four factors were prominent in their observations:

1. A minimal afferent discharge may be effective, as with the rapid displacement of a single hair.

2. Spatial summation of impulses triggered paroxysms of pain at lower stimulus strength and with a shorter latent period. Temporal summation of impulses was evident in all but a few patients. The length of time necessary to evoke an attack of pain by repetitive stimuli of a small trigger zone was "largely dependent on the stimulus intensity," indicating that an excitatory state necessary to fire an attack may be built up over a considerable period of time by the temporal summation of afferent impulses. To test how quickly the effect of a stimulus subsided, the trigger threshold was determined at different intervals after a conditioning stimulus, both at threshold and subthreshold. The test stimulus was delivered at 5, 10, 15, and 25 seconds after the conditioning stimulus. The time lapse necessary to again reach trigger threshold was measured. Test results on patients with stable trigger thresholds were consistent in three of five individuals.

3. The response to an effective stimulus was occasionally one of paresthesia but more commonly was a jab of pain, which repeated itself in rapid succession when the stimulus was continued. Occasionally when the stimulus had been prolonged, the pain became sustained, continuing even after the stimulus had been discontinued.

4. Refractory periods were examined, since this is a classical feature of the clinical syndrome. In most cases the refractory interval could be broken by stimulating at higher intensities, although usually the pain was then diminished in intensity. The duration of refractory phases appeared to be a function of the duration and intensity of the preceding pain attack rather than of any characteristic of the stimulus.

They concluded that touch, and possibly tickle, were the adequate stimuli to precipitate an attack of pain and that the largest touch fibers alone were not involved. Fibers transmitting touch stimuli along trigeminal pathways project into the main sensory nucleus as well as into the spinal nuclear complex.

In summary, while a single hair was sometimes sufficient to provoke an attack, spatial and temporal summation were more commonly necessary. A rapid fall in excitability following the cessation of the stimulus might persist for 20 seconds. An attack of pain was followed by a refractory period of 2 or 3 minutes depending on the duration and intensity of pain. Lidoacaine and hydantoine raised the threshold for these effective stimuli and shortened the duration of an attack. The long summation times, the tendency of the attack to be self-maintained, the effect of anti-epileptic drugs, and the long-lasting refractory period suggested to them that the mechanism responsible for the paroxysm of pain was situated centrally and probably in the brain.
system, in structures related to the spinal fifth nucleus. While their detailed clinical study does not specifically consider the cause of tic douloureux, but rather its mechanism, it does focus our attention on central relay pathways and reminds us of Kinnier Wilson’s suggestion that we consider efferent inhibitory mechanisms.177

Lewy and Grant111 suggested the possible role of efferent (parasympathetic) fibers from the mesencephalic trigeminal nucleus extending through the trigeminal system to the periphery. Since no such morphologic structure has been demonstrated, this possibility and its further interpretation by Gardner54 is mentioned only in passing.

List and Williams118 thought it unlikely that a cortical or thalamic lesion could account for the remarkable precision and spatial restriction of trigger zones and the divisional distribution of the pain. They concluded, as did Crue,23 that the “focus” of the tic mechanism should be sought at the level of the lower brainstem and that the abnormal over-excitability of the neural elements in this region could be caused by senescent changes in cells of the trigeminal nucleus. Morphological or functional vascular disturbances were not considered primary factors in the production of the attacks of pain.

If one records from the stimulated infraorbital nerve of a cat or monkey, the afferent spike is followed within 3 to 5 msec by a complex sequence of deflections conducted from the brain stem to the periphery.155 The same response may be evoked by stroking vibrissae. This element of the evoked response is conducted in an efferent direction over the larger sensory fibers. Either a posterior rhizotomy or a spinal fifth nucleo-tractotomy rostral to the nucleus caudalis gradually augments this response, which can be interrupted by small intravenous doses of mephenesin or dilantin.98 Mild, graded pressure on the posterior root will completely interrupt the conduction of the dorsal root reflex at the site of pressure, so that the response no longer reaches the cutaneous elements. In the cat, the increased amplitude of this dorsal root reflex, after strychnine, is associated with marked over-reaction to a light tactile stimulus in the distribution of the trigeminal nerve.8 If cats are stimulated several weeks after the injection of alumina gel in the region of the nucleus caudalis, the response is similar.100

Control injections in the region of the root entry zone of the main sensory nucleus and the adjacent reticular formation were never followed by an electrophysiological change in the dorsal root reflex or by a behavioral change in the chronic preparations.

Deafferentation of chronic cat preparations was accomplished either by removal of skin or undercutting of skin on the affected side, or by compression of the posterior root so that the trigeminal dorsal root reflex is no longer propagated from its locus of origin to the skin. In animals so prepared, no over-reaction follows stimulation of the nucleus caudalis or the peripheral elements of the trigeminal nerve. Perhaps an analogous observation has been made in two tic-douloureux patients whose trigger points were in the second division and whose pain radiated to the vertex in the first division. In each instance, a band of novocaine injected above the eyebrow interrupted the radiation of pain distally to the vertex, although pain in the upper eyelid and eyebrow persisted. There is little information with respect to the presence of this efferently conducted potential in human beings. Although we have few observations in this respect, a potential much like that seen in the experimental animals has been recorded from the third division of a patient with tic douloureux under local anesthesia.

These alterations in the trigeminal dorsal root reflex were clearly shown to depend on a relay at the nucleus caudalis, which was secondarily reflected to higher relays. Small lesions in the nucleus caudalis of the cat, when studied by the Nauta-Gygax technique for tracing degeneration patterns, showed dense bundles of ascending degeneration within each element of the trigeminal spinal complex, as far as the mesencephalic nucleus.156 Bilateral projections were demonstrated in the medial lemniscus, the arcuate nucleus, the pars magnocellularis of the medial geniculate, and the reticular formation; they passed through the centrum medianum-parafascicularis to the nucleus centralis centralis and nucleus centralis lateralis. Responses with low thresholds, evoked by infraorbital nerve stimulation but dependent upon a relay at the nucleus caudalis, were demonstrated in the midbrain reticular formation and in the pars magnocellularis of the medial geniculate. The evoked responses in the medial lemniscus and in the region of the red nucleus did not appear
to be dependent on that relay. In more rostral elements of the thalamus, evoked responses in the region of the centrum medianum were entirely dependent on a relay at the nucleus caudalis, while those in other elements of the posterior thalamus were not. Following the application of strychnine to the nucleus caudalis, a response in the centrum medianum evoked by the infraorbital nerve or the nucleus caudalis was augmented following spinal fifth nerve tractotomy. This response persisted following stimulation of the nucleus caudalis but was abolished by stimulation of the infraorbital nerve. In a similar fashion, the evoked response in the midbrain reticular formation incident to infraorbital nerve and nucleus caudalis stimulation was augmented by strychnine, but did not persist following infraorbital nerve stimulation and tractotomy of the spinal fifth rostral to the nucleus caudalis. In the nucleus ventralis posterior medialis, however, the evoked response was not augmented following the application of strychnine and persisted in reduced amplitude following the tractotomy.

Projections from the central nervous system to the substantia gelatinosa of the nucleus caudalis have been identified in Nauta-Gygax preparations following minimal lesions of the lateral cuneate nucleus, the cuneate nucleus, the contralateral nucleus caudalis, the adjacent reticular formation, the sensory motor cortex, the C-1 and C-2 dorsal roots, and the seventh, ninth, and tenth cranial nerves. No ascending elements from spinal cord segments below the C-2 root entry zone have been identified.

The significance of these projections is not certain. A conditioning stimulus alters the excitability of elements activated by the test stimulus. The conditioning stimulus can be applied to the contralateral or ipsilateral sensory motor cortex, the reticular formation of the pons or the medulla, the contralateral infraorbital nerve, the dorsal columns, the ipsilateral or contralateral radial or sciatic nerves, or the ipsilateral optic nerves. When the sequence of conditioning and test stimuli are applied to the isolated dorsal column, the amplitude of the antidromic spike of the primary afferent neuron is clearly increased while the amplitude of the secondary neuron and dorsal root responses are diminished. This altered state of excitability, while maximum in the cat at approximately 30 to 50 msec, persists for a period up to 400 to 500 msec. The same phenomenon can be demonstrated in the squirrel monkey. A tractotomy at the obex allows further depolarization of the afferent terminals in the rostral relay nuclei, suggesting that the nucleus caudalis may maintain a hyperpolarizing influence on the primary afferent terminals of the rostral relay nuclei.

We have used these illustrations of evoked response to demonstrate that, within the trigeminal system and specifically related to the nucleus caudalis, a centrifugally-conducted dorsal root reflex can be elicited by stimulation of touch fibers in the trigeminal distribution. They also show that conditioning stimuli from a wide number of related remote loci within the central nervous system may materially alter (inhibit) the excitability of the central terminals of primary afferent trigeminal neurons. Several other workers have also demonstrated this phenomenon. To this limited extent, there is evidence in cats and monkeys of mechanisms suggestive of Kinnier Wilson's efferent sensory inhibitory concept. Of course neither animal has been known to have tic douloureux.

Harris, in 1936, Surtees, in 1954, and Chakrovorty, in 1966 have each stated that tic douloureux is probably a syndrome and not a disease. They, and Professor Dott, implied that this syndrome may have many causes, some remote from any morphologic component of the trigeminal nerve. Their hypotheses suggest that there are unusual characteristics of the trigeminal nerve which determine the unique pain pattern we call tic douloureux.

The pathologic process we seek need not be restricted to the immediate elements of the trigeminal nerve or its primary relay nuclei. In cases of tic douloureux with multiple sclerosis, it is evident that the central nervous system has been altered in many regions besides that of the trigeminal system. We cannot exclude the possibility that multiple lesions and multiple agents may lead to secondary changes within the trigeminal complex, and that tic douloureux may become manifest because a physiological mechanism has been triggered by any one of a number of "etiologic" circumstances.
Summary and Conclusions
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Summary

Anatomy of the Normal Trigeminal Ganglion. We have reported descriptions of normal trigeminal ganglia studied under both light and electron microscopes.

It is now clear that the trigeminal ganglion and root visualized at the “mesoscopic” level (with binocular microscope) include a group of intermediate fibers which appear distinct from the sensory and motor roots at the pons.

Comparison of human autopsy and biopsy material with animal ganglia fixed by immersion or perfusion have provided a basis for evaluation of pathological material and the recognition of artifacts. Focal degenerative changes independent of specific disease were noted in all species. In man, myelin alterations appeared to increase with advancing age. Nissl-body breakdown, lipolysis of myelin sheaths, and myelin deformation due to mechanical stress could be readily identified. Except for pigment content and a generally more complex neuron-satellite cell relationship, the fine structure of the trigeminal ganglion of man resembled that in other mammals.

Microscopic Characteristics of Trigeminal Neuralgia. Electron microscopically, trigeminal neuralgia is characterized by vacuolated but intact ganglion cells, degenerative hyper-myelinization, segmental demyelination with denuding of the axis cylinders, and axonal thickenings and tortuosities. The abnormalities are much more marked than the degenerative changes seen in normal ganglia. Many of these findings can be seen under the light microscope as well. We concluded that the concept that there are no pathological changes in the Gasserian ganglion associated with trigeminal neuralgia is no longer tenable. Many illustrations demonstrating this evidence have been included.

Anatomical Variations Contributing to the Mechanism of Trigeminal Neuralgia. We have reviewed evidence for the following:

1. Trigeminal compression at the petrous ridge including alteration in the angle of the dural band capable of narrowing the opening into the cavum, and preliminary clinical results following the cutting of this band.

2. The significance of vascular variations associated with trigeminal neuralgia and the preliminary results of surgical correction of arterial compression on the ganglion or root.

3. The clinical significance of the “intermediate fibers” in the ganglion.

4. Structural mechanisms capable of accounting for the initial good results with compression and decompression procedures.

Peripheral Versus Central Etiology. We have discussed:

1. The specific characteristic clinical features of trigeminal neuralgia, such as divisional distribution, location of trigger zones, and female and right-sided predominance that provide evidence for a peripheral etiology.

2. The evidence dealing with clinico-physiological (paroxysmal) manifestations indicative of a primary central disturbance and some recent experimental findings concerning the trigeminal “efferent” (dorsal root) reflex.

Conclusions

Consistent pathological changes of unknown etiology have been demonstrated in the trigeminal ganglia; they are most marked in patients with trigeminal neuralgia. These “degenerative” changes could be the basis for altered neuronal function. Mechanical factors such as petrous ridge compression or vascular pulsation, which might otherwise be of little significance, may combine with “degenerative” changes to produce an abnormal paroxysm of pain in response to a light sensory stimulus applied to the appropriate receptive field (trigger point).

We can make no definitive statement as to the neurophysiological mechanism responsible for the paroxysm of pain.
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