Stereotaxic Midbrain Lesions for Central Dysesthesia
and Phantom Pain*

Preliminary Report

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It was shown 25 years ago by Walker that sectioning of the lateral spinothalamic and quintothalamic tracts in the mesencephalon at the level of the superior callosum resulted in analgesia and thermanesthesia over the opposite half of the body.\textsuperscript{11} The open operation did not gain wide acceptance because of the occurrence of severe postoperative dysesthesias. In 1948, Spiegel and Wycis developed a stereotaxic approach coagulating the lateral tegmentum at the level of the posterior commissure.\textsuperscript{23} Recent reports of significant relief of pain by this method of treatment prompted our own study of the problem.\textsuperscript{5,7,16,21,26,28}

The present report concerns the attempts to relieve phantom arm pain and central pain in a group of 15 patients.

Characteristics Selected

The 15 patients suffered from spontaneous intractable pains due to pathological lesions at various sites in the central nervous system, which included the spinal cord, medulla, mesencephalon, thalamus, and cerebral cortex (Table 1). The criteria for selection were as follows: the pains must have been severe and intractable and not relieved by the usual methods of treatment. The duration of the pains ranged from 1 to 38 years. Numerous therapeutic attempts had been made to relieve the pains and had included such procedures as frontal leukotomy, alcohol injection of the peripheral nerves of the face and limbs, amputation of the painful limb, stellate block and cervical sympathectomy, dorsal rhizotomy, cervical percutaneous and open chordotomy, and psychotherapy. All these therapies had failed. Many of the patients were on large doses of analgesics and tranquilizers, two were addicted to drugs, and two had threatened suicide and required hospitalization. Severe depressions had occurred in 50% of these persons.

Only persons considered to be suffering from central dysesthesia were selected. It is well known that pains of central origin often result from spontaneous pathological lesions which only partially involve the ascending pain pathways. We define central dysesthesia as the type of pain described by Riddoch as, “spontaneous pain and painful over-reaction to objective stimulation resulting from lesions confined to the substance of the central nervous system, including dysesthesia of any kind.”\textsuperscript{110}

There were nine persons with central pains that were due to pathological involvement at five different sites in the central nervous system, including the anterolateral quadrant of the cervical cord, the lateral medullary plate of the medulla, the dorsal lateral mesencephalon, the posterior ventral nucleus of the thalamus, and the parietal lobe. The causes of the original pathological insults to the CNS were varied and included percutaneous and open chordotomy, mesencephalic tractotomy, arachnoiditis, spontaneous subarachnoid hemorrhage, cerebral vascular thrombosis, encephalomyelitis, multiple sclerosis, and cerebral trauma. Two of the patients had developed secondary dysesthesias following surgical procedures to relieve their pain. One woman (Case 15) had bilateral percutaneous cervical chordotomies and the other patient (Case 7) had a previous stereotaxic mesencephalic tractotomy. Five of the patients (Cases 1–5) suffered from phantom arm pain, and one man had a severe brachial causalgia.

Phantom arm pain usually has not been considered of central origin. However, we believe that in certain instances phantom

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pain may represent a form of central pain due to neurological involvement not only in the brachial plexus but also in the region of the spinal cord where the neural roots were avulsed. Our evidence for this interpretation is based on several observations. Many of our patients had a history of a severe traction injury to the arm or neck. Due to the injury, the arm was flaccid, areflexic, and all sensations abolished except for the complaint of the pain in the denervated limb. In addition to the neural injury, the arm had been traumatically amputated in one man (Case 1) while the other four men (Cases 2–5) had surgical amputations done at various times after the injury in an effort to relieve the pain. This left them with pain plus a phantom sensation.

Examination of the patients revealed clinical evidence of total avulsion of the brachial plexus and its cervical nerve roots; cervical myelograms showed extensive deformity and disruption of the cervical nerve root sleeves with extravasation of Pantopaque outside the dural sac. In one patient (Case 2) a cervical laminectomy had been done, and when the dura was opened the cervical roots (5 through 7) were found avulsed from the cord. Even though direct pathological evidence was lacking, it is possible that the injury extended into the spinal cord involving the synaptic regions of the substantia gelatinsosa.

The phantom arm pain was usually centered in the hand and fingers. The fingers of the phantom were described as clenched tightly in the hand, and there were complaints of distorted postures of the phantom

### TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age</th>
<th>Etiology</th>
<th>Pathologic Site</th>
<th>Type of Pain and Duration</th>
<th>Response to Therapeutic Lesions</th>
<th>Follow-up (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>62</td>
<td>traumatic avulsion of arm</td>
<td>brachial plexus</td>
<td>phantom pain 2 yrs</td>
<td>relief</td>
<td>alive 24</td>
</tr>
<tr>
<td>2</td>
<td>29</td>
<td>traumatic avulsion of brachial plexus</td>
<td>plexus and spinal roots in cord</td>
<td>phantom pain 2 yrs</td>
<td>relief</td>
<td>alive 24</td>
</tr>
<tr>
<td>3</td>
<td>68</td>
<td>traumatic avulsion of brachial plexus</td>
<td>brachial plexus</td>
<td>phantom pain 27 yrs</td>
<td>no relief</td>
<td>dead 12</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>traumatic avulsion of brachial plexus</td>
<td>brachial plexus</td>
<td>phantom pain 3 yrs</td>
<td>partial relief</td>
<td>alive 24</td>
</tr>
<tr>
<td>5</td>
<td>43</td>
<td>traumatic avulsion of plexus, incomplete</td>
<td>brachial plexus</td>
<td>causalgia 1 yr</td>
<td>relief</td>
<td>alive 18</td>
</tr>
<tr>
<td>6</td>
<td>47</td>
<td>traumatic avulsion of brachial plexus</td>
<td>brachial plexus</td>
<td>phantom pain 15 yrs</td>
<td>no relief</td>
<td>alive 9</td>
</tr>
<tr>
<td>7</td>
<td>61</td>
<td>trauma—cerebral stereotactic lesion</td>
<td>parietal lobe, midbrain</td>
<td>central pain 38 yrs</td>
<td>relief</td>
<td>dead 24</td>
</tr>
<tr>
<td>8</td>
<td>48</td>
<td>vascular thrombosis, post. inferior : cerebellar artery</td>
<td>lateral medullary plate syndrome</td>
<td>central pain 1 yr</td>
<td>no relief</td>
<td>dead 9</td>
</tr>
<tr>
<td>9</td>
<td>66</td>
<td>vascular thrombosis</td>
<td>sensory thalamus, internal capsule</td>
<td>central pain, face-arm 1 yr</td>
<td>relief</td>
<td>alive 15</td>
</tr>
<tr>
<td>10</td>
<td>58</td>
<td>vascular thrombosis</td>
<td>sensory thalamus, internal capsule</td>
<td>central pain, face-arm 2 yrs</td>
<td>no relief</td>
<td>alive 15</td>
</tr>
<tr>
<td>11</td>
<td>63</td>
<td>vascular thrombosis</td>
<td>sensory thalamus, midbrain</td>
<td>central pain 2 yrs</td>
<td>partial relief</td>
<td>alive 24</td>
</tr>
<tr>
<td>12</td>
<td>49</td>
<td>cerebral hemorrhage, intracranial aneurysm</td>
<td>thalamus, midbrain</td>
<td>central pain 2 yrs</td>
<td>relief</td>
<td>dead 29</td>
</tr>
<tr>
<td>13</td>
<td>44</td>
<td>encephalomyelitis, thrombosis ?</td>
<td>thalamus</td>
<td>central pain, arm 3 yrs</td>
<td>relief</td>
<td>alive 30</td>
</tr>
<tr>
<td>14</td>
<td>37</td>
<td>thrombosis ? multiple sclerosis ?</td>
<td>thalamus</td>
<td>central pain, arm 15 yrs</td>
<td>partial relief</td>
<td>alive 24</td>
</tr>
<tr>
<td>15</td>
<td>47</td>
<td>percutaneous chordotomy, arachnoiditis</td>
<td>spinal cord, multiple levels</td>
<td>central pain 20 yrs, arnts, chest, phantom leg pain</td>
<td>relief, only unilateral no relief</td>
<td>alive 3</td>
</tr>
</tbody>
</table>

* Bilateral operation.
limb. When the intensity of the patient's pain increased in the phantom, either spontaneously or with stimulations in the midbrain, he experienced an increase in his awareness of the phantom limb sensation.

There was more variation in the location and distribution of the dysesthesia in those patients who had suffered from spontaneous lesions at higher levels in the CNS. Each one of these patients exhibited some degree of hemiparesis and altered sensation in the painful area. The dysesthesia was localized for the most part to the face, arm, and chest, and was described as a “burning,” “tearing,” “unpleasant coldness,” or “raw flesh.” These pains tended to vary in intensity but were often exaggerated by the slightest external stimuli such as a touch or pressure in the hyperpathic cutaneous area. When emotionally upset, the patient often complained more of pain. Most patients exhibited marked emotional lability associated with bouts of crying and episodes of depression that required psychiatric treatment.

**Stereotaxic Procedure**

Two separate operative procedures were performed. At the first operation, a burr hole was made, and stereotaxic implantation of multiple chronic electrodes into the thalamus and midbrain carried out. The method of implantation and the results of stimulations in the midbrain and thalamus have been reported in detail.14

At the second operation, a lesion probe was introduced through the previously placed burr hole. The insertion of the lesion probe often paralleled closely, if not exactly, the electrode tracts, so that a close approximation of the lesion and the area of stimulation could be carried out (Fig. 1). The lesions were made with a high frequency coagulator,11 300 W working output. The high frequency lesion probe* is gold-tipped, measuring 2 × 4 mm, and contains a thermistor for measuring the heat generated by the probe.31 Each lesion was produced at 70–72°C for 30 seconds. In two patients, a string electrode* was used for the second operation. This electrode was introduced 5 to 7 mm from the midline with the string extended 3 to 4 mm laterally, and the lesion produced for 30 seconds at 100–110 mA. A total of 66 lesions were made in 15 patients (Fig. 2). One woman had bilateral mesencephalic lesions made 3 months apart.

If more than one lesion was planned, the initial lesion was situated 1 to 5 mm caudal to the posterior commissure, 3 to 5 mm below the horizontal extension of the AC-PC line, and 5 mm from the midsagittal plane. For a second coagulation, the lesion probe was withdrawn from the initial site and the second lesion made more laterally at a point 8 to 12 mm from midline. The dorsoventral orientation of the lesion probe was also aided by superimposing on the lateral roentgenogram the shadow of the lesion probe over the silhouette of the aqueduct beneath the collicular plate. Lesions of three sizes were produced; the smallest consisted of 1 or 2 coagulations (estimated size 30 to 60 mm²), a medium lesion of 4 coagulations (60 to 100 mm²), and a large lesion of 5 to 10 coagulations (100 mm³ + ).

**Results of Midbrain Lesions**

The lesions in the dorsolateral midbrain resulted in relief of pain in 69% of the patients, total in 44%, partial in 25%. The remaining 31% were regarded as failures (Table 2). Reoperations were done in six persons, with partial improvement in three, one with phantom arm pain and two with central dysesthesia experienced no relief. A woman (Case 15) with bilateral dysesthesia over her chest and arms due to previous bilateral percutaneous chordotomies had only partial relief of pain over one shoulder and arm following a series of bilateral mesencephalic lesions. The average duration of follow-up has been 18 months, with the longest period of significant relief lasting 30 months. No deaths were associated with the surgery. Three of the patients have died since operation, at 6 months to 2 years postoperative.

**Significance of Size and Location of Lesions.** The smallest lesion that resulted in the relief of phantom arm pain or central dysesthesia was situated 4.5 mm caudal to and 3.0 mm below the posterior commissure, and 8 mm from the midsagittal plane of the Sylvian aqueduct. Six such persons with small lesions (30 to 60 mm³) were either partially or totally relieved of their pain.

* Designed by Mundinger, Riechert, and Gabriel and manufactured by F. L. Fischer, Freiburg, West Germany.
One man (Case 5) did not have a thermal coagulation because his causalgic arm pain was relieved as the result of introducing the three chronic depth electrodes into the dorsolateral tegmentum. He developed a mild analgesia over his painful arm; the pain subsided over a period of several days, and relief has continued for over 18 months. Another man (Case 6) with a medium-size lesion (60–100 mm³) was not relieved of his phantom arm pain, while a third (Case 2) with phantom arm pain was relieved following enlargement of the original small lesion 6 months later.

The largest lesions (100 mm³) were the result of multiple operations, and these were done in five persons whose pain failed to respond to earlier operations. One woman (Case 12) with a central dysesthesia of the face caused by subarachnoid hemorrhage involving the thalamus did have a large lesion made initially at one operation; her pain had been relieved for a period of 2½ years when she died of a heart attack. In one man (Case 8) a series of 10 coagulations made at three operations failed to relieve the facial pains

Fig. 1. Case 7. Lateral x-ray films of a 61-year-old man with central pain of 38 years’ duration due to parietal lobe trauma. A. Visualization of ventricles and aqueduct with air and Pantopaque. B. First operation: probe for introduction of chronic electrode passing into the midbrain just below the posterior commissure and parallel to the aqueduct. The tip of the probe is approximately 3 mm from the midpoint of the aqueduct as seen in the anteroposterior view. Second operation: lesion probe introduced in same plane for high frequency lesion in midbrain.

Fig. 2. Extent of the stereotaxic lesions in the 15 patients. Each cross represents the center of lesion as determined from the lateral roentgenogram. Overlapping lesions were omitted. The crosses above the posterior commissure represent second and third operations extending the lesion in a more rostral direction. The crosses below the aqueduct were those associated with diplopias and spasticity in the leg.
TABLE 2

Results of mesencephalic lesions in 15 patients*

<table>
<thead>
<tr>
<th>Relief of Pain</th>
<th>No. of Patients</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Relief</td>
<td>8</td>
<td>44%</td>
</tr>
<tr>
<td>Partial Relief</td>
<td>3</td>
<td>25%</td>
</tr>
<tr>
<td>No Relief</td>
<td>5</td>
<td>31%</td>
</tr>
</tbody>
</table>

* Percentages based on 16 mesencephalic operations in 15 patients, 14 patients with unilateral operations, 1 patient with bilateral operations.

associated with a lateral medullary plate syndrome. At the postmortem examination 3 months after the last surgery, examination of the brain revealed that the entire dorsolateral tegmentum had been destroyed from the inferior colliculus into the thalamus, where the nuclei ventralis posterior, parafascicularis, and two thirds of the centrum medianum had been destroyed.

The position of the therapeutic lesion in relationship to the midline of the mesencephalon was related to the extent of the distribution of the analgesia over the contralateral half of the body. In some patients the analgesia did not extend exactly to the midline on the face. Five patients had a crescent-shaped area of spared or reduced pain and thermal sensation which extended along the lateral aspect of the nose and the adjacent medial portion of the cheek. In four of these patients the most medial extent of the lesion was estimated to lie 6 mm from the midline of the mesencephalon. When a lesion was placed more medially, the resultant analgesia always extended to the midline of the face and included the inside of the mouth, posterior pharyngeal wall, palate, and tongue, and also extended posteriorly over the head and ear to the skin at external auditory canal where sensation was normal. The nasal tickle and corneal response were also abolished. Although the corneal reflex was lost in 12 of the patients, they did experience some vague feeling when the cornea was touched. Pulling the hairs on the analgesic skin was not felt and testicular pressure was not painful.

In this group of patients the extent of the lower level of the analgesia began to fade into an area of normal sensation between the T-6 and T-8 dermatomes with varying degrees of the milder analgesia extending into the leg (Fig. 3). There were only three persons in whom a moderate degree of analgesia involved the leg, and these were due to the production of a large lesion which extended far laterally into the tegmentum.

Analgesia and Thermal Anesthesia. A marked to moderate degree of analgesia and thermal anesthesia accompanied the relief of the spontaneous central pains in all but one patient (Case 8) who suffered from facial pain due to a medullary infarction and the occurrence of a lateral medullary plate syndrome. The smallest lesion that resulted in a moderate to marked analgesia over the painful area reduced the spontaneous pains as effectively as the larger lesions. The common factor associated with relief of the pain was the production of a moderate to marked analgesia over the cutaneous region from which the spontaneous pain originated. Appreciation of pinprick sensation and deep muscle pinch was also reduced in the analgesic region. Position sense and vibration sensation were not altered postoperatively in the patients with small and medium lesions. In the persons with the central dysesthesia it was often difficult to assess the degree of appreciation of light touch preoperatively because of the aberration of the sensations originating from the painful area. Some degree of light touch sensation remained postoperatively in the analgesic zone.

Three persons exhibited a dissociation between the appreciation of hot and cold sensation despite the presence of analgesia and relief of pain. Two patients noted a greater reduction in appreciation of cold than hot sensation while the reverse was true in the third person.

Postoperative Dysesthesia. Five of the patients whose original pain was relieved by operation continued to complain of some degree of disagreeable sensations originating spontaneously from the area of postoperative analgesia. These patients described “aching” or “gnawing” sensations localized in their bones and joints. They admitted that these feelings had been present preoperatively but were overshadowed by the severe dysesthesia. Three patients (Cases 1, 2, 13) complained of a postoperative dysesthesia, mild in two but severe in the third. The dysesthesia originated from the region of the skin
FIG. 3. The sensory, motor, and ocular changes occurring with small to medium sized lesions in the mesencephalon. Note the sparing of the mid-portion of face centering the lesion at 6 mm from midsagittal plane.

where the postoperative analgesia faded into the zone of normal sensation. One woman (Case 2) whose central dysesthesia was relieved in her arm noted postoperatively a "burning" and "raw" sensation along the upper lip and in the roof of her mouth. Analgesia did not extend into this part of her face. She did not consider this new pain incapacitating nor has it become so after a follow-up period of 30 months. Two other patients had small areas of postoperative dysesthesia on the chest wall between T-6 and T-8. There was a narrow strip of skin which was hypersensitive to pinprick, light touch, and cold. One man (Case 1) complained bitterly of this postoperative thoracic hyperalgesia. His phantom arm pain had been relieved by the operation, but he still was depressed. Prompt treatment of the depression resulted in improvement of the postoperative dysesthesia although on clinical examination the small area of hyperalgesia persisted.

**Effect on Phantom Limb.** The pain in the phantom limb was markedly intensified in one man (Case 6) for 24 hours postoperatively. The therapeutic lesion had produced a moderate analgesia in his painful arm but despite this he was only relieved for a period of 2 weeks when the pain recurred. Reoperation resulted only in partial relief. Another man (Case 4) with phantom limb pain complained of a "flapping" motion localized to his phantom limb. It occurred immediately after the production of the therapeutic lesion and over the next 5 minutes the muscles in his stump could be seen twitching involuntarily at a rate of 1 per second.

The phantom limb sensation tended to
fade gradually over a period of several months but it was never completely abolished even with significant relief of the pain. The families of these patients reported a decrease in the patient’s attention to his phantom whereas preoperatively the patient’s every waking hour was often involved with some aspect of the phantom limb and the pain.

**Motor Sequellae.** Two men (Cases 1 and 4) developed postoperative weakness of the contralateral leg. In the first, the weakness cleared in 3 weeks with only a mild residual ataxia for another month. In the second, the weakness was more pronounced and he developed a spastic gait, which has persisted.

A woman (Case 11) with a central dysesthesia due to a cerebral thrombosis complained of a painful arm and an unpleasant coldness over half of her body. In addition, she also exhibited an intention tremor in the painful arm and an ataxic gait. Involuntary muscular twitches were noted in her facial muscles on the side of the pain. The therapeutic lesion in her midbrain not only partially relieved the dysesthesia in the arm but also abolished the tremor and the facial tic.

The woman (Case 12) with facial dysesthesia that was relieved after a single large lesion in the mesencephalon also suffered from generalized epileptic seizures as the result of a frontal lobectomy done 3 years before, which failed to relieve her pain. The epileptic seizures could not be controlled. After the mesencephalic lesion, however, small amounts of anticonvulsant medication brought the seizures under control.

**Ocular Complications.** A series of complex ocular defects occurred following the placement of therapeutic lesions in this region of the mesencephalon. These defects consisted of permanent loss of conjugate upward gaze (15 patients), temporary loss of conjugate downward gaze (6 patients), and transient nystagmus retractorius (13 patients). Diplopia was often a prominent initial complaint and occurred in 7 patients but persisted in only 3; two others were corrected by prisms. Pupillary changes consisted of bilateral miosis with the pupil on the side of the lesion 1 to 2 mm larger. The pupillary reactions to light for both the direct and the consensual light reflexes were sluggish or absent in the immediate postoperative period. The vertical optokinetic reflexes were also depressed or absent in those patients with disturbance of upward or downward conjugate gaze. Four persons exhibited within a few seconds after the production of the therapeutic lesion a forced conjugate deviation of eyes downward toward the side of the lesion.

**Severe Depression.** In 50% of these patients severe depression was a preoperative symptom, and most have had psychiatric treatment. In one man (Case 5) depressive symptoms cleared spontaneously within 1 week after relief of his pain. Two other patients (Cases 8 and 10) who had not been relieved despite multiple reoperations continued in a state of depression which required protective hospitalization. One of these patients (Case 10) suffered from thalamic pain localized in his face; he exhibited marked emotional lability and pseudo-bulbar signs both before and after surgery and his pain was not relieved. Four patients continued to have intermittent bouts of depression for months postoperatively, even after relief of pain. Preoperatively all of the depressed patients had been severely disabled because of their pain. Postoperatively three of the patients returned to full activity. One man (Case 5) manages a farm, another man (Case 2) an automobile agency. One of the women (Case 13) has resumed her household activities. Of the two patients with narcotic addiction, only one (Case 2) was able to stop drugs.

**Discussion**

Stereotaxic midbrain lesions to relieve intractable pain have not been used widely because of the high mortality of the earlier operations, the technical difficulties encountered in the precise localization of the therapeutic lesion to the midbrain target, a lack of understanding of the anatomical and physiological factors involved in the transmission of painful sensations in this region of the mesencephalon, the tendency of the analgesia to fade, the possibility of disagreeable postoperative dysesthesias, and the high incidence of ocular disturbances.

**Operative Mortality.** There has been a substantial reduction in the operative mortality in mesencephalic tractotomy from 24% (non-stereotaxic) to 7.4% (early stereo-
Stereotaxic Midbrain Lesions for Pain

Stereotaxic Technique. The first stereotaxic operation on the midbrain was devised by Spiegel and Wycis.23-24 The target area included the lateral spinothalamic tract, the quintothalamic tract, and the diffuse spinoreticular system at the level of the posterior commissure. The probe was introduced in a dorsoventral direction into the mesencephalon along a plane of 34° behind the interaural line. Leksel and others modified the original stereotaxic approach by introducing the probe into the brain stem anteriorly through the frontal region.7-14,17 We also used this anterior approach and found it well suited for the insertion of chronic electrodes. The anterior approach has one advantage in that the probe enters the dorsal tegmentum in a rostrocaudal direction paralleling the ascending sensory pathways. A disadvantage to the anterior approach may be the necessity to carry out more than one puncture if several lesions are planned side by side. With the probe oriented in this way there is less chance of entering the ventral tegmentum than with the more posterior approach of Spiegel and Wycis.24

Sensory Response to Stimulation. In cross sections of the midbrain the lateral spinothalamic and quintothalamic tracts appear as discrete localized bundles of afferent fibers which pass through the tegmentum lateral to the periaqueductal gray.29 Stimulation of the lateral regions of the dorsolateral tegmentum often results in a variety of sensory responses from more extensive areas of the midbrain than those occupied by these ascending pain pathways.2,10,14 A definite topographic arrangement of the afferent fibers from the face, arm, trunk, and leg fibers exists in the dorsolateral midbrain. The facial region is medial while the arm, trunk, and leg fibers lie laterally.22 There exists a region in or near the periaqueductal gray from which electrical stimulation evokes autonomic and emotional responses and sensations referred to the midportion of the face, plus the nasal and oral cavities.14,24

Facial Analgesia. Previous clinical reports indicate differences in the extent of the analgesia on the face.25,26,31 Walker and Torvik noted the preservation of the sensations in the center of the face but offered no explanation for this phenomenon.28,29 We noted sparing of sensation over the center of the face and in the oral cavity in four patients. It was due to the position of the therapeutic lesion in relationship to the mid sagittal plane of the midbrain. In our group of patients with spared midline facial sensation, we estimated that the medial edge of the therapeutic lesion responsible was approximately 6 mm lateral to the Sylvian aqueduct. Electrical stimulation in man has shown that facial, oral, and nasal sensations could be evoked from periaqueductal gray.13,14 Observations based on anatomical studies have also demonstrated afferent tracts in the periaqueductal region which may be associated with the transmission of various modalities of sensations from the medial facial, oral and nasal regions.25,30 A failure to include this mesial region of the midbrain in the lesion may in part explain the failure to relieve the facial pain in certain patients.

Various Target Areas. The relief of the pain was not only related to the degree of analgesia but to the extent of the analgesia in the painful area.26,31 To prevent a postoperative reduction of the analgesia and recurrence of pain, Spiegel and others advocated the placement of a large lesion in the midbrain involving both lateral tegmentum and mesial regions near the periaqueductal gray.33,17,26 In our experience the phantom pain was relieved by a small laterally placed lesion which spared the mesial region. A single coagulation in the dorsolateral mesencephalon has relieved one man (Case 1) of phantom pain for over 24 months while another man (Case 5) with a causalgic arm pain was simply relieved after the introduction of the depth electrode for stimulation. Pain originating from segments of the body such as the arm or hand appear to be relieved by small lesions localized in the region of the lateral spinothalamic tract. Spiegel and Wycis believed that the failure to control central pain might be due to a sparing of the diffuse ascending pain pathways.13,25 The larger lesions appeared more effective for relief of the central dyesthesia due to thrombotic lesions in the midbrain or the thala-
mus. Pains in the facial region seemed especially difficult to relieve. One of the reasons may be the presence of multiple and bilateral afferent pathways for facial sensations which ascend in the medial portion of the tegmentum and periaqueductal gray regions, and could only be completely destroyed by bilateral lesions.2,10,14 The most difficult pains to relieve appear to be those which originate from multiple insults to the sensory pathways within the CNS such as those that occurred in one of our patients (Case 15) who had had multiple bilateral chordotomies at dorsal and cervical levels. Her post chordotomy dysesthesia was only partially relieved on one side of her body even after extensive bilateral midbrain lesions.

Relief of Phantom Pain. White and Sweet have repeatedly commented on the difficulty in relieving brachial pain.23 Although high cervical chordotomy has been successful in a few instances there has been a tendency for the level of analgesia to fall followed by the return of the pain.5 We have been impressed that small and medium lesions in the midbrain successfully reduced the pain in four of the six patients with phantom arm pain. A longer follow-up period will be necessary before complete assessment of the result because failures occurring years after operation have been reported.25,26,53 Our findings agree with those of others who noted that the phantom sensation does not entirely disappear even with improvement in the pain.5,16,17,53 However, the patient was rarely as concerned about his phantom sensation if he was free of pain. Riddoch suggested that "it is as if the postural model had become frozen when normal sensation ceased."20 We observed that the pain in the phantom limb could be intensified when the mesencephalic region in or near the lateral spinothalamic tract was stimulated. It was of interest that the patient never did consider the effect of electrical stimulation as an exact reproduction of his pain. This suggested to us that the phantom pains may be caused by a series of complex afferent inputs from other sensory pathways, but the spinothalamic input seems of prime importance. The phantom sensation was less in its intensity but never completely disappeared after the lesions in the midbrain. We agree with White and Sweet who concluded that an essential portion of the neural mechanism responsible for the painful phantom must reside in the lateral spinothalamic tract.53

Postoperative Dysesthesia. Most neurosurgeons have considered the occurrence of a postoperative dysesthesia a serious deterrent to a midbrain tractotomy.21,25,33 It has been reported more often following the open operation than with the stereotaxic coagulations. We agree with Spiegel and Wycis that this complication is serious when it occurs but should not be considered as a contraindication for operation in persons with severe intractable pain who have failed to respond to other treatment.55 The influence of psychogenic and emotional factors must be considered always in any patient with severe pain, and these factors may play a role in the patient's reaction to his original pain or a postoperative dysesthesia.17 This was true in one of our patients (Case 1) who was severely depressed and complained bitterly of a postoperative dysesthesia which subsided with successful treatment of his depression.

Ocular Complications. The variety of ocular changes noted in this group of patients was related to the variation in the extent and position of the lesion in the midbrain. The dorsal midbrain represents an important neural region for the integration of complex ocular functions such as conjugate eye and head movements plus the pupillary reflexes.13 The most consistent ocular defect was a loss of vertical conjugate gaze, absence of the Bell's phenomenon, and depressed vertical optokinetic reflexes. Although the loss of upward gaze appeared permanent it did not cause a serious ocular disability because the patient was often not aware of the deficit. Bender has contended that a loss of upward gaze occurred only as the result of bilateral lesions in this dorsal region of the midbrain,3 but clinical observations by Cogan and others do not support this contention.4,6,13 The therapeutic lesions in these 14 patients were considered unilateral and appeared so in the postmortem examinations in two patients. The complaints of diplopia which occurred immediately postoperatively in every patient persisted in only three. In the early postoperative period the visual complaints were related to the complex forced ipsilateral deviations of the eye, nystagmus retractorius, and conver-
gence nystagmus which cleared within a few days or weeks. Two patients had complaints of diplopia months after operation; it was related to ocular fatigue and was not considered disabling. The most disabling diplopia resulted in the woman with bilateral midbrain lesions that only partially relieved her pain. The ocular disturbances should not deter the surgeon from recommending operation if the patient has severe intractable pain that has not been relieved by other treatment. It is important, however, to discuss in detail these ocular changes with the patient and family prior to the operation.

Contralateral Leg Pareisis. Associated with the relief of pain have been reports of postoperative weakness and spasticity in the opposite leg. This deficit was thought by Spiegel to be due to an injury of the cortical leg area by the stereotaxic probe. Torvik suggested that the cause was an injury to the efferent fibers of the extra-pyramidal motor system in the ventral tegmentum. He reported this complication in one patient after a midbrain lesion for relief of pain and the postmortem analysis revealed sparing of the pyramidal tract but a partial destruction of the ventral tegmentum. Two of our patients who developed weakness and spasticity in the contralateral leg also suffered from persistent diplopia due to partial 3rd nerve involvement indicating encroachment of the lesion into the ventral tegmentum.

Summary

A group of 15 patients suffering from intractable phantom arm pain and central pain have been treated by stereotaxic high frequency coagulation of the dorsolateral tegmentum of the mesencephalon at or below the posterior commissure. There have been varying degrees of relief of pain in the majority of the patients. Mesencephalic lesions may result in complex ocular and motor effects which must be taken into account before recommending the operation for relief of pain.

References