Somatosensory function following dorsal root entry zone lesions in patients with neurogenic pain or spasticity

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The goal of this study was to assess the effects of the dorsal root entry zone (DREZ) lesioning procedure, microsurgical DREZ-otomy (MDT), on spinal cord somatosensory function based on peri- and intraoperative clinical and electrophysiological data. The study was performed prospectively on a series of 20 patients suffering from either chronic neurogenic pain or spasticity. Physiological observations were made of the intraoperative evoked electrospinographic recordings as collected from the surface of the spinal cord.

The MDT procedure produced analgesia or severe hypalgesia, moderate hypesthesia, and only slight deficits in proprioception and cutaneous spatial discrimination on the body segments operated on. These clinical data correlated well with evoked electrospinographic recordings, which showed a moderate effect of MDT on presynaptic compound action potentials recorded from the spinal cord (N1 and N2), a partial or even reversible effect on the cortical postcentral N20 wave, a more marked effect on the postsynaptic dorsal horn waves N13 and N24, related to large primary afferent fibers, and a disappearance of dorsal horn waves related to finer afferents (N2 and possibly N3). These data provide evidence for an acceptably selective action of MDT on spinal cord nociceptive mechanisms, and for a partial, often slight, involvement of the other somatosensory domains. The presence of abnormal evoked electrospinographic waves is discussed in relation to the mechanisms of neurogenic pain and spasticity. The hypothesis of a "retuning" of the dorsal horn as the mode of action of MDT is presented.

Key Words • somatosensory evoked potentials • dorsal root entry zone lesion • electrospinogram • pain • spasticity

Promoted by the gate control theory of Wall, various surgical approaches have appeared since 1965 with the goal of alleviating chronic pain through an alteration in pain modulation at the dorsal horn level. Rather than stimulating inhibitory structures, one of the authors (M.S.) chose to explore the possibilities of interrupting or selectively destroying the paths or centers playing an excitatory role in pain modulation. With this rationale, the dorsal root entry zone (DREZ) lesioning procedure, microsurgical DREZ-otomy (MDT), was introduced in 1972 after anatomical studies of the human spinal cord revealed the existence in the DREZ of a spatial segregation of primary afferent fibers according to their size. This segregation has been confirmed in the monkey and is characterized by the convergence of fine group III and IV afferents in the ventrolateral part of the DREZ (Fig. 1). The MDT (Fig. 1) consists of a microsurgical lesion in this location, aiming to selectively interrupt the pain-related small myelinated and unmyelinated afferent fibers and to destroy the medial, pain-activating part of Lissauer's tract. A destructive effect of MDT on the most dorsal laminae of the dorsal horn could also be anticipated. On the other hand, MDT was conceived to spare the structures dealing with the inhibitory side of the gate control: that is, large-diameter primary afferents, the lateral part of Lissauer's tract, and the dorsolateral funiculus. Over the past 17 years, the MDT has been used to treat chronic nociceptive pain due to cancer, with an overall 83% good results.

In patients suffering from chronic noncancerous neurogenic pain, long-term satisfactory relief of the allodynic component was obtained in 88.2%, with less satisfactory effects (61%) on those with spontaneous pain.

As MDT was shown to produce a decrease of muscular tone and a diminution of stretch reflexes in the territories of the cord segments operated on, the procedure was applied as early as 1973 to chronic severe spasticity localized on the upper or the lower extremities. Its effects could be explained 1) through an involvement of some Ia fibers placed laterally enough in the DREZ to be interrupted by the MDT operation, and 2)
Somatosensory function after DREZ lesions for pain

![Diagram of fiber organization in the spinal cord](image)

**FIG. 1.** Schematic representation of the fiber organization in the dorsal root entry zone (DREZ) and of the microsurgical DREZ-otmy (MDT) (large black and open arrows). A group \(a\) myotatic reflex arc axon (myot.) is represented by a thick black fiber, two group II axons are drawn open, and six group III and IV axons are represented as thin black fibers. PR = pial ring (the area of junction of central and peripheral glia); TL = tract of Lissauer; DH = dorsal horn; DF = dorsal funiculus; IN = interneuron; MN = mononeuron; SRT = spinoreticular tract; and STT = spinothalamic tract.

by a suppression of the influence of fine, group III and IV, nociceptive fibers on the interneuronal pools, in particular the flexor reflex afferent interneurons. The MDT procedure achieved good or excellent long-lasting effects in 75% to 88% of the patients who underwent surgery for spasticity in the upper\(^1\) or the lower\(^6\) limb(s).

The present work, already presented in preliminary form,\(^20\) has been devoted to evaluating the effects of MDT on spinal cord somatosensory function. For this purpose, we studied a group of 20 patients with spasticity or neurogenic pain who were treated with MDT between September, 1987, and September, 1988, and who underwent both clinical and electrophysiological investigations pre-, intra-, and postoperatively. The physiology was based on the intraoperative recording of somatosensory evoked potentials from determined loci on the spinal cord surface. The spinal cord field potentials, collected either on the skin of the cervical or lumbosacral areas or directly on the spinal cord surface intraoperatively, are here called "evoked electrospinogram," according to Ertelkin.\(^15\) They have been the subject of many fundamental\(^2,3,7,8,25,26,42,46\) and human\(^6,9,10,12,13,15,20-22,28,31,33\) studies.

Although this study is prospective, its pre-established protocol was adapted to each clinical situation, which caused some variability in the methods used. First, the group of patients was heterogeneous, presenting with different, often extensive lesions in the peripheral or central nervous systems. Second, the MDT, which is an open, long-duration operation applied to often-debilitated patients, imposed limitations on intraoperative recording times. We therefore attempted to collect a maximum amount of significant data for each case in a minimum amount of time. The interest of this study resides, consequently, not in the statistical study of a large homogeneous group, but specifically 1) in a variety of studies of spinal cord physiological events as recorded in the immediate vicinity of their generators, 2) in the analysis of the effects of the MDT lesion on these events, and 3) in the study of some revealing clinico-physiological correlations.

**Clinical Material and Methods**

**Patient Population**

This series (detailed in Table 1) included 20 consecutive patients operated on between September, 1987, and September, 1988. There were 10 men and 10 women, whose ages ranged from 21 to 74 years. Thirteen patients suffered from chronic neurogenic pain, affecting the upper limb in seven and the lower limb in six. Five patients presented with chronic severe spasticity, located in the upper limb in two and the lower limb(s) in three. Two patients had a spastic bladder. Etiologies were traumatic in nine cases, multiple sclerosis (MS) in five, postirradiation plexopathies in two, post-herpetic neuralgia in one, intraspinal spontaneous hematoma in one, cerebral abscess in one, and Wilson's disease in one. Clinically, the cervicothoracic cord segments were involved in nine cases, the lower thoracic segments in one, the thoracolumbar segments in one, the lumbosacral segments in seven, and the sacral segments in two.

All patients had intraoperative evoked electrosynaphographic recordings. For practical organizational reasons, only 16 of the 20 patients underwent preoperative skin evoked electrosynaphographic and scalp somatosensory evoked potentials (SSEP's). Only five patients were recorded postoperatively; for the other 15, it was assumed from their intraoperative evoked electrosynaphographic and preoperative physiological assessment that no additional significant information would be collected from postoperative recording.

**Surgical Procedure**

The detailed description of the operative technique for the cervicothoracic\(^17\) as well as for the lumbosacral\(^6\)
### TABLE 1
Pre- and post-MDT somatosensory clinical and electrophysiological data

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Diagnosis &amp; Extent of Lesions</th>
<th>MDT Levels</th>
<th>Preop Skin EESG &amp; SSEP</th>
<th>Intraop EESG</th>
<th>Somatosensory Effects of MDT</th>
<th>Postop Skin EESG &amp; SSEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40, M</td>
<td>Neurogenic pain on avulsion of Lt brachial plexus; atrophy of DR C5, C6, T2; complete avulsion of DR C7–T1</td>
<td>C5–T2 on Lt</td>
<td>silent from lt SL, normal from rt SL</td>
<td>silent from lt SL, normal from rt SL &amp; lt IL</td>
<td>hypealgesia ++ --- hypalgesia ++ --- anesthesia on C-5, C-6, C-7, T-2</td>
<td>---</td>
</tr>
<tr>
<td>2</td>
<td>58, M</td>
<td>Neurogenic pain on avulsion of Lt brachial plexus; atrophy of DR C5; complete avulsion of DR C6–T1</td>
<td>C5–T1 on Lt</td>
<td>silent from lt SL, normal from rt SL</td>
<td>silent from lt SL, normal from rt SL &amp; lt IL</td>
<td>hypealgesia ++ --- hypealgesia +++ on T-2</td>
<td>---</td>
</tr>
<tr>
<td>3</td>
<td>21, M</td>
<td>Neurogenic pain on avulsion of Lt brachial plexus; complete avulsion of DR C4–T1</td>
<td>C4–T1 on Lt</td>
<td>silent from lt SL</td>
<td>unable to monitor during whole MDT, of ascending cord volley on lt cervical fasciculus gracilis, &amp; during stimulation of homolateral trilateral nerve</td>
<td>hypealgesia ++ --- hypealgesia +++ on C-5</td>
<td>---</td>
</tr>
<tr>
<td>4</td>
<td>74, M</td>
<td>Neurogenic pain on rt C5–7 post-herpetic neuraplexia; atrophic DR C-5, C-6</td>
<td>C5–8 on rt</td>
<td>only Pt identifiable at rt SL stimulation</td>
<td>after MDT: maintenance of Pt, maintenance of N, decrease of N,+++, suppression of N, P</td>
<td>dysesthesia &amp; hyperalgesia on C-5-7 (mild hypealgesia &amp; hyperalgesia probably present); proprioception possibly normal</td>
<td>---</td>
</tr>
<tr>
<td>5</td>
<td>69, F</td>
<td>Neurogenic pain on rt postradianation brachial plexopathy</td>
<td>C5–T1 on Lt &amp; cranial half of T-2, on Lt</td>
<td>silent from lt SL, normal from rt SL</td>
<td>silent from lt SL, normal from rt SL</td>
<td>normal</td>
<td>---</td>
</tr>
<tr>
<td>6</td>
<td>59, F</td>
<td>Neurogenic pain on rt postradianation brachial plexopathy</td>
<td>C5–T2 on rt</td>
<td>silent from rt SL</td>
<td>normal on lt side, decreased, prolonged, &amp; hyperalgesia ++ --- hypealgesia + --- anesthesia on C-5</td>
<td>---</td>
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</tr>
</tbody>
</table>

*For all patients, the extent of the caustive lesion, the operated levels, and all recordings and clinical somatosensory examinations performed are displayed. DREZ = dorsal root entry zone; MDT = microsurgical DREZ-cotomy; EESG = evoked electromyogram; SSEP = somatosensory evoked potentials; DR = dorsal root; SL = superior limb; IL = inferior limb; DF = dorsal funiculus; MS = multiple sclerosis; --- = no data available. For wave demaration and the gradient of the effects of MDT in pluses, see text. The column for somatosensory effects of MDT does not include the segments with total preoperative somatosensory losses.*

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TABLE 1 (continued)

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs.)</th>
<th>Diagnosis &amp; Extent of Lesions</th>
<th>MDT Levels</th>
<th>Preop Skin EESG &amp; SSEP</th>
<th>Intraop EESG</th>
<th>Somatosensory Effects of MDT</th>
<th>Postop Skin EESG &amp; SSEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>39, F</td>
<td>neurogenic &amp; causalgic pain on traumatic peripheral nervous lesion (rt middle finger); algoneurodystrophy</td>
<td>C6-8 on rt</td>
<td>normal from rt SL</td>
<td>after MDT: maintenance of ( P_0 ), decrease ++ of ( N_1 ), suppression +++ of ( N_2 )</td>
<td>normal somesthesia except middle finger</td>
<td>from 7th postop day on, normal from rt, SL, except absence of ( N_3 )</td>
</tr>
<tr>
<td>8</td>
<td>41, F</td>
<td>neurogenic pain on missile injury of rt cauda equina; atrophic DR L4-S5</td>
<td>caudal half of L-3, L4-S1 &amp; cranial half of S-2, on rt</td>
<td>silent from rt IL</td>
<td>normal from rt IL</td>
<td>hypalgesia +, hypesthesia +++</td>
<td>analgesia on L-4, anesthesia on L-4</td>
</tr>
<tr>
<td>9</td>
<td>38, M</td>
<td>neurogenic pain on traumatic lesion of rt conus medullaris &amp; cauda equina</td>
<td>L2-S5 on rt</td>
<td>only dorsal root potentials recorded on rt side</td>
<td>hypalgesia ++</td>
<td>hypalgesia +++</td>
<td>analgesia on L-4, anesthesia on L-5, S-1, S-2</td>
</tr>
<tr>
<td>10</td>
<td>46, M</td>
<td>neurogenic pain on extensive traumatic lesions of conus medullaris &amp; cauda equina</td>
<td>L2-S1 on rt</td>
<td>silent from rt IL</td>
<td>after MDT: decrease of ( N_2 ), suppression of ( P )</td>
<td>hypalgesia +++ on L-2, L-3, S-2</td>
<td>unchanged</td>
</tr>
<tr>
<td>11</td>
<td>66, M</td>
<td>neurogenic pain on compressive injury of rt cauda equina; atrophic DR L4-S1</td>
<td>L4-S1 on rt</td>
<td>silent from rt IL</td>
<td>normal on rt with prominent ( N_2 ) delayed, prolonged &amp; low-amplitude negative wave on DF on il, flat recording after MDT</td>
<td>hypalgesia ++ on L-4</td>
<td>unchanged</td>
</tr>
<tr>
<td>12</td>
<td>56, M</td>
<td>neurogenic pain on complete traumatic lesion of spinal cord (T-10)</td>
<td>T-10 corpectomy; MDT T-8, T-9 on both sides</td>
<td>silent from both IL's</td>
<td>delayed, prolonged, &amp; low-amplitude negative wave on DF</td>
<td>analgesia from T-10 down</td>
<td>analgesia from T-8 down</td>
</tr>
<tr>
<td>13</td>
<td>25, F</td>
<td>neurogenic pain on missile injury of rt conus medullaris &amp; cauda equina; atrophic DR L1-S5</td>
<td>T12-L5 on rt</td>
<td>silent from both IL's</td>
<td>delayed, prolonged, &amp; low-amplitude negative wave on DF</td>
<td>analgesia from L-2 down &amp; hypalgesia ++ on L-1</td>
<td>analgesia from T-12 down, anesthesia from L-2 down &amp; hypalgesia ++ on L-1</td>
</tr>
</tbody>
</table>

* For all patients, the extent of the causative lesions, the operated levels, and all recordings and clinical somatosensory examination performed are displayed. DREZ = dorsal root entry zone; MDT = microneurological DREZ-ectomy; EESG = evoked electromyogram; SSEP = somatosensory evoked potentials; DR = dorsal root; SL = superior limb; IL = inferior limb; DF = dorsal funiculus; MS = multiple sclerosis; — = no data available. For wave denomination and the grading of the effects of MDT in pluses, see text. The column for somatosensory effects of MDT does not include the segments with total preoperative somatosensory losses.
cord segments have been published elsewhere. Anesthesia was induced with a short-lasting barbiturate and maintained with isoflurane and nitrous oxide at as low a concentration as possible, with the addition of a narcotic analgesic when needed. Short-lasting curare derivatives were administered only at and just after induction to allow for the identification of motor responses during root or median/tibial nerve stimulation.

The main surgical steps of the procedure are illustrated in Fig. 2. Root identification was performed using radiological and anatomical landmarks and the motor responses to electrical stimulation, produced by a bi-

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<th>Preop Skin EESG &amp; SSEP</th>
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<th>Somatosensory Effects of MDT</th>
<th>Postop Skin EESG &amp; SSEP</th>
</tr>
</thead>
<tbody>
<tr>
<td>14</td>
<td>48, M</td>
<td>harmfull spasticity &amp; plegia of lt SL after rt frontal hemicraniotic lesions</td>
<td>C5-8 &amp; rostral 3/4 of T-1 on lt</td>
<td>normal except low-amplitude N9</td>
<td>after MDT: maintenance of P7 decrease + of N11 decrease ++ of N24 suppression of N2, N5, P</td>
<td>hypalgesia +</td>
<td>low-amplitude N9</td>
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<td></td>
<td></td>
<td>analgesia on C5-T1</td>
<td></td>
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<tr>
<td>15</td>
<td>22, F</td>
<td>harmfull hypertonia of lt SL in Wilson's disease</td>
<td>C5-T1 on lt</td>
<td>--</td>
<td>after MDT: maintenance of P7 decrease + of N11 decrease ++ of N24 suppression of N2 &amp; P</td>
<td>normal</td>
<td>very low-amplitude N9</td>
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<td></td>
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<td></td>
<td></td>
<td></td>
<td>analgesia on C5-T1 &amp; half of T-2</td>
<td></td>
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<tr>
<td>16</td>
<td>42, F</td>
<td>harmfull spasticity on both IL's on MS</td>
<td>L1-S4 on both sides</td>
<td>silent from both IL's</td>
<td>after MDT: disappearance of small N2; decrease ++ of N24 decrease ++ of N2 suppression of P</td>
<td>analgesia &amp; anesthesi</td>
<td>silent from both IL's</td>
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<td></td>
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<td></td>
<td>L1-S5</td>
<td></td>
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<tr>
<td>17</td>
<td>54, M</td>
<td>harmfull spasticity of both IL's on MS</td>
<td>L2-S2 on both sides</td>
<td>silent from both IL's</td>
<td>after MDT: maintenance of P7 decrease of N24 ++ suppression of P</td>
<td>hypalgesia +++ from T-6 down</td>
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<td></td>
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<td></td>
<td></td>
<td>analgesia on caudal part of L-1 &amp; on L2-S5</td>
<td>unchanged from T-6 down</td>
</tr>
<tr>
<td>18</td>
<td>40, F</td>
<td>harmfull spasticity of both IL's on MS</td>
<td>L1-S3 on both sides</td>
<td>--</td>
<td>after MDT: maintenance of P7 suppression of N24, N2, N5, P</td>
<td>hypalgesia ++</td>
<td></td>
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<td></td>
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<td></td>
<td></td>
<td>analgesia on L3-S2</td>
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<td></td>
<td></td>
<td></td>
<td>hypalgesia ++ on L-1, L-2, &amp; S-3, S4</td>
<td></td>
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<tr>
<td>19</td>
<td>64, F</td>
<td>spastic bladder on MS</td>
<td>S2-4 on both sides</td>
<td>--</td>
<td>after MDT: maintenance of N24, N2, N5, P</td>
<td>hypalgesia ++ on S2-5</td>
<td>hypalgesia ++ on S2-5</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>analgesia on S-2</td>
<td></td>
</tr>
<tr>
<td>20</td>
<td>39, F</td>
<td>spastic bladder on MS</td>
<td>S2-5 on both sides</td>
<td>silent from both IL's</td>
<td>after MDT: maintenance of N24, N2, N5, P</td>
<td>hypalgesia ++</td>
<td>hypalgesia ++ on S-2</td>
</tr>
</tbody>
</table>

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Somatosensory function after DREZ lesions for pain

![Operative views showing the four main steps of the cervical microsurgical dorsal root entry zone (DREZ)-otomy (MDT) with exposure of the right dorsolateral aspect of the cervical cord. A: The rootlets of the selected dorsal root (DR) are displaced dorsally and medially with a hook (H) to obtain access to the ventrolateral aspect of the DREZ in the dorsolateral sulcus. Using microscissors (S), the arachnoid adhesions are cut between the cord and dorsal rootlets. DF = dorsal funiculus; DLF = dorsolateral funiculus. B: A thin, sharp bipolar microforceps (F) coagulates exclusively the tiny pial vessels in the lateral border of the dorsolateral sulcus. C: The microscalpel (S) is in position to start the incision, with an angle of 45° to the midline and to a depth of 2 mm. D: The bipolar microforceps (F) coagulates the dorsal part of the dorsal horn, which appears as a darker zone in the depth of the cord incision (arrow). A = aspirator.](image)

A bipolar electrode, with a 2.5-Hz frequency and increasing voltages from 1 to 6 V. Evoked electrospinographic recordings were obtained, as described below, before and after the MDT, and from the same locations.

In the majority of cases, where the dorsal roots were still present, the junction between them and the dorsolateral funiculus on the lateral border of the DREZ was identified and penetrated to a depth of 2 mm and at an angle of 45° with the midline. When the root was avulsed, the dorsolateral sulcus was identified under high magnification and then penetrated down to the dorsal horn, at the same 45° angle but to a depth of 3 to 4 mm. In one case, a cordectomy was performed in addition to the more proximal microsurgical DREZ lesion.

*Intraoperative Evoked Electrospinographic Recording*

The active recording electrode was a silver ball, 750 μ to 1 mm long and 500 to 700 μ wide, which was soldered at the tip of a Teflon-insulated silver wire (Fig. 3). The electrode was placed at the contact of the pial surface of the spinal cord, and was maintained in position by a wet (saline) small cotton pad. The recording positions used in this study were the dorsal funiculus position (Fig. 4A), and a location on the most dorsal part of the lateral funiculus, close to the lateral border of the DREZ, the dorsolateral position (Fig. 4C). The cord segments recorded extended from C4–T1 to from L1–S3, in different cases. The reference used was always noncerebral; the knee was used for lumbosacral cord studies and the shoulder for cervical studies, both contralateral to the stimulated peripheral nerve. Reference electrodes were subcutaneous stainless steel needles 0.4 mm in diameter.

Bipolar stimulation with a proximal cathode was applied 1) to the median nerve at the wrist for cervical evoked electrophysiological recordings, and 2) to the tibial nerve at the ankle (or rarely in the popliteal fossa)
for lumbosacral recordings and for the study of the ascending spinal cord volley at cervical levels. Subcutaneous stainless steel needle electrodes were used. Monophasic square waves with a duration of 0.2 msec were delivered at a frequency of 6 Hz by a constant-current isolated stimulator. The intensities were just above motor threshold and up to three times higher for the study of N20 and N2. This threshold was shown to be significantly variable from one patient to another because of the often extensive neurological and orthopedic lesions affecting them.

Responses were recorded and averaged between 20 and 200 times by electromyogram with a bin width of 137 μsec, analysis times of 60 msec (upper limb) and 90 msec (lower limb), and a filter bandpass of 2 Hz to 2 kHz (6 dB per octave). Peaks were labeled from their polarity and peak latency, according to the Desmedt and Cheron designation, except for the late components for which we have kept the experimental labels N2, N3, and P.

The effects of MDT on the various components of the evoked electrophysiological signals were graded from mild (+), moderate (++), or severe (+++) decrease to suppression. A more precise numerical quantification was not attempted because of large evoked electrophysiological amplitude variations which were probably due to variations in the quality of the contact of the electrodes with the pial surface, or to differences in the exact position of the electrode in relation to the strictly oriented dorsoventral dipoles under study. The possibility of interindividual amplitude variations due to pathology was also considered (see below). The recording conditions in the operating theater are, moreover, well known to be most demanding, and called for a high level of quick adaptability in the midst of a variable and difficult electrical environment. A significant amount of artifactual interference could be controlled by the connection of all reference leads to a common earth and by switching off and, when feasible, unplugging all possible electrical installations during evoked electrophysiological recordings. These amplitude variations of the recorded potentials did not prevent their easy recognition, however, thanks to highly reproducible peak latencies and waveforms.

Pre- and Postoperative Recordings

Preoperative (16 patients) and postoperative (five patients) skin evoked electrophysiological recordings and scalp SSEP's were recorded for upper or lower limbs, respectively, from Erb's point or popliteal fossa, over the C-6 or L-1 spinous processes or corresponding portions of the operative scar, and from contralateral parietal cortex or vertex positions. In this study, these preoperative recordings played a secondary role, as we soon found that they represented only a low-amplitude, low-resolution image of the intraoperative evoked electrophysiological findings. Nevertheless, they allowed a targeted preparation of the intraoperative evoked electrophysiological recording for each patient. In three of the five patients recorded postoperatively, there was an identifiable cortical N20 wave, which allowed study of its post-MDT stability or recovery.

Pre- and Postoperative Clinical Testing

Pain and spasticity were graded as mild (+), moderate (++), marked (+++), or severe (++++) Global results were defined as failure (0% improvement), fair (25%), moderate (50%), good (75%), and excellent (100%), or any value between 0% and 100% if the patient was willing to quantify his postoperative state himself. Decrease of spasticity and spasms, or postural or motor improvements were graded from mild (+), moderate (++), marked (+++), to normalization. The neurological examination included qualitative but detailed somatosensory testing, with an analysis of esthesia (light superficial tactile stimulation of the skin with cotton), algasia (pinprick), pallesthesia or vibratory sense, kinesthesia or position sense, spatial two-point discrimination, and topoesthesia (point localization). The file of each patient included, for each sensory modality, a filled-in map of body segmental innervation displaying the extent of pre- and postoperative sensory deficits. These were graded from light (+) to moderate (++) and severe (++++), down to complete loss of the given modality.

Results

The analysis of our data will be accompanied by the description of some illustrative cases.

Effects of MDT on Neurogenic Pain and Spasticity

As stated in the introduction, this study is an analysis of the effects of MDT on somatosensory function, and not a long-term review of its effects on pain and spasticity. For the sake of completeness, we nevertheless give here and in Table 2 the short-term results of MDT for this series of patients.

For the 13 patients affected with pain (Cases 1 to 13), we have subdivided the various components of neurogenic pain into three categories (Table 2): 1) "spontaneous paroxysmal pain" describes the short, abrupt, and unevoked appearance of multifocal painful sensations (burning, tearing, cutting, and electrical shock); 2) "allodynia" characterizes the appearance of painful sensations evoked by stimuli which do not normally provoke pain; and 3) "permanent pain" describes the presence of continuous dysesthesias, mainly characterized by burning, tearing, compressive, or electrical phenomena.

Table 2 displays the global results, followed by the detailed effects of surgery on the three pain components. At follow-up periods ranging from 2 to 33 weeks, we have noted global improvement between 25% and 100%, and the disappearance of spontaneous paroxysmal pain in 100% of the cases.
Somatosensory function after DREZ lesions for pain

TABLE 2

Symptomatology and results on pain and spasticity

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Symptomatology</th>
<th>Maximum Follow-up Time (wks)</th>
<th>Results at Last Follow-up Examination</th>
<th>Deficits Secondary to MDT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>patients with neurogenic pain</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>SPP ++++, allodynia +++, PP +++++</td>
<td>17</td>
<td>global improvement 75%, suppression of SPP, suppression of allodynia, PP +++++</td>
<td>light distal weakness of Lt IL, no pyramidal signs</td>
</tr>
<tr>
<td>2</td>
<td>SPP +++, allodynia +++, PP +++++</td>
<td>19</td>
<td>global improvement 25%-50%, SPP +++, allodynia +++, PP + to +++</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>no SPP, no allodynia, PP ++++</td>
<td>33</td>
<td>global improvement 98%, suppression of PP</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>SPP +++, allodynia +++, PP +++++</td>
<td>12</td>
<td>global improvement 50%-75%, suppression of SPP, suppression of allodynia, PP +++++</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>no SPP, no allodynia, PP +++</td>
<td>2</td>
<td>global improvement 95%, suppression of PP</td>
<td>light &amp; reversible weakness of Lt IL</td>
</tr>
<tr>
<td>6</td>
<td>SPP +++, allodynia +++, PP +++++</td>
<td>11</td>
<td>global improvement 50%-75%, suppression of SPP, suppression of allodynia, PP + to +++</td>
<td>light &amp; reversible weakness of rt IL</td>
</tr>
<tr>
<td>7</td>
<td>no SPP, allodynia +++, PP +++++</td>
<td>20</td>
<td>global improvement 25%-50%, suppression of allodynia, PP +++++</td>
<td>urinary retention related to liberation of light numerous arachnoidal adhesions on conus medullaris &amp; cauda equina</td>
</tr>
<tr>
<td>8</td>
<td>SPP +++, no allodynia, no PP</td>
<td>16</td>
<td>global improvement 75%-80%, suppression of SPP, appearance of paresthesiae + to ++ on L-3</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>no SPP, no allodynia, PP +++</td>
<td>18</td>
<td>global improvement 80%, PP +</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>no SPP, no allodynia, PP ++++</td>
<td>4</td>
<td>global improvement 100%, suppression of PP</td>
<td></td>
</tr>
<tr>
<td>11</td>
<td>SPP +++, allodynia +++, PP +++++</td>
<td>31</td>
<td>global improvement 25%-50%, suppression of SPP, suppression of allodynia, PP +++++</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>no SPP, no allodynia, PP +++</td>
<td>3</td>
<td>global improvement 50%, PP +</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>SPP +++, no allodynia, PP + to +++</td>
<td>13</td>
<td>global improvement 100%, suppression of SPP, suppression of PP</td>
<td></td>
</tr>
<tr>
<td>patients with spasticity</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>harmful spasticity of Lt SL ++++</td>
<td>6</td>
<td>global improvement 80%, decrease of spasticity &amp; spasm +++, improvement of posture +++, improvement of voluntary motricity ++</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>harmful hypertonia of Lt SL ++++</td>
<td>3</td>
<td>global improvement 75%, decrease of hypertonia +++, improvement of posture +++</td>
<td></td>
</tr>
<tr>
<td>16</td>
<td>harmful spasticity of both IL’s ++</td>
<td>11</td>
<td>global improvement 50%, decrease of spasticity and spasm +++, improvement of posture +++, improvement of voluntary motricity ++</td>
<td></td>
</tr>
<tr>
<td>17</td>
<td>harmful spasticity of both IL’s ++</td>
<td>2</td>
<td>global improvement 80%, decrease of spasticity and spasm +++, improvement of posture +++</td>
<td></td>
</tr>
<tr>
<td>18</td>
<td>harmful spasticity of both IL’s ++</td>
<td>10</td>
<td>global improvement 80%, decrease of spasticity &amp; spasm +++, improvement of posture +++</td>
<td></td>
</tr>
<tr>
<td>19</td>
<td>spastic bladder ++++</td>
<td>13</td>
<td>global improvement 50%, decrease of bladder spasticity ++</td>
<td>postoperative cortisone-dependent increase of IL spasticity (probably due to new MS episode)</td>
</tr>
<tr>
<td>20</td>
<td>spastic bladder +++</td>
<td>11</td>
<td>global improvement 50%-75% decrease of bladder spasticity +++</td>
<td></td>
</tr>
</tbody>
</table>

* For all patients, description of the symptomatology, follow-up times, postoperative results, and neurological complications are presented. DREZ = dorsal root entry zone; MDT = microsurgical DREZ-otomy; SPP = spontaneous paroxysmal pain; PP = permanent pain; SL = superior limb; IL = inferior limb; MS = multiple sclerosis. For the gradation of symptoms, pluses, and %, see test.

With the persistence (in this case minimally distressful) of significant permanent dysesthesiae. Case 3 is at variance with the tendency of this group, showing a subtotal decrease of isolated incapacitating permanent pain.

The seven patients with spasticity (Cases 14 to 20) were clinically assessed according to Sindou and Jeanmonod. The assessment is summarized in Table 2.
under the two items 1) spasticity and spasms, and 2) abnormal postures. The MDT procedure diminished or suppressed spasms, spontaneous as well as provoked by noxious or innocuous stimuli. The global improvement percentages after surgery went from 50% to 80%. These values include the two patients operated on for a spastic bladder. In two patients we noted a moderate improvement of the voluntary motricity of the disabled limb(s). Case 14 illustrates an excellent postoperative outcome of spasticity and abnormal postures, and a significant improvement of the voluntary motor control of the upper limb.

The Normal Evoked Electrospinographic Recording

Figure 4 is a synthetic diagram displaying the different normal cervical evoked electrospinographic events as recorded in various intraoperative recording positions. The P₀ peak is a ubiquitous positive deflection corresponding to a far-field compound action potential originating in the proximal part of the brachial plexus. The P₀ peak is immediately followed by a large negative slow wave, designated N₁₀, on the ascending slope of which dwell a succession of small sharp negative peaks, regrouped under the label N₁₁. These peaks represent near-field presynaptic compound action potentials, probably generated in the proximal portion of the dorsal root, the dorsal funiculus, and the large-diameter afferent collaterals to the dorsal horn. The N₁₃ wave originates from a dorsoventral postsynaptic dipole, generated in laminae IV and V of the dorsal horn during the activation of large-diameter, group I and II afferent fibers. It is followed, at higher stimulus intensities, by the negative N₂ wave, a component of another dorsoventral postsynaptic dipole generated in laminae IV to VI and activated by medium-diameter, group II and III afferent fibers. The latest event, until now only obtained inconsistently and not represented in Fig. 4, is the negative N₃ wave, possibly corresponding to a similarly oriented postsynaptic dipole generated by cells in the most dorsal part of the dorsal horn and in laminae IV–VI during the activation of group III afferent fibers. The P wave is a late prolonged positive deflection which carries the N₂ and N₃ waves. It is the manifestation of presynaptic inhibition on primary afferent fibers.

In addition, the dorsal root position (Fig. 4B) records the compound action potential N₁₀, originating exclusively in the proximal part of the dorsal root and buried in the depth of the P₀ wave. The ipsilateral ventral funiculus position (Fig. 4E) shows a reversal of N₁₀, N₂, and P into P₀, P₂, and N waves, proving the existence of a dorsoventral dipolar generator for these three potentials. The distant rostral position (Fig. 4G) records an ascending spinal cord volley after tibial nerve stimulation. This volley is composed of a long succession of sharp and low-amplitude peaks corresponding to pre- and postsynaptic afferent compound action potentials.

The lumbosacral evoked electrospinographic tracing is similar to the cervical one, the lumbosacral counterparts of the cervical waves being P₀ for P₀, N₁₀ for N₁₁, N₁₃ for N₁₃, and N₂₄ for N₁₄. The other waves are labeled identically in cervical and lumbosacral areas (N₂, N₃, and P).

Practical Use of Evoked Electrospinographic Recordings During MDT

In agreement with other authors,⁹,³¹ we have found the evoked electrospinographic recording extremely useful during surgery in the DREZ. Having studied the peak latencies and baseline-to-peak amplitudes of well-identified cervical N₁₀ and lumbosacral N₂₄ waves (D Jeanmonod, et al., in preparation), we were able to determine the longitudinal distribution of these potentials along the dorum of the spinal cord, characterized by maximum amplitudes at C-7 and C-8 for N₁₃, and at L-5 and S-1 for N₂₄, and a progressive decrease rostrally and caudally from these dominant or “main entry” segments of the stimulated median and tibial nerves respectively (see also Shimooji, et al.⁵³). This knowledge was used to identify, by a minimum of three quick recordings on the dorsal funiculus, the surgically exposed cord segments. The same study performed after MDT allowed assessment of the desired effect of the therapeutic lesions on the postsynaptic activity of the target segments, that they were neither too extensive (respecting, as they should, the neighboring cord segments, Fig. 5) nor too limited (producing a too-modest decrease of the potentials concerned, Fig. 6). It was even possible to leave an electrode in position on the dorsal funiculus during the entire MDT procedure on the recorded segments, thus checking intraoperatively the diminution of N₁₃ (or N₂₄) and the integrity of N₁ (or N₁₃) (Fig. 7).

We also studied intraoperatively the ascending spinal cord volley, as evoked by tibial nerve stimulation and recorded at the cervical level, to assess the preservation of the ascending spinal cord tracts rostral to a cervical MDT operation in cases with cervical root avulsions. Figure 8 demonstrates, in Case 3, the presence of two dominating peaks inside the succession of small and sharp events. Neither the beginning of the volley nor the latency of the first larger sharp peak have shown any significant alterations during the entire C₄–T₁ MDT procedure, which was performed without radical landmarks in this patient with extensive cervical dorsal root avulsions.

Effects of MDT on Somatosensory Examination

Table 1 displays, in the column headed “Somatosensory effects of MDT,” the pre- and postoperative somatosensory examination of all dermatomes, sclerotics, and myotomes affected by the MDT. We chose to omit those segments involved in the operation but presenting with a preoperative total loss of sensation.

We have derived from Table 1 all body segments
Somatosensory function after DREZ lesions for pain

Fig. 4. Artist's diagram displaying a typical normal evoked electrophysiological sample for each one of our intraoperative cervical recording positions. A cervical cord segment is represented, containing one large primary afferent axon from a dorsal root ganglion cell. Its collateral ends in layers IV to VI of the dorsal horn. Another broken axon travels rostrally from the lumbosacral levels in the fasciculus gracilis. A: Dorsal funiculus position, showing the succession of the \( P_0, N_{11}, N_{13}, N_2, \) and \( P \) waves. B: Dorsal root position, with the compound action potential \( N_{10} \) buried in the depth of the \( P_0 \) wave. C: Dorsal position, displaying maximally the succession of sharp peaks composing the presynaptic \( N_{11} \) potential (between the two diverging arrows). D: Lateral funiculus position showing two sharp peaks, possibly \( N_{11} \), followed by a small \( N_{13} \) wave. E: Ipsilateral ventral funiculus position, with a maintenance of \( P_0 \) and probably \( N_{11} \), but a reversal of \( N_{10}, N_2 \), and \( P \) into \( P_{13}, P_2, \) and \( N \) waves. F: Contralateral ventral funiculus position. The \( P_0 \) wave is followed by two sharp peaks, possibly \( N_{11} \), and a small \( P_3 \) wave. G: Distant rostral position, with the ascending spinal cord volley, composed of a long succession of sharp and low-amplitude peaks. The first arrow points to the beginning of the volley, the second arrow to the first of two larger-amplitude peaks. H: Dorsal funiculus position, but more rostral than the entry segment of the axon. Its collateral to the dorsal horn of this rostral level has not been represented. Note the \( N_{13} \) is smaller than in A but \( N_{11} \) is evident. The horizontal bars represent 10 msec, and the vertical bars 10 \( \mu V \) except in G where it is 1 \( \mu V \).

Presenting preoperatively with a normal examination or only a slight deficit, and not situated on the rostral or caudal limits of the operated segments. Four such dermatomes (in three patients) presented postoperatively with a severe (+++) hypalgesia, and nine others (in five patients) with analgesia. Light touch sensation decreased to moderate (+++) hypesthesia on 11 dermatomes (in six patients) and to light hypesthesia on one dermatome. Proprioception was lightly affected (+) on 11 scleromyotomes (in six patients). Discriminatory cutaneous functions were lightly affected (+) on two dermatomes (in two patients). The rest of the body...
segments displayed in Table 1, which were either more heavily affected preoperatively or on the border of the operated segments, showed similar but more variable post-MDT results. In other words, MDT has a severe effect or suppresses nociceptive sensation, affects moderately or lightly the sense of light touch, and has only mild effects on proprioception and cutaneous discriminatory functions.

Case 7 (Table 1), as an example, presented with the complete typical profile of post-MDT somatosensory status. This patient had, at the operative C6–8 levels, analgesia and severe hypesthesia, light distal proprioceptive deficits, and a minimal decrease of spatial cutaneous discrimination.

Electrophysiological Effects of MDT

Table 1 details the evolution of the various intraoperative evoked electrospinographic components after MDT. If MDT affected at least the two main entry segments of the stimulated nerve, changes were as follows (Fig. 9, 1) The cervical far-field compound action potential P2 and the lumbosacral P2 remained unchanged in all six patients in whom they could be identified before MDT. 2) The cervical presynaptic compound action potential N1, remained unchanged in one patient, was lightly diminished in two, and moderately decreased in one. The lumbosacral N2, was much less easily recognized than N1, and the effects of MDT on it could thus not be analyzed. 3) Among the eight patients in whom they could be recorded, the cervical postsynaptic dorsal horn N1, and the lumbosacral N2 potentials were moderately diminished in three, severely decreased in four, and suppressed in one. 4) Among the seven patients in whom they could be recorded, the later events (the N2, N3, and P waves) were all indistinguishable after MDT. One exception was seen in Case 16 (Fig. 6). In the two patients (Cases 19 and 20) in whom the main entry segments of the stimulated nerve were not involved by MDT, the evoked electrospinographic traces remained unaltered (Fig. 5).

In two patients (Cases 6 and 11) with extensive subtotal deafferentation of the recorded segments, we noted the presence of an abnormal delayed, prolonged, and low-amplitude negative wave (N2 in Fig. 10A), followed by a late slow deflection (P2). The MDT left a flat recording in these two situations (Fig. 11). In three patients, we have been able to record the N29 cortical postcentral potential postoperatively (1 week after the MDT). In two of them it was easily identifiable, but
Somatosensory function after DREZ lesions for pain

**Fig. 7.** Intraoperative evoked electrospinographic recordings from Case 4, all on the right dorsal funiculus of cord segment C-7. Right median nerve stimulation at the wrist. A: Recording taken before the microsurgical dorsal root entry zone (DREZ)-otomy (MDT) section was started. Note the N₁ wave, and the presence of two small sharp peaks as N₁. B: Recording obtained after MDT was completed on cord segments C-5 and C-6 and while it was being performed on C-8. C: Recording taken after the MDT at C-8 was completed and while it was beginning on C-7. D: Recording obtained after the Cₕ–8 MDT was complete. Note here the clear-cut final amplitude loss of N₁, but the maintenance of the N₁ sharp peaks on its ascending slope. The _horizontal bars_ represent 10 msec, and the _vertical bars_ 10 µV.

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decreased in amplitude. In the third patient (Case 7, Fig. 12), it was again normal after 1 week and 3 months.

**Discussion**

**Anatomoelectroclinical Correlations and Effects of MDT on Somatosensory Function**

This study has shown that the MDT produced analgesia or severe hypalgesia, moderate hypesthesia, and a slight decrease of proprioception and cutaneous spatial discrimination on the operated body segments. Post-MDT analgesia can be correlated to the interruption of the majority of group III and IV fibers destined for the dorsal horn, mainly laminae I and V. From this interruption will ensue an isolation of these laminae from fine fiber inputs, supposedly interrupting nociceptive inflow in the spinthalamic, spinoreticular, and spino-mesencephalic tracts, known or thought to be important for primate nociception. The subtotal clinical effects (hypalgesia) of MDT could be due to the maintenance after surgery of some particularly medially placed fine fibers, but also of residual intersegmental group III and IV fiber inflow to the operated cord segments, spared by incomplete destruction of Lissauer's tract. These clinical data find physiological support from the suppression after MDT of the late N₃ and N₄ waves, known to correspond to postsynaptic dorsal horn activity after stimulation of the group III fibers. The "suppression" of these two late waves has, however, to be considered critically: 1) they are such slow and low-amplitude events that only a slight decrease might cause them to be lost in the background noise of the recording; and 2) the N₃ wave was found too inconsistently and rarely to allow safe conclusions to be drawn.
Fig. 9. Effects of microsurgical dorsal root entry zone (DREZ)-otomy (MDT) on evoked electrophysio-
graphic recordings: pre- and post-MDT intraoperative samples from two patients with surgery at the cervical 
(Ce) level (A-B and C-D) and two others with surgery at the lumbar-sacral (LS) level (E-F and G-H). 
A and B: Recordings on the left dorsal funiculus on the C-7 and C-5 cord segments, with stimulation of the left median 
nerve at the wrist, before (A) and after (B) a left MDT from C-5 to the rostral three-quarters of T-1. A slight 
decrease of N\textsubscript{11} and a moderate decrease of N\textsubscript{13} are seen; N\textsubscript{2} is indistinguishable. 
C and D: Recordings on the right dorsal funiculus of segment C-7, with right median nerve stimulation at the wrist, before (C) and 
after (D) a right C5-8 MDT. Maintenance of N\textsubscript{13}, and a marked decrease of N\textsubscript{13} are evident. E and F: 
Recordings on the right dorsal funiculus at the junction of segments L-5 and S-1, with right tibial nerve 
stimulation in the popliteal fossa, before (E) and after (F) a right L2-S2 MDT. There is severe decrease of N\textsubscript{24}, 
suppression of P, and maintenance of a very discrete N\textsubscript{21}. G and H: Recordings on the right dorsal funiculus 
on segment L-5, with right tibial nerve stimulation at the ankle, before (G) and after (H) a right L1-S4 MDT. 
A marked decrease of N\textsubscript{24} and N\textsubscript{23} and suppression of P are observed. All horizontal bars represent 10 msec, 
and the vertical bars 10 \(\mu\)V.

from it. It remains a finding to be confirmed by more 
pre- and post-MDT recordings. A marked action of 
MDT on nociception has as a matter of fact also been 
demonstrated by a study of the nociceptive flexion re-
flex, showing its reduction or suppression after MDT.\textsuperscript{18} 
Experimental studies\textsuperscript{15} have shown that the sense of 
touch-pressure is transmitted in primates through many 
pathways, including the dorsal, the dorsolateral, and 
the ventrolateral funiculi. The fact that the MDT pro-
duced a moderate hypesthesia could be explained by 
the physiological data presented in this study. We have 
indeed found a light-to-moderate decrease of the pre-
synaptic compound action potentials penetrating the 
dorsal funiculus (N\textsubscript{i1}), a moderate-to-severe effect on 
the postsynaptic dorsal horn waves related to large pri-
mary afferent fibers (N\textsubscript{13} or N\textsubscript{33}), and a partial or re-
versible decrease of the cortical N\textsubscript{20} wave, the postcen-
tral endpoint of the lemniscal pathway. All of these 
data prove a partial involvement of the touch-pressure 
channels by the MDT, a portion of the large group II 
tactile primary afferents probably being placed laterally 
elsewhere in the DREZ to be caught in the MDT lesion. 
The light effects of MDT on proprioceptive and spa-
tial discrimination functions are explained by the fact 
that the procedure at least partially spares the dorsal 
and dorsolateral funiculi, where vibration and position 
senses have been shown to travel.\textsuperscript{41,65} 
All of these data argue firmly for at least a subtotal, 
and acceptably selective, action of MDT on nociceptive 
mechanisms and structures, but for a partial, often slight 
involvement of the other sensory domains. This evi-
cence is confirmed by post-MDT histological studies of
Somatosensory function after DREZ lesions for pain

Fig. 10. Two examples of pathological evoked electrospinographic waves. A: Delayed, prolonged, and low-amplitude waves (N? and P?) recorded intraoperatively, before microsurgical dorsal root entry zone (DREZ)-otomY (MDT), on the left dorsal funiculus of cord segment L-3 in a patient suffering from neurogenic pain (Case 11). Stimulation of the left tibial nerve at the ankle. B: High-amplitude N2 wave in a spastic patient (Case 16), recorded intraoperatively on the right dorsolateral funiculus of cord segment L-5, before MDT. Stimulation: the right tibial nerve at the ankle. The horizontal bars represent 10 msec, and the vertical bars 10 μV.

Fig. 11. A: Delayed, prolonged, and low-amplitude waves (N? and P?) recorded intraoperatively on the left dorsal funiculus of cord segment L-3 in Case 11 before microsurgical dorsal root entry zone (DREZ)-otomy (MDT) for neurogenic pain. Stimulation: the left tibial nerve at the ankle. B: Recording after the L4-S1 MDT with same recording position and same stimulation as in A. Disappearance of the slow abnormal waves is seen (arrow). The horizontal bars represent 10 msec, and the vertical bars 10 μV.

Anatomoelectroclinical Correlations in Neurogenic Pain

In two cases of marked deafferentations (Cases 6 and 11), delayed, prolonged, and low-amplitude evoked electrospinographic abnormal waves were recorded over the affected cord segments (Fig. 10). These slow waves have been totally silenced by MDT in the two patients (Fig. 11) who both experienced the postopera-

four personal cases (unpublished data), which showed: 1) a destruction of the DREZ and the medial part of Lissauer's tract; 2) a partial lesion of the superficial half of the dorsal horn (laminae I to III) and of the large-diameter primary afferent fibers in the dorsal funiculus; and 3) a preservation of the dorsolateral funiculus and of the deep (IV to VI) dorsal horn laminae.
tive disappearance of their spontaneous paroxysmal pain episodes and allodynia, but the persistence of some permanent pains. More data are needed to determine if the correlation between this slow abnormal evoked electrospinographic event and spontaneous pain paroxysms and allodynia is significant.

Specific deafferentation neuronal hyperactivities have been discovered in the dorsal horn of patients suffering from neurogenic pain,2,19,27 in the dorsal horn and thalamus of a corresponding animal model,1 as well as in the human thalamus.24 Three of the four deafferented hyperactive units that we have personally recorded in human dorsal horns19 were located in the area of laminae IV or V, and one slightly more superficial. On the other hand, we have mentioned above preliminary histological postoperative evidence for a preservation by MDT of the deep half of the dorsal horn, contrary to what was intended by others.30,32,40,47 Thus, MDT might act through the selective and massive interruption, in the ventrolateral part of the DREZ and the medial part of Lissauer's tract, of the remaining excitatory peripheral inputs to the deep dorsal horn. An additional important feature is the concomitant preservation of the inhibitory peripheral and central inputs (large-diameter afferents, lateral part of Lissauer's tract, descending tracts in the dorsolateral funiculus). This "retuning" of the dorsal horn toward inhibition might remain at least partially significant in cases of dorsal root avulsions as well, where even long-distance remaining intersegmental Lissauer's tract inflow could be influenced by MDT in the same therapeutic way.

Another mode of action of MDT could be the destruction of the nociceptive spinothalamic neurons located in lamina I of the dorsal horn. The concomitant surgical destruction of inhibitory interneurons in lamina II, however, does not follow the desired trend of MDT to "retune" the dorsal horn toward inhibition. In addition, the suppression by MDT of the P wave, the manifestation of presynaptic inhibition, can also be seen as an undesired surgical elimination of inhibitory mechanisms. This must be envisaged, however, as the direct consequence of the demonstrated partial moderate-to-strong surgical involvement of group I and II collaterals to the dorsal horn, as shown by the postoperative decrease of N1, and N2. More favorable to the "retuning" hypothesis would be the evidence that fine group III and IV afferent terminals also receive presynaptic endings.

Recent anatomical data indicate a strong predominance (80%) of peripheral afferents in Lissauer's tract.16 These data do not question the dual physiological role of this tract11 as discussed above: the peripheral afferents have indeed been shown to be most concentrated in the medial part of the tract, probably leaving space laterally for rarer axons of dorsal horn intersegmental inhibitory interneurons.

As mentioned in the Introduction and evident from our clinical data, MDT is only partially effective against permanent pain. The mechanisms of this form of pain could well be seated at higher levels than the dorsal horn, for example in the thalamus, thus escaping control by MDT. Conversely, the good response of allodynia after MDT fits well with its supposed spinal mechanisms.

**Electroclinical Correlations in Chronic Spasticity**

As stated in the Introduction, spasticity is usually considered an anarchic hyperactivity of the motoneuronal pool secondary to the suppression of descending inhibitory influences on spinal cord interneurons.4,14 Concerning the mechanisms of action of MDT in spasticity, our results allow us to argue that they are not restricted only to the involvement of some la fibers placed laterally enough in the DREZ to be interrupted by the surgical lesion. A suppression of the influence of fine group III and IV fibers on the operated cord segments must also play a significant role. It was indeed clinically evident that MDT had a suppressive effect on the hyperactive flexion reflexes, as manifested by flexion spasms evoked by noxious cutaneous stimulation. The fact that this was true not only for spasms provoked by noxious but also by innocuous stimuli could be explained by the partial involvement by MDT of larger afferent fibers, as discussed above. We have moreover shown in this study that MDT produces a decrease or a disappearance of the postsynaptic dorsal horn waves recorded on the operated cord segments. Case 16 exhibited evoked electrospinographic recordings with an abnormally large N2 wave before MDT. The effect of the MDT was less marked in this patient than in all others, with an exceptional maintenance of the N2 wave (Fig. 6). This patient then presented with the poorest postoperative result (50% improvement) of our series of spastic patients. Experimental data indicate that suprasegmental lesions of the spinal cord can cause the appearance of increased postsynaptic dorsal horn potentials.17,25,29 These dorsal horn potentials could thus be supposed to manifest at least partly the hyperactivity of spinal cord interneurons. One might thus tentatively conclude that MDT relieves spasticity by silencing hyperactive interneuronal pools in the affected cord segments. In some patients, like Case 16, the abnormal interneuronal activity could be such that an interruption limited mainly to the fine afferent fiber group would be insufficient to bring satisfactory relief of spasticity.

**Conclusions**

The clinical and electrophysiological correlative data presented here indicate that the goal, in performing the MDT procedure, of placing a restricted and specific lesion was achieved. Our results provide evidence for an acceptably selective action of MDT on spinal cord nociceptive mechanisms, and for a partial, often slight, involvement of the other somatosensory domains. Our intraoperative evoked electrospinographic recordings have also given promising insights into the physiopathology of neurogenic pain and spasticity. The hypothe-
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sis of a "returning" of the deafferented or spastic spinal cord segment toward inhibition finds substantial clinical and physiological support in our results.

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