Spinal cord stimulation and thalamic surgery for the treatment of central post–stroke pain

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Abstract

Objective. We studied the surgical results and indications of spinal cord stimulation and thalamic surgery for the purpose of treating central post–stroke pain (CPSP).

Subjects and Methods. CPSP due to unilateral cerebrovascular disease (CVD) lesion was studied in 17 subjects. Pain was localized on the limb (localized type CPSP) in 7 cases and distributed on the hemibody (diffuse type CPSP) in the other 10 cases. All cases of localized type CPSP were treated by epidural spinal cord stimulation. For cases with diffuse type CPSP, stereotactic Vim–Vcpc thalamotomy was performed in 6 cases and thalamic Vim–Vcpc stimulation in 4 cases using depth micro–recording. Evoked pain was dominant in the former and continuous, sometimes intermittent, pain was dominant in the latter. Prior to the operation, we studied somatosensory evoked potentials (SEP) in all cases of localized type CPSP and in 6 cases with diffuse type CPSP treated by thalamotomy. In cases involving thalamic surgery, electrophysiological data obtained during surgery included background neural activity (BNA), sensory response (SR) and burst discharge (burst), and responses to micro–stimulation in the thalamic sensory nucleus.

Results. In 7 cases with localized type CPSP treated by spinal cord stimulation, sufficient pain relief was achieved in 4 cases, moderate in 2 cases and fair in one case. In 10 cases with diffuse type CPSP, good was achieved in 3, moderate in one, fair in 2 out of 6 cases treated by thalamotomy, and good in 2 and moderate in 2 out of 4 cases treated by thalamic stimulation. With the exception of one “fair” respondent to spinal cord stimulation, we recognized definite SEP originating in the sensory cortex (N20). However, SEPs were flat in 4 cases and the amplitude of N20 markedly decreased in the other 2 cases treated by thalamotomy. Using results taken from the electrophysiological study during thalamic surgery, these patients could be classified into 3 groups. In group A (3 cases), we found SR frequently and rarely encountered bursts in a wide area of the thalamic sensory nucleus. In group B (5 cases), we encountered marked burst discharges with high amplitude and of various duration. These were recognized around decreased or voided areas of thalamic neural activity. In 2 cases, which were classified as group C, we found a decrease of BNA, few bursts

and no SR in the sensory thalamus. We found response to micro-stimulation in all groups. Pain control was achieved by thalamic surgery in both group A and B.

Conclusions. Spinal cord stimulation proved to be an effective treatment for localized type CPSP in those cases in which the lemniscal system was preserved. In cases with diffuse type CPSP, we found both dysfunction of the lemniscal system and functional change of the spino-thalamic system. We were able to ameliorate this intractable pain by stereotactic Vim-Vcpc thalamic surgery in those cases in which the spino-thalamic system had been preserved or was hyperactive.

Keywords

Central post-stroke pain; Surgical treatment; Spinal cord; Thalamus

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INTRODUCTION

Recent studies in the field of neuroscience have revealed several important mechanisms of intractable pain. Pharmacological approaches have been used to resolve most pain problems²²⁾. However, treating central pain, particularly central post-stroke pain (CPSP), is difficult. As Dejerine and Roussy made clear in their precise clinico-pathological study²⁾, CPSP can be ascribed to a thalamic vascular lesion mainly on the ventro-postero-lateral thalamic area. Though many hypotheses concerning the factors which cause this intractable pain have been proposed^{1,20)}, the real mechanism of CPSP remains problematic^{13,17)} and systematic studies have been lacking. We carried out epidural spinal cord, thalamic, internal capsule (posterior limb), or precentral cortical stimulation²¹⁾ for the treatment of CPSP^{6,8,9)}.

We examined the pathophysiological mechanism of CPSP using neuroimaging and depth micro-recording. These studies revealed that widespread dysfunction occurred on the thalamus in cases of CPSP, particularly various kinds of functional changes which develop on the relay nuclei around the cerebrovascular disease (CVD) lesion in the sensory thalamus^{4,5,7)}.

In our previous study, we examined the somatosensory evoked potentials (SEP) before surgery and thalamic sensory neuronal activity and response to micro-stimulation during surgery in patients with CPSP¹¹⁾. We found both dysfunction of the lemniscal system and functional change of the spino-thalamic system in these cases. In this paper, we have examined the surgical results and indications of spinal cord stimulation and thalamic surgery for the purpose of treating CPSP based on those electrophysiological findings.

Fig.1 Selection of surgical method for the treatment of patients with central post-stroke pain.

SUBJECTS AND METHODS

Subjects consisted of 17 cases of CPSP due to unilateral CVD lesion. All cases complained of continuous intractable pain. None of a variety of treatments, including analgesic or injection therapy, had proved effective. Pain was localized (localized type CPSP) in 7 cases, and was distributed on the hemibody (diffuse type CPSP) in the other 10 cases with CPSP. In the latter, evoked pain was dominant in 6 cases and continuous, sometimes intermittent, pain was dominant in the other 4 cases. The method of treatment for each CPSP patient was determined according to the localization and the characteristics of pain. For cases with localized type CPSP, we treated this intractable pain by epidural spinal cord stimulation (SCS). For cases with diffuse type CPSP, in which evoked pain (allodynia) was dominant, we performed stereotactic Vim (Ventralis intermedius)–Vcpc (Ventralis caudalis parvocellularis) thalamotomy using depth microrecording^{18,19}. For cases with diffuse type CPSP, in which continuous, sometimes intermittent, pain was

dominant, we carried out thalamic Vim–Vcpc stimulation (Fig.1). Clinical changes in pain intensity were evaluated by visual analogue scale (VAS) and subsequently classified into three stages. Good: VAS improvement was more than 50% of that before treatment. Moderate: VAS improvement was 20–50%. Fair: VAS improvement was less than 20%.

Spinal cord stimulation

SCS was performed in 7 cases with CPSP. Pain was localized in these patients (localized type CPSP); 3 cases on the distal side of the arm and 4 cases on the distal side of the leg, and 3 cases on the left side and 4 on the right. The subjects, 5 males and 2 females, ranged from 50 to 77 years old. Pain was caused by unilateral thalamic CVD lesion in 3 cases, putaminal lesion in 2 cases, and putamino–thalamic or pontine lesion in one case each (Table 1). A wire-type stimulation electrode was implanted, the top of which was around C2 on the spinal dura for cases with arm pain and around Th8–9 for cases with leg pain based on the test stimulation data.

Table 1 Subjects of central post-stroke pain treated by various kinds of surgical method

Case	Age / Sex	Lesion	side	CVD	Pain		Treatment	
1.	54 m	Th	rt	hem	loc.	arm	SCS	S-C
2.	73 f	Th	lt	hem	loc.	leg	SCS	S-T
3.	70 m	Put	lt	inf	loc.	leg	SCS	S-T
4.	77 m	Th	lt	inf	loc.	arm	SCS	S-C
5.	50 f	Pons	rt	hem	loc.	leg	SCS	S-T
6.	57 m	Put	rt	hem	loc.	leg	SCS	S-T
7.	63 m	Th-Put	lt	hem	loc.	arm	SCS	S-C
8.	62 f	Th	lt	hem	dif.	hemi	Th-C	Vim-Vcpc
9.	64 m	Th	lt	hem	dif.	hemi	Th-C	Vim-Vcpc
10.	63 m	Th	rt	inf	dif.	hemi	Th-C	Vim-Vcpc
11.	58 m	Th	lt	hem	dif.	hemi	Th-C	Vim-Vcpc
12.	60 m	Th	rt	hem	dif.	hemi	Th-C	Vim-Vcpc
13.	63 m	Th	rt	hem	dif.	hemi	Th-C	Vim-Vcpc
14.	61 m	Th	rt	hem	dif.	hemi	Th-S	Vim-Vcpc
15.	60 f	Sp	rt	hem	dif.	hemi	Th-S	Vim-Vcpc
16.	68 f	Th	rt	hem	dif.	hemi	Th-S	Vim-Vcpc
17.	65 m	Th	lt	hem	dif.	hemi	Th-S	Vim-Vcpc

Abbreviation: Put: putamen, Sp: spinal cord, Th: thalamus, rt: right, lt: left, hem: intracerebral hemorrhage, inf: cerebral infarct, loc.: localized, dif.: diffuse, hemi: hemibody, SCS: spinal cord stimulation, Th-C: thalamotomy, Th-S: thalamic stimulation, S-C: cervical portion of spinal cord, S-T: thoracic portion of spinal cord, Vim: nucl. ventralis intermedius, Vcpc: nucl. ventralis caudalis parvocellularis

Thalamic surgery

Thalamic surgery was carried out in the other 10 cases with CPSP. Pain was distributed on the hemibody (diffuse type CPSP); 6 cases on the left side and 4 on the right. The subjects, 7 males and 3 females, ranged from 58 to 68 years old. Pain was caused by unilateral thalamic CVD lesion in 9 cases and spinal cord lesion (cavernous angioma) in one case (Table 1). Stereotactic thalamic surgery was performed under local anesthesia. After fixation of a stereotactic frame on the patient's head with pins, we performed an MRI study for target calculation. During the operation, we studied background neural activity (BNA),

sensory response (SR) and burst discharge (burst), or response to micro-stimulation (amplitude 0.1 – 1.0 mA, width 1 milliseconds, frequency 60 Hz, duration 1 second) in the thalamic sensory nucleus. During micro-stimulation on the sensory thalamus, we examined sensory response or change of pain. Referring to the recording data, we made a therapeutic lesion by electrocoagulation using a high-frequency sine wave current generator in the thalamotomy cases. In the thalamic stimulation cases, we implanted a therapeutic electrode on the thalamus. We subsequently connected with the IPG installed on the anterior chest and stimulated the thalamus con-

Table 2 Subjects and results of spinal cord stimulation for cases with localized type central post-stroke pain

Case	Age / Sex	Lesion	CVD	Pain	Duration after op.	Result
1.	54 m	Th rt	hem	arm	8y 2m	<i>good</i>
2.	73 f	Th lt	hem	leg	4y 8m	<i>good</i>
3.	70 m	Put lt	inf	leg	9m	<i>good</i>
4.	77 m	Th lt	inf	arm	3m	<i>good</i>
5.	50 f	Pons rt	hem	leg	4y 10m	<i>moderate</i>
6.	57 m	Put rt	hem	leg	2y 10m	<i>moderate</i>
7.	63 m	Th-Put lt	hem	arm	3y 6m	<i>fair</i>

Abbreviation: Put: putamen, Th: thalamus, rt: right, lt: left, hem: intracerebral hemorrhage, inf: cerebral infarct

tinuously under optimum stimulating conditions. In 3 of these cases, a drug challenge test using ketamine and morphine was additionally performed.

Somatosensory evoked potentials (SEP) study

Before the operation, we studied SEP to contralateral median and posterior tibial nerve stimulation in 7 cases with localized type CPSP and to contralateral median nerve stimulation in 6 cases with diffuse type CPSP, in which evoked pain was dominant. We measured the amplitude and latency of evoked potentials, mainly originating on the thalamus (N13) and sensory cortical area (N20) to contralateral median nerve stimulation or originating on the sensory cortical area (N45) to posterior tibial nerve stimulation. Results were obtained after adding and averaging 200 electrical stimulations on each nerve. The stimulating condition was determined by reference to a flexion of the thumb or the foot joint on the ipsilateral side to electrical stimulation (amplitude 5.0 – 10.0 volt, width 0.2 milliseconds, frequency 2 Hz). We defined the difference of latency and amplitude of SEP (N13, N20 or N45) as below. Delay: The latency during contralateral nerve

stimulation delayed over 2 milliseconds compared to that during ipsilateral stimulation. Decrease: The amplitude of SEP during contralateral nerve stimulation decreased to more than half that recorded during ipsilateral stimulation.

RESULTS

Spinal cord stimulation for localized pain dominant CPSP

Seven cases with localized CPSP treated by epidural spinal cord stimulation, were followed from three months to 8 years and 2 months. Good pain control was achieved in 4 cases, moderate in 2 cases and fair in one case (Table 2). We were able to maintain long-term stable effects in “good” pain control cases, which lasted for 3.5 years mean. Definite SEPs were recognized in 6 successful cases with spinal cord stimulation, which originated on the sensory cortical area. We could not find any obvious difference of SEPs between cases with good and those with moderate pain control. The latency of

Table 3 SEP studies for cases with localized type central post-stroke pain

Case	Age / Sex	Lesion		Pain		SEP (MN)			SEP (pTN)		Op. result
						Thal (N13) latency	SCx (N20) latency	amp	SCx (N45) latency	amp	
1.	54 m	Th	rt	arm	lt	same	delay	dec.	same	same	<i>good</i>
2.	73 f	Th	lt	leg	rt	same	delay	dec.	delay	dec.	<i>good</i>
3.	70 m	Put	lt	leg	rt	same	same	same	same	dec.	<i>good</i>
4.	77 m	Th	lt	arm	rt	same	delay	dec.	n. ex.	n. ex.	<i>good</i>
5.	50 f	Pons	rt	leg	lt	same	same	same	delay	dec.	<i>moderate</i>
6.	57 m	Put	rt	leg	lt	same	delay	dec.	delay	dec.	<i>moderate</i>
7.	63 m	Th-Put	lt	arm	rt	(flat)	(flat)		(flat)		<i>fair</i>

Abbreviation: MN: median nerve, pTN: posterior tibial nerve, Thal, Th: thalamus, SCx: sensory cortex, amp: amplitude, Put: putamen, rt: right, lt: left, dec.: decrease, n.ex.: not examined

The definition of *delay* and *dec.* are explained in the *Subjects and Methods*.

Table 4 Subjects and results of thalamic ablative surgery for cases with diffuse type central post-stroke pain

Case	Age / Sex	Lesion	CVD	Op. Target	Recording resp.	Burst	Duration after op.	Op. result
1.	62 f	Th lt	hem	Vim-Vcpc	●	—	5y 5m	<i>good</i>
2.	64 m	Th lt	hem	Vim-Vcpc	●	—	5y 6m	<i>good</i>
3.	63 m	Th rt	inf	Vim-Vcpc	●	—	1y	<i>good</i>
4.	58 m	Th lt	hem	Vim-Vcpc	●	●	6y 8m	<i>moderate</i>
5.	60 m	Th rt	hem	Vim-Vcpc	—	●	1y 7m	<i>fair</i>
6.	63 m	Th rt	hem	Vim-Vcpc	—	●	1y 10m	<i>fair</i>

Abbreviation: resp.: response, Th: thalamus, rt: right, lt: left, hem: intracerebral hemorrhage, inf: cerebral infarct, Vim: nucl. ventralis intermedius, Vcpc: nucl. ventralis caudalis parvocellularis

The size of closed circle in the column of recording indicated the frequency of sensory responses or burst discharges which were encountered during the surgery.

evoked potentials (N20 or N45) to contralateral corresponding nerve stimulation, which innervates the body part suffering intractable pain, delayed or its amplitude decreased (Table 3).

Thalamotomy for evoked pain dominant CPSP

In 6 thalamotomy cases with diffuse type CPSP followed from one year to 6 years and 8

months, in which evoked pain was dominant, good pain control was achieved in 3 cases, moderate in 1 case and fair in 2 cases (Table 4). Before surgery, SEP study showed that the evoked potentials (N13 and N20) were not recognized (flat) during contralateral median nerve stimulation in 4 out of 6 cases. In the other 2 cases, the latency of N20 delayed or its amplitude decreased. There appeared to be no relationship

Table 5 SEP studies for cases with diffuse type central post-stroke pain

Case	Age / Sex	Lesion	CVD	Pain	SEP (MN)			Op. result
					Thal (N13) latency	SCx (N20) latency	amp	
1.	62 f	Th lt	hem	h. rt	(flat)	(flat)		<i>good</i>
2.	64 m	Th lt	hem	h. rt	(flat)	(flat)		<i>good</i>
3.	63 m	Th rt	inf	h. lt	(flat)	(flat)		<i>good</i>
4.	58 m	Th lt	hem	h. rt	same	delay	dec.	<i>moderate</i>
5.	60 m	Th rt	hem	h. lt	same	same	dec.	<i>fair</i>
6.	63 m	Th rt	hem	h. lt	(flat)	(flat)		<i>fair</i>

Abbreviation: MN: median nerve, Thal, Th: thalamus, SCx: sensory cortex, amp: amplitude, rt: right, lt: left, hem: intracerebral hemorrhage, inf: cerebral infarct, dec.: decrease

The definition of *delay* and *dec.* are explained in the *Subjects and Methods*.

between the SEP findings and operative results (Table 5). As electrophysiological details have been previously reported¹¹⁾, we have mentioned those findings only briefly in this paper. These cases could be classified into 3 groups. In group A (3 cases), we found SR frequently and rarely encountered bursts in a wide area of the thalamic sensory nucleus. Micro-stimulation on the thalamus evoked paresthesia on the corresponding body part, but somatotopic distribution of the perceptive field was narrower than that of the receptive field to peripheral natural stimulation. In group B (1 case), we encountered frequent bursts, which were recognized around decreased or voided areas of thalamic neural activity. We found SR in a limited area of the thalamic sensory nucleus. Micro-stimulation on the thalamus also evoked paresthesia on the corresponding body part, and somatotopic distribution of the perceptive field was also narrower than that of the receptive field (group B-1). In the other 2 cases, which were classified into group C, we found a decrease of BNA, few bursts, and no SR in the sensory thalamus. However, micro-stimulation on the thalamus evoked paresthesia on the corresponding body part with organized soma-

totopic distribution of perceptive fields. We were able to achieve good pain control in group A but were not in group C by thalamotomy.

Thalamic stimulation for continuous or intermittent pain dominant CPSP

In 4 thalamic stimulation cases with diffuse type CPSP followed from 6 months to 3 years and 2 months, in which continuous or sometimes intermittent pain was dominant, good pain control was achieved in 2 cases, and moderate in 2 cases (Table 6). Electrophysiological study showed thalamic BNA increasing rather than being preserved in all 4 cases. We encountered frequent bursts, which were recognized around the decreased or voided areas of thalamic neural activity (group B). In one of these cases, we found SR in the limited area of the thalamic sensory nucleus (group B-1). In this case, studied using two (A and B) electrodes 3 mm apart each during surgery, marked burst discharges with high amplitude and of various duration were frequently recognized and SR was rare in the bottom of the thalamus on the anterior A electrode. On the posterior B electrode, neural activity was almost silent at the dorsal to middle

Table 6 Subjects and results of thalamic electrical stimulation treatment for cases with diffuse type central post-stroke pain

Case	Age / Sex	Lesion	CVD	Op. Target	Recording resp.	Burst	Duration after op.	Op. result
1.	61 m	Th rt	hem	Vim-Vcpc	●	●	1y 8m	<i>good</i>
2.	60 f	Sp rt	hem	Vim-Vcpc	—	●	1y	<i>good</i>
3.	68 f	Th rt	hem	Vim-Vcpc	—	●	3y 2m	<i>moderate</i>
4.	65 m	Th lt	hem	Vim-Vcpc	—	●	6m	<i>moderate</i>

Abbreviation: resp.: response, Sp: spinal cord, Th: thalamus, rt: right, lt: left, hem: intracerebral hemorrhage, Vim: nucl. ventralis intermedius, Vcpc: nucl. ventralis caudalis parvocellularis

The size of closed circle in the column of recording indicated the frequency of sensory responses or burst discharges which were encountered during the surgery.

part and very low in the bottom part of the thalamus. We were unable to find SR in the thalamic sensory nucleus in the other 3 cases (group B-2). In one of these 3 cases, marked burst discharges of various duration were frequently recognized on the dorsal part of the sensory thalamus. In both cases, insertion of the therapeutic electrode without stimulation resulted in a decrease of the intractable pain. Although pain recurred a few days later, it was successfully controlled by electrical stimulation. We were able to achieve moderate pain control in the other 2 cases. In 3 cases of thalamic stimulation, a drug challenge test using morphine and ketamine showed that they were not available for pain relief. One patient, however, was slightly satisfied by using ketamine.

DISCUSSION

Previous reports have found spinal cord stimulation to be ineffective in the treatment of CPSP¹⁴⁾. However, our study revealed that relatively long-term and stable positive effects were

possible with SCS in limited cases of CPSP. In particular, positive effects were obtainable in those cases in which pain was distributed on a localized body area (localized type CPSP). In cases for which spinal cord stimulation was successful, we were able to recognize definite SEP originating in the sensory cortex ipsilateral side to the CVD lesion. We could not find an obvious difference of SEPs between cases with good and those with moderate pain control. Our previous study suggested that spinal cord stimulation was effective for cases with localized type CPSP, in which the sensory function had been preserved¹⁰⁾. This study revealed that in effective cases, the lemniscal function appeared to have been preserved¹²⁾. Spinal cord stimulation was considered to activate spinal posterior funiculus orthodromically, to relay on the brain stem, sensory thalamus, cerebral cortex, or on the descending pain inhibition system, resulting in the inhibition of nociceptive input from the peripheral receptors¹⁵⁾. Several studies have shown that the lemniscal system modulates the spinothalamic system on the thalamic level¹⁶⁾. Spinal cord stimulation may reduce pain by affecting the function of supraspinal structures, particularly that of the thalamus, through the

lemniscal system¹⁵⁾. Therefore, for spinal cord stimulation to ameliorate CPSP, it is essential that the lemniscal function is preserved. Although CPSP is a highly complex dysfunction which is often resistant to usual pain therapies, spinal cord stimulation proved to be effective in limited cases with localized type CPSP, in which SEP or spinal cord test stimulation revealed that the lemniscal function had been preserved.

Our study suggested that severe dysfunction of the lemniscal system and mild to moderate dysfunction of the spinothalamic system occurred in cases with diffuse type CPSP. In cases with diffuse type CPSP treated by thalamotomy, SEP study showed that evoked potentials on the side of CVD lesion were not recognized (flat) or the latency of N20 delayed and its amplitude decreased during contralateral median nerve stimulation. It was proved that remarkable damage occurred in the lemniscal system, which differs markedly from those cases with localized type CPSP. It was suggested that the majority of the VC nucleus had suffered remarkable damage by CVD lesion, which was the main terminal nucleus of the lemniscal system in the thalamus. We could not expect to ameliorate diffuse type CPSP by spinal cord stimulation because it appeared unable to affect the function of supraspinal structures through the lemniscal system. Micro-recording study on the lateral thalamus during thalamic surgery (thalamotomy and thalamic stimulation) showed the functional changes of the spinothalamic system in cases with diffuse type CPSP. These cases could be classified into 3 groups. Group A: The lateral spino-thalamic pathway was regarded as slightly irritated and showed mild functional change. We found SR frequently and rarely encountered bursts in the wide area of the thalamic sensory nucleus. Group B: The lateral spino-thalamic pathway was regarded as remarkably irritated

and showed moderate to severe functional change. We encountered bursts frequently and found SR in the limited area of the thalamic sensory nucleus or did not. Particularly, in cases treated by thalamic stimulation, we found an area of marked burst discharges with occasional high amplitude and with slow oscillation in the sensory thalamus. In groups A and B, the perceptive field to micro-stimulation in the thalamus was not compatible with the receptive field to peripheral natural stimulation. Group C: The lateral spino-thalamic pathway was regarded as slightly irritated and showed a decrease of sensory function. We found a decrease of BNA, few bursts, and no SR to peripheral natural stimulation in the sensory thalamus. However, we could find sensory effects to micro-stimulation in the thalamus. These findings revealed that the sensory thalamic function had not been completely destroyed in cases of group C.

In the evoked pain or allodynia dominant cases in the group of diffuse type CPSP, pain was evoked by peripheral natural stimulation. Ascending sensori-motor signals from the periphery were relayed on the Vim nucleus in the thalamus^{18,19)}. Abnormal reception or misconduction of the sensori-motor impulses of the spino-thalamic system may have developed due to functional change of the Vim nucleus on the lateral sensory thalamus, resulting in the genesis of movement-related pain. This intractable pain may be ameliorated by the treatment of dysfunction of the thalamic Vim nucleus around the CVD lesion. In the continuous or sometimes intermittent pain dominant cases in the group of diffuse type CPSP, electrophysiological study suggests that marked burst discharges with occasional high amplitude and slow oscillation, which was recognized on the Vim or Vcpc nucleus around CVD lesion in the sensory thalamus, played an important role in the genesis

of diffuse type CPSP. Pain appeared in a spontaneous fashion, but was not evoked by peripheral natural stimulation. Spontaneous or intermittent excitation of the Vim or Vcpc nucleus in the lateral sensory thalamus may develop due to functional changes of these nuclei, resulting in the genesis of continuous or sometimes intermittent pain. Though it was transient, pain was ameliorated or disappeared during surgery, immediately after implantation of the therapeutic electrode on the irritable thalamus. Pain control was achieved by therapeutic stimulation on the area of marked burst discharges with slow oscillation in the sensory thalamus. Though the role of bursting activity mediating central neuropathic pain is unclear³⁾, this study suggests that dysfunction or hyperactivity at the spino-thalamic terminal nucleus on the thalamus (Vim or Vcpc nucleus) around CVD lesion is one of the important factors in the genesis of diffuse type CPSP. We have been able to ameliorate this intractable pain by reducing the function of abnormal sensory nuclei or modulating hyperactive sensory nuclei directly at the thalamic level. Indeed, pain control was achieved for a long time in cases with diffuse type CPSP by making a therapeutic lesion on the functionally changed area or by stimulation on the area of marked burst discharges with slow oscillation in the lateral sensory thalamus.

CONCLUSION

We were able to ameliorate localized type CPSP by spinal cord stimulation in cases in which the lemniscal system appeared to have been preserved. Spinal cord stimulation was considered to be the first treatment for localized

type CPSP. We found both dysfunction of the lemniscal system and functional change of the spino-thalamic system in cases with diffuse type CPSP. These functional changes appeared to affect perception or conduction of sensory impulses in the lateral sensory thalamus, resulting in the genesis of CPSP. We were able to ameliorate this intractable pain by stereotactic Vim-Vcpc thalamic surgery in those cases in which the spino-thalamic system had been preserved or was hyperactive.

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