INTRODUCTION

“Frau R. Suicidium.” (Edinger 1891)

DEFINITIONS

Ever since Dejerine and Roussy’s description of central pain (CP) after thalamic stroke in 1906, thalamic pain (itself part of the thalamic syndrome) has remained the best-known form of CP and it has often — misleadingly — been used for all kinds of CP. Since CP is due to extrathalamic lesions in the majority of patients, this term should be discarded in favor of the terms central pain of brain—brainstem or cord origin (BCP and CCP). Other terms that are now obsolete and should be discarded include pseudothalamic pain (i.e., CP caused by extrathalamic lesions) and anesthesia dolorosa, when this refers to CP in an anesthetic region caused by neurosurgical lesions. If a stroke at whatever level is the cause of CP, the term central post-stroke pain (CPSP) is used. Even though some clinical features are similar, peripheral neuropathic pain (PNP) is not CP.

CP is akin to central dysesthesias/paresthesias (CD) and central neurogenic pruritus (CNP): actually, these are facets of a same disturbance of sensory processing following central nervous system (CNS) lesions. Dysesthesias and paresthesias differ from pain in their being abnormal unpleasant and non-unpleasant sensations with a nonpainful quality. While contributing to suffering, they can also be found in PNP. Dyesthetic pain used as a synonym of CP must also be abandoned.

Since 1978 there has been a tendency to combine CP and PNP under the general rubric of deafferentation pain on account of “shared clinical features,” both being due to a decrease in afferent input into the CNS and consequent sensitization (see Tasker 2001). Deafferentation pain has never been included in the taxonomy published by the International Association for the Study of Pain (IASP) (Merksey and Bogduk 1994), and actually indiscriminate lumping of all neuropathic pains under this term
has created much confusion and even contradictions, often hindering assessment of therapeutic strategies for single disease entities. The term neural injury pain should also be discarded. In 1990 a consensus group (Devor et al. 1991) concluded that: “The term ‘deafferentation pain’ as presently used is misleading and should perhaps be abandoned altogether for purposes of clinical diagnosis.”

Virtually all kinds of slowly or rapidly developing disease processes affecting the spino- and quintothalamic pathways (STT/QST), i.e., the pathways that are most important for the sensibility of pain and temperature, at any level from the dorsal horn/sensory trigeminal nucleus to the parietal cortex, can lead to CP/CD/CNP. These do not depend on continuous receptor activation.

The IASP defines CP as “pain initiated or caused by a primary lesion or dysfunction of the central nervous system” (Merksey and Bogduk 1994), i.e., of the spinal cord, brainstem or cerebral hemispheres. This definition is too extensive, as it includes pain associated with motor disorders (Parkinson’s disease and dystonia) and painful fits, which — although being CNS disorders — are not strictly CP: impairment of spinothalamocortical conduction, a cardinal finding of CP, is not seen in these conditions. However, there are cases of bona fide CP without clinical or electrophysiological signs of such impairment. We propose that CP/CD/CNP be considered only “spontaneous, constant and/or evoked pain, dysesthesia or pruritus initiated by a CNS lesion impinging on or interfering with the spinothalamoparietal path.” Since CP appears to be the most frequent of these three conditions, we will generally refer to CP throughout the text. Parkinson’s disease, epileptic pains and perhaps other diseases with a painful CP-like component should be classified as central pain-allied conditions (CPAC).

Once thought an uncommon neurological curiosity, CP is an important and underrecognized condition. CP produces immense suffering (“a great burden”), even when intensity is low: its generally very unpleasant and irritating, largely constant character makes it incomprehensible by almost all sufferers. Patients can be completely disabled and CP may be so devastating as to override any other disability in the chronic stage. By dominating the sensorium, interfering with the thought processes and undermining the morale, CP frequently alters mood, intellect and behavior with deterioration of personality, depression and neurotic tendencies, interfering with rehabilitation, and impairing daily activities and quality of life. Many patients with severe persistent pain undergo a progressive physical deterioration caused by disturbance of sleep and appetite, a restriction in physical and daily activities, and often become addicted to medications, all of which contribute to general fatigue, increased irritability and decreased libido and sexual activity. The social effects are equally devastating: many patients have progressively greater problems with their families and friends, reduce their social interactions and activities and are unable to work (Widar et al. 2004). There are hints that chronic pain may suppress the immune system and even alter insulin sensitivity. Some patients with severe persistent pain become so discouraged and desperate that they commit suicide, and usually not because of depression. Last, but not least, CP financially burdens both society and patients. Thus, it represents a true challenge.
"Those who cannot remember the past are condemned to repeat it." (G. Santayana)

Cases of CP following brain or cord damage have most certainly been observed since antiquity, but never understood as such. We have to wait until the nineteenth century for published descriptions of what we now understand to be CP (Table 1.1) in western medicine (there appear to be reports of what is most likely CP in ancient Chinese medicine, this being the result of a deficiency of the Qi and attendant blood stasis, in turn depriving the nourishing of meridians and tendons; see Kuong 1984).

**TABLE 1.1. Historic highlights of central pain in the western literature (from Garcin 1937; DeAjuriaguerra 1937)**

<table>
<thead>
<tr>
<th>Year</th>
<th>Author</th>
<th>Description</th>
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<tbody>
<tr>
<td>1811</td>
<td>Marcet</td>
<td>Describes pain after bulbar lesions</td>
</tr>
<tr>
<td>1822</td>
<td>Fodera</td>
<td>Describes pain after spinal hemisection</td>
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<tr>
<td>1850</td>
<td>Brown-Sequard</td>
<td>Describes the syndrome named after him; confirms previous description of hyperesthesia below lesion level on the plegic side</td>
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<tr>
<td>1860—70s</td>
<td></td>
<td>Descriptions of pain after spinal trauma during the U.S. Civil War</td>
</tr>
<tr>
<td>1875</td>
<td>Marot</td>
<td>Further describes pain after bulbar lesions</td>
</tr>
<tr>
<td>1879</td>
<td>Nothnagel</td>
<td>First precise description of constant pain following tumors of the pons Varolii (mentioned by other authors) and other sites</td>
</tr>
<tr>
<td>1883</td>
<td>Page</td>
<td>Describes pain in spinal cord injury patients</td>
</tr>
<tr>
<td>1891</td>
<td>Edinger</td>
<td>Birth of the concept of central pain</td>
</tr>
<tr>
<td>1891</td>
<td>Hardy</td>
<td>Describes pain of cortical origin</td>
</tr>
<tr>
<td>1892</td>
<td>Mann</td>
<td>Matches CP to infarctions of medulla at nucleus ambiguous level</td>
</tr>
<tr>
<td>1889</td>
<td>Gilles de la Tourette</td>
<td>Describes syringomyelic pain</td>
</tr>
<tr>
<td>1895</td>
<td>Wallenberg</td>
<td>Describes the syndrome named after him; insisted on facial pains; ascribed it to PICA embolism (verified autopathically in 1901)</td>
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<tr>
<td>1897</td>
<td>Reichenberg</td>
<td>Describes CP as resulting from parietal stroke (autopsy confirmed)</td>
</tr>
<tr>
<td>1899</td>
<td>Link</td>
<td>Describes CP as resulting from pontobulbar lesions</td>
</tr>
<tr>
<td>1906</td>
<td>Dejerine and Roussy</td>
<td>Describe the syndrome named after them</td>
</tr>
<tr>
<td>1919</td>
<td>Holmes</td>
<td>&quot;Typical thalamic pain&quot; observed in spinal cord injured patients (World War I soldiers)</td>
</tr>
<tr>
<td>1910, 1920s—30s</td>
<td>Souques, Guillain and Bertrand, Davison and Schick, Schuster, Wilson, Parker</td>
<td>Autopic confirmation that CP may arise without thalamic involvement</td>
</tr>
</tbody>
</table>

*A great many authors described CP, but dates are not available through the two cited reviews: these authors include Halische, Joly, Duchek, Biemacki, Oppenheim, Bechterew (pre-1900), Bamm, Elsberg (cordonal pain), Foerster (dorsal horn pain), Vulpian, Gowers, Gerhardt (recognized CP in multiple sclerosis), Schlesinger, Lhermitte and DeMassary-Bonhomme (hematobulbia), Mills, Mattiolo, Hansen, and many others.
However, the possibility of centrally arising pains was simply dismissed by most authorities.

It was only in 1891 that Edinger, a German neurologist, challenging the prevailing opinion of the day, and “avec une rare sagacité” (with rare sagacity; Garcin 1937), introduced the concept of centrally arising pains. In his landmark paper “Giebt es central entstehende Schmerzen? Mittheilung eines Falles von Haemorrhagie in den Nucleus externus Thalami optici und in das Pulvinar, dessen wesentliche Symptome in Hyperaesthesie und furchtbaren Schmerzen in der gekreuzten Seite, ausserdem in Hemiatherose und Hemianopsie bestanden haben” (Are there centrally arising pains? Description of a case of bleeding in the nucleus externus thalami optici and in the pulvinar, whose essential symptom consisted in hyperesthesia and terrible pains in the contralateral side, besides hemiathetosis and hemianopsia), he remarked how only a few cases of pains associated with damage of the brain, brainstem and spinal cord were on record (“Die Durchsicht der Literatur nach aehnlichen Beobachtungen hat nur wenig ergeben” — A literature review of similar cases has borne little fruit), but that other reasons were adduced to explain them (generally peripheral nerve causes or muscle spasms). One of the few “well investigated” cases was that of Greiff (1883), concerning a 74-year-old woman who developed “Hyperaesthesie und reissenden Schmerzen im linkem Arm, geringgradiger im linkem Beine” (hyperesthesia and tearing pains in the left arm and of lesser intensity in the left leg) as a consequence of several strokes and which lasted for two months until death. At autopsy, two areas of thalamic softening were found, one of which was in what appears to be ventrocaudalis (Vc). Greiff commented on vasomotor disturbances as a possible cause of pain. According to Edinger “Vielleicht giebt es auch corticale Schmerzen” (perhaps there are also cortical pains), and he cited as evidence “…schmerzhaften Aura bei epileptischen, abnorme Sensationen bei Rindenherden und Reizerscheinungen im Bereich des Opticus bei Affektionen des Hinterhaupts-lappens” (…painful aura in epileptics, abnormal sensations in cortical foci and signs of excitation in the territory of the opticus following diseases of the occipital lobe). Edinger reported on “einen Krankheitsfall…in dem als Ursache ganz furchtbaren Schmerzen post mortem ein Herd gefunden wurde, der dicht an die sensorische Faserung grenzend im Thalamus lag. Der Fall erscheint dadurch besonders beweiskraftig fuer die Existenz 'centraler Schmerzen', weil die Hyperaesthesie und die Schmerzen sofort nach dem Insulte und monatelang vor einer spater auftretenden Hemichorea sich zeigten” (a patient…in whom the origin of truly terrible pains was at autopsy a lesion that impinged on the fibers abutting the thalamus. This case is thus especially convincing evidence for the existence of “central pains,” as the hyperesthesia and the pains showed immediately after the insult and months before a later arising hemichorea). The patient was “Frau R.” (Mrs. R.), aged 48, who developed “heftige Schmerzen und deutliche Hyperaesthesie in den gelaehmten Gliedern” (violent pains and clear-cut hyperesthesia in the paretic limbs: right arm and leg); “Wegen der furchtbaren Schmerzen Suicidium 1888” (due to the terrible pains, suicide 1888). This woman developed an intense tactile allodynia for all stimuli bar minimal, which hindered all home and personal activities (e.g., dressing) and made her cry; also “Laues wasser wurde als sehr heiss, kaltes als unertraeglich schmerzend” (lukewarm water was felt as very hot, and cold water as intolerably painful) in both limbs. Very high doses
of “Morphium” were basically ineffective. This patient's pain reached intolerable peaks, but sometimes could be tolerated for a few hours or at most half a day before shooting up again. In this patient, “Vasomotorische Stoerungen, wie sie in dem Lauenstein (D.Arch.f.klin.Med. Bd XX u. A.)'schen... Falle bestanden haben, sind nicht zur Beobachtung gekommen” (vasomotor disturbances, as present in Lauenstein's case, were nowhere to be observed). At autopsy, “Der Herd im Gehirn nimmt also den dorsalen Theil des Nucleus externus thalami und einen Theil des Pulvinar ein, er erstreckt sich lateral vom Pulvinar fuer 1 mm in den hintersten Theil der inneren Kapsel hinein. Der Faserausfall, der dort in Betracht kommt, ist sehr gering.” (The brain lesion involved the dorsal portion of the nucleus externus thalami and a portion of the pulvinar, extending laterally from pulvinar for 1 mm into the most posterior part of the inner capsule. The loss of fibers, that can be observed at this point, is minimal.) Thus, in Greiff's and Edinger's patients lesions were respectively found at autopsy in right thalamic nucleus internus and ventral thalamus and in thalamic nucleus externus and pulvinar.

Edinger should be given the credit as the one who introduced the concept of CP to neurology, as he wrote: “Man kommt zum Schlusse, dass hier wahrscheinlich durch directen Contact der sensorischen Kapselbahn mit erkranktem Gewebe die Hyperaesthesie und die Schmerzen in der gekreuzten Koerperhaelfte erzeugt worden sind” (one concludes that here both the hyperesthesia and the pains in the crossed half of the body have been likely caused by direct contact of injured tissue with the sensory path coursing in the internal capsule), actually being the first to propose an irritative theory of CP. Incidentally, he stressed the importance of the internal capsule, a forerunner of our theory (Canavero 1994).

One year later, Mann (1892), another German neurologist, concluded, in Edinger's wake, that CP can be also observed outside the thalamus, namely in the medulla oblongata, thus antedating Wallenberg's classic description (autopsy of this patient performed in 1912 confirmed Mann’s clinical diagnosis and the involvement of the spinothalamic tract). Thereafter, an explosion of reports ensued. In the first decade of the twentieth century, Dejerine and Roussy (1906) described six cases of what they called “Syndrome thalamique,” whose signs and symptoms were summarized by Roussy (1906) in his thesis:

1) slight hemiparesis usually without contracture and rapidly regressive;
2) persistent superficial hemianesthesia of an organic character which can in some cases be replaced by cutaneous hyperesthesia, but always accompanied by marked and persistent disturbances of deep sensations;
3) mild hemiataxia and more or less complete astereognosis.

To these principal and constant symptoms are “ordinairement” (ordinarily) added:

1) severe, persistent, paroxysmal, often intolerable pain on the hemiparetic side unyielding to any analgesic treatment;
2) choreoathetotic movements in the limbs on the paralyzed side.

On the basis of an autopsy study of three cases, they concluded that the lesion is localized to the external, posterior and inferior region of the thalamus (thus including the main sensory nucleus Ventrocaudalis, or Vc), impingings on the median
nuclei and, to a lesser extent, involves a part of the posterior limb of the internal capsule. Certainly, the complete syndrome is very rare. In their original paper on the syndrome thalamique, Dejerine and Roussy evaluated microscopically the thalamic lesion responsible for the syndrome. In their first case they noted a lesion in the posterior thalamus, involving both external and internal nuclei and the internal capsule. The lesion impinged more diffusely on the external thalamic nucleus. In their second case the lesion again impinged more on the external thalamic nucleus, but they also noted the lesion of the internal and median nuclei, internal capsule and pulvinar. The lesion also impinged on the posterior pulvinar. In their third case a less extended lesion was noted, impinging on the posterior part of the thalamic external nucleus, the internal and median nuclei, the posterior part of the internal capsule and part of the lenticular nucleus. They concluded that the thalamic syndrome follows a lesion of the postero-external part of the external thalamic nucleus, impinging also on part of median and internal thalamic nuclei and on the near part of the internal capsule.

A few years later, Head and Holmes (1911), on the basis of personal and literature autopic evidence, concluded that thalamic pain depends on the destruction of the posterior part of the external thalamic nucleus. In their book-size article, they provide the best and first quantitative description ever of somatosensory alterations in CP patients (Chapter 2).

During World War I several observations on “thalamic pains” associated with spinal cord war lesions were published, as previously done during the U.S. Civil War in the 1860s.

The term central pain was first used in the English literature by Behan (1914). In 1933 Hoffman reported a tiny lesion in the most basal part of the Vc, where spinothalamic fibers end (Hassler’s Vcpc). This is probably the smallest reported lesion causing CP.

In the 1930s three major reviews on CP were published (De Ajuriaguerra 1937; Garcin 1937; Riddoch 1938). Here, the interested reader will find an unparalleled review of the literature of the nineteenth and early twentieth centuries, plus unsurpassed descriptions of CP, whose ignorant neglect (admittedly also out of language barriers) on the part of modern investigators is responsible for several “re-discoveries.” Nothing new has been basically added to the clinical literature since.

Riddoch gave this definition: “By central pain is meant spontaneous pain and pain-ful overreaction to objective stimulation resulting from lesions confined to the sub-stance of the central nervous system including dysesthesiae of a disagreeable kind.”

It was clear how “thalamic pains” could follow a lesion of the lateral thalamic area, in the territories of the lenticulo-optic, thalamo-geniculate and thalamo-perforating arteries, but also of the cortex (rarely), internal capsule, medulla oblongata and less frequently the pons (no mesencephalic lesions were on record) and the spinal cord (not infrequently; particularly following injury and syringomyelia). Thermoalgesic sensory loss and somatotopographical constraints were clearly delineated. However, De Ajuriaguerra, based on a patient with a thalamic lesion and CP without sensory derangement described by Lhermitte, concluded against a role of the sensory relay nuclei in the genesis of CP (actually that patient had minimal sensory loss and loss of cells and fibers also included Vc).
The most frequent cause of CP appeared to be vascular at all levels, except the brainstem, where tumors, tuberculomas, multiple sclerosis, syringobulbia and hematobulbia contributed; Mills’s 1908 patient suffered mostly central paresthesias. Epileptic pains were also considered CP.

Unfortunately, over the years, despite ample evidence that other lesions can cause CP as well, the term thalamic syndrome became synonymous with CP, despite it being clear to many that it was not so.

In 1969 Cassinari and Pagni, in their monograph Central Pain: A Neurosurgical Survey, wrote: “the conclusions of the various workers who have tried . . . to identify the structure in which lesions are responsible for the onset of central pain sometimes conflict. The divergence of opinion is fairly easily explained by the fact that spontaneous lesions are usually extensive, difficult to define, often plurifocal, and affect several systems with different functions.” By studying iatrogenic “pure” lesions (which they equated to “experimental lesions”) giving rise to CP, they reached the conclusion that the essential lesion was damage to the pain-conveying spinothalamocortical tract. Also, they observed how operations that interrupt the central pain pathways in order to allay pain may themselves originate CP (sometimes more severe than the pain that led to the operation), an occurrence practically impossible to foresee. However, the genesis of CP remained an enigma.

Thereafter, the subject received little additional attention (the “hidden disorder”: Schott 1996). CP remained a neglected field among most medical educators and also among neurologists and neurosurgeons at large. Bonica (1991) found that, of 26,281 pages of text in 14 textbooks of neurology, neurosurgery, medicine and surgery, only 6.5 (0.025%) dealt specifically with CP, a situation that persists almost unchanged to this day. Consequently, most physicians in practice have little or no awareness of the subject.

Until the mid 1980s, little or no research on the clinical characteristics as well as the basic mechanisms and pathophysiology of CP was done, with only a handful of basic and clinical scientists devoting efforts to these objectives. Not even the establishment of the IASP in 1973 and of the journal Pain in 1975 changed this dismaying panorama. At the end of the 1990 Ann Arbor symposium on central pain syndromes (Casey 1991), Lindblom epitomized the problem: “The pain mechanisms of central pain syndromes are virtually unknown and specific analgesic measures are lacking for the vast majority of patients”: CP remained a “puzzling mystery” (Pagni 1989).

The extent of the “puzzle” is given by the bewildering array of theories proposed over 100 years, several directly contradicting one another:

1) Irritation of cells and fibers of spinothalamic and lemniscal systems.
2) Irritation of the sympathetic system, outside the CNS, central cerebrospinal sensory pathways being destroyed.
3) Diversion of pain impulses to the hypothalamus.
4) Summation and wrong integration of pain impulses on a few spared nociceptive neurons.
5) Loss of inhibitory pain mechanisms exerted by thalamus, cerebral cortex, striopallidum, medial lemniscus, brainstem.
6) Activation of alternative secondary pathways, not usually opened and not used when conduction via the spinothalamic complex is available.

7) Abnormal spontaneous or provoked activity in deafferented central sensory neuronal pools which may act as spontaneous dysesthesia and pain-generating mechanisms.

8) Hypersensitivity of deafferented medial midbrain tegmentum, posterior thalamus, thalamic radiations and somatosensory cortex.

9) Activation of nonspecific polysynaptic pathways (paleospinothalamic system), i.e., the neospinothalamic complex and lemniscal system being damaged, nociceptive stimuli are conveyed to the conscious level on this diffuse network of short neurons.

Much has changed over the past 15 years, with several groups applying modern neuroimaging and neurophysiologic techniques to the study of CP. In particular, it is our contention that an explanation and a cure for this “enigma” can now be offered.
CENTRAL PAIN OF BRAIN ORIGIN

EPIDEMIOLOGY AND CLINICAL FEATURES

1. Lesions causing CP and location (Table 2.1a,b)

BCP has been caused by all kinds of lesions at any level along the spinothalamo-parietal path, from brainstem to cortex, a fact already appreciated in the 1930s (Garcin 1937; DeAjuraguerra 1937; Riddoch 1938). These include rapidly or slowly developing processes, apparently without differences in probability of triggering CP (but systematic studies have not been conducted), compressive or disruptive/distractive (these latter perhaps being more often associated with CP).

Stroke, either hemorrhagic or ischemic, is the commonest cause of BCP (without differences between the two); dismayingly, iatrogenic CP is not rare. In agreement with their known incidence, in all studies, infarcts are more common than hemorrhages (roughly 4:1).

When the lesion is thalamic, Vc is always involved (the case of Gonzales et al. [1992] had signs of capsular involvement). Contrary to previous belief, one third or even less of BCP cases are purely thalamic (e.g., Hirato et al. 1993; Andersen et al. 1995; Tasker 2001b; Widar et al. 2002; Oliveira et al. 2002; see also Schmahmann 2003) and complete thalamic syndromes are exceptional. CP does not arise following thalamic lesions only damaging the kinesthetic afferent pathway and probably the spindle afferent pathway as well (Ohye 1998). In all other cases, lesions are cortico-subcortical, in the brainstem, capsulothalamic or lenticulocapsular, or diffuse. Most CPSP is supratentorial (roughly 80%; Tasker 2001).

All cortical lesions responsible for CP involve, exclusively or in combination, the parietal lobe, and specifically SI (and also SII) (e.g., Bassetti et al. 1993). Pain occurring acutely immediately after traumatic cortical injury (e.g., penetrating head injuries) — a lancinating pain felt by the patient at the very moment of injury — has been considered CP of cortical origin (Garcin 1937); it fades away rather quickly (hours to days).

The most common site of brainstem lesions (either stroke or hematobulbia, syringobulbia, tumors and MS) is the medulla oblongata, with few cases of pontine and no pure midbrain spontaneous CP having been reported. However, this may actually be an underestimation, as a brainstem lesion was found in 70% of stroke patients in whom MRI was performed (Vestgaard et al. 1995). CP of bulbar origin is generally due to thrombosis of the posteroinferior cerebellar artery (PICA) giving rise to Wallenberg’s syndrome, in which a lesion impinges on the spinothalamic
TABLE 2.1. Lesions causing CP and their location

<table>
<thead>
<tr>
<th>a: Lesions causing BCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>(1) Vascular lesion (ischemia/infarct*, hemorrhage, including intracerebral and subarachnoid (independent of surgery, due to spasm and infarction or direct brain injury), vascular malformations (AVM through compression, theft, ischcea or hemorrhage, cavernomas through hemorrhage and perhaps compression, compressing nonhemorrhagic saccular aneurysm, venous angiomia), migraine-induced vasospasm). [est. 85%]</td>
</tr>
<tr>
<td>(2) Penetrating trauma [est. 1-2%]</td>
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<tr>
<td>(3) Inflammation: MS, etc.</td>
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<tr>
<td>(4) Infection: abscess (e.g., toxoplasma), gumma, tuberculoma, encephalitis, etc. [est. 4%]</td>
</tr>
<tr>
<td>(5) Tumor (glioma, meningioma, etc., including intratumoral hemorrhage) [est. 1-2%]</td>
</tr>
<tr>
<td>(6) Epilepsy</td>
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<td>(7) Iatrogenic*</td>
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</table>

* There appears to be no difference between hemorraghes and infarcts as regards the tendency to induce CP but infarcts being more frequent (85% vs. 15%) are more commonly cause of CPSP. Likewise, about 80% of all infarcts occur in the caudal territory and engage the thalamus (thalamogeniculate and thalamotiostite arteries), while PICA strokes engage the lower hemisphere. Ischemic lesions may be multiple, often small infarcts especially in the corona radiata and brainstem.

1 Intracerebral hemorrhages may act like tumors and provoke CP by compression.

Also includes one patient with a thalamic DBS apparatus for motor control who developed CP after cardioversion, patients with resected vestibular schwannomas and cerebellar tumors.

b: Site of lesions causing brain central pain

Kameyama (1976)

Anatomopathological study on 87 patients with thalamic lesions mainly involving the Vc nucleus.

Author's conclusion: Thalamic pain is more common in right-sided lesions. Thalamic pain is more frequent in lesions confined to Vc (16% out of 38 cases) or involving Vc nucleus and extending to internal capsule (13% out of 31 cases). Patients with combined Vc and pulvinar lesions (19 cases) developed thalamic pain in 11% of cases. Vc lesions extending to nucleus centrum medianum (11 cases) were never associated with the development of CP. This group of patients, however, suffered from more frequent disturbances of deep sensation. As individual variations are common in this area, even very similar lesions do not necessarily produce the same syndrome.

Graff-Radford et al. (1985)

CT study of 25 patients with non-hemorrhagic thalamic infarction. The location of the lesion was determined by CT slices plotted on appropriate templates of human brain. Blinded assessment. Patients were divided into 4 groups with different clinical hallmarks:

(1) posterolateral thalamic infarcts (9 cases, geniculothalamic artery infarct): sensory loss in all primary modality, without major cognitive deficits or aphasia; dysesthesia, hemiparesis, hemianopia and choreiform movements may be associated

(2) anterolateral thalamic infarcts (5 cases, tuberotalamic artery [paramedian thalamic artery, anterior internal optic artery or premamillary pedicle] infarct): normal sensory findings or transient proprioceptive loss

(3) medial thalamic infarcts (3 cases, deep intrapallial and thalamoperforating pedicle) infarct: normal sensory findings or transient proprioceptive loss

(4) lateral thalamus and posterior internal capsule lesions (depending on the partial or whole involvement of the anterior choroidal territory): diminished pinprick in 4/8 pts

Author's conclusion: Geniculothalamic (posteriorlateral) infarction may cause Dejerine-Roussy syndrome. Dysesthesia developed in 4/9 pts. A geniculothalamic lacuna in primary sensory nuclei (Vc) causes a “pure sensory loss.” Complete geniculothalamic infarction causes a contralateral loss of both proprioception and pain sensation. A partial lesion (lacuna) in the same territory causes impaired pain and light touch sensation but preserved proprioception or vibratory sensibility.