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Nondermatomal somatosensory deficits in chronic pain patients: Are they really hysterical?

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ABSTRACT

Patients with chronic pain disorders frequently show nondermatomal somatosensory deficits (NDSDs) that are considered to be functional. Typically, NDSDs show quadratomal or hemibody distribution ipsilateral to the areas of chronic pain. According to the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition and the *International Classification of Diseases*, 10th revision, such functional somatosensory deficits are classified in the chapter "conversion disorder." Many publications also used the term "hysterical sensory loss." However, doubts are increasing about this one-sided psychiatric view. We aimed to better characterize the biopsychosocial factors associated with NDSDs. Therefore, we compared 2 groups of inpatients with chronic pain disorder, of whom 90 suffered from NDSDs and 90 did not. The patients with NDSDs all showed widespread somatosensory deficits with hemibody distribution. On logistic regression analysis, history of a prior physical trauma was positively predictive for patients with NDSDs. Personality disorder and adverse childhood experiences were positively predictive for the control group with chronic pain disorders without NDSDs. The frequencies of comorbid depression and anxiety disorder did not differ statistically between groups. In conclusion, pain patients with NDSDs are, psychopathologically, by no means more noticeable personalities than patients with chronic pain disorder without NDSDs. Similar to complex regional pain syndromes, we assume a multifactorial etiology of NDSDs, including stress. Based on our observations, terms like "hysteric" should not be applied any longer to patients with NDSDs who suffer from chronic pain.

1. Introduction

This study focuses on widespread nondermatomal somatosensory deficits (NDSDs) occurring in the context of chronic pain disorders. NDSDs are associated with reduced sensitivity of the skin surface to touch and temperature [19,27]. They do not reflect the distribution of peripheral nerves or dermatomes [19,27]. The anatomic extent of the NDSDs usually exceeds the area of pain. NDSDs typically show quadratomal or hemibody distribution, ipsilateral to the areas of chronic pain, with or without facial involvement. In marked cases, subtle motor symptoms (weakness, abnormal posturing, and gait abnormalities) can be observed concomitantly [19].

NDSDs with hemibody distribution were described in patients suffering from chronic pain as early as the 1920s [23]. Over the past decade, an increasing number of studies have begun to explore the clinical phenomenology of functional somatosensory deficits in the context of chronic pain. In doing so, the merely descriptive term of "nondermatomal somatosensory deficits" (NDSDs), promoted by the Canadian duo of Mailis-Gagnon and Nicholson [19], has established itself. Depending on the sample examined, the prevalence of NDSDs in chronic pain patients varies between 25% and 40% [8,16,25]. Despite their frequent occurrences, NDSDs tend to be underreported in scientific publications. On the one hand, this can possibly be explained by the still poorly understood origins of NDSDs, which, on the other hand, also pose some nosological difficulties. Both the *Diagnostic and Statistical Manual of Mental Disorders*, 4th edition (DSM-IV) [28] and the *International Classification of Diseases*, 10th revision (ICD-10) [4] classify functional somatosensory deficits collectively as *mental disorders* (chapters on conversions and dissociation/conversion,

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respectively). Accordingly, they have often been referred to as "hysterical" in the literature [17,21,36].

NDSDs are considered to be *functional* since there is no evidence of *structural* injuries or affections of the somatosensory system, as in the case of neuropathies. Mailis-Gagnon and Nicholson describe a partial or total reversibility in cases of NDSDs after intravenous applications of sodium amytal, which suggests an inhibitory neurofunctional mechanism in NDSDs [19]. The fact that NDSDs habitually appear after physical injuries and are associated with neurological deficits that can be assessed objectively points to an "organic" basis [5,6,27]. Rommel et al. have described NDSDs with hemisensory distribution in patients with physical injuries as *failed back surgery* and *complex regional pain syndrome* (CRPS) [26,27]. Moreover, experimental studies with *artificially* induced local NDSDs [9,14] and results of studies investigating NDSDs with functional brain imaging also suggest a biological basis for NDSDs [5,6,17].

Nevertheless, patients with NDSDs often show a characteristic *psychological* strain profile, too. Previous clinical data indicate high comorbidity rates with depressive and anxiety disorders in patients with chronic pain disorders and NDSDs [6,19]. In itself, this aspect is also well known in patients with chronic pain disorder *without* NDSDs [34].

The purpose of our study was to further elucidate the specific characteristics of NDSDs. We compared 2 cohorts of patients with chronic pain disorders whose primary distinguishing feature was the presence or absence of NDSDs. With regard to the traditional "conversion hypothesis," special attention was paid to sociodemographic and psychological parameters. Our primary question was whether evidence for a specific clinical profile could be identified in patients with NDSDs.

2. Patients and methods

We compared 2 groups of inpatients from the same tertiary university center for multimodal pain therapy. The specialized ward closely collaborates with physician consultants from adjacent specialties (eg, rheumatology, neurology, anesthesiology, orthopedic surgery). All patients suffered from chronic pain disorders, 90 with and 90 without concomitant NDSDs. Patients included with NDSDs all showed widespread somatosensory deficits with *hemibody distribution*.

The patients were examined by internal medicine residents trained in Engel's biopsychosocial interview method [7] and supervised by board-certified internists and psychiatrists trained in psychosomatic medicine, including pain medicine. The interview included a standardized history with structured demographic data forms. Additionally, a detailed psychosocial history was obtained, including specific questions about adverse life events.

The clinical examination of all patients included a thorough psychiatric and neurological examination. Psychiatric comorbidities were assessed with a semi-structured clinical interview following ICD-10 criteria and validated by means of clinical observation over a period of more than 3 weeks of inpatient care. Selective psychometric assessments were carried out to consolidate the diagnoses (Hospital Anxiety and Depression Scale, Montgomery-Åsberg Depression Scale, Clinician-Administered PTSD [posttraumatic stress disorder] Scale and Clinical Global Impressions). Furthermore, all available charts including assessments by other physicians or health care professionals, as well as psychological reports and other psychometric data, were thoroughly reviewed.

The neurological examination included a cranial nerve examination, a comprehensive reflex assessment, and motor function tests. The examination to diagnose NDSDs included sensory testing of superficial touch and heat perception, performed by side-to-side comparison of the whole body [19]. Heat perception was tested by applying hot-water-filled plastic tubes (45°C). Deep-pressure pain testing was performed by side-to-side comparison of manual pressure of approximately 250 kPa/cm^2 applied to multiple bone prominences in the upper and lower extremities. All observed findings and data were documented in our consult notes, body drawings, and the electronic study protocol.

For the statistical analyses, we used SPSS 17 for Windows (SPSS Inc., Chicago, IL, USA). We used descriptive statistics to compare sociodemographic data, clinical symptoms, pain history, history of adverse life events, psychiatric co-diagnoses, and medications between the 2 patient groups. Group comparisons for categorical variables used Pearson χ^2 or Fisher's exact test where appropriate. Mann-Whitney test was used for group comparisons on continuous variables. Variables were expressed as percentages and mean values with SD. The level of significance was set at $P < 0.05$ (2-tailed).

Logistic linear regression analysis was used to identify odds ratios (95% confidence intervals) of predictors of NDSDs and Nagelkerke R^2 statistics to estimate effect sizes.

3. Results

A summary of patient characteristics is given in Table 1. With respect to sociodemographic data, the patient groups showed no significant differences regarding *age* and *gender*, but significantly more pain patients with NDSDs relative to those without NDSDs were married and (still) able to work. In pain patients *with NDSDs*, there was a noticeably high proportion of persons of *Eastern European* origin. Of all patients with NDSDs, 57.8% originated from regions of former Yugoslavia.

As far as NDSDs are concerned, they affected the left half of the body in 58.9%, and the right half of the body in 41.1%. In 45.6% of the patients with NDSDs, the sensory abnormality also involved the ipsilateral face region.

With respect to pain history, both patient groups reported several years of pain, which, moreover, was indicated with relatively high intensity per scores on the VAS. Pain intensity was greater in patients *with* NDSDs than in those *without* NDSDs ($P < 0.001$).

There was a significant group difference in pain onset; three quarters of patients with NDSDs (74.4%) mentioned an acute nociceptive trauma (eg, an accident) as the trigger of their pain condition. The triggering event was consistently located ipsilateral to the ensuing pain disturbance with NDSDs. In contrast, more than half of the patients without NDSDs (56.7%) were not able to describe a distinct triggering event and tended to associate their pain with some sort of degenerative ailment that gradually became worse.

In none of the 180 enrolled patients could we completely and solely explain their chronic pain condition (ie, type, localization, and severity of pain) on examination by a persistent somatic nociceptive lesion only. In 22.2% of pain patients *with* NDSDs and in 26.7% of patients *without* NDSDs, the examining physician could not objectify any ongoing nociceptive peripheral cause of pain at all. These patients suffered mostly from *functional pain syndromes* [20]. Functional pain syndromes occurred in both groups, in part concomitantly with other pain disorders. However, *fibromyalgia*, *chronic low back pain,* and *atypical chest pain* were observed significantly more often in the patient group without NDSDs.

Both patient groups were characterized by a high frequency of adverse life events. Domestic distress (marital violence, worries about family members in war zones) occurred to approximately the same extent. Significant differences, though, were noted with respect to *strain during childhood*: the group of patients *with* NDSDs showed significantly fewer adverse childhood experiences. In turn,

Table 1
Patient characteristics per NDSDs group.

		Patients without NDSDs n = 90	Patients with NDSDs n = 90	Significance
Summary of sociodemographic data				
Age	Mean (range)	46.5 years (16-80)	44.2 years (19-76)	0.14
Gender	Female	51.1%	55.6%	0.37
	Male	48.9%	44.4%	
Marital status	Married	58.9%	84.4%	0.00**
	Not married	41.1%	15.6%	
Ability to work	Disabled/unemployed	86.6%	80%	0.01*
	Working	13.3%	20%	
Origin[a]	Middle Europe[a]	38.9%	16.7%	0.00**
	Southern Europe[a]	7.8%	6.7%	0.78
	Eastern Europe[a]	37.8%	68.9%	0.00**
	Other	15.6%	7.8%	0.10
Clinical symptoms and pain history				
Pain duration	Mean (range)	93.3 months (0-540)	71.7 months (3-264)	0.63
Intensity of pain on VAS	Mean (range)	6.38 (4-10)	7.62 (4-10)	0.00**
History of a painful inciting physical trauma[b]	Minor trauma[b]	26.7%	40%	0.00**
	Major trauma[b]	16.7%	34.4%	
	Without	56.7%	25.6%	
Concomitant postaccidental pain syndromes	CRPS Type I or II	3.3%	4.4%	0.70
	Whiplash-associated pain	11.1%	21.1%	0.07
Concomitant functional pain syndromes	Fibromyalgia	36.7%	14.4%	0.00**
	Low back pain	55.6%	30%	0.00**
	Tension headache	50%	44.4%	0.46
	Irritable bowel syndrome	13.3%	7.8%	0.23
	Atypical thoracic pain	12.2%	3.3%	0.05*
Main adverse life events (multiple answers allowed)				
Imprisonment for political reasons		2.2%	12.2%	0.02*
Directly involved in war		7.8%	16.7%	0.07
Family members involved in war		38.9%	42.2%	0.65
Marital violence		12.2%	13.3%	0.14
Adverse childhood experiences[c]		58.9%	30%	0.00**
History of severe accidents		17.8%	42.2%	0.00**
Concomitant psychiatric diagnoses and symptoms				
Depression[d]		83.3%	77.8%	0.35
Anxiety disorder[e]		31.1%	21.1%	0.13
Insomnia		84.4%	84.4%	1.0
Posttraumatic stress disorder		12.2%	26.7%	0.01*
Personality disorder		25.6%	3.3%	0.00**
Neurotropic medications				
Antidepressants		82.2%	89.9%	0.16
Benzodiazepines		31.1%	26.6%	0.52
Weak opioids (eg, Tramadol)		18.9%	30%	0.08
Strong opioids		15.6%	8.9%	0.17
Anticonvulsants (eg, Pregabalin)		26.7%	27.8%	0.86

NDSDs, nondermatomal somatosensory deficits; VAS, visual pain analogue scale; CRPS, complex regional pain syndrome.
* $\leqslant .05$.
** $\leqslant .001$.
[a] Origin: *Middle Europe* = Switzerland, Germany, Netherlands, and Austria; *Southern Europe* = Italy, Spain, and Portugal; *Eastern Europe* = countries of former Yugoslavia and Turkey; *Other* = countries from Africa, Asia, and other continents.
[b] History of a painful antecedent physical trauma: *Minor trauma* = a non-life-threatening event without significant injuries or with injuries usually healing in a short period of time was considered minor. *Major trauma* = a life-threatening event or the experience of significant violence by another person was considered major.
[c] Adverse childhood experience = emotional or sexual abuse in childhood, repeated physical violence, loss or severe illness of a parent, persistent, serious family conflict.
[d] Anxiety disorder = including generalized anxiety disorder, panic disorder, social phobia.
[e] Depression = including major or minor depression, recurrent, with or without somatic syndrome.

this group was exposed to a greater number of stressors during adulthood (experience of political violence, major accidents).

Both groups of patients with chronic pain showed similarly elevated comorbidity rates as regards depression, anxiety disorders, and insomnia. The PTSD rate in patients *with* NDSDs was significantly higher in that every fourth patient with NDSDs had a co-diagnosis of PTSD, whereas personality disorders were observed to a significantly lesser extent in patients with NDSDs.

The 2 groups examined did not show significant differences with regards to the intake of neurotropic medications (*antidepressants, opioid analgesics, antiepileptics, benzodiazepines*).

On logistic regression analysis, *history of inciting physical trauma* was significantly predictive for a diagnosis of a chronic pain disorder with NDSDs. Conversely, the presence of *adverse childhood experiences* and of a *personality disorder* was significantly predictive for the diagnosis of a chronic pain disorder *without* NDSDs (Table 2).

4. Discussion

The patients examined suffering from NDSDs with hemibody distribution all had a *chronic pain condition*. Most of them showed an accompanying depression and insomnia. However, these accompanying symptoms of psychological distress did not occur more frequently in this group than in the control group of 90 patients without NDSDs. But contrary to what would be expected from the traditional hysteria concept, our patients with NDSDs were by no means more noticeable personalities than the control

Table 2
Logistic linear regression analysis.

Variables entered	Step 1	Step 2	Step 3
Sociodemographic data			
Age	.979 (.953-1.006)	.993 (.963-1.024)	.996 (.965-1.029)
Gender	.649 (.351-1.201)	.623 (.320-1.211)	.730 (.356-1.496)
Migration	2.819 (1.367-5.811)	1.834 (.823-4.089)	1.254 (.504-3.115)
History			
History of prior physical trauma		3.825 (1.916-7.635)	3.773 (1.779-8.000)
History of adverse childhood experience		.334 (.169-.662)	.281 (.135-.584)
Concomitant psychiatric diagnoses			
Depression			.642 (.254-1.624)
Anxiety disorder			.623 (.274-1.415)
Posttraumatic stress disorder			1.781 (.688–4.611)
Personality disorder			.104 (.026-.412)
Model statistics	R^2 = .082*	R^2 = .247***	R^2 = .357**

The table shows the odds ratios with corresponding confidence intervals.
Migration (only step 1) and *History of prior physical trauma* (steps 2 and 3) are positively predictive for nondermatomal somatosensory deficits (NDSDs) in patients with chronic pain disorders. *Adverse Childhood Experience* and *Personality disorder* are positively predictive for the control group with chronic pain disorders without NDSDs.
* $P < 0.05$.
** $P < 0.01$.
*** $P < 0.001$.

group of patients with chronic pain disorder without NDSDs. The prevalence value for personality disorders in the NDSDs group (3.3%) is much the same as in the normal population, where it is estimated to range from 1% to 9% [11,15].

In a recent review article, Mailis-Gagnon and Nicholson reported several case series of patients with NDSDs and chronic nonmalignant pain syndromes [19]. Generally, clinical findings and symptoms closely match the findings in our case series. Mailis et al. reported a large case series of patients with chronic pain and NDSDs; the history and sociodemographic data are almost parallel [16]. Compared to the whole spectrum of NDSDs, our group is characterized by its uniform hemisensory symptomatology and the virtual absence of motor abnormalities.

The earliest comparable case series with *"hysterical anesthesia"* and pain following an industrial accident was reported by Moldofsky and England in 1975 [21]. At a time when Europe was dominated by psychoanalysis, both *unexplained pain syndromes* and *neurofunctional deficits* (eg, NDSDs) were usually suspected to be *"hysterically induced"* and, following Freud's concept of conversion, they were interpreted as psychogenic – psychogenic in a psychodynamic sense, referring, for example, to repressed conflicts in immature personalities [30]. In common parlance, the terms "psychogenic" and "hysterical" have been used for decades to describe neurofunctional deficits in the implication of *unreal* or *self-caused*. In this traditional context, the dualistic understanding of the human body has always diametrically opposed "psychogenic" and "somatogenic."

In this respect, the picture is more differentiated nowadays. The advent of DSM-IV and ICD-10 allowed for less rigid interpretations of these neurofunctional disorders. Nevertheless, in many cases, a monocausalistic psychiatric perception still prevails. Recently, further progressive differentiations and readjustments are developing with regard to the diagnosis of *conversion* and *dissociation* [22,29,32]. We have to decide whether we want to change the *terms* or redefine their *meaning*. Concerning NDSD, we have to note its strong compound with pain.

With regard to NDSDs, simply defining the symptoms as "psychogenic" or "hysterical" is no longer accurate; the setting is clearly more complex. Sudeck's comment on CRPS is likely to hold true for NDSDs, too: "One should be careful not to simply assume psychological reasons for pain disorders one does not sufficiently understand" [33]. Similar to CRPS, in 3 of 4 patients with NDSDs, there is a datable inciting physical trauma giving rise to the pain disorder. As with CRPS, however, there is no relationship between the intensity of the somatic trigger event and the degree of the ensuing pain disorder.

Moreover, patients with NDSDs are also clearly distinct with respect to their social history: in patients *with* NDSDs, the focus is significantly more often on *exogenous stressors experienced in adulthood* (eg, migration, political violence, and accidents). One fourth of these patients are suffering from PTSD. The context of experienced violence and stress may also explain the high percentage of patients from the war-ridden former Yugoslavian regions. This *exogenous* reaction profile in adulthood is in contrast to the profile in the comparator group of people suffering from pain disorders without NDSDs in whom we can see significantly more adverse life events in childhood; this also might be the explanation why *personality disorders,* which have their onset in childhood, are more often diagnosed in the control group [2].

For some time, it has been suspected that chronic pain conditions develop against a background of stress-related strain in the patient's biography [1,24,35]. Generally speaking, it would seem that exposing the central nervous system to high levels of stress increases the risk of hyperalgesic pain processing. In a recent meta-analysis, this association was even confirmed for patients with CRPS [3].The overlap with CRPS is noteworthy to the extent that, in patients with CRPS, per se, NDSDs are also frequently seen [25]. Hemisensory deficits, as described in patients with CRPS [27], exactly correspond in qualitative terms to the hemisensory NDSDs described in the present and our previous studies [6]. Similar to CRPS [13], peripheral, autonomic, and central factors are involved in the occurrence of chronic pain disorders with NDSDs [27]. Therefore, one might wonder whether NDSDs with hemibody distribution are not some type of CRPS. But there are also differences: the typically deep, inner pain in NDSD patients by definition lacks superficial allodynia, while CRPS patients are moreover characterized by their local and primarily positive sensory disturbances. Furthermore, the pain diagnoses of our patients with NDSDs are heterogeneous; according to current criteria of definition [31], only 4 patients showed a co-diagnosis of a clear CRPS. Rather, our data show that NDSDs seem to have the capacity to graft themselves as an *epiphenomenon* onto a variety of chronic pain disorders, *including* CRPS.

Finally, in NDSDs, multiple pathophysiological factors are suspected. The hemisensory pattern and the results of the functional brain imaging suggest a superior dysfunction of the central

nervous system [5,6,17]. On the other hand, pain character and local symptoms suggest, similar to many cases with CRPS, a kind of *sympathetically maintained pain* [12]. Already in the early 1980s, a case report by Gross on a bullet wound of the subclavian artery revealed that the impairment of the periarterial sympathetic plexus can change the sensitivity (especially sensitivity to touch and temperature) [10]. Several cases of NDSDs with typical clinical signs of a dysregulated autonomic nervous system are reported [5,19]. Maybe the thesis of a stress-related (centrally mediated) dysfunctional autonomic nervous system could someday also explain the *nondermatomal* anatomical distribution of these sensory abnormalities.

In brief, signs and symptoms provoked by an exogenous somatic trigger (as with CRPS) plus a context of frequent experience of exogenous violence (as in the case of PTSD) suggest a complex reactive phenomenology, including peripheral, autonomic, and stress-associated central aspects that do not follow a dichotomous classification pattern (either psyche or soma). Both the somatic *and* the stress-associated psychological factors call for an integrative approach to better understand this type of pain disorder. In our previous positron-emission tomography study on pain patients suffering from NDSDs, we observed a cerebral involvement of both the *somatosensory systems and* the *emotional areas* of the prefrontal-limbic systems [6]. Angela Mailis-Gagnon wrote an editorial for that journal issue entitled "Nondermatomal Somatosensory Deficits: A Neuropsychobiological Phenomenon?" [18]. In light of the available evidence, there now are even stronger reasons to answer this question with "yes".

Conflict of interest statement

The authors declare that they have no conflicts of interest.

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References

[1] Anton F. Chronic stress and pain – a plea for a concerted research program. PAIN® 2009;143:163–4.
[2] Battle CL, Shea MT, Johnson DM, Yen S, Zlotnick C, Zanarini MC, Sanislow CA, Skodol AE, Gunderson JG, Grilo CM, McGlashan TH, Morey LC. Childhood maltreatment associated with adult personality disorders: findings from the Collaborative Longitudinal Personality Disorders Study. J Pers Disord 2004;18:193–211.
[3] Beerthuizen A, Van 't Spijker A, Huygen FJPM, Klein J, de Wit R. Is there an association between psychological factors and the Complex Regional Pain Syndrome type 1 (CRPS1) in adults? A systematic review. PAIN® 2009;145:52–9.
[4] Dilling H, Mombour W, Schmidt MH (Hrsg). Internationale Klassifikation psychischer Störungen, ICD-10, Kapitel V(F). Bern/Göttingen/Toronto/Seattle: Hans Huber; 2000.
[5] Egloff N, Gander ML, Gerber S, von Känel R, Wiest R. Halbseitenstörung nach Unfall. Praxis 2010;99:797–801.
[6] Egloff N, Sabbioni ME, Salathé C, Wiest R, Juengling FD. Nondermatomal somatosensory deficits in patients with chronic pain disorder: clinical findings and hypometabolic pattern in FDG-PET. PAIN® 2009;145:252–8.
[7] Engel GL, Morgan WL. The clinical approach to the patient. Philadelphia: WB Saunders Co.; 1969.
[8] Fishbain DA, Goldberg M, Rosomoff RS, Rosomoff H. Chronic pain patients and the nonorganic physical signs of nondermatomal sensory abnormalities (NDSA). Psychosomatics 1991;32:294–303.
[9] Geber C, Magerl W, Fondel R, Fechir M, Rolke R, Vogt T, Treede RD, Birklein F. Numbness in clinical and experimental pain—a cross-sectional study exploring the mechanisms of reduced tactile function. PAIN® 2008;139:73–81.
[10] Gross D. Therapeutische Lokalanästhesie [German]. Stuttgart, Germany: Thieme Verlag; 1982.
[11] Gustavsson A, Svensson M, Jacobi F, Allgulander C, Alonso J, Beghi E, Dodel R, Ekman M, Faravelli C, Fratiglioni L, Gannon B, Jones DH, Jennum P, Jordanova A, Jönsson L, Karampampa K, Knapp M, Kobelt G, Kurth T, Lieb R, Linde M, Ljungcrantz C, Maercker A, Melin B, Moscarelli M, Musayev A, Norwood F, Preisig M, Pugliatti M, Rehm J, Salvador-Carulla L, Schlehofer B, Simon R, Steinhausen HC, Stovner LJ, Vallat JM, den Bergh PV, van Os J, Vos P, Xu W, Wittchen HU, Jönsson B, CDBE2010Study Group. Cost of disorders of the brain in Europe 2010. Eur Neuropsychopharmacol 2011;21:718–79.
[12] Jänig W. Autonomic nervous system and pain. In: Bushnell MC, Basbaum AI, editors. Pain. The senses: a comprehensive reference, vol. 5. San Diego: Academic Press; 2008. p. 193–225.
[13] Jänig W, Baron R. Complex regional pain syndrome: mystery explained? Lancet Neurol 2003;2:2687–97.
[14] Leffler AS, Kosek E, Hansson P. Injection of hypertonic saline into musculus infraspinatus resulted in referred pain and sensory disturbances in the ipsilateral upper arm. Eur J Pain 2000;4:73–82.
[15] Lenzenweger MF, Lane MC, Loranger AW, Kessler RC. DSM-IV personality disorders in the national comorbidity survey replication. Biol Psychiatry 2007;62:553–64.
[16] Mailis A, Papagapiou M, Umana M, Cohodarevic T, Nowak J, Nicholson K. Unexplainable nondermatomal somatosensory deficits in patients with chronic nonmalignant pain in the context of litigation/compensation: a role for involvement of central factors? J Rheumatol 2001;28:1385–93.
[17] Mailis-Gagnon A, Giannoylis I, Downar J, Kwan CL, Mikulis DJ, Crawley AP, Nicholson K, Davis KD. Altered central somatosensory processing in chronic pain patients with "hysterical" anesthesia. Neurology 2003;60:1501–7.
[18] Mailis-Gagnon A, Nicholson K. Nondermatomal somatosensory deficits (NDSDs): a neuropsychobiological phenomenon? PAIN® 2009;145:12–3.
[19] Mailis-Gagnon A, Nicholson K. On the nature of nondermatomal somatosensory deficits. Clin J Pain 2011;27:76–84.
[20] Mayer EA, Bushnell MC. Functional pain syndromes: presentation and pathophysiology. Seattle: IASP (International Association for the Study of Pain) Press; 2009.
[21] Moldofsky H, England RS. Facilitation of somatosensory average-evoked potentials in hysterical anesthesia and pain. Arch Gen Psychiatry 1975;32:193–7.
[22] Nicholson TR, Stone J, Kanaan RA. Conversion disorder: a problematic diagnosis. J Neurol Neurosurg Psychiatry 2011;82:1267–73.
[23] Pette H. Das Problem der wechselseitigen Beziehung zwischen Sympathikus und Sensibilität [German]. Dtsch Z Nervenheilk 1927;100:143–8.
[24] Raphael KG, Widom CS, Lange G. Childhood victimization and pain in adulthood: a prospective investigation. PAIN® 2001;92:283–93.
[25] Rommel O, Gehling M, Dertwinkel R, Witscher K, Zenz M, Malin JP, Jänig W. Hemisensory impairment in patients with complex regional pain syndrome. PAIN® 1999;80:95–101.
[26] Rommel O, Maercklin A, Eichbaum A, Kuprian A, Jäger G. Hemisensorische Störungen bei neuropathischen Schmerzen im Rahmen chronischer Nervenwurzelreizsyndrome [German]. Der Schmerz 2005;19:59–64.
[27] Rommel O, Malin JP, Zenz M, Jänig W. Quantitative sensory testing, neurophysiological and psychological examination in patients with complex regional pain syndrome and hemisensory deficits. PAIN® 2001;93:279–93.
[28] Sass H, Wittchen HU, Zaudig M, Houben I (Hrsg). Diagnostisches und Statistisches Manual Psychischer Störungen - Textrevision, DSM-IV-TR, Kapitel Somatoforme Störungen. Göttingen/Bern/Toronto/Seattle: Hogrefe Psychologie; 2003.
[29] Spitzer C, Barnow S, Freyberger HJ, Grabe HJ. Recent developments in the theory of dissociation. World Psychiatry 2006;5:82–6.
[30] Spitzer C, Freyberger HJ, Kessler C. Hysterie, Dissoziation und Konversion. Eine Übersicht zu Konzepten, Klassifikation und diagnostischen Erhebungsinstrumenten [German]. Psychiatr Prax 1996;23:63–8.
[31] Stanton-Hicks M, Jänig W, Hassenbusch S, Haddox JD, Boas R, Wilson P. Reflex sympathetic dystrophy: changing concepts and taxonomy. PAIN® 1995;63:127–33.
[32] Stone J, Carson A, Aditya H, Prescott R, Zaubi M, Warlow C, Sharpe M. The role of physical injury in motor and sensory conversion symptoms: a systematic and narrative review. J Psychosom Res 2009;66:383–90.
[33] Sudeck P. Die sogenannte akute Knochenatrophie als Entzündungsvorgang [German]. Chirurg 1942;15:449–58.
[34] Tunks ER, Crook J, Weir R. Epidemiology of chronic pain with psychological comorbidity: prevalence, risk, course, prognosis. Can J Psychiatry 2008;53:224–34.
[35] Van Houdenhove B, Luyten P. Beyond dualism: the role of life stress in chronic pain. PAIN® 2005;113:238–9.
[36] Vuilleumier P, Chicherio C, Assal F, Slosman D, Landis T. Functional neuroanatomical correlates of hysterical sensorimotor loss. Brain 2001;124:1077–90.