

# PSYCHIATRIC DIFFERENTIAL ASPECTS OF PAIN

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## Introduction

Pain represents a frequent symptom in various psychiatric disorders ranging from so called psychogenic or somatoform disorders to depression, to schizophrenia and to organic brain syndromes including dementia. Thalamic lesions are long known to induce painful symptoms. The cortex is involved in pain awareness. The locus coeruleus modulates pain perception. Various facets of pain in a range of psychiatric syndromes is here addressed, as a means of clinical differential diagnosis:

## Somatoform disorders

Paradigmatic disorders with difficult to explain pain is somatoform disorders (F45.4 ICD-10 or DSM-IV 381) and the respective chronic variant (ICD-10 F45.4 and DSM-IV 307.80).

There is some difference between the understanding of somatoform disorder in ICD-10 vs. DSM-IV: in ICD-10 there is more the assumption that no 'real' organic basis for the symptoms of pain may be found whereas according to DSM-IV the uncertainty and the possible involvement of a bodily disorder as a triggering state is assumed, but the severity of symptoms should appear 'inadequate'. The diffuse pain syndrome is mainly considered to be triggered by emotional conflicts or psychosocial problems with, according to a clinical evaluation, enough importance to explain the intrapsychic conflict. The mechanisms to explain this type of pain in neurobiological frameworks remains open. To be differentiated is migraine or increased muscle tensions as consequence of other somatic disorders. This etiological classification depends heavily

from the quality of differential diagnostic attempts and of assessments especially whether positive indicators of a severe intrapsychic conflict to explain a psychogenic disorder are present or not, also the availability of diagnostic means or the psychopathological understanding of respective possible pathomechanism. The unsolved problem is seen in the syndromes included into F45.4 such as psychalgia or psychogenic lumbago. It is apparent that the assessments, e.g. of muscle tension, is rather arbitrary and in so far the organic basis of pain remains of considerable uncertainty.

A teaching example of uncertainties is the recent description of improved diagnostics in a subgroup of chronic fatigue syndromes respectively fibromyalgia syndromes and somatization disorder which often include anxiety and depression in parallel: it appears now, with considerable plausibility, that in a subgroup of cases fibromyalgia seems related to slight compression of the upper spinal cord either on the basis of congenital or spongiotic cervical stenosis or brain stem compression due to tonsillar ectopia [Heffez et al, 2007].

Also another view found in the scientific literature may be relevant in a considerable subgroup: a broad array of somatoform disorders with overlapping or rather similar symptoms such as chronic fatigue, fibromyalgia, or chronic pain and burn-out, may be considered as hypo-cortisolemic disorders [Heim et al 2000]. A close brain body connection evoked under stress circumstances, has been clearly shown in experiments: the ergotropic vs. trophotropic stress response according to Hess [Hess 1961] was central for understanding this interconnection, and appears to be complemented by autonomic mechanisms through neuroendocrine pathways [Hellhammer and Hellhammer 2008]. It is interesting, that even single interventions into the reactivity or (spontaneous) activity, (in two directions of

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hypo- or hyper-) of the locus coeruleus (LC) and other stress brain senses may have lasting effects guided by norepinephrine and the sympathetic nervous system. Because the LC is also directly involved to mediate peripheral pronociceptive and peripheral antinociceptive effects by modulating the output.

### Pain and depression

There is abundant literature about a frequent association between pain and depression in general, pain found in up to 60 or 80% of patients with depression [Karp et al 2005; Emptage et al 2005; Simon et al 1999]. Pain in depression is usually explained by central mechanisms, e.g. plasticity of the cortex. The locus coeruleus and the thalamus appear involved. Nevertheless, the exact neurobiological basis of pain in depression remains in various aspects unclear. Many believe that subgroups exist and not infrequently a mixed pathogenesis of psychogenic mechanisms and biological mechanisms may occur. However, even when central mechanism is involved, peripheral mechanisms may be triggers.

Inflammatory pathomechanisms become increasingly plausible to be involved in subgroups of depression: recent psychoimmunological results showed abnormal CSF contents in a large subgroup of patients with depression [Bechter et al 2010; Maxeiner et al, 2009]. In this context, it is interesting that following inflammation or nerve injury simple stimuli which are normally perceived as innocuous, can evoke persistent pain [Drew and Dermore 2009], mediated by previously unknown small sensory neurons that respond to light touch [Seal et al 2009]. Mild CNS inflammation, represented in slightly abnormal CSF contents, might reach the peripheral nerve and even free nerve ends according to a recent hypothesis [Bechter 2007]. Such mechanisms could explain both pain and slight muscle lesions, a long known rather surprising finding in depression and in schizophrenia [Meltzer and Crayton 1972], in a new way. Over this, pain was rapidly improved with cerebrospinal fluid filtration in single case studies [Bechter 2007 and submitted].

### Schizophrenia and pain

In some types of schizophrenia painful symptoms can prevail, prevalent together with other somatop-

sychic symptoms, termed coenesthetic symptoms. Gerd Huber included in his definition of coenesthetic schizophrenia only cases which showed in parallel psychotic schizophrenic first and second rank symptoms according to Kurt Schneider [Huber G, 1957]. A detailed discussion of the various concepts of coenestopathy and the like, and the coenesthetic subtype of schizophrenia was recently given [Wichowicz and Cubala 2008]. Various somatopsychic symptoms including even severe pain symptoms are frequent in schizophrenia. This type of sensory disturbances was usually understood to originate from central brain mechanisms (compare depression). Based on the mild encephalitis hypothesis of a subgroup of psychoses one may understand pain and other bodily symptoms in a new way: cerebrospinal fluid efflux along peripheral nerves (and brain nerves), a pathway described as extralymphatic pathways in animals, and direct CSF-nerve interaction could induce such symptoms (see depression).

### Organic brain syndromes and pain

Pain can be a leading symptom in the so called 'Durchgangssyndromes', i.e. acute reversible organic psychoses. This would match with the so called hyperesthetic emotional neuropathy (synonymous to reversible pseudoneurasthenic syndromes). A specific subtype would represent meningoencephalitis. In the differential diagnosis of organic pain syndromes it is important, to perform careful neurologic examination, brain imaging and often CSF investigation.

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