

## PAIN AND EMOTIONAL PROCESSING IN PSYCHOLOGICAL TRAUMA

## SCHMERZ UND EMOTIONALER VERARBEITUNG IM PSYCHOLOGISCHEN TRAUMATA

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### SUMMARY

Extreme psychological and physical traumas cause dramatic symptom patterns which are insufficiently described by the psychiatric diagnostic criteria of post traumatic stress disorders (PTSD). Additionally, due to the neurobiological proximity and similarity of processing mechanisms of physical and psychological pain stimulation and extremely negative emotions, the patients often suffer from persistent pains even after the somatic healing process is completed. Epidemiological studies confirm the joint occurrence of pain and PTSD. The close relationship and the etiological and behavioral similarities of both disorders have led to the development of joined vulnerability and mutual maintenance models. The particular suffering of patients with PTSD due to chronic pain necessitates pain-therapeutic interventions. On the other hand, in chronic pain patients, the etiological role of severe traumas should be considered.

**Key words:** trauma – pain – stress - emotion

### ZUSAMMENFASSUNG

Extreme psychische und körperliche Traumatisierungen bewirken dramatische Symptommuster, die durch die psychiatrischen Diagnosekriterien der Posttraumatischen Belastungsstörung (PTBS) nur unzureichend beschrieben werden. Aufgrund ähnlicher neurophysiologischer Verarbeitungen von psychischer und körperlicher Schmerzen und intensiver, negativer Emotionen leiden Menschen nach Traumatisierungen oft unter persistierenden Schmerzen – auch wenn die körperlichen Folgen der Traumata geheilt sind. Die vorliegenden epidemiologischen Studien bestätigen das simultane Auftreten von PTBS und chronischen Schmerzen. Die enge Beziehung zwischen Schmerzen und Symptomen der PTBS führten zu gemeinsamen Krankheitsmodellen zur Vulnerabilität und gegenseitigen Aufrechterhaltung der Symptome (vulnerability and mutual maintenance models). Daraus folgt die Notwendigkeit das Schmerzleiden von Patienten mit PTBS spezifisch schmerztherapeutisches zu behandeln. Andererseits sollte der ätiologischen Bedeutung von traumatischen Ereignissen für die Schmerzentstehung und –aufrechterhaltung bei Schmerzpatienten beachtet werden.

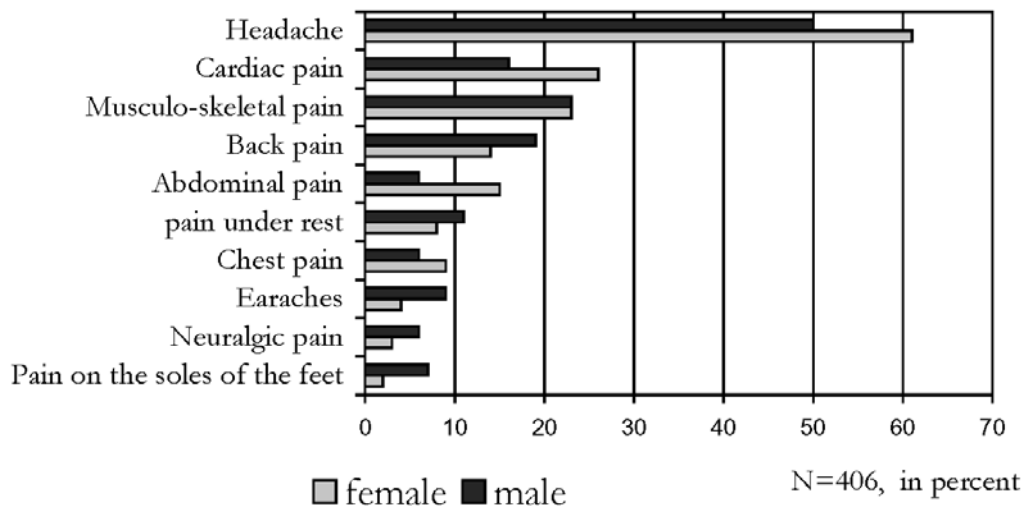
**Schlüsselwörter:** Trauma – Schmerz – Stress - Emotion

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### INTRODUCTION

Due to traumatic experiences, many patients suffer from prolonged complex disruptions of their somatic and mental functions, which are summarized under the concept of post traumatic stress disorder (PTSD). These disruptions are obviously severe and more persistent if they are the result of man-made disaster (Herman 1994). A characteristic of this kind of traumatization is the deliberate infliction of extreme emotional states and physical damages that are accompanied by strong emotions and often by considerable pain. The extent (i.e., the intensity and quality) of pain and physical injuries depends on the type of trauma. While psychological traumatization causes a general vulnerability to pain, physical traumatizations lead to somatic disorders and specific pains. Since physical trauma simultaneously constitutes extreme psychological stress, a complex symptom pattern usually results. Complex posttraumatic stress disorder (C-PTSD) usually results from prolonged exposure to a traumatic event or series thereof and is characterized by long-lasting problems with many aspects of emotional and social functioning.

Pains due to traumatic effects on the body are initially acute, but may later persist for a long time; much like scars, alterations in connective tissue, muscles, or even the skeletal apparatus. Blows to the soles of the feet, for example, may damage or destroy the connective tissue in such a way that walking and running becomes very painful, necessitating comprehensive orthopedic care. Yanking up the arms of the victims or the so-called “banana tie” overstretches joints, connective tissue, and muscles lead to long-term bodily disorders. Figure 1 shows the enormous pain of traumatized patients from the Ulmer Behandlungszentrum für Folteropfer (Ulm Treatment Center for Torture Victims). It is misleading, however, if, during the usual diagnoses of posttraumatic stress disorder, the physical symptoms are exclusively considered as a mental phenomenon leading to somatic manifestation of psychological injuries. In the worst case, this can become a failure to provide necessary (somatic) treatments. Indeed, the general interactions between trauma and pain (vulnerability) and the particular effects of physical traumas are difficult to distinguish in individual cases (Traue et al. 2010).



**Figure 1.** Pain symptoms (in %) in N=406 patients suffering from torture-induced trauma

If one includes the data from the diagnostic interviews for each individual and correlates them with the mentioned symptoms to arrive at an estimate of the extent of traumatization, the correlation between trauma intensity and pathology is between  $r=0.29$  and  $r=0.52$ . The higher the frequency and therefore also the dura-

tion, the higher the number of subsequent pain symptoms (see Table 1).

Traumas associated with sexual violence, such as humiliation, rape, obscene insults, or corresponding threats lead to an accumulation of abdominal pains and headaches in men and women (see Table 2,  $**<0.01$ ).

**Table 1.** The correlation between the extent of traumatization and later complaints (adapted from Leißner et al. 2007).

Frequency of trauma events	Number of psychological symptoms	Number of physical symptoms	Pain	Number of overall symptoms
Women	0.30**	0.40**	0.29**	0.43**
Men	0.49**	0.35**	0.39**	0.52**

**Table 2.** Comparison between sexual and other traumas with regard to resulting pains (adapted from Leißner et al. 2007,  $**<0.01$ ,  $*<0.05$ ,  $^†<0.10$ ).

	Rape and sexual abuse N=114 between 17 and 55 years of age	Other traumas N=92	Chi square
Abdominal pain	24 (21%)	7 (8%)	6.2**
Headaches	78 (68%)	46 (52%)	5.9*
Painful micturition	6 (5%)	0	4.8 <sup>†</sup>
Kidney pain	9 (8%)	1 (1%)	4.9 <sup>†</sup>

### COGNITIVE-BEHAVIORAL INTERACTIONS BETWEEN TRAUMA AND PAIN

Although traumatic experiences can neither be cognitively nor behaviorally coped with in the way everyday stress can be coped with, the brain tries to avoid acute extreme stress through central nervous and psychophysiological activity. Nevertheless, the memory trace of this emotional event remains retrievable. As memory consolidation takes place during sleep, memory contents are unpredictably activated, triggering nightmares. It is important to know that the adverse effects of trauma on memory and cognitive functions interfere with therapeutic interventions that draw on

social and cognitive learning processes (Nilges & Traue 2007).

As one of the brain survival mechanisms during extreme traumatization, the victims dissociate the unbearably intense feelings of fear and panic from their current consciousness (dissociation). The emotional experience of fear and physical pain is kept away from the current, conscious experience only by means of major psychological effort. Later, the survivor permanently attempts to contain their experiences; an effort which requires a great deal of psychological energy and leads to a state of exhaustion and disturb the ability to control pain.

Since the trauma cannot be coped with immediately, the high emotional arousal acts internally and cannot be

converted into actions that facilitate coping. Although the organism becomes extremely activated, it essentially remains ineffective regarding the traumatic situation. The emotional arousal explodes inwardly; it turns into an emotional implosion (Traue & Deighton 2007). The consequences of this particular form of emotional repression are expressed in the aforementioned central nervous and psychophysiological hyperactivity and emotional numbing, which is accompanied by a retreat from the social environment, intrusions including flashbacks and attempted suppression, verbal emotional poverty, and activation of cognitive fear networks. The situational inevitability of the trauma substantially contributes to this processing. Due to the high intensity of the experience, such conditions very quickly lead to learned helplessness and therefore feelings of guilt and depression, which, in turn, result in various pain processes (Traue et al. 1997).

People have a primary need for closeness to other people, which is interindividually more or less pronounced. Stressors and dangers increase the need for closeness and social support (Schachter & Singer 1962). This can directly affect behavior, but may also be mediated cognitively through an internal image of common values or beliefs. This feeling of being on the same wavelength with others is an important protection mechanism against stressors. Particularly man-made traumas, such as criminal violence, rape, and torture involve a massive disruption in the feeling of belonging. Physically and psychologically, the victim is abruptly torn away from their social relationships. Since the emotional experience during extreme trauma is greatly characterized by fear, panic, and feelings of abandonment, it lastingly undermines the victims trust in interpersonal relationships (Janoff-Bulman 1992).

The theoretical model of joined vulnerability of PTSD and pain is therefore based on three etiological mechanisms: increased levels of anxiety induced by the trauma, increased levels of arousal, and a common genetic pathway for both. The model of mutual maintenance postulates multiple mutual interactions on the basis of attentional bias, fear sensitivity, trauma memory, depression with diminished activity level, changed body awareness, and cognitive stress. This model includes nearly all aspects, but empirical studies particularly confirm the aspect of a joined influence of stress, changed attention, fear sensitivity and depression.

Liedl and Knaevelsrud (2008) take up the idea of common etiological and maintaining factors, but consider as the core of both disorders avoidance behavior concerning trauma memories in PTSD patients and pain-relevant movements in pain patients. As described in the “fear avoidance model” by Norton and Asmundson (2003), fear loaded with catastrophizing thoughts is causative for the avoidance of activities with painful consequences. Psychological energy maintains this interaction on the basis of physiological

overexcitation common to both disorders. The etiology of PTSD is expressed quite similarly: Initially the affected persons react with strong psychophysiological arousal to the extreme momentary stressor and later to their memory of it. Since these states are avoided, psychological and physical stress results equally by virtue of emotional inhibition and stressful memories (Traue & Deighton 2007). The cycles of fear and avoidance caused by trauma experiences and pain interlock in a barely distinguishable fashion.

## **THE PSYCHOBIOLOGY OF THE INTERACTIONS BETWEEN TRAUMA AND PAIN**

Modern neurobiological imaging techniques have raised great expectations for psychological pain research. Chen (2001) summarized the results of this research: The brain structures fundamentally involved in pain perception have been determined. These brain structures are not limited to one pain center, but represent a neuronal matrix, which includes all areas that are activated by sensory, affective and cognitive information processes, particularly the primary sensory cortex, insular cortex, cingulate gyrus, periaqueductal grey, and frontal cortical area.

One consequence of frequent pain in PTSD is the expectation of new pain (Otis et al. 2003). Bischoff et al. (1982) showed that the cognitive expectation of pain lead to physiological reactions similar to those originating from actual pain stimulation shortly before acute nociceptive stimulation. In a fMRI experiment Sawamoto and his team (2000) prove that such a pain expectation during nonpainful stimulation causes a neuronal excitation pattern resembling that caused by pain stimulation itself. As another pathway the dysregulation of the noradrenaline (NA) system is essential for the interaction between trauma and pain: In PTSD patients NA is continuously increased, while the number of receptors is reduced by up to 50% due to chronicity of the overactivation. This correlates with cardiovascular reactions triggered by trauma-relevant stimuli. If the NA level is pharmacologically lowered, patients exhibit panic attacks and flashbacks of the trauma (Southwick et al. 1993). Confrontation of traumatized persons with their experiences (sometimes merely isolated aspects are sufficient) leads to increased activity of endogenous opioids, which are, however, depleted in case of over excitation. Continuous stress caused by an unprocessed trauma therefore leads to dysregulation of the endogenous opioid system, resulting in hyper- or hyposensitivity to pain or fluctuations between these poles. When exposed to trauma-related stimuli, some patients with PTSD react with an increase in endogenous opioids that corresponds to an 8 mg dose of morphine. Saporita & van der Kolk (1992) interpret this opioid reaction as a common cause for emotional numbing and bodily pain.

Pain and negative emotions are processed in the same neuronal matrix comprised of the primary sensory cortex, insular cortex, cingulate gyrus, periaqueductal grey, and frontal cortical areas. Several of these areas are more strongly activated in the right hemisphere of the brain (Chen 2001). This corresponds to the idea of a right dominant lateralized localization of attention control through pain stimuli, negative emotions (Coan & Allen 2004), and vegetative excitation (Traue 1998).

fMRI studies concerning feelings of social isolation are groundbreaking for the relationship between social isolation and pain vulnerability. The observed neuronal excitation patterns in the anterior cingulate cortex (ACC) correspond to sensory pain stimulation: the (psychologically) painful experience of social marginalization is processed similarly to acute painful stimulation (Eisenberger et al. 2003). It is remarkable how clearly this effect can be detected even in existentially unimportant game settings. Recent volumetric studies among PTSD patients (Geuze et al. 2007, Kasai et al. 2008) demonstrate reduced ACC volumes (which might be due to alcohol abuse), but also reduced neuronal function of the ACC (Schuff et al. 2008). This might conflict with findings regarding increased ACC activation in case of social marginalization. However, the alteration of the ACC is the cause for resistance to extinction of stressful stimuli (negative emotions, pain, memory contents), and therefore also for vegetative hyperreactivity with respect to trauma-relevant stimuli. Therapeutic interventions on the basis of confrontation could be less efficient for PTSD, because reduced neuronal function of the ACC has a less inhibiting effect on the amygdala-based fear response (Schuff et al. 2008).

Frequently, traumatized persons exhibit a tendency towards self-destructive behavior. States of great arousal with emotional experience content can be regulated, in a more or less socially conspicuous fashion, by means of self-injury or addictive behavior, including drugs, alcohol or pharmaceuticals. In connection with this, Kosten and Krystal (1998) pointed out the arousal-dampening effect of the brain's opioids and posed the question of whether painful self-injury could possibly stimulate the opioid system of the brain and maintain self-destructive behavior through the learning mechanism of negative reinforcement. This poses the risk of overstimulation and depletion of pain inhibitors.

## **INTEGRATION OF PAIN TREATMENT INTO A TRAUMA TREATMENT CONCEPT**

The treatment of posttraumatic stress disorder focuses on confrontation with the trauma. Not all trauma therapists agree with the primacy of this approach, however, nor does it represent the reality of treatment in treatment centers (Deighton et al. 2007).

There is no cohesive treatment concept, but rather treatment elements that resemble a phase-like course of therapy, which should be imagined as a spiral rather than linearly. The initial phase focuses on establishing security and trust. Subsequently the treatment of psychosomatic problems should take priority. This includes pain therapeutic interventions, restoration of a positive relationship with one's own body, healthy sleep habits, and sometimes necessary somatic treatments. Interdisciplinary forms of treatment are favorable during which the patients learn to relax, newly acquire pleasant bodily sensations, and receive physiotherapy. The most difficult phase of therapy concentrates on therapeutically guided confrontation with the experienced trauma. Afterward one can therapeutically work on reinterpreting the experience (normalization of the abnormal; sensu Herman 1994) and strive for social integration (Maercker 2003, Traue et al. 1997, Schwarz-Langer et al. 2010).

### **Regaining control over the body, emotions, and thoughts**

Systematic therapeutic strategies must take into account the patient's need for security in all of these areas. At the beginning the therapist must explain to the patient the different interactions of trauma experience, PTSD, and pain in such a way that the patient does not experience their own reactions as strange and pathological, but rather as a normal reaction in an abnormal stress situation. Physical therapy, medication, relaxation techniques and interventions for guiding attention, the interpretation of symptoms in the complex disease model, and the development of a trusting therapeutic relationship are important elements at the beginning of therapy. Relaxation should be coupled with the promotion of physical activity. This therapeutic step should be aided by contact with support groups, mobilization of the social environment, and creation of a safe ground. Connection with pre-traumatic resources and social activation are helpful during this process.

In the next phase of treatment, the therapeutic focus is on improving the disturbed bodily self-experience, the coping with pain, and lowering hyperactive arousal. Relaxation techniques are as useful as physical activity. Biofeedback treatment can be useful if muscle tension or reduced peripheral circulation (usually in connection with stress) is involved in the occurrence of pain. Since biofeedback also improves body awareness, this intervention influences several mechanisms involved in the maintenance of pain. Body focused interventions are advantageous, as many of the patients exhibit a somatic disease concept. All of these interventions must take into account available resources and cultural characteristics as well as the type of traumatization. Furthermore, the high arousal level of the patients can be reduced, and, in combination with physical therapy, the patients again begin to experience their bodies in a positive manner.

A first application of neurofeedback related to ACC activation in an experiment with artificial pain and in a small number of chronic pain patients by Christopher deCharms and his team (deCharms et al. 2005) is very promising. In several sessions the subjects and patients received feedback on a screen (as is also customary in case of vegetative biofeedback) regarding the activity of their ACC. They were asked to increase or decrease the activity by means of different mental strategies. After three learning trials, the experimental group was able to suppress ACC activity and decrease pain perception by between 23% and 38%. The chronic pain patients were similarly successful: They achieved a reduction of pain by between 44% and 64% and also a change in ACC activity. Such a procedure could be used to directly treat dysfunctional central nervous system activities of the hyperactive neuronal pain matrix.

### Remembering and grieving

The basic principle of personality strengthening and special attention to the need for security applies here as well. Exploration of the victims' life before the trauma and the circumstances which caused the trauma (the flow of time), followed by the reconstruction of the trauma and all sensations involved; often with the help of nonverbal methods. The trauma does not change during this stressful period of reconstruction; it rather becomes more concrete, more real. Through repetitive, controlled reliving of one memory after another, transformation by means of advanced behavioral therapeutic techniques of stimulus confrontation or creation of witness reports aims at removing the horror from the events. The mourning process is accompanied by processing of revenge fantasies and wishes for compensation, deep feelings of guilt and shame, but also a search for positive experiences and strengthening of bonding and relationship skills.

The reconstruction of the trauma can not be fully completed during therapy. Every new stage of life is accompanied by new conflicts and challenges which inevitably reawaken the trauma and bring to light a new aspect of the traumatic experience. The main work is completed when the trauma can be considered to lie in the past on a time continuum and the patient participates in life again with new hope and strength.

### Reentering

Creating a future, developing a new "self", socializing, searching for meaning and new activities, adapting to a new situation. Problems of the first phase are often tackled again during this phase; not defensively, but now actively. The patient should be prepared for the fact that symptoms of posttraumatic stress can reoccur in new life phases and in times of great stress.

### Medication

Patients with chronic pain are usually also treated with analgesics; either by prescription or with medication they procured for themselves. This is true for traumatized persons with migration backgrounds, particularly if they find themselves in a situation where healthcare is limited to acute complaints. Due to their cultural background or the very basic general medical care offered, such patients often emphasize their pain and receive analgesics. It is therefore important to ensure that the right medication and dosing regimen are used so as to avoid pain due to overuse. In the course of (pain) psychotherapeutic treatment one can work towards reducing the use of analgesics. Sometimes it is useful to supplement (pain) psychotherapeutic treatment with antidepressants or with an atypical neuroleptic agent (Schwarz-Langer et al. 2006), or to create conditions suitable for psychotherapeutic interventions with the help of medication.

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