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POSTTRAUMATIC STRESS DISORDER

Bio-Psycho-Social Aspects, Eye Movement Desensitisation and Reprocessing and Autogenic Training in Persistent Stress**

Case Study, Part 1

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‘There is one more thing I would like to add:
It wouldn’t be bad if for once truth
and love prevailed over lies and hatred.’

(Václav Havel, 1997, our trans.)

The inclusion of the diagnostic category Posttraumatic Stress Disorder (PTSD) in both the American and the international diagnostic systems, and the definition of the ‘exceptional stressor’ has probably contributed to the recent boom in psychotraumatology research. The bio-psycho-social model seems to be the most adequate account with regard to the multiple layers of the problem. The authors provide an overview of recent research findings. Numerous scientific studies have focused on finding effective treatment methods for trauma-related disorders. Both pharmacologic and psychotherapeutic approaches have been found effective. Nowadays, the authors consider psychotherapy to be more essential in the treatment approach for traumatogenic disorders.

Keywords: posttraumatic stress disorder, bio-psycho-social model, therapy, psychotherapy

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1 Original text: 'Pravda a láska musí zvítězit nad lží a nenávisti.' In a speech at a demonstration in Prague on 10 Dec 1989, shown live on the Czechoslovak TV, Václav Havel said, ‘Truth and love must prevail over lies and hatred’ (our trans.). Later, it became one of Havel’s most famous remarks. His critics, however, ridiculed it for being naive, simplistic and childish. Years later (on 1 Jan 1997), Havel self-ironically alluded to this quote in another TV interview when his last words to the camera were: ‘There is one more thing I would like to add: It wouldn’t be bad if for once truth and love prevailed over lies and hatred’ (our trans.). Original text: ‘A ještě jeden pocit bych chtěl říct. Vůbec by nezaškodilo, kdyby tu a tam zvířela lásku nad lží a nenávisti.’

Schlüsselbegriffe: posttraumatische Belastungsstörung, das bio-psycho-soziale Modell, Therapie, Psychotherapie

1. Introduction

Psychological trauma or repeated traumata can play an important role in the etiology of several mental illnesses. The most characteristic clinical picture resulting from a psychological trauma is described by both the international and American diagnostic system as posttraumatic stress disorders (PTSD). According to epidemiological studies, incidence of psychological trauma in PTSD represents a serious medical and social problem.

In this short overview, we will first focus on the description of PTSD. In the second part, we describe and discuss the case of our patient. Using the bio-psycho-social model of mental illness, we will specially focus on its social aspects. Eye Movement Desensitisation and Reprocessing (EMDR) has become an important component in the treatment of posttraumatic stress disorders. The name of this method is historical, as it has been discovered that bilateral stimulation in trauma treatment does not have to use eye movements. Bilateral tactile or auditory stimuli are equally effective, and for some patients more acceptable. In case of ongoing traumatisation, EMDR as well as other exposure psychotherapeutic methods are considered contraindicated, relatively contraindicated or at least complicating the therapy. Autogenic training (AT) is a rarely mentioned component of PTSD treatment. It is usually considered as a contraindication in acutely symptomatic patients.

In our case study, we illustrate a meaningful and effective use of both these methods in the treatment of a patient with PTSD after type II trauma (repeated traumatisation). EMDR was used to reduce the PTSD symptoms; AT to increase the resistance towards ongoing stressors. The case has been closely related to recent social and political processes. Therefore, we considered it important to focus on the social level of the bio-psycho-social model of mental health and illness.
2. Diagnosis

Posttraumatic stress disorder is established according to the International Classification of Diseases and Health Related Problems (World Health Organisation 2004) and the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) (American Psychiatric Association 2000). The diagnostic criteria in the ICD-10 and the DSM-IV are very similar (Smolík 2002). The ICD-10 code (WHO 2004) for PTSD is F43.1. The diagnosis requires the fulfilment of 5 criteria, A–E:

A) The patient must have been exposed to a stressful event or situation (of either brief or long duration) of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone.

B) There must be persistent remembering or reliving of the stressor in intrusive flashbacks, vivid memories or recurring dreams, or in experiencing distress when exposed to circumstances resembling the stressor.

C) The patient must exhibit an actual or preferred avoidance of activities and situations reminiscent of the trauma.

D) Either of the following must be present:
   – Inability to recall either partially or completely some important aspects of the period of exposure to the stressor, or
   – Persistent symptoms of increased psychological sensitivity and arousal by difficulty falling or staying asleep, irritability or outbursts of anger, difficulty concentrating, hypervigilance, and exaggerated startle response.

E) Criteria B, C and D must all arise within 6 months of the period of stress. However, a delayed onset is possible, too.

The DSM-IV-TR (APA 2000) and ICD-10 criteria (WHO 2004) for the diagnosis of PTSD are similar. The DSM-IV criteria are, however, more precisely formulated, and there are also some differences. For example, the DSM-IV defines a traumatic event (criterion A) as exposure to, witnessing or learning about an event or events that involve actual or threatened death or serious injury or other threat to one’s physical integrity or the physical integrity of another person. The person’s response to the event must involve intense fear, helplessness or horror. There is a possibility of illusions, hallucinations, and dissociative flashbacks, especially after waking up or when intoxicated (criterion B). Further symptoms may include (criterion C) diminished interest or participation in significant activities, feelings of detachment or estrangement, restricted range of affect (e.g. inability to have loving feelings), and sense of foreshortened future (e.g. no expectations to experience a career, marriage, children or a normal life span). The symptoms should last more than one month (criterion E). In the DSM-IV-TR, there is also criterion F: the disturbance causes clinically significant distress or impairment in social, occupational or other important areas of functioning.
Table 1
PTSD criteria according to DSM-IV (APA 2000) and ICD-10 (WHO 2004)

<table>
<thead>
<tr>
<th>Criteria</th>
<th>DSM-IV</th>
<th>ICD-10</th>
</tr>
</thead>
<tbody>
<tr>
<td>Criteria of a traumatic event</td>
<td>A1: Event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others. A2: Person’s response involving intense fear, helplessness, or horror</td>
<td>A stressful event or situation of threatening nature which would likely cause pervasive distress in almost anyone</td>
</tr>
<tr>
<td>Sufficient symptoms</td>
<td>Symptoms from following areas: B: Intrusions (at least one) C: Avoidance / emotional numbness (at least three) D: Autonomous hyper-arousal (at least two)</td>
<td>Persistent remembering or reliving of the stressor in intrusive flashbacks, vivid memories or recurring dreams</td>
</tr>
<tr>
<td>Duration of the symptoms</td>
<td>E: At least four weeks; acute: 1–3 months; chronic: 3 months and longer</td>
<td>No specifications</td>
</tr>
<tr>
<td>Onset of the symptoms</td>
<td>E: No specifications. Delayed onset when at least six months have passed between the stressor and the onset of the symptoms</td>
<td>Within six months of the period of stress</td>
</tr>
<tr>
<td>Clinical impairments</td>
<td>F: The disturbance causes clinically significant impairment in important areas of functioning</td>
<td>No specifications</td>
</tr>
</tbody>
</table>
3. Problematic definition of a traumatic event in posttraumatic stress disorder (PTSD)

According to the DSM-IV-TR, the person’s response to a traumatic event must involve intense fear, helplessness or horror. These reactions can be observed in most typical cases. These criteria, however, do not allow the diagnosis of PTSD in cases of dissociative amnesia, or when the predominant acute response of the person involves stupefaction, indifference or derealisation. Another problem arises when the patient displays typical symptomatology, but the traumatic event was not of an exceptionally catastrophic nature.

MOL et al. (2005) compared the incidence of PTSD symptoms in people experiencing a traumatic event according to the DSM-IV to those experiencing a stressful life event not fulfilling the A1 criterion according to the DSM-IV. The authors included serious illness, chronic illness of a close person, and problems related to work and relationships. They found that PTSD symptoms were more frequent in people experiencing a stressful life event within the last 30 years, although this event did not fulfil the A1 criterion (DSM-IV).

In clinical practice, one often distinguishes between type I and type II trauma (TERR 1991). Type I trauma refers to a single traumatic event of a brief duration; type II trauma describes long-lasting and/or repeated traumatisation. One possible reaction to type II trauma is the ‘complex PTSD’ (HERMAN 2001) or so-called disorder of extreme stress not otherwise specified (DES-NOS) (LUXENBERG et al. 2001). This disorder has not yet been included in diagnostic manuals. It can partially overlap with borderline personality disorder (F60.3) or the ‘enduring personality change after catastrophic experience’ (F62.0) according to the ICD-10 (WHO 2004).

Psychoanalytically oriented authors have used the term trauma/psychological trauma in a broader sense, meaning, for example, emotional overload. This was even before traumatic event was defined in the DSM-III, IV and ICD-10. Similarly, the term neurosis was broader and did not describe only neurotic disorders as we know them today, but also syndromes like PTSD, adjustment disorders, and personality disorders, especially those in the B cluster (anxious personality disorder). In his encyclopaedic monograph mapping the development of dynamic psychological approaches, Leonhard SCHLEGEL writes about trauma:

Von Traumen wird in der Tiefenpsychologie übrigens keineswegs nur gesprochen, wenn es sich um akute Ereignisse oder schockartige Erlebnisse handelt. Als Ursprung der Neurosen spielen vielmehr langdauernde Konfliktsituationen, jahrelanger Druck durch eine lieblose Erziehung und ähnliches, sozusagen also ‘chronische Traumen’ eine viel wichtigere Rolle. Auch wo einzelne Ereignisse offensichtlich die Bedeutung von Traumen zu haben scheinen, kann doch in ihnen gleichsam wie in einer Zusammenfassung sich eine längerdauernde belastende Situation symbolisieren.

(1972, 212)
He added the following sentence to the Slovakian edition of his book: ‘I define a psychological trauma leading to neurotic disorders as an *emotional overload.*’ (SCHLEGEL 2005, 30, our trans.).

In childhood, physical and emotional neglect are as harmful as sexual, physical, and emotional abuse. The long-standing effects of emotional cruelty and neglect on neurobiology and mental health might even be more significant than those of physical abuse (TEICHER et al. 2006). Several clinical pictures may arise as a consequence of experiencing a traumatic event. This remains true even if we define trauma according to the current diagnostic criteria. A traumatic event may lead to an acute stress reaction and consequently to PTSD. A traumatic event may also lead to a picture similar to adjustment disorder (F43.2, F43.8). A trauma might, however, also lead to a depressive episode, anxiety disorder, somatisation disorder, substance dependency or dissociative disorder, either directly or through an adjustment disorder or PTSD (FLATTEN et al. 2004). The further development may contain personality alteration (the aforementioned ‘complex PTSD’). Type II trauma in young age may play an important role in the etiology of a borderline personality disorder, with unresolved trauma, disorganized and insecure-ambivalent attachment style (WÖLLE 2006, 3, 70). In these cases, trauma is diagnosed as one of the factors influencing health status and coded with a Z code according to the ICD-10. Comorbidity with other psychiatric disorders is common. According to various authors, the comorbidity rates are up to 80%. Apart from all aforementioned pathogenetic factors of trauma, in some cases, thankfully, mental health is preserved in spite of a trauma. Trauma either does not lead to a mental disorder, or a subsequent mental disorder ends with a full remission (salutogenesis). Furthermore, as our clinical experience teaches us, a psychological trauma might lead to a seemingly paradoxical personality development, deepening and broadening of the consciousness, increase in responsiveness to human suffering, improvement in the ability to protect oneself and others from trauma, and willingness to help with their processing. A person unfolds an altruistic mode of mind and behaviour and expands the ability to mentalise.

4. Epidemiology of PTSD and its various courses

KESSLER et al. (1995) studied both point and lifetime prevalence of PTSD in a representative sample of 6000 subjects. The study was conducted in the USA. The lifetime prevalence was 10.4% in women and 5% in men; the point prevalence in the last month was 2.8% (average women and men). However, approximately 51% of women and 61% of men in the study experienced a stressor corresponding to the DSM-IV criteria. Therefore, most people experiencing trauma do not develop PTSD;

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2 Original text: ‘Ja vymedzujem psychickú traumu, ktorá vedie k neurotickým poruchám, ako emocionálne preťaženie.’ And in the German manuscript: ‘Ich umschreibe das psychische Trauma, das zu neurotischen Störungen führt, als emotionale Überforderung.’
they recover spontaneously. On the other hand, 17% of men had intrusive memories, but the traumatic event did not fulfil the DSM-IV criteria. In some cases, a *subliminal* PTSD arises, meaning that symptoms do not reach the diagnostic threshold. Another possibility is *partial* PTSD, when after a traumatic event, the person experiences symptoms which are not otherwise typical of PTSD. In some of these cases, the symptomatology might lead to severe impairment in important areas of functioning. In a study focusing on 185 fire and car accident victims, 22.7% fulfilled the criteria of PTSD, but another 16.7% with partial PTSD fulfilled the F criterion referring to the impairment in everyday functioning (Mylle & Maes 2004).

Various possibilities of coping with trauma are shown in the following scheme in *Figure 1* (Tagay et al. 2013).

![Diagram](https://example.com/diagram.png)

*Figure 1*

Scheme: Possible developmental courses after a traumatic event

(Copyright Tagay et al. 2013)
In Germany and in Europe, the lifetime prevalence in people younger than 65 years is 1–3%. In the German population older than 65 years, the lifetime prevalence is threefold. This is due to World War II experiences (FROMMBERGER & MAERCKER 2011). Psychological traumatisation of ‘child soldiers’ leads to PTSD in only about 20% of cases. The longer they participate in fighting, the smaller this percentage. ‘Child soldiers’ are approximately eight-year-old boys kidnapped by the rebels and trained to kill, as it often happens in some African countries, but also in Asia and South America. By contrast, PTSD was found in 80% of refugees from Sudan. Apparently, ‘child soldiers’ learn to view killing as an ‘exciting activity’. According to ELBERT (2010), they switch to a ‘hunter mode’. They need to see blood and hear the cries of the dying. They cut off their ears, noses or extremities. Some of them become cannibals.

In a psychiatric and psychotherapeutic hospital in Germany, the therapists (psychiatrists, physicians with training in psychosomatics or clinical psychologists) considered trauma as a reason for mental disorder in 31.5% of patients. This group included all PTSD patients, 12% of patients with an affective disorder, 26% of anxiety disorders patients, and 19% of patients with a personality disorder. In a questionnaire assessing psychotraumatic events, these patients listed 6 and more events (UNGERER et al. 2010).

In our hospital in Trenčín, Slovakia, we assessed all patients hospitalised in the first half of 2004. The assessment included the structured clinical DSM-IV interview focusing on psychological trauma and PTSD (WITTCHEN et al. 1997). The total number of patients admitted to the ward was 577. Out of these patients, we assessed 532. Those not assessed included mostly patients with various types of dementias. A
A history of a traumatic event was found in 27%. In 8%, we found a serious stressor not fulfilling the DSM-IV criteria of a traumatic event. 64% of patients did not indicate a traumatic event in their history (Figure 2). PTSD was found in 35% of patients listing a traumatic event in their history, and subliminal PTSD was found in 26% of those patients. In 38.6% of patients, no symptoms of PTSD were found, although they had a history of a traumatic event (Figure 3). We did not assess possible time and content relationships between psychological traumatisation and psychiatric syndromes other than PTSD. The highest comorbidity was found between PTSD and depressive disorders (HAŠTO et al. 2011).

![Figure 3](image)

Psychological traumata (N = 145; 27%) and PTSD or subsyndromal PTSD in the group of patients with a history of psychological trauma

One week after psychological trauma, PTSD is found in 94% of adult patients. Three months after a traumatic event, PTSD is found in only 47%. After years, PTSD is found in 10–25% of people that have experienced psychological trauma (summarised according to FROMMBERGER & MAERCKER 2011). These data show that although a considerable number of people heal spontaneously, there is a relatively high risk of chronification of untreated PTSD. In some samples, chronic PTSD was found in 25% of people with a history of psychological trauma.

5. Risk and protective factors

The following factors have been identified to play a role in the development of PTSD: subjective experience of loss of control, anticipated negative consequences regard-
ing a person’s own health and future, event intensity and duration, physical injury or injury or death of another person. Other risk factors include: insufficient familial and social support, female gender, experience of early separation, and former somatic illness (FROMMBERGER & MAERCKER 2011). Although these risk factors do statistically increase the probability of PTSD development, according to the same authors, in individual cases PTSD does not develop even when these factors are present. On the contrary, PTSD after a serious trauma may develop even in a person with stable personality and no risk factors. Factors indicating diminished risk of PTSD include stability prior to the trauma, ability to mobilise individual resources and problem coping strategies from the past, introspection, and ‘sense of coherence’ according to Antonovsky. A developed sense of self-efficacy, active engagement of personal problems rather than avoidance, and an ability to share difficulties are positive prognostic factors (FLATTEN et al. 2004; FROMMBERGER & MAERCKER 2011). The ability to share difficulties might be connected to secure attachment style (HAŠTO 2005). Finally, it goes without saying that respect for the victim and their trauma reduces the risk of chronic PTSD (FROMMBERGER & MAERCKER 2011).

5.1. Biological level

Genetic vulnerability explains about one third of the variance in PTSD incidence. In animals, early negative experience leads to increased and longer-lasting stress reactions (RUEGG 2010, 158). In PTSD, increased amygdala activation has been observed, as well as decreased prefrontal cortex activation and inhibition of Broca’s motor speech centre when speaking of the trauma (HOFMANN 2006). Thus, the turn of phrase ‘speechless from fear’ found in many Slovak folk tales has a neurobiological explanation. Increased noradrenergic stress response leads to memory consolidation of the experience (FROMMBERGER & MAERCKER 2011). The interpretation of volume reduction in hippocampal formation remains controversial, but one possible explanation is chronically increased activation in the hypothalamic-pituitary-adrenal axis (BRÜNE 2012; WARNER et al. 2006).

KAMPFHAMMER (2006) summarises the findings as follows: Basal hypothalamic-pituitary-adrenal axis activity and reactivity is increased. There are signs of increased adrenergic (both central and peripheral) reactivity and decreased vagal control. Objective signs of increased startle reactions and hypervigilance (enhanced P300 amplitude as a reaction to loud noise) have been found. In Vietnam veterans, increased incidence of A1 allele of the DA-2 receptor as well as increased incidence of specific DA transporter mutation has been found in chronic PTSD. Some findings suggest that reduction of hippocampal formation might be a premorbid vulnerability factor rather than a consequence of PTSD.

In each mental disorder or predisposition to a mental disorder, the considerations of the biological aspects should include evolutionary considerations, as was suggested for example by Nesse and Brüne. Evolution shaped us to be social crea-
tures, but at the same time vulnerable. Chances of survival and reproduction are higher when we are alert to potential threats, including those inside our social group. In the course of human evolution, neuroanatomical structures involved in evaluation of potentially dangerous situations have become bigger. In normal circumstances, it is advantageous to have mechanisms enabling us to make use of previous experience to avoid present or future risks. In PTSD, this mechanism is pathologically hyperactive, and it inhibits rather than supports adaptive reactions (BRÜNE 2012). The more extreme the traumatic event and the more the individual was made sensitive by previous traumata, presumably also by cumulative micro-traumata, and/or the higher the genetic vulnerability, the easier it is for this mechanism to become pathologically unbalanced. On the other hand, a sufficient amount of sensitive interactions with significant others, supporting inner working models of secure attachment, may increase the resilience of the individual.

There is only a limited number of ways to prevent the onset of PTSD after a traumatic event. The efficacy of psychological interventions remains questionable (FROMMBERGER & MÄRCKER 2011); further studies might confirm the effectiveness of certain techniques elaborated by cognitive-behavioural therapists. Benzodiazepines are commonly prescribed for acute adjustment disorder, but they are probably counterproductive, as higher PTSD and depression incidence has been found in people treated with benzodiazepines in comparison with control groups (FROMMBERGER & MÄRCKER 2011). According to our knowledge, prophylactic treatment with other psychotropic drugs has not yet been fully explored.

Several studies have found that up to 50% of patients experiencing acute psychosis later show symptoms similar to PTSD as a reaction to the experience of helplessness and disintegration (FLATTEN et al. 2004). We consider this finding important for clinical practice and therapy planning for psychotic patients. In his study of 208 patients suffering from schizophrenia, M. BLEULER (1978) found secondary pathogenic influence of the psychotic experience that can even lead to a personality change. In the ICD-10 (WHO 2004), this is categorised as F62.1. Furthermore, according to BLEULER (1978), schizophrenic illness tends to run a more severe course in patients coming from broken homes where serious traumatisation can be assumed.

Serious dissociative symptoms are an interesting aspect of posttraumatic symptomatology. They can occasionally resemble or even equal psychotic experiences including Schneider’s first-rank symptoms (thought insertion, flashbacks escalating to hallucinations, voices representing dissociated parts of the personality, feelings of being influenced by external agents). They represent a real challenge for differential diagnosis (MOSKOWITZ et al. 2008).

Some findings suggest that early psychological traumatisation might influence the manifestation, course and symptomatology of bipolar affective disorder, including suicidality and functioning between the episodes (ÉTAÍN et al. 2008). Several retrospective and (for now only sporadic) prospective studies have confirmed connections between adverse childhood experiences and numerous mental disorders, such as borderline personality disorder, certain types of depression, dissociative disorders, some
types of bipolar affective disorder, schizophrenia, substance dependency and others. Recently, some studies have focused on similar connections in somatic illnesses.

Childhood psychological traumatisation might sometimes indirectly lead to somatic illness through problematic behaviour such as smoking, alcohol and drug consumption, and sexual promiscuity (sexually transmitted diseases). Early psychological traumata and their accumulation might cause life-lasting wounds that are not healed with time. Or, to put it more cautiously, they are not always healed with time. There are reasons to doubt the common consolation, ‘time heals all wounds’.

Vincent J. FELLITTI with his co-authors (1998) and their Adverse Child Experience (ACE) Study deserve credit for studying this problem. Fellitti is professor in San Diego, and Anda in Atlanta. Between 1995 and 1997, 26,000 patients undergoing a routine health screening were asked about adverse experiences in childhood. Informed consent for use of personal data for study purposes was given by 71% of patients (54% females and 46% males, average age 57 years), representing a sample of 18,175 subjects. All patients possessed health insurance, and thus were not necessarily representative of the American population; most belonged to the middle or upper middle classes. However, 64% listed at least one traumatic childhood event. One third of the subjects had two or more traumatic events in childhood (younger than 18 years). A part of the sample was followed prospectively and the whole sample retrospectively. Eight categories of childhood abuse and adverse experience of parent-child relationship were studied: emotional and physical abuse, sexual violence/abuse, substance abuse, serious mental illness, violence toward the mother, a family member in prison, and parental separation or divorce were considered indicators of a disintegrated family system. The ACE Score attributes one point for each category of exposure to child abuse and/or neglect. Thus, if a person indicated three types of traumatisation, his/her score was 3, etc.

5.2. An excerpt from the ACE Study questionnaire listing the percentage of positive response in the sample N = 18,175

**Adverse childhood experience – maltreatment, abuse**

*Emotional* – 10.3%

– Swear at you, insult you, put you down, or humiliate you?
– Act in a way that made you afraid that you might be physically hurt?

*Physical* – 28.0%

– Push, grab, slap, or throw something at you?
– Ever hit you so hard that you had marks or were injured?
Sexual – 20.4%
Did an adult or person at least 5 years older than you ever
– Touch or fondle you or have you touch their body in a sexual way?
– Attempt or actually have oral, anal, or vaginal intercourse with you?

Severely damaged family system / domestic violence

Dependency – 26.6%
Did you live with anyone who was a problem drinker or alcoholic or who used street drugs?

Mental illnesses – 19.0%
Was a household member depressed or mentally ill, or did a household member attempt suicide?

Violence towards the mother – 12.6%
Was your mother or stepmother:
– Often or very often pushed, grabbed, slapped, or had something thrown at her?
– Sometimes, often, or very often kicked, bitten, hit with a fist, or hit with something hard?
– Ever repeatedly hit at least for a few minutes or threatened with a gun or knife?

Household member in prison – 4.5%
Did a household member go to prison?

Parental separation or divorce – 22.8%
Were your parents ever separated or divorced?

The results of the ACE Study indicate that there is a connection between psychological traumatisation in childhood and pregnancies in adolescence. Adolescence pregnancies, for their part, are connected with long-lasting psychosocial consequences and an increased foetal death risk. The higher the ACE Score, the higher the lithium and antipsychotic prescription rates. When the ACE Score is 5 or higher, there is a tenfold increase in these prescription rates. The ACE Score also positively correlates with frequency of work- and relationship-related problems in adulthood, financial problems, absences from work, emotional stress, somatic symptoms, and substance abuse. The higher frequency of somatic and mental illnesses leads to a higher mortality. The ACE Score also positively correlates with nicotine dependency. Nicotine might be understood as ‘self-medication’; it is probably an attempt to influence the negative emotional consequences of neurobiological and social adverse child experience influences. Each ACE Point increases the probability of drug abuse two- to fourfold. Intravenous drug abuse, on its part, leads to a higher hepatitis and endocarditis risk. Hallucinations represent a nonspecific psychopathological symptom, as they can be found in affective disorders, schizophrenia, schizoaffective disorders, serious posttraumatic stress disorders, dissociative and other disorders. If the ACE Score is 7 and more, the risk of psychotic symptoms increases fivefold. Each ACE Point increases the risk of suicidal behaviour. This relationship remains even after correcting for alcohol and drug dependency depression. Two thirds of
all suicidal attempts in adulthood can be understood as long-term consequences of childhood adverse experience. Liver diseases, especially cirrhosis, are one of the prevailing causes of death both in Europe and the USA. The most frequent cause of cirrhosis is hepatitis and alcoholism. For hepatitis C, the risk of chronic course is 70%. Similarly, 40% of patients suffering from a liver disease have hepatitis C.

Illegal drug consumption and hazardous sex behaviour increase the risk of hepatitis B and C. Combination with alcohol consumption accelerates cirrhosis and increases the risk of hepatic cancer. The ACE Score positively correlates with chronic liver illnesses. Risk factors of cardiac disease need to be revised, too. ‘Conventional’ risk factors, such as smoking, diabetes, physical inactivity and hypertension explain only half of the variance of the cardiac disease incidence. According to Harald Schickendanz and his colleagues (SEIDLER 2011), several prospective studies suggest that psychosocial factors such as depression, anger and hostility support development of a coronary illness. Furthermore, they may play a role in frequency of fatal complications, such as heart attack. An ACE Score of 9 significantly increases the risk of a coronary illness (FELLIOTTI et al. 1998). There is a direct connection between chronic obstructive pulmonary disease and nicotine abuse – cigarette smoking. An ACE Score of 4 increases its risk by 93%. The ACE Study shows that there is a connection between all illness-related deaths (deaths due to coronary illness, cancer, chronic pulmonary disease, accidents with a skeletal injury) and frequency of childhood traumatic experiences.

In a big sample of somatically ill patients, the ACE Study confirms what we suspected based on the clinical practice. According to SCHICKENDANZ & PLASSMANN: ‘The results clearly show that psychosocial stressors in childhood are destructive. Furthermore, they have life-long consequences. They are the most important factor deciding about health, wellbeing, and individual and social efficiency’ (2011, 447).

According to some critics derogating the findings of this and similar studies, subjects might tend to exaggerate childhood traumatic events due to the methodology. However, this criticism is not fully justified. Patients with positive family experience have little reason to speak badly of them. On the contrary, we should assume higher rates of traumatisation than those found in the ACE Study. This is due to the fact that many victims experience amnesia after a traumatic event. Partial or complete amnesia, either permanent or in some periods of life, is found in 5–20% of traumatised subjects, most frequently after sexual abuse (HOFMANN 2006). There were no direct questions assessing emotional or physical neglect, which might be considered a minor flaw of the ACE Study. The methodology only allows indirect inference of neglect.

6. Therapy of posttraumatic stress disorder

PTSD treatment, similarly to the treatment of other mental disorders, might include psychotherapy and pharmacotherapy. In pharmacological studies of PTSD treatment,
a 30% reduction of symptomatology is considered an improvement. In randomised controlled trials, such an improvement is observed in 50% of cases. 

Sertraline and paroxetine have been found to have the most positive effects. The effectiveness of mirtazapine and venlafaxine is similar. Pharmacological treatment in PTSD is not as effective as it is in depression. Compared to the treatment of depressions, PTSD treatment requires higher dosage, starting from a low dose and keeping the full dose for at least eight weeks, since the medication may not be immediately effective. The recommended duration of pharmacological treatment is 1–2 years. There is an increased relapse risk after the discontinuation of the medication. In case of psychosis-like symptomatology, an atypical antipsychotic might be considered. Some findings suggest that olanzapine and quetiapine have an additional positive effect on sleep. In case of aggression, valproate may be used (Benkert & Hippius 2011).

7. Psychotherapy of posttraumatic stress disorder

The authors of the monograph on supportive psychotherapy, the most prevalent psychotherapeutic approach in the USA (Novalis et al. 1999), describe several principles that are used by other psychotherapeutic approaches too. They include communication style leading to strong therapeutic alliance, repeated psychoeducation, and encouragement to verbalise details of the traumatic event and related emotions. The therapist has to be ready to listen to the story again and again, to repeatedly reassure and to retain a realistic therapeutic optimism. Sigmund Freud (1969) was the first one to point out the therapeutic importance of reliving and re-suffering the traumatic event. This is often connected with an affective catharsis. He encouraged the patients to make the trauma fully accessible to the consciousness, including all its aspects and details, verbalisations and related affective reactions. This happens within a safe therapeutic relationship.

In psychotherapy of a trauma, it is important to consider a specific way of confronting the patient with the traumatic memories. This enables new learning and reworking of the dysfunctional memory material (Bob 2011). Typically, traumatic memories emerge uncontrollably. Their repeated presentation does not, however, have a positive effect on emotional burden and on extinguishing the conditioned reaction. On the contrary, re-traumatisation happens. The negative affective memories are strengthened, and they are generalised to other stimuli. One of the most common flaws in psychotherapy, potentially leading to iatrogenic damage, is an uncontrolled emotional abreaction, including full reliving and free expression of related emotions with no therapeutic guidance of the process. The therapeutic effect of the sole ‘emotional discharge’ has been questioned (Hart & Brown 1992). Remembering the traumatic event must always be preceded by sufficient stabilisation of the patient. This is especially important when treating massively and repeatedly traumatised patients. The stabilisation includes building sufficient affective tolerance and the ability to maintain dual attention through the whole period of trauma confrontation.
Dual attention means sufficient contact with the traumatic memories on one hand, and contact with the situation here and now on the other. This ability to integrate the past into the full consciousness of the present represents the main therapeutic goal. Important aspects are activation of inner sources of safety (positive inner objects) and of the present safe therapeutic relationship (psychotherapy as a secure base, Bowlby) (Hašto 2005). This goal is reached in three phases of trauma therapy: stabilisation, confrontation with the trauma, and reintegration. These therapy stages were proposed already by P. Janet (Hart & Dorahy 2009).

Methodical guidelines for PTSD treatment were formulated by authors belonging to various therapeutic schools. Some of them are mainly dynamically oriented; others describe themselves as cognitive-behavioural. The biggest body of empirical evidence confirms the effectiveness of cognitive-behavioural techniques, especially exposition and cognitive therapy (Beneđek & Wynn 2011). We often encounter integrative approaches or pragmatic eclecticism. The aforementioned supportive psychotherapy can be classified as a dynamic approach or as a supportive pole of dynamic (Gabbard 2005) or psychoanalytic (Wöller & Kruse 2005) psychotherapy. It chooses the best techniques according to what is needed to solve a specific problem (Novalis et al. 1999). Methodically structured guidelines of PTSD treatment that could be classified rather as psychodynamic have been formulated by Horowitz (2003), Levenson et al. (2005), Reddemann (2004), Sachsse (2004), and Steiner & Krippner (2006). The latter three use techniques originating from katathym-imaginative psychotherapy (KIP). According to Lamprecht (2000), EMDR (Hofmann 2006; Shapiro 1998) can be understood as an integration of psychodynamic and cognitive approaches. It is based on free associations focused on the trauma and work with cognitions. Several useful techniques originate in hypnotherapy (Phillips & Frederic 2007) that also has an interactively eclectic character. Approaches described by the authors with a background in KIP and hypnotherapy are oriented rather on type II trauma; they use especially stabilisation techniques and techniques activating the inner resources. Other approaches in PTSD treatment include the Imagery Rescripting and Reprocessing Therapy (IRRT) (Smucker et al. 1995) and the Narrative Exposition Therapy (NET) (Schauer et al. 2005). They also use the exposition principle. A theoretical concept and treatment methodology for patients with serious structural personality dissociations (usually as a result of type II trauma beginning in early childhood) have been formulated by Helga Matthias, together with Ellert Nijenhuis (2006).

A methodically precise concept of PTSD exposition therapy and work with cognitions and homework within the frame of cognitive-behavioural therapy has been described by Ehlers (1999). In most circumstances, there are 15 sessions lasting 90 minutes each. The first 8–12 sessions take place weekly, the last 3 monthly. The in vivo exposure can take an additional 2 hours. While the metaanalysis of CBT for PTSD treatment conducted by Etten and Taylor in 1998 found the effect size $d = 1.27$, Ehlers’ work group reports effect size $d = 2.6–2.8$. The effects stay stable even
In a one-year follow-up. After the treatment, 80–90% of the patients do not fulfil PTSD diagnostic criteria. Drop-out rate was only 5%.

In the last two decades, the development of psychotraumatology has markedly accelerated, and the effectiveness of psychotherapeutic techniques used in PTSD treatment has been the subject of intense scientific interest. The authors of an overview of present knowledge on PTSD treatment (FROMMBERGER & MAERCKER 2011) state that PTSD symptoms can be reduced by both psychotherapy and pharmacotherapy (paroxetine, sertraline). They quote the review of neurobiological studies conducted by Jetzke et al., and state that the anterior gyrus cinguli activity is increased, while the amygdala remains unchanged. Thus, the emotion control improves, but we can hardly count with influencing the anxious conditioning of the amygdala. Considering psychotherapy, the biggest effect sizes have been found for CBT and EMDR. According to LAMPRECHT (2000), EMDR accelerates the therapeutic process. Thus, less time is needed for the therapy. Meta-analyses comparing CBT exposure techniques and EMDR suggest that EMDR reaches the same results in 40–50% of the time needed when using CBT.

In our patient, we used EMDR as well as autogenic training. Therefore, we shortly describe both methodologies. EMDR is an integrative method which involves a specific approach for confronting trauma. Following a structured manualised approach, the therapist helps the patient remember the traumatic event in as much detail (tactile, cognitive, affective and somatic) as possible. After the memory is fully active in the patient’s consciousness, the therapist starts the bilateral stimulation at a frequency of approximately 1 Hz. (The therapist usually asks the patient to follow his fingers with their eyes. The fingers move alternately from side to side so that the patient’s eyes also move back and forth. The stimulation can also be tactile or auditory. The therapist either alternately touches the patient’s right and left hand, or the patient has headphones on and listens to sounds played alternately in their left and right ear.) One set usually consists of 25–30 stimuli, followed by a pause of several seconds. In this time, the therapist and the patient talk about the experience. In the stimulation phase, the patient’s task is to freely associate and to impartially observe the associations (free associations and activation of the watchful ego). In this phase, the therapist does not interfere, trusting the patient’s salutogenic ability facilitated by the therapeutic situation and the bilateral stimulation. The therapist only takes short pauses in the stimulation and asks the patient about any changes in their feelings. This process continues until the memory is fully emotionally neutral. In the next step, the original memory is paired with a new – positive – cognitive evaluation. Bodily reaction to the memory is tested, since traumatic memories are most deeply kept on the somatic level. EMDR uses two subjective scales measuring the therapeutic effect. One of them is the Subjective Units of Disturbance Scale (SUDS) originating from WOLPE (1969). It is a scale of 0 to 10, where 0 means ‘no distress’ and 10 means ‘maximal distress’. The other one is the Validity of Cognition Scale (VoC) measuring the validity of the positive cognition connected to the traumatic event. It is a scale of 1 to 7, where 1 means ‘completely false’ and 7 equals ‘completely true’. The de-
sensitisation phase ends when the SUDS has reached 0 and VoC has reached 7. This result might be reached after one session, but sometimes several sessions are needed.

Hypotheses about a possible ‘mechanism of action’ of EMDR remain open. Further research is needed to confirm them. According to present knowledge, bilateral stimulation during trauma reliving leads to specific activation changes in the central nervous system. These changes, caused mainly by autonomic nervous system regulations (VOJTOVÁ & HASTO 2009), increase the information processing capacity. Already ten seconds after the beginning of the bilateral stimulation on a frequency of 1 Hz during the trauma reliving, there is a decrease of sympathicotonia, decrease of heart rate and increase in vagal activation (ELOFSSON et al. 2008; SACK et al. 2008). The brain information processing system is probably activated. Orientation reaction is provoked and neural activity is similar to REM sleep (STICKGOLD 2002). PANKSEPP (1998) points out that similar neural structures are activated by play, exploratory behaviour and REM sleep. These neurophysiological processes are supposed to facilitate the development of associative connections with neural networks where positive experiences are stored. Thus, the trauma is being rewritten (RALAUS 2006). According to one hypothesis, there is a greater participation of the left hemisphere in problem solving (HOFMANN 2006). All this happens within the accepting and safe frame potentiated by the rhythmic bilateral signals and the therapist’s presence. Furthermore, the therapist explicitly verbalises his/her trust in the patient’s salutogenic processes that can now be activated. From the attachment theory point of view (HASTO 2005), this can be understood as ritualised attachment behaviour: a competent, supporting person is close to an extremely distressed individual. If we move to the endocrine-molecular level, oxytocin (BRÜNE 2012) may play an important role here.

Autogenic training (AT) according to J.H. Schultze (HAŠTO 2006) is a well-known and empirically tested method of concentrative self-relaxation. It uses mental repeating of autosuggestion formulas to reach full physical and mental relaxation, the so-called auto-hypnoid state. In the AT basic stage, the patient repeats sentences creating sensations of heaviness and warmth, calm heart rate and breathing, feelings of radiating warmth in the abdomen, and feelings of freedom and clarity in the head. In further course, it is possible to use ‘individually tailored’ formulas corresponding to the patient’s specific difficulties (intermediate stage). Finally, it is possible to deepen and broaden the consciousness using imaginative meditations (higher stage). The result of autogenic training is a state of deep psychophysical well-being having both a therapeutic and preventive effect on stress-related difficulties. However, we consider the Autogenic Training as contradictory in florid PTSD symptomatology. In our case study, we will show its usefulness in the last phase of treatment and also its importance as a preventing factor.

End of Part 1.
The case study with discussion will be presented in Part 2.
References


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