Dissociation, forced normalization and dynamic multi-stability of the brain

Petr Bob

Center for Neuropsychiatric Research of Traumatic Stress & Department of Psychiatry,
1st Faculty of Medicine, Charles University, Prague, Czech Republic

Correspondence to: Petr Bob, Ph.D.
Department of Psychiatry, Charles University, 1st Faculty of Medicine
Ke Karlovu 11, 128 00 Prague, Czech Republic
TEL. +420 224965314; FAX: +420 224923077
E-MAIL: petrbob@netscape.net

Submitted: May 17, 2007 *Accepted: May 27, 2007*

Key words: **alternative psychosis; dissociation; depression; epilepsy; forced normalization; multi-stable perception; psychosis**

Neuroendocrinol Lett 2007; **28**(3):231–246 NEL280307A02

Abstract

Dissociated states represent pathological conditions where psychological trauma may emerge in a variety of forms such as psychic dissociative symptoms (hallucinations, derealization etc.) or on the other hand as somatoform symptoms (paroxysms, loss of motor control, involuntary movements etc.). Recent findings suggest that neurophysiological level of dissociative phenomena may be linked to the same neurophysiological principles that emerge in multi-stable perception of ambiguous stimuli likely caused by competing interpretations with mutual exclusivity. At this time there is evidence that temporal lobe seizure activity can produce dissociative syndrome and from these findings may be inferred that temporal lobe epileptic activity existing independently of neurological focal may share common neurobiological mechanims with dissociative symptoms. This conceptualization of dissociative phenomena is also in accordance with findings that originate from the study of the relationship between epilepsy and mental illness. The relationship was for the first time described in Meduna's concept of antagonism between epilepsy and psychosis and from the study of forced normalization introduced by Landolt in 1950s. The findings reported similar pathological conditions as in dissociative states when psychopathological symptoms and paroxysms may represent two different forms of the pathological process. Following the concept of forced normalization Tellenbach in 1965 introduced the term alternative psychosis implicating that stopping seizures does not mean vanishing or inactivity of the pathological state and that the epilepsy is still active subcortically and supplies energy for psychopathological symptoms. In the present review chaos in brain neural networks as a possible explanation of the relationship between dissociation and epileptic activity has been suggested that represents testable hypothesis for future research.

INVITED REVIEW

The concept of dissociation was initiated mainly in the work done by Pierre Janet (Janet, 1890). The development continued in the works of dynamic psychiatrist and depth psychologist (e.g. S. Freud, C.G. Jung, A. Adler and others) (Ellenberger, 1970). The contemporary state in the field supports the validity of these findings for explanation of many psychopathological symptoms and the other psychological phenomena (Frankel, 1996; Butler *et al.* 1996).

Concept of dissociation elaborated by Pierre Janet includes pathological conditions when psychological trauma may emerge in various forms such as psychopathological symptoms, somatoform symptoms and paroxysms (Ellenberger, 1970; Bob, 2003a; van der Hart and Friedman, 1989). Similar pathological conditions occur also in the cases of forced normalization and alternative psychosis that describe the relationship between epilepsy and mental diseases that may emerge as reciprocity between seizures and psychopathological symptoms (Krishnamoorthy *et al.* 2002). These empirical findings seem to be very important in search of common basis of these extraordinarily interesting phenomena that occur at the interface of psychology, psychiatry and neurology.

DISSOCIATION, SUBLIMINAL PROCESSES AND THE UNCONSCIOUS

Scientific history of the unconscious became in the field of psychopathology connected to the study of trauma and dissociation. Dissociation is often understood as inability to integrate some contents of thinking or feeling into the consciousness and lead to the category of the unconscious. The term dissociation has its origin in the constituent parts of the term 'dis-association' which means disconnecting or lowering the strength of associative connections. Even before Janet, in the year 1845, Moreau de Tours used the term psychological dissolution (désagrégation psychologique) (van der Hart and Friedman, 1989). Analogically Hughlings Jackson (Meares, 1999) used the term "dissolution" and also the term "dreamy state" which meant splitting consciousness leading to amnesia and other symptoms, such as depersonalization, derealization, hallucination or disagregation of perception. Morton Prince, one from Janet's contemporaries, used the term "co-conscious" in the sense that two consciousness are isolated from one another (Hilgard, 1974). Max Desoir identified two main streams of mental activity as upper or lower consciousness where the lower one may emerge - for example, in hypnosis (Hilgard, 1974). F. Myers introduced the term subliminal Self which was later used also by William James (Hilgard, 1974).

Janet initially elaborated the concept of dissociation in his work Psychological Automatism (Havens, 1966; Janet, 1890; van der Hart and Friedman, 1989), where he sketches his notion of psychic functions and structures. He dealt with psychological phenomena often observable in hysteria, hypnosis and states of suggestion or possession. From 1889 his work was greatly influenced by his collaboration with J.M. Charcot in the Parisian hospital Salpetrière.

During complete psychological automatism (van der Hart and Friedman, 1989), consciousness is totally dominated by repeating past experiences, such as in somnambulism or hysterical crises. In the case of partial automatism, only a part of the consciousness is dominated. In the case of complete or partial automatism systems of unconscious fixed ideas play an important role and repress conscious control and perception. They may emerge in many forms of psychopathological or somatoform symptoms, for example paroxysm, which may be understood as a representation of psychological trauma when a fixed idea is transformed into hallucinations and body movements (Ellenberger, 1970; van der Hart and Friedman, 1989; Bob, 2003b). Janet considered hysteria as a defect of the psychic wholeness. Similarly in cases of abulia he viewed the degeneration of will as a consequence of the influence of unconscious processes which lead to a repression of conscious psychic activity. Janet described many forms of somnambulism, which represent abnormal states of consciousness with their own memory that are inaccessible for the normal state of consciousness. Fixed ideas are also presented in the form of dreams and dissociative episodes (e.g. hysterical attacks) or during hypnosis as a secondary consciousness. A characteristic feature of these states is a lowering of the mental level (abaissement du niveau mental), which is manifested by increased dissociation and mental depression connected to the reduction of psychological tension. It leads to integration at the lower psychological level corresponding to the level of the dominant fixed idea and its psychological automatism (Ellenberger, 1970; van der Hart and Friedman, 1989; Bob, 2003b).

Some new interest in the theory of dissociation appeared after the Second World War along with a restoration of interest in the study of hypnosis. A great interest in psychoanalysis after the First World War led to a state when its own roots at the French school in Salpetrière were forgotten (S. Freud studied there in 1885-86 in J.M. Charcot) (Ellenberger, 1970; Haule, 1984). According to Freud, "dissociated states" are elicited by the repression of the libido energy that is of a sexual nature. Other authors representing the depth psychological trends explained libido differently than Freud. According to C.G. Jung, the libido represents general psychic energy, for Alfred Adler it represents a will for power, and Viktor Frankl believed the libido to be the meaning of life (Bob, 2003b). Modern history of dissociation began in the work of Ernest R. Hilgard who continued in Janet's tradition. His neodissociation theory is sketched in the work "Toward a Neodissociation Theory: Multiple Cognitive Controls in Human Functioning" (Hilgard, 1974) and is described in detail in his book (Hilgard, 1986). Following modern findings show that dissociative processes must be seen as clinical reality (Frankel, 1996).

On the other hand recent findings in cognitive neuroscience show that next to psychopathology there is other source for the evidence of the unconscious (implicit) processes. Modern study of the cognitive unconscious defined explicit and implicit perception, i.e. explicit perception means perception immediately presented to consciousness of the subject while implicit perception is not accessible for the awareness of the subject and cannot be verified immediately in the response of the subject but only indirectly by observation or measurement (Kihlstrom, 1987, 2004; Bob, 2003). Implicit perception represents process on the unconscious level in which introspection is not possible. But also this subliminal information that remains under the limit of consciousness and is perceived unconsciously influences the organism (Kihlstrom, 1987, 2004; Bob, 2003b). A suitable method for the study of implicit perception became the subliminal stimulation (for example very short projection of an image or special forms of auditory stimulation). Libet (1979) shows that all stimuli lasting less than 500 ms with respect to a nature of the stimulus most frequently are not present in awareness of the subject. Great interest in subliminal phenomena arose from experiment performed in 1957 that led to the restriction of subliminal advertising. During a movie presentation were projected two verbal messages: "Drink Coca-Cola" and "Eat popcorn" which led to increased popcorn consumption about 58% and Coca-Cola consumption about 18% (Wortman *et al.*, 1992). It lead to many controversies but other findings also confirmed the existence subliminal perception and information processing (Crick and Koch, 1995; Marcel, 1983; Kihlstrom, 1987; Shevrin, 2001; Brazdil *et al.* 2001) and the method of subliminal activation has been proved as a suitable instrument for the study of the dynamic and cognitive unconscious.

Further findings about subliminal processes come also from the field of neurophysiological study of the dynamic and cognitive unconscious and show complexity of information processing without awareness of the subject. It is focused mainly on the study of the dynamic unconscious connected with emotional conflict. Experimental measurement linked to the subliminal stimulation, for example in the form of two different subliminally presented pictures enables to distinguish these different subliminally presented pictures, e.g. one which is connected to the inner conflict (for example picture of a well-known person evoking phobia) and the other which is not associated with the conflict, without any conscious activity of the patient. For example using measurement of changes in skin resistance it was shown that the neutral stimulus does not lead to any observable changes but the picture associated with the phobia results in a measurable response (Poetzl, 1960).

The phenomena of subliminal stimulation were also studied by analyzing Event Related Potentials (ERP) using P3 (also called P300) wave, which is a positive wave with latency 300 ms, after presentation of a stimulus. These studies performed on emotionally disturbed patients show that the P3 wave reflects the neurophysiological changes associated with the subliminal stimulus connected with the emotional conflict (Wong *et al.* 1994, 2004). Additionally, the P3 wave was able to demonstrate that the prosopagnostic patient could distinguish (albeit unconsciously) between familiar and unknown faces (Reanault *et al.* 1989). Other data also indicate that threshold of consciousness may change with respect to experimental conditions, for example Stross and Shevrin (1962, 1968, 1969) have shown alterations of thought contents under hypnosis in investigation of "freely evoked images" after the subliminal presentation. Their major conclusion was that hypnosis leads to heightened access to subliminal stimuli and that thought organization during hypnosis shares some common elements with thought organization during dreaming. It corresponds to similar findings when subliminally presented images were found in dreams (Fischer, 1954; Poetzl, 1960). There are some studies, which confirm a common view that the manifestations of post hypnotic suggestion are very similar to some psychopathological phenomena (Huston *et al.* 1934; Vermeten and Bremner, 2003) and it is probable that psychopathological phenomena lead to a dissociated state by lowering the corresponding psychic content beneath the threshold of consciousness (Bob, 2003b).

DISSOCIATION AND TRAUMA

Dissociation represents a special form of consciousness in which events that would ordinarily be connected are divided from one another (Li and Spiegel, 1992) or is also often less generally understood as inability to integrate some psychic contents into the consciousness (Bernstein and Putnam, 1986). Dissociation is defined in DSM-III-R and DSM-IV as "a disturbance or alteration in the normally integrative functions of identity, memory or consciousness" and leads also to characteristic somatoform changes (Nijenhuis *et al.* 1996) such as alterations in sensation of pain (analgesia, kinesthetic anesthesia), painful symptoms, perception alterations, motor inhibition or loss of motor control, gastrointestinal symptoms and dissociative seizures (Brown and Trimble 2000; Kuyk *et al.* 1999).

The group of syndromes which are immediately bound to dissociative processes were elaborated by John Nemiah (1981). Main features are these: 1. alteration of identity as a consequence of dissociative reaction and 2. disturbance of memory of an individual during dissociative states. These principles were used for the first definition of diagnostic classification of dissociative states in the DSM III frame. The third important principle defined by Putnam (1989) is based on experiences from the study of dissociative reaction where the major part of dissociative disorders was induced by traumatic events. The most important traumas originate in childhood due to physical or sexual abuse with following develop-

ment of symptoms often after many years. Dissociative symptoms also frequently occur due to traumatic event after serious accidents or natural calamities. Symptoms of disintegration often develop in the connection to post-traumatic stress disorder (Spiegel and Cardena, 1991). Characteristic features of these dissociative symptoms are changes in notion of identity as depersonalization or in serious cases multiple personality disorder. Another experienced symptoms represent changes in notion of external world such as derealization, hallucinations or changes of memory, for example psychogenic amnesia or multiple personality disorder (Spiegel and Cardena, 1991). For example Chu and Dill (1990) investigated dissociation by means of Dissociative Experiences Scale (DES) in 98 females and found significantly higher dissociation in patients which were exposed to emotional or physical abuse. Coons, Bowman and Pellow (1989), in their study of prevalence of traumas both in childhood and adults in clinical population found that 100% patients with atypical dissociative disorders and 82% diagnosed as psychogenic amnesia documented physical or verbal abuse or neglect in childhood. About half of patients experienced also significant trauma in adulthood. Briere and Conte (1989) have documented that 59,6% from the group of 468 patients with proven history of sexual abuse in childhood were not able to remember the episodes of abuse from the past. There is growing evidence that child abuse is a very important factor in many psychiatric disorders and that dissociative symptomatology often occurs due to child abuse especially in cases of chronic emotional, physical or sexual abuse (Spiegel and Cardena, 1991; Putnam, 1997, Teicher *et al.* 2003).

Generally these findings implicate that any exposed or reported trauma may be important for the development of dissociative symptomatology and may be closely related to many symptoms such as depresssion, hallucinations and other. On the other hand it is necessary to mention that in ICD-10 is also defined the organic dissociation that concerns dissociative symptoms and disorders including amnesia, fugue, depersonalization, multiple personality, automatisms, and certain furors which can be induced by a variety of medications, abuse of drugs, and medical illnesses or conditions affecting cerebral functions. It is important to note that organic dissociation can be distinguished from intoxication, amnestic disorder, and delirium (Good 1993). It is important that dissociation as a reaction to psychological stressors and/or physical traumatic insult has various neurobiological consequences. Typical reactions are disturbances of self-regulatory systems such as hypothalamus-pituitary-adrenal axis (HPA) resulting in hyperarousal, tachycardia or other symptoms of autonomic nervous system instability (Read *et al.* 2001). HPA is functionally closely related to neuroendocrinological balance, control hormonal levels, energetic metabolism, neuroimmunomodulation and disturbances of memory during stress reaction (Teicher *et al.* 2003; Read *et al.* 2001; King *et al.* 2005; Gavrilovic and Dronjak, 2005; Nakayama *et al.* 2005; Takahashi *et al.* 2005; Payne *et al.* 2006; Umegaki *et al.* 2006). According to neurodevelopmental research are most serious disturbances of HPA axis caused by traumatic events such as childhood abuse or neglect in the first years of life and often have long-term impact on emotional, behavioral, cognitive, social and physiological functions and vice versa love and social care also may influence these functions and improve dissociative disturbances (Teicher *et al.* 2003; Read *et al.* 2001; Esch and Stefano, 2005a,b; Stefano and Esch, 2005).

The special literature expresses in many forms the view (Putnam, 1989) that dissociation is not only pathological but it also represents some adaptive functions. It corresponds to the dissociative continuum based on the fact that some dissociative phenomena also occur in the normal population.

Pierre Janet as mentioned above, in his work about psychological automatisms (Janet, 1890; Ellenberger, 1970), defines dissociation as a defect of the associated system that creates the secondary consciousness, which he called the subconscious fixed idea. Similarly, Sigmund Freud and Joseph Breuer consider double consciousness in "Studies in hysteria" (Breuer and Freud, 1895) as a pathological phenomenon. In contrast to that, Carl Gustav Jung considered the dissociation of personality not only a pathological phenomenon (Jung, 1972a), but saw the dissociation of the psyche as a fundamental psychological process that makes differentiation and specialization of psychic processes possible. An example of this is the focusing of will or concentration on a single target, which often is a prerequisite for the development of the personality (Jung 1972b). During these processes, psychic entities are created and associated with certain contents of memory, patterns of behavior, and emotional charges. Jung called these entities "psychic complexes". The most often dominant one is the ego-complex. Jung identified these psychic contents in his experiments in Burghölzl and described them in his studies of word associations (Jung, 1973). He found that when a defect occurs in free associations it is caused by a complex (Jung, 1972c, 1973). These complexes are, according to Jung, created out of inborn and inherent dispositions and their ethological manifestation is the resulting pattern of behavior. These dispositions act as ordering factors that organize psychic contents, perceptions, and fantasies into complex psychic structures. In the outer world, they are projected by neural firing patterns that affect muscular activity and glands that are involved in the ethological manifestation (pattern of behavior) (Bob, 2003b). The existence of these ordering factors were recognized by Jung in his study of psychic regression in schizophrenic patients as well as their parallels in mythology and dream production (Jung, 1968). He called these emerging complex psychic structures "archetypes." A complex always has its own autonomy and behaves as a split part of the psyche. When a complex is evoked into the consciousness, its physiological or pathological influence depends on a degree of its autonomy or, contrary

to that, compatibility with other complexes respective to the ego-complex (Bob, 2003b, 2004). In the case of pathological influence the complex leads to a lowered mental level (Janet, 1890; van der Hart and Friedman, 1989). As Janet suggested, the fundamental causes of the etiology of pathological complexes are mainly traumatic events, which produce traumatic memories. Complexes thus generate alternate fields of the psyche, and it is possible, by means of these complexes, to explain extreme cases of dissociation which occur in multiple personality disorder (MPD) (Bob, 2004).

Deeper understanding of dissociative processes suggests the connectionist models of memory and illustrates the theoretical view of dissociative continuum. These models of memory based on neural networks were elaborated during 1980s and are known as connectionist models or parallel distributed processing (Yates and Nasby, 1993). Dissociation became important for further explanation of memory phenomena and for development of memory models in normal and also pathological memory processes. Increased clinical interest in dissociation led to further development of models of dissociation based on associative neural networks. According to these findings emotions and inhibitory mechanisms as participants in organization of memory play a key role in dissociative processes (Yates and Nasby, 1993).

Affect plays a crucial role in modulation of memory processes, which in extreme cases, lead to dissociative defects. Another important factor is neural inhibition resulting from fundamental excitatory or inhibitory synaptic processes that are necessary for brain functions. Inhibition of very intensive and negatively modulated states enables us to describe memory phenomena such as amnesia, fugues, MPD and other manifestations of dissociation in normal or pathological states. Closely related to these findings is the notion of schizophrenia as a loss of dissociative inhibitory connections within the memory leading to serious defects in associative connections as, for example, the so-called "word salad" (Yates and Nasby, 1993).

In other words, dissociated fragments, i.e., complexes, are pathologically disintegrated due to abnormal intensive affect compensated by neural inhibition. Failure of this inhibition manifests itself as a continuum of pathological dissociation from mild forms, such as repression, to serious forms, such as splitting or word salad.

From the point of view emphasized in associative models of memory, pathological dissociative processes may be conceptualized as a failure of inhibition and normal dissociative functions leading to unresolved intrapsychic conflict with serious consequences for the course and organization of psychic functions, for example reactive creation of splitting or repression. As a consequence of splitting, the personality becomes alternating and dissociated and in extreme cases several personalities may be distinguishable in one person. Diagnostic criteria of the multiple personality were introduced for the first time in the DSM III (Diagnostic and Statistical Manual of Mental Disorders) (American Psychiatric Association, 1987) in 1980 and in ICD 10 (World Health Organization, 1993) it belongs to the group of dissociative disorders (F 44). The revised classification criteria for multiple personality (i.e. dissociative identity disorder) according to DSM IV were formulated as follow (American Psychiatric Association, 1994):

1. The presence of two or more distinct identities or personality states (each with its own relatively enduring pattern of perceiving, relating to, and thinking about the environment and self).
2. At least two of these identities or personality states recurrently take control of the person's behavior.
3. Inability to recall important personal information that is too extensive to be explained by ordinary forgetfulness.
4. The disturbance is not due to the direct physiological effects of a substance (e.g., blackouts or chaotic behavior during Alcohol Intoxication) or a general medical condition (e.g., complex partial seizures).

Note: In children, the symptoms are not attributable to imaginary playmates or other fantasy play.

Multiple personalities in one person typically may include the birth personality that develops in the individual from birth, while the personality that controls the body for most of the time, analogical to the ego-complex, is called the host personality. Birth and host personalities are called primary, while other personalities are called secondary. The presenting personality is the one actually present at a given moment (Ellenberger, 1970; Boleloucky, 1986; Putnam, 1989; Bob, 2004).

The creation of personalities and dissociated structure was also defined by Eugen Bleuler in schizophrenia (Bleuler, 1924; Bottero, 2001; Rosenbaum, 1980; Scharfetter, 1998). In his Text-book of psychiatry he wrote (Bleuler, 1924): "It is not alone in hysteria that one finds an arrangement of different personalities one succeeding the other. Through similar mechanism schizophrenia produces different personalities existing side by side." (p. 138) Bleuler's introduction the group of schizophrenias in 1911 replaced Kraepelin's term dementia praecox. A review of Index Medicus from 1903 to the revival of interest in multiple personality in 1978 shows a dramatic decline in the number of reports of multiple personality, which indicates that many patients with multiple personality had been diagnosed and treated as schizophrenics (Rosenbaum 1980). It is in agreement with findings that a substantial number of patients with dissociative identity disorder (DID) have previous diagnoses of schizophrenia (Elason and Ross, 1995). It is mainly due to the presence of positive symptoms of schizophrenia in patients with dissociative identity disorder that report more positive symptoms of schizophrenia than schizophrenics. It is important to note that schizophrenics report more negative symptoms and therefore a primary emphasis on

positive symptoms may result in false-positive diagnoses of schizophrenia and false-negative diagnoses of dissociative identity disorder (Elason and Ross, 1995). On the other hand there are findings that show markedly high level of dissociation in schizophrenic patients (Bernstein and Putnam, 1986; Read *et al.* 2001; Spitzer *et al.* 1997; Startup, 1999).

Etiology of schizophrenia is according to Bleuler explained along the lines of Janet (Bleuler, 1924; Boleloucky, 1986; Rosenbaum, 1980) as a consequence of dissociative reaction, analogous to somnambulism, fugue states, hypnosis or psychogenic amnesia, most often as a consequence of abuse or traumatic experiences mainly occurring between the ages of four to eight. It is a splitting of psychic connections similar to hysteria, but in an extreme version. Psychotic decompensation of some personality with corresponding symptoms, such as hallucinations may occur.

From the perspective of the Jung's complex theory, it is very interesting that in hypnosis components of the personality very similar to subpersonalities of the multiple personality were found also in normal individuals (Barret, 1995; Bowers and Brecher, 1955; Lynn *et al.* 1994; Merskey, 1992; Rickeport, 1992; Watkins, 1993; Watkins and Watkins, 1979-80). For example, Bowers and Brecher (1995) reported interesting material involved in the emergence of multiple personality structure under hypnosis. The authors conclude that this structure was not produced by the hypnosis, but preceded the beginning of the hypnotic work. The patient in the case under discussion had not shown the multiple structure in clinical and psychological examinations prior to the hypnosis. In his conscious state the patient was not aware of his three underlying personalities, each of which reported distinctive dream material and Rorschach responses (Bowers and Brecher, 1995). Barret (1995) describes similarities between the states of dreaming and MPD including amnesia and other alterations of memory. This suggests the dream character as a hallucinated projection of aspects of the self that can be seen as a prototype for the alter personalities. Extreme early trauma may mutate or overdevelop these dissociated parts, inducing them to function in the external world, and thus leading to development of MPD (Barret, 1995).

Jung's complex theory in many of these aspects corresponds to modern formulation of discrete behavioral states by Frank Putnam (1997). Discrete behavioral states (DBS) provide alternative perspective for the understanding of dissociation. The term DBS originates from the study of infant mental states. Infant behavioral states can be defined by a set of observable continuous and dichotomous variables. The number of infant states and their levels of interconnection increase with development and are responsible for the infant's growing behavioral repertoire. Healthy children are born with basic set of behavioral states. Fundamental features of a system of discrete states of consciousness are different state-dependent behaviors in response to the same stimulus. In adults, this type of differential responsiveness is most apparent in such disorders as bipolar illness or MPD (Putnam, 1997). State defining variables may be continuous or dichotomous and define behavioral state space. It means that individual behavioral states existing within larger multidimensional framework or space, defined by a chosen set of variables, occupy discrete volumes of the state space. An individual's behavior traverses the state space in a series of discontinuous jumps or switches from one state to another (Putnam, 1997). The state space may be vast but individual regularly occupies those regions in which one has created stable discrete states (Putnam, 1997). The discrete states as transitory behavioral structures are linked together by directional pathways forming behavioral architecture that defines an individual's personality. Transition between behavioral states is manifested as "switch" that represents abrupt change in the values of the constellation of state defining variables, for example transition from waking to sleeping or in bipolar illness from mania to depression (Putnam, 1997). DBS model defines "pathological dissociation" as a trauma induced discrete behavioral states that are widely separated in multidimensional state space from normal states of consciousness and it corresponds to conventional definition, which emphasize the separation or segregation of specific ideas or affects from normal mental phenomena (Putnam, 1997; Kaplan and Sadock, 1991). When two types of states are significantly different, then the states are separated by a wide gap in state space that determines pathological dissociative states. Observable differences between two discrete states are not a simple function of moving up or down. But there is probably nonlinear dynamic features connected to chaos (Putnam, 1997). For example Wolff (1987) highlights differential responsiveness as an example of the nonlinearity of input-output relation in different states of consciousness and conceptualizes the relevance of nonlinear dynamic systems theory to discrete behavioral states where switches between behavioral states constitute nonlinear transitions. Further recent studies give stronger evidence that rapid shifts in mood and behavior corresponds to nonlinear dynamic processes (Putnam, 1997; Gottschalk *et al.* 1995).

PERCEPTUAL CONSCIOUSNESS AND DISSOCIATION

According to the modern definition selective attention can be defined as selection among potential conscious contents (Baars, 1988, 2002) and specific function attentional mechanisms whose function is to bring different events to consciousness. It leads to global distribution of information that is located in brain regions underlying conscious vision and separated from those involved in the selection of visual objects and events (Baars, 1997,1999, 2002).

The study of the neural correlates of consciousness (Crick and Koch, 1995) or the neural correlates of perceptual awareness opens again fundamental question whether all perception is accompanied by awareness or not. Extensive evidence from behavioral studies of normal subjects as well as neurological patients show that perceptual information can be represented in the mind/brain without the subject's awareness of that information (Kanwisher, 2001).

It opens the question regarding conditions that are needed for conscious experience of a neural representation. It is possible that even a strong neural representation may not be sufficient for awareness and there is behavioral evidence that perceptual awareness involves not only activation of the relevant perceptual properties but the further construction of an organized representation in which these visual properties are attributed to their sources in external objects and events (Kanwisher, 2001; Baars, 1988).

Simple examples of these cases provide ambiguous stimuli in the cases when perceptual experience alternates between two different states and lead to perceptual bistability in cases such as Necker cube, Rubin's face/vase or in experiments of binocular rivalry in which different images are projected to each eye (Kanwisher, 2001; von Helmholtz, 1962). Although the stimulus itself does not change the human observer sees only one of them, instead of seeing a mixture of the two images and their perceptual experience seems to reflect a dynamic competition between the two inputs. When for example, vertical stripes are presented to the left eye and horizontal stripes to the right eye, the viewer is likely to see not a superimposition of the two patterns (i.e. a crosshatching plaid pattern), but an alternating sequence in which only vertical stripes will be seen for one moment, and only horizontal stripes the next. Although the precise mechanisms underlying binocular rivalry are a matter of some debate it is clear that experience alternates in a bistable fashion between being dominated by the input to one eye and being dominated by the input to the other eye (Blake *et al.* 1998; Kanwisher, 2001).

Important phenomena that affect the contents of perceptual awareness include attention, mental imagery, and changing states of consciousness. For each of these phenomena, neural signals have been shown to be connected with perceptual awareness. As in cases described above only simple focusing of visual attention on different aspects of an unchanging stimulus has a strong effect on the content and intensity of perceptual awareness (Rees *et al.*, 1999). Closely following the effect of attention on subjective experience, numerous studies using single-unit recordings (Desimone and Duncan, 1995), ERPs (Luck and Girelli, 1998), and brain imaging have shown clear modulations of sensory responses by attention, even for a constant stimulus and even in primary visual cortex (Kanwisher, 2001). A rather different manipulation of perceptual awareness occurs during mental imagery, in which no stimulus is present at all and selective activation has been reported during mental imagery of motion (Goebel *et al.* 1998) or for face and place imagery (Kanwisher, 2001). In each of these cases, the activations during mental imagery are weaker than the corresponding stimulus activations.

Theoretical explanation of the observed phenomena proposed Desimone and Duncan (1995) in the concept of 'interactive competition'. According to this model competitive interactions across cortical areas result in domination of perceptual representations by properties of a single object. This competition can be biased by either bottom-up factors (e.g. stimulus salience) or top-down factors (e.g. endogenous attention). In either case the net result is that the various properties of an object, represented in distinct cortical regions, enhance each other and suppress the representation of competing objects. On this view, attention and awareness are global properties of the entire perceptual system that connect multiple cortical areas (Kanwisher, 2001). These principles of perceptual bistability are closely linked to dissociated states in which multistable competitive interactions and alterations in perception, emotion and identity were described. For example, in mild forms in cases of splitting in borderline personality disorder perception of close person as totally "good" or "bad" significantly alternates.

These findings suggest that awareness of perceptual information requires not only a strong representation of the contents of awareness, but access to that information by other parts of the mind/brain (Baars, 1988, 2002). The limits on conscious access to perceptual information may not be immutable and for example, brain damage may disrupt neural pathways and the perceptual information represented in one neural structure is not accessed by other parts of the system (Baars, 2002). On the other hand conscious access to perceptual information may also change over time even in undamaged brains (Kanwisher, 2001). According to conscious access hypothesis consciousness might be a gateway to brain integration that enables access between otherwise separate neuronal functions (Baars, 2002). In accordance with this explanation there is evidence for a mutual dependence between consciousness and executive input and a loss of one executive interpreter's access to conscious events while another was dominant in cases of multiple personality, dissociative fugue and during hypnosis was reported (Hilgard, 1988; Putnam, 1995; Baars, 2002). This suggests a binding between conscious contents and self functions, and similar dissociation may be found in split-brain patients where each hemisphere executive control over one side of the body, based on conscious input, is limited to half of the visual field (Gazzaniga and Sperry, 1967; Sperry, 1967). From this point of view consciousness may enable access to self functions as well (Baars, 2002).

Perceptual bi-stability as a class of phenomena in which a particular stimulus gives rise to two different interpretations therefore may reveal general principles

about brain architecture (Rubin, 2003). Generally such cases results in multi-stability when each of the competing interpretations have periods of dominance and the (three or more) percepts alternate in dominance in a seemingly random manner. Multi-stable phenomena thus suggest an underlying principle of mutual exclusivity (Rubin, 2003) that is typical for perceptual consciousness as well as for dissociative phenomena. The quasistability related to different levels of interpretation process has been suggested to be connected with dynamic nonlinear chaotic processes and self-organization (Aks and Sprott, 2003) similarly as switches between dissociated behavioral states (Putnam, 1997). These findings may represent significant outlet for future research of common basis for interpretative functions in multistable perception and dissociative phenomena.

DISSOCIATION AND TEMPORAL LOBE EPILEPSY

Recent data indicate that traumatic stress and dissociation might be significantly related to epileptic activity (Putnam, 1997; Teicher *et al.* 2003). Because epileptic activity represents typical example of chaos in neural organization (Korn and Faure, 2003; Tirsch, 2004) and dissociation is hypothetically attributed to neural chaotic organization it is needed to more comprehensively consider the data that have documented a relationship between dissociation and epileptic activity. The epilepsy/temporal lobe dysfunction model of dissociation was for the first time proposed by Charcot in 1892 (Putnam, 1997). This concept for explanation of neurobiological basis of dissociation is related to clinical data that prevalence of seizure disorders is much higher in MPD patients (Mesulam, 1981; Schenk and Bear, 1981; Benson, 1986; Perrine, 1991; Putnam, 1997). On the other hand dissociation-like symptoms such as depersonalization, fugues, amnesias and autoscopy (seeing an externalized image of oneself) are sometimes reported ictally and periictally, by seizure patients (Putnam, 1997). Kindling as neurobiological mechanism (Goddard *et al.* 1969) can potentially explain how epileptic-like phenomena might arise from repeated trauma (Post *et al.* 1995; Putnam, 1997; Teicher *et al.* 2003). The kindling related to traumatic stress similarly as experimental kindling likely is caused by progressively increasing response of groups of neurons due to repetitive subthreshold stimulation that may later lead to epileptic activity. It corresponds to clinical data that raise the possibility that temporal lobe abnormalities may play a role in pathological dissociation (Putnam, 1997). Typical EEG abnormalities found in traumatized and dissociated patients often involve temporal or frontal slow wave activity and also may involve frontotemporal spikes or sharp waves predominantly in the left hemisphere (Putnam, 1997; Teicher *et al.* 2003). Recent studies (Putnam, 1997; Ito *et al.* 1993; Teicher *et al.* 1993, 2003) have found frequent and unusual EEG abnormalities in victims of child abuse and also several imaging studies describe hippocampal abnormalities in trauma patients (Bremner *et al.* 1995; Putnam 1997; Bremner, 2006; Teicher *et al.* 2003, 2006). Although dissociative disorders cannot be generally explained on the basis of neurological dysfunction, contemporary data supports the suggestive evidence that temporal lobe seizure activity can produce dissociative syndrome, which is similar to that observed in functional cases (Spiegel, 1991). From these findings it may be inferred that temporal lobe epileptic activity is important in the generation of dissociative symptoms without neurological focal lesion (Spiegel, 1991). It is in accordance with evidence linking dissociative symptoms to the temporal lobe activity and corresponds to clinical data that the dissociative symptoms in temporal lobe epileptics occur during interictal periods and not during the ictal state (Spiegel, 1991). Epileptic activity in interictal periods in temporal lobe epileptics also may produce characteristic symptoms called complex partial seizure-like symptoms that appear as intrusions into the normal state of consciousness in the form of cognitive, psychosensory or affective symptoms (Roberts, 1993; Roberts *et al.* 1990; Silberman *et al.* 1985; Hines *et al.* 1995). Many of these symptoms were already defined by Hughlings Jackson in his classical studies (Roberts *et al.* 1990; Dreifuss, 1981; Roberts *et al.* 1992). Modern findings support the view that these symptoms have, similarly as dissociation (Bernstein and Putnam, 1986), in the general population a continuous character (Roberts *et al.* 1990, 1992). The continuum of complex partial seizure-like symptoms begins in healthy state without the symptoms via transitional pathological states until the symptoms of complex partial epilepsy with all typical manifestations. Between these opposites a broad spectrum of different clinical dysfunctions with good response to anticonvulsant drugs occurs. It concerns most often affective diseases or atypical psychoses with characteristic manifestations of the symptoms of temporal lobe epilepsy in nonepileptic conditions and are called Epilepsy Spectrum Disorders (Roberts *et al.* 1992; Hines *et al.* 1995; Jampala *et al.* 1992). According to some findings (Roberts, 1993, Bob *et al.* 2005, 2006) these symptoms are in close relationship to increased sensitivity on parental influence and dissociative tendency due to traumatic or aversive events most often in connection to child abuse. Two main characteristic features of these patients are enhanced dissociative capacity and abnormal electrophysiological activity although it must not be present in scalp EEG (Roberts, 1993; Hines *et al.* 1995).

There were also described clinical cases of EEG abnormalities with dissociative symptomes, several dissociative syndromes (including the patients with multiple personality) and also identity shift in temporal lobe epilepsy (Schenk and Bear, 1981; Mesulam, 1981; Coons *et al.* 1982; Benson *et al.* 1986; Spiegel, 1991; Ahern *et al.* 1993; Hersh *et al.* 2002). For example Ahern *et al.* (1993) examined the relationship of "multiple personality

disorder" in two patients with temporolimbic epilepsy to certain types of hemispheric interaction. Both patients had presented with different "personalities" in a characteristic temporal relationship to their seizures. These two patients with temporolimbic epilepsy were considered to be surgical candidates referred for the intracarotid amobarbital sodium procedure. Both patients have demonstrated outbursts of emotional behavior during inactivation of the left hemisphere. These "different personalities" were known by the patient's families to manifest themselves in the postictal period.

Dissociative states are also present during the so-called altered states of consciousness such as possession, out of body experiences, near death experiences (Putnam, 1989) and several studies reported epileptic activity to religious experiences (Saver and Rabin, 1997) or out of body experiences (Blanke *et al.* 2002).

Dissociation is traditionally connected to inner conflict and several clinical studies indicate that activation of inner conflict during stressful interview may produce seizure activity in epileptics (Stevens 1959, Faber *et al.* 1996) or activates burst waves in closed eyes in normal healthy people (Berkhout *et al.* 1969). There are also studies describing subcortical epileptiform activity during intensive emotional and psychopathological states (Faber and Vladyka, 1987; Heath, 1962, 1975; Groethuysen *et al.* 1957; Monroe, 1978, 1982; Stevens, 1999; Alvarez, 2001).

Frequent occurrence of epileptic activity and epileptiform abnormalities in dissociative states and disorder suggest that close relationship between epileptic activity and kindling might have crucial importance for understanding of dissociative processes. Kindling mechanism caused by stress may involve typical inhibitory failure caused by overloading of defensive mechanisms such as denial or "repression" that has been conceptualized for understanding dissociative states (Yates and Nasby, 1993). This process therofore leads to similar lack of inhibiton as epilepsy and therefore it may also cause similar pathological electrophysiological changes as found in epilepsy. Preliminary findings suggest that mental stress may cause increased chaos and neural complexity (Redington and Reidbord, 1992, Bob, 2007) and repeated stress may hypothetically represent neurophysiological mechanism inducing epileptic activity as a typical form of chaotic process (Bob *et al.* 2006). This implicates that dissociation is not synonymous as epileptic activity but in agreement with these findings we may consider the hypothesis that reported relationship between dissociation and epileptic activity might represent manifestation of underlying mechanims related to neural chaos.

NEURAL NETWORKS, DISSOCIATION AND CHAOS IN THE BRAIN

As outlined above recent findings suggest the hypothesis that dissociative states are linked to chaotic processes and that perception and processing of internal conflict related to dissociative states may share the common neurobiological substrate with phenomena of multistable perception of ambiguous stimuli that may be explained by interactive competition model (Desimone and Duncan, 1995). Proposed model of chaos and dissociative states by Putnam (1997) seems to be relevant with respect to chaotic nature of epileptic activity occurring during dissociative states and kindling hypothesis as a candidate mechanism for explanation of repeated traumatic stress and dissociation. More specific model for this relationship might be elaborated using concepts of neural networks. Already proposed models of neural networks provide an attractive framework for modeling dissociative mental processes and dissociative mechanism. They require the parallel operation of two or more information processors (Li and Spiegel, 1992). These processor systems are disintegrated and it represents the problem rather than the competition of these subsystems (Li and Spiegel, 1992). The first successful model of a neural network suitable for the study of dissociative processes was suggested by Bower (Bower, 1981; Butler *et al.*, 1996). In this model memory is saved in single elements of the network. The memory content may be excited when its corresponding memory element is activated upon reaching a threshold. There are elements with excitation influences on memory contents and also elements that inhibit them. Bower assumes that dissociative disorders can be the consequence of state dependent learning (Yates and Nasby, 1993).

Other classes of models represent neural networks in which memory is parallel distributed in the space of the network (Butler *et al.* 1996; Li and Spiegel 1992; Mc Clelland and Rumelhart 1986). This network is able to explain a wider class of dissociative processes, such as the course of posthypnotic amnesia. Parallel distributed processing is a model for the microstructure of cognition (Mc Clelland and Rumelhart 1986) where the activities of many neurons are described as configurations or neural patterns and their psychological correlates are called mental representations (Butler *et al.* 1996; Li and Spiegel, 1992; Mc Clelland and Rumelhart, 1986). In this description the neural network state is described by the superposition of neuronal patterns. Potiential neural patterns and configurations in their "superposition" are in "prespace", which might be attributed to psychic space and the active neural patterns are selected from this superposition (Butler *et al.* 1996; Li and Spiegel, 1992; Mc Clelland and Rumelhart, 1986).

Because dissociation represents an inability to integrate some psychic contents, such as from memory into the consciousness (Bernstein and Putnam, 1986) the disintegration represents the problem rather than the

competition of these subsystems (Li and Spiegel, 1992). Dissociative states thus represent mental representations, which are inaccessible to dominant interpreter's access. In another words it means that dominant ego-complex, i.e. dominant consciousness (or dominant subpersonality in MPD), has not access to certain contents of memory, consciousness or identity that represent dissociated part of the mind in accordance with Baars' (2002) conscious access hypothesis. From this inaccessibility and other aspects of dissociative states may be inferred that among the certain brain states an antagonistic competitive relationship may occur. These states can be modeled by parallel distributed processing in neural networks (Butler *et al.* 1996; Li and Spiegel, 1992; Mc Clelland and Rumelhart, 1986). Many activity configurations in parallel distributed processing networks are represented as points in an N-dimensional plane, where N represents the number of neurons in the network. In a simple case we have two neurons and all their possible activities, which are given by synaptic strength, can be represented by two axes. The third axis represents the probability of a given configuration. This produces a three-dimensional plan (landscape) and "peaks" in this plane represent favored activity states. Isolated peaks in this plane represent dissociated states (Li and Spiegel, 1992). All configurations in the N-dimensional plane represent a multistable dynamic system, which is changed over time with each new input. This PDP model may be used for the modeling of some pathological states, for example functional amnesia, multiple personality disorder and post-traumatic stress disorder (Butler *et al.* 1996; Li and Spiegel, 1992; Mc Clelland and Rumelhart, 1986; Bob, 2003a). Dissociation on the neurophysiological level therefore may be described using the concept of brain complexity. Generally, the complexity of the system means simply its composition from simple units or its dimensionality and tends to evolve over time (Coveney and Highfield, 1996). Structures, which have a higher number of dimensions, are generally viewed as more complex. In the case of neural networks or an electroencephalogram this means that there is competition among oscillating neuronal cell assemblies (neural configurations). Complexity is, for example, higher during divergent (creative) thinking than during convergent thinking (analytical thought) (Mölle *et al.* 1996), which leads to the suppression of competition among neural assemblies. Also, people with higher intelligence have higher EEG complexity (Lutzenberger *et al.* 1992). Complexity and competition among neural cell assemblies is represented by the number of simultaneously active neuronal assemblies involved in performing of a task. For example, during convergent analytical thought all information irrelevant for solving the problem is reduced and at the same time the number of competitive neural assemblies is reduced and the complexity decreases. Higher competition during creative thinking leads to the establishment of new associations among neural representations of mental states (Mölle *et al.* 1996). These findings implicates transient periods of high complexity of the EEG during activity of independent areas that enables fast parallel information processing which runs in a distributed mode. It means that numerous processes from sensory and cognitive channels are executed simultaneously and this desynchronized neural state may be related to active information processing in the cortex (Tirsch *et al.* 2004).

Competition among cortical neural cell assemblies which excite one another and are unable to agree on a common frequency of oscillations (Freeman, 1993) thus may be used as appropriate neurophysiological equivalent to dissociated and disintegrated competitive mental states in PDP model (Bob, 2003a). When the associated strength in these newly activated ranges of the neural network is low, it leads to strong competition among the neural assemblies (Freeman, 1993), and dissociated mental representations (Bob, 2003a). In parallel distributed processing model these dissociated states with low associated strength are represented by isolated "peaks" (Mc Clelland and Rumelhart, 1986; Li and Spiegel, 1992; Butler *et al.*, 1996).

The cases of 'antagonistic' competition or the so-called winnerless competition among neural assemblies according to several models may cause chaotic states in the brain dynamics (Korn and Faure, 2003). These chaotic states does not mean only an absence of order and unpredictability but the term chaos implies the idea of underlying structure and the potential for describing a complex system with the aid of relatively simple mathematical formulations with a basis in nonlinear mathematics. Generally, chaos in the neural system may leads to a low-dimensional aperiodic signal that may be used for describing behavior resulting from very many degrees of freedom in systems with very high complexity (Elbert *et al.* 1994; Tirsch *et al.* 2004). Freeman proposed that brain chaos probably arises from the competition of two or more parts of the brain represented by neuronal assemblies (Freeman, 1991; Elbert *et al.* 1994) and this process may represents neurophysiological substrate for competition of mental representations (Bob, 2003b). Chaos leads to an often instantaneous reduction of excitatory thresholds of many neural populations not excited in that particular combination before (Freeman, 1991; Elbert *et al.* 1994). Additionally, chaos may lead to a process known as bifurcation. Bifurcations characterize networks that are sensitive to very weak changes of initial conditions. As Freeman suggested the bifurcations may lead neural activity that can be observed as unexpected original ideas or in pathological cases as epileptic paroxysms (Elbert *et al.* 1994; Freeman, 1991). A characteristic feature of neural activity due to brain chaos is synchronous collective activity - a burst (Freeman 1991). The burst waves often have a frequency of about 40 Hz and high amplitudes and this process also may produce epileptic (or epileptiform) activity that represents a typical form of chaotic behavior (Freeman 1991; Elbert *et al.* 1994; Korn and Faure, 2003; Tirsch *et al.* 2004). Freeman (1991) suggested that chaos underlies the ability of the

brain to respond flexibly to the outside world and to generate novel activity patterns, including those that are experienced as fresh ideas. Chaos from this point of view expresses the underlying unpredictable order of attractors and enables the complex behavior of the brain (Freeman 1991, 2000, 2001; Skarda and Freeman 1987).

According to these connections dissociative states understood as the competitive states disintegrated mental representations with resulting chaotic states might explain the mechanism that produces hypersynchronized epileptic activity as a consequence of chaotic competition among neural assemblies and corresponding mental representations of conflicting mental states (Bob, 2003a). In these cases conflict of mental representations may include also interhemispheric competition and dissociation of cerebral hemispheres. On the "macro"-neurophysiological level dissociative processes may be understood in the connection of possible reversible blocking of the transmission from one hemisphere to the other across the corpus callosum and other commissural fibers. It may lead to the competition and chaotic states. This postulate may motivate similarities between some cases of psychic dissociation (or repression) and split brain patients (Galin, 1974) or epilepsy due to defective hemispheric interaction as Ahler *et al.* (1993). Dissociation from this point of view may be understood as a blocking of communication between verbal (conscious) left and the other side of the brain (Galin, 1974; Spitzer *et al.* 2004). On the other hand human creativity represents high level of psychic integration and corresponds to integration of left-hemispheric verbal analytical thought and holistic thought of the right hemisphere (Gallin, 1974; Bogen and Bogen, 1969). These connections suggest direct relationship between Epilepsy/Temporal Lobe Dysfunction model (Putnam 1997) and Cerebral Hemispheric Laterality Model which represents the theory that either an anatomical or a functional disconnection between the two hemispheres of the brain is the source of "double personality" (Putnam, 1989, 1997; Ellenberger, 1970; Quen, 1986). Competitive inter-hemispheric alter personality states connected to dissociated mental representations of corresponding neural assemblies thus suggest the explanation of repeated clinical observations that document laterality differences across alter personalities in MPD patients (Brende 1984; Henninger 1992; Le Page *et al.* 1992; Ahern 1993; Putnam 1997) such as mentioned cases when an intracarotid injection of amobarbital (Wada test) was used to anesthetize the hemisphere and this reproduced the alter personality changes associated with seizure activity in these two patients (Ahern *et al.* 1993). On the other hand there are controlled test of the laterality that did not find evidence of shifts in lateralization of galvanic skin response across repeatedly randomized testing of alter-personality states (Putnam 1997) that might be explained by dissociation without inter-hemispheric competition.

FORCED NORMALIZATION AND RELATIONSHIP BETWEEN EPILEPSY AND MENTAL ILLNESS

Because chaos according to this model of dissociation may explain the relationship between dissociation and epileptic activity the model is also in accordance with findings that epileptic activity may produce, similarly as dissociation, seizures and psychopathological symptoms. The relationship between seizures and psychopathology is historically linked to biological antagonism between epilepsy and psychosis that was for the first time investigated by a Hungarian physician László von Meduna (Meduna, 1934; Wolf and Trimble, 1985; Krishnamoorthy *et al.*, 2002). After graduation Meduna was interested in brain anatomy and in the year 1927 began his psychiatric research. At the time of his most important works he was leading physician in Royal Asylum in Budapest. There he dealt with experimental epilepsy and his findings confirmed that the relationship between epileptic paroxysms and psychotic manifestations is not only random (Wolf and Trimble, 1985; Krishnamoorthy *et al.* 2002). Steiner and Strauss (Wolf and Trimble, 1985) presented 6000 schizophrenic patients and found typical epileptic paroxysms in these cases very rare. In his work from 1935 Meduna introduced the study of 176 patients from which 95 were epileptics and had at the same time also psychotic symptoms (Meduna, 1935). Meduna confirmed that next to the antagonism may be also combination between epilepsy and psychosis. In his practical therapy he used convulsive drugs, for example camphor or penetrazol, for the treatment of schizophrenia. These drugs often led to convulsions and lowering of schizophrenic symptoms (Wolf and Trimble, 1985).

Discussions following Meduna's works initiated the development of convulsive therapy in psychiatry but the relationship between epilepsy and psychosis was neglected for a time. Its investigation was renewed in 1950s when Heinrich Landolt, director of Swiss asylum for epileptics in Zürich, by means of electroencephalographical methods reported his findings on forced normalization (Wolf and Trimble, 1985; Landolt, 1953). Landolt introduced the term forced normalization for the reaction of the organism, which represents the defense of the brain against epileptic discharges (Wolf and Trimble, 1985). According to his findings this reaction may begin spontaneously or as a consequence of antiepileptic medication. At first Landolt studied forced normalization in patients with temporal lobe epilepsy and later also in patients with focal cortical epileptic seizures. In 1954 he used succinimid medication in patients with generalized epilepsy of the type petit mal and in twilight states that represent qualitative changes of mental state dissociated from normal state of consciousness similar to schizophrenic symptoms.

Effects of forced normalization were also reported in cases of neurosurgical treatment focused on inactivation of the epileptic focus (Mace and Trimble, 1991; Blumer *et al.* 1998). It was suggested that the effects might explain kindling in mesolimbic dopaminergic system that has a relationship of reciprocity with regard to similar EEG activity in temporal neocortex in patients with temporal lobe epilepsy (Pakalnis *et al.* 1988).

Similar relationship of reciprocity was found also between epilepsy and depression (Jobe *et al.* 1999; Kanner and Balabanov, 2002; Chaplin *et al.* 1990; Trimble, 1996). Historical roots of this problem are also connected to the term forced normalization and contemporary is thought that people with epilepsy exhibit a higher incidence of depression compared to people in general population (Jobe *et al.* 1999; Kanner and Balabanov, 2002).

Several sources show that there is a relationship between seizures and affective disorders (Jobe *et al.* 1999; Kanner and Balabanov, 2002). First reason is that electroconvulsive therapy has a high degree of efficacy in the treatment of depression as well as in the treatment of manic states and similarly it is also for chemically induced seizures (Jobe *et al.*, 1999). Second reason is that the manifestations of forced normalization emerge in epileptic patients as an increasing occurrence of depressive symptoms when the frequency of seizures decreases. Next to a response to anticonvulsant therapy worsening of affective disorders or psychotic episodes due to the sharply reduced number of seizures postsurgically also may occur (Jobe *et al.* 1999; Kanner and Balabanov, 2002). Some investigators now believe that antidepressant therapy is crucial for significant number of patients after surgical treatment of epilepsy and that the symptoms of interictal dysphoric disorder tend to occur as chronic seizure activity is suppressed (Jobe *et al.* 1999; Kanner and Balabanov, 2002).

There is growing body of evidence that anticonvulsant medications have emerged as powerfull agents for the treatment of bipolar disorders as well as schizoaffective disorder or for the treatment of refractory depression (Jobe *et al.* 1999; Chaplin *et al.* 1990; Trimble, 1996).

On the other side there is an extensive body of evidence that clinically useful antidepressant drugs can both prevent and cause seizures (Jobe *et al.* 1999; Chaplin *et al.* 1990; Trimble, 1996). According to contemporary literature antidepressant drugs suppress seizures when blood and brain concentration are relatively low. In contrast to that seizures may occur as a response to antidepressants in overdoses or in response to excessive blood levels (Jobe *et al.* 1999). Also people with epilepsy exhibit anticonvulsant effects in response to antidepressants and the use of these drugs often represents a safe therapeutic approach in epileptic patients with interictal dysphoric disorder (Jobe *et al.* 1999).

On the other hand as mentioned above there is a great subgroup of depressive patients and schizophrenic patients without epilepsy which manifest temporal lobe lability and have complex partial seizure-like symptoms with good response to anticonvulsive medication (Roberts *et al.* 1992; Roberts, 1993; Hines *et al.* 1995; Bob *et al.* 2005, 2006).

ALTERNATIVE PSYCHOSIS AND DISSOCIATION

Further connection between forced normalization and dissociation represents the term alternative psychosis. From the clinical point of view, forced normalization is often connected with decreasing epileptic changes in EEG and improving the control of seizures. On the other hand for example psychotic symptoms appear. The clinical manifestations of forced normalization also include dysphoric states, hysteria and hypochondria, affective disorders, and miscellanea (twilight states). Forced normalization can be observed in both generalized and partial epilepsies as a rare complication. It is relatively frequently observed in adults with persistent absence seizures (Wolf, 1991; Kanner, 2000, 2001; Schmitz *et al.* 1999; Marsh and Rao, 2002). Because the term forced normalization is often considered in connection with EEG, Tellenbach in 1965 introduced in this connection the term alternative psychosis implicating that stopping seizures does not mean vanishing or inactivity of the pathological state (Wolf and Trimble, 1985; Krishnamoorthy *et al.* 2002). Several studies suggest that in the patients who display alternative psychosis subcortical epileptic discharges are continuously present (Wolf and Trimble, 1985) Heath (1962, 1975) pointed out that at the level of subcortical structures epilepsy and manifestations of psychotic symptoms in certain cases might have the same neurobiological substrate in the from of epileptic activity. Similar findings reported also other authors (Monroe, 1982; Walter, 1944; Goon *et al.* 1973; Alvarez, 2001), who reported that manifestations of schizophrenic pathology are correlated by subcortical spikes. Evoking epileptic seizure, as a consequence of electroconvulsive therapy or other convulsive methods which are connected with improvement of psychotic symptoms, points to effects of forced normalization also in cases of "pure" psychosis or depression and it suggest common neurobiological mechanisms between mental illness and epilepsy. Similarly, epileptic discharges located in subcortical structures were observed also in patients with dissociative seizures (Wieser, 1979) which strongly suggest that clear boundary cannot be determined between dissociative and epileptic seizures and that common pathophysiological processes for dissociation and epilepsy might be found.

CONCLUSION

At this time the pathogenesis of forced normalization is still unresolved. It has been postulated that amygdaloid and limbic kindling may play a role in the development of this phenomenon and there are also neurochemical changes that accompany forced normalization (Krishnamoorthy *et al.* 2002). A more comprehensive hypothesis is that the epilepsy is still active subcortically and provides energy for psychopathological symptoms (Wolf, 1991), and it has often been postulated that subcortical and subclinical electrophysiological activity, particularly in the limbic system, may be responsible for the development of forced normalization (Krishnamoorthy *et al.* 2002). It is likely that secondary epileptogenesis and other related phenomena may enable continuing of epileptiform activity in limbic areas with predominant psychopathological manifestations (Smith and Darlington, 1996; Stevens, 1999; Krishnamoorthy *et al.* 2002). These alterations and co-occurrence of symptoms are similar to certain conditions that occur during dissociative states when traumatic stress may emerge in a variety of symptoms such as psychopathological symptoms, seizures or other somatoform disturbances. Because dissociation is at least in several reported cases linked to epileptic activity common neurobiological mechanisms of traumatic stress and epileptic activity likely must be present. Modern formulation of dissociation in the terms of discrete behavioral states seems to be able to link cognitive processes related to dissociation and multi-stable perception of ambiguous stimuli with neural network model of dissociative states. Agreement of these conceptual approaches enables to reformulate the concept of dissociation in neuroscientific terms that may include also organic etiology of these pathological conditions. These findings and further data reported in this review strongly suggest that neurobiological concept of dissociation linked to nonlinear chaotic processes in the brain might provide possibilities for future research that may help more comprehensively understand the neurobiological consequences of the traumatic stress and also the relationship between dissociative states and epileptic activity.

Acknowledgement:

Author thanks for support by research grants MSM0021620849, MSM0021622404, IGA MZ NR 8824-4 and support of research project of Centre for Neuropsychiatric Research of Traumatic Stress 1M06039.

REFERENCES

1 Ahern GL, Herring AM, Tackenberg J and Seeger JF (1993). The association of multiple personality and temporolimbic epilepsy: Intracarotid amobarbital test observations. Arch Neurol. **50**: 1020–1025.
2 Aks DJ and Sprott JC (2003). Resolving perceptual ambiguity in the Necker cube: A dynamical systems approach. *Nonlinear Dynamics Psychol Life Sci.* **7**: 159–178.
3 Alvarez J (2001). Neural hypersynchronization, creativity and endogenous psychoses. *Med Hypotheses* **56**: 672–685.
4 American Psychiatric Association (1987). *DSM III-R, Diagnostic and Statistical Manual of Mental Disorders*, fourth ed. Washington DC: American Psychiatric Association.
5 American Psychiatric Association (1994). *DSM IV, Diagnostic and Statistical Manual of Mental Disorders*, fourth ed. Washington DC: American Psychiatric Association.
6 Baars B (1988). *A cognitive theory of consciousness*, Cambridge, MA: Cambridge University Press.
7 Baars BJ (1997). Some essential differences between consciousness and attention, perception, and working memory. *Conscious Cogn.* **6**: 363–371.
8 Baars BJ (1999). Attention versus consciousness in the visual brain: differences in conception, phenomenology, behavior, neuroanatomy, and physiology. *J Gen Psychol.* **126**: 224–233.
9 Baars BJ (2002). The conscious access hypothesis: origins and recent evidence. *Trends Cogn Sci.* **6**: 47–52
10 Barret, D. 1995. The dream character as prototype for the multiple personality alter. *Dissociation.* **8**: 66–68.
11 Benson DF (1986). Interictal behavior disorders in epilepsy. *Psychiatr Clin North Am*. **9**: 283–292.
12 Benson F, Miller BL and Signer SF (1986). Dual personality associated with epilepsy. *Arch Neurol* **43**: 471–474.
13 Berkhout J, Walter DO and Adey WR (1969). Alteration of the Human Electroencephalogram Induced by Stressful Verbal Activity. *Electroencephalogr Clin Neurophysiol*. **27**: 457–469.
14 Bernstein EM, Putnam FW (1986). Development, Reliability, and Validity of a Dissociation Scale. *J Nerv Ment Dis*. **174**: 727–735.
15 Blake R, Yu K, Lokey M, Norman H (1998). Binocular rivalry and motion perception. *J Cogn Neurosci.* **10**: 46–60.
16 Blanke O, Ortigue S, Landis T, Seeck M (2002). Neuropsychology: Stimulating illusory own-body perceptions. *Nature* **419**: 269–270.
17 Bleuler E (1924). Textbook of Psychiatry. A.A Brill (trans.). New York: Macmillan Publishing Co. Inc.
18 Blumer D, Wakhlu S, Davies K, Hermann B (1998). Psychiatric outcome of temporal lobectomy for epilepsy: incidence and treatment of psychiatric complications. *Epilepsia* **39**: 478 –86.
19 Bob P (2003a). Dissociation and Neuroscience: History and New Perspectives. *Int J Neurosci.* **113**: 903–914.
20 Bob P (2003b). Subliminal Processes, Dissociation and the "I". *J Anal Psychol* **48**: 307–316.
21 Bob P (2004). Dissociative processes, multiple personality and dream functions. *Am J Psychother.* **58**: 139–149.
22 Bob, P. (2007). Chaotic patterns of electrodermal activity during dissociated state released by hypnotic abreaction. *Int J Clin Exp Hypn.* **55** (4).
23 Bob P, Susta M, Pavlat J, Hynek K, Raboch J (2005). Depression, traumatic dissociation and epileptic-like phenomena. *Neuro Endocrinol Lett.* **26**: 321–325.
24 Bob P, Glaslova K, Susta M, Jasova D, Raboch J (2006). Traumatic dissociation, epileptic-like phenomena, and schizophrenia. *Neuro Endocrinol Lett.* **27**: 321–326.
25 Bob, P., Susta, M., Prochazkova-Vecerova, A., Kukleta, M., Pavlat, J., Jagla, F, & Raboch, J. (2006). Limbic irritability and chaotic neural response during conflicting Stroop task in the patients with unipolar depression. *Physiol Res.* **55** (Suppl. 1): 107–112.
26 Bogen JE, Bogen GM (1969). The other side of the brain: III. The corpus callosum and creativity. *Bull Los Angel Neurol Soc.* **34**: 191–220.
27 Boleloucky Z (1986). Multiple, dissociated personality – new interest in an old problem. *Cesk Psychiatr*. **82**: 318–327.
28 Bottero A (2001). A history of dissociative schizophrenia. *Evol Psychiatr.* **66**: 43–60.
29 Bower GH (1981). Mood and Memory. *Am Psychol*. **36**: 129–148.
30 Bowers MK, Brecher S (1955). The emergence of multiple personalities in the course of hypnotic investigation. *Int J Clin Exp Hypn*. **3**: 188–199.
31 Brazdil M, Rektor I, Daniel P, Dufek M, Jurak P (2001). Intracerebral event-related potentials to subthreshold target stimuli. *Clin Neurophysiol.* **112**: 650–61.
32 Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, Delaney RC, Charney DS (1995). MRI-based measurement of hippocampal volume in combat-related posttraumatic stress disorder. *Am J Psychiatry* **152**: 973–981.

33 Bremner JD (2006). The relationship between cognitive and brain changes in posttraumatic stress disorder. *Ann N Y Acad Sci.* **1071**: 80–6.
34 Brende JO (1984). The psychophysiologic manifestations of dissociation: Electrodermal responses in a multiple personality patient. *Psychiatr Clin North Am*. **7**: 41–50.
35 Breuer J and Freud S (1895). *Studies in hysteria.* New York: Basic Books.
36 Brewin CR and Andrews B (1998). Recovered memories of trauma: Phenomenology and cognitive mechanisms. *Clin Psychol Rev*. **18**: 949–970.
37 Briere J and Conte J (1989). Amnesia and adults molested as children: Testing theories of repression. Paper presented at the 97th Annual Convention of the American Psychological Association, New Orleans.
38 Brown RJ and Trimble MR (2000). Dissociative psychopathology, non-epileptic seizures, and neurology. *J Neurol Neurosurg Psychiatr* **69**: 285–9.
39 Butler LD, Duran REF, Jasiukaitis P, Koopman CH, Spiegel D (1996). Hypnotizability and Traumatic Experience: A Diathesis-Stress Model of Dissociative Symptomatology. *Am J Psychiatry* (Festschrift Supplement) **153**: 42–62.
40 Coveney P and Highfield R (1996). *Frontiers of Complexity*. London: Faber and Faber.
41 Chaplin J, Yepez R and Shorvon S (1990). A quantitative approach to measuring the social effects of epilepsy. *Neuroepidemiology* **9**: 151–158.
42 Chu J and Dill D (1990). Dissociative Symptoms in Relation to Chidhood Sexual and Physical Abuse. *Am J Psychiatry* **147**: 887–892.
43 Colman AM (2003). *A Dictionary of Psychology*. New York: Oxford University Press.
44 Coons PM, Bowman ES and Pellow TA (1989). Post-traumatic aspects of the treatment of victims of sexual abuse and incest. *Psychiatr Clin North Am*. **12**: 325–327.
45 Coons PM, Milstein V and Marley C (1982). EEG studies of two multiple personality and a control. *Arch Gen Psychiatry* **39**: 823–825.
46 Crick F and Koch C (1995). Are we aware of neural activity in primary visual cortex? *Nature* **375**: 121–123.
47 Desimone R and Duncan J (1995). Neural mechanisms of selective visual attention. *Annu Rev Neurosci.* **18**: 193–222.
48 Dreifuss F (1981). Proposal for revised clinical and electroencephalographic classification of epileptic seizures. *Epilepsia* **22**: 489–501.
49 Elbert T, Ray WJ, Kowalik ZJ, Skinner JE, Graf KE, Birbaumer N (1994). Chaos and Physiology: Deterministic Chaos in Excitable Cell Assemblies. *Physiol Rev.* **74**: 1–47.
50 Ellason JW and Ross CA (1995). Positive and negative symptoms in dissociative identity disorder and schizophrenia: a comparative analysis. *J Nerv Ment Dis.* **183**: 236–41.
51 Ellenberger HF (1970). The Discovery of the Unconscious: The History and Evolution of Dynamic Psychiatry. New York: Basic.
52 Esch T and Stefano GB (2005a). The neurobiology of love. *Neuro Endocrinol Lett.* **26**: 175–92.
53 Esch T and Stefano GB (2005b). Love promotes health. *Neuro Endocrinol Lett*. **26**: 264–7.
54 Faber J and Vladyka V (1987). Epileptogenesis and Psychosogenesis, antithesis or synthesis? *Acta Univ Carol Med*. **34**: 245–312.
55 Faber J, Vladyka V, Dufkova D, Faltus F, Jirak R, Pavlovsky J, Smidova E, Zvolsky P, Zukov I, Klar I, Posmurova M, Srutova L (1996). "Epileptosis"– A syndrome or useless speculation. *Sb Lek*. **97**: 71–95.
56 Fisher, R.S., Webber, W.R., Lesser, R.P., Arroyo, S., Uematsu, S. 1992. High-frequency EEG activity at the start of seizures. J. Clin. Neurophysiol. 9, 441–448.
57 Fischer, C. (1954) Dreams and perception: The role of preconscious and primary modes of perception and dream formation. *Journal of the American Psychoanalytic Association* 2: 389–445.
58 Freeman WJ (1991). The Physiology of Perception. *Sci Am.* **264**: 34–41.
59 Frankel FH (1996). Dissociation: The Clinical Realities. *Am J Psychiatry* **153** (Suppl.) 64–70.
60 Freeman WJ (2000). Mesoscopics neurodynamics: From neuron to brain. *J Physiol*. (Paris) **94**: 303–322.
61 Freeman WJ (2001). Biocomplexity: Adaptive behavior in complex stochastic dynamical systems. *BioSystems* **59**: 109–123.
62 Galin D (1974). Implications for psychiatry of left and right cerebral specialization. *Arch Gen Psychiatry* **31**: 572–583.
63 Gavrilovic L and Dronjak S (2005). Activation of rat pituitary-adrenocortical and sympatho-adrenomedullary system in response to different stressors. *Neuro Endocrinol Lett.* **26**: 515–20.
64 Gazzaniga MS and Sperry RW (1967). Language after section of the cerebral commissures. *Brain* **90**: 131–148.
65 Goebel R, Khorram-Sefat D, Muckli L, Hacker H, Singer W (1998). The constructive nature of vision: direct evidence from functional magnetic resonance imaging studies of apparent motion and motion imagery. *Eur J Neurosci.* **10**: 1563–1573.
66 Goddard GV, McIntyre GC and Leech CK (1969). A permanent change in brain function resulting from daily electrical stimulation. *Exp Neurol*. **25**: 295–330.
67 Good MI (1993). The Concept of an Organic Dissociative Syndrome: What is the Evidence? *Harv Rev Psychiatry* **1**: 145–57.
68 Goon Y, Robinson S and Lavy S. (1973). Electroencephalographic changes in schizophrenic patients. *Isr Ann Psychiatr Relat Discip*. **11**: 99–107.
69 Gottschalk A M, Bauer MS and Whybrow PC (1995). Evidence of chaotic mood variation in bipolar disorder. *Arch Gen Psychiatry* **52**: 947–959.
70 Groethuysen VC, Robinson DB, Haylett CH, Estes HR, Johnson AM (1957). Depth electrographic recording of a seizure during a structured interview. *Psychosom Med.* **19**: 353–362.
71 Haule JR (1984). From Somnambulism to the Archetypes: The French Roots of Jung's Split with Freud. *Psychoanal Rev.* **71**: 636–689.
72 Havens LL (1966). Pierre Janet. *J Nerv Ment Dis.* **143**: 383–398.
73 Heath RG (1962). Common Characteristic of epilepsy and schizophrenia: Clinical Observation and Depth Electrode Studies. *Am J Psychiatry* **118**: 1013–1026.
74 Heath RG (1975). Brain Function and Behavior. *J Nerv Ment Dis.* **160**: 159–175.
75 Henninger P (1992). Conditional handedness: Handedness changes in multiple personality disorder subjects reflect shifts in hemispheric dominance. *Conscious Cogn.* **1**: 265–287.
76 Hersch J, Yiu-Chung C and Smeltzer D (2002). Identity shifts in temporal lobe epilepsy. *Gen Hosp Psychiatry* **24**: 185–187.
77 Hilgard ER (1974). Toward a Neodissociation Theory: Multiple Cognitive Controls in Human Functioning. *Perspect Biol Med.* **17**: 301–316.
78 Hilgard ER (1986). *Divided Consciousness. Multiple Control in Human Thought and Action.* New York: Wiley.
79 Hilgard ER (1988). Commentary. Professional skepticism about multiple personality. *J Nerv Ment Dis.* **176**: 532.
80 Hines M, Swan C, Roberts RJ, Varney NR (1995). Characteristics and mechanisms of epilepsy spectrum disorder: An explanatory model. *Appl Neuropsychol*. **2**: 1–6.
81 Huston P, Skehow J and Erickson M (1934). A study of hypnotically induced complexes by means of the Louria technique. *J Gen Psychol.* **11**: 65–97.
82 Ito Y, Teicher M, Gold C, Harper D, Magnus E, Gelbard H (1993). Increased prevalence of electrophysiological abnormalities in children with psychological, physical and sexual abuse. *J Neuropsychiatry Clin Neurosci*. **5**: 401–408.
83 Jampala V, Atre-Vaidya N and Taylor MA (1992). A profile of psychomotor symptoms (POPS) in psychiatric patients. *Neuropsychiatry Neuropsychol Behav Neurol.* **5**: 15–19.
84 Janet P (1890). *L'Automatisme Psychologique.* Paris: Felix Alcan.
85 Jobe PC, Dailey JW and Wernicke JF (1999). A noradrenergic and serotoninergic hypothesis of the linkage between epilepsy and affective disorders. *Crit Rev Neurobiol*. **13**: 317–356.
86 Jung CG (1968). *Analytical Psychology: Its Theory and Practice*. London: Routledge and Kegan Paul.
87 Jung CG (1972a). On the Nature of the Psyche. The structure and dynamics of the psyche. In: *Collected Works of C.G. Jung 8*. Princeton: Princeton University Press.
88 Jung CG (1972b). Psychological factors determining human behaviour. The structure and dynamics of the psyche In: *Collected Works of C.G. Jung 8*. Princeton: Princeton University Press.

89 Jung CG (1972c). A Review of the Complex Theory. The structure and dynamics of the psyche. In: *Collected Works of C.G. Jung 8*. Princeton: Princeton University Press

90 Jung CG (1973). *Collected Works of C. G. Jung 2*. Princeton: Princeton University Press.

91 Kanner AM (2000). Psychosis of Epilepsy: A Neurologist's Perspective. *Epilepsy Behav.* **1**: 219–227.

92 Kanner AM (2001). The Behavioral Aspects of Epilepsy: An Overview of Controversial Issues, *Epilepsy Behav*. **2**: 8–12.

93 Kanner AM and Balabanov A (2002). Depression and Epilepsy. How closely related are they? *Neurology* **58**: 827–839.

94 Kanwisher N (2001). Neural events and perceptual awareness. *Cognition* **79**: 89–113.

95 Putnam FW (1995). Investigating multiple personality disorder. *Br J Psychiatry* **166**: 122–123. Gazzaniga MS and Sperry RW (1967). Language after section of the cerebral commissures. *Brain* **90**: 131–148.

96 Kaplan HI and Sadock BJ (1991). *Comprehensive Glossary of Psychiatry and Psychology*. Baltimore: Williams and Wilkins.

97 Kihlstrom JF (1987). The Cognitive Unconscious. *Science* **237**: 1445–1452.

98 Kihlstrom JF (2004). Availability, accessibility, and subliminal perception. *Consciousness and Cognition* **13**: 92–100.

99 King JA, Rosal MC, Ma Y, Reed GW (2005). Association of stress, hostility and plasma testosterone levels. Neuro Edocrinol Lett **26**: 355–60.

100 Korn H and Faure P (2003). Is there chaos in the brain? II. Experimental evidence and related models. *C R Biol* **326**: 787–840.

101 Krishnamoorthy ES, Trimble MR, Sander JW, Kanner AM (2002). Forced normalization at the interface between epilepsy and psychiatry. *Epilepsy Behav.* **3**: 303–308.

102 Kuyk J, Spinhoven P, Van Emde Boas W, Van Dyck R (1999). Dissociation in Temporal Lobe Epilepsy and Pseudo-Epileptic Seizure Patients. *J Nerv Ment Dis.* **187**: 713–720.

103 Landolt H (1953). Some clinical electroencephalographical correlations in epileptic psychosis (twilight states). *Electroencephalogr Clin Neurophysiol.* **5**: 121.

104 Le Page KE, Schafer DV and Miller A (1992). Alternating unilateral lachrymation. Am J Clin Hypn **34**: 255–260.

105 Li D and Spiegel D (1992). A Neural Network Model of Dissociative Disorders. *Psychiatr Ann.* **22**: 144–47.

106 Libet B, Wright EW Jr, Einstein B, Pearl DK (1979). Subjective Refferal of the Timing for a Conscious Sensory Experience. *Brain* **102**: 193–224.

107 Luck SJ and Girelli M (1998). Electrophysiological approaches to the study of selective attention in the human brain. In: Parasuraman R, Editor. Cambridge, MA: *The attentive brain*, MIT Press, p. 71–94.

108 Lutzenberger W, Birbaumer N, Flor H, Rockstroh B, Elbert T (1992). Dimensional Analysis of the Human EEG and Inteligence. *Neurosci Lett*. **143**: 10–14.

109 Lynn S J, Maré C, Kvaal S, Segal D, Sivec H (1994). The hidden observer, hypnotic dreams, and age regression: Clinical implications. *Am J Clin Hypn*. **37**: 130–142.

110 Mace CJ and Trimble MR (1991). Psychosis following temporal lobe epilepsy: a report of six cases. *J Neurol Neurosurg Psychiatry* **54**: 639–644.

111 Marcel AJ (1983). Conscious and Unconscious Perception: An Approach to the Relations Between phenomenal experience and perceptual processes. *Cognit Psychol.* **15**: 238–300.

112 Marsh L, Rao V (2002). Psychiatric complications in patients with epilepsy: a review. *Epilepsy Res* **49**: 11–33.

113 Mc Clelland JL, Rumelhart DE and the PDP research group (1986). *Parallel Distributed Processing I, II*. Cambridge: MIT Press,.

114 Meares R (1999). The Contribution of Hughlings Jackson to an Understanding of Dissociation. *Am J Psychiat*. **156**: 1850–1855.

115 Meduna L (1935). Versuche über die biologische Beeinflussung des Ablaufes der Schizophrenie, in: Campher- und Cardiazolkräpfe. *Zeitschrift für Neurologie und Psychiatrie* **15**: 235–262.

116 Meduna L (1934). Über experimentelle Campherepilepsie. *Archiv für Psychiatrie*, **102**: 333–339.

117 Merskey, H. 1992. The manufacture of personalities. The production of multiple personality disorders. Br. J. Psychiatry 160, 327–40.

118 Mesulam MM (1981). Dissociative states with abnormal temporal lobe EEG. *Arch Neurol.* **38**: 176–181.

119 Monroe RR (1982). Limbic Ictus and Atypical Psychoses. *J Nerv Ment Dis*. **170**: 711–716.

120 Monroe RR (1978). The Episodic Psychoses of Vincent van Gogh. *J Nerv Ment Dis*. **166**: 480–488.

121 Mölle M, Marshall L, Lutzenberger W, Pietrowsky R, Fehm HL, Born J (1996). Enhanced Dynamic Complexity in the Human EEG during Creative Thinking. *Neurosci Lett.* **208**: 61–64.

122 Nakayama Y, Takahashi T, Radford MH (2005). Cortisol levels and prospective and retrospective memory in humans. *Neuro Endocrinol Lett.* **26**: 599–602.

123 Nemiah JC (1981). Dissociative Disorders. In: Freeman AM, Kaplan HI editors. *Comprehensive Textbook of Psychiatry*. Baltimore: Williams and Wilkins.

124 Pakalnis A, Drake ME, Kuruvilla J, Blake JK (1988). Forced normalization. *Arch Neurol*. **45**: 139.

125 Payne JD, Jackson ED, Ryan L, Hoscheidt S, Jacobs JW, Nadel L (2006). The impact of stress on neutral and emotional aspects of episodic memory. *Memory* **14**: 1–16.

126 Perrine KR (1991). Psychopathology in epilepsy. *Semin Neurol*. **11**: 175–181.

127 Poetzl O (1960). The Relationship between Experimentally Induced Dream Images and Indirect Vision. *Psychol Issues Monograph* **2**: 46–106.

128 Post RM, Weis SR and Smith MA (1995). Sensitization and kindling. In: Friedman MJ, Charney DS, Deutch AY, editors. *Neurobiological and clinical consequences of stress: From normal adaptation to posttraumatic stress disorder*. Philadelphia: Lipincott-Raven.

129 Putnam, F.W. 1989. Diagnosis and Treatment Multiple Personality Disorder, The Guilford Press, New York, London.

130 Putnam FW (1995). Investigating multiple personality disorder. *Br J Psychiatry* **166**: 122–123.

131 Putnam F (1997). *Dissociation in Children and adolescents. A developmental Perspective.* London, New York: The Guilford Press

132 Quen JM (1986). *Split Minds/Split Brains*. New York: New York University Press.

133 Read J, Perry BD, Moskowitz A, Connolly J (2001). The contribution of early traumatic events to schizophrenia in some patients: a traumagenic neurodevelopmental model. *Psychiatry* **64**: 319–45.

134 Reanault B, Signoret JL, Debruille B, Breton F, Bolgert F (1989). Brain Potentials Reveal Covert Facial Recognition in Prosopagnosia. *Neuropsychologia* **27**: 905–912.

135 Redington DJ and Reidbord SP (1992). Chaotic dynamics in autonomic nervous system activity of a patient during a psychotherapy session. *Biol Psychiatry* **31**: 993–1007.

136 Rees G, Russell C, Frith CD, Driver J (1999). Inattentional blindness versus inattentional amnesia for fixated but ignored words. *Science* **286**: 2504–2507.

137 Rickeport MM (1992). The interface between multiple personality, spirit mediumship and hypnosis. *Am J Clin Hypn*. **34**: 168–77.

138 Roberts RJ, Varney NR and Paulsen JS (1990). Dichotic listening and complex partial seizures. *J Clin Exp Neuropsychol.* **12**: 448–458.

139 Roberts RJ, Gorman LL, Lee GP, Hines ME, Richardson ED, Riggle TA, Varney NR (1992). The phenomenology of multiple partial seizure like symptoms without stereotyped spells: An epilepsy spectrum disorder? *Epilepsy Res.* **13**: 167–177.

140 Roberts RJ (1993). Commentary; Positive associations among dichotic listening errors, complex partial epileptic-like signs, and paranormal beliefs. *J Nerv Ment Dis.* **131**: 668–671.

141 Rosenbaum M (1980). The role of the term schizophrenia in the decline of diagnoses of multiple personality. *Arch Gen Psychiat*. **37**: 1383–1385.

142 Rubin N (2003). Binocular rivalry and perceptual multi-stability. *Trends Neurosci.* **26**: 289–291

143 Saver JL and Rabin J (1997). The neural substrates of religious experience. *J Neuropsychiatry Clin Neurosci.* **9**: 498–510.

144 Scharfetter C (1998). Dissociation and schizophrenia. Schizophrenias- a dissociative nosopoietic construct? *Fortschr Neurol Psychiatr.* **66**: 520–3.

145 Schenk L and Bear D (1981). Multiple personality and related dissociative phenomena in patients with temporal lobe epilepsy. *Am J Psychiatry* **138**: 1311–1316.

146 Schmitz EB, Robertson MM and Trimble MR (1999). Depression and schizophrenia in epilepsy: social and biological risk factors. *Epilepsy Res.* **35**: 59–68.
147 Shevrin H (2001). Event-related markers of unconscious processes. *Int J Psychophysiol.* **42**: 209–218.
148 Silberman E, Post R, Nurenberger J, Theodore W, Boulenger J (1985). Transient sensory, cognitive, and affective phenomena in affective illness: A comparison with complex partial epilepsy. *Br J Psychiatry* **146**: 81–89.
149 Skarda, CA and Freeman WJ (1987). How Brains Make Chaos in Order To Make Sense of the World. *Behav Brain Sci*. **10**: 161–95.
150 Smith PF and Darlington CL (1996). The development of psychosis in epilepsy: a reexamination of the kindling hypothesis. *Behav Brain Res*. **75**: 59–66.
151 Sperry RW (1968). Hemisphere deconnection and unity in conscious awareness. *Am Psychol.* **23**: 723–733.
152 Spiegel D (1991). Neurophysiological correlates of hypnosis and dissociation. *J Neuropsychiatry Clin Neurosci*. **3**: 440–5.
153 Spiegel D and Cardena E (1991). Disintegrated Experience: The Dissociative Disorders Revisited. *J Abnorm Psychol.* **100**: 366–376.
154 Spitzer C, Haug HJ and Freyberger HJ (1997). Dissociative symptoms in schizophrenic patients with positive and negative symptoms. *Psychopathology* **30**: 67–75.
155 Spitzer C, Willert C, Grabe H, Rizos T, Moller B, Freyberger HJ (2004). Dissociation, hemispheric asymmetry, and dysfunction of hemispheric interaction: A transcranial magnetic stimulation approach. *J Neuropsychiatry Clin Neurosci.* **16**: 163–169.
156 Startup M (1999). Schizotypy, dissociative experiences and childhood abuse: relationships among self-report measures. *Br J Clin Psychol*. **38**: 333–44.
157 Stefano GB and Esch T (2005). Love and stress. *Neuro Endocrinol Lett*. **26**:173–4.
158 Stevens JR (1959). Emotional activation of the electroencephalogram in patients with convulsive disorders. *J Nerv Ment Dis.* **128**: 339–351.
159 Stevens JR (1999). Epilepsy, schizophrenia and the extended amygdala. *Ann NY Acad Sci.*: **156**: 548–561.
160 Stross L and Shevrin H (1962). Differences in thought organization between hypnosis and the waking state: An experimental approach. *Bull Menninger Clin.* **26**: 237–247.
161 Stross L and Shevrin H (1968). Thought organization in hypnosis and the waking state. *J Nerv Ment Dis.* **147**: 272–288.
162 Stross L and Shevrin H (1969). Hypnosis as a method for investigating unconscious thought processes. *J Am Psychoanal Assoc.* **17**: 100–135.
163 Takahashi T, Ikeda K, Ishikawa M, Kitamura N, Tsukasaki T, Nakama D, Kameda T. (2005). Anxiety, reactivity, and social stress-induced cortisol elevation in humans. *Neuro Endocrinol Lett*. **26**:351–4.
164 Teicher M, Glod C, Surrey J, Swett C (1993). Early childhood abuse and limbic system ratings in adult psychiatric outpatients. *J Neuropsychiatry Clin Neurosci*. **5**: 301–306.
165 Teicher M, Andersen SL, Polcari A, Anderson CM, Navalta CP, Dennis M, Kim DM (2003). The neurobiological consequences of early stress and childhood maltreatment. *Neurosci Biobehav Rev*. **27**: 3–44.
166 Tirsch WS, Stude Ph, Scherb H, Keidel M (2004). Temporal order of nonlinear dynamics in human brain. Brain Res Rev. **45**: 79–95.
167 Trimble MR (1996). Anticonvulsant-induced psychiatric disorders. The role of forced normalization. *Drug Saf.* **15**: 159.
168 Umegaki H, Yamamoto A, Suzuki Y, Iguchi A (2006). Stimulation of the hippocampal glutamate receptor systems induces stress-like responses. *Neuro Endocrinol Lett*. **27**: 339–43.
169 van der Hart O and Friedman B (1989). A Reader's Guide to Pierre Janet on Dissociation: A Neglected Intelectual Heritage. *Dissociation* **2**: 3–16.
170 Vermetten E and Bremner JD (2004). Functional brain imaging and the induction of traumatic recall: a cross-correlational review between neuroimaging and hypnosis. *International J Clin Exp Hypn.* **52**: 218–312.
171 Walter WG (1944). Electroencephalography. *J Ment Sci.* **90**: 64.
172 Watkins JG and Watkins HH (1979–80). Ego states and hidden observers. *J Altered States of Consciousness* **5**: 3–18.
173 Watkins HH (1993). Ego-state therapy: an overview. *Am J Clin Hypn*. **35**: 232–40.
174 Wieser HG (1979). The stereoencephalographic correlate of psychical seizures. *EEG EMG Z Elektroenzephalogr Elektromyogr Verwandte Geb.* **10**: 197–206.
175 Wolf P (1991). Acute behavioral symptomatology at disappearence of epileptiform EEG abnormality. Paradoxical or 'forced' normalization. *Adv Neurol*. **55**: 127–142.
176 Wolf P and Trimble MR (1985). Biological Antagonism and Epileptic Psychosis. *Br J Psychiatry* **146**: 72–276.
177 Wolff PH (1987). *The development of behavioral and emotional states in infancy*. Chicago: University Chicago Press.
178 World Health Organization (1993). *The ICD-10. Classification of Mental and Behavioural Disorders. Diagnostic Criteria for Research*. Geneva: World Health Organization.
179 Yates JL and Nasby W (1993). Dissociation, Affect, and Network Models of Memory: An Integrative Proposal. *J Trauma Stress* **6**: 305–326.
180 von Helmholtz H (1962). *Helmholtz's treatise on physiological optics* (J. P. C. Southall, Trans.). New York: Dover. (Original work published 1866).
181 Wong PS, Bernat E, Snodgrass M, Shevrin H (2004). Event-related brain correlates of associative learning without awareness. *Int J Psychophysiol.* **53**: 217–31.
182 Wortman CB, Loftus EF and Marshall ME (1992). Sensation and Perception. In: Wortman CB, Loftus EF, Marshall ME, editors. *Psychology*. New York: Mc Graw-Hill.