IDEAS & PERSPECTIVES

DISSOCIATION, EPILEPTIFORM DISCHARGES AND CHAOS IN THE BRAIN: TOWARD A NEUROSCIENTIFIC THEORY OF DISSOCIATION

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Abstract

Dissociated states represent pathological conditions when psychological trauma may emerge in a variety of forms such as psychic dissociative symptoms or, on the contrary, as paroxysms or other somatoform symptoms. There is evidence that epileptic activity plays an important role in the generation of dissociative states and it is able to generate various psychopathological processes as well as a wide spectrum of somatic symptoms or seizures. For the explanation of these connections between dissociative states and epileptic discharges the author proposes a neuroscientific model of dissociation based on the theory of competitive neural assemblies which can lead to chaotic self-organization in brain neural networks. This model is suggested as an integrative view interconnecting the various psychopathological and somatoform manifestations of dissociative states and suggests further possibilities for future research regarding common pathogenic mechanisms among epilepsy and mental disorders.

Key words: Dissociation; Stress; Chaos; Complexity; Epileptiform discharges

1. INTRODUCTION

Scientific history of the dissociation began in the French psychiatry of the 19th century mainly in the Pierre Janet’s work. 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 & 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 disaggregation 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 Automatisms” (Havens 1966; Janet 1890; van der Hart & 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 he was greatly influenced by his collaboration with J.M. Charcot in the Parisian hospital Salpetrière.

During complete psychological automatism (van der Hart & 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. An example of this is during hypnotic anesthesia, when something, such as the touching of an object, is not registered by the consciousness but may be registered by the secondary consciousness, called by Hilgard “the hidden observer” (Hilgard, 1986). In the case of complete or partial automatism, systems of unconscious fixed ideas play an important role and may 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 (van der Hart & Friedman, 1989). In this context, 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 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.

![Figure 1](image_url)

*Figure 1.* Dissociation produces psychopathological as well as somatoform symptoms.

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)
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. 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). Modern findings show that dissociative processes must be seen as clinical reality (Frankel, 1996).

In the following section further historical and recent findings about dissociative processes are reviewed. Then a neuroscientific model of dissociation and its empirical evidence is discussed and it is shown how this model, jointly constrained by psychopathological and neuroscientific data, can help to explain phenomena of somatoform dissociative symptoms, forced normalization and alternative psychosis.

2. DISSOCIATION AND TRAUMA

Dissociation may be defined as partial or total disconnection between memories of the past, awareness of identity and of immediate sensations, and control of bodily movements often resulting from traumatic experiences, intolerable problems, or disturbed relationships (Colman, 2003). Dissociation represents a special form of consciousness in which events that would ordinarily be connected are divided from one another (Li & Spiegel, 1992) or it is also often less generally understood as inability to integrate some psychic contents into the consciousness (Bernstein & 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 & Trimble 2000; Kuyk et al., 1999).

The groups of syndromes which are immediately bound to dissociative processes were described by John Nemiah (1981). Main criteria are: 1. alteration of identity as a consequence of dissociative reaction and 2. disturbances 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 development of symptoms often after many years. Dissociative symptoms also often occur due to traumatic events after serious accidents or natural disasters. Symptoms of disintegration often develop in the connection to posttraumatic stress disorder. 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 & Cardena, 1991). For example, Chu and Dill (1990) investigated dissociation by means of DES in 98 females and found significantly higher dissociation in patients who 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.

Generally these findings implicate that any exposed or reported trauma may be important for the development of dissociative symptoms and may be closely related to many symptoms such as depression, 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).

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

Pierre Janet as mentioned above, in his work about psychological automatisms (Janet, 1890), 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 & 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 contents in his experiments in Burghölzl and described them in his studies of word associations (Jung, 1973). 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). The existence of these ordering factors was 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. In the case of pathological influence the complex leads to a lowered mental level (Janet, 1890; van der Hart & 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 & Nasby, 1993).
Figure 2. Traumatic influence leads to dissociation which varies from mild forms to serious such as multiple personality disorder (MPD).

In further research dissociation became unnecessary 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 & Nasby, 1993).

In this context, affect plays a crucial role in modulation of memory processes, which, in extreme cases, lead to dissociative deficits. 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 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 deficits in associative connections as, for example, the so-called “word salad” (Yates & Nasby, 1993).

In other words, dissociated fragments, i.e., complexes which represent organized collections of ideas, emotions, impulses, and memories that share a common emotional tone and that have been excluded either partly or entirely from consciousness but continue to influence a person’s thoughts, emotions, and behavior (Colman, 2003), are pathologically disintegrated due to an 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. Inhibited negative emotion is dissociated from the consciousness and it implicates the problem of unconscious emotions which in serious states of dissociation lead to phenomenological fragmentation of the Self to multiple Selves (Lambie & Marcel, 2002, 2004; Dalgleish & Power, 2004).

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 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.

The creation of personalities and dissociated structure has been defined also 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). The creation of personalities in 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. Bleuler proposed that schizophrenia 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. 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 corresponds to findings that a substantial number of patients with dissociative identity disorder (DID) have previous diagnoses of schizophrenia. 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 schizophrenic patients. It is important to note that schizophrenic patients report more negative symptoms. A primary emphasis on positive symptoms may result in false-positive diagnoses of schizophrenia and false-negative diagnoses of dissociative identity disorder (Elason & Ross, 1995). On the other hand there are findings that show markedly high level of dissociation in schizophrenic patients (Bernstein & Putnam 1986; Read et al., 2001; Spitzer, Haug, & Freyberger, 1997; Startup 1999). The above data suggest close relationship between schizophrenia and dissociative disorders.

Close relationship between dissociation and traumatic experiences has been also found in modern studies of depression. According to Beck’s cognitive theory of depression (Beck et al., 1979) many aspects of depressive cognition reflect the fact that the individuals predominantly cannot recall negative past episodes. It is linked to activation of generalized schemas that consist of negative information about the self and activate specific less accessible autobiographical memories. The study reported by Spenceley and Jerrom (1997), suggests that defensive reaction which leads to an effort to eliminate these negative memories increases intrusive thoughts and at this time it is known that intrusive memories have an important role in depression (Brewin & Andrews, 1998). It implicates that the effort to avoid bad experiences paradoxically may increase their accessibility to the consciousness (Wegner, 1994). According to information-processing theories of depression, the greater accessibility of negative memories should lead to more severe and prolonged depression and predict it (Brewin & Andrews, 1998). Kuyken and Brewin (1995) reported close relationship between intrusive autobiographical memories of childhood abuse and other aspects of
depressive cognition. In addition, depressive patients (although less than PTSD patients) experience very vivid and distressing memories (Brewin & Andrews, 1998). The majority of the memories are accompanied by feelings and physical sensations reliving a traumatic event and represent dissociated fragments (or complexes) separated from normal mental phenomena.

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 & Brecher, 1955; Lynn et al., 1994; Merskey, 1992; Rickeport, 1992; Watkins, 1993; Watkins & 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 in this study 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. Barret (1995) also 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.

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. According to this Putnam’s concept 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. State defining variables may be continuous or dichotomous and define behavioral state space. It means that individual behavioral states exist within larger multidimensional framework or space defined by a chosen set of variables and 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. The state space may be vast but the individual regularly occupies those regions in which one has created stable discrete states. 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. 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, which corresponds to conventional definition of dissociation that emphasizes the separation or segregation of specific ideas or affects from normal mental phenomena (Putnam, 1997; Kaplan & Sadock, 1991). When two types of the states are significantly different, then the states are separated by a wide gap in the state space that determines pathological dissociative states. Putnam also proposed that observable differences between two discrete states are not a simple function of moving up or down but there are probably nonlinear dynamic features connected to chaos (Putnam, 1997). Similaryl for example Wolff (1987) highlighted differential responsiveness as an example of the nonlinearity of input output relation in different states of consciousness and conceptualized the relevance of nonlinear dynamic
systems theory to discrete behavioral states where switches between behavioral states constitute nonlinear chaotic transitions. Further recent studies give preliminary evidence that rapid shifts in mood and behavior correspond to nonlinear chaotic processes (Putnam, 1997; Gottschalk, Bauer, & Whybrow, 1995). Chaotic features between dissociated states mean that there is unpredictable and seemingly random behavior occurring in a system that should be governed by deterministic laws. This may occur when the psychological process involves a large number of complex interlinked and simultaneously active states which can lead to selforganization as the spontaneous order arising in a system when certain parameters of the system reach critical values (Isaacs, Daintith, & Martin, 2003).

3. NEURAL NETWORKS AND DISSOCIATION

Certain data suggest that neural networks may provide an attractive framework for modelling dissociative mental processes and dissociative mechanism. They require the parallel operation of two or more information processors which correspond to dissociated states (Li & Spiegel, 1992). These processor systems are disintegrated and it represents the problem rather than the competition of these subsystems (Li & Spiegel, 1992). Neural networks in which memory is parallel distributed in the space of the network (Jordan, 1986) represent important classes of models for the study of dissociative processes. This network is able to explain a wider class of dissociative processes, such as the course of posthypnotic amnesia. Parallel distributed processing (PDP) in neural networks is a model for the microstructure of cognition (Mc Clelland & Rumelhart, 1986), where the activities of many neurons are described as configurations or neural patterns and their psychological correlates are called mental representations. Using this theoretical concept the neural network state is described by the superposition of the neuronal patterns. The neural patterns (configurations) in the superposition are in “prespace”, which corresponds to psychic space and active neural patterns are selected from this superposition (Butler et al., 1996; Li & Spiegel, 1992; Mc Clelland & Rumelhart, 1986; Yates & Nasby, 1993). 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, 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 & 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 is used for the modeling of some pathological states, for example functional amnesia, multiple personality disorder and post-traumatic stress disorder.

In summary, neural network models and mainly parallel distributed model of dissociation suggest useful scheme for understanding dissociative processes in the real brain with respect to binding of neural assemblies and competitive relationships between them when they operate in the parallel distributed mode.

4. BINDING OF NEURAL ASSEMBLIES, CHAOS AND DISSOCIATION: A NEUROSCIENTIFIC THEORY OF DISSOCIATION

Recent experimental evidence suggests that neuronal synchronization is necessary for the large-scale integration of distributed neuronal activities. There is also increasing experimental evidence that coherent neuronal assemblies in the brain are functionally linked by phase synchronization between simultaneously recorded EEG signals and that this time-dependent synchrony between various discrete neuronal assemblies represents neural
substrate for mental representations such as perception, cognitive functions and memory (Lachaux et al., 1999; Varela et al., 2001; van Puten, 2003). These functions are related to distributed macroscopic patterns of neuronal activity which involve multiple neuronal subsystems bound into a coherent whole (Braitenberg, 1978; van Puten & Stam, 2001). A mechanism which enables binding of distributed macroscopic patterns of neuronal activity represented by neural assemblies into the coherent whole is still unresolved and represents fundamental problem in neuroscience (the so-called binding problem), i.e. how the brain codes and integrates distributed neural activities during processes connected to perception, cognition and memory (Woolf & Hameroff, 2001; Lee et al., 2003). The theory of feature binding originates in the theory of distributed coding which states that neurons involved in the processing of a single object will tend to synchronize their firing with each other, while simultaneously desynchronizing their firing from the remaining neurons not involved in the processing of that object (von der Malsburg & Schneider, 1986). An essential feature of a neuronal assembly coding is that individual neurons or subsystems can participate at different times in an almost unlimited number of different assemblies (Sannita, 2000; Varela et al., 2001). It implicates that the same neurons can participate in different perceptual events and that different combinations of these neurons represent different perceptual objects. Synchronization of these different perceptual objects is related to the integration of perceptions into a coherent whole (Singer & Gray, 1995). As a candidate mechanism for the integration or binding of distributed brain activities has been proposed synchronized high frequency electrical brain activity, the so-called Gamma activity, which represents high frequency oscillations at about 40 Hz, but often varying from 30 to 90 Hz. This activity occurs synchronously across brain regions and underlies the integration of diverse brain activities (Singer & Gray, 1995). Although the majority of research on feature binding has focused on synchronous Gamma activity (35–50 Hz), there is some evidence that synchronous activities in other frequency bands, may also participate in functional integration of distributed neural activities into the coherent whole (Bressler et al., 1993; Lee et al., 2003).

When there is a lack of synchronization then the system involves a large number of complex interlinked and simultaneously active neural assemblies and runs in desynchronized parallel distributed mode which can lead to self-organization. Parallel operation of two or more information processors systems leads to competition of disintegrated subsystems (Li & Spiegel, 1992) during numerous active information processing in sensory and cognitive channels of the cortex in desynchronized and simultaneously executed neural state. It 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 (Tirsch et al., 2004). Higher competition tends to the establishment of new associations among neural representations of mental states (Mölle et al., 1996) and decreasing complexity defined by number of oscillating neuronal cell assemblies. When the associated strength in these activated ranges of the neural network is low, it leads to the strong competition among the neural assemblies (Freeman, 1991). When two types of neural states are significantly different, then the states are separated by a wide gap in a neural state space as for example in PDP models. Freeman conceptualizes the relevance of nonlinear dynamic systems theory for these states of competition between neural assemblies and suggests that chaotic states between neural assemblies lead to an unpredictable and seemingly random behavior occurring in a system that should be governed by deterministic laws (Freeman, 1991, 2000, 2001; Skarda & Freeman, 1987). Chaos in the brain may occur when there is large number of complex interlinked and simultaneously active states. This can lead to the spontaneous order (self-organization) arising in a system when certain parameters of the system reach critical values (Isaacs, Daintith, & Martin, 2003). According to Freeman, the critical state can be reached by intensive competition among cortical neural assemblies with low associated strength which excite one another and they are unable to agree on a common frequency of oscillations (Freeman, 1991). Chaos leads to an often instantaneous reduction of excitatory thresholds of many neural
populations not excited in that particular combination before. A characteristic feature of neural activity due to brain chaos is a burst of synchronous collective activity in gamma band (Freeman, 1991). The burst waves often have a frequency at about 40 Hz and high amplitudes. Increasing competition as a consequence of failure of associative connections without possibility to agree on a common frequency of oscillations may lead after extremely intense and hypersynchronous gamma rhythms to epileptiform discharges and decreasing gamma oscillations (Medvedev, 2001, 2002; Alarcon et al., 1995; Allen et al., 1992; Fisher et al., 1992; Huang & White, 1989). In this context, epileptiform activity has been suggested as an antibinding mechanism which is related to a failure of associative connections between neural assemblies (Medvedev, 2001, 2002) and corresponding fragmentation of related mental representations characteristic for dissociative states. As a consequence chaotic self-organization during these processes may lead to pathological states such as epileptic paroxysms (Elger et al., 2000; Korn & Faure, 2003) or other manifestations of epileptiform activity when competitive neural assemblies remain disassociated or as unexpected original ideas due to successful creating of new associations during the chaotic competition (Elbert et al., 1994; Freeman, 1991, 2000, 2001; Skarda & Freeman, 1987; Mölle et al., 1996; Lutzenberger et al., 1992; Bob, 2003). According to propositions of PDP models the competition among neural assemblies on the psychological level is correlated by competition among mental representations (e.g., ideas, feelings or memories) and competitive neural assemblies with low associative strength represent neural base for dissociated mental states. The above suggest a key proposition for neuroscientific theory of dissociation which states that the competitive neural assemblies with low associative strength represent neural substrate for dissociated mental states which in the parallel distributed processing model are represented by isolated “peaks” (McClelland & Rumelhart, 1986; Li & Spiegel, 1992; Butler et al., 1996).

![Diagram](image)

**Figure 3.** The neuroscientific theory of dissociation deals with parallel levels of psychological and neural chaotic self-organization which leads on the neural level to epileptiform discharges. Epileptiform discharges as a probable consequence of dissociative states may produce a wide variety of psychopathological symptoms, somatoform symptoms or seizures which can alternate each other.
A key prediction of the theory is close relationship between manifestation of the dissociative state linked to extreme competition with chaotic features (on both neural and mental level) and epileptiform EEG activity.

5. DISSOCIATION AND EPILEPTIFORM DISCHARGES

Empirical confirmation with respect to prediction of the neuroscientific theory of dissociation is related to data which led to the epilepsy/temporal lobe dysfunction model of MPD that was at first mentioned by Charcot in 1892 (Putnam, 1997). This model corresponds to data that prevalence of seizure disorders is much higher in MPD patients (Mesulam, 1981; Schenk & 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 perictally, by seizure patients (Putnam, 1997). A neurobiological mechanism of a progressively increasing response of a group of neurons when exposed to a repetitive electrical stimulation mediated mainly by neurons in the hippocampus and amygdala called kindling (Colman, 2003) can potentially explain how epileptic-like phenomena might arise from repeated traumas (Post, Weis, & Smith, 1995; Putnam, 1997). It corresponds to clinical data that raise a possible role of temporal lobe abnormalities in pathological dissociation. Typical EEG abnormalities found in dissociative patients involve temporal or frontal slow wave activity (Putnam, 1997). Recent studies (Teicher et al., 1993; Ito et al., 1993) 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). There were also described connection of EEG abnormalities with dissociative symptoms, several dissociative syndromes including the patients with multiple personality, where epileptic activity during personality alterations was reported and also identity shift observed in temporal lobe epilepsy (Schenk & Bear, 1981; Mesulam, 1981; Coons, Milstein, & Marley, 1982; Benson, Miller, & Signer, 1986; Spiegel, 1991; Hersch, Yiu-Chung, & Smeltzer, 2002). 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) or mystical experiences which characteristically lead to changes in perception and identity. There are several findings which attribute epileptic activity to religious experiences (Saver & Rabin 1997; Alvarez, 2001) or out of body experiences (Blanke et al., 2002). Similar altered states of consciousness may manifest in creative artists. For example, Monroe (1978, 1982) reported that burst which is present as subcortical ictus correlates at times with a creative or an inspirational thought. Inspirational thoughts represent new ideas or creativity and in certain persons it correlates with epileptiform discharges, as for example in Vincent van Gogh (Monroe, 1982).

However contemporary literature does not support the concept that dissociative disorders can be generally explained on the basis of neurological dysfunction (Spiegel, 1991). Contemporary data support the suggestive evidence that temporal lobe seizure activity can produce dissociative syndrome, which is similar to that observed in functional cases. From these findings it may be inferred that temporal lobe epileptic activity is important in the generation of dissociative symptoms without neurological focal lesion. It corresponds to the evidence linking dissociative symptoms to the temporal lobe activity and indicates that the dissociative symptoms in temporal lobe epileptics occur during interictal periods and not during the ictal state (Spiegel, 1991). It is known that epileptic activity in interictal periods in temporal lobe epileptics produces characteristic symptoms called complex partial seizure-like symptoms. These symptoms represent intrusions into the normal state of consciousness in the form of cognitive, psychosenzory or affective symptoms (Roberts, 1993; Neppe, 1983; Roberts, Varney, & Paulsen, 1990; Silberman et al., 1985; Hines et al., 1995). Many of these
symptoms were already defined by Hughlings Jackson in his classical studies (Roberts, Varney, & Paulsen, 1990; Dreifuss, 1981; Roberts et al., 1992).

Modern findings support the view that these symptoms have, similarly as dissociation (Bernstein & Putnam, 1986), in the general population a continuous character (Roberts, Varney, & Paulsen, 1990; Roberts et al., 1992). This continuum of complex partial seizure-like symptoms begins in a healthy state without the symptoms via transitional pathological states until the symptoms of complex partial epilepsy with all typical manifestations. Between these opposites occurs a broad spectrum of different clinical dysfunctions with good response to anticonvulsant drugs. It concerns most often affective diseases or atypical psychoses with characteristic manifestations of temporal lobe epilepsy called as Epilepsy Spectrum Disorders (Roberts et al., 1992; Hines et al., 1995; Jampala, Atre-Vaidya, & Taylor, 1992). According to some findings (Roberts 1993) 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 abnormal electrophysiological activity and enhanced dissociative capacity (Roberts, 1993; Monroe, 1982). These data suggest close relationship between dissociative symptoms and interictal epileptic discharges. Further research may confirm this relationship by examining correlations between dissociation and complex partial seizure-like symptoms in epileptic patients as well as in patients with various psychiatric diagnoses. In addition further research may also confirm or falsify relationship between measurement of chaotic instability by Lyapunov exponents in biological signals (such as EEG, electrodermal activity etc.) and dissociation by using of sensitive measures of dissociative states, as for example State Scale of Dissociation (Kruger & Mace, 2002).

6. EPILEPSY/TEMPORAL LOBE DYSFUNCTION MODEL, HEMISPHERIC LATERALITY MODEL AND THEIR RELATIONSHIP TO THE NEUROSCIENTIFIC THEORY OF DISSOCIATION

According to neuroscientific theory of dissociation neurobiological manifestations of dissociative states are linked to competition between neural states and epileptiform discharges. Dissociative states are understood as a competition among disintegrated mental representations which represent the process that corresponds to probable EEG correlate of dissociative processes in the form of hypersynchronized epileptic (or epileptiform) activity, mediated by chaotic competition of neural assemblies corresponding to mental representations of conflicting psychic contents. In these cases conflict of dissociated mental representations may also include interhemispheric competition and dissociation of cerebral hemispheres as several studies suggest (Spitzer et al., 2004). On the “macro”-neurophysiological level dissociative processes in the case of interhemispheric competition 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 which in cases of very intensive competition may lead to chaotic self-organization. This postulate may motivate similarities between some cases of psychic dissociation (or repression) and split brain patients (Galin, 1974). Dissociation from this point of view may be understood as a blocking of communication between verbal (conscious) left and the right side of the brain which leads to competition between hemispheres. 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 & Bogen, 1969).

With respect to neuroscientific model of dissociation proposed above direct relationship between Epilepsy/Temporal Lobe Dysfunction model (Putnam, 1997) and Cerebral Hemispheric Laterality Model may be suggested. Cerebral Hemispheric Laterality Model 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, Schafer, & Miller, 1992; Ahern, 1993; Putnam, 1997). For example, Ahern et al. (1993) examined the relationship of “multiple personality disorder” in two patients with temporolimbic epilepsy to certain types of hemispheric interactions. 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 to the patient’s families to manifest themselves in the postictal period. These observations suggest that the association of multiple personality and temporolimbic epilepsy is not dependent on seizure discharges per se, but rather may be related to certain types of hemispheric interaction.

On the other hand there are controlled tests 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 likely are not bound to inter-hemispheric competition.

This suggests that competition between dissociated alter personalities and their neural representations may course in the form of intra-hemispheric as well as inter-hemispheric competition. This depends on the predominant qualities of competitive alter personalities. For example when the first alter will have left hemispheric dominance and the second right hemispheric dominance then the identity shift will be linked to laterality differences. When both alter personalities will have the right (respectively left) hemispheric dominance then the identity shift will not be connected to laterality differences.

7. TRAUMATIC AND UNACCEPTABLE MEMORIES AND THEIR RELATIONSHIP TO EPILEPTIFORM DISCHARGES

Dysfunctions in accessibility of memory traces linked to traumatic and other negative past experiences as well as intrusive autobiographical memories of childhood abuse are closely related to dissociation that represents the separation or segregation of specific ideas or affects from normal mental phenomena. Dissociation leads to an effort to eliminate these negative memories and increases intrusive thoughts connected to inner conflict due to contradictory tendencies when unacceptable or traumatic memory is released to the consciousness. There are several findings that activating of inner conflict due to stressful interview produce seizure activity in epileptics (Groethuysen et al., 1957; Stevens, 1959; Faber et al., 1996). Similar findings were reported also in normal healthy people during stressful interview concerned on embarrassing themes such as masturbation or homosexual relationships. During the interview burst waves in closed eyes similar to epileptiform discharges were activated (Berkhout, Walter, & Adey, 1969). There are also studies describing subcortical epileptiform activity during intensive emotional and psychopathological states which represented dissociated states separated from normal mental phenomena (Faber & Vladyka, 1987; Heath, 1962, 1975; Groethuysen et al., 1957; Monroe, 1978, 1982). For example, Monroe reported (1978, 1982) that epileptiform discharges within the subcortical emotional circuits were correlated with intense dysphoric and aversive affects as well as impulsive behavior.

Similar findings have been also documented by measurements of bilateral electrodermal activity in dissociated states during abreactive hypnosis. Hypnotic abreaction represents a useful therapeutic method for treatment of posttraumatic stress disorder. It involves re-living and re-experiencing an emotionally traumatic event under hypnosis and releases dissociated
contents with strong negative affect (Putnam, 1992). Psychophysiology of bilateral electrodermal activity (EDA) in these pathological phenomena is at this time poorly understood. Important data for this research represent intracranial stimulation studies which show dominant modulatory influences on EDA originating from ipsilateral limbic structures, where increased stimulation was accompanied by increased EDA response (Mangina & Beuzeron-Mangina, 1996). From these data it may be deduced that unilateral or bilateral changes in activity of limbic structures are reflected in ipsilateral changes of EDA and EDA may serve as a window into the limbic system. Reported material when re-living and re-experiencing of trauma from early childhood was linked to fast oscillations of bilateral EDA in the form of “spikes” similar to epileptiform EEG activity thus seems support existing data that epileptic type activity in the limbic structures and temporal lobe reflects important processes in the psychophysiology of dissociative states (Bob, 2004). Rapid fluctuations of bilateral electrodermal activity similar to “spikes” were also observed by Brende in a very upset and traumatized patient with multiple personality disorder (Brende, 1982, 1984). Chaotic states were also found in autonomic activity of a patient during psychotherapy in cardiac responses associated with psychologically meaningful events which displayed nonlinear characteristics indicative of chaos (Redington & Reidbord, 1992). Also these connections and experimental findings may be important for future research and the further understanding of the relationship between epileptiform activity and dissociative states.

8. DISSOCIATIVE SEIZURES, SOMATOFORM DISSOCIATION AND EPILEPTIC DISCHARGES

Dissociative seizures also called psychogenic nonepileptic seizures are in DSM IV and ICD 10 diagnosed as dissociative disorders. Main etiological factor is dissociative mechanism connected to conversion into somatic symptoms. This diagnostic classification of psychogenic nonepileptic seizures is due to growing evidence that these patients have a great deal of dissociative symptoms (Kuyk et al., 1999; Brown & Trimble, 2000). Prevalency of these pseudoseizures is not accurately known but probably it is 10-25% of all the patients visiting epileptologist specialists (Kuyk et al., 1999). Coexisting pseudoseizures and epilepsy have about 12-36% (Kuyk et al., 1999) and according to more recent data about 9-50% of all the visiting patients (Brown, Trimble, 2000). In epileptologist practice pseudoseizures and epilepsy are differentiated by finding of epileptic discharges on scalp EEG although also in patients with dissociative seizures subcortical epileptic discharges in limbic structures probably occur (Wieser, 1979).

This situation when seizures hardly differentiable from “true” epileptic seizures are produced by dissociative mechanism suggests interesting and important possibility to understand other somatoform manifestations of dissociation due to experienced stress and trauma in the so-called somatoform disorders as a category of mental disorders characterized by somatic symptoms of a general medical condition, but without independent evidence of any diagnosable general medical condition (Colman, 2003). Historically are these pathological manifestations associated with the term hysteria described by Pierre Janet, Joseph Breuer and Sigmund Freud in which mental and somatic factors are closely connected and understood as different aspects of a unit (Ellenberger, 1970). These pathological conditions were later re-defined by Janet as a consequence of dissociative mechanism which can leads to psychopathological as well as somatoform symptoms. Epileptic discharges as well are able to produce a wide range of psychopathological symptoms such as depression, psychosis, anxiety and other (Mace, 1993; Roberts et al., 1992) as well as a wide spectrum of somatic manifestations mainly due to autonomic symptoms of epileptic seizures (Baumgartner, Lurger, & Leutmezer, 2001). Autonomic symptoms accompany other seizure symptoms or may occur as sole or predominant seizure manifestation due to an activation of the central autonomic network. Spectrum of autonomic seizure manifestations is wide
ranging and can be divided into cardiovascular changes, respiratory manifestations, gastrointestinal symptoms, cutaneous manifestations, pupillary symptoms, genital and sexual manifestations as well as urinary symptoms etc.

Autonomic symptoms may be also lateralized as a consequence of a hemispheric-specific representation of the central autonomic network. It is known that these symptoms may occur similarly as various motor seizures also in non-epileptic conditions where represent difficult problem for differential diagnostics (Baumgartner, Lurger, & Leutmezer, 2001; Freeman & Schachter, 1995; Reeves, 1997). These data suggest close relationship between several somatoform dissociative states and non-convulsive epileptic activity in autonomic nervous system mainly in non-epileptic conditions when subcortical epileptic discharges related to dissociative processes may produce a wide spectrum of autonomic somatoform symptoms. With respect to other known symptoms of epileptic activity bound to sensory, sensitive and motor manifestations of seizures (Dreifus, 1981; Barry et al., 1985; Ghosh, Mohanty, & Prabhakar, 2001) may non-convulsive epileptic activity as a consequence of dissociation serve as a model also for other forms of somatoform dissociative symptoms such as alterations in sensation of pain (analgesia, kinesthetic anesthesia), painful symptoms, perception alterations, motor inhibition or loss of motor control, psychogenic blindness etc. which occur in conversion and somatoform disorders.

9. RELATIONSHIP BETWEEN EPILEPSY AND MENTAL ILLNESS: IMPLICATIONS OF THE THEORY

Biological antagonism between epilepsy and psychosis was at first investigated by a Hungarian physician László von Meduna (Meduna, 1934; Wolf & Trimble, 1985; Krishnamoorthy et al., 2002). After graduation his interest was concerned on brain anatomy and in 1927 he began his psychiatric career. At the time of his most important discoveries 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 & Trimble, 1985; Krishnamoorthy et al., 2002). Steiner and Strauss (Wolf & Trimble, 1985) presented 6000 schizophrenic patients and found that typical epileptic paroxysms in these cases are 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 along the antagonism between epilepsy and psychosis there may be a coexistence of both. In his practical therapy he used convulsive drugs, for example camphor or penetrazol, for the treatment of schizophrenia. These drugs led in many cases to convulsions and lowering of schizophrenic symptoms.

Following discussions of Meduna’s works initiated the development of convulsive therapy in psychiatry, nevertheless the relationship between epilepsy and psychosis was neglected for a period of time. His findings were resurrected 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 & 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 & Trimble, 1985). Forced normalization is often connected with decreasing epileptic changes in EEG and improving the control of seizures. On the other hand psychotic symptoms, as for example hallucinations may appear. These clinical manifestations of forced normalization may also include dysphoric states, hysteria and hypochondria, affective disorders, and miscellanea (twilight states). These reactions may begin spontaneously or as a consequence of antiepileptic medication. 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 anticonvulsant medication in patients with generalized epilepsy of the petit mal
type and in twilight states. Twilight states represent qualitative changes of mental state due to epileptic discharges which are very similar to manifestations of schizophrenia.

Recent findings suggest close relationship between epilepsy and schizophrenia (Stevens, 1999; Mace 1993). Schizophrenia as well as epilepsy is related to loss of physiological balance between excitation and inhibition. Epilepsy is linked to overexcitation and on the contrary schizophrenia is connected to focal overexpression of inhibition in the nuclei of the limbic system, hypothalamus and their projection sites (Stevens, 1999). Normal equilibrium between excitation and inhibition is in epilepsy permanently altered by repeated focal excitation or kindling resulting in a permanent state of excessive focal excitability and spontaneous seizures (Stevens 1999; Goddard, McIntyre, & Leech, 1969). Similar “kindling” or sensitization may be induced in inhibitory systems in response to focal physiological pulsed discharges of limbic and hypothalamic neurons and this excess of inhibitory factors may then be manifested as a psychosis (Stevens, 1992, 1999). This might correspond to intracranial EEG studies in schizophrenia patients which reported epileptic discharges in limbic structures (Heath, 1962, 1975; Monroe, 1982; Walter, 1944; Goon, Robinson, & Lavy, 1973; Sem-Jacobsen & Torkildsen, 1960). These data suggest that schizophrenia may represent a compensatory error which is physiologically reciprocal to epilepsy (Stevens, 1999) and it would correspond to observed reciprocity between epilepsy and psychosis in cases of forced normalization.

Effects of forced normalization may also occur in many cases of neurosurgical treatment focused on inactivation of the epileptic focus (Mace & Trimble, 1991; Blumer et al., 1998). Some authors thus point to the fact that kindling in mesolimbic dopaminergic system (characteristic for schizophrenia) 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 as between epilepsy and psychosis has also been found between epilepsy and depression (Jobe, Dailey, & Wernicke, 1999; Kanner & Balabanov, 2002; Chaplin, Yepez, R., & Shorvon, 1990; Trimble, 1996). Historical roots of this problem are connected to the term of forced normalization. Contemporary is thought that people with epilepsy exhibit a higher incidence of depression compared with people in general population (Jobe, Dailey, & Wernicke, 1999; Kanner & Balabanov, 2002).

Several studies also show that there is a relationship between seizures and affective disorders. 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 (Jobe, Dailey, & Wernicke, 1999). Similar situation is also in chemically induced seizures. Second reason is that the manifestations of forced normalization emerge in epileptic patients as an increasing of the occurrence of depressive symptoms when the frequency of seizures decreases. The response to anticonvulsant therapy may also cause worsening of affective disorders or psychotic episodes due to the sharply reduced number of seizures postsurgically. 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, Dailey, & Wernicke, 1999; Kanner & Balabanov, 2002).

There is growing body of evidence that anticonvulsant medications have emerged as powerful agents for the treatment of bipolar disorders as well as schizoaffective disorder or for the treatment of refractory depression (Jobe, Dailey, & Wernicke, 1999; Chaplin, Yepez, & Shorvon, 1990, 1990; Trimble, 1996).

On the other hand there is an extensive body of evidence that clinically useful antidepressant drugs can both prevent and cause seizures. According to contemporary literature antidepressant drugs suppress seizures when blood and brain concentration are relatively low. In contrast to this, seizures occur as a response to many antidepressants ingested in overdoses or in response to excessive blood levels (Jobe, Dailey, & Wernicke, 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, Dailey, & Wernicke, 1999). On the other hand there
is a great subgroup of depressive patients without epilepsy who manifest temporal lobe lability and have complex partial seizure-like symptoms and manifest a good response to anticonvulsant medication (Roberts et al., 1992; Roberts, 1993). The above data suggest a certain continuum between epilepsy and depression which may be explained by common pathogenic mechanisms of both, such as kindling (Kanner & Balabanov, 2002).

From the pharmacological point of view there is a similar relationship of reciprocity also between epilepsy and schizophrenia. Whereas epilepsy is associated with a decreased threshold for seizures, people with schizophrenia have an increased threshold. Anticonvulsant drugs which are used for treating epilepsy reduce brain excitability by blocking of Ca⁺ or Na⁺ channels or by GABA and monoamine enhancement. By contrast to that neuroleptics which are used for treatment of schizophrenia have antagonistic proconvulsive influence and are able to restore more normal excitability in overinhibited brain regions in schizophrenia. This corresponds to overexcitation in epilepsy and overinhibition in schizophrenia (Stevens, 1999).

In summary, forced normalization is often connected with decreasing epileptic changes in EEG and improving the control of seizures. On the other hand psychotic symptoms, as for example hallucinations may appear. On the other hand the clinical manifestations of forced normalization include also dysphoric states, hysteria, 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 & Rao, 2002). The term forced normalization is often considered in connection with EEG but 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 & Trimble, 1985; Krishnamoorthy et al., 2002). Some studies based on experimental findings show that in these psychotic patients subcortical epileptic discharges are present (Wolf & Trimble, 1985) as well as in patients with dissociative seizures (Wieser, 1979). Heath (1962, 1975) proposed that at the level of subcortical structures are epilepsy and manifestations of psychotic symptoms, for example hallucinations in schizophrenia, undistinguishable. Similar findings reported also other authors (Monroe, 1982; Walter, 1944; Goon, Robinson, & Lavy, 1973; Groethuysen et al., 1957; Sem-Jacobsen & Torkildsen, 1960) and suggest that manifestations of schizophrenic pathology are correlated with 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 corresponds in certain cases to the relation of reciprocity between mental illness and epilepsy.

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). There are also findings that secondary epileptogenesis and other connected phenomena may enable continuing of epileptiform activity in limbic areas with predominant psychopathological manifestations (Smith & Darlington, 1996; Krishnamoorthy et al., 2002).

Similar concept as Tellenbach’s alternative psychosis with respect to the transformation of epileptic and psychopathological symptoms were also observed in the study of dissociated states, where traumatic insult 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.). In this context, dissociation understood as psychobiological process of competitive neural assemblies connected to
Chaotic brain states and epileptiform activity suggests possible integrative view interconnecting the alternative psychosis, forced normalization and dissociative phenomena and suggests further possibilities for future research regarding common pathogenic mechanisms among epilepsy and several mental disorders.

10. CONCLUSION

Modern formulation of dissociation in the terms of discrete behavioral states and corresponding competing neural assemblies model presented above enables to reformulate the concept of dissociation in neuroscientific terms which is able to include also organic etiology of these pathological states. Definition of dissociation on the level of competitive mental representations related to competitive neural assemblies links dissociation with nonlinear chaotic self-organization which at certain circumstances can lead to decreasing gamma oscillations due to a lack of binding and manifestations of epileptiform discharges in the brain. From this point of view the relationship between dissociated states and epileptic activity suggests the explanation for the wide spectrum of psychopathological or somatoform dissociative symptoms.

This relationship between dissociated states and epileptic activity also explanatory framework for phenomena of forced normalization and alternative psychosis as alteration of symptoms due to persistent epileptic activity, hypothetically linked to chaotic brain dynamics. The neuroscientific theory of dissociation may serve as a theory for dissociative disorders and also for other mental disorders (for example schizophrenia or depression) or psychosomatic disorders which have in their pathogenesis dissociative mechanism as an important factor. A role of trauma and dissociation in etiology of schizophrenia and depression is still not fully understood but may represent important factor with respect to kindling hypothesis of both diseases. This model although is consistent with recent evidence requires further research which may enable integrated view of several psychopathological disorders, psychosomatic disorders and epilepsy.

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