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Psychogenic Pseudoepileptic Seizures – From Ancient Time to the Present

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1. Introduction

Clinicians who work with patients with epilepsy are confronted with many diagnostic and therapeutic challenges when have to differentiate between epileptic and psychogenic nonepileptic seizures (PNES). At the end of the twentieth century, the introduction of electroencephalography (EEG) recording with simultaneous monitoring of patient behaviour helped to correct false positive and false negative diagnoses of the nature of convulsive conditions. This technological advancement sensitized physicians to the high incidence of patients with PNES receiving referrals to clinical centres specializing in the treatment of epilepsy. When PNES is erroneously diagnosed as epilepsy, patients are at risk of prolonged, unnecessary, and above all, ineffective treatment with antiepileptic drugs. These drugs do not reduce the number of psychogenic convulsive incidents. Moreover, ineffective treatment leads to frequent visits to outpatient clinics and hospitalizations. It also leads to frequent change of doctors, strategies and forms of treatments. All this increases the cost of erroneous diagnosis and inadequate treatment.

PNES are defined as “episodes of altered movement, sensation or experience similar to epilepsy, but caused by a psychological process and not associated with abnormal electrical discharges un the brain” (Reuber and Elger, 2003) In current diagnostic schemes PNES are categorized as a manifestation of dissociative or somatoform (conversion) disorder (ICD-10).

This mean that they are caused by unconscious, symbolically expressed psychological processes leading to conversion, i.e. the pressing need to interpret one’s problems in ways which are both rationally and socially acceptable. This psychological mechanism has tangible gains, such as reduction of anxiety, and is a specific defence against the experience of other powerful and negative emotions. The external observer often feels that the patient is faking symptoms, unwittingly or even deliberately. He/she responds with irritation or even wants to blame the patient and make him/her feel guilty. The patient is not simulating, however. The misery associated with dysfunction is genuine and tangible. It is true,

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however, that patients with PNES do sometimes want to achieve certain goals and benefits which they could not naturally achieve by other means.

The role of cerebral organic factors in PNES is unclear. This type of seizures often coincides with mild brain injury. It has been reported for a long time that patients with nonepileptic seizures have more or less pronounced changes in their EEG recordings between seizures. They may even demonstrate typical epileptic EEG discharges. Changes in the brain structures have also been found using neuroimaging techniques. Both types of changes lead to the false diagnosis of epilepsy, especially when focal or generalized sharp waves, spikes or complexes of these elements and short waves are present. EEG changes have been variously interpreted in terms of cortical-subcortical connections or brain tissue hyper-reactivity, both of which can cause psychogenic disorders directly or indirectly. It therefore seems legitimate to assume that the predisposition to psychogenic seizures may be discussed in psychodynamic or psychosocial terms in lieu of brain dysfunction diagnosed earlier on the basis of EEG and neuroimaging. (Reuber et al., 2002, Jędrzejczak et al., 2004, Reuber, 2009, Auxéméry et al, 2010)

Treatment of PNES is equally controversial. There is no unified method of treatment as there is in epilepsy. Because PNES is so complex and multifaceted and is often accompanied by anxiety, depression or attention deficit, it is highly advisable that both psychiatrists and psychologists cooperate in the treatment. At present the diagnosis and treatment of PNES is largely in the hands of neurologists (epileptologists). It is currently thought that the doctor must first either rule out the epileptological nature of the disorder or diagnose the comorbidity of epilepsy and PNES and determine the proportion of both types of seizures. He/she can then begin to treat the PNES. Rational psychotherapy based on meticulous diagnosis and tailored to the specific patient can be effective.

2. Historical background

Psychogenic nonepileptic seizures have been traditionally associated with the concept of hysteria. However, reports of PNES are as old as reports of epilepsy. In various ancient cultures and civilizations the causes of various types of seizures were found. We also find accounts of various methods of prevention and treatment. Ancient written sources can roughly be divided into two groups: philosophical-medical and religious. The religious literature quotes and reflects the knowledge presented in philosophical and medical texts to various degrees. Historical sources often contain many superstitions and prejudices about epilepsy. Many people suffered not so much from the seizures themselves but from misunderstandings concerning their prevention and treatment. Today we can assert that the attitudes of whole societies toward epilepsy and the way patients who suffered from epileptic incidents were treated attested to the level of their social conventions and the progressiveness of their culture.

2.1 Accounts of epileptic seizures in the religious literature

In the Old Testament, all diseases are viewed as punishments for the sins of individuals or entire societies and as cautions against the violation of divine laws. Epilepsy was no exception except that it was called the “falling disease” in the original text. Yahve was the one who inflicted people with epileptic attacks and only he could alleviate their suffering. In

chapter 21 of the First Book of Samuel we find the story of young David escaping from Saul to the Philistines. In order to avoid being taken hostage he simulates epileptic seizures whose symptoms, according to contemporary nomenclature, resembled tonic-clonic seizures: “So he pretended to be insane in their presence: and while he was in their hands he acted like a madman, making marks on the doors of the gate and letting saliva run down his beard” (I Sam 21:13). This is surely the oldest existing account of nonepileptic seizures.

Where there are nonepileptic seizures there must be a model which can be emulated. Without much further searching we find in the same book a description of the peculiar behaviour of Saul, king of Israel, who discards his clothes then falls to the ground and lies there all day and all night. This reminds us of an epileptic state probably preceded by a psychomotor seizure. These seizures were a punishment which God inflicted on Saul for his vile and disobedient acts. Saul repeatedly tried to kill David who was designated to be the next king of Israel. In those days seizures were thought to be symptoms of a mysterious malady but their specific form, course and circumstances were submitted to rational rules and interpretations in terms of cause, negative consequences and advantages.

In the fourth book of the Pentateuch we find the story of Balaam, a Midianite, who prophesied the coming of the Holy Spirit. He saw this prophesy in his mind's eye when he was stricken to the ground and learned the will of the God Most High. Inscriptions discovered in the Middle Jordan Valley and dated at 750 BCE attest to the activity of Balaam, son of Beor, in the days of the Middle Kingdom. Known for his talents, he was summoned by Balak, king of the Moabites, to curse the group of Israelites who settled in Moab upon leaving Egypt. Balaam was promised a handsome reward for his efforts. Three times did Balak make offerings to God in Balak's presence and three times did Yahve bless the Israelites, enraging the king. In the biblical account of this event we read that Balaam fell to the ground during the blessings. The ancient Hebrew term for this falling is *nophel*, the same term that was used to describe Saul's seizures. The word evidently conveys the action of the spirit, be it odd or impure. This analysis of the stories of Saul, David and Balaam is testimony to the belief, which was widespread in ancient times but is also quite popular today, in the tragic inevitability of poor prognosis in patients, their inferiority, and interpretation of human seizures in terms of God's punishment for their evil deeds and vile life. Seizures are repeatedly described in the Pentateuch. From our contemporary perspective it is hard to say if these were epileptic seizures or conversion dissociative attacks. What is typical, however, is that they always occur in unusual circumstances, as the only solution to a seemingly impossible situation which cannot be resolved happily or even satisfactorily.

2.2 Middle ages

In ancient times Hippocrates and Aretaeus of Cappadocia left extremely accurate accounts of both types of seizures (Veith, 1965; Lennox & Lennox, 1960). Treatment varied in various cultures depending on the level of medical and philosophical knowledge and was the domain not only of medics but often also of shamans, exorcists and other religious hierarchs. Throughout the Middle Ages, but also partly in modern times, both epilepsy and hysteria were believed to be signs of possession by the devil but were thought nevertheless to be two distinct conditions. The concept of hysteria is rooted in the idea, already discernible in Egyptian medicine that functional disorders are caused by the wandering of

the uterus (Ebbel, 1937). As it wandered to different parts of the body, the uterus caused a variety of symptoms. The Hippocrates (2005) school attributed hysterical disorders in women to sexual abstinence. Abstinence was thought to make the uterus “anxious” and to move to the upper parts of the body. If, for example, it reached the respiratory system, the patient had difficulty breathing and turned blue. Restricted breathing was a source of “anguish” and made the patient ill. Mainly spinsters and widows were prone to hysterical attacks and prompt marriage was thought to be the best cure. Fumigation of the uterus was another radical remedy. Hippocrates is also believed to have been the first writer to differentiate between genuine epilepsy and hysterical seizures. He recommended the following diagnostic procedure: press the abdomen energetically above the ovaries. If this evokes a seizure, hysteria shall be diagnosed. Like all his contemporaries, Hippocrates believed epilepsy to be a male disease and hysteria to be a typically female disease (Trillat, 1993). This belief is surprisingly consistent with contemporary observations that many more women than men have psychogenic nonepileptic seizures. (Jedrzejczak et al., 1998, Lesser, 1996, Selkirk et al., 2008). Roman medical authorities discarded the wandering uterus hypothesis but continued to associate hysteria with the female reproductive system. In the 17th century English physicians declared that hysterical attacks originated in the brain, not the abdomen, but they continued to endorse the idea that epileptic attacks and hysterical attacks were related and that they shared the same pathophysiology. Some of them argued that hysteria was caused by an excess of passion (Arts, 2001).

2.3 From the Middle Ages to the Enlightenment

In the Middle Ages hysteria, although not so well-understood as epilepsy, was extremely widespread. The supra-naturalistic climate is partly responsible for this. Demons, devils and other evil powers reigned in both theological and medical circles, affecting social ideas concerning hysterical phenomena. Fear of the devil of panic proportions and religious ecstasy were fertile soil for the development of morbid affective states and hysteria. Bourneville, a representative of the Charcot school, threw some light on this problem. In the last decade of the 19th century he published a series of monographs entitled *Collection Bourneville* or *Bibliothèque diabolique* containing many original 16th and 17th century documents. In this excellent work he compared historical examples of alleged witches and demoniacs with contemporary forms of hysteria pointing that in Middle Ages powerful Catholic church forced a spiritual interpretation of disease – servants of Satan tried to confuse the minds of people (Szumowski, 1994).

The term *demonopathia hysterica* was coined in the Middle Ages. According to magic-theologic theory, hysteria is the consequence of possession. It is a malady of the soul which manifests itself via somatic symptoms. Possessed women, stigmatized with behaviour which others could not understand, were submitted to cruel exorcisms and other bestial procedures, often widely condoned, which frequently maimed them or even killed them (Fig. 1).

Evidence of these murky times and the opinions and customs which affected the lame and the sick can be found in the Dominican textbook whose full title is *Malleus Maleficarum. Acts of witchcraft, and how to protect oneself against them, and remedies, all encompassed in two parts* (Sprenger & Instytor, 1992). This book was written by two inquisitors designated by Pope

Innocent VIII and sent to south Germany to purge those lands from heretics and practitioners of Satanism. The book contains unequivocal evidence that it was not difficult in those days to be accused of witchcraft or black magic. It was hard not to admit to such practices when one was being submitted to elaborate and cruel tortures. People who looked odd or were behaving violently were thought to be possessed by the devil or practicing witchcraft and were submitted to the water test. The suspect (usually female) was thrown into the river to see if she would drown or not. If she didn't, this meant that impure forces were keeping her afloat. If she sank, this meant that she had been too hastily and probably incorrectly accused. One way or another, women accused of witchcraft and submitted to such brutal practices lost their lives. Many centuries had to elapse before the Enlightenment dispersed the darkness of superstition, prejudice and stigma engulfing men and women suffering from psychogenic nonepileptic seizures.



Fig. 1. Hundreds of innocent people were burnt at the stake because of the inquisitors' fear of witchcraft and diabolic possession.

Other group incidences of hysterical behaviour were also observed and described in the Middle Ages. Particularly famous were the epidemics of mass dancing, called the dancing plague or tarantism, which overcame even several thousand people at a time (Fig. 2).

A barking epidemic broke out in France in 1609. In a small town 40 people barked like dogs during hysterical attacks (Szumowski, 1994). However, possession epidemics usually broke out in monasteries. The monastery epidemics in Loudon and Louviers in the 15th century were famous. Possession could also be sexually motivated. This was particularly obvious during the epidemic which broke out in a monastery in Auxonne. Mediaeval hysteria can clearly be divided into demonic hysteria and mystical hysteria. Demonic hysteria is

probably more akin to what we typically call hysteria. But mystic hysteria often conveys the image of a person capable of sacrifice and suggestible. Many of these phenomena were viewed as supernatural and perhaps we should view them as meta-psycho-physiological phenomena.



Fig. 2. Peasants in the throes of hysteria manifested in mass dancing.

Wier, a distinguished 16th century physician and expert in diabolic phenomena, wrote that “the number of patients who say that they are possessed by the devil is infinite” (Szumowski, 1994). In the Middle Ages, hysterical women prone to suggestion guessed the exorcist’s intentions and demonstrated various symptoms. They often had split personalities and gave the impression of being possessed by the devil. Their movements were lewd and lascivious, their language was obscene, and the devil was allegedly to blame. One nun spat out the Holy Communion straight in the priest’s face. When in a state of hysterical frenzy, people (usually women) exhibited extraordinary physical strength. “One heroine of the madness epidemic in Auxonne, a woman of slight build, grasped a stone holy water stoup which normally could barely be lifted by two men, lifted it from its pedestal and threw it to the ground with the greatest ease” (Szumowski, 1994). The typical symptoms of hysteria include complete or partial dermal anaesthesia. Patients did not react to pricking with a needle or touching with a hot object. These changes in sensory sensitivity played an important role in mediaeval witchcraft trials because the sites of anaesthesia were thought to signify a pact with the devil.

During the Enlightenment many monographs extracting hysterical behaviour from the darkness of shamanism and superstition were written. Susceptibility to hysteria now began to be associated with personal disposition, temperament, or other genetically determined traits and hysterical attacks were attributed to traumatic life events. In the 17th century Sydenham drew attention to the similarity between high fever states and hysterical behaviour. He found that one-sixth of all patients who consulted a physician were hysterics (Pechy, 1772). In the 18th century Boerhaave coined the term “nervous disease” and it was he who introduced a simple albeit drastic diagnostic method, burning with a hot iron, during the epidemic of hysteria. Seizures which did not subside in response to this cruel treatment were diagnosed as epileptic (Sokołowski, 1950).

Pierre Briquet (1796-1881) published the first systematic review of hysteria, *Traité Clinique et Therapeutique de L'Hysterie*, in 1859. He presented three main types of seizures: spasmodic seizures, *syncopal attack* and hysterical convulsions. Hysterical convulsions were the most frequent type. Their course and clinical manifestations resembled epileptic seizures most closely. Briquet also found that his patients were most familiar with epileptic seizures. According to his ideas – very modern at the time – hysterical symptoms were closely related to their causes which were largely emotional. Briquet’s theory was based on the assumption that every painful and unpleasant event is registered in the brain’s emotional centres and it is that part of the brain which reacts, resulting in external manifestations. This could help to differentiate between hysterical and epileptic attacks. In hysterical attacks “convulsions are an expression of feelings, passions or life events”. In epilepsy “convulsions are a kind of tetanus contraction, quite unlike the movements which occur in physiological conditions” (Briquet, 1859). From our contemporary point of view this is an extremely simplified idea but it is worth pointing out that, unlike, nonepileptic seizures, epileptic attacks reflect the cortical motor representation. Also, automatisms reproduce everyday movements such as buttoning one’s clothes. Today it is hard to accept that hysterical attacks are simply the direct outcomes of cerebral emotional states.

2.4 The 19th century – “grand hystérie”

It was not until the 19th and 20th centuries that hysteria began to be understood better. In 1853 Carter presented an extremely mature monograph of hysteria in which he suggested three etiological factors: disposition, circumstances and undisclosed causes (usually sexual passion) (Carter, 1853).

Most of the data on hysteria in those days came from France where Jean Martin Charcot and his students at La Salpêtrière clinic made hysteria the focus of their attention. The unfortunate notion of hysteroepilepsy was introduced in France at that time, suggesting a link between the two disorders. The term was used to describe the coexistence of epileptic and hysterical attacks in the same patient, epileptic attacks triggered by suggestion (what we now call psychogenic nonepileptic seizures) and finally hysteroform epileptic attacks considered to be an intermediate form between epilepsy and hysteria. According to Charcot, hysteria was a disorder of the nervous system, a “neurosis”, like epilepsy and migraine. He believed that hysteria, just like the two other diseases, was the consequence of structural or functional damage. In hysteria, the damage is caused by the coexistence of inherent dispositions and an external trigger such as physical or emotional shock (Charcot & Marie, 1892). Richter, a student of Charcot’s, clearly established that hysteria was a defined disease

quite unrelated to either supernatural forces or epilepsy (Temkin, 1945). Charcot gave extremely detailed accounts of hysterical symptoms which he described, analyzed and photographed but unlike Freud, who also studied hysteria, he was not interested in his patients' life histories. Charcot distinguished two main forms of hysteria – with and without convulsions. Most varieties of hysteria have features which enable differentiation between hysterical and nonhysterical disorders. In hysterepilepsy the most reliable factor was allegedly the “tubal symptom”, hypersensitivity in the vicinity of the fallopian tubes. Application of pressure to this area could trigger an attack of hysteroepilepsy but not an epileptic attack. This way, the history of medicine travelled full circle from Charcot to Hippocrates and back.

In 1880 Charcot and Paul Richer (1849-1933), one of his students and collaborators, began an extensive study of over 100 female patients with hysteroepilepsy. They published the results in the book *Études cliniques sur l'hystéro-épilepsie ou grand hystérie* in 1885. The book was richly illustrated by Paul Richer.

According to Charcot, grand (convulsive) seizures can occur in cases of severe hysteria. The patient has contractions, like the ones experienced by patients with epilepsy, then falls but usually does herself no harm, has facial grimaces and violent movements of the extremities, clenches her fist as if she were angry, screams, whines, laughs or weeps (Figs. 3, 4, 5).

One of the classical symptoms of the grand hysterical attack is the arching forward of the whole trunk (*opisthonus*) so that the body, when lying on the floor or the bed, rests only on the head and the heels (*arc de cercle*) and the abdomen protrudes upward (Fig. 6).



Fig. 3. The initial phase of a hysterical attack, Paul Richer, *Études cliniques sur l'hystéro-épilepsie ou grand hystérie* (Paris, 1885).

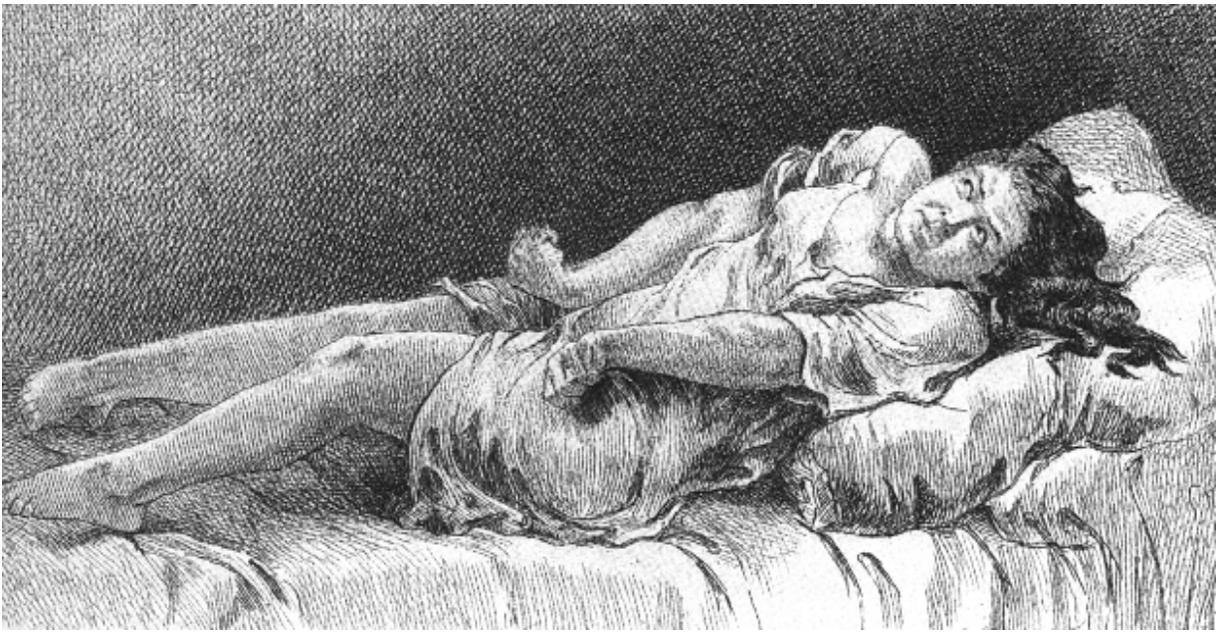


Fig. 4. The tonic phase of a hysterical attack, Paul Richer, *Études cliniques sur l'hystéro-épilepsie ou grand hystérie* (Paris, 1885).



Fig. 5. The motor phase of a hysterical attack, Paul Richer, *Études cliniques sur l'hystéro-épilepsie ou grand hystérie* (Paris, 1885).

If these seizures are frequent enough, the two ends of the arch, the head and the heels, can touch. The main characters of the Auxonne monastery madness even “walked” in this pose. The body poses and facial countenance sometimes express feelings such as surprise,

religious ecstasy, anger or erotic passions (Charcot's *attitudes passionnelles*), and the body moves in harmony with these feelings (Figs. 7 and 8).



Fig. 6. The circular arc – “arc de cercle”, Paul Richer, *Études cliniques sur l’hystéro-épilepsie ou grand hystérie* (Paris, 1885).



Fig. 7. Bodily poses and facial countenances expressing intense emotions – attitudes passionnelles, Paul Richer (*Études cliniques sur l’hystéro-épilepsie ou grand hystérie* (Paris, 1885).



Fig. 8. Bodily poses and facial countenances expressing intense emotions – attitudes passionnelles, Paul Richer (*Études cliniques sur l'hystéro-épilepsie ou grand hystérie* (Paris, 1885).

Richer also suggested that the attack of *grand hystérie* goes through four typical stages. In the first, epileptoid, stage the patient has a tonic seizure, usually preceded by an aura. She then begins to have acrobatic movements (*grands mouvements*) in which she assumes spectacular poses like the aforementioned *arc de cercle*. This second stage is followed by the expression of emotions by means of gestures and words (*attitudes passionnelles*). Finally in the fourth stage the attack ends with a kind of delirium. This fourth stage was often omitted in later publications (Arts, 2001).

The cases from Charcot's clinic reported in *Iconographie photographique de la Salpêtrière* are extremely instructive. The images captured in the clinic match the experiences of mediaeval hysterical women who claimed to have had sexual intercourse with the devil. The patient would lie on the bed in a cataleptic slumber and have, for example, sexual visions accompanied by appropriate words and movements. Following the attack the patient would fall into a deep sleep lasting even several days or weeks. This sleep resembled lethargy where breathing and heart function are so weak that the patient looked as if she were dead. The author also reports a case of authentic hysterical agony. On the other hand, some patients manifest enormous physical strength during a hysterical attack. One male patient from Charcot's clinic, barely seventeen years old, unscrewed the iron bed railings, pulled out an iron stair balustrade, lifted a whole heavy iron bedstead together with the bedclothes and threw it onto the neighbouring bed when in the throes of a hysterical attack (Szumowski, 1994).

Many writers tried to refute Charcot's ideas after his death. The Belgian physician Joseph Delboeuf and the Swedish physician Axel Munthe helped to discredit the *Salpêtrière* school. They argued that the patients who demonstrated hysterical attacks at Charcot's Tuesday lectures were coached by a team of doctors and that, in addition to the pleasure they gleaned from their demonstrations, they were rewarded for their convincing performances.

Babiński, himself once a member of the *Salpêtrière* school, also became a critic but his was a global criticism of the entire concept of hysteria (Arts, 2001). On the other hand, his work should be seen as the beginning of psychological interpretation of traumatic neurosis.

Conversion disorders were initially thought to be neurological because their symptoms resembled the symptoms of somatic diseases. Hysteria was called the great malingerer. Freud and Breuer (1896) argued that conversion disorders were psychogenic and were related to early childhood sexuality (Aleksandrowicz, 1998). Freud had the greatest influence on the understanding of hysterical disorders in the 20th century. His libido concept and his principles of psychoanalysis as a method of understanding and solving unconscious internal conflicts continue to be known and applied, with certain modifications, today. According to psychoanalytic assumptions, conversion disorders were symbolic expressions of repressed experiences. Contemporary psychoanalytic formulations do not limit the range of these experiences to the sphere of incestuous desires, however.

3. Contemporary conceptualizations of conversion and dissociative disorders

3.1 Paroxysmal disorders

In the 19th and 20th centuries the concept of hysteria evolved within the concepts of conversion and dissociation. Earlier classifications qualified dissociative disorders as a hysterical *neurosis pithiatica* syndrome, i.e. the co-occurrence of conversion symptoms and specific personality traits (Aleksandrowicz, 1998). Modern psychiatry (particularly American) tends to separate the two disorders and view them as two distinct diseases. The DSM-IV places conversion in the somatoform disorder group and dissociation in a quite separate group, the dissociative disorder group (American Psychiatric Association, 1994). The DSM-IV has completely eschewed the category of neurotic disorders and classifies psychogenic non-epileptic seizures as conversion disorders. The ICD-10 is less explicit on this issue. In accordance with the European psychiatric perspective, neurotic disorders are included and classified as a subgroup of the larger class of dissociative (conversion) disorders. This group includes amnesia, fugue, ataxia, apraxia and anaesthesia. Meanwhile, depersonalization and derealisation, which can also imitate epileptic seizures and are usually associated with the concept of dissociation, are classified as “other neurotic disorders”. Finally, psychogenic pain and vegetative disorders are classified as “somatoform disorders”.

These confusions suggest, on the one hand, that it is difficult to diagnose patients with nonepileptic seizures and, on the other hand, that we need to identify the pathological mechanisms of these disorders in order to determine whether the same mechanism is leading to this or that symptom or whether the variety and abundance of symptoms is a reflection of the heterogeneity of the etiopathogenic context (Jędrzejczak, 2007).

To understand paroxysmal disorders (both epileptic and pseudoepileptic), we must understand both the physical and the psychological factors influencing each individual patient. This is particularly important when we want to come to grips with somatoform disorders which greatly contribute to the etiology of nonepileptic seizures. It is now thought that conversion disorders are psychogenic and that their onset strictly coincides with traumatic events, difficult or “unbearable” situations or dysfunctional interpersonal

relations. Symptoms often reflect patients' fantasies concerning the course of somatic illness. Symptoms may be symbolically linked to the psychological cause. For example, the patient who saw terrifying scenes becomes blind. Pseudoepileptic seizures in conversion disorders are probably indirect (symbolic) manifestations of anger, fear, helplessness, or loss of control (Betts, 1997). In men seizures symbolizing helplessness can be found in victims of war trauma.

3.2 Affect conversion

The estimated prevalence of conversion disorders in general practice is from 15% to 30% of all hospital admissions (Folks et al., 1984; Wolańczyk et al., 1994, Jedrzejczak et al., 1999, Szaflarski et al., 2000, Angus -Leppan, 2008). Conversion disorders often give the impression that the patient wants to be ill, "is escaping into illness", and unconsciously or even consciously feigning illness. This impression sometimes causes irritation with the patient and the wish to blame the patient and make him/her feel guilty. This reaction is unjustified, however, because the suffering which accompanies the dysfunction is genuine. It is true, however, that patients do sometimes have their own personal agendas and seek certain benefits which they cannot achieve by other means.

The current classification of conversion and dissociative disorders is rooted in 19th century research on hysteria and defence mechanisms. Early psychological theories conceptualized these disorders as unconscious conversions into physical symptoms in order to resolve conflict or reduce anxiety. Stress caused the symptoms. In cases of acute reactions to stress this is probably correct. The trouble begins when, as is sometimes the case, patients benefit from a particular response to stress. If this behaviour is reinforced or rewarded it may be repeated and become chronic. However, it is not always easy to trace the direct and obvious relations between symptom onset (cause) and its indirect psychological consequences and therefore it is hard to reconstruct the psychological mechanism underlying the repetitive disorder.

3.3 Dissociative disorders

Dissociative disorders are processes leading to various clinical symptoms and these symptoms represent a broad range of problems confronting neurologists and psychiatrists.

ICD-10 (1992) gives the following definition: "what dissociative and conversion disorders have in common is partial or complete loss of normal integration between memories of the past, sense of identity, sensory sensations and control of body movements" which are normally under conscious control. In dissociative disorders conscious, selective control of these functions is reduced. The patient is no longer able to manage his/her behaviour at will or move his/her body in a controlled way. "Conversion" sets in, i.e. emotions are transformed into physical manifestations of unpleasant experiences. Dissociation is thought to be caused by stress and stressful life events with which the patient is unable to cope, and the most important consequence is loss of voluntary control of somatic functions which cannot be attributed to organ or system condition (Aleksandrowicz, 1998). Definitions of this group of neurotic syndromes tend to ignore the sphere of disordered experience, particularly unconscious experience. In the psychodynamic approach to neurotic systems, the natural functioning of the human psyche includes both spheres which are accessible to

awareness and those which are unconscious and conceal unbearable problems, experiences and memories (Kokoszka, 1998). This approach gave rise to the concept of defence mechanisms, of which repression is the most important. Defence mechanisms help to keep these contents unconscious. If repression is not quite successful, other defence mechanisms such as intellectualisation, isolation or rationalisation are resorted to (Meissner, 1985). If they too are unsuccessful, neurotic symptoms develop. These symptoms serve defensive functions. They reduce conscious anxiety or other unbearable emotions (“primary gain”) and the symptom is easier to accept than its cause (Kokoszka, 1988). The symptom symbolizes the concealed repressed content. Patients also glean secondary gains from their neurotic symptoms when the environment responds sympathetically and reduces the misery caused by the symptoms.

3.4 Difficulty classifying paroxysmal disorders

The DSM-IV also includes dissociative disorders in its classification system but distinguishes them from conversion disorders and defines them (e.g. fugue, depersonalization) as rupture of the normally integrated functions of consciousness, identity and environmental perception. It defines conversion disorders as symptoms or deficits concerning any motor functions and sensory functions, suggesting neurological or other health disorders. Conversion is understood as a symptom, not a disease, and it can have various causes.

Most of the dissociative disorders specified in separate ICD-10 categories are syndromes whose occurrence is associated with a single psychological trauma and in which there is one dominant symptom, often with sudden onset, which usually recedes within several weeks or months. These disorders become chronic when there are underlying insolvable problems. The most important dissociative (conversion) disorders are:

- dissociative amnesia
- dissociative fugue
- dissociative stupor
- dissociative motor disorders
- dissociative convulsions
- dissociative anaesthesia and loss (disturbance) of sensation.

Psychogenic nonepileptic seizures are behaviour disorders which resemble epileptic attacks in which the focal or generalized neuronal discharges which are typical for epilepsy are absent. According to the ICD-10 classification system, dissociative (conversion) disorders also include dissociative stupor and dissociative convulsions whose most frequent manifestations can be erroneously confused with epileptic attacks. In dissociative stupor the main symptom is psychomotor inhibition – loss or serious reduction of voluntary movements, lack of response to external stimuli such as noise, voice or touch, loss of contact. Patients usually recline, immobile and speechless. In dissociative convulsions consciousness is fully retained or altered like in stupor. Paroxysmal attacks may resemble the “hysterical attacks” so often described in the 19th century. Dissociative stupor and dissociative convulsions are the forms most frequently confused with epileptic attacks and that is why they are called psychogenic pseudoepileptic seizures.

A review of the literature shows quite clearly that the physician's behaviour and expectations usually modified or intensified the phenomena they described. Charcot and Freud's work illustrates this very well. One extremely interesting question is why non-epileptic seizures were so popular in times when epilepsy was much more stigmatised than it is today and there was much greater risk of detention in the uncomfortable psychiatric hospital. From our contemporary perspective, perhaps non-epileptic seizures symbolized problems with which patients could not possibly cope?

It is still unclear what causes various types of disorders. Many symptoms found in psychogenic nonepileptic seizures can also be found in the course of dissociative (conversion) disorders. These disorders do not have a homogenous clinical picture. On the contrary, their symptomatology is typically heterogeneous (Jędrzejczak, 2007).

Many questions and doubts come to mind.

Why do some patients with conversion disorders have fugues or dissociative amnesia, for example, whereas others have PNES? And pursuing this query further, what is the difference (if there is any difference) between patients who develop dissociative stupor and patients who develop dissociative convulsions? The observation that conversion disorders assumed many different forms throughout the ages further confirms that this is a mutable and heterogeneous phenomenon. Also, not everyone who is stressed develops conversion disorders. Whether they do or not probably depends on many factors such as culture, socioeconomic circumstances, level of education, life experience, sex, etc. Other important factors are personality and habitual coping mechanisms. Finally we must also consider the contribution of the history of mental and somatic illness. It is worth pointing out that culture does not have a major effect on the expression of psychogenic pseudoepileptic seizures. Although the course of PNES varies greatly, similar patterns can be found worldwide. The semiology of PNES is similar in western countries, Taiwan, Lebanon and India (De Paola, 2003; Lai and Yu, 2003; Riachi, 2003; Krisznanmorrthy et al., 2003).

PNES occur with a variety of psychiatric diagnoses, with a variety of psychic risk factors but without a uniform psychological/psychiatric profile. Reuber et al (2004) found distinct types of maladaptive personality disorders as the psychiatric basis for PNES. However, most experts assume that no single mechanisms or even contributing factors has been identified that is sufficient to explain PNES in all patients. PNES occur in a heterogeneous patient population and in contrast to epileptic seizures no commonly accepted classification of PNES exists. It is important to find PNES subtypes that differ with respect to etiology. The first step is to use objective instruments to study clinical semiology of PNES. Using quantitative cluster analysis based on 125 patients with PNES (Jędrzejczak 2007) developed a classification system with four different types of PNES. All types have a characteristic pattern, i.e., motor symptoms intensify progressively starting with immobility (PS4), through simple motor symptomatology (PS2), trembling of the body (PS3) and ending with complex, sudden motor symptoms (PS1). Gröppel et al. (2000) presented such an analysis but it was based on a very small sample (29 patients). They identified three clusters: seizures with a clearly expressed motor component, seizures with minor motor symptoms and trembling and a third cluster of atonic seizures, which they defined as falling down without motor manifestations (only one patient in Gröppel et al.'s sample had this symptom). As far as diagnostics are concerned, this distinction helps to identify differential criteria for psychogenic pseudoepileptic seizures and epileptic seizures. It also provides a new

perspective on prognosis. If we manage to distinguish homogeneous groups of patients with psychogenic pseudoepileptic seizures, this may be a milestone on the road to developing a more detailed description of such patients, particularly from the point of view of etiology and the pathophysiology of psychogenic seizures.

Models explaining the pathogenic mechanisms leading to these events are limited. In paper of Baslet (2011) presented is a hypothetical pathophysiological model suggesting an alteration in the influence and connection of brain areas involved in emotion processing onto other brain areas responsible for sensorimotor and cognitive processes. Integrating this information, PNES are conceptualized as brief episodes facilitated by an unstable cognitive-emotional attention system. (Baslet 2011). According to Reuber (2009) PNES should be understood based on multifactorial etiologic model. He differentiates predisposing factors (abuse, neglect, interpersonal problems) means increase vulnerability to development of PNES. The next group covers precipitating factors (stressful experience, physical or mental health symptoms) which can occur days or months before onset of PNES and cause PNES to start. Perpetuating factors that inhibit ability to gain control over seizures. Anxiety was identified as relevant perpetuating factor in more than 50% of patients with PNES (Reuber, 2009).

What we want to know is whether the observed heterogeneity of clinical symptoms is caused by one and the same etiology or whether each clinical manifestation has its own specific pathophysiology. Our present state of knowledge does not allow us to answer this question unequivocally.

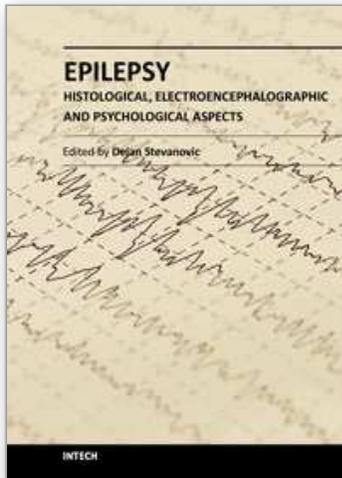
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