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ORGANIC CATATONIA: A REVIEW

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ABSTRACT

Catatonia is a clinical syndrome associated with a wide variety of psychiatric, medical and neurological disorders. Despite several reports in the literature of a wide range of medical and neurological diseases causing catatonia, there has been a tendency to consider catatonia as purely psychiatric disorder. The review attempts to look at the concept of organic catatonia from a historical viewpoint, including its place in the psychiatric classification, discusses the various etiological causes of organic catatonia, and them goes through some important management issues in organic catatonia. The review suggests that organic catatonic disorder must be first considered in every patient with catatonic signs, particularly in a patient with new onset catatonia.

Key words: Catatonia, organic catatonia, organic catatonic disorder

There has been a tendency to consider catatonia as a purely psychiatric disorder, despite many case reports demonstrating a wide range of medical and neurological diseases, as well as psychiatric causes, associated with catatonic symptomatology (Barnes et al, 1986, Clark & Rickards, 1999a). The common occurrence of catatonic symptoms in schizophrenia has in fact diminished, with most catatonic symptoms presenting now in association with either mood disorders or general medical conditions (Blumer, 1997). Actually, few phenomena in psychiatry or neurology are as enigmatic as catatonia (Lohr & Wisniewski, 1987). This is partly due to the many contradictions surrounding the concept of catatonia. Catatonia has been variously described as a disease (Kahlbaum, 1874), a syndrome (Gelenberg, 1976), a non-specific symptom (Peralta et al.,1997), a subtype of schizophrenia (Kraepelin, 1919; Bleuler, 1950), and as a symptom seen more commonly in mood disorders (Abrams & Taylor, 1976). Strangely enough, it is both caused as well as ameliorated by neuroleptics (Lohr & Wisniewski, 1987).

Historical perspective : In a science beleaguered by a plethora of soft signs and absence of demonstrable physical signs, catatonia appears to be one of the more "medical" appearing conditions in psychiatry. The debate regarding whether catatonia constitutes a disease entity or merely a conglomeration of signs and symptoms was almost nipped in the bud by Kahlbaum, when he christened catatonia as a "symptom complex" (Ahuja, 2000).

Karl Ludwig Kahlbaum first described catatonia (meaning, "to stretch tightly") (Johnson, 1993) in 1868 during a lecture and later published his work in a small monograph, "The Tonic Mental Disorder or the Tension Insanity" (English Translation 1973). To clarify the issue further, his pupil Hecker (1871) wrote that "to consider these symptomatological patterns as diseases would be the same as to consider, in somatic medicine, headache, chest pain, and abdominal pain as disease entities." It is, however, to be noted that after describing catatonia as a "symptom complex", Kahlbaum went beyond the mere conglomeration of signs and symptoms, and actually paid a lot of emphasis on the entire "disease" process and "course" of the illness. In

Kahlbaum's opinion, he had described a distinct entity of equal importance to GPI, but with better prognosis. He stressed that catatonia was often associated with affective illness (mood disorder), occurring in both mania and depression. He also gave examples of other diseases that are associated with catatonia, namely alcoholism, epilepsy, malaria, syphilis, post-partum states, post-traumatic states, and tuberculosis (though the tuberculosis in his cases may well have been due to long periods of debilitation) (Barnes et al, 1986).

His autopsy studies showed abnormalities in the vicinity of the Sylvian fossa, and of the second and third frontal gyrus, i.e. in the area already known as the site of speech formation. Kahlbaum judged this finding noteworthy with regard to the clinical symptoms of mutism and verbigeration so prominent in catatonia, but he justly considered his findings as preliminary (Blumer, 1997). He was convinced that ultimately the etiology of catatonia lay in an "anato-mico-pathological" (sic) change in the brain. He believed that the patients could die of catatonia. The fatal form of catatonia was later termed as "lethal catatonia" by Stauder in 1934. Although believing firmly in the "organic" etiology of catatonia, Kahlbaum also recognized the existence of psychogenic as well epidemic forms of catatonia.

The book on catatonia passed relatively unnoticed until 1896, when Emil Kraepelin incorporated catatonia (as well as hebephrenia) as a subtype of dementia praecox (later termed as schizophrenia by Eugene Bleuler). This amalgamation had a major influence on the future of the term "catatonia", it being regarded as a purely psychiatric condition, commonly associated with schizophrenia. Kraepelin also put forward a psychological interpretation (as did Eugene Bleuler later) of the catatonic phenomena as being due of 'mental blocking". This was a direct antithesis of Kahlbaum's view of catatonia as an organic disease However, surprisingly both Kraepelin and Bleuler recognized that catatonia can occur secondary to a variety of other functional and organic disorders, but this fact has not been given much publicity.

The historical association of catatonia with chronic schizophrenia has often engendered a feeling of therapeutic nihilism. Barnes et al (1986) have therefore recommended that catatonia be called as "Kahlbaum's syndrome" to honour the man who so accurately described the disorder over a century ago. That catatonia is a syndrome with multiple etiologies is brilliantly illustrated in the landmark article by Gelenberg (1976) where he described the catatonic syndrome. In fact this article by Gelenberg, and another by Abrams and Taylor (1976), laid the foundation for the origin of the concept of "Organic catatonic syndrome" (Ahuja & Nehru, 1989). Though Gelenberg (1976) was one of the first to list the several causes of catatonia, since then many more associations have been published. Recently, there has been an emergence of realization that neuroleptic malignant syndrome (NMS) may also be another form of catatonia (Ahuja & Nehru, 1990; White & Robins,1992, Fink et al.,1993).

It has been suggested that the prevalence of catatonia has decreased over time (Mahendra, 1981), at least in the developed countries. Although the prevalence of catatonia is supposed to be much lower in the developed countries as compared to developing ones. Carroll & Spetie (1994) found 1.6% of all patients on a consultation-liaison service met "restrictive criteria" for catatonic disorder. Rosebush et al. (1990) found an even higher figure of 9% (of all admissions), again using strict criteria (requiring four or more signs for diagnosis). Catatonic signs and symptoms are found in 7-14% (Ungvari et al, 1994; Bush et al, 1996a) of all new psychiatric admissions in some other studies. These findings support the theory that catatonia is often under-recognized and under-diagnosed. According to Fink et al. (1993), catatonia appears in many guises and is often difficult to diagnose. The more dramatic features are uncommon, encouraging the belief that catatonia has become rare. Several authors have found that a significant percentage (20-39%) of catatonic patients suffer from organic catatonia (Barnes

et al.,1986; Wilcox, 1986, Bush et al , 1996a). More prior brain injury and physical illness at onset was found in catatonic patients (Wilcox, 1986).

Organic catatonia and classification: In ICD-9, DSM-III and DSM-III-R, catatonia was categorized only as a subtype of schizophrenia, following the tradition of Kraepelin and Bleuler. It has been pointed out that catatonia should be separated from schizophrenia in the psychiatric nomenclature and that organic catatonia should be classified separately (Ahuja & Nehru, 1989; Fink & Taylor, 1991).

DSM-IV includes catatonia as a specifier for mood disorders ("with catatonic features") and as a separate disorder ("catatonic disorder due to general medical condition"), in addition to a subtype of schizophrenia. The diagnostic criteria for "catatonic disorder due to general medical condition" include presence of catatonia (as manifested by motoric immobility, excessive motor activity that is apparently purposeless and not influenced by external stimuli, extreme negativism or mutism, peculiarities of voluntary movement, or echolalia or echopraxia). The criteria require evidence from history, physical examination, or laboratory findings that the disturbance is a direct physiological consequence of a general medical condition, and the disturbance is not better accounted for by another mental disorder (e.g. manic episode). The disturbance should not occur exclusively during the course of delirium.

The "organic catatonic disorder" (F06.1) is described in both versions of ICD-10. CDDG (Clinical Descriptions and Diagnostic Guidelines) and DCR (Diagnostic Criteria for Research), though the DCR criteria are more stringent. In addition to the condition that the general criteria for F06 ("Other mental disorders due to brain damage and dysfunction and to physical disease") are met, the ICD-10 requires the presence of stupor and/or negativism, and catatonic excitement, with rapid alteration between stupor and excitement. The confidence in the diagnosis is increased by the presence of additional catatonic phenomena (e.g. stereotypies, waxy flexibility). The DCR criteria also stress on the need to exclude delirium before diagnosing organic catatonic disorder.

The inclusion of organic catatonia in DSM-IV and ICD-10 will help in better recognition and diagnosis of catatonic symptomatology. As mentioned earlier, catatonic signs are perhaps the only "real" objective signs in psychiatry. Attempts have therefore been made to examine these signs by using rating scales, so as to objectively quantify the severity of the disorder. Early identification of catatonic features is important as the symptoms may require specific treatment with benzodiazepines and/or ECT (Bush et al., 1996b; Rosebush et al., 1990). The various rating scales available for catatonia include Bush-Francis Catatonia Rating Scale and a shorter Bush-Francis Catatonia Screening Instrument (Bush et al., 1996a), Modified Rogers Scale, Lohr and Wisniewski's scale (1987), Northoff Catatonia Scale (Northoff et al , 1999b), and Catatonia Rating Scale (Braunig et al., 2000).

Etiology: Any review of literature on catatonia can not fail to be struck by the diversity of etiologies of catatonia. Acute catatonic syndrome is a condition that can be caused by a variety of metabolic, neurological, psychiatric, and toxic conditions, including neuroleptic malignant syndrome. The neurological causes of catatonia include a variety of lesions that may affect any level of the central nervous system from the brain stem to the cerebral hemispheres (Patterson, 1986)

The first attempts to simulate catatonia experimentally were made in laboratory animals in the beginning of 20th century. DeJong induced catatonia in laboratory in 1945, the first experimental model psychosis to be produced. In his book, "Experimental Catatonia", he described 23 different methods of inducing catatonia in primates, e.g bulbocapnine, mescaline, cannabis, CO, and production of Eck's fistula (anastomosing the inferior vena cava and the portal vein). In DeJong's view, catatonia was similar to epilepsy, a general neurological reaction of the central nervous system to a specific toxin. According to Taylor

(1990), catatonia is a state phenomenon. It results from dysfunction of the brain's motor regulation centres, is not the result of permanent structural brain changes, and regardless of co-occurring symptoms, resolves quickly with treatment.

The neurochemical and neuroanatomical basis of catatonia remains unknown, though several theories exist. It seems that several neurotransmitters are critical in causation of catatonia. Dopaminergic pathways are implicated because of several reasons. Drugs, that are either dopamine depleters or are antidopaminergic, are associated with occurrence of catatonia as well as NMS. Neuroleptics that block dopamine can worsen catatonia and may precipitate NMS, particularly in catatonic patients (White & Robins, 1992; Koch et al., 2000). GABA-ergic systems too have been implicated, keeping in view the effectiveness of benzodiazepines in the treatment of catatonia (including NMS). There is evidence that GABA regulates dopamine activity in the mesolimbic and mesostriatal systems (Fricchione et al., 1983). Benzodiazepines, acting as GABA facilitators, probably alleviate a dopamine blockade in the mesostriatal and mesolimbic systems, thereby stimulating the motor systems and releasing the inhibited catatonic behaviour (Salam et al., 1987). Northoff et al. (1996) have suggested that the different subtypes of catatonia may represent dysfunction of different neurotransmitter systems. The dyskinetic subtype (with high CPK) may signify GABA-ergic dysfunction, with good response to lorazepam. The parkinsonic subtype (with low CPK) may indicate dopaminergic disturbance in the cortico-striatal-thalamo-cortical circuit (the so-called motor loop), with a poor response to lorazepam. The neuroanatomical localization of catatonia can be inferred from the various medical and neurological disorders that cause catatonic signs.

1. Neurological disorders

Organic catatonic disorder occurs due a variety of neurological disorders, mostly affecting the basal ganglia, limbic system, frontal and temporal lobes, and diencephalon. Northoff et al. (1999a) conducted a functional Magnetic Resonance Imaging (fMRI) study in two catatonic patients as compared to healthy controls. They found decreased activation in contralateral motor cortex during a motor task (sequential finger opposition), along with evidence of disturbance of hemispheric localization of activity. However, the patients were diagnosed as catatonic schizophrenia and schizoaffective disorder, and whether the findings were due to the catatonic state or underlying disorder, was not clear. Their earlier SPECT study (Northoff et al, 1997; cited in Northoff et al, 1999a) on a large number of catatonics showed decrease of r-CBF in right fronto-parietal cortex. In another study, Northoff et al. (1999c) found decreased density of GABA-A receptors in the left sensorimotor cortex and right parietal cortex in akinetic catatonia. This finding is important keeping in mind the good response of "akinetic" catatonia to lorazepam and other benzodiazepines, which act on GABA-A receptor.

A. Brain stem, diencephalic, and basal ganglia disorders: One of the recurring themes in the etiology of organic catatonia (as well as akinetic mutism) is the lesions in and around the III ventricle. These include traumatic haemorrhage in III ventricle (Newmann,1955), epidermoid cyst III ventricle (Cairns,1941), thalamotomy and thalamic lesions (Kleist,1960), subthalamic mesencephalic tumours (Neumann, 1996), and neoplasms involving III ventricle (Newmann, 1955). Hoenig and Toakley (1959) reported a patient with recurrent stupor presenting as a "mixed neurotic illness", who was found to have a craniopharyngioma pressing on the brain stem, on autopsy. Globus pallidus lesions, either bilateral (Mettler,1955) or focal (Kleist,1960), parkinsonism (Mettler,1955, Patterson,1986), especially atherosclerotic parkinsonism (Brain,1962), neoplasms and space occupying lesions, and dystonia (Carroll et al.,1994) may also be associated with catatonic features. Chemical lesions of caudate (particularly bilateral and extensive) in animals can produce catatonia (Spiegel & Szekely, 1961). However, if the amygdala or the amygdala-striatal pathway is

destroyed prior to the caudate lesion, catatonia can no longer be produced. It is interesting to note that amygdala, under conditions of extreme fear and arousal, can produce catatonic-like frozen states, including waxy flexibility, presumably by acting on the stratum and SMA (supplementary motor area). Similarly, massive bilateral lesions of caudate and anterior putamen have been known to produce catatonic features (Joseph,1996).

B. Frontal lobe disorders: Catatonia has also been thought to be a manifestation of frontal lobe lesions (Kleist, 1960; Taylor, 1990). Kretschmer supported this view and called the condition as apallic syndrome (Johnson, 1993). Freeman and Watts (1942) described development of waxy flexibility, catatonia, and related symptoms after a gunshot wound, in which the bullet passed completely through the frontal lobes. Similarly, Hillbom (1951) reported frontal lobe injuries due to missile wounds with catatonic features. This view is supported by occurrence of catatonic signs in frontal lobe lesions, like frontal lobe contusion (Rommel,1998), frontal lobe atrophy (Ruff,1980), and neoplasms and space occupying lesions in frontal lobe. Deep medial lesions of the frontal lobe, particularly those involving the SMA (supplementary motor area), may give rise to emotional blunting, decreased speech output or mutism, perseveration, severe apathy, posturing and catatonic-like symptoms (like gegenhalten and waxy flexibility) (Joseph,1996).

C. Parietal lobe disorders: Lesions of parietal lobe too can lead to organic catatonia, like biparietal infarct (Tippin & Dunner,1981 Howards & Low-Beer, 1989), right hemispheric infarct, mostly involving parietal lobe [presenting with only left-sided posturing and left hemineglect (Saver et al.,1993)], arachnoid cyst in right parietal lobe (Wolaczyk et al.,1997), and neoplasms and space occupying lesions in the parietal lobe. As mentioned above, Northoff et al. (1999c) found (right) parietal cortex to be of importance in causation of catatonia.

D. Limbic and temporal lobe disorders: There are several reports of appearance of catatonia in lesions affecting the limbic system (especially the cingulated gyrus and its connections to the frontal lobe) and the temporal lobe. The reported lesions include temporal lobe infarct (Malamud & Boyd, 1929), focal temporal encephalomalacia (Sours, 1962), septum pellucidum tumours (impinging on fornix) (De Morsier, 1968), neoplasms and space occupying lesions, and limbic encephalitis.

E. Generalized CNS disorders: Many times, catatonia can occur due to diffuse brain pathology, such as closed head injury and diffuse brain trauma (Sutter et al.,1959), dementia (Valenstein et al.,1985), diffuse cerebral atrophy, and multiple sclerosis (Mathews,1979; Pine et al.,1995; Mendez,1999).

F. Infections: Catatonic states occur in both the acute and post-encephalitic states of encephalitis lethargica (Tilney & Howe,1920, Cheyette & Cummings,1995, Dekleva & Husain,1995; Johnson & Lucey,1987), and may develop quite independently of any psychotic symptoms (Davison & Bagley,1969). The amyostatic-akinetic form of encephalitis lethargica was the third most common form in the 1918 epidemic, and was known as "epidemic stupor" (Johnson & Lucey,1987). In this disorder, catatonic signs and obsessive symptoms may often co-exist. Recently, several reports have appeared in literature of appearance of catatonic signs in HIV encephalopathy or AIDS dementia (Volkow et al.,1987, Scamvougeras & Rosebush,1992; Carroll et al.,1994; Brough et al.,2000). Catatonic symptomatology can occur in presence of encephalitis both viral and bacterial.

Among the viral encephalitis (Wilson, 1976, Glaser & Pincus 1969); herpes (Raskin & Frank,1974), chicken pox (Blau & Averbuck, 1936), encephalitis lethargica, atypical viral hemorrhagic tick-borne encephalitis (Abczynska & Terminska,1995), and rabies (Prabhakar et al., 1992) have been associated with catatonia, while the bacterial meningo-encephalitis (Orland & Daghestani,1987) have included borrelia burgdorferi encephalitis (Neumarker et al.,1989, Pfister et al.,1993), and acute Chagas' encephalitis (Sevlever et al.,1987). The other infections include hydatid disease (Brage et al.,

1956), post-immunization encephalopathy (Kim & Perlstein, 1970), subacute sclerosing panencephalitis (Koehler & Jakumeit,1976), neurosyphilis and GPI (Kahlbaum,1874; Herman et al.,1942), progressive multifocal leukoencephalopathy (Carroll et al.,1994), and encephalomyelitis disseminate (Boerner,1996). In developing countries, malaria (Durrant,1977), tuberculosis (Kahlbaum,1874), and typhoid (Breakey & Kala,1977; Skrabanek,1977) are important, though under-recognized, causes of organic catatonia.

G. Epilepsy: The association of epilepsy with catatonia has been pointed out repeatedly (Gomez et al.,1982; Lim et al.,1986; Primavera et al.,1994), beginning with the original work of Kahlbaum. Kanemoto et al.(1999) described ictal catatonia as a manifestation of de novo absence status epilepticus, following benzodiazepine withdrawal in a 78 year old patient. In addition to the fixed and glassy-eyed stare, the patient exhibited waxy flexibility, which responded to re-administration of benzodiazepines. Thompson & Greenhouse (1968) and Gomez et al. (1982) have also described catatonic signs in association with absence status (Petit mal status). Lim et al. (1986) reported 3 cases of ictal catatonia, a non-convulsive status epilepticus, which responded dramatically to IV phenytoin. Primavera et al. (1994) stressed on the importance of EEG in detection of seizure activity in patients with acute catatonic syndrome. Organic catatonia has also been described in patients with complex partial epilepsy (Kirubakaran et al.,1987; Wallis et al., 1993), post-ictal state, "spike-wave" stupor, and psychomotor status (Kramer,1977; Shah & Kaplan,1980).

H. Miscellaneous neurological disorders: Cerebrovascular lesions, like cortical venous thrombosis (Gangadhar et al.,1983), subarachnoid haemorrhage (Hanson & Brown, 1973; Thompson,1970; Solomon,1978), subdural hematoma (Woods,1980, Jain et al., 1993), and bacillar aneurysms, AVM of posterior cerebral vessels and ACA aneurysms, may also present with catatonia. Degenerative lesions [e.g. cerebro-macular degeneration, hereditary cerebellar ataxia (Folkerts et al.,1998), and central pontine myelinolysis (Chalela & Kattah, 1999)] and neoplasms (Furtado & Freitas,1946; De Morsier,1968; Steriade et al.,1961) [e.g. pinealoma, and craniopharyngioma (Hoenig & Toakley,1959), and paraneoplastic encephalopathy (Tandon et al.,1988)] may also be associated with catatonic signs. Other disorders which can cause catatonia, include cerebral lupus erythematosus (Kronfol et al.,1977; Fricchione et al.,1990) and non-cerebral lupus erythematosus (Daradkeh & Nasrallah,1987), hydrocephalus (Foltz & Ward, 1956), narcolepsy (Cave,1931), tuberous sclerosis (Critchley & Earl,1932), Wilson's disease (Akil et al.,1991, Davis & Borde,1993), Tay-Sach's disease (Rosebush et al., 1995), and Prader-Willi syndrome (Dhossche & Bouman,1997).

II. Metabolic, Systemic and Endocrine Disorders

Gjessing (1976) conducted metabolic studies in periodic catatonia (a rare disorder described by Kraepelin). The disorder is characterized by episodes of cataleptic states alternating with mental excitement. He showed that during the cataleptic state there was nitrogen retention, which he attributed to the accumulation of a toxic substance. He advocated treatment with large doses of thyroid extract, in an attempt to stimulate catabolism of the toxin. Several other metabolic derangements can cause catatonic symptomatology. These include diabetic keto-acidosis (Katz,1934), homocystinuria (Freeman et al.,1975), hypercalcemia, e.g. secondary to parathyroid adenoma (Cooper & Schapira,1973), acute intermittent porphyria (Freeman & Kolb,1951), hereditary coproporphyria (Offenstadt et al., 1980), hepatic failure (Jaffe,1967), renal failure (Carroll et al.,1994), membranous glomerulonephritis, and hyponatremia (Lee & Schwartz,1997).

Among the endocrine disorders, catatonia has been described with hypothyroidism, hyperthyroidism (Farah & McCall,1995), hyperparathyroidism (Cooper & Schapira,1973), and adrenal carcinoma (Steinberg,1996).

III. Nutritional Deficiency

The nutritional causes of catatonia include pellagra (Gelenberg,1976), nicotinic acid deficiency (Teare et al.,1993), Wernicke's encephalopathy (Teare et al.,1993), and Vitamin B12 deficiency (Catalano et al.,1998).

IV. Drugs and Toxic Agents

Catatonic signs can occur both during intoxication as well as withdrawal from drugs of dependence. Some reported cases include: alcohol intoxication (Bender & Schilder,1933), opiates, e.g. morphine (Enquist et al.,1980), glutethamide withdrawal (Good, 1976; Campbell et al.,1983), amphetamines (Warner & Pierozynski,1977; Chern & Tsai,1993), e.g. methylphenidate (Janowsky et al.,1973), MDMA intoxication (Maxwell et al.,1993; Lee,1994), cannabis (Warner & Pierozynski,1977), mescaline intoxication (Gjessing,1976), LSD intoxication, phencyclidine intoxication (Baldridge & Bessen,1990), benzodiazepine withdrawal (Hauser et al.,1989; Rosebush & Mazurek,1996), and isopropyl alcohol poisoning (Wiernikowski et al.,1997). They have also been reported with disulfiram (Reisberg,1978; Weddington et al.,1980; Fisher, 1989; Schmuecker,1992).

Neuroleptics have long been used for the treatment of catatonic schizophrenia, as well as catatonia occurring in other disorders (like mood disorders). However, neuroleptics have also been reported to cause catatonic symptoms, particularly in high doses (May, 1959; Gelenberg & Mandel,1977; Weinberger & Kelly,1977; Fricchione et al.,1983; Taylor,1990; Bush et al., 1996b; Blumer,1997; Clark & Rickards,1999b). Recently, catatonia has been reported with atypical antipsychotics like risperidone (Bahro et al.,1999), though in a patient with frontal lobotomy. Neuroleptics are also associated with the occurrence of NMS, which has been recently thought to be a subtype of catatonia (Ahuja & Nehru,1989). Patients with present or past catatonic symptoms are particularly vulnerable to NMS (Blumer,1997). Multiple catatonic signs are present in NMS and the severity of NMS predicts the number of catatonic signs (Koch et al.,2000). Lethal (or pernicious or malignant) catatonia is now considered clinically inseparable from NMS (Ahuja & Nehru,1989, Koch et al., 2000), although not all workers agree with this view (Castillo et al.,1989).

Intoxication with pharmacological agents, such as aspirin (Herman et al.,1942), lithium, NMDA antagonists (Lees,1997), baclofen (Pauker & Brown, 1986), and steroids/ACTH (Sullivan & Dickerman,1979, Fink,1991), may cause catatonic features. In other cases, anticonvulsants (Sher et al.,1983), gabapentin withdrawal (Rosebush et al.,1999), antibiotics, e.g. ciprofloxacin (Akhtar & Ahmad, 1993), bupropion (Jackson et al.,1992), allopurinol (Collins et al.,1991) and levodopa (Barbeau,1972) have been incriminated. Toxic agents, like fluorinated hydrocarbons [illuminating gas (Schwab & Barrow,1964)], coal gas (Herman et al.,1942) and CO poisoning, can also cause catatonia. Bulbocapine was used by DeJong (1945) to produce experimental catatonia in cats.

V. Miscellaneous Disorders

Catatonia has also been described to occur secondary to electrocution, burns (Zarr & Nowak,1990), toxic epidermal necrolysis (Lyell syndrome) (Weller et al.,1992), ligation of hepatic artery and portocaval anastomoses, autoimmune disorders (Lichtenstein et al ,1989; Nasierowski & Piotrowski,1997), hepatic amebiasis (Suner-Churlaud et al.,1992), and thrombotic thrombocytopenic purpura (Read,1983).

VI. Psychiatric Disorders

Of course, apart from the organic causes, catatonia can also occur in various psychiatric disorders. These disorders include schizophrenia, mood disorders (Taylor & Abrams,1977), dissociative/conversion disorders (Gelenberg, 1976; Ungvari et al.,1994), OCD (Hermesh, 1989), reactive psychosis, acute and transient psychotic disorder (Banerjee & Sharma,1995; Payee et al.,1999), postpartum/puerperal catatonia (Bach-y-Rita & De Ranieri,1992; Ranzini et al.,1996), PTSD (Shiloh et al.,1995), and under hypnosis (Kornfeld,1985). Catatonic signs are also seen in autistic disorder (pervasive developmental disorder) (Dhossche,1998; Zaw

et al.,1999) and autism spectrum disorders (Wing & Shah,2000). Autistic disorder shares some symptoms with catatonia, namely mutism, echopraxia and echolalia, and stereotypies (Realmuto & August,1991). Little is written of the association of autism and catatonia to clarify the possibility of catatonia as a variant or as a sign of a comorbid condition.

Benegal et al. (1993) have described an idiopathic variety of catatonia, encompassing those patients with catatonia who can not be fitted into the existing classification systems.

Some Management Issues: In the management of catatonia, particularly organic catatonic disorder, it is important to treat at the earliest. Catatonia is associated with excess early mortality when it is unrecognized or inadequately treated (McCall et al.,1995). Catatonic patients were more likely to die of pulmonary embolism, particularly after the second week of appearance of catatonic symptoms and often occurred without warning (McCall et al.,1995). Prompt resolution of the catatonic syndrome with benzodiazepines, barbiturates, or electroconvulsive therapy is the best way to reduce risk of pulmonary embolism, in addition to risks of infection and other medical complications. Also, prompt resolution of catatonic symptoms (like mutism and negativism) allows the clinician to obtain a more detailed information from the patient, necessary for correct diagnosis and management.

Since 1930, amobarbital (amytal) and other barbiturates have been used effectively (Bleckwenn,1930) for producing temporary improvement in catatonic symptoms, allowing for feeding and for interviewing the patient. There were also some reports of temporary relief from catatonic symptoms by the use of stimulants like amphetamines. Traditionally, amytal or pentothal interview was used to differentiate between organic and functional catatonic stupor, and it also helped in ameliorating, even though transiently, the functional catatonic stupor (Gelenberg,1976; McCall et al.,1992). The organic stupor seemed to deteriorate, or not improve, with the intravenous use of these short-acting barbiturates. However, recently lorazepam has been shown to be effective in producing a beneficial response even in catatonia with organic etiology (Fricchione et al.,1983; Rosebush et al.,1990), though not all authors agree (Salam & Kilzieh,1988). The safer benzodiazepines have virtually replaced the barbiturates for the diagnosis and acute management of catatonic syndrome, though rigorous, controlled studies comparing amobarbital with placebo and benzodiazepines in specific patient populations are needed (Kavirajan,1999).

Hawkins et al. (1995) reviewed the recent literature and found that the most commonly reported treatment for catatonia was with benzodiazepines. Lorazepam exhibited the highest frequency of use and a 79% complete response rate. The usual dose is 1-2 mg IV (or IM) and 3-6 mg/day orally. The initial positive response to IV lorazepam may predict the final response of catatonia to oral lorazepam (Bush et al.,1996). Payee et al (1999), in the only study till date of the use of lorazepam in catatonia from our country, also found the usefulness of lorazepam in non-organic catatonia. Although treatment of catatonia should be based on the underlying cause of catatonia, when identifiable (Ahuja,1988), lorazepam appears to be a safe and effective first-line treatment of acute catatonia. In addition, there are now several reports of use of lorazepam and diazepam in the treatment of NMS (Koch et al.,2000). Its use in chronic catatonic states has, however, been doubted (Ungvari et al.,1999). Favourable reports of use of other benzodiazepines, like diazepam (the first report of a benzodiazepine use in catatonia) (McEvoy & Lohr,1984), midazolam (DeLisle,1991, Morena et al.,1994) and oxazepam (Schmider et al.,1999), in retarded catatonia have also been described. Smith & Lebegue (1991), however, did not find clonazepam to be effective in a catatonic patient, where lorazepam was found useful.

Similar to amytal test, zolpidem (7.5-10 mg) has been used for transient amelioration of catatonic symptoms, sometimes when amytal

has failed (Zaw and Bates,1997) or even lorazepam and ECT have failed (Mastain et al., 1995; Thomas et al.,1997). GABA-A agonists enhance GABA activity that may reverse abnormal basal ganglia dopaminergic activity which has been postulated to explain the motor abnormalities of catatonia (Thomas et al.,1997).

In Hawkins et al. (1995) review, antipsychotics demonstrated poor efficacy in treatment of catatonia. In fact, neuroleptics may exacerbate catatonia (Gelenberg & Mandel, 1977; Fricchione et al.,1983; Taylor,1990; Bush et al.,1996b; Clark & Rickards,1999b). It has also been shown that catatonia may be a risk factor for the development of NMS, when neuroleptic drugs are administered (White & Robins,1991). 32% of the catatonic patients in one series (Bush et al.,1996b) did not even have any psychosis, further supporting the need to avoid neuroleptics in catatonic patients. A conservative approach would be to discontinue neuroleptics whenever lethal catatonia or NMS is suspected. IV antidepressants (like clomipramine) have also been found useful by some in the treatment of catatonia associated with mood disorders (Barroso et al.,1999). There are occasional reports of improvement of catatonia with carbamazepine (Rankel & Rankel,1988) and lithium (Pheterson et al.,1985).

The efficacy of ECT in the management of catatonia is impressive (Fink et al.,1993). It is no surprise that both Meduna (for camphor treatment in 1934) and Cerletti and Bini (for ECT treatment in 1938) selected catatonic patients for their initial treatments; catatonic stupor for camphor treatment and catatonic excitement for ECT treatment.(Fink,1984; Bush et al. 1996) Hawkins et al. (1995) found ECT to be efficacious in catatonia (85%) and found it to provide a positive outcome in cases of malignant catatonia. ECT should be considered when rapid resolution is necessary (e.g., malignant catatonia) or when an initial lorazepam trial fails (Hawkins et al.,1995; Bush et al.,1996b; Yeung et al.,1996). A favorable outcome can be expected when ECT is started early, provided the underlying pathological process is treatable and there are no structural lesions in the central nervous system. Several instances of good response of ECT in organic catatonia have been described in the literature. Among theses include cases of catatonia secondary to encephalitis lethargica (Dekleva & Husain,1995), hyperthyroidism (Farah & McCall,1995), typhoid (Breakey & Kala, 1977), lupus erythematosus (Fricchione et al., 1990), hereditary cerebellar ataxia (Folkerts et al.,1998), toxic epidermal necrolysis (Weller et al.,1992) and NMS (Troller & Sachdev,1999).

In conclusion, catatonia is a clinical syndrome associated with a wide variety of psychiatric, medical and neurological disorders. As Gelenberg (1976) said, it "...is not a rare phenomenon; does not automatically imply a 'functional' disorder; and certainly does not always indicate schizophrenia." The physicians and psychiatrists are advised to be aware of firstly, the presence of catatonic signs in a variety of disorders (Carroll et al.,1994) and secondly, the potentially serious organic illnesses which may underlie the catatonic syndrome (Talbot-Stern et al.,2000). Catatonic disorder due to general medical condition (or Organic catatonic disorder) must be first considered in every patient with catatonic signs, particularly in a patient with new onset catatonia. In addition to a careful psychiatric and medical history (including history of drug use), and a detailed neurological, medical and psychiatric examination, diagnostic procedures like CT and MRI should be used, if indicated by the patient's history. Like hypertension or parkinsonism, catatonia discovered on physical examination should spur a medical investigation to uncover the probable cause (Gelenberg, 1976)

REFERENCES

Abczynska,M. & Terminska,K. (1995) Psychopathological symptoms in atypical viral hemorrhagic tick-borne encephalitis. *Psychiatria Polska*, 29, 547-551.

Abrams,R. & Taylor,M.A.(1976)

Catatonia : A prospective clinical study. *Archives of General Psychiatry*, 33, 579-581.

Ahuja,N. (2000) Pioneers in Psychiatry: Karl Ludwig Kahlbaum (1828-1899). *Delhi Psychiatry Society Bulletin*, Submitted and Accepted for Publication.

Ahuja,N. & Nehru,R. (1990) Neuroleptic malignant syndrome : a subtype of lethal catatonia? *Acta Psychiatrica Scandinavica*, 82, 398

Ahuja,N. (1988) Lorazepam : Therapeutic for catatonic schizophrenia? *American Journal of Psychiatry*, 145, 1047.

Ahuja,N. & Nehru,R. (1989) The case for the diagnosis of organic catatonic syndrome. *American Journal of Psychiatry*, 146, 1351-1352.

Akhtar,S. & Ahmad,H. (1993) Ciprofloxacin-induced catatonia. *Journal of Clinical Psychiatry*, 54, 115-116.

Akil,M., Schwartz,J.A., Dutchak,D., Yuzbasiyan-Gurkan,V. & Brewer, G.J. (1991) The psychiatric presentations of Wilson's disease. *Journal of Neuropsychiatry and Clinical Neuroscience*, 3, 377-382.

Bach-y-Rita,G. & De Ranieri,A. (1992) Medicolegal complications of postpartum catatonia. *Western Journal of Medicine*, 156,417-419.

Bahro,M., Kampf,C. & Strnad,J. (1999) Catatonia under medication with risperidone in a 61-year-old patient. *Acta Psychiatrica Scandinavica*, 99, 223-226.

Baldridge,E.B. & Bessen,H.A. (1990) Phencyclidine. *Emergency Medical Clinics of North America*, 8, 541-550.

Banerjee,A. & Sharma,L.M. (1995) Catatonic incidence in acute psychiatric admission. *Indian Journal of Psychiatry*, 37, 35-40.

Barnes,M.P., Saunders,M., Walls,T.J., Saunders,I., & Kirk,C.A. (1986) The syndrome of Karl Ludwig Kahlbaum. *Journal of Neurology Neurosurgery and Psychiatry*, 49, 991-996.

Barbeau,A. (1972) Dopamine and mental function. In : *L-Dopa and Behaviour*, (Ed.) Malitz,S., pp. 9-31, New York : Raven Press.

Barroso,C.A., Jimenez,C.J.P. & Granada,J.O. (1999) Clomipramine in the treatment of catatonia. *Actas Espanolas de Psiquiatria*, 27, 273-276.

Bender,L. & Schilder,P. (1933) Encepholopathia alcoholica (polioencephalitis hemorrhagica superior of Wernicke). *Archives of Neurology and Psychiatry*, 29, 990-1053.

Benegal,V., Hingorani,S. & Khanna,S. (1993) Idiopathic catatonia: Validity of the concept. *Psychopathology*, 26, 41-46.

Blau,A. & Averbuck,S.H.(1936) Catalepsy or cerea flexibilitas in a 3 year old child. *American Journal of Psychiatry*, 92, 967-977.

Bleckwenn,W.J. (1930) Narcosis as therapy in neuropsychiatric conditions. *Journal of American Medical Association*, 95, 1168-1176.

Bleuler,E. (1950) Dementia praecox or the Group of Schizophrenias. (Transl.), Zinkin, J. New York: International University Press.

Blumer,D. (1997) Catatonia and the neuroleptics: psychobiologic significance of remote and recent findings. *Comprehensive Psychiatry*, 38, 193-201.

Brage,D., Pedace,E.A., Napolitano,A.G. (1956) Estupor hippotalimico por hidatide diencefalohipofisaria. *Prensa Med Arg*, 43, 2954-2959.

Brain,R. (1962) Diseases of the Nervous System. London: Clarendon Press.

Braunig,P., Kruger,S., Shugar,G., Hoffler,J. & Borner,I. (2000) The catatonia rating scale I—development, reliability, and use. *Comprehensive Psychiatry*, 41, 147-158.

Breakey,W.R. & Kala,A.K. (1977) Typhoid catatonia responsive to ECT. *British Medical Journal*, 2, 357-359.

Brough,A.S., Stewart,F. & Zackowski,S. (2000) Rapid onset of stupor. *Southern Medical Journal*, 93, 70-71.

Bush,G., Fink,M., Petrides,G., Dowling, F. & Francis,A. (1996a) Catatonia. I. Rating scale and standardized examination. *Acta Psychiatrica Scandinavica*, 93, 129-136.

Bush,G., Fink,M., Petrides,G., Dowling, F. & Francis,A. (1996b) Catatonia. II. Treatment with lorazepam and electroconvulsive therapy. *Acta Psychiatrica Scandinavica*, 93, 137-143.

Cairns,H., Oldfield,R.C., Pennybacker, J.B., & Whitteridge, D. (1941) Akinetic mutism with an epidermoid cyst of the third ventricle. *Brain*, 64, 273-290.

Campbell,R., Schaffer,C.B. & Tupin,J. (1983) Catatonia associated with glutethimide withdrawal. *Journal of Clinical Psychiatry*, 44, 32-33.

Carroll,B.T., Anfinson,T.J., Kennedy, J.C., Yendrek,R., Boutros,M. & Bilon,A. (1994) Catatonic disorder due to general medical conditions. *Journal of Neuropsychiatry and Clinical Neuroscience*, 6, 122-133.

Carroll,B.T. & Spetie,L.(1994) Catatonia on the consultation-liaison service : a replication study. *Interational Journal of Psychiatry in Medicine*, 24, 329-337.

Castillo,E., Rubin,R.T. & Holsboer-Traschler,E. (1989) Clinical differentiation between lethal catatonia and neuroleptic malignant syndrome. *American Journal of Psychiatry*, 146, 324-328.

Catalano,G., Catalano,M.C., Rosenberg, E.I., Embi,P.J. & Embi,C.S. (1998) Catatonia. Another neuropsychiatric presentation of vitamin B12 deficiency? *Psychosomatics*, 39, 456-460.

Cave,H.A. (1931) Narcolepsy. *Archives of Neurology and Psychiatry*, 26, 50-101.

Chalela,J. & Kattah,J. (1999) Catatonia due to central pontine and extrapontine myelinolysis: case report. *Journal of Neurology, Neurosurgery and Psychiatry*, 67, 692-693.

Chern,C.H. & Tsai,W.J. (1993) Acute amphetamine intoxication with catatonia: a case report. *Chung Hua I Hsueh Tsa Chih (Taipei)*, 51, 322-327.

Cheyette,S.R. & Cummings,J.L. (1995) Encephalitis lethargica : Lessons for contemporary neuropsychiatry. *Journal of Neuropsychiatry and Clinical Neuroscience*, 7, 125-134.

Clark,T. & Rickards,H. (1999a) Catatonia. 1 : History and clinical features. *Hospital Medicine*, 60, 740-742.

Clark,T. & Rickards,H. (1999b) Catatonia. 2 : Diagnosis, management and prognosis. *Hospital Medicine* 60, 812-814.

Collins,C.E., Thomas,D.J. & Gumpel, J.M. (1991) Catatonia in the allopurinol hypersensitivity syndrome. *British Medical Journal*, 302, 970.

Cooper,A.F. & Schapira,K. (1973) Case report: depression, catatonic stupor, and EEG changes in hyperparathyroidism. *Psychological*

Medicine, 3, 509-515.

Critchley,M. & Earl,C.J.C.(1932) Tuberose sclerosis and allied conditions. *Brain*, 55, 311-346.

Daradkeh,T.K. & Nasrallah,N.S. (1987) Lupus catatonia : a case report. *Pharmatherapeutica*, 5, 142-144.

Davis,E.J. & Borde,M. (1993) Wilson's disease and catatonia. *British Journal of Psychiatry*, 162, 256-259.

Davison,K. & Bagley,C.R. (1969) Schizophrenia-like psychoses associated with organic disease of the central nervous system. In : *Current Problems in Neuropsychiatry*, (Ed.), Herrington R.N., Ashford : Headley.

DeJong,H.H. (1945) Experimental Catatonia, Baltimore : Williams and Wilkins.

Dekleva,K.B. & Husain,M.M. (1995) Sporadic encephalitis lethargica : A case treated successfully with ECT. *Journal of Neuropsychiatry and Clinical Neuroscience*, 7, 237-239.

DeLisle,J.D. (1991) Catatonia unexpectedly reversed by midazolam. *American Journal of Psychiatry*, 148, 809.

Dhossche,D.M. & Bouman,N.H. (1997) Catatonia in an adolescent with Prader-Willi Syndrome. *Annals of Clinical Psychiatry*, 9, 247-253.

Dhossche,D. (1998) Brief report: catatonia in autistic disorders. *Journal of Autism and Developmental Disorders*, 28, 329-331.

Durrant,W. (1977) Catatonia after malaria. *British Medical Journal*, 2, 893.

Enquist,A., Chraemmer,J.B. & Andersen,H.B. (1980) Epidural morphine-induced catatonia. *Lancet*, 2, 984.

Farah,A. & McCall,W.V. (1995) ECT administration to a hyperthyroid patient. *Convulsive Therapy*, 11, 126-128.

Fink,M. (1984) Meduna and the origins of convulsive therapy. *American Journal of Psychiatry*, 141, 1034-1041.

Fink,M. & Taylor,M.A. (1991) Catatonia: A Separate Category in DSM-IV? *Integrative Psychiatry*, 7, 2-10.

Fink,M. (1991) Steroid-induced catatonia. *British Journal of Psychiatry*, 159, 445.

Fink,M., Bush,G. & Francis,A. (1993) Catatonia : A treatable disorder, occasionally recognized. *Directions in Psychiatry*, 13, 3, 1-8.

Fisher,C.M. (1989) Catatonia due to disulfiram toxicity. *Archives of Neurology*, 46, 798-804.

Folkerts,H.W., Stadtland,C. & Reker,T. (1998) ECT for organic catatonia due to hereditary cerebellar ataxia. *Journal of ECT*, 14, 53-55.

Foltz,E.L. & Ward,A.A. (1956) Communication hydrocephalus from sub-arachnoid bleeding. *Journal of Neurosurgery*, 13, 546-566.

Freeman,W & Watts,J.W. (1942) Psychosurgery, Springfield. IL . Charles C Thomas.

Freeman,J.G. & Kolb,L.C. (1951) Acute intermittent porphyria Associated psychiatric symptoms treated by electroshock. *Proceedings of Mayo Clinic*, 26, 401-405.

Freeman,J.M., Finkelstein,J.D. & Mudd,

S.H. (1975) Folate-responsive homocystinuria and schizophrenia. *New England Journal of Medicine*, 291, 491-496.

Fricchione,G.L., Cassem,N.H., Hooberman,D. & Hobson,D.(1983) Intravenous lorazepam in neuroleptic-induced catatonia. *Journal of Clinical Psychopharmacology*, 3, 338-342.

Fricchione,G.L., Kaufman,L.D., Gruber, B.L. & Fink,M. (1990) Electroconvulsive therapy and cyclophosphamide in combination for severe neuropsychiatric lupus with catatonia. *American Journal of Medicine*, 88, 442-443.

Furtado,D. & Freitas,A. (1946) Catatonie par compression diencephalique. *Revista de Neurologia*, 78, 499-500.

Gangadhar,B.N., Keshavan,M.S., Goswami,U. & Rao,T.V. (1983) Cortical venous thrombosis presenting as catatonia : A clinico-pathological report. *Journal of Clinical Psychiatry*, 4, 109-110.

Gelenberg,A.J. (1976) The catatonic syndrome. *Lancet*, i, 1339-1341.

Gelenberg,A.J. & Mandel,M.R. (1977) Catatonic reactions to high potency Neuroleptic drugs. *Archives of General Psychiatry*, 34, 947-950.

Glaser,G.H. & Pincus,J.H. (1969) Limbic encephalitis. *Journal of Nervous and Mental Disease*, 149, 59-67.

Good,M.I. (1976) Catatonic-like symptomatology and withdrawal dyskinesias. *American Journal of Psychiatry*, 133, 1454-1456.

Gomez,F.A., Comstock,B.S. & Rosario, A. (1982) Organic versus functional etiology in catatonia: Case report. *Journal of Clinical Psychiatry*, 43, 200-201.

Gjessing,R.R. (1976) Contribution to the Somatology of Periodic Catatonias. Oxford: Pergamon Press.

Hanson,G.D. & Brown,M.J. (1973) Waxy flexibility in a post-partum woman *Psychiatric Quarterly*, 47, 95-103.

Hauser,P., Devinsky,O., De Bellis,M., Theodore,W.H. & Post,R.M. (1989) Benzodiazepine withdrawal delirium with catatonic features. Occurrence in patients with partial seizure disorders. *Archives of Neurology*, 46, 696-699.

Hawkins,J.M., Archer,K.J., Strakowski, S.M. & Keck,P.E. (1995) Somatic treatment of catatonia. *International Journal of Psychiatry in Medicine*, 25, 345-369.

Hecker,E. (1871) Die Hebephrenie. *Archiv fur Pathologische Anatomie und Physiologie und fur Klinische Medizin*, 25, 202.

Herman,M., Harpham,D. & Rosenblum, M. (1942) Nonschizophrenic catatonic states. *New York State Journal of Medicine*, 42, 624-627.

Hermesh,H., Hoffnung,R.A., Aizenberg, D., Molcho,A. & Munitz,H. (1989) Catatonic signs in severe obsessive compulsive disorder. *Journal of Clinical Psychiatry*, 50, 303-305

Hillbom,E. (1951) Schizophrenia-like psychoses after brain trauma *Acta Psychiatrica*, 60, 36-47

Hoenig,J & Toakley,J.G. (1959) The diagnosis of stupor. *Psychiatry et Neurology*, 137, 128-144.

Howards,R.J. & Low-Beer,T.S. (1989) Catatonia following biparietal infarction with spontaneous recovery. *Postgraduate Medical Journal*, 65, 316-317.

Jackson,C.W., Head,L.A. & Kellner,C.H. (1992) Catatonia associated with bupropion treatment. *Journal of Clinical Psychiatry*, 53, 210.

Jaffe,N. (1967) Catatonia and hepatic dysfunction. *Diseases of Nervous System*, 28, 606-608.

Jain,A., Sharma,A.K. & Kar,P. (1993) Subdural hematoma presenting as catatonia. *Journal of Association of Physicians in India*, 41, 615.

Janowsky,D.S., El-Yousef,M.K., Davis, J.M. & Sekerke,H.J. (1973) Provocation of schizophrenic symptoms by intravenous adminstration of methylphenidate. *Archives of General Psychiatry*, 28, 185-191.

Johnson,J. & Lucey,P.A. (1987) Encephalitis lethargica, a contemporary cause of catatonic stupor: A report of two cases. *British Journal of Psychiatry*, 151, 550-552.

Johnson,J. (1993) Catatonia: The Tension Insanity. *British Journal of Psychiatry*, 162, 733-738.

Joseph,R. (1996) Neuropsychiatry, Neuropsychology, and Clinical Neuroscience, Baltimore : Williams and Wilkins.

Kahlbaum,K.L. (1874) Die Katatonie Oder das Spannungsirresein. (Transl.) Levij, Y & Pridan, T, Baltimore : John Hopkins University Press, 1973.

Kanemoto,K., Miyamoto,T. & Abe,R. (1999) Ictal catatonia as a manifestation of de novo absence status epilepticus following benzodiazepine withdrawal. *Seizure*, 8, 364-366.

Katz,S.E. (1934) A catatonic syndrome associated with diabetes mellitus. *Archives of Neurology and Psychiatry*, 31, 880-881.

Kavirajan,H. (1999) The amobarbital interview revisited: a review of the literature since 1966. *Harvard Review of Psychiatry*, 7, 153-165.

Kim,C.M. & Perlstein,M.A. (1970) Encephalitis with catatonic schizophrenic symptoms. *Illinois Medical Journal*, 138, 503-507.

Kirubakaran,V., Sen,S. & Wilkinson, C.B. (1987) Catatonic stupor: unusual manifestation of temporal lobe epilepsy. *Psychiatric Journal of University of Ottawa*, 12, 244-246.

Kleist,K. (1960) Schizophrenic symptoms and cerebral pathology. *Journal of Mental Science*, 106, 246-255.

Koch,M., Chandragiri,S., Rizvi,S., Petrides,G. & Francis,A. (2000) Catatonic signs in neuroleptic malignant syndrome. *Comprehensive Psychiatry*, 41, 73-75.

Koehler,K. & Jakumeit,U. (1976) Subacute sclerosing panencephalitis presenting as Leonhard's speech-prompt catatonia. *British Journal of Psychiatry*, 129, 29-31.

Kornfeld,A.D. (1985) A spontaneous catatonic-like reaction associated with hypnotic regression: a brief case report. *American Journal of Clinical Hypnosis*, 27, 180-182.

Kraepelin,E. (1919) Dementia praecox and paraphrenia, (Transl.), Barclay, R.M., Edinburg : Livingstone.

Kramer,M.S. (1977) Menstrual epileptoid psychosis in an adolescent girl. *American Journal of Diseases of Children*, 68, 1271-1279.

Kronfol,Z., Schlesser,M. & Tsuang,M.T. (1977) Catatonia and systemic lupus erythematosus. *Diseases of Nervous System*, 38, 729-731.

Lee,T.H., Chen,S.S., Su,S.L. & Yang, S.S. (1992) Baclofen intoxication : report of four cases and review of the literature. *Clinical Neuropharmacology*, 15, 56-62.

Lee,J.W. (1994) Catatonic stupor after "ecstasy". *British Medical Journal*, 308, 717-718.

Lee,J.W. & Schwartz,D.L. (1997) Catatonia associated with hyponatremia. *Neuropsychiatry Neuropsychology Behavavioural Neurology*, 10, 63-64.

Lees,K.R. (1997) Cerestat and other NMDA antagonists in ischemic stroke. *Neurology*, 49 (Suppl. 4), S66-99.

Lichtenstein,A., Calish,I., Oliveira,R.M., Miguel,F.E.C. & Rocha,A.S. (1989) Catatonic syndrome caused by autoimmune disease: spontaneous remission. Revista Do Hospital Das Clinicas Faculdade de Medicinia de Sao Paulo, 44, 312-315.

Lim,J., Yagnik,P., Schraeder,P. & Wheeler,S. (1986) Ictal catatonia as a manifestation of nonconvulsive status epilepticus. *Journal of Neurology, Neurosurgery and Psychiatry*, 49, 833-836.

Lohr,J.B. & Wisniewski,A.A. (1987) Movement Disorders : A Neuropsychiatric Approach. New York : Guilford.

Mahendra,B. (1981) Where have all the catatonics gone? *Psychological Medicine*, 11, 669-671

Malamud,N. & Boyd,D.A. (1929) Sudden brain death in schizophrenia with extensive lesions in the cerebral cortex. *Archives of Neurology and Psychiatry*, 41, 252-364.

Mastain,B., Valva,G., Guerouaou,D., Pommery,J. & Thomas,P. (1995) Dramatic improvement of catatonia with zolpidem. *Revista de Neurologia (Paris)*, 151, 52-56.

Mathews,W.B. (1979) Multiple sclerosis presenting with acute remitting psychiatric symptoms. *Journal of Neurology, Neurosurgery and Psychiatry*, 42, 859-863.

Maxwell,D.L., Polkey,M.I. & Henry,J.A. (1993) Hyponatraemia and catatonic stupor after taking "ecstasy". *British Medical Journal*, 307, 1399.

May,R.H. (1959) Catatonic like states following phenothiazine therapy. *American Journal of Psychiatry*, 115, 1119-1120.

McCall,W.V., Shelp,F.E. & McDonald, W.M. (1992) Controlled investigation of the amobarbital interview for catatonic mutism. *American Journal of Psychiatry*, 149, 202-206.

McCall,W.V., Mann,S.C., Shelp,F.E. & Caroff,S.N. (1995) Fatal pulmonary embolism in the catatonic syndrome : two case reports and a literature review. *Journal of Clinical Psychiatry*, 56, 21-25.

McEvoy,J.P. & Lohr,J.B. (1984) Diazepam for catatonia. *American Journal of Psychiatry*, 141, 284-285.

Mendez,M.F. (1999) Multiple sclerosis presenting as catatonia. *International Journal of Psychiatry in Medicine*, 29, 435-441

Morena,G., Sunderland,B. & Billig,N. (1994) Midazolam and the treatment of catatonia in major depression in an older adult. *Psychosomatics*, 35, 392-395.

Mettler,F.A. (1955) Perceptual capacity, function of the corpus striatum and schizophenia. *Psychiatric Quarterly*, 29, 89-111.

Nasierowski,T. & Piotrowski,T. (1997) Mental disorders in the course of scleroderma:

case reports. *Psychiatria Polska*, 31, 215-221.

Neumarker,K.J., Dudeck,U. & Plaza,P. (1989) Borrelia encephalitis and catatonia in adolescence. *Nervenarzt*, 60, 115-119.

Neuman,E., Rancurel,G., Lecrubier,Y., Fohanno,D. & Boller,F.(1996) Schizophreniform catatonia on 6 cases secondary to hydrocephalus with subthalamic mesencephalic tumor associated with hypodopaminergia. *Neuropsychobiology*, 34, 76-81.

Newmann,M.A. (1955) Periventricular diffuse pinealoma. *Journal of Nervous and Mental Disease*, 121, 193-204.

Northoff,G., Wenke,J. & Pflug,B. (1996) Increase of serum creatine phosphokinase in catatonia : An investigation in 32 acute catatonic patients. *Psychological Medicine*, 26, 547-553.

Northoff,G., Braus,D.F., Sartorius,A., Khoram-Sefat,D., Russ,M., Eckert,J., Herrig, M., Leschinger, A., Bogerts, B. & Henn, F.A. (1999a) Reduced activation and altered laterality in two neuroleptic-naive catatonic patients during a motor task in functional MRI. *Psychological Medicine*, 29, 997-1002.

Northoff,G., Koch,A., Wenke,J., Eckert, J., Boker,H., Pflug,B. & Bogerts,B. (1999b) Catatonia as a psychomotor syndrome: A rating scale and extrapyramidal motor symptoms. *Movement Disorders*, 14, 404-416.

Northoff,G., Steinke,R., Czcervenka,C., Krause,R., Ulrich,S., Danos,P., Kropf,D., Otto, H. & Bogerts,B. (1999c) Decreased density of GABA-A receptors in the left sensorimotor cortex in akinetic catatonia: Investigations of in-vivo benzodiazepine receptor binding. *Journal of Neurology, Neurosurgery and Psychiatry*, 67, 445-450.

Offenstadt,G., Bienvenu,M.P., Hericord, P., Tawil,I.S. & Amstutz,P. (1980) Severe catatonia, schizophrenia and hereditary coproporphyria. A case report. *Seminars in Hospital*, 56, 1727-1730.

Orland,R.M. & Daghestani,A.N. (1987) A case of catatonia induced by bacterial meningoencephalitis. *Journal of Clinical Psychiatry*, 48, 489-490.

Patterson,J.F. (1986) Akinetic parkinsonism and the catatonic syndrome : an overview. *South Medical Journal*, 79, 682-685.

Pauker,S.L. & Brown,R. (1986) Baclofen-induced catatonia. *Journal of Clinical Psychopharmacology*, 6, 387-388.

Payee,H., Chandrasekaran,R., Raju, G.V.L. (1999) Catatonic syndrome: Treatment response to lorazepam. *Indian Journal of Psychiatry*, 41, 49-53.

Peralta,V., Cuesta,M.J., Serrano,J.F. & Mata,I. (1997) The Kahlbaum syndrome: a study of its clinical validity, nosological status, and relationship with schizophrenia and mood disorder. *Comprehensive Psychiatry*, 38, 61-67.

Pfister,H.W., Preac-Mursic,V., Wilske, B., Rieder,G., Forderreuther,S., Schmidt,S. & Kapfhammer,H.P. (1993) Catatonic syndrome in acute severe encephalitis due to Borrelia burgdorferi infection. *Neurology*, 43, 433-435.

Pheterson,A.D., Estroff,T.W. & Sweeney,D.R. (1985) Severe prolonged catatonia with associated flushing reaction responsive to lithium carbonate. *Journal of American Academy of Child & Adolescent Psychiatry*, 24: 235-237.

Pine,D.S., Douglas,C.J., Charles,E., Davies,M. & Kahn,D. (1995) Patients with multiple sclerosis presenting to psychiatric hospitals. *Journal of Clinical Psychiatry*, 56, 297-306.

Prabhakar,S., Malhotra,R.M. & Goel, D.S. (1992) Rabies presenting as catatonic stupor. *Journal of Association of Physicians of India*, 40, 272-273.

Primavera,A., Fonti,A., Novello,P., Roccatagliata,G. & Cocito,L. (1994) Epileptic seizures in patients with acute catatonic syndrome. *Journal of Neurology, Neurosurgery and Psychiatry*, 57, 1419-1422.

Rankel,H.W. & Rankel,L.E. (1988) Carbamazepine in the treatment of catatonia. *American Journal of Psychiatry*, 145, 361-362.

Ranzini,A.C., Vinekar,A.S., Houlihan, C., Scully,J., Cho,S.C. & Vintzileos,A. (1996) Puerperal psychosis mimicking eclampsia. *Journal of Maternal and Fetal Medicine*, 5, 36-38.

Raskin,D.E. & Frank,S.W. (1974) Herpes encephalitis with catatonic stupor. *Archives of General Psychiatry*, 31, 544-546.

Read,S.L. (1983) Catatonia in thrombotic thrombocytopenic purpura: case report. *Journal of Clinical Psychiatry*, 44, 343-344.

Realmuto,G.M. & August,G.J. (1991) Catatonia in autistic disorder : a sign of comorbidity or variable expression? *Journal of Autism and Developmental Disorders*, 21, 517-528.

Reisberg,B. (1978) Catatonia associated with disulfiram therapy. *Journal of Nervous and Mental Disease*, 166, 607-609

Rommel,O., Tegenthoff,M., Widdig,W., Braunig,P. & Malin,J.P. (1998) Organic catatonia following frontal lobe injury: response to clozapine. *Journal of Neuropsychiatry and Clinical Neuroscience*, 10, 237-238.

Rosebush,P.I., Hildebrand,A.M., Furlong, B.G. & Mazurek,M.F. (1990) Catatonic syndrome in a general psychiatric inpatient population: Frequency, clinical presentation, and response to lorazepam. *Journal of Clinical Psychiatry*, 51, 357-362.

Rosebush,P.I., MacQueen,G.M., Clarke, J.T., Callahan,J.W., Strasberg,P.M. & Mazurek,M.F. (1995) Late-onset Tay-Sachs disease presenting as catatonic schizophrenia: diagnostic and treatment issues. *Journal of Clinical Psychiatry*, 56, 347-353.

Rosebush,P.I. & Mazurek,M.F. (1996) Catatonia after benzodiazepine withdrawal. *Journal of Clinical Psychopharmacology*, 16, 315-319.

Rosebush,P.I., MacQueen,G.M. & Mazurek,M.F. (1999) Catatonia following gabapentin withdrawal. *Journal of Clinical Psychopharmacology*, 19, 188-189.

Ruff,R.L. (1980) Catatonia with frontal lobe atrophy. *Journal of Neurology, Neurosurgery and Psychiatry*, 43, 185-187.

Salam,S.A., Pillai,A.K. & Beresford,T.P. (1987) Lorazepam for psychogenic catatonia. *American Journal of Psychiatry*, 144, 1082-1083.

Salam,S.A. & Kilzieh,N. (1988) Lorazepam treatment of psychogenic catatonia: An update. *Journal of Clinical Psychiatry*, 49, (12 Suppl.), 16-21.

Saver,J.L., Greenstein,P., Ronthal,M. & Mesulam,M.M. (1993) Asymmetric catalepsy after right hemisphere stroke. *Movement Disorders*, 8, 69-73.

Scamvougeras,A. & Rosebush,P.I. (1992) AIDS-related psychosis with catatonia responding to low-dose lorazepam. *Journal of Clinical Psychiatry*, 53, 414-415.

Schmider,J., Standhart,H., Deuschle, M., Drancoli,J. & Heuser,I. (1999) A double-

blind comparison of lorazepam and oxazepam in psychomotor retardation and mutism. *Biological Psychiatry*, 46, 437-441.

Schmuecker,J.D., Meloy,J.R. & Williams,D.J. (1992) Disulfiram toxicity and catatonia in a forensic outpatient. *American Journal of Psychiatry*, 49, 1275-1276.

Schwab,J.J. & Barrow,M.V. (1964) A reaction to organic fluorides simulating classical catatonia. *American Journal of Psychiatry*, 120, 1196-1197.

Sevlever,G., Taratuto,A.H., de las Carreras,M.C., Leiguarda,R. & Nogues,M. (1987) Catatonia secondary to acute Chagas' encephalitis. *Journal of Neurology, Neurosurgery and Psychiatry*, 50, 1244.

Shiloh,R., Schwartz,B., Weizman,A. & Radwan,M. (1995) Catatonia as an unusual presentation of posttraumatic stress disorder. *Psychopathology*, 28, 285-290.

Skrabanek,P. (1977) Typhoid catatonia and ECT. *British Medical Journal*, 2, 892-893.

Shah,P. & Kaplan,S.L. (1980) Catatonic symptoms in a child with epilepsy. *American Journal of Psychiatry*, 137, 738-739.

Sher,A., Andersen,J.M. & Bhatia,S.C. (1983) Primidone-induced catatonic schizophrenia. *Drug Intelligence in Clinical Pharmacology*, 17, 551-552.

Smith,M & Lebegue,B.(1991) Lorazepam in the treatment of catatonia. *American Journal of Psychiatry*, 148, 1265.

Snyder,S., Prenzlauer,S., Maruyama,N. & Rose,D.N. (1992) Catatonia in a patient with AIDS-related dementia. *Journal of Clinical Psychiatry*, 53, 414.

Solomon,J.G. (1978) Distinguishing catatonia. A case report. *Psychosomatics*, 19, 299-307.

Sours,J.A. (1962) Akinetic mutism simulating catatonic schizophrenia. *American Journal of Psychiatry*, 119, 451-455.

Spiegel,E.A. & Szekely,E.G. (1961) Prolonged stimulation of the head of the caudate nucleus. *Archives of Neurology*, 4, 67-77.

Stauder,K:H. (1934) Die Todliche Katatonie. *Arch Psychiatr Nervenkr*, 102, 614-634.

Steinberg,P. (1996) Adrenal carcinoma and hypertension presenting with catatonic stupor. *Australian and New Zealand Journal of Psychiatry*, 30, 146-149.

Steriade,M., Botez,M.I. & Petrovoco,I. (1961) On certain dissociations of consciousness levels within the syndrome of akinetic mutism. *Psychiatr Neurol*, 141, 38-58.

Sternbach,H. & Yager,J.(1981) Catatonia in the presence of midbrain and brainstem abnormalities. *Journal of Clinical Psychiatry*, 42, 352-353.

Sullivan,B.J. & Dickerman,J.D. (1979) Steroid associated catatonia : Report of a case. *Pediatrics*, 63, 677-679.

Suner-Churlaud,A., Favre,J.D., Vallet, D., Payen,A. & Dubelle,P. (1992) Catatonia and hepatic amebiasis. A case report. *Annals of Medical Psychology (Paris)*, 150, 407-413.

Sutter,J.M., Bardinat,C., Pheline,C. & Coudray,J.P. (1959) La catatonie postraumatique ses rapports avec les 'etats de decerebration' et les 'comas prolongees'. *Revista de Neurologia*, 101, 524-535.

Talbot-Stern,J.K., Green,T. & Royle,T.J. (2000) Psychiatric manifestations of systemic illness. *Emergency Medical Clinics of North America*, 18, 199-209.

Tandon,R., Walden,M. & Falcon,S. (1988) Catatonia as a manifestation of paraneoplastic encephalopathy. *Journal of Clinical Psychiatry*, 49, 121-122.

Taylor,M.A. & Abrams,R. (1977) Catatonia : Prevalence and importance in the manic phase of manic-depressive illness. *Archives of General Psychiatry*, 34, 1223-1225.

Taylor,M.A. (1990) Catatonia: A review of the behavioral neurologic syndrome. *Neuropsychiatry Neuropsychology and Behaviour Neurology*, 3, 48-72.

Teare,J.P., Hyams,G. & Pollock,S. (1993) Acute encephalopathy due to coexistent nicotinic acid and thiamine deficiency. *British Journal of Clinical Practice*, 47, 343-344.

Thomas,P., Rascle,C., Mastain,B., Maron,M. & Vaiva,G. (1997) Test for catatonia with zolpidem. *Lancet*, 349, 702.

Thompson,G.H. & Greenhouse,A.H. (1968) Petit mal status in adults. *Annals of Internal Medicine*, 68, 1271-1279.

Thompson,G.H. (1970) Cerebral lesions simulating schizophrenia : Three case reports. *Biological Psychiatry*, 2, 59-64.

Tiiney,F. & Howe,H.S. (1920) Epidemic Encephalitis. New York : Paul B. Hoebner.

Tippin,J. & Dunner,F.J. (1981) Biparietal infarctions in a patient with catatonia. *American Journal of Psychiatry*, 138, 1386-1387.

Troller,J.N. & Sachdev,P.S. (1999) Electroconvulsive treatment of neuroleptic malignant syndrome : A review and report of cases. *Australian and New Zealand Journal of Psychiatry*, 33, 650-659.

Ungvari,G.S., Leung,C.M., Wong,M.K. & Lau,J. (1994) Benzodiazepines in the treatment of catatonic syndrome. *Acta Psychiatrica Scandinavica*, 89, 285-288.

Ungvari,G.S., Chiu,H.F., Chow,L.Y., Lau,B.S. & Tang,W.K. (1999) Lorazepam for chronic catatonia: a randomized, double-blind, placebo-controlled cross-over study. *Psychopharmacology (Berlin)*, 142, 393-398.

Valenstein,M., Maltbie,A. & Kaplan,P. (1985) Catatonia in the emergency department. *Annals of Emergency Medicine*, 14, 359-361.

Volkow,N.D., Harper,A., Munnisteri,D. & Clother,J. (1987) AIDS and catatonia. *Journal of Neurology, Neurosurgery and Psychiatry*, 50, 104.

Walls,M.J., Bowers,T.C., Dilsaver,S.C. & Swann,A.C. (1993) Catatonia associated with depression secondary to complex partial epilepsy. *Journal of Clinical Psychiatry*, 54, 73.

Warner,A.M. & Pierozynski,G. (1977) Pseudocatatonia associated with abuse of amphetamine and cannabis. *Postgraduate Medicine*, 61 275-277.

Weddington,W.W. Jr., Marks,R.C. & Verghese,J.P. (1980) Disulfiram encephalopathy as a cause of the catatonia syndrome. *American Journal of Psychiatry*, 137, 1217-1219.

Weinberger,D.R. & Kelly,M.J. (1977) Catatonia and malignant syndrome : A possible complication of neuroleptic administration. *Journal of Nervous and Mental Disease*, 165, 263-268.

Weller,M., Kornhuber,J. & Beckmann,

H. (1992) Electroconvulsive therapy in treatment of acute life threatening catatonia in toxic epidermal necrolysis. *Nervenarzt*, 63, 308-310.

Wiernikowski,A., Piekoszewski,W., Krzyzanowska-Kierepka,E. & Gomulka,E. (1997) Acute oral poisoning with isopropyl alcohol in alcoholics. *Przegl Lek*, 54, 459-463.

White,D.A.C. & Robins,A.H. (1991) Catatonia: Harbinger of the neuroleptic malignant syndrome. *British Journal of Psychiatry*, 158, 419-431.

Wilcox,J.A. (1986) Organic factors in catatonia. *British Journal of Psychiatry*, 149, 782-784.

Wing,L. & Shah,A. (2000) Catatonia in autism spectrum disorders. *British Journal of Psychiatry*, 174, 357-362.

Wison,L.G. (1976) Viral encephalopathy mimicking functional psychosis. *American Journal of Psychiatry*, 133, 165-170.

Wolaczyk,T., Komender,J. & Brzozowska,A. (1997) Catatonic syndrome preceded by symptoms of anorexia nervosa in a 14-year-old boy with arachnoid cyst. *European Child and Adolescent Psychiatry*, 6, 166-169.

Woods,S.W. (1980) Catatonia in a patient with subdural hematoma. *American Journal of Psychiatry*, 137, 983-984.

Yeung,P.P., Milstein,R.M., Daniels,D.C. & Bowers,M.B.Jr. (1996) ECT for lorazepam-refractory catatonia. *Convulsive Therapy*, 12,31-35.

Zarr,M.L. & Nowak,T. (1990) Catatonia and burns. *Burns*, 16, 133-134.

Zaw,F.K. & Bates,G.D. (1997) Replication of zolpidem test for catatonia in an adolescent. *Lancet*, 349, 1914.

Zaw,F.K., Bates,G.D., Murali,V. & Bentham,P. (1999) Catatonia, autism, and ECT. *Developmental Medicine and Child Neurology*, 41, 843-845

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