Stupor is an unusual but striking phenomenon generally recognized but difficult to define precisely. This condition is clinically characterized by the basic triad of akinesia, mutism and clear consciousness (Joyston-Bechal 1966). However, the degree of akinesia and mutism may vary. The patient characteristically assimilates the external stimuli but the output is curtailed.

There are a number of causes for stupor and catatonic schizophrenia is one of the important ones (Gelenberg 1976). Neurological disorders (Plum 1972; Penn et al 1972), metabolic conditions including psychotomimetic agents such as mescaline and phencyclidine, ethyl alcohol and amphetamine (Gatewood et al 1975, Jaffe 1967), and pharmacological agents include aspirin, ACTH, and neuroleptics (Weinberger and Kelly 1978) produce a catatonic state. Therefore, catatonia is not always due to a functional psychiatric disorder and thus does not imply an automatic diagnosis of schizophrenia.

In every case of a catatonic syndrome, a process of clarification of the etiological diagnosis must proceed systematically. Such a procedure will assist in making the proper diagnosis and administration of appropriate therapy. As the initial step, it is important to ascertain whether the patient is suffering from an organic or a functional illness by means of a good history, physical examination, and the results of laboratory investigations. A clear consciousness is more in favour of a functional disorder. Subsequently, it is imperative to search thoroughly for the etiology and then clinical diagnosis in functional stupor.

To illustrate the importance of making the proper diagnosis of catatonic stupor, we wish to report the case history of a patient who presented with typical catatonic stupor and on investigation she was found to have temporal lobe epilepsy (Partial Complex Seizure).

**Case History**

A 24-year-old black female, unwed mother of a two-year-old, was hospitalized in the Harbor General Hospital on 8-10-83 because of persistent thoughts of suicide either by shooting herself or running in front of a car. This was her third hospitalization within a two month period. She was initially hospitalized following an overdose of aspirin a month before and rehospitalized after complaining of feeling depressed, empty, suicidal and insomnia three weeks prior to the current admission.

On this admission, there were no precipitants noted. She stated clearly that she wanted to die and had intermittent feelings of not belonging, being empty since the age of twelve. At that time, her parents divorced and her mother was diagnosed as suffering from schizophrenia with subsequent multiple psychiatric hospitalizations.
The patient expressed fears of having her mother's illness and joining her in a board and care home. She also revealed that she was a victim of several rapes resulting with the birth of her daughter. She had a recurrent thought that no one would attend the funeral. Her mental status examination was unremarkable except that her mood and behavior fluctuated from being smiling and engaging to being withdrawn and sad. Physical examination was unremarkable. Routine laboratory tests including hemogram, urinanalysis, VDRL, total bilirubin, alk. phosphatase, SGOT, total protein albumin, inorganic phosphorus, uric acid, BUN, creatinine, chloride, bicarb K, Na, Ca, glucose, cholesterol, lactate dehydrogenase were all within normal limits except for microcyclic anemia (hemoglobin 11.5 G).

Patient is the oldest of seven children. The father is a truck driver and is absent from home for long periods of time. Therefore, the patient assumed the responsibility of raising her six brothers and sisters. Her relationship with her father was cold and distant. She had a difficult childhood and was regularly beaten with an extension cord.

On the third day of hospitalization, she became catatonic, totally mute and unresponsive to external environment. During this five hour period, she also manifested posturing with waxy flexibility. She was given 2 mg of haloperidol and within one hour after the injection became responsive to her environment although somewhat sedated. At that time she had no recall of her catatonic behavior and expressed fear of losing control of her emotions. A sleep deprived EEG with naso-pharyngeal leads the following morning revealed diffuse slowing and spiking in the temporal region suggesting a generalized seizure disorder with a temporal lobe focus. A detailed neurological examination, CAT scan and examination of the cerebrospinal fluid obtained by a lumbar puncture revealed no abnormality. She was stabilized on Dilantin 400 mg daily and was discharged home two weeks later.

Discussion

Our patient presented with the typical clinical features of an abrupt onset of catatonic stupor including mutism, posturing, akinesia and waxy flexibility. Her two past hospitalizations, the age of onset, a family history of schizophrenia in her mother and waxy flexibility were all indicative of a diagnosis of catatonic schizophrenia. However, she improved with haloperidol. As she did not have any memory of her catatonic episode, organicity was suspected. In addition her EEG demonstrated a temporal lobe focus. She progressed well on antiepileptic drugs. Thus, this case illustrates the difficulty to distinguish a functional stupor from the organic. Absence of memory for the event and alteration of consciousness are more in favor of an organic diagnosis while a past family history of psychosis favor the diagnosis of a functional illness. In doubtful cases, it is necessary to weigh the clinical data to exclude the conditions that may produce a catatonic picture and investigate appropriately to establish an etiological diagnosis.

Temporal lobe epilepsy is well known to produce schizophrenia- like syndromes (Slater and Beard 1963), as well as affective disorders (Dongier 1959). In addition, catatonia occurring in these patients has long been recognized. Drake and Coffey (1983) reported two patients who developed stupor and the clinical recognition was hindered because of features resembling psychogen-
ic or feigned unconsciousness. Slater and Beard (1963) found two patients with catatonic stupor among the 69 patients of schizophrenia-like psychosis with epilepsy. Gomez et al (1981) reported that a 38-year-old catatonic worsened on neuroleptics and was later found to have had epilepsy. Hence, it is not infrequent that a catatonic clinical picture may be associated with epilepsy.

Two aspects of treatment are important. Firstly, neuroleptics may or may not be effective (Gomez et al 1982) and secondly they may even induce catatonic states (Geleberg and Mandel 1977; Nakra and Hwu 1982). Hence neuroleptic therapy should await the definitive diagnosis in all cases of catatonic stupor.

References

DONGIER, S. (1959), Statistical study of clinical and electroencephalographic manifestations of 536 psychotic episodes occurring in 516 epileptics between clinical seizures, Epilepsia, 1, 117-142.

DRAKE, M. E. & COFFEE, C. E. (1983), Complex partial status epilepticus simulating psychogenic unresponsiveness, American Journal of Psychiatry, 140, 800-801.

GATEWOOD, J. W., ORGAN, C. H. & MEAD, B. T. (1975), Mental changes associated with hyperparathyroidism, American Journal of Psychiatry, 132, 129-132.

GELENGERG, A. J. (1976), The catatonic syndrome. Lancet 1, 1339-1341.

GELENGERG, A. S. & MENDEL, M. R. (1977), Catatonic reactions to highpotency neuroleptic drugs, Archives of General Psychiatry, 34, 947-950.

GOMEZ, E. A., COMSTOCK, B. S. & ROSARIO, A. (1982), Organic vs. functional etiology in catatonia: case report, Journal of Clinical Psychiatry, 43, 200-201.

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

JOYSTON-BECHEL, M. P. (1966), The clinical features and outcome of stupor, British Journal of Psychiatry, 112, 967-981.

PENN, H., RORY, J. & LAPHAM, L. (1972), Catatonic behaviour, viral encephalopathy, and death. The problem of fatal catalonce, Archives of General Psychiatry, 27, 758-761.

PLUM, F. (1972), Prospects for research on schizophrenia-3. Neuropsychology, Neuropathological findings, Neuroscience Research Progress Bulletin, 10, 384-388.

SLATER, E. & BEARD, A. C. W. (1963), The schizophrenia like psychoses of epilepsy-I. Psychiatric aspects, British Journal of Psychiatry, 109, 95-115.

S. NAKRA, B. R. S. & Hwu, H. (1982), Catatonic-like syndrome during neuroleptics therapy, Psychosomatics 23, 769-770.

TAYLOR, M. A. & ABRAMS, R. (1977), Catatonia, Archives of General Psychiatry, 34, 1223-1225.

WEINBERG, D. R. & KELLY, M. J. (1978), Catatonic stupor and neuroleptic drugs, Journal of American Medical Association, 239-1846.