cated by respiratory paralysis resulting in postanoxic encephalopathy and a PVS. After 27 months she was still labeled with PVS by a prominent neurologist specializing in PVS. His videotape reflected a classical neurological evaluation of such a patient. Responses to stimuli were labeled as reflexive and aversive: Indeed, many were (plantar extensor, startle response, palmarmental reflex). No detailed clinical higher brain evaluation was performed. During neuropsychiatric examination shortly thereafter, however, she showed intermittently differential responses to both sensory stimuli and to verbal instructions from her caregivers (consistent nursing staff, family members) compared with those of the evaluating neuropsychiatrist. This early differential response was well demonstrated in nine different ways, sometimes using an ABA design. For example, when told the neuropsychiatrist would put his finger in her mouth, she clenched tightly. When the nurse asked her sweetly to open her mouth for her finger, she immediately complied. When the neuropsychiatrist requested the same, she clenched tightly again. These differential factors put the patient in a MCS because of the demonstrated intermittent minimal consciousness.

The medical confirmation of MCS provided impetus for more appropriate follow-through on her responses and targeted rehabilitation, This diagnostic awareness produced earlier interventions, jumpstarting the relatively small improvements. By a year later, her MCS was clear as she exhibited consistent nonreflex responses to certain musical and TV-show stimuli, occasional verbalizations and ostensible awareness of when to pass urine and stools leading to contemplated potty training.

This earlier diagnosis using differential cerebral cortical testing with consequent rehabilitation could be generalized to others ultimately leading to more standardized subtle cerebral cortical approaches to MCS and PVS. These specialized examinations optimally extend the neuropsychiatric and behavioral neurological domains using subtle behavioral differential responses that are not in the usual armamentarium of the classical psychiatric or neurological examinations.

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The Schizophrenic Disguise of Complex Partial Seizures

SIR: The call for integration of Neurology in Psychiatric training has a long history.[1] The following case is reported from the United Kingdom where the divide between neurology and psychiatry is deepening everyday as opposed to the American model.[2]

Case Report

Ten years ago AB presented to a child psychiatrist when she was 16 with gradual withdrawal and unprovoked attack on classmates. Her younger brother had learning difficulties and episodic behavioral dyscontrol and was suspected to have epilepsy. Her mother was worried whether AB used drugs. She claimed hearing abusive voices in third person discussing her actions. But she did not appear too distressed. She also felt that she was being watched and her food might be poisoned. She became increasingly disruptive at home, physically fighting with brother and peers. She also threatened to harm her mother. There were incidents when she appeared drunk but not smelling of alcohol. She claimed the house was being bugged and that the TV is a "special camera observing her." She was admitted informally for an assessment but later detained due to lack of diagnostic progress in wake of troublesome behavior and abuse of cannabis, amphetamines and ecstasy at hospital. She was diagnosed to have schizophrenia and treated with haloperidol which was later changed to sulpiride. Her EEG showed bilateral minor sharp waves of unknown significance especially on right side which was regarded nonspecific and insignificant. CT Brain done around this period was normal. She made a slow recovery with a high degree of non compliance and presented to adult psychiatric services with depression 4 years later when she stopped all antipsychotics. She was started on sertraline which she stopped on her own after a very short course. By this time she was married and had had another episode of depression when she was noted to wander and "going into daze." Change of antidepressants proved unhelpful and she started reporting new symptoms of déjà vu and "reading others minds." She also reported seeing flashes of events before they occur with increasing paranoia and referential ideas. During this period she would stare at space and 'lose sense of things around her' with electric sensations at the back of her head and spasmodic jerks of one arm or leg and facial droop. Most of her symptoms were seen as psychotic features with drug induced side effects. Switching various atypical antipsychotics proved unhelpful, increasing the frequency of her déjà vu at times. An EEG was done

which picked up persistent paroxysmal right temporal lobe activity. MRI did not show any structural anomalies. The diagnosis was revisited after 8 years from initial presentation and she was started on carbamazepine after stopping atypical antipsychotic. She made a remarkable improvement from symptoms of déjà vu and "psychotic" features. Symptoms of derealisation decreased considerably and she is now under shared neurology and psychiatric care. Retrospectively, her depression seems to be an interictal phenomenon, relatively resistant to antidepressants with ictal frequency increasing on psychotropic treatment.

Some important learning points transpire from this case. Temporal lobe epilepsy has been a great disguiser at psychiatric clinics. But missing a diagnosis for nearly a decade occurs rarely.[3] It is well noted that a single EEG will not rule out possible seizure disorder.[4] In this case the non specific reported findings were taken for schizophrenia related variation from normal. It is clear that lack of clarity about EEG findings in a psychiatric illness and general hesitancy in utilizing repeat neurophysiological investigations could lead to potential misdiagnosis.

The importance of neurological training for a psychiatrist is clearly illustrated by this case. The time-honored approach of detailed analysis of representative cases should serve to improve our practice and convergence of neurology and psychiatry within the framework of neuroscientific clinical practice.[1] Even today in some countries including U.K it is possible to qualify as a psychiatrist without spending a single day in a neurology clinic.

Inspite of increasing evidence that first-rank symptoms have a wider prevalence, their strong influence on tilting the diagnostic balance toward schizophrenia, at least in Europe, is immense as in this case.[5]

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Amantadine in Catatonia Due to Major Depressive Disorder in a Medically Ill Patient

The treatment of catatonia has been well established with trials of lorazepam and ECT. Recent treatment guidelines list these as effective in acute and chronic catatonia; however, if patients do not respond to these primary treatments, clinicians have few guidelines to guide treatments for refractory catatonia.[1,2] Amantadine has been demonstrated to be effective in refractory catatonic schizophrenia, but little evidence exists supporting its potential use in catatonia secondary to other conditions including mood disorders.[3,4]

Case: A 38-year-old male was admitted to the internal medicine service with failure to thrive and end stage renal failure with uremia requiring hemodialysis. His clinical course was notable for personal neglect and a severe decline in function over the previous 3 months due to worsening major depressive disorder. A psychiatry consult was requested from the medical team due to prominent akinetic catatonic features including severe alexithymia, immobility, speech-prompt mutism, staring, and reduced oral intake resulting in a Clinical Global Impression Scale Score of Marked Severity. The patient was not considered appropriate for trial of lorazepam due to intermittent symptomatic hypotension with systolic blood pressure of 60–90 mmHg. The patient was begun on sertraline 50 mg daily, risperidone 1 mg daily, and methylphenidate 5 mg daily, with only minimal improvement after 2 weeks, whereupon he was referred for ECT.

All psychiatric medications were discontinued on the day prior to beginning ECT. He received 18 bilateral index ECT treatments with a mean EEG-ictal seizure length of 68 sec; however, he remained markedly catatonic throughout the course of ECT. At this point the patient started amantadine 100 mg daily for 12 days with partial improvement in his catatonic symptomatology. The patient became able to answer simple questions, staring resolved, and he displayed occasional spontaneous movement, even though he continued to manifest significant speech prompt mutism and a paucity of spontaneous movement (CGI-I = Mild Improvement). The dose was increased to 100 mg twice daily, and after 2 weeks, the patient showed marked improvement with resolution of his catatonic presentation (CGI-I = Very much improved).

This case is notable in that it demonstrates an episode of catatonia responsive to amantadine in a medically ill individual that was