The Catatonia Foundation

What's your experience treating patients with catatonia?

Dr. Caroff:
Because of that orientation at Stony Brook, I continue to have a great interest. I have observed and written about patients with catatonia ever since I was very fortunate to be at the referral center at the VA, because I saw some wonderful, amazing cases in the beginning. In fact, I remember we were using sodium amytal. Benzodiazepines weren't yet considered first-line treatment for catatonia, so we used that older barbiturate, and it was a great ceremony to have patients with catatonia and infuse sodium amytal and watch them wake up.

Like Dr. Sacks wrote about encephalitis lethargica, we really saw awakenings in people who were stuporous, nonresponsive — we give them sodium amytal, they'd wake up, start talking, and we can do a diagnostic interview.

So I was around even then, until Greg Fricchione up at Harvard reported on lorazepam being a great treatment to reverse the effects of Catatonia, which had a dramatic effect on the field. And he, of course, continues to do great research on catatonia.

But as I said, we saw our own patients, and patients around the state of Pennsylvania were referred for ECT. And what struck me — the cases of stuporous catatonia, we're familiar with and can very effectively treat with benzodiazepines or ECT. But I was also impressed with the variations of catatonia and how common it was, which people didn't respect. A lot of people thought catatonia had disappeared in the mid- and late-20th century. It's still a mystery to this day how that could be ignored. But we saw a lot of interesting patients with catatonia.

I became very interested in drug-induced catatonia. A lot of my research has to do with antipsychotic treatment, and we were very aware and saw a lot of patients who were treated with antipsychotics or various disorders and then developed catatonia. That resulted in what was described by Ira Brenner as “the catatonic dilemma” you may have heard about, where people with schizophrenia would come in, would be treated with one of these old antipsychotics, and then would become catatonic. And as a clinician, you didn't know if the catatonia was due to the antipsychotic or to their underlying condition. That was called “the catatonic dilemma.”

We realized that the thing to do was to stop the antipsychotic. And we would change the antipsychotic, and if that was the cause of the catatonia, it would resolve or go away.

I think in those days, when the more potent antipsychotics were being used, a drug-induced cause of catatonia was very common. In fact, it may have been the most common form of

catatonia in those days. And, like I said, we became very aware of the need to stop the antipsychotic, and catatonia would often go away. Whereas nowadays, there are better, newer, safer drugs that don't cause catatonia as often.

So now I think catatonia is more likely to be caused by other medical conditions or other disorders, and less so by the antipsychotics. But again, over the years, especially in the early years, I was very impressed with that. In fact, in the severe forms of drug-induced catatonia, like neuroleptic malignant syndrome — you may have heard of NMS — was also something that we had a direct hand in trying to make people more aware of the fact that people with these severe forms of catatonia didn't have schizophrenia. They had a drug-induced cause of catatonia.

We saw that, and because we were interested in those severe forms of catatonia, we got a lot of referrals of people who had encephalitis and also had catatonia. So we went back and looked at the literature on all the cases reported of people who had encephalitis and presented with catatonia. Even before the NMDAR observations, we were aware and reported on the fact that catatonia is very common in people who have infections or inflammation of the brain, like an encephalitis. Something like 30 to 40 percent of those cases are people who presented with psychosis that was caused by encephalitis. People with catatonia who really had encephalitis, that occurred like, 30, 40 percent of people with encephalitis. So now, fortunately, it's better recognized that if you have someone who shows up with catatonia, no mental illness background, you have to consider encephalitis. So we saw that.

I was also impressed with patients I saw who had repeated episodes of catatonia without any underlying cause. We couldn't find underlying causes — they didn't have schizophrenia, they didn't have mania. They just kept coming in with catatonia. So we're impressed with those so-called “idiopathic recurrent forms” of catatonia, often misdiagnosed. They wound up being labeled as schizophrenia, schizoaffective disorder.

Unfortunately, our diagnostic system, even the DSM, doesn't have a good way to categorize these people who have these various kinds of catatonia. A great thanks to Andy Francis and Max Fink and Mickey Taylor for lobbying DSM people to change catatonia and bring it more up to date, and separate it from schizophrenia so people wouldn't think that catatonia was synonymous with schizophrenia and mistreat the patients. So now at least catatonia has a home of its own, as they said. So they deserve a lot of credit for lobbying for that over the years. And it was very successful.

So catatonia throughout my career has been an interest because I think it's a common problem and it has a lot of importance in terms of proper treatment and even implications for research.