The Catatonia Foundation

What is ECT?

Dr. Fricchione:
I'm not an expert, but I think ECT is — there's another area that deserves more research: trying to figure out why it works so well. And I think that there are different paradigms that you could explore to get an understanding of ECT's mechanism of action. One of them is the seizure threshold mechanism of action because we know that ECT has been used for intractable seizures.

You can wind up increasing seizure threshold if you give ECT. And, of course, that reminds us of that earlier discussion we just had about seizure threshold, kindling, the relationship between bipolar illness and catatonia. Why is it so good for bipolar illness? Why is it so good for catatonia?

I had that mentor, George Murray, he was fond of saying, “You always have to be on the lookout for seizures when you're thinking as a psychiatrist.” And he would say, “Trying to find a seizure is like going fishing in a lake. The fact that you don't catch anything doesn't mean there are no fish.” And he would then go on to tell us about how hard it is to pick up seizures in the hippocampus because of the structure of the hippocampus. It's not a sort of layered system where the impulse can travel up the layers; the impulse can be going around in a circle in the hippocampus until it escapes.

So there are probably a lot of patients out there who are walking around with hippocampal dysrhythmias that are not being picked up on surface EEGs. Not only that, but there are probably other parts of the brain where there are these subcortical dysrhythmias. Who knows? I mean, catatonia could, in a fair number of cases, be related to subcortical dysrhythmias, and there may be a certain group of bipolar patients who are bipolar because of subcortical dysrhythmias in certain parts of the brain. What's causing those subcortical dysrhythmias? They may be excitatory-inhibitory imbalances in parts of the person's brain.

So, why is ECT beneficial? It's because it's doing something perhaps to that dysrhythmia by allowing seizures to occur. In a kindled brain, you may be actually providing therapeutic benefit. There's a force normalization hypothesis that too much control of seizures actually leads to psychotic behavior because there are certain patients who need a discharge of this buildup of subcortical dysrhythmia. So that's one paradigm that you've seen.

Another one is inflammatory, that ECT will have effects on inflammatory markers, and there is a good story to tell about neuroinflammation in catatonia. We're learning more and more about microglial cells and how important they are for neurological functioning, how important they're gonna be for those four operations I was talking about, and how susceptible we are to stress-

induced neuroinflammation. And so going way back to what we started with in terms of stress and how people are responding to stress in different ways, there may be a neuroinflammatory connection from toxic stress leading to mitochondrial changes that, through oxidative stress, will lead to neuroinflammation. So that's another big one. And then, you know, there are effects of ECT on transmitters.

No one I think has a real clear picture of that. Someone may — I may not be aware of it, but so there's a neuromodulation hypothesis. But again, I'm not an expert on it.