The Catatonia Foundation

What is catatonia?

Dr. Fricchione:
There's a whole evolutionary story because over the course of a hundred million years, over 300 million years ago, mammals developed, and their survival strategy was an absolute requirement for parental and social attachment. And we are very highly ordered conscious mammals, so this is what is most important for all of us. And of course, when you look at culture, when you look at society, it's staring you in the face. The thing that hurts us the most is social rejection.

I had a sabbatical once, and I spent it at the Brain Evolution Lab at NIMH in 1990 with a mentor by the name of Paul MacLean, who's the father of the limbic system. He taught me that the most painful mammalian condition was separation. And there's a particular part of your brain that Paul studied really, really closely — the anterior cingulate and the surrounding medial prefrontal cortex, which is absolutely essential for parental and social attachment.

It's amazing how that area is the convergence of pretty much everything that makes our lives important and worth living. Nature, evolution has said for you, as a mammal with an anterior cingulate, it's even more important sometimes for you to be socially accepted, and when you're not, that pain is gonna be so important for your decision making. Which the anterior cingulate also is extraordinarily important for the decisions you make. We are gonna put it there in the anterior cingulate, right next to the same area of the brain that tells you when you're a kid, don't put your hands on the stove. You're not going to make that decision again once your anterior cingulate tells you that that's excruciating, right?

So when you talk about a patient with catatonia, this area of the brain is so important for human life that for me, that has been a central area of trying to understand why patients succumb to catatonia when they're extraordinarily fearful.

The only way to understand that given the phenomenology, the symptom complex that we call the catatonic syndrome is to sort of say, you've got areas of that medial prefrontal cortex, not only anterior cingulate, but also of the orbital frontal cortex and so on that speak for the anterior cingulate — goes up and it speaks to the premotor cortex, to the supplementary motor cortex and to the primary motor cortex, and it's creating a template for movement.

So remember, sensory motor analyzer, effector, you're getting impulses about your environment. They're traveling up to this region of the brain where you're analyzing that data with your past history and what has happened, and the emotionality from the limbic system, which meet up in the anterior cingulate. And then you're making a decision in order to actually

affect that decision. You move that information up road to the premotor cortex, you form a template for movement, supplementary motor cortex and M1, the primary motor cortex.

So there's a plan for what the anterior cingulate has decided to do, but the brain, through evolution, is extraordinarily adept at trying to keep you safe. So it also sends a message down into the basal ganglia laterally to the motor part of the system. They're talking to one another, the primary motor cortex, which has gotten the template and the basal ganglia, which is getting feedback information as things are happening that, “Okay, this is safe, it's a good decision. You're not going to suffer consequences by continuing to carry out this plan.” And they work together, and the person moves.

So what happens to the individual who, for a medical disorder reason, for a neurological disorder reason, or for a psychiatric reason, the thalamus — the filter — is malfunctioning? The information is getting to this very important area to make decisions, and it's all jumbled. But it's also giving the patient a sense of paranoia or fear that if they move, they're going to make a mistake and they're going to suffer. They may even die. You know, some patients with catatonia have Cotard's, where they think they're dead.

What's happening with poor catatonic patients is they’re removing themselves from life. Not that they're not sensing, but they have really dammed up the analyzer effector part of their system. And this is why the old writers, the German writers, called it a problem of volition, right? Of motivation — when they were talking about the psychiatric patients.

But of course, as I mentioned, you can gum up the works neurologically, either with lesions or with modulator problems. So it's even more complicated.