What studies are you working on?

Dr. Northoff:
We developed this rating scale, as I said. Also, we compared subjective experience in Parkinson's patients with akinesia versus catatonia and say, “Okay, this is really different to psychomotor versus motor.”

Based on these clinical observations, you really develop this concept of psychomotor versus motor so that effective cognitive experiences/events translate into motor behavior changes. And accordingly, we developed this scale for subjective experience. Then we developed a ball-throwing test. And then we also did corresponding imaging data — FMRI, EEG, and said, “Okay, this really seems to be,” for instance, “the orbital frontal cortex or subcortically could be the raphe nucleus serotonin,” which then modulates dopamine. So we could really see the motor cortex is just the output, but it's not the source.

And we have now released a paper, also psychomotor versus motor, where we directly compared these two hypotheses. It's really clear you have motor cortex changes, no doubt, you have subcortical basal ganglia changes, you have probably dopamine changes, but these are secondary to some other changes in the cortex or other subcortical regions, like for instance, raphe nucleus serotonin.

So that's why we speak of psychomotor mechanisms. And I think that's very important for treatment. So this is diagnostically important because you can use some of these markers. That's what our hope is for differentiating a primary movement disorder, extra primary movement disorder like Parkinson's or dystonia or tremor, or even Huntington’s, from catatonia.

And so that's what we hope that we're now working on that we really try to develop. Maybe we can use one of these psychomotor mechanism connections between cortical regions and the motor regions or modulation of the dopamine system by serotonin as biomarkers for catatonia as distinguished from, let's say, other disorders — pure psychosis without catatonia or depression without catatonia.