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LETTERS TO THE EDITORS

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Comments on "Diminished cerebral metabolic response to motor stimulation in schizophrenics" by Guenther et al. (Eur Arch Psychiatry Clin Neurosci, 1994, 244: 115–125)

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Dear Sirs:

In their paper entitled "Diminished cerebral metabolic response to motor stimulation in schizophrenics: a PET study" (244: 115–125, 1994), Guenther et al report that patients with schizophrenia failed to show normal activation of contralateral motor areas, despite making the same actual finger movements as healthy controls. In discussing their findings the authors take the important step of moving beyond prevailing notions of psychiatric illness as localized dysfunction of a specific brain locus. They note a similarity between their findings and the fact that "other laboratories have reported failures to activate in response to behavioral challenges of various cortical regions," and astutely "propose, therefore, that schizophrenia involves a derangement of brain organization that limits the ability of these patients to produce a focal metabolic response to stimulation of a number of functionally distinct cortical regions."

I find this proposal particularly pleasing because in 1991 I made the same suggestion in a paper entitled "Failure at task-specific regional brain activation: new conceptualization of a disease entity" (J Neuropsychiatry, 3, 94–98, 1991). I cited previous work by Guenther and colleagues that also demonstrated that patients failed to normally activate the motor cortex during a motor task (Guenther et al. 1986), as well as two other cerebral blood flow studies cited in the current paper by Guenther et al. (Weinberger et al. 1986; Buchsbaum et al. 1984), additional blood flow studies by Gur and colleagues (1983, 1984), and my own dichotic listening studies. I "proposed the existence of a disease characterized by abnormality in the general physiological mechanism of task-specific regional brain activation." It is encouraging to find that other investigations have come to a conclusion so similar to my own.

Both Guenther et al. and I note that patients fail to show normal blood flow activation despite performing the same task or receiving the same somatosensory stimuli as controls. This led me to suggest that "what differentiated patient and control subjects was that control subjects showed a regional spread of activation beyond that minimally required by the task. The absence of such elaborative activation may be the neural basis of the poverty of thought and the inability to sustain attention and interest seen in patients diagnosed as being either negative-symptom schizophrenic or endogenously depressed. A failure of elaborative activation could also be responsible for the absence of social relatedness and responsiveness seen in these patients."

There is another difference between my paper and that of Guenther et al. I proposed that the physiological failure at task-specific regional brain activation be the defining characteristic of a newly conceived disease entity. I predicted that this disease entity would include some, but not all, patients who carry a symptom-based diagnosis of schizophrenia, as well as some patients currently diagnosed as depressed. This prediction was based in part on the previous study by Guenther and colleagues (1986) in which both negative-symptom schizophrenic patients and endogenously depressed patients failed to show normal activation of the motor area whereas positive-symptom schizophrenic patients actually showed excessive activation. Given the limitations inherent in psychiatry's current symptom-based nosology, it is essential that investigators begin using physiological abnormalities as primary diagnostic constructs, rather than attempting to fit new findings into current, flawed, diagnostic categories (Wexler 1992).

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