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Editorial Comment

Johnsen, Fasmer, Van Waginengen et al. present a preliminary study of the effect of the glutamate antagonist memantine on the variability of motor activity in normal humans compared with a group of schizophrenic patients. The study of animal movement within a range has been studied with the model of Fourier transformation for years by ecologists. This was my first exposure to its use in human behaviour (1). It appears to provide meaningful results, and this group seems likely to continue to apply and refine the approach. Their results are interesting, and seem to support the hypothesis that motor abnormalities associated with the disease of schizophrenia are mediated by changes in *N*-Methyl-D-aspartate (NMDA) receptors, their neurons, and their circuits (2). Where results seem inconsistent, they may clarify what we know about long-term potentiation at NMDA receptors involvement in learning (3–5). For example, the greater effect of memantine on movement of the dominant arm might be evidence of the learning of skills in the dominant hand and arm. The matter deserves further study, as potentiation of neurotransmission at NMDA receptors is involved in all learning processes that have been studied. Thus, we see a pharmacological probe getting at something that may be at the core of how the brain and body work within and on the environment.

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