spines of the striatal nigral projection neurons. Synaptic transmission in these pathways is blocked by glutamate antagonists. The non-competitive antagonist of NMDA receptors which act at the receptor-operated ion channels is particularly interesting, because it includes phencyclidine and chlorpromazine, which have psychotomimetic and antipsychotic properties respectively.

It is possible that during severe psychosocial stresses the excitatory amino acid pathways are overstimulated, which leads to an increase of glutamate transmission and calcium influx. This results in excitotoxicity effect on the striatal neurons where NMDA receptors are located. This in turn activates the dopaminergic pathways which synapse on the dendritic neck of the same neurons. This overreaction and degeneration lead to the schizophreniform reaction and chronic schizophrenic process as mentioned earlier, depending on the severity of the damage done and the vulnerability of the dopamine system.

According to this hypothesis, NMDA antagonists and calcium channel antagonists may be effective in preventing the progression from acute to chronic schizophrenia. Yet non-competitive NMDA antagonists such as MK 801 and phencyclidine are found to have psychotomimetic effects. The possible reason is that when the NMDA ion channels are blocked, there is increased transmission through the non-NMDA receptors as well as activation of dopamine-containing elements in the striatum. More research in this area is awaited.

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Sir: Frith & Done (*Journal*, October 1988, **153**, 437–443) provide an elegant account of the symptoms of schizophrenia in relationship to the self-monitoring of actions. The positive symptoms are regarded as a failure of monitoring, because of the degradation of information about willed intentions. In contrast, negative symptoms are associated with a difficulty in forming willed intentions. I would suggest that such an explanation is both faulty and unneccesarily complicated.

The concepts of 'the will' and 'intention', and the associated 'act', are notoriously problematic. (What is an 'act of will' in this context?) Nevertheless, if we in some form accept the existence of 'willed intent' we readily recognise Drs Frith & Done's suggestion that this in some way fails in the schizophrenic with negative symptoms. However, does it not fail in all of his actions, whether generated from stimuli or from a willed intent? The inability of social stimuli to generate 'normal' and acceptable behaviour is a familiar problem with such patients. Furthermore, we know that the acutely psychotic patient can be disinhibited and also fail to respond to social mores. We therefore have to elaborate the theory, perhaps by arguing that such patients are unable to understand these stimuli. Alternatively, we can make the much simpler statement that all schizophrenics have potential difficulty in forming 'willed intentions' for all kinds of acts. Or should we merely reassert Bleuler's contention that ambivalence of the will is a fundamental symptom of schizophrenia?

Considering now the positive symptoms, the theory has the advantage of describing percepts, beliefs, and emotions as irreducible mental events. "First rank symptoms are essentially abnormal experiences and do not necessarily result in obvious changes in behaviour." It thus becomes easier to understand the fact that a psychotic patient can experience such phenomena at the same time as experiencing both the real world and other people's reactions to him; and that he can use any combination of phenomena to inform his actions. Furthermore, 'insight' can also be seen as a process separate to this event. It can be understood within the context of Drs Frith & Done's theory as the appraisal of the correlation of this mental event with reality, by a further monitoring system (which may or may not be intact).

However, having accepted such mental events as irreducible, and subject to monitoring systems, how should we classify these events? It seems straightforward to classify physical actions (and the preceding neural activity within the motor cortex) as 'acts', subject to the constraints outlined above. It seems less easy, but possible, to classify thoughts as 'acts'; we are given a helpful quotation from Piaget: "thought is internalised action". However, it does not seem possible to classify percepts (or hallucinations), beliefs (or delusions), and emotions as actions. Drs

Frith & Done avoid this in part by flaunting the convention of classical phenomenology and attributing hallucinations to thoughts, rather than treating them as percepts. If they do accept hallucinations as percepts then they themselves would place this phenomenon in a category entirely separate to actions, and rightly so, since the notion of 'willing' a percept is not convincing. (One may will an image, which is why percepts and images are entirely distinct experiences.) Is a belief an 'action'? A belief is a predisposition to act. The associated mental 'action' is the thought which is used to rehearse the belief. Is an emotion an 'action'? Anyone who has been unable to change their feelings but has been able to change their actions could describe the difference, and would say without doubt that emotion is most definitely not the result of 'willed intent'.

I would therefore suggest that physical actions and thoughts do have a willed intention, although this is rarely conscious outside of situations of conflict. However, percepts, beliefs, and emotions are not produced by willed intention – other than those intentions of which we are as unconscious as the schizophrenic person. These irreducible mental events are different both from actions and from each other. The Frith & Done theory thus succeeds only as a neuropsychology for the abnormal schizophrenic experiences of the passivity phenomena of thoughts and actions, and the willed intention to think, i.e. *Gedankenlautwerlen*. (The only experimental evidence cited is in fact for the monitoring of motor actions by schizophrenic patients.) It is not convincing for the entirely different experiences of percepts, beliefs, and feelings.

If such events are not willed, where do they come from and how do they arise? We must assume that percepts arise from sensory experiences, and that beliefs and emotions arise from the black box of our consciousness as a consequence of other inputs. They do so in a way that we can make some sense of – i.e. in an 'understandable' fashion. Thus we feel justified in labelling these events, as we do, as our own. Here Dr Frith points out (in my view correctly) that we differ in this way from the schizophrenic person who sometimes fails to achieve correct 'labelling' of these events. I would suggest that this is simply because these events appear to arise *de novo*, not because they are a consequence of unmonitored intentions. They could appear to arise *de novo* because they occur as a consequence of a structural or biochemical abnormality, or because they arise from one's consciousness in an non-understandable way – presumably for the same reasons. Given their non-understandable origin, these events will sometimes be recognised as 'not of self' (i.e. not arising from one's own consciousness). They will then be (incorrectly) attributed to externally generated sensations, producing hallucinations, or to external agents, producing the passivity phenomena of sensations and feelings. They could also be attributed to thoughts and actions, allowing one to abandon the faulty internal monitor altogether. Sometimes they will be accepted as 'of self', resulting in delusions which are accorded the same special status as religious insights: not arising from data from the outside world, and therefore not requiring testing against it. If 'will' or 'willed intention' is yet another mental event, there is no reason why these should not also arise apparently *de novo* in the schizophrenic person, thus creating conflicting wishes and intentions, and the ambivalence of the will.

Such a hypothesis draws directly on the theories of Hughlings Jackson: any cerebral lesion results in negative symptoms, due to loss of tissue, and to positive symptoms, since loss of tissue results in the 'release' of other areas of the brain. It is both more parsimonious than that offered by Drs Frith & Done, and in line with the traditional phenomenological and philosophical understanding of such mental events. It explains how pathological, physiological, and biochemical changes at almost any level of the central nervous system can result in psychotic phenomena of any sort, in any illness, simply because they can produce these mental events. It seems superfluous to invoke a final common pathway through an 'internal monitor'. Schizophrenia may well be a distinct illness, but it is not its capacity to cause psychotic phenomena that makes it distinctive.

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SIR: I would like to add to the model of Drs Frith & Done by putting forward some ideas derived from my interest in artificial cognition. Hofstadter (1982) has pointed out that thought has a monitoring quality with increasing levels of abstraction and breadth of observation. He likened this to a mathematical regression, but pointed out that the regression is not infinite as the self-watching nature of a monitor at some level of abstraction detects infinite, or even mildly repetitive, patterns of thought. The lowest level of monitoring would be the conscious channel through which our thoughts run, analogous to a central processing unit (mainly processing, little abstraction), and the equivalent of the comparator of Gray (1981). I propose changing the schema illustrated by Drs Frith & Done in their Fig. 1 by suggesting that