A Rating Scale for Psychotic Symptoms (RSPS):
Part II: Subscale 2: distraction symptoms (catatonia and passivity experiences); Subscale 3: delusions and semi-structured interview (SSCI-RSPS)

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Abstract

In the second paper on the Rating Scale for Psychotic Symptoms (RSPS), distraction symptoms (Subscale 2) and delusions (Subscale 3) are described. Subscale 2 includes distraction symptoms, which arise from one of two mechanisms: the symptom may arise either by loss of attentional focus (LAF) due to a competing channel of information, or by the intrusion of the competing channel into the focus of attention [attentional intrusions (AI)]. The symptom classes resulting from loss of attentional focus (LAF) include motor catatonia, negativism, and thought blocking; the attentional intrusions (AI) symptoms rated include three types of passivity experiences (Schneiderian symptoms): (1) thought insertion, (2) movements or action controlled, and (3) speech controlled by an external force. Subscale 3, consisting of delusions, is organized on the basis of content identification. Nineteen types of delusions are rated. Each item of delusional content is rated along three axes (active elaboration, persistence and active extinction) and complexity, and optionally if primary or mixed (i.e. primary with secondary elaborations). The last section of the paper includes the semi-structured interview (SCI-RSPS) for each of the items, as well as guidelines for practical application of the interview. © 1999 Elsevier Science B.V. All rights reserved.

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1. Subscale 2: Distraction symptoms

1.1. Theoretical basis

The second class of symptoms, classified as distraction symptoms, derives from the concept of selective attention and its dysfunctions, and the evidence for the role of dopamine in attentional processes (Miller,
One central feature of attention is that there is competition between different channels of information. As a result, under normal circumstances, one channel of information comes to command all attentional resources, while competing channels are suppressed. Miller and Wickens (1991) have previously suggested a neural model by which such competitive effects appear, these being dependent on the inhibitory interaction between neighboring neurons in the striatum. The competitive interactions arising in the striatum may then also be imposed on the different cell assembly configurations in the cerebral cortex, so that only one of these can be activated at once.

Under the abnormal circumstance of enhanced striatal dopaminergic activity, the competitive processes underlying normal attention become dysfunctional. Instead of a stable state created by the complete dominance of one channel of information and the suppression of competitors, an unstable state is created. As a result, it becomes difficult to maintain attention for long on one channel because there is a tendency to continual interference from competing channels. This type of pathology has been documented in a number of experiments in rats given stimulant drugs. The exact explanation in terms of a neural model of dopaminergic effects on synaptic strengthening and the consequent change of network dynamics is not yet clear (Miller, 1993).

In humans prone to psychosis, a predisposition to such attentional disturbance is predicted by the hypothesis that dopamine overactivity is the basis of psychosis. However, this topic becomes complex because attentional dysfunction in the schizophrenic psychoses probably has more than one component. It is known that persons vulnerable to psychosis are often extremely sensitive to environments where several different channels of information compete (e.g., crowded rooms). However, the underlying vulnerability is not the same as the psychotic state itself, and the attentional disorder in patients, who are not at the time psychotic, is likely to have a basis unrelated to the dopaminergic factors. Experimental studies comparing attentional dysfunction during psychosis and after recovery from this state generally show a more severe disorder during the acute psychotic state. This has been shown by comparing acutely ill new admissions with the same patients after stabilization on neuroleptics (Maloney et al., 1976; Baruch et al., 1988). It has also been shown in chronic hospitalized patients, comparing those stably maintained on neuroleptics with those after drug withdrawal (Orzack et al., 1967; Kornetsky, 1972; Oltmanns et al., 1978). Attentional disorders are also more severe in schizophrenic patients with positive symptoms than in those with negative symptoms (Cornblatt et al., 1985). Thus, despite the fact that the attentional abnormalities are a trait variable in those prone to psychosis, there is good reason to relate the additional attentional problems which occur during the psychotic state itself to a state in which dopaminergic overactivity plays a major role. The animal studies referred to in the previous paragraph are likely to be a good model of the latter feature of psychosis.

In general terms, the attentional disturbance common to psychosis is a form of hypervigilance or some similar state characterized by greater than normal vulnerability to distraction. This state is different from the lapses of attention which occur during fatigue, when vigilance fails, and when brain functions are significantly impaired (e.g., by alcohol). The difference between the mechanisms producing such rather similar effects is a theoretical one. In distraction symptoms, attention deficits occur due to interference between two or more strong channels of information, leading to a deficient focus of attention; in contrast, in lapses of vigilance, attention fails because no channel of information carries cues which are salient enough to capture attention. The attentional dysfunction in the psychotic state is also different from that in ‘attention deficit disorder/hyperactivity syndrome’ found in children and adolescents because, in the latter syndrome, there is not the complementarity of symptoms due on one hand to loss of attentional focus and on the other to attentional intrusions. When disturbance of attention alone is rated, often no distinction is made between these two mechanisms.

As in animal studies, attentional problems specific to psychotic states can be described using the model of interference between two channels during information processing. One channel does the interfering and
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