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# A Rating Scale for Psychotic Symptoms (RSPS) Part I: theoretical principles and subscale 1: perception symptoms (illusions and hallucinations)

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**Abstract**

The authors present a new rating scale for the psychotic symptoms of schizophrenia and related psychoses. The scale links specific symptoms of psychopathology to dysfunction and overactivity of dopaminergic mechanisms underlying the processes of reward and selective attention. The Rating Scale for Psychotic Symptoms (RSPS) is a 44-item rating instrument with a seven-point severity scale for each item. Psychotic symptoms are classified into three groups: Pathological amplification of mental images (perception symptoms) (subscale 1), Distraction symptoms (including catatonia and passivity experiences) (subscale 2), and Delusions (subscale 3). A dimensional, rather than a categorical, conceptualization of psychosis is assumed. Rating is accomplished through a manual and a semi-structured interview (SSCI-RSPS). In this first of two papers, general issues about the construction of the scale and the derivation of symptom groups are discussed. Dopamine-mediated modification of cortico-striatal synapses is seen as being of critical importance in all three groups of symptoms. In this first paper, we present subscale I (perception symptoms), which includes both amplified perceptual images (illusions) and hallucinations. A total of seven illusions and 11 hallucinations are rated as individual items. 

*Keywords:* Dopamine; Hallucination; Mania; Positive symptoms; Psychosis; Rating scale; Schizoaffective disorder; Schizophrenia; Synaptic modification

## 1. Introduction

An essential starting point in the development of an objective quantitative rating scale for a psychiatric syndrome is the creation of a qualitative and descriptive classification system. A fundamental question to be asked of such a system concerns the conceptual status of the variables it measures. Their validity is

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open to question until it has been established that the items of the descriptive system, as well as their interrelationships, are founded on a coherent set of scientific concepts.

There are two possible approaches for achieving this aim: one theoretical and the other empirical. The theoretical approach involves constructing explanatory arguments that link the psychological phenomena documented at the ''molar'' or ''whole person'' level to processes in the brain at the ''molecular'' or neuronal level. In this approach, scientific reasoning, which crosses between levels of description, is required. There are several examples of such reasoning in the physical sciences, and some in biology and general medicine. However, in psychology and psychiatry, this approach to validating concepts of normal or abnormal functions has not yet been adopted. Historically, it has been generally accepted that the complexity of the intact brain and its resultant psychological functions is such that ''across level'' explanatory arguments are not yet possible.

Therefore, an alternative empirically-based strategy has been developed for defining concepts of function in psychology and psychiatry. This process has involved devising multi-item rating scales for documenting those features, which are immediately seen by the psychologist or psychiatrist, and then establishing inter-rater reliability and collecting data from a large number of human subjects. When large data sets are available, they are analysed by processes such as factor analysis or cluster analysis, with the aim of identifying those variables, whose measures tend to be correlated across subjects. The factors or clusters so obtained are then regarded as preliminary concepts allowing a more economical description of the phenomena under study than was possible with the original multi-item scales. The validity of these preliminary concepts can be examined further by exploring whether external variables correlate with the factor scores in a manner that respects the different factors. Such concepts rely mainly on the statistics of clusters and correlations, rather than on causal reasoning.

There is merit in both methods of validating scientific concepts, and the fact that the second rather than the first of the above has been used for deriving psychiatric rating scales reflects more uncertainty about how to proceed with a theoretically-based system, rather than fundamental opposition to such a project. However, there has been a rapid growth of neuroscience knowledge, and, within the basic sciences of brain and behavior, cross-level explanatory arguments are becoming more common and more rigorous. The time may therefore be appropriate for introducing such explanatory arguments within psychiatry, and for basing rating scales upon them. In fact, if mental disorders are disorders of the brain, there are strong reasons for basing systems for description, measurement and diagnosis of mental disorders on concepts that have theoretical validity deriving from basic neuroscience. In particular, such systems are likely to embody the key variables measured in biological studies more accurately than do empirically-based ones, and thus may be more valuable for biological and pharmacological research of mental illness.

Construction of cross-level theories in psychiatry is still prone to error. Therefore, theoretically based rating scales require the support of empirically based rating scales, and vice versa. The system we present is not meant to undermine the empirically based systems of description of psychosis, but rather to supplement them. We anticipate that revision of both sorts of scale will be required before they become congruent. When this happens, however, we will have rating scales that are based on the best available neuroscience concepts and are compatible with empirical evidence both at the biological level and at the level of psychiatric phenomenology.

At present, several empirically based systems are in use for the rating of symptoms of psychosis. However, these are not specific to psychosis nor to schizophrenia, having been drawn from descriptive systems applicable to a wide range of psychiatric symptoms. The most widely used rating scales for schizophrenia have been the Brief Psychiatric Rating Scale (BPRS) (Overall and Gorman 1962) and the Inpatient Multidimensional Psychiatric Scale (IMPS) (Lorr et al., 1963; Lorr and Klett 1966). These scales were originally designed to rate psychiatric symptoms along three axes: (1) anxiety, (2) depression, and (3) psychosis. The BPRS itself has four items related to psychotic positive symptoms (conceptual

disorganization, suspiciousness, unusual thought content, and grandiosity) and two items related to deficit symptoms (emotional withdrawal and blunted affect).

The Positive and Negative Symptoms Scale (PANSS) (Kay et al., 1987, 1988), now replacing the BPRS, includes the 18 items of the BPRS and 12 items from the Psychopathology Rating Schedule (Singh and Kay, 1975). The PANSS constitutes a major improvement over the BPRS. Although the same items as those that appeared in previous scales are used in the PANSS, they have all been redefined. The originality of the PANSS is the redefining of symptoms included in the BPRS and in descriptions of every score as an anchoring point for each item. The PANSS permits excellent interrater reliability and establishes the severity of symptoms according to the degree of functional impairment. However, since the majority of the items of the PANSS derive from the BPRS, it is not specific to psychosis. As a result, the majority of the symptoms rated are not primary psychotic symptoms but reflect the consequences (anxiety, depression, somatic concern, disorientation, etc.) of the schizophrenic or psychotic process.

An independent tradition for rating symptoms of schizophrenia is represented by the Scale for Assessment of Negative Symptoms (SANS: Andreasen, 1981), the Scale for Assessment of Positive Symptoms (SAPS: Andreasen, 1983) and the Scale for Assessment of Thought Language and Communication (TLC: Andreasen, 1979). The most relevant of these for the present work is the SAPS. It rates 33 items, under the main headings of hallucinations, delusions, bizarre behavior, and positive formal thought disorder. Data sets obtained with this system have been incorporated into factor analyses in several studies (e.g. Silver et al., 1993; Maziade et al., 1995; Basso et al., 1998). The SAPS has something in common with the scale that we present (RSPS). However, there are items included in the SAPS that we do not include (such as Positive Formal Thought Disorder), and many aspects of psychosis that are rated in the RSPS, but not covered in the SAPS. In addition, a fundamental difference is that the SAPS originates from traditional clinical description, whereas the RSPS is derived from an underlying psychobiological theory (Miller 1984, 1987, 1993; Miller et al., 1990).

With regard to Positive Formal Thought Disorder, this is a positive symptom, found especially in schizophrenia. However, positive symptoms are not necessarily features of psychosis, and it is debatable whether positive formal thought disorder is a symptom of psychosis. Different scales for assessing thought disorder define it in differing ways. The definitions of positive thought disorder in the SAPS are derived from the TLC. Using that instrument, measures of positive thought disorder show little change in the transition from overt psychosis to the state of stable impairment after treatment (Andreasen and Grove, 1986; Mazumdar et al., 1995). Thus, whereas thought disorder according to other definitions may be prominent in many psychotic states, thought disorder as assessed with the SAPS is mainly an aspect of the ongoing trait abnormalities, found especially in schizophrenia. Those definitions of thought disorder that have been used commonly for rating the disordered thinking that occurs during psychosis [such as the Thought Disorder Index (Johnston and Holzman, 1979) or the Index of Bizarre and Idiosyncratic Thinking (Marengo et al., 1986)] are likely to have a statistical association with items rated in the RSPS but in our opinion are less directly related to underlying brain mechanisms than the items we include. For these reasons, thought disorder is not explicitly included in the RSPS, though there will undoubtedly be many areas of conceptual overlap between items in the RSPS and thought disorder such as can be detected during psychosis. In a similar manner, we regard the items of the SAPS included under ''Bizarre behavior'' as related only very indirectly to the brain mechanisms underlying psychosis, and so these are not included in the present scale.

The items rated in the RSPS, but not covered in the SAPS, derive directly from the theory for psychosis that we present. We derive passivity symptoms (Schneiderian symptoms) from attentional theory and its biological substrate. Therefore, all such symptoms are grouped together in a separate section of the RSPS, whereas in the SAPS, items in this area are to be found in the sections dealing both with hallucinations and delusions. The theory for the formation of delusions leads us to place some emphasis on the dynamic aspects of delusions, that is, the temporal aspects of formation, persistence and extinction of delusional

beliefs. Scales are devised in the RSPS for rating these features, and we are aware of no other scale that does this explicitly. Within the area of delusions, items are also grouped according to content, in so far as they imply similar underlying psychobiological disturbance.

It has not been possible to assess the conceptual validity of any of the previous scales (BPRS, IMPS, PANSS or SAPS) in terms of neurobiological concepts due to the lack of a theoretical system derived from psychobiology of psychosis (including that in schizophrenia). However, a theory of psychosis has been advanced in recent years (Miller 1984, 1987, 1993; Miller et al., 1990), based on the relation between the psychological functions of the neurotransmitter dopamine, as studied in animal experiments, and features of the psychotic phases in humans. The theoretical derivation of the symptoms of the psychotic phases, although incomplete, is based on two concepts in psychology: reward and attention. These concepts can be understood in biological as well as psychological terms. Psychopharmacological studies have shown that both reward and attention are related to the functioning of the neurotransmitter dopamine. The reward concept has also been expressed in terms of electrophysiological evidence (Wickens et al., 1995), the latter being also related to intracellular neurochemistry (Colwell and Levine, 1995).

It is not surprising that these two psychological concepts, once translated into the language of dopaminergic mechanisms, should provide a theoretical basis for rating psychotic symptoms. The dopamine (DA) hypothesis proposes that a relative overactivity of mesolimbic, mesocortical, or nigrostriatal dopaminergic neurons is present in schizophrenic patients, especially when they are acutely psychotic. It has been suggested that this increase relates to specific symptoms (i.e. delusions or hallucinations) or to specific mechanisms (i.e. attentional impairment) (Meltzer, 1987). A relation between dopaminergic neurotransmission and the most acute psychotic symptoms has long been recognized, based on the therapeutic effects of antipsychotic drugs such as the classical neuroleptics, which block DA receptors, and that indirect DA agonists such as amphetamine, which release dopamine in active form in the brain, can produce or exacerbate psychotic symptoms. The evidence for a DA disturbance in psychosis remains indirect. Although a consensus has yet to be reached on the exact nature of dopaminergic overactivity and the origin of psychotic symptoms, there are several promising lines of investigation that support the dopamine hypothesis (Seeman et al., 1993; Reith et al., 1994; Pearlson et al., 1995).

This paper provides a psychobiological theory of brain mechanisms from which a descriptive and classificatory system for psychotic symptoms has been derived. The new rating scale is designed for the quantitative assessment of a variety of psychotic symptoms, defined and grouped according to the premises of this theory. However, before describing the Rating Scale for Psychotic Symptoms (RSPS), a summary of the neurological principles believed to underlie each major grouping of symptoms will be presented.

## 2. General principles underlying the RSPS

The focus of the scale is the primary symptoms of psychosis that dominate the acute stages of psychosis in schizophrenia and related disorders. These psychotic symptoms are classified into three groups: pathological amplification of mental images (perception symptoms) (subscale I), distraction symptoms (including catatonia and passivity experiences) (subscale II), and delusions (subscale III). These symptoms have been associated mainly with the psychotic phases of schizophrenic illnesses but also occur in other related illnesses such as schizoaffective, bipolar disorders and other psychotic disorders.

Whenever possible, the grouping of symptoms and their rating follow neurological principles. For example, for illusional perceptions (subscale IA) and hallucinations (subscale IB), separate items refer to disturbances in information processing with respect to separate sensory systems that are located in different regions of the cerebral cortex (or other brain structures). Similarly, amongst the passivity experiences (distraction symptoms, subscale IIB), we characterize the effects of cognitive interference or distraction

on different response systems (e.g. general bodily movements vs. speech). For the symptoms of delusions, we apply neuropsychological concepts derived from the study of normal memory (formation, persistence and extinction) to the abnormal configurations of information that delusions constitute.

In the RSPS, we also allow the rating of the region of sensory space (i.e. right/left) where an hallucination is experienced. In this context, it is known that lateralization of psychological functions is a significant factor in schizophrenia (e.g. Flor-Henry, 1969, 1983; Gruzelier, 1991), and it is likely that this will be one of the influences determining the form of psychotic symptoms (Flor-Henry, 1983; Bracha et al., 1985; McGilchrist and Cutting, 1995; Nayani and David, 1996). Attempts have been made to show the theoretical links between psychotic symptoms and lateralization of psychological functions at this stage of the illness (Flor-Henry, 1983). While these endeavors are incomplete at present, we think that lateralization of symptoms should be included in some of the items below, and we tentatively identify the side and location in the hemispheres having a particular relation to specific symptoms.

The strategy outlined in the previous two paragraphs presumes that the etiology of symptoms can be theoretically derived from a knowledge of brain mechanisms and their dynamic disturbance. We envisage that this approach to psychotic symptoms will make the RSPS particularly useful in brain scanning studies and clinical trials.

Although our proposed classification of symptoms has a neurological basis, its principles are more dynamic than for most neurological disorders. This is appropriate because psychotic symptoms can fluctuate rapidly, even second by second, and can be intermittent like epileptic symptoms. Moreover, some symptoms (i.e. delusions) may be present for a long period of time, yet have definable dynamic aspects to their initial formation and gradual disappearance. We propose that there are neuroprocesses, corresponding to symptoms, that show rapid or more gradual dynamic fluctuations and may be measurable by brain scanning or modern EEG analyses. Therefore, the RSPS includes a rating of the temporal aspects of the formation and disappearance of persistent symptoms.

While our approach is one of ''dynamic neurology'', our strategy is also to relate psychotic symptoms as far as possible to normal brain mechanisms and normal psychology. For this reason, we favor a dimensional rather than a categorical conceptualization of the factors characteristic of a given syndrome. Thus, the rating of the level of psychopathology for each item begins with the prepsychotic level of each symptom, which describes phenomena that may sometimes occur in normal persons. By emphasizing the relation between normal and psychotic psychology, we aim to present a more coherent account of psychosis.

Many details of the RSPS, including the progression from prepsychotic experiences to psychotic symptoms, which is implicit in our scale, reflect a theoretical perspective that we adopt, based on brain mechanisms. It is possible that the hypotheses about mechanisms are incorrect or incomplete in some respects. This is an empirical question to be addressed by use of the scale in its present form. This descriptive scheme, the RSPS, should thus be viewed as a prototype for a theoretically based rating scale for the specific and primary symptoms of psychosis in schizophrenia and related psychotic disorders. In the same way, the PANSS can be viewed as the third version of a continually modified scale for psychotic symptoms (whose antecedents were the IMPS and BPRS).

The negative symptoms of schizophrenia characteristic of the prodromal or chronic phases of illness are less specific to schizophrenia than the positive symptoms (Klosterkotter et al., 1995) and more diverse in origin. Some of them may have a derivation from an underlying structural disturbance of the brain (e.g. increased ventricular size and cortical atrophy) not directly related to the acute psychotic disease process. Others, however, may be secondary consequences of adverse psycho-social sequelae of the illness rather than due to the illness itself. For instance, a parallel has been drawn between the negative symptoms of schizophrenia and posttraumatic stress disorder as seen in war veterans (McGorry et al., 1991). Some negative symptoms may be related to extrapyramidal side-effects of medication. Moreover, cognitive impairment, seen in chronic psychotic illnesses, may arise from neuropathological processes different from

those at work in the psychotic phases. For instance, cognitive deficits are reported to be worse in chronic than in first-episode cases (Bilder et al., 1992), and in some patient groups, there is an association between cognitive deterioration and appearance of tardive dyskinesia (Waddington et al., 1990; Davis et al., 1992). Thus, many of the non-psychotic symptoms of schizophrenia are either non-specific or not directly related to the fundamental disease process underlying the psychosis itself. Due to the complexity and lack of specificity of these symptoms, it becomes more difficult to derive them in a logical fashion from a psychobiological theory. Therefore, in providing a rating scale for specific, primary symptoms of psychosis in schizophrenia and related illnesses, we focus on degrees of severity of positive symptoms during the psychotic phase, occurring as precursors of this phase, or present in its immediate aftermath. Some of the negative symptoms may, however, be derivable from neurological principles, and in the future, we hope to develop a scale for such symptoms to complement the RSPS.

## 3. Overall conceptual issues

The fundamental biological process believed to underlie all the symptoms rated below is the action of dopamine as a signal that reinforces certain components of neural activity and corresponding aspects of thought or behavior. In animal psychopharmacology, this role for dopamine is well known from studies of instrumental conditioning and related processes, where it is referred to as the reward or incentive function. A variety of specific behavioral paradigms have been devised for evaluating the process by which sensory stimuli are made more attractive, programs for behavioral acts are reinforced, or the two processes occur in combination [reviewed by Beninger (1983), Miller et al. (1990), Beninger (1992) and Miller (1993)].

From the available literature, we know that reward mediates a class of learning processes. It is generally assumed that the biological basis of learning is the modification (strengthening or weakening) of selected synapses. We propose that the role of dopamine as a rewarding influence at the ''molar'' level corresponds at the ''molecular'' level to a type of synaptic strengthening in which dopamine is an essential catalyst. The best-known paradigm for synaptic strengthening is Hebb's rule, where strengthening is dependent on a coincidence of activity at the level of a single neuron between the presynaptic and the postsynaptic side of a synaptic junction (Hebb 1949). In earlier works, Miller (1981, 1988) and Wickens (1988) suggested that reward-mediated learning at the behavioral level requires a more complex rule for synaptic change than Hebb's Rule, in which a dopaminergic ''reward'' signal controlled by macroscopic events, as well as factors local to each neuron, is needed to achieve the strengthening of a synapse. The most likely population of synapses that might be subject to such a dopamine-dependent modification are the cortico-striatal synapses, composed of terminals of axons projecting from the cerebral cortex upon the dendritic spines of the principal neurons of the striatum. The hypothesis that cortico-striatal synapses are subject to dopamine-dependent synaptic change has recently received empirical support from Wickens et al. (1995). It was reported that responses in striatal neurons to stimulation of the cortex were enhanced if dopamine was applied as a brief pulse at the time of the response. Conjunction of presynaptic and postsynaptic activity alone, in this class of synapses, was insufficient for synaptic strengthening to occur in this pathway.

The relevance of this variety of synaptic change for an understanding of psychotic symptoms is clear from the evidence relating psychosis to dopamine. The indirect evidence, based on the beneficial action of dopamine-blocking, antipsychotic drugs and the psychotogenic effects of dopamine-releasing drugs such as amphetamine, has already been mentioned. The search for direct evidence has followed two avenues: the hypothesis of an excess of dopamine receptors in psychosis-prone subjects and the hypothesis of excessive release of dopamine in such subjects. Most work has concentrated on the former of these two possibilities (Seeman et al., 1993; Pearlson et al., 1995). A recent study investigated the second of these possibilities and has shown, using positron emission tomography, an excess conversion of L-DOPA to dopamine in actively psychotic patients (Reith et al., 1994). To understand the theoretical basis of the RSPS, it is not necessary to be specific about which of these two possible causes of dopamine overactivity

applies. That some variety of dopamine overactivity underlies psychosis is, however, a necessary premise for the theoretical understanding of psychotic symptoms as presented below.

The psychotic symptoms are classified into three groups: pathological amplification of mental images (perception symptoms) (subscale I), distraction effects (including catatonia, passivity experience and some other symptoms) (subscale II), and delusions (subscale III). The first two of these are momentary or intermittent experiences that occur at any specific point in time. These experiences may be remembered, but the symptoms themselves do not persist over time (even though, in severe illness, they may occur so frequently during the day as to be more or less continuous). The delusions, however, are persistent beliefs. In this case, it may be possible to document the exact time at which such a symptom first appears; but once it has appeared, it endures as a symptom for a period lasting from days to years. Momentary experiences can be combined with longstanding delusional elaborations.

Both momentary abnormal experiences and enduring delusions recruit learning and memory globally, but in different ways. A memory of a momentary experience in the recent or distant past does not constitute a re-enactment of the experience since that experience is known to have occurred at a discrete time in the past. However, a belief, whether normal or delusional, may lie dormant for long periods but can be completely reactivated at any time. The initial experiences through which the belief came to be formed and internalized may not even be remembered because they are not easily distinguished from the periodic reactivations of those experiences. As a result, a particular configuration of information comes to be an enduring part of a person's belief system rather than being seen as an experience that ''happened'' to that individual at some discrete time in the past. Broadly speaking, the difference between the memory of a momentary experience and an enduring delusional belief in psychopathology is similar to that between episodic and semantic memory in normal cognitive psychology. There is evidence that episodic memory involves the frontal lobes to a significant degree, whereas semantic memory does not (Tulving, 1989). This may be particularly relevant to the distinction between momentary experiences of psychosis versus delusional beliefs.

In terms of brain dynamics, a dopamine-dependent synaptic change in the striatum is presumed to underlie both abnormal momentary experiences and enduring delusions. The fact that both types of symptom can recruit long-term memory is taken to indicate that synaptic change, occurring in the striatum, influences patterns of dynamic activity throughout the cerebral cortex. This is plausible because outflow pathways from the striatum are known to project to the cerebral cortex (via a number of intervening structures, such as globus pallidus or pars reticulata of the substantia nigra, and the ''motor'' thalamus) (Alexander et al., 1986; Joel and Weiner, 1994). However, the way in which cerebral cortical activity is ''turned on'' is envisaged to be quite different for momentary experiences as compared to enduring beliefs.

In the case of momentary and intermittent experiences, signals transmitted from striatum to cortex are believed to inscribe or leave a trace on the latter structure in the form of patterns of modified synapses that correspond to the formation of a memory of the experiential episode. In the case of delusional beliefs, this also happens. However, each assembly of cortical cells so activated is envisaged to contain elements that project back to the striatum. Thus, there is a potential connectional loop from striatum, via the pallidum and thalamus, to the cortex and back to the striatum (Alexander et al., 1986; Joel and Weiner, 1994). If suitably triggered, such a loop is capable of maintaining a state of elevated circulating activity. The theory for the formation and operation of such loops, designated as ''cortico-striatal cell assemblies'' is described by Miller and Wickens (1991). The effect of activity in such a configuration is that a belief can be reactivated with a vividness comparable to that at the time of its formation.

Having established this broad distinction between momentary psychotic experiences and enduring delusional beliefs, we will consider the derivation of various types of psychotic symptom in each class. The momentary psychotic experiences, to be discussed first, fall into two types, those involving the pathological amplification of mental images (subscale I) and those involving distraction effects (subscale II).

## 4. Amplification of mental images: perception symptoms

### *4.1. Theoretical basis*

The concept underlying this group of symptoms applies to the amplification of perceptions produced in response to external stimuli (illusions) and to mental images generated internally rather than in response to an external stimulus (hallucinations). The latter can be referred to generically as "thoughts". Use of the word, as an internally generated mental image, is somewhat wider than its usual use. It applies to the full range of internally generated mental images. Most people have only a limited subjective awareness of many of these images until they become amplified by mental illness. Our use of the word "thought" is a logical extension of its everyday use, given the common mechanism we envisage for amplification of a wide variety of internal mental images. Thus, amplification, both of perceptions and of thoughts, is envisaged to rely on the same mechanisms, namely, the strengthening, under the influence of dopamine, of synapses in the striatum activated from the cerebral cortex.

Parallels of such symptoms have been identified in animals in cases where modifications in the activity of the dopamine system have occurred (Miller and Beninger, 1991). For instance, when rats with unilateral lesions of the midbrain dopamine neurons are injected with dopamine agonists, the animals move around in circles. While several factors contribute to this effect, the heightened attractiveness of sensory stimuli contralateral to the striatum with the greatest dopamine activity appears to be one of the factors responsible (Miller and Beninger, 1991). In unlesioned animals given dopamine antagonists, such heightened sensory attractiveness likewise appears to be a factor in the stereotyped behavior that these animals display (Miller and Beninger, 1991). An effect in the opposite direction is also well known: animals depleted of striatal dopamine exhibit a pronounced multisensory unresponsiveness to stimuli delivered contralateral to the depleted striatum. Such unresponsiveness, far from being a symptom of psychosis in humans, can be viewed as a pathology in the opposite direction (i.e. negative symptoms such as apathy and anhedonia, subjectively a reduction in the amplification of mental images) occurring due to a reduction, rather than an enhancement, of dopaminergic activity (Chouinard and Jones 1978, 1979).

The reason for distinguishing between symptoms due to amplification of perceptions and those due to amplification of thoughts is not because the mechanism is categorically different, but because this distinction corresponds to traditional classifications of symptoms of psychosis. Amplification of thoughts is regarded as the psychological process underlying the production of hallucinations. When purely internal mental images are greatly amplified, we propose that they are experienced not as being internally produced but as a subjective response to real external stimuli. We distinguish this from symptoms due to the amplification of perceptions of actual stimuli, which are described in this paper as illusions. Although the basic process is the same as for hallucinations, illusional amplification of perceptions does not produce an equally dramatic and categorical departure from normal sensory experience and, therefore, is often not included amongst the symptoms of psychosis. However, accounts of the prodromes to psychosis, or the early stages of psychosis, commonly mention such symptoms (e.g. Bowers, 1974). The fact that they are not often noted during the height of a psychotic episode may be because they are then overshadowed by the dramatic symptoms of the more floridly psychotic state. An important reason for wanting to rate the symptoms of amplified illusional perceptions, as well as hallucinations, is that we suspect that brain scanning or EEG mapping studies may be able to demonstrate the involvement of different brain regions in the case of perceptual amplification as compared to those involved in hallucinations. Specifically, we anticipate that amplified illusional perceptions will be associated with activation of primary sensory or unimodal sensory association areas of cortex, or of the putamen (the sensorimotor part of the striatum), whereas hallucinations will be associated with additional activation of cognitive regions of the brain (such as prefrontal cortex, and the caudate nucleus).

Symptoms in subscale I are all subjective experiences, whether or not they are triggered by an external

stimulus, which have increased intensity, impact or vividness for the patient. They are subdivided into "illusional amplification of perception" (IAP) and hallucinations (H). The former of these concepts has a relation to traditional definitions of an illusion. Classically, an "illusion" is defined as a misperception or misinterpretation of a real external sensory stimulus (Kaplan et al., 1994). The concept that we refer to as an "illusional amplification of perception" is defined as an amplification of the subjective response to a real stimulus. This amplification manifests itself as an increase in the vividness, attractiveness or memorability of the stimulus. It is these features that lead, in psychosis, to the misperception or misinterpretation referred to in the classical definition. An hallucination is classically defined as a false sensory perception not associated with a real external stimulus (Kaplan et al., 1994). Our definition attempts to indicate an underlying mechanism and requires us first to define a "thought" as a mental image without an explicit, external stimulus. An "hallucination" can, therefore, be defined as the amplification of a thought to the point where it seems no longer like a thought, but more like a subjective response to a real stimulus. Since the subjective response is then a perception with an origin different from normal (i.e. not associated with a real external stimulus), it is a false perception, as in the classical definition.

Symptoms that are produced by the amplification of mental images (whether perceptions or thoughts) may be differentiated according to their empirical manifestation and presumed etiology. The cortex and striatum both appear to function as repeated sets of information-processing subunits. The logical operation performed in each subunit of one of these structures is assumed to be the same, but since inputs and outputs are different, so too would the psychological functions of each subunit be different. Thus, the symptoms identified in subscale I, which are all produced by the same mechanism, have different but analogous psychopathologies: when dopamine is overactive, different symptoms are generated, corresponding to the excessive influence of different information systems, all relayed through the striatum. Specifically, they are believed to be produced in different processing subunits in the striatum, in the cortex, or in different circuits including corresponding processing subunits in the cortex and the striatum. In theory, the subdivision of symptoms that we propose in subscale I could be carried out in as much detail as can the regional division/localization of function in the cortex. However, the items that we rate in subscale I are those that we consider to occur most often and are most easily identified.

The information systems involved in formation of amplified perceptions include any sensory system. In subscale 1A, we include visual, auditory, olfactory, gustatory, tactile (haptic), co-enesthetic and kinesthetic systems. Amplified tactile perceptions arise from the external surface, i.e. from the skin or under the skin. Amplified co-enesthetic perceptions arise from within the body. The details of the types of sensory experiences in each sensory system which, when amplified, may lead to symptoms are given in the schedule for the semi-structured interview. We also include the item "amplification of perception of coincidence". While this symptom is not directly related to a single sensory system, it is a common symptom in which perception of the stimuli in general is distorted. When this symptom is present, there may be increased significance attributed to the concurrence of external events that occur together only once. Alternatively, increased significance is given to the occurrence of an external event immediately after a movement, action or thought of the patient, this conjunction again occurring only once.

Persons vulnerable to psychosis may be particularly sensitive to sensory stimuli (for example sounds) when they are in complete remission. This is rated at a level of 2 for isolated symptoms on the items for illusions. However, we acknowledge the possibility that such abnormal sensitivity to sensory stimuli seen in remitted patients may have a different basis in brain mechanisms from the illusions occurring in the same patients when they are becoming psychotic. This is one of the areas where theory development is as yet incomplete.

In the case of hallucinations, we subdivide those in the auditory domain because these are recognized to be the most common, and the subdivision may have significance with respect to brain location or laterality. The thoughts from which auditory hallucinations arise are thus designated as verbal, musical, or other non-verbal auditory thoughts. Auditory hallucinations are divided into three types corresponding to these three items. Each type is assumed to be associated with a different brain region or laterality: verbal thoughts with predominantly dominant, superior, temporal regions; musical thoughts with right

temporal regions; and other non-verbal auditory thoughts with non-dominant temporal regions (Flor-Henry, 1983, 1990). We include visual images without external stimuli as "visual thoughts", which are proposed as the basis for visual hallucinations. We divide visual hallucinations into two items: simple, elementary visual thoughts associated with bilateral (right and left) occipital regions, and complex visual thoughts associated with either bi-temporal or right hemispheric regions (Flor-Henry, 1983, 1990). Also, we include olfactory (frontal limbic with right predominance), gustatory (bilateral temporal mesolimbic/uncus), tactile (haptic), co-enesthetic (bilateral, postero-parietal with right predominance), kinesthetic or vestibular images without external stimuli as the basis for corresponding hallucinations. Details of the hallucinatory experiences that may arise in each of these information channels are given in SSCI-RSPS.

## 5. Rating Scale for Psychotic Symptoms (RSPS). I = illusions, H = hallucinations

### *5.1. Subscale 1: Pathological amplification of mental images*

#### *5.1.1. 1A: Illusional amplification of perception (IAP)*

$I_1$. Amplified visual perceptions. Features: colours, shapes, faces, persons, animals, scenes, pictures, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Absent | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_2$. Amplified auditory perceptions. Features: noises, musical sounds, other recognizable sounds, voices, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_3$. Amplified olfactory perceptions. Features: food cooking, car exhaust, smoke, pollution smells, odours, perfumes, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_4$. Amplified gustatory perceptions. Features: tastes, flavors, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_5$. Amplified co-enesthetic perceptions. Features: touch, pain, itchiness, aches, numbness, awareness of heart beat, hot flushes, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_6$. Amplified tactile perceptions. Features: sensation from clothes, touching by another person (which may be the subject of immediate delusional elaboration), etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_7$. Amplified kinesthetic perceptions. Features: walking, jogging, eating, washing, playing a musical instrument, etc.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Clearly present, mildly amplified and vivid, awareness of the abnormality | | 2 | 3 | 4 |
| Strikingly amplified and vivid, or involving more than one feature mentioned above | | 3 | 4 | 5 |
| Disturbingly amplified and involving more than one feature, or delusional elaboration | | 4 | 5 | 6 |

$I_8$. Amplification in perception of coincidence [increased significance attributed to either: (1) concurrence of external events that occur together only once; or (2) occurrence of an external event immediately after a movement, action or thought of the patient, with this conjunction occurring only once].

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Coincidences seem to be happening more than normal; patient is uncertain whether this is real or not | | 2 | 3 | 4 |
| Coincidences are definitely more significant than normal; actions and thoughts definitely have unusual consequences | | 3 | 4 | 5 |
| All events in the world appear to be causally related to each other. Actions and thoughts all appear to be controlling events in the world | | 4 | 5 | 6 |

### 5.1.2. 1B: Hallucinatory amplification of thoughts (H)

$H_1$. Amplification of verbal thoughts [one's own verbal thoughts more active than normal, hearing one's own voice talking when one is not speaking, hearing external voices when no one is speaking, voice of an identifiable person (patient's doctor, parents, etc.), the voice of God, attractive voices, threatening voices, voices talking about oneself, voices telling one to do something, several voices at once].

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupation with verbal thoughts, or verbal thoughts more active than usual (but still identified as one's thoughts) | | 2 | 3 | 4 |
| Definite external voices, or one's own voice (when one or no one is speaking), including simple comments, or faint or unidentifiable voices | | 3 | 4 | 5 |
| Disturbing, or emotionally intense voices, or several voices at once (a babble), or elaborated voices (e.g. identifiable voices), or delusional identification of voices | | 4 | 5 | 6 |

$H_2$. Amplification of musical thoughts (songs, tunes, rhythms).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupation with musical thoughts, or musical thoughts more active than usual (but still identified as one's thoughts) | | 2 | 3 | 4 |
| Music heard as if playing outside of one's head, or inside one's head | | 3 | 4 | 5 |
| Many musical sounds at once, or recognizable musical form (e.g. tune plus rhythm or harmony), emotionally intense or delusional identification of type of music | | 4 | 5 | 6 |

$H_3$. Amplification of other non-verbal auditory thoughts (noises, buzzing, ringing, hisses, whistles, tinnitus, whirring, etc.). Exclude noises inside one's head arising from peripheral lesions of the auditory system.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with sounds; may experience sounds during silence and is unsure whether they really exist | | 2 | 3 | 4 |
| Hearing distinct external sounds or noises, or noises "inside one's head", when thereis no stimulus | | 3 | 4 | 5 |
| Hearing noises which are familiar and identifiable, or of greater complexity than usual, or strange, emotionally intense or with delusional identification, but with no identifiable stimulus | | 4 | 5 | 6 |

$H_4$. Amplification of simple, elementary, visual, thoughts (patterns, points, shadows, clouds, patches of colour, flashes of light). Exclude unusual visual images arising from peripheral lesions of the visual system.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Definite but indistinct simple images seen when eyes closed; or, if eyes are open, images which patient suspects have no basis | | 2 | 3 | 4 |
| Distinct simple images seen with eyes open, patterns, shades, colours, which are simple and emotionally neutral, and definitely no external stimulus | | 3 | 4 | 5 |
| Recognizable patterns, patches of colour seen with eyes open, which are emotionally intense, or with delusional identification, and definitely no external stimulus | | 4 | 5 | 6 |

$H_5$. Amplification of complex visual thoughts (faces, angels, devils, Jesus, the Virgin Mary, animals, insects, skulls, flying saucers, Martians, monsters, ghosts, etc.).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Definite but indistinct complex images seen when eyes closed; or, if eyes are open, images which patient suspects have no basis | | 2 | 3 | 4 |
| Distinct complex images seen with eyes open, recognizable objects, faces or persons, if simple and emotionally neutral, and definitely no external stimulus | | 3 | 4 | 5 |
| Recognizable objects, faces or persons seen with eyes open, which are complex or emotionally intense, or with delusional identification, and definitely no external stimulus | | 4 | 5 | 6 |

$H_6$. Amplification of olfactory thoughts (smells, odors, perfumes, gas, garbage, animal smells, etc.).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with smells; or definite but indistinct smells, and is unsure whether they really exist | | 2 | 3 | 4 |
| Distinct or recognizable smells, which are simple and emotionally neutral, and definitely no external stimulus | | 3 | 4 | 5 |
| Recognizable smells, which are complex or emotionally intense, or subject to delusional elaboration, and definitely no external stimulus | | 4 | 5 | 6 |

$H_7$. Amplification of gustatory thoughts (tastes, flavors, etc.).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with taste, awareness of unusual tastes, but unsure whether they are real | | 2 | 3 | 4 |
| Distinct or recognizable tastes with nothing in mouth to produce them | | 3 | 4 | 5 |
| Recognizable tastes, which are emotionally intense and distinctly unpleasant or pleasant or subject to delusional elaboration, with nothing in mouth to produce them | | 4 | 5 | 6 |

$H_8$. Amplification of co-enesthetic thoughts (changes in body image, size or shape of body parts, or more often internal feelings for which a stimulus cannot be identified). These symptoms (co-enesthetic hallucinations) are distortions of the idea of body image, rather than of perceptions due to actual external somatic stimuli. The hallucination that there is an object within the body or head (rated 4, 5, 6) is often subject to immediate delusional elaboration (e.g. identified as a microphone, snake, etc.).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with body image, or internal sensations; strange internal sensations, uncertain whether they are real | | 2 | 3 | 4 |
| Definite abnormal internal sensations, or sensations of changes in size or shape of body parts | | 3 | 4 | 5 |
| Complex internal sensations, including some object inside or on the surface of the body, emotionally intense or subject to delusional elaboration | | 4 | 5 | 6 |

$H_9$. Amplified tactile thoughts (sensations of touch, burning, biting, or of something moving or crawling on or under the skin (parasites, snakes, etc.) for which no stimulus can be identified. May be subject to immediate delusional elaboration.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with sensation of touch and uncertain whether tactile sensations are real | | 2 | 3 | 4 |
| Definite abnormal sensations of touch, although there is no stimulus that can cause them | | 3 | 4 | 5 |
| Abnormal tactile sensations that are complex or recognizable, emotionally intense or subject to delusional elaboration | | 4 | 5 | 6 |

$H_{10}$. Amplified kinesthetic thoughts (sensations of movement of body parts when they are stationary). Hallucination of a specific movement of a sporting activity which is an example of a complex and recognizable movement that is rated at a minimum of 4.

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with body movement; strange sensations of movement of body parts still identified as one's thoughts, but unsure whether they really exist | | 2 | 3 | 4 |
| Definite abnormal sensations of movement of body parts, not complex, although one can see that they are stationary | | 3 | 4 | 5 |
| Abnormal sensations of movement of body parts that are complex or recognizable, emotionally intense or subject to delusional elaboration | | 4 | 5 | 6 |

$H_{11}$. Amplified vestibular thoughts (sensations of movement of whole body, of balancing, or of loss of balance). It is assumed that the sensation of whole-body movement arises mainly from hallucinations in the vestibular sense (i.e. from the organs of balance).

| | | Occasional | Frequent | Almost continual |
|---|---|---|---|---|
| Normal | 0 | | | |
| Borderline | 1 | | | |
| Preoccupied with whole-body movement or balance; strange sensations of whole-body movement or balance, still identified as one's thoughts, but unsure whether they really exist | | 2 | 3 | 4 |
| Definite abnormal sensations of whole-body movement, or balance, or falling, not complex, without identifiable causes | | 3 | 4 | 5 |
| Sensations of movement of the whole body that are complex, emotionally intense or recognizable, or subject to a delusional elaboration | | 4 | 5 | 6 |

## 6. Semi-Structured Clinical Interview (SSCI-RSPS)—Part I

### *6.1. Subscale 1: Pathological amplification of mental images*

#### *6.1.1. 1A: Illusional amplification of perception (illusion)*

1. Amplified visual perceptions (colours, shapes, faces, persons, animals, scenes, pictures, etc.).
Questions:
- Are colours brighter than usual?
- Do you see things more clearly than normal?
- Are shapes more interesting than usual?
- Are faces more distinctive than normal?
- For any of these four, ask: Was it pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

2. Amplified auditory perceptions (noises, musical sounds, other recognizable sounds, voices, etc.).
Questions:
- Are noises louder than usual?
- Is music more enjoyable than normal?
- Are sounds more distinctive than normal?
- Are voices more distinctive than normal?
- For any of these four, ask: Was it pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

3. Amplified olfactory perceptions (food cooking, car exhaust, smoke, pollution smells, odors, perfumes, etc.).
Questions:
- Are smells stronger than normal?
- Are you more affected by cigarette smoke than normal?
- Does food cooking smell more appealing than normal?
- For any of these, ask: Was it pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

4. Amplified gustatory perceptions (tastes, flavors, etc.).
Questions:
- Are some or all tastes more intense than usual? Which ones?
- Do you like some tastes more or less than before?
- For any of these, ask: Was it pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

5. Amplified co-enesthetic perceptions (touch, pain, itchiness, aches, numbness, awareness of heart beat, hot flushes, etc.).
Questions:
- Do you feel increased body sensations?
- Do you have exaggerated feelings of pain or aches?
- Do you have unusual bodily sensations?
- For any of these, ask: Was it pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

6. Amplification of tactile perceptions [sensation from clothes, touching by another person (which may be subject to immediate delusional elaboration)].

Questions:

- Is your sense of touch exaggerated?
- Are you more sensitive than normal when someone else touches you?
- For any of these, ask: Was this pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

7. Amplification in kinesthetic perceptions (walking, jogging, eating, washing, playing a musical instrument, etc.)

Questions:

- Are you more aware of your body movements than normal, for instance in walking, eating, washing, etc.?
- Does your sense of movement or do parts of your body seem different from normal?
- For any of these, ask: Was this pleasant? Was it striking? Was it disturbing?
- How many times per day or per week does this happen?

8. Amplification in perception of coincidence (increased significance attributed to either: concurrence of external events that occur together only once; or occurrence of an external event immediately after a movement, action or thought of the patient).

Questions:

- Have you been aware of any unusual coincidences in the last few days?
- Have you been aware of any strange consequences of your actions, or of your thoughts?
- Do you think that these things are just coincidence?
- Might they have some significance?
- Do you feel that your thoughts are controlling events in the world?
- How many times per day or per week does this happen?

*6.1.2. 1B: Hallucinatory amplification of thoughts (hallucination)*

1. Amplification of verbal thoughts [one's own verbal thoughts more active than normal, hearing one's own voice talking when one is not speaking, hearing external voices when no one is speaking, voice of an identifiable person (patient's doctor, parents, etc.), the voice of God, attractive voices, threatening voices, voices talking about oneself, voices telling one to do something, several voices at once].

Questions:

- Do you spend more time thinking than usual?
- Are you more preoccupied with your thoughts than normal?
- Are your thoughts more active than usual?
- Do sentences sometimes go through your mind?
- Are sentences going through your mind more intensely than normal?
- Do you feel as if you are hearing voices?
- Are you sure about this?
- Do you hear a voice telling you what to do?
- Can you say who is speaking? Your doctor? Your father/mother? God/the devil? Man/woman? (etc.)
- Are they nice voices? Or bad voices?
- Are they threatening you?
- Are they saying bad or good things about you?
- Are they telling you to do bad things (e.g. killing yourself, or other people)?
- How many voices do you hear? Are they all speaking at once?

- Do you hear these things mainly on only one side? Left or right? (please circle R/L)
- How many times per day or per week does this happen?

2. Amplification of musical thoughts (songs, tunes, rhythms).

Questions:

- Do you have tunes (songs, rhythms) running through your head?
- Is this happening more often than normal? Are the tunes (etc.) more vivid than normal?
- Do you actually hear music inside your head, or does it seem to be outside as if it was really being played somewhere?
- Do you hear these things mainly on only one side of your head? Left or right? (Please circle R/L)
- Do you have a special interest in music? (Professional performer/composer; educated non-professional; amateur)
- How many times per day or per week does this happen?

3. Amplification of other non-verbal, auditory thoughts (noises, buzzing, ringing, hisses, whistles, tinnitus, whirring, etc.).

Questions:

- Are there any sounds or noises running through your mind?
- Can you tell whether they come from outside, or are only in your mind?
- Can other people hear them too?
- Are they indistinct or clear?
- Can you recognize the sounds or noises you hear (e.g. telephone ringing or a bell ringing)?
- Could you describe these noises?
- Can you give me an example of what these noises sound like?
- Do you hear these things mainly on only one side? Right or left? (Please circle R/L)
- How many times per day or per week does this happen?

4. Amplification of simple, elementary, visual thoughts (patterns, points, shadows, clouds, patches of colour, flashes of light).

Questions:

- Do you see unusual things?
- What do you see?
- Do you see points of light, patterns on the wall, shadows, patches of colour, etc.?
- When you see these things, are your eyes open or closed?
- Do you see these things mainly on one side? Right or left? (Please circle R/L)
- How many times per day or per week does this happen?

5. Amplification of complex visual thoughts (faces, angels, devils, Jesus, the Virgin Mary, animals, insects, skulls, flying saucers, Martians, monsters, ghosts, etc.).

Questions:

- Do you see visions?
- Do you see things that other people cannot see?
- What do you see? Are they recognizable objects or faces?
- Do you see faces? Persons? Animals? Insects? Frightening things? Angels? God? etc.
- When you see these things, are your eyes closed or open?
- Do you see these things mainly on only one side? Right or left?
- How many times per day or per week does this happen?

6. Amplification of olfactory thoughts (smells, odors, perfumes, gas, garbage, animal smells, etc.).
Questions:

- Are you aware of smelling any strange smells?
- Are you sure there is really something causing them?
- Do you smell things that other people cannot smell?
- Are you bothered by these smells?
- Are they pleasant? Or unpleasant?
- Can you recognize them? (smoke, perfume, gas, animal smells, etc.)
- What are they like?
- How many times per day or per week does this happen?

7. Amplification of gustatory thoughts (tastes, flavors, etc.).
Questions:

- Are you aware of unusual tastes but unsure if they really exist?
- Are these tastes recognizable?
- Do you have anything in your mouth that could produce these tastes?
- Are they pleasant or unpleasant?
- How many times per day or per week does this happen?

8. Amplification of co-enesthetic thoughts (changes in body image, size or shape of body parts, or more often internal feelings for which a stimulus cannot be identified). These symptoms (co-enesthetic hallucinations) are distortions of the idea of body image, rather than of perceptions due to actual external somatic stimuli. The hallucination that there is an object within the body or head is often subject to immediate delusional elaboration.
Questions:

- Do you have strange sensations inside your body? Or in your brain?
- Do you think something is changing within your body? Or in your brain?
- Do you have a burning sensation in a part of your body?
- Do you feel the size or shape of a part of your body is changing?
- Do you feel there is something strange inside your body?
- Or inside your head? What is it? Microphones? Electrodes? Microprocessor? Animals? Knives? etc.
- How many times per day or per week does this happen?

9. Amplified tactile thoughts (sensations of touch, burning, biting, or of something moving or crawling on or under the skin (parasites) for which no stimulus can be identified. May be subject to immediate delusional elaboration).
Questions:

- Do you have sensations of being touched? Or being bitten? Or of something (like insects, or animals, or snakes) moving across or under your skin?
- How many times per day or per week does this happen?

10. Amplified kinesthetic thoughts (sensations of movement of body parts when they are stationary), e.g. hallucination of a specific movement of a sporting activity.
Questions:

- Do you keep imagining body movements?
- Do you feel that some parts of your body are moving, even though you can see that they are stationary?
- Can you recognize what these movements are?
- Are you sure that these sensations come from movements that are actually happening?

- Do you feel that one part of your body is flying?
- How many times per day or per week does this happen?

11. Amplified vestibular thoughts (sensations of movement of whole body, of balancing, or of loss of balance). It is assumed that the sensation of whole-body movement arises mainly from hallucinations in the vestibular sense (i.e. from the organs of balance).

Questions:

- Do you keep imagining that your whole body is moving, or that you are losing your balance?
- Are you certain that these sensations are connected to something that is really happening to your body?
- Do you sometimes feel that your whole body is moving when you can see that it is not?
- Do you feel you are flying? Or falling? Or that your body is rising from the ground?
- Can you recognize the body movement you feel?
- How many times per day or per week does this happen?

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