Discussion

Summary of Findings

In everyday situations, when the head position remains stable, both the pursuit and saccadic systems work interactively in order to maintain the image of a moving target on the retina (dual-mode pursuit: Eckmiller, 1987). In the current study, participants with schizophrenia, at a group level, were able to track a moving stimulus equally as well as neurologically-intact control observers using dual-mode pursuit at all but the fastest target velocity examined in the current study (35°/sec). Isolating the pursuit system (single-mode gain) yielded similar results, with clinical participants performing equitably with controls at slow to moderate target velocities (5°/sec to 20°/sec), but poorly when higher speeds were required (25°/sec to 35°/sec). In contrast, the clinical participants produced a significantly greater number of positively-directed saccades at all target velocities, although the magnitude of this difference was reduced at high target speeds (30°/sec and 35°/sec). Only very few saccades were made in the opposite direction to the target by either group, with the clinical group producing a significantly greater number of such reversal saccades only at the extremely slow target speed (5°/sec).

Using qualitative ratings of combined eye tracking performance, around half of the participants with schizophrenia were classified as eye-tracking disordered (scoring a rating of three or above on average qualitative ratings of tracking a 5, 10 and 15°/sec target on the four-point Benitez 1970 scale). Consistent with the classification, those categorised as eye tracking disordered produced significantly lower single-mode gain, as well as more saccades, than those patients that were classified as having normal tracking.
There were no differences between the eye-tracking disordered participants and others with schizophrenia in terms of age, duration of illness, medication level, extrapyramidal symptoms or total psychopathology assessed through the PANSS. Those classified as having disordered eye tracking did, however, have significantly lower WAIS-III full-scale IQ scores as well as poorer sensitivity scores on the Continuous Performance Task of sustained visual attention. Consistent with this, the only symptom grouping on either the three-, five- or eleven-dimension models of psychopathology that differentiated the groups was the degree of ‘cognitive dysfunction’ under the eleven-factor model (clinical judgements of attention and abstraction problems), on which the patients with eye tracking disorder were significantly more affected than the other clinical participants.

Relationships between symptom dimensions and components of eye tracking were examined, with the hypotheses that the degree of ‘cognitive dysfunction’ and ‘negative signs’ defined in the eleven-dimension model of symptomatology would be related to the adaptive aspects of tracking (gain and positive-directed saccades), and the ‘conceptual disorganisation’ grouping related to the disinhibitive aspects of tracking (most closely related to reversal saccade frequency). Consistent with the hypothesis, only the degree of ‘cognitive dysfunction’ in the eleven-dimension model, and none of the symptom groupings in the three- or five-dimensional models predicted single-mode gain. However, this only related to performance at the medium (15-20°/sec) and fast (25-35°/sec) velocities and even then was only weakly associated (explaining 6-7% of variance), showing little improvement over a null model. Inconsistent with the hypotheses, positive-direction saccades were predicted by the degree of ‘social dysfunctions’ rather than ‘negative signs’ in the eleven-dimensional symptom model, and only at the fast target speeds. While the broad ‘negative’ symptom grouping under the five-factor model also emerged as a predictor of positive saccade frequency during fast
target speeds, neither these nor the more closely-defined ‘social dysfunction’ symptoms explained a substantial amount variance in the dependent measure (less than 10% each). Reversal saccades were very few in number, displayed a mixed pattern of symptom relationships under the three models at different velocities, and, in difference to the hypotheses, were unrelated to ‘conceptual disorganisation’, ‘negative signs’ or ‘cognitive dysfunction’. At the slow target velocities (5-10°/sec), the only speeds at which the patient group displayed an abnormal frequency of reversal saccades, these were predicted by the degree of ‘positive’ symptoms under the three-factor model of symptomatology; by the degree of ‘positive’ and ‘mood’ symptoms under the five-factor model; and by the degree of ‘loss of boundary delusions’ in the eleven-dimension approach; however, as per all other symptom relationships, these correlations explained just 10% or less of the variance in reversal saccade frequency.

**Consistency of group level findings with previous literature**

The clinical participants displayed adequate dual-mode pursuit at all but the highest target velocity, a point at which even the tracking of neurologically-intact participants was reduced (the mean dual-mode gain of the control participants was 0.85 at 35°/sec). The performance of the clinical participants using single-mode pursuit was indistinguishable from that of controls at all but the fastest three target velocities (25-35°/sec). While previous studies have identified deficiencies in gain at moderate target velocities (Clementz et al., 1990; Friedman et al., 1995; Friedman, Jesberger & Meltzer, 1991; Levy et al., 1993; Sweeney et al., 1994), this finding has not been perfectly consistent (Allen, 1997; Thaker et al., 1999). The patterns of performance seen here follow the general trend shown in several studies, whereby the disparity between the groups increases with
target speed (Abel et al., 1991; Levin et al., 1988), and specifically replicates the Hutton (et al., 2001) study findings, whereby group differences were most apparent at target speeds of 30°/sec and above. The identification of superior single mode pursuit performance in the clinical group at 10°/sec is inconsistent with a great deal of previous research (Levy et al., 1993; 1994). This may, however, reflect an anomaly of this dataset, particularly in light of the fact that saccadic activity was at its greatest at the slow target velocities (5 and 10°/sec: Table 76), for the clinical group in particular, and as such, there would have been a smaller amount of time spent in smooth pursuit available for scoring at these velocities. Moreover, given the finding of significantly greater saccadic activity among the clinical participants with dysfunctional eye tracking, it would be these poor trackers in particular that would be spending the least time in score-able single-mode pursuit, which may underlie the unusual finding of superior tracking in the clinical group at this velocity.

The increased overall frequency of positively-directed saccades among the clinical participants is consistent with previous findings in terms of an increased rate of CUS (which are by far the most prevalent positively-directed saccade: Levy et al., 2000) among observers with schizophrenia (Fletchner et al., 1997; Levy et al., 1993; Ross et al., 1996; 2001; Sweeney et al., 1994). Similarly, the decreased magnitude of the group difference at high target speeds has been identified in previous research, as even the neurologically-intact participants cannot keep up with the target with the single-mode pursuit system alone at high target speed, and so produce an increasing number of compensatory saccades with increasing target velocity (Abel et al., 1991; Mather et al., 1992; Schalen, 1980). However, the measure of positively-directed saccades represents a composite of compensatory CUS, and the intrusive SWJ, AS and LS. The finding of a greater number of positive saccades among the observers with schizophrenia, in the context of equal (or
indeed superior) single-mode gain at low and moderate target velocities, suggests that this difference between groups may reflect an increased presence of intrusive saccades. This is consistent with the identification of a greater frequency of reversal saccades in the clinical group at the 5°/sec target speed (Table 76). Indeed, the greatest amount of saccadic activity in both clinical and control groups were identified at the lowest target velocities examined (5 and 10°/sec: Table 76). This has been identified by other researchers for observers with schizophrenia, with, for example, Mather, Neufeld, Merskey and Russell (1992), identifying a greater saccadic frequency among observers with paranoid schizophrenia when tracking 11°/sec targets than during tracking 22, 33, 44, 55, or 66°/sec targets. However, both the Mather (et al., 1992) study and multiple other authors (such as Schalen, 1980) have shown that saccade frequency steadily increases with target velocity in neurologically-intact controls.

A number of possible causes may be suggested for this unexpected pattern of findings. Firstly, this may be an effect of methodology: the eye movement trials at different velocities were not presented in counterbalanced order, and it is possible that performance during the initial trials (always the 5°/sec target speed) may have been affected by participant or performance anxiety. However, any such effect would have dissipated quickly, and so does not explain why saccadic activity was greatest when following the 10°/sec target, as this was target was presented after five full tracking cycles at the lower tracking velocity (40 seconds), and any initial anxiety in regard to task complexity would likely have been addressed by this point. Alternatively, as single-mode gain for both groups steadily reduced with increasing target speed, the increased level of saccadic activity may reflect less tolerance for position error at low velocities (in which case, a greater number of small-amplitude CUS would occur), or poor accuracy in predicting target location when making corrective saccades (for example, CUS
overshooting the target and hence requiring corrective BUS). Finely detailed analyses of saccadic type and amplitude across velocities would be required to examine this possibility. A third possible suggestion may be that disinhibition of the saccade system may be responsible for the increased saccadic activity at low target velocities. The extremely slow-moving targets at 5 and 10°/sec are very undemanding cognitive tasks. It is possible that, following a Yerkes-Dodson-type ‘inverted-U’ performance pattern, arousal is low at the undemanding target velocities (5-10°/sec) and hence intrusions of saccades into pursuit are not actively inhibited. An ‘inverted-U’ type effect of arousal on performance on several visual attentional tasks has previously been shown (Parrott & Craig, 1992). More pertinent to the current discussion, Matsue (et al., 1986) showed that frequency of intrusive saccades increases during a fixation task as the intensity of the fixation target decreases (fixation on a visible target, versus fixation on an imagined target in the dark, versus closed eyes with no target), with this effect of concentration of attention apparent in both those with schizophrenia and controls, and exacerbated in the clinical group. Moreover, the frequency of saccades during fixation on an imagined target was shown to be significantly related to those seen during eye tracking (r=0.6, p<0.05: Matsue et al., 1986).

While only specific studies will resolve the reason behind this effect of increased saccadic activity at extremely low target speeds, findings in regard to between-group differences in terms of saccadic activity have been equivocal both for compensatory (Abel et al., 1991; Hutton et al., 2001) and intrusive (Ross et al., 1999; 2001; Sweeney et al., 1992; 1994) saccades, and the findings here suggest that some of the reason for the discrepant findings between studies may relate to the velocity of the target stimuli employed. Most studies in the area of eye tracking in schizophrenia only make assessments at a single target velocity, and given the findings by Ross and colleagues that AS and LS may be
particularly important as phenotype markers (Ross et al., 1998; 2002: who commonly employ a target moving at 16.7°/sec) it would be suggested that these studies may be best employed at slow target speeds in order to maximise the presence of saccadic activity, and hence the methodological power of such experiments.

Qualitatively-defined eye tracking disorder groups

The scale of Benitez (1970) was used to rate the quality of eye tracking performance of participants in response to 5, 10, and 15°/sec targets. Using an (arbitrary) cut off point of mean performance of three or above on this four-point performance scale, approximately half of the participants with schizophrenia were classified as exhibiting an eye tracking disorder (ETD). This rate is consistent with the findings of other authors (Levy et al., 2000; and Allen, 1997, applying global qualitative or quantitative measures respectively), but is perhaps overly conservative when compared to the findings of the admixture studies of Ross and colleagues (Ross et al., 1996; 1997; 2002), showing that 21-34% of observers with a diagnosis of schizophrenia produce global eye tracking performances that fall into a distribution that is qualitatively disordered compared to neurologically-intact observers. The results here, showing that the group of clinical participants classified as ETD did not differ from other observers with schizophrenia in terms of variables such as age, duration of illness, medication level or extrapyramidal side effects are consistent with research suggesting that these factors are not responsible for the presence of eye tracking disorder in schizophrenia (Levy et al., 1994; Ross et al., 1998).

The finding that there were no difference between those clinical participants with ETD and those without ETD on any grouping of symptoms under either the three- or the
five-factor models of symptomatology in schizophrenia has also often been suggested in previous research (Keefe et al., 1989; Levy et al., 2000), and is consistent with the view that ETD itself is a trait, rather than state, effect (Clementz & Sweeney, 1990; Iacono, 1998). This, however, is problematic for our hypotheses of syndrome relations to specific measures of eye tracking, and will subsequently be discussed in greater detail. The only distinguishing group of symptoms between the clinical participants with ETD and without was the ‘cognitive dysfunction’ grouping as defined under the eleven-dimensional model of symptomatology. This symptom group comprises items that are clinical assessments of dysfunctions of attention and abstraction, and consistent with this, the ETD group, who displayed a worse mean ‘cognitive dysfunction’ assessment, also had significantly lower WAIS-III full-scale IQ scores and a trend toward poorer sensitivity on the Continuous Performance Task (CPT). This latter finding, of a relatively small difference between these clinical groups on the CPT, is consistent with the weak relationships between this task and eye tracking performance identified in previous research (Grawe & Levender, 1995; van der Bosch, 1984). Moreover, as CPT performance may be reduced among participants with lower general intelligence (Carter & Swanson, 1995; Herman, Kirchner, Streissguth & Little, 1980; Kirby, Nettelbeck & Bullock, 1978; Tomporowski & Allison, 1988), this CPT performance difference between ETD groups may be a secondary reflection of the lower general intellectual performance among the clinical participants with ETD.

Several studies have failed to identify relationships between eye tracking disorder and general intellectual ability, when this relationship is examined on a correlational basis (Hutton et al., 2004; Grawe & Levander, 1995), although others have identified weak relationships that do not survive correction for duration of education (Katsanis & Iacono, 1992). However, these studies have treated all those with a diagnosis of
schizophrenia as a uniform group, and have not examined differences between qualitatively ‘good’ and ‘poor’ eye trackers. Among a sample of university students, general intelligence deficits have been related to eye tracking performance, but only among those that display poor eye tracking (Coursey, Lees & Siever, 1989). As such, if only around half of any sample of schizophrenia observers display ETD (Allen, 1997), and most studies examining neuropsychological deficits in relation to eye tracking treat observers as a homogeneous (ETD) group, then such a methodological approach may obscure identification of cognitive deficits in those that experience ETD. Given such a concern, the question as to whether eye tracking dysfunction does relate to a generalised neuropsychological deficit is a question worth revisiting using the more sensitive methodological approach of only examining those displaying disordered tracking.

While no differences between the ETD and non-ETD classified participants with schizophrenia emerged in terms of symptoms (other than the ‘cognitive dysfunction’ symptom trio), a significant discriminant function was identified, with ‘cognitive dysfunction’, as well as the ‘conceptual disorganisation’ (positive formal thought disorder) symptom groupings from the eleven-dimension model contributing to this function for categorising clinical participants as ETD and non-ETD. This finding was consistent with the hypothesised relationship of ‘conceptual disorganisation’ to eye tracking disorder. While no difference was identified in the level of ‘conceptual disorganisation’ symptoms between ETD and non-ETD participants in the current cohort, other investigations have identified just such an effect (Keefe et al., 1989; Solomon et al., 1987). However, while the discriminant function using the degree of ‘conceptual disorganisation’ and ‘cognitive dysfunction’ symptoms to differentiate between ETD and non-ETD clinical participants was consistent with the hypotheses and with previous research, the equation, although statistically significant, was overly
Does this study provide evidence for the validity of the eleven-factor model of symptoms?

This is the key question that this study was designed to investigate. In order to examine the evidence in support of the validity of the eleven-factor model, the findings of the current study are reviewed in relation to three aspects of this question, namely: whether symptom grouping measures were useful predictors of eye tracking performance; whether application of the eleven-factor model of symptoms yielded any advantage over the more parsimonious three- or five-factor models in terms of such predictions; and finally, whether there is evidence that the symptom groupings under the three- and five-factor models harbour heterogeneous sub-groups, which is a fundamental assumption of the eleven-factor model.

1. Were symptom measures useful predictors of eye tracking performance?

There were statistically significant relationships identified between aspects of symptomatology defined under the eleven-dimensional model and each of the three measures of eye movement performance examined in the current study. While not simply reflective of chance associations, these relationships were uniformly moderate. The specific findings in regard to each measure of eye tracking performance are discussed individually below.
Single-mode gain

In terms of single-mode gain, differences emerged between participants with schizophrenia and neurologically-intact controls at fast target velocities (25°/sec and above), with performance in the clinical group equivalent to that of controls at lower target speeds (and possibly even being superior at 10°/sec). On the basis of findings from several studies identifying relationships between ‘negative’ symptoms, broadly defined, and global eye tracking performance or gain (Blackwood et al., 1991; Katsanis & Iacono, 1991; Lees Roitman et al., 1997; Sweeney et al., 1992; 1994), and in particular, studies suggesting that the ‘negative’-type subgroups of alogia (a component of the ‘negative signs’ grouping in the eleven-dimension model) and the SANS ‘attention’ subscale (a component of the ‘cognitive dysfunction’ grouping in the eleven-dimension model) were related to eye tracking performance (Ciuffreda et al., 1993; Malaspina et al., 2002), it was hypothesised that the eleven dimensional model groupings of ‘negative signs’ and ‘cognitive dysfunctions’ would be related to the compensatory aspects of eye tracking performance.

Only a single symptom grouping under any of the three-, five- or eleven- dimensional models emerged as a significant predictor of single-mode gain, this being the degree of ‘cognitive dysfunction’ from the eleven-dimension model. While this relationship emerged as hypothesised, it was only related to single-mode gain performance at the medium (15-20°/sec) and fast (25-35°/sec) target velocities, and was also only a very weak predictor of performance (explaining 6-7% of the variance in single mode gain at the medium or fast target velocities). The fact that the correlates of single-mode gain at the slow (5 and 10°/sec) and the medium to fast (15-35°/sec) velocities were different is actually in keeping with the findings of greater saccadic activity at the slow velocities than others (Table 76), and suggests that there are subtly different processes occurring at the
low velocities in comparison to those during the moderate and faster target speeds. The finding of correlations between the ‘cognitive dysfunction’ grouping and single-mode gain is in keeping with the very mixed pattern of findings in relation to the relationships between ‘negative’ symptoms and eye tracking performance (for example Katsanis & Iacono, 1991; Lees Roitman et al., 1997, compared with Lee et al., 2001; Sweeney et al., 1998), in that the broad concept of ‘negative’ symptoms, defined through instruments such as the SANS, was subdivided under the eleven-dimension model into groupings of ‘negative signs’, ‘social dysfunction’ and ‘cognitive dysfunction’ – and only the latter of the three sub-groupings displayed any relationship with single-mode gain. As such, the discrepant findings in previous literature may relate to the application of different definitions of ‘negative’ symptoms, or use of scales other than the SANS, as it may be the case that relationships are only able to be identified when variance due to ‘cognitive dysfunction’-type symptoms are included within the definition of ‘negative’ symptoms. This is exactly the effect that occurred in the Lee (et al., 2001) study: when a broad definition of ‘negative’ symptoms was applied, significant relationships were identified with global eye tracking performance; however, when the variance due to the item ‘difficulties in abstract thinking’ (one of the three symptoms within the ‘cognitive dysfunction’ subgroup) was removed, this relationship disappeared.

However, the relationships identified between ‘cognitive dysfunction’ and single-mode gain was only weak (explaining just 6-7% of variance), and it is possible that this reflects a secondary relationship arising from shared relationships with some intervening variable. Alternatively, this may represent the small role that general intellectual function plays in eye tracking performance, which may be consistent with the significantly poorer ratings of ‘cognitive dysfunction’ and WAIS-III Full Scale IQ apparent among those participants classified as exhibiting ETD when compared to those with normal tracking ability.
Indeed, some authors have suggested an association between frontal-lobe mediated tasks and poor eye tracking performance, on the assumption that if performance is poor on ‘frontal’ tasks, then this deficit may extend into the frontal eye fields, which are important in mediating eye tracking (Katsanis & Iacono, 1991; Tregallas et al., 2004). There is, however, no reason for this to be the case other than the proximity of the structures involved, and several studies have identified no relationships between neuropsychological tasks with heavy demands on frontal lobe function, such as the Wisconsin Card Sorting Test, and eye tracking performance. (Friedman et al., 1995; Grawe & Levander, 1995).

In sum, among this cohort of observers with a diagnosis of schizophrenia, no measures of clinical symptoms showed any relationships with single-mode gain. The only identified associate, the degree of clinically-assessed cognitive-dysfunction, although hypothesised, was only weakly related with this measure (sharing around 6-7% of variance) and may simply reflect the minor influence of general intellectual decline on this task.

*Positive saccade frequency*

The frequency of positive saccades was shown to be increased in the observers with schizophrenia relative to control participants at all target velocities, although the magnitude of the difference reduced at high target velocities (remaining significant or trending towards being so: Table 76). As it had been shown that alogia (but not social dysfunctions or emotional blunting: Ciuffreda et al., 1993; Malaspina et al., 2002) and attention (but not avolition, anhedonia or affective flattening: Ciuffreda et al., 1993) were related to saccadic frequency during eye tracking, it was hypothesised that the eleven-dimensional model symptom groups that incorporated these symptoms (‘cognitive dysfunction’ and ‘negative signs’) would relate to the adaptive components of eye
tracking – and hence, both single-mode gain and positively-directed saccades (given that compensatory CUS are likely to comprise approximately 90% of the variance in this measure: Levy et al., 2000). Regression models showed that the degree of ‘social dysfunction’ predicted positive saccade frequency, but only at the fastest target velocities (25-35°/sec), and that neither of the hypothesised symptom groupings displayed any trend toward correlating with this dependent measure. The five factor model conception of ‘negative’ symptoms also emerged as a statistically significant predictor of performance at the fast target velocities (25-35°/sec) but it could be shown that this relationship arose due to the presence of the same symptom items (those defined as ‘social dysfunctions’ in the eleven-dimensional model) within this grouping.

What are the possible reasons for this finding? Firstly, the hypothesised relationships (of ‘negative signs’ and ‘cognitive dysfunction’ relating to the frequency of positive saccades) were identified on the basis of a study conducted at slow to moderate target velocities (4.3 and 11.9°/sec: Ciuffreda et al., 1993), however, in the current study no symptom relations were identified at these speeds – instead, the single identified relationship was apparent only at the fastest target velocities (25-35°/sec). Secondly, some studies have identified a relationship between poor eye tracking performance and poor outcome (Beiser et al., 1994; Katsanis, Iacono & Beiser, 1996; Lieberman et al., 1993), which is a concept highly interwoven with the symptoms incorporated with the ‘social dysfunctions symptom grouping (Green & Nuechterlein, 1999). This is certainly consistent with the findings of increased presence of eye tracking disorder among those categorised with the deficit syndrome, a diagnosis that has also been associated with poor outcome (Kirkpatrick, Buchanan, Ross & Carpenter, 2001). However, if such an association was to explain the underlying eye tracking deficit in these participants, then it could be anticipated that differences on the ‘social dysfunctions’ grouping would have been
apparent when comparing clinical participants classified as having ETD with those that were not – which did not occur; moreover, given the intermediate relationship between neurocognitive performance and outcome measures (Green & Nuechterlein, 1999), then if a link with poor outcome is to explain eye tracking performance, then one would expect the positive saccades to be related to both social dysfunction and cognitive dysfunctions – which was not the case; finally, if ‘social dysfunctions’ is a particularly important correlate of performance then relationships would have been identifiable at all target velocities. Thirdly, positive saccades are a composite of compensatory CUS and intrusive SWJ, AS and LS. At the fast target speeds applied here (25-35°/sec), it has been shown that there are little differences between observers with schizophrenia, and neurologically-intact controls in CUS frequency (Abel et al., 1991; Mather et al., 1992). As such, the identified difference between clinical and control groups in positive saccades at these frequencies may reflect a greater presence of intrusive saccades (a suggestion consistent with the trend to greater reversal saccades at these target velocities: Table 76). If this is the case, then the identification of a relationship between positive saccades and social dysfunctions at the fastest (25-35°/sec), but not the slow or moderate target velocities (5-20°/sec) may suggest that the relationships arises from the influence of the intrusive rather than the compensatory aspects of eye tracking, which are likely to arise from distinct underlying neurological causes (Abel & Ziegler, 1988; Hutton & Kennard, 1998). However, this proposal is unable to be examined in further detail in the current study.

Finally, however, the relationship between ‘social dysfunction’ symptoms and the frequency of positive saccades’ identified here are extremely small (8% of shared variance), and, while they may not simply be due to chance, they represent an extremely small influence on the dependent measure of the frequency of positive saccades (Table 82 and 84). There certainly has not been a great deal of precedent for symptom
relationships of saccadic activity during pursuit: Nkam (et al., 2001) and Hong (et al., 2003) found no difference between schizophrenia observers with and without the deficit syndrome (deficit patients, by definition, would have poor ratings on the ‘social dysfunction’ grouping: Kirkpatrick et al., 2001; Ross, 2000) on SWJ or AS frequency; and while some studies have identified symptom relationships with saccade frequency in general (Ciuffreda et al., 1993) or specific saccade types (Sweeney et al., 1992; 1994); others have failed to identify such relationships (Flechner et al., 1997; King et al., 1999).

It is, however, possible that the presence of a real saccade/symptom grouping relationship has been obscured by either incorrect definitions of symptom groupings, or, more likely, the combination of compensatory and intrusive saccades into a single dependent measure (such as is the case with examining all forward-directed saccades in combination), particularly as there is some evidence to suggest that these aspects of performance may arise from different neural structures (Hutton & Kennard, 1998; MacAvoy & Bruce, 1995). Alternatively, the poor symptom relationship here may simply be an artefactual relationship arising from shared correlations with some third intervening variable. While these are intriguing possibilities amenable to future investigations with more targeted studies, at present it can simply be concluded that the identified relationship between the degree of ‘social dysfunction’ and positive-directed saccades is statistically significant, but very weak, and, when combined with the poor relationships between symptom groupings and single-mode gain, is not consistent with the concept of a syndrome-related deficit.
Reversal saccade frequency

The presence of saccades made in the opposite direction to that of the movement of the target – reversal saccades – was small among both clinical and control cohorts, and reduced steadily in frequency with increasing target velocity (Table 76). Observers with schizophrenia, at a group level, produced a significantly greater number of reversal saccades at the extremely slow target velocity (5°/sec), and a trend was apparent for a greater presence of reversal saccades in the clinical group at the 25 and 35°/sec target speeds. However, in general, reversal saccades were very infrequent for all participants, falling from a mean of two saccades per five cycles of tracking at the 5°/sec target velocity, to less than one at a 35°/sec target speed among clinical observers.

Reversal saccades will represent a combination of compensatory BUS and intrusive SWJ, although several studies have suggested that compensatory backward saccades may be more frequent than SWJ (with BUS representing 12-20% of all saccadic activity during pursuit, in comparison to 4-6% of activity due to SWJ: Levy et al., 2000). From the earlier review of symptom relationships with eye tracking performance, it was hypothesised that the eleven-dimension symptom model grouping of ‘conceptual disorganisation’ would relate to the intrusive aspects of saccadic activity during eye tracking performance, and that the ‘cognitive dysfunction’ and ‘negative signs’ groupings would be related to the compensatory aspects of eye tracking performance. In contrast to the findings for the limited relationships between symptoms and single-mode gain or positive saccades, a number of statistically significant symptom relationships were identified with reversal saccade frequency. These symptom relationships appeared to differ across the target velocities (Table 89). Considering the symptom relationships at the target velocities where reversal saccade frequency in the clinical group was most
deviant (slow target velocities of 5 to 10°/sec), raw correlations showed that the degree of ‘positive’ symptoms under the three- and five– factor models of psychopathology trended towards significance, with the eleven-dimensional model suggesting that this relationship was largely due to the presence of delusional and hallucinatory symptoms rather than paranoia (which are the symptom combined together under the broad ‘positive’ symptom dimension in the three- factor model: Tables 86, 89, 90, and 91).

These relationships were actually inverse relationships, suggesting that increased reversal saccades were related to lower levels of ‘positive’, or, in particular ‘loss of boundary delusions’, ‘grandiosity’ or ‘auditory hallucinations’. This is an unusual relationship, and while inverse relationships have previously been identified between broad ‘positive’ symptom groupings and measures of global eye tracking performance (Bartfai et al., 1985; Katsanis & Iacono, 1991; Kelly et al., 1990), it is difficult to suggest a meaningful interpretation for this finding.

At moderate target velocities (15 to 20°/sec), the frequency of reversal saccades was related to the degree of ‘anxious introspunitiveness’, and to the anxiety symptoms within this group in particular (a relationship with this group of symptoms also emerged when applying the five-factor model of symptoms at low target speeds of 5 to 10°/sec: Table 89). This was a positive association, whereby increased anxiety was associated with an increased frequency of reversal saccades. This may be interpreted in terms of increased distractibility: early studies suggested that by increasing stress, eye tracking performance in neurologically-intact participants can be made to resemble the disorder seen amongst observers with schizophrenia (Brezinova & Kendall, 1977), and similarly, studies in neurologically-intact observers have shown that increasing distraction, a common symptom of anxiety, produces greater saccadic activity, particularly AS toward distracting stimuli and subsequent compensatory BUS to refoveate the target (Kaufman & Abel,
1986; Nolte et al., 1999). At high target velocities (25 to 35°/sec), the relationships between symptom groupings and reversal saccade frequency change (in the case of those under the three- or eleven-dimensional models of symptomatology), or disappear completely (in the case of the five-factor model). These changes are likely to arise due to the extremely low presence of reversal saccades at these target speeds, as the mean presence of reversal saccades was less than one per five tracking cycles (Table 76).

The identified relationships between symptom groupings and reversal saccade frequency are universally low (as has been the case with other measures of eye tracking performance) and explained less than ten percent of the variance in the dependent measure in all instances. These correlations are likely to be particularly effected by the reduced range of performance in the frequency of reversal saccades (Nunnally, 1978). With the exception of the slowest target velocity (5°/sec), where half of the clinical participants either produced no or single reversal saccades, at all higher target velocities, on average 80% of clinical participants produced nil or single reversal saccades (Table 76). This range of performance was also apparent in the control cohort. As such, this reduction of range will either curtail the magnitude of correlation possible, or will render the analysis susceptible to the identification of artefactual relationships on the basis of similar distributions (Maxwell, 1972). The finding of different relationships under each of the symptom models and across target velocities is consistent with the suggestion that these relationships, particularly those at the high target velocities (25-35°/sec), may simply arise from such chance factors.

In sum, as per the findings for single-mode gain and for positive saccades, while there are some promising relationships identified (such as for anxiety) with frequency of reversal
saccades, the relationships only contribute to the explanation of a minor amount of the variance in the dependent measure.

2. Did the eleven-factor model of symptoms show any advantages over the more parsimonious symptom models?

In terms of the prediction of single-mode gain, there were no symptom groupings under the three- or five-factor model of symptomatology of schizophrenia that contributed to the prediction of single mode gain performance at any target velocity. Using the eleven-dimensional model, however, the grouping ‘cognitive dysfunction’ did emerge as a significant predictor of performance at the medium and high target velocities (15 to 35°/sec). However, this explained just 6-7% of the variance in the dependent measure, and did not produce a significantly better prediction of single-mode gain over that provided by the control measure of duration of illness (Table 80).

In the prediction of positive-duration saccades, no symptom groupings under the three-factor model of symptoms predicted positive saccadic frequency at any target velocity. However, adopting the five-factor model, the degree of ‘negative’ symptoms predicted positive saccade frequency only at fast (25 to 35°/sec) target speeds; and similarly, the eleven-dimensional model grouping of ‘social dysfunction’ (a sub-group of the broad ‘negative’ concept in the five-factor model) emerged as a significant predictor at these target velocities. The correlation between symptom groupings and the frequency of positive saccades was greatest using the eleven-dimensional model: under the three- and five-factor symptom models, the relationship occurred only at trend level (r=0.19 and r=0.21 for the three- and five-dimensional models respectively; Table 81), but when
these broad conceptions of ‘negative’ symptoms were subdivided into ‘negative signs’ and ‘social dysfunctions’ subgroups through the application of the eleven-factor model, a significant relationship between positive saccade frequency and ‘social dysfunctions’ was apparent ($r=0.30, p<0.01$), while the other group of symptoms were unrelated to this measure (‘negative signs’ $r=-0.09, p>0.05$: Table 81). Inspection of the relationships between the individual symptom items contained within each of these symptom groupings and the dependent measure showed greater uniformity when the grouping was tightly defined (i.e. the use of the ‘social dysfunctions’ grouping rather than a broader ‘negative’ grouping). Additionally, the more broadly defined symptom groups (‘negative’ symptoms under the three- and five-factor models) encompassed items that shared no relationship with the frequency of positive saccades along with those displaying moderate relationships, as is captured by the zero-order relationship identified for the ‘negative signs’ group of symptoms with the dependent measure, but a moderate relationship for the ‘social dysfunctions’ symptom group (Table 81 and 85). However, the eleven-factor model ‘social dysfunctions’ grouping explained only a small amount of variance in the dependent measure, and did not produce a statistically superior prediction of the frequency of positive saccades in comparison to that provided by the control measures of illness duration or extrapyramidal symptoms alone (Table 84), and nor was it statistically superior to the prediction afforded through the application of the five-factor model of symptoms (Table 84).

In the prediction of reversal saccade frequency, a mixed pattern of correlations emerged under each model of symptomatology and at each target velocity. Considering the frequency of reversal saccades overall (averaged over the 5-35°/sec target velocities), when the three-factor model is applied, the ‘positive’ symptom group emerged as a statistically significant ($r=-0.24, p<0.05$: Table 86), but minor predictor. Under the five-
factor model, ‘positive’ symptoms remained a predictor of performance, and additionally, the ‘mood’ symptom grouping, which is not included within the spectrum of psychopathology examined in the three-factor model, also emerged as a predictor of reversal saccade frequency ($r=-0.28, p<0.01; r=0.24, p<0.05$ respectively; Table 86). The ‘mood’ group in the five-factor model has the exact same content as the ‘anxious intropunitiveness’ symptom group within the eleven-factor symptom model, and as such this also entered the regression models as a significant predictor of performance when the eleven-factor model was applied ($r=0.24, p<0.05$; Table 86). The ‘positive’ symptom grouping that emerged as a significant predictor using the three- and five-factor symptom models is similar in content under each approach, however, under the eleven-factor model, this is sub-divided into four: ‘loss of boundary delusions’; ‘grandiosity’; ‘auditory hallucinations’; and ‘paranoia’. Examining correlations for each of these groupings and the frequency of reversal saccades, a very similar, moderate magnitude relationship with the dependent measure was apparent for the first three of these groupings, and not for ‘paranoia’, with the groupings of ‘auditory hallucinations’ and ‘loss of boundary delusions’ emerging as significant predictors respectively at different target velocities (Table 89). However, despite the differences in relationships identified when different symptom models were employed, there was no statistically significant benefit in terms of the prediction of reversal saccade frequency arising from the application of the more refined eleven-dimensional model of symptoms over that provided by the symptom groupings defined under the three- or five-factor models (Table 89).

Taken together, symptom groupings under the more complex eleven-factor model did not produce a statistically significant improvement in the prediction of any of the three aspects of eye movement assessed in the current study, when compared with the prediction produced when control measures (extrapyramidal symptoms) or the symptom
groupings defined under other models (the three- and five- factor models). However, this may not preclude the appropriateness or validity of the eleven-dimension model: indeed, the presence of symptoms may simply contribute little to the variance of these measures regardless of the specific nature of their combination – in fact, in no model was more than 16% of the variance of any eye tracking measure explained by symptom groups.

What is apparent in terms of the advantage of the eleven-dimensional model was that it was able to produce correlations that were clearer to interpret: while the broad symptom groupings in the more parsimonious symptom models did identify some relationships between symptom groupings and eye movement variables, the use of the more tightly-defined groupings in the eleven-dimensional model showed that the relationships with external measures were undermined through their encompassing of symptom items that did not share uniform patterns of relationships with the dependent measures. This is exemplified in the case of prediction of positive saccade frequency, where the five-factor model ‘negative’ symptom group predicted performance, however, when these symptoms were subdivided into ‘negative signs’ and ‘social dysfunctions’ subgroups in the eleven-factor model, only the latter group of symptoms showed any relationship with performance (Table 81, 82, and 85). These particular aspects of the current findings are discussed in more detail in the subsequent section.
3. Is there evidence that the symptom groupings in the three- and five-factor symptom models harbour heterogeneous sub-groups?

In the first study of this series, it was apparent that the symptom groupings under the three- and five-factor models of symptomatology combined poorly related symptoms into groups. To better account for the relationships between symptoms, eleven homogeneous groupings of symptoms were defined, largely reflecting subdivisions of the coarse combinations defined under the three- and five-factor models. The logic of the current study was that, if it could be shown that these subdivisions of symptom factors in the existing models displayed differential relationships with external variables that have been shown to play a role in schizophrenia, then this would support the independence of such groupings. For example, the ‘negative’ dimension under the three-factor model of psychopathology was divided into three by the eleven-factor approach. If all three of these groups showed identical relationships with an external measure, then there is little evidence to support the validity of their division; however, if these groupings differ in their external relationships, then their independence is apparent. Table 93 summarises the evidence provided by the current study toward this end: reviewing the intercorrelations between the symptom groupings defined in the eleven-factor model in terms of their parent combinations in the three- and five-factor models, and the relationships between each of these eleven symptom groupings and the measures of eye tracking performance. The evidence for the independence of the symptom subgroups in the eleven-factor model from those they are combined with in other approaches will be reviewed in turn below.
The ‘reality distortion’ factor under the three-factor model of symptomatology (and, similarly, the parallel ‘positive’ symptom grouping in the five-factor model) was shown in the first study of this thesis to encompass a very heterogeneous group of symptoms. In the development of the eleven-factor model, this was subdivided into four groupings (with a further two groupings tentatively suggested but unable to be examined in the current study due to low symptom prevalence). As shown in Table 93, these fine-grained symptom groupings were all poorly inter-related, with the exception of ‘auditory hallucinations’ and ‘loss of boundary delusions’ (Pearson’s $r=0.43$, $p<0.01$, <19% shared variance), and to a lesser extent, ‘auditory hallucinations’ and ‘paranoia’ ($r=0.28$, $p<0.05$, <8% shared variance), with the presence of ‘grandiosity’ symptoms clearly separable from others. Examining the patterns of correlations between these four symptom groupings and the measures of eye tracking performance (Table 93), there were no
correlations greater than $r=0.1$ (1% shared variance) with single mode gain or positive saccade frequency. In term of reversal saccade frequency, however, ‘grandiosity’, ‘auditory hallucinations’ and ‘loss of boundary delusions’ all showed relationships with the dependent measure of very similar magnitude, with both the degree of ‘auditory hallucinations’ and ‘loss of boundary delusions’ entering the predictive regression equations as significant predictors of performance at different target frequencies (although ‘loss of boundary delusions’ alone entered as a predictor when reversal saccades were averaged across all target frequencies: Table 89). However, the ‘paranoia’ symptom grouping, that was most clearly distinguished from the others phenomenologically (in terms of symptom relationships) did not display this same relationship with the frequency of reversal saccades (Table 93), and as such, there is some suggestion from this study for the independence of ‘paranoia’ from the other groupings.

The internal consistency process in the derivation of the eleven-factor model identified that the three-factor model conception of ‘negative’ symptoms could instead be better considered as three subgroups. As discussed previously (in Study Two), there is substantial evidence for the independence of the ‘cognitive dysfunction’ symptom grouping from the other two subcomponents of this broad group (here defined as ‘negative signs’ and ‘social dysfunctions’) in terms of being poorly related to the other two groupings phenomenologically (sharing less than 6% of variance with either measure: Table 93), and showing strong interrelationships with neuropsychological measures of attention that were not apparent with the ‘negative signs’ and ‘social dysfunctions’ groupings. This differentiation was not as apparent in the assessment of gain: while ‘cognitive dysfunction’ did show a significant relationship with single-mode gain that was not present with either the ‘social dysfunctions’ or ‘negative signs’, and this did remain significant after partialling out the variance associated with other symptom...
groups (Table 78), the inclusion of this variable did not produce a statistically significant improvement in prediction of gain over a null model (using only illness duration as a predictor). The ‘cognitive dysfunction’ grouping did not show a significant relationship with frequency of positively or negatively directed saccades.

The other two subcomponents, ‘negative signs’ and ‘social dysfunctions’, together comprise the ‘negative’ symptom dimension in the five-factor model. These groupings and are closely related phenomenologically (in current cohort: Pearson’s r=0.51, p<0.01, staring approximately 26% of variance), and produce similar magnitude correlations with the frequency of reversal saccades. However, when the relationships with other eye movement measures are examined, the ‘negative signs’ and ‘social dysfunctions’ groupings are clearly differentiated. As shown in Table 93, ‘negative signs’ showed zero-order relationships with single mode gain, while ‘social dysfunctions’ displayed a moderate relationship (r=0.01 and r=0.16 respectively, both p>0.05). Similarly, ‘social dysfunctions’ emerged as a significant predictor of the frequency of positive saccades while ‘negative signs’ did not (Table 93). However, while it should be noted that the use of the ‘social dysfunction’ grouping did not produce a statistically significant improvement over regression models using control measures only (duration of illness and extrapyramidal side effects), these findings do provide some evidence in support of the independence of the ‘negative signs’ and ‘social dysfunctions’ groupings.

Affective-type symptoms were not present in the three-factor model of symptomatology, and were grouped similarly under the five- and eleven-factor models of symptoms. These appeared unrelated to single-mode gain and positive saccade frequency, however, emerged as a statistically significant predictor of reversal saccade frequency (Table 93). While this did not produce a significant improvement in the prediction of reversal
saccades over a control regression model (incorporating the degree of extrapyramidal symptoms only), this still provides some support for the independence of ‘anxious intropunitiveness’ from other symptom groupings examined in the eleven-dimensional model.

The remaining symptoms assessed in the SANS, SAPS and BPRS/PANSS item set are grouped very differently in the three- and five- factor models. The three-factor model ‘disorganisation’ grouping was, under the eleven-factor model, subdivided in the groups that were poorly intercorrelated (‘hostility’, ‘conceptual disorganisation’ and ‘bizarre behaviour’, with none sharing more than 10% of variance: Table 93). Under the five-factor model, this ‘disorganisation’ factor is split into two, namely, the ‘cognitive’ factor and the group of ‘hostility’ symptoms (along with others that were not prevalent enough for reliable examination in the current cohort) forming an ‘excitement’ factor. The symptoms in the ‘cognitive’ factor were poorly intercorrelated (Table 93), and subdivided into groupings of ‘bizarre behaviour’ ‘conceptual disorganisation’ and ‘cognitive dysfunction’ in the eleven-dimension model. Examining the relationships between symptom subgroupings and eye movement measures, in terms of the five-factor model ‘cognitive’ dimension, the ‘cognitive dysfunction’ subgroup was clearly independent from the other sub-groupings of this dimension, being significantly correlated with single-mode gain, while both the ‘bizarre behaviour’ and ‘conceptual disorganisation’ symptom groupings were unrelated with this or any other eye movement variable (Table 93). The symptom subgroups within the three-factor model ‘disorganisation’ dimension, defined in the eleven-dimension model as ‘bizarre behaviour’, ‘conceptual disorganisation’ and ‘hostility’, were all unrelated to any eye movement measure (Table 93), and hence there was no evidence in support of their independence provided by this study.
In conclusion, while there was good evidence within the current cohort for the phenomenological independence of all of the symptom groupings proposed within the eleven-dimension model, not all of these were able to be validated as independent symptom dimensions through examination of relationships with eye movement variables. While the contributions of symptom groupings to the prediction of eye movement variables was in no case sufficient to improve the prediction quality above that afforded by the use of control measures alone (such as illness duration or extrapyramidal symptoms), statistically significant correlations did occur, and provided evidence to suggest that the broad concept of ‘negative’ symptoms in the three-factor model is instead composed of three independent subgroups: ‘cognitive dysfunctions’, ‘social dysfunctions’ and ‘negative signs’ that display distinct patterns of relationships with external variables. Additionally, there was support for the independence of ‘paranoia’ from the other symptoms commonly combined into the broad ‘positive’ or ‘reality distortion’ symptom groupings in the three- and five-factor models; as well as the independence of affective ‘anxious intropunitiveness’ symptoms from others in the SANS/SAPS/PANSS/BPRS item set.

Methodological limitations

While it is often suggested in the literature that eye tracking disorder in schizophrenia is a trait deficit (Clementz & Sweeney, 1990; Iacono, 1998), in the introduction to this study multiple lines of evidence were reviewed that showed that eye tracking dysfunctions were only present in a subset of those diagnosed with schizophrenia (Levy et al., 2000; Malaspina et al., 2002; Ross et al., 2002) and that multiple studies had suggested that some particular syndromes may share underlying pathology with those underlying eye
tracking disorder (Hutton et al., 2004; Lee et al., 2001). In the current study, some such relationships between eye movements and symptom groupings were indeed identified; however, these were uniformly mild, sharing only a minority of variance with the target measures (Table 93). Possible methodological limitations that may have impaired the ability of the current study to identify relationships between symptom groupings and eye movement performance will be discussed briefly under several main headings: the approach adopted for the assessment of eye movements, the nature of the symptom groupings used as predictors; characteristics of the participant group; and the experimental design approach applied.

**Symptom Groupings**

Part of the logic behind the current thesis was that, since there was substantial evidence suggesting that many of the symptom dimensions proposed under the three- and five-factor models of symptomatology were heterogeneous, then correlations between these broad groupings and external variables would be attenuated (due to the broad groupings combining symptoms that, due to their differing cause, were inconsistent in their relationships with the external measure). As such, it was proposed that the use of the more refined symptom groupings identified in the eleven-factor model would enhance any relationships that exist. However, if the groupings defined under the eleven-factor model were themselves also heterogeneous, then this problem would still remain, and relationships with external measures would still be obscured.

It is unlikely, however that this is the underlying cause of the lack of identification with external measures. Firstly, the clustering procedure and the detailed examination of symptom intercorrelation applied in the first study produced highly internally consistent
symptom groupings that were also clinically logical. Secondly, where the broad groupings of symptoms (in the 3- or 5-factor models) showed any useful predictive relationship with the target variables, the correlations between all of the individual symptom measures in this factor and the neuropsychological variable were examined. This did not reveal any example in which a subset of symptoms in the groups defined under the eleven-factor model showed strong external relationships (>10% of shared variance) yet were excluded from predictive equations.

Alternatively, if the symptom groupings defined under the eleven factor model represented over-splitting of symptom dimensions, this would not have obscured any external relationships, as all of the sub-groupings in question would have been identified as significant correlates of the measure in question in the initial correlation matrix. Although there was evidence that some of the eleven factor model groupings that reflected sub-divisions of the simple model factors exhibited uniformly poor relations with external variables (as was the case with the ‘bizarre behaviour’ and ‘hostility’ groups), there was no case where it could be suggested that over-splitting of symptom dimensions that were strongly related with the target variables had occurred.

The nearest example of possible ‘over-splitting’ in the current study arose in the case of the eleven-factor model subdivisions of ‘positive’ symptoms (from the three- and five-factor models) in the prediction of the frequency of reversal saccades (Table 93). Here, three of the four symptom groupings that represented sub-divisions of the ‘positive’ symptom concept showed significant or trend relationships of similar magnitude with the frequency of reversal saccades, however, one (‘paranoia’) did not. This provides evidence in support of the distinction of ‘paranoia’ from the other symptoms within the broad ‘positive’ factor. On the other hand, it is not possible to conclude from the current study
as to whether the distinction made in the eleven-factor model between the remaining three sub-groupings of the ‘positive’ factor (‘auditory hallucinations’, ‘loss of boundary delusions’ and ‘grandiosity’) is appropriate or otherwise.

**Participant Characteristics**

Correlations between variables will be attenuated if there is a poor spread of performance on one of both of the measures under examination (Nunnally, 1967). There are a number of characteristics of the current cohort that may have contributed to the lack of identification of any relationships between the eye movement variables and measures of symptoms. Firstly, the current participant sample was predominantly chronic in nature, with many of those interviewed accessed through community-based treatment services rather than in-patient services. As such, it could be argued that the range of symptomatology experienced among the cohort may have been attenuated. While this was certainly the case for a number of variables (such as the ‘somatisation’, ‘visual hallucination’, ‘excitement’, and ‘inappropriate affect’ symptom groups), these symptom groupings were excluded from analysis in the current study, and there was a reasonable range of severity of experience for each of the included variables (with distribution tables provided in Study 1).

Another aspect that may have affected performance is the presence of antipsychotic medications, or neural changes arising from the effects of chronic administration of antipsychotic medications. While multiple studies have shown that the presence of neuroleptics is unlikely to cause eye tracking disorder in observers with schizophrenia (Levy et al., 1993), there are, however, indications that acute administration of antipsychotics may affect the intricacies of saccadic activity during tracking (Rea et al.,
1989; Spohn, Coyne & Spray, 1988), and gain deficits may be exacerbated either through chronic medication administration (Hutton et al., 2001) or through use of the atypical antipsychotic, clozapine (Friedman, Jesberger & Meltzer, 1992; Litman et al., 1994). The largely chronic nature of the current sample means that the effects of long-term medication are relevant to the current study, and similarly, the vast majority of participants (81 of 95) were receiving treatment with atypical antipsychotic medications. There have been limited studies of the potential effects of these medications on eye tracking performance, and it is possible that these factors may have contributed additional variance to eye tracking performance. While exploratory analyses suggested that there was no relationship between medication level and any of the measures of eye tracking (Tables 77, 81 and 86), if, as suggested by some authors (Friedman et al., 1992; Litman et al., 1994; Rea et al., 1984; Spohn et al., 1988) the presence of medication produces changes in the relationships between saccades and gain, then medication impacts may have an insidious effect on these measures of eye movement that would be difficult to account for through statistical adjustment approaches.

The optimal environment for the current study would have been a diagnostically-broad, recent-onset, neuroleptic-naïve, population-based sample. While replication of the findings identified within the current cohort in such a sample would be recommended, it is extremely unlikely that the lack of identification of poor relationships between those symptoms at the conceptual ‘core’ of schizophrenia and measures of eye tracking performance is simply an artefact of the characteristics of the participants that contributed to this study. As shown in the literature overview at the start of this chapter, while this is the first study to have systematically examined the relationship between performance of all of the components of an eleven-factor model of symptomatology, consistent with the findings of the current study, symptom-task relationships identified in
the literature to date for all measures remained relatively slight regardless of the refinement of the symptom groupings or the characteristics of the participants involved.

**Research design**

Multiple studies have shown that disorders of eye tracking are not present in all of those diagnosed with schizophrenia (Levy et al., 2000; Malaspina et al., 2002; Ross, et al., 2002). The finding that half of the clinical participants, on the basis of a qualitative distinction would be classified as experiencing eye tracking disorder is consistent with several previous studies (Keefe et al., 1989; Levy et al., 2000; Malspina et al., 2002). Such assertions assume that the distribution of eye tracking performance in schizophrenia is unimodal, and simply downshifted in comparison to that apparent amongst neurologically-intact observers, to the extent that half of the clinical participants are so impaired in eye tracking that they perform two standard deviations below the mean of the neurologically-intact observers (Allen, 1997; Sweeney et al., 1994). Importantly, Ross and colleagues (Ross et al., 1996; 1997; 2002) and others (Iacono et al., 1992) have shown that eye tracking disorder in schizophrenia is best characterised by a bimodal distribution, with only one of these distributions reflective of poor eye tracking (encompassing 21-34% of participants with schizophrenia: Ross, 2000), and that those performing in the other distribution show no differences in eye tracking performance on any eye movement measure when compared to neurologically-intact observers (Levy, 2000; Ross et al., 1997).

If this is the case, then it would be more methodologically powerful to examine the relationships between symptoms and eye tracking only among participants that perform within that ‘eye tracking disordered’ distribution, rather than examining a cohort defined
simply on the basis of a diagnosis of schizophrenia. This is because, if symptom relationships do exist with the processes underlying disorders of eye tracking, they will be apparent only amongst this subset of participants displaying eye tracking dysfunction. The symptoms experienced by participants that perform in the non-eye tracking disordered distribution may then simply add extraneous ‘noise’ to the dataset, and hence, when hunting for relationships between eye tracking performance and symptoms amongst the whole cohort, the observable magnitude of any such relationships would be diminished, as they would only be apparent amongst a minority of the sample.

Perhaps driven by the illusory validity of diagnostic category, or by the still open question as to the ‘trait’ nature of eye tracking deficits, to date no studies have examined relationships between symptoms and eye tracking performance among only participants that perform within an ‘eye tracking disordered’ dimension (identified on an empirical basis such as admixture analysis, rather than on an arbitrary basis), although some have examined other aspects of performance among observers identified in such a fashion (Ross et al., 2002). This is an approach that merits investigation in future studies, although one that will demand an extremely large investment in terms of sample size.

*Eye tracking recording technique*

The current study used infra-red oculography (IR) to record eye movements. This is the superior technique of the two most commonly applied approaches, as the other, electro-oculography (EOG) is susceptible to artefact from bioelectric noise (muscle artefacts) and hence less reliable at identifying small-amplitude eye movement events (i.e. a lower resolution compared to IR: Clementz & Sweeney, 1990; Iacono & Lykken, 1983). One of the challenges to interpretation of recordings made through IR, is that the presence of
eye blinks tends to produce artefactual activity in the trace, characteristically being ‘spiky’ or polarity reversal events on the eye position record (Iacono & Lykenen, 1983). These must be removed prior to analysis in order for appropriate interpretation of the eye tracking record, and many groups (including our own) do so on the basis of manual inspection of eye recordings, as the appearance of eye blinks can be variable and hence difficult to develop reliable algorithms for identification. Participants with schizophrenia display an increased rate of eye blinks relative to neurologically intact participants (Amador et al., 1991), and extended periods of recording with IR equipment tends to irritate the eye, leading to automatic blink responses (Iacono & Lyken, 1983).

Vertical EOG, measuring changes in electrical potential that arise from the movement of the lid over the eyeball, allows accurate identification of blinks (Calkins et al., 2001). Calkins (et al., 2001) have shown that the ‘characteristic’ artefact in IR recordings during eye blinking does not always occur, and that blinks may indeed produce a response in IR records that is identical to that produced by saccades. In their study, Calkins (et al, 2001), compared vertical EOG recordings to IR recordings alone in response to 30 seconds of recording of tracking of an 0.4 Hz sinusoidal stimulus. They showed that 41% of eye blinks identifiable in the EOG were not identifiable on the IR recording, and that one-third of eye blinks were misclassified as saccades when using the IR trace alone. In all cases, these artifactual ‘saccades’ were interpreted as positively-directed AS or CUS, and never SWJ or reverse-direction BUS.

In the current study, we gathered IR recordings only. As such, it is likely that these artefacts may have added extraneous variance to the ‘positive’ saccade measure, which may have obscured potential findings in terms of saccade-symptom relationships. Moreover, given that the IR recording system applied in the Calkins (et al., 2001) study
had a markedly superior temporal resolution than that applied in the current study (250 Hz sampling of eye position in the Calkins et al., 2001 study, compared to 100 Hz sampling in the current study), it is possible that this misclassification variance may have been exacerbated further in the current experiment. While this would only impact on the measure of ‘positive saccades’ and not on single-mode gain or reversal saccades, this is a likely source of additional extraneous variance and it would be preferable for future studies to apply both VEOG and IR techniques simultaneously.

Saccade identification

In the current study, saccades were defined as any eye movement greater than $60^\circ$/sec over a spatial distance of $0.2^\circ$ during pursuit. This is a reasonable definition on the basis of the known metrics and neurophysiology of the pursuit and saccadic systems (Wurtz & Goldberg, 1989), and is certainly unlikely to falsely classify pursuit as saccades, given that the pursuit system tracks to a maximum of $100^\circ$/sec (MacAvoy & Bruce, 1995) but tends to only be used at target velocities of less than $30^\circ$/sec (Stark, 1983). However, some investigators, such as Holzman, Levy, Matthyssse and Abel (1997), have argued for a stricter definition of saccades, more specifically defined in terms of the velocity of the target. They argue that a definition such as that applied in the current study ($60^\circ$/sec over an amplitude of $0.2^\circ$) would theoretically allow eye movements as fast as $59^\circ$/sec to be classified as pursuit – at a target velocity of $5^\circ$/sec, this would represent a possible gain of up to 11.8, which would be extremely high. Their argument extends to suggest that if these high gain eye movements are included in measures of average velocity, then this will artificially inflate gain scores; additionally, if moderate-speed saccades are classified as pursuit, these movements would not be included in saccade frequency counts – leading to a possible underestimation of the number of saccades. This would effect the
interpretation of all participants’ eye tracking, but perhaps would disproportionately affect the analyses of those with poor eye tracking.

Most researchers in this area do not present the specifics of their definitions of saccades, however, the Ross group in Maryland define saccades as any movement with a 35°/sec peak velocity, initial acceleration greater than 2000°/s^2, and a minimum duration of 9 milliseconds or greater while tracking a constant velocity target at 16.7°/sec (Ross, Olincy & Radant, 1999). This tolerates gain values of up to 2.10 prior to the classification of a movement as a saccade, and suggests that the threshold applied in the current study (60°/sec) is reasonable given that we examined target speeds up to 35°/sec. Re-analysis of a sub-sample of the current dataset using a lower velocity threshold for saccade identification (50°/sec over 0.2° amplitude: Slaghuis, Hawkes, Holthouse & Bruno, *in press*) showed that this change had negligible effects on gain or saccadic values. While it may be advantageous to apply a more targeted definition of saccades (for example, including an assessment of initial velocity), and a definition that shifts relative to the velocity of the target, this was not possible with the current experimental setup in our laboratory, and it is unlikely that the more general definition of saccadic activity applied here had caused any substantial effect on the variables assessed in the current study.

*Taxonomy of saccades*

Early studies in this field used qualitative inspection of records to classify the eye movements of participants with schizophrenia as either ‘normal’ or ‘disordered’. These studies could identify the presence of disrupted eye tracking, but the global qualitative measures applied were not sufficiently specific to be able to identify whether the differentiating aspect was an abnormality of pursuit or an abnormality *during* pursuit (Abel
& Ziegler, 1988). As was discussed in the introduction to this study, eye tracking is an activity governed by the interaction of the function of pursuit and saccadic eye systems (Eckmiller, 1987; Rashbass, 1961).

Careful investigation of the intricacies of eye tracking have further shown that performance that can be qualitatively categorised as ‘disordered’ represents a combination of reduced gain with corresponding compensatory saccadic activity, as well as intrusions of inappropriate saccadic activity (Levy et al., 2000; Ross et al., 1998). The neurological structures and pathways subserving the pursuit and saccadic systems are at least partially distinct (Hutton & Kennard, 1998; MacAvoy & Bruce, 1995) and saccadic disinhibition underlying intrusive saccades may reflect distraction or fatigue (Abadi & Gower, 2004; Kaufman & Abel, 1986; Nolte et al., 1998). Given this multivariate nature of eye tracking performance, and the suggestions that pursuit, compensatory, and disinhibitive saccades may have distinct underlying mechanisms (Abel & Ziegler, 1988), very specific examination of these individual components is crucial to advancing our understanding of the exact process(es) underlying the deficits seen among some of those individuals diagnosed with schizophrenia (Braff, 1998).

While single-mode gain, representing the functioning of the pursuit system, was adequately separated out in the analyses of the current study, the assessment of saccades derived here were heterogeneous measures that comprised components of both anticipatory and intrusive saccadic activity. The positive saccade variable, all saccades made in the same direction of the target stimulus, comprises variance due to compensatory CUS, as well as intrusive AS, LS and SWJ activity; and the ‘reversal’ saccade measure – saccades in the direction opposite to the target – are a combination of compensatory BUS and SWJ. Interpretation of the meaning of correlates of these
measures was heavily dependent on reports in the existing literature in regard to the relative frequency of each type of saccade that were combined within these measures. As such, both the possibility of identification of external correlates of these measures was compromised by the use of variables with such complex contributions to variance, and the interpretation of the few correlates identified necessarily limited by a lack of clarity in regard to the nature or function of each saccadic measure under examination.

Friedman, Abel, Jesberger, Malki, Brar and Meltzer (1992) have detailed specific amplitude and timing criteria for the definition of each type of saccade occurring during eye tracking in psychiatric patients. However, in recent years Ross and colleagues in Maryland have applied substantially more specific definitions of saccadic activity through the inclusion of criteria including relative position error and post-saccadic slowing, as well as broadening the amplitude range for several measures. The recording equipment available to Ross and colleagues provides a much greater sampling resolution than that available in our laboratory (the Maryland group samples eye movement at 500 Hz, compared to our system which samples at 100 Hz). The highly specific criteria for the identification and classification of saccadic activity proposed by Ross and colleagues requires assessment of movements at the threshold of our recording resolution and as such may be of questionable reliability/validity if applied to these records.

In sum, the restriction of the current study to analyses of saccadic activity during eye tracking simply in terms of frequency of saccadic activity in the same or opposite direction to the target was a limitation driven by practical aspects of our equipment. This has limited the potential for identification of symptom grouping correlations with specific aspects of saccadic activity during eye tracking, and it is recommended that future studies apply specific differentiation of both the frequencies and amplitudes of
each saccadic component during eye tracking, following the explicit criteria of Friedman (et al., 1991) and Ross and colleagues (Ross, Olincy & Radant, 1999; Roy-Byrne, Radant, Wingeresen & Cowley, 1995).

**Summary**

The ability to follow a moving target with one’s eyes is a complex task that requires the involvement of two independent oculomotor control systems: the pursuit system, in order for the eyes to keep pace with the movement of the target; and the saccadic system, to quickly replace the target into central vision should the eye fall behind the target while tracking. Participants with schizophrenia often display difficulty with this task, and, when compared to neurologically-intact controls, tend to display a low gain – that is, their eyes lag behind when following a target; and consequently, also produce greater numbers of compensatory saccades to rapidly refoveate the target (‘catch-up saccades’). Additionally, groups with schizophrenia diagnoses exhibit more frequent intrusions of non-functional saccades during tracking than do control participants, particularly in the case of saccades made to a point ahead of the position of the target (‘anticipatory saccades’). These deficits do not appear to be due to the effects of medication, inattention or clinical phase and are more prevalent among individuals with a diagnosis with schizophrenia than those with other psychiatric diagnoses. Eye tracking disorder may be particularly important for understanding ‘schizophrenia’ itself, as unaffected twins and close relatives of individuals with schizophrenia also display similar performance on this task, suggesting a genetic association that may be useful in endophenotype definition.
However, while eye tracking disorder is common among those with a schizophrenia diagnosis, it is not ubiquitous, with only around half this group also categorised as producing disordered eye tracking on the basis of qualitative examination of eye movement recordings. More sophisticated analyses suggest a bimodal distribution of performance in schizophrenia, whereby 21-34% perform within a distribution that is extremely poor in eye tracking quality, with the remaining patients performing on a comparable level with neurologically-intact controls. In attempting to identify the factors that differentiate patients with good and poor eye tracking, several studies have identified moderate associations between the presence of negative symptoms and the global quality of eye tracking performance. This literature has, however, been mixed, with multiple replication failures and others suggesting that only sub-groups of ‘negative’ symptoms, or that ‘disorganisation’ and not ‘negative’ symptoms, are related to eye tracking performance.

In this study, we set out with the implicit hypothesis that the mixed findings in the literature to date in regard to symptom relationships with eye tracking performance were due to the application of coarse psychopathological models (the three- and five-factor models) which applied heterogeneous groupings of symptoms as dependent measures, which may obscure the identification of potential relationships. On the basis of the limited findings in the existing literature, it was hypothesised that eye tracking performance would be most related to the ‘cognitive dysfunction’, ‘negative signs’ and ‘conceptual disorganisation’ groupings defined according to the eleven-factor model of schizophrenia identified in the first study of this thesis.

Eye tracking in response to a constant velocity target, at seven individual target speeds between 5 and 35°/sec, was examined in a medicated, mixed in- and out- patient group
of 95 largely chronic observers with schizophrenia (as well as 102 neurologically intact comparison participants). Half of the clinical sample was classified as having qualitatively disordered eye tracking, and there were no differences apparent between these patients in terms of demographic or clinical characteristics, although those with disordered eye movements exhibited poorer general intellectual performance and ‘cognitive dysfunction’ as defined in the eleven-factor model of symptomatology.

Clinical participants produced generally lower gain when compared to control observers at high target speeds, and the degree of ‘cognitive dysfunction’ was the only symptom group – under any of the 3-, 5- or 11-factor models – to predict performance. However, these symptoms only related to performance at moderate target speeds (15-35°/sec). Positively-directed saccades, largely but not exclusively compensatory in nature, were more frequent among patients than controls and most common when the target was moving slowly. The only symptom grouping predicting frequency of positively-directed saccades was the degree of ‘social dysfunctions’ defined in the eleven-factor model, but this only related to performance at fast velocities, where there was little difference between the performance of patients and controls. Finally, saccades in the direction opposite to the target during tracking, representing both compensatory and disinhibitory aspects of saccade performance were infrequent in both patients and controls but most common when the target was moving slowly. The presence of these saccades was predicted by the degree of affective symptoms (and anxiety in particular), and the ‘positive’ symptom groups of ‘loss of boundary delusions’ and ‘auditory hallucinations’.

These results provided external validation for some aspects of the eleven-dimensional model of symptomatology. Firstly, the eleven-factor model symptom groupings of ‘cognitive dysfunction’ and ‘social dysfunctions’ emerged as significant predictors of gain.
and positively-directed saccades respectively. These relationships were totally obscured when these symptoms were combined into the broader groupings defined in the three- and five-factor models of psychopathology. Secondly, of the diverse group of symptoms defined as ‘positive’ symptoms in the three- and five-factor models, only loss of boundary delusions and auditory hallucinations related to the presence of reversal saccades, however, paranoia did not. This is consistent with the phenomenological findings of the first study of this thesis, that suggest that the three- and five-factor models mask heterogeneity within their symptom groupings, with the broad ‘negative’ dimension encompassing three independent subgroups that display differential external relationships; and that ‘paranoia’ is independent from other delusions and hallucinations that are commonly grouped together.

However, while the use of the eleven-factor model allowed for the identification of relationships that were otherwise obscured when more parsimonious models of symptoms were applied, these identified relationships did not significantly improve the prediction of eye tracking variables over comparison equations (using duration of illness or extrapyramidal effects as predictors) as the symptom relationships with eye tracking were uniformly moderate in magnitude.

The characteristics of the experimental equipment limited the definitions of saccadic activity examined here to heterogeneous combinations grouped on the basis of their direction. As compensatory and intrusive aspects of saccadic activity during eye tracking likely reflect the functioning of distinct neural systems, greater clarity of interpretation, and possibly more robust symptom relationships, may be identified if more specific analyses are performed.
Finally, this is one of very few studies that have examined eye tracking performance among participants with schizophrenia as a function of target velocity. A peripheral finding of the current study was that saccadic activity during pursuit is greatest at low target velocities (5 and 10°/sec), despite their being little need for compensatory saccadic activity at these speeds. This additional activity is likely to reflect intrusive saccades, which is particularly relevant given recent findings that anticipatory intrusive saccades may have the most potential for clear endophenotype definition (Ross et al., 2002). Most studies to date examine eye tracking at moderate target velocities (15 to 25°/sec), and it is hence recommended that analysis of performance at lower velocities may induce greater levels of saccadic activity, and hence maximise the experimental power of studies in this area.
Summary: Overview of findings and future directions

“Insanity is doing the same thing over and over again and expecting different results”
- Albert Einstein

Study Rationale

Numerous accounts open their description of schizophrenia declaring it a ‘heterogeneous disorder’ or as being ‘characterised by extreme heterogeneity’. Yet these same articles tend to directly follow such statements of caution with the use of terminology that implies discussion of a disorder that is clear, discrete and valid – which is a situation far from clinical reality (Andreasen et al., 1995; Boyle, 1990; Peralta & Cuesta, 2000 van Praag, 1997). Take, for example, the contradiction – and understatement – inherent in this excerpt from the DSM-IV guidebook:

“Schizophrenia is a clinical syndrome of unknown aetiology and pathophysiology. No symptoms are pathognomonic to schizophrenia, and there are no clinically useful laboratory or imaging markers. The characteristic features of schizophrenia are heterogeneous, and the boundaries with other disorders can be difficult to delineate. All of this makes for quite a diagnostic challenge, as well as creating problems for research.”

(Frances, First & Pincus, 1996: Guidebook for the DSM-IV, p. 166, emphasis added)
This thesis was designed to examine this ‘heterogeneity problem’ in schizophrenia (Heinrichs, 2001), and in particular, to investigate the validity of a low-order dimensional approach to this diagnostic concept.

A review of the literature showed that the DSM-IV concept of schizophrenia does provide a reliable definition, but one that is not concordant with other definitions in use around the world (such as the ICD-10), nor with historical definitions of the concept (Daradekeh et al., 1997; McGorry et al., 1995; Peralta & Cuesta, 2000). Moreover, it is a definition without a theory (APA, 1994), composed of remnants of historical Kraepelinian, Bleulerian and Schneiderian concepts without fully supporting any (Peralta & Cuesta, 2000), and, as mentioned above, an unvalidated concept with unclear borders from other psychiatric disorders (van Praag, 1997). Importantly, the pragmatic adoption of polythetic inclusion criteria in the DSM-IV and ICD-10 systems actively enshrines the heterogeneity that is so problematic to research progress in this area (Jablensky, 1999; Rosenman et al., 2000).

There are three major ways of conceptualising the cause of heterogeneity within the diagnostic category of schizophrenia: as the pleiotropic manifestations of a single disease process; as the results of multiple disease processes, each with unique pathophysiologies, leading to the general manifestations of ‘schizophrenia’ (subgroups of the disorder); or that ‘schizophrenia’ represents the composite of a series of sign and symptom complexes, each with an independent pathophysiological process (Buchanan & Carpenter, 1994). It is this latter approach – a dimensional one – that was examined in the current thesis. The dimensional approach to addressing heterogeneity divides the symptoms experienced by individuals diagnosed with schizophrenia – rather than the individuals themselves – into groups, on the assumption that these symptom groupings
may represent possible ‘syndromes’ with independent pathologies. This approach is enticing because it is in step with clinical practice (for example, medications such as lithium and dopamine agonists are equally efficacious at ameliorating elevated mood, delusions or hallucinations regardless of diagnosis: Johnstone et al., 1988); has the potential to cut across diagnostic boundaries where similar symptoms occur in different categorical diagnoses; is consistent with current genetic models of familial transmission of schizophrenia (Tsuang, Stone & Faraone, 2000); places symptoms on a spectrum from ‘normality’; and, importantly, allows a more informative portrait of the clinical situation of an individual, when compared to the use of gross diagnostic labels.

Dimensional models of schizophrenia have been increasing in popularity over the past two decades. Crow’s (1980) articulation of a two-dimensional hypothesis (‘positive’ and ‘negative’ syndromes) helped lead to advances in the neural underpinnings and, importantly, in the medications available for the treatment of ‘schizophrenia’. The development of assessment inventories designed directly to examine Crow’s hypothesis: the SANS, SAPS (Andreasen, 1981; 1983) and PANSS (Kay, Opler & Fiszbein, 1986), led to the revision of this model and three ‘syndromes’: ‘positive’ (reality distortion), ‘negative’ and ‘disorganised’ (as well as the, less prominent, extension of these with additional affective and ‘excitement’ ‘syndromes’ when applying the PANSS) are commonly currently advanced as an adequate description of the processes occurring in ‘schizophrenia’ – and have been proposed for enshrinement in the DSM-V.

These ‘syndrome’ models have achieved an impressive replicability, which has in turn served to obscure their important limitations: firstly, they are developed from symptom inventories that do not necessarily examine a representative array of symptoms; secondly, these inventories explicitly combine the assessed symptoms into ‘subscale’ groupings that
are assumed to be heterogeneous by the authors, despite this not being consistent with empirical findings – and these are used as the ‘building blocks’ of the ‘syndrome’ models; and finally, compounding these challenges, there appears to be an under appreciation of the limitations of the statistical techniques applied to the examination of relationships between these heterogeneous symptom subscale groupings (Stuart, Pantelis, Klimidis & Minas, 1999). As such, the dimensional approach to addressing the heterogeneity of schizophrenia is extremely promising but has been poorly implemented: critically, if these ‘syndromes’ indeed mask heterogeneity, then research applying these will suffer the same failings as the use of the parent diagnosis of ‘schizophrenia’, an outcome which contradicts the very reason for the application of these dimensional models.

However, there is a literature of dimensional research into schizophrenia that first emerged more than five decades ago, but has apparently been lost to the memory of schizophrenia research until a revival in very recent years. These pioneering authors developed syndrome models that did not suffer from any of the problems plaguing more modern research: applying detailed symptom inventories that were broad in scope and with a careful understanding of the statistical concepts involved. A review of the findings of this early literature identified – instead of three – at least 12, and possibly 17, observable ‘syndromes’ present within the clinical pictures of those experiencing psychoses. The relatively small modern literature that has similarly risen to the challenge of complexity has also identified at least 11 ‘syndromes’ within those diagnosed as experiencing functional psychosis.
Study One: Phenomenology

Study one attempted to follow in the tradition of authors such as Lorr, Stuart, Peralta and Cuesta by examining the symptom interrelationships present amongst a sample of 100 participants with a schizophrenia spectrum diagnosis. Participants were recruited from in- and out-patient services and interviewed with a semi-structured instrument that comprised items from the SANS, SAPS, BPRS-E and PANSS symptom sets. Cluster analysis was applied to the symptom correlation matrix to identify potential groupings of symptoms and these subjected to careful examination of homogeneity and internal consistency. Eleven homogeneous and clinically recognisable symptom groupings were identified as potentially independent clusters, with a further four groupings possible but poorly represented in the cohort. With reference to the predominantly espoused three ‘syndrome’ model, these represented a subdivision of the ‘negative syndrome’ concept into three groupings, labelled ‘negative signs’, ‘social dysfunctions’, and ‘cognitive dysfunction’; division of the ‘disorganisation syndrome’ into three: defined as ‘hostility’, ‘conceptual disorganisation’ and ‘bizarre behaviour’ (with a further two possible groupings: ‘excitement’ and ‘inappropriate affect’); and a split of the ‘positive syndrome’ into four more tightly defined groupings: ‘grandiosity’, ‘auditory hallucinations’, ‘loss of boundary delusions’ and ‘paranoia’ (with a possibility of additional ‘somatisation’ and ‘visual hallucinations’ groupings). Affective symptoms formed an additional independent group (labelled ‘anxious introjectiveness’).

A review of the literature showed that these eleven symptom groupings proposed here were potentially generalisable beyond the current participant sample, as they were consistent with the findings of studies of symptomatology conducted in first-episode schizophrenia and psychosis, among medicated and medication-free patients, in acute, chronic and mixed, population-representative patient samples, using different symptom
inventories, different statistical techniques and participants from around the world. Importantly, they also showed historical continuity with the early literature in this area, with 9 of the 11 groupings very similar to those identified by Lorr and colleagues in research in the 1960s (Lorr, McNair & Klett, 1966).

However consistent these groupings may be, their identification is not an end in itself. The rationale for examining which symptoms tend to co-occur assumes that these will also share pathophysiological mechanisms distinct from those of other symptom groupings. The identified relationships remain simply descriptive until they can be shown to have conceptual, clinical, diagnostic or biological meaning (Andreasen et al., 1995). As such, in order to be a useful description of symptomatology, the validity of these fine symptom grouping distinctions needs to be empirically demonstrated. In particular, they need to be shown to provide a superior account of symptomatology in comparison to the better-established and more parsimonious three- and five-factor symptom models. The second and third studies of this thesis were designed to directly examine the validity of the eleven symptom groupings through investigation of their relationships with two areas of deficit that appear particularly relevant to ‘schizophrenia’ (Heinrichs, 2001) – attentional dysfunction and disruption of pursuit eye movement.

**Study Two: Neuropsychology**

Disorders of attention have been long been reported among individuals diagnosed with schizophrenia, and are one of the strongest variables in differentiating patients from controls in meta-analytic studies of effect size (Heinrichs, 2001), so much so that they have been proposed as possible associates of the schizophrenia genotype. However, ‘attention’ itself is a complex concept, a composite outcome of the functioning of
multiple sub-systems, and performance on tests of attention is heterogeneous amongst those diagnosed with schizophrenia. Recent meta-analytic studies have shown that performance on specific aspects of attention may be related to particular ‘syndromes’ within schizophrenia, rather than being a trait characteristic of the diagnosis itself. These relationships are typically identified between the ‘negative’ or ‘disorganised’ dimensions of the three-factor symptom model (or the ‘cognitive’ grouping in the five-factor model) and measures of attention, however they are largely weak to moderate in magnitude. In this study, we set out with the implicit hypothesis that the weak to moderate correlations identified between aspects of attention and symptoms were due to the use of coarse psychopathological models which apply heterogeneous groupings of symptoms as dependent measures, hence obscuring potential relationships.

The ‘negative’ component of the three-factor symptom model was related to neuropsychological measures of several components of attention. Under the eleven-dimension model of symptoms identified in the first study, this ‘negative’ concept was subdivided into three groups, and it emerged that the relationship identified with the neuropsychological measures was almost entirely due to one of these three subgroups alone – the degree of ‘cognitive dysfunction’. Moreover, application of this group of symptoms alone produced a significantly better prediction of performance than could be identified through using a broader definition of ‘negative’ symptoms. As such, this validated the independence of the ‘cognitive dysfunction’ grouping from other components of the eleven-factor model.

Applying the eleven-factor model of symptomatology, two other symptom groupings, namely ‘hostility’ and ‘social dysfunctions’ were shown to contribute significantly to the prediction of neuropsychological performance independent of others. However, careful
examination of these relationships in light of the existing literature in regard to neurocognition and functional outcome (Green & Nuechterlein, 1999) suggested that the experience of symptoms in these two groupings were likely to be consequences of neuropsychological dysfunction, rather than the cause of these relationships. As such, to the extent that the ‘cognitive dysfunction’ dimension of the eleven-factor model is a valid index of neurocognition, the ‘hostility’ and ‘social dysfunction’ groupings in this model may simply be secondary factors: important for a description of the full clinical picture of patients but not as targets in biological research. The validity of the remaining symptom groupings proposed in the eleven-factor model remains an open question.

**Study Three: Psychophysicsology**

The ability to follow a moving target with one’s eyes is a complex task that requires the involvement of two independent oculomotor control systems: the pursuit system, in order for the eyes to keep pace with the movement of the target; and the saccadic system, to quickly reposition the target into central vision should the eye fall behind while tracking. Participants with schizophrenia often display difficulty with this task, and, when compared to neurologically-intact controls, tend to display a low gain – that is, their eyes lag behind when following a target; and consequently, also produce greater numbers of compensatory saccades to rapidly refoveate the target. Additionally, groups with schizophrenia diagnoses exhibit more frequent intrusions of non-functional saccades during tracking than do control participants. Disorders of eye tracking have been proposed as an ‘endophenotype’ for schizophrenia as close relatives of those with the diagnosis often exhibit similar problems. However, while eye tracking disorder is common among those with a schizophrenia diagnosis, it is not ubiquitous, and a bimodal distribution of performance is apparent, whereby 21-34% of patients perform within a
distribution with extremely poor eye tracking quality, while the remainder perform on a comparable level with neurologically-intact controls (Ross, 2000). In attempting to identify the factors that differentiate patients with good and poor eye tracking, several studies have identified moderate associations between the presence of negative symptoms and the global quality of eye tracking performance. However, this literature has been mixed, with multiple replication failures and others suggesting that only subgroups of ‘negative’ symptoms, or instead that ‘disorganisation’ symptoms, are related to eye tracking performance. It was hypothesised that the use of the more homogeneous eleven-factor model would identify a clearer pattern of relationships with eye-tracking variables.

Eye tracking in response to a constant velocity target was examined at seven individual target speeds between 5 and 35°/sec. These results provided external validation for some aspects of the eleven-dimensional model of symptomatology. Firstly, the eleven-factor model symptom groupings of ‘cognitive dysfunction’ and ‘social dysfunctions’ emerged as significant predictors of gain and the largely compensatory positively-directed saccades respectively. These relationships were totally obscured when these symptoms were combined into the broader groupings defined in the three- and five-factor models of psychopathology. Additionally, saccades in the direction opposite to the target during tracking, representing both compensatory and disinhibitory aspects of saccade performance, were infrequent but predicted by the degree of ‘anxious intropunitiveness’ symptoms. Secondly, of the diverse group of symptoms defined as ‘positive’ symptoms in the three- and five-factor models, loss of boundary delusions and auditory hallucinations related to the presence of reversal saccades, however, paranoia did not.
These findings are consistent with the phenomenological findings of the first study of this thesis that suggest that the three- and five-factor models mask heterogeneity within their symptom groupings: as the three subgroups of the broad ‘negative syndrome’ grouping displayed differential external relationships; and ‘paranoia’ appeared independent from other delusions and hallucinations that such symptoms are commonly grouped with in the broad ‘positive’ dimension. However, while the use of the eleven-factor model allowed for the identification of relationships that were otherwise obscured when more parsimonious models of symptoms were applied, these identified relationships did not significantly improve the prediction of eye tracking variables over comparison equations (using duration of illness or extrapyramidal effects as predictors) as the symptom relationships with eye tracking were uniformly moderate in magnitude (although there were some characteristics of the experimental equipment that may have limited the identifications of relationships).
Summary of evidence for the validity and independence of symptom groupings

A summary of the symptom groupings identified by the current thesis and the evidence in support of these is provided in Table 94 below.

Table 94: Summary of evidence in support of the independence and external validity of the symptom groupings identified in the current thesis

<table>
<thead>
<tr>
<th>Symptom groups identified in current study</th>
<th>Study I. Phenomenological</th>
<th>Study II. Neuropsychological</th>
<th>Study III. Psychophysiological</th>
<th>Support for independence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Paranoia</td>
<td>✓</td>
<td>?</td>
<td>(✓)</td>
<td>Tentative support</td>
</tr>
<tr>
<td>Grandiosity</td>
<td>✓</td>
<td>?</td>
<td>?</td>
<td>Possible over-splitting</td>
</tr>
<tr>
<td>Auditory Hallucinations</td>
<td>✓</td>
<td>?</td>
<td>(✓)</td>
<td>Unclear, shares relationships with loss of boundary delusions</td>
</tr>
<tr>
<td>Loss of Boundary Delusions</td>
<td>✓</td>
<td>(✓)</td>
<td>(✓)</td>
<td>Unclear, shares relationships with auditory hallucinations</td>
</tr>
<tr>
<td>Visual Hallucinations</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Not examined</td>
</tr>
<tr>
<td>Somatization</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Not examined</td>
</tr>
<tr>
<td>Negative Signs</td>
<td>✓</td>
<td>(✓)</td>
<td>(✓)</td>
<td>Support</td>
</tr>
<tr>
<td>Social dysfunctions</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>Support, but possibly secondary</td>
</tr>
<tr>
<td>Cognitive dysfunction</td>
<td>✓</td>
<td>✓</td>
<td>✓</td>
<td>Strong support</td>
</tr>
<tr>
<td>Bizarre behaviour</td>
<td>✓</td>
<td>?</td>
<td>?</td>
<td>Possible over-splitting</td>
</tr>
<tr>
<td>Conceptual disorganisation</td>
<td>✓</td>
<td>?</td>
<td>?</td>
<td>Possible over-splitting</td>
</tr>
<tr>
<td>Hostility</td>
<td>✓</td>
<td>✓</td>
<td>?</td>
<td>Support, but possibly secondary</td>
</tr>
<tr>
<td>Excitement</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Not examined</td>
</tr>
<tr>
<td>Inappropriate affect</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>Not examined</td>
</tr>
<tr>
<td>Anxious intropunitive</td>
<td>✓</td>
<td>(✓)</td>
<td>✓</td>
<td>Support</td>
</tr>
</tbody>
</table>

Key: ✓: entered predictive equations, but as a minor contribution to variance; (✓): did not enter predictive equations, but appeared to relate differently to external variables when compared to others within the broad grouping of symptoms defined under the three-factor model; ? no evidence available; ✓: evidence for independence; (✓): clear evidence for independence
The eleven symptom groups identified in the current cohort using the items from the SANS, SAPS, PANSS, and BPRS-E inventories are consistent with multiple independent studies, many of which have identified similar structures despite investigating cohorts at different illness phases, using different symptom instruments, and applying different statistical techniques. Several of these symptom groupings have been demonstrated to have independent relationships with external measures in the current series of studies, as well as in the existing literature (Barta et al., 1990; Erkwoh et al., 1999; Flaum et al., 1995; Kwon et al., 1999; Strauss, Carpenter & Bartko, 1974; Vita et al., 1995).

In defining a ‘syndrome’ it is explicitly assumed that all the symptoms comprising this group are manifestations of a single pathology, and will therefore share equal relationships with external variables. In direct contradiction to the ‘syndrome’ status of the groupings defined in the three- and five-factor models, several of the subgroupings of these (‘cognitive dysfunction’, ‘social dysfunctions’, ‘hostility’ and ‘paranoia’) displayed relationships with external variables that were independent of the other symptoms they were combined with under the coarse factor models. The finding that the ‘syndromes’ of the three- and five-factor models in fact harbour heterogeneity is particularly important as, where correlates are shown, it is often assumed that the relationships are reflective of the causes of the symptoms at the conceptual ‘core’ of these groupings (for example, affective blunting in the core of the ‘negative syndrome’ or formal thought disorder as the core of the ‘disorganisation syndrome’), when it may, as was demonstrated here, relate only to a subgroup of symptoms within that category (Stuart et al., 1999). In further support of the tightly-defined, homogeneous groupings of the eleven-dimension model, use of these groupings, despite their inclusion of variance from a smaller number of symptoms, actually produced statistically significant improvements in the prediction of
external variables when compared to the predictions possible using the more parsimonious symptom models.

The strongest lessons from the current study are in regard to the broad ‘negative syndrome’ concept in the three-factor model (likely reflecting the predominantly chronic nature of the current participant sample), as the results of Studies Two and Three demonstrated that the ‘cognitive dysfunction’ grouping of symptoms is clearly responsible for much of the external relationships that emerge when the broad ‘negative’ grouping is applied. ‘Social dysfunctions’ also appear distinct from ‘negative signs’ despite their moderate phenomenological intercorrelation, as a dissociation of sorts was identified between these (given that ‘social dysfunctions’, but not ‘negative signs’, were related to eye movement variables). The implications for the three-factor model ‘positive syndrome’ construct was less clear, but there is certainly phenomenological and some external suggestions that ‘paranoia’ is independent of others in this broad group, while other proposed subgroupings (‘auditory hallucinations’; ‘loss of boundary delusions’; and, to a lesser extent, ‘grandiosity’) appeared to share similar relationships with the external measures. Finally, in terms of the ‘disorganisation’ three-factor grouping, ‘cognitive dysfunctions’, which sometimes forms part of this definition, is clearly distinct from these symptoms; likewise ‘hostility’ appears independent, although is possibly an assessment of secondary clinical problems; while little can be concluded in regard to the validity of the distinction between ‘conceptual disorganisation’ and ‘bizarre behaviour’ from these studies.

Three clear statements can be made from these findings in relation to the popular three-factor model of symptoms. Firstly, the three-factor model is inadequate in terms of the scope of symptoms that it describes. Secondly, the application of the three-factor model,
rather than facilitating research, actively undermines the identification of relationships between ‘syndromes’ and underlying mechanisms. This has been suggested as a possible consequence of the limitations of this model by some authors (Cuesta & Peralta, 2001; Stuart et al., 1999), but has been explicitly demonstrated in the current thesis. Finally, the application of the three-factor model can actually imply misleading relationships with external variables - due to correlations being identified with only a subgroup of symptoms contained within a heterogeneous dimension.

The eleven symptom groupings identified in the current thesis and similar literature (Cuesta & Peralta, 2001; Lorr, 1986; Stuart et al., 2002) improves on the three-factor model by expanding the breadth of coverage of symptoms to better reflect the complexities of clinical presentations. Moreover, by identifying more homogeneous groupings, this has improved predictive power, and also produced superior phenotype candidates, than those provided by the more ‘parsimonious’ symptom models. This has been produced through the disentangling of the independent groupings present within the ‘syndromes’, and also by differentiating symptom groupings that may be primary and secondary manifestations of underlying neural dysfunctions (for example, differentiating ‘cognitive dysfunctions’ and ‘hostility’).

It is important to make clear that the author is not contending that these eleven-dimensions reported here represent a definitive model of the ‘syndromes of schizophrenia’. It is only proposed that this provides a superior approach to the conceptualisation of the relationships between the symptom items in the SANS, SAPS, BPRS-E and PANSS among these participant cohorts than is produced through the more ‘parsimonious’ three- and five- factor models that are currently de rigueur in schizophrenia research.
Future directions

With complex models of psychopathology consistently emerging from multiple independent research teams, it is clearly time to look towards the validation of these constructs. In order to do so, it needs to be determined as to whether the different symptom groupings correspond to distinct psychopathological mechanisms (Cuesta, Peralta, Gil & Artamendi, 2003). As part of this approach, all aspects of the model should be explicitly targeted, applying an approach similar to the ‘double dissociation’ methodology applied in neuropsychology (Lezak, 1995). The current thesis took the very difficult route of examining relationships between symptoms and deficits that are considered ‘endophenotypic’ of schizophrenia (attention and eye tracking dysfunctions) in order to determine external validation for the model. These are existing theories about the evolution of particular symptom groups (for example, Frith, 1992; Harvey, 2000; Gilloen & David, 2005), and targeted research programs could be developed using these theories to demonstrate distinction in cause between these symptom groupings and others in the eleven-dimension model. Preferably, these studies should be carried out among unmediated cohorts experiencing a first episode of psychosis in order to maximise the power of the research design.

While the symptom groupings identified here have also emerged in studies of symptom structure among cohorts assessed at all phases of illness (first episode, chronic and mixed), these remain beset with the limitations of cross-sectional studies. The symptom groupings identified here require replication in prospective, longitudinal studies, in samples sufficiently large to determine the independence of the symptom groupings that were only able to be suggested in the current cohort (‘excitement’, ‘inappropriate affect’, ‘visual hallucinations’ and ‘somatisation’). Similarly, the symptom structure requires investigation with confirmatory factor analysis to determine the goodness of fit of these
models to the complexities of clinical reality. Additionally, it would be very advantageous to implement more sophisticated investigations of the structure of these symptom groupings in order to, in van Praag's (1997) terms, ‘verticalise’ this model – differentiating primary and secondary symptom combinations. Cuesta and Peralta (Cuesta & Peralta, 2001; Cuesta et al., 2003) have begun this process with their proposals of symptom hierarchies, whereby phenotypic targets at different levels of complexity can be selected depending on the requirements of the research program involved.

Furthermore, these complex symptom models should be extended to large and broad psychiatric samples in order to determine the potential for these to cut across existing categorical diagnostic boundaries. This line of research has already been initiated by several groups (Peralta & Cuesta, 1999; Cuesta & Peralta, 2001; Stuart et al., 2002; Toomey et al., 1997), and holds great promise for conceptualising all present diagnoses in a dimensional rather than categorical framework. As part of this process, the symptom groupings identified here, and in other, comparable research, should be used as starting points from which to begin the process of improving the representativeness and content of instruments available for the assessment of psychosis, in much the same way that the original work of Lorr and colleagues led to the development of the BPRS (Overall & Gorham, 1962). The scales that are predominantly used in schizophrenia research currently were designed explicitly in order to examine the positive and negative ‘syndromes’, and the spectre of these concepts continues to pervade research adopting these instruments (Peralta & Cuesta, 2000; Stuart et al., 1999).

Finally, the identification and validation of these fine-grained symptom groupings suggests a new ‘default analysis’ for schizophrenia research (c.f. Kirkpatrick & Ryan, 2000): where significant relationships are identified between symptom groupings using
broad ‘syndrome’ constructs (such as the three- and five- factor models) and dependent measures, these should be examined for their consistency across all of the sub-groupings suggested by the current thesis and related literature. For example, in any study applying the three factor model, if symptom relationships are identified between negative symptoms and a dependent measure, then the relationships between ‘negative signs’, ‘social dysfunctions’ and ‘cognitive dysfunction’ symptom items should be also examined, in order to ensure that the correct interpretations are made in regard to these findings (for example ensuring that the relationships with the broader ‘negative’ grouping are not solely due to a subgroup of symptoms).
The rationale for the push toward greater complexity

“The causes of dementia praecox are at the present time still wrapped in impenetrable darkness” (Kraepelin, 1913, p. 224)

“We have entered the second century of schizophrenia research. Despite a massive accumulation of data on brain structure and function, and our luck in inventing more friendly drugs, the understanding of the pathologic mechanisms is still very limited and does not mirror the invested work. The crucial problem seems to me to reside in the inadequacies of psychopathology as it is theoretically conceptualised and de facto practiced...the operational diagnostic criteria, usually considered a decisive step forward on the way to truth, have created an atmosphere of illusory diagnostic precision and reliability” (Parnas, 2000, p. 415).

Revisiting the quotations that opened this manuscript, it is a sobering reminder that, despite the forest of studies examining ‘schizophrenia’, we have made comparatively little headway in more than a century of research. Heinrichs’ (2001) meta-analysis of effect sizes in 54 research literatures (neurobiological, psychophysiological and cognitive) identified no deficits that occurred in all those with the diagnosis, with the effects of largest magnitude, for dysfunctions of eye movement and attention, only moderate in effect size, suggesting relative deficits in 50-70% of patients. As noted in the reviews of these literatures in the second and third studies of this manuscript, even these ‘robust’ findings give way to heterogeneity, and Heinrichs (2001) concludes that close to 40% of all of the biological findings in the reviewed literature are so weak as to represent “minor, unimportant and chance abnormalities with no intrinsic link to schizophrenia” (p. 259).
In the face of all this evidence, is psychiatric research, as suggested by the Einstein quotation in the opening of this section, ‘doing the same thing over and over again, and expecting different results’? Is the commitment to a single disease process model, despite there being no serious evidential support for its validity, actively harming the progress of research in this field (Heinrichs, 2001; Poland, *in press*; van Praag, 1997)? After all, calls for the reconsideration of the concept of ‘schizophrenia’ are not new, and have been raised consistently, both at the socio-political (Foucault, 1988; Laing, 1965; Szasz, 1970) and scientific levels (Bentall, 1990; Boyle, 1990; Maj, 1998; Rodnick, 1997; Sarbin, 1990; Tsuang, Stone & Faraone, 2000). The majority of research in this field, however, appears to accept what Heinrichs (2001) terms the ‘promissory note’ view of the existing evidence: the implicit assumption is made that our conception of ‘schizophrenia’ is accurate, and if the prevailing conception is consistently applied the causes of the disorder will emerge once our knowledge of the brain, and the methods available for its’ study, have advanced. While the sciences of genetics and neuroscience may indeed be relatively immature, the refusal to seriously challenge the ‘schizophrenia’ concept – when there are viable alternatives – may very well be a larger contributing factor than neurobiological methodology to the lack of research progress made in the last century (Peralta & Cuesta, 2000; van Praag, 1997). Clearly, if the inconsistency and weakness of the existing evidence is due to the existence of different kinds of illness captured within the ‘schizophrenia’ concept, then continuing to study such etiopathologically diverse conditions as a single disease state is guaranteed to continue the output of inconsistent findings (Heinrichs, 2001; van Praag, 1997).

Problematically, the minority of researchers that have considered these challenges and adopted a dimensional, multiple disease process model of ‘schizophrenia’ appear to have
settled on a syndromal model that is seductive in its simplicity, but limited in its empirical validity (Peralta & Cuesta, 1999). Adoption of the three- or five-dimensional models will short-circuit advances in research in the very same way as would the maintenance of a unitary disease concept of ‘schizophrenia’ as the heterogeneity problem remains encapsulated within the very models that are designed to address it.

Studies and reviews questioning the methodology and assumptions of the three-syndrome (and five-syndrome) model have appeared (Peralta & Cuesta, 2001; Stuart et al., 1999) but are drowned out by the sheer weight of studies seeking simple methods for reducing the complexity of the challenge presented by ‘schizophrenia’. Moreover, the illusory legitimacy of these models increases with each meta-analytic review (for example: Grube, Bilder & Goldman, 1998; Smith, Mar & Turoff, 1998) and mention in strong secondary sources such as the DSM and psychiatric texts (Boyle, 1990). Ashamedly, the criticisms of these models raised in this thesis and by others are not new, and were problems that were addressed in the careful, pioneering work of early researchers in this field (such as Wittenborn, Lorr, Klett, McNair and Lasky), and our psychopathological conceptions would likely be more advanced had subsequent research followed in their traditions.

The stakes are high. The experience of psychosis has a profound and devastating effect on people’s lives. We owe it to the afflicted, their families and loved ones not to continue this pattern of limited gains into the next century of research. Success in improvement of treatment and research depends largely on an accurate understanding of cause (Tsuang et al., 2000), and this is likely to remain in the ‘impenetrable darkness’ until the heterogeneity at the clinical level can be resolved (Peralta & Cuesta, 2000; van Praag, 1997). It is absolutely critical that we challenge the inertia of our well-entrenched
practises (Poland, *in press*) and smash through our current barriers to progress. A vigorous path of research adopting a rigorously-based dimensional approach to ‘schizophrenia’, and psychosis generally, offers a recipe for such a revolution (Poland, *in press*) that is long overdue.
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*New Trends in Experimental and Clinical Psychiatry, 10*, 75-83.


Appendix 1: Semi-structured Interview Schedule
Participant Code Number____________

Date of Assessment _______________

Standardised Clinical Interview Forms

Covering:

Background Demographic Information

PANSS (Kay, Opler & Fiszbein, 1992)  SOS (Perkins et al, 2000)
SAPS (Andreasen, 1983)              SDS (KirKPatrick et al, 1988)
BPRS-E (Lukoff, Nuechterlein & Ventura, 1986)  EPSE (Simpson & Angus, 1970)
Data on “Lack of Spontaneity and Flow of Conversation” (N6), “Poor Rapport” (N3) & “Conceptual Disorganisation” (P2)

Hi I’m … We’re going to be spending the next 30 to 40 minutes talking about you. Maybe you can start by telling me something about yourself and your background?

(Instruction to interviewer: Allow a few minutes for a non-directive phase, serving to establish rapport in the context of an overview before proceeding to the specific questions)
QLS09 Are you working? Doing any courses? Looking for work? ………………………………………………………………
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QLS09 How much time does that take per week? ………………………………………………………………………………………………………
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SN-AA02/QLS10 How are things going at work/school? ………………………………………………………………………………………………………
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QLS12 Do you like your work / school? ………………………………………………………………………………………………………
QLS12 Do you find it rewarding or fulfilling? Does it make you feel good? ……………………………………………………………
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SN-ANH01/SDS1D What are your interests? SDS1B What things do you really enjoy doing? ………………………………………
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SDS1D Where do you find out about that? What do you do to find out more about this? …………………………………………
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T1-Loose Assoc. T2-Tangential T3-Salad T4-Illogical T5-Circumstant. T6-Pressured T7-Distractible T8-Clanging BL6-Inapp. Aff. AL1-Speech Pov. AL2-Content Pov. AL3-Blocking AL4-Inc. Latency G13-Indecision
**1. Background & Demographic Information**

<table>
<thead>
<tr>
<th>Name</th>
<th>Sex</th>
<th>Address</th>
<th>Phone</th>
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<thead>
<tr>
<th>Date of Birth</th>
<th>Age</th>
<th>Season of Birth</th>
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<thead>
<tr>
<th>Country of Birth</th>
<th>Year of Arrival</th>
<th>Handedness</th>
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<thead>
<tr>
<th>Marital Status</th>
<th>1 Never Married; 2 De Facto; 3 Divorced; 4 Unknown; 5 Married; 6 Widowed; 7 Separated</th>
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<thead>
<tr>
<th>Accommodation Type</th>
<th>1 Private Flat/House; 2 Boarding House; 3 Residential Support Service; 4 Nursing Home; 5 Caravan; 6 Homeless; 7 Other; 8 Unknown</th>
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<tr>
<th>Living With</th>
<th>1 Alone; 2 Partner; 3 Parent; 4 Siblings; 5 Relatives; 6 Friends; 7 Child; 8 Unknown</th>
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<tr>
<th>Employment Status</th>
<th>1 Full Time; 2 Part Time; 3 Unemployed; 4 Home Duties; 5 Student; 6 Retired; 7 Pensioner; 8 Other</th>
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<tr>
<th>Education</th>
<th>1 Never Attended; 2 Year 1-6; 3 Year 7-10; 4 Year 11-12; 5 Tertiary Commenced; 6 Tertiary Completed; 7 Unknown</th>
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<tr>
<th>Highest Year Obtained</th>
<th>Qualifications</th>
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<table>
<thead>
<tr>
<th>Years Smoked Tobacco</th>
<th>Date Last Smoked</th>
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<table>
<thead>
<tr>
<th>Quantity Smoked Per Day</th>
<th>Strength</th>
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<thead>
<tr>
<th>Years Consumed Alcohol</th>
<th>Date Last Consumed</th>
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<thead>
<tr>
<th>Estimated Amount Per Week (type / quantity by glass)</th>
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<table>
<thead>
<tr>
<th>Years Smoked Cannabis</th>
<th>Date Last Smoked</th>
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<table>
<thead>
<tr>
<th>Estimated Amount Smoked Weekly (cost, grams, or cones)</th>
<th></th>
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<tr>
<th>Any other Significant Substance Use History (what, how often, time since last use)</th>
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</table>
Current Diagnosis

Other Diagnoses

Past Diagnoses

When first diagnosed with schizophrenia

Age of Onset
Years of Illness

Prodrome length (time unwell before referral)

Number of hospitalizations

Total Length of Hospitalization

Family Psychiatric History

Significant Medical History

Current Status: inpatient outpatient

Casemanager by Worker

Current Medications (specify name, dosage in mg/day, duration)

Past Medications (specify name, dosage in mg/day, duration)
Course of Illness (participant’s words; can ask to draw a plot if useful - use scale below to code)

1. Single psychotic episode, followed by complete remission
2. Single psychotic episode, followed by incomplete remission
3. Single psychotic episode followed by one or more psychotic episodes, with complete remissions between all or most of the episodes
4. Single psychotic episode followed by one or more non-psychotic episodes, with incomplete remissions between all or most of the episodes
5. Two or more psychotic episodes, with complete remissions between all or most of the episodes
6. Two or more psychotic episodes, with incomplete remissions between all or most of the episodes
7. Continuous psychotic illness (no remission); psychotic symptoms present most of the time
8. Continuous non-psychotic illness (no remission); psychotic symptoms may be present for some time, but non-psychotic symptoms predominate throughout
9. Information inadequate for rating the pattern of course
Onset of illness: (What sort of things were happening the first time you became unwell? How long did you experience these things before you went into hospital?; Use following table to code) .................................................................

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>A florid psychotic state developing within days (up to a week); mild (‘suggestive’, non-psychotic) prodromal signs or symptoms may have been absent (sudden onset) or present (precipitous onset).</th>
</tr>
</thead>
<tbody>
<tr>
<td>2</td>
<td>Subacute</td>
<td>Symptoms appearing and developing into a clear psychotic state over a period of up to one month</td>
</tr>
<tr>
<td>3</td>
<td>Gradual</td>
<td>Slow, incremental development of psychotic symptoms over a period exceeding one month; prodromal signs or symptoms (if any) cannot be clearly distinguished from overt psychotic symptoms as regards their timing because of gradual transition from one to the other</td>
</tr>
<tr>
<td>4</td>
<td>Insidious</td>
<td>No clear demarcation can be made between premorbid personality and mental illness, and onset as such cannot be rated; included are also cases in which no overt psychotic symptoms were present at the time of examination but the investigator had a strong suspicion of an underlying psychotic illness</td>
</tr>
</tbody>
</table>

Onset of the disorder is defined as the beginning of the first psychotic episode, manifested in the emergence of the following signs and symptoms:

A. At least one overt psychotic symptom or sign:
   a. Hallucinations or pseudohallucinations (in any modality)
   b. Delusions
   c. Thought and speech disorder (incoherence, irrelevance, blocking, neologisms, incomprehensibility of speech)
   d. Qualitative psychomotor disorder (negativism, mutism, or stupor; catatonic excitement, constrained attitudes and postures)
   e. Bizarre or grossly inappropriate behaviour; or:

B. The simultaneous presence of two or more ‘suggestive’ signs or symptoms
   a. Marked reduction of interests, initiative, and drive leading to a deterioration in performance
   b. Marked social withdrawal
   c. Severe excitement, purposeless destructiveness or aggression (frequent episodes or continuous)
   d. Persistent, pervasive fear or anxiety
   e. Gross self-neglect

*Any of the latter would be regarded as a prodromal phenomenon, if it appeared in isolation prior to the outbreak of overt psychotic symptoms.*
### Interim Rating of SANS/SAPS Items

<table>
<thead>
<tr>
<th>Item Code</th>
<th>Description</th>
<th>Rating</th>
<th>None</th>
<th>Questionable</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
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<tbody>
<tr>
<td>SP-B02</td>
<td>Bizarre social &amp; sexual behaviour (inappropriate to norms, e.g. sexual; talk to self)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>SN-BL01</td>
<td>Unchanging facial expression (wooden, no change w/ emotional content of speech)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>SN-BL02</td>
<td>Decreased spontaneous movements (e.g. shifting position, moving legs/hands)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>SN-BL03</td>
<td>Paucity of expressive gestures (no use of body in expression; hand gestures/leaning in/out)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>SN-BL04</td>
<td>Poor eye contact (avoids looking @ others; stares into space; doesn’t use eyes for expression)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
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<tr>
<td>SN-BL05</td>
<td>Affective (non)responsivity (failing to laugh/smile appropriately)</td>
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<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<td>SN-BL07</td>
<td>Lack of vocal inflections (monotone; no emphasis, etc.)</td>
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<tr>
<td>SN-AT01</td>
<td>Social Inattentiveness (looks away while talking, uninvolved, spacey, ends abruptly)</td>
<td></td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<tr>
<th>Item Code</th>
<th>Description</th>
<th>Rating</th>
<th>Absent</th>
<th>Minimal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
<th>Extreme</th>
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<tr>
<td>P4</td>
<td>Excitement (hyperactivity – accelerated motor behaviour, hypervigilance, lability)</td>
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<td>P7</td>
<td>Hostility (expressions of anger – sarcasm, passive-aggressive, verbal/physical abuse)</td>
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<td>7</td>
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<tr>
<td>N1</td>
<td>Blunted Affect (Diminished emotional responsiveness:no facial expression/gestures)</td>
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<td>1</td>
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<tr>
<td>N2</td>
<td>Emotional Withdrawal (lack interest/involvement/emotional commitment to life’s events)</td>
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<tr>
<td>N3</td>
<td>Poor rapport (lack interpersonal empathy/openness/closeness &amp;reduced conversation)</td>
<td></td>
<td>1</td>
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<td>4</td>
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<td>7</td>
</tr>
<tr>
<td>N6</td>
<td>Lack of Spontaneity &amp; Flow of Conversation (restricted /halting conversation)</td>
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<td>N7</td>
<td>Stereotyped Thinking (inflexible thinking: rigid/repetitious/barren thought content)</td>
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<td>G4</td>
<td>Tension (overt physical manifestations – shaking, stiffness, sweating, restlessness)</td>
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<td>G7</td>
<td>Motor Retardation (reduced motor activity – slow movts./speech, poor body tone)</td>
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<td>G8</td>
<td>Uncooperativeness (refusal to comply – distrust,stubbornness, negativism, hostility)</td>
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<td>G11</td>
<td>Poor Attention (distraction from internal/external stimuli or probs. shifting focus)</td>
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<td>G15</td>
<td>Preoccupation (with internally generated thoughts/feelings to detriment of external)</td>
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<td>E23</td>
<td>Motor Hyperactivity (increased energy level – frequent movement/rapid speech)</td>
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Data on “Anxiety” (G2)

Have you been feeling worried or nervous in the past week?  .................................................................
   If NO: Would you say that you’re usually calm & relaxed? .................................................................
   If YES: What’s been making you feel nervous (worried, uncalm, unrelaxed)? .................................

Just how nervous (worried) have you been feeling? ..............................................................................
Have you been shaking at times, or has your heart been racing? ............................................................
Do you get into a state of panic? ............................................................................................................
Has this affected your sleep, eating, or participation in activities? ............................................................

Data on “Delusions (General)” (P1) and “Unusual Thought Content” (G9)

E22 Has anything been bothering you lately? .........................................................................................

E22 Can you tell me something about your thoughts on life and its purpose? ........................................

Do you follow a particular philosophy? ....................................................................................................

(Some people tell me that believe in the devil; what do you think?) .............................................................

SP-D09 Can you read other people’s minds? ............................................................................................
   IF YES: How does that work? ..................................................................................................................
   How often do you get this feeling? ............................................................................................................

SP-D09 Can others read your mind? ...........................................................................................................
   IF YES: How can they do that? ..................................................................................................................
   Is there any reason that someone would want to read your mind? ..........................................................

T1-Loose Assoc.  T2-Tangential  T3-Salad  T4-Illogical  T5-Circumstant.  T6-Pressed  T7-Distractible
T8-Clanging  BL6-Inapp. Aff.  AL1-Speech Pov.  AL2-Content Pov.  AL3-Blocking  AL4-Inc. Latency  G13-Indecision
SP-D10 Have you heard your own thoughts out loud, as if they were a voice outside your head?  

SP-D10 Have you ever felt your thoughts were broadcast so that other people could hear them?  

   IF YES: How often does this happen?  

SP-D08 Who controls your thoughts?  

   IF NOT ME: SP-D11 Have you felt that thoughts were being put into your head by some outside force?  

   IF YES: How often does this happen?  

   IF NOT ME: SP-D12 Have you felt that your thoughts were being taken away by some outside force?  

   IF YES: How often does this happen?  

Data on “Suspiciousness/Persecution” (P6), “Passive/Apathetic Social Withdrawal” (N4), “Active Social Avoidance” (G16), “Poor Impulse Control” (G14), “Hostility” (P7) & “Emotional Withdrawal” (N2)  

SN-AA03/ANH01 How do you spend your time these days?  

QLS17/SDS1E Have you spent much time just sitting around, in bed, or watching TV?  

   IF YES: were you interested in the programs, or were you just passing the time?  

QLS16 Do you get bored much?  

QLS14/SDS1E Have there been things that you’ve wanted to do recently, but didn’t because you somehow didn’t get around to them?  

   IF YES: Why was that?  

SDS2/3 Does this change when you’re feeling well?  

T1-Loose Assoc.  T2-Tangential  T3-Salad  T4-Illogical  T5-Circumstant  T6-Pressed  T7-Distractible  
T8-Clanging  BL6-Inapp. Aff.  AL1-Speech Pov.  AL2-Content Pov.  AL3-Blocking  AL4-Inc. Latency  G13-Indecision  

760
Some people like to be with others and some prefer to be alone – how would you describe yourself?  

IF WITHDRAWN: Can you tell me why you prefer to be alone?

LIVES: WITH OTHERS WITH FAMILY ALONE (GO TO QLS02)

QLS01 Are you especially close with any of the people that you live with, or your immediate family?

Can you discuss personal matters with them?

QLS01/06 When you’re at home, do you spend much time with the others in your house, or do you generally do things alone?

SN-ANH03/04/QLS02 Do you have friends that you are especially close with other than the people you live with (or family)?

Can you discuss personal matters with them?

How often have you spoken with them recently? (in person or on phone)

QLS03 If some important or exciting thing happened to you, who would you contact?

SN-ANH03/04/QLS03 Other than close personal friends, are there many people that you know that you enjoy doing things with?

IF NO: Just a few?

IF NO: Any? .. Why?

IF YES: Why Just a few friends?

IF YES: How often do you get together?

What sort of things do you do when you get together?

SN-AA03/ANH01 ONLY IF NO TO PREVIOUS - Do you join in activities with others?

IF NO: QLS07 Can you tell me why not? (Are you uncomfortable with people, or do you dislike them?)

IF YES: Tell me about it
Do you usually ask people to do (social-type) things with you, or do you usually wait for others to ask you?

Have you turned down offers to do things with other people?

IF HAVEN'T BEEN ASKED: Would you if you were asked?

IF YES TO EITHER: Have you done (would you do) this even if you have nothing to do?

Why?

Have you been able to enjoy yourself lately?

How often do you do things for enjoyment that involve other people?

What sort of things do you do?

Do other people seem to get more enjoyment out of things than you do?

(OONLY IF EXPERIENCING CLEAR WITHDRAWAL/ASOCIAL/ANHEDONIC SYMPTOMS)

So, it sounds like you recently – do you feel this way most of the time? Or do things change when you are feeling more well?

Do you have a (girl/boy) friend?

IF YES: How’s the relationship going? Are you happy? Are you very close?

IF NO: Are you on the lookout? What sort of things are you doing?
Do you feel that you can trust most people? .................................................................

IF NO: Why not? ........................................................................................................

SP-D02 Are there some people in particular that you don’t trust? ...................................

IF YES: Can you tell me who they are? .................................................................

Why don’t you trust people (or name specific person)?........................................

........................................................................................................................................

IF “don’t know” or “DON’T WANT TO SAY”: Do you have a good reason not to trust ...?

........................................................................................................................................

Is there something that ... Did to you? .................................................................

........................................................................................................................................

Perhaps might do to you now? ...............................................................................

IF YES: Can you explain to me? ............................................................................

........................................................................................................................................

SP-D01/B03 Do you get along well with others? .........................................................

IF NO: What’s the problem? ................................................................................

SP-B03 Do you have a quick temper? ........................................................................

SP-B03/D-01 Do you get into fights? .................................................................

IF YES: How do these fights start? .................................................................

Tell me about these fights......................................................................................

How often does this happen? ...............................................................................

SP-D06/B03 Do you sometimes lose control of yourself? ...........................................

........................................................................................................................................

SP-D01 Do you like most people? ..............................................................................

IF NO: Why not? ......................................................................................................

........................................................................................................................................

QLS20 Are you affected very much by how other people feel?............................

........................................................................................................................................

........................................................................................................................................

SP-D01 Are there perhaps some people who don’t like you? ..............................

IF YES: For what reason? ......................................................................................

........................................................................................................................................
Do others talk about you behind your back? .................................................................

IF YES: What do they say about you? .............................................................................
........................................................................................................................................

Why?................................................................................................................................
........................................................................................................................................

How often do you feel this happens?..............................................................................
........................................................................................................................................

Have you walked into a room and thought people were talking or laughing about you? .
........................................................................................................................................

How often do you feel this happens?..............................................................................
........................................................................................................................................

Does anyone ever spy on you or plot against you? ......................................................
........................................................................................................................................

Do you sometimes feel in danger? ..................................................................................

IF YES: Would you say that your life is in danger? .......................................................  

Is someone thinking of harming you or perhaps even thinking of killing you? ...........
........................................................................................................................................

Have you gone to the police for help? ...........................................................................
........................................................................................................................................

Do you sometimes take matters into your own hands or take action on those who might harm you?
........................................................................................................................................

IF YES: What have you done? .........................................................................................
........................................................................................................................................

IF PARTICIPANT IS IN A RELATIONSHIP: Have you ever worried that your partner might be

unfaithful to you? ..............................................................................................................

IF YES: What evidence do you have for this happening? ..............................................
........................................................................................................................................

........................................................................................................................................
........................................................................................................................................
Data on “Hallucinatory Behaviour” (P3) and associated delusions

Do you, once in a while, have strange or unusual experiences? 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………………………………………………………
Do you sometimes receive personal communications from the radio or TV? ...........................................
 IF YES: How often? .................................................................................................................................
 IF NO: From God or the Devil? ................................................................................................................
 Can you tell me about these communications? ....................................................................................... 766

Do ordinary things sometimes look strange or distorted to you? ..............................................................

Do you sometimes have “visions” or see things that other’s can’t see? ....................................................
 IF YES: For example? ...............................................................................................................................
................................................................................................................................................................
 Do these visions seem very real or life-like? .................................................................................................
 How often do you have these experiences? .................................................................................................
 Does this occur mainly when you are falling asleep or waking up?...........................................................

Do you sometimes smell things that are unusual or that other’s don’t smell? ........................................
 IF YES: Can you tell me about these? ........................................................................................................
................................................................................................................................................................
 How often does this occur?........................................................................................................................

Do you get any strange or unusual sensations from inside your body? ......................................................
 IF YES: Tell me about this. ..........................................................................................................................
................................................................................................................................................................
 How often does this occur?........................................................................................................................

---

T1-Loose Assoc.  T2-Tangential  T3-Salad  T4-Ilogical  T5-Circumstant.  T6-Pressed  T7-Distractible
T8-Clanging  BL6-Inapp. Aff.  AL1-Speech Pov.  AL2-Content Pov.  AL3-Blocking  AL4-Inc. Latency  G13-Indecision
### Interim Ratings of SAPS/PANSS Items (II)

<table>
<thead>
<tr>
<th>Item</th>
<th>None</th>
<th>Questionable</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
</tr>
</thead>
<tbody>
<tr>
<td>SP-B02 Bizarre social &amp; sexual behaviour (inappropriate to norms, e.g. sexual; talk to self)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>SN-BL01 Unchanging facial expression (wooden, no change w/ emotional content of speech)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>SN-BL02 Decreased spontaneous movements (e.g. shifting position, moving legs/hands)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>SN-BL03 Paucity of expressive gestures (no use of body in expression; hand gestures/leaning in/out)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<tr>
<td>SN-BL05 Poor eye contact (avoids looking @ others; stares into space; doesn’t use eyes for expression)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>SN-AT01 Social Inattentiveness (looks away while talking, uninvolved, spacey, ends abruptly)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<table>
<thead>
<tr>
<th>Item</th>
<th>Absent</th>
<th>Minimal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Mod/Sev</th>
<th>Severe</th>
<th>Extreme</th>
</tr>
</thead>
<tbody>
<tr>
<td>P4. Excitement (hyperactivity – accelerated motor behaviour, hypervigilance, lability)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>P7. Hostility (expressions of anger – sarcasm, passive-aggressive, verbal/physical abuse)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<td>7</td>
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<tr>
<td>N1. Blunted Affect (Diminished emotional responsiveness/no facial expression/gestures)</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>N2. Emotional Withdrawal (lack interest/involvement/emotional commitment to life’s events)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<td>7</td>
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<tr>
<td>N3. Poor rapport (lack interpersonal empathy/openness/closeness &amp; reduced conversation)</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>N6. Lack of Spontaneity &amp; Flow of Conversation (restricted/halting conversation)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>N7. Stereotyped Thinking (inflexible thinking; rigid/repetitious/barren thought content)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>G4. Tension (overt physical manifestations – shaking, stiffness, sweating, restlessness)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
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<td>7</td>
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<tr>
<td>G7. Motor Retardation (reduced motor activity – slow movts./speech, poor body tone)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>G8. Uncooperativeness (refusal to comply – distrust, stubbornness, negativism, hostility)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>G11. Poor Attention (distraction from internal/external stimuli or probs. shifting focus)</td>
<td>1</td>
<td>2</td>
<td>3</td>
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<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>G15. Preoccupation (with internally generated thoughts/feelings to detriment of external)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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<tr>
<td>E23. Motor Hyperactivity (increased energy level – frequent movement/rapid speech)</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
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</table>

T1-Loose Assoc.  T2-Tangential  T3-Salad  T4-Illogical  T5-Circumstant.  T6-Pressured  T7-Distractible
T8-Clanging  BL6-Inapp. Aff.  AL1-Speech Pov.  AL2-Content Pov.  AL3-Blocking  AL4-Inc. Latency  G13-Indecision

![Image of table with ratings of SAPS/PANSS items]

**Notes:**
- Interim Ratings of SAPS/PANSS Items (II) table includes items for Bizarre social & sexual behaviour, Unchanging facial expression, Decreased spontaneous movements, Paucity of expressive gestures, Poor eye contact, Affective (non)responsivity, Lack of vocal inflections, Social Inattentiveness, Excitement, Hostility, Blunted Affect, Emotional Withdrawal, Poor rapport, Lack of Spontaneity & Flow of Conversation, Stereotyped Thinking, Tension, Motor Retardation, Uncooperativeness, Poor Attention, Preoccupation, Motor Hyperactivity.

**Scoring:**
- Interim Ratings use a scale from None to Severe, with additional categories for Absent, Minimal, Mild, Moderate, Marked, Severe, Extreme.
Data on “Somatic Concern” (G1)

How have you been feeling in terms of your health? .................................................................

If OTHER THAN GOOD: What has been troubling you? ..........................................................

If GOOD: Do you consider yourself to be in top health? .........................................................

If NO: What has been troubling you? ........................................................................................

SP-D06 Do you have any medical illness or disease? .................................................................

..................................................................................................................................................

SP-D06 Has any part of your body been troubling you? .............................................................

..................................................................................................................................................

If NO: How is your head? Your Heart? Stomach? The rest of your body? ..............................

..................................................................................................................................................

If YES: Could you explain? .........................................................................................................

..................................................................................................................................................

SP-D04/D06 Has your head or body changed in shape or size? ..................................................

If YES: Please explain. (Query frequency) ..................................................................................

..................................................................................................................................................

What is causing these changes? .................................................................................................

..................................................................................................................................................

SP-D04 Are there any things that you do over and over? ..........................................................

If YES: Please tell me more (query frequency) ..........................................................................

..................................................................................................................................................

T1-Loose Assoc.  T2-Tangential  T3-Salad  T4-Ilogical  T5-Circumstant.  T6-Pressured  T7-Distractible
T8-Clanging  BL6-Inapp. Aff.  AL1-Speech Pov.  AL2-Content Pov.  AL3-Blocking  AL4-Inc. Latency  G13-Indecision
How has your mood been in the past week: mostly good or mostly bad? ...........................................

IF MOSTLY GOOD: Have there been times in the past week that you were feeling sad or unhappy? .......................................................... IF YES, NEXT QUESTION:

IF MOSTLY BAD: Is there something in particular that is making you sad? ...........................................

How often do you feel sad? ..........................................................

Just how sad have you been feeling? ..................................................

Have you been crying lately? ..........................................................

Has your mood in any way affected your sleep? ..................................................

Can you tell me about how well you are sleeping? ..................................................

Has it affected you appetite? ..........................................................

Do you participate less in activities on account of your mood? ..................................................

Have you had any thoughts of harming yourself? ..................................................

IF YES: Any thoughts about ending your life? ..................................................

IF YES: Have you attempted suicide? ..................................................

Note: If concerned, consider the following:

<table>
<thead>
<tr>
<th>Variable</th>
<th>Risk Factors</th>
<th>Present?</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographics</td>
<td>Male, &gt;45, unemployed</td>
<td></td>
</tr>
<tr>
<td>Relationships</td>
<td>Isolated, conflictual, strained</td>
<td></td>
</tr>
<tr>
<td>Health</td>
<td>High substance intake, somatic focus, hopelessness</td>
<td></td>
</tr>
<tr>
<td>Suicidal Ideation</td>
<td>Frequent, intense, prolonged; Unambiguous wish to die; High self-blame</td>
<td></td>
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<tr>
<td>Suicide Attempt</td>
<td>Multiple attempts</td>
<td></td>
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<tr>
<td>Suicide Plan</td>
<td>Clear plan, method available</td>
<td></td>
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</tbody>
</table>

Actions

- What do you do when you feel like this?
- Is there somebody you can contact / talk to that is helpful if you are feeling like this?
- Have you talked to your case manager about this?
- Can you/we talk to your case manager about this?

(I’m concerned about your safety and I have a duty of care for you because of your involvement in this research. I am legally obliged to contact your case manager to discuss this matter with them so that we can help you and make sure that your are safe).
Data on “Guilt Feelings” (G3) and “Grandiosity” (P5)

If you were to compare yourself to the average person, how would you come out: a little better, maybe a little worse, or about the same? ……………………………………………………………………………………………………………………………

IF WORSE: Worse in what ways? ……………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………………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Are you a religious person? .................................................................
  IF YES: What was your religious training as a child? .........................
  Have you had any unusual religious experiences? ..............................
  Are you close to god? .................................................................
  IF YES TO EITHER: Had God assigned you some special role or purpose? .................................................................
  Can you be one of God’s messengers or angels? ..............................
    IF YES: What special powers do you have as God’s messenger (angel)? ........................................................................................................

Do you perhaps consider yourself to be god? .....................................

Do you have some special mission in life? .........................................
  IF YES: What is your mission? .......................................................
  Who assigned you to that mission? ...............................................
Data on “Disorientation” (G10)

Can you tell me today’s date? .................................................................
What is the name of the place that you are in now? ................................
What is your address? ...........................................................................
If someone had to reach you by phone, what number would that person call? .........................................................
What is the name of your casemanager (doctor, psychiatrist)? .......................
Do you know who is now the prime minister of Australia? ...................................
Who is the premier of Tasmania? .............................................................
Who is the Mayor of this city? ..................................................................

Have you been reading newspapers or listening to the TV or radio recently? ..................
IF YES: Have there been any things happening that you were really interested in? ...............
..................................................................................................................
..................................................................................................................
Did you do anything to find out more about them? ...............................................
..................................................................................................................
..................................................................................................................

QLS18: Commonplace objects

Are you carrying any of the following items?
1. a wallet or purse?
2. keys?
3. drivers license?
4. watch?
5. a bank card?
6. a health care or medicare card?

Do you have at your house any of the following?
1. map of the area?
2. your own alarm clock?
3. a comb or hairbrush?
4. an overnight bag?
5. a library card?
6. postage stamps?

QLS19: Commonplace activities

In the past two weeks, which of the following have you done?
1. read a newspaper?
2. paid a bill?
3. wrote a letter?
4. gone to a movie?
5. driven a car or caught a bus alone?
6. shopped for food?
7. shopped for things other than food?
8. eaten out?
9. taken something out of the library?
10. participated in some sort of public event?
11. gone to watch some sporting event?
12. visited a park or something like that?
Data on “Difficulty in Abstract Thinking” (N5)

I’m going to now say a pair of words, and I’d like you to tell me in what important way they’re alike. Let’s start, for example, with the words ‘apple’ and ‘banana’ How are they alike – what do they have in common?

IF ‘THEY’RE BOTH FRUIT’: good, now what about ……? (select 3 other items at varying levels of difficulty)

IF AN ANSWER IS GIVEN THAT IS CONCRETE, TANGENTAL, OR IDIOSYNCRATIC, E. G. ‘THEY BOTH HAVE SKINS’, ‘YOU CAN EAT THEM’ OR ‘MONKEYS LIKE THEM’: OK, but they’re both fruit. Now how about … and …: how are these alike?

<p>| | |</p>
<table>
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<tbody>
<tr>
<td>1. Ball and an Orange?</td>
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<tr>
<td>2. Apple and a banana?</td>
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<tr>
<td>3. Pencil and pen?</td>
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<td>4. Nickel and dime?</td>
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<td>5. Table and Chair?</td>
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<td>6. Tiger and elephant?</td>
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<td>7. Hat and shirt?</td>
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<td>8. Bus and Train?</td>
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<td>9. Arm and leg?</td>
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<td>10. Rose and Tulip?</td>
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<td>11. Uncle and cousin?</td>
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<td>12. The sun and the moon?</td>
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<td>13. Painting and poem?</td>
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<tr>
<td>14. Hilltop and Valley?</td>
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<tr>
<td>15. Air and Water?</td>
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<td>16. Peace and prosperity?</td>
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</table>
You’ve probably heard the expression “carrying a chip on the shoulder” - what does this really mean?
There’s a very old saying “Don’t judge a book by its cover” – what is the deeper meaning of this proverb?
(select 2 other proverbs from the list at varying levels of difficulty)

<table>
<thead>
<tr>
<th>What does the saying mean:</th>
<th></th>
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<tbody>
<tr>
<td>1. Plain as the nose on your face</td>
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<tr>
<td>2. Carrying a chip on your shoulder</td>
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<tr>
<td>3. Two heads are better than one</td>
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<td>4. Too many cooks spoil the broth</td>
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<td>5. Don’t judge a book by its cover</td>
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<td>6. One man’s food is another man’s poison</td>
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<td>7. All that glitters is not gold</td>
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<td>8. Don’t cross the bridge until you come to it</td>
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<td>9. What’s good for the goose is good for the gander</td>
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<td>10. The grass always looks greener on the other side</td>
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<td>11. Don’t keep all your eggs in one basket</td>
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<td>12. One swallow does not make a summer</td>
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<td>13. A stitch in time saves nine</td>
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<td>14. A rolling stone gathers no moss</td>
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<td>15. The acorn never falls far from the tree</td>
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<tr>
<td>16. People who live in glass houses should not throw stones at others</td>
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</tbody>
</table>
Data on Attention (SANS-AT01/02; PANSS G11)

How would you say your attention is?........................................................................................................
Do you have any problems, say, watching TV, following a movie, or reading a book?...........................
.................................................................................................................................................................. 

FOR THE FOLLOWING, GIVE A SCORE OUT OF FIVE (ONE FOR EACH CORRECT PART)

Spell the word ‘world’ backwards for me: (D-L-R-O-W) .................. Score = ......................

Subtract back from 100 by seven (if education > grade 10; by 3 if > grade 6). Stop after five subtractions.
(100, 93, 86, 79, 72, 65)....................................................................................................................... Score = ....................

☐ Made error, but self-corrected ☐ Completed in halting manner
Data on “Lack of Judgement and Insight” (G12)

How long have you been in the hospital (patient of … clinic etc)? ……………………………………………

Why did you come to the hospital (clinic etc)?…………………………………………………………………
…………………………………………………………………………………………………………………

Did you need to be in a hospital (clinic etc.)?……………………………………………………………………

IF NO: Did you have a problem that needed treatment? ……………………………………………………………

IF YES: Would you say that you had a psychiatric or mental problem? …………………………………………

IF NO: Why are you taking medications / still in the hospital (clinic etc)?………………………………………
………………………………………………………………………………………………………………………………

IF YES: Why? …would you say that you had a psychiatric or mental problem? ……………………………….……

IF YES: Can you tell me about it and what it consists of? ……………………………………………………………
…………………………………………………………………………………………………………………………

In your own opinion, do you need to be taking medications?……………………………………………………
…………………………………………………………………………………………………………………………

Why? … Do the medications help you in any way? …………………………………………………………………
…………………………………………………………………………………………………………………………

SDS1E/QLS13 Do you have any plans for your life over the next year or so – personal as well as work-related
ones?……………………………………………………………………………………………………………………
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SDS1E Have you been doing things lately to work toward those goals? What sort of things?………………….
…………………………………………………………………………………………………………………………
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Well, that’s about all I have to ask you about these forms. Are there any questions that you might like to ask of
me at this stage?
**Simpson-Angus Scale (Short Form)**

a. **Tremor**
*Participant is observed during the interview.*
0. Absent
1. Mild finger tremor, obvious to sight and touch
2. Moderate tremor of hand and arm occurring spasmodically
3. Marked tremor on one or more limbs
4. Whole body tremor

b. **Salivation**
*Participant is observed while talking and then asked to open his mouth and elevate his tongue.*
0. Absent
1. Slight salivation to the extent that pooling takes place if the mouth is open and tongue raised
2. Excess salivation is present and might occasionally result in difficulty in speaking
3. Speaking with difficulty because of marked salivation
4. Frank drooling

c. **Rigidity**
*This represents increased muscle tone of a uniform and general nature. It is observed on the basis of a uniform, steady resistance to passive movements of the limbs. Special importance is attached to the muscles around the elbow joints Procedure: The elbow joints are separately bent at right angles and passively extended and flexed, with the participant’s biceps observed and simultaneously palpated. Rate resistance to this procedure.*
0. Absent
1. Doubtful or very slight rigidity
2. Moderate rigidity in neck, shoulder and extremities. It must be possible to observe the rigidity on the basis of resistance to passive movements of elbow joints.
3. Marked rigidity assessed on the basis of resistance to passive movements of, for instance, elbow joints
4. Very marked rigidity

Note **Presence of cogwheel rigidity:** present / absent

d. **Gait**
*The participant is examined as he walks into the examining room, his gait, the swing of his arms, his general posture, all form the basis for an overall score for this item.*
0. Absent
1. Diminution in swing while the participant is walking
2. Moderate diminution in swing with obvious rigidity in the arm
3. Stiff gait with arms held rigidly before the abdomen
4. Stooped shuffling gait with propulsion and retropulsion

e. **Dystonic Reaction**
*Dystonia in form of tonic muscular contractions localized to one or several muscle groups, particularly in the mouth, tongue or neck. The rating is to be made on the basis of the 72 hours preceeding the examination.*
0. Absent
1. Doubtful or very slight dystonia
2. Moderate and short spasms, for instance, in the musculature of the jaws or the neck
3. More markedly pronounced contractions of a longer duration or of a wider localization
4. Very marked forms, for instance, oculogyric crises or opisthotonia
f. Akathisia

This represents the subjective feeling of muscle unrest, particularly in the lower extremities, so that it may be difficult for the participant to remain seated. The assessment of this item is based on clinical signs observed during the interview, as well as on the participant’s report.

0. Absent
1. Doubtful or very slight akathisia
2. Moderate akathisia, however, the participant can keep still without effort
3. Marked akathisia; the participant can, however, with effort, remain sitting during the interview.
4. The participant rises several times during the interview because of akathisia.

g. Arm Dropping*

The participant and examiner both raise their arms to shoulder height and let them fall to their sides. In a normal participants stout slap is heard as the arms hit the sides. In a participant with extreme Parkinson’s syndrome, the arms fall very slowly:

0. normal, free fall with loud slap and rebound
1. fall slowed slightly with less audible contact and little rebound
2. fall slowed, no rebound
3. marked slowing, no slap at all
4. arms fall as though against resistance; as though through glue

References:


SDS3 Do your medications make you feel sleepy/tired/bog you down? .................................................................
........................................................................................................
........................................................................................................
SDS3 (Do you have more energy when you aren’t on the medications?) .........................................................
........................................................................................................
........................................................................................................
### Rating of SANS/SAPS/PANSS Items (III) – On basis of whole interview

<table>
<thead>
<tr>
<th>Item</th>
<th>None</th>
<th>Questionable</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
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<tbody>
<tr>
<td><strong>SP-B02</strong> Bizarre social &amp; sexual behaviour (inappropriate to norms, e.g. sexual; talk to self)</td>
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<td><strong>SN-BL02</strong> Unchanging facial expression (wooden, no change w/ emotional content of speech)</td>
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<td><strong>SN-BL03</strong> Decreased spontaneous movements (e.g. shifting position, moving legs/hands)</td>
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<td><strong>SN-BL04</strong> Paucity of expressive gestures (no use of body in expression; hand gestures/leaning in/out)</td>
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<td><strong>SN-BL05</strong> Poor eye contact (avoids looking @ others; stares into space; doesn't use eyes for expression)</td>
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<td><strong>SN-BL06</strong> Affective (non)responsivity (failing to laugh/smile appropriately)</td>
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<td><strong>SN-BL07</strong> Lack of vocal inflections (monotone; no emphasis, etc.)</td>
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<td><strong>SN-AT01</strong> Social Inattentiveness (looks away while talking, uninvolved, spacey, ends abruptly)</td>
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<td><strong>SP-B01</strong> Bizarre clothing and appearance (dress unusual/inappropriate to situation etc.)</td>
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<tr>
<td><strong>SN-AA01</strong> Grooming and hygiene (level of self-care)</td>
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<table>
<thead>
<tr>
<th>Item</th>
<th>Absent</th>
<th>Minimal</th>
<th>Mild</th>
<th>Moderate</th>
<th>Marked</th>
<th>Severe</th>
<th>Extreme</th>
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<tr>
<td><strong>P4. Excitement</strong> (hyperactivity – accelerated motor behaviour, hypervigilance, lability)</td>
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<td><strong>P7. Hostility</strong> (expressions of anger – sarcasm, passive-aggressive, verbal/physical abuse)</td>
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<tr>
<td><strong>N1. Blunted Affect</strong> (Diminished emotional responsiveness:no facial expression/gestures)</td>
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<td><strong>N2. Emotional Withdrawal</strong> (lack interest/involvement/emotional commitment to life’s events)</td>
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<td><strong>N3. Poor rapport</strong> (lack interpersonal empathy/openness/closeness &amp; reduced conversation)</td>
<td>1</td>
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<td><strong>N6. Lack of Spontaneity &amp; Flow of Conversation</strong> (restricted / halting conversation)</td>
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<tr>
<td><strong>N7. Stereotyped Thinking</strong> (inflexible thinking: rigid/repetitious/barren thought content)</td>
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<td><strong>G4. Tension</strong> (overt physical manifestations – shaking, stiffness, sweating, reslessness)</td>
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<td><strong>G7. Motor Retardation</strong> (reduced motor activity – slow movts./speech, poor body tone)</td>
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<tr>
<td><strong>G8. Uncooperativeness</strong> (refusal to comply – distrust, stubbornness, negativism, hostility)</td>
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<td><strong>G11. Poor Attention</strong> (distraction from internal/external stimuli or probs. shifting focus)</td>
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<td><strong>G15. Preoccupation</strong> (with internally generated thoughts/feelings to detriment of external)</td>
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<td><strong>E23. Motor Hyperactivity</strong> (increased energy level – frequent movement/rapid speech)</td>
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