The nature and impact of negative symptoms of psychosis – a discussion

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Discussion Paper

Introduction
There is now general agreement that there are three groups of symptoms associated with a diagnosis of psychotic illness:

1. **Positive symptoms** – hallucinations, delusions, ideas of reference, passivity phenomenon such as thought insertion, withdrawal, broadcast.

2. **Negative symptoms** – social withdrawal, apathy, demotivated behaviour, flattened mood and a lack of pleasure or interest.

3. **Cognitive / disorganised symptoms** - disordered thinking leading to difficulty in forming thoughts, poor speech content and flow, poor concentration, poor memory and difficulties in learning, problem-solving and abstract thinking (Morrison et al 2004).

Negative and cognitive symptoms, which some would argue are inter-related (Stolar and Grant 2007, Milev et al 2005), appear to be overlooked as a treatment priority. Historically, little attention has been paid to negative or cognitive symptomatology in psychotic illness (Stolar and Grant 2007, Birchwood and Trower 2006). It has been argued that the emphasis on positive symptomatology may lie with both the under-reporting of negative symptoms, as they are nearer to usual behaviour, and the intolerant social reaction to positive symptomatology (Stolar 2004, Rector et al 2005).

It is clear that such symptoms represent a source of poorer outcomes in quality of life issues, such as relationships, social and vocational roles (Bromet et al 2005, Fowler et al 1998) and in some cases this may contribute to relapse and co-morbidity (Birchwood 2003, Kuipers 2006).
For example, there may be an increased potential for the exacerbation and entrenchment of delusional beliefs due to a lack of disconfirmation opportunities (Freeman 2007). Dam (2006) further highlights the correlation between depression, adaptation to psychotic experience (Fowler et al 1998) and suicide. Suicide rates of up to 10% have been found in those diagnosed with schizophrenia, over a 10 year follow up period (Bromet et al 2005). Other authors cite the spiral of social decline and an eroding of the personality that is so often witnessed in individuals with a diagnosis of psychotic illness, as mainly related to apathy, social withdrawal and poor interpersonal functioning – commonly referred to as negative symptoms (Milev et al 2005). The financial implication of these medication resistant symptoms is also well documented in terms of direct and indirect costs – for example: benefits claims, poly-pharmacy, hospital admissions and family burden (Mino et al 2007). However, the origins and maintenance factors implicated in these symptoms, if this is indeed the correct terminology (Fowler et al 1995, Beck - Sander 1998), are complex and less easily described or understood.

**What are Negative Symptoms?**

Although Bleuler and Kraepelin described emotional and social difficulties in their descriptions of schizophrenia (Beck and Rector 2005), Crow (1980, cited in Morrison et al 2004) introduced the concept of a cluster of symptoms specific to the diagnosis of schizophrenia:

- **flattening of affect** – blunted, unresponsive and / or a limited range of emotion;
- **alogia** – poverty of speech, limited verbal interaction and poverty of thought (this latter issue is debatable as there may be a thought but a lack of desire or sense of purpose to express the thought (Rector et al 2005, Stolar 2004);
- **anhedonia** – an inability to experience pleasure (although it has been argued that there may be an inability to initiate pleasure-inducing activities, individuals may still be able to enjoy these as well as anyone else once the activity is initiated (Rector et al 2005));
- **avolition** – a lack of will / motivation;
- **apathy** – a lack of interest;
- **asociality** – a lack of social interaction (Morrison et al 2004).

Although the presence of these features in association with psychosis is not disputed, the aetiology is widely debated (Fowler et al 1995, Stolar &
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Grant 2007). Indeed some authors argue for a move away from a discrete diagnosis of schizophrenia, as it objectifies a heterogeneous range of experience, to one that is more sensitive to the subjective experience (Levin 2006).

Theories of Negative Symptoms
It is hypothesised that negative symptoms can be further subdivided into primary and secondary symptoms (Beck & Rector 2005).

*Primary negative symptoms* refer to the attitudes, beliefs and behaviours (or lack of) resulting from neurobiological and/or pathological processes (Morrison et al 2004).

Some studies have been offered as evidence of this theory, showing that those experiencing negative symptoms of schizophrenia have enlarged ventricles in the brain (Rector et al 2005, Littrell et al 1997). Stolar (2004) hypothesises that resulting neurobiological disturbances or other anomalies in connectivity may cause disruption in various inter-related aspects of cognitive processing, which include memory, attention, and reward systems, as shown in Fig 1.1.

**Fig 1.1 Implications of Inter-related Cognitive Processes**

Milev et al (2005) demonstrated this inter-relationship, identifying a slower pupillary response, an indicator of information processing speed, in a letter
recognition task in individuals diagnosed with psychotic illness. They found that impaired information processing speed, which is linked to memory and attention, was predictive of poorer outcomes in terms of social, vocational and global psychological functioning. They also highlighted a link between a decreased level of cognitive functioning at first episode and progressive severity of negative symptoms over a seven year period. Although this provides evidence of impairment, it is not concrete evidence that these changes are directly caused by a disease process. It does however highlight the need for early and sustained intervention.

There is some suggestion that abnormal cell pruning in the womb or during adolescence may be responsible for poor connectivity (Morrison et al 2004) and may be a vulnerability factor for psychosis in the first instance, rather than a symptom of psychotic illness (Fowler et al 1998). Furthermore enlarged brain ventricles have been found in individuals with alcohol, depression and other disorders (Littrell et al 1997), such that the evidence, whilst convincing, is inconclusive.

Rector et al (2005) take a different view and propose a link between premorbid schizotypal and schizoid personality traits in those with a stable core of negative symptoms. They propose a continuum of experience and behaviour, for example, in those individuals with pre-morbid difficulties or a lack of interest with initiating, understanding and maintaining social relationships, which may be exacerbated by the experience of psychotic illness. They argue that 85% of outpatients with schizophrenia have a diagnosis of personality disorder, mainly of the afore-mentioned variety. However, these theories fail to explain negative symptoms in those with healthy pre-morbid functioning.

Secondary negative symptoms refer to the attitudes, behaviours and beliefs associated with (or secondary to) the experience of psychosis.

The impact of the experience of psychosis can be multi-faceted, from the treatment options to the personal and societal response, and can influence the recovery process (Birchwood et al 2006). For example, anti-psychotic medication generally works by blocking selected dopamine pathways (Farine 1994), which can mean an under-stimulation of the mesocortical pathway that affects frontal lobe executive functioning (Littrell et al 1997) – a global term for planning and co-ordinating activity. Further histamine
blockage by serotonin action can induce sedation and weight gain by increasing appetite (Farine 1994), which can lead to a lack of activity and social withdrawal, as described in Fig 1.1, compounded by tiredness and poor self esteem.

These issues can be further compounded by the widely described experience of shame and stigma associated with a diagnosis of psychotic illness (Birchwood et al 2006). Social rank theory suggests that we all have a desire to present ourselves as attractive to others (Gilbert 2000). Being marked by the self or others, particularly with something as taboo as a diagnosis of schizophrenia, can lead to opting out, avoidance and symptoms of social anxiety, which are similar in presentation to both depression and negative symptomatology (Gumley et al 2004, Dam 2006, Birchwood et al 2006).

Furthermore, trauma occurring either in response or as a trigger to psychosis (Calcott et al 2004) can influence memory. Indeed structural changes in the hippocampus (the area of the brain associated with memory, learning and contextual information processing) have been found to occur in (sexual abuse and combat) trauma victims (Morrison et al 2004). Calcott et al (2004) suggest that reliving distressing experience, which is a trademark of post traumatic stress that results from a perceived threat to life or integrity, experienced through hallucinations and delusions, can influence memory and affect. As mentioned earlier, memory difficulties impede an individual’s ability to plan and initiate activity. The consequent lack of stimulation can induce a blunting of affect / numbness that is, again, characteristic in post trauma but interestingly virtually indistinguishable from negative symptomatology.

Furthermore, the experience of trauma can shatter pre-morbid core beliefs and assumptions of a sense of self efficacy and safety (Birchwood et al 2006). Trauma may also exacerbate existing beliefs and attitudes in terms of Schizotypal and Schizoid personality traits (Rector et al 2005) and the meanings attached to expressing wishes / desires, forming relationships and engaging in activities. Consequent diminished anticipation for pleasure and success can result in apparent apathy, avolition and indeed alogia (Gumley et al 2004, Beck and Rector 2002). Individuals may not see the point in engaging socially (Gumley et al 2004, Beck and Rector 2005).
Other authors have suggested that negative symptoms may actually be coping strategies for managing distressing residual positive symptoms (Birchwood 2003) or perhaps an avoidance of high expressed emotion in the family. However, what we do know is that these issues are exacerbated as the social decline resulting from negative symptomatology spirals (Milev et al 2005, Morrison et al 2004). As we have demonstrated, the causes are inter-related and complex and it would follow that treatment is also variable. Consequently it is difficult to be discrete about the cause and effect and it may be more useful to take a stress-vulnerability perspective (Zubin and Spring 1977).

Rationale and Evidence Base for CBTp for negative symptoms
Most studies of cognitive behavioural therapy for psychosis (CBTp) have tended to concentrate on interventions for the management of positive symptoms (Stolar 2004). The general theme of these studies appears to be favourable in terms of reduced distress and improved coping and subsequent impact on negative symptoms (Rector and Beck 2001, Haddock et al 2003, Sensky et al 2000, Granholm et al 2005, Wykes et al 2007) (Fig. 1.2). Furthermore the National Institute for Clinical Excellence (2003) indicates CBTp as a core treatment option for both positive and negative symptomatology.

<table>
<thead>
<tr>
<th>Study</th>
<th>Experimental Group</th>
<th>Control Group</th>
<th>Outcome measure</th>
<th>Outcome</th>
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<tr>
<td>Sensky et al 2000</td>
<td>90 clients offered 20 x 45 min sessions of CBT + RC + RC</td>
<td>20 x 45 min sessions of befriending + RC</td>
<td>CPRS, SANS, MADRS</td>
<td>at immediate follow-up all groups showed significant improvement; at 9/12 FU, significant improvement for CBT group</td>
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<td>Pinto et al 1999 (Wykes et al 2007)</td>
<td>19 clients offered 24 x 1 hr sessions of CBT + social skills training + RC</td>
<td>18 clients offered 24 x 1 hr psycho-education, crisis management, advocacy (housing etc...) + RC</td>
<td>BPRS, SAPS, SANS</td>
<td>at immediate follow-up, significant improvement; at 6/12 FU, slippages in control group but experimental group remain improved</td>
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<td>Daniels 1998 (Wykes et al 2007)</td>
<td>20 clients offered 16 x 50 min sessions twice weekly of</td>
<td>20 clients as a waiting list control – not specified</td>
<td>CGI, QOL, SANS, BPRS, GAF, PANSS</td>
<td>improved social competence, psychosocial &amp; global functioning at</td>
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<td>Group CBT + Group Process Strategies</td>
<td>Post Treatment</td>
<td>Some Deterioration in Control Group</td>
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<td>Haddock et al 2003</td>
<td>17 clients offered 29 sessions of individual CBT + motivational intervention &amp; 15 carers offered 10-16 sessions of Behavioural Family Therapy + RC</td>
<td>18 clients offered RC &amp; 18 carers offered a family support worker (advice and management)</td>
<td>GAF, PANSS, SFS, TLFB, client service receipt inventory, salary of therapist; GHQ, BDI, RCNS with carers</td>
<td>Signif improv in positive symptoms over 12 but not 18 months, though GAF remained improved; no signif cost effectiveness found</td>
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<tr>
<td>Rector et al 2002</td>
<td>24 clients offered 20 x weekly sessions of CBT + enhanced RC</td>
<td>18 clients offered 20 x weekly sessions of enhanced RC (psycho-ed, meds management, housing, OT &amp; crisis support)</td>
<td>PANSS, BDI</td>
<td>At 6 month follow up: 65% vs 62% improvement in +ve symptoms; 67% vs 31% improvement in –ve symptoms</td>
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<td>Granholm et al 2005</td>
<td>37 older clients offered 24 x 2hr group therapy, CBT &amp; social skills training + RC</td>
<td>39 clients offered RC</td>
<td>ILS, PBSA, PANSS HADRS, BCIS</td>
<td>Improvement in general functioning and skills over control group but no change in +ve / -ve symptoms</td>
</tr>
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</table>

**Abbr:** RC = routine care; CBT = cognitive behaviour therapy; OT = occupational therapy; CPRS = comprehensive psychiatric rating scale; SAPS = scale for assessment of positive symptoms; SANS = scale for assessment of negative symptoms; BPRS = brief psychiatric rating scale; GHQ = general health questionnaire; QOL = quality of life scale; CGI = clinical global improvement scale; HADRS = hospital anxiety and depression scale; GAF = global assessment of functioning scale; SFS = social functioning scale; BDI = Beck depression inventory; TLFB = time line follow back; ILS = independent living skills; PBSA = performance based skills assessment; BCIS = Beck cognitive insight scale.

Although generally positive, the studies above incorporated varied interventions under the umbrella of CBTp. Consequently it is difficult to state with certainty the elements of CBTp that were effective for negative symptomatology. However, Wykes et al (2007) and Rector and Beck (2001) have reviewed several studies of CBTp that included those targeting social functioning, social anxiety and negative symptomatology. The factors identified for success with negative symptoms are: shared goals; psycho-education techniques; compliance; and, behavioural strategies. A further
study by Massari and Hallam (2003) identified the fundamental characteristics as humanistic and collaborative.

Although there does not appear to be a specific model of CBT for negative symptoms, it is generally agreed that therapy should be formulation focused (Morrison et al 2004, Rector et al 2002) but with the following general principles in mind:
1. to reduce distress and disability;
2. to reduce emotional disturbance;
3. to increase active participation of the client in managing self, risk and social implications (Fowler et al 1995).

There is also general agreement that the behavioural features of CBT, including homework, are most beneficial for negative symptomatology (Rector and Beck 2001, Jacobson et al 2001). In keeping with this philosophy, activity scheduling, which includes mastery and pleasure ratings (Beck and Rector 2005) is used to:
- monitor current activities and beliefs;
- begin to address inactivity by scheduling pleasurable and meaningful activities (Jacobson et al 2001) and rating the extent to which these are completed and enjoyed;
- breaking tasks into smaller manageable and repeated tasks (Milev et al 2005).

**Conclusion**
In conclusion, the debates surrounding the aetiology of psychosis are endless. There is general agreement that psychotic illness is not a discrete disease process and many authors now accept theories based on several inter-related factors (Zubin and Spring 1977, Calcott et al 2004, Fowler et al 1995). From this standpoint, it seems sensible to conclude that the same is true for the causes and effects of primary and secondary negative symptoms in relation to psychotic illness. Consequently, it may be more useful to take a more holistic and person-centred approach when attempting to understand and support individuals in managing these symptoms.
At present the philosophy of cognitive behavioural intervention seems most appropriate as it encourages the client to explore their current situation and identify personally meaningful areas for change, and collaborate on the method of the change process and the pace at which
the change will occur. This would appear to be most important for regaining a sense of control and purpose for those whose illness may have externalised, in actual or perceived terms, a significant proportion of their locus of control.

References


