Cognitive approach to depression and suicidal thinking in psychosis

1. Ontogeny of post-psychotic depression†

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Background Depression in schizophrenia is a rather neglected field of study, perhaps because of its confused nosological status. Three course patterns of depression in schizophrenia, including post-psychotic depression (PPD), are proposed.

Aims We chart the ontogeny of depression and psychotic symptoms from the acute psychotic episode over a 12-month period and test the validity of the proposed course patterns.

Method One hundred and five patients with ICD–10 schizophrenia were followed up on five occasions over 12 months following the acute episode, taking measures of depression, positive symptoms, negative symptoms, neuroleptic exposure and side-effects.

Results Depression accompanied acute psychosis in 70% of cases and remitted in line with the psychosis; 36% developed PPD without a concomitant increase in psychotic symptoms.

Conclusions The results provided support for the validity of two of the three course patterns of depression in schizophrenia, including PPD. Post-psychotic depression occurs *de novo* without concomitant change in positive or negative symptoms.

Declaration of interest This research was supported by the Mental Health Foundation and Department of Health.

Depression in schizophrenia recently has enjoyed a revival in research settings, although depression-like pathology has been acknowledged in the literature since the earliest observations (Bleuler, 1950). The major shift in this position has been the observation that depression may also occur independently of the symptoms of schizophrenia and several months after recovery from an acute episode, i.e. post-psychotic depression, in up to 30% of cases (Siris, 1995); it is known to be both a precursor and a concomitant feature of hopelessness and suicidal thinking (Drake & Cotton, 1986). Several theories attempting to account for the onset of secondary depression have been postulated. The 'intrinsic' theory suggests that depression is an essential aspect of the schizophrenic process and as such should be discernible at one or more stages during the course of an acute psychotic episode (Hirsch & Jolley, 1989). 'Pharmacogenic' theories have suggested that the depression observed in psychosis may be simply a drug-induced akinetic dysphoria (Van Putten & May, 1978). Results from Birchwood *et al* (1993) and Rooke & Birchwood (1998) show that depression following acute psychosis may be a psychological response (demoralisation) to an apparently uncontrollable life event (the psychosis) and its attendant disabilities. Leff (1990) proposes three types of clinical course for secondary depression in schizophrenia: depressive symptoms at the acute stage that are ameliorated as psychotic pathology recedes and do not re-emerge after recovery; depressive symptoms that emerge at the point of remission of the acute psychosis; and depressive symptoms that emerge independently of the acute psychosis at a point several months after recovery (post-psychotic depression). In this study we investigate the ontogeny of depression from the acute episode onwards and put these models and course patterns to the test. We report the evolution of depression over a 12-month follow-up period and its link with positive and negative symptoms.

METHOD

Design

The course of psychotic and dysphoric symptoms was investigated by longitudinal assessment of symptoms during the acute stage, on recovery and at follow-up interviews 4, 8 and 12 months after recovery. Specific instruments were employed to assess the course of positive and negative symptoms, depression and suicidal ideation.

Case identification

The study commenced in 1994 and all patients were recruited from acute services in northern Birmingham (population 650 000). Inclusion required that patients fulfilled ICD–10 (World Health Organization, 1992) criteria for schizophrenia or a related disorder (F20, F22 and F23), in the absence of organic disorder or a current diagnosis of mania or bipolar disorder or mood-congruent delusions and hallucinations. All participants were aged between 18 and 65 years inclusive.

Daily contact with admission units ensured that all new admissions were screened within 3 days of their arrival by examining medical files for exclusion criteria and current symptoms/diagnosis and through discussion with ward nursing and psychiatric staff. Patients satisfying screening criteria were interviewed within the next 2 days; thus, no more than 5 days elapsed between admission and first interview. The timing of the recovery interview corresponded with each patient's discharge from the ward and employed recovery criteria described by Drury *et al* (1996). Patients were re-interviewed until recovery had occurred.

Assessments

Each interview was conducted using a battery of measures. Negative and positive symptoms were measured using the Scale for the Assessment of Negative Symptoms (SANS; Andreason, 1982) and the Scale for the Assessment of Positive Symptoms (SAPS; Andreason, 1986) respectively. Two measures were used to assess depressive pathology: the self-report Beck Depression Inventory (BDI; Beck, 1967) and the interview-based Calgary Depression Scale for Schizophrenia (CDSS; Addington *et al*,

†See pp. 522–528, this issue.

https://doi.org/10.1192/bjp.177.6.516 Published online by Cambridge University Press

1993), which also provides sub-scales for hopelessness and suicidal ideation. The CDSS was developed specifically with the aim of avoiding phenomenological overlap between depression and negative symptoms. The presence and severity of pharmacogenic side-effects were assessed using the Abnormal Involuntary Movements Scale (AIMS; Wojcik *et al*, 1980) and the chlorpromazine equivalent dosages were calculated from the September 1996 *British National Formulary* (British Medical Association & Royal Pharmaceutical Society of Great Britain, 1996). Basic demographic and clinical data were collected on a standardised form.

Patients' beliefs about their illness (Personal Beliefs about Illness Questionnaire, PBIQ; Birchwood *et al*, 1993), self-esteem (Crown Self-Esteem Scale; Crown *et al*, 1977) and insight (Insight Scale; Birchwood *et al*, 1994) were also assessed. Additional 'one-off' measures were employed during the follow-up phase to evaluate cognitions pertaining to the self, namely the Possible Selves Questionnaire (PosSQ; Marcus & Nurius, 1986) and the Depressive Experiences Questionnaire (DEQ; Blatt *et al*, 1976). The data from the PBIQ, Crown Self-Esteem Scale, Insight Scale, PosSQ and DEQ will be commented upon in our subsequent companion paper.

Each item pertaining to a symptom on either the SANS and SAPS is assessed on a six-point scale. A score of zero denotes the absence of the symptom whereas a rating of 1–5 denotes the presence of the target symptom in increasing severity. The SANS has five categories: affective blunting and flattening, alogia, avolition–apathy, anhedonia–associality and attention. The SAPS consists of four categories: hallucinations, delusions, bizarre behaviour and positive formal thought disorder. In addition to the symptom items, which comprise each SANS or SAPS category, an additional 'global rating' item for the category as a whole is included.

Reliability of SANS and SAPS ratings

Following a period of training in the instruments, mental state assessments carried out by the second author (Z.I.) were subject to a reliability check to prevent drift in accuracy of ratings across the study. A sample (n=6) from the present study was selected randomly at different points and re-interviewed by M.B. blind to the original assessment. Sufficient concordance between the two raters was established for both SANS (r=0.95, $P<0.01$) and SAPS (r=0.86, $P<0.05$) total scores.

Hypotheses

(a) Depression and positive symptoms will 'follow the same course' during recovery from the acute phase.

(b) Depressive symptoms that emerge at the post-psychotic stage will do so independently of the course of negative or positive symptoms or neuroleptic side-effects.

RESULTS

Of the 105 subjects recruited into the study, a subsample of 24 (22.8%) dropped out during follow-up: 87 (83%) completed two observations, 81 (77%) completed three or four observations and 78 (74.3%) completed all five interviews over the duration of the study. The subsample of subjects (n=27) who did not complete all assessments was found to differ significantly from the main sample in two respects: the subsample was significantly older at the first onset of psychosis and also in chronological age (see Table 1). Completion of all five follow-up assessments was not linked to use of the Mental Health Act during hospital admission (χ^2=1.0, NS).

Definition of post-psychotic depression

The ICD–10 definition of post-psychotic depression (PPD, F20.4) requires that, along with general criteria for schizophrenia during the previous 12 months, the patient must still exhibit persistent hallucinations, thought disorder or negative symptoms not due to depression or neuroleptic medication. These criteria make many of the assumptions embodied in the theories outlined in the introduction, whereas in this study we are making no such assumptions and indeed we are attempting to discern empirical support for them. Hence, our definition revolves around depressed mood *per se*.

Table 1 Mean values for complete and incomplete data subgroups at onset and recovery interviews

Variable	Onset				Recovery			
	Complete (n=78)	Incomplete (n=27)	t	P	Complete (n=78)	Incomplete (n=15)	t	P
BDI score	21.09	17.37	1.37	NS	12.99	17.07	1.48	NS
CDSS score	9.20	9.18	0.01	NS	5.87	6.00	0.11	NS
Negative symptoms	11.44	9.63	1.72	NS	7.86	8.33	0.41	NS
Positive symptoms	7.94	8.58	0.94	NS	3.37	3.33	0.44	NS
Suicidal ideation	0.77	0.59	0.81	NS	0.26	0.40	0.72	NS
Hopelessness	1.24	1.04	0.86	NS	0.87	0.80	0.36	NS
Duration of admission	–	–	–	–	72.47	62.85	0.58	NS
Age at first onset	–	–	–	–	25.29	32.07	2.96	0.004
Total admissions	–	–	–	–	5.17	6.67	0.98	NS
Age	–	–	–	–	33.52	39.85	2.33	0.022

BDI, Beck Depression Inventory; CDSS, Calgary Depression Scale for Schizophrenia.

https://doi.org/10.1192/bjp.177.6.516 Published online by Cambridge University Press

Given that we are concerned with depressed mood and not necessarily formal major depression, the definition of caseness is arbitrary; however, for reasons outlined in the introduction, we used a definition of 'at least moderate depression' (BDI score $\geqslant$15). The correlation between the self-report BDI and the interview-based CDSS approached unity (r=0.91) and the thresholds proposed by Addington *et al* (1993) conformed almost exactly with the subgroups defined below using the BDI. The BDI criteria will be used in this and our subsequent companion paper.

Using this definition, 70% of the sample were depressed at onset (n=55): of these, 48 (87%) dipped below the threshold for at least one post-recovery follow-up observation, 26 (54%) of whom became depressed once again and the remaining 22 (46%) stayed below threshold; 5 (9%) were depressed at all follow-up points; and 2 (4%) were depressed at all but the final follow-up point.

Twenty-three subjects were not depressed at onset: thirteen (56%) exceeded the threshold on at least one occasion (post-recovery), nine (39%) remained below threshold and one (4%) became depressed at the recovery observation but remained below threshold at all future follow-up points.

Course types

Using these criteria, all but eight patients were assigned to the following groups:

Post-psychotic depression (PPD, n=*39)*

Group I: Depressed at onset, improvement and return of depression (n=26)

Group II: No depression at onset; depressed at one or more follow-up points (n=13)

No post-psychotic depression (non-PPD, n=*31)*

Group III: Depressed at onset; no depression throughout the follow-up period (n=22)

Group IV: No depression at onset or throughout the follow-up period (n=9)

Five patients did not recover from their acute illness throughout this follow-up period and a further three patients could not be assigned to the above groups. Of the three patients unable to be assigned, two were above threshold for all but the final follow-up point and the third was depressed at the recovery observation.

The potential problem of observer bias was controlled by allocation of subjects to one of the four groups once the collection of data had been completed.

For those patients with PPD, we defined the follow-up point immediately prior to its appearance as 'pre-PPD': in other words, the point in the follow-up period where the patient manifested no depression prior to the onset of PPD.

The individual subject variation in the timing of PPD, and therefore pre-PPD points, would make any attempt at analysing the data virtually impossible using the original temporal sequence. We therefore examined data at the pre-PPD and PPD stages by realigning individual subject data at the appropriate follow-up points. As the average number of days from the discharge interview to the pre-PPD observation was 167.5 (s.d.=124.9), the default 'pre-PPD' and PPD observations for those subjects who did not develop PPD (n=31) were those at 4 and 8 months after recovery, respectively.

The PPD groups

Figure 1 shows the evolution of PPD in each of the two PPD groups (groups I and II) juxtaposed with that for positive symptoms (SAPS total score) and negative symptoms (SANS total score).

The SAPS scores remained unchanged during the PPD episode in both group I (pre-PPD *v.* PPD: t=1.84, P=0.08) and group II (t=1.34, NS). The SANS scores were also stable in group I (t<1, NS) and group II (t<1, NS).

Although the BDI score in group I reverted to that observed in the acute episode (BDI score=25), the SAPS score rose only slightly (but non-significantly).

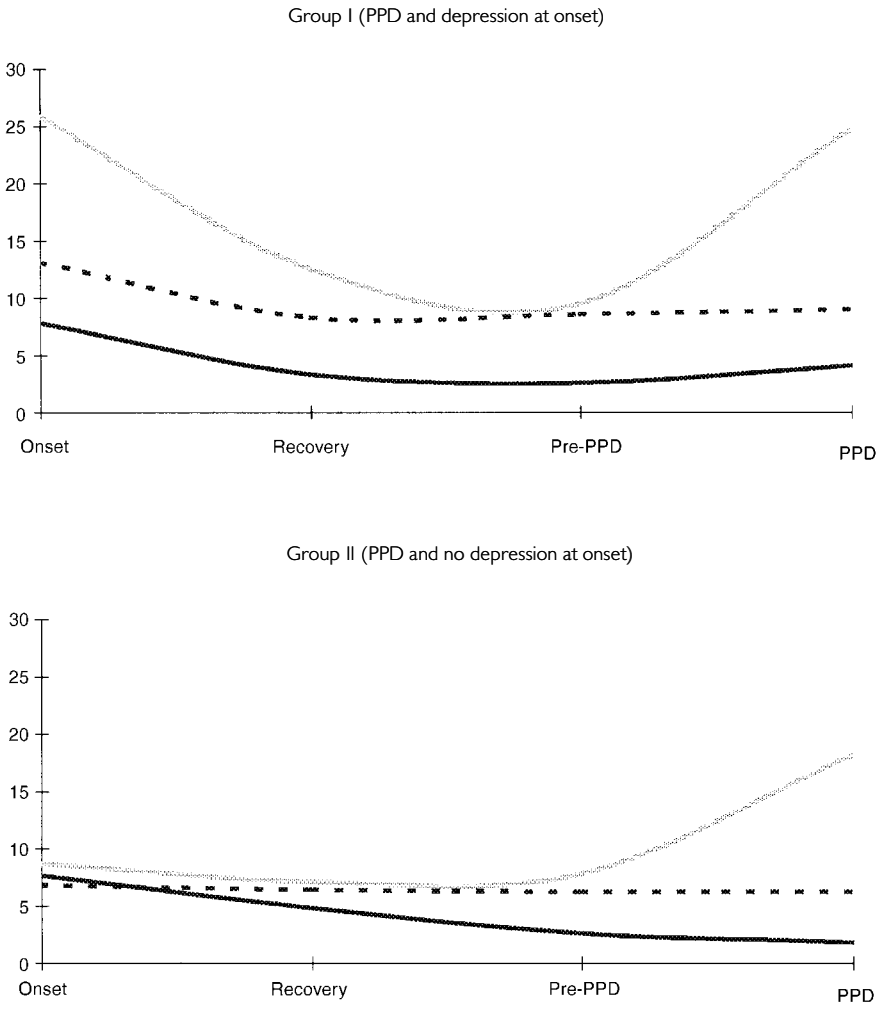

Fig. 1 Graphs depicting mean values for the course of depression and positive and negative symptoms for post-psychotic depression (PPD) groups I and II. ——, Scale for the Assessment of Positive Symptoms; ·····, Scale for the Assessment of Negative Symptoms; ·····, Beck Depression Inventory.

https://doi.org/10.1192/bjp.177.6.516 Published online by Cambridge University Press

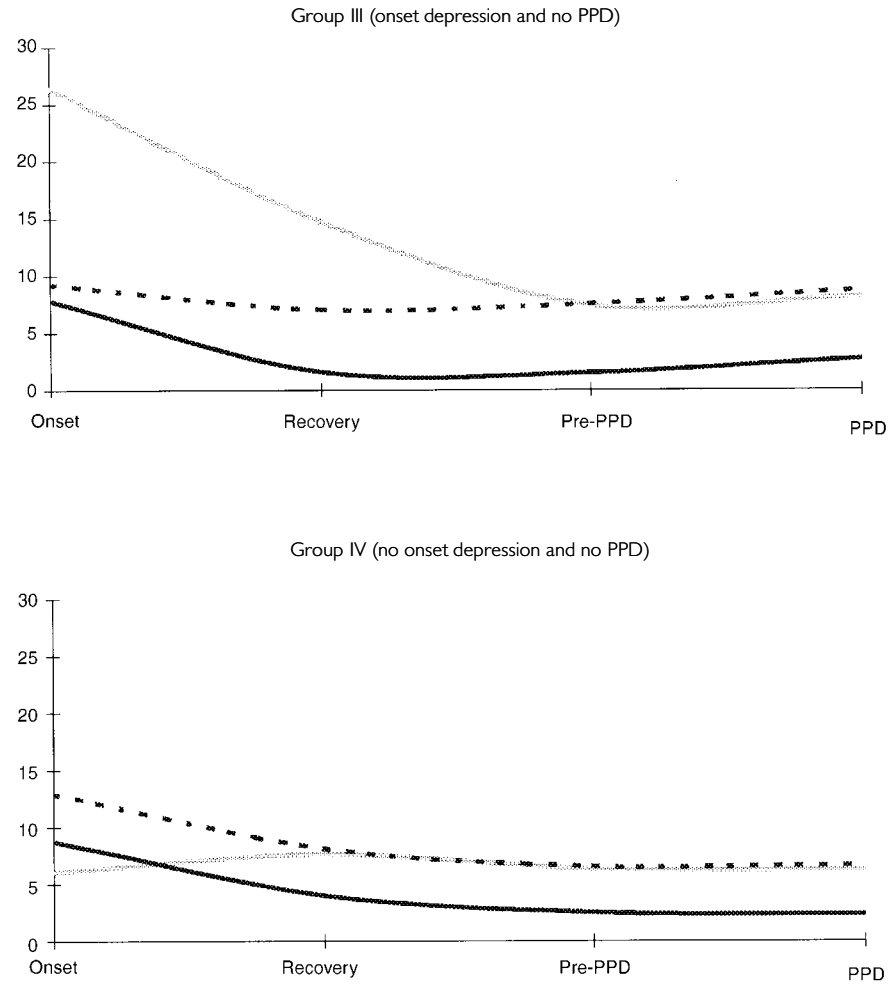

Fig. 2. Graphs depicting mean values for the course of depression and positive and negative symptoms for non-post-psychotic depression (PPD) groups III and IV. ——, Scale for the Assessment of Positive Symptoms; ·····, Scale for the Assessment of Negative Symptoms; ·····, Beck Depression Inventory.

Table 2 Mean BDI, SANS and SAPS scores for groups I–IV at onset, prior to and during post-psychotic depression (PPD)

Group	Measure	Onset (s.d.)	Follow-up point pre-PPD (s.d.)	PPD (s.d.)
PPD				
I (n=26)	BDI	25.9 (7.0)	9.5 (4.1)	24.7 (9.4)
	SAPS	7.8 (3.5)	2.7 (2.7)	4.1 (3.1)
	SANS	13.1 (4.6)	8.6 (4.8)	9.0 (3.6)
II (n=13)	BDI	8.7 (4.7)	7.7 (3.4)	18.2 (3.3)
	SAPS	7.7 (2.8)	1.5 (2.3)	2.6 (3.3)
	SANS	9.2 (4.0)	6.2 (5.7)	8.6 (4.4)
Non-PPD				
III (n=22)	BDI	26.4 (8.3)	7.4 (4.9)	8.1 (5.1)
	SAPS	8.7 (2.4)	2.5 (3.0)	2.3 (2.1)
	SANS	13.0 (4.4)	6.5 (3.7)	6.6 (3.1)
IV (n=9)	BDI	6.0 (4.6)	6.5 (3.8)	6.2 (4.4)
	SAPS	7.6 (2.7)	2.6 (3.0)	1.8 (2.9)
	SANS	6.7 (2.7)	7.5 (3.6)	6.2 (7.3)

BDI, Beck Depression Inventory; SAPS, Scale for the Assessment of Positive Symptoms; SANS, Scale for the Assessment of Negative Symptoms.

In group II the BDI score rose to 18.2, which is an unprecedented rise in this particular group, without a concomitant increase in SAPS. It should be noted that the mean level of depression during PPD was 'moderate' for the latter group and 'severe' for the former, using conventional criteria (see Williams, 1992, for a review).

The non-PPD groups

Again, the evolution of depression in patients not manifesting PPD is juxtaposed with that for SAPS (Fig. 2). In the case of group III, SAPS declines in line with depression, whereas in group IV, by definition, the decline in SAPS does not coincide with that for depression.

Overall then, in groups I and III depression and positive symptoms follow the same course (combined n=48) during the acute episode, whereas in groups II and IV (n=22) depression and positive symptoms are not synchronous at the acute episode. However, on comparing patients with and without depression at onset (BDI score $\geqslant$15), there was no significant difference in SAPS score at onset, confirming that, in general terms, any link between positive symptoms and depression is confined to a subgroup.

Comparison of PPD with non-PPD groups

Positive symptoms

In order to determine whether the presence of PPD was the result of variation in positive symptoms, the groups were compared at each follow-up point. For this analysis groups I and II (PPD) and (III and IV) (non-PPD) were combined. Table 2 suggests a small mean rise in SAPS in each of the PPD groups; combining these groups into a single PPD group (n=39) revealed a modest significant rise in SAPS from 2.3 (2.6) to 3.6 (3.2) (t=2.3, $P<0.05$) during PPD; no overall change in SANS was apparent.

This result suggests the presence of a subgroup showing a rise in positive symptoms, beyond the chance level, ranging from a 'mild' increase through to a full relapse. Thus we defined the threshold for a rise in positive symptoms equivalent to at least a 'mild' increase in symptoms; our operational definition was a rise equivalent to *at least* the margin of measurement error in the SAPS scale. From the interrater reliability data, the maximum discrepancy in

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SAPS global score ratings by M.B. and Z.I. was 3, which is equivalent to a single standard deviation. By defining subgroups of the PPD subjects using this discrepancy as a cut-off, 28 out of 39 (72%) did not display an increase in positive symptoms (i.e. a change of <3 in the SAPS global score) whereas SAPS scores for 11 subjects (28%) increased at PPD by at least the maximum interrater discrepancy. The mean SAPS global score for these 11 subjects at PPD was not significantly different from their SAPS score at onset (t=2.03, NS), suggesting that the rise in depression and positive symptoms in this subgroup was concordant with symptoms indicative of a psychotic relapse, which indeed conformed with our clinical impression (e.g. some were hospitalised). We conclude, therefore, that the remaining 28 subjects (36% of the whole sample) developed PPD *without* any marked increase in positive symptoms and were hence not displaying signs of psychotic relapse. This now becomes the new 'pure strain' PPD sample.

Negative symptoms

A similar investigation of the depressed and non-depressed subjects' extent of negative symptoms during the pre-PPD and PPD stages was also conducted. No overall change in SANS was apparent (Table 2). These results clearly indicate the stability of negative symptoms during the pre-PPD and PPD stages as subjects become depressed, irrespective of changes in positive symptoms.

Drugs and side-effects

Information on prescribed neuroleptics was obtained at each of the follow-up points and converted to chlorpromazine equivalents (*British National Formulary*, 1996). In accordance with pharmacogenic theories of depression in schizophrenia, the two groups were compared at each follow-up point to determine whether the presence of PPD was the result of neuroleptic exposure. No differences were observed at any of the follow-up points ($F<1$ for all).

Similarly, the two groups were compared for side-effects using the AIMS scale. At no point in the follow-up period did the PPD group differ in severity of side-effects using either the global or composite item measures.

Is PPD just an early sign of relapse?

It can be argued that the rise in depression during the PPD phase may be an early sign of a further psychotic episode, because dysphoria is a known precursor of psychotic relapse (Birchwood *et al*, 1989), rather than PPD *per se*. One way of addressing this issue is to establish the presence of psychotic symptoms *following* PPD. A comparison of positive symptoms after the PPD stage could be investigated only for those subjects who developed PPD at the third or fourth observation points, where follow-up data would therefore be available (i.e. at the fourth or fifth observation, respectively). Hence, there would be no follow-up data for subjects developing PPD at the fifth (and final) observation. Sixty-four per cent of subjects (n=18) who developed PPD had follow-up data after the PPD stage. For these cases, the mean SAPS global score was not higher at the subsequent observation than that observed at PPD (2.52 (2.50) *v.* 2.76 (2.66); t=0.38, NS) and thus does not support the argument that the rise in depression at PPD is a precursor to the onset of relapse.

Who develops PPD?

Of the 28 patients developing PPD, 68% were also depressed at onset. A total of 22/48 (46%) with depression at onset did not develop PPD and conversely 9/28 (32%) with PPD were not depressed at onset. Overall, the presence of depression at onset did not raise the risk of PPD (χ^2=0.63, NS). There were no differences between the groups with (n=28) and without PPD (n=31) for: age at onset, duration of illness or total number of admissions; gender, marital status or ethnicity; or being under a section of the Mental Health Act.

Depression during acute psychosis

Definition

The wide variation in depression during the acute episode was not anticipated and here we distinguish those individuals with and without depression at onset using the same criteria (BDI score $\geqslant$15). This yields a group of patients with acute psychosis who are not depressed: 39/105 (37%) for the whole cohort and 22/78 (28%) for the sample completing all follow-up points. The mean BDI score for the depressed group was 27.7 (8.5) and for the non-depressed was 7.4 (4.4), indicating that the depressed group were, on average, 'severely' depressed. Contrary to expectation, there was no difference in SAPS global score between these depression groups: depressed=8.2 (3.7); not depressed=7.8 (3.1). Similarly, there was no difference between these groups in SANS global score: depressed=11.8 (4.7); not depressed=11.2 (4.9).

Who develops depression at onset?

People with depression at onset were younger at their first episode (30 *v.* 25 years; F=6.1, $P<0.01$) and younger at inclusion to the study (40 *v.* 32; F=10.8, $P<0.001$). There were no differences between the groups in neuroleptic dose or drug side-effects.

Hopelessness and suicidal thinking

A total of 54% of non-relapsing depressed patients at the PPD stage also reported suicidal thinking or worse on the CDSS, including 18% who had made suicidal plans and 4% who had undertaken a suicide attempt designed to end in death. Similarly, 36% of those with PPD expressed a concurrent persistent sense of hopelessness about the future ($P<0.01$ for both).

One-year prevalence

In order to gauge the level of occurrence of these features using this unique longitudinal perspective, we calculated the prevalence of at least moderate severity levels of depression *or* hopelessness *or* suicidal thinking across the three follow-up points over the 12-month envelope. Excluding the acute psychotic and recovery points, the 12-month prevalence for subjects with moderate levels of depression *and* hopelessness amounted to one-third (33.3%) of the sample, whereas that for subjects who were moderately depressed *and* suicidal was 20.5%. Finally, 61% during the 12 months after discharge from hospital experienced at least moderate levels of depression *or* suicidal ideation *or* hopelessness at a follow-up observation.

DISCUSSION

To the best of our knowledge, this is the first study to investigate the course of depression in schizophrenia within a longitudinal framework using this density of follow-up observation, the nearest comparable study being that of Leff *et al* (1988). Given the

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frequency of follow-up and the assessments required, the level of concordance with the research protocol was exceptional.

On the basis of our definition of PPD, 36% (28/78) of patients in the study developed depressive symptoms of at least moderate intensity during the 12 months following an acute psychotic episode. It is important to point out that at the follow-up point immediately preceding the onset of PPD (i.e. pre-PPD) all these subjects were *not* depressed. The average time to PPD after recovery was 248 days or about 8 months, whereas the average number of days to PPD from the onset of psychosis was 304. Earlier studies have observed prevalence rates for PPD at 38% (Van Putten & May, 1978), 39% (Roy, 1981) and 45% (Leff *et al*, 1988). Our sample consists of a high proportion of chronic and multiple-relapse cases within a socio-economically depressed inner-city area, more so than in previous studies, although these similar rates of PPD suggest that chronicity or relapse history may not alone be predictive of PPD and its origin may lie elsewhere. This is underlined by recent observations of a high rate of depression in first-episode subjects (Addington *et al*, 1998), and in the present study 50% of first-episode subjects developed PPD compared with 32% for those with multiple relapses ($t=3.1$, $P<0.01$). Overall, then, the sample's bias towards youth and the differential rate of PPD in the first-episode group need to be kept in mind when considering the generalisability of the data.

Analyses reveal that, for the patients developing 'pure strain' PPD, no significant changes are observed in positive symptoms, negative symptoms, the severity of side-effects or drug dose during the follow-up period.

The results clearly indicate the stability of negative and positive symptoms during the pre-PPD and PPD stages as subjects become depressed. To our surprise, correlational analyses between SANS global score and sub-scales and BDI for depressed subjects at the PPD phase indicate no significant associations between PPD and negative symptoms (all $r<0.16$, NS). We support Siris's (1995) position that the major difference between depression and negative symptoms is 'blue mood', a valuable indicator of depression that differentiates it from symptoms such as anhedonia, alogia, affective flattening and apathy, providing a clear rationale for distinguishing negative symptoms from depression. Finally, the risk of PPD was unaffected by the presence of depression during the acute psychotic stage. This suggests that the processes underlying PPD and depression at onset are different, and in line with the theories outlined in the introduction.

An unexpected finding was that about one-third of patients in acute psychosis were not depressed and the remaining two-thirds displayed mostly severe depression. Furthermore, there were no significant differences between the positive symptoms, drug dosage or severity of side-effects between these two groups at onset. Finally, there is substantial evidence that suicidal thinking and hopelessness accompany depressive pathology in the period following remission of acute psychosis. In the context of our 1-year prevalence rates for suicidal ideation, parasuicide and suicide of 23%, which are in line with previous results of 25% (Roy, 1986) and 10% (Mortensen & Juel, 1993), there is a considerable need to establish a theoretical framework and preventive therapy for this most serious of problems, which shows a clear linkage with PPD.

The course patterns

We find evidence to support two of the course patterns outlined by Leff (1990). Depression can follow the same course as positive symptoms (during acute psychosis and relapse) and present *de novo* during follow-up without a change in positive symptoms (i.e. PPD). We found no evidence that depression emerged immediately following the remission of positive symptoms. Pharmacogenic theories are not supported either during acute psychosis or in the following 12 months. Similarly, negative symptoms remained stable whether PPD was observed or not. In addition, the study attempted to employ stringent criteria at recruitment and to define clearly the nature of PPD, and therefore we do not believe that diagnostic variation provides an explanation for these results.

Methodological considerations

In order to control for interviewer bias the BDI (self-report) and CDSS (observer-rated), two highly correlated and virtually interchangeable depression inventories (Addington *et al*, 1993), were utilised. These measures were highly intercorrelated ($r=0.91$). Interrater reliability for the SANS and SAPS was also established at various junctures throughout the study, and shown to be satisfactory.

We conclude that the experiment embodied little measurement error, and that insufficient to distort the main results.

What is PPD ?

The ICD–10 definition of PPD (F20.4) requires that, alongside a general criterion for schizophrenia during the previous 12 months, the patient must still exhibit persistent hallucinations in any modality, symptoms of thought disorder or certain negative symptoms that are not due to depression or neuroleptic medication. Based upon these criteria, none of our PPD subjects would conform to ICD PPD but would be simply depressed. We suggest that there is a strong case for further investigation of the diagnostic criteria for PPD. The ICD–10 criteria seem to be rooted in the assumption that depression in schizophrenia is an endemic feature of the psychotic process.

We have argued that although PPD is embedded in the realities of a psychotic illness, it is the beliefs or appraisals about psychosis made by those who experience it that are important (Birchwood & Iqbal, 1998; Rooke & Birchwood, 1998). In our subsequent companion paper this hypothesis is put to the test.

(First received 15 December 1999, final revision 8 June 2000, accepted 9 June 2000)

References, authors' details, clinical implications and limitations are at end of the next paper

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