Correspondence

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Collaborative therapy: framework for mental health

We agree with the sentiments expressed by Lester & Gath (2006) in their recent editorial on promoting a collaborative model of recovery, and believe that the collaborative therapy model which we have developed goes some way towards meeting the need for ‘a collaborative approach…to the development of high-quality recovery-oriented mental healthcare’.

One of the key aspects of collaborative therapy is recognising that ‘recovery’ and chronic models of healthcare are not dichotomous. Recovery is an individual process that can be assisted through the application of a model of care embracing the reality of mental illness and encompassing both the management of acute episodes and long-term health needs.

Collaborative therapy is a comprehensive therapeutic framework for consumers, clinicians, services and others to work systematically towards the achievement of optimal mental health outcomes. The aim is to deliver, within mainstream services, comprehensive psychosocial treatments that are evidence-based and individually tailored. The ability to individually tailor treatments thus allows for the incorporation of personal definitions of recovery.

The collaborative therapy framework has three components that can be applied across the spectrum of mental disorders. Engagement encompasses a comprehensive screen for issues that may be barriers to treatment (including psychiatric comorbidity), as well as a mapping of ‘collaborative partners’, who agree to be involved in the individual’s care. These may include the general practitioner, case manager, psychiatrist, vocational worker and family members. All collaborative partners are explicitly involved in the planning of healthcare for the individual.

The therapy itself can be conducted in a group or one to one with a case manager. It is run over 8–12 weeks, followed by booster sessions over a further 9 months, is based on an adaptation of the stress vulnerability model (stress vulnerability—self-efficacy) and utilises self-efficacy and self-reliance as part of the process. It provides core components of the therapeutic interventions that have established efficacy across a wide range of diagnosis, including psychoeducation, coping and relapse prevention strategies. An allied tool is the collaborative treatment journal, a small pocket journal that can chart stressors, early warning signs, coping strategies, supports and other factors that influence the course and management of an individual’s health. It is held by the consumer and places them at the centre of their treatment.

The unique appeal of the collaborative therapy framework is that it is sensitive to the structure, resources and staff-mix of particular services, and meets all consumers’ needs. This helps to ensure that there is maximum likelihood that the components developed within the collaborative therapy framework are taken up within routine service delivery structures. It has been adopted by a number of Australian mental health services, and is well accepted by consumers, their families, and health professionals.


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Physical contact with child and adolescent patients

Although I recognise and share some of the concerns about the appropriateness of certain types of physical contact with patients, I was surprised by several aspects of the survey by Blower et al (2006) of the views of child and adolescent psychiatrists.

First, I was puzzled by the fact that 1% of respondents selected the ‘do nothing’ option in response to the clinical vignette of a distressed child running towards a busy road and that the implications of such a response were not commented on by the authors.

Second, although Blower et al referred to physical examination in their discussion, the participants do not seem to have been asked about their views on this in either the questionnaire or the telephone interviews. The authors then seemed to downplay the role of physical examination and treatment in child psychiatry, both of which are becoming increasingly important.

Physical examination is essential in the assessment and management of many psychiatric conditions, including attention-deficit hyperactivity disorder, eating disorders and severe depression. Specific syndromes associated with behavioural disorders, particularly those accompanied by learning disability, may be missed without appropriate examination (Devlin, 2003). In addition, physical examination is necessary before initiating drug treatments and in monitoring for adverse effects, particularly when using stimulant drugs or atypical antipsychotics.

Knowledge, understanding and practical experience of the use of physical treatments is required as part of specialist registrar training in child and adolescent psychiatry, alongside the use of other treatments, including the various psychotherapies (Royal College of Psychiatrists, 1999). If trainees or consultants lack confidence or skills in physical examination and treatment, or feel uneasy with the physical contact this requires, it would be appropriate for them to seek further training as part of continuing professional development.


Acute and transient psychotic disorders and puerperal psychosis

Marneros (2006) addresses an important issue in his editorial on the concept of acute and transient psychosis, which is a challenge to the Kraepelinian dichotomy. He argues that acute and transient psychoses are separate from schizophrenia, schizoaffective disorder or affective disorder, based on the clinical manifestations, but he did not mention puerperal or post-partum psychosis, which also lacks a consensus of definition (Kohl, 2004). Post-partum psychosis has been described as functional psychosis with good prognosis and clinical presentation similar to acute and transient psychosis (Kendell et al, 1987). Despite a varying symptomatology, women with schizophrenia rarely experience arousal of their symptoms after childbirth (Meltzer & Kumar, 1985). Puerperal psychosis appears to occupy a clinical position which is different from schizophrenia and affective disorder.

It is of interest that acute and transient psychosis mainly affects females (Marneros, 2006), and suggests a link between puerperal psychosis and acute and transient psychosis. I therefore suggest that the concept of puerperal psychosis should be included in discussions of the concept of acute and transient psychosis.


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Author’s reply: Dr Lewin is right that puerperal psychoses are of special interest in the context of acute and transient psychoses. To our knowledge there is consensus that post-partum disorders are not distinct nosological entities (Brockington, 2004; Riecher-Rossler & Rohde, 2005) with neither ‘post-partum depression’ nor ‘post-partum psychosis’ having specific aetiology. ‘Giving birth to a child with all its biological and psychosocial consequences seems to act as a major stressor, which – within a general vulnerability-stress-model – can trigger the outbreak of all classical disorders in predisposed women’ (Riecher-Rossler & Rohde, 2005). Hence it is evident that the situation after delivery can be typical for triggering acute and transient psychoses.

Re-evaluation of our own sample of 61 women (Rohde & Marneros, 1993) with first onset of psychosis after delivery showed that according to ICD–10 criteria 18 (29.5%) should be classified as having acute and transient psychosis (Rohde & Marneros, 2000); all other diagnostic categories were also present (schizoaffective and affective disorders, schizophrenic and organic psychoses). In our sample the frequency of acute and transient psychoses was much higher than expected from the general prevalence. This might be a reason for the frequent observation that puerperal psychoses are mainly very acute, short episodes with a ‘colourful’ psychopathology and good prognosis.

Considering the few available studies we conclude that in the post-partum period acute and transient psychoses represent a disorder that is different from other psychiatric disorders but is part of a psychotic continuum.


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Cognitive impairment in euthymic patients with bipolar disorder

By prospectively verifying euthymia and controlling for the effect of residual affective symptoms Goswami et al (2006) make a significant contribution to the existing evidence on cognitive impairments in euthymic patients with bipolar disorder. However, they did not define euthymia and the diagnosis of DSM–IV bipolar I disorder, verification of euthymia and exclusion of current and past psychiatric illness or substance use disorders in patients and controls were made without structured assessments. Controls were relatives of participating patients. In addition, exclusion criteria make no mention of birth injuries, the handedness of patients and whether patients had received electroconvulsive therapy. All these factors influence results of tests for cognitive function (Ferrier & Thompson, 2002).

As the Schedule for Assessment of Psychiatric Disability assesses marital and occupational functioning, details of the patients’ marital or occupational status would have helped to better interpret the data. There is also no mention of the duration of illness (in Table 1, p. 368, duration spent in episodes is erroneously labelled as duration of illness). This variable has implications for the generalisability of findings.

A measure of the reliability and validity of the modified Kolakowska battery is not provided. The use of more systematic and better-validated instruments such as the Cambridge Neurological Inventory (Chen et al, 1995) and more than one rater to reduce assessment bias would have allowed better characterisation of neurological soft signs. About 92% of healthy controls in the current study had neurological soft signs. This unusually high prevalence could...
reflect the inappropriateness of the control group.

In the Rey Auditory Verbal Learning Test, significantly lower scores on lists A1–A5 were taken to infer a reduction in verbal memory. However, there was no difference between patients and controls for lists A6 and A7. The percentage of words retained between trials A5 and A7 would provide a purer index of retention (Thompson et al., 2005) and would help to better interpret the data.

In the future, meta-analyses of existing data and studies involving assessment of cognitive function and neuroimaging in euthymic patients with bipolar disorder should help elucidate a profile of cognitive deficits and their underlying neurobiological bases.


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Authors’ reply Certain aspects of methodology were left out of our paper owing to space constraints. Bharadwaj cites Ferrier & Thompson (2002) when questioning the exclusion criteria used in our study. Both are co-authors of our paper, which is a result of collaborative research between the Department of Psychiatry in New Delhi and Newcastle since 1998. Whenever possible, similar tests and criteria for euthymia have been used in both centres with occasional variations to respect cultural differences. Use of spouses and siblings as members of the control group was acceptable, as it brought together people of broadly similar backgrounds. Although this might have resulted in the inclusion of a limited number of controls who were at risk of developing bipolar disorder, it minimised differences between people with bipolar disorder and controls without greatly confounding our results.

For verification of euthymia participants were seen at least twice, separated by a minimum of 1 month, before they were tested. Clinical judgement of euthymia was reinforced by a Hamilton Rating Scale for Depression score <8 and a score <20 on Bech’s modification of Beigel’s Manic State Rating Scale on both occasions. The stability of mood during the intervening period was assessed clinically on a weekly basis. We were not aware of any Hindi version of the Structured Clinical Interview for DSM–III – patient version. The exclusion of other psychiatric morbidity was based on clinical interviews by two highly experienced psychiatrists, complemented by careful mapping of life charts using the techniques of Post et al. (1998).

So soft neurological signs were assessed with the widely used modified Kolakowski battery. We are unsure whether the use of other batteries, such as the Cambridge Neurological Inventory, would substantially alter our findings. Involving a second rater would perhaps increase reliability but would extend the assessment time unreasonably.

Not surprisingly, soft signs were found in the control group, but only at about one-quarter of the severity seen in people with bipolar disorder. The maximum score on the modified Kolakowski battery was 45. The maximum score for controls was 9 whereas the mean for patients was 13.9. Control scores mainly comprised minimum scores on a few of the 15 items. In a subsequent article (further details available on request) we report high levels of soft signs in the youngest patients with bipolar disorder. There is no evidence that soft signs progress with age in bipolar disorder, whereas in controls there is significant (P < 0.01) progression with age.

List A7 of the Rey Auditory Verbal Learning Test measures retention after 20 min. We have further analysed these data and found no difference between the groups.

We agree that ‘duration of illness’ actually describes ‘duration of illness episodes’. The actual mean duration of illness was 9.1 years (s.d. = 6.0). Data concerning marital status and occupation were collected but were omitted for brevity. We did not wish to control for handedness or birth injuries as potential confounders as we regarded these differences to be part of the spectrum of people with bipolar disorder. We did not include those who had recently received electroconvulsive therapy (> 6 months). Finally, we would agree that there is a need for meta-analyses and have recently published such a study (Robinson et al, 2006).


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Delay in onset of action of antidepressants

In an important editorial, Mitchell (2006) marshalls evidence to show that we do not have to wait 2 weeks for antidepressants to work.

Why has it been so difficult so far to show that they work in the first few days? In addition to the reasons that Mitchell sets out, I should like to mention a further problem. If you analyse the results on a day-by-day basis, it is hard to obtain sufficient statistical power to distinguish the early response to the drug from the response to the placebo, since you have just the scores for that day.

In 1996 my colleagues and I published evidence that the fall in scores on the Hamilton Rating Scale for Depression followed an exponential decay curve with a correlation coefficient of 0.99 (Priest et al, 1996; Livingston & Clark, 1997). This observation corresponds with Mitchell’s remarks on the steep fall in scores in the first 2 weeks. A comparison of the slope of the curve for the active drug with the placebo, using all of the data, gives a very sensitive way of testing for efficacy.

By plotting the log of the depression scores against time, a straight line is obtained. Thus the recovery from depression
is of one piece, with a smooth process throughout. The clear implication is that there is no delay in the onset of action, either of the active drug or of the placebo. By using the slope of the graph, one can use all of the trial results, not just those on a particular day. The statistical power is greatly increased and the distinction between drug and placebo enhanced.


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Author’s reply I thank Professor Priest for further insights concerning the difficulty in obtaining sufficient statistical power to distinguish the early drug-placebo response. This is correct but the reason for the difficulty is mainly that the absolute difference between drug and placebo is initially small and when combined with typically low sample sizes the overall power to detect a true difference is insufficient. That said, if the aim of the study is to discover how early an effect is manifest there is no alternative to regular early measures.

Professor Priest then anticipates our next piece of work to which I previously eluded – examination of the trajectories of antidepressant response. He quite cleverly observes that a comparison of the slope of the response curves for the active drug and placebo is in effect a test of efficacy. It is also suggested that the response is often neither delayed nor steady but actually rapid (or perhaps more accurately ‘accelerated’). Allow me to illustrate this point further (Fig. 1). The rate of change of those taking placebo is poor compared with a hypothetical antidepressant with a ‘steady’ or ‘rapid’ onset. A delayed onset, as so often suggested, is not illustrated, but I expect readers will be able to sketch their own view of the delayed trajectory. In fact several ‘delayed’ paths are possible, depending on whether there is a catch-up with the steady path and if so, when.

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Primary agoraphobia as a specific phobia

The elegant study of 1920 participants from the Baltimore Epidemiologic Catchment Area programme concluded that ‘the implied one-way causal relationship between spontaneous panic attacks and agoraphobia in DSM–IV appears incorrect’ (Bienvenu et al, 2006). Bienvenu et al echo the arguments of many researchers, beginning with Marks (1987), that agoraphobia without panic attacks (primary agoraphobia) should be reinstated in DSM–V as a stand-alone diagnosis as in ICD–10.

It has been argued that evolutionary biological reasoning predicts the existence of a ‘hard-wired’ primary stand-alone agoraphobia, which should be classified with other specific phobias (Bracha, 2006). Specific phobias have been considered as conserved traits that enhanced survival during the human era of evolutionary adaptedness (Nesse, 1999; Bracha, 2006). Primary agoraphobia may similarly be traced back to the fact that humans relied on arboreality as a major escape response long after they diverged from chimpanzees. Homo sapiens expanded beyond its densely forested East-African indigenous niche into sparsely wooded habitats (savannahs and water-front dunes) only about 70,000 years ago. In sparsely wooded habitats, anxiety in wide-open spaces was arguably a survival-enhancing trait since opportunities for arboreal escape from large predators were limited (Bracha, 2006). These arguments may be relevant to psychiatric classification and contribute to the ‘neuroscience research agenda to guide development of a pathophysiologically based classification system’ emphasised in the research agenda for DSM–V (Kupfer et al, 2002).

If, as one of us (Bracha, 2006) has argued, the two types of agoraphobia have different modes of acquisition, there might be some clinical implications. Primary agoraphobia might, like other specific phobias, be especially amenable to virtual reality exposure treatment. In contrast, agoraphobia secondary to panic attacks can be classified in DSM–V and treated along with post-traumatic stress disorder (and other fear-memory-overconsolidation disorders, which are misclassified as specific phobias in DSM–IV–TR, e.g. hospital phobia, dentist phobia, dog phobia, bird phobia, and bat phobia).

Finally, contrary to myth, predictions based on brain evolution are eminently testable/falsifiable (Nesse, 1999). Some 30 such predictions are elaborated elsewhere (Bracha, 2006).


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Authors’ reply We are grateful to Bracha et al for their interesting comments regarding primary agoraphobia as a potential evolutionary adaptation. First, we should clarify that we did not propose an additional diagnostic category; rather, we proposed that agoraphobia itself should be a stand-alone diagnosis in DSM–V (as in ICD–10), like other phobias. Subdividing what has historically been called agoraphobia may be useful, but we are concerned that clinicians and researchers are adopting Klein’s narrower conceptualisation of agoraphobia as simply fear of panic in typical agoraphobic situations (Klein, 1980), without considering the possibility that a broader conceptualisation may be useful. Epidemiologists are increasingly adopting the definition of agoraphobia as ‘fear of fear’ (e.g. Grant et al, 2006), rather than the broader fear of difficulty in escaping, etc., in characteristic situations. As noted in our article, what has historically been called agoraphobia is strongly but not exclusively associated with panic, and, when the association exists, agoraphobia is not always preceded by panic.

The concept of an evolutionary basis for the development of phobias is not new (Seligman, 1971; Marks, 1987). Nevertheless, the reasons why people with agoraphobia develop fear and avoidance of particular situations remain important. Bracha et al suggest that fear of open spaces is an evolutionary remnant of primates’ use of trees to escape from predators. However, although some people with agoraphobia are fearful of open spaces, the list of typical agoraphobic situations is broad (Marks, 1987). Thus, hypotheses with an evolutionary basis to explain agoraphobia will be expected to cover reasons why persons fear and avoid a variety of situations. Although it is difficult to ‘prove’ such hypotheses, we agree with Bracha et al that researchers can make falsifiable predictions that can continue to illuminate the field.

We agree that cognitive–behavioural techniques may be particularly important for persons whose agoraphobia is primary. However, many people with agoraphobia can benefit from such treatment, whether the syndrome is primary or secondary (Klein, 1980).
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