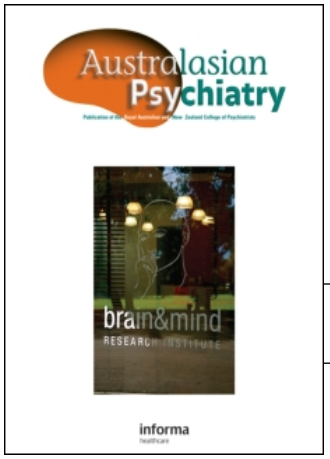

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CORRESPONDENCE

Paediatric bipolar disorder

DEAR SIR,

We thank Jairam for his thoughtful comment[1] on our paper concerning the controversy surrounding the diagnosis of paediatric bipolar disorder (PBD) especially among pre-pubertal children.[2] In response, we emphasize that the British National Institute for Health and Clinical Excellence (NICE) guidelines on bipolar disorder (BD) regard the bipolar spectrum in children as highly speculative and the case for inclusion in evidence-based practice remains unproven. They advocate caution until further research evidence becomes available.[3]

The main PBD hypotheses are that BD can be recognized reliably in childhood, and that early intervention will improve the course of the disorder. This line of research encounters the well recognized difficulty of long-term prediction in psychiatry, especially if using relatively frequent and non-specific symptoms to predict the emergence of uncommon conditions at a much later developmental stage. Jairam references Chang's paper, ''Adult bipolar disorder is continuous with paediatric bipolar disorder'',[4] which highlights the similar phenomenology between children and adults with BD. However, Chang's review in the *Canadian Journal of Psychiatry* was accompanied by a review by Duffy,[5] concluding there is no evidence of BD in pre-pubertal children. In particular, Duffy noted that in longitudinal studies of high-risk children of parents with well-characterized BD, ''there have been no observations of diagnosable BD in children under the age of 12 years''.

Alternative clinical hypotheses are evident in nearly all the studies of PBD, which has remarkably high levels of comorbidity; in fact, this is one of the most reproducible findings about the various childhood BD phenotypes.[6] These 'comorbidities' could be viewed as differential diagnoses and potential explanations for the mood dysregulation. For example, Rucklidge, in a ground breaking study of the psychosocial factors associated with a diagnosis of 'narrow phenotype' PBD in Christchurch,[7] found that over 50% had a history of trauma and that 21% met criteria for lifetime posttraumatic stress disorder (compared with 10% trauma exposure, 0% posttraumatic stress disorder among controls). Childhood trauma may well be implicated in these cases of mood dysregulation rather than PBD.

As NICE recommend, the threshold for a diagnosis of early-onset BD should be set particularly high as the implications are serious. PBD has been conceptualized as a life-long condition requiring long-term medical management beginning early in childhood. The risk of excessive pharmacotherapy, particularly in pre-pubertal children cannot be understated. In our paper,[2] we alluded to this with a reference to Wonodi *et al.*,[8] who examined 118 paediatric psychiatric patients aged 5–18 years on atypical antipsychotics for at least 6 months and found that 11 (9%) had tardive dyskinesia. There have been large numbers of fatalities relating to atypical antipsychotics[9] in the USA. The diagnosis of PBD is a major driving force for atypical antipsychotics being prescribed as long-term 'mood stabilizers' to children. Another aspect worthy of further discussion is the effect on a child's developing narrative of self and the meaning of their emotional life in the context of the PBD label.[10]

REFERENCES

1. Jairam R. Comment. *Australasian Psychiatry* 2007; **16**: 85–86.
2. Parry P, Allison S. Pre-pubertal paediatric bipolar disorder: A controversy from America. *Australasian Psychiatry* 2007; **16**: 80–84.
3. National Institute for Health and Clinical Excellence. *National Clinical Practice Guideline Number 38: Bipolar Disorder: the Management of Bipolar Disorder in Adults, Children and Adolescents in Primary and Secondary Care.* London: National Collaborating Centre for Mental Health, 2006.
4. Chang K. Adult bipolar disorder is continuous with paediatric bipolar disorder. *Canadian Journal of Psychiatry* 2007; **52**: 418–425.
5. Duffy A. Does bipolar disorder exist in children? A selected review. *Canadian Journal of Psychiatry* 2007; **52**: 409–417.
6. Biederman J, Faraone SV, Wozniak J *et al.* Clinical correlates of bipolar disorder in a large, referred sample of children and adolescents. *Journal of Psychiatric Research* 2005; **39**: 611–622.
7. Rucklidge JJ. Psychosocial functioning of adolescents with and without paediatric bipolar disorder. *Journal of Affective Disorders* 2006; **91**: 181–188.
8. Wonodi I, Reeves G, Carmichael D *et al.* Tardive dyskinesia in children treated with atypical antipsychotic medications. *Movement Disorders* 2007; **22**: 1777–1782.
9. Moore TJ, Cohen MR, Furberg CD. Serious adverse drug events reported to the Food and Drug Administration, 1998–2005. *Archives of Internal Medicine* 2007; **167**: 1752–1759.
10. Purcell WJ. Pediatric bipolar disorder and the destruction of lived experience: a case study. In: Olfman S, ed. *Bipolar Children: Cutting-Edge Controversy, Insights, and Research.* Westport, CT: Praeger, 2007; 95–106.

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Paediatric bipolar disorder

DEAR SIR,

In their recent publication, Parry and Allison set out to explore the rapid rise in the diagnosis of bipolar disorder in the paediatric, particularly pre-pubertal, age group in the USA over the past decade and to look at associated controversies.[1] It is important that clinicians in Australia have our own debate on this particularly important subject, and to do that we must be adequately informed and unbiased. It is not just an American phenomenon. The international literature[2–6] refutes the rise in diagnosis and highlights the need for clinical epidemiological reliability and diagnostic validity studies in view of the claims that paediatric bipolar disorder (PBD) carries high morbidity and psychosocial dysfunction because of its chronic and frequently rapid-cycling symptoms, high comorbidity with disruptive behaviour disorders and relative treatment resistance.[7–9] This is currently one of the most active and controversial areas of clinical and research interest in child psychiatry.

There seems a pervading sense of urgency in the US as public health implications of the increasing diagnosis of bipolar disorder affecting children and adolescents are publicized. Current literature depicts the disease as devastating, with substantial impairment across psychosocial domains, high risk of suicide, psychosis, significant familial aggregation, and protracted illness course in which the classically described cycles of disease followed by well periods are rarely observed.[10] Early recognition is called

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for in both acute and maintenance treatment of bipolar spectrum disorders in children and adolescents in order to ameliorate ongoing symptoms and reduce or prevent serious psychosocial morbidity that usually accompanies this illness.[11]

The diagnosis of PBD poses problems as the diagnostic criteria are softened and radically inflated numbers result. Comorbidity reportedly between attention deficit hyperactivity disorder (ADHD) and bipolar disorder, oppositional defiant disorder (ODD), conduct disorder, posttraumatic stress disorder (PTSD) and other disorders of mood regulation are not considered and present enormous diagnostic and treatment challenges, evidenced by emerging reports that mania is being misdiagnosed as ADHD.[12] In children who present with both the DSM-IV and non-DSM-IV phenotypes, assessment should include careful evaluation and systematic monitoring of all abnormal behaviours to explore stability and change over time in diagnosis and impairment and medication used with the utmost responsibility. I would stress the complexities of making such a serious diagnosis at a stage of crucial development, in view of ''lack of maternal warmth'' being quoted by proponents of the PBD diagnosis in this age group as a predictor of faster relapse after recovery from mania[13] and in view of the role of the family in the onset and outcome of childhood disorders. Family dynamics are vitally important and there is a need to empirically assess which family processes are important for specific childhood disorders.[14] Discrepancies between reports of mother, child and father in childhood disorders are an inherent difficulty as sometimes parents are relieved when a diagnosis explains their concerns to date.

Research evidence is still unconvincing. The paediatric samples followed up have been of small to modest sizes, and subjects have been followed up infrequently or for relatively brief periods. So far, no study has prospectively collected syndromal and subsyndromal course data on children and adolescents representing the full spectrum of bipolar phenotypes, in particular bipolar disorder not otherwise specified.[15] Many children and adolescents cannot be meaningfully diagnosed using DSM-IV, and the variety of bipolar phenotypes observed in clinical practice remains unclarified.[8] Several research groups have published studies using semi-structured interviews to examine the cross-sectional presentation of bipolar 1 disorder in child and adolescent cohorts.

Traditional views have been shaped by the DSM, and PBD will be no exception. If DSM-V was to encompass some of the postulated PBD phenotypes, my major concern is that the role of developmental theory would take a backstage. Of further concern is that the symptom checklist type of diagnosis of PBD is dangerous, and so also is the recommended treatment, mainly because the symptoms can also be found in other disorders such as complex PTSD, ADHD and ODD, and the pharmacotherapy used in one may not be suitable for the other and in fact may be dangerous.

Has the relationship between the pharmaceutical industry, academic medicine and the national drug authorities affected the clinical practice of child psychiatry? ''Many leading researchers in this area,'' says Mary Burke, ''have financial relationships with the manufacturers of the drugs recommended for the treatment of PBD and although such relationships are not illegal, our credibility with the public is being jeopardized and constantly questioned''.[16] The reduction of child psychiatry to a biological model is in competition with those of us who espouse an integrated perspective of developmental psychopathology without blinding ourselves to the fact that some seriously disturbed children do require pharmacotherapy. The role of cumulative trauma, including attachment trauma in early life, the role of environmental stressors and family relationships cannot be disregarded. Yet, the biomedical model appears to be taking over and limiting treatment options available to psychiatrists. The bio-psycho-social model is dying. Psychiatry journals lately are full of multicolour scans and complex genetic maps indicative of the fantastic progress being made in understanding the biology of mental disorders. Actual human beings with mental disorders have practically disappeared from their pages. The patient is 'disappearing'[17] from psychiatry. Are we now running the risk of children and families 'disappearing' from child psychiatry?

REFERENCES

1. Parry P, Allison S. Pre-pubertal paediatric bipolar disorder: a controversy from America. *Australasian Psychiatry* 2008; **16**: 80–84.
2. Srinath S, Janarolha N, Reddy YC. A prospective study of bipolar disorder in children and adolescents from India. *Acta Psychiatrica Scandinavia* 1998; **98**: 437–442.
3. Moreno C, Laje G, Blanco C. National trends in the outpatient diagnosis and treatment of bi-polar disorder in youth. *Archives General Psychiatry* 2007; **64**: 1032–1039.
4. Holtman M, Bolte S, Poustka F. Rapid increase in rates of bi-polar diagnosis in youth: True bipolarity or misdiagnosed severe disruptive behaviour disorders? *Archives General Psychiatry* 2008; **65**: 324–327.
5. Meltzer H, Gatward R, Goodman R. Mental health of children and adolescents in Great Britain. *Advances in psychiatric Treatment* 2004; **10:** 1–3.
6. Holtman M, Bolte S, Goth K. Prevalence of the CBCL-paediatric bipolar disorder phenotype in a German general population sample. *Bipolar Disorders* 2007; **9**: 895–900.
7. Staton D, Volness LJ, Beatty WW. Diagnosis and classification of paediatric bipolar disorder. *Journal of Affective Disorders* 2008; **105**: 205–212.
8. Geller B, Zimmerman B, Williams M. Six month stability and outcome of prepubertal and early adolescent bipolar disorder phenotype. *Journal Child Adolescent Pharmacology* 2000; **10**: 165–173.
9. Finding RL, Gracious BL, McNamara NK. Rapid continuous cycling and psychiatric co-morbidity in paediatric bipolar 1 disorder. *Bipolar Disorder* 2001; **3**: 202–210.
10. Axelsom D, Birmaher B, Strober M. Phenomenology of children and adolescents with bipolar spectrum disorders. *Archives General Psychiatry* 2006; **63**: 1139–1148.
11. Harrington R, Myatt T. Is preadolescent mania the same condition as adult mania? A British perspective. *Biological Psychiatry* 2003; **53**: 961–969.
12. Biederman J, Klein RG, Pine DS. Resolved: Mania is mistaken for ADHD in prepubertal children. *Journal of the American Academy Child Adolescent Psychiatry* 1998; **37**: 1091–1099.
13. Geller B, Tillman R, Craney J. Four year prospective outcome and natural history of mania in children with a pre-pubertal and early adolescent bipolar disorder phenotype. *Archive General Psychiatry* 2004; **61**: 459–467.
14. Wamboldt M, Walmboldt F. Role of the family in the onset and outcome of childhood disorders: selected research findings. *Journal American Academy Child Adolescent Psychiatry* 2000; **39**: 1212–1219.
15. Weller RA, Weller EB, Tucker SG. Mania in pre-pubertal children: has it been underdiagnosed? *Journal of Affective Disorders* 1986; **11**: 151–154.
16. Burke MG. Commentary by a child psychiatrist practicing in a community setting. *Journal of Child and Adolescent Psychopharmacology* 2007; **17**: 295–299.
17. Hellerstein DJ. The disappearing patient. *Medscape General Medicine* 2007; **9**: 34 (webcast video editorials).

Joan Haliburn
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Paediatric bipolar disorder

DEAR SIR,

In his comment on the recent article by Parry and Allison,[1] Jairam mentions that paediatric bipolar disorder had been recognised 150 years ago by Esquirol.[2] This is somewhat misleading.

The first descriptions of what might now be called bipolar disorder in adults came from the 1850s. It was not until the end of the 19th century that manic depressive illness was outlined and not until the 1920s that the term came into widespread use for any age group.[3]

At the time Esquirol was writing, mania was a common term used for insanity in general, but despite this Esquirol did not use the term mania for the case to which Jairam refers. The case, which was that of an 8-year old boy, was described in a section headed 'Folie', an even more generic term for insanity, and was preceded by a statement that infancy is secure from insanity.[4]

The case that supposedly is a first description of paediatric bipolar disorder is as follows:

> "In 1814, I took over the care of an 8 year old child. The child was physically healthy and had normal cognitive function. He had been frightened badly by his governess during the siege of Paris. A lot of what he had to say was appropriate. But nothing could restrain him. He frequently ran away from his mother and governess and wandered around the city. He often went down into the court of the hotel and ordered a team of horses, pretending to be the master. He would claim confidently that he had won a large sum of money in the lottery. If he passes by a stall, or a shop, he might grab the money his mother or other customers had paid for their purchases. He often insults, provokes, or strikes people he meets, especially those visiting his mother. As soon as he sits down anywhere he falls asleep. When he wakes up, he creates pandemonium. He regularly abuses his mother and is unwilling to do anything she asks." (p. 30)[4]

This is all the information Esquirol offers on this case. He gives no diagnosis. Elsewhere in the book, he picks out overactivity, disinhibition and loquacity as the leading features of hysteria. It would be interesting to obtain readers' formulations of this case.

REFERENCES

1. Parry P, Allison S. Pre-pubertal paediatric bipolar disorder: a controversy from America. *Australasian Psychiatry* 2008; **16**: 80–84.
2. Jairam R. Comment. *Australasian Psychiatry* 2008; **16**: 85–86.
3. Healy D. *Mania.* Baltimore: Johns Hopkins University Press, 2008.
4. Esquirol JED. *Des Maladies Mentales. Considéeréees sous les rapports Médical, Hygiéniques et Médico-Légal.* Paris: JB Bailliere, 1838.

David Healy
Cardiff, UK

Acute trauma response at a conference abroad

DEAR SIR,

It is relatively uncommon for clinicians dealing with post-traumatic stress disorder (PTSD) to be called upon to use practice guidelines in an acute setting, given that it is unlikely that this type of practitioner would be present in a situation where the skills and knowledge can be directly applied in this context. We report the case of a traumatic incident affecting a large group of people, where recently released guidance[1] was utilized in a practical setting.

The Pharmaceutical Society of Australia regularly conducts off-shore educational events, and in 2007 a pre-conference seminar based in Morocco attracted 190 conference delegates. Early in the course of this event, an incident involving a violent attack by a mentally ill man resulted in the hospitalization of two members of the conference party in the city of Marrakech. As a result of machete wounds that included serious lacerations and fractures, these patients required an overnight stay in local hospital, sutures, intravenous antibiotics, CT scans, x-rays, and tetanus inoculations. Both were subsequently transported from Morocco for further treatment. A security guard from the hotel complex was also seriously injured. The assailant was alleged to have been an ex-employee of the resort hotel, said to have been recently released from a psychiatric hospital about 700 kilometres from the place of the attack. Considerable information detailing the attack was reported in Australian and other international media within hours of the event. Particularly in the period immediately after the attack, many of the people present were disturbed and unsettled by these events. At the time, a travel advisory from the Australian Department of Foreign Affairs and Trade suggested that Australians travelling to Morocco should exercise a high degree of caution because of a high threat of terrorist attacks against Western interests and recent suicide bombings, and some delegates were unsettled by the possibility that the attack might have been related to terrorism.

The conference organizers were in a unique position of coordinating a debriefing session provided by a speaker at the conference (LM). Prior to the session, which was attended by approximately 70 of the conference delegates, the Australian Centre for Posttraumatic Mental Health (ACPMH) guidelines for people exposed to traumatic events[1] were made available to all delegates. Given the proximity of the event to the release of the document, it is probable that this occasion was the first time that it had been used 'in the field' for a group of people affected by a traumatic event. The debriefing allowed the conference staff to provide clarification of the events surrounding the incident, as well as the opportunity to convey apologies and messages of empathy from various parties including the King of Morocco's delegation, the Mayor of Marrakech, the Moroccan Ministry of Tourism, the police, the manager of the hotel, and the conference staff. There was also discussion surrounding the role of the press. The session then focused on an exploration of the thoughts of the participants regarding the incident, discussing feelings such as anxiety and helplessness. The facilitator encouraged the participants to view the incident from a positive perspective, emphasizing strengths such as coping skills, supports and the sense of bonding that had emerged among those affected.

A feedback questionnaire was designed after detailed discussion with conference organizers, and the availability of the form was announced in a subsequent conference session. The voluntary nature of participation was stressed, and the questionnaire was copied by conference staff and left on

tables at tea breaks and lunchtime. Reponses received from 71 of approximately 190 delegates who had been on site in Marrakesh were completed 7–9 days after the incident. Twenty-five respondents described some form of first-hand relationship to the incident: 10 had arrived at the scene shortly afterwards, four respondents had arrived shortly afterwards and assisted, five reported that they had witnessed the incident first-hand, three provided personal assistance but not at the scene of the incident, and three responded that they had both witnessed the incident and provided personal assistance. The remaining 46 respondents reported that they had only heard about the incident afterwards. Descriptors most commonly selected by respondents were 'sad' ($n = 46$), 'anxious' ($n = 27$) and 'powerless' ($n = 21$). Younger respondents (aged < 60 years) were more likely to report feeling either fearful or helpless ($p < 0.001$ for each). Those with first-hand exposure to the incident were more likely to report that they felt 'panicky' than others who had heard about the event afterwards ($p < 0.05$). Self-help measures most often described as useful were 'talking with friends' ($n = 58$) and 'talking with the victims' ($n = 40$). In addition, 23 respondents described specific benefit from attending the debriefing session and 21 reported that they derived benefit from 'drawing on past experiences'. Most respondents reported that they expected ''not to think about the incident very much at all in the future'' ($n = 12$) or ''I may think about the incidents from time to time but don't expect to be troubled by them'' ($n = 57$). Three participants selected ''I may think about the incidents from time to time and they may continue to upset me'' and one respondent chose the alternative that specified ''I may need to seek some form of help or counselling about the incident at some time''. Several participants reported that the incident had prompted them to recall other previous traumatic events such as armed robberies of their pharmacies.

It is thought that this instance is the first time where the recently released ACPMH guidelines for helping others following frightening or distressing events have been used for a group of people in a practical setting. This report documents a unique first-hand perspective of trauma in an unfamiliar setting that resulted in an opportunity to counsel, assist victims and participants, and provide specialist advice to the conference organizers.

REFERENCE

1. PFA. *Helping Others*. Melbourne: Australian Centre for Posttraumatic Mental Health. https: www.unimelb.edu.au. Accessed 16 July 2007.

Linda McCarthy and Christopher Alderman
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The gain of suicide

DEAR SIR,

The recent publication, *The Loss of Sadness*,[1] helps to explain one of the discrepancies of suicide scholarship. In 1964, Stengel reported that 33.3% ''of people who commit suicide have been suffering from a neurosis or psychosis or severe personality disorder''.[2] Less than two decades later, Robins found mental disorder in 94% of patients who completed suicide;[3] that is, more than 2.5 times higher. Subsequent psychological autopsies (Robins' report was an early example) have supported this higher rate.

Robins[3] used the St Louis Suicide Study Criteria, which were closely related to the Feigner Criteria, which in turn, were a forerunner of the DSM criteria.

Horwitz and Wakefield are critical of the DSM criteria of major depressive disorder (MDD), stating that this a list of symptom which pays no heed to contextual matters (loss and other unwelcome events).[1] They state that the DSM MDD criteria lead to normal sadness being misdiagnosed as a disorder. They cite sociological studies that provide evidence that stressful events can result in features ('symptoms') that are indistinguishable from the DSM MDD criteria, but that when taken in context, these do not result in the diagnosis of MDD. Individuals who experience sad events may construe their symptoms as the natural reaction to their experience, may not consider themselves disordered and generally do not seek medical assistance.

The point we make is that the criticism made by Horwitz and Wakefield[1] can also be leveled at the diagnostic criteria used by Robins[3] because the St Louis Suicide Study Criteria and the Feigner Criteria are lists of symptoms with no contextual considerations.

Robins gives case vignettes of 63 people who were diagnosed with depression prior to suicide. Case 051 was a 61-year-old male who had been ''a highly successful lawyer until a few months before his death''. A long time gambler, he generated serious gambling debts. He embezzled money from his firm and was asked to resign. He was forced to sell his house but lost the proceeds gambling, and 1 week before his suicide he had to take his wife to live with their son. Case 056 was a 56-year-old man who had been living with a female partner for 20 years. She suffered a stroke and was moved to a nursing home 4 months before his suicide. ''From the time the wife was taken away, he seemed totally lost and despondent and would ask his neighbors the same question over and over: 'What am I going to do?''. Case 075 was a single 86-year-old never married retired dentist. He was suffering ''1) chronic asthmatic bronchitis, 2) prostatic hypertrophy, 3) hypotensive vascular heart disease, and 4) generalized arteriosclerosis''. He had prostatic surgery. He developed a tumor under his nipple which was surgically removed 5 weeks before his suicide. The only time he mentioned ''insomnia, anorexia and depression'' was after his last discharge from hospital. Case 011 was a 57-year-old woman with rheumatic heart disease who had a foot severely crushed in an automobile accident. ''The informant believed that the leg injury and the feeling of disgrace concerning her appearance and intelligence were the chief stresses that may have contributed to her suicide.''

An older diagnostic system listed endogenous/biological depression and reactive depression. Endogenous depression was usually a severe depression, the hallmark feature being that it arose without external cause. Reactive depression was the depression which arose in the aftermath of an unwelcome event. The demarcation between normal sadness and reactive depression is indistinct and relies on the attitude of the 'patient' and the diagnostician.

In 1955, Sainsbury made a useful contribution.[4] He studied 390 suicides and concluded that mental disorder

was the principal factor in 37% of cases (similar to the 33.3% of Stengel) and a contributory factor in 47% of cases. The sum of these principal and contributory factors is 94% (exactly that of Robins).

When considering the role of mental disorder in suicide, it is important to think about primary and secondary categories, and to place cases in context. Psychiatry can be expected to do something about mental disorders, but little about most other aspects of life. A diagnostic system that acknowledges the importance of context would provide revised psychological autopsy outcomes.

REFERENCES

1. Horwitz A, Wakefield J. *The Loss of Sadness: How Psychiatry Transformed Normal Sorrow into Depressive Disorder*. Oxford: Oxford University Press, 2007.
2. Stengel E. *Suicide and Attempted Suicide*. Middlesex: Penguin Books, 1964.
3. Robins E. *The Final Months*. London: Oxford University Press, 1981.
4. Sainsbury P. *Suicide in London*. London: Chapman and Hall, 1955.

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DSM-IV cure for post-traumatic stress disorder

DEAR SIR,

For those that struggle with the challenge of providing exposure therapies and other treatments for post-traumatic stress disorder (PTSD), there is good news. Medico-legal reports by learned colleagues have convinced me there is an easier remedy.

Let us take an uncomplicated case, say a horrific motor vehicle accident without physical injury or other ongoing stressor. When first assessed at say 9 months, PTSD is diagnosed and a suitable prognosis is proffered – full or partial but useful degrees of recovery are possible, but chronicity with a fluctuating course may yet ensue. It is too early to allocate an assessment of permanent psychiatric impairment. On review at 2 years, symptoms have moderated somewhat (at least at the time of the reassessment) and the DSM-IV PTSD criteria are no longer met. A diagnosis of an adjustment disorder with anxiety is allocated. Now here is the brilliance of the cure. The simple act of diagnosing an adjustment disorder (instead of PTSD in partial remission) means that the patient has to recover within at most 6 months and probably a lot sooner, as it is now some time since the original stressor. So zero impairment can be confidently assessed and there is no need for more of that troublesome exposure work, or any other treatment for that matter.

Importantly, for this cure to be effective in practice it is essential to wear a cross-sectional pair of spectacles – longitudinal ones don't work. It is also preferable not to have read conclusions that have been drawn from the Australian National Survey of Mental Health and Well-Being,[1] where the group in the population who have sub-syndromal symptoms were found to carry at least half the population burden of impaired mental health and social role performance.

REFERENCE

1. Korten A, Henderson S. The Australian National Survey of Mental Health and Well-Being: Common psychological symptoms and disablement. *British Journal of Psychiatry* 2000; **177**: 325–330.

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