

There is another aspect of this issue that is worthy of particular emphasis – the use of long-acting benzodiazepines in older people, as opposed to short-acting agents. There is evidence that elderly people who are taking benzodiazepines with a long duration of action are particularly prone to suffer falls, and Hemmelgarn *et al*, (1997) have shown that there is a significantly increased risk of being involved in a motor vehicle crash for an older person on a long-acting, but not a short-acting, benzodiazepine. While Taylor *et al* (1998) refer to advice that diazepam and chlorthalidopoxide may be inappropriate for use in older people, they do not specifically address this aspect of benzodiazepine use. On the basis of the information given with regard to benzodiazepine type, 274 (44%) of 621 benzodiazepine users in the Liverpool sample were taking a long-acting agent (elimination half-life ≥ 24 hours). Comparative data for 1985 in North America (Ray *et al*, 1989) showed that one-third of older benzodiazepine users were taking long-acting drugs and, therefore, the even higher use in the 1990s is of concern, given the increasing availability of short-acting alternatives. The Liverpool data would also appear to suggest that the use of long-acting benzodiazepines is more common in the anxiolytic class (88/135, 65%) than in the hypnotic class (186/486, 38%) of benzodiazepines. This may be explained by the frequent attention placed on the hangover effect of long-acting hypnotics, with the adverse effects of long-acting daytime anxiolytics receiving less attention.

It is worth noting that these findings refer to the community-dwelling elderly and that many such older people remain independent and continue to drive and, therefore, may be vulnerable to the adverse effects of long-acting benzodiazepines in the course of their normal daily activities.

Hemmelgarn, B., Suissa, S. & Huang, A. (1997) Benzodiazepine use and the risk of motor vehicle crash in the elderly. *Journal of the American Medical Association*, **278**, 27–31.

Ray, W. A., Griffin, M. R. & Downey, W. (1989) Benzodiazepines of long and short elimination half-life and the risk of hip fracture. *Journal of the American Medical Association*, **262**, 3303–3307.

Taylor, S., McCracken, C. F. M., Wilson, K. C. M., et al (1998) Extent and appropriateness of benzodiazepine use. Results from an elderly urban community. *British Journal of Psychiatry*, **173**, 433–438.

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Early psychosis

Sir: The June 1998 supplement of the *British Journal of Psychiatry* was devoted to a discussion of early psychosis. Patrick McGorry, the Guest Editor, introduced the subject with an extract from a 1938 article by D. Ewen Cameron. McGorry credited Cameron with originally foreshadowing this form of preventive intervention and he was apparently using Cameron as a source of authority. It is uncertain whether McGorry is aware of Cameron's unsavoury reputation as a CIA-funded unethical experimenter. Cameron attempted to erase his patients' self-identities using electroconvulsive therapy and deep sleep. McGorry's judgement in openly citing Cameron as a source of authority is unsound.

Perhaps McGorry has not read the Discussion which follows Cameron's 1938 article. Harry Stack Sullivan's adverse comments about Cameron's thinking and the general idea of pre-psychotic detection and intervention do not accord with McGorry's beliefs. Sullivan (1938) wrote:

"I would be very deeply disturbed if, as is implied by the last speaker [Cameron], people who show signs of personality disorders, early mental disorder of an indeterminate kind, were to be rushed through treatment with insulin, metrazol and camphor on the chance that they might otherwise have developed schizophrenia. I privately have a suspicion that might have a distinctly unfavourable effect on the general intelligence level and so on of the community. What does it mean that a person will have schizophrenia which can be detected by the intelligent layman months to years before the schizophrenia appears? In seven and half years of exclusive preoccupation with the schizophrenia problem I was unable to put my finger on anything sufficiently simple and obvious to service this purpose".

Was Sullivan right to nip this scheme in the bud in the late 1930s? Cameron proved to have many very bad ideas yet McGorry draws on Cameron as a source of authority. Should we allow McGorry to persuade us that among all of Cameron's bad ideas, this one is an exception?

McGorry, P. (1998) Preventive strategies in early psychosis: verging on reality. *British Journal of Psychiatry*, **172** (suppl. 33), 1–2.

Sullivan H. S. (1938) 'Discussion' in D. Ewen Cameron. 'Early schizophrenia'. *American Journal of Psychiatry*, **95**, 579.

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Author's reply: Gosden takes me to task for quoting Ewen Cameron in the *British Journal of Psychiatry* Supplement 33 on early psychosis, because of his subsequently highly problematic career. I was made aware for the first time by Canadian colleagues, after the acceptance of the Supplement for publication, of Cameron's dubious later activities, but originally quoted him on the merits of his landmark paper from the *American Journal of Psychiatry* in 1938. Although he was not the first to emphasise the importance of early intervention in schizophrenia and related psychoses (in fact, Sullivan had done so a decade earlier), his paper highlighted how little was known about the appropriate ways and means of intervening at such an early stage. This challenging yet cautionary note, stressing the level of ignorance which existed and continued for a prolonged period afterwards, is actually the point of the quote I selected. Ironically, it is quite at odds with the interpretation adduced by Gosden, namely a feared headlong rush to apply dubious treatments to arguably healthy individuals. Furthermore, the remainder of the introduction and the Supplement as a whole clearly reflects a belief in the need to collect and weigh the evidence in relation to all aspects of early psychosis.

In the discussion which accompanied the paper, which I had certainly read and from which I have also quoted in scientific forums, Sullivan, while not eschewing his support for a preventive approach, quite appropriately argued against the widespread application of treatments which were unproven and carried the potential for harm. He stressed the need for very accurate prediction of incipient psychosis, if specific treatment were to be considered prior to onset of psychosis. Such caution is appropriate today, particularly in relation to those at increased risk of psychosis, but who have not yet met diagnostic thresholds, and we have been active in research aimed at improving our predictive capacities (Yung & McGorry, 1997). In such potentially prodromal cases, desire for treatment, level of distress and disability, risk of harm and risk of early transition to psychosis are factors influencing the decision to offer treatment when it is sought. It is clearly premature to offer antipsychotic medications in a widespread fashion to such potentially pre-psychotic patients until a series of well-conducted randomised controlled trials from several

sites have examined the issue. Many cases will prove to be 'false positives'. Other people, even if truly vulnerable to a psychotic disorder, may respond to purely psychosocial interventions or require other forms of drug therapy, for example antidepressants. In others, it may turn out that neuroleptics help to reduce the risk of a first and potentially destructive episode of psychosis. We simply do not yet have sufficient knowledge to be clear about specific treatment-matching at this phase, so further research is urgently required. On the other hand, psychiatrists still need to respond to those in distress and people with substantial levels of disability and risk. For example, in our Personal Assessment and Crisis Evaluation (PACE) clinic which provides assistance for at-risk patients who are actively seeking and accepting help, the rate of deliberate self-harm and disability prior to entry is high (Yung *et al.*, 1996). Better access to interventions for adolescents and young adults with psychosocial disorders is an urgent priority (Rutter & Smith, 1995); specificity of treatment is essentially a second-order issue.

In first-episode psychosis, the evidence suggests the real problem is excessive caution, with delayed treatment all too common. While it remains to be definitively proven that shortening delays in treatment strongly affects the long-term outcome, the increasing safety and effectiveness of modern treatments support a policy of early intervention once a psychotic illness is clearly present. Obviously, such a proactive stance must respect the rights and wishes of consumers; indeed, in doing so, the chances of a collaborative long-term relationship, the cornerstone of a positive outcome in psychosis, are usually enhanced.

I do not believe that Sullivan either sought to or succeeded in 'nipping this scheme in the bud'. The notion of early intervention is a sound one throughout medicine, provided the maxim *primum non nocere* is adhered to. Cameron merely argued for this principle to be applied to the most severe disorders that psychiatrists treat. Perhaps the time was not right in the sense that few effective or specific treatments existed then, but the idea was a not a bad one. It is important to separate people from ideas.

Rutter, M. & Smith, D. J. (eds) (1995) *Psychosocial Disorders in Young People: Time Trends and their Causes*. Chichester: John Wiley and Sons.

Yung A. R. & McGorry, P. D. (1997) Is pre-psychotic intervention realistic in schizophrenia and related

disorders? *Australian and New Zealand Journal of Psychiatry*, **31**, 799–805.

—, —, **McFarlane, C. A., et al (1996)** Monitoring and care of young people at incipient risk of psychosis. *Schizophrenia Bulletin*, **22**, 283–303.

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Emotional functions and cognitive processes

Sir: Taylor & Liberzon (1999) provide a valuable overview of current knowledge of the neurobiology of emotion, and its implications for the study of schizophrenia. One important aspect which was not fully explored is the possible link between emotional function and the capacity for abstract thought, both of which may be abnormal in schizophrenia.

Lesion studies in monkeys indicate the importance of the orbitofrontal cortex in mediating the inhibition of action when this inhibition is required for reward. Damasio (1996) has shown that in man this area is not only essential in inhibiting automatic responses, but also in recognising bodily states associated with reward and punishment. This recognition is necessary to allow the abstraction of rules about which categories of responses are likely to be rewarded. The study quoted used a gambling paradigm. Subjects selected cards at will from two packs; one pack provided rewards of large sums of money, but an overall loss in the course of the experiment, while the second pack produced more modest wins but a consistent gain over the experiment. Subjects with orbitofrontal lesions consistently selected cards from the first pack, whereas those with normal frontal lobe function shifted strategy to select cards from the second pack. Subjects without orbitofrontal lesions were able to 'sense' that one pack was 'better' than the other although they were unable to say why this was.

The suggestion is that in order to be able to shift attention from the immediate consequences of a response (in terms of reward or punishment) to being able to think symbolically about categories of response, systems involved in reward, punishment and the unconscious and conscious appraisal of emotion are required, as well as systems involved in sustained attention, memory and information-processing. Social

interaction requires the ability to abstract rules of behaviour, while the ability to form mental representations of categories of rewarding and aversive stimuli may represent evolutionarily early stages of abstract thought.

These possible links between emotional functions and cognitive processes are of considerable interest in the study of the disease process of schizophrenia, which involves affective blunting, concrete thought processes, impaired function on tests of cognitive set-shifting and abnormal categorical thinking.

Damasio, A. R. (1996) The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society of London, Series B: Biological Sciences*, **351**, 1413–1420.

Taylor, S. F. & Liberzon, I. (1999) Paying attention to emotion in schizophrenia. *British Journal of Psychiatry*, **174**, 6–8.

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Authors' reply: We are grateful to Barbel for pointing out an important and complementary approach to the study of emotion and cognition. Whereas we sought in our editorial to emphasise the distinction between cognitive and emotional themes in schizophrenic pathology (Taylor & Liberzon, 1999), it is equally interesting to understand how emotional responses also have cognitive functions. Barbel makes the intriguing suggestion that some emotional responses might demonstrate "early stages of abstract thought". As patterned responses, presumably selected by evolutionary pressures, emotions should reflect the output of specific neural systems which adapted to handle recurring situations. As such, specific emotions occur in specific environmental contexts, which clearly delineate categories of behaviour, or rules.

We agree with Barbel's observation that a disease process affecting cognitive functions, such as set-shifting, and emotional functions, such as affective range, points to both cognition and emotion at the root of pathophysiology. In general, we reject the use of rigid, dichotomous formulations of emotion *v.* cognition, both on theoretical and on empirical grounds. Students of emotion recognise that in spite of their heuristic value as psychological categories, emotion and cognition do not constitute mutually exclusive functional