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Correspondence

Editor: Ian Pullen

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Interpretation of historical evidence

SIR: May I reply to the letter of Persaud & Allderidge (*Journal*, May 1989, **154**, 719–720), criticising aspects of my paper on 'Schizophrenia as a recent disease' (*Journal*, October 1988, **153**, 521–531)?

Concerning Andrew Harper, it seems trivial to belittle his work on the ground that he had been an army surgeon. One might with better reason belittle Pinel on the ground that he had been a teacher of mathematics. Dr Persaud and Miss Allderidge have overlooked the fact that Harper (1789, pp vii, 9) defined insanity in terms of what we would now call the functional psychoses, and that he specifically excluded mental disorders due to bodily disturbances or toxins (Leigh (1961, p. 68), in criticising Harper's book, seems also to have overlooked this fact). If organic psychoses are excluded, then Harper's statement of insanity as being very rare in young people may properly suggest that adolescent schizophrenia was rare or absent in his time.

Harper's book was quoted with approval by Pinel (1801, p. 111 of the translation), and the adverse comments made in a footnote by his translator are dismissed by Cranefield (1962). Haslam's criticism of Harper (Leigh, 1961, p. 121) is specious, and perhaps reflects no more than his general quirkiness and his annoyance at Harper's assertion of the "ignorance and absurdity" of immediately confining victims of insanity to "the cells of Bedlam". Although limited in scope, Harper's book is straightforward and sensible and gives a good description (p. 68) of the intellectual deterioration associated with chronic mania, a condition much discussed during the 19th century and whose relation to schizophrenia has yet to be determined.

I can see no reason whatever to think that Haslam's evidence is 'more reliable' than Harper's. We know little of Harper's life, but we know enough of Haslam's (from the 1815 Select Committee) to know that his probity was not above suspicion. In any case, the statistics for admissions to Bethlem during 1784–1794 were not personally collected by Haslam, since he did not take up his appointment there until 1795; they were collected by his predecessor, John Gozna (Leigh, 1961, p. 103). At Bethlem, 'insanity' was used in its wider sense and included organic as well as functional psychoses (see Haslam's definition, 1798, p. 10). In citing the Bethlem statistics as evidence against the recency hypothesis, Dr Persaud and Miss Allderidge have overlooked this wider usage of insanity, for they seem to suppose that all young people admitted as 'insane' would have suffered from the 'adolescent insanity' described in the latter part of the 19th century. But it is much more likely that many, and perhaps most, of the young Bethlem patients suffered from organic psychoses, because the causes of organic psychoses – fever with delirium, encephalitis, head injury, vitamin deficiences, toxins, the effects of malnutrition, etc. – would have been far commoner then than now. Haslam (1809, p. 209) gives a list of such causes, and devotes a chapter (Ch. IV) to cases of "insane children", where he describes patients aged 3, 7, and 10 who evidently suffered from organic mental disorder.

Unlike Dr Persaud and Miss Allderidge, Wilkins (1987) was careful to point out that Bethlem admissions would not have been representative of the country as a whole. It is a pity that Wilkins confined his study to teenagers, for during the 19th century adolescence was generally taken as continuing up to the age of 25 (Clouston, 1892, p. 361). Thus Clouston (p. 362) says that developmental insanities occur most frequently during adolescence "from twenty-one to twenty-five". But so far from contradicting the recency hypothesis, Wilkins' study perhaps lends it some support. He found a significant increase with time in the proportion of patients having visual and auditory hallucinations; and whereas visual hallucinations are most commonly the consequence of fever, drugs, or toxins, the commonest cause of auditory hallucinations in young persons is schizophrenia.

Dr Persaud and Miss Allderidge are incorrect in saying that I argued that descriptions of recognisably schizophrenic symptoms are difficult to find at the

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beginning of the 19th century. What I said was that, on the recency hypothesis, "descriptions corresponding to adolescent insanity [in Clouston's sense of the term] will hardly be found in pre-1800 records"; and I said the earliest probable description I had found was in Cox's book of 1806 (the description is also given in his first edition of 1804). Their quotations from Haslam's books of 1809 and 1810 are therefore irrelevant as arguments against the recency hypothesis. Moreover, they fail to notice that I drew attention in my paper to the differences between Haslam's *Observations* of 1798 and his second edition of 1809. The remarkable passage in the second edition about "a form of insanity in young persons" does not occur in the first edition – that was my point. It is also noteworthy that Haslam says little about auditory hallucinations (and nothing about "voices") in his first edition (p. 5), but much more about them in his second edition (p. 68). In the second edition, but not in the first, he refers to 'the alarming increase in insanity'; and as late as 1843 he was still concerned with this increase, which he believed to be a real one (Leigh, 1961, p. 134).

Haslam's book of 1810 is entitled 'Illustrations of Madness, exhibiting a *singular* case of Insanity . . .' (my italics). Precise in his use of words and a student of etymology (Leigh, 1961, p. 116), Haslam would have chosen the word 'singular' with care. Its meaning was the same in his day (Johnson, 1807) as it is now – unique, extraordinary, unexampled, peculiar. Why then, we may wonder, should Dr Persaud and Miss Allderidge consider this case as an instance of what was 'so common, obvious and typical' as to be 'hardly worth mentioning'?

I am sure that many cogent arguments may be urged against the recency hypothesis, but I find little or nothing of substance in the criticisms of Dr Persaud and Miss Allderidge. And that leads me to think that the remarks in their final paragraph are out of place.

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Assessment of outcome

SIR: Tarrier *et al* (*Journal*, May 1989, **154**, 625–628) report a substantial rate of "relapse" of schizophrenia after 24 months of aftercare that included 9 months of a specific psychological intervention as an adjunct to drug therapy. They comment that several other recent studies of psychosocial and drug combinations have shown an increase in psychopathology once the psychosocial intervention has been completed, and advocate the continuation of such treatment for a longer period. They fail to note that the one study that did follow this valuable advice not only showed continued low levels of schizophrenic and affective psychopathology, but also showed a substantially higher recovery rate, in terms of both the clinical condition and social disability (Falloon, 1985).

The preoccupation of schizophrenia researchers with preventing "relapse", a state poorly defined in most studies (Falloon *et al*, 1983), has led to a neglect of measurements that reflect the quality of life of the patient and his caregivers. I suggest that the aim of long-term management of any chronic disorder characterised by exacerbations and remissions should focus on maximising functioning and minimising handicaps, as well as controlling symptoms. A longitudinal approach that targets the continuing and changing needs of patients and their support systems seems essential. Continued targeting of psychosocial interventions as well as drug therapies may yet lead to an enhanced rate of clinical and social recovery from schizophrenia. The advocates of such a strategy are urged to conduct further studies to assess the long-term benefits of continuing optimal multimodel therapies.

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