

order, with some resemblances to folie à deux . . ." which is created by clinicians, the media, and the health care system through "selective reinforcement of symptoms". Simpson is but one of a number of clinicians recently who have professed to have seen either none or at the most one patient with MPD, yet find themselves qualified to comment on the diagnosis, aetiology, and treatment of MPD (Levitt, 1988; Fahy, 1988). What has become of the scientific method? Conclusions about such matters should only be made after thoroughly reviewing the literature or after accumulating a robust series of cases.

The history of MPD dates back to antiquity (Ross, 1989). Since 1860 over 700 scientific articles, chapters, books, etc. have appeared on MPD (Coons, 1986–89). Half of this literature has appeared since 1980, when MPD was first recognised as a diagnosis in the DSM-III.

Although there are a few 'media multiples' who derive much secondary gain from their illness as Simpson suggests, this pattern is not common. Most patients with MPD are deeply troubled by their illness and therapy can be very arduous. I've found few clinicians who make a spectacle out of demonstrating their "talented offspring" and showing their "latest cute trick".

Professor Simpson is incorrect when he states that "a very small number of clinicians report the great majority of case reports". The 211 case reports in the English literature have been reported by 180 different clinicians. The International Society for the Study of Multiple Personality and Dissociation has a membership of over 1300 clinicians, predominately in the United States and Canada, and most have seen patients with MPD. I have personally corresponded with numerous clinicians from throughout North America, several Latin American nations, numerous European countries, and Australia who have encountered MPD. Analogues of MPD exist in Africa and Asia. The few clinicians who have reported large series of cases have amassed these over 10–15 years and have large referral areas encompassing several states or provinces.

A recent issue of *Dissociation* (Kluft, 1989) was devoted to the iatrogenic aspects of the diagnosis and treatment of MPD. It is clear that dissociative symptomatology may be worsened by improper forms of treatment. In occasional instances new personalities may form over the course of treatment secondary to new traumatisation. At this time, however, the most common iatrogenic problem is not making a diagnosis of MPD and instituting proper treatment.

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SIR: Simpson (*Journal*, October 1989, **155**, 565) has stated that multiple personality disorder is an iatrogenic artifact, and that it does not occur as a natural condition. Neither he nor Fahy (*Journal*, November 1988, **153**, 597–606), upon whom Simpson comments, refer to the substantial evidence accumulated in North America that MPD is a relatively common post-traumatic disorder of dissociative type, linked to severe chronic child abuse.

Rather than disagreeing with Professor Simpson on an ideological level, I would like to point out that these two competing hypotheses about MPD could be tested using the Dissociative Experiences Scale (Bernstein & Putnam, 1986) and the Dissociative Disorders Interview Schedule (Ross, 1989; Ross *et al.*, 1989).

If all individuals admitted to an acute care adult psychiatric in-patient unit in Britain or South Africa were screened with the Dissociative Experiences Scale, I predict that about 15% would score above 30. Of these individuals, one-third would meet DSM-III-R criteria for MPD on the Dissociative Disorders Interview Schedule, and one-third would have another dissociative disorder.

Review of the past histories of these individuals with MPD would reveal that they had been in the mental health system for an average of between five and ten years. Only part of their complex symptom clusters would have been documented during this period. The dissociative disorders would not have been diagnosed because of failure to enquire systematically about dissociative symptoms.

In some countries the prevalence of dissociative disorders would vary from that in North America, perhaps due to chronic childhood trauma in the form of war, famine, and natural disaster, or a different prevalence of child abuse. Although an actual calculation would be difficult to make, conceptually the correction factor for the prevalence of dissociative disorders in other parts of the world compared with that in North America would be the ratio of trauma in those countries compared with that in North America. MPD would occur in response to chronic

severe trauma only within a window of vulnerability ending in early adolescence.

If anyone in Britain, South Africa, or any other country outside North America would like to conduct such a study, I can be contacted at the address below. Once such studies had been conducted, we could then begin a scientific discussion.

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Life events and management in schizophrenia

SIR: We have previously reported (TARRIER *et al*, *Journal*, October 1988, 153, 532–542) on the success of a behavioural intervention with families to reduce relapse rates in schizophrenic patients living with high expressed emotion (EE) families compared with a short educational intervention and routine treatment. We also found that in families receiving the family intervention there were significantly greater changes in the relatives' ratings of EE from high EE to low EE over the nine month follow-up. Hence there appears to be an association between relapse rates and change in the relatives' EE ratings. It could be hypothesised that reduction in the relatives' EE resulted in reduced relapses, although these data do not provide unequivocal support for this hypothesis. We were interested in examining alternative hypotheses for the different relapse rates. It could be possible that the family intervention resulted in the patient having greater contact with the psychiatric services in general, or receiving higher doses of medication or showing greater medication compliance. However, we could find no evidence to support these alternative hypotheses.

A further possibility that we did not examine at the time, although the data were collected, is that patients in the high-EE education and routine treatment groups experienced more independent life

events than patients in the family intervention groups, the occurrence of major life events being associated with relapse. Data on life events over the nine month follow-up period was collected on 77 patients. Of these, 50 (65%) did not experience a life event. Three patients (16%) from the low-EE groups, six (20%) from the high-EE education and routine treatment groups, and 17 (61%) from the behavioural intervention group experienced at least one life event. A Kruskal-Wallis one-way ANOVA demonstrated that this difference was highly significant ($\chi^2 = 19.02$, $P < 0.001$) due to the very high frequency of life events in the behavioural intervention group. The hypothesis that a higher frequency of life events would be associated with the higher relapse rates in the high-EE education and routine treatment group was not confirmed. In fact, patients in the behavioural intervention group experienced more frequent life events and showed a decreased frequency of relapses. Similar results have been reported by Falloon and his colleagues in their intervention study (Hardestry *et al*, 1985).

These results suggest that family interventions designed to improve the family members' ability to cope with stress are successful in reducing the negative effects of major life events. This is evidenced by one patient in the family intervention group who had a 25-year history of schizophrenia involving 13 hospital admissions. She experienced seven independent life events over the 9 month post-discharge period, but remained symptom free over this period and was still well at two-year follow-up.

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Pimozide in pathological jealousy

SIR: It was with considerable dismay that I read Cohen's scathing attack (*Journal*, November 1988, 155, 714) upon our brief report (*Journal*, August 1988, 155, 249–251). Our suggestion that pimozide