

Violence and sudden death

SIR: I read with great interest the paper on "Suicide and unexpected deaths among psychiatric in-patients" by Morgan & Priest (*Journal*, March 1991, 158, 368–375). They have raised some very important issues.

Drs Morgan & Priest indicated that communication difficulties, low levels of available staff, or absence on leave may have been important in some cases of their series of suicides. Our work on violence (including self-harm) has already demonstrated this quantitatively (e.g. James *et al*, 1990). Of particular interest is the positive relationship between levels of agency (temporary) nursing staff and violence, and a negative relationship between levels of permanent nursing staff and violence. Other studies of in-patient suicides have indicated authoritarian attitudes and under-involvement of medical staff (Langley & Bayatti, 1984) and increased vulnerability among nursing staff in conflict (Morgan & Priest, 1984) as contributory factors. All these studies indicate that adequate provision of well trained staff with an appropriate hierarchical support structure is vital, particularly in the modern era of community psychiatry, cost cutting and audit.

In a further study of violence on a high-dependency mental handicap ward (Shah & Piachaud, 1988), 620 violent incidents over a 21-month period were identified. Four patients accounted for 74% of this violence, and two of these four most violent patients suffered sudden unexpected deaths during the study period. Both these patients were in their early twenties and physically well. In one case the cause of death was spontaneous jejunal rupture with peritonitis and in the other case, dissecting aneurysm of the thoracic aorta. Both these causes of death are rare even in the surgical literature. Shortly after completion of the study, a third violent patient died suddenly from acute pneumonia. Although the numbers were small for statistical analysis, this apparent relationship between violent behaviour and sudden death is of interest. It could be argued that these sudden deaths could be avoided if strategies to reduce violent behaviour were available. This is another area that needs systematic exploration and I would be interested to hear of such cases.

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Treatment-resistant depression

SIR: We noted the letter by Malizia (*Journal*, July 1990, 157, 145). Regarding the Tyrer & Murphy article also published in the *Journal* (January 1990, 156, 115–118) on a case of treatment-resistant depression, we are in agreement with Dr Malizia. However, we believe that the following issues deserve further comment. Treatment resistance must be clearly defined. Adequate duration of treatment, dose, and blood levels must be established. The differential efficacy within the same individual of one antidepressant over another must be determined.

The issues of diagnostic criteria, comorbidity and personality disorders are increasingly complex. The pharmacological factors may be simplest to define. It would seem appropriate, then, to work towards the establishment of a common system, regarding duration of treatment, dose, and blood levels, lest the important and painstaking work of investigators such as Drs Tyrer & Murphy and others be marred by vague concepts of treatment resistance.

Ayd (1983) highlighted the problem of treatment resistant depression, noting the recommendations of the World Psychiatric Association. He discusses the distinction of absolute and relative treatment resistance, the former being defined as failure to respond to 150 mg per day of imipramine, or its equivalent, for four to six weeks, and the latter as failure to respond to an inadequate course of treatment. This clarification fails to consider well established findings of the 'relative' efficacy of the 150 mg per day dose of imipramine. Simpson *et al* (1976) report that 300 mg of imipramine is clearly superior to 150 mg per day. The same effect has been demonstrated for other antidepressants.

It is imperative that we arrive at a consensus opinion regarding criteria to define the adequacy of a treatment trial. It has been relatively well established, at least for imipramine and nortriptyline, that blood levels may be used to guide therapy. In cases where blood levels are not available, it is increasingly advisable to use imipramine at 300 mg per day, or

its equivalent, before declaring the patient a non-responder.

The important review of Quitkin *et al* (1984) addresses the question regarding the length of treatment and its relation to treatment resistance. He suggests that there is a paucity of treatment trials extending beyond four weeks, although some literature suggests that treatment extended to six weeks may improve response, particularly in those patients without melancholia. He strongly argues for the extension of treatment trials to at least six weeks. It has not been established whether one tricyclic is more effective than another. If a patient cannot tolerate an 'adequate' dose of a tertiary amine, a trial of a secondary amine may be indicated. A monoamine oxidase inhibitor (MAOI) may be indicated subsequently, if one has not already been employed at a dose corresponding to 90 mg of phenelzine or 60 mg of tranylcypromine. Alternative therapy such as fluoxetine, psychostimulants, lithium or thyroid augmentation, as well as combination regimens, may be considered when first line treatments have failed.

Until the problem of classification is resolved, we suggest that investigators report diagnostic criteria applied and the parameters of pharmacological treatment employed, including duration of treatment, dose of drugs, concomitant medications, and blood level data when available. Perhaps a better way of defining treatment resistance might be lack of response to 300 mg of a tertiary amine or a secondary amine such as nortriptyline at a blood level of 100 ng/ml, 90 mg of phenelzine, or 60 mg of tranylcypromine with platelet MAOI inhibition of 80% for a minimum of six weeks. These criteria may help to define treatment resistance. While this may not always be data based, it would allow for a certain amount of standardisation across sites.

Finally, it is well established that comorbidity on both axes I and II effect outcome. A new and more inclusive taxonomy should be multidimensional and allow for the characterisation of depressive subtypes, comorbid anxiety and psychotic disorders, character disorders and patterns of substance use, as well as definitive pharmacological parameters to guide treatment.

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QUITKIN, E., RABKIN, J., ROSS, D., *et al* (1984) Duration of antidepressant drug treatment. What is an adequate trial? *Archives of General Psychiatry*, 41, 238–245.

SIMPSON, G., LEE, J., CUCULIC, Z., *et al* (1976) Two dosages of imipramine in hospitalized endogenous and neurotic depressives. *Archives of General Psychiatry*, 33, 1093–1102.

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Psychiatry and post-traumatic stress disorder

SIR: Kohen's letter on the "Psychological sequelae of torture" (*Journal*, February 1991, 158, 287) and her reference to Turner & Gorst-Unsworth (*Journal*, October 1990, 157, 475–480) underlines the reluctance of the body of British (and Australian) psychiatrists to grasp the nettle of the significance to psychiatry of the concepts embodied in the diagnosis of post-traumatic stress disorder (PTSD). Her concise but dramatic picture of the consequences of the widespread use of torture in Turkey – without mention of PTSD – illustrates the wide-ranging pathology in both the persecuted and the persecutors, and intergenerational effects. If one extends this view of torture to terrorism and violence generally, the social, as well as psychiatric, consequences of the sort of events associated with a PTSD become monstrous (Borges Watson, 1990).

The scanty involvement of British psychiatrists at the Second European Conference on Traumatic Stress held at Leeuwenhorst in The Netherlands in September 1990, compared with the large number of psychologists, is a cause for concern. Perhaps after side-stepping the psychological consequences of trauma for nearly 100 years, psychiatry finds it difficult to reassess its position. There is abundant evidence that violence breeds violence and, as Bowlby (1984) said "It has been extremely unfashionable to attribute psychopathology to real life experience". Perhaps even more telling is Guntrip's (1971) comment "An aggressive society becomes self-perpetuating, a nearly insoluble problem".

Dr Kohen is not alone in seeing a connection between unbridled violence, terror and human degradation and a wide variety of presenting psychopathological states (Veterans' Administration, 1985; Ulman & Brothers, 1988), although the relationship and the extent of the relationship remains to be determined. Is psychiatry going to leave the responsibility of investigating this primarily in the hands of psychologists?

BOWLBY, J. (1984) Violence in the family as a disorder of the attachment and caregiving systems. *American Journal of Psychoanalysis*, 44, 9–27.