Patients with mild heart disease (for example, a history of myocardial infarction or mild angina) can probably be treated with any antidepressant. There is no clear evidence that some drugs are safer than others and, though mianserin and trazodone may prove to be such safer drugs, further studies are needed in patients with heart disease. In the light of recent reports it would seem wise to be very careful about the use of tricyclic antidepressants in patients with severe heart disease. In this category I would place patients suffering from heart failure, patients with bundle branch block, or those with heart block in their electrocardiograms, and patients who have recently had a myocardial infarction.

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Violence and mental illness

The case of McNaughten in 1843 focused public and legal attention on the relation between psychosis and homicide, but the issues are still hotly debated. Several series have shown that serious crime may be an early indication of schizophrenia.[1-3] Hallucinations, delusions, and conceptual disorganisation create obvious hazards, and psychotic manifestations of this kind—particularly when associated with thought disorder—have been shown to predict violence on an acute admission ward.[4]

Psychopathy may be considered as part of what Inouye was the first to call the schizophrenia spectrum of disorders[5]—a concept supported by their having some physiological similarities.[6] Schizophrenia and alcoholism also overlap genetically: follow up studies have shown that pathologically aggressive boys are likely to abuse alcohol as young men,[7] and schizophrenic patients are more likely than other groups to abuse illicit drugs.[8] How ironic, therefore, that Taylor and Gunn (p 9) should have found that drug abuse and alcholism are great impediments to the transfer of prisoners on remand to hospital.

What sort of people are prone to violence? Some support has been found for Megaragee's view that they are distributed biomodally[9 10]; at one end of the curve are the over controlled, highly provoked individuals who eventually react with homicide rather than abuse or lesser violence, while at the other end—more commonly—are the under controlled, explosive psychopaths. The temperamental component in some violent behaviour may be recognised early in childhood.[11] Recurrent problems with feeding, bathing, and dressing, together with loud crying, protest at novelty, and tantrums are all more common in children who are later found to have "conduct disorders,"[12] and such a history is general in adult criminals.[13] Further evidence of a temperamental factor has come from a series of identical twins separated in early life who independently became criminal, sometimes in curiously similar ways.

Neurological lesions do not seem to be a prime cause of behavioural problems, but they may sensitise children to adverse environmental influences.[14] Both mental handicap and epilepsy are associated with violence.[15] Neurophysiological mechanisms may be disordered in explosive violence. The electroencephalogram often shows non-specific slow wave abnormalities, and similar features are said to be present in psychosis,[16] sometimes detectable only by spectral and coherence analysis.[17]

A child who has serious difficulties with his parents will necessarily be disturbed, and the father's violence towards the mother was found to be the most important contributing factor in a study of 21 homicidally aggressive children.[15] Psychodynamic formulations relate aggression to depression,[18] and, indeed, aggression sometimes precedes depression,[19] while suicidal behaviour has been associated with outwardly directed violence in children.[15] Suicide is most common in late life, while violence is greatest in the prime, but nevertheless a relation has been found between aggression, suicidal behaviour, and 5-hydroxytryptamine metabolites in the cerebrospinal fluid of people with personality[20] and borderline personality disorders.[21] Lithium,[22] which, among its several actions increases release of 5-hydroxytryptamine,[23] together with its precursors,[24] has been advocated as treatment for violence (as have antiandrogens[25] and amygdalectomy[26]).

The findings of Taylor and Gunn (p 9) reinforce the linkage

of violence with mental illness. Their analysis of the records of 1241 men remanded in Brixton prison showed that in the group charged with homicide just over a third were psychiatrically abnormal; five (11%) of those convicted were schizophrenic. Despite the risk of undercounting this was a disproportionately large group in relation to expectations from epidemiological data, and the number of schizophrenic patients who had been violent towards others without resulting fatality exceeded expectations by over 22 times.

In 1939 Lionel Penrose showed a negative correlation between the prison population, the homicide rate, and the availability of mental hospital beds. Since 1954 the number of patients in mental hospitals in England and Wales has declined by 67 000, but only 3010 expatients are supported by local authorities.[27] These figures suggest one explanation for the discrepancy between the current findings of Taylor and Gunn and those of several previous studies,[28–30] all of which failed to find any appreciable excess of schizophrenic patients prosecuted for violent crimes. As a society we have been failing in our care of schizophrenics. Their difficulties in obtaining psychiatric treatment,[27 31] their high suicide rates after discharge from hospital,[32] and the prevalence of serious mental illness among the destitute and in prison,[33 34] are further indictments of "community care" policies.[35–37] The links between violence in our society and the neglect of mental illness by successive governments need wide publicity —and the government should not be allowed to remain silent on this issue.

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Rifampicin in non-tuberculous infections

A recent review of the use of rifampicin in non-tuberculous infection in North America raises the question of current practice in Britain.[1] Should rifampicin be used more widely? Or should it be reserved for a few specific indications?

Rifampicin was synthesised in 1965 and rapidly recognised as having bacterial activity against *Mycobacterium tuberculosis*. It also has a broad antimicrobial spectrum against Gram positive and Gram negative organisms and can penetrate most tissues because it is lipid soluble. Despite these attributes, however, it has been little used in non-tuberculous infections owing to fears of the development of resistance by *M tuberculosis* and other bacteria. Rifampicin is also relatively expensive—though considerably cheaper than many of the more recent cephalosporins—and potentially toxic.

One problem encountered early in clinical experience was the rapid development of bacterial resistance when rifampicin was used alone. Rifampicin's antibacterial activity depends on its ability to bind and inactivate bacterial DNA dependent RNA polymerase. Resistant strains occur during monotherapy by a modification altering the binding of rifampicin to this target enzyme. This mechanism of resistance is specific to rifampicin.

Rifampicin is said to be the most potent antistaphylococcal agent available, but it must be used with another agent to avoid resistance.[2] One report described synergy between rifampicin and oxacillin, which was influenced by both the concentration of the antibiotics and the bacterial inoculum used in laboratory studies—but these did not necessarily predict the clinical outcome.[3] In clinical practice rifampicin is a valuable reserve drug for use in refractory *Staphylococcus aureus* infections, including endocarditis, and is effective for staphylococcal infections in patients with chronic granulomatous disease because of its effective penetration of neutrophils.[4] Rifampicin is usually active against *Staph aureus* resistant to methicillin and might be considered for use in individual patients infected with a resistant strain, but experience shows that its use on a large scale would inevitably be associated with the appearance of rifampicin resistant *Staph aureus*.[5] *Staph epidermidis* has a less predictable sensitivity pattern than *Staph aureus*: it is often multiresistant, though usually sensitive to rifampicin. Rifampicin has been recommended as an initial choice of treatment for endocarditis due to *Staph epidermidis*,[6] and also in infections of cerebrospinal fluid shunts due to this organism.[7]

The intracellular penetration of rifampicin suggests that it should be effective in infections caused by typically intracellular organisms such as *Chlamydia* spp and *Brucella* spp. Rifampicin is the most active agent available against *C trachomatis* but may rapidly develop resistance in vitro.[8] Rifampicin and chlortetracycline ointments were found to be equally effective in sexually transmitted *C trachomatis* infection of the eye,[9] and rifampicin has also been reported effective in endocarditis due to *C psittaci*.[10] Because of the combination of the rapid development of resistance and the availability of other effective agents such as tetracyclines or erythromycin rifampicin should not be used routinely in chlamydial infection, but it is valuable in deep seated infection. Rifampicin has also been used in combination with erythromycin to treat serious infections due to