Management of perinatal loss of a twin

We are all indebted to Dr Elizabeth M Bryan for her contributions to our knowledge of the social and psychological effects of twinning and to Dr Manny Lewis for his studies of the need for mourning in relation to stillbirth—their latest joint paper (19 November, p 1327) being no exception. I wonder, however, whether they have sufficiently emphasised the conflict in a mother between mourning for the dead baby and taking pleasure in the living one. Although it is probably true that the loss of one of a pair of twins. Either she postpones her mourning, repressing the feelings that go with it, or she indulges her grief at the expense of the survivor, depression being incompatible with the lively communication, based on primary maternal preoccupation, that Murray has shown to be so important for a baby's emotional and intellectual development.

Many of your readers will have personal or professional experience of the problem that this presents to families; and it is difficult to know how to cope with what on the face of it is an insoluble dilemma. Perhaps the best answer would be for the mother to be helped to get over her mourning before taking up the care of the second baby, bonding in our species being almost certainly a postponable if necessary process but susceptible to permanent wrecking.

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Sexual drive of patients in psychiatric hospitals

An anonymous correspondent (20-27 August, p 1120) and Dr J C C Chase (29 October, p 1129) expressed concern about sexual activity in psychiatric hospitals, including rape. Rape may not be uncommon in such settings, and we are aware of three cases in units in which we have recently worked, one of a live one in the case of the death of feeling, it is only one example of the violence that is commonplace in psychiatric institutions. The amount of violence is thought to be rising, and the Department of Health has recently issued further guidelines.1 There is, however, no consensus on whether or in what circumstances patients should be prosecuted for acts of violence committed in psychiatric hospitals.

Many among psychiatrists is divided on the question of prosecution. Some believe that those admitted to psychiatric wards enjoy a state of asylum which should, in most circumstances, extend to immunity from prosecution. Though few would dissent when acts of violence arise out of psychotic experience, many would contend that those who do not fall into this category should be held responsible for their actions and subject to the due process of law. Health care workers, particularly nurses, are increasingly reluctant to accept physical assault as an occupational hazard, and several cases have recently come to our attention of staff pressing charges against patients for violent attacks. These actions were initiated without the help or authority of the hospital authorities and were met with hesitance from the police, who may be used to viewing psychiatric admission as an alternative to prosecution.

Though it remains uncommon for patients to be charged after assault on staff, the prosecution of patients for assault on other patients is, we suspect, less common still. We wonder which factors underlie whether the option of pressing charges is adopted or even considered. The reactions to the rape cases that we encountered bear examination in this respect. One case, the rape of a woman in her 70s by a young schizophrenic, resulted in a decision to press charges. The patient’s age, the feelings of her relatives, and the brutality of the attack may have influenced this choice of action. In the two remaining cases young female schizophrenics who were not acutely ill were raped by an outpatient and by a patient from an open ward. Staffadoption of strategies to prevent the rape, and the pressing of charges was not actively considered. We doubt that such an approach would have been chosen if the victims had been members of the nursing or medical staff.

The ethical, legal, and therapeutic implications of decisions to press charges against patients for assault remain largely unexplored.2 We are conducting a survey of the attitudes to and the practice of pressing charges and of the factors that influence these. We would be interested to hear from those with experience of decisions to press charges or otherwise, particularly regarding the eventual therapeutic implications.

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Phenytoin toxicity produced by tobitumabate

M ELIZABETH BEECH, DR S V S MATHUR, AND DR B P HARROLD (Luton and Dunstable Hospital, Luton, Bedfordshire) write: We describe a patient maintained on phenytoin who developed phenytoin toxicity when given tobitumabate. A 48 year Asian woman with idiopathic epilepsy, diabetes mellitus, and ischaemic heart disease was transferred from another hospital. She was being maintained on phenytoin 200 mg once daily, and her phenytoin concentration was 31·3 mg/l. She had developed polydipsia and polyuria and her blood glucose concentration was 12·5 mmol/l (228 mg/dl). Her tobitumabate 500 mg 3 times daily was started. Her symptoms did not resolve and after five days the tobitumabate was increased to 1 g 3 times a day. Within 48 hours she developed headache, nausea, cerebellar ataxia, and tinnitus. Her dose of phenytoin was reduced to 150 mg at night and she was changed to insulin the same day. The phenytoin value the next day was 27·4 mg/l. The features of toxicity resolved completely within 48 hours. The rest of her haematological and biochemical parameters were normal. She had been treated successfully for a year with phenytoin 100 mg at night and tobitumabate 1 g three times a day.

Wesseling and Molks-Thurkow investigated 17 epileptics who were taking phenytoin who were given tobitumabate 500 mg two to three times a day.1 The total plasma phenytoin concentration fell, but the total phenytoin area under the plasma concentration curve was not significantly reduced, and the area under the plasma phenytoin concentration curve was even reduced.2 Phenytoin was also reduced in isolated uptake in vivo by tobitumabate, although reversibility was not demonstrated.4 In the study on oral administration, the area under the plasma concentration curve was again not significantly reduced.2 This indicates that the plasma protein binding of phenytoin is not affected by tobitumabate.3,4 There is some evidence that the phenytoin concentration in the cerebrospinal fluid may also be reduced,5 but this needs confirmation. Moreover, it has been suggested that the intracellular uptake of phenytoin may be reduced by tobitumabate.6 The reduction in phenytoin plasma concentration may affect patients already taking phenytoin, and we have shown that the oral bioavailability of phenytoin is reduced by tobitumabate.2,7 Phenytoin plasma concentrations were reduced in patients taking tobitumabate on several occasions.8,9 This indicates that the bioavailability of phenytoin is reduced in patients taking tobitumabate. Such a reduction may be of clinical importance.

We report a case of phenytoin toxicity caused by tobitumabate. The patient improved slowly when the dose of tobitumabate was reduced and when phenytoin was added. She was able to continue to work with a lower dose of phenytoin and tobitumabate.