have many more observations, which I quoted earlier (8 October, p 918).

I accept their point about the shorter latent period for melanoma; but the evidence for non-linearity in the incidence-exposure relation for melanoma is also relevant. Our differences of emphasis may reflect a concentration on shorter term effects by Professor MacKie and Dr Ryecroft and my concentration on the longer term outlook.

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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 rearing of a live one in the case of the death 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.[1]

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 561) 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. Although rape arouses particular strength 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,[1] and the Department of Health has recently issued further guidelines.[2] There is, however, no consensus on whether or in what circumstances patients should be prosecuted for acts of violence committed in psychiatric hospitals.

Opinion 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 active support of 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 assaults 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. Staff adopted a "hot bath and cup of tea" approach, 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.[3,4] 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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Drug Points

Immune thrombocytopenia and interferon alfa

Drs EDWARD J KANFER and DONALD M MCCARTHY (Department of Haematology, Charing Cross and Westminster Medical School, Westminster Hospital, London SW1P 2AP) write: Recombinant interferon alfa has become a common treatment in patients with hairy cell or chronic granulocytic leukaemia. Its efficacy has also been investigated in several other haematological disorders. A thrombocytopenic effect of this agent has been observed frequently and this finding has been used in the management of essential thrombocythaemia.[1] We have recently seen a patient with lymphoma who developed a severe immune thrombocytopenia while receiving interferon alfa.

A 43 year old woman presented in 1982 with stage IV non-Hodgkin's lymphoma (small lymphocytic). Remission was induced, but this was followed by multiple relapses, culminating in severely symptomatic advanced disease. She achieved a partial remission with combination chemotherapy and in March 1988 began receiving interferon alfa-2b (3 million units, 3 times weekly). At the start of this treatment her blood count showed a haemoglobin of 131 g/l, white cell count $5{\cdot}3\times10^9$/l, and platelet count 200×10^9/l. Seven weeks later the dose of interferon was reduced to twice weekly because of leucopenia and thrombocytopenia (white cell count of $2{\cdot}3\times10^9$/l and platelet count of 30×10^9/l). Over the next six weeks the cell count rose to $5{\cdot}0\times10^9$/l, although the platelet count remained low at 36×10^9/l. Two weeks later she presented with widespread purpura. A blood count showed a haemoglobin of 115 g/l, white cell count of $4{\cdot}6\times10^9$/l, and platelet count of 7×10^9/l. Bone marrow examination showed a cellular marrow with many more megakaryocyes than normal. Platelet associated immunoglobulin studies showed an IgG of 3800 ng/10^6 platelets (normal 2-10), IgM of 200 ng/10^6 (normal $<2{\cdot}5$), and C3d of 40 ng/10^6 (normal $<3{\cdot}3$). The interferon was discontinued and she was started on prednisolone (1 mg/kg/day). A rapid rise in her platelet count followed and the steroid dose was tapered over the next few weeks without event.

Interferon may cause thrombocytopenia by an inhibitory action on megakaryocyte progenitor cells. A recent study of patients receiving interferon has, however, shown raised levels of platelet associated immunoglobulins,[2] suggesting that immune mediated platelet destruction may be important. The association of immune thrombocytopenia with non-Hodgkin's lymphoma is well recognised,[3] but its development in our patient six years after presentation suggests that interferon may have been the precipitating cause. This interpretation is supported by both the study quoted above and previous reports which have associated interferon treatment with immune thrombocytopenia.[4,5] Physicians using interferon should be aware of this potential complication.

We thank Dr U Hegde for performing the platelet associated immunoglobulin investigation.

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Phenytoin toxicity produced by tolbutamide

MS 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 tolbutamide. A 48 year Asian woman with idiopathic epilepsy, diabetes mellitus, and ichaemic 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 12-15 mmol/l. Tolbutamide 500 mg three times daily was started. Her symptoms did not resolve and after five days the tolbutamide was increased to 1 g three times a day. Within 48 hours she developed headache, nausea, cerebellar ataxia, and nystagmus. 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 medications remained unaltered. In 1982 she had been treated successfully for a year with phenytoin 100 mg at night and tolbutamide 1 g three times a day.

Wesseling and Mols-Thurkow investigated 17 epileptic patients taking phenytoin who were given tolbutamide 500 mg two to three times a day.[1] The total plasma phenytoin concentration fell, but the proportion of free plasma phenytoin increased by 44·6% of control values. This displacement of phenytoin from plasma proteins lasted four days. None of their patients showed features of phenytoin toxicity during that phase. There is a single case report of ataxia and drowsiness when phenytoin and tolazamide were given for several weeks.[2]