A child who plays out of sight and hurts himself, typically first starts to run towards the parents, and only after a while begins to cry. Such observations indicate that there is no simple connection between the pain felt and its expression even in children.

In contrast to an idea of naturally expressed pain, is also the fact that children with early infantile autism may hurt themselves without showing any visible reactions. For example, Mahler (1952) reports that an autistic child, seeing his mother light a cigarette with a lighter from the dashboard of the car, seized the lighter and burned his mouth, apparently without feeling any pain. This failure to express pain has been interpreted as a sign that autistic children suffer from some defect in the nervous system which make them insensitive to pain (Rimland, 1964). There is, however, another possibility; that expressing pain is primarily communicative.

Several case histories of autistic children support the idea that expression of pain is linked with communication in a wider sense. If children who show insensitivity to pain improve to the point where they begin to communicate, they typically also begin to express pain. This might be due to some improvement of the nervous condition of these children, but this is not a likely explanation since comparable courses of development are found among the congenitally blind, and children raised in institutions (Keeler, 1958; Provence and Lipton, 1962). These children may fail to show pain at an early age, but at later ages communicate both their pains and pleasures. There is no reason to believe that they share some neurological pathology with the autistics. The children do, however, face difficulties during their development which may hamper the acquisition of communicative skills, indicating that expression of pain may be dependent upon communicative ability.

Furthermore, if the expression of pain depends upon the child's communicative ability, it follows that children who differ in their means of communication should also differ in their expressions of pain. A case which apparently demonstrates this has been brought to our attention. A fifteen-month-old normal hearing boy with deaf parents fell down the stairs and cut his forehead. He bled freely, but did not cry. He did, however, run to his parents pointing demonstratively to his forehead. Thus the boy expressed his pain according to his parents' communicative abilities, and was duly rewarded with attention and care. This happened while the family were on a visit to hearing relatives for a couple of months. The relatives were shocked by the boy's 'unnatural' reactions, and as a consequence of this the deaf parents employed a hearing housekeeper when they returned home. During the stay with his hearing relatives the boy more frequently started to cry when hurting himself.

The case of this boy, as well as the other above mentioned observations makes it a warranted suggestion that pain has no natural expression.

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MORE ON ALCOHOLISM AND DEPRESSION

DEAR SIR,

Galdi and Bonato (*Journal*, August 1977, **131**, pp 221–2) took an interesting epidemiologic approach to elucidate the relation between alcoholism and psychiatric disorder. However, a number of conceptual and methodologic reservations must be raised in addition to those mentioned by them.

Hospital admission rates reflect treated prevalence, not incidence, especially in the case of depression. Over a third of affectively ill patients in Helgasson's epidemiologic study were not admitted to hospital anywhere for their illness (2). This rate is subject to the vagaries of fashion, accessibility, administrative and clinical interest, budgeting, reporting, social attitudes, etc, in addition to actual changes in level of need.

It is noteworthy that admission rates for psychiatric disorders also increased significantly during the six-year period in question. There is evidence that in the long run this rate as well as the prevalence of psychiatric disorders should remain steady (1, 3). Therefore such a marked increase may reflect transitory, atypical trends in Sweden.

Further, patients with so-called depressive spectrum disease (DSD) (6), a depression closely linked to alcoholism familially, have a different rate of hospital

admission from patients with pure depressive disease (PDD). Van Valkenberg *et al* (4), in a study comparing the two groups found that DSD patients had a 26 per cent chance of being re-admitted after the index admission, compared to 44 per cent of patients with PDD ($P < 0{\cdot}025$). This reflected a 23 per cent rate of one or more relapses among the DSD patients, compared to 48·7 per cent relapse rate among PDD patients ($P < 0{\cdot}005$).

DSD patients might then be underrepresented in rates of hospital admission for psychiatric disorder as compared to other depressed patients. This would be particularly true if DSD patients suffered from less severe symptoms than the PDD patients. That there may be a differential severity in different types of depressions has already been shown, e.g. Weissman *et al* (5) demonstrated that secondary depressions have fewer symptoms than primary depressions.

A loosening of restrictions on alcohol should not strongly affect such rates, since DSD patients tend not to abuse it (6). Patients in whom alcoholism is a primary disease would need hospital admission for alcoholism; however, it is their non-drinking relatives (usually females) who typically need to enter psychiatric hospitals for depression. Whether the alcoholic male relations of depression spectrum patients would suffer depression if the source of alcohol were denied is currently unknown. It is certainly worth investigating. Failure to show an inverse relation in admission rates does not disprove a link between alcoholism and psychiatric disorder.

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THE RIGHT WAY TO TREAT SCHIZOPHRENIA?

DEAR SIR,

A schizophrenic patient in her thirties, after occasionally taking trifluperazine for two years and having four hospital admissions in that time, was put on to depot flupenthixol in 1973 and after eleven months developed fairly severe tardive dyskinesia with generalized chorea.

Her neuroleptic was replaced with pimozide 4 mg every morning, and in the course of the next five months her abnormal movements disappeared entirely. Shen then refused to take her tablets, but four months later appeared in out-patients again complaining of auditory hallucinations and ideas of reference. Shen then took her pimozide in a dose of 4 mg every morning for six months, when it was reduced to 2 mg, and for the next two years I thought that she had religiously taken this medication with complete control of her illness. In fact she now tells me that she takes the pimozide for two months, and then leaves it off until her voices return, which usually takes about twelve weeks, to re-start the antipsychotic for another two months.

She seems to have achieved the object that we should all be striving for, and that is to control psychosis with the smallest dose of neuroleptic. Would that all patients had such insight.

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AN OPEN LETTER ON WARD ROUNDS

DEAR SIR,

I have recently been a patient in a large psychiatric hospital. Each week there was a ward round or meeting, as it was called. I would like to question the therapeutic value of these.

I believe that it is current psychiatric practice to interview the patient in front of the whole psychiatric team; some of whom have nothing to do with that