CASE REPORT

Five months after sustaining a head injury associated with fracture of the left side of the vault of the skull the patient was able to resume work as engineer supervisor of maintenance of aircraft. His only residual complaint when he was seen two years after the accident was a persistent tinnitus. He discharged satisfactorily his exacting duties, which entailed personal contact with his superiors, peers, and subordinates. The accident resulted in litigation, in the course of which the patient met the defendant's solicitor, who described him as being "an extraordinarily pleasant plaintiff." Concurrently, the patient showed personality changes at home, which his wife described as follows: ". . . little things provoke a temper; he shows a lack of consideration for his family. He used to have good relations with his son, but since the accident, no matter what they discussed, they used to finish in a slanging match. If my son or his wife put the towel in the wrong place he would go for them. I believe [the patient's] temper was the reason why they emigrated. When, last Christmas, I told them, 'I can't like Dad anymore,' my son said, 'I can't blame you.' These have been the unhappiest years in my whole life. Even if he improves these two years have done something which cannot be repaired; it hurts when he says nasty things. There was a phase when everything I said was wrong—I still can't do anything right—he always finds fault with everything. First thing in the morning is the worst time. If you burn the toast that's a terrible thing and he flares up . . . he has almost gone berserk on occasions—mad—almost frighteningly so, as if he has lost his senses—shouts and carries on—just goes mad with temper. Though you try to make allowances for his accident it still affects you if you get it day in and day out. When the children were staying with us he would say: 'I heard what you said,' thinking we were talking about him when we weren't. He smokes and drinks more than he used to . . . he is spending money foolishly; he goes into the bar and, irrespective of who is there, buys them drinks. I have learnt to think before I say anything. When he is upset his favourite expression is 'You bloody idiot.' Many a time I would have run away from him if I had somewhere to go—I even thought of taking a bed-sitting room."

Discussion

These cases are presented as a phenomenological study of a group sharing a similar behaviour disorder, the essential element of which is an abrupt onset of hostility towards a few people with whom the male patient has close emotional ties, especially his wife. This enmity may also extend towards parents, siblings, and children. The sustained increase in hostility may first show itself in acts aimed at injuring or denigrating the spouse or some close member of the family. These acts may occur under the influence of alcohol—so often one of the preconditions of wife-battering—or under chlordiazepoxide medication. One patient on chlordiazepoxide physically assaulted his wife for the first time in 20 years of marriage.[1]

In Capgras's syndrome hostility is similarly projected on to close members of the family, ostensibly on the grounds that they are impostors disguised as exact doubles. As in the group of patients presented here head injuries and other cerebral conditions precede the onset of Capgras's symptoms in some cases.[2 3] This pattern of antisocial behaviour within the family milieu may be due to release from inhibition, as a result of which there is a regression—in hierarchical terms—to a less adult mode of behaviour. Previous conditioning and past experience may facilitate the development of the behavioural disorder. Flugel's observation may provide an explanation for these phenomena, "It is man's unique and inevitable tragedy—due to his long period of helpless infancy—that he is compelled to hate those whom he also most loves."[4] The poet, John Clare (1793-1864), who spent many years in the General Lunatic Asylum at Northampton, described this mental state in similar terms:

> And even the dearest that I love the best
> Are strange—nay, rather stranger than the rest.

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SHORT REPORTS

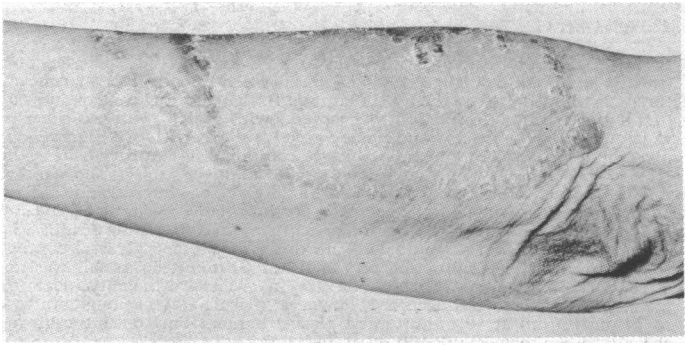
Oculocutaneous Reaction to Oxprenolol

A patient developed a psoriasiform eruption and dryness of the eyes after six months' treatment with oxprenolol. Forty-eight hours after oral challenge, the skin eruption recurred in the same places. Further challenge with practolol failed to reproduce the lesions.

Case Report

In November 1973 a 65-year-old woman was treated for hypertensive heart disease with methyldopa 250 mg twice daily, digoxin 0·25 mg daily, and oxprenolol 20 mg twice daily. There was no past or family history of skin disease. In April 1974 she developed symmetrical psoriasiform plaques on the extensor aspects of both forearms (see fig.) and on both cheeks. The plaques had prominent marginate scaling with some clearing at the centre. The palmar and plantar skin was not affected. She complained of eye dryness, and tear secretion measured by Schirmer's test was reduced bilaterally. Oxprenolol was stopped. The skin eruption improved after seven days, beginning as a reduction in the peripheral scaling, and disappeared after two months. Treatment with methyldopa and digoxin continued. Skin biopsy showed epidermal oedema and the epidermis was invaded in places by lymphocytes and histiocytes. There were scattered foci of swollen eosinophilic cells, some of which had pyknotic nuclei. There was oedema and a cellular infiltrate composed of lymphocytes and histiocytes in the papillary and reticular dermis. Direct immunofluorescence of the diseased skin showed a fine granular deposit of IgM and complement at the dermo-epidermal junction. The antinuclear factor test was negative.

In January 1975 after two days of oxprenolol (20 mg twice daily) she developed erythema in the same places as before and, after five days, scaling appeared at the margins of the erythema. Oxprenolol was discontinued, and the eruption subsided within two weeks. She was later given practolol 100 mg twice daily for seven days. The skin lesions did not recur and the drug was discontinued.



Psoriasiform plaques on extensor aspect of left forearm.

Discussion

Many cases of cutaneous and ocular reactions to practolol have been reported[1-3] but there have been no confirmed reports of such reactions in patients receiving oxprenolol. This patient had a psoriasiform eruption which recurred in the same places after oral challenge. The eruption was not preceded by scaling and thickening of the palmar and plantar skin, a feature which was invariably present in patients developing a psoriasiform eruption on practolol.[2] Direct immunofluorescence showed a pattern similar to that described by these authors. Oxprenolol has been prescribed as an alternative β-blocking agent in patients who have developed cutaneous and ocular reactions to practolol. This report emphasizes the importance of

closely watching patients receiving oxprenolol so that any adverse reactions may be detected at an early stage.

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Glycerol Therapy for Cerebral Oedema Complicating Fulminant Hepatic Failure

A recent analysis of the main causes of death in a consecutive series of 132 patients (96 deaths) with fulminant hepatic failure (F.H.F.) seen over a seven-year period showed cerebral oedema in 35 (31%) of the 92 cases in which the brain was examined at necropsy. In 20 of these there was associated brain herniation, and in some cases death appeared to be due to cerebral oedema at a time when liver function was improving. The mechanism of the development of the oedema and its relation to the encephalopathy is unknown but clearly an effective means of control might improve the survival rate in this condition. Because of experience in neurosurgery, both dexamethasone and mannitol have been advocated in F.H.F. but the former may lead to gastrointestinal bleeding and the latter is often followed by a rebound increase in intracranial pressure. Glycerol given intravenously as a 10% solution in a dose of 50 g over 24 hours reduces the neurological deficit resulting from a cerebrovascular accident, possibly by limiting cerebral oedema,[1] and there is one report of its successful use in F.H.F.[2] As the liver is an important disposal site of endogenous glycerol liberated during lipolysis we have measured its concentration in blood in patients with F.H.F. and have also investigated the effect of infusion of a 10% solution.

Case Histories

Eighteen untreated patients with F.H.F. who were in grade 4 hepatic coma showed significantly higher ($P<0.05$) basal whole blood glycerol concentrations (mean 0·354 ± SEM 0·118 mmol/l) than fasting control subjects (0·074 ± 0·005 mmol/l). Five further patients with F.H.F. who had deteriorated to grade 4 hepatic encephalopathy were treated with intravenous glycerol in a dose of 50 g/24 hours but we could find no improvement in the level of consciousness. Further studies in two volunteer subjects showed that this dosage led to only a minor rise in blood glycerol concentration and no change in plasma osmolality. To produce an appreciable rise in blood glycerol concentration a further fasted control patient was given a loading dose of 6·5 g followed by 13 g over 40 minutes. With this dosage blood glycerol concentration rose to a maximum of 2·64 mmol/l at 40 minutes but this was not associated with an appreciable increase in plasma osmolality. Definite intravascular haemolysis occurred, however: plasma free haemoglobin rose to 70 mg/100 ml at 40 minutes and plasma haptoglobin concentration fell sharply to less than 10 mg/100 ml at 24 hours.

Discussion

Both endogenous and exogenous glycerol are removed principally in the liver and kidney and possibly this process is impaired in hepatic disease, which explains the significantly increased blood glycerol levels found in patients with F.H.F. The low dosage of intravenous glycerol (50 g/24 hours) given to these patients did not improve their level of consciousness and the benefit seen in patients with stroke given the same dose may be due to a direct effect on infarcted brain. Higher doses of intravenous glycerol may benefit cerebral oedema by an osmotic action and in patients with stroke given 17 g/h there was an associated increase in cerebral hemisphere blood flow and a decrease in the respiratory quotient. Cerebrospinal fluid pressure decreased and no rebound occurred.[3] Our results show, however, that similar doses may result in intravascular haemolysis and this has also been reported using other dosage schedules (60-80 g of glycerol as a 20% solution over 15-60 minutes and also after 50 g as a 10% solution over 60-90 minutes).[4,5]

We conclude that intravenous infusion of 10% glycerol (50 g/day) is ineffective in improving encephalopathy in patients with F.H.F. and that higher doses may result in dangerous intravascular haemolysis.

The continued support of the Medical Research Council is gratefully acknowledged.

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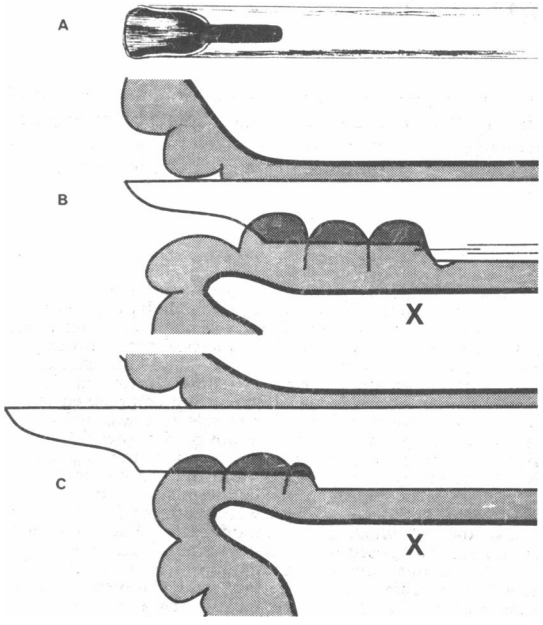
Modified Oesophagoscope for Injecting Oesophageal Varices

Emergency operations for the arrest of bleeding from oesophageal varices carry a high mortality rate.[1-4] Better results have been reported using injection of sclerosants through the oesophagoscope,[5] but manipulating accurately the point of the needle at the distal end of the oesophagoscope is difficult. The technique is greatly simplified by modifying the 50-cm Negus oesophagoscope after the style of the Gabriel proctoscope.

Modification and Technique

The modification consists of cutting a slot 0·5 cm × 4·0 cm in the lower end of the instrument diagonally opposite the beak (see fig. (a)).



Modified oesophagoscope. (a) Slot cut in distal end. (b) Prolapse of varix and overlying mucosa into slot, which allows needle to be advanced directly into mass of varix. (c) Oesophagoscope advanced to compress site where injection has been given.