that people are not too grievously exploited in one way or another by those higher up the scale. An excellent example of this sort is the National Association for Mental Health. For many years it has snapped at the ankles of the Establishment in defence of one particularly inarticulate and powerless minority, the mentally disordered.

From time to time the N.A.M.H. has published reports subtly entitled *Mind*. The tenth in the series[1] was recently issued with the subtitle "Patients' Rights: the Mentally Disordered in Hospital." It is mainly concerned with the deprivations that patients admitted to psychiatric hospitals suffer as the result of the exercise of compulsory orders, the most important being the loss of liberty. Though compulsion is always to be regretted, it is at times necessary in the interests of the patient and of society at large. But it would not be too cynical to point out that, in contrast to the special hospitals, to abscond from most conventional mental hospitals presents no great difficulty. Indeed it is sometimes said that to abscond is far too easy and that it is just those patients who are most dangerous who may be the ones to go.

This illustrates the anomalous position in which mental hospitals have been thrust by virtue of the two incompatible roles, therapeutic and custodial, they are expected to fulfil as an unforeseen consequence of the Mental Health Act, 1959. And the custodial function is increasing in importance. This is reflected in the statistics given in the *Mind* report for compulsory admissions in the years 1962 and 1970. A comparison of these two years shows that the number of orders under Section 135-6 (Place of Safety Orders) have more than doubled—namely, from 740 to 1,493. Furthermore, if it is assumed that "other admissions" refers to those under Part V of the Act (admission of patients concerned in criminal proceedings, etc). it is to be noted that here again the numbers have increased—namely, from 1,584 to 1,725. This means that there has been a substantial increase in the number of mentally abnormal offenders compulsorily admitted, either without prosecution in the case of Sections 135-6 or as a result of prosecution under Part V, for whom the medical profession is not initially responsible.

The report then explores the way in which patients may be subject to inadequate or unsatisfactory treatment or exploitation. It takes up the familiar theme of the scant attention paid to long-term patients by consultant psychiatrists. Unfortunately the case made out is all too convincing and confirms what has long been recognized—the inadequate medical staffing of our mental hospitals. Just as important is the right of patients to refuse treatment. The report stresses the failure of the 1959 Act to say anything about the treatment of people against their will. A demand is made that the Department of Health and Social Security should give firm guidance on the subject to the Responsible Medical Officers. It is only fair to comment that the Secretary of State, Sir Keith Joseph, in a written answer in the House of Commons on 23 January stated briefly but succinctly the position as he sees it.[2] His reply was as follows: "I am advised that, in the case of a patient detained for treatment under the Mental Health Act, any recognized form of treatment which is considered necessary for such disorder may lawfully be administered without the consent of the patient. Where, however, the patient is capable of understanding what is proposed, it is the normal practice to explain this to him and, if possible, to obtain his agreement."

In an attempt to safeguard patients against exploitation the report discusses the danger of their use as "cheap labour" and the unfairness of not being paid the rate for the job. It emphasizes too the golden rule that in all forms of employment the therapeutic work programmes should be made for the patients rather than "patients being made to fit into an arbitrary programme of little relevance to their usual way of life." It deals next with the satisfaction of ordinary human needs, in particular adequate feeding and the right, when possible, of patients to wear their own clothes and to acquire possessions of their own. All in all it recommends measures to improve their way of life and advocates the means whereby they may retain their dignity. The danger to civil rights is stressed, in particular the disfranchisement of those patients in hospital for the mentally disordered who have no home address. Commenting on the review procedures for detained patients the report deplores the rubber-stamp method of extending compulsory orders by hospital management committees. And it asks for patients appealing to mental health review tribunals to be represented either by qualified lawyers or by a lay "patient's friend," for when this is done the number of recommendations for discharge is substantially increased.

The points discussed, though not necessarily new, are nevertheless of importance, and none of the recommendations made can be ignored. But perhaps the most important is contained in the final paragraph. This refers not to the sins of the hospitals themselves but to the lack of facilities in the community to support the patients after they leave. The last sentence should be pondered: "The right to leave means nothing if there is nowhere else to go."

[1] *Mind Report* No. 10. National Association for Mental Health, 39 Queen Anne Street, London W1M 0AJ.
[2] *Hansard*, 23 January 1973, col. 77.

Clinical Diagnosis of Reye's Syndrome

Reye's syndrome is characterized pathologically by cerebral oedema without cellular infiltration or demyelination, and fatty degeneration of the viscera, especially the liver.[1] Its cause is unknown. Mortality in reported series varies from 25% to 80%.[2-4] Recovery may be complete[4] or associated with serious neurological deficits.[2] Whether the syndrome is a distinct entity has been questioned, but the clinical, biochemical, and pathological abnormalities in childhood[1 4-7] are remarkably consistent.

The disorder occurs in children aged 2 months to 15 years. The typical case presents as an acute encephalopathy with seizures and disturbances of consciousness proceeding rapidly to deep coma. Sustained seizures, with periods of tonic posturing with flexed elbows, clenched fists, and extended legs and hyperpnoea or irregular respiration are symptoms that suggest the diagnosis. There are no focal neurological signs or meningismus. Mild to moderate hepatomegaly is the only clinical evidence of visceral disease, and even this is lacking in 50% of cases. Deepening coma with death in 24 to 48 hours is the usual course. Clinical diagnosis is based on these findings in the absence of other apparent causes of acute encephalopathy. Confirmation is obtained by finding at biopsy intense fatty infiltration of the liver, seen as diffuse vacuolation of the hepatocytes without nuclear displacement and no hepatocellular necrosis.[7] The nucleoli may be enlarged and irregular. Elec-

tron-microscopy shows all the hepatocytes to be affected by a process which results in pleomorphic, swollen mitochondria, proliferation of smooth endoplasmic reticulum, a great increase in peroxisomes, and accumulation of triglyceride in small droplets.[8] Post-mortem material shows massive changes, with fat deposition also in the renal tubules and myocardium.

Liver biopsy is not always possible because the hepatic lesions are often accompanied by low prothrombin activity, which is a contraindication. But readily available laboratory tests will support a presumptive diagnosis. The cerebrospinal fluid is normal except for its low sugar content and in rare cases increase in lymphocytes. Acidosis, hypoxia, and raised serum potassium levels are prominent. Most important are biochemical abnormalities suggesting hepatic disease. These have been systematically studied in the most recent[9] of a series of well documented reports[5 6 10 11] from the Medical Research Laboratory, Seato Medical Project, Bangkok, and the Khon Kaen Provincial Hospital in Thailand, where Reye's syndrome occurs with great frequency and is a major cause of death in children 1-6 years old. Circumstantial evidence suggests that ingestion of aflatoxin (a fungal poison) may have an aetiological role in that region.

In a prospective study of 81 children admitted with acute encephalopathy and no immediately apparent cause A. M. Glasgow and colleagues[9] considered criteria for the non-pathological diagnosis of Reye's syndrome and tried to evaluate the role of excess blood ammonia in the encephalopathy. Forty-three cases were considered to have Reye's syndrome on the basis of three criteria: (1) acute encephalopathy of no apparent cause; (2) typical hepatic histological features or two out of three abnormal biochemical indicators of malfunction—namely, serum asparate transaminase level two and a half times normal, a prothrombin activity of less than 60% of normal, a blood sugar of less than 60 mg/100 ml, or cerebrospinal fluid sugar of less than 40 mg/100 ml; and (3) no other reasonable explanation for the cerebral or biochemical abnormality. The control group were 27 children in whom a definite diagnosis other than Reye's syndrome was finally established. The majority of these had encephalitis. A further 11 children remained undiagnosed. Only 4 of the 39 cases of Reye's syndrome did not satisfy the histological features, while 2 in the unknown group were considered probable cases on histological grounds. Twenty of the Reye's syndrome cases satisfied three of the biochemical criteria, 40 satisfied two, as did one of the control cases, a child with tuberculous meningitis. The authors thus considered that abnormality in any two of the three biochemical tests, while not specific, afforded a satisfactory way of diagnosing the disorder. Indeed, in a separate article[12] they describe in detail two cases from the series with typical clinical and biochemical features in which the hepatic lesion was limited to swollen, pale hepatocytes with enlarged nuclei and irregular nucleoli but only minimal fatty infiltration seen as fine droplets at the periphery of the lobule. They admit that this is outside the usual definition of Reye's syndrome but suggest that it is one end of a spectrum of fatty infiltration, which is a variable manifestation of hepatic injury rather than its cause.

An excess of ammonia in the blood, or hyperammonaemia, is another presumed sign of hepatic injury and was a common finding in these patients. Levels greater than 0·1 μmol/ml (normal range 0·025—0·095 μmol/ml) were found in 32 of 40 cases of Reye's syndrome as compared with only 7 of 38 in the other groups. Only in Reye's syndrome were levels greater than 0·15 μmol/ml recorded, and in nine cases levels of more than 0·5 μmol/ml were found. The highest levels were found in the most deeply comatose patients, but many with normal or only slightly raised levels were also deeply comatose. In many instances raised concentrations of blood ammonia fell to normal values after admission to hospital but the patient remained deeply comatose and died. Cleansing enemata to reduce colonic production of ammonia had no apparent effect. Two patients with hyperammonaemia and deep coma responded rapidly to the standard management of intravenous infusion of 20 ml of 50% glucose. Though blood ammonia levels are only an indirect estimate of cerebral ammonia and may not be the best measure of ammonia intoxication, these observations do suggest that factors other than ammonia retention and hypoglycaemia are important in the encephalopathy of Reye's syndrome. Raised non-esterified fatty acid,[13] a known cause of coma,[13 14] may also be important. In a variety of experimental animals raised concentrations of ammonia and free fatty acids have an additive effect in producing coma.[15]

Apart from lack of jaundice the biochemical abnormalities in Reye's syndrome are similar to those of acute hepatocellular failure and are consistent with the hypothesis that hepatic dysfunction may be the primary event in the syndrome, leading to the encephalopathy. The ultrastructural changes in the hepatocytes are likely to be associated with other abnormalities of intermediary metabolism, involving possibly amino-acid and catecholamine metabolism, both of which are deranged in hepatic encephalopathy.[16]

The high mortality of the disorder and lack of effective treatment are again highlighted by Glasgow and his co-workers. Only 12 of 43 patients survived. In general the more severe the biochemical abnormality the worse the prognosis, but, more specifically, 7 out of 8 patients with normal blood ammonia levels survived and only 1 out of 23 with a potassium level of greater than 5mEq/litre did so. In sporadic cases these observations may be of value in assessing the efficacy of treatment, which remains empirical.

Hypoglycaemia, hypoxia, acidosis, electrolyte abnormalities, and tendency to bleeding require correction.[17] A low protein diet with sufficient carbohydrate intake to minimize endogenous protein metabolism, neomycin by nasogastric tube, and enemata as used in hepatic encephalopathy seem rational and are commonly used. Dexamethasone or mannitol infusion to minimize cerebral oedema, and peritoneal dialysis or exchange transfusion to remove hypothetical toxins, are advocated[3 4 7 8 18] and must be considered in such a serious disorder. By defining criteria for a non-pathological diagnosis of Reye's syndrome and clarifying some prognostic indices Glasgow and his colleagues have facilitated assessment of the efficacy of such treatment by controlled trials. The sporadic nature of the syndrome in most places may require such trials to be multicentric, perhaps based on units experienced in the management of acute hepatic failure.

Early investigation of children with acute encephalopathy of no apparent cause is mandatory if treatment is to be applied before irreversable changes occur. Clearly the investigation of any child presenting with coma or seizures of no apparent cause should include determination of blood sugar, serum aspartate transaminase levels, prothrombin activity, and, if possible, blood ammonia levels.

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Calcaneal Fractures

Fractures of the os calcis are important because of the severe disability they cause. They occur mainly in men whose work requires them to climb heights or who are heavy manual workers. They incapacitate patients frequently at the peak of their earning power and at a time when their financial commitments are greatest.

The fractures were classified by R. Watson-Jones into three types.[1] This classification is useful because it reflects the degree of trauma causing the injury and it indicates the prognosis. Type A fractures are isolated fractures of the calcaneum, and they may be comminuted. They are caused by less severe trauma than the others, and the resultant prognosis is good. Type B fractures have minimal injury to the posterior talo-calcaneal joint, and type C show significant displacement and involvement of this joint. The last two types of fracture are rarely shown in full by the standard radiographs of the bone, and special views of the posterior talo-calcaneal joint are essential.

S. Nade and P. R. W. Monahan have reported a series of 203 fractures of the calcaneum in 185 patients seen at the Radcliffe Infirmary, Oxford.[2] Almost a quarter were occupied in climbing heights and another quarter were heavy manual workers. Children were rarely affected. Falls from heights were responsible for most of the type C fractures, whereas less severe accidents, such as twisting, falls from kerbs or stairs, or crush injuries, were responsible mainly for type A and B fractures. Type C was the most common fracture and type B was the least common. In this series 28% of the patients had other injuries. These were mainly to the same foot or limb, but five patients had vertebral fractures and 13 had other lesions.

Pain, bruising, and swelling of the heel are the presenting features of this fracture, and the heels are tender to touch on examination. In the face of such an obvious injury further examination of the patient might be thought unnecessary, but this is essential because of the risk of other injuries, especially vertebral fractures. The pain from these calcaneal fractures is so severe that it may mask the pain from the other injuries. A full radiographic investigation of all tender areas is essential.

The treatment reported by Nade and Monahan in their study was not uniform, and this variation reflects the general opinion among surgeons. While all their patients were treated conservatively, they recommend that the place of surgery should be reviewed. Most surgeons treat their patients conservatively, but the exact method of treatment varies. Crepe and wool bandages and raising the affected foot with the patient rested in bed comprise the simplest treatment. Admission to hospital is indicated if there are other severe injuries. Analgesics initially are essential and trypsin-chymotrypsin tablets may help to disperse the haematoma. The application of a well-padded plaster below the knee often provides the patient with more comfort. Because the os calcis in type C fractures is broadened when viewed from behind, manipulation under anaesthetic during application of the plaster is sometimes undertaken in an attempt to mould the bone into a more normal shape. This enables the patient to wear a normal shoe later when he has recovered. Surgical treatment is essential in all open fractures, but in closed fractures opinions vary about surgery. Operative procedures include raising the superior border of the os calcis with a Steinmann pin inserted through the heel and bone-grafting the os calcis to achieve the same result and finally fusion. Early fusion was advocated by Watson-Jones because he was so disappointed by the long-term results of these fractures, but most people wait to see the end product before embarking on this procedure. The fusion undertaken might be a local subtalar fusion or even a triple fusion involving the subtalar and midtarsal joints.

Nade and Monahan's patients who were admitted to hospital stayed for an average duration of 6·7 days and were away from work for 15·6 weeks, the range for the latter being 7·3 weeks for type A and 23 weeks for type C. Symptoms can be expected to improve for two years, but even after this time patients may have complaints. These are pain, local swelling, stiffness, cold feet, and cramps. Patients with type C have the worst symptoms. In Nade and Monahan's study 57% of patients were so affected, and many suffered loss of agility, being unable to stand unaided on tiptoes, walk on uneven ground, and climb ladders; 17% had been granted a governmental disability pension ranging from 5 to 20%. Of the patients with bilateral fractures, only one from whom the information was available returned to work within six months.

Patients suffering these fractures are faced with a long period of pain and disability, loss of pay, loss of function, and perhaps loss of work. A satisfactory treatment has yet to be devised.

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Glucagon and Diabetes

Diabetes mellitus is characterized by relative or absolute deficiency of insulin. Three of its main biochemical features—hyperglycaemia, increased mobilization of body fat, and increased protein catabolism—appear to be primarily caused by lack of the normal restraining influence of insulin on gluconeogenesis, triglyceride hydrolysis, and protein turnover respectively. Insulin's action in regulating fat mobilization and protein turnover appears to be mainly at the level of the peripheral tissues. By favouring protein synthesis and inhibiting proteolysis, insulin controls the flow of glucogenic amino-acids to the liver, where they may be subsequently converted to glucose.[1] In a similar manner insulin promotes