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Patients who take overdoses

Recent guidance issued by the DHSS on the management of deliberate self harm[1] is based on advice from a group set up by the Royal College of Psychiatrists. In 1968 the department endorsed the report of Sir Denis Hill's committee on the hospital management of acute poisoning.[2] The Hill report covered both the medical and the psychiatric aspects of poisoning, and it conveyed a sense of urgency about a growing problem that was then not too widely understood. We are now resigned to its magnitude, and the new report reflects that existing responses are reasonable. Its thrust is towards greater efficiency and better coordination of professional services—and especially more effective aftercare.

The principal innovation lies in setting aside the earlier recommendations that every poisoned patient should be seen by a psychiatrist before discharge from hospital. The Hill committee agonised over whether that counsel of perfection was capable of realisation. Very largely it has been. Sixteen years later the new guidance is that the "psychosocial" history taking and the psychological as well as the physical assessment of "cases" (patients?) may be carried out by junior doctors on medical wards or in accident and emergency departments. This belief has been shown to be correct and reflects a triumph of psychiatric education.[3] Doctors qualifying today are aware of the factors associated with deliberate self harm and know how to assess suicidal risk. Some 10% of acute medical admissions are instances of self poisoning. Such an incidence demands that doctors should possess the necessary psychological and epidemiological understanding, and the proper skills and uncensorious attitudes required to elicit the somatic, psychological, and social information relevant to making decisions about individual patients. The guidance rightly insists that psychiatric help must always be procurable, "by telephone if necessary." "In person if necessary" would have been better. The report advises that instruction should be given to each new intake of junior medical staff and to other professional staff in order to reinforce the skills and knowledge of junior doctors. The procedures may be found set out with clarity and conciseness in a recent booklet aimed at all professions concerned.[4]

Other health professionals may carry out the psychosocial assessments. Nurses[5] and social workers[6]—when given intensive psychiatric training—have proved able to do this competently, but this training cannot go by default. The generality of nurses and social workers cannot undertake this task simply by virtue of their basic training. When so trained they can provide valuable help to a hard pressed psychiatric service, but the assessment and treatment of patients must remain a medical responsibility. The guidance welcomes such assistance where appropriate.

District health authorities are urged, in the current jargon, "to take the initiative for preparing . . . a clearly laid down policy or code of practice agreed by" all concerned. There should also be "a local multidisciplinary group which should include representatives of nursing, social work, and general practice." When a poisoned patient gets to a hospital the report says there must be both a full physical assessment and a psychosocial assessment (equally full?) carried out before he is discharged. If, however, it is the general practitioner who sees the patient first he "may, if he believes there is no risk to life, decide not to send the patient to hospital and to manage the case at home." The hospital doctor's thoroughness, it seems, need only be matched by the general practitioner's belief. The DHSS ought to have eliminated such tendentious double talk.

The report, like its predecessor, urges special consideration for children and adolescents. (The Royal College of Psychiatrists' guidelines on these are excellent[7].) It points to the, often unrecognised, part that may be played by alcoholism and drug dependence. It emphasises that the extent of physical harm cannot be used as the yardstick to determine the extent of psychological illness or the need for psychological or social help. It is a pity that it omits the warning not to accept readily that an adult's poisoning has been accidental or inadvertent.

The report recommends that patients should be sent to particular medical wards. Refined resuscitation facilities are rarely required, however, and I do not support this suggestion.

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Readers are reminded that the *BMJ*'s current classified job advertisements are also available on Prestel.

It is better for the patients if they are not singled out, sequestered, and so stigmatised. Similarly I doubt whether it is practical to have special nurses, some with psychiatric training, to look after them, or for specialist social workers to devote a substantial part of their time to such patients. On the other hand, the report gives sound advice on developing a network of medical, nursing, and social work services, including the primary health care team, to provide any necessary aftercare and follow up. This is where present arrangements are weakest.

This sensible report takes account of the changing medical situation and advocates simple policies for seeing that patients are properly evaluated and treated. Purged of its overzealous organisational urgings, as it surely will be in practice, it deserves widespread support. I hope that it will come to be recognised that these patients need treatment not just to prevent later suicide but because they are presently distressed and suffering.

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1 Department of Health and Social Security. *The management of deliberate self-harm*. London: DHSS, 1984. (Health Notice HN(84)25.)
2 Ministry of Health. *Hospital treatment of acute poisoning*. London: HMSO, 1968.
3 Gardner R, Hanka R, Roberts SJ, Allon-Smith JM, Kings AA, Nicholson R. Psychological and social evaluation in cases of deliberate self-poisoning seen in an accident department. *Br Med J* 1982;**284**:491-3.
4 Gardner R. *The overdose patient—assessment and management*. Cambridge: Addenbrooke's Hospital, 1983.
5 Catalan J, Marsach P, Hawton KE, Whitwell D, Fogg J, Bancroft JHJ. Comparison of doctors and nurses in the assessment of deliberate self-poisoning patients. *Psychol Med* 1980;**10**:483-1.
6 Gibbons JS, Butler J, Urwin P, Gibbons JL. Evaluation of a social work service for self-poisoning patients. *Br J Psychiatry* 1978;**133**:111-8.
7 Royal College of Psychiatrists. The management of parasuicide in young people under sixteen. *Bulletin of the Royal College of Psychiatrists* 1982;**6**:182-5.

Functional diarrhoea: the acid test

Many physicians dislike making the diagnosis of functional diarrhoea—the painless diarrhoea variant of the irritable bowel syndrome. They view the diagnosis as one made by excluding all likely organic disorders; indeed, they may be tempted to regard it as a diagnosis of last resort made by a doctor who is intellectually destitute.

Certainly there are so many organic causes of diarrhoea that the physician often worries that he may have missed one. But the temptation and this worry are based on ignorance—ignorance of the commonness of chronic painless diarrhoea. Nearly 4% of apparently healthy people questioned in Bristol and Gosport admitted that their stools were frequently loose and passed with urgency,[1] while functional diarrhoea was the main diagnosis in over 5% of 2000 patients referred to gastroenterology clinics in Bristol.[2] The outpatient investigated in hospital for persistent loose stools is most likely to have functional diarrhoea—after inflammatory bowel disease has been excluded.

For the doctor the difficult question is always how far to investigate the patient. No investigations except sigmoidoscopy and rectal biopsy are needed when the patient is under 40, has had diarrhoea off and on for many years, when his symptoms are closely related to stress and anxiety, and when his weight is steady and his stool is free of occult blood. Many patients need a few investigations and a few need many; clinical judgment must decide. The severity of the diarrhoea gives some guidance. Patients with functional diarrhoea do not get dehydrated or hypokalaemic, and their stool weight is seldom more than 200 g a day in women or 300 g a day in men.[3,4] Some patients, especially women, actually have normal sized stools but are troubled by urgency of defecation and feelings of incomplete evacuation.

Food intolerance, especially to wheat, corn, and dairy products, seems to be a common cause of functional diarrhoea in Cambridge,[5] but this is yet to be confirmed in other centres. Nevertheless, it is reasonable to try an exclusion diet in selected cases.

While he is thinking about the diagnosis the physician must ask himself two questions. Firstly, is the diarrhoea self induced? Laxative abuse is probably the most common cause of obscure diarrhoea, at any rate when there is hypokalaemia.[6] The personality of the patient will usually give a clue to this. The second question is whether the cause might be bile acid induced diarrhoea. The laxative properties of bile have been known and exploited for hundreds of years.[7] Dried ox bile was a popular laxative until the present century, and even today preparations containing extract of ox bile are available over the counter. These laxative properties are due to the dihydroxy bile acids—deoxycholate and chenodeoxycholate. Some authors have argued that bile acids are the body's "built in" preventive of constipation.[8] They certainly promote water and electrolyte secretion in the large intestine,[9] and there is some evidence that the colon of patients with the irritable bowel syndrome is excessively sensitive to their action.[10]

Spontaneous bile acid diarrhoea occurs whenever the terminal ileum fails in its job of reabsorbing bile acids so that the amount of bile acid escaping into the colon increases. The most important causes are extensive Crohn's disease and surgical resection of the ileum. In the early 1970s occasional patients with structurally normal intestines were found to have malabsorption of bile acids and diarrhoea.[11,12] Previously these patients had been labelled as having functional diarrhoea, but with the cause uncovered effective treatment proved possible with cholestyramine, a resin which binds and inactivates bile acids.

Until recently no simple test had been available to detect malabsorption of bile acids and physicians had to resort to a therapeutic trial with a bile acid binding agent. They may now use SeHCAT, a radiolabelled bile acid analogue which emits γ rays and so can be counted easily with little or no handling of stools. In a recent issue Merrick and his colleagues reported the use of SeHCAT to confirm that bile acid malabsorption is frequent in patients whose chronic diarrhoea had until then been put down to the irritable bowel syndrome.[13]

This is a useful reminder to doctors to think of idiopathic malabsorption of bile acids in all patients with chronic diarrhoea of unknown cause. But it is far from certain that it is a disease in its own right; the defect of bile acid absorption may be non-specific. Patients with functional diarrhoea tend to have rapid small bowel transit,[4] and in some there may simply not be enough time for bile acids to be absorbed in the terminal ileum. Bile acid diarrhoea often responds to standard antidiarrhoeal drugs such as loperamide, which slow down transit in both the small and the large bowels.[14]

The "logical" treatment for bile acid diarrhoea is a bile acid binding resin such as cholestyramine, but in its usual form this has disadvantages. Cholestyramine is unpalatable, and it interferes with the absorption of lipids by inactivating bile acids in the proximal small intestine. These objections