At the end of the 1970s the MRC was funding nearly 400 students at a cost of about £800 000 (around 1% of its budget). A few students were funded by local education authorities, "soft money," or themselves. The MRC had started supporting students in 1964 after prompting from the then Ministry of Education and in its first year supported just 12. Once this source of funding was advertised the numbers grew rapidly, reaching a peak in 1979-80, and the proportion doing the degrees became similar in all medical schools, though each medical school was left entirely to itself to decide which students would be supported.

In 1980 the MRC decided that the money it was spending on these students was growing too fast, and it capped the number at 380. Since then their numbers have been reduced by 10% a year. Some 304 will be supported in 1986—or perhaps none at all. The question mark is due to a decision by the Exchequer and Audit Department (now the National Audit Office) to question the council's funding of these students. It is arguing not only that the MRC should not be paying this money because it is not for research but also that it is insufficiently discriminating in where the money goes. The MRC is now waiting to see whether its reply to this charge will be accepted. If it is not the matter will be referred to the Public Accounts Committee, and the council could be told to stop the funding.

The MRC responded to the challenge by informing all deans that it would have to start discriminating between schools, departments, and courses. Conditions attached to funding would include the schools explaining how students will be selected, the courses including some research that would have to be written up, and reports being produced on the outcome of each award. The council also announced its intention to monitor results. Clearly such a system would consume time and money that could be better spent.

The MRC has also looked for other ways of funding the students—and drawn something of a blank. For the Department of Education and Science to take over the funding, even if the will and the money were available, would require legislation, and hard pressed local education authorities, the other potential source, are unlikely to support many students when the grants remain discretionary.

The Kafkaesque lunacy of all this is that no one seems to dispute the value of these degrees. A paper we publish today (p 1646) shows not only that 40% of graduates who had done intercalated degrees in pathology in Edinburgh went into academic medicine but also that they did better in the rest of the academic curriculum than a group of students of matched ability who did not do intercalated degrees. A study from Queensland of 99 doctors who had taken degrees in basic medical sciences showed that they were 20 times more likely than their controls to go into full time research, four times more likely to graduate with an honours degree, and much more likely to have published papers in refereed scientific journals.¹ Further British research has also shown that of a sample of 262 doctors in research orientated posts almost a third had taken intercalated degrees.²

Many of these self selected doctors might, of course, have gone into academic medicine anyway, but much of the value of these courses is not quantifiable in these ways. Most of those who do intercalated degrees (and I was one) speak very positively of the experience. "That was the year," they say, "that I learnt to think and to question and to find out things for myself." That is where the degrees may be so important—they prepare students for a lifetime of learning. Many are driven into intercalated degrees by the unremitting dullness of preclinical courses, but many emerge newly inspired and

ready not only to soak up clinical medicine but also to push forward its boundaries.

For these degrees to be lost simply for lack of somebody with common sense in government would be a tragedy. Somebody—probably the education departments—must pick up the bill.

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Adolescent suicide: preventive considerations

Suicide in prepubertal children is rare, and the rates appear stable over time.¹⁻³ By contrast, the rates of suicide and attempted suicide in older teenagers have increased appreciably during the past 30 years in most countries where accurate statistics are kept, whereas in older people they have remained stable or declined.^{3,4} In 1984 in England and Wales 112 people under 20 killed themselves.⁵ In Japan, with roughly twice the population, 572 under 19 year olds killed themselves.⁶ In the United States suicide is now the third most common cause of death in 15-24 year old white young men.⁷

In most countries suicides by men outnumber those by women by at least 2:1, although attempted suicide is far commoner in girls. The methods used by boys and men are more violent (firearms, jumping from high places, and hanging) and more likely to result in death. Women and girls favour overdosing, where resuscitation is more likely.³ The two groups, suicide and attempted suicide, vary in many ways but 46% of young suicides (under 14) had made threats or attempts before the act,¹ and in the follow up study of youngsters aged 10-20 who had attempted suicide 10% of the boys and 3% of the girls eventually killed themselves, usually within two years.⁸ Repetition rates in a later study were much lower (0.24%)⁹ probably because the earlier one included all forms of attempted suicide.

The impact on those most closely connected with a young person who kills himself are profound. Family, teachers, friends, and workmates experience guilt and stigma. Reactions of denial and blame are common. Mourning may be more difficult for the family because friends avoid talking to them about such deaths.³

If we are to prevent or reduce the number of adolescent suicides we must understand the causes. The problems of studying causation when of necessity the study must be retrospective have been summarised by Hawton.³ Among adults committing suicide 90% have psychiatric disorders, especially depression and schizophrenia.¹⁰ The rate in young people is probably less than 50% but there is often a family history of both psychiatric disorder and suicidal behaviour.¹¹ There may therefore be a genetic predisposition to depression or other psychiatric disorders. In addition the behaviour (taking pills, attempting suicide) may be imitated. Finally, living with a mentally ill parent, especially a single parent, may be intolerable. Most young people who kill themselves have high levels of family disruption and in addition may

have suffered physical abuse or possibly sexual abuse. A recent study using controls has come from Boston where 159 youngsters attempting suicide were carefully matched and the registry of the department of social services searched for the names in both groups.¹² Prior contact with social services was three to six times more likely for probands of both sexes than controls. Most of these young people would have experienced child physical abuse or neglect.

A follow up study of 201 families in which intrafamilial sexual abuse had been substantiated found that 11 had experienced suicidal attempts within 2½ years, in most cases by the daughter-victim.¹³ There was no control group. Since boys more commonly kill themselves than girls, sexual abuse is less likely to be a major factor in adolescent suicide as opposed to attempted suicide.

In Japan and elsewhere educational pressures may lead young people to experience hopelessness and lowered self esteem when they fail. Possibly teachers and schoolmates may increase the sense of isolation.⁶ Finally, young people may be unable to establish themselves in work, love, or living apart from their family and this may heighten the sense of failure. This may account for the high suicide rate in young married Hindu women in Fiji.¹⁴ Probably many of these factors will interact to produce the final suicidal behaviour.

What can doctors and other health professionals contribute to the primary prevention of suicide in young people? Probably their most important contribution is to recognise the early signs of mental illness, especially depression and schizophrenia. Depression may present with school failure after normal achievement, hypochondriasis, or social withdrawal more often than with complaints of depression or anxiety by the youngster. If in addition the family background is disturbed, there is a family history of psychiatric illness, or child abuse is suspected or known then referral for specialist help may avert tragedy. The fact that prepubertal children and black American adolescents, both of whom may have high levels of family and social stresses, have lower suicide rates points to the possibility that protective factors may be operating.²

Secondary prevention implies the prevention of subsequent, possibly fatal, suicidal attempts in youngsters after their first attempt. This requires the energetic, persistent, and effective treatment of all youngsters attempting suicide.^{3,15,16} If they are living with their families the latter will need to be included in treatment. Although family therapy seems an obvious choice, no treatments have been adequately evaluated in this age group. In my own practice most young people who have taken an overdose are given a card enabling them to readmit themselves to the paediatric ward without question. This approach awaits evaluation but so far has been used responsibly.

Finally, tertiary prevention implies the prevention of avoidable handicap in the members of the family and wider network of a young person who commits suicide. Probably the normal techniques of bereavement counselling are needed here, and particularly when siblings are present, but professional help may be needed to prevent morbid grief reactions in the survivors.^{17,18}

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Non-steroidal anti-inflammatory drugs and the kidney

Non-steroidal anti-inflammatory drugs have been an important part of our therapeutic armament for many years. It was only in 1971, however, that these drugs were shown to work by inhibiting prostaglandin synthesis.¹ Since then the number of non-steroidal agents has increased very considerably, the indications for their use have widened—and increasing attention has been paid to the risk benefit ratio. In some cases the side effects have led to a drug's product licence being withdrawn or modified (for example, phenylbutazone, zomepirac, fenclofenac, and the Osmosin preparation of indomethacin). All doctors are well aware that non-steroidal anti-inflammatory drugs produce some side effects such as gastric erosions, but they may not realise that more severe side effects, in particular renal damage, may also occur.

Isolated cases of renal failure have been reported for some time,^{2,3} but recognition of renal problems has been slow—possibly because the response of the kidney to non-steroidal anti-inflammatory drugs is different in people with abnormal physiological function from that in normal people, and initial studies at least tend to be performed in normal volunteers. Prostaglandins are synthesised in the kidney, and the principal prostaglandins E₂, D₂, and I₂ (prostacyclin) are powerful vasodilators. In normal conditions these prostaglandins do not play a big part in the maintenance of the renal circulation, and drugs such as indomethacin have little effect on the kidney.^{4,5} In a patient with increased amounts of vasoconstrictor substances such as angiotensin II, nor-adrenaline, or antidiuretic hormone in the blood stream, however, vasodilatory prostaglandins become important in maintaining renal blood flow.⁶ Clinical conditions such as congestive cardiac failure, cirrhosis of the liver with ascites, diuretic induced volume depletion, salt restriction, and the nephrotic syndrome will cause the release of vasoconstrictor substances to maintain blood pressure. In those circumstances inhibition of prostaglandin synthesis (cyclo-oxygenase inhibition) by non-steroidal anti-inflammatory drugs may cause unopposed renal arteriolar constriction, leading to acute renal insufficiency or renal tubular necrosis. This is probably the most common form of renal toxicity due to non-steroidal drugs.⁷⁻⁹

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