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 time 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 E_2, D_2, and I_2 (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, noradrenaline, 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]

Other factors predisposing to acute renal insufficiency induced by these drugs are advanced age, renovascular disease, systemic lupus erythematosus, and gout. All non-steroidal anti-inflammatory drugs have been implicated in causing acute renal insufficiency, but indomethacin has been cited most commonly, perhaps owing to its greater use.[10] The drug sulindac has less effect on renal prostaglandin synthesis than other non-steroidal anti-inflammatory drugs,[11] but it may nevertheless inhibit renal prostaglandin synthesis[12] and cause renal failure.[10] Fortunately this acute renal insufficiency is usually reversible on stopping the non-steroidal anti-inflammatory drug.

The second form of renal toxicity that may be induced by non-steroidal anti-inflammatory drugs is hyperkalaemia. Prostaglandins directly stimulate the release of renin, and thus the inhibition of cyclo-oxygenase by non-steroidal anti-inflammatory drugs leads directly to a hyporeninaemic hypoaldosteronism with subsequent hyperkalaemia.[3 13] Stopping the drug quickly reverses the hyperkalaemia, but the serum potassium concentration may need to be monitored in patients treated with non-steroidal anti-inflammatory drugs, particularly those having potassium supplements or potassium sparing diuretics.

Renal papillary necrosis is well recognised with analgesics such as phenacetin,[14] but it may also result from long term use of non-steroidal anti-inflammatory drugs such as aspirin and indomethacin.[9] Acute interstitial nephritis and the nephrotic syndrome are two further renal lesions that have been associated with the use of non-steroidal anti-inflammatory drugs.[15 16] The mechanism responsible here is uncertain—it may represent an allergy to the drug mediated through cytotoxic T cells,[15 17] and the mean duration of exposure to the drug before diagnosis has been six to eight months.[7] Renal biopsy specimens usually show a minimal change glomerular lesion with lymphocytic infiltration of the interstitium. Although many non-steroidal anti-inflammatory drugs have been implicated in this condition there does seem to be an undue preponderance of cases associated with fenoprofen.[7 9] In most cases the prognosis is good after stopping the drug, though treatment with steroids in high doses has been used to hasten recovery.[17]

As if this catalogue of renal disease induced by non-steroidal anti-inflammatory drugs were not enough, Adams *et al* have added another problem.[18] They reported that out of 17 patients presenting over three years with renal disease induced by these drugs, six had chronic renal failure. In contrast to the remaining 11, the renal failure in these six patients did not improve when the drug was stopped. The lesion was papillary necrosis in one patient and interstitial fibrosis in the remaining five; this might possibly be the end stage of uncorrected interstitial nephritis. These authors suggested that the non-steroidal anti-inflammatory drugs with long plasma half lives were the ones most likely to cause this problem, but this remains speculation.[18]

What is clear is that many patients taking non-steroidal anti-inflammatory drugs are at risk of developing renal disease and that this risk is greatest in elderly patients; patients with cirrhosis, congestive heart failure, renovascular disease, or gout; and patients who are volume depleted or salt depleted. The largest group in this last category are those patients taking diuretics, whether loop diuretics such as frusemide or milder diuretics such as thiazides. Particular care needs to be taken when giving non-steroidal anti-inflammatory drugs to patients taking diuretics, since many of these drugs inhibit the diuretic response to loop diuretics[19] and the hypotensive response to thiazides[20] in addition to their adverse effects on renal function.

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Confidential inquiry into perioperative deaths

A confidential inquiry into all perioperative deaths in three regions seemed impossible just over a year ago, but it started before Christmas in the South Western, North East Thames, and Northern regions. The inquiry will determine the incidence of particular causes of death and the part played by the anaesthetic, the surgery, and any other causes. Standards of care and clinical and organisational deficiencies will also be investigated. Funded by the Nuffield Provincial Hospitals Trust and King Edward's Hospital Fund for London, the inquiry is a joint effort between anaesthetists and surgeons—between the Association of Anaesthetists and the Association of Surgeons of Great Britain and Ireland.

After consultation with all the relevant clinical organisations, a working party prepared a protocol that received general approval. This scheme was then tested in Darlington, Exeter, and the Middlesex Hospital. A final questionnaire was then designed to show broadly what was done in surgical and anaesthetic management. It was intended neither as a detailed investigation nor to show the virtues of a particular approach but rather to describe the delivery of surgical and anaesthetic care in Britain today.