The epidemiology of malpractice

Adverse events common, negligence cases rare

Roughly one in 25 patients admitted to hospital experiences harm as a result, and as many as a fifth of deaths in hospital may be caused by something going wrong. About a quarter of these adverse events result from negligence. These figures are derived from the only two large population studies of the incidence of adverse events in hospital, both of them American.[1,2] If they apply in Britain then about 300 000 patients a year may experience an adverse event while in hospital, 45 000 may die in part because of the event, and 75 000 cases of potential negligence may arise from hospital admissions. These numbers do not include the problems that arise from outpatient encounters or consultations in general practice. Clearly, few cases of negligence in Britain result in a claim and even fewer in compensation.

The two American studies have both demanded huge amounts of work and even larger amounts of political finesse. Doctors do not want their dirty linen catalogued. Yet studies of the epidemiology of malpractice are just as important as studies of the epidemiology of disease—and for the same reasons: they allow insights into causative factors and ways of reducing the harm. In particular, they help with working out whether errors happen to all practitioners or are concentrated among the few—the "bad apples" whom we hear so much about. Every country needs such a study. At least one funding body would probably be willing to finance a British study, but nobody has had the stomach to make it happen. Britain has, however, for many years had its inquiries into maternal deaths, and the Confidential Enquiry into Perioperative Deaths found that 7% of deaths within 30 days of operation were caused solely by the surgery and that anaesthesia played some part in 14% of deaths.[3]

The latest contribution to the epidemiology of malpractice is the 5 cm thick report from the Harvard Medical Practice Study of malpractice in New York State.[1] Interestingly, its results are remarkably similar to those from the only other population study—one done in California in the 1970s.[2] The Harvard study looked at 30 121 records of admissions to 51 hospitals in 1984 and found 1133 (3·8%) adverse events arising from medical mismanagement; 280 (24·7%) were judged to be due to negligence. For all hospital admissions in New York State these figures would have meant almost 99 000 adverse events and just over 24 000 cases of negligence. During 1984 between 2967 and 3888 malpractice claims were filed, meaning that about eight times as many patients were injured through negligence as filed a claim—and 16 times as many as received compensation. In California in 1974 the researchers found 959 (4·6%) "potentially compensable events" among 20 864 records, about 17% of which were judged to have been caused by negligence. About 1 in 10 of the negligent injuries resulted in a claim, and 40% of the claims resulted in a payment. These results are, remember, from the litigious United States. In Britain the proportion of patients injured through negligence who receive compensation is almost bound to be much lower.

Over half (57%) of the adverse events recorded in the New York study led to only minimal and transient disability, but 14% of patients died in part because of their adverse event and another 9% had disability lasting longer than six months. In the Californian study four fifths of adverse events were categorised as temporary, but a tenth were fatal. In both studies negligent events resulted in greater disability, and over half of the deaths in the New York study arising from adverse events resulted from negligence.

In both studies the risk of an adverse event increased with age. In New York those aged over 65 had twice the chance of sustaining an adverse event of those aged 16-44. There were no gender differences in rates of adverse events, but blacks had higher rates. Overall rates of adverse events varied 10-fold among hospitals, and the rate was four times higher in hospitals in New York City than in non-metropolitan hospitals. About half the adverse events occurred in patients undergoing surgery, but only about 14% of the adverse events in the operating room resulted from negligence compared with over 70% of those in the emergency room. Over a third of the adverse events were categorised as performance errors (three quarters of which were technical errors), while a fifth were prevention errors (almost half of which were failing to prevent falls), 14% were diagnostic errors, and 9% errors in drug treatment.

Because of their interest in the economics of a no fault scheme the Harvard researchers calculated that the value of the losses (past and future) to all patients injured in New York hospitals in 1984 was $894m (in 1989 dollars). In 1988 New York doctors paid $850m in insurance premiums, and self insured hospitals paid at least another $150m. The researchers calculated that a no fault scheme covering wage losses and medical losses would cost about $335m, suggesting that there is considerable room within current expenditure for funding a scheme. These data have been used by New York State health commissioner David Axelrod to support his call for a state no fault system for medical injury.[4]

These are important data, and the researchers are to be congratulated on jumping the many hurdles necessary to collect them. The report contains a mass of detail that will be helpful to health professionals and managers everywhere, but because the overall number of adverse events was small the study does not help much with evaluating whether many of the adverse events result from particular doctors, although the concentration in particular hospitals is clear. Another recent American study, this time from Florida (the state with the highest malpractice insurance premiums), has, however, looked at this question and concluded that the "bad apple theory" has merit.[5]

The Florida researchers examined almost 6000 claims made in 1975-80 and found that 85% of the doctors in medical specialties, two thirds of those in obstetrics or anaesthetics, and half of those in surgical specialties had not had a claim made against them. But in all three groups many of the payments made resulted from the actions of few doctors—for example, in the medical group 85% of payments were made on behalf of 3% of doctors. The researchers looked at the training, age, gender, and place of practice of the doctors but found no strong patterns.

In pursuit of bad apples the Florida researchers discovered that twice as many doctors in the high payment group as in the no payment group had had complaints filed against them with the Florida Department of Professional Regulation. Another central finding was that doctors' experience with claims in 1975-80 predicted their experience in 1981-3. Here then is some evidence for the bad apple theory, but an important problem with this study is that it did not adjust for risk. Thus

doctors may have had more claims because they took on more difficult cases.

Debate will continue on whether a small group of incompetent doctors produce much of the malpractice problem, but the Office of Technology Assessment has suggested that malpractice information should be collected and used for quality screening by hospitals, licensing boards, and others (perhaps even patients).[6] Indeed, a national database of doctors who have lost malpractice suits went into operation on 1 September (22 September, p 569): hospitals are required to check the database before hiring doctors, but the data are not yet available to patients. The Florida researchers argue that this would be premature, but research in the epidemiology of malpractice must continue so that we have data on which to base such proposals.

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Regular Review

Interventions in chronic renal failure

Treatment may slow progression in some cases

Studies of chronic renal failure in a variety of animal models suggest that once the number of functioning nephrons is reduced to a critical proportion a self perpetuating and progressive deterioration in renal function develops, leading eventually to terminal uraemia. One of the main planks in this explanation is the hyperfiltration hypothesis.

This hypothesis postulates that loss of nephrons owing to different causes leads to changes in glomerular haemodynamics in the remaining functioning nephrons with a rise in glomerular capillary pressure (glomerular hypertension) and an increase in filtration rates in the individual nephrons (hyperfiltration). These changes seem to produce increasing proteinuria and progressive glomerulosclerosis culminating in terminal uraemia due to a progressive reduction in the numbers of functioning residual nephrons.[1-5] It has proved possible in experiments in animals to delay or prevent the development of progressive glomerulosclerosis by ameliorating the haemodynamic changes—either by reducing protein intake or by treatment with angiotensin converting enzyme inhibitors. The converse has also been shown: either a high protein diet or treatment with glucocorticoids increases both the glomerular hypertension and the degree of glomerulosclerosis.

Some observations in these animal models do not, however, support a simple causal relation between the abnormal glomerular haemodynamic changes and progressive glomerulosclerosis. Other mechanisms have been suggested to account for the progressive nature of chronic renal failure. The precipitation-calcification hypothesis postulates that an excess of phosphate causes deposition of calcium phosphate and resultant interstitial and tubular damage.[6-8] Hyperlipidaemia may be another factor; accumulation of lipid within the mesangial cells may result in the development of focal glomerulosclerosis.[9,10] Again in animals treatment of hyperlipidaemia has reduced the degree of albuminuria and the incidence of glomerulosclerosis.[11]

It is a big step, however, to attempt to extrapolate the results of the mass of research work in animal models of chronic renal failure (mainly in the rat) to chronic renal failure in humans. Firstly, there is little evidence in humans that reduction of renal mass compromises the function of the remaining nephrons. Long term follow up of donors of kidneys for transplantation has shown only a slightly increased incidence of mild hypertension and proteinuria.[12] In follow up studies of more than 10 years the function of the remaining kidney has not deteriorated.[13] A long term follow up of 32 patients for a mean of 23 years after unilateral nephrectomy in childhood (for various reasons) showed no evidence of an increased incidence of renal impairment or hypertension.[14]

Clinical studies have confirmed that not all patients with chronic renal failure progress inexorably towards terminal uraemia. In a recent study progression of chronic renal failure was analysed in 108 patients by plotting the slope of the reciprocal of the plasma creatinine concentration against time.[15] Seventy patients showed a pattern of linear deterioration and 15 showed non-linear deterioration. In 23 patients, however, the chronic renal failure was stable. Progressive renal failure was usual in patients with chronic glomerulonephritis, diabetic nephropathy, reflux nephropathy, and polycystic kidney disease. By contrast, most of the patients with hypertensive nephrosclerosis, analgesic nephropathy, and renal impairment after acute renal failure were stable. Among patients with linear deterioration the rate was faster in those with chronic glomerulonephritis and diabetic nephropathy than in those with reflux nephropathy and polycystic kidney disease. The underlying renal disease seems, therefore, to be important in determining progression of chronic renal failure and also the rate at which this deterioration occurs. Clearly clinicians need accurate methods of measuring renal function in order to determine whether chronic renal failure is stable or is progressively deteriorating. Plots of the reciprocal of the serum creatinine concentration against time have frequently been used for this purpose.[16,17] However, the validity of this method has been seriously questioned, as has the use of measurement of creatinine clearance.[18,19] Isotopic methods of determining the glomerular filtration rate are to be preferred.

Deterioration in chronic renal failure may be due to continuing activity of the underlying renal disease—as in systemic lupus erythematosus. Several other factors that may result in acute deterioration in chronic renal failure need to be sought and excluded. Their prompt identification and correction may enable renal function to improve and even stabilise at the previous level. These factors include acute sodium and water depletion due to vomiting or diarrhoea, or