patients admitted to hospitals in New York State in 1984, estimated that 1% of admissions were complicated by an adverse event due to negligent care. Fewer than 1 in 50 of these, however, led to malpractice claims.⁵ Furthermore, out of the 47 malpractice claims that were identified in the study population, only eight were clearly associated with substandard care. This is consistent with a review of medico-legal cases in the United Kingdom that found that 44 out of 100 claims were not associated with any serious clinical error.⁶ It seems, therefore, that many patients who are injured as a result of poor care do not sue their doctor. Paradoxically, many patients who start legal proceedings do so without clear evidence of error. Unsurprisingly, communication and record keeping, cornerstones of clinical risk management, are identified as key issues in minimising litigation.⁷

Does managing clinical risk work? The United States has considerable experience of risk management techniques, but their effect on the cost of negligence claims has rarely been subjected to formal evaluation.⁸ Such evaluation is difficult because claims are rare and resolving them can take a long time. A comparison of 40 hospitals in Maryland found that hospitals with specific risk management programmes had fewer claims made against them than those that did not.⁹ A study in California assessing incident reporting systems found that claims were resolved more rapidly if an incident report was made.¹⁰ This was despite the incident reports involving more serious injuries. Neither study was randomised, and confounding factors may have accounted for the apparent effect of risk management. In the Maryland study hospitals that provided better quality care may also have been more likely to have a risk management programme; in the Californian study characteristics of events that were not reported (for example, a late complication after hospital discharge) may have made claims more difficult to resolve. Given the substantial differences in both the health care and legal systems between the United States and the United Kingdom, the findings may be of limited relevance to the NHS.¹¹

Despite the lack of evidence of the effectiveness of clinical risk management, any attempt to minimise malpractice claims and improve patient safety must be taken seriously. So how should doctors and managers respond to the intuitive appeal of clinical risk management? Given its overlap with clinical audit it should be closely linked to existing audit procedures. Risk management programmes that are set up should be carefully evaluated. The NHS Executive has urged that purchasers should assess the effectiveness of a clinical treatment before buying it.¹² The same criteria should apply to management interventions.

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Treating hypertension after stroke

Hold off for 10 days unless hypertensive encephalopathy, aortic dissection, or intracerebral bleeding is present

Hypertension is the most important potentially reversible risk factor for cerebrovascular disease in all age groups. A continuous relation exists between increasing blood pressure and the likelihood of stroke, such that a 7 mm Hg increment in diastolic blood pressure is associated with a doubling of the relative risk of stroke.¹ The preventive role of antihypertensive treatment in reducing morbidity and mortality from stroke is well established: treatment that produces a long term reduction in diastolic blood pressure of 5-6 mm Hg results in a 35-40% fall in the risk of stroke.^{2,3}

About four fifths of patients with stroke have raised blood pressure on admission to hospital and about one third have a history of hypertension.⁴ Raised blood pressure usually falls spontaneously within a few days⁵: 10 days after an ischaemic stroke two thirds of patients are normotensive.⁶ Doctors are therefore faced with the problem of deciding in whom and when to treat raised blood pressure.

There is conflicting evidence about the prognostic significance of hypertension immediately after acute stroke. Using multivariate analysis, Carlberg and colleagues found that previous hypertension most strongly predicted raised

initial blood pressure in patients admitted to an acute stroke unit.⁷ Hypertension preceding stroke is, however, not invariably associated with an adverse prognosis in terms of either early fatality or functional recovery.⁸⁻¹⁰ Although early studies suggested that hypertension immediately after stroke was associated with increased fatality and poor functional recovery,^{11,12} more recent work has suggested that raised blood pressure early after cerebral haemorrhage or infarction is related to 30 day fatality only in patients with impaired consciousness,¹³ which is the single most important adverse prognostic factor for survival after stroke.¹⁴ In addition, Levy and colleagues found no association between blood pressure at admission and recovery in a series of patients with acute stroke.⁸ These results explain the controversy surrounding the early use of hypotensive treatment after acute stroke.^{15,16}

In health, cerebral circulation is kept constant by autoregulation at a mean systemic arterial pressure (diastolic + (systolic - diastolic)/3) between 55 and 125 mm Hg. This capacity to regulate cerebral blood flow is reduced in elderly people.¹⁷ In patients with chronic hypertension autoregulation begins at a higher systemic pressure, and below

this level there is a direct relation between cerebral blood flow and mean arterial pressure.

After an ischaemic stroke, the normal mechanisms of cerebral circulatory autoregulation are impaired. Perfusion in the ischaemic penumbra, the potentially viable neural tissue surrounding the infarcted area, becomes pressure dependent.¹⁸ A rise in systemic arterial pressure may therefore be an adaptive response to maintain the blood flow to this vulnerable area. For this reason, hypotension after stroke should also be sought and treated to avoid further anoxic cerebral damage in parts of the brain with marginal circulation.

The decision to treat hypertension immediately after ischaemic stroke cannot be taken lightly, especially given the well known hazards of cerebral hypoperfusion from overzealous early blood pressure reduction.^{19,20} It is important to identify those few patients with hypertensive encephalopathy or aortic dissection whose initial clinical presentation may mimic acute cerebral infarction and who obviously require urgent management of their hypertension. But the more familiar clinical picture is of an elderly patient, often with a history of hypertension, who has substantially raised blood pressure in the first few days after admission to hospital with a stroke. Should such patients receive antihypertensive treatment; which drugs should be used; and how long after the stroke should treatment begin?

No consensus has emerged regarding treatment of hypertension in the acute phase after stroke. Marked hourly fluctuations in blood pressure have been shown in the first 24 hours,²¹ and in most patients hypertension will settle gradually without specific treatment.^{5,6} Furthermore, patients with acute stroke show extreme hypersensitivity to hypotensive treatment⁴ especially if they are elderly or if the cerebral autoregulatory curve has been shifted as a result of pre-existing longstanding hypertension. No randomised controlled trials looking specifically at antihypertensive treatment in acute stroke have been reported. Hypotension was recognised as a predictable side effect of treatment in studies examining the potential cerebral protective effects of β blockers and nimodipine in acute ischaemic stroke; these studies showed no beneficial effect on mortality or morbidity for these drugs.²²⁻²⁴

Extreme and persistent hypertension is more likely with intracerebral haemorrhage than with infarction.²⁵ Once again, no randomised trials have been performed that would allow clear clinical guidelines to be drawn up. Nevertheless, very high blood pressures (systolic pressure > 200 mm Hg; diastolic pressure > 120 mm Hg) associated with intracerebral bleeding (established by computed tomography or magnetic resonance imaging) should be treated early, to limit the development of vasogenic oedema due to disruption of the blood-brain barrier in the ischaemic area around the haemorrhage.²⁰

Much stroke care occurs in the community, where computed tomography is not available, and a more pragmatic approach is necessary. It may therefore be prudent not to lower the diastolic blood pressure to below 110 mm Hg soon after acute stroke.²⁶ In North America, the treatments advocated for blood pressure reduction soon after stroke tend to be short acting vasodilator drugs such as sodium nitroprusside and labetalol.^{4,20} Careful monitoring of blood pressure is required with these agents, and they may therefore be difficult to use outside an intensive care unit or designated stroke unit. Current British practice favours the use of short acting oral vasodilators such as nifedipine.²⁷ Such agents may be given sublingually or through a nasogastric tube if swallowing is impaired.

No consensus exists regarding the optimal timing to initiate long term hypotensive treatment in patients with persistently raised blood pressure a few days after a stroke. But because in

most patients blood pressure has stabilised within seven to 10 days, many doctors start treatment at about this time in those remaining hypertensive.⁶ Antihypertensive treatment that has been initiated in hospital should certainly be reviewed after discharge. In a recent study, two thirds of patients who left hospital after recovering from stroke had significantly raised blood pressures throughout the first year's follow up.²⁸

Although trials in older adults have shown the efficacy of antihypertensive treatment in reducing morbidity and mortality from first ever stroke,^{29,30} the long term benefits of secondary stroke prevention by treating hypertension are uncertain and extrapolating from studies of primary prevention may be inappropriate. Few data on the role of blood pressure reduction in decreasing the risk of recurrent stroke are available from clinical trials. In a small randomised study, treatment of hypertension after stroke failed to alter the rates of recurrence.³¹ Trials of long term secondary stroke prevention by treating hypertension with angiotensin converting enzyme inhibitors or diuretics are currently under way in China and Australasia. Much work remains to be done regarding the behaviour of hypertension after stroke and the effects of its treatment.

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