INTRODUCTION
Blood pressure is the single most important modifiable risk factor for stroke. Key epidemiological data about the relationship between blood pressure and stroke come from both prospective observational (cohort) studies and randomised controlled trials. Cohort studies provide information about the effects of prolonged blood pressure differences between groups of people (MacMahon et al. 1990), while clinical trials provide evidence about the effects of short-term reductions in blood pressure (Collins et al. 1990). Cohort studies in both Western (Prospective Studies Collaboration 1995) and Eastern (Eastern Stroke and Coronary Heart Disease Collaborative Research Group 1998; Asia Pacific Cohort Studies Collaboration 1999) populations have established a strong and direct association between the risk of first-ever stroke and level of blood pressure, with no detectable lower level of blood pressure below which the risk of stroke does not continue to decline (Fig. 1). A similar association is evident among patients who have had a stroke or transient ischaemic attack (TIA) (Rodgers et al. 1996). Thus, blood pressure predicts stroke recurrence as well as stroke occurrence (Fig. 2).

Most of the data on the effects of blood pressure lowering therapy come from trials of the primary prevention of stroke, confirming beyond doubt the benefits of treatment with a wide variety of agents in preventing first-ever stroke in middle-aged men and women with and without ‘hypertension’. Moreover, the relative reductions in primary stroke rates of 35–40% in response to reductions of 10–12 mmHg in systolic blood pressure (SBP) and 5–6 mmHg in diastolic blood pressure (DBP) are remarkably in line with predictions based on the observational epidemiological studies.

THERAPEUTIC INTERVENTIONS
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Most of the data on the effects of blood pressure lowering therapy come from trials of the primary prevention of stroke, confirming beyond doubt the benefits of treatment with a wide variety of agents in preventing first-ever stroke in middle-aged men and women with and without ‘hypertension’. Moreover, the relative reductions in primary stroke rates of 35–40% in response to reductions of 10–12 mmHg in systolic blood pressure (SBP) and 5–6 mmHg in diastolic blood pressure (DBP) are remarkably in line with predictions based on the observational epidemiological studies.

An outstanding issue has, until recently, been whether it is also beneficial to lower blood pressure after the onset of stroke to improve long-term outcome and prevent recurrent stroke, and indeed other serious vascular events such as myocardial infarction (i.e. secondary prevention). In addition, whether the beneficial effects of treatment apply to an important patient subgroup, such as those with ‘normal’ or lower levels of blood pressure after a stroke. These questions are particularly pertinent to reducing the global burden of stroke. According to the World Health Organization Global Burden of Disease study (Murray & Lopez 1996), stroke was the second most common cause of death (4.4 million or 8.7% of deaths) after ischaemic heart disease (6.3 million or 12.5% of deaths), and the sixth most common cause of premature death and disability in 1990. However, stroke is predicted to rank fourth in terms of global disease burden by 2020 because stroke rates rise steeply with age and the world population is ageing rapidly, and adverse lifestyles are being adopted in developing countries. Because individuals with a history of stroke or TIA are at very high risk of recurrent stroke and other vascular events, and because three-quarters of all strokes occur in individuals with ‘normal levels’ of blood pressure, even modest reductions in blood pressure from widely applicable treatments could confer major absolute benefits on the incidence of stroke and other cardiovascular events. This paper reviews the evidence of the effectiveness of blood pressure lowering for the secondary prevention of stroke in the light of recent data from randomised trials, in particular the results of the Perindopril Protection against Recurrent Stroke Study (PROGRESS) Collaborative Group 2001.

LETTER TO THE EDITOR
Long-term management of blood pressure after stroke
A major role of your journal is to provide ‘jobbing neurologists’ like myself with didactic practical advice for everyday patient management. Whilst finding Craig Anderson’s review helpful (Practical Neurology, 2, 272–279), it fell short of my expectations in the section headed ‘How much should blood pressure be reduced?’ Despite the title I am really none the wiser. If the BP is persistently 190/110, how low should it go? What if it is only 130/80 – is a reduction to 120/75 satisfactory, or is 110/70 better? Is 100/60 too low? In such a situation, perhaps a suggestion for starting doses of indapamide and perindopril would be appropriate. A little more didactic advice would be much appreciated.

**RESPONSE**

Craig Anderson

Clinical Trials Research Unit, Department of Medicine, The University of Auckland, Private Bag 92019, Auckland, New Zealand; Email: c.anderson@ctru.auckland.ac.nz

Dr Hilton-Jones has raised some important issues. The epidemiological and clinical trials evidence is now quite robust. Blood pressure and stroke risk are directly and positively related – so the lower the blood pressure, the lower the risk of recurrent stroke. Ideally therefore we should aim for the lowest tolerable blood pressure in all patients with stroke, irrespective of their age, sex, pathological type of stroke, or other factors. Viewed simply, it has been argued that we no longer need to measure blood pressure because there is no ‘normal’ safe level. Rather, we should just introduce blood pressure lowering agent(s) until our patients feel dizzy on standing, and then reduce the dose! In clinical practice, though, the hazards of postural hypotension, particularly in older patients, are such that we would want to introduce treatment cautiously and to check the blood pressure levels to confirm response to therapy and any postural hypotension.

The starting dose and titration of medication clearly depend on the baseline blood pressure, the degree of blood pressure reduction expected, and the physical condition (or age) of the patient. I start with an ACE inhibitor and then add a diuretic if the patient is free of symptoms, and not on the basis of blood pressure levels, and with ‘intermediate strength’ doses as was used in PROGRESS. For the frailer older patient with stroke, I halve the starting dose and increase it much more slowly, whereas combination therapy can be introduced in the younger ‘hypertensive’ adult without much cause for concern.

The issue of blood pressure targets is more controversial. While it make little sense epidemiologically to have for example, ‘low’ (<140/90), ‘medium’ (140–160/90–100), or ‘high’ (>160/100) categories, they do help in the management of patients who generally like and expect to have their blood pressure monitored and to receive appropriate feedback. However, notwithstanding the random error associated with individual blood pressure readings, including the ‘white coat’ effect, the significance of ‘average’ blood pressure levels should be put in the context of the absolute risk of stroke in the individual patient. Aggressive management of vascular risk, including lowest possible blood pressure levels, is most appropriate in those at high overall risk of disease.