ing,” but would emphasize that those were trials of isovolemic hemodilution. Hypervolemic hemodilution offers greater opportunity to improve cerebral perfusion because of its positive effect on cardiac function. Isovolemic and hypervolemic hemodilution are two different methods aimed at augmenting reperfusion. Isovolemic hemodilution probably does not improve outcome. We feel that our study of hypervolemic hemodilution provides enough encouragement to conclude that further study of this technique is definitely needed.

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References

Relationship of Treatment for Hypertension and Stroke Mortality

To the Editor:
Klag et al1 clearly attach considerable importance to the lack of correlation in eight age-race-sex groups between the rank order of average annual decline in stroke mortality and the rank order of change in hypertension treatment (assessed in NHES and NHANES I and II) over the last 20 years. It seems of possible interest to look at the sexes separately (Table 1). The results for women are compatible with the decline in stroke mortality being related to an increase in the quantity and quality of treatment for hypertension. The results for men are much less tidy; indeed, even the two measures of antihypertensive treatment are unrelated to each other. Perhaps the problem lies in the quality of the data for treatment in men rather than in a lack of benefit from antihypertensive treatment? It should also be noted that the range of annual decline in stroke mortality among the male groups was very small (6.8–7.5%), making the rank order relatively unimportant.

In support of their negative findings, Klag et al cite a report by Bonita and Beaglehok2 stating that “only approximately 10% of the reduction in stroke mortality in New Zealand can be attributed to treatment of hypertension.” In my opinion, the calculations of Bonita and Beaglehoke greatly underestimate the contribution of antihypertensive therapy to the reduction of stroke deaths in New Zealand. These calculations make use of the Hypertension Detection and Follow-up Program (HDFP)3 and Australian4 trial results, but pay no attention to the fact that in HDFP, about 60% of the control group were given antihypertensive therapy while in the Australian study, the most hypertensive 12% of the control group received antihypertensive treatment. In addition, the Australian subjects were a very low-risk group. A more detailed exposition of these points has been published,5 as well as the reply of Bonita and Beaglehok6 in which they estimate the contribution of antihypertensive treatment to the reduction in stroke mortality to be 20%. Whether the true figure is 20%, 30%, or 40% is simply not known at present, but one can be sure that it is neither 100% nor 10%.

Klag et al also cite another paper from New Zealand by Bonita et al7 noting that “the population-attributable risk of stroke for cigarette smokers was higher than for hypertensives.” What Bonita et al actually stated was that “. . . in this population roughly 37% of stroke events may be attributed to cigarette smoking and 36% to hypertension.” It is surely wrong to imply that these two figures are different. In addition, the diagnosis of hypertension in this study was based on the Hypertension Detection and Beaglehok8 in which they estimate the contribution of antihypertensive therapy to the reduction of stroke mortality to be 20%. Whether the true figure is 20%, 30%, or 40% is simply not known at present, but one can be sure that it is neither 100% nor 10%

Klag et al also cite another paper from New Zealand by Bonita et al7 noting that “the population-attributable risk of stroke for cigarette smokers was higher than for hypertensives.” What Bonita et al actually stated was that “. . . in this population roughly 37% of stroke events may be attributed to cigarette smoking and 36% to hypertension.” It is surely wrong to imply that these two figures are different. In addition, the diagnosis of hypertension in this study was based on whether the stroke victim was taking antihypertensive treatment at the time of the stroke,7 which would have led to underestimation of the risk attributable to hypertension. First, any stroke victim who was hypertensive but not treated would be counted as normotensive, and, second, the greater the success of antihypertensive treatment in the community in preventing strokes, the lower the apparent contribution of hypertension to stroke.

The utmost care must be taken in interpretation of this kind of data.

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References

Table 1. Data From Klag et al,1 Subdivided by Sex

<table>
<thead>
<tr>
<th>Age-race-sex group</th>
<th>Average annual decline in stroke mortality (1973-1981) %</th>
<th>Rank order</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Women</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55–64 BF</td>
<td>8.3</td>
<td>1</td>
</tr>
<tr>
<td>65–74 BF</td>
<td>7.9</td>
<td>1</td>
</tr>
<tr>
<td>65–74 WF</td>
<td>6.6</td>
<td>3</td>
</tr>
<tr>
<td>55–64 WF</td>
<td>5.8</td>
<td>4</td>
</tr>
<tr>
<td><strong>Men</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>55–64 WM</td>
<td>7.5</td>
<td>1</td>
</tr>
<tr>
<td>65–74 WM</td>
<td>7.3</td>
<td>2</td>
</tr>
<tr>
<td>55–64 BM</td>
<td>6.9</td>
<td>1</td>
</tr>
<tr>
<td>65–74 BM</td>
<td>6.8</td>
<td>4</td>
</tr>
</tbody>
</table>

*Data from Figure 5.
3. Five-year findings of the hypertension detection and follow-up program. 1. Reduction in mortality of persons with high blood pressure, including mild hypertension. JAMA 1979; 242:2562–2571
5. Simpson FO: No evidence that treatment of hypertension does not explain much of the decline in mortality from stroke in New Zealand. NZ Med J 1987;100:452–453

The following is in reply:

To the Editor:

We appreciate Dr. Simpson’s thoughtful comments and agree that ecologic analyses such as ours must be interpreted with caution. We set out to demonstrate an association between hypertension treatment and decline in stroke mortality, and we, too, are bothered by the lack of such a relationship. From our review of the data, however, this lack of association is almost certainly due to the uniformity in the rates of decline in stroke mortality, not to the quality of the antihypertensive treatment data.1 Except for that in the group of 75–84-year-old black men and women, the rates of decline were not significantly different among age-race-sex groups. This lack of variability in rates and the onset of stroke decline in 1973 in each age-race-sex group argue strongly for an environmental change that affected all demographic groups equally.

Dr. Simpson’s reanalysis by gender is interesting but necessitates subdividing an already-small number of data points.

Dr. Simpson raises cogent questions about the previous studies by Bonita et al. Although one can argue that these studies using population-attributable risk estimates may have underestimated the contribution of blood pressure treatment to stroke decline, these criticisms do not mitigate our findings, which are based on different methodology. This discussion highlights the need for further work to delineate the underlying reasons for the salutary changes in stroke mortality.

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Reference

Pure Motor Hemiplegia due to Pontine Hemorrhage

To the Editor:

Pure motor hemiplegia (PMH) is a well-defined clinical syndrome. Since the advent of computed tomography (CT), reported sites and etiologies of PMH have been varied. Besides lacunar infarction or hemorrhage as major causes of PMH, other rare etiologies such as glioma, abscess, metastatic brain tumor, subdural hematoma, postoperative epidural hematoma, saccular aneurysm of the basilar artery, and neurocysticercosis have been reported. Although hemorrhage in the putamen, thalamus, and internal capsule have been described as the origin of PMH, we have known only two reported cases of PMH attributable to pontine hemorrhage,1,2 and these have both occurred in the last 4 years.

A 74-year-old hypertensive woman noticed a worsening left hemiparesis. On admission, her blood pressure was 176/92 mm Hg. She was alert, with dysarthric speech due to an operation for right maxillary cancer 14 years before. She had severe hemiparesis on the left side with grade 0 faciocutaneous paralysis in the upper limb and grade 2 in the lower limb. Babinsky’s sign was present on the left side. There was no facial palsy or ataxia, and all sensation was intact. Computed tomography revealed a small hematoma in the right pontine base. The patient walked home at discharge.

The other patient was a 54-year-old hypertensive man who noticed weakness on the right side while driving his car. He had neither headache nor nausea. His blood pressure was 190/100 mm Hg. There was no arrhythmia. He was alert and his speech was dysarthric. He had mild facial palsy and right hemiparesis with grade 0 in the upper limb and grade 2 in the lower limb. Sensory impairment was not demonstrated. Deep tendon reflexes were normal and Babinski’s sign was not present. A CT scan demonstrated a small hematoma in the left pontine base. The auditory brain stem response and short-latency somatosensory evoked potentials were within normal limits. Vertebral angiography showed diffuse sclerosis. The patient was able to walk without a cane at discharge. His hemiparesis completely resolved 3 months after onset.

Primary pontine hemorrhage localized within the basis pontis is a rare occurrence. As our cases demonstrate, it allows the patient good recovery of motor function, presumably due to partial involvement of the pyramidal tract, which is dispersed in the pontine base.

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References

Patch Grafting in Carotid Endarterectomy

To the Editor:

We read with great interest the review by Awad and Little1 in the March issue of Stroke on patch angioplasty in carotid endarterectomy and agree with the conclusion that patch angioplasty may be beneficial. Unfortunately, the authors referred to our prospective randomized study2 as though it were not published, when it actually appeared in the Journal of Vascular Surgery 6 months before they submitted their review.

Carotid patching reduced the restenosis rate significantly (3.5% vs. 21%; p=0.006). Its effect was most evident in women. Recurrent stenosis occurred more frequently when residual lesions were seen on early postoperative digital subtraction angiography. Long-term follow-up showed that differences in restenosis rates occur in the first postoperative year while late
Relationship of treatment for hypertension and stroke mortality.

F O Simpson

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