Dr. Lamy that such rates are probably a much better reflection of current risk. These studies do not specify the rates for the subgroup with relevant carotid lesions, however, making the problem of finding appropriate "historical controls" even more vexing.

Fortunately, it appears that definitive resolution will become available in the next few years, when several large European and North American controlled studies are completed. Since aspirin treatment, apparently the best available medical therapy, does not seem to help women and only partially reduces the excess S+D rate in men, I hope fervently that surgery will be proven to be of value and that we will have a clear definition of the circumstances in which it is applicable.

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Thalamic Lesion Producing Ataxic Hemiparesis

To the Editor:

We read with great interest Dr. Murthy’s letter1 reporting a patient with ataxic hemiparesis who had suffered a "thalamic" lesion. We find disturbing, however, that the lesion depicted by the computed tomogram (CT scan) is actually not localized in the thalamus. The CT scan shows a slice through the body of the lateral ventricles; the lateral walls of these are formed by the thalamus. The CT scan shows a slice through the body of the lateral ventricles. In any case, this thalamic component would be posterior to where the lesion of Dr. Murthy's patient is shown.

Perhaps lower slices of the same CT scan, which could more clearly demonstrate thalamic involvement, might be shown. The previously reported cases2,3 to which Dr. Murthy refers actually show lesions that are lower than that shown in the CT scan presented by him. Although we don't doubt that thalamic lesions may give rise to the syndrome of ataxic hemiparesis, it is our opinion that the documentation of this lesion should be clarified. Enough uncertainty already surrounds the localization of lesions that can cause this syndrome, and it seems that the one questioned here is no different than some of the others previously described.4

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References

Multicenter Trial of Hemodilution in Acute Ischemic Stroke

To the Editor:

Recently, the Scandinavian Stroke Study Group1 investigated the effects of hemodilution in a general stroke population. The study design, which had been previously reported,2 indicated that the major outcome measures were the proportion of institutionalized patients among the survivors at 3 months and the proportion of all patients entering the trial who were home at 3 months. A single-center trial3 was the source of background information, that is, the expected proportions in the control group.

There are major difficulties in interpreting the results of this study. First, analysis based on all patients enrolled in the trial is more appropriate than analysis based on survivors since the subgroup of survivors may be influenced by the treatment, leading to a selection bias. This is the case regardless of the fact that the treatment and control group mortality rates are similar and regardless of demonstrable similarity of the survivors in each group with respect to measured baseline characteristics. Second, sample size calculations for the second outcome measure were based on conservative estimates of the proportion of all patients entering the trial who were at home at 3 months (see Figure 7 of Strand et al). From the single-center trial,4 44% of the control group patients were at home at 3 months, with a 95% confidence interval of 30–58%. Using a more conservative estimate for the control group proportion, 45%, the power curve for a two-sided p<0.05 test of differences in proportions is given in Figure 1. It is clear from the power curve that differences of ≤15% would be very difficult to detect with a total sample size of 373 patients and maximum power of 0.80. Further, the power to

FIGURE 1. Power curve for two independent proportions.