Letters to the Editor

To Heparinize or Not: An Unsettled Issue

To the Editor:

In separate statements in the February 1989 edition of Stroke, Phillips and Scheinberg each firmly and unequivocally urge against the use of heparin in patients with brain ischemia. They conclude from past studies that heparin is ineffective and probably risky. Phillips further argues on theoretical grounds that "platelets, not fibrin, are the main components of arterial thrombi" and, since heparin effects on platelets are, if anything, prothrombotic, it should not prevent arterial thromboses.

I disagree with these opinions on two major points. First, I feel strongly that prior studies are hopelessly inadequate to conclude anything. All studies cited defined patients only by the time course of ischemia (TIA, progressing stroke, etc.) except for anecdotal results in patients with lacunar stroke in which heparin was predictably ineffective. Time courses do not define the nature of the causative vascular lesions, which are heterogeneous and range from platelet emboli arising in irregular plaques, to fresh red clots occluding stenotic, large arteries, to cardioemboli, to penetrating artery lipohyalinosis. No medicine or treatment is likely to be effective in all of these situations.

Second, hematologists characterize clots as "white" or "red." White clots are composed of platelets, often mixed with fibrin, which are more likely to form in fast-moving arterial streams that harbor irregular endothelial surfaces. Red clots are fibrin-dependent thrombi that are more likely to form in stagnant, low-flow zones of vessels, as, for example, in large arteries with very tight stenoses or on the tail of arterial occlusive thrombi. White clots could theoretically be prevented by agents that decrease platelet aggregation and agglutination, while white or red clots might work against red clots. Unfortunately, few, if any, studies have characterized the nature of thrombi in the cerebrovascular bed, and none have studied the effectiveness of platelet antiaggregants versus heparin or warfarin in situations that would favor white or red thrombi.

For these reasons, in contrast to Phillips and Scheinberg, I find the data inconclusive. The jury should still be out. To continue to study patients by time course alone seems futile. Until we have adequate studies in patients with defined vascular lesions, I think I made my position quite clear in the editorial on heparin coagulation. I have not even hinted that the issue is settled. I have simply stated that there are no studies yet that support its use; that heparin administration entails a substantial risk; and that use of a dangerous drug for an unproven objective is hazardous and should be avoided unless "... there is a strong suggestion of its probable value and with full understanding of its empirical basis or in a planned, controlled experimental clinical trial. . . ."

That hardly fits the characterization of my posture in Dr. Caplan's letter.

As an aside, it would be interesting to know how and why Dr. Caplan uses heparin in patients with acute ischemia and how he judges its effectiveness. I seriously question that he would advocate its use on the basis of anecdotal observations.

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Carotid Endarterectomy in a Teaching Hospital

To the Editor:

We read with interest the articles by Friedman et al regarding recent mortality and morbidity statistics for carotid endarterectomy in a community hospital. We have recently completed a similar study in a veterans administration medical center with comparable results.

The San Diego Veterans Administration Medical Center is a general teaching hospital affiliated with the University of California at San Diego School of Medicine. Surgery is performed by senior residents under the direct supervision of attending surgeons. We reviewed the records of 151 veterans who underwent cerebral angiography between January 1, 1985, and April 30, 1987. Of these, 51 patients subsequently had a total of 55 carotid endarterectomies. We defined a major complication as death, heart attack, or stroke. Demographic data were noted on each patient and the records were searched for details of the angiography, the operation, and the outcome. For those patients who underwent carotid endarterectomy, we recorded the name of the
surgeon, duration of cross-clamping, stump pressure, EEG findings if intraoperative monitoring was done, and whether a shunt was used. A major complication was assumed to be due to angiography if it occurred within 48 hours of the procedure and to surgery if it occurred within 96 hours of operation.

There were major complications following five surgeries (9%), but three of these were associated with a single surgeon. We found no correlation between the use of a shunt, clamp time, stump pressure, or EEG findings and neurologic outcome: clamp time was 33.8±28 minutes in patients with no complications compared to 41.5±11 minutes in the group suffering a major complication. During 166 angiograms, there were three major complications (1.8%) attributable to the procedure. There was no correlation between the occurrence of a major complication and the amount of time spent in the angiography suite or the amount of dye used: patients with complications spent 75±35 minutes in the suite compared to 42±43 for those without complications. Those with complications received 74 cc dye compared to 129±68 cc dye in those without complications.

Our results agree with those of others in showing a low incidence of complications during carotid angiography and surgery and confirm in a teaching hospital the encouraging results previously observed in community hospitals. The data emphasize once again that an important factor in reducing mortality and morbidity during carotid endarterectomy is the choice of the surgeon since three fifths of the major complications in our medical center occurred with one operator. Other important factors include careful preoperative cardiac evaluations, choice of anesthesia, and patient selection.

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