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 cardioembolic, to penetrating artery lipohyalinosis. No medicine or treatment is likely to be effective in all of these situations.

Seconds, hematologists characterize clots as "white" or "red." White clots are composed of platelets, often mixed with fibrin, which theoretically 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 heparin and warfarin 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 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 reasonable clinicians should keep their minds open. Until then, I, like Miller and Hart, continue to use heparin acutely in selected patients with brain ischemia. While Miller and Hart use antiaggregants in relation to temporal course, I use it as acute therapy patients with brain ischemia. While Miller and Hart use antiaggregants in relation to temporal course, I use it as acute therapy patients with brain ischemia.

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