Heparin Anticoagulation

Peritz Scheinberg, MD

It may well be the frustration we all feel at our inability to treat acute strokes effectively or to prevent a cerebral infarct following transient ischemic attacks (TIAs) or to inhibit the progression of a stroke that motivates our continued use of unproven and potentially harmful treatment modalities. There is increasing, if not universal, agreement that the indications, if any, for carotid endarterectomy are unknown, yet it is still widely used. The value of long-term administration of anticoagulants in preventing strokes has never been satisfactorily demonstrated, with the probable exception of some types of cardiogenic cerebral emboli, but anticoagulants are still used in many patients despite the recognized risk of hemorrhagic complications. In the above circumstances, lack of proof of efficacy in the literature has not been an effective deterrent.

In this issue of Stroke, Dr. Stephen J. Phillips has briefly reviewed the literature pertaining to heparin anticoagulation in the treatment of acute focal brain ischemia and concludes that the risk: benefit ratio is unacceptably high. His review and opinion were prompted at least partially by the article by Miller and Hart, which made recommendations for the use of heparin in certain patients with acute cerebral ischemia or infarction despite a lack of evidence of its efficacy and its known significant complication rate. By and large, heparin has fallen into disfavor in the management of TIAs, completed strokes, or acute stable partial strokes on the basis of published data, but there are still advocates for heparinization of patients with progressing stroke and immediately following cerebral embolization. Progressing stroke or stroke in evolution are generic terms describing progression of neurologic signs presumably caused by propagation of an intra-arterial thrombus. The pathophysiology of these syndromes is not actually known, and proof that they are indeed caused by a propagating thrombus is lacking.

Although it may be difficult not to treat a patient with presumed progressive vertebrobasilar insufficiency and brainstem ischemia, there has yet been no study that proves the value of heparinization in this circumstance, and there is certain evidence of its potential complications. It is unlikely that a prospective, randomized, controlled study will ever successfully address the issue of heparinization as a treatment modality for progressing stroke despite the obvious importance of the problem. We should at least be aware now that heparinization cannot be recommended on the basis of existing data.

Some studies have emphasized that the incidence of recurrent cerebral embolization of cardiac origin is approximately 12–15% during the first 2 weeks after the ictus, so that early anticoagulation has been recommended. How early and what kind of anticoagulation remain disputed points in view of the risk of hemorrhage into a cerebral embolic infarction. In my judgment, no proof has yet been offered that the risk of immediate heparinization of these patients is justified. There is ample time to use warfarin if anticoagulation is deemed appropriate therapy. It should also be emphasized that although the high incidence of cerebral embolization in patients with nonvalvular atrial fibrillation has been verified, no study has yet established the value of anticoagulants in these patients.

As Dr. Phillips points out in his review, the mechanism of action of heparin is complex, but since heparin has no known thrombolytic effect, its action upon an intra-arterial or intracardiac clot could only be to inhibit further propagation of that clot and not necessarily to prevent recurrent embolization from the thrombus. In addition to the well-known hemorrhagic complications of heparinization, thrombocytopenia and arterial thrombosis have been described and should be considered as liabilities of heparin administration. In view of these various serious potential complications, heparin should be used only if there is convincing scientific, not anecdotal, evidence of its efficacy. In the absence of such proof, recommendations for heparinization should be made only if 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.

References


**KEY WORDS** • cerebrovascular disorders • heparin
Heparin anticoagulation.
P Scheinberg

Stroke. 1989;20:173-174
doi: 10.1161/01.STR.20.2.173

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://stroke.ahajournals.org/content/20/2/173.citation

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Stroke can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Stroke is online at:
http://stroke.ahajournals.org/subscriptions/