LETTERS TO THE EDITOR

Regarding "Prospective randomized study of carotid endarterectomy with PTFE versus collagen impregnated Dacron (Hemashield) patching: Perioperative (30-day) results"

I read Dr James Edwards' comments (J Vasc Surg 2002;35:176-7) on our paper, "Prospective randomized study of carotid endarterectomy with PTFE versus collagen impregnated Dacron (Hemashield) patching: Perioperative (30-day) results" (J Vasc Surg 2002;35:125-30)[1] with great interest, and I appreciate the opportunity to respond.

Dr Edwards cited other reasons to explain the better outcome of PTFE in regards to perioperative carotid thrombosis and stroke, and the incidence of early (>50%) restenosis, other than increased thrombogenecity of the Hemashield patch. We did not conclude in our article that this was the only possible reason for the thrombosis, and we have no explanation for these higher rates of thrombosis and stroke. We simply raised the question as to whether the Hemashield patch, impregnated with collagen, can be thrombogenic in this location. This issue should be addressed by future studies.

We would also like to point out to the readers that not all collagen impregnated Dacron patches are the same as the conventional Hemashield type that was used in our study. In the last few years, a few authors, particularly surgeons in Europe, have reported using the Finesse Hemashield with satisfactory results.[2] The Finesse Hemashield has a different thickness from the conventional Hemashield, which has been used in many medical centers in the USA over the last several years.

Dr Edwards also stated that the technical performance, preoperative antiplatelet/anticoagulation medication, patient selection, and statistical aberrancy might explain the results of the superiority of PTFE versus Dacron. We agree that these are possibilities but don't feel that technical performance was a factor in this study because the two surgeons who contributed to this study (AFA and JHK) have performed a few thousand carotid endarterectomies over the last 20 years. The senior author (AFA) has reported the results of carotid endarterectomy in two randomized trials over the last several years using patch materials other than Hemashield, such as saphenous vein, internal jugular vein, or PTFE patching, with a perioperative stroke rate of 0.9%.[3,4] The routine protocol used for this study was similar to what has been used over the last 20 years and has been published previously.[3] However, it's conceivable that a statistical error may have been a factor in this study because of the sample size (200), even though we exceeded the required sample size recommended by our statistician.

Dr Edwards also indicated that the 5% acute carotid occlusion rate was quite unusual. We agree with him, and as a matter of fact, it was surprising to us. If you take the senior surgeon's (AFA) experience, the perioperative occlusion rate in this study was 3 out of 90 (3.3%) in the Hemashield patch group, which is relatively close to what has been reported by others (close to 2%, as reported by Ricco et al[2]). Our perioperative carotid occlusion rate in past endarterectomy trials has been <1%, which is similar to that reported by Dr Edwards' group, but different patches were used in these (eg, saphenous vein or PTFE).

In summary, based on our study, we still believe that conventional Hemashield collagen impregnated Dacron patches, used with carotid endarterectomy, likely carry higher perioperative carotid thrombosis and stroke rates than PTFE or saphenous vein patch. Further long-term studies are indicated if this patch is to be used in the future. Perhaps the Finesse Hemashield patch may prove to be a better alternative for those who prefer not to use PTFE patches.

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Regarding "Heparin modulates integrin function in human platelets"

Sobel et al elegantly and convincingly show that unfractionated heparin (UH) binds to the platelet integrin $\alpha_{IIb}\beta_3$ (Gp IIb/IIIa).[1] This binding then induced aggregation by outside-in signaling. A few additional comments are of relevance.

The effect of UH on platelets is not uniform. UH is a more powerful stimulator of in vitro and ex vivo aggregation in situations where platelet hyperactivity is present (eg, peripheral vascular disease,[2] coronary artery disease,[3,4] and anorexia nervosa[2]). The stimulatory effect depends on which anticoagulant preparation is used. Low molecular weight heparin(oid)s (LMWH) are less powerful stimulators than UH.[5-8] Moreover, porcine mucosa UH (used by Sobel et al) is a less powerful stimulator of aggregation than bovine lung UH.[5]

The UH effect on aggregation is enhanced by fibrinogen added in vitro.[7] This finding may be relevant because UH increases fibrinogen binding to platelets.[1]

UH also affects an early stage of platelet aggregation—the shape change.[8,10]