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Regarding "Intraoperative use of dextran is associated with cardiac complications after carotid endarterectomy"

We read with great interest the recent article by Farber et al¹ on behalf of the Vascular Study Group of New England regarding the high cardiovascular complication rate associated with the use of dextran as an antithrombotic agent after carotid endarterectomy (CEA). The strongest evidence to support intravenous dextran after CEA is based on its selective use when patients have persistently high transcranial Doppler (TCD)-detected microembolization.²

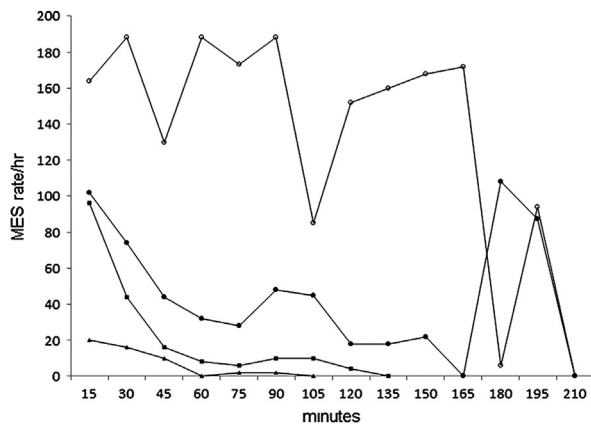


Fig. Microemboli signals (MES) after carotid endarterectomy in patients on single-antiplatelet therapy before surgery. ●, Dextran-40 (n = 28); ○, patients (n = 4) who developed early stroke, despite dextran-40 infusion; ■, tirofiban (n = 32); ▲, no additional antiplatelet treatment (n = 36).⁶ Reproduced with permission from Saedon et al.⁶

Cerebral microemboli appear to be primarily solid platelet aggregates. Microemboli are associated with a high risk of subsequent stroke and can be successfully controlled with a number of antiplatelet agents, including aspirin, dextran, and S-nitrosoglutathione.³ We found that it was possible to control microemboli in patients with recurrent or crescendo transient ischemic attacks using preoperative TCD-directed dextran therapy, and so safely defer CEA until the next elective list.⁴ However in this small cohort, the cardiovascular side effects of dextran appeared excessive. We therefore attempted to use a more selective TCD-directed intravenous antiplatelet agent, a glycoprotein IIb/IIIa receptor antagonist (tirofiban), to control transient cerebral microemboli both before and after CEA.⁵ In view of our previous experience of the effectiveness and tolerability of the intravenous glycoprotein IIb/IIIa antagonist antiplatelet agent (tirofiban),⁵ we have recently compared the efficacy of dextran with tirofiban and found that TCD-directed tirofiban therapy appears more effective than dextran-40 in suppression of cerebral microemboli after CEA, with a lower side effect profile⁶ (Fig).

Our findings support the authors' conclusions that dextran has a limited role in carotid endarterectomy. In our opinion, TCD-directed tirofiban would be a safer and more efficacious approach to reduce stroke risk after carotid endarterectomy in these patients.

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Reply

Multiple studies have demonstrated the efficacy of transcranial Doppler (TCD)-directed dextran therapy during carotid endarterectomy (CEA).¹⁻³ In our recent review of 6641 CEAs performed within the auspices of the Vascular Study Group of New England,⁴ we found perioperative infusion of dextran was not correlated with lower stroke rates but rather was associated with increased postoperative cardiac complications, including myocardial infarction and congestive heart failure. We concluded that there was limited clinical utility for the routine use of dextran during CEA.

We read with great interest the article by Saedon et al⁵ regarding the efficacy of TCD-directed antiplatelet therapy during CEA. These authors found TCD-directed infusion of the glycoprotein IIb/IIIa antagonist tirofiban was more effective than dextran in reducing transient cerebral microemboli before and after CEA.⁵ Their conclusions suggest a potential role of TCD-directed tirofiban infusion during CEA. Although these findings are provocative, widespread use of tirofiban cannot be recommended from these data alone. Further investigation of this agent in CEA patients is warranted.

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