Response to comments on: "The Influence of Wall Stress on AAA Growth and Biomarkers"

Dear Editor,

I thank Dr. Georgakarakos and colleagues for their letter in response to our article. We attempted to predict AAA growth based on relative wall stress. We found a lower growth rate for the group of AAAs with a relative low wall stress.[1] However, AAA growth is most likely a multi-factorial phenomenon, possibly including the effects of flow through the AAA. It is known that AAAs grow faster when they increase in size. Also the amount of thrombus grows, narrowing the flow lumen to a comparable size as the healthy aorta, only the flow lumen is more tortuous in AAAs. Additionally, the endothelial cells normally covering the vessel wall are destroyed and can therefore not respond to shear stresses induced by secondary flows. Our group previously developed FSI models of AAAs[2] and, among others, we will further investigate the relation between aneurysmal flow, thrombus and AAA growth. Additionally we will evaluate these models to study the relation between wall stress, flow effects, circulating biomarkers and AAA growth in a larger patient population.

References

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2 Wolters BJ, Rutten MC, Schurink GW, Kose U, de Hart J, van de Vosse FN, et al. A patient-specific computational model of fluid-structure interaction in abdominal aortic aneurysms. *Med Eng Phys* 2005;**27**(10):871–83.

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Comment on "Variations in the Pharmacological Management of Patients Treated with Carotid Endarterectomy: A Survey of European Vascular Surgeons"

Dear Editor,

We noted with interest the persistent variation in perioperative Clopidogrel management at the time of carotid endarterectomy (CEA) as documented by Hamish et al (*Eur J Vasc Endovasc Surg* 2009;**38**:402–7). The authors demonstrate that 43% and 55% of surgeons queried would stop Clopidogrel prior to CEA for both symptomatic and asymptomatic patients respectively.

Furthermore, the authors noted that over 49% of surgeons would stop Clopidogrel more than 7 days prior to surgery, irrespective of a patient's symptomatic status. Presumably, these clinical biases reflect a presumption that Clopidogrel is associated with increased serious bleeding complications at the time of CEA. Further, in symptomatic patients, it suggests that surgeons who stop Clopidogrel are more concerned about operative bleeding than about antiplatelet efficacy perioperatively.

In a recent study conducted by the Vascular Study Group of New England, we evaluated 4587 CEAS performed by 66 surgeons in the United States. Though our analysis focused

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on the impact of protamine sulfate on bleeding and thrombotic complications after CEA, we found no evidence that Clopidogrel was associated with serious bleeding complications. Reoperation for bleeding after CEA occurred in 1.0% of patients on Clopidogrel vs 1.2% in patients not on Clopidogrel $P = 0.67$). Nearly all patients were on anti-platelet therapy at the time of surgery (73% aspirin only, 3% Clopidogrel only, 13% aspirin and Clopidogrel). Based on these data, it is our practice to routinely perform CEA in patients taking Clopidogrel for an appropriate indication, especially symptomatic carotid artery disease. We do not believe that Clopidogrel increases serious bleeding after CEA.

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Response to comment on "Variations in the Pharmacological Management of Patients Treated with Carotid Endarterectomy: A Survey of European Vascular Surgeons"

Dear Editor,

We would like to thank Dr Stone and colleagues for their comments. The main purpose of the survey was to highlight the variations in the pharmacological practice around the time of CEA in Europe. It is certainly the practice of the senior author not to stop any anti-platelet agents at the time of CEA.

We hope that this survey will stimulate discussion about the optimal pharmacological management for patients in the peri-operative phase.

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