Response to Letter by Karapanayiotides and Devuyst

Response:

We thank Drs Karapanayiotides and Devuyst for their interest in our recent study, which gives us the opportunity to discuss our results further.

We would like firstly to point out that our model should in no way be used yet for clinical decision-making because further refinement is required to this idealized model as well as testing on more patient data. The title of the article is not misleading and states exactly what has been performed in the study. We are not sure how Drs Karapanayiotides and Devuyst conclude that the model “would better represent atheromatous disease in the common carotid artery, distal internal carotid artery, the coronaries or the lower limb arteries?”

There is growing evidence to suggest that plaque morphology is important for risk stratification of patients with carotid atheroma especially for asymptomatic patients with moderate carotid stenosis. Our study aimed at providing a theoretical explanation underlying these clinical findings. The assumptions and limitations of using an idealized model to illustrate a complex clinical problem have been extensively discussed. The NASCET trial although it concluded patients with stenosis of <50% did not benefit from surgery, the absolute statement “we know for sure that patients with symptomatic carotid stenosis <50% do not benefit from endarterectomy” is not tenable.

The calculation from our model suggested that the plaque stress was high when the fibrous cap thickness was <0.1 mm. In practice, patients with a small degree of carotid stenosis have atheromatous plaque that may be at early stage of development and lipid core can often be hardly detected. Moreover, this model was purely a structural mechanics model and any biochemical and biological contributions to risk have not been taken into account. Our simulation suggested that the maximal stress for rupture was reached when the fibrous cap thickness was <0.1 mm based on the assumption that the rupture stress threshold for carotid atheroma was 300 kPa. We used coronary data as the concept for the vulnerable plaque because there is data to suggest that coronary physiology may also be applicable to the carotid circulation.4–4 Certainly, further studies need to be done to test the mechanical property of carotid plaque. We are currently investigating the mechanical property of the fibrous cap using carotid endarterectomy specimens.

Our results also suggest that stress was higher at the shoulder regions of the plaque. This was attributable to the constant thickness of the fibrous cap used in the model. One of the reasons why carotid plaques rupture at the midpoint of the plaque is that the fibrous cap may be thinner over much of the lipid core. We have used finite element analysis based on in vivo MRI to investigate the stress distribution in carotid atheroma.5 The largest stress concentration can often be found at the thinnest portion of the fibrous cap or at the shoulder regions of the plaque.

We are pleased to read that despite the obvious limitations of ultrasound, Devuyst’s study corroborates our results with respect to critical fibrous cap thickness that is vulnerable to rupture. The fact that our model was limited by the constant thickness of the fibrous cap placed all over the plaque means that with further refinement of the simulation we can develop a more accurate description in the future, which may be a useful tool for risk stratification of patients particularly with an asymptomatic moderate carotid stenosis.

Disclosures

None.

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