stress independently of the wall thickness. We have chosen these 2 cases (no treatment and 100% occlusion) because the actual Von Mises stresses for any packing degree should be a value somewhere between them.

In summary, we have shown that the hydration of the coils, up to 93% volume filling, does not lead to an increase in the Von Mises stresses acting on the aneurysm wall. Our original measurements[1] and the results presented here are consistent with the filling characteristics of the HydroCoil reported by others (73% for HydroCoil vs 32% for platinum[3,4]).

References

1. Cantón G, Levy DI, Lasheras JC. **Changes in the intra-aneurysmal pressure due to HydroCoil embolization**. *AJNR Am J Neuroradiol* 2005;26:904–07
2. Boutin C. **Study of permeability by periodic and self-consistent homogenisation**. *Eur J Mech A-Solids* 2000;19:603–32
3. Cloft HJ, Kallmes DF. **Aneurysm packing with HydroCoil embolic system versus platinum coils: initial clinical experience**. *AJNR Am J Neuroradiol* 2004;25:60–62
4. Raymond J, Guilbert F, Weill A, et al. **Long-term angiographic recurrences after selective endovascular treatment of aneurysms with detachable coils**. *Stroke.* 2003;34:1398–403

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Clinical Experience of Selective Intra-Arterial Nimodipine Treatment for Cerebral Vasospasm Following Subarachnoid Hemorrhage

We read with interest the recent report by Firat et al entitled "Selective Intraarterial Nimodipine Treatment in an Experimental Subarachnoid Hemorrhage Model."[1] Nimodipine, a calcium channel blocker, is used orally and intravenously for the treatment of cerebral vasospasm after aneurysmal subarachnoid hemorrhage. In their article, the authors report the effects of selective intra-arterial nimodipine infusion in the treatment of experimentally induced cerebral vasospasm in a rabbit model. We congratulate the authors for their article but disagree with their claim that there are no experimental or clinical studies investigating the effects of intra-arterial nimodipine infusion. In the June 2004 issue of the *AJNR*,[2] we published the first preliminary clinical and angiographic results in a series of 25 consecutive patients treated with selective intra-arterial nimodipine for symptomatic aneurysmal cerebral vasospasm. There is a paucity of data available on this topic, and the contribution of Firat et al is appreciated by all of us in the field. However, readers would have benefited more from their work had they been informed of our prior article because they would have known that similar results had been achieved by independent investigators. Knowledge that the experimental work of Firat et al confirmed earlier clinical work would give physicians additional confidence in this important therapeutic option in aneurysmal cerebral vasospasm.

References

1. Firat MM, Gelebek V, Orer HS, et al. **Selective intraarterial nimodipine treatment in an experimental subarachnoid hemorrhage model.** *AJNR Am J Neuroradiol* 2005;26:1357–62
2. Biondi A, Ricciardi GK, Puybasset L, et al. **Intra-arterial nimodipine for the treatment of symptomatic cerebral vasospasm after aneurysmal subarachnoid hemorrhage: preliminary results.** *AJNR Am J Neuroradiol* 2004;25:1067–76

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Reply:

Thank you very much for the opportunity to give our reflections on comments by Biondi and colleagues regarding our study showing the effectiveness of intra-arterial nimdipine treatment in an animal subarachnoid hemorrhage model. We completely agree with them that their work, which is in complete agreement with ours, yet published earlier, merits being cited in our article, and we therefore were wrong to say, "There are no experimental or clinical studies investigating the effects of intraarterial nimodipine infusion." However, their article was published at approximately the same time that we submitted our manuscript for publication (our very first correspondence was initiated on June 1, 2004), and it was undeniably, but surely inadvertently, omitted. Neither we nor the reviewers were able to catch that missing citation in the process of publication. We take this opportunity to present our apologies to the authors. Nevertheless, we also want to point out that a preliminary account of this study was already presented in The Fourth Asian-Oceanian Congress of Neuroradiology and Head & Neck Radiology in Parallel with the Sixth Congress of World Federation of Interventional and Therapeutic Neuroradiology, which was held in Seoul, Korea, on September 21–25, 2001.[1] Publication delay was due to a reshuffling of our research group in the course of the study.

We believe that animal experimentation plays a key role in the development of new therapeutic modalities with the ease of performing controlled experiments and with the option to test both current and investigational drugs. In this respect, it is of value to note that the vehicle injections showed no reversal of the vasospasm in our rabbit model, a point of concern raised by Biondi et al regarding the alcohol content of the product (nimodipine, Nimotop) used in the clinical study.

Reference

1. Gelebek V, Firat MM, Orer HS, et al. **Intraarterial nimodipine infusion in an experimental rabbit cerebral vasospasm model.** Paper presented at The Fourth Asian-Oceanian Congress of Neuroradiology and Head & Neck Radiology in Parallel with the Sixth Congress of World Federation of Interventional and Therapeutic Neuroradiology; September 21–25, 2001; Seoul, Korea

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