

- Van Driest SL, Vasile VC, Ommen SR, et al. Myosin binding protein C mutations and compound heterozygosity in hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2004;44:1903–10.
- Jaaskelainen P, Kuusisto J, Miettinen R, et al. Mutations in the cardiac myosin-binding protein C gene are the predominant cause of familial hypertrophic cardiomyopathy in eastern Finland. *J Mol Med* 2002;80:412–22.
- Erdmann J, Raible J, Maki-Abadi J, et al. Spectrum of clinical phenotypes and gene variants in cardiac myosin-binding protein C mutation carriers with hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2001;38:322–30.
- Niimura H, Bachinski LL, Sangwatanaroj S, et al. Mutations in the gene for cardiac myosin-binding protein C and late-onset familial hypertrophic cardiomyopathy. *N Engl J Med* 1998;338:1248–57.
- National Center for Biotechnology Information. Available at: <http://www.ncbi.nlm.nih.gov/>. Accessed April 1, 2005.
- DNA Mutation Database. Familial Hypertrophic Cardiomyopathy. Available at: <http://www.angis.org.au/Databases/Heart/heartbreak.html>. Accessed April 1, 2005.

## Effect of Two Different Neuroprotection Systems on Microembolization During Carotid Artery Stenting

After reading the study titled “Effect to Two Different Neuroprotection Systems on Microembolization During Carotid Artery Stenting” by Schmidt et al. (1) in the *Journal* and after having an extensive experience with a system that holds several similarities with the MO.MA device (Invatec s.r.l., Roncadelle, Italy), I can make the following comments.

The investigators stated that 71% of their patients had microembolic signals (MESs) after balloon dilation of the stent; MESs during stent placement and balloon dilation were more than six times higher than during wire passage. This latter observation suggests that antegrade flow was still present using the MO.MA device.

The utilization of balloon occlusion of the common carotid artery (CCA) and external carotid artery (ECA) and the description of continuous MESs during carotid artery stenting (CAS) using a filter device was reported earlier by us (2). The researchers did not reference this original study.

Using occlusion of the CCA and ECA, we experienced, as did Schmidt et al. (1), that MESs were still present; we attributed them to antegrade flow through branches not occluded by the balloon. The other potential explanation is a Venturi effect of the circle of Willis suctioning from the column of stagnant flow in the internal carotid artery after balloon occlusion.

Finally, owing to the above-mentioned findings, we added flow reversed to the occlusion of the CCA and ECA.

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## REFERENCES

- Schmidt A, Diederich CW, Scheinert S, et al. Effect of two different neuroprotection systems on microembolization during carotid artery stenting. *J Am Coll Cardiol* 2004;44:1966–9.

- Parodi JC, LaMura R, Ferreira LM, et al. Initial evaluation of carotid angioplasty and stenting with three different cerebral protection devices. *J Vasc Surg* 2000;32:1127–36.

## REPLY

Dr. Parodi mentions his experience with a similar protection device, the PAES (Parodi antiembolism system) (1). Because of the requirement of brevity of our study (2), the comparative technical discussion of both systems (MO.MA vs. PAES) was not possible. An advantage of the PAES over the MO.MA system could be the potential continuous retrograde flow through the target lesion during the intervention. Establishment of a continuous retrograde flow in the internal carotid artery using this concept has been demonstrated in an animal model (3). However, there are no scientific data demonstrating and quantifying that, during the critical phases of stent placement, delivery and postdilation of the PAES permits an effective retrograde flow in humans. Dr. Parodi mentioned in his study (1) the use of this protection device in nine patients. Transcranial Doppler monitoring revealed no microembolic signals (MESs) during clamping of the common carotid artery in these patients. In our study, using the MO.MA system there was also a considerable number of subjects (48% of 21 patients) showing no MESs during stent deployment and during balloon dilation (29% of 21 patients). It is regrettable that a controlled multicenter registry, showing the safety and feasibility of the PAES, as it has been conducted recently using the MO.MA system (4), is not yet available. A randomized comparison between the PAES and MO.MA device using transcranial Doppler with detection of MESs would be of interest.

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doi:10.1016/j.jacc.2005.04.024

## REFERENCES

- Parodi JC, LaMura R, Ferreira LM, et al. Initial evaluation of carotid angioplasty and stenting with three different cerebral protection devices. *J Vasc Surg* 2000;32:1127–36.
- Schmidt A, Diederich KW, Scheinert S, et al. Effect of two different neuroprotection systems on microembolization during carotid artery stenting. *J Am Coll Cardiol* 2004;44:1966–9.
- Bates MC, Dorros G, Parodi J, Ohki T. Reversal of the direction of internal carotid artery blood flow by occlusion of the common and external carotid arteries in a swine model. *Catheter Cardiovasc Interv* 2003;60:270–5.
- Reimers B, Sievert H, Schuler G, et al. Proximal endovascular flow blockage for cerebral protection during carotid artery stenting: results from a prospective multicenter registry. *J Endovasc Ther* 2005;12:156–65.