

Regarding "Cerebral microembolization after protected carotid artery stenting in surgical high-risk patients: results of a 2-year prospective study"

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Hammer et al¹ have performed an elegant study of the true incidence of cerebral lesions after carotid artery stenting (CAS). Most of the microinfarctions that were noted had no clinical consequences, but the authors correctly point out that the late effects of these "silent" infarctions on cognitive function are unknown.

The authors, as well as the invited commentators, both conclude that endovascular manipulation of the internal carotid artery (ICA) is not responsible for these subclinical lesions. This is based on the fact that the topography of the microinfarctions was not limited to the territory of the stented ICA. Instead, blame is placed on manipulations of the aortic arch or common carotid arteries (CCAs). This absolution of ICA stenting appears to us to be rather disingenuous, because aortic and CCA manipulation cannot be separated from the specific technique of carotid dilation.

In any event, we question whether the observed lesions were indeed independent of the CAS itself. According to the data presented, 14 patients had ipsilateral lesions (8 alone and 6 associated with other lesions), while 7 had lesions elsewhere than in the ipsilateral hemisphere. In other words, there were almost two times more microinfarctions in the ipsilateral hemisphere, especially if we note that one of the seven patients with contralateral lesions had complete occlusion of that side's CCA and could not, therefore, have had embolization originating from it. Furthermore, that patient had a patent anterior communicating artery. Thus, even though it is clear that some microembolization originates from other than ICA manipulations, the significantly higher incidence of ipsilateral lesions forces us to consider the role of intra-ICA manipulations themselves.

We therefore cannot be completely reassured concerning the innocuousness of ICA manipulations, nor can we share the commentators' optimism in emphasizing the specific precautions used in their center (minimization of aortic arch manipulation, meticulous technique, and so on). These comments raise the question that every reader asks: "under the optimized conditions at your center, what is the real incidence of microinfarctions?"

While we again congratulate the authors for their comprehensive study, we also regret that once again the utility of carotid interventions (carotid endarterectomy [CEA] or CAS) for asymptomatic stenoses (66% of patients) and in elderly patients (41% older than 75 years) is not discussed. The Asymptomatic Carotid Surgery Trial study showed a beneficial effect of CEA that was limited to a 2.5% reduction in the incidence of cerebrovascular accidents over 5 years.² Furthermore, this marginal benefit became apparent (under the best operative conditions) only after 2 years.

If the cost of this limited benefit (assuming that CAS has the same effects as CEA) is a 40% incidence of silent microinfarction, one would be justified in doubting the rationale of performing CAS in asymptomatic elderly patients. Indeed, one might wonder if these microembolizations hasten the onset, or worsen the severity, of senile dementia.

REFERENCES

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Reply

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We appreciate the letter from Pr. R. Limet, but we want to clarify the following points. First, we have not concluded that "endovascular manipulations of the internal carotid artery are not responsible for subclinical lesions."

From our study we can conclude that a significant number of focal diffusion-weighted imaging (DWI) lesions could have resulted only from manipulations in the aortic arch or arch vessels, especially the lesions located in brain territories other than the ipsilateral anterior circulation. The exact number of positive DWI cases was, we must correct Pr. Limet's numbers, 8 for ipsilateral brain lesions, 6 for a combination of ipsilateral and other territories, and 7 for lesions located exclusively in other vascular territories. Migration through the circle of Willis seems to play a minor role, according to our analysis of intracranial angiograms. Patients who had a contralateral common carotid catheterization for diagnostic purposes only, by using a 4F catheter, had a rate of ipsilateral DWI lesions of 33%, in comparison to 9.4% when this artery was not probed. It seems therefore logical to find a higher incidence of microembolization in the ipsilateral brain when treating an internal carotid artery (ICA) stenosis, because the endovascular maneuvers are more aggressive: placement of a stiff exchange wire, cannulation of the common carotid artery (CCA) with a 6F sheath positioned close to the CCA bifurcation, repeated contrast injections and flushes, advancement of a folded filter device (external diameter, 2.9-3.9F) through the ICA stenosis, and retrieval of the filter (during which material can be pressed through the filter pores or meshwork). On top of this comes, obviously, the stenting and dilation itself. We admit that this step of the procedure cannot be dissociated from most of the other maneuvers, but we do not know how many new DWI lesions this generates, which supposes that some emboli are not captured by the filter or pass between the arterial wall and the filter. From a theoretical point of view, an elegant way to assess this would be to perform a study comparing filters with flow-reversal devices (Parodi antiembolism system). In addition, we do not know what kind of emboli (thrombus, air, or atheroma) can induce small incidental DWI lesions and how large the fragments have to be. According to animal studies,¹ it seems possible that fragments even smaller than the pores of filters, which therefore cannot be captured, could induce such lesions. This could explain why we found no significant difference in ipsilateral DWI lesions whether the filters contained macroscopic debris or not after the procedures (28% vs 24%).

Carotid artery stenting (CAS) obviously requires adequate training and an adequate environment. Unnecessary catheterization of other arch vessels should be avoided and certainly can reduce the overall rate of DWI lesions. Technical skills alone can probably to a large extent not avoid such lesions. We observed, for example, no reduction of the number of DWI lesions over the 2 years of our patient inclusion: 11 positive cases in the 27 first patients of this study vs 10 in the 26 last patients.

We are convinced, according to our data, that since the introduction of protective devices too much attention has been placed on the carotid lesions themselves and their structure to explain or try to predict cerebral embolization during CAS. Because it seems that the femoral approach will remain the privileged route for endovascular treatment of ICA stenoses, a direct puncture of the CCA carrying a too-important risk of complications, especially in patients premedicated with antiaggregants and heparin, we are not particularly optimistic about seeing an important reduction of DWI lesions in the future. The only way to reassure us would be to demonstrate that those lesions are innocuous — especially that they induce no negative effects on cognitive functions or acceleration of intellectual decline.

Second, concerning the utility of carotid interventions in asymptomatic patients, we agree that this topic is still controversial and that the indications have to be restrained. Local center expertise and complication rates, the patient's condition and life expectancy, and the degree of ipsilateral and contralateral stenosis are some of the key points that need to be assessed. The group of asymptomatic patients who we treated was carefully selected on the basis of local and general criteria, and we considered, after a multidisciplinary discussion, that CAS was a better alternative than surgery or conservative management.

We did not treat patients who were elderly and asymptomatic. The asymptomatic group was in fact significantly younger than the symptomatic group (mean age, 69.5 vs 77.7 years) and had in general a good life expectancy. Their stenoses were very tight and bilateral in 46% of the cases. Twenty-nine percent of the patients had a hostile neck that precluded any surgical procedure. Forty-three percent had to undergo cardiac surgery in the near future, and in this group of patients, we considered that CAS followed within several days or weeks by cardiac surgery was potentially less invasive than a combined carotid and cardiac surgical procedure, which is known to carry a higher rate of mortality and morbidity.

Finally, none of these highly selected asymptomatic patients had a neurologic deficit, and their rate of new silent DWI lesions was less than that in the symptomatic group (34% vs 50%). We therefore think that our attitude was justified, but we agree that the indications to treat them must be restrictive and that the follow-up is very important.

Third, observation of such a high incidence of focal DWI lesions, especially in asymptomatic patients, should indeed be a concern. Fortunately, a recent study² has demonstrated that most of those DWI lesions (97%) do not evolve to macroscopic brain infarction and are nondetectable on magnetic resonance imaging follow-up examinations at 6 months. In addition, stenting of the ICA stenosis could prevent further embolization from the carotid plaque and, in some cases, restore cerebral perfusion, therefore potentially improving some cerebral cognitive functions. This remains nevertheless speculative, and when considering the high rate of DWI lesions, we definitively agree with Pr. Limet that the short- and long-term evolution of cognitive functions in CAS patients needs to be evaluated and compared with carotid endarterectomy.

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