Cerebral Microembolism During Coronary Angiography
A Randomized Comparison Between Femoral and Radial Arterial Access

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Background and Purpose—Microemboli observed during coronary angiography can cause silent ischemic cerebral lesions. The aim of this study was to investigate if the number of particulate cerebral microemboli during coronary angiography is influenced by access site used.

Methods—Fifty-one patients with stable angina pectoris referred for coronary angiography were randomized to right radial or right femoral arterial access. The number of particulate microemboli passing the middle cerebral arteries was continuously registered with transcranial Doppler.

Results—The median (minimum–maximum range) numbers of particulate emboli were significantly higher with radial 10 (1–120) than with femoral 6 (1–19) access. More particulate microemboli passed the right middle cerebral artery with the radial access.

Conclusions—This study indicates that the radial access used for coronary angiography generates more particulate cerebral microemboli than the femoral access and thus may influence the occurrence of silent cerebral injuries. (Stroke. 2011; 42:1475-1477.)

Key Words: coronary angiography • microemboli • radial • transcranial Doppler

Clinically apparent stroke is seen in <0.3% of patients examined by coronary angiography, but the incidence of asymptomatic cerebral infarctions may be considerably higher.1 Microemboli are observed to enter the middle cerebral arteries during coronary angiography.2 Although their clinical relevance is undecided, recent studies with diffusion-weighted MRI confirm that such microemboli may cause silent ischemic cerebral lesions.3–5 In a nonrandomized study, more cerebral microemboli were detected during coronary angiography via the radial compared to the femoral arterial access.5 This randomized study was designed to test the hypothesis that there is no difference in the occurrence of cerebral microemboli according to access site during coronary angiography.

Patients and Methods
Patients with stable angina pectoris without a history of coronary artery bypass surgery, valvular heart disease, or advanced kidney disease referred for coronary angiography at the Karolinska University Hospital were eligible. The study was approved by the regional ethics committee.

Data Collection
Coronary angiography was performed according to standard procedures. Both middle cerebral arteries were continuously monitored with a multifrequency Doppler system (Embodop, DWL), and cerebral microemboli were automatically identified, counted, and differentiated as gaseous or particulate.6,7 Data registration was divided into stages reflecting catheter exchanges and coronary examination.

Statistical Analysis
The Mann–Whitney U test (2-tailed) was used for continuous variables. Categorical data were compared with the χ2 test and Fisher exact test. Using data of Lund et al8 who reported 40% fewer particulate microemboli via the femoral (n=10) versus the radial (n=37) artery, ~22 patients had to be randomized to detect a difference between the access sites with 80% power at P<0.05.

Results
From February 2007 to June 2008, 340 patients were screened. Fifty-one were included and randomized to radial (n=28) or femoral (n=23) access. Baseline characteristics are shown in the Table. All patients were using aspirin; the radial group also received 5000 IU heparin.

Eight patients were converted from radial to femoral access and thus were excluded from the analysis. In the radial group, more patients had normal coronary arteries and the fluoroscopy time was also longer (Table).

Cerebral microemboli were detected in all patients. The majority was gaseous, most occurring during flushing of the catheters, and the rate did not differ between the groups (Table).
Significantly more (67%) particulate microemboli were detected in the radial group (Figure 1). In both groups, more particulate microemboli occurred during catheter exchanges than during examination of the coronary arteries (7 [range, 0–51] versus 1 [range, 0–67]; \( P/\text{H11021} < 0.001 \)).

Separate analyses for the right and left middle cerebral arteries, respectively, revealed that more particulate microemboli passed the right middle cerebral artery with the radial access, predominantly at catheter exchanges (Figure 2). There were no overt strokes.

**Discussion**

In this randomized study, we found that cerebral microemboli occurred in all patients. In accordance with the nonrandomized study by Lund et al., the radial access generated significantly more particulate microemboli than the femoral access. Interestingly, in contrast to the femoral access, more particulate microemboli entered the right than the left middle cerebral artery with the radial access, especially during catheter exchanges. Particulate microemboli are probably more harmful because they are more likely to permanently occlude cerebral microvessels. With the right radial approach, the catheters have to pass the apertures of the right brachiocephalic artery and bend sharply into the ascending aorta, which may disrupt atherosclerotic plaques with subsequent embolization. Given the longer duration of angiography with the radial access, the catheters may themselves constitute an additional embolic source. These findings favor the use of only 1 catheter.

Diffusion-weighted MRI examination of patients with vascular dementia implied that small clinically silent lesions may contribute to cognitive deterioration. Five prospective studies with diffusion-weighted MRI have detected new silent ischemic cerebral lesions after cardiac catheterization, the incidence ranging between 5% and 22%\(^{4,5,9–11}\). The occurrence of particulate microemboli was verified with transcranial Doppler in 3 of the studies\(^{4,5,10}\). Moreover, in 1 of these studies neuropsychological tests indicated a causal link between microemboli and cognitive impairment,\(^{5}\) demonstrating the clinical relevance of the present findings. In conclusion, the access site for diagnostic coronary angiography may have an impact on the occurrence of clinically silent cerebral injuries.

**Sources of Funding**

The Karolinska Institute, the Swedish Heart and Lung Foundation, and Stockholm County Council contributed to this study.

**Disclosures**

None.

**References**


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Stroke. 2011;42:1475-1477; originally published online March 10, 2011;
doi: 10.1161/STROKEAHA.110.608638

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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