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Atheroemboli to the brain: Size threshold for causing acute neuronal cell death * ** *

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Abstract

Objective: The objective of this study was to investigate the dilemma posed by the observations that carotid angioplasty dislodges significant numbers of plaque fragments but is reported to have a low rate of neurologic consequences. We examined the fragments released by ex vivo carotid angioplasty. The smaller and most numerous were separated by size and injected into rats to determine the tolerance of the brain to microemboli. **Methods:** Ex vivo angioplasty was performed on a total of 20 human carotid plaques removed en bloc. Plaques were placed within polytetrafluoroethylene grafts, and three manipulations were performed: guide wire insertion, 3.5- or 4.0-mm balloon angioplasty, and 5-mm angioplasty with or without a Palmaz stent. After each manipulation, the lumen was flushed, effluent was collected, and fragments were

counted under 100 \times magnification. Using 200- μ m and 500- μ m micropore mesh, we separated fragments by size into two groups: (1) less than 200 μ m and (2) 200 to 500 μ m. We then injected rats with saline alone (Group A), with 100 fragments less than 200 μ m (Group B), or with 100 fragments 200 to 500 μ m (Group C). Animals were euthanized at 1, 3, and 7 days, and brain sections were examined for cell viability and expression of HSP-72. **Results:** The total number of fragments dislodged from the plaques varied from 30 to 553. The mean number of fragments released with each manipulation was as follows: guide wire passage, 24; initial balloon angioplasty, 97; second balloon angioplasty, 68; and second angioplasty plus stent, 172. Sixteen of the 20 plaques dislodged fragments that were 1 mm or more in greatest dimension. There was no evidence of brain ischemia in Group A at any time. Group B also showed no injury at 1 or 3 days. However, injection of 200- to 500- μ m fragments (Group C) caused a scattered pattern of neuronal cell death. At 7 days, brain sections from both Group B and Group C animals had a scattered pattern of ischemic neurons. There were no classic wedge-shaped infarctions. **Discussion:** The brain appears to have a surprising tolerance for microembolization in the acute setting. Thus, carotid angioplasty may dislodge plaque fragments, but there may still be a low incidence of stroke. However, even small plaque fragments, less than 200 μ m, may cause neuronal ischemia at later time points. Periprocedural microemboli could cause subtle neurologic dysfunction in late follow-up. (J Vasc Surg 2000;32:68-76.)



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* Competition of interest: nil.

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