AJNR

Analysis of Slipstream Flow in Two Ruptured Intracranial Cerebral Aneurysms

Steven G. Imbesi and Charles W. Kerber

AJNR Am J Neuroradiol 1999, 20 (9) 1703-1705
http://www.ajnr.org/content/20/9/1703

AJNR Am J Neuroradiol 20:1703–1705, October 1999

Technical Note

Analysis of Slipstream Flow in Two Ruptured Intracranial Cerebral Aneurysms

Steven G. Imbesi and Charles W. Kerber

Summary: Replicas of ruptured posterior communicating and basilar artery aneurysms were created from cadaveric specimens and then were placed in a circuit of pulsating non-Newtonian fluid. Individual fluid slipstreams were opacified with isobaric dyes, and images were recorded on film. The slipstreams entered the distal aneurysm neck with impact against the distal lateral wall of the aneurysm. They then swirled slowly in a reverse vortical pattern within the aneurysm sac. Fluid exited the aneurysm at the proximal neck. The flow pattern clearly shows the impact zone of entering slipstreams (the point of aneurysm rupture) and provides information pertaining to aneurysm growth and formation.

Flow dynamics in the intracranial circulation have been illustrated previously with human vascular casts and models (1, 2). Flow dynamics of intracranial aneurysms, however, have been reported using mainly numerical or created models, which are simplified representations (3, 4). We recently obtained two fresh cadaveric specimens of patients who had died as a result of ruptured intracranial aneurysms, made accurate castings and reproductions of the aneurysms and surrounding arterial systems, and studied the flow dynamics in the systems. The resulting silicone replicas were accurate to within 1% of the original cadaveric specimens, providing highly realistic aneurysm geometry. We now report the results of those experiments.

Technique and Observations

We created castings of a posterior communicating artery aneurysm and a basilar artery aneurysm from fresh human cadavers by using the lost-wax technique. Details of this process have been described previously (5). Although the aneurysms had ruptured, no modification of this procedure was necessary because thrombus had sealed the aneurysm sac. This tiny clot at the point of rupture was the only source of potential error in the replication process; however, it did not alter the aneurysm geometry. The clear elastic silicone replicas made from the original castings were placed in a circuit of pulsatile non-Newtonian fluid that mimics the rheologic properties of blood (6). A blood pump (model 1421 Harvard apparatus, S. Natick, MA), cycling at one pulse per second, provided fluid flow. Flows were adjusted to replicate human physiological flow profiles with a Square Wave Electromagnetic Flowmeter (Carolina Medical Electronics, Inc., King, NC), so that there was 40% forward flow during diastole compared with flow during systole. We studied internal carotid artery flow rates of 400 mL per minute and vertebral artery flow rates of 100 mL per minute, similar to that found in a normal human. The fluid slipstreams were opacified with isobaric dyes after insertion of 30-gauge needles into the vessel sidewall. Images were recorded on 35-mm film and on a super VHS video at shutter speeds of 1/1000 second.

Analysis of flow in the internal carotid and basilar arteries showed parallel fluid slipstreams of generally equal velocity except at the artery wall's boundary layer where little, if any, flow exists. Flow often assumed a helical pattern, especially in the regions of vessel curvature. At the aneurysm origin, flow was noted to enter via the distal aneurysm neck and impact against the distal lateral aneurysm wall. Disturbed, but nonturbulent, flow then continued along the aneurysm wall in a vortical pattern, opposite the direction of the parent vessel flow. The opacified slipstreams slowly swirled in this reverse vortical pattern within the aneurysm sac and eventually were sucked out of the aneurysm peripheral to the central distal incoming jet, usually at the proximal aspect of the aneurysm neck (Figs 1, 2). Because there is always forward flow in the intracranial circulation, both during diastole and systole, this pattern remained constant while the velocities changed through the different phases of the cardiac cycle. Although the two aneurysms studied were quite different (the internal carotid posterior communicating artery aneurysm was a small, narrow-necked, bifurcation aneurysm of the anterior circulation and the basilar artery aneurysm was a large, wide-necked, sidewall aneurysm of the posterior circulation), the flow patterns were essentially identical.

Discussion

Intravascular flow dynamics are generally thought to be responsible for inducing changes in the vessel wall (7–9). Pathologic mechanisms that result in these problems are complex and not fully understood. The main factors are hemodynamic, namely blood pressure (a measure of force) and blood flow (the applied direction of that force). The use of clear elastic silicone replicas from human cadaveric sources allows for high-resolution detailed analysis of individual slipstream flow patterns in an accurate, reproducible, in vitro setting.

Our observation and interpretation of the presented data are as follows. In the internal carotid and basilar artery parent vessels, opacified fluid slipstreams were generally parallel to the vessel sidewall, characteristic of the flow profile found in

Received February 16, 1999; accepted after revision May 5.

From the Hospital of the University of Pennsylvania (S.G.I.), Philadelphia, PA, and the University of California, San Diego Medical Center (C.W.K.), San Diego, CA.

Presented at the 1998 Annual Meeting of the American Society of Neuroradiology.

Address reprint requests to Steven G. Imbesi, MD, Assistant Professor of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104.

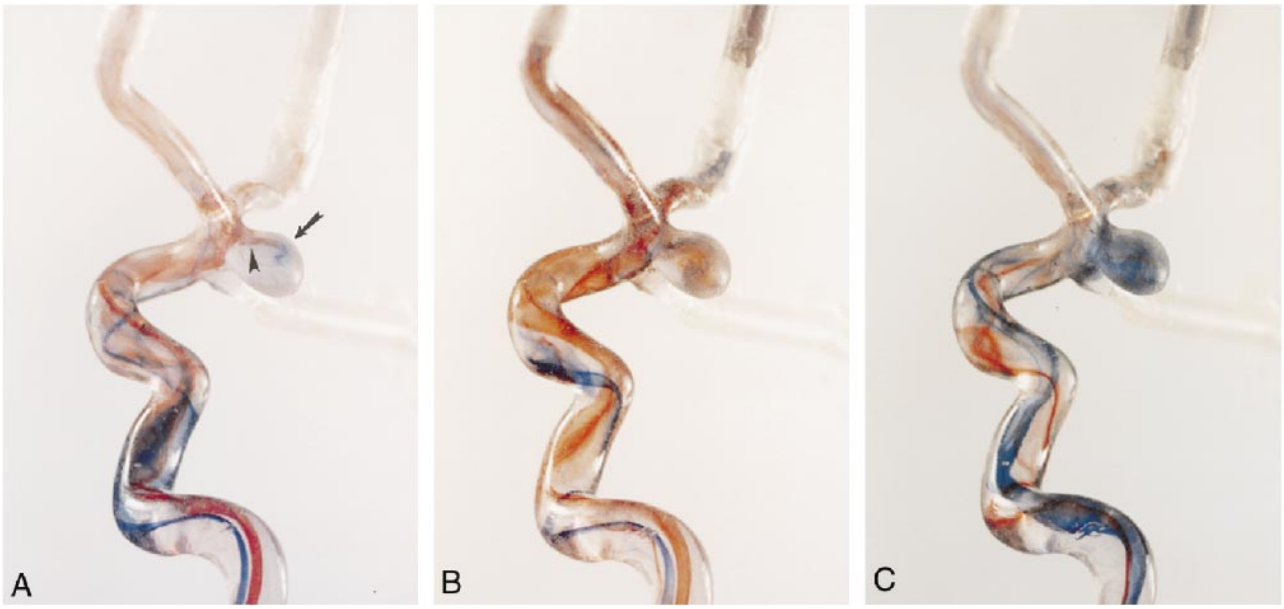

FIG 1. Posterior communicating artery aneurysm.
A, Opacified fluid slipstreams enter the aneurysm sac via the distal neck (*arrowhead*) and impact upon the distal lateral aneurysm wall (*arrow*).
B, Disturbed, nonturbulent reverse vortical flow is seen within the aneurysm.
C, Flow exits the aneurysm peripheral to the central incoming jet, usually at the proximal portion of the neck.

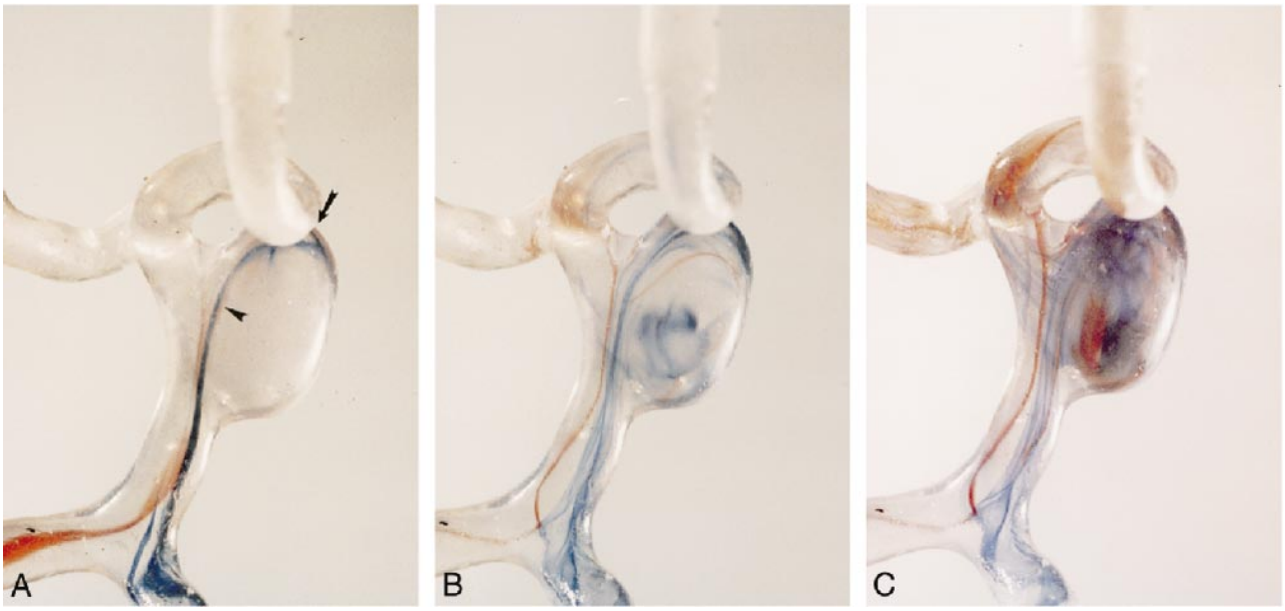

FIG 2. Basilar artery aneurysm.
A, Similarly, slipstreams enter via the distal aneurysm neck (*arrowhead*) and impact against the distal lateral aneurysm wall (*arrow*), the site of aneurysm rupture.
B, Reverse vortical flow is seen within the aneurysm sac.
C, Flow exits the proximal neck peripheral to the distal central incoming jet.

normal human vessels by Doppler sonography. The slipstreams often assumed a helical pattern in the regions of vessel curvature. At the aneurysm origin, slipstreams entered through the distal aneurysm neck and struck the distal lateral aneurysm wall. After this impact, sluggish, less energetic slipstreams flowed along the aneurysm dome in a swirling reverse vortical pattern. A previous publication by Benndorf et al (10) has shown a decrease in flow velocity from measurements at the aneurysm dome compared with measurements at the aneurysm neck with intravascular Doppler son-

ography. We believe these observations confirm transfer of kinetic energy from the flowing fluid to the impact site of the aneurysm wall. These impact sites, at the distal lateral aspect of the aneurysm wall, were the aneurysm rupture foci found in the cadaver specimens.

Understanding how these slipstreams impact against the aneurysm wall also provides information concerning aneurysm growth and may provide clues to de novo aneurysm formation. Aneurysms develop at regions of vessel bifurcation. Therefore, it is logical to conclude that the interaction between the fluid slipstream, with its inherent kinetic energy, and the vessel wall flow divider at the bifurcation, should induce small tears of the superficial inner vascular lumen similar to that observed at the aneurysm rupture site (11, 12). These small intimal tears may be the precursors to subsequent aneurysm formation. Finally, from a more practical standpoint, knowledge of intra-aneurysmal flow dynamics, including an understanding of the aneurysm inflow and outflow zones as well as the point of rupture, offers the promise of improving our ability to treat these lesions by endovascular techniques.

Acknowledgements

We gratefully acknowledge Shelley Van Buren for invaluable assistance in preparing the manuscript.

References

1. Kerber CW, Heilman CB. **Flow dynamics in the human carotid artery.** *AJNR Am J Neuroradiol* 1992;13:173–180
2. Chong BW, Kerber CW, Buxton RB, et al. **Blood flow dynamics in the vertebrobasilar system.** *AJNR Am J Neuroradiol* 1994;15:733–745
3. Burleson AC, Strother CM, Turitto VT. **Computer modeling of intracranial saccular and lateral aneurysms for the study of their hemodynamics.** *Neurosurgery* 1995;37:774–782
4. Liou TM, Chang WC, Liao CC. **Experimental study of steady and pulsatile flows in cerebral aneurysm model of various sizes at branching site.** *J Biomech Eng* 1997;119:325–332
5. Kerber CW, Heilman CB, Zanetti PH. **Transparent elastic arterial models. I. A brief technical note.** *Biorheology* 1989;26:1041–1049
6. Mann DE, Tarbell JM. **Flow of non-Newtonian blood analog fluids in rigid curved and straight artery models.** *Biorheology* 1990;27:711–733
7. Hashimoto N, Handa H, Hazama F. **Experimentally induced cerebral aneurysms in rats.** *Surg Neurol* 1983;19:107–111
8. Kim C, Kikuchi H, Hashimoto N, Hazama F. **Histopathological study of induced cerebral aneurysms in primates.** *Surg Neurol* 1989;32:45–50
9. Crompton MR. **Mechanisms of growth and rupture in cerebral berry aneurysms.** *Br Med J* 1966;1:1138–1142
10. Benndorf G, Wellnhofer E, Lanksch W, Felix R. **Intra-aneurysmal flow. Evaluation with Doppler guidewires.** *AJNR Am J Neuroradiol* 1996;17:1333–1337
11. Greenhill NS, Stehbens WE. **Scanning electron-microscopic study of experimentally induced intimal tears in rabbit arteries.** *Atherosclerosis* 1983;49:119–126
12. Kim C, Cervos-Navarro J, Patzold C, Tokuriki Y, Takebe Y, Hori K. **In vivo study of flow pattern at human carotid artery bifurcation with regard to aneurysm development.** *Acta Neurochir (Wien)* 1992;115:112–117