Are your MRI contrast agents cost-effective? Learn more about generic Gadolinium-Based Contrast Agents.





Flow in experimental berry aneurysms: method and model.

C W Kerber and C B Heilman

AJNR Am J Neuroradiol 1983, 4 (3) 374-377 http://www.ajnr.org/content/4/3/374

This information is current as of April 23, 2024.

Flow in Experimental Berry Aneurysms: Method and Model

Charles W. Kerber¹ and Carl B. Heilman¹

This study addresses two basic questions: What are the flow dynamics in aneurysms? Can these flows be modified to enhance retention of adhesive? Using Pyrex glass bifurcations, fluid flow was studied in a variety of aneurysms placed at varying positions around the bifurcations. Indicators injected into the slipstreams were recorded and studied both by stop-frame high-speed movie analysis and with 35 mm slides. Even at low-flow rates, a central slipstream strikes the apex of bifurcations, and may be partly responsible for the initial production of berry aneurysms. A lowpressure zone occurs at the lateral angle of bifurcations, probably explaining the formation of endovascular cushions. Flow into symmetrically placed narrow neck aneurysms did not occur. Indicator entered the aneurysm in a turbulent fashion only when there was orifice (lip) asymmetry. Both lip asymmetry and rapid flow favor intraaneurysmal turbulent flow.

Advances in the microneurosurgery of aneurysms and safer neuroanesthesia should have improved the chances and quality of survival in patients who have ruptured aneurysm. The facts are otherwise. Of the nearly 30,000 aneurysms that rupture each year in the United States and Canada, fewer than 5,000 patients are cured [1]. The Rochester group [2] surveyed three decades of results (1945-1975) and showed no significant reduction in morbidity after aneurysm rupture. Of 100 patients in that community, only 42 survived the initial hemorrhage to the end of the first month; at 6 months only 39 of the original 100 were in good neurologic condition. Other studies suggest similar outcomes [3-6]. Apparently, the good surgical results come by allowing patients to die, leaving a group that can tolerate the surgical manipulation. We wonder what would happen if percutaneous techniques of aneurysm obliteration were available. Would we then be able to take critically ill patients, occlude the aneurysm during the diagnostic angiogram, prevent the feared rebleeding, then improve cerebral perfusion to combat the effects of spasm?

Before attempting percutaneous treatment in humans, though, it seems worthwhile to identify flow patterns within human intracranial aneurysms. With this knowledge, one might better plan an approach, develop a methodology to allow aneurysm entry, and identify factors that would enhance deposition and retention of some occluding agent within the aneurysm sac.

Background Principles

In the human circulation, nutrients move by bulk flow, and it is necessary to understand rudimentary principles of flow dynamics in tubes. Poiseuille, a French physician, related the variables: flow =*pressure change* × $\pi/8 \times 1/viscosity \times radius^4/length$. Thus, flow will increase linearly if pressure increases and decrease if the viscosity of the fluid or the length of the tubing increases. Of most importance is the radius, as it is a fourth-power function. Doubling the radius increases flow 16 times.

Reynolds related the variables in a different fashion: $Re = density \times velocity \times tube diameter/viscosity$. The result is a dimensionless number that, if kept constant, allows one experimenter to compare his results with a different system from another laboratory. Beyond a certain Reynolds number (Re), turbulent flows occur. Nonturbulent, or laminar, flow is the normal state in most human arteries. Laminar intraarterial flow is best thought of as a series of concentric thin sleeves of material, with little flow at the artery wall (the boundary layer), and faster and faster flow as one approaches the center. Cutting the artery lengthwise shows the flow vectors to have a parabolic shape (fig. 1A). Curved tubes (as all arteries are) skew the parabola to the outside of the curve (fig. 1B).

Two types of flow disturbances may occur. An eccentric web or plaque generally forms downstream vortices (eddy shedding) (fig. 1C). Eddy shedding produces a high-pitched single-tone musical murmur. Increasing the Reynolds number beyond a certain point or creating a rough constriction in the stream yields real turbulance (random chaotic movement) (fig. 1D). Hydrodynamic theory states that no flow occurs with turbulence, but in the body movement does occur because of downstream runoff.

Materials and Methods

As it is difficult to see through blood and arteries of the size we must study, an optically clear system was made. Glass bifurcations were constructed of varying size and with varying bifurcation angles. Spherical aneurysms were placed at varying points around the bifurcation. Fluid flowing at varying velocities was passed through the system and indicators, either isobaric dyes or neutral-density spheres, were injected into the flowing fluid. The path and behavior of the indicators were recorded on high-speed 16 mm movie film and 35 mm frame film. Pattern analysis was done both by analog and by stop-frame movie analysis. A perfusion apparatus modified from Stehbens [8] was used, which consisted of a constant pressure reservoir with the pressure level kept constant by an overflow. This reservoir was connected by Tygon tubing to the glass model. The outflow passed through stopcocks and was pumped back into the overhead reservoir. Graduated cylinders placed under the stopcocks measured flow. All bifurcations were placed horizontally to avoid gravity effects. A 26 gauge needle used to inject the materials caused no appreciable flow disturbance.

Results

Figure 2 shows one model as seen from above. Direction of flow is indicated by the arrow. The flowing fluid in a more lateral slip-

¹ Neurodiagnostic Center of San Diego, 4020 Fifth Ave., Suite 301, San Diego, CA 92103. Address reprint requests to C. W. Kerber.

AJNR 4:374-377, May/June 1983 0195-6108/83/0403-0374 \$00.00 © American Roentgen Ray Society

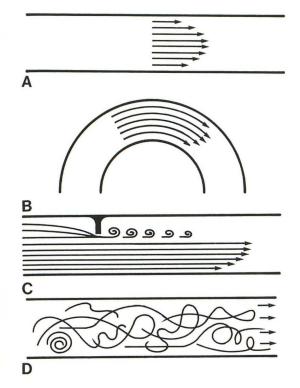


Fig. 1.—A, Longitudinal section through central plane of tube. Nonturbulent (laminar) flow from left to right. Velocity vectors form parabola, with most rapid flow in central slipstream, and almost no flow along wall (boundary layer). B, Modified laminar. Curved tubes (as all arteries are) skew parabola. C, Vortex formation (eddy shedding) beyond partial obstruction. Eddies gradually cease, dampened by viscous forces in fluid. D, Turbulence occurs when flow velocity exceeds critical number. Motion is random, chaotic, and antithesis of laminar flow. (Adapted from [3].) stream passes gently into the side arm of the bifurcation. As it passes beyond the right lateral angle it moves away from the wall, and the boundary layer widens.

Figure 3 demonstrates injection of indicator into the most central (fastest flowing) lamina. Note how the fluid strikes the apex of the bifurcation.

Figure 4 shows an aneurysm constructed at the apex of the bifurcation. The lips or entry area into the aneurysm are symmetrical, and the opening is small (about one-half aneurysm diameter). The central slipstream impinges on the centrum of the orifice but, surprisingly, does not enter the aneurysm. Even with turbulent flow, no entry occurs. The indicator rebounds from this stagnant area as though no aneurysm were present and then passes distally down one of the limbs.

Figure 5 shows an aneurysm constructed at the bifurcation but with its location not exactly at the center of the apex of the bifurcation. Its lips are thus asymmetric. The indicator entered this model with ease. Increasing the Reynolds number just to the point of turbulence caused turbulent flow in the aneurysm.

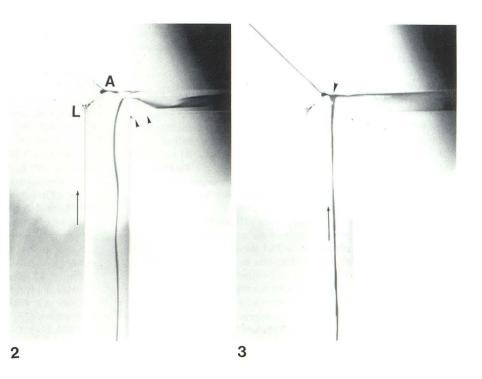
Discussion

The physical factors influencing blood flow have received scant attention until recently. In 1960 McDonald's book, *Blood Flow in Arteries* [9], ushered in a period of rapid growth of knowledge. In 1972, *Cardiovascular Fluid Dynamics* [10] summarized and updated the knowledge to that point. Since then, the number of rheologic studies has increased exponentially.

Studying flow in biophysical models outside the body is risky, as not only is bifurcation geometry important, but vessel tethering modifies fluid behavior too. Roach [11] stated that, in general, "The models used to date seemed to have produced less useful information than one might expect . . . probably because of a lack of some biological data with which to test them." Now, there is a

Fig. 2.—Glass bifurcation model, viewed from above. Flow direction (*arrow*). Left lateral angle (L) = 45° ; right lateral angle (*arrowheads*) = 90° . Indicator placed in nearly central slipstream enters downstream limb, but attempts to continue in straight line. Boundary layer widens markedly (cf. fig. 5A). Slipstreams swirl gently, then straighten again in a few tube diameters. A = apex.

Fig. 3.—Indicator injected into central slipstream vigorously strikes apex (*arrowhead*). At higher flow rates, it rebounds, swirling gently, then enters limbs. Direction of flow (*arrow*).



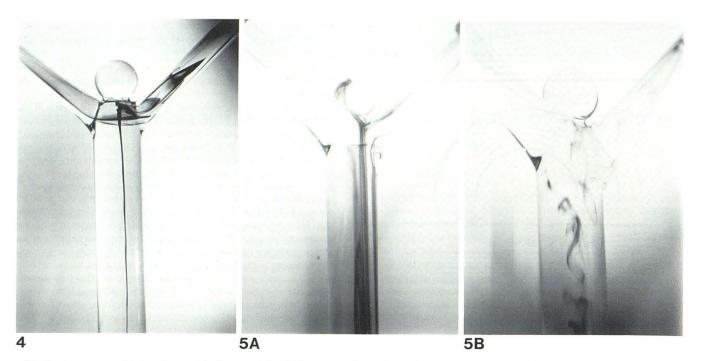


Fig. 4.—Aneurysm model placed symmetrically at apex of 90° bifurcation. Indicator in central slipstream surprisingly fails to enter aneurysm, and rebounds, swirls, then passes into limbs. Orifice size is one-half aneurysm diameter.

Fig. 5.—Aneurysm placed asymmetrically into apex of 90° bifurcation. Even at low flow, indicator enters dome. **B**, Increasing flow rate to point of turbulence causes indicator to enter aneurysm in turbulent fashion.

general agreement that the quality of blood flow at bifurcations produces disease such as atherosclerosis and aneurysms, and many have studied this critical site both with mechanical [12–16] and mathematical [17, 18] models to elucidate the pathologic physiology there.

Figure 2 shows the flow changes encountered at the lateral angles of a 90° bifurcation. Note how the boundary layer widens as the slipstream tries to continue in a straight line. The reattachment point downstream becomes more distal as the Reynolds number increases. This widening is thought to be responsible for the formation of endovascular cushions [15, 19, 20], which are widely regarded as precursors of atheromatous disease.

As aneurysms invariably occur at the apex of a bifurcation [21], the visualization of the forceful impingement of the central slipstream there (fig. 3) leads one to postulate that this force vector is important in the initiation and growth of human saccular aneurysms. What influence the force will have on both the initial catheterization of the sac and on occluding agent retention must be studied.

It was surprising to us that this central slipstream did not enter our aneurysm (fig. 4) as it has in other models [22]. Further, we were disturbed by the laminar flow in our aneurysms, because Ferguson [23, 24] has directly measured turbulence in human intracranial aneurysms during surgery. Thus, our model must be invalid. Further analysis (and discussion with M. Roach, personal communication) suggested orifice or lip asymmetry, as well as orifice size in the production of intraaneurysmal turbulence. She and coworkers had already stressed the importance of the bifurcation geometry [22]. We then made a model with asymmetric lips, that is, the aneurysm did not originate exactly from the apex of the bifurcation (fig. 5). Then, even at moderate Reynolds number (about 250), but especially with turbulent flow, the indicator entered the aneurysm, and flow in the sac was turbulent. In summary, nondistensible models are of some value as they allow development and refinement of methodology and may also yield some data about basic flow patterns. The geometry of bifurcations is so complex though, and the stakes are so high, that we must not be satisfied with anything less than direct castings of the circle of Willis and viscoelastic production from those castings. Only then will we approximate the conditions found in life.

REFERENCES

- Drake CG. Management of cerebral aneurysm. Stroke 1981;12:273-283
- Phillips LH II, Whisnant JP, O'Fallon WM, Sundt TM Jr. The unchanging pattern of subarachnoid hemorrhage in a community. *Neurology* (NY) 1980;30:1034–1040
- Post KD, Flanum ES, Goodgold A, Ransohoff F. Ruptured intracranial aneurysms: care, morbidity and mortality. J Neurosurg 1977;46:290–295
- Sundt TM Jr, Whisnant JP. Subarachnoid hemorrhage from intracranial aneurysms. Surgical management and natural history of the disease. N Engl J Med 1978;299:116–122
- Nishimoto A (Okayama University Medical School, Okayama Japan). Paper presented at the meeting of the Japanese Neurosurgical Society, Tokyo, October 1979
- Pakarine WS. Incidence, aetiology of primary subarachnoid hemorrhage. Acta Neurol Scand [Suppl] 1967;43:10–27
- Roach MR, Boughner DR. Biophysics principles in the cardiovascular system. In: Goodwin JW, ed. *Perinatal medicine*. Baltimore: Williams & Wilkins, **1976**:119–133
- Stehbens SE. Turbulence of blood flow. Q J Exp Physic/ 1959;44:110-117

- 9. McDonald DA, ed. Blood flow in arteries, 1st ed. London: Arnold, 1960
- Bergel DH, Derek H, eds. Cardiovascular fluid dynamics. New York: Academic Press, 1972
- 11. Roach MR. Biophysical analyses of blood vessel walls and blood flow. *Annu Rev Physiol* **1977**;39:51–71
- Brech R, Bellhouse BJ. Flow in branching vessels. Cardiovasc Res 1973;7:593-600
- Ferguson GG, Roach MR. Flow conditions at bifurcations as determined in glass models, with reference of the focal distribution of vascular lesions. In: Bergel DH, Derek H, eds. *Cardiovascular fluid dynamics*. New York: Academic Press, 1972:142–156
- Feuerstein IA, El Masry OA, Round GF. Arterial bifurcation flows: effects of flow rate and area ratio. Can J Physiol Pharmacol 1976;54:595–808
- 15. Fox JA, Hugh AE. Localization of atheroma: a theory based on boundary layer separation. *Br Heart J* **1966**;28:388–399
- Fry DL. Hemodynamic forces in atherogenesis. In: Scheinberg P, ed. Cerebrovascular diseases. New York: Raven, 1976
- Cox RH. A model for the dynamic mechanical properties of arteries. J Biomech 1972;5:135–152

- Tin-kan H, Naff SA. A mathematical model for systolic blood flow through a bifurcation. Presented at the meeting of the HICMBE, Chicago, July 1969
- Stehbens WE. Focal intimal proliferation in the cerebral arteries. Am J Pathol 1960;36:289–301
- Caro CG, Fitz-gerald JN, Schroter RC. Wall shear rate in arteries and distribution of early atheroma. In: AGRD conference proceedings. #65. Specialists meeting on fluid dynamics of blood circulation and respiratory flow, Naples, May 1970. London: U.S. Dept. of Commerce, 1970:13.1–13.8
- Hassler O. Morphologic studies on the large cerebral arteries with reference to the aetiology of subarachnoid hemorrhage. *Acta Psychiatr Neurol Scand [Suppl]* 1959;136:154–158
- Roach MR, Scott S, Ferguson GG. The hemodynamic importance of the geometry of bifurcations in the circle of Willis (glass model studies). *Stroke* 1972;3:255-267
- 23. Ferguson GG. Turbulence in human intracranial saccular aneurysms. J Neurosurg 1970;33:485–497
- Ferguson GG. Physical factors in the initiation, growth, and rupture of human intracranial saccular aneurysms. *J Neurosurg* 1972;37:666–677