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M F Brothers and R C Holgate

Intracranial Angioplasty for Treatment of Vasospasm After Subarachnoid Hemorrhage:
Technique and Modifications to Improve Branch Access

Michael F. Brothers[1,2]
Richard C. Holgate[1]

Angioplasty of the proximal portions of major cerebral arteries at the base of the brain has shown promise as a therapy for symptomatic vasospasm after subarachnoid hemorrhage. The blind-ended, single-lumen balloon-dilatation catheter most widely used to date lacks steerability, limiting its application to unbranched stems and single branches at bi- or trifurcation points. To extend the capabilities of cerebral angioplasty, we describe two modifications of the basic technique that have allowed increased selectivity and successful angioplasty of multiple branches, both proximal and distal, involved by vasospasm. Of four patients treated, three showed early improvement in their clinical condition, likely attributable to the angioplasty procedure.

Our modifications to the basic angioplasty technique enhanced its success. Further refinement of this technique in the treatment of vasospasm will make it safer in treating this serious and widespread disorder.

Intracranial arterial vasospasm remains a major cause of morbidity and mortality despite successful cerebral aneurysm surgery. Medical modes of therapy have achieved only limited success [1]. Recently, angioplasty of these narrowed vessels has been found to result in both angiographic and clinical improvements in this condition [2–4].

Inherent in the design of the cerebral angioplasty balloon catheters currently available is a limited ability to (1) turn sharp corners around acute angles and (2) selectively enter narrowed branches at a bi- or trifurcation after one major branch at that point has been dilated, as the resultant increase in flow into the one dilated branch can act to pull the balloon into it. Thus, these systems work well for angioplasty of the distal internal carotid artery, vertebral-basilar arteries, and proximal (M1) segment of the middle cerebral artery. There has been considerable difficulty in gaining access to the anterior cerebral artery, and in entering more than one branch of each middle cerebral or posterior cerebral artery. We describe two modifications of the original technique designed to help overcome these problems of branch access, and the results of cerebral angioplasty in four patients.

Materials and Methods

We use the same approach to patient selection, neurologic monitoring, and basic angiographic technique as that described by others recently for intracranial angioplasty [3, 4] of vasospasm. Briefly, a delayed ischemic neurologic deficit in a patient at risk for cerebral vasospasm [1] is first evaluated clinically. Typically, a reduced level of consciousness or coma, with or without focal neurologic signs, is present. If other causes of deterioration can be excluded (e.g., hypoxia, hydrocephalus, sepsis, complications of surgery, or repeat hemorrhage), angiography is performed to confirm the diagnosis of vasospasm [5, 6]. Routine medical measures (e.g., expansion of blood volume) should be tried first; if further deterioration occurs, angioplasty is considered.

We begin with the most symptomatic vessels (based on correlation of clinical findings with the arteriogram) and may proceed to dilate all significant stenoses (caliber reduced by ≥50% diameter) in the other territories at the same session. This is important, particularly with inaccessible vasospasm of distal and multiple branches because an increased leptomeningeal collateral supply that can result from proximal dilatation may prevent infarction. Immediately after angioplasty, angiography is performed to document the results of treatment. Procedural time, including pre- and postangioplasty studies, varies from 2 to 6 hr (depending mainly on whether secondary and tertiary branches need to be dilated or more than one territory has to be treated). Because these patients usually are quite ill, it is important to arrange adequate clinical monitoring of respiratory status and vital signs during the procedure. Because the vasospasm apparently does not recur in arteries once dilated [2–4], delayed follow-up angiography has not been performed routinely. However, if neurologic deterioration should recur some days after intracranial angioplasty, and is otherwise unexplainable (e.g., by brain CT findings or cardiorespiratory compromise), the possibility of worsening spasm in untreated vessels should be excluded angiographically. Postprocedural clinical monitoring in a neurologic intensive care unit is essential.

"Unmodified" Technique

The device predominantly used to date for cerebral angioplasty consists of a 0.85 by 4.1 mm (uninflated) silicone balloon (Interventional Therapeutics Corp., South San Francisco, CA) permanently attached either to a 2-French polyethylene nontapered catheter [3] or to a Tracker catheter (Target Therapeutics, San Jose, CA) [4]. These balloon catheters are blind-ended, have limited steering ability, and are strongly influenced by flow direction. Both can be shaped by steam, and, in the case of the Tracker catheter, some additional distal curve and stiffness can be achieved with the use of a curved-tip guidewire within the catheter. We prefer the Tracker-based system. The balloon/catheter is prepared at the time of the procedure by flushing it with nonionic contrast material to clear air bubbles. Because the silicone balloon is semipermeable, it will allow slow egress of air (over 10–20 min) during injection of contrast material into the catheter; thus, back-and-forth exchange of contrast material for air is avoided. Alternatively, air can be flushed retrogradely from the distal end by use of a 0.29-mm "vent tube" (Interventional Therapeutics Corp.) advanced to the balloon neck; contrast material injected through it will wash air back to the catheter hub, and then it is withdrawn. Once flushed, a 0.016- or 0.018-in. (0.041- or 0.046-cm) steerable tapered guidewire (Target Therapeutics) with a distal curve is advanced into the catheter to the balloon neck, through a Toughey-Borst side arm. The wire provides some axial stiffness to allow the catheter to be advanced manually. With systemic heparinization, the balloon/catheter/wire combination is delivered through a nontapered 7- to 8-French catheter already positioned in the cervical internal carotid or vertebral artery. Continuous saline perfusion of the guiding catheter is maintained around the balloon catheter. The balloon is then pushed to the proximal intracranial arterial segment. Under the guidance of digital "road-mapping" fluoroscopy, the angioplasty balloon is advanced into the first narrowed segment encountered, for example, the supraclinoid segment of the internal carotid artery, and inflated and deflated very briefly, occluding flow for not more than 8–10 sec/cycle. This is repeated until the vessel is dilated to nearly normal caliber size (determined from the preoperative angiogram if available) or until severe focal narrowings are effaced (Fig. 1). The actual injection volume is determined by fluoroscopic observation of the balloon's size and shape during inflation. The maximum inflation volume of 0.1 cm^3 yields a maximum balloon dimension of 3.5 by 12 mm. Because the premorbid caliber of the internal carotid artery

requires a greater balloon dimension for full dilatation, alternatively a 1.5-mm (uninflated diameter) silicone balloon (Interventional Therapeutics Corp.) can be used (in this vessel only), which may be inflated up to a diameter of 5–8 mm. Unlike the customary angioplasty balloons, the silicone balloon is highly deformable. Inflation pressure measurement does not appear to be necessary or useful. Usually, arteries narrowed by vasospasm dilate readily. To prevent catastrophic vascular injury, it is critical not to distend the vessel to a size greater than its premorbid caliber. It seems preferable to err on the size of slight underdilatation. Intimal tearing is not desirable in this procedure; overdistension may result in arterial occlusion [4] or rupture [3].

The catheter is then advanced further into the vessel (Fig. 1) and subsequent segments are dilated in a stepwise fashion. From this point on, usually only one branch at any bifurcation can be entered because of vessel shape, catheter shape, and preferential flow into the first dilated branch. To overcome this problem, either or both of the two maneuvers described below may be successful.

Modifications of Basic Technique

Wire-in-balloon.—This method requires no alteration of the basic balloon/catheter/wire system, but rather a change in the way it is used. If the distally curved wire is advanced to the tip of the uninflated cylindrical balloon, and then pushed slightly farther, the ballon and wire tip will deform into a curve (Fig. 2) in response to the elastic pull of the balloon. The radius of this curve will decrease as more wire is advanced. The balloon can be steered by rotating the wire. The result is a steerable balloon tip with controlled variable stiffness and variable radius curve.

With the use of road-map fluoroscopy, the balloon is positioned just proximal to the origin of the desired branch and multiple short advances are made, adjusting catheter torque and balloon curve with each sweep until the origin is slightly engaged. Next, some of the balloon tension is released by slightly retracting the wire, ideally keeping its tip near the balloon's tip. The balloon is then advanced by applying a rapid series of gentle short thrusts to the catheter (Figs. 2F–2I).

The silicone balloon tolerates this internal pressure from the wire. In one case, we were able to use a single balloon and wire to dilate the A1 and A2 portions of the anterior cerebral artery, as well as the M1 and three of the M2 branches of the middle cerebral artery. By the end of the procedure, the balloon had developed a very small leak, which did not interfere with its function. The imposed increase in wire/balloon stiffness that results from this technique is minimal and, so far, apparently safe. No angiographic evidence of vascular injury resulted from its use.

Wire-through-leak balloon.—The original Interventional Therapeutics silicone balloon/Tracker combination is assembled as above, then modified as follows. A hole is made in the exact center of the distal end of the contrast-filled angioplasty balloon with a 25-gauge needle (ideally round, rather than bevel-tipped). A 0.016- or 0.018-in. (0.041- or 0.046-cm) tapered guidewire is advanced several centimeters through the hole, enlarging it slightly. Only after the wire protrudes from the balloon should it have a curve added; otherwise it is difficult to guide the wire tip through the small balloon hole. The wire, when protruding from the balloon, largely obturates the hole. The balloon then behaves much like the unaltered angioplasty balloon, with a slight to moderate "leak" of contrast material around the wire. When the wire is withdrawn back into the catheter, the leak will be much faster, but the balloon can still be used for angioplasty, as well as for superselective angiography. The rate of balloon inflation and deflation will depend both on the hole size and force applied to the operator's syringe.

Fig. 1.—Case 1.

A, Preoperative right carotid angiogram, anteroposterior view, shows middle cerebral trifurcation aneurysm.

B, 4 days after successful clipping. Aneurysm is occluded, but there is new, severe narrowing of M1 segment, plus contralateral shift of anterior cerebral artery.

C, Angioplasty balloon (0.85 mm) is being inflated in proximal M1 segment. Note conical deformity of balloon tip (wedged in an as yet undilated portion of artery).

D, Right carotid angiogram after angioplasty of proximal two-thirds of M1 segment. Distal M1 spasm persists.

E, Immediate postangioplasty arteriogram reveals normal caliber of M1 segment and improved filling of distal branch.

A

B

C

D

E
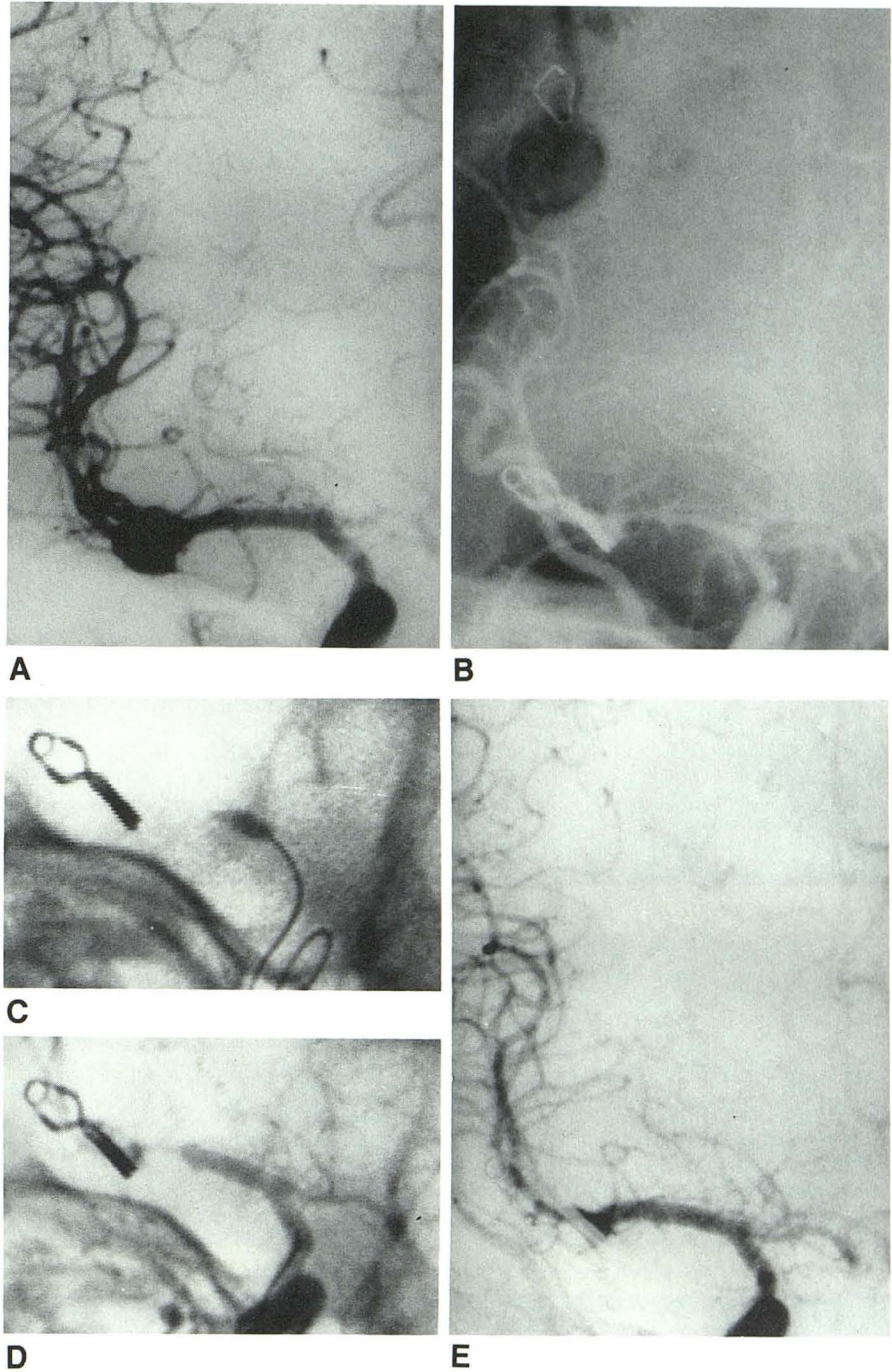
If the catheter does not advance easily, the balloon should be inflated where it lies at the origin, to dilate the vessel orifice (Fig. 3). Though this may propel the balloon backward out of the branch, subsequent engagement and entry of the vessel will have been facilitated, by even slight dilatation. This applies equally to unmodified or wire-in-balloon techniques.

This modification provides the ability to selectively steer into multiple narrowed branches otherwise not accessible. It has two drawbacks: (1) The hole enlarges with multiple guidewire movements through it, leading eventually to an inability to inflate the balloon; thus, several balloon catheters may be required to complete a procedure.

(2) Larger volumes of contrast material are delivered to ischemic brain. (The manufacturer is attempting to develop a modified balloon with a reinforced hole at its tip, which may reduce these problems.)

Results

To date, four patients have been treated for symptomatic cerebral vasospasm with intracranial angioplasty. In our first case only the unmodified technique was required, as spasm was limited to the stem of the middle cerebral artery. In
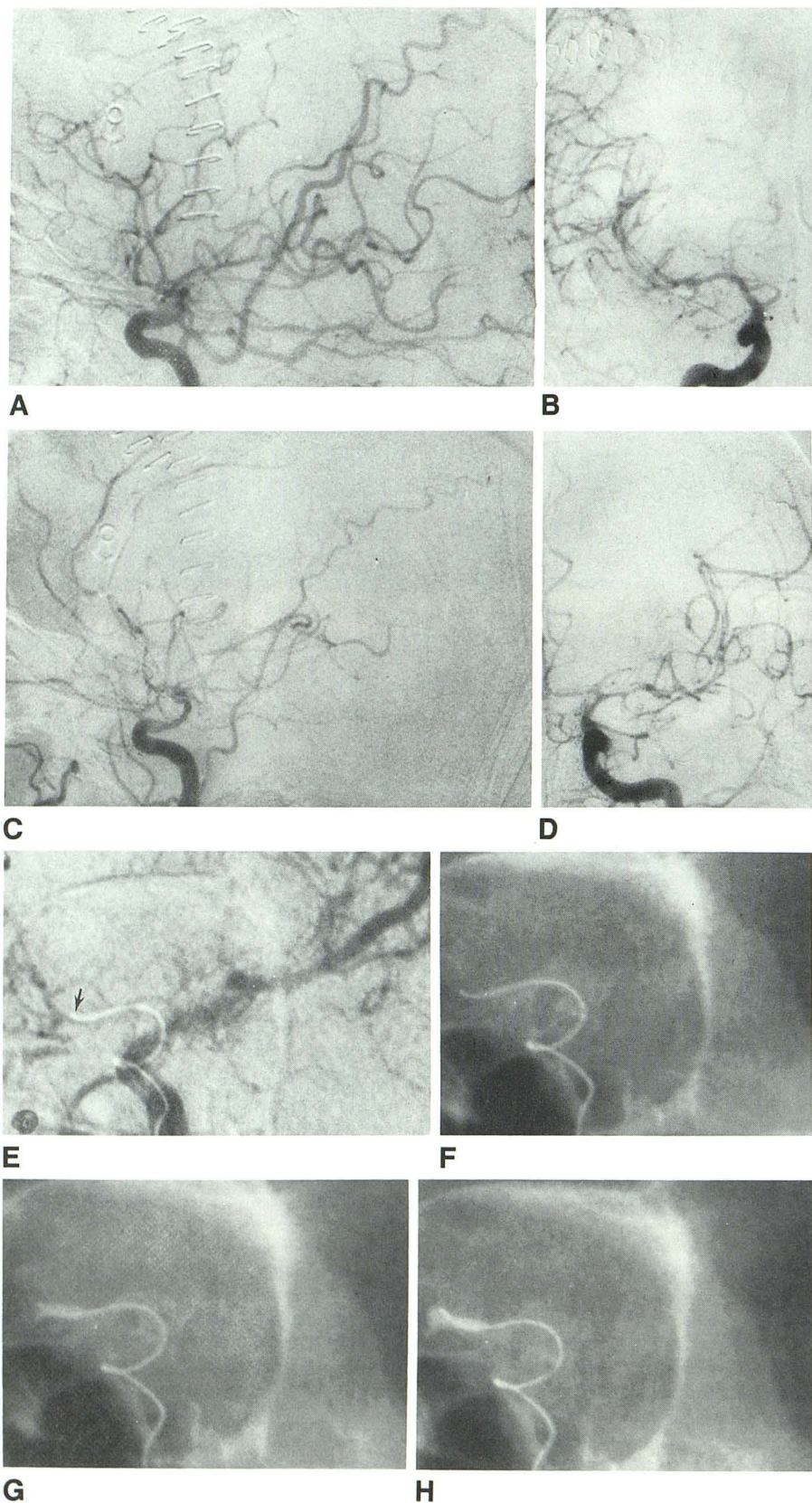
A

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Fig. 2.—Case 2.

A and *B*, Right carotid angiograms 1 week postoperatively show severe, diffuse vasospasm.

C and *D*, Left carotid angiograms 1 week postoperatively. Clipping has obliterated pericallosal aneurysm. There is severe, diffuse vasospasm.

E, Left carotid angiogram, oblique view, after partial angioplasty. Supraclinoid internal carotid artery, M1 segment, and a single M2 branch are already dilated by unmodified technique. A1 segment of left anterior cerebral artery has been entered using wire-in-balloon technique. Radiopaque wire is seen superimposed on vessels; tip of balloon is now directed into left A2 segment (*arrow*).

F–I, Sequential left orbital oblique spot films show advancement of wire-in-balloon up A2 segment with angioplasty.

Fig. 2—(continued).

J and *K*, After completion of left hemisphere angioplasties, vessel caliber and circulation time are much improved. Note persistent, severe spasm in untreated anterior cerebral territory distal to aneurysm clip.

L, Anteroposterior spot film. After angioplasty of right supraclinoid internal carotid artery, right M1 segment, and one right M2 segment "branch," right A1 (1) and A2 (2) segments were engaged and entered by using wire-in-balloon technique. Note focal curving (*arrows*) of distal wire (intraballoon segment) due to elastic pull of balloon (see text).

M and *N*, Right carotid angiogram immediately after completion of right hemisphere angioplasties. Right anterior cerebral artery was dilated to approximate level of clip; distal untreated spasm persists.

J

K

L

M

N
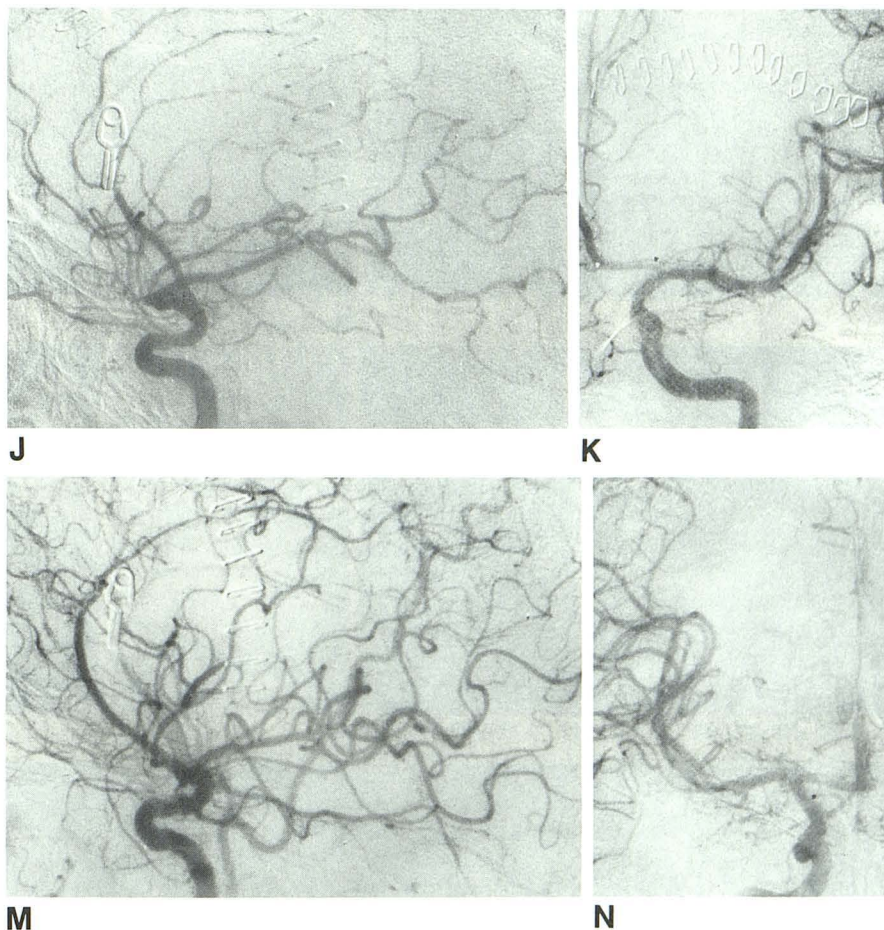
subsequent cases, spasm was both proximal and distal and involved multiple branches. Each procedure was begun by using the unmodified technique; access to the anterior cerebral and branches of middle cerebral arteries necessitated use of one of the modified techniques. In all, 27 arterial stems and branches were dilated in the four patients. In all cases, the progressive clinical deterioration of the patients ceased with the angioplasty. There were no significant complications.

Case Reports

Case 1

A 50-year-old man developed delayed obtundation and mild hemiparesis 3 days after the clipping of an unruptured right middle cerebral artery aneurysm (Fig. 1A). CT showed minimal hemisphere swelling without hematoma or hydrocephalus; a 6-hr course of medically induced hypertension and volume expansion produced no improvement. Angiography revealed severe vasospasm limited to the M1 segment of middle cerebral artery (Fig. 1B), presumably caused by surgical manipulation during dissection. The angioplasty balloon was introduced, and dilatation of the M1 segment was carried out using the unmodified angioplasty technique (guidewire retained within the catheter proper). Successive dilatations and advancements (Figs. 1C and 1D) resulted in a normal-caliber vessel (Fig. 1E). The patient showed immediate moderate improvement in level of consciousness

and was talking the next day. He was neurologically intact when discharged 1 week later.

Case 2

A 55-year-old man experienced a rapidly decreasing level of consciousness 1 week after the clipping of a ruptured pericallosal artery. A 2-day course of medical therapy had failed, and he was referred for angioplasty. Angiography revealed diffuse vasospasm of the bilateral anterior circulation: it was particularly severe in both anterior cerebral arteries (Figs. 2A–2D). At the start of the procedure the patient was mute to all stimuli and withdrew his limbs to pain. Angioplasty of the bilateral distal internal carotid artery as well as the bilateral middle cerebral M1 segment was performed with the unmodified technique. Next, a single M2 branch was dilated on each side; then, subsequent M2 branches as well as bilateral anterior cerebral A1 and A2 segments were entered successively, using the wire-in-balloon technique (Figs. 2E–2N). By 48 hr, the patient was uttering confused phrases and occasionally followed commands. He showed slow, continued improvement. Two months later, he was fully oriented, able to dress and feed himself, and was ambulating with a walker.

Case 3

A 60-year-old woman developed severe progressive obtundation and was comatose with left decerebration 1 week after rupture of a
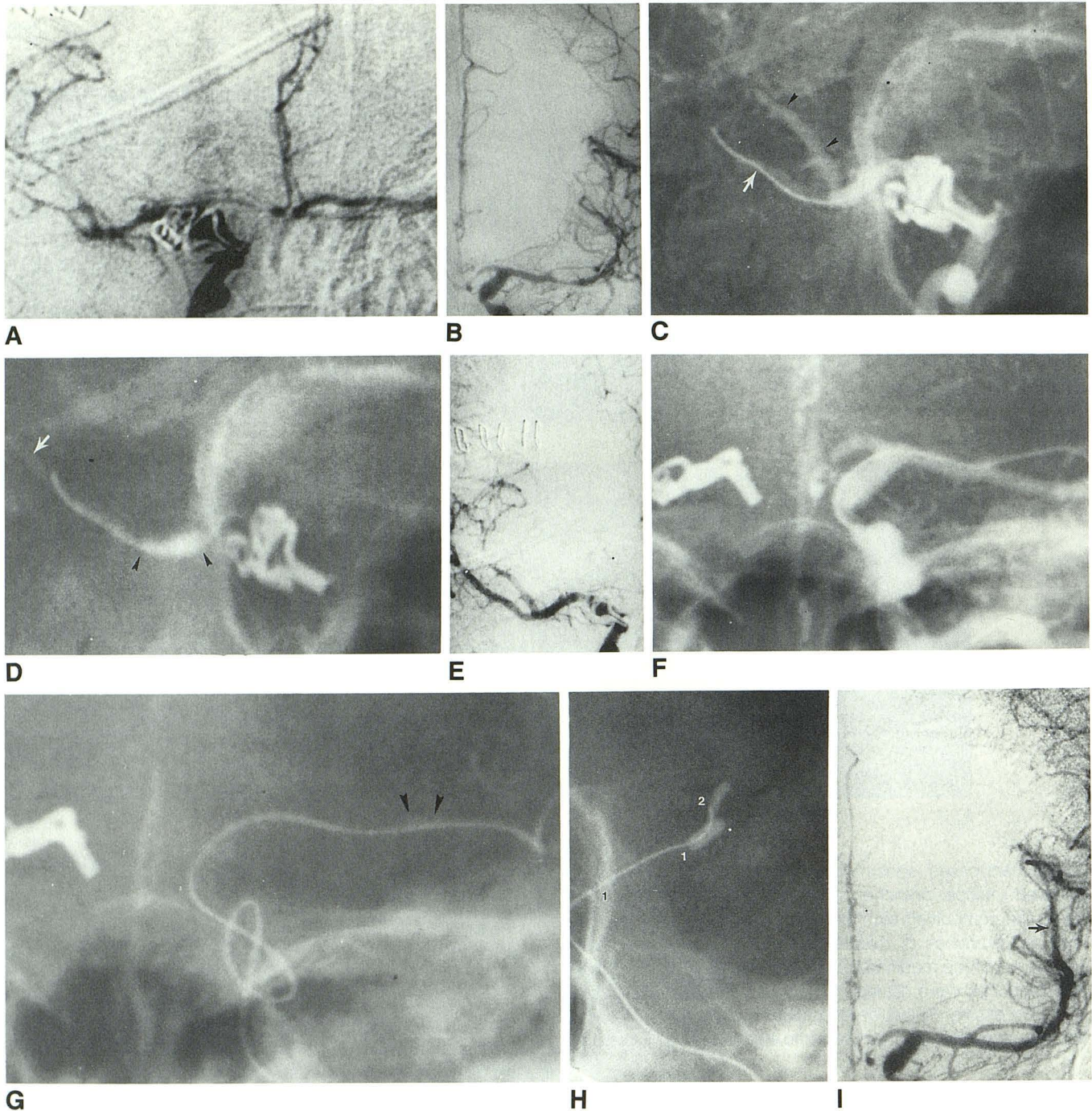
Fig. 3.—Case 3.

A, Right carotid angiogram, oblique view, with contralateral carotid compression 11 days after successful clipping of right posterior communicating aneurysm. There is severe, diffuse vasospasm.

B, Left carotid angiogram, same day. Spasm spares M1 segment, but involves all three M2 segment "branches" of left middle cerebral artery, as well as A1 and A2 segments of left anterior cerebral artery.

C, Right carotid angiogram, oblique view. Right supraclinoid carotid artery, right M1 segment, and uppermost (*arrowheads*) of two main M2 segments are already dilated by unmodified technique. Wire had been advanced through leak-balloon hole, well out lowermost branch (*arrow*), to allow angioplasty to sylvian fissure portions.

D, Inflation of leak balloon with contrast material (*arrowheads*) results in dilatation of proximal segment of this branch. By advancing or withdrawing balloon along wire, remaining narrowed portions were treated. Note faint opacification of distal branch due to leaking contrast material (*arrow*).

E, Right carotid angiogram after completion of right middle cerebral angioplasties. Note untreated spasm of right anterior cerebral A1 segment.

F, Left carotid angiogram after dilatation of centermost portions of three M2 branches of left middle cerebral artery with unmodified technique. Wire has been advanced out of balloon hole to allow selective placement in uppermost M2 branch.

G, Inflation of leak balloon (*arrowheads*) results in dilatation of segment of M2 branch. Remainder was treated by moving balloon along wire and inflating narrow segments.

H, Left oblique spot film. Origin of uppermost M2 branch had been skipped initially, but was angioplastied successfully after distal portions. Fresh balloon catheter was required, as leak hole of first catheter had enlarged excessively with use. By initially using wire-in-balloon technique (without a hole), curved tip has been steered into narrowed vessel origin. Note how balloon has deformed to fit both M1 (1) and "branch (2)" sections it crosses during inflation.

I, Left carotid angiogram after angioplasty of all three M2 "branches," as well as more distal portions in sylvian fissure (*arrow*). Distal part of left A1 segment was not accessible owing to acute angulation of origin off internal carotid artery.

right posterior communicating artery aneurysm. A 48-hr regimen of medical management had failed. Angiography revealed severe, diffuse vasospasm in the anterior circulation (Figs. 3A and 3B). We performed balloon angioplasty (using the unmodified technique) of both supraclinoid carotid arteries, the M1 segments of the middle cerebral artery bilaterally, as well as a single M2 segment beyond the trifurcation of the middle cerebral artery on each side. Secondary branches of the middle cerebral artery were entered selectively using the wire-through-leak balloon technique and successfully dilated (Figs. 3C–3I). This technique failed to provide access to the anterior cerebral artery owing to the very acute angle of its origin off the internal carotid artery in this patient. The 0.016-in. (0.041-cm) guidewire could engage the A1 segment, but the balloon could not be made to follow without "kicking out" the guide. A direct carotid puncture was used for access because of a severely tortuous aortic arch; the procedure was complicated by a neck hematoma necessitating endotracheal intubation. The patient showed no significant early improvement, but deterioration ceased after the procedure; at 2 weeks she was hemiplegic on the left, but speaking. After 3 months, she could converse, was oriented to herself and her surroundings, but remained hemiplegic.

Case 4

A 48-year-old man presented with subarachnoid hemorrhage, drowsiness, and aphasia (Fig. 4A); clipping of a ruptured left middle cerebral trifurcation aneurysm was performed the same day. The next day transtentorial herniation resulted from brain swelling; his pupils were fixed and dilated, and an emergency left frontal lobectomy was required. Postoperatively, gradual improvement was seen over the next 4 days, but right-sided weakness and dysphasia persisted. One week after presentation a right arm monoplegia was noted and worsening drowsiness and dysphasia developed. CT the next day revealed a new 3-cm infarct in the left parietal lobe. This had developed despite aggressive systemic blood volume and pressure management as well as pharmacologic prophylaxis with a calcium channel blocker (nimodipine). Angiography 9 days after hemorrhage revealed severe proximal and distal vasospasm in the left middle cerebral and in the proximal left anterior cerebral territories (Fig. 4B). Accordingly, angioplasty was performed, first in the M1 segment using the unmodified technique; then, each of the main trifurcation branches was

entered selectively using the wire-in-balloon technique. These latter branches were dilated out under the operculum. Finally, the A1 segment of the left anterior cerebral artery was entered and dilated using the wire-in-balloon technique, as was the left A2 segment in its first proximal 2 cm (Fig. 4C). There were no complications. Within 24 hr the patient had definite improvement in level of consciousness, was more alert, and had some return of speech. Right arm movement returned within 3 days. Steady improvement was seen over the subsequent month.

Discussion

Intracranial arterial angioplasty for vasospasm is in its infancy, and further technical advances are expected. The initial reports of successful dilatation of the proximal stems of some major cerebral arteries [2–4] provide an encouraging basis for attempts to extend the technique to the more distally affected branches and those more difficult to access, such as the anterior cerebral complex. The simple modifications we describe here facilitate this access.

The techniques described are complementary. The wire-through-leak balloon provides the greatest steerability when entering narrow branches owing to the leading guidewire tip. However, the distal guide is so floppy that several centimeters of length must be advanced for support before the balloon can be pushed into the vessel. The wire-in-balloon, in contrast, provides a slightly stiffened, curved balloon; while less able to select the narrowest branches, it can provide more stable access to vessel origins that are short and sharply angled, such as the A1 segment of the anterior cerebral artery. It also has the advantage of requiring no additional preparation time, and is our first-choice maneuver when greater selectivity is desired.

The best correlation with clinical deficits is with the "diffuse" type of vasospasm on angiography—involving both proximal and distal segments and multiple vessels [5, 6]. The more common "segmental" variety, usually involving proximal stems in a limited distribution, correlates less well with neu-
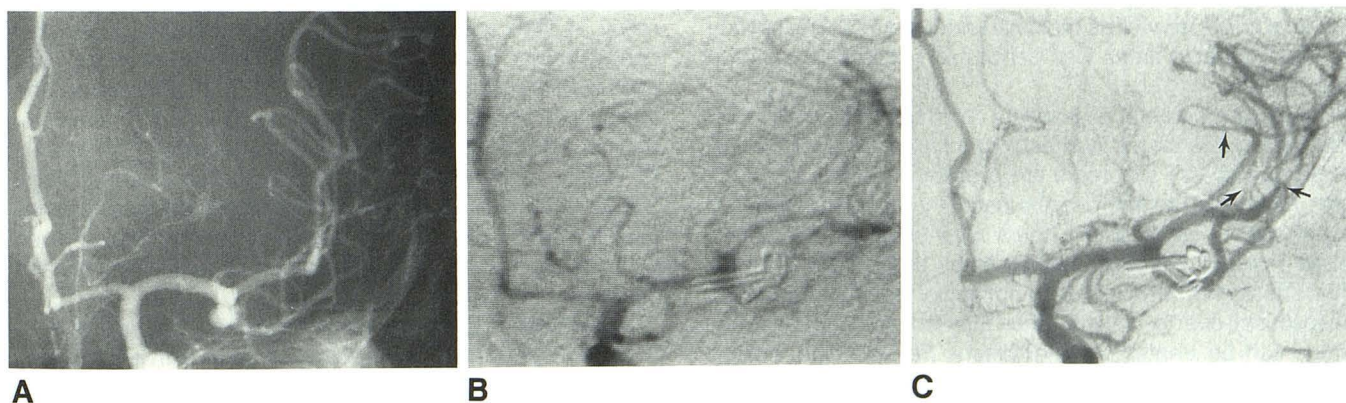

A **B** **C**

Fig. 4.—Case 4.
A, Preoperative left carotid angiogram, anteroposterior view, shows middle cerebral trifurcation aneurysm. Note shifted anterior cerebral arteries owing to combination of sylvian subarachnoid hematoma and left hemisphere swelling. Successful clipping was confirmed by angiogram the next day.
B, 10 days later, left carotid angiogram shows severe vasospasm involving both proximal and distal portions of middle cerebral artery territory; left anterior cerebral artery is also involved.
C, Immediate postangioplasty appearance. Unmodified and wire-in-balloon techniques were used to dilate A1 and proximal A2 segments, M1 segment, and all three middle cerebral artery trifurcation branches, well out into sylvian fissure. Note abrupt tapering at limits of angioplasty (*arrows*).

rologic deterioration. Thus, the ability to access and treat distal and multiple branches beyond the circle of Willis likely will be critical to the successful application of angioplasty in many symptomatic patients. On preangioplasty angiography, it may sometimes be difficult to decide whether the distal narrowing is due to actual distal disease or is simply the result of reduced flow owing to proximal stenosis. Dilatation of limited, truly proximal spasm may, through improved flow, lead to an improved caliber of the distal vessel [2, 4]. However, when vasospasm is diffuse, distal narrowing persists after proximal dilatation (as in cases 2–4).

It is logical that the angioplasty procedure should encompass as much affected territory as feasible, working proximally to distally, with serial angiographic assessments of progress. In the case of distal spasm (beyond the M1, A1, and P1 segments), the question of how far distally the angioplasty should be carried out cannot yet be answered. The caliber of most accessible major cortical branches of the middle [7], anterior, and posterior cerebral arteries in the normal adult should permit passage of the 0.85-mm balloon. Very severe narrowing of even the most proximal arterial stems, however, occasionally can prevent balloon entry. In one instance (case 3), this problem was overcome by first advancing the guide alone into the vessel (wire-through-leak balloon), which itself dilated the vessel slightly, then by advancing the uninflated balloon to dilate it further. Risk of vessel rupture might increase when far distal vessels are treated unless extreme care is taken to limit balloon inflation to a size less than or equal to the premorbid luminal caliber. Interestingly, despite untreated persistent spasm far distally, dilatation of stems and major branches more proximally led to early improvement in two patients (cases 2 and 4).

We initially have been hesitant to dilate the arterial segment at the site of the clipped aneurysm for fear that the wall here may not withstand the dilatation or that the clip might be displaced. When the aneurysm location was distal (as in case 2), considerable dilatation proximal to the level of the clip could be done. However, because most aneurysms are located proximally, the need for treatment may mandate dilatation at the clip site. For example, in case 4, severe spasm was seen at the clip site at the trifurcation of the middle cerebral artery, as well as proximal and distal to it. Angioplasty of these affected areas was performed successfully without complication.

We believe cerebral angioplasty could be considered when the appropriate clinical and angiographic features of the delayed vasospasm syndrome are present and medical measures have failed to improve the patient (i.e., volume loading, hypertension) within a few hours after onset of symptoms. Major residual neurologic deficits (such as in cases 3 and 4) might be averted with more prompt angioplasty. Symptomatic vasospasm due to causes other than subarachnoid hemorrhage can be treated by angioplasty. For example, spasm after vessel manipulation, either due to surgical dissection (as in our case 1) or induced by microcatheters during endovascular interventional procedures [8], has responded favorably. Further verification of the value of cerebral angioplasty awaits the results of a larger clinical series. A controlled randomized

trial might be difficult to justify, for several reasons. The natural history of severe vasospasm when well established is usually progressively downhill. The available evidence [2–4] for significant early benefit from angioplasty appears strong, with approximately two thirds of patients responding favorably, usually within 48 hr. Finally, the complication rate for cerebral angioplasty, while as yet uncertain, is likely sufficiently low [2–4] to support the use of the technique as a lifesaving measure, in the absence of other efficacious treatment.

The presence of an unclipped ruptured aneurysm is likely not an absolute contraindication [2], but would seem to be a cause for concern and great caution. All our patients were treated postoperatively after successful clipping of an aneurysm. Most surgeons will not operate in the presence of severe vasospasm, and likely it will occasionally be necessary to perform angioplasty preoperatively. It must be recognized that abrupt changes in arterial pressure and flow could put the unoperated ruptured aneurysm at risk of rerupture during or after angioplasty. An extensive recent cerebral infarction in an involved territory is probably a contraindication, as it may become hemorrhagic after angioplasty [3]. However, smaller infarctions may not pose a prohibitive risk (as in case 4), particularly when the patient shows continued global deterioration due to vasospasm in other vascular territories. Other complications described to date are few, but include vessel rupture [3] and occlusion [4].

Vasospasm may be a misnomer, as it appears that simple muscular contraction may not be responsible for the arterial narrowing observed in this syndrome [9, 10]. Histologic findings in this setting have been variable, but there is evidence that an arteriopathy develops after subarachnoid hemorrhage. Pharmacologic manipulations [9] have proved unsuccessful. Mechanical dilatation results in a stable increase in luminal diameter, without evidence of intimal injury or perforator occlusion (based on clinical and radiographic criteria) at sites of successful angioplasty using this balloon catheter system. Presumably, the dilatation results in stretched internal elastic laminae and tunicae media. Without an additional injury (i.e., a second subarachnoid hemorrhage), there is no impetus for further arterial pathologic reaction, and vasospasm does not recur. In vitro angioplasty of pharmacologically constricted canine basilar artery [11] to 100–104% of the native size caused mild histologic changes including media thinning and dehiscence of the internal elastic lamina (sparing perforator origins), without intimal fracture. After angioplasty, the vessels no longer responded to a vasoconstrictive pharmacologic agent. Similarly, human cadaveric middle cerebral arteries dilated by angioplasty to increase the lumen by 30% showed compressed intima and stretched internal elastic laminae and media, but no intimal tearing [12].

As experience with intracranial angioplasty grows, it is likely that the demand for the procedure will grow with it. Familiarity with intracranial catheterization techniques and balloon devices is a prerequisite to its safe performance. The widespread nature of the problem of vasospasm may well require acquisition of these specialized skills by most major centers. Future work should address these unresolved issues: the

time "window" after symptom onset for optimal, safe, and efficacious treatment; how far distally it is necessary or desirable to carry out angioplasty in cases of diffuse vasospasm; and identification of absolute contraindications to cerebral angioplasty.

Addendum

Recently, a fifth patient, who had severe bilateral anterior cerebral vasospasm, was successfully treated with the wire-in-balloon technique. Both pericallosal arteries were accessed via the left A1 segment and dilated to a level above the genu of corpus callosum.

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