AJNR

Reply

Shinichi Nakano

AJNR Am J Neuroradiol 1999, 20 (10) 2022-2023
http://www.ajnr.org/content/20/10/2022

Safety of Angioplasty for Intracranial Artery

We read with interest the article ''Intracranial Angioplasty: Experience and Complication'' by Takis et al (1) in the October 1997 issue of the *American Journal of Neuroradiology*. The authors performed angioplasty in intracranial arteries in 10 patients with TIA or minor stroke and reported a relatively high rate of intraprocedural complications, including vasospasm (63%), dissection (25%), and compromise of perforating vessels (25%). We speculate that perhaps technical differences in their procedures may have contributed to their unfavorable results. In our previous study of angioplasty for the basilar artery (2), we suggested less invasive techniques, such as lowering inflation pressure (<3 atm), dilating fewer times (once or twice), and using a smaller balloon catheter (2.0 mm). It is also important to inflate the balloon catheter slowly. These techniques could minimize the intimal damage and prevent occlusion of the perforating artery and excessive dissection. In addition, significant hemodynamic disturbance is reported to occur only with more than 70% stenosis; further dilatation of less than 50% stenosis may not always be necessary to achieve adequate cerebral blood flow. The purpose of angioplasty should be to provide sufficient perfusion to reduce ischemic symptoms, not to achieve an angiographic cure, which is often associated with an unacceptable complication rate. Nevertheless, the restenosis rate of our techniques is to be determined by a larger number of patients and long-term follow-up data. In our limited experience with 25 cases that have been followed up more than one year, the restenosis rate has been less than 10% (two cases). This is relatively low when compared with the reported data regarding angioplasty for the intracranial artery (30% [3]) and angioplasty for the coronary artery. Most important, our complication rate, based on our 30 cases, is much lower (6%, unpublished data) than that reported by Takis et al.

In general, mechanical vasospasm is temporary and responsive to vasodilators, as suggested by the authors. Persistent vasospasm is mostly related to arterial dissection, particularly the dissection of small vessels, such as intracranial arteries, and may not always be apparent on cerebral angiograms. Because silent dissection does not commonly cause stroke, systemic heparinization administrated for 3 days after angioplasty is the preferred treatment of patients with potential arterial dissection in our institution.

Toshihiro Ueda, MD, PhD
William T. C. Yuh, MD
Division of Neuroradiology and MRI Center
Department of Radiology
The University of Iowa College of Medicine
Iowa City, IA

References

1. Takis C, Kwan E, Pessin M, Jacobs D, Caplan L. **Intracranial angioplasty: experience and complication.** *AJNR Am J Neuroradiol* 1997;18:1661–1668
2. Nakatsuka H, Ueda T, Ohta S, Sakaki S. **Successful percutaneous transluminal angioplasty for basilar artery stenosis: technical case report.** *Neurosurgery* 1996;39:161–164
3. Mori T, Mori K, Fukuoka M, et al. **Percutaneous transluminal angioplasty for total occlusion of middle cerebral arteries.** *Neuroradiology* 1997;39:111–116

Reply

We appreciate the interest of Drs Ueda and Yuh in our article *Intracranial Angioplasty: Experience and Complication.* We agree that submaximal percutaneous transluminal angioplasty (PTA) of intracranial arteries with balloons inflated slowly and at low pressure is probably a safer technique, because it is less traumatic to the intima and media of treated arteries. Nevertheless, only careful long-term follow-up in a larger series of patients can determine the efficacy and restenosis rate with this approach.

Eddie Kwan, MD
New England Medical Center
Boston, MA

Direct Angioplasty for Acute Occlusion of Intracranial Artery

We have read with interest the article *Direct Percutaneous Transluminal Angioplasty for Acute Middle Cerebral Artery Occlusion* by Nakano and colleagues (1). The authors report their experience using direct percutaneous angioplasty (PTA) as the sole means of treating 10 patients with acute middle cerebral artery (MCA) occlusion when the initial CT scans demonstrate early ischemic changes or involvement of lenticulostriate arteries or both. The authors' rationale for choosing direct PTA alone to establish blood flow without using thrombolysis is based on the high risk of hemorrhagic complications in this group of patients. The authors believe that avoiding thrombolytic therapy can reduce such a risk. The angiographic success rate in their patients was relatively high (80%), and there were no hemorrhagic complications; however, only two patients had full recovery. With 20% clinical recovery and 20% distal embolization, the rationale for their method becomes debatable despite a high rate of angiographic success without hemorrhagic complications.

We wonder whether the authors may have overlooked the fundamental pathophysiology that causes hemorrhagic complications during acute ischemic stroke. Patients with early ischemic findings on initial CT scans have a high risk of hemorrhage after re-established blood flow primarily because of the high incidence of reperfusion of irreversibly damaged ischemic tissue. The thrombolytic agent

can contribute to but is not the primary cause of hemorrhagic complications (ie, reperfusion of dead tissue). The most effective way to prevent such complications is either to avoid reperfusion of irreversibly damaged tissue or to recanalize the occluded vessel as early as possible. In some patients, the blood flow of the cortex in the distal MCA territory can be rescued by recanalization of the occluded M1 segment by using direct angioplasty. Angioplasty alone, however, will not dissolve the clot nor re-establish the blood flow effectively, particularly in the perforators, but will further propagate the clot distally. Therefore, the relatively low rates of hemorrhage and clinical recovery suggest that their technique of performing angioplasty alone may not be as effective in re-establishing the blood flow. If the authors believe that early ischemic findings on the initial CT scan suggest irreversibly damaged tissue and a high risk of hemorrhage, then early interventional treatment, including PTA, should not be performed in patients who have such findings.

One important question in the treatment of acute stroke is whether we are treating reversible ischemia. Our previous reports suggest that reversibility of ischemic tissue can be assessed by single-photon-emission CT of pretreatment CBF, which can help in the selection of appropriate patients for thrombolysis by reducing hemorrhagic complications and improving outcome (2, 3). Our previous experience also suggests that a combination of thrombolysis and angioplasty is effective in failed thrombolysis cases or reocclusion cases (4). We strongly believe that angioplasty is an effective option in reperfusion therapy for acute ischemic stroke and can shorten the duration of ischemia and improve the success rate of recanalization. Most importantly, the purpose of angioplasty should be to improve the neurologic systems of stroke patients by increasing CBF, not to improve angiographic results.

Toshihiro Ueda, M.D., Ph.D.
William T.C. Yuh, M.D.
Division of Neuroradiology and MRI Center
Department of Radiology
The University of Iowa College of Medicine

References

1. Nakano S, Yokogami K, Ohta H, Yano T, Ohnishi T. **Direct percutaneous transluminal angioplasty for acute middle cerebral artery occlusion.** *AJNR Am J Neuroradiol* 1998;19:767–772
2. Ueda T, Hatakeyama T, Kumon Y, Sakaki S, Uraoka T. **Evaluation of risk of hemorrhagic transformation in local intra-arterial thrombolysis in acute ischemic stroke by initial SPECT.** *Stroke* 1994;25:298–303
3. Ueda T, Sakaki S, Yuh W, Nochide I, Ohta S. **Outcome in acute stroke with successful intra-arterial thrombolysis and predictive value of initial SPECT.** *J Cereb Blood Flow Metab*1998:(in press)
4. Ueda T, Hatakeyama T, Kohno K, Kumon Y, Sakaki S. **Endovasular treatment for acute thrombotic stroke of the middle cerebral artery: local intra-arterial thrombolysis combined with percutaneous transluminal angioplasty.** *Neuroradiology* 1997;39: 99–104

Reply

We appreciate the interest of Ueda and Yuh in our report about direct percutaneous transluminal angioplasty (PTA) for acute middle cerebral artery (MCA) occlusion. To our regret, however, they misinterpreted our thesis and results. We reported that four (57%) of seven patients with embolic MCA trunk occlusion showed marked clinical improvement. All patients had early ischemic findings on the initial CT scan, however, and lenticulostriate artery (LSA) involvement, both of which have been reported to be predictive signs of hemorrhagic complications after thrombolytic therapy (1, 2). This good result indicates that early ischemic findings on the initial CT don't always suggest irreversibly damaged tissue.

In patients with these findings, urgent recanalization should be undertaken prior to the onset of irreversible brain damage. In patients with embolic MCA trunk occlusion, the embolus is often so large that it is resistant to thrombolysis and mechanical crushing of the embolus by direct PTA is preferred to time-consuming thrombolytic therapy. Our rationale for choosing direct PTA for these patients is based on the high risk of hemorrhagic complication when time-consuming high-dose thrombolytic therapy is performed. We chose direct PTA in order to achieve rapid recanalization, not to avoid using thrombolytic agents. We agree with Ueda and Yuh that angioplasty is an effective option in reperfusion therapy for acute ischemic stroke and it can achieve rapid recanalization (3).

In patients with embolic MCA trunk occlusion, conservative treatment often leads to extended space-occupying cerebral edema or massive intracerebral hemorrhage owing to late spontaneous recanalization after complete damage of the vessel wall (4). Even if most of the ischemic tissue cannot escape cerebral infarction, therapeutic recanalization might be effective if recanalization could be performed without hemorrhagic complications and the goal of rehabilitation could be improved. The purpose of recanalization therapy should be to improve clinical outcome, not solely to achieve an excellent full recovery. We have never aimed to improve angiographic results. We do aim to improve clinical outcome.

In our study, three (43%) of seven patients with embolic MCA occlusion had cerebral infarctions in spite of rapid recanalization, suggesting irreversible ischemic damage. In these three patients, however, neither space-occupying cerebral edema nor massive intracerebral hemorrhage was found in the course of treatment because of the rapid recanalization prior to the damage of the vessel wall. Rehabilitation of these three patients went well and we believe that their clinical outcome was improved by the urgent recanalization therapy.

We have also demonstrated that direct PTA alone could achieve complete recanalization in five (71%) of seven patients with embolic MCA occlusion. Crushed fragments of the embolus migrate distally and often lyse spontaneously, resulting in complete recanalization without thrombolysis. In the other two patients, additional thrombolysis was required because of the distal embolization. Although distal embolization by crushed fragments is a noteworthy problem of direct PTA for cerebral embolism, thrombolysis of these fragments is likely to be easy with small amounts of thrombolytic agents. We agree with Ueda and Yuh that a combination of angioplasty and thrombolysis is effective in some patients. In order to recanalize the occluded vessel as early as possible, direct PTA and subsequent thrombolysis of crushed thrombi should be effective.

Angioplasty is effective in patients with atherothrombotic stroke, particularly in failed thrombolysis or reocclusion cases; however, in patients with atherothrombotic MCA branch occlusion, sufficient arterial patency was not achieved with the minimum dilatation force of 2 to 3 atm because of the small diameter of the vessel.

In summary, angioplasty is an effective option in reperfusion therapy for acute MCA occlusion, particularly in patients with atherothrombotic stroke. Even in patients with embolic MCA occlusion, when early ischemic findings and LSA involvement is present, urgent recanalization by direct PTA should be performed and additional thrombolysis may be required in some patients.

Shinichi Nakano
Department of Neurosurgery
Junwakai Memorial Hospital
Miyazaki, Japan

References

1. Yokogami K, Nakano S, Ohta H, Goya T, Wakisaka S. **Prediction of hemorrhagic complications after thrombolytic therapy for middle cerebral artery occlusion: value of pre- and post-therapeutic computed tomographic findings and angiographic occlusive site.** *Neurosurgery* 1996;39:1102–1107
2. Larrue V, von Kummer R, del Zoppo G, Bluhmki E. **Hemorrhagic transformation in acute ischemic stroke. Potential contributing factors in the European Cooperative Acute Stroke Study.** *Stroke* 1997;28:957–960
3. Nakano S, Yokogami K, Ohta H, Goya T, Wakisaka S. **Direct percutaneous transluminal angioplasty for acute embolic middle cerebral artery occlusion: report of two cases.** *Angiology* 1997;6:254–256
4. von Kummer R, Meyding LU, Forsting M, et al. **Sensitivity and prognostic value of early CT in occlusion of the middle cerebral artery trunk.** *AJNR Am J Neuroradiol* 1994;15:9–15

In Re: Reversible Ischemia Determined by Xenon-Enhanced CT after 90 Minutes of Complete Basilar Artery Occlusion

In their case report, *Reversible ischemia determined by xenon-enhanced CT after 90 minutes of complete basilar artery occlusion*, Levy et al describe a patient with acute basilar artery occlusion whose right occipital lobe remained viable after reperfusion, despite 90 minutes with blood flow to this region reduced to 6 mL /100 g per minute (1). This finding—the rationale for their report—suggests a lower threshold for reversible ischemia at 90 minutes (at least in the posterior circulation) than the 10–12 mL /100 g per minute that is generally accepted.

The basis for their diagnosis of reversible ischemia, however, is the finding of resolution of hypoattenuation in the medial right occipital lobe on a CT scan done 12 days after stroke, as compared with a scan obtained 2 days after stroke. The authors do not provide a late CT scan (2 to 3 months after ictus), an MR scan, or an autopsy report that documents that cavitation has not subsequently evolved. Such documentation is necessary to prove that the normal attenuation coefficients found at 12 days were because of preservation of normal tissue rather than the result of a more likely phenomenon—fogging of an evolving infarction (2–4).

Infarct fogging, the apparent normalization of the CT scan hypodensity associated with an acute stroke, typically occurs between the second and third weeks after infarction. It is believed to be caused by the capillary proliferation and macrophage invasion into infarcted brain parenchyma that occurs after the resolution of acute edema but before the development of tissue cavitation.

Michael H Lev, MD
Director of Emergency Neuroradiology
Department of Radiology
Massachusetts General Hospital

Robert H Ackerman, MD
Director of the Neurovascular Laboratory
Departments of Radiology and Neurology
Massachusetts General Hospital

References

1. Levy EI, Scarrow AM, Kanal E, Rubin, G, Yonas H, Kirby L. **Reversible ischemia determined by xenon-Enhanced CT after 90 minutes of complete basilar artery occlusion.** *AJNR Am J Neuroradiol* 1998;19:1943–1946
2. Skriver EB, Olsen TS. **Transient disappearance of cerebral infarcts on CT scan, the so-called fogging effect.** *Neuroradiology* 1981;22:61–65
3. Becker H, Desch H, Hacker H, Pencz A. **CT fogging effect with ischemic cerebral infarcts.** *Neuroradiology* 1979;18:185–192
4. Ackerman RH, Lev MH, Alpert NM, et al. **Infarct fogging on CT: prevalence, features, and PET/MRI correlates.**In: Proceedings of the 28th Annual Meeting of the American Society of Neuroradiology; 1990; Los Angeles, CA

Reply

In regard to the comment by Lev and Ackerman concerning our article, *Reversible Ischemia Determined by Xenon-Enhanced CT After 90 Minutes of Complete Basilar Artery Occlusion*, we agree that

the fogging effect may have initially introduced error into our ability to define infarction. Nonetheless, subsequent studies not cited in this article demonstrated retained tissue volume. Additionally, it must be noted that hemorrhage transformation of infarcted tissue occurred only on the left, where the PCA territory remained occluded despite attempted thrombolysis. The fact that hemorrhagic transformation was absent on the right, with Xe/CT and angiographic evidence of normal parenchymal blood flow, leads us to believe that this tissue was viable with intact perfusion regulatory mechanisms.

It should be clarified that the region with flow values less than 6 cc/100 g/min were primarily within the deep white matter of the occipital lobe. These therefore did not represent the more traditional mixed cortical flow values for the threshold of infarction that are more commonly referred to in the physiologic literature.

Howard Yonas, M.D.
Peter J. Jannetta, M.D.
Elad Levy, M.D.
Department of Neurological Surgery
University of Pittsburgh
School of Medicine

Pathogenesis of Syringomyelia

I read with great interest the article, *The Presyrinx State: A Reversible Myelopathic Condition that May Precede Syringomyelia,* by Nancy J. Fischbein et al (1). Although their theory of pathogenesis for the formation of the presyrinx state and syringomyelia is provocative, several areas need further clarification. The authors propose, as have others, that increased pressure in the subarachnoid space of the spinal canal in patients with Chiari malformation or other causes of relative blockage of CSF flow at the foramen magnum would cause the CSF to flow along the perivascular spaces and accumulate either in the central canal, causing syringomyelia, or in the cord substance itself, causing a ''presyrinx'' state. Nonetheless, as has been pointed out by others, increased pressure applied to a fluid (CSF) surrounding a distensible semisolid structure (the spinal cord) would have a tendency to compress that structure rather than force fluid into it (2). The only way there can be net flow of fluid into the spinal cord from the CSF surrounding it is if there is a pressure differential from the subarachnoid space to the central canal or to the spinal cord substance.

The authors state it has been proved that CSF flows from the subarachnoid space into the perivascular spaces of the spinal cord and from there along the interstitial spaces toward the central canal. For support of this, the authors reference Stoodley et al, among others (3). To summarize the experiment of Stoodley et al briefly, they injected horseradish peroxidase into the subarachnoid space of sheep and, by reducing arterial pulsations by ligating the brachiocephalic trunk, they demonstrated a reduction in the distribution of the horseradish peroxidase tracer through the perivascular spaces and central canal. They felt this experiment supported the hypothesis of arterial-driven flow of fluid from the subarachnoid space into the perivascular spaces across the interstitial space and into the central canal. In my opinion, however, this does not prove that there is bulk flow of fluid in that direction. It only proves that the cerebrospinal fluid is anatomically continuous with the central canal through a series of perivascular and interstitial spaces (4) and that when you impart energy (arterial pulsations) to a solid (horseradish peroxidase tracer) in solution (CSF) that solid will be distributed more rapidly and over a greater volume than if no energy is imparted to the mixture.

Liquids flow along the path of least resistance and from regions of high pressure to regions of low pressure. In order for there to be a net flow of fluid from the subarachnoid space through the very small (high resistance) perivascular and interstitial spaces into the very small (high resistance) central canal of the cord, and then possibly out the central canal at the level of the obex, there must be a significant pressure differential between the cerebrospinal fluid surrounding the spinal cord and the pressure in the central canal. Or, the resistance to flow from the subarachnoid space surrounding the spinal cord to the cisterna magna or basilar subarachnoid space must be greater than the combined resistances of the perivascular spaces, interstitial spaces, and central canal. This seems unlikely.

As we have previously proposed, we believe that the spinal cord is a net producer of extracellular fluid (5), and that this fluid normally flows along the perivascular spaces either into the subarachnoid space or possibly even into the central canal and is ultimately absorbed at the arachnoid villi with the rest of the CSF (6). We feel that increased resistance to flow at the foramen magnum by either Chiari I malformations or other causes prevents this extracellular fluid from exiting, and it accumulates in the spinal cord. This explanation appears more reasonable to us than the hypothesis that cerebrospinal fluid is somehow forced from the subarachnoid space through channels of very high resistance into the spinal cord and accumulates there.

William C. Olivero, M.D.
Associate Professor of
Neurological Surgery and Pediatrics
University of Illinois
College of Medicine at Peoria

References

1. Fischbein NJ, Dillon WP, Cobbs C, Weinstein PR. **The "presyrinx" state: a reversible myelopathic condition that may precede syringomyelia.** *AJNR Am J Neuroradiol* 1999;20:7–20

2. Cahan LD, Bentson JR. **Considerations in the diagnosis and treatment of syringomyelia and the Chiari malformation.** *J Neurosurg* 1982;57:24–31
3. Stoodley MA, Brown, SA, Brown, CJ, Jones NR. **Arterial pulsation-dependent perivascular cerebrospinal fluid flow into the central canal in the sheep spinal cord.** *J Neurosurg* 1997;86:686–693
4. Milhorat TH, Nakamura S, Heger IM, Nobanegani F, Murray S. **Ultrastructural evidence of sink function of central canal of spinal cord as demonstration by clearance of horseradish peroxidase.** *Procedure, Electronic Microscope Society, America* 1992;50: 700–701
5. Sato O, Asai T, Amano Y, Hara M, Tsugane R, Yagi M. **Extraventricular origin of the cerebrospinal fluid: formation rate quantitatively measured in the spinal subarachnoid space of dogs.** *J Neurosurg* 1972;36:267–282
6. Olivero WC, Dinh Z. **Chiari I malformation with traumatic syringomyelia and spontaneous resolution: case report and literature review.** *Neurosurgery* 1992;30:758–760

Reply

We appreciate Dr. Olivero's letter and his interest in our article. In their paper (1), Drs. Olivero and Dinh describe a patient with post-traumatic acquired Chiari I malformation and syringomyelia that spontaneously resolved without intervention. They review multiple theories of the pathogenesis of syringomyelia and hypothesize that head trauma in this 28-year-old woman resulted in elevated intracranial pressure and secondary tonsillar herniation that then led to obstruction of CSF flow at the level of the foramen magnum. They hypothesize that this resulted in elevated pressure in the spinal subarachnoid space, preventing egress of fluid normally produced in the spinal cord and resulting in syrinx formation. When the elevated intracranial pressure resolved, normal CSF flow across the foramen magnum was reestablished, and the syrinx resolved.

We appreciate Dr. Olivero's emphasizing that the source of CSF within hydromyelic cavities associated with the Chiari I malformation is controversial and by no means firmly established. He reviews the evidence that, in the context of a Chiari I malformation and accentuation of systolic pressure waves within the spinal subarachnoid space, CSF may not be driven into the spinal cord parenchyma along the perivascular spaces. Rather, the CSF produced within the cord substance may be prevented from exiting because of elevated pressure in the spinal subarachnoid space. We failed to mention this theory in our report (2). Nonetheless, if true, this theory regarding the directionality of CSF flow is not inconsistent with our hypothesis that a presyrinx state may depend upon the patency of the central canal and, more importantly, is associated with CSF obstruction which, if relieved, may reverse the condition.

Nancy J. Fischbein, M.D.
William P. Dillon, M.D.
Department of Radiology
University of California, San Francisco
San Francisco, CA

References

1. Olivero WC, Dinh DH. **Chiari I malformation with traumatic syringomyelia and spontaneous resolution: case report and literature review.** *Neurosurgery* 1992;30:758–760
2. Fischbein NJ, Dillon WP, Cobbs C, Weinstein PR. **The "presyrinx" state: a reversible myelopathic condition that may precede syringomyelia.** *AJNR Am J Neuroradiol* 1999;20:7–20

Erratum:

The correct references for the letter to the editor—Hawley RJ, Payne JM, Giannola LS. **The Use of Hyperventilation in Contrast-Enhanced MR Imaging of Brain Tumors.** *AJNR Am J Neuroradiol* 1999;20:1184–1185—are:

1. Pronin IN, Holodny AI, Kornienkio VA, Petraikin AV, Golovanov AV, Lee HJ. **The use of hyperventilation in contrast-enhanced MR of brain tumors.** *AJNR Am J Neuroradiol* 1997;18:1705–1708
2. Ross RT. **Transient tumor attacks.** *Arch Neurol*1983;40:633–636
3. UK TIA Study Group. **Intracranial tumors that mimic transient cerebral ischemia: lessons from a large multicentre trial.** *J Neurol Neurosurg Psychiatry*1993;56:563–566
4. Cameron EW. **Transient ischemic attacks due to meningioma: report of 4 cases.** *Clin Radio*1994;49:416–418.
5. Schregel W, Geissler C, Winking M, Schaefermeyer H, Cunitz G. **Transcranial Doppler monitoring during induction of anesthesia: effects of propofol, thiopental, and hyperventilation in patients with large malignant brain tumors.** *J Neurosurg*1993;5: 86–93
6. Pronin IN, Kornienko VN, Petriaikin AV, Golanov AV. **The use of hyperventilation for improving the visualization of glial tumors of the brain in magnetic resonance tomography using the contrast substance Gd-DTPA.** *Zh Vopr Neirokhir Im N Burdenko*1995;3:10–13