Focal Headache During Balloon Inflation in the Internal Carotid and Middle Cerebral Arteries

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Although a number of reports are available on the occurrence of headache in patients with ischemic cerebrovascular disease, most studies have recorded the frequency but not the specific sites of the pain. We report 18 patients who underwent balloon inflation in the distal internal carotid artery and middle cerebral artery stem during embolization therapy for intracerebral arteriovenous malformations. Eleven patients had reproducible patterns of headache during balloon inflation. Inflation in the proximal middle cerebral artery stem produced pain primarily in the ipsilateral temple, that in the middle of the middle cerebral artery stem produced pain referred primarily retro-orbitally, and inflation in the distal middle cerebral artery stem produced pain referred primarily to the forehead. Experimental studies have demonstrated similar patterns of referred pain. The fact that these areas of referred pain are so reproducible is of potentially great clinical importance in the approach to management of patients with cerebrovascular disease. (Stroke 1990;21:555-559)

Although a number of reports are available on the occurrence of headache in patients with ischemic cerebrovascular disease, most studies have recorded the frequency but not the specific sites of the pain. Some authors have noted that carotid-distribution headaches tend to be located anteriorly and that vertebrobasilar-associated headaches tend to be located posteriorly. The fact that in most studies of cerebrovascular disease only a minority of patients are reported to develop headache has lead some clinicians to dismiss its importance. Unfortunately, from the reports of headache in patients with cerebrovascular disease it has not been possible to determine precisely the site, and frequently the type, of arterial disease. In part, this has resulted from an inability to define precisely the location of the arterial injury. In addition, it is frequently difficult to obtain an accurate history from patients who are acutely ill and/or neurologically impaired. Thus, the potential utility of localized head pain associated with focal arterial disease may have been overlooked. Our experience with balloon inflation within the intracranial arteries during therapeutic embolization of intracerebral arteriovenous malformations (AVMs) demonstrates that there are specific sites of referred pain from distension of these vessels. Knowledge of these specific patterns may prove to be useful clinically.

Subjects and Methods

Between July 1983 and June 1985, as part of the clinical management of AVMs at the New York Neurological Institute, 82 patients underwent therapeutic embolization of AVMs with Silastic pellets.

In 18 patients a flow-directed balloon catheter was used to temporarily occlude the distal internal carotid artery (ICA) and middle cerebral artery (MCA) stem. Either normal vessels (to prevent their possible embolization during the procedure) or one of the arteries supplying the AVM was occluded, thus allowing selective embolization of other arteries supplying the AVM.

As part of a protocol for the embolization of AVMs, a neurologist performed a complete history and neurologic examination of each patient during the 24 hours before the procedure and after each Silastic pellet embolization. In addition, the neurologist performed repeated brief neurologic evaluations every 3–5 minutes during balloon inflation.

The study patients comprised nine women and nine men, aged 19–55 years. All were in good general
health and had either no or only a minimal neurologic deficit. Patients were typically premedicated with droperidol, fentanyl, and diazepam. A catheter was introduced through the femoral artery under local anesthesia, usually bupivacaine. An initial angiogram was performed to identify the arterial supply to the AVM. A dual-lumen, flow-directed balloon-tipped catheter was then introduced and positioned under magnification fluoroscopy. There were no balloon occlusions in the middle to proximal ICA. During the procedure the balloon was frequently deflated and reinflated, either to allow perfusion of the normal distal arterial bed or to reposi- tion the balloon. This allowed observations on the effect of repeated inflation in the same location, as well as in different locations. The balloon was inflated manually until there was angiographic evidence of occlusion of the vessel. There were no precise criteria for volume or pressure in the balloon. The inflations lasted for variable times, typically at least 5 minutes.

All patients were alert and fully cooperative at the time of balloon inflation. Before inflation the patients had no complaint of headache. The first patient who underwent this protocol complained spontaneously of focal headache at the time of balloon inflation. Following this observation, all patients were asked at the time of balloon inflation if they had any discomfort or pain. If the response was “yes,” they were asked to describe the intensity, site, and size of the discomfort; to confirm the description the examiner would map out the area of pain on the patient’s head with his index finger. If the response was “no,” the patients were asked specifically if they had any head pain, but no location was suggested by the question. If they volunteered the presence of pain or discomfort, the patients were asked to describe its size and site. The severity of discomfort was graded as + if the patient was aware of the discomfort but did not seem disturbed by its presence, as ++ if the patient complained of moderate discomfort, and as +++ if the patient volunteered the presence of pain or when agitation indicated obvious discomfort. No patient developed a neurologic deficit that interfered with the assessment of the pain.

For descriptive purposes the MCA stem is divided into the proximal, middle, and distal thirds.

**Results**

During manipulation, the balloon catheter was often briefly inflated in the distal ICA, and a number of patients complained of discomfort in an area adjacent to the ipsilateral lateral canthus. None complained of pain above the eye or on the cheek. However, as the balloon inflation at this site was usually very brief, more detailed data could not be collected.

Eleven patients developed focal headache upon balloon inflation; the pain resolved with balloon deflation and recurred upon reinflation. In the remaining seven patients, specific questioning failed to elicit any complaint of head discomfort. In the 11 patients experiencing pain, the precise patterns of pain were reproducible upon reinflation during one session; in two of the three patients who underwent repeat embolization, the patterns of pain were also reproducible (Table 1). The headache pattern was consistent not only within patients but also between patients. With balloon inflation in the proximal third of the MCA stem, pain appeared in a 3–5-cm diameter area adjacent to the ipsilateral lateral canthus and slightly above the zygomatic arch (Figure 1). With balloon inflation in the distal third of the MCA stem, pain developed in a 2–3-cm diameter area above the ipsilateral eye (Figure 1). Retro-orbital pain frequently accompanied inflation in the proximal or distal MCA stem but was generally much less intense than the discomfort in the temple or forehead. Inflation in the middle third of the MCA stem produced retro-orbital pain almost exclusively, with only minimal pain in the temple or forehead.

Six of nine patients undergoing left-sided balloon inflation and five of nine patients undergoing right-sided balloon inflation developed focal head discomfort.

**Illustrative Case**

A 28-year-old right-handed man (patient 5) with a left parieto-occipital AVM supplied by branches of the anterior, middle, and posterior cerebral arteries underwent therapeutic embolization. When the balloon was inflated in the distal third of the left MCA stem, he promptly complained of pain in an area approximately 3 cm in diameter above his left eye and had a less severe complaint of retro-orbital discomfort. When the balloon was deflated the pain resolved completely almost immediately. The balloon was then pulled into the proximal third of the MCA stem, where inflation resulted in pain in the left temple, approximately 2 cm behind the lateral canthus. When the balloon was deflated, there was complete relief from pain in < 1 minute. The balloon was repositioned slightly more distally in the MCA stem (in the middle third), where inflation produced pain behind the left eye and adjacent to and slightly above the lateral canthus. At this point the balloon was deflated, with prompt relief from pain, and then reinflated with prompt recurrence of the pain in the left temple as before, with perhaps more retro-orbital complaint.

Patient 5 had seven balloon inflations during this embolization, and focal headache occurred with each inflation. The embolization procedure was repeated 2 months later. On balloon inflation in the proximal MCA, he again had headache in the temple, as on the previous occasion. There was no focal tenderness over the site of head pain either during or after balloon inflation.

**Discussion**

Our study documents reproducible patterns of referred pain during balloon occlusion of the distal
### TABLE 1. Sites of Balloon Inflation and Severity of Referred Pain in 18 Patients Undergoing Balloon Occlusion During Therapeutic Embolization of Arteriovenous Malformations

<table>
<thead>
<tr>
<th>Pt</th>
<th>Site of balloon inflation</th>
<th>Temple</th>
<th>Retro-orbital</th>
<th>Forehead</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>R proximal MCA</td>
<td>+++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>R distal ICA</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>R distal MCA</td>
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<td>0</td>
<td>0</td>
</tr>
<tr>
<td>4</td>
<td>L proximal MCA</td>
<td>+++</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>L distal MCA (x4)</td>
<td>0</td>
<td>+</td>
<td>++</td>
</tr>
<tr>
<td></td>
<td>L proximal MCA (x3)</td>
<td>+</td>
<td>+</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>L middle MCA (x2)</td>
<td>0</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
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<td>L proximal MCA</td>
<td>+++</td>
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<td>0</td>
</tr>
<tr>
<td>6</td>
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<td>++</td>
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<tr>
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<td>0</td>
</tr>
<tr>
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<td>R proximal MCA</td>
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<td>0</td>
</tr>
<tr>
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<td>0</td>
<td>+++</td>
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<td>0</td>
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<tr>
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</tr>
<tr>
<td>13</td>
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<td>+++</td>
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<td>+</td>
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<td>0</td>
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</tr>
<tr>
<td>18</td>
<td>L proximal MCA</td>
<td>+</td>
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<td>0</td>
</tr>
</tbody>
</table>

Pt, patient; R, right; L, left; MCA, middle cerebral artery; ICA, internal carotid artery; 0, no admission of discomfort on questioning; +, patient aware of discomfort but not disturbed by its presence; ++, patient complained of moderate discomfort; ++++, patient volunteered presence of pain or agitation indicated obvious discomfort. A indicates second procedure.

ICA and the MCA stem. Pain from the distal ICA and proximal MCA was referred ipsilaterally to an area adjacent to the lateral canthus, pain from the middle MCA was referred primarily retro-orbitally, and pain from the distal MCA stem was referred to an area above the eye. Although there is some overlap in these patterns, they are distinct and reproducible.

The lack of pain in certain patients with balloon inflation may be due to the lack of a standardized way to inflate the balloon to a specific pressure or size; the inflations may not have produced identical amounts of stretch of the vessel, thereby failing to stimulate the pain receptors in some cases. Also, some of the arteries that supply an AVM may be dysplastic, and it is possible that they lack normal innervation.

Focal referred pain has been demonstrated in studies employing stimulation of the distal ICA and MCA stem. In four of five patients, Northfield noted that pressure or displacement of the ICA adjacent to the anterior clinoid process resulted in pain behind the ipsilateral eye or in the ipsilateral temple. Fay noted that "electric stimulations, clamping, or traction of the large arterial branches produced ipsilateral pain referred deep in the eye, vaguely distributed to the temples, as is the case with the middle meningeal and middle cerebral arteries." Penfield noted in two patients that stimulation of the MCA referred pain to the ipsilateral temporal region in one and to the forehead above the eye in the other. The occurrence and patterns of referred pain were demonstrated by Ray and Wolff with repeated...
farad stimulations of the MCA in awake patients. Stimulation of the distal ICA and proximal MCA in three patients produced pain in the sites that we observed with balloon inflation in the proximal MCA (Figure 1).

Headache has been noted in from 9% to >30% of patients with ischemic stroke. Although some authors have felt its presence to be of little significance, others have commented on the potential utility of headache in localizing the site of vascular disease. Fisher described pain associated with embolism and thrombosis of the MCA. He noted the development of pain in the forehead above the eye with MCA thrombosis and in the frontotemporal region near the lateral canthus with MCA embolism. The location of the latter is similar to that seen in our patients with balloon inflation in the proximal MCA (a likely site of lodgement of embolic particles). Auerbach et al also reported a patient who suffered an embolus to the left MCA stem that produced pain in the “left temple.” The locations of the headache produced by balloon inflation in the MCA are also similar to those reported in patients with dissection of the ICA. No patient had pain in the neck or cheek as a result of balloon inflation in the MCA, so presumably such pain is solely ICA-radiated pain. In support of this, Fay noted that faradic stimulation of the carotid bifurcation results in pain referred to the teeth, gums, scalp, cheek, jaw, and the face about the orbit. Although headache is frequently reported by patients with intracranial arterial dissection, most of the descriptions of this subject simply note the presence of headache, described variously as diffuse or ipsilateral, but rarely adding any details on the location of the pain.

The location of referred pain in cases of stroke in which the site of the vascular lesion has been determined is similar to that seen in our study and in previous experimental stimulation studies. Stretching of the artery by intraluminal inflation of a balloon, as well as by pressure or faradic stimulation applied to the external aspects of the MCA stem, all result in similar, reproducible patterns of referred pain. Thus, both clinical and experimental observations suggest that focal head pain in patients with acute stroke may be the result of local arterial disease. However, there are other potential mechanisms that may result in headache at the time of stroke. As originally suggested by Willis, it is possible that dilation of collateral channels causes headache. In addition, the stress of the event could trigger unrelated vascular headache. Further, pain referred to these sites is not entirely specific for the distal ICA or the proximal MCA. Stimulation of the superficial temporal and middle meningeal arteries, as well as of the large intracranial venous channels, may also result in similar patterns of referred pain. However, well-localized headache in the areas we describe, particularly in the clinical setting of acute stroke, should alert the clinician to the possibility of arterial disease in specific locations.

Both myelinated and unmyelinated nerves, generally of unspecified origin, follow the intracranial arteries, but the precise nerves involved in the mediation of the perceived pain from the distal ICA and proximal MCA remain poorly defined. Older studies have shown that the ophthalmic division of the trigeminal nerve contributes many fibers to this area and to other structures above the tentorium. Moskowitz as well as Yamamoto et al have recently confirmed this observation histochromically. Mayberg et al have demonstrated the presence of fibers from both superior cervical ganglia running to this area. Fay has demonstrated that despite sectioning of the trigeminal nerve, patients still felt pain upon stimulation of the large intracerebral vessels. In fact, even after sectioning the trigeminal and glossopharyngeal nerves and upper cervical nerve roots, he frequently was able to produce a “dull throbbing pain referred to the field of the large vascular branches, and tenderness as well as pain on direct stimulation could be obtained in the facial, temporal, occipital, and carotid arteries.” He felt that the vagus had a major role in the perception of painful stimuli applied to the intracranial arteries but cautioned that the only patients in whom sectioning of the tenth cranial nerve had been successful in the relief of pain had other procedures performed, typically sectioning of the fifth, ninth, eleventh, twelfth, and cervical sympathetic chain or ganglia. Despite Fay's observations, the consensus of previous studies is that section of the trigeminal nerve above the level of the tentorium results in pain referred to the distribution of the ophthalmic portion of the trigeminal nerve, primarily those areas we describe for balloon inflation.

Our observations of the sites of pain that result from balloons' stretching of the intracranial distal ICA and proximal MCA complement the experimental observations of Ray and Wolff, as well as the clinical observations of Fisher, demonstrating a consistent pattern of referred pain in response to faradic stimulation, stretching, or dissection for these intracranial vessels. Although the precise pathways of the referred pain are as yet not fully known, the fact that these areas of referred pain are so reproducible is of potentially great clinical importance in the approach to management of patients with cerebrovascular disease.

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References


**KEY WORDS** • arterial venous malformations • embolizations, therapeutic • headache
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