Distribution of Intraarterially Administered Papaverine in Endovascular Treatment of Delayed Cerebral Vasospasm

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Abstract

The distribution of selectively administered papaverine was determined in nine patients with delayed cerebral vasospasm in the territories of the anterior (ACA) and/or middle cerebral arteries (MCA) secondary to aneurysmal subarachnoid hemorrhage by simultaneous infusion with technetium-99m-hexamethylpropyleneamine oxime ($^{99m}$Tc-HMPAO). Four of the nine patients had a ruptured anterior communicating artery aneurysm, four had an internal carotid artery aneurysm, and the remaining one had a MCA aneurysm. Trapping of anterior communicating artery was carried out in one case and clipping of aneurysms in other eight cases. Neurological deterioration with hemiparesis, paraparesis, and/or somnolence appeared between postsurgical Days 8 and 13 due to delayed cerebral vasospasm in all patients. Intraarterial infusion of 40 mg of papaverine containing 37 MBq of $^{99m}$Tc-HMPAO was performed from the C$_1$ segment in seven of the nine patients and from the C$_2$ segment in the other two patients. $^{99m}$Tc-HMPAO was distributed in the territories of the ACA and MCA in the two patients who were treated with intraarterial infusion of papaverine from the C$_2$ segment, but was distributed only to the territory of the ACA in four patients who were treated with intraarterial infusion of papaverine from the C$_1$ segment at 1 ml/min. In contrast, $^{99m}$Tc-HMPAO was distributed in the territories of the ACA and MCA in the three patients who were treated with papaverine from the C$_1$ segment at 2 ml/min, although most $^{99m}$Tc-HMPAO was distributed in the territory of the ACA. Vasospasm of the ACA can be treated by intraarterial infusion of papaverine from the C$_1$ segment at 1 ml/min when selective catheterization to the ACA is difficult to perform.

Key words: subarachnoid hemorrhage, delayed cerebral vasospasm, anterior cerebral artery, papaverine, $^{99m}$Tc-HMPAO, single photon emission computed tomography

Introduction

The treatment of cerebral vasospasm has improved greatly with the use of percutaneous transluminal angioplasty (PTA) and/or superselective infusion of papaverine.$^5,8,1°,12,17$ The first treatment of cerebral vasospasm after subarachnoid hemorrhage (SAH) with transluminal balloon angioplasty was reported in 1984.$^{17}$ Subsequent applications of this technique have generally achieved favorable results in patients with symptomatic vasospasm refractory to conventional therapy.$^3,19$ However, the balloon catheter currently available for PTA is limited for reaching distally affected vessels or vessels with short and sharply angled origins, such as the distal segments of the middle cerebral artery (MCA) and the A$_1$ segment of the anterior cerebral artery (ACA). Moreover, superselective catheterization of the ACA using the Tracker-18 or Tracker-10 is sometimes difficult to perform.$^8$

Papaverine is only effective on vessels when delivered by blood flow. Papaverine will be relatively ineffective, for example, in cases of ACA spasm with a normal MCA, since the blood flow preferentially delivers papaverine infused into the internal carotid artery (ICA) to the MCA. However, this problem can be circumvented by transiently occluding the MCA with a balloon and infusing papaverine in the more proximal portion of the ICA.$^{30}$ Superselective catheterization of the A$_1$ segment just proximal to a spastic portion of A$_1$, followed by selective infusion of papaverine, achieved dilation of the A$_1$ segment.$^6$ Superselective catheterization of the M$_1$ segment of the MCA is thought to be relatively easy, whereas catheterization of the A$_1$ segment is sometimes difficult and the guidewire may
injure the arterial wall or perforate the spastic vessel.

This study determined the distribution of papaverine infused from the C1 segment of the ICA simultaneously with technetium-99m-hexamethylpropyleneamine oxime (99mTc-HMPAO) as a tracer.

**Clinical Materials and Methods**

I. Clinical materials

This study included nine patients with vasospasm of the ICA or MCA treated between August 1993 and January 1995. Four patients had a ruptured anterior communicating artery (AcomA) aneurysm, four had a ruptured ICA-posterior communicating artery aneurysm, and the other had a ruptured MCA aneurysm. Seven patients were classified in clinical grade II, and two in clinical grade III.

Computed tomography (CT) performed on admission in all patients demonstrated group 3 SAW.

Surgery was performed on the day of admission following completion of diagnostic angiography. Trapping of the AcomA aneurysm was performed in Case 2, and aneurysmal neck clipping in the other eight cases. All patients had clear consciousness and no neurological deficit the day after the operation. Delayed ischemic neurological deficits appeared in all patients (Table 1).

II. Treatment and distribution study

A Tracker-10 microcatheter and guidewire system (Target Therapeutics, Inc., Fremont, Cal., U.S.A.) was advanced coaxially to the C1 or C4 segment through a 5 French angiographic catheter. In seven (Cases 1–7) of the nine patients, 99mTc-HMPAO and papaverine diluted with normal saline was infused through the microcatheter located in the C1 segment. In the remaining two patients (Cases 8 and 9), papaverine was infused through the microcatheter in the C4 segment (Table 1).

Selective infusion was performed using a transfemoral approach under local anesthesia with full heparinization. The patient's neurological condition was carefully monitored throughout the procedure. After completion of papaverine infusion, selective angiograms of the ICA were obtained through the guiding catheter. A 99mTc-HMPAO single photon emission computed tomography (SPECT) study was performed after the angiography. The distribution of 99mTc-HMPAO was determined and compared with perfusion maps obtained before (Cases 2, 5, 6, and 7) or just after (Cases 1, 3, 4, 8, and 9) the appearance of symptomatic vasospasm.

III. Control studies

The control group consisted of four patients in whom cerebral angiography showed neither stenosis nor occlusion in the territories of the ACAs or MCAs. The four patients underwent subtotal resection of glioblastoma multiforme followed by conventional radiation therapy, and were treated with 1-[4-aminoethylpyridine-5-yl]-methyl-3-[2-chloroethyl]-3-nitrosourea hydrochloride (ACNU) infused from the C1 segment between July 1993 and January 1994. A total of 37 MBq of 99mTc-HMPAO and 50 mg of ACNU were mixed and diluted with 20 ml of normal saline and infused at 1 ml/min through a Tracker-10 located in the C1 segment (Table 2).

**Results**

I. Angiographic and clinical results

The spastic ACAs became dilated just after termination of infusion of papaverine in Cases 2–4 and all three manifested improvement of level of paraparesis and/or consciousness. The spastic ACA and MCA became dilated just after termination of infusion of papaverine in Cases 8 and 9, and both manifested improvement of hemiparesis and/or level of consciousness. Vasospasm in the territory of the right MCA showed no improvement after completion of infusion of papaverine from the right C1 segment in Case 1, but improved just after completion of infusion of papaverine from the right M1 segment. Vasospasm in the territory of the MCA showed slight improvement after completion of infusion of papaverine from the C1 segment in Cases 5–7. Eight patients were discharged without neurological deficit (Glasgow Outcome Scale) 34 to 42 days after the onset of SAH. The other patient (Case 5) was discharged with right hemiparesis 55 days after the onset of SAH (Table 1).

II. Distribution of 99mTc-HMPAO

99mTc-HMPAO was homogeneously distributed to the territories of the ACA and MCA in Cases 8 and 9. 99mTc-HMPAO was confined to the territory of the left ACA in Case 2, and to the territories of the bilateral ACAs in Cases 3 and 4. 99mTc-HMPAO was also confined to the territory of the right ACA in Case 1. The territory of the MCA was not perfused with 99mTc-HMPAO in the four patients who received infusion of papaverine at 1 ml/min in the C1 segment (Cases 1–4). 99mTc-HMPAO was distributed to the territories of the ACA and MCA in Cases 5–7. However, most of the 99mTc-HMPAO was distributed to the territory of the ACA (Table 1).

99mTc-HMPAO was confined to the territory of the ACA in the four control patients (Table 2).
Table 1 Summary of nine patients with delayed cerebral vasospasm treated by intraarterial infusion of papaverine

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/ Sex</th>
<th>Location of aneurysm</th>
<th>Clinical grade*</th>
<th>SAH group**</th>
<th>Operation (Day)</th>
<th>Neurological deficits (Day)</th>
<th>Spastic vessels (parent ICA)</th>
<th>Doses of papaverine (Infusion site, infusion rate)</th>
<th>Immediate clinical results</th>
<th>Distribution of ⁹⁹ᵐTc-HMPAO</th>
<th>Outcome***</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>63/F</td>
<td>lt ICA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>lt hemiparesis (10)</td>
<td>rt ACA (rt)</td>
<td>40 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>rt ACA</td>
<td>good recovery (6)</td>
</tr>
<tr>
<td>2</td>
<td>48/M</td>
<td>AcomA</td>
<td>II</td>
<td>3</td>
<td>trapping (0)</td>
<td>paraparesis, somnolence (13)</td>
<td>lt ACA (rt)</td>
<td>40 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>lt ACA</td>
<td>good recovery (5)</td>
</tr>
<tr>
<td>3</td>
<td>54/F</td>
<td>AcomA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>paraparesis, somnolence (8)</td>
<td>bil ACAs (lt)</td>
<td>40 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>bil ACAs</td>
<td>good recovery (6)</td>
</tr>
<tr>
<td>4</td>
<td>67/F</td>
<td>AcomA</td>
<td>III</td>
<td>3</td>
<td>clipping (0)</td>
<td>paraparesis, somnolence (10)</td>
<td>bil ACAs (rt)</td>
<td>40 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>bil ACAs</td>
<td>good recovery (6)</td>
</tr>
<tr>
<td>5</td>
<td>62/F</td>
<td>lt ICA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>rt hemiparesis, somnolence (8)</td>
<td>lt MCA (lt)</td>
<td>40 mg/40 ml saline (rt C₁, 60 ml/hr)</td>
<td>unchanged</td>
<td>lt ACA (lt MCA)</td>
<td>severe disability (8)</td>
</tr>
<tr>
<td>6</td>
<td>55/M</td>
<td>AcomA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>rt hemiparesis, somnolence (9)</td>
<td>lt ACA (lt)</td>
<td>40 mg/40 ml saline (rt C₁, 120 ml/hr)</td>
<td>unchanged</td>
<td>lt ACA (lt MCA)</td>
<td>good recovery (6)</td>
</tr>
<tr>
<td>7</td>
<td>37/M</td>
<td>lt ICA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>rt hemiparesis (10)</td>
<td>lt MCA (lt)</td>
<td>40 mg/40 ml saline (rt C₁, 120 ml/hr)</td>
<td>improved</td>
<td>lt ACA (lt MCA)</td>
<td>good recovery (5)</td>
</tr>
<tr>
<td>8</td>
<td>55/F</td>
<td>rt MCA</td>
<td>II</td>
<td>3</td>
<td>clipping (0)</td>
<td>lt hemiparesis (10)</td>
<td>rt ACA (rt)</td>
<td>80 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>rt ACA</td>
<td>good recovery (5)</td>
</tr>
<tr>
<td>9</td>
<td>61/M</td>
<td>rt ICA</td>
<td>III</td>
<td>3</td>
<td>clipping (0)</td>
<td>lt hemiparesis, somnolence (6)</td>
<td>rt MCA (rt)</td>
<td>80 mg/20 ml saline (rt C₁, 60 ml/hr)</td>
<td>improved</td>
<td>rt MCA</td>
<td>good recovery (6)</td>
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</table>

*Hunt and Kosnik classification. **Subarachnoid hemorrhage (SAH) grouping according to Fisher et al. ***Glasgow Outcome Scale score. Papaverine was infused with technetium-⁹⁹ᵐ-hexamethylpropyleneamine oxime (⁹⁹ᵐTc-HMPAO). Most infused ⁹⁹ᵐTc-HMPAO was distributed to the territory of the anterior cerebral artery (ACA) in Cases 5–7. AcomA: anterior communicating artery, ICA: internal carotid artery, MCA: middle cerebral artery.
Table 2  Summary of four control patients with glioblastoma multiforme treated by intraarterial infusion of ACNU

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age/ Sex</th>
<th>Location of tumor</th>
<th>Histology</th>
<th>Operation</th>
<th>Irradiation (Gy)</th>
<th>Doses of ACNU (Infusion site)</th>
<th>Distribution of 99mTc-HMPAO</th>
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</thead>
<tbody>
<tr>
<td>10</td>
<td>68/F</td>
<td>rt frontal</td>
<td>glioblastoma</td>
<td>subtotal</td>
<td>50</td>
<td>50 mg (rt C1)§</td>
<td>rt ACA</td>
</tr>
<tr>
<td>11</td>
<td>48/F</td>
<td>rt fronto-parietal</td>
<td>glioblastoma</td>
<td>subtotal</td>
<td>60</td>
<td>50 mg (rt C1)§</td>
<td>rt ACA</td>
</tr>
<tr>
<td>12</td>
<td>79/F</td>
<td>lt frontal</td>
<td>glioblastoma</td>
<td>subtotal</td>
<td>50</td>
<td>50 mg (lt C1)§</td>
<td>lt ACA</td>
</tr>
<tr>
<td>13</td>
<td>38/M</td>
<td>rt frontal</td>
<td>glioblastoma</td>
<td>subtotal</td>
<td>60</td>
<td>50 mg (rt C1)§</td>
<td>rt ACA</td>
</tr>
</tbody>
</table>

§1-(4-aminomethylpyridine-5-yl)-methyl-3-(2-chloroethyl)-3-nitrosourea hydrochloride (ACNU) was infused with technetium-99m-hexamethyl-propyleneamine oxime (99mTc-HMPAO). ACA: anterior cerebral artery.

Fig. 1  Case 2.  Left internal carotid angiograms (anteroposterior views), before operation demonstrating an anterior communicating artery aneurysm (left), on Day 13 demonstrating severe and diffuse vasospasm in the territory of the left anterior cerebral artery (ACA) (center), and just after intraarterial infusion of papaverine demonstrating dilation of the left ACA (right).

Illustrative Cases

Case 2: A 48-year-old male was admitted with SAH due to rupture of an AcomA aneurysm (Fig. 1 left). He was grade II clinically with group 3 CT findings. Trapping of the aneurysm was performed on the day of admission (Day 0). He had clear consciousness without neurological deficit the day after operation. 99mTc-HMPAO SPECT was performed on Day 10 when he became slightly somnolent, showing an inhomogeneous region of low perfusion bilaterally (Fig. 2 upper row). On Day 13, he became paraparetic and somnolent. Cerebral angiography disclosed severe vasospasm in the bilateral ACAs and development of collaterals bilaterally via the posterior pericallosal arteries, and from the MCAs (Fig. 1 center). A Tracker-10 was introduced into the C1 segment just distal to the origin of the anterior choroidal artery of the right ICA through a 5 French angiographic catheter, and 40 mg of papaverine was infused, resulting in dilation of the right spastic ACA. The Tracker-10 was then introduced into the C1 segment of the left ICA (Fig. 3), and 40 mg of papaverine and 37 MBq of 99mTc-HMPAO were in-
fused at 1 ml/min, resulting in dilation of the spastic left ACA (Fig. 1 right). Just after termination of interventional treatment, $^{99m}$Tc-HMPAO SPECT was performed to obtain a $^{99m}$Tc-HMPAO distribution map. $^{99m}$Tc-HMPAO was present only in the territory of the left ACA (Fig. 2 lower row). His neurological deficit completely disappeared on Day 14, and he was discharged on Day 34 without neurological deficit.

**Fig. 2** Case 2. Technetium-99m-hexamethyl-propyleneamine oxime ($^{99m}$Tc-HMPAO) single photon emission computed tomography scans, axial (left column), coronal (center column), and sagittal (right column) planes, before endovascular treatment of delayed vasospasm showing inhomogeneously low perfusion in both cerebral hemispheres (upper row), and after intraarterial infusion of 40 mg of papaverine with $^{99m}$Tc-HMPAO into the left C1 segment showing $^{99m}$Tc-HMPAO distribution restricted to the territory of the left anterior cerebral artery (lower row).

**Fig. 3** Case 2. Angiogram (anteroposterior view) showing the anterior and middle cerebral arteries visualized after bolus injection of contrast medium through the Tracker-10 selectively introduced into the left C1 segment just distal to the anterior choroidal artery. The arrow indicates the tip of the Tracker-10.
Case 9: A 61-year-old male was admitted with SAH due to rupture of a right ICA-posterior communicating artery aneurysm. He was grade III clinically and had group 3 CT findings. Neck clipping of the aneurysm was performed on the day of admission, and his consciousness was clear without neurological deficits the day after operation.

Left hemiparesis and somnolence developed on Day 8. $^{99m}$Tc-HMPAO SPECT performed on the same day demonstrated bilateral inhomogeneous regions of low perfusion in both cerebral hemispheres (upper row), and after intraarterial infusion of 80 mg of papaverine with $^{99m}$Tc-HMPAO from the right C4 segment showing $^{99m}$Tc-HMPAO in the territories of the right anterior and middle cerebral arteries (lower row).

$$\text{Fig. 4 Case 9. Technetium-99m-hexamethylpropyleneamine oxime (}^{99m}\text{Tc-HMPAO) single photon emission computed tomography scans, axial (left column) and coronal (right column) planes, before endovascular treatment of delayed vasospasm disclosing inhomogeneously low perfusion in both cerebral hemispheres (upper row), and after intraarterial infusion of 80 mg of papaverine with }^{99m}\text{Tc-HMPAO from the right C}_4\text{ segment showing }^{99m}\text{Tc-HMPAO in the territories of the right anterior and middle cerebral arteries (lower row).}$$

99mTc-HMPAO SPECT was performed on Day 7 when he showed slight somnolence. Cerebral perfusion in the left cerebral hemisphere was slightly lower than that in the right hemisphere (Fig. 6 upper row). On Day 9, he showed right hemiparesis and somnolence. Cerebral angiography disclosed vasospasm in the left ACA and ipsilateral MCA. A Tracker-10 was introduced into the C1 segment just distal to the origin of the anterior choroidal artery of the left ICA through a 5 French angiographic catheter (Fig. 7), and 40 mg of papaverine and 37 MBq of $^{99m}$Tc-HMPAO were infused resulting in dilation of the spastic vessels. Just after termination of interventional treatment, $^{99m}$Tc-HMPAO SPECT was performed to obtain a distribution map. $^{99m}$Tc-HMPAO was present in the territories of the right ACA and MCA (Fig. 4 lower row). The left hemiparesis disappeared on Day 9 and his consciousness became clear on Day 10. He was discharged on Day 40 without neurological deficit.

Case 6: A 55-year-old male was admitted with SAH due to a ruptured AcomA aneurysm. He was grade II clinically and had group 3 CT findings. Neck clipping of the aneurysm was performed on the day of admission, and he manifested clear consciousness without neurological deficits the day after operation.

$^{99m}$Tc-HMPAO SPECT was performed on Day 7 when he showed slight somnolence. Cerebral perfusion in the left cerebral hemisphere was slightly lower than that in the right hemisphere (Fig. 6 upper row). On Day 9, he showed right hemiparesis and somnolence. Cerebral angiography disclosed vasospasm in the left ACA and ipsilateral MCA. A Tracker-10 was introduced into the C1 segment just distal to the origin of the anterior choroidal artery of the left ICA through a 5 French angiographic catheter (Fig. 7), and 40 mg of papaverine and 37 MBq of $^{99m}$Tc-HMPAO in 40 ml of normal saline were infused at 2 ml/min, resulting in partial dilation of the left ACA and MCA. Just after termination of interventional treatment, $^{99m}$Tc-HMPAO SPECT...
was performed to obtain a distribution map. $^{99m}$Tc-HMPAO was present in the territories of the left ACA and the ipsilateral MCA. However, most $^{99m}$Tc-HMPAO was distributed in the territory of the left ACA (Fig. 6 lower row). His neurological deficit completely disappeared on Day 14, and he was discharged on Day 40 without neurological deficit.

**Case 10:** A 66-year-old female had undergone subtotal removal of glioblastoma multiforme located in the right frontal lobe on May 18, 1993, followed by conventional radiation therapy with a total dose of 50 Gy. She had left hemiparesis (4/5) after the treatment. On July 17, 1993, a Tracker-10 was advanced to the right C1 segment. A total of 37 MBq of $^{99m}$Tc-HMPAO and 50 mg of ACNU in 20 ml of normal saline was infused at 1 ml/min through the Tracker-10. $^{99m}$Tc-HMPAO was present only in the territory of the right ACA (Fig. 8).

**Discussion**

A previous study observed intravascular streaming after intracarotid infusion of cisplatin, which was reduced with diastole-phased pulsatile infusion, suggesting that extensive and variable intravascular streaming occurs in infusions into the supraophthalmic segment of the ICA. The infusion rate was between 0.4 and 5.0 ml/min. The patients were divided into two groups: patients who received infusion of the anticancer drug from the infrarophthalmic segment of the ICA, and patients who received infusion from the supraophthalmic segment of the ICA. In the former group, a minimally heterogeneous distribution of the infusate was detected. In contrast, heterogeneous distribution of infusate was detected in the territories of the ACA and MCA in the latter group who received continuous infusion. The magnitude of streaming could be substantially reduced or eliminated with diastole-phased pulsatile infusion. However, patients with infusion from the supraophthalmic segment were not divided into subgroups of patients with infusion from the C1 segment, and patients with infusion from the site proximal to the C1 segment.

In our study, $^{99m}$Tc-HMPAO infused with papaverine into the C1 segment reached the territories of the ACA and MCA. In contrast, $^{99m}$Tc-HMPAO...
simultaneously infused with papaverine into the C1 segment at 1 ml/min reached only the territory of the spastic ACA, even when the MCA was of normal caliber on angiograms. $^{99m}$Tc-HMPAO was confined to the territory of the ACA even in the control patients who had neither stenosis nor occlusion in the territories of the ACA and the MCA. $^{99m}$Tc-HMPAO simultaneously infused with papaverine into the C1 segment at 2 ml/min reached the territories of both the ACA and MCA. However, most infused $^{99m}$Tc-HMPAO was distributed to the territory of the ACA.

Infusion rates of papaverine for the treatment of delayed vasospasm have ranged from 1.0 to 9.0 ml/min. Our findings indicate that cerebral vasospasm in the territory of the ACA can be treated selectively by continuous infusion of papaverine in saline into the C1 segment at 1 ml/min. The intracranial portion of the ICA runs dorso-laterally at the C1 segment, and the tip of the microcatheter delivered to the C1 segment was always attached to the medial inner lumen of the ICA. Therefore, if contrast medium or papaverine is injected as a bolus or at 2 ml/min, it is distributed to both the ACA and MCA. If papaverine is continuously infused at a low flow rate (1 ml/min), it runs along the medial inner lumen of the ICA (laminar flow effect) and enters only the ACA.

When treatment of vasospasm in the territory of the ACA is necessary and selective catheterization to the ACA is difficult to perform, infusion of papaverine at 1 ml/min in the C1 segment will deliver the agent selectively to the ACA.

**References**


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Commentary

It is easy to imagine that, within the intracranial portion of the internal carotid artery, the blood stream is largely divided into two stream lines, going to the anterior cerebral artery and to the middle cerebral artery. The authors of the present paper used this idea to selectively deliver papaverine to the anterior cerebral artery undergoing cerebral vasospasm. The drug intraarterially injected in this way was proved to reach only the territory of the anterior cerebral artery by SPECT during which 99mTc-HMPAO was simultaneously administered. Furthermore, the efficacy of this mode of treatment was supported by the immediate improvement of neurological deficits in some of the cases and the overall good outcome. The technique suggested in this paper certainly appears to be a useful method worth remembering.

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This is a very interesting article that helps considerably to clarify the question of what happens with intra-arterial injections, particularly when one of the runoff arteries is severely constricted by vasospasm. One would expect this to mean automatically that the infusion cannula must be advanced into the artery with the spastic segment, but the authors have demonstrated convincingly that this is not necessarily the case, and that the flow depends more on other factors.

These include particularly the location in the internal carotid artery from which an injection is made. As one would expect, if this is more proximal as with the C1 segment, mixing of the infusate so that it flows into both anterior (ACA) and middle cerebral arteries (MCA) occurs. If the catheter is advanced into the C2 segment distal to the anterior choroidal branch, where an infused solution goes to will depend on the rate of infusion-injection at 2 ml/min will again lead to distribution to both MCA and ACA, but with a slower infusion of 1 ml/min it will tend to pass selectively...
into the ACA, even when that vessel is the only one in spasm.

This has important implications in the management of vasospasm by arterial papaverine infusion. It appears that for predominantly MCA spasm the cannula may be best passed selectively into that artery. If the ACA is the main target, a distal intracarotid placement will suffice, without the need for balloon occlusion of the MCA to direct flow.

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The authors successfully demonstrated that slow injection (1 ml/min) from a microcatheter tip located at the C1 portion of the carotid artery results in predominant distribution in the ACA area and that vasodilating or anti-cancer drug can be delivered into the ACA area by these methods. They attributed this specific distribution pattern to several factors; the catheter tip locating medially in the C1 lumen and in the laminar flow to the ACA area, and slow injection at 1 ml/min rather than bolus injection.

While the results and conclusion seem reasonable, one basic question still arises. It is well known that velocity profile and secondary motions of the intravascular flow change greatly and momentarily depending on the geometrical difference of the arterial tree and pulsatility of the blood stream.1,2) Accordingly, greater variations of these factors in a more heterogeneous group of patients may make the location of the catheter tip or volume of the injection less meaningful.

This study would be more useful if the authors investigate details of the distribution pattern of injected materials in terms of the different location of catheter tip within the C1 or C2 segment of the ICA, a wider range of injection rate, and changing parameters of the systemic cardiovascular system in individual patients.

References


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