Artificial Slow Flow Carotid Angiography

Carotid Angiography with Compression Proximal to the Site of Injection*

HENK VERBIEST, M.D., AND THOMAS A. DOKKUM, M.D.

Neurosurgical Department, State University of Utrecht, Utrecht, The Netherlands

VARIOUS arterial compression tests have been introduced in cerebral angiography to produce alterations in blood flow. There is a limited number of papers on carotid angiography with compression or occlusion of the carotid artery proximal to the site of injection. The early cerebral angiographies reported in 1931 by Moniz were performed with injection of the common carotid and proximal occlusion of this vessel. Moniz noted that under these conditions contrast medium passed more readily through the external than through the internal carotid, which was why he also occluded the external carotid. He explained this finding by a greater resistance to the passing of the contrast medium in the internal carotid at the levels of the carotid foramen and the siphon. He also noted that in a number of cases the anterior cerebral artery and its ramifications were not filled, while the Sylvian group was well visualized. Three years later he reported that these inconveniences were avoided when injecting the nonoccluded common carotid artery. No further attention was given to carotid angiography under proximal occlusion since, at the time, all efforts were directed toward obtaining a perfect intracranial angiogram. Therefore, Engeset in 1944 rejected the proximal occlusion because it decreased the density of the angiographic picture. In later years, various authors described angiography with injection distal to the site of compression or ligation of the carotid artery in cases of intracranial aneurysms in order to check the effects of ligation on the aneurysm.

No attention has been given to using proximal compression in carotid angiography for other diagnostic purposes. Recently one of us (H. V.) found this method useful for demonstrating arteriovenous aneurysms supplied by the external carotid artery, and consequently we decided to examine its diagnostic value systematically. Our experiences with this method of proximal compression (which we shall call “PC”) are classified into angiograms of the following groups:

Group 1. Patients (classified as normal) suffering from headaches, epilepsy, migraine, trigeminal neuralgia, etc. (22 cases).

Group 2. Patients with increased intracranial pressure due to non-meningiomatosus tumors (13 cases).

Group 3. Patients with meningiomas and arteriovenous aneurysms supplied by the external carotid artery (14 cases).

Group 4. Patients with stenotic or occlusive disease of the internal carotid artery (5 cases).

Group 5. Patients with diagnostic peculiarities in the intracranial internal carotid PC angiogram (6 cases).

In this preliminary study, blood flow and pressure in the internal and external carotid arteries following occlusion of the common carotid were not measured.

Technique

The carotid artery is punctured at the level of C-4 or C-5, the bevel of the needle tip being directed upward. After the vessel is entered, the bevel is turned downward and the needle is gently advanced into the vessel. After the routine angiograms are completed,
Slow Flow Carotid Angiography

the PC angiograms are made, with the anesthetist exercising compression with a sterile gloved finger on the ipsilateral common carotid artery at the C-6 level below the site of puncture. It is not easy to be sure whether the compression results in complete occlusion. Tindall, et al., showed that reduction of blood flow or pressure above the site of graded occlusion did not occur until a 70% to 90% reduction in cross-sectional area of the lumen was obtained. One of our cases of stenotic lesions of the internal carotid artery with reduction to approximately one ninth of the cross-sectional area of its lumen showed a normal angiographic circulation time, while another patient with reduction to one thirtieth of the cross-sectional area displayed properties of slow flow at routine angiography. Sweet and Bennett stressed how difficult it is to secure through the intact skin a full occlusion of the vessel that consistently results in a reliable and maximal drop in pressure. We used as criteria the disappearance of a pulse in the temporal artery and the cessation or reduction of reflux of blood from the angiographic needle.

The resistance to injection of contrast medium in PC angiography is increased in the majority of cases but there was no correlation between this increase and the angiographic effect; in some instances there was no increased resistance at all. The most reliable criterion of proximal occlusion was found in the PC angiograms. With effective occlusion, the internal and external carotid arteries or one of them were well visualized for 3 sec following injection of the contrast medium. Using this radiographic criterion we had to discard six of the early angiograms because of inadequate compression. With increasing experience, the number of cases showing defective compression during the first shot became negligible. A 17- or 18-gauge needle was used. After digital proximal compression of the common carotid was established, which took place in 3 or 4 sec, 8 cc of 60% urografin were injected within about 1.5 sec. When 3 cc were left in the syringe, the first film was exposed, followed by two other exposures with intervals of 1.5 sec. After the third film had been exposed, digital compression was released. We found that rapid serial angiography did not give better information with this form of slow flow angiography. The total duration of digital compression did not exceed 9 to 10 sec; the prolonged staining of the carotids was 3 sec.

Results

In our series of 60 patients (plus 27 subsequent ones), no clinical complications were observed. The results are shown in Table 1. The total number of cases, however, is too small for an accurate evaluation of the complication rate.

**Table 1**

<table>
<thead>
<tr>
<th>Result</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral arteries not opacified; arrest of contrast medium in carotid</td>
<td>29</td>
</tr>
<tr>
<td>Shortlasting early or late staining of middle cerebral artery</td>
<td>20</td>
</tr>
<tr>
<td>Prolonged staining (3 sec): Middle cerebral artery</td>
<td>9</td>
</tr>
<tr>
<td>Middle and anterior cerebral arteries</td>
<td>2</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
</tr>
</tbody>
</table>

**Group 1 (Normal) and Group 2 (Increased Intracranial Pressure)**

Internal Carotid PC Angiograms. Group 1 consisted of 22 patients and Group 2 of 13 patients. Table 2 shows a higher incidence of arrest of the contrast medium in the internal carotid artery in the PC angiograms of Group 2 than in the normal Group 1 series. Filling of the anterior and posterior cerebral arteries was suppressed in nearly all PC angiograms, facts already described by other authors as a sign of compression of the carotid artery in the neck. The various types of intracranial circulation are demonstrated in Figs. 1 and 2. Even with a poorly visualized middle cerebral artery, leptomeningeal vessels were stained in some of the angiograms. In some cases the transport of contrast medium through the middle cerebral artery seemed to be affected by the collateral circulation on the carotid siphon, the latter being washed out while the internal carotid remained well opacified in all three films (Fig. 2). In 15 patients,