Femorocerebral catheter angiography has been the accepted technique of choice in evaluating the arterial tree of the head and neck for many years. It has virtually replaced direct puncture of the axillary, brachial, carotid, or vertebral arteries except in very rare cases. The risks of this procedure have been studied by many investigators1-27 over the last 20 years and appear to have gradually decreased (Table 1). This is probably due to marked improvements in angiographic equipment, operator dexterity, and the more widespread use of small catheters and better-designed guidewires. The use of heparinized catheters and guidewires2-35 may have contributed to this decrease. Systemic heparinization may offer additional protection to selected patients,26-30 but this remains to be proven.

Classically, studies evaluating the risks of cerebral angiography have been retrospective, and none included events occurring >24 hours after angiography. At the time this study was commenced, there were no detailed prospective studies in the literature, although 1 has since been published.9

We prospectively evaluated clinical events occurring up to 72 hours after cerebral angiography in 1,002 consecutive procedures performed on 724 patients. Our purpose was to identify predictable risk factors and to compare these with risk factors previously identified by other groups. As well, we recorded events occurring at 24-72 hours after angiography in an attempt to document either delayed cerebral ischemia following angiography or a trend in the patients’ natural history of those diseases that had led to the diagnostic procedure.

Subjects and Methods

One thousand two consecutive procedures performed on 724 patients over a 15-month period from March 1983 to May 1984 were evaluated prospectively. Each patient had a neurologic examination performed by the same neurologist prior to, at 24 hours after, and at 72 hours after angiography. Patients with events were followed until recovery or stabilization. In 25.8% of the cases, part of the clinical information had to be retrospectively gathered from the chart records. This most commonly consisted of urgent cases where the preliminary neurologic examination was done by a different physician. Since these patients were being monitored closely, it is doubtful that significant changes in status were overlooked in the postangiography follow-up period.

Records were made of age, sex, indication for angiography, medical history [stroke, transient ischemic attacks (TIAs), subarachnoid hemorrhage, myocardial infarction, angina, intermittent claudication, hypertension, seizures, headache, diabetes, smoking], medication (i.e., aspirin, aminocaproic acid, etc.), relevant neurologic symptoms and dates, abnormal neurologic findings (evaluated by the same neurologist both before and after angiography), pertinent laboratory values (blood urea nitrogen, creatinine, prothrombin time, partial thromboplastin time, hemoglobin, hema-
Blood pressure was recorded in both arms, and the patients were evaluated for the presence of cervical and other bruits. Peripheral pulses were examined and graded (0–3). Blood urea nitrogen and creatinine were studied 24 hours after angiography.

Parameters recorded in the neuroangiography suite included premedication (type and dosage, route of administration); type of anesthesia (local, general); vessels catheterized, volume and number of injections per vessel; type and total amount of contrast used; administration of an intra-arterial heparin bolus (2,000 units); reversal of heparinization with protamine (usually 20 mg protamine at the end of the procedure). A combination of careful double-flush technique and constant heparinized saline infusion (3,000 units/500 ml normal saline) was used. Our standard angiographic protocol was tailored to different types of suspected pathologic processes as follows: for TIA and/or stroke — anteroposterior and lateral views of both carotid arteries (head and neck), right posterior oblique view of the aortic arch; for aneurysms — anteroposterior, lateral, and oblique views of both carotid arteries (head), anteroposterior Towne and lateral views of 1 vertebral artery; for arteriovenous malformations (AVMs) — anteroposterior and lateral views of both carotid arteries, anteroposterior Towne and lateral views of 1 vertebral artery; and for post-operative — anteroposterior and lateral views of the artery or territory that was involved. Additional projections were added as appropriate. All injections were performed with a power injector. Conray 60 (Mallinkrodt Canada, Pointe-Claire, Canada) was employed in all cases except for aortic arch angiography, in which MD-76 (Mallinkrodt) was used. Standard injection rates and volumes were as follows: for common carotid artery, 8–10 ml/sec for 12 ml; for internal carotid artery, 8 ml/sec for 10 ml; for external carotid artery, 2–3 ml/sec for 5 ml; for vertebral artery, 7 ml/sec for 9 ml; for subclavian artery, 8 ml/sec for 16 ml with inflated arm cuff; and for aortic arch, 25 ml/sec for 50 ml. During this study, virtually all filming was done by film changer. Pressure was applied at the puncture site manually. All procedures were performed by senior radiology residents or neuroradiology fellows under the close supervision of a staff neuroradiologist, who became technically involved in more difficult cases.

A neurologic event was defined as any neurologic sign or symptom occurring during the procedure or in the subsequent 72 hours, whether it was considered a manifestation of the primary disease or not, and was
defined as transient if it lasted < 1 week and had no permanent sequelae and as permanent if it lasted > 1 week or carried permanent sequelae. A nonneurologic event was defined as any sign or symptom occurring either locally at the puncture site or systemically (i.e., angina, shortness of breath, etc.).

Neurologic events were recorded according to time, occurring either immediately or up to 30 minutes following the procedure, 30 minutes to 24 hours, 24–48 hours, and 48–72 hours. Events were also categorized as recurrence of the preangiographic symptoms, as worsening of a patient’s condition, or as de novo occurrences. When hematomas occurred, they were classified as small (< 5 cm in diameter), as moderate to large but not requiring therapy (5–10 cm), or as large (> 10 cm) requiring some form of therapy (correction of hypotension or surgery).

Tests of validity were performed as follows: Student’s t tests were used to evaluate the associations of continuous variables; a corrected χ2 analysis was applied to assess the associations of discrete variables. We defined p ≤ 0.05 as significant and a p value between 0.1 and 0.05 as a trend.

Results

Five hundred seven procedures (50.6%) were in males and 495 (49.4%) in females. Ages ranged from 4 to 78, with a mean of 47.6 years. A little more than 50% of the patients were > 50 years of age. The following histories were present: smoking (44.8%), subarachnoid hemorrhage (35.6%), hypertension (29.3%), TIA (19%), stroke (17.4%), seizures (13.1%), headaches (10.4%), angina (7.8%), diabetes (7.1%), myocardial infarction (6.6%), and claudication (4.6%). At the time of the procedure, 25.7% of the patients were taking aspirin, 2.4% persantine, 0.1% sulfipyrazone; 0.6% were receiving anticoagulant therapy and 8.6% ampicinocortic acid therapy. Evaluation of TIA and/or stroke was the most common indication, followed closely by postoperative evaluation of aneurysms and preoperative evaluation of aneurysms and AVMs (Table 2). The relatively high proportion of subarachnoid hemorrhages, AVMs, and aneurysms is due to our referral pattern. Other indications such as vasculitis, assessment of endarterectomy and extracranial–intracranial bypass, amyotrophic lateral sclerosis, and moyamoya disease accounted for 6.4%. Systolic hypertension (blood pressure ≥ 160 mm Hg) was present in 9.9% and diastolic hypertension (blood pressure ≥ 100 mm Hg) in 3.7% of our patients at the time of angiography; the presence of a carotid bruit was recorded in 15.3%. The preangiographic creatinine level was available in 962 procedures; it was normal (< 120 IU) in 93%. In 653 procedures, the postangiographic creatinine level was available; it was normal in 89%. Of the 692 procedures in which both the pre- and post-angiographic creatinine levels were available, 27 (3.9%) declined to an abnormal laboratory level of renal function (the creatinine increase usually not > 30 IU), but none had any clinical manifestations. Only 2 of the 27 patients in that group had received > 200 ml contrast material during the procedure.

Catheterizations were performed on 2,438 arteries for a total of 4,176 injections. The distribution of catheterized arteries expressed as percent of the 1,002 angiographic procedures was as follows: right common carotid artery (67.7%), left common carotid artery (67.4%), left vertebral artery (45.3%), aortic arch (21.3%), right vertebral artery (12.7%), right internal carotid artery (6.2%), left internal carotid artery (6.1%), left subclavian artery (5%), right subclavian artery (4.6%), right external carotid artery (4.2%), and left external carotid artery (3.3%). Five hundred twelve patients had 1 angiogram, 160 had 2, 40 had 3, 10 had 4, and 2 had 5. In 42% of the procedures, 3 vessels were studied, while in 24 and 22%, respectively, 1 and 2 vessels were studied; 4–6 vessels were studied in 11.5% of the patients. Three patients underwent angiography but vessel catheterization was unsuccessful. In all procedures, a 5-French catheter was used as the initial catheter; in 278 procedures, a 6.5-French catheter was also used. The mean number of catheters for the entire population was 1.4; only 1 catheter was used in 67.8% of the procedures, 2 in 24%, 3 in 5.7%, and 4–6 in 1.9%. Although 6 guidewires were used in 1 procedure, only 1 guidewire was used in 30% and 2 in 57.9% of the procedures. A heparin bolus (systemic heparinization) was given in 32.3% of procedures. The mean volume of contrast material used per angiogram was 93 ml, with a maximum of 408 ml in 1 instance; in only 5.2% of the procedures was > 200 ml necessary. The mean duration of the procedure for the entire group was 50.2 minutes. Two percent of the procedures lasted > 2 hours, and 94% lasted < 90 minutes. The mean compression time was 22 minutes, but it was increased by approximately 5–6 minutes by using > 1 catheter, by using a 6.5-French rather than a 5-French catheter, and by the presence of atherosclerosis.

Tables 2, 3, 4, and 5 summarize the event rate observed in this study according to indications for angiography, age, type of event (repeat, worsening of a preexisting status, or de novo), category of event (transient neurologic, permanent neurologic, or nonneurologic), and time of onset (during the procedure to 30 minutes after, 30 minutes to 24 hours, 24–48 hours, or 48–72 hours).

Neurologic Events

The neurologic event rate between 0 and 24 hours was 1.3%. Only 1 patient (0.1%) suffered a permanent neurologic event in this period; he was a 44-year-old man with a Grade IV subarachnoid hemorrhage (secondary to a ruptured posterior fossa AVM) who rebled 24 hours after angiography and had a brainstem stroke. There were no deaths (Table 2). There was no significant increase in neurologic events within the first 24 hours in patients who had been maintained on ampicinocortic acid therapy prior to the procedure (8.9%). It is interesting to note that in the first 24 hours, slightly over one half of the neurologic events occurred in the
Table 2. Neurologic Events According to Indication and Angiographic Technical Data

<table>
<thead>
<tr>
<th>Indication</th>
<th>No. of procedures (N = 1,002)</th>
<th>Mean age (years)</th>
<th>No. catheters</th>
<th>Duration (min)</th>
<th>Neurologic Transient 0-24 hrs</th>
<th>Neurologic Permanent 0-24 hrs</th>
<th>Non-neurologic 0-24 hrs</th>
</tr>
</thead>
<tbody>
<tr>
<td>TIA/stroke</td>
<td>28.4</td>
<td>57.7</td>
<td>1.9</td>
<td>60.4</td>
<td>2.5% (7)</td>
<td>1.4% (4)</td>
<td>0.7% (2) 14.0%</td>
</tr>
<tr>
<td>Known aneurysm and AVM</td>
<td>21.8</td>
<td>43.1</td>
<td>1.2</td>
<td>53.4</td>
<td>0.9% (2)</td>
<td>0.9% (2)</td>
<td>0.4% (1) 6.6%</td>
</tr>
<tr>
<td>Postoperative aneurysm</td>
<td>22.6</td>
<td>47.9</td>
<td>1.3</td>
<td>37.6</td>
<td>1.3% (3)</td>
<td>0.9% (2)</td>
<td>0.4% (1) 3.7%</td>
</tr>
<tr>
<td>SAH (recent)</td>
<td>12.0</td>
<td>43.9</td>
<td>1.3</td>
<td>51.7</td>
<td>0.8% (1)</td>
<td>2.5% (3)</td>
<td>0.9% (1) 5.0%</td>
</tr>
<tr>
<td>Postoperative AVM</td>
<td>8.8</td>
<td>33.7</td>
<td>1.1</td>
<td>44.4</td>
<td>3.4% (3)</td>
<td>0.0% (1)</td>
<td>2.3% 4.6%</td>
</tr>
<tr>
<td>Others</td>
<td>6.4</td>
<td>46.1</td>
<td>1.3</td>
<td>50.3</td>
<td>1.6% (1)</td>
<td>1.6% (1)</td>
<td>0.2%</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>47.6</td>
<td>1.4</td>
<td>50.2</td>
<td>1.2% (12)</td>
<td>1.5% (15)</td>
<td>0.1% (1) 0.3% (3) 7.2%</td>
</tr>
</tbody>
</table>

TIA, transient ischemic attack; AVM, arteriovenous malformation; SAH, subarachnoid hemorrhage.

TIA group, which represents only 28.4% of the population studied (Table 3). However, after subjecting this to $\chi^2$ analysis, we could demonstrate only that patients in this group showed a trend toward having more neurologic events in the first 24 hours ($p = 0.09$, $\chi^2$). None of the other groups were shown statistically to be at higher risk.

Patients in the TIA group were significantly older (mean age 57.7 years), more catheters and guidewires were used during angiography, which lasted longer, when compared with the rest of the patients (Table 2). One may then deduce that this group could have suffered more events on the basis of the aforementioned factors; however, when identical statistical analysis was applied to the same factors within the TIA group, none significantly increased the likelihood of an event within 24 hours. It is thus possible that merely being part of that group is the cause for the increased number of events.

Of all the parameters studied (Table 3), the only 2 factors that significantly increased neurologic risk in the first 24 hours were procedure lasting >60 minutes ($p = 0.03$, $\chi^2$) and systolic hypertension ($p = 0.04$, $\chi^2$). Other factors that showed a trend included volume of contrast material used ($p = 0.07$, t test), use of ≥3 catheters ($p = 0.08$, $\chi^2$), and a preangiographic creatinine level of >120 IU ($p = 0.08$, $\chi^2$). In this last group, we observed a 4.1% (3 patients) incidence of neurologic events between 0 and 24 hours compared with 1.0% for the rest of the group. The fact that we could show only a trend is probably due to the relatively small number of patients in this group. Elevated creatinine level was first described in 1983 by Earnest et al9 as a significant angiographic risk factor.

Table 3. Significant Factors and Trends

<table>
<thead>
<tr>
<th>Type of event</th>
<th>Significant (p≤0.05)</th>
<th>Probability level</th>
<th>Trend (0.05≤p≤0.1)</th>
<th>Probability level</th>
</tr>
</thead>
<tbody>
<tr>
<td>Neurologic 0-24 hrs</td>
<td>Duration &gt;60 min</td>
<td>0.03</td>
<td>Contrast volume</td>
<td>0.07</td>
</tr>
<tr>
<td>Neurologic 24-72 hrs</td>
<td>Contrast volume</td>
<td>0.001</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diabetes mellitus</td>
<td>0.03</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neurologic 24-72 hrs</td>
<td>Contrast volume</td>
<td>0.0000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>TIA as indication</td>
<td>0.0000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Age</td>
<td>0.0000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥3 catheters</td>
<td>0.0000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>6.5-French</td>
<td>0.000</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Duration &gt;60 min</td>
<td>0.002</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Carotid bruit</td>
<td>0.006</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systolic + diastolic hypertension</td>
<td>0.007</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Diastolic hypertension</td>
<td>0.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Systolic hypertension</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Any bruit</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Number of guidewires used</td>
<td>0.02</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

TIA, transient ischemic attack.
Some of the parameters that did not significantly influence the occurrence of a neurologic event in the first 24 hours included concurrent aminocaproic acid therapy, sex, the use of only 5-French catheters vs. 5-French and 6.5-French, and the number and type of vessels injected per procedure.

Fifteen patients (1.5%) suffered a transient neurologic event 24–72 hours after angiography (Table 2). Three (0.3%) had a permanent neurologic event: a 75-year-old man with preangiographic TIAs in the left middle cerebral artery territory who had a hemorrhagic infarction 48 hours after angiography and moderate right hemiparesis and aphasia, a 71-year-old man with preangiographic TIAs in the right middle cerebral artery territory who had a permanent moderate left hemiparesis 72 hours after angiography, and finally, a 54-year-old man who had angiography (which demonstrated moderate to severe posterior fossa vasospasm) to verify a basilar aneurysm clip placement suffered a brainstem infarct 72 hours after angiography.

Factors that significantly increased the neurologic events between 24 and 72 hours (Table 3) included volume of contrast material used ($p = 0.001$, $t$ test) (this remained significant within the TIA group, $p = 0.02$, $t$ test), age ($p = 0.02$, $t$ test), and the presence of diabetes mellitus ($p = 0.03$, $\chi^2$).

Table 4 lists neurologic events by age groups. No neurologic event (reversible or permanent) occurred in patients <30 years of age (17.4% of the procedures). In patients >30 years of age, we demonstrated a significant increase in neurologic events between 24 and 72 hours with advancing age ($p = 0.02$, $t$ test).

In Table 5, neurologic events are classified according to type: repeat of a preangiographic event, worsening of preangiographic situation, or de novo occurrences. De novo events accounted for 1.1% of the total (3.1%), but their proportion was higher (0.7%) in the first 24 hours, and especially in the first 30 minutes following angiography (0.5% of 0.6%), when compared with the 24–72-hour period (0.4% of 1.8%). These are the events that were most likely to have been caused directly by angiography, and not by the primary disease. Repeat and worsening events accounted for 2.0% (0.7 and 1.3%, respectively) of the total (3.1%). These could have been caused indirectly by angiography (i.e., delayed contrast-induced platelet aggregation, hypotension secondary to groin hematoma, etc.) or may actually represent an ongoing manifestation of the primary problem.

### Table 4. Neurologic Events According to Age Group

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>% (N)</th>
<th>% Transient (n)</th>
<th>% Permanent (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–9</td>
<td>0.8 (8)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>10–19</td>
<td>4.8 (48)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>20–29</td>
<td>11.8 (118)</td>
<td>0 (0)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>30–39</td>
<td>14.4 (144)</td>
<td>1.4 (2)</td>
<td>2.0 (3)</td>
</tr>
<tr>
<td>40–49</td>
<td>15.4 (154)</td>
<td>0.7 (1)</td>
<td>1.3 (2)</td>
</tr>
<tr>
<td>50–59</td>
<td>26.4 (265)</td>
<td>1.9 (5)</td>
<td>1.4 (4)</td>
</tr>
<tr>
<td>60–69</td>
<td>20.9 (210)</td>
<td>1.4 (3)</td>
<td>1.9 (4)</td>
</tr>
<tr>
<td>70–79</td>
<td>5.5 (55)</td>
<td>1.8 (1)</td>
<td>3.6 (2)</td>
</tr>
<tr>
<td>Total</td>
<td>100 (1,002)</td>
<td>1.2 (12)</td>
<td>1.5 (15)</td>
</tr>
</tbody>
</table>

### Table 5. Type of Neurologic Events Between 0 and 72 Hours

<table>
<thead>
<tr>
<th>Time of onset (hrs)</th>
<th>Repeat</th>
<th>Worsening*</th>
<th>De novo</th>
<th>All</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–0.5</td>
<td>0.1%</td>
<td>0%</td>
<td>0.5%</td>
<td>0.6%</td>
</tr>
<tr>
<td>0.5–24</td>
<td>0.2%</td>
<td>0.3%</td>
<td>0.2%</td>
<td>0.7%</td>
</tr>
<tr>
<td>24–48</td>
<td>0.3%</td>
<td>0.5%</td>
<td>0.3%</td>
<td>1.1%</td>
</tr>
<tr>
<td>48–72</td>
<td>0.1%</td>
<td>0.5%</td>
<td>0.1%</td>
<td>0.7%</td>
</tr>
<tr>
<td>Total</td>
<td>0.7%</td>
<td>1.3%</td>
<td>1.1%</td>
<td>3.1%</td>
</tr>
</tbody>
</table>

*All 4 permanent events in this category.

### Table 6. Hematomas

<table>
<thead>
<tr>
<th>Age group (yrs)</th>
<th>Small</th>
<th>Moderate-to-large</th>
<th>Large with therapy</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–29</td>
<td>174</td>
<td>4.23</td>
<td>0.0</td>
<td>4.23</td>
</tr>
<tr>
<td>30–39</td>
<td>144</td>
<td>3.21</td>
<td>1.07</td>
<td>4.28</td>
</tr>
<tr>
<td>40–49</td>
<td>154</td>
<td>4.26</td>
<td>2.6</td>
<td>9.58</td>
</tr>
<tr>
<td>50–59</td>
<td>265</td>
<td>12.4</td>
<td>3.11</td>
<td>16.6</td>
</tr>
<tr>
<td>60–69</td>
<td>210</td>
<td>21.00</td>
<td>5.24</td>
<td>26.12</td>
</tr>
<tr>
<td>70–79</td>
<td>55</td>
<td>7.127</td>
<td>3.55</td>
<td>10.18</td>
</tr>
<tr>
<td>Total</td>
<td>1,002</td>
<td>51.51</td>
<td>10.51</td>
<td>69.51</td>
</tr>
</tbody>
</table>
Discussion

Cerebral angiography is already established as a safe way of investigating the cerebral vascular tree, although it does carry a small, but real, risk that has been investigated by many authors.1-27 This risk is usually in the form of neurologic symptoms (transient or permanent), local complications (groin hematoma, distal limb ischemia), or systemic derangements (angina, renal failure, etc.). While the mechanisms and causes for the latter two categories are fairly limited, there exist many possible causes for the occurrence of neurologic symptoms secondary to cerebral angiography; these range from mechanical detachment of arterial wall plaques by the catheter or guidewire with distal embolization, to clot formation in or around the catheter as well as on guidewires, again with distal embolization, to platelet activation,44-46 change in red blood cell morphology,45,46 and blood viscosity,47 to endothelial damage caused by contrast agents22,2348 and direct neurotoxic effect from contrast material49; as well, the presence of a catheter in a neck vessel is also capable of producing spasm, which may lead to neurologic symptoms.

Although the custom is to assume that any event following angiography has been caused by it, the culprit may be the primary disease, which continues to manifest itself. Baum et al50 in 1966 published an interesting report on the complication of “no angiography” in which the clinical complication rate of patients scheduled for peripheral angiography but whose examination was subsequently cancelled was close to that of patients who had undergone angiography. The natural history of disease may account for a proportion of so-called complications.

The present study was undertaken because of the lack of large-scale prospective studies on the risks of cerebral angiography. One has since been published.6 Prior studies consistently stopped monitoring patients for adverse effects that might have resulted from angiography 24 hours after the procedure if nothing abnormal had occurred. In our study, patients were followed for 72 hours after angiography to detect any possible delayed events.

Our population contained a lesser proportion of patients with TIA and/or stroke as an indication for angiography when compared with other studies. Earnest et al6 reported a higher (but not significant) percent of reversible (3.6%) and permanent (0.6%) neurologic events at 24 hours in patients investigated for cerebrovascular disease when compared with their overall rates of 2.3 and 0.3%, respectively. In this study (Table 2), the same pattern was observed for reversible (2.5%) but not permanent (0%) events in the same type of patients when compared with our overall rates of 1.2 and 0.1%. An identical pattern persisted into the 24-72-hour observation period for both reversible (1.4%) and permanent (0.7%) events.

Another point of difference with most other studies is that a full detailed neurologic examination was performed by a neurologist (and not a radiologist) before as well as 24 and 72 hours following angiography; it is much less likely that subtle changes were missed. In most previous publications, complications were identified either by retrospectively reviewing the chart or after a radiology resident or staff member had examined the patient. Not every investigator considered worsening of the patient’s status as a complication, although Earnest et al,6 Faught et al,2 and Olivecrona1 recorded events according to this method, avoiding biased judgments.

Our reversible neurologic event rate of 1.2% within the first 24 hours compares favorably with that of other reports (Table 1), which range between 0.9% (Mani et al4,6) and 4.9% (Chynn10). The permanent neurologic event rate within 24 hours is surprisingly constant throughout the years and for different investigators, ranging between 0% (Chynn,10 Kerber et al3) and 0.33% (Earnest et al6); it is 0.1% in our report.

The only factors that significantly increased the risk for neurologic events within 24 hours were duration of procedure >60 minutes and systolic hypertension, although we showed trends with increasing volume of contrast material, the use of ≥3 catheters, TIA as an indication, and a preangiographic creatinine level >120 IU (Table 2). Most authors agree that patients with atherosclerosis are at increased risk. Faught et al2 and Earnest et al6 could not show an increased neurologic risk with hypertension. Mani et al4,6 had results similar to ours in that they demonstrated increased risk with duration >80 minutes, but Olivecrona1 did not find this factor significant. He showed more risk in the left carotid and right vertebral arteries compared with the right carotid and left vertebral; we could not show any pattern with vessel injected.

Using smaller, softer 5-French catheters might logically decrease the rate of neurologic complications (Kerber et al,3 Mani et al,4,6 Eisenberg et al19,20), but we were not able to prove this relation. As well, we were unable to corroborate Faught et al2 who found women to be at significantly increased risk.

Although Earnest et al6 showed that the use of ≥1 catheter was a significant neurologic risk, we were not able to show an increase in risk until 3 catheters had been used. The same group was the first to identify an increased creatinine level (>120 IU) as a neurologic risk factor; in our study, we paralleled that finding by showing a trend (p = 0.08, χ²); we suspect that, had we studied more procedures, we would have shown the same relation.

Many factors (volume of contrast material, duration of procedure, TIA as an indication for procedure, age, number of catheters and guidewires used, size of catheters used, presence of a carotid or any vascular bruit, systolic and diastolic hypertension) were shown to significantly increase the likelihood of nonneurologic events occurring (Table 3). Adequate hydration may have played a role in reducing the number of patients with altered renal function studies (3.9%).

Many of the measured parameters were intricately related; for example, a long procedure was likely to have resulted from more contrast material, the use of more catheters and guidewires, and was usually per-
formed in older patients with atherosclerosis. The low frequency of complications precluded further multivariate analysis. Digital subtraction angiography, the use of 4-French catheters, the increased use of duplex ultrasonography, and new nonionic contrast media may further decrease the dangers of cerebral angiography.

Acknowledgments

The authors wish to thank the following neurologists and neuroradiologists for their assistance: V. Hachinski, F. Vinuela, D.M. Pelz, W. Wong, T. Constantino, J.A. Rem, S. Spiegel, J.K. Chang, Y.H. Liu, and R. Ferguson, as well as Matthew Maynard for statistical analysis, and Sylvia Boulanger and Ann Hammond for secretarial assistance.

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Key Words: cerebral angiography • complications of cerebral angiography
Clinical events following neuroangiography: a prospective study.
J E Dion, P C Gates, A J Fox, H J Barnett and R J Blom

Stroke. 1987;18:997-1004
doi: 10.1161/01.STR.18.6.997

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