Duplex Scanner Study of Carotid Artery Dissection Following Surgical Treatment of Aortic Dissection Type A

H.R. Zurbrügg, MD, F. Leupi, MD, P. Schüpbach, MD, and U. Althaus, MD

In patients suffering from aortic dissection, persistent perfusion of the false lumen distal to the implanted graft is frequent. Postoperative follow-up examinations of the carotid arteries of these patients were performed by duplex scanner and correlated with clinical symptoms. Thirty-nine patients who survived the surgical treatment of acute type A aortic dissection had duplex sonography of both common carotid arteries after an average postoperative follow-up of 53 months. In 21 cases a composite graft and in 18 cases a supracoronary prosthetic vascular graft were implanted. No sign of residual dissection of the common carotid arteries was seen in 23 patients; in nine there was a dissection of both common carotid arteries, and seven patients had a unilateral carotid dissection (five right, two left). There were nine symptomatic patients with the following symptoms: transient ischemic attack (four), amaurosis fugax (four), stroke with incomplete recovery (one). Two symptomatic patients had a corresponding dissection. The generally good prognosis of all these patients suggests a conservative nonoperative treatment. (Stroke 1988;19:970-976)

After surgical treatment of type A aortic dissection, persistent perfusion of the false lumen of the aorta distal to the implanted graft is a frequent occurrence.1-3 The incidence and clinical importance of asymptomatic or symptomatic dissection of the carotid-innominate vessels secondary to aortic dissection have not been investigated. This article deals with the problems of diagnosis, prognosis, and treatment of patients with these conditions.

Subjects and Methods
Since 1969, 61 patients with type A aortic dissection were successfully operated on at our clinic. Type A classification is based on the presence of dissection of the ascending aorta, whereas in type B only the descending aorta is involved. Type A includes DeBakey types I and II; type B corresponds to DeBakey type III (Figure 1). It was decided to conduct a follow-up investigation of the carotid-innominate vessels in these patients. At the time of the investigation, 46 patients were still alive; 15 patients had died due to sudden cardiac death (5), aortic rupture (3), cancer (2), subdural hematoma (1), reoperation (1), or unknown causes (3), and seven patients refused to take part in the study. The 39 patients who agreed to participate were examined in August-September of 1986. On the day of the study, the patients were interviewed and underwent a complete clinical examination. The mean ± SD age of the seven women and 32 men at that time was 56 ± 12 years; the mean postoperative follow-up was 53 ± 50 months. Patients who were on antihypertensive or antidiabetic drugs were considered as hypertensive or diabetic, respectively. Patients who consumed at least one cigarette/day were considered smokers. Patients whose body weight in kilograms was greater than their stature in centimeters minus 100 were considered obese.

Duplex sonography of both common carotid arteries of all these patients was performed with a duplex scanner (Diasonics DRF 400 ADA) equipped with a 10-MHz probe. Dissection of the common carotid artery (CCA) was diagnosed only when the intimal flap was clearly identified in the transverse and in two different longitudinal sections. The longitudinal sections were examined from the anterior and lateral approaches so that the angle between the sectional planes was about 90°.

The risk of stroke complicating open heart surgery ranges between 2% and 5%.7,8 The most common causes of brain infarction during or after operation are embolism related to microaggregates, cross-clamping of a brittle aorta, air in the heart or...
bypass lines, mural or valvular thrombus, or dysrhythmia. The precise etiology of intraoperative or early postoperative neurologic events is, in most cases, speculative. As this article deals exclusively with the prognosis of cerebrovascular events related to the carotid vessels, only clear neurovascular events with hemispheric or monocular symptoms occurring >30 days after surgery were included.

**Results**

No signs of residual dissection were seen in 23 patients. In nine there was a dissection of both CCAs (Figure 2), and seven patients had a unilateral carotid dissection (Figures 3 and 4, Table 1). The right side was more commonly affected (14 times) than the left (11 times). In only one patient was the false lumen occluded; this patient suffered a dense left hemiplegia. In all other dissected vessels both the false and true lumens were perfused. In at least one lumen a broad systolic peak and positive end-diastolic blood flow velocity was observed. In six patients the other lumen showed not the characteristic Doppler signal of a CCA but a reverberating flow. Identification of the true lumen was possible in some cases. Atherosclerotic plaques (Figure 2) or a clear intima signal may point to the true lumen.

Patients with carotid dissection had a mean follow-up of 43 ±38 months, patients without dissection a mean follow-up of 60 ±57 months. During follow-up, nine patients suffered cerebrovascular events; in only two was there a corresponding dissection. In the remaining seven patients, the neurologic symptoms were attributed to the contralateral side. Four patients had transient ischemic attacks, and four had amaurosis fugax. One patient had a stroke with incomplete recovery. The mean ± SD age of these patients at the time of the event was 58 ± 8 years (Table 2). Other risk factors for cerebrovascular events (hypertension, diabetes, smoking, obesity) are shown in Table 3.

Among the nine patients with carotid dissection, the average interval from surgery to the first neurovascular event was 44 months; however, variation was considerable (Table 2). Four of the nine had had at least one recurring attack. Of the nine, seven had been treated with anticoagulants before the attack. In the other two, anticoagulation therapy was started after the neurovascular event. At no time was any patient treated with platelet inhibitors.

Among the 21 implantations of composite grafts (10 Björk-Shiley, 11 St. Jude Medical valves; Figure 5) six cases of cerebrovascular events were observed. There was, however, only one dissection with corresponding symptoms and signs (Patient 2,
FIGURE 3. Top: right common carotid artery (CCA) in left: longitudinal and right: transverse sections. Arrows point to intimal flap. Bottom: same vessel with time-velocity curves of blood in true and false lumens. Almost-reverberating, i.e., zero blood flow during cardiac cycle, is observed in smaller lumen. Areas under curve during systole and diastole do not differ significantly.
FIGURE 4. Top: left common carotid artery (CCA) in left: longitudinal and right: transverse sections. Arrows point to dissecting membrane. Bottom: same vessel with corresponding time-velocity curves of blood in true and false lumens. As in Figure 3, almost-reverberating blood flow is seen in one lumen.
Table 1. Patients With or Without Dissection of Common Carotid Arteries by Duplex Sonography After Surgery for Type A Aortic Dissection

<table>
<thead>
<tr>
<th>Dissection</th>
<th>Number</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bilateral</td>
<td>9</td>
</tr>
<tr>
<td>Unilateral</td>
<td>7</td>
</tr>
<tr>
<td>Right</td>
<td>5</td>
</tr>
<tr>
<td>Left</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>23</td>
</tr>
<tr>
<td>Total</td>
<td>39</td>
</tr>
</tbody>
</table>

Table 2). In 18 patients a supracoronary prosthetic graft was implanted. Neurologic events occurred in three of these, with a corresponding dissection in one patient (Patient 3, Table 2).

Discussion

Neurologic findings are common symptoms of type A aortic dissection. The prevalence and variability of these manifestations on admission, after surgery, and during the long-term course have been reported.6-13 However, these data cannot be compared with each other due to different selection criteria. In our group, 16 of 39 patients (41%) had a dissection of at least one CCA. This rate is markedly higher than those reported by other authors. In autopsy cases, the reported incidence of carotid involvement varies between 8% and 15%.14,15 This difference could be explained by two hypotheses. First, after the acute onset of aortic dissection and surgery, the persistent false lumen has a tendency to extend peripherally. Such a mechanism has not been reported for the carotid-innominate vessels but has been described for the descending aorta.6,12 On the other hand, no findings suggested that improvement or disappearance of some dissections could occur with time because the mean follow-up of patients with dissection (43 ± 38 months) is not significantly lower than that of patients without dissection (60 ± 57 months) (Student's t test, p<0.05).

We intend to investigate this problem in a further study. Second, the detection of a collapsed false lumen postmortem may be more difficult than detection of a filled false lumen in vivo. At autopsy it is sometimes very difficult by macroscopic and microscopic techniques to differentiate between artificial and true dissection. Furthermore, in our study, false-positive diagnosis of the additional lumen due to ultrasound reflections or electronic noise was unlikely as the dissection membrane was visualized in three different sections.

The postoperative cerebrovascular events corresponded with the dissection in only two of the nine symptomatic patients. Therefore, a carotid dissection probably need not be considered a risk factor for neurologic events. The prognosis usually is good, even with the high recurrence rate (44%). Only one symptomatic patient suffered a stroke with incomplete recovery, whereas all other symptomatic patients had only transient signs and symptoms. The prognosis of spontaneous acute and isolated carotid dissection seems to be much worse, with reports of one third to one half of these patients suffering a stroke.16-18 All these patients

Table 2. Age at First Neurologic Event, Postoperative Interval, and Number of Recurrences With Reference to Type of Graft, Initial Anticoagulation, and Corresponding Dissection After Surgery for Type A Aortic Dissection

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Interval (mo)</th>
<th>Recurrences (no.)</th>
<th>Type of graft</th>
<th>Initially anticoagulated</th>
<th>Corresponding dissection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. BJ</td>
<td>59</td>
<td>1</td>
<td>0</td>
<td>SJM</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>2. HP</td>
<td>68</td>
<td>1</td>
<td>0</td>
<td>BS</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>3. KH</td>
<td>48</td>
<td>3</td>
<td>5</td>
<td>SCG</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>4. ME</td>
<td>61</td>
<td>27</td>
<td>1</td>
<td>SJM</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>5. NP</td>
<td>62</td>
<td>12</td>
<td>0</td>
<td>BS</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>6. OF</td>
<td>66</td>
<td>24</td>
<td>1</td>
<td>BS</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>7. RF</td>
<td>44</td>
<td>155</td>
<td>3</td>
<td>SCG</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>8. SR</td>
<td>59</td>
<td>60</td>
<td>0</td>
<td>BS</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>9. WA</td>
<td>53</td>
<td>114</td>
<td>0</td>
<td>SCG</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

SJM, St. Jude Medical valve (composite graft); BS, Björk-Shiley valve (composite graft); SCG, supracoronary artery graft.
had an acute occlusion of the cervical internal carotid artery (ICA) when they became symptomatic. To our knowledge, patients without symptoms but with isolated dissection of the CCA or ICA have not been previously reported. Spontaneous reopening of the occluded vessel happens in approximately 40–60%. In these cases a persistent dissecting membrane usually does not occur. Therefore, spontaneous carotid dissection has to be clearly differentiated from chronic persistent dissection due to type A aortic dissection. Similar conclusions have been drawn by Zirkle et al, who described one case of carotid involvement in aortic dissection.

The interval from surgery to the first neurovascular event is not predictable for each individual case (see Table 2). It appears that at the time of release from the hospital, patients are at no more risk than those with long, symptom-free follow-ups. Anticoagulation therapy probably does not prevent cerebrovascular events. The reasons for subsequent neurovascular events, therefore, are unclear or only highly speculative, and there are no consistent data on the use of anticoagulant therapy in the literature or in our present study.

The risk of suffering a cerebrovascular event seems to be associated with the type of composite graft implanted. In our study, patients with a Björk-Shiley valve were at higher risk. However, the number of patients investigated was too small to draw any firm conclusion. Moreover, the patients had not been prospectively randomized, and the decision to implant a composite or a supracoronary graft was influenced mainly by the severity of the aortic disease. Due to the limited number of patients, a statistical analysis of other risk factors (Table 3) is of restricted value. Moreover, the possibility of finding a false-positive difference among nine statistical tests at a 5% significance level is 45%.

Our results indicate that persistent carotid dissection after surgical treatment of type A aortic dissection need not be considered a risk factor, and the generally good prognosis of these patients suggests a conservative nonoperative treatment.

References


**Key Words**  aorta • carotid arteries • dissection • ultrasonics
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