Assessment by Fluorescein Angiography of Surgical Treatment of Occlusive Carotid Artery Disease


Fundus fluorescein angiography was performed in 10 cases of occlusive carotid artery disease at presentation and after surgery or medical treatment. An improvement in the microcirculation of the retina was observed in 4 cases after carotid endarterectomy and in 1 case after carotid endarterectomy and extracranial–intracranial bypass. Improvement was not observed in 2 cases after extracranial–intracranial bypass alone or in 3 cases in which surgical intervention was not undertaken. Our results suggest that carotid endarterectomy is a more effective procedure than extracranial–intracranial bypass in improving retinal perfusion when compromised by ipsilateral carotid obstruction. (Stroke 1987;18:585–590)

Occlusive disease of the carotid artery may produce ocular signs of retinal emboli, chronic ocular ischemia, neovascular glaucoma, or venous-stasis retinopathy. This latter term was used by Kears and Hollenhorst for the combination of dilated veins, blossom-shaped hemorrhages, microaneurysms, and sludging of blood in the veins. Venous-stasis retinopathy has been found in 12% of patients with unilateral carotid occlusion and 17% of patients with bilateral carotid occlusion.

Fluorescein angiography is more sensitive than ophthalmoscopy in detecting minor microvascular abnormalities in the retina. Performing fluorescein angiography in patients with carotid occlusion or stenosis, we found abnormalities in 67% of cases. Fluorescein angiography may also be used in this group to assess whether carotid surgery improves retinal ischemia. This report reviews the findings of fluorescein angiography at diagnosis and follow-up in 10 patients with carotid occlusion or stenosis. Five patients had carotid artery surgery, 2 patients had extracranial–intracranial (EC–IC) anastomosis alone, and 3 patients had medical treatment only.

Subjects and Methods

These 10 cases may be considered in 4 groups depending on their management (Table 1). Two patients had superficial temporal-to-middle cerebral artery (ST–MCA) anastomosis alone (Group A), 1 patient had an external and internal carotid endarterectomy as well as a ST–MCA anastomosis (Group B), 4 patients had internal carotid endarterectomies (Group C), and 3 patients had medical treatment only (Group D).

Group A

A.D. first developed amaurosis fugax affecting the right eye in 1970 when he was aged 57. These attacks were accompanied by an ache in the right eye, but these symptoms resolved spontaneously over a few months. He had a history of iliac endarterectomy for intermittent claudication in 1969. In 1983 he had a recurrence of amaurosis fugax affecting both eyes. Visual acuities were 6/6 right and left. A left fundus fluorescein angiogram showed mild macular edema (Figure 1). Bilateral internal carotid artery (ICA) occlusion was confirmed by arterial angiography. In December 1983 a left ST–MCA anastomosis was performed. Following surgery he had no more attacks of amaurosis fugax, but he did experience occasional episodes of formed visual hallucinations in the left visual field. A fundus fluorescein angiogram repeated in September 1985 showed persistence of the left macular edema (Figure 2).

E.S. presented with left amaurosis fugax in 1978 when he was aged 60. Visual acuities were 6/6 right and left. A cholesterol embolus was seen in the left retina. A left carotid angiogram showed an atheromatous plaque at the origin of the left ICA. In January 1979 a left internal carotid endarterectomy was performed. In 1981 he developed attacks of blurring of right vision associated with photopsia and headache. In April 1981 he had an inferior myocardial infarction. In 1984 he developed left amaurosis fugax and attacks of weakness of the right hand. Visual acuities were 6/6 right and left. A left fundus fluorescein angiogram showed a few microaneurysms temporal to the fovea (Figure 3). Digital subtraction angiography (DSA) of the carotid arteries now showed an occlusion of the left ICA and a stenosis of the right ICA. A left ST–MCA anastomosis was performed in January 1985. The left fundus fluorescein angiogram repeated in June 1985 showed persistence of microaneurysms temporal to the fovea (Figure 4).


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Table 1. Carotid Status and Fluorescein Findings

<table>
<thead>
<tr>
<th>Patient</th>
<th>Group</th>
<th>Age</th>
<th>Carotid status</th>
<th>Fluorescein findings</th>
<th>Surgery</th>
<th>Follow-up</th>
<th>Fluorescein</th>
</tr>
</thead>
<tbody>
<tr>
<td>A.D.</td>
<td>A</td>
<td>57</td>
<td>Bilateral IC O</td>
<td>L macular edema</td>
<td>L ST-MCA</td>
<td>21</td>
<td>No change</td>
</tr>
<tr>
<td>E.S.</td>
<td>A</td>
<td>60</td>
<td>L IC O, R IC ST</td>
<td>L microaneurysms</td>
<td>L ST-MCA</td>
<td>6</td>
<td>No change</td>
</tr>
<tr>
<td>F.A.</td>
<td>B</td>
<td>64</td>
<td>L IC O, L EC ST, R IC ST</td>
<td>L slow A-V transit, L microaneurysms, L macular edema</td>
<td>L EC END, R IC END, LST-MCA</td>
<td>24</td>
<td>Improved</td>
</tr>
<tr>
<td>M.F.</td>
<td>C</td>
<td>59</td>
<td>R IC ST</td>
<td>R slow A-V transit, R microaneurysms</td>
<td>R IC END</td>
<td>6</td>
<td>Improved</td>
</tr>
<tr>
<td>F.W.</td>
<td>C</td>
<td>63</td>
<td>L IC ST</td>
<td>L slow A-V transit, L microaneurysms, L macular edema</td>
<td>L IC END</td>
<td>1</td>
<td>Improved</td>
</tr>
<tr>
<td>A.S.</td>
<td>C</td>
<td>63</td>
<td>L CC O, R IC ST</td>
<td>R microaneurysms</td>
<td>R IC END</td>
<td>1</td>
<td>Improved</td>
</tr>
<tr>
<td>N.S.</td>
<td>C</td>
<td>48</td>
<td>Bilateral IC ST</td>
<td>R microaneurysms</td>
<td>R IC END</td>
<td>5</td>
<td>Improved</td>
</tr>
<tr>
<td>L.S.</td>
<td>D</td>
<td>58</td>
<td>L CC O, R IC ST, R EC ST</td>
<td>L slow A-V transit, L microaneurysms, L macular edema</td>
<td>None</td>
<td>24</td>
<td>No change</td>
</tr>
<tr>
<td>M.M.</td>
<td>D</td>
<td>56</td>
<td>L CC O, R SC ST</td>
<td>L slow A-V transit, L microaneurysms, L macular edema</td>
<td>None</td>
<td>24</td>
<td>No change</td>
</tr>
<tr>
<td>W.B.</td>
<td>D</td>
<td>66</td>
<td>L IC O</td>
<td>L slow A-V transit, L microaneurysms, L macular edema</td>
<td>None</td>
<td>5</td>
<td>No change</td>
</tr>
</tbody>
</table>

R, right; L, left; IC, internal carotid; EC, external carotid; CC, common carotid; SC, subclavian; O, occlusion; ST, stenosis; ST-MCA, superficial temporal-middle cerebral artery anastomosis; A-V, arteriovenous; END, endarterectomy. Age in years; follow-up in months.

Group B

F.A. presented with sudden loss of vision in the right eye in 1978 when he was aged 64. Macular edema secondary to hypertensive retinopathy was diagnosed, and his vision improved over the next 5 months on antihypertensive treatment. In 1983 he developed amaurosis fugax affecting the left eye. Visual acuities were 6/6 right and 6/12 left. A left fundus fluorescein angiogram showed slow arteriovenous transit of dye, microaneurysms, and macular edema. DSA revealed a left ICA occlusion and a stenosis of the right ICA. A left carotid angiogram also showed a stenosis of the left external carotid artery. The left ophthalmic artery...
FIGURE 3. Left fundus fluorescein angiogram showing microaneurysms temporal to the fovea in E.S.

Filled via anastomosis from the left maxillary artery. In October 1983 a left external carotid endarterectomy was performed. Attacks of left amaurosis fugax persisted postoperatively. In September 1984 a right internal carotid endarterectomy was performed. In February 1985 a left ST-MCA anastomosis was done. A left fundus fluorescein angiogram in September 1985 showed resolution of microaneurysms and macular edema and more rapid arteriovenous transit of dye.

**Group C**

M.F. presented in May 1985 when she was aged 59 with a 6-month history of attacks of shaking of the left arm and leg. Attacks occurred once a day, lasted a few minutes, and were not accompanied by disturbance of consciousness. She had a history of bilateral iliac endarterectomies and a mitral valve replacement for rheumatic heart disease. Visual acuities were 6/9 right and 6/6 left. There were dilated retinal vessels and multiple hemorrhages in the right eye. A right fundus fluorescein angiogram showed delayed arteriovenous transit of dye and multiple microaneurysms. Carotid angiography revealed a long stenosis at the origin of the right ICA. A right internal carotid endarterectomy in June 1985 was followed immediately by a left hemiparesis that slowly improved over the next month. A right fundus fluorescein angiogram repeated in January 1986 showed a reduction in the arteriovenous transit time and fewer microaneurysms in the temporal retina.

F.W. presented in July 1985 when he was aged 63 with frequent episodes of left-sided amaurosis fugax. Visual acuities were 6/12 right (his right eye was ambyopic) and 6/5 left. In the left fundus the retinal veins were dilated, there were multiple hemorrhages, and the central retinal artery pressure was reduced. A left fundus fluorescein angiogram showed delayed arteriovenous transit of dye, peripheral microaneurysms, and areas of diffuse leakage at the posterior pole (Figure 5). DSA showed a tight stenosis of the left ICA at its origin. In April 1986 a left internal carotid endarterectomy was performed. A left fundus fluorescein angiogram repeated in May 1986 showed reduction in arteriovenous transit time, fewer microaneurysms, and resolution of the excess leakage of dye at the posterior pole (Figure 6).

A.S. presented in April 1986 when he was aged 63 with 2 episodes of transient right-sided weakness. He then developed amaurosis fugax affecting the right eye. Visual acuities were 6/6 right and left. A right fundus fluorescein angiogram showed peripheral microaneurysms. DSA showed occlusion of the left com-
FIGURE 6. Left fundus fluorescein angiogram showing less leakage at the posterior pole in F.W.

mon carotid artery and stenosis at the origin of the right ICA. In May 1986 a right internal carotid endarterecto-
my was performed. Right fundus fluorescein angiography repeated in June 1986 showed fewer microan-
eurysms in the periphery of the retina.

N.S. presented in November 1985 when he was aged 48 with episodes of blurred vision occurring espe-
cially when stretching his neck. Visual acuities were 6/4 right and left. A right fundus fluorescein angiogram showed a few microaneurysms at the posterior pole. DSA showed bilateral ICA stenosis. While under investigation he developed a mild left hemiparesis. A right internal carotid endarterectomy was performed in December 1985. A right fundus fluorescein angiogram repeated in May 1986 showed persistence of only 1 microaneurysm at the right posterior pole.

Group D

L.S. presented in 1980 when he was aged 58 with 4 episodes of expressive dysphasia. He also complained of transient graying of vision lasting a few minutes induced by exercise. He had a history of aortic and femoral endarterectomies for intermittent claudication in 1976 and an inferior myocardial infarction in 1980. In 1984, visual acuities were 6/6 right and left. There were dilated veins, multiple peripheral hemorrhages, and a low central retinal artery pressure in the left eye. A left fundus fluorescein angiogram showed delayed arteriovenous transit of dye, microaneurysms, and macular edema. Carotid angiography showed left common carotid artery occlusion and stenosis of the right internal and external carotid arteries. Carotid artery bypass surgery was not performed. A left fundus fluorescein angiogram repeated in January 1986 showed no change in the appearances noted 2 years previously (Figure 7).

M.M. presented in 1983 when she was aged 56 with attacks of blurred vision and dazzle on exposure to bright light in her left eye. Visual acuities were 6/6 right and left. There were dilated retinal veins, multiple hemorrhages, and a low central retinal artery pressure on the left. A left fundus fluorescein angiogram showed delayed arteriovenous transit of dye, microaneurysms, and macular edema. A week later she developed dysphasia and later a right hemiplegia. An arch
Fluorescein angiography in carotid artery disease

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Discussion

Carotid endarterectomy may directly increase the flow to the ipsilateral eye and increases the pressure in the ophthalmic artery. This operation is technically feasible when the carotid is stenosed but rarely when it is occluded. A report by Neuport and others documented photographically the rapid resolution of venous-stasis retinopathy in 1 case after internal carotid endarterectomy. In our 4 cases treated by endarterectomy of the ipsilateral ICA (Group C), fluorescein angiography provided evidence of improvement in retinal ischemia as early as 1 month after surgery. In all cases microaneurysms were fewer after surgery, and in 2 cases arteriovenous transit time was reduced. These cases support the view that retinal ischemia improves after carotid endarterectomy.

In 1 case (Group B), both endarterectomy and EC-IC bypass surgery were performed. The right internal carotid endarterectomy is unlikely to have produced an improvement in fluorescein angiographic findings in the left eye. It was shown that the left ophthalmic artery filled via anastomosis from the left maxillary artery, so it is likely that the left external carotid endarterectomy rather than the left EC–IC bypass was responsible for the improvement in retinal ischemia.

ST–MCA anastomosis was introduced by Yasargil as a form of EC–IC bypass. The rationale for this operation in the management of ocular ischemia is to increase hemispheric perfusion pressure and collateral blood flow. This operation may be performed when the ipsilateral carotid artery is completely occluded. Kearns and others reported the resolution of venous-stasis retinopathy by ST–MCA anastomosis in 1 case. Edwards and others also reported the reversal of signs of chronic ocular ischemia by ST–MCA anastomosis in 1 case. In our 2 cases treated by this operation alone (Group A), fluorescein angiographic abnormalities were unchanged after surgery. Therefore, our 2 cases do not support the claim that EC–IC bypass reverses retinal ischemia.

In none of the remaining 3 cases treated by medical measures alone (Group D) did fluorescein angiography provide evidence of improvement in retinal ischemia. The period of follow-up ranged from 5 months to 2 years. These case histories suggest that it is unlikely that retinal ischemia due to occlusive carotid disease improves spontaneously.

The improvement in the microcirculation of the retina occurring after carotid endarterectomy may be matched by similar changes in the brain. Whether these changes are associated with a reduction in the risk of stroke or death in these patients was the subject of a large controlled clinical trial in 1970 comparing a group treated by endarterectomy with a group treated by medical means alone. Although surgery appeared to improve survival and stroke rate, this advantage was offset by mortality and morbidity in the perioperative period. When this was taken into account there was no significant difference between the 2 groups. The result of a further international randomized trial of carotid endarterectomy vs. medical treatment is awaited with interest. Although we did not observe an improvement in the microcirculation of the retina after EC–IC bypass, study of the cerebral circulation reactivity to CO₂ has shown an improvement after this procedure. A recent randomized controlled clinical trial of EC–IC bypass has shown no benefit in the risk of stroke or death for the patients treated surgically, even in patients with extensive or bilateral extracranial occlusion.

The ready availability of the retinal circulation and its sensitivity to minor subclinical changes in perfusion pressure make fluorescein angiography a safe and convenient method of assessing any improvement in retinal perfusion that may follow surgery. Our results suggest that carotid endarterectomy is a more effective procedure than EC–IC bypass in improving retinal blood flow.
References

KEY WORDS • fluorescein • carotid • endarterectomy
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