Non-Invasive Evaluation of Patients with Extracranial to Intracranial Bypass

ANDREW C. HAYES, PA-C, WILLIAM H. BAKER, M.D., AND O. HOWARD REICHMAN, M.D.

SUMMARY In selected patients with cerebrovascular insufficiency, an extracranial-intracranial bypass is indicated to increase cerebral blood flow. To assess the effect of this operation upon routine non-invasive testing, 15 patients had oculoplethysmography, carotid phonoangiography and Doppler testing. None of those with a preoperative abnormality were changed after surgery, despite angiographically proven anatomic patency. Whereas non-invasive tests may correctly identify severe internal carotid stenosis, use of these modalities in their routine form does not predict extracranial-intracranial bypass patency.

IN 1966 DONAGHY AND YASARGIL reported on microvascular extracranial-intracranial bypass (EC-IC) for distal cerebrovascular lesions. Since then this procedure has been utilized in selected patients to bypass middle cerebral artery lesions, internal carotid siphon stenosis and internal carotid occlusion. In 1977 the Peripheral Vascular Lab at the Loyola University Medical Center began to evaluate a series of EC-IC patients pre- and post-operatively. Specifically, we hoped that routine noninvasive cerebrovascular testing could assess EC-IC bypass patency. The following is a report of our experience.

Methods and Materials

A battery of three non-invasive tests — supraorbital Doppler ultrasound evaluation, carotid phonoangiography, and oculoplethysmography (Kartchner) — were performed on each patient. In our laboratory this multiple modality testing detects 85% of carotids with > 75% stenosis and is normal in 94% of carotids with < 50% stenosis.

In the Doppler evaluation, a pencil probe is placed over the frontal artery. Direction of flow and signal response to sequential compression of the superficial temporal, facial, infraorbital and common carotid arteries is noted. Normally ophthalmic artery flow and hence frontal artery flow is antegrade out of the eye and is not effected by digital compression of the external carotid artery branches. If compression of an external carotid branch (frontal, infraorbital, superficial temporal artery) reduces the audible signal, a functioning collateral is demonstrated. An absence of signal diminution to common carotid compression likewise indicates an abberant source of ipsilateral frontal artery blood flow (i.e. contralateral carotid or vertebral arteries). An abnormal response equates to a > 75% ipsilateral internal or common carotid area stenosis.

Phonography assessment of cervical bruits and oculoplethysmographic evaluation of ocular pulse volume changes as described by Kartchner et al. are
subsequently performed. A systolic cervical bruit that lasts until diastole indicates at least a 50% area stenosis. The absence of a bruit or a shorter bruit may indicate a lesser degree of stenosis (< 50%) or total or near occlusion. The OPG compares the timed volume changes in each eye to each other as well as to an ear. The instrument generates not only a direct tracing from each eye but also a differential tracing [2 (R-L)] designed to diagnose lesser degrees of stenosis. By our criteria a differential shift alone indicates a 50–75% area stenosis. A visible eye pulse delay in respect to an ear or contralateral eye pulse is diagnostic of > 75% carotid stenosis.

Fifteen patients who underwent 16 superficial temporal to middle cerebral artery bypasses were assessed. The vascular lesion was a middle cerebral artery stenosis in two, an internal carotid occlusion in 12, and bilateral siphon stenoses in one patient. In addition to EC-IC bypass, one patient had an ipsilateral common carotid endarterectomy to re-establish external carotid artery blood flow, one had ipsilateral external carotid endarterectomy, one had contralateral internal carotid endarterectomy and one patient had dilatation of fibromuscular dysplasia of the contralateral internal carotid artery. Ten patients were studied both pre and postoperatively. In eight, the lesion was correctly diagnosed preoperatively. In all 10 patients post-operative exams were unchanged (table).

Five patients were tested only after EC-IC bypass. One patient with middle cerebral artery stenosis had normal testing. One patient with bilateral siphon stenosis and bilateral bypass procedures was abnormal on both sides by non-invasive testing. Two patients with total internal carotid occlusion had abnormal tests after bypass. One patient with an occlusion ipsilateral to the EC-IC bypass and a contralateral 90% internal carotid stenosis had normal testing bilaterally both after bypass and subsequent contralateral endarterectomy. Compression of the left superficial temporal artery (the donor artery) did not alter test results.

Follow-up contrast angiography was obtained in 11 patients with anastomotic patency demonstrated uniformly. One patient had an anastomotic stenosis that was not diagnosed by these techniques (he was also normal pre-operatively). These angiographic results are a reflection of our usual 96% patency rate.

**Discussion**

Ausman and Diaz assessed 66 EC-IC bypass procedures in 55 patients with a directional Doppler and predicted patency as seen on postoperative angiography. Patency was assumed if the extracranial arterial flow could be detected and followed to the cranial defect, and was diminished by digital compression of the donor artery. The authors then performed follow-up angiography.

**Table: Summary of Patient Testing**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Pre-op angio</th>
<th>Pre-op CV exam</th>
<th>Operation</th>
<th>Post-op angio</th>
<th>Post-op CV exam</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>Rt. ICA occ</td>
<td>Rt &gt; 75%</td>
<td>Rt EC-IC</td>
<td>Patent EC-IC</td>
<td>Rt &gt; 75%</td>
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<td>Rt EC-IC</td>
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<td>Rt &gt; 75%</td>
</tr>
<tr>
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<tr>
<td>4</td>
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</tr>
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<tr>
<td>7</td>
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<td>Lt &gt; 75%</td>
<td>Lt CCA TEA and Lt EC-IC</td>
<td>Patent CCA and EC-IC</td>
<td>Lt &gt; 75%</td>
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<td>8</td>
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<td>Patent Lt EC-IC and Rt ICA</td>
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<tr>
<td>9</td>
<td>Rt. ICA occ</td>
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<td>Rt EC-IC</td>
<td>Patent EC-IC</td>
<td>Normal</td>
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<tr>
<td></td>
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<td></td>
<td>with stenosis</td>
<td></td>
<td>Normal</td>
</tr>
<tr>
<td>10</td>
<td>Lt. MCA stem</td>
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<td>Lt EC-IC</td>
<td>Patent EC-IC</td>
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<tr>
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<td>Rt. and Lt. siphon sten</td>
<td>Not done</td>
<td>Lt EC-IC</td>
<td></td>
<td>Normal</td>
</tr>
</tbody>
</table>

**Abbreviations:** occ = occlusion; sten = stenosis; ICA = internal carotid artery; MCA = middle cerebral artery; CCA = common carotid artery; ECA = external carotid artery; FMD = fibromuscular dysplasia; TEA = thromboendarterectomy; EC-IC = extracranial to middle cerebral artery bypass; Lt = left; Rt = right.
the artery at a site proximal to the probe. In 58 of 63 studied anastomoses, the Doppler criteria were met, and the bypass was patent by angiography. Three were thought to be patent by Doppler but angiographically only the superficial temporal artery was seen to be patent. However, in two of these patients, a subsequent arteriogram showed EC-IC bypass patency. One patient had equivocal Doppler findings and was found to be angiographically occluded. Later testing became positive for patency and a repeat angiogram confirmed that the anastomosis was indeed patent. Finally, one case diagnosed by Doppler to be patent was occluded. In all cases, direction of flow in the donor artery was toward the cranial defect.

Whereas the above cited technique evaluates direction of flow in the donor artery, the techniques evaluated in our study judge patterns of collateral circulation and the equality of timed eye volume changes. The supraorbital Doppler evaluation of external-supraorbital (or frontal)-ophthalmic-distal internal carotid artery collateral flow is abnormal when the pressure in the external carotid branches is greater than the ophthalmic artery pressure. For this test to revert to normal, the ophthalmic artery pressure must be increased to greater than external carotid branch pressures. The OPG is abnormal when the timed volume changes of the eyes are dysynchronous, i.e. one globe increases its volume at a slower rate than the normal globe. For this test to revert to normal, the reduced volume flow in the abnormal ophthalmic artery must be increased to match the normal contralateral side.

Stephens measured intra-arterial pressures of the donor superficial temporal artery in recipient middle cerebral artery branch. He found an average drop of 35 mm Hg in the donor artery versus systemic pressure and a further average 45 mm Hg pressure gradient between the donor and recipient artery. That is, if a patient had a systemic systolic pressure of 130 mm Hg, then the superficial temporal artery pressure would be 105 mm Hg and the middle cerebral artery pressure would be 60 mm Hg. These values may be erroneously low due to manipulation induced spasm. If intervening stenotic lesions between the site of anastomosis and the ophthalmic artery are present, then larger gradients may exist.

Still, despite these measured gradients, ophthalmic artery pressures do increase following EC-IC bypass. Gee, et al. reported on 35 patients after a variety of reconstructions using pneumo-oculoplethysmography as his pre- and post-operative testing technique. This method measures an ophthalmic artery pressure which is compared to the contralateral ophthalmic artery pressure as well as the systemic systolic pressure. In 17 patients, carotid endarterectomy was performed for internal carotid stenosis contralateral to internal carotid artery occlusion. In all patients, post-operative pressures were raised ipsilaterally and in 76% contralaterally. Eighteen patients underwent EC-IC bypass, six bilaterally. Seventeen had post-operative ophthalmic artery pressure elevations. Four occurred unilaterally, eight occurred definitely bilaterally, and five occurred possibly bilaterally. Carney also reported that ipsilateral ophthalmic artery pressure as measured by the OPG-Gee increased after EC-IC bypass. Furthermore, digital compression of the superficial temporal artery decreased these readings demonstrating anastomotic patency.

These above changes are obviously not of the magnitude required to cause reversion of abnormal collateral flow patterns back to normal, as our supraorbital Doppler examinations continued to show abnormal patterns of flow despite patent EC-IC bypasses. Kartchner has reported that a visible eye pulse delay correlates with a greater than 40% reduction of internal carotid artery flow. Normally internal carotid artery flow is approximately 250 cc/minute. A greater than 40% reduction would be less than 150 cc/minute. Reichman using cineangioarteriography to estimate EC-IC graft flow in 20 patients demonstrated that the average flow was 86 cc/minute (range 39-240 cc/minute) with only 7 patients having flow greater than 100 cc/minute. These average flow rates are obviously not enough to overcome testing abnormalities as measured by the fluid filled OPG in our noninvasive vascular laboratory. Therefore, a persistently abnormal OPG cannot be taken as evidence of graft occlusion.

This report should not be interpreted as a condemnation of EC-IC bypass grafting for selective patients with cerebrovascular insufficiency. Operative morbidity of permanent neurologic deficit of 1% with 2.7% operative mortality is comparable to statistics for carotid endarterectomy. Graft patency is high, approaching 97%. Improvement in regional cerebral blood flow using Xenon-133 techniques has been demonstrated after anastomosis. This increased blood flow results not only in improved local perfusion but also protects the watershed area from emboli arising from established proximal atherosclerotic lesions.

This report does establish that conventional supraorbital Doppler evaluation and the fluid filled OPG (Kartchner) are insensitive to the quantity of change in pressure and flow created by a functioning EC-IC bypass, and thus these noninvasive procedures cannot be used to assess the patency of these anastomoses.

References

Cerebral Edema Following Experimental Subarachnoid Hemorrhage

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SUMMARY The development of cerebral edema after experimental subarachnoid hemorrhage (SAH) was studied in cats by determining regional brain tissue water content with the microgravimetric technique as well as the drying-weighing method. SAH was induced by withdrawing needles previously pierced into one or both infraclinoid internal carotid arteries through a unilateral transorbital approach. Serial determinations of regional cerebral blood flow (rCBF) by labelled microspheres, and monitorings of vital signs such as intracranial pressure (ICP), blood pressure and EEG were carried out up to 24 h after SAH. Animals could be classified into three grades according to the severity of SAH. In grade I, the increase of ICP was transient and minor. In grade II, ICP increased up to 200 mm Hg with a marked reduction of rCBF below 20% of control in cerebral hemispheres. Following subsequent reduction of ICP, rCBF increased over control, indicating reactive hyperemia. Thereafter, a great reduction of rCBF was again observed. In grade III, rCBF was sustained at essentially zero flow with the presence of continuously increased ICP above 100 mm Hg. Cerebral edema was observed particularly in the parasagittal water-shed areas of all grade II animals. It is concluded that cerebral edema complicating SAH is caused by the combination of an initially induced global cerebral ischemia and the subsequent recovery of cerebral circulation. Post SAH hypertension is another factor to exacerbate the development of cerebral edema.

The Pathophysiology of subarachnoid hemorrhage (SAH) has been widely studied in both its clinical and experimental aspects, being related to the cerebral circulation,1-4 neuronal function,5-7 intracranial pressure (ICP),9,10 and cerebral vasospasm.5,12 On the other hand, the development of cerebral edema in SAH has rarely been investigated.13 However, it is becoming more important to understand cerebral edema as a complication in patients with SAH, because an increasing number of patients with ruptured cerebral aneurysms are undergoing early surgery.14,15

Some difficulties are still involved in investigating cerebral edema after experimental SAH. First, to simulate human SAH due to a ruptured cerebral aneurysm, the following three conditions are required: 1: arterial wall injury; 2: acute increase of ICP; 3: a sufficient amount of subarachnoid blood clots. At the time of aneurysm surgery in the very acute stage of SAH, widely spread subarachnoid blood clots usually observed to cover the cerebral convexity may contribute to the alterations of cerebral microcirculation. In experimental SAH usually induced by cisternal blood injection or arterial puncture, such a condition cannot be easily obtained warranting a more advanced method. Secondly, changes of brain water content must be precisely assessed for the quantitative determination of brain edema. For this purpose, the recently developed gravimetric technique may be more advantageous for determining specific gravity (SG) of very small tissue fragments than the conventional drying-weighing pro-
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