Selective cerebral revascularization as an adjunct in the treatment of giant anterior circulation aneurysms

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Cerebral revascularization, an indispensable component of neurovascular surgery, has been performed in the treatment of cranial base tumors, complex cerebral aneurysms, and occlusive cerebrovascular disease. The goal of a revascularization procedure is to augment blood flow distally. It can therefore be used as an adjunctive measure in the treatment of complex neurosurgical disease processes that require parent artery sacrifice for definitive treatment. In the treatment of giant anterior circulation aneurysms, for instance, a cerebral revascularization procedure may be considered in patients in whom the collateral circulation is marginal and in whom lesions may be treated either using a Hunterian-based strategy or clip-assisted reconstruction requiring a prolonged period of temporary occlusion. To date, there is no entirely effective method known to produce long-term tolerance to carotid artery (CA) sacrifice and, largely for that reason, some neurovascular surgeons advocate universal revascularization. The authors of this report, however, prefer to perform revascularization only in the limited subset of patients in whom preoperative assessment has revealed risk factors for cerebral ischemia due to hypoperfusion. In this paper, the authors introduce their protocol for assessing cerebrovascular reserve capacity, indications for cerebral revascularization in the treatment of complex anterior circulation aneurysms, and discuss their rationale for choosing to practice selective, rather than universal, revascularization.

KEY WORDS • revascularization • giant aneurysm • extracranial–intracranial bypass • balloon occlusion • cerebrovascular reserve capacity • Hunterian

The ideal treatment of a giant intracranial aneurysm is definitive exclusion from the circulation by performing microneurosurgical clip–assisted reconstruction in which all afferent and efferent arterial branches in the aneurysm segment are preserved. Unfortunately, because these lesions very frequently involve both the parent artery and critical neighboring branches, reconstruction is rarely a straightforward task. In fact, for most giant aneurysms, clip reconstruction requires a prolonged period of temporary occlusion. In some cases, usually because of inaccessibility or an unfavorable neck configuration, clip-assisted reconstruction is not feasible; the giant aneurysm must then be treated using a Hunterian strategy. In his considerable experience treating 174 patients who harbored giant intracranial aneurysms, Drake reported that 62% of the lesions were deemed unamenable to placement of a clip; his data underscored the difficulty of direct approaches in the management of these complex pathological entities, even in the best of hands.

It is widely accepted that a subset of patients who are treated with a Hunterian occlusion–based strategy are at risk of suffering clinically significant ipsilateral cerebral hypoperfusion. The same holds true for patients requiring prolonged temporary occlusion for clip reconstruction of the aneurysmal segment. Some neurovascular surgeons have performed prophylactic revascularization in all patients harboring complex aneurysms, whereas others have implemented a selective cerebral revascularization paradigm based on extensive data derived from preoperative testing of cerebrovascular reserve capacity. In the latter of these two schemes, which is our institutional practice, revascularization is considered to be an adjunctive measure only in patients in whom impairment of cerebrovascular reserve is demonstrated. Moreover, based on both their angiographically depicted neurovascular anatomy and results of CRT, the most appropriate bypass conduit is then selected. In this review, which is based on the neurovascular practice of the senior author (H.H.B.), we describe our approach to evaluation and surgical management of patients with giant anterior circulation aneurysms with a particular focus on our usage of adjunctive cerebral revascularization. We describe the

Abbreviations used in this paper: BTO = balloon test occlusion; CA = carotid artery; CBF = cerebral blood flow; CCA = contralateral CA; CRT = cerebrovascular reserve test; CT = computerized tomography; EC–IC = extracranial–intracranial; ECA = external CA; EEG = electroencephalography; ICA = internal CA; MCA = middle cerebral artery; MR = magnetic resonance; SAH = subarachnoid hemorrhage; SPECT = single-photon emission CT.
management of a patient with a giant supraclinoid CA aneurysm to illustrate further our general principles of practice. Finally, our rationale for advocating selective revascularization is discussed.

SELECTIVE CEREBRAL REvascularization

Initial Neuroradiological Evaluation

In all patients who will undergo surgical treatment of a giant intracranial aneurysm, the following neurodiagnostic studies, at the very least, have been performed: non-contrast head CT scanning, diffusion-weighted MR imaging, and four-vessel cerebral angiography. It has become our practice to obtain a three-dimensional CT angiogram in every patient with a giant aneurysm in whom some form of surgical treatment is planned. Computerized tomography angiography plays a complementary role to conventional cerebral angiography in the evaluation of aneurysms, primarily by allowing the easy visualization of intraaneurysm and neck thrombosis or calcification, as well as depicting the relationship of the lesion to neighboring arteries and important cranial base structures such as the clinoid process. Diffusion-weighted MR imaging is an extremely sensitive modality by which to detect subtle ischemic changes that may predict diminished tolerance to extended arterial occlusion.

Cerebrovascular Reserve Testing

Assessment of cerebrovascular reserve, which is composed of four principal elements-neuroclinical, hemodynamic, neurophysiological, and provocative-is used in an effort to predict tolerance to parent artery sacrifice. The cornerstone of the CRT is the BTO, which has been widely described in the literature and involves monitoring the awake patient in whom an artery is temporarily occluded by using nondetachable endovascular balloons. Cerebrovascular reserve testing is routinely performed in all our patients with unruptured giant intracranial aneurysms prior to any surgical treatment. In general, however, CRT should be performed in patients harboring aneurysms for which a deconstructive strategy is anticipated or, based on the assessment of an experienced neurovascular surgeon, a prolonged period of temporary occlusion will be required for clip-assisted reconstruction. After the CRT, the case is then stratified based on the outcome. The CRT results not only allow us to predict tolerance to parent artery sacrifice with a high degree of certainty, but they also enable us to select the most appropriate type of bypass conduit should revascularization be necessary as an adjunctive measure.

Investigators in the Cooperative Study of Intracranial Aneurysms and Subarachnoid Hemorrhage reported that the rates of ischemic cerebral-related complications attributed to abrupt CA occlusion were 49 and 28% for ICA and CCA sacrifice, respectively. This wide discrepancy in values has been attributed to several factors. First, in cases requiring CCA sacrifice, because the ICA frequently remains fully patent, clinically significant thromboembolic phenomena affecting the ipsilateral hemisphere are less likely to occur. Usually retrograde flow through the ipsilateral ECA allows substantial orthograde ICA flow, thereby limiting ischemic sequelae. Moreover, the dysautoregulatory state associated with SAH limits the capacity of regional cerebral circulation vasodilatation to maintain adequate perfusion during CA occlusion. In cases in which the BTO is clinically tolerated-the neuroclinical arm of the CRT-the rate of cerebral ischemia following CA sacrifice decreases to somewhere between 5 and 20%. With the goal of improving the predictive ability of the BTO, quantitative hemodynamic assessment in the form of radionuclide SPECT imaging—either using 99mTc HMPAO or 133Xe—has been added to the CRT protocol. Augmentation of the CRT schema with these CBF studies has met with some success, but not overwhelming, at improving our ability to predict tolerance to parent artery sacrifice. In a study that combined neuroclinical assessment during BTO and nonquantitative 99mTc HMPAO SPECT, Peterman, et al. found that two of 17 patients in whom BTO was tolerated asymmetrical cerebral perfusion was demonstrated on radionuclide CBF studies. One of these two patients is currently undergoing follow-up study, whereas significant EEG slowing was demonstrated in the other during temporary arterial occlusion. Conversely, one of the 15 patients in their series in whom BTO was tolerated and symmetrical perfusion was revealed on 99mTc-HMPAO-SPECT imaging experienced a watershed infarction during a period of intraoperative hypotension. Results obtained in this study, and others just like it, clearly underscore the multifactorial complexity of arterial blood flow to the brain and its potential vulnerability to a variation in systemic hemodynamics, particularly hypotension, volume depletion, and arrhythmias. As a consequence of these factors, the CRT now commonly incorporates periods of hypotension—provocative testing—that are iatrogenically induced using either sodium nitroprusside or labetalol to achieve a blood pressure roughly two thirds of mean arterial pressure. In their seminal study on this topic, Standard and colleagues retrospectively reviewed 47 cases of BTO performed at the State University of New York at Buffalo. Of the 19 patients who underwent iatrogenic CA sacrifice after a clinically tolerated BTO involving hypotensive challenge, only one experienced a delayed ischemic insult—a minor cerebral infarction in the distribution of the distal posterior division of the MCA, certainly due to thromboembolism. Of interest, the surgical procedure in this patient involved Hunterian ligation, which is thought to be associated with a higher rate of thromboembolic complications than endovascular CA occlusion due to, among other things, formation of thrombus in CA “dead space.”

As previously mentioned, our institutional protocol for CRT assessment involves the following in four dimensions: neuroclinical, hemodynamic, neurophysiological, and provocative. Neuroclinical evaluation, the cornerstone of the CRT, involves the continual monitoring of the patient for signs of cerebral ischemia for a period of approximately 20 minutes during which a nondetachable balloon is inflated in the parent artery and the patient is kept in the normotensive range. The most rudimentary hemodynamic evaluation involves simply observing the cross-flow of contrast material to the ipsilateral hemisphere via the communicating arteries as well as the lep-
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tomeningeal inflow and venous outflow in the involved territory. This, in our experience, has minimal value for predicting tolerance to parent artery sacrifice. A more useful hemodynamic assessment involves the aforementioned radionuclide CBF studies. Neurophysiological monitoring is accomplished very easily using EEG during the BTO. This adjunct confers another level of sensitivity to the evaluation in that, because it can potentially show changes due to ischemia in watershed zones, for instance, it can detect perfusion problems not evidenced by focal neurological deficits. The fourth dimension of the CRT as it is performed at our institution is provocative testing.

After patients undergo the CRT, we then stratify them into one of the following three categories based on their results: Group 1, passed in all four dimensions of testing; Group 2, passed neuroclinically but failed in another dimension; and Group 3, failed neuroclinically.

Group 1 patients do not undergo revascularization and their aneurysm may be treated using a Hunterian strategy or prolonged temporary occlusion if necessary. If a Hunterian procedure is required or prolonged temporary occlusion is necessary for clip-assisted reconstruction, Group 2 and 3 patients undergo a low-flow arterial bypass or a high-flow venous interpositional bypass procedure, respectively. An important point to note is that rarely is stratification into these groups cut and dry. Often times, patients who fall somewhere between two groups are placed into the group of criteria for which their CRT results most closely resemble, a concept that will be described in the proceeding case illustration.

CASE ILLUSTRATION

Giant Supraclinoid Carotid Aneurysm

This 49-year-old Asian woman without a significant medical history except for right-sided retroorbital pain was admitted to Northwestern Memorial Hospital with an incidentally discovered, partially thrombosed, giant supraclinoid CA aneurysm. On admission, the patient was alert and oriented in all three dimensions, and her neurological examination was unremarkable. Cerebral angiography demonstrated that the aneurysm imposed significant mass effect on both the A<sub>1</sub> and M<sub>1</sub> segments (Fig. 1). Our therapeutic plan was to attempt definitive exclusion of the lesion from the circulation by performing clip-assisted reconstruction with the understanding that, because of the complexity of the lesion and the presence of a hypoplastic efferent CA, a prolonged period of temporary CA occlusion or even intraoperative loss of the CA was possible. Cerebrovascular reserve testing showed very minimal left upper-extremity drift in conjunction with mild slowing and amplitude loss in the right anterior midtemporal leads on the EEG just after the balloon was inflated in the cerebral right ICA. Both the clinical and neurophysiological deficits were transient and resolved in roughly 1 to 2 minutes. For the remaining 20 minutes of normotensive BTO, she was entirely neuroclinically intact and the EEG values were normal. When a hypotensive challenge was provoked, there were no signs of neurological impairment for the entire 5-minute period.

Because the BTO was essentially tolerated neuroclinically although there was minor evidence of transient impairment, the patient was considered to best belong to the Group 2 classification. As such, she was taken to the operating room at which point a superficial temporal artery–MCA bypass was performed. Immediately after patency of the low-flow bypass was verified angiographically (Fig. 2), clip-assisted reconstruction was conducted with the aid of retrograde suction decompression as described by Batjer and Samson.4

Routine intraoperative cerebral angiography demonstrated exclusion of the lesion from the intracranial circulation and satisfactory clip placement (Fig. 3). Of minor note, a transient third nerve palsy developed postoperatively. On routine postoperative cerebral angiography performed 5 days postoperatively, residual filling of the aneurysm sac was surprisingly demonstrated (Fig. 4). It was our assumption that a small amount of intraaneurysm thrombus was forced posteriorly by the clip closure and must have temporarily obliterated a tiny remaining neck deep to the blades. Ultimately, natural thrombolysis must have restored a channel of communication between the aneurysm remnant and the intracranial circulation. We considered endovascular coiling of the tiny remaining neck, but the sac had been widely opened for thrombus decompression at time of the first procedure, and we were concerned the coils might simply pass into the subarachnoid space. As a result, the patient was returned to the operating room at which point the clips were reapplied to the aneurysm neck to secure it and again exclude it from the circulation. A final postoperative cerebral angiogram

![Image 300x445 to 540x715]

Fig. 1. Preoperative neurodiagnostic studies. Upper Left: Axial MR image of the lesion. Upper Right: A CT angiogram of the lesion demonstrating the complexity of its neck. Lower Left and Right: Anteroposterior (lower left) and lateral (lower right) cerebral angiograms obtained after right ICA injection, demonstrating a giant aneurysm arising from the supraclinoid CA.
(Fig. 5) revealed complete exclusion of the aneurysm from the circulation with preservation of all afferent and efferent vasculature. She has made a full recovery.

DISCUSSION

Giant intracranial aneurysms, first defined by Morley and Barr as being greater than 25 mm in maximum diameter, are uncommon clinical entities, although uniformly formidable from a technical standpoint. Excellent reviews on the natural history and epidemiology of these lesions have been published. Not dissimilar to their nongiant counterparts, giant aneurysms most frequently present clinically with either SAH, mass effect, or cerebral ischemia. Of note, giant aneurysms presenting with massive cerebral edema have also been reported. Several investigators have reported a dismal natural history of intradural giant aneurysms starting from the moment they become symptomatic in any manner, with most patients either dead or severely debilitated within only a few years. Because of this, some form of intervention is almost always warranted to cure or alter the natural history of this life-threatening condition.

The therapeutic approach to giant CA aneurysms is dichotomously divided into reconstructive and deconstructive strategies. Reconstructive strategies, accomplished by either clip placement or endovascular insertion of detachable coils, are preferred because they preserve cerebral hemodynamics. Because giant aneurysms frequently possess wide-based necks, endovascular aneurysmal thrombosis has produced less than optimal success rates. Deconstructive strategies, which principally involve surgical or endovascular Hunterian proximal arterial occlusion, are comparatively less desirable than direct clip-assisted reconstruction because they alter the postoperative cerebral hemodynamics and allow for potential new aneurysms to develop in the collateral system. Nevertheless, some giant aneurysms, either before or after intracranial exploration, prove unamenable to clip-assisted reconstruction and therefore must be, by default, treated by deconstructive measures.

Shortly after its introduction in 1967 by Yaşargil and Donagh and Yaşargil, cerebral revascularization became extensively described in the neurosurgical literature as an adjunctive measure used in the treatment of complex aneurysms and cranial base tumors. It has been traditionally undertaken using an EC–IC bypass; however, technically sophisticated high-flow IC–IC venous interposition constructs have also been described. As previously described, in an effort to prevent cerebral ischemia when a Hunterian strategy is applied to treat a giant aneurysm or when prolonged temporary occlusion is anticipated, some neurovascular surgeons perform a prophylactic bypass in every patient, eliminating the necessity to determine cerebrovascular reserve capacity. The argument proposed by advocates of universal cerebral revascularization has focused on the delayed risk of cerebral ischemia, which they have asserted, is reduced by an EC–IC bypass following proximal parent arterial sacrifice. Additional reasons that universal revascularization is contented to be superior include a reported decreased rate of either de novo aneurysm formation or undetected aneurysm enlargement contralateral to a sacrificed CA and avoidance of the inherent risks associated with the BTO. By contrast, as proponents of a selective revascularization paradigm, we base our defense of this strategy on four central themes on which we will expound. 1) A small but significant inherent risk is associated with revascularization even in the best of hands. 2) Most cerebral ischemia following iatrogenic CA sacrifice is caused by embolic phenomena that have not been shown to be prevented or even abated after revascularization. 3) Both de novo aneurysm formation or existing aneurysm expansion, and even subsequent hemorrhage, may occur despite a bypass. 4) Balloon test occlusion-related risks have been exaggerated and, moreover, its predictive
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Fig. 4. Postoperative right ICA angiogram obtained after the first operation demonstrating residual filling of the aneurysm neck. Although there is smooth narrowing of the A1 and M1 segments due to mass effect from the aneurysm, it is considerably diminished compared with the preoperative studies.

Fig. 5. Oblique (left) and lateral (right) right ICA angiograms obtained after the second operation at which time the clips were repositioned to exclude the aneurysm from the circulation. Of note, the M1 segment of the right MCA is severely stenotic and there is delayed transit time into the MCA territory, a finding consistent with cerebral vasospasm.

complicated their postoperative course. Its development was presumably related to the anticoagulation or antiplatelet therapy necessary to maintain patency of the graft. An additional point of contention related to surgery-induced complications is the diminished experience younger-generation neurosurgeons will have with performing the EC–IC bypass. Because of the unfavorable results reported by the EC/IC Study Group in 1985,12 cerebral revascularization has been largely reserved for the treatment of complex aneurysms, trauma, and cranial base tumors. This considerable reduction in the number of EC–IC bypasses performed by neurosurgeons today stands to reduce significantly the opportunity for the newer generation of neurovascular and cranial base surgeons to cultivate the technical skills needed to achieve excellent results. The overall complication rate of 10% reported by Lawton and colleagues, in light of this, should be considered a floor value, not an expected norm.

A second argument against universal revascularization relates to the incidence and cause of cerebral ischemic events following the use of Hunterian occlusion. Cerebral ischemia following parent artery sacrifice can be classified dichotomously with respect to time of occurrence—immediate and delayed. A second, and more meaningful, manner by which to classify postocclusive ischemia is based on pathophysiological mechanism—thromboembolism compared with cerebral hypoperfusion. The earlier neurosurgical literature on CA occlusion in the treatment of intracranial aneurysms indicates that ischemic complications occurred in roughly 25% of patients. Of these, many were likely due to thromboembolic phenomena or cerebral vasospasm, an entity not widely recognized at the time. Certainly some cases of ischemia were due to a low-flow state that could be attributed to insufficient collateral circulation in the occluded arterial territory.

The authors of extensive reviews have determined that most ischemic events during or after therapeutic CA occlusion to promote aneurysmal thrombosis were thromboembolic in nature. For this reason, patients who, based on the CRT are classified as having Group 1 findings and
should therefore tolerate CA occlusion due to their adequate collateral circulation, are still at risk of stroke because of distal embolization from the occluded CA segment. Despite this information, proponents of universal revascularization claim that an EC–IC bypass performed in patients undergoing parent artery sacrifice will significantly reduce the incidence of postocclusive stroke. Certainly a subset of postocclusive ischemia—that related to cerebral hypoperfusion in patients with marginal collateral circulation—will be alleviated by an EC–IC bypass that will augment CBF. Not only is there no evidence to support a reduced incidence of ischemic stroke in patients with known adequate collateral circulation undergoing parent artery sacrifice and who have undergone revascularization, but because the principal mechanism of ischemia is thromboembolic there is no intuitive rationale either.

A study frequently quoted by advocates of universal revascularization is the retrospective review of 57 consecutive patients in whom intracranial aneurysms were treated with CA ligation. In this study Roski and colleagues, after a mean follow-up period of 12.5 years, reported an annual risk of ischemia-related neurological deficits, either transient or permanent, to be approximately 2.6%. Significantly, 48 of the 57 patients presented with SAH. There is little doubt in our minds that many of these patients, reported to have suffered an ischemic event attributed to CA ligation, in fact, suffered vasospasm-induced cerebral ischemia, a major cause of delayed neurovascular insult which, at the time of this publication, was not widely appreciated by the neurosurgical community. Because the principal mechanism of postocclusive cerebral ischemia is distal thromboembolism due to intravascular CA “dead space” or the aneurysm itself, periprocedural anticoagulation therapy, intracranial trapping, and endovascular release of detachable balloons at or proximal to the aneurysm neck are viable strategies by which to reduce the incidence of delayed stroke in patients undergoing a Hunterian occlusion and with confirmed adequate collateral circulation to sustain normal neuronal function.

A third point of contention in the debate between selective and universal revascularization relates to the incidence of de novo aneurysm formation, existing aneurysm expansion, and subsequent SAH in patients who have undergone CA sacrifice. Proponents of universal revascularization have asserted that the bypass-induced restoration of blood flow would minimize the hemodynamic changes occurring after parent artery sacrifice, which lead to de novo aneurysm formation and existing aneurysm expansion and rupture. Intuitively, there is no doubt that the greatly increased hemodynamic stress imposed on the contralateral CA tree after application of a Hunterian strategy to treat an aneurysm will yield to a higher incidence of aneurysm growth and subsequent rupture.

What makes us think then that ipsilateral cerebral revascularization will attenuate these risks? Certainly a high-flow interposition saphenous vein graft causing reversal of flow in the MCA territory appears just as likely to cause the growth and rupture of aneurysms. In the two-stage treatment of a giant basilar caput aneurysm published by Heros and Ameri, Heros initially created an interpositional saphenous vein graft—assisted bypass to the P segment; the plan was to occlude the proximal basilar artery by using a Drake tourniquet the following day. Before the basilar artery could be sacrificed, the aneurysm ruptured and the patient suffered a massive, fatal aneurysmal SAH. This case clearly demonstrates that an EC–IC bypass can alter the cerebral hemodynamics in such a manner as to cause rupture of an aneurysm. One should also infer from this report that it is likely, because of the hemodynamic stress imposed by a bypass graft, that revascularization could also lead to aneurysm growth much in the same manner as CA occlusion in the absence of a bypass. Carotid artery occlusion simply imposes heightened hemodynamic stress on the contralateral circulation in an orthograde manner whereas the typical EC–IC bypass frequently results in increased flow in the ipsilateral circulation in a retrograde fashion.

Our fourth, and final, major reason for practicing selective revascularization is that it is our belief the aforementioned risk of the CRT, which is attributable to the BTO, has been exaggerated. Contemporary balloons are softer and less traumatic to tissue than earlier versions. Moreover, the predictive value of the BTO in patients in whom parent artery sacrifice will be tolerated and who will not sustain an ischemic event due to cerebral hypoperfusion is, in our experience, quite good. In the large series of 300 patients who underwent BTO of the cerebrovascular reserve capacity published in 1991, Turr and colleagues found that 3.7% of patients suffered a procedure-related complication, the most severe of which was stroke. In an even larger experience reported by Mathis and coworkers the authors reviewed their experience performing 500 BTO procedures at the University of Pittsburgh between 1985 and 1993. The overall procedure-related complication rate was 3.2%. Of note, however, there was a mere 1.2 and 0.4% incidence of transient and permanent neurological complications, respectively, due to the procedure. Although based solely on anecdotal evidence, it has been our institutional experience and that of others (Heros, RC: personal communication, 2003) that the BTO-related risk of complications is much lower, on the order of 1.5%. Additionally, based on the favorable results of parent artery occlusion using an endovascular detachable balloon strategy, this technique is essentially associated with the same risk as the BTO.

With regard to the efficacy of the BTO in predicting tolerance to parent artery sacrifice in patients with complex CA aneurysms, Drake and colleagues reported ischemic complications in only three of their 114 patients in whom a Hunterian strategy—based procedure was performed in the absence of an adjunctive bypass to augment CBF, all of which were the result of a thromboembolic process. With the continual improvement of balloon-related technology and pericranial pharmacology, it is our belief that both the risks associated with the BTO and endovascular Hunterian arterial occlusion will diminish. Equally impressive are the strides being made to improve the predictive value of a preoperative CRT. In their recently published retrospective account of 29 patients with large or giant CA aneurysms in whom therapeutic occlusion was contemplated, van Rooij and coworkers conducted, in addition to the BTO with both neuroclinical and neurophysiological monitoring, angiography to determine the presence or absence of synchronous venous filling of the two hemispheres to identify patients with adequate collat-
eral circulation that would prevent low-flow ischemia. Of the 17 patients in whom evidence of neuroclinical, neurophysiological, and angiographic tolerance to CA occlusion was demonstrated, all then underwent endovascular CA sacrifice. In a mean follow-up period of 21 months, not one of these 17 patients experienced a delayed cerebral ischemic event. One patient in whom the BT0 was tolerated but asynchronous venous filling was visualized on angiography, experienced a delayed ipsilateral watershed infarction when CA sacrifice was performed urgently because of a rapidly expanding CA bifurcation aneurysm causing progressive compression of the optic apparatus.

CONCLUSIONS

Giant aneurysms of the anterior circulation are complex pathological entities that are both uncommon and technically challenging to treat. Therapy, either reconstructive or deconstructive, should be preceded by extensive testing of cerebrovascular reserve capacity. In patients known to be at risk of cerebral ischemia due to cerebral hypoperfusion, a revascularization procedure must be considered. The modalities by which tolerance to parent artery sacrifice are assessed, although currently imperfect, are improving. It should be understood, however, that these hemodynamically based studies cannot predict which patients will develop thromboembolic injuries after arterial sacrifice even when adequate collateral circulation exists. Nevertheless, it is our practice to undertake revascularization in patients in a selective, rather than universal, manner based on results of their cerebrovascular reserve assessment. It is our expectation that the tool of cerebral revascularization, a critical part of the armamentarium necessary to practice neurovascular surgery, will continue to play a dominant role in the management of complex cerebrovascular disease.

References


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