## High flow extracranial to intracranial vascular bypass procedure for giant aneurysms: indications, surgical technique, complications and outcome

H. C. PATEL and P. J. KIRKPATRICK

Department of Academic Neurosurgery, Addenbrooke's Hospital, University of Cambridge, Cambridge, UK

With 5 Figures

# Contents

Abstract	1
Introduction	2
Surgical technique	7
Cranial exposure	9
Cervical exposure	0
Saphenous vein exposure	1
Pre auricular tunnel	2
Anastamoses	3
Distal anastamosis	3
External carotid anastamosis	4
Closure and post operative care	7
Discussion	7
Comparison of outcomes	7
Choosing the type of graft	8
Long term patency of grafts	9
Ischaemic complications	9
Anticoagulation related morbidity	1
Conclusion	1
References	1

## Abstract

High flow extracranial-intracranial (hfEC-IC) vascular bypass remains an important surgical technique in selected patients. For example, in those with

giant aneurysms where the natural history of the condition is poor, and direct surgical approaches are recognised as excessively hazardous. hfEC–IC also allows for major carotid vessel occlusion in the treatment of skull base tumours which would otherwise be untreatable. We describe the indications, techniques, complications, and outcomes of this procedure in an era where few neurosurgeons are exposed to high volume vascular neurosurgery, and fewer still are trained to perform hfEC–IC. We emphasise the need for a stereo-typed and meticulous technique, highlighting key points at each stage of the operation, to ensure graft survival and minimal chances of morbidity.

Keywords: Giant aneurysm; high flow EC-IC bypass.

### Introduction

The aim of this chapter is to outline the current indications, techniques, complications and outcomes of high flow extracranial–intracranial (hfEC–IC) bypass for extreme cerebral vascular disease.

hfEC–IC is a procedure that results in the formation of a sizable vascular conduit between the extracranial circulation and the intracranial circulation, allowing the opportunity to sacrifice a major intracranial parent vessel. The method continues to be recognised as technically demanding with significant hazards, all the more pertinent given reduced training opportunities in vascular neurosurgery. Despite these changes in neurosurgical training culture, the technique remains important for assisting the small numbers of individuals who present with extreme pathological challenges.

hfEC–IC has been performed since 1953 when Conley, an Ear Nose and throat surgeon used a Saphenous vein graft (SVG) to bypass the cervical internal carotid artery to aid resection of a tumour [6]. Whilst Yasargil *et al.* in 1967 reported on surgical techniques of revascularization in dogs, it was Lougheed *et al.* in 1971 who reported the first a successful "high flow" common carotid artery-to-intracranial carotid SVG bypass for athero-occlusive extracranial carotid disease [18, 38]. However, it was not until 1986 that a sizable clinical series emerged (with surgical technique and outcome) with 77 patients undergoing high flow was reported by Sundt [31]. They reported on successfully applying the technique for SVG bypasses into the M2 segment of the MCA and the P2 segment of the PCA.

The indications for a hfEC–IC can broadly be categorized into those required for carotid sacrifice to aid the complete resection of a neoplastic lesion at the skull base or high cervical region, and those required for cerebrovascular pathologies [28, 37] (see Table 1). The number of patients in which hfEC–IC is indicated remains small (Table 2). Indeed, over a cumulative period of 55 years only 460 hfEC–IC bypasses were reported world wide. Moreover, with improvements in stent technology and use of stereotactic radiosurgery, the potential need for hfEC–IC may decline although there is little evidence of this occurring to date [26].

A wide range of benign and malignant tumours of the skull base and head and neck have been treated with radical excision and bypass. Decision to bypass is made on tumour type, tumour location, tumour growth dynamics, extent of invasion and respectability of tumour. Bypass is rarely performed as a palliative procedure although in cases with a high incidence of complications arising from carotid invasion and destruction bypass has been performed.
Complete acclusion of the caretid with associated
"haemodynamic" tia's from misery perfusion.
Giant aneurysms
<ul> <li>that are inaccessible (cavernous sinus aneurysms)</li> <li>when prolonged temporary clipping during</li> </ul>
aneurysm surgery is anticipated
• in those with in whom surgical or endovascular
treatment is not possible because of;
wide neck,
<ul> <li>Dissecting aneurysms, fusiform aneurysms.</li> </ul>
or aneurysms with branches coming of the neck
may also require consideration of bypass.
<ul> <li>Bypass is also considered when a patient has bilateral giant aneurysms and when bilateral carotid sacrifice may be contemplated.</li> </ul>

**Table 1.** Indications for cerebral revascularisation (tia's-transient ischaemic attacks)

Patient selection is complex since there is no absolute indications for hfEC–IC; decisions are made on a case by case basis with the involvement of a multi-disciplinary team, the expertise and opinions of which may vary between institutions, and indeed within institutions as a function of time taking into account the pathology, physiological age of the patient, and the skill and availability of the surgical team, and access to other options.

One of the most robust indications for hfEC–IC is in the treatment of giant aneurysms. Giant aneurysms comprise 3–5% of all intracranial aneurysms [1]. The majority present with symptoms associated with mass effect, usually visual failure, cranial nerve palsies, seizures or hemipariesis [1, 7]. Drake reported that 35% of patients present with a subarachnoid haemorrhage, and some patients will have symptoms of transient ischaemic attacks from aneurysm associated thromboembolic disease [7].

lable z. Jun	Indry or	DUILOUT	ies reported by surgical tea	אווויא הפווטוווווט בר-ור מאמי	SSP	
Study	Study period	Patient no.	Indication	Bypass type	Graft patency	Complications
Regli <i>et al.</i> [26]	1979– 1992	202	Aneurysm (37%), Carotid occlusive disease (63%)	High flow (SVG) ICA-ICA ICA/ECA/CCA/M2 ICA/ECA/CCA-P2	86% patency at 1 year; 82% at 5 years and 73% at 13 vears	15% overall mortality from graft related occlusion causing infarction.
Hacein-Bey <i>et al.</i> [10]	1992– 1997	6	ICA aneurysms	High flow and low flow (SVG and STA) ECA-M2	89% patency	11% neurological morbidity
Houkin <i>et al.</i> [13]	1989– 1998	43	ICA anerysms	High flow (radial artery) ECA-M2	95%	4% ischemic complications
Morgan <i>et al.</i> [22]	1990– 2001	55	Aneurysm (22%), Carotid occlusive disease (28%); neoplastic (30%)	High flow (SVG) ECA-supraclinoid ICA	93%	25% post operative deficits 7% mortality
Jafar <i>et al.</i> [14]	1990– 1999	30	ICA aneurysms	High flow (SVG) ECA/CCA-MCA (84%) ECA/CCA-ICA(10%)	93%	3% Ischemic complications 3% Mortality
Van-Doormaal <i>et al.</i> [35]	1999– 2004	34	Giant ICA aneurysms (with and without SAH)	High flow (SVG) ECA-ICA	100% (17% revision rate)	6% Mortality 12% Ischemic complications 3% technical failures
Mohit <i>et al.</i> [21]	1998– 2005	101	Not specified	High flow (77%) 50% radial grafts; 50% SVG	%66	14% Ischemic complications 4% permanent neurological morbidity: 3% mortality
Kocaeli <i>et al.</i> [15]	2003– 2007	13	Giant aneurysms (76%)	High flow radial graft ECA-M2 70%	100%	23% Ischemic complications
Kirkpatrick <i>et al.</i> (unpublished)	2005- 2007	00	Giant aneurysms (100%)	High flow (SVG) ECA-M2	100% (1 graft revision)	12% anticoagulation related complications 12% graft complications (aneurysm)
SVG Saphenot cerebral arteny	us vein gra 7; P2 post	aft; /CA erior ce	internal carotid artery; CCA erebral artery.	common carotid artery; EC	A external carotid artery; //	CA internal carotid artery; M2 middle

בר ור א Y + 1--:-1 1-+ Ч Ċ

Toblo J

The natural history of giant aneurysms is universally poor. Drake reported a mortality of 68% at 2 years and 80% mortality at 5 years in his series of giant aneurysms treated conservatively [3, 7]. Therefore, a conservative strategy would not be appropriate in the majority of cases, particularly in the younger age groups. Direct surgical treatment of giant aneurysms by means of clipping and/or reconstruction is not possible in up to 50% of patients with anterior circulation aneurysms, and in 70% of patients harbouring posterior circulation abnormalities [8]. Whilst the advent of endovascular therapies has increased the number of such aneurysms amenable to treatment, this technique is also limited in the majority of cases [5]. The success of coiling of a giant aneurysm is dictated by the aneurysm's geometry including neck size and the presence of intra-luminal thrombus. For endovascularly treated aneurysms, there remains a high rate of recanalisation and rerupture even in tightly packed aneurysms. Increasingly with the advances in endovascular techniques, with the use of stents (covered and uncovered) and balloon remodelling to help to keep loops of coils inside the lumen, more aneurysms are amenable to treatment, although this is still fraught with a high rate of recurrence and exposes the patient to the risk of haemorrhage from an untreated lesions, as well as the potential complications from multiple endovascular procedures [5].

In the situation of an inability to surgically or endovascularly treat the aneurysm, because of the poor natural history, the default treatment is usually parent vessel occlusion: Hunterian ligation is a well accepted form of treatment for the treatment of giant aneurysms resulting in aneurysm obliteration in over 95% of patients with a giant aneurysms [8].

Whilst Hunterian ligation is widely accepted as a good management strategy of giant aneurysms, the indications for bypass remain controversial [17, 23]. Not all surgeons agree whether patients should undergo a balloon test occlusion first, with bypass being offered to only those that do not tolerate the trial occlusion [17, 23]. Those advocating a non selective revascularization strategy have argued that the results of bypass are better than the risks of balloon occlusion, that significant ischemia results with parent vessel occlusion without bypass, and that parent vessel sacrifice without bypass leads to an increased risk of aneurysm formation [17].

History reveals that surgical carotid occlusion for the treatment of an aneurysm results in cerebral infarction in upto 40% of patients, even if parent vessel occlusion is graded suggesting that a high proportion of patients cannot tolerate direct carotid occlusion [25]. Because of this high risk of stroke a number of techniques have been developed to ascertain the feasibility of parent vessel sacrifice. The contemporary method for evaluation of stroke risk from parent vessel occlusion is a test balloon occlusion test. This is a test which involves a 20–30 min occlusion of the parent vessel by a deflatable balloon, carried out under radiological guidance, endovascularly, in awake patients that

are clinically monitored. Adjuncts to clinical testing to increase the specificity and sensitivity of cerebrovascular reserve and stroke risk during test balloon occlusion have included measurement of carotid stump pressure, transcranial doppler of MCA flow velocities, SSEP monitoring, Xenon CT and SPECT imaging of cerebral blood flow [2, 4, 19, 29].

The overall complication rate following a test balloon occlusion is reported to be between 0-8% [20]. In the largest series published on the complications of balloon test occlusion (without deployment) the reported overall adverse incident rate was 3.2% [20]. This included 8 patients (1.6%) who developed were asymptomatic, either from a dissection, carotid pseudoaneurysm, or an embolus [20]. Transient neurological deficits were reported in 1.6%, and the overall permanent neurological deficit rate was 0.4%. The complications essentially represent those reported for diagnostic cerebral angiography which all patients undergoing evaluation for giant aneurysm would have to have and therefore does not constitute an additional risk [34].

The risks of cerebral ischaemia or infraction after internal carotid sacrifice after patients have clinically tolerated balloon occlusion are a different proposition. In this group of patients the permanent neurological morbidity rate is reportedly 1.5–4.8% whilst 10–12% of patients experience continuing transient ischemic attacks [9, 16, 30, 32]. It has also been suggested that cerebral ischaemia may not be apparent early, and the risk of delayed ischemia occurring has also been quoted at a rate of 1.4% per year [27]. However most of this risk is observed in the first year of carotid ligation, and it remains unclear whether these risks are from spasm related ischemia or a result of thrombosis and embolisation from the aneurysm, as both are a cause cerebral ischemia in this group of patients [16, 27, 36].

In reality of those patients that have clinically passed a balloon occlusion, only about 10% of patients have problems with delayed cerebral ischemia [4]. Most of this is not acute ischaemia, but that akin to 'misery perfusion' as seen with occlusive carotid disease. Given this, we advocate a selective revascularisation approach, reserving a bypass for those patients that have failed to tolerate 20 min a trial balloon occlusion based on clinical features, as well as angiographic findings of absence of synchronous venous filling and >50% drop in the middle cerebral artery blood flow velocities. All our patients are given acetazolamide pre procedure so that the balloon occlusion is performed in patients with the neurovascular bed 'under stress'. Patients that have developed cerebral ischemia (invariably presenting with transient ischaemic symptoms rather than acute stroke) in a delayed fashion can be re evaluated, undergo cerebral blood flow imaging pre and post acetazolamide, and if necessary hfEC-IC. Further, it is important to appreciate that low blood flow states are dynamic, and may improve with time with the increase in leptomeneingeal collateralization. We emphasize again that decisions need to be made in an individualized manner, because what is appropriate for a patient with for example an unruptured aneurysm may not be appropriate for a patients that present with a subarachnoid haemorrhage.

The other major concern of surgeons favouring a universal revascularization strategy is the formation of denovo aneurysm as a result of the change in flow dynamics (mainly increased flow in the contralateral carotid) after balloon occlusion [17]. These aneurysms have been reported in the territory of the occluded vessel, and also remote from it, and have been reported in up-to 10% of patients [17]. Whilst undoubtedly the flow dynamics of the cerebrovasculature is changed following parent vessel occlusion, most of the literature on denovo aneurysm formation is from the 1960s and early 1970s and may represent missed aneurysms at the time of initial angiography from poorer quality examinations. Further there is no evidence to suggest that an interposition graft is not associated with denovo aneurysm formations because the long term results are not yet available. There are certainly case reports that suggest that hfEC–IC can alter cerebral haemodynamics enough to cause rupture of an unprotected aneurysm [12, 24].

To summarise, we reserve consideration of an hfEC–IC bypass to those patient who have major vascular pathology who attract a poor natural history, and who would otherwise be expected to survive for some years. They all must have failed a test balloon occlusion test judged on clinical and transcranial Doppler and angiographic criteria. Those who have initially tolerated carotid occlusion but then have developed ischaemic symptoms, cerebral hypoperfusion syndrome is confirmed with appropriate cerebral blood flow imaging.

#### Surgical technique (see Table 3)

The key theme to performing a hfEC–IC is the introduction of multiple quality control measures testing each end point of each part of the procedure (see Table 3 for surgical check list used for surgery). The consequence of failure at any point of surgery is at best graft failure, at worst a life threatening complication. Unless these quality insurances are meet, it is our view the patient would be best served avoiding such a procedure.

The operation usually lasts around 4–5 hours and hence is always performed under full general anaesthesia with urinary catheterisation and a central line to ensure optimal fluid management. Rheological agents, such as mannitol, are avoided until the graft is established (vide infra). Sites of proposed incision are discussed with the anaesthetist before induction of anaesthesia to ensure that the necessary central and arterial line placement is not at the site of proposed incisions. All patients receive antibiotic prophylaxis at induction.

All patients undergoing bypass for giant aneurysms are commenced on Aspirin 300 mg a day to minimize the risk of thromboembolic complications and encourage graft survival in the early phase.

Cranial exposure	<ul> <li>Meticulous attention to bleeding and appropriate haemostasis</li> <li>Appropriate, exposure and mobilization of recipient vessel to provide room for formation of anastamosis with temporary clips in situ</li> <li>Absence of perforating vessels on recipient vessel</li> </ul>
Comical averaging	Appropriate caliber of vessel to receive SVG
Cervical exposure	Meticulous attention to naemostasis     Adoquate exposure of ECA
	<ul> <li>Adequate exposure of ECA</li> <li>Oval arteriotomy in ECA when required</li> </ul>
Pre auricular	Create early to allow for the subcutaneous ooze to settle
tunnel	<ul> <li>Generous subcutaneous dissection to ensure no</li> </ul>
	compression of graft
Graft extraction	• Adequate length of graft (20–25 cm)
	<ul> <li>Ligation of side vessels flush with the graft</li> </ul>
	<ul> <li>Graft left in situ until required</li> </ul>
Graft preparation	Valvulotomy and pressure distention to ensure flow
	in both directions
	• Removal of adventia at both ends of the graft
	Ensure no large gratt/recipient vessel mismatch
Anastamosis	Check haemostasis     Arteriotomy to match size of SVC
	Artenotomy to match size of svG     Secure heal and anex of graft first
	<ul> <li>Secure fileer and apex of grant first</li> <li>Ensure subsequent sutures are placed starting as close</li> </ul>
	to the apex and heel of the graft
	<ul> <li>Ensure back wall not compromised with the sutures</li> </ul>
	<ul> <li>Graft allowed to back fill with blood before tunneling</li> </ul>
	<ul> <li>Avoidance of redundant intracranial graft to prevent</li> </ul>
	kinking of graft
Pre closure	• Doppler assessment of the MCA with graft occluded
assessment	Back bleeding through side port with graft occluded just
of vasculature	distal to ECA
	<ul> <li>Back bleeding through side port with graft occluded</li> </ul>
	proximal to MCA
	Closure of side port
	• Doppler assement pre closure of whole length of graft
	Break for 30 min before reassessment of graft
Closure	• Attention to closure to ensure no graft compression from
	replaced bone flap, muscle closure, skin closure
Post operative care	IViaintain normovolaemia, normotension
	<ul> <li>Avoid induversent external graft compression from face masks etc.</li> </ul>
	Regular hand held Donnler surveillance of graft
	<ul> <li>Post operative angiography to ensure graft patency prior</li> </ul>
	to parent vessel occlusion

Table 3. Check list of measures considered to optimize surgical success and graft survival

We have a long history of intraoperative monitoring, but for this procedure no monitoring is routinely used. We feel that the ischemic insult caused by bypass on to the M2 branch of the middle cerebral is minimal, and generally well tolerated. In this setting, we feel that monitoring with somatosensory evoked potentials as advocated by some authors may lead to pressure to rush the anastamosis which is a critical part of the procedure. We accept however that when the bypass is performed onto a more proximal vessel, monitoring would be beneficial particularly to guide the need for 'neuroprotective' adjuncts such as burst suppression.

The head is fixed and held in a Mayfield clamp, and the patient is placed supine with the head turned  $90^{\circ}$  away from the side of surgery. A sandbag is placed under the contralateral shoulder to aid rotation of the head, and the head of the operating table is elevated by  $30^{\circ}$ . A large sandbag is also placed underneath the ipsilateral thigh to aid slight external rotation of the lower limb that facilitates access the Saphenous vein in the leg. As with all neurosurgical procedures, attention to position, and pressure points is routine.

The procedure is lengthy, technically challenging, and requires a meticulous approach at all times of the surgery. Because of this and to minimise the effects of tiredness, we normally have two senior surgeons involved in the surgery. Typically, surgeon A would perform the craniotomy and exposure of vessels in head ( $\sim$ 45–60 min) and in the neck (cervical exposure 30 min). Whilst surgeon A takes a break surgeon B proceeds to harvest and prepare the graft ( $\sim$ 90 min). Surgeon A rested then performs the proximal and distal anastamosis which are the most challenging and tiring part of the procedure.

#### Cranial exposure

Through a standard curved hairline incision, a Pterional craniotomy is fashioned. Following removal of the sphenoid wing, the dura is opened based on the sphenoid wing. The dura is also tacked back over linteen strips at the edge of the wound to minimize ooze from the wound contaminating the field adjacent to the anastamosis. Attention to haemostasis is critical at every step of the procedure from skin to dural opening because of the need to anticoagulate the patient during vessel cross clamping. Dural hitch sutures are not used routinely, as in our experience they do not serve a purpose.

The Sylvian fissure is split widely to identify an M2 branch of the Middle cerebral artery (MCA). The M1 branch is not generally used because of the presence of the perforating arteries. Mohit *et al.* have suggested that in cases where the M1 segment is short, there may be perforating branch of the M2 segment which would make these vessels unsuitable [21]. In this situation he recommended that the communicating segment of the ICA is used as the site of the distal anastamosis. However, this has not been our experience and we have yet to be unable to anastamose to one of the proximal M2 segments.



**Fig. 1.** (A) Graft exposure demonstrating the length of dissection and the number of side branches that need to be ligated. (B) Creation of pre auricular tunnel (C–E) preparation of the graft with destruction of the valves with a disc dissector to allow for flow reversal, and pressure distention to check there are no leaks and that there are no patent valves before implantation. (F, G) Removal of graft adventitia (H) M2 recipient vessel mobilized and prepared for graft implantation

The size of the M2 segment is important and we tend to target the most generous branch. Once the Sylvian fissure is split, and the appropriate M2 branch is identified, the vessel is freed of arachnoid adhesions allowing mobility of the segment. A piece of coloured card (usually derived from the packaging of the 10/0 nylon suture) is softened in saline, shaped and sized, and inserted underneath the site of proposed anastamosis to provide contrast to allow for better visualization of the vessel wall during the anastamosis (Fig. 1). The wound is then filled with warm saline.

#### Cervical exposure

A transverse skin crease incision at the level of the hyoid is made to expose the Carotid bifurcation. The incision is transverse mainly for cosmetic reasons, but can be extended longtitudinally in cases where the carotid bifurcation is difficult to expose. The Sternomastoid muscle is mobilized laterally, to expose the deep cervical fascia and the Carotid sheath both of which need to be opened to expose the Carotid artery. We usually plan for an end to side anastamosis between the graft and external carotid artery (ECA). This requires exposure of the ECA for about 2 cm beyond the bifurcation so that there is enough room to create the bypass after the application of temporary vascular clamps. Avoiding the internal carotid artery (ICA) is clearly important in the context

of intracranial flow arrest during anastamosis, which takes approximately 30–45 min. These patients have already failed a balloon occlusion test, and therefore, this length of time would not be tolerated. If the ICA has to be involved due to anatomical variations and an inadequate ECA, due consideration to a carotid shunt is necessary.

#### Saphenous vein exposure (Fig. 1)

The Saphenous vein consistently runs from anteriomedial to the medial Malleolus where it can invariably be seen or palpated, to the knee where it lies a hands width posteriomedial to the patella. From this point it passes upwards on the medial aspect of the thigh to join the femoral vein in the groin. We harvest the saphenous vein from the ankle to the lower thigh. Pre operatively it is important to check that the patient does not have varicosities of the Saphenous vein, and they have not had any previous surgery that may have compromised the Saphenous vein. We also routinely examine and mark the course of the Saphenous (at the ankle and lower leg) vein pre-operatively with the patient in the standing position. At the time of surgery, the vein is identified first at the ankle before the dissection proceeds toward the knee. Approximately 20-25 cm of vein is exposed and mobilized. During the mobilization procedure all the side branches are ligated with a 4.0 silk suture or secured with titanium haemoclips (Fig. 1). It is important that the ligation is flush with the vessel wall without compromising the lumen. We purposefully do not ligate flush one or two of the larger side branches. These are left so that the side branches can be reopened after the completion of the anastamosis so that flow in both directions of the bypass checked, and air can be vented. They also act as portals to allow graft exploration with various probes if a technical problem has arisen and the graft is found not be conducting appropriately.

Once the graft is prepared, it is left in situ until it is required. Thus the cranial and cervical vessels have been fully exposed and prepared, and haemostasis ensured. At this point it is ligated at both ends and removed. We flush the graft with heparinized saline, and clean the graft of the redundant connective tissue and adventitia at both ends of the graft (Fig. 1). The removal of the adventitia at the proposed cranial end of the graft is cleaned for approximately 2 cm (Fig. 1). This is to minimize the sticking of the sutures to the side walls of the vessel during the time of the anastamosis. The valves in the graft are broken down by passing a series of disc dissectors up and down the graft. Multiple passes of the dissector, oriented in slightly different radial positions, ensures that each valve is sliced in multiple directions to ensure bi-directional and seamless flow. The patency is confirmed by flushing with heparinized saline in both directions. Finally, a probe is passed down the lumen of the graft which is scrolled up along the shaft to allow some degree of mechanical dilation. We cannot stress enough the importance of this manoeuvre, since



**Fig. 2.** These are the images of a 56 year old lady who presented with a cavernous sinus syndrome on the left side. Her contrast enhanced CT scan (A: arrow demarcated aneurysm) and cerebral angiogram (B) revealed a left sided intracavernous aneurysm which was treated with ECA-M2 bypass followed by balloon occlusion 4 days following surgery. The flow into the aneurysm stopped immediately following the balloon occlusion (D), and the aneurysm was noted to remain obliterated on the 3 month contrast enhanced CT scan (C). These images also demonstrate balanced flow in the graft and the internal carotid on a common carotid injection (E–H). This argues against concerns expressed by a number of surgeons who maintain that early/immediate closure of the parent vessel is necessary to encourage flow in the graft

failure of the graft can be a consequence of inadequate graft preparation. Compensation for the valves by reversing the direction of the graft (ankle end for proximal anastamosis) is not something we recommend since the flow dynamics favour a tapering graft which narrows towards the distal anastamosis (Fig. 2). This promotes an acceleration of flow, a factor which may bear importance in maintaining the graft when no pressure gradient exists (i.e. before balloon occlusion of the parent ICA some days later). We also believe that a valved graft will not allow bi-directional flow which may encourage thrombosis within the pockets (flow shadows) of each valve. The smaller ankle end of the graft is also more suitably sized for anastamosis to the M2 branches and vice versa.

Once devalved and dilated, the cranial (ankle) end of the graft is cut obliquely, and fishmouthed in preparation for the distal (cranial) anastamosis (Fig. 1).

#### Pre auricular tunnel

A pair of Roberts forceps is inserted proximal to distal into the pre-auricular tunnel and the lower end of the graft is grasped before the temporary clip is removed. The deflated graft is pulled through with enough tension to ensure that there is no redundant intracranial loop, and that there is no twist or kink in the graft. As a quality check the graft patency can be ensured after this part of the procedure by releasing the distal graft clip and allowing back flow towards the cervical incision. Once this has been observed, the graft is again refilled with heparinised saline as for above (Fig. 1).

## Anastamoses

Just prior to creation of the anastamosis, the patient is given a 3000 unit bolus of Heparin intravenously. We do not routinely perform this procedure under burst suppression or hypothermic conditions, although we do advocate that the blood pressure is maintained at a slightly elevated level (as compared to preoperative state) to optimize flow though the collateral circulation.

# Distal anastamosis (Fig. 3)

We perform the distal anastamosis first allowing maximal mobility of the graft which can be flipped over to allow for better visualization of the posterior wall of the vessel. Following application of proximal and distal temporary clips low pressure clips oval arteriotomy of approximately 5 mm in length (to match the size of the opening of the harvested saphenous vein) in the recipient vessel is created. Sutures at the apex and the heel of the graft are placed to stabilize the



Fig. 3. Intracranial anastamosis

graft against the recipient vessel. The intracranial anastamosis is performed with interrupted 10-0 nylon sutures starting at a point closest to the heel or apex of the graft. The technique preferred for sitting the interrupted sutures is to lay flat all sutures for one side cut to a similar length. Once in place they are tied in sequence starting at the end which most recently received the suture since these ends will overlap earlier sutures. On completion of each knot the ends are trimmed to reduce clutter.

Once one side is complete, the graft is flipped over to access the remaining side. Improved exposure and access of the unsutured side can often be achieved by holding the graft in place by means of additional low pressure clips, or even a fixed retractor blade (Fig. 2). Lumen patency is checked whilst closing the posterior wall to ensure that the lumen has not been compromised by inadvertent stitch placement. The use of a blunt hook inserted into the M2 vessel (both directions) is carried out on a regular basis.

The clamp time to allow for the creation of the anastamosis should ideally be no more than 30 min. However, given that the donor vessel is an M2 branch we believe that collateral flow at this level to be reasonable as assessed by the back flow pressures usually seen. Longer clamp times are therefore likely to be tolerated without too much concern.

After the anastamosis is complete, the temporary clips are removed, and the graft is allowed to 'back bleed' and fill up before a temporary clip is placed at the midpoint of the graft. This implies patency of the distal anastamosis. The site of the anastamosis is also checked to ensure there is no leak that requires a further suture. The site at which leaks are most commonly noted are in the region of the apex or heel of the graft, emphasizing the importance of placing the first sutures as close to the anchoring sutures as possible. Small leakages are tolerated and surgical wrapped around the graft to encourage spontaneous haemostasis. However, a significant pulsatile haemorrhage will usually need a further suture which is best placed with the graft and anastamotic site inflated to prevent inadvertent catching of the opposite vascular walls.

Back bleeding of the graft is important also to perfuse the graft endothelium to minimise the effects of ischaemia. After this, and once reasonable haemostasis is secured, heparinised saline is introduced into the free end of the graft and irrigation provided whilst an aneurysm temporary clip is applied close to the anastamosis site. The rest of the procedure is carried out with the graft full of heparinised saline, although if there are any delays with the proximal anastamosis, the graft is back bleed again to perfuse to minimise the effects of endothelial ischaemia.

#### External Carotid anastamosis

The external carotid anastamosis is performed with interrupted (9.0 nylon) suture in an end to side fashion on the ECA. Artery slings can be manipulated

to allow the donor vessel to be presented more favourably into the wound since the depth of the vessels can present difficulties in carrying out the anastamosis. After application of temporary clips on the proximal and distal ECA, an oval arteriotomy is created. Direct vision into the lumen of the external carotid is an essential part of this section of the operation since intimal flaps can be readily missed. A simple slit incision, in our experience, is insufficient and will often seal resulting in early graft failure. The SVG is cut to size (to minimize excessive kinks and redundant loops) and fish-mouthed as already described. The extracranial anastamosis also proceeds in the same fashion as the described for the intracranial anastamosis with the same check points applicable.

Following completion of the bypass, the temporary clip on the distal graft is released to re-confirm back filling and vent air. If haemostasis is reasonable the external carotid clamps are removed to establish anterograde flow in the graft. Any major leaks can be addressed with interrupted 9/0 sutures.

The availability of side venting ports are invaluable in quality assurance assessing competency of flow and direction (it is not impossible for the graft to cause reverse flow if the proximal anastamosis is compromised). A hand held intraoperative Doppler probe is also essential at this point to assure the surgeon that antegrade flow is present within the graft, and flow is continuous in both systole and diastole.

Though a combination of application of temporary clips proximal or distal to the venting port, and insonation with a handheld Doppler probe, flow and patency of various portions of the graft can be ascertained. Once the operator is satisfied that good quality anterograde flow has been achieved, the patient is given 200 ml of 20% mannitol as a rheological agent to increase graft and cerebral blood flow, and as an anti-oxidant agent to improve the health of the graft which may have been rendered ischaemic during the non-flow stage of the surgery.



**Fig. 4.** These images demonstrate the application of transcranial colour doppler (TCCD) for graft surveillance. (A, B) Demonstrate that the proximal (B) and distal (A) anastamoses are patent, whilst (C) shows that flow is in an antegrade manner; i.e from the carotid to the middle cerebral as measured at the midpoint of the graft



**Fig. 5.** These images demonstrate a typical case from our series of hFEC–IC bypass for giant aneurysms. These images of a 60 year old patient who presented with visual deterioration in the right eye. Her CT head scan (A, B) revealed a right paraclinoid mass which was demonstrated to be a giant carotico-opthalmic aneurysm on a CT angiogram (C). She underwent cerebral angiography which demonstrated good anterior circulation crossflow, but she failed a test balloon occlusion (D, E). She therefore underwent an ECA to M2 bypass, and had a delayed (2 days post bypass) occlusion of her carotid artery endovascularly. The post balloon angiogram demonstrated filling of the right anterior cerebral from the left side as noted preoperatively, and the right middle cerebral territory supplied by the graft (F, G; right common carotid injection; I–K left carotid injection). Following the balloon occlusion, the aneurysm was still noted to be filling (F, G). The patient was reviewed 4 months later when she remained well, the graft (arrows) was patent, and the aneurysm was no longer filling (L). She had no objective change in her visual symptoms

We then recommend observing the graft for a full 30 min and re-checking the end-points of the operation before closing.

#### Closure and post operative care

Once the patency of the vasculature has been confirmed, the side arm of the graft is closed using a silk suture or haemoclip. Closure of all the wounds proceeds in a layered fashion with a repeat attention paid to haemostasis prior to closure because the heparin is not reversed. Attention to closure around the graft is also critical to ensure that the graft is not compromised by replacement of the bone flap, closure of temporalis, or poorly placed sutures in the head and neck. Post operatively external compressions from facemask elastic to securing for an endotracheal tube if necessary also need to be avoided. The patient is managed on a critical care unit with strict instructions to maintain euvolaemia, and normotension. The graft is monitored with a hand held Doppler.

Long term surveillance is performed with a transcranial Doppler ultrasound which assesses flow, direction of flow, and allows visualisation of the entire bypass quickly, and non invasively in the clinic setting (Fig. 4).

Using this technique we have performed 9 hfEC–IC bypass grafts over the last 2 years as part of the treatment for giant intracranial aneurysms. Our median follow-up is currently 12 months. Although, we have had one early graft failure that required revision all grafts were patent and revascularising the hemisphere at the time of balloon occlusion (Fig. 5). We have experienced no ischemic complications resulting from the surgery. One patient in this series developed an anticoagulant related complication (subdural haematoma). This was managed conservatively and there were no resultant long term complications. One patient developed a graft site aneurysm in the preauricular region. This was at the site of a side vessel that was not appropriately ligated flush with the graft vessel and required surgical excision and repair of the graft site.

### Discussion

#### Comparison of outcomes

The results of the experience of a selection of surgeons performing hfEC–IC are summarized in Table 2. Most of these series represents the overall experience of the respective surgical teams and do not stratify results by pathology. This makes overall outcome in patients undergoing hfEC–IC difficult to interpret because of the wide range of pathologies for which hfEC–IC is performed. Even for results published for giant aneurysms, the heterogeneity introduced by whether patients have presented with a subarachnoid haemorrhage or not, makes interpretation of results difficult. Overall outcomes in these situations tend to be driven by the underlying disease process and cannot

entirely be related to surgical morbidity associated with the creation of bypass. These problems in assessing outcome overall is a result of the low number of patients that require bypass.

Outcome however can be assessed on the basis of surgical factors such as graft patency and surgical complications. Graft patency rates are high, and average 93%, and most surgical series agree that early graft failures can be related to intra-operative problems and are due to technical problems [13, 15, 21]. This emphasizes the importance of surgical technique and having in place quality control measures for each section of the operative procedure as emphasized in this article.

### Choosing the type of graft

In general, when major parent vessel occlusion is planned, "low flow" vessels (which typically support flows of 25-30 ml/min) such as the superficial temporal or the occipital artery are not normally sufficient to revascularise a whole hemisphere. In this situation, "higher flow" vessels such as the radial artery (supports flow of between 50 and 150 ml/min) or saphenous vein (100–200 ml/min) are necessary. In addition to flow, graft selection depends on, availability of the graft, size of the recipient vessels, as well as the preference of the surgical team. Both saphenous and radial artery grafts have practical advantages and disadvantages. Radial artery grafts are more difficult to harvest. They are notoriously prone to severe spasm leading to shortening of the harvested vessel. These problems with graft shortening due to spasm can be overcome by the use of judicious application of pressure distention and by soaking in Papaverine [21]. The advantages of working with a radial artery grafts is that there is less of a size mismatch between the graft and the recipient vessel, and therefore there is less turbulent flow at the level of the anastamosis. These features also reportedly make the distal anastamosis easier to perform [13, 15, 21].

The argument for the use of SVG is that it is easier to harvest, supports greater flow, and that grafts are not prone to significant spasm. This is balanced against the disadvantages of the size mismatch, and the presence of a fragile endothelium which contribute in making the distal anastamosis more difficult to perform.

We prefer to use a SVG because of ease of harvesting, and because of ease of handling due to lack of spasm. Our experience of radial artery graft is that it causes significant forearm morbidity. Further getting the vessel back to a suitable length for the bypass requires considerable pressure distension and Papaverine. We are however mindful of the mismatch in the size between the graft and proximal receiving vessel but have had no problems that have required changing the site of anastamosis or graft to date.

#### Long term patency of grafts

Long term patency results are unfortunately limited, although a series from the Mayo clinic using Saphenous grafts with follow-up for 13 years, has suggested a 1-1.5% attrition rate for SVG grafts [26]. In comparing graft survival according to type of graft used, some have argued based on the cardiovascular literature, and experimental literature (porcine venous grafts were more prone to accumulation of low density lipoprotein deposits, and developed accelerated atherosclerosis as compared to arterial grafts) suggests that radial (indeed arterial) grafts may be better, but this is not substantiated in the neurosurgical literature [15]. In the long term, it is unclear as to how long a graft needs to last for, with the logical assumption being that the graft would be required for the life of the patient. One of our cases who having failed a balloon occlusion underwent an hfEC-IC graft for treatment of a giant ophthalmic aneurysm. Four months later, she developed a pre-auricular graft site aneurysm which needed resection and repair with an interposition graft. At the time of surgery, the back pressure in the distal portion of the graft was very high suggesting that the graft may not be required as a result of substantial leptomeningeal collateral development.

Anecdote aside, late graft failure remains an important consideration. The cause of this is thought to be because of a combination of 'arterialisation' of the graft with resultant intimal and smooth muscle hypertrophy, and resultant or coincident accelerated atherosclerosis [26, 15]. It is critical therefore, that post operative care includes medical and life-style strategies to optimize cardiovas-cular health. We routinely ask that the patient refrains from smoking, and that they submit to annual medical surveillance for hypertension and hypercholes-trolaemia.

### Ischaemic complications

One of the great concerns of hfEC–IC is the potential risk of cerebral infarction as a result of clamp time on the distal (intracerebral vasculature) required to perform the distal anastamosis. This clamp time varies according to surgical expertise and familiarity, and is estimated at between 20 and 50 min, and depends on the site of the anastamosis. The risk of cerebral ischemia intuitively is increased when the distal anastamosis is performed on the ICA and may be reflected in the high risk of post operative neurological deficits reported by Morgan *et al.* [22] (14%) and Lawton *et al.* [17] (16%) who performed bypass onto the supraclinoid ICA [17, 22]. This is contrast to those contemporary series including our own that have reported an ischaemia related neurological morbidity of between 0 and 6% with a hfEC–IC onto the middle cerebral branches [14, 21]. That major branch occlusion results in increased ischemic complications is further suggested by the reluctance to bypass onto the M1 because the territory supplied by the M1 perforating arteries is intolerant of ischemic insults [21]. Because of this risk of infarction, we constantly perform our distal bypass onto the M2 to good effect.

More recently, the excimer lazer assisted technique (ELANA) has been pioneered to allow an anastamosis to be performed without parent vessel occlusion [35, 33]. Whilst this technique is a significant aid to performing bypass, it still requires the technical skill and quality control measures necessary to perform bypass because the failure rate of the laser to create a suitable opening is high, and this may necessitate the use of conventional techniques. Initial results whilst encouraging they have not reported a reduced stroke risk as compared to series in which patients underwent bypass without the ELANA technique [14, 17, 21, 22, 35]. However, it has to be noted that the reports are on patients that have had bypass for giant aneurysms some of whom have presented with a subarachnoid haemorrhage.

Early graft occlusion is responsible for a significant proportion of cerebral ischemia related morbidity [35]. This again emphasizes that need for quality control through out the procedure, and mindful of this, a number of surgical teams have come to rely on intraoperative angiography as part of the quality control for the procedure [21]. Indeed this is thought to be one of the major reasons why some authors report graft patency rates of 99-100% [15, 21] compared to the series from Sundt et al. [31] that reported a graft patency rate of 83%. Intraoperative angiography is not widely available, and good graft patency rates can be achieved without intraoperative angiography using techniques as described above. We use a number of measure to check patency before the patient leaves the operating theatre that have been documented above. We feel that the quality of the Doppler signal from the graft particularly hearing flow in both systole and diastole combined with the other measure including rechecking the graft 20-30 min after creation of the anastamosis is a robust enough quality control measure to ensure graft patency. More recently, with the advent of indocyanine green angiography technology, on table "angiography" to assess graft patency has become easier at centres that have this facility.

One of the other important factors with regard to morbidity associated with early graft failure however may be related to the timing of the parent vessel occlusion. The risks of occlusion if a graft fails with early/immediate parent vessel occlusion are high, and in this setting, we would agree that contemporary practice dictates that intraoperative or pre-parent vessel sacrifice angiography to demonstrate graft patency is mandatory. However, in most cases, immediate parent vessel occlusion is not necessary, although there are reports of early aneurysm rupture from change in flow dynamics in the intracranial circulation. Again decisions with regard to this have to be made on a case by case basis, mindful of potential complications, and the available quality control measures. We routinely perform delayed (2–4 days) cerebral angiography to assess the patency and quality of the graft before proceeding to balloon occlusion of the parent vessel. There have also been concerns from some authors that early parent vessel occlusion is necessary to encourage flow the graft. This is not borne out by our experience and as illustrated in Fig. 2, balanced flow is achieved between the graft and the carotid using the technique described above.

### Anticoagulation related morbidity

The final major concern with regard to bypass related morbidity the need to anticoagulate the patient during the procedure. One patient in our series developed an acute subdural post operatively. Lawton et al. reported that 5 out of 61 patients (8%) had a post operative extradural haematoma, and Hecin-Bey et al. (although in a smaller series) reported a anticoagulation complication rate of 33% (this included 2 femoral pseudoaneurysms) [10, 17]. As a result of these complications some authors have advocated routinely reversing the heparin given as part of surgery once the graft is established. In these series, the post operative problems resulting from a bleeding diathesis are significantly lower (0-3%) [14, 21]. Indeed, there are some surgical teams that do not systemically anticoagulate their patients, preferring to use copious amounts of heparinised saline during the operative procedure. The potential for thromboembolic complications in the graft in this setting are higher, and mandates the availability of appropriate quality control measures such as intraoperative angiography, and or intraoperative Doppler. We routinely do not reverse the anticoagulation because of the potential thrombotic complications. This approach necessitates meticulous attention to bleeding from the start of the operation. It is important to point out that most of the bleeding related complications reported in the literature occur remote from the anastamosis site emphasising the importance of haemostasis at each phase of the procedure.

## Conclusion

hfEC–IC bypass to allow parent vessel occlusion is an important treatment for patients with giant intracerebral aneurysms. Surgery for hfEC–IC bypass is technically challenging. However, many authors have performed this surgery with good graft patency rates and low morbidity. We advocate performing this surgery in a stereo-typed and meticulous fashion with multiple quality control checks at each phase of the procedure.

### References

- 1. Anson JA (1995) Epidemiology and natural history. In: Awad I, Barrow D (eds) Giant intracranial aneurysms. Thieme, Stuttgart
- Barker DW, Jungreis CA, Horton JA, Pentheny S, Lemley T (1993) Balloon test occlusion of the internal carotid artery: change in stump pressure over 15 minutes and its correlation with xenon CT cerebral blood flow. Am J Neuroradiol 14: 587–90

- 3. Barrow DL, Alleyne C (1995) Natural history of giant intracranial aneurysms and indications for intervention. Clin Neurosurg 42: 214–44
- Carlson A, Yonas H (2006) Monitoring carotid interventions with Xenon CT. In: Gillard J, Graves M, Hatsukami T, Yuan C (eds) Carotid disease: role of imaging in diagnosis and management. Cambridge University Press, Cambridge
- 5. Choi IS, David C (2003) Giant intracranial aneurysms: development, clinical presentation and treatment. Eur J Radiol 46: 178–94
- Conley JJ (1953) Free autogenous vevin graft to the internal and common carotid arteries in the treatment of tumours in the neck. Ann Surg 137: 205–14
- Drake CG (1979) Giant intracranial aneurysms: experience with surgical treatment in 174 patients. Clin Neurosurg 26: 12–95
- Drake CG, Peerless SJ, Ferguson GG (1994) Hunterian proximal arterial occlusion for giant aneurysms of the carotid circulation. J Neurosurg 81: 656–65
- Fox AJ, Vinuela F, Pelz DM, Peerless SJ, Ferguson GG, Drake CG, et al. (1987) Use of detachable balloons for proximal artery occlusion in the treatment of unclippable cerebral aneurysms. J Neurosurg 66: 40–46
- Hacein-Bey L, Connolly ES Jr, Mayer SA, Young WL, Pile-Spellman J, Solomon RA (1998) Complex intracranial aneurysms: combined operative and endovascular approaches. Neurosurgery 43: 1304–12; discussion 1312–13
- Herkes GK, Morgan M, Grinnell V, Sorby W, Wong J, Rowe D, et al. (1993) EEG monitoring during angiographic balloon test carotid occlusion: experience in sixteen cases. Clin Exp Neurol 30: 98–103
- 12. Heros RC, Ameri AM (1984) Rupture of a giant basilar aneurysm after saphenous vein interposition graft to the posterior cerebral artery. Case report. J Neurosurg 61: 387–90
- Houkin K, Kamiyama H, Kuroda S, Ishikawa T, Takahashi A, Abe H (1999) Long-term patency of radial artery graft bypass for reconstruction of the internal carotid artery. Technical note. J Neurosurg 90: 786–90
- 14. Jafar JJ, Russell SM, Woo HH (2002) Treatment of giant intracranial aneurysms with saphenous vein extracranial-to-intracranial bypass grafting: indications, operative technique, and results in 29 patients. Neurosurgery 51: 138–44; discussion 144–46
- 15. Kocaeli H, Andaluz N, Choutka O, Zuccarello M (2008) Use of radial artery grafts in extracranial-intracranial revascularization procedures. Neurosurg Focus 24: E5
- Larson JJ, Tew JM Jr, Tomsick TA, van Loveren HR (1995) Treatment of aneurysms of the internal carotid artery by intravascular balloon occlusion: long-term follow-up of 58 patients. Neurosurgery 36: 26–30; discussion 30
- 17. Lawton MT, Hamilton MG, Morcos JJ, Spetzler RF (1996) Revascularization and aneurysm surgery: current techniques, indications, and outcome. Neurosurgery 38: 83–92; discussion 92–4
- Lougheed WM, Marshall BM, Hunter M, Michel ER, Sandwith-Smyth H (1971) Common carotid to intracranial internal carotid bypass venous graft. Technical note. J Neurosurg 34: 114–18
- Marshall RS, Lazar RM, Young WL, Solomon RA, Joshi S, Duong DH, et al. (2002) Clinical utility of quantitative cerebral blood flow measurements during internal carotid artery test occlusions. Neurosurgery 50: 996–1004; discussion 1004–05
- Mathis JM, Barr JD, Jungreis CA, Yonas H, Sekhar LN, Vincent D, et al. (1995) Temporary balloon test occlusion of the internal carotid artery: experience in 500 cases. Am J Neuroradiol 16: 749–54

- Mohit AA, Sekhar LN, Natarajan SK, Britz GW, Ghodke B (2007) High-flow bypass grafts in the management of complex intracranial aneurysms. Neurosurgery 60: ONS 105–22; discussion ONS 122–23
- 22. Morgan MK, Ferch RD, Little NS, Harrington TJ (2002) Bypass to the intracranial internal carotid artery. J Clin Neurosci 9: 418–24
- O'Shaughnessy BA, Salehi SA, Mindea SA, Batjer HH (2003) Selective cerebral revascularization as an adjunct in the treatment of giant anterior circulation aneurysms. Neurosurg Focus 14: e4
- Plangger CA, Mohsenipour I, Grunert V, Twerdy K (1989) Rupture of a giant aneurysm of the inferior wall of the internal carotid artery after saphenous vein interposition graft to the middle cerebral artery. Zentralbl Neurochir 50: 61–63
- Polevaya NV, Kalani MY, Steinberg GK, Tse VC (2006) The transition from hunterian ligation to intracranial aneurysm clips: a historical perspective. Neurosurg Focus 20: E3
- 26. Regli L, Piepgras DG, Hansen KK (1995) Late patency of long saphenous vein bypass grafts to the anterior and posterior cerebral circulation. J Neurosurg 83: 806–11
- 27. Roski RA, Spetzler RF, Nulsen FE (1981) Late complications of carotid ligation in the treatment of intracranial aneurysms. J Neurosurg 54: 583–87
- Sekhar LN, Kalavakonda C (2002) Cerebral revascularization for aneurysms and tumors. Neurosurgery 50: 321–31
- Standard SC, Ahuja A, Guterman LR, Chavis TD, Gibbons KJ, Barth AP, et al. (1995) Balloon test occlusion of the internal carotid artery with hypotensive challenge. Am J Neuroradiol 16: 1453–58
- Sudhakar KV, Sawlani V, Phadke RV, Kumar S, Ahmed S, Gujral RB (2000) Temporary balloon occlusion of internal carotid artery: a simple and reliable clinical test. Neurol India 48: 140–43
- Sundt TM Jr, Piepgras DG, Marsh WR, Fode NC (1986) Saphenous vein bypass grafts for giant aneurysms and intracranial occlusive disease. J Neurosurg 65: 439–50
- 32. Swearingen B, Heros RC (1987) Common carotid occlusion for unclippable carotid aneurysms: an old but still effective operation. Neurosurgery 21: 288–95
- Tulleken CA, Verdaasdonk RM, Berendsen W, Mali WP (1993) Use of the excimer laser in high-flow bypass surgery of the brain. J Neurosurg 78: 477–80
- 34. U-King-Im JM, Gillard J (2006) Conventional digital subtraction angiography for carotid disease. In: Gillard J, Graves M, Hatsukami T, Yuan C (eds) Carotid disease: role of imaging in diagnosis and management. Cambridge University Press, Cambridge
- 35. van Doormaal TP, van der Zwan A, Verweij BH, Langer DJ, Tulleken CA (2006) Treatment of giant and large internal carotid artery aneurysms with a high-flow replacement bypass using the excimer laser-assisted nonocclusive anastomosis technique. Neurosurgery 59: ONS 328–34; discussion ONS 334–35
- van Rooij WJ, Sluzewski M, Slob MJ, Rinkel GJ (2005) Predictive value of angiographic testing for tolerance to therapeutic occlusion of the carotid artery. Am J Neuroradiol 26: 175–78
- Wolfe SQ, Tummala RP, Morcos JJ (2005) Cerebral revascularization in skull base tumors. Skull Base 15: 71–82
- Yasargil MG (1967) Experimental small vessel surgery in the dog including patching and grafting of cerebral vessels in the form of functional extracranial-intracranial shunts. St Louis, Mosby