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Carotid Artery Stenosis

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INTRODUCTION

Recent analysis by the American Heart Association **(AHA)** shows an annual stroke rate of approximately 795,000 strokes in the United States of America [1]. Nearly 85% of strokes are ischemic, with an estimated >50% due to large-vessel atherosclerosis [2]. As part of a systemic thromboembolic disease, the carotid bifurcation is by far the most frequently affected site of atheroma formation in the carotid arteries. An arterial bifurcation causes turbulent flow that leads to an increased risk of intimal damage, initiating atherosclerotic plaque formation. Due to progressive intima damage, lipid accumulation, platelet deposition, calcification and fibroplasias, the atheromatosis causes intima-media thickness and eventual occlusion of the artery when the plaque ruptures [2]. The risk factors for the development of carotid artery stenosis are similar to those for peripheral atherosclerotic disease, including increased age, male gender, hypertension, smoking, diabetes mellitus and dyslipidaemia.

A focal ipsilateral neurologic deficit, e.g. contralateral paraplegia, higher cortical dysfunction or amaurosisfugax, marks a carotid artery stenosis as symptomatic. Depending on the duration of the symptoms, the neurological deficit can be classified as followed: Transient Ischemic Attack **(TIA)** (resolution in < 24 hours), reversible Ischemic Neurologic Deficit **(RIND)** (resolution in 24 hours – 6 weeks) or, minor (non-disabling) or major (disabling) strokes [3]. The majority of strokes are due to thrombo-embolic events of a ruptured unstable plaque. Only a minority of strokes are caused by direct flow-limiting stenosis.

A multidisciplinary consensus has developed a set of criteria to estimate carotid stenosis via duplex ultrasound examination (Table 1) [4-6]. The proposed system mainly estimates the degree of stenosis based on the primary parameters being the peak systolic flow **(PSV)** and visible lumen decrease due to plaque presence. An additional set of parameters was adopted to confirm stenosis rates and to allow extra evidence in cases of confounding technical or clinical factors. If significant stenosis is suspected, a Computed Tomography **(CT)** or Magnetic Resonance Imaging **(MRI)** angiography should be performed for optimal pre-operative workup.

Table 1: Duplex Criteria for ICA Stenosis Estimation.

	Primary parameters		Additional parameters	
Degree of ICA stenosis (%)	PSV (cm/s)	Plaque diameter estimation (%)	ICA/CCA PSV ratio	EDV (cm/s)
Normal	< 125	None	< 2.0	< 40
< 50	< 125	< 50	< 2.0	< 40
50 – 69	125 - 230	≥ 50	2.0 - 4.0	40 - 100
≥ 70	> 230	≥ 50	> 4.0	> 100
Occlusion	Undetectable	100; no lumen detectable	NA	Undetectable

PSV = Peak Systolic Velocity, ICA = Internal Carotid Artery, CCA = Common Carotid Artery, EDV = End Diastolic Velocity, NA = Not Applicable

Indications for surgical intervention in symptomatic patients are mainly based on twolarge Randomised Control Trials **(RCT):** the North American Symptomatic Carotid Endarterectomy Trial **(NASCET)** and the European Carotid Surgery Trial **(ECST)** [7,8]. In symptomatic patients with a high grade stenosis (70-99%), surgery provides a clear significant reduction in mortality and recurrent ipsilateral cerebrovascular events when compared to best medical therapy. In cases of moderate stenosis (50-69%), a significant ipsilateral stroke reduction was seen in the Carotid Endarterectomy **(CEA)** group. Therefore, surgery is the preferred treatment of choice in this population. In the age group ≥75 years of age, the advantage of CEA over best medical therapy was especially strong in the reanalysis of the ECST and NASCET populations. In patients with only a minor stenosis (<50%), there was no benefit from CEA. Therefore, best medical therapy should be applied [7-9].

When surgery is indicated, the optimal interval between the onset of the ischemic event and the performance of CEA remains unclear. Early surgical treatment may lead to an increased risk of

postoperative haemorrhage, brain oedema or extension of the ischemic infarction. Hence, surgery is often delayed by six to eight weeks [10]. However, recent research, including a further analysis of the NASCET and ECST trials, suggests a beneficial outcome when surgery is performed just two weeks after a cerebral vascular event, if the patient is neurologically stable [11].

Asymptomatic patients with a routine finding of a carotid bruit have a 1-3% yearly stroke rate, yet the presence of carotid bruits seemingly has a greater risk of generalised cardiovascular disease and mortality than cerebrovascular accidents [12]. Treatment recommendations in cases of asymptomatic patients are mainly based upon the results of two large randomized controlled trials: the Asymptomatic Carotid Atherosclerosis Study **(ACAS)** and the Asymptomatic Carotid Surgery Trial **(ACST)** [13,14]. In cases of low-grade stenosis (<50%), surgical treatment is not indicated and best medical therapy should be applied. The stenosis grade of 50-69% requires a yearly follow-up and best medical therapy, but the benefit of revascularisation still remains a topic of debate [15]. When a stenosis grade of ≥70% is present, surgical treatment is beneficial and indicated unless substantial comorbidities contraindicate major surgery [9,15-17]. As in symptomatic patients, the morphology of the plaque, e.g. ulceration or soft unstable plaques (Figure 1), is not yet included in the treatment algorithm, as it lacks proper routine identification methods [18]. Research on plaque characteristics as part of the pre-operative workup is still ongoing.

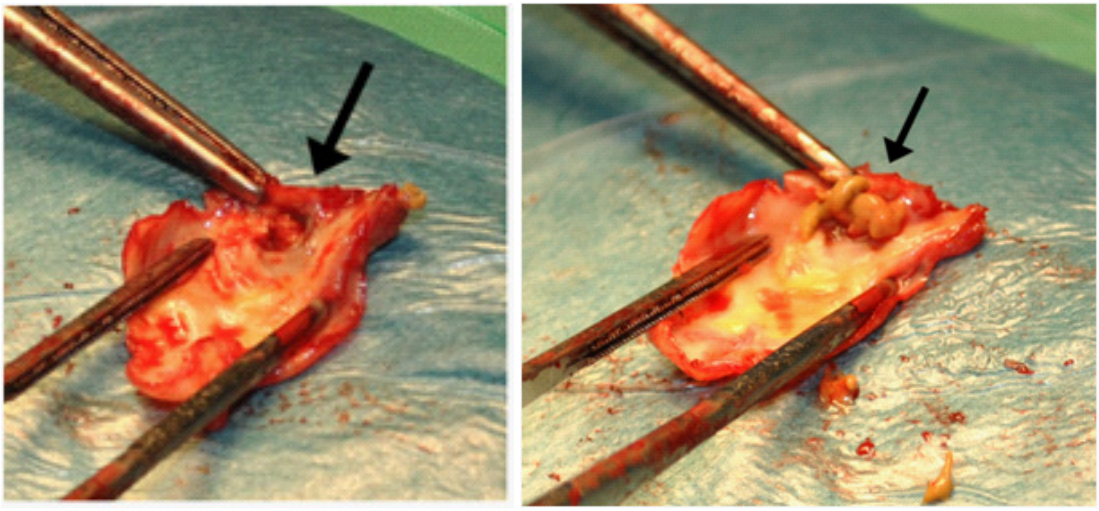

Figure 1: Plaque post-endarterectomy. The arrow points at an ulceration (1a) with soft tissue debris evacuation on gentle palpation (1b).

Best medical therapy involves the admission of statins for dyslipidaemia, quitting smoking, weight control, correction of glycaemia in diabetic patients, correction of hypertension, antiplatelet therapy (e.g. low-dose aspirin) and stimulation of an overall healthy lifestyle [19-21].

Other, less frequent carotid pathologies are carotid dissection, carotid aneurysm and carotid body tumors, but these will not be discussed in this chapter.

ANAESTHESIA

Pre-operative workups must include a cardiac evaluation due to the nature of the arteriosclerotic disease. CEA may be performed under local anaesthesia, and most frequently a combination of a superficial cervical plexus block combined with a deep cervical plexus is used. A superficial cervical plexus block is performed by injecting a local anaesthetic at the midpoint of the posterior wall of the sternocleidoid muscle, blocking the cutaneous branches of the cervical plexus. A deep cervical block is obtained by infiltration of C2-C4 via a single injection [22,23]. In the General Anaesthesia Versus Local Anaesthesia **(GALA-)** Trial for Carotid Surgery, data was collected from 3,526 patients over an eight year period. It was found that a superficial block provides sufficient anaesthesia and that the addition of a deep block does not increase patient comfort, but does increase the risk of local complications. The most important complications include convulsions, subarachnoid injection, paralysis of the phrenic nerve, significant local hematoma and dysphagia [23,24]. Local anaesthesia did reduce the need for shunting from 43% to 14%, but there was no significant difference in stroke rate or mortality [25].

A Cochrane meta-analysis showed that the use of local anesthetic was associated with a reduction in the risk of local haemorrhage within 30 days after surgery. However, there was no evidence of perioperative stroke reduction [27]. The choice of anaesthesia mainly depends on the preferences of the patient, anesthesiologist and surgeon. The major advantage of local anaesthesia is that it allows for direct neurologic monitoring of the conscious patient. Altered contra lateral motor deficiency or altered consciousness after clamping has a specificity of 59-91%, and sensitivity is 57-99% [26].

Cerebral oxygenation monitoring during general anaesthesia remains an important issue. Several monitoring techniques have been proposed, including Electro-Encephalogram **(EEG)** monitoring, Near-Infrared Spectroscopy **(NIRS)** monitoring, Transcranial Doppler Ultrasonography **(TCD)** and Somatosensory Evoked Potentials **(SSEPs)** [27-39]. However, these techniques often require a certain expertise in interpretation, especially when considering the effect of general anaesthesia on cerebral function the anesthesiologist should provide a constant state of increased blood pressure to allow for adequate contralateral perfusion via the circle of Willis unless a shunt is placed.

CAROTID ENDARTERECTOMY

Place the patient in the supine position with the head in slight extension and contralateral rotation. Make sure the head of the patient remains supported. After careful palpation of the sternocleidomastoid muscle, incise longitudinally along the anterior board. Dissect through the platysma down until the sternocleidomastoid muscle. If encountered, ligate the external jugular vein and mobilize the great auricular nerve posteriorly. Dissect past the anterior border of the sternocleidomastoid muscle. If necessary, the superior belly of the omohyoid muscle can be

divided to increase exposure. Identify and ligate the common facial vein, as it is an important landmark that often overlays the carotid bifurcation. Mobilize the jugular vein posteriorly and expose the carotid arteries. When exposing the carotid bifurcation, take care in identifying and sparing the vagal nerve, hypoglossal nerve and ansacervicalis. After intravenous heparinization, the Internal Carotid Artery **(ICA)** is clamped, followed by the clamping of the Common Carotid Artery **(CCA),** External Carotid Artery **(ECA)** and superior thyroid artery (Figure 2).

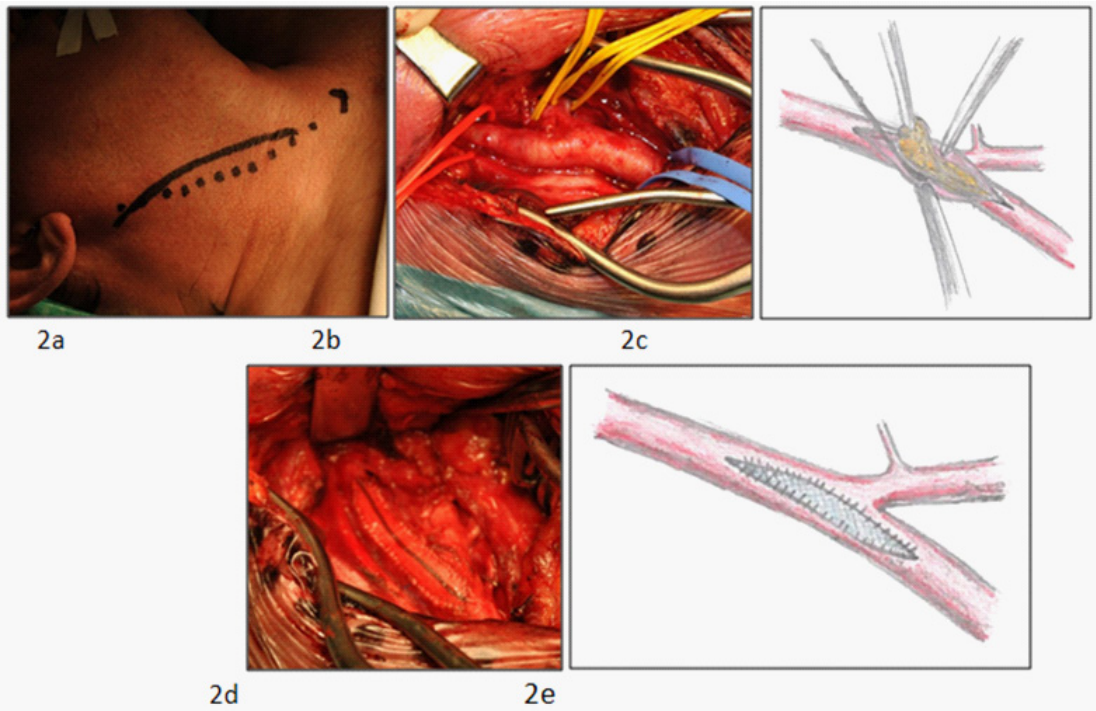

Figure 2: Surgical procedure of a carotid endarterectomy. Figure 2 summarizes a carotid endarterectomy procedure. The anterior border of the sternocleidomastoid muscles (dotted line, 2a) and the incision line (straight line, 2a) are marked. The carotid bifurcation and superior thyroid artery are identified (2b). An endarterectomy is performed by using gentle traction on the plaque (arrow, 2c) and separating it from the arterial wall using a spatula (2c). A patch is then sutured via a running suture (2d, 2e).

A longitudinal arteriotomy is made from the common carotid arteryto the internal carotid artery until the plaque is sufficiently exposed. The decision to place a shunt should then be made. The removal of the carotid plaque is performed by dividing the media and adventitia walls by an using a endarterectomy spatula. Fixation stitches should be placed on the distal transition line of the endarterectomy border to prevent flapping of the residual distal wall after restoring blood flow. Evert and remove the plaque at the base of the external carotid artery. Measure the required patch size and cut the patch to its appropriate length. Suture the patch with a running

suture. Several trials have showed that patch angioplasty favors primary closing [40,41]. Before finalizing the suture, flush out any debris. Restore vascularization by releasing the internal and external carotid artery clamps first, prior to releasing all other clamps. Administer intravenous protamine and suture the wound in layers.

Shunting allows for constant ipsilateral perfusion (Figure 3). The placement of a shunt reduces the time pressure, but increases important risks, including the dislodging of the shunt with significant blood loss, plaque embolization during insertion, shunt occlusion, intima damage, postoperative thrombosis and, technical challenges for the endarterectomy [42]. In cases of selective shunting, the major criterion for shunt placement is the so called stump pressure. After clamping, a pressure needle is inserted in the ICA in order to measure the local blood pressure. The minimally required stump pressure is still undetermined, but typically a stump pressure of 40-50 mmHg systolic is accepted as safe and does not require shunting [43].

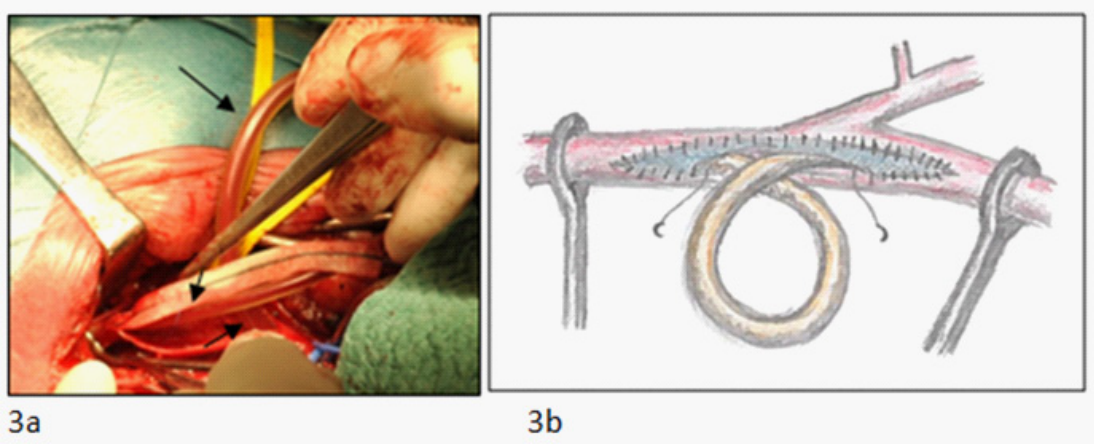

Figure 3: Carotid artery shunting. The arrows (1a) point at the visible parts of the carotid shunt. Figure 1b depicts the positioning of a carotid shunt nearing the completion of a patch suturing.

EVERSION ENDARTERECTOMY

Eversion endarterectomy is a technique first described by DeBakey in 1959 [44]. Initial dissection is identical to a conventional endarterectomy, including the clamping of the carotid arteries. However, instead of a longitudinal arteriotomy of the carotid bifurcation, the ICA is transected at the ostium. The edge of the plaque is then visualized. Fixate the plaque and evert the internal carotid artery away from the plaque with constant traction, essentially peeling the artery of the plaque. Inspect the remaining adventitia for residual debris. After removal, a circular endarterectomy is performed at the CCA and ECA. Re-anastomose of the CCA and ICA is done via a running suture. Remove any debris by flushing the vessel before final closure. The ICA should be unclamped last to avoid debris migration [45].

Eversion CEA (eCEA) is a safe alternative to conventional CEA, but its superiority to conventional CEA is still unproven [46]. According to a Cochrane review of 2002, there is still insufficient data to finalize conclusions on better outcomes in stroke rate and mortality. The Cochrane review did show an overall statistically significant decrease in restenosis or occlusion for eCEA in comparison to conventional CEA of 2-5% versus 5-2%, respectively, even though the individual studies found no significant difference in restenosis rate. The large multicenter EVEREST trial showed a 30 day event rate of 13.3% and an overall mortality and major stroke rate at the 30day of 1.3% [47,48]. A point of concern with the eversion method is the inability to control the distal plaque, possibly leading to flow limitation after reperfusion, but the occurrence of distal restenosis does not seem to be a problem [48].

CAROTID ARTERY STENTING

Using the Seldinger technique, introduce a dilatator in the common femoral artery. A diagnostic catheter is then advanced and a perioperative angiogram is performed to identify and assess the aortic, carotid, vertebral and intra-cranial circulation. Introduce a 0.035 inch stiff guidewire in the carotid artery and past the atherosclerotic lesion. Achieve anticoagulation by intravenous admission of heparin or a direct thrombin inhibitor (e.g. bivalirudin). The diagnostic catheter is replaced with a guidance sheath up to the arteriosclerotic plaque. The stiffwire is then replaced with a 0.014 inch guide wire and an embolic protection device is advanced past the lesion using a rapid-exchange monorail system. Using angiography, measure the appropriate balloon and stent size. Pre-dilation with an undersized balloon is often required to allow passage of the stent delivery catheter. The stent is deployed over the entire lesion and dilated using a balloon catheter. When the control angiogram shows a successful result, remove the catheters. Compress or apply a closure device at the puncture site. All patients require clopidogrel 75mg ones daily for at least once month.

In symptomatic patients, endarterectomy is associated with a lower risk of death or stroke in the 30 day postoperative period and hence is the preferred treatment of choice. When investigating age as a modifier, an age greater than 70 years significantly increased the rates of death or any stroke in the 30-day postoperative period after endovascular treatment. Similar results were not found when comparing asymptomatic patients [49,50].

The results of the largest RCTs comparing CAS to CEA are summarized in table 1. CAVATAS, CREST, EVA-3s and SPACE showed an increased restenosis rate in long term follow-up (Table 2) [51-55]. It remains unclear whether the embolic protection decrease the risk of preoperative stroke. Subgroup analysis of the International Carotid Stenting Study **(ICSS)** actually showed an increase in ischemic brain lesions in MRIs with the application of the embolic protection devices [56].

Table 2: Results of large randomized controlled trials comparing carotid endarterectomy to carotid artery stenting.

Procedure	Randomised controlled trials	30 day stroke (%)	30 day mortality (%)	Cumulative ≥70 Restenosis (%)
CEA	CREST, 2010 [54]	2,3	0,3	6,3 (2 years)
	SPACE, 2008 [52,69]	6,2	0,9	4,6 (2 years)
	EVA-3S, 2006 [70,71]	3,9†		2,8 (3 years)
	SAPPHIRE, 2004 [72]	9,9†		NA
	CAVATAS, 2001 [55,73]	8,3	1,6	5,1 (1 year)
CAS	CREST, 2010 [54]	4,1	0,7	6,0 (2 years)
	SPACE, 2008 [52,69]	7,5	0,7	10,7 (2 years)
	EVA-3S, 2006[70,71]	9,6†		3,3 (3 years)
	SAPPHIRE, 2004 [72]	4,4†		NA
	CAVATAS, 2001 [55,73]	10,9	2,8	22,0 (1 year)

CAROTID BYPASS AND INTERPOSITION GRAFTING

With CEA as the gold standard, carotid bypass grafting is generally reserved for challenging cases, such as extensive disease or challenging anatomy, post radiotherapy (peri)vasculitis, restenosis after a previous CEA, malignant invasion of the carotid body, or artery or patch infection from a previous CEA. Several small studies have been conducted on both carotid artery bypass and interposition grafting. In cases of carotid bypass surgery, several techniques have been described, including carotid-carotid bypass, common-internal carotid artery bypass and, bypass from the ascending carotid artery or subclavian artery to the internal carotid artery. There is great variance in published restenosis rates of 3.2 % - 16.4%, postoperative cerebral vascular events of 0.5-5% and low perioperative mortality of 0-1.8% [57-63]. In the case of interposition grafting the diseased artery is resected. Results for interposition grafting are based on studies with smaller sample sizes, often with only a minority of the patients actually undergoing anatomical interposition grafting. Results varied greatly but overall showed high restenosis rates of 2.2 - 16%, postoperative cerebral vascular events of 0 - 5% and mortality rates of 0-4% [64-67].

However, a recent publication on a population of 103 interposition grafts showed excellent long term patency up to 7.5 years follow-up (mean 29.1 months). This is, to our knowledge, the largest study of interposition grafting yet, especially on the primary treatment of significant carotid stenosis. The technique applied in this study was named Carotid Bifurcation and Interposition of a (PTFE Graft (BRIG) and was performed by a single experienced surgeon, P. De Vleeschauwer. In the BRIG technique, the diseased carotid bifurcation is completely removed until a viable proximal and distal carotid artery is identified. The external carotid artery is routinely ligated. A 6-mm thin wall PTFE graft is then sutured via an end to end running suture between the CCA and ICA (Figure 4). Only in cases of a contralateral occlusion or previous BRIG procedure was, the external carotid artery revascularized via an end to side PTFE anastomosis on the already inserted interposition

graft. Results showed a 1% early postoperative mortality rate and a 1.9% minor strokes rate, with near full recovery in long term follow-up. Kaplan Meier analysis showed a significant lower restenosis rate compared to CEA, with 0% restenosis after two years of follow-up [68].

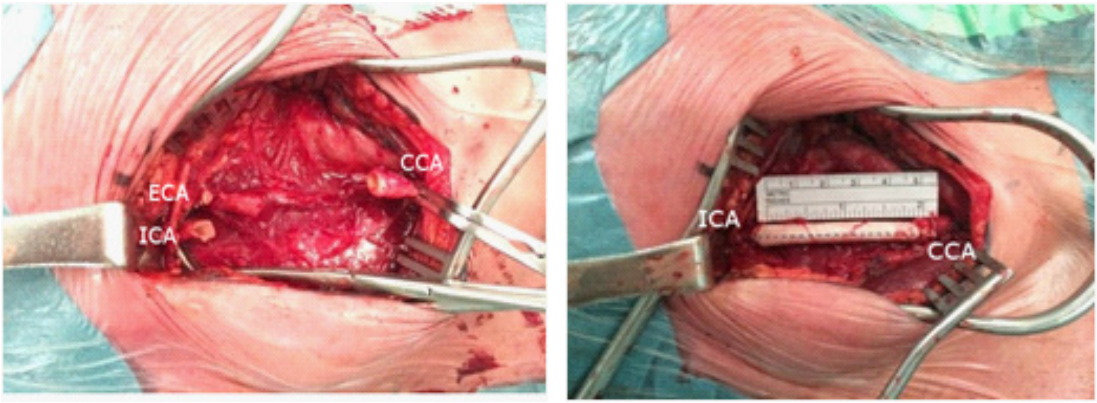

Figure 4: BRIG procedure. Peroperative images of a BRIG procedure showing the resected carotid bifurcation (4a) and interpositioning of a PTFE graft between the CCA and ICA.

BRIG = Carotid Bifurcation Resection and Interposition of a PTFE Graft, ECA = External Carotid Artery, ICA = Internal Carotid Artery, CCA = Common Carotid Artery, PTFE = Polytetrafluoroethylene.

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