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## Review

## Should intentional endovascular stent-graft coverage of the left subclavian artery be preceded by prophylactic revascularisation?

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## Summary

Thoracic endovascular aortic repair (TEVAR) has emerged as a promising therapeutic alternative to conventional open aortic replacement but it requires suitable proximal and distal landing zones for stent-graft anchoring. Many aortic pathologies affect in the immediate proximity of the left subclavian artery (LSA) limiting the proximal landing zone site without proximal vessel coverage. In patients in whom the distance between the LSA and aortic lesion is too short, extension of the landing zone can be obtained by covering the LSA's origin with the endovascular stent graft (ESG). This manoeuvre has the potential for immediate and delayed neurological and vascular symptoms. Some authors, therefore, propose prophylactic revascularisation of the LSA by transposition or bypass, while others suggest prophylactic revascularisation only under certain conditions, and still others see no requirement for prophylactic revascularisation in anticipation of LSA ostium coverage. In this review about LSA revascularisation in TEVAR patients with coverage of the LSA, we searched the electronic databases MEDLINE and EMBASE historically until the end date of May 2010 with the search terms left subclavian artery, covering, endovascular, revascularisation and thoracic aorta. We have gathered the most complete scientific evidence available used to support the various concepts to deal with this issue. After a review of the current available literature, 23 relevant articles were found, where we have identified and analysed three basic treatment concepts for LSA revascularisation in TEVAR patients (prophylactic, conditional prophylactic and no prophylactic LSA revascularisation). The available evidence supports prophylactic revascularisation of the LSA before ESG LSA coverage when preoperative imaging reveals abnormal supra-aortic vascular anatomy or pathology. We further conclude that elective patients undergoing planned coverage of the LSA during TEVAR should receive prophylactic LSA transposition or LSA-to-left-common-carotid-artery (LCCA) bypass surgery to prevent severe neurological complications, such as paraplegia or brain stem infarction.

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## 1. Introduction

Most thoracic aortic pathologies are serious conditions demanding referral to highly specialised cardiovascular

centres with a sustained expertise with complex cases. The estimated yearly incidence of thoracic aortic aneurysms (TAAs) is six cases per 100,000 [1,2], and of acute aortic dissection 10–20 cases per million [1]. Conventional treatment consists of surgical replacement of the diseased aortic segment with a vascular graft: this requires thoracotomy, aortic cross-clamping and, occasionally, cardiopulmonary bypass with hypothermic circulatory arrest and antegrade selective cerebral perfusion. Some operative procedures are extensive, often technically complex and may be associated with substantial morbidity, including bleeding, paraplegia,

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stroke, renal insufficiency and the need for prolonged ventilatory support [3–5], as well as substantial mortality [6–8]. Thoracic endovascular aortic repair (TEVAR) for aortic disease has, since its introduction by Dake [9], emerged as a promising therapeutic alternative to conventional operative treatment. It is a less invasive method associated with considerably lower mortality and morbidity [10–15]. This treatment method is based on the principle of segmental exclusion with an endovascular stent graft (ESG) [11,14,16]. However, TEVAR requires suitable proximal and distal landing zones for stable ESG fixation. It has been suggested [17,18] that 2 cm of normal aortic wall are required for adequate and stable sealing. Unfortunately, many diseases and injuries of the thoracic aorta occur in the immediate proximity of the origin of the supra-aortic branches, such as the left subclavian artery (LSA). In patients in whom the distance between the LSA and the aortic lesion is too short or in those in which the LSA ostium is located within the aortic pathology, extension of the landing zone can be obtained by covering the LSA's ostium with the ESG or positioning the proximal margin of the endoprosthesis directly on the ostium of the LSA [19,20].

However, there are reports of delayed onset of vertebrobasilar insufficiency and arm ischaemia [21,22], following LSA covering with ESGs. Insufficient blood supply to the posterior cerebral circulation may evolve in the presence of hypoplasia of the right vertebral artery (VA) and/or posterior communicating arteries (PCOMAs) exits [23]. Surgical transposition of the LSA to the left common carotid artery (LCCA) or LCCA-to-LSA bypass prior to TEVAR with LSA covering may preserve blood flow to the brain stem and spinal cord [1,12]. Alternatively, a branched ESG approach can be implemented [24,25].

In the current review, we outline various strategies that have been advocated to manage and treat patients requiring covering of the LSA with ESG.

## 2. Material and methods

A review of the relevant available literature was performed by searching the complete electronic MEDLINE and EMBASE databases historically until the end date of May 2010. The terms left subclavian artery, covering, endovascular, revascularisation and thoracic aorta were used as search terms. All relevant publications were selected and analysed. Only cases of isolated complete covering of the LSA ostium were included. All complications were documented, independent of interpretation.

## 3. Results of the review

### 3.1. Prophylactic LSA revascularisation

Some authors recommend prophylactic LSA transposition or LSA bypass prior to intentional LSA covering. In a series by Grabenwöger et al. in 2000 [26], 21 patients received TEVAR, of whom 19 presented a descending aortic aneurysm and two an aortic dissection. In that series, nine patients (42.9%) underwent prophylactic LSA transposition before covering

the LSA because of a short proximal neck. There were no neurological complications, although the distal thoracic aorta was covered in seven patients without postoperative paraplegia. The authors suggested two possible explanations for their observations. First, in the case of atherosclerotic aneurysms, collaterals supply the spinal cord, and many intercostal branches may be already occluded by thrombotic material. Second, the sudden deployment of the ESG followed by the occlusion of the intercostal branches does not produce a steal phenomenon in spinal cord perfusion [27]. In a subsequent study [28], 19 patients with acute thoracic aortic syndrome were treated with TEVAR, and only one suffered left-arm ischaemia requiring secondary revascularisation by LSA transposition. Another patient without LSA revascularisation developed paraparesis, which was managed successfully by cerebrospinal fluid drainage for 72 h without residual neurological deficit. These authors seem to have changed their strategy and decided not to perform prophylactic LSA transposition before covering the LSA.

Heijmen et al. [29] treated 28 TAAs in 27 patients with TEVAR. In five patients (18%), the LSA was transposed first as a separate procedure to lengthen the proximal landing zone. None developed paraplegia or paraparesis. The authors argued that inadvertent ESG placement across the LSA's origin may lead to the development of an occlusive thrombus, resulting in left-upper-extremity ischaemia, despite unimpeded blood flow and the absence of a pressure gradient at completion in angiography [12,30]. Moreover, they argued that LSA obstruction may lead to cerebrovascular insufficiency in compromised patients due to flow reversal in the ipsilateral VA, and because that vessel also contributes to the anterior spinal artery, it is conceivable that flow reversal may increase the risk of spinal cord ischaemia. Furthermore, persistent flow in the LSA could result in a retrograde type-II endoleak [31].

In 2006, Czerny et al. [32] published a series of 11 patients with TAA or acute dissection. TEVAR was performed after autologous sequential transposition of the LCCA into the brachiocephalic trunk and of the LSA into the already transposed LCCA, or by total arch rerouting with an extra-anatomical vascular prosthesis. The authors routinely perform LSA transposition in any type of chronic disease of the distal aortic arch prior to ESG placement, with the primary intention of preserving posterior cerebral circulation, and not to maintain antegrade perfusion of the left upper extremity. Furthermore, as do other investigators [29,31], they emphasise that retrograde perfusion of the aneurysmal sac via the LSA may result in type-II endoleak formation.

Cambria et al. performed TEVAR in 28 patients [33], six of whom (21%) required a preliminary LSA transposition to provide an adequate proximal neck for ESG fixation. All patients with LSA covering underwent prophylactic revascularisation. The authors' argument for this was not primarily a concern for any neurologic or vascular symptoms, but their concern for significant endoleaks, as they believe that a majority of the patients will tolerate the LSA's sacrifice.

A multicentre registry analysis by Buth et al. [34] assessed the incidence and risk factors for paraplegia and/or paraparesis and stroke by both univariate and multivariate regression models of 606 patients with thoracic aortic

disease, who underwent TEVAR. The authors found among other factors an independent correlation between spinal cord ischaemia (SCI) and LSA covering without revascularisation (odds ratio 3.9,  $p = 0.27$ ). In addition, a neurologic complication (paraplegia or stroke) developed in 8.4% of the patients in whom LSA covering was required, compared with 0% of patients following prophylactic revascularisation ( $p = 0.049$ ).

### 3.2. Conditional prophylactic LSA revascularisation

Other authors propose the concept of prophylactic LSA revascularisation only under certain conditions. Criado et al. [1] describe their experience with TEVAR in 47 patients with TAA or type-B aortic dissections. Proximal ESG attachment was distally within 2 cm of the LSA in 21 patients and proximal to the LSA in 10. In five cases, the ESG was placed across the origin of the LSA without prior revascularisation, and in eight patients, prophylactic transposition or bypass surgery was performed to revascularise the LSA to allow ESG placement more proximally into the mid/distal aortic arch. The authors did not experience any instance of paraplegia or stroke. They believe that LSA coverage is generally safe, even without prior transposition or bypass, but they recommend ascertaining the angiographic patency of the contralateral VA beforehand. Secondary revascularisation is easily achievable should vertebrobasilar or arm ischaemia develop. On the other hand, preliminary transposition of the LSA is the preferred strategy to prevent a type-II endoleak [29,31–33].

Schoder et al. [15] treated 58 patients with TEVAR, who required fixation of the ESG at the origin of arch vessels. In nine patients with TAA, the LSA originated within the aneurysm. The remaining patients presented a distance between the LSA and aneurysm or intimal tear of the dissection of less than 15 mm. Intentional coverage of the LSA resulted in complete occlusion in eight and partial occlusion in 24 patients. Primary surgical revascularisation of the LSA was performed in patients with a dominant left VA, with a marked stenosis of the right VA or an occluded internal carotid artery. In that study, 26 patients underwent surgical revascularisation of the supra-aortic vessels before TEVAR. Intentional covering of the LSA without previous revascularisation was performed in patients in whom two VAs were of good and equal calibre. Five of eight patients (63%) with complete occlusion of the LSA without prophylactic LSA revascularisation experienced adverse events. Two patients suffered major peri-procedural complications (one had paraplegia and the other critical arm ischaemia), and three had minor complications (two incomplete subclavian steal syndromes with temporary dizzy spells and one with temporary arm claudication). Secondary transposition of LSA was necessary in two patients, one to treat critical arm ischaemia, and one to treat a type-II endoleak. The authors argue that a lack of collaterals may lead to acute and sub-acute ischaemic symptoms following acute LSA occlusion, compared with the absence of symptoms in patients with atherosclerotic LSA stenosis or occlusion, where the slow progression of the stenosis promotes collateral vessel development [35]. They also emphasise that the combination of LSA coverage and the presence of a severely stenotic or hypoplastic right VA can cause vertebrobasilar ischaemia

and, thereby, irreversible neurological complications due to the incapacity of the contralateral VA to supply blood to the posterior circulation. In accordance with other studies [36,37], it is suggested that a non-compromised blood supply between the anterior and posterior circulation via the posterior communicating artery (PCOMA) [23] seems essential to avoid cerebral infarction. The authors therefore conclude that, before intentional LSA occlusion, one should evaluate the carotid arteries, VAs as well as the circle of Willis, to minimise the risk for ischaemic cerebral disorders. Surgical revascularisation of the LSA is recommended in patients in whom any concern for an ischaemic event exists. Transposition of the LSA or LSA-to-LCCA bypass surgery followed by ligation of the proximal LSA was also recommended to avoid retrograde perfusion of the aneurysm sac in distal arch aneurysms or to avoid retrograde perfusion of the false lumen in dissections.

Weigang et al. [38] evaluated the neurological complication rate in 20 patients with TAA or type-B aortic dissections undergoing TEVAR with complete ( $n = 14$ ) or partial ( $n = 6$ ) coverage of the LSA. All patients were subjected to a detailed preoperative exploration of vascular anatomy and pathology by means of Doppler ultrasound, computed tomography (CT) or magnetic resonance imaging (MRI) scan. In one patient with right carotid artery and VA occlusion, a LCCA-to-LSA bypass was performed prophylactically. After surgery, all patients underwent neurological examination and Doppler ultrasound to detect any neurological and peripheral vascular complications. Two patients developed late adverse neurological events: one developed a brainstem infarction due to an acute postoperative occlusion of the right VA causing left-sided facial paresis, dysarthria, hemiparesis and hemihypoaesthesia. That patient underwent a secondary LSA transposition to prevent further brainstem infarction due to impaired perfusion of the vertebrobasilar arteries. Another patient developed impaired binocular vision in conjunction with dizziness. Peripheral symptoms related to the LSA occlusion were observed in five of 14 patients as sensory and motoric deficits of the left hand and arm. However, the symptoms were mild and in four of them, they improved over time, making LSA revascularisation unnecessary. One was treated surgically with LCCA-to-LSA bypass and proximal LSA ligation, leading to an improvement of symptoms. Several groups of patients have been identified as needing prophylactic revascularisation of the LSA. These include coronary bypass grafting (CABG) patients with a patent left internal mammary artery (LIMA) because LSA occlusion in such cases may cause myocardial ischaemia [39]. Further, carotid artery or VA stenosis, as well as anatomic variants, such as the origin of the left VA from the aortic arch, the absence of fusion between the VAs and the basilar artery, an otherwise functionally compromised circle of Willis or aberrant subclavian arteries (lusoria) do not permit LSA occlusion without previous revascularisation, as in the presence of a covered lusorian artery, it carries the increased risk of consecutive cerebellar infarction [35,40]. Weigang et al. recommended that in patients with supra-aortic vessel pathology, prophylactic transposition of the LSA or LCCA-to-LSA bypass is required prior to LSA covering.

In 2006, Peterson et al. [18] attempted to develop guidelines for managing patients scheduled for TEVAR, who

present with aortic pathology adjacent to the supra-aortic branches. Their experience consisted of 70 patients, of whom 30 (43%) presented involvement of the proximal aortic landing segment or in whom it was adjacent to the supra-aortic branches. In all 30 patients, the LSA was covered by the proximal end of the ESG. Coverage of the LSA was managed by transposition of the LSA into LCCA (20 patients) and by bypass (two patients). In eight patients, no prophylactic revascularisation was performed. In the latter group, five patients experienced acute complications, including four patients who experienced a stroke and one who developed symptomatic subclavian steal syndrome requiring secondary LSA transposition. Of the four stroke patients, two showed infarction confined to distribution in the posterior circulation and ipsilateral to the covered LSA. Further imaging examination revealed the absence of the contralateral VA in both of those patients. In the two other patients (one of whom died), stroke was likely due to athero-embolisation from the aortic arch. By contrast, there were no strokes and no procedure-related deaths in the group of 22 patients, who had undergone prophylactic revascularisation. In the remaining 40 patients with thoracic aortic pathology distal to the supra-aortic branches, there was only one reported stroke. Furthermore, no late endoleaks related to retrograde aneurysmal sac perfusion from the LSA were reported. Based on their experiences, Peterson et al. recommended prophylactic LSA revascularisation before TEVAR in case of proximal thoracic aortic pathology arising within 15 mm from the LSA, especially when the dominant VA arises from the ipsilateral LSA. Moreover, prophylactic revascularisation should also be considered when multiple intercostal arteries are covered in patients with extensive descending thoracic aortic pathology. In line with Weigang et al. [38], LSA transposition is indicated in patients, who may undergo future coronary revascularisation (with a LIMA graft), and an LSA-to-LCCA bypass rather than LSA transposition (to circumvent unsafe clamping proximal to the LIMA) should be performed in patients, who have already undergone CABG with LIMA as bypass graft. In accordance with Schoder et al. [15], prophylactic revascularisation should be considered to prevent type-II endoleak in these patients.

Görich et al. [30] performed TEVAR with full coverage of the LSA without prior revascularisation in 23 patients. Three patients (13%) experienced mild neurovascular symptoms after a mean follow-up of  $12.1 \pm 7.3$  months. These include a temperature difference between the upper extremities with no loss of strength, exercise-dependent paraesthesia as well as non-exercise dependent, intermittent and completely reversible dizziness. They documented preoperative patency of both VAs in 18 patients. The remaining five patients underwent LSA coverage in an emergency setting, without prior demonstration of contralateral VA patency. The authors concluded that covering the LSA is well tolerated. However, similar to Criado and Schoder [1,15], Görich et al. emphasise that the LSA should only be covered in patients whose right VA is not pathological and no anatomical variant is present.

Tiesenhausen et al. [22] published a series of 10 patients suffering from TAA or aortic dissection and scheduled for TEVAR: two underwent prophylactic LSA transposition. During the mean follow-up of 18 months, two patients with aortic dissection as a primary pathology and without

prophylactic revascularisation developed a type-II endoleak with retrograde flow via re-entries and a false aortic channel. Because of the absence of thrombosis, one patient underwent transposition and the other LCCA-to-LSA bypass. A third patient developed subclavian steal syndrome with vertigo and left-arm claudication resolved by a postponed LSA transposition. Another two patients suffered from occasional vertigo but refused further surgical intervention. Like other authors [1,15,29,30,38], we recommend that significant stenosis or obstruction in the VAs or internal carotid arteries should be ruled out before TEVAR with intentional LSA occlusion to guarantee sufficient collateral blood flow. Tiesenhausen et al. [22] also consider, along with Heijmen [29] that intentional occlusion of the LSA might influence the spinal cord's upper blood supply. Tiesenhausen et al. [22] conclude that intentional occlusion of the LSA (without prophylactic revascularisation) in TEVAR seems to be a safe procedure. Although all patients with intentionally occluded LSA did not develop postoperative neurological symptoms or left-arm ischaemia, three of eight patients (37.5%) without prior revascularisation developed a subclavian steal syndrome with vertigo and resting pain or claudication during follow-up. The indication for a secondary revascularisation was set in all three patients (of whom only one was revascularised). Two additional patients, who had a type-II endoleak after the primary procedure, received revascularisation 3 months later. Five of eight patients (62.5%) with intentional occlusion of the LSA without prophylactic revascularisation were said to require late revascularisation, which seems to contradict the authors' own conclusion.

Rehders et al. [40] reported the clinical history of 22 TEVAR patients, who underwent occlusion of the LSA during ESG placement. The need for post-interventional transposition of the LSA was evaluated. They performed either high-resolution contrast-enhanced CT or three-dimensional (3D) MR angiography (MRA) before TEVAR to exclude vertebral/carotid or coronary stenoses and to visualise the LSA in relation to the aortic pathology. If intentional coverage of the LSA ostium was unavoidable, a balloon occlusion test of the LSA was performed to screen for left-arm ischaemia. In their series of 22 patients in whom the LSA was occluded by the ESG, no patient was symptomatic during the 30-min LSA balloon occlusion test. Further, ESG occlusion of the LSA was followed by a mean 26% differential in systolic blood pressure between the right and left arms. However, no patient showed any signs of malperfusion during hospitalisation. During a mean follow-up of  $24 \pm 15.8$  months, 15 (68.2%) patients remained completely asymptomatic, revealing no functional deficit or temperature difference between the arms, while seven patients reported mild subclavian-steal-syndrome symptoms. No patient required any secondary surgical intervention. Rehders et al. challenge the need for prophylactic transposition or bypass of the LSA. They cite publications [41,42] that show that most patients with an ultrasound-documented subclavian steal are asymptomatic, and argue that collateral perfusion of the left arm seems adequate as flow inversion from a normal contralateral VA to the left VA can compensate for intentional LSA coverage. They point out that surgical treatments correlate with a mortality of 1.2–5% when performed on occlusive LSA lesions, thereby increasing the overall risk of TEVAR, and



recommend that LSA transposition or bypass procedures should be reserved for those patients who develop ischaemic symptoms or for those who have a potentially compromised collateral arm supply, including patients presenting previous CABG with LIMA, a critically stenosed carotid and/or VAs, or functional compromise of the circle of Willis or anatomical variants. They maintain that pre-interventional balloon testing is unnecessary as long as appropriate functional and anatomical connections to the basilar artery have been documented by MRA and normal flow directions in both VAs are visible in Doppler flow investigations.

Caronno et al. [17] also advocate individual prophylactic bypass or transposition of the LSA after coverage of the LSA ostium. They performed a retrospective analysis of 11 patients with TEVAR and intentional coverage of the LSA ostium. Mean systolic pressure in the left arm decreased by  $38 \pm 17$  mmHg, and no patient showed any signs of left-arm malperfusion during hospitalisation. Paraplegia was not observed, but one patient developed a transient ischaemic attack. During a mean follow-up of  $19 \pm 8$  months, all patients were completely asymptomatic, presenting no functional deficits or temperature differentials between the two arms. Caronno et al. also mention that, besides the VA, other muscular arterial branches in the neck and shoulder region serve as collaterals when occlusion of LSA occurs [30,43,44]. They conclude that intentional occlusion of the LSA is well tolerated, that surgical LSA revascularisation procedures add to TEVAR's invasiveness and overall risk and that (in accordance with Rehders et al.) these procedures should be reserved for those patients who develop ischaemic symptoms or who may have a compromised collateral arm supply. Again, in accordance with many authors [1,15,22,29,30,38,40], they emphasise that intentional occlusion of the LSA ostium may influence the perfusion of the upper spinal cord segment. They also recommend careful screening for anomalies of the supra-aortic arteries, as have many authors [15,29,31–33,38]. Caronno et al. did not record cases of type-II endoleak from the LSA in their series, and they suggest that coil embolisation can be easily performed in such a case through a catheter placed in the LSA from the left arm.

Reece et al. [45] report a series of 27 patients undergoing TEVAR with LSA coverage. Seven patients (25.9%) required prophylactic LSA revascularisation (all had LSA bypass) based on their specific vascular anatomy and the estimated risk of ischaemic complications (e.g., presence of LIMA graft). Three patients developed anterior cerebral neurological deficits after TEVAR (one patient with prophylactic LSA revascularisation and two without). Symptoms resolved during initial hospital stay in two of these patients, including

the one with revascularisation. The third experienced persistent memory difficulties. All three neurological events were attributed to embolic phenomena rather than hypoperfusion due to LSA exclusion. No patient suffered from paraplegia. The authors concluded that preoperative imaging predicted well the need for prophylactic LSA revascularisation, but did not eliminate the occurrence of cerebrovascular accidents (CVAs), which were most likely embolic in nature. Four other patients (20%) developed symptoms of subclavian steal syndrome or late left-arm claudication (between 3 and 26 months after TEVAR) and underwent LSA revascularisation with LSA-to-LCCA bypass. These authors prefer selective revascularisation of the LSA using preoperative imaging in case of vertebrobasilar isolation: aberrant vascular and patent internal mammary conduits to the coronaries (Table 1). They argue that their data suggest that the need for LSA revascularisation may be greater than that reported by most other groups performing these procedures.

In a single-centre, retrospective investigation by Woo et al. [46], the LSA was covered in 70 of 308 patients with TEVAR. Forty-two patients underwent elective revascularisation of the LSA, consisting of both LSA transposition ( $n = 5$ ) and LSA bypass (with ligation  $n = 3$  and with coil embolisation  $n = 34$ ). Mean follow-up was 11 months. No paraplegia developed, the stroke rate was 8.6% and no strokes were related to LSA coverage because there were no posterior strokes. Stroke rates were similar in both groups with or without revascularisation (7% vs 11%). No left-arm symptoms developed in the LSA-revascularised patients, whereas the five (18%) patients without LSA revascularisation developed symptoms in the left arm. Two required LSA revascularisation, and no permanent left-upper-extremity dysfunction or ischaemia developed in any patient. They conclude that prophylactic LSA revascularisation is not absolutely necessary except in patients with a patent LIMA bypass; however, they also mention in the article that they now perform LCCA-to-LSA bypass on all elective zone 2 TEVAR cases. Melissano et al. [44] performed TEVAR in 30 patients, 18 of whom underwent LSA ostium coverage. Prophylactic revascularisation of the LSA was done in the first three patients only. Paraplegia or neurologic sequelae were not observed in this group. Five cases of type-II endoleak were observed that resolved after prevertebral ligation of the vessel during the same procedure. This group also suggests that prophylactic revascularisation of the LSA is only necessary (1) when the contralateral VA and the branches from which it originates are not patent, (2) in the presence of a previous myocardial revascularisation with the LIMA, (3) a lusoria artery or (4) an AV shunt for haemodialysis in the left arm and (5) finally in left-handed patients. Prophylactic revascularisation of the

Table 1. Indications and considerations for LSA revascularisation in TEVAR with LSA coverage.

Indications for preoperative LSA revascularisation	Considerations for preoperative LSA revascularisation
Isolated posterior cerebral circulation	Risk for type-II endoleak from covered LSA
Dominant left vertebral artery	Potential need for LIMA for future CABG
Patent LIMA to LAD	Potential extensive intercostals coverage (paraplegia risk)
Anomalous subclavian or vertebral arteries	Younger patients
	Left-handed professionals

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CABG: coronary artery bypass grafting; LAD: left anterior descending artery; and LIMA: left internal mammary artery.

covered LSA is also recommended in cases of extensive stent covering of large parts of the thoracic aorta in combination with previous abdominal aortic surgery, to prevent paraplegia. These authors conclude that prophylactic surgical revascularisation is required for cases involving the left common carotid and innominate arteries, not for coverage of the LSA.

In an extension of their first study [47], Melissano et al. report coverage of the LSA ostium in 27 patients, 11 of whom underwent prophylactic revascularisation. They observed two cases of delayed (range 1–3 days) transitory paraparesis (which resolved by cerebral spinal fluid (CSF) drainage) in patients with extensive descending thoracic aorta involvement and LSA coverage without prior revascularisation.

### 3.3. No prophylactic LSA revascularisation

Riesenmann et al. [48] do not support prophylactic revascularisation. They described TEVAR in 24 patients with partial (10 patients, 42%) or complete (14 patients) coverage of the LSA. None underwent prior revascularisation. Mild left-upper-limb symptoms developed that did not warrant intervention in three (15.8%) patients, and rest pain developed in one (5.3%), which was treated by insertion of an LSA stent. Three CVAs (10.7%) were observed. Two occurred in patients in whom the LSA had been completely covered: one was due to embolism and the other to hypotension that occurred during an intra-operative cardiac arrest, leading to a CVA in a left frontoparietal watershed distribution. The third CVA was assessed as an embolic event in a patient with partial covering of the LSA. In this study, four of 19 patients (21%) with complete cessation of antegrade flow through the LSA experienced left-upper-extremity symptoms, but only one (5%) required an intervention. They confirmed that the 21% incidence of left-upper-extremity symptoms is consistent with previous observations after LSA coverage without revascularisation [22,30,40,44]. However, they also recommend pre-procedural imaging to evaluate the contralateral VA, carotid arteries and the presence of an intact circle of Willis.

A meta-analysis and systematic review by Cooper et al. [49] reveals a higher risk of both CVA and SCI with coverage of the LSA during TEVAR. However, regarding the higher risk of CVA after LSA coverage, they found that relatively few of the published studies reached statistical significance alone owing to insufficient numbers. In addition, they posit that CVA after TEVAR has multiple causes, thus making prophylactic revascularisation not definitively protective. However, the risk for SCI did not differ significantly between patients with no LSA coverage and those with LSA coverage and revascularisation, suggesting a protective role for prophylactic revascularisation in preventing SCI (although relevant study numbers were very low in that regard). They concluded that prophylactic revascularisation does not seem to prevent the occurrence of CVAs, but that it may reduce the risk of SCI.

In an article published 2 years earlier by the Tiesenhausen's team, Hausegger [50] presented three case reports of patients with aortic type-B dissection and one patient with a thoracic aneurysm, who underwent TEVAR during which the LSA ostium was covered. All their patients underwent TEVAR without prior surgical LSA revascularisa-

tion. None had any symptoms suggestive of a subclavian steal syndrome or left-arm ischaemia. However, left-arm radial pulses and blood pressure remained significantly reduced. The follow-up period ranged from 14 to 20 months. On follow-up duplex scanning, all four patients presented inverse flow in the left VA. Secondary transposition of the LSA to the LCCA had therefore not been required. The authors state that they rely on natural collateralisation via the left VA to sustain perfusion of the left arm after covering the LSA, supporting that position by the fact that their initial experience with LSA occlusion did not cause problems in patients undergoing TEVAR, and that the only measurable effect was a significant drop in arterial blood pressure and reversed flow in the left VA. In addition, owing to collateral flow via the VA, most patients with subclavian steal syndrome are asymptomatic, as are children with aortic coarctation in whom the LSA is transected and used as an aortic interposition graft without bypass to the distal subclavian artery stump. Furthermore, the perfusion of the spinal cord, which derives some of its blood supply from the VA, should be maintained regardless of the direction of the flow in the VA. They also emphasise the additional invasiveness and overall risk of treating TAA or dissection patients, most of whom have significant co-morbidities. Such risk is mirrored by mortality rates of 1.2–5% reported for LSA-to-LCCA bypass or LSA transposition, when performed on occlusive lesions of the LSA.

In a separate article by Görlich's working group, Pamler et al. [51] reported intentional covering of the LSA in nine of 14 patients with isolated type-B aortic dissection who underwent TEVAR. The authors did not perform any prophylactic revascularisation of the LSA before coverage. An incomplete paraparesis developed in one patient due to acute anterior spinal artery syndrome at level T10/T11. The neurological symptoms decreased within 24 h without any further management (i.e., no CSF drainage). Spiral CT revealed complete thrombosis of the false lumen from the LSA to superior mesenteric artery. Furthermore, they observed eight pairs of intercostal arteries originating from the false lumen, thereby explaining the anterior spinal artery syndrome. The authors observed no clinical signs of malperfusion of the left arm or reperfusion of the false lumen (endoleak) via the LSA in any of these nine patients, who had undergone coverage of the LSA and no prophylactic revascularisation procedures.

In their subsequent report [52], Sunder-Plassmann et al. describe the experience with TEVAR in 45 patients with aneurysms of the descending thoracic aorta in elective and emergency cases. The LSA had to be covered without prior revascularisation in 12 patients. The LSA was transposed later in one patient, who had requested the surgery. The authors carried out a pre-procedural duplex evaluation of the ipsi- and contralateral carotid circulation to rule out occlusions and stenoses, observing no cases of left-hand ischaemia, subclavian steal syndrome or clinically relevant spinal cord ischaemia. One transient neurological deficit occurred in a patient, who died 7 days later from undetected iatrogenic aortic dissection. Another patient died after 4 months from brain infarction (Table 2).

Table 2. Relevant studies concerning endograft coverage of the LSA and prophylactic LSA revascularisation: Diagnoses, surgical therapy, complications (all in number of patients) and cumulative risk (in %) for neurological complications as related to treatment option.

Authors [Ref.] (PR)	Diagnosis total patient group	Patients (n) with isolated coverage of LSA	Primary T or BP No T or BP	Sec. T or BP or LSA stent	Complications <sup>a</sup>			Type-II endoleak	Cumulative risk for neurological complications
					Stroke	Paraplegia	Arm malperfusion		
Grabenwöger [26]	TAA 19, diss. 2, other 0	9	9 T 0	0	0	0	0	0	0%
Grabenwöger [28]	TAA 2, diss. 11, traum 2, other 4	19	0 19	0 1	0 0	0 1	0 1	0 1	0% 5.2%
Heijmen [29]	TAA	5	8 T 0	0	0	0	0	4 of 28 (whole group)	0%
Czerny [32]	TAA 8, diss. 3	9	9 T 0	0	0	0	0	0	0%
Cambria [33]	TAA 18, diss. 4 (chronic), other 6	6	6 T 0	0	0	0	0	3	0%
Buth [34]	TAA 291, other 315 (inc. 67 traum)	159	40 T/BP 119	0 0	0 5	0 6	0 0	0 0	0% 9.2%
Authors [Ref.] (cond. PR)									
Criado [1]	TAA 31, diss. 16	5	0 5	0	0	0	0	0	0% 0%
Schoder [15]	TAA 32, diss. 19, traum. 4, other 3	8	0 8	0 2	0 2	0 1	0 2	0 2	0% 37.5%
Weigang [38]	TAA 10, diss. 10 (chronic 4)	11	1 T 10	0 1 T	0 2	0 0	0 0	0 0	0% 20%
Peterson [18]	TAA 15, diss. 9 (chronic 5), traum 6	30	20 T, 2 BP 8	0 1 T	0 5	1 0	0 1	0 0	4.5% 62.5%
Görich [30]	TAA 3, diss. 9, other 11	23	0 23	0 0	0 0	0 0	0 2	0 0	0% 0%
Tiesenhausen [22]	TAA 3, diss. 7	10	2 T 8	0 2 T, 1 BP	0 0	0 0	0 1	0 2	0% 0%
Rehders [40]	TAA 39, diss. 128 (all elective cases), other 4	22	0 22	0 0	0 1	0 0	0 7	0 0	0% 4.5%
Caronno [17]	TAA 3, diss. 5, other 3	11	0 11	0 0	0 1	0 0	0 0	0 0	0% 9%
Reece [45]	TAA 15, diss. 3 (chronic), other 9 (acute traum 7)	27	1 T, 6 BP 20	0 4 BP	0 3	0 0	0 3	0 0	0% 15%
Woo [46]	TAA 47, diss. 16 (chronic 1), other 7 (traum 2)	70	5 T, 37 BP 28	0 2 BP	3 3	0 0	0 5 (18%)	0 0	7% 11%
Melissano [44]	TAA 11, diss. 2, other 5	18	3 BP 15	0 0	0 0	0 0	0 0	5 0	0% 0%
Melissano [47]	TAA 53, diss.3, other 8 (traum 2)	27	2 T, 9 BP 16	0 0	0 0	0 2	0 0	0 0	0% 12.5%

Table 2. (Continued)

Authors Ref. (no PR)																									
Riesemann [48]	TAA 10, diss. 8, other 4 (traum 6)	14	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0%	0%	14.3%		
Hausegger [50]	TAA 1, diss. 3	4	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0%	0%	
Pamler [51]	diss. 14 (chronic 13)	9	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0%	0%
Sunder-Plassmann [52]	TAA 45, diss. 15 (traum 15)	12	1 T	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0%	0%
		11	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0%	0%

[Ref]: Reference #; PR: prophylactic revascularisation; TAA: thoracic aortic aneurysm; diss.: thoracic aortic dissection; traum: acute traumatic transection; inc.: inclusive; cond.: conditional; LSA: left-subclavian artery; T: transposition; BP: bypass; and Sec.: secondary.

<sup>a</sup> =both reversible and irreversible complications. Stroke = irrespective of location or aetiology. Central neurological symptoms of subclavian steal syndrome are classified as neurological brainstem complications.

#### 4. Summary

ESG placement in the proximal part of the descending aorta often requires covering the LSA ostium to extend the proximal landing zone with a minimum of 2 cm of normal aortic wall. Covering the LSA can lead to vascular and neurological complications and to type-II endoleak by retrograde perfusion from the LSA into the aneurysm sac or the dissection’s false lumen in certain patients. Several authors [6,29,32,33,38], therefore, recommend prophylactic revascularisation of the LSA before coverage in high-risk patients. Others [48,50,51] criticise every additional surgical procedure by suggesting that they add to the invasiveness and overall treatment of TAA or dissection patients [50]. In addition, they draw attention to the 1–5% mortality rates described in association with LSA-to-LCCA bypass or LSA transposition [50], although these data refer to occlusive atherosclerotic lesions of the LSA and not to LSA coverage by ESGs. Another argument in favour of a procedure without prophylactic LSA revascularisation is that LSA coverage is a well-tolerated procedure in patients with normal (no apparent angiographic lesions) supra-aortic branches [30,50]. When collateral blood flow from the muscular arterial branches in the neck and shoulder girdle [17] and via the contralateral VA [50] is adequate, most patients with subclavian steal syndrome are asymptomatic [50]. This may be difficult to maintain for incipient aortic conditions, for instance, in acute aortic dissection or some traumatic transections. After systematically probing the literature to analyse whether the hypothesis that the putative absence of a collateral network in acute aortic conditions leads to a higher frequency of neurological events with coverage of the LSA ostium is true, we found that unequivocal information on this paradigm is lacking. This is due to low patient numbers and few specifics by the authors in differentiating emergency, acute, chronic and elective cases and in reporting (neurologic) complications.

Over the past years, most authors have concluded that prophylactic LSA transposition or LSA-to-LCCA bypass surgery is advantageous. This approach demands careful preoperative evaluation by Doppler ultrasound, computed tomography angiography (CTA) or MRA of supra-aortic vascular anatomy and pathology (significant stenoses and occlusions) before deciding whether prophylactic LSA transposition or LSA-to-LCCA bypass is necessary. For instance, a study by Manninen et al. [23] addressed the risk for posterior cerebral infarction that can evolve in the presence of vertebrobasilar insufficiency following LSA coverage. They performed a post-mortem anatomic study on 62 forensic autopsy cases in which the existence and diameter of the right VA and PCOMA were assessed with craniocervical CT and MRA. In 5.4% of cases, they were able to identify a substantial risk for acute neurological complications after unprotected closure of the LSA due to the hypoplasia of the right VA above the posterior inferior cerebellar artery in conjunction with an incomplete PCOMA, and in 3.3% the risk for neurologic complications was considered possible because of isolated right VA hypoplasia. They concluded that when the LSA is closed, insufficient posterior cerebral circulation due to individual anatomy occurs relatively infrequently, but to avoid complications, careful imaging of the right VA up to the basilar artery is



mandatory, and if proven hypoplastic, PCOMAs' imaging is necessary.

Another point has been raised by Botta et al. while studying endovascular treatment for acute traumatic transection of the descending aorta in 31 patients [53]. They remark that flow compensation throughout the circle of Willis is a dynamic process, which can be influenced by the diameter of the posterior communicating arteries and vasomotor regulation which, in turn, may be influenced by cerebral oedema or general anaesthesia. In any case, we believe that these traumatic patients should undergo treatment as soon as the surgeon perceives that there is no other limiting concomitant injury more ominous than the aortic disease.

However, recent data [34,45,49] reveal that a high percentage of patients undergoing TEVAR with LSA coverage need conditional prophylactic LSA transposition or LSA-to-LCCA bypass surgery due to abnormal supra-aortic vascular anatomy or existing supra-aortic pathology. Moreover, the same studies report a significant incidence of devastating neurologic complications in the remaining non-revascularised group.

The reported incidence of stroke varies from 2% to 15% [15,18,34,38,45,46,48], whereby the causes seem multifactorial. Although a significant number of strokes occur in the posterior circulation and can be attributed to localised anatomical vascular malformations or vascular pathology directly linked to LSA coverage, still another significant number does occur in other parts of the brain and has other aetiology, for instance, hypoperfusion or emboli. It is generally known that LSA covering without prophylactic revascularisation leads to a significant higher incidence of stroke compared with LSA covering with prophylactic revascularisation. Nevertheless, a substantial part of all presenting strokes do occur in patients with TEVAR without LSA coverage, and even in some with LSA covering with prophylactic revascularisation. TEVAR patients, therefore, still present an elevated risk of perioperative stroke (due to guidewire manipulations and ESG deployment during the procedure).

SCI is another serious potential complication of TEVAR with an incidence of between 0% and 5%, although few data are available. Proposed pathophysiologic mechanisms include, for instance, LSA coverage leading to reduced blood flow to the VA and thus to the spinal cord via the spinal artery and/or to reduced anterior spinal and costocervical blood flow by coverage of intercostal vessels by one or more stent grafts. Further suggested risk factors for compromised spinal cord perfusion in TEVAR are the occlusion of intercostal arteries by the stent grafts at the T8-12 level, previous abdominal aortic surgery, occlusion of internal iliac arteries and renal failure. Overall, the risk of SCI after TEVAR is significant and is not to be underestimated because of the condition's severity.

Unfortunately, many of these neurological conditions do not resolve after secondary revascularisation. It thus may be necessary to perform prophylactic LSA transposition or LSA-to-LCCA bypass surgery in elective patients scheduled for TEVAR with LSA coverage, also because their additional surgical risk is low as they are usually free of occlusive atherosclerotic lesions of the LSA.

Subclavian steal syndrome is common after TEVAR with LSA coverage, with an incidence of 5–37.5%. Most of these patients are asymptomatic due to the flow inversion from the VA and the presence of collaterals in the neck and shoulder girdle, as symptoms, if they occur, tend to be mild and transient.

The incidence of left-arm ischaemia is reported between 0% and 36%. Symptoms are mostly transient and consist of a cooler hand, exercise-induced paraesthesias of the left arm and hand, claudication and rest pain or even distal digital trophic changes. Prophylactic revascularisation prevents such symptoms.

Overall, left-upper-extremity symptoms occur in about 20% of patients, who undergo TEVAR and LSA coverage without prior revascularisation. When one considers the high frequency of this complication together with the substantial threat of severe neurologic sequela (i.e., stroke or SCI after TEVAR with LSA coverage and without prophylactic revascularisation), a prophylactic revascularisation is strongly recommended.

By contrast, covering of the LSA without prophylactic revascularisation of the LSA is justified in acute unstable patients (e.g., patients with aortic rupture) because of the shortage of time to perform prophylactic LSA transposition or LSA-to-LCCA bypass surgery. As we anticipate the absence of a patent collateral network in those conditions, secondary revascularisation of the LSA is indicated after the emergency situation.

The LSA-to-LCCA bypass or LSA transposition procedure may itself be hazardous and there are few reports that document cases that do not proceed to TEVAR because of complications related to the preparatory LSA revascularisation. Future studies are necessary to distinguish whether the morbidity of TEVAR without prophylactic revascularisation is significantly greater than the combined morbidity of TEVAR with prophylactic revascularisation. Until databases and reports include all patients on an intention-to-treat basis, definitive evidence that one approach is superior to another is difficult to acquire. Nevertheless, such pathology-based rather than procedure-based databases would incorporate the added complication rate of the prophylactic surgical procedure in the total equation.

Finally, prophylactic LSA transposition or LSA-to-LCCA bypass surgery and TEVAR is recommended for elderly and high-risk patients not suitable for conventional open surgery. This is based on the fact that, so far, long-term results regarding integrity and durability of the stent-graft device are missing [53]. However, in traumatic aortic rupture, descending aneurysm rupture and in acute aortic dissection type-B patients with complications, where conventional surgery exhibits high mortality and morbidity rates, TEVAR is recommended as first-line therapy.

## 5. Conclusion

Prophylactic revascularisation of the LSA before coverage through a stent graft is necessary when careful preoperative imaging reveals abnormal supra-aortic vascular anatomy or supra-aortic pathology. To avoid potentially devastating neurological complications in the absence of revascularisation, elective patients undergoing LSA coverage during TEVAR

should be subjected to prophylactic LSA transposition or LSA-to-LCCA bypass surgery before TEVAR. It would appear that only in acute unstable patients immediate covering of the LSA without prophylactic revascularisation of the LSA is justified. In these cases, LSA revascularisation should be done after the emergency situation.

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