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Review

How to manage the left subclavian artery during endovascular stenting of the thoracic aorta☆

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Summary

We performed a systematic review of the literature to establish whether revascularisation of the left subclavian territory is necessary when this artery is covered by a stent. We retrieved data from 99 studies incorporating 4906 patients. Incidences of left-arm ischaemia (0.0% vs 9.2%, $p = 0.002$) and stroke (4.7% vs 7.2%, $p < 0.001$) were significantly less following revascularisation, although mortality (10.5% vs 3.4%, $p = 0.032$) and endoleak incidence (25.8% vs 12.6%, $p = 0.008$) were increased. No significant differences in spinal-cord ischaemia were seen. Revascularisation may reduce downstream ischaemic complications but can cause significant risk. Indications must be carefully considered on an individual patient basis.

Keywords: Anatomy; Aorta; Thoracic; Aortic surgery; Endovascular; Stents

1. Introduction

Thoracic aortic pathology has traditionally been treated by open surgery. The development of thoracic endovascular aortic repair (TEVAR) has introduced an attractive alternative with reported reduced morbidity and perioperative mortality [1]. Advantages such as negating the need for thoracotomy and aortic cross-clamping must be tempered by consideration of the complications. Management and, especially, stenting of the aortic arch present a specific challenge in view of the head-and-neck vessel origins because a key factor in the successful deployment of a stent is the provision of a suitable proximal landing zone (LZ), which should be at least 15–20 mm [2,3].

Endovascular management in the vicinity of the left subclavian artery (LSA) origin may necessitate incursion of that boundary to create an adequate LZ. Stents have, therefore, been deployed partially or completely across the LSA origin. The LSA is not only the main source of perfusion of the left arm but also the origin of three important branches: the left internal mammary artery (LIMA), the vertebral artery and the costocervical trunk. The LIMA is the preferred donor conduit for coronary artery bypassing. The vertebral artery supplies the posterior part of the circle of Willis with the basilar artery and also contributes to spinal-cord perfusion via the anterior spinal and posterior spinal arteries. The costocervical trunk can also contribute to spinal-cord perfusion [3].

As a result, LSA coverage has been associated with downstream ischaemic complications such as left-arm ischaemia, spinal-cord ischaemia and stroke [2–4]. Myocardial ischaemia in patients with LIMA to coronary artery bypass graft (CABG) has also been reported. However, coverage of the LSA origin has also been shown to be complication-free with no downstream ischaemic consequences [3].

To prevent or to treat coverage complications, it is possible to revascularise the LSA territory, before or after TEVAR, respectively, usually by LSA to left-carotid-artery bypass or transposition [5]. The revascularisation itself is associated with mortality and morbidity such as nerve injury, graft infection, lymphatic leakage and stroke [6].

The optimal management of the LSA in the context of TEVAR, therefore, remains unclear and guidelines do not exist, especially with regard to the revascularisation requirement.

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Different practice strategies exist with some centres electively covering the LSA in isolation without performing surgical revascularisation [3]. This may be appropriate in some but not all patients because complications occur. Specific indications where collateral prior revascularisation must be performed are recognised. Examples include patients with previous LIMA conduits for CABG, left-handed professionals, dialysis patients with left-arm arteriole-venous shunts, patients with anatomical variations such as a common origin of the LSA and left common carotid artery and those with vertebral or carotid artery stenosis [7]. In addition, direct involvement of the LSA in the underlying aortic pathology, such as aneurysmal disease, may oblige pre-emptive revascularisation. Other centres perform revascularisation if deemed necessary as a second step on a 'wait-and-see' basis in response to ensuing coverage complications such as signs of ischaemia or malperfusion.

The question remains as to who should receive which treatment and when. Recent studies addressing this controversy have been published though this study represents the largest quantitative and comprehensive approach to date [2,4,8].

1.1. Study aims

Aims were to establish the evolution of clinical practice regarding LSA coverage and revascularisation and to compare outcomes in patients with LSA coverage with and without LSA revascularisation. We also aimed to identify the complications of LSA revascularisation and their incidence. Our final goal was to try to develop an evidence-based approach to management of the LSA during thoracic-aortic stenting.

2. Material and methods

2.1. Literature search

An extensive multilayer literature search using a broad comprehensive searching protocol was performed to ensure capture of as many relevant studies as possible. First, Medline, Ovid, Embase, Cochrane and the UK National Library for Health databases were searched for all relevant studies up to and including November 2008 using the following MeSH search headings: 'subclavian artery/blood supply' OR 'subclavian artery/surgery' AND 'aorta, thoracic/surgery' AND 'complications' OR 'intraoperative complications' OR 'postoperative complications'. Searches were also performed using the terms: *Subclavian artery AND thorac* AND (stent* OR graft* OR endovasc*). Abstracts of all articles resulting from the search were reviewed. The search was then broadened by reviewing abstracts of 'related articles'. Finally, the references of all recent review articles were considered. In this way, we identified articles not brought to our attention by the keyword search.

2.2. Study selection and data extraction

Two reviewers (Syed Rehman and Ryan Perera) independently extracted the data from each study using a predefined protocol. Studies were selected according to eligibility, exclusion criteria and where primary outcomes, as defined below, were available. Data extracted included: first author, year of publication, study population characteristics, study design, number of LSA covered, number of LSA revascularised before thoracic-aortic stenting, stenting indication, urgency, complications in patients without LSA coverage, with LSA coverage with and without pre-stenting LSA revascularisation and complications due to LSA revascularisation.

Initially, all titles and abstracts (and full text where abstracts were unavailable) were reviewed. All full texts were then reviewed to generate the final study group (see Fig. 1).

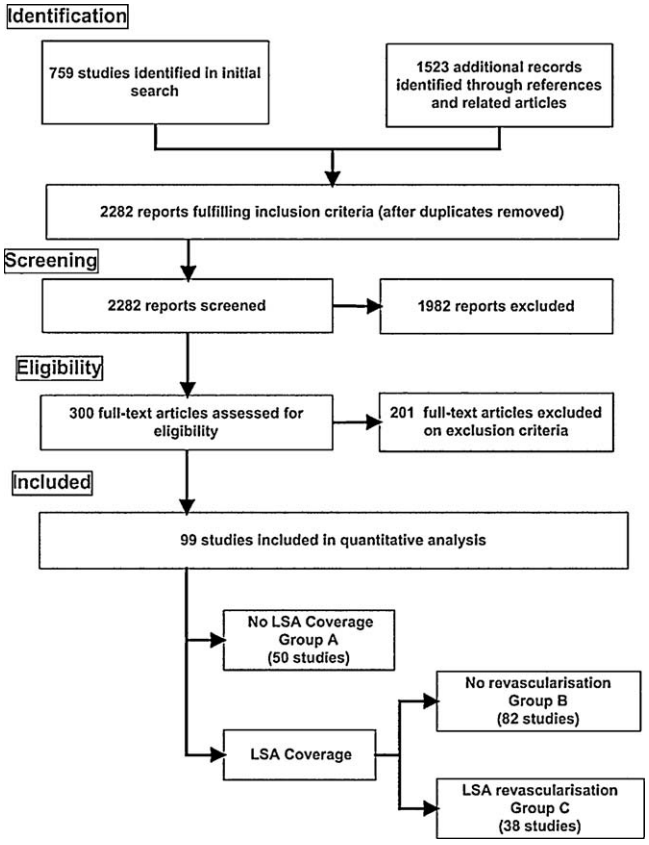

Fig. 1. Search strategy and outcome.

2.3. Eligibility criteria

We included all studies in which the LSA was covered by thoracic-aortic stents for any aortic pathology. Further, all studies in which the LSA (Zone 2) was covered with or without revascularisation, which reported on primary or secondary outcomes as defined below, were reported. (To facilitate LZ selection, Ishimaru described a classification system where each zone (Z0—Z4) is bordered by tangents aligned with the distal sides of each orifice of the arteries branching off the aortic arch.) [9].

2.4. Exclusion criteria

We excluded studies where the predefined primary or secondary outcomes were not extractable, studies in which thoracic-aortic-stent insertion was not radiologically guided

or where fenestrated or branched stent grafts were used, studies with a patient population less than five and studies not published in the English language. Where there was overlap in data in studies reported by the same institution, the analysis included either the better quality or more recent publication and the 'other' study was excluded.

2.5. Primary outcomes

These included left-arm ischaemia (which was defined as critical arm ischaemia and left-arm claudication symptoms but not an asymptomatic reduction in blood pressure in the left arm), stroke, spinal-cord ischaemia (which was defined as paraplegia and paraparesis), endoleak (including all types I–IV), stent migration and overall mortality.

2.6. Secondary outcomes – complications of LSA revascularisation

Secondary outcomes were nerve injury (including Horner's syndrome, partial plexus palsy, phrenic nerve palsy and recurrent laryngeal nerve palsy), lymphatic leak, post-operative thrombosis, graft infection, haematoma and other.

2.7. Group comparison and sensitivity analysis

All study participants were classified into three defined study groups (see Fig. 1) enabling comparison of outcomes: group A who had with no LSA coverage, group B who underwent LSA coverage without LSA revascularisation and group C who underwent LSA coverage with LSA revascularisation.

Statistical analysis was performed on all studies for each outcome comparing groups A, B and C (see Tables 2–4). There were three stages of analysis. The first stage was to examine the total incidence of a specific outcome between the two groups. To examine this, we recorded the total incidence of the complication for each group. We classified these as 'all studies' and report the tabulated findings with the number of studies shown where the incidence was reported. The second stage was to refine this search further by selecting only those studies where a direct comparison was made of the outcome between the two groups. In this way, studies where the incidence was reported in only one group were rejected. The remaining studies were classified as 'comparative studies' and are tabulated with the respective number of studies and subjects. The third stage of analysis was to perform a subgroup analysis (sensitivity analysis) on studies published before and after 2004 to determine if developments in endovascular stenting in the last 5 years have impacted on the selected outcomes. A subgroup analysis was also performed to compare outcomes according to the underlying aortic pathology (dissection, aneurysm and trauma) and degree of clinical urgency.

Through performing this sensitivity analysis, an assessment of the risk of bias was made at study level. This approach not only enabled selection of only higher quality studies, consequently reducing the risk of selection bias, but also provided a mechanism for assessing the robustness of results under different circumstances.

Statistical analysis was performed using the epidemiological software Epi Info™ Version 3.5.1 (Centres for Disease Control and Prevention, Atlanta, GA, USA). Statistical significance was achieved at $p < 0.05$. Statistical tests performed were chi-square with or without Yate's correction.

3. Results

The initial MeSH term search generated 759 studies. A further 1523 studies were identified from references and related articles of the original search. Of these studies, 1982 studies were excluded. On expanding to full-text review, a further 201 studies were excluded. This resulted in a final study group of 99 publications, which constituted 'all studies' (see Fig. 1). From this, a variable number of studies (ns) were excluded, generating groups of 'comparative studies' for specific subgroup analysis. Demographic and clinical data are presented in Table 1. In total, there were 4906 patients with at least 1607, who were LSA covered.

Table 1. Studies selected with mean age, LSA management strategy and stenting indication.

Study	Mean age	LSA management		Stenting indication						
		Covered	Revascularised before stenting	AD	AA	AA rupture	PAU	IMH	Trauma	Other
Buth et al.[a] [15]	63.2	159	40	215	317	0	0	0	67	7
Thompson et al.[a] [13]	64	56	14	67	88	0	3	1	21	0
Feezor et al.[a] [16]	68.6	80	11	34	103	0	32	0	9	18
Farber and Criado[a] [17]	66.5	9	0	11	0	11	0	0	0	0
Woo et al.[a] [18]	67	70	42	16	44	5	0	0	4	1
Morales et al.[a] [11]	71	66	0	52	116	0	0	0	10	8
Khoynezhad et al.[a] [19]	71	43	2	67	91	0	14	0	12	0
Schoder et al.[a] [20]	61.8	58	25	20	29	3	2	0	4	0
Melissano et al.[a] [21]	71	30	5	2	24	0	2	0	2	0
Peterson et al.[a] [5]	62	30	22	5	45	0	0	6	14	0
Morasch and Peterson [22]	NS	28	21	NS	NS	NS	NS	NS	NS	NS
Reece et al. [23]	60.4	27	7	3	17	0	0	0	7	0
Ferreira et al.[a] [24]	NS	17	4	NS	NS	NS	NS	NS	NS	NS
Appoo et al.[a] [25]	73.1	20	20	0	99	0	0	0	0	0
Steingruber et al.[a] [26]	38.7	20	0	0	0	0	0	0	22	0
Weigang et al.[a] [27]	64.3	20	1	10	9	1	0	0	0	0
Chung et al.[a] [28]	46	17	0	0	0	0	0	0	26	0

Table 1 (*Continued*)

Study	Mean age	LSA management		Stenting indication						
		Covered	Revascularised before stenting	AD	AA	AA rupture	PAU	IMH	Trauma	Other
Scharrer-Pamler et al. [29]	69	15	1	0	34	11	0	0	0	0
Alsac et al.[a] [30]	45	13	0	0	0	0	0	0	28	0
Sunder-Plassmann et al. [31]	69	12	0	0	30	15	0	0	0	0
Czerny et al. [32]	72.3	11	11	3	8	0	0	0	0	0
Galili et al.[a] [33]	NS	11	2	0	10	1	0	0	0	0
Melissano et al. [34]	71.4	10	0	0	21	0	0	0	0	0
Teisenhausen et al.[a] [35]	NS	10	2	7	3	0	0	0	0	0
Buz et al. [36]	36	20	1	0	0	0	0	0	39	0
Pamler et al. [37]	60.3	9	0	14	0	0	0	0	0	0
Reisenman et al. [38]	70	9	0	4	35	7	2	0	2	0
Schumacher et al. [39]	65	25	25	2	23	NS	0	0	0	0
Bent et al. [40]	43.2	8	0	0	0	0	0	0	13	0
Orend et al. [41]	NS	8	0	0	0	0	0	0	8	0
Schoder et al.[a] [42]	71.6	12	8	0	28	0	0	0	0	0
Schumacher et al. [43]	71	8	8	2	0	6	0	0	0	0
Hughes et al. [44]	63	13	0	1	12	0	0	0	0	0
Kutty et al. [45]	36	9	6	0	9	0	0	0	0	0
Lambrechts et al.[a] [46]	64	7	1	11	12	0	0	0	3	0
Matravers et al. [47]	71	7	NS	9	10	1	3	0	1	0
Pearce et al.[a] [48]	61	7	0	15	0	0	0	0	0	0
Rousseau et al. [49]	37	7	0	0	0	0	0	0	9	0
Tse et al.[a] [50]	73.2	7	5	6	18	2	0	8	3	0
Midulla et al.[a] [51]	47.6	6	3	0	8	0	0	0	0	0
Dagenais et al. [52]	63.3	6	0	2	11	0	6	0	4	1
Fattori et al.[a] [53]	39.4	6	0	0	0	0	0	0	19	0
Fu et al. [54]	47	6	4	9	1	0	0	0	0	0
Lawlor et al.[a] [55]	42.3	6	1	0	0	0	0	0	7	0
Neschis et al. [56]	40	6	0	0	0	0	0	0	20	0
Czerny et al. [57]	79.5	5	5	0	5	0	0	0	0	0
Destrieux-Garnier et al. [58]	62	5	0	17	5	0	5	0	5	0
Ferrari et al. [59]	40.7	4	0	0	0	0	0	0	18	0
Hausegger et al. [60]	NS	5	1	5	0	0	0	0	0	0
McPhee et al.[a] [61]	30.8	4	0	0	0	0	0	0	8	0
Yamane et al. [62]	39	4	0	0	0	0	0	0	14	0
Bockler et al. [63]	66	4	2	7	17	4	0	0	0	0
Czermak et al. [64]	67	3	NS	7	0	0	0	0	0	0
Daenen et al.[a] [65]	46.9	3	1	0	0	0	0	0	7	0
Ianelli et al.[a] [66]	58.3	3	0	8	0	4	0	0	3	0
Inglese et al.[a] [67]	69.3	3	0	6	24	5	4	0	1	1
Amabile et al. [68]	32	2	0	0	0	0	0	0	9	0
Balzer et al.[a] [69]	61.3	6	0	8	9	7	0	0	2	0
Chan et al.[a] [70]	64.8	16	2	6	9	1	0	0	0	0
Czerny et al. [71]	63	NS	4	6	0	0	0	0	0	0
Kato et al.[a] [72]	56	2	0	0	0	0	0	0	10	0
Matsumura et al.[a] [73]	72	NS	NS	0	137	0	23	0	0	0
Pitton et al. [74]	53.9	20	0	13	8	1	2	0	9	4
Saratzis et al. [75]	29.5	2	0	0	0	0	0	0	9	0
Teisenhausen et al. [76]	70	3	1	4	0	0	0	0	0	0
Brueck et al. [77]	62	NS	2	5	4	0	0	0	0	0
Fattori et al. [78]	40.6	8	2	0	0	0	0	0	51	0
Gonzalez-Fajardo et al. [79]	57	12	0	12	0	0	0	0	0	0
Grabenwoger et al. [80]	60	NS	NS	11	0	0	6	0	2	0
Schoder et al.[a] [81]	70.6	2	0	0	0	0	8	0	0	0
Orend et al. [82]	34	NS	NS	0	0	0	0	0	11	0
Neuhauser et al. [83]	39	8	0	0	0	0	0	0	13	0
Amabile et al. [84]	66	8	3	17	26	0	5	0	18	1
Apple et al.[a] [85]	72	8	6	1	21	0	2	0	3	0
Attia et al.[a] [86]		2	0	6	20	0	3	0	11	0
Bergeron et al. [87]	71.5	25	0	11	14	0	0	0	0	0
Bockler et al.[a] [88]	57	13	1	37	0	0	0	0	0	0
Botta et al.[a] [89]	71.8	1	1	0	0	0	19	0	0	0
Buffolo et al.[a] [90]		14		120	61	0	6	0	4	0
Czerny et al.[a] [71]	71.8	66	66	0	79	0	0	0	0	0
Di Tommaso et al.[a] [91]	63.5	10	0	26	18	0	0	0	7	0
Dick et al. [92]	68.8	13	0	37	9	0	0	0	0	6
Eggebrecht et al.[a] [93]	62.2	13	0	38	0	0	0	0	0	0
Go et al.[a] [94]	44	2	0	0	0	0	0	0	10	0
Go et al.[a] [95]	71	29	28	0	142	0	0	0	0	0
Gorich et al. [96]	51	23	0	9	3	11	0	0	0	0
Kokotsakis et al.[a] [97]	26.2	2	0	0	0	0	0	0	22	0

Table 1 (*Continued*)

Study	Mean age	LSA management		Stenting indication						
		Covered	Revascularised before stenting	AD	AA	AA rupture	PAU	IMH	Trauma	Other
Marcheix et al.[a] [98]	40	9	0	0	0	0	0	0	33	0
Marcheix et al.[a] [99]	68	6	0	0	45	0	0	0	0	0
Midgley et al.[a] [100]	43.8	8	0	0	0	0	0	0	12	0
Palma et al.[a] [101]	57.6	14	0	58	0	0	6	6	0	0
Patel et al.[a] [102]	67.4	16	3	0	0	0	0	0	0	0
Pauls et al.[a] [103]	74	4	0	0	0	0	12	0	0	0
Piffaretti et al.[a] [104]		17	1	13	14	0	12	0	8	0
Rodriguez et al. [105]	72	37	13	82	183	0	34	0	11	14
Sandroussi et al.[a] [106]	61.5	21	0	23	31	0	2	0	9	0
Schoder et al.[a] [107]	57	26	4	0	0	0	0	0	0	0
Xu et al.[a] [108]	50.4	16	0	63	0	0	0	0	0	0
Yang et al.[a] [109]	48.4	10	0	0	0	0	0	0	0	0
Total	–	1561	438	1243	2139	96	213	21	674	61

LSA: left subclavian artery; AD: aortic dissection.; AA: aortic aneurysm; PAU: penetrating aortic ulcer; IMH: intramural haematoma; NS: not stated.
[a] Comparative study.

The primary outcome findings of the comparison among groups A, B and C are presented in Tables 2–4. These tables demonstrate the results of subgroup analyses for the primary outcomes utilising 'all studies', 'comparative studies' and pre- and post-2004.

3.1. Group A versus group B — comparison of primary outcomes between no LSA coverage and LSA coverage without revascularisation (Table 2a and b)

Left-arm ischaemia was increased throughout all groups; 'all studies' ($p = 0.000$), 'comparative studies' ($p < 0.001$), pre-2004 ($p = 0.008$) and post-2004 ($p < 0.001$). The incidence of stroke was also greater in 'all studies' ($p = 0.076$) and post-2004 studies only ($p = 0.049$). The incidence of endoleak was increased in 'all studies' ($p = 0.066$) and, especially, pre-2004 ($p = 0.035$).

When covering the LSA without revascularisation, the incidence of spinal-cord ischaemia was significantly reduced in 'comparative studies' ($p = 0.017$).

3.2. Group A versus group C — comparison of primary outcomes between no LSA coverage and LSA coverage with revascularisation (Table 3a and b)

In those undergoing coverage with revascularisation, the incidence of stroke was elevated in analysing 'all studies' only ($p = 0.013$). The incidence of endoleak was greater in 'all studies', 'comparative studies' ($p < 0.001$ and $p = 0.010$, respectively) and after 2004 ($p = 0.002$). The incidence of mortality was also increased ($p = 0.003$) in 'all studies' when revascularising the LSA.

There was no statistically significant difference in spinal-cord ischaemia between the groups ($p = 0.51$) though there

Table 2. (a) Comparison of primary outcomes between no LSA coverage and LSA coverage without revascularisation. (b) Subgroup analysis before and after 2004.

Outcome	All studies							Comparative studies					
	A(o)	A(n)	ns	B(o)	B(n)	ns	*p* value	A(o)	A(n)	B(o)	B(n)	ns	*p* value
Left-arm ischaemia	0 (0.0)	955	29	59 (9.2)	640	68	0.000	0 (0.0)	472	8 (4.8)	168	24	0.000
Stroke	68 (3.6)	1901	39	35 (5.1)	683	52	0.076	47 (3.3)	1434	20 (4.4)	458	33	0.27
Spinal-cord ischaemia	35 (2.4)	1456	40	16 (3.0)	540	46	0.48	14 (3.7)	378	21 (1.7)	1250	34	0.017
Endoleak	7 (6.1)	115	10	25 (12.6)	198	16	0.066	1 (2.5)	40	3 (5.6)	54	6	0.84
Stent migration	0 (0.0)	54	7	2 (1.8)	112	11	0.82	0 (0.0)	50	0 (0.0)	25	7	–
Mortality	1 (0.8)	129	10	7 (3.4)	207	20	0.25	1 (0.8)	129	0 (0.0)	72	10	1.00

Outcome	Pre-2004							Post-2004					
	A(o)	A(n)	ns	B(o)	B(n)	ns	*p* value	A(o)	A(n)	ns	B(o)	B(n)	ns	*p* value
Left-arm ischaemia	0 (0.0)	132	8	7 (6.7)	104	17	0.008	0 (0.0)	555	22	55 (10.3)	532	52	0.000
Stroke	0 (0.0)	66	5	0 (0.0)	38	7	–	68 (3.9)	1731	34	36 (5.8)	618	44	0.049
Spinal-cord ischaemia	2 (0.6)	316	10	1 (1.0)	99	12	1.00	33 (2.3)	1416	32	16 (3.3)	480	36	0.23
Endoleak	1 (3.1)	32	3	11 (22.9)	48	6	0.035	6 (7.2)	83	7	18 (10.9)	165	11	0.36
Stent migration	0 (0.0)	45	4	1 (2.9)	35	6	0.89	0 (0.0)	13	3	1 (1.3)	77	6	1.00
Mortality	0 (0.0)	41	3	2 (4.4)	45	5	0.52	1 (1.1)	88	7	6 (3.2)	185	16	0.54

A(o): number (% incidence) of patients without LSA coverage who experienced outcomes listed; A(n): total patients without LSA coverage; B(o): number (% incidence) of patients with LSA coverage without pre-stenting revascularisation who experienced outcomes listed; B(n): total patients with LSA coverage without pre-stenting revascularisation; ns: number of studies used in analysis.

Table 3. (a) Comparison of primary outcomes between no LSA coverage and LSA coverage with revascularisation. (b) Subgroup analysis before and after 2004.

Outcome	All studies							Comparative studies					
	A(o)	A(n)	ns	C(o)	C(n)	ns	*p* value	A(o)	A(n)	C(o)	C(n)	ns	*p* value
Left-arm ischaemia	0 (0.0)	955	29	0 (0.0)	113	20	–	0 (0.0)	206	0 (0.0)	91	10	–
Stroke	68 (3.6)	1901	39	24 (7.1)	340	27	0.013	40 (3.5)	1149	12 (5.7)	210	18	0.12
Spinal-cord ischaemia	35 (2.4)	1456	40	3 (1.4)	213	21	0.51	16 (2.5)	643	1 (0.8)	125	12	0.40
Endoleak	7 (6.1)	115	10	24 (25.8)	93	11	0.000	1 (2.9)	35	3 (42.9)	7	3	0.010
Stent migration	0 (0.0)	54	7	0 (0.0)	37	5	–	0 (0.0)	5	0 (0.0)	2	2	–
Mortality	1 (0.8)	129	10	9 (10.5)	86	11	0.003	0 (0.0)	42	0 (0.0)	1	2	–

Outcome	Pre-2004							Post-2004					
	A(o)	A(n)	ns	C(o)	C(n)	ns	*p* value	A(o)	A(n)	ns	C(o)	C(n)	ns	*p* value
Left-arm ischaemia	0 (0.0)	132	8	0 (0.0)	18	5	–	0 (0.0)	555	22	0 (0.0)	95	15	–
Stroke	0 (0.0)	66	5	1 (5.9)	17	3	0.46	68 (3.9)	1731	34	19 (5.9)	323	24	0.11
Spinal-cord ischaemia	2 (0.6)	316	10	0 (0.0)	18	5	1.00	33 (2.3)	1416	32	3 (1.6)	184	16	0.74
Endoleak	1 (3.1)	32	3	1 (11.1)	9	2	0.92	6 (7.2)	83	7	23 (24.5)	94	9	0.002
Stent migration	0 (0.0)	45	4	0 (0.0)	9	2	–	0 (0.0)	13	3	0 (0.0)	28	3	–
Mortality	0 (0.0)	41	3	1 (12.5)	8	1	0.36	1 (1.1)	88	7	8 (10.3)	78	11	0.025

A(o): number (% incidence) of patients without LSA coverage who experienced outcomes listed; A(n): total patients without LSA coverage; C(o): number (% incidence) of patients with LSA coverage with pre-stenting revascularisation who experienced outcomes listed; C(n): total patients with LSA coverage with pre-stenting revascularisation; ns: number of studies used in analysis.

was a relative reduction of 42% when performing LSA revascularisation.

3.3. Group B versus group C — comparison of primary outcomes between LSA coverage without and with revascularisation, respectively (Table 4a and b)

When comparing LSA coverage with and without revascularisation, significant differences were seen in the incidence of left-arm ischaemia, which was reduced by revascularisation in 'all studies', 'comparative studies' and after 2004 (all p values = 0.002). Stroke incidence was also relatively reduced when analysing 'comparative studies' only ($p = 0.007$).

However, revascularisation was also related to a significant increase in the incidence of endoleak in 'all studies' ($p = 0.008$) and after 2004 ($p = 0.004$) and mortality in 'all studies' ($p = 0.032$) and post-2004 ($p = 0.021$).

No significant differences in spinal-cord ischaemia ($p = 0.33$) were seen though there was a relative reduction of 53% when revascularising the LSA territory.

3.4. Secondary outcomes — complications of LSA revascularisation

The secondary outcome findings of all studies that reported LSA revascularisation complications are summarised in Table 5. In total, this included 278 patients. The overall complication rates were nerve injury 8.6%, lymphatic leak 2.5%, postoperative thrombosis 1.1%, graft infection 0.0%, haematoma 0.4%, haemorrhage 1.1%, wound dehiscence 0.4%, stroke 0.7% and mortality 0.0%.

Table 4. (a) Comparison of primary outcomes between LSA coverage without and with revascularisation respectively. (b) Subgroup analysis before and after 2004.

Outcome	All studies							Comparative studies					
	B(o)	B(n)	ns	C(o)	C(n)	ns	*p* value	B(o)	B(n)	C(o)	C(n)	ns	*p* value
Left-arm ischaemia	59 (9.2)	640	68	0 (0.0)	113	20	0.002	17 (10.2)	167	0 (0.0)	103	17	0.002
Stroke	35 (5.1)	683	52	24 (7.1)	340	27	0.21	30 (7.2)	415	12 (4.7)	257	22	0.007
Spinal-cord ischaemia	16 (3.0)	540	46	3 (1.4)	213	21	0.33	7 (3.4)	207	1 (0.8)	123	13	0.27
Endoleak	25 (12.6)	198	16	24 (25.8)	93	11	0.008	7 (10.3)	68	11 (31.4)	35	5	0.33
Stent migration	2 (1.8)	112	11	0 (0.0)	37	5	1.00	0 (0.0)	21	0 (0.0)	4	3	–
Mortality	7 (3.4)	207	20	9 (10.5)	86	11	0.032	0 (0.0)	71	0 (0.0)	32	6	–

Outcome	Pre-2004							Post-2004					
	B(o)	B(n)	ns	C(o)	C(n)	ns	*p* value	B(o)	B(n)	ns	C(o)	C(n)	ns	*p* value
Left-arm ischaemia	7 (6.7)	104	17	0 (0.0)	18	5	0.56	55 (10.3)	532	52	0 (0.0)	95	15	0.002
Stroke	0 (0.0)	38	7	1 (5.9)	17	3	0.68	36 (5.8)	618	44	19 (5.9)	323	24	0.97
Spinal-cord ischaemia	1 (1.0)	99	12	0 (0.0)	18	5	1.00	16 (3.3)	480	36	3 (1.6)	184	16	0.36
Endoleak	11 (22.9)	48	6	1 (11.1)	9	2	0.73	18 (10.9)	165	11	23 (24.5)	94	9	0.004
Stent migration	1 (2.9)	35	6	0 (0.0)	9	2	1.00	1 (1.3)	77	6	0 (0.0)	28	3	1.00
Mortality	2 (4.4)	45	5	1 (12.5)	8	1	0.94	6 (3.2)	185	16	8 (10.3)	78	11	0.021

B(o): number (% incidence) of patients with LSA coverage without pre-stenting revascularisation who experienced outcomes listed; B(n): total patients with LSA coverage without pre-stenting revascularisation; C(o): number (% incidence) of patients with LSA coverage with pre-stenting revascularisation who experienced outcomes listed; C(n): total patients with LSA coverage with pre-stenting revascularisation; ns: number of studies used in analysis.

Table 5. Secondary outcomes – complications of LSA revascularisation.

Author	Patients (total, % revascularised)	M:F	Age (mean, range)	Complications					
				Nerve injury	Lymphatic leak	Postoperative thrombosis	Graft infection	Haematoma	Other
Domenig et al.[a] [6]	150, 100	76:74	60.2, –	18	5	3	0	1	5
Woo et al. [18]	70, 63	53:17	67, –	1	0	0	0	0	0
Saleh [110]	16, 38	12:4	67, 45–82	0	1	0	0	0	0
Brueck et al. [77]	9, 22	5:4	62, 44–70	0	0	0	0	0	0
Czerny et al. [32]	11, 100	7:4	72.3, –	0	0	0	0	0	1
Peterson et al. [5]	70, 31	44:26	62, 22–85	2	0	0	0	0	0
Schoder et al. [20]	58, 47	45:13	61.8, 21–84	2	0	0	0	0	0
Cambria et al. [111]	28, 21	16:12	71, 36–91	0	0	0	0	0	0
Criado et al. [112]	47, 17	33:14	–, 33–88	0	1	0	0	0	0
Yano et al. [113]	50, 4	–	–, –	1	0	0	0	0	0

M:F: male:female. Other complications in Domenig et al. [6] included two strokes and three haemorrhages and in Czerny et al. [32] included one wound dehiscence.
[a] *Note*: In this study only 26 out of the 150 patients underwent thoracic aortic endovascular stenting.

Table 6. Aortic pathology and outcome.

Outcome	A(o)	A(n)	ns	B(o)	B(n)	ns	*p* value
Discussion							
Left-arm ischaemia	0 (0.0)	161	22	4 (4.0)	101	14	0.043
Stroke	2 (1.4)	142	10	9 (9.0)	100	15	0.013
Endoleak	2 (4.0)	50	10	12 (29.3)	41	9	0.002
Aneurysm							
Left-arm ischaemia	0 (0.0)	87	8	5 (8.6)	58	12	0.020

Please note that only statistically significant findings are shown. A(o): number (% incidence) of patients without LSA coverage who experienced outcomes listed; A(n): total patients without LSA coverage; B(o): number (% incidence) of patients with LSA coverage without pre-stenting revascularisation who experienced outcomes listed; B(n): total patients with LSA coverage without pre-stenting revascularisation; ns: number of studies used in analysis.

3.5. Time-trend analysis in LSA coverage and revascularisation

Chronological trends in LSA coverage with and without revascularisation from 1997 to 2008 are demonstrated in Fig. 2.

3.6. Underlying aortic pathology and outcome

Comparison of groups A, B and C according to three different aetiologies (dissection, aneurysm and trauma) demonstrated that in the case of dissection, the incidences of left-arm ischaemia, stroke and endoleak were all increased when covering the LSA in comparison to leaving the origin uncovered ($p = 0.043$, 0.013 and 0.002, respectively; see Table 6). The incidence of left-arm ischaemia was also elevated in those with aneurysms when covering the LSA origin ($p = 0.020$). No other statistically significant findings were demonstrated. Therefore, those with dissections (and less so aneurysms) are more likely to develop LSA coverage complications.

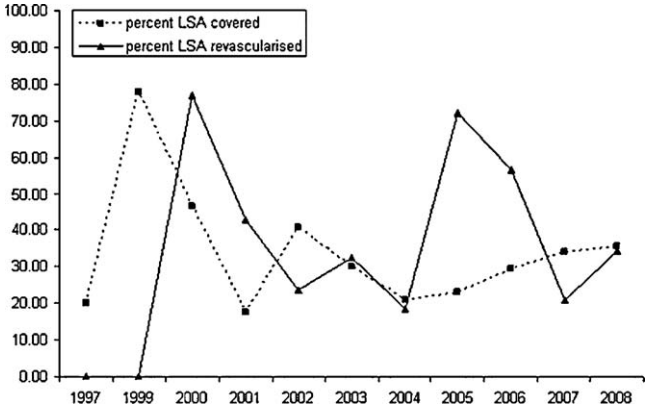

Fig. 2. Trends in LSA coverage and LSA revascularisation.

3.7. Clinical urgency

We assessed all studies to assess for a relationship between clinical urgency and primary outcome. The only statistically significant finding was for stroke where we found that this outcome was more likely in elective patients than emergencies (4.5% vs 0.7%, $p = 0.004$).

3.8. Types of endoleaks

We performed a subgroup analysis to compare the incidence of different types (I and II) of endoleaks among groups A, B and C. The results were similar as those for endoleaks in general, except that the increase in incidence of type I endoleaks in group C compared with group B was not statistically significant ($p = 0.34$).

4. Discussion

We established chronological trends in coverage and revascularisation rates (Fig. 2) such that, initially, LSA coverage was very high (1999) closely followed by a

revascularisation peak (2001). Trends then returned to baseline until, in 2005, another revascularisation surge was seen. By 2008, there were fairly equal coverage and revascularisation incidences, suggesting a relative increase in revascularisation uptake. This variation represents a lack of consensus for optimal LSA management during TEVAR. Potential influencing factors include varying surgical preferences and institution of guidelines (or, in this case, lack of them).

The impact of LSA coverage without revascularisation, as expected, demonstrated a significant increase in left-arm ischaemia (9.2% vs 0.0%). This complication was obliterated by revascularisation.

LSA coverage *per se* with or without revascularisation was also found to increase the risk of stroke by 50% and 30% in comparison to no LSA coverage. When revascularising, the risk of stroke was relatively reduced by 30% with analysis of 'comparative studies' only demonstrating a statistically significant reduction in the incidence of stroke.

The relative incidence of spinal-cord ischaemia was also found to be reduced after revascularisation though this was not statistically significant. There was a relative reduction seen of 53% from 3.0% to 1.4%.

Important negative implications of revascularisation were revealed in that the incidence of mortality and endoleak were significantly increased. The rate of endoleak increased from 13% to 26% (relative increase of 51%) when undergoing LSA revascularisation. We expected to see a reduction in stent migration in favour of LSA origin coverage in view of the improved LZ; however, our results did not corroborate this. This may be explained by the small number of patients reported (35 vs 9 patients). Mortality, however, was significantly different between the groups with a relative increase of 68% when undergoing LSA revascularisation.

In addition, revascularisation was associated with further independent complications. Nerve injuries were observed in 8.6% (24 out of 278 patients). However, 75% of these were transient and resolved within 6 months of surgery [6]. The rates of the other minor complications were all minimal.

We also demonstrated that in considering the underlying aortic pathology where LSA coverage was required, those with dissection (and, to a lesser extent, aneurysms) were more likely to develop complications. We found no statistically significant data to demonstrate that revascularisation was better or worse for differing aortic aetiologies. Furthermore, the urgency influenced only the outcome of stroke and the incidence of this complication was elevated in the elective cases.

These conclusions provide the current available evidence base to facilitate decision making in LSA management during TEVAR. When it is necessary to cover the LSA origin to facilitate the LZ, subsequent revascularisation reduces the incidence of left-arm ischaemia and stroke. However, this intervention may also be associated with an increase in the rate of mortality, endoleak and morbidity. This precludes a 'blanket statement' approach to LSA management and necessitates a thorough understanding of each clinical scenario. We propose that proficient decision making in LSA management requires a multifactorial thought process to calculate an appropriate risk–benefit ratio, which may be divided into patient, operator and hospital factors.

4.1. Patient factors

This includes assessment of the individual's specific anatomy and consideration of the underlying pathology in the context of its clinical urgency.

Anatomical assessment of the individual enables planning the endovascular approach and mapping out the collateral anatomy enabling prediction of downstream ischaemia. Direct imaging of the aorta, and carotid and vertebral arteries by ultrasound duplex, computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) are available options [10,11]. Specific posterior cerebral circulation layouts preclude the risk of stroke on occluding the dominant artery and Feevor and Lee describe that where they identified specific at-risk posterior circulations and performed 'expectant' revascularisation, they reduced their stroke rate from 6.4% to 2.3% [2].

In addition, LSA occlusion tests also enable prediction of the consequences of LSA coverage. This involves performing neurological tests while the LSA is temporarily occluded using a balloon catheter to demonstrate potential complications such as left arm, cerebellar, brain stem or spinal-cord ischaemia [12]. Kurimoto et al. employed these tests to demonstrate symptoms of vertebro-basilar insufficiency and vertebral artery abnormalities in 6.5% and 6.7% of patients, respectively. As a result, they performed revascularisation surgery or fenestrated stent grafting in these patients to preserve LSA perfusion and successfully prevent the occurrence of stroke [12]. In addition, the individual presentation must be considered to ensure that there are no specific influencing factors such as a previous LIMA harvest for CABG. The underlying aortic pathology and clinical urgency did not appear to significantly influence the outcome, according to our results, despite the expectation that the extent of aortic dissection may influence the rate of spinal-cord ischaemia due to varying degrees of interruption of spinal-cord perfusion [13].

4.2. Operator factors

Individual surgeons have preferences as to how they implement their clinical practice and are influenced by the evidence base for specific interventions and their personal experience and training pathway.

4.3. Hospital factors

Facilities need to be in place to enable a multidisciplinary approach; hence, 'hospital factors' are a further important consideration. In addition, the cost–benefit ratio and available budget must be considered because the added cost of revascularisation surgery is considerable. The mean hospital stay for patients with and without pre-stenting LSA revascularisation, where data were available, was 14.9 days and 10.9 days, respectively. It would be useful to perform a cost-effectiveness analysis to evaluate this for consideration in decision making of management of patients with LSA coverage.

Stent-graft type and availability are also influential. We expect that the type of stent graft used (fenestrated/branched) may also affect outcome though the different

degrees of aortic manipulation associated with their insertion may confound these findings. We excluded graft type as a distinction from the study because none of the studies included in the analysis used fenestrated or branched grafts. This turned out to be beneficial because this has enabled the focus to be on approved techniques/devices and not of investigational tools and experimental techniques.

Combining these findings, we emphasise that LSA management decision making requires careful considered action. There are currently no Agency for Healthcare Research and Quality (AHRQ, USA) or National Institute for Health and Clinical Excellence (NICE, UK) guidelines available. If LSA coverage is necessary to create an optimal LZ, then, a decision needs to be made on whether or not to perform LSA revascularisation. The decision needs to be made on an individual basis taking into account patient, operator and hospital factors in the context of the clinical urgency.

4.4. Strengths and limitations

An important limitation is that the study was not based on randomised evidence and enabled comparison of only a limited number of comparative studies in some subgroup analyses. A randomised controlled trial would be valuable but would be expensive and impractical. Exclusions also limited the study to English-only articles.

It is important to be aware of potential statistical and clinical heterogeneity, which could influence the findings. Statistical heterogeneity was limited as much as possible through careful consideration of the inclusion and exclusion criteria and robust application of the search protocol.

In such a study, clinical heterogeneity can be introduced because numerous operators (of a presumed varied skill base) in different centres were included. In addition, there were confounding factors which we could not account for. We expect that variation in peri-procedural blood pressure would affect outcomes such as spinal-cord ischaemia [14]. We presume that variations in haemodynamic parameters, recording, reporting and blood-pressure management protocols existed between studies but these data were not available for extraction. In addition, the length of thoracoabdominal aorta covered by a stent graft or the degree of LSA coverage would be expected to influence the outcome and this information too was not available. The degree of pre-existing disease of cerebral and intra-arch vasculature is also known to affect the stroke rate and this was not factored for in this study [13].

5. Conclusions

Management of the LSA during TEVAR is complex. Coverage of the LSA origin may cause downstream ischaemic complications, which may be overcome by revascularising the LSA. However, these strategies themselves may worsen the overall patient outcome. Therefore, taking into consideration the availability of time and facilities, we propose assessing each individual carefully by taking into account patient, operator and hospital factors. In this way, a bespoke approach is necessary to achieve the best risk—benefit ratio for the individual patient.

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