特集／大動脈瘤診療のめざましい進歩

各種動脈瘤に対する最新の治療

解離性大動脈瘤

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はじめに

今回，解離性大動脈瘤の最新の治療について述べるが，最近では瘤化を示さないものも含めて，大動脈解離の呼称で呼ばれることが多い。またこの大動脈解離には多くの分類が存在し，その分類された形態，時期によって治療法が異なるため，最初に大動脈解離の分類を中心とした大動脈解離の基礎について述べる。

Ⅰ．大動脈解離の基礎

大動脈解離の病型分類は1）解離範囲による分類，2）偽腔の血流状態による分類，3）病期による分類がある（表1）。一般的に多く用いられているStanford分類，DeBakey分類は解離範囲による分類であり，このDeBakey分類には亜型分類が存在し，逆行性Ⅲ型解離，弓部型，腹部型が存在する。また，Stanford分類とDeBakey分類の違いは前者が解離の存在する範囲でA型とB型に分類されているのに対し，後者では内膜亀裂が生じた部位と解離範囲を組み合わせた分類になっている。偽腔の血流状態による分類では偽腔開存型と偽腔血栓閉塞型（早期血栓閉塞型）に分類され[1)-3)]，偽腔血栓閉塞型は欧米ではintramural hematoma（IMH）と称されることが多く，ほぼ同様の病態であるが，欧米では大動脈壁内に血腫を形成したことに重点を置き，内膜亀裂の存在を重視していない定義である。本邦では大動脈壁内に血腫を形成する際は多くは内膜亀裂が存在することにより壁内に血液が流入し血腫を形成することが多いとの判断からの定義と考えてよい。しかし，全く内膜亀裂が存在せず，純粋に血管壁のvasa vasorumの破綻のみによるIMHが存在することも事実である[4)-8)]。次に病期による分類では急性期（発症から2週間以内）と亜急性期（3週目から2ヵ月まで）と慢性期（2ヵ月以降）に分類されている。急性期の2週間以内は最も破裂やタンポナーデや臓器虚血などの急激な変化を来す危険な時期でStanford A型では多くが緊急手術となる。亜急性期でも同様な急激な変化を来すことは見受けられるが，比較的希である。2ヵ月以降の慢性期では急激な変化はほとんどなく，解離腔の瘤化や血栓の状態の変化などがほとんどで，緩徐な変化が主体である。

以上の大動脈解離の定義，分類を十分に理解し，遭遇した患者さんが上記のどこに属するかを正確

表　1

1. 解離範囲による分類
   Stanford分類
   A型：上行大動脈に解離があるもの
   B型：上行大動脈に解離がないもの
   DeBakey分類
   Ⅰ型：上行大動脈に内膜亀裂があり弓部大動脈より末梢に解離が及ぶもの
   Ⅱ型：上行大動脈に解離が限局するもの
   Ⅲ型：下行大動脈に内膜亀裂があるもの
   Ⅲa型：腹部大動脈に解離がおよばないもの
   Ⅲb型：腹部大動脈に解離が及ぶもの
   DeBakey分類に際しては以下の亜型分類を追加できる
   逆行性Ⅲ型解離：内膜亀裂が下行大動脈にあり逆行性に解離が弓部から近位に及ぶもの
   弓部型：弓部に内膜亀裂があるもの
   弓部限局型：解離が弓部に限局するもの
   弓部広範型：解離が上行または下行大動脈に及ぶもの
   腹部型：腹部に内膜亀裂があるもの
   腹部限局型：腹部大動脈のみに解離があるもの
   腹部広範型：解離が胸部大動脈に及ぶもの
2. 偽腔の血流状態による分類
   偽腔開存型：偽腔に血流があるもの。部分的な血栓の存在はこの中に入れる
   偽腔血栓閉塞型：偽腔が血栓で閉塞しているもの
3. 病期による分類
   急性期：発症2週間以内。この中で発症48時間以内を超急性期とする
   亜急性期：発症後3週日（15日目）から2ヵ月まで
   慢性期：発症後2ヵ月を経過したもの

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に判定することで，治療方針まで決定できる分類方法となっている。例えば，急性Stanford A型大動脈解離，DeBakey I型，偽腔開存型との診断になれば，当然のことではあるが，緊急手術の適応であり，手術術式まで推定できる。このような診断を行うためには病歴，現症に加えて近年では画像診断の重要性が増しており，特にMDCTは欠かせない画像診断法である。画像診断の進歩により，解離に類似した病態も存在するようになった。

Ⅱ．大動脈解離に関連した病態の解説

急性大動脈解離の基礎および病態については上記したが，付随した病態や解離類似の病態が存在する。それらについては言葉の定義が問題となるが，ここではそれらの定義について説明を加えておく。

1. Ulcer like projection（ULP）

偽腔閉塞型大動脈解離の動脈造影で見られる潰瘍様小突出像で大部分が血栓閉塞した解離腔の中で内膜亀裂が存在する部位のみが消化管造影での潰瘍像のように動脈造影で突出して見える所見である。

2. Intramural hematoma（IMH）

CT scanを中心とした画像診断の進歩により，大動脈中膜が血腫により剥離しているが，内膜亀裂が見られない病態であり，自然消退するものがある一方，大動脈解離や大動脈瘤へ進展するものも認められる。これらは大動脈解離のVariantとして考えられている[4)-8)]。しかし，偽腔閉塞型大動脈解離との違いは内膜亀裂が存在するか否かであり，全ての内膜亀裂を画像診断で描出できるとは限らないため，どちらであるかの確定診断は困難である。IMHと偽腔閉塞型大動脈解離は類似の病態と考えておいてよいであろう。またその後の経過観察が予後を左右するため，綿密な観察が必要である。

3. Penetrating atherosclerotic ulcer（PAU）

大動脈の粥状硬化性病巣が潰瘍を形成して中膜より外層まで及ぶことがあり，これをPAUと称した。症例によっては血管外へ血腫を形成するものも存在する。解離へ進展することも考えられるが，まだ不明な点も多い[9)]。

4. Acute aortic syndrome（急性大動脈症候群）

胸痛や背部痛を伴って突然発症する大動脈に関連した疾患群の総称であり，急性大動脈解離，大動脈瘤破裂，大動脈瘤切迫破裂，IMH，PAUなどが含まれる。

表 2　急性A型大動脈解離の早期手術成績

対象症例	232例	
期間	1991年～2009年	
平均年齢	66±13歳（25～87）	
男性：女性	101：131	
手術術式		
上行近位弓部置換（部分弓部置換を含む）		182例
上行全弓部置換		18例
大動脈基部上行置換		14例
大動脈基部上行弓部全置換		3例
大動脈弁温存基部置換		3例
その他		12例
手術成績（病院死亡）	23例（9.9%）	

Ⅲ．大動脈解離の最新の治療

大動脈瘤の治療に関しては近年のステントグラフトによる治療（TEVAR）の進歩を外しては話ができないであろう。動脈硬化性の大動脈瘤の治療では現時点で約40%程度がTEVARによる治療に変わりつつある。しかし大動脈解離に関してはTEVARによる治療はまだ一般的ではなく，ある特殊な症例に対して行われているのが実情である。大動脈解離の治療を解説する上で全てを同時に解説することは困難であり，この項ではStanford分類を基準に話を進めるが，TEVARによる大動脈解離の治療も含めて解説する。

1. Stanford A

Stanford A型大動脈解離（以下A型解離と略す）の解離腔開存型の治療に関しては従来からの外科手術による上行大動脈からエントリー部までの人工血管置換術が現在においても基本である。これは急性期症例でも慢性期症例でもほぼ同様であるが，急性期症例では可能な限り早急に手術すべきである。近年，このようなA型解離に対する外科手術成績は非常に良好であり（表2），90%を越える急性期生存率を示す施設も少なくない。また，このような典型的な上行大動脈にtearが存在するような症例に対するステントグラフトでの治療はまだ行われていない。

A型解離の血栓閉塞型またはIMHの急性期症例の治療に関して多くの論文で検討されているが，上行弓部までにULPが存在する症例や上行弓部の大動脈径が5 cm以上の症例あるいは血栓閉塞した解離腔が10㎜以上の症例などに関しては急性期

表　3　上行大動脈血栓閉塞型大動脈解離の比較

	High risk group 9例	Low risk group 18例	P値
年齢	67.1±11.0	64.0±9.3	0.4588
男性：女性	2：7	7：9	0.3865
CTの所見			
上行大動脈外径（入院時）	54.1±9.3	46.5±4.5	0.0178
上行大動脈外径（入院7日後）	59.4±10.5	47.3±5.3	0.0074
上行大動脈内径（入院時）	39.3±9.2	36.6±3.6	0.3452
解離腔血栓の厚さ	11.7±4.0	8.8±2.0	0.0207
心嚢液の厚さ	4.7±3.5	5.5±3.4	0.5897

High risk group：6ヵ月以内の外科手術と死亡症例
Low risk group：6ヵ月以後の外科手術と内科的治療症例

外科手術を奨励する論文が多い。当科の症例での検討でも約10年間の経過観察でほぼ治癒に到った症例は27例中4例のみであり，やはり外科手術を推奨すべき結果であった（表3）。しかし，このような血栓閉塞型を呈する症例にはA型解離であっても，左鎖骨下動脈末梢に内膜亀裂（tear）が存在する症例が多く認められ，最近ではこのような症例に対して左鎖骨下動脈を閉塞させるようにステントグラフトを留置させる治療も限られた施設では行われている。しかし，このような急性期の脆弱な血管壁にステントグラフトを留置して慢性期まで，新たなtearを生じず，解離腔を隔離・血栓閉塞できるかどうかは今後の検討課題である。

2. Stanford B

Stanford B型大動脈解離の基本的な治療方針は血圧コントロールによる保存的治療である。急性期の外科的治療の対象は大動脈破裂，腹部および下肢などの虚血，持続する胸背部痛，コントロールできない高血圧等があげられる。このような合併症や現症を有する症例は急性B型解離の約10%に存在し，これらに対しては胸部下行大動脈のエントリーの存在する部位の大動脈置換術や腹部大動脈の内膜切除やカテーテルによる開窓術や腋窩大腿動脈バイパス術などが行われてきた。しかしこれらの外科的手技の成績は決して良好とは言えず，新しい治療法の出現が待たれていた。最近になってこれらB型大動脈解離に対するTEVARの治療成績が多く報告されるようになってきた。

まず，2009年にCirculationに掲載されたINSTEAD trial[10]では発症から2週間以上を経過した亜急性期症例150例に対して72例はTEVARを施行し，68例は内科的治療のみを行った。追跡2年のSiurvival rateはTEVAR 88.9%，内科的治療95.6%であり，両群間に差は認めなかった（p＝0.15）。また，大動脈関連死についても差は認めなかった。しかし，Aortic remodelingについてはTEVAR群で有意に認められ，長期遠隔では差が認められる可能性を示唆する所見であった。これらは主として2週間以上を経過した亜急性期症例であったが，急性B型大動脈解離症例に関しては2008年のTHE ANNALS OF THORACIC SURGERYにOutcome of Endovascular Treatment of Acute Type B Aortic Dissection[11]と言う形で29の論文をまとめて942症例のcomplicated acute type B dissection（合併症を有する急性B型大動脈解離）の成績をReviewとして報告しているが，病院死亡は9%であり，外科手術成績（29%）と比較して有意に良好であったことを報告している。しかし，その他の主要合併症として脳梗塞，対麻痺，A型解離への進展，腸管虚血，下肢切断などは8.1%に認められて，それ以外に20ヵ月の経過観察期間に追加のTEVARが10.4%，大動脈の破裂が0.8%に認められているが，これらの合併症を考慮してもcomplicated acute type B dissectionに対するTEVAR治療はOpen surgeryよりも有意に良好ではないかと，評価している。その他にも多くの論文でcomplicated acute type B dissectionに対するTEVAR治療は効果的であることが示され，一つの治療体系となりつつある。しかし，合併症を有しない急性B型解離に対するTEVAR治療の有効性はまだ示されていない。しかし今後，ステントグラフトデバイスの進歩や外科手術との組み合わせによるHybrid治療などの進歩により，TEVAR治療の適応は拡大して行くであろう。ここで，当科における1症

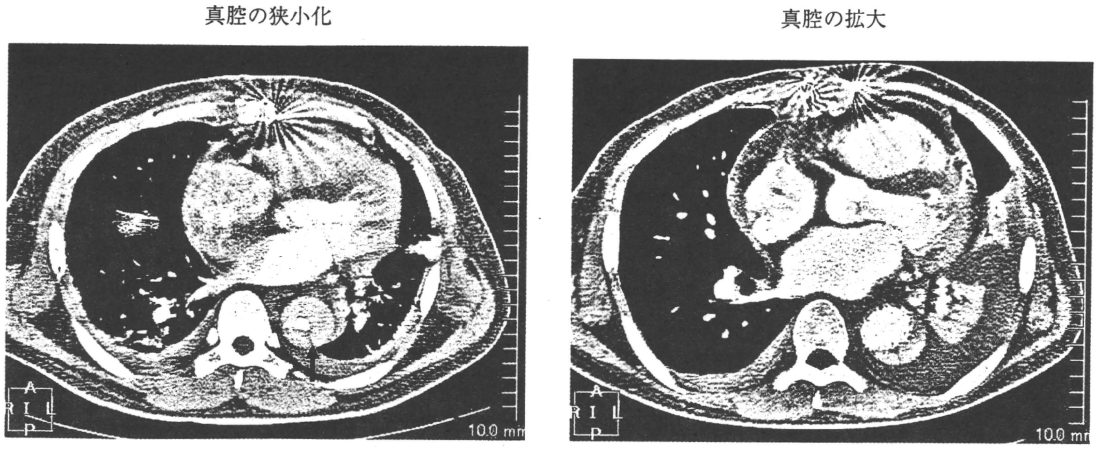


図　1

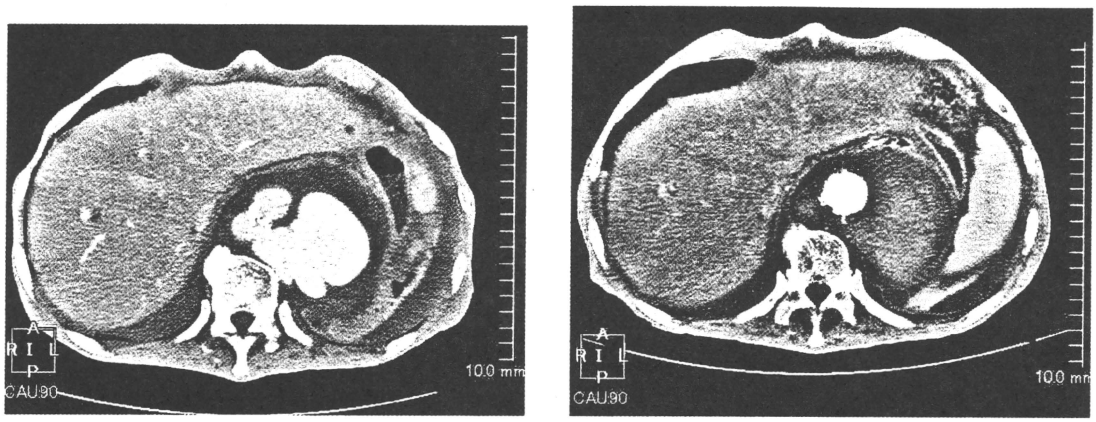
例を示すが，この症例は急性A型解離で，上行部分弓部置換を行った症例であったが，術後に腹痛を認め，尿量減少し，造影検査の結果，術中に発見できなかったTearが近位下行大動脈に存在し，真腔が狭小化し，腹部臓器虚血を来しているものと判断し，胸部下行大動脈にGORE-TAGのステントグラフトを挿入・留置した症例である。Tearを完全に閉塞することはできなかったが，TEVAR後から尿量は改善し，腹痛も消失した（図1）。このようにcomplicatedな症例に対して，有効な手段であると考えられる。

3. PAUに対する治療

既に上記したPAUについても，最近ではTEVARの良い適応となることが多い。PAUは動脈硬化性潰瘍が破綻して周囲組織へ穿破または破裂し，嚢状瘤の形態をとるものであり，突然，仮性瘤となることもある。周囲組織で破裂口を抑えられ，周囲に出血を認めない症例も存在する。しかし，これらはacute aortic syndromeの範疇に入り，多くは胸痛を伴う。この病態では病変部位と周囲の正常大動脈壁とに明らかな境界が存在し，大動脈の主要分枝から2㎝以上の距離が確保できれば，TEVARの良い適応となる。大量出血による術前の危機的状態が無ければ多く症例が劇的に改善する。当科の1症例を示す（図2）。この症例は80歳の男性で，1ヵ月前に胃癌に対して胃全摘手術を行われ，順調に回復していたが，突然，胸腹部痛と背部痛が出現し，ショック状態となった。その後，輸血にて100㎜Hgの血圧まで回復した状態で当科へ搬送となった。CT検査で胸部下行大動脈遠位部の腹腔動脈から5㎝中枢に石灰化を認め，同部位からの出血，仮性瘤形成，左胸腔内血腫を認め，PAUの破裂と診断した。1ヵ月前のCTでは大動脈瘤は全く存在していなかった。この症例の胸部下行大動脈にGORE-TAGのステントグラフトを挿入・留置し，造影剤の漏れは消失し，救命し，2週間後に紹介医へ転院となった。手術時間は2時間であった。このように開胸開腹を想定すると多大な侵襲が考えられる症例でも，低侵襲で短時間，短期間で回復が可能な画期的な治療法となっている。

ま　と　め

大動脈解離の治療成績は近年急激に進歩した。一般的にA型解離は現時点でも胸骨正中切開下の人工血管置換手術が主流であるが，手術が可能であった全症例での術後早期の手術成績でも急性期が約90%で，慢性期症例ではそれ以上の成績であり，この成績は欧米の手術成績を遙かに凌いでいる。術前状態が良好な患者では95%以上の成績を残すことができるレベルまで達している。B型解離の従来の手術成績の方がやや不良であり，特にcomplicated acute type B dissection（急性B型大動脈解離の合併症症例）では治療成績の改善が望まれている。その中で近年，TEVARによる治療成績が散見されるようになり，比較的良好であり，今後はcomplicated acute type B dissectionに対する主要な治療法となるであろう。しかし，non-complicated acute type B dissectionに対する治療成績は今でも保存的治療が最良の方法であるが，TEVARのデバイスの改良やHybrid治療などの導入で治療体系が変化する可能性を秘めて



留置前　　留置後

図　2　TEVAR by GORE-TAG for PAU rupture

いる。治療全てを総括して述べると，現時点ではTEVARの適応を慎重に検討したうえで，TEVAR可能と判断した症例に対してはTEVARを第1選択とし，それ以外の症例に対してはopen surgeryによる外科手術か内科的治療を選択することが最良の方法になりつつあるようである。

参考文献

1) Matsuo, H.: The thrombosed type of aortic dissection-its clinical features and diagnosis. International J of Angiol, 7: 329-334, 1998.
2) 松尾汎，中島伸之ほか：'いわゆるThrombosed type'急性大動脈解離の臨床像の検討．脈管学，27: 1088, 1987.
3) Nakajima, N., Matsuo, H. et al.: 'Thrombosed type aortic dissection', its clinical features and long term follow up results. Advances in vascular pathology (ed. by Strano, A. and Nobo, S.), 1989. Elsevier Science Publishers B. V., 1325-1330, 1989.
4) Heiberg, E., Wolverson, M. et al.: CT findings in thoracic aortic dissection. AJR Am J Roentgenol, 136: 13-17, 1981.
5) Robbins, R. C., Mcmanus, R. P., Mitchell, R. S., Latter, D. R., Moon, M. R., Olinger, G. N., Miller, D. C.: Management of patients with intramural hematoma of the thoracic aorta. Circulation, 88[part 2]: 1-10, 1993.
6) Erbel, R., Oelert, H., Meyer, J., Puth, M., Mohr-Katoly, S., Hausmann, D., Daniel, W., Maffei, S., Caruso, A., Covino, F. E.: Effect of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography. Implications for prognosis and therapy. The European Cooperative Study Group on Echocardiography. Circulation, 87: 1604-1615, 1993.
7) 栗林幸夫ほか：三次元CT血管造影法による大動脈および分枝疾患の診断－大動脈解離：電子ビームCTによる診断．臨床画像，12: 661-667, 1996.
8) Tyson, M. D.: Dissecting aneurysm. Am J Pathol, 7: 581-603, 1931.
9) Stanson, A. W., Kazmier, F. J., Hollier, L. H. et al.: Penetrating atherosclerotic ulcers of the thoracic aorta: natural history and clinicopathologic correlations. Ann Vasc Surg, 1: 15-23, 1986.
10) Christoph A. Nienaber, Hervé Rousseau, Holger Eggebrecht, Stephan Kische, Rossella Fattori, Tim C. Rehders, Günther Kundt, Dierk Scheinert, Martin Czerny, Tilo Kleinfeldt, Burkhart Zipfel, Louis Labrousse, Hüseyin Ince for the INSTEAD Trial. Randomized comparison of strategies for type B aortic dissection: The investigation of stent grafts in aortic dissection (INSTEAD) trial. Circulation, December 22/29, 120: 2519-2528, 2009.
11) Jun D. Parker and Jonathan Golledge: Outcome of endovascular treatment of acute type B aortic dissection. Ann Thorac Surg, 86: 1707-1712, 2008.

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EDITORIAL

Is hypothermia a reliable adjunct for spinal cord protection in descending and thoracoabdominal aortic repair with regional or systemic cooling?

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Since DeBakey first carried out thoracoabdominal aortic repair with an aortic homograft using a temporary arterial shunt in 1956,[1] multidisciplinary approaches have been directed toward minimizing spinal cord ischemic injury during surgery of the descending thoracic and thoracoabdominal aorta. The principal modality has been distal aortic perfusion during the aortic cross-clamping, which was passive in the past with a temporary shunt tube or bypass and that currently has been active with a left heart bypass or a cardiopulmonary bypass (CPB). Other approaches include the following: intraoperative monitoring of spinal cord ischemia with somatosensory evoked potentials and transcranial motor evoked potentials; reattaching the responsible intercostal artery; increasing collateral flow by controlling the back-bleeding of the patent intercostal arteries with high cardiac output and arterial pressures with cerebrospinal fluid (CSF) drainage; increasing ischemic tolerance with pharmacological adjuncts including naloxane, steroid, barbiturates, and with hypothermia; reducing excitotocixity from neuronal ischemia with hypothermia, naloxane, and steroid; and attenuating reperfusion injury with steroid, hypothermia, and free radical scavengers. Particularly, in Japan, with recent great advances in diagnostic imaging modalities, preoperative demonstration of the arteria radicularis magna (Adamkiewicz artery) by magnetic resonance imaging (MRI) or computed tomography (CT) scans has been highlighted as a reliable guide for reattachment or preservation of the responsible intercostal arteries including the collaterals.

Since the 1950s hypothermia has been proven effective for protecting organs, including the central nervous system, in the cardiovascular surgical field.[2–5] When focusing on milestones of aortic surgery, systemic profound hypothermia at 12°–15°C was applied to aortic arch surgery for brain protection in 1975.[6] Thereafter, during the 1980s, based on systemic hypothermia, more sophisticated antegrade or retrograde cerebral perfusion has been established for more definitive cerebral safety.[7,8] For aortic surgery through a left thoracotomy, Kouchoukos et al. employed systemic deep hypothermia to protect the spinal cord as well as the brain during descending thoracic and thoracoabdominal aortic repair in 1995.[9] In most, an open aortic anastomotic technique was used with circulatory arrest, avoiding aortic cross-clamping. On the other hand, mild hypothermia around 32°C with distal perfusion of a partial CPB has been our routine, as in most of Japan.[10] During the surgery, transcranial motor evoked potentials are used to monitor spinal cord ischemia continuously. A couple of the intercostal arteries responsible for the spinal cord ischemia are aggressively reattached according to the preoperative demonstration of the Adamkiewicz artery by MRI and CT, while controlling back-bleeding from the other intercostal arteries without delay. In selected "high-risk" patients having an extent I and II thoracoabdominal aortic aneurysm, however, our tactics have lately been shifted to deep hypothermic surgery (around 20°C) with total CPB for more rigorous spinal safety. Even in this setting, motor evoked potentials can be recorded above

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25°C with adjusted anesthetics. Early rewarming after the proximal anastomosis has been another refinement for minimizing some of the adverse effects of systemic hypothermia by reducing the time of ventricular fibrillation and CPB.

As described above, systemic hypothermia has some drawbacks, such as coagulopathy, pulmonary dysfunction, cardiac arrhythmia leading to cardiac arrest or ventricular fibrillation, systemic edema due to fluid shift, in part due to prolonged CPB. To eliminate these shortcomings, the usefulness of regional cooling of the spinal cord was addressed in surgery for the descending thoracic and thoracoabdominal aorta. In 1975, Hansebout et al. first highlighted the effect of local hypothermia and steroid on recovery from experimental spinal cord compression injury.[11] Between 1992 and 1994, several impressive reports were published on regional cooling of the spinal cord in animal models.[12–14] In our country, Tabayashi and colleagues also addressed the impact of epidural cooling on the spinal cord protection in an animal experiment.[15] The rationale for this technique is to increase the ischemic tolerance of the spinal cord during the critical periods of aortic cross-clamping associated with the reduction of the spinal cord perfusion pressure. Around the same time, Davison, Cambria, and colleagues (Massachusetts General Hospital, or MGH, group) first adopted this technique clinically in eight patients undergoing descending thoracic and thoracoabdominal aortic resection.[16] With their method, at least 30 min (average 50 min) before the aortic cross-clamping, 4°C saline solution was infused into the epidural catheter until the CSF temperature decreased to 25°C. Another subarachnoidal catheter was also used to measure CSF pressure and, if necessary, to drain the CSF. They have continued ongoing efforts for the development of this technique. In the latest report of 240 patients requiring extent I to III thoracoabdominal aneurysmal repairs, the incidence of spinal cord injury of any severity was 12.1%, including the results of emergency cases.[17]

In this volume, Tabayashi and colleagues (Tohoku University group) reported their 10-year experience of epidural perfusion cooling with outstanding outcomes—spinal cord injury in 3.9% and hospital deaths in 5.9%—for 102 patients undergoing descending thoracic and thoracoabdominal aortic repair. This regional cooling has been applied for the spinal cord only by them in Japan,[18,19] as the MGH group has advocated over the last decades.[20,21] Between them, the techniques of regional epidural cooling are similar in conjunction with the CSF drainage and measurement of CSF temperature. The outcome of the Tohoku University group was more favorable than that of the MGH group. One reason might be employment of distal perfusion with CPB or left heart bypass with mild systemic hypothermia at 31°–32°C by the Tohoku University group, which differed from the fundamental "clamp-and-saw" technique with normothermia without any assisted circulation by the MGH group. Meticulous surgical techniques of antegrade or retrograde segmental sequential repair for reduced spinal cord ischemia by the Tohoku group might be another factor for more favorable spinal safety, in conjunction with aggressive reattachment of the responsible intercostal arteries based on preoperative CT or MRI findings. Although the statistical analysis did not reveal any relevant factors for spinal cord injury, they noted that the main cause of spinal cord injury was likely due to unstable hemodynamics caused by massive bleeding. However, the causes of spinal cord injury are supposed to be multifactorial. Even with efficient epidural cooling, the spinal tolerance should be limited owing to relatively lower perfusion pressure of the spinal cord. Rushed reattachment of the responsible intercostal artery has technical difficulties. Given real-time monitoring of spinal cord ischemia, such as with transcranial motor evoked potentials, it would have been feasible to clarify the cause of the paraplegia.[10]

Some drawbacks of this novel epidural cooling have been recognized. One is a sharp rise in CSF pressure coincident with epidural cooling in some instances, which might be a risk factor for spinal cord ischemia.[22,23] The MHG group also warned about this increase in CSF pressure. The two groups tolerated this "modest and transient" increase because it can be controlled well with further CSF drainage or epidural cooling fluid drainage and because the risk of spinal cord ischemia is thought to be outweighed by the potential neuroprotective effect of epidural cooling.[20,21] From this point of view, it is important to maintain an arbitrary 30–40 mmHg gradient between the mean arterial pressure and the CSF pressure before aortic cross-clamping, with some caution. Otherwise, given a newly designed cooling catheter system, for example, with a closed countercurrent lumen, it is theoretically feasible to avoid this CSF pressure elevation.[23] On a related issue, one patient suffered an unexplained cerebrovascular accident associated with the highest CSF pressure. Interestingly, the MGH group experienced a similar case and recognized the difficulty of excluding the possibility of the increased CSF pressure contributing to the highly unusual but devastating lower cervical or upper thoracic spinal cord injury. Another technical question is about the temperature measurement of the spinal cord. The both groups measured the temperature of CSF—not that of the spinal cord—based on the correlation of the temperature between the spinal cord and the CSF. The practical con-

sideration of the degree of hypothermia required for spinal safety has been answered though a variety of animal experiments, which suggested 25°–27°C for uniform protection.[12–14] However, homogeneous cooling of the spinal cord might be uncertain. In this context, the local cooling times are all different and prolonged to an average of 50 min as reported by the MGH group, which is a defect of this method.[17]

Hypothermia is still the principle for organ protection, particularly for cerebral and spinal safety. Regional perfusion cooling into the epidural space is a unique and reliable method for spinal protection, minimizing the adverse effects of systemic hypothermia. Tabayashi and colleagues are to be congratulated for their outstanding outcomes and ongoing efforts in the development of epidural cooling. However, the unsolved complexity of this novel technique for spinal safety, including some drawbacks, seems to keep it from being in routine use.[23]

References

1. Creech O JR, DeBakey ME, Morris GC Jr. Aneurysm of thoracoabdominal aorta involving the celiac, superior mesenteric, and renal arteries; report of four cases treated by resection and homograft replacement. Ann Surg 1956;144: 549–73.
2. Bigelow WG, Callaghan JC, Hopps JA. General hypothermia for experimental intracardiac surgery; the use of electrophrenic respirations, an artificial pacemaker for cardiac standstill and radio-frequency rewarming in general hypothermia. Ann Surg 1950;132:531–9.
3. Lewis FJ, Taufic M. Closure of atrial septal defects with the aid of hypothermia; experimental accomplishments and the report of one successful case. Surgery 1953;33:52–9.
4. Kirklin JW, Dawson B, Devloo RA, Theye RA. Open intracardiac operations: use of circulatory arrest during hypothermia induced by blood cooling. Ann Surg 1961;154:769–76.
5. Barratt-Boyes BG, Simpson M, Neutze JM. Intracardiac surgery in neonates and infants using deep hypothermia with surface cooling and limited cardiopulmonary bypass. Circulation 1971;43(suppl):I25–30.
6. Griepp RB, Stinson EB, Hollingsworth JF, Buehler D. Prosthetic replacement of the aortic arch. J Thorac Cardiovasc Surg 1975;70:1051–63.
7. Ueda Y, Miki S, Kusuhara K, Okita Y, Tahata T, Yamanaka K. Surgical treatment of aneurysm or dissection involving the ascending aorta and aortic arch, utilizing circulatory arrest and retrograde cerebral perfusion. J Cardiovasc Surg 1990;31:553.
8. Kazui T, Washiyama N, Muhammad BA, Terada H, Yamashita K, Takinami M, et al. Total arch replacement using aortic arch branched grafts with the aid of antegrade selective cerebral perfusion. Ann Thorac Surg 2000;70:3–8.
9. Kouchoukos NT, Daily BB, Rokkas CK, Murphy SF, Bauer S, Abboud N. Hypothermic bypass and circulatory arrest for operations on the descending thoracic and thoracoabdominal aorta. Ann Thorac Surg 1995;60:67–76.
10. Ogino H, Sasaki H, Minatoya K, Matsuda H, Yamada N, Kitamura S. Combined use of Adamkiewicz artery demonstration and motor-evoked potentials in descending and thoracoabdominal repair. Ann Thorac Surg 2006;82:592–6.
11. Hansebout RR, Kuchner EF, Romero-Sierra C. Effects of local hypothermia and of steroids upon recovery from experimental spinal cord compression injury. Surg Neurol 1975;4: 531–6.
12. Berguer R, Porto J, Fedoronko B, Dragovic L. Selective deep hypothermia of the spinal cord prevents paraplegia after aortic cross-clamping in the dog model. Vasc Surg 1992;15:62–71.
13. Marsala M, Vanicky I, Galik J, Radonak J, Kundrat I, Marsala J. Panmyelic epidural cooling protects against ischemic spinal cord damage. J Surg Res 1993;55:21–31.
14. Allen BT, Davis CG, Osborne D, Karl I. Spinal cord ischemia and reperfusion metabolism: the effect of hypothermia. J Vasc Surg 1994;19:332–9.
15. Tabayashi K, Niibori K, Konno H, Mohri H. Protection from postischemic spinal cord injury by perfusion cooling of the epidural space. Ann Thorac Surg 1993;56:494–8.
16. Davison JK, Cambria RP, Vierra DJ, Columbia MA, Koustas G. Epidural cooling for regional spinal cord hypothermia during thoracoabdominal aneurysm repair. J Vasc Surg 1994; 20:304–10.
17. Conrad MF, Crawford RS, Davison JK, Cambria RP. Thoracoabdominal aneurysm repair: a 20-year perspective. Ann Thorac Surg 2007;83:S856–61.
18. Motoyoshi N, Takahashi G, Sakurai M, Tabayashi K. Safety and efficacy of epidural cooling for regional spinal cord hypothermia during thoracoabdominal aneurysm repair. Eur J Cardiothorac Surg 2004;25:139–41.
19. Tabayashi K, Motoyoshi N, Saiki Y, Kokubo H, Takahashi G, Masuda S, et al. Efficacy of perfusion cooling of the epidural space and cerebrospinal fluid drainage during repair of extent I and II thoracoabdominal aneurysm. J Cardiovasc Surg (Torino) 2008;49:749–55.
20. Cambria RP, Davison JK, Zannetti S, L'Italien G, Brewster DC, Gertler JP, et al. Clinical experience with epidural cooling for spinal cord protection during thoracic and thoracoabdominal aneurysm repair. J Vasc Surg 1997;25:234–41.
21. Cambria RP, Davison JK, Carter C, Brewster DC, Chang Y, Clark KA, et al. Epidural cooling for spinal cord protection during thoracoabdominal aneurysm repair: a five-year experience. J Vasc Surg 2000;31:1093–102.
22. Meylaerts SA, Kalkman CJ, de Haan P, Porsius M, Jacobs MJ. Epidural versus subdural spinal cord cooling: cerebrospinal fluid temperature and pressure changes. Ann Thorac Surg 2000;70:222–7.
23. Yoshitake A, Mori A, Shimizu H, Ueda T, Kabei N, Yozu R, et al. Use of an epidural cooling catheter with a closed countercurrent lumen to protect against ischemic spinal cord injury in pigs. J Thorac Cardiovasc Surg 2007;134:1220–6.

Multidisciplinary Approach to Prevent Spinal Cord Ischemia After Thoracic Endovascular Aneurysm Repair for Distal Descending Aorta

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Background. This article discusses the multidisciplinary approach to prevent spinal cord ischemia (SCI) with reference to the incidence of SCI after thoracic endovascular aneurysm repair (TEVAR) associated with closure of the intercostal-lumbar artery that supplies the Adamkiewicz artery (ICA-AKA).

Methods. We reviewed 60 patients [49 men, 57 to 89 years old] who underwent TEVAR (TAG [W. L. Gore & Associates, Flagstaff, AZ] 42; the Matsui-Kitamura (Kanazawa, Japan) 10; Talent [Medtronic Inc, Santa Rosa, CA] 5; TAG and Talent 3) for part of or the entire distal descending aorta between T7 and L2. These patients had frequently undergone aortic surgeries: ascending aorta (4), aortic arch (25), descending aorta (4), thoracoabdominal aorta (3), and abdominal aorta (19). The multidisciplinary approach consists of identification of the ICA-AKA by magnetic resonance angiography or computed tomographic angiography to avoid unnecessary coverage of the ICA-AKA, in combination with monitoring of motor evoked potentials, augmentation of mean arterial pressure (> 80 mm Hg), and cerebrospinal fluid drainage.

Results. Spinal cord ischemia occurred in 4 patients and patent ICA-AKAs were covered in 3 of them. The overall incidence of SCI was 6.7% and 9.4% in the group of 32 patients whose patent ICA-AKAs were covered by TEVAR. After treatment for SCI, 3 patients regained full ambulatory ability. Significant risk factors were identified as the artificial graft at the proximal landing zone, the number of covered zones (>8), the length of aortic coverage (>250 or >300 mm), and the length of the uncovered distal aorta (<60 mm).

Conclusions. A multidisciplinary approach is essential to prevent SCI after TEVAR for the distal descending aorta. This approach includes the preservation of patent ICA-AKAs after their identification, early diagnosis of SCI during TEVAR by monitoring motor evoked potentials, and prophylaxis and treatment of SCI by increasing mean arterial pressure to at least 80 mm Hg and performing cerebrospinal fluid drainage.

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Thoracotomy, aortic clamp, and distal perfusion under extracorporeal circulation become unnecessary when thoracic endovascular aortic repair (TEVAR) is used. As perfusion of the spinal cord is maintained during the procedure and the hemodynamics remain stable, the spinal cord is not at risk of reperfusion injury. Thoracic endovascular aortic repair has thus been considered as less invasive for the spine than open repair [1].

However, TEVAR requires covering of the intercostal-lumbar arteries (ICAs) to extend the landing zones and the optimal method for revascularization of ICAs after TEVAR has not been established. Moreover, the incidence of spinal cord ischemia (SCI) is not insignificant after TEVAR for the distal descending aorta, from which the intercostal-lumbar artery that supplies the Adamkiewicz artery (ICA-AKA) usually branches off [2].

In the case of open repair, various postoperative measures for spinal protection have been reported [3, 4]. In this retrospective study we discuss our multidisciplinary approach to prevent SCI after TEVAR with reference to the incidence of SCI associated with closure of the ICA-AKA and the significant risk factors for SCI after TEVAR.

Patients and Methods

Of the 112 patients operated on during the preceding 32 months at our department, we reviewed the clinical records of 60 who underwent TEVAR for part of or for the entire distal descending aorta after the ICA-AKA was identified by magnetic resonance angiography (MRA) or computed tomographic angiography (CTA). Our institution approved this retrospective study and waived patient consent requirements if patients could not be identified. The distal descending aorta was defined as the segment between T7 and L2 [5]. Patients who developed an aortic dissection or

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pseudoaneurysm after aortic replacement were not included, but two patients who developed a thin and thrombosed aortic dissection on a degenerative aneurysm were included. Three patients whose spinal cord function could not be evaluated due to, respectively, intraoperative death, cervical spondylotic myelopathy, and cerebral infarction were also excluded.

There were 11 women and 49 men (57 to 89 [77] years old), 53 of whom were 70-years old or older. According to the American Society of Anesthesiologists physical status classification, 17 patients were class 2, 28 class 3, 14 class 4, and 1 class 5. They were also characterized by a history of frequent aortic surgeries: ascending aorta (4 patients), aortic arch (25; TEVAR 1), descending aorta (4; TEVAR 1), thoracoabdominal aorta (3), abdominal aortic aneurysm (AAA) replacement (18), and EVAR (1).

Identification of the ICA-AKA

When the stent graft was expected to cover the distal descending aorta, the ICA-AKA was first identified by MRA or CTA. Contrast MRA and CTA were performed as previously reported in detail [2, 6]. In principle, we preferred to use MRA (52 patients) in order to avoid influence of the spine and to accurately differentiate the AKA from the anterior radicular vein [7]. However, as MRA was not always possible for emergency diagnosis of ICA-AKA, CTA was used instead for 8 patients.

The ICA-AKA was patent in 48 patients, occluded at its origin in 8, and double "patent" ICA-AKAs were identified in 4 patients. In total, 60 ICA-AKAs were confirmed (Table 1). In 4 patients, the ICA-AKA could not be confirmed by MRA and was considered to be "absent."

The MRA also revealed that the vertebral level of the orifice of the ICA-AKA did not always coincide with the vertebral number of the ICA-AKA. When they differed, the orifice of the ICA-AKA was usually located at the vertebral level below; for example, the Th10 ICA-AKA would branch at the Th11 vertebral level of the descending aorta. Closure of the ICA-AKA was defined by the placement of the stent graft at the origin of the ICA-AKA, and in patients with double ICA-AKAs it was defined by closure of one or both ICA-AKAs.

TEVAR

When planning the TEVAR, a sufficiently long landing zone was considered to be the first priority and preservation of the ICA-AKA patency the second. Coverage of the patent ICA-AKA was unavoidable in 32 patients.

Table 1. Adamkiewicz Artery Distribution

	Right	Left	Total
Th7		1	1 (1.6%)
Th8	2	6	8 (13.3%)
Th9	1	19	20 (33.3%)
Th10	2	13	15 (25.0%)
Th11	1	5	6 (10.0%)
Th12	3	3	6 (10.0%)
L1	1	2	3 (5.0%)
L2		1	1 (1.6%)
Total	10	50	
	(16.7%)	(83.3%)	

The thoracic stent grafts used were the Gore TAG (W. L. Gore & Associates, Flagstaff, AZ) for 42 patients, the Matsui-Kitamura stent graft (Kanazawa, Japan) [8] for 10 patients, the Talent (Medtronic Inc, Santa Rosa, CA) for 5, and both Gore TAG and Talent for 3 patients.

To create suitable landing zones, the following aortic branches were closed: left carotid artery (2 patients), left subclavian artery (4), celiac axis (5), and mesenteric and renal arteries (2). All left carotid, left subclavian (LSCA), mesenteric, and renal arteries were reconstructed with bypass surgery. Except for one patient who underwent bypass surgery after division of the celiac axis (CA), the CA was closed by means of coil embolization using the balloon occlusion test of the celiac artery [9]. Bypass surgery to prepare for the closure of major aortic branch(es) was usually performed one week before TEVAR and supraaortic bypass was performed concomitant with TEVAR for 2 patients.

Intervention for AAA was concurrently performed for 5 patients: AAA replacement for 3 and endovascular aneurysm repair for 2. In 17 patients whose external iliac and (or) femoral arteries were not large enough, an 8 or 10 mm conduit was connected to the common or external iliac artery.

Thoracic endovascular aortic repair was carried out under general anesthesia. The duration of the operation varied (51 to 406 [130] minutes) due to the need for concomitant performance of the procedures mentioned above. Twenty patients required blood transfusion. An artificial graft was used for the landing zone for endografting in 24 patients in view of their history of aortic surgeries. An artificial graft was used at the proximal landing zone for 20 patients after total arch replacement (TAR) with or without elephant trunk installation and for 3 patients after TEVAR for aortic arch or proximal descending aorta. For 11 of the patients subjected to TAR, TEVAR was performed as the second-stage surgery for extensive aneurysm in 7 patients or multiple aneurysms in 4. As for the distal landing zone, 5 patients underwent thoracoabdominal replacement and 2 AAA replacements in conjunction with debranching bypass for the visceral arteries. Artificial grafts were used for both landing zones in 4 patients.

Monitoring of Motor Evoked Potentials and Cerebrospinal Fluid Drainage

Transcranial motor evoked potentials of the anterior tibialis and thenar muscles were monitored in all patients every five minutes after stent grafting under general anesthesia [10, 11].

A cerebrospinal fluid drainage (CSFD) tube [12] was positioned in 41 patients prior to TEVAR. The CSFD tube could not be inserted due to exigency in 2 patients, to bleeding tendency in 5, and to preservation of ICA-AKA patency in 12. In 1 patient, who developed paraplegia the day after TEVAR due to rupture, a CSFD tube was inserted immediately after the onset of paraplegia.

Table 2. Risk Factors for Spinal Cord Ischemia (χ^2 Test)

Variables	SCI (−) (n = 56)	SCI (+) (n = 4)	*p* Value
75 years old or older	22 (39.3%)	3 (75.0%)	0.5734
Male gender	44 (78.6%)	2 (50.0%)	0.1337
History of total arch replacement	22 (39.3%)	3 (75.0%)	0.1611
History of AAA surgery	24 (42.9%)	0 (0.0%)	0.0747
Patent left internal iliac artery[a]	49 (87.5%)	4 (100%)	0.3103
Operation time ≥ 240 minutes	6 (10.7%)	2 (50.0%)	0.6360
Blood transfusion	16 (28.6%)	4 (100%)	0.0022
Emergency	2 (3.6%)	1 (25.0%)	0.1506
Concomitant surgery[b]	25 (44.6%)	3 (75.0%)	0.2328
Artificial graft at proximal landing zone	24 (33.9%)	4 (100%)	0.0043
Artificial graft at distal landing zone	5 (8.9%)	0 (0.0%)	0.3956
Number of covered zones ≥ 8	31 (55.4%)	4 (100%)	0.0336
Aortic coverage ≥ 250 mm	10 (17.6%)	4 (100%)	0.0004
Aortic coverage ≥ 300 mm	1 (1.8%)	3 (75.0%)	0.0001
Distal uncovered aorta ≤ 60 mm	28 (50.0%)	4 (100.0%)	0.0234
Coverage of ICA-AKA	29 (51.8%)	3 (75.0%)	0.3562
Reexploration for bleeding	1 (1.8%)	2 (50.0%)	0.0041
Prophylactic CSFD	9 (16.1%)	0 (0.0%)	0.2453

[a] Left subclavian and right internal iliac arteries were patent in all patients. [b] Concomitant surgery includes supraaortic bypass, AAA replacement, and iliac conduit.

AAA = abdominal aortic aneurysm; CSFD = cerebrospinal fluid drainage; ICA-AKA = the intercostal-lumbar artery that supplies the Adamkiewicz artery; SCI = spinal cord ischemia.

Treatment and Prophylaxis of SCI

Immediately after stent grafting, the mean arterial pressure (MAP) was raised to above 80 mm Hg with the aggressive use of catecholamines. This increase in MAP was maintained for two days.

When the amplitude of motor evoked potentials declined under general anesthesia, or when symptoms and signs of SCI were noted during the postoperative period, the MAP was raised to above 90 mm Hg and CSFD was started at 12 cm H_2O. The drainage gradient was adjusted between 10 and 15 cm H_2O to control the drainage volume at less than 20 mL per hour and 300 mL per day.

After the onset of SCI, methylprednisolone (30 mg/kg bolus and 5.4 mg/kg/hour for 23 hours followed by 2.7 mg/kg/hour for 2 days) and naloxone (1,200 μg/day) were infused [13]. This intensive spinal care, together with the augmentation of MAP, CSFD, methylprednisolone, and naloxone was discontinued 24 hours after full recovery or continued for 72 hours if the symptoms did not resolve.

During the early stages, CSFD was not indicated as a prophylactic measure. However, after the clinical data of this study had been accumulated, the indication of CSFD was modified. In the 9 most recent patients whose ICA-AKA had been patent and covered by TEVAR, prophylactic CSFD was started when the patient was transferred to the intensive care unit and continued for more than 12 hours.

Measurement of the Aortic Length

The length of the proximal uncovered aorta (from the LSCA to the stent graft), of aortic coverage by the stent graft, and of the distal uncovered aorta (from the stent graft to CA) was measured at the centerline of the aorta on CTA in 59 patients [2]. After TAR with a multibranch graft, the length of aortic coverage was measured from the distal anastomosis, which coincided with the origin of the elephant trunk. After replacement of the descending aorta or the thoracoabdominal aorta, the position of LSCA and (or) CA served as the point of reference for the measurement.

Statistical Analysis

Values show the mean ± SD. Data were analyzed with the χ^2 test for categoric variables and continuous variables with analysis of variance. The level of statistical significance was set at a *p* value less than 0.05.

Results

Spinal cord ischemia occurred in 4 patients and patent ICA-AKAs were covered in 3 of them. The overall incidence of SCI was 6.7% and 9.4% in the group of 32 patients whose patent ICA-AKAs were covered by using TEVAR, 2 of whom needed reoperation for hemostasis of a retroperitoneal hematoma and bleeding at the puncture site. In another patient, whose patent ICA-AKA was left uncovered, TEVAR was performed while the patient was in shock due to rupture, and the anastomosis side of the iliac conduit became occluded and it had to be reanastomosed 6 hours after TEVAR. One patient developed SCI as evidenced by a drop in motor evoked potential amplitude during TEVAR and the symptom was confirmed when the patient awoke from general anesthesia. The incidence of SCI, which was confirmed by a drop in motor evoked potential amplitude was 1.7% for all patients. Three other patients developed

Ann Thorac Surg
2010;90:561–5

SCI 10 hours (1 patient) and 24 hours (2 patients) after TEVAR [14].

After treatment for SCI, 3 patients regained full ambulatory ability. The incidence of permanent SCI was 1.7% after TEVAR for the distal descending aorta and 3.1% for patients whose patent ICA-AKAs were covered.

There were 2 hospital mortalities. One was a patient who died of methicillin-resistant *Staphylococcus aureus* mediastinitis 4 months after undergoing TEVAR subsequent to TAR, the other a patient who had undergone TAR and thoracoabdominal aortic grafting but died 2 months after TEVAR due to infective endocarditis.

Other intraoperative complications and postoperative comorbidities, one per patient, were iliac artery injury, femoral artery thrombosis, and tracheostomy for pneumonia. Spinal bleeding occurred in a patient who started walking before removal of the inserted CSFD tube.

During the follow-up period, 5 patients underwent a secondary intervention: repeated TEVAR for type I endoleak in 2 patients, for type III endoleak and for endotension in 1 patient each, and the ligation of intercostal arteries for type II endoleak in 1 patient.

The common risk factors for SCI were analyzed with the χ^2 test (Table 2). Four patients with SCI had no history of AAA surgery, but even with a history of frequent TAR and (or) AAA surgery, the LSCA and right internal iliac artery were patent and the patency of the left internal iliac was not a significant risk factor for SCI. Due to the small number of patients, parameters showing an obvious difference between the patients with and without SCI, such as long operation time (>4 hours), emergency TEVAR, concomitant surgeries including supraaortic bypass, and utilization of an iliac conduit were not significant risk factors for SCI in this study. On the other hand, artificial graft at the proximal landing zone, number of covered zones (>8), length of aortic coverage (>250 or >300 mm) and length of the uncovered distal aorta (<60 mm) were significant risk factors. Of the 4 patients who developed SCI, the ICA-AKA was covered in 3 patients and preserved in 1 who underwent emergency TEVAR while in shock after rupture of the aneurysm.

Comment

The risk for SCI is considered to be lower after TEVAR than with open aortic repair [1], but in case of the former, additional ICAs must be sacrificed for the landing zones and revascularization of the ICAs is impossible after TEVAR. We have been using a multidisciplinary approach to prevent SCI after TEVAR that consists of preoperative identification of the ICA-AKA and planning to avoid its coverage, early diagnosis of SCI during TEVAR through motor evoked potential monitoring, and prophylaxis and treatment of SCI by increasing MAP and performing CSFD.

In this retrospective study, we reviewed the clinical records of patients who underwent TEVAR for part of or for the entire distal descending aorta. Coverage of eight or more spinal zones, longer (>250 mm or >300 mm) aortic coverage, and the shorter (<60 mm) length of uncovered distal aorta were identified as significant risk factors for SCI. All these factors involve the sacrifice of a large number of ICAs [15–18]. Another significant risk for SCI was the artificial graft at the proximal landing zone; that is, the elephant trunk with TAR and the proximal descending graft or stent graft. The artificial graft at the proximal landing zone may be in relation with other factors because it also results in the sacrifice of a large number of ICAs. Of these factors leading to the closure of ICAs, aortic coverage with a length of greater than 250 mm was the most sensitive, aortic coverage of greater than 300 mm showed the lowest p value, and the number of covered zones could be counted most easily in a clinical situation. However, the most appropriate parameter to show the number of sacrificed ICAs remains to be determined. Nevertheless, the risk involved in long coverage of the descending aorta must be emphasized.

The LSCA and internal iliac arteries are usually considered to constitute possible collateral sources of spinal blood supply. In our study, these arteries proved not to be important risk factors for SCI because of the high percentage of patency, 100% of LSCA and right internal iliac artery, and 88% of the left internal iliac. Lumbar arteries are the other collateral source of spinal blood supply. Open and endoluminal AAA surgeries, which involve the sacrifice of several pairs of lumbar arteries, have thus been described as significant risks for SCI after TEVAR [1, 19]. However, a history of or concomitant performance of AAA surgery including three EVARs did not constitute a risk factor for SCI in our study because none of the patients who experienced SCI had a history of AAA surgery. Aguiar Lucas and colleagues [20] reported that multilevel aortic disease was not associated with an increased incidence of SCI, possibly due to the avoidance of thoracic aortic cross-clamping resulting in no reperfusion injury and maintenance of adequate spinal perfusion during the perioperative period [21–24].

After open repair of the thoracoabdominal aorta, perioperative hemodynamic stability is essential for the prevention of SCI. In particular, the duration of hypotension after termination of the partial cardiopulmonary bypass has proved to be an independent risk factor for paraplegia [25]. Bleeding was recognized as a risk factor for SCI in our study, so to achieve stable perioperative hemodynamics after TEVAR, bleeding and subsequent hypotension should be avoided. Two of our patients showed a tendency to bleed during TEVAR in the form of difficulties with hemostasis at the anastomosis of the conduit and bleeding from the puncture site. In these patients, extensive TEVAR from the elephant trunk to the subtotal descending aorta was indicated. Moreover, consumption coagulopathy due to massive coagulation inside an extended aneurysm may be indirectly related to SCI after TEVAR.

After closure of several pairs of ICAs, collateral flow was supplemented by increasing MAP. We initially maintained MAP at 80 mm Hg and further increased it to 90 mm Hg after the onset of SCI. This, together with concomitant CSFD and pharmacologic intervention, resolved SCI in 3 of 4 patients. Our current strategy is to maintain MAP above 90 mm Hg with the aggressive use of catecholamines for 48 hours to prevent SCI, including delayed-onset SCI [20, 26].

To break the vicious cycle of spinal ischemia and edema, perioperative CSFD has been confirmed to reduce paraplegia after open repair of extent I and II thoracoabdominal aneurysms [27, 28]. We could successfully perform CSFD after TEVAR, except for one patient who started walking before the CSFD tube had been removed and developed spinal bleeding.

In the early stages, a CSFD tube was positioned in all patients who underwent TEVAR for the distal descending aorta and CSFD was started only after the onset of SCI. However, due to the low sensitivity for SCI (9.7%) among the patients whose patent ICA-AKA was covered and high specificity (96.6%) among those whose patent ICA-AKA was left uncovered, no CSFD tube was inserted when the ICA-AKA remained patent. On the other hand, when coverage of the patent ICA-AKA is required, prophylactic CSFD was performed after TEVAR in the most recent nine cases. However, since the usefulness of prophylactic CSFD has not been demonstrated, its indication remains to be evaluated.

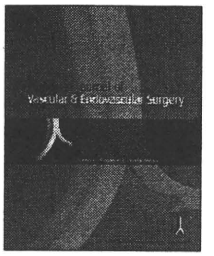
Coverage of the ICA-AKA was not a significant risk factor for SCI. This may be erroneously interpreted as meaning that identification of ICA-AKA is unnecessary provided MAP is increased to at least 80 mm Hg and prophylactic CSFD is executed. However, in our patients the aim of every preoperative and intraoperative attempt to plan and perform TEVAR for distal descending aorta was to avoid coverage of the ICA-AKA, although the effect of unnecessary or unexpected closure of ICA-AKA remains unknown.

In conclusion, a multidisciplinary approach is essential to prevent SCI after TEVAR for the distal descending aorta. This approach includes the preservation of patent ICA-AKAs after their identification, early diagnosis of SCI during TEVAR by monitoring motor evoked potentials, and prophylaxis and treatment of SCI by increasing MAP to at least 80 mm Hg and performing CSFD.

References

1. Baril DT, Carroccio A, Ellozy SH, et al. Endovascular thoracic aortic repair and previous or concomitant abdominal aortic repair: is the increased risk of spinal cord ischemia real? Ann Vasc Surg 2006;20:188–94.
2. Matsuda H, Fukuda T, Iritani O, et al. Spinal cord injury is not negligible after TEVAR for lower descending aorta. Eur J Vasc Endovasc Surg 2010;39:179–86.
3. Svensson LG, Crawford ES, Hess KR, Coselli JS, Safi HJ. Experience with1509 patients undergoing thoracoabdominal aortic operations. J Vasc Surg 1993;17:357–68.
4. Coselli JS, LeMaire SA, Schmittling ZC, Köksoy C. Cerebrospinal fluid drainage in thoracoabdominal aortic surgery. Semin Vasc Surg 2000;13:308–14.
5. Ishimaru S. Endografting of the aortic arch. J Endovasc Ther 2004;11(Suppl 2):II62–71.
6. Yamada N, Takamiya M, Kuribayashi S, Okita Y, Minatoya K, Tanaka R. MRAof the Adamkiewicz artery: a preoperative study for thoracic aortic aneurysm. J Comput Assist Tomogr 2000;24:362–8.
7. Ogino H, Sasaki H, Minatoya K, Matsuda H, Yamada N, Kitamura S. Combined use of adamkiewicz artery demonstration and motor-evoked potentials in descending and thoracoabdominal repair. Ann Thorac Surg 2006;82:592–6.
8. Sanada J, Matsui O, Terayama N, et al. Clinical application of a curved nitinol stent-graft for thoracic aortic aneurysms. J Endovasc Ther 2003;10:20–8.
9. Libicher M, Reichert V, Aleksic M, Brunkwall J, Lackner KJ, Gawenda M. Balloon occlusion of the celiac artery: a test for evaluation of collateral circulation–prior endovascular coverage. Eur J Vasc Endovasc Surg 2008;36:303–5.
10. Jacobs MJ, Meylaerts SA, de Haan P, de Mol BA, Kalkman CJ. Assessment of spinal cord ischemia by means of evoked potential monitoring during thoracoabdominal aortic surgery. Semin Vasc Surg 2000;13:299–307.
11. Kawanishi Y, Munakata H, Matsumori M, et al. Usefulness of transcranial motor evoked potentials during thoracoabdominal aortic surgery. Ann Thorac Surg 2007;83:456–61.
12. Miyamoto K, Ueno A, Wada T, Kimoto S. A new and simple method of preventing spinal cord damage following temporary occlusion of the thoracic aorta by draining the cerebrospinal fluid. J Cardiovasc Surg 1960;1:188–97.
13. Acher CW, Wynn MM, Hoch JR, Popic P, Archibald J, Turnipseed WD. Combined use of cerebral spinal fluid drainage and naloxone reduces the risk of paraplegia in thoracoabdominal aneurysm repair. J Vasc Surg 1994;19:236–46.
14. Safi HJ, Miller CC 3rd, Azizzadeh A, Iliopoulos DC. Observations on delayed neurologic deficit after thoracoabdominal aortic aneurysm repair. J Vasc Surg 1997;26:616–22.
15. Kawaharada N, Morishita K, Kurimoto Y, et al. Spinal cord ischemia after elective endovascular stent-graft repair of the thoracic aorta. Eur J Cardiothorac Surg 2007;31:998–1003.
16. Feezor RJ, Martin TD, Hess PJ Jr, et al. Extent of aortic coverage and incidence of spinal cord ischemia after thoracic endovascular aneurysm repair. Ann Thorac Surg 2008;86:1809–14.
17. Amabile P, Grisoli D, Giorgi R, Bartoli JM, Piquet P. Incidence and determinants of spinal cord ischaemia in stent-graft repair of the thoracic aorta. Eur J Vasc Endovasc Surg 2008;35:455–61.
18. Greenberg R, Resch T, Nyman U, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-term follow-up. J Vasc Surg 2000;31:147–56.
19. Schlösser FJ, Verhagen HJ, Lin PH, et al. TEVAR following prior abdominal aortic aneurysm surgery: increased risk of neurological deficit. J Vasc Surg 2009;49:308–14.
20. Aguiar Lucas L, Rodriguez-Lopez JA, Olsen DM, Diethrich EB. Endovascular repair in the thoracic and abdominal aorta: no increased risk of spinal cord ischemia when both territories are treated. J Endovasc Ther 2009;16:189–96.
21. Castelli P, Caronno R, Piffaretti G, et al. Endovascular repair for concomitant multilevel aortic disease. Eur J Cardiothorac Surg 2005;28:478–82.
22. Gawenda M, Aleksic M, Heckenkamp J, Reichert V, Gossmann A, Brunkwall J. Hybrid-procedures for the treatment of thoracoabdominal aortic aneurysms and dissections. Eur J Vasc Endovasc Surg 2007;33:71–7.
23. Black SA, Wolfe JH, Clark M, Hamady M, Cheshire NJ, Jenkins MP. Complex thoracoabdominal aortic aneurysms: endovascular exclusion with visceral revascularization. J Vasc Surg 2006;43:1081–9.
24. Roselli EE, Greenberg RK, Pfaff K, Francis C, Svensson LG, Lytle BW. Endovascular treatment of thoracoabdominalaortic aneurysms. J Thorac Cardiovasc Surg 2007;133:1474–82.
25. Kawanishi Y, Okada K, Matsumori M, et al. Influence of perioperative hemodynamics on spinal cord ischemia in thoracoabdominal aortic repair. Ann Thorac Surg 2007;84:488–92.
26. Azizzadeh A, Huynh TT, Miller CC 3rd, et al. Postoperative risk factors for delayed neurologic deficit after thoracic and thoracoabdominal aortic aneurysm repair: a case-control study. J Vasc Surg 2003;37:750–4.
27. Coselli JS, Lemaire SA, Köksoy C, Schmittling ZC, Curling PE. Cerebrospinal fluid drainage reduces paraplegia after thoracoabdominal aortic aneurysm repair: results of a randomized clinical trial. J Vasc Surg 2002;35:631–9.
28. Piano G, Gewertz BL. Mechanism of increased cerebrospinal fluid pressure with thoracic aortic occlusion. J Vasc Surg 1990;11:695–701.

Eur J Vasc Endovasc Surg (2010) 39, 179–186



Spinal Cord Injury is Not Negligible after TEVAR for Lower Descending Aorta☆

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Abstract *Objectives:* To clarify the incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR), we investigate the intercostal/lumbar arteries that supply the Adamkiewicz artery (ICA-AKA).
Patients: Among 81 patients subjected to TEVAR, we retrospectively reviewed the clinical records of 50 patients (range: 57–86 (median age: 77) years, 41 males) who underwent TEVAR for part of or the whole distal descending aorta (T7 to L2) after identification of ICA-AKA by magnetic resonance angiography (MRA) or computed tomography angiography (CTA).
Results: The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered, group B: 24 patients whose ICA-AKA was covered and group C: nine patients in whom no patent ICA-AKA was identified. Only three patients in group B suffered paraplegia and of them two recovered full ambulation. The estimated incidence of permanent and transient paraplegia was 3.7% in all TEVAR patients, 6.0% when part of or the entire distal aorta was covered and 12.5% when the patent ICA-AKA was covered. The length of aortic coverage in patients with paraplegia was >300 mm.
Conclusions: Paraplegia after TEVAR occurred in one of eight patients in whom the stent graft covered ICA-AKA. Long coverage of the aorta including the ICA-AKA was critical. To prevent this serious complication, identification of the ICA-AKA is crucial.

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The incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR) has been reported to vary according to the demographics of the patients.[1–20] Whether the integrity of the Adamkiewicz artery (AKA) is essential for spinal cord function is still to be investigated.[21] However, after reattachment of the intercostal/lumbar arteries, which supply AKA (ICA-AKA), or of the adjacent intercostal/lumbar arteries during thoracoabdominal aortic replacement, motor-evoked potentials (MEPs) recover.[22] TEVAR has been reported to reduce SCI.[12] In principle, the longer the length of the aorta including both landing zones that is covered by TEVAR, the larger the number of ICAs that will be sacrificed and whose revascularisation will be impossible.[23]

To clarify the incidence and cause of SCI after TEVAR, we have investigated the patency of ICA-AKA in relation to other factors which may cause SCI.

Materials/Methods

Patient demographics

In the past 27 months, of 81 patients, we performed TEVAR with Gore TAG (W. L. Gore & Associates, Flagstaff, AZ, USA) in 47 patients, Talent thoracic stent graft (Medtronic, Inc., Santa Rosa, CA, USA) in five, both TAG and Talent in one and Matsui-Kitamura (MK) stent graft in 28 patients.[24] In this study, we included 50 patients who underwent TEVAR for part of or the whole distal descending aorta after ICA-AKA was identified by magnetic resonance angiography (MRA) or computed tomography angiography (CTA). The distal descending aorta was defined as the segment between T7 and L2.[25] Fifteen patients who underwent TEVAR above T6 and 16 patients who had not undergone MRA or CTA to identify ICA-AKA were not included in this investigation.

In general, the patients were senescent, debilitated and presented co-morbidities. (Table 1) Thirty-seven patients were ≥75 years old and the median age was 77. Thirty-seven patients were in ASA class 3 or 4, and 32 patients had a history of aortic surgery (48 surgeries in total).

Of the 18 patients who had undergone AAA repair, TEVAR had been indicated more than 1 year later in 11 patients, scheduled within 3 months in five and performed simultaneously in two. Emergency TEVAR was performed in three for haemoptysis, acute aneurysm dissection and persistent back pain. They were haemodynamically stable and could undergo CTA for ICA-AKA.

In all patients, another CTA was carried out to precisely measure the aneurysm and access. CTA also revealed the patency of the left subclavian (LSCA) and bilateral internal iliac arteries (IIA). Occlusion of left IIA (LIIA) was confirmed in three patients but LSCA and right IIA (RIIA) were patent in all the patients regardless of whether total arch replacement (TAR) or AAA repair was performed.

Identification of ICA-AKA

ICA-AKA was identified by MRA in 39 and by CTA in 11 patients.

The details of contrast MRA were previously reported by Yamada et al.[26] For the CTA, an Aquilion 16 multi-detector row CT scanner (Toshiba, Tokyo, Japan) was used. To detect AKA, the reconstruction field of view was set to the area around the aorta and spine. The images were processed in a workstation (Ziostation; Amin, Tokyo, Japan). Volume-rendered images of the entire aorta were routinely generated. Multiplanar reformation (MPR) images, including oblique coronal images with craniocaudal angulations and curved planar reformation images, were reconstructed to investigate the side and level of the origin of AKA.

Diagnostic criteria for the anterior spinal artery and AKA were as previously reported.[26] We preferred MRA as CTA is disadvantageous due to the influence of the spine and lack of accurate differentiation of the AKA from the anterior radicular vein.[22] However, the selection of MRA or CTA

Table 1 Patient demographics.

Number of Patients	50			
Age	57–86 [median 77] year-old			
Gender	41 male			
ASA class	Class 2: 13, Class 3: 19, Class 4: 18			
History of aortic surgery	Root to Ascending	3		
	Arch	21	Total arch replacement	20
			TEVAR after debranch	1
	Descending		Replacement	3
			TEVAR	1
	Thoraco-abdominal	2		
	AAA	18	Replacement	17
			EVAR	1
Aortic pathology	Degenerative aneurysm	39		
	Chronic dissection	3		
	Acute dissection on aneurysm	2		
	Penetrating atherosclerotic ulcer	3		
	Anastomotic false aneurysm	3		

Table 2 Distribution of ICA-AKA.

	Right	Left	(Occlusion at origin)
Th7	0	1	
Th8	1	6	(2)
Th9	0	18	(1)
Th10	1	10	
Th11	0	7	(1)
Th12	2	4	(2)
L1	0	1	
L2	0	0	
Total	4	47	(6)

ICA-AKA: intercostal/lumbar arteries which supplies Adamkiewicz artery.

depended on the availability of the equipment. CTA was used in all three emergency cases.

When AKA was not identified by MRA, it was diagnosed as 'absent' ($n = 3$). In 47 patients, 51 ICA-AKAs were identified (Table 2). In four patients, there were double ICA-AKAs. Occlusion of ICA-AKA at its origin was diagnosed in six patients, in all of them on the left side. When the ICA-AKA was occluded, blood supply from adjacent intercostal or lumbar arteries was suspected to be significant. However, we were unable to distinguish the critical collateral flow to AKA.

TEVAR

To create a landing zone, a carotid–subclavian bypass was performed in two and visceral vessel bypass was performed in one. In nine patients who had extensive/multiple aneurysm(s) from the aortic arch to the descending aorta, TAR was performed using elephant trunk (ET) implantation. Regarding patients who had a history of aortic surgery, an artificial graft was used to create a proximal landing zone in 19 and a distal landing zone in three.

In all patients, TEVAR was carried out under general anaesthesia. The access route for TEVAR was a native artery in 35, an iliac conduit in 13 and a graft limb or a side branch of AAA graft in two patients.

MEP monitoring and cerebrospinal fluid drainage

In all patients trans-cranial MEPs were monitored during TEVAR and a cerebrospinal fluid drainage (CSFD) tube was placed before TEVAR in 31 patients.

Immediately after the stent graft was placed, the mean blood pressure was raised above 80 mmHg and MEP was monitored every 5 min. When the amplitude of MEPs decreased under general anaesthesia, or when symptoms and signs of SCI were noted during the postoperative period, CSFD (<15 cmH_2O) was started with the infusion of methylprednisolone (30 mg kg^{-1} bolus and 5.4 mg kg^{-1} h^{-1} for 23 h followed by 2.7 mg kg^{-1} h^{-1} for 2 days) and naloxone (1200 μg day^{-1}). Intensive spinal care with CSFD, methylprednisolone and naloxone was continued for 72 h if the symptom did not resolve or was discontinued 24 h after full recovery.

CSFD was started only after paraplegia or a decrease of less than 25% of the amplitude of MEPs was noted. CSFD was not indicated as a prophylactic measure after TEVAR.

Measurement of the aortic length

The length of 'proximal uncovered aorta' (from LSCA to stent graft), 'aortic coverage' by stent graft and 'distal uncovered aorta' (from stent graft to coeliac axis (CA)) was measured on CTA using curved planar reformation images processed in a workstation (GE Advantage workstation 4.3).

After TAR with a multibranch graft, the length of aortic coverage was measured from the distal anastomosis. This site coincided with the origin of ET and was several centimetres distal to the branch graft of LSCA. When ET was installed, the proximal edge of the stent graft was positioned inside the multibranch graft and not only inside ET. After replacement of the descending or the thoracoabdominal aorta, the position of LSCA and/or CA served as the point of reference for the measurement.

Statistical analysis

Values are the mean ± SD. Data were analysed using the chi-square test for categorical variables, and continuous variables were examined using analysis of variance (ANOVA). The level of statistical significance was set at $p < 0.05$.

Results

Mortality and morbidity

Initial success of TEVAR was achieved in all patients except for two patients with Type I endoleaks detected by CTA who were successfully treated by a repeat TEVAR. No operative (30 days) death was encountered. Injury and occlusion of access arteries occurred in one. Two patients were complicated with cerebral embolism due to the guidewire pull-through technique and atrial fibrillation.

The following three patients were complicated with paraplegia: Patient 1 was a 59-year-old man with a history of closure of ventricular septal defect, aortic valve replacement and repair of a Valsalva sinus aneurysm. He also suffered from liver cirrhosis. He developed aneurysmal dilatation of the whole thoracic aorta and underwent TAR with ET installation as the first-stage repair. MRA revealed the AKA arose from the left Th9-ICA. TEVAR with Gore TAG was performed 5 weeks later from ET (Z3) to T11. The iliac conduit was connected to the right common iliac artery but the haemostasis was time consuming because of obvious coagulopathy due to liver cirrhosis. Paraplegia was confirmed 24 h after TEVAR after the patient suffered much pain. Despite treatment for SCI, the patient could not ambulate. Retroperitoneal haematoma had to be removed twice. He eventually died from methicillin-resistant *Staphylococcus aureus* (MRSA) mediastinitis 4 months after TEVAR. The length of aortic coverage from the origin of ET to the distal flair was 325 mm.

Patient 2 was an 81-year-old man with ascending, arch and descending aorta aneurysms. MRA revealed the AKA branching from the left Th9-ICA. Four weeks after TAR with ET installation, TEVAR with TAG was performed from ET (Z3) to T12 (Fig. 1). The iliac conduit was required and

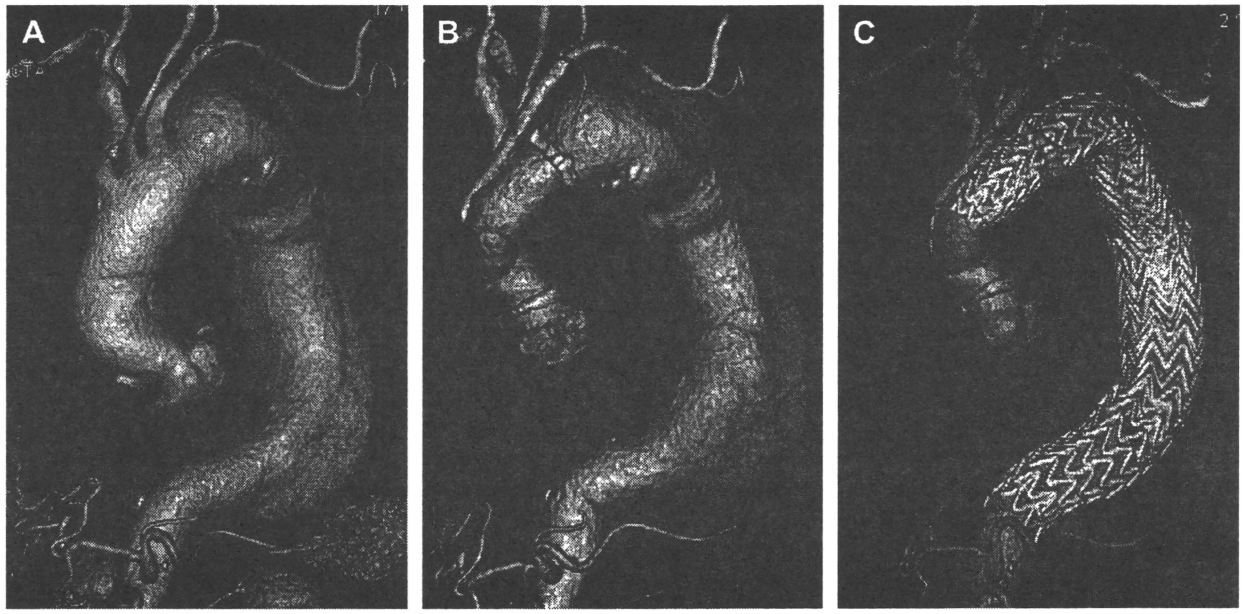

Figure 1 Sequence of CTA in Patient 2. Panel A: Preoperative, Panel B: After total arch replacement, Panel C: After TEVAR.

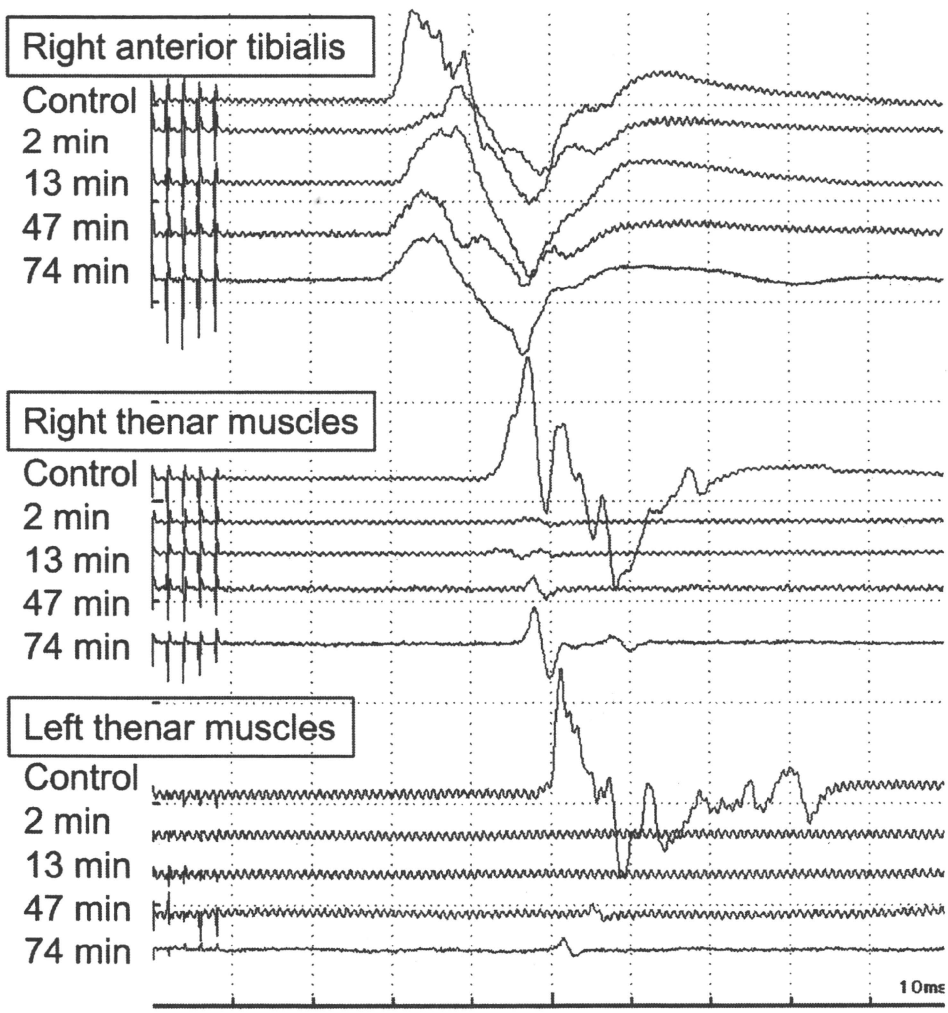

Figure 2 Sequence of MEPs in Patient 3. MEPs of the right anterior tibialis and both thenar muscles before TEVAR over Th9-ICA (control) and 2, 13, 47, and 74 minutes after TEVAR.

haemostasis took a long time due to co-existing consumption coagulopathy caused by aortic lesions. Six hours after TEVAR, the patient suddenly complained of back pain and paraplegia was confirmed. One hour after intensive spinal treatment, he could move his legs and on the next morning he could walk. The retroperitoneal, femoral and brachial haematomas were removed twice. The length of aortic coverage was 302 mm.

Patient 3 was a 78-year-old woman who had undergone TEVAR (Z3 to T7) for a proximal descending aortic aneurysm 6 months earlier. MRA revealed the ICA-AKA branching from the left Th9-ICA. Due to the rapid growth of the distal descending aortic aneurysm, TEVAR was performed again from the previous stent graft to L1. The CA was closed to create a distal landing zone. Immediately after the deployment over the Th9-ICA, the MEPs of both thenar muscles diminished and the amplitude of the MEPs of the right anterior tibialis decreased about 50% from the control amplitude (Fig. 2). Despite treatment for SCI, ankle dorsiflexion was slight when she awoke from anaesthesia. Intensive spinal care was continued and she gradually gained muscle strength within 3 h after TEVAR. On the following morning she could ambulate. The length of aortic coverage after the first TEVAR was 157 mm and was extended to 308 mm by the second TEVAR.

Incidence of paraplegia

The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered by TEVAR, group B: 24 patients whose ICA-AKA was covered by TEVAR and group C: nine patients in whom no patent ICA-AKA was identified. Group C included six patients whose ICA-AKA occluded at its origin and three patients whose ICA-AKA was absent.

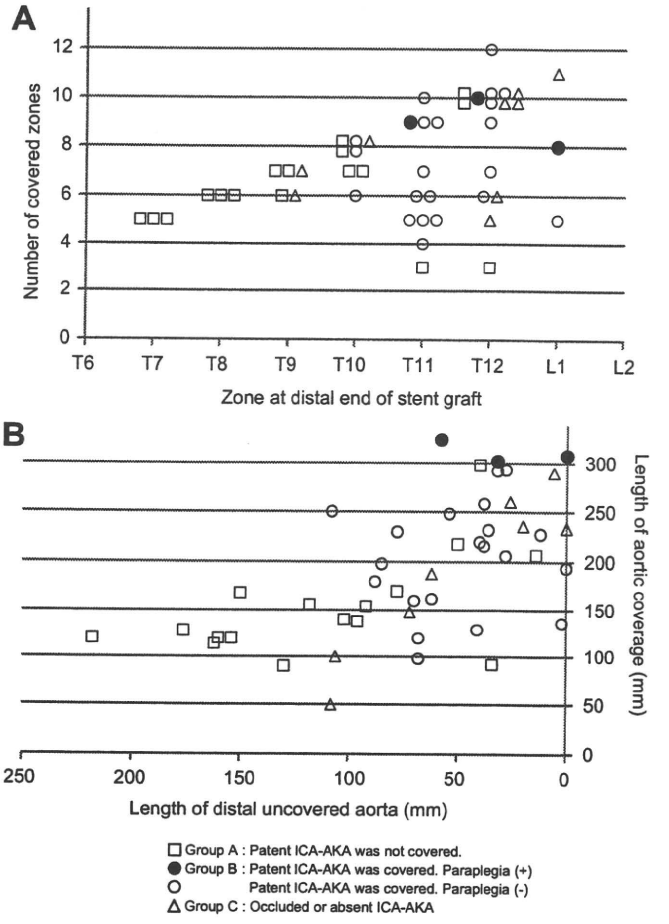

Figure 3 Distribution of patients with paraplegia in accordance with covered aorta and distal uncovered aorta expressed as number of aortic zones (Panel A) and measured length (Panel B).

Table 3 Comparison of patients with and without paraplegia.

	Paraplegia $N = 3$	No paraplegia $N = 47$	p
Age (year-old)	72.7 ± 11.9	76.1 ± 6.1	.3736
Male gender	2 (67%)	39 (83%)	.5094
ASA classification	3.7 ± 0.6	3.1 ± 0.8	.2026
Renal dysfunction	1 (33%)	18 (38%)	.3362
History of aortic repair (descending, thoracoabdominal, abdominal)	1 (33%)	19 (40%)	.8060
LSCA patency	3 (100%)	47 (100%)	—
RIIA patency	3 (100%)	47 (100%)	—
LIIA patency	3	44	.5359
Op time (minutes)	252 ± 117	141 ± 76	.0200
Blood loss (ml)	557 ± 274	363 ± 423	.4482
Use of an iliac conduit	2 (67%)	15 (32%)	.2335
Zones of aortic coverage	9 ± 1	7.2 ± 2.2	.1691
Proximal uncovered aorta (mm)	0	36 ± 49	.2191
Aortic coverage (mm)	312 ± 12	179 ± 64	.0009
Distal uncovered aorta (mm)	30 ± 29	72 ± 52	.1839
Hypotension	2 (67%)	3 (6%)	.0116

LSCA: Left subclavian artery, R(L)IIA: Right internal iliac artery.

205 mm was the critical length of aortic coverage for SCI.[18] Feezor et al. described that both the extent (>200 mm) and distal location of aortic coverage (20 mm from CA) were associated with an increased risk for SCI.[16]

We tried to locate the critical segment for paraplegia by dividing the aorta into zones but the length measured by CTA demonstrated the critical length of aortic coverage more clearly. This particular threshold, 300 mm, might vary in the future as experience accumulates and the index is modified, for instance, according to height. Nevertheless, it can be emphasised that long aortic coverage is another important risk factor for paraplegia. Long coverage of the aorta including the patent ICA-AKA is critical.

We found intra-operative coagulopathy related to prolonged operation time and postoperative retroperitoneal bleeding in patient 1 and patient 2. Hypotension associated with retroperitoneal bleeding contributes to SCI.[6] Consumption coagulopathy is another risk which is heightened by long coverage of the aorta.

Paraplegia occurred when the stent graft covered the zones at T11 or was placed less than 60 mm from CA. It can be concluded that the zones above T10 or a distance of more than 60 mm from CA are safe. However, the length of distal uncovered aorta would only express the probability of the occlusion of the ICA-AKA according to its distribution. The length of distal uncovered aorta might be less significant than the closure of the ICA-AKA or the length of aortic coverage.

Similarly, high percentages of paraplegia, 12.5% and 14.3%, after TEVAR were reported in patients with prior AAA repair.[12,20] In our series, a history of abdominal, thoraco-abdominal, or descending aneurysm repair was not a significant risk for paraplegia. However, AAA repair sacrifices several pairs of lumbar arteries that significantly contribute to spinal cord perfusion and/or IIA, which are the possible sources of direct or collateral blood flow to spinal arteries. Indeed, previous AAA repair was described as a risk factor in various other reports.[2,6,12,27,31]

Limitations of this study include the retrospective review of prospectively collected data, the retrospective measurement of aortic length and the small number of patients. Further accumulation of patients treated by TEVAR after identification of ICA-AKA is crucial for more precise diagnosis of the risk for paraplegia after TEVAR.

Conclusions

Paraplegia after TEVAR occurred in 1 of 8 (12.5%) patients in whom the stent graft covered the distal descending aorta below Th7. Long (>300 mm) coverage of the aorta including the ICA-AKA is a critical risk factor for SCI and paraplegia. To prevent this serious complication, it is imperative to identify the ICA-AKA before performing TEVAR.

Conflict of Interest/Funding

None.

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References

1 Mitchell RS, Miller DC, Dake MD, Semba CP, Moore KA, Sakai T. Thoracic aortic aneurysm repair with an endovascular stent graft: the "first generation" *Ann Thorac Surg* 1999;**67**:1971–4.
2 Gravereaux EC, Faries PL, Burks JA, Latessa V, Spielvogel D, Hollier LH, et al. Risk of spinal cord ischemia after endograft repair of thoracic aortic aneurysms. *J Vasc Surg* 2001;**34**:997–1003.
3 Criado FJ, Clark NS, Barnatan MF. Stent graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. *J Vasc Surg* 2002;**36**:1121–8.
4 Ellozy SH, Carroccio A, Minor M, Jacobs T, Chae K, Cha A, et al. Challenges of endovascular tube graft repair of thoracic aortic aneurysm: midterm follow-up and lessons learned. *J Vasc Surg* 2003;**38**:676–83.
5 Orend KH, Scharrer-Pamler R, Kapfer X, Kotsis T, Görich J, Sunder-Plassmann L. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. *J Vasc Surg* 2003;**37**:91–9.
6 Cheung AT, Pochettino A, McGarvey ML, Appoo JJ, Fairman RM, Carpenter JP, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. *Ann Thorac Surg* 2005;**80**:1280–8.
7 Bell RE, Taylor PR, Aukett M, Sabharwal T, Reidy JF. Mid-term results for second-generation thoracic stent grafts. *Br J Surg* 2003;**90**:811–7.
8 Bergeron P, De Chaumaray T, Gay J, Douillez V. Endovascular treatment of thoracic aortic aneurysms. *J Cardiovasc Surg (Torino)* 2003;**44**:349–61.
9 Czerny M, Cejna M, Hutschala D, Fleck T, Holzenbein T, Schoder M, et al. Stent-graft placement in atherosclerotic descending thoracic aortic aneurysms: midterm results. *J Endovasc Ther* 2004;**11**:26–32.
10 Makaroun MS, Dillavou ED, Kee ST, Sicard G, Chaikof E, Bavaria J, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. *J Vasc Surg* 2005;**41**:1–9.
11 Greenberg RK, O'Neill S, Walker E, Haddad F, Lyden SP, Svensson LG, et al. Endovascular repair of thoracic aortic lesions with the Zenith TX1 and TX2 thoracic grafts: intermediate-term results. *J Vasc Surg* 2005;**41**:589–96.
12 Baril DT, Carroccio A, Ellozy SH, Palchik E, Addis MD, Jacobs TS, et al. Endovascular thoracic aortic repair and previous or concomitant abdominal aortic repair: is the increased risk of spinal cord ischemia real? *Ann Vasc Surg* 2006;**20**:188–94.
13 Kawaharada N, Morishita K, Kurimoto Y, Hyodoh H, Ito T, Harada R, et al. Spinal cord ischemia after elective endovascular stent-graft repair of the thoracic aorta. *Eur J Cardiothorac Surg* 2007;**31**:998–1003.
14 Morales JP, Taylor PR, Bell RE, Chan YC, Sabharwal T, Carrell TW, et al. Neurological complications following endoluminal repair of thoracic aortic disease. *Cardiovasc Intervent Radiol* 2007;**30**:833–9.
15 Sandroussi C, Waltham M, Hughes CF, May J, Harris JP, Stephen MS, et al. Endovascular grafting of the thoracic aorta, an evolving therapy: ten-year experience in a single centre. *ANZ J Surg* 2007;**77**:974–80.
16 Feezor RJ, Martin TD, Hess Jr PJ, Daniels MJ, Beaver TM, Klodell CT, et al. Extent of aortic coverage and incidence of spinal cord ischemia after thoracic endovascular aneurysm repair. *Ann Thorac Surg* 2008;**86**:1809–14.
17 Greenberg RK, Lu Q, Roselli EE, Svensson LG, Moon MC, Hernandez AV, et al. Contemporary analysis of descending

thoracic and thoracoabdominal aneurysm repair: a comparison of endovascular and open techniques. *Circulation* 2008;**118**: 808–17.
18 Amabile P, Grisoli D, Giorgi R, Bartoli JM, Piquet P. Incidence and determinants of spinal cord ischaemia in stent-graft repair of the thoracic aorta. *Eur J Vasc Endovasc Surg* 2008;**35**:455–61.
19 Hnath JC, Mehta M, Taggert JB, Sternbach Y, Roddy SP, Kreienberg PB, et al. Strategies to improve spinal cord ischemia in endovascular thoracic aortic repair: outcomes of a prospective cerebrospinal fluid drainage protocol. *J Vasc Surg* 2008;**48**:836–40.
20 Schlösser FJ, Verhagen HJ, Lin PH, Verhoeven EL, van Herwaarden JA, Moll FL, et al. TEVAR following prior abdominal aortic aneurysm surgery: increased risk of neurological deficit. *J Vasc Surg* 2009;**49**:308–14.
21 Griepp RB, Ergin MA, Galla JD, Lansman S, Khan N, Quintana C, et al. Looking for the artery of Adamkiewicz: a quest to minimize paraplegia after operations for aneurysms of the descending thoracic and thoracoabdominal aorta. *J Thorac Cardiovasc Surg* 1996;**112**:1202–13.
22 Ogino H, Sasaki H, Minatoya K, Matsuda H, Yamada N, Kitamura S. Combined use of adamkiewicz artery demonstration and motor-evoked potentials in descending and thoracoabdominal repair. *Ann Thorac Surg* 2006;**82**:592–6.
23 Bicknell CD, Riga CV, Wolfe JH. Prevention of paraplegia during thoracoabdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2009;**37**:654–60.
24 Sanada J, Matsui O, Terayama N, Kobayashi S, Minami T, Kurozumi M, et al. Clinical application of a curved nitinol stent-graft for thoracic aortic aneurysms. *J Endovasc Ther* 2003;**10**: 20–8.
25 Ishimaru S. Endografting of the aortic arch. *J Endovasc Ther* 2004;**11**(Suppl 2):II62–II71.
26 Yamada N, Takamiya M, Kuribayashi S, Okita Y, Minatoya K, Tanaka R. MRA of the Adamkiewicz artery: a preoperative study for thoracic aortic aneurysm. *J Comput Assist Tomogr* 2000;**24**: 362–8.
27 Cooper DG, Walsh SR, Sadat U, Noorani A, Hayes PD, Boyle JR. Neurological complications after left subclavian artery coverage during thoracic endovascular aorticrepair: a systematic review and meta-analysis. *J Vasc Surg* 2009;**49**: 1594–601.
28 Buth J, Harris PL, Hobo R, van Eps R, Cuypers P, Duijm L, et al. Neurologic complications associated with endovascular repair of thoracic aortic pathology: incidence and risk factors. a study from the European Collaborators on Stent/Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) registry. *J Vasc Surg* 2007; **46**:1103–10.
29 Carroccio A, Marin ML, Ellozy S, Hollier LH. Pathophysiology of paraplegia following endovascular thoracic aortic aneurysm repair. *J Card Surg* 2001;**11**:359–66.
30 Greenberg R, Resch T, Nyman U, Lindh M, Brunkwall J, Brunkwall P, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-term follow-up. *J Vasc Surg* 2000;**31**:147–56.
31 Mitchell RS, Miller DC, Dake MD. Stent-graft repair of thoracic aortic aneurysms. *Semin Vasc Surg* 1997;**10**:257–71.