

保たれていることに着目し、自己弁を温存したまま拡張した大動脈壁-Valsalva洞-のみを切除し人工血管に置換する術式が発表された。いわゆるDavid手術(aortic root reimplantation)と称される、直管状人工血管を左室流出路側より刺入した縫合糸で大動脈弁輪に固定して、その内側に大動脈弁尖を縫着する術式である⁸⁾。次いで1993年、弁輪縫合を省いて舌状に切り込みを入れた人工血管でValsalva洞を置換するYacoub手術(aortic root remodeling)が発表され⁹⁾、自己弁温存術式はこの両術式をもって広く普及した。

(2) 自己弁温存大動脈基部置換術の論点

第一の論点は、“DavidかYacoubか?”ということである。David手術においては当初、比較的小径の人工血管が用いられることが多く、弁開放時に弁尖が人工血管壁と干渉して変性をきたす危険性が危惧された。一方のYacoub手術では舌状の人工血管がValsalva洞様のふくらみを形成するため、生理的な形態が保たれることが利点とされた。しかし、Yacoub手術では弁輪への介入がなされないため、遠隔期に弁輪拡張による弁逆流再発が問題となった。弁輪への補強縫合を別個に追加する改良も考案されたが成績としては不確実で、またDavid手術の二重の縫合ラインに対してYacoub手術では1ラインのみであるため出血の危険性も高いという弱点も指摘された。そこで、今日ではValsalva洞形態を付加した人工血管

表1 自己弁温存術式の変遷(C. Millerによる命名)

David-I	reimplantation
David-II	remodelingと同様の術式
David-III	弁輪への補強を追加したreimplantation
David-IV	大径人工血管を用い、ST-junction部を縫縮
David-V	Valsalva洞形態を再現したreimplantation

でDavid手術を行う、David-V手術が最も信頼性のある術式と考えられるようになった(表1)。

もう一つの論点は、術式的前提となる“弁尖は正常”であることが、本当にMFSのAAEにもあてはまるのか、という疑問である。David自身は、MFSでも遠隔成績に問題はないとしている¹⁰⁾。しかし、症例数の豊富なStanfordからは、やはり弁の耐久性に問題があること、弁輪拡大の観点からDavid手術が望ましいこと、が報告されている¹¹⁾。またJohns Hopkinsの報告では、Bentall手術と比べると再手術のリスクが高くてもイベント発生率と遠隔生存率で有利¹²⁾とされており、結論を得るに至っていない。それでも、ある程度の-生体弁の耐久性を上回るべきだが-長期間にわたり抗凝固療法なく生活できるならば、少なくとも若年女性、特に妊娠・育児希望のある女性には大きな利点のある術式であることは異論のないところである。

(3) 当科での術式：

David-V UT modification

当科では1998年に自己弁温存術式を導入し、当初はMFSにはDavid手

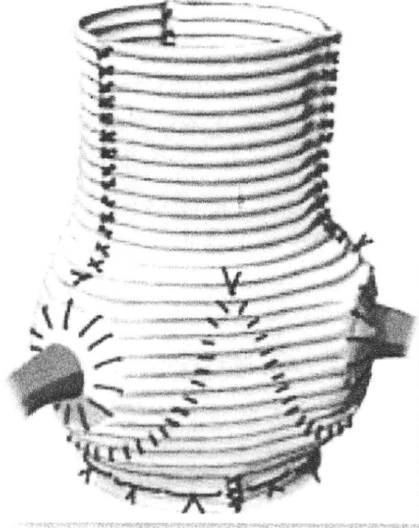
術を、non-MFSにはYacoub手術を選択していた。2004年より自己弁温存全症例にDavid-V手術の東京大学変法：David-V UT modification¹³⁾を標準術式として適用している。これは径32~34mmの大径人工血管を用い、弁尖縫着後にST-junctionから遠位側吻合部までの人工血管を3カ所で縫縮してValsalva洞を形成する方法で、特別な人工血管を必要とせず、かつデザイン自由度の高い術式である(図1)。2010年5月までに45例のDavid-V UT modificationを施行し、手術死亡なし、現時点での平均AR I度、弁置換なし、と成績は良好である。しかし、遠隔死亡が3例：不整脈死2、分娩後脳出血1、とMFS特有の疾患で失っており、今後の経過観察上での大きな問題と捉えている。

MFSにおける その他心疾患治療の変貌

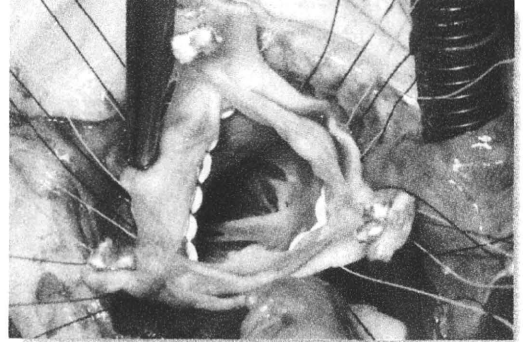
(1) 僧帽弁逸脱症の治療

MFSに合併する僧帽弁逸脱症の頻度は、従来は40~80%といわれてきたが、昨今の厳密な診断基準では30%程度と

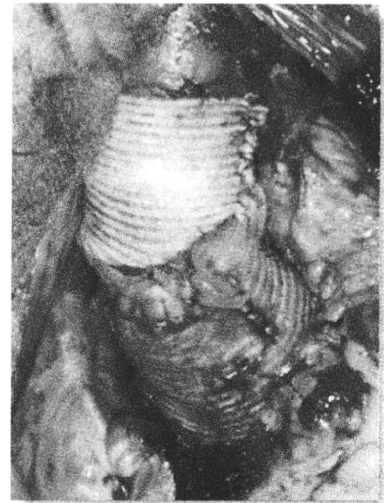
a: 人工血管のデザインと完成模式図



b: 弁輪への縫合糸



d: 残存上行大動脈のラッピング

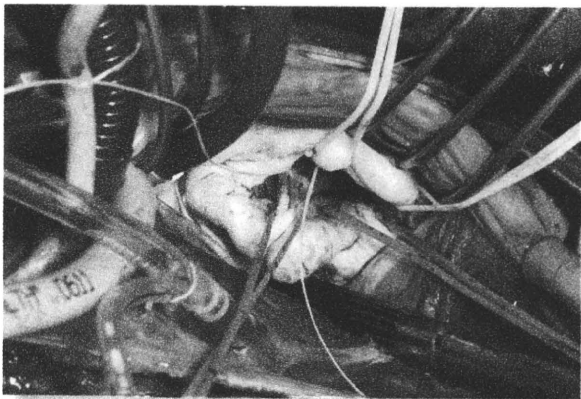


c: 接合良好となった弁尖



図1 David-V UT modification

a: 前後尖の広範逸脱



b: 人工腱索で逆流は消失した

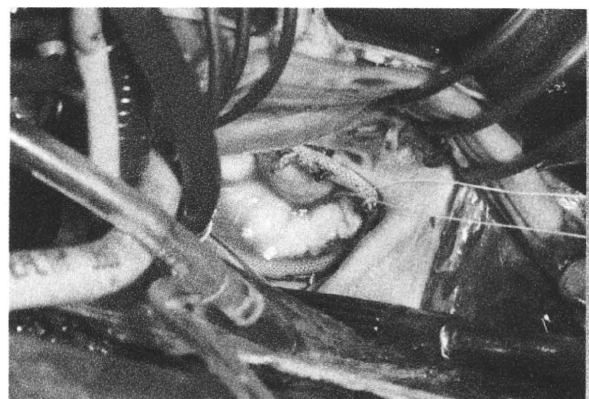


図2 人工腱索を用いた僧帽弁形成術

考えられている¹⁴⁾。前後尖の広範な逸脱を呈することが多く、これまでは多くの症例で人工弁置換術が施行されてきた。僧帽弁に関しても大動脈弁と同様に弁形成技術の進歩は著しく、人工腱索の長期遠隔成績が実証されるに至って、広範逸脱や小児期の重症僧帽弁閉鎖不全症にも形成術が適用されるようになり、良好な長期成績をあげている¹⁵⁾(図2)。大動脈弁・僧帽弁ともに人工弁使用を回避できることの意義は大きいと考えられる。

(2) 心室性不整脈

MFS患者の生命予後を規定する重

要な因子として、心室性不整脈が注目されている¹⁶⁾。当科でも、大動脈基部と僧帽弁の修復がされながら不整脈死を遂げた症例を複数経験しており、リスクの予知と防止が課題となっている。DCGに代表される従来の検査法だけではなく、TDIを用いた心筋障害の検出¹⁷⁾などの今後の発展に期待したい。

MFSにおけるステントグラフト治療の問題点

胸部下行大動脈瘤および解離において、従来の開胸手術に対する血管内治

療(thoracic endovascular aneurysm repair; TEVAR)の施行比率は急速に上昇している。低侵襲性は当然のこととして、企業製デバイスの供給により合併症も減少し遠隔成績も向上してきている。しかし、現時点ではMFSへの積極的適応を示唆するエビデンスには乏しく、高率なエンドリークや新規解離発症などの報告も多く、遠隔成績はいまだ不明である¹⁸⁻²⁰⁾。TEVARは“健常部にランディングする”ことが基本であり、びまん性の中膜病変を有するMFSへの適用、特に解離への適用には慎重な配慮が必要と思われる。

文献

- 1) Van Karnebeek DD, Naeff MS, Mulder BJ, et al: Natural history of cardiovascular manifestations in Marfan syndrome. *Arch Dis Child* 84: 129-137, 2001.
- 2) Judge DP, Dietz HC: Therapy of Marfan syndrome. *Annu Rev Med* 59: 43-59, 2008.
- 3) Wheat MW, Wilson JR, Bartley TD: Successful replacement of the entire ascending aorta and aortic valve. *JAMA* 188: 717-719, 1964.
- 4) Bentall H, DeBono A: A technique for complete replacement of the ascending aorta. *Thorax* 23: 338-339, 1968.
- 5) Piehler JM, Pluth JR: Replacement of the ascending aorta and aortic valve with a composite graft in patients with nondisplaced coronary ostia. *Ann Thorac Surg* 33: 406-409, 1982.
- 6) Cabrol C, Pavie A, Mesnildrey P, et al: Long-term results with total replacement of the ascending aorta and reimplantation of the coronary arteries. *J Thorac Vasc Surg* 91: 17-25, 1986.
- 7) Kouchoukos NT, Marshall WG, Jr, Wedig-Stecher TA: Eleven-year experience with composite graft replacement of the ascending aorta and aortic valve. *J Thorac Cardiovasc Surg* 92: 691-705, 1986.
- 8) David TE, Feindel CM: An aortic valve-sparing operation for patients with aortic incompetence and aneurysm of the ascending aorta. *J Thorac Cardiovasc Surg* 103: 617-622, 1992.
- 9) Sarsam MAI, Yacoub M: Remodeling of the aortic valve annulus. *J Thorac Cardiovasc Surg* 105: 435-438, 1993.
- 10) David TE, Armstrong S, Maganti M, et al: Long-term results of aortic valve-sparing operations in patients with Marfan syndrome. *J Thorac Cardiovasc Surg* 138: 859-864, 2009.
- 11) Miller DC: Valve-sparing aortic root replacement in patients with the Marfan syndrome. *J Thorac Cardiovasc Surg* 125: 773-778, 2003.
- 12) Patel ND, Weiss ES, Alejo DE, et al: Aortic root operations for Marfan syndrome: a comparison of the Bentall and valve-sparing procedures. *Ann Thorac Surg* 85: 2003-2010, 2008.
- 13) Takamoto S, Nawata K, Morota T: A simple modification of 'David-V' aortic root reimplantation. *Eur J Cardiothorac Surg* 30: 560-562, 2006.
- 14) Taub CC, Stoler JM, Perez-Sanz T, et al: Mitral valve prolapse in Marfan syndrome: An old topic revisited. *Echocardiography* 26: 357-364, 2009.
- 15) Everitt MD, Pinto N, Hawkins JA, et al: Cardiovascular surgery in children with Marfan syndrome or Loeys-Dietz syndrome. *J Thorac Cardiovasc Surg* 137: 1327-1332, 2009.
- 16) Yetman AT, Bornemeier RA, McCrindle BW, et al: Long-term outcome in patients with Marfan syndrome: is aortic dissection the only cause of sudden death? *J Am Coll Cardiol* 41: 329-332, 2003.
- 17) Rybczynski M, Koschik DH, Aydin MA, et al: Tissue Doppler imaging identifies myocardial dysfunction in adults with Marfan syndrome. *Clin Cardiol* 30: 19-24, 2007.
- 18) Cooper DG, Walsh SR, Sadat U, et al: Treating the thoracic aorta in Marfan syndrome: surgery or TEVAR? *J Endovasc Ther* 16: 60-70, 2009.
- 19) Nordon IM, Hinchliffe RJ, Holt PJ, et al: Endovascular management of chronic aortic dissection in patients with Marfan syndrome. *J Vasc Surg* 50: 987-991, 2009.
- 20) Russo BL, La Palombara C, Rosati M, et al: Stent graft repair of descending aortic dissection in patients with Marfan syndrome: an effective alternative to open reoperation? *J Thorac Cardiovasc Surg* 138: 1108-1114, 2009.

Expert Opinion

1. Introduction
2. Coronary arterial disease and periodontitis
3. Peripheral arterial disease and periodontitis
4. Aortic aneurysm and periodontitis
5. Vitamin D is a key factor for periodontitis and cardiovascular diseases
6. Conclusion
7. Expert opinion

Periodontitis and cardiovascular diseases

Jun-ichi Suzuki[†], Norio Aoyama, Masahito Ogawa, Yasunobu Hirata, Yuichi Izumi, Ryoza Nagai & Mitsuaki Isobe

[†]University of Tokyo, Department of Advanced Clinical Science and Therapeutics, Tokyo, Japan

Periodontitis is characterized by gingival inflammation and periodontopathic bacteria generate immunological inflammatory responses. Recent epidemiological reports suggest that periodontitis is one of the key risk factors for the onset of cardiovascular diseases. Several studies reported that periodontal bacteria in cardiovascular specimens were frequently detected. We revealed that patients with acute coronary syndrome showed significantly higher serum IgG titers to a strain of periodontopathic bacteria compared with patients with chronic coronary disease. Periodontopathic bacteria were also present in a high percentage of specimens of diseased arteries from patients with Buerger disease or abdominal aortic aneurysm. Although periodontopathic bacteria may play a role in the development of cardiovascular diseases, the influence of these bacteria on the disease has not yet been proven. In this article, we review the relationship between periodontopathic pathogens and cardiovascular diseases to conduct further clinical and experimental investigations in near future.

Keywords: aorta, bacteria, cytokine, inflammation, periodontitis

Expert Opin. Ther. Targets (2010) 14(10):1023-1027

1. Introduction

Periodontitis is a chronic inflammatory disease that degrades the attachment apparatus of the teeth, leading to tooth loosening. Clinical signs of the disease are often seen in middle age and it is a very common disease in adults [1,2]. Epidemiological studies showed that periodontitis significantly increased the risk of cardiovascular disease (CVD) [3-6]. Although data was adjusted for known CVD risk factors such as smoking, diabetes, hypertension and socioeconomic conditions, other points might still explain the apparent association. Levels of risk markers for CVD have been reported to be elevated in patients with periodontitis. Furthermore, animal studies demonstrated an association between the prevalence of periodontal pathogens, bacterial products, periodontitis and the incidence of CVD-related events [7,8]. Although DNA from oral bacteria has been found in atherosclerotic plaque in animal experimental models [9] and humans [10], the contribution of these bacteria to plaque formation remains unknown. Periodontal pathogens and their products were reported to be a trigger of the atherosclerotic process in animal studies [7,8]. However, their effects in the human system remain unclear. The release of host-derived inflammatory mediators, such as cytokines from the chronically inflamed periodontal tissues into the circulation, may provide a link between periodontal disease and CVD [11,12]. Altered serological profiles of risk markers in patients with periodontitis may result from an invasion of bacteria. Additionally, entry of their products from the periodontal lesion into the blood stream and the consequential induction and maintenance of a chronic inflammatory state also contribute to the progression of CVD.

informa
healthcare

In this small article, we review pathological and immunological influence of periodontal pathogens to CVD and some promising methodologies for prevention of the disease.

2. Coronary arterial disease and periodontitis

Recent studies suggest that chronic inflammation plays an important role in the development of coronary arterial disease (CAD). Because periodontal disease is an enhancer of several chronic inflammatory factors such as MMPs [13-15], an etiological relationship between periodontal disease and CAD was proposed. For these reasons, there is strong interest in evaluating whether periodontal disease is independently associated with CAD [16-18]. Humphrey *et al.* revealed that periodontal disease is associated with increased risk of CAD using a meta-analysis [19]. They concluded that periodontal disease is a risk factor or marker for CAD, and is independent of traditional CAD risk factors. Nakajima *et al.* also revealed that periodontitis is associated with increased risk of CAD through dysfunction of endothelial cells, induced by either periodontopathic bacteria or their products [20]. CRP concentrations were higher among patients who subsequently developed myocardial infarction compared with those without the disease. However, there was no report to elucidate the relationship between specific gingival bacteria infection and CAD.

We recently revealed that there is an association between periodontitis and CAD, particularly acute coronary syndrome (ACS). A total of 28 CAD patients participated in the study. Coronary angiography, periodontal examination and dental radiography were performed in all patients. Subgingival plaque, saliva and blood samples were analyzed for the periodontopathogens *Aggregatibacter* (formerly *Actinobacillus*) *actinomycetemcomitans*, *Porphyromonas gingivalis*, *Tannerella forsythia*, *Treponema denticola*, and *Prevotella intermedia* using PCR. Specific serum antibody titers to the five periodontal pathogens were determined by ELISA. We found that 33% of the ACS patients harbored *A. actinomycetemcomitans* in oral samples, whereas no *A. actinomycetemcomitans* was found in the patients with chronic CAD. Furthermore, ACS patients showed significantly higher serum IgG titers to *A. actinomycetemcomitans* compared with chronic CAD patients. Thus, we concluded that a specific periodontal pathogens may play a crucial role in the development of ACS [21].

3. Peripheral arterial disease and periodontitis

There are several papers demonstrating the relationship between peripheral arterial disease (PAD) and periodontitis. Buhlin *et al.* revealed the association by determining the plasma levels of some risk markers for PAD in cases with periodontitis [22,23]. Statistical analyses revealed a significant association between periodontitis and high levels

of C-reactive protein (CRP), fibrinogen, IL-18 and antibodies against heat shock protein (Hsp) 65 and 70. They also showed the effect of infection control of periodontitis on the prevalence of the risk factors. One year after the initial treatment, IL-18 and other levels decreased. Thus, standard treatment for periodontal disease induces systemic changes in several biochemical markers that reflect the risk for PAD.

Chen *et al.* also revealed that periodontitis was associated with PAD using tissue specimens [24]. They identified *P. gingivalis*, *T. denticola*, *A. actinomycetemcomitans*, *P. intermedia* in tissue specimens taken from the anastomotic site of distal bypasses PCR. In the study, periodontopathic bacteria were detected in 52% of atherosclerotic specimens. Severe (Fontaine grade III or IV) patients showed higher detection frequency of *P. gingivalis* than mild (Fontaine grade II) patients. After adjusting for age, sex, diabetes and smoking, periodontitis increased fivefold the risk of having PAD. They also showed that periodontitis was associated with increased serum IL-6 and TNF- α concentrations.

Buerger disease also showed the significant relationship to periodontitis. Iwai *et al.* revealed that DNA of oral bacteria was detected in 13 of 14 arterial samples and all oral samples of patients with Buerger disease [25]. While *T. denticola* was found in 86% of the arterial samples, other pathogens were found in 14 to 43% of the samples. A pathological examination revealed that arterial specimens showed the characteristics of an intermediate-chronic-stage or chronic-stage lesion of Buerger disease. They reported that the patients with Buerger disease had high prevalence of severe periodontitis with higher serum IgG titers against *T. denticola*, *P. gingivalis* and *A. actinomycetemcomitans* [26]. They also found that the patients had increased titers of serum anti-cardiolipin antibody compared with healthy subjects [27]. These results suggest that periodontitis influences the development of PAD.

4. Aortic aneurysm and periodontitis

Abdominal aortic aneurysm (AAA) is a common and lethal disorder in the aging population [28,29]. Inflammation and MMPs appear to play a critical role in AAA development and progression [30]. Human AAA tissue samples demonstrated severe inflammatory infiltrates in both the media and adventitia [31,32]. An increased expression of MMPs has been observed in human aneurysm tissue specimens [33-37]. It is well known that MMPs play key roles in periodontal diseases. Periodontopathic bacteria generate host immunological inflammatory responses, thus resulting in the secretion of cytokines and MMPs [38], and eventually leading to the extracellular matrix destruction of the periodontal tissues [39]. Some studies reported the detection of periodontal bacteria in AAA specimens. Periodontopathic bacteria, especially *P. gingivalis* was present in a high percentage of specimens of AAA and were also found throughout the whole aneurysmal wall [40]. Thus, periodontopathic bacteria may play a role in the development of AAA, but the influence of these bacteria on the aneurysmal wall has not yet been

proven. To determine the effect of the periodontal microorganism on the AAA, we made a novel murine AAA model, which was produced by the periaortic application of 0.25 M CaCl₂. The mice received inoculations of either live *P. gingivalis*, *A. actinomycetemcomitans* or vehicle. Four weeks after the application of CaCl₂, the *P. gingivalis*-challenged mice showed a significant increase in the aortic diameter in comparison with the vehicle control mice while the *A. actinomycetemcomitans*-challenged mice showed no significant increase. Immunohistochemically, the CD8- and MOMA2-positive cells and the level of MMP-2 in the aneurysmal samples of *P. gingivalis*-challenged mice were also significantly higher than that inoculated with vehicle. We found that the *P. gingivalis*, but not *A. actinomycetemcomitans*, infection accelerated the progression of AAA due to the increased expression of MMPs (Aoyama N, unpublished).

5. Vitamin D is a key factor for periodontitis and cardiovascular diseases

Although vitamin D is well known to regulate calcium and phosphorus metabolism, it also has a physiological effects beyond its role in skeletal homeostasis. Recently, it was revealed that vitamin D is an immunomodulator which targets various immune cells, and modulates both innate and adaptive immune responses. Thus, vitamin D plays a crucial role in maintenance of immune homeostasis [41]. Several epidemiological studies have linked inadequate vitamin D levels to a higher susceptibility to immune-mediated disorders [42,43], including cardiovascular diseases [44]. It is believed that maintaining adequate vitamin D levels might in part prevent these common diseases [45]. It has been reported that low serum vitamin D levels were independently associated with

periodontal and cardiovascular diseases [46,47]. Notably, vitamin D insufficiency is associated with increased circulating CRP levels, and vitamin D supplementation decreases circulating CRP levels [48]. Thus, the elevated CRP levels observed in periodontal and cardiovascular disease might be a surrogate for vitamin D insufficiency.

6. Conclusion

In this brief article, we have demonstrated the relationship between periodontopathic pathogens and cardiovascular diseases. We have also elucidated that each gingival bacterium caused different condition of cardiovascular diseases.

7. Expert opinion

Although several periodontopathic bacteria play a serious role in the development of cardiovascular diseases, the influence of these bacteria has to be elucidated because of the lack of appropriate investigations. Thus, further experimental and clinical studies should be conducted to elucidate the pathophysiology and relationship between periodontitis and cardiovascular diseases. Meanwhile, clinicians should optimize the periodontal conditions in patients with cardiovascular risk factors for primary and/or secondary prevention. Finally, it is plausible that this simple treatment of periodontitis might provide as much or even more benefit than the standard treatments for cardiovascular diseases.

Declaration of interest

The authors state no conflict of interest and have received no payment in preparation of this manuscript.

Bibliography

Papers of special note have been highlighted as either of interest (●) or of considerable interest (●●) to readers.

1. Boehm TK, Scannapieco FA. The epidemiology, consequences and management of periodontal disease in older adults. *J Am Dent Assoc* 2007;138:26S-33S
2. Jain N, Jain GK, Javed S, et al. Recent approaches for the treatment of periodontitis. *Drug Discov Today* 2008;13:932-43
3. Friedewald VE, Kornman KS, Beck JD, et al. The American Journal of Cardiology and Journal of Periodontology Editors' Consensus: periodontitis and atherosclerotic cardiovascular disease. *Am J Cardiol* 2009;104:59-68
- **This article shows a standard consensus for physicians and dentists.**
4. Andriankaja OM, Genco RJ, Dorn J, et al. Periodontal disease and risk of myocardial infarction: the role of gender and smoking. *Eur J Epidemiol* 2007;22:699-705
5. Bahcekar AA, Singh S, Saha S, et al. The prevalence and incidence of coronary heart disease is significantly increased in periodontitis: a metaanalysis. *Am Heart J* 2007;154:830-7
6. Cairo F, Castellani S, Gori AM, et al. Severe periodontitis in young adults is associated with sub-clinical atherosclerosis. *J Clin Periodontol* 2008;35:465-72
7. Herzberg MC, Meyer MW. Dental plaque, platelets, and cardiovascular diseases. *Ann Periodontol* 1998;3:151-60
8. Dorn BR, Dunn WA, Progulski-Fox A. Invasion of human coronary artery cells by periodontal pathogens. *Infect Immun* 1999;67:5792-8
9. Zhang MZ, Li CL, Jiang YT, et al. Porphyromonas gingivalis infection accelerates intimal thickening in iliac arteries in a balloon-injured rabbit model. *J Periodontol* 2008;79:1192-9
10. Gaetti-Jardim E Jr, Marcelino SL, Feitosa AC, et al. Quantitative detection of periodontopathic bacteria in atherosclerotic plaques from coronary arteries. *J Med Microbiol* 2009;58:1568-75
11. Espinola-Klein C, Rupprecht HJ, Blankenberg S, et al. AtheroGene investigators. Impact of infectious burden on extent and long-term prognosis of atherosclerosis. *Circulation* 2002;105:15-21
12. Desvarieux M, Demmer RT, Rundek T, et al. Periodontal microbiota and carotid intima-media thickness: the Oral Infections and Vascular Disease Epidemiology Study (INVEST). *Circulation* 2005;111:576-82
13. Sorsa T, Tjaderhane L, Kontinen YT, et al. Matrix metalloproteinases: contribution to pathogenesis, diagnosis and treatment of periodontal inflammation. *Ann Med* 2006;38:306-21
14. Sorsa T, Tjaderhane L, Salo T. Matrix metalloproteinases (MMPs) in oral diseases. *Oral Dis* 2004;10:311-18
15. Olsen I. Update on bacteraemia related to dental procedures. *Transfus Apher Sci* 2008;39:173-8
16. Nakib SA, Pankow JS, Beck JD, et al. Periodontitis and coronary artery calcification: the Atherosclerosis Risk in Communities (ARIC) study. *J Periodontol* 2004;75:505-10
17. Higashi Y, Goto C, Hidaka T, et al. Oral infection-inflammatory pathway, periodontitis, is a risk factor for endothelial dysfunction in patients with coronary artery disease. *Atherosclerosis* 2009;206:604-10
18. Zaremba M, Gorska R, Suwalski P, Kowalski J. Evaluation of the incidence of periodontitis-associated bacteria in the atherosclerotic plaque of coronary blood vessels. *J Periodontol* 2007;78:322-7
19. Humphrey LL, Fu R, Buckley DI, et al. Periodontal disease and coronary heart disease incidence: a systematic review and meta-analysis. *J Gen Intern Med* 2008;23:2079-86
20. Nakajima T, Yamazaki K. Periodontal disease and risk of atherosclerotic coronary heart disease. *Odontology* 2009;97:84-91
21. Sakurai K, Wang D, Suzuki J, et al. High incidence of Actinobacillus actinomycetemcomitans infection in acute coronary syndrome. *Int Heart J* 2007;48:663-75
22. Buhlin K, Hultrin M, Norderyd O, et al. Risk factors for atherosclerosis in cases with severe periodontitis. *J Clin Periodontol* 2009;36:541-9
23. Buhlin K, Hultrin M, Norderyd O, et al. Periodontal treatment influences risk markers for atherosclerosis in patients with severe periodontitis. *Atherosclerosis* 2009;206:518-22
24. Chen YW, Umeda M, Nagasawa T, et al. Periodontitis may increase the risk of peripheral arterial disease. *Eur J Vasc Endovasc Surg* 2008;35:153-8
25. Iwai T, Inoue Y, Umeda M, et al. Oral bacteria in the occluded arteries of patients with Buerger disease. *J Vasc Surg* 2005;42:107-15
26. Chen YW, Iwai T, Umeda M, et al. Elevated IgG titers to periodontal pathogens related to Buerger disease. *Int J Cardiol* 2007;122:79-81
27. Chen YW, Nagasawa T, Wara-Aswapati N, et al. Association between periodontitis and anti-cardiolipin antibodies in Buerger disease. *J Clin Periodontol* 2009;36:830-5
28. Ernst CB. Abdominal aortic aneurysm. *N Engl J Med* 1993;328:1167-72
29. Alcorn HG, Wolfson SK Jr, Sutton-Tyrrell K, et al. Risk factors for abdominal aortic aneurysms in older adults enrolled in The Cardiovascular Health Study. *Arterioscler Thromb Vasc Biol* 1996;16:963-70
30. Longo GM, Xiong W, Greiner TC, et al. Matrix metalloproteinases 2 and 9 work in concert to produce aortic aneurysms. *J Clin Invest* 2002;110:625-32
31. Bobryshev YV, Lord RS, Parsson H. Immunophenotypic analysis of the aortic aneurysm wall suggests that vascular dendritic cells are involved in immune responses. *Cardiovasc Surg* 1998;6:240-9
32. Pearce WH, Koch AE. Cellular components and features of immune response in abdominal aortic aneurysms. *Ann NY Acad Sci* 1996;800:175-85
33. Freestone T, Turner RJ, Coady A, et al. Inflammation and matrix metalloproteinases in the enlarging abdominal aortic aneurysm. *Arterioscler Thromb Vasc Biol* 1995;15:1145-51

34. Davis V, Persidskaia R, Baca-Regen L, et al. Matrix metalloproteinase-2 production and its binding to the matrix are increased in abdominal aortic aneurysms. *Arterioscler Thromb Vasc Biol* 1998;18:1625-33
35. Rizas KD, Ippagunta N, Tilson MD III. Immune cells and molecular mediators in the pathogenesis of the abdominal aortic aneurysm. *Cardiol Rev* 2009;17:201-10
36. Takagi H, Manabe H, Kawai N, et al. Circulating matrix metalloproteinase-9 concentrations and abdominal aortic aneurysm presence: a meta-analysis. *Interact Cardiovasc Thorac Surg* 2009;9:437-40
37. Aziz F, Kuivaniemi H. Role of matrix metalloproteinase inhibitors in preventing abdominal aortic aneurysm. *Ann Vasc Surg* 2007;21:392-401
38. Salvi GE, Lang NP. Host response modulation in the management of periodontal diseases. *J Clin Periodontol* 2005;32(Suppl 6):108-29
39. Ashley RA. Clinical trials of a matrix metalloproteinase inhibitor in human periodontal disease. SDD Clinical Research Team. *Ann NY Acad Sci* 1999;878:335-46
40. Kurihara N, Inoue Y, Iwai T, et al. Detection and localization of periodontopathic bacteria in abdominal aortic aneurysms. *Eur J Vasc Endovasc Surg* 2004;28:553-8
41. Backe F, Takiishi T, Korf H, et al. Vitamin D: modulator of the immune system. *Curr Opin Pharmacol* 2010; published online 27 April 2010, doi:10.1016/j.coph.2010.04.001
42. Pilz S, Tomaschitz A, Obermayer-Pietsch B, et al. Epidemiology of vitamin D insufficiency and cancer mortality. *Anticancer Res* 2009;29:3699-704
43. Shoenfeld N, Amital H, Shoenfeld Y. The effect of melanism and vitamin D synthesis on the incidence of autoimmune disease. *Nat Clin Pract Rheumatol* 2009;5:99-105
44. Judd SE, Tangpricha V. Vitamin D deficiency and risk for cardiovascular disease. *Am J Med Sci* 2009;338:40-4
45. Goldstein MR, Mascitelli L, Pezzetta F. Periodontitis, atherosclerotic cardiovascular disease and vitamin D. *Am J Cardiol* 2009;104:1164
46. Dietrich T, Joshipura KJ, Dawson-Hughes B, et al. Association between serum concentrations of 25-hydroxyvitamin D3 and periodontal disease in the US population. *Am J Clin Nutr* 2004;80:108-13
47. Kendrick J, Targher G, Smits G, et al. 25-Hydroxyvitamin D deficiency is independently associated with cardiovascular disease in the Third National Health and Nutrition Examination Survey. *Atherosclerosis* 2009;205:255-60
48. Elliott P, Chambers JC, Zhang W, et al. Genetic loci associated with C-reactive protein levels and risk of coronary heart disease. *JAMA* 2009;302:37-48

Affiliation

Jun-ichi Suzuki¹, Norio Aoyama², Masahito Ogawa¹, Yasunobu Hirata¹, Yuichi Izumi², Ryozo Nagai⁴ & Mitsuaki Isobe³

¹Author for correspondence

¹University of Tokyo, Department of Advanced Clinical Science and Therapeutics, 7-3-1 Hongo, Bunkyo, Tokyo 113-8655, Japan
Tel: +81 3 5800 9116; Fax: +81 3 5800 9182;
E-mail: junichisuzuki-circ@umin.ac.jp

²Tokyo Medical and Dental University, Department of Periodontology and GCOE Program, Tokyo, Japan

³Tokyo Medical and Dental University, Department of Cardiovascular Medicine, Tokyo, Japan

⁴University of Tokyo, Department of Cardiovascular Medicine, Tokyo, Japan

Aorta Movement in Patients With Scoliosis After Posterior Surgery

Katsushi Takeshita, MD,* Toru Maruyama, MD,† Yusuke Nakao, MD,† Takashi Ono, MD,*
Yuki Taniguchi, MD,* Hirotaka Chikuda, MD,* Naoki Shoda, MD,* Yasushi Oshima, MD,*
Akio Higashikawa, MD,* and Kozo Nakamura, MD*

Study Design. Retrospective analysis.

Objective. To evaluate movement of the aorta in patients with scoliosis who have undergone the posterior correction and fusion.

Summary of Background Data. Surgeons check preoperative imaging for pedicle screw placement, but past analyses indicated that the aorta shifts after scoliosis surgery. Few studies, however, evaluated the aorta movement in detail.

Methods. A total of 22 patients with a right thoracic curve underwent posterior instrumentation and fusion. The average age at surgery was 17.2 years. The average of the preoperative Cobb angle was 65.2° which decreased to 20.0°.

Computed-tomographic data were analyzed by multiplanar reconstruction. In our coordinate system, the middle of the base of the left superior facet was set as the origin and a line connecting the middle points of both bases of the superior facets was defined as the X-axis. We defined the angle and the distance to describe the aorta position and analyzed the movement of the aorta relative to the spine. Deformity parameters were examined to determine their correlation with the aorta parameters.

We simulated variable pedicle screw placement and defined a warning pedicle when the aorta enters the expected area of the screw and examined them in 24 scenarios.

Results. The aorta moved 4.7 ± 3.0 mm on an average. The aorta had a tendency to migrate in the anteromedial direction and this movement correlated with preoperative apical vertebral translation, preoperative sagittal alignment, and change of sagittal alignment. The ratio of warning pedicles at the middle thoracic level (T7–T9) increased after deformity correction.

Conclusion. The aorta moved anteromedially relative to the spine after the posterior correction and the risk of the aorta by a pedicle screw increased by correction of the deformity at the middle thoracic spine. Surgeons are recommended to anticipate the aorta movement in the surgical planning.

Key words: scoliosis, pedicle screw, aorta, computed tomography. *Spine* 2010;35:E1571–E1576

Posterior correction and fusion by instrumentation is popular in the deformity surgery and pedicle screws have been the dominant anchors for the last decade. However, several authors^{1–3} reported a possible risk of aorta injury by a pedicle screw. Although surgeons use preoperative radiographic imaging in placing pedicle screws to prevent the aorta containment, the aorta may move after surgical correction of the spinal deformity. Few analyses of the movement of this organ after posterior surgery have been reported. The purpose of the present study was to evaluate the aorta movement after the posterior correction and fusion in scoliosis surgery.

Materials and Methods

A total of 37 patients with scoliosis underwent posterior instrumentation and fusion at the University Hospital between 2005 and 2007 and 22 patients with a right thoracic curve were included in this study. Scoliosis was idiopathic in 18 patients, Chiari-syrinx in 2, multiple epiphyseal dysplasia in 1, and Noonan syndrome in 1. A total of 15 patients were excluded: 5 patients with congenital scoliosis, 4 with idiopathic scoliosis with no thoracic curve, 3 with Marfan syndrome who might have had abnormal vascular movement, 2 with idiopathic scoliosis with left thoracic curve, and 1 with tubular sclerosis with left thoracic curve. Patient age at surgery was 10 to 29 (mean, 17.2) years old and 18 were women and 4 were men. Lenke's classification of scoliosis was type 1 in 8 patients, type 2 in 5, type 3 in 1, type 4 in 4, type 5 in 1, and type 6 in 3. The preoperative Cobb angle averaged $65.2 \pm 11.6^\circ$ (range, 50° – 88°) and corrected to $36.3 \pm 12.0^\circ$ (range, 18° – 70°) on bending films, and to $26.6 \pm 10.0^\circ$ (range, 13° – 44°) on fulcrum-bending films.⁴ The apex vertebra of the thoracic curve ranged from T5–T10 (T5:1, T7:2, T8:6, T9:5, T10:8). All patients were treated by posterior correction and fusion by pedicle screw instrumentation. The average number of instrumented vertebrae was 12.2 ± 1.6 (9–16 vertebrae). Postoperative Cobb angle averaged $20.0 \pm 7.7^\circ$ (range, 11° – 39°) and correction rate was $69.6 \pm 8.5\%$ (53%–83.1%). Cincinnati correction index⁵ was 1.74 ± 0.57 (0.95–3.38) and Fulcrum bending correction index⁶ was 1.18 ± 0.16 (0.78–1.48).

The patients were evaluated by computed tomography (CT) before and after surgery. Preoperative examination was for the computer-assisted placement of pedicle screws and the postoperative one was to confirm the location of pedicle screws. There was no need to replace any screw. The preoperative CT was obtained from the upper thoracic to the lower lumbar spine with a width of 1.25 mm as directed by a naviga-

From the *Department of Orthopaedic Surgery, the University of Tokyo, Tokyo, Japan; and the †Department of Orthopaedic Surgery, the Saitama Medical Center, Saitama, Japan.

Acknowledgment date: April 17, 2009. First revision date: August 8, 2009. Second revision date: November 23, 2009. Third revision date: November 30, 2009. Acceptance date: December 1, 2009.

The manuscript submitted does not contain information about medical device(s)/drug(s).

No funds were received in support of this work. Although one or more of the author(s) has/have received or will receive benefits for personal or professional use from a commercial party related directly or indirectly to the subject of this manuscript, benefits will be directed solely to a research fund, foundation, educational institution, or other non-profit organization which the author(s) has/have been associated.

Address correspondence and reprints requests to Katsushi Takeshita, MD, Department of Orthopaedic Surgery, The University of Tokyo, 7-3-1, Hongo, Bunkyo-ku, Tokyo, Japan 113-8655; E-mail: Takeshita-ort@h.u-tokyo.ac.jp

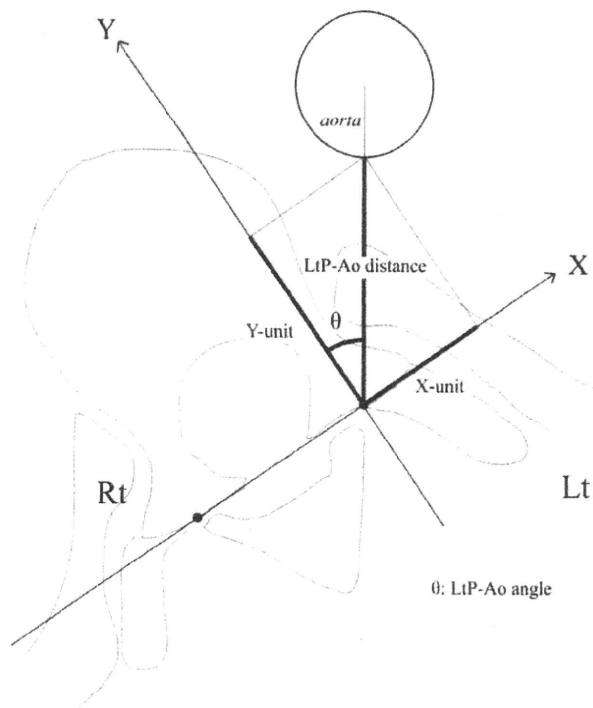


Figure 1. Aorta parameters. The origin is set in the middle of the base of the left superior facet. A line connecting the 2 middle points of both bases of the superior facets is defined as X-axis. LtP-Ao distance indicates the left pedicle-aorta distance; LtP-Ao angle, the left pedicle-aorta angle.

tion protocol, and the postoperative CT was obtained with a helical scan and developed with a width of 1.00 mm less than 2 weeks after surgery. All Digital Imaging and Communication in Medicine data were transferred to a personal computer and analyzed by Digital Imaging and Communication in Medicine or DICOM software (ExaView LITE: Ziosoft, Tokyo, Japan). In the present study, we used our original Cartesian coordinate system and measured parameters describing the location of the aorta from T4 to L4 of the 22 patients. We selected the middle of the base of the left superior facet as the point of origin of this coordinate system

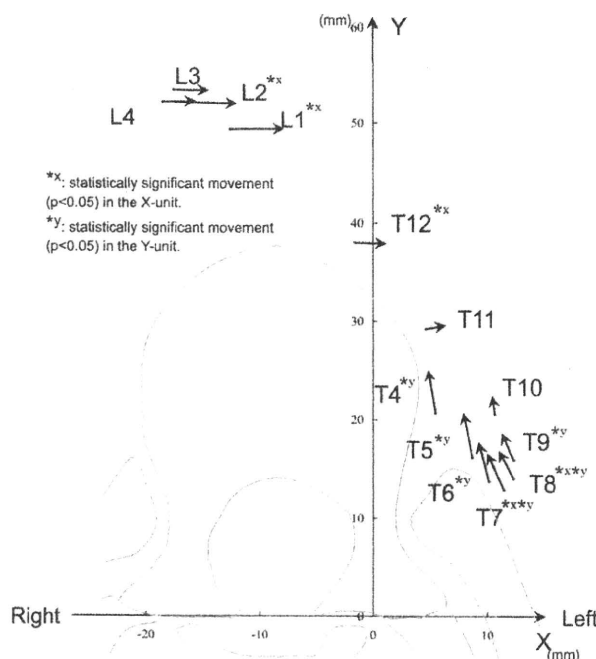


Figure 2. The aorta movement relative to the spine before and after posterior correction and fusion in our Cartesian coordinate system. The aorta moved to the anterior direction at the upper and middle thoracic levels and to the medial direction at the lower thoracic and lumbar levels.

(Figure 1) because the most probable threat to the aorta is by a pedicle screw on the left side at the thoracic spine. A line connecting the 2 middle points of both bases of the superior facets was defined as the X-axis; the Y-axis is determined to be parallel to the upper endplate of each vertebral body. The angle formed by the Y-axis and a line connecting the origin and the center of the aorta was defined as the left pedicle-aorta (LtP-Ao) angle and length of a line connecting the origin and the edge of the aorta as the LtP-Ao distance. Two parameters and the X- and Y-units of the LtP-Ao distance at 240 vertebral bodies were measured pre- and postsurgery after excluding vertebrae with incomplete data. From the repeatability test from our previous study,⁷ interclass corre-

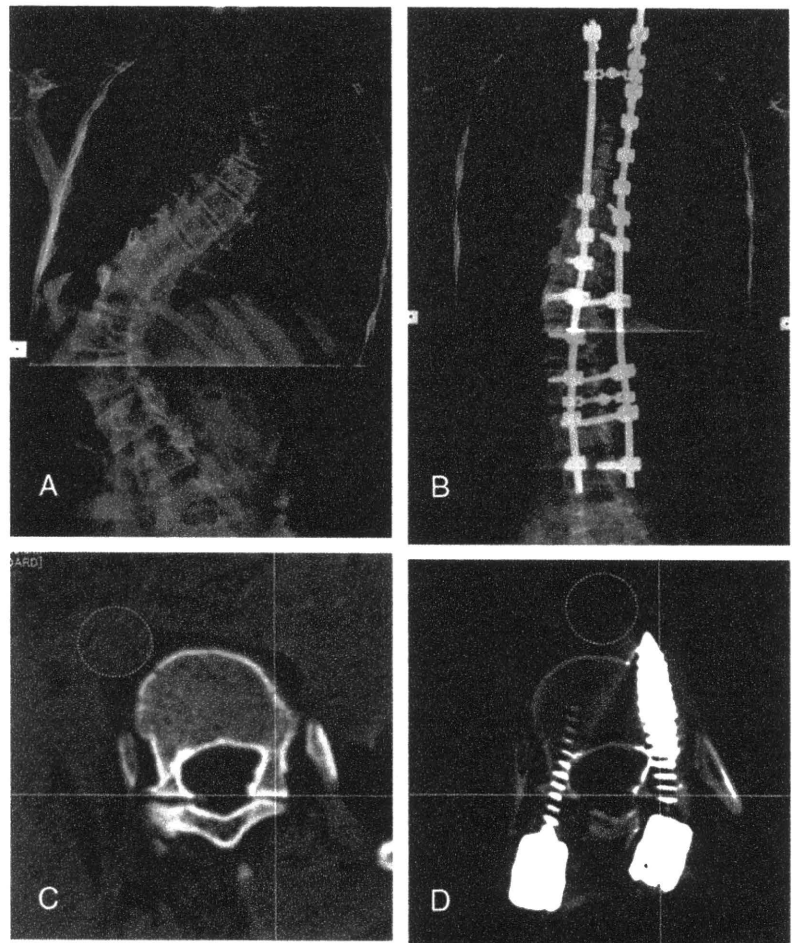
Table 1. Aorta Parameters Before and After Surgery

Level	n	LtAo-Angle (deg)			LtAo-Distance (mm)			Movement (mm)
		Preop	Postop	Change	Preop	Postop	Change	
T4	6	22.3 ± 30.9 (-6 to 65)	16.2 ± 24.6 (-18 to 55)	-4.1 ± 13.0 (-28 to 9)	22.0 ± 8.4 (11 to 32)	25.8 ± 8.3 (14 to 39)	3.6 ± 3.1* (-1 to 8)	5.9 ± 4.2 (1 to 12)
T5	20	30.8 ± 21.0 (-1 to 78)	23.8 ± 18.0 (-10 to 67)	-7.2 ± 8.9* (-23 to 12)	19.9 ± 4.7 (12 to 32)	23.6 ± 6.0 (14 to 37)	4.3 ± 3.7* (-1 to 14)	5.4 ± 2.9 (1 to 13)
T6	22	37.3 ± 16.8 (9 to 74)	30.2 ± 14.2 (3 to 59)	-7.1 ± 9.1* (-22 to 8)	18.1 ± 3.7 (13 to 28)	20.7 ± 5.0 (11 to 33)	2.6 ± 2.7* (-2 to 9)	4.6 ± 2.7 (1 to 13)
T7	22	42.3 ± 14.0 (17 to 74)	32.6 ± 11.4 (12 to 59)	-10.0 ± 7.7* (-22 to 3)	17.9 ± 3.7 (12 to 26)	19.7 ± 4.5 (12 to 33)	1.9 ± 2.6* (-3 to 7)	4.1 ± 2.6 (0 to 9)
T8	22	42.4 ± 11.1 (16 to 65)	34.5 ± 8.5 (20 to 55)	-7.9 ± 7.1* (-20 to 4)	19.2 ± 4.3 (13 to 30)	20.2 ± 3.7 (14 to 29)	1.0 ± 2.6 (-6 to 4)	3.9 ± 2.1 (1 to 8)
T9	22	39.2 ± 11.5 (13 to 53)	32.5 ± 8.8 (9 to 46)	-6.8 ± 7.0* (-23 to 7)	20.8 ± 4.9 (14 to 32)	21.8 ± 4.2 (16 to 31)	1.0 ± 2.4 (-6 to 5)	3.8 ± 2.2 (1 to 9)
T10	22	29.9 ± 14.1 (-13 to 51)	27.1 ± 10.3 (0 to 49)	-2.7 ± 7.5 (-15 to 13)	24.1 ± 6.3 (15 to 36)	25.0 ± 5.2 (17 to 34)	0.9 ± 3.2 (-5 to 6)	4.3 ± 2.8 (0 to 10)
T11	22	13.0 ± 20.4 (-46 to 44)	13.9 ± 13.6 (-21 to 37)	0.8 ± 10.0 (-17 to 25)	31.8 ± 8.0 (19 to 46)	31.3 ± 6.7 (20 to 43)	-0.5 ± 3.1 (-7 to 4)	5.1 ± 4.4 (0 to 19)
T12	21	0.4 ± 16.2 (-26 to 28)	3.7 ± 13.2 (-18 to 34)	3.3 ± 6.4* (-10 to 13)	39.4 ± 8.1 (25 to 52)	38.2 ± 7.4 (25 to 55)	-1.0 ± 2.5 (-7 to 4)	5.0 ± 3.0 (1 to 10)
L1	21	-12.6 ± 14.1 (-36 to 13)	-8.0 ± 10.6 (-29 to 9)	4.8 ± 5.5* (-8 to 12)	50.3 ± 6.8 (36 to 63)	49.0 ± 6.2 (39 to 59)	-1.5 ± 3.3* (-7 to 6)	6.0 ± 3.1 (1 to 13)
L2	21	-16.0 ± 10.3 (-38 to 2)	-12.0 ± 8.8 (-28 to 3)	4.1 ± 4.6* (-3 to 14)	54.5 ± 4.5 (44 to 61)	52.8 ± 4.4 (44 to 61)	-1.6 ± 1.5* (-7 to 2)	5.2 ± 3.2 (2 to 14)
L3	14	-17.6 ± 5.2 (-27 to -10)	-14.0 ± 5.3 (-23 to -5)	2.6 ± 4.0* (-4 to 8)	55.4 ± 4.5 (47 to 63)	54.4 ± 4.3 (48 to 62)	-1.0 ± 2.9 (-5 to 4)	4.4 ± 2.6 (1 to 8)
L4	5	-19.6 ± 5.9 (-28 to -12)	-17.0 ± 4.6 (-25 to -13)	2.4 ± 3.0 (-1 to 7)	53.2 ± 3.3 (48 to 57)	53.2 ± 4.7 (48 to 59)	0.0 ± 3.1 (-3 to 4)	3.9 ± 2.0 (2 to 7)
Total	240	17.9 ± 27.4 (-46 to 78)	15.4 ± 21.6 (-29 to 67)	-2.5 ± 9.1 (-28 to 25)	31.3 ± 15.3 (11 to 63)	31.9 ± 13.9 (11 to 62)	0.7 ± 3.3 (-7 to 14)	4.8 ± 3.2 (0 to 20)

*P < 0.01.

LtAo-Angle indicates left pedicle-aorta angle; LtAo-Distance, left pedicle-aorta distance; Preop, preoperative; Postop, postoperative.

Figure 3. **A**, Standing anteroposterior spinal radiograph of a 14-year-old girl with a 69° right thoracic curve and 88° left lumbar curve. She had only 11 thoracic vertebrae (no T12) and the apical vertebral translation was 69.5 mm. She had had foraminal magnum decompression and duroplasty 16 months before spinal surgery. **B**, Standing coronal radiograph 2 weeks after segmental pedicle screw instrumentation from T2 to L4 demonstrating thoracic curve correction to 19° and lumbar curve correction to 25°. The apical vertebral translation decreased to 30.1 mm. **C**, Preoperative computed tomography by multiplanar reconstruction at T11. The aorta (dotted circle) located in front of the right side of the vertebral body. **D**, Postoperative computed tomography adjusted by multiplanar reconstruction to match the preoperative imaging. The aorta (dotted circle) had moved 19.5 mm to the bicortical pedicle screw of the left side.



lation coefficients were 0.922 to 0.957 in the intraobserver measurement and 0.896 to 0.929 (0.864–0.961) in the interobserver measurement.

We analyzed the movement of the aorta relative to the spine in each level. To determine the relationship between the thoracic main curve and thoracic coronal/sagittal alignment, we selected patients who had their main curve in the thoracic spine. In the 17 selected patients, we measured the Cobb angle and the apical vertebral translations (AVT) of the main curve, and the sagittal alignment (the Cobb angle at T5–T12) before and after surgery. These deformity parameters were examined for their correlation, with the maximum movement of the aorta in the main thoracic curve. Statistical analysis was performed by SPSS 17.0 (SPSS Inc., Chicago, IL).

We simulated placement of the pedicle screw with a direction different from the ideal trajectory. Sensitivity analysis was performed by varying the direction error and the length of the screw independently. The direction error started from 10° up to 30° with 10° increments (3 scenarios). The length of the screw started from 25 to 40 mm with increments of 5 mm (4 scenarios). Therefore, we set up total of 24 scenarios in the preoperative or postoperative state. We defined a warning pedicle as being when the aorta enters the expected area of the screw. Ratio between the number of warning pedicles and the number of the examined pedicles at 1 spine level was calculated from T4 to L4 in every scenario.

■ Results

The LtP-Ao angle changed significantly from T5 to T9 and from T12 to L3 (*t* test, $P < 0.01$) (Table 1), whereas the LtP-Ao distance changed significantly from T4–T7, L1 and L2. The average of the aorta movement in the examined 240 vertebrae was 4.7 ± 3.0 mm. The aorta moved more than 10 mm in 14 vertebrae (5.8%), and had a tendency to migrate in the anteromedial direction (Figure 2). A representative case is shown in Figure 3.

In the 17 patients who had the main curve in the thoracic spine, the maximum movement of the aorta in the main thoracic curve was 8.9 ± 3.5 mm (range, 3.9–14.9). Level of the maximum movement was T4 in 1 case, T5 in 1, T6 in 1, T7 in 2, T9 in 1, T10 in 3, T11 in 1, T12 in 1, L1 in 3, L2 in 2, L3 in 0, and L4 in 1. Level of the maximum movement was periapical (± 1 vertebra of the apex) in 8 cases. The maximum movement of the aorta correlated with the preoperative AVT (Pearson correlation coefficient, -0.55 ; $P = 0.02$), preoperative sagittal alignment (-0.52 , $P = 0.03$), and change of the sagittal alignment (0.57 , $P = 0.02$) (Figure 4).

Sensitivity analysis (Tables 2–4) revealed that long pedicle screw (40 mm) with moderate direction error

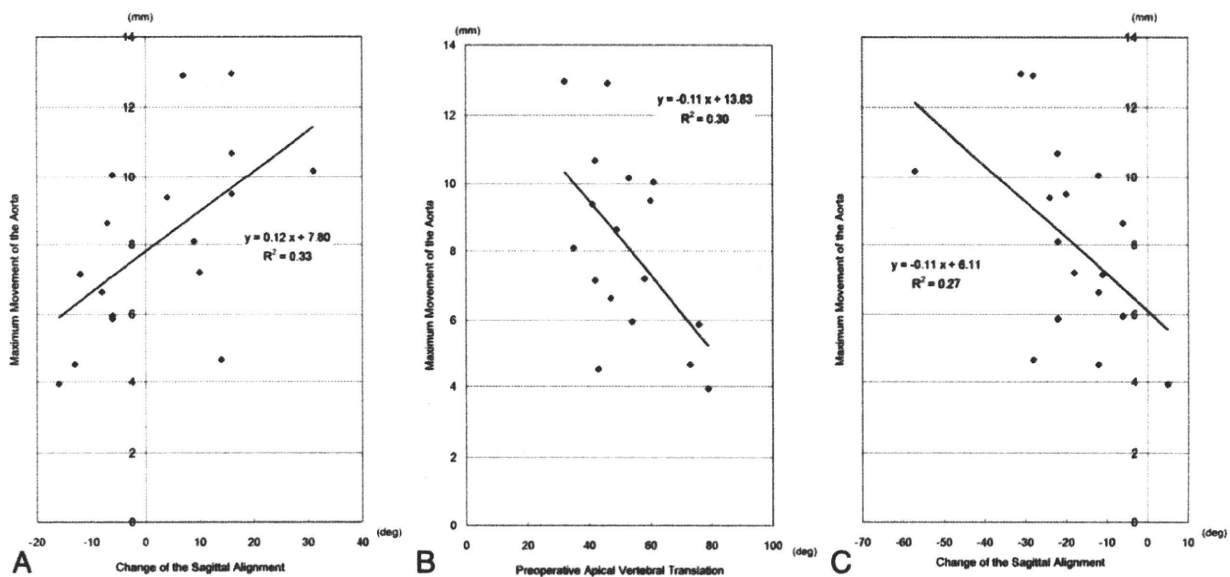


Figure 4. **A**, Relationship between change of the sagittal alignment (T5–T12) and the maximum aorta movement in the main thoracic curve of 17 patients. Positive change denotes more kyphotic change. Pearson correlative coefficient was 0.571 ($P = 0.017$); *i.e.*, more correction of the main curve meant greater aorta movement. **B**, Relationship between the postoperative apical vertebral translation and the maximum aorta movement. Pearson correlative coefficient was -0.550 ($P = 0.022$); *i.e.*, lower apical vertebral translation after surgery meant greater aorta movement. **C**, Relationship between the preoperative sagittal alignment (T5–T12) and the maximum aorta movement in the main thoracic curve of 17 patients. Positive change denotes more kyphotic change. Pearson correlative coefficient was -0.521 ($P = 0.032$); *i.e.*, more correction of the main curve meant larger aorta movement.

imposed risk on the aorta, but a large direction error (30°) by itself put the aorta at a high risk in any spine level regardless of the length of the pedicle screw.

There were only 5 warning pedicles in 96 examined lumbar spine and all were first lumbar spine. Therefore, further analysis was limited only in the thoracic spine. The distribution of the warning pedicles revealed 3 groups. The middle thoracic level (T7–T9) had a low ratio of warning pedicles before surgery, and the ratio increased statistically significantly after deformity correction. The upper thoracic level (T4–T6) as well as the lower thoracic level (T10–T12) had a moderate ratio of warning pedicles before surgery, and did not change considerably after deformity correction.

■ Discussion

The present study revealed that the aorta moved more than 10 mm in 17 of the examined 240 spines (5.8%), with a shift to the anterior and medial positions after posterior surgery. From our previous study,⁸ the aorta may be at risk at left concave pedicle at T4, T5, and T10–T12 before surgery, and there was a relative safety of the aorta for pedicle screw placement at the apical level. The present study showed that the dangerous pedicle ratio increased at the midthoracic level after surgery. At the apical level, the aorta often resides far lateral of the vertebral body which is far from the axis of the trunk. Surgeons assume from the preoperative imaging that the

Table 2. Warning Pedicles—Direction Error Within 10°

Screw Length	Level	Preoperative			Postoperative			Difference
		Warning (+)*	Warning (-)†	Ratio	Warning (+)*	Warning (-)†	Ratio	
25 mm	T4–T6	2	46	4.2%	0	48	0.0%	-4.2%
25 mm	T7–T9	0	66	0.0%	0	66	0.0%	0
25 mm	T10–T12	0	65	0.0%	0	65	0.0%	0
30 mm	T4–T6	5	43	10.4%	2	46	4.2%	-6.2%
30 mm	T7–T9	0	66	0.0%	1	65	1.5%	+1.5%
30 mm	T10–T12	0	65	0.0%	1	64	1.5%	+1.5%
35 mm	T4–T6	7	41	14.6%	5	43	10.4%	-4.2%
35 mm	T7–T9	0	66	0.0%	1	65	1.5%	+1.5%
35 mm	T10–T12	4	61	6.2%	6	59	9.2%	+3.0%
40 mm	T4–T6	7	41	14.6%	5	43	10.4%	-4.2%
40 mm	T7–T9	0	66	0.0%	1	65	1.5%	+1.5%
40 mm	T10–T12	8	57	12.3%	11	54	16.9%	+4.6%

*Number of the warning pedicles.
†Number of the nonwarning pedicles.

Table 3. Warning Pedicles—Direction Error Within 20°

Screw Length	Level	Preoperative			Postoperative			Difference
		Warning (+)*	Warning (-)†	Ratio	Warning (+)*	Warning (-)†	Ratio	
25 mm	T4–T6	7	41	14.6%	5	43	10.4%	-4.2%
25 mm	T7–T9	1	65	1.5%	0	66	0.0%	-1.5%
25 mm	T10–T12	1	64	1.5%	1	64	1.5%	0
30 mm	T4–T6	11	37	22.9%	12	36	25.0%	+2.1%
30 mm	T7–T9	2	64	3.0%	1	65	1.5%	-1.5%
30 mm	T10–T12	1	64	1.5%	5	60	7.7%	+6.2%
35 mm	T4–T6	13	35	27.1%	16	32	33.3%	+6.2%
35 mm	T7–T9	4	62	6.1%	2	64	3.0%	-3.1%
35 mm	T10–T12	10	55	15.4%	14	51	21.5%	+6.1%
40 mm	T4–T6	13	35	27.1%	18	30	37.5%	+10.4%
40 mm	T7–T9	4	62	6.1%	2	64	3.0%	-3.1%
40 mm	T10–T12	19	46	29.2%	21	44	32.3%	+3.1%

*Number of the warning pedicles.

†Number of the nonwarning pedicles.

aorta stays out of the spine and become less careful of this organ during screw placement. In fact, after correction of the scoliosis in some cases, the vertebrae return to a more physiologic position, which is the center of the body: this movement of the spine results in the medialization of the aorta relative to the spine and the risk of the aorta by a pedicle screw increased by correction of the deformity at the middle thoracic spine. Accordingly, all left pedicles have substantial risk of indenting the aorta indentation if a pedicle screw breaches outside the pedicle.

Few authors have reported change of the aorta position after deformity surgery. The first analysis was reported by Bullmann *et al.*⁹ They analyzed the aorta movement in their experience of anterior surgery and found that the aorta migrates from a more posterolateral to a more anteromedial position in relation to the thoracic vertebrae. However, patients were scanned in supine position for preoperative CT and in a lateral decubitus position for postoperative magnetic resonance imaging. As the aorta location depends on the patient position at examination especially in the midthoracic

level as clearly shown by the study of Huitema *et al.*¹⁰ the aorta movement in Bullman's report may come from a difference in the patient's position in the 2 examinations.

Recently, Wang *et al.*¹¹ analyzed the change of the position of the aorta after anterior or posterior instrumentation of type I Lenke curve and concluded that the aorta moved more in anterior surgery than in posterior surgery. They measured by 2 methods: one was from the aorta to the closest point of the cortex of the vertebral body and the other was from the posterior wall of the aorta to the anterior edge of the left rib head, neither of which was associated with the pedicle screw impingement. They measured 2 angles which were not suitable to describe the aorta position as for pedicle screw placement. Accordingly, parameters they adopted could not clarify the aorta movement relative to the spine, as do our results.

The present analysis indicated that the aorta position has a relationship with the curve characteristics of spinal deformity. The aorta movement highly correlated with the deformity characteristics: change of the sagittal alignment, preoperative AVT and sagittal alignment. Therefore, the

Table 4. Warning Pedicles—Direction Error Within 30°

Screw Length	Level	Preoperative			Postoperative			Difference
		Warning (+)*	Warning (-)†	Ratio	Warning (+)*	Warning (-)†	Ratio	
25 mm	T4–T6	15	33	31.3%	15	33	31.3%	0
25 mm	T7–T9	3	63	4.5%	15	51	22.7%	+18.2%‡
25 mm	T10–T12	6	59	9.2%	8	57	12.3%	+3.1%
30 mm	T4–T6	19	29	39.6%	23	25	47.9%	+8.3%
30 mm	T7–T9	9	57	13.6%	22	44	33.3%	+19.7%§
30 mm	T10–T12	14	51	21.5%	18	47	27.7%	+6.2%
35 mm	T4–T6	21	27	43.8%	27	21	56.3%	+12.5%
35 mm	T7–T9	11	55	16.7%	23	43	34.8%	+18.1%§
35 mm	T10–T12	24	41	36.9%	30	35	46.2%	+9.3%
40 mm	T4–T6	21	27	43.8%	29	19	60.4%	+16.6%
40 mm	T7–T9	11	55	16.7%	23	43	34.8%	+18.1%§
40 mm	T10–T12	33	32	50.8%	38	27	58.5%	+7.7%

*Number of the warning pedicles.

†Number of the nonwarning pedicles.

‡ $P < 0.01$ (Fisher exact test).§ $P < 0.05$.

degree of the aorta movement may be estimated from preoperative deformity and the degree of correction.

We did not measure and analyze the rotation of the spine as most CT did not include the pelvis because of the retrospective nature of this study. Accordingly, we could not estimate the effect of derotation.

In summary, the aorta moved anteromedially relative to the spine after the posterior correction and the risk of the aorta by a pedicle screw increased by correction of the deformity at the middle thoracic spine. Surgeons are recommended to anticipate the aorta movement in the surgical planning.

■ Key Points

- We evaluated the aorta positions before and after scoliosis surgery by multiplanar reconstruction of computed tomography.
- The aorta had a tendency to migrate to the anteromedial direction after corrective surgery of the scoliosis.
- The risk of the aorta by a pedicle screw increases by correction of the deformity at the middle thoracic spine.

References

1. Kakkos SK, Shepard AD. Delayed presentation of aortic injury by pedicle screws: report of two cases and review of the literature. *J Vasc Surg* 2008; 47:1074–82.
2. Minor ME, Morrissey NJ, Peress R, et al. Endovascular treatment of an iatrogenic thoracic aortic injury after spinal instrumentation: case report. *J Vasc Surg* 2004;39:893–6.
3. Wegener B, Birkenmaier C, Fortner A, et al. Delayed perforation of the aorta by a thoracic pedicle screw. *Eur Spine J* 2008;17(suppl 2):S351–4.
4. Cheung KM, Luk KD. Prediction of correction of scoliosis with use of the fulcrum bending radiograph. *J Bone Joint Surg Am* 1997;79:1144–50.
5. Vora V, Crawford A, Babekhir N, et al. A pedicle screw construct gives an enhanced posterior correction of adolescent idiopathic scoliosis when compared with other constructs: myth or reality. *Spine* 2007;32:1869–74.
6. Luk KD, Cheung KM, Lu DS, et al. Assessment of scoliosis correction in relation to flexibility using the fulcrum bending correction index. *Spine* 1998; 23:2303–7.
7. Takeshita K, Maruyama T, Chikuda H, et al. Diameter, length, and direction of pedicle screws for scoliotic spine- analysis by multiplanar reconstruction of computed tomography. *Spine* 2009 59 34:798–803.
8. Takeshita K, Maruyama T, Chikuda H, et al. New parameters to represent the position of the aorta relative to the spine for pedicle screw placement. Paper presented at: The Scoliosis Research Society Annual Meeting; 2008; Salt Lake City, UT.
9. Bullmann V, Fallenberg EM, Meier N, et al. The position of the aorta relative to the spine before and after anterior instrumentation in right thoracic scoliosis. *Spine* 2006;31:1706–13.
10. Huitema GC, Cormips EM, Castelijns MH, et al. The position of the aorta relative to the spine: is it mobile or not? *Spine* 2007 20;32:1259–64.
11. Wang W, Zhu Z, Zhu F, et al. The changes of relative position of the thoracic aorta after anterior or posterior instrumentation of type I Lenke curve in adolescent idiopathic thoracic scoliosis. *Eur Spine J* 2008;17:1019–26.

New parameters to represent the position of the aorta relative to the spine for pedicle screw placement

Katsushi Takeshita · Toru Maruyama · Takashi Ono · Satoshi Ogihara · Hiroataka Chikuda · Naoki Shoda · Yusuke Nakao · Ko Matsudaira · Atsushi Seichi · Kozo Nakamura

Received: 26 January 2009 / Revised: 13 January 2010 / Accepted: 16 January 2010 / Published online: 4 February 2010
© Springer-Verlag 2010

Abstract Parameters of the position of the aorta in previous reports were determined for anterior surgery. This study evaluated the relative position of the aorta to the spine by new parameters, which could enhance the safety of pedicle screw placement. Three parameters were defined in a new Cartesian coordinate system. We selected an entry point of a left pedicle screw as the origin. The transverse plane was determined to include both the bases of the superior facet and to be parallel to the upper endplate of the vertebral body. A line connecting the entry points of both sides was defined as the *X*-axis. The angle formed by the *Y*-axis and a line connecting the origin and the center of the aorta was defined as the left pedicle–aorta angle. The length of a line connecting the origin and the aorta edge was defined as the left pedicle–aorta distance. Distance from the edge of the aorta to the *X*-axis was defined as the pedicular line–aorta distance. These parameters were measured preoperatively in 293 vertebral bodies of 24 patients with a right thoracic curve. We simulated the placement of the pedicle screw with variable length and with some direction error. We defined a warning pedicle as that when the aorta enters the expected area of the screw.

Sensitivity analysis was performed to find the warning pedicle ratio in 12 scenarios. The left pedicle–aorta angle averaged 29.7° at the thoracic spine and -16.3° at the lumbar spine; the left pedicle–aorta distance averaged 23.7 and 55.2 mm; the pedicular line–aorta distance averaged 18.3 and 51.0 mm, respectively. The ratio of warning pedicles was consistently high at T4–5 and T10–12. When a left pedicle screw perforates an anterior/lateral wall of the vertebral body, the aorta may be at risk. These new parameters enable surgeons to intuitively understand the position of the aorta in surgical planning or in placement of a pedicle screw.

Keywords Scoliosis · Pedicle screw · Aorta · Computed tomography

Introduction

Several authors have reported serious injuries of the aorta due to inappropriate placement of screws or plates in anterior surgery [8, 9]. Sucato et al. [10] reported that 12% (13/106) of vertebral screws in right thoracic scoliosis created some contour defect in the aorta on the contralateral side of the vertebral body, although patients had no sequela. They subsequently analyzed the position of the aorta in patients with scoliosis compared to those with non-scoliotic spine and found that the aorta often resides on the lateral side of the vertebral body and concluded that a potential risk of the aorta by a vertebral screw increases in the scoliotic spine. Maruyama et al. [7] studied the spatial relations between the spine and the aorta in adolescent idiopathic scoliosis and concluded that the aorta can be located in the direction of the screw passage in 33 of 40 vertebrae (83%) between T6 and T9. These studies,

K. Takeshita (✉) · T. Ono · S. Ogihara · H. Chikuda · N. Shoda · Y. Nakao · K. Matsudaira · K. Nakamura
Department of Orthopaedic Surgery, Faculty of Medicine,
The University of Tokyo, 7-3-1, Hongo, Bunkyo-ku,
Tokyo 113-8655, Japan
e-mail: Takeshita-ort@h.u-tokyo.ac.jp; dtstake@coral.ocn.ne.jp

T. Maruyama
Department of Orthopaedic Surgery,
The Saitama Medical Center, Saitama, Japan

A. Seichi
Department of Orthopaedics, Jichi Medical University,
Tochigi, Japan

however, paid less attention to the relationship between the aorta and a pedicle screw. Accordingly, parameters describing the position of the aorta in these reports were not intuitive and surgeons have had difficulty utilizing these values in posterior surgery. The purpose of the present study was to evaluate the relative position of the aorta to the spine by new parameters, which can enhance the safety of pedicle screw placement.

Materials and methods

Thirty-seven patients with scoliosis underwent posterior instrumentation and fusion at the University Hospital from 2005 to 2007. Patients with congenital scoliosis were excluded. A total of 24 patients with a right thoracic curve were included in this study. Scoliosis was idiopathic in 17 patients, Chiari-syrinx in 2, Marfan syndrome in 2, multiple epiphyseal dysplasia in 1, Noonan syndrome in 1, and tuberous sclerosis in 1. Age at surgery was 10–29 (mean 17.1) years; 19 patients were women and 5 were men. Lenke's classification of scoliosis [4] was type 1 in eight patients, type 2 in five, type 3 in two, type 4 in four, type 5 in one, and type 6 in four. Preoperative Cobb angle averaged 66.4° (50° – 103°). The apex vertebra ranged from T5 to T10 (median T10). All patients were treated by posterior correction and fusion by pedicle screw instrumentation. One patient with a curve of 103° had undergone anterior release before posterior spinal fusion. Computed tomography was taken before surgery and pedicle screws were placed with guidance of the CT-based navigation system. Postoperative Cobb angle averaged 20.3° (11° – 39°).

A computer tomography was taken from the upper thoracic to the lower lumbar spine with a width of 1.25 mm for navigation. All DICOM data were transferred to a personal computer and analyzed by DICOM software (ExaView LITE: ©Ziosoft, Tokyo, Japan). We defined three parameters in a new Cartesian coordinate system and those parameters from T4 to L4 were measured in 293 vertebral bodies of 24 patients. We selected the middle of the base of the left superior facet as the origin of this coordinate system (Fig. 1), because the most probable threat to the aorta by a pedicle screw is on the left side at the thoracic spine. The transverse plane was determined to include both the bases of the superior facet and to be parallel to the upper endplate of the vertebral body. A line connecting the middle points of both bases of the superior facets is defined as the pedicular line (PL) (X-axis). The Y-axis perpendicular to the X-axis is drawn ventrally from the origin. The angle formed by the Y-axis and a line connecting the origin and the center of the aorta is defined as the left pedicle–aorta angle length of a line connecting the origin and the edge of the aorta as the left pedicle–aorta

distance, and distance from the edge of the aorta to the X-axis as the pedicular line–aorta distance. Moreover, we break down the left pedicle–aorta distance into the X- and Y-unit. The X-unit is the rectangular component of the left pedicle–aorta distance to the X-axis and the Y-unit is that to the Y-axis.

We simulated placement of the pedicle screw with a direction different from the ideal trajectory. Sensitivity analysis was performed by changing the direction error and the length of the screw independently. The direction error started from 10° up to 30° with 10° increments (three scenarios). The length of the screw started from 25 to 40 mm with increments of 5 mm (four scenarios). We set up a total of 12 scenarios (three by four). We defined a warning pedicle as that when the aorta enters the expected area of the screw. The ratio of warning pedicles was calculated from T4 to L4 in the 12 scenarios (Fig. 2). From the repeatability test from our previous study, interclass correlation coefficients were 0.922–0.957 in the intraobserver measurement and 0.896–0.929 (0.864–0.961) in the interobserver measurement [12].

To determine the relationship of the location of the aorta and the characteristics of scoliosis, the Cobb angle of the main thoracic curve, apical vertebral translations of the main thoracic curve, and the angle at T5–T12 in the sagittal plane were measured, and correlations between these

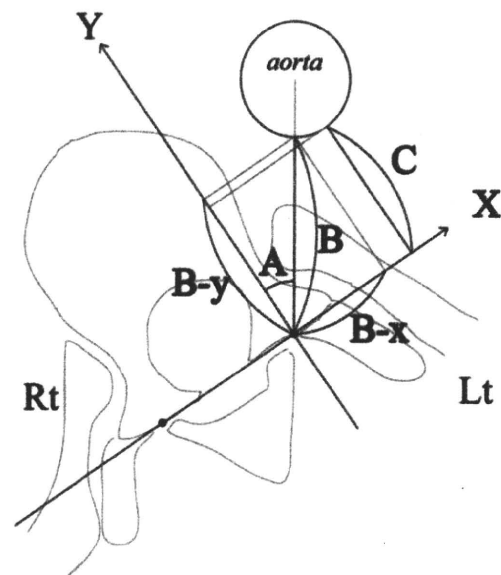


Fig. 1 Measurement of new parameters. The origin is set at the middle of the base of the left superior facet. A line joining the middle points of both bases of the superior facets is defined as the X-axis (the pedicular line). The Y-axis, perpendicular to the X-axis, is drawn ventrally from the origin. **A** The left pedicle–aorta angle. **B** The left pedicle–aorta distance. **B-x** the X-unit of the left pedicle–aorta distance. **B-y** the Y-unit of the left pedicle–aorta distance. **C** the pedicular line–aorta distance

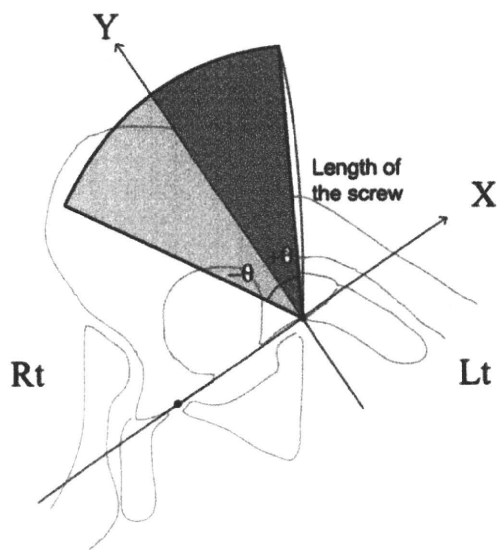


Fig. 2 The expected area of the pedicle screw. We simulated the pedicle screw placement with some direction error ($\pm\theta$) and the variable length (length of the screw) with sensitivity analysis. We defined a warning pedicle as that when the aorta enters this zone

parameters and the X-and Y-unit of the left pedicle–aorta line at the apex were calculated.

Results

The relative position of the aorta to the spine changed dramatically at the thoracic spine (Table 1; Fig. 3). The left pedicle–aorta angle spanned from -46° to 78° (average 29.7°) at the thoracic spine and from -38° to 13° (average -16.3°) at the lumbar spine; the left pedicle–

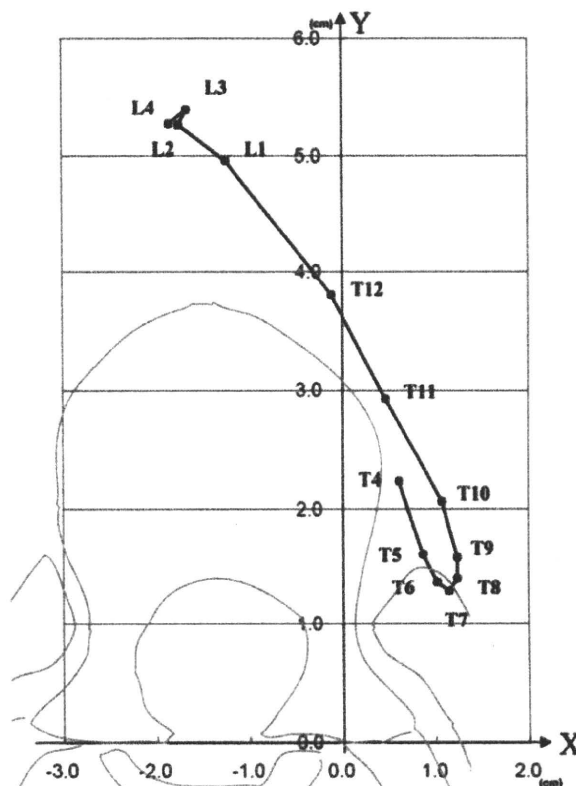


Fig. 3 The average course of the aorta relative to the spine. The origin is set at the middle of the base of the left superior facet. A line joining the middle points of both bases of the superior facets is defined as X-axis. The aorta begins to be seen from T4. It descends laterally and posteriorly and turns back at T7. At T12, the aorta is in front of the left pedicle and moves to the right side at the lumbar level. Attention should be paid to the spine drawn in the figure, because the size of the spine changed considerably at the level of the spine

Table 1 Distribution of the left pedicle–aorta angle, the left pedicle–aorta distance, and the pedicular line–aorta distance

	Left pedicle–aorta angle ($^\circ$)	Left pedicle–aorta distance (mm)	The pedicular line–aorta distance (mm)
T4	20.1 ± 22.7	24.5 ± 6.2	20.9 ± 9.7
T5	32.1 ± 20.3	19.4 ± 4.4	14.1 ± 8.5
T6	39.5 ± 17.3	17.8 ± 3.5	10.9 ± 7.2
T7	43.8 ± 13.6	17.6 ± 3.6	10.1 ± 6.3
T8	42.8 ± 11.9	19.0 ± 4.2	11.4 ± 6.5
T9	40.0 ± 12.8	20.6 ± 4.9	13.6 ± 7.2
T10	30.2 ± 15.1	24.0 ± 6.4	19.2 ± 8.6
T11	13.0 ± 19.9	31.5 ± 8.1	29.1 ± 11.0
T12	0.3 ± 15.6	39.3 ± 8.3	36.9 ± 8.4
L1	-12.9 ± 13.3	52.4 ± 7.1	48.3 ± 7.0
L2	-17.7 ± 10.3	56.2 ± 6.2	51.2 ± 5.2
L3	-16.9 ± 6.8	56.7 ± 5.4	52.9 ± 4.2
L4	-19.4 ± 5.0	56.0 ± 5.0	52.4 ± 7.4

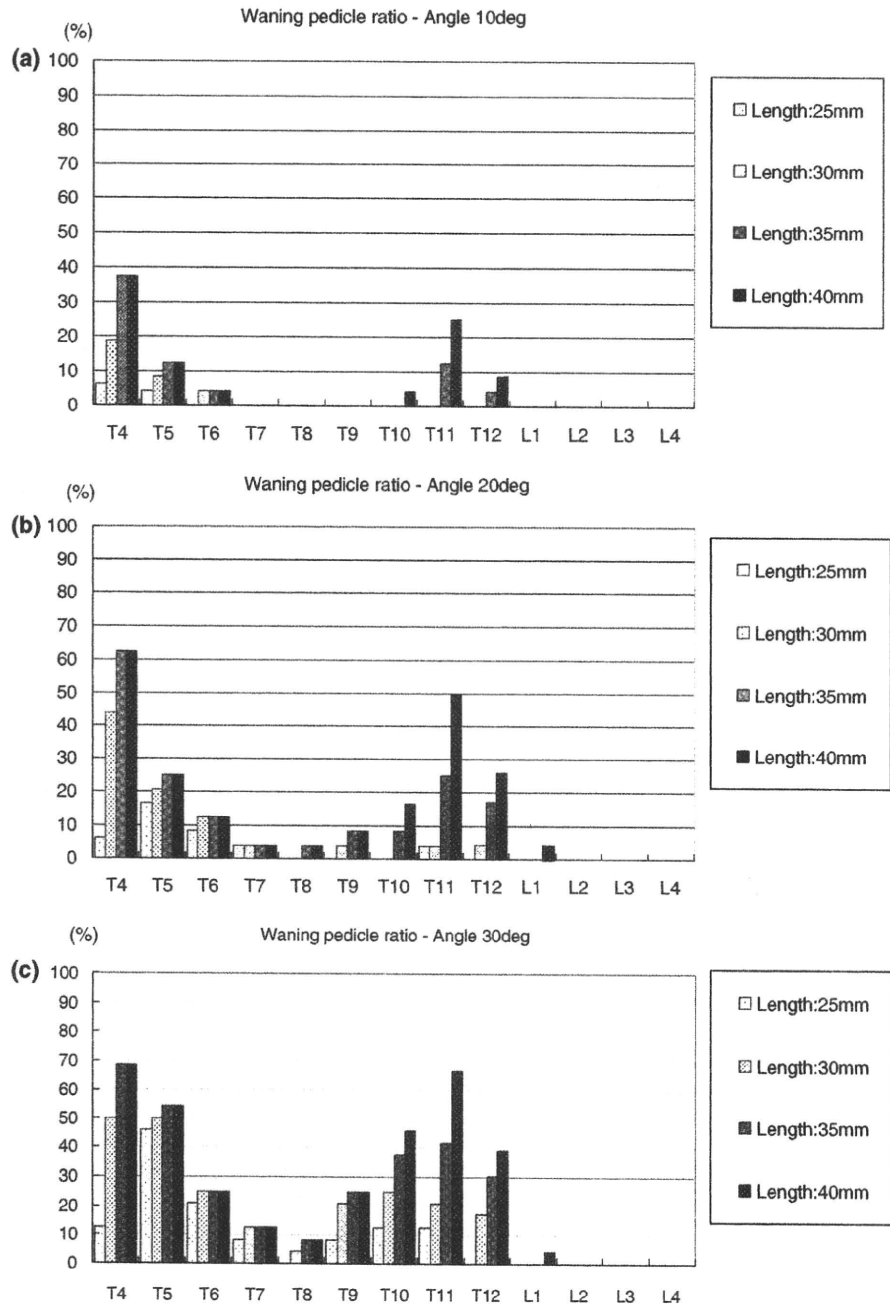
aorta distance ranged from 11 to 52 mm with an average of 23.7 mm and from 36 to 72 mm with an average of 55.2 mm, respectively; the pedicular line–aorta distance ranged from –4 to 59 mm (average 18.3 mm) and from 33 to 75 mm (average 51.0 mm), respectively. From the cephalad to the caudal direction, the aorta was seen at the antero-lateral position of the vertebral body of T4 or T5. The aorta moved to the left side laterally and posteriorly as it descended, changing its course at T7 and moving medially and anteriorly. It located in front of the vertebral

body at the left T12 pedicle. At the lumbar spine, the aorta moved to the right side.

The ratio of warning pedicles increased as the direction error or the screw length increased (Fig. 4). When the direction error was within 30° and the screw length was 40 mm, the ratio was highest at T4 with 69%, followed by T11 (67%), T5 (54%), T10 (46%) and T12 (39%), and this trend was consistent in any scenario.

No parameter of the main thoracic curve correlated with the X-unit of the left pedicle–aorta distance at the apex.

Fig. 4 Distribution of warning pedicle ratios from T4 to L4 in 12 scenario. In any scenario, there was a high percentage of warning pedicles at the thoracic spine except at T7 and T8. **a** The warning pedicle ratio when the direction error is within 10° and the screw length changes from 25 to 40 mm. **b** The warning pedicle ratio when the direction error is within 20° and the screw length changes from 25 to 40 mm. **c** The warning pedicle ratio when the direction error is within 30° and the screw length changes from 25 to 40 mm



Sagittal angle at T5–T12 in the sagittal plane significantly correlated with the *Y*-unit (“anterior–posterior” direction for the spine) of the left pedicle–aorta distance at the apex (-0.44 ; $p = 0.03$).

Discussion

Liljenqvist et al. [5] measured the distance from the aorta to the vertebral body, and reported that the closest distance averaged 6–7 mm between T4 and T9 and <5 mm between T10 and L4. Sucato and Duchene [11] analyzed the position of the aorta in patients with idiopathic scoliosis in magnetic resonance scans and found that the thoracic aorta in idiopathic scoliosis is positioned more posteriorly and laterally compared with straight spines. From their analysis, the aorta begins to be seen as the aortic arch in front of the T4 vertebral body and changes its position posteriorly and laterally as it descends. The aorta turns back anteriorly and medially at the apical region and passes in front of the T12 through the hiatus of the diaphragm. The present study supports their analyses.

Vaccaro et al. [13] analyzed a non-scoliotic thoracic spine and found that the aorta and the esophagus are at greatest risk of injury when a pedicle screw penetrates an anterior cortex of the vertebral body. Liljenqvist et al. [6] analyzed 22 patients with idiopathic scoliosis by computed tomography postoperatively. They found that 3 of 120 pedicle screws penetrated the anterior vertebral cortex and 1 of these three screws was replaced because of its direct proximity to the thoracic aorta.

When a pedicle screw is placed by a free-hand technique [3] or with a fluoroscope, the direction of placement largely depends on several landmarks of the explored surface of the spine: facet joints, transverse processes and laminae. Our new parameters defined by both sides of superior facet are easy to comprehend in posterior surgery. Additionally, we could compare the relative risk of pedicle screw placement between spine levels in various settings by the sensitivity analysis.

The present study elucidated that the aorta usually stays on the anterior or left-lateral side of the vertebral body at T4, T5 and at T10–T12, and a small breach of a pedicle screw outside the vertebral body at these levels may result in indentation of the aorta. Faro et al. [1] studied the influence of indentation of the aorta by a screw in their bovine model and found that the major impingement of vertebral screws on the aorta caused acute and chronic histopathologic and biomechanical changes in the vessel wall. Though sequelae of moderate to mild indentation of the aorta have not yet been known, screws will stay inside the body for over tens of years in

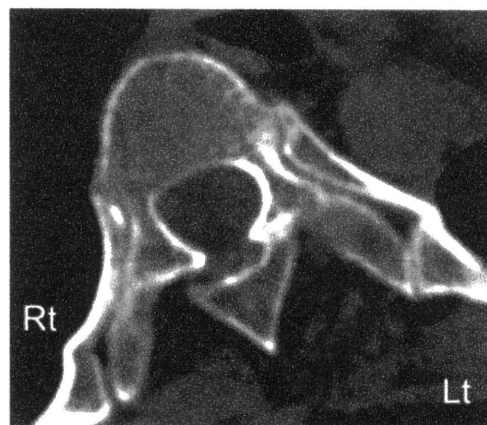


Fig. 5 A case with a typical position of the aorta around the apex level. Though a distance to the left base of the superior facet (an insertion point of a pedicle screw) is closer than other levels, the aorta often resides in the most lateral position from the spine and allows wider maldirection of the pedicle screw

this young population. It is recommended that any screw either in anterior or in posterior surgery be placed away from the aorta.

The present study shows that the aorta at the middle thoracic spine is often located away from the spine and resides in front of a left rib (Fig. 5), which leads to a low percentage of dangerous pedicles at T7 and T8. However, in turn, the spinal cord deviates to the left concave pedicles at the apical area at the right thoracic spine [5]. Moreover, the aorta may not stay in the same position. Huitema et al. [2] examined 50 patients by computed tomography or magnetic resonance scans before surgery, and reported that the aorta moves more anteromedially in a prone position than in a supine position especially at levels T5–T10. Their study indicates that the aorta is fairly mobile at the mid-thoracic level when a subject changes his position. Though the present study showed a relative safety of the aorta at T6–T9, the aorta might reside closer to the spine when a subject is at another position. Admittedly, segmental pedicle screw instrumentation is a most powerful construct for correction and maintenance in spinal deformity. Surgeons, however, must be vigilant about the positions of the aorta and the spinal cord in placement of pedicle screws, especially on the left side, and screw breach may necessitate reoperation for replacement.

In summary, new parameters enable surgeons to intuitively understand the position of the aorta in their preoperative planning or during placement of a pedicle screw. When a left pedicle screw perforates an anterior/lateral wall of the vertebral body, the aorta may be at risk, especially at T4, T5 and T10–T12.

References

1. Faro FD, Farnsworth CL, Shapiro GS et al (2005) Thoracic vertebral screw impingement on the aorta in an in vivo bovine model. *Spine* 30(21):2406–2413
2. Huitema GC, Cornips EM, Castelijns MH et al (2007) The position of the aorta relative to the spine: is it mobile or not? *Spine* 32(12):1259–1264
3. Kim YJ, Lenke LG, Bridwell KH et al (2004) Free-hand pedicle screw placement in the thoracic spine: is it safe? *Spine* 29:333–342
4. Lenke LG, Betz RR, Harms J et al (2001) Adolescent idiopathic scoliosis: a new classification to determine extent of spinal arthrodesis. *J Bone Joint Surg Am* 83(8):1169–1181
5. Liljenqvist UR, Allkemper T, Hackenberg L et al (2002) Analysis of vertebral morphology in idiopathic scoliosis with use of magnetic resonance imaging and multiplanar reconstruction. *J Bone Joint Surg Am* 84(3):359–368
6. Liljenqvist UR, Halm HF, Link TM (1997) Pedicle screw instrumentation of the thoracic spine in idiopathic scoliosis. *Spine* 22(19):2239–2245
7. Maruyama T, Takeshita K, Nakamura K et al (2004) Spatial relations between the vertebral body and the thoracic aorta in adolescent idiopathic scoliosis. *Spine* 29:2067–2069
8. Matsuzaki H, Tokuhashi Y, Wakabayashi K et al (1993) Penetration of a screw into the thoracic aorta in anterior spinal instrumentation. A case report. *Spine* 18:2327–2331
9. Ohnishi T, Neo M, Matsushita M et al (2001) Delayed aortic rupture caused by an implanted anterior spinal device. *J Neurosurg* 95(2 Suppl):253–256
10. Sucato DJ, Kassab F, Dempsey M (2004) Analysis of screw placement relative to the aorta and spinal canal following anterior instrumentation for thoracic idiopathic scoliosis. *Spine* 29(5):554–559
11. Sucato DJ, Duchene C (2003) The position of the aorta relative to the spine: a comparison of patients with and without idiopathic scoliosis. *J Bone Joint Surg Am* 85(8):1461–1469
12. Takeshita K, Maruyama T, Chikuda H et al (2009) Diameter, length, and direction of pedicle screws for scoliotic spine: analysis by multiplanar reconstruction of computed tomography. *Spine* 34:798–803
13. Vaccaro AR, Rizzolo SJ, Balderston RA et al (1995) Placement of pedicle screws in the thoracic spine. Part II. An anatomical and radiographic assessment. *J Bone Joint Surg Am* 77(8):1200–1206