労災疾病研究事業費補助金

# 脊椎インストゥルメンテーション患者に
# アフターケアは本当に必要か？
# 全国労災病院と産業医科大学を含む多施設大規模調査

平成２７年度総括研究報告書

研究代表者　須田　浩太

平成２８年５月

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# 全国労災病院と産業医科大学を含む多施設大規模調査

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研究要旨

脊椎インストゥルメンテーション技術の大幅な進歩に伴い、治療成績は向上したものの、長期にわたる治療が必須な症例も少なくない。

アフターケアの「適応基準」を定め、その際に生じる「医療コスト」を予測する取組を３年計画で包括的に推進することとした。初年度の検討結果は以下である。

1）労災患者における脊椎インストゥルメンテーション症例のデータ集積

全労災病院（全国に３４）と産業医科大学の過去５年を目処に労災患者における脊椎インストゥルメンテーション症例のデータを集積する。

2）労災患者以外の脊椎インストゥルメンテーション症例のデータ集積

労災患者以外の脊椎インストゥルメンテーション症例のデータを集積し、同様の手法で中央集約管理を行う。

3）データの解析

集積したデータを基にアフターケアの該当基準や頻度を解析する。

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A. 研究目的

脊椎インストゥルメンテーション技術の大幅な進歩に伴い、従来は不可能であった大規模な脊椎再建術が平常的に行われるようになった。治療成績は飛躍的に向上し、後遺症が減ったものの、中には長期にわたる経過観察や治療が必須な症例も少なくない。そこで、脊椎インストゥルメンテーションを行った症例に関して、1）症状固定後に如何なる症状が生じえるか？2）如何なる措置がどの程度の期間必要か？3）どの程度の症例数が見込まれるのか？を明らかにすることを目的とする。

限りある財源の有効活用、効率的分配は労災補償行政において重要課題である。脊椎インストゥルメンテーションにおいてアフターケアが必要な症例は極めて少ないと予想するが皆無ではない。どの様な症例が該当するのか、どの程度の頻度で存在するのか把握することが第一義である。「適応基準」を定め、その際に生じる「医療コスト」を予測することが労災補償行政に必須である。

本研究には大きな特色が3つある。

①労災医療における国内最大組織（全労災病院＋産業医科大学）による国内最大規模調査
②労働者健康安全機構が持つ職業調査データベース
③複数の大学・施設・診療科の集学的協力体制

全国には34労災病院があり（総合せき損センター及び吉備高原医療リハビリテーションセンター含む）、総数 13,000 以上のベッド、約

15,000 人のスタッフを有し、最高水準の労災医療を提供している。また、産業医科大学は産業医学の中枢であることは言うまでもない。両施設は我が国における労災医療の中心であり、両施設の共同体がスケールメリットを生かして全国調査を行う意義は非常に大きい。労災医療の調査としては国内最大のスケールとなる。また、労働者健康安全機構が持つ巨大な職業調査データベースから職業別の要因なども解析可能である。更に特殊症例の検討を目的として北海道大学と獨協医科大学の協力を得たこと、社会医学、統計解析のスペシャリストにも協力を得たことも大きな利点である。

1）症状固定後に如何なる症状の動揺を来すおそれがあるのか、また、2）如何なる措置がどの程度の期間必要とされるか、さらには、3）どの程度の症例数が見込まれるのかといった、といった疑問全てに回答すべく研究体制を整備した。

日本国内における脊椎インストゥルメンテーション手術は年間6万件と目されている。そのうち、麻痺のない労災患者を 2%と仮定すると年間症例数は 1200 例。アフターケアにかかる費用は当院平均で年間 30 万円なので、全例を 20 年間アフターケアとすれば 72 億円を要す。しかし、脊椎インストゥルメンテーションを行った症例全てがアフターケアを要するとは考えづらく、ごく限られた特殊な症例が該当するであろうと予測している。症状固定後に後遺症状に動揺をきたす症例、後遺障害に付随する疾病を発症させる症例はどのような条件が揃えば生じえるのか解析し、アフターケアの適応条件を定めることができれば労災補償行政に資するところ大である。

①どのような症例が対象か？

②その程度の期間が必要か？

③その頻度や症例数は？

④追加で必要な医療コストは？

アフターケアを導入する際の適応基準となりえるもので、労災補償行政の施策上、必須のデータになる。また、アフターケアを導入した場合の医療コストを予想することが可能となる。適正なアフターケアの適応基準を定めれば 5%以下の症例しか該当しないであろうと予想され、年間72億円を3億程度まで圧縮する科学的根拠となるであろう。アフターケアが「真に必要な症例」、「本来は不要な症例」を見定めることこそ、限りある労災補償財源の適正分配につながる。

B.研究計画、方法

（1）労災患者における脊椎インストゥルメンテーション症例のデータ集積（2 年間）

北海道中央労災せき損センター、総合せき損センター、吉備高原医療リハビリテーションセンター、産業医科大学をコアとして全労災病院（全国に３４）と産業医科大学の過去５年を目処に労災患者における脊椎インストゥルメンテーション症例のデータを集積する。

データ集積用のクラウドを準備し、中央集約管理を行う。一定の基幹施設にコンピューターと作業員を配置する。また、多施設間の連絡を密にするため iPad など情報端末を全施設の担当者へ配備し、計画の進捗状況を観察し、適宜、適切な補助・助言を行う。

対象施設は以下の 35 施設。

1.北海道中央労災病院、2.北海道中央労災病院せき損センター、3.釧路労災病院、4.青森労災病院、5.東北労災病院、6.秋田労災病院、7.福島労災病院、8.鹿島労災病院、9.千葉労災病院、10.東京労災病院、11.関東労災病院、12.横浜労災病院、13.燕労災病院、14.新潟労災病院、15.富山労災病院、16.浜松労災病院、17.中部労災病院、18.旭労災病院、19.大阪労災病院、20.関西労災病院、21.神戸労災病院、22.和歌山労災病院、23.

山陰労災病院、24.岡山労災病院、25.中国労災病院、26.山口労災病院、27.香川労災病院、28.愛媛労災病院、29.九州労災病院、30.九州労災病院門司メディカルセンター、31.長崎労災病院、32.熊本労災病院、33.吉備高原医療リハビリテーションセンター、34.総合せき損センター、35.産業医科大学

協力医師
油川修一、池田天史、岡崎裕司、奥山幸一郎、加治浩三、川添泰弘、日下部　隆、楠瀬浩一、武田宏史、千葉光穂、信田進吾、馬場秀夫、放生憲博、山縣正庸、渡邉健一、舘田聡、伊藤圭吾、岩崎幹希、岡部　聡、笠原孝一、河本正昭、菊地　廉、傳田博司、花林昭裕、平野典和、三上容司、三好光太、湯川泰紹、安藤宗治、大園健二、大西英生、大和田哲雄、岡野　徹、城戸研二、木戸健司、楠城誉朗、國司善彦、佐々木俊二、笹重善朗、壺内　貢、富永俊克、生熊久敬、前原　孝、原田良昭

実務担当：須田浩太（北海道中央労災せき損センター；以下道央せき損）、三浪明男（道央せき損）、松本聡子（道央せき損）、小松　幹（道央せき損）、牛久智加良（道央せき損）、芝　啓一郎（総合せき損センター；以下総合せき損）、植田尊善（総合せき損）、前田　健（総合せき損）、坂井宏旭（総合せき損）、徳弘昭博（吉備高原医療リハビリテーションセンター；以下吉備リハ）、古澤一成（吉備リハ）、酒井昭典（産業医科大学；以下産業医大）、中村英一郎（産業医大）

（２）労災患者以外の脊椎インストゥルメンテーション症例のデータ集積（3年間）

労災患者以外の脊椎インストゥルメンテーション症例のデータを集積し、同様の手法で中央集約管理を行う。
対象施設は以下の5施設。
1.北海道中央労災せき損センター、2.総合せき損センター、3.産業医科大学、4.北海道大学、5.獨協医科大学
担当：須田浩太（道央せき損）、三浪明男（道央せき損）、松本聡子（道央せき損）、小松　幹（道央せき損）、牛久智加良（道央せき損）、芝　啓一郎（総合せき損）、植田尊善（総合せき損）、前田　健（総合せき損）、坂井宏旭（総合せき損）、酒井昭典（産業医大）、中村英一郎（産業医大）、岩崎倫政（北海道大学；以下北大）、高畑雅彦（北大）、種市　洋（獨協医科大学；以下獨協医大）、稲見　聡（獨協医大）、森平　泰（獨協医大）

（３）データの解析（2年間）

集積したデータを基に下記の7点につき明らかにする。
1.労災患者での脊椎固定頻度
2.手術内容
3.治癒までの期間
4.治癒後の加療要否
5.麻痺患者との比較
6.アフターケアの適応基準
7.医療コスト
担当：伊藤陽一（北大）、須田浩太（道央せき損）、坂井宏旭（総合せき損）、中村英一郎（産業医大）、高畑雅彦（北大）、森平　泰（獨協医大）

C.研究結果

全国34労災病院（約13,000床、約15,000人スタッフ）よりデータを収集している。2009年1月から2014年12月までの5年間を対象とした調査を行った。当該機関に全国の労災病院にて入院加療を行った労災保険患者は18371名であった。うち労災保険適応の手術は16093件あり、このうち、脊椎手術を受けた患者は514名、脊椎手術総件数は594件であった。その中で脊椎インストゥルメンテーション手術は348件であることが判明した。現在、これらの患者を対

象に疾患名、術式、固定部位、固定椎間数、矯正の有無、アウターケアの有無、アフターケアの内容、医療費などの調査を開始したところである。各病院の症例数内訳は、北海道中央労災病院：1、北海道中央労災病院せき損センター：65、釧路労災病院：8、青森労災病院：2、東北労災病院：3、秋田労災病院：4、福島労災病院：2、鹿島労災病院：2、千葉労災病院：14、東京労災病院：4、関東労災病院：17、横浜労災病院：10、新潟労災病院：3、富山労災病院：6、浜松労災病院：3、中部労災病院：26、旭労災病院：1、大阪労災病院：3、関西労災病院：8、神戸労災病院：3、和歌山労災病院：2、山陰労災病院：8、岡山労災病院：4、中国労災病院：6、山口労災病院：9、香川労災病院：14、愛媛労災病院：2、九州労災病院：6、長崎労災病院：26、熊本労災病院：12、総合せき損センター：29であった。

関連する他研究の調査：脊椎インストゥルメンテーションに起因する隣接椎間障害についての検討

頸椎インストゥルメンテーション

外傷

小島孝太らは中下位頸椎損傷に対して PS と LMS を施行した 4 例(男性 3 例、女性 1 例、手術時年齢 25-52 歳)、CF 型 3 例(C4/5 脱臼 1 例、C4/5 脱臼骨折 1 例、C5/6 脱臼骨折 1 例)、VC 型 1 例(C6 圧迫骨折)を調査し 1 例で隣接椎間障害を認めた。すなわち外傷では隣接椎間障害は少なかった。

慢性関節リウマチ

一方で、林 協司らは慢性関節リウマチ（以下 RA）性中下位頸椎病変 19 例(男 2 例、女 17 例、平均 62 歳).Steinbrocker 分類では stage III が 2 例、IV が 17 例で、術式は椎弓形成術 8 例(A 群)、椎弓切除+後方固定術 8 例(B 群)、前方除圧固定術 3 例(C 群)を術後 14〜55 ヵ月まで調査した。A 群では Ranawat の疼痛スコアで 1 段階改善が 5 例、2 段階改善が 3 例であった。脊髄症スコアでも術直後は全例 1 段階改善したが、1 例で彎曲異常が増強し、除圧術を追加した。C 群では疼痛スコアが 1 段階改善 2 例、2 段階改善 1 例であったが、脊髄症スコアは全例改善を認めなかった。X 線所見は、A 群では 1 例にのみ不安定性の増強が認められ、C 群も全例骨癒合は得られたが、移植骨の圧潰を認め、2 例に隣接椎間の新たなすべり、不安定性が出現していた。納田真也らは同様に RA 環軸椎亜脱臼に対する環軸椎固定術の手術成績を調査し、軸椎下亜脱臼(SAS)の発生と進行が手術成績に及ぼす影響を調査した。術後 18 ヵ月以上経過した 10 症例とした。神経症状が悪化した 4 例中 3 例では、SAS の出現あるいは進行時期に一致して神経症状が悪化しており、新たな SAS の発生または SAS の進行が術後の神経症状に影響していた。最終調査時に SAS を認めた 7 例中 5 例はムチランス型であり、SAS 発生と進行の要因としてムチランス型の自然経過が最も大きいと考えられた。7 例で骨癒合が後頭骨や第 3 頸椎まで多椎間に及んでいたことから、上位頸椎の多椎間に及ぶ骨癒合によって下位頸椎の負担が増大したために生ずる隣接椎間障害も、SAS の要因となり得ると考えられムチランス型に対しては、全頸椎固定を考慮する必要を示唆した。

透析

透析に関しては土屋邦喜らが頸椎後方固定術を施行した血液透析(HD)患者 24 例を検討している。椎弓根スクリュー(PS)が最も多く 13 例に使用されていた。脊髄症の平均改善率は 30.6%であった。骨癒合は 21 例で獲得され、骨癒合率は 87%であった。インプラントの折損を 2 例に認めた。隣接椎間障害が 5 例に認められた。スク

リューを用いた後方固定術は長期 HD 患者に対して適した方法と考えられるが隣接椎間障害に関しては十分な注意が必要であると結論した。宮崎幸政らは透析患者で上位頸椎病変を含む手術を施行した 7 例(男性 4 例、女性 3 例)を検討した。平均年齢は 65.1 歳、平均透析歴 23.4 年、病態は、歯突起偽腫瘍 2 例、環軸関節亜脱臼 2 例、DSA に伴う固定術後の隣接椎間障害 2 例、C2 椎体内骨嚢胞を伴う脊髄症 1 例であった。Magerl スクリューを用いた 1 例で骨癒合不全、隣接椎間障害の 2 例でインプラントの破損を認めた。岸田俊一らは後頭胸椎固定術を 4 例(男 2 例・女 2 例)に施行した。平均年齢は 64.5 歳、平均透析期間は 23 年であった。病態は歯突起偽腫瘍と下位頸椎 DSA の合併、環軸関節亜脱臼と下位頸椎 DSA の合併、後頭胸椎固定術後の implant failure、固定術後の隣接椎間障害であり、3 例に多数回手術を行っていた。1 例にロッド折損による再手術を施行したが、最終的には全例骨癒合が得られ、おおむね ADL は保たれた。術後の平均経過観察期間は 5.9 年であり、期間中に 3 例が死亡したと報告した。平野 徹らは透析性脊椎症の頸椎病変に対する手術例 29 例(男 20 例、女 9 例、手術時平均年齢 59 歳)の術後経過を調査した。術式は前方固定術 7 例、後方除圧固定術 8 例、椎弓形成術 14 例であり、周術期合併症として、大量出血、血腫、instrumentation 脱転、早期隣接椎間障害、せん妄などを認めた。日本整形外科学会頸髄症判定基準は術前平均 6.4 点から術後 9.9 点と改善した(改善率 47%)が、最終時 8.9 点であった。3 例に術後平均 4 年で頸椎隣接椎間障害を生じ、9 例に術後平均 3 年で腰椎病変の出現/悪化を生じた。重度脊髄障害の透析患者を作らないためには、患者啓発および実地透析医に施行可能なスクリーニング導入による早期診断と、脊椎手術を受けた透析患者に対する専門医の継続的観察が重要である。以上より、透析患者においては周術期管理が厳重である必要があるとともに、隣接椎間障害の発生が多い。これは脊椎インストゥルメンテーションによるものと言うより、透析性変化と判断できる。

脳性麻痺

三原久範らはアテトーゼ型脳性麻痺に伴う頸椎症性脊髄症に対する手術 89 例中 27 例の再手術例を経験した。術後 6 ヵ月以内の早期再手術例は 6 例あり、固定術後の内固定材の破綻が 4 例(80 例中)、椎弓形成術後の症状悪化が 2 例(4 例中)で、いずれもわれわれのアテトーゼ強度分類で Grade 4(徒手矯正不能な強度)以上の症例であった。一方、術後 6 ヵ月以上経過後の再手術は 21 例あり、抜釘を除くと隣接椎間障害(5 例)と頭蓋頸椎移行部不安定症(2 例)が原因であった。下川 宣幸らは手術治療を行ったアテトーゼ頸髄症患者 7 例(男性 4 例、女性 3 例、平均 59.7 歳)を対象に、術前の画像評価、三原らのアテトーゼ強度分類で「アテトーゼ強度」を評価して術式を選択した。観察期間は平均 23.7 ヵ月であった。アテトーゼ強度が軽度(grade 1、2)で 20 度以上の局所後彎がなければ椎弓形成術を行い、20 度以上の局所後彎合併例に後彎矯正固定術を併用し、椎間孔狭窄に後方椎間孔開放術を行った。アテトーゼ強度が中等度(grade 3)には A 型ボツリヌス毒素製剤を過緊張筋に術前投与し、後方徐圧固定術を行った。アテトーゼ強度が重度(grade 4、5)には過緊張の選択的筋解離術を併用して後方徐圧固定術を行った。平均 JOA スコアは術前 7.7 点→最終時 10.6 点、平均改善率は 31.2%であった。術後に固定に伴う嚥下障害、隣接椎間障害、頭蓋頸椎移行部の不安定性出現は認めず、後方徐圧例で術後に後彎変形が出現して神経症状を呈する症例も認めなかった。上記 2 論文からは適切な治療選択により隣接椎間障害や再手術は軽減できるものの、限界があることが示され、原因は脊椎インストゥルメンテ

ーションではなくアテトーゼ型脳性麻痺といえる。

腰椎インストゥルメンテーション

英文、和文の関連論文を用いて Meta analysis を腰椎インストゥルメンテーションに関して行った。まとめを別添資料とした。

広範囲インストゥルメンテーション

清水敬親らは骨粗鬆性脊柱矢状面配列異常に対して、上中位胸椎(Th3～7)～骨盤(仙骨又は腸骨)に至る広範囲固定を施行した 22 症例(手術時 54～80 歳・平均 70 歳、術後経過観察期間 1.5～8 年・3.1 年)の臨床像・治療結果について検討し固定頭側端は T3:8 例・T4:3 例・T5:9 例・T6:1 例・T7:1 例、固定尾側端は S1:3 例・S2:14 例・腸骨:5 例であった。術後固定頭側の隣接椎間障害は全例で認めず、フックやスクリューの脱転やワイヤーのカットアウトは認めなかったが、ロッド折損を 2 例で認め、1 例は偽関節であったと報告している。隣接椎間障害は予想以上に少ないと感がえる。

D.考察

本調査では労災保険が適応されて脊椎インストゥルメンテーション手術を受けた患者の大半が脊椎損傷であり、大部分が脊髄損傷や馬尾損傷などの神経損傷を伴っていた。神経損傷によって引き起こされる麻痺は後遺症として残存しやすい。上肢、下肢、体幹に留まらず排尿障害や排便障害も引き起こすため永続的な治療を要す。すなわち、これがアフターケアの根拠となっている。麻痺がない患者で脊椎インストゥルメンテーション手術を受けた場合のアフターケアの要否は不明のままであるが、脊椎インストゥルメンテーション手術に関連する他研究から後遺症、隣接椎間障害など長期にわたり治療を要する病態は乏しいと推察できる。

E.結論

労災患者以外のコントロールとして脊椎インストゥルメンテーション手術を受けた労災保険以外の患者データを 1.北海道中央労災せき損センター、2.総合せき損センター、3.産業医科大学、4.北海道大学、5.獨協医科大学にて分担して調査している。2 年間はデータの集積業務が主となるため解析作業は行っていないが、次年度から以下の解析を行う予定である。

1.労災患者での脊椎固定頻度
2.手術内容
3.治癒までの期間
4.治癒後の加療要否
5.麻痺患者との比較
6.アフターケアの適応基準
7.医療コスト

F.健康危険情報

該当なし

G.研究発表

1.論文発表

データ集積および解析後に予定

2.学会発表

データ集積および解析後に予定

H.知的財産権の出願・登録状況（予定を含む）

1.特許取得

なし

2.実用新案登録

なし

3.その他

| Summary | Article Number |
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| A total of 15 studies consisting of 1474 patients were included.<br>•ASD were lower in motion-preservation procedures group than in the fusion group (P = 0.001; P = 0.0004; P < 0.0001).<br>•Moreover, shorter length of hospital stay was found in motion-preservation procedures group (P < 0.0001).<br>• No difference was found in terms of operation time (P = 0.57), blood loss (P = 0.27), VAS (P = 0.76) and ODI improvement (P = 0.71) between the two groups. | #1 |
| A total of 13 studies with 1,270 patients were included.<br>•ASD were lower in motion-preservation procedures group than in the fusion group (P = 0.001; P = 0.0004; P < 0.0001).<br>•Moreover, shorter length of hospital stay was found in motion-preservation procedures group (P < 0.0001).<br>No difference was found in terms of operation time (P = 0.57), blood loss (P = 0.27), VAS (P = 0.76) and ODI improvement (P = 0.71) between the two groups. | #2 |
| A total of 31 articles with 4206 patients were included<br>•ASDeg (degeneration) was 5.9% , and ASDis (disease) was 1.8%per year.<br>•The incidence of ASDeg is higher with more motion segments.<br>•Sex, age, segmental sagittal alignment, fusion methods, and instrumentation were not associated with an increased risk of ASD.<br>•Radiographic ASDeg did not show strong correlation with clinical outcomes. | #3 |
| CONSENSUS STATEMENTS:1. The risk of developing CASP after lumbar fusion occurs at a mean annual incidence of 0.6% to 3.9%. Strength of Statement: Strong.<br>2. Patients older than 60 years or who have pre-existing facet/disc degeneration may have an increased risk of developing CASP. Strength of Statement: Strong.<br>3. The risk of developing CASP may be greater after multilevel fusions and fusions adjacent to but not including the L5–S1 level, and may increase when performing a laminectomy adjacent to a fusion. Strength of Statement: Strong. | #4 |
| In patients who underwent fusion in the lumbar spine for degenerative reasons, the radiographical adjacent segment pathology (RASP) rate averaged 12.4% during an average of 5.6-year follow-up. There is insufficient evidence in the literature to determine whether the indication/reason for fusion affects the risk of RASP in the lumbar spine | #5 |
| The incidence of adjacent segment disease in the lumbar spine to range from 2% to 14%. Damage to the posterior ligamentous complex and sagittal imbalances are important risk factors for both degeneration and disease. | #6 |

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| CONSENSUS STATEMENTS:<br>1. The risk of clinical ASP is likely greater after fusion than the other motion-sparing devices with fusion but the risk is still quite rare. The increased risk compared to TDR could be ***as small as less than 1% or as great as 10%***. Strength of Statement: Weak.<br>2. There is insufficient evidence to make a definitive statement regarding fusion versus other motion-sparing devices with respect to the risk of ASP. | #7 |
| No studies comparing operative with nonoperative management or comparing operative with operative management of CASP were identified in the literature. | #8 |
| 314 / 926 patients in the arthrodesis group (34%) and 31 / 313 patients in the total disc replacement group (9%) developed ASDeg. 173/1216 patients in the arthrodesis group (14%) and 7/595 patients in the arthroplasty group (1%) developed ASDis<br><br>higher odds of ASDeg :<br>・older patients ($P < 0.001$)<br>・arthodesis ($P = 0.0008$)<br>・and longer follow-up ($P = 0.0025$).<br><br>higher odds of ASDis:<br>・with fusion ($P < 0.0001$)<br>・higher percentages of male patients ($P = 0.0019$)<br>・shorter follow-up ($P < 0.05$). | #9 |
| Adjacent segment disease and degeneration remain a multifactorial problem with several techniques being developed recently to minimize them | #10 |
| [redacted] | |

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| Plain standing lateral radiographs were obtained at LTFU (mean, 13 ± 4 yr postrandomization) in 229 of 464 (49%) patients randomized to surgery and 140 of 303 (46%), to nonoperative care.<br><br>Both treatment groups showed significantly lower values for disc space height of the adjacent segment than norm values.<br><br>There was a significant difference between treatment groups for the disc space height of the cranial adjacent segment<br><br>The treatment effect of fusion on adjacent segment disc space height was -0.44 SDs; there was no group difference for posteroanterior displacement<br><br>Adjacent level disc space height and posteroanterior displacement were not correlated with Oswestry Disability Index or pain scores | #11 |
| 148 patients with severe chronic low back pain were randomly selected for ALIF+PLF or for PLF alone. 95 patients participated.<br><br>ASD was similar between randomization groups<br><br>Patients without disc height reduction over time were significantly younger than patients with disc height reduction. | #12 |
| 74 consecutive patients who underwent instrumented lumbar/lumbosacral fusion<br>Radiographic ASD occurred in 20.6% (14/68) of patients<br>Preoperative disc degeneration at an adjacent segment was a risk factor for ASD<br>Age, gender, preoperative diagnosis, length of fusion, instrumentation configuration, sagittal alignment and lumbar or lumbosacral fusion were not significant risk factors for the development of ASD | #13 |
| A total of 137 patients<br>9% required a follow-up operation for ASD<br><br>Predictors of developing ASD included<br>・antidepressant use (OR=5.4),<br>・diagnosis of degenerative scoliosis (OR=34.2), fusion of L4-S1 (OR=56.5),<br>・having no decompressions adjacent to the fusion,<br>・low sacral slope (OR=0.9). | #14 |

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| 511 patients<br><br>Patients with fusions excluding the sacrum (floating fusions) were more likely to develop ASD than those with fusion constructs ending at S-1 distally (p = 0.030) but were less likely to develop postoperative radiculopathy (p = 0.030)<br><br>In the floating fusion cohort, 31 (12.11%) of 256 patients had cephalad ASD, whereas 39 (15.29%) of 255 patients in the lumbosacral cohort had cephalad ASD development; this was not statistically different (p = 0.295)<br>(; Suggesting that caudad ASD development in the floating fusion cohort is due to the added risk of an unfused L5-S1 vertebral level. ) | #15 |
| Age was a risk factor for radiologic degeneration<br><br>109 patients who underwent PLIF for degenerative instability at L4/5<br>Group A : without ASD, Group B: with ASD.<br>11 patients (22%) classified into Group A<br><br>No significant difference : the lordosis angle at L1 and S1, the laminar inclination at L3, the pre-existing L3-L4 disk degeneration, the lordosis angle of L4-L5, the lumbosacral joint angle and preoperative BMD (P > 0.05)<br><br>Significant differences : age | #16 |
| 81 patients<br><br>The incidence of ASD development after lumbar surgery was 11.1% (9 of 81) in this study<br><br>Risk factor for ASD development: Age > 50 years at primary surgery<br>Not : number of fusion levels, initial diagnosis and type of fusion | #17 |
| 154 patients,<br>103 (66.8%) showed ASD<br>92 (59.7%): the cranial ASD<br>61 (39.6%): the caudal ASD<br>10 (6.4%): required second operation<br><br>Age, body mass index (BMI), and preexisting stenosis at the cranial adjacent segment: risk factors for clinical ASD | #18 |
| 43 patients (67 segments) treated with the Graf ligament<br>angular instability : 19 of the 67 segments (28%)<br>translational instability: five (7%).<br>The disc height decreas : 93% → 82%<br>adjacent segmental instability at the upper segment: 18 (42%)<br>The adjacent segment instability at the lower segment: 13 (30%) | #19 |
| 275 patients who underwent instrumented PLF<br>59 (21.5%) patients required reoperation for ASD<br>the predicted ASD-free survival was 79.7% (95% CI, 72.3 - 85.3) at 5 years and 53.4% (95% CI, 40.0 - 65.0) at 10 years.<br><br>the only significant factor associated with ASD : increasing age<br>sex, preoperative diagnosis, and the numbers of segments fused were not significantly associated with ASD. | #20 |

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| 102 patients<br>L5/S1 showed a significant ($P<0.05$) lower risk for ASD than patients with fusions L4/5 (20% vs. 46%).<br>Compared with L4/5 fusions, bisegmental L4-S1 fusions showed a similar trend ($P=0.06$) with a lower risk for ASD (24%).<br><br>Patients suffering from ASD showed significant ($P<0.05$) reduced sacral inclination and lumbar lordosis angles | #21 |
| 1250 patients who underwent posterior lumbar fusion and pedicular fixation<br>13 patients with symptomatic ASD who underwent secondary surgery<br>22 patients without symptomatic ASD<br><br>Radiographic risk factors for the development of a symptomatic ASD were increased sagittal balance, loss of lordosis, and adjacent disc space collapse<br><br>increased BMI, preoperative ADD and disc bulge on MRI have a statistically significant increased risk of developing symptomatic ASD. | #22 |
| 98 patients who had undergone surgical correction and fusion with instrumentation for degenerative lumbar scoliosis<br><br>ASD was present in 44 (44.9%) patients at an average period of 48.0 months (range 6-98 months)<br><br>Risk Factors for ASD<br>・preoperative existence of disc degeneration (as revealed by MRI)<br>・ age at operation ($P = 0.0001, 0.0364$).<br><br>There were no statistically significant differences between radiological adjacent segment degeneration and clinical results (VAS, $P = 0.446$; ODI, $P = 0.531$). | #23 |
| 401 patients with spondylolisthesis at the L4-5 or L4-5-S1 level<br>CASD :clinically symptomatic adjacent segment degeneration<br><br>Fusion extension surgery was performed on 33 patients due to CASD at the L3-4<br><br>3-, 5-, and 10-year disease-free survival rates of 99.20, 96.71, and 76.93 %<br><br>Risk factors for CASD : age, low overall lordosis, low segmental lordosis, progression of facet degeneration, total laminectomy-treated isthmic spondylolisthesis, and PLF-alone rather than IBF alone or IBF + PLF | #24 |

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| 54 patients who underwent L4/5 PLIF for L4 degenerative spondylolisthesis<br><br>31 patients (57.4%) : ASD<br>The length of follow-up and simultaneous decompression surgery at L3/4 were statistically significant factors for ASD<br><br>7 patients (13.0%) had symptomatic ASD: 6 in the decompression group (16.2%) and 1 in the PLIF-only group (5.9%).<br>Simultaneous decompression surgery did not reduce the incidence of symptomatic ASD.<br><br>Local lordosis at the fused segment and the sagittal angle of the facet joint at L3/4 were statistically significant predictors of symptomatic ASD | #25 |
| 52 L4/5-PLIF patient<br><br>Progressive disc degeneration at L3/4 was found significantly more frequently in patients with aggravation of facet degeneration<br><br>The severities of preoperative facet degeneration, facet sagittalization and tropism were not associated with progressive disc degeneration or spinal stenosis.<br><br>Patients who underwent L4 total laminectomy had significantly more frequent R-ASD compared to those who received bilateral fenestration at L4/5 (P<0.01). | #26 |
| 85 patients with L-4 spondylolisthesis treated by L4-5 PLIF<br><br>The L4-5 disc space distraction by cage insertion ...<br>3.1 mm : without ASD<br>4.4 mm : with radiographic ASD<br>6.2 mm : with symptomatic ASD<br><br>Multivariate analysis showed that distraction was the most significant risk factor. | #27 |
| 1:1 (50 vs 50) pair analysis matched by age, sex, fusion level, and follow-up period.<br><br>higher BMI , preoperative facet degeneration, disc degeneration, and a smaller relative paraspinal muscle cross-sectional area (CSA) were significant factors for predicting the development of ASD. | #28 |

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| 69 patients who underwent L4/5 PLIF with no definitive degenerated disc in adjacent segments on preoperative MRI and plain radiographs<br><br>group I was isthmic spondylolisthesis patients and group II was degenerative spondylolisthesis<br><br>postoperative L4-L5 segmental lordotic angle at about 20° or more is important for prevention of clinical ASD | #29 |
| 490 patients who had undergone lumbar spinal fusion<br>PLF :103 , PLIF: 387<br><br>23 patients (4.7%) had undergone additional surgery for ASD<br>disease-free survival rate of adjacent segments in 94.2% of patients at 5 years and 89.6% at 10 years after operation<br><br>PLIF was associated with 3.4 times higher incidence of ASD than PLF.<br>Older than 60 years were 2.5 times than younger than 60 years.<br><br>no significant differences : sex, preoperative diagnosis, number of fused segments, and concomitant laminectomy to adjacent segment. | #30 |
| 3,799 patients who underwent posterior lumbar fusion<br>A total of 28( 0.74%) patients with symptomatic ASD surgery were identified.<br>Matched 56 for control group<br><br>Strong risk factors :<br>preoperative distance from L1 to S1 sagittal plumb line, preoperative lumbar lordosis , and preoperative adjacent disc height.<br><br>BMI, preoperative adjacent disc degeneration (ADD) on MRI , and disc bulge in preoperative CT maintained their significance in predicting likelihood of symptomatic adjacent segment degeneration surgery. | #31 |
| 73 patients , long-term (15 years) , PLIF<br><br>At 10- and 15-year follow-up, ODI scores return to preoperative scores in patients without revision surgery.<br><br>At 15-year follow-up, nine patients were lost and a total of 24 (37.5 %) required a new surgical treatment because of ASD.<br><br>The occurrence of revision surgery because of symptomatic ASD was highly dependent of the age of patients at the first surgery and the number of fused levels. | #32 |

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| 120 PLIF patients<br>first group included 60 patients: 3椎間以上<br>second group included 60 patients :2椎間以下<br><br>Long: sagittal imbalance have a statistically significant increased risk of ASD<br><br>Short: preoperative degenerative changes are more important factors | #33 |
| 48 patients<br><br>ASD was found in 30 (62.5%) patients<br><br>Risk:age, difference in the degree of preoperative from postoperative lumbar lordosis, and the proportion of patients who underwent anterior lumbar interbody fusion<br><br>radiographic evidence of ASD does not necessarily correlate with a poor outcome | #34 |
| The purpose of the current study was to investigate the degeneration in decompressed adjacent segments after PLIF.<br><br>In the 19 segments with ASD, ASD occurred in 16 of 25 (64.0%) segments at decompressed sites compared with 3 of 15 (20.0%) non-decompressed sites. | #35 |
| 101 patients<br><br>degenerative changes<br>・disc height: 12 (cranial), 3 (Caudal)<br>・vertebral slip: 36cranial), 6 (Caudal)<br>・intervertebral angle: 17 (cranial), 11 (Caudal)<br><br>・Increased disc degeneration : 62 (cranial), 25 (Caudal)<br>・stenosis: 68 (cranial), 12 (Caudal)<br><br>Ten patients (9.9%) required reoperation<br><br>High pelvic incidence was a risk factor for developing early-onset radiographical ASD | #36 |
| 20 patients who underwent additional surgery for ASD after L4/5 posterior lumbar interbody fusion (PLIF) for L4 degenerative spondylolisthesis.<br><br>Lamina inclination angle was significantly higher in the ASD group than in the control group.<br><br>facet tropism was more significant in the early group than in the late and control groups. | #37 |

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| 190 patients undergoing lumbar fusion surgery for degeneration<br><br>ASD was identified in 13 of the 190 patients(6.8%)<br><br>BMI was a risk factor for adjacent segment disease after lumbar fusion for degenerative spine diseases (odds ratio, 1.68; 95% confidence interval, 1.27-2.21; P < .001) | #38 |
| 45 patients (ASDis) were identified that underwent revision surgery for adjacent segment disease after on average 49 months (7-125)<br><br>Patients with such PI - LL >10° mismatch exhibit a 10-times higher risk for undergoing revision surgery | #39 |
| French Spine Surgery Society clinical data that included 3338 patients<br><br>ASD requiring revision surgery was predicted in 5.6%<br><br>Initial spinal disease affected the risk of ASD requiring surgery (p = 0.0003). The highest risk was observed for degenerative spondylolisthesis. | #40 |
| 55 patients who were followed up for more than 5 years after lumbar fusion<br><br>There were 21 patients with adjacent segment degeneration.<br><br>there was little relationship between ASD and gender, age, residence, fusion method, number of fusion segments, degree of preoperative adjacent disc degeneration in MRI, or preoperative and postoperative LL<br><br>Good:fusion segment lordotic angle per level (FSLA per level) >15° | #41 |
| 217 patients who underwent lumbar fusion<br><br>The patients were divided into 3 groups: fusion was carried out on 1 , 2, and more than 3 segments<br><br>The impact of ASD on clinical outcome after fusion showed a significant correlation with number of fusion segments. | #42 |
| open vs minimally invasive (MI) TLIF<br><br>Risk of ASD did not differ significantly by surgical approach | #43 |

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| Spondylolytic Spondylolisthesis vs Degenerative Spondylolisthesis vs Spinal Stenosis<br><br>sixty-three patients<br><br>no significant difference in the superior adjacent segment degeneration and instability | #44 |
| Of 161 patients, 22 patients (13.7 %) had additional surgeries at cranial segments<br><br>Pre-existing canal stenosis ≥47 % at the adjacent segment on myelography, greater facet tropism, and high BMI were significant risk factors for C-ASP<br><br>spinal canal stenosis on myelography, but not on MRI, was found to be a significant risk factor for ASD | #45 |
| [redacted] | |
| 腰椎後側方固定術(PLF)を施行した腰椎変性疾患28例<br><br>再手術の原因は固定上位での狭窄3例、固定上位での椎間板ヘルニア1例、instrumentation下端でのPLF偽関節と再狭窄1例<br><br>画像上の隣接椎間障害は17例に認め、前方すべり2例、後方すべり8例、側彎1例、終板硬化像7例 | #46 |
| L4腰椎変性すべり症,L4/5PLIF,2年以上,85名<br><br>L4/5固定椎間高は,<br>ASD(-)群(58例)の3.1/1.3mm<br>,画像的ASD(+)群(14例)は4.4/1.5mm<br>臨床的ASD(+)群(13例)は6.2/4.3mm<br><br>過度の持ち上げがASDの危険因子 | #47 |
| 過度の持ち上げがASDの危険因子 | #48 |
| Graf制動術の方が後側方固定術に比べ椎間板変性の進行の頻度は低<br><br>隣接椎間障害に対する追加手術率も,Graf制動術で5.6%,後側方固定術で18.5%と,Graf制動術で低率 | #49 |
| 腰部脊柱管狭窄症23例<br><br>ASDの発生は12例(52%)<br>発生率は1椎間固定よりも2椎間固定で高い<br><br>ASD群と非ASD群の間で椎間高、椎間角に有意差はみられなかった | #50 |
| 1椎間PLIF＜ 1椎間PLIF＋1椎間PLF ＜ 2椎間PLIFで隣接椎間障害(ASD)発生率高 | #51 |

| | |
|---|---|
| L4/5椎間後方除圧固定術を行った腰椎変形すべり症21例<br><br>隣接椎間障害をH-graft群6例(66.7%)、PLF群6例(75%)、PLIF群3例(75%)に認めた | #52 |
| すべり矯正を行った矯正群15例(平均年齢65.2歳、平均観察期間46ヵ月)<br>vs in situ固定を行った非矯正群17例<br><br>固定椎間の前彎角および最終調査時の隣接椎間障害、隣接椎間可動域、隣接椎間高は両群、有意差なし | #53 |
| TLIF36例 vs SSCS)を用いたdinamic stabilization 41例<br><br>椎間板の変性進度は、全椎間でSSCS群に比しTLIF群の変性が有意に強 | #54 |
| 腰椎変性疾患に対して後方固定術を行った65歳以上の患者42例<br>ASD発生を認めた症例は11例(26.2%)<br><br>固定椎間数と矯正前彎角が危険因子<br>患者背景、種々の術前画像所見、隣接椎間への除圧単独の追加はASD発生の危険因子ではない | #55 |
| PLIFを施行した167症例のうち、術後10年以上経過観察可能であった101例<br><br>10例(9.9%)が経過観察中に隣接椎間障害に対し再手術<br><br>危険因子はPI値の大きな症例 | #56 |
| L4/5単椎間固定術を施行した91例<br>術後隣接椎間変化を認めた例は33例(36%) | #57 |
| 不安定性腰椎病変に腰椎固定術を行った透析患者47例<br>隣接椎間障害は47例中5例(10.6%) | #58 |
| PLIF47例<br>ASDは14例(29.8%)に認め、うち3例は有症状で加療<br><br>1椎間25.9%、2椎間35%であった。<br>JOA改善率は有意差なし | #59 |
| 後側方固定術(PLF)施行後,10年以上経過観察可能であった31例<br>X線上術後10年以上では100%に隣接椎間になんらかの変化を認め,<br>そのうち約40%に異常可動性或いはMRI上の狭窄を認め,経年的に増加 | #60 |
| 隣接椎間障害は67例中27例<br><br>腰仙椎前彎角が10度以上低下する症例にも腰痛が出現し易い | #61 |
| L4変性すべり症に対しL4/5単椎間の除圧固定術を施行した20例<br><br>上位隣接椎間障害ありの症例は隣接椎間障害なしの症例に比較して、調査時の固定椎間角が有意に小 | #62 |

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|---|---|
| PLFを行った症例のうち、4年以上経過観察可能であった32症例<br><br>32例中15例(46.8%)で隣接椎間のすべりや狭小化<br>隣接椎間障害の有無と最終経過観察時のJOAスコアー改善率の間に有意な相関関係なし<br>固定椎間の%slip・lumbosacral joint angle・腰椎前彎角・disc angle、いずれの項目も有意な差なし | #63 |
| 腰椎後方固定術を施行し、2年以上経過観察可能であった73例<br><br>症候性ASD群は11例<br>非症候性ASD群は15例<br>非ASD群が47例<br><br>ASD群全体では近位端発生が24例、遠位端発生は2例<br><br>症候性ASD群で腰椎前彎矯正角が有意に少なかった<br>年齢、性別、経過観察期間、病型分類，他X線学的因子すべての項目で有意差なし | #64 |

論文(英文)

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| | | |
|---|---|---|
| Nakashima H, Yukawa T, Suda K, Yamagata M, Ueta T, Kato F | Abnormal Findings on Magnetic Resonance Images of the Cervical Spines in 1,211 Asymptmatic Subjects. | Spine(Phila Pa 1976).2015 Mar 15;40(6):392-398 |
| Matsumoto S, Suda K, Iimoto S, Yasui K, Komatsu M, Ushiku C, Takahata M, Kobayashi Y, Tojyo Y, Fujita K, Minami A | Prospective study of deep vein thrombosis in patients with spinal cord injury not receiving anticoagulant therapy. | Spinal Cord(2015)53, 306-309 |
| Kato M, Taneichi H, Suda K. | Advantage of Pedicle Screw Placement into the Sacral Promontory (Tricortical Purchase) on Lumbosacral Fixation. | J Spinal Disord Tech.28,6 July 2015 |
| Nakashima H, Yukawa T, Suda K, Yamagata M, Ueta T, Kato F | Cervical Disc Protrusion Correlates With the Severity of Cervical Disc Degeneration | SPINEe(Phila Pa 1976).2015 Mar 13;40(13):E774-E779 |

(独)労働者健康安全機構　総合せき損センター

| | | |
|---|---|---|
| Jun Tanaka, MD, Itaru Yugue, MD, PhD,y Keiichiro Shiba, MD, PhD,y Akira Maeyama, MD, PhD, and Masatoshi Naito, MD, PhD | A Study of Risk Factors for Tracheostomy in Patients With a Cervical Spinal Cord Injury | SPINE Volume 41, Number 9, pp 764–771 2016 Wolters Kluwer Health, Inc. All rights reserved |
| Itaru Yugué, Seiji Okada, Muneaki Masuda, Takayoshi Ueta, Takeshi Maeda & Keiichiro Shiba | Risk factors for adjacent segment pathology requiring additional surgery after single-level spinal fusion: impact of preexisting spinal stenosis demonstrated by perative myelography | Eur Spine J (2016) 25:1542-1549 DOI 10.1007/s00586-015-4185-6 |
| A Matsushita, T Maeda, E Mori, I Yugue, O Kawano, T Ueta and K Shiba | Subacute T1-low intensity area reflects neurological prognosis for patients with cervical spinal cord injury without major bone injury | Spinal Cord (2016) 54, 24–28 & 2016 International Spinal Cord Society All rights reserved 1362-4393/16 |
| Eiji Mori,Takayoshi Ueta,Takeshi Maeda,Itaru Yugué,Osamu Kawano,and Keiichiro Shiba | Effect of preservation of the C-6 spinous process and its paraspinal muscular attachment on the prevention of postoperative axial neck pain in C3-6 laminoplasty. | J Neurosurg Spine Month Day,2014 |
| tetsuo hayashi, md, phd,1,2 michael d. daubs, md,1 akinobu Suzuki, md, phd,1trevor p. Scott, md,1 Kevin h. phan, md,1 monchai ruangchainikom, md,1 Shinji takahashi,md, phd,1 Keiichiro Shiba, md, phd,2 and Jeffrey c. wang, md1 | Motion characteristics and related factors of Modic changes in the lumbar spine | J Neurosurg Spine 22:511-517,2015 |

(独)労働者健康安全機構　吉備高原リハビリテーションセンター

| | | |
|---|---|---|
| Furusawa K and Tajima F | Geriatric Spinal Cord Injuries: Rehabilitation Perspective | Harvinder Singh Chhabra eds. ISCoS Textbook on COMPREHENSIVE MANAGEMENT OF SPINAL CORD INJURIES. Wolters Kluwer. pp960-967, 2015. |

北海道大学大学院医学研究科　機能再生医学講座　整形外科学分野

| | | |
|---|---|---|
| Masahiko Takahataa, Kuniyoshi Abumia, Hideki Sudoa, Ken Nagahamaa & Norimasa Iwasakia | Cervical myelopathy due to atraumatic odontoid fracture in patients with rheumatoid arthritis: A case series | Mod Rheumatol. 2015 Mar 16:1-14. |

独協医科大学医学部医学科　整形外科学

| | | |
|---|---|---|
| Satoshi Inami, Hiroshi Moridaira,Daisaku Takeuchi, Yo Shiba, Yutaka Nohara & Hiroshi Taneichi | Optimum pelvic incidence minus lumbar lordosis value can be determined by individual pelvic incidence | Eur Spine J DOI 10.1007/s00586-016-4563-8 |

Spine

SPINE Volume 40, Number 6, pp 392-398


DIAGNOSTICS

# Abnormal Findings on Magnetic Resonance Images of the Cervical Spines in 1211 Asymptomatic Subjects

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**Study Design.** Cross-sectional study.

**Objective.** The purpose of this study was to determine the prevalence and distribution of abnormal findings on cervical spine magnetic resonance image (MRI).

**Summary of Background Data.** Neurological symptoms and abnormal findings on MR images are keys to diagnose the spinal diseases. To determine the significance of MRI abnormalities, we must take into account the (1) frequency and (2) spectrum of structural abnormalities, which may be asymptomatic. However, no large-scale study has documented abnormal findings of the cervical spine on MR image in asymptomatic subjects.

**Methods.** MR images were analyzed for the anteroposterior spinal cord diameter, disc bulging diameter, and axial cross-sectional area of the spinal cord in 1211 healthy volunteers. The age of healthy volunteers prospectively enrolled in this study ranged from 20 to 70 years, with approximately 100 individuals per decade, per sex. These data were used to determine the spectrum and degree of disc bulging, spinal cord compression (SCC), and increased signal intensity changes in the spinal cord.

**Results.** Most subjects presented with disc bulging (87.6%), which significantly increased with age in terms of frequency, severity, and number of levels. Even most subjects in their 20s had bulging discs, with 73.3% and 78.0% of males and females, respectively. In contrast, few asymptomatic subjects were diagnosed with SCC (5.3%) or increased signal intensity (2.3%). These numbers increased with age, particularly after age 50 years. SCC mainly involved 1 level (58%) or 2 levels (38%), and predominantly occurred at C5–C6 (41%) and C6–C7 (27%).

**Conclusion.** Disc bulging was frequently observed in asymptomatic subjects, even including those in their 20s. The number of patients with minor disc bulging increased from age 20 to 50 years. In contrast, the frequency of SCC and increased signal intensity increased after age 50 years, and this was accompanied by increased severity of disc bulging.

**Key words:** magnetic resonance image (MRI), abnormal findings, asymptomatic, cervical, disc degeneration, disc bulging, spinal cord compression, increased signal intensity, cervical myelopathy, aging, cross-sectional study.

**Level of Evidence:** 2

**Spine 2015;40:392–398**

Magnetic resonance image (MRI) is a useful tool for the diagnosis of cervical spine disorders. Surgeons plan spinal surgical procedures based on neurological symptoms and abnormal MRI findings. However, there is an ongoing debate on the validity of abnormal MRI findings to make such decisions because they are also frequently reported in asymptomatic subjects.[1–9] The relevance of abnormalities on MR image depends on the frequency and spectrum of asymptomatic structural abnormalities.

To our current knowledge, most of the previous studies relating to asymptomatic abnormal findings on cervical spine MR image were limited to small cohort studies[1–8] and the population were not equally distributed in each decade.[1–9] Moreover, few studies investigated abnormal findings in the spinal cord,[1,5,7,9] whereas majority of the studies reported on disc degeneration.[1–7] Finally, there are little data available on the frequency or severity of asymptomatic cervical spinal canal stenosis, or increased signal intensity (ISI) changes,[9,10] which is the representative sign on MR image for cervical compressive myelopathy.[11]

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In cervical compressive myelopathy, static and dynamic factors are the main contributing factors of cervical spinal cord compression (SCC).[12,13] The static factors are the structural spondylotic changes causing canal stenosis and subsequent compression.[12,13] Disc degeneration is suspected as the initiating event of these spondylotic changes that might result in SCC.[12,13] However, there are no data available on the relationship between disc degeneration and SCC.

The purpose of this study was to determine the frequency and severity of abnormal findings on cervical spine MR image in a large cohort of asymptomatic subjects, namely disc bulging, SCC, and ISI changes, and investigate the spatial relationship between disc bulging and SCC.

## MATERIALS AND METHODS

A total of 1230 healthy volunteers were examined using cervical spine MR image between February 2006 and February 2008. Subjects recruited were between 20 and 79 years. We recruited the patients *via* newspaper advertisements and posters in facilities having some sort of relationship with our hospital. Thus, the majority of subjects were not patients at our hospital but healthy residents of the area. The hospital where this study was performed is in one of the biggest cities, Nagoya in Japan, and the majority of the subjects lived within its city limits. The exclusion criteria included a history of brain or spinal surgery, comorbid neurological disease (*e.g.*, cerebral infarction or neuropathy), symptoms related to sensory or motor disorders (numbness, clumsiness, motor weakness, or gait disturbances), or severe neck pain. Pregnant females, and individuals who received workmen's compensation, or presented with symptoms after a motor vehicle accident were also excluded. Subjects with other comorbidities (smoking, diabetes, hypertension, and others) were included in this study. This study was approved by the institutional review board, and each patient signed a written consent form.

All participants underwent imaging analysis and clinical examination by 2 spinal surgeons (F.K. and K.S.). The MRI data from 1211 subjects were included in the analysis, after excluding those with measurement difficulties resulting from artifacts, such as motion or metals. MRIs were performed with a 1.5-T superconductive magnet (Signa Horizon Excite HD version 12; GE Healthcare, Britain, United Kingdom). The scans were taken at slice thicknesses of 3 and 4 mm in the sagittal and axial planes, respectively. T2-weighted images (fast spin echo TR, 3500 ms; TE, 102 ms) were obtained in sagittal scans. Axial scans were performed using T2-weighted images (fast spin echo TR, 4000 ms; TE, 102 ms). All images were transferred to a computer as Digital Imaging and Communications in Medicine data to measure the anteroposterior diameter of the spinal cord, disc bulging diameter, and axial cross-sectional area of the spinal cord, both at the disc and midvertebral level, using imaging software (Osiris4; Icestar Media Ltd., Essex, United Kingdom). Disc bulging, SCC, and ISI change in T2 sagittal images were individually recorded.

By definition, SCC was identified when the anteroposterior diameter of the spinal canal at the narrowest level is less than or equal to the anteroposterior diameter of the spinal cord at the mid C5 vertebral body level (Figure 1).[14] This definition is based on the fact that (1) a sagittal diameter of the spinal canal at the C5 vertebral body level on radiograph is generally used to define developmental stenosis of the cervical spinal canal and (2) there was no case of SCC at the mid C5 vertebral body level in our previous report.[14] Disc bulging was defined as the intervertebral disc protruding posteriorly by more than 1 mm. ISI changes in the spinal cord were classified into 3 groups based on sagittal T2-weighted images as shown in our previous article[10]: grade 0, none; grade 1, light (increased intensity, but less intense compared with cerebrospinal fluid signal); and grade 2, intense (similar intensity to cerebrospinal fluid signal). Grades 1 and 2 signal-intensity changes were included in this study.

### Statistical Analysis

The Fisher exact test or *t* test was used to evaluate differences in abnormal findings between 2 consecutive decades. We plotted receiver operating characteristic analysis to determine the cutoff value to know (1) how big of a disc-bulge diameter would cause SCC to occur more frequently, and (2) how much SCC would increase ISI incidence. A $P$ value less than 0.05 was considered statistically significant. All analyses were conducted using SPSS version 21 (SPSS, Chicago, IL).

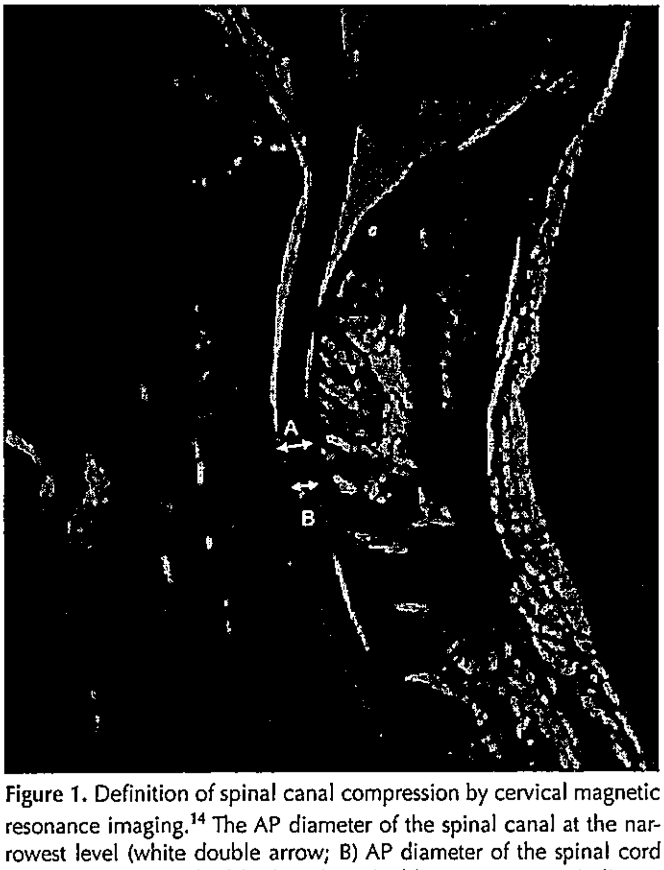


Figure 1. Definition of spinal canal compression by cervical magnetic resonance imaging.[14] The AP diameter of the spinal canal at the narrowest level (white double arrow; B) AP diameter of the spinal cord at the mid C5 vertebral body (white double arrow; A). AP indicates anteroposterior.

## RESULTS

The 1211 asymptomatic volunteers included in this study were equally distributed among age classes, from the third to the eighth decade of life (Table 1). Approximately, 50% of the subjects had passive occupations, mainly as office workers, teachers, or service providers, whereas 28% of them had physically demanding occupations, like housekeepers, builders, and manufacturers (Table 2).

### Disc Bulging

Most asymptomatic volunteers (87.6%) had significant disc bulging. The incidence was already very high in the subjects in their 20s, with 73.3% of the males and 78.0% of the females having disc bulging (Figure 2A). The frequencies tended to increase with age from the 20s to the 50s, with a significant increase from the 30s to the 40s in males ($P < 0.05$). The number of bulging discs in each subject also increased with age (Figure 2B). In the subjects in their 20s, the average number of levels implicated was 1.5 ± 1.3 and 1.0 ± 1.4 for males and females, respectively. Thereafter, the sex difference was lost as the number of levels increased significantly from the 20s to 40s ($P < 0.05$ to 0.001), and reached a plateau (approximately 2 levels) in the 40s. The average disk displacement gradually increased with age from the 30s to the 60s ($P < 0.05$; Figure 2C), reaching 2.5 ± 0.7 mm and 2.0 ± 0.7 mm in males and females in their 70s, respectively.

### Spinal Cord Compression

The diagnosis of SCC was confirmed in 64 (5.3%) subjects. The age and sex distribution of the SCC cases is presented in Figure 3A. Ossification of the posterior longitudinal ligament (OPLL) was observed in 5 people (0.4%) as in our previous report.[14] Although our population was Japanese, the majority of SCC cases were due to other degenerative changes. There was no case of SCC in the subjects in their 20s, and the number increased gradually with age. In addition, SCC was more common in males than in females in all generations. Thirty-seven cases had SCC in 1 level, 24 in 2 levels, 2 in 3 levels, and 1 in 4 levels; they were located predominantly at C5–C6 (41%) and C6–C7 (27%; Figure 3B). The axial cross-sectional area of the dural sac was 112.5 ± 23.3 $mm^2$ in cases of SCC. The most severe case of SCC had a 77.6% reduction in cross-sectional area at C5–C6, compared with the C5 mid-vertebral body (Figure 4).

TABLE 1. Age and Sex of 1211 Asymptomatic Subjects

| Age (yr) | Males | Females |
|---|---|---|
| 20–29 | 101 | 100 |
| 30–39 | 104 | 99 |
| 40–49 | 100 | 100 |
| 50–59 | 99 | 103 |
| 60–69 | 101 | 103 |
| 70–79 | 101 | 100 |
| Total | 606 | 605 |

TABLE 2. Occupation of 1211 Asymptomatic Subjects

| Occupation | No. |
|---|---|
| Office workers | 196 |
| Teachers | 196 |
| Service providers | 101 |
| Doctors, nurses, and medical coworkers | 58 |
| Sales persons | 57 |
| Students | 16 |
| Subtotal = 624 (51.5%) | |
| Housekeepers | 193 |
| Builders | 78 |
| Manufacturers | 54 |
| Carriers | 15 |
| Farmers | 3 |
| Subtotal = 343 (28.3%) | |
| Unemployed persons | 124 |
| Others | 100 |
| Unknown | 20 |
| Subtotal = 244 (20.1%) | |
| Total | 1211 |

### Increased Signal Intensity

A small fraction of the subjects (N = 28; 2.3%) exhibited significant changes in ISI on T2 sagittal images. The distribution of ISI cases per decade and sex is shown in Figure 5A. This MRI abnormality was more common in males than females of all generations, as in the case of SCC. The incidence of ISI increased with age, particularly after the 50s, reaching 9% and 4% for males and females in their 70s, respectively. Most cases of ISI (89%) involved 1 level. Every ISI coincided with the level of SCC, primarily at C4–C5 (36%) and C5–C6 (54%) (Figure 5B).

### The Relationship Between Disc Bulging, SCC, and ISI

A disc bulge of more than 1.35 mm was a risk factor for SCC (area under curve = 0.87, $P < 0.0001$, Figure 6A), and an SCC area of less than 128.5 $mm^2$ was a risk factor for ISI (area under curve = 0.92, $P < 0.0001$, Figure 6B).

### Presentation of the Most Severe Case of SCC

The patient was a 77-year-old male with no clinical subjective symptoms, such as gait disturbance or numbness in his extremities. His manual muscle test results were 5. The result of the 10-second grip and release test[15] was 21/22 times in the

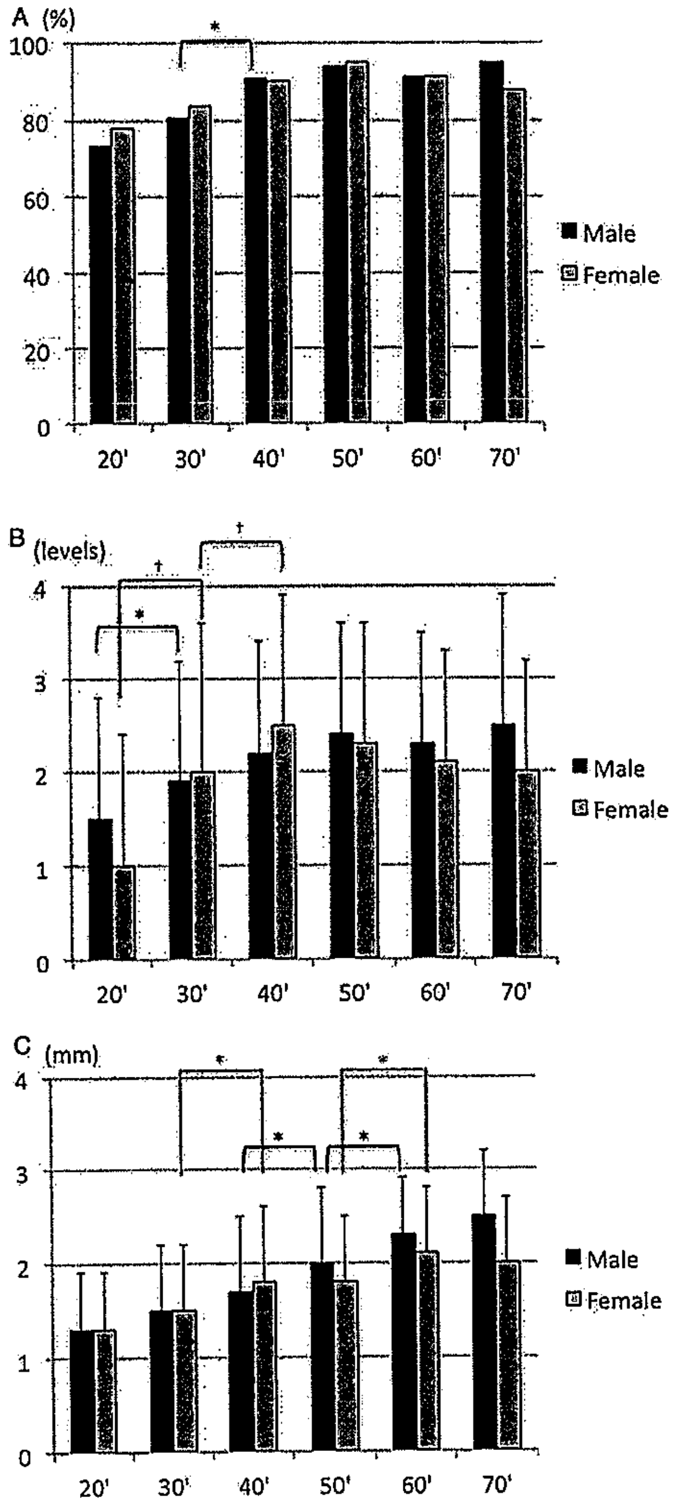


Figure 2. Frequency distribution of disc bulging in asymptomatic subjects. A, Frequency distribution of disc bulging with age and sex. B, Frequency distribution of the number of levels involved in disc bulging. C, Impact of age and sex on disc displacement (mm). Values are mean + SD. $*P < 0.05$, $\dagger P < 0.001$. SD indicates standard deviation.

right/left hand, respectively, and the result of the 10-second step test[16] was 15. The deep tendon reflex, triceps, patella, and Achilles tendon reflex were hyper, whereas the deltoid,

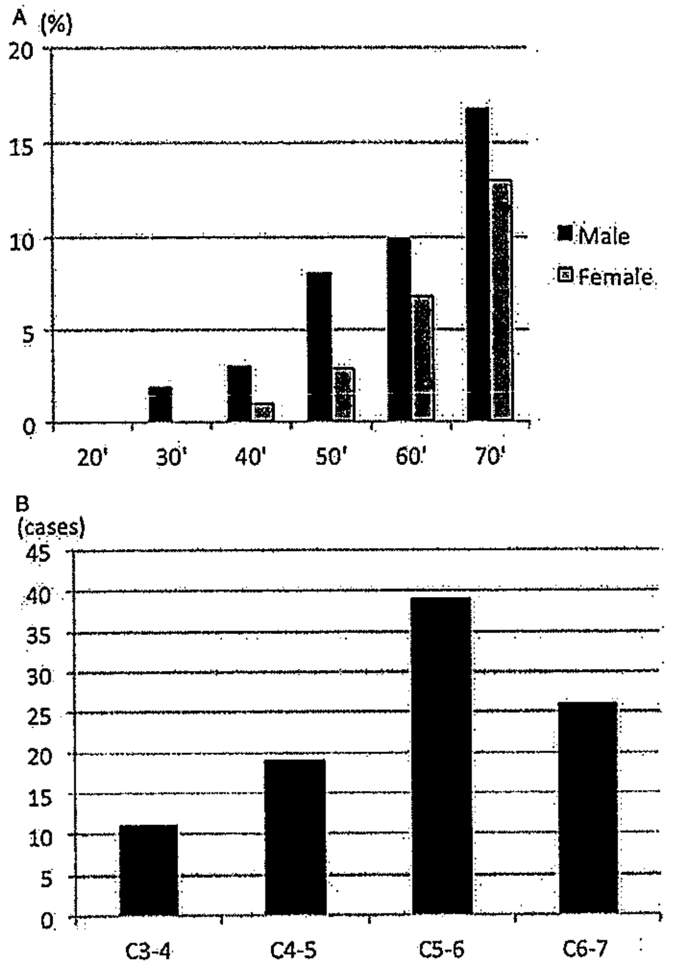


Figure 3. Frequency distribution of cervical SCC in asymptomatic subjects. A, Frequency distribution of SCC with age and sex. B, Frequency distribution of SCC along the spine. SCC indicates spinal cord compression.

biceps, and brachioradialis tendon reflex were normal. Tromner-Hoffman sign[17] were negative on both sides, but the Wartenberg sign[15] was positive on both sides. MR image showed fusion of the C5 and C6 vertebrae, and local kyphosis at C4–C6 (Figure 4). SCC was detected at C4–C5 and C5–C6, with ISI at C5–C6.

## DISCUSSION

This study constituted the largest prospective evaluation of cervical spine MR image in asymptomatic subjects. This comprehensive survey demonstrated that small disc bulging was frequently observed even in the subjects in their 20s. In addition, the number of patients with minor disc bulging and the number of levels with small disc bulging increased from age 20 to 50 years. In contrast, the frequency of SCC and ISI increased after age 50 years, and this was accompanied by increased severity of disc bulging.

The increasing incidence of cervical disc bulging with aging among asymptomatic subjects has been extensively documented.[1–8] Cervical disc degeneration or bulging is frequently reported in asymptomatic subjects in their 40s and 50s.[1,3] In

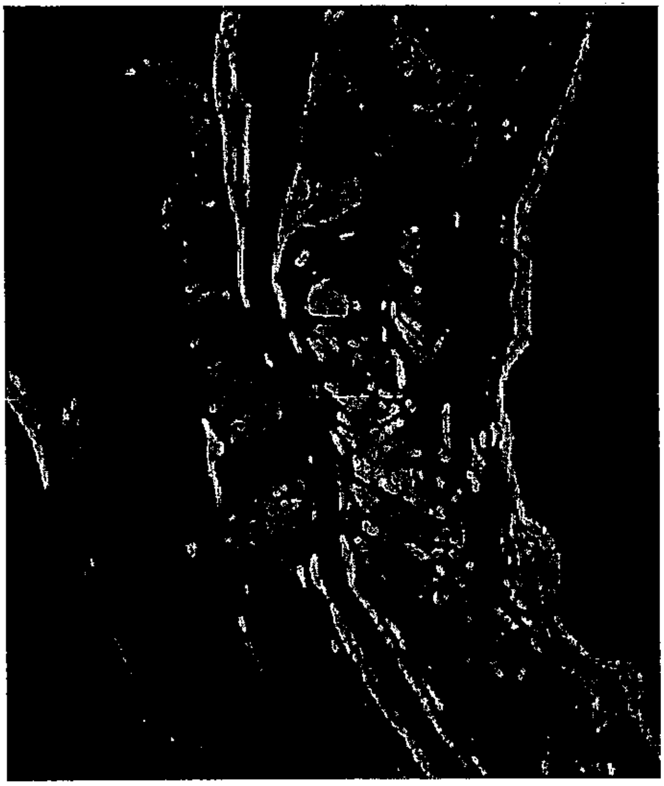

Figure 4. Spine magnetic resonance imaging T2-weighted sagittal image of a 77-year-old asymptomatic male. There is fusion of the C5 and C6 vertebrae, and local kyphosis at C4–C6. Spinal cord compression detected at C4–C5 and C5–C6, with high-signal intensity change at C5–C6.

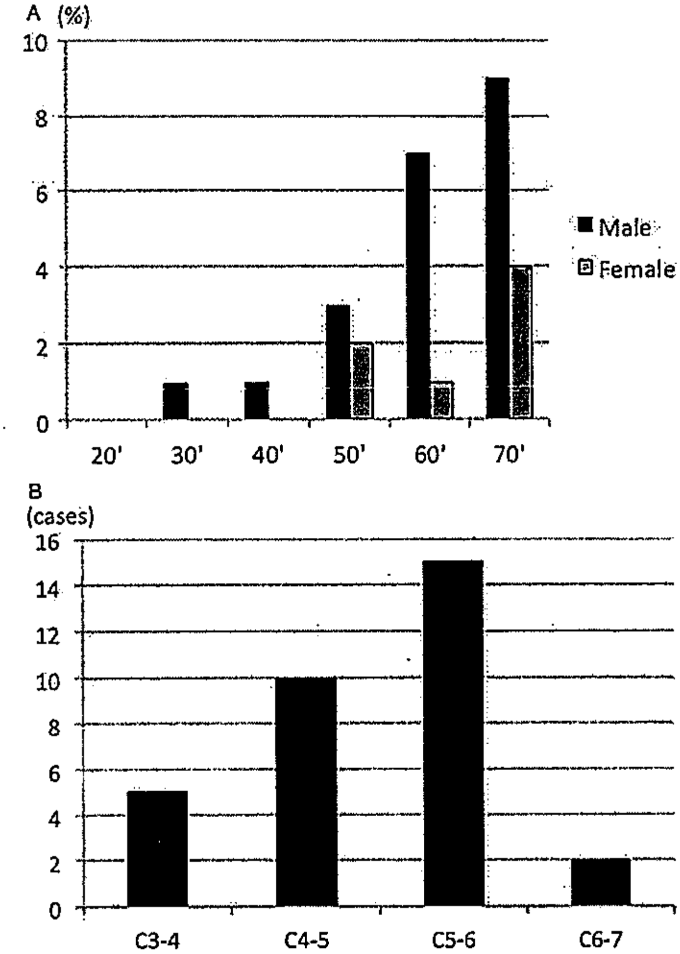


Figure 5. Frequency distribution of ISI changes on MR image in asymptomatic subjects. A, Frequency distribution of ISI with age and sex. B, Frequency distribution of ISI along the spine. ISI indicates increased signal intensity; MRI, magnetic resonance image.

this study, the number of cases and levels with small disc bulging increased and reached a plateau in the 50s. However, the severity of disc displacement continued to increase even after the 50s. Such disc bulging enlargement with age is highly suspected as a cause of spinal canal stenosis after the 50s.

Spinal canal stenosis is also known to gradually increase with age.[1,9,14] The reduction in spinal canal size occurred more frequently at the disc level than at the midvertebral level, particularly at C5–C6.[14] In addition to disc bulging, cervical spine alignment change could be another cause of SCC. Cervical lordosis in the neutral position increases with age, particularly in the 60s.[18] Changes in cervical alignment could compensate for the growing spinal canal stenosis with age. Spinal canal stenosis occurs by pincers effect, which defines pinching of the spinal cord between the ligamentum flavum and intervertebral disc. This effect is more pronounced in the lordotic alignment.[19] Accordingly, more severe pincer effects could occur in older populations with large disc bulging and lordotic alignment.

Few reports mentioned the severity of spinal canal stenosis in an asymptomatic population.[7] Teresi *et al*[7] noted that the reduction in cross-sectional area of the spinal cord never exceeded 16% in the asymptomatic population. However, in this study, the most severe case of SCC showed the reduction of the cross-sectional area exceeding 75%. Although the critical value at which symptoms manifest is not clear, a significant degree of SCC can be tolerated without any symptoms. Hamburger *et al*[20] reported the axial cross-sectional area of the dural sac in patients with cervical myelopathy; the preoperative and postoperative areas were 92 ± 37 mm$^2$ and 154 ± 36 mm$^2$, respectively. The axial cross-sectional area of the dural sac was 113 mm$^2$ in cases of SCC in our asymptomatic subjects, and the cross-sectional area in cases of SCC in asymptomatic subjects was not as severe as in cases with symptomatic cervical myelopathy. The severity of stenosis was just midway between that of the pre- and postoperative conditions of patients with symptomatic cervical myelopathy. This result could be valuable for knowing the degree of stenosis in symptomatic patients.

Although we performed receiver operating characteristic analysis to detect a relationship between disc bulging, SCC and ISI, disc bulge of more than 1.35 mm is not particularly severe, and so it seems likely that the combination of developmental canal stenosis, hypertrophy of ligamentum flavum,

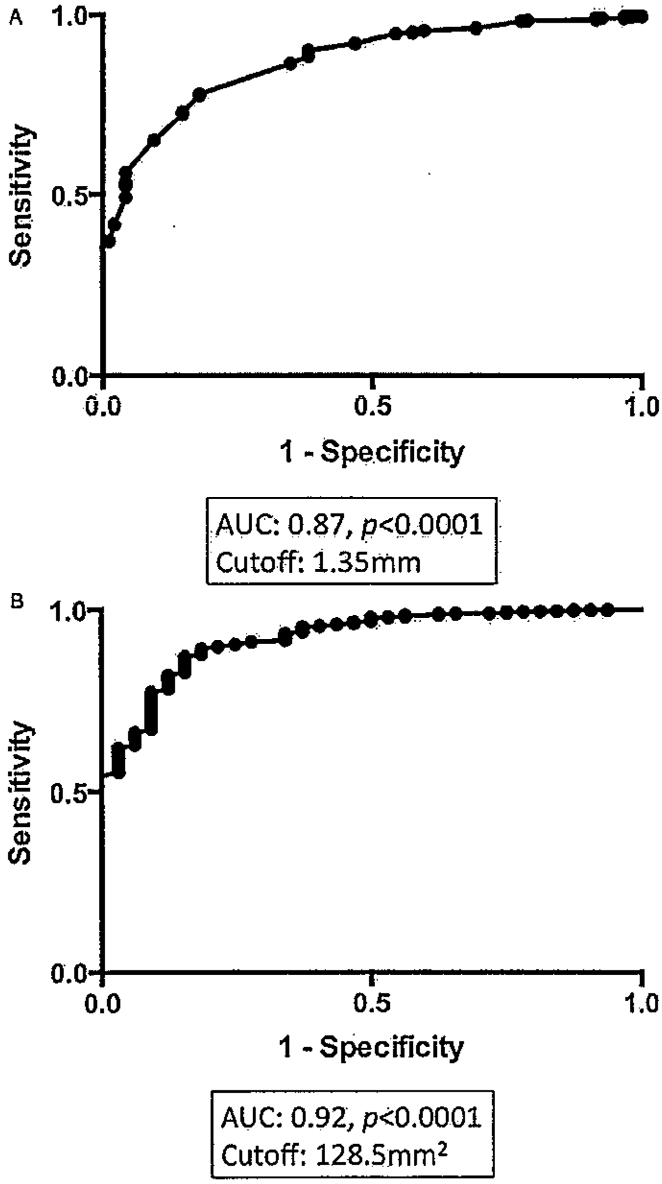


**Figure 6.** The ROC curves to determine (A) how big of a disc-bulge diameter would cause SCC to occur more frequently, and (B) how much SCC would increase ISI incidence. A, The relationship between disc bulging and SCC. B, The relationship between SCC and ISI. SCC indicates spinal cord compression; ISI, increased signal intensity; ROC, receiver operating characteristic.

and deformity of cervical spine is what is important for the occurrence of SCC.[12,13]

This study has some limitations. First, the survey was limited to the Japanese population, which does not rule out racial differences. Second, this large cohort included subjects with a wide variety of occupations in terms of physical demand, which may influence the progression or severity of cervical degenerative disease. Third, we used our original definition of SCC. To objectively and quantitatively evaluate SCC in asymptomatic subjects, we newly established this definition of SCC. In the other previous articles, SCC was defined as the presence of a defect in the cord, a definition that was subjective and not quantitative.[1,21] Especially in asymptomatic cases, interobserver reliability in SCC is not very high because canal compression in those cases was not severe.[21] Our definition is useful in asymptomatic subjects, however this might not be useful in symptomatic cases, especially ones with severe deformity or continuous type OPLL, and so further discussion is needed.

## CONCLUSION

This large prospective analysis of cervical spine MRI data in asymptomatic subjects demonstrates the high frequency and multiple levels of degenerative change in the spinal cord and discs. Then, it is dangerous to make interventional decisions only by judging degenerative changes using MR images alone.

The results in this study alerted us to the fact that clinical decision making should be prudent, correlating MRI findings with clinical signs and symptoms. Future studies are required to monitor the progression of asymptomatic SCC to identify the MRI abnormalities that would predict the emergence of symptomatic cervical degenerative disease.

### Key Points

- Cervical disc bulging, SCC, and ISI changes were evaluated on cervical MR images of 1211 healthy volunteers.
- Most subjects presented with disc bulging (87.6%); the frequency, severity, and number of levels involved significantly increased with age.
- The frequency of SCC and ISI was 5.3% and 2.3%, respectively.
- The number of patients with minor disc bulging and the number of levels with small disc bulging increased from age 20 to 50 years.
- In contrast, the frequency of SCC and ISI increased after age 50 years, and this was accompanied by increased severity of disc bulging.

## References

1. Boden SD, McCowin P, Davis D, et al. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg* 1990;72:1178–84.
2. Schellhas KP, Smith MD, Gundry CR, et al. Cervical discogenic pain: prospective correlation of magnetic resonance imaging and discography in asymptomatic subjects and pain sufferers. *Spine* 1996;21:300–11.
3. Matsumoto M, Fujimura Y, Suzuki N, et al. MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg Br* 1998;80:19–24.
4. Lehto I, Tertti M, Komu M, et al. Age-related MRI changes at 0.1 T in cervical discs in asymptomatic subjects. *Neuroradiology* 1994;36:49–53.

5. Matsumoto M, Okada E, Ichihara D, et al. Age-related changes of thoracic and cervical intervertebral discs in asymptomatic subjects. *Spine* 2010;35:1359–64.
6. Siivola SM, Levoska S, Tervonen O, et al. MRI changes of cervical spine in asymptomatic and symptomatic young adults. *Eur Spine J* 2002;11:358–63.
7. Teresi LM, Lufkin RB, Reicher MA, et al. Asymptomatic degenerative disk disease and spondylosis of the cervical spine: MR imaging. *Radiology* 1987;164:83–8.
8. Okada E, Matsumoto M, Fujiwara H, et al. Disc degeneration of cervical spine on MRI in patients with lumbar disc herniation: comparison study with asymptomatic volunteers. *Eur Spine J* 2011;20:585–91.
9. Nagata K, Yoshimura N, Muraki S, et al. Prevalence of cervical cord compression and its association with physical performance in a population-based cohort in Japan: the Wakayama Spine Study. *Spine* 2012;37:1892–8.
10. Yukawa Y, Kato F, Yoshihara H, et al. MR T2 image classification in cervical compression myelopathy: predictor of surgical outcomes. *Spine* 2007;32:1675–8.
11. Kalsi-Ryan S, Karadimas SK, Fehlings MG. Cervical spondylotic myelopathy the clinical phenomenon and the current pathobiology of an increasingly prevalent and devastating disorder. *Neuroscientist* 2013;19:409–21.
12. Karadimas SK, Erwin WM, Ely CG, et al. The pathophysiology and natural history of cervical spondylotic myelopathy. *Spine* 2013;38:S21–36.
13. Karadimas SK, Gatzounis G, Fehlings MG. Pathobiology of cervical spondylotic myelopathy. *Eur Spine J* 2014:1–7.
14. Kato F, Yukawa Y, Suda K, et al. Normal morphology, age-related changes and abnormal findings of the cervical spine. Part II: magnetic resonance imaging of over 1200 asymptomatic subjects. *Eur Spine J* 2012;21:1499–507.
15. Ono K, Ebara S, Fuji T, et al. Myelopathy hand. New clinical signs of cervical cord damage. *J Bone Joint Surg Br* 1987 ;69:215–9.
16. Yukawa Y, Kato F, Ito K, et al. "Ten second step test" as a new quantifiable parameter of cervical myelopathy. *Spine (Phila Pa 1976)* 2009;34:82–6.
17. Chang C-W, Chang K-Y, Lin S-M. Quantification of the Tromner signs: a sensitive marker for cervical spondylotic myelopathy. *Eur Spine J* 2011;20:923–7.
18. Yukawa Y, Kato F, Suda K, et al. Age-related changes in osseous anatomy, alignment, and range of motion of the cervical spine. Part I: radiographic data from over 1200 asymptomatic subjects. *Eur Spine J* 2012;21:1492–8.
19. Muhle C, Metzner J, Weinert D, et al. Classification system based on kinematic MR imaging in cervical spondylitic myelopathy. *Am J Neuroradiol* 1998;19:1763–71.
20. Hamburger C, Büttner A, Uhl E. The cross-sectional area of the cervical spinal canal in patients with cervical spondylotic myelopathy: correlation of preoperative and postoperative area with clinical symptoms. *Spine* 1997;22: 1990–4.
21. Nagata K, Yoshimura N, Hashizume H, et al. The prevalence of cervical myelopathy among subjects with narrow cervical spinal canal in a population-based MRI study: The Wakayama Spine Study. *Spine J* 2014;14:2811–7.

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ORIGINAL ARTICLE

# Prospective study of deep vein thrombosis in patients with spinal cord injury not receiving anticoagulant therapy

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**Study Design:** Prospective cross-sectional study.
**Objectives:** To investigate the timing of deep vein thrombosis (DVT) onset secondary to spinal cord injury without anticoagulant therapies.
**Setting:** Spinal Cord Injury Center in Hokkaido, Japan.
**Methods:** Between November 2012 and June 2013, patients with spinal cord injury who were admitted to our hospital within 1 day after the injury and treated surgically within 24 h underwent a neurological examination, leg vein ultrasonography and D-dimer test 1, 3, 7, 14 and 28 days after surgery. All patients received treatment with intermittent pneumatic compression and elastic stockings, but without any anticoagulant.
**Results:** DVT developed in 12 patients (11 men and 1 women), with a mean age of 62.2 years (range, 41–80 years; mean age of total sample, 63.2 years (range, 25–78 years)), all distal to the popliteal vein. DVT occurred more often with a more severe paralysis (66.3%, AIS A and B). The median (± standard error) length of time from the operation to DVT detection was 7.5 ± 2.2 days. The mean D-dimer level upon DVT detection was 14.6 ± 11.8 μg ml$^{-1}$, with no significant differences between those who developed DVT and those who did not at any of the time points.
**Conclusion:** These results suggest that DVT can develop at the very-acute stage of spinal cord injury and the incidence increases with a more severe paralysis. DVT detection was more reliable with ultrasonography, which should be used with DVT-preventive measures, beginning immediately after the injury, for the management of patients with spinal cord injury.
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## INTRODUCTION

Deep vein thrombosis (DVT) is one of the most dangerous complications of a spinal cord injury. DVT has a high incidence in patients with spinal cord injury as compared with patients with other diseases[1] with a high mortality rate if complicated by pulmonary embolism.[2]

Although prevention, early detection and timely treatment of DVT are important during the management of patients with spinal cord injury, the timing of DVT onset secondary to a spinal cord injury has not yet been determined. In order to clarify these problems, this study aimed to investigate the timing of DVT onset by prospectively analyzing the patients immediately after a spinal cord injury.

## MATERIALS AND METHODS

A prospective study was conducted in all patients with spinal cord injury admitted to our hospital between November 2012 and June 2013 within 1 day after the injury and treated surgically within 24 h. A neurological examination, leg vein ultrasonography and clotting system test (D-dimer) was performed 1, 3, 7, 14 and 28 days after surgery. Leg vein ultrasonography was carried out by using an Aplio XG (TOSHIBA, Tokyo, Japan) by experienced sonographers.

Intermittent pneumatic compression (IPC) with a calf pump (Kendall SCD-EXPRESS, COVIDINE, Dublin, Ireland) and elastic stockings (ES) were used in all cases. The patient were kept foot pump attached throughout the day (except when the patient left the bed) for at least 2 consecutive weeks. The use of ES began on the day after the operation, as far as possible, with a median of 3 days between the operation and their use. Patients were advised to keep them on, except when bathing. One patient refused to wear them, and two patients had difficulty wearing them because of skin irritation. The other patients continued to use them until discharge from the hospital.

As a rule, we allowed the patients to leave the bed by using a wheelchair from the day after the surgery, with a median (± standard error) of 3 ± 0.7 days from the surgery. Ten patients were unable to leave their bed on the day after the operation because of elective surgery (six cases), mechanical ventilation (two cases) and chest tube (two cases).

We certify that all applicable institutional and governmental regulations concerning the ethical use of human volunteers were followed during the course of this research.

## RESULTS

The sample included 25 men and 4 women with a mean age of 63.2 years (25–78 years). The spinal cord injury occurred at the cervical segment in 19 cases and thoracic/lumbar segment in 10 cases. The cause of spinal cord injury was a fall from a great height in 11 cases, fall from a low height in 2 cases, same-level fall in 8 cases, traffic accident in 4 cases, fall from a staircase in 1 case and sports accident in 3 cases. The median period from injury to hospitalization was 0 day (0–1 day). Twenty-four patients were admitted to our hospital on the day of the injury and 5 were hospitalized on the following day. The severity of paralysis at admission (American Spinal Cord Association

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(ASIA) Impairment Scale (AIS)) was A in 9 cases, B in 2 cases, C in 8 cases and D in 10 cases. The paralyzed level (ASIA Neurological Level of Injury (NLI)) was C3–4 in 11 cases, C5–8 in 8 cases, T1–12 in 6 cases and L1–4 in 4 cases. DVT developed in 12 patients (41.4%; 11 men and 1 woman), with a mean age of 62.2 years (range, 41–80 years). In all of these cases, the DVT was located distal to the popliteal vein. DVT affected both sides in 2 cases, the left side in 5 cases and the right side in 5 cases. Among these 12 patients, the injured level was the cervical segment in 7 cases and thoracic/lumbar segment in 5 cases, and the paralyzed level according to the NLI classification was C3–4 in 4 cases, C5–8 in 2 cases, T1–12 in 3 cases and L1–4 in 3 cases. The severity of paralysis (AIS) was A in 7 (58.3%) of these 12 cases, B in 1 case (8.3%), C in 3 cases (25.0%) and D in 1 case (8.3%). Thus, DVT occurred more often with paralysis of AIS grades A and B (complete motor paralysis) than with a partial motor paralysis (Tables 1a and b). DVT was detected on postoperative day 3 in 3 cases (25.0%), postoperative day 7 in 5 cases (41.7%), postoperative day 14 in 1 case (8.3%) and postoperative day 28 in 3 cases (25.0%). The median length of time from the operation to DVT detection was $7.5 \pm 2.2$ days ($\pm$ standard error) (Table 2).

The mean D-dimer level at DVT detection was $14.6 \pm 13.5\ \mu g\ ml^{-1}$ (range, 2.78–44.3 $\mu g\ ml^{-1}$). There was no significant difference between the DVT negative group and DVT positive group in terms of D-dimer level upon admission or on postoperative day 1, 3, 7, 14 or 28 (Mann–Whitney test, $P = 0.117$, 0.059, 0.028, 0.075, 0.306 and 0.124, respectively).

The cutoff D-dimer levels determined from the receiver operator characteristic (ROC) curve was 7.34, 7.88, 5.82, 10.99, 11.44, and 5.88 at admission and on the postoperative days 1, 3, 7, 14 and 28, respectively. The sensitivity/specificity (%) at these points of time were 75.0/70.6, 41.7/94.1, 72.7/76.5, 55.6/88.2, 60.0/93.3 and 75.0/93.3, respectively (Table 3).

**Table 1a** Participants' characteristics ($N = 29$)

| *Variable* | *Total (N = 29)* n(%) | *DVT Positive (N = 12)* % |
|---|---|---|
| *Age (median years)* | 63.2 (25–78) | 62.2 (41–80) |
| *Gender* | | |
| Male | 25 (86) | 11 (92) |
| Female | 4 (14) | 1 (8) |
| *Lesion level* | | |
| Cervival | 19 (66) | 7 (58) |
| Thoracic | 4 (14) | 2 (17) |
| Lumbar | 6 (20) | 3 (25) |
| *Causes* | | |
| High fall | 11 (38) | 5 (42) |
| Low fall | 2 (7) | 3 (25) |
| Fall at ground level | 8 (28) | 2 (17) |
| Road traffic accidents | 4 (14) | 1 (8) |
| Stairs | 1 (3) | 0 |
| Sports | 3 (10) | 1(8) |
| *AIS* | | |
| A | 9 (31) | 7 (58) |
| B | 2 (7) | 1 (8) |
| C | 8 (28) | 3 (25) |
| D | 10 (34) | 1 (8) |
| *Neurological level of injury* | | |
| C3–4 | 11 (38) | 4 (27) |
| C5–8 | 8 (27) | 2 (17) |
| T1–12 | 6 (21) | 3 (25) |
| L1–4 | 4 (14) | 3 (25) |
| *Comorbid injury* | | |
| Fracture | | |
| Rib | 1 (3) | 0 |
| Clavicle | 1 (3) | 0 |
| Scapula | 1 (3) | 0 |
| Radius | 2 (7) | 0 |
| Patella | 0 | 1 (8) |
| Skull | 1(3) | 0 |
| Pneumo/hemothorax | 2 (7) | 0 |
| Period from injury to hospitalization (median days) | 0 (0–1) | 0 (0) |

Abbreviations: AIS, The American Spinal Cord Injury Association Impairment scale; DVT, deep vein thrombosis; n, number of subject; U/E, upper extrimities; L/E, lower extrimities.
Multiple injury of the same patient.

**Table 2** Days of DVT detection

| *Variable* | |
|---|---|
| *Day detected (days)* | |
| POD 1 | 0 (0) |
| POD 3 | 3 (25) |
| POD 7 | 5 (41) |
| POD 14 | 1 (8) |
| POD 28 | 3 (25) |
| Days from operation (mean, s.d.) | 7.2 (2.2) |

**Table 1b** DVT participants' characteristics ($N = 12$)

| *Variable* | *Case* 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Age | 78 | 72 | 63 | 60 | 66 | 41 | 63 | 63 | 53 | 49 | 80 | 67 |
| Gender | Male | Male | Male | Male | Female | Male | Male | Male | Male | Male | Male | Male |
| Lesion level | Cervival | Lumbar | Cervival | Thoracic | Lumbar | Thoracic | Cervival | Cervival | Cervival | Cervival | Lumbar | Cervival |
| Causes | Low fall | Low fall | High fall | High fall | High fall | Sports | Low fall | Ground | High fall | Traffic | High fall | Ground |
| AIS | A | C | A | A | C | B | A | A | A | A | C | D |
| Neurological level of injury | C6 | L1 | C3 | T11 | L3 | T6 | C4 | C4 | C3 | C5 | L2 | T8 |
| Comorbid injury | – | – | – | – | – | – | – | – | – | Patella Fx | – | – |
| Period form injury to hospitalization | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 1 |
| From injury to operation | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 0 | Day 1 | Day 1 |
| Day of DVT detetion form injury | Day 3 | Day 7 | Day 28 | Day 7 | Day 7 | Day 3 | Day 7 | Day 28 | Day 7 | Day 14 | Day 29 | Day 4 |
| From operation | Day 3 | Day 7 | Day 28 | Day 7 | Day 7 | Day 3 | Day 7 | Day 28 | Day 7 | Day 14 | Day 28 | Day 3 |
| Ventilator Tx at DVT detection | – | – | – | – | – | – | + | – | – | – | – | – |

Abbreviations: AIS, The American Spinal Cord Injury Association Impairment scale; DVT, deep vein thrombosis; Ground, fall at ground level; Traffic, road traffic accident; Tx, treatment; –, none.

Table 3 Value of D-dimer in DVT positive group

| Variable | Mean D-dimer level (range) (μg ml⁻¹) | | | Cutoff (μg ml⁻¹) | Sensitivity (%) | Specificity (%) |
|---|---|---|---|---|---|---|
| | DVT positive | DVT negative | P-value | | | |
| At admission | 30.9 (1.2–99.6) | 16.7 (1.1–84.0) | 0.117 | 7.34 | 75 | 70.6 |
| POD 1 | 7.5 (2.9–21.0) | 3.8 (0.8–7.9) | 0.059 | 7.88 | 41.7 | 94.1 |
| POD 3 | 11.6 (1.8–35.4) | 5.4 (1.5–16.4) | 0.0287 | 5.82 | 72.7 | 76.5 |
| POD 7 | 13.5 (4.6–44.3) | 6.2 (1.6–16.8) | 0.075 | 10.99 | 55.6 | 88.2 |
| POD 14 | 8.5 (2.8–14.5) | 6.9 (1.5–22.0) | 0.306 | 11.44 | 60 | 93.3 |
| POD 28 | 9.9 (1.9–21.0) | 4.7 (1.1–15.0) | 0.124 | 5.88 | 75 | 86.7 |

Abbreviation: DVT, deep vein thrombosis.
Mann–Whiteney U test.

## DISCUSSION

### Timing and incidence of DVT in spinal cord injury patients

Concerning the pathogenesis of DVT, the three major factors in Virchow's triad (blood retention, capillary wall disorder and blood coagulopathy) are well known.[3] After spinal cord injury, paralysis is accompanied by reduced vascular contraction (because of a sympathetic nerve disorder) and reduced venous return (because of disturbed muscle contraction), leading to blood retention.[4] Furthermore, a possible loss of the circadian variation in hemostatic and fibrinolytic function has also been suggested in patients with spinal cord injury. Thus, DVT is quite likely to develop in the presence of spinal cord injury. Considering the known mechanisms for the onset of DVT, we need to bear in mind that the risk for DVT increases with more severe paralysis and the incidence of DVT is particularly high in patients with complete motor paralysis associated with spinal cord injury.[5,6] In other words, the incidence of DVT is high in paralyzed patients with spinal cord injury; therefore, early detection and treatment are essential in such cases.

For early detection, we need to understand the timing associated with DVT onset; however, we have little information regarding the timing of DVT onset. In a retrospective analysis of 52 patients, Sugimoto *et al.*[7] reported that they observed DVT 2–13 days after injury of the cervical segment of the spinal cord, suggesting that DVT can develop soon after spinal cord injury. To the best of our knowledge, the present study is the first to prospectively document the timing of DVT onset in patients with a spinal cord injury, with analysis commencing immediately after their injury. We detected no DVT positive patient on Day 1 but 3 patients were positive on Day 3, 5 on Day 7, 1 on Day 14 and 3 on Day 28.

As a result, DVT was detected 3 days after injury in 25% of the patients who developed DVT. Especially in the group of AIS A as complete paralysis, DVT occurred in 78% of patients. These findings indicate that proactive DVT prevention and diagnosis, beginning immediately after the injury, are indispensable in the management of the patients with a spinal cord injury.

### How to diagnose DVT in spinal cord injury patients

D-dimer measurement and leg ultrasonography are predominantly used for the diagnosis of DVT. However, according to a study of patients who developed DVT after total knee or hip replacement surgery, both the sensitivity and specificity of D-dimer are low, indicating that D-dimer alone does not provide a sufficient means of DVT diagnosis.[8,9] Similar results were obtained in the present study, and the D-dimer level varied depending on the timing of the measurement, indicating a limitation in the accuracy of diagnosis based on D-dimer levels. DVT-inducing pulmonary embolism is a potentially fatal complication, and it seems advisable to use D-dimer only as an auxiliary indicator.

Diagnostic reliability is highest with leg ultrasonography. The sensitivity of ultrasonography in detecting leg DVT is reported to be 98–100% and its specificity to be 75–100%.[10–12] This is an excellent method for the noninvasive screening of DVT.[13] Shortcomings of this modality include the amount of time and labor required. For spinal cord injury accompanied by paralysis, periodic leg ultrasonography is indispensable. The results of the present study suggest that frequent leg ultrasonography, beginning immediately after injury, is needed for patients with severe paralysis (AIS A-C).

### Prevention of DVT

In patients with a spinal cord injury, the incidence of DVT is quite high, and active measures for prevention are important, beginning from the acute stage of injury.[1] The basic principles for DVT prevention include the prevention of venous retention, stimulation of venous return, and anticoagulation, with the use of ES, IPC and low-dose unfractionated heparin (LDUH), respectively. In the present study, ES and IPC were used immediately after injury, but this approach could not prevent DVT from occurring. These results indicate that it is necessary to include LDUH in the treatment of paralyzed patients with spinal cord injury. However, since patients with spinal cord injury often have hemorrhaging around the spine or spinal cord, physicians are often cautious about the use of LDUH because bleeding inside/outside the spinal cord is likely to aggravate the paralysis.

Recommendations provided in the American College of Chest Physicians Guidelines[13] include the use of low-molecular weight heparin (LMWH) upon confirmation of hemostasis (Grade 2B), combined use of IPC and LDUH (Grade 2C), combined use of IPC and ES in cases where anticoagulants are contraindicated (Grade 1C+), and treatment with LMWH or an oral-dose vitamin K antagonist during rehabilitation (Grade 1C). Christie *et al.*[14] recommended treatment with LMWH within 72 h after injury or re-treatment with the same drug within 24 h after surgery. Further, no difference has been detected in the incidence of DVT or hemorrhagic complications between patients receiving 5000 U of unfractionated heparin every 8 h and those receiving 30 mg LMWH every 12 h, if treatment was started within 72 h after injury.[15] We have no objection to the view that LMWH or LDUH has a central role in the prevention of DVT among patients with spinal cord injury, even in the patients treated surgically. However, there is a particular need to establish the use of these drugs during the acute stage of spinal cord injury.

## CONCLUSION

1. DVT can develop at the very-acute stage of spinal cord injury.
2. The incidence of DVT increased with more severe paralysis.
3. Ultrasonography is a simple and valid means of detecting DVT.
4. Frequent ultrasonogtraphy during early stage is useful for detecting asymptomatic DVT in acute SCI patients.

## DATA ARCHIVING

There were no data to deposit.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

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1 Teasell RW, Hsieh JT, Aubut JA, Eng JJ, Krassioukov A, Tu L *et al.* Venous thromboembolism following spinal cord injury. *Arch Phys Med Rehabil* 2009; 90: 232–245.
2 DeVivo MJ, Krause JS, Lammertse DP. Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil* 1999; 80: 1411–1419.
3 Furlan JC1, Fehlings MG. Cardiovascular complications after acute spinal cord injury: pathophysiology, diagnosis, and management. *Neurosurg Focus* 2008; 25: E13.
4 Miranda AR, Hassouna HI. Mechanisms of thrombosis in spinal cord injury. *Hematol Oncol Clin North Am* 2000; 14: 401–416.
5 Jones T, Ugalde V, Franks P, Zhou H, White RH. Venous thromboembolism after spinal cord injury: incidence, time course, and associated risk factors in 16,240 adults and children. *Arch Phys Med Rehabil* 2005; 86: 2240–2247.
6 Waring WP, Karunas RS. Acute spinal cord injuries and the incidence of clinically occurring. *Paraplegia* 1991; 29: 8–16.
7 Sugimoto Y, Ito Y, Tomioka M, Tanaka M, Hasegawa Y, Nakago K *et al.* Deep venous thrombosis in patients with acute cervical spinal cord injury in a Japanese population: assessment with Doppler ultrasonography. *J Orthop Sci* 2009; 14: 374–376.
8 Chen CJ, Wang CJ, Huang CC. The value of D-dimer in the detection of early deep-vein thrombosis after total knee arthroplasty in Asian patients: a cohort study. *Thromb J* 2008; 6: 5.
9 Shiota N, Sato T, Nishida K, Matsuo M, Takahara Y, Mitani S *et al.* Changes in LPIA D-dimer levels after total hip or knee arthroplasty relevant to deep-vein thrombosis diagnosed by bilateral ascending venography. *J Orthop Sci* 2002; 7: 444–450.
10 Mattos MA, Melendres G, Sumner DS, Hood DB, Barkmeier LD, Hodgson KJ *et al.* Prevalence and distribution of calf vein thrombosis in patients with symptomatic deep venous thrombosis: a color-flow duplex study. *J Vasc Surg* 1996; 24: 738–744.
11 Persson AV, Jones C, Zide R, Jewell ER. Use of the triplex scanner in diagnosis of deep venous thrombosis. *Arch Surg* 1989; 124: 593–596.
12 Comerota AJ, Katz ML, Greenwald LL, Leefmans E, Czeredarczuk M, White JV. Venous duplex imaging: should it replace hemodynamic tests for deep venous thrombosis? *J Vasc Surg* 1990; 11: 53–59.
13 Hirsh J, Guyatt G, Albers GW, Harrington R, Schünemann HJ. American College of Chest Physicians. Executive summary: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). *Chest* 2008; 133: 71S–109S.
14 Christie S, Thibault-Halman G, Casha S. Acute pharmacological DVT prophylaxis after spinal cord injury. *J Neurotrauma* 2011; 28: 1509–1514.
15 Spinal Cord Thromboprophylaxis Investigators. Prevention of venous thromboembolism in the acutetraumatic phase after spinal cord injury: a randomized, multicenter trial comparing low-dose heparin plus intermittent pneumatic compression with enoxaparin. *J Trauma* 2003; 54: 1116–1124; discussion 1125–1126.

# Advantage of Pedicle Screw Placement Into the Sacral Promontory (Tricortical Purchase) on Lumbosacral Fixation

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**Study Design:** Retrospective clinical study.

**Objective:** To evaluate the clinical outcome of the tricortical method for lumbosacral fixation.

**Summary of Background Data:** Despite advances in surgical techniques, failure to achieve solid arthrodesis of the lumbosacral junction continues to be significant clinical problems. To overcome these problems, tricortical purchase fixation has recently been advocated and studied. In this method, a trajectory directly into the medial sacral promontory is used to gain purchase in the dorsal, anterior, and superior cortices. This fixation method has been shown to double the insertional torque of the classic bicortical technique.

**Methods:** Patients who had undergone lumbosacral fixation were included in this study. The average area of fusion was 1.7 segments. The patients were divided into a tricortical fixation group (TF, n = 98) and a nontricortical fixation group (non-TF, n = 33). We examined clinical outcome [Japanese Orthopaedic Association scoring system (JOA score)], fusion status, and the characteristics and safety of pedicle screwing in both groups. To identify risk factors for postoperative loss of lordosis (postoperative loss of >5 degrees in L5/S1 disk angle), risk factor analysis was performed by multivariate logistic regression.

**Results:** In TF and non-TF, the JOA score changed from 13.4 and 13.8 points at surgery to 24.9 and 23.8 points, respectively, at final follow-up, and the recovery rate was 73.7% and 64.2%, respectively. Pseudoarthrosis of the fused L5/S1 occurred in 3 patients in whom the lumbosacral spine had not been fixed by tricortical purchase. The screw angle was 22.0 and 16.1 degrees in TF and non-TF, respectively, that is, a significant difference was shown. Significantly fewer TF cases encountered the risk of injured vascular tissue compared with non-TF. Non-TF (OR, 3.37) and correction of the L5/S1 disk angle (OR, 1.11) were significant risk factors for postoperative loss of lordosis.

**Conclusions:** In patients who underwent short-segment lumbosacral fusion, TF enhanced postoperative stability at the lumbosacral junction. Pseudoarthrosis did not occur in patients who underwent TF, and the risk of vascular injury was less. TF is regarded as a successful technique in short-segment lumbosacral fixation.

**Key Words:** lumbosacral spine, fixation, pedicle screw, pseudoarthrosis

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Despite advances in surgical techniques, failure to achieve solid arthrodesis of the lumbosacral junction continues to be a significant clinical problem. This is the result of various anatomic, biological, and mechanical factors relating to the sacrum, such as the fused structure of bony somites, low bone quality, a thin cortical shell, and very large, medially convergent S1 and sub-S1 pedicles.[1–3] Thus, indications of lumbosacral fixation include complications such as pseudoarthrosis, loss of lordosis, sacral fracture, and failure of instrumentation (loosening, pullout, screw breakage, implant migration, and so on).[4–8] An understanding of the sacral bony anatomy and the structures at risk is key to a well-considered sacral fixation.

The principal starting point for any sacral fixation is usually the use of S1 pedicle screws. There are 2 basic insertion techniques for S1 pedicle fixation, unicortical and bicortical. Because of the largely cancellous nature of the S1 pedicle, unicortical insertion results in poor fixation with toggling, loss of fixation, and screw pullout. Consequently, bicortical purchase has been the standard for many years. The classic trajectory is described as paralleling the S1 endplate, with appropriate medial convergence necessary to avoid the common iliac vessels located on the anterior surface of the sacrum.[9,10] However, the ideal method for fusion to the sacrum thus remains controversial. Options for posterior fixation to the sacrum, including sacral screws with extension to the ilium or combined anterior and posterior surgery, have been selected in cases of severe osteoporosis or in those requiring long fusion.

To overcome these problems, tricortical purchase fixation has recently been advocated and studied

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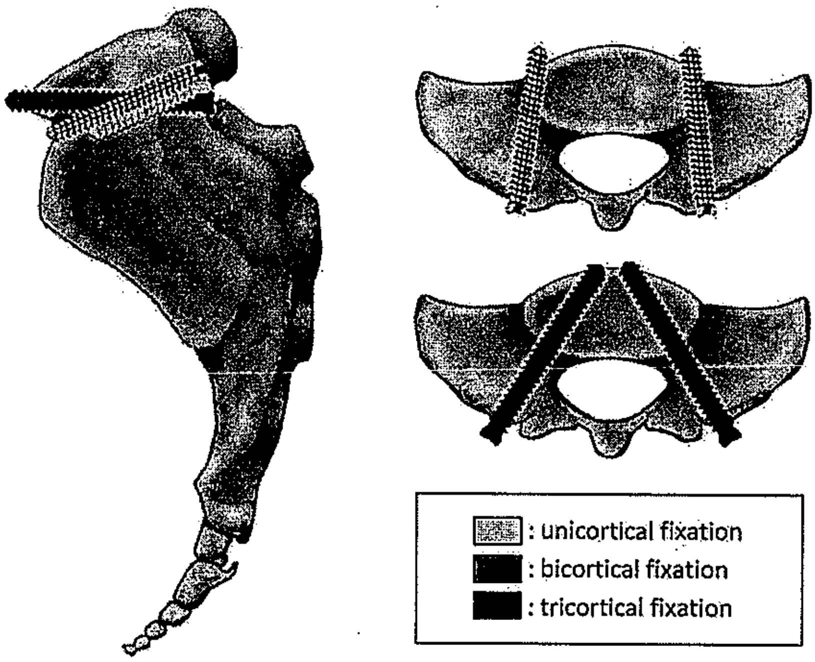


FIGURE 1. A lateral view of the sacrum demonstrates the 3 trajectories of S1 pedicle screwing including unicortical, bicortical, and tricortical fixation.

(cf. Fig. 1).[11,12] In this method, a trajectory directly into the medial sacral promontory is used to gain purchase in the dorsal, anterior, and superior cortices. This fixation method has been shown to double the insertional torque of the classic bicortical technique.[13] In addition, this trajectory is considered to introduce less risk of vascular injury compared with the trajectory of the bicortical method. Although this new fixation technique has been studied, its clinical advantages in short-segment lumbosacral fixation have not been documented. The purpose of this study was to evaluate the fixative efficacy and safety of tricortical purchase fixation in lumbosacral fixation.

## MATERIALS AND METHODS

Between 2002 and 2006, 131 patients at the Hokkaido Chuo Rosai Hospital (formerly Bibai Rosai Hospital) who had undergone lumbosacral fusion with bilateral lumbar and sacral pedicle screws systems for degenerative lumbar disease were included in this study (80 men and 51 women). The average age of subjects at the time of surgery was 56.5 years (range, 27–85 y) and the mean follow-up period was 31 months (range, 24–41 mo). The initial consecutive material comprised 155 patients, 24 of whom were lost to follow-up. These comprised 38 cases of congenital or isthmic spondylolisthesis, 37 of degenerative spondylolisthesis, 18 of lumbar canal stenosis with instability, 17 of lumbosacral foraminal stenosis, 8 of degenerative lumbar scoliosis, and 13 others (multiple operative back, and so on). The area of fusion was L3–S1 in 21 patients, L4–S1 in 57 patients, and L5–S1 in 53 patients, the average being 1.7 segments. The fusion method was either transforaminal lumbar interbody fusion (TLIF) or posterior lumbar interbody fusion (PLIF) with pedicle screws. Pedicle screw fixation using the Kaneda Anterior Posterior Spinal System was performed. In both TLIF and PLIF procedures, a carbon open box cage was placed anteriorly at L5–S1.

### Surgical Procedure

We aimed to fuse the lumbosacral joint with tricortical fixation (TF) using transparent lateral imaging. Our starting point for S1 screw insertion was the inferolateral angle of the S1 superior articular process. Orientation of the screw trajectory was directed as medially as possible to sacral midline in the horizontal plane. Under lateral fluoroscopy, we inserted into the anterior and superior cortices of the sacral promontory in the sagittal plane. When the ilium obstructed the trajectory of the pedicle screw, compromising tricortical purchase, the ilium was partially resected to facilitate implantation of the screw. Surgeons aimed to penetrate the anterior cortex of the sacrum by concomitantly probing and checking the screw position on lateral fluoroscopy. After inspecting the tunnel with a sounder, the screw was inserted with an identical trajectory.

### Evaluation

Patients were divided into 2 groups, a TF group (n = 98) and a non-TF group (n = 33). The success of TF was evaluated based on the direct trajectory of the S1 pedicle screw into the medial sacral promontory to gain purchase in the dorsal, anterior, and superior cortices using axial and sagittal plane computed tomography (CT) images. We examined the clinical outcome (fusion status) and surgical features of pedicle screwing in both groups. Clinical outcome was evaluated using the Japanese Orthopaedic Association scoring system (JOA score) and postoperative recovery rate (JOA recovery rate), which was calculated as follows: recovery rate (%) = (postoperative JOA score − preoperative JOA score) × 100/(29 − preoperative JOA score). Fusion status was evaluated on the final follow-up radiographs, including dynamic lateral flexion-extension films, and was assigned according to the Brantigan criteria[14]; that is, fusion (bone in the fusion area is radiographically more dense and more mature than originally achieved at surgery) and probable fusion (bony bridge in the fusion area is at least as dense as that originally achieved at surgery) were both considered to represent radiographically demonstrable fusion.

To assess the surgical features of pedicle screwing, the authors investigated the length, diameter, angle of the screw, and fenestration of the anterior cortex. The safety aspect of the pedicle screw's trajectory was also evaluated by verifying the relative positions of the internal iliac artery and the trajectory on postoperative CT images. The authors concluded that screwing is a high-risk technique whenever a screw and iliac artery come into contact, and that it is low risk when there is no contact (cf. Fig. 2). We compared the risk between the 2 groups.

Statistically significant differences in these studies were determined by 1-way ANOVA, Mann-Whitney $U$ test, or $\chi^2$ tests. $P$-values $< 0.05$ were considered significant.

To identify risk factors for postoperative loss of lordosis after short-segment lumbosacral fixation, risk factor analysis was performed by multivariate logistic regression with a software analysis system (JMP version 7). Postoperative loss of lordosis was defined as a postoperative loss of > 5 degrees in the L5/S1 disk angle. For statistical analysis, postoperative loss of lordosis was used as a dependent variable, along with the independent variables age, sex, number of fixed vertebrae, correction of L5/S1 disk angle, sagittal alignment of total spine, depth of L5 within the pelvis, form of promontory, average length and diameter of S1 pedicle screw, implementation of TF, and implementation of penetration of the anterior sacral cortex with the S1 pedicle screw. The quality of the fit and significance of the model were evaluated with likelihood ratio test statistics in a stepwise manner. The sagittal balance was quantified by translation of the C7 plumb line from a vertical line drawn from the posterior-superior cortex of the S1 vertebral body. The depth of L5 within the pelvis was assessed by measuring the vertical distance between the midpoint of the L5 pedicles and the interiliac crest line on the preoperative coronal standing radiograph. When the pedicles of L5 were positioned below the interiliac crest line, the depth was considered "deep" and, conversely, the measurement was "shallow" when the pedicles were above the interiliac crest line. The form of promontory was decided as follows: angles of the anterior-superior cortical surface of the S1 vertebral body < 70 and > 70 degrees were judged as sacral and lumbar type, respectively (Fig. 3).

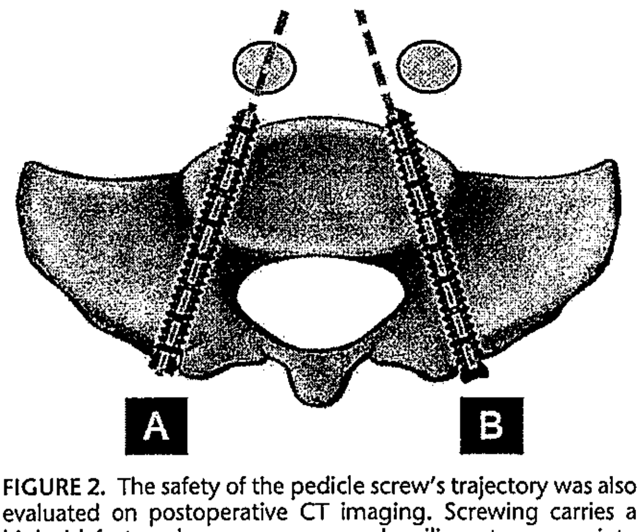


FIGURE 2. The safety of the pedicle screw's trajectory was also evaluated on postoperative CT imaging. Screwing carries a high-risk factor whenever a screw and an iliac artery come into contact (A, screw), but the risk is low when the iliac artery and screw trajectory make no contact (B, screw). Circles represent blood vessels (common iliac artery and vein).

## RESULTS

### Clinical Outcome

The demographic characteristics for patients are shown in Table 1. Among the 98 TF patients and 33 non-TF patients there were no significant differences in demographic characteristics. Over both groups, the average ratio of recovery of JOA score was 71.3%. No patients exhibited serious neurological complications or iliac vascular injury. Disturbance of adjacent segments after lumbosacral fusion necessitating reoperation occurred in 3 cases (2 TF and 1 non-TF). All 3 cases were fused at L4–S1, as the L3/4 disks were impaired. Adding on fracture was present in 1 case, and 1 patient suffered infection accompanied with meningitis 4 years after the operation.

For TF patients, the JOA score increased from 13.4 at surgery to 24.9 at final follow-up, and the recovery rate was 73.7%. For non-TF patients, the JOA score increased from 13.8 to 23.8, and the recovery rate was 64.2%. Patient recovery rate for TF was high compared with non-TF, although this difference was not significant.

Pseudoarthrosis of the fused L5/S1 occurred in 3 cases in which the lumbosacral vertebrae were not fixed by TF. These cases were fused at L3–S1, L4–S1, and L5–S1, respectively; the screws had become loosened by surrounding osteolytic change, with 1 case requiring reoperation. Pseudoarthrosis of fused vertebrae at other level (L4/5) occurred in 2 cases fixed by non-tricortical technique. The recovery rate of JOA score in these 3 cases

 

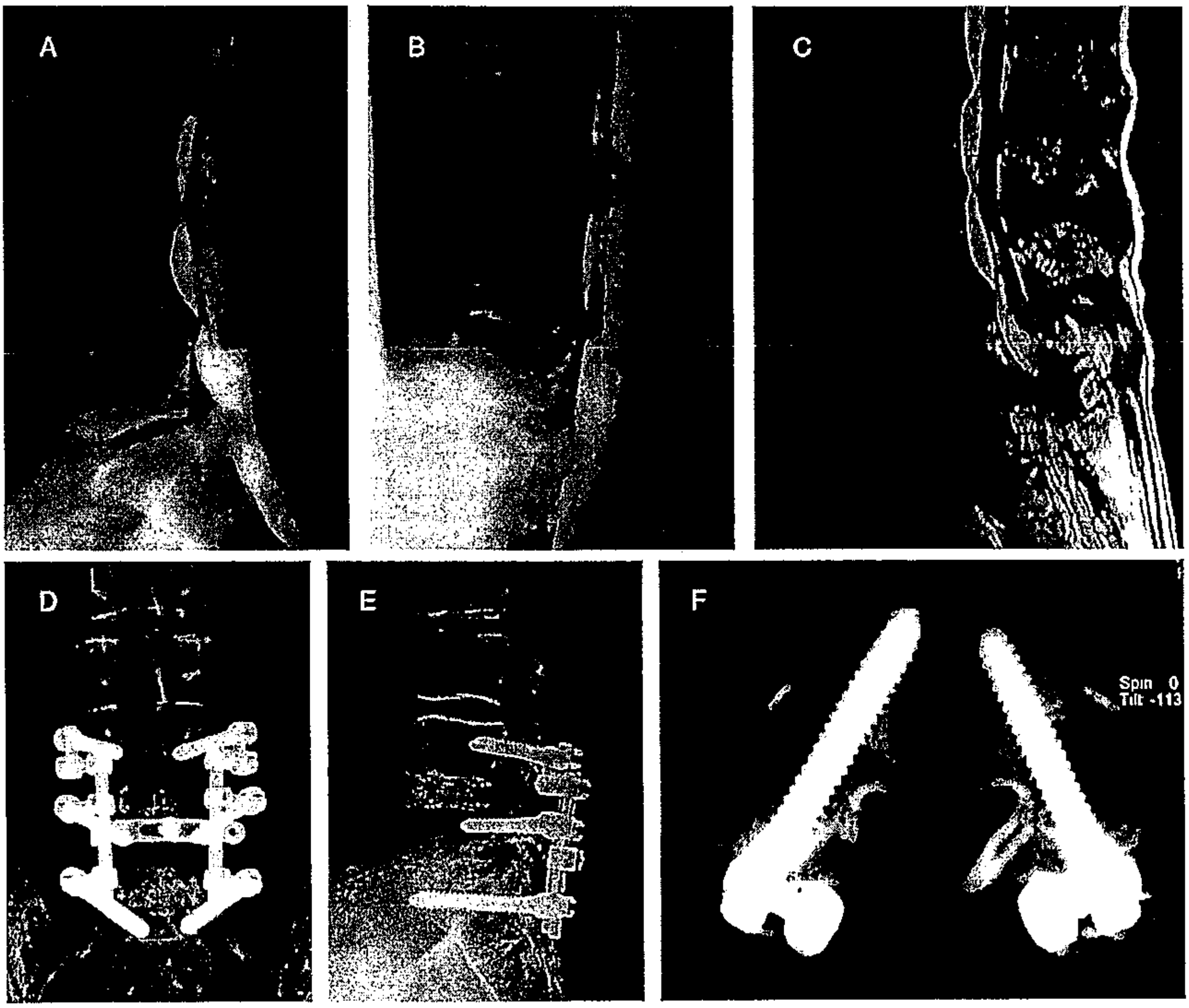


FIGURE 3. A 67-year-old woman with L4 degenerative spondylolisthesis and L5/S1 degenerative disk underwent L4–S1 transforaminal lumbar interbody fusion. Preoperatively, she had pain in the right lower limb and lower back. The lumbosacrum was fixed using the tricortical method. The JOA score was 19 preoperatively and 28 postoperatively. A and B, Preoperative myelogram (A, extension; B, flexion). C, Preoperative T2-weighted MR image (sagittal plane). D and E, Postoperative plain roentgenogram (D, AP view; E, lateral view). F, Postoperative CT image.

of pseudoarthrosis of the fused L5/S1 in non-TF patients was 31.3%, which was significantly lower than that of cases without lumbosacral nonunion.

## Characteristics and Safety of Pedicle Screwing

Screw length was 45.0 and 38.9 mm, and diameter 6.9 and 6.8 mm, in TF and non-TF, respectively, that is, there were no significant difference between groups. However, there was a significant difference in the angle of the screw (TF, 22.0 degrees; non-TF, 16.1 degrees). To achieve tricortical screwing, the surgeons performed partial resection of the ilium in 29 TF cases (29.6% of all TF cases), and this was considered to have resulted in securing a deeper angle for implant trajectory. Evaluation of the penetration of the anterior cortex of the sacrum by the S1 screw revealed that the right side was penetrated in 80 TF and 29 non-TF cases, the left side in 81 TF and 30 non-TF cases, and bilaterally in 77 TF and 29 non-TF cases. The prevalence of penetration through the anterior sacral cortex with the S1 pedicle screw was lower in TF than in non-TF (78% and 87%, respectively). Table 2 summarizes the characteristics of pedicle screwing.

In TF, the trajectory of the S1 pedicle screw put 26 cases at risk of vascular injury (26.5%), whereas 15 non-TF cases (45.4%) were at risk. Significantly fewer TF cases encountered the risk of injured vascular tissue compared with non-TF (Table 3).

## Loss of Lordosis

Postoperative loss of lordosis was present in 23 patients, with an incidence of 19%. Factors examined in stepwise analysis were age, sex, implementation of TF,

 

TABLE 1. Demographic Characteristics

| Characteristic | Group TF (98 cases) | Group Non-TF (33 cases) |
|---|---|---|
| Age (y) | 56.1 (15.6) | 57.7 (13.4) |
| Sex (male/female) | 60/38 | 20/13 |
| Preoperative JOA score (points) | 13.4 (4.2) | 13.8 (4.1) |
| Time of operation (min) | 289 (90.1) | 305 (106) |
| Blood loss during operation (mL) | 868 (491) | 884 (467) |
| Follow-up period (mo) | 30 (8.5) | 34 (9.9) |
| No. fused segment | 1.7 (0.95) | 1.7 (0.89) |
| Sagittal balance (mm) | 45.9 (41.1) | 39.6 (44.4) |
| Depth of L5 within pelvis (case) | | |
| Fair | 62 | 19* |
| Deep | 25 | 12 |
| Shallow | 11 | 2 |
| Form of promontory (case) | | |
| Sacral type | 87 | 17 |
| Lumbar type | 11 | 16* |

Values are mean (SD).
*Significant difference in comparison with group TF.
TF indicates tricortical fixation.

sagittal alignment of total spine, correction of L5/S1 disk angle, and depth of L5 within the pelvis. The implant angle, length, and diameter of the S1 pedicle screw, and achieving anterior sacral cortical penetration by the S1 pedicle screw in TF were not significant risk factors. Table 4 shows the adjusted effect estimates [odds ratio (OR)] for the final predictive model as determined by multivariate logistic analysis. Nonimplementation of TF (OR, 3.37) and correction of the L5/S1 disk angle (OR, 1.11) were significant risk factors for postoperative loss of lordosis. These findings demonstrate that the implementation of TF decreases the occurrence of postoperative loss of lordosis.

## DISCUSSION

As lumbosacral fixation techniques and their related theories have continued to develop, several studies have reported various instrumentation strategies for lumbosacral fixation and the use of anterior grafts, anterior instrumentation, supplementary points of sacral fixation, or iliac extension to improve stabilization of the lumbosacral junction. However, increasing the level of internal fixation may increase implant-related complications. Methods to expand an anchor to the ilium feature longer lever arms but are associated with the problems of iliac implant breakage or sacroiliac joint arthritis and pain.[15] Bulky implants are associated with prominent and painful hardware.[16,17] Placement of an intrasacral rod through the S1 screw into the ala of the sacrum, as reported by Jackson, is a complicated operative technique.[18] To achieve solid arthrodesis in long fusion to the sacrum, such as that required for the treatment of neuromuscular scoliosis and severe degenerative scoliosis and in patients with severe osteoporosis, fixation to expand an anchor to the ilium may be needed. However, an S1 pedicle screw system alone is ideal for short-segment fixation of the lumbosacral spine.

The present study revealed that TF effectively enhanced the postoperative stability of lumbosacral reconstruction in patients with short-segment fusion. In this method, which does not require a complex surgical technique, the lumbosacral spine is fixed by an independent sacral anchor. This fixation technique may be useful as an alternative to the standard bicortical approach. One principal reason for the biomechanical superiority of S1 pedicle screw fixation, using the tricortical technique, over unicortical or bicortical fixation is that the bone mineral density (BMD) of the sacral promontory is higher than that of other S1 body layers and the bone in the lateral sacral alar region.[19] It has been reported that BMD is a useful preoperative predictor of sacral screw fixation strength. With increase in BMD comes an increase in screw insertion torque and potentially, a subsequent increase in screw pullout strength after cyclic loading.[20–22] Lehman et al[13] reported that TF doubled the insertional torque of the classic bicortical technique. In this study, pseudoarthrosis of the fused L5/S1 did not occur in cases of lumbosacral vertebrae with TF, and we assume it did not occur due to strong fixation of the lumbosacral spine. Furthermore, in cases of the lumbosacral joint being fused by the tricortical technique, postoperative lumbar lordosis was maintained. Many studies have shown the negative effects of reduced lumbar lordosis with a fixed spine after spinal instrumentation.[23–25] Optimal lumbar lordosis prevents the anticipated progression of degenerative change that follows lumbar spine-instrumented fusion. Pseudoarthrosis

TABLE 2. Characteristics of Pedicle Screwing

| Characteristic | Group TF | | Group Non-TF | |
|---|---|---|---|---|
| | Right | Left | Right | Left |
| Screw length (mm) | 45 (4.8) | 45 (4.5) | 38.5 (4.3) | 39 (5.3) |
| Screw diameter (mm) | 7.0 (0.2) | 6.9 (0.2) | 6.8 (0.2) | 6.8 (0.2) |
| Angle of screw (deg.) | 22.2 (4.7) | 21.8 (5.2) | 16.1 (3.8)* | 16 (2.4)* |
| Fenestration of anterior cortex (case) | 80 | 81 | 29 | 30 |

Values are mean (SD).
*Significant difference in comparison with group TF.
TF indicates tricortical fixation.

TABLE 3. Risk of Vascular Injury*

| Risk | Group TF | Group Non-TF |
|---|---|---|
| Risk (+) | 26 | 15 |
| Risk (−) | 72 | 18 |

Values are case.
*Significant difference by the $\chi^2$ tests.
TF indicates tricortical fixation.

of the fused L4/5 occurred in 2 cases wherein the lumbosacral vertebrae were not fused by TF. This occurrence might be attributable to nonstrong fixation and the loss of fused lumbar lordosis, both of which can result in the screw loosening. TF can reduce the pseudoarthrosis of the fused spine and the postoperative lumbar lordosis that could induce poor clinical outcome, so TF is regarded as a successful technique for lumbosacral fixation.

The structures that surgeons usually put at risk while implanting S1 pedicle screws include the common iliac artery and vein, the L5 nerve root, the colon, and the sympathetic chain.[10,26,27] The classic trajectory is described as paralleling the S1 endplate with the appropriate medial convergence necessary to avoid the common iliac vessels located on the anterior surface of the sacrum. Several studies have suggested that anteromedial screw placement also provides a larger "safe zone" for avoiding neurological injury.[1,28] Furthermore, while implanting bicortical S1 pedicle screws, the surgeon must penetrate the sacral anterior cortex to obtain sufficient biomechanical strength, yet this procedure of penetration might enhance the risk that the screws will cause neurovascular injuries. Therefore, we wanted to confirm that the trajectory of tricortical screwing is more convergent than that of classic screwing as well as to examine whether penetrating the sacral anterior cortical bone is necessary for implanting tricortical S1 screws. The present study showed that the screw trajectory in TF is more convergent and incurs a lower risk of vascular injury. In addition, the strength of fixation obtained by the tricortical technique, which provides sufficient purchase in the hard sacral promontory, is such that it is not always necessary for the screw to penetrate the anterior cortex. In contrast, the bicortical method requires that the screw penetrate the anterior cortex, thus increasing the risk of vascular injury.

TABLE 4. Risk Factors for Postoperative Loss of Lordosis

| Variables | OR (95% CI) | P |
|---|---|---|
| Age | 1.02 (0.56–4.2) | 0.22 |
| Men (vs. women) | 0.97 (0.93–1.01) | 0.39 |
| Non-TF (vs. TF) | 3.37 (1.24–9.26) | 0.016* |
| Sagittal balance | 1.05 (0.99–1.01) | 0.41 |
| Correction of L5/S1 disk angle | 1.11 (1.03–1.2) | 0.033* |
| Deep-seated L5 | 0.6 (0.24–1.42) | 0.252 |

*Significant difference.
CI indicates confidence interval; OR, adjusted odds ratio; TF, tricortical fixation.

The tricortical method is therefore beneficial by reducing the risk of neurovascular injury.

Compared with conventional fixation (unicortical and bicortical), tricortical purchase requires accurate targeting of the apex with the exact length of screw, which is time consuming and technically difficult. Use of transparent lateral imaging and partial resection of the ilium to obtain a proper implant angle for the screw are keys to success. Kaptanoglu et al[29] suggested that the level of iliac resection should be decided preoperatively by evaluating CT scans acquired parallel to the trajectory of the S1 screws. In this study, 98 patients (74.8%) were fused by TF, of which approximately one third required partial resection of the ilium for an appropriate implant angle to be achieved. Ota et al[30] reported that the paraspinal approach can lead to easier placement of medially oriented screws. TF may be regarded as an implant method that is not technically difficult using the above techniques.

There are some limitations to this study: the first and major being that it was retrospective. This study was not a precise comparative study and may include bias. A prospective comparative study would be necessary to demonstrate the efficacy of TF. The present study demonstrated that TF effectively enhances the postoperative stability of lumbosacral reconstruction in patients with relatively short-segment fusion. Failure to achieve solid arthrodesis in long fusion, including the lumbosacral junction, continues to be a significant clinical problem. Further research is needed to determine the effectiveness of a combination of TF and other spinopelvic fixation (ie, Jackson technique or iliac screw fixation) for long fusions to the sacrum, such as those required for treatment of neuromuscular scoliosis and severe degenerative scoliosis and in patients with severe osteoporosis.

## CONCLUSIONS

TF enhanced postoperative stability at the lumbosacral junction in patients who underwent short-segment lumbosacral fusion. This study demonstrated that pseudoarthrosis did not occur in patients after TF, in comparison with those undergoing non-TF; tricortical screwing carries a lower risk of vascular injury. TF is regarded as a successful technique in lumbosacral fixation.

## REFERENCES

1. Mirkovic S, Abitbol JJ, Steinman J, et al. Anatomic consideration for sacral screw placement. *Spine*. 1991;16:S289–S294.
2. Smith SA, Abitbol JJ, Carlson GD, et al. The effects of depth of penetration, screw orientation, and bone density on sacral screw fixation. *Spine*. 1993;18:1006–1010.
3. Xu R, Ebraheim NA, Yeasting RA, et al. Morphometric evaluation of the first sacral vertebra and the projection of its pedicle on the posterior aspect of the sacrum. *Spine*. 1995;20:936–940.
4. Molinari RW, Bridwell KH, Lenke LG, et al. Complications in the surgical treatment of pediatric high-grade, isthmic dysplastic spondylolisthesis. A comparison of three surgical approaches. *Spine*. 1999;24:1701–1711.
5. Pihlajamaki H, Bostman O, Ruuskanen M, et al. Posterolateral lumbosacral fusion with transpedicular fixation: 63 consecutive cases followed for 4 (2-6) years. *Acta Orthop Scand*. 1996;67:63–68.

6. Pihlajamaki H, Myllynen P, Bostman O. Complications of transpedicular lumbosacral fixation for non-traumatic disorders. *J Bone Joint Surg Br*. 1997;79:183–189.
7. Rechtine GR, Sutterlin CE, Wood GW, et al. The efficacy of pedicle screw/plate fixation on lumbar/lumbosacral autogenous bone graft fusion in adult patients with degenerative spondylolisthesis. *J Spinal Disord*. 1996;9:382–391.
8. Wood KB, Geissele AE, Ogilvie JW. Pelvic fractures after long lumbosacral spine fusions. *Spine*. 1996;21:1357–1362.
9. Carlson GD, Abitbol JJ, Anderson DR, et al. Screw fixation in the human sacrum. An in vitro study of the biomechanics of fixation. *Spine*. 1992;17:S196–S203.
10. Esses SI, Botsford DJ, Huler RJ, et al. Surgical anatomy of the sacrum. A guide for rational screw fixation. *Spine*. 1991;16:S283–S288.
11. Linville DA, Dmitriev AE. Lumbosacral fixation: an update. *Curr Opin Orthop*. 2005;16:137–143.
12. Polly DW, Latta LL. Spinopelvic fixation biomechanics. In: *Semin Spine Surg*. Elsevier; 2004:101–106.
13. Lehman RA Jr, Kuklo TR, Belmont PJ Jr, et al. Advantage of pedicle screw fixation directed into the apex of the sacral promontory over bicortical fixation: a biomechanical analysis. *Spine*. 2002;27:806–811.
14. Brantigan JW, Steffee AD. A carbon fiber implant to aid interbody lumbar fusion. Two-year clinical results in the first 26 patients. *Spine*. 1993;18:2106–2107.
15. Devlin VJ, Boachie-Adjei O, Bradford DS, et al. Treatment of adult spinal deformity with fusion to the sacrum using CD instrumentation. *J Spinal Disord*. 1991;4:1–14.
16. Emami A, Deviren V, Berven S, et al. Outcome and complications of long fusions to the sacrum in adult spine deformity: luque-galveston, combined iliac and sacral screws, and sacral fixation. *Spine*. 2002;27:776–786.
17. Tsuchiya K, Bridwell KH, Kuklo TR, et al. Minimum 5-year analysis of L5-S1 fusion using sacropelvic fixation (bilateral S1 and iliac screws) for spinal deformity. *Spine*. 2006;31:303–308.
18. Jackson RP, McManus AC. The iliac buttress. A computed tomographic study of sacral anatomy. *Spine*. 1993;18:1318–1328.
19. Zheng Y, Lu WW, Zhu Q, et al. Variation in bone mineral density of the sacrum in young adults and its significance for sacral fixation. *Spine*. 2000;25:353–357.
20. Hadjipavlou AG, Nicodemus CL, al-Hamdan FA, et al. Correlation of bone equivalent mineral density to pull-out resistance of triangulated pedicle screw construct. *J Spinal Disord*. 1997;10:12–19.
21. Myers BS, Belmont PJ Jr, Richardson WJ, et al. The role of imaging and in situ biomechanical testing in assessing pedicle screw pull-out strength. *Spine*. 1996;21:1962–1968.
22. Zhu Q, Lu WW, Holmes AD, et al. The effects of cyclic loading on pull-out strength of sacral screw fixation: an in vitro biomechanical study. *Spine*. 2000;25:1065–1069.
23. Kumar MN, Baklanov A, Chopin D. Correlation between sagittal plane changes and adjacent segment degeneration following lumbar spine fusion. *Eur Spine J*. 2001;10:314–319.
24. Lazennec JY, Ramare S, Arafati N, et al. Sagittal alignment in lumbosacral fusion: relations between radiological parameters and pain. *Eur Spine J*. 2000;9:47–55.
25. Korovessis P, Stamatakis M, Baikousis A. Segmental roentgenographic analysis of vertebral inclination on sagittal plane in asymptomatic versus chronic low back pain patients. *J Spinal Disord*. 1999;12:131–137.
26. Licht NJ, Rowe DE, Ross LM. Pitfalls of pedicle screw fixation in the sacrum. A cadaver model. *Spine*. 1992;17:892–896.
27. Sabatier JP, Guaydier-Souquieres G. Noninvasive methods of bone-mass measurement. *Clin Rheumatol*. 1989;(suppl 2):41–45.
28. Krag MH, Beynnon BD, Pope MH, et al. An internal fixator for posterior application to short segments of the thoracic, lumbar, or lumbosacral spine. Design and testing. *Clin Orthop Relat Res*. 1986;203:75–98.
29. Kaptanoglu E, Okutan O, Tekdemir I, et al. Closed posterior superior iliac spine impeding pediculocorporeal S-1 screw insertion. *J Neurosurg*. 2003;99:229–234.
30. Ota M, Neo M, Fujibayashi S, et al. Advantages of the paraspinal muscle splitting approach in comparison with conventional midline approach for s1 pedicle screw placement. *Spine*. 2010;35:E452–E457.

Spine

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CERVICAL SPINE

# Cervical Disc Protrusion Correlates With the Severity of Cervical Disc Degeneration

## *A Cross-Sectional Study of 1211 Relatively Healthy Volunteers*

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**Study Design.** Cross-sectional study.

**Objective.** The purposes of this study were (1) to investigate the frequency and degree of cervical disc degeneration and protrusion on cervical spine magnetic resonance (MR) images and (2) to analyze the correlation between the severity of disc degeneration and disc protrusion.

**Summary of Background Data.** Cervical disc degenerative changes or protrusion is commonly observed on MR images in healthy subjects. However, there are few large-scale studies, and the frequency and range of these findings in healthy subjects have not been clarified. Moreover, there are no reports regarding the correlation between cervical disc degeneration and disc protrusion.

**Methods.** Cervical disc degeneration and protrusion were prospectively measured using magnetic resonance imaging in 1211 relatively healthy volunteers. These included at least 100 males and 100 females in each decade of life between the 20s and the 70s. Cervical disc degeneration was defined according to the modified Pfirrmann classification system, and the amount of disc protrusion was evaluated using the anteroposterior diameter of disc protrusion on sagittal MR image.

**Results.** Mild disc degeneration was very common, including 98.0% of both sexes in their 20s. The severity of cervical disc degeneration significantly increased with age in both sexes at every level. The disc degeneration predominantly occurred at C5–C6 and C6–C7. The difference between sexes was not significant except for individuals in their 50s. The average anteroposterior diameter of disc protrusion increased with aging, especially from the 20s to the 40s. The anteroposterior diameter of disc protrusion increased with a progression in the disc degeneration grade.

**Conclusion.** Cervical disc degeneration and protrusion were frequently observed in healthy subjects even in their 20s and deteriorated with age. Cervical disc protrusion was significantly correlated with cervical disc degeneration, and spatial cervical disc protrusion was affected by biochemical degenerative changes as observed on MR images.

**Key words:** cervical, disc degeneration, disc protrusion, asymptomatic, magnetic resonance images, cross-sectional study, aging.

**Level of Evidence:** 2

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The human intervertebral disc forms a fibrocartilaginous joint between the vertebrae. The intervertebral disc consists of 3 distinct components, including the nucleus pulposus, the annulus fibrosis, and the cartilaginous endplates.[1] Progressive morphologic and cellular changes occur with age and degeneration in intervertebral discs.[2,3] Biochemically, proteoglycan fragmentation increases with aging, and the overall proteoglycan and water content of the disc decreases, especially in the nucleus.[2,3]

Magnetic resonance imaging (MRI) provides a noninvasive morphologic evaluation of the cervical spine. The signal intensity of the disc on magnetic resonance (MR) image is indicative of the chemical composition and likely histological changes.[4,5] The reduction of signal intensity on T2-weighted MR image correlates with progressive degenerative changes of the intervertebral disc.[5] The brightness of the nucleus correlates particularly well with the proteoglycan concentration of the disc.[4] Although these degenerative changes in the cervical disc are often seen on MR image in healthy subjects,[6,7] some of them could be pathological, leading to associated clinical symptoms. It is difficult to distinguish between aging discs and pathologically degenerated discs that lead to clinical symptoms. Thus, it is crucial to analyze the frequency and range of degenerative changes observed on MR images in healthy subjects.

As well as signal intensity changes in the disc, disc protrusion is also frequently observed with aging. "Protrusion" is commonly used in a nonspecific general sense to signify any displacement of the disc.[8,9] A disc herniation, on the contrary, results when a crack in the outer layer of cartilage allows some of the inner fragmented annulus, nucleus, cartilage, and fragmented apophyseal bone to protrude out of the disc.[8–10] Disc herniation is usually a further development of a previously existing disc protrusion.[11]

Cervical compressive myelopathy is one of the most common causes of neurological symptoms associated with the cervical spine.[12] Disc protrusion has been suspected to initiate cervical spinal cord compression.[7] However, to our knowledge, there are no data about whether this spatial disc protrusion has any correlation with biochemical disc degeneration (DD).

The primary aim of this study was to prospectively investigate the frequency and range of cervical DD and protrusion observed on cervical spine MR image in a large cohort of relatively healthy volunteers. We also analyzed the correlation between the severity of DD and the spatial changes associated with disc protrusion and then evaluated the influence of DD on disc protrusion.

## MATERIALS AND METHODS

A total of 1230 relatively healthy volunteers were examined by cervical spine MRI between February 2006 and February 2008. The subjects were relatively healthy volunteers without neurological symptoms, and the subjects recruited were approximately 100 males and 100 females in each decade of life between 20 and 79 years of age (Table 1).[13] We recruited the subjects by using newspaper advertisements and posters in facilities having some sort of relationship with our hospital. Thus, the majority of subjects were not patients at our hospital but relatively healthy residents of the area. The exclusion criteria included a history of brain or spinal surgery, comorbid neurological disease (*e.g.*, cerebral infarction or neuropathy), symptoms related to sensory or motor disorders (numbness, clumsiness, motor weakness, or gait disturbances), or severe neck pain. We excluded pregnant females, individuals who received workmen's compensation, and those who presented with the symptoms after a motor vehicle accident. Subjects with other comorbidities (smoking, diabetes, hypertension, and others) were included in this study. The institutional review board approved this study, and each patient signed a written consent form.

All participants underwent imaging analysis by 2 spinal surgeons (F.K. and K.S.). Finally, The MRI data from 1211 subjects were included in this analysis, after excluding 19 subjects with measurement difficulties resulting from artifacts, such as motion artifacts or the presence of metals. The MR images were obtained with a 1.5-Tesla superconductive magnet (Signa Horizon Excite HD version 12; GE Healthcare, Britain, United Kingdom). The images were obtained at a slice thickness of 3 mm in the sagittal plane. T2-weighted images (fast spin echo TR, 3500 ms; TE, 102 ms) were obtained in sagittal images. All images were transferred to a computer as Digital Imaging and Communications in Medicine data to measure the anteroposterior (AP) diameter of disc protrusion using imaging software (Osiris 4; Icestar Media Ltd, Essex, United Kingdom). We used a slice in which the disc protrusion was most prominent in the sagittal images and measured the AP diameter of disc protrusion. The posterior aspect line between the rostral and caudal vertebral body was used as the standard, and we measured the AP diameter of the disc protrusion from the standard line to the posterior top of the disc protrusion (Figure 1). Cervical DD was defined according to the modified Pfirrmann classification system (Figure 2).[4] To facilitate comparison of the severity of DD between decades or sexes, a total score of DD (DD score) at the 6 levels (from C2–C3 to C7–T1) was calculated by the summation of individual Pfirrmann scores at each level.

### Statistical Analysis

Fisher exact test was used to compare the differences between decades and sexes in DD scores. The Jonckheere-Terpstra trend test was used to assess whether the AP diameter of disc protrusion increased depending on DD severity and is indicated by a letter "trend," followed by a *P* value. A *P* value of less than 0.05 was considered statistically significant. All analyses were conducted using SPSS version 21 (SPSS, Chicago, IL).

## RESULTS

The severity of cervical DD increased with age at every level in both sexes (Figure 3). Mild DD was observed in the 20s (98% in both sexes). Although grade VI DD was not identified in the 20s and the 30s, it appeared after the 40s and then increased further with age. The C5–C6 and C6–C7 discs were the first and second most severely degenerated discs in every decade.

TABLE 1. Age and Sex of 1211 Asymptomatic Subjects

| Age (yr) | 20–29 | 30–39 | 40–49 | 50–59 | 60–69 | 70–79 | Total |
|---|---|---|---|---|---|---|---|
| Male | 101 | 104 | 100 | 99 | 101 | 101 | 606 |
| Female | 100 | 99 | 100 | 103 | 103 | 100 | 605 |

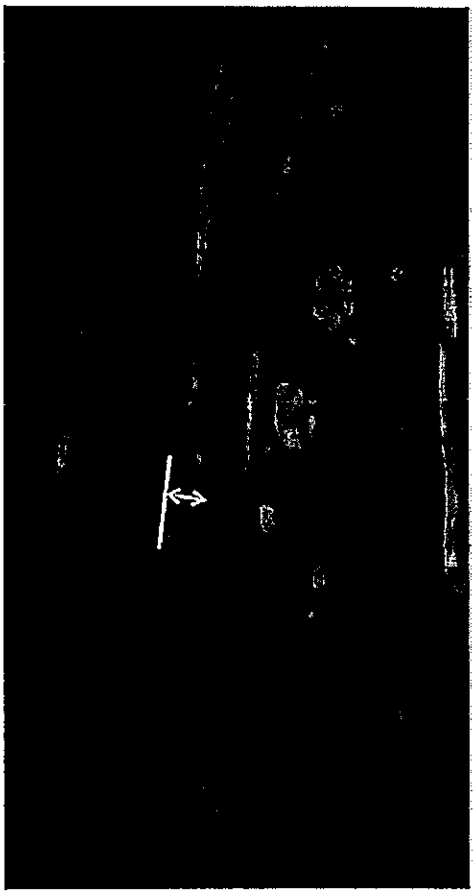

Figure 1. The measurement method for the anteroposterior diameter of the disc protrusion on magnetic resonance image. The posterior aspect line between the rostral and caudal vertebral body was used as the standard, and we measured the anteroposterior diameter of the disc protrusion from the standard line to the posterior top of the disc protrusion (white double arrow).

The DD score increased with age in both sexes (Figure 4). A DD score of 6 (the minimal score) was found only in the 20s. A DD score of 13 or more was found in more than half of the cases belonging to the 50s or older age groups. The total score was 13 or more in more than 95% of the cases in the 70s. The number of DD scores of 13 or more significantly increased with aging ($P < 0.05$ to $P < 0.001$), and the amount of the increase was very significant from the 20s to the 50s ($P < 0.005$). The difference between sexes was significant only in the 50s ($P = 0.03$), in which a DD score of 13 or more was more frequent in males.

The average AP diameter of disc protrusion increased with aging, especially from the 20s to the 40s (Figure 5). The disc protrusion at C5–C6 was most prominent, and the disc protrusion at C6–C7 was second most prominent in all decades and both sexes. On the contrary, the disc at C2–C3 protruded the least.

The relationship between the severity of DD and disc protrusion is shown in Figure 6. The AP diameter of cervical disc protrusion increased with a progression in the DD grade ($P < 0.05$ to $P < 0.001$).

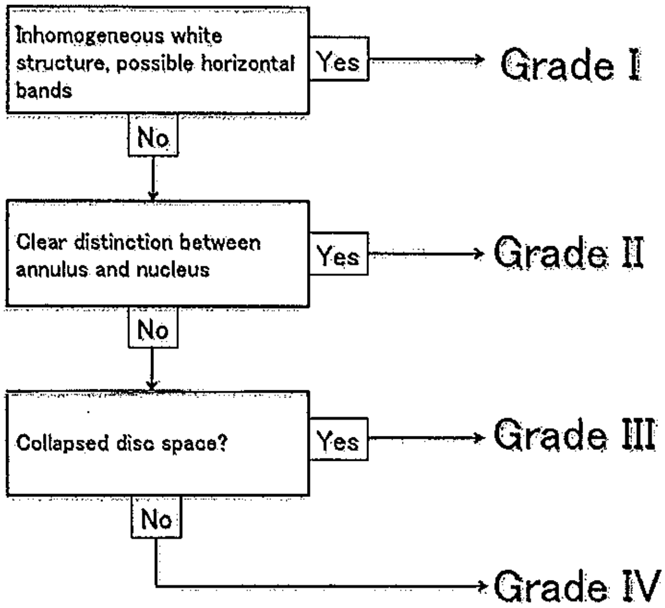


Figure 2. Algorithm for the grading system and assessment of cervical disc degeneration grade using the modified Pfirrmann classification system.

## DISCUSSION

This largest cross-sectional study of cervical MR images in relatively healthy subjects showed that (1) mild cervical DD and protrusion started in the 20s, (2) cervical DD and protrusion progressed with age, especially from the 20s to the 50s, mainly at C5–C6 and C6–C7, and (3) the degree of cervical disc protrusion was significantly correlated with the severity of DD.

Cervical DD and protrusion are some of the abnormal findings most commonly seen on MR images in relatively healthy subjects, and they are frequently observed in subjects aged 40 to 50 years.[6,7] These degenerative changes, which occur throughout life, have been biochemically analyzed.[2,3] Proteoglycan fragmentation starts during childhood, and the overall proteoglycan and water content of the disc decreases with increasing age, especially in the nucleus.[2,3] These changes are enhanced by adverse genetic, biomechanical, and nutritional factors.[3]

The reduction of signal intensity on T2-weighted MR image correlates with the progressive degenerative changes of the intervertebral discs.[5] The brightness of the nucleus correlates particularly well with the proteoglycan concentration.[4] These indicators of DD on MR image are observed even in a younger population; Matsumoto *et al*[7] reported 17% and 12% prevalence in males and females in their 20s, respectively, and Boden *et al*[6] reported 8% in patients younger than 40 years. In the present largest cohort study, mild DD of at least 1 level was observed in 98% of both sexes in their 20s. DD was identified more frequently in subjects in their 20s than we previously expected, although evaluation methods of DD were different across studies. Several reports mentioned that lumbar discs in males were more degenerated than those in females,[14–16] but there were few differences in

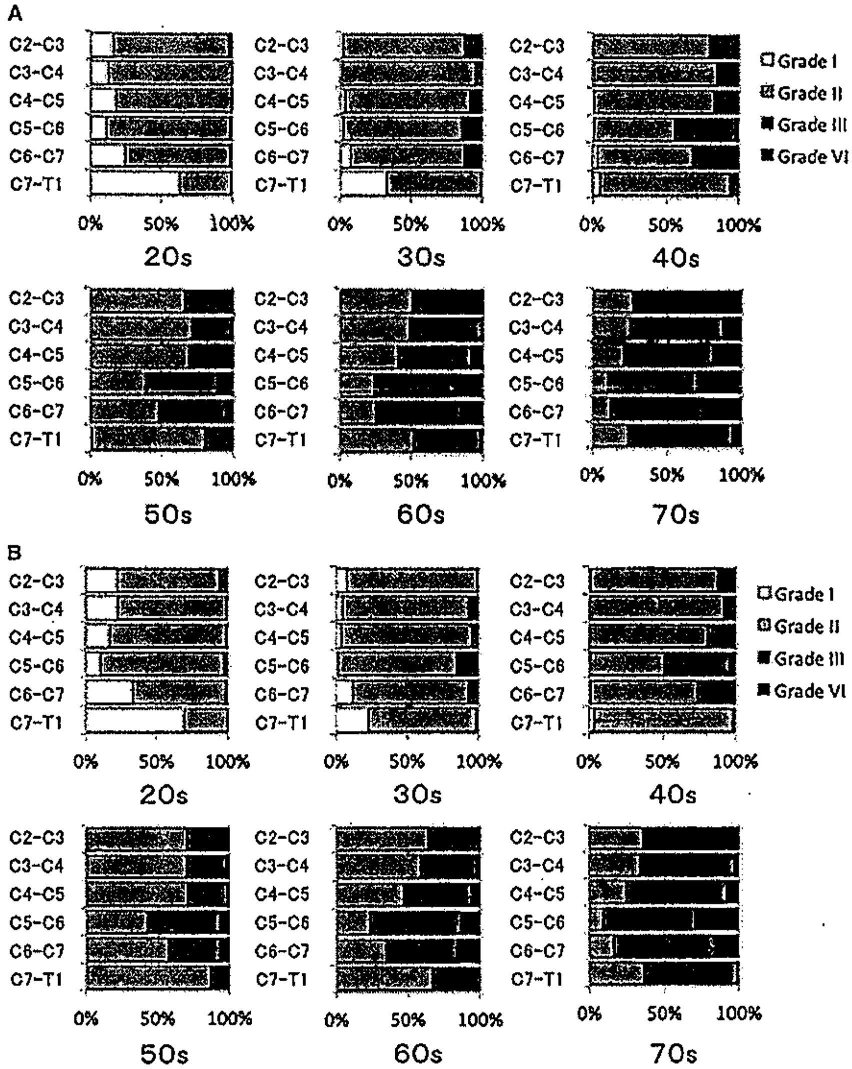


Figure 3. Cervical disc degeneration using the modified Pfirrmann classification system in each decade in (A) males and (B) females.

cervical discs. We think that the differences between lumbar and cervical disc changes are due to sex-related differences in the loading of the discs based on weight or profession. Further studies are needed to elucidate the different prevalence of DD dependent on sex between the cervical and lumbar spine.

Disc protrusion starts from an annulus tear, and this tear allows gross migration of the nucleus.[2] Disc protrusion has been simulated in cadaveric discs, and mechanically induced prolapse occurs most in discs from patients aged 30 to 40 years.[17] It is difficult to induce disc protrusion in severely degenerated discs in the laboratory. It has been

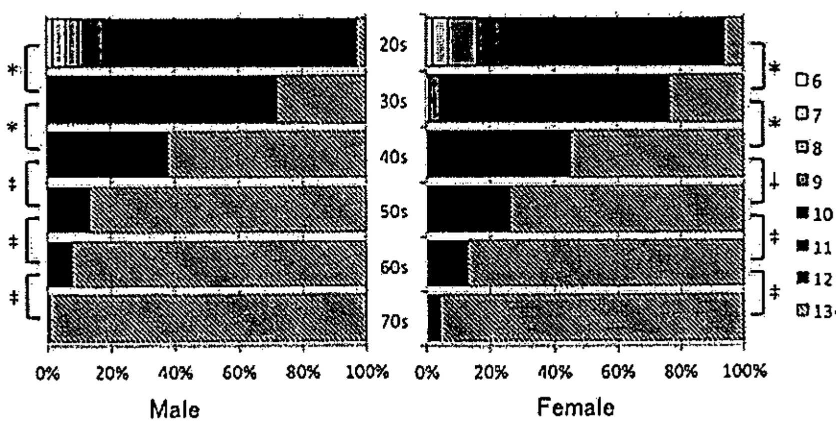


Figure 4. Disc degeneration scores in each decade and sex. *$P < 0.05$; †$P < 0.005$; ‡$P < 0.001$.

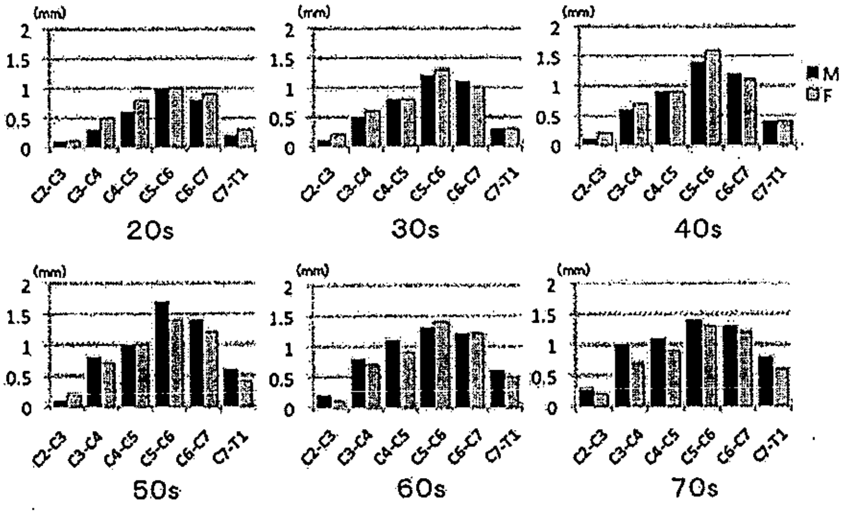


Figure 5. Mean anteroposterior diameter of cervical disc protrusion in each sex. M indicates male; F, female.

suggested that this is because the nucleus is no longer able to exert hydrostatic pressure on the annulus.[2,17] However, the amount of disc protrusion was significantly larger in the severely degenerated discs in the current study. Proteoglycan works as a resistance against compression forces.[3] Discs might protrude further if relatively large vertical forces were loaded onto severely degenerated discs. Thus, cervical disc protrusion was highly correlated with biochemical degeneration in the disc.

There were some limitations to the present study. First, the survey was limited to the Japanese population, so racial differences were not taken into account. Second, only relatively healthy subjects were enrolled in this study. This could have led to a selection bias in favor of relatively healthy participants. Third, we compared different individuals between ages because this study was cross-sectional rather than longitudinal. Fourth, although the subjects were all relatively healthy, some had pathological backgrounds or occasional mild neck pain, and these pathological or mild symptomatic backgrounds could affect the results. Fifth, the MR images were not evaluated by radiologists but spine surgeons. This might have affected the sensitivity and specificity of the radiographical evaluation. Sixth, the AP diameter of the disc protrusion was measured in sagittal images to reduce interobserver measurement error. This was because, in the axial images, the location of the anterior standard point from which the AP diameter was measured differed from observer to observer. Care must be taken when the measurement data are used in future because the AP diameters measured in axial and sagittal images are different.

In conclusion, this large-scale cross-sectional analysis of cervical spine MRI data in healthy subjects demonstrates that cervical DD and protrusion occur more frequently at an earlier age than what was previously reported. Spatial disc protrusion is highly correlated with the progression of DD. By comparing the DD and protrusion data from the relatively healthy subjects in this study with that of symptomatic patients in future, we think that the current data in relatively healthy volunteers could be helpful for determining the DD pattern resulting in symptoms.

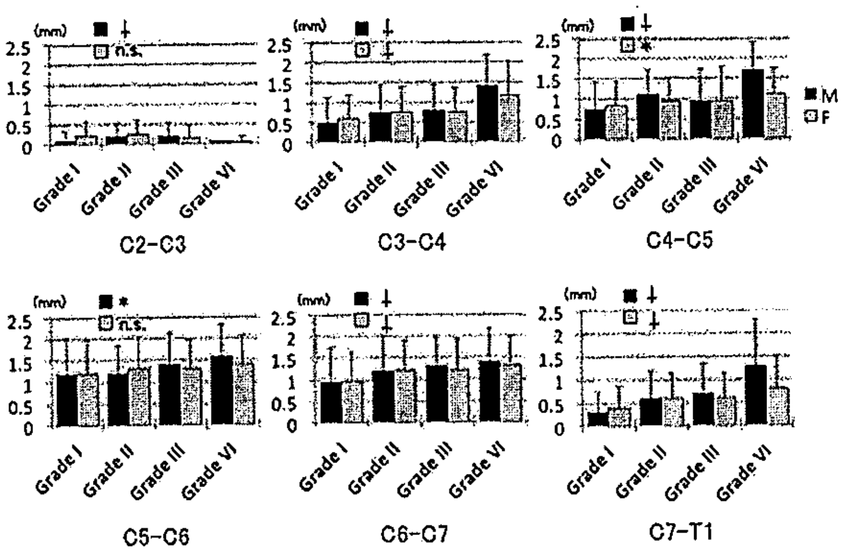


Figure 6. Mean anteroposterior diameter of cervical disc protrusion in each cervical disc degeneration grade at each level. Values are mean ± SD. *$P < 0.05$; †$P < 0.001$. M indicates male; F, female; ns, not significant.

## ➢ Key Points

- ❑ Cervical disc degeneration and protrusion were evaluated on cervical MR images of 1211 relatively healthy volunteers.
- ❑ Mild cervical disc degeneration of at least 1 level was observed in 98% of healthy subjects in their 20s, and it progressed with aging.
- ❑ The average amount of cervical disc protrusion increased with aging, especially from the 20s to the 40s.
- ❑ Both cervical disc degeneration and protrusion were predominantly observed at the C5–C6 and C6–C7 levels.
- ❑ The degree of cervical disc protrusion was significantly correlated with the severity of disc degeneration as observed on MR image.

## References

1. Cheung KM, Karppinen J, Chan D, et al. Prevalence and pattern of lumbar magnetic resonance imaging changes in a population study of one thousand forty-three individuals. *Spine* 2009;34:934–40.
2. Adams MA, Roughley PJ. What is intervertebral disc degeneration, and what causes it? *Spine* 2006;31:2151–61.
3. Roughley PJ. Biology of intervertebral disc aging and degeneration: involvement of the extracellular matrix. *Spine* 2004;29:2691–9.
4. Pfirrmann CW, Metzdorf A, Zanetti M, et al. Magnetic resonance classification of lumbar intervertebral disc degeneration. *Spine (Phila Pa 1976)* 2001;26:1873–8.
5. Tertti M, Paajanen H, Laato M, et al. Disc degeneration in magnetic resonance imaging: a comparative biochemical, histologic, and radiologic study in cadaver spines. *Spine* 1991;16:629–34.
6. Boden SD, McCowin P, Davis D, et al. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation. *J Bone Joint Surg* 1990;72:1178–84.
7. Matsumoto M, Fujimura Y, Suzuki N, et al. MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg Br* 1998;80:19–24.
8. Fardon DF. Nomenclature and classification of lumbar disc pathology. *Spine* 2001;26:461–2.
9. Fardon DF, Milette PC. Nomenclature and classification of lumbar disc pathology: recommendations of the combined task forces of the North American Spine Society, American Society of Spine Radiology, and American Society of Neuroradiology. *Spine* 2001;26: E93–113.
10. Masui T, Yukawa Y, Nakamura S, et al. Natural history of patients with lumbar disc herniation observed by magnetic resonance imaging for minimum 7 years. *J Spinal Disord Tech* 2005;18: 121–6.
11. Vernon-Roberts B, Moore RJ, Fraser RD. The natural history of age-related disc degeneration: the pathology and sequelae of tears. *Spine* 2007;32:2797–804.
12. Fehlings MG, Wilson JR, Kopjar B, et al. Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy results of the AOSpine North America Prospective Multi-Center Study. *J Bone Joint Surg* 2013;95:1651–8.
13. Kato F, Yukawa Y, Suda K, et al. Normal morphology, age-related changes and abnormal findings of the cervical spine. Part II: magnetic resonance imaging of over 1,200 asymptomatic subjects. *Eur Spine J* 2012;21:1499–507.
14. Miller J, Schmatz C, Schultz A. Lumbar disc degeneration: correlation with age, sex, and spine level in 600 autopsy specimens. *Spine* 1988;13:173–8.
15. Lebkowski WJ. [Autopsy evaluation of the extent of degeneration of the lumbar intervertebral discs]. *Polski merkuriusz lekarski: organ Polskiego Towarzystwa Lekarskiego* 2002;13:188–90.
16. Takatalo J, Karppinen J, Niinimaki J, et al. Prevalence of degenerative imaging findings in lumbar magnetic resonance imaging among young adults. *Spine (Phila Pa 1976)* 2009;34:1716–21.
17. Adams M, Hutton W. Prolapsed intervertebral disc: a hyperflexion injury. *Spine* 1982;7:184–91.

Spine

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CLINICAL CASE SERIES

# A Study of Risk Factors for Tracheostomy in Patients With a Cervical Spinal Cord Injury

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**Study Design.** A retrospective, consecutive case series.
**Objective.** To determine the risk factors for a tracheostomy in patients with a cervical spinal cord injury.
**Summary and Background Date.** Respiratory status cannot be stabilized in patients with a cervical spinal cord injury (CSCI) for various reasons, so a number of these patients require long-term respiratory care and a tracheostomy. Various studies have described risk factors for a tracheostomy, but none have indicated a relationship between imaging assessment and the need for a tracheostomy. The current study used imaging assessment and other approaches to assess and examine the risk factors for a tracheostomy in patients with a CSCI.
**Methods.** Subjects were 199 patients who were treated at the Spinal Injuries Center within 72 hours of a CSCI over 8-year period. Risk factors for a tracheostomy were retrospectively studied. Patients were assessed in terms of 10 items: age, sex, the presence of a vertebral fracture or dislocation, ASIA Impairment Scale, the neurological level of injury (NLI), $PaO_2$, $PaCO_2$, the level of injury on magnetic resonance imaging (MRI), the presence of hematoma-like changes (a hypointense core surrounded by a hyperintense rim in T2-weighted images) on MRI, and the Injury Severity Score.
Items were analyzed multivariate logistic regression, and $P<0.05$ was considered to indicate a significant difference.
**Results.** Twenty-three of the 199 patients required a tracheostomy, accounting for 11.6% of patients with a CSCI. Univariate analyses of the risk factors for tracheostomy revealed significant differences for six items: age, Injury Severity Score, presence of fracture or dislocation, ASIA Impairment Scale A, NLI C4 or above, and MRI scans revealing hematoma-like changes. Multivariate logistic regression analyses revealed significant differences in terms of two items: NLI C4 or above and MRI scans revealing hematoma-like changes. Thirty patients had both an NLI C4 or above and MRI scans revealing hematoma-like changes. Of these, 17 (56.7%) required a tracheostomy.
**Conclusion.** Patients with an NLI C4 or above and MRI scans revealing hematoma-like changes were likely to require a tracheostomy. An early tracheostomy should be considered for patients with both of these characteristics.
**Key words:** arterial blood gases, ASIA impairment scale, cervical spinal cord injury, hematoma-like changes on magnetic resonance imaging, imaging assessment, injury severity score, neurological level of injury, risk factors, tracheostomy, vertebral fracture or dislocation.
**Level of Evidence:** 3
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Respiratory status cannot be stabilized in patients with a cervical spinal cord injury (CSCI) for various reasons, so a number of these patients require long-term respiratory care and a tracheostomy. Numerous studies have reported that respiratory dysfunction is closely associated with morbidity and mortality in CSCI,[1–3] and respiratory dysfunction leads to massive financial expenses.[3] The cause of death for patients with a CSCI is often a urinary complication or a respiratory complication.[4,5] A recent study from abroad has reported that respiratory complications represent the leading cause of death in patients with a CSCI,[5] and the same is true in Japan.[4]

In the acute phase of CSCI, spinal shock can have an effect and the patient's respiratory status might be unstable, so temporary ventilator management is often required.[6–16] If the patient's respiratory status fails to improve with temporary ventilator management and long-term intubation is required, a tracheostomy is often performed. Performing a tracheostomy early on is known to be useful in reducing respiratory complications, as various studies have reported[6,9,16–18]; however, unnecessary intubation and unnecessary tracheostomies are known to increase

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the risk of complications in both the short and long term.[9] Exaggerating the usefulness of an early tracheostomy can result in unnecessary tracheal intubation or an unnecessary tracheostomy. Thus, predicting true risk factors for intubation or a tracheostomy in patients with a CSCI is important.

Various studies have described risk factors for a tracheostomy in patients with a CSCI. These include advanced age,[7,12] complete paralysis,[1,6–8,14,17,19–22,23] a high level of neurological paralysis,[6,12,14,21] the patient's general condition prior to the injury and a prior history of lung disease,[1,8,12] a high injury severity score (ISS),[8,17] a history of smoking,[19] and a low forced vital capacity upon admission.[7,9]

Nevertheless, no previous studies have indicated a relationship between imaging assessment (radiographs, computed tomography, or magnetic resonance imaging [MRI]) and the need for a tracheostomy. The current study used imaging assessment and other approaches to assess and examine the risk factors for a tracheostomy in patients with a CSCI.

## METHODS

Subjects were 199 patients who were treated by the Department of Orthopedic Surgery of the Spinal Injuries Center within 72 hours of a CSCI over 8-year period from January 1, 2005 to December 31, 2012. Patients consisted of 165 males and 34 females ranging in age from 14 to 91 years with a mean age of 61.9 years.

All patients were examined by two or more physicians and a physical therapist upon admission, and patients were assessed neurologically. In addition, the presence or absence of a vertebral fracture or dislocation was assessed using X-ray films or computed tomography scans and the presence or absence of cord damage was assessed using MRI scans in all patients upon admission. Surgery (anterior spine fusion, posterior spine fusion, or anterior spine fusion and posterior spine fusion,) was performed on patients with apparent spinal instability. Arterial blood gases were measured in all patients upon admission. If patients had apnea or ventilatory failure prior to initial admission and they needed assistance breathing, a tracheostomy was performed on the day of admission. If, however, a patient's respiratory status gradually worsened after admission, intubation was performed at the discretion of the patient's primary physician in accordance with the patient's respiratory status. When long-term ventilator management was considered necessary, a tracheostomy was performed. This Center has not formulated definite standards for intubation and tracheostomy, although blood gas results of $PaO_2$ 70 mm Hg or less and $PaCO_2$ at least 50 mm Hg serve as somewhat of a guide, regardless of whether $O_2$ is administered.

Medical records, the patient's discharge summary, and imaging findings upon admission and discharge were retrospectively studied.

The following items were studied retrospectively: age, sex, the presence or absence of a vertebral fracture or dislocation at the level of injury, the American Spinal Association (ASIA) Impairment Scale (AIS), the neurological level of injury (NLI), $PaO_2$ according to a blood gas analysis, $PaCO_2$ according to a blood gas analysis, the level of injury on MRI, hematoma-like changes on MRI (presence or absence of a hypointense core surrounded by a hyperintense rim in T2-weighted images), and the ISS.

Statistical analyses were done using the Jump11 statistical software package from SAS Institute Inc. (Cary, NC).

In instances wherein there was no avulsion fracture of the anterior aspect of the vertebral body or a spinous process fracture in conjunction with a hyperextension injury, no bone injury on MRI scans, and no need for surgery due to the lack of spinal instability, bone injury or dislocation was deemed to be absent. In addition, the NLI was the most caudal segment wherein normal motor and sensory function were intact. If hematoma-like changes were present, the level of those changes served as the level of injury on MRI. If those changes were absent, the segment wherein the center of a wider ranging hyperintensity was located on T2-weighted images served as the level of injury on MRI (Figure 1A, B).

Age, $PaO_2$, $PaCO_2$, and the ISS were assessed as continuous variables. To increase statistic power, the AIS, the NLI, and the level of injury on MRI were dichotomized as an AIS A or not, an NLI C4 or above or not, and a level of injury on MRI C3/4 or less or not.

## RESULTS

A total of 199 patients with a cervical spinal cord injury were treated at this center over 8-year period. Patients consisted of 165 males and 34 females ranging in age from 14 to 91 years with a mean age of 61.9 years. All of the patients had suffered blunt trauma. Sixty patients (30.1%) had a vertebral fracture or dislocation.

The extent of paralysis upon admission was AIS A in 66 patients (33.2%), AIS B in 38 (19.1%), AIS C in 51 (25.6%), and AIS D in 44 (22.1%). The NLI upon admission was C2 in 1 patient (0.5%), C3 in 10 (5.0%), C4 in 90 (45.2%), C5 in 62 (31.2%), C6 in 19 (9.6%), C7 in 6,(3.0%), C8 in 1 (0.5%), and T1 in 10 (5.0%) (Table 1).

All of the patients underwent an MRI upon admission, and hyper-intensity changes on T2-weighted images were noted in all of the patients. The level of injury on MRI was C2/3 in 5 patients (2.5%), C3 in 2 (1.0%), C3/4 in 70 (35.2%), C4 in 4 (2.0%), C4/5 in 49 (24.6%), C5 in 10 (5.0%), C5/6 in 29 (14.6%), C6 in 4 (2.0%), C6/7 in 24 (12.1%), C7 in 1 (0.5%), and C7/T1 in 1 (0.5%). Hematoma-like changes were noted in 46 patients (23.1%) and such changes were not noted in 153 (76.9%) (Table 2).

Twenty-three of the 199 patients required a tracheostomy, accounting for 11.6% of patients with a cervical spinal cord injury. The average time from injury until a tracheostomy was performed was 4.69 days (day of injury–13 days later). Details on the 23 patients who underwent a tracheostomy are indicated below (Table 3).

Eleven patients had a vertebral fracture or dislocation; a bone injury was not noted in 12 patients. Seventeen patients had an AIS A, 4 had AIS B, and 2 had AIS C. The NLI was C2

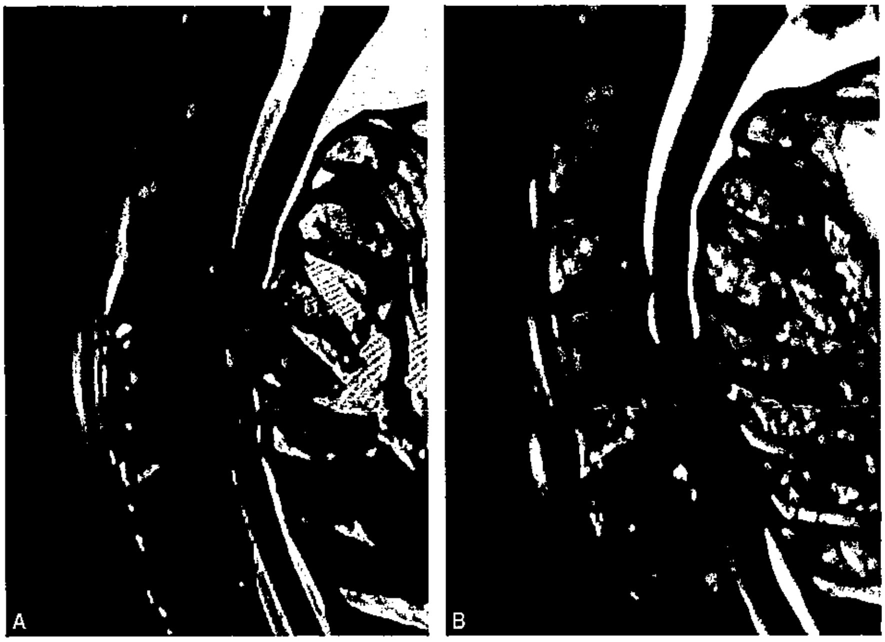

Figure 1. "Hematoma-like changes" and the level of injury on MRI. A, MRI findings of a hypointense core surrounded by a hyperintense rim in a T2-weighted image. We described these changes as "hematoma-like changes." When these changes were present, the level of the changes served as the level of injury on MRI. This patient's level of injury on MRI was "C3/4". B, When hematoma-like changes were absent, the segment containing the center of a wider-ranging hyperintensity area served as the level of injury on MRI. This patient's level of injury on MRI was "C5/6." MRI indicates magnetic resonance imaging.

in 1 patient, C3 in 5, C4 in 15, C5 in 1 and T1 in 1. The level of injury on MRI scans was at C2/3 in 1 patient, at C3/4 in 9, at C4/5 in 7, at C5/6 in 5, and at C6/7 in 1. Hematoma-like changes were noted on MRI images of 17 patients.

The final outcome (state at final follow-up) was death for one patient and permanent ventilator management for seven. The respiratory status of 15 patients stabilized, and they were weaned from the ventilator.

TABLE 1. Patients Demographic Data

| Mean ± SD | Overall (n = 199) | Tracheostomy (n = 23) | No Tracheostomy (n = 176) |
|---|---|---|---|
| Age (yrs) | 61.9 ± 17.7 | 69.1 ± 15.8 | 60.9 ± 17.8 |
| Sex | | | |
| Male | 165 (82.9%) | 17 (73.9%) | 148 (84.1%) |
| Female | 34 (17.1%) | 6 (26.1%) | 28 (15.9%) |
| Fracture or dislocation | | | |
| + | 60 (30.1%) | 11 (43.5%) | 49 (27.8%) |
| − | 139 (69.9%) | 12 (56.5%) | 127 (72.2%) |
| Initial AIS | | | |
| A | 66 (33.2%) | 17 (73.9%) | 49 (27.8%) |
| B | 38 (19.1%) | 4 (17.4%) | 34 (19.4%) |
| C | 51 (25.6%) | 2 (8.7%) | 49 (27.8%) |
| D | 44 (22.1%) | 0 | 44 (25.0%) |
| Initial NLI | | | |
| C2 | 1 (0.5%) | 1 (4.4%) | 0 |
| C3 | 10 (5.0%) | 5 (21.7%) | 5 (2.8%) |
| C4 | 90 (45.2%) | 15 (65.3%) | 75 (42.6%) |
| C5 | 62 (31.2%) | 1 (4.4%) | 61 (34.7%) |
| C6 | 19 (9.6%) | 0 | 19 (10.9%) |
| C7 | 6 (3.0%) | 0 | 6 (3.4%) |
| C8 | 1 (0.5%) | 0 | 1 (0.5%) |
| T1 | 10 (5.0%) | 1 (4.4%) | 9 (5.1%) |
| Arterial blood gases | | | |
| $PO_2$ (mm Hg) | 91.9 ± 40.7 | 104.1 ± 71.2 | 90.3 ± 34.9 |
| $PCO_2$(mm Hg) | 38.3 ± 5.1 | 39.9 ± 5.4 | 38.1 ± 5.0 |
| ISS | 21.4 ± 10.6 | 31.7 ± 18.9 | 20.1 ± 8.2 |

*AIS indicates American Spinal Association impairment scale; ISS, injury severity score; NLI, neurological level of injury; SD, standard deviation.*

**TABLE 2. Level of Spinal Cord Injury and Hematoma-Like Changes on MRI**

| | Overall (n = 199) | Tracheostomy (n = 23) | No tracheostomy (n = 176) |
|---|---|---|---|
| Level of spinal cord injury on MRI | | | |
| C2/3 | 5 (2.5%) | 1 (4.4%) | 4 (2.3%) |
| C3 | 2 (1.0%) | 0 | 2 (1.1%) |
| C3/4 | 70 (35.2%) | 9 (39.1%) | 61 (34.7%) |
| C4 | 4 (2.0%) | 0 | 4 (2.3%) |
| C4/5 | 49 (24.6%) | 7 (30.4%) | 42 (23.9%) |
| C5 | 10 (5.0%) | 0 | 10 (5.7%) |
| C5/6 | 29 (14.6%) | 5 (21.7%) | 24 (13.6%) |
| C6 | 4 (2.0%) | 0 | 4 (2.3%) |
| C6/7 | 24 (12.1%) | 1 (4.4%) | 23 (13.1%) |
| C7 | 1 (0.5%) | 0 | 1 (0.5%) |
| C7/T1 | 1 (0.5%) | 0 | 1 (0.5%) |
| Hematoma-like changes on MRI | | | |
| + | 46 (23.1%) | 17 (73.9%) | 29 (16.5%) |
| – | 153 (76.9%) | 6 (26.1%) | 147 (83.5%) |

*MRI indicates magnetic resonance imaging; SD, standard deviation.*

Univariate analyses of the risk factors for tracheostomy revealed significant differences for six items: age ($P = 0.0422$, odds ratio [OR] = 1.035), ISS ($P = 0.0002$, OR = 1.065), presence of fracture or dislocation ($P = 0.0209$, OR = 2.827), AIS A ($P < 0.0001$, OR = 7.344), NLI C4 or above ($P = 0.0008$, OR = 12.599), and MRI scans revealing hematoma-like changes ($P < 0.0001$, OR = 9.504) (Table 4).

The aforementioned items with significant differences in the univariate analyses were further analyzed using multivariate logistic regression. The results revealed significant differences for two items: NLI C4 or above ($P = 0.0058$, OR = 9.681) and MRI scans revealing hematoma-like changes ($P = 0.0212$, OR = 3.941) (Table 5).

In addition, 17 of 30 patients (56.7%) who had both an NLI C4 or above and MRI scans revealing hematoma-like

**TABLE 3. Demographic Data of Tracheostomy Patients**

| Case No. | Age | Sex | Type of Fracture | AIS | NLI | T2 High Level | Hematoma-like changes on MRI | PO2 on Admission (mm Hg) | PCO2 on Admission (mm Hg) | Final Form |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | 80 | F | C6 fracture dislocation | A | C4 | C6/7 | + | 74.73 | 45.2 | Trach collar |
| 2 | 47 | M | C3 tear drop fracture T4 fracture dislocation | A | T1 | C3/4 | – | 80.9 | 36 | Closed |
| 3 | 49 | M | C5 burst fracture | A | C4 | C5/6 | + | 66.7 | 41.8 | Closed |
| 4 | 64 | M | C4 fracture dislocation | A | C4 | C4/5 | + | 72.1 | 36.5 | Trach collar |
| 5 | 82 | F | C3 fracture dislocation | C | C4 | C3/4 | + | 145.7 | 45.1 | Closed |
| 6 | 84 | M | C4 fracture dislocation | A | C3 | C4/5 | + | 74.5 | 34.4 | Mechanical ventilation |
| 7 | 75 | M | C5 fracture (post ASF) | C | C3 | C4/5 | – | 45.8 | 46.6 | Closed |
| 8 | 61 | F | C5 fracture dislocation | A | C4 | C5/6 | + | 116.9 | 37.4 | Closed |
| 9 | 80 | F | C3 burst fracture | B | C4 | C3/4 | + | 59.0 | 40.0 | Closed |
| 10 | 76 | M | C5 fracture dislocation | A | C4 | C5/6 | + | 161.0 | 33.0 | Trach collar |
| 11 | 70 | M | C3 fracture dislocation | A | C3 | C3/4 | + | 166.0 | 46.7 | Mechanical ventilation |
| 12 | 51 | M | No fracture | A | C3 | C5/6 | – | 97.0 | 49.0 | Mechanical ventilation |
| 13 | 77 | M | No fracture | A | C4 | C4/5 | + | 48.6 | 47.6 | Mechanical ventilation |
| 14 | 22 | M | No fracture | A | C3 | C2/3 | + | 392 (intubated) | 40.7 (intubated) | Closed |
| 15 | 63 | M | No fracture | B | C4 | C3/4 | – | 63.4 | 33.8 | Closed |
| 16 | 89 | M | No fracture | A | C4 | C4/5 | + | 93.6 | 34.8 | Died |
| 17 | 72 | M | No fracture | B | C4 | C3/4 | – | 86.5 | 33.4 | Closed |
| 18 | 69 | F | No fracture | A | C4 | C3/4 | + | 123 | 43.9 | Closed |
| 19 | 61 | M | No fracture | B | C2 | C3/4 | + | 127 | 35.7 | Mechanical ventilation |
| 20 | 85 | F | No fracture | A | C4 | C4/5 | + | 69.5 | 43.1 | Closed |
| 21 | 83 | M | No fracture | A | C5 | C5/6 | – | 72.5 | 37.2 | Closed |
| 22 | 66 | M | No fracture | A | C4 | C4/5 | + | 72.7 | 31.6 | Mechanical ventilation |
| 23 | 84 | M | No fracture | A | C4 | C3/4 | + | 86.2 | 43.9 | Mechanical ventilation |

*AIS indicates American Spinal Association impairment scale; ASF, anterior spine fusion; MRI, magnetic resonance imaging; NLI, neurological level of injury.*

TABLE 4. Results of the Simple Logistic Regression Model

| | *P* | OR | 95% Confidence Interval |
|---|---|---|---|
| Age | 0.0422 | 1.035 | 1.004–1.074* |
| ISS | 0.0002 | 1.065 | 1.032–1.106* |
| $PO_2$ on admission | 0.1445 | 1.006 | 0.997–1.014 |
| $PCO_2$ on admission | 0.1055 | 1.076 | 0.986–1.183 |
| Sex | 0.2284 | 1.866 | 0.629–4.939 |
| Fracture or dislocation (+) | 0.0209 | 2.827 | 1.166–6.931* |
| AIS A | <0.0001 | 7.344 | 2.870–21.353* |
| NLI ≥C4 | 0.0008 | 12.599 | 3.550–80.260* |
| Injury level on MRI ≥C3/4 | 0.6169 | 1.251 | 0.508–3.004 |
| Hematoma-like changes on MRI | <0.0001 | 9.504 | 3.780–25.604* |

*Age, ISS, $PO_2$, and $PCO_2$ were calculated by the continuous variable function unit odds ratio.*
*AIS indicates American Spinal Association impairment scale; ISS, injury severity score; MRI, magnetic resonance imaging; NLI, neurological level of injury; OR, odds ratio.*
*$^*P<0.05$.*

changes required a tracheostomy, whereas 15 of 40 patients (37.5%) who had both an NLI C4 or above and AIS A on admission required a tracheostomy.

## DISCUSSION

Various complications can occur after a CSCI, although the most frequent are respiratory complications.[1–5] Typical complications include atelectasis, pneumonia, and ventilatory failure. These complications often occur in the acute phase within 5 days of injury.[13]

The diaphragm is innervated by nerves originating from C3–C5 (primarily from C4), and damage to the spinal cord at a higher level will immediately necessitate ventilator management. Sputum is expelled and coughing is accomplished primarily with the intercostal muscles and abdominal muscles. Even if the injury occurred at a lower level and the diaphragm still functioned, paralysis of these muscles causes sputum to pool, thereby facilitating atelectasis and pneumonia.[10,16] In the acute phase of injury, the sympathetic nerves are interrupted and the vagus nerve predominates. Furthermore, tracheobronchial secretions increase and the airway constricts. The amount of sputum increases and the sputum cannot be readily expelled. This phenomenon is one reason for why respiratory complications are so frequent.

In the first week after injury, a patient's vital capacity will decrease by 30% or more. About 5 weeks after injury, vital capacity will begin to recover, and 3 months after injury the vital capacity will double.[11,16] If the period of an unstable respiratory status in the acute-subacute phase of injury can be weathered, then the respiratory status will subsequently stabilize for the most part. Thus, predicting risk factors for intubation or tracheostomy in patients with a CSCI is important.

Studies cite widely differing figures for the percentage of patients requiring a tracheostomy after CSCI. These figures range from 15.2% to 81%, and recent studies have noted that a relatively high percentage of those patients require a tracheostomy.[6–9,12,17,19,21,24] Numerous studies have reported that performing a tracheostomy early on is useful in reducing respiratory complications.[6,9,16–18] This might be the reason for the substantial difference in the percentage of patients with a tracheostomy. That said, one possibility is that intubation or tracheostomy is performed unnecessarily, and such situations should be avoided.

The current study found that 11.6% of patients with a CSCI require a tracheostomy, and this is lower than the percentages described in other studies. This Center is a dedicated facility with a Department of Orthopedic Surgery and in principle this facility does not accept patients in the

TABLE 5. Results of the Multiple Logistic Regression Model

| | *P* | OR | 95% Confidence Interval |
|---|---|---|---|
| Age | 0.0782 | 1.031 | 0.999–1.071 |
| ISS | 0.2784 | 1.025 | 0.983–1.077 |
| Fracture or Dislocation (+) | 0.2477 | 2.049 | 0.597–6.970 |
| AIS A | 0.2180 | 2.329 | 0.600–9.154 |
| NLI ≥ C4 | 0.0058 | 9.681 | 2.362–66.748* |
| Hematoma-like changes on MRI | 0.0212 | 3.941 | 1.243–13.131* |

*Age and ISS were calculated by the continuous variable function unit odds ratio.*
*AIS indicates American Spinal Association impairment scale; ISS, injury severity score; MRI, magnetic resonance imaging; NLI, neurological level of injury; OR, odds ratio.*
*$^*P<0.05$.*

acute phase of multiple trauma. This Center sees a small proportion of patients with complications such as a brain contusion, multiple rib fractures, a hemopneumothorax, or injuries to the abdominal viscera, which might explain why so few of the current patients required a tracheostomy. The aforementioned reasons are also presumably the reason why tracheal intubation or a tracheostomy is so often indicated for a simple CSCI seen at this Center. In this study, univariate analyses and multivariate analyses yielded significant differences in terms of two items: an NLI C4 or above and MRI scans revealing hematoma-like changes. The aforementioned reasons are presumably why these two items were predictors for a tracheostomy, in a true sense, in patients with a CSCI.

Numerous studies have reported that complete paralysis is a risk factor for intubation or a tracheostomy.[1,6–8,14,17,19–22,23] In the current study, univariate analyses indicated that AIS A was a risk factor for tracheostomy, whereas multivariate analyses revealed no significant differences in AIS A.

This might be because complete paralysis means that the intercostal muscles and abdominal muscles are paralyzed, regardless of the level of the CSCI; however, Yugue *et al*[7] reported that 18.8% of patients who were AIS A upon admission had an improved level of paralysis (a grade other than A) at final follow-up. Similarly, 66 patients in the current study were AIS A upon admission, although eight (12.1%) had improvement at final follow-up. The period of spinal shock might have been included in the 72 hours after injury. Given this possibility, improvement in the level of paralysis needs to be assessed.

The ISS was similarly found to be a risk factor for tracheostomy according to univariate analyses. ISS is a score for the severity of multiple trauma. Several studies have reported that a high ISS is a predictor for tracheostomy in patients with a CSCI[8,21]; however, Velmahos *et al*[21] stated that assessment of the ISS is difficult in the acute phase of trauma, and they recommended that the ISS not be used as a predictor of tracheostomy.

No studies have indicated the relationship between imaging assessment and the need for a tracheostomy. The current study is the first to do so. In the acute phase of injury, blurred hyperintensity on T2-weighted MRI indicates spinal cord contusion or edema.[25–27] This imaging finding suggests injury to the spinal cord. If cord damage is severe and hematomyelia is present, hypointensities will be found inside hyperintensities in T2-weighted images. This finding mostly suggests severe spinal cord injury,[28,29] and we described those change as "hematoma-like changes." Bozzo *et al*[29] reported that 93% of patients with MRI scans revealing a hypointense core surrounded by a hyperintense rim in T2-weighted images (hematoma-like changes) were AIS A upon admission. Of these, 95% were AIS A at final follow-up. In the current study, 82.6% of patients who were found to have hematoma-like changes were AIS A upon admission. All of the patients were AIS A at final follow-up, and this grade might be correlated with severe paralysis.

In the current study, univariate analyses and multivariate analyses revealed significant differences in MRI scans revealing hematoma-like changes. The AIS grade might change over time because of its relationship to spinal shock. Consequently, the ISS might change as well. Thus, these indicators change. In contrast, MRI images revealing hematoma-like changes do not change with spinal shock, making these MRI findings an independent indicator.

Of course, MRI was performed within a few hours after the injury. Therefore, there was a possibility that hematoma-like changes (hypointense core surrounded by a hyperintense rim) did not appear on T2-weighted images, even if a hematomyelia occurred, reflecting the intracellular oxyhemoglobin[30]; however, if hypointensity changes were revealed on T2-weighted image within 72 hours after the injury, they reflected deoxyhemoglobin, suggesting that a hemorrhage had occurred in the spinal cord and that the damage was severe.

In addition, significant differences in the level of injury on MRI scans were not noted, although an NLI C4 or above was a risk factor for tracheostomy.

The level of injury on MRI and the NLI did not necessarily coincide. Results suggested that the NLI is important because of its greater clinical significance.

Several studies have reported that an NLI at a high level is a predictor for tracheotomy.[6,12,14,21] Nerves innervating the diaphragm originate at levels C3–C5. The diaphragm is

**TABLE 6. Results of the Multiple Logistic Regression Model in CSCI Patients Who Had NLI ≥ C4 on Admission**

| | *P* | OR | 95% Confidence Interval |
|---|---|---|---|
| Age | 0.1383 | 1.031 | 0.993–1.078 |
| ISS | 0.4848 | 1.016 | 0.974–1.066 |
| Fracture or dislocation (+) | 0.1476 | 2.617 | 0.101–1.411 |
| AIS A | 0.2180 | 1.705 | 0.386–7.369 |
| Hematoma-like changes on MRI | 0.0049 | 6.101 | 1.779–22.842* |

*Age and ISS were calculated by the continuous variable function unit odds ratio.*

*AIS indicates American Spinal Association impairment scale; CSCI, cervical spinal cord injury; ISS, injury severity score; MRI, magnetic resonance imaging; NLI, neurological level of injury; OR, odds ratio.*

*$P < 0.05$.

involved in about 65% of breathing. The current study yielded significant differences at C4 or above. Paralysis due to an injury at C4 or above causes motor paralysis of the diaphragm, which is likely to result in the patient's respiratory status worsening.

Several studies have reported that the risk of tracheostomy is related to age[7,12]; however, multivariate analyses revealed no significant differences in patient age. In addition, this study found no significant differences in patient sex.

Studies have reported that the forced vital capacity upon admission is correlated with the risk of tracheostomy,[7,9] although this characteristic might present problems for facilities that do not have a simple spirometer on hand. Blood gas analysis is simple and convenient, and the results might serve as an indicator in place of forced vital capacity. In actuality, however, significant differences in blood gas results were not evident.

An extensive search yielded no studies indicating a relationship between bone injury or dislocation and the risk of tracheostomy. The current study noted no significant differences in terms of the presence or absence of a vertebral fracture or dislocation in multivariate analyses. This is probably because the damage to the spinal cord is a more important factor than the presence or absence of a vertebral fracture or dislocation.

In the current study, multivariate analyses revealed significant differences in terms of two items: an NLI C4 or above and MRI scans revealing hematoma-like changes.

Multivariate-analyses revealed significant differences in terms of two items in 30 patients. Of these patients, 17 (57%) required a tracheostomy, suggesting that patients with both of the aforementioned characteristics are likely to require a tracheostomy.

In addition, we examined the risk factors for a tracheostomy in a total of 101 CSCI patients who had NLI C4 or above on admission. As a result, hematoma-like changes on MRI only showed a significant difference in the multivariate logistic regression model ($P=0.0049$, $OR=6.101$) (Table 6). This finding raises the possibility that hematoma-like changes on MRI are an optimal indictor of the risk for tracheostomy in patients with a CSCI.

The current study had several limitations. One is that this study was a retrospective study that studied a relatively small sample over an 8-year period. In addition, this study was conducted at a single special facility, that is, the Spinal Injuries Center, and not at other facilities. In addition, definite eligibility criteria for a tracheostomy have yet to be formulated at this facility, and the final decision is left to the discretion of the patient's primary physician. In addition, there is a possibility that hematoma-like changes did not appear on T2-weighted images, when MRI was performed within a few hours after the injury. Moreover, this study did not examine aspects such as patient history, original respiratory status, or whether or not the patient smoked.

Despite these limitations, however, the current study identified significant differences in terms of the two items: an NLI C4 or above and MRI scans revealing hematoma-like changes. These two items are statistically independent risk factors. If patients have both of these characteristics, they are extremely likely to have respiratory failure even if they had a satisfactory respiratory status upon admission. These characteristics can serve as important indices with which to study early tracheal intubation and early tracheostomy.

## ➢ Key Points

- ❑ The current study used imaging assessment and other approaches to assess and examine the risk factors for a tracheostomy in patients with a CSCI.
- ❑ Univariate analyses of the risk factors for tracheostomy revealed significant differences for six items: age, ISS, presence of fracture or dislocation, AIS A, NLI C4 or above, and MRI scans revealing hematoma-like changes.
- ❑ Multivariate logistic regression analyses of the risk factors for tracheostomy revealed significant differences in terms of two items: NLI C4 or above and MRI images revealing hematoma-like changes.
- ❑ Spinal shock has an effect in the acute phase of injury, hampering assessment of the AIS. In contrast, MRI scans revealing hematoma-like changes do not change with spinal shock, making these MRI findings an independent indicator.
- ❑ Patients with an NLI C4 or above and MRI scans revealing hematoma-like changes were likely to require a tracheostomy.

## References


1. Ellen MH, Stein AL, Tiina R, et al. Mortality after traumatic spinal cord injury: 50 years of follow-up. *J Neurol Neurosurg Psychiatry* 2010;81:368–73.
2. Reines David H, Harris Robert C. Pulmonary complications of acute spinal cord injuries. *Neurosurgery* 1987;21:193–6.
3. Winslow C, Rozovsky J. Effect of spinal cord injury on the respiratory system. *Am J Phys Med Rehabil* 2003;82:803–14.
4. Nakajima A, Honda S, Yoshimura S, et al. The disease pattern and causes of death of spinal cord injured patients in Japan. *Paraplegia* 1989;27:163–71.
5. Sekhon LH, Fehlings MG. Epidemiology, demographics, and pathophysiology of acute spinal cord injury. *Spine* 2001;26: 2–12.
6. Como JJ, Sutton ER, McCunn M, et al. Characterizing the need for mechanical ventilation following cervical spinal cord injury with neurologic deficit. *J Trauma* 2005;59:912–6.
7. Yugue I, Okada S, Ueta T, et al. Analysis of the risk factors for tracheostomy in traumatic cervical spinal cord injury. *Spine* 2012;37:E1633–8.
8. Branco BC, Plurad D, Green DJ, et al. Incidence and clinical predictors for tracheostomy after cervical spinal cord injury: a national trauma databank review. *J Trauma* 2011;70:111–5.
9. Berney SC, Gordon IR, Opdam HI, et al. A classification and regression tree to assist clinical making in airway management for patients with cervical spinal cord injury. *Spinal Cord* 2011;49: 244–50.

10. McMichan JC, Michel L, Westbrook PR. Pulmonary dysfunction following traumatic quadriplegia. *JAMA* 1980;243:528–31.
11. Ledsome JR, Sharp JM. Pulmonary function in acute cervical cord injury. *Am Rev Respir Dis* 1981;124:41–4.
12. Harrop LS, Sharan AD, Scheid EH Jr, et al. Tracheostomy placement in patients with complete cervical spinal cord injuries: American Spinal Injury Association Grade A. *J Neurosurg* 2004;100:20–3.
13. Berly M, Shem K. Respiratory management during the first five days after spinal cord injury. *J Spinal Cord Medicine* 2007;30:309–18.
14. Berney S, Bragge P, Granger C, et al. The acute respiratory management of cervical spinal cord injury in the first 6 weeks after injury: a systematic review. *Spinal Cord* 2011;49:17–29.
15. Biering-Sorensen M, Biering-Sorensen F. Tracheostomy in spinal cord injured: frequency and follow up. *Paraplegia* 1992;30:656–60.
16. Perry AB. Critical care of spinal cord injury. *Spine* 2001;26: S27–30.
17. Leelapattana P, Fleming JC, Gurr KR, et al. Predicting the need for tracheostomy in patients with cervical spinal cord injury. *J Trauma Acute Care Surg* 2012;73:880–4.
18. Romero J, Vari A, Gambarrutta C, et al. Tracheostomy timing in traumatic spinal cord injury. *Eur Spine J* 2009;18:1452–7.
19. Nakashima H, Yukawa Y, Imagama S, et al. Characterizing the need for tracheostomy placement and decannulation after cervical spinal cord injury. *Eur Spine J* 2013;22:1526–32.
20. Hassid VJ, Schinco MA, Tepas JJ, et al. Definitive establishment of airway control is critical for optimal outcome in lower cervical spinal cord injury. *J Trauma* 2008;65:1328–32.
21. Velmahos GC, Toutouzas K, Chan L, et al. Intubation after cervical spinal cord injury: to be done selectively or routinely? *Am Surg* 2003;69:892–4.
22. Call MS, Kutcher ME, Izenberg RA, et al. Spinal cord injury: outcomes of ventilator weaning and extubation. *J Trauma* 2011;71:1673–9.
23. Menaker J, Kufera JA, Glaser J, et al. Admission ASIA motor score predicting the need for tracheostomy after cervical spinal cord injury. *J Trauma Acute Care Surg* 2013;75:629–34.
24. Lemons VR, Wagner FV Jr. Respiratory complications after cervical spinal cord injury. *Spine* 1994;15:2315–20.
25. Fujii H, Yone K, Sakou T. Magnetic resonance imaging study of experimental acute spinal cord injury. *Spine* 1993;18:2030–4.
26. Ohshio I, Hatayama A, Kaneda K, et al. Correlation between histopathologic features and magnetic resonance images of spinal cord lesions. *Spine* 1993;18:1140–9.
27. Machino M, Yukawa Y, Ito K, et al. Can magnetic resonance imaging reflect the prognosis in patients of cervical spinal cord injury without radiographic abnormality? *Spine* 2011;36: E1568–72.
28. Schaefer DM, Flanders AE, Osterholm JL, et al. Prognostic significance of magnetic resonance imaging in the acute phase of cervical spine injury. *J Neurosurg* 1992;76:218–23.
29. Bozzo A, Marcoux J, Radhakrishna M, et al. The role of magnetic resonance imaging in the management of acute spinal cord injury. *J Neurotrauma* 2011;28:1401–11.
30. Bradley WG Jr. MR appearance of hemorrhage in the brain. *Radiology* 1993;189:15–26.

# *Risk factors for adjacent segment pathology requiring additional surgery after single-level spinal fusion: impact of pre-existing spinal stenosis demonstrated by preoperative myelography*

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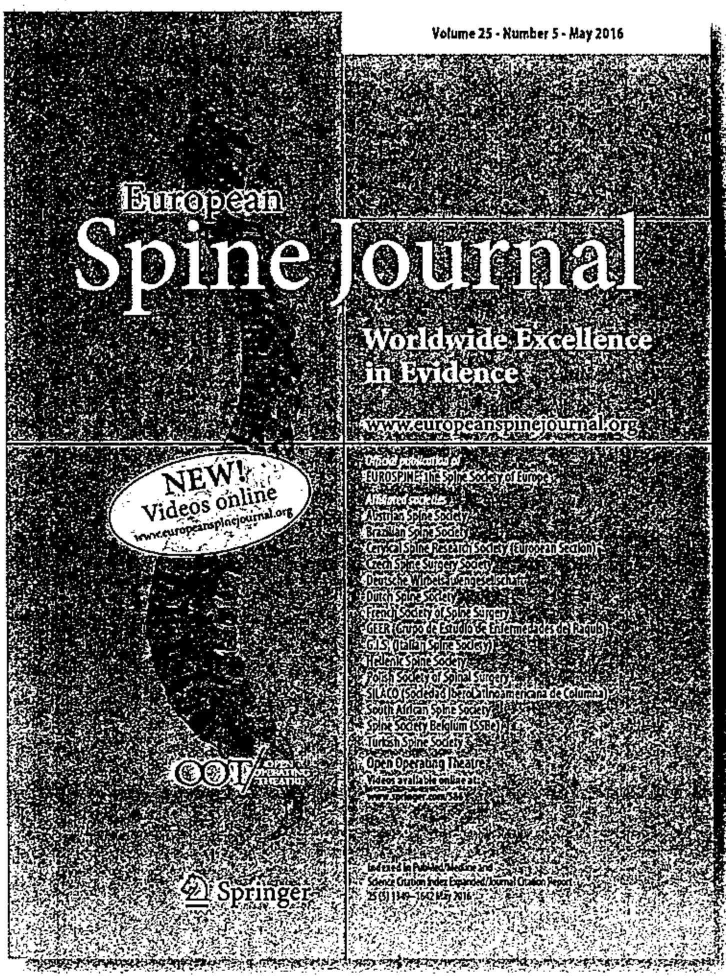


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ORIGINAL ARTICLE

# Risk factors for adjacent segment pathology requiring additional surgery after single-level spinal fusion: impact of pre-existing spinal stenosis demonstrated by preoperative myelography

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**Abstract**

*Purpose* We determined the incidence of and risk factors for clinical adjacent segment pathology (C-ASP) requiring additional surgeries among patients previously treated with one-segment lumbar decompression and fusion surgery.

*Methods* We retrospectively analysed 161 consecutive patients who underwent one-segment lumbar decompression and fusion surgery for L4 degenerative spondylolisthesis. Patient age, sex, body mass index (BMI), facet orientation and tropism, laminar inclination angle, spinal canal stenosis ratio [on myelography and magnetic resonance imaging (MRI)], preoperative adjacent segment instability, arthrodesis type, pseudarthrosis, segmental lordosis at L4–5, and the present L4 slip were evaluated by a log-rank test using the Kaplan–Meier method. A multivariate Cox proportional-hazards model was used to analyse all factors found significant by the log-rank test.

*Results* Of 161 patients, 22 patients (13.7 %) had additional surgeries at cranial segments located adjacent to the index surgery's location. Pre-existing canal stenosis ≥47 % at the adjacent segment on myelography, greater facet tropism, and high BMI were significant risk factors for C-ASP. The estimated incidences at 10 years postoperatively for each of these factors were 51.3, 39.6, and 32.5 %, and the risks for C-ASP were 4.9, 3.7, and, 3.1 times higher than their counterparts, respectively. Notably, spinal canal stenosis on myelography, but not on MRI, was found to be a significant risk factor for C-ASP (log-rank test $P < 0.0001$ and 0.299, respectively).

*Conclusions* Pre-existing spinal stenosis, greater facet tropism, and higher BMI significantly increased C-ASP risk. Myelography is a more accurate method for detecting latent spinal canal stenosis as a risk factor for C-ASP.

**Keywords** Degenerative spondylolisthesis · Adjacent segment pathology · Pre-existing spinal stenosis · Body mass index · Facet tropism

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

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## Introduction

During the past few decades, spinal arthrodesis has become a common treatment component for a variety of spinal disorders. However, it alters the biomechanical and kinematic properties of the lumbar spine [1, 2]. Pathological development at mobile segments above or below the site of spinal fusion is known as adjacent segment pathology (ASP). ASP is considered a potential late complication of spinal arthrodesis that requires further surgical treatment. The clinical failure rate of adjacent segments at 5 years after the index spinal fusion surgery has been reported to range from 3 to 32.3 % [3–7].

Several risk factors for ASP have been reported, such as age [4, 5, 7], sex [4], multilevel arthrodesis [3–5], sagittal imbalance [8], the type of arthrodesis [7], facet tropism [9], and laminar inclination [9]. However, few studies have focused on asymptomatic pre-existing spinal stenosis as a risk factor for clinical ASP (C-ASP) that requires additional surgery at an adjacent segment [10]. In fact, when patients demonstrate asymptomatic spinal stenosis adjacent to the fusion segment, there is often controversy as to whether the segment should be included within the surgical site or not.

This study analysed the preoperative prognostic risk factors for C-ASP, and we calculated the survival times of the patients with significant risk factors.

## Materials and methods

From January 2000 to December 2006, 204 L4 degenerative spondylolisthesis (DS) patients with radicular pain and/or neurological claudication after unsuccessful conservative treatment underwent either instrumented posterolateral fusion (PLF) or posterior lumbar inter-body fusion (PLIF) at the single level of L4–5. All surgeries were performed using the same procedures at a single institution. Patients with an acute fracture, dislocation, or malignancy were excluded. Informed consent was obtained from all patients. Medical records of all patients were reviewed, and this study was approved by our local ethics committee. Forty-three patients were excluded because of a short follow-up (<2 years) or a lack of preoperative magnetic resonance imaging (MRI) and/or myelography data. The remaining 161 patients with a follow-up period of longer than 2 years were finally selected.

PLF had been performed in 137 patients (85 %) and PLIF in 24 patients (15 %). In all patients undergoing PLF, autogenous cancellous iliac bone was used as a graft. PLIF was performed using a rectangular ceramic cage with morselised local bone from neural decompression in all patients. When patients had spinal stenosis on myelography or MRI at the L3–4 segment, as well as neurological findings (including the presence of patellar tendon reflex and no sensory and motor disturbance associated with the L4 nerve root) and negative findings of L4 nerve root infiltration, we did not include the L3–4 segment in the operation site.

### Radiographic evaluation

In all patients, computed tomography (CT), myelography, and MRI were performed within 2 weeks before the index fusion surgery. In this series, no patients required additional surgery at the L5–S1 segment during follow-up, so radiographic evaluations were performed at the L3–4 and L4–5 segments. Standard biplanar anteroposterior, lateral radiography with the lumbosacral spine in neutral, flexion, and extension positions was performed preoperatively, at 24 months after surgery, and at the final follow-up.

The anteroposterior vertebral slip and intervertebral disc angle were measured on lateral radiographs of the L3–4 and L4–5 taken in the neutral, flexion, and extension positions. To minimise the errors due to different magnifications, the vertebral slip was expressed as a percentage of the caudal vertebral body width (% slip). The ranges of motion (ROM) of the L3–4 and L4–5 segments were defined as the sum of the intervertebral disc angles in the flexion/extension view (Fig. 1). Pseudarthrosis was present if there was no continuity in the PLF fusion mass between the cephalad and caudad transverse processes, no continuity between graft bone and vertebra in PLIF fusion, or if lateral flexion–extension radiographs demonstrated >2° of angular motion or >2 mm of sagittal motion at L4–5 [11].

The criteria for adjacent segment instability were well-defined spondylolisthesis or dynamic instability with slippage >4 mm and/or an ROM >10° [12]. The laminar inclination angle at L3 was measured as previously described [9] on lateral radiographs (Fig. 2a). Facet orientation and tropism were determined by CT images that were coplanar with the disc and transected the facet joints, as described previously [9]. The sum of the right and left facet angles and the difference between the right and left facet angles were defined as the facet orientation and tropism, respectively (Fig. 2b).

### Myelography measurements

After lumbar puncture and injection of radiographic contrast material into the dural sac under fluoroscopic guidance, the physician moved the patient's lower back to maximum flexion and extension in the left lateral decubitus position and obtained lateral radiographs in the neutral, flexion, and extension positions under fluoroscopy. The narrowest anteroposterior dural sac diameter at L3–4 was measured on lateral myelography in the neutral, flexion, and extension positions and on the sagittal view of T2-weighted MRI. The dural sac diameter at the midpoint of the L2 vertebral body was also measured. The spinal canal stenosis ratio (SCSR) was calculated as $x/y \times 100$ (Fig. 3).

All measurements were performed twice by two independent observers blinded to the patient name and clinical findings using an electronic digitiser (MicroAnalyzer; Japan Poladigital Corp., Tokyo, Japan) with an accuracy of 0.01 mm: measurements were averaged. The inter-observer correlation of all measurement was evaluated by the Pearson's correlation coefficient test. The kappa statistic was used to assess inter-observer agreement of pseudarthrosis and preoperative instability at L3–4.

### Statistical analysis

The final follow-up examination was defined as the last visit. In patients undergoing re-operation at L3–4, the survival period was defined as the interval from the index operation to the second operation due to C-ASP. C-ASP

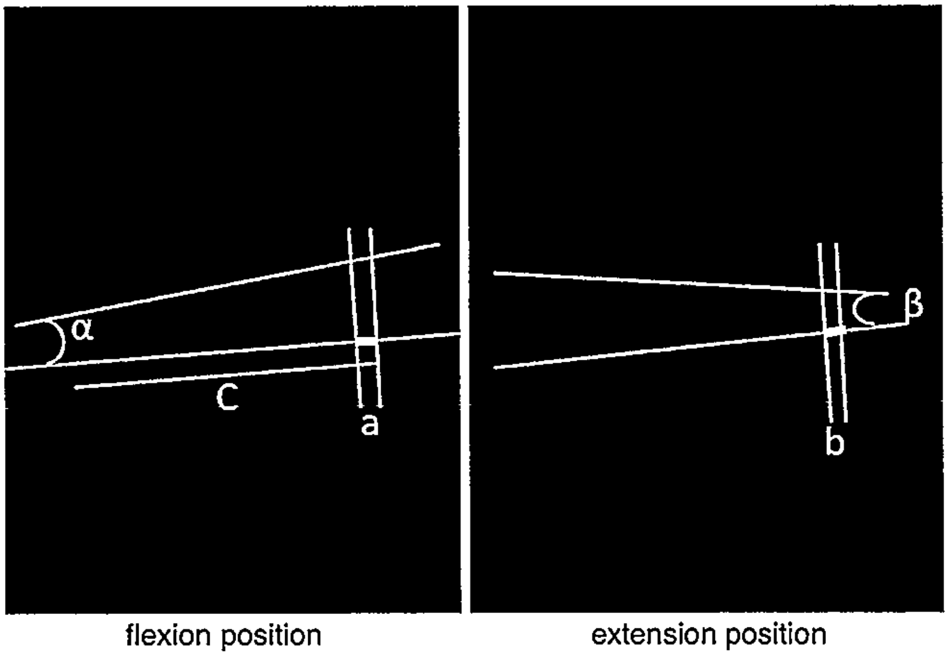


Fig. 1 Plain radiography measurement. *a* Anterior slip in the flexion position, *b* posterior slip in the extension position, *c* vertebral body width. The total percent slip is $(a + b)/c \times 100$. $\alpha$ Intervertebral disc angle in flexion position, $\beta$ intervertebral disc angle in the extension position. The range of motion is $\alpha + \beta$ in degrees

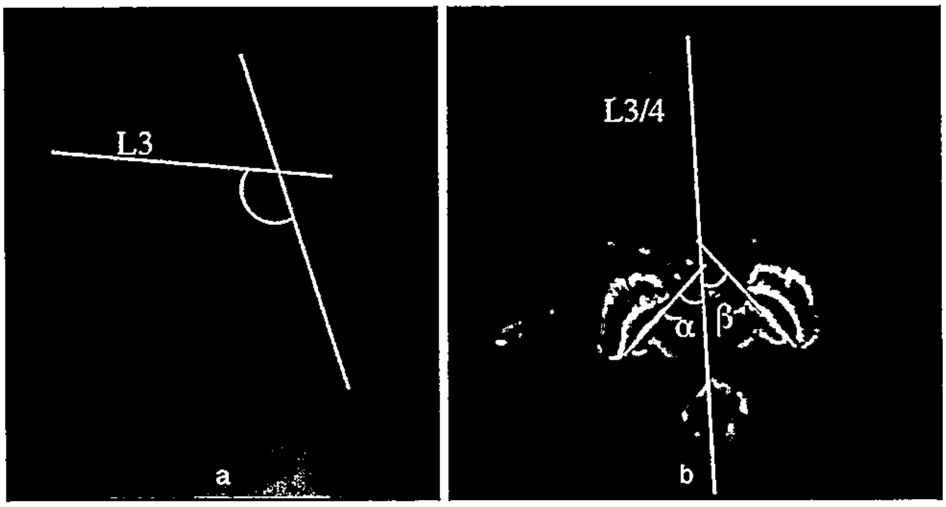


Fig. 2 *a* The laminar inclination angle at L3 was defined as the angle formed by a *straight line* connecting the base of the superior facet with the base of the inferior facet, and a *straight line* connecting the midpoints of the anterior and posterior L3 vertebral cortices on lateral radiographs. *b* Facet orientation and tropism were determined by computed tomography images that were coplanar with the disc and transected the facet joints. The sum of the right and left facet angles and the difference between the right and left facet angles were defined as the facet orientation and tropism, respectively

was defined as a condition where an additional surgery at L3–4 was required to treat symptomatic neurological deterioration.

The following prognostic risk factors were examined: age, sex, body mass index (BMI), facet orientation, facet tropism, laminar inclination angle, SCSR by myelography and MRI, preoperative adjacent segment instability, type of fusion, presence of pseudarthrosis, segmental lordosis at L4–5, and the %slip of L4–5 after 2 years postoperatively. Continuous variables were dichotomised to increase the statistical power using the Youden index from the receiver operating characteristic curve (ROC).

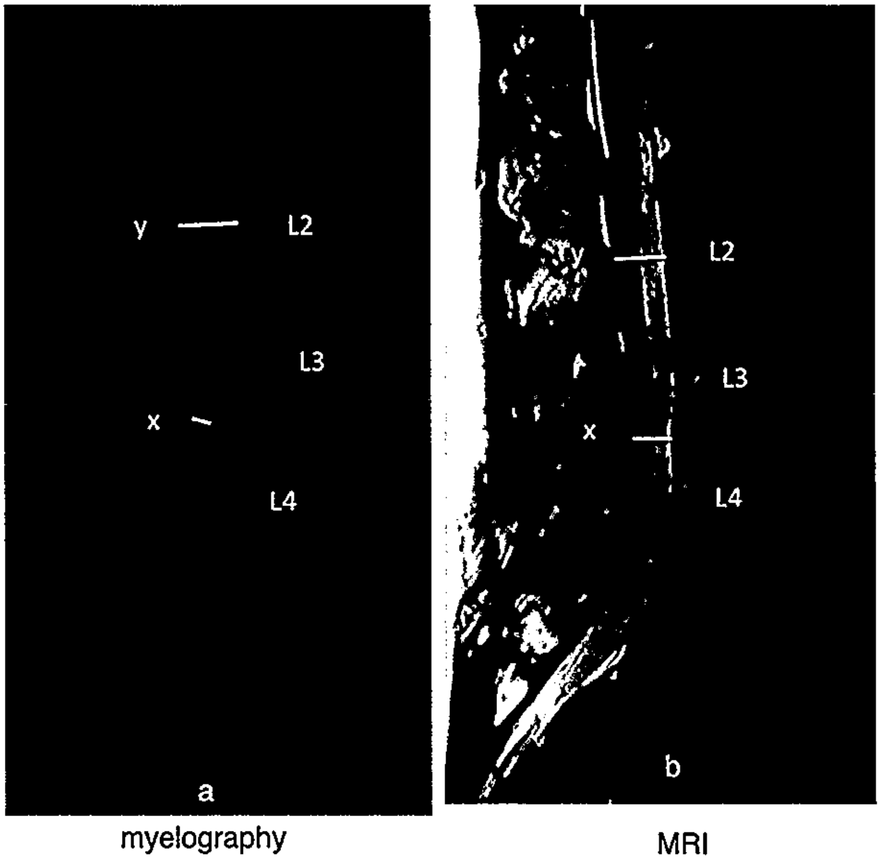


**Fig. 3** The narrowest anteroposterior dural sac diameter at L3–4 (*x*) was measured on lateral myelographs taken in the extension position (a) and on MRI (b) in the same patient. The dural sac diameter at the midpoint of the L2 vertebral body (*y*) was also measured. The spinal canal stenosis ratio (SCSR) was calculated as $x/y \times 100$

A log-rank test was used for univariate analyses using the Kaplan–Meier method, and survival curves for all patients with significant risk factors were constructed to calculate the survival time. A multivariate Cox proportional-hazards model was used to assess all factors demonstrated to be significant by the log-rank test to adjust for confounding factors.

Statistical analyses were performed using the JMP 10 statistical software package (SAS Institute Inc. Cary, NC). A value of $P < 0.05$ was considered to be statistically significant.

## Results

There were 56 males and 105 females. The mean age at index surgery was 65.4 years (range 40–87 years). The average follow-up period was 77.3 months (range 24–183 months). The follow-up rate was 78.9 %. Among the 161 patients, 22 (13.7 %) underwent subsequent procedures at cranial segments adjacent to the L4–5 segment; five patients underwent decompression surgery with arthrodesis and 17 underwent decompression surgery alone. After the additional surgery, all patients show improved neurological symptoms. The mean duration between the index surgery and the additional surgery was 75.9 months (range 24–141 months).

The inter-observer correlation is shown in Table 1. The kappa coefficient for pseudarthrosis rated between observers was 0.82 ($P < 0.0001$) and that of preoperative instability at L3–4 was 0.89 ($P < 0.0001$).

Patients with a BMI $\geq$ 25 kg/m$^2$ had a significantly lower survival rate than their counterparts in a univariate analysis (log-rank test: $P = 0.0497$). The incidence of C-ASP in patients with a BMI $\geq$ 25 kg/m$^2$ was estimated to be 32.5 % at 10 years. Conversely, the incidence of C-ASP in patients with a BMI < 25 kg/m$^2$ was lower, at 21.1 % at 10 years. The median survival time for patients with a BMI $\geq$ 25 kg/m$^2$ was 141 months (Fig. 4).

Patients with facet tropism $\geq$11° demonstrated a lower survival rate than their counterparts (log-rank test: $P = 0.0178$). The incidence of C-ASP among patients with facet tropism $\geq$11° was 39.6 %, while for facet tropism <11° it was 19.4 % at 10 years after the initial operation (Fig. 5).

Regarding SCSR, patients with an SCSR $\geq$47 % on myelography in the extension position showed a significantly lower survival rate than their counterparts

Table 1 Inter-observer correlations for study parameters

| Parameters | P value | Pearson correlation coefficient |
|---|---|---|
| Facet orientation | <0.001 | 0.91 |
| Facet tropism | <0.001 | 0.92 |
| Laminar inclination angle | <0.001 | 0.86 |
| Segmental lordosis at L4–5 | <0.001 | 0.87 |
| %Slip of L4 | <0.001 | 0.89 |
| SCSR of MRI | <0.001 | 0.91 |
| SCSR of myelography (neutral position) | <0.001 | 0.88 |
| SCSR of myelography(flexion position) | <0.001 | 0.87 |
| SCSR of myelography(extension position) | <0.001 | 0.89 |

Significance was set at $P < 0.05$

*SCSR* spinal canal stenosis ratio, *MRI* magnetic resonance imaging

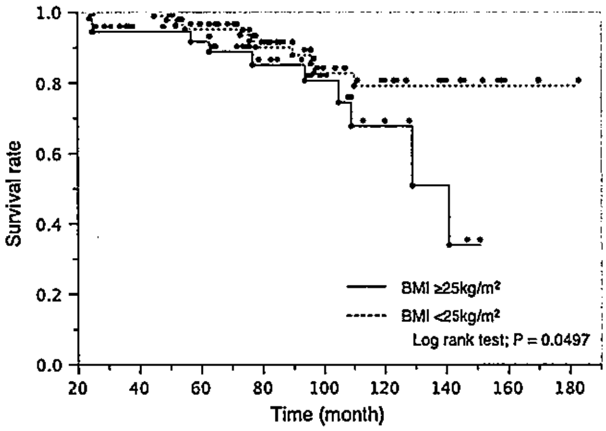


Fig. 4 The Kaplan–Meier survivorship curve of patients with BMI $\geq$ 25 kg/m$^2$ versus those with BMI < 25 kg/m$^2$

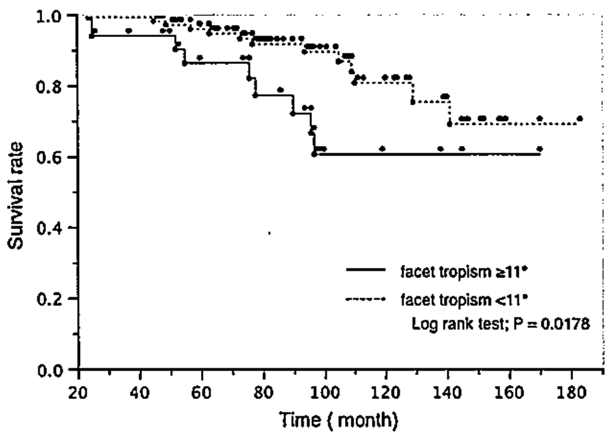


Fig. 5 The Kaplan–Meier survivorship curve of patients with facet tropism $\geq$11° versus those with facet tropism <11°

($P < 0.0001$). In these patients, the prevalence of C-ASP requiring reoperation was 51.3 % at 10 years, whereas it was 11.4 % in patients with an SCSR <47 %. The median

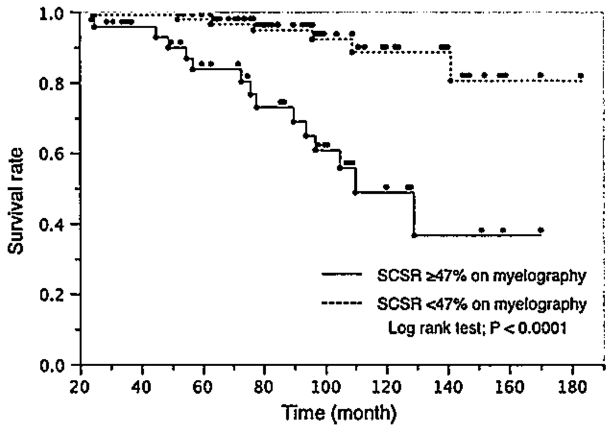


Fig. 6 The Kaplan–Meier survivorship curve of patients with an SCSR $\geq$ 47 % versus those with an SCSR < 47 % on myelography. *SCSR* spinal canal stenosis ratio

survival time for patients with an SCSR $\geq$47 % was 110 months (Fig. 6).

Interestingly, SCSR determined by MRI was not a significant risk factor (log-rank test: $P = 0.2990$). Since a factor with a higher discrimination ability makes an ROC curve closer to the top left corner, the area under the ROC curve (AUC) is used to indicate the sensitivity and specificity of each factor. We compared the AUC values of MRI and myelography in the extension positions. In this analysis, the AUC of the SCSR determined by myelography was significantly higher than that of the SCSR determined by MRI (Fig. 7). In fact, 13.2 % of patients who exhibited SCSR on MRI <50 % had an SCSR $\geq$50 % on myelography in the extension position.

Other potential risk factors, such as age, sex, facet orientation, laminar inclination angle, preoperative adjacent segment instability, type of fusion, pseudarthrosis, segmental lordosis, the %slip, and SCSR on myelography in the neutral and flexion positions, were not statistically significant (Table 2). A multivariate Cox proportional-

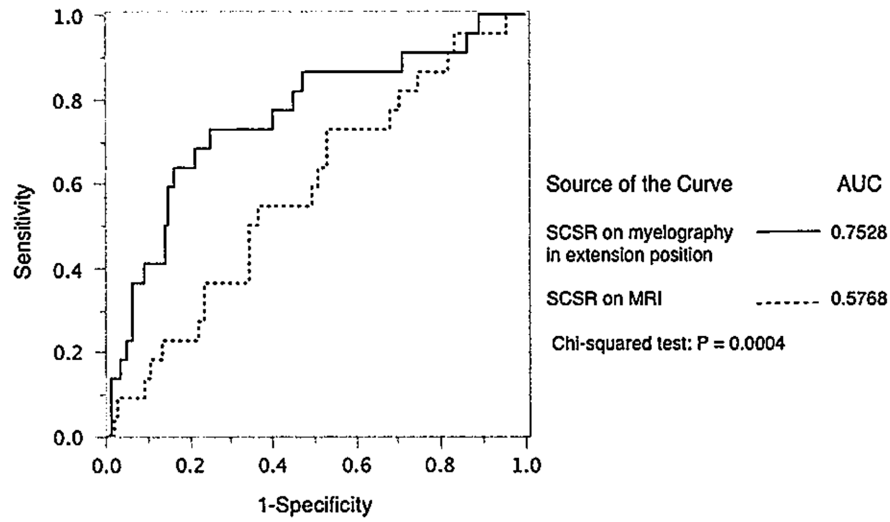


**Fig. 7** Receiver operating characteristic curves of the SCSR on myelography at extension and the SCSR on MRI. *AUC* area under the ROC curve, *SCSR* spinal canal stenosis ratio

**Table 2** Potential risk factors for clinical adjacent segment pathology after lumbar spinal fusion based on the log-rank test

| Risk factor | *P* value |
|---|---|
| Sex (female) | 0.3530 |
| Age ≥ 68 years | 0.3989 |
| BMI ≥ 25 kg/m$^2$ | 0.0497* |
| Facet orientation ≥65° | 0.2272 |
| Facet tropism ≥11° | 0.0178* |
| Laminar inclination ≥120° | 0.6325 |
| SCSR on MRI ≥ 35 % | 0.2990 |
| SCSR on myelography in extension ≥47 % | <0.0001* |
| SCSR on myelography in neutral ≥33 % | 0.0757 |
| SCSR on myelography in flexion ≥18 % | 0.1467 |
| Preoperative instability | 0.6902 |
| Type of fusion | 0.4737 |
| Pseudarthrosis | 0.2086 |
| Segmental lordosis at L4–5 ≥ 6.3° | 0.2278 |
| %Slip of L4 ≥ 13.4% | 0.1465 |

Significance was set at $P < 0.05$*

*BMI* body mass index, *SCSR* spinal canal stenosis ratio, *MRI* magnetic resonance imaging

hazards model revealed a BMI ≥ 25 kg/m$^2$, facet tropism ≥11°, and SCSR ≥47 % on myelography to be significant risk factors, and patients with these factors had 3.1-, 3.7-, and 4.9-fold higher risks of adjacent segment reoperation than their counterparts, respectively (Table 3).

## Discussion

The definition of C-ASP has often been reported as adjacent segment pathology, manifesting radiculopathy, neurogenic intermittent claudication, back pain, or a combination of any of these [13], and the need for additional surgeries [3–5, 8–10] on the index fusion segments. Park et al. [3] reported the incidence of C-ASP to range from 5.2 to 18.5 %. The term 'degeneration' itself suggests a time-dependent phenomenon. Therefore, the survival function estimated by the Kaplan–Meier method and the multivariate Cox regression model are good ways to analyse the development of ASP as a late complication of spinal arthrodesis.

Regardless of the use of spinal arthrodesis, the clinical course of patients with severe spinal stenosis often deteriorates over time during conservative treatments [14]. This indicates that pre-existing spinal stenosis in itself may be a significant risk factor for C-ASP. Cho et al. [10] reported a significant relationship between pre-existing spinal stenosis and C-ASP. However, they did not indicate a cutoff point for spinal stenosis that may increase the likelihood of C-ASP. In the current study, patients with an SCSR ≥47 %

**Table 3** Risk factors for clinical adjacent segment pathology after lumbar spinal fusion

| Risk factor | *P* value | Hazard ratio | 95 % confidence interval |
|---|---|---|---|
| BMI ≥ 25 kg/m$^2$ | 0.0212* | 3.12 | 1.18–8.49 |
| Facet tropism ≥11° | 0.0114* | 3.74 | 1.35–10.30 |
| SCSR in myelography ≥47 % | 0.0003* | 4.87 | 2.05–12.78 |

Significance was set at $P < 0.05$*

*BMI* body mass index, *SCSR* spinal canal stenosis ratio on myelography in the extension position exhibited a 4.87-fold higher risk of adjacent segment reoperation than their counterparts. Interestingly, spinal stenosis demonstrated by MRI was not a significant factor. We attempted to change the cutoff point for SCSR on MRI from 35 to 60 %; however, the MRI findings were not a significant factor. Moreover, the AUC of SCSR on myelography in the extension position (0.7528) was significantly larger than that on MRI (0.5768) (Chi squared; $P = 0.0004$). These results suggest that myelography has a significantly higher sensitivity and specificity to detect not only latent spinal canal stenosis, but also the risk of ASP requiring additional surgery.

While these results indicate that pre-existing severe stenosis can be a significant risk factor for C-ASP, this does not lead directly to a recommendation for performing laminectomy during the index surgery, since performing laminectomy adjacent to a fusion segment demonstrated a significant association with ASP [5, 15–17]. Imagama et al. [15] recommended that the adjacent segment with asymptomatic spinal stenosis should not be subjected to a concomitant decompression from the viewpoint of preventing ASP. We therefore recommend that surgeons should have thorough discussions with patients to determine whether a concomitant operation at adjacent segments with asymptomatic stenosis should be performed.

Facet tropism is defined as asymmetry in the facet joint that causes an abnormal rotation of the spinal segment, which increases the mechanical stress on the disc and could lead to lumbar degeneration or disc herniation [18, 19]. Several studies have reported greater facet tropism to have a significant relationship with C-ASP [9]. In the current study, patients with facet tropism $\geq 11°$ had a 3.74-fold higher risk of adjacent segment reoperation compared to their counterparts. We hypothesised that facet tropism may affect the rotational stability, which accelerates the thickening of the ligamentum flavum, thus resulting in spinal canal stenosis. However, some authors have reported no association between facet tropism and the occurrence of C-ASP [10]. Further studies are required to clarify this issue.

It remains controversial as to whether an association exists between BMI and ASP. Some studies reported no association between BMI and radiographic ASP [20]; however, Cho et al. [10] reported BMI to be a significant risk factor for C-ASP, and Liuke et al. [21] reported that a BMI $\geq 25$ kg/m$^2$ increased the risk of lumbar disc degeneration on MRI. It is assumed that being overweight might cause disc degeneration [22], resulting in earlier ASP over the long term. In this series, a BMI $\geq 25$ kg/m$^2$ was identified as a significant risk factor for C-ASP, and patients with this factor had a 3.12-fold higher risk of needing adjacent segment reoperation than their counterparts. Patients with high BMI appear to have a higher risk of C-ASP; however, a large prospective study is needed to confirm this finding.

In this study, other factors were not significant risk factors for C-ASP. The association between the conditions of fused segments and the occurrence of ASP also remains controversial. Since almost all past studies were retrospective analyses that contained potential bias, a randomised prospective study will be necessary to resolve these issues.

There are several possible limitations associated with this study. First, it was a retrospective study. Second, the predictors derived were not prospectively validated in an independent population. Third, the sample size was relatively small. Finally, whole spinal radiographs were not routinely taken for DS patients who underwent one-segment spinal fusion and decompression and, therefore, a whole spinal radiographic analysis was not possible in this study. Despite these limitations, the factors identified in this study may assist both surgeons and patients when making decisions about whether or not to include an adjacent segment at the time of index fusion surgery.

In conclusion, a BMI $\geq 25$ kg/m$^2$, facet tropism $\geq 11°$, and pre-existing stenosis $\geq 47$ % demonstrated on myelography in the extension position were found to be important risk factors for C-ASP requiring a second operation. Careful consideration of the type and extent of surgery is therefore necessary when these risk factors are present.

**Compliance with ethical standards**

**Conflict of interest** No funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

**Ethical standards** The study was approved by the local ethics committee.

## References

1. Axelsson P, Johnsson R, Strömqvist B (1997) The spondylolytic vertebra and its adjacent segment. Mobility measured before and after posterolateral fusion. Spine (Phila Pa 1976) 22(4):414–417
2. Bastian L, Lange U, Knop C et al (2001) Evaluation of the mobility of adjacent segments after posterior thoracolumbar fixation: a biomechanical study. Eur Spine J 10(4):295–300
3. Ghiselli G, Wang JC, Bhatia NN et al (2004) Adjacent segment degeneration in the lumbar spine. J Bone Joint Surg Am 86-A(7):1497–1503
4. Ahn DK, Park HS, Choi DJ et al (2010) Survival and prognostic analysis of adjacent segments after spinal fusion. Clin Orthop Surg 2(3):140–147
5. Sears WR, Sergides IG, Kazemi N et al (2011) Incidence and prevalence of surgery at segments adjacent to a previous posterior lumbar arthrodesis. Spine J 11(1):11–20

6. Celestre PC, Montgomery SR, Kupperman AI et al (2014) Lumbar clinical adjacent segment pathology: predilection for proximal levels. Spine (Phila Pa 1976) 39(2):172–176
7. Lee JC, Kim Y, Soh JW, Shin BJ (2014) Risk factors of adjacent segment disease requiring surgery after lumbar spinal fusion: comparison of posterior lumbar interbody fusion and posterolateral fusion. Spine (Phila Pa 1976) 39(5):E339–E445
8. Kumar MN, Baklanov A, Chopin D (2001) Correlation between sagittal plane changes and adjacent segment degeneration following lumbar spine fusion. Eur Spine J 10(4):314–319
9. Okuda S, Oda T, Miyauchi A et al (2008) Lamina horizontalization and facet tropism as the risk factors for adjacent segment degeneration after PLIF. Spine (Phila Pa 1976) 33(25):2754–2758
10. Cho TK, Lim JH, Kim SH et al (2013) Preoperative Predictable Factors for the Occurrence of Adjacent Segment Degeneration Requiring Second Operation after Spinal Fusion at Isolated L4–L5 Level. J Neurol Surg A Cent Eur Neurosurg [Epub ahead of print]
11. Kornblum MB, Fischgrund JS, Herkowitz HN et al (2004) Degenerative lumbar spondylolisthesis with spinal stenosis: a prospective long-term study comparing fusion and pseudarthrosis. Spine (Phila Pa 1976) 29(7):726–733
12. Chen WJ, Lai PL, Niu CC et al (2001) Surgical treatment of adjacent instability after lumbar spine fusion. Spine (Phila Pa 1976) 26(22):E519–E524
13. Liao JC, Chen WJ, Chen LH et al (2011) Surgical outcomes of degenerative spondylolisthesis with L5–S1 disc degeneration: comparison between lumbar floating fusion and lumbosacral fusion at a minimum 5-year follow-up. Spine (Phila Pa 1976) 36(19):1600–1607
14. Minamide A, Yoshida M, Maio K (2013) The natural clinical course of lumbar spinal stenosis: a longitudinal cohort study over a minimum of 10 years. J Orthop Sci 18(5):693–698
15. Imagama S, Kawakami N, Kanemura T et al (2013) Radiographic adjacent segment degeneration at five years after L4/5 posterior lumbar interbody fusion with pedicle screw instrumentation: evaluation by computed tomography and annual screening with magnetic resonance imaging. J Spinal Disord Tech [Epub ahead of print]
16. Miyagi M, Ikeda O, Ohtori S et al (2013) Additional decompression at adjacent segments leads to adjacent segment degeneration after PLIF. Eur Spine J 22(8):1877–1883
17. Lai PL, Chen LH, Niu CC et al (2004) Relation between laminectomy and development of adjacent segment instability after lumbar fusion with pedicle fixation. Spine (Phila Pa 1976) 29(22):2527–2532
18. Noren R, Trafimow J, Andersson GB et al (1991) The role of facet joint tropism and facet angle in disc degeneration. Spine (Phila Pa 1976) 16(5):530–532
19. Karacan I, Aydin T, Sahin Z et al (2004) Facet angles in lumbar disc herniation: their relation to anthropometric features. Spine (Phila Pa 1976) 29(10):1132–1136
20. Ha KY, Son JM, Im JH et al (2013) Risk factors for adjacent segment degeneration after surgical correction of degenerative lumbar scoliosis. Indian J Orthop 47(4):346–351
21. Liuke M, Solovieva S, Lamminen A et al (2005) Disc degeneration of the lumbar spine in relation to overweight. Int J Obes (Lond) 29(8):903–908
22. Weiler C, Lopez-Ramos M, Mayer HM et al (2011) Histological analysis of surgical lumbar intervertebral disc tissue provides evidence for an association between disc degeneration and increased body mass index. BMC Res Notes 4:497

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ORIGINAL ARTICLE

# Subacute T1-low intensity area reflects neurological prognosis for patients with cervical spinal cord injury without major bone injury

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**Study design:** A retrospective imaging and clinical study.
**Objectives:** To evaluate the relationship between magnetic resonance imaging (MRI) features and neurological prognosis in patients with traumatic cervical spinal cord injury (CSCI) without major bone injury.
**Methods:** A total of 72 patients with CSCI without major bone injury were treated conservatively in our hospital. MRI was performed for all patients at admission and 1 month following injury. We measured the antero-posterior and cranio-caudal diameter of intramedullary intensity changed area with T1-weighted images at the injured segment. Neurological evaluations were performed using the American Spinal Injury Association (ASIA) motor score and the modified Frankel grade at the time of admission and discharge.
**Results:** There was a significant relationship between the antero-posterior diameter ratio of the T1-weighted low-intensity area on MRI at the subacute stage and the ASIA motor score. The optimal threshold of the T1-weighted low-intensity diameter ratio for predicting the patient's ability to walk with or without assistance at discharge was determined to be 46%. Moreover, 96.8% of the patients with <50% T1-weighted low-intensity area recovered to walk with or without a cane at discharge.
**Conclusion:** The T1-low intensity area may be an important predictive factor for the neurological recovery of CSCI without major bone injury.
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## INTRODUCTION

The incidence of cervical spinal cord injuries (CSCIs) without major bone injury has been increasing. Patients with these injuries show no evidence of fracture or dislocation of the spine on plain radiographs or computed tomography scans.[1,2] The incidence, pathogenesis and severity of CSCI without major bone injury are different for different age groups because of the anatomical and biomechanical differences in the spine.[3,4]

Magnetic resonance imaging (MRI) is the best clinical tool for evaluating traumatic CSCI and is therefore invaluable for examining patients with CSCI without major bone injury. MRI can show the degree of spinal canal stenosis, as well as reveal the intramedullary state of the spinal cord in detail.[5]

To our knowledge, there have been only few reports on the MRI features of patients with CSCI without major bone injury.[6,7] The most common acute MRI pattern described is no change of signal intensity on T1-weighted images with a blurred high-intensity area on T2-weighted images. In the subacute and chronic stages, a low-intensity area begins to appear on T1-weighted images. The characteristic finding in the chronic stage is usually an oval-shaped area of signal change.[8] On the basis of previous studies on the histopathological features of SCI, the blurred high-intensity area on T2-weighted images is thought to represent edema or petechial hemorrhage. On the other hand, the low-intensity area on T1-weighted images obtained in the subacute and chronic stages is thought to indicate necrosis, myelomalacia or an intramedullary cyst.[5,9]

Some papers have described the early relationship between MRI features and clinical outcomes in patients with CSCI at the acute stage following trauma.[8,10,11] However, to the best of our knowledge, there are few papers reporting about this relationship at subacute or chronic stages following trauma.

In the present study, we evaluated T1- and T2-weighted MRI findings of patients with CSCI in the subacute stages. The purpose was to investigate the relationship between the MRI features and neurological prognosis of patients with CSCI without major bone injury at subacute and chronic stages.

## MATERIALS AND METHODS

Patients with acute CSCI without major bone injury who were admitted to our hospital within 10 days after trauma were included in this study. The patients' imaging (radiographs, CT, MRI) was reviewed when they were admitted to our hospital. Small avulsion fractures of the vertebral body, spinous process fractures or bone bruises in the vertebral body without noticeable vertebral collapse were considered to be minor bony injuries. Any patient who had undergone previous cervical surgery and those who were likely to have recoil flexion injury were excluded, because of the artifacts from surgery implants in MRI. Recoil flexion injury requires fixation. It was difficult to do a correct evaluation in surgery cases. We also excluded patients who had no cord signal changes on MRI to rule out patients with cervical concussion or hysteria or those with a cord compression rate >20%. The rate of spinal cord

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compression was measured on sagittal MRI. The spinal cord diameter was measured at both the non-compressed and injured levels on T1-weighted MRI images, and the percent change was calculated by the following equation:

(antero-posterior diameter of the cord at no compression level − antero-posterior diameter of the cord at compression level)/antero-posterior diameter of the cord at no compression level × 100% (Figure 1). A value of 20% was defined as a cut-off for the spinal cord compression rate based on a previous report.[12]

From January 2005 to May 2010, 72 consecutive patients were included in this study. The mean patient age at the time of trauma was 62.4 years (34–81 years), and there were 60 men and 12 women. Using the American Spinal Injury Association (ASIA) impairment scale, paralysis at the time of admission was graded as A in 4 patients, B in 16, C in 43 and D in 9. The mean period from traumatic injury to admission was 1.5 days (0–10 days), and the mean duration of hospital stay was 240.9 days (35–1423 days). All patients were treated conservatively and rehabilitated as quickly as possible with a simple neck brace.

MRI was performed in all patients at hospital admission (acute stage) and 1 month after trauma (subacute stage). Using sagittal MRI images, an intramedullary high-intensity changed area on T2-weighted MRI images was determined to be the injured level of the cervical spinal cord. We measured the antero-posterior diameter and cranio-caudal diameter of intramedullary intensity changed area on sagittal T1-weighted images (Figure 2). It was difficult to detect the intensity changed area on axial T1-weighted image. The measurement was taken at the largest spinal cord slice. The T1-weighted low-intensity changed area ratio (T1-LCAR) was calculated by the following equation: antero-posterior diameter of low-intensity changed area/spinal cord antero-posterior diameter × 100% (Figure 2).

All measurements were performed by two observers. Each measurement was performed three times, and the average value was calculated. All MR imaging examinations were performed with a 1.5-T magnet (MAGNETOM; Siemens Healthcare, Munich, Bayern, Germany). Standardized MR imaging protocols for the acutely injured spine were used: for sagittal T1-weighted imaging, a two-dimensional spin-echo sequence was performed by using a conventional imaging option with no phase wrap, 525/11 (repetition time msec/echo time msec), a receiver bandwidth of 147 Hz/Px, a matrix of 320 × 224 (frequency encoding × phase encoding), three acquired signals and no phase correction. For sagittal T2-weighted imaging, a two-dimensional fast-recovery fast spin-echo (accelerated) sequence was performed by using imaging options that included the following: no phase wrap, an extended dynamic range, tailored radiofrequency and fast recovery; 3500/99; an echo train length of 15; a receiver bandwidth of 150 Hz/Px; a matrix of 384 _ 229; four acquired signals; and phase correction. Both the T1- and the T2-weighted examinations were performed in the antero-posterior frequency direction by using an Neck Matrix coil, a section thickness of 3.0 mm, an intersection gap of 0.3 mm, a 24-cm field of view and no contrast medium enhancement.

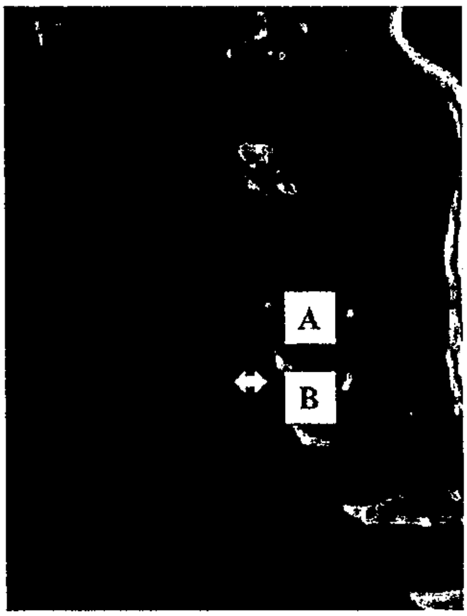


Figure 1 T1-weighted image. Black arrow (A) intact cervical spinal cord. White arrow (B) cervical spinal cord compressed by a disc and ligament flavum. Compression rate was 34%.

The ASIA impairment scale and the ASIA motor score (ranging from 0 to 100) were documented at admission and discharge for each patient. Neurologic recovery was evaluated as the following:

Improvement rate (%) = ((motor score at discharge − motor score at admission)/(100 − motor score at admission)) × 100.[13]

All neurological evaluations were performed by senior spinal surgeons. In addition, we also documented each patient's score with the modified Frankel grading system (Table 1).[14,15]

All data were analyzed using the JMP 8.0.2 software program (SAS Institute, Cary, NC, USA). The relationships between T1-LCAR and the ASIA motor scores and the recovery rate were analyzed using the Spearman rank-correlation coefficient. $P<0.05$ was considered statistically significant. The relationships between cranio-caudal diameter and the ASIA motor scores were also analyzed using the Spearman rank-correlation coefficient. The chance-corrected κ-coefficient was calculated to determine intra-observer agreement. Intra-observer reliability was almost good ($\kappa=0.92$, $P<0.001$).

## RESULTS

The neurologic statuses as evaluated by the ASIA and modified Frankel grading scales at admission and discharge are summarized in Tables 2 and 3. None of the patients demonstrated neurological deterioration during follow-up, and 60 (81.9%) and 68 (94.4%) demonstrated neurological recovery as evaluated by ASIA and modified Frankel grading, respectively.

The mean ASIA motor scores at admission and discharge were $37.3\pm29.2$ and $78.3\pm22.8$, respectively. The mean improvement rate of the motor score was $68.9\pm24.7\%$, which was significant.

T1-weighted low-intensity changed areas were observed in 53 cases (73.6%) at 1 month after injury. A total of 16 patients with final ASIA D and 3 patients with final ASIA E did not demonstrate intramedullary low-intensity change on T1-weighted MRI at the time of discharge. The T1-LCAR of these 19 patients was set as 0%. The correlation between the T1-CAR and the ASIA motor score at discharge and the improvement rate were examined in all 72 patients, as shown in Figures 3 and 4. There was a significant negative correlation between T1-LCAR and ASIA motor score at discharge ($P<0.0001$, Figure 3). There was also a significant negative correlation

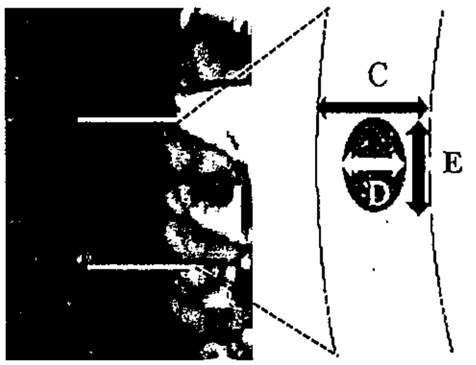


Figure 2 T1-weighted image. Black arrow (C) cervical spinal cord antero-posterior diameter. White arrow (D) T1-low antero-posterior diameter. T1-low diameter ratio was D/C(%). Gray arrow (E) T1-low cranio-caudal diameter (mm).

**Table 1** The modified Frankel grading system

| *Grade* | *Neurological status* |
|---|---|
| A | Complete: no motor or sensory function |
| B | Sensory only: some sensation preserved, no motor function |
| B1 | Touch sensation remains in only sacral lesion |
| B2 | Touch sensation remains in L/E |
| B3 | Pain sensation remains in sacral lesion or L/E |
| C | Motor useless: some sensory and motor function, but motor function not useful |
| C1 | Unable to flex the hip and knee from supine (Hip flexors 0–2) |
| C2 | Able to flex the hip and knee from supine (Hip flexors 3–5) |
| D | Motor useful: sensory function preserved, moter function weak but useful |
| D0 | MMTs of L/E are 4–5, but because of an acute phase, it is impossible to test the walking ability |
| D1 | Able to walk with a walker, but not practiced, usually use a wheel chair |
| D2 | Independent gait with a cane |
| D3 | Independent gait without a cane |
| E | Normal: normal sensory and motor function (hyperreflexia and numbness are permitted) |

**Table 2** Diagram showing change in American Spinal Injury Association (ASIA) impairment scale between the status at admission (vertical axis) and at discharge (horizontal axis)

| discharge / admission | A | B | C | D | E |
|---|---|---|---|---|---|
| A | 1 | 0 | 3 | 0 | 0 |
| B | 0 | 3 | 3 | 10 | 0 |
| C | 0 | 0 | 2 | 39 | 2 |
| D | 0 | 0 | 0 | 7 | 2 |

between T1-LCAR and the recovery rate ($P=0.0072$, Figure 4). On the other hand, there was no correlation between cranio-caudal diameter of T1 low-changed area and the ASIA motor score at discharge ($P=0.2797$). There was also no correlation between cranio-caudal diameter of T1 low-changed area and the recovery rate ($P=0.2184$).

A total of 60 patients (83.3%) recovered to walk with or without a cane (higher or equal Frankel D). Receiver operator characteristic curve analysis demonstrated that the optimal T1-LCAR cut-off value for patients who were able to walk at discharge was 46% (Figure 5). If the T1-LCAR cut-off value was <50%, there was a significant positive correlation with being able to walk at discharge ($P<0.0001$, Pearson's $\chi^2$ test, Table 4).

## DISCUSSION

The reported incidence of CSCI without major bone injury ranges from ~10 to 16% of all cervical cord injury in North America and India,[6,16] but they are the largest proportion of cervical cord injury in Japan as described by Koyanagi *et al.*[16] The number of CSCI without major bone injury has been increasing as the population ages; data from our institution showed an annual rate of 38.2% in 1990 and 63.2% in 2005.[17] CSCIs without major bone injury have consistently

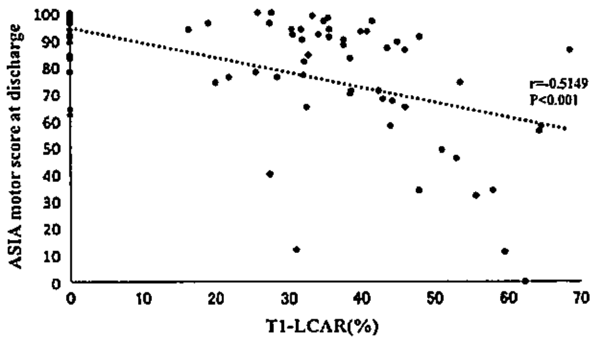


**Figure 3** The relationship between the ASIA motor score at discharge and the T1-LCAR. There was a significant negative relationship between the two parameters, indicating that a larger T1-LCAR was associated with more severe paralysis.

**Table 3** Diagram showing change in modified Frankel grade between the status at admission (vertical axis) and at discharge (horizontal axis)

| discharge / admission | A | B1 | B2 | B3 | C1 | C2 | D1 | D2 | D3 | E |
|---|---|---|---|---|---|---|---|---|---|---|
| A | 1 | 0 | 0 | 0 | 2 | 1 | 0 | 0 | 0 | 0 |
| B1 | 0 | 0 | 2 | 0 | 1 | 0 | 2 | 2 | 0 | 0 |
| B2 | 0 | 0 | 2 | 0 | 1 | 0 | 1 | 0 | 0 | 0 |
| B3 | 0 | 0 | 0 | 0 | 1 | 0 | 2 | 2 | 1 | 0 |
| C1 | 0 | 0 | 0 | 0 | 1 | 1 | 9 | 7 | 7 | 0 |
| C2 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 7 | 4 | 0 |
| D0 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | 11 | 3 |

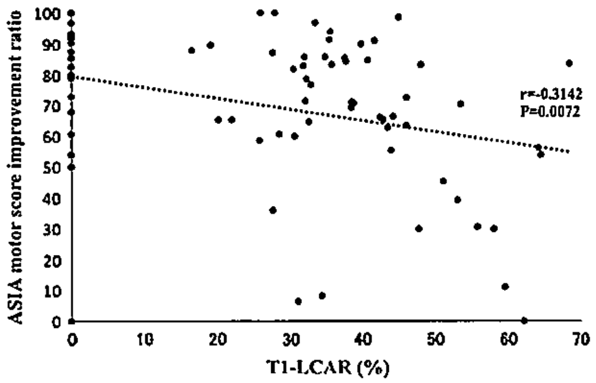


Figure 4 The relationship between the ASIA motor score improvement ratio at discharge and the T1-LCAR. There was a significant negative relationship between the two parameters, indicating that a larger T1-LCAR was associated with poor improvement.

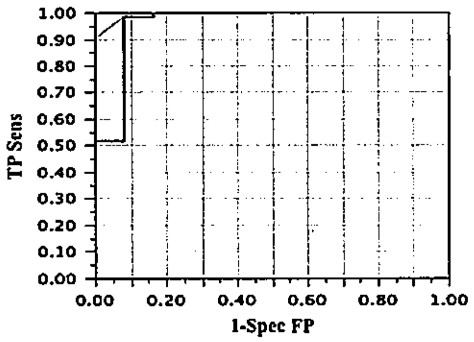


Figure 5 ROC curve of higher or equal Frankel grading D and T1-LCAR at 1 month after injury. AUC=0.95833. FP, false positive; Sens, sensitivity; Spec, specificity; TP, true positive.

been treated conservatively and rehabilitated as early as possible at our institution.

MRI is being increasingly used in the evaluation of post-traumatic myelopathy. It is useful in that it allows imaging of the injured cord, as well as an ability to predict patient outcome. The most common acute MRI pattern for CSCI is a blurred high-intensity area on T2-weighted images and no change in signal intensity on T1-weighted images. The subacute MRI pattern is iso-intensity or high-intensity on T2-weighted images, but a circumscribed low-intensity area is noted on T1-weighted images. This low-intensity area appears 3 to 6 weeks after injury.[8] On the basis of previous studies of the histopathological features of SCI, the blurred high-intensity area on T2-weighted MRI is thought to represent edema or petechial hemorrhage. On the other hand, the low-intensity area on T1-weighted MRI in the subacute and chronic stages is thought to indicate necrosis or myelomalacia.[5,9] We thought that the T1-weighted low-intensity area on MRI at the subacute stage reflected the damage area of spinal cord. It was difficult to measure accurately the changed area of the spinal cord by edema in T2-weighted image. As there was no edema change in T1-weighted image, we could measure accurately the changed area of spinal cord.

Shimada and Tokioka[18] identified four distinct patterns of MR signal intensity changes that correlated well with spinal cord damage severity and clinical outcome. Ishida and Tominaga[19] assessed predictors of neurologic recovery in patients with acute central cervical SCI and found that an absence of MR signal intensity in the spinal

**Table 4 T1-LCAR and modified Frankel grade at discharge. If the cut-off point for the T1-LCAR was <50%, a significant positive correlation was found between T1-LCAR <50% and the patients who recovered to ASIA D or higher or to modified Frankel D or higher (*P*<0.0001)**

| | T1-LCAR<50% | T1-LCAR>50% |
|---|---|---|
| Lower Frankel D | 2 | 10 |
| Higher or Equal Frankel D | 60 | 0 |

cord and good early neurologic improvement were important predictors of long-term neurologic function improvement. However, no studies have reported on T1-weighted low-intensity changed area in CSCI patients. We hypothesized that the T1-weighted low-intensity area on MRI at the subacute stage of injury was predictive of neurological outcome. Indeed, we identified a significant negative correlation between the T1-LCAR and the ASIA motor score at discharge. There was also a significant negative correlation between the T1-LCAR and the recovery rate. These results suggest that there might be significant correlation between the T1-weighted low-intensity changed area on MRI and post-traumatic neurological outcome. If the T1-LCAR was <50%, the patients achieved a neurological status higher or equal ASIA D or modified Frankel D. These patients were able to walk with or without a cane at discharge.

In this study, we researched the relationship between the outcome and the MRI 1 month after injury. All patients who did not have low-intensity area 1 month after injury were able to walk at discharge. Two months after injury, the low-changed area might be getting larger. However, this is the relationship between the outcome and the MRI 1 month after injury. MRI 1 month after injury reflects the patient's ability to walk.

This study had several limitations; it was retrospective, and the number of patients was small. Further research with larger patient populations and prospective evaluation may help resolve the questions raised in this study. In this study, the relationship between the imaging immediately after injury and the outcome was not performed. We did not consider the relationship between the outcome and the transverse diameter, cross-sectional area and volume of signal alteration, which will be considered in future study. Moreover, the etiology of CSCI without major bone injury should be studied in more detail.

In conclusion, we identified a significant relationship between T1-weighted low-intensity areas on T1-weighted MRI 1 month following injury and neurological recovery prognosis at discharge. Low-intensity changed area on T1-weighted MRI may be an important predictive factor in the natural course of neurological recovery for CSCI.

## DATA ARCHIVING

There were no data to deposit.

## CONFLICT OF INTEREST

The authors declare no conflict of interest.

1 Gupta SK, Rajeev K, Khosla VK, Sharma BS, Paramjit, Mathuriya SN *et al.* Spinal cord injury without radiographic abnormalities in adult. *Spinal Cord* 1999; **37**: 726–729.
2 Shen H, Tang Y, Huang L, Yang R, Wu Y, Wang P *et al.* Applications of diffusion-weighted MRI in thoracic spinal cord injury without radiographic abnormality. *Int Orthop* 2007; **31**: 375–383.
3 Pang D, Wilberger JE. Spinal cord injury without radiographic abnormalities in children. *J Neurosurg* 1982; **57**: 114–129.
4 Bhatoe HS. Cervical spinal cord injury without radiological abnormality in adults. *Neurol India* 2000; **48**: 243–248.
5 Ohshio I, Hatayama A, Kaneda K, Takahara M, Nagashima K. Correlation between histopathologic features and magnetic resonance images of spinal cord lesions. *Spine* 1993; **18**: 1140–1149.
6 Tewari MK, Gifti DS, Singh P, Khosla VK, Mathuriya SN, Gupta SK *et al.* Diagnosis and prognostication of adult spinal cord injury without radiographic abnormality using magnetic resonance imaging: analysis of 40 patients. *Surg Neurol* 2005; **63**: 204–209.
7 Kasimatis GB, Panagiotopoulos E, Megas P, Matzaroglou C, Gliatis J, Tyllianakis M *et al.* The adult spinal cord injury without radiographic abnormalities syndrome: magnetic resonance imaging and clinical findings in adults with spinal cord injuries having normal radiographs and computed tomography studies. *J Trauma* 2008; **65**: 86–93.
8 Takahashi M, Harada Y, Inoue H, Shimada K. Traumatic cervical cord injury at C3-4 without radiographic abnormalities: correlation of magnetic resonance findings with clinical features and outcome. *J Orthop Surg* 2002; **10**: 129–135.
9 Weirich SD, Cotler HB, Narayana PA, Hazle JD, Jackson EF, Coupe KJ *et al.* Histopathologic correlation of magnetic resonance imaging signal patterns in a spinal cord injury model. *Spine* 1990; **15**: 630–638.
10 Machino M, Yukawa Y, Ito K, Nakashima H, Kanbara S, Morita D *et al.* Can magnetic resonance imaging reflect the prognosis in patients of cervical spinal cord injury without radiographic abnormality? *Spine* 2011; **36**: E1568–E1572.
11 Miyanji F, Furlan JC, Aarabi B, Arnold PM, Fehlings MG. Acute cervical traumatic spinal cord injury: MR imaging findings correlated with neurologic outcome–prospective study with 100 consecutive patients. *Radiology* 2007; **243**: 820–827.
12 Kawano O, Ueta T, Shiba K, Iwamoto Y. Outcome of decompression surgery for cervical spinal cord injury without bone and disc injury in patients with spinal cord compression: a multicenter prospective study. *Spinal Cord* 2010; **48**: 548–553.
13 Ishida Y, Tominaga T. Predictors of neurologic recovery in acute central cervical cord injury with only upper extremity impairment. *Spine* 2002; **27**: 1652–1658.
14 Frankel HL, Hancock DO, Hyslop G, Melzak J, Michaelis LS, Ungar GH *et al.* The value of postural reduction in the initial management of closed injuries of the spine with paraplegia and tetraplegia. *I Paraplegia* 1969; **7**: 179–192.
15 Hayashi T, Kawano O, Sakai H, Ideta R, Ueta T, Maeda T *et al.* The potential for functional recovery of upper extremity function following cervical spinal cord injury without major bone injury. *Spinal Cord* 2013; **51**: 819–822.
16 Koyanagi I, Iwasaki Y, Hida K, Akino M, Imamura H, Abe H. Acute cervical cord injury without fracture or dislocation of the spinal column. *J Neurosurg* 2000; **93**: 15–20.
17 Okada S, Maeda T, Ohkawa Y, Harimaya K, Saiwai H, Kumamaru H *et al.* Does ossification of the posterior longitudinal ligament affect the neurological outcome after traumatic cervical cord injury? *Spine* 2009; **34**: 1148–1152.
18 Shimada K, Tokioka T. Sequential MR studies of cervical cord injury: correlation with neurological damage and clinical outcome. *Spinal Cord* 1999; **37**: 410–415.
19 Ishida Y, Tominaga T. Predictors of neurologic recovery in acute central cervical cord injury with only upper extremity impairment. *Spine* 2002; **27**: 1652–1658.

Authors: Please review all authors' names, academic degrees, affiliations, and contributions, as well as the Disclosure, for spelling and accuracy.

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JNS SPINE

CLINICAL ARTICLE

# Effect of preservation of the C-6 spinous process and its paraspinal muscular attachment on the prevention of postoperative axial neck pain in C3–6 laminoplasty

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**OBJECT** Axial neck pain after C3–6 laminoplasty has been reported to be significantly lesser than that after C3–7 laminoplasty because of the preservation of the C-7 spinous process and the attachment of nuchal muscles such as the trapezius and rhomboideus minor, which are connected to the scapula. The C-6 spinous process is the second longest spinous process after that of C-7, and it serves as an attachment point for these muscles. The effect of preserving the C-6 spinous process and its muscular attachment, in addition to preservation of the C-7 spinous process, on the prevention of axial neck pain is not well understood. The purpose of the current study was to clarify whether preservation of the paraspinal muscles of the C-6 spinous process reduces postoperative axial neck pain compared to that after using nonpreservation techniques.

**METHODS** The authors studied 60 patients who underwent C3–6 double-door laminoplasty for the treatment of cervical spondylotic myelopathy or cervical ossification of the posterior longitudinal ligament; the minimum follow-up period was 1 year. Twenty-five patients underwent a C-6 paraspinal muscle preservation technique, and 35 underwent a C-6 nonpreservation technique. A visual analog scale (VAS) and VAS grading (Grades I–IV) were used to assess axial neck pain 1–3 months after surgery and at the final follow-up examination. Axial neck pain was classified as being 1 of 5 types, and its location was divided into 5 areas. The potential correlation between the C-6/C-7 spinous process length ratio and axial neck pain was examined.

**RESULTS** The mean VAS scores (± SD) for axial neck pain were comparable between the C6-preservation group and the C6-nonpreservation group in both the early and late postoperative stages (4.1 ± 3.1 vs 4.0 ± 3.2 and 3.8 ± 2.9 vs 3.6 ± 3.0, respectively). The distribution of VAS grades was comparable in the 2 groups in both postoperative stages. Stiffness was the most prevalent complaint in both groups (64.0% and 54.5%, respectively), and the suprascapular region was the most common site in both groups (60.0% and 57.1%, respectively). The types and locations of axial neck pain were also similar between the groups. The C-6/C-7 spinous process length ratios were similar in the groups, and they did not correlate with axial neck pain. The reductions of range of motion and changes in sagittal alignment after surgery were also similar.

**CONCLUSIONS** The C-6 paraspinal muscle preservation technique was not superior to the C6-nonpreservation technique for preventing postoperative axial neck pain.

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**KEY WORDS** laminoplasty; axial neck pain; paraspinal muscle; cervical spine; spinous process; trapezius muscle

Laminoplasty, which evolved from extensive laminectomy in the treatment of cervical spinal cord compression, is an established posterior cervical decompression procedure for multisegmental cervical myelopathy.[5,9,20] Although acceptable neurological improvement after laminoplasty is achieved, postoperative complications or problems such as axial neck pain, reduction of cervical range of motion (ROM), and changes in spinal alignment are often induced.[1,3,7,12] Axial neck pain, typified by persistent pain around the neck and shoulder, is a notorious postoperative complication after laminoplasty.[8,12,18] The incidence of axial neck pain has been reported

**ABBREVIATIONS** CSM = cervical spondylotic myelopathy; JOA = Japanese Orthopaedic Association; OPLL = ossification of the posterior longitudinal ligament; ROM = range of motion; VAS = visual analog scale.

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**DISCLOSURE** The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

to range from 5% to 80%.[6,13,18] Several techniques have been developed by surgeons who attempted to reduce axial neck pain after laminoplasty.[14,17,21] In a comparative study between C3–6 and C3–7 laminoplasty, Hosono et al.[6] reported that postoperative axial neck pain was significantly less severe after C3–6 laminoplasty than after C3–7 laminoplasty. They suggested that preservation of the C-7 spinous process and the origin of the trapezius and rhomboid minor muscles on the C-7 spinous process was key to preventing postoperative axial neck pain. The C-7 spinous process is an important muscular attachment point for the trapezius and rhomboid minor muscles connecting to the scapula. They hypothesized that disruption of the musculotendinous connection between the C-7 spinous process and the scapula was one of the causes of postoperative axial neck pain. The C-6 spinous process is the second longest spinous process after the C-7 process, and it also serves as an attachment point for the rhomboid minor and the speculum rhomboideum section of the trapezius, which is composed of the middle fibers of the trapezius and contains a very strong tendinous component.[16] Therefore, we postulated that preservation of the paraspinal muscles of the C-6 spinous process in addition to the C-7 spinous process might decrease postoperative axial neck pain. The relationship between preservation of the paraspinal muscles of the C-6 spinous process and axial neck pain is not well understood. The purpose of the current study was to clarify whether preservation of the paraspinal muscles of the C-6 spinous process reduces postoperative axial neck pain.

## Methods

### Patient Population

In total, 81 patients with no history of cervical surgery underwent a C3–6 spinous process–splitting double-door laminoplasty for the treatment of cervical spondylotic myelopathy (CSM) or cervical ossification of the posterior longitudinal ligament (OPLL) between January 2009 and December 2010. Thirty-three patients were treated with a paraspinal muscle preservation technique at the C-6 level (C6-preservation group), and 48 were treated with a paraspinal muscle nonpreservation technique at the C-6 level (C6-nonpreservation group). A surgeon (I.Y.) who was interested in preservation techniques performed the C6-preservation laminoplasties, and others (T.U. and T.M.) performed the C6-nonpreservation laminoplasties, our conventional technique. Although selection of the surgeon and the patients was not randomized in the current study, the patients were not informed about the C-6 spinous process preservation procedure. Of the patients, 60 were followed up and examined for more than 1 year. The ethics committee of our institution approved the study. Of the 60 patients, 44 were men, and the mean age of the group was 66.5 years (range 47–81 years). Forty-three patients had CSM and 17 had cervical OPLL. The mean follow-up durations were 3 years 1 month (range 1.8–3.8 years) in the C6-preservation group and 2 years 10 months (range 1.5–3.8 years) in the C6-nonpreservation group. Twenty-five patients underwent C6-preservation laminoplasty, and 35 underwent C6-nonpreservation laminoplasty. No significant differences in age, sex, or diagnosis were observed between the 2 groups (Table 1).

### Surgical Procedures

In both of the groups, a midline skin incision was made from the C-2 spinous process to the C-7 spinous process. The nuchal ligament was incised in the midline. Then, the incision was continued between the bilateral splenius capitis and semispinalis capitis down to the spinous processes from C-3 to C-6. All the bilateral muscles attached to the C-2 and C-7 spinous processes were preserved by the procedures performed in both groups.

In the C6-preservation procedure, the paraspinal muscles were dissected, detached from the C-3 to C-5 posterior aspect while the attachment points of the paraspinal muscles such as rotators, multifidus, semispinalis cervicis, rhomboid minor, and the speculum rhomboideum of the trapezius (if attached to the spinous processes of C-6) were left intact, and retracted to ensure exposure of the laminae and the medial border of the facet joints. The epidural space was exposed after opening the C-3 split spinous process using a surgical bur, with lateral gutters at the medial border of the facet joint bilaterally. After midline blunt dissection with cutting of the interspinous process ligament between the C-6 and C-7 spinous processes, the epidural space was minimally exposed. The attachments of the paraspinal muscles to the C-6 and C-7 spinous processes were left intact. The C-4, C-5, and C-6 spinous processes were split longitudinally by a thread saw passed through the epidural space from C-3 to C-6/C-7, and lateral gutters were made bilaterally. At the C-6 level, bilateral gutters were made after minimal exposure of the medial border of the facet joint by retracting the paraspinal muscles from the lamina with a Penfield elevator while leaving the muscular attachment to the spinous process undisturbed. Bilateral halves of split spinous processes and laminae were lifted and bilaterally opened. A ceramic spacer was placed between the split spinous processes at each level (Fig. 1).

In the C6-nonpreservation procedure, the paraspinal muscles were dissected and detached thoroughly from the C-3 to C-6 posterior aspect. Longitudinal splitting of the spinous process from C-3 to C-6 was performed with a thread saw passed through the epidural space from C-6/C-7 to C-2/C-3. The procedures for opening the split

**TABLE 1. Patient characteristics and surgical invasion**

| Characteristic | C6-Preservation Group (n = 25) | C6-Nonpreservation Group (n = 35) | p Value |
|---|---|---|---|
| Mean age in yrs (range) | 66.1 (47–79) | 66.7 (48–81) | NS |
| Sex (male/female)* | 19:6 | 25:10 | NS |
| CSM* | 17 | 26 | NS |
| OPLL* | 8 | 9 | |
| Mean op time (mins)† | 132.5 ± 34.2 | 81.8 ± 20.9 | <0.0001 |
| mean EBL (g)† | 60.2 ± 41.7 | 40.6 ± 32.0 | <0.05 |

EBL = estimated blood loss; NS = not significant.
* Values are the number of patients.
† Values are mean ± SD.

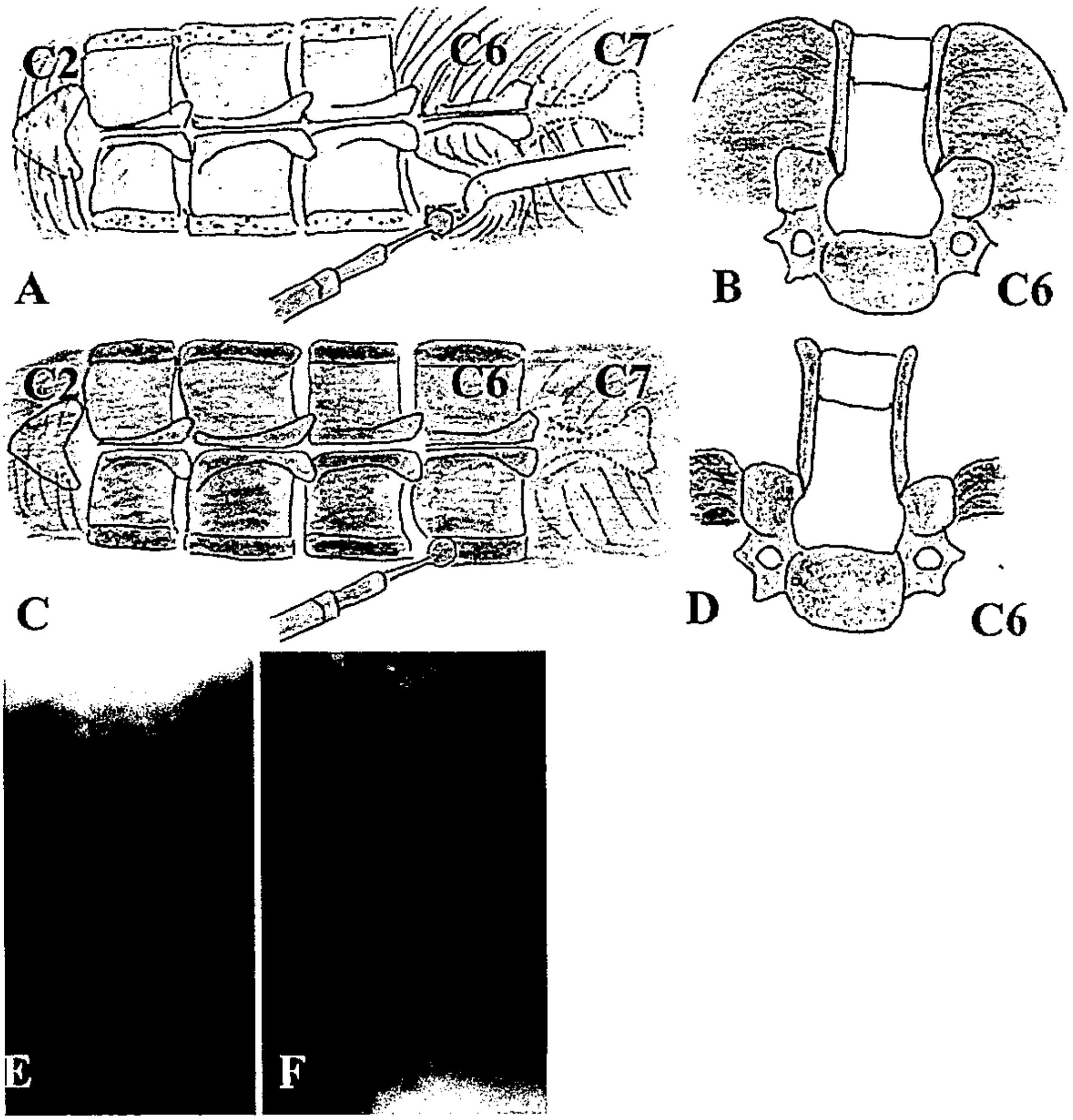


FIG. 1. **A and B:** In the C6-preservation procedure, the paraspinal muscles were dissected and detached from the C3–5 posterior aspects. The C3–6 spinous processes were cut longitudinally at the midline using a thread saw, and the attachments of the paraspinal muscles, such as rotators, multifidus, semispinalis cervicis, rhomboid minor, and the speculum rhomboideum of the trapezius, if it was attached, to the spinous processes of C-6 were left intact. At the C6 level, bilateral gutters were made after minimal exposure of the medial border of the facet joint by retracting the paraspinal muscles from the lamina with a Penfield elevator while leaving the muscular attachment to the spinous process undisturbed. **C and D:** In the C6-nonpreservation procedure, the paraspinal muscles were dissected and detached thoroughly from the C3–5 and C-6 posterior aspects. **E and F:** Anteroposterior and lateral radiographs after the C6-preservation procedure. Copyright (A–D) Eiji Mori. Published with permission.

spinous processes and placement of ceramic spacers were the same as those described for the C6-preservation procedure (Fig. 1).

Each patient in both groups wore a cervical orthosis for 10 days after surgery.

### Clinical Examination

A 10-point visual analog scale (VAS) and VAS grading were used to assess axial neck pain 1–3 months after surgery and at the final follow-up observation. Pain around the neck and shoulder persisting for more than 1 week in the 1–3 months after surgery or for more than 1 month after surgery was considered postoperative axial neck pain. Patients were asked to mark their level of pain under the condition of no analgesics. The VAS grades were defined as follows: Grade I, 0–2.5 points; Grade II, 2.6–5.0 points; Grade III, 5.1–7.5 points; and Grade IV, 7.6–10.0 points. Axial neck pain was classified as 1 of 5 types as follows: pain, stiffness, tension, tightness, or traction. The axial neck pain areas of distribution were defined as follows: nuchal region, suprascapular region, superior angle of the scapular region, scapular region, and interscapular region (Fig. 2).

The type(s) and area(s) of distribution of axial neck pain (patients were allowed to select multiple types and/or areas) were examined and defined as positive when patients experienced pain 1–3 months after surgery or at the final follow-up observation. The examination for axial neck pain was performed by a clerical nonsurgical staff member who was blinded to the patients' surgical status. The correlation between the ratio of the length of the C-6 spinous process to that of the C-7 spinous process (C-6/C-7 spinous process length ratio) and the VAS score for axial neck pain was examined after surgery or at the final follow-up. These items concerning axial neck pain were compared between the C6-preservation and C6-nonpreservation groups. Surgical invasion was also compared between the groups. The ROM and sagittal alignment from C-2 to C-7 were examined before and after surgery and compared between the 2 groups. Neurological function was assessed by using the Japanese Orthopaedic Association (JOA) scale (full score 17), which was administered by a doctor who was blinded to the patients' surgical status.

### Statistical Analysis

The clinical parameters of the 2 groups were compared using the Mann-Whitney U-test and the Fisher exact test. The correlation between the C-6/C-7 spinous process length ratio of each patient and his or her VAS score for axial pain was analyzed using Pearson's correlation coefficient. Statistical analysis was performed using the JMP program (version 8; SAS Institute Japan). p values of < 0.05 were considered statistically significant. Means are presented ± SD.

## Results

The mean surgical time was significantly longer and the estimated blood loss was significantly greater in the C6-preservation group than in the C6-nonpreservation group (Table 1).

### VAS Scores for Axial Neck Pain and VAS Grades

The mean VAS score for axial neck pain in the C6-preservation group was equivalent to that in the C6-nonpreservation group 1–3 months after surgery (4.1 ± 3.1 vs 4.0 ± 3.2, respectively). Moreover, the mean VAS scores for axial neck pain were comparable between the 2 groups at the final follow-up observation (3.8 ± 2.9 vs 3.6 ± 3.0, respectively) (Fig. 3). The VAS grade distributions were comparable between the groups at both 1–3 months after surgery and the final follow-up observation (Fig. 4).

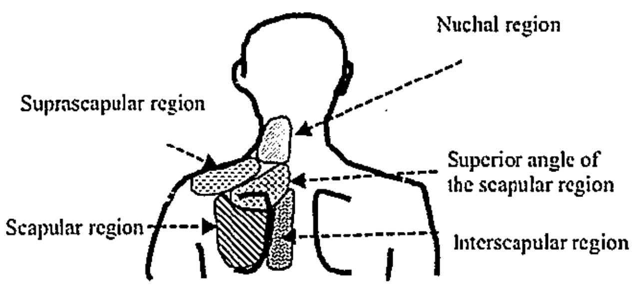


FIG. 2. Locations of axial neck pain (5 separate regions).

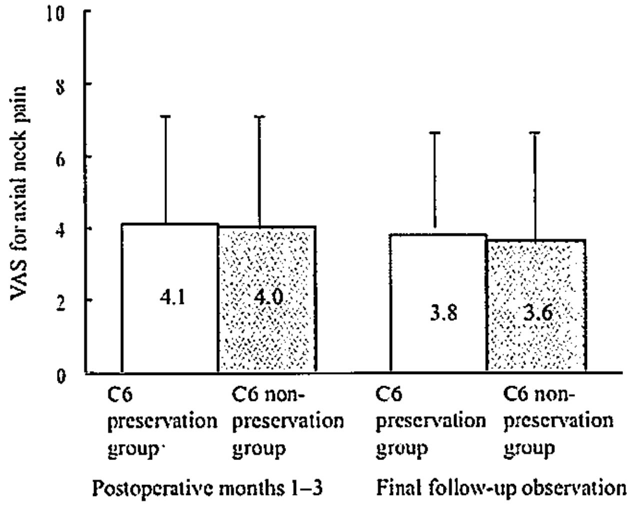


FIG. 3. VAS scores for axial neck pain in the C6-preservation and C6-nonpreservation groups 1–3 months after surgery and at the final follow-up observation. The *white columns* denote the C6-preservation group, and the *gray columns* denote the C6-nonpreservation group. There were no differences in the mean VAS scores of the 2 groups at either postoperative stage.

### Type of Axial Neck Pain

Among the types of axial neck pain, stiffness was the most prevalent complaint in both the C6-preservation and C6-nonpreservation groups (64.0% and 54.5%, respectively). Subsequently, pain was prevalent in both groups (40.0% and 39.4%, respectively). The prevalences of the types of axial neck pain were comparable between the groups for all 5 types (Fig. 5).

### Location of Axial Neck Pain

Among the locations of axial neck pain, the suprascapular region was the most common site in both the C6-preservation and C6-nonpreservation groups (60.0% and 57.1%, respectively). Subsequently, the nuchal region was commonly affected in both groups (48.0% and 45.7%, respectively). The prevalences of axial neck pain were comparable between the groups at all 5 regions (Table 2).

### C-6/C-7 Spinous Process Length Ratio and Axial Neck Pain

The mean C-6/C-7 spinous process length ratios were comparable between the C6-preservation and C6-nonpreservation groups (76.9 ± 9.9 vs 78.0 ± 11.2, respectively). The C-6/C-7 spinous process length ratios of the patients were not significantly correlated with the VAS scores for axial neck pain either 1–3 months after surgery or at the final follow-up in the C6-preservation group (Fig. 6). Similarly, no significant correlation between the C-6/C-7 spinous process length ratios and the VAS scores for axial neck pain was observed for either postoperative stage in the C6-nonpreservation group (Fig. 7).

### Radiographic and Neurological Outcomes

The reductions of ROM at the final follow-up visit, which were greater in the subgroup of patients with OPLL than in

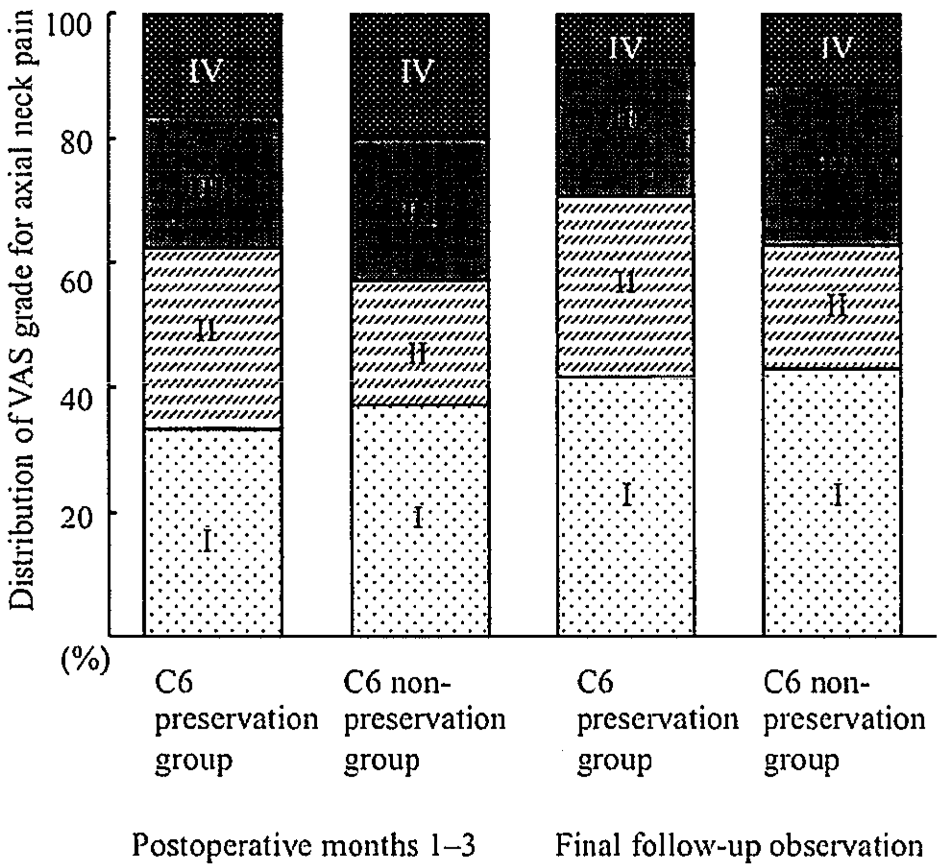


FIG. 4. The distribution of VAS grades for axial neck pain in the C6-preservation and C6-nonpreservation groups 1–3 months after surgery and at the final follow-up observation. There were no differences in the distributions of VAS grades between the 2 groups for either postoperative stage.

the subgroup of patients with CSM, were comparable between the C6-preservation and C6-nonpreservation groups in both of these subgroups (Table 3). Neither fracture of the lamina nor dislodgement of a spacer was observed in either of the 2 groups in the follow-up period. The sagittal alignment conversion of preoperative lordosis to postoperative kyphosis was observed in only 2 patients in the C6-nonpreservation group. A loss of C2–7 lordosis of > 10° was observed in 2 patients (8.0%) in the C6-preservation group and in 3 patients (8.6%) in the C6-nonpreservation group. The JOA score and its recovery rate were not significantly different between the 2 groups (Table 4). The mean hospital stay was 43.7 ± 21.3 days in the C6-preservation group and 42.5 ± 16.0 days in the C6-nonpreservation group. The patients participated in a rehabilitation program after surgery during their stay in the hospital. Therefore, they were not discharged soon after their surgery. In the C6-preservation group, 3 patients (12%) experienced dysesthesia in the upper extremities after surgery. These unpleasant symptoms were relieved in a few weeks with conservative treatment. One patient had cerebrospinal fluid pooling in the surgical site, which was identified after surgery and was associated with headache and nausea. He was treated conservatively and successfully without neurological worsening. Another patient had night delirium that lasted only a few nights. In the C6-nonpreservation group, there were 5 patients (14.3%) who experienced postsurgical dysesthesia in the upper extremities, which was relieved with conservative treatment in a few weeks. One patient was diagnosed with pneumonia after surgery and was treated successfully with antibiotics. One patient with an electrolyte abnormality and the patient with night delirium were managed conservatively. It was fortunate that neither neurological worsening (including C-5 paresis) nor any other serious complications, such as surgical site infection, nerve injury, or hematoma, occurred in either group.

## Discussion

Postoperative axial neck pain typified by persistent pain around the neck and shoulders is a common problem associated with cervical laminoplasty.[7,8,12,18] There have been wide variations in the incidence of axial neck pain after various types of laminoplasty techniques.[6,13,18] Several posterior cervical decompression techniques, such as skip laminectomy,[21] segmental partial laminectomy,[17] and muscle-preserving laminoplasty,[14] have been introduced in an attempt to reduce postoperative complications, including axial neck pain. In terms of preventing axial neck pain, the results were not always favorable.[22,23] Although the exact cause of axial neck pain has not been detected, intraoperative invasion to the nuchal muscles is presumed

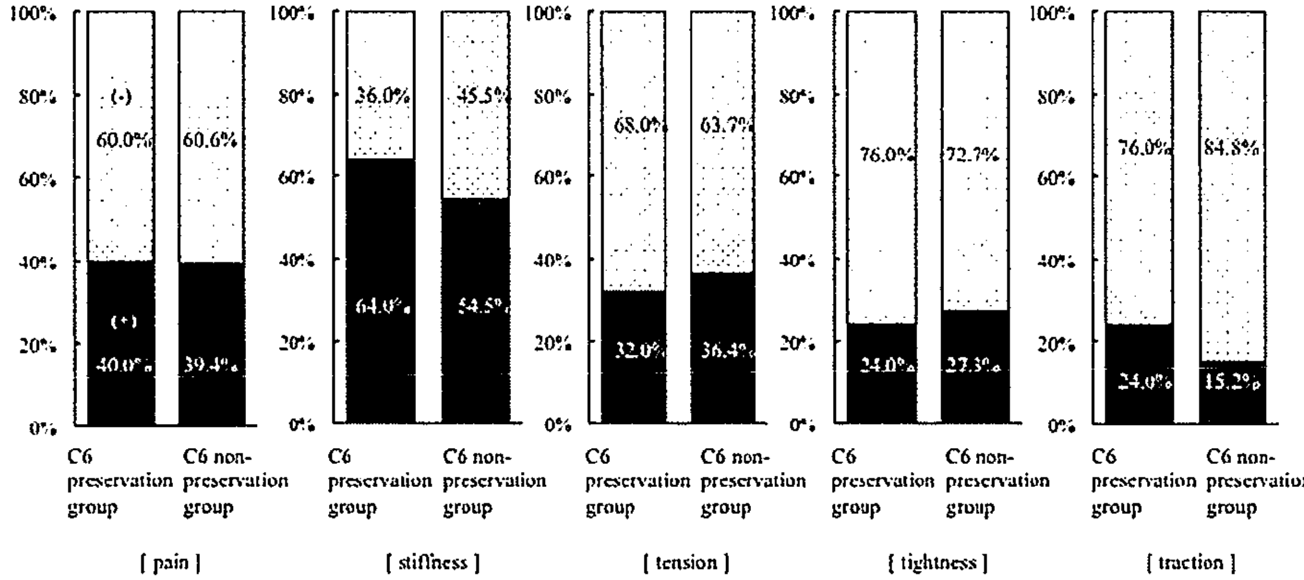


FIG. 5. Types of axial neck pain. The *dark columns* denote positivity, and the *spotted columns* denote negativity concerning the type of pain. Stiffness was the most common complaint in both groups, and pain was the second most prevalent complaint. The prevalences of axial neck pain were comparable between the 2 groups for all 5 types.

to be involved in this complication.[14,17,21] Nuchal muscles, such as the trapezius, rhomboid minor, splenius capitis, and serratus posterior superior muscles, are more densely and closely bound to the C-7 spinous process than other upper or middle cervical vertebrae.[11] Moreover, the maximum force generated by the trapezius has been reported among the cervical vertebrae to be the greatest at C-7.[10] In clinical research, the authors of one study reported that axial pain was induced after upper thoracic surgery and required detachment and spreading of the spinous process or vertebral arch at the cervicothoracic junction; these results were in contrast to those after middle or lower thoracic surgery, which was associated with no strong postoperative pain around the surgical wound.[19] These findings suggest that the connection of the nuchal muscles with the C-7 spinous process is very important and related to the pathology of postoperative axial neck pain. Focusing on preserving the large spinous process of C-7 as the important origin of the nuchal muscles, Hosono et al.[6] conducted a comparative study of conventional C3–7 laminoplasty and C3–6 laminoplasty by limiting the range of decompression with preservation of the C7 spinous process. They demonstrated the superiority of C3–6 laminoplasty over C3–7 laminoplasty in regard to axial neck pain; the incidence of postoperative axial neck pain was only 5.4% after C3–6 laminoplasty versus 29% after the C3–7 procedure. They emphasized the importance of preserving the C--7

TABLE 2. Prevalence of axial neck pain at each of the 5 locations*

| Region | C6-Preservation Group (n = 25) | C6-Nonpreservation Group (n = 35) | p Value |
|---|---|---|---|
| Nuchal | 12 (48.0) | 16 (45.7) | NS |
| Suprascapular | 15 (60.0) | 20 (57.1) | NS |
| Superior angle of the scapula | 4 (16.0) | 7 (20.0) | NS |
| Scapular | 1 (4.0) | 3 (8.6) | NS |
| Interscapular | 1 (4.0) | 4 (11.4) | NS |

* Values represent number (%) of patients.

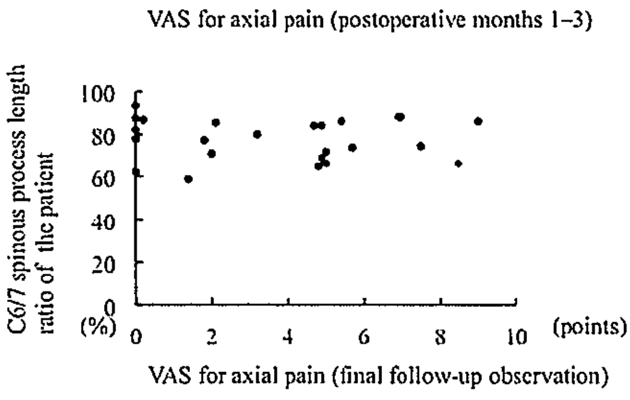


FIG. 6. Correlation between the C-6/C-7 spinous process length ratios of the patients and the VAS scores for axial neck pain in the C6-preservation group. The C-6/C-7 spinous process length ratios were not significantly correlated with the VAS scores for axial neck pain either 1–3 months after surgery *(upper)* or at the final follow-up observation *(lower)*.

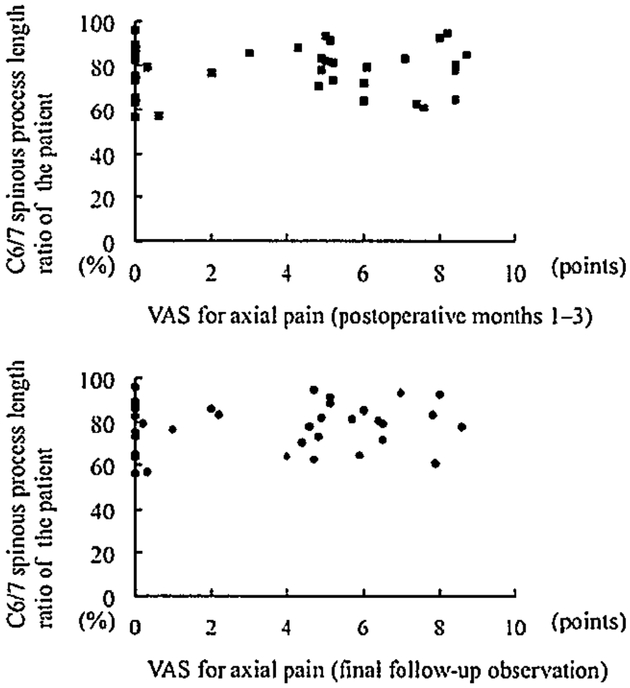


FIG. 7. Correlation between the C-6/C-7 spinous process length ratios and the VAS scores for axial neck pain in the C6-nonpreservation group. The C-6/C-7 spinous process length ratios were not significantly correlated with the VAS scores for axial neck pain either 1–3 months after surgery *(upper)* or at the final follow-up observation *(lower)*.

spinous process and the origin of the trapezius and rhomboid minor muscles on the C-7 spinous process for preventing postoperative axial neck pain. Patients with axial neck pain often experience increasing pain in the sitting or upright position, in contrast to decreasing pain in a lying position. Hosono et al.[6] provided the following explanation for the pain pattern. Downward displacement of the upper extremities occurs in the sitting position because of gravity. The trapezius and rhomboid minor muscles connecting the C-7 spinous process and the scapula are stretched by adduction of the scapula with downward displacement of the upper extremities. The disruption of the connection at the C-7 spinous process, serving as the important muscular attachment point for these muscles, might increase the stress on the remaining muscles attached to other spinous processes, resulting in persistent axial pain.

**TABLE 3. ROM before and after surgery**

| Condition and Parameters | C6 Preservation Group (n = 25) | C6 Nonpreservation Group (n = 35) | p Value |
|---|---|---|---|
| No. of patients w/ CSM | 17 | 26 | |
| Preop ROM (°)* | 35.2 ± 11.7 | 30.1 ± 13.8 | NS |
| Final FU ROM (°)* | 28.8 ± 11.2 | 25.8 ± 11.5 | NS |
| % ROM† | 81.8 | 85.7 | NS |
| No. of patients w/ OPLL | 8 | 9 | |
| Preop ROM (°)* | 23.5 ± 9.0 | 21.1 ± 13.4 | NS |
| Final FU ROM (°)* | 18.5 ± 9.7 | 14.8 ± 7.7 | NS |
| % ROM (%)† | 78.7 | 70.1 | NS |

FU = follow-up.
* Values are presented as the mean ± SD.
† % ROM indicates (preop ROM/ROM at final follow-up) × 100.

**TABLE 4. Neurological outcome**

| Outcome | Mean ± SD | | p Value |
|---|---|---|---|
| | C6-Preservation Group (n = 25) | C6-Nonpreservation Group (n = 35) | |
| Preop JOA score | 10.2 ± 2.9 | 9.8 ± 2.7 | NS |
| Final FU JOA score | 14.1 ± 3.1 | 13.6 ± 3.0 | NS |
| Recovery rate (%)* | 60.8 ± 21.3 | 57.8 ± 29.5 | NS |

* Recovery rate calculated as follows: (JOA at final follow-up – preop JOA/17 – preop JOA) × 100.

The attachment of the trapezius ranges widely from the occipital bone to the T-12 spinous process. In particular, the speculum rhomboideum section, which is composed of the middle fibers of the trapezius, is very strong because of the presence of a very strong tendinous component. From an anatomical study using 50 cadavers, Ono et al.[16] reported that the speculum rhomboideus of the trapezius attached to the spinous process between C-3 and T-3, and its center was located at the C-7 spinous process in more than half of the individuals. The attachment of the rhomboid minor muscle to the spinous process occurred between C-5 and C-7. The C-6 spinous process is the second longest spinous process next to the C7 in the cervical region, and it plays a role as an attachment point for these muscles. Ono et al.[16] also stated that > 50% preservation of the attachment of the speculum rhomboideum of the trapezius was possible in 72% of the patients who underwent C3–6 laminoplasty with preservation of the C-7 spinous process and in 88% of the patients who underwent C3–C5 laminoplasty. The rhomboid minor muscle was possibly spared without complete dissection of the muscular attachment at the spinous process in 35% of the patients who underwent C3–6 laminoplasty and in > 50% of the patients who underwent C3–5 laminoplasty. Therefore, preserving the attachment of the paraspinal muscles to the C-6 spinous process in addition to the C-7 spinous process increases the preservation of the muscular attachment at the spinous process in the central area of the speculum rhomboideus of the trapezius and the rhomboid minor, potentially reducing axial neck pain more than that after using the preservation technique on the C-7 spinous process alone. However, the relationship between preservation of the C-6 spinous process and axial neck pain has not been studied thoroughly. Contrary to our expectations, the mean VAS scores and the distributions of VAS grade for axial neck pain were amazingly comparable between the C6-preservation and C6-nonpreservation groups in both the early and late postoperative stages. In addition, neck pain as assessed by VAS scores and the type of pain and its location were similar between the 2 groups. These results demonstrate that our paraspinal muscle preservation procedure for the C-6 spinous process was not superior to the C6-nonpreservation technique in terms of reducing postoperative axial neck pain.

We assume that the reason that the C6-preservation procedure did not achieve a successful result regarding axial neck pain includes anatomical and technical aspects. We did not know the range of attachment of the speculum rhomboideum of the trapezius and the rhomboid minor to the spinous process in each patient; therefore, we did not understand the extent of preservation of these muscles with regard to treatment of the paraspinal muscles at the C-6 spinous process. The maximum degree of preservation of these muscles and the resulting effect on the control of axial neck pain might be reached with preservation of the C-7 spinous process regardless of the C-6 procedure. Although we also examined the relationship between the length of the C6 spinous process and axial neck pain, the C-6/C-7 spinous process length ratios were similar in the 2 groups, and furthermore, they did not correlate with axial neck pain. From a technical viewpoint, our preservation technique for paraspinal muscles at the C-6 spinous process did not clarify whether the C-6 spinous process and its attachment of paraspinal muscles were left intact. The longitudinally split C-6 spinous process and lamina were spread similar to opening a double door while leaving the muscular attachment to the split spinous processes undisturbed, but the connections of the spinous process to the paraspinal muscles and nuchal ligament were different than those in the original alignment. Our preservation technique at the C-6 level might be insufficient for preventing axial neck pain, but another decompression technique allowing the C-6 spinous process and its paraspinal muscle attachment to be left intact could be successful.

Stiffness and tension, which were frequently observed in the patients in our study, seem to be similar to symptoms derived from back muscle diseases after lumbar surgery.[15] Almost half of the patients in the present study presented with pain in the nuchal or suprascapular region, and these areas seem to correspond to those in which nuchal muscles are stretched between the scapula and spinous processes by adduction of the scapula with downward displacement of the upper extremities. These findings suggest that the pathology of axial neck pain is mainly attributable to intraoperative invasion of the nuchal muscles.

The pathology of neck pain after posterior cervical surgery is multifactorial. Highsmith et al.[4] conducted a comparative study of laminoplasty and laminectomy with fusion in patients with cervical stenotic myelopathy. Preoperative neck pain VAS scores of 3.2 increased slightly postoperatively to 3.4 in the laminoplasty group, whereas in the fusion group, VAS scores significantly improved from 5.8 to 3.0. The authors concluded that cervical fusion significantly reduces neck pain in patients with cervical stenotic myelopathy. Motion-related pain, which is improved by stabilization with fusion, is an important factor in neck pain. This clinical study demonstrated this kind of neck pain. The pathology of preoperative neck pain is possibly different from that of postoperative neck pain. Although pain was reduced after fusion, postoperative neck pain in the fusion group was comparable to that in the laminoplasty group. Spinous processes and paraspinal muscular attachments were not preserved in either of the groups. Postoperative neck pain might have been related to the resection of spinous processes associated with the dissection and detachment of paraspinal muscles.

We used only pain characteristics such as intensity, pattern, and location to evaluate axial neck pain. We believe that our assessment for neck pain was not sufficient, because neck pain is an individual unpleasant sensory and emotional experience that is affected by various factors, including cervical pathologies and psychological issues. We found no differences in the VAS scores for axial neck pain between the CSM and OPLL subgroups 1–3 months after surgery or at the final follow-up observation (3.8 ± 3.2 vs 4.7 ± 3.0 [$p > 0.05$] and 3.5 ± 3.1 vs 4.0 ± 2.5 [$p > 0.05$], respectively). We should have adopted the use of additional instruments such as the Neck Disability Index,[24] which is most widely used to assess the effect of neck pain on the ability to manage everyday tasks, and the Self-rating Depression Scale,[2] which is used for the assessment of psychological factors. These additional evaluations probably would have helped us identify the differences in axial neck pain in our 2 groups of patients.

When VAS Grade III and IV axial neck pain (> 5.0 VAS points) in the present study was considered nearly comparable to moderate and severe axial pain (medicine or physical therapy for the painful muscles regularly needed), respectively, the incidence of axial neck pain in the late postoperative period was 34% in our study compared with 5.4% in the study of Hosono et al.,[6] in which these grades of pain were defined as axial neck pain. Although it is not precise to compare the results directly between these 2 studies because of the differences in axial neck pain measurements, the incidence of postoperative axial neck pain was higher in our study than in the study of Hosono et al. despite the use of C3–6 laminoplasty with C-7 spinous process preservation in both studies. This finding suggests that the difference in the laminoplasty techniques, double-door laminoplasty in our study and open-door laminoplasty in their study, possibly contributes to the pathology of axial neck pain. The purpose of the present study was to examine the efficacy of preserving the paraspinal muscular attachment to the C-6 spinous process in reducing postoperative axial pain. Our technique for preserving the C-6 spinous process did not effectively diminish pain.

The current study has some limitations. The study was retrospective, and the patients were not randomly assigned to their treatment group. Not only the individual surgeons but also the number of surgeons between the 2 groups were different. There are biases based on the technical variety of the surgeons and patient selection. A randomized prospective study without any biases would have been ideal. However, this was a preliminary nonrandomized comparative study aimed at investigating the advantages of the paraspinal muscle preservation technique before a randomized study is conducted. Each surgeon in each group was an expert spine surgeon with more than 10 years of experience who had no technical difficulty in handling the paraspinal muscles. Therefore, the difference in the surgeons between our 2 groups does not seem to highlight a difference in the surgeons' skills. The sample size was small, and it included a combined population of patients with CSM and cervical OPLL. There was no image assessment of the paraspinal muscles at the C-6 level, including atrophy or signal changes of muscles concomitant with postoperative clinical evaluations. A prospective randomized study with a large sample size would be required to compare the effect of differences in axial neck pain on the clinical outcome of C3–6 laminoplasty versus that of C3–5 laminoplasty with the preservation of the C-6 spinous process out of the range of decompression.

## Conclusions

The VAS scores for axial neck pain and the distribution of VAS grades in both the early and late postoperative stages and the types and locations of axial neck pain were comparable between the C-6 paraspinal muscle preservation procedure and the C6-nonpreservation procedure. The C6-preservation technique was not superior to the C6-nonpreservation technique in preventing postoperative axial neck pain.

## References


1. Chiba K, Ogawa Y, Ishii K, Takaishi H, Nakamura M, Maruiwa H, et al: Long-term results of expansive open-door laminoplasty for cervical myelopathy—average 14-year follow-up study. **Spine (Phila Pa 1976) 31:**2998–3005, 2006
2. Estlander AM, Takala EP, Verkasalo M: Assessment of depression in chronic musculoskeletal pain patients. **Clin J Pain 11:**194–200, 1995
3. Fujimori T, Le H, Ziewacz JE, Chou D, Mummaneni PV: Is there a difference in range of motion, neck pain, and outcomes in patients with ossification of posterior longitudinal ligament versus those with cervical spondylosis, treated with plated laminoplasty? **Neurosurg Focus 35(1):**E9, 2013
4. Highsmith JM, Dhall SS, Haid RW Jr, Rodts GE Jr, Mummaneni PV: Treatment of cervical stenotic myelopathy: a cost and outcome comparison of laminoplasty versus laminectomy and lateral mass fusion. **J Neurosurg Spine 14:**619–625, 2011
5. Hirabayashi K, Miyakawa J, Satomi K, Maruyama T, Wakano K: Operative results and postoperative progression of ossification among patients with ossification of cervical posterior longitudinal ligament. **Spine (Phila Pa 1976) 6:**354–364, 1981
6. Hosono N, Sakaura H, Mukai Y, Fujii R, Yoshikawa H: C3-6 laminoplasty takes over C3-7 laminoplasty with significantly lower incidence of axial neck pain. **Eur Spine J 15:**1375–1379, 2006
7. Hosono N, Sakaura H, Mukai Y, Yoshikawa H: The source of axial pain after cervical laminoplasty-C7 is more crucial than deep extensor muscles. **Spine (Phila Pa 1976) 32:**2985–2988, 2007
8. Hosono N, Yonenobu K, Ono K: Neck and shoulder pain after laminoplasty. A noticeable complication. **Spine (Phila Pa 1976) 21:**1969–1973, 1996
9. Itoh T, Tsuji H: Technical improvements and results of laminoplasty for compressive myelopathy in the cervical spine. **Spine (Phila Pa 1976) 10:**729–736, 1985
10. Johnson G, Bogduk N, Nowitzke A, House D: Anatomy and actions of the trapezius muscle. **Clin Biomech (Bristol, Avon) 9:**44–50, 1994
11. Johnson GM, Zhang M, Jones DG: The fine connective tissue architecture of the human ligamentum nuchae. **Spine (Phila Pa 1976) 25:**5–9, 2000
12. Kawaguchi Y, Kanamori M, Ishihara H, Ohmori K, Nakamura H, Kimura T: Minimum 10-year followup after en bloc cervical laminoplasty. **Clin Orthop Relat Res (411):**129–139, 2003
13. Kawaguchi Y, Matsui H, Ishihara H, Gejo R, Yoshino O: Axial symptoms after en bloc cervical laminoplasty. **J Spinal Disord 12:**392–395, 1999
14. Kotani Y, Abumi K, Ito M, Sudo H, Takahata M, Ohshima S, et al: Minimum 2-year outcome of cervical laminoplasty with deep extensor muscle-preserving approach: impact on cervical spine function and quality of life. **Eur Spine J 18:**663–671, 2009
15. Mori E, Okada S, Ueta T, Itaru Y, Maeda T, Kawano O, et al: Spinous process-splitting open pedicle screw fusion provides favorable results in patients with low back discomfort and pain compared to conventional open pedicle screw fixation over 1 year after surgery. **Eur Spine J 21:**745–753, 2012
16. Ono A, Tonosaki Y, Yokoyama T, Aburakawa S, Takeuchi K, Numasawa T, et al: Surgical anatomy of the nuchal muscles in the posterior cervicothoracic junction: significance of the preservation of the C7 spinous process in cervical laminoplasty. **Spine (Phila Pa 1976) 33:**E349–E354, 2008
17. Otani K, Sato K, Yabuki S, Iwabuchi M, Kikuchi S: A segmental partial laminectomy for cervical spondylotic myelopathy: anatomical basis and clinical outcome in comparison with expansive open-door laminoplasty. **Spine (Phila Pa 1976) 34:**268–273, 2009
18. Ratliff JK, Cooper PR: Cervical laminoplasty: a critical review. **J Neurosurg 98 (3 Suppl):**230–238, 2003
19. Sakaura H, Hosono N, Mukai Y, Fujii R, Iwasaki M, Yoshikawa H: Persistent local pain after posterior spine surgery for thoracic lesions. **J Spinal Disord Tech 20:**226–228, 2007
20. Seichi A, Takeshita K, Ohishi I, Kawaguchi H, Akune T, Anamizu Y, et al: Long-term results of double-door laminoplasty for cervical stenotic myelopathy. **Spine (Phila Pa 1976) 26:**479–487, 2001
21. Shiraishi T: A new technique for exposure of the cervical spine laminae. Technical note. **J Neurosurg 96 (1 Suppl):**122–126, 2002
22. Sivaraman A, Bhadra AK, Altaf F, Singh A, Rai A, Casey AT, et al: Skip laminectomy and laminoplasty for cervical spondylotic myelopathy: a prospective study of clinical and radiologic outcomes. **J Spinal Disord Tech 23:**96–100, 2010
23. Yukawa Y, Kato F, Ito K, Horie Y, Hida T, Ito Z, et al: Laminoplasty and skip laminectomy for cervical compressive myelopathy: range of motion, postoperative neck pain, and surgical outcomes in a randomized prospective study. **Spine (Phila Pa 1976) 32:**1980–1985, 2007
24. Vernon H: The Neck Disability Index: state-of-the-art, 1991-2008. **J Manipulative Physiol Ther 31:**491–502, 2008


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# Motion characteristics and related factors of Modic changes in the lumbar spine

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**OBJECT** Most studies of Modic changes (MCs) have focused on investigating the relationship between MCs and low-back pain, whereas the kinematic characteristics and degenerative disc disease associated with MCs are not well understood. To the authors' knowledge, no previous study has reported on the kinematics of MCs. The purpose of this study was to elucidate the relationship of MCs to segmental motion and degenerative disc disease.

**METHODS** Four hundred fifty symptomatic patients underwent weight-bearing lumbar kinematic MRI in the neutral, flexion, and extension positions. Segmental displacement and intervertebral angles were measured in 3 positions using computer analysis software. Modic changes, disc degeneration, disc bulging, spondylolisthesis, angular motion, and translational motion were recorded, and the relationship of MCs to these factors was analyzed using a logistic regression model. To control the influence of disc degeneration on segmental motion, angular and translational motion were analyzed according to mild and severe disc degeneration stages. The motion characteristics and disc degeneration among types of MCs were also evaluated.

**RESULTS** Multivariate analysis revealed that age, disc degeneration, angular motion, and translational motion were factors significantly related to MCs. In the severe disc degeneration stage, a significant decrease of angular motion and significant increase of translational motion were found in segments with MCs, indicating that a disorder of the endplate had an additional effect on segmental motion. Disc degeneration increased and angular motion decreased significantly and gradually as the type of MC increased. Translational motion was significantly increased with Type 2 MCs.

**CONCLUSIONS** Age, disc degeneration, angular motion, and translational motion were significantly linked to MCs in the lumbar spine. The translational motion of lumbar segments increased with Type 2 MCs, whereas angular motion decreased as the type of MC increased, indicating that Type 2 MCs may have translational instability likely due to degenerative changes. A disorder of the endplates could play an important role in spinal instability.

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**KEY WORDS** Modic changes; lumbar spine; kinematic analysis; magnetic resonance imaging; logistic regression analysis; degenerative disc disease

Modic changes (MCs) are bone marrow and endplate changes visible on MRI of patients with degenerative disc disease.[10,11] Modic Type 1 changes are hypointense on T1-weighted MRI and hyperintense on T2-weighted MRI, and are detected in areas with inflammation. Modic Type 2 changes are hyperintense on both T1- and T2-weighted MR images and are detected in areas with fatty degeneration. Modic Type 3 changes are hypointense on both T1- and T2-weighted MR images and detected in areas with sclerosis.

**ABBREVIATIONS** CI = confidence interval; MC = Modic change; OR = odds ratio.

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Many previous papers[2,10,11,16] associated with MCs in the lumbar spine have been published, but most of them are focused on the relationship between MCs and low-back pain.[5,14,17] Some previous studies[1,8,15] demonstrated the relationship between nonfusion after surgery and MCs, and have reported on the instability related to bone marrow changes without using MCs. To our knowledge, no study has reported on the kinematics of MCs. In addition, few studies have reported on the relationship between MCs and degenerative disc disease.

We hypothesized that lumbar MCs have some relationship to segmental motion and degenerative disc disease in the process of degenerative changes. The objective of this study was to evaluate how MCs relate to segmental lumbar motion and degenerative disc disease using kinematic MRI.

## Methods

### Patient Population

Four hundred fifty consecutive symptomatic patients (267 men and 183 women) with an average age of 44.6 ± 12.2 years (range 17–77 years) were examined from March 2011 to October 2011. The inclusion criteria were defined as patients who had low-back pain with or without neurological symptoms. The exclusion criteria were trauma, infection, rheumatoid arthritis, spinal tumors, and history of lumbar spine surgery. A total of 2250 lumbar discs from L1–2 to L5–S1 were retrospectively evaluated for all patients in this study. The Institutional Review Board of the University of California at Los Angeles approved this study and informed consent was obtained from all participants.

### Kinematic MRI

MRI of the spine was performed using a 0.6-T MRI machine (Upright Multi-Position, Fonar Corp.). Two vertically orientated, opposing magnetic doughnuts placed 18 inches apart were used, allowing scanning of the patient sitting in an upright, axially loaded position. A quad-channel planar coil was used to obtain images. We examined the T1-weighted sagittal spin echo images (TR 671 msec, TE 17 msec, thickness 4.0 mm, field of view 30 cm, matrix 256 × 224, number of excitations 2) and T2-weighted fast spin echo images (TR 3000 msec, TE 140 msec, thickness 4.0 mm, field of view 30 cm, matrix 256 × 224, number of excitations 2) of each patient. All patients were scanned in the flexion, neutral, and extension positions.

### MRI Analysis

Midsagittal T2-weighted MR images were marked for digitization by 3 spine surgeons on the flexion, neutral, and extension position images. Vertebral bodies were marked at 4 points (anterior-inferior, anterior-superior, posterior-superior, and posterior-inferior) from L-1 to S-1. The lowest lumbar vertebra was defined as L-5. All MRI parameters were recorded by computer-based measurements, and all calculations were performed with the MRI Analyzer anatomical software (version 3, Truemetric Corp.) for objective quantification as described in prior publications.[6,13,19,20]

Disc bulging was measured as the extension of the disc beyond the intervertebral space, with a greater value representing greater posterior bulging.[19,20] Spondylolisthesis was measured as the displacement of 1 vertebral body on the adjacent level below in the anterior or posterior direction as observed on the neutral image.[13]

Segmental mobility was measured in terms of translational motion and angular motion (Fig. 1). Translational motion was defined as an anterior to posterior shift of the vertebral body during translation and was calculated by measuring the distance of one segment over another in millimeters using T2-weighted sagittal images.[3,6,13] Angular motion was defined as the angle of difference between each vertebral body in flexion and extension.[3,6,13] This was measured by drawing lines along the superior borders of adjacent vertebrae of each motion segment and extending them until they joined. The difference between the two angles, which were not direction dependent, was then calculated.

### Assessment of MCs

Modic changes were also evaluated for 2250 segments from L1–2 to L5–S1 and classified into none or Types 1, 2, and 3, according to their signal patterns on T1- and T2-weighted sagittal MR images. Type 1 MCs had a hypointense signal on T1-weighted sequences and a hyperintense signal on T2-weighted sequences. Type 2 MCs had a hyperintense signal on T1-weighted sequences and hyper- or isointense signal on T2-weighted sequences. Type 3 MCs had a hypointense signal on T1- and T2-weighted sequences.[10,11] The intra- and interobserver reliability of the ratings for MCs were assessed using the $\kappa$ value with a subset of 50 cases (250 intervertebral levels).

### Assessment of Degenerative Disc Change

Disc degeneration was classified into 5 grades using T2-weighted sagittal MR images according to the grading system proposed by Pfirrmann et al.[12] Grade I indicated normal, whereas Grade V indicated the most advanced disc degeneration. Intra- and interobserver reliability for this grading system was previously reported.[13]

### Statistical Analysis

The Mann-Whitney U-test was used for comparisons of disc degeneration, disc bulging, spondylolisthesis, angular motion, and translational motion. According to correction with Bonferroni's inequality in the Kruskal-Wallis test, a p value less than 0.0125 (0.05/4) was considered statistically significant for comparison among types of MCs. After the variables were categorized, the chi-square test was used for the comparisons. Logistic regression analyses were used to compute odds ratios (ORs) and 95% confidence intervals (CIs) and detect the association between the existence of MCs and related factors. The variables in the multivariate model were clinically important from previous reports, regardless of statistical significance.[9,14] Disc degeneration grade was categorized into mild (Grades I–III) and severe (Grades IV and V). Age, spondylolisthesis, disc bulging, angular motion, and translational motion were di-

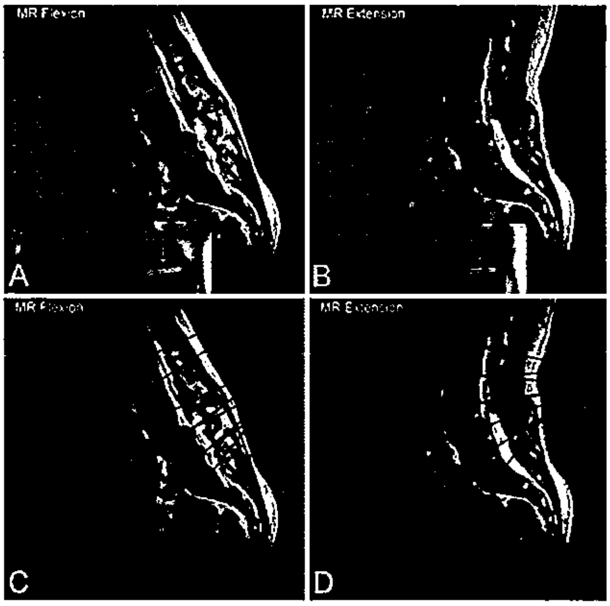


**FIG. 1.** Examples of translational and angular motion from flexion to extension as measured by MR Analyzer computer-based software on sagittal MR images. **A:** Translational motion in flexion. **B:** Translational motion in extension. Total translational motion was calculated as the absolute value of the difference between flexion and extension. **C:** Angular motion in flexion. **D:** Angular motion in extension. Total angular motion was calculated as the absolute value of the difference between the angle between adjacent vertebral bodies in flexion and in extension in degrees. Tan et al: Kinetic magnetic resonance imaging analysis of lumbar segmental mobility in patients without significant spondylosis. *Eur Spine J 21*:2673–2679, 2012. Copyright Springer. Reproduced with kind permission from Springer Science and Business Media. Figure is available in color online only.

vided into 2 groups, establishing the cutoff points at the median because no definitive cutoff point was found in a previous paper. Statistical analyses were performed using SPSS computer software (version 20, IBM Corp.) and values were expressed as mean ± SD. A p value less than 0.05 was considered statistically significant. According to Landis and Koch,[7] the reliability of diagnosis was assessed as follows: κ = 0–0.2 indicated slight agreement, 0.21–0.4 fair agreement, 0.41–0.6 moderate agreement, 0.61–0.8 substantial agreement, and 0.81–1 excellent agreement.

## Results

### Prevalence of MCs

Modic changes were observed in 129 (28.7%) of 450 patients. One hundred sixty-four (7.3%) of 2250 lumbar segments had MCs: Type 1 in 43 segments, Type 2 in 115, and Type 3 in 6. Modic changes were most frequent at L5–S1 followed by L4–5 and L3–4.

### Reliability of Diagnosis of MCs

The interobserver agreement was substantial with a κ value of 0.77. Also, the intraobserver agreement was excellent with a κ value of 0.82.

### Link to Disc Degeneration, Disc Bulging, Spondylolisthesis, Angular Motion, and Translational Motion at Each Level

The relationship between MCs and disc degeneration grade, disc bulging, spondylolisthesis, angular motion, and translational motion at each level are shown in Table 1. Analysis of disc degeneration revealed significant differences between the group with MCs and the group without MCs in all segments, indicating that the segments with MCs had more severe disc degeneration. A significant increase in disc bulging was observed at all segments except L5–S1 between units with MCs and those without MCs. Also, a significant increase in spondylolisthesis was observed at L1–2, L4–5, and L5–S1 in the group with MCs. In terms of kinematic analyses, a significant decrease in angular motion was found at L2–3, L3–4, and L4–5 in units with MCs, and a significant increase in translational motion was observed at all segments with MCs except L5–S1.

**TABLE 1. Comparison between patients with and without MCs at each level**

| Variable | MCs | No MCs | p Value |
|---|---|---|---|
| Disc degeneration grade | | | |
| L1–2 | 3.7 ± 1.3 | 2.1 ± 0.7 | 0.001 |
| L2–3 | 4.1 ± 0.9 | 2.2 ± 0.8 | <0.001 |
| L3–4 | 4.0 ± 1.0 | 2.3 ± 0.9 | <0.001 |
| L4–5 | 4.1 ± 0.7 | 2.6 ± 1.3 | <0.001 |
| L5–S1 | 4.2 ± 0.7 | 2.7 ± 1.2 | <0.001 |
| Disc bulge (mm) | | | |
| L1–2 | 3.5 ± 1.1 | 2.6 ± 0.9 | 0.038 |
| L2–3 | 3.7 ± 1.2 | 2.9 ± 1.0 | 0.01 |
| L3–4 | 3.7 ± 1.3 | 3.2 ± 1.1 | 0.03 |
| L4–5 | 4.3 ± 1.7 | 3.6 ± 1.3 | 0.006 |
| L5–S1 | 4.3 ± 1.9 | 4.1 ± 1.7 | 0.39 |
| Spondylolisthesis (mm) | | | |
| L1–2 | 1.5 ± 0.9 | 0.6 ± 0.6 | 0.001 |
| L2–3 | 1.1 ± 1.0 | 0.8 ± 0.7 | 0.24 |
| L3–4 | 2.0 ± 2.5 | 0.8 ± 0.7 | 0.07 |
| L4–5 | 2.1 ± 2.3 | 1.2 ± 1.1 | 0.02 |
| L5–S1 | 3.3 ± 2.7 | 2.4 ± 1.7 | 0.007 |
| Angular motion (°) | | | |
| L1–2 | 7.8 ± 4.8 | 6.6 ± 3.7 | 0.44 |
| L2–3 | 6.0 ± 2.8 | 8.1 ± 3.9 | 0.03 |
| L3–4 | 5.5 ± 3.5 | 8.2 ± 4.3 | 0.002 |
| L4–5 | 6.0 ± 3.5 | 7.9 ± 5.0 | 0.039 |
| L5–S1 | 5.2 ± 3.9 | 5.8 ± 4.5 | 0.31 |
| Translational motion (mm) | | | |
| L1–2 | 2.6 ± 1.4 | 1.3 ± 0.9 | 0.003 |
| L2–3 | 2.3 ± 1.7 | 1.4 ± 1.0 | 0.03 |
| L3–4 | 2.3 ± 2.7 | 1.1 ± 0.9 | 0.03 |
| L4–5 | 1.7 ± 2.0 | 1.0 ± 0.9 | 0.03 |
| L5–S1 | 1.2 ± 1.4 | 1.0 ± 0.9 | 0.47 |

**TABLE 2. Difference between segments with and without MCs in the mild and severe disc degeneration groups**

| Motion | Mild (Grade I–III) | | | Severe (Grade IV & V) | | |
|---|---|---|---|---|---|---|
| | No MCs | MCs | p Value | No MCs | MCs | p Value |
| Angular | 7.5 ± 4.3 | 6.8 ± 3.6 | NS | 6.8 ± 4.6 | 5.3 ± 3.7 | 0.002 |
| Translational | 1.2 ± 0.9 | 1.5 ± 1.3 | NS | 1.2 ± 1.0 | 1.6 ± 2.0 | 0.04 |

NS = Not significant.

### Segmental Motion With and Without MCs in Mild and Severe Disc Degeneration Stage

To control the influence of disc degeneration on segmental motion, angular and translational motion were analyzed according to mild (Grades I–III) and severe (Grades IV and V) disc degeneration stage (Table 2). No significant differences were observed between the groups without and with MCs with mild disc degeneration, whereas a significant decrease of angular motion and significant increase of translational motion were found in the group with MCs with severe disc degeneration. This result indicates that segmental motion is affected by the presence of MCs in patients with severe disc degeneration. The disorder of the endplate would have an additional effect on segmental motion.

### Factors Related to MCs

Factors potentially related to the changes were evaluated using the chi-square test and logistic regression analysis to control confounding factors (Table 3). Using univariate analysis, significant differences were found in age, disc degeneration, spondylolisthesis, disc bulging, and angular motion. Next, a multiple logistic regression model was used to adjust for age, sex, levels, disc degeneration, spondylolisthesis, disc bulging, angular motion, and translational motion, which were factors likely to relate to MCs and segmental motion. After adjustment for potential confounding factors, significantly elevated ORs were observed in segments with age > 45 years (OR 2.11, 95% CI 1.41–3.15), severe disc degeneration (OR 11.3, 95% CI 7.33–17.4), angular motion ≤ 6.8° (OR 1.70, 95% CI 1.17–2.48), and translational motion > 1.0 mm (OR 1.45, 95%

**TABLE 3. Analysis of related factors to MCs**

| Factor | No. of Discs (%) | | p Value* | Crude OR (95% CI) | Adjusted OR†† (95% CI) |
|---|---|---|---|---|---|
| | No MCs (n = 2086) | MCs (n = 164) | | | |
| Age (yrs) | | | | | |
| ≤45 | 1108 (53) | 42 (26) | | Reference | Reference |
| >45 | 978 (47) | 122 (74) | <0.001 | 3.29 (2.29–4.72)† | 2.11 (1.41–3.15)† |
| Disc degeneration | | | | | |
| Mild (Grades I–III) | 1704 (82) | 31 (19) | | Reference | Reference |
| Severe (Grades IV–V) | 382 (18) | 133 (81) | <0.001 | 19.1 (12.7–28.7)† | 11.3 (7.33–17.4)† |
| Spondylolisthesis (mm) | | | | | |
| ≤0.8 | 1123 (54) | 45 (27) | | Reference | Reference |
| >0.8 | 963 (46) | 119 (73) | <0.001 | 3.08 (2.16–4.39)† | 1.47 (0.98–2.20) |
| Disc bulge (mm) | | | | | |
| ≤3.1 | 1100 (53) | 46 (28) | | Reference | Reference |
| >3.1 | 986 (47) | 118 (72) | <0.001 | 2.86 (2.01–4.07)† | 1.10 (0.74–1.65) |
| Angular motion (°) | | | | | |
| ≤6.8 | 1026 (49) | 112 (68) | | 2.23 (1.58–3.13)† | 1.70 (1.17–2.48)† |
| >6.8 | 1060 (51) | 52 (32) | <0.001 | Reference | Reference |
| Translational motion (mm) | | | | | |
| ≤1.0 | 1120 (54) | 84 (51) | | Reference | Reference |
| >1.0 | 966 (46) | 80 (49) | 0.541 | 1.10 (0.80–1.52) | 1.45 (1.01–2.09)† |

* Calculated using the chi-square test.
† $p < 0.05$, calculated by the univariate and multivariate analyses.
†† The logistic regression model was adjusted for age, sex, level, disc degeneration, spondylolisthesis, disc bulge, angular motion, and translational motion.

CI 1.01–2.09). Although the crude OR for translational motion was not significant (OR 1.10, 95% CI 0.80–1.52), it increased to significance after adjustment for several potential confounders.

### Motion Characteristics of Types of MCs

The mean value of the disc degeneration grade in each MC type was 2.4 ± 1.0 for Type 0, 3.8 ± 0.9 for Type 1, 4.2 ± 0.7 for Type 2, and 5.0 ± 0.0 for Type 3. The analysis of disc degeneration among types of MCs showed a significant increase between Type 0 and Type 1, Type 1 and Type 2, and Type 2 and Type 3 ($p < 0.001$, $p = 0.004$, and $p = 0.004$, respectively) (Fig. 2). The analysis of angular motion among types of MCs showed Type 3 had significantly less than other types, and Type 2 was significantly less than Type 0 (Type 1 and 3, $p = 0.003$; Type 2 and 3, $p = 0.003$; Type 0 and 3, $p = 0.004$; Type 0 and 2, $p < 0.001$), indicating Types 2 and 3 had less angular motion (Fig. 3). The analysis of translational motion among types of MCs revealed a significant increase between Type 0 and 2 ($p = 0.006$), indicating Type 2 changes had more translational instability than Type 0 (Fig. 4).

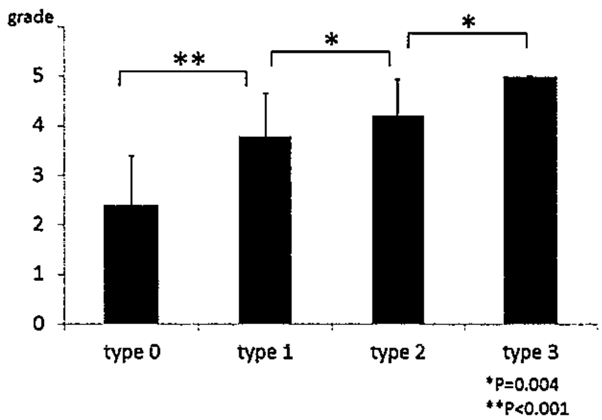


FIG. 2. Bar graph of disc degeneration grade (1–5) and types of MC. A significant increase of disc degeneration grade was observed between Type 0 and Type 1, Type 1 and Type 2, and Type 2 and Type 3. Figure is available in color online only.

## Discussion

A thorough understanding of lumbar segmental motion is valuable to treat patients with degenerative lumbar disease, but kinematics associated with MCs in the lumbar spine have not been well understood. Our multivariate analysis showed MCs were significantly related to angular motion and translational motion. In addition, we were able to identify the details of the motion change among the types of MCs. Our study showed significant decreases in angular motion of Type 2 and Type 3 MCs and a significant increase in translational motion of Type 2. To the best of our knowledge, this is the first study assessing the relationship between segmental motion and MCs.

The reason for the decreased angular motion of Type 2 and Type 3 may be related to the disc degeneration grade. The relationship between disc degeneration and motion was studied by Kong et al.,[6] who reported a kinematic analysis of the relationship between the grade of disc degeneration and motion of the segmental unit of the lumbar spine, and demonstrated that angular motion significantly decreased in severely degenerated segments (Grade V). Their study was consistent with our results because we showed a significant increase of disc degeneration grade associated with an increase in the type of MCs (Fig. 2). Decreased or collapsed disc height due to degenerative change would result in decreased angular motion.

The reason for the increased translational motion also might be related to the disc degeneration grade. Translational motion was reported to be significantly increased in more advanced disc degeneration grade except for Grade V.[6] In addition, Kirkaldy-Willis and Farfan[4] postulated 3 stages with different conditions of stability and motion in the degenerative lumbar spine: dysfunction, instability, and stabilization. Their reports were consistent with our results of translational motion. Increased translational motion at Type 2 may indicate the stage of instability. The mean values of angular motion and translational motion in Type 3 were least among all types, suggesting that the segments with Type 3 may tend to ankylose and lose mobility with severe degeneration.

Interestingly, the significant difference of segmental motion between groups with and without MCs in the severe disc degeneration group suggests that segmental motion may not only be affected by disc degeneration but also by MCs themselves. Spinal instability associated with endplate disruption was reported by Zhao et al.[18] in a cadaveric motion segment experiment. They demonstrated that endplate disruption contributed more to instability than disc dehydration. Thus, in addition to disc degeneration, a disorder of the endplate would also play an important role in segmental instability and have an additional effect on segmental motion.

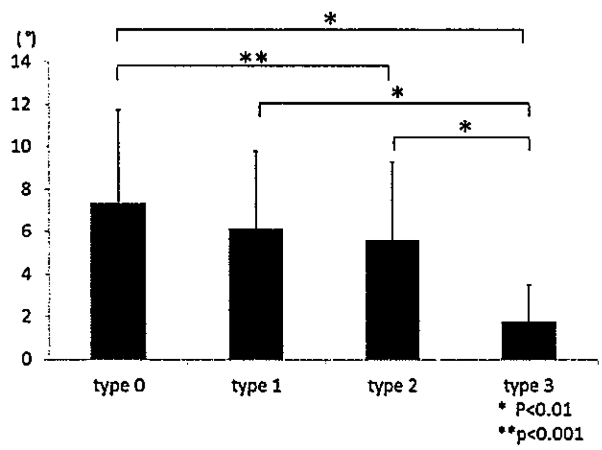


FIG. 3. Bar graph of angular motion (°) and types of MC. A significant decrease in angular motion was found between Type 3 and the other types, and between Type 2 and Type 0. Figure is available in color online only.

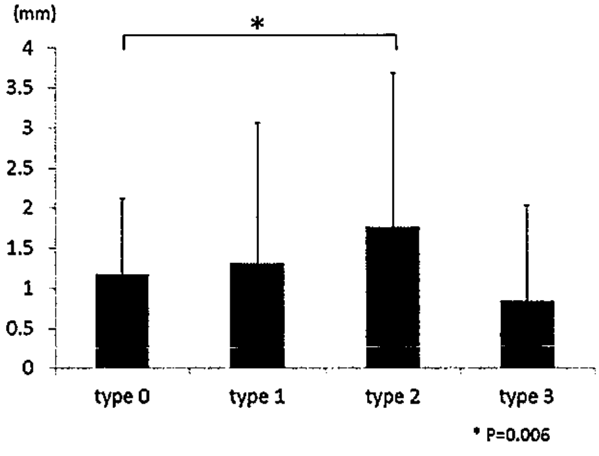


FIG. 4. Translational motion (mm) and types of MC. A significant increase in translational motion was found between Type 0 and 2. Figure is available in color online only.

Although MCs are widely accepted as the change associated with degenerative disc disease or spondylosis, few previous papers have reported on the relationship between MCs and disc bulging and between MCs and spondylolisthesis. Thompson et al.[14] reported that Type 1 MCs show some value in predicting disc herniation and spondylolisthesis compared with non–Type 1 MCs. Jensen et al.[2] reported that bulges or herniations at 40 years of age were the only predictors of new vertebral endplate signal changes at 44 years of age. Mann et al.[9] reported in their study of cervical MCs that patients with MCs are more likely to have a disc herniation at the same level. Our study showed disc bulging and spondylolisthesis were significantly related to MCs using the chi-square test and univariate regression analyses; however, after adjustment for confounding factors, the significant difference disappeared. Because disc degeneration would be the most influential factor in any kind of degenerative disc disease, the variables of disc bulging and spondylolisthesis may be statistically skewed in the multivariable analysis.

The present study has certain limitations. A relatively small number of segments with MCs, especially Type 3, may have reduced the statistical power, although the prevalence of MCs was consistent with previous studies.[17] Larger numbers of older patients in future research may resolve this limitation. Although the common method to assess the motion would be flexion and extension radiographs, we evaluated segmental motion with MRI. This difference might affect the values of measurements.

Clinically, our study suggests that the segments with MCs should be evaluated carefully because MCs may be a sign of a disc bulge or herniation. Moreover, MCs are one of the factors associated with segmental motion. The presence and status of MCs should be taken into consideration when evaluating stability in the lumbar spine. Characterizing the type of MCs observed on MRI may be one of the important factors to take into account when making a decision for or against spinal fusion.

## Conclusions

Patient age, disc degeneration, angular motion, and translational motion are significantly linked to MCs in the lumbar spine. Disc degeneration grade increased significantly as the type of MC increased. Angular motion decreased as the type of MC increased, and translational motion significantly increased with Type 2 MCs compared with Type 0 MCs.

## References

1. Buttermann GR, Heithoff KB, Ogilvie JW, Transfeldt EE, Cohen M: Vertebral body MRI related to lumbar fusion results. **Eur Spine J 6**:115–120, 1997
2. Jensen TS, Kjaer P, Korsholm L, Bendix T, Sorensen JS, Manniche C, et al: Predictors of new vertebral endplate signal (Modic) changes in the general population. **Eur Spine J 19**:129–135, 2010
3. Keorochana G, Taghavi CE, Lee KB, Yoo JH, Liao JC, Fei Z, et al: Effect of sagittal alignment on kinematic changes and degree of disc degeneration in the lumbar spine: an analysis using positional MRI. **Spine (Phila Pa 1976) 36**:893–898, 2011
4. Kirkaldy-Willis WH, Farfan HF: Instability of the lumbar spine. **Clin Orthop Relat Res (165)**:110–123, 1982
5. Kjaer P, Korsholm L, Bendix T, Sorensen JS, Leboeuf-Yde C: Modic changes and their associations with clinical findings. **Eur Spine J 15**:1312–1319, 2006
6. Kong MH, Morishita Y, He W, Miyazaki M, Zhang H, Wu G, et al: Lumbar segmental mobility according to the grade of the disc, the facet joint, the muscle, and the ligament pathology by using kinetic magnetic resonance imaging. **Spine (Phila Pa 1976) 34**:2537–2544, 2009
7. Landis JR, Koch GG: The measurement of observer agreement for categorical data. **Biometrics 33**:159–174, 1977
8. Lang P, Chafetz N, Genant HK, Morris JM: Lumbar spinal fusion. Assessment of functional stability with magnetic resonance imaging. **Spine (Phila Pa 1976) 15**:581–588, 1990
9. Mann E, Peterson CK, Hodler J: Degenerative marrow (Modic) changes on cervical spine magnetic resonance imaging scans: prevalence, inter- and intra-examiner reliability and link to disc herniation. **Spine (Phila Pa 1976) 36**:1081–1085, 2011
10. Modic MT, Masaryk TJ, Ross JS, Carter JR: Imaging of degenerative disk disease. **Radiology 168**:177–186, 1988
11. Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR: Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. **Radiology 166**:193–199, 1988
12. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N: Magnetic resonance classification of lumbar intervertebral disc degeneration. **Spine (Phila Pa 1976) 26**:1873–1878, 2001
13. Tan Y, Aghdasi BG, Montgomery SR, Inoue H, Lu C, Wang JC: Kinetic magnetic resonance imaging analysis of lumbar segmental mobility in patients without significant spondylosis. **Eur Spine J 21**:2673–2679, 2012
14. Thompson KJ, Dagher AP, Eckel TS, Clark M, Reinig JW: Modic changes on MR images as studied with provocative diskography: clinical relevance—a retrospective study of 2457 disks. **Radiology 250**:849–855, 2009
15. Toyone T, Takahashi K, Kitahara H, Yamagata M, Murakami M, Moriya H: Vertebral bone-marrow changes in degenerative lumbar disc disease. An MRI study of 74 patients with low back pain. **J Bone Joint Surg Br 76**:757–764, 1994
16. Wu HL, Ding WY, Shen Y, Zhang YZ, Guo JK, Sun YP, et

al: Prevalence of vertebral endplate modic changes in degenerative lumbar scoliosis and its associated factors analysis. **Spine (Phila Pa 1976) 37**:1958–1964, 2012
17. Zhang YH, Zhao CQ, Jiang LS, Chen XD, Dai LY: Modic changes: a systematic review of the literature. **Eur Spine J 17**:1289–1299, 2008
18. Zhao F, Pollintine P, Hole BD, Dolan P, Adams MA: Discogenic origins of spinal instability. **Spine (Phila Pa 1976) 30**:2621–2630, 2005
19. Zou J, Yang H, Miyazaki M, Morishita Y, Wei F, McGovern S, et al: Dynamic bulging of intervertebral discs in the degenerative lumbar spine. **Spine (Phila Pa 1976) 34**:2545–2550, 2009
20. Zou J, Yang H, Miyazaki M, Wei F, Hong SW, Yoon SH, et al: Missed lumbar disc herniations diagnosed with kinetic magnetic resonance imaging. **Spine (Phila Pa 1976) 33**:E140–E144, 2008

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CHAPTER 64

# Geriatric Spinal Cord Injuries: Rehabilitation Perspective

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## Learning Objectives

At the end of this chapter, you will be able to:

- Describe the differences in etiology, complications, and outcomes between younger and older population with spinal cord injury.
- Identify the medical and social problems in older population.
- Prepare treatment strategies specifically for geriatric population.

## INTRODUCTION

Elderly people (age ≥65 years) constitute a large proportion of the population (15–20%) in high-income countries, and many require hospitalization for old age-related diseases.[1] The steady increase in the age of the general population will also probably result in a larger proportion of elderly patients with spinal cord injuries (SCIs).[2]

Elderly patients with SCI have reduced functional reserves and greater comorbidity[3] and are more likely to have been physically disabled prior to the SCI, compared with younger patients with SCI.[4] Elderly patients with SCI do not need special techniques for rehabilitation therapy, but they require specialized medical consideration and rehabilitation strategies during hospitalization.

SCI in the elderly can be divided in two categories based on the time of onset: (a) onset of SCI at old age (or late-onset SCI) and (b) onset at young age with survival to old age (i.e., early-onset SCI). In this chapter, we focus on individuals with late-onset SCI.

## EPIDEMIOLOGY OF TRAUMATIC SCI IN GERIATRIC POPULATION

### INCIDENCE

Understanding the epidemiological pattern of SCI is the first step toward preventive strategies. However, the epidemiology of SCI varies in different countries and there is relatively little information on geriatric SCI.

The incidence is far higher in the elderly population than in the younger population.[5] One study from Canada showed that the age-adjusted incidence rates were 41.79 per million per year among adults (aged 15–64 years), and 50.87 per million per year for adults 65 years and older.[5]

In developed countries, the incidence of geriatric SCI is increasing. In Finns aged 50 years or older, the raw incidence of fall-induced cervical SCI (fracture, cord injury, or both) rose considerably between 1970 and 2004, from 52 in 1970 to 120 in 2004.[6] Among persons enrolled in the United States combined data set, the mean age at injury has increased from 28.3 years during the 1970s to 37.1 years between 2005 and 2008.[7] The proportion of participants with new injuries who were at least 60 years of age at injury increased from 4.6% in the 1970s to 13.2% between 2005 and 2008.[7] Two recent Scandinavian studies have also evaluated trends in the incidence rates over the past few decades.[8,9] In Norway, incomplete cervical SCI increased especially among men >60 years of age,[8] and in Finland, there was an increase in the SCI incidence rate among persons aged >55, including both tetraplegia and incomplete SCI.[9] These increases probably reflect the increase in aging population and the propensity for older patients to sustain SCI.[10] Interestingly, the mean age of patients

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with traumatic SCI seems to be lower in less developed countries than in developed countries.[11]

### ETIOLOGY

The mechanisms of injury in elderly patients with SCI differ substantially from those in younger patients.[10] Elderly patients are susceptible to minor trauma and much more likely to sustain SCI secondary to a fall.[5,6,10] A study from the United States[10] (SCI database at a single institution, $n = 3481$) examined the etiology of SCI in elderly patients (≥70 years of age) and indicated that falls accounted for 74% of SCI in elderly patients. Another study from Canada showed that falls were responsible for 63% of SCI among patients older than 65 years.[5] As mentioned above, the incidence of fall-induced severe cervical SCI seems to show an alarming rise in Finnish persons aged 50 years or older.[6]

### LEVEL AND SEVERITY OF INJURY

The high rate of tetraplegic injuries account for a greater percentage of SCI in elderly patients than in adults patients.[5,10] Pickett et al.[5] reported that cervical SCI accounted for 94% of SCI in older patients (age > 60 years), compared with only 70% of SCI in patients younger than 60 years. Elderly patients, with a high incidence of ground-level falls, tend to suffer injury of the upper cervical spine when the head strikes the ground upon falling.[10]

As elderly patients often sustain vertebral spine and SCI from low-velocity trauma, they are more likely to present with incomplete, as opposed to complete neurologic, injuries.[4] Fassett et al.[10] showed that 63% of elderly patients sustained AIS grades C and D injuries compared with 40% among the younger patients.

The increase in SCI among geriatric persons poses great clinical and social concerns. The results of the studies on epidemiology of SCI indicate that preventive programs for SCI in the geriatric population should focus on the vulnerability to low falls.

**Points to Remember**

- The incidence of SCI in geriatric population is higher than in younger people.
- There has been an increase recently in the incidence of SCI in the geriatric population in developed countries.
- Elderly patients are susceptible to minor trauma and they are much more likely to sustain incomplete SCI secondary to a fall.

## SPECIAL ISSUES IN ASSESSMENT AND CLASSIFICATION OF TRAUMA-RELATED GERIATRIC SCI

### INJURY TYPES AND CLASSIFICATIONS

Assessment of elderly patients with SCI should be undertaken with understanding of the unique pathophysiology, age-related disorders, and associated comorbidities in these patients. Elderly patients are especially vulnerable to traumatic SCI due to the following factors; (a) age-related changes in bone quality,[10] (b) age-related predisposition to cervical spinal stenosis,[10] (c) propensity for fall-related injuries due to poor vision, vestibular system dysfunction, and abnormal proprioception in the lower extremity,[12] and (d) high rate of motor vehicle accidents per mile driven.[10]

In comparison with younger patients, elderly patients are less likely to have severe neurological deficits (greater percentage of AIS grades C and D injuries).[10] The International Neurological Standards Committee classified incomplete SCI into five types: central cord syndrome, Brown–Séquard syndrome, anterior cord syndrome, conus medullaris, and cauda equina syndromes. Elderly patients are at high risk of cervical spine injury.[13] The central cord syndrome (CCS), a common injury usually sustained as a result of an extension injury to the cervical spine, often occurs in elderly patients with underlying spondylotic changes. These patients experience variable degrees of functional recovery, but some degree of residual deficit and spasticity is likely. Traumatic CCS (TCCS) is generally considered to be associated with good prognosis.[14] However, the outcome is often worse in the elderly than younger people.[15,16]

It is necessary to evaluate cognitive abnormalities that can sometimes impede rehabilitation, especially in elderly patients with SCI. Age is recognized as a significant factor that negatively affects cognitive function. Furthermore, SCIs in elderly patients tend to be associated with traumatic brain injury (TBI), because trauma associated with falls that causes SCI also often results in TBI, and traumatic cervical SCI is also associated with a high rate of TBI but not more severe injuries.[17] As mentioned above, the geriatric population is prone to falls-related cervical SCI.

**Point to Remember**

- When we assess elderly patients with SCI, it is important to understand the unique pathophysiology, age-related disorders, and associated comorbidities, including cognitive abnormalities.

## SPECIAL ISSUES IN MEDICAL MANAGEMENT AND REHABILITATION

General physical examination is important in elderly patients, due to the high post-SCI morbidity and mortality rates in this population.[18] DeVivo et al.[19] reported that the 7-year survival rate is significantly lower in patients with SCI over 50 years of age (22.7%), compared with that of the general population (86.7%). The aging-related decline in organ function is a problem faced by all elderly individuals and susceptibility to illness is one of the many consequences of aging.[20] Moreover, immobilization after injury increases the likelihood of disuse-related complications in the elderly compared with younger patients. The high mortality rate in elderly patients with SCI is probably due to limited physiological reserve.[10]

Pneumonia is the leading cause of death irrespective of the postinjury time period (up to 30 years after SCI).[21] The level and severity of SCI correlate directly with the development of respiratory complications.[22] Other factors associated with pulmonary complications in the elderly are age, preexisting medical illnesses (especially pulmonary), smoking, and major trauma.[23] In addition, dysphagia and regurgitation of gastric contents can cause aspiration pneumonia. Dysphagia is a relatively common secondary complication known to occur after acute cervical SCI, especially in the elderly. The expedient diagnosis of dysphagia is imperative to reduce the risk of development of pulmonary complications.

In geriatric population, the existing neurological conditions should be examined. SCI in neuromuscular disorders, such as Parkinson's disease has been reported.[24,25] Because neuromuscular disorders produce tremor, muscular rigidity, shuffling gait, and stooped posture, patients with these diseases are at greater risk of falling. Moreover, because patients with Parkinson's disease also usually have poor bone quality,[24] falls produce an increased risk of spinal fracture. The existing neuromuscular disorders should be reflected in any goal setting.

DeVivo et al.[4] analyzed the data of 866 in-hospital patients between 1973 and 1985 and compared the relative risks for various comorbidities in elderly (≥61 years) and young patients with SCI (Table 64.1). Outcomes were measured at discharge and 2 years after injury. Patients who were at least 61 years of age were 2.1 times more likely to have developed pneumonia, 2.7 times more likely to have experienced a gastrointestinal hemorrhage, 5.6 times more likely to have developed pulmonary emboli, and 16.8 times more likely to have had renal stones prior to first definitive discharge than their 16- to 30-year-old counterparts.

Patients who were at least 61 years of age were 3.9 times more likely to have been rehospitalized during the second postinjury year than patients in the 16- to 30-year-old age group; 2.1 times more likely to have required artificial ventilatory support prior to discharge; 22.7 times more likely to have been discharged to a nursing home; 71.8 times more likely to be in a nursing home 2 years after injury during the second postinjury year.

There is no specific information about special techniques for rehabilitation of geriatric SCI. However, the elderly patients with SCI have the following unique characteristics: (a) age-related complications and premorbid diseases such as cognitive abnormalities and pain, (b) limited physiological reserve, including difficulty to increase muscle strength, (c) a lot of incomplete neurological injuries, (d) short life expectancy, all of which should be taken into account when we determine the goal. The geriatric patients present with a complex set of issues and associated disability. This complexity requires comprehensive assessment and an integrated approach.

**Table 64.1 Relative Risk of Rehabilitation Outcome in SCI Patients Aged ≥61 Years and Younger Patients[4]**

| Condition | Relative Risk |
|---|---|
| Pneumonia | 2.1 |
| Gastrointestinal hemorrhage | 2.7 |
| Pulmonary emboli | 5.6 |
| Renal stones before first discharge | 16.8 |
| Rehospitalization during second postinjury year | 3.9 |
| Artificial ventilator support before discharge | 2.1 |
| Discharge to nursing home | 22.7 |
| Nursing home placement within 2 years | 71.8 |

**Points to Remember**

- The morbidity and mortality rates following SCI are higher in elderly patients, probably due to the limited physiological reserve.
- Pneumonia is the leading cause of death during all postinjury time periods after SCI.
- Dysphagia is a relatively common secondary complication known to occur after acute cervical SCI, especially in the elderly. Early diagnosis of dysphagia is imperative to reduce the risk of the development of pulmonary complications.
- The geriatric patients present with a complex set of issues and associated disability. This complexity requires comprehensive assessment and an integrated approach.
- The basis of rehabilitation in elderly patients is mobilized with physical training to increase residual physical functions.

## PROGNOSIS AND GOAL SETTING

Fassett et al.[10] maintained SCI registry of 3481 patients at a single institution during the period extending from 1978 to the end of 2005. They demonstrated that in comparison with younger patients, senior patients (defined as ≥70 years of age) were found to be less likely to have severe neurological deficits, but the mortality rates were higher in the older age group both for the period of hospitalization (27.7% compared with 3.2%, $p < 0.001$) and during the 1-year follow-up period. The mortality rates in the elderly directly correlated with the severity of neurological injury (1-year mortality rate, AIS grade A 66%, grade D 23%, $p < 0.001$). The mortality rate in elderly patients with SCI was stable over the two decades of the above study, but the 1-year mortality rate was greater than 40% in all the analyzed time periods.

Rehabilitation produces gains in activities of daily living comparable to younger patients,[20] yet older patients achieve less objective neurological recovery and independent walking.[4] In addition, patients with concomitant neuromuscular disorders, which tend to be mostly elderly patients, are also reported to have worse outcome.[24] In addition, ventilatory impairment is a common problem in elderly patients recovering from SCI, and is associated with high morbidity rate.[26]

Although functional decline is inevitable, research suggests lack of decline in psychological well-being with aging. Despite several reports of high rates of depression in the elderly, there is evidence that perceived quality of life (QOL) is not necessarily worse in the elderly, even in those with chronic illnesses.[27] In addition, elderly patients may develop expectations that are more commensurate with adaptation to illness than younger individuals and may be more able to cope with life stresses.

**Points to Remember**

- In comparison with younger patients, the mortality rates are higher in the older age group.
- Goal setting is often difficult in geriatrics medicine for the following reasons:
  - They often develop age-related complications, premorbid diseases, and cognitive disorders.
  - They are more likely to present with incomplete as opposed to complete neurological injuries.

## COMPLICATIONS

### PRESSURE ULCER

Despite the progress in medicine, such as universal use of specialty beds and early nutrition, pressure ulcers are a common secondary condition associated with SCI. Pressure ulcers can potentially interfere with activities of daily living, occupational duties, and in severe cases, they may threaten life.

Aging itself might be a risk factor of pressure ulcers. Eastwood et al.[28] analyzed the data of 3904 persons with SCI discharged from the Model Systems between 1990 and 1997, who had follow-up interviews at 1 year postinjury. They showed that at first anniversary, lower discharge motor Functional Independence Measure, injury level, and age were related to the presence of pressure ulcers, rehospitalization, residence, and time spent out of residence in logistic regressions.

Rochon et al.[29] examined risk factors for pressure ulcers in 364 patients with SCI. They identified a pressure ulcer in 81 of 364 patients and revealed that in the univariate analyses, pressure ulcers were associated with Frankel groups A to B with an odds ratio (OR) of 5.7, low albumin with an OR of 4.9, low hemoglobin with an OR of 2.5, age > or = 60 years with an OR of 1.9 and three independent measures of comorbidity: Cumulative Illness Rating Scale with an OR of 3.7, Charlson Index with an OR of 2.2, and International Classification of Diseases, Ninth Revision, Clinical Modification count with an OR of 4.2.

Geriatric persons with SCI are more likely to develop a pressure ulcer for the following reasons: (a) these patients have increased arteriosclerotic risk factors and small vessel circulation is decreased, (b) it is difficult for them to take a posture to reduce the buttock pressure, (c) some older persons are malnourished and show extreme bony prominence. To prevent the pressure ulcer in the elderly, special consideration for pressure relief maneuvers and nutritional status is required.

### PAIN

Neuropathic pain is a serious clinical symptom in SCI patients. SCI-related pain interferes with daily activities and significantly influences QOL.[30] Modirian et al.[30] studied the type and site of pain as well as the factors that exacerbate and alleviate such pain in 1295 SCI patients. Spinal cord-related pain was reported by 64.9% of their patients, while 8.8% reported history of pain but had no complaint at the time of examination, and 26.3% had never suffered any pain. The most common sites of pain were the distal lower extremities (46.5%), proximal lower extremities (40.9%), pelvic girdle (24.5%), and upper limbs (5.7%). The intensity of neuropathic pain was more severe at night than other times of the day.[31] The meta-analysis study of Dijkers et al.,[32] who analyzed 42 studies, indicated that completeness of injury (at least as simplified to complete versus incomplete) did not correlate with differences in pain prevalence. Furlan and Fehlings[33] analyzed data of the Third National Spinal Cord Injury Study and reported no significant correlation between age at the time of trauma and change in pain scores at all stages after SCI. These findings reinforce the need for continued research and education on neuropathic pain in SCI.

## SPASTICITY

Assessment of spasticity is also important in the assessment of prognosis. Spasticity does have some potential benefits as it increases muscle bulk and tone, reduces osteoporosis, and enhances standing.[34] It is important to understand the balance between advantages and disadvantages of spasticity in patients with SCI.

As stated above, elderly patients are less likely to have severe neurological deficits.[10] Mobile patients require optimal spasticity with orthosis under the control of medications. Antispastic drugs are effective in reducing stretch reflexes without substantially reducing volitional torque. The differential effects of tizanidine and baclofen on reflexes of flexor and extensor muscles warrant further investigation into patient-specific management of antispastic drugs.[35] It is reported that locomotor training can reduce spasticity as measured by decreased plantar flexor excitability, ankle clonus, and quadriceps spasm.[36]

## NEUROGENIC BOWEL

Neurogenic bowel represents colonic dysfunction resulting from lack of control of the central nervous system, and is a major physical and psychological problem for persons with SCI.[37]

Colorectal function can have a devastating effect on the daily life of elderly patients with SCI.[38] For example, while constipation is a frequently reported problem in numerous studies of SCI irrespective of age,[38] it is more prevalent in the elderly and those with a long history of injury.[39]

Fear of fecal incontinence frequently restricts patients from home activities.[40] Lynch et al.[41] assessed 467 persons with SCI and 668 age- and gender-matched controls, and demonstrated that continence deteriorates with increasing age in the general population but does not change with increasing age or time since injury in persons with SCI ($n$ = 467). However, another study reported a larger proportion of elderly individuals with SCI suffered fecal incontinence.[42]

TCCS is considered the most prevalent incomplete SCI[43] and frequently occurs in elderly individuals with cervical spondylosis who sustain hyperextension injuries without spine fractures in falls. TCCS is generally considered to be associated with good prognosis and complete neurological and functional recovery.[43] One study investigated in detail the effects of age on bowel management and bowel care-related activities of daily living in individuals with TCCS.[15] The results identified significantly fewer patients aged ≥70 years with "continent spontaneous defecation" or "independent for bowel care", compared with younger patients.

The above studies suggest that management of neurogenic bowel in the geriatric SCI population can be challenging. However, alleviating bowel problems might help improve the QOL of these patients.

## NEUROGENIC BLADDER

In the general population, aging is accompanied by diminished bladder capacity and urethral compliance and an increase in uninhibited detrusor contractions and residual bladder volume.[38] In addition, elderly individuals appear to be at increased risk for urinary tract infection and a gradual decline in kidney function.[38]

These changes also seem to affect individuals with SCI. However, the incidence of bladder problems, such as urinary tract infections and urinary incontinence, is reported to correlate with advancement of age (older > younger) in this population,[44,45] although other studies showed no such correlation.[42] The likely reasons for this discrepancy are differences in study methodologies and/or definition of urinary problems including urinary tract infections and urinary incontinence.

Young patients with SCI have a better outcome with regard to bladder management. Scivoletto et al.[2] compared outcome measures in elderly and young patients with SCI. With regard to the bladder, more patients of the young age group reached independence in voiding,[2,46] suggesting that the following account for the difficulties in bladder rehabilitation in elderly individuals with SCI, (a) diminished ability of elderly people to cope with the new situation, (b) pre-existing factors interfering with micturition, such as benign prostate hyperplasia and cystocele, (c) delayed or weak detrusor reflex activity, and (d) the erroneous belief, by both physicians and nurses, that continuous catheter bladder drainage is a safe and comfortable way to solve the problem.

The management of bladder dysfunction in elderly individuals with SCI is difficult, as it is in younger ones. Treatment of neurogenic bladder in the geriatric population with SCI requires comprehensive understanding of the above-mentioned factors, in addition to the neurological findings and activities of daily living.

## RESPIRATORY AND CARDIOVASCULAR SYSTEMS

Rabadi et al.[47] studied the predictors of mortality in 147 veterans with traumatic SCI and concluded that there were three major causes of death: infection, such as pneumonia (21%), urinary infection (14%), and infection of pressure ulcers (11%); cardiovascular diseases, such as congestive heart failure (16%), coronary arterial disease (13%), and atrial fibrillation (2%); and malignancies (16%). In the same study, Cox regression analysis showed the age at the time of injury was the main predictor of SCI-related mortality. Their study suggested that old age at the time of injury is a significant predictor of mortality following SCI and that such patients are more likely to die from cardiovascular deaths than the general population.

Osterthun et al.[48] studied cardiovascular and respiratory functions in individuals who suffered traumatic or nontraumatic SCI during a follow-up period of 6.2 years and found that 27 persons (12.2%) died during the follow-up and the main causes of death were cardiovascular disease (37.0%), pulmonary disease (29.6%), and malignancy (14.8%). Old age at injury, nontraumatic SCI, and history of other medical conditions were independent predictors of death. They concluded that the main causes of death were cardiovascular and pulmonary diseases.

Glaser[49] indicated that cardiopulmonary (aerobic) fitness is difficult to develop and maintain and this can often be exacerbated by a sedentary lifestyle. It is evident that exercise training programs utilizing appropriate techniques can markedly improve the physical fitness, functional independence, and rehabilitation outcome of wheelchair users. Shiba et al.[50] measured maximum oxygen consumption ($VO_{2max}$) of eight individuals with SCI in 1986–1988 and in 2006 and found stable $VO_{2max}$ while continuously maintaining sports activities. The results indicated that physical capacity reflected the level of sports activity in individuals with SCI who continuously performed sports activities.

### Points to Remember

- To prevent and/or treat complications appropriately, we should understand the differences in complications affecting younger and older patients with SCI.
- Aging itself might be a risk factor of pressure ulcers.
- Elderly patients are less likely to have severe neurological deficits. Geriatric mobile patients greatly require optimal spasticity with orthosis under the control of medications.
- Constipation is more prevalent in the elderly and those with a long history of injury.
- In TCCS, geriatric patients are less likely to be "continent spontaneous defecation" or "independent for bowel care", compared with younger patients.
- Cardiovascular and pulmonary diseases are main causes of death in patients with SCI. Old age at the time of injury might be a significant predictor of mortality following SCI.
- Geriatric patients with SCI, just as the younger patients, should receive intensive and long-term rehabilitation to improve the physical fitness, functional independence, and rehabilitation outcome.

## Key Points

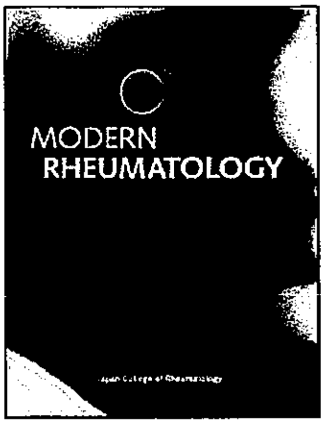
- Recently, there has been an increase in the incidence of SCI in the geriatric population in developed countries.
- Elderly patients are susceptible to minor trauma and they are much more likely to sustain incomplete SCI secondary to a fall.
- Elderly patients with SCI have reduced functional reserves and greater comorbidity prior to SCI compared with younger patients with SCI.
- A significant proportion of elderly patients with SCI die from cardiovascular disease or pneumonia than the general population.
- Geriatric patients with SCI should receive intensive and long-term rehabilitation as well as younger patients.
- Geriatric patients present with a complex set of issues and associated disability. This complexity requires comprehensive assessment and an integrated approach.
- After rehabilitation, geriatric patients with SCI should maintain physical activities and/or sports to prevent any negative impacts of SCI.

## REFERENCES

1. WHO Regional Office for Europe's Health Evidence Network (HEN): do current discharge arrangements from inpatient hospital care for the elderly reduce readmission rates, the length of inpatient stay or mortality, or improve health status? WHO; 2005.
2. Scivoletto G, Morganti B, Ditunno P, Ditunno JF, Molinari M. Effects on age on spinal cord lesion patients' rehabilitation. *Spinal Cord* 2003;41:457-64.
3. New PW, Epi MC. Influence of age and gender on rehabilitation outcomes in nontraumatic spinal cord injury. *J Spinal Cord Med* 2007;30:225-37.
4. DeVivo MJ, Kartus PL, Rutt RD, Stover SL, Fine PR. The influence of age at time of spinal cord injury on rehabilitation outcome. *Arch Neurol* 1990;47:687-91.

5. Pickett GE, Campos-Benitez M, Keller JL, Duggal N. Epidemiology of traumatic spinal cord injury in Canada. *Spine (Phila Pa 1976)* 2006;31:799-805.
6. Kannus P, Palvanen M, Niemi S, Parkkari J. Alarming rise in the number and incidence of fall-induced cervical spine injuries among older adults. *J Gerontol A Biol Sci Med Sci* 2007;62:180-3.
7. DeVivo MJ, Chen Y. Trends in new injuries, prevalent cases, and aging with spinal cord injury. *Arch Phys Med Rehabil* 2011;92:332-8.
8. Hagen EM, Eide GE, Rekand T, Gilhus NE, Gronning M. A 50-year follow-up of the incidence of traumatic spinal cord injuries in Western Norway. *Spinal Cord* 2010;48:313-8.
9. Ahoniemi E, Alaranta H, Hokkinen EM, Valtonen K, Kautiainen H. Incidence of traumatic spinal cord injuries in Finland over a 30-year period. *Spinal Cord* 2008;46:781-4.
10. Fassett DR, Harrop JS, Maltenfort M, et al. Mortality rates in geriatric patients with spinal cord injuries. *J Neurosurg Spine* 2007;7:277-81.
11. Rahimi-Movaghar V, Sayyah MK, Akbari H, et al. Epidemiology of traumatic spinal cord injury in developing countries: a systematic review. *Neuroepidemiology* 2013;41:65-85.
12. Richardson JK, Hurvitz EA. Peripheral neuropathy: a true risk factor for falls. *J Gerontol A Biol Sci Med Sci* 1995;50:M211-5.
13. The Spinal Cord Injury Rehabilitation Evidence (SCIRE). Available from: http://www.scireproject.com/rehabilitation-evidence
14. Tow AP, Kong KH. Central cord syndrome: functional outcome after rehabilitation. *Spinal Cord* 1998;36:156-60.
15. Furusawa K, Tokuhiro A, Ikeda A, et al. Effect of age on bowel management in traumatic central cord syndrome. *Spinal Cord* 2012;50:51-6.
16. Penrod LE, Hegde SK, Ditunno JF. Age effect on prognosis for functional recovery in acute, traumatic central cord syndrome. *Arch Phys Med Rehabil* 1990;71:963-8.
17. Macciocchi S, Seel RT, Thompson N, Byams R, Bowman B. Spinal cord injury and co-occurring traumatic brain injury: assessment and incidence. *Arch Phys Med Rehabil* 2008;89:1350-7.
18. Liang HW, Wang YH, Lin YN, Wang JD, Jang Y. Impact of age on the injury pattern and survival of people with cervical cord injuries. *Spinal Cord* 2001;39:375-80.
19. DeVivo MJ, Kartus PL, Stover SL, Rutt RD, Fine PR. Seven-year survival following spinal cord injury. *Arch Neurol* 1987;44:872-5.
20. Kennedy P, Evans MJ, Berry C, Mullin J. Comparative analysis of goal achievement during rehabilitation for older and younger adults with spinal cord injury. *Spinal Cord* 2003;41:44-52.
21. DeVivo MJ, Krause JS, Lammertse DP. Recent trends in mortality and causes of death among persons with spinal cord injury. *Arch Phys Med Rehabil* 1999;80:1411-9.
22. Shem K, Castillo K, Wong SL, Chang J, Kolakowsky-Hayner S. Dysphagia and respiratory care in individuals with tetraplegia: incidence, associated factors, and preventable complications. *Top Spinal Cord Inj Rehabil* 2012;18:15-22.
23. Lemons VR, Wagner FC, Jr. Respiratory complications after cervical spinal cord injury. *Spine* 1994;19:2315-20.
24. Babat LB, McLain RF, Bingaman W, Kalfas I, Young P, Rufo-Smith C. Spinal surgery in patients with Parkinson's disease: construct failure and progressive deformity. *Spine* (Phila Pa 1976) 2004;29:2006-12.
25. Koller H, Acosta F, Zenner J, et al. Spinal surgery in patients with Parkinson's disease: experiences with the challenges posed by sagittal imbalance and the Parkinson's spine. *Eur Spine J* 2010;19:1785-94.
26. Chaudhry S, Sharan A, Ratliff J, Harrop JS. Geriatric spinal injury. *Semin Spine Surg* 2007;19:229-34.
27. George LK. Social factors and illness. In: Binstock RH, George LK, editors. Handbook of ageing and the social sciences. 4th ed. San Diego: *Academic Press*; 1996. p. 229-52.
28. Eastwood EA, Hagglund KJ, Ragnarsson KT, Gordon WA, Marino RJ. Medical rehabilitation length of stay and outcomes for persons with traumatic spinal cord injury–1990-1997. *Arch Phys Med Rehabil* 1999;80:1457-63.
29. Rochon PA, Beaudet MP, McGlinchey-Berroth R, et al. Risk assessment for pressure ulcers: an adaptation of the National Pressure Ulcer Advisory Panel risk factors to spinal cord injured patients. *J Am Paraplegia Soc* 1993;16:169-77.
30. Modirian E, Pirouzi P, Soroush M, Karbalaei-Esmaeili S, Shojaei H, Zamani H. Chronic pain after spinal cord injury: results of a long-term study. *Pain Med* 2010;11:1037-43.
31. Celik EC, Erhan B, Lakse E. The clinical characteristics of neuropathic pain in patients with spinal cord injury. *Spinal Cord* 2012;50:585-9.
32. Dijkers M, Bryce T, Zanca J. Prevalence of chronic pain after traumatic spinal cord injury: a systematic review. *J Rehabil Res Dev* 2009;46:13-29.
33. Furlan JC, Fehlings MG. The impact of age on mortality, impairment, and disability among adults with acute traumatic spinal cord injury. *J Neurotrauma* 2009;26:1707-17.
34. Teasell R, Allatt D. Managing the growing number of spinal cord-injured elderly. *Geriatrics* 1991;46:78-89.
35. Chu VW, Hornby TG, Schmit BD. Effect of antispastic drugs on motor reflexes and voluntary muscle contraction in incomplete spinal cord injury. *Arch Phys Med Rehabil* 2014;95(4):622-32.
36. Manella KJ, Field-Fote EC. Modulatory effects of locomotor training on extensor spasticity in individuals with motor-incomplete spinal cord injury. *Restor Neurol Neurosci* 2013;31:633-46.
37. Krassioukov A, Eng JJ, Claxton G, Sakakibara BM, Shum S. Neurogenic bowel management after spinal cord injury: a systematic review of the evidence. *Spinal Cord* 2010;48:718-33.
38. Jha A, Charlifue S. Aging in SCI. In: Kirshblum S, Campagnolo DI, editors. Spinal cord medicine. 2nd ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2010. p. 500-13.
39. Faaborg PM, Christensen P, Finnerup N, Laurberg S, Krogh K. The pattern of colorectal dysfunction changes with time since spinal cord injury. *Spinal Cord* 2008;46:234-8.
40. Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: clinical evaluation and rehabilitative management. *Arch Phys Med Rehabil* 1997;78:S86-102.
41. Lynch AC, Wong C, Anthony A, Dobbs BR, Frizelle FA. Bowel dysfunction following spinal cord injury: a description of bowel function in a spinal cord-injured population and comparison with age and gender matched controls. *Spinal Cord* 2000;38:717-23.
42. Weitzenkamp DA, Jones RH, Whiteneck GG, Young DA. Ageing with spinal cord injury: cross-sectional and longitudinal effects. *Spinal Cord* 2001;39:301-9.
43. Bosch A, Stauffer ES, Nickel VL. Incomplete traumatic quadriplegia. A ten year review. *JAMA* 1971;216:473-8.
44. Hitzig SL, Tonack M, Campbell KA, et al. Secondary health complications in an aging Canadian spinal cord injury sample. *Am J Phys Med Rehabil* 2008;87:545-55.

45. Drake MJ, Cortina-Borja M, Savic G, Charlifue SW, Gardner BP. Prospective evaluation of urological effects of aging in chronic spinal cord injury by method of bladder management. *Neurourol Urodyn* 2005;24:111-6.
46. Madersbacher G, Oberwalder M. The elderly para- and tetraplegic: special aspects of the urological care. *Paraplegia* 1987;25:318-23.
47. Rabadi MH, Mayanna SK, Vincent AS. Predictors of mortality in veterans with traumatic spinal cord injury. *Spinal Cord* 2013;51:784-8.
48. Osterthun R, Post MW, van Asbeck FW, van Leeuwen CM, van Koppenhagen CF. Causes of death following spinal cord injury during inpatient rehabilitation and the first five years after discharge. A Dutch cohort study. *Spinal Cord* 2014;52:483-8.
49. Glaser RM. Arm exercise training for wheelchair users. *Med Sci Sports Exerc* 1989;21:S149-57.
50. Shiba S, Okawa H, Uenishi H, et al. Longitudinal changes in physical capacity over 20 years in athletes with spinal cord injury. *Arch Phys Med Rehabil* 2010;91:1262-6.

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# Cervical myelopathy due to atraumatic odontoid fracture in patients with rheumatoid arthritis: A case series

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CASE REPORT

# Cervical myelopathy due to atraumatic odontoid fracture in patients with rheumatoid arthritis: A case series

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**Abstract**

To highlight the risk of cervical myelopathy due to occult, atraumatic odontoid fracture in patients with rheumatoid arthritis, we retrospectively reviewed radiographic findings and clinical observations for 7 patients with this disorder. This fracture tends to occur in patients with long-lasting rheumatoid arthritis and to be misdiagnosed as simple atlantoaxial dislocation. Since this fracture causes multidirectional instability between C1 and C2 and is expected to have poor healing potential due to bone erosion and inadequate blood supply, posterior spinal arthrodesis surgery is indicated upon identification of the fracture to prevent myelopathy.

**Keywords**

Atlantoaxial instability, Atraumatic occult fracture, Myelopathy, Odontoid fracture, Rheumatoid arthritis

**History**

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## Introduction

Upper cervical spine lesions frequently occur in patients with rheumatoid arthritis (RA) [1]. Synovitis at C1–2 results in erosion of the odontoid process as well as rupture of the transverse ligament, leading to atlantoaxial instability and subluxation. Bone atrophy and erosion of the base of the odontoid process as well as instability at C1–2 may cause a fragility fracture of the odontoid process. The incidence of odontoid fracture in patients with RA, however, remains unclear. Although there are some case reports describing occult fractures of the odontoid process in patients with RA [2–4], it is important to emphasize the potential for these fractures because they can result in severe myelopathy.

The present study is a retrospective review of the clinical and radiologic findings, as well as the surgical outcomes of atraumatic odontoid fractures with myelopathy in patients with RA.

## Materials and methods

### Design

This is a retrospective case series review of seven RA patients with cervical myelopathy due to atraumatic odontoid fracture.

### Patients

All of the patients were treated at the Hokkaido University Hospital between 2007 and 2012. We identified and reviewed the medical records of seven patients who underwent posterior spinal arthrodesis surgery for atraumatic odontoid fracture with myelopathy.

### Assessment of clinical features and surgical outcomes of cervical myelopathy due to atraumatic odontoid fracture

We reviewed the medical records of the seven patients and obtained data regarding patient characteristics, symptoms, activities of daily living, and the clinical status of RA. The Ranawat classification of neurologic impairment was used to assess paralysis and walking disturbances. Imaging studies included plain radiograms, three-dimensional computed tomography (CT) angiograms, and magnetic resonance imaging (MRI) of the cervical spine and spinal cord.

## Results

From 2007 to 2012, a total of 47 RA patients underwent surgery for atlantoaxial instability. This includes 7 RA patients with cervical myelopathy due to atraumatic odontoid fracture (Tables 1 and 2). They were 1 male and 6 females, with an average age at surgery of 67 (range, 44–88 ) years. Despite trends suggesting older age at surgery and higher risk in female in the 7 patients with cervical myelopathy due to atraumatic odontoid fracture compared with the remaining 40 patients (average age at surgery 63 years, 12 males and 28 females), these differences were not statistically significant. The mean follow-up period was 3 (range, 1–6 ) years. Average duration of RA was 15 (range, 9–28 ) years and rheumatic inflammation was poorly controlled in three of seven patients due to resistance to medications.

The preoperative symptoms were neck and occipital pain and quadriplegia. Five of seven cases were initially diagnosed as atlantoaxial subluxation (AAS) by plain radiograms and odontoid fracture was identified after examining results of MRI and CT. Three of seven patients were unable to walk when they were admitted. Radiologic studies revealed posterior shift or instability of fractured odontoid process in the three non-ambulatory patients, indicating that posterior instability of odontoid process is associated with severity of myelopathy. None of the patients in this series had anomalies of vertebral artery but we found that anterior and posterior ascending arteries, which provide blood supply to the odontoid process [5,6], were not depicted in any patients of this series on CT angiograph. All seven patients underwent posterior decompression and fusion surgery with autogenous iliac bone graft. The Ranawat classification of neurologic deficits improved level after surgery in all patients except 1 patient (Patient 1). Postoperative deep wound infection occurred in 1 patient (Patient 5).

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Table 1. Patient characteristics, disease activity, and medications.

| Patient | Sex | Age at onset (years) | Duration of RA (years) | Steinblocker stage | Classification of functional status* | CRP (mg/mL) | ESR (mm/h) | MMP-3 (ng/mL) | Medications |
|---|---|---|---|---|---|---|---|---|---|
| Patient 1 | F | 78 | 15 | IV | IV | 1.7 | 74 | N/A | Etanercept, Prednisolone 10 mg/day (Resistant to Bucillamine, Salazosulfapyridine, and Tacrolimus) |
| Patient 2 | F | 58 | 10 | III | III | 0.02 | 20 | 192 | Tocilizumab, MTX 6 mg/w, Methylprednisolone 2 mg/d (Resistant to Etanercept and Infliximab) |
| Patient 3 | F | 75 | 12 | III | II | 0.24 | 16 | 79 | Tacrolimus 3 mg/d, Prednisolone 5 mg/d, (Intolerant to MTX and Adalimumab) |
| Patient 4 | F | 44 | 16 | IV | IV | 2.6 | N/A | 165 | Bucillamine 300 mg/d, Prednisolone 10 mg/d, (Intolerant to MTX) |
| Patient 5 | M | 88 | 9 | II | II | 2.7 | N/A | 409 | Tacrolimus 1 mg/d, Predonisolone 12.5 mg/d |
| Patient 6 | F | 55 | 16 | III | II | 0.2 | 7 | 246 | MTX 6 mg/w, Prednisolone 10 mg/d, Bucillamine 300 mg/d |
| Patient 7 | F | 71 | 28 | III | II | 0.5 | 28 | 96 | Bucillamine 300 mg/d, Prednisolone 5 mg/d |

*CRP* C-reactive protein, *ESR* erythrocyte sedimentation rate, *MMP-3* matrix metalloproteinase-3, *N/A* not available, *MTX* methotrexate.
*Functional status due to RA before onset of myelopathy.



Bone union at graft site was achieved within 1 year postoperatively in all 7 cases; however, none of the 7 cases achieved bone union at fracture site on X-ray or CT despite posterior fusion. One patient (Patient 4) required additional posterior decompression surgery for recurrent myelopathy due to adjacent segment degeneration 1 year after O–C3 fusion surgery. During follow-up, two patients died. Patient 1 died due to pneumonia 1 year after surgery and Patient 3 died due to cardiac arrest 6 years after surgery, respectively.

## Case presentations

### Patient 1

A 78-year-old woman with RA presented with neck pain and quadriplegia. She was resistant to conventional disease-modifying anti-rheumatic drugs. She suffered from neck pain for 1 year and was diagnosed with an AAS 1 year prior to the onset of myelopathy. Her neck pain worsened without any history of trauma 7 days before presenting to our clinic. Five days after the exacerbation of her neck pain, she became unable to stand or even sit unassisted. The patient was then referred to our clinic for evaluation and treatment for AAS.

On admission, she exhibited severe quadriplegia and astasia. Neurologic examination revealed motor weakness and sensory disturbance in all four extremities, clumsy hand movements, increased deep tendon reflex of the elbows and knees, and a positive Babinski's reflex.

Plain radiography revealed posterior dislocation of the odontoid process and C1 (Figure 1A–D). CT scanning revealed an odontoid fracture with extensive erosion of the fractured region (Figure 1E and F). MRI showed impingement of the spinal cord by the fractured odontoid process, periodontoid granulation tissue, and C1 posterior arch (Figure 1G).

The patient underwent a posterior occipitocervical (O–C2) fixation and C1 laminectomy (Figure 1I). Her neck pain was relieved immediately after surgery. Her motor weakness gradually improved, and she was able to sit, stand, and transfer to a wheelchair on her own at 4 weeks after surgery. Although the Ranawat classification of her neurologic deficits remained Class IIIb, her activities of daily living were recovered to the level prior to the onset of myelopathy. The patient died due to pneumonia 1 year after surgery.

### Patient 3

A 75-year-old woman with a 12-year history of RA presented with neck and occipital pain, numbness in her feet, and a slight gait disturbance 3 months before admission to our clinic. She had been treated with Tacrolimus for 6 years. She was diagnosed with asymptomatic AAS 4 years before the onset of her symptoms.

On admission, she exhibited an unsteady gait. Neurologic examination revealed a hyperactive deep tendon reflex in her elbows, knees, and ankles, and motor weakness in both lower extremities. Her fingers and feet were hypoalgesic. Plain radiograms revealed anterior dislocation of the C1 and gross instability between C1 and C2 (Figure 2A–F). CT revealed a fracture of the odontoid process and vertical subluxation of C2 (Figure 2G and H). MRI showed spinal cord impingement by the fractured odontoid process and C1 posterior arch (Figure 2I).

The patient underwent posterior occipitocervical (O–C2) fusion surgery to correct the vertical subluxation (Figure 2J). Her neck pain was relieved and her motor function recovered from Ranawat Class IIIa to Class II after the surgery. She died due to cardiac arrest 6 years after surgery, at which time her motor function was sustained.

Table 2. Duration of neck pain, neurologic impairment, and surgical procedures.

| Patient | Duration of neck pain (months) | Ranawat classification of neurologic impairment: Preop | Follow-up | Posterior shift or instability of odontoid process | Surgical procedure (fusion levels) |
|---|---|---|---|---|---|
| Patient 1 | 12 | IIIB | IIIA | + | O–C2 |
| Patient 2 | 24 | IIIA | II | + | C1–2 |
| Patient 3 | 3 | IIIA | II | − | O–C2 |
| Patient 4 | 8 | IIIB | IIIA | + | O–C3 |
| Patient 5 | 13 | IIIB | IIIA | + | O–C2 |
| Patient 6 | 24 | II | I | − | O–C2 |
| Patient 7 | 1 | II | I | − | O–C2 |

*O–C* occipitocervical.

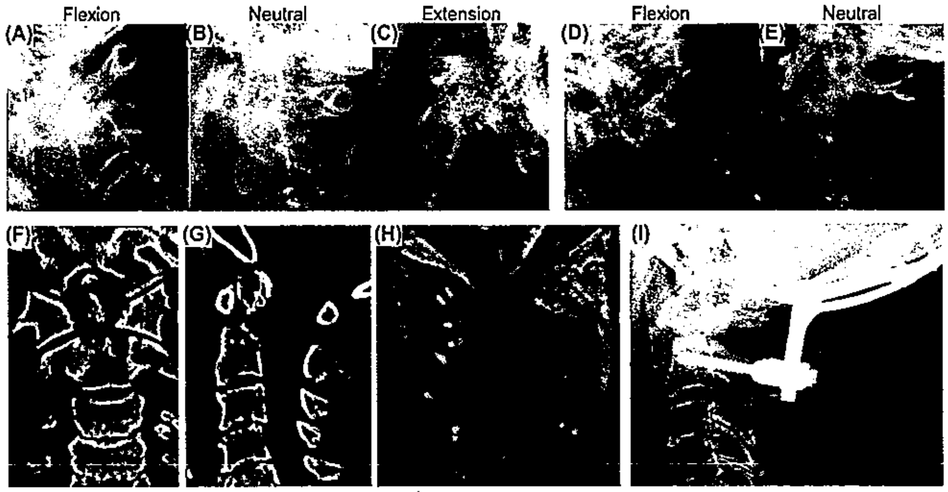


Figure 1. Patient 1. A 78-year-old woman with RA. Lateral plain radiographs in flexion (A), neutral position (B), and extension (C) at 1 year before the onset of myelopathy showing no odontoid fracture, but atlantoaxial subluxation. Lateral radiographs in flexion (D) and neutral position (E) after the onset of myelopathy, showing an odontoid fracture and posterior subluxation of C1 in neutral position. Coronal and sagittal CT scan reconstructions show the odontoid fracture and bone erosion at the base of the odontoid process (F and G). Sagittal T2-weighted MRI showing impingement of the spinal cord by the fractured odontoid process, periodontoid granulation tissue, and C1 posterior arch (H). The patient underwent a C1 laminectomy and posterior instrumented occiput–C2 fusion with an iliac bone graft (I).

### Patient 5

An 88-year-old man with RA presented with gait disturbance and restriction of fine finger motion. He had a 9-year history of RA. He had slight neck and occipital pain for 2 years. Numbness in his hands and fingers appeared without any history of trauma, and motor weakness in the upper and lower extremities developed gradually. Additionally, urinary incontinence and encopresis developed. The patient was diagnosed with AAS and referred to our clinic.

On admission, he exhibited quadriplegia and astasia. Neurologic examination revealed a hyperactive deep tendon reflex in the elbows, knees, and ankles, and motor weakness in all four extremities. All four extremities were hypoalgesic. He had bowel and bladder dysfunction. CT and MRI showed C1–2 instability with an odontoid fracture. The patient underwent O–C2 fusion surgery and his motor weakness was fully recovered after surgery. Deep wound infection occurred 2 weeks after surgery. Bacterial culture revealed that the infecting organism was *Corynebacterium* species. The patient underwent open irrigation surgery without removing the instrument and was treated with antibiotics for 3 weeks. The signs of infection disappeared. Additional bone graft surgery was performed 3 months after the first surgery.

### Discussion

Findings from this case series suggest that occult atraumatic fractures of the odontoid process should be suspected in patients with long-standing RA complaining of neck pain and myelopathy. There are some case reports describing occult odontoid fractures in patients with RA, but most reported cases had only vague neck pain [2,3], indicating that not all patients with atraumatic odontoid fractures develop myelopathy. Toyama et al. reported that fracture of odontoid process was seen in 6 of 58 RA patients (10%) with atlantoaxial subluxation, who underwent surgery, and that 2 of the 6 patients with spontaneous odontoid fractures had myelopathy [4]. This is not surprising because 75% of patients with traumatic odontoid fracture are reported to be neurologically intact [7]. Atraumatic odontoid fractures, however, may have a greater chance of causing myelopathy because odontoid fractures are likely to become non-union fractures, especially in patients with RA, and non-union odontoid fractures may cause progressive myelopathy [8].

Possible risk factors for atraumatic odontoid fractures with myelopathy include long-standing RA and poorly controlled rheumatic inflammation. Average duration of RA in this series was 15 years and rheumatic inflammation was poorly controlled in three of seven patients due to resistance to medications. Given that long-standing poorly controlled RA results in severe bone erosion of the odontoid process, atlantoaxial instability, and systemic bone loss, patients with long-standing RA have a potential risk for the onset of fragility odontoid fractures without major trauma.

Myelopathy caused by atraumatic odontoid fracture is likely to be misdiagnosed as AAS or exacerbation of polyarthritis. We think

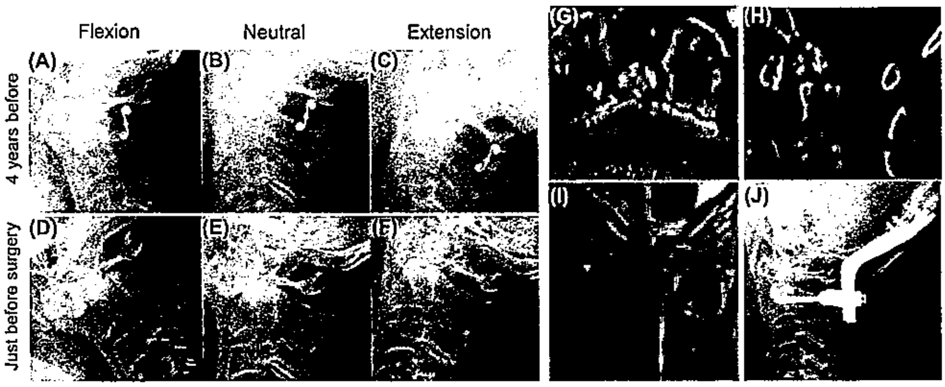


Figure 2. Patient 3. A 75-year-old woman with RA. Lateral plain radiographs in flexion (A), neutral position (B), and extension (C) demonstrating mild AAS, but no odontoid fracture 4 years before the onset of myelopathy. Lateral radiographs in flexion (D), neutral position (E), and extension (F) after the onset of myelopathy showing odontoid fracture and gross instability at C1–2. Coronal and sagittal CT reconstruction images demonstrating the odontoid fracture and vertical and anterior subluxation at C1–2 (G and H). Sagittal T2-weighted MRI image of the upper cervical region showing spinal cord compression by the base of odontoid process and C1 posterior arch (I). The patient underwent posterior correction and occiput–cervical fusion with an iliac bone graft (J).

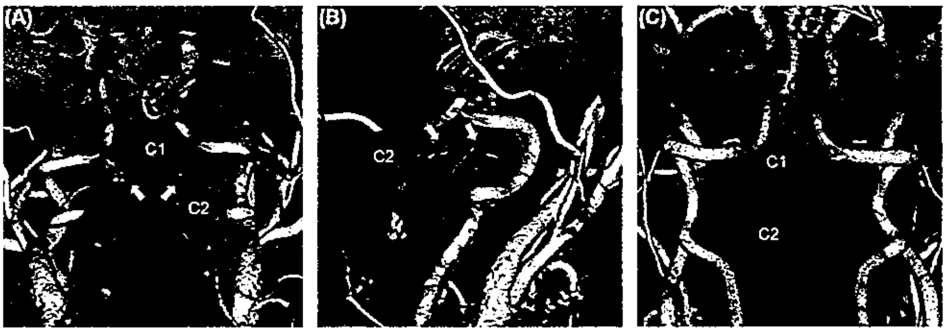


Figure 3. Poor arterial blood supply of the odontoid process in patients with an atraumatic odontoid fracture. Examples of three-dimensional CT angiography images in the posterior–anterior view (A) and oblique view (B) depicting anterior and posterior ascending arteries of the odontoid process (white arrows) in a patient with early RA. Three-dimensional CT angiogram of Patient 2 showing no ascending arteries of the odontoid process (C). Ascending arteries of the odontoid process were not observed in any patients of this series on CT angiography.



that myelopathy is caused by the progression of AAS, because AAS is very common in RA [1], while myelopathy caused by atraumatic odontoid fractures is rare. This is a very important point because the examination as well as treatment for AAS may be harmful to the spinal cord in cases of odontoid fracture. In most cases of AAS, cervical extension is safe because anterior subluxation can be reduced by extension and posterior subluxation of the C1 can be blocked by the odontoid process. On the other hand, in cases of odontoid fracture, cervical extension could cause posterior displacement of the fractured odontoid process, which compresses the spinal cord. Therefore, when patients with RA show abnormal neurologic findings on examination and posterior displacement of the C1 on X-ray, MRI and CT are indicated prior to functional plain X-ray examination.

Posterior spinal arthrodesis surgery is indicated for atraumatic odontoid fractures in patients with RA. Osteosynthesis through the anterior approach is not recommended because fracture union is difficult to achieve in these cases. None of the 7 cases in this study achieved fracture union despite posterior fusion. Possible reasons for this are bone erosion at the fracture site due to chronic inflammation and poor blood supply, which impair bone healing. We could not observe anterior and posterior ascending arteries, which provide blood supply to the odontoid process [5,6], in any patients of this series on CT angiography, supporting the idea that poor arterial blood supply to the fractured odontoid process impairs healing (Figure 3).

The difference between traumatic odontoid fractures and atraumatic odontoid fractures in terms of the surgical procedure is that traumatic odontoid fractures can be stabilized by direct anterior screw fixation as in osteosynthesis [9,10] or posterior C1–2 arthrodesis [11], but atraumatic odontoid fractures tend to require posterior occipitocervical (O–C) fusion. As described, atraumatic odontoid fractures occur in patients with long-standing poorly controlled RA and many of them have destructive bone lesions in the upper cervical spine. If patients have vertical instability, destruction of the C1–2 joints, or a retro-odontoid pannus formation that requires C1 laminectomy for spinal cord decompression, O–C fusion is indicated. Indeed, six of seven patients in this series required O–C fusion.

In conclusion, atraumatic odontoid fractures should be suspected in patients with long-standing RA complaining of neck pain and myelopathy. This condition is likely to be misdiagnosed as AAS or exacerbation of polyarthritis. Since this fracture causes multidirectional instability between C1 and C2, examination as well as treatment should be carefully performed to prevent deterioration of the myelopathy. Posterior spinal arthrodesis surgery is indicated for this fracture.

## Conflict of interest

None.

## References

1. Dreyer SJ, Boden SD. Natural history of rheumatoid arthritis of the cervical spine. Clin Orthop Relat Res. 1999;366:98–106.
2. Al Khayer A, Sawant N, Emberton P, Sell PJ. Spontaneous odontoid process fracture in rheumatoid arthritis: diagnostic difficulties, pathology and treatment. Injury. 2006;37(7):659–62.
3. Lewandrowski KU, Park PP, Baron JM, Curtin SL. Atraumatic odontoid fractures in patients with rheumatoid arthritis. Spine J. 2006;6(5):529–33.
4. Toyama Y, Hirabayashi K, Fujimura Y, Satomi K. Spontaneous fracture of the odontoid process in rheumatoid arthritis. Spine. 1992;17(10 Suppl):S436–41.
5. Anderson LD, D'Alonzo RT. Fractures of the odontoid process of the axis. J Bone Joint Surg Am. 1974;56(8):1663–74.
6. Crockard HA, Heilman AE, Stevens JM. Progressive myelopathy secondary to odontoid fractures: clinical, radiological, and surgical features. J Neurosurg. 1993;78(4):579–86.
7. Althoff B, Goldie IF. The arterial supply of the odontoid process of the axis. Acta Orthop Scand. 1977;48(6):622–9.
8. Schiff DC, Parke WW. The arterial supply of the odontoid process. J Bone Joint Surg Am. 1973;55(7):1450–6.
9. Bohler J. Anterior stabilization for acute fractures and non-unions of the dens. J Bone Joint Surg Am. 1982;64(1):18–27.
10. Borne GM, Bedou GL, Pinaudeau M, Cristino G, Hussein A. Odontoid process fracture osteosynthesis with a direct screw fixation technique in nine consecutive cases. J Neurosurg. 1988;68(2):223–6.
11. Jeanneret B, Magerl F. Primary posterior fusion C1/2 in odontoid fractures: indications, technique, and results of transarticular screw fixation. J Spinal Disord. 1992;5(4):464–75.

# *Optimum pelvic incidence minus lumbar lordosis value can be determined by individual pelvic incidence*

**Satoshi Inami, Hiroshi Moridaira, Daisaku Takeuchi, Yo Shiba, Yutaka Nohara & Hiroshi Taneichi**

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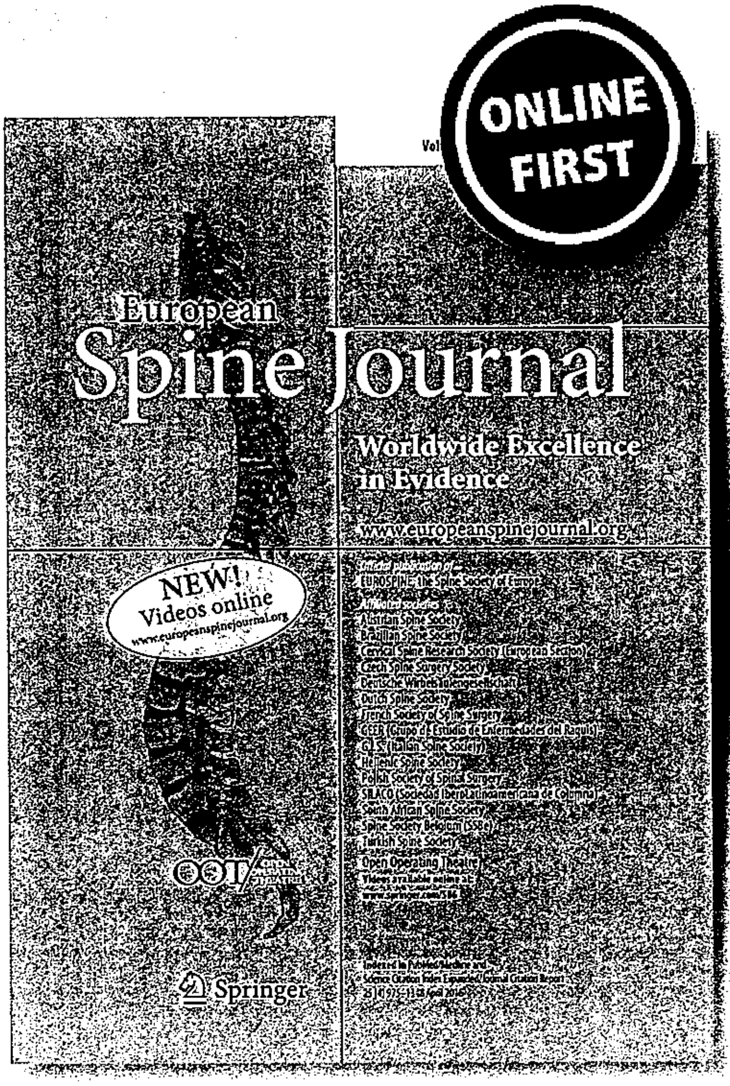


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ORIGINAL ARTICLE

# Optimum pelvic incidence minus lumbar lordosis value can be determined by individual pelvic incidence

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**Abstract**
*Purpose* Adult spinal deformity (ASD) classification showing that ideal pelvic incidence minus lumbar lordosis (PI-LL) value is within 10° has been received widely. But no study has focused on the optimum level of PI-LL value that reflects wide variety in PI among patients. This study was conducted to determine the optimum PI-LL value specific to an individual's PI in postoperative ASD patients.
*Methods* 48 postoperative ASD patients were recruited. Spino-pelvic parameters and Oswestry Disability Index (ODI) were measured at the final follow-up. Factors associated with good clinical results were determined by stepwise multiple regression model using the ODI. The patients with ODI under the 75th percentile cutoff were designated into the "good" health related quality of life (HRQOL) group. In this group, the relationship between the PI-LL and PI was assessed by regression analysis.
*Results* Multiple regression analysis revealed PI-LL as significant parameters associated with ODI. Thirty-six patients with an ODI <22 points (75th percentile cutoff) were categorized into a good HRQOL group, and linear regression models demonstrated the following equation: PI-LL = 0.41PI–11.12 ($r = 0.45$, $P = 0.0059$).
*Conclusions* On the basis of this equation, in the patients with a PI = 50°, the PI-LL is 9°. Whereas in those with a PI = 30°, the optimum PI-LL is calculated to be as low as 1°. In those with a PI = 80°, PI-LL is estimated at 22°. Consequently, an optimum PI-LL is inconsistent in that it depends on the individual PI.

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**Keywords** Adult spinal deformity · Sagittal alignment · Pelvic incidence · Lumbar lordosis

## Introduction

Adult spinal deformity (ASD) is often associated with sagittal plane malalignment [1, 2]. The correlations between sagittal radiographic parameters and self-reported pain and disability have been shown in several studies, and it has been demonstrated that sagittal plane malalignment results in the disability most consistently expressed in ASD patients [3–6].

Numerous studies have been conducted to better understand the ideal sagittal global alignment, including the pelvis. Since the work by Duval-Beaupère et al. several authors have attempted to clarify that pelvic incidence (PI) is primary importance in regulating sagittal spinal alignment, and the relationship between lumbar lordosis (LL) and PI is admitted [7–9].

Nowadays, in an effort to achieve patient-specific alignment treatment, some formulas have been developed to calculate the ideal LL matching with the PI, which helps in surgical planning when deciding the amount of correction needed. The Scoliosis Research Society (SRS)-Schwab classification has a global classification paradigm with three sagittal modifiers: PI minus LL (PI-LL), sagittal vertical axis (SVA), and pelvic tilt (PT). These parameters were classified into three categories (i.e., non-pathological, moderate, and marked) on the basis of health-related quality of life (HRQOL) [10]. In this classification, the non-pathological cutoff value of PI-LL is within 10°, and a

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excessive PI-LL mismatch is considered as a possible risk factor for adjacent segment disease and lumbopelvic fixation failure [11, 12].

To improve knowledge about spinopelvic harmonized alignment, the question to consider is whether or not the PI-LL value for sagittal balance is fixed (i.e., <10), without taking of a variety of PI values into account. The PI value varies widely among individuals. It has been demonstrated in a past study that a lower value of PI is approximately 30° and a higher value is approximately 85° [13–16]. PI determines the relative position between the sacral plate and femoral heads, and it has been shown that the PI is related to the capacity to compensate for sagittal balance through pelvic retroversion [13]. In our clinical experience, patients with a large PI sometimes have good surgical results, even with a postoperative PI-LL >10° (Fig. 1). Additionally, Yamada et al. reported that 23 % of postoperative patients acquired good SVA and satisfaction nevertheless they had PI-LL >10° [17]. An optimal formula that would incorporate these factors has not yet been proposed.

## Materials and methods

In this study, we intended to investigate the relationship between PI-LL and PI in postoperative ASD patients and to determine the optimum PI-LL, specific to the individual PI value, in patients who had undergone successful surgery. Our hypothesis is that the optimal PI-LL is not constant, but changes based upon individual differences in PI.

This is a single-institutional retrospective study with institutional review board approval of consecutive patients undergoing surgery for ASD between the years 2007 and 2012. Inclusion criteria for this study were as follows: adult patients (>40 years old) presenting pain/disability caused by spinal deformity; available complete coronal and sagittal radiography from preoperative visits to final follow-up, and complete health related quality of life (HRQOL) questionnaires in final follow-up.

### Data collection and radiographic measurement

All patients were observed for at least 2 years. Demographic and surgical data were retrospectively recorded from clinical charts. Preoperative and final follow-up (minimum 2 years) radiographic data consisted of full-length standing coronal and sagittal radiographs obtained in the freestanding position with fingers on clavicles and shoulders in 45° of forward elevation [18]. All radiographic measurements were made by digital analysis, using the Centricity™ Enterprise Web, version 3.0 (GE Healthcare Japan, Tokyo).

Spinal radiographic measurements included T5–T12 thoracic kyphosis (TK), T10–L2 thoracolumbar kyphosis (TLK), T12–S1 lumbar lordosis (LL), SVA, maximal coronal Cobb angle (scoliosis), and coronal balance, i.e., the distance between C7 plumb line and central sacral vertical line. Pelvic morphology and orientation were measured by PT, sacral slope (SS), and PI. PI-LL was also calculated from these data. The Oswestry Disability Index (ODI) at final follow-up was investigated for HRQOL evaluation.

### Statistical analysis

Stepwise multiple regression analysis was used to determine the factors associated with good clinical results using the ODI score as the dependent variable and the above-mentioned radiographic parameters as independent variables. Patients with a postoperative ODI value less than the 75th percentile cutoff were designated into the "good" HRQOL group. Then, in this group, the relationship between the PI-LL and PI was assessed by linear regression analysis to determine the optimum PI-LL value based upon the individual PI. Furthermore, post hoc power analysis was performed, using G*Power (free program; http://www.gpower.hhu.de/) to estimate the effect size that could be detected with a sample size of good HRQOL group. The level of significance was set at 0.05.

## Results

### Patient data

The patients included 35 women and 13 men, with a mean age of 60.6 years (SD 9.7 years; range 40–75 years). Categorization by SRS-Schwab classification system was marked deformity in 43 patients, moderate deformity in three patients, in at least one sagittal modifier. And coronal curve types were TL/lumbar only ($n = 22$), double curve ($n = 7$), and no major coronal deformity ($n = 19$).

Forty-two patients had a posterior-only spinal fusion, five had an anterior and posterior spinal fusion, and one had an anterior fusion. Osteotomies included pedicle subtraction osteotomy ($n = 13$), vertebral column resection ($n = 2$), and Ponte osteotomy ($n = 27$). The most common uppermost instrumented vertebrae (UIV) were T10 ($n = 34$), followed by L2 ($n = 4$), T11 and T12 ($n = 2$ each), and T3, T4, T7, L1, and L3 ($n = 1$ each). The most common lowest instrumented vertebrae (LIV) were S1 ($n = 30$), followed by L5 ($n = 14$), and L3 and L4 ($n = 2$ each).

### Preoperative versus final follow-up radiographic findings

The deformity correction surgery led to an increase in TK (from 14° preoperatively to 30° at follow-up), a decrease in

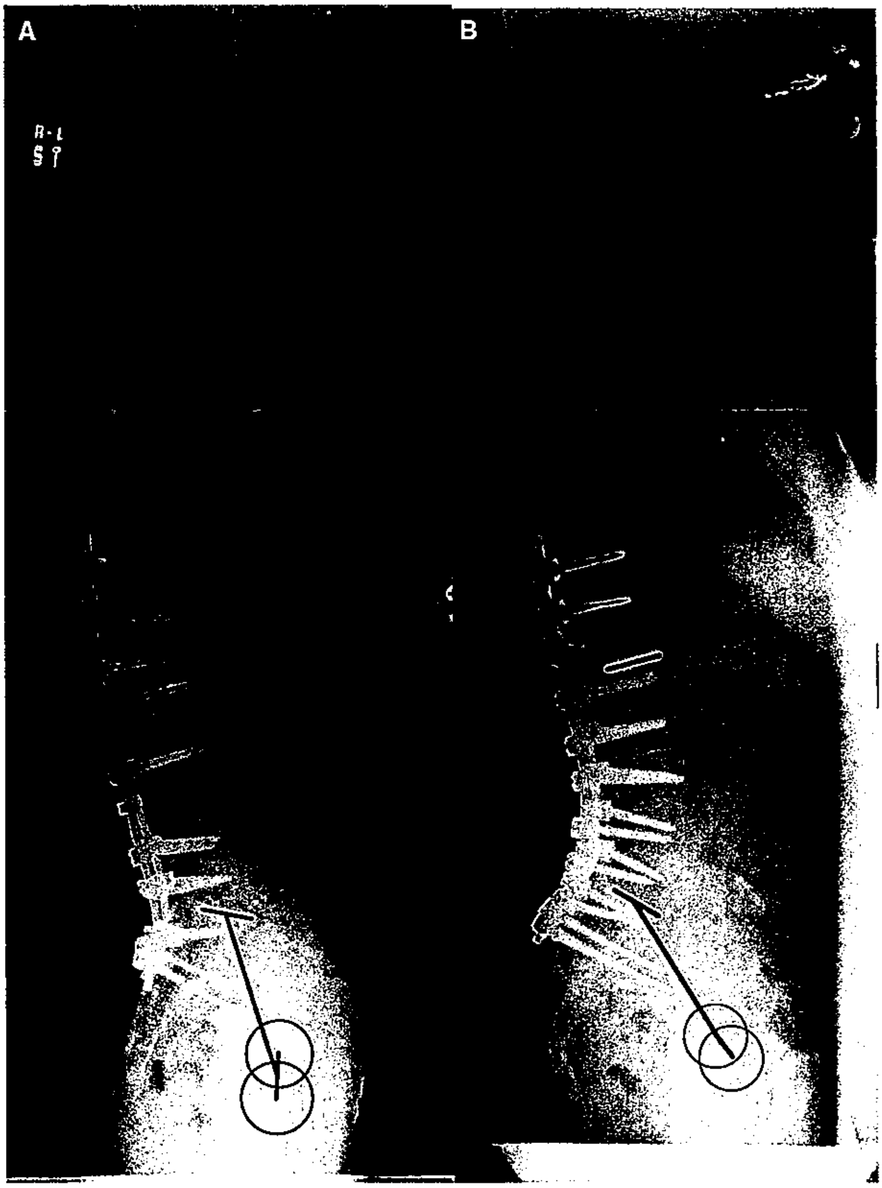


**Fig. 1** A wide variety of postoperative sagittal alignment among the patients with good surgical results. Sagittal postoperative radiograph shows a case with PI-LL of 2° (PI = 29°, LL = 27°), ODI score of 10 points (a). Sagittal postoperative radiograph shows a case with PI-LL of 21° (PI = 65°, LL = 44°), ODI score of 16 points (b)

TLK (from 20° preoperative to 9° at follow-up), and an increase in LL (from 6° preoperative to 39° at follow-up). PI was 49° preoperatively and 50° at follow-up. PT decreased at follow-up (from 33° to 25°). PI-LL decreased at follow-up (from 43° to 11°), and SVA decreased at follow-up (102–37 mm) (Table 1).

### QOL analysis

The median ODI score improved from 47.5 points (range 20–66) before surgery to 14.4 points (range 0–35.6) at follow-up (Table 2).

### Factors associated with good clinical outcomes

Multiple regression analysis revealed TK ($P = 0.00278$), PT ($P = 0.0339$) and PI-LL ($P = 0.0001$) as the radiographic parameters most significantly associated with ODI (Table 3).

### Relationship between PI-LL and PI

When subdividing the study population into the good HRQOL group by ODI score of less than 22 points (75th percentile cutoff) (Table 2), 36 patients were classified into

**Table 1** Spinopelvic parameters between preoperative and final follow-up

| | Preoperative (mean ± SD) | Final follow-up (mean ± SD) |
|---|---|---|
| Thoracic kyphosis (°) | 13.9 ± 15 | 30 ± 13.9 |
| Thoraco-lumbar kyphosis (°) | 19.9 ± 14 | 9.2 ± 7.5 |
| Lumbar lordosis (°) | 6.4 ± 21.7 | 38.6 ± 11 |
| Pelvic tilt (°) | 32.8 ± 13.1 | 24.9 ± 11.4 |
| Sacral slope (°) | 16.6 ± 12.9 | 24.8 ± 8.6 |
| Pelvic incidence (°) | 49.4 ± 11.2 | 49.7 ± 11.4 |
| PI-LL (°) | 43 ± 21.1 | 11 ± 12.6 |
| Sagittal vertical axis (mm) | 102.3 ± 69.5 | 36.6 ± 44.6 |
| Scoliosis (°) | 49.2 ± 16 | 13.3 ± 10.2 |
| Coronal balance (mm) | 41.4 ± 33.4 | 11.3 ± 9 |

**Table 2** Distribution of ODI score at final follow-up

| | Minimum | 25-percentile | Median | 75-percentile | Maximum |
|---|---|---|---|---|---|
| ODI (points) | 0 | 6.7 | 14.4 | 22.2 | 35.6 |

**Table 3** Stepwise multiple regression analysis

| Variables | *P* |
|---|---|
| Thoracic kyphosis | 0.0028* |
| Thoraco-lumbar kyphosis | 0.7375 |
| Lumbar lordosis | 0.3194 |
| Pelvic tilt | 0.0339* |
| Sacral slope | 0.3194 |
| PI-LL | 0.0001* |
| Sagittal vertical axis | 0.7577 |
| Coronal balance | 0.2836 |

* $P < 0.05$

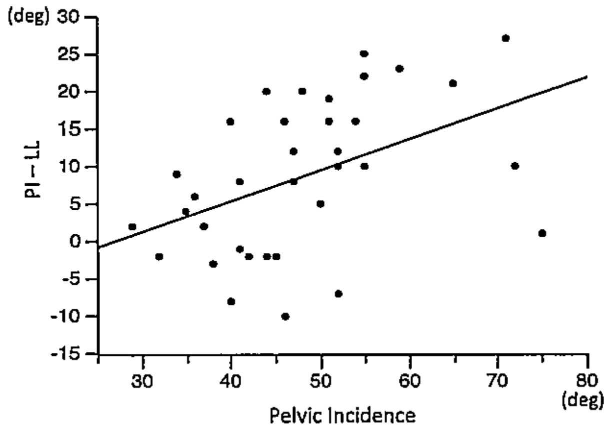


**Fig. 2** Relationship between the PI-LL and pelvic incidence in the good HRQOL group

the good HRQOL group. In this group, a significant association was noted between the PI-LL and PI with a linear regression model leading to the following equation (Fig. 2):

$$\mathrm{PI} - \mathrm{LL} = 0.41 \times \mathrm{PI} - 11.12 (r = 0.45, P = 0.0059).$$

Parameters used for post hoc power analysis were, effect size = 0.45, two tailed alpha = 0.05, sample size = 36. The power was calculated as 0.8066.

## Discussion

Several authors have established equations for predicting LL from pelvic and spinal parameters [8, 9, 19–22] and have improved surgical planning for the treatment of ASD. On the other hand, their efficacy and clinical relevance remain in question throughout the postoperative period; in particular, how would patients respond to a given LL predicted before surgery, because the subjects of these studies are healthy adult population [8, 9] or preoperative ASD patients [19]. Furthermore, even the formulas calculated for postoperative ASD patients did not incorporate the QOL outcome [21, 22]. To answer these questions, we intended to establish a formula from postoperative patients with successful outcomes.

Our findings showed that the PI-LL is a factor that was consistently associated with HRQOL during the postoperative period; and significant correlation was shown between PI-LL and PI. On the basis of the above-mentioned equation, we propose that an optimal PI-LL is inconsistent in that it depends on the individual PI. For example, in patients with a normal PI (PI = 50°), the optimum PI-LL is 9°, which corresponds to a normal value in the established classification system [10], whereas, in those with a small PI (PI = 30°), the optimum PI-LL is calculated to be as low as 1°. Then, in those with a large PI (PI = 80°), the ideal PI-LL is estimated at 22°.

Consequently, a smaller PI results in a smaller PI-LL, i.e., close to or equivalent to 0°, while a larger PI allows for a larger PI-LL, approximately 20°.

Other studies provide solid rationale to explain our results particularly, that an optimal PI-LL is not constant but variable, depending upon the individual PI. Duval-Beaupère et al. have expressed that low values of PI decrease the chance of an economical standing posture, because this induces a short lever arm of the hip extensor muscle [7]. Additionally, they have shown in their clinical experience that there are constant action potentials of the gluteus maximus in patients with a small PI in the standing position. This standing condition might suggest a tendency to fall into a non-economical standing condition. Roussouly et al. advocated that the ability to retrovert the pelvis is limited by a patient's own PI, and patients with a small PI have a small capacity to compensate for their sagittal balance through pelvic retroversion [23]. For reasons mentioned above, it seems reasonable to suppose that a small PI demands a strictly matched LL.

In contrast, a patient with a large PI has a long lever arm of the hip extensor muscle, and furthermore has a wide posterior offset between the sacrum and the femoral heads in the sagittal plane [13, 23]. Legaye et al. defined the overhanging of S1 as the distance between the bi-coxofemoral axis and the projection to this level of the midpoint of the sacral plate, and reported a significant correlation between the PI and the overhanging of S1; i.e., the larger the value of the PI, the larger the value of the adapted overhanging of S1 [8]. These mechanisms and anatomical features of patients with a large PI may help to keep the position of the C7 plumb line behind the femoral heads, and it is reasonable to think that a large PI value would allow a larger spinopelvic mismatch, even if the PI-LL is above 10.

As PI-LL is one of the most important indicators for successful corrective surgery [10], we aimed to determine the optimum value of the PI-LL in the specific setting of an individual's PI value. This is the reason why the PI-LL was selected as a dependent variable in the following linear equation: $\mathrm{PI\text{-}LL} = 0.41\mathrm{PI}-11.12$. Whereas, this equation could be rearranged simply as: $\mathrm{LL} = 0.59\mathrm{PI} + 11.12$.

Legaye et al. have studied the correlation between the pelvic and spinal sagittal parameters and have developed a formula indicating an ideal LL by using the PI [24]:

$$\begin{aligned}\mathrm{LL} &= ((0.5481\mathrm{PI} + 12.7) \times 1.087 + 21.61)\\ &= 0.596\mathrm{PI} + 35.415\end{aligned}$$

The important point to note is that these two linear equations have a common factor (i.e., the coefficient is approximately 0.6). We would like to emphasize that the rise of the ideal LL (y-axis) is only 60 % of every increase in PI (x-axis) in these equations, whereas the value of the y-intercept is different between the Eqs. (11 versus 35), and we speculate that the main cause is the difference in the material population, i.e., the Legaye et al. study involved normal volunteers without surgery (mean age 24 years), whereas the current study included postoperative ASD patients (mean age 61 years) with successful outcomes.

The results of this study show that a smaller PI results in a smaller PI-LL, while a larger PI allows for a larger PI-LL. Our conclusion is that the optimal PI-LL is not a fixed but rather a flexible value reflecting the specific PI of the individual. We argue that this data will have a significant clinical impact on patient-specific surgical planning for procedures aimed at increasing lordosis in the lumbar spine. The majority of UIV in this study were below T10, therefore there would be a potential intraspinal compensatory mechanism [25]. Thus, a limitation of this study is that the applicability to patients with instrumentation that includes the upper thoracic spine is unknown. For the patients with thoracic malalignment being required correction, surgical planning including realignment of thoracic spine would be effective [26]. In addition, not only magnitude but also shape of LL [23] that represents geometry of lordosis should have been evaluated in the present study. Although significant correlations between PI and LL have been reported in the several manuscripts including the present study [8, 9, 24], PI can be influenced by the other parameters such as PT, SS, and so on [7]. Furthermore, LL can also be affected by TK as a reciprocal mechanism [25]. Therefor, newly developed formulas regarding the relationship between PI and LL should receive careful validation by future investigations.

Some might argue that the formula is based on only 36 patients with successful outcomes and HRQOL was evaluated by only ODI, so that the results should not be applied to all ASD patients. No doubt that higher value for power with a bigger sample size would be useful to have a good chance of detecting the effect, however power analysis in the current study calculated a power of 80 % that is the common acceptable level to detect the effect. In regarding to HROQL, evaluation with multiple instruments could bring more detailed information of disability; yet, the reason why ODI was used in the current study is that non-pathological cutoff value of radiographic parameters (i.e., PI-LL, SVA, PT, etc.) has been evaluated based on ODI in past studies [4, 5, 10, 16, 19]. It follows from what has been said that our findings would afford new perspectives on surgical planning for LL correction. Future studies addressing to the shape of LL [23] with more patients and multiple HRQOL evaluations will assist in clarifying the interaction between PI and LL in the setting of surgical treatment.

## Conclusion

Our findings show that the PI-LL was consistently associated with a patient's quality of life during the postoperative period, and a significant correlation was shown between the PI-LL and PI. On the basis of our equation, we demonstrate that an optimal PI-LL value is not a constant value, e.g., the PI-LL value is 1° with a PI of 30°, whereas the value is 22° with a PI of 80°. Our study confirmed our hypothesis that an optimal PI-LL value is not constant and changes based upon the difference in PI value, and will have a clinical impact in patient-specific surgical planning to make lordosis in lumbar spine.

**Compliance with ethical standards**

**Conflict of interest** None.

## References


1. Schwab F, Smith VA, Biserni M, Gamez L, Farcy JP, Pagala M (2002) Adult scoliosis: a quantitative radiographic and clinical analysis. Spine 27:387–392
2. Gelb DE, Lenke LG, Bridwell KH, Blanke K, McEnery KW (1995) An analysis of sagittal spinal alignment in 100 asymptomatic middle and older aged volunteers. Spine 20:1351–1358
3. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F (2005) The impact of positive sagittal balance in adult spinal deformity. Spine 30:2024–2029
4. Lafage V, Schwab F, Patel A, Hawkinson N, Farcy JP (2009) Pelvic tilt and truncal inclination: two key radiographic parameters in the setting of adults with spinal deformity. Spine 34:E599–E606
5. Protopsaltis T, Schwab F, Bronsard N, Smith JS, Klineberg E, Mundis G et al (2014) The T1 pelvic angle, a novel radiographic measure of global sagittal deformity, accounts for both spinal inclination and pelvic tilt and correlates with health-related quality of life. J Bone Joint Surg Am 96:1631–1640
6. Schwab F, Farcy JP, Bridwell K, Berven S, Glassman S, Harrast J et al (2006) A clinical impact classification of scoliosis in the adult. Spine 31:2109–2114
7. Duval-Beaupère G, Schmidt C, Cosson P (1992) A barycentremetric study of the sagittal shape of spine and pelvis: the conditions required for an economic standing position. Ann of Biomed Eng 20:451–462
8. Legaye J, Duval-Beaupère G, Hecquet J, Marty C (1998) Pelvic incidence: a fundamental pelvic parameter for three-dimensional regulation of spinal sagittal curves. Eur Spine J 7:99–103
9. Boulay C, Tardieu C, Hecquet J, Benaim C, Mouilleseaux B, Marty C et al (2006) Sagittal alignment of spine and pelvis regulated by pelvic incidence: standard values and prediction of lordosis. Eur Spine J 15:415–422
10. Schwab F, Ungar B, Blondel B, Buchowski J, Coe J, Deinlein D et al (2012) Scoliosis research society-Schwab adult spinal deformity classification: a validation study. Spine 37:1077–1082
11. Rothenfluh DA, Mueller DA, Rothenfluh E, Min K (2015) Pelvic incidence-lumbar lordosis mismatch predispose to adjacent segment disease after lumbar spinal fusion. Eur Spine J 24:1251–1258
12. Cho W, Mason JR, Smith JS, Shimer AL, Wilson AS, Shaffrey CI (2013) Failure of lumbopelvic fixation after long construct fusion in patients with adult spinal deformity: clinical and radiographic risk factors. J Neurosurg Spine 19:445–453
13. Le Huec JC, Aunoble S, Philippe L, Nicolas P (2011) Pelvic parameters: origin and significance. Eur Spine J 20:S564–S571
14. Roussouly P, Gollogly S, Noseda O, Berthonnaud E, Dimnet J (2006) The vertical projection of the sum of the ground reactive forces of a standing patient is not the same as the C7 plumb line: a radiographic study of the sagittal alignment of the 153 asymptomatic volunteers. Spine 31:E320–E325
15. Berthonnaud E, Dimnet J, Roussouly P, Labella H (2005) Analysis of the sagittal balance of the spine and pelvis using shape and orientation parameters. J Spinal Disord Tech 18:40–47
16. Schwab F, Patel A, Ungar B, Farcy JP, Lafage V (2010) Adult spinal deformity-postoperative standing imbalance: how much can you tolerate? An overview of key parameters in assessing alignment and planning corrective surgery. Spine 35:2224–2231
17. Yamada K, Abe Y, Yanagibashi Y, Hyakumachi T, Satoh S (2015) Mid- and long-term clinical outcomes of corrective fusion surgery which did not achieve sufficient pelvic incidence minus lumbar lordosis value for adult spinal deformity. Scoliosis 10:S17
18. Horton WC, Brown CW, Bridwell KH, Glassman SD, Suk SI, Cha CW (2005) Is there an optimal patient stance for obtaining a lateral 36″ radiograph? Acritical comparison of three techniques. Spine 30:427–433
19. Schwab F, Blondel B, Bess S, Hostin R, Shaffrey CI, Smith JS (2013) Radiographical spinopelvic parameters and disability in the setting of adult spinal deformity. Spine 38:E803–E812
20. Boissiere L, Bourghli A, Vital JM, Gille O, Obeid I (2013) The lumbar lordosis index: a new ratio to detect spinal malalignment with a therapeutic impact for sagittal balance correction decisions in adult scoliosis surgery. Eur Spine 22:1339–1345
21. Lafage V, Schwab F, Vira S, Patel A, Ungar B, Farcy JP (2011) Spino-pelvic parameter after surgery can be predicted. Spine 36:1037–1045
22. Rose PS, Bridwell KH, Lenke LG, Cronen GA, Mulconrey DS, Buchowski JM et al (2009) Role of pelvic incidence, thoracic kyphosis, and patient factors on sagittal plane correction following pedicle subtraction osteotomy. Spine 34:785–791
23. Roussouly P, Pinherio-Franco JL (2011) Biomechanical analysis of the spino-pelvic organization and adaptation in pathology. Eur Spine J 20:S609–S618
24. Legaye J, Duval-Beauere G (2005) Sagittal plane alignment of the spine and gravity: a radiological and clinical evaluation. Acta Orthop Belg 71:213–220
25. Lafage V, Ames C, Schwab F, Klineberg E, Akbania B, Smith J et al (2012) Change in thoracic kyphosis negatively impact sagittal alignment after lumbar pedicle subtraction osteotomy. Spine 37:E180–E187
26. Berjano P, Langella F, Ismael M-F, Damilano M, Scopetta S, Lamertina C (2014) Successful correction of sagittal imbalance can be calculated on the basis of pelvic incidence and age. Eur Spine J 23:S587–S596