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[Intervention Protocol]

# Anterior versus posterior spinal correction and fusion for adolescent idiopathic scoliosis

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## ABSTRACT

This is a protocol for a Cochrane Review (Intervention). The objectives are as follows:

To examine which intervention (anterior or posterior spinal correction and fusion) has the best functional clinical outcome (in terms of pain, participant satisfaction and function), the best radiographic results and the lowest complication rates in people with AIS.

## BACKGROUND

Clinical trials have shown that in anterior adolescent idiopathic scoliosis (AIS) surgery fewer fusion levels are needed and blood loss may be diminished compared with posterior AIS surgery (Lowe 2003). However, anterior surgery may impair pulmonary function (Graham 2000; Lenke 2004; Vedantam 2000; Wong 1996). Currently there are no (systematic) reviews comparing anterior versus posterior instrumentation and spinal correction and instrumented fusion for the surgical treatment of AIS.

### Description of the condition

Scoliosis is a multi-factorial, three-dimensional deformity of the spine and the trunk (Grivas 2006a; Grivas 2008a; Grivas 2008b). It occurs in 3% to 5% of the adolescent population (SOSORT 2012). The most common type of scoliosis (80%) is AIS, which presents shortly before, or during, puberty (Kanayama 1996; Stokes 1996). Approximately 10% of these diagnosed cases require conservative treatment and approximately 0.1% to 0.3% require operative correction of the deformity. Progression of AIS is much more frequently seen in females. When the Cobb angle is 10° to 20°, the ratio of affected girls to boys is similar (1.3 : 1), increasing to 5.4 : 1 for Cobb angles between 20° and 30°, and 7 : 1 for angle values above 30° (Lonstein 2006; Parent 2005). If the scoliosis angle at completion of growth exceeds a 'critical threshold' (most authors assume it to be between 30° and 50°), there is a higher risk of health problems in adult life, decreased quality of life, cosmetic deformity and visible disability, pain and progressive functional limitations (Lonstein 2006; Negrini 2006).

Most people with untreated AIS function at or near normal levels, even though pain is more prevalent. Self image is often slightly diminished. Mental health is usually normal (Asher 2006).

The aetiology of scoliosis has not been fully elucidated (Burwell 2000; Burwell 2009; Grivas 2002; Machida 1999; Sevastik 1997; Stokes 1997). Based on the variety of opinions on idiopathic scoliosis development, we can assume a multi-factorial origin (Burwell 2000; Burwell 2009; Grivas 2002; Nachemson 1977).

The potential for curve progression is related to several factors, including the person's gender, age, curve magnitude, bone maturity, rate of growth and growth potential at presentation. The pubertal growth spurt is the period of the most marked progression of AIS (Grivas 2006b; Wong 2005).

The assessment of the Cobb angle is essential for diagnosis, follow-up and evaluation of treatment results (Lonstein 1995).

The basic objectives of comprehensive conservative treatment of idiopathic scoliosis are:

1. to stop curve progression at puberty (or possibly even reduce it);
2. to prevent or treat respiratory dysfunction;
3. to prevent or treat spinal pain syndromes;
4. to improve aesthetics via postural correction (SOSORT 2012).

Curves up to 25° Cobb angle can be observed every six months in order to monitor progression of the curve. Immature people with curves 25° to 40° are at risk for curve progression and should be treated with a brace. In severe scoliosis with curves of more than

45°, surgery is often necessary. Surgical fusion of the curve is still the standard treatment.

Surgical treatment of idiopathic thoracolumbar scoliosis aims to correct the deformity and to regain or maintain trunk balance through as short a fusion as possible in order to prevent degeneration of the open discs underneath the fusion (Asher 2006; Goldberg 2002).

### Description of the intervention

In severe scoliosis with curves of more than 45°, surgery is often necessary. Surgical fusion of the curve is still the standard treatment. The aim of scoliosis surgery is to obtain a well-balanced spine, maximum correction of the scoliotic curves and maximum functionality of the vertebral column (Asher 2006; Goldberg 2002).

The spine can be approached from the anterior or posterior. The posterior approach is most commonly utilised. Posterior instrumentation and fusion disrupts the paraspinal musculature and may, therefore, have long-term health consequences with respect to back pain. The posterior approach often requires fusion to a more distal (caudal) extent than anterior surgery. The use of pedicle screws permits manipulation of the spine to a desired correction by cantilevering, segmental in situ bending, translation, direct vertebral rotation and incomplete rod rotation.

The anterior approach utilises instrumentation with vertebral-body screws, which are then connected by single or dual rods to gain correction. The correction manoeuvres are cantilevering and compression, although rod rotation has also been utilised. The advantage of anterior surgery for a selective fusion is that, in general, fewer motion segments are fused (Betz 1999; Lowe 2003; Sweet 1999). However, anterior surgery of the spine requires entry to the chest in thoracic scoliosis, or entry to the retroperitoneal space in lumbar scoliosis. This anterior approach may injure vital organs and structures such as the aorta and the lungs.

### How the intervention might work

The aim of AIS surgery is to prevent progression of the curve, to obtain a well-balanced spine with 1. maximum correction of the scoliotic curves, 2. maximum maximal functionality of the vertebral column and 3. minimal pain and complications.

### Why it is important to do this review

Clinical trials have shown that in anterior AIS surgery fewer fusion levels are needed and blood loss may be diminished compared with posterior AIS surgery (Delorme 2002; Lee 1997; Lowe 2003; Noordeen 1999; Sanders 1997). However, anterior surgery may impair pulmonary function (Graham 2000; Lenke 2004; Vedantam 2000; Wong 1996). Currently there are no (systematic) reviews comparing anterior versus posterior instrumentation and spinal correction and fusion for of AIS.

## OBJECTIVES

To examine which intervention (anterior or posterior spinal correction and fusion) has the best functional clinical outcome (in terms of pain, participant satisfaction and function), the best radiographic results and the lowest complication rates in people with AIS.

## METHODS

### Criteria for considering studies for this review

#### Types of studies

The primary analysis will combine the results of randomised controlled trials (RCTs) and quasi-randomised controlled trials (QRCTs). We will also include prospective non-randomised studies (NRSs) with a control group because it is anticipated that we will find very few RCTs. We will include primary studies that compare anterior versus posterior instrumentation and spinal correction and instrumented fusion.

#### Types of participants

People (aged 10 to 21 years) with AIS who underwent either anterior or posterior spinal correction and instrumented fusion.

#### Types of interventions

Anterior (either thoroscopically, via standard open thoracotomy or a mini-open approach combined with thoracoscopy) compared with posterior spinal correction and instrumented fusion.

#### Types of outcome measures

Clinical, functional and radiological outcome parameters. Minimal duration of follow-up of 12 months.

A clinically important effect size for outcomes is difficult to establish. However, we will check whether authors of original studies reported expected effect sizes (e.g. in sample size calculations).

#### Primary outcomes

- Pain, participant satisfaction and function: 22-item Scoliosis Research Society (SRS-22) questionnaire, 36-item Short Form (SF-36), Oswestry Disability Index (ODI), EQ-5D, EuroQol Visual Analogue Scale (EQ-VAS), Quality of Life Profile for Spinal Deformities (QLPSD).

#### Secondary outcomes

- Complications (neurological, vascular, infection, pseudarthrosis).
- Pulmonary function.
- Correction percentage on radiographs, spinal balance.

### Search methods for identification of studies

#### Electronic searches

##### Search strategy:

Searches will be undertaken to identify relevant papers on the surgical treatment of Adolescent Idiopathic Scoliosis. The search strategies (keywords) will be developed specifically for each database. Only studies conducted on humans will be sought.

The following databases will be searched from inception to current:

- Medline (OvidSP)
- Medline In-Process & Other Non-Indexed Citations (OvidSP)
- Medline Daily Update (OvidSP)
- Embase (OvidSP)

- Cochrane Central Register of Controlled Trials (CENTRAL) (Wiley) (The Cochrane Library, Latest Issue)
- [ClinicalTrials.gov](http://ClinicalTrials.gov)
- World Health Organization International Clinical Trials Registry Platform ([WHO ICTRP](http://WHO ICTRP))

The main Embase strategy was independently peer reviewed by a second Information Specialist, using the CADTH checklist ([CADTH 2013](http://CADTH 2013)), and can be found in [Appendix 1](#).

#### Searching other resources

A supplementary search will be undertaken on the following resource to identify grey literature:

- [OpenGrey](http://OpenGrey) (Internet)

#### Data collection and analysis

The search will be conducted by the team. One review author (GH) will retrieve the full-text articles to be evaluated, and contact authors for additional information if necessary.

The Cochrane team for this review will primarily communicate by email and occasionally by videoconferencing. The review authors GH, PW and LvR will meet every three months.

#### Selection of studies

Two review authors (GH and PW) will independently perform the first screening based on title and abstract and subsequently apply the selection criteria to the articles. If we cannot reach consensus we will consult a third review author (LvR). We will assess the risk of bias of included studies using methods recommended by the Cochrane Back Review Group ([Furlan 2009](http://Furlan 2009)), and The Cochrane Collaboration ([Higgins 2011](http://Higgins 2011)).

#### Data extraction and management

We will use five questions to determine if the results of the review are clinically relevant ([Furlan 2009](http://Furlan 2009); [Appendix 2](#)).

One review author (GH) will extract data and enter them into Review Manager 5 ([RevMan 2012](http://RevMan 2012)); a second review author (PW) will check entries.

We will identify studies by the main study name/identifier. Where this is not available, we will use the surname of the first study author and year of the main report/publication. To avoid the duplication of data where studies (or study populations) have multiple publications, we will use the most recent and complete report as the main reference, but we will extract additional details from the other publications as necessary.

For each study, we will record the following general types of information/data:

- study ID or name (if reported or otherwise surname of first study author);
- year of publication;
- other related publications;
- study group (if reported);
- study country(ies);
- study aim;

- treatment type;
- study funding.

Examples of specific details to be extracted include:

- sample size;
- location/setting;
- methods (e.g. randomisation and allocation concealment, blinding), extracted as part of the quality assessment process;
- participant baseline characteristics (e.g. age, diagnosis, comorbidities, previous and concomitant treatments, etc.);
- interventions/study arms compared (description of interventions and comparators);
- outcomes assessed (e.g. definition of outcome, when assessed, who assessed, methods used to assess outcome(s));
- results (e.g. numbers, percentages and effect sizes with confidence intervals (CI) (where relevant)).

### Assessment of risk of bias in included studies

We will assess the risk of bias for both RCTs and NRSs using the criteria recommended by the Cochrane Back Review Group (Furlan 2009; Higgins 2011), together with items from the Downs & Black checklist (Downs 1998). See Appendix 3 for a detailed description of the criteria for risk of bias. For each included study, we will rate each criterion as high, low or unclear and enter it into the 'Risk of bias' table in Review Manager 5 (RevMan 2012). We will pilot and test the 'assessment of risk of bias' form for intraobserver and interobserver reliability. Two review authors will independently assess the internal validity of the included studies. The review authors will resolve any disagreements by discussion; we will consult a third independent review author if disagreements persist. We will score the risk of bias criteria as high, low or unclear and will report them in the 'Risk of bias' table. We will then rate the overall extent of risk of bias within each bias category (e.g. performance bias) as 'bias' or 'no bias'. In the case that one of the review authors is also an author of trials, this review author will be excluded in the assessment of their own trial.

Relevant major confounding variables will be age, curve location, curve magnitude and the number of fused levels.

We will consider the fact that confounding due to selection bias is possible. Especially in small, NRSs large clinically important group differences may not to achieve statistical significance. We will consider this fact in interpreting results and in adjustment if possible.

When it comes to grading the quality of the evidence, we will not downgrade evidence from studies judged 'no bias' for all five categories. We will downgrade (-1 point) evidence when we judge three or fewer categories for each study to have bias. We will downgrade evidence by -2 points when four or more categories for each study are judged to have bias. See the [Data synthesis](#) section for additional details on quality assessment for each outcome.

Two review authors will independently assess study quality and any discrepancies will be resolved through discussion or the intervention of a third review author.

### Measures of treatment effect

For dichotomous outcomes that were measured with the same instrument across trials, we will preferably use risk ratios (RR) and for continuous outcomes, we will use mean differences (MD). We will use standardised mean differences (SMD) if various instruments are used for the same outcome. We will use hazard ratios (HR) for time to event data. Results will be expressed with their 95% CIs.

### Unit of analysis issues

We do not expect any unit of analysis issues in this review.

### Dealing with missing data

For recent papers (within five years), we will endeavour to collect missing data by contacting the study authors. When data are insufficient to be entered into the meta-analysis (even after contacting the study authors), we will assume that missing values have a poor outcome and we will impute that value.

### Assessment of heterogeneity

We will base the judgement of clinical homogeneity on the baseline characteristics of the trial populations (e.g. baseline risk, age, disease stage). We will assess statistical homogeneity by means of the  $I^2$  statistic. This will be assessed by measuring the degree of inconsistency between the study results and describes the percentage of total variation across studies that is due to heterogeneity rather than the play of chance. The value of the  $I^2$  statistic lies between 0% and 100%.

An approximate guide to interpretation is as follows:

- 0% to 40%: might not be important;
- 30% to 60%: may represent moderate heterogeneity\*;
- 50% to 90%: may represent substantial heterogeneity\*;
- 75% to 100%: considerable heterogeneity\*.

We will consider studies to be sufficiently similar for the purposes of pooling if the  $I^2$  statistic is less than 75%.

Because of the expected statistical heterogeneity, we will use the random-effects model.

### Assessment of reporting biases

We will assess publication bias where there are sufficient numbers of trials (i.e. six trials) using funnel plots (i.e. SE (log [RR]) versus RR; SE (MD) versus MD; and SE (log[HR]) versus HR; where SE = standard error).

### Data synthesis

Regardless of whether there are sufficient data available to use quantitative analyses to summarise the data, we will assess the overall quality of the evidence for each outcome. To accomplish this, we will use the GRADE approach, as recommended in *The Cochrane Handbook for Systematic Reviews of Interventions* (Higgins 2011), and adapted in the updated Cochrane Back Review Group method guidelines (Furlan 2009). Factors that may decrease the quality of the evidence include study design and risk of bias, inconsistency of results, indirectness (not generalisable), imprecision (sparse data) and other factors (e.g. reporting bias). We will reduce the quality of the evidence for a specific outcome by a

level, according to the performance of the studies against these five factors.

- **High-quality evidence:** there are consistent findings among at least 75% of RCTs with low risk of bias, consistent, direct and precise data and no known or suspected publication biases. Further research is unlikely to change either the estimate or our confidence in the results.
- **Moderate-quality evidence:** one of the domains is not met. Further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate.
- **Low-quality evidence:** two of the domains are not met. Further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate.
- **Very-low-quality evidence:** three of the domains are not met. We are very uncertain about the results.
- **No evidence:** no studies were identified that addressed this outcome

#### **Summary of findings table**

We will summarise the main findings of our review in one or more 'Summary of findings' tables. Our main comparison

is anterior spinal correction and fusion versus posterior spinal correction and fusion. Our main outcomes for inclusion in the table(s) are complications (neurological, vascular, infection and pseudarthrosis), pain, disability, pulmonary function, correction on radiographs and spinal balance.

#### **Subgroup analysis and investigation of heterogeneity**

Where there are at least three clinically and statistically similar trials, we will consider subgroup analyses to investigate the potential effects of the following factors: study quality (blinded versus open and concealed allocation versus no/unclear), age in years (categories to be defined).

#### **Sensitivity analysis**

If possible, we will do separate analyses according to study design (RCTs versus other studies) in the event that we will include observational studies; and according to study quality (blinded versus open and concealed allocation versus unclear).

#### **ACKNOWLEDGEMENTS**

We would like to thank Dr Teresa Marin for her help with our queries in completing this protocol.

## REFERENCES

### Additional references

#### Asher 2006

Asher MA, Burton DC. Adolescent idiopathic scoliosis: natural history and long term treatment effects. *Scoliosis* 2006;**1**(1):2.

#### Betz 1999

Betz RR, Harms J, Clements DH 3rd, Lenke LG, Lowe TG, Shufflebarger HL, et al. Comparison of anterior and posterior instrumentation for correction of adolescent thoracic idiopathic scoliosis. *Spine* 1999;**24**:225-39.

#### Burwell 2000

Burwell R, Dangerfield P. Adolescent idiopathic scoliosis: hypothesis of causation. State of the art reviews. *Spine* 2000;**14**(2):319-32.

#### Burwell 2009

Burwell RG, Aujla RK, Grevitt MP, Dangerfield PH, Moulton A, Randell TL, et al. Pathogenesis of adolescent idiopathic scoliosis in girls - double neuro-osseous theory involving disharmony between two nervous systems, somatic and autonomic expressed in the spine and trunk: possible dependency on sympathetic nervous system and hormones with implications for medical therapy. *Scoliosis* 2009;**4**:24.

#### CADTH 2013

Canadian Agency for Drugs and Technologies in Health. CADTH peer review checklist for search strategies [Internet]. <http://www.cadth.ca/en/resources/finding-evidence-is-17-07-2013:3>.

#### Delorme 2002

Delorme S, Labelle H, Aubin CE. Is Cobb angle progression a good indicator in adolescent idiopathic scoliosis?. *Spine* 2002;**27**:E145-51.

#### Downs 1998

Downs S, Black N. The feasibility of creating a checklist for the assessment of the methodological quality both of randomised and non-randomised studies of health care interventions. *Journal of Epidemiology and Community Health* 1998;**52**:377-84.

#### Furlan 2009

Furlan AD, Pennick V, Bombardier C, van Tulder M. 2009 updated method guidelines for systematic reviews in the Cochrane Back Review Group. *Spine* 2009;**34**(18):1929-41.

#### Goldberg 2002

Goldberg C, Moore D, Fogerty E, Dowling F. The natural history of early onset scoliosis. *Studies in Health Technology and Informatics* 2002;**91**:68-70.

#### Graham 2000

Graham EJ, Lenke LG, Lowe TG, Betz RR, Bridwell KH, Kong Y, et al. Prospective pulmonary function evaluation following open thoracotomy for anterior spinal fusion in adolescent idiopathic scoliosis. *Spine* 2000;**25**:2319-25.

#### Grivas 2002

Grivas TB, Samelis P, Chadziargiropoulos T, Polyzois B. Study of the ribcage deformity in children with 10 degrees-20 degrees of Cobb angle late onset idiopathic scoliosis, using rib-vertebra angles - aetiologic implications. *Studies in Health Technology and Informatics* 2002;**91**:20-4.

#### Grivas 2006a

Grivas TB, Burwell GR, Vasiliadis ES, Webb JK. A segmental radiological study of the spine and rib-cage in children with progressive infantile idiopathic scoliosis. *Scoliosis* 2006;**1**:17.

#### Grivas 2006b

Grivas TB, Vasiliadis E, Mouzakis V, Mihas C, Koufopoulos G. Association between adolescent idiopathic scoliosis prevalence and age at menarche in different geographic latitudes. *Scoliosis* 2006;**1**:9.

#### Grivas 2008a

Grivas TB, Vasiliadis ES, Rodopoulos G, Bardakos N. The role of the intervertebral disc in correction of scoliotic curves. A theoretical model of idiopathic scoliosis pathogenesis. *Studies in Health Technology and Informatics* 2008;**140**:33-6.

#### Grivas 2008b

Grivas TB, Vasiliadis ES, Rodopoulos G. Aetiology of idiopathic scoliosis. What have we learned from school screening?. *Studies in Health Technology and Informatics* 2008;**140**:240-4.

#### Higgins 2011

Higgins JPT, Green S (editors). *Cochrane Handbook for Systematic Reviews of Interventions* Version 5.1.0 [updated March 2011]. The Cochrane Collaboration, 2011. Available from [www.cochrane-handbook.org](http://www.cochrane-handbook.org).

#### Kanayama 1996

Kanayama M, Tadano S, Kaneda K, Ukai T, Abumi K. A mathematical expression of three dimensional configuration of the scoliotic spine. *ASME Journal of Biomechanical Engineering* 1996;**2**(118):247-52.

#### Lee 1997

Lee CS, Nachemson AL. The crankshaft phenomenon after posterior Harrington fusion in skeletally immature patients with thoracic or thoracolumbar idiopathic scoliosis followed to maturity. *Spine* 1997;**22**:58-67.

#### Lenke 2004

Lenke LG, Newton PO, Marks MC, Blanke KM, Sides B, Kim YJ, et al. Prospective pulmonary function comparison of open versus endoscopic anterior fusion combined with posterior fusion in adolescent idiopathic scoliosis. *Spine* 2004;**29**:2055-60.

#### Lonstein 1995

Lonstein J, Bradford D, Winter R, Ogilvie J. *Moe's Textbook of Scoliosis and other Spinal Deformities*. Vol. **1**, Philadelphia: WB Saunders, 1995.

**Lonstein 2006**

Lonstein JE. Scoliosis: surgical versus nonsurgical treatment. *Clinical Orthopaedics and Related Research* 2006;**443**:248-59.

**Lowe 2003**

Lowe TG, Betz R, Lenke L, Clements D, Harms J, Newton P, et al. Anterior single-rod instrumentation of the thoracic and lumbar spine: saving levels. *Spine* 2003;**28**(20):S208-16.

**Machida 1999**

Machida M. Cause of idiopathic scoliosis. *Spine* 1999;**24**(24):2576-83.

**Nachemson 1977**

Nachemson A, Sahlstrand T. Etiologic factors in adolescent idiopathic scoliosis. *Spine* 1977;**1**(2):176-84.

**Negrini 2006**

Negrini S, Grivas TB, Kotwicki T, Maruyama T, Rigo M, Weiss HR. Why do we treat adolescent idiopathic scoliosis? What we want to obtain and to avoid for our patients. SOSORT 2005 Consensus paper. *Scoliosis* 2006;**1**:4.

**Noordeen 1999**

Noordeen MH, Haddad FS, Edgar MA, Pringle J. Spinal growth and a histologic evaluation of the Risser grade in idiopathic scoliosis. *Spine* 1999;**24**:535-8.

**Parent 2005**

Parent S, Newton PO, Wenger DR. Adolescent idiopathic scoliosis: etiology, anatomy, natural history, and bracing. *Instructional Course Lecture* 2005;**54**:529-36.

**RevMan 2012 [Computer program]**

The Nordic Cochrane Centre, The Cochrane Collaboration. Review Manager (RevMan). Version 5.2. Copenhagen: The Nordic Cochrane Centre, The Cochrane Collaboration, 2012.

**Sanders 1997**

Sanders JO, Little DG, Richards BS. Prediction of the crankshaft phenomenon by peak height velocity. *Spine* 1997;**22**:1352-6.

**Sevastik 1997**

Sevastik B, Xiong B, Sevastik J, Lindgren U, Willers U. Rib-vertebral angle asymmetry in idiopathic, neuromuscular and experimentally induced scoliosis. *European Spine Journal* 1997;**6**:84-8.

**SOSORT 2012**

Negrini S, Aulisa A, Aulisa L, Circo A, De Mauroy J, Durmala J, et al. SOSORT guidelines: orthopaedic and rehabilitation treatment of idiopathic scoliosis during growth. *Scoliosis SOSORT Guidelines* 2012;**7**(1):3.

**Stokes 1996**

Stokes I, Spence H, Aronsson D, Kilmer N. Mechanical modulation of vertebral body growth: implications for scoliosis progression. *Spine* 1996;**21**(10):1162-7.

**Stokes 1997**

Stokes I. Analysis of symmetry of vertebral body loading consequent of lateral spinal curvature. *Spine* 1997;**22**:2495-503.

**Sweet 1999**

Sweet FA, Lenke LG, Bridwell KH, Blanke KM. Maintaining lumbar lordosis with anterior single solid-rod instrumentation in thoracolumbar and lumbar adolescent idiopathic scoliosis. *Spine* 1999;**24**:1655-62.

**Vedantam 2000**

Vedantam R, Lenke LG, Bridwell KH, Haas J, Linville DA. A prospective evaluation of pulmonary function in patients with adolescent idiopathic scoliosis relative to the surgical approach used for spinal arthrodesis. *Spine* 2000;**25**:82-90.

**Wong 1996**

Wong CA, Cole AA, Watson L, Webb JK, Johnston ID, Kinnear WJ. Pulmonary function before and after anterior spinal surgery in adult idiopathic scoliosis. *Thorax* 1996;**51**:534-6.

**Wong 2005**

Wong HK, Hui JH, Rajan U, Chia HP. Idiopathic scoliosis in Singapore school children: a prevalence study 15 years into the screening program. *Spine* 2005;**30**(10):1188-96.

## APPENDICES

### Appendix 1. Example search strategy

#### Embase (OvidSP)

AIS + Surgery + (No animals)

1. AIS.ti,ab,ot.
2. ((Adolescen\$ or juvenile\$) adj3 idiopath\$ adj3 scolios\$).ti,ab,ot,hw.
3. adolescent idiopathic scoliosis/
4. or/1-3
5. exp scoliosis/
6. (Scolios\$ or ((spine\$ or spinal) adj3 curv\$)).ti,ab,ot,hw.
7. or/5-6
8. exp Adolescence/

9. (Teen or teens or teenage\$ or teen-age\$ or juvenile\$ or Adolescen\$ or immatur\$ or youth\$ or "young adult\$" or "young people \$").ti,ab,ot.
- 10.or/8-9
- 11.7 and 10
- 12.4 or 11
- 13.(surger\$ or surgic\$ or operat\$).ti,ab,ot.
- 14.Screw\$.ti,ab,ot.
- 15.(Decompress\$ or de-compress\$ or fuse\$ or fusing or fusion\$ or correct\$).ti,ab,ot.
- 16.spinal cord decompression/
- 17.((Harrington or magnetic\$ control\$ grow\$ or Cotrel-Dubousset) adj3 (rod\$ or pin\$ or instrument\$ or equipment\$ or tool\$ or bar or bars or pole\$ or shaft\$ or device\$ or apparatus or appliance\$ or implement\$)).ti,ab,ot,hw.
- 18.Harrington instrumentation/
- 19.exp spine fusion/
- 20.thoracotomy/ or thoracoscopy/ or spondylodesis/ or (spondylodes\$ or spondylosyndes\$ or thoracotom\$ or thoracoscop\$ or pleuroscop \$ or (pleural adj3 endoscop\$)).ti,ab,ot.
- 21.(vertebra\$ adj3 condensat\$).ti,ab,ot.
- 22.((posterior\$ or anterior\$ or dorsal\$ or front or back or rear or ventral\$) adj3 instrument\$).ti,ab,ot.
- 23.or/13-22
- 24.12 and 23
- 25.adolescent idiopathic scoliosis/su
- 26.24 or 25
- 27.animal/
- 28.animal experiment/
- 29.(rat or rats or mouse or mice or murine or rodent or rodents or hamster or hamsters or pig or pigs or porcine or rabbit or rabbits or animal or animals or dogs or dog or cats or cow or bovine or sheep or ovine or monkey or monkeys).ti,ab,ot,hw.
- 30.or/27-29
- 31.exp human/
- 32.human experiment/
- 33.or/31-32
- 34.30 not (30 and 33)
- 35.26 not 34

## Appendix 2. Questions for clinical relevance

1. Are the participants described in detail so that you can decide whether they are comparable to those that you see in your practice?
2. Are the interventions and treatment settings described well enough so that you can provide the same for your patients?
3. Were all clinically relevant outcomes measured and reported?
4. Is the size of the effect clinically important?
5. Are the likely treatment benefits worth the potential harms?

## Appendix 3. Criteria for assessing risk of bias for internal validity for randomised and non-randomised studies (Downs and Black 1998; Furlan 2009)

### 1. Random sequence generation

There is a low risk of selection bias if the investigators describe a random component in the sequence generation process such as: referring to a random number table, using a computer random number generator, coin tossing, shuffling cards or envelopes, throwing dice, drawing of lots, minimisation (minimisation may be implemented without a random element, and this is considered to be equivalent to being random).

There is a high risk of selection bias if the investigators describe a non-random component in the sequence generation process, such as: sequence generated by odd or even date of birth, date (or day) of admission, hospital or clinic record number, or allocation by judgement of the clinician, preference of the participant, results of a laboratory test or a series of tests, or availability of the intervention.

If it is a non-randomised study, this will be rated as high bias.

## 2. Allocation concealment

There is a low risk of selection bias if the participants and investigators enrolling participants could not foresee assignment because one of the following, or an equivalent method, was used to conceal allocation: central allocation (including telephone, web-based and pharmacy-controlled randomisation); sequentially numbered drug containers of identical appearance or sequentially numbered, opaque, sealed envelopes.

There is a high risk of bias if participants or investigators enrolling participants could possibly foresee assignments and thus introduce selection bias, such as allocation based on: using an open random allocation schedule (e.g. a list of random numbers); assignment envelopes were used without appropriate safeguards (e.g. if envelopes were unsealed or non-opaque or not sequentially numbered); alternation or rotation; date of birth; case record number or other explicitly unconcealed procedures.

If it is a non-randomised study, this will be rated as high bias.

## 3. Selection bias (population)\*

There is low risk of selection bias if participants in different intervention groups are recruited from the same population.

## 4. Selection bias (timing)\*

There is low risk of selection bias if participants in different intervention groups are recruited over the same time. Surgical studies must be under 10 years old for low risk of selection bias.

## 5. Adjustment for confounding\*

There is low risk if no significant group differences were shown. There is high risk if the effect of the main confounders was not investigated or no adjustment was made in the final analyses.

### Performance bias

#### 6a. Blinding of participants

There is a low risk of performance bias if blinding of participants was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.

#### 6b. Blinding of personnel/care providers

There is a low risk of performance bias if blinding of personnel was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding.

## 7. Compliance (adherence)

There is low risk of bias if compliance with the interventions was acceptable, based on the reported intensity/dosage, duration, number and frequency for both the index and control intervention(s). For single-session interventions (e.g. surgery), this item is irrelevant.

## 8. Co-interventions

There is low risk of bias if there were no co-interventions or they were similar between the index and control groups.

### Attrition bias

#### 9a. Incomplete outcome data

There is a low risk of attrition bias if there were no missing outcome data; reasons for missing outcome data were unlikely to be related to the true outcome (for survival data, censoring unlikely to be introducing bias); missing outcome data were balanced in numbers, with similar reasons for missing data across groups; for dichotomous outcome data, the proportion of missing outcomes compared with the observed event risk was not enough to have a clinically relevant impact on the intervention effect estimate; for continuous outcome data, the plausible effect size (difference in means or standardised difference in means) among missing outcomes was not enough to have a clinically relevant impact on observed effect size, or missing data were imputed using appropriate methods (if drop-outs are very large, imputation using even 'acceptable' methods may still suggest a high risk of bias). The percentage of withdrawals and drop-outs should not exceed 20% for short-term follow-up and 30% for long-term follow-up and should not lead to substantial bias (these percentages are commonly used but arbitrary, not supported by literature).

#### 9b. Intention-to-treat analysis

There is low risk of bias if all randomised participants were reported/analysed in the group to which they were allocated by randomisation.

## Measurement/detection

### 10. Blinding of outcome assessment

There is low risk of detection bias if the blinding of the outcome assessment was ensured and it was unlikely that the blinding could have been broken; or if there was no blinding or incomplete blinding, but the review authors judge that the outcome is not likely to be influenced by lack of blinding, or:

- for participant-reported outcomes in which the participant was the outcome assessor (e.g. pain, disability): there is a low risk of bias for outcome assessors if there is a low risk of bias for participant blinding;
- for outcome criteria that are clinical or therapeutic events that will be determined by the interaction between participants and care providers (e.g. co-interventions, length of hospitalisation, treatment failure), in which the care provider is the outcome assessor: there is a low risk of bias for outcome assessors if there is a low risk of bias for care providers;
- for outcome criteria that are assessed from data from medical forms: there is a low risk of bias if the treatment or adverse effects of the treatment could not be noticed in the extracted data.

### 11. Timing of outcome assessments

There is low risk of bias if all important outcome assessments for all intervention groups were measured at the same time, or if analyses adjust for different lengths of follow-up.

## Selective reporting

### 12a. Data dredging

There is low risk of bias if all analyses were planned at the outset of the study.

There is high risk of bias if analyses were conducted retrospectively (e.g. retrospective unplanned subgroup analyses).

### 12b. Outcome measures

There is low risk of reporting bias if the study protocol is available and all of the study's pre-specified (primary and secondary) outcomes that are of interest in the review have been reported in the pre-specified way, or if the study protocol is not available but it is clear that the published reports include all expected outcomes, including those that were pre-specified (convincing text of this nature may be uncommon).

There is a high risk of reporting bias if not all of the study's pre-specified primary outcomes have been reported; one or more primary outcomes is reported using measurements, analysis methods or subsets of the data (e.g. subscales) that were not pre-specified; one or more reported primary outcomes were not pre-specified (unless clear justification for their reporting is provided, such as an unexpected adverse effect); one or more outcomes of interest in the review are reported incompletely so that they cannot be entered in a meta-analysis; the study report does not include results for a key outcome that would be expected to have been reported for such a study.

**\*Items 3, 4 and 5 are only relevant to non-randomised studies.**

## CONTRIBUTIONS OF AUTHORS

Gerian Huitema: protocol, search, quality assessments, data extraction, report.

Paul Willems: protocol, quality assessments, data extraction, draft review.

Jos Kleijnen: protocol, draft review.

Chris Shaffrey: protocol, draft review.

Lodewijk van Rhijn: protocol, third review author consultation, draft review.

## DECLARATIONS OF INTEREST

The review authors are not involved in an RCT concerning the topic of this review. None of the review authors receive any funding or grants for this study.

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