ELSEVIER
SAUNDERS

Neurosurg Clin N Am 16 (2005) 595–602

NEUROSURGERY
CLINICS
OF NORTH AMERICA

Biomechanics of Lumbar Arthroplasty

Neil R. Crawford, PhD

Department of Neurosurgery Research, Barrow Neurological Institute, 350 W. Thomas Road, Phoenix, AZ 85013, USA

There are many issues to consider when evaluating the biomechanics of lumbar arthroplasty, which may consist of a nucleus replacement, a total disc replacement, or a mobile posterior device. The goal of spinal arthroplasty is to replicate or augment the function of the normal spinal elements, by taking into consideration both in the quantity and quality of motion that occurs across the replaced joint. This article describes the relevant parameters for studying the biomechanics of lumbar arthroplasty and briefly summarizes the current knowledge with regard to those parameters in some well-known lumbar nucleoplasty, facet replacement, and total disc arthroplasty devices.

Biomechanical parameters

Range of motion

The simplest but most pertinent parameter for evaluating biomechanical effectiveness of a device for spinal arthroplasty is the physiologic range of motion (ROM), which is defined as the amount of motion possible across the joint at a pre-chosen nondestructive load. ROM can be evaluated in terms of a translation along any axis or a rotation about any axis. Whether angular or linear ROM is studied depends largely on the type of loading applied to the joint [1]. For example, it would be sensible to concentrate on measuring axial translational ROM during axial compression, while measuring sagittal plane rotational ROM when the patient is asked to flex and extend their back.

In vitro cadaveric studies commonly choose a load that causes normal specimens to move (on average) to an angular or linear displacement that matches clinically observed voluntary limits and define the ROM as the displacement achieved at that pre-selected load. Therefore, in a laboratory study of arthroplasty, the same pre-selected load is applied before and after arthroplasty, and the effectiveness of the arthroplasty device is evaluated by comparing the ROM after arthroplasty to the normal ROM [2,3].

Clinically, it is not as easy to evaluate ROM before and after application of an arthroplasty device. The patient does not have precise control of the load applied to their spine, and the replaced disc is abnormal to begin with, which means its ROM should not necessarily be replicated. However, since the mean normal ROM across each level of the lumbar spine is known ([4], Table 1), the effectiveness of an intervertebral disc prosthesis in replicating normal ROM in vivo can be estimated by evaluating how closely the ROMs at the replaced and adjacent levels match the corresponding mean values. It should be expected that the ROMs at least be proportionally equivalent to normal if the arthroplasty is effective. For example, if arthroplasty is applied at the L4-5 and, after surgery and recovery, the patient's flexion and extension bilateral ROMs at L3-4, L4-5, and L5-S1 are 6°, 6°, and 9° respectively, then apparently the patient has a less mobile spine than normal (adding flexion and extension columns in Table 1). Scaling the ROMs from Table 1 by 62% to match the patient's L3-4 and L5-S1 ROMs, it is expected that the ROM at L4-5 should be about 8°. Because the patient's ROM is smaller than the expected value, it would appear that the hypothetical arthroplasty allows insufficient joint mobility.

E-mail address: Neil.Crawford@chw.edu

1042-3680/05/$ - see front matter
doi:10.1016/j.nec.2005.06.002

Table 1
Mean angular range of motion ± standard deviation (in°) at all levels of the lumbar spine [4]

Lumbar level	Flexion	Extension	Lateral bending (L or R)	Axial rotation (L or R)
L1-L2	5.0 ± 1.0	4.1 ± 1.5	4.2 ± 0.6	1.8 ± 1.1
L2-L3	7.0 ± 1.2	3.3 ± 1.2	5.8 ± 1.6	2.2 ± 0.9
L3-L4	7.3 ± 1.5	2.6 ± 1.2	5.3 ± 0.8	2.2 ± 0.8
L4-L5	9.1 ± 2.5	3.6 ± 1.5	5.1 ± 1.0	1.6 ± 1.0
L5-S1	9.0 ± 2.0	5.3 ± 2.0	4.3 ± 0.8	1.1 ± 0.9

Axis of rotation

When examining the biomechanical parameters in evaluating lumbar arthroplasty, the axis of rotation (AOR) is probably a close second in importance after the ROM. The AOR is the line in space about which rotation occurs during any motion of the spine. In purely linear motions, the AOR is at infinity (infinitely anterior or posterior to the spine during compression or tension, and infinitely rostral or caudal to the spine during anterior or posterior translation). During the more common bending and twisting motions, the AOR normally lies in or near the disc space (Fig. 1).

Since the spine does not bend or twist (eg, like a hinge), the AOR does not remain at a fixed position, which makes thorough characterization of the AOR difficult. By evaluating instantaneous AORs in small steps through the bending or twisting of the spine, the path or centrode of the AOR is created [5]. Typically, the AOR is represented as a dot on a two-dimensional planar image of the spine, and it is assumed that the AOR extends vertically in and out of the page. This representation is sometimes referred to as the center of rotation.

The AOR is extremely sensitive to small inaccuracies in frames of data [6]. As technology has improved, more precise AORs have become possible, but it is still difficult to measure a three-dimensional AOR from in vivo images. Typically,

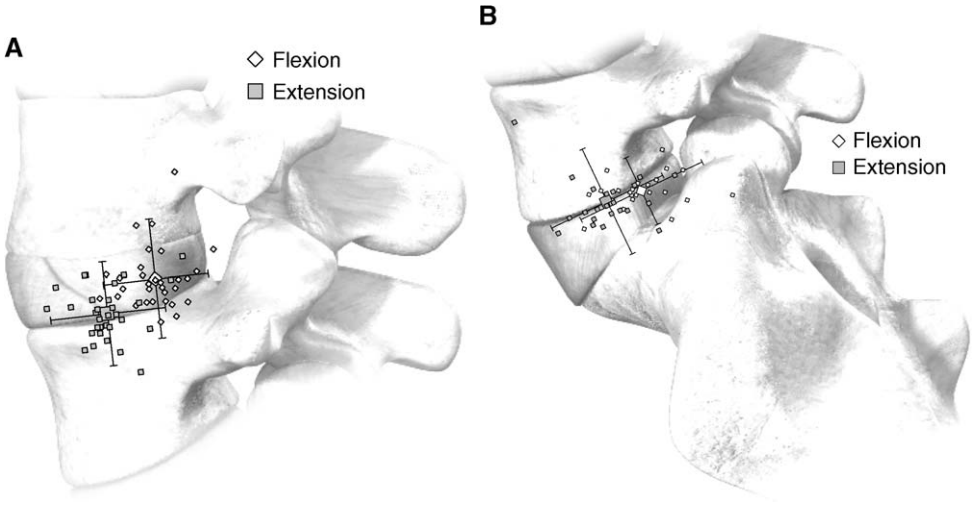

Fig. 1. Axes of rotation (AORs) at (*A*) L4-5 and (*B*) L5-S1 in the intact lower lumbar spine evaluated as a single average for neutral-to-fully-flexed and neutral-to-fully-extended motions in vitro. AORs were calculated in three dimensions from high-precision optical marker data. Data were recorded during flexibility tests where pure nonconstraining moments of 7.5 Nm were applied while holding a constant axial compressive follower load of 400 N. The plots show the intersection of the three-dimensional AOR with the mid-sagittal plane. Specimens in which the AOR was greater than 20° from perpendicular to the mid-sagittal plane were excluded. Each small data point represents the AOR for one specimen, adjusted for differences in anatomical size ($N = 31$ for L4-5 and $N = 23$ for L5-S1). Large data points represent mean values; error bars show the 95% confidence interval for this data set (± 2 standard deviation). Figure used with permission from the Barrow Neurological Institute.

AORs measured in vivo are two-dimensional approximations taken from two-dimensional planar x-ray data. In vitro, where it is possible to mount high-precision optical markers to the vertebrae, the AOR can be measured precisely in three dimensions, although limited information on lumbar AORs has yet been tabulated.

It has been shown that the location of the AOR varies depending upon the type of loading applied to the spine [7]. Therefore, although better precision in measuring the AOR can be achieved in vitro, these measurements may be less accurate than measurements made in vivo, because loads used in laboratory experiments are a simplification of true in vivo loading. This caveat may explain why the high-precision AORs generated from laboratory data—in which pure moments with a follower load were applied (see Fig. 1)—show the location of the AOR during flexion to be posterior to the location during extension; whereas, low-precision AORs generated from x-rays in living subjects [8,9], show the AOR during flexion to be anterior to that during extension.

The AOR is an important parameter because it helps to define how the quality of the motion changes with pathology or arthroplasty. If the spine moves pathologically, bony structures may collide with neural structures, causing pain. A spine in which arthroplasty has been applied may have exactly the same ROM as the pathological or normal condition, but pain may be gone because spinal motion occurs in such a way that the bony and neural elements no longer collide. That is, the AOR may have shifted from a pathological to a physiological location. Thus, restoration of normal ROM is necessary but insufficient to declare an arthroplasty device effective; restoration of both normal ROM and normal AOR are required. If pathology or arthroplasty shifts the AOR so that the facets no longer slide smoothly over each other, but instead are forced together, then eventual facet hypertrophy, or other physiological compensation such as osteophyte growth, would be expected. The exact time course for such events is unknown but is presumed to be years.

Zone of laxity

A measure of the passive motion at the replaced level provides additional insight into the biomechanics of the joint. The neutral zone (NZ) and lax zone (LZ) are measures of the joint laxity that are used with different experimental methods [10]. Both parameters are subsets of the ROM and quantify the amount of the ROM taken up by relatively unrestricted motion. The NZ quantifies the range through which the joint can move with only frictional joint resistance, and the LZ quantifies the range through which the joint can move before substantial ligamentous resistance begins to occur [10]. In the normal lumbar spine, the NZ takes up on average 13% of the ROM, and the LZ takes up on average 46% of the ROM (Table 2). Either measure is very sensitive to alterations in the loading of the joint, which makes measures of joint laxity sensitive indicators of instability under some circumstances. However, it is not possible to measure either parameter in vivo, limiting these measurements to laboratory investigations.

With regard to spinal fusion, NZ and LZ have provided valuable information on stability. When comparing two devices for spinal fusion, if one device allows a larger NZ or LZ, then it has

Table 2
Mean angular neutral zone and lax zone ± standard deviation at all levels of the lumbar spine expressed as % of total ROM (unpublished data, N = 7; 6.0 Nm maximum pure moments; no compressive follower load)

Laxity measure	Level	Flexion-extension	Lateral bending	Axial rotation
NZ	L1-L2	14 ± 12	11 ± 8	11 ± 3
	L2-L3	23 ± 34	11 ± 8	11 ± 4
	L3-L4	10 ± 9	13 ± 13	9 ± 4
	L4-L5	16 ± 17	18 ± 19	8 ± 5
	L5-S1	17 ± 13	19 ± 16	9 ± 4
LZ	L1-L2	46 ± 16	48 ± 13	29 ± 20
	L2-L3	46 ± 19	49 ± 16	33 ± 22
	L3-L4	45 ± 21	51 ± 18	38 ± 18
	L4-L5	57 ± 14	60 ± 14	44 ± 13
	L5-S1	61 ± 15	48 ± 17	31 ± 15

Abbreviations: LZ, lax zone; NZ, neutral zone.

a greater region of "slop," where the hardware is moving relative to the bone without actually tensioning. The device with the larger zone of laxity has less stabilizing potential and is less likely to provide a good environment for fusion. In arthroplasty, however, the interpretation of LZ and NZ is less straightforward. The presence of a larger LZ or NZ than normal can indicate that the arthroplasty device is not taking as large a part in resisting motion as the native tissues took. Or, a larger zone of laxity can indicate that the arthroplasty device simply has less friction than the native tissues had. Although measurements of LZ or NZ provide insight into the joint's mechanical response, it is not necessarily possible to extract information about stability of an arthroplasty device from these measures. As long as the arthroplasty and remaining native tissues can limit the total ROM, it is theoretically possible for the NZ or LZ to take up nearly 100% of the ROM after arthroplasty, without the occurrence of instability. In vivo, the muscles should easily compensate for any stability lost through enlarged zone of laxity.

Coupling

Coupling refers to the secondary motions that occur in addition to the primary, expected motion [1]. The best known pattern of coupling in the lumbar spine is the coupled lateral bending that occurs during axial rotation [11]. When transverse plane muscle forces in vivo or loading apparatus forces in vitro are applied to cause primarily axial rotation to the left, the upper lumbar spine tends to bend laterally toward the left, and the lower lumbar spine tends to bend laterally toward the right as coupled motions (Fig. 2). The inverse coupling pattern is not necessarily present; that is, when applied forces cause primarily lateral bending, little coupled axial rotation occurs simultaneously (see Fig. 2). Coupling occurs as a result of the sloped facet joints. The presence of substantial coupled lateral bending during axial rotation and the absence of substantial coupled axial rotation during lateral bending indicates that the lumbar facets interact to a greater extent during axial rotation than during lateral bending, which is intuitively apparent.

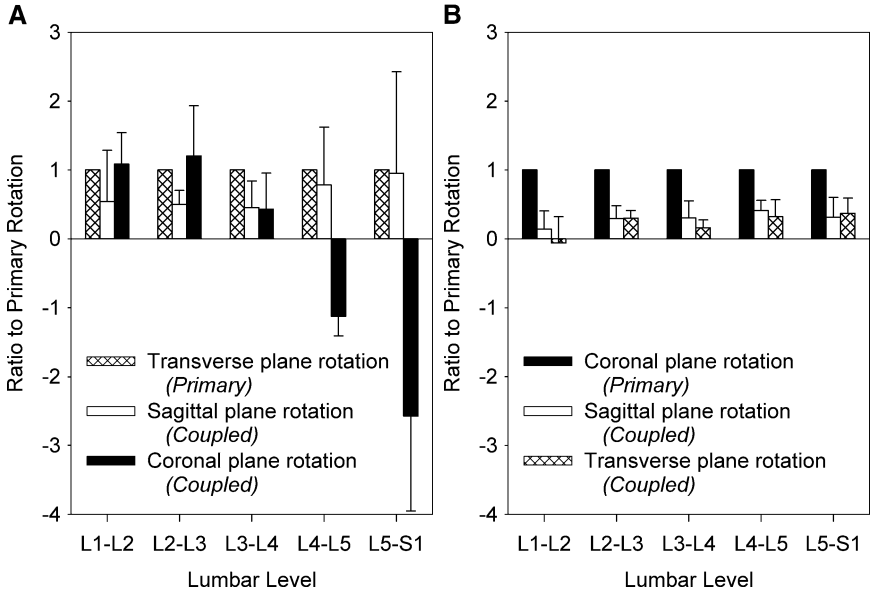

Fig. 2. Normal pattern of coupling between lateral bending and axial rotation in the lumbar spine represented as dimensionless ratios relative to the main or primary expected plane of rotation. (*A*) Coupling that occurs during loading intended to produce primarily axial rotation, (*B*) coupling that occurs during loading intended to produce primarily lateral bending. Flexion, left axial rotation, and right lateral bending are represented as positive sagittal plane rotation, positive coronal plane rotation, and positive transverse plane rotation respectively. Error bars show standard deviation. Coupling data are represented as ratios. For example, at L5-S1, for every degree of primary left axial rotation, 2.6° of coupled right lateral bending occurs. (*Data from* Panjabi M, Yamamoto I, Oxland T, et al. How does posture affect coupling in the lumbar spine? Spine 1989;14(9):1002–11.)

Coupling is an important biomechanical parameter because it is an indicator of the three-dimensional quality of spine motion. Biomechanically, lumbar arthroplasty should maintain the normal coupling pattern of the spine. If the arthroplasty disrupts the normal coupling pattern, tissues become stressed that are not used to being stressed, and the body tends to compensate with facet hypertrophy, osteophyte formation, or other means. Coupling and AOR are closely related, because both parameters describe how motion occurs. When coupling is present, the AOR is not perpendicular to the plane of the primary motion, disallowing the representation of the AOR as a dot on a two-dimensional planar image of the spine.

Adjacent segment biomechanics

One common claim of arthroplasty versus fusion is that adjacent levels are less likely to suffer from degeneration after arthroplasty than after fusion, because there is less alteration in the adjacent segment biomechanics [12]. It would, presumably, be a worthwhile goal to quantify in some way the change in biomechanics at the adjacent level after arthroplasty, which some studies have done in vitro [2,13]. Unfortunately, this approach is likely to give misleading results because of the simplified loads typically applied in the laboratory. In vivo, multiple muscles attach at multiple locations and work with precise timing to apply forces of varying and unknown magnitudes to the spine to achieve the desired motion. Conversely, a single simplified pure moment or offset force is the common loading modality used in cadaveric experiments in vitro [2,13,14]. Pure-moment loading distributes loads evenly to all levels regardless of the stability or instability of any particular level; conversely, offset-force loading distributes loads differently among levels based on the geometrical relationship to the force vector at each level [15]. Therefore, pure-moment loading would consistently under-represent the effect of arthroplasty at adjacent levels, and offset forces could unpredictably over- or under-represent the effect of arthroplasty at adjacent levels (depending on how the muscles would truly have loaded the spine in vivo). Because no in vitro study of adjacent level biomechanics has validated that the lab apparatus accurately mimics physiologic loads, current in vitro information regarding adjacent level biomechanics is unreliable. Therefore, it is best to use in vivo measurements of AOR, ROM, and coupling before and after arthroplasty to draw conclusions about how adjacent segment biomechanics are altered by lumbar arthroplasty.

Arthroplasty devices

Nucleoplasty

Nucleoplasty refers to the replacement of the nucleus pulposus with an artificial device. The best known nucleoplasty device is the PDN (Prosthetic Disc Nucleus) by Raymedica, Inc. (Bloomington, Minnesota). The natural nucleus functions biomechanically to help the disc behave both as a tension band and as a buttress. For example, during flexion, the posterior portion of the disc is under tension (tension band) and the anterior and central portions of the disc are under compression, which prevents the vertebrae from colliding (buttress). When the nucleus degenerates, the height of the disc is lost, and the tension band is less effective. The ability of the degenerated disc to act as a buttress is diminished in the same way as a deflated tire is less able to support the weight of an automobile. The biomechanical function of the nucleoplasty prosthesis is to restore the disc height, restore the buttress, and restore the ability of the remaining annulus to act as a tension band.

The PDN was evaluated biomechanically in the laboratory [16]. It was shown that after nucleotomy, the LZ increased by 65%–106% and the ROM increased by 39%–62%. Although not studied, it could be predicted that the AOR and the coupling pattern would have been altered to reflect increased interaction of the facets because of the deflated disc. After subsequent introduction of two PDN devices, the LZ and ROM values were restored to rates not significantly different from those in the normal condition. These findings reflect the theoretical prediction based on loss and restoration of the tension band and buttress as described above. Interestingly, the restored specimens had slightly greater ROM than normal in flexion, extension, and lateral bending, but slightly smaller ROM than normal in axial rotation. This may reflect a different biomechanical mechanism—friction of the device against the endplates—at work in resisting axial rotation that is minimal in the other modes.

Total disc arthroplasty

Total disc arthroplasty (TDA) refers to a device that replaces the majority of the disc, and relies on

only a small portion of the annulus for tension band stability. The intention of the TDA is to restore the motion segment to a condition mimicking the biomechanics of an intact motion segment, but, unlike in nucleoplasty, this goal is not necessarily achieved by the TDA mimicking the biomechanics of the natural disc.

Because the natural disc is a mass of woven tissues with a fluid-filled center, a design for a TDA that would seem intuitively plausible would be a thick-walled balloon or a flexible elastomeric prosthesis that is firmly attached to each endplate. Such a device was developed and underwent clinical trials (AcroFlex Lumbar Disc; DePuy Spine, Inc., Raynham, Massachusetts). However, clinical trials were aborted because of mechanical failure of the elastomer and osteolysis caused by the released particles of elastomer [17]. Biomechanically, tests with the device produced a smaller ROM than the normal spine or other TDA devices in all three planes of rotation [3,18]. For this prosthesis to work, a different elastomer is needed that is more durable and facilitates ROM that is closer to the normal spine. It is unknown whether an elastomer with the desired properties exists.

Rather than aspiring to create a TDA that matches the properties of the natural disc, an alternative approach has been taken in which the TDA changes the nature of the intervertebral disc joint from a deformable cushion to a sliding rotational joint. This approach seems to be unique to the spine—other types of arthroplasty (knee, hip, and so forth) do not typically change the intrinsic nature of the joint. There is mounting clinical evidence that the sliding-rotation joint approach is effective [19,20]. A sliding joint TDA has been shown to result in a greater ROM than a flexible core TDA [3]. The sliding joint TDA would theoretically rely more on the remaining native tissues to limit ROM than a flexible core TDA, reducing the mechanical requirements placed on the TDA. It may be that it is necessary to minimize the mechanical requirements of the TDA in this way to make the TDA adequately durable.

Some sliding joint TDA devices have a mobile, sliding core (eg, SB Charité III; DePuy Spine, Inc.), and others have semi-constrained ball-and-socket type joints (eg, Prodisc II, Synthes Spine, Paoli, Pennsylvania). The mobile core of the Charité device makes it possible for the AOR to shift anteriorly during extension and posteriorly during flexion [21]. This pattern is exactly what is seen in the normal spine in vitro (but not necessarily in vivo), both at the L4-5 and the L5-S1 levels (see Fig. 1). Conversely, the fixed AOR of the Prodisc becomes the fixed AOR of the motion segment, which is most likely not exactly the natural position of the motion segment's AOR. The better ability of the Charité TDA to allow the joint to find the natural location of the AOR has been described as the biggest biomechanical advantage of the Charité over the Prodisc [22]. Similarly, the mobile core would seem to facilitate normal patterns of coupling. This hypothesis has not been tested. The advantage of the Prodisc over the Charité appears to be its better ability to limit the ROM, particularly the linear anteroposterior translational ROM in response to anterior and posterior shear loads. Limitation of ROM is an important factor if stability is in question, such as in multi-level disc replacement. Although linear ROM has not been directly compared between the two TDAs, one laboratory study showed the Prodisc to allow smaller angular ROMs than Charité, especially during lateral bending [23]. Of particular concern in both the Charité and Prodisc TDAs is the unconstrained nature of axial rotation. In this mode, the TDA relies heavily on the remaining annulus and posterior elements to resist motion, and provides only a guiding effect to ensure that the facets interact squarely (more so with the ball-and-socket Prodisc than the mobile core Charité). However, such a function is probably effective since it is known that the facets are excellent resistors to rotation that provide the final "stop" to axial rotation [24].

Mobile posterior devices

Mobile posterior arthroplasty devices are intended to replace the facet joints or augment the function of the posterior elements. An indication for a mobile posterior device is painful excessive pathological motion. Therefore, the biomechanical function of a mobile posterior device would be twofold. First, it would need to limit ROM, and second, it would need to establish a kinematic pattern (AOR) that avoids pathological interaction between neural and bony structures.

Some devices achieve these goals with a flexible or semiconstrained implant (eg, Dynesys, Zimmer Spine, Minneapolis, Minnesota; X STOP Interspinous Process Decompression, St. Francis Medical Technologies, Alameda, California). Such devices wedge or prop open the posterior intervertebral space to increase the foraminal area and unload the facets, and they also limit

extension ROM by a buttress or cantilever effect without necessarily affecting lateral bending or axial rotation ROM [13,25]. In doing so, the AOR shifts toward the device, which acts as a new fulcrum for bending and twisting. Clinical outcomes of these devices are still being evaluated; there have been reports of good clinical performance [26,27] and suboptimal clinical performance [28]. Biomechanically, it might be suspected that suboptimal clinical performance could be caused by the device forcing an unnatural AOR, which might alleviate pain at the foramina but might load other tissues pathologically and create new pathology such as kyphosis. The amount by which these devices shift the AOR away from normal has not been quantified.

A seemingly more elegant and more recent posterior mobile stabilization strategy is facet arthroplasty (eg, TFAS, Archus Orthopedics, Inc., Redmond, Washington), which would focus specifically on restoring the function of normal facet joints. Theoretically, a well-designed facet arthroplasty could limit extension ROM and restore foraminal space, and at the same time guide the AOR to a normal location. A recent in vitro study of one facet arthroplasty device, showed successful restoration of normal ROM and AOR at L4-5 after injury and device implantation [29]. As with other forms of disc arthroplasty, current biomechanical knowledge is limited and further biomechanical comparisons in vitro and in vivo are needed.

Summary

- Biomechanical factors to consider when evaluating lumbar arthroplasty include: range of motion, zone of laxity, axis of rotation, and coupling. These factors are relevant both at the level of arthroplasty and at motion segments adjacent to arthroplasty.
- The range of motion should remain the same before and after arthroplasty. This condition is necessary but not sufficient for the success of the arthroplasty.
- The axis of rotation should be maintained at the same position before and after arthroplasty. The axis of rotation is more precisely measured in vitro and more accurately measured in vivo.
- The strong coupled lateral bending that occurs during axial rotation should be maintained after arthroplasty to ensure normal loading of tissues.
- Although it is desirable to maintain the same zone of laxity after arthroplasty as in the normal spine, this biomechanical parameter is probably minimally important when evaluating the success of the arthroplasty.

References

[1] Goel VK, Winterbottom JM, Weinstein JN, et al. Load sharing among spinal elements of a motion segment in extension and lateral bending. J Biomech Eng 1987;109:291–7.
[2] Cunningham BW, Gordon JD, Dmitriev AE, et al. Biomechanical evaluation of total disc replacement arthroplasty: an *in vitro* human cadaveric model. Spine 2003;28(20S):S110–7.
[3] Cunningham BW, Dmitriev AE, Hu N, et al. General principles of total disc replacement arthroplasty: seventeen cases in a nonhuman primate model. Spine 2003;28(20S):S118–24.
[4] Panjabi MM, Oxland TR, Yamamoto I, et al. Mechanical behavior of the human lumbar and lumbosacral spine as shown by three-dimensional load-displacement curves. J Bone Joint Surg (Am) 1994; 76A:413–24.
[5] Gertzbein SD, Seligman J, Holtby R, et al. Centrode patterns and segmental instability in degenerative disc disease. Spine 1985;10(3):257–61.
[6] Panjabi MM, Goel VK, Walter SD. Errors in kinematic parameters of a planar joint: guidelines for optimal experimental design. J Biomech 1982;15(7): 537–44.
[7] Panjabi MM, Krag MH, Dimnet JC, et al. Thoracic spine centers of rotation in the sagittal plane. J Orthop Res 1984;1(4):387–94.
[8] Pearcy MJ, Bogduk N. Instantaneous axes of rotation of the lumber intervertebral joints. Spine 1988; 13(9):1033–41.
[9] Yoshioka T, Tsuji H, Hirano N, et al. Motion characteristic of the normal lumbar spine in young adults: Instantaneous axis of rotation and vertebral center motion analyses. J Spinal Disord 1990;3(2): 103–13.
[10] Crawford NR, Peles JD, Dickman CA. The spinal lax zone and neutral zone: measurement techniques and parameter comparisons. J Spinal Disord 1998; 11(5):416–29.
[11] Panjabi M, Yamamoto I, Oxland T, et al. How does posture affect coupling in the lumbar spine? Spine 1989;14(9):1002–11.
[12] Hilibrand AS, Robbins M. Adjacent segment degeneration and adjacent segment disease: the consequences of spinal fusion? Spine J 2004;4(6 Suppl): 190S–4S.
[13] Lindsey DP, Swanson KE, Fuchs P, et al. The effects of an interspinous implant on the kinematics of the instrumented and adjacent levels in the lumbar spine. Spine 2003;28(19):2192–7.

[14] Chen WJ, Lai PL, Tai CL, et al. The effect of sagittal alignment on adjacent joint mobility after lumbar instrumentation–a biomechanical study of lumbar vertebrae in a porcine model. Clin Biomech 2004; 19(8):763–8.
[15] Panjabi MM. Biomechanical evaluation of spinal fixation devices: 1: a conceptual framework. Spine 1988;13(10):1129–34.
[16] Eysel P, Rompe J, Schoenmayr R, et al. Biomechanical behaviour of a prosthetic lumbar nucleus. Acta Neurochir (Wien) 1999;141(10):1083–7.
[17] Fraser RD, Ross ER, Lowery GL, et al. AcroFlex design and results. Spine J 2004;4(6 Suppl): 245S–51S.
[18] Cunningham BW, Lowery GL, Serhan HA, et al. Total disc replacement arthroplasty using the AcroFlex lumbar disc: a non-human primate model. Eur Spine J 2002;11(Suppl 2):S115–23.
[19] Guyer RD, McAfee PC, Hochschuler SH, et al. Prospective randomized study of the Charite artificial disc: data from two investigational centers. Spine J 2004;4(6 Suppl):252S–9S.
[20] Zigler JE. Lumbar spine arthroplasty using the ProDisc II. Spine J 2004;4(6 Suppl):260S–7S.
[21] Link HD. History, design and biomechanics of the LINK SB Charite artificial disc. Eur Spine J 2002; 11(Suppl 2):S98–105.
[22] Huang RC, Girardi FP, Cammisa FP Jr, et al. The implications of constraint in lumbar total disc replacement. J Spinal Disord Tech 2003;16(4):412–7.
[23] DiAngelo D, Foley KT, Morrow B, et al. Biomechanical comparison of the Charité and Prodisc lumbar disc prostheses. Poster presentation at the Global Symposium on Motion Preservation Technology, 5th Annual Meeting of the Spinal Arthroplasty Society. New York, May 4–7, 2005.
[24] Ahmed AM, Duncan NA, Burke DL. The effect of facet geometry on the axial torque-rotation response of lumbar motion segments. Spine 1990;15(5): 391–401.
[25] Schmoelz W, Huber JF, Nydegger T, et al. Dynamic stabilization of the lumbar spine and its effects on adjacent segments: an *in vitro* experiment. J Spinal Disord Tech 2003;16(4):418–23.
[26] Lee J, Hida K, Seki T, et al. An interspinous process distractor (X STOP) for lumbar spinal stenosis in elderly patients: preliminary experiences in 10 consecutive cases. J Spinal Disord Tech 2004; 17(1):72–7.
[27] Stoll TM, Dubois G, Schwarzenbach O. The dynamic neutralization system for the spine: a multicenter study of a novel non-fusion system. Eur Spine J 2002;11(Suppl 2):S170–8.
[28] Grob D, Benini A, Junge A, et al. Clinical experience with the Dynesys semirigid fixation system for the lumbar spine: surgical and patient-oriented outcome in 50 cases after an average of 2 years. Spine 2005; 30(3):324–31.
[29] Oxland TR, Zhu Q, Larson CR, et al. Biomechanical evaluation of the total facet arthroplasty system (TFAS) kinematics. Poster presentation at the Global Symposium on Motion Preservation Technology, 5th Annual Meeting of the Spinal Arthroplasty Society. New York, May 4–7, 2005.