



Review Article

ISSN : 2277-3657
CODEN(USA) : IJPRPM

The Role of Total Disc Replacement in Degenerative Disc Degeneration, Literature Review

Wafaa Sulaiman Alhifzi¹, Fahad Mohammed Alqahtani^{1*}, Wael Abdulrahman Al
Luhayb¹, Abdulaziz Musallam S Aljohani², Mohanad Misfer S Alkathami³, Jamal Abdulghani
Taj⁴, Shahd Mohammed Alanzan⁵, Marwan Salah Alsaadi⁶, Abdullah Ahmed Amin⁶, Safar
Dhawi Saleh Alyami⁷, Abdullah Matuq Alaithan⁸

¹Faculty of Medicine, King Khalid University, Abha, KSA.

²Primary Care of Prince Mohammad bin Abdulaziz International Airport, Madinah, KSA.

³Faculty of Medicine, Bisha University, Bisha, KSA.

⁴Faculty of Medicine, King Saud bin Abdulaziz University for Health Sciences, Jeddah, KSA.

⁵Faculty of Medicine, King Saud University, Riyadh, KSA.

⁶Faculty of Medicine, Ibn Sina National College of Medicine, Jeddah, KSA.

⁷Faculty of Medicine, Jordan University of Science and Technology, Irbid, KSA.

⁸Faculty of Medicine, King Faisal University, Al Ahsa, KSA.

*Email: dr_Fh2a3d_mujary@hotmail.com

ABSTRACT

Degenerative disc degeneration (DDD) alludes to the progressive collapse of the disc to execute its function, resulting in a diminished range of motion and back inconvenience. Even though it is debilitating, the majority of people see their symptoms slowly go away without the requisite for surgery. However, numerous cases require surgical intercession, and spinal integration has been considered the gold standard for DDD in the past years. Nevertheless, spinal integration has shown to be related to a few complications. Subsequently, total disc restoration (TDR) surgery recently has been replacing spinal integration within the therapy of DDD. To review the role of TDR in DDD cases and provide a competent survey that covers the main aspects of this topic. PubMed database was utilized for articles selection, and the following keys were utilized within the mesh ((“degenerative disc degeneration”[Mesh]) AND (“surgical management”[Mesh]) OR (“total disc replacement”[Mesh])). TDR works by mimicking the biomechanical function of an unscathed intervertebral disc, conserving physiological versatility and perhaps lowering the risk of adjacent segment degeneration as compared to lumbar fusion.

Key words: Degenerative disc degeneration, Total disc restoration, Surgery, Management

INTRODUCTION

DDD alludes to the progressive decline in the performance of the disc, which leads to a diminished range of movements and back distress [1, 2]. Aging, mechanical stress, and a few hereditary variables have all been connected to DDD. The disc is a structure that contains vessels, making it defenseless against injury and incapable to regenerate viably. This helps to clarify why DDD is predominant [3]. The disc can function as a shock absorber between the vertebrae in terms of physiology. It moreover aids within the support of spinal arrangement and the facilitation of a range of movement. The failure of arrangement, the collapse of the intervertebral disc produce

pressure over the facet joint, and degeneration, impingement on neural structures, and pressure on the paraspinal muscles [4].

DDD may develop anywhere along the spine, although it is more predominant within the cervical and lumbar regions, resulting in neck and back distress. Even though it is debilitating, the majority of individuals see their symptoms slowly go away without the require for surgery [1, 5]. However, numerous cases require surgical intercession, and spinal fusion has been considered the gold standard for DDD in the past years. Nevertheless, spinal fusion has appeared to be related to a few complications. Hence, TDR surgery recently has been supplanting spinal fusion in the treatment of DDD. In this article, we aim to survey the role of TDR in DDD cases and provide a sufficient review that covers most aspects of this topic.

MATERIALS AND METHODS

PubMed database was utilized for articles draft, and the following keys were utilized in the mesh (“degenerative disc degeneration”[Mesh]) AND (“surgical management”[Mesh]) OR (“total disc replacement”[Mesh]).

In respect to the incorporation criteria, the articles were chosen based on the incorporation of one of the following points: degenerative disc degeneration, and total disc substitution. Exclusion criteria were all other articles, which did not have one of these points as their essential endpoint.

RESULTS AND DISCUSSION

Epidemiology

The predominance of Degenerative Disc Disease is somehow related to age, with forty percent of those aged forty years having Degenerative Disc Disease and eighty percent of those matured 80 years or beyond having DDD. Lumbar DDD affects individuals as early as thirty years old, and the highest proportion can occur around the age of 40. Cervical DDD affects essentially the middle-aged and elderly, which is one of the major causes of nerve root dysfunction and cervical spinal cord in individuals aged 55 and over. While the majority of individuals over 50 years of age experiences DDD related to their spine, only around a third of them are present symptoms. Lumbar DDD is characterized by low back pain, while cervical DDD is characterized by neck and arm pain. Low back pain is anticipated to have a point predominance of between 4% and 33%, with a 1-year predominance rate of 73% and a lifetime prevalence rate of between 58% and 84%. The prevalence of low back pain, on the other hand, changes depending on the criteria applied and the populace surveyed. Despite the significant lifetime frequency of low back pain, it is predicted that 80% to 90% of all lower back pain can be treated non-surgically. However, nonsurgical therapy alleviates pain in only roughly 30% to 50% of individuals with cervical myelopathy and 75% of patients with cervical radiculopathy [3, 6, 7].

Pathophysiology and risk factors

Nutrition, oxygen, and chemo-modulators must all be delivered through the numerous small blood arteries that inundate the nerve roots and spinal cord. These structures' compression reduces their capacity to transport essential nutrients, ensuing in an ischemia impact on the structure. Ischemic pain occurs onward the course of this nerve root as a result of the inflammation caused by intervertebral disc compression. Cytokines increment is seen when the disc compresses the nerve. Macrophages and Tumor necrosis factor-alpha and enlistment. Remarkably, a positive body mass index can be affiliated with several histologic changes inside the intervertebral disc appeared, which further suggest obese individuals are more likely to endure degeneration.

Within the middle of the intervertebral disc is where the nucleus pulposus is located, the location of the annulus fibrosus on the periphery, cranially and caudally at the intersection of the vertebral bodies are the locations for cartilaginous interplaits. The existence of proteoglycans within the nucleus pulposus enables water retention. This characteristic of the nucleus pulposus is critical for the intervertebral disc's axial load management. Type II collagen is the most periodic type of collagen found within the nucleus pulposus of a healthy plate. The annulus fibrosus is made up mainly of type I collagen and encompasses the nucleus pulposus [7].

A few decades back, explanations of the innervation of the intervertebral plate were displayed. The discogenic back pain for the neurologic foundation is theorized to incorporate divisions of the spinal nerves, sinus-vertebra nerve, and gray rami communicantes. There has been a report of an increment in fibers of nerve and blood vessels within the excruciating disc, reaching regions of the nerve free pulposus, annulus fibrosus, and nucleus within the well-functioning plate, and an interface between these perception and neurotrophin expression levels has been proposed [8, 9]. In numerous regards, the method of degeneration is comparable to the method of aging. Disc

degeneration, on the other hand, is established at a higher pace in working-age individuals, making DDD a common paradox. A process is known as chondrosis intervertebralis which occurs when the water content of the intervertebral disc diminishes as individuals get older, and crevices within the nucleus pulposus, potentially extending into the annulus fibrosus might appear. This process can further flag the onset of deterioration of the intervertebral disc, endplates, and vertebral bodies [10]. DDD is a complicated degenerative process caused by changes in the disc's molecular composition as it ages. This cascade has biomechanical and, in certain cases, clinical consequences that might cause significant disability in the person affected. Environmental factors are an auxiliary concern to the hereditary component of DDD. Nonetheless, the impact of environmental factors on DDD is anything but inconsequential. A little interface between smoking and disc degeneration has been found, indicating that chemical exposures may play a role [7, 11]. Moreover, a connection of atherosclerotic injuries in the aorta with low back pain has been portrayed, indicating a potential relationship between atherosclerosis and DDD [12].

Clinical feature

The timing of pain, pain radiation, and triggering events should all be addressed in the patient's history. Patients commonly report discomfort spreading down their glutes and lower terminus. It is important to pay close attention to previous trauma incidents. The important scenario is to figure out whether the discomfort is limited to the lower back or has spread to the legs. The presence of radiating pain is a sign of canal stenosis. When the major problem is radiating pain, the surgical outcome is considerably more predictable than when the problem is non-specific lower back pain caused by muscle exhaustion and tension.

Back discomfort with a mechanical component, such as pain that only occurs with specific motions, might suggest uncertainty or a deteriorating pars fissure. Inspecting circulation is particularly crucial in any examination of extremity problems since intermittent claudication can mirror or imitate neurogenic abnormalities.

An appraisal of the patient's walking ability is fundamental for determining the daily impact of the pain/deficit. One systematic sequence is to have the patient rise from the chair, walk on his or her heels and toes, and then sit on the examination table for strength, reflex, and straight leg raise (SLR) tests. A Trendelenburg stride, which can show an underlying weakness in the gluteus medius, which is innervated by the L5 nerve root, should be ruled out during the stride evaluation.

The neurologic function of the arms, legs, bladder, and intestines will be evaluated throughout all physical examinations. Organization and patience are the keys to a comprehensive examination. Not just strength but also sensitivity and reflexes should be surveyed. It is also vital to examine the skin around the back and note any pain from compression or previous surgical scars.

A supine patient's fully extended leg is passively stretched from 0 to roughly 80 degrees in the SLR test. A diagnosis of a stenotic canal is supported by the start of radiating back discomfort in either leg. Ankle dorsiflexion weakness and large toe extension are symptoms of a herniation compressing the L5 nerve root. The Achilles tendon reflex may also be impaired as a result of this deficiency. Quadriceps weakness and a reduced patellar tendon response are common symptoms of L4 radiculopathy.

Non-organic causes of low back pain/symptoms should also be ruled out during a thorough examination. When a physician detects psychological reasons, the following should be taken into account: Non-dermatomal patterns of distribution of symptoms, pain with axial load/rotational movements, negative SLR with patient diversion (one approach incorporate having the patient sit in a chair and replicating the SLR's environment), and pain out of proportion on the exam are all illustrations of non-specific symptoms or irregularity. These are called Waddell's signs [11, 12].

Evaluation

In patients without red flags, magnetic reverberation imaging (MRI) should not be done at the initial presentation of suspected acute disc herniations, since these patients will likely gain from a 6-week course of physical therapy. Anterior-posterior and lateral radiographs of the influenced region are commonly utilized within the assessment of individuals with low back pain. Radiographs of the entire spine might be requested by a few physicians. Within the first presentation, an MRI is most certainly an unnecessary cost and utilization burden. If the symptomatology endures at the follow-up, an MRI can be procured amid that time. The T2 weighted sagittal and axial scans ought to be the priority since they will show any compression of neurologic components. Both symptomatic and asymptomatic disc herniations will shrivel in size on MRI over time. The presence of disc malady-like

degeneration or herniation on an MRI scan does not necessarily affect the probability of diligent pain or the need for surgery in the future [13, 14].

Management

Nonsurgical and surgical therapies are available to alleviate pain and reduce the disability caused by DDD. Physical treatment, facet joint infusions, epidural steroids, needle therapy, behavior alteration, ultrasound, anti-inflammatory drugs, pain relievers, muscle relaxants, lumbosacral stabilization treatment, and orthotic management are a few of the nonsurgical therapies accessible. Periradicular treatment (injection of local anesthetic and/or glucocorticoids), percutaneous laser discectomy, and intradiscal electro-thermal therapy are all minimally invasive techniques for pain relief. Nonsurgical therapies are expected to be ineffectual in 10% to 20% of individuals with lumbar DDD and up to 30% of those with cervical DDD [15, 16].

Surgical therapy is explored in certain instances. The technique of fusing or connecting two bones is known as spinal fusion or arthrodesis, and it is regarded as the surgical gold standard for DDD. The reason for spinal fusion is to reduce any strain on the spinal nerves and to reestablish the spine's arrangement and steadiness [17].

Discectomy or the removal of all or portion of the degenerative intervertebral disc that is considered to be the source of pain is required for fusion. The surgeon can either take off the intervertebral disc-less space vacant or fill it with a bone transplant after the plate is removed. The bone transplant fills the gap, encouraging fusion and giving stability [18]. In 70% to 80% of instances, spontaneous fusion is expected without the need for a bone transplant [17].

Over the last 20 years, new surgical techniques, equipment, and bone graft origin for spinal fusion have been presented. The most common source of bone grafts is the patient's own hip bone (autograft), although they can also be obtained from a donor (allograft). Bone morphogenic proteins, a synthetic bone grafting substance, may also be employed. The surgical techniques of spinal fusions include posterolateral and posterior fusion; anterior or posterior interbody fusion; and the circumferential technique, which combines anterior interbody fusion and posterior or posterolateral fusion [19].

The insertion of hardware such as screws or plates to give stability to the fused spinal segment and reduce the risk of pseudo fusion is known as instrumental or inadequate fusion [20]. The lack of mobility in the spine, which is regarded to cause adjoining segment degeneration, is one of the disadvantages of surgical fusion. The degeneration of the vertebrae above or below the fusion site is known as adjacent segment degeneration. Greater mobility of neighboring vertebrae, as well as increased stress on adjacent intervertebral discs as a result of motion transfer from fused vertebrae, are considered to have a role in the development of adjacent segment degeneration. However, it is uncertain whether adjacent segment degeneration is caused primarily by the spinal fusion process or by normal vertebral deterioration. Adjacent segment degeneration following spinal fusion is frequent, according to radiographic data, and is expected to develop at rates ranging from 8% over 4 years to 100% over 6 years. After spinal fusion, the prevalence of symptomatic adjacent segment degeneration is decreased, ranging from 5.2% in 13 years to 18.5% in 5 years [21]. This emergence of adjacent segment degeneration is problematic because it may necessitate more surgery if it produces symptoms such as aches and impairment. Therefore, the surgical treatment of persistent low back pain caused by DDD is still debatable.

Regarding the efficacy of the fusion approach on the quality of life correlated with other types of surgical and nonsurgical treatments, fusion has shown no significant superiority. Pseudo-arthritis (15%), distress and infection at the bone graft donor site (hip bone) (11%), instrument failure (7%), and neurological injuries (3%) are all common consequences of spinal fusion surgery [22, 23]. Anterior approach spinal fusion surgeries have a higher risk of gastrointestinal and vascular problems, whereas posterior approach spinal fusion procedures have a higher rate of dura- and neurology-related complications. After anterior lumbar fusion surgery, vascular damage has been found to occur at a rate of 18%. Dural tears have been observed to ensue at a rate ranging from 0.3% to 13% after spinal surgery [20].

Total disc replacement (TDR)

Artificial intervertebral disc restoration has become a viable alternative to fusion surgery. This therapy includes replacing the damaged disc with an artificial disc that can maintain normal segmental mobility and hence minimize the likelihood of neighboring segment degeneration [24, 25].

TDR has been shown safe and successful especially in the treatment of lumbar discogenic low back pain caused by DDD during the last decade. TDR works by imitating the biomechanical function of an intact intervertebral disc, preserving physiological mobility and perhaps decreasing the risk of adjacent segment degeneration as

compared to lumbar fusion [26, 27]. An indirect comparison by Zigler et Delamarter between TDR and lumbar fusion patients found that TDR patients had a much lower rate of progression in radiographic adjacent-level degeneration over 5 years than lumbar fusion patients [28, 29]. Furthermore, lumbar TDR improves neighboring segment pathology and index-level range of motion much more than fusion [26].

Selection criteria

It is critically important, like with any operation, to establish adequate patient selection criteria [30]. The distinctions between fusion and arthroplasty must be underlined to comprehend the unique indications for lumbar TDR. Initially, any lumbar pain producer, such as the disc, facet joints, and adjacent tissues, is meant to be fused to minimize mobility and hence the discomfort. Because arthroplasty retains motion while addressing just the disc space, it can only treat disc-related pain and not pain from other causes such as facet joint pain [31].

Facet anatomy is evaluated before surgery when planning for TDR. Patients with an extreme pelvic predominance (more than 65°) are more likely to develop an arthritic deficiency in the facet joints. These individuals are not excellent candidates for TDR because their mobility may be restricted following surgery due to hypertrophy facet joints. Second, TDR isn't outlined to preserve the spinal column and ought not to be utilized in individuals with spondylolisthesis or other translational abnormalities. Third, in patients with diminished mobility owing to segmental auto fusion like ankylosing spondylitis, a motion preserving or restoring TDR treatment is not suitable. Finally, solid attachment to the bone is required for disc arthroplasty. Fascination failure and vertebral body fissure may occur in patients with the low bone trait [31, 32].

Therefore, lumbar TDR may be considered a feasible cure alternative in patients with painful DDD who have failed to respond to more than 6 months of nonsurgical approach and who have diagnostic studies confirming the disc as the likely source of pain, but who do not have substantial facet joint degeneration, defects, anomalies, or osteoporosis [30, 33].

Surgical procedure

The patient is positioned in da Vinci position which is a supine posture with the legs spread, allowing the surgeon to operate immediately anterior to the disc area by standing between the legs. When pathing L4–L5 or above, an oximeter on the left hallux ought to be utilized to check for ischemia caused by retractors shortening the left iliac artery for a brief time. To guarantee visualization of the operative level as well as neutral alignment of the lumbar spine, anterior-posterior and lateral pictures are taken [34, 35].

The anterior route is the most prevalent method of lumbar implant implantation. Except for L5–S1, a left retroperitoneal approach is suggested at all levels. The anatomic placement of the major vessels on the left side is the reason for the preference. The aorta (on the left) is not only simple to recognize, but it also has a stronger wall than the inferior vena cava (on the right). However, to prevent damage to the superior hypogastric plexus (placed in the left anterior portion of the promontory) and subsequent retrograde ejaculation, right-sided access is recommended to reach L5–S1. Another advantage of reaching L5–S1 from the right side is that if the patient needs a second anterior approach to a more cephalad lumbar disc in the future, a virgin left-sided retroperitoneal plane is retained [35, 36].

Vessels are the most challenging part of getting to the disc, with L4–5 being the most difficult. It is necessary to have access to general and vascular surgeons in the event of visceral or vascular damage, especially during revision procedures. The ascending lumbar vein, which should be ligated if required, should always be given special attention. The disc is removed, as well as the cartilaginous endplates. To avoid subsidence, special attention should be paid to the integrity of the bone endplates upon which the prosthesis will rest [30, 35, 37, 38]. After the annulus fibrosus has been revealed, the center of the vertebral disc space is located and marked on the cranial and caudal vertebrae using fluoroscopic imaging. This allows for more precise implant placement after the disc area has been prepared. The annulus fibrosus is dissected anteriorly, and a partial discectomy is done, leaving the annulus intact on the lateral and posterior sides. When dissecting near to the osseous endplates, more caution is required to preserve the endplates' integrity, which is critical for long-term implant success. Depending on the implant, the trial, insertion, and placement differ. The rectus sheath and linea-alba are sutured, and the skin is then closed [1]. The irrigated surgical site and hemostasis are maintained utilizing hemostatic medications or cautery once implantation is finished and sufficient is confirmed fluoroscopically.

Possible obstacles facing TDR

The ability to restore sagittal equilibrium is one of the aims of TDR surgery. According to current research, raising lordosis to achieve physiologic sagittal alignment is linked to better clinical results [1, 39]. Whereas this is also genuine for TDR, there is proof that arthroplasty patients are an advantage from avoiding significant lordosis. Enormous lordosis, which causes the embed endplate to impinge on each other amid expansion, might confine the range of movement of the disc embed postoperatively. Furthermore, it might place greater stress on the implant, resulting in deterioration and ultimate implant failure [40].

Because the patient is in a supine posture, assessing segmental lordosis intraoperatively might be difficult. A parameter was suggested in a study, which includes measuring segmental angle intraoperatively while inside the index disc space, the spacer is being inserted. This measurement has been found to predict postoperative segmental lordosis accurately in the standing position [41]. Implementing facet replacement in combination with TDR is another innovative surgical approach. Degenerative disc disease can be linked to Facet arthrosis and the two diseases frequently coexist. Facet arthrosis, on the other hand, is a contraindication to TDR. As a result, fusion surgery is the sole choice for a substantial percentage of individuals. Therefore, Nayak *et al.* conducted biomechanical research to look at unilateral facet joint restoration and lateral TDR. When compared to an undamaged spine, the research found no significant differences in spinal motion characteristics [42].

The most significant factor impacting implant success is wear. The most prevalent cause of disc replacement failure is aseptic loosening caused by implant wear. Debris accumulates in the region surrounding the prosthesis due to implant wear. This fragment triggers an inflammatory response that looks like a foreign body reaction, complete with granulomas and bone resorption. In addition, the inflammatory environment activates nearby sensory fibers, amplifying the recurrence of painful feelings.

Several researchers have looked into ways to decrease this immunologic response, and immunomodulation has been suggested as a potential therapeutic target for aseptic implant loosening. Several inflammatory indicators have been discovered in the inflammatory response pathway that leads to osteolysis [43]. Etanercept, a TNF inhibitor, has been proposed as a potential treatment for limiting osteolysis, although its usefulness has yet to be shown clinically.

More modern implants are subjected to an extra stage of gamma radiation, which increases the implant's resistance to oxidative damage. The quantity of worn debris should be reduced as a result of this [44].

The facet and sacroiliac joints are additional sources of pain. Due to the progression of facet degeneration, patients with underlying low-grade facet arthrosis are more prone to have early post-operative discomfort. Excessive stress on the facet owing to the loss of supporting ligaments is the most common cause of facet joint degeneration. Facet joint arthrosis was also observed to be more common in arthroplasty performed at the L5-S1 level than at other levels [45].

A broad analysis of the American College of Surgeons National Surgical Quality Improvement Program database for all spinal operations (36,440 patients) showed that arthroplasty had the lowest infection rate which was 0%, while posterolateral fusion had a 1.04% infection rate [46]. However, Implant loosening can be caused by a surgical site infection, which is an uncommon but still probable cause.

Another possible side effect of TDR surgery is heterotopic ossification. Heterotopic ossification may restrict spinal mobility and trigger adjacent segment degeneration, as well as induce radiculopathy symptoms. At 5 years, the incidence of heterotopic ossification following TDR varies from 20% to 50%, and at 10 years, it might be as high as 71%. Even though heterotopic ossification has a substantial impact on a range of motion, many studies have demonstrated that it does not result in worse patient outcomes [47].

CONCLUSION

Surgical treatment of DDD is still debatable. Nevertheless, regarding the efficacy of the combination approach on the quality of life analyzed with other sorts of surgical and nonsurgical therapy, fusion has shown no significant superiority.

Artificial intervertebral plate rearrangement has become a viable elective to fusion surgery. This treatment involves replacing the damaged disc with an artificial disc that can maintain normal segmental mobility and hence minimize the likelihood of neighboring segment degeneration.

TDR has been shown safe and successful especially in the therapy of degenerative lumbar lower back pain caused by DDD during the last decade. TDR works by mimicking the biomechanical function of an intact intervertebral disc, preserving physiological mobility and perhaps lowering the risk of adjacent segment degeneration as compared to lumbar fusion.

ACKNOWLEDGMENTS : None

CONFLICT OF INTEREST : None

FINANCIAL SUPPORT : None

ETHICS STATEMENT : None

REFERENCES

1. Othman YA, Verma R, Qureshi SA. Artificial disc replacement in spine surgery. *Ann Transl Med.* 2019;7(Suppl 5). doi:10.21037/atm.2019.08.26.
2. Choi YS. Pathophysiology of degenerative disc disease. *Asian Spine J.* 2009;3(1):39. doi:10.4184/asj.2009.3.1.39.
3. Hicks GE, Morone N, Weiner DK. Degenerative lumbar disc and facet disease in older adults: prevalence and clinical correlates. *Spine.* 2009;34(12):1301-6. doi:10.1097/brs.0b013e3181a18263.
4. Palepu V, Kodigudla M, Goel VK. Biomechanics of disc degeneration. *Adv Orthop.* 2012;2012:1-17. doi:10.1155/2012/726210.
5. Kalichman L, Hunter DJ. Diagnosis and conservative management of degenerative lumbar spondylolisthesis. *Eur Spine J.* 2008;17(3):327-35. doi:10.1007/s00586-007-0543-3.
6. Tulder MV, Koes B, Bombardier C. Low Back Pain. *Best Pract Res Clin Rheumatol.* 2002;16(5):761-75. doi:10.1053/berh.2002.0267.
7. Taher F, Essig D, Lebl DR, Hughes AP, Sama AA, Cammisa FP, et al. Lumbar degenerative disc disease: current and future concepts of diagnosis and management. *Adv Orthop.* 2012;2012:1-7. doi:10.1155/2012/970752.
8. Takahashi Y, Ohtori S, Takahashi K. Peripheral nerve pathways of afferent fibers innervating the lumbar spine in rats. *J Pain.* 2009;10(4):416-25. doi:10.1016/j.jpain.2008.10.012.
9. García-Cosamalón J, Del Valle ME, Calavia MG, García-Suárez O, López-Muñiz A, Otero J, et al. Intervertebral disc, sensory nerves and neurotrophins: who is who in discogenic pain?. *J Anat.* 2010;217(1):1-5. doi:10.1111/j.1469-7580.2010.01227.x.
10. Prescher A. Anatomy and pathology of the aging spine. *Eur J Radiol.* 1998;27(3):181-95. doi:10.1016/s0720-048x(97)00165-4.
11. Battié MC, Videman T. Lumbar disc degeneration: epidemiology and genetics. *JBJS.* 2006;88(suppl_2):3-9. doi:10.2106/jbjs.e.01313.
12. Kurunlahti M, Tervonen O, Vanharanta H, Ilkko E, Suramo I. Association of atherosclerosis with low back pain and the degree of disc degeneration. *Spine.* 1999;24(20):2080. doi:10.1097/00007632-199910150-00003.
13. Shah LM, Ross JS. Imaging of degenerative and infectious conditions of the spine. *Neurosurgery.* 2016;79(3):315-35. doi:10.1227/neu.0000000000001323.
14. Yao Q, Wang S, Shin JH, Li G, Wood KB. Lumbar facet joint motion in patients with degenerative spondylolisthesis. *J Spinal Disord Tech.* 2013;26(1):E19. doi:10.1097/bsd.0b013e31827a254f.
15. Rodts MF. Total Disc Replacement Arthroplasty. *Orthop Nurs.* 2004;23(3):216-9. doi:10.1097/00006416-200405000-00012.
16. McAfee PC. The indications for lumbar and cervical disc replacement. *Spine J.* 2004;4(6):S177-81. doi:10.1016/j.spinee.2004.07.003.
17. Jacobs W, Anderson PG, Limbeek JV, Willems PC, Pavlov P, Bartels R. Single or Double-Level Anterior Interbody Fusion Techniques for Cervical Degenerative Disc Disease. *Cochrane Database Syst Rev.* 2004. doi:10.1002/14651858.cd004958.
18. Bono CM, Lee CK. Critical analysis of trends in fusion for degenerative disc disease over the past 20 years: influence of technique on fusion rate and clinical outcome. *Spine.* 2004;29(4):455-63. doi:10.1097/01.brs.0000090825.94611.28.
19. Lee CK, Langrana NA. A review of spinal fusion for degenerative disc disease: need for alternative treatment approach of disc arthroplasty?. *Spine J.* 2004;4(6):S173-6. doi:10.1016/j.spinee.2004.07.002.

20. Scaduto AA, Gamradt SC, Warren DY, Huang J, Delamarter RB, Wang JC. Perioperative complications of threaded cylindrical lumbar interbody fusion devices: anterior versus posterior approach. *Clin Spine Surg.* 2003;16(6):502-7. doi:10.1097/00024720-200312000-00003.
21. Park P, Garton HJ, Gala VC, Hoff JT, McGillicuddy JE. Adjacent segment disease after lumbar or lumbosacral fusion: review of the literature. *Spine.* 2004;29(17):1938-44. doi:10.1097/01.brs.0000137069.88904.03.
22. Gibson JNA, Waddell G, Grant IC. Surgery for Degenerative Lumbar Spondylosis. *Cochrane Database Syst Rev.* 2000. doi:10.1002/14651858.cd001352.
23. Frelinghuysen P, Huang RC, Girardi FP, Cammisa FP. Lumbar total disc replacement part I: rationale, biomechanics, and implant types. *Orthop Clin.* 2005;36(3):293-9. doi:10.1016/j.ocl.2005.02.014.
24. Thavaneswaran P, Vandepeer M. Lumbar artificial intervertebral disc replacement: a systematic review. *ANZ J Surg.* 2014;84(3):121-7. doi:10.1111/ans.12315.
25. Fritzell P, Berg S, Borgström F, Tullberg T, Tropp H. Cost effectiveness of disc prosthesis versus lumbar fusion in patients with chronic low back pain: randomized controlled trial with 2-year follow-up. *Eur Spine J.* 2011;20(7):1001-11. doi:10.1007/s00586-010-1607-3.
26. Yue JJ, Garcia R, Blumenthal S, Coric D, Patel VV, Dinh DH, et al. Five-year results of a randomized controlled trial for lumbar artificial discs in single-level degenerative disc disease. *Spine.* 2019;44(24):1685-96. doi:10.1097/brs.0000000000003171.
27. Ren C, Song Y, Liu L, Xue Y. Adjacent segment degeneration and disease after lumbar fusion compared with motion-preserving procedures: a meta-analysis. *Eur J Orthop Surg Traumatol.* 2014;24(1):245-53. doi:10.1007/s00590-014-1445-9.
28. Zigler JE, Delamarter RB. Five-year results of the prospective, randomized, multicenter, Food and Drug Administration investigational device exemption study of the ProDisc-L total disc replacement versus circumferential arthrodesis for the treatment of single-level degenerative disc disease. *J Neurosurg Spine.* 2012;17(6):493-501. doi:10.3171/2012.9.spine11498.
29. Zigler JE, Blumenthal SL, Guyer RD, Ohnmeiss DD, Patel L. Progression of adjacent-level degeneration after lumbar total disc replacement: results of a post-hoc analysis of patients with available radiographs from a prospective study with 5-year follow-up. *Spine.* 2018;43(20):1395-400. doi:10.1097/brs.0000000000002647.
30. Salzman SN, Plais N, Shue J, Girardi FP. Lumbar disc replacement surgery—successes and obstacles to widespread adoption. *Curr Rev Musculoskelet Med.* 2017;10(2):153-9. doi:10.1007/s12178-017-9397-4.
31. Uschold TD, Fusco D, Germain R, Tumialan LM, Chang SW. Cervical and lumbar spinal arthroplasty: clinical review. *Am J Neuroradiol.* 2012;33(9):1631-41. doi:10.3174/ajnr.a2758.
32. Gamradt SC, Wang JC. Lumbar disc arthroplasty. *Spine J.* 2005;5(1):95-103. doi:10.1016/j.spinee.2004.09.006.
33. Büttner-Janzen K, Guyer RD, Ohnmeiss DD. Indications for lumbar total disc replacement: selecting the right patient with the right indication for the right total disc. *Int J Spine Surg.* 2014;8:12. doi:10.14444/1012.
34. Vital JM, Boissiere L. Total disc replacement. *Orthop Traumatol Surg Res.* 2014;100(1):S1-4. doi:10.1016/j.otsr.2013.06.018.
35. Tropiano P, Huang RC, Girardi FP, Cammisa Jr FP, Marnay T. Lumbar Total Disc Replacement. *J Bone Joint Surg.* 2006;88(1):50-64. doi:10.2106/jbjs.e.01066.
36. Park CK. Total disc replacement in lumbar degenerative disc diseases. *J Korean Neurosurg Soc.* 2015;58(5):401. doi:10.3340/jkns.2015.58.5.401.
37. Frelinghuysen P, Huang RC, Girardi FP, Cammisa FP. Lumbar total disc replacement part I: rationale, biomechanics, and implant types. *Orthop Clin.* 2005;36(3):293-9. doi:10.1016/j.ocl.2005.02.014.
38. Dehn T. Degenerative disc disease: disc replacement. *Ann R Coll Surg Engl.* 2007;89(1):6. doi:10.1308/003588407x160792.
39. Glassman SD, Bridwell K, Dimar JR, Horton W, Berven S, Schwab F. The impact of positive sagittal balance in adult spinal deformity. *Spine.* 2005;30(18):2024-9. doi:10.1097/01.brs.0000179086.30449.96.
40. Rundell SA, Day JS, Isaza J, Guillory S, Kurtz SM. Lumbar total disc replacement impingement sensitivity to disc height distraction, spinal sagittal orientation, implant position, and implant lordosis. *Spine.* 2012;37(10):E590-8. doi:10.1097/brs.0b013e318241e415.

41. Laouissat F, Allain J, Delecrin J. Intraoperative determination of lumbar prosthesis endplate lordotic angulation to improve motion. *Orthop Traumatol Surg Res.* 2015;101(1):109-13. doi:10.1016/j.otsr.2014.11.008.
42. Nayak AN, Doarn MC, Gaskins RB, James CR, Cabezas AF, Castellvi AE, et al. Postero-lateral disc prosthesis combined with a unilateral facet replacement device maintains quantity and quality of motion at a single lumbar level. *Int J Spine Surg.* 2014;8. doi:10.14444/1031.
43. Werner JH, Rosenberg JH, Keeley KL, Agrawal DK. Immunobiology of periprosthetic inflammation and pain following ultra-high-molecular-weight-polyethylene wear debris in the lumbar spine. *Expert Rev Clin Immunol.* 2018;14(8):695-706. doi:10.1080/1744666x.2018.1511428.
44. Veruva SY, Lanman TH, Isaza JE, MacDonald DW, Kurtz SM, Steinbeck MJ. UHMWPE wear debris and tissue reactions are reduced for contemporary designs of lumbar total disc replacements. *Clin Orthop Relat Res.* 2015;473(3):987-98. doi:10.1007/s11999-014-4029-4.
45. Beatty S. We need to talk about lumbar total disc replacement. *Int J Spine Surg.* 2018;12(2):201-40. doi:10.14444/5029.
46. De la Garza-Ramos R, Abt NB, Kerezoudis P, McCutcheon BA, Bydon A, Gokaslan Z, et al. Deep-wound and organ-space infection after surgery for degenerative spine disease: an analysis from 2006 to 2012. *Neurol Res.* 2016;38(2):117-23. doi:10.1080/01616412.2016.1138669.
47. Park HJ, Lee CS, Chung SS, Park SJ, Kim WS, Park JS, et al. Radiological and clinical long-term results of heterotopic ossification following lumbar total disc replacement. *Spine J.* 2018;18(5):762-8. doi:10.1016/j.spinee.2017.09.003