

Interspinous Posterior Devices IPD: A Miracle Cure for the Lumbar Spinal Stenosis?

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Abstract

Interspinous Posterior Device (IPD) is a term used to identify a relatively recent group of implants used to treat lumbar spine stenosis with the presumed aim of a dynamic motion control systems. In the last 10 years there was a very large use of these implants. Despite this, no long-term clinical follow-up are available. In the literature is evident the high rate of reoperation, recurrence of symptoms and progression of degenerative changes. But his main question is: if these devices are effectively a miracle cure for the common problem of the lumbar spinal stenosis, why actually the use of IPD remains extremely controversial and should be investigated further?

Keywords: IPD; IFD; Biomechanics; Lumbar stenosis; Interspinous devices; Lumbar instability

Interspinous Posterior Device (IPD) is a term used to identify a relatively recent group of implants used to treat lumbar spine stenosis with the presumed aim of a dynamic motion control systems [1-4]. All of IPD are small devices implanted between the vertebral spinous processes [5-11]. After implantation, the device must be opened or expanded to distract (open) the neural foramen and decompress the roots [5-7]. These implants aim to restrict painful motion while otherwise enabling normal motion [12,13]. These devices (also called interspinous spacers) distract the laminar space and/or spinous processes and restrict extension. This procedure theoretically, enlarges the neural foramen and decompresses the roots, and could decompress the cauda equina in patients with spinal stenosis and neurogenic claudication [11-15].

The IPD have evolved over the years, being classified into bound or not bound depending on the presence or absence of a dynamic movement control only of the extension or both flexion-extensions [16]. A further evolution has also led to the development of IFD or Interspinous Fusion Devices. These implants have as their goal the interspinous bone fusion and, in my opinion, they cannot be classified as dynamic motion control systems because their target is metameric fusion [17-22]. In the last 10 years there was a very large use of these implants. Despite this, no long-term clinical follow-up are available. In the literature is evident the high rate of reoperation, recurrence of symptoms and progression of degenerative changes [7-26]. But his main question is: if these devices are effectively a miracle cure for the common problem of the lumbar spinal stenosis, why actually the use of IPD remains extremely controversial and should be investigated further? If we exclude the problems of the system's high cost, in my opinion, the main problem remains the pathologic substrate on which the devices have to act.

Biomechanical Consideration

If it is true that such devices can be used in patients with mild to moderate stenosis [5-8], either central or foraminal, or in low-grade spondylolisthesis without lysis (with poor or at least questionable results), it is also true that such devices can be used in cases in which the lumbar degenerative cascade is in active phase. The lumbar degenerative cascade, when is in active phase, has as its first step the disc degeneration, more or less advanced in relation to the extension and the continuity in time of the injury itself. Normally, as defined by Kirkaldy-Willis [27], the biomechanics of the lumbar spine follows a law that is called "rule of spine loading", in which the axial load of the body is distribute for the 80% on the intervertebral disc and 20% on the

posterior structures (joints, ligaments and muscles) [1-4,21-25]. Disc degeneration transfers the axial load posteriorly, reversing the load distribution. This leads to the overload of the facet joints resulting in joint laxity, reduced competence of the joint capsules and hypermobility. The hypermobility stimulates the inflammatory reaction of adjacent tissues, this activates the Fractalkine in the yellow ligament [27] causing the increasing of the inflammatory cells recruitment which degrade the extracellular matrix of the ligament making it lose elasticity and causing hypertrophy. It is well documented the role of fractalkine in the development of numerous inflammatory diseases (rheumatoid arthritis, dermatitis, etc.) and in ligaments and joints involved in inflammatory processes caused by instability (eg, joint capsules, ligaments, and synovium). The inflammatory process involves these tissues so the fractalkine over expression is activated; thus causing the recruitment of mononuclear cells within the LF feeding the inflammation and causing vascular injury and angiogenesis [27]. Moreover such an increase in mononuclear activity cause a proliferation of fibroblasts, (for over expression of TGF beta mRNA resulting in increased collagen fibers) and inflammatory cells in LF. This inflammatory cells activity in the LF causes rupture of the extracellular matrix (for activation of metalloproteinase MMP2) due to the elastin degradation, resulting in loss of elasticity of the ligament and subsequent hypertrophy [27]. In addition, the disk protrusion and prolapse and the yellow ligament hypertrophy cause reduction of the spinal canal diameter causing stenosis. In this phase, in which the articular hypertrophy generates foramina stenosis, and the collapse of the disc generates ligamentous hypertrophy, the stenosis becomes symptomatic, but the main substrate remains hypermobility anyway.

The *non bound IPD* is implanted between the vertebral spinous processes [28]. After implantation, the device is opened to distract the neural foramen and decompress the nerves. This procedure brings to the transfer of the axial load anteriorly on an already degenerated disc. In addition, the distraction that has to be made to open the foramina

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alters the sagittal balance of the lumbar spine. The sagittal balance refers to the axial load in relation to the external environment, and its integrity gives the elastic properties to the spine.

The sagittal balance may be defined by the proper balancing of the physiological curvature, which is able to transfer the axial load through the pelvis and the femoral heads to the ground. This is possible by a perpendicular vector obtained from the external acoustic meatus, passing by the middle of the L5 endplate, the femoral heads and that reaches the floor. This line has to be posteriorly to the line between the femoral heads and it is essential that the curvatures of the spine are kept as physiological as possible. In particular, it is essential, at the lumbar level, the preservation of lordosis [1-4]. The goal of such devices is the insertion between the spinous processes and their distraction, as well as the transfer of the axial load anteriorly on a degenerated disc. These processes cause the alteration of the spinal biomechanics, impacting negatively on the sagittal balance. These actions cause changes in posture, spinal- pelvic axis rotations and alteration of the dorsal and cervical curvatures, which have as their purpose to compensate the alteration of the sagittal balance, but which have the effect of increasing the progression of spinal degeneration. Such patients are in a condition of spinal imbalance [7-9,12,14,16,18,29-34].

If such devices give an immediate improvement of the symptoms thanks to the foramen opening, long-term alteration of the biomechanics causes an acceleration of the degenerative process, either of the treated level or of the adjacent ones. Moreover, the overload of the spinous processes can result in their fractures or in lacerations of the posterior longitudinal ligament, causing the mobilization of the device [7-14,35,36].

The *bound IPD* that have the presumed function of neutralizing the excessive movement in flexion-extension of the spine, when inserted have the goal of distraction of the spinous processes to open the foramina, which alters the lumbar biomechanics. Then even if these are able to control the excessive degrees of movement in flexion and extension, they have as consequence the non-physiological movement of spinal unit, with the same consequences as described before for the non bound IPD.

Specifically we can assure that the binding and unbinding properties are specific for the IPD; in particular the bound IPD have a particular concept in materials and design for which it must be adherent to the above and below spinal process (such as WALLIS implant or DIAM implant for example). This design complains laces, strings and much more. The unbound IPD instead have no adherence to the spinal processes (such as APERIUS, X-STOP, BACJAC ecc). This difference in design reflects a difference in biomechanical behaviour: in fact the unbound devices restrict (and no arrest) the motion only in flexion and the bound devices restrict the motion both in flexion than in extension.

Moreover the metameric instability is not limited to flexion-extension movements, but also and above all of lateral bending and axial rotation. These movements are often associated with the flexion-extension when complex movements are done. An interspinous device cannot control the rotation and the lateral bending in any way. Those movements are burdened by excessive load after the insertion of the device, which then enhances and accelerates the degenerative process [7-13,37,38].

Conclusions

The real problem is the biomechanic behaviour of the spine when these devices are implanted. So in my opinion the real indications for IPD implants are extremely restricted, and reserved only to the bound

IPD, because this type of devices is the only ones with a slight control of the hypermovement. But when there are clear signs of metameric instability, these devices should never be implanted.

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