Sitting versus standing: Does the intradiscal pressure cause disc degeneration or low back pain?

Andrew Claus a,*, Julie Hides a, G. Lorimer Moseley b, Paul Hodges a

a Division of Physiotherapy, School of Health and Rehabilitation Sciences, The University of Queensland, Brisbane, St. Lucia, Qld 4072, Australia
b Department of Physiology, Anatomy & Genetics & fMRJB Centre, University of Oxford, Oxford, UK

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Abstract

Studies of lumbar intradiscal pressure (IDP) in standing and upright sitting have mostly reported higher pressures in sitting. It was assumed clinically that flexion of the lumbar spine in sitting relative to standing, caused higher IDP, disc degeneration or rupture, and low back pain. IDP indicates axial compressive load upon a non-degenerate disc, but provides little or no indication of shear, axial rotation or bending. This review is presented in two main parts. First, in vivo IDP data in standing and upright sitting for non-degenerate discs are comprehensively reviewed. As methodology, results and interpretations varied between IDP studies, in vivo studies measuring spinal shrinkage and spinal internal-fixator loads to infer axial compressive load to the discs are also reviewed. When data are considered together, it is clear that IDP is often similar in standing and sitting. Secondly, clinical assumptions related to IDP in sitting are considered in light of basic and epidemiologic studies. Current studies indicate that IDP in sitting is unlikely to pose a threat to non-degenerate discs, and sitting is no worse than standing for disc degeneration or low back pain incidence. If sitting is a greater threat for development of low back pain than standing, the mechanism is unlikely to be raised IDP.

Keywords: Intervertebral disc; Intradiscal pressure; Lumbar; Sitting; Low back pain

1. Introduction

Intradiscal pressure (IDP) is the hydrostatic pressure measured in the nucleus pulposus of a non-degenerate intervertebral disc. Standing and sitting are everyday activities, which has made the findings of posture and lumbar IDP studies immediately relevant and memorable. Much lower IDP has been reported in sitting than in standing. It has been reported that “in standing, the disc pressure is about 35% of the pressure in relaxed sitting without back support” (Andersson et al., 1975). Sitting on a horizontal surface involves lumbar flexion relative to standing (Maksous et al., 2003; Scannell and McGill, 2003), and it has been assumed clinically that lumbar flexion was the cause of higher IDP, tensile stress to the annulus, disc degeneration and low back pain (Nachemson and Morris, 1964; Andersson and Ortengren, 1974; Andersson et al., 1974a,b; Nachemson, 1975). IDP data and related assumptions have formed a basis for clinical advice to advocate erect sitting postures for the prevention of spinal complaints (Brunswic, 1984; Pynt et al., 2002). However, some studies have reported near identical pressures “with the subject either sitting [relaxed without back support] or standing... the intradiscal pressure was approximately 300 kilopascals [0.30 MPa]” (Schultz et al., 1982). Similar pressures in standing and sitting would undermine a common clinical basis for advocating erect sitting postures, and encourage broader inquiry into aetiology for pathology and pain at the lumbar spine.

This paper uses two approaches to examine IDP in upright sitting (relaxed without back support). First, in vivo IDP data for non-degenerate lumbar discs in standing and upright sitting are comprehensively reviewed, with consideration for measurement methodologies, results, and
interpretations. Second, the assumed clinical links between IDP in sitting, disc degeneration and low back pain are considered through basic in vitro studies (IDP and degeneration), spinal imaging and epidemiologic studies (sitting, disc degeneration and low back pain). The biomechanical emphasis of this review is the axial compressive load affecting intervertebral discs, as this relates to IDP. Spinal pain can relate to other mechanisms of loading, load-bearing structures, neuro-muscular control, tissue physiology and psychosocial factors, but those aspects are beyond the scope of this review.

2. IDP measurement: development of methodology

Over the past thirty years, there has been a clear progression of the accuracy and sensitivity of in vitro IDP measurement. It is important to review these developments in methodology, to provide context for interpretation of results. With IDP measurement, the pioneering work of Nachemson, Morris, Elfstrom, Andersson and Ortingen in the 1960s and 1970s, helped to pave the way in the study of spinal biomechanics. The first pressure transducer used a polyethylene membrane (elastic tubing) over the tip of a hollow liquid-filled needle, connected to an electromanometer (Nachemson, 1963; Nachemson and Morris, 1964; Nachemson, 1965, 1966; Okushima, 1970). Research subjects for these studies had no apparent disc degeneration on radiograph or discography, but did suffer from low back pain. Early studies showed similar pressure readings with changes in transducer orientation, which provided evidence that the nucleus pulposus of a non-degenerative intervertebral disc could behave hydrostatically. Unfortunately, apparatus used in early studies had a number of limitations. The polyethylene membrane, covering the needle tip, had insufficient sensitivity for dynamic pressure measurement. The fluid-filled needle could also alter the measured pressures if it was bent more than 20°. Movement and changes in the research subject’s posture could induce at least this degree of bend in the needle (Nachemson and Elfstrom, 1970). Table 1 shows a list of studies that reported IDP data from standing and sitting in non-degenerate discs (with population studied and transducer type).

In the 1970s, major advances in transducer technology and calibration increased accuracy, and produced results ~25–33% lower than earlier measures (as shown in Figs. 1 and 3). Calibration to body temperature, rather than room temperature, improved precision of recordings; and the liquid-filled needle was replaced with a piezoresistive semiconductor strain gauge (embedded in an epoxy resin, and covered with a pressure sensitive membrane, at the tip of a 0.8 mm diameter transducer needle) (Nachemson, 1975). It was reported that this new transducer needle could bend as much as 40° without influencing accuracy of pressure measurement (Nachemson and Elfstrom, 1970). Furthermore, research subjects with non-degenerate discs and no history or low back pain were used, but it is uncertain whether a history of back pain influenced IDP measurements. From 1983, there were no further studies of IDP with non-degenerate discs in standing and sitting until the late 1990s.

In 1999, two separate research teams reported IDP measurements. One team from Japan (Sato et al., 1999) used a similar design to the 1970s apparatus, but with the piezoresistive sensor positioned laterally in the transducer needle. In this way, the pressure was detected at a window in the guiding needle, rather than at the needle tip. Another team in Germany (Wilke et al., 1999) avoided the problem of having a needle in situ, by using telemetry to record from an implanted transducer measuring 1.5 mm diameter, by 7 mm in length. This implanted transducer had the advantage of transmitting data during a variety of daily activities over 24 h, compared with other studies that reported trials of ~15 s or less (Schultz et al., 1982).

3. Comparison of IDPs in standing and sitting

3.1. Method for comparison of studies

IDP results from all published studies with individual subject data from non-degenerate discs in standing and sitting were converted to megapascals (MPa), and are shown in Fig. 1. Broad error bars shown in Fig. 1 denote the range of data values for each column, except for one study with narrow error bars to denote the standard deviation (range data were not available). Two studies that did not report individual data were also included because they involved authors from earlier studies, and the lower pressures reported in these papers were an important finding. The

Table 1

Comparison of subjects and studies that report standing and upright sitting IDP in non-degenerate discs

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>L3-4, n</th>
<th>L4-5, n</th>
<th>Low back pain</th>
<th>Transducer type</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nachemson*</td>
<td>1964/1965</td>
<td>6</td>
<td>4</td>
<td>Yes</td>
<td>Liquid-filled</td>
</tr>
<tr>
<td>Okushima</td>
<td>1970</td>
<td>10</td>
<td>20</td>
<td>Yes</td>
<td>Liquid-filled</td>
</tr>
<tr>
<td>Nachemson and Elfstrom</td>
<td>1970</td>
<td>7</td>
<td>No</td>
<td>Piezoresistive</td>
<td></td>
</tr>
<tr>
<td>Andersson et al.</td>
<td>1974</td>
<td>4</td>
<td>No</td>
<td>Piezoresistive</td>
<td></td>
</tr>
<tr>
<td>Schulze et al.</td>
<td>1982</td>
<td>4</td>
<td>No</td>
<td>Piezoresistive</td>
<td></td>
</tr>
<tr>
<td>Sato et al.</td>
<td>1999</td>
<td>8</td>
<td>No</td>
<td>Piezoresistive, side-window</td>
<td></td>
</tr>
<tr>
<td>Wilke et al.</td>
<td>1999</td>
<td>1</td>
<td>No</td>
<td>Piezoresistive, implanted</td>
<td></td>
</tr>
</tbody>
</table>

* Data from the Nachemson and Morris (1964) and Nachemson (1965) papers are combined, as indicated by the author in 1965.

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Data from subsequent studies were all from subjects with no history of back pain (Table 1).

To assist interpretation of pressures in MPa, Fig. 2 shows early (Nachemson and Morris, 1964) and recent IDPs (Sato et al., 1999) relative to common fluid pressures. Pressure results in Figs. 1–3 from early studies of IDP (\(p_a\)) were multiplied by 0.098 to convert results from kg/cm² to MPa, (Nachemson and Morris, 1964; Nachemson, 1965; Okushima, 1970).

3.2. IDP results in standing and sitting

The earliest data, obtained with a polyethylene tipped liquid-filled transducer, in subjects with a history of low back pain, showed the mean pressure in sitting to be 1.24 MPa (11 atmospheres, shown in Fig. 1) (Nachemson, 1965). In contrast, more recent studies with piezoresistive transducers and subjects with no history of low back pain, have reported smaller pressures of 0.5–0.6 MPa (~5–6 atmospheres) for standing or sitting (Sato et al., 1999; Wilke et al., 1999). Aside from large variation in means between studies, there is also large variation between subjects (Okushima, 1970; Andersson et al., 1982; Sato et al., 1999).

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In contrast, data from the implanted transducer in a single subject indicated marginally higher IDP in standing (0.49 MPa) than in sitting (0.47 MPa, Figs. 1 and 3) (Wilke et al., 1999). The implanted transducer presented advantages for data collection and accommodation of the subject to the apparatus; being a smaller apparatus that did not protrude from the disc, and in situ for recording over 24 h. Despite these advantages, results from a single-subject study must be considered with some caution. At present, the only alternative methods to infer IDP differences between standing and sitting are by studies of change in spinal height or load upon internal fixators.

3.3. Spinal height and load cell measures in standing and sitting

In vitro studies have shown a near-linear relationship between increased axial compressive load applied to an intervertebral disc and increased IDP (Nachemson, 1963; Berkson et al., 1979). In vivo, progressively higher pressures measured from lying, to standing, to lifting a weight, provide evidence that greater compressive load upon the disc is associated with greater IDP (Nachemson and Elfstrom, 1970; Schultz and Anderson, 1981). Several studies have sought to measure disc compression either indirectly (stadiometry) or directly (load-cell equipped spinal fixators). Although IDP has not been concurrently measured with disc compression in vivo, compression measures in standing and sitting permit inference as to which posture causes a higher IDP.

Stadiometry is the measurement of spinal height, and is used to infer the effect of disc compression in different activities. Activities with greater compressive load cause lesser disc hydration, and thus reduce spinal height (Eklund and Corlett, 1984; Kingma et al., 2000; Hutton et al., 2003). Stadiometry has the advantages of providing a non-invasive, intra-subject comparison of sustained disc compression (e.g. 30 min or more) with different postures. Subjects commonly lose more height in standing than they do in sitting (1–5 mm difference) (Althoff et al., 1992; Lievseth and Drerup, 1997), which infers that standing causes more disc compression than sitting.

Load cells within steel bars for surgically implanted spinal fixators have been used as a more direct measure of compression and flexion torque between two spinal segments (Rohllmann et al., 2001). Data from load-cell fixators have been reported for ten subjects, with spinal fixators applied to various levels from T1-L4 (data transmitted by telemetry). Again, sitting postures showed lower compression than upright standing, consistent with implanted-transducer disc pressure and stadiometry, but contrary to needle-transducer results.

Each experimental approach has limitations. Stadiometry of different postures has a small effect size, but relatively large variation between subjects and between trials for the same subject (Althoff et al., 1992; McGill et al., 1996; Lievseth and Drerup, 1997; van Deursen et al., 2005). The load-cell fixator study was a series of single cases, with...
fixators at a variety of spinal levels (Rohllmann et al., 2001).
However, taken together these studies provide evidence in
support of results from the implanted-transducer study,
to show that sitting may be associated with less disc com-
pression than standing.

3.4. Interpretation of pressure in standing and sitting:
developments with methodology

Results obtained with IDP measurement appear to be
greatly influenced by the experimental methodology.
Consequentely, there has been a parallel development of
interpretations for the results, and how they inform rehabil-
itation and ergonomic advice. For example, initial
in vitro results from 1963 showed similar pressures in exten-
sion or flexion of a loaded spinal segment (Nachemson,
1963). Subsequent in vivo studies reported a difference
between pressures in standing (extension) or sitting (flex-
ion) (Nachemson and Morris, 1964), which led the author
to state, “it might appear unreasonable that the load
decreases in shifting from sitting to standing” (Nachemson,
1966). At the time it was hypothesised that sitting might
have caused higher muscle activity in psosas major, and thus
higher IDP. It was also suggested that disc degeneration
was a result of tensile fractures of the annulus fibrosus
(Nachemson and Morris, 1964). Pressures in sitting of
~1.2 MPa were considered to support a mechanical theory
of ruptures to the posterior annulus fibrosus, leading to
irritation of free nerve endings in the posterior disc, thus
contributing to low back pain (Nachemson and Morris,
1964).

In the 1970s, electromyography showed that muscle
activity of psosas major did not explain IDP differences
between standing to sitting. The new hypothesis was that
lumbar flexion caused higher IDP than lumbar extension
(Andersson et al., 1974a). Several papers reported a direct
link between increased lumbar flexion and increased IDP in
sitting (Andersson and Ortnegren, 1974; Andersson et al.,
1974a,b; Andersson et al., 1975; Nachemson, 1975). Based
on the assumption that lower disc pressure was advanta-
geous for reducing low back pain, advice followed the
mechanical theory for disc ruptures causing chemical irri-
tation and pain. ‘Back Schools’ recommended postures and
movements that had been shown to involve lower disc pres-
sures, including the ‘take home’ message for Physiothera-
pist’s ergonomic advice that “straight standing is better
than unsupported sitting” (Nachemson, 1975).

Notably, the same authors recognised that there was no
direct evidence to link sitting and the aetiology of disc
degeneration, mechanical ruptures, or pain (Nachemson,
1975). However, with the high incidence of disc degenera-
tion, pain and loading the spine in sitting, correlation of
these variables may have been interpreted as causality.

The difficulty in determination of pain aetiology, and a lack
of alternative disc degeneration paradigms may have
strengthened assumptions that IDP in sitting could be
adverse for disc health.

With progression of transducer technology and calibra-
tion, the lower pressures reported gave reason to question
assumptions about disc health being affected by the IDP
in sitting. One example was a lifting-task study, which
moved away from labeling the IDP in sitting as threaten-
ing, with the statement that “it is clear that in the upright
sitting and standing positions there are small differences
[in pressure]” (Nachemson and Elfstrom, 1970). By the late
1970s, standing pressure was reported as low as 0.33 MPa
(3 atmospheres) at the L3-4 disc (Andersson et al., 1978),
and a 1982 lifting-study reported that pressures in sitting
and standing were similar (Schultz et al., 1982).

Further challenging previous interpretation of the data,
evidence has suggested that the position of spinal flexion or
extension is a poor predictor of IDP. Lumbar flexed postu-
tures, such as slumped sitting with elbows resting on thighs,
can demonstrate similar pressure to upright sitting
(Andersson et al., 1974a) and lower pressure than standing
(Wilke et al., 1999). Studies of lifting also measured IDP
with flexed and straight spinal postures. Although some
studies reported higher IDP in lumbar flexed (stoop lift)
than straight spine (squat lift) strategies (Nachemson and
Elfstrom, 1970; Wilke et al., 2001), others showed similar
pressures for both alternatives (Andersson et al., 1976;
Andersson et al., 1978). This variability suggests that the
degree of spinal flexion or extension is a lesser determinant
than other variables affecting IDP (e.g. individual variation
in muscle activity, facet joint load or ligamentous tension
for a given task).

In summary, pressures for upright standing and sitting
appear to be ~0.5–0.6 MPa (~5–6 atmospheres) with
inter-subject variation of ~0.2–0.3 MPa (~±2–3 atmos-
pheres), based on mean pressures from piezoresistive nee-
dle-transducer studies (Andersson et al., 1974a; Schultz
et al., 1982; Sato et al., 1999). Contrary to results from
needle-transducer studies, data from an implanted-transducer
(Wilke et al., 1999), studies of spinal shrinkage and internal
fixator loads (Althoff et al., 1992; Lievseth and Drenter,
1997; Rohllmann et al., 2001) infer that similar or margin-
ally greater IDP is likely in standing than sitting. Thus, a
pertinent question is whether IDP of 0.3–0.7 MPa in sitting
poses a mechanical threat to the annulus fibrosus of a non-
degenerate disc, an elevated risk of low back pain or both.

4. Does IDP in sitting pose a threat to the annulus fibrosus or
low back pain?

4.1. Does IDP in sitting threaten or protect the annulus
fibrosus in vitro?

In the 1960s and 1970s, horizontal force of IDP upon
the annulus was proposed as the mechanical cause for ten-
sile rupture of disc fibres (Nachemson and Morris, 1964;
Nachemson, 1975). Three assumptions linked this tensile
rupture model to the IDP of non-degenerate discs in sit-
ting. First, it was theorised that an optimum IDP for inter-
vertebral discs would be lower than that the IDP in sitting

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(Nachemson, 1975), despite little data on annulus cell physiology. Second, higher IDP was equated with higher risk of annulus rupture and low back pain, because tensile stress to annulus fibres was mainly equated with compressive load (Nachemson and Elfström, 1970), with less attention to loads in shear, axial rotation or bending. Thirdly, hydrostatic behavior of the nucleus pulposus indicated an even pressure per unit area at the annulus fibrosus and adjacent endplates (Nachemson and Elfström, 1970), but not the load distribution within the annulus. Advances with in vitro research methods have provided new insights into annulus response to IDP in sitting, biomechanics of disc rupture and load distribution within the annulus.

One aspect affecting annulus fibrosus integrity is the metabolic response of disc cells to compressive loading. Composition and structure of the annulus matrix are determined by the balance of protein synthesis and degradation. Cells harvested from the inner annulus fibrosus and nucleus pulposus undergo metabolic change in response to hydrostatic pressure in vitro (Handa et al., 1997). Pressure of 0.3 MPa applied for 2 h (the lower limit of 0.3–0.7 MPa in sitting or standing) can stimulate proteoglycan synthesis and inhibit degradation. In contrast, 0.10 MPa pressure (similar to the pressure in lying down) can inhibit synthesis and facilitate degradation. Pressures within normal physiological range appear to provide an essential mechanical stimulus for maintenance of the proteoglycan matrix, and consequent load bearing capacity of the intervertebral disc.

Investigation of disc biomechanics with axial compressive loading has indicated that tensile rupture of annulus fibres is unlikely with compression alone (Adams and Dolan, 1995; Fujita et al., 1997). An in vitro study of 43 isolated spinal motion segments showed that a mean repetitive load of 3076 N compression (range: 1500–6000 N) applied at 0.66 Hz for 4 h, resulted in trauma at the vertebral endplates or anterior vertebral bodies, but not the disc (Adams and Hutton, 1983). Twenty of the spinal motion segments were then loaded to failure, with the pressure increased by a further 3000 N/s. Only 3 of the 20 segments suffered a disc protrusion or prolapse. Failure most commonly occurred at the vertebral body (Adams and Hutton, 1983). Similar results were found in a more recent study with eight in vitro spinal segments (Ranu, 1990). A mean critical compressive load of 4370 N (SD 1030) was associated with a mean IDP of 3.02 MPa (SD 0.76) for vertebral endplate failure. In summary, compressive loads ~10 times the magnitude of upper-body weight rarely cause annulus fibrosus failure in a non-degenerate disc. With axial compression alone, IDP of ~0.5–0.6 MPa in sitting appears trivial for non-degenerate discs, when compared with ~2.26–3.78 MPa required for endplate failure. Loading mechanisms other than simple axial compression must be involved in initiating disc degeneration.

Combined loading of spinal motion segments with compression (1,334 N), flexion (7°) and axial rotation (<3°) for 3–13 h, has been observed to cause disc failure in all 14 discs that were tested (Gordon et al., 1991). While this study aimed to simulate repetitive loading in a very flexed position, combined loading can also occur throughout range. Even in mid-range spinal positions, neuromuscular control is essential to prevent spinal motion segments from buckling in shear, axial rotation and/or bending (Crisco and Panjabi, 1992; Adams, 1995). The potential for these combined loads to occur, and evidence that combined loads can cause disc trauma, raise the question of how combined loads affect disc integrity differently to axial compression alone.

Structurally the annulus fibrosus is a composite material, with 20–40 layered sheets (lamellae) of collagen fibres in a proteoglycan matrix. Although pressure is relatively evenly distributed in the nucleus of a healthy disc (i.e. hydrostatic behaviour) (Sato et al., 1999), this even load distribution from nucleus to annulus does not indicate how load is distributed within the annulus fibrosus. Profilometry was developed as a progression from IDP measurement, to measure both IDP and annulus fibrosus stress profiles (McNally and Adams, 1992; McNally et al., 1992). Typically profilometry utilizes a piezoelectric transducer in a lateral window of the needle (similar to that used by Sato et al.) to measure changes in compressive load as the transducer is drawn across a diameter of an intervertebral disc (McNally and Adams, 1991). Non-degenerate discs loaded in a neutral position with axial compression, show uniform stress profiles at the nucleus pulposus and inner - middle annulus fibrosus layers (lower stress at outer annulus). With combined loading in compression plus antero-lateral bending, stress profiles in the annulus fibrosus were not uniform, and 10 of the 22 segments failed at the disc (McNally et al., 1993) (compared with 3 out of 20 with axial compression alone (Adams and Hutton, 1983)). All 10 discs that failed in combined loading showed concentrations of higher stress in the annulus fibrosus (especially the posterior annulus). Of 12 that failed at the vertebral endplate, only two demonstrated stress concentrations in the annulus. That is, combined loading can produce regions of higher stress in the posterior annulus (~1.5 × IDP), and these stress concentrations were strongly associated with disc prolapse (McNally et al., 1993).

Identification of these stress concentrations with combined loading, shed new light on disc pathomechanics. Ensuing studies of non-degenerate discs have demonstrated annulus stress concentrations as a consequence of reduced IDP, caused by trauma to vertebral endplates (0–80% reduction in IDP) (Adams et al., 2000a) or sustained compressive load (13–36% reduction in IDP with 3–6 h at 2–4 × upper body weight) (Adams et al., 1996). Degenerate discs commonly display this combination of endplate trauma, reduced IDP (relative to annulus stress) and stress concentrations in the annulus fibrosus. Instead of higher IDP being adverse for the disc, reduced IDP relative to concentrations of annulus stress appear more indicative of adverse loading.

In non-degenerate discs, horizontal stress applied by IDP acts to maintain the annulus lamellae in close apposition. If...
IDP is lower than compressive load to the annulus, inner and outer laminae can buckle and collapse away from middle laminae, causing failure of the inter-lamina matrix, cellular trauma and separation of the lamellae (Adams et al., 1994; Adams and Dolan, 1995; Bruehlmann et al., 2004). Separation and collapse of inner lamellae towards the nucleus is observed in early stages of disc degeneration in vitro (Seroussi et al., 1989), as well as formation of local and circumferential annulus tears in animal models (Iatridis and Gwynn, 2004). This breakdown or delamination of the disc’s composite structure make it more vulnerable to subsequent loading. Trauma to vertebral endplates and interlaminar matrix will also affect disc nutrition, cell physiology and distribution of load to facet joints, but examination of these changes is beyond the scope of this IDP review.

In summary, health and integrity of the annulus fibrosus appears dependent upon many variables including hydrostatic pressure as a stimulus for matrix metabolism, IDP to support and distribute load to the annulus, and vertebral endplate integrity. Compression alone (measured via IDP) is unlikely to pose a threat to a non-degenerate disc, but combinations of shear, axial rotation or bending with compression may. Current research methods preclude prediction of relationships between common postures, loads and initiation of disc degeneration. Further tools and methods will be needed to predict the complex interactions between variables such as spinal physiology, an individual’s spinal anatomy and neuro-motor control (Adams et al., 2000b; Natarajan et al., 2004).

4.2. Does sitting predict disc degeneration or low back pain?

Considering that IDP is of similar magnitude in standing and sitting, it is necessary to evaluate the clinical assumption that exposure to sitting poses a greater risk of disc degeneration and low back pain than exposure to standing. In 1975, an exploratory epidemiological study of people seeking acute medical care, was the first to report a link between sedentary occupations (sitting), reduced disc space on X-ray and acute low back pain (Kelsey, 1975; Kelsey and Hardy, 1975). Occupational motor vehicle driving was reported to have an even stronger relative-risk for reduced disc space and low back pain. The author cautioned that the methodology had limitations, such as the population studied, questionnaire design, response rate, and the large number of associations tested (Kelsey, 1975). The author also stressed that further investigations were needed to confirm or refute the association of sitting, disc degeneration and low back pain. A subsequent study with 45 pairs of identical twins (Battie et al., 2002) refuted the association. One individual from each of the pairs, had more than five times the lifetime exposure to driving a motor vehicle than their twin. No difference in lumbar disc degeneration on MRI, or incidence of low back pain occurred with prolonged exposure to vibration in sitting (Battie et al., 2002). Likewise, greater exposure to sitting at work was no worse for disc degeneration (Battie et al., 1995). Given that sitting commonly involves lumbar flexion relative to standing (Makhsous et al., 2003; Scannell and McGill, 2003) and recent epidemiologic studies (Battie et al., 1995, 2002), lumbar flexion associated with sitting appears to be no worse for disc health or LBP than relative extension in standing.

5. Conclusions and implications

Since the initial studies of IDP, new tools and methods have continued to advance understanding of pathomechanics and neuro-muscular control related to spinal degeneration and low back pain. Current evidence shows similar IDP in standing and upright sitting postures. In vitro biomechanics show that the axial compression in sitting is unlikely to pose a threat to non-degenerate discs. Epidemiology shows that sitting is no worse than standing for incidence of disc degeneration or low back pain. If sitting is a greater threat for development of low back pain than standing, the mechanism is unlikely to be raised IDP.

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References


Andrew Claus MPhty (Manip), BPhty (Hons), is completing PhD studies with Paul Hodges, Julie Hides and Lorimer Moseley advising for the project, using fine-wire EMG to examine spinal extensor muscles with sitting postures (The University of Queensland, School of Health and Rehabilitation Sciences, Australia). Physical interests include rowing, infantry training, road cycling, track & field, long-track speed skating, physical theatre and hiking, etc. but recent years have mostly involved standing and sitting. Future studies will continue to focus upon neuro-muscular coordination of the lumbar spine.

Julie Hides PhD, MPhtySt, BPhty is a Senior Lecturer in the Division of Physiotherapy at the University of Queensland, and the Clinical Supervisor of the UQ/ Mater Hospital Back Stability Clinic in Brisbane, Australia. Her research interests include therapeutic exercise for low back patients and the use of real-time ultrasound imaging in rehabilitation. She was recently invited by the European Space Agency to be a member of the Topical Team for Low Back Pain and is currently involved in research involving lumbo-pelvic stability in elite sporting teams.

Lorimer Moseley PhD, BAppSc(Phyt)(Hons) is a clinical and research physiotherapist. He has doctorate from University of Sydney Faculty of Medicine, was awarded a Research Fellowship from the Australian National Health & Medical Research Council and then a Senior Lectureship in Pain Sciences at the University of Sydney School of Physiotherapy. He is now Nuffield Medical Research Fellow at Oxford Centre for fMRI of the Brain and the Pain Imaging Neuroscience Group, University of Oxford, United Kingdom. His current research focuses on cortical mechanisms underlying chronic and complex pain states and the development of non-pharmacological treatment strategies.

Professor Paul Hodges PhD, MedDr, BPhty(Hons) is a Principal Research Fellow (National Health and Medical Research Council) at the University of Queensland where he heads the Human Neuroscience Unit. He has doctorates in both physiotherapy and neuroscience and his work blends neurophysiological and biomechanical methods to study the control of movement and stability of the spine and limbs and how this changes in pain. Paul was awarded the 2006 ISSLS Prize for spinal research from the International Society for the Study of the Lumbar Spine. His primary research interests include investigation the relationship between pain and motor control; the coordination of the multiple functions of the trunk muscles; pain rehabilitation; and the biomechanical mechanisms for control of the spine.