SPINE Volume 33, Number 11, pp E334–E341
©2008, Lippincott Williams & Wilkins

Differences in Standing and Forward Bending in Women With Chronic Low Back or Pelvic Girdle Pain

Indications for Physical Compensation Strategies

Jan-Paul van Wingerden, BSc, PT, Andry Vleeming, PhD, and Inge Ronchetti, MSc

Study Design. This cohort study compares motion characteristics during forward bending of a group of chronic female patients either with low back pain (LBP) or pelvic girdle pain (PGP) and healthy subjects using computer-video analysis.

Objective. This study determines whether subcategories of back pain patients could be distinguished by motion characteristics of the pelvis and lumbar spine.

Summary of Background Data. Compared with healthy subjects, patients with low back pain bend forward in distinct manners. Clustering these motion patterns into specific patient subgroups has been challenging since a basis for subcategorizing was lacking. Chronic LBP can be distinguished from PGP using specific evidence-based diagnostic tests. This allows comparing the motion characteristics of subgroups of chronic patients with either LBP or PGP.

Methods. Forward bending was recorded in both female patients groups and healthy female individuals, using a computer video analysis system. Trunk motion, pelvic tilt, and lumbar lordosis are represented as sagittal plane angles. From these angles, the relative contribution of the lumbar spine and hip joint to forward bending can be derived.

Results. Specific and discriminating motion characteristics were found between groups. During erect stance in the PGP group, the pelvis is significantly tilted backwards. At maximally forward bending, the ROM of the trunk is limited in all patient groups, but only the PGP group has significantly limited hip motion. During the initial part of forward bending, lumbar motion is increased in PGP patients and decreased in LBP patients. In the final part of forward bending contribution of the lumbar spine is increased in both patient groups.

Conclusion. LBP and PGP patients show specific, consistent, and distinct motion patterns. These motion patterns are assumed to be functional compensation strategies, following altered neuromuscular coordination.

Key words: low back pain, pelvic girdle pain, motion analysis, lumbopelvic motion, subclassification. **Spine 2008; 33:E334–E341**

From the Spine & Joint Centre, the Netherlands, Westerlaan, 3016 CK Rotterdam, The Netherlands.
Acknowledgment date: July 2, 2007. Revision date: November 13, 2007. Acceptance date: December 18, 2007.
The manuscript submitted does not contain information about medical device(s)/drug(s).
No funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.
Address correspondence and reprint requests to Jan-Paul van Wingerden, BSc, PT, Spine & Joint Centre, the Netherlands, Westerlaan 10, 3016 CK Rotterdam, The Netherlands; E-mail: wingerden@spineandjoint.nl

One of the main problems in nonspecific low back pain is the lack of adequate subcategories allowing specific fine-tuning of therapeutic interventions. Since in the past subcategorizing on a structural basis was found to be fruitless, present research predominantly aims at subcategorization based on functional analysis.[1–11] For successful functional subcategorization, both an adequate parameter and a preliminary subclassification preferably based on a "gold standard" are essential. This study aims at subcategorizing 2 back pain groups using forward bending as a discriminating tool.

In healthy subjects, forward bending consists of trunk flexion, which is flexion of the (lumbar) spine combined with pelvic tilt (hip flexion). The coordination of the lumbar spine and pelvis during this motion is not arbitrary, but specifically and consistently coupled.[12–19]

In the 1960s, Cailliet described the specific motion pattern of spine and pelvis, coined the lumbar-pelvic rhythm, similar to the scapulothoracal rhythm.[20] Compared with healthy subjects, patients with low back pain (LBP) usually bend forward in a distinct manner.[5,7,15,20–28]

Because of the assumed relation between low back pain and the specific motion patterns during forward bending, this subject has been well studied.[4,5,7,10,15,21,22,26,27,29] Most studies found differences in the motion patterns between healthy individuals and LBP patients. However, attempts to cluster motion patterns of specific subgroups of "nonspecific" patients with low back pain remains difficult especially because of the considerable variation in the motion patterns found and also the lacking of evidence-based diagnostic tests to discriminate subgroups.[1,29–31]

With respect to the latter problem, in a recent European Guideline on Pelvic Girdle Pain (PGP), a definition was constructed for pelvic musculoskeletal pain as follows:

PGP generally arises in relation to pregnancy, trauma, or reactive arthritis. Pain is experienced between the posterior iliac crest and the gluteal fold, particularly in the vicinity of the sacroiliac joints (SIJ). The pain may radiate in the posterior thigh and can also occur in conjunction with/or separately in the symphysis. The endurance capacity for standing, walking, and sitting is diminished. The diagnosis of PGP can be reached after exclusion of lumbar causes. The pain or functional disturbances in relation to PGP must be reproducible by specific clinical tests.[32]

This European PGP guideline considers, among valid tests like the Gaenslen and Patrick's Faber test, specific tests such as the Active Straight Leg Raise (ASLR), the Long Dorsal Ligament (LDL), and the Posterior Pelvic Pain Provocation (PPPP) test valuable in discriminating PGP patients from healthy subjects and patients with low back pain.[31–35] Functionally, PGP patients can be distinguished from regular LBP patients by certain motion characteristics like in walking.[3,11,36] As shown in the study by Wu *et al*, PGP patients do not only walk at lower speed, their coordination during walking is also distinct from LBP patients and healthy subjects.[11] This leads to the assumption in the present study that also the coupled motion of lumbar spine and pelvis could differ between LBP and PGP patients.

Comparison and analysis of the motion patterns of LBP and PGP patients may provide new insight in the etiology of chronic of low back and pelvic pain.

The specific patient population of a Dutch rehabilitation center specialized in the treatment of severe low back pain and pelvic girdle pain allowed to compare the motion patterns of LBP and PGP patients.

The aim of the present study was to demonstrate that consistent and discriminating motion patterns exist for the mentioned subgroups. When this assumption is proven correct, analysis of coordination may provide useful information for therapy of LBP and PGP.

■ Materials and Methods

Subjects

In a Dutch rehabilitation center, as part of the standard diagnostic procedure, motion of the lumbar spine and pelvis during forward bending was recorded using video analysis. From the general patient population, a group with specific PGP (29 women, age 33 years, SD: 5 years) was selected. The cut off scores for the inclusion criteria for PGP were raised to exclusively select severe PGP patients in this group. In the PGP group, pain was mainly experienced in the pelvic area and commenced during pregnancy or within 3 weeks after delivery. There was no history of low back pain. The Active Straight Leg Raise test (ASLR test) was positive (score summed for both legs was more than 4 on a scale of 0–10). The score of the LDL test, summed for left and right posterior superior iliac spine, was more than 2 and the PPPP test was positive.

In the group with LBP (22 women, aged 36 years, SD: 9 years), patients were selected whose pain had no relation with pregnancy; they had explicit pain in the lumbar spine but no pain in the pelvic area. The ASLR test was over all negative (summed score of both legs not more than 2 [0.9 on average for both sides]). The summed score of the LDL test (left and right posterior superior iliac spine) was less than 2 (0.4 on average for both sides), and the PPPP test was negative.

In both patient groups, complaints were present for more than 3 months. Impact of the complaints on daily life was measured using the Quebec Disability scale, experienced pain was measured with VAS scales, and the Tampa scale for kinesiophobia was used to record fear avoidance beliefs. Furthermore, as measure of physical impairment, abduction and adduction strength of the hips was measured using a hand-held dynamometer. Finally, patients were asked how long they could stand, walk, sit, or lie down before their pain significantly increased. An overview of these results is presented in Table 1.

Table 1. Overview of Severity and Impact of Complaints of LBP and PGP Group

	LBP	PGP
Quebec*	45 ± 15	61 ± 10
Pain actual	55 ± 25 mm	54 ± 24 mm
Pain minimal	32 ± 20 mm	28 ± 16 mm
Pain maximal	86 ± 15 mm	89 ± 11 mm
Tampa	33 ± 10	36 ± 7
Abduction strength*	245 ± 83 N	146 ± 74 N
Adduction strength*	176 ± 55 N	83 ± 51 N
Standing time	12 ± 13 min	10 ± 9 min
Walking time	30 ± 20 min	17 ± 14 min
Sitting time	22 ± 18 min	27 ± 16 min
Lying down time	37 ± 25 min	45 ± 20 min

Presented are: limitations in daily life (Quebec Disability Scale), experienced pain (actual, minimal, and maximal), Tampa list for kinesiophobia, measured abduction and adduction strength of the hips and duration of standing, walking, sitting or lying down before experienced pain significantly increases. Values are mean ± SD.
*Difference between LBP and PGP significant at $P < 0.001$.
mm indicates millimetres; N, newtons; min, minutes.

Both patient groups were compared with a control group of 53 healthy women (aged 25 years, SD: 9 years). In this control group, none of the women had any history of spine, pelvic, hip, knee, or ankle complaints.

Video Method

Women were instrumented with 4 markers (infrared LEDs, Figures 1–3) attached to the skin: 1 directly to the lateral side of the anterior superior iliac spine, 1 in the middle on the sacrum at the level of the posterior superior iliac spine, 1 at the level of the spinal process of the first lumbar vertebra (L1), and 1 rigidly connected to the marker on L1 (7 cm above the L1 marker).

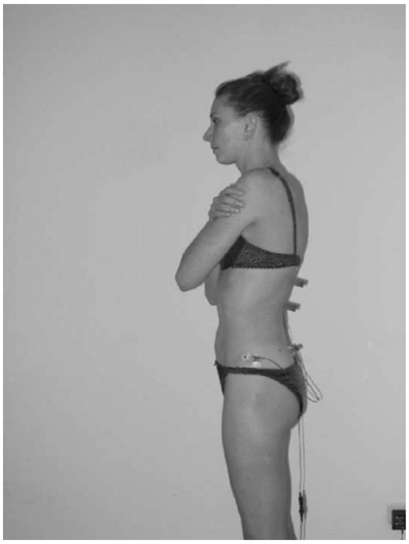

Figure 1. Upright position at the beginning of the measurement. Note the marker positions on spine and pelvis.

Figure 2. Maximally flexed posture during measurement. Note the marker positions on spine and pelvis.

Marker positions were recorded in the sagittal plane using a CCD video-camera (Javelin JE7642) equipped with a black filter. Frames were sampled at 50 Hz by a standard Personal Computer (Windows based) equipped with a customized video digitizer board (M3156b) and customized software.

Accuracy, inter- and intraobserver reliability and reproducibility of the method were extensively tested with good results (accuracy: 1°, interobserver reproducibility 0.80, internal data).

Recording

At the beginning of the recording, the subject stood upright for 1 second, with both hands on the contralateral shoulder to avoid the arm crossing the anterior pelvic marker (Figure 1). Next, subjects were asked to bend forward with straight knees as far as possible in a moderate pace without forcing or jerking and then return to the initial position (Figure 2). The motion was repeated 5 times without interruption. Minimally, 3 repetitions are used for analysis.

Analysis

The 4 pairs of coordinates obtained from each video image were converted into 3 angles in the sagittal plane (Figure 3):

α, the angle between the horizontal and the line perpendicular to the tangent of the lumbar curve at the level of L1. This angle represents the combined pelvic tilt (hip flexion) and lumbar lordosis (*trunk flexion*). α as shown in Figure 3 has a negative value.

β, the angle between the horizontal and the line through the pelvic markers, representing pelvic tilt (hip flexion).

γ, representing the lumbar lordosis was calculated by subtracting angle β from angle α as described by Gracovetsky *et al.*[14,37]

Regressions were performed on the lumbar lordosis (γ) as a function of trunk flexion (α) for the first and final one third of trunk flexion ROM. In this study, ROM was measured from

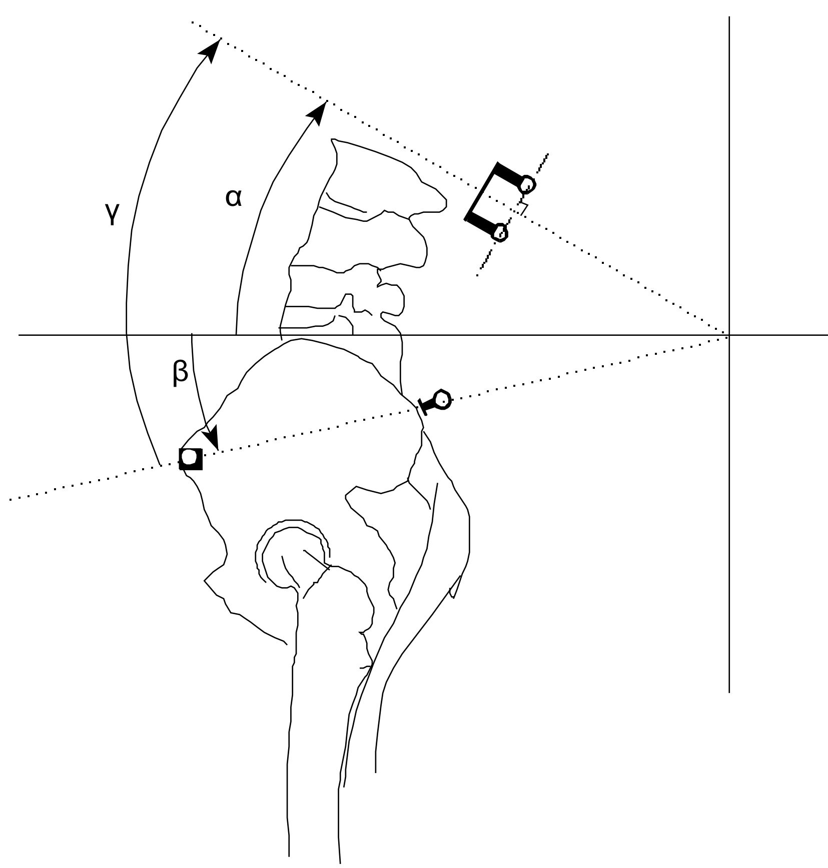

Figure 3. Outline of LEDs and calculated angels of trunk (α), pelvis (β), and lumbar spine (γ).

Table 2. Upright Position of Trunk, Pelvis and Shape of Lumbar Spine of the No Complaints Group, Low Back Pain (LBP) and Pelvic Girdle Pain (PGP) Patients

	Trunk (°)	Pelvis (°)	Lumbar Spine (°)
No complaints	−14 ± 5	11 ± 6	−25 ± 7
LBP	−13 ± 5	10 ± 5	−23 ± 6
PGP	−13 ± 5	7 ± 4*	−20 ± 6*

Values are mean ± SD.
*Compared with no complaints group difference significant at $P < 0.001$.

the upright position to maximal flexion as obtained during the video recording.

The slopes, resulting from the regression analysis, represent the relative contribution of the lumbar spine (lordosis) and pelvis to flexion. A slope of 100 reflects exclusively lumbar motion, whereas a slope of 50 indicates that 50% of the motion consists of lumbar motion and 50% of pelvic tilt.

For between group comparison, an unpaired *t* test was used. A $P \leq 0.05$ was considered significant for all tests.

■ Results

The Quebec and Pain scores as presented in Table 1 show that both patient groups are mildly to severely impaired. Although they experience equal pain, the impact on daily life is significantly higher in the PGP group. Furthermore, strength of the hips is lower in the PGP group, and walking is significantly more limited.

The data in Table 2 show that while standing upright, the position of the trunk was similar in all 3 groups (13°–14°) and pelvic tilt was similar between subjects without complaints (11°) and LBP patients (10°). In PGP patients, however, there was a significant backward tilt of the pelvis (7°) compared with both the healthy group and LBP patients. In PGP patients, lumbar lordosis was significantly flattened (20°) compared with the healthy subjects (25°), but not with LBP patients (23°) (Table 2 and Figures 3–6).

Table 3 shows that, compared with the healthy group (116°), the ROM of the trunk was significantly decreased in both LBP and PGP patients (81° and 83°, respectively). However, in the LBP group, this diminished motion is caused by a specific limitation of the lumbar motion (30°), whereas in the PGP group, not only lumbar motion is limited (47°), but also pelvic tilt (37°) is significantly limited. There is a significant difference in both pelvic tilt (51° and 37°, respectively) and lumbar motion (30° and 47°, respectively) between the LBP and PGP patients (Table 3).

Table 4 provides data on the relative contribution of the lumbar spine and pelvis to forward bending (slope 1 and slope 2). Slope 1 represents the initial one third and slope 2 represents the final one third of the forward bending motion.

For the LBP patients, the slope 1 is significantly smaller compared to healthy controls (57.7% and 66.9%, respectively), indicating that LBP patients maintain lordosis in the initial flexion. In contrast, the slope 1 of PGP patients (71.2%) is significantly increased compared with healthy controls (Table 4 and Figures 4–6). This result shows that, in contrast to both healthy subjects and LBP patients, PGP patients emphasize lumbar motion in the initial phase of forward bending.

In the final phase of forward bending, the slope 2 is significantly increased in both LBP and PGP patients compared with healthy controls, demonstrating that both patient groups have more lumbar motion in the final stage of flexion.

■ Discussion

This study investigated motion strategies in female patients with female chronic LBP and chronic PGP compared with healthy female controls. First, before the initiation of movement, PGP patients stand with especially more backward pelvic tilt but also with a slight flattened

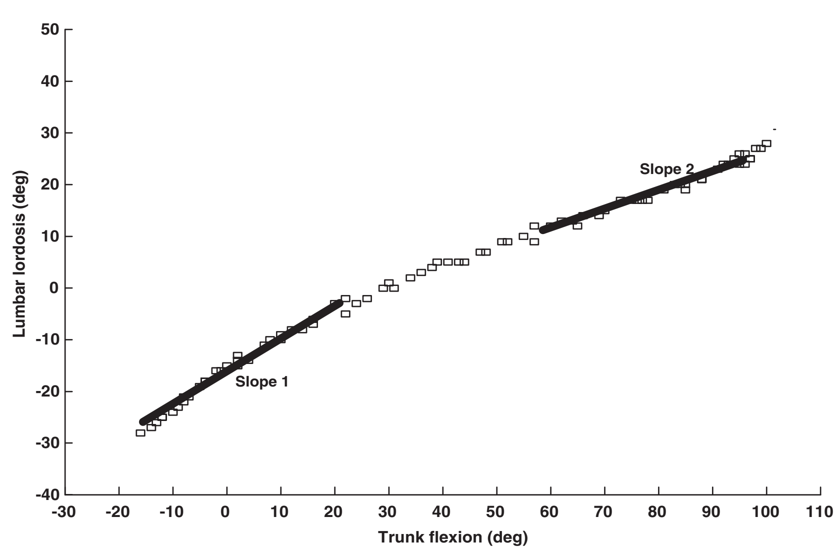

Figure 4. Example of motion pattern typical for healthy subjects, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope 1 and slope 2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion, respectively.

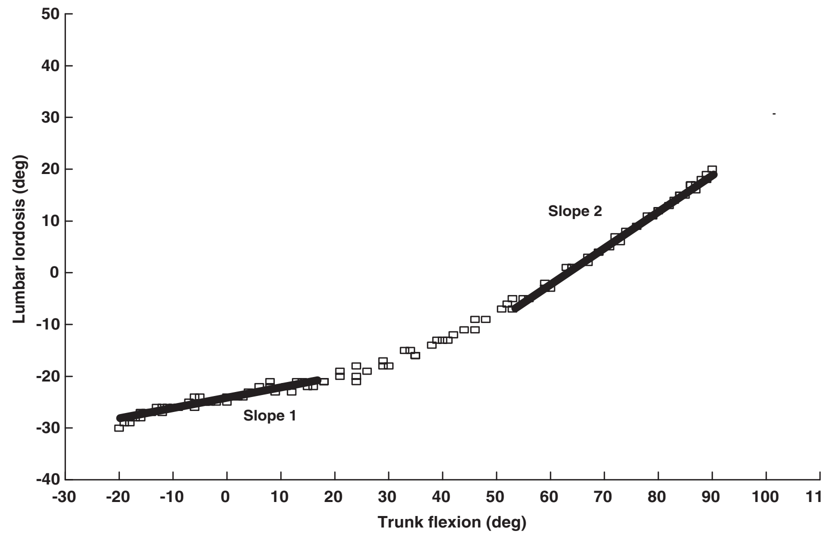

Figure 5. Example of motion pattern typical for LBP patients subjects, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope 1 and slope 2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion, respectively.

lordosis compared with both healthy subjects and LBP patients. Second, during forward bending the coupled motion of lumbar spine and pelvis during the initial phase of the motion differed significantly between both patient groups. Especially during the first one third of forward bending, LBP patients tend to maintain lordosis, whereas PGP pain patients emphasize lumbar flexion. Although the coupled motion of lumbar spine and pelvis has been well investigated, this distinct motion pattern between 2 groups of "nonspecific" back pain patients has not been reported previously. In 2 earlier studies, LBP patients could be divided in 2 subgroups: 1 with normal coupled motion of lumbar spine and pelvis and 1 with altered coupled motion[7,31]; however, no satisfactory reason for these differences was provided. In the study by Paquet *et al*, it is unclear whether male or female subjects (or both) were included,[7] and so it is likely that LPB and PGP patients were mixed. In the study by Porter *et al*,[26] a subgroup was found with reduced hip flexion (*e.g.*, limited pelvic tilt during forward bending). Although this motion pattern is similar to that found in the PGP group in this study, there are no clear indications in the Porter study that their subgroup had PGP rather than LBP. This also applies to the study by Esola *et al*, which compared the coupled motion of spine and pelvis during forward bending of 14 males and 6 females.[9] In their study, the spine/hip ratios (as a measure of relative contribution of spine and pelvis to forward bending) have large standard deviations, especially for the first part of the flexion, indicating a substantial variation in the spine/hip ratios. Such variation can occur when LBP and PGP patients, with distinct motion patterns as shown in the present study, are mixed in the same study population.

Possible Explanations Why LBP Patients Maintain Lordosis

In contrast to the motion strategies of healthy subjects, LBP patients tend to maintain lordosis during forward bending. Many authors consider this specific motion pattern as a natural protection response of the body during a back problem.[14,22,28,35,37–42] Consequently, this mo-

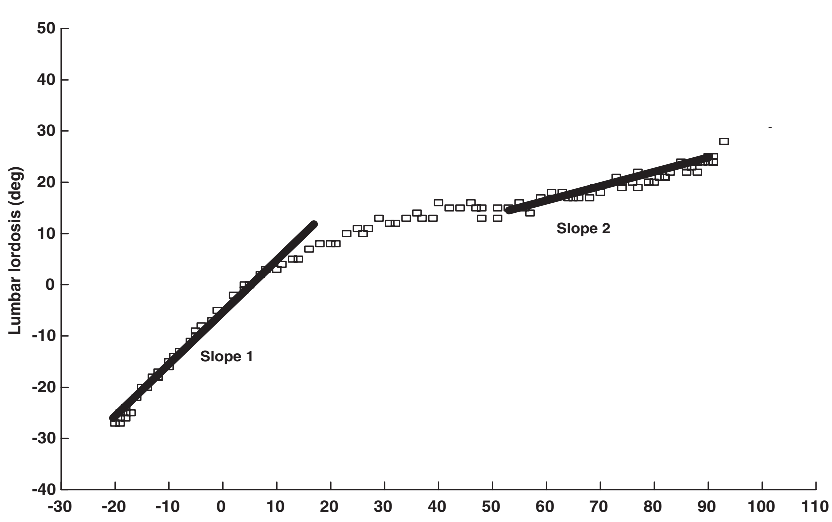

Figure 6. Example of motion pattern typical for PGP patients, with trunk flexion (α) on the x-axis and lumbar lordosis (γ) on the y-axis. Slope 1 and slope 2 represent the relative contribution of the lumbar spine (lordosis) to the first and final one third of flexion, respectively.

Table 3. Range of Motion to Flexion From Upright Position of Trunk, Pelvis and Shape of Lumbar Spine of No Complaints Group, Low Back Pain (LBP) and Pelvic Girdle Pain (PGP) Patients

	Trunk (°)	Pelvis (°)	Lumbar Spine (°)
No complaints	116 ± 14	56 ± 13	60 ± 9
LBP	81 ± 23*	51 ± 18	30 ± 16*
PGP	83 ± 28*	37 ± 19*	47 ± 14*

Values are mean ± SD.
*Compared with no complaints group difference significant at $P < 0.001$.

tion pattern is often advised to patients as the "squat" lifting technique.[43] However, it can be argued that maintaining lordosis is not a solution for a back problem, but a direct consequence of the back problem. In LBP patients, the recruitment pattern of the m. multifidus frequently changes, diminishing its anticipatory, stabilizing effect.[15,39,44,45] To guarantee stability, despite this altered activity of multifidus, other muscles (especially the m. erector spinae) become more active.[2,39] Because of its anatomic orientation, the m. erector spinae does not stabilize the lumbar spine on a segmental level, but merely increases compression, pulling the lumbar spine into lordosis. Consequently, coordination of segmental motion during forward bending is disturbed when using m. erector spinae predominantly. Therefore, it could be speculated that when m. erector spinae activity is increased to compensate for diminished m. multifidus activity this results in maintained lordosis during forward bending, as shown in the present study. Since many other factors could lead to the described patterning, this is still an incomplete analysis, which requires further study.

Possible Explanations Why PGP Patients Emphasize Lumbar Flexion

In contrast to LBP patients, PGP patients emphasize lumbar flexion in the initial phase of forward bending. Like in the LBP group, the motion pattern found could be a consequence of the specific pelvic problem.

To comprehend stability of the pelvis, a joint model of form and force closure has been introduced.[46] According to this model, several structures surrounding the SIJ can stabilize the joint by increasing joint compression.[19,34,46–49] The sacrotuberous ligament is one such structure that stabilizes the SIJ.[32,49,50] Since the sacrotuberous ligament is connected to the long head of the m. biceps femoris and the m. gluteus maximus, by increasing tension of the sacrotuberous ligament, these muscles can dynamically stabilize the SIJ.[13,19,32,48,49] Indahl *et al* showed that, in analogy to zygapophysial joints, the capsule of the SIJ plays an important role in the neuromuscular control of its stabilizing muscles.[51] When neuromuscular control of the SIJ is disturbed, compensatory means of stabilization could be addressed, such as increased activation of the biceps femoris muscle, which was elegantly explained in a study by Hungerford *et al*, but also indicated by other studies.[3,5,6,32,49,50] Because increased tension of the biceps femoris or m. gluteus maximus also resists the pelvic rotation in the hip joint this limits the contribution of the pelvis to forward bending. Consequently, lumbar motion will be emphasized, as was shown in the present study. However, other explanations for the presented phenomenon cannot be excluded.

The present study compared 2 specifically selected groups of patients with low back problems. However, it can be expected that when groups with less outspoken differences are compared, the motion patterns will be less distinct: the lumbar spine and pelvis are not separate entities but are, from a functional perspective, mutually dependent systems.[19,46,34] Low back problems and their compensatory strategies will have an impact on pelvic function, and *vice versa*. The explicit distinction between groups in this study was made for methodologic reasons but LBP and PGP can occur in mixed variations. Therefore, in a clinical setting, pelvic function should also be examined in LBP patients, and consequently, lumbar function should be analyzed in PGP patients.

In this study, no men were included because it was assumed that specific differences in motion patterns could occur between sexes. Motion patterns in men, as the differences in motion patterns between men and women, needs further study.

Table 4. Relative Contribution (RC) of Lumbar Spine to Forward Bending of No Complaints Group, Low Back Pain (LBP) and Pelvic Girdle Pain (PGP) Patients

	Slope 1 (%)	Slope 2 (%)
No complaints	66.9 ± 7.4	29.6 ± 12.0
LBP	57.7 ± 14.7*	49.3 ± 17.5*
PGP	71.2 ± 12.7*	47.0 ± 17.3*

Values are mean ± SD.
*Compared with no complaints group difference significant at $P < 0.05$.
Slope 1 = the relative contribution of the lumbar spine (lordosis) to the first one third of flexion.
Slope 2 = the relative contribution of the lumbar spine (lordosis) to the final one third of flexion.

■ Conclusion

This study shows that coupled motion of the low back and pelvis, studied in strictly classified subgroups of LBP and PGP patients, is specific and discriminating between groups. It is postulated that the specific motion patterns in patients could be functional compensation strategies of the body possibly following adjusted neuromuscular coordination.[45,51]

The distinct coupled motion of lumbar spine and pelvis, combined with more impaired walking and lower hip strength in PGP patients emphasizes the notion that LBP and PGP patients belong to distinct patients groups. Analysis of their specific compensatory patterns may elucidate how our body attempts to compensate for functional disturbances. In the clinical setting, this may enable more specific exercise programs to be developed for both LBP and PGP patients.

■ Key Points

- In chronic back pain patients, coordination of the lumbar spine and hip joints during forward bending is not arbitrary but serves a specific functional goal.
- Based on these coordination patterns, different subcategories of former nonspecific chronic back pain patients can be specified.
- Subcategorization of patients with low back pain, based on motion patterns during forward bending, allows for more specific and thus more effective therapeutic interventions.

References

1. Dankaerts W, O'Sullivan P, Burnett A. Straker L. Altered patterns of superficial trunk muscle activation during sitting in non-specific chronic low back pain patients: importance of subclassification. *Spine* 2006;31:2017–23.
2. Danneels LA, Coorevits PL, Cools AM, Vanderstraeten GG, Cambier DC, Witvrouw EE. Differences in electromyographic activity in the multifidus muscle and the iliocostalis lumborum between healthy subjects and patients with sub-acute and chronic low back pain. *Eur Spine J* 2002;11:13–9.
3. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. *Spine* 2003 28:1593–600.
4. Leinonen V, Kankaanpaa M, Airaksinen O, Hanninen O. Back and hip extensor activities during trunk flexion/extension: effects of low back pain and rehabilitation. *Arch Phys Med Rehabil* 2000;81:32–7.
5. Neblett R, Mayer TG, Gatchel RJ, Keeley J, Procter T, Anagnostis C. Quantifying the lumbar flexion-relaxation phenomenon: theory, normative data, and clinical applications. *Spine* 2003;28:1435–46.
6. O'Sullivan PB, Beales DJ, Beetham JA, et al. Altered motor control strategies in subjects with sacroiliac joint pain during the active straight-leg-raise test. *Spine* 2002;27:E1–8.
7. Paquet N, Malouin F, Richards CL. Hip-spine movement interaction and muscle activation patterns during sagittal movement in low back pain patients. *Spine* 1994;19:596–603.
8. Pirouzi S, Hides J, Richardson C, Darnell R, Toppenberg R. Low back pain patients demonstrate increased hip extensor muscle activity during standardized submaximal rotation efforts. *Spine* 2006;31:E999–E1005.
9. Pool-Goudswaard AL, Vleeming A, Stoeckart R, Snijders CJ, Mens JMA. Insufficient lumbopelvic stability: a clinical and biomechanical approach to 'specific' low back pain. *Man Ther* 1998;3:2–20.
10. Silfies SP, Squillante D, Maurer P, Westcott S, Karduna AR. Trunk muscle recruitment patterns in specific chronic low back pain populations. *Clin Biomech* 2005;20:456–73.
11. Wu W, Meijer OG, Jutte PC, et al. Gait in patients with pregnancy-related pain in the pelvis: an emphasis on the coordination of transverse pelvic and thoracic rotations. *Clin Biomech* 2002;17:678–86.
12. Adams MA, Dolan P, Marx C, Hutton WC. An electronic inclinometer for measuring lumbar curvature. *Clin Biomech* 1986;1:130–34.
13. Gajdosik RL, Hatcher CK, Whitsell S. Influence of short hamstring muscles on the pelvis and lumbar spine in standing and during the toe-touch test. *Clin Biomech* 1992;7:38–42.
14. Gracovetsky S, Kary M, Levy S, Ben Said R, Pitchen I, Helie J. Analysis of spinal and muscular activity during flexion/extension and free lifts. *Spine* 1990;15:1333–9.
15. Sihvonen T, Partanen J, Häninnen O, Soimakallio S. Electric behaviour of low back muscles during lumbar pelvic rhythm in low back pain patients and healthy controls. *Arch Phys Med Rehabil* 1991;72:1080–7.
16. Stokes IA, Abery JM. Influence of the hamstring muscles on lumbar spine curvature in sitting. *Spine* 1980;6:525–8.
17. Toppenberg R, Bullock M. The interrelation of spinal curves, pelvic tilt and muscle length in the adolescent female. *Aust J Physiother* 1986;32:6–12.
18. Tully EA, Wagh P, Galea MP. Lumbofemoral rhythm during hip flexion in young adults and children. *Spine* 2002;27:432–40.
19. van Wingerden JP, Vleeming A, Stam HJ, Stoeckart R. Interaction of spine and legs: influence of hamstring tension on lumbo-pelvic rhythm. Proceedings of second interdisciplinary world congress on low back pain, San Diego, 1995.
20. Cailliet R. *Low Back Pain Syndrome*. 2nd ed. Philadelphia: F.A. Davis Company, 1968;23–6.
21. Esola MA, McClure PW, Fitzgerald GK, Siegler S. Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. *Spine* 1996;21:71–8.
22. Kaigle AM, Wessberg P, Hansson TH. Muscular and kinematic behaviour of the lumbar spine during flexion-extension. *J Spinal Disord* 1998;11:163–74.
23. Mayer TG, Tencer AF, Kristoferson S, Mooney V. Use of non-invasive techniques for quantification of spinal range-of-motion in normal subjects and chronic low-back dysfunction patients. *Spine* 1984;9:588–95.
24. Mellin G. Correlations of hip mobility with degree of back pain and lumbar spinal mobility in chronic low-back pain patients. *Spine* 1988;13:668–70.
25. Nelson JM, Walmsley RP, Stevenson JM. Relative lumbar and pelvic motion during loaded spinal flexion/exension. *Spine* 1995;20:199–204.
26. Porter JL, Wilkinson A. Lumbar-hip flexion motion. A comparative study between asymptomatic and chronic low back pain in 18- to 36-year-old men. *Spine* 1997;22:1508–13.
27. Shirado O, Ito T, Kaneda K, Strax TE. Flexion-relaxation in the back muscles. A comparative study between healthy subjects and patients with chronic low back pain. *Am J Phys Med Rehabil* 1995;74:139–44.
28. Toussaint HM, de Winter AF, de Haas Y, de Looze MP, van Dieen JH, Kingma I. Flexion-relaxation during lifting: implications for torque production by muscle activity and tissue strain at the lumbo-sacral joint. *J Biomech* 1995;28:199–210.
29. McClure PW, Esola M, Schreier R, Siegler S. Kinematic analysis of lumbar and hip motion while rising from a forward, flexed position in patients with and without a history of low back pain. *Spine* 1997;22:552–8.
30. Östgaard HC, Andersson GBJ, Karlsson K. Prevalence of back pain in pregnancy. *Spine* 1991;16:549–52.
31. Östgaard HC, Zetherstrom GBJ, Roos-Hansson E. The posterior pelvic pain provocation test in pregnant woman. *Eur Spine J* 1994;3:258–60.
32. Vleeming A, Albert H, Östgaard HC, Stuge B, Sturesson B. COST B14, Working Group 4 on Pelvic Girdle. European guidelines for the diagnosis and treatment of pelvic girdle pain. *Eur Spine J* 2008 [Epub ahead of print].
33. Mens JMA. Pregnancy Related Low Back Pain. PhD thesis, Erasmus University, Rotterdam, the Netherlands, 2000.
34. Vleeming A, Pool-Goudzwaard AL, Hammudoghlu D, Stoeckart R, Snijders CJ, Mens JM. The function of the long dorsal sacroiliac ligament: its implication for understanding low back pain. *Spine* 1996;21:556–62.
35. Vleeming A. de Vries HJ, Mens JMA, van Wingerden JP. Possible role of the long dorsal sacroiliac ligament in women with peripartum pelvic pain. *Acta Obstet Gynecol Scand* 2002;81:430–6.
36. Lamoth CJ, Meijer OG, Daffertshofer A, Wuisman PI, BeekPJ. Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. *Eur Spine J* 2006;15:23–40.
37. Gracovetsky S, Kary M, Pitchen I, Levy S, Ben Said R. The importance of pelvic tilt in reducing compressive stress in the spine during flexion-extension exercises. *Spine* 1989;14:412–6.
38. Adams MA, Bogduk N, Burton K, Dolan P. *The Biomechanics of Back Pain*. Churchill Livingston, Edinburgh, 2002;95–105, Ch 7.
39. van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. *Spine* 2003;28:834–41.
40. Dolan P, Mannion AF, Adams MA. Passive tissues help the back muscles to generate extensor moments during lifting. *J Biomech* 1994;27:1077–85.
41. Macintosh JE, Bogduk N, Gracovetsky S. The biomechanics of the thoracolumbar fascia. *Clin Biomech* 1987;2:78–83.
42. McGill SM, Kippers V. Transfer of loads between lumbar tissues during the flexion-relaxation phenomenon. *Spine* 1994;19:2190–6.
43. van Dieen JH, Hoozemans MJ, Toussaint HM. Stoop or squat: a review of biomechanical studies on lifting technique. *Clin Biomech* 1999;14:685–96.
44. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. *Spine* 1996;21:2763–9.
45. Indahl A, Kaigle AM, Reikeras O, Holm SH. Interaction between the porcine lumbar intervertebral disc, zygapophysial joints, and paraspinal muscles. *Spine* 1997;22:2834–40.
46. Snijders CJ, Vleeming A, Stoeckart R. Transfer of lumbosacral load to iliac bones and legs. Part 1 and 2. *Clin Biomech* 1993;8:285–94.
47. Vleeming A, Pool-Goudzwaard AL, Stoeckart R, Wingerden JP van, Snijders CJ. The posterior layer of the thoracolumbar fascia; its function in load transfer from spine to legs. *Spine* 1995;20:753–8.
48. van Wingerden JP, Vleeming A, Snijders CJ, Stoeckart R. A functional-

anatomical approach to the spine-pelvis mechanism: interaction between the biceps femoris muscle and the sacrotuberous ligament. *Eur Spine J* 1993;2: 140–4.

49. van Wingerden JP, Vleeming A, Buyruk HM, Raissadat K. Stabilization of the sacroiliac joint in vivo: verification of muscular contribution to force closure of the pelvis. *Eur Spine J* 2004;13:199–205.
50. Vleeming A, Stoeckart R, Snijders CJ. The sacrotuberous ligament: a conceptual approach to its dynamic role in stabilizing the sacroiliac joint. *Clin Biomech* 1989;4:201–3.
51. Indahl A, Kaigle A, Reikeras O, Holm S. Sacroiliac joint involvement in activation of the porcine spinal and gluteal musculature. *J Spinal Disord* 1999;12:325–30.