

This is the accepted version of the following article: Pesesse, Pierre, Sebastien Wolfs, David Colman, Stephanie Grosdent, Marc Vanderthommen, Christophe Demoulin. « Straight Leg Raise versus Knee Extension Angle: Which Structure Limits the Test in Asymptomatic Subjects? » *Journal of Manual & Manipulative Therapy*, 24 février 2025, 1-9.
<https://doi.org/10.1080/10669817.2025.2465739> which has been published in final form at:
<https://www.tandfonline.com/doi/full/10.1080/10669817.2025.2465739>

Straight Leg Raise versus Knee Extension Angle: which structure limits the test in asymptomatic subjects?

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Clinical trial number: NCT05899244

Biographical note:

Pierre Pesesse graduated as a physiotherapist in 2013 from the University of Liege (Belgium). He obtained a certificate in manual therapy in 2018 (IFOMPT) from the University of Liège, where he works as an assistant and a PhD student. The aim of his thesis is to contribute to the development of knowledge regarding the effectiveness of neurodynamic techniques and manual therapy in the management of lumbosacral radicular pain. Additionally, he is a lecturer in the orthopaedic manual therapy certificate program of the University of Liège.

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David Colman graduated as a physiotherapist in 2007 from the University of Liège. He began training in manual therapy in 2011 (IMTA, level 2b) and obtained a certificate in manual therapy (IFOMPT) in 2014 in Liège. After 10 years of full-time clinical practice in the musculoskeletal field (including the Back Clinic Department at CHUOA, Liège), he was hired as an assistant and PhD student in the Department of Sport Sciences and Rehabilitation at ULiège. The aim of his thesis is to contribute to improving knowledge on the evaluation and rehabilitation of cervical extensor muscle dysfunction. He is also a lecturer in orthopaedic manual therapy at the University of Liège and UCLouvain.

Stéphanie Grosdent holds a degree in physiotherapy and a PhD from the University of Liège. Her thesis focused on the assessment and rehabilitation of athletes with non-specific low back pain. She is currently working as an assistant professor at the University of Liège and also works part-time as a manual therapist at the Spine Center of the University Hospital Center of Liège.

Marc Vanderthommen graduated as a physiotherapist from the University of Liège in 1987 and defended his doctoral thesis in 1994. After finishing a post-doc in Paris (France), he was appointed Professor in the Department of Physical Activity and Rehabilitation Sciences of the Faculty of Medicine (ULiège) in 2002. He completed an IFOMPT-recognized course in manual therapy in 2015. In addition to teaching and research activities (H-index of 15), focusing on neuromuscular electrical stimulation, adapted sports and spinal pain, dysfunction and rehabilitation, he continued to practise as a clinician in the multidisciplinary program offered by the Spine Clinic of the Liège University Hospital Centre until 2020.

Christophe Demoulin graduated as a physiotherapist from the University of Liège in 2002 and defended his doctoral thesis on the evaluation and rehabilitation of people with chronic low back pain in 2008. After a post-doc in Maastricht (Netherlands), he completed an IFOMPT-recognised manual therapy course. He has been a guest lecturer at the Faculty of Motor Sciences of UCLouvain since 2015 and was appointed Professor within the Department of Physical Activity and Rehabilitation Sciences of the Faculty of Medicine (ULiège) in 2021. In addition to teaching and research activities (H-index of 20), focusing on spinal pain and dysfunction, chronic pain, manual therapy, clinical reasoning, communication, and the evidence-based practice approach, he works as a clinician in the multidisciplinary program offered by the Spine Clinic of the Liège University Hospital Centre. He is also a member of the Editorial Committee of the journals JOSPT and JMMT, the President of the Belgian French-speaking Scientific Society of Physiotherapy (SSFK) and has been President of the Belgian Back Society (BBS) since 2021.

Abstract

Objectives: This study aimed to determine if the first onset of symptoms (discomfort) during the straight leg raise (SLR) (hip flexion with an extended knee) and the Knee Extension Angle (KEA) tests (knee extension with 90° of hip flexion) results from nervous or muscular structures in asymptomatic individuals. The secondary objective was to investigate if the gender influences the structure related to the discomfort.

Methods: This cross-sectional study consisted of a single assessment session during which the structure related to participants' discomfort during the KEA and SLR was identified. For this identification, a structural differentiation (SD) was conducted during both tests using passive mobilisation of the cervicothoracic spine in flexion and extension. Changes in participants' discomfort were monitored during the SD to determine whether a change or lack of change was consistent with variations in the load applied to the suspected structures either muscular or neural. If the structure related to the participants' discomfort could not be identified, two additional tests were conducted: the lateral SLR and the Slump test.

Results: One hundred and seventy-eight individuals were included. Median [IQR] age was 21 years [20;23], and 57.3% were female. The structure related to participants' discomfort was similar for the SLR and the KEA ($p=0.451$): neural for 72.5 % of participants in the SLR and 75.8% in the KEA. Gender only influenced the structure identified in the KEA test, with a significantly higher rate of nerve-related discomfort in females than males and a significantly higher rate of muscle-related discomfort in males ($p=0.002$).

Conclusion: In asymptomatic individuals, the discomfort induced by the SLR and the KEA tests could be related to either muscular or neural structures. Therefore, structural differentiation is necessary to identify the structure causing the discomfort in both research and clinical practice.

Keywords: "Straight leg raise test", "Knee extension angle test", "Structural differentiation", "Mechanosensitivity", "Hamstring muscles", "Range of motion, Articular"

Introduction

A common finding associated with musculoskeletal disorders is reduced passive range of motion (ROM) of the lower limbs (1). This reduction can also be present in individuals without musculoskeletal disorders. Reduced ROM might result from various anatomical structures such as ligaments, joint capsules, bones, muscles (e.g., hamstring, soleus, gastrocnemius) and neural structures (e.g., sciatic or femoral nerve, nerve roots and the associated nerve bed). However, accurately identifying the structure responsible for the ROM limitation is challenging.

Studies evaluating the lower limb ROM used the straight leg raise test (SLR) or the knee extension angle test (KEA), also referred to as the passive knee extension test (2–4). However, these tests were used and interpreted as they were first described in the 1980s without questioning their validity in the light of recent findings. The SLR test consists of passively raising the leg with the knee extended and the person in supine. During the 1980s and 1990s, several studies evaluated the validity of the SLR test for the assessment of hamstring extensibility in asymptomatic individuals (5–8). Some studies reported that the test induced considerable pelvic rotation (7,8). The authors also stated that the limitation during the SLR could be related to neural structures as the test had been described for the assessment of people with lumbosacral nerve root involvement (9) because it generates movement of neural structures (10).

To reduce the amount of pelvic rotation during the assessment of hamstring extensibility, the KEA test and some variations were developed (11–13). The KEA test involves passive knee extension while maintaining the hip at 90° of flexion, with the person in supine. Since the KEA induces considerably less pelvic rotation than the SLR (mean pelvic rotation 4.1° - 5.0° vs 24.9°) (7,11,14), several authors have concluded that it is the most valid test to assess hamstring extensibility (12,14). These findings have also been used as indirect evidence that the hamstring is the most probable limiting factor during this test, a hypothesis that many researchers accept. Although reducing pelvic rotation increases the reliability of ROM assessment, it does not define the structure causing the limitation or discomfort during the test.

This statement is supported by a recent study using in vivo ultrasound imaging, which reported that hip flexion induces a longitudinal movement of the sciatic nerve that is significantly higher with the knee in flexion than with the knee in extension (11.7 ± 4.8 vs 7.0 ± 3.8 mm) (15). Furthermore, mobilising the knee in extension induces a similar longitudinal excursion of the sciatic nerve when comparing mobilisation starting from hip in neutral position to hip in flexion (6.9 ± 2.5 vs 8.8 ± 3.5 mm) (15). Therefore, these findings suggest that both the KEA and SLR could induce movement and tension of neural structures and cause nerve-related sensations that limit the test ROM.

Symptoms can also arise from non-neural tissues (i.e., muscle tissues) during the SLR and KEA tests (16); therefore, it is necessary to identify the structure that limits the ROM and causes the symptoms. A recent systematic review of the SLR concluded that a process of structural differentiation (SD) should be used to identify the structure related to the person's symptoms (17). This process consists of analysing if the person's symptoms change (increase or decrease) when passively mobilising a joint remote from the symptom location in a way that loads or unloads the nervous system without modifying the load applied on other structures that might also be responsible for the symptoms (17,18). If the movement loading and unloading the nervous system consistently modifies the symptoms, a neural origin of the symptoms and/or ROM limitation is likely.

To our knowledge, none of the previous studies using SLR and KEA tests have properly used SD to identify the structure related to participants' discomfort during these tests.

Therefore, the primary aim of this study was to use SD to determine if the discomfort induced during the SLR and KEA tests is related to neural or muscular structures in asymptomatic individuals. The secondary aim was to determine the influence of gender on the structure related to the discomfort.

Methods

Study design and setting

This cross-sectional study was conducted in the Department of Physical Activity and Rehabilitation Sciences of the University of Liege (Belgium). This article reports the primary results of a larger project, which aims to determine the influence of muscle stretching and neural mobilisations on lower limb range of motion in asymptomatic individuals. The study was approved by the Liege University Hospital Human Ethics Committee (Nr: EudraCT: B7072021000077) and registered at clinicaltrials.gov (NCT05899244).

Participants:

Asymptomatic people aged between 18 and 65 years were invited to participate. A non-probabilistic recruitment method was used: participants were recruited using convenience sampling via mailing lists and announcements posted on social networks (Facebook and Instagram). Recruitment was conducted from June 2023 to October 2023.

The exclusion criteria included any of the following: 1) low back pain requiring medical care during the last 6 months; 2) a peripheral or central neurological condition such as lumbosacral radicular pain, multiple sclerosis, stroke, herpes zoster, polyradiculoneuropathy or meningitis; 3) diabetes mellitus; 4) present or past muscle injury of the lower limbs (e.g. tendinopathy or muscle tear); and 5) history of lower limb surgery of the tested leg. Furthermore, participants who exhibited full knee extension without any discomfort during the KEA at the assessment session were excluded. All participants were informed of the objective of the project and took part in the study after providing informed consent.

Procedures:

Assessment session

Three investigators were involved in the evaluation process. Investigator 1 was a qualified physiotherapist (IFOMPT manual therapist). The two other investigators were final-year physiotherapy students.

After checking exclusion criteria, demographic data were collected (age, gender, height and weight). Then, the SLR and KEA tests were conducted in a randomised order across participants.

To standardise the assessments, participants were positioned supine with the cervical spine in a neutral position, the upper part of the head at the edge of the examination table, and both hands placed under the lumbar spine to prevent lumbar flexion. The examination table used during the assessment (Enraf-Nonius, Rotterdam, Netherlands) had a moveable headset allowing mobilisation of the thoracic spine in flexion/extension. A belt over the anterior superior iliac spines fixed the pelvis to the examination table to avoid any pelvic movement.

Description of the two main tests

SLR: Investigator 1 passively flexed the hip while maintaining the knee in extension, with one hand placed on the distal anterior part of the thigh and the other on the posterior distal part of the leg (19) (**Figure 1**).

KEA: Investigator 1 placed the participant's leg in the starting position, i.e., 90° hip flexion (verified using a gravity inclinometer placed on the anterior part of the thigh) with the knee flexed. He then passively extended the knee (**Figure2**).

In both tests, participants were asked to say "stop" when they felt any discomfort in the leg and to tell the investigator where the sensation was located (i.e., at the posterior part of the buttock, thigh, knee, calf or foot). Investigator 2 measured the hip ROM for the SLR and the

knee ROM for the KEA when the participant said “stop”. This measurement was done with an electronic inclinometer (DXL306S, resolution 0.01°, WIWU 2013) positioned over the proximal part of the tibial tuberosity, used as a reference for the device placement. For both tests, a structural differentiation process was then conducted.

Structural differentiation (SD):

SD was performed according to the participant’s response to the previous question. While investigator 1 maintained the position of the leg (during SLR or KEA) at which the discomfort was felt, two additional steps were conducted:

Step 1: Investigator 3 passively flexed the thoracic spine using the movable cranial part of the table (45° of movement), then manually flexed the cervical spine until full ROM was reached. Full ROM was defined as the point at which the investigator perceived strong resistance at the end of the potentially available movement. If the participant reported an intolerable increase in lower limb discomfort, the cervical flexion was stopped. The participant was then asked to report any change in the tested limb discomfort using the following predefined descriptors: “increase”, “decrease” or “unchanged”.

Before step 2, the participant was re-positioned in the neutral cervicothoracic position and asked to report if the discomfort was still present and if it was the same as before step 1. If the discomfort had disappeared or decreased, the ROM of the tested lower limb was slightly increased until participant felt the same discomfort as at baseline.

Step 2: Investigator 3 passively extended the thoracic spine using the movable part of the table (25° of movement), and manually extended the cervical spine until full ROM was reached. The participant was then asked to report any changes in the lower limb discomfort induced by the test using the descriptors mentioned above.

As indicated in **Figure 3**, if neither step of the SD changed the lower limb discomfort, a “muscular origin of the discomfort” was suspected (and the participant was categorised in the “muscle discomfort” category). A “neural origin of the discomfort” was suspected (“nerve discomfort” category) if the discomfort increased during step 1 and decreased during step 2. If a different combination was observed (inconsistent observations between steps 1 and 2), an additional test was performed.

Additional tests:

If the origin of the discomfort could not be determined during the SLR or the KEA tests, additional tests were performed, the lateral SLR test (after the SLR) or the Slump test (after the KEA) (**Figure 3**).

To interpret the outcome of the additional test, the discomfort felt in the lower limb had to be exactly the same (in terms of location, quality and intensity) as the discomfort reported during

the main test (KEA or SLR). If this was not the case, the participant was excluded. Again, participants were asked to say “stop” at the onset of discomfort.

Lateral SLR test: Participants were placed in side-lying position with the tested limb on the upper side and the cervical, thoracic and lumbar spine in full flexion. As previously described, full ROM was defined as the point at which the investigator perceived strong resistance at the end of the potentially available movement. Investigator 2 maintained the thoracic and lumbar flexion, and investigator 3 maintained the cervical spine flexion. Investigator 1 then flexed the hip while maintaining the knee fully extended.

The slump test: Participants were seated in a slumped posture (cervical, thoracic and lumbar spine in full flexion) with the popliteal fossae against the edge of the table. The vertical position of the sacrum was checked by investigator 2 using an electronic goniometer. Investigator 3 stabilised the thoracic, lumbar and sacral positions. Investigator 1 stabilised the cervical spine flexion with his elbow over the cervicothoracic junction and his hand on the participant’s head and then extended the knee.

Structural differentiation (SD):

SD was performed according to the participant’s response to the previous question. While investigator 1 maintained the position of the lower limb (during lateral SLR or slump) at which the discomfort was felt, two additional steps were conducted. Investigator 2 manually and passively induced a complete extension (step 1) and then flexion (step 2) of the cervical spine. The participant was then asked to report any change in the tested limb discomfort using the previously described descriptors.

If neither of the SD steps changed the lower limb discomfort, a “muscular origin of the discomfort” was suspected (and the participant was categorised in the “muscle discomfort” category). A “neural origin of the discomfort” was suspected if the discomfort decreased during step 1 and increased during step 2 (“nerve discomfort” category) (**Figure 3**). If the responses to both steps were inconsistent, the participant was excluded from the study as the origin of the discomfort could not be identified.

Statistical analysis

Statistical analysis was performed using R (Version 4.0.3, R foundation). For quantitative variables, the normality of the distribution was assessed using histograms, quantile-quantile plots and the Shapiro-Wilk test. Since none of the quantitative variables were normally distributed, they are described by the median and interquartile range, except for ROM measured during the SLR and KEA, which was normally distributed and is expressed as the mean and standard deviation. Median age and BMI values were compared between the female and male groups using the Kruskal-Wallis test. The McNemar test compared the proportion of neural and muscular structures related to the discomfort during the SLR and KEA tests in the

whole sample. A Pearson's chi-squared test compared the proportion of neural and muscular structures related to the discomfort between the male and female groups.

Results

Participants

In total, 184 people were recruited, and 178 were included in the analyses (**Figure 4**). Only three participants were excluded because the origin of the discomfort could not be identified at one test (KEA or SLR) after all the tests. The median [IQR] age of the included participants was 21 [20;23] years, body mass index (BMI) was 22.0 [20.7;23.9], and 57.3% (n=102) were female (**Table 1**). Mean \pm SD SLR ROM was $63.9^\circ \pm 14.1^\circ$ and mean KEA ROM was $48.3^\circ \pm 20.2^\circ$.

Suspected structural limitation

The suspected origin of the discomfort (first onset of symptoms) was neural in 72.5% (n=129) of participants with the SLR and 75.8% (n=135) with the KEA (**Table 2**). These proportions did not differ significantly ($p=0.45$).

Among the participants, 61.8% presented a suspected neural origin of the discomfort and 13.5 % presented a muscular origin of the discomfort at **both** SLR and KEA tests (**Table 3**). In contrast, 24.7% of the participants had a different discomfort origin at the KEA and SLR (mixed limitation).

The results of the two-step process of SD for both SLR and KEA are presented in **Table 4**. To identify the structure related to the discomfort, additional tests had to be performed in 61.2% of participants for the SLR and 53.9% for the KEA.

Influence of gender on the suspected limitation

The comparison of the demographic characteristics of the male and female groups only found a significant difference for BMI (higher in the male group; $p=0.002$) (**Table 1**).

The proportion of each suspected structure related to participants' discomfort during the SLR test (i.e., neural origin for most participants) did not differ between genders ($p=0.08$). For the KEA, the percentage of participants with a neural origin of the discomfort was significantly higher in the female group, and the percentage with a muscular origin was higher in the male group ($p=0.002$).

Discussion

The results of this study suggest that the origin of the discomfort in the SLR and KEA tests in asymptomatic individuals is mostly neural (72.5-75.8%). Importantly, in contrast with common assumptions, the SLR does not only evaluate neural structures mechanosensitivity, and the KEA is not only limited by hamstring stiffness. These findings highlight that structural differentiation is necessary to identify the structure related to participants' discomfort when using these tests.

Our findings are partially supported by those of Lopez-de Celis et al. (20) who reported a neural origin of the discomfort in 75.8% of dominant limbs and 72.7% of non-dominant limbs when using the SLR associated with SD in healthy participants. The trend of results for the KEA was similar in that study and the present study, with most discomfort being related to neural structures. However, we found a much higher proportion of neural origin of the discomfort in comparison with the study of Lopez-de-Celis et al. (75.8% vs 57.6%) (20). This difference might be explained by the less valid methodology they used for the SD. The SD was performed using passive ankle dorsiflexion during the SD for all the participants for both the KEA and SLR tests, despite the fact that some participants reported symptoms located in the posterior part of the calf or at the popliteal fossa (20). As mentioned before, during SD the therapist try to modify the load on neural structures without modifying the load on other structures that could be responsible for patients' symptoms (18,21). Therefore, to properly conduct the SD, the joint mobilised should be remote from the pain location. Pesonen et al. suggested using medial hip rotation for the SD when a subject presents a pain located below the popliteal fossa (22). Passive ankle dorsiflexion is recommended when the pain is located in the gluteal region or above the popliteal fossa (22,23). In other words, these authors advocate that the joint used for the SD should at least be separate by another joint from the symptom location. Therefore, SD using the ankle should not be performed when the symptoms are located in the posterior part of the calf or the popliteal fossa, which are frequent locations of participants' symptoms during the SLR and KEA test in asymptomatic individuals (24).

The results of this study brings meaningful considerations for clinical practice and for research in the domain. They indicate that a single test is insufficient to identify the type of limitation of the participants. To the best of our knowledge, none of the studies analysing the effectiveness of muscle stretching techniques or neural mobilisations on lower limb ROM have properly identified the limitation of their participants. Therefore, the data in the literature may not be meaningful or valid for the selection of the most effective technique for improving ROM for a given limitation. Therefore, we recommend that future studies in this domain properly identify the type of limitation or the structure related to the symptoms, using at least a SD.

No difference was found between males and females for the origin of the discomfort for the SLR. However, for the KEA, the proportion of females with a neural origin of the discomfort was higher than that of males (84.3% vs 64.5%; $p=0.002$). Although our results are in accordance with those of Lopez-de-Celis et al. for the SLR test (20), they differ regarding the KEA test since

that study reported no differences between males and females. As mentioned above, the SD method used in that study might explain this partial discordance.

During this study, additional tests were conducted in 53.9% of participants for the KEA and 61.2% for the SLR because the modification or the absence of modification of participant discomfort during SD was uninterpretable. Our SD process was based on previous findings showing that cervicothoracic spine flexion and extension respectively increase and decrease tension on the nervous system (25,26) and are expected to increase and decrease symptoms accordingly (27,28). Surprisingly, some participants only reported a modification of their discomfort during one of the steps of the SD, and others reported a contradictory response (decreased discomfort during cervicothoracic spine flexion and inversely during extension). Participants might not have been fully relaxed (antagonist muscle activation) during the passive cervicothoracic movement, preventing maximal flexion or extension, which could explain the partial modification of discomfort with SD. Another explanation could be that, despite our efforts to stabilise the lumbopelvic region, participants may have activated their rectus abdominis during the passive thoracic flexion or extension, inducing slight pelvic retroversion, which could reduce the load on the hamstring muscles.

The contradictory responses observed in some participants could, at first glance, be considered unintuitive. However, it can be explained by the fact that nervous structures can also be sensitive to mechanical constraints other than tension. Historically, nervous responses during mechanosensitivity tests are linked to tension constraints; however, a reduction of the sliding capacity of the neural structures could also cause discomfort during these tests. Kobayashi et al., have shown in people suffering from lumbosacral radicular pain, that the angle at which people experienced pain during the SLR were related to a reduction of the neural structure sliding capacity, measured during surgical intervention, caused by adhesive fibrosis between the nerve roots and a herniated disc. The lack of sliding is also associated with reduced blood flow (29,30). Furthermore, still in people suffering from lumbosacral radicular pain, Pesonen et al., have showed using MRI imaging a strong correlation between a reduction of the sliding of the conus medularis and patient pain intensity (31). Moreover, they found that restoration of the conus medularis sliding capacity was correlated with patient recovery (31).

These findings could be extrapolated to asymptomatic individuals in which physiological structures (such as the intraforaminal ligament (32), meningovertebral ligament (33) or denticulate ligament (34)) or asymptomatic structural modifications (disc bulging, foraminal or spinal canal stenosis) could reduce neural distal sliding and consequently the blood flow. Therefore, in participants with contradictory responses to SD, cranial movement of the neural structures could reduce discomfort through the reduction of the tension on physiological structure or by displacing neural structures away from structural modifications, potentially improving blood perfusion and therefore reducing the discomfort.

Limitations

Despite the strengths of our study (use of a SD in a large sample, main investigator being a physiotherapist with 10 years of experience), it has several limitations. The study sample was relatively young (median age of 21 years); therefore, the results might not reflect asymptomatic people of all ages. The criterion used to stop the test was participant's discomfort, which is a subjective parameter. The results might have been different if the criterion had been pain reported by the participant. As our sample consisted of asymptomatic participants, the results cannot be extrapolated to people with radicular pain. Although involving 3 investigators to conduct the tests was necessary to limit compensatory movements as much as possible and to be able to conduct a proper SD, such a methodology is not applicable in clinical practice.

Finally, the specific action of the SD on neural structures has been challenged by authors who suggested that the continuity of the fascial system (from the thoraco-lumbar fascia to the biceps femoralis muscle) could explain the modification of discomfort during flexion and extension of the cervicothoracic spine (35). However, two studies showed that neither cervical nor thoracic movement modifies the load on the proximal tendon of the hamstrings or on the soleus during the slump test (16,36), reducing the likelihood of fascia involvement in discomfort modification during SD.

Conclusion

The results of this study show that in most young, asymptomatic individuals, the discomfort during the SLR and KEA tests was induced by neural structures. These results underscore the importance of structural differentiation to identify the structure related to the discomfort.

Disclosure statement:

The authors report there are no competing interests to declare.

Funding details:

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All the authors have approved the version of the paper to be published and agree to be accountable for all aspects of the work.

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Tables

	n	BMI	Age (Years)
Males	76	22,5 [21,5;24,5]	21 [19;23]
Females	102	21,7 [20,1;23,2]	21 [20;23]
Total	178	22.0 [20.7;23.9]	21 [20;23]

Table 1. Demographic characteristics of the participants (median values and interquartile ranges [IQ 25;75]). BMI: body mass index.

	SLR			KEA		
	Males	Females	Total	Males	Females	Total
Suspected nervous origin (%)	65.8 (n=50)	77.5 (n=79)	72.5 (n=129)	64.5 (n=49)	84.3 (n=86)	75.8 (n=135)
Suspected muscular origin (%)	34.2 (n=26)	22.5 (n=23)	27.5 (n=49)	35.5 (n=27)	15.7 (n=16)	24.2 (n=43)

Table 2. Proportion of participants with discomfort of neural and muscular origin during the straight leg raise (SLR) and the knee extension angle (KEA) tests. (Proportions are expressed as percentages by column)

	Males	Females	Total
Nervous related discomfort for KEA and SLR (%)	48.7 (n=37)	71.6 (n=73)	61.8 (n=110)
Discomfort related structure ≠ for KEA and SLR (%)	32.9 (n=25)	18.6 (n=19)	24.7 (n=44)
Muscle related discomfort for KEA and SLR (%)	18.4 (n=14)	9.8 (n=10)	13.5 (n=24)

Table 3. Origin of the discomfort for the knee extension angle (KEA) and straight leg raise (SLR) tests (n=178). (Proportions are expressed as percentages by column)

	SLR		KEA	
	Structural differentiation using cervicothoracic spine passive mobilization (n=178)			
	Flexion	Extension	Flexion	Extension
Increased discomfort (%)	51.7 (n=92)	24.2 (n=43)	48.3 (n=86)	17.4 (n=31)
Decreased discomfort (%)	28.0 (n=50)	43.8 (n=78)	23.6 (n=42)	50.6 (n=90)
Unchanged discomfort (%)	20.2 (n=36)	32.0 (n=57)	28.1 (n=50)	32.0 (n=57)
Coherent responses (%)	38.8 (n=69)		46.1 (n=82)	
Unclear responses (%)	61.2 (n=109)		53.9 (n=96)	

Table 4. Participant's responses to passive cervicothoracic mobilisation (in flexion and extension) applied during the structural differentiation for the straight leg raise (SLR) and knee extension angle (KEA) tests.

Figures

Figure 1.



Figure 2.



Figure 3

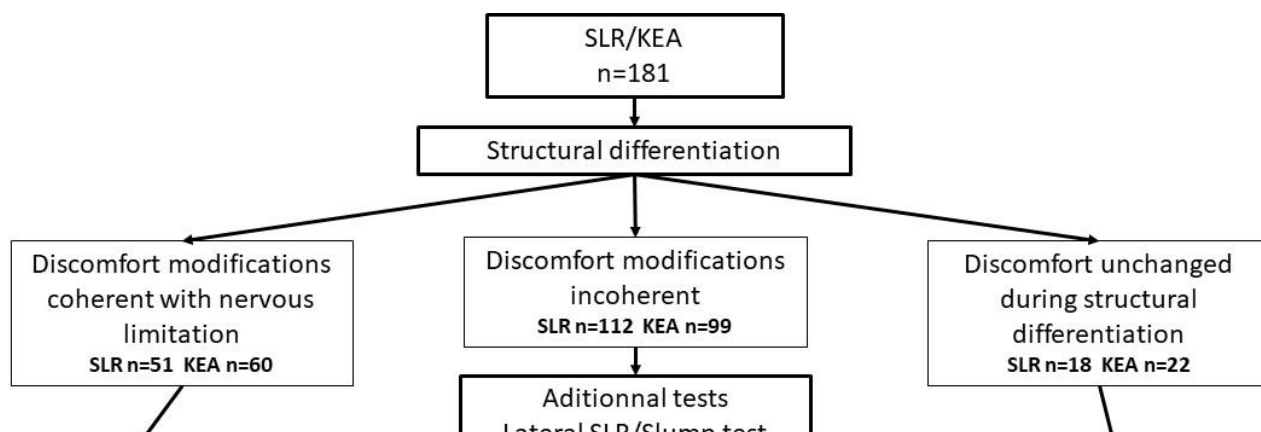
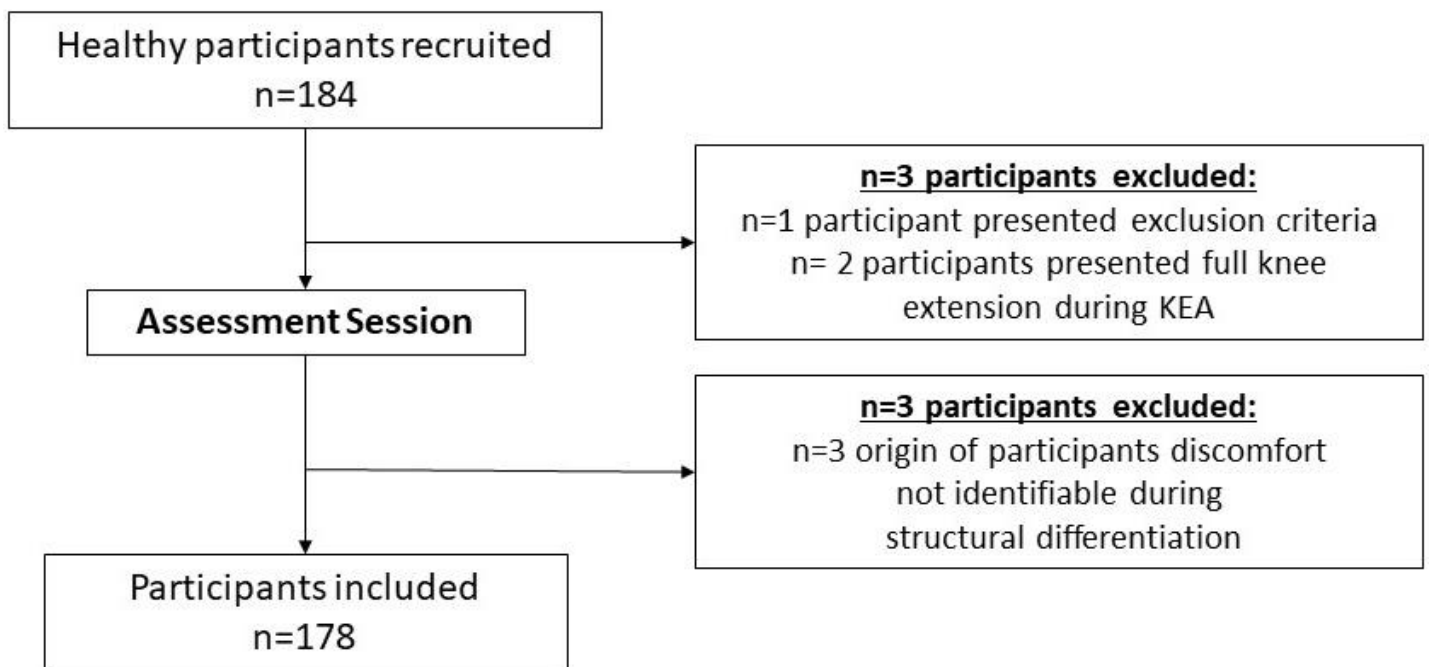


Figure 4



Figures captions

Figure 1. Assessment of the hip range of motion during the Straight Leg raise test.

Figure 2. Assessment of the knee range of motion during the Knee extension angle test.

Figure 3. Diagram representing the process used to identify the structure related to participants' discomfort during the straight leg raise test (SLR) and knee extension angle test (KEA) using structural differentiation.

Figure 4. Study flow Chart.