University Outpatient Clinic Potsdam, Sports Medicine & Sports Orthopaedics
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Motor control strategies in response to unexpected disturbances of dynamic postural control in people with and without low back pain

Dissertation

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ABSTRACT

Background: Low back pain (LBP) represents one of the world wide leading causes of limited activity and disability. Impaired motor control has been found to be one of the possible factors related to the development or persistence of LBP. In particularly, motor control strategies seemed to be altered in situations requiring reactive responses of the trunk counteracting sudden external forces. However, muscular responses were mostly assessed in (quasi) static testing situations under simplified laboratory conditions. Whether observed muscular responses following isolated trunk loading experiments are comparable to real-life response strategies under less restricted and dynamic conditions has been questioned. Hence, comprehensive investigations in motor control strategies during dynamic everyday situations are lacking. The present research project aimed to investigate muscular compensation strategies following unexpected gait perturbations in people with and without LBP. A novel treadmill stumbling protocol was tested for its validity and reliability to provoke muscular reflex responses both at the trunk and the lower extremities in an asymptomatic cohort (study 1). Thereafter, motor control strategies in response to sudden perturbations were compared between people with LBP and asymptomatic controls (CTRL) (study 2). In accordance with more recent concepts of motor adaptation to pain, it was hypothesized that pain may have profound consequences on motor control strategies in LBP. Therefore, it was investigated whether differences in compensation strategies were either consisting of changes local to the painful area at the trunk, or also being present in remote areas such as at the lower extremities.

Methods: All investigations were performed on a custom build split-belt treadmill simulating trip-like events by unexpected rapid deceleration impulses (amplitude: 2 m/s; total duration: 100 ms deceleration; 200 ms after heel contact) at 1m/s baseline velocity. A total number of 5 (study 1) and 15 (study 2) right sided perturbations were applied during each walking trial. Muscular activities were assessed by surface electromyography (EMG), recorded at 12 trunk muscles and 10 (study 1) respectively 5 (study 2) leg muscles. EMG latencies of muscle onset [ms] were retrieved by an automatic detection method followed by visual inspection. EMG amplitudes (root mean square (RMS)) were assessed within 200 ms post perturbation, normalized to RMS amplitudes of full strides prior to any perturbation [RMS%]. Latency and amplitude investigations were performed for each muscle individually, as well as for pooled data of muscles grouped by location and function. Characteristic pain intensity scores (CPIS; 0-100 points, von Korff questionnaire) based on mean intensity ratings reported for current, worst and average pain over the last three months were used to allocate participants into LBP (≥30 points) or CTRL (≤10 points). Test-retest reproducibility between measurements was determined using a compilation of absolute and relative measures of

reliability. Differences in muscular activities between LBP and CTRL were analysed descriptively (means with standard deviations and 95% confidence intervals) for individual muscles; differences based on grouped muscles were statistically tested by a general linear model, using a multiple analysis of variance (MANOVA, α =0.05; Pillai's trace test; post hoc comparisons with Bonferroni corrections).

Results: Thirteen individuals were included into the analysis of study 1. EMG latencies revealed reflex muscle activities following the perturbation (average: 89 ms; range: 75 to 117 ms). Respective EMG amplitudes were on average 5-fold of those assessed in unperturbed strides (range: 106 RMS% to 909 RMS%), though being characterized by a high inter-subject variability. Test-retest reliability of muscle latencies showed a high level of reproducibility, both for muscles at the trunk and the legs. In contrast, reproducibility of amplitudes was only weak to moderate for individual muscles, but increased when being assessed as a location specific outcome summary of grouped muscles. Seventy-six individuals were eligible for data analysis in study 2. Group allocation according to CPIS resulted in n=25 for LBP and n=29 for CTRL. Descriptive analysis of activity onsets revealed longer delays for all assessed muscles within LBP compared to CTRL (trunk muscles: average 10 ms; leg muscles: average 3 ms). Onset latencies of grouped muscles revealed statistically significant differences between LBP and CTRL for both right (p=0.009) and left (p=0.007) sided abdominal muscle groups. EMG amplitude analysis showed a high variability in activation levels between individuals, independent of group assignment or location. Statistical testing of grouped muscles indicated no significant difference in amplitudes between LBP and CTRL.

Discussion: The present research project could show that perturbed treadmill walking is suitable to provoke comprehensive reflex responses at the trunk and lower extremities, both in terms of sudden onsets and amplitudes of reflex activity. Moreover, it could demonstrated that sudden loadings indirectly applied at the trunk under dynamic conditions provoked altered reflex timing of muscles surrounding the trunk in people with LBP compared to CTRL. In line with previous investigations, compensation strategies seemed to be deployed in a task specific manner, with differences between LBP and CTRL being evident predominately at ventral sides. No muscular alterations exceeding the trunk (area of pain) could be found when being assessed under the automated task of locomotion. While rehabilitation programs tailored towards LBP are still under debate, it is tempting to urge the implementation of dynamic sudden loading incidents of the trunk to enhance motor control and thereby to improve spinal protection. Moreover, in respect to the consistently observed task specificity of muscular compensation strategies, such a rehabilitation program should be rich in variety.

ZUSAMMENFASSUNG

Hintergrund: Unterer Rückenschmerz (LBP) stellt eine der weltweit führenden Ursachen für eine eingeschränkte körperliche Funktion und Belastbarkeit dar. Defizite in der neuromuskulären Ansteuerung gelten als einer der möglichen Faktoren im Zusammenhang mit der Entstehung und Persistenz von LBP. Insbesodere in Situationen, die eine aktive Kompensation von plötzlich auftretenden Lasten am Rumpf beinhalten, konnten veränderte Strategien in der muskulären Antwort bei LBP aufgezeigt werden. Allerdings basierten solche Untersuchungen meistens auf (quasi) statischen Testsituationen unter vereinfachten Laborbedingungen. Ob die beobachteten muskulären Reaktionen isolierter Rumpfbelastungen repräsentativ sind für eine neuromuskuläre Ansteuerung unter dynamischen Alltagsbedingungen ist bisher nicht geklärt. Ziel der vorliegenden Arbeit war es, muskuläre Kompensationsstrategien in Folge unerwarteter Gangperturbationen bei Personen mit und ohne LBP zu untersuchen. Um muskuläre Reflexantworten sowohl am Rumpf als auch an den unteren Extremitäten zu provozieren, wurde ein neu entwickeltes Laufband-Stolperprotokoll auf seine Validität und Reliabilität getestet (Studie 1, asymptomatische Kohorte). Darauf aufbauend erfolgte der Vergleich neuromuskulärer Antworten in Reaktion auf plötzlich applizierte Gangperturbationen zwischen Personen mit LBP und asymptomatischen Kontrollpersonen (CTRL) (Studie 2). In Übereinstimmung mit aktuellen Modellen zu motorischen Anpassung bei Schmerzen wurde untersucht, ob Unterschiede in den beobachteten Kompensationsstrategien auf lokale Veränderungen am Rumpf reduziert sind, oder ebenfalls in rumpffernen Körperregionen auftreten.

Methoden: Alle Untersuchungen wurden mit Hilfe eines Spezial-Laufbands durchgeführt, welches mittels unerwarteter schneller Abbremsimpulse (Amplitude: 2 m/s, Gesamtdauer: 100 ms Verzögerung, 200 ms nach Fersenkontakt) die Simulation von Stolperereignissen während der Gangbewegung (1 m/s) erlaubt. Eine Gesamtanzahl von 5 (Studie 1) bzw. 15 (Studie 2) rechtsseitigen Perturbationen wurde während des Verlaufs des Stolperprotokolls appliziert. Muskuläre Aktivitäten wurden mittels Oberflächen-Elektromyographie (EMG) von 12 Rumpfmuskeln sowie 10 (Studie 1) bzw. 5 (Studie 2) Beinmuskeln aufgezeichnet. EMG-Latenzen wurden mit Hilfe eines automatisierten Detektions-Verfahrens mit anschließender visueller Überprüfung ermittelt. Die Berechnung der EMG Amplituden (RMS) erfolgte für den Zeitraum von 200 ms nach Perturbation, normiert auf den gesamten Schrittzyklus des unperturbierten Ganges [%]. Latenz- und Amplituden-Messgrößen wurden sowohl für jeden Muskel individuell, als auch für gepoolte Daten (gruppiert nach Lokalisation) berechnet. Charakteristische Schmerzintensitätswerte (CPIS, 0-100 Punkte, von Korff Fragebogen), basierend auf gemittelten Intensitätswerten (akute Schmerzen, sowie höchste und durchschnittliche Schmerzen der letzten drei Monate) wurden zur Einteilung in LBP (≥30 Punkte) und

CTRL (≤ 10 Punkte) verwendet. Zur Beurteilung der Test-retest Reliabilität wurden sowohl absolute als auch relative Reliabilitätsparameter herangezogen. Unterschiede in den Muskelaktivitäten zwischen LBP und CTRL wurden für individuelle Muskeln deskriptiv (Mittelwerte mit Standardabweichungen und 95% Konfidenzintervallen) analysiert. Gepoolte Daten gruppierter Muskeln wurden mittels multipler Varianzanalyse (MANOVA; $\alpha = 0,05$; Pillai's trace test; post hoc Vergleich mit Bonferroni-Korrektur) statistisch getestet.

Ergebnisse: Ergebnisse von 13 Probanden wurden für die Analyse von Studie 1 herangezogen. EMG-Latenzen zeigten Muskelaktivitäten repräsentativ für Reflexantworten im Nachgang applizierter Gangperturbationen, sowohl an Rumpf- als auch an Beinmuskulatur (Mittelwert: 89 ms, Range: 75 bis 117 ms). EMG-Amplituden erreichten im Durchschnitt ein 5-fach erhöhtes Aktivitätniveau innerhalb des 200 ms Zeitfensters nach Perturbation (Range: 106 RMS% bis 909 RMS%), jedoch gezeichnet von einer hohen interindividuellen Variabilität zwischen den Probanden. Eine hohe Reproduzierbarkeit für EMG-Latenzen konnte anhand der Reliabilitätsparameter aufgezeigt werden. EMG-Amplituden dagegen erwiesen sich als nur geringfügig reliabel bei der Betrachtung individueller Muskeln. Sechsundsiebzig Probanden waren für die Datenanalyse in Studie 2 geeignet. Die Gruppenzuteilung nach CPIS ergab n = 25 für LBP und n = 29 für CTRL. EMG-Latenzen zeigten eine erhöhte Aktivitätsverzögerung aller erfassten Muskeln für LBP im Vergleich zu CTRL (Rumpf: Mittelwert 10 ms; Bein: Mittelwert 3 ms). EMG-Latenzen gruppierter Muskeln zeigten statistisch signifikante Unterschiede zwischen LBP und CTRL sowohl für rechtsseitige ($p=0,009$) als auch für linksseitige ($p=0,007$) abdominale Muskelgruppen. EMG-Amplituden waren geprägt von einer hohen interindividuellen Variabilität, unabhängig von Gruppenzuordnung oder Lokalisation.

Diskussion: Das vorliegende Forschungsprojekt konnte belegen, dass Gangperturbationen dafür geeignet sind, umfassende Reflexantworten am Rumpf und den unteren Extremitäten zu provozieren. Darüber hinaus konnte gezeigt werden, dass unerwartete Gangperturbationen zu einer zeitlich verzögerten Reflexantwort der rumpfumgreifenden Muskulatur bei Personen mit LBP im Vergleich zur Kontrollgruppe führen. In Übereinstimmung mit den Ergebnissen vorheriger Untersuchungen erscheinen dabei die gewählten Kompensationsstrategien aufgabenspezifisch angepasst zu sein. Veränderte muskuläre Reaktionsmuster abseits des Rumpfes konnten trotz Einbezug weiterer Lokalisationen nicht gefunden werden. Gegenüber isolierten Rumpfbelastungen erlaubt der Einsatz indirekter Perturbationsbelastungen während des Ganges alltagsrelevante situationsspezifische Defizite neuromuskulärer Kontrolle gezielt zu untersuchen. Bei der Erstellung neuer Theapiekonzepte zur Steigerung der neuromuskulären Kontrolle sollte in diesem Zusammenhang die Einbindung alltagsähnlicher indirekter Belastungsformen des Rumpfes diskutiert werden.

BACKGROUND

1.1 Low back pain

Low back pain (LBP) represents one of the most prevalent disorders in western society, causing a substantial personal, societal and financial burden (Murray et al., 2015; Choi et al., 2010; Dionne et al., 2006; Ihlebaek et al., 2006; Neuhauser et al., 2005; Rapoport et al., 2004; Andersson, 1999). Moreover, during the last decades it has been shown that LBP has also become a major problem in low- and middle-income countries (Hoy et al., 2003; Jin et al., 2004; Hoy et al., 2010a) elevating LBP to one of the leading causes of activity limitations and work absence around the world (Lidgren, 2003). In the United States, LBP was estimated to be causing 149 million workdays lost per year, rendering the total costs of LBP to $14 billion per year (Gou et al., 1999). In the United Kingdom estimated indirect costs were found to be £10668 million (Maniadakis & Gray, 2000). While estimated costs of LBP vary due to the different approaches and definitions of costs related to LBP, it is apparent that LBP represents an important economic burden wherever it is studied (Dagenais et al., 2008). Despite the enormous impact of LBP, there is little known about the underlying mechanisms causing and preserving LBP for most people suffering from this condition (Andersson, 1999; Krismer & van Tulder, 2007; Airaksinen et al., 2006; Taylor et al., 2014).

Based on several epidemiological investigations LBP has been linked to a lifetime prevalence of up to 85% and a point prevalence varying from 12% to 38% (Hoy et al., 2012; Ihlebaek et al., 2006; Andersson, 1999). Furthermore, individual studies reported substantial rates of recurrence ranging from 20% to 90% (Hoy et al., 2010a; Carey et al., 1999; Abenhaim et al., 1988) within one year following initial occurrence. Heterogeneity of case definitions and prevalence periods of LBP among various studies might cause such significant differences in reported prevalence and recurrence rates (Hoy et al., 2010a). Results of a recently performed systematic review of global prevalence of LBP indicated a lifetime prevalence of 39% and a point prevalence of 18.3% for LBP worldwide (Hoy et al., 2012). According to this data, LBP is highest between the ages of 40 and 69 years, higher among females than males in all age groups, and more common in countries with high-income economies. LBP was also counted as one of the five leading causes of disability adjusted life years (DALYs) in “The Global Burden Disease Study 2013” (Murray et al., 2015). This measure represents the

overall disease burden, expressed as the number of years lost due to poor health, disability or early death. Longitudinal data could show that DALYs attributed to LBP increased over the last 23 years (1990 to 2013) moving LBP from rank 7 to rank 4 among the most common global burden diseases (Murray et al., 2015).

There is no consensual definition of low back pain available (Dionne et al., 2008; Ozguler et al., 2000; Hoy et al., 2010a). However, as a general definition LBP can be described as pain and discomfort, localized below the costal margin and above the gluteal crease, with or without referred leg pain (Airaksinen et al., 2006; Van Tulder et al., 2006). The addition of a minimum severity criterion: *"…bad enough to limit your usual activities or change your daily routine for more than one day"* has been proposed by an international panel of back pain experts in search of standardized case definitions of LBP (Dionne et al., 2008). Two important criteria to further discriminate LBP are its duration and underlying cause. Duration of pain is used to distinguish between acute and chronic forms of LBP. Thresholds used for differentiations between acute and chronic LBP vary among different studies (Koes et al., 2010; Majid & Truumees, 2008; Ganesh et al., 2014). However, prolonged pain for at least 3 months is often used to discriminate chronic LBP from its acute or sub-acute forms (Parthan et al., 2006; Airaksinen et al., 2006; Bogduk & Bogduk, 2004; Hoy et al., 2014, 2010b; Koes et al., 2010; Krismer & van Tulder, 2007). Furthermore, LBP is often characterized by fluctuations over time, thus pain appears in recurrent episodes with changes in intensities (van Tulder et al., 2002; Airaksinen et al., 2006). This condition can be referred as either chronic or recurrent LBP, based on the different existing definitions among studies (Stanton et al., 2010). In most individuals acute LBP will disappear within six to twelve weeks, only in 10% - 30% LBP becomes a chronic symptom (Parthan et al., 2006; van Tulder et al., 2002; Oliveira et al., 2012; Majid & Truumees, 2008; Andersson, 1999). However, rates of pain recurrence within a period of one year were found to be ranging between 35% - 70% (Pengel et al., 2003; Hestbaek et al., 2003; Abenhaim et al., 1988), rendering LBP itself as one of the best predictors for future LBP (Taylor et al., 2014; Hestbaek et al., 2003). Therefore, the traditional concept of LBP being defined as either single episodes of acute LBP or chronic LBP has been questioned (Von Korff & Saunders, 1996; Cedraschi et al., 1999; van Tulder et al., 2002).

As a crucial differentiation, LBP is often categorized as being specific or unspecific (Krismer & van Tulder, 2007; World Health Organization, 2003; Airaksinen et al., 2005). Specific LBP is referred to the presence of a localized source of pain with a specific structure of the spine being painful (Krismer & van Tulder, 2007). Therefore, specific LBP allows for specific diagnoses to characterize the cause of pain, such as compression fractures, neoplasm, spondyloarthropathies, scoliotic deformities or spinal infections (Airaksinen et al., 2006; World Health Organization, 2003). However, specific causes of LBP are only seen in about 10% - 20% of LBP patients (Airaksinen et al., 2006; Krismer & van Tulder, 2007; Cedraschi et al., 1999). Unspecific LBP is determined by exclusion, being not attributable to a specific pathological mechanism or anatomical source of pain and accounts for about 80% -95% of all LBP cases (Airaksinen et al., 2005; Cedraschi et al., 1999; Hoy et al., 2010a; Ehrlich, 2003). Thereby, unspecific LBP refers more to a symptom rather than to a diagnosis for most individuals suffering from LBP (Cedraschi et al., 1999).

Heterogeneity of research methods, case definitions and populations challenge the identification of risk factors linked to LBP (Hoy et al., 2010a; van Tulder et al., 2002). Thereby, history of LPB remains the most predictive risk factor for the development of future LBP (Taylor et al., 2014; Hestbaek et al., 2003). Identified risks have often been categorized into biomechanical, psychosocial and individual risk factors (Ferguson et al., 2012). Among biomechanical risk factors, increased durations of trunk flexion or rotation, prolonged standing and lifting heavy weights at work have been linked to LBP (Hoogendoorn et al., 2000a; Ferguson et al., 2012; Matsui et al., 1997; Magnusson et al., 1996; Taylor et al., 2014). Moreover, the occurrence of unexpected loading situations at the trunk, such as during slips, trips and falls, as well as bending and twisting while lifting have been repeatedly related to low back injuries (Radebold et al., 2000; Bigos et al., 1986; Frymoyer et al., 1983; Kelsey et al., 1984; Omino & Hayashi, 1992; Troup et al., 1981). All in common, those situations require a sudden muscle force generation to stabilize the human system and thereby increase the risk of large compressive and shear forces at the spine (Radebold et al., 2000). Among psychological factors, distress and in particular anxiety, depression and certain types of pain behavior were shown to be related to LBP (Hoy et al., 2010a; Vroman et al., 2009; Bailly et al., 2015; Bunzli et al., 2013). Psychosocial factors associated with LBP were found to encompass a lack of emotional support, low job satisfaction, monotonous tasks and poor work relations (Bailly et al., 2015; Andersson, 1999;

Hoogendoorn et al., 2000b). Furthermore, psychosocial factors were linked with the transition from acute to chronic LBP (Linton, 2000; Pincus et al., 2002). Among individual factors, overall prevalence of LBP has been found to increase with age until 40-69 years, and then to gradually decline (Stratford, 1999; Hoy et al., 2012). Evidence concerning the association of LBP prevalence with age has been however questioned, due to large heterogeneity of methods and prevalence figures used in different studies (Dionne et al., 2006). Sex differences in prevalence of LBP have been identified in some studies, with females being more prone to LBP than males (Hoy et al., 2012, 2010a). Other studies could not reveal gender as a risk factor for the development of LBP (van Tulder et al., 2002). Besides the high number of risk factors related to LBP, the underlying causes for the development and persistence of LBP remain almost elusive (Andersson, 1999; Krismer & van Tulder, 2007; Airaksinen et al., 2006; Taylor et al., 2014). Taken together, risk factors associated with the development of LBP are vast, however, most of them are not robust, replicable, yet modifiable (Taylor et al., 2014; Pincus et al., 2002).

Clinical guidelines for the management of LBP have been proposed, based on the evidence of numerous clinical trials and reviews (Middelkoop et al., 2013; Airaksinen et al., 2006). Treatment recommendations for acute forms of LBP have been found to be mostly consistent among published clinical guidelines (Koes et al., 2006; Middelkoop et al., 2013). Key elements of the proposed treatment strategy involve, among others, the advice to stay active and if necessary the prescription of pain relieving medications (Koes et al., 2006). Treatment strategies for unspecific chronic LBP mostly incorporate some form of supervised exercise intervention (Airaksinen et al., 2006; Koes et al., 2006; Callaghan & Nelson-wong, 2013; Burton et al., 2005). Thereby, exercise is often recommended as either stand-alone treatment or in combination with other strategies such as manual therapy and cognitive behavioral therapy (Koes et al., 2006; Nelson-Wong & Callaghan, 2010). However, no consensus exists about the appropriate types of exercise. While some clinical studies suggest specific back pain related exercises, recent systematic reviews constantly show that there is no evidence for a single best type of exercise (Choi et al., 2010; Poquet et al., 2016; Saragiotto et al., 2016).

In Summary, LBP is a very common but also quite heterogeneous condition, with pain being the best dominator and predictor for future development. While specific causes of pain can

be identified in some people, the underlying mechanisms and contributing factors remain unknown for the majority of individuals suffering from this condition (Taylor et al., 2014; Krismer & van Tulder, 2007). Hence, so far unspecific LBP is often described as a biopsychosocial phenomenon, as defined by the World Health Organization (Waddell & Burton, 2005; Kamper et al., 2015).

1.2 Motor control and the spine

"Motor control is a term that can be used to refer to all aspects of control of movement" (Hodges et al., 2013b). It is a process containing the decisions to move, the integration and processing of sensory inputs to the system providing information about the body segments as well as the environment, and finally the motor output by coordinated muscle activities to fulfil the desired movement (Rosenbaum, 1991; Hodges et al., 2013b; Wise & Shadmehr, 2002). Thereby, motor control relies on a cooperative interaction between central neuronal circuits and peripheral skeletomuscular activities (Chiou et al., 2014).

Motor control is vital for the spine as it is a complex structure composed of 24 individual vertebral bodies, the sacrum, intervertebral disks in between as well as numerous ligaments and muscles (Ebenbichler et al., 2001; Ferguson, 2008; Van Dieën & Kingma, 2013). Protections of the spinal cord, transfer of loads from the head and the trunk to the pelvis and vice versa are principal functions of spine (Ferguson, 2008; Panjabi, 1992). Yet, these tasks have to be performed under various conditions, requiring the spine to change its characteristics from being flexible to rigid within a short time (Panjabi, 1992; Hammill et al., 2008). Active control is crucial for such an adaptation and in particularly challenging in dynamic situations, when the spine is exposed to reactive forces (Ebenbichler et al., 2001). Panjabi et al (1992) introduced a theoretic model of three subsystems associated with the control of the spine: the passive system, the active system and the neuronal control system (figure 1). The passive system, consisting of discs, ligaments, joints and capsules is thought to play a role in restricting the range of spinal motions, as well as providing feedback about vertebral positions and motions. Therefore, it also could be seen as a dynamic monitoring system, providing the control system with required information about the current state. The active system, comprised of various muscles and tendons around the spine, is thought to generate the necessary forces to stabilize the spine as well as to provide the control system

with information about acting forces. The neuronal control system is thought to process the information received from numerous transducers of the passive and active subsystem. Also, it determines the necessary actions to be executed by the active subsystem, adjusted to the specific situation (Panjabi, 1992).

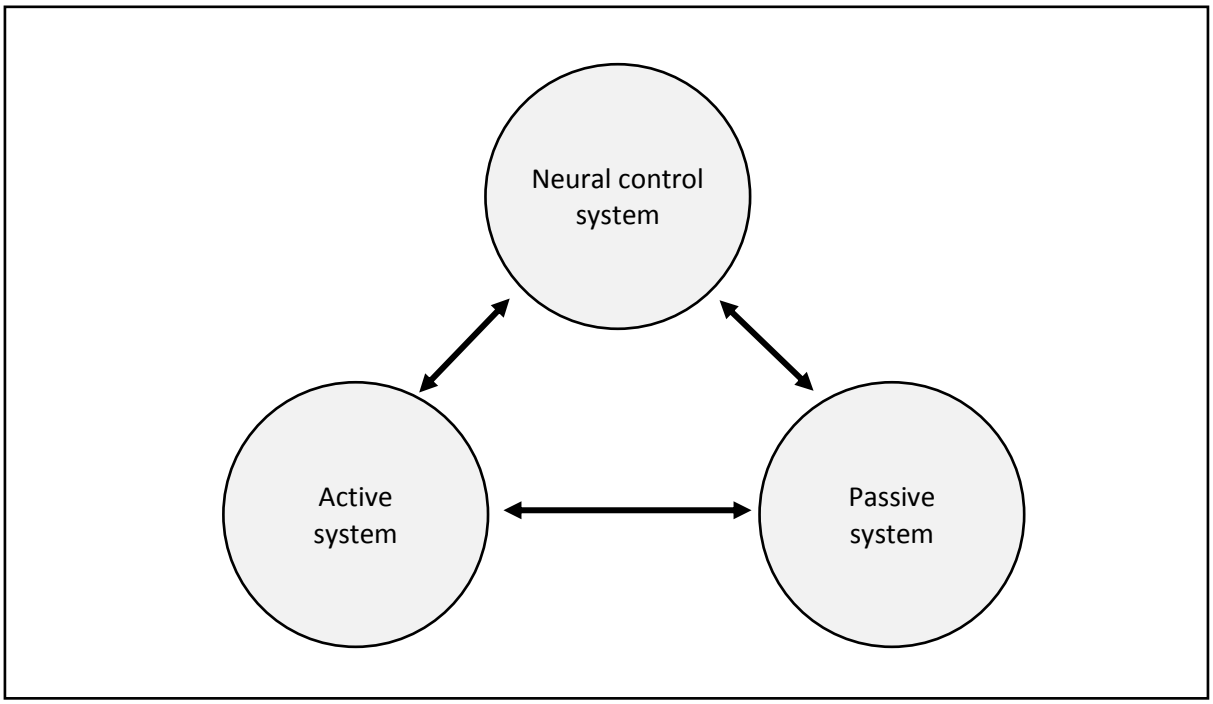

Figure 1: Panjabi's theoretic model of the three subsystems associated with the control of the spine; adapted from Panjabi 1992: The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *Journal of spinal disorders*, 5(4), pp.383–9

Constant feedback and control of spinal movement and position seems to be a key element of spinal function (Brumagne et al., 2013). Deficits in one of the underlying subsystems could potentially be enough to challenge the whole system to work properly (Panjabi, 1992; Hammill et al., 2008; Radebold et al., 2001). The need of constant control of the spine is already given by its multi segmental nature, as its biomechanical characteristics are comparable to an inverted pendulum (Reeves et al., 2007). Thereby, simply keeping posture in a static situation already requires active adjustments and re-adjustment to maintain static equilibrium (Massion, 1992). Dynamic situations with external threats to dynamic equilibrium require the deployment of an even greater amount of reactive mechanisms (Ferber et al., 2002; Brumagne et al., 2013). Control of the spine during locomotion, for example, needs to adapt to the changing base of support during each stride with proactive

and reactive response strategies; especially when exposed to additional external perturbations (Marigold & Patla, 2002; Patla, 1996).

Muscular support has been shown repeatedly to play an important role in maintaining spinal control under various conditions (Ebenbichler et al., 2001; Granata & Marras, 1995; Gardner-Morse & Stokes, 1998; Barr et al., 2005; Stokes et al., 2000). Muscular activity is necessary to counter-act forces as well as to absorb potentially harmful impacts applied to the spine by either agonistic or antagonistic work or by muscular co-contractions (McGill et al., 2003). Functionally, the trunk musculature can be divided into flexors (abdominal muscles and psoas muscles) and extensors (sacrospinalis group, transversospinal group and short back muscle group) (Ferguson, 2008). Stabilization of the spine is acquired by an coordinated interaction of all of these muscles (Ebenbichler et al., 2001; Wirth et al., 2016). However, it is hypothesized that not all muscles contribute via the same mechanisms to this overall goal (Borghuis et al., 2008). Therefore, trunk musculature were further grouped into local stabilizers, global stabilizers as well as global mobilizers (Ferguson, 2008; Borghuis et al., 2008; Comerford & Mottram, 2001). According to this categorization, local stabilizers are paravertebral muscles, usually spanning single spinal segments. Their main function is thought to be stiffening of the spinal segment and to control motion rather than to induce motions, especially in neutral position of intervertebral joints, where passive support from ligaments and capsules are minimal (Comerford & Mottram, 2001; Borghuis et al., 2008). Important contributors of the local stabilization have been found to be for example the Mm. multifidii, intertransversarii and interspinales (Anderson & Behm, 2005; Borghuis et al., 2008). The global stabilizing system is built by polysegmental paravertebral muscles, primarily balancing the external loads to minimize resulting forces at the spine (Borghuis et al., 2008). Therefore, the global stabilizers are thought to be primarily responsible for concentrically shortening into full physiological inner range position, isometrically hold position and eccentrically control or decelerate functional load against gravity (Comerford & Mottram, 2001). Lastly, global mobilizers are defined as larger, torque producing muscles, being the originator of movement. Their primary function is thought to be the stabilization under high load or strain, such as during lifting, pushing, pulling or ballistic shock absorption, as well as to enhance or reduce spinal rigidity (Comerford & Mottram, 2001). Important global stabilizing muscles where found to be for example Mm. rectus abdominis, external

oblique, internal oblique and latissimus dorsi (Anderson & Behm, 2005; Sciascia & Cromwell, 2012; Danneels et al., 2001; Anderson et al., 2011).

While muscular support is important for spinal function, it is pointless without an adequate recruitment and timing of muscle activities (Ebenbichler et al., 2001; Borghuis et al., 2008; Radebold et al., 2000; Dolan & Adams, 2013). Therefore, the muscular system relies on the processing of sensory information from multiple sensory inputs, such as from peripheral mechanoreceptors (located in the skin, joints, ligaments, tendons and muscles) , vestibular receptors and visual input (Biedert, 2000; Lephart et al., 1997). Processing of afferent sensory information and initiation of efferent information flow to allow motor responses is deployed under three levels within the central nervous system: the spinal cord, the lower regions of the brain and the cerebral cortex (Biedert, 2000; Guyton & Hall, 2006; Lephart et al., 1997; Radebold et al., 2001). At the spinal cord afferent impulses of mechanoreceptors are processed within the gray matter, eliciting both local segmental reflexes, as well as transmitting signals to higher levels within the central nervous system. Motor neurons are located at the anterior horns of the cord gray matter to innervate muscle fiber via specific motor neurons. Continuous sensory feedback of muscle function is provided by muscles spindles and golgi tendon organs, reporting about tension, length and its rate of change (Shumway-Cook & Woollacott, 2007). Excitations of muscle spindles causing contractions of skeletal muscle fibers within the same muscle are the simplest form of reflex activity, named simple stretch reflexes (Riemann & Lephart, 2002). Consequently, the cord level of motor control serves to elicit immediate muscular excitations as well as to forward constant sensory information to higher levels of the central nervous system. More complex motor responses are processed at the lower regions of the brain such as the brain stem, the basal ganglia and the cerebellum. Every sensory and motor nerve passes through the brain stem (Biedert, 2000). Receiving commands of higher centers to modify specific control functions throughout the body, as well as the control of body movement and equilibrium are among its core functions (Shumway-Cook & Woollacott, 2007). Timing of motor activities and rapid progression from one movement to another is coordinated within the cerebellum (Biedert, 2000; Kenney et al., 2012). Here, the intended program of muscle contraction gets compared with the continuously updated information from the peripheral areas and, if necessary, corrective adjustments are determined to produce the desired movement. Therefore, all conscious decisions of motor activities initiated by the motor cortex are

transmitted to the cerebellum. At the highest level of motor control, the cerebral cortex is involved in all conscious motor activities. However, successful performance of the desired movement involves simultaneous activation of different functions in the spinal cord, brain stem, basal ganglia and the cerebellum (Biedert, 2000; Guyton & Hall, 2006). Thereby, motor control relies on the interaction of all three levels of the central nervous system with individual contributions being dependent on the specific situation (figure 2) (Biedert, 2000; Radebold et al., 2001; Shumway-Cook & Woollacott, 2007).

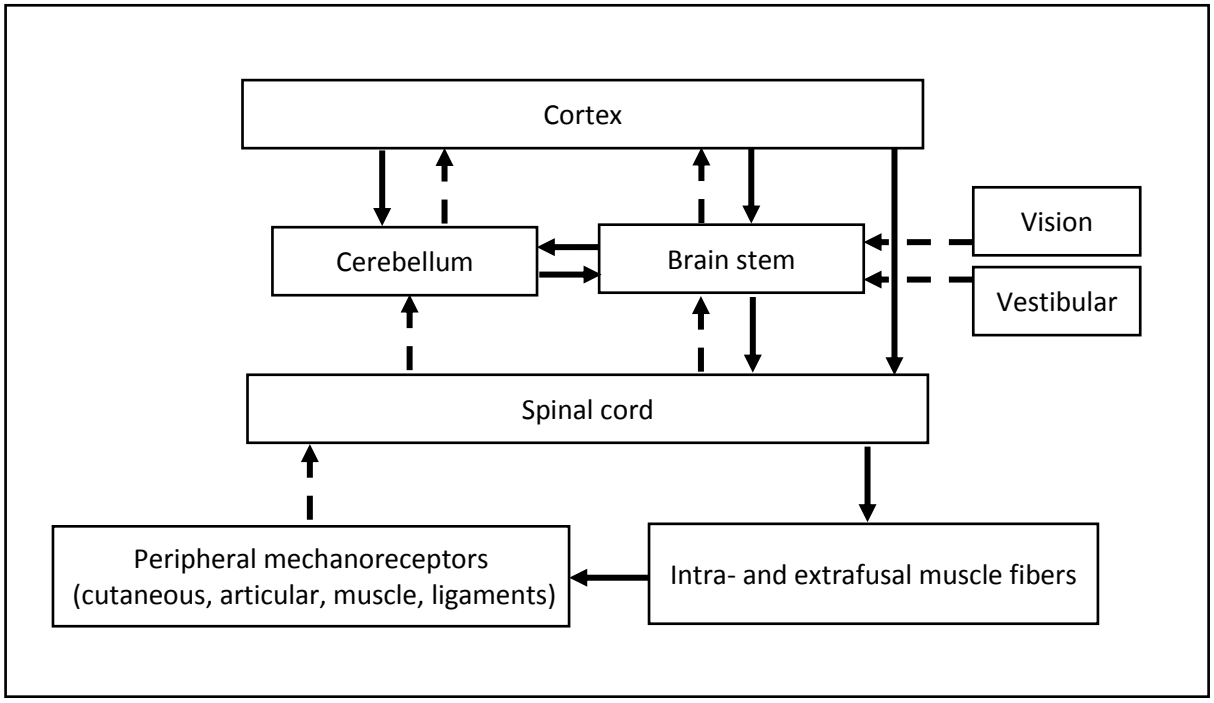

Figure 2: Afferent (dotted lines) and efferent (solid lines) pathways of the central nervous system involved in motor control; adapted from Riemann et al. 2002: The sensorimotor system, part I: The physiologic basis of functional joint stability. *Journal of Athletic Training*, 1(1), pp.71–79

As the spine is often considered to be an unstable mechanical system, clinicians and researchers often use the term stability in the context of motor control of the spine (Van Dieën & Kingma, 2013). This term is, however, discussed controversially, as clear definitions are lacking (Van Dieën & Kingma, 2013; Reeves et al., 2007; Panjabi, 1992; Borghuis et al., 2008). In a seminal paper, Reeves et al (2007) explained, that *"stability... is a term that appears to change depending upon the context, and as such, appears to have unstable definitions."* From a mechanical perspective, a systems' stability would be typically tested by applying a perturbation and comparing the new behavior to the old behavior. The system

would be considered stable if the old behavior stays principally unaltered. If the new behavior is significantly different to the old behavior, the system would be described as unstable (Reeves & Cholewicki, 2013; Reeves et al., 2007). Accordingly, there is no degree of stability; the system would be either stable or unstable. This definition becomes challenged when being seen from a clinical perspective (Van Dieën & Kingma, 2013). Stability of the spinal system would not necessarily be considered a simple return to the behavior or trajectory of the initial task, but rather be dependent on whether coping with the new situation can be accomplished without generating injurious forces or excessive tissue strain (Reeves et al., 2007). Also, the term stability has frequently been used as synonymous to stiffness of the spine. While stiffness of the spine is an important factor in some situations to prevent spinal injury, other situations may require adapting with a more subtle, well-coordinated muscular interaction, specific to the situation (Hodges, 2013; Borghuis et al., 2008). Being a dynamic system, the spine constantly has to adapt its characteristics (its behavior) according to the changing demands (Hammill et al., 2008; Ebenbichler et al., 2001). Moreover, real-world perturbations are rarely applied at the spine exclusively. Therefore, spinal control has to be deployed in conjunction with other strategies, such as e.g. performing a step in response to a perturbation during bipedal stance (Van Dieën & Kingma, 2013; Marigold & Misiaszek, 2009).

In summary, motor control of the spine is founded by the interaction of several contributors, being of both active and passive nature. The mechanical system is characterized by a vast redundancy, the control systems are manifold and the sensory systems are endowed with a variety of receptors. It almost seems, as if the spine could easily cope with arising deficits in one or another of the connected subsystems (Parnianpour, 2013). However, at closer inspection it becomes apparent that this complex system relies on each of its contributors in order to adapt itself from being flexible to being rigid and vice versa in an unpredictable and changing environment (Parnianpour, 2013; Hodges & Richardson, 1996; Cholewicki et al., 2000; Stokes et al., 2000). Also, it becomes obvious, that the definition and exact understanding of motor control is not always consistent, but rather dependent upon the perspective.

1.3 Impairments of motor control in people with LBP

In both clinical practice and research, impaired motor control has been proposed as one of the underlying contributors for the development or persistence of LBP (Hodges & Richardson, 1996; Jones et al., 2012a; Radebold et al., 2000; Stokes et al., 2006; Cholewicki & McGill, 1996; Panjabi, 1992; Hodges et al., 2013b). Though the exact mechanisms leading to alterations in motor control remain mostly unknown, cross-sectional investigations revealed a variety of changes in people with LBP, such as increased postural sway, decreased postural control, delayed muscle responses to sudden spine loadings, delayed muscle shut-off following load releases and imbalances of contra-lateral muscle activities (Parkhurst & Burnett, 1994; Newcomer et al., 2000; Luoto et al., 1998; Nies & Sinnott, 1991; Radebold et al., 2001; Reeves et al., 2005; Magnusson et al., 1996; Cholewicki et al., 2000; Larivière et al., 2005).

Some changes in motor control became apparent in fairly simple tasks of postural control. Repositioning accuracy of the spine in sitting or standing position has been investigated in a variety of studies (Brumagne et al., 2000; Gill & Callaghan, 1998; O'Sullivan et al., 2003; Brumagne et al., 2013). Most of these investigations reported increased reposition errors in LBP patients compared to controls; however, some studies found no differences at all (Newcomer et al., 2000; Asell et al., 2006; Descarreaux et al., 2005). Other studies were targeted towards balance control focusing on postural sway in a variety of conditions. Alhough simple standing seemed not to be valid to distinguish alterations in postural control, more challenging situations, e.g. uni-pedal stance or unstable support surface, could show significant increases in postural sway in people with LBP (Mientjes & Frank, 1999; della Volpe et al., 2006; Luoto et al., 1998; Henry et al., 2006). Deficits in proprioception are often hypothesized to be a plausible mechanisms for altered motor control during these tasks (Gill & Callaghan, 1998; Brumagne et al., 2000; Lamoth et al., 2006). While all of these situations are characterized by the demand to precisely control the spinal position, most of them do not require reactive responses to sudden changes in the environment. However, the latter seems to be especially challenging for motor control in people with LBP (Radebold et al., 2000, 2001; Cholewicki et al., 2005; Reeves et al., 2005). Investigations in neuromuscular responses to sudden external loadings revealed a variety of changes in motor control in people with LBP. Muscular activity recorded by

electromyography (EMG) could show changes in activation level, timing of muscle activation and changes in intermuscular recruitment pattern of the muscles surrounding the trunk (Navalgund et al., 2013; Larivière et al., 2010; Radebold et al., 2000). In early investigations, Hodges at al. (1996) identified delayed muscular responses of M. transversus abdominis following rapid shoulder movements in people with LBP. These changes were hypothesized to result in an insufficient muscular stabilization of the spine (Hodges & Richardson, 1996). Responses to sudden load release in standing position by Magnusson et al. (1996) further indicated increased latencies of M. erector spinae in people with LBP (Magnusson et al., 1996). A series of investigations using a custom build apparatus (figure 3) to test muscular responses of the major trunk muscles subsequent to quick load releases showed further alterations in motor control (Radebold et al., 2000, 2001; Cholewicki et al., 2005; Reeves et al., 2005). Following load release during isometric contractions in flexion/extension and lateral bending, LBP patients experienced delayed shut-off times of agonistic muscles compared to controls (Radebold et al., 2000). Moreover, their response demonstrated a change in pattern of co-contraction strategies, as agonistic muscles remained active while antagonistic muscles already switched on. Investigations in activity levels of EMG demonstrated elevated activation levels prior to and reflexive of sudden perturbations in people with LBP compared to controls (Stokes et al., 2006; Larivière et al., 2010). Though, other studies found contrary results with EMG amplitudes being lower in LBP patients (MacDonald et al., 2010). In conclusion, reactive responses to isolated trunk loading or unloading repeatedly showed evidence for changes in motor control in people with LBP. Direct load application at the trunk enabled the minimization of potential confounding variables, such as reactive responses of other parts of the body (Maaswinkel et al., 2016). Few studies have investigated muscular responses to sudden load application both at the trunk and offsite the trunk in people with LBP in order to identify if changes in motor control exceed the area of the trunk (Jacobs et al., 2011; Jones et al., 2012b). By using support surface translations in standing position, Jacobs et al (2011) identified higher normalized baseline EMG amplitudes at the trunk (abdominal and back) and the lower extremities (ankle) as well as lower incidents of EMG burst onsets at the trunk and distal leg muscles. In addition, in a study by Jones et al. (2012) LBP patients demonstrated increased muscle activity following perturbations in directions where muscles acted as prime mover and reduced muscle activity in opposing directions, both evident in trunk muscles and lower

extremities (Jones et al., 2012b). Based on their results, the authors suggested that motor control in LBP might be influenced by central nervous systems processing containing changes both at the trunk and offsite the trunk. Changes in preparatory and triggered reactions of upper limb loading and impaired postural stability during one-footed and externally disturbed two-footed stance in people with LBP might further provide indications for changes in motor control exceeding the trunk (Luoto et al., 1998; Leinonen et al., 2007). However, some of those findings might also just show the consequences of an altered control at the level of the trunk.

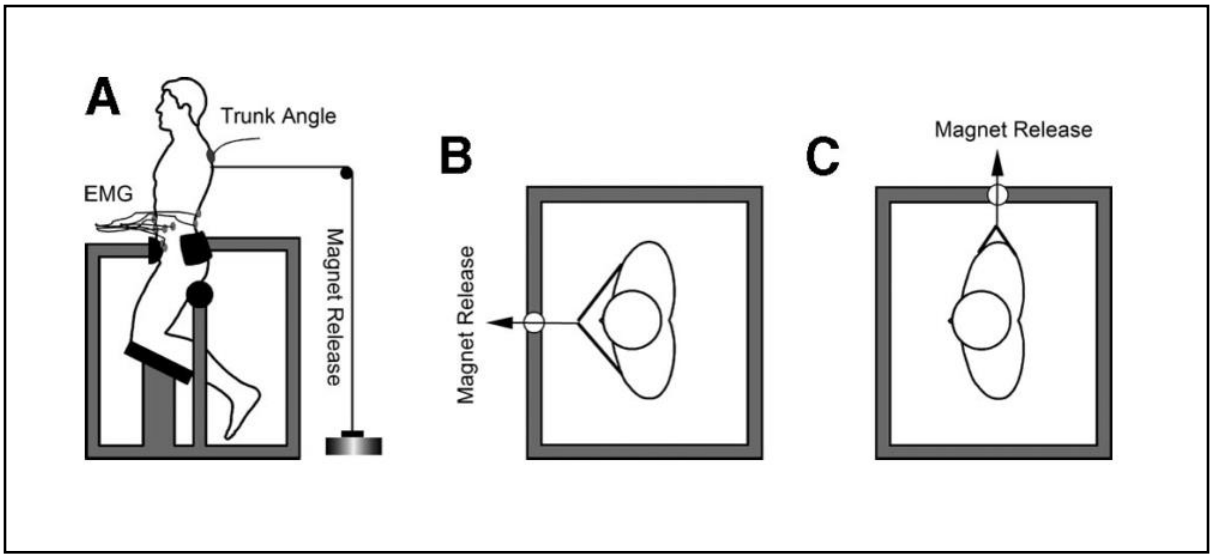

Figure 3: Apparatus for quick-releases with semi seated position of the subjects; Flexion (A), extension (B), lateral bending (C) loads were applied via a system pulleys; out of: Cholewicki, J., Silfies, S. P., Shah, R. A, Greene, H. S., Reeves, N. P., Alvi, K. and Goldberg, B. (2005) 'Delayed trunk muscle reflex responses increase the risk of low back injuries.', Spine, 30(23), pp. 2614–20

1.4 Motor control and pain

Impairment of motor control may arise from a variety of potential sources, hence the number of proposed theories of underlying mechanisms is vast (Reeves & Cholewicki, 2013). A well-adjusted interaction of passive, active and controlling contributors is crucial for motor function, as impairments of each sub-system may cause the whole system to be degraded (Panjabi, 1992; Hammill et al., 2008; Radebold et al., 2001). Impairments of the active sub-system such as decreased muscle strength indicated alterations in muscular capacity in LBP (Dvir & Keating, 2003; Sjölie & Ljunggren, 2001; Yahia et al., 2011; Thomas et

al., 2008), with signs of reorganized muscle fiber characteristics (Mannion et al., 2000) and muscle atrophy (Danneels et al., 2000). However, the majority of identified alterations in motor control in people with LBP might be attributed to the controlling system. Decreased proprioceptive and kinesthetic awareness of the trunk were repeatedly reported in people with LBP (Henry et al., 2006; Mok et al., 2004; Radebold et al., 2000; Reeves et al., 2005; Brumagne et al., 2000; O'Sullivan et al., 2003). Only a few studies could not detect such changes (Descarreaux et al., 2005), which might be attributed to the heterogeneity between investigated populations (O'Sullivan et al., 2003). Consequently, decreased sensory function and thereby an insufficient feedback mechanism was often discussed as one of the possible mechanisms linked to LBP (Borghuis et al., 2008). However, there is also growing belief that people with LBP may suffer from a faulty control logic, lacking flexibility in control strategies which are normally available in asymptomatic individuals (Reeves & Cholewicki, 2013; Hodges & Cholewicki, 2007). Dominated by the goal to protect the painful area from further pain, the deployed control strategies might foster other detrimental effects, such as premature fatigue of the surrounding musculature (Hodges & Tucker, 2011). It was hypothesized that changes in motor control might also represent a centrally generated change in muscle synergies (Jacobs et al., 2011; Jones et al., 2012b). However, while numerous studies investigated in local changes in motor control at the area of the trunk, only a few studies investigated changes upstream in the motor control system in people with LBP (Van Dieën & Kingma, 2013). Evaluations of transcranial magnetic stimulations revealed a reorganization of trunk muscle representation at the motor cortex in people with LBP (Tsao et al., 2008). Also, EEG investigations showed altered late-phase cortical processing of postural perturbations in co-existence with altered kinematic and muscle responses in people with LBP (Jacobs et al., 2016). Though functional consequences of these preliminary findings remain unclear, they might indicate that LBP is associated with changes at several levels of the motor control system.

From a more theoretical background, adaptations of motor behavior in pain have been commonly interpreted by the two following models: the 'pain-spasm-pain' model and the 'pain-adaptation' model (van Dieën et al., 2003; Travell et al., 1942; Lund et al., 1991). According to the 'pain-spasms-pain' model (also called the vicious cycle theory) pain results in an increased activity which in return will lead to an accumulation of metabolites and consequently further increase pain, regardless of the task (Hodges & Tucker, 2011; Roland,

1986). This mechanism is thought to be beneficial in response to acute trauma, as intense contractions of the muscles surrounding the injured site would decrease motion by co-contraction and therefore prevent further injury. However, in non-traumatic pain, this reaction could rather be detrimental, as over time pain would lead to more pain (van Dieën et al., 2003). Two different underlying pathways of the 'pain-spasm-pain' model have been proposed. According to Wyke (1987) nociceptive afferent information is travelling via the spinal cord to both higher nervous centers for pain perception and to alpha motor neurons on segmental level causing an increase in muscle activation (spasm) (Wyke, 1987). Alternatively, Johansson and Sojka (1991) proposed a pathway where nociceptive information travels to gamma motoneurons, affecting increased muscle spindle output and thereby resulting in an hyperexcitability of the alpha motoneuron pool (Johansson & Sojka, 1991).

The 'pain-adaptation' model in contrast postulates that pain will decrease activity in muscles acting as agonist and increase activity in muscles acting as antagonist, thereby being adaptive to the function of each muscle (van Dieën et al., 2003). A reduced movement velocity and limited movement excursion are thought to be the goal of such an adaptation to pain. The respective pathway was proposed to be based on nociceptive information transferred either via inhibitory or excitatory interneurons to the alpha motoneuron pool (van Dieën et al., 2003). Domination of either excitatory or inhibitory interneurons is thereby controlled by the central nervous system, in dependence of the motor command (van Dieën et al., 2003). Both models consider changes in motor control to be an adaptation to pain and not vice versa. However, it should be mentioned, that this conclusion can't be drawn in relation to LBP with the existing data (Callaghan & Nelson-wong, 2013). Also, both models are primarily based on animal studies or experimentally induced acute pain via noxious substances (van Dieën et al., 2003). This might explain why observations in LBP are only partially in congruence with the two existing theories (Callaghan & Nelson-wong, 2013). As described before, changes in motor control related to LBP are variegated and not as uniform as required to be related to either the 'pain-spasm-pain' model or the 'pain-adaptation' model (Hodges, 2013). As an example, delayed muscular latencies in response to sudden loading could be interpreted as an indicator of a reduced excitability being in line with the 'pain-adaptation' model, observations of an increase in activity levels would be more in line with the 'pain-spasm-pain' model predicting an increased muscular activity in

response to pain. In summary, the observed alterations in motor control related to LBP do not follow the theoretic predication of either a uniform inhibition ('pain adaptation') or facilitation ('pain-spasm-pain') of muscle activity being the cause of pain or causing painful movements (Hodges & Tucker, 2011; van Dieën et al., 2003). In a more recent approach Hodges and Tucker (2011) introduced a new model of 'motor adaptation to pain' based on the assumption that"…*adaptation to pain aims to reduce pain and protect the painful part, but with a more flexible solution than currently proposed*" (Hodges & Tucker, 2011). According to this model, it is hypothesized that motor adaptation to pain (1) aims to protect from further pain or injury, (2) involves redistributions of activity within and between muscles, which (3) leads to changes in mechanical behavior and (4) involves changes at multiple levels of the motor system, which may be complementary, additive or competitive, (5) with short-term benefits, but potential long-term consequences (Hodges & Tucker, 2011). Protection of the painful region from further pain is assumed to be the driving factor to deploy new strategies, both in the presence of real or perceived risks of pain or injury (figure 4).

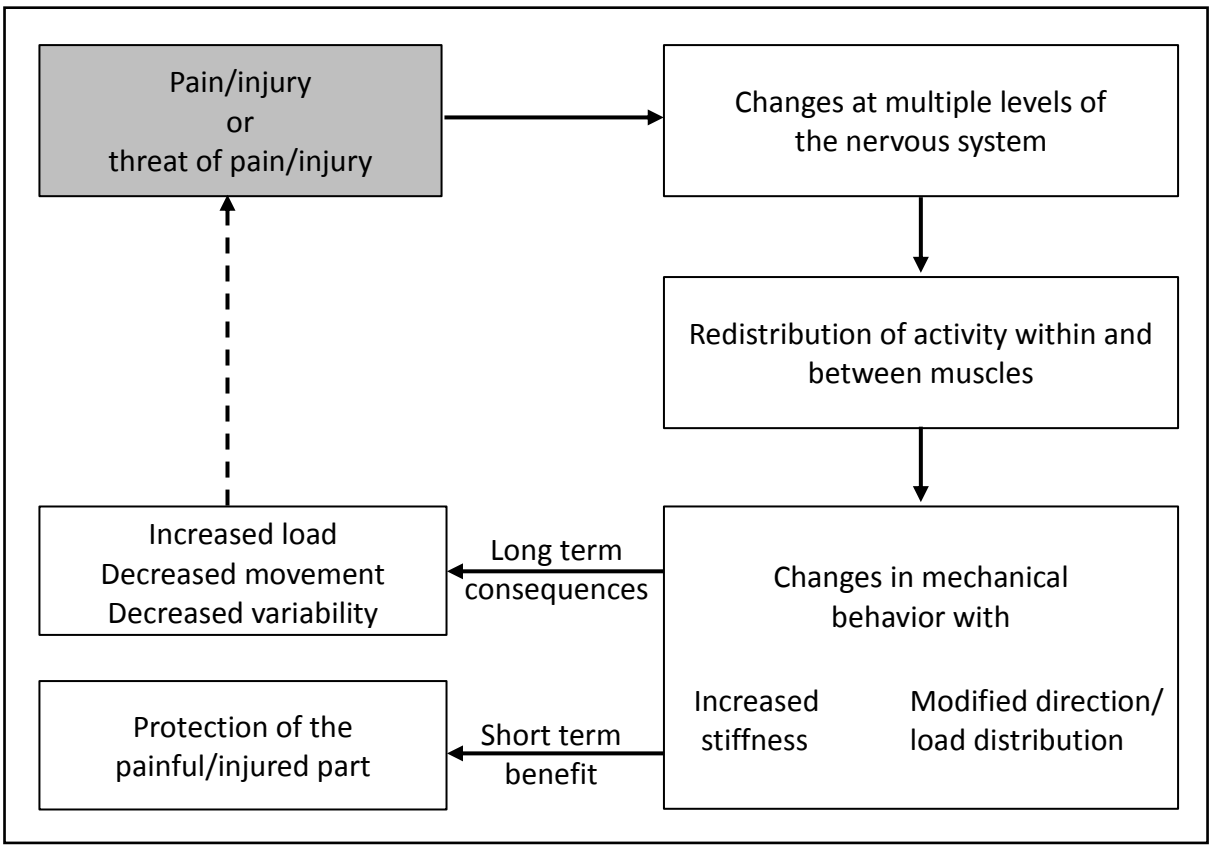

Figure 4: New theory of 'motor adaptation to pain'; adapted from Hodges, P. W. and Tucker, K. (2011) 'Moving differently in pain: a new theory to explain the adaptation to pain, Pain. International Association for the Study of Pain, 152(3 Suppl), pp. 90-8

In contrast to previous theories, the outlined model is based on the idea that the nervous system may reorganize the activity between muscles in a more complex way to find a new solution to the supposedly harmful situation. Also, changes in to motor control would require higher motor function and motor planning to contribute to the new motor behavior, such as the recruitment of a more protective strategy in advance of movements or reorganizations of cortical regions (Tsao et al., 2008; Hodges & Tucker, 2011). As the final element of the proposed framework, adaptations may in the short-term protect from further pain or injury, but in the long term may have consequences that could lead to further problems, such as increased loads, decreased movement and variability (Hodges & Tucker, 2011). A variety of changes in motor adaptation in people with LBP are in congruence with this new theory of 'adaptation to pain'. From isolated trunk loading experiments, to more complex investigations in changes of muscle activities proximally and distally to the trunk, to transcranial magnetic stimulations, changes in motor control have been proven to involve more complex neural processes than those proposed by the existing theories that advocate stereotypical changes in the presence of LBP (Tsao et al., 2008; Jacobs et al., 2011; Radebold et al., 2001). However, the hypothesis of the established framework will need further validation by researching more thoroughly the deployed motor control strategies related to LBP.

2 RESEARCH PARADIGM

Impaired motor control of the lumbar spine has been proposed as one of the possible mechanisms underlying LBP (Panjabi, 1992; Cholewicki & McGill, 1996; Hodges et al., 2013b). Previous investigations detected alterations in motor control at the trunk particularly in situations of sudden load changes (Radebold et al., 2000; Cholewicki et al., 2005; Reeves et al., 2005). Thereby, LBP patients showed changes in muscle recruitment patterns in muscles of the trunk, with muscular responses being delayed or altered in activity levels and patterns of co-activations (Radebold et al., 2000; Cholewicki et al., 2005; Reeves et al., 2005; Stokes et al., 2006; Jacobs et al., 2011; Jones et al., 2012b). One limitation of previous studies is however that muscular responses were mostly tested under static and simplified conditions with external loads being applied directly at the trunk in (quasi) static standing or half seated positions (Radebold et al., 2000, 2001; Cholewicki et al., 2005; Reeves et al., 2005). While these specific testing situations enabled the study of isolated trunk responses to sudden external loading, they did not allow the investigation of reactive responses as they may occur under real life circumstances (Arendt-Nielsen et al., 1996). Outside laboratory conditions, external loadings are rarely applied directly at the trunk, but usually transferred indirectly to the trunk via upper or lower extremities (Marigold & Misiaszek, 2009). Thereby, trunk responses have to be realized in conjunction with responses of other contributors, such as the lower extremities in situations of perturbed postural control. Only a few studies investigated changes in motor control offsite the trunk in response to sudden perturbations (Jacobs et al., 2011; Jones et al., 2012b). Those investigations showed an altered response pattern at the trunk as well as at the lower extremities in response to sudden surface translations in free standing. Moreover, these first findings support the notion that motor adaptation in pain may not simply consist of changes in excitability at the painful area, but rather cause a comprehensive restructure of motor control, both at the region of pain and offsite (Hodges & Tucker, 2011).

In summary, motor control in LBP has been mostly assessed in isolated loading situations and few studies have been conducted to investigate in more comprehensive loading situations, requiring compensation strategies of the trunk in conjunction with those offsite the trunk (figure 5). Moreover, the latter was exclusively applied during static situations, with stiffening strategies such as co-contractions of large flexor and extensor muscles being

thought to be the primary mechanisms to maintain postural control (Hodges, 2013). Comprehensive investigations in motor control during dynamic situations are lacking. Dynamic control is the ultimate goal of motor control of the human system, as the body must continuously respond to the changing circumstances (Brumagne et al., 2013). Furthermore, dynamic situations might also be well suited to assess impairments of motor control, as they rely on a variety of control strategies ranging from co-contractions to carefully timed alternating bursts of muscle activities (Brumagne et al., 2013; Hodges, 2013).

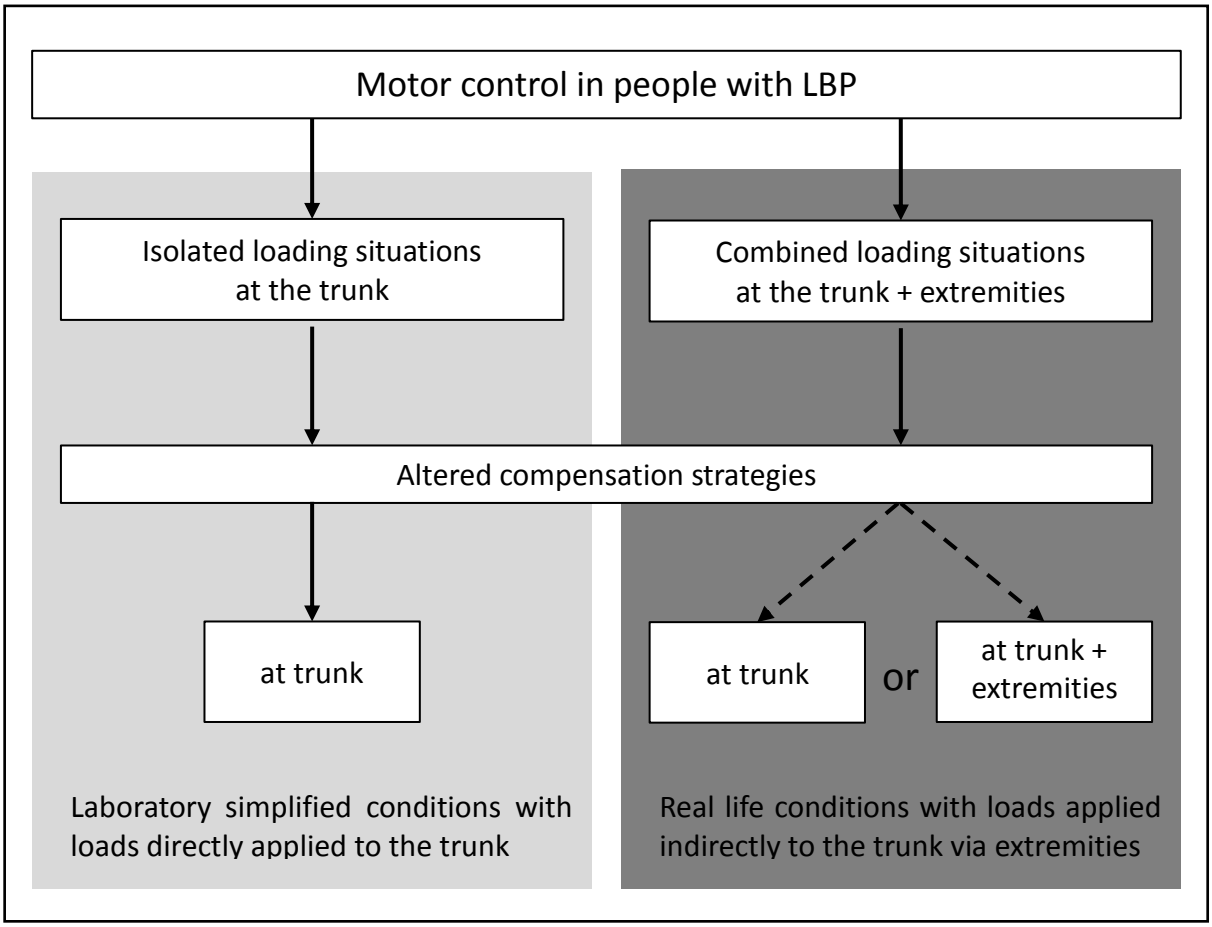

Figure 5: Load compensation strategies in simplified laboratory conditions with loads directly applied at the trunk vs. real life conditions with loads applied indirectly to the trunk via extremities

This research project aims to investigate motor control strategies at the trunk and lower extremities following unexpected disturbances under dynamic conditions resembling real life circumstances. Therefore, muscular response strategies are evaluated following sudden perturbations applied during walking to cause trip-like events. Walking was chosen, as it represents an everyday motor task for the individuals which is quite stable over time and enables the investigation of muscular strategies repeatedly under similar dynamic

conditions (Terrier & Dériaz, 2011). Moreover, walking requires constant dynamic adjustments (Massion, 1992; Iosa et al., 2015) and thereby might in particular be suited to discover impairments of motor control in consequence of sudden loading incidents. Previous investigations often used experimentally induced stumbling to evaluate muscular responses of the lower extremities (Granacher et al., 2006; Forner Cordero et al., 2003; Sessoms et al., 2014). Few studies applied this approach to quantify muscular responses following the stumbling incident at the trunk, using for example movable plates or sudden obstacles dropped on the walk way (van der Burg et al., 2005; Tang et al., 1998).

In the present research project, walking perturbations are intended to be provoked by sudden treadmill speed alterations. Therefore, a new setup has to be developed, with belt perturbations intense enough to provoke detectable muscular responses at the trunk, despite being indirectly transferred via the lower extremities. At the same time, perturbations need to be explicitly short to avoid mechanical influences on subsequent muscular activities (Sloot et al., 2015). The technical feasibility of the protocol has been confirmed prior to this project (Engel et al., 2013), however data on validity and reliability regarding timing and magnitude of muscular responses at the trunk and lower extremities are lacking. Hence, the suitability of the developed protocol to provoke muscular reflex responses following walking perturbations, as well as day-to-day reliability of their assessment, is addressed in a first investigation (Study 1).

In a second investigation (Study 2) motor control strategies in response to sudden perturbations of the established protocol will be analyzed between people with LBP and asymptomatic controls (CTRL). In accordance with more recent concepts of motor adaptation to pain, it is hypothesized that pain may have profound consequences on motor control strategies in people with LBP. Especially in situations where trunk function is embedded in comprehensive motor tasks, new strategies may involve the reorganization of muscular activity exceeding the area of the trunk. It will be determined whether changes in muscular response strategies in timing and level of activity are limited to the painful area (trunk) or being also present in remote areas (lower extremities).

2.1 Research questions

In agreement with the research paradigm described previously, the following main research questions and their respective sub questions are raised:

Research question RQ1 (sub questions RQ1a, RQ1b)

Does perturbed treadmill walking represent a suitable testing situation to provoke muscular reflex responses at the trunk and lower extremities?

a) *Does the stumbling incident lead to detectable onsets of muscular activity at the trunk and lower extremities representative of reflex responses, quantified by the latency of bursts in electromyographic signal activity?*

b) *Does the stumbling incident lead to a detectable increase of muscular activity at the trunk and lower extremities, quantified by the amplitude of electromyographic activity within a time window of reflex responses?*

Research question RQ2 (sub questions RQ2a, RQ2b)

Does perturbed treadmill walking allow a reliable assessment of provoked muscular activity responses at the trunk and lower extremities on different days?

a) *How reliably can muscular activity levels (EMG amplitudes) at the trunk and lower extremities in response to treadmill walking perturbations be assessed on different days?*

b) *How reliably can muscular activity onsets (EMG latencies) at the trunk and lower extremities in response to treadmill walking perturbations be assessed on different days?*

Research question RQ3 (sub questions RQ3a, RQ3b, RQ3c)

Do people with LBP show an altered muscular compensation strategy in response to perturbed treadmill walking in comparison to CTRLs, either locally at the painful area of the trunk or also remotely at the lower extremities?

a) *Do people with LBP show altered latencies of muscular activity onsets at the trunk in response to the stumbling incident applied at the lower extremities?*

b) *Do people with LBP show altered levels of muscular activities at the trunk in response to the stumbling incident applied at the lower extremities?*

c) *Do people with LBP deploy altered compensation strategies (activity onset and/or levels of activity) restricted to the area of pain (trunk) or exceeding the area (lower extremities) in order to compensate the stumbling incident applied at the lower extremities?*

3 MATERIAL AND METHODS

Two independent studies were conducted in succession to answer the proposed research questions (figure 6). The first study (Study 1: Validation of the testing situation) served to assess muscular responses following walking perturbations in a test-retest design, conducted in a cohort of asymptomatic participants. Suitability of the developed protocol to provoke muscular reflex responses following walking perturbations (RQ1), as well as day-to-day reliability of their assessment (RQ2) were addressed in this investigation. Following the refinement of the testing protocol, a second study (Study 2: Differences in activation strategies between low back pain and controls) was conducted to apply the investigations in a cross-sectional study design. This investigation assessed differences in muscular activation strategies at the trunk and lower extremities following walking perturbations between LBP and CTRL (RQ3).

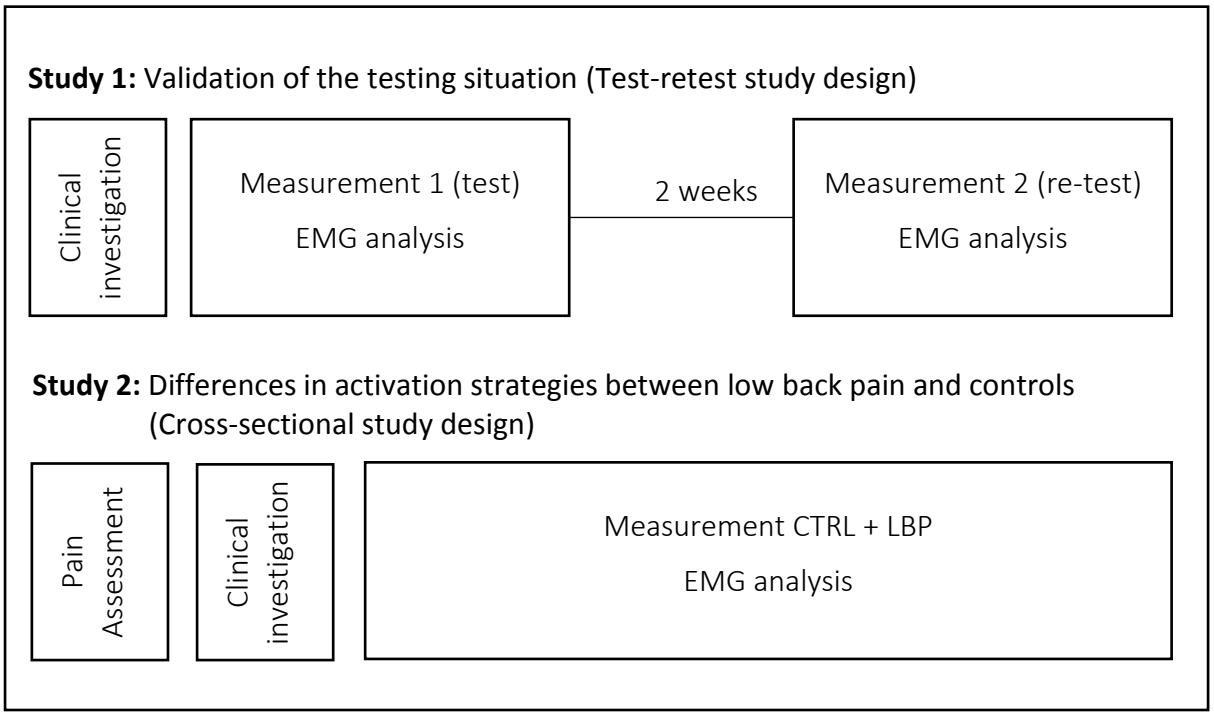

Figure 6: General structure of the research project and study designs of the two empirical investigations (CTRL: asymptomatic controls; LBP: low back pain)

3.1 Treadmill walking perturbations

All investigations (Study 1 and Study 2) were performed on an instrumented split-belt treadmill (Woodway® GmbH, Weil am Rhein, Germany; max. acceleration: 40 m/s^2). This

treadmill, powered by two separate electric engines, was used to generate rapid impulses of velocity alterations of each belt independently. Baseline velocity of the belts (1 m/s) and superimposed impulses of velocity alterations were controlled by a custom software solution (stimuli, pfitec biomedical systems, Endingen, Germany). Applied perturbation stimuli consisted of rapid velocity decreases with amplitudes of 2 m/s, resulting in a reverse of movement direction of baseline velocity (figure 7). Total duration of the stimuli was preset to 100 ms (50 ms deceleration, 50 ms acceleration). The Perturbation trigger was given by a plantar pressure measurement insole (Pedar® X, Novel GmbH, Muenchen, Germany; sampling rate 50 Hz; threshold load 40 kPa) inside the right shoe (standardized footwear; Nike®, Air Pegasus, 2002; figure 8). Right sided perturbations were applied with a delay of 200 ms after initial heel contact of the right foot to be released during mid-stance phase of walking (Winter & Yack, 1987). Left sided perturbations served only to avoid unidirectional gait changes and were not used for later analysis. Therefore, left sided perturbations were triggered indirectly by the signal of the right sided heel contact with an additional time delay (stride length) which was measured during warm up trials (3D motion capture system, Vicon MX3, 8 cameras, 200 Hz, Vicon, Oxford, UK). This technique allowed an approximation of left sided mid-stance phases during walking.

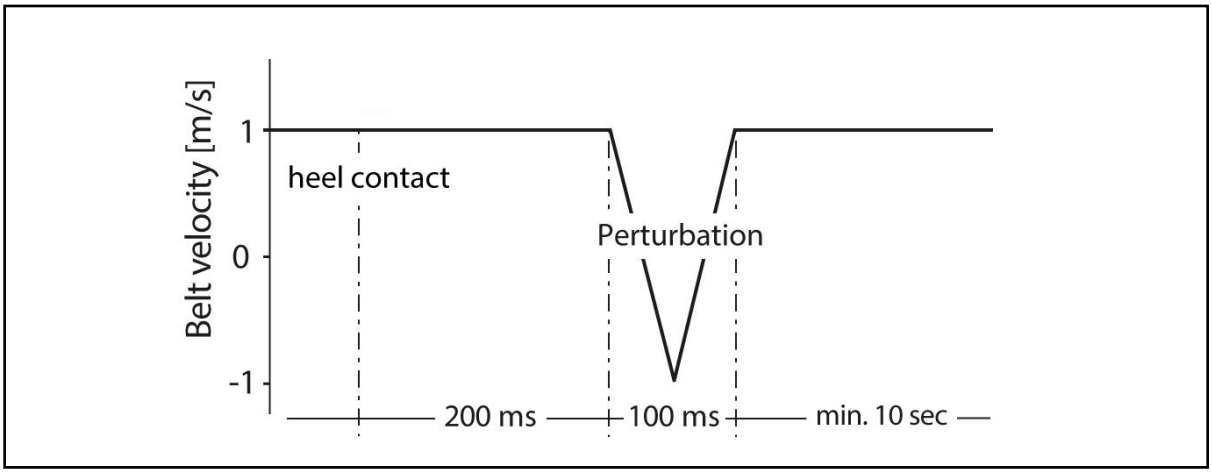

Figure 7: Characteristics of perturbation stimuli (100 ms duration; 2 m/s amplitude) applied during treadmill walking at 1m/s baseline velocity.

Left and right sided belt perturbations were applied in random time intervals, with a refractory period of at least 10 seconds (several strides) between stimuli to ensure regain of normal walking pattern for the subsequent perturbation (Forner Cordero et al., 2003).

Wireless acceleration sensors (ACC) measuring linear acceleration in three dimensions (Myon 320s, myon AG, Switzerland) served to detect time points of heel contact and time points of applied perturbation stimuli during walking. Acceleration data of ACC sensors attached at the back side of the shoes were captured synchronously to the EMG signal using the same wireless transmission system. This approach ensured identical transmission latencies (14 ms) of both EMG and ACC data and therefore allowed a precise identification of time differences between perturbation and EMG responses during later analysis. Technical validity and reliability of this new perturbation setup were investigated in a pilot study preceding this research project (Engel et al., 2013). Results of this study indicated a high accuracy between observed and pre-defined perturbation characteristics (timing, amplitude and duration) as well as a high reliability of the repeated application of the protocol.

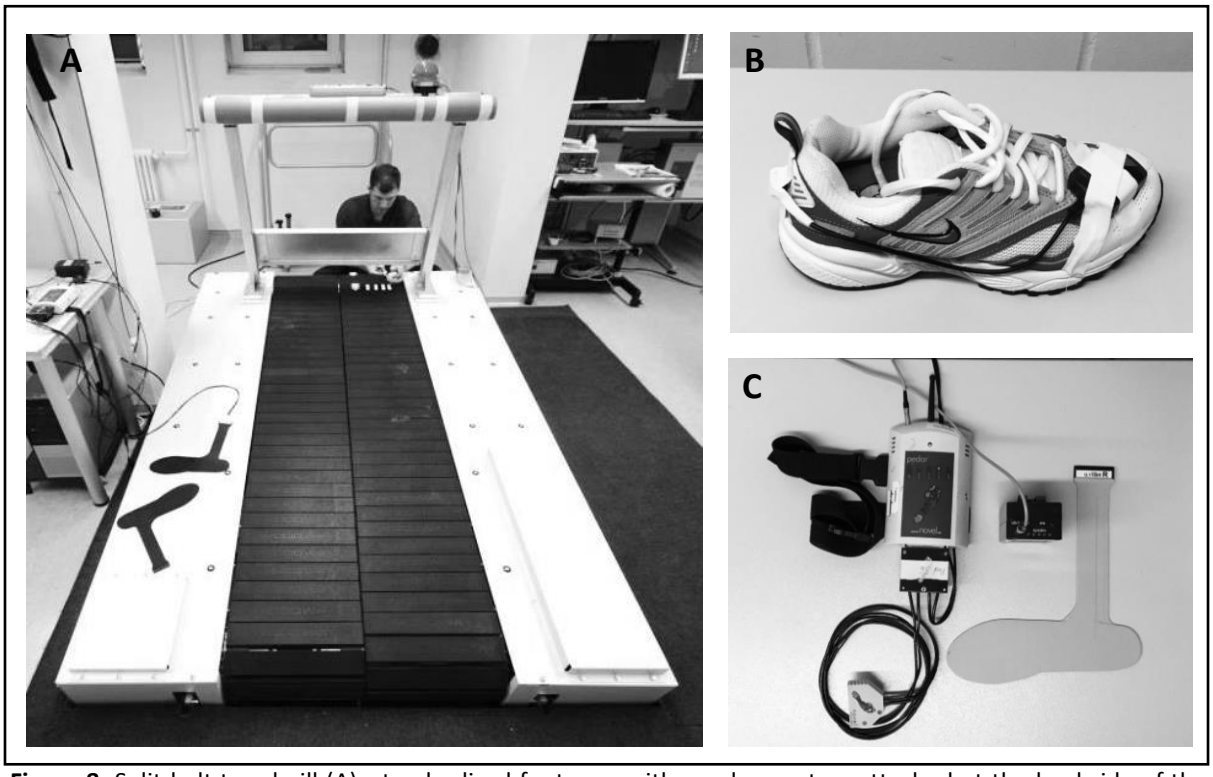

Figure 8: Split-belt treadmill (A), standardized footwear with accelerometers attached at the heel side of the shoe(B) and plantar pressure insole for detection of heel contact (C)

3.2 Electromyography (EMG)

Muscular activities of the trunk and lower extremities were assessed by surface electromyography (EMG) during all investigations (figure 9). EMG signals of the trunk were

recorded from 12 major trunk muscles (right/left): M. rectus abdominis (RRA/LRA), M. externus obliquus (REO/LEO), M. internus obliquus (RIO/LIO), M. latissimus dorsi (RLD/LLD) and M. erector spinae thoracic (REST/LEST) and lumbar (RESL/LESL)(Radebold et al., 2000). EMG signals of the lower extremities were recorded from 10 leg muscles (right/left): M. vastus medialis (RVM/LVM), M. biceps femoris (RBF/LBF), M. gastrocnemius medialis (RGM/LGM), M. peroneus longus (RPL/LPL) and M. tibialis anterior (RTA/LTA).

Table 1: Location of EMG electrodes at the 12 trunk and 10 legs muscles

Muscle	**Electrodes localization**
M. rectus abdominis	5 cm lateral to the umbilicus – oriented rostral-caudally
M. externus obliquus	15 cm lateral to the umbilicus – oriented rostral-caudally
M. internus obliquus	midway along the line between anterior-superior iliac spine and the symphysis pubis, above the inguinal ligament
M. latissimus dorsi	Lateral to the 9th thoracic segment – inferior to the scapula over the muscle belly when the arm was positioned in the shoulder mid-range
M. erectus spinae (thoracic)	At the 9th thoracic spine segment, 5 cm lateral to the thoracic segment – oriented rostral-caudally
M. erectus spinae (lumbar)	At the 3rd lumbar spine segment, 3 cm lateral to the lumbar segment – oriented rostral-caudally
M. vastus medialis	At 4/5 on the line between the anterior spina iliaca superior and the joint space in front of the anterior border of the medial ligament
M. biceps femoris	At 1/2 way on the line between the ischial tuberosity and the lateral epicondyle of the tibia
M. gastrocnemius medialis	Over the area of greatest muscle bulk on the medial calf – oriented rostral-caudally
M. peroneus longus	Midway along the line between the head of the fibula and the lateral malleolus, (in a more proximal position (1/4 of distance)
M. tibialis anterior	Over the area of greatest muscle bulk lateral to the crest of the tibia on the proximal half of the leg

Bipolar EMG electrodes (pre-gelled (Ag/AgCl); Ambu, Medicotest, Denmark, type P-00-S) were placed on the skin above the respective muscles. Skin areas selected for electrode placement were shaved, sandpapered and cleaned with alcohol to reduce skin impedance

below 5 kΩ (Hogrel et al., 1998). Localizations of EMG electrodes were determined according to Radebold et al. (2000), Winter and Yak (1987) and the SENIAM guidelines (2000) as shown in table 1. Surface electrode pairs were positioned with a constant inter-electrode distance of 2 cm and longitudinal axes of the electrodes were in line with the presumed direction of the underlying muscle fibers.

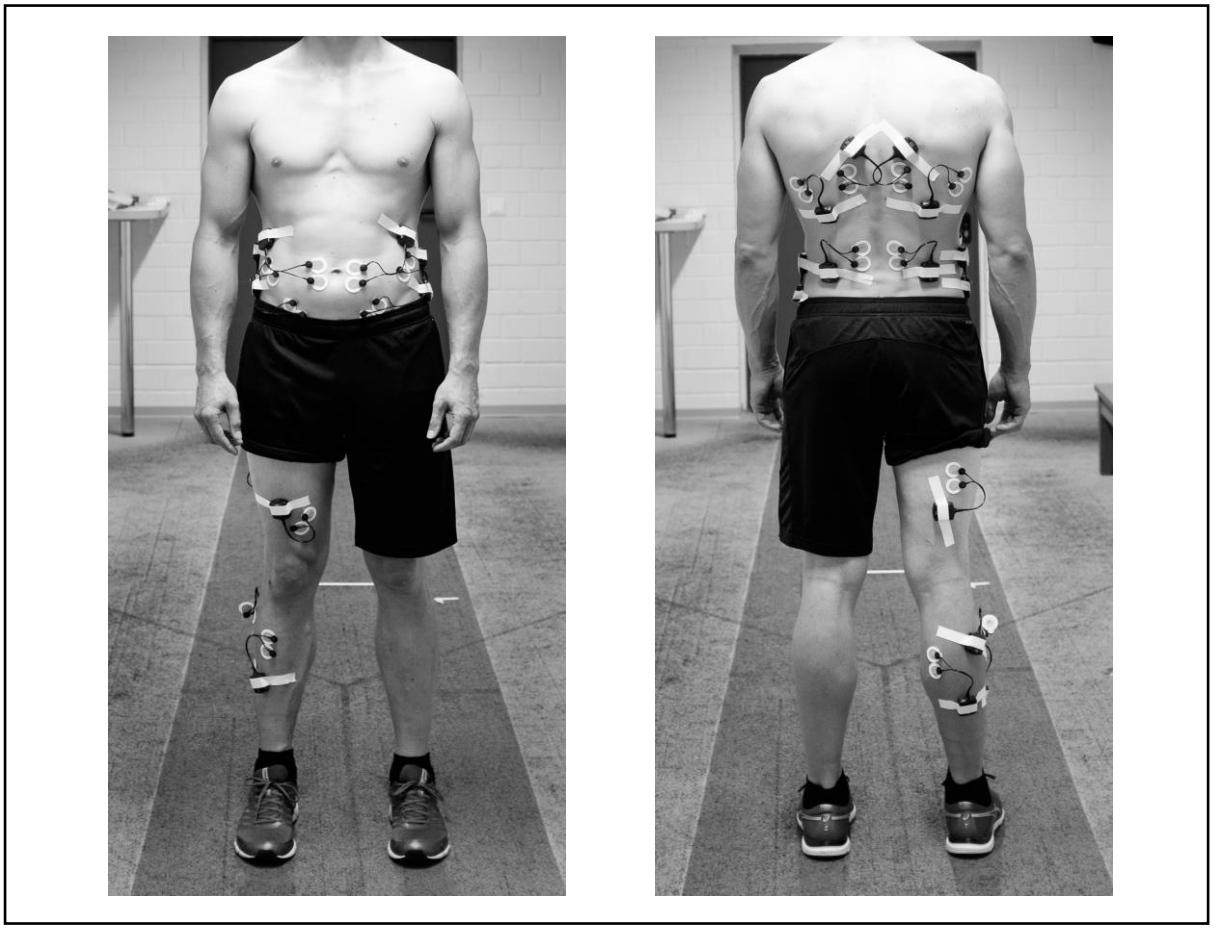

Figure 9: EMG Setup for trunk and leg muscles; electrodes placed according to SENIAM guidelines; wireless transmitters connected via short cables attached at the skin by sticky tape

A wireless EMG capture system (Myon 320, RFTD-32, sampling frequency 4000Hz, myon AG, Switzerland) was used to record muscular activities during perturbed treadmill walking. Wireless transmitters (m320TXA) were placed at the skin and connected to the EMG electrodes by short cables forwarding the signal to a central receiver unit (m320RX, bandwidth: 5-500 Hz, butterworth filter 4^{th} order, digitized). Finally, signals were A/D-converted (NI PCI 6229, 250 kS/s, 16-Bit, National Instruments®, Austin, TX, USA) and stored on a personal computer (IMAGO record master, LabView®-based, pfitec, biomedical systems, Endingen, Germany).

Post processing of the EMG and ACC data was done using a customized software solution (IMAGO process master, LabView®-based, pfitec, biomedical systems, Endingen, Germany). Events of heel contact and onsets of perturbation detected by the ACC sensors attached at the shoes were used for trigger generation prior to EMG signal treatment. Only right sided perturbations and respective heel contacts were triggered for later analysis. In addition, also the last 5 steps prior to the first applied perturbation were triggered by heel contacts of the right foot for normalization of amplitude investigations. Event triggers of the ACC signal were placed manually by visual inspection of the recordings. Clear acceleration characteristics for both heel contact and perturbation onset allowed for a precise event determination (see figure 10 for an example of visual event detection).

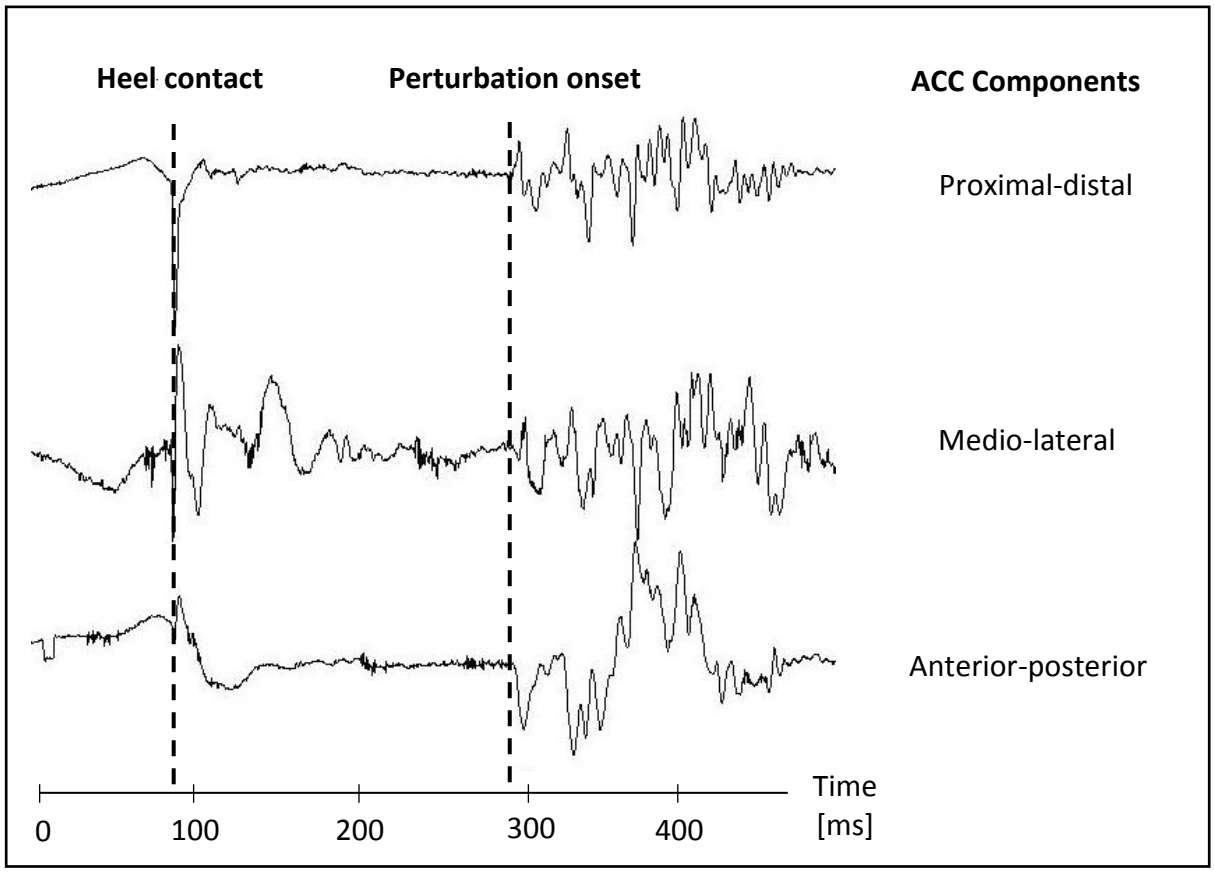

Figure 10: Example of an acceleration signal (ACC) attached at the right foot for detection of initial heel contact and perturbation onset

Subsequent EMG data treatment was dependent on whether it was part of the analysis in the amplitude or time domain. For latency investigations, a semi-automated detection method (IMAGO process master, LabView®-based, pfitec, biomedical systems, Endingen,

Germany) was used (figure 11 A). A rise of the averaged EMG signal of all perturbations (ensemble average; filter: 4th order moving average) above 2 standard deviations from baseline level was defined as the criteria for automatic onset detection for each muscle individually (Baur et al., 2010; O'Connell et al., 2016). Visual inspection of the automatically detected onsets was used to manually set the time point of activity onset where automatic detection failed or was not considered plausible due to signal artefacts. This semi-automated procedure is considered to secure both high standardization and validity of onset detection(Hodges & Bui, 1996).

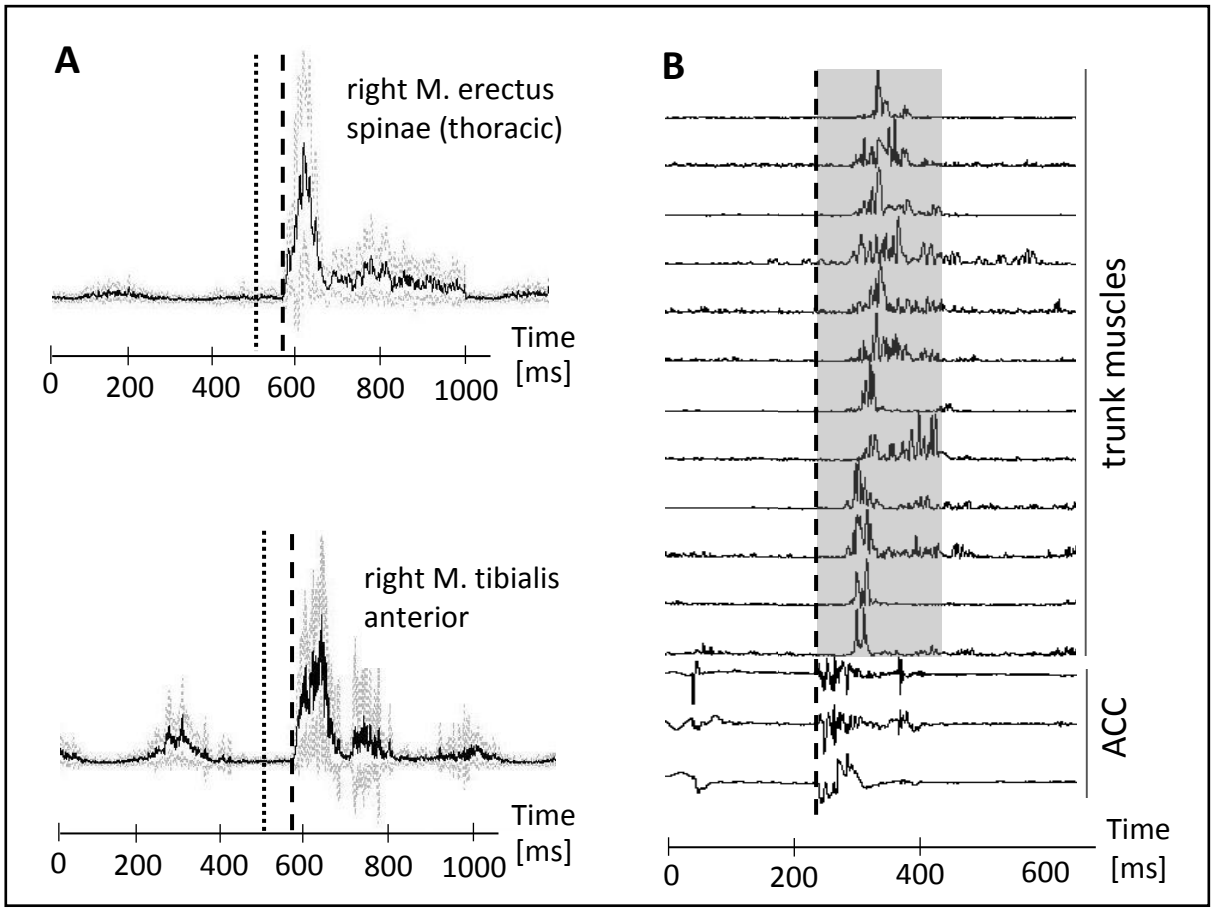

Figure 11: (A) Example of the ensemble average signal (15 perturbations) of two exemplary muscles, showing the mean signal (black lines) with standard deviations (grey lines) and automatically detected onset of muscle activity (dashed line) following the rise of EMG activity above 2SD of the silence period (dotted line); (B) Example of an EMG recording for amplitude investigations, showing the rectified signal of trunk muscles for a single perturbation (greyed area indicates the time window of 200 ms)

For amplitude investigations, EMG signals were rectified and the root mean square (RMS) amplitude of individual muscles was calculated over a time window of 200 ms after the onset of perturbations (figure 11 B). This time window was used to cover neuromuscular

responses comprising mono- and polysynaptic reflex activities following the perturbation stimulus (de Freitas et al., 2010; Oliveira et al., 2012; Taube et al., 2007). RMS amplitudes of the unperturbed stride cycles prior to any perturbation were used to normalize the EMG amplitude output at perturbed strides (Granacher et al., 2010).

Latency and amplitude investigations were performed for each of the assessed muscles individually, as well as for pooled data of muscles grouped by location and function (figure 12). Trunk muscles were grouped into four quadrants of the torso: ventral right (RRA, REO, LEO), ventral left (LRA, LEO, LIO), dorsal right (RLD, REST, RESL) and dorsal left (LLD, LEST, LESL) as previously reported in literature (McGill et al., 2013). Leg muscles were grouped into upper leg right (RVM, RBF), upper leg left (LVM, LBF), lower leg right (RGM, RPL, RTA) and lower leg left (LGM, LPL, LTA), in order to distinguish muscular reactions by their distance to the area of perturbation (Study 2: only right sided leg muscles were assessed).

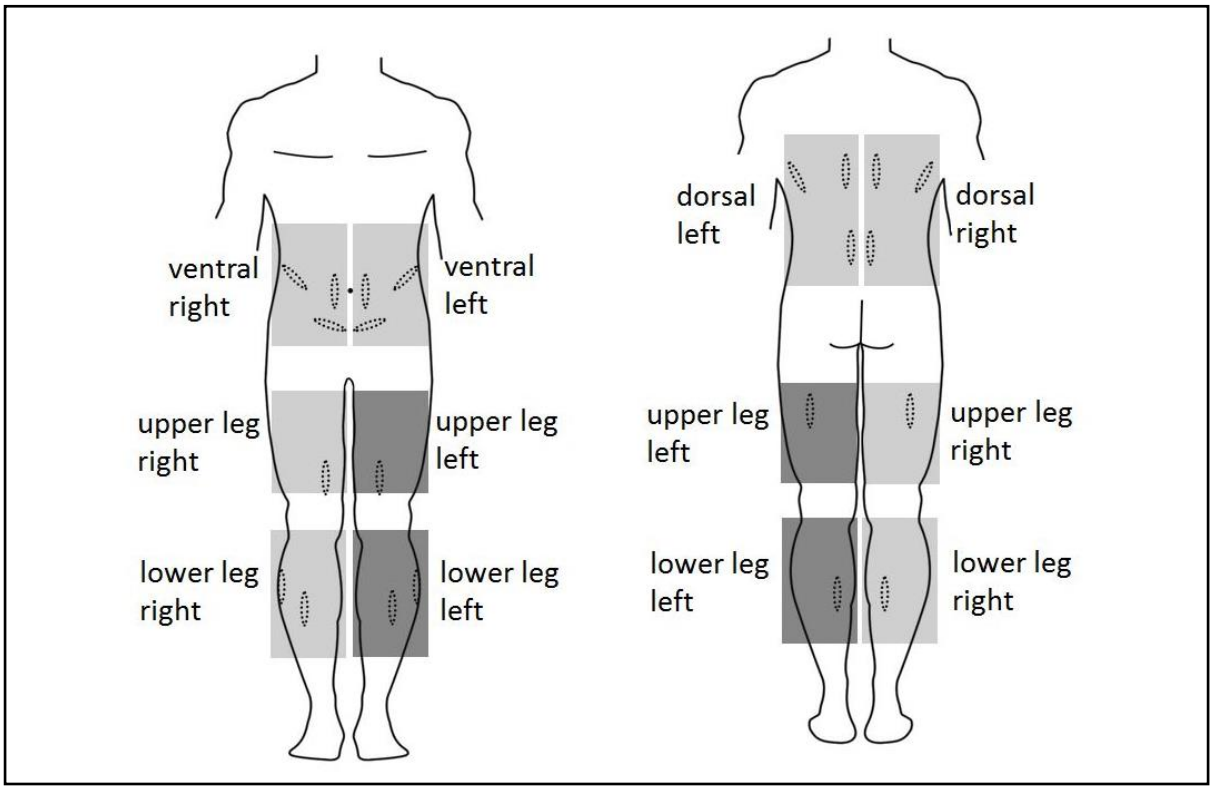

Figure 12: Muscles grouped according to location into ventral right/left, dorsal right/left, upper leg right/left and lower leg right/left; upper leg left and lower leg left (dark grey filled areas) were only assed in study one (Validation of the testing situation)

Muscle co-contraction pattern of selected muscle pairs were additionally compared between LBP and CTRL within the population of “Study Two”. Interactions between muscle pairs were analyzed by ratios of RMS% signal (amplitudes) and by differences of muscle

onsets following perturbation (latencies). Activity pattern were therefore compared (A) between right and left sided trunk muscles (ventral and dorsal), (B) within ventral muscles and dorsal muscles, (C) between ventral and dorsal muscles and (D) between trunk muscles (ventral and dorsal) and lower leg muscles (figure 13).

$$\text{RMS\% ratio} = \frac{\text{Muscle 1 [RMS\%]}}{\text{Muscle 2 [RMS\%]}} \; ; \; \text{Onset differences} = \text{Muscle 1 [ms]} - \text{Muscle 2 [ms]}$$

		Muscle 1	Muscle 2
(A)	right vs. left ventral	right M. rectus abdominis (RRA)	left M. rectus abdominis (LRA)
	right vs. left dorsal	right M. erector spinae lumbar (RESL)	left M. erector spinae lumbar (LESL)
(B)	ventral muscles	M. rectus abdominis (RA)	M. externus obliquus (EO)
	dorsal muscles	M. erector spinae thoracic (EST)	M. erector spinae lumbar (ESL)
(C)	ventral vs. dorsal	M. rectus abdominis (RA)	M. erector spinae lumbar (ESL)
(D)	ventral vs. lower leg	M. rectus abdominis (RA)	M. tibialis anterior (TA)
	dorsal vs. lower leg	M. erector spinae lumbar (ESL)	M. tibialis anterior (TA)

Figure 13: selected muscle pairs and calculation formula for muscle co-contraction pattern following perturbation stimulus; averaged signal of right and left sided abdominal and back muscles for condition (B), (C) and (D).

3.3 Pain assessment

Back pain condition was assessed using the German version of “The Chronic Pain Grade” questionnaire (CPG) by Von Korff (Von Korff et al., 1992; Klasen et al., 2004). This brief questionnaire is based on 7 questions addressing characteristic pain intensity and pain related disability in the context of back pain. (Original and German translated questions can be found in the appendix.) Respondents are asked to answer each question by an 11-point numeric rating scale, ranging from 0-10. Resulting scores of each question are used to calculate the following 3 subscales: A) the characteristic pain intensity score (CPIS; 0-100), which represents the mean intensity ratings reported for current, worst and average pain over the period of the last three months (mean of the 3 items x 10); B) the disability score (0-100), which is based on the mean ratings for the difficulty to perform daily, social, and work activities (mean of the 3 items x 10) and C) the disability points score (0–3), which is

derived from a combination of ranked categories of the number of disability days and the disability score (Von Korff et al., 1992). Finally all subscale scores are combined to calculate a chronic pain grade (CPG) that enables a classification of the respondents into five hierarchical categories from grade 0 to grade 4 (table 2; for detailed calculations see appendix).

Table 2: CPG classification with grade 0-4 based on pain intensity and disability points

Grade	Characteristics	
0	Pain free	No pain problem
1	Low disability – low intensity	Characteristic Pain Intensity less than 50, and less than 3 Disability Points
2	Low disability – high intensity	Characteristic Pain Intensity of 50 or greater, and less than 3 Disability Points
3	high disability – moderately limiting	3-4 Disability Points, regardless of Characteristic Pain Intensity
4	high disability – severely limiting	5-6 Disability Points, regardless of Characteristic Pain Intensity

Lower grades (1 and 2) are characterized by low disability and low to high pain intensity, whereas higher grades (3 and 4) are characterized by moderately to severely limiting disability, in disregard of pain intensity. This hierarchical relationship between pain intensity and disability was proposed to allow a better discrimination within higher levels of pain severity, where pain intensities alone may not be sufficient enough (Von Korff et al., 1992).

In the present research project two different categorization strategies were used for allocating individuals into pain group (LBP) and asymptomatic controls (CTRL) (figure 14). In a first analysis, chronic pain grades (0-4) served as the allocation criteria, with participants being classified as CTRL at grade 0 and as LBP at grade 2 and above. This allocation scheme was chosen to distinguish muscular reflex responses between people being free of any pain within the last three months and people suffering from high pain intensity and moderate to severely limiting disability related to low back pain. However, this strategy did not account for participants falling into chronic pain grade 1, a potentially quite heterogeneous group of people reporting pain intensities varying between 1 and 49 points (of 100 max) on the characteristic pain intensity score (CPIS). It was therefore hypothesized that CPG

classification may not be sensitive enough for the present research project to allow a differentiated group allocation based on pain intensity. Moreover, with regards to the inclusion criteria, it was anticipated that the majority of participants of the study population would fall in a range of mild to medium levels of pain and only minor levels of pain related disabilities.

Therefore, a second analysis was performed using reported characteristic pain intensity scores (CPIS; 0-100) exclusively. CTRL group allocation was based on CPIS below or equal to 10 points, whereas LBP groups were defined by CPIS of 30 points or above. Definition of pain threshold for CTRL (≤10% of maximum score) was derived by levels used for CTRL allocations in previous studies using a visual analogue scale (VAS) (Nelson-Wong & Callaghan, 2010; Müller et al., 2014; Callaghan & Nelson-wong, 2013). The definition of pain threshold for LBP was chosen in accordance with previous classifications of LBP, where this threshold was described as a transition point from mild to moderate pain intensities (Cedraschi et al., 1999; Von Korff & Miglioretti, 2005).

Group allocation by chronic pain grade (CPG, grade 0 - 4)

CTRL: Grade = 0	absence of pain and pain related disabilities
LBP: Grade ≥ 2	high pain intensity and low to high disability

Group allocation by characteristic pain intensity scores (CPIS, score 0 - 100)

CTRL: Score ≤ 10	pain free to low level of chronic pain
LBP: Score ≥ 30	moderate to high levels of chronic pain

Figure 14: Group allocation into CTRL (control) and low back pain (LBP) according to chronic pain grade (CPG) and characteristic pain intensity scores (CPIS)

3.4 Methods - Validity and reliability of the testing situation

3.4.1 Participants

Fourteen asymptomatic volunteers (table 3) were recruited for study participation. All participants were supposed to meet the following inclusion criteria: (1) being physically active two times per week at minimum, (2) age of 18-50 years, (3) no pain/discomfort at the musculoskeletal system at the time point of the study. Exclusion criteria were defined as: (1)

pain/discomfort at the musculoskeletal system, (2) acute or chronic infection. All participants underwent a clinical investigation by a physician, to rule out underlying pathologies at the trunk and lower extremities. Written informed consent was provided by all volunteers after being fully informed about the respective test procedure. The ethics committee of the local university approved the study.

Table 3: Study 1 - Participants' anthropometrics

Gender	Age [years]	Weight [kg]	Height [cm]
8 M , 6 F	27 ± 3	76 ± 13	179 ± 10

Age and anthropometrics of participants (males (M) and females (F)); data are given as mean ± SD.

3.4.2 Test protocol

All participants underwent two identical testing situations (M1 and M2), with two weeks in between each testing day (figure 15). Being classified as eligible (inclusion criteria; clinical examination) for study participation, volunteers were prepared for the data acquisition of EMG at 12 muscles at the trunk (RRA, LRA, REO, LEO, RIO, LIO, RLD, LLD, REST, LEST, RESL, LESL) and 10 muscles at the lower extremities (RVM, LVM, RBF, LBF, RGM, LGM, RPL, LPL, RTA, LTA). Standardized foot wear was provided with a plantar pressure insole placed inside for stride detection, accelerometers (ACC) were mounted at the back side of the shoes. For safety reasons, a chest harness system connected to an emergency stop of the treadmill was provided. Prior to the stumbling protocol, participants were accustomed to walking on the spilt-belt treadmill at 1 m/s for a warm-up period of 5 min, without the occurrence of perturbations. The subsequent test protocol consisted of 8 minutes walking with a total of 10 perturbations (5 x at left and 5 x at right foot), applied randomly over time and side.

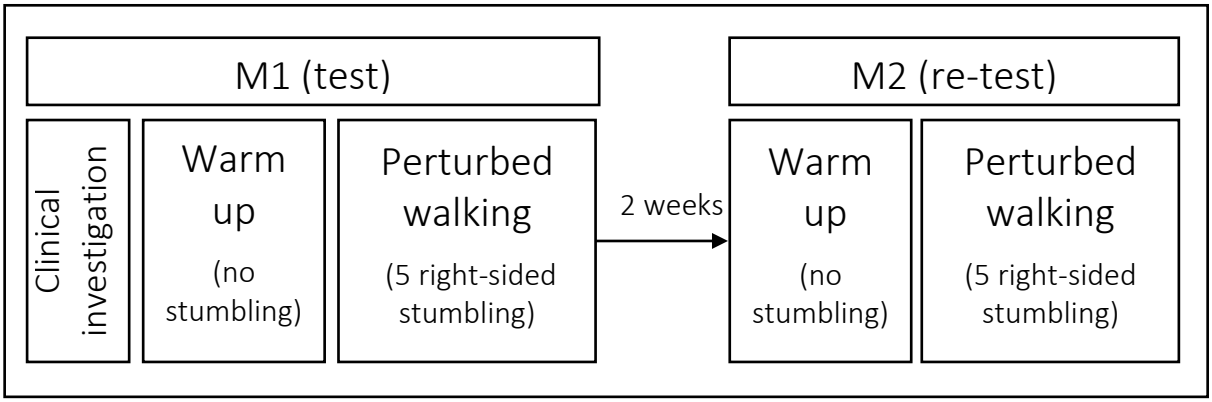

Figure 15: Validation of the testing situation (Test-retest study)

3.4.3 Statistics

All acquired data was transferred into a data matrix (Microsoft® excel 2010). Final outcome variables were tested for plausibility by range check within the final data matrix (Microsoft® excel 2010, JMP® statistical software Package Version 9.0, depending on data type). Outliers were compared with original data and if necessary corrected, recalculated or deleted. Data distributions were examined by Shapiro-Wilk tests and inspection of histograms for all investigations. Calculations of mean and standard deviation (SD) were used to summarize data descriptively, unless stated differently.

The level of reliability between measurements was determined using the intra-class correlation coefficient (ICC 2,1) and calculations of stand error of measurements (SEM: SD * (square root of (1-ICC) as estimate of the precision of measurement) (Shrout & Fleiss, 1979; Denegar & Ball, 1993). Within-subject variability was investigated by Bland-Altman analysis with calculation of bias (systematic error) and limits of agreement (bias ± 1.96*SD; LoA) (Bland & Altman, 1986; Hopkins, 2000). Relative differences between measurements were assessed by calculating test-retest variability (TRV [%]: (| xi - yi | / 0.5 (xi + yi) * 100), where xi represents the amplitude/latency values of M1 and yi are those of M2 for subject i) (König et al., 2012).

3.5 Methods - Differences in activation strategies between LBP and CTRL

3.5.1 Participants

Eighty five participants (table 4) were recruited out of the clinical routine assessment of the University Outpatient Clinic Potsdam, independent of absence or presence of LBP. As inclusion criteria all volunteers had to be aged between 18-50 years. Exclusion criteria were defined as: (1) acute or chronic infection, (2) pregnancy, (3) postural disabilities, (4) general dispositions, contraindicating the participation in physical activity. Back pain condition was assessed using the German version of “The Chronic Pain Grade” questionnaire (CPG) by Von Korff (Von Korff et al., 1992; Klasen et al., 2004). Furthermore, all participants underwent a clinical investigation by a physician to assess clinical state and to rule out contraindicated pathologies prior to study participation. Back pain grading (CPG scores) as well as sub scores

of pain intensity (characteristic pain intensity scale; CPIS) were used for allocation of participants into CTRL and LBP group. All participants provided written informed consent after being fully informed about the respective test procedure. The ethics committee of the local university approved the study.

Table 4: Study 2 - Participants' anthropometrics

Gender	Age [years]	Weight [kg]	Height [cm]
31 M, 54 F	29 ± 8	71 ± 13	174 ± 10

Age and anthropometrics of participants (males (M) and females (F)); data are given as mean ± SD.

3.5.2 Test Protocol

Participants were initially asked to complete the German version of "The Chronic Pain Grade" questionnaire (von Korff; chapter 3.3) on a computer (web based database, Pro WebDB®) followed by the clinical investigation (figure 16). Thereafter, participants were prepared for the data acquisition of EMG at 12 muscles at the trunk (RRA, LRA, REO, LEO, RIO, LIO, RLD, LLD, REST, LEST, RESL/LESL) and at 5 muscles at the right sided lower extremities (RVM, RBF, RGM, RPL, RTA). Standardized foot wear was provided with an insole placed inside for stride detection, accelerometers (ACC) were mounted at the back side of the shoes. A chest harness system connected to an emergency stop of the treadmill was provided for safety reasons. Prior to the stumbling protocol, participants were accustomed to walking on the spilt-belt treadmill at 1 m/s for a warm-up period of 5 min (unperturbed). The subsequent test protocol consisted of 10 minutes walking with a total of 30 perturbations (15 x at left and 15 x at right foot), applied randomly over time and side.

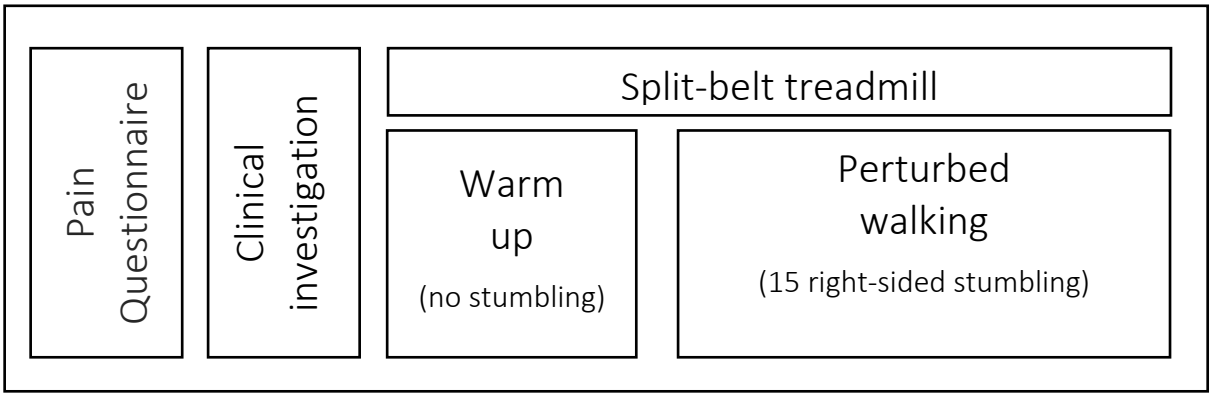

Figure 16: Differences in activation strategies between low back pain and controls (cross-sectional)

3.5.3 Statistics

All acquired data was transferred into a data matrix (Microsoft® excel 2010). Final outcome variables were tested for plausibility by range check within the final data matrix (Microsoft® excel 2010, JMP® statistical software Package Version 9.0). Outliers were compared with original data and if necessary corrected, recalculated or deleted. Data distributions were examined by Shapiro-Wilk tests and inspection of histograms for all investigations. Calculations of mean and standard deviation (SD) were used to summarize data descriptively, unless stated differently. Significant differences in anthropometrics between LBP and CTRL groups were tested by independent samples t-tests ($p<0.05$). Differences in muscular activities (amplitudes and latencies) between LBP and CTRL were analyzed descriptively with means and 95% confidence intervals for individual muscles (Altman & Gardner, 2000). Differences between LBP and CTRL based on grouped muscles (ventral left, ventral right, dorsal left, dorsal right, upper leg right, lower leg right) were statistically tested by a general linear model, using a multivariate analysis of variance (MANOVA, α =0.05; Pillai's trace test for differences between LBP and CTRL). When Pillai's trace test reached level of significance, post hoc comparisons were performed by independent samples t-tests ($p<0.01$) with Bonferroni corrections applied to account for family wise error rate. Alterations in muscle co-contraction pattern between LBP and CTRL, assessed by differences in delays of muscle onset [ms] and by ratios of RMS% amplitudes between selected muscles pairs, were tested by independent samples t-tests with Bonferroni correction applied to account for multiple testing ($p<0.01$). All statistical analyses were performed using SPSS® IBM Version 22, JMP® statistical software Package Version 9.0 and Microsoft® excel 2010.

4 RESULTS

4.1 Results - Validity and reliability of the testing situation

Recordings from 13 out of the 14 recruited participants were used for data analysis, after the exclusion of one individual due to inadequate EMG signal quality. Occasionally, single muscles had to be excluded from data analysis caused by EMG signal artefacts. Therefore, actual numbers of included participants are provided for each muscle separately in tables 5 to 9. No incidents of falls were caused by the applied walking perturbations. Visual inspection indicated kinematic reactions of the whole body following the stumbling stimuli.

4.1.1 Muscular responses following perturbations

Latency analysis of EMG activities revealed onset delays of 82 ms to 106 ms for trunk muscles and 75 ms to 117 ms for leg muscles following the perturbation stimulus. Muscles onset latencies of individual muscles following the perturbation are given in figure 17. Mean latency times resulted in 89 ms, both for trunk muscles and leg muscles. Muscle latencies could be retrieved from 515 of 572 onset events following perturbations. Automatic onset detection had to be manually corrected 78 times (15%) by visual inspection, due to signals artefacts or pre-activity of the respective muscle. Onset at right sided gastrocnemius medialis (RGM) could not be detected according to the defined onset criteria, due to an unclear/inconsistent EMG signal change post perturbation.

Amplitude analysis showed EMG increases (normalized to full strides of unperturbed walking) of 352% to 909% for trunk muscles and 106% to 718% for leg muscles within the time window of 200 ms following the perturbation. Amplitude responses of individual muscles at the trunk and lower extremities are shown in figure 18. EMG responses showed higher amplitudes (normalized to full strides of unperturbed walking) in trunk muscles compared to leg muscles (mean amplitudes: 530% for trunk muscles and 383% for leg muscles). Highest variability in amplitudes between individuals was found in abdominal trunk muscles (RRA, LRA, REO and LEO) and muscles at the upper legs (RVM, LVM, RBF and LBF) as shown in figure 18 by high standard deviations.

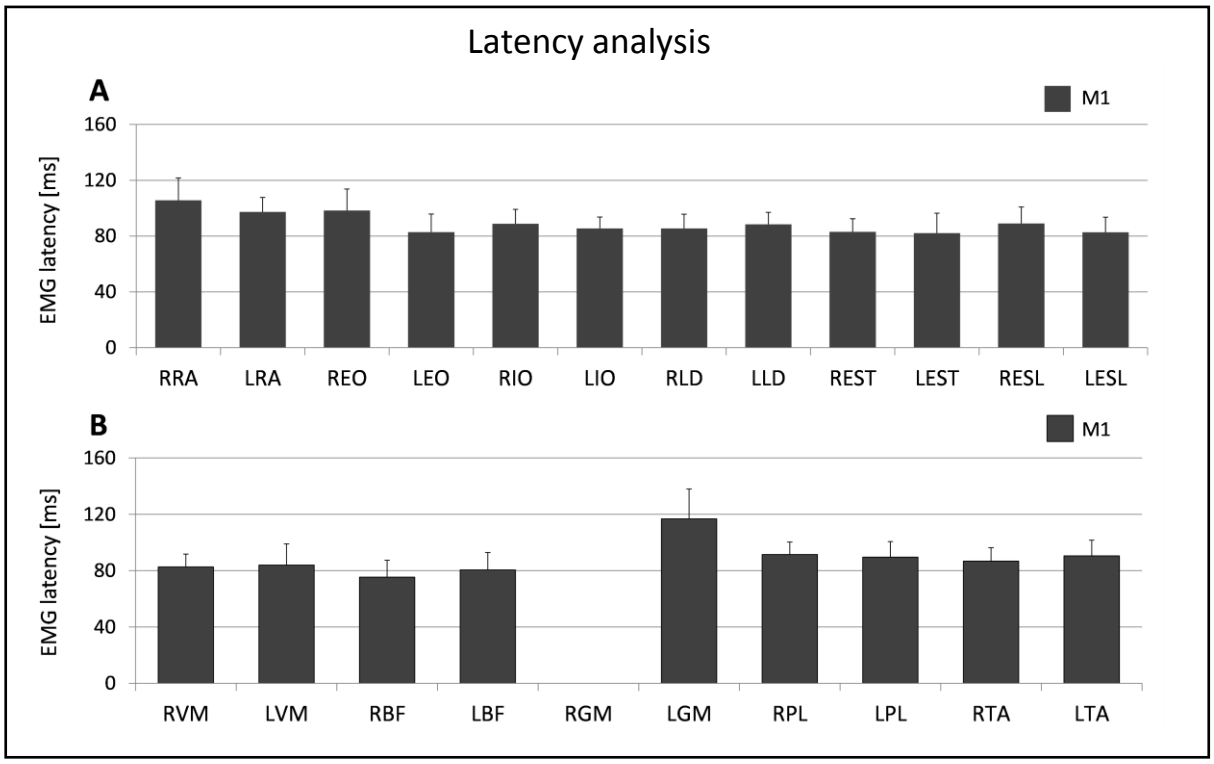

Figure 17: EMG latencies (ms) of M1 for trunk muscles (A) and leg muscles (B); data presented in means ±SD; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left M. peroneus longus

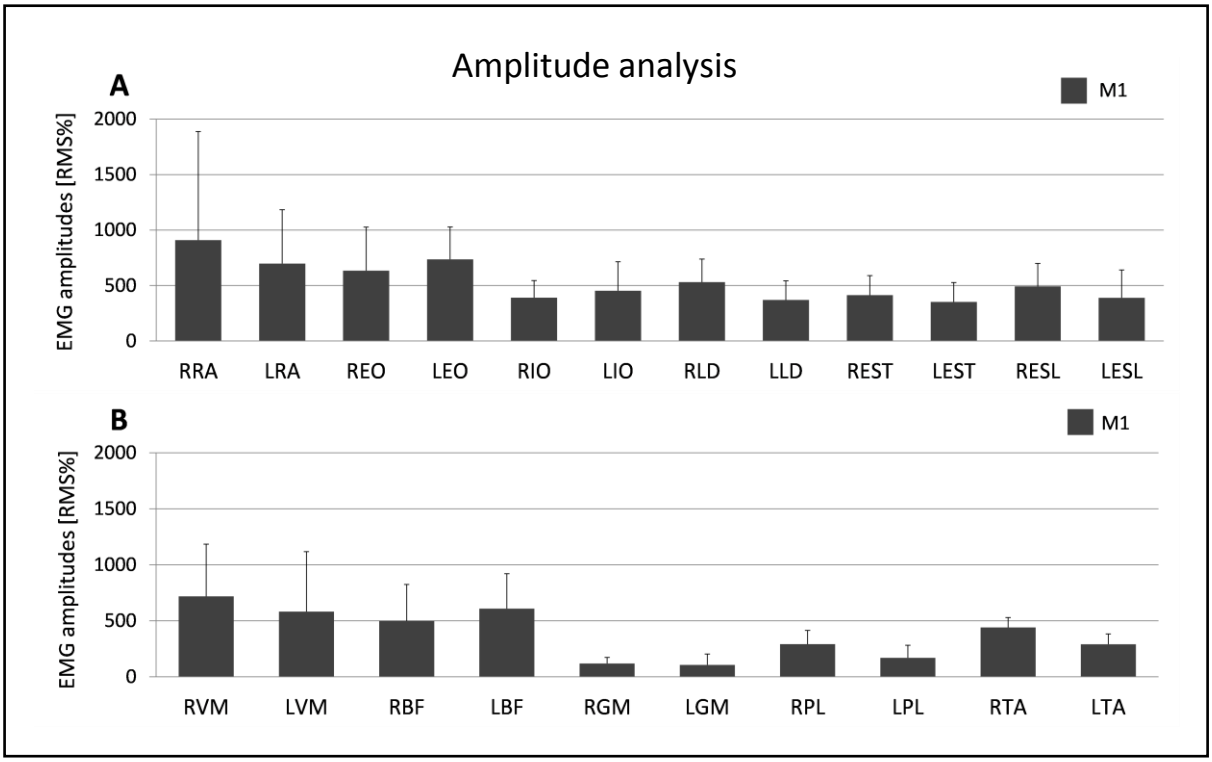

Figure 18: EMG amplitudes (%, normalized to full stride of unperturbed walking) of M1 for trunk muscles (A) and leg muscles (B); data presented in means ±SD; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left M. peroneus longus

4.1.2 Reliability of muscular activities following perturbations

Measures of reliability for latency investigations showed an ICC in mean of 0.71, ranging from 0.24 (RPL) to 0.92 (RRA) for all assessed muscles (table 5 and 6). Test-retest variability resulted in 5.0% (RRA) to 10.5% (LEO) for trunk muscles and 6.3% (RTA) to 13.4% (LBF) for leg muscles. SEM showed in mean 5 ms, ranging from 4 to 8 ms across all assessed muscles. Bland Altman analysis revealed a bias ranging from -7 ms to 4 ms. Figure 19 shows in more details differences between the two measurement days (bias) and 95% LoAs (1.96* SD) in a Bland Altman plot, highlighting exemplary characteristics of absolute reliability for single muscles at the trunk and lower extremities. Detailed results of all indicators of reliability between test day M1 and M2 are given in table 5 for trunk muscles and table 6 for muscles of the lower extremities.

Table 5: Indicators of reliability - latency analysis of trunk musculature

Muscle	N	Latency [ms]		ICC	SEM	TRV [%]	BA [ms]
		M1(mean ± SD)	M2(mean ± SD)	(95% CI)	[ms]	(mean ± SD)	(bias ± 1.96*SD)
RRA	8	106 ± 16	103± 15	0.92 (0.70 - 0.98)	4	5.0 ± 4.7	-3 ± 11
LRA	8	97 ± 10	101± 13	0.72 (0.31 - 0.97)	6	7.6 ± 3.9	4 ± 15
REO	12	98 ± 15	91± 16	0.86 (0.15 - 0.98)	6	7.9 ± 6.3	-7 ± 10
LEO	13	83 ± 13	86± 12	0.74 (0.18 - 0.92)	6	10.5 ± 7.4	3 ± 22
RIO	10	89 ± 10	85± 8	0.75 (0.43 - 0.96)	4	6.0 ± 4.2	-3 ± 11
LIO	8	85 ± 8	85± 9	0.58 (-0.46 - 0.92)	5	9.1 ± 6.4	-1 ± 18
RLD	10	86 ± 10	82± 8	0.67 (-0.14 - 0.92)	5	8.7 ± 7.1	-3 ± 16
LLD	11	88 ± 9	82± 11	0.63 (0.03 - 0.89)	6	10.0 ± 5.6	-6 ± 14
REST	11	83 ± 9	80± 8	0.50 (-0.28 - 0.87)	6	9.2 ± 7.9	-3 ± 19
LEST	11	82 ± 14	81± 12	0.79 (0.39 - 0.94)	6	8.2 ± 5.6	-1 ± 16
RESL	13	89 ± 12	86± 10	0.77 (0.28 - 0.93)	5	7.7 ± 7.2	-3 ± 18
LESL	13	83 ± 11	82± 9	0.77 (0.41 - 0.92)	5	6.0 ± 4.6	-1 ± 13

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left m. peroneus longus

Table 6: Indicators of reliability - latency analysis of leg musculature

Muscle	N	Latency [ms] M1(mean ± SD)	Latency [ms] M2(mean ± SD)	ICC (95% CI)	SEM [ms]	TRV [%] (mean ± SD)	BA [ms] (bias ± 1.96*SD)
RVM	12	83 ± 9	81± 9	0.73 (0.05 - 0.92)	4	7.9 ± 6.6	-2 ± 16
LVM	13	84 ± 15	86± 11	0.79 (0.32 - 0.94)	6	9.8 ± 8.9	2 ± 21
RBF	12	75 ± 12	69± 10	0.75 (0.12 - 0.93)	5	9.5 ± 9.8	-6 ± 16
LBF	10	81 ± 12	76± 15	0.67 (-0.12 - 0.84)	8	13.4 ± 13.5	-4 ± 25
RGM	-	-	-	- -	-	-	-
LGM	7	117 ± 21	109± 11	0.73 (0.10 - 0.95)	9	10.1 ± 7.1	-5 ± 23
RPL	8	91 ± 9	86± 7	0.24 (-0.47 - 0.72)	7	8.8 ± 7.7	-5 ± 19
LPL	11	90 ± 11	86± 9	0.79 (0.28 - 0.94)	4	7.6 ± 5.7	-4 ± 15
RTA	13	87 ± 9	85± 6	0.78 (0.31 - 0.93)	4	6.3 ± 4.4	-2 ± 13
LTA	12	91 ± 11	88 ± 11	0.71 (-0.15 - 0.88)	6	8.2 ± 7.4	-2 ± 20

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left m. peroneus longus

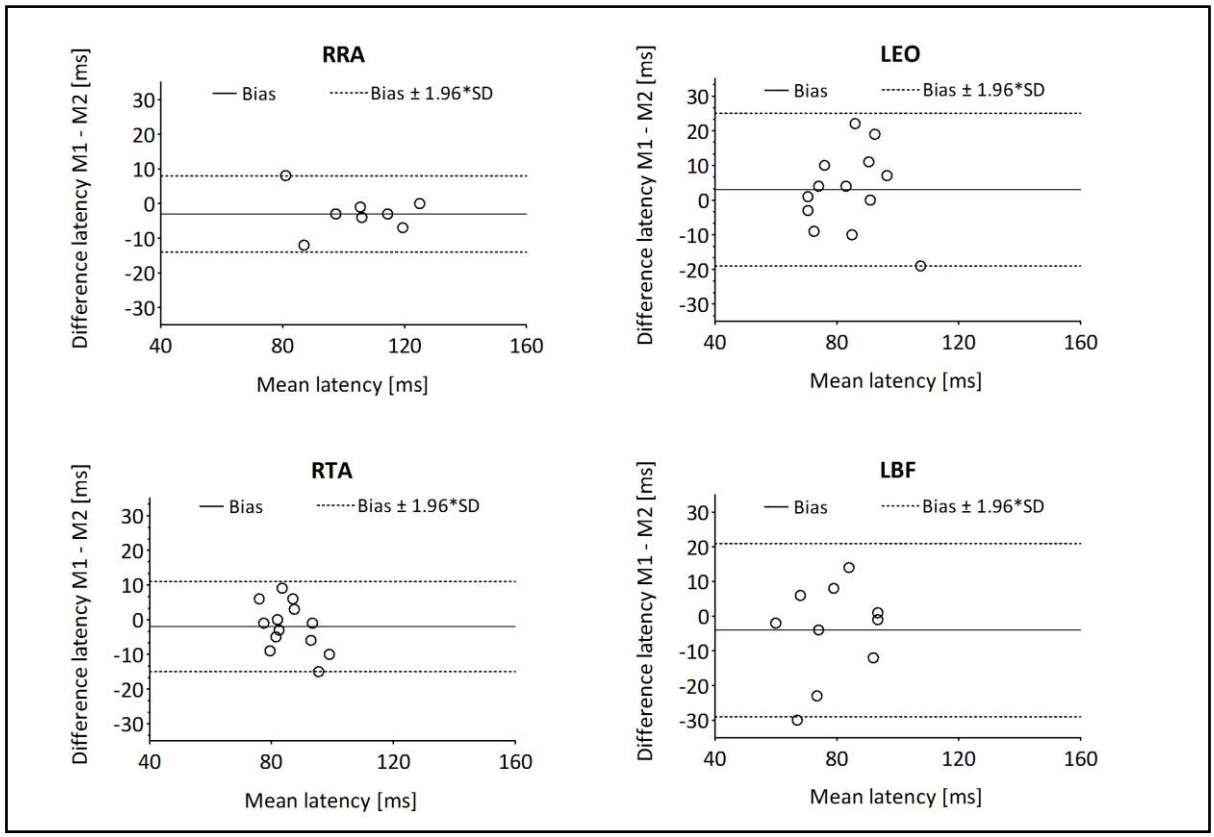

Figure 19: Bland Altman plots of selected muscles for EMG latencies [ms]; data presented in bias and limits of agreement (1.96*SD) for right M. rectus abdominis (RRA), left M. (LEO), right M. tibialis anterior (RTA) and left M. biceps femoris (LBF)

Measures of reliability for amplitude investigations differed between muscles assessed at the perturbed walking trials. ICC values ranged from 0.31 (LLD) to 0.95 (RRA) for all assessed muscles (table 7 and 8). Test retest variability resulted in 20.2% (LRA) to 37.1% (RLD) for trunk muscles and 15.6% (LTA) to 41.5% (LGM) for leg muscles. SEM showed in mean 148 RMS% for trunk muscles and 103 RMS% for leg muscles. Bland Altman analysis revealed a bias ranging from -83 RMS% to 129 RMS%. More detailed results of systematic errors (bias) and 95% LoAs (1.96* SD) are shown in Bland Altman plots (figure 20), highlighting exemplary different characteristics of absolute reliability for single muscles at the trunk and lower extremities. Detailed results of all indicators of reliability between test day M1 and M2 are given in table 7 for trunk muscles and table 8 for muscles of the lower extremities.

Table 7: Indicators of reliability - amplitude analysis of trunk musculature

Muscle	N	Amplitude [RMS%] M1(mean ± SD)	Amplitude [RMS%] M2(mean ± SD)	ICC (95% CI)	SEM [RMS%]	TRV [%] (mean ± SD)	BA [RMS%] (bias ± 1.96*SD)
RRA	12	909 ± 979	885 ± 1264	0.95 (0.85 - 0.98)	242	25.9 ± 18.1	24 ± 666
LRA	13	698 ± 486	781 ± 670	0.90 (0.71 - 0.97)	178	20.2 ± 14.4	-83 ± 497
REO	12	634 ± 392	680 ± 543	0.78 (0.42 - 0.93)	213	34.5 ± 24.5	-46 ± 594
LEO	13	736 ± 292	741 ± 218	0.57 (0.23 - 0.85)	162	33.4 ± 25.7	6 ± 461
RIO	12	390 ± 154	361 ± 246	0.69 (0.23 - 0.90)	110	31.4 ± 20.8	29 ± 308
LIO	11	453 ± 261	448 ± 243	0.51 (-0.14 - 0.84)	168	37.0 ± 23.0	5 ± 478
RLD	12	531 ± 207	473 ± 288	0.39 (-0.21 - 0.78)	189	37.1 ± 23.3	58 ± 521
LLD	12	369 ± 172	367 ± 169	0.31 (-0.36 - 0.74)	136	36.2 ± 24.2	2 ± 383
REST	13	413 ± 177	387 ± 193	0.75 (0.37 - 0.92)	89	22.0 ± 15.5	26 ± 249
LEST	13	352 ± 175	333 ± 100	0.57 (0.43 - 0.85)	90	27.5 ± 19.9	19 ± 253
RESL	12	492 ± 206	473 ± 245	0.70 (0.27 - 0.90)	119	29.2 ± 19.3	19 ± 336
LESL	13	389 ± 251	326 ± 172	0.86 (0.55 - 0.96)	78	20.1 ± 13.3	63 ± 188

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left M. peroneus longus

Table 8: Indicators of reliability - amplitude analysis of leg musculature

Muscle	N	Amplitude [RMS%] M1(mean ± SD)	Amplitude [RMS%] M2(mean ± SD)	ICC (95% CI)	SEM [RMS%]	TRV [%] (mean ± SD)	BA [RMS%] (bias ± 1.96*SD)
RVM	13	718 ± 466	721± 556	0.88 (0.66 - 0.96)	171	25.2 ± 23.2	-2 ± 487
LVM	13	582 ± 535	538± 437	0.90 (0.71 - 0.97)	149	40.9 ± 23.5	45 ± 421
RBF	13	499 ± 326	527± 239	0.27 (-0.32 - 0.70)	236	32.5 ± 28.3	-28 ± 658
LBF	13	609 ± 312	480± 250	0.63 (0.18 - 0.87)	170	34.4 ± 24.0	129 ± 424
RGM	13	119 ± 54	121± 59	0.45 (-0.12 - 0.79)	41	27.8 ± 23.6	-2 ± 115
LGM	13	106 ± 97	89± 81	0.70 (0.29 - 0.89)	47	41.5 ± 33.8	17 ± 132
RPL	13	292 ± 122	298± 97	0.61 (0.10 - 0.87)	66	28.1 ± 18.1	-6 ± 187
LPL	13	170 ± 112	156± 82	0.77 (0.42 - 0.92)	45	28.0 ± 26.1	13 ± 127
RTA	13	441 ± 88	457± 94	0.43 (-0.14 - 0.79)	67	17.0 ± 11.7	-16 ± 185
LTA	13	290 ± 92	299 ± 92	0.83 (0.56 - 0.94)	37	15.6 ± 11.2	-9 ± 102

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; RRA/LRA: right/left M. rectus abdominis, REO/LEO: right/left M. externus obliquus, RIO/LIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM/LVM: right/left M. vastus medialis, RBF/LBF: right/left M. biceps femoris, RGM/LGM: right/left M. gastrocnemius medialis, RTA/LTA: right/left M. tibialis anterior, RPL/LPL: right/left M. peroneus longus

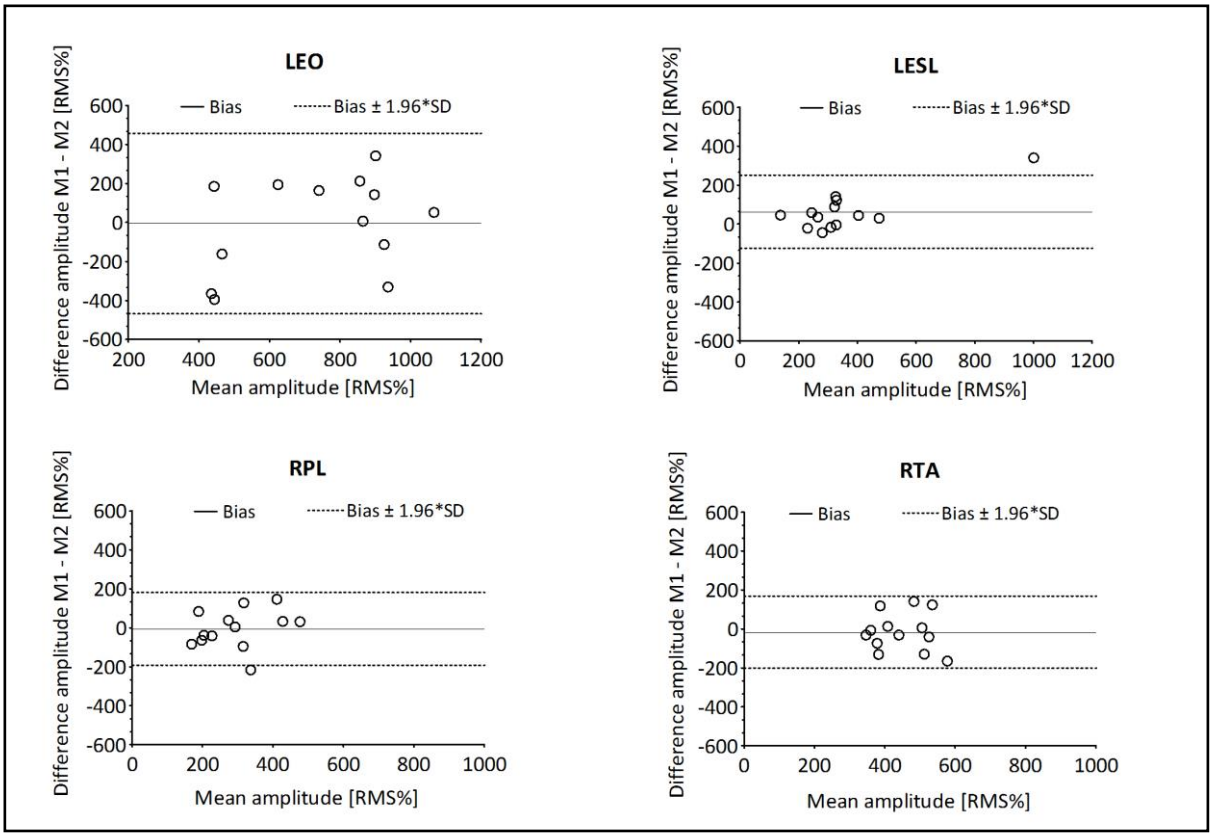

Figure 20: Bland Altman plots of selected muscles for EMG amplitudes [RMS%]; data presented in bias and limits of agreement (1.96*SD) for left M. externus obliquus (LEO), left M. erectus spinae lumbar (LESL), right M. peroneus longus (RPL) and right M. tibialis anterior (RTA).

Indicators of reliability for pooled data of muscle groups at the trunk (ventral right (RRA, REO, RIO), ventral left (LRA, LEO, LIO), dorsal right (RLD, REST, RESL), dorsal left (LLD, LEST, LESL)) and legs (upper leg right (RVM, RBF), upper leg left (LVM, LBF), lower leg right (RPL, RTA), lower leg left (RPL, RTA)) are presented in table 9 (latency investigations) and table 10 (amplitude investigations). Grouped latency analysis showed an ICC ranging from 0.12 (dorsal right) to 0.82 (ventral right), SEM ranging from 3 ms to 5 ms and TRV ranging from 4.6% to 9.2%. Bland Altman analysis revealed a bias of -4 ms to 4 ms. Grouped amplitude analysis showed an ICC ranging from 0.77 (dorsal right) to 0.93 (ventral right), SEM ranging from 24 RMS% to 132 RMS% and TRV ranging from 15.4% to 35.0%. Bland-Altman analysis revealed a bias of -28 RMS% to 81 RMS%.

Table 9: Indicators of reliability - latency analysis of grouped muscles (trunk + legs)

Muscle group	N	Latency [ms] M1(mean ± SD)	Latency [ms] M2(mean ± SD)	ICC (95% CI)	SEM [ms]	TRV [%] (mean ± SD)	BA [ms] (bias ± 1.96*SD)
ventral right	12	97 ± 8	92 ± 8	0.82 (0.05 - 0.96)	4	4.6 ± 3.3	-4 ± 6
ventral left	13	89 ± 10	90 ± 9	0.74 (0.15 - 0.92)	5	7.2 ± 5.3	1 ± 17
dorsal right	13	86 ± 5	84 ± 6	0.12 (-0.50 - 0.58)	5	6.6 ± 5.7	-2 ± 14
dorsal left	13	83 ± 9	80 ± 8	0.88 (0.61 - 0.96)	3	5.1 ± 4.6	-3 ± 10
upper leg right	12	79 ± 8	75 ± 6	0.60 (-0.17 - 0.88)	4	7.2 ± 6.8	-4 ± 13
upper leg left	13	82 ± 9	81 ± 9	0.63 (0.12 - 0.80)	5	9.2 ± 5.8	-1 ± 17
lower leg right	13	88 ± 8	85 ± 5	0.77 (0.29 - 0.93)	3	5.9 ± 3.6	-3 ± 10
lower leg left	11	89 ± 7	87 ± 6	0.74 (0.12 - 0.93)	3	5.3 ± 4.4	-3 ± 11

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg right: right M. vastus medialis, right M. biceps femoris; upper leg left: left M. vastus medialis, left M. biceps femoris; lower leg right: right M. tibialis anterior, right peroneus longus; lower leg left: left M. tibialis anterior, left M. peroneus longus

Table 10: Indicators of reliability - amplitude analysis of grouped muscles (trunk + legs)

Muscle group	N	Amplitude [RMS%] M1(mean ± SD)	Amplitude [RMS%] M2(mean ± SD)	ICC (95% CI)	SEM [RMS%]	TRV [%] (mean ± SD)	BA [RMS%] (bias ± 1.96*SD)
ventral right	13	637 ± 293	665± 312	0.93 (0.81 - 0.98)	132	24.6 ± 19.6	5 ± 342
ventral left	13	622 ± 456	616± 573	0.85 (0.57 - 0.95)	113	22.5 ± 14.1	-28 ± 317
dorsal right	13	370 ± 120	341± 114	0.77 (0.24 - 0.93)	83	27.5 ± 14.2	34 ± 294
dorsal left	13	475 ± 145	441± 210	0.82 (0.41 - 0.95)	48	17.9 ± 14.8	29 ± 172
upper leg right	13	582 ± 370	501± 291	0.82 (0.44 - 0.94)	121	31.3 ± 18.2	-17 ± 437
upper leg left	13	598 ± 271	614± 320	0.84 (0.57 - 0.95)	129	35.0 ± 19.0	81 ± 358
lower leg right	13	187 ± 77	181± 53	0.84 (0.49 - 0.95)	26	15.4 ± 7.0	-8 ± 94
lower leg left	13	276 ± 72	284± 60	0.86 (0.56 - 0.95)	24	17.4 ± 14.7	6 ± 89

ICC: intraclass correlation coefficient with 95% confidence interval (95%CI); SEM: standard error of measurement; TRV: test retest variability; BA: Bland Altman analysis; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg right: right M. vastus medialis, right M. biceps femoris; upper leg left: left M. vastus medialis, left M. biceps femoris; lower leg right: right M. tibialis anterior, right peroneus longus; lower leg left: left M. tibialis anterior, left M. peroneus longus

4.2 Results - Differences in muscular compensation pattern between LBP and CTRL

Eighty five participants meeting the eligibility criteria enrolled for study participation. During data analysis nine participants had to be excluded from final data set due to missing pain data or inadequate EMG quality as shown in the flow diagram (figure 21).

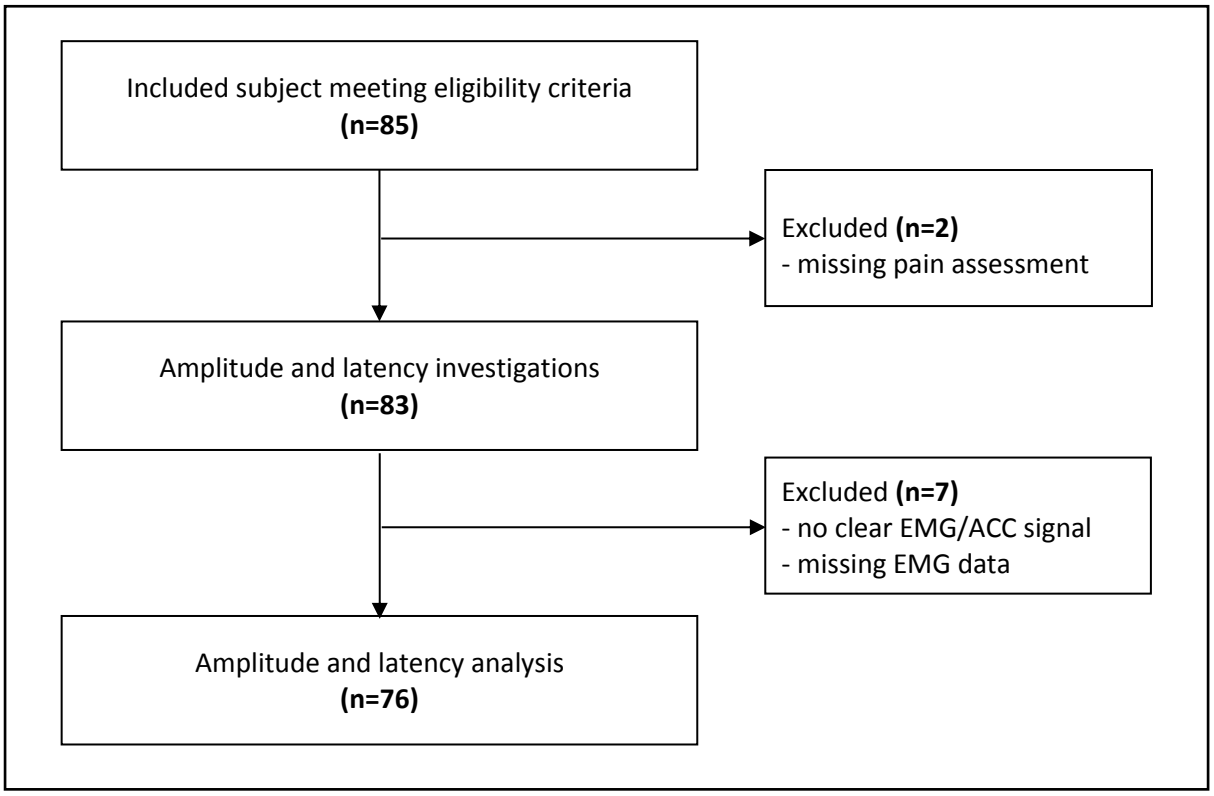

Figure 21: Flow diagram of enrolled participants of study two, stating numbers of subjects included/excluded for amplitude and latency investigations

4.2.1 Pain distribution

According to chronic pain grades (CPG), 12% of participants were categorized as pain free (grade 0), 75% as pain grade 1 (low disability, low intensity), 8% as pain grade 2 (low disability, high intensity), 4% as pain grade 3 (high disability, moderately limiting) and 1% as pain grade 4 (high disability, severely limiting). Group allocation based on the a priori defined grouping criteria (LBP: grade ≥ 2; CTRL: grade 0; chapter 3.3) resulted accordingly in a cohort size of 10 participants for LBP and 9 participants for CTRL group. Anthropometric data (table 11) showed significant differences between groups only for age of participants ($p=0.03$).

Table 11: Participants' anthropometrics - LBP and CTRL based on CPG classification

Group	Gender	Age [years]	Weight [kg]	Height [cm]
CTRL	3 M, 6 F	24 ± 4	71 ± 9	172 ± 9
LBP	4 M, 6 F	32 ± 9	70 ± 14	174 ± 11

Age and anthropometrics of participants (males (M) and females (F)); CTRL: control group; LBP: low back pain group; data are given as mean ± SD.

Distribution of characteristic pain intensity scores (CPIS; 0-100) for the 76 included participants is presented in figure 22. Group allocation based on the a priori defined grouping criteria (CTRL: pain intensity score 0 – 10; LBP: pain intensity score 30 – 100; chapter 3.3) resulted in a cohort size of 25 participants (38%) for LBP and 29 participants (32%) for CTRL group. No significant differences in anthropometric data (table 12) were shown between groups ($p>0.05$).

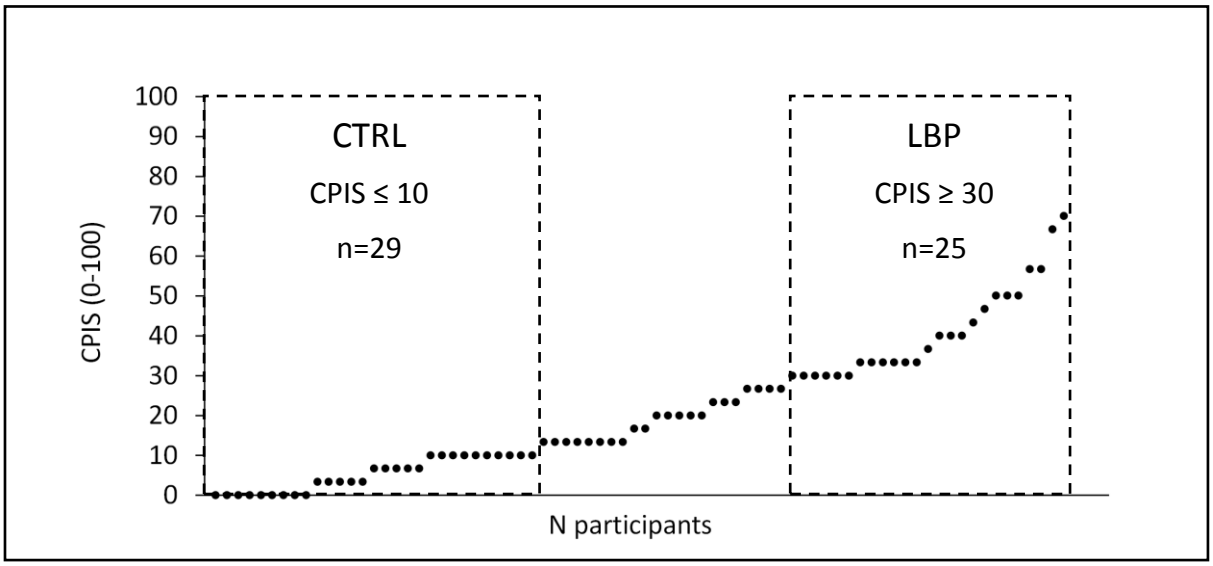

Figure 22: Group allocation according to distribution of characteristic pain intensity scores (CPIS) for the 76 included participants; CTRL: control group; LBP: low back pain group

Table 12: Participants' anthropometrics - LBP and CTRL based on CPIS classification

Group	Gender	Age [years]	Weight [kg]	Height [cm]
CTRL	13 M, 16 F	26 ± 7	72 ± 12	175 ± 11
LBP	9 M, 16 F	31 ± 9	73 ± 14	175 ± 12

Age and anthropometrics of participants (males (M) and females (F)); CTRL: control group; LBP: low back pain group; data are given as mean ± SD

Individuals of the study population were only in minority either categorized as pain free (absence of pain over the last three months) or as being in severe pain or suffering from pain related disability. Therefore, group allocation according to characteristic pain intensity scores seemed to be more suited and was used for the subsequent analysis of differences in muscular responses between people with LBP and pain free controls. However, analysis based on pain grade classification was performed additionally and can be found in the appendix at the end of this thesis.

4.2.2 Muscular responses to sudden perturbations in LBP and CTRL

EMG latencies for the cohort defined by characteristic pain intensity classification (CPIS) ranged between 65 ± 13 ms (RBF in CTRL) and 126 ± 30 ms (LRA, LBP) (table 13). EMG latencies at right gastrocnemius medialis (RGM) could not be assessed, due to an unclear on/off activation status. Mean latencies of the muscles at the trunk reached 90 ± 12 ms and mean latencies of the leg muscles 81 ± 10 ms. LBP showed longer latencies for all of the 16 assessed muscles compared to CTRL. Differences between CTRL and LBP for trunk muscles showed in mean 10 ± 8 ms (ranging from 2 ms to 30 ms) and for leg muscles 3 ± 2 ms (ranging from 1 ms to 5 ms). Largest differences of EMG latencies between LBP and CTRL were found for right and left M. rectus abdominis (RRA, LRA) and left M. internus obliquus (LIO). The overall activation pattern of EMG latencies in LBP and CTRL for the 16 analyzed muscles is shown in figure 23.

EMG amplitudes ranged between 150 ± 91 RMS% (RGM in LBP) and 889 ± 642 RMS% (RVM in CTRL) (table 13). Mean amplitude of muscles at the trunk reached 555 ± 109 RMS% and mean amplitude at the leg muscles 424 ± 214 RMS%. Differences between LBP and CTRL for trunk muscles showed in mean -13 ± 69 RMS% (ranging from -115 RMS% to 108 RMS%) and for leg muscles -84 ± 122 RMS% (ranging from -302 RMS% to -18 RMS%). Only minor differences between LBP and CTRL were found for all muscles, except for RVM showing increased amplitude for CTRL. The overall activation pattern of LBP and CTRL for all 17 assessed muscles is shown in figure 24 based on mean values and 95% confidence intervals for both groups.

Table 13: EMG latencies and amplitudes in CTRL and LBP

Muscle		EMG latencies [ms]		EMG amplitudes [RMS%]	
		CTRL	LBP	CTRL	LBP
ventral	RRA	99 ± 17	115 ± 19	503 ± 275	569 ± 401
	LRA	98 ± 21	117 ± 27	497 ± 266	548 ± 353
	REO	88 ± 14	96 ± 13	748 ± 420	708 ± 401
	LEO	80 ± 13	85 ± 13	690 ± 311	644 ± 254
	RIO	88 ± 13	95 ± 15	524 ± 355	632 ± 339
	LIO	90 ± 17	102 ± 24	546 ± 484	471 ± 296
dorsal	RLD	79 ± 10	84 ± 10	669 ± 306	722 ± 536
	LLD	80 ± 7	87 ± 11	589 ± 371	474 ± 253
	REST	77 ± 9	84 ± 10	554 ± 155	493 ± 191
	LEST	79 ± 10	85 ± 12	368 ± 143	309 ± 125
	RESL	79 ± 10	85 ± 9	554 ± 191	570 ± 233
	LESL	79 ± 9	82 ± 10	490 ± 258	442 ± 220
lower extremities	RVM	89 ± 15	93 ± 16	889 ± 642	587 ± 377
	RBF	64 ± 11	67 ± 16	501 ± 239	455 ± 226
	RGM	- -	- -	168 ± 150	150 ± 91
	RTA	80 ± 7	82 ± 7	438 ± 117	415 ± 113
	RPL	83 ± 8	84 ± 9	336 ± 136	302 ± 98

RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RGM: right M. gastrocnemius medialis, RTA: right M. tibialis anterior, RPL: right M. peroneus longus; CTRL: control group; LBP: low back pain group

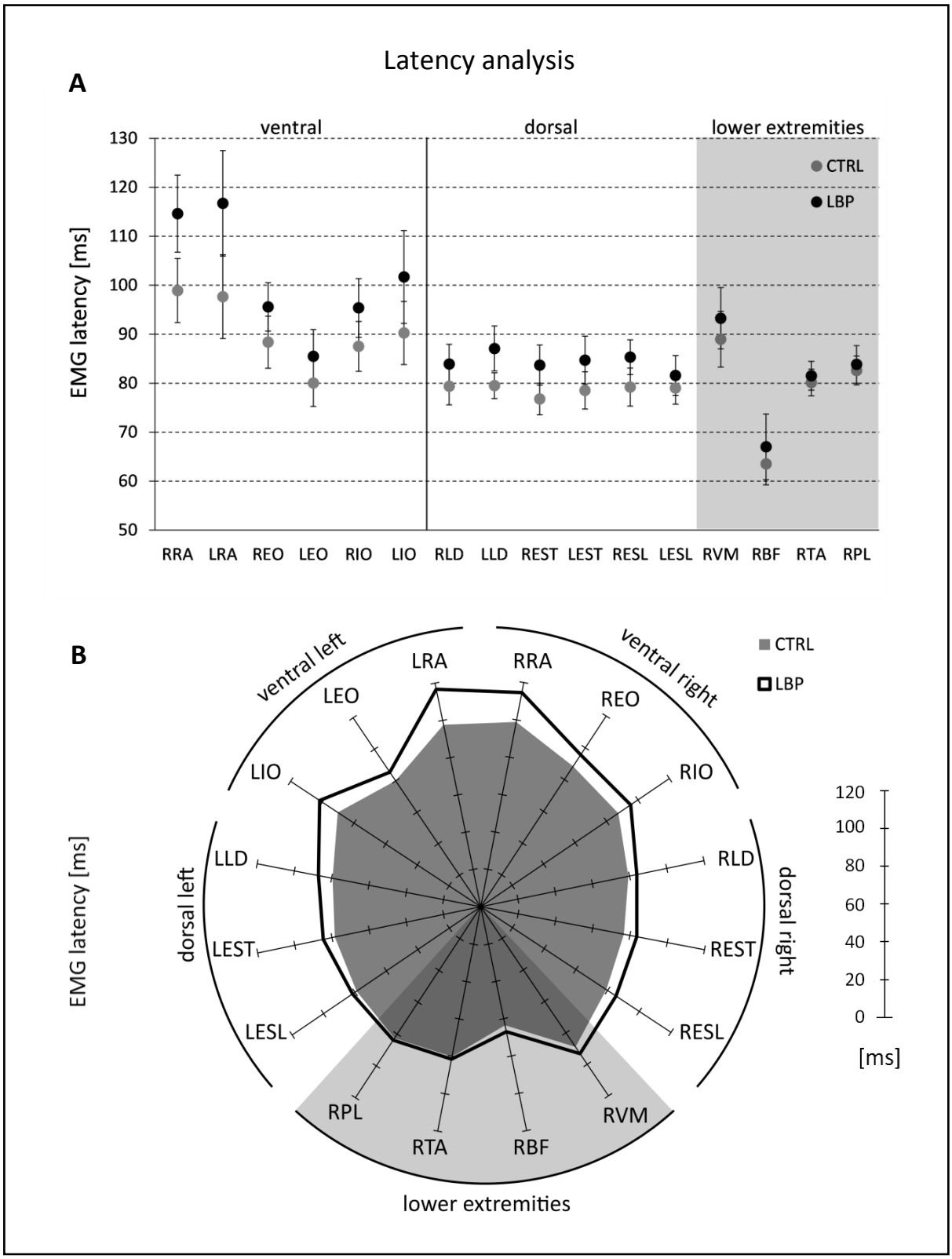

Figure 23: EMG latencies (ms) for low back pain (LBP) and controls (CTRL); data presented for individual muscles with means (A, B) and 95% confidence intervals (A); RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RTA: right M. tibialis anterior, RPL: right M. peroneus longus

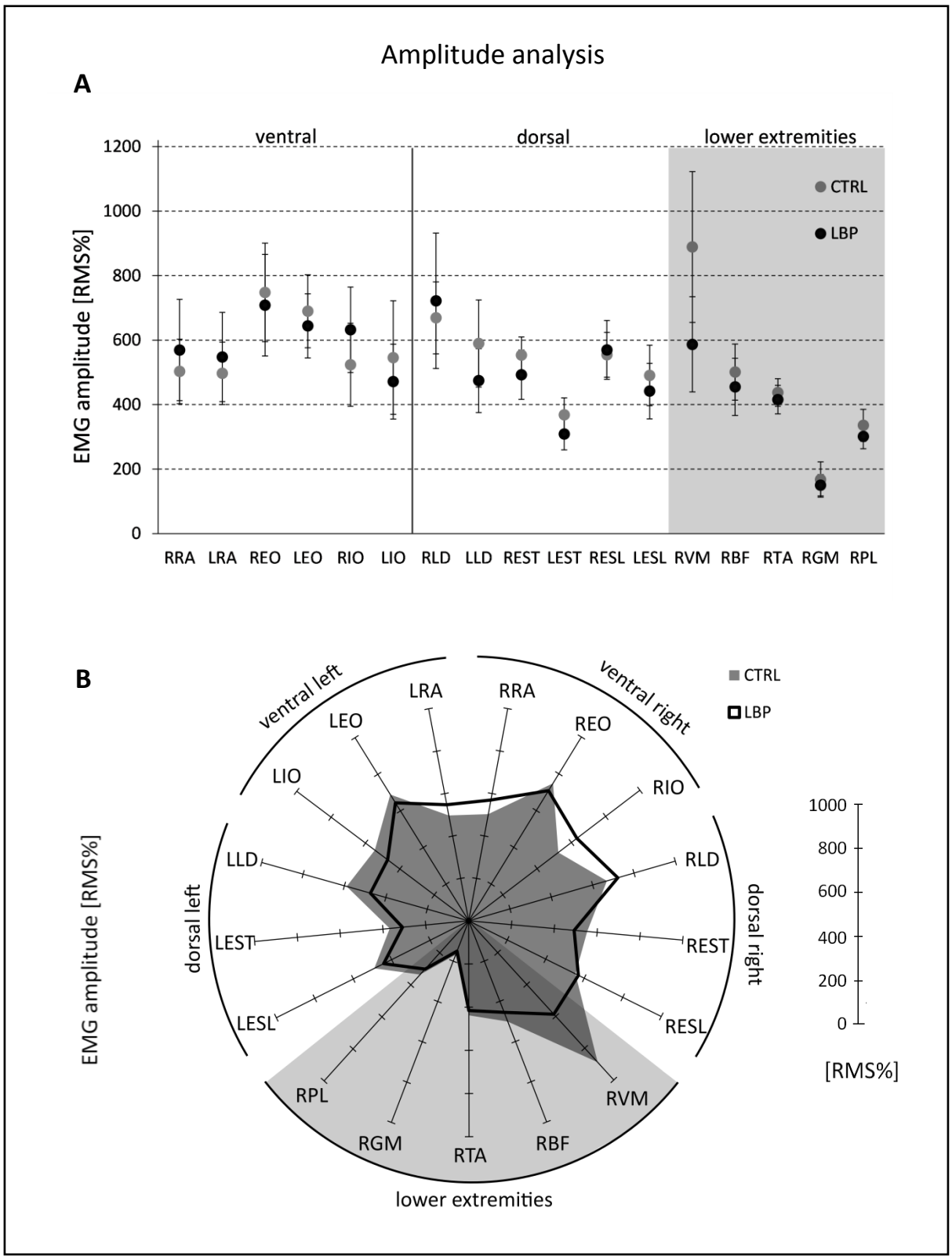

Figure 24: EMG amplitudes (RMS%, normalized to full stride of unperturbed walking) for low back pain (LBP) and controls (CTRL); data presented for individual muscles with means (A, B) and 95% confidence intervals (A); RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RGM: right M. gastrocnemius medialis, RTA: right M. tibialis anterior, RPL: right M. peroneus longus

Results of pooled muscles for latencies and amplitudes are shown in figure 25. To improve consistency between amplitude and latency investigations, lower leg muscles were pooled out of right tibialis anterior and peroneus longus (RTA, RPL) exclusively. Differences in EMG latencies between LBP and CTRL were highest ventral right (10 ms) and left (13 ms). Multivariate analysis of variance (MANOVA) revealed a statistically significant difference (V=0.23, F (6,47)=2.39, p=0.04) for muscle latencies between LBP and CTRL. Post hoc comparisons indicated that latencies were significantly differences at ventral right (p=0.009) and ventral left (p=0.007) (table 14). Differences in amplitudes between the groups were highest in dorsal left (74 RMS%) and upper leg (152 RMS%), being higher in CTRL compared to LBP. However, no statistically significant differences for amplitudes of grouped muscles were shown by MANOVA testing (V=0.18, F (6,47)=1.66, p=0.15) (table 14).

Table 14: EMG latencies and amplitudes in CTRL and LBP for grouped muscles

Muscle group	**EMG latencies [ms]**				**EMG amplitudes [RMS%]**			
	CTRL	**LBP**	**MANOVA**	**Post hoc**	**CTRL**	**LBP**	**MANOVA**	**Post hoc**
ventral right	92 ± 12	102 ± 13		p=0.009*	591 ± 253	636 ± 310		
ventral left	89 ± 15	102 ± 20		p=0.007*	578 ± 280	554 ± 214		
dorsal right	78 ± 9	84 ± 8	F=2.39	p=0.015	592 ± 161	608 ± 307	F=1.66	
dorsal left	80 ± 8	84 ± 10	p=0.04*	p=0.055	483 ± 211	408 ± 129	p=0.15	
upper leg	76 ± 11	83 ± 18		p=0.085	695 ± 337	521 ± 228		
lower leg	81 ± 7	82 ± 8		p=0.511	387 ± 97	359 ± 72		

Asterisks indicate significant differences; CTRL: control group; LBP: low back pain; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg: right M. vastus medialis, right M. biceps femoris; lower leg: right M. tibialis anterior, right M. peroneus longus

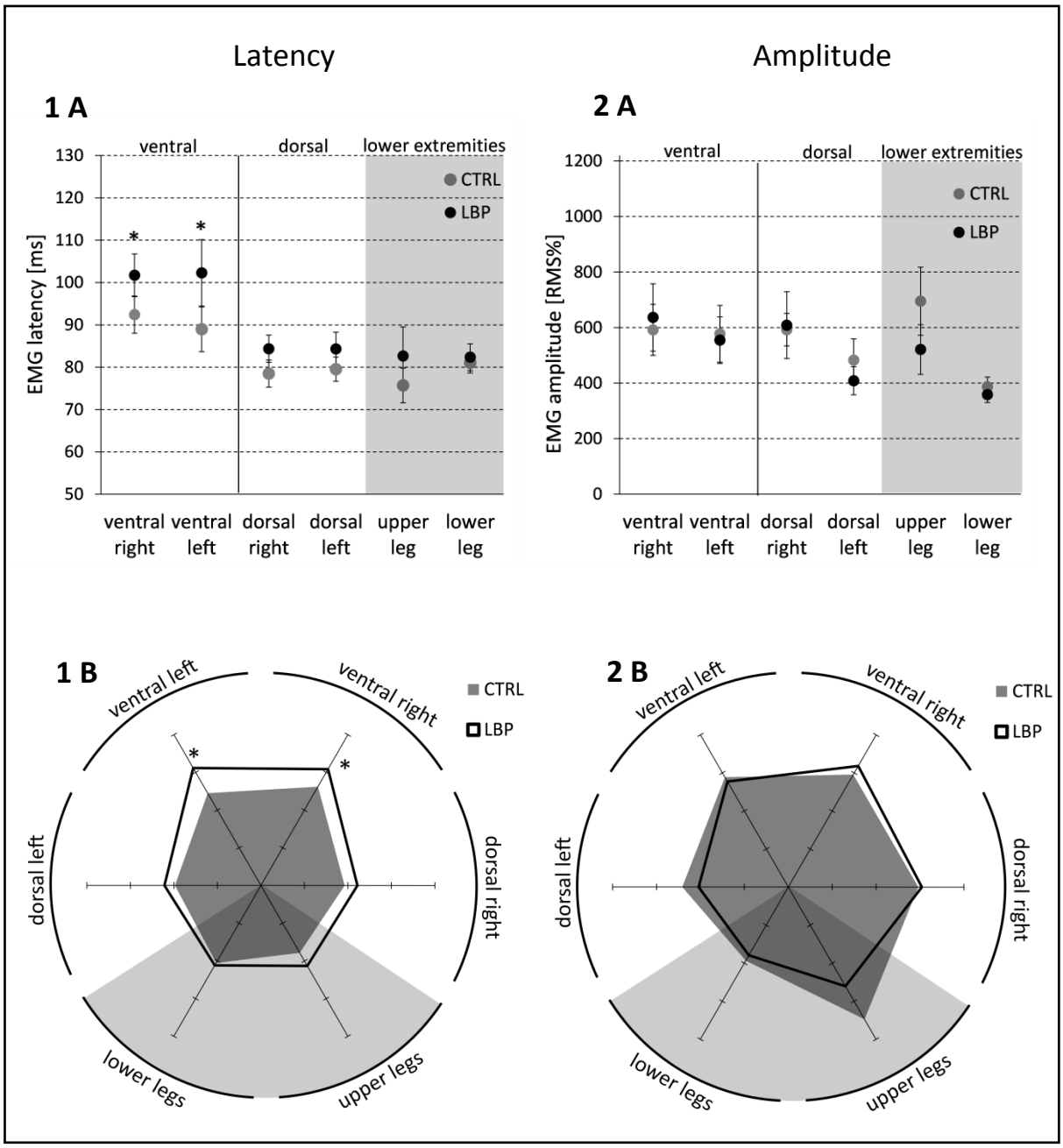

Figure 25: EMG latencies (1A,1B) and amplitudes (2A, 2B) for low back pain (LBP) and controls (CTRL); data presented for grouped muscles with means (1A, 1B, 2A, 2B) and 95% confidence intervals (1A, 1B); asterisks indicate significant differences ; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg: right M. vastus medialis, right M. biceps femoris; lower leg: right M. tibialis anterior, right M. peroneus longus

Differences of activation onset pattern between selected muscles pairs (table 15) showed temporal alterations between LBP and CTRL ranging from 0 ms (EST-ESL) to 13 ms (RA-TA). For co-contraction ratios (table 16) differences between LBP and CTRL ranged from 0 (EST:ESL) to 0.3 (RESL:LESL). No statistical differences were indicated for both differences in activation onset or co-contraction ratios of all assessed muscle pairs (table 15 and 16).

Table 15: EMG activation onset pattern between selected muscles pairs

Comparison	Muscle pair	EMG onset differences [ms]		
		CTRL	LBP	t-test (p)
right vs. left ventral	RRA - LRA	4 ± 10	1 ± 11	0.350
right vs. left dorsal	RESL - LESL	0 ± 9	3 ± 11	0.386
ventral only	RA - EO	15 ± 14	24 ± 18	0.089
dorsal only	EST - ESL	-1 ± 6	-1 ± 7	0.817
ventral vs. dorsal	RA - ESL	20 ± 18	31 ± 23	0.084
ventral vs lower leg	RA - TA	19 ± 16	32 ± 20	0.021
dorsal vs. lower leg	ESL - TA	-1 ± 11	2 ± 8	0.218

Delay of muscle onset (muscle 1 - muscle 2) for selected muscle comparisons for low back pain (LBP) and controls (CTRL); data presented in mean±SD; RRA: right M. rectus abdominis, LRA: left M. rectus abdominis; RESL: right M. erectus spinae (lumbar); LESL: left M. erectus spinae (lumbar); RA: averaged right and left M. rectus abdominis; EO: averaged right and left M. externus obliquus; EST: averaged right and left M. erectus spinae (thoracic); ESL: averaged right and left M. erectus spinae (lumbar); TA: right M. tibialis anterior

Table 16: EMG co-contraction ratios between selected muscles pairs

Comparison	Muscle pair	EMG amplitude ratios [arb. unit]		
		CTRL	LBP	t-test (p)
right vs. left ventral	RRA : LRA	1.1 ± 0.5	1.0 ± 0.3	0.609
right vs. left dorsal	RESL : LESL	1.3 ± 0.6	1.6 ±0 0.7	0.297
ventral only	RA : EO	0.8 ± 0.5	1.0 ± 0.6	0.421
dorsal only	EST : ESL	0.9 ± 02	0.9 ± 0.2	0.292
ventral vs. dorsal	RA : ESL	1.0 ± 0.4	1.2 ± 0.8	0.224
ventral vs lower leg	RA : TA	1.2 ± 0.8	1.4 ± 1.4	0.423
dorsal vs. lower leg	ESL : TA	1.3 ± 0.6	1.4 ± 1.3	0.790

Co-contraction ratios (muscle 1 : Muscle 2) for selected muscle comparisons for low back pain (LBP) and controls (CTRL); data presented in mean±SD; RRA: right M. rectus abdominis, LRA: left M. rectus abdominis; RESL: right M. erectus spinae (lumbar); LESL: left M. erectus spinae (lumbar); RA: averaged right and left M. rectus abdominis; EO: averaged right and left M. externus obliquus; EST: averaged right and left M. erectus spinae (thoracic); ESL: averaged right and left M. erectus spinae (lumbar); TA: right M. tibialis anterior

4.3 Summary of results

The first study investigated the validity and reliability of a newly developed testing situation, which aimed to assess muscular reflex activities in a situation of perturbed dynamic postural control. Evaluations of EMG activities revealed that the deployed protocol was able to provoke muscular reflex responses detectable both at the trunk and the lower extremities. EMG latency analysis indicated reflexive muscle activity following the stumbling incident with an average onset delay of 89 ms across all assessed muscles. EMG amplitude investigations revealed increased muscle activities following the stumbling (200 ms post perturbation), being on average 5 times higher than activations during unperturbed walking strides. Day-to-day reliability of muscle latency investigations showed a high level of reproducibility, both for muscles at the trunk and the lower extremities (table 5 and 6). In contrast, reproducibility of amplitude investigations was found to be only weak to moderate for individual muscles (table 7 and 8). EMG analysis of pooled muscles, representing a combined location specific outcome summary, showed no change in reliability indicators for latency analysis, but an increased reliability for amplitude analysis (table 9 and 10).

In the second study the evaluated and refined protocol was used to investigate differences in compensation strategies, quantified by muscular reflex responses, between people with LBP and a group of asymptomatic CTRLs. Assessment of pain severity (including intensity and disability measures) indicated an overall moderate level of pain severity in the study population. Characteristic pain intensity scores were used to allocate participants into either LBP or CTRL for the subsequent analysis (figure 22). Motor control strategies in response to sudden perturbations revealed longer delays for all assessed muscles within the LBP group compared to CTRL (table 13). Trunk muscles delays were found to be on average 10 ms longer compared to asymptomatic CTRLs, where differences at the lower extremities reached on average 3 ms. Onset latencies of grouped muscles according to location revealed statistically significant differences between LBP and CTRL for both right ($p=0.009$) and left ($p=0.007$) sided abdominal muscle groups (table 14). No statistically significant differences were found for muscle groups located at the lower extremities. Differences of activation onset pattern between selected muscles pairs showed highest alterations between LBP and CTRL, when comparing M. rectus abdominis to any other selected muscle, though not reaching a level of statistical significance (table 15). EMG amplitude analysis showed a high

variability in activation levels between individuals, independent of group assignment or location (table 16). No clear pattern of redistributed activity levels between muscles were observed in LBP. Also statistical testing of grouped muscles according to the location indicated no significant difference in amplitudes between LBP and CTRL. Ratio analysis of selected muscle pairs revealed as well no clear shift in activation pattern between LBP and CTRL. In summary, altered muscle activations in response to perturbations were characterized by longer latencies in LBP compared to CTRL at the trunk, predominantly at ventral sides. No clear differences could be obtained in terms of EMG activation level between LBP and CTRL, neither at the trunk nor at the lower extremities.

5 DISCUSSION

This research project aimed to investigate differences in compensation strategies in response to sudden walking perturbations in people with and without LBP. While previous investigations revealed altered muscle recruitment pattern and activation level at the trunk in response to sudden loading situations, these findings were mostly based on isolated and quasi-static trunk loading experiments. Therefore, the present research project proposed a testing situation, which intends to explore muscular responses during a dynamic task, being more representative for real life loading incidents. Two separate empirical investigations were conducted to address the formulated research questions.

5.1 Discussion - Validity and reliability of the testing situation

A new protocol was developed to investigate muscular compensation strategies at the trunk and lower extremities following walking perturbations. Therefore, evaluation of validity (RQ1) and reliability (RQ2) of the testing situation was a key element prior to its application. Muscular reflex responses provoked by stumbling were found to be clearly distinguishable by EMG signals from ongoing muscular activities of the underlying walking task. Considerable increases in activity levels in the assessed muscles indicated high velocity contractions following the perturbation within the targeted time window of 200 ms post perturbation (Ball et al., 2010). Onset delays ranging from 69 ms to 117 ms across all assessed muscles indicated response times within the range of polysynaptic reflex responses (50-80 ms) and triggered reactions (80-120ms) (Wilder et al., 1996; Milosevic et al., 2015; Eng et al., 1994). Previous studies investigating muscular responses following a sudden perturbation reported similar response times, with latencies at lower extremities ranging from 65 ms to 140 ms and latencies at the trunk ranging from 60 ms to 190 ms (Tang et al., 1998; Nashner, 1980; van der Burg et al., 2005). Relatively wide ranges between onset times of the individual studies might be explained by the differences in type of perturbations, directions and magnitudes, as well as by differences in used onset detection methods (Milosevic et al., 2015; Preuss & Fung, 2008; Nashner, 1980; Tang et al., 1998; Carter & Gutierrez, 2015; Hodges & Bui, 1996; Ebenbichler et al., 2001). However, the commonly reported onset delays indicated rather a polysynaptic response mechanism, than simple

stretch reflexes. Also, observations from Van der Burg (2005) revealed that muscular responses at the trunk following perturbations preceded mechanical trunk displacements, supporting the notion that other mechanisms than stretch reflex stimulations are responsible for triggering those reactive responses (van der Burg et al., 2005). In the present study, the longest onset delays were evident at the abdominal muscles, in particular at the M. rectus abdominis, which at the same time showed the highest EMG amplitudes following perturbations. This response pattern might indicate that delayed abdominal activities were compensated by increased activity levels to counteract the sudden forces. Contrary to this, van der Burg (2005) reported that abdominal muscles showed the shortest onset delays. Such contradicting results might show that deployed compensation strategies are adapted to the specific requirements of the situation. Others have shown that response times changed even dependent on the phase of the gait cycle in which the perturbation occurred (Berger et al., 1984). At the lower extremities, only muscular activity of the right M. gastrocnemius medialis was missing a clear onset in the present investigation, which was previously explained by a subsequent inhibition of the muscle following the perturbation (Tang et al., 1998; Ferber et al., 2002). Side differences between muscle onsets were small at all assessed muscles, with only slightly shorter response times at the right side of the body. Therefore, it seems that triggered compensation strategies rather deployed a bilateral approach of immediate muscle activation to react to the sudden disturbances of dynamic postural control. Furthermore, onset latencies were found to be quite homogeneous among individuals. In line with previous investigations, this observation might highlight the inherent stability of reflex timing in responses to sudden load changes, even though being tested under various conditions (Cholewicki et al., 2005; van der Burg et al., 2005; Radebold et al., 2000). Day to day reliability of latency analysis revealed a high level of reproducibility between testing days (Atkinson & Nevill, 1998; Hopkins, 2000), both at muscles surrounding the trunk and the lower extremities. Overall, test-retest variability stayed below 9%, SEM was on average 5 ms and systematic errors ranged between -7 ms and 8 ms. Only ICC values indicated poor reliability for some muscles (right M. peroneus longus). However, this was not confirmed by other indicators of reliability and might be explained by the fact that ICC values tend to report decreased reliability in homogeneous data distributions between individuals (Atkinson & Nevill, 1998). Despite the high reliability in latency analysis, muscle onsets could only be detected on average in 10 out of 13 participants. Furthermore,

automatic onset detection had to be corrected by visual inspection in 15% of all cases due to underlying muscle activity of the walking task. While this might introduce some form of subjectivity to the onset detection (Allison, 2003), visual determination of EMG onsets in previous studies was found to be a reliable method when performed by an experienced investigator (Hodges & Bui, 1996).

Amplitude investigations revealed the highest activity levels following perturbations at abdominal muscles (particularly M. rectus abdominis and M. externus obliquus) and upper leg muscles (M. vastus medialis and M. biceps femoris). Contrary, M. gastrocnemius medialis was found to be almost as low as during unperturbed strides. It is tempting to relate these findings to a specific reaction pattern in response to the applied perturbations. Previous studies revealed that abdominal muscles play an important role in counteracting walking perturbations, supporting this idea (van der Burg et al., 2005). Also, suppressive muscle inhibitions at the M. gastrocnemius in response to the stumbling incident are in line with previous investigations (Ferber et al., 2002; Tang et al., 1998). However, as amplitudes were normalized to unperturbed walking strides, it should be noted that activity levels of each muscle should rather be understood as a relative measure of their activity compared to normal walking. This also explains why EMG amplitudes were higher at muscles located at the trunk in comparison to muscles at lower extremities. Contrary, evaluations of side differences should more precisely describe actual differences in muscle activation levels, assuming equal activation levels for both sides during the locomotive reference task in asymptomatic individuals. Despite the fact that perturbations were exclusively applied at right heel contacts, side differences in EMG amplitudes were small. Only in M. peroneus longus and M. tibialis anterior, right sided amplitudes were higher compared to its left counterpart. This might indicate that the initial compensation strategy following the applied perturbations consists of a rather symmetrically pattern in terms of activation levels at the trunk and upper legs. Contrary, lower leg muscles might respond more unequally, as their task at the time point of perturbation is quite different between both sides. Previous investigations also could show that muscular responses at the lower extremities were dependent on their specific function during the gait cycle (Berger et al., 1984). Early studies further hypothesized that reactive strategies from distal leg muscles would be the primary mechanism for balance recovery during perturbed walking (Berger et al., 1984). However, in line with the findings of the present study, more recent investigations could show that

muscular contributions of proximal muscles around the hip and trunk also play an important role in compensation to walking perturbations (van der Burg et al., 2005; Stanek et al., 2011). Besides the considerable increase in activity levels, muscular responses were found to be highly variable between individuals. This might be partly attributed to movement variability and changes in posture during the walking task (Santos et al., 2011), despite that average measures of EMG amplitudes (5 perturbations) were used to account for such potential effects. Moreover, this high inter subject variability might indicate that individualised muscular compensation strategies in terms of activation level may serve to cope with the sudden perturbations. Similarly, measures of reproducibility indicated on average only weak to moderate levels of reliability for EMG amplitude when tested at different days. While systematic errors (bias) did not indicate learning effects between days, random errors reaching up to 666 RMS% and test-retest variabilities of up to 41% revealed a low level of reliability for repeated testing. Yet varying levels of reproducibility where not found to be location specific. Again, constant adjustments in muscle activity during the dynamic task might be responsible for the magnitude of variability at the level of single muscle contribution. Reliability of variables used in sudden loading studies is generally rarely documented in literature (Santos et al., 2011). However, Santos et al. (2011) revealed similar levels of reliability (ICC up to 0.62) for EMG responses following sudden loadings at the trunk, though measured in a static position with stabilized lower limbs and pelvis (Santos et al., 2011). While the authors suggested that a minimum of 8 perturbations should be used to derive more reliable average measure of EMG response activities following the sudden load application, such recommendations cannot be generalized as they are specific to the testing situation.

From a methodological perspective, the normalization of EMG amplitudes might also have affected the reliability of repeated assessment of muscle activity levels (Murley et al., 2010). Especially in a dynamic situation, where relative movements of electrodes above the skin might contribute to an increased variability in the EMG signal, normalization of EMG amplitudes is critical (Farina, 2006). In contrast to static conditions, isometric maximum voluntary contractions (MVC) may not adequately represent the maximum level of muscle activation during dynamic contractions (Ball & Scurr, 2013; Burden, 2010). Also, valid MVCs may not be obtainable in certain populations with symptomatic individuals such as LBP, because of pain or pain-related fear (Larivière et al., 2010; Thomas et al., 2008). Lastly, when

multiple muscles need to be analysed, measuring MVCs for each muscles is impractical (Ball & Scurr, 2013; Burden, 2010). Therefore, a variety of normalization techniques have been applied for investigations in dynamic conditions, such as normalizing EMG amplitudes to peak activities or to submaximal reference values (Granacher et al., 2010; Oliveira et al., 2013). In line with previous investigations, the present study normalized EMG signals to the averaged activity of unperturbed gait cycles (Granacher et al., 2010; Sloot et al., 2015). Though submaximal activities used as a reference value to normalize EMG amplitudes have been proven to be a reliable approach (Murley et al., 2010), low muscle activity levels during normal walking at the trunk might be seen as a potential risk for an increased variability in normalized EMG amplitudes. It should be noted however, that indicators of reliability for muscles at the trunk and muscles at the lower extremities (with higher unperturbed walking activities) did not show a systematic difference in reliability outcomes. Finally, random errors between measurements might be caused by other technical considerations of EMG assessment, such as variations in electrode placements on different measurement days (Murley et al., 2010). Even though electrode placement was conducted by an experienced examiner and controlled by the use of distinct anatomical landmarks such a source of error can't be ruled out completely.

A few potential limitations of the testing situation also need to be considered. Despite aiming for a testing situation resembling every day conditions, perturbed treadmill walking should still be regarded as an artificial stumbling situation (Forner Cordero et al., 2003; Sloot et al., 2015). Participants were required to walk at a constant pre-selected velocity and perturbation stimuli were always applied at the same phase of gait (during ground contact). Moreover, participants were aware that sudden perturbations may occur at some point of the testing trial, which may have altered their usual walking pattern (Forner Cordero et al., 2003).

In conclusion, the newly developed testing situation proved to be suited to provoke comprehensive muscular responses following sudden walking perturbations, both at the trunk and the lower extremities. Thereby, the used protocol might allow evaluating compensation strategies under perturbed dynamic postural control, which is highly relevant in real life situations. Both activation timing (EMG latencies) and activation level (EMG amplitudes) proved to be clearly detectable subsequent to the walking perturbations. While

observed latencies were found to be quite homogeneous between individuals, a high variability of amplitudes indicated a rather individualised muscular activation pattern in response to the stumbling incident. Similarly, reliability indicators revealed a high level of reproducibility in EMG onset detection between testing days and only weak to moderate levels of reproducibility in EMG amplitudes (Atkinson & Nevill, 1998; Fleiss, 1999). Previous studies identified differences in timing of muscle activation between CTRLs and people with LBP of around 10 ms (15%) when tested in quasi-static seated positions (Cholewicki et al., 2000). Thereby, expected differences in muscle timing under dynamic conditions are assumed to exceed the test-retest variability and standard error of measurements found in the present investigation. In contrast, the use of EMG amplitudes might only be suited for populations where high differences in activation levels between patients and asymptomatic controls are to be expected due to the inherent variability of EMG activation levels. Never the less, as there are no previous data available comparing muscular compensation strategies under similar conditions, the expected differences in activation level in people with LBP remain elusive. In consequence of the presented results, a few minor modifications were made to the testing protocol prior to its transfer to the second study (RQ3). To account for the potential effects of task variability between strides, both amplitude and latency analyses were based on average measures of 15 instead of 5 perturbations. Also, at lower extremities, muscular activity was exclusively assessed at right sides, where perturbations were directly applied during mid stance phase and hence represented a more controlled testing situation compared to the contra lateral lifted foot.

5.2 Discussion - Differences in muscular compensation pattern between LBP and CTRL

Assessment of pain was a fundamental requirement to answer the third research question of this research project. Following an acute trauma, pain is often assessed by simply asking the affected about their current subjective pain experience based on a rating scale system (Breivik et al., 2008). When pain becomes a chronic condition, however, valid pain evaluation becomes more demanding, as the natural course of prolonged pain has been found to be characterized by fluctuations over time (Breivik et al., 2008; van Tulder et al., 2002; Airaksinen et al., 2006). A variety of questionnaires have been developed to assess

chronic pain and its impact on function in the context of back pain (Longo et al., 2010; Morris et al., 2015). Von Korff et al. (1992) introduced a brief questionnaire ("The Chronic Pain Grade" questionnaire), which assesses both pain intensity at the current time and in the past (worst and average pain) as well as pain related disability in different environmental conditions (daily, social and work activities). Classification into chronic pain grades (CPG; 0-4), as well as scores of the underlying sub scales of characteristic pain intensity (CPIS, 0-100) and of disability (0-100) are finally used to quantify global severity, or pain intensity and pain related disability respectively (Khan et al., 2013; Von Korff et al., 1992). CPG grades were used as one approach to allocate participants (study two) into either the CTRL or LBP group in the present investigation. Based on this approach 12% of individuals were assigned to CTRL (grade 0), 13% to LBP (grade ≥ 2) and 75% of the study population was falling in between both group classifications (grade 1). Consequently, CPG classifications allowed distinguishing between pain free individuals and those suffering from high pain intensity or high pain related disability in the present cohort. However, group allocation based on CPG scheme might have some inherent drawbacks. CTRL group allocation (grade 0) requires the absence of any back pain over the time period of the last three months, as otherwise individuals are already categorized into the lowest grade of pain severity (grade 1). Therefore, the inclusion criteria for the CTRL group might be considered questionable, as single pain events over such a long time period may be unrelated to chronic back pain. On the other hand, allocation into LBP group is also challenging, as only individuals indicating high levels of pain intensity or pain related disability (grade ≥ 2) can reasonably be allocated into the LBP group. All ratings of lower pain intensity (CPIS score 0-49) fall into the same grade as ratings of almost no pain (grade 1). Therefore, a second allocation scheme was conducted based on CPIS measures which allowed a more sensitive group allocation of individuals related to pain intensity with allocation thresholds corresponding to those found in previous studies (Callaghan & Nelson-wong, 2013; Müller et al., 2014; Cedraschi et al., 1999; Von Korff & Miglioretti, 2005). Based on this approach, 38% of the population were assigned to CTRL (CPIS 0-10), 32% to LBP (CPIS 30-100) and 30% falling outside both group classifications. Consequently, in the present study population, CPG classification, which intends to distinguish especially between higher levels of pain severity (Von Korff et al., 1992), was not well suited for group allocation. Only strict comparisons between absence of pain and high pain severity are viable when using such a

classification scheme. CPIS scores on the other hand allowed a more subtlety graded group allocation of individuals based on a composite measure of three different pain intensity characteristics (current, past worst and past average). Accordingly, CPIS was used as the main group allocation into LBP and CTRL for this research project.

Muscular compensation strategies in LBP and CTRL subsequent to the stumbling incident were analyzed to answer the final research question of this thesis (RQ3). Thereby it was investigated whether trunk muscle latencies (RQ3a) and trunk muscle amplitudes (RQ3b) differed between LBP and CTRL in response to the walking perturbations. Furthermore it was investigated whether differences in compensation strategies were either consisting of changes local to the painful area at the trunk, or also in remote areas such as at the lower extremities (RQ3c).

Descriptive analysis of muscle latencies at the trunk revealed that muscular responses were delayed in LBP compared to CTRL. Predominantly muscle response times increased at the ventral side of trunk in LBP, with longest latencies occurring at both right and left sided M. rectus abdominis (RRA: 16 ms LRA: 19 ms). Those alterations in latencies between groups clearly exceeded the magnitude of measurement error observed in the previous test-retest investigations. At other sites of the trunk, differences in muscle onset between LBP and CTRL were lower, however still at a level well around the indicated measurement error. Statistical testing by general linear model revealed significant differences between LBP and CTRL for ventral right and ventral left muscle groups, whereas during post hoc comparisons both dorsal groups missed the significance level (Bonferroni correction; $p<0.01$). A number of previous studies repeatedly identified delayed trunk muscles responses in the context of LBP (Wilder et al., 1996; Radebold et al., 2000; Magnusson et al., 1996; Hodges & Richardson, 1996; Reeves et al., 2005). At a closer look, however, those detected alterations seemed also to be dependent on the specific testing situation. Magnusson et al (1996) reported of delayed muscular responses of M. erectus spinae at lumbar level in response to perturbations applied at the trunk in standing position. Unfortunately, absolute values of muscle latency times were not presented and response activation pattern limited to one specific back muscle. Studies by Radebold et al. (2000) and Reeves et al. (2005) revealed delayed shut-off times and switch-on times in LBP compared to controls, following a sudden load release at the trunk in a semi-seated position. Using force releases in three directions

(flexion, extension and lateral bending), grouped activities of both abdominal and back muscles seemed to be affected whether working as agonists or antagonists (Radebold et al., 2000). Onset delays between perturbation and muscle activation were found to be indicative for reflex activities with muscles typically responding within 100 ms following the load-release. Differences in latencies reached around 13 ms for switch-on events and 17 ms for switch-off events (Radebold et al., 2000). Contrary to applying load releases directly at the trunk, Hodges et al. (1996) used rapid shoulder movements during free standing (flexion, abduction and extension) to provoke trunk perturbations and related muscle activities. Although assessing EMG activities of abdominal muscles, lumbar multifidus, and the contralateral deltoid muscle, only contractions of transverse abdominis were significantly delayed in patients with LBP. In contrast to the externally triggered perturbations during the quick release experiments, self-initiated perturbations by rapid shoulder movements allowed the implementation of feed forward mechanisms. Thereby, a presumably optimized preparation for the load onset may have influenced the deployed response strategy (Hodges & Richardson, 1996). Similarly to the arm raising protocol by Hodges et al. (1996), in the present study perturbations were applied indirectly at the spine; however, onsets of perturbations were not anticipated by the participants as they were introduced by randomly timed stumbling incidents. Again, the observed compensation pattern seemed to be very specific to the testing situation. In response to the walking perturbations (rapid reversal of treadmill belt movement directions), predominantly ventral muscles were required to initiate a counteracting response to the occurring forces at the trunk. This might explain why differences between LBP and CTRL become more pronounced at ventral muscles where demands of reflex responses were particularly high. Relative differences in reflex timing showed that dorsal muscles responded prior to ventrally located muscles following the perturbations. In contrast to static investigations where co-contractions were found to be deployed to stiffen the trunk, a well-coordinated interaction of all involved muscles might be the key to maintain postural control in the constantly changing environment within dynamic tasks (Hodges, 2013; Borghuis et al., 2011, 2008). Besides the task specific differences of muscular response pattern between studies, detected delays of muscle onsets in LBP may critically alter the capabilities of the neuromuscular system to counteract sudden forces and thereby to protect and stabilize the spine (Reeves & Cholewicki, 2013; Santos et al., 2011). While reflex control inherently

involves a delay, even small increases in delays may result in considerably increased loadings of the spine (Van Dieën & Kingma, 2013). Additional delays in LBP as identified in the present investigation as well as in previous studies increased muscle onsets by 15-20% compared to asymptomatic controls (Radebold et al., 2000; Reeves et al., 2005). Those delays may become even more problematic, when occurring during fast dynamic situations (Reeves & Cholewicki, 2013). Lastly, in a prospective study by Cholewicki et al. (2005) it was shown that muscle reflex latencies in response to a quick force release at the trunk were predictive for future low back injury when tested in a population of athletes. Though history of LBP seemed to be the strongest predictor of future LBP, the odds of sustaining a low back injury increased with each millisecond of additional delay in muscle shot-off latencies by 3% (Cholewicki et al., 2005).

Descriptive analysis of EMG amplitudes at the trunk in the present study indicated that there was no group difference between LBP and CTRL in activation levels within 200 ms post perturbation. High standard deviations showed that muscular responses following the perturbations were characterized by a considerable inter subject variability, independent of the presents or absence of LBP. Also, statistical testing of combined outcome measures of muscles grouped according to their location indicated no significant differences for EMG amplitudes between LBP and CTRL. In contrast to these findings, previous studies identified changes in activation levels at the trunk following sudden loading incidents in people with LBP (Larivière et al., 2010; MacDonald et al., 2010). However, conflicting results were reported in regards of the direction of changed muscle activation levels. Larivière et al. (2010) discovered significantly higher amplitudes of trunk muscles in LBP compared to CTRLs, following sudden forward perturbations at the trunk in half seated position (quick loading apparatus). Findings from MacDonald et al. (2010) on the other hand showed a decreased EMG activity for both deep and superficial trunk muscles following sudden external loadings indirectly applied via the upper extremities in standing position (load drop into extended arms). Though differences in study populations and methodological considerations may have contributed to those oppositional responses, muscular reflex activities may likely have been adapted to the different requirements of each testing situation. Contrary to previous studies, the present study investigated in muscular responses under dynamic conditions. While during static postural tasks control of the centre of mass is the most fundamental requirement to maintain balance, in dynamic tasks

mechanical parameters in relation to the movement, such as linear and angular momentums, have to be controlled in addition (van der Burg et al., 2005). Constant adjustments in muscle activity during the dynamic task might explain the degree of variability in amplitudes for single muscle contribution. However, high variability in muscular reflex amplitudes following the perturbation may also indicate that there are several possible response strategies available to recover from a sudden loading incident. Previous investigations acknowledged that the redundancy of force generators evident at the trunk may allow to use various activation strategies resulting in the same kinematic output of the trunk (Reeves et al., 2005). Considering that muscle activity responses were often found to be rather individual-specific than stereotypically altered in the presence of pain (Hodges et al., 2013a), potential changes in muscular activation levels related to LBP may have been covered by the overall diversity of deployed response strategies provoked by the stumbling incident.

Finally, it was investigated whether potential changes in muscular activations may exceed the area of the trunk in a dynamic situation, which requires more than an isolated response strategy of the trunk. Previous research mostly assessed muscular activities following perturbations under (quasi) static conditions, with the load being applied directly on the trunk and the lower limbs and pelvis being well stabilized (Radebold et al., 2000; Santos et al., 2011; Reeves et al., 2005). While such an experimental approach might allow more precise research into isolated response strategies of the trunk, it has been questioned, to what extend resulting reflex responses are relevant to real-life perturbations (Santos et al., 2011). Few studies investigated response strategies, where muscular contributors of postural control offsite the trunk were additionally involved (Jacobs et al., 2011; Jones et al., 2012b). Moreover, none of those studies deployed sudden indirect loading incidents in a dynamic testing situation. Jacobs et al. (2011) reported altered muscular activity pattern both at the trunk and at lower legs in response to sudden surface translations during free standing. According to their results, individuals with LBP experienced fewer early and late phase activations at both muscles located at the trunk and at lower extremities following the sudden loading. Also, EMG amplitudes were higher in assessed leg muscles following the perturbations in LBP compared to CTRLs (Jacobs et al., 2011). Based on these results, the authors suggested that LBP might be linked to a central change in multi segmental muscle coordination. In another study, Jones et al. (2012) assessed muscle activations of ten

muscles surrounding the trunk and two additional muscles in the lower legs in response to multi-directional surface translations. EMG responses were reported to be increased following perturbations, when muscles were acting as prime movers and reduced in opposing directions, both in muscles located at the trunk and at the lower extremities (Jones et al., 2012a). Again, it was hypothesized that the observed changes in the response pattern in LBP may likely reflect the influence of a central nervous system processing to cope with the sudden disturbances. Further research identified altered triggered reactions of the biceps brachii following expected limb loading and impaired postural stability following disturbances of postural control (Leinonen et al., 2007; Luoto et al., 1998). While these findings may only provide a first insight on how extensive pain may influence motor behavior, they demonstrate that changes in LBP can only partially be explained by traditional pain models, such as the 'pain spasm pain' theory or the 'pain adaptation' theory. Rather, observations in LBP seem to be represented by a more recent model on 'motor adaptation to pain', which assumes that adaptations to pain may aim to reduce pain or protect the painful part with a more flexible solution (Hodges & Tucker, 2011). In the present study, reflex responses both of the trunk and the lower extremities were assessed under perturbed dynamic control. It was targeted to challenge motor control strategies as they may occur in real life situations, where muscular trunk responses have to be deployed in conjunction with muscles located at the lower extremities to counteract sudden forces. Contrary to static investigations, altered response timing in LBP was predominantly evident at the trunk. Minor increases in response delays at the lower extremities in LBP were neither statistically significant nor may they be of practical relevance. In addition, analysis of reflex amplitudes showed a high variability between individuals at all assessed muscles, independent of the absence or presence of pain. Thereby, no indication for a reorganization of muscular activities exceeding the trunk could be revealed for muscular responses in perturbed walking causing trip-like events. However, in line with previous research, a task specific response pattern was observed that consisted of alterations in muscular response timing not restricted to back muscles, but rather predominantly present in abdominal muscles in people with LBP. Based on recent models of motor adaptation in pain, delayed reflex activities might be interpreted as an attempt to prevent the painful area from further pain or injury (Hodges & Tucker, 2011). Further, such a strategy might be beneficial in short term prevention by reducing rapid motion during (voluntary) movements, but becoming

maladaptive over a longer time and in particularly in situations, where fast responses are required to prevent the spine from excessive strain, such as in sudden perturbations (Hodges & Tucker, 2011; Hodges, 2013; van Dieën et al., 2003). Missing alterations in amplitude measures in the present investigation might either be caused by the observed individual-specific variability of single muscle contribution or by the fact that no such alterations were existent in people with LBP. Many factors may influence the chosen response strategy, such as habitual strategies, postural parameters, functional demands or anthropometrics (Hodges, 2013), however the actual selection processes remain elusive. Another explanation for the absence of altered motor responses at the lower extremities could be that the repetitive rhythmic motor pattern of human locomotion is less susceptible to altered motor control strategies deployed in LBP. Indeed, its repetitive quality allows locomotion to be controlled at relatively low levels of the nervous system without intervention by higher central nervous centers (Kandel et al., 2000). Although, caused by unpredictable environmental changes, locomotor movements are continually modified, to adapt to the new requirements (Kandel et al., 2000). Also, it should be noted that severity of pain may as well play an important role regarding the adaptation of motor behavior. Results of the presented study may therefore only be representative for a population reporting a rather moderate level of pain intensity. However, additional analysis based on CPG classifications, allocating only people with higher pain severity into the LBP group, did not change the overall results of this investigation (see appendix for results based on CPG classification).

A few limitations of the presented study design should be addressed. While perturbed treadmill walking may resemble a situation of disturbed dynamic postural control occurring under real life circumstances, it should still be considered an artificial laboratory assessment (Forner Cordero et al., 2003; Sloot et al., 2015). Also, it should be noted that fear of pain may have altered normal walking behavior in people with LBP. Previous studies showed, that anticipation of pain is already sufficient to change motor control (Moseley et al., 2004). Another potential limitation might be that muscular responses were only assessed by superficial muscles. Surface EMG signals recorded at the trunk may often be just a composite of activities from various muscles in different layers (Kawchuk, 2013). Therefore, the understanding of deep muscle contributions to the deployed compensation strategies may be limited. Furthermore, baseline EMG levels prior to perturbations were not assessed

in the present study due to the underlying task activity. Investigations in baseline activities, however, identified increased baseline activity levels in previous studies (Larivière et al., 2010; Jacobs et al., 2011; Stokes et al., 2006). While it was hypothesized that such a strategy might be selected as an attempt to stiffen and stabilize the trunk under (quasi) static conditions (Stokes et al., 2006), a statement regarding dynamic conditions is still missing. Using EMG as the exclusive outcome measure of response activations also may have limited the explanatory power of altered motor control strategies found in this study. A more detailed picture of potential changes upstream the motor system would therefore require the recordings from techniques such as electroencephalography (Van Dieën & Kingma, 2013). Only a few studies have used such techniques to investigate changes at a cortical level (Tsao et al., 2008; Strutton et al., 2005; Chiou et al., 2014). Lastly, the question of whether LBP represent rather the cause or the effect of the observed alterations in motor control remains unanswered (Callaghan & Nelson-wong, 2013; Moseley, 2013). Although it is often assumed that pain may lead to motor adaption, prospective studies providing evidence for such a claim are rare (Moseley, 2013).

6 SUMMARY

Motor control is vital for the spine (Ferguson, 2008; Van Dieën & Kingma, 2013). To protect itself from harmful injury, the spine has to adapt its characteristics from being flexible to rigid and vice versa within fractions of seconds in a constantly changing environment (Panjabi, 1992; Hammill et al., 2008). In LBP, motor control has been found to be impaired, leading to a variety of alterations in motor behavior related to the trunk (Newcomer et al., 2000; Reeves et al., 2005; Magnusson et al., 1996; Cholewicki et al., 2000; Larivière et al., 2005). In particular, control strategies seemed to be altered in situations requiring a reactive response of the trunk in the event of sudden external forces (Radebold et al., 2000, 2001; Cholewicki et al., 2005; Reeves et al., 2005). Recordings of EMG activities revealed changes in muscular activation level, timing of muscle activation and changes in intermuscular recruitment pattern of the muscles surrounding the trunk (Navalgund et al., 2013; Larivière et al., 2010; Radebold et al., 2000). However, muscular responses were mostly assessed in (quasi) static testing situations under simplified conditions (Radebold et al., 2000, 2001;

Cholewicki et al., 2005; Reeves et al., 2005). Whether observed muscular responses following isolated trunk loading experiments are comparable to real-life response strategies under less restricted circumstances has been questioned (Santos et al., 2011). Especially as muscular response strategies seemed to be depending on the specific requirements of the testing situation (Milosevic et al., 2015). Only a few studies investigated in muscular activation strategies deployed both at the trunk and offsite in response to sudden surface translations (Jacobs et al., 2011; Jones et al., 2012b). Those studies identified modified response strategies in LBP both at the trunk and offsite the trunk, likely reflecting the influence of an altered central nervous system processing to cope with the sudden disturbances of postural control (Jones et al., 2012b). Still, research was lacking in muscular response strategies in dynamic situations, challenging the motor control system to deploy reactive responses under constantly changing environmental conditions. Therefore, the present research project aimed to investigate muscular compensation strategies following unexpected gait perturbations in individuals with and without LBP. A new treadmill stumbling protocol was developed and tested for its validity and reliability to provoke muscular reflex responses both at the trunk and the lower extremities. Results of this first investigation demonstrated the feasibility of the proposed testing situation. Applied stumbling incidents during locomotion led to clearly detectable reflex responses subsequent to the perturbation, with EMG amplitudes being on average 5 times higher compared to unperturbed walking strides. However, a high inter-subject variability in activation amplitudes suggested that either constant adjustments in muscle activity during locomotion may lead to changes in single muscle contributions or that a variety of response strategies may be available to recover from the sudden loading incident. Similarly, day-to-day reliability of reflex latencies indicated a high reproducibility of muscle latency assessment, whereas a high variability in activation levels was observed in EMG amplitude analysis. In the second investigation, the validated testing protocol was used to evaluate differences in muscular compensation strategies between people with and without LBP. A composite of three different pain intensity characteristics (current pain, past worst and past average pain) was used to allocate participants into LBP or CTRL group, according to self-reported pain intensities. Muscular reflex responses at the trunk were found to be delayed in LBP compered to CTRL. Thereby, indirect loadings led to a similar response pattern compared to onset delays observed in isolated trunk loading experiments in previous studies. However,

alterations in LBP seemed to be deployed in a task specific manner, with onset delays predominantly occurring at ventral muscles sites. In contrast, activity levels of reflex responses did not show any statistically significant differences between LBP and CTRL. It might be speculated, that potential changes in muscular activation levels may have been covered by the overall diversity of deployed response strategies following the perturbation. Evaluation of muscles activities at the lower extremities did not reveal any differences, neither in timing nor in activation levels between LBP and CTRL. Contrary to previous studies investigating under static conditions, under dynamic conditions no indication for a reorganization of muscular activities exceeding the trunk could be observed.

In conclusion, the present research project showed that sudden loadings indirectly applied at the trunk under dynamic conditions provoked an altered reflex timing of muscles surrounding the trunk in people with LBP. Though similar to findings of isolated trunk loading experiments, compensation strategies seemed to be deployed in a task specific manner. No muscular alterations could be found exceeding the area of pain when being assessed under the automated task of locomotion. While rehabilitation programs tailored towards LBP are still under debate, it is tempting to urge the implementation of sudden loading incidents to enhance motor control and thereby to improve spinal protection. Moreover, in respect to the consistently observed task specificity of muscular compensation strategies, such a rehabilitation program should be rich in variety.

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10 LIST OF ABRIVIATIONS

ACC	accelerometer
BA	Bland-Altman analysis
CPG	chronic pain grade
CPIS	characteristic pain intensity scale
CTRL	control
EMG	electromyography
EO	right and left M. externus obliquus (average)
ESL	right and left M. erectus spinae (lumbar) (average)
EST	right and left M. erectus spinae (thoracic) (average)
ICC	intra class correlation coefficient
LBF	left M. biceps femoris
LBP	low back pain
LEO	left M. externus obliquus
LESL	left M. erectus spinae (lumbar)
LEST	left M. erectus spinae (thoracic)
LGM	left M. gastrocnemius medialis
LIO	left M. internus obliquus
LLD	left M. latissimus dorsi
LoA	Limits of agreement
LPL	left M. peroneus longus
LRA	left M. rectus abdominis
LTA	left M. tibialis anterior
LVM	left M. vastus medialis
M1	measurement 1
M2	measurement 2
MANOVA	multivariate analysis of variance
RA	right and left M. rectus abdominis (average)
RBF	right M. biceps femoris
REO	right M. externus obliquus
RESL	right M. erectus spinae (lumbar)
REST	right M. erectus spinae (thoracic)
RGM	right M. gastrocnemius medialis
RIO	right M. internus obliquus
RLD	right M. latissimus dorsi
RMS	root mean square
RPL	right M. peroneus longus
RQ	research question
RRA	right M. rectus abdominis
RTA	right M. tibialis anterior
RVM	right M. vastus medialis
SEM	standard error of measurement
TRV	test-retest variability
VAS	visual analogue scale

11 APPENDIX

11.1 Korff questionnaire - Original/German version

Table 17: Questions of "The Chronic Pain Grade" questionnaire (CPG) by Von Korff in German language as provided to the participants and its respective English translations

Nr.	Question
1	Wie würden Sie Ihre Schmerzen, wie sie in diesem Augenblick sind, einstufen? (How would you rate your pain on a 0-10 scale at the present time, that is right now, where 0 is „no pain" and 10 is „pain as bad as could be?")
2	Wenn Sie an die Tage denken, an denen Sie in den letzten drei Monaten Schmerzen hatten, wie würden Sie Ihre stärksten Schmerzen einstufen? (In the past three months, how intense was your worst pain rated on a 0-10 scale, where 0 is „no pain" and 10 is „pain as bad as could be"?)
3	Wenn Sie an die Tage denken, an denen Sie in den letzten drei Monaten Schmerzen hatten, wie würden Sie die durchschnittliche Stärke der Schmerzen einstufen? (In the past three months, on average, how intense was your pain rated on a 0-10 scale where 0 is „no pain" and 10 is „pain as bad as could be"? (That is, your usual pain at times you were experiencing pain.))
4	In welchem Maße haben die Schmerzen in den letzten drei Monaten Ihren Alltag (Ankleiden, Waschen, Essen, Einkaufen etc.) beeinträchtigt? (In the past three months, how much has back pain interfered with your daily activities rated on a 0-10 scale where 0 is „no interference" and 10 is „unable to carry out on activities"?)
5	In welchem Maße haben die Schmerzen in den letzten drei Monaten Ihre Freizeitaktivitäten oder Unternehmungen im Familien- oder Freundeskreis beeinträchtigt? (In the past three months, how much has back pain changed your ability to take part in recreational, social and family activities where 0 is „no change" and 10 is „extreme change"?)
6	In welchem Maße haben die Schmerzen in den letzten drei Monaten Ihre Arbeitsfähigkeit (einschließlich Hausarbeit) beeinträchtigt? (In the past three months, how much has pain changed your ability to work (including housework) where 0 is „no change" and 10 is „extreme change"?)
7	An ungefähr wie vielen Tagen konnten Sie in den letzten drei Monaten aufgrund von Rückenschmerzen Ihren üblichen Beschäftigungen (Beruf, Schule/Studium, Hausarbeit) nicht nachgehen? (About how many days in the last three months have you been kept from your usual activities (work, school or housework) because of back pain?)

11.2 Calculation of chronic pain grades

Calculation of chronic pain grades and the respective sub scores is based on the following scoring system (Von Korff et al., 1992):

Characteristic Pain Intensity is a 0-100 score derived from question 1-3:
Mean (Pain Right Now, Worst pain, Average Pain) x 10

Disability Score is a 0-100 score derived from questions 5-7:
Mean (Daily Activities, Social Activities, Work Activities) x 10

Disability Points: add the indicated points (table 18)
for Disability Days (question 4) and for Disability Score

Table 18: Calculation of Disability Points by "Disability Days" and "Disability Score":

Disability points			
Disability days (0-180)		Disability score (0-100)	
0-6 Days	0 Point	0-29	0 Point
7-14 Days	1 Point	30-49	1 Point
15-30 Days	2 Point	50-69	2 Point
31+ Days	3 Point	70+	3 Point

Scores of *Characteristic Pain Intensity* and *Disability Points* are subsequently used to build the different chronic pain grades as following (table 19):

Table 19: CPG classification with grade 0-4 based on pain intensity and disability points

Grade	**Characteristics**	
0	Pain free	No pain problem
1	Low disability – low intensity	Characteristic Pain Intensity less than 50, and less than 3 Disability Points
2	Low disability – high intensity	Characteristic Pain Intensity of 50 or greater, and less than 3 Disability Points
3	high disability – moderately limiting	3-4 Disability Points, regardless of Characteristic Pain Intensity
4	high disability – severely limiting	5-6 Disability Points, regardless of Characteristic Pain Intensity

11.3 Muscular responses based on chronic pain grade classification (CPG)

EMG amplitudes and latencies for the cohort defined by CPG classification are shown in table 20. The amplitude activation pattern of all 17 assessed muscles is shown in figure 26 for LBP and CTRL group for CPG, provided as mean values and 95% confidence intervals. Respectively, the latency activation pattern of all 17 assessed muscles is shown in figure 27.

Table 20: EMG amplitudes and latencies for CTRL and LBP

	Muscle	EMG amplitudes [RMS%]		EMG latencies [ms]	
		CTRL	LBP	CTRL	LBP
ventral	RRA	370 ± 167	646 ± 531	93 ± 9	112 ± 21
	LRA	437 ± 201	576 ± 343	96 ± 20	126 ± 30
	REO	642 ± 427	820 ± 522	91 ± 13	99 ± 15
	LEO	626 ± 249	740 ± 257	81 ± 14	85 ± 13
	RIO	464 ± 235	628 ± 404	91 ± 13	94 ± 20
	LIO	463 ± 222	461 ± 156	89 ± 18	106 ± 34
dorsal	RLD	616 ± 276	736 ± 638	79 ± 12	86 ± 10
	LLD	520 ± 271	554 ± 268	80 ± 9	86 ± 8
	REST	591 ± 193	446 ± 152	79 ± 8	86 ± 10
	LEST	403 ± 134	291 ± 87	81 ± 10	90 ± 12
	RESL	581 ± 184	577 ± 227	77 ± 8	86 ± 10
	LESL	399 ± 152	406 ± 163	80 ± 8	82 ± 13
lower extremities	RVM	635 ± 331	583 ± 259	90 ± 11	93 ± 13
	RBF	613 ± 352	361 ± 120	65 ± 13	69 ± 21
	RGM	148 ± 93	144 ± 70	- -	- -
	RTA	394 ± 147	442 ± 91	81 ± 6	82 ± 8
	RPL	307 ± 120	250 ± 94	84 ± 8	87 ± 13

RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RGM: right M. gastrocnemius medialis, RTA: right M. tibialis anterior, RPL: right M. peroneus longus; CTRL: control group; LBP: low back pain group

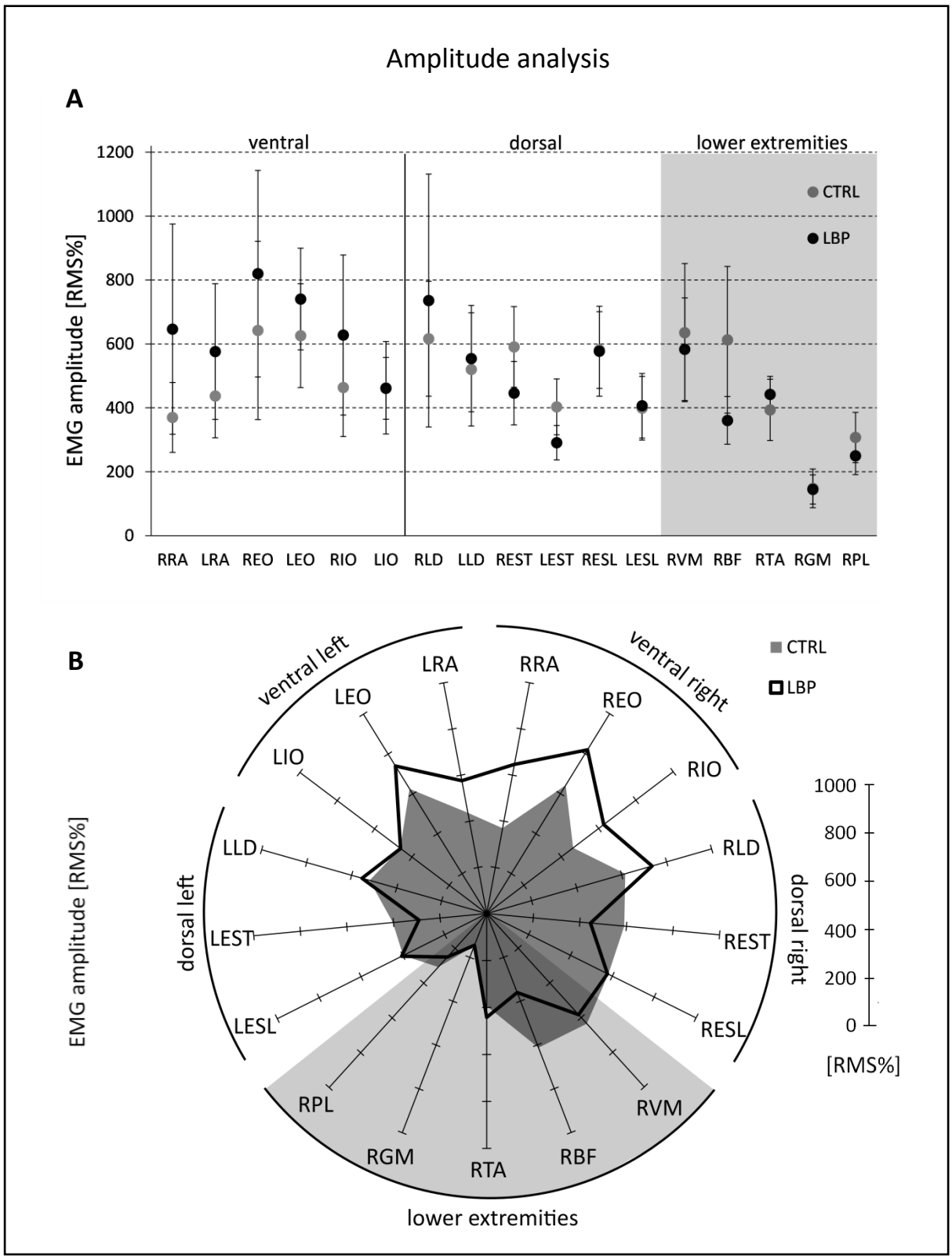

Figure 26: EMG amplitudes (RMS%, normalized to full stride of unperturbed walking) for low back pain (LBP) and controls (CTRL) according to chronic pain grades; data presented for individual muscles with means (A, B) and 95% confidence intervals (A); RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RGM: right M. gastrocnemius medialis, RTA: right M. tibialis anterior, RPL: right M. peroneus longus

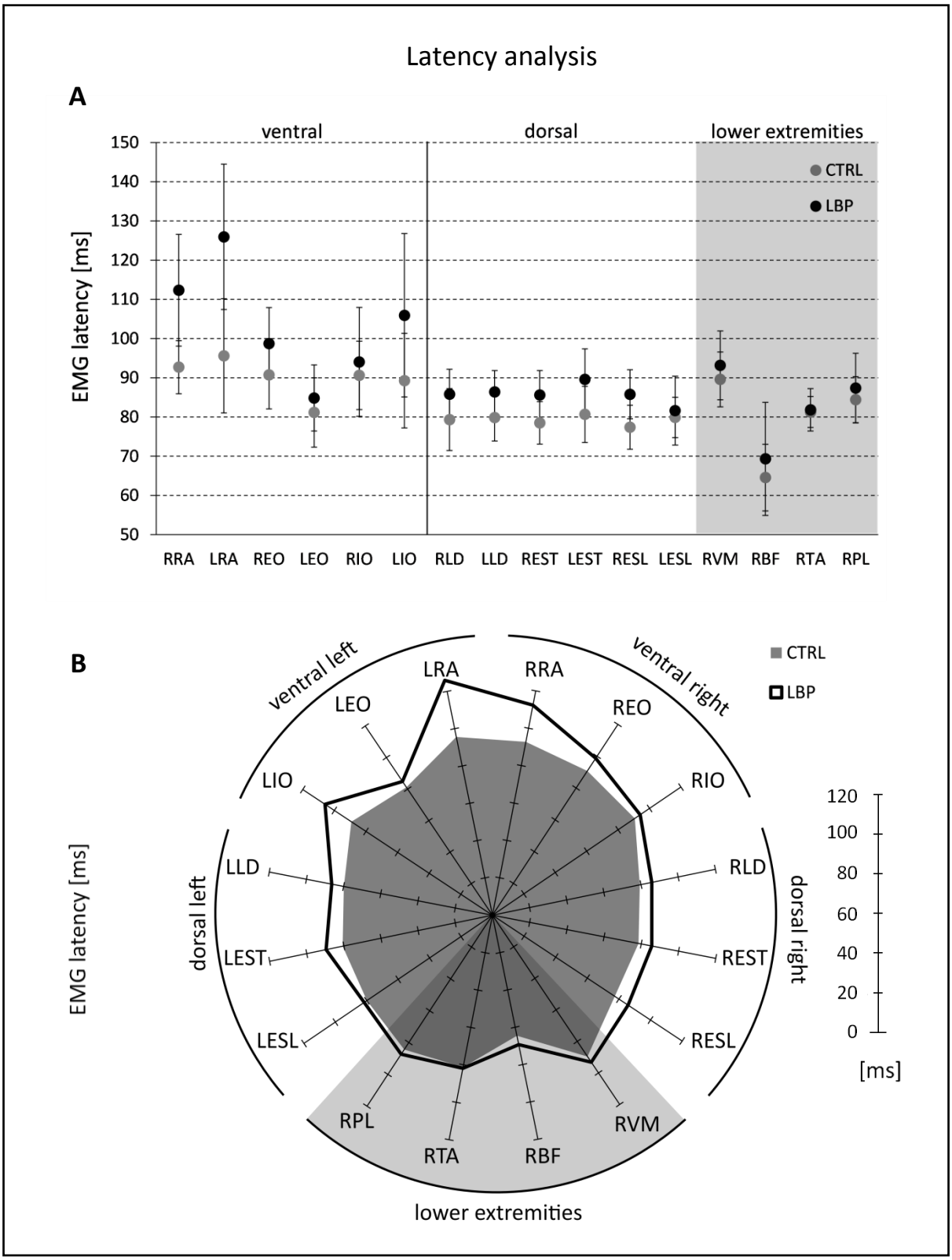

Figure 27: EMG latencies (ms) for low back pain (LBP) and controls (CTRL)according to chronic pain grades; data presented for individual muscles with means (A, B) and 95% confidence intervals (A); RRA/LRA: right/left M. rectus abdominis, LEO/REO: right/left M. externus obliquus, LIO/RIO: right/left M. internus obliquus, RLD/LLD: right/left M. latissimus dorsi, REST/LEST: right/left M. erectus spinae (thoracic), RESL/LESL: right/left M. erectus spinae (lumbar), RVM: right M. vastus medialis, RBF: right M. biceps femoris, RTA: right M. tibialis anterior, RPL: right M. peroneus longus

Results of pooled muscles (ventral right, ventral left, dorsal right, dorsal left, upper leg, lower leg) for amplitudes and latencies based on CPG classification are shown in table 21 figure 28.

Table 21: EMG amplitudes and latencies for CTRL and LBP – grouped muscles (CPG classification)

Muscle group	**EMG amplitudes [RMS%]**				**EMG latencies [ms]**			
	CTRL	**LBP**	**MANOVA**	**Post hoc**	**CTRL**	**LBP**	**MANOVA**	**Post hoc**
ventral right	492 ± 225	698 ± 423			93 ± 9	102 ± 16		
ventral left	509 ± 148	593 ± 198			88 ± 14	108 ± 27		
dorsal right	596 ± 163	622 ± 363	F=1.44 p=0.28		79 ± 8	86 ± 9	F=0.88 p=0.54	
dorsal left	441 ± 155	417 ± 121			80 ± 8	86 ± 11		
upper leg	624 ± 254	472 ± 151			77 ± 9	83 ± 21		
lower leg	350 ± 73	346 ± 76			82 ± 7	84 ± 11		

CTRL: control group; LBP: low back Pain; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg: right M. vastus medialis, right M. biceps femoris; lower leg: right M. tibialis anterior, right M. peroneus longus

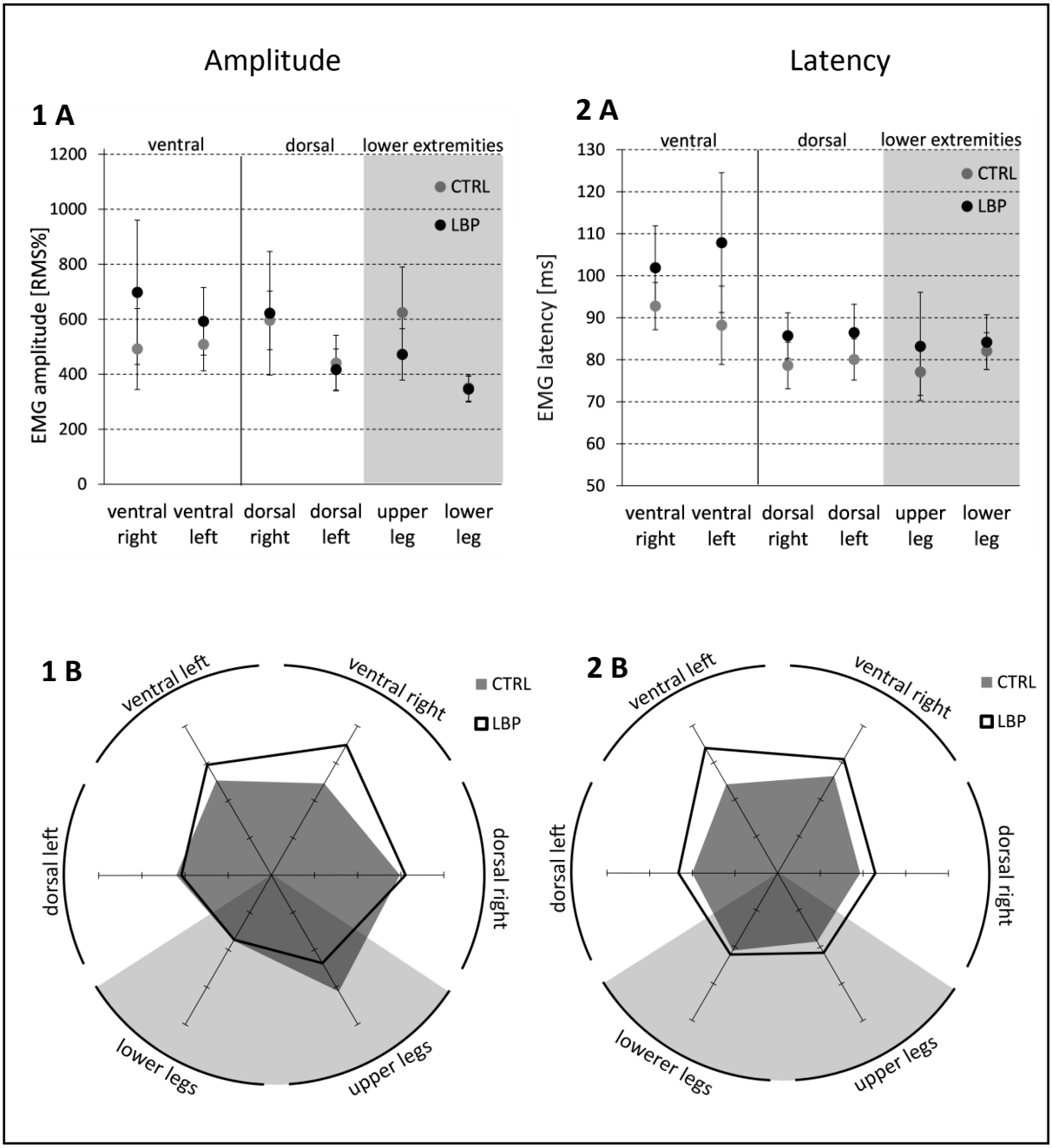

Figure 28: EMG amplitudes (1A,1B) and latencies (2A, 2B) for low back pain (LBP) and controls (CTRL) according to chronic pain grades; data presented for grouped muscles with means (1A, 1B, 2A, 2B) and 95% confidence intervals (1A, 1B); asterisks indicate significant differences; ventral right: right M. rectus abdominis, right M. externus obliquus, right M. internus obliquus; ventral left: left M. rectus abdominis, left M. externus obliquus, left M. internus obliquus; dorsal right: right M. latissimus dorsi, right M. erectus spinae (thoracic), right M. erectus spinae (lumbar); dorsal left: left M. latissimus dorsi, left M. erectus spinae (thoracic), left M. erectus spinae (lumbar); upper leg: right M. vastus medialis, right M. biceps femoris; lower leg: right M. tibialis anterior, right M. peroneus longus

Latency comparisons and co-contraction ratios of muscle activity and muscle between selected muscle pairs are shown in table 22 and 23.

Table 22: EMG activation onset pattern between selected muscles pairs

Comparison	Muscle pair	EMG onset differences [ms]		
		CTRL	LBP	t-test (p)
right vs. left ventral	RRA - LRA	7 ± 7	-5 ± 17	0.102
right vs. left dorsal	RESL - LESL	-2 ± 5	2 ± 14	0.500
ventral only	RA - EO	10 ± 6	21 ± 8	0.015
dorsal only	EST - ESL	2 ± 5	1 ± 6	0.739
ventral vs. dorsal	RA - ESL	14 ± 9	32 ± 17	0.044
ventral vs lower leg	RA - TA	10 ± 6	34 ± 17	0.006
dorsal vs. lower leg	ESL - TA	-2 ± 7	2 ± 10	0.303

Delay of muscle onset (muscle 1 - muscle 2) for selected muscle comparisons for low back pain (LBP) and controls (CTRL); data presented in mean±SD; RRA: right M. rectus abdominis, LRA: left M. rectus abdominis; RESL: right M. erectus spinae (lumbar); LESL: left M. erectus spinae (lumbar); RA: averaged right and left M. rectus abdominis; EO: averaged right and left M. externus obliquus; EST: averaged right and left M. erectus spinae (thoracic); ESL: averaged right and left M. erectus spinae (lumbar); TA: right M. tibialis anterior

Table 23: EMG co-contraction ratios between selected muscles pairs

Comparison	Muscle pair	EMG amplitude ratios [arb. unit]		
		CTRL	LBP	t-test (p)
right vs. left ventral	RRA : LRA	0.9 ± 0.3	1.0 ± 0.4	0.329
right vs. left dorsal	RESL : LESL	1.6 ± 0.6	1.6 ± 0.8	0.989
ventral only	RA : EO	0.8 ± 0.5	0.8 ± 0.5	0.897
dorsal only	EST : ESL	1.0 ± 0.2	0.9 ± 0.4	0.168
ventral vs. dorsal	RA : ESL	0.9 ± 0.4	1.3 ± 0.9	0.186
ventral vs lower leg	RA : TA	1.3 ± 1.1	1.4 ± 1.1	0.792
dorsal vs. lower leg	ESL : TA	1.5 ± 0.8	1.1 ± 0.4	0.288

Co-contraction ratios (muscle 1 : Muscle 2) for selected muscle comparisons for low back pain (LBP) and controls (CTRL); data presented in mean±SD; RRA: right M. rectus abdominis, LRA: left M. rectus abdominis; RESL: right M. erectus spinae (lumbar); LESL: left M. erectus spinae (lumbar); RA: averaged right and left M. rectus abdominis; EO: averaged right and left M. externus obliquus; EST: averaged right and left M. erectus spinae (thoracic); ESL: averaged right and left M. erectus spinae (lumbar); TA: right M. tibialis anterior

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13 AFFIDAVITS

According to the doctoral degree regulations (§ 4 (2), sentences No. 4 and 7) of the Faculty of Human Sciences, University of Potsdam:

I hereby declare that this thesis entitled “Motor control strategies in response to unexpected disturbances of dynamic postural control in people with and without low back pain” is the original work of the author. I did not receive any help or support from commercial consultants. All sources and/or materials applied are listed and specified in the thesis. All parts or single sentences, which have been taken analogously or literally from other sources, are identified as citations. Furthermore, I declare that this thesis or parts of the thesis have not yet been submitted for a doctoral degree to this or any other institution neither in identical nor in similar form.

Place, Date	Tilman Engel