Aus der Klinik für Anästhesiologie
(Prof. Dr. med. K. Meissner)
der Medizinischen Fakultät der Universität Göttingen

The effect of acute evoked muscular pain at the lower back on motion capture and surface EMG during a repetitive movement task

INAUGURAL-DISSERTATION

zur Erlangung des Doktorgrades
der Medizinischen Fakultät der
Georg-August-Universität zu Göttingen

vorgelegt von

Franziska Butterwegge

aus

Kassel

Göttingen 10.07.2021

Dekan:	Prof. Dr. med. W. Brück

Betreuungsausschuss

Betreuer/in	Prof. Dr. med. F. Petzke
Ko-Betreuer/in:	Prof. Dr. med. M. Sommer

Prüfungskommission

Referent/in	Prof. Dr. med. F. Petzke
Ko-Referent/in:	Prof. Dr. med. M. Sommer
Drittreferent/in:	Prof. Dr. mult. T. Meyer

Datum der mündlichen Prüfung: 09.06.2022

Hiermit erkläre ich, die Dissertation mit dem Titel "The effect of acute evoked muscular pain at the lower back on motion capture and surface EMG during a repetitive movement task" eigenständig angefertigt und keine anderen als die von mir angegebenen Quellen und Hilfsmittel verwendet zu haben.

Göttingen, den …… ……

(Unterschrift)

Table of Content

List of Figures

List of Tables

List of Abbreviations

ADC	Analog to digital converter
ARV	Average rectified value
BL1	Baseline 1
BL2	Baseline 2
C	Cervical vertebra
CNS	Central nervous system
CoV	Coefficient of variations
CPM	Central inhibition of pain
CV	Conduction velocity
DASS	Depression-Anxiety-Stress-Scale
DFNS	Deutscher Forschungsverbund Neuropathischer Schmerzen
EIH	Exercise induced hypoalgesia
EMD	Electromechanical delay
EMG	Electromyography
HDs-EMG	High-density surface electromyography
HYP	Hypertonic saline injection
ISO	Isotonic saline injection
L	Lumbar vertebra
LBP	Low back pain
LED	Light emitting diodes
LL	Lateral left column
LR	Lateral right column
M.	*Musculus*
MDF	Median frequency
ML	Medial left column
MNF	Mean frequency
MR	Medial right column
NRS	Numeric rating scale
PPT	Pressure pain threshold
Procc.	*Processus (pl.)*
QST	Quantitative sensoric testing
RKI	Robert Koch Institute
RM-ANOVA	Repeated measurement analysis of variance

RMS	Root mean square
SEC	Serial elastic components
SF-12	Short form 12

1 Introduction

1.1 Relevance of low back pain

Low back pain (LBP) is one of the most common reasons for consulting health care services, as "70-85 % of all people have back pain at some time in life" (Andersson 1999). The annual incidence of LBP within the general population is estimated around 18 % (Cassidy et al. 2005). LBP is the leading reason for work incapacity, hospitalisation, and permanent disability. Although 90 % of all patients recover within nine months, many of them have a recurrence of LBP. Patients who do not recover in this time are most likely to get well very slowly (Andersson 1999). Overall, LBP causes costs of about nine billion Euros per year (Raspe 2012). According to a study of the health insurance BARMER in 2007 in Germany (Plamenig 2018), the costs per patient vary between 661 EUR (acute) and 6319 EUR (chronic). However, not only medical treatment but also loss of workdays and disability cause high costs.

A statistic of the health insurance BARMER (Plamenig 2018) states that the musculoskeletal diseases caused 26.5 % of all work incapacity in 2012 whereof back pain is the leading reason. Data from the "Bundesanstalt für Arbeitsschutz und Arbeitsmedizin" (Figure 1) show similar results (BAuA 2015).

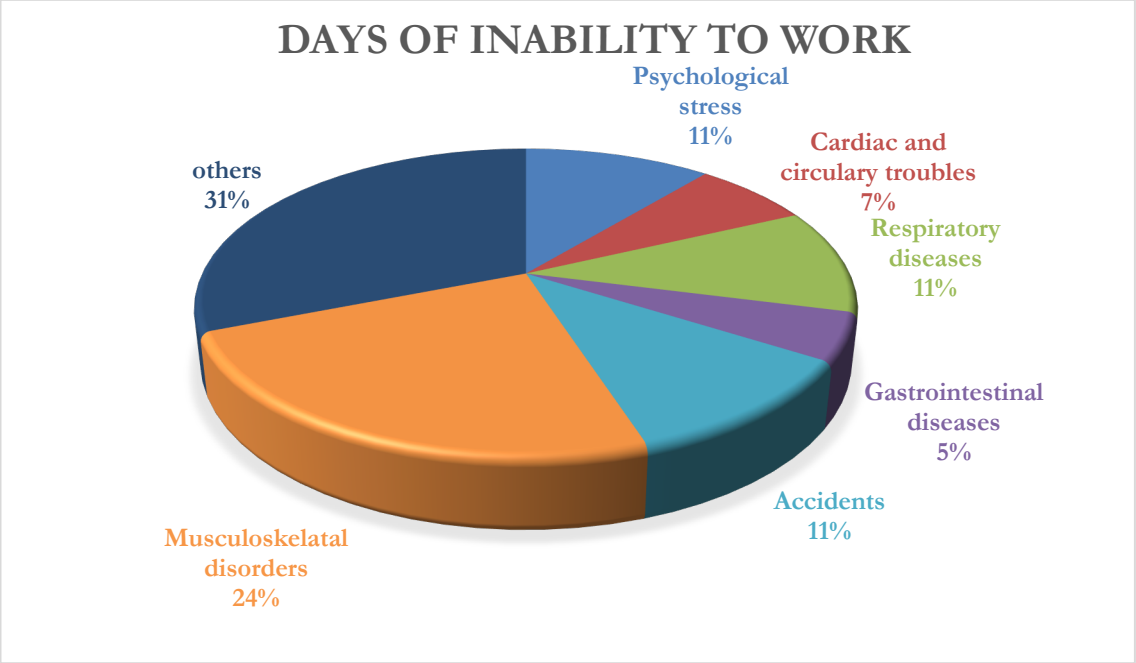

Figure 1: Percentages of days of inability to work, data adopted from BAuA (2015)

According to the Robert Koch Institute (RKI), every fifth woman und every seventh man in Germany suffer from chronic LBP. This represents a global trend, as "Worldwide, the Global Burden of Disease Study 2010 now ranks LBP first […] in years lived with disability" (Vos et al. 2012).

Men have a higher risk of recurrence than women. In general, people aged 25 to 44 years have the highest rate of recurrence (Andersson 1999).

LBP and the risk of chronicity can develop in early life (Vasseljen et al. 2013). According to the survey of the BARMER (Plamenig 2018), in 2013 about 16 % of the interviewed students had back pain during the last six weeks. "There is good evidence that more than 50 % of the general population have already experienced an episode of LBP before the end of adolescence" (Cassidy et al. 2005).

The survey of the RKI shows that even small children are affected. This shows that LBP is not only an issue for adults representing a sign of wear but a current and important topic for the whole society.

In any case, given its high prevalence the prognosis for LBP is quite positive. Most patients recover within a few weeks, 60-70 % recover within six weeks, and 90 % within twelve weeks (Andersson 1999).

Recovery beyond this time is rather poor and long lasting. 68-86 % return to work within one month, 93 % within six months (Pengel 2003). Improvement is rapid within the first month; smaller improvement occurs until three months (Pengel et al. 2003).

One problem is that recurrence is quite likely. 73 % of the patients have at least one recurrent period of back pain within twelve months (Pengel et al. 2003). The longer the recovery lasts, the more likely it comes to a chronification (Balagué et al. 2007). The best predictor for chronic LBP is a history of LBP (Cholewicki et al. 2005), and "5 % of people with an acute episode of low back pain develop chronic low back pain and related disability" (Koes et al. 2006).

Considering the aforementioned high rates of acute and chronic LBP and its economic consequences including the cost of medical interventions to prevent or reverse chronicity, it becomes obvious that early diagnosis and appropriate treatment have great importance. This requires an understanding of the in vivo physiological and pathophysiological mechanisms related to the experience of (acute) back pain. Thus, the ultimate aim of this thesis is to contribute to the understanding of the effects of an acute painful episode on the neuromuscular, kinematic and biomechanical aspects of lower back function.

1.2 Somatic and neurodegenerative causes of LBP

It has long been documented that only 10-15 % of low back pain has a specific somatic cause (Deyo et al. 1992). The predominant reasons among those are degenerative changes due to old age, osteoporotic compression fractures and disc prolapse (Müller 2001). On the other hand, the majority of diagnosed LPB (85-95 %) does not reveal any specific somatic causes. They are classified as non-specific low back pain (Deyo et al. 1992; Koes et al. 2006). Although the prognosis is quite good (see 1.1), different risk factors can lead to the development of chronic pain (Bundesärztekammer and Kassenärztliche Bundesvereinigung 2017). On the one hand so-called "yellow flags" such as psychological and social aspects are highly associated with chronic LBP. A low social status, depression, dissatisfaction and different coping strategies are assumed to lead to recurrence and persistence of LBP (Lethem et al. 1983; Carroll et al. 2004; Geyer et al. 2006). On the other hand an impaired motor control and delayed reaction to sudden movement of the trunk related to loss of functional capacity due to pain increases the risk of tissue injury (Cholewicki et al. 2005). Moreover, lumbar shear forces, disc compression, repetitive and sustained low level as well as heavy

work are associated with LBP (Kerr et al. 2001; Hodges and Smeets 2015). To this respect, it can be concluded that acute and chronic LBP have a multifactorial nature. Many studies agree that the best predictor for chronic LBP is a history of LBP (Bigos et al. 1992; Greene et al. 2001; Kerr et al. 2001).

1.3 Influence of chronic pain on muscle activity, force output and movement

It has been shown that neuromuscular activity of trunk muscles is altered during pain (Falla et al. 2007a; van der Hulst et al. 2010). This altered neuromuscular activity emerges with a deviant motor control and motion strategies in the persistence or recurrence of low back pain (Falla et al. 2014). Patients with LBP show an impaired muscle reaction to sudden movement which is assumed to hold a risk for injury (Hodges and Richardson 1999; Cholewicki et al. 2005). Moreover, they exhibit trunk stiffening by a co-activation of muscles in order to minimize the forces on the trunk, which means that most muscles are permanently active during a task (Boston et al. 1995; Jacobs et al. 2011; Jones et al. 2012b). Their movement is less coordinated compared to healthy controls, they move slower and show a decreased endurance in work as well as less willingness to apply maximum force, as studies show that they could reach higher forces when they were challenged (Boston et al. 1995; Jones et al. 2012b). However, it is difficult to differentiate neuromuscular consequences of pain from psychological aspects like fear of movement and early onset fatigue (Falla et al. 2007b).

Thus, when investigating the processes that take place in clinical LBP there are many confounding aspects including psychological variability and systemic reactions (Reddy et al. 2012). Moreover, the variability of dysfunction makes it difficult to guarantee an adequate level of reproducibility. In addition to this it is hardly possible to select "matched" healthy controls to the LBP patients because of high individual intrinsic variability of movement (Ross et al. 2015).

In summary, many studies have investigated the kinesiology of chronic and acute low back pain and suggest various mechanisms underlying the deviant motor control and motion strategies, most likely due to the high variation of symptoms and reactions among the patients (Hodges et al. 2013). Therefore, examining the contribution of individual mechanisms influenced by the experience of pain requires a standardized experimental set

up to study. The induction of controlled experimental pain is a well-established method that provides these steady state conditions (Graven-Nielsen et al. 1997a; Falla et al. 2007b; Falla et al. 2007a). It allows to investigate a cause-effect relationship between pain and (dys-)function that is mediated by kinematic and neuromuscular adjustments during painful movement (Graven-Nielsen and Arendt-Nielsen 2008).

1.4 Experimental pain

Experimental pain gives the opportunity to investigate pain mechanisms in controlled settings under standardized conditions (Reddy et al. 2012). For inducing pain, different methods are applied. One of the commonly used methods is electrically stimulating a particular dermatome in order to activate nociceptive receptors. However, this is an unselective method that may lead to an unnatural synchronic excitation of afferent nerve fibres (Handwerker and Kobal 1993). A faster and more precise method is stimulating noxious receptors with ultrasound, but the exact effects are poorly understood (Handwerker and Kobal 1993). Thermal stimulation is an alternative method, however, the reliability and accuracy is doubtable (Handwerker and Kobal 1993; Reddy et al. 2012). One method is to apply laser-generated heat pulses on the superficial skin which excite the free nerve ends of nociceptive fibres (Truini et al. 2004). Studies show that the laser evoked potentials have a high correlation with the reported pain (Bromm and Chen 1995). Other studies use CO_2 Laser to investigate the neurophysiological pathways of pain (Valeriani et al. 2003). However, this is mostly used for investigations different from the study at hand.

Instead, more "natural" noxious stimuli can be used. Mechanical stimulation is one of the oldest forms of experimental pain whereof induction of pressure is the most popular one (Handwerker and Kobal 1993). It is for example one part of the QST battery, which is used as a standardized method to investigate different sensory aspects (Rolke et al. 2006a). One of the main shortcomings is the difficulty to control the rapidity of onset and termination of pain. Moreover, it is quite unspecific since deeper tissues might be co-activated (Reddy et al. 2012).

For examining acute pain related to LBP, stimulating noxious muscle afferents with the intramuscular injection of chemical substances like capsaicin, glutamate and hypertonic saline has been accepted as an experimental analogue of acute muscular pain (Graven-Nielsen et

al. 1997a; Reddy et al. 2012). Capsaicin evokes pain and hyperalgesia by either injecting or applying it topically on the skin (Reddy et al. 2012). It stimulates the TRPV1-nociceptor with only little effect on other somatosensory modalities (Ross et al. 2015). Hypertonic saline is often used because its effects are comparable to acute clinical pain. "Although hypertonic saline injection is unlikely to simulate all aspects of mechanical LBP, it provides a model to investigate the effect of nociceptor stimulation and pain […]" (Hodges et al. 2003). Multiple previous studies showed that the injection of hypertonic saline leads to pain sensation around the injection point as well as typical patterns of referred pain. Thus, experimental pain reproduces aspects of pain sensation similar to clinical pain (Graven-Nielsen et al. 1997b). Another advantage of experimental pain is that the pain can be focused on the region of interest (Graven-Nielsen et al. 1997b; Graven-Nielsen and Arendt-Nielsen 2008).

1.5 Pain and motor control of movement

The complex process of motion needs an adequate recruitment of motor units in the musculature, the correct timing and a sufficient amount of muscle force. While healthy people show a quite homogenous course of action during motion it is highly variable in patients with LBP (Radebold et al. 2000).

Numerous studies have investigated the processes that take place in the muscles of patients with LBP. Increased stiffening of the spine (Hodges et al. 2013), increased trunk stiffening during unexpected movements (Jones et al. 2012a), increased activity of the M. erector spinae during gait (Lamoth et al. 2006), less relaxation (van der Hulst et al. 2010) and a redistribution of activity (Graven-Nielsen et al. 1997a; Hodges et al. 2013) are phenomena that are observed when LBP is present.

Different theories try to explain these changes in motor control. The vicious cycle theory states that pain and muscle spasms affect each other. Under pain the muscle would stiffen in a stereotypical way (independent from the required task) which leads to ischemia. As a result, metabolites accumulate and worsen the pain. This theory assumes that the muscle activity increases with pain. However, it cannot explain a reduced activity in some parts and non-systematic changes (Johansson and Sojka 1991).

The pain adaption theory assumes that pain leads to an inhibition of agonistic and an excitation of antagonistic muscles, which results in a decreased activity in painful muscles, less force and a reduced movement velocity (Lund et al. 1991). Still, it cannot explain non-

systematic changes. On the other hand, a new theory for motor adaption to pain assumes that the brain uses a multimodal approach to cope with pain (Hodges and Tucker 2011). The central aspect in this model is the intention to protect the "damaged area" from further pain. Changes take place on different levels of the motor system. The redistribution of muscle activity can reduce the pressure on the painful areas. Changes in mechanical behaviour like stiffening occur. Although there are fundamental features, the exact behaviour of the motor system is variable between individuals and dependent on the patient as well as on the required task. Accordingly, investigations show that individual patients use different strategies to avoid pain (Hodges et al. 2013). The choice of the strategy is influenced by habitation, body composition, location of pain etc. Patients use the same movement strategy for any specific task, which provides short-term benefit since they reduce the pain and reduce the load on damaged tissues. Nevertheless, long-term consequences of this mechanism are unfavourable, causing an overloading of neighbouring parts and worsening or spatially extending the pain. While pain is the stimulus for pain adaption, there is no stimulus terminating adaption (Hodges and Tucker 2011), which means that some patients seem to retain the stiffening mechanism even after the healing process is completed (Hodges et al. 2013).

Healthy people show a change in the distribution of activity when performing repetitive or monotonous tasks (Farina et al. 2008; Tucker et al. 2009). This is important especially in muscles like the M. erector spinae which experiences repetitive or constant activation over long times. The redistribution is sensible to avoid overload (Andersson and Ortengren 1984). Furthermore, variability is necessary to keep up motor output under different conditions, for example fatigue or pain (Farina et al. 2008). Consequently, reduced variability as a continuing dysfunction of the trunk muscles is estimated to have an impact on recurrence and persistence of LBP (Cholewicki et al. 2005). It is supposed that especially repetitive tasks hold a high risk for neuromuscular irritation (Lu et al. 2008).

1.5.1 Examples of experimental pain investigation

Previous studies have already investigated the effect of experimental pain on motion and muscle activation. Examinations of the M. trapezius during shoulder flexion show that in acute pain reorganization of the activation pattern occurs (Falla et al. 2007a). Moreover, the motion strategy is altered compared to healthy controls (Falla et al. 2007a). Experimental pain applied to the Mm. tibialis anterior and gastrocnemius in a static task and during gait

leads to an increased activation of the antagonistic muscles and a decreased activation of the agonistic muscles, which is interpreted as a protection mechanism for the painful area (Graven-Nielsen et al. 1997a). Similar changes are observed during unanticipated postural perturbations. In this case, a unilateral injection of hypertonic saline leads to a bilateral delay in activation onset of the M. erector spinae and a bilateral reduction of EMG amplitude compared to healthy participants (Boudreau et al. 2011). With regard to these findings the major interest of the study at hand is to analyse the behaviour of muscle activation, especially the onset, the distribution and the intensity of activity during acute experimentally induced LBP (also see 1.8).

1.6 Methodological background

1.6.1 Pressure pain threshold

Quantitative sensory testing (QST) is a non-invasive method to test the somatosensory nervous system. It is used to diagnose different forms of musculoskeletal and neuropathic pain disorders since it shows a full somatosensory phenotype of the patient. It usually combines various measures in a test battery to investigate the different fibres that are responsible for the perception of somatosensory sensation. It focuses primarily on innervation of the skin investigating touch, vibration, heat and cold perception as well as pin prick sensation, identifying and quantifying loss (like thermoanaesthesia, mechanical analgesia) and gain of function (like wind-up, hyperalgesia and allodynia). Deep tissues are only assessed by pressure pain thresholds (PPTs).

QST is a semi-objective method because it is dependent on the cooperation and judgement of the patient. Moreover, the situation as well as the behaviour of the investigator have an influence on the data. The respective sensory thresholds are influenced by the region tested as well as age and sex of the patient (Rolke et al. 2006a; Rolke et al. 2006b; Pavlaković and Petzke 2010). To standardize this method, the DFNS (Deutscher Forschungsverbund Neuropathischer Schmerzen) developed a QST battery which was validated in earlier publications (Rolke et al. 2006a).

Changes in perception can be a sign for central and/or peripheral sensitization. While psychological, demographic and structural factors alone often fail to explain the cause for LBP, recent studies suggest that there is connection between LBP and a change in central

pain processing (Giesecke et al. 2006). It has been shown that healthy people with acute pain show an increased activity only in the brain region involved in acute pain, whereas chronical pain patients show a high activity in the emotion-related circuity (Hashmi et al. 2013). The critical turning point seems to be approximately after one year of lasting pain (Hashmi et al. 2013).

It is assumed that central sensitization is caused by a lack of descending pain inhibition and an increased excitability. Changes in the dorsal horn of the spinal cord may cause a hyperexcitability. Possible results are hyperalgesia in the muscle as well as referred pain (Cruccu and Truini 2006; Pavlaković and Petzke 2010).

The so-called peripheral sensitization is mediated via C-fibres (Cruccu and Truini 2006). A flow of metabolic and chemical substances around a nociceptor causes a hyper response. This means, the nociceptor produces a greater action potential than normally when being stimulated or responds to lower stimulation intensities.

For this study only changes in the pressure pain threshold at the lower back were measured. Clinical examinations have proven an increased generalized sensitivity for pressure pain (mechanical sensitization) in patients with LBP (Giesecke et al. 2006), others a more localized sensitivity depending on the extent of clinical pain (Gerhardt et al. 2016). According to Greenspan (Cruccu and Truini 2006) pressure allodynia (or better hyperalgesia) is caused by a peripheral sensitization with a central component via C-fibres and a central sensitization via Aδ-fibres.

1.6.2 Surface electromyography signal and interventions

In clinical routine surface electromyography (EMG) is used to detect and analyse pathological processes in the muscles. Moreover, one can distinguish myopathic and neuropathic origin of neuromuscular disorders (Mumenthaler and Mattle 2006; Berlit 2013). Surface EMG is a measure of electrical activity of the muscles which is commonly used to interpret the excitability of the interested muscle in different conditions as well as during pain (Hodges et al. 2003; Kramer et al. 2005; Hodges et al. 2013; Falla et al. 2014). The features/variables extracted from EMG signal, such as onset, amplitude and mean frequency, can be used to understand changing muscle activity during acute (i.e. fatigue, acute pain) and chronic (i.e. sport exercise, aging, chronic pain) states (Merletti and Farina 2016b).

The surface EMG signal is a representation of the electrical potential field generated by the depolarization of the muscle fibre membrane - the sarcolemma (Merletti and Farina 2016a). These electrical changes are detected by the electrode which is fundamentally an electrochemical transducer. The shape of the surface EMG signal depends on two fundamental factors:

a) Physiological factors:

The muscle anatomy, the composition of the motor units that are recruited in certain contractions, the statistical distribution of muscle fibre conduction velocity, blood flow and temperature can change the amplitude and the frequency content of surface EMG (Merletti et al. 2001). A thick tissue (like subcutaneous fatty tissue) between muscle and electrode, for example, reduces the amplitude and the signal bandwidth (Merletti and Farina 2016b). The amplitude of the EMG shows the "activity" of the muscle. It represents the overlapping of the action potential of all recorded motor units. When more motor units are recruited and the firing rate increases the amplitude rises (Konrad 2011). This means that the EMG amplitude increases with increasing contraction force in relation with the number of recruited motor units. The EMG signal is a non-periodic signal that carries the fundamental discharge frequencies of motor units modulated by various neural drives from different volleys. Their allocation can be seen in a frequency power spectrum. The mean or median frequency of this spectrum indicates the changes in the combination of neural drive contributing to various muscle contraction strategies (Konrad 2011). For example, development of muscle fatigue leads to a shift in mean and median frequencies (Gilmore and Meyers 1983).

b) Electrical factors:

The impedance of the electrode skin surface couple, the recording mode (i.e., monopolar or differential), the specifications of the electronic system that is used to acquire EMG signals affect the amplitude and frequency content. Beside the EMG signal from the muscle, non-biological signals as well as the signals from other muscles are recorded. These unwanted disturbances to the signal of interest are described as noise. In order to maximize the fidelity of the signal, these undesirable surrounding signals are filtered.

1.6.3 Recording EMG signal

Voluntary activated surface EMG is a stochastic (random) signal characterized by its amplitude and its energy (frequency bandwidth). The amplitude of the signal can range from 0 to 10 mV (peak-to-peak). The full frequency bandwidth of the EMG signal is between 20 and 500 Hz. This implies that the energy spectrum of a surface EMG signal would involve all the frequencies in this range. These signal characteristics are considered by the design of the recording system. A basic recording system consists of (Figure 2): 1) sensors (electrodes), 2) an amplifier for increasing the power of the signal with a certain gain factor, 3) an electronic filter for attenuating the power of undesirable signals (noise) contributing to the raw EMG signal, 4) an analogue to digital converter (ADC) for recording the EMG signal with a computer for interpretation/analysis.

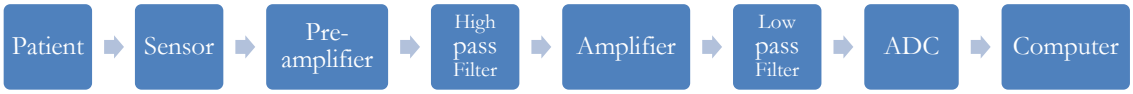

Figure 2: Components of EMG recording

The most critical aspects of recording are the electrode design and the recording modality. There are two recording modalities: the differential mode implies detecting EMG signals at two different locations. The amplifier subtracts two signals and then amplifies with a certain gain. Thus, any signal that originates far away from the detection sites will be a common signal (time independent), whereas signals in the immediate vicinity of the detection surfaces will be different and consequently will be amplified (De Luca 2002). The alternative mode is monopolar recording. This technique implies amplifying the difference between one detection site and the ground (non-active recording site). The differential modality provides a higher signal-to-noise ratio (stronger noise elimination), however the EMG signals that originates from relatively far (deeper) muscles will be attenuated as well, depending on inter electrode distances (Beck et al. 2007). On the other hand, the monopolar recording enables us to record from relatively deeper muscles, however with a lower noise-to-signal ratio (Merletti et al. 1990).

1.6.4 EMG variables

Surface EMG is a stochastic signal that is generated by convolved and filtered muscle fibre compound action potentials. Due to the stochastic nature of the EMG signal, its amplitude and energy cannot be measured directly. Therefore, an EMG signal is quantified by some descriptive variables (Konrad 2011).

The effective amplitude of the EMG signal is defined by root mean square (RMS) or the average rectified value (ARV) (Merletti and Farina 2016; Merletti et al. 1990). While ARV is an estimation for amplitude deportment, RMS reflects the mean power of the signal (Luca 2002; Konrad 2011). Essentially, both values indicate the activity (excitation) level of the interested muscle during a specific task, contraction type or condition (i.e. pain, fatigue) (Konrad 2011). Both values are related to the area under the curve and the mean power of the signal within a specified time window and allow a description of amplitude variations (Merletti et al. 1990).

The spectral density of surface EMG exhibits the distribution of frequency content of the signal during sustained voluntary contraction. The amplitude of the spectrum shows the energy of EMG signal in the relevant bandwidth. Additionally, two spectral indices mean (MNF) and median frequency (MDF) are utilized as appropriate indicators of spectral compression (Merletti et al., 1990). Changes in these indices over time can be associated with changes in the activity of muscle fibres contributing to muscle contraction during sustained contraction. For example, a reduction in MNF or MDF is interpreted as less contribution of fast-twitch type muscle fibres during sustained contraction. This phenomenon is typically observed during muscle fatigue (Merletti et al. 1990).

Together with MDF, MNF and RMS, muscle fibre conduction velocity (CV) is a variable indicating the change in muscle contraction strategy. During a long sustained contraction a decrease in MNF, MDF and CV is accompanied by an increase in ARV and RMS (Merletti et al. 1990). This happens because the spectral density is compressed to the lower frequencies over time (Gilmore and Meyers 1983; Merletti; and Parker 2013; Merletti and Farina 2016b) and impairment in action potential propagation (and consequently in CV) by fatigue. (Potvin and Bent 1997; Konrad 2011).

1.6.5 Advanced electromyography method: high-density surface EMG

The intention of EMG is to get information about the myoelectric signal. The big shortcoming of the often-used single sampling electrodes is that they are only able to present the temporal aspects of activation. Moreover, their signal is dependent on their location on the muscle of interest (e.g., near innervation zones etc.). Previous studies have shown that the muscle does not activate in a homogeneous way, but is dependent on joint position and contraction conditions (Rojas-Martínez et al. 2012). Since single electrodes are unable to give information about the spatial distribution of activity the physiological propagation of activity cannot be measured. Due to the loss of information misleading and conflicting conclusions might be drawn (Rojas-Martínez et al. 2012).

In contrast, high-density surface electrodes are able to overcome this problem. They consist of panels with multiple, closely spaced electrodes. This design (Figure 18) offers a topographical overview to observe the localization of activation in the muscle. The advantage is that changes in distribution of activity, the location of maximal amplitude and information of the direction and origin of the action potentials can be detected and analysed. Moreover, information on the neuromuscular strategies of the CNS can be derived (Zwarts and Stegeman 2003; Merletti et al. 2008; Rojas-Martínez et al. 2012). The importance of this novel technique is proved by the heterogenic behaviour of motor units under different conditions and in different individuals (Falla et al. 2007c; Tucker et al. 2009).

1.7 Investigating the movement of body segments

Everyday observations demonstrate that pain has an effect on movement. Several studies have investigated the effect of pain-related dysfunction and changes in motion (Boston et al. 1995; Marras et al. 1995; Shum et al. 2007; Scholtes et al. 2009; Sánchez-Zuriaga et al. 2011; Falla et al. 2014). Motion capture systems are a well-established method to analyse human biomechanics and to study movement in the context of different activities (Sánchez-Zuriaga et al. 2011). This method has been widely used in the clinic, sports and entertainment industry (Sahrmann et al. 2017). There are different commercial systems available, like Vicon (Oxford Metrics Ltd., UK), Qualysis (Gothenburg, Sweden) and Motion Analysis (Motion Analysis Corp., Santa Rosa, CA, USA). The system consists of a number of cameras which track the position of markers that are placed on the body. These markers may be reflecting light, transmit LED or magnetic waves, which are received by the cameras. The markers are

typically placed on the joints of the human body. Their position and speed are measured. Moreover, the angle between two markers can be estimated to monitor the motion of the body and extremities.

Previous studies show significant differences in the motion of people with LBP and healthy controls during standardized tasks (Boston et al. 1995; Shum et al. 2007; Lee et al. 2011). Patients with LBP have a reduced velocity and acceleration in the sagittal plane when bending forward (Shum et al. 2007). During a lifting task people with LBP reveal a less smooth and coordinated motion. Moreover, they compensate a reduced trunk flexion using leg lift (Boston et al. 1995). This might be a result of either the pain itself or fear avoidance behaviour (Lee et al. 2011). According to Scholters et al., LPB patients also display more mobility in the lumbopelvic region, which can be interpreted as a degeneration of tissue resulting in instability (Scholtes et al. 2009). These changes are supposed to contribute to the development or persistence of LBP (Scholtes et al. 2009). The reduced velocity during extension has a sensitivity of 90 % and a specificity of 80 % to detect the presence of LBP (Lee et al. 2011). In addition to this, motion analysis serves as a quantitative evaluation of rehabilitative arrangements (Marras et al. 1995).

1.8 Aim of the study

A recent study (Falla et al. 2014) shows, that healthy subjects performing a repetitive lifting task over three minutes have a shift in the centre of activation of their muscle fibres in the erector spinae, while patients with non-specific LBP have less or no variation. Moreover, the pressure pain threshold before and after the task remains stable in the control group and decreases in the LBP group. This indicates that the motor control strategies to maintain task performance are different and probably less efficient in people with chronic LBP.

The intention of our study is to obtain further insights into the habituation of motor control under pain. Our aim is to investigate the effect of experimentally induced acute pain (using hypertonic saline) on the muscle activation pattern. The central questions we intend to answer are:

- Does acute pain induced in the lower back muscles result in an immediate reduction of the variability of activation as seen in chronic low back pain?
- Does this reduction correlate with the intensity of the experienced pain?
- Does acute pain cause changes in the course of movement and postural strategies?
- Does it have any effect on the local pressure pain sensitivity?

For this purpose, we use a 2-dimensional high-density EMG recording providing information about the distribution of muscle activity and changes of the activation pattern (Zwarts and Stegeman 2003). The functional status of the participant is measured by a motion capture system which allows a classification on the basis of the biomechanical analysis (Sánchez-Zuriaga et al. 2011). Pressure pain thresholds as described in a standard quantitative sensory testing (QST) protocol are used to investigate potential somatosensory changes (Rolke et al. 2006a; Pavlaković and Petzke 2010).

We assume that our results provide important information to improve the understanding of the pathophysiology of LBP, with relevance for treatment and prevention.

2 Material and Methods

The experimental protocol of the present study was approved by the Human Ethics Committee of the Universitätsmedizin Göttingen (number of application 13/12/15) in accordance with the Declaration of Helsinki. Each participant provided written informed consent prior to the conduction of the experimental session. All measurements took place in the laboratory of the Department of Neurorehabilitation Engineering, of the Bernstein Center of Computational Neuroscience.

2.1 Subject recruitment

In total, 26 healthy people were recruited via placard. A first screening took place via telephone when the inclusion criteria were examined. The participants had to be able to give their consent. Participants' age was between 18 and 55 years. Participants with no history of back pain or pain in the lower limb, for at least three years, were eligible. Any injuries, chronic pain or receiving pain-related medical treatment were criteria for exclusion. Additionally, they should not have any rheumatic, internal medical, neuro-musculoskeletal, or psychiatric disorders or a recent pregnancy. Participants on any pain reliever or medication that has effects on the CNS, were also excluded.

Prior to the experimental session, participants were informed about risks and benefit of the study as well as inclusion and exclusion criteria and asked to sign informed consent.

2.2 Protocol

2.2.1 Preparation

Prior to the experimental task, each participant was informed about the experimental protocol, related procedures and the instruments used. They were asked to wear clothes allowing for appropriate marker and electrode placement on the day of the experiment.

Subsequently the participant's ability to move was examined. The distance between fingers and floor when bending forward was measured. Moreover, measurements according to Schober and Ott were scanned standing and during flexion.
The participants were familiarised with the lifting task before electrode placements. The markers for the motion capture were placed first (see 2.3.3). Afterwards one electrode was placed on each side on the main body of the M. erector spinae/ M. multifidus lumborum. To do so the participant's' skin was pre-treated with alcohol pads and abrasive gel. If necessary, the hairs were removed by shaving to improve skin-electrode contact. Then the electrodes were placed and fixed with tapes. They were connected with the preamplifiers, which were connected to the amplifier. Two reference electrodes were placed on the Procc. spinosi over the upper edge of the electrode. In order to reduce moving artefacts, the preamplifiers were tied around the waist with a bandage.
Finally, the signal quality of electromyography and marker position was tested to ensure good electrode placement and camera setting of the motion capture system.

2.2.2 Experimental task

The experimental task implied lifting a box from a lower shelf at knee level (lateral epicondylus of femur was the reference point) to a higher shelf at shoulder level (acromio-clavicular joint was the reference point) and lowering back down and lifting again repetitively (Figure 3). While the participants performed this task, their pain sensation, based on a numerical rating scale (NRS), was asked at the very beginning of the task, every fifth cycle (5, 10, 15, 20, 25) of the lifting task and three minutes after they had finished the task. NRS range was between 0 corresponding to no pain and 10 to strongest possible pain. The participants were asked to lift a box (40x20x30 cm) with a weight of 5 kg from the lower shelf to the higher shelf within one second. They paused the box there for about three seconds while continuing to hold the box. The box was lowered after an acoustic trigger and held again at the lower shelf for three seconds. The rhythm and acoustic triggers of this task were given using a metronome that was set to 60 beats per minute. While the beats indicating lifting or lowering the box were in high tone, the beats for waiting at the shelf were in low tone. The task was repeated 25 times lasting about three minutes. The postural position while performing the task was standardized among subjects. Namely, the subjects were asked to lift or lower the box with merely bending the trunk. The knees were kept extended at all times. To ensure adequate task performance the subjects were shown it for a few times and

underwent a short test run until they felt familiar and comfortable with the timing and execution of the task. During the whole experimental session, the lifting task was repeated four times. The first run was performed without any injection, the two subsequent runs with or without an injection of normal saline in a balanced and randomized order. The final run followed an injection of hypertonic saline. After each run pain pressure thresholds (PPT) were measured. The details of PPT protocol is explained below.

The task is equivalent to an earlier study by our group (Falla et al. 2014) which showed that patients are able to complete the task without interruption due to fatigue or pain.

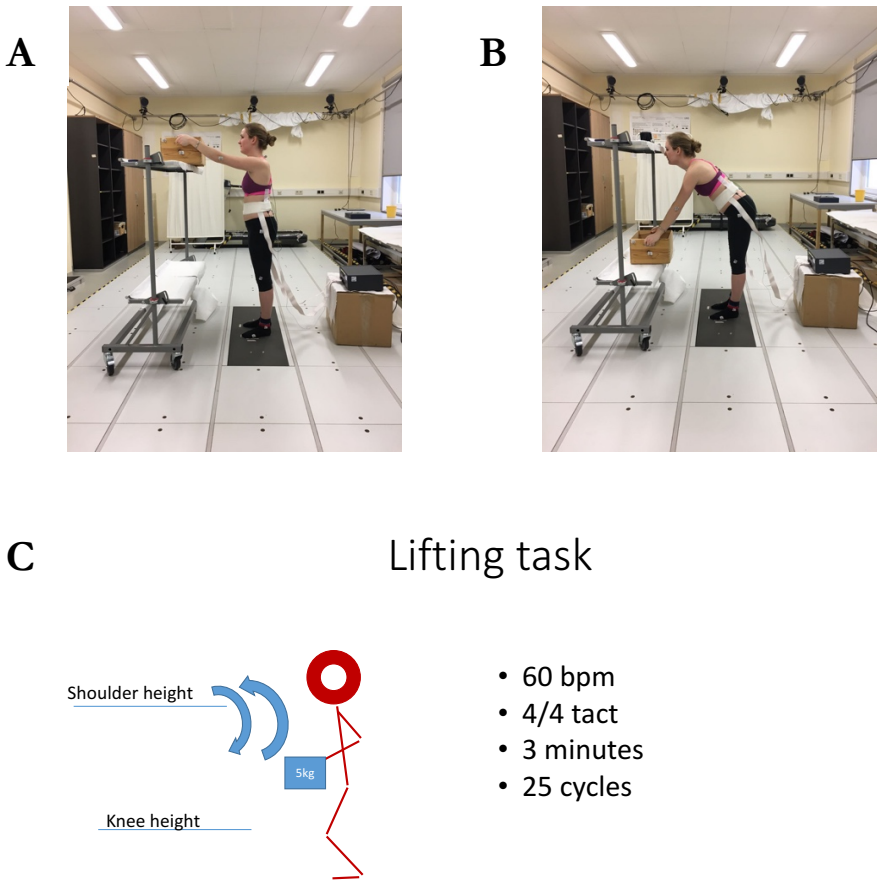

Figure 3: Composition of the lifting task
A: Lifting of the box
B: Lowering of the box
C: Specification of the lifting task

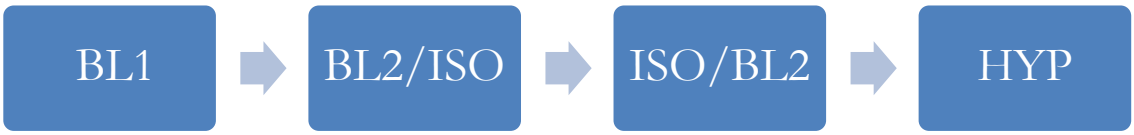

Figure 4: Chronology of conditions. Baseline measure (BL1) was followed randomly by either a repeat baseline (BL2) or injection with isotonic saline (ISO), injection with hypertonic saline (HYP) was the fourth and last run

2.2.3 Experimental muscle pain

In order to find out if the variability of muscular activation can be influenced by acutely evoked pain the participants received two intramuscular injections of 1 ml saline each. One being isotonic with a concentration of 0.9 %, the other one being hypertonic with a concentration of 5.89 %. All subjects were informed, that they were going to receive injections that might be painful, prior to two of the four runs, and that the first run was without injection (Figure 4). To avoid psychological bias, the participants did not know the order of the injection. In fact, isotonic saline was always injected prior to hypertonic saline. This ensured that the (higher) pain typically provoked by hypertonic saline did not have any long-term interference with subsequent runs. The saline was injected into the paravertebral muscles on the right side of the back: Insertion of the 50 mm needle was at the midlevel of the surface electrode (most likely close to the L3 level). The needle was directed laterally and anteriorly and fully inserted to inject the saline below the centre of the surface electrode into the erector spinae muscle. In a subset of patients an ultrasound exam was performed prior to electrode placement to monitor depth of subcutaneous tissue.

Graven-Nielsen et al. investigated the effect of different parameters of an intramuscular injection on the pain perception. Increased volume and concentration led to a higher intensity of pain. Moreover, a fast infusion led to an earlier onset, higher intensity but shorter duration of pain (Graven-Nielsen et al. 1997b). As it was the intention to mimic acute LBP, injection of the saline was done in one slow manual injection over three seconds. Hypertonic saline was used to evoke acute back pain. The objective of the injection of isotonic saline was to control for pain from the needle stick and changes in sensation due to the additional

volume in the muscle. The order of second and third run was randomized to control for anticipatory psychological effects, such as fear of injection.
After each injection, the pain level was immediately recorded. Thereafter the subjects started the lifting task (25 cycles). Right after completing the task PPT was measured. Prior to the next injection they had 20 minutes of rest. During this time, the effect of the saline was expected to fade. In the meantime, the participants had to fill in some questionnaires.

2.2.4 Questionnaires

The participants were asked to fill in two questionnaires (see attachment). Firstly, the general health status was examined with the SF-12 Health Survey (Brazier, Harper et al. 1992). The SF-12 is a short version of the SF-36 Questionnaire, which is a measure for health-related quality of life (Ellert and Kurth 2004). The questionnaire consists of 12 questions which give information about physical and psychological issues. Although the validity of the SF-36 is not reached completely the SF-12 it is much shorter. With regard to our demands the shorter SF-12 is considered an adequate measure to check the general health of the participants.

Secondly, the participants' stress level, and symptoms of depression or anxiety were explored by the DASS (Depression, Anxiety, Stress Scale; Lovibond and Lovibond 1995). This questionnaire is used in clinic to screen pain patients for depression and stress (Nilges and Essau 2015). It consists of three scales (Depression, Anxiety and Stress) with seven items each.

2.3 Recording

2.3.1 Pressure pain threshold

To explore changes in pain sensitivity at the lower back the pressure pain threshold (PPT) was measured using a pressure algometer providing information about the characteristics of deep pain (Rolke et al. 2005). In this study an electronic pressure algometer (Somedic Production, Stockholm, Sweden) was used (Figure 5). The algometer reported the applied pressure on a digital display. The pressure itself was applied by hand and slowly increased with 30 kPa/s. The participants were asked to report when they started to feel the perception

of pressure change to a perception of pain. The respective value for each location was documented. To avoid injuries the maximally applied pressure was limited to 1000 kPa. If a subject did not report any pain until this level 1000 was taken as a threshold value.

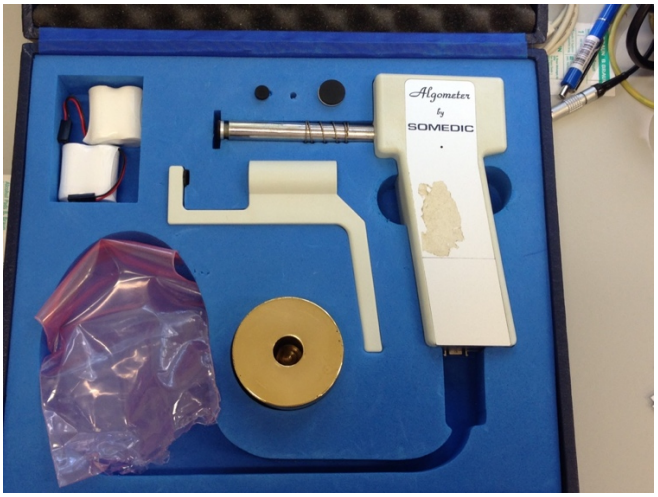

Figure 5: Pressure pain algometer

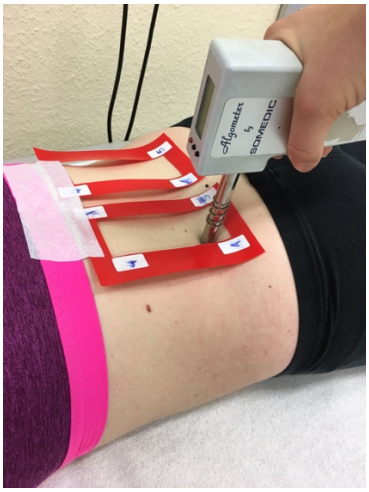

Figure 6: Measuring PPT at the back of a participant

The pressure pain level was examined in 16 points at the lower back.

A template was placed so that the medial lower edge of the inner frame was over the Processus transversus of L5.

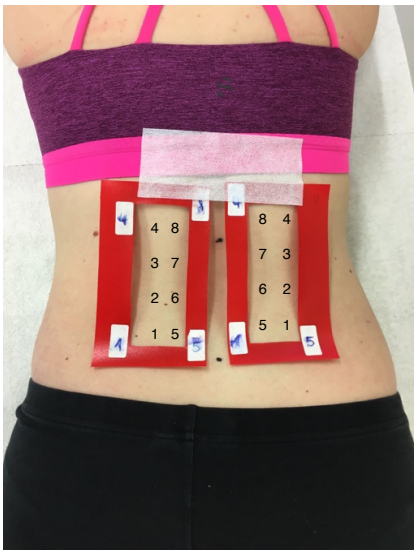

Figure 7: Location of the Pressure Points

The measurement started at the lower edge on the lateral side of the template and repeated four times in cranial direction. The fifth measurement was done on the lower medial edge of the template (Figures 6 and 7).

The distance between the single points was 2.5 cm. This examination was done at the very beginning and repeated after each task and in the very end. All in all, PPT was measured at six time points. The first and the last time it was measured using the template (Figure 7). Between the lifting tasks while the electrodes were stacked on the back, the PPT was measured around them.

2.3.2 EMG recording

The muscle activity was detected using 64 channel high-density surface EMG electrodes (ELSCH064NM2, OT Bioelettronica, Torino, Italy) with 13 x 5 grid, 8 mm of inter-electrode distance and dimension of 120 x 48 mm (Figure 8).

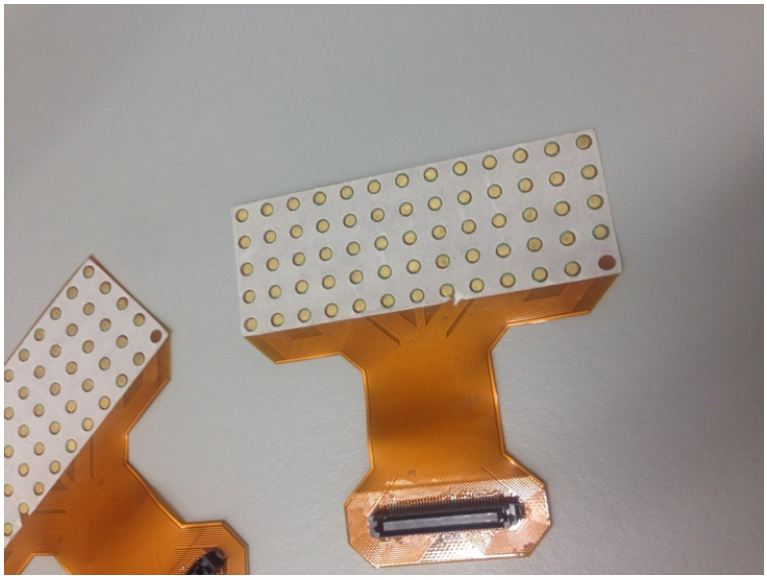

Figure 8: High-density surface electrodes

The skin of the recording region was cleaned using abrasive gel and 70 % ethanol before electrodes were placed. Two high-density EMG electrodes were placed symmetrically bilateral over the bulk of the M. erector spinae/M. multifidus. The medial edges of the electrodes (the side without connector) were 2 cm away from the spinal column and the caudal edge was on the level of L5. The electrodes were covered with conductive gel (filling the cavities of the electrode completely) to ensure good contact to the subjects' skin. Two reference electrodes (Ambu electrode, Spacemedica) were placed on the Procc. spinosi over the upper edge of the electrode (Figure 9).

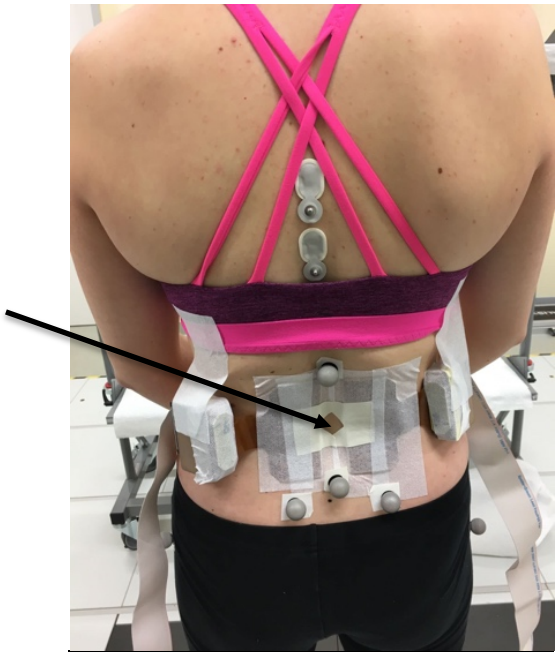

Figure 9: Overview of the participant's back after preparation. The arrow marks the location where the saline injection was performed

The surface EMG was recorded using a 256 channel EMG acquisition system (EMG-USB2, OT Bioelettronica, Torino, Italy) at 2048 sample/s with resolution of 12 bit and gain factor of 1000 or 2000 in monopolar mode. Monopolar mode provides detection of the activity from deeper muscles (Beck et al. 2007). The EMG signal was filtered using analogue filters during recording at 500 Hz low-pass and 20 Hz high-pass cut-off frequencies.

2.3.3 Motion capture system

The lifting task was performed in the sagittal plane (Marras et al. 1995). As the weight of the lifted box has a strong influence on the lumbar-pelvic coordination (increasing the lumbar contribution with increasing weight) (Mitnitski et al. 1998; Granata and Sanford 2000), this study used a standardised weight for all participants to reduce this effect. As the pelvic leg angle is affected while bending knees the lifting task was performed with straight knees. This restricted moving differences between the participants in the spine and pelvic regions (Granata and Sanford 2000).

The movement of subjects was detected by an eight-camera system (Oqus 300+, Qualisys Gothenburg, Sweden) placed in every corner of the room and in the middle of each side

(Figure 10). These cameras radiated infrared rays which were reflected by the markers and received by the digital cameras. Overall, 16 markers were attached to anatomic landmarks of the patient (Table 1). At least three cameras had to track one marker. The information of the markers' location was transmitted to a computer. A software (Track manager, Qualisys AB, Gothenburg, Sweden) converted this information into a 3-dimensional rendering of the participant.

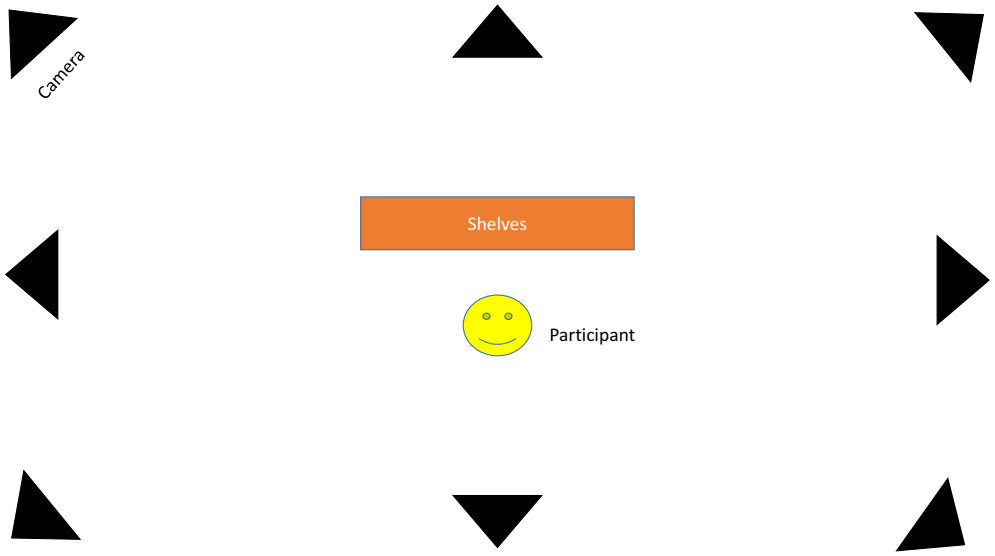

Figure 10: Positioning of the camera system

Additional eight markers were placed at the box and the two shelves. Two markers were placed on the edge of the lower shelf and two on the upper one. From this the height of the two shelves could be recorded. Four markers were placed on the box to show and monitor the position of the lifted object.

The motion capture was used to track the position of the markers and their speed. The markers were placed on joints that played an important role during the task. For example, the angles between C7 and L5 (trunk bending) and compensating movements in the hip were analysed. Moreover, the rhythm of the lifting task could be checked i.e., whether the lifting was consistent or the rhythm changed under perceived pain.

Table 1: Location of the reflecting markers (ri = right side, li=left side)

C7
Acromion ri
Acromion le
Epicondylus humeri ri
Epicondylus humeri le **L1 (upper edge of EMG)** **L5 (lower edge of EMG)** **triangle under L5 (1 finger below L5, 2 fingers laterally)**
Trochanter maj. of femur ri
Trochanter maj. of femur le
lat. Condylus fem. ri
lat. Condylus fem. le **lat. Malleolus ri**
lat. Malleolus le
5 th Metatarsus ri
5 th Metatarsus le

2.4 Signal analysis

2.4.1 NRS

For analysing the pain intensity, the mean value and standard deviation across all participants were calculated. From these data the development of pain sensation over the whole trial and differences between the different conditions were investigated.

2.4.2 PPT

For analysing the PPT, the mean over all participants was calculated for each point and each condition, so PPT was taken from the beginning, after each task and at the very end of the whole session. Thereof changes of the pressure pain threshold between conditions were

estimated. The mean of all points for every single condition was calculated. Moreover, the mean for left and right was compared in every condition. In addition to this, the mean of the single rows and columns was compared in every condition.

2.4.3 EMG

The EMG signals from 128 channels were stored in a computer for an offline analysis. The signals were pre-processed and analysed using custom programs written in MATLAB programming software. The 4th order Butterworth band pass filter was used to filter the EMG signal with a band pass of 20Hz and 500Hz. If single channels had an insufficient contact to the skin, they were substituted by the average of neighbouring channels.

To be able to get information about the standard amplitude the signals were rectified. Otherwise the sum would be zero (Konrad 2011). From this full wave rectification standard amplitude parameters were applied to the curve (Konrad 2011). As the EMG activity is of random nature digital smoothing algorithms were applied. This makes it possible to eliminate non-reproducible peaks in EMG signal (Konrad 2011). In the present study root mean square (RMS) was calculated for each monopolar recording. It gives information about the mean power of the signal and is the preferred recommendation for smoothing (Luca 2002; Konrad 2011). To calculate RMS the squares of all amplitude values were added and divided through the number of values. Subsequently the root was extracted.

$$\sqrt{\frac{1}{n} * (x1^2 + x2^2 + \cdots xn^2)}$$

For analysing the frequency, the mean frequency (MNF) and the frequency spectrum were calculated. By using Fast Fourier Transformation, the raw EMG was assumed to be an overlap of many sinusoid curves of different frequencies. Subsequently, the single frequencies were assigned to special Hertz ranges. This showed the distribution of frequencies and made is possible to graph the Total Power Spectrum (Konrad 2011). "The MNF showed the mathematical mean of the spectrum curve" (Konrad 2011).

MNF and RMS were calculated for each monopolar recording. The average RMS was calculated over all 64 monopolar electrodes to get information about the overall activity. The NormRMS values were graphed with regard to the repetitions of lifting and lowering of each side in every single condition. The activity pattern was calculated from the weighted centre of mass from the RMS map (centroid). The shift of the centroid of activity was mapped in X- and Y- axis.

2.4.4 Motion capture

The motion capture data were analysed with the corresponding software Qualisys track manager. The Software estimates the precise location of the reflecting markers in a coordination system with X-, Y-, and Z-axis. A change of their location was measured between the markers and with regard to the spatial axes. Every marker was defined with a special position on the body (e.g., C7, L5). From this information the exact movement of the body was calculated as well as the position of the box (Figure 11).
The cameras recorded 64 frames per second to capture the movement. The signal was filtered with a time window of 0.5s. The data was exported to MATLAB.
The frames were geared to the cycles of lifting and lowering. The spine angle in the ZY and ZX plane for C7 and L5 was analysed for each frame. Moreover, the angular velocity and the variability of the repetitions was analysed and compared between the different conditions.

The Z-coordinates were used to analyse the movement and speed of the four markers on the box along the Z-axis. The on and off-set of each cycle and the single sub-phases were analysed. To be able to better compare subjects a metronome was used to provide a standardised rhythm.

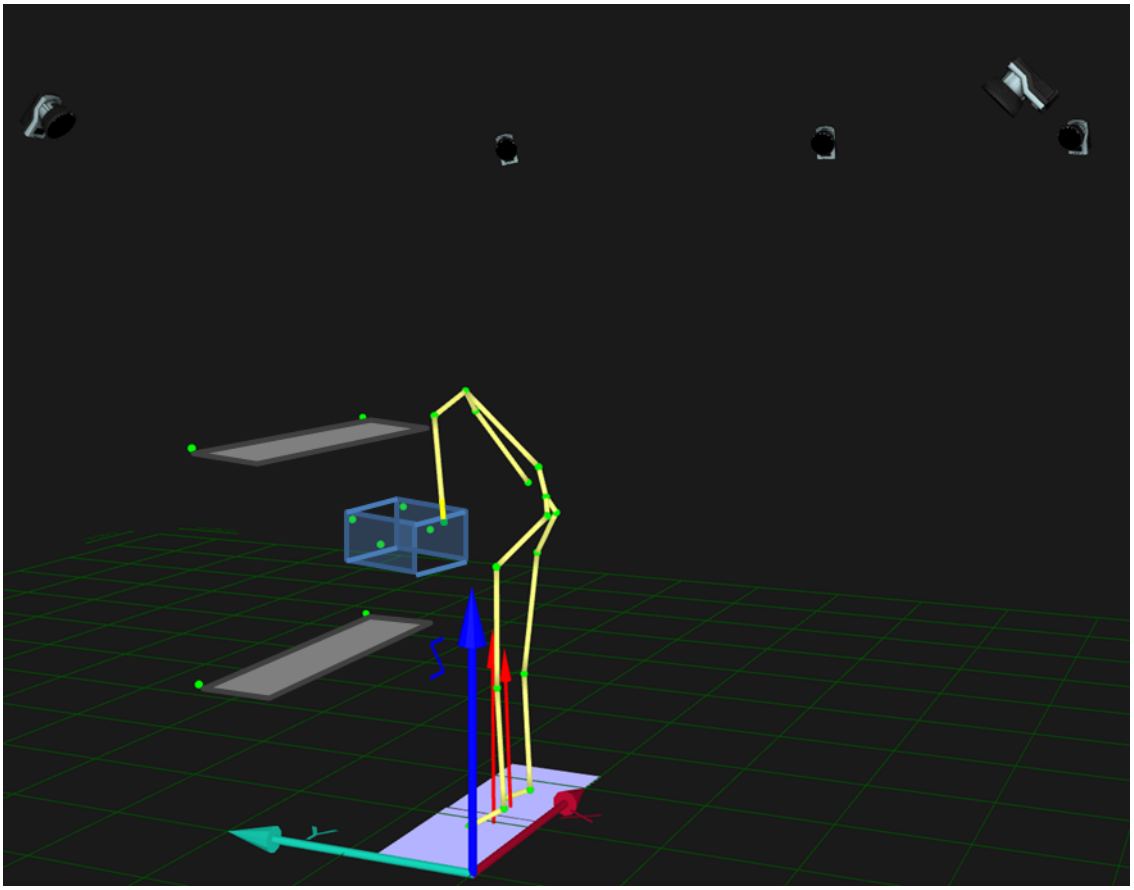

Figure 11: Example of the 3D model created from the coordinates of the reflecting markers

Additionally, the data of Motion Capture and EMG were combined. The onset of EMG and the resulting onset of motion were compared to calculate the average electromechanical delay (EMD in ms). The average delay was compared between the different conditions.

2.5 Statistical analysis

Mean, standard deviation and standard error were calculated with Excel (Microsoft Excel for MAC, Version 15.27). The statistical analysis was performed using SPSS. All data were approximately normally distributed. For better comparison between conditions we used ANOVA for each calculation. For the demographic statistics, the mean and standard error of the age were calculated for both male and female participants. Moreover, the mean and standard error over all participants were calculated. There was the intention to have an equal distribution of age between male and female participants. In addition to this, mean and

standard error were calculated from the weight, height, and finger-floor distance over all participants as well as for male and female participants separately. For the NRS, the mean value and standard error of all participants were calculated for each time of the trial. The DASS values were also calculated in Excel taking the mean value of the scores of all participants for depression, anxiety, and stress. For the SF-12 the physical and psychological summary scores were calculated. The statistical analysis was performed using SPSS with the appropriate template. Then the mean value for each score was calculated.

The statistical analysis of the PPT was generated with SPSS. A general linear model with repeated measurement ANOVA was used. The interrelation between the factors condition (baselines, isotonic and hypertonic), side (LL, ML, MR, LR) and the recording altitude (P1-P4) was investigated. The level of significance was defined as $p < 0.05$ (5 %). All factors were analysed within the subject. Multivariate tests were applied for interactions, then marginal means were calculated for each factor. Bonferroni was used as a post hoc test.

The signals from EMG and Motion Capture were processed and analysed with a programme written in MATLAB R2015b numerical programming software (The MathWorks, Inc.) following the details above. The resulting values of the EMG were also analysed in SPSS using a general linear model in RM-ANOVA. The factors condition, side (left vs right), task (lifting vs lowering) and time (19-time windows throughout each task) were analysed. Therefore, multivariate tests were made to check rough correlations. The single factors were investigated using marginal means with pairwise comparisons. The centroid was also analysed in a general linear model in RM-ANOVA. There the factors were task (lifting/lowering), side (right/left), condition and time.

For Motion Capture data, the movements were divided into lifting and lowering (task). The mean angles (AvgAng) of each condition (BL1, BL2, ISO, HYP) were compared using RM-ANOVA. In order to investigate the regularity of the movements, the coefficient of variations (CoV) was used. It is defined as the ratio of the standard deviation of the AvgAng to the mean. The results were analysed using RM-ANOVA, as mentioned above.

The EMD was also analysed in SPSS using general linear model. A single factor ANOVA was used. The factor was condition.

3 Results

3.1 Subjects

Twenty-six participants (13 male and 13 female) took part in the study. They were aged between 20 and 54 years. The mean age was 34 ± 2 years for the male and 34 ± 2 years for the female participants. Table 2 shows the physical characteristics of the subjects, Figure 12 the age distribution.

Table 2: Physical characteristics of the participants with mean and standard error.

	Mean ± SE
Age	34 ± 2
Gender (% male)	50
Age male	35 ± 3
Age female	35 ± 3
Height (cm)	176 ± 1
Height male (cm)	182 ± 2
Height female (cm)	170 ± 1
Weight (kg)	721± 3
Weight male (kg)	82 ± 2
Weight female (kg)	61 ± 4
Finger-floor-distance (cm)	7 ± 2
Finger-floor-distance male (cm)	9 ± 3
Finger-floor-distance female (cm)	5 ± 2
Schober (cm)	15 ± 0
Schober male (cm)	15 ± 0
Schober female (cm)	14 ± 0
Ott (cm)	32 ± 0
Ott male (cm)	32 ± 0
Ott female (cm)	33 ± 0

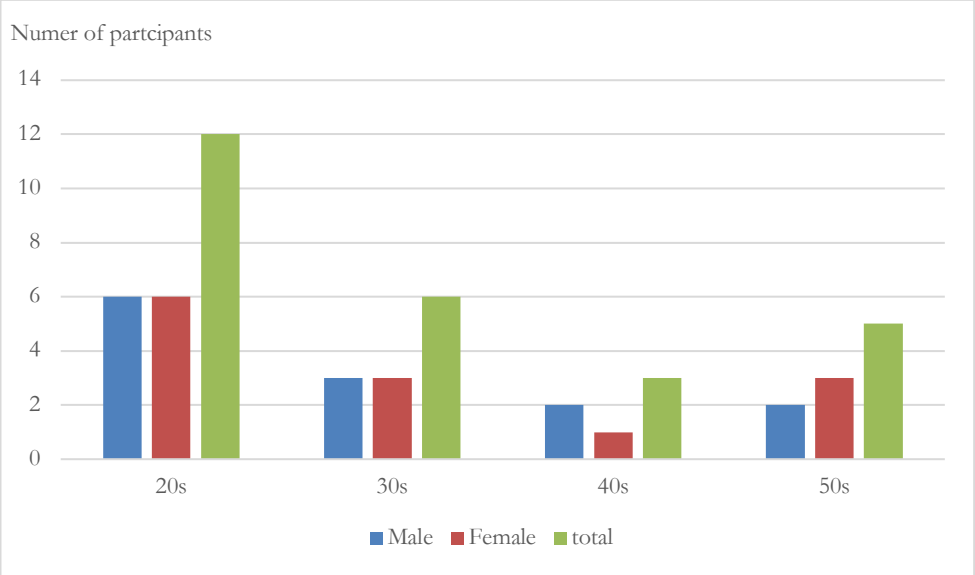

Figure 12: Distribution of the participants' age groups in the total cohort, for male and female.

The two questionnaires (DASS and SF-12) were analysed with standardised templates. As the preliminary screening required participants to not have any physical or psychological impairments the results were inconspicuous. The results of the questionnaires are shown in Table 3. There were no relevant differences between female and male participants.

Table 3: Evaluation of the questionnaires (DASS and SF-12).

Questionnaire	item	mean	SE
DASS	Depression	2	0
	Anxiety	1	0
	Stress	4	1
SF-12	Physical score	55	1
	Psychological score	53	1

3.2 Pain level

At the very beginning of the task no participant reported pain on the NRS (BL1). During the first three minutes of the lifting task the pain level reached a maximal mean value of 1 ± 0.2 after 20 cycles. The other run without injection (BL2) showed similar results (maximal mean value 1 ± 0.2).

Right after the injection of isotonic saline (ISO) the participants reported a pain level of 1 ± 0.3. This level decreased over the three minutes of the lifting task. In contrast, pain right after the injection of hypertonic saline was reported with 4 ± 0.4 and stayed quite stable during the first 15 cycles (HYP). At the end of the whole experiment the participants reported a pain level of 1 ± 0.2. The pain level and its development are summarised in Table 4 and in Figure 13 and 14. Figure 14 shows a time course of the development of pain intensity over the whole session. Moreover, the development of the pain intensity had a different shape for isotonic and hypertonic saline. Whereas isotonic saline gave its pain maximum at the time of injection, hypertonic saline raised in the pain level and then slowly faded .

There was a significant higher pain level after the hypertonic saline injection compared to all other conditions ($p < 0.05$) for all time points. There was no significant difference between isotonic saline injection and the two baseline runs at any time point.

Table 4: Pain perception for each trial (mean ± standard error).

	Beginning	5 Cycles	10 Cycles	15 Cycles	20 Cycles	25 Cycles
BL1	0 ± 0	0 ± 0	0 ± 0	0 ± 0,1	1± 0.2	0 ± 0.1
BL2	0 ± 0	0 ± 0.1	0 ± 0.2	1 ± 0.2	1 ± 0.2	0 ± 0.2
ISO	1 ± 0.3	1 ± 0.3	1 ± 0.2	1 ± 0.2	0 ± 0.1	0 ± 0.1
HYP	4 ± 0.4	4 ± 0.4	4 ± 0.4	4 ± 0.4	3 ± 0.4	2 ± 0.3

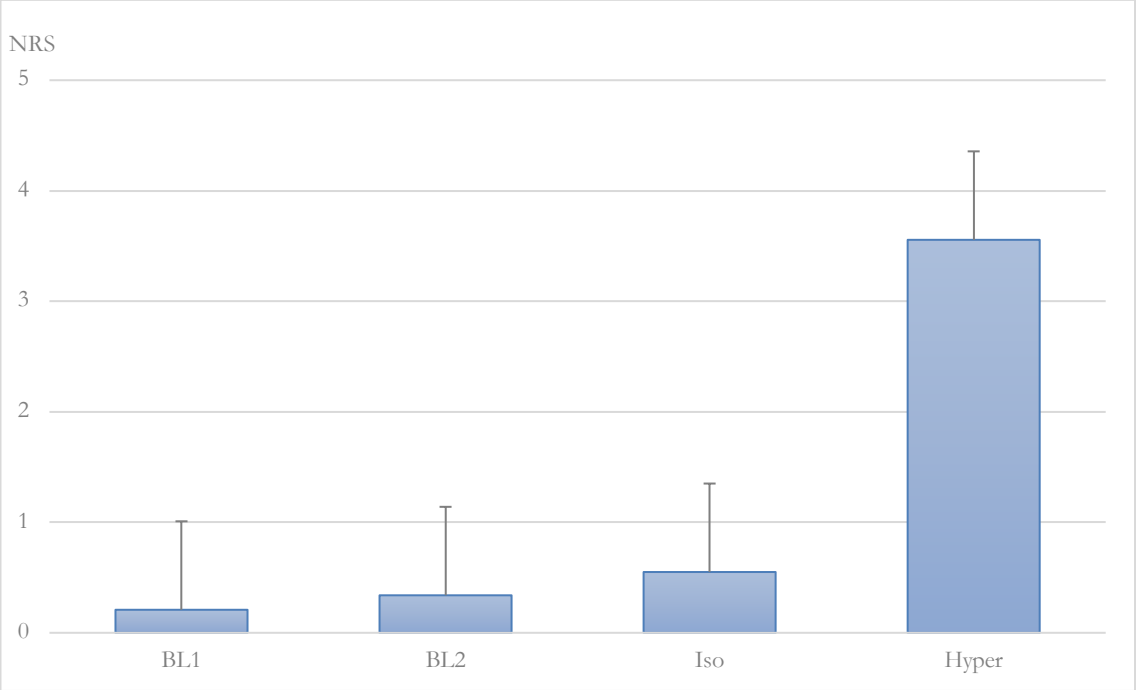

Figure 13: Average pain intensity for every condition on a numeric rating scale. The bar chart presents the mean values with the standard errors.

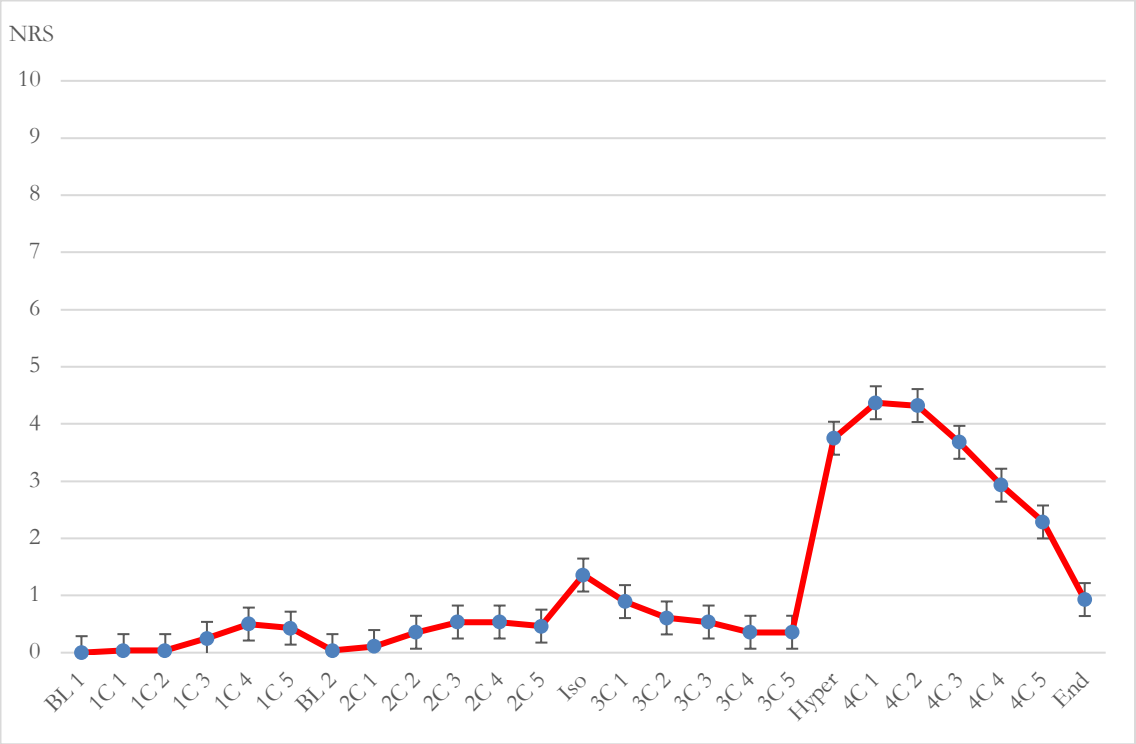

Figure 14: Development of pain perception of the whole session (mean with SE). BL2 and ISO were in reverse order for half of the subjects. Since there were no significant differences between BL2/ISO versus ISO/BL2, the same sequence is shown for all subjects

3.3 Pressure pain threshold

The PPT was measured on 16 (2 x 8) different points on the lower back on each side of the spinal column in six different conditions: beginning (beginning), 1st task (BL1), without injection (BL2), after isotonic injection (ISO), after hypertonic injection (HYP), after removing the electrodes (end). For analysis a general linear model was used in RM-ANOVA.

Regarding the multi-variate tests, both condition ($F=17.636$; $p < 0.05$), side ($F = 32.79$; $p < 0.05$) and altitude ($F = 2.687$; $p = 0.07$) had a significant effect on PPT. There was no condition-side interaction ($F = 1.487$; $p = 0.256$) but a condition-altitude interaction ($F = 15$; $p < 0.05$) and a side-altitude interaction ($F = 11.943$; $p < 0.05$).

Regarding the conditions, it becomes obvious that the PPT was significantly lowest at the beginning ($F = 17.636$; $p < 0.005$). After the injection of hypertonic saline, the PPT was significantly higher than all other conditions ($p < 0.05$) except for BL2.

Within any condition there was no significant difference between injected and non-injected side, even for the hypertonic saline condition. Regarding the mean of all points for each condition, the values after removing the electrodes (End) were significantly lower than BL2 ($p = 0.01$) and HYP ($p = 0.007$). There was no significant difference between the other conditions. The mean values are summarised in Table 5.

Table 5: PPT (kPa) for each condition.

	Mean of both sides ± SE	Mean left side ± SE	Mean right side ± SE
Beginning	536 ± 9	541 ± 10	532 ± 8
BL1	649 ± 12	657 ± 11	641 ± 13
BL 2	682 ± 12	687 ± 10	677 ± 13
ISO	664 ± 12	678 ± 11	650 ± 12
HYP	695 ± 13	698 ± 11	691 ± 15
End	614 ± 14	629 ± 13	600 ± 15

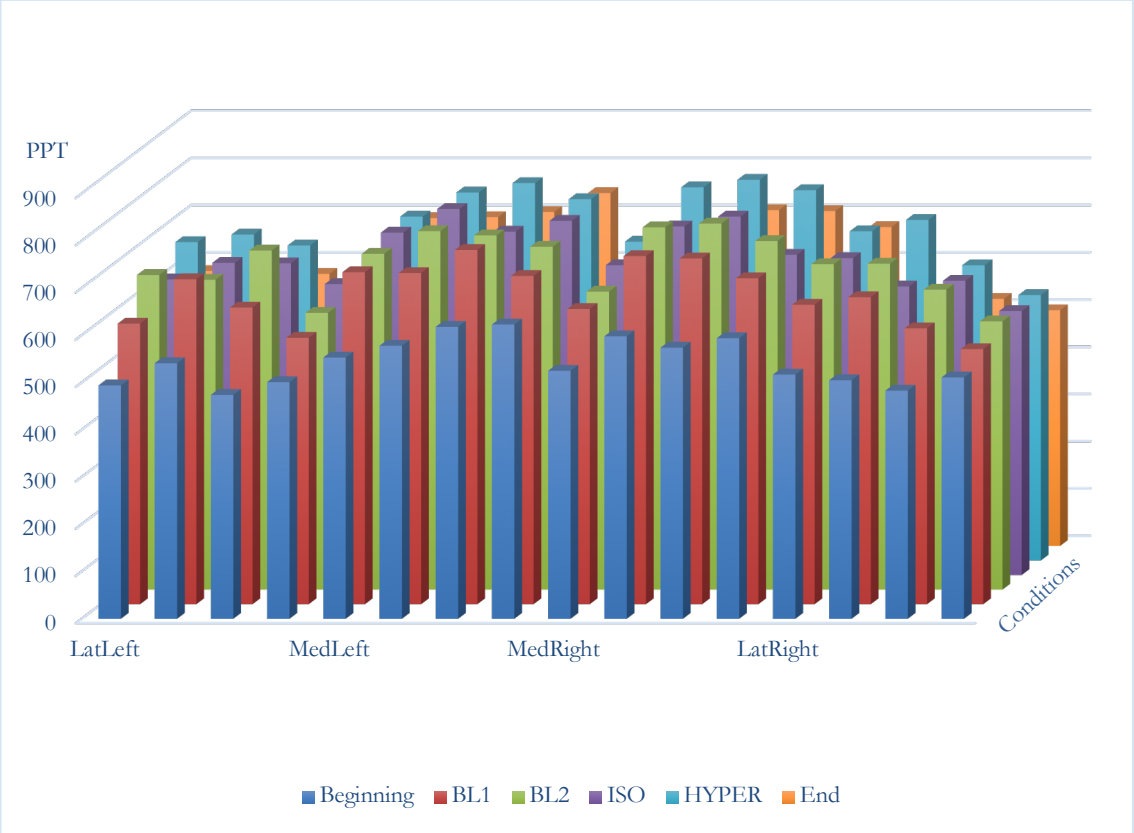

Figure 15: Mean PPT (kPa) data from all measurement points (LatLeft to Lat Right) over all conditions

The distribution of PPT was analysed through conditions and measurement points using a Two-Way Repeated Measures ANOVA. Figure 15 shows mean data from all measurement points and conditions, Figure 18 the anatomical positions of the points. The multivariate tests showed that both factors, condition and measurement points had a significant effect on the PPT. However, there was no significant difference between the injected (right) side and the non-injected (left) side over the whole session (Figure 16).

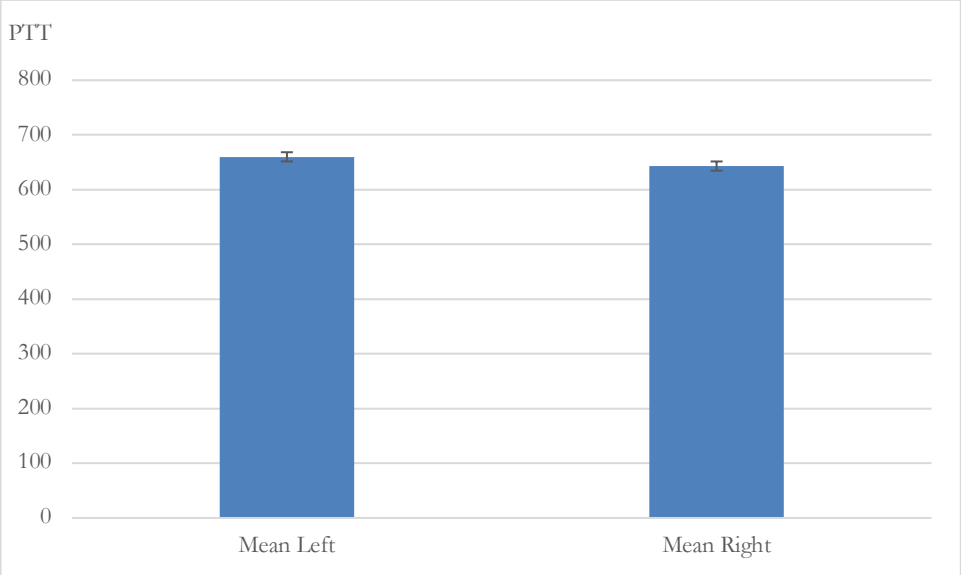

Figure 16: Comparison of the PPT (kPa, mean ± SE) : left side vs right side

The differences between the single conditions were calculated. Therefor the points were grouped in columns and rows.

Firstly, the values of medial and lateral columns were compared. There was a significant difference between the lateral and the medial rows. In the LL Row the PPT was significantly lower than in the ML und MR row ($p < 0.05$), whereas there was no difference between LR and LL. However, the ML had a significantly higher PPT than the MR row.

Regarding the graphs, it becomes obvious that the PPT behaved laterally reversed. The medial points overall had a higher threshold than the lateral points. This effect was significant for every condition ($p<0.05$). (Figure 17).

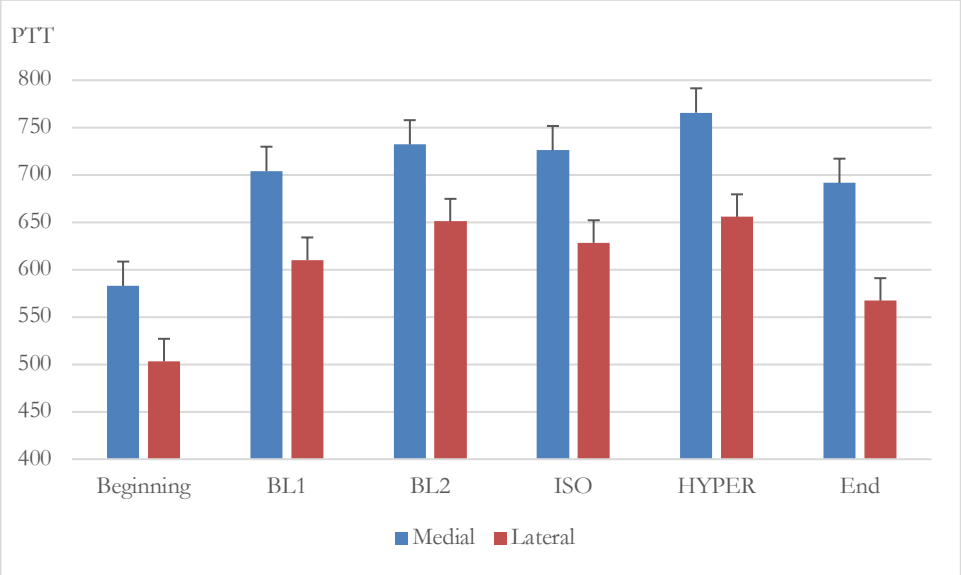

Figure 17: PPT (kPa, mean ± SE) from medial rows compared with lateral rows for each condition

Secondly, the rows were analysed (Figure 18-22). Therefor pairwise comparisons were used. In every row the PPT was significantly lowest at the very beginning ($p < 0.05$).
In the most caudal row P1 and in the most cranial row P4 the PPT after hypertonic saline injection was significantly higher than PPT after BL1 and End. There were no differences between HYP, BL2 and ISO.
In the rows P2 and P3, which were closer to the injection point the PPT after HYP was significantly higher compared to BL1, ISO and End. There was no significant difference between HYP and BL2.

As the injection was performed between P2 and P3 on the right side it was of major interest to see if there was a significant difference between the left and the right medial point of row 3 and 2. The t-test showed that there was no significant difference between injected and non-injected side ($p = 0.767$ for P2 and $p = 0.818$ for P3).

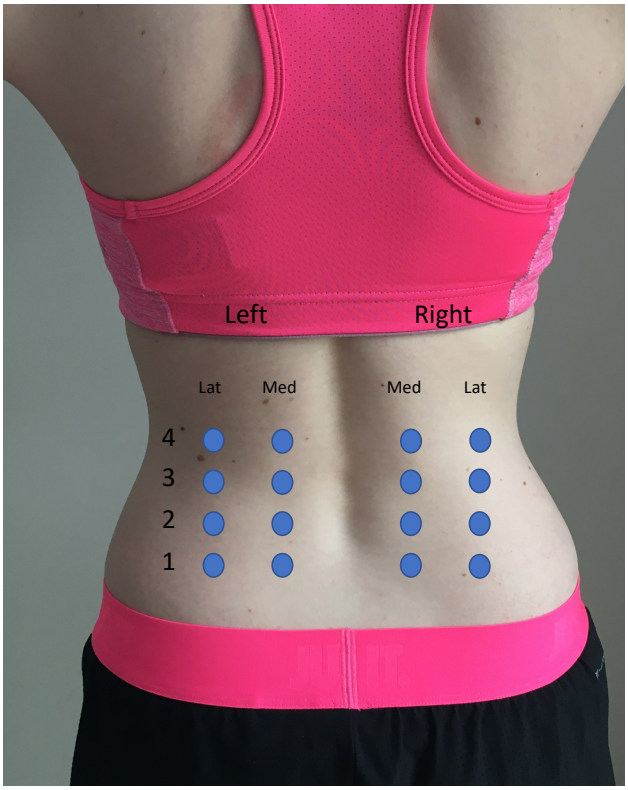

Figure 18: Overview of the anatomical location of the pressure points

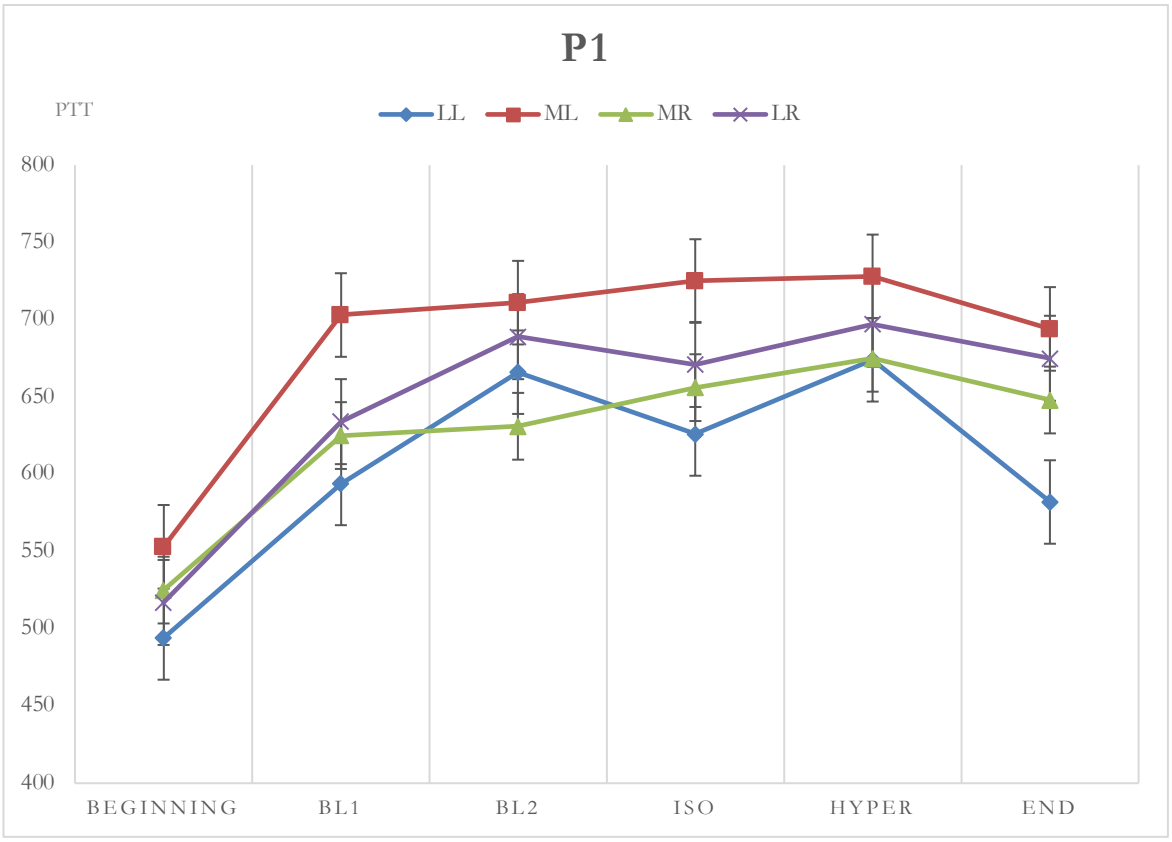

Figure 19: PPT (kPa, mean ± SE) for P1 for each condition

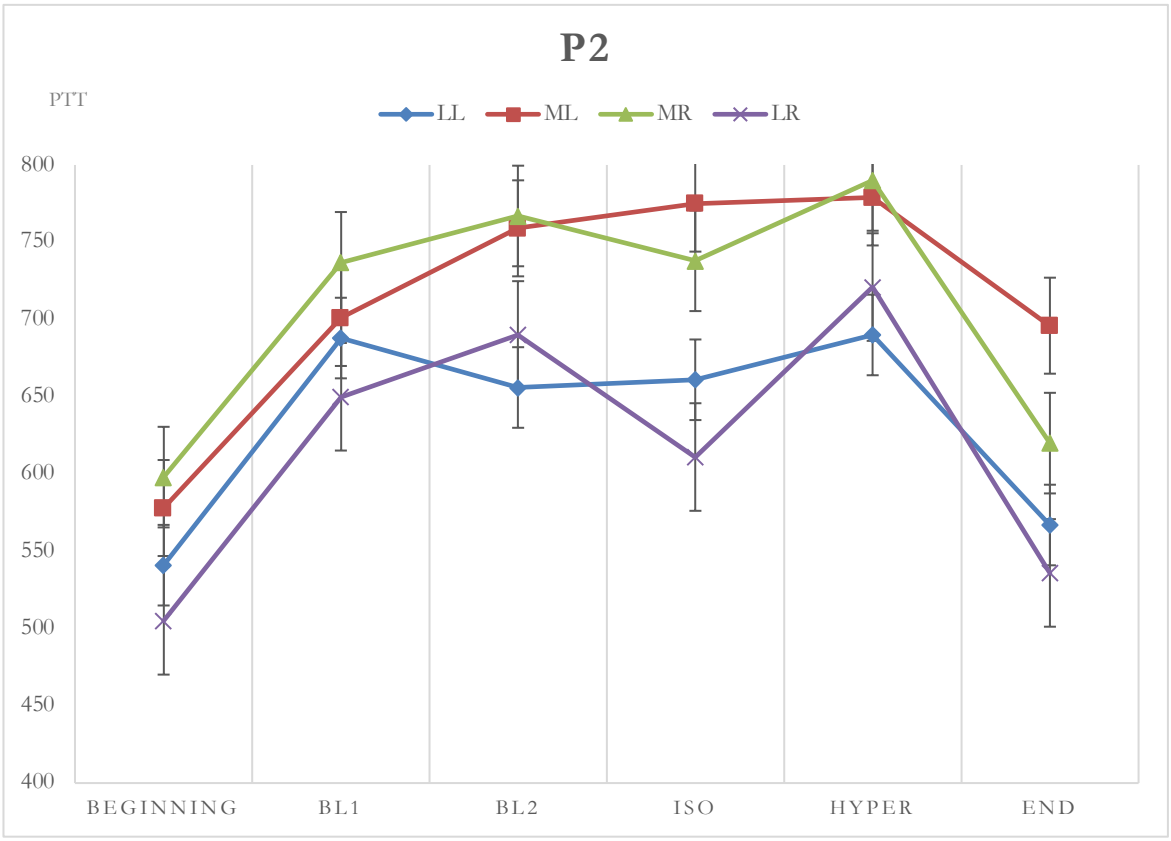

Figure 20: PPT (kPa, mean ± SE) for P2 for each condition

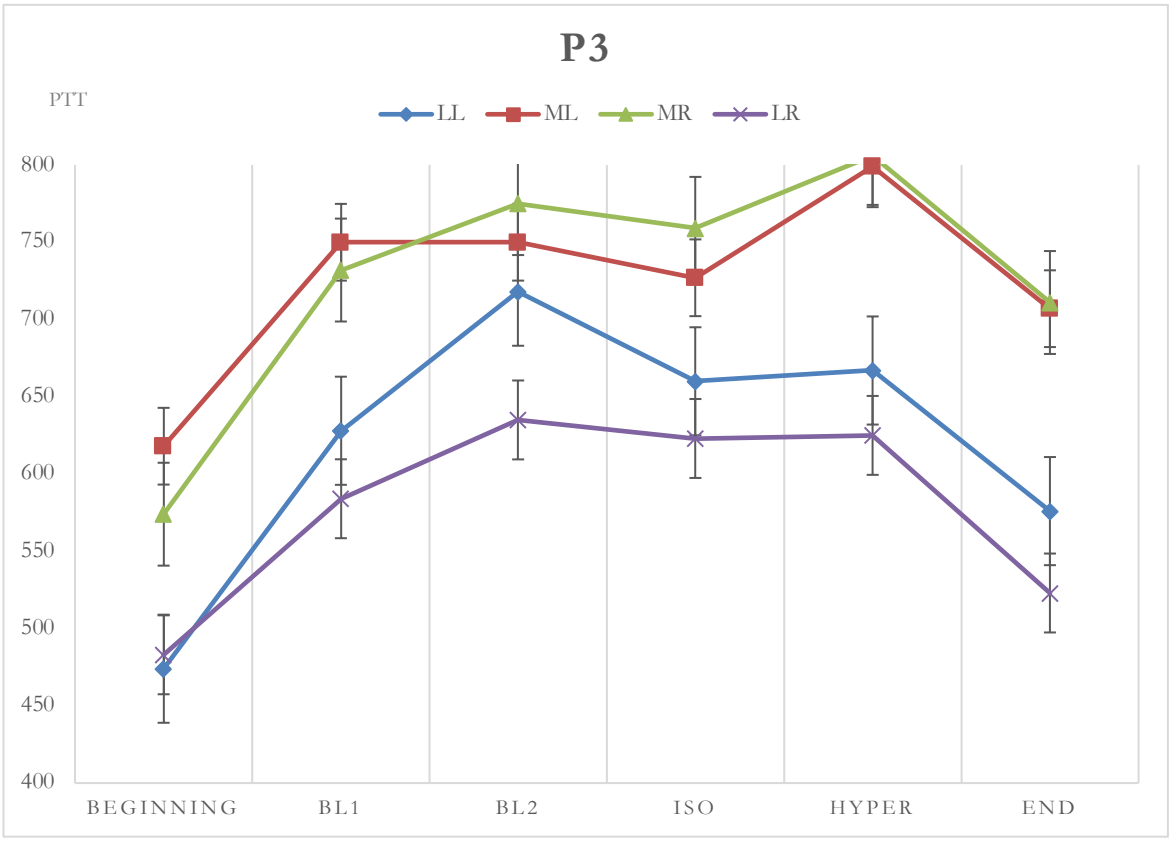

Figure 21: PPT (kPa, mean ± SE) for P3 for each condition

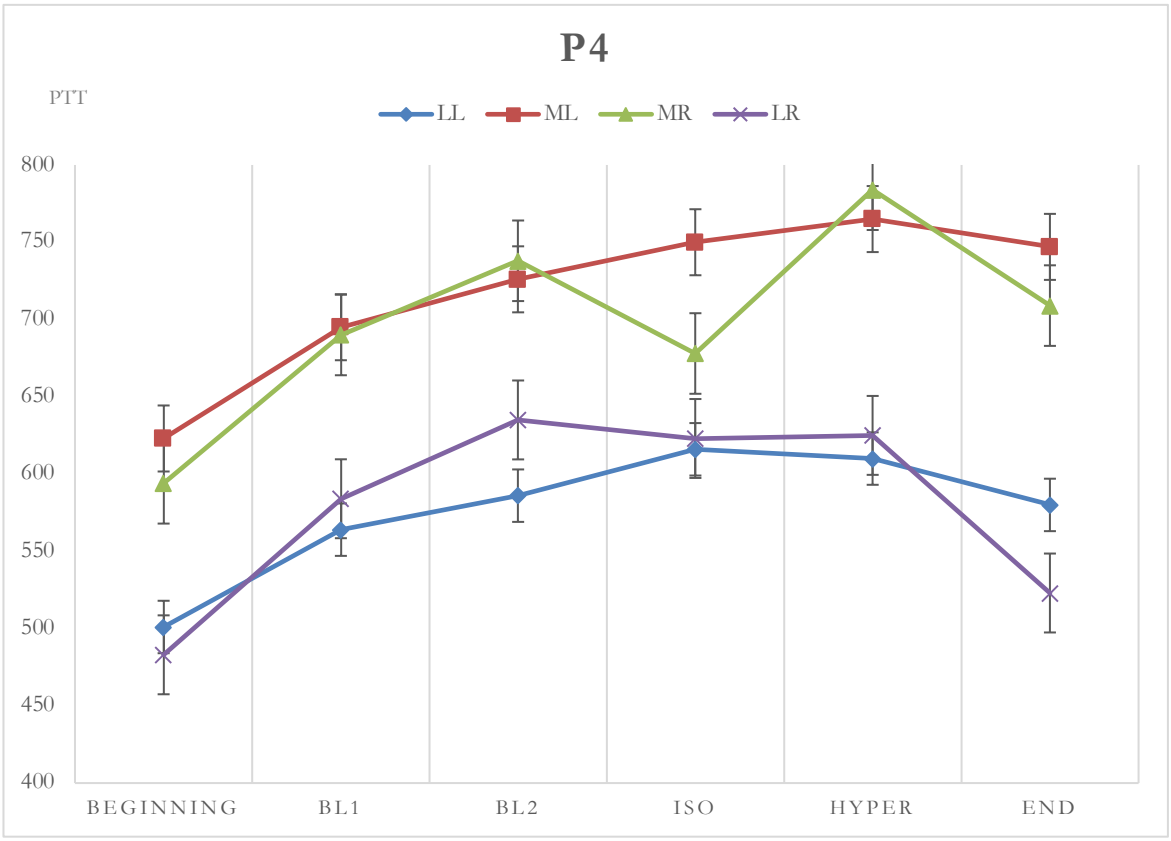

Figure 22: PPT (kPa, mean ± SE) for P4 for each condition

3.4 EMG

3.4.1 RMS

The EMG data were separated in a lifting and a lowering period (task) and the RMS for each condition, side and task was calculated (Table 6 and 7). For further analysis, a general linear model was used.

Table 6: RMS (no unit since normalised to baseline) values for lifting period

Condition	Mean	SE	Condition	Mean	SE
BL1 Left	331.5	± 22.4	BL1 Right	333.5	± 22.4
BL2 Left	313.1	± 25.4	BL2 Right	315.7	± 25.7
ISO Left	311.2	± 24.7	ISO Right	311.5	± 23.6
HYP Left	303.4	± 26.7	HYP Right	292.5	± 26

Table 7: RMS (no unit since normalised to baseline) values for lowering period

Condition	Mean	SE	Condition	Mean	SE
BL1 Left	274.9	± 17.1	BL1 Right	280.1	± 20.4
BL2 Left	256.1	± 19.4	BL2 Right	261.3	± 22.8
ISO Left	256.8	± 18.3	ISO Right	262.2	± 22.4
HYP Left	257.4	± 25.3	HYP Right	245.4	± 21.7

First multivariate tests were applied. The task (lifting/lowering) had a significant effect on RMS ($F = 37.654$; $p < 0.05$) with higher values recorded during lifting. Moreover, a significant overall effect of condition was observed irrespective of the task ($F = 5.369$; $p = 0.03$).

In contrast to this the side (left/right) did not show a significant effect ($F = 0$; $p = 0.99$).

Importantly, there was also neither a significant task-side interaction ($F = 0.128$; $p = 0.725$) nor a task-condition interaction ($F = 0.823$; $p = 0.49$). However, there was a side- condition

interaction ($F = 14.717$; $p < 0.05$). A task-side-condition interaction was not observed ($F= 0.392$; $p = 0.759$).

The investigated factors "Condition" (BL, HYP etc.) and "Task" (lifting and lowering) both had a significant effect on the RMS variation. The three minutes of lifting and lowering include 19 repetitions each (Figure 23).

Table 8: Marginal means from RM-ANOVA for the task periods

Task	Mean	SE
Lifting	314	20
Lowering	261.8	17.8

Regarding the marginal means it becomes obvious that the RMS was significantly higher ($p<0.05$) during the lifting task compared with lowering (Table 8).

Table 9: Mean RMS (no unit since normalised to baseline) on the right side

Condition	Mean	SE
BL1	310.7	20
BL2	294.5	22
ISO	293.9	20
HYP	276	22

Table 10: Mean RMS (no unit since normalised to baseline) on the left side

Condition	Mean	SE
BL1	309.8	22
BL2	292.9	23
ISO	291.9	22
HYP	284.5	24

Regarding the conditions for the right side (Table 9) both condition ($p < 0.05$; $F = 8.99$), and time ($p < 0.05$; $F = 2.71$) had a significant effect. During HYP the RMS was significantly lower compared to the other conditions ($p < 0.05$). There was no significant difference between the other three conditions.

The significance varied over time. So, there was no overall effect.

On the left side (Table 10) there was no significant difference between the four conditions.

Regarding the lowering phase there were no significant differences between all conditions and both sides.

In summary, the EMG amplitude was significantly lowest during lifting on the injected side ($p < 0.05$; $F = 8.99$) (Table 9, Figure 23; right lifting; purple graph).

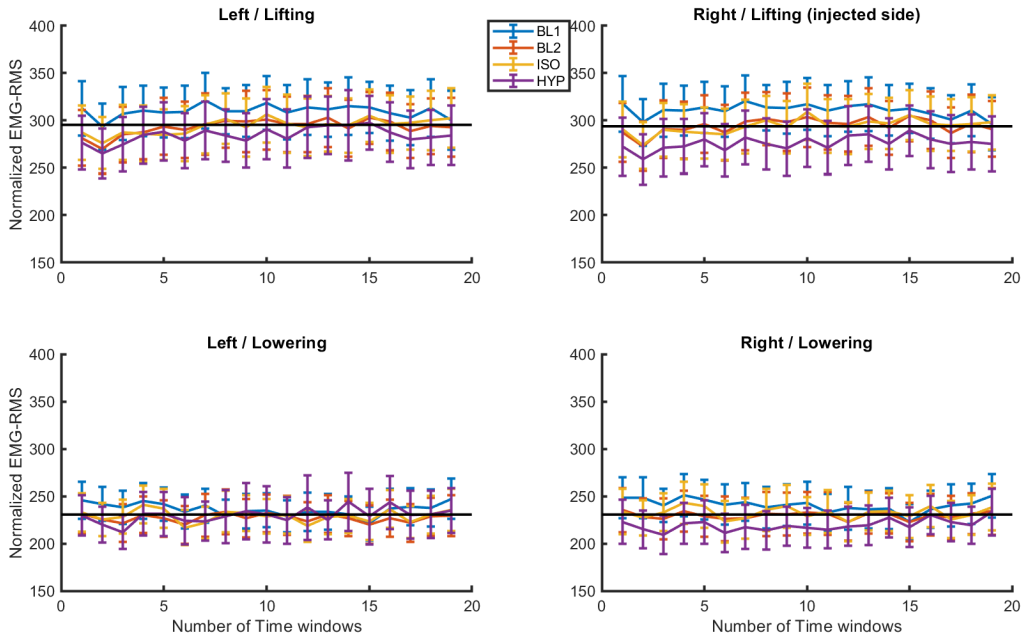

Figure 23: Assessment of EMG-RMS variation through 19 repetitions normalised to the baseline (ground activity during quiet standing). The black line represents the overall mean. Time windows here represent consecutive cycles over the repetitive task

Moreover, the time course of the EMG amplitudes over the lifting and lowering periods was examined (Figure 24). Therefor they were divided into eight time windows each. As expected, there was a significant reduction of the EMG amplitude in HYP during lifting compared to

the other three conditions on the injected side, but no different time courses between the conditions, task periods and sides.

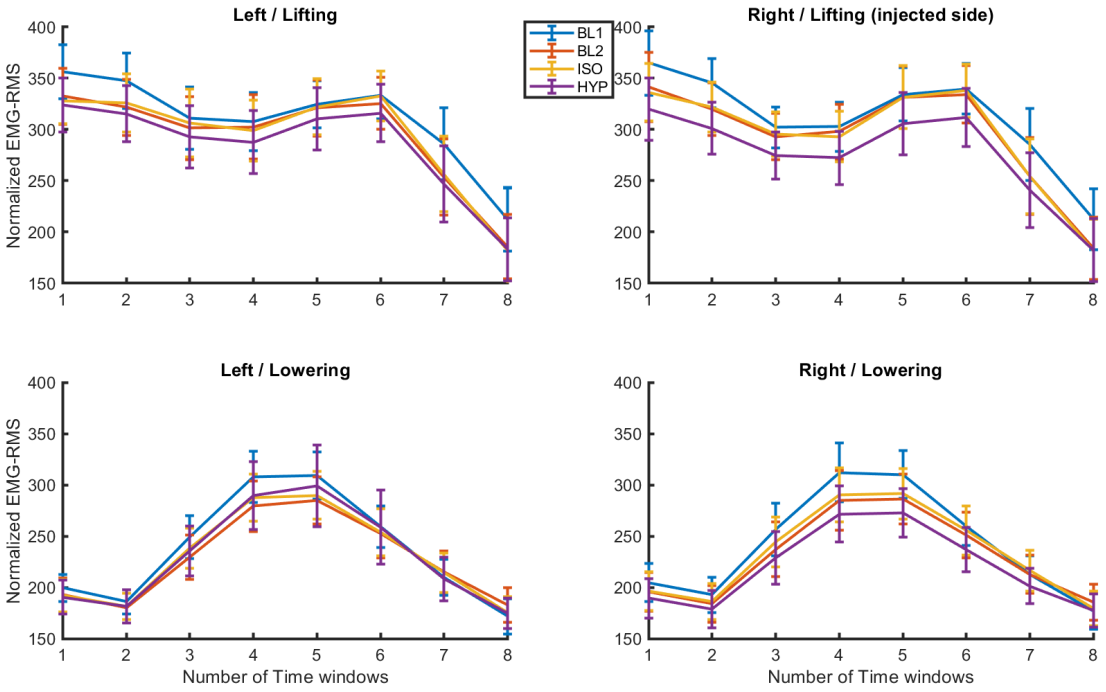

Figure 24: Assessment of EMG-RMS variation divided in lifting and lowering task. Each task was divided into eight time windows. The time windows represent different time segments during the respective phases averaged across all repetitions

3.4.2 MNF

Moreover, the mean frequency (MNF) was analysed for each task, condition, and side. The investigation of MNF showed a significant difference between lifting and lowering, however no significant other effects for neither condition nor side and interactions.

Table 11: MNF (Hz) estimates for lifting and lowering

Task	Mean	SE
Lifting	81.5	3.4
Lowering	90.4	3.4

It turned out that the MNF was significantly higher during lowering ($p < 0.05$; $F = 15.566$) (Table 11). Regarding the side there was no significant difference between left and right($p = 0.308$; $F = 1.103$) (Table 12).

Table 12: MNF (Hz) estimates of left and right

Side	Mean	SE
Right	86.294	3.324
Left	85.634	3.128

There were no significant differences for any condition ($p = 0.096$; $F = 2.535$) (Table 13).

Table 13: MNF (Hz) estimates for conditions

Condition	Mean	SE
Baseline 1	87.966	3.45
Baseline 2	84.248	3.139
Iso	97.168	4.006
Hyper	84.473	2.551

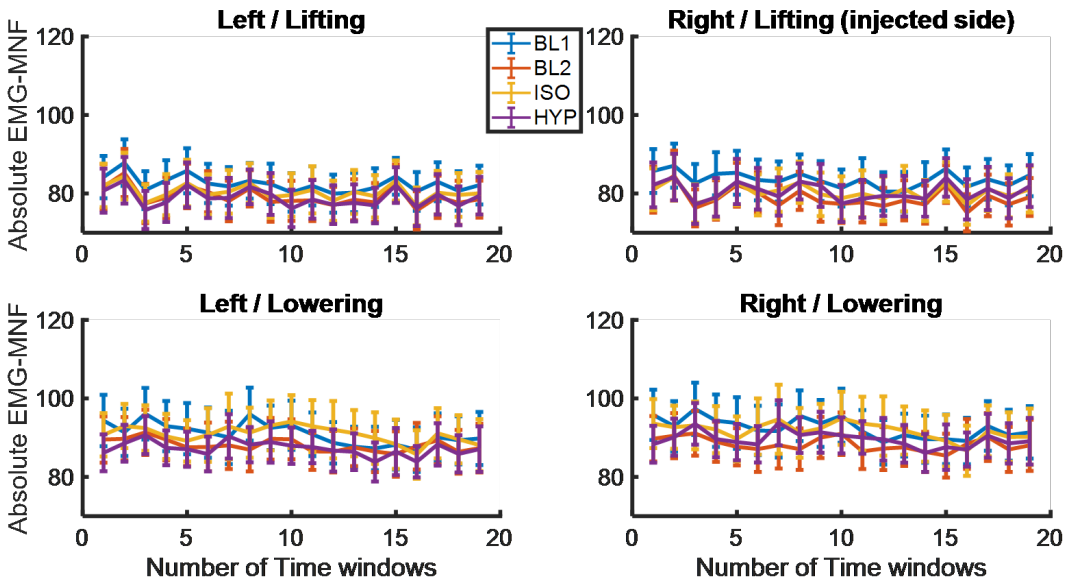

Figure 25: Assessment of MNF (Hz) divided in lifting and lowering

3.4.3 Centroid

The centroid was calculated from the RMS maps, Figure 26 shows a sample presentation.

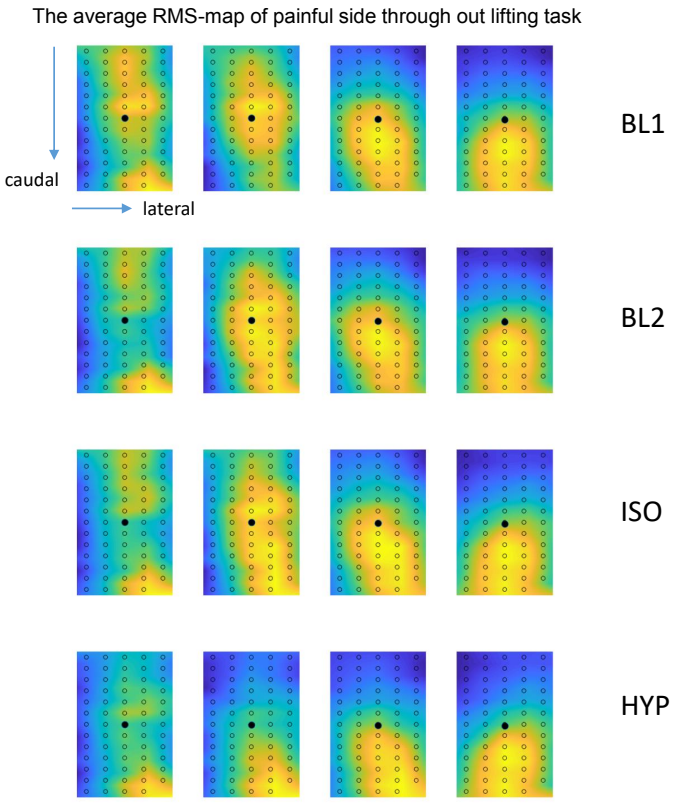

Figure 26: Example presentation of the right 2D high-density electrode signals for the four conditions, each divided and averaged over four time windows of the lifting task. The focus of activity is marked yellow. The X-axis represents the lateral/medial shift, the Y-axis represents the caudal/cranial shift

Firstly, the shift of the centroid was analysed within one task. Therefor the lifting as well as the lowering period were divided into eight time windows . The effect of task, side, condition, and time were investigated.

Within one task there were no significant differences between lifting and lowering ($p = 0.64$; $F = 0.237$), side ($p = 0.89$; $F = 0.019$), condition ($p = 0.95$; $F = 0.123$) and time ($p = 0.11$; $F = 2.866$) in X-axis.

In Y-axis, there were no significant differences between lifting and lowering ($p = 0.12$; $F = 2.63$), side ($p = 0.35$; $F = 0.92$) and condition ($p = 0.79$; $F = 0.11$). Over time there was a significant shift in caudal direction during lifting ($p < 0.05$; $F = 10.94$) (Figure 27), during lowering this effect was less evident (Figure 28).

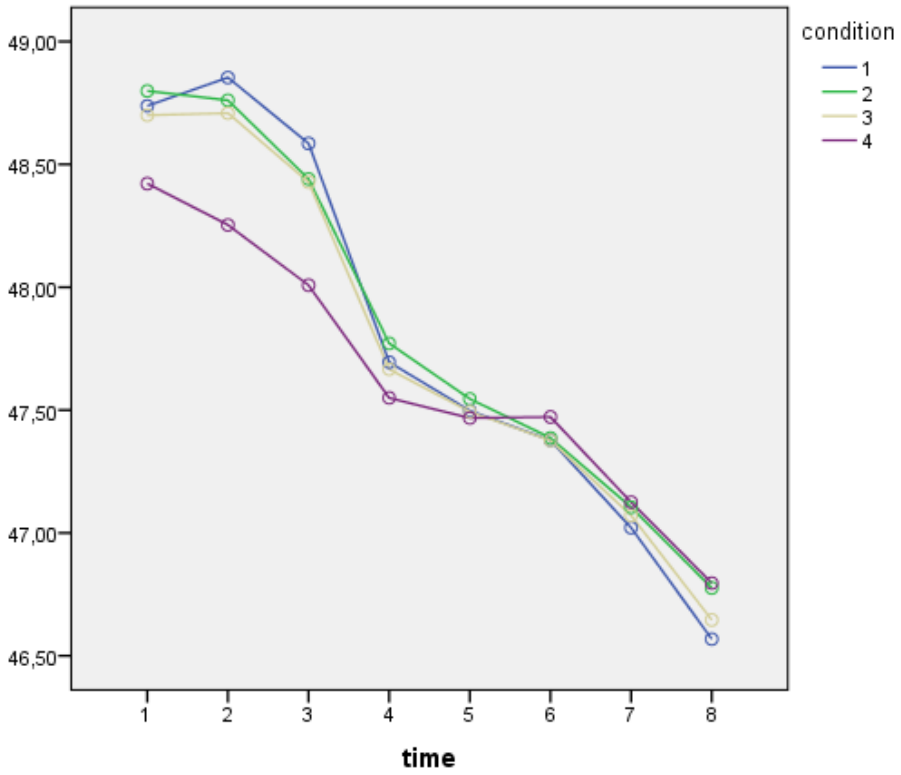

Figure 27: Shift of centroid (in mm) in Y-axis during lifting (1 = BL1; 2 = BL2; 3 = ISO; 4 = HYP), with the lifting phase divided into eight intervals

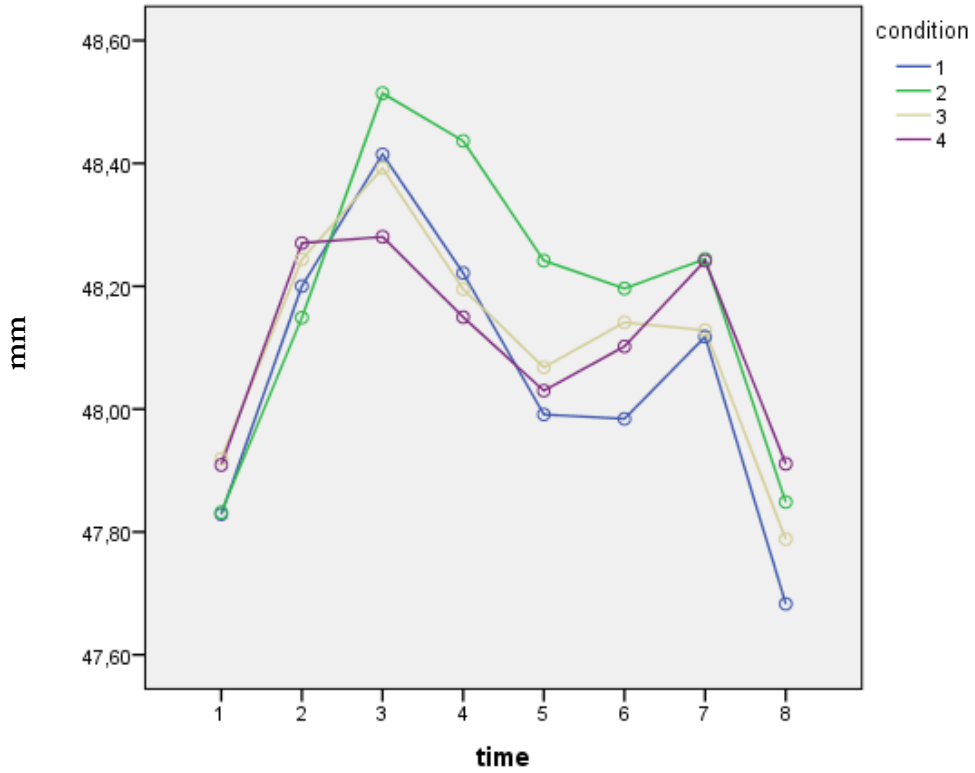

Figure 28: Shift of centroid (in mm) in Y-axis during lowering (1 = BL1; 2 = BL2; 3 = ISO; 4 = HYP), with the lowering phase divided into eight intervals

Moreover, the behaviour of the centroid along the repetitions was analysed.

There was no significant effect of the shift in X-axis (lateral/medial) for any condition ($p = 0.945$; $F = 0.123$).

In Y-axis (cranial/caudal), there was no significant difference between task (lifting and lowering) ($p = 0.08$; $F = 4.1$), side ($p = 0.48$; $F = 0.55$) and condition ($p = 0.36$; $F = 1.14$). Over the whole time of three minutes there was a small but significant shift in cranial direction ($p < 0.05$; $F = 3.83$), however there was no significant difference between the four conditions. Figure 29 shows this change of the centroid over the whole period of the task.

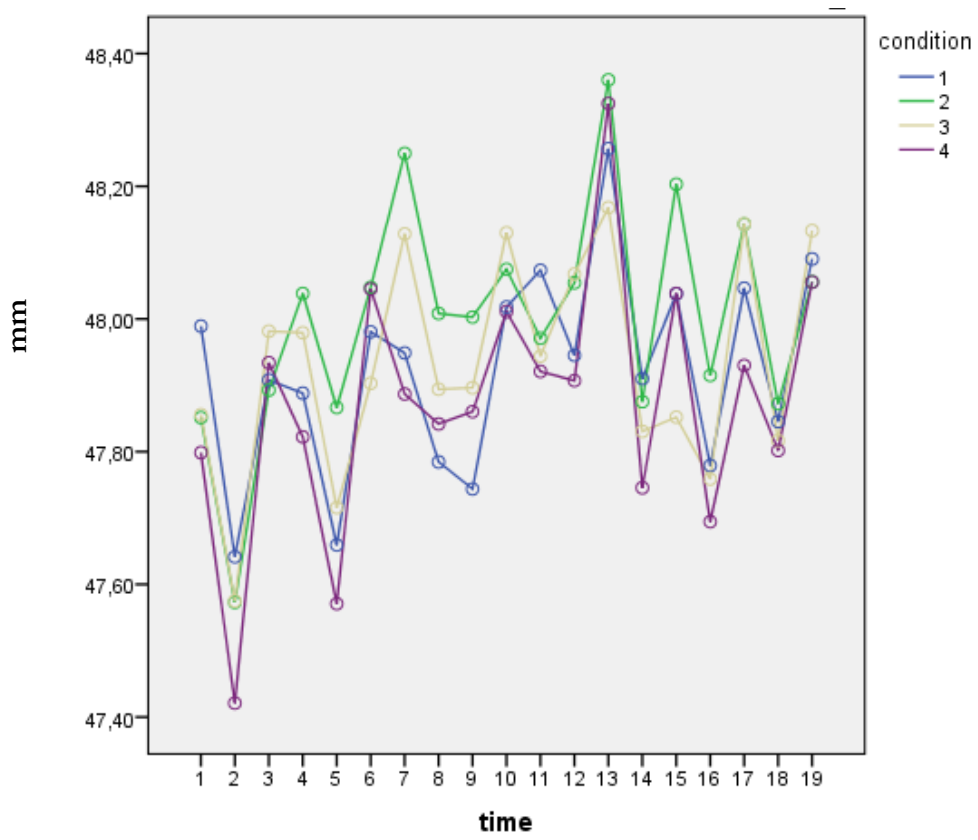

Figure 29: Shift of centroid (in mm) along repetitions in Y-axis (1 = BL1; 2 = BL2; 3 = ISO; 4 = HYP), with the whole session divided into 19 time intervals.

3.5 Motion of the spine during tasks.

3.5.1 Angles

The motion capture system provides information about the consistence of the repetitions and the spine angle. We calculated the mean spine angle between C7 and L5 in each condition for lifting and lowering for both sagittal (Figure 30) and coronal (Figure 32) planes. Then we applied RM-ANOVA to investigate differences.

The mean angles for the ZY plane for lifting are summarised in Table 14, the one for lowering in Table 15. Figure 31 shows the variation of the angles over the time of the task divided into 600 frames. The mean angles for the ZX plane for lifting are summarised in Table 16, the one for lowering in Table 17. The corresponding graphs are shown in Figure 33.

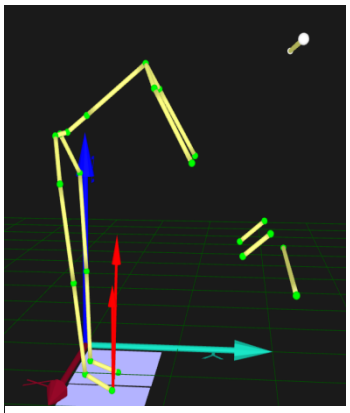

Figure 30: Model of the motion capture. The green arrow shows the Y-axis, the blue one the Z-axis, and the brown one the X-axis in a 3- dimensional coordinate system. The ZY plane represents the sagittal plane during movement

Table 14: Descriptive statistics of the average C7/L5 angle in ZY plane during lifting

Average Angle Lifting (degree)	Mean	SE
BL1	155	1
BL2	155	1
ISO	155	1
HYP	156	1

Table 15: Descriptive statistics of the average C7/L5 angle in ZY plane during lowering

Average Angle Lowering (degree)	Mean	SE
BL1	148	1
BL2	149	1
ISO	148	1
HYP	149	1

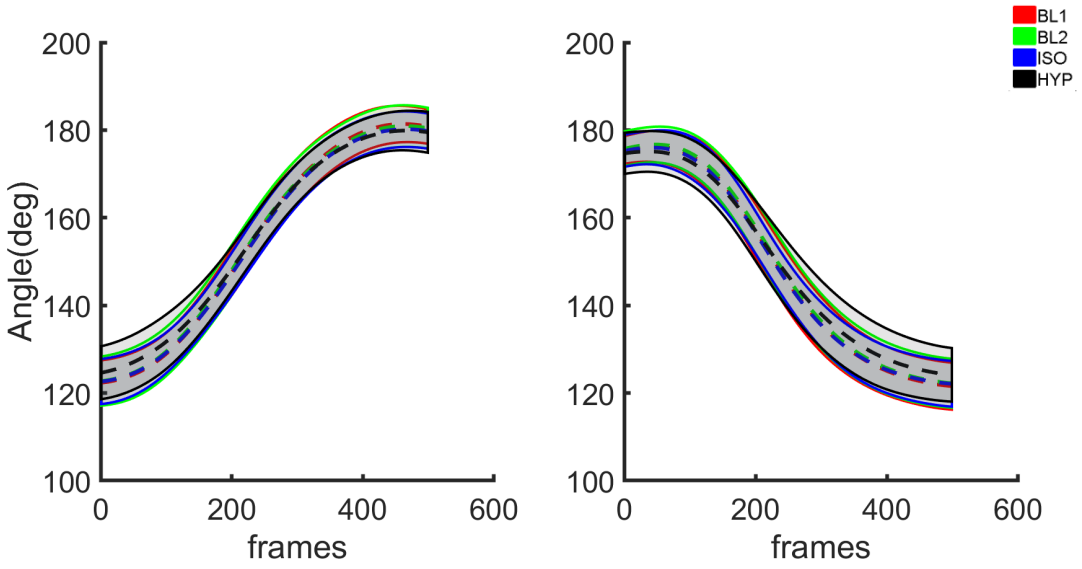

Figure 31: Spine angle between C7 and L5 and the Y-axis in the ZY plane during lifting (left graph) and lowering (right graph). The angles are the average over the whole time of the task. The grey area represents the variation of the data, the curves show the edges of the data. The data for the left graph are summarised in Table 14, the data for the right graph are summarised in Table 15

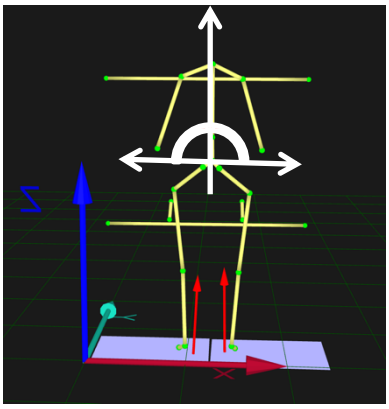

Figure 32: Model of the motion capture. The green arrow shows the Y-axis, the blue one the Z-axis, and the red one the X-axis in a 3- dimensional coordinate system. The ZX plane represents the coronal plane during movement

Table 16: Descriptive statistics of C7/L5 angle in ZX plane during lifting

Average Angle Lifting (degree)	Mean	SE
BL1	181	1
BL2	181	1
ISO	181	1
HYP	181	1

Table 17: Descriptive statistics of C7/L5 angle in ZX plane during lowering

Average Angle Lifting (degree)	Mean	SE
BL1	181	1
BL2	181	1
ISO	181	1
HYP	180	1

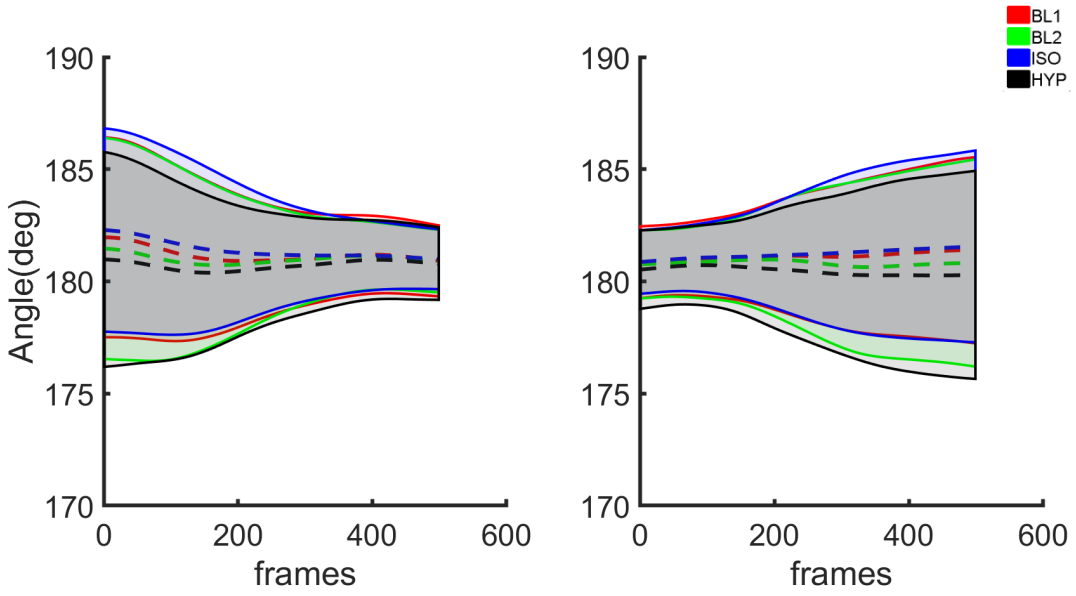

Figure 33: Spine angle between C7 and L5 and the X-axis in the ZX plane during lifting (left graph) and lowering (right graph). The angles are the average over the whole time of the task. The grey area represents the variation of the data, the curves show the edges of the data. The data for the left graph are summarised in Table 16, the data for the right graph are summarised in Table 17

There was no deviation of the spine angle in the ZY plane (Figure 31) in any condition ($p = 0.19$; $F = 1.68$). There were minor effects in the ZX plane ($p = 0.006$; $F = 4.68$) but significant only between ISO and HYP (Figure 33).

3.5.2 Coefficient of variations

Moreover, the regularity of lifting and lowering were investigated. Therefor the coefficient of variations (CoV) was calculated, likewise for both coronal and sagittal planes. It is defined as the ratio of the standard deviation to the mean. It is used to show the variability of the degrees related to the mean value. The mean values of the CoV in the ZY plane are summarised in Table 18 for lifting and in Table 19 for lowering. Figure 34 shows the mean CoV over the whole task divided into 600 frames for each condition.

Table 18: Descriptive statistics for regularity in ZY plane during lifting

Coefficient of variations Lifting (%)	Mean	SE
BL1	1.17	0.11
BL2	1.75	0.1
ISO	1.73	0.07
HYP	1.95	0.1

Table 19: Descriptive statistics for regularity in ZY plane during lowering

Coefficient of variations Lowering (%)	Mean	SE
BL1	2.02	0.1
BL2	1.97	0.1
ISO	2.06	0.13
HYP	1.99	0.1

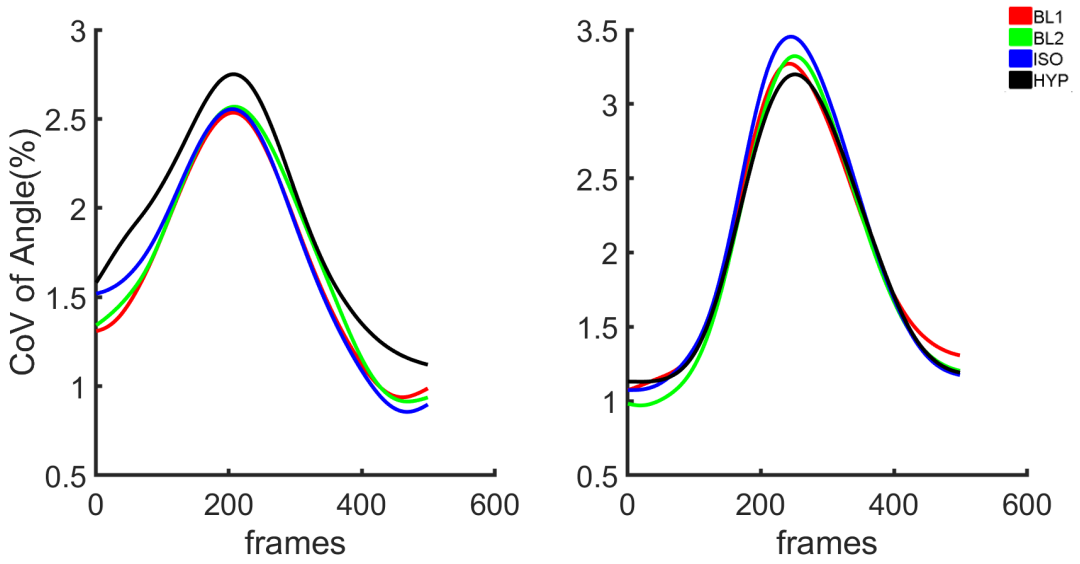

Figure 34: Average variability of movement in ZY plane during lifting (left graph) and lowering (right graph). The mean data of the CoV in ZX plane are summarised in Table 20 for lifting. Table 21 shows them for lowering. Figure 35 shows the mean CoV over all tasks for lifting and lowering divided into 600 frames for each condition.

Table 20: Descriptive statistics of regularity in ZX plane during lifting

Coefficient of variations Lifting (%)	Mean	SE
BL1	0.56	0.04
BL2	0.57	0.06
ISO	0.55	0.06
HYP	0.6	0.05

Table 21: Descriptive statistics of regularity in ZX plane during lowering

Coefficient of variations Lowering (%)	Mean	SE
BL1	0.63	0.05
BL2	0.63	0.07
ISO	0.62	0.05
HYP	0.67	0.07

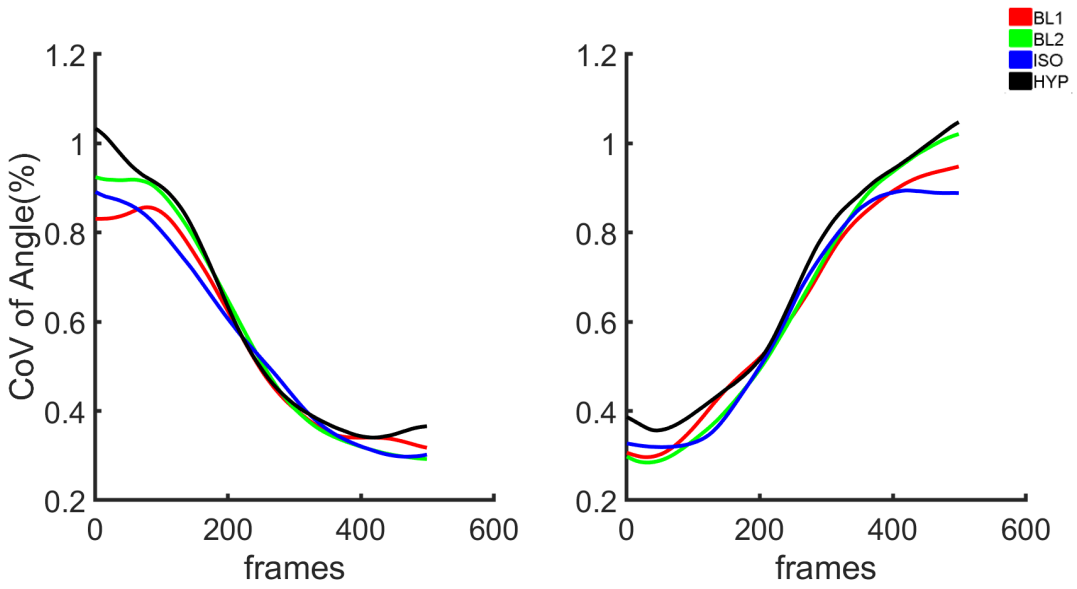

Figure 35: Average variability of movement in ZX plane during lifting (left graph) and lowering (right graph)

Calculations showed that there was no significant variability neither in ZY ($p = 0.326$; $F = 1.19$) nor in ZX ($p = 0.68$; $F = 0.35$) plane. Regarding the graphs, it seems as if the variability during hypertonic condition was higher during lifting in the ZY plane, compared to the other conditions (Figure 34, left graph, black curve). However, this tendency was not significant in our calculations.

3.6 Biomechanical assessment

Combining the information of the EMG and motion capture systems, it was possible to analyse the biomechanical delay which is the time interval between the muscle activation (EMG) and the beginning of the movement (motion capture). The EMD is explained in Figure 36. For the calculations, a repeated measurement ANOVA was used.

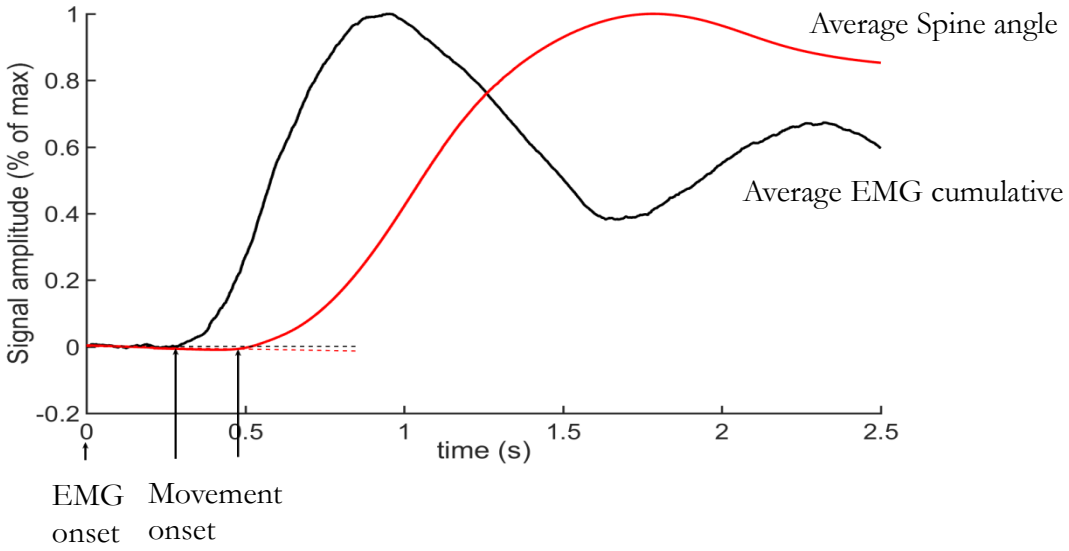

Figure 36: Chronological development of EMG signal and Motion capture. The EMG activity first increases and then oscillates due to muscle activity, followed by a detectable movement onset in the capture system. The EMD represents the difference between the two onsets (in ms).

Table 22 shows the mean values of the EMD for each condition. Figure 37 shows the corresponding graphs.

Table 22: Average electromechanical delay (time in ms)

	Mean	SE
Baseline 1	180	23
Baseline 2	172	24
ISO	163	30
HYP	116	10

It became apparent that the condition had a significant effect on the EMD ($F = 4.46$; $p < 0.05$). The delay was significantly lower in painful condition compared to BL1 in the post hoc tests (Table 23). There was no significant difference between the other conditions.

Table 23: Post hoc test (Bonferroni) of EMD in different conditions, corrected for multiple comparisons.

Sample	Significance
BL1-BL2	1
BL1- ISO	1
BL1 – HYP (*)	0.042
BL2 - ISO	1
BL2- HYP	0.104
ISO - HYP	0.645

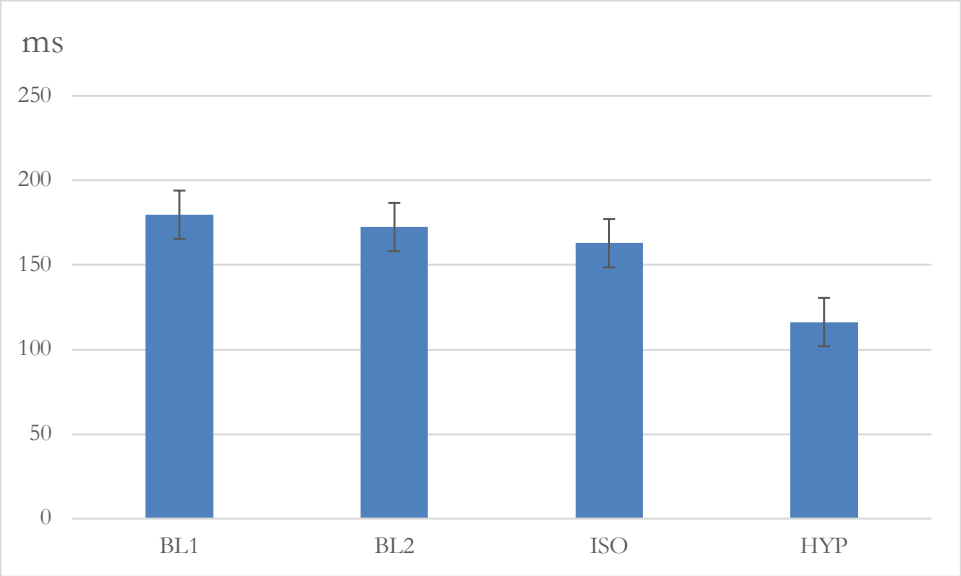

Figure 37: Average electromechanical delay (mean ± SE).

4 Discussion

The aim of the study was to characterize which changes in muscle activity and motion are triggered by an acute pain stimulus. We hypothesized that the injection of hypertonic saline into the back muscles would induce an acute pain sensation, which has an immediate effect on pain perception, the neuromuscular activity, as well as movement strategies. We assumed that this would be a consequence of exited activation of the nociceptive feedback system and suboptimal cortical control of movement.

4.1 Summary of results

Regarding the subjects' data, it became obvious that we worked with a homogenous group of 50 % male and 50 % female participants. Therefore, we did not divide our analysis into gender groups. Of all conditions, the participants reported the highest pain perception in the task after the hypertonic saline injection. Their PPT after the lifting task with hypertonic saline was significantly increased. This phenomenon was most explicit in the region of injection (P2, P3).

Moreover, the EMG activity was lower in painful condition than in the other conditions. It was lowest in painful condition on the injected side during lifting. The variation of the EMG amplitudes was also reduced in painful condition. The centroid shifted in longitudinal plane over the task irrespective of the condition. However, no shift in the transversal plane took place.

Regarding the motion, there was no deviation in the coronal plane nor in the sagittal plane. Moreover, there was no significant increase of variability of motion, although the graphs suggest an increase of variability in painful condition.

Combining the information of EMG and Motion Capture, it became obvious that the time interval between the onset of EMG and the onset of motion decreases after the injection of hypertonic saline compared to the non-painful trials.

4.2 Pain perception

With regard to the questionnaires, it can be assumed that all participants were pain free at the beginning of the experiment. Consequently, the pain they reported came solely from the repetitive lifting task and the injections. The very low pain intensity reported in condition BL1 and BL2 can be assessed as a slight exhaustion they experienced over the 3 minutes the lifting task lasts. In the ISO condition there was a slightly higher initial pain, probably related to the injection procedure quickly reaching the baseline levels. Although isotonic saline is not expected to induce pain due to its more "physiological" concentration, it still can lead to discomfort in thin muscles because of the volume of bolus (Graven-Nielsen et al. 1997b). This is coherent with our findings, that the participants reported only a very low or no pain sensation after the injection of isotonic saline. In contrast, there was a significant increase in pain after the hypertonic saline injection. Previous studies report similar pain levels following injection of hypertonic saline. Arendt-Nielsen et al. state that an intramuscular injection of hypertonic saline evokes a pain which is comparable to acute clinical muscle pain (Arendt-Nielsen and Graven-Nielsen 2008). A change of pain intensity over 30 % compared to the baseline is clinically relevant in LBP patients (Ostelo et al. 2008) having chronic pain. In this study, pain of about 4 on a NRS may thus reflect mild to moderate acute LBP. One limitation and difference to clinical pain, however, is the short duration of the induced pain that lasted only a few minutes.

4.3 Sensory adaptation

The PPT at the very beginning of the experiment was always the lowest value. This might be due to the fact that the participants needed some time to familiarise themselves with this method. Repetition of the measurement might be associated with less anxiety and more relaxation leading to higher thresholds. Another possible explanation might be that their muscles were still "cold" at the very beginning, while after the lifting task the pain sensitivity was reduced due to muscle activation and warm-up. Different forms of exercises also reduce pain sensitivity especially for pressure stimuli (Naugle et al. 2012; Misra et al. 2014; Jones et al. 2016; Vaegter et al. 2017). The so-called exercise-induced hypoalgesia (EIH) can be found in healthy subjects after exercises as an increased PPT (Vaegter et al. 2017). But not every

tissue type has the same effect. It has been shown that the activation of muscles, especially deeper muscles, causes a stronger descending inhibition in the dorsal horn of the spinal cord than cutaneous input. So muscle input may have a stronger hypoalgesic effect than cutaneous input (Vaegter et al. 2017). As underlying mechanisms the activation of A-delta- and C-fibres during a contraction might lead to an activation of segmental anti-nociceptive effects (Kosek and Lundberg 2003) and/or the activation of the central nociceptive descending system (Misra et al. 2014; Vaegter et al. 2017). This also explains why there is a slight increase of PPT over the whole time of the experiment. Moreover, the secretion of ß-endorphins and endogenous opioids to the peripheral blood may lead to altered pain perception (Kosek and Lundberg 2003).

In the present study, there was a trend for the hyperalgesic condition to have the highest pressure pain threshold. This means that after the most painful trial participants tended to be less sensitive for painful pressure stimulation (Figure 19-22). Especially in the region of P2 and 3, which were the location of the injection, the PPT is significantly higher than for all other conditions, interestingly in a bilateral pattern. This means that it comes to a hypoalgesia in the region where the hypertonic saline most likely has the highest concentration and effect. This is not coherent with findings of Arendt-Nielsen and Grave-Nielsen, who describe a decreased PPT after the injection of Capsaicin (Arendt-Nielsen and Graven-Nielsen 2008). However, another study with hypertonic saline described that an intramuscular injection of hypertonic saline led to a cutaneous hypoalgesia to pin prick stimuli (Graven-Nielsen et al. 1997b).

Pain perception is modified by several aspects related to nociceptive processing as well as psychological factors. Multiple studies have shown a relation between pain perception and emotions. Especially positive emotions are assumed to reduce pain (Villemure and Bushnell 2002). In our experiment PPT was measured a few minutes after the experimentally induced pain had mostly resolved possibly influencing the PPT-result for hypertonic saline. Another important aspect that needs to be considered is the "pain inhibits pain" model. The CPM (Central inhibition of pain) is the central inhibition of pain in a local area by a second painful stimulus which is applied elsewhere in the body (Willer et al. 1984). This means that the residual pain from the hypertonic saline and the pressure pain assessment may interact resulting in a CPM related increase in pain threshold.

While healthy individuals seem to be less sensitive to pain after a painful task it has been shown to be the other way around for patients with chronic low back pain. A recent study

with the same repetitive lifting task demonstrated decreased pain thresholds in patients with mild chronic low back pain (Falla et al. 2014). This supports the hypothesis that chronic pain patients express an altered pain processing resulting in a central sensitisation (Giesecke et al. 2006). Related to our initial question this means that an acute experimental pain stimulus mimicking acute low back pain is associated with decreased pain sensitivity and not with central sensitisation, seen in the processing of chronic pain.

4.4 Variation in muscle activity

The most outstanding result regarding muscle activity was that the muscle activity decreased in the painful condition. This phenomenon was most distinct on the injected side and is consistent with several previous studies (Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997a; Boudreau et al. 2011). Boudreau et al. investigated the activation of the M. erector spinae and M. obliquus externus during unexpected postural perturbations. They analysed the different behaviour of the muscles in healthy subjects before and after the injection of hypertonic saline. In their study, they describe a reduction of the EMG amplitude during postural perturbation after an unilateral injection of hypertonic saline (Boudreau et al. 2011). They state that although the injection as well as the reported pain were unilateral, the amplitude of the EMG was reduced bilaterally. Compared with our study, we also observed a reduction of the EMG activity. However, this effect was most obvious on the side of injection. A possible explanation might be that the lifting task in our study required different and possibly less complex muscle activation than the postural task. With regard to chronic pain patients, similar effects have been observed. Studies report a decreased EMG activation associated with LBP (Jacobs et al. 2011). Hodges et al. (2001) had a closer look at the muscle activation in LBP patients. They investigated different abdominal muscles during feedforward postural exercises. In this context it has been shown that muscle activity is reduced in LBP when the muscle works as an agonist while it is increased when it works as an antagonist (Hodges 2001). Consequently, it is suggested that the activity of the muscle is dependent on the work pattern (Arendt-Nielsen et al. 1996). This shows that not only the painful muscle but also the activities of synergistic and antagonist muscle are influenced by pain. This change in coordination is assumed to be the reason for overall altered motor control in pain. This may result in overloading of other regions and extended stress on parts of the muscles that take over the task of the painful muscle possibly causing referred pain

and worsening chronification (Arendt-Nielsen and Graven-Nielsen 2008). The findings of the current study suggest that the decline in muscle activity is an immediate result of pain itself as it is not dependent on the duration of pain. It is suggested that the decreased activity represents a (dys-)functional muscle adaptation to the nociceptive stimulus in order to reduce motion (Arendt-Nielsen and Graven-Nielsen 2008). Previous studies suggest that nociception influences the input from the motor cortex to motor neurons (Graven-Nielsen et al. 1997a; Yavuz et al. 2015). During pain the CNS is focusing on the pain stimulus which leads to a reduced performance in motor control (Moseley and Hodges 2005). It is hypothesized that the aim of the CNS is to reduce the stress on the painful tissue (van den Hoorn et al. 2015). The decreased neural drive to the painful back extensor leads to a decrease in EMG activity (Chiou et al. 2014). In parallel, a compensatory increase in muscle activity in non-painful regions in order to complete the required task has been described (Falla et al. 2007a). However, such a compensatory mechanism was not observed in the present study. This difference is most likely due to the functional role of the studied muscle since Falla et al. (2007) investigated the M. trapezius and not the M. erector spinae. On the other hand, the shift of the centroid, discussed in detail below, may be a compensatory mechanism in the current experimental set up. In addition, the altered motor control under experimental pain seems to be a predictor for future pain problems. Arendt-Nielsen and Graven-Nielsen suggest that the individual muscle activity during a repetitive task gives a forecast who of the trial subjects will develop chronic pain. They state that a decreased frequency and increased amplitude was found in patients who later developed chronic neck-pain. (Arendt-Nielsen and Graven-Nielsen 2008).

We investigated the behaviour of the centroid of EMG activity in two different perspectives, one within the overall average of all cycles of lifting and lowering the box across all subjects and repetitions and the second overall cycles over the whole three minutes of the repetitive lifting and lowering, averaged only across subjects. Both were calculated for each condition. Regarding the centroid of the activity distribution within one cycle of the lifting task there was a significant shift in caudal (Y-axis) direction during lifting. The shift was correlated with the trajectory of the movement during lifting. It has been shown that during bending forward and straightening up there is an increasing tension during extension and a relaxation during flexion in the M. erector spinae (Tanii and Masuda 1985). Investigations of the different back muscles indicate that during extension the M. erector spinae and M. multifidus are active to compensate gravity as well as weight while they are inactive in full extension (Tanii and Masuda 1985). A variation in force triggers the substitution of motor units and leads to a

redistribution of activity (Falla and Farina 2007). The shift of centroid might be the result of different parts of the muscles being active in order to stabilise and move the trunk during the lifting. Another possibility is that the shift reflects the straightening of the spine, evident by the correlation between the trajectory of the lifting movement and the observed shift. During lowering, the effect was less apparent which might be due to the less differentiated activity required to lower down the box.

Following the centroid along the time course over the repetitions of the task there was a significant cranial shift in any condition towards the end of the three minutes. One possible explanation is that the participants were exhausted, and the shift was caused by fatigue. Changes in the activation pattern are due to a variation in motor unit activity during a fatiguing task (Falla and Farina 2008). In order to fulfil a demanding task, there is both a recruitment of additional motor units and a rotation between the active muscle fibres. Units that were initially active reduce their firing rate while new motor units are recruited to keep up force. It is supposed that the accumulation of metabolites and afferent reflexes of motor neurons lead to this reorganisation of the activity pattern (Falla and Farina 2008). It should be noted that the electrical discharge is dependent on the region of the body. There is a heterogeneous reaction to fatigue within a muscle (Farina et al. 2008). Different regions of the muscle are innervated independently. Farina et al. saw a preference for recruiting cranial fibres of the trapezius during a submaximal isometric contraction over time. This led to a shift of the centre of activity from caudal to cranial which was measured as a shift of the centroid (Falla and Farina 2007; Falla and Farina 2008). A recent study by Falla et al. performed an investigation with a similar lifting task. They found that healthy people exhibit a caudal shift of activation, whereas people with chronic LBP did not show any variability of muscle activation (Falla et al. 2014). It has also been described that during fatiguing exercise activity increases in lower lumbar muscles rather than in parts situated more cranial. This has been observed for both isometric (van Dieen et al. 1993) and repetitive tasks (Bonato et al. 2002).

A study from Madeleine at al. investigated the shift of the centroid in the upper trapezius muscle in two conditions. During sustained contraction the centroid shifted in cranial direction (Madeleine et al. 2006). The observed changes were assumed to be caused by additional motor recruitment in a fatiguing task which is in line with our findings. Moreover, they investigated the behaviour of the centroid following induction of experimental pain.

The RMS was reduced, and the centroid shifted in caudal direction after the injection of hypertonic saline. Madeleine at al. suggest that local excitation of nociceptive afferents result in an increased variability in force modulation (Madeleine et al. 2006). The centroid therefore shifts away from the region where the saline was injected. In our study there was no significant shift of the centroid in the painful condition compared to the others. However, similar observations regarding the decrease of RMS were made. Since there were different observations in previous studies, we assume that the effect in our study possibly was too subtle. During the repetitive movement, some electrodes lost contact over the tasks, which might be the reason why some data were not as precisely as they should. In pain a muscle would typically stiffen which leads to a shortening of fibres. The combination of the information of EMG and motion capture support this theory (see electromechanical delay). Moreover, the muscle parts around the area of injection exhibit a reduced force production capacity as mentioned above. Previous studies with experimentally induced pain state that not only the non-affected parts but rather all agonistic parts are recruited in order to fulfil the required task (van den Hoorn et al. 2015). This means that the centroid of activation shifts away from the directly pain affected fibres, while the more cranial or caudal ones take over the task. According to the pain adaption model (Lund et al. 1991) the activity in pain is decreased in the agonist muscle and increased in the antagonist muscle. It has been shown that the injection of hypertonic saline reduces the discharge rate of motor units due to central inhibitory mechanisms (Farina et al. 2004). In order to fulfil the required task a spatial redistribution within the painful muscle occurs.

It has been shown that a greater change of activity is associated with a reduced fatigue (Falla and Farina 2007). Previous investigations have shown that both muscle fatigue and pain can lead to impaired spinal control and therefore hold an increased risk of injury (Jubany et al. 2017). The redistribution of activity protects the muscle from regional overload and allows a longer endurance of the force (Mathiassen et al. 2003; Farina et al. 2008).

4.5 Movement strategy in induced pain

The physiology of the musculoskeletal system is based on a bilateral and symmetric architecture. However, pain and limitations of motion often occur unilaterally (Kim et al.

2013b). Impaired motion can lead to imbalance in load on passive and active tissue which might lead to trauma or worsen the symptoms (Hernandez et al. 2017). Motion Capture systems are a well-established method to investigate the range, execution, and acceleration of movement (see chapter 1.7). It has been utilized for assessment of painful movement in order to qualify the relationship between motion, posture and the development and persistence of LBP (Hernandez et al. 2017). With regard to this several studies investigated differences in the motion patterns of patients with LBP and healthy controls. While most studies agree that lumbopelvic motion is altered in LBP there are controversial findings according to the kind of change. Some studies report that lumbar spine movement is reduced in LBP (Paquet et al. 1994) whereas other studies show an increase in spinal motion (Esola et al. 1996). It is supposed that these inconsistent findings are due to the heterogeneity of LBP symptoms, locations and compensatory strategies. For that reason LBP patients have been divided into subgroups according to their symptoms (Kim et al. 2013a; Gombatto et al. 2017). However, the present study investigated experimental pain in healthy participants. Therefore, a division into different symptom related subgroups is not relevant for this study.

In the study, the angle of C7 and L5 was calculated during lifting and lowering. Participants did not reveal any significant differences between the non-painful and the painful condition in any measurement. This means that acute (unilateral) pain seems to have no effect on the spine angle. In contrast to this previous studies report that patients with LBP exhibit a reduced movement range (Boston et al. 1995) and a reduced velocity (Lee et al. 2011) and acceleration (Granata and Sanford 2000). A possible explanation is that chronic pain patients develop adaptive mechanisms to reduce spinal pain over time. Moreover, patients show an impaired mobility in the lumbar spine and hip, which leads to a compromised function (Shum et al. 2005). Depending on the task, LBP patients exhibit kinematic differences (Shum et al. 2005; Shum et al. 2007). LBP in the lumbar spine contributes less to the total movement (Shum et al. 2005). Especially in the sagittal plane the motion of the lower lumbar spine is reduced (Hernandez et al. 2017; Mitchell et al. 2017). In order to accomplish the required task compensatory movements from other joints are utilized (Gombatto et al. 2017; Hernandez et al. 2017; Mitchell et al. 2017). A recent study by Dubois et al. compared people with chronic LBP and healthy subjects with experimental acute pain in a trunk flexion task. They found that although both groups showed changes in muscle activity, only the chronic LBP group adopted a different motion strategy with reduced contribution of the lower back (Dubois et al. 2011). This is in line with the observation that healthy participants with acute pain do not change the degree of flexion.

Boston et al. (1995) suggest that patients with LBP try to minimise the pain by compensating a decreased spine motion with alternative lifting methods, for example leg lift (Boston et al. 1995). This leads to increased stress on the lower extremities, which might cause further injuries and/or pain. Furthermore, LBP patients exhibit greater axial rotation than healthy controls (Kim et al. 2013b; Hernandez et al. 2017; Mitchell et al. 2017). This compensatory mechanism puts an increased stress on the lumbopelvic region and therefore leads to instability and injury (Kim et al. 2013b). Thus, one can assume that compensatory strategies of motor control resulting in adaptive motion may lead to a progressive increase in chronicity.

The origin of the limited motion in chronic LBP has not been clarified yet. A possible explanation is the intention to protect the painful tissue from further damage or termination of movement due to pain, or psychological factors (Shum et al. 2005). Moreover, the long lasting character of LBP might have caused alteration in the painful tissue such as stiffening and shortening directly reducing the lumbar motion (Gombatto et al. 2017).Not only pain itself may contribute to altered lifting strategies, also fear of pain can cause avoidance behaviours (Pfingsten 2004). This leads to the assumption that changes in motion strategies have a multifactorial origin that needs some duration of pain to develop. This hypothesis is in line with our findings since the acute pain stimulus was not "clinically relevant" enough to transfer the participants into reduced motion.

Although there was no significantly higher variability of movement during pain, the graph (Figure 34, left graph, black curve) and the clinical observations suggest that the lifting task was completed less consistently during pain especially during lifting in the sagittal plane. This goes with the findings of previous studies. This lifting period is of major interest, as it is the time when the lumbar muscles have the highest activity. There was no significant deviation in the coronal plane. It has been suggested that movement variability may serve as an "index of motor control" (Mitchell et al. 2017). This means that a high variability corresponds to a decreased motor control. However, this statement has caused considerable controversy. Lamoth et al. (2006) state that the variability is dependent on the investigated plane describing a reduced kinematic variability in the transversal plane during gait, as well as an increased variability in the coronal plane (Lamoth et al. 2006). Healthy people show changes in motion during a fatiguing task (Bonato et al. 2002), suggesting that healthy people use a

high variability in motion in order to apportion the stress between the muscle fibres. In contrast, people with LBP show a rigid repertoire of motion strategies assuming that they should stabilise the spine (Jacobs et al. 2009; Boudreau et al. 2011). This strategy carries a high risk of overload and due to the fatigue a higher risk of injury (Magnusson et al. 1996). Therefore, it seems like the variability of motion in pain is a distinctive feature for patients with chronic LBP and healthy people. Regarding our study there probably was a significant effect in certain episodes during the lifting but the statistical analysis of the mean values was not significant. A possible explanation is that the pain faded over the 3 minutes of lifting. By using the mean of all liftings during the 3 minutes slight effects might disappear. For more meaningful statements a different experimental set-up might be sensible (e.g., a constant injection of hypertonic saline by pump).

4.6 Muscle stiffness and pain relation

The combined information of EMG and Motion Caption showed that the electromechanical delay is significantly shorter in the painful condition compared to the other conditions. The electromechanical delay (EMD) is defined as the latency between the onset of activity in the skeletal muscle and the development of tension or movement at the joint (Cavanagh and Komi 1979). This delay corresponds to the time lapse between the translation of tension to the joint and the arrival of an action potential at the tubuli starting contraction of the sarcomeres (Cavanagh and Komi 1979). However, the greatest amount of electromechanical delay is assumed to be the time interval from the contraction of the contractile elements to the point when the serial elastic components (SEC) are stretched (Hill 1950; Cavanagh and Komi 1979; Moore et al. 2002). Consequently, any tension of the muscle is supposed to reduce the EMD by pre-stretching the SEC (Zhou et al. 1995, Yavuz et al. 2010). Injection of hypertonic saline shortened the EMD. The resulting pre-tension has been observed in previous studies where an increased muscle activity has been found after the injection of hypertonic saline. Although the muscle was expected to be silent at that point there was an increased EMG activity in the painful condition (Arendt-Nielsen and Graven-Nielsen 2008). The reduction of EMD after the injection of hypertonic saline cannot be explained with fatigue over time. Previous studies report an increase in EMD in a fatiguing task. This can be explained with an impairment of the action potential travelling along the muscle membrane (Yavuz et al. 2010). The reduced conductivity leads to a decrease in calcium ion

release from the sarcoplasmic reticulum, eventually resulting in a reduced rate of force (Zhou et al. 1996a).

The effect of pre-tension has also been observed in chronic LBP patients and is assumed to be a strategy to protect painful regions (Magnusson et al. 1996). It is suggested that especially repetitive tasks with a high frequency of cycling load, as it was demanded in the study at hand, carry a high risk for neuromuscular disorders. The recurrent activity allows only little rest and can lead to trauma and pain. Consequently, protecting mechanisms like stiffening occur (Lu et al. 2008). Jones et al. (2012b) suggest that the stiffening leads to a co-activation of trunk muscles in order to improve the response to external perturbations (Jones et al. 2012b). Pretension also seems to be a sensible compensating mechanism since the reaction time is increased in patients with LBP. Different studies report a longer onset latency and slower reaction time for trunk extensors in a reaching task and reaction to sudden load in people with LBP (Radebold et al. 2000; Thomas et al. 2007). A possible explanation is either the inability or unwillingness to use the muscle effectively (Boston et al. 1995). Additionally, faster developing fatigue due to less training might be a reason. Delayed muscle response is associated with a higher risk of injuries (Cholewicki et al. 2005; Yavuz et al. 2010). For patients with LBP this means that an impairment of spinal control carries a high risk of injury (Hodges and Richardson 1999). In LBP muscle fibre atrophy occurs (Chiou et al. 2014). It has been shown that special training decreases EMD (Zhou et al. 1996b) whereas immobility increases the EMD (Kubo et al. 2000). Increasing slackness of the SECs results in a longer EMD which means that the protecting mechanism might fail after a long period of rest due to pain.

In summary, in a healthy participant the painful sensation leads to a stiffening of the muscle in order to stabilise and protect the spine from further damage. When these protection mechanisms fail, due to fatigue or a muscle loss, the risk of further injury increases.

4.7 Methodological limitations

We used high-density surface EMG electrodes in order to examine the variation in muscle activity during experimentally induced pain. The high-density surface EMG (HDsEMG) is a convenient method for estimating special distribution of muscle activity as well as the variation in the weighted centre of this distribution through different conditions (baseline, test and control) (Falla et al. 2007a; Arendt-Nielsen and Graven-Nielsen 2008; Boudreau et al. 2011). Moreover, we placed two HDsEMG electrodes bilaterally in order to compare muscle activity from the injected (right side) and the non-injected side (left side). With this set up also indirect effects of the acute pain stimulus could be detected. The electrodes were placed on the lower back paraspinal from the level of L5 to approximately L1 to obtain the highest signal from the M. erector spinae. The EMG signal was pre-processed in order to eliminate noise components. Therefore, well-established digital filtering methods were used (Konrad 2011). The HDsEMG implies recording from 64 individual channels, where an improper contact to skin surface may be observed occasionally. In case of poor connections, the signal obtained from that individual electrode cannot be filtered using known filtering methods. We tried to fix this problem with thorough fixation and preparation. Before the actual test run took place, the EMG signal was checked and if any channel gave a poor signal the skin contact was improved by additional fixation. In case we were not able to enhance the signal of one single channel, a mean signal from the surrounding channels was calculated We recorded the EMG signal in monopolar mode. The advantage of recording monopolar is that it gives information about the deeper muscles like the M. erector spina (Merletti et al. 1990). In the study these were muscles of our major interest. Moreover, monopolar recording provides more signal information that are lost in differential mode. Because of this we accepted the disadvantage of this mode that there is more noise contaminating the signal than in the differential mode. We counteracted this problem with amplification and filtering. However, this might be the explanation that the results of centroid motion were rather poor. In order to examine if the changes correlate with the intensity of the experienced pain the participants were asked to rate the pain on a scale from 0 to 10. By reporting the pain level orally, it was possible to note changes immediately during the task. The Numeric Rating Scale is a standard item to rate pain in clinical practise. Moreover, we expected changes in motion under pain. We used a motion capture system to get information about the range of motion during the different conditions. Motion capture is a well-established method to gain information about altered motion strategies in LBP (Boston et al. 1995; Bonato et al. 2003; Kim et al. 2013b). Finally, we were interested in the question if the experience of muscular

pain leads to a change in pressure pain perception. Although some participants struggled to report the exact pressure level when they started to feel pain at the very beginning, they quickly got used to this method. This method was inspired by the study of Falla et al. (Falla et al. 2014), where changes in PPT have been observed in participants with LBP after a lifting task. In our study, the PPT was measured at the very beginning and after each trail to be able to get both changes due to exercise (BL1, BL2) and changes due to pain (Iso, Hyper). However, there was of course an overlap after the hypertonic injection.

The main shortcoming was the limitation of the maximal PPT. When a participant did not report any pain until a pressure of 1000 kPa, the value 1000 was reported. This leads to insecurity about the real pressure pain threshold and might have caused inaccuracy in out estimations.

5 Summary

The study investigated the effect of an acute noxious stimulus on pain perception, muscle activity and movement during a repetitive lifting task.

Although the hypertonic saline injection led to a pain intensity that is comparable to short lasting mild to moderate pain in patients with (acute) LBP, the effects of acute pain in healthy participants are quite different from the effects of chronic LBP in affected patients. First of all, the participants were less sensitive to pressure pain stimuli after the painful injection. This can be seen as a result of segmental inhibition of pain, increased descending inhibition and/or exercise-induced hypoalgesia.

Secondly, the reduction of muscle activity in pain seems to be an immediate result and is independent of the duration of pain. It is assumed that this may serve to protect the painful parts from further damage. Just as healthy people, the participants under acute pain showed slight shifts of the centre of activity during the task avoiding an overload to certain regions of the muscle. This mechanism seems to be lost or ineffective in chronic pain.

Regarding the Motion Capture, no change of the spine angles C7 and L5 could be observed during pain. It seems like there was a tendency for increased variability during pain, although the calculations did not reveal a significant effect. Again, this could be a difference between chronic and acute low back pain as chronic LBP patients maintain a quite rigid strategy to complete the required task.

Finally, the electromechanical delay between neuronal input and muscle movement decreased in acute pain possibly due to the pretension of the muscle. In contrast, patients with LBP show a slower reaction.

In summary, healthy people with an acute back pain stimulus exhibit a quite different behaviour of muscle activity and motion than chronic pain patients. Probably these mechanisms shall avoid further damage. In contrast to this, these mechanisms are lost in chronic pain patients. We assume that this holds the key for further therapy approaches in chronic pain patients.

6 Appendix

Check List Injection

	Check	Addition
Preparation		
30 small Tapes		
10 big Tapes		
26 Reflectors		
Wet bracelet		
Calibration		
Prepare box and shelf (reflectors, no other reflection), ultrasound, saline iso- and hyper, syringe, needles, iPad		
Patient/Subject Treatment		
Sex		
Age		
Height		
Weight		
Information handed		
Explanation of task		
Questionnaire		
Distance finger/floor		
Schober (S1+10) Flex. (5cm)		

	Check	Addition
Ott (C7-30) Flex. (3-4 cm)		
Mark standing position		
Numeric pain scale (0-10)		
Pain Pressure Threshold (lying)		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L		_____/_____
2. R/L **3. R/L**		_____/_____ _____/_____
4. R/L **5. R/L** **6. R/L**		_____/_____ _____/_____ _____/_____
7. R/L **8. R/L**		_____/_____ _____/_____
Need to go to the toilet?		
Show task to subject		
Ask subject to take off clothes		
Fix electrodes		

	Check	Addition
C7 **Acromion ri**		
Acromion le		
Epicondylus humeri ri		
Epicondylus humeri le		
L1 (upper edge of EMG)		
L5 (lower edge of EMG) **Triangle under L5 (1 finger below L5, 2 fingers laterally)**		
Trochanter maj. of femur ri		
Trochanter maj. of femur le		
lat. Condylus fem. ri **lat. Condylus fem. le**		
lat Malleolus ri		
lat Malleolus le **5. Metatarsus ri** **5. Metatarsus le**		
Fix EMG		
Clean with abrasive gel and alcohol pads **2 cm lat L5-L2 ri** **2 cm lat L5-L2 le**		
Create model (before every task step		

	Check	Addition
on force plate)		
show task to subject **64 sec quiet standing**		
64 sec closed eyes		
Lifting task test run 1 min (10 cycles) beat 60 (without weight) **Lifting task 3 min (25 cycles)**		
Numeric pain scale (0-10)		
5 cycles		
10 cycles		
15 cycles		
20 cycles **25 cycles**		
Pain Pressure Threshold (around EMG)		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L		_____ / _____
2. R/L		_____ / _____
3. R/L		_____ / _____
4. R/L		_____ / _____
5. R/L		_____ / _____
6. R/L		_____ / _____
7. R/L		_____ / _____

	Check	Addition
8. R/L		_____ / _____
Injection isotonic saline/without		
Numeric pain scale (0-10)		
Lifting task 3 min (25 cycles)		
Numeric pain scale (0-10)		
5 cycles		
10 cycles		
15 cycles		
20 cycles		
25 cycles		
Pain Pressure Threshold (around EMG)		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L		_____ / _____
2. R/L		_____ / _____
3. R/L		_____ / _____
4. R/L		_____ / _____
5. R/L		_____ / _____
6. R/L		_____ / _____
7. R/L		_____ / _____
8. R/L		_____ / _____
Wait 20 minutes (sitting)		

	Check	Addition
Injection isotonic saline/without		
Numeric pain scale (0-10)		
Lifting task 3 min (25 cycles)		
Numeric pain scale (0-10)		
5 cycles		
10 cycles		
15 cycles **20 cycles**		
25 cycles		
Pain Pressure Threshold (around EMG)		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L **2. R/L**		_____ / _____ _____ / _____
3. R/L **4. R/L**		_____ / _____ _____ / _____
5. R/L **6. R/L**		_____ / _____ _____ / _____
7. R/L **8. R/L**		_____ / _____ _____ / _____
Wait 20 minutes (sitting)		
Injection hypertonic saline		

	Check	Addition
Numeric pain scale (0-10)		
Lifting task 3 min (25 cycles)		
Numeric pain scale (0-10)		
5 cycles		
10 cycles		
15 cycles		
20 cycles **25 cycles**		
Pain Pressure Threshold		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L		_____ / _____
2. R/L		_____ / _____
3. R/L		_____ / _____
4. R/L		_____ / _____
5. R/L		_____ / _____
6. R/L		_____ / _____
7. R/L **8. R/L**		_____ / _____ _____ / _____
Wait 20 minutes		
Lifting task 3 min (25 cycles)		
Numeric pain scale (0-10)		

	Check	Addition
5 cycles **10 cycles**		
15 cycles		
20 cycles		
25 cycles		
Put off EMG		
Pain Pressure Threshold		„wenn Sie den Reiz als Schmerz empfinden“
1. R/L		_____/_____
2. R/L		_____/_____
3. R/L **4. R/L**		_____/_____ _____/_____
5. R/L		_____/_____
6. R/L **7.R/L** **8. R/L**		_____/_____ _____/_____ _____/_____
Control tibialis ant R		

	Check	Addition
Control tibialis ant L **Control thumb R**		
Control thumb L		
Put off all systems		
Pain scale after 3 min (after end of task)		

Sf 12

Wie würden Sie Ihren Gesundheitszustand im Allgemeinen beschreiben?

ausgezeichnet	sehr gut	gut	weniger gut	schlecht
☐	☐	☐	☐	☐

Im Folgenden sind einige Tätigkeiten beschrieben, die Sie vielleicht an einem normalen Tag ausüben.

Sind Sie durch Ihren derzeitigen Gesundheitszustand bei diesen Tätigkeiten eingeschränkt? Wenn ja, wie stark?

	ja, stark eingeschränkt	ja, etwas eingeschränkt	nein, überhaupt **nicht** eingeschränkt
Mittelschwere Tätigkeiten, z.B. einen Tisch verschieben, staubsaugen, kegeln, Tennis spielen	☐	☐	☐
Mehrere Treppenabsätze steigen	☐	☐	☐

Hatten Sie in den vergangenen 4 Wochen *aufgrund Ihrer körperlichen Gesundheit* irgendwelche Schwierigkeiten bei der Arbeit oder anderen alltäglichen Tätigkeiten im Beruf bzw. zu Hause?

	ja	nein
Ich habe weniger geschafft als ich wollte	☐	☐
Ich konnte nur bestimmte Dinge tun	☐	☐

Hatten Sie in den vergangenen 4 Wochen *aufgrund seelischer Probleme* irgendwelche Schwierigkeiten bei der Arbeit oder anderen alltäglichen Tätigkeiten im Beruf bzw. zu Hause (z.B. weil Sie sich niedergeschlagen oder ängstlich fühlten)?

	ja	nein
Ich habe weniger geschafft als ich wollte	☐	☐

Ich konnte nicht so sorgfältig wie üblich arbeiten	☐	☐

Inwieweit haben die Schmerzen Sie in den vergangenen 4 Wochen bei der Ausübung Ihrer Alltagstätigkeiten zu Hause und im Beruf behindert?

überhaupt nicht	ein bisschen	mäßig	ziemlich	sehr
☐	☐	☐	☐	☐

In diesen Fragen geht es darum, wie Sie sich fühlen und wie es Ihnen in den vergangenen 4 Wochen ergangen ist.

Wie oft waren Sie in den vergangenen 4 Wochen…

	immer	meistens	ziemlich	manchmal	selten	nie
... ruhig und gelassen	☐	☐	☐	☐	☐	☐
... voller Energie	☐	☐	☐	☐	☐	☐
... entmutigt und traurig	☐	☐	☐	☐	☐	☐

Wie häufig haben Ihre körperliche Gesundheit oder seelischen Probleme in den vergangenen 4 Wochen Ihre Kontakte zu anderen Menschen (Besuche bei Freunden, usw.) beeinträchtigt?

immer	meistens	manchmal	selten	nie
☐	☐	☐	☐	☐

Schmerzfragebogen Seite 5 Schmerzklinik UMG

Fragen zu Ihrem Befinden
Bearbeitungshinweis: Bitte lesen Sie jede Aussage und kreuzen Sie die Zahl 0, 1, 2 oder 3 an, die angeben soll, wie sehr die Aussage **während der letzten Woche** auf Sie zutraf. Es gibt keine richtigen oder falschen Antworten. Versuchen Sie, sich spontan für eine Antwort zu entscheiden.

0 Traf **gar nicht** auf mich zu
1 Traf **bis zu einem gewissen Grad** auf mich zu oder **manchmal**
2 Traf **in beträchtlichem Maße** auf mich zu oder **ziemlich oft**
3 Traf **sehr stark** auf mich zu oder **die meiste Zeit**

1. Ich fand es schwer, mich zu beruhigen.	0	1	2	3	S
2. Ich spürte, dass mein Mund trocken war.	0	1	2	3	A
3. Ich konnte überhaupt keine positiven Gefühle mehr erleben.	0	1	2	3	D
4. Ich hatte Atemprobleme (z.B. übermäßig schnelles Atmen, Atemlosigkeit ohne körperliche Anstrengung).	0	1	2	3	A
5. Es fiel mir schwer, mich dazu aufzuraffen, Dinge zu erledigen.	0	1	2	3	D
6. Ich tendierte dazu, auf Situationen überzureagieren.	0	1	2	3	S
7. Ich zitterte (z.B. an den Händen).	0	1	2	3	A
8. Ich fand alles anstrengend.	0	1	2	3	S
9. Ich machte mir Sorgen über Situationen, in denen ich in Panik geraten und mich lächerlich machen könnte.	0	1	2	3	A
10. Ich hatte das Gefühl, dass ich mich auf nichts mehr freuen konnte.	0	1	2	3	D
11. Ich bemerkte, dass ich mich schnell aufregte.	0	1	2	3	S
12. Ich fand es schwierig, mich zu entspannen.	0	1	2	3	S
13. Ich fühlte mich niedergeschlagen und traurig.	0	1	2	3	D
14. Ich reagierte ungehalten auf alles, was mich davon abhielt, meine momentane Tätigkeit fortzuführen.	0	1	2	3	S
15. Ich fühlte mich einer Panik nahe.	0	1	2	3	A
16. Ich war nicht in der Lage, mich für irgendetwas zu begeistern.	0	1	2	3	D
17. Ich fühlte mich als Person nicht viel wert.	0	1	2	3	D
18. Ich fand mich ziemlich empfindlich.	0	1	2	3	S
19. Ich habe meinen Herzschlag gespürt, ohne dass ich mich körperlich angestrengt hatte (z.B. Gefühl von Herzrasen oder Herzstolpern).	0	1	2	3	A
20. Ich fühlte mich grundlos ängstlich.	0	1	2	3	A
21. Ich empfand das Leben als sinnlos.	0	1	2	3	D

Diese Zeile bitte nicht ausfüllen:
D: _____ A: _____ S: _____

7 Literature

Andersson GB (1999): Epidemiological features of chronic low-back pain. Lancet 354, 581–585

Andersson GB, Ortengren R (1984): Assessment of back load in assemblyline work using electromyography. Ergonomics 27, 1157–1168

Arendt-Nielsen L, Graven-Nielsen T (2008): Muscle pain: Sensory implications and interaction with motor control. Clin J Pain 24, 291–298

Arendt-Nielsen L, Graven-Nielsen T, Svarrer H, Svensson P (1996): The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 64, 231–40

Balagué F, Mannion AF, Pellisé F, Cedraschi C (2007): Clinical update: low back pain. Lancet 369, 726–728

Beck TW, Housh TJ, Cramer JT, Malek MH, Mielke M, Hendrix R, Weir JP (2007): A comparison of monopolar and bipolar recording techniques for examining the patterns of responses for electromyographic amplitude and mean power frequency versus isometric torque for the vastus lateralis muscle. J Neurosci Methods 166, 159–167

Berlit P: Basiswissen Neurologie. Springer-Verlag 2013

Bigos SJ, Battie MC, Spengler DM, Fisher LD, Fordyce WE, Hansson T, Nachemson AL, Zeh J (1992): A longitudinal, prospective study of industrial back injury reporting. Clin Orthop Relat Res 279, 21–34

Bonato P, Boissy P, Della Croce U, Roy SH (2002): Changes in the surface EMG signal and the biomechanics of motion during a repetitive lifting task. IEEE Trans Neural Syst Rehabil Eng 10, 38–47

Bonato P, Ebenbichler GR, Roy SH, Lehr S, Posch M, Kollmitzer J, Della Croce U (2003): Muscle Fatigue and Fatigue-Related Biomechanical Changes During a Cyclic Lifting Task. Spine (Phila Pa 1976) 28, 1810–1820

Boston JR, Rudy TE, Lieber SJ, Stacey BR (1995): Measuring treatment effects on repetitive lifting for patients with chronic low back pain: speed, style, and coordination. J Spinal Disord 8, 342–351

Boudreau S, Farina D, Kongstad L, Buus D, Redder J, Sverrisdóttir E, Falla D (2011): The relative timing of trunk muscle activation is retained in response to unanticipated postural-perturbations during acute low back pain. Exp Brain Res 210, 259–267

Bromm B, Chen AC (1995): Brain electrical source analysis of laser evoked potentials in response to painful trigeminal nerve stimulation. Electroencephalogr Clin Neurophysiol 95, 14–26

Bundesärztekammer, Kassenärztliche Bundesvereinigung: Nationale VersorgungsLeitlinie Nicht-spezifischer Kreuzschmerz – Langfassung, 2. Auflage. Version 1; 2017

Carroll LJ, Cassidy JD, Côté P (2004): Depression as a risk factor for onset of an episode of troublesome neck and low back pain. Pain 107, 134–139

Cassidy JD, Côté P, Carroll LJ, Kristman V (2005): Incidence and course of low back pain episodes in the general population. Spine (Phila Pa 1976) 30, 2817–2823

Cavanagh PR, Komi P V. (1979): Electromechanical delay in human skeletal muscle under concentric and eccentric contractions. Eur J Appl Physiol Occup Physiol 42, 159–163

Chiou SY, Shih YF, Chou LW, McGregor AH, Strutton PH (2014): Impaired neural drive in patients with low back pain. Eur J Pain (United Kingdom) 18, 794–802

Cholewicki J, Silfies SP, Shah RA, Greene HS, Reeves PN, Alvi K, Goldberg B (2005): Delayed trunk muscle reflex responses increase the risk of low back injuries. Spine (Phila Pa 1976) 30, 2614–2620

Cruccu G, Truini A (2006): Assessment of neuropathic pain. Neurol Sci 27, s288–s290

De Luca CJ (2002): Surface electromyography : detection and recording. DelSys Inc 10, 1–10

Deyo RA, Rainville J, Kent DL (1992): What can the history and physical examination tell us about low back pain? Ration Clin Exam 268, 760–765

Dubois JD, Piché M, Cantin V, Descarreaux M (2011): Effect of experimental low back pain on neuromuscular control of the trunk in healthy volunteers and patients with chronic low back pain. J Electromyogr Kinesiol 21, 774–781

Ellert U, Kurth B-M (2004): Methodische Betrachtungen zu den Summenscores des SF-36 anhand der erwachsenen bundesdeutschen Bev lkerung. Bundesgesundheitsblatt - Gesundheitsforsch - Gesundheitsschutz 47, 1027–1032

Esola MA, McClure PW, Fitzgerald GK, Siegler S (1996): Analysis of lumbar spine and hip motion during forward bending in subjects with and without a history of low back pain. Spine (Phila Pa 1976) 21, 71–8

Falla D, Farina D (2007): Periodic increases in force during sustained contraction reduce fatigue and facilitate spatial redistribution of trapezius muscle activity. Exp Brain Res 182, 99–107

Falla D, Farina D (2008): Non-uniform adaptation of motor unit discharge rates during sustained static contraction of the upper trapezius muscle. Exp Brain Res 191, 363–370

Falla D, Farina D, Graven-Nielsen T (2007a): Experimental muscle pain results in reorganization of coordination among trapezius muscle subdivisions during repetitive shoulder flexion. Exp Brain Res 178, 385–393

Falla D, Farina D, Dahl MK, Graven-Nielsen T (2007b): Muscle pain induces task-dependent changes in cervical agonist/antagonist activity. J Appl Physiol 102, 601–609

Falla D, Farina D, Graven-Nielsen T (2007c): Spatial dependency of trapezius muscle activity during repetitive shoulder flexion. J Electromyogr Kinesiol 17, 299–306

Falla D, Gizzi L, Tschapek M, Erlenwein J, Petzke F (2014): Reduced task-induced variations in the distribution of activity across back muscle regions in individuals with low back pain. Pain 155, 944–953

Farina D, Arendt-Nielsen L, Merletti R, Graven-Nielsen T (2004): Effect of experimental muscle pain on motor unit firing rate and conduction velocity. J Neurophysiol 91, 1250–1259

Farina D, Leclerc F, Arendt-Nielsen L, Buttelli O, Madeleine P (2008): The change in spatial distribution of upper trapezius muscle activity is correlated to contraction duration. J Electromyogr Kinesiol 18, 16–25

Gerhardt A, Eich W, Janke S, Leisner S, Treede R-D, Tesarz J (2016): Chronic widespread back pain is distinct from chronic local back pain. Clin J Pain 32, 568–579

Geyer S, Hemström O, Peter R, Vågerö D (2006): Education, income, and occupational class cannot be used interchangeably in social epidemiology. Empirical evidence against a common practice. J Epidemiol Community Health 60, 804–810

Giesecke T, Gracely RH, Clauw DJ, Nachemson A, Dück MH, Sabatowski R, Gerbershagen HJ, Williams DA, Petzke F (2006): Zentrale Schmerzverarbeitung bei chronischem Rückenschmerz. Der Schmerz 20, 411–417

Gilmore KL, Meyers JE (1983): Using surface electromyography in physiotherapy research. Aust J Physiother 29, 3–9

Gombatto SP, D'Arpa N, Landerholm S, Mateo C, O'Connor R, Tokunaga J, Tuttle LJ (2017): Differences in kinematics of the lumbar spine and lower extremities between people with and without low back pain during the down phase of a pick up task, an observational study. Musculoskelet Sci Pract 28, 25–31

Granata KP, Sanford AH (2000): Lumbar – pelvic coordination is influenced by lifting task parameters. Spine (Phila Pa 1976) 25, 1413–1418

Graven-Nielsen T, Arendt-Nielsen L (2008): Impact of clinical and experimental pain on muscle strength and activity. Curr Rheumatol Rep 10, 475–481

Graven-Nielsen T, Svensson P, Arendt-Nielsen L (1997a): Effects of experimental muscle pain on muscle activity and co-ordination during static and dynamic motor function. Electroencephalogr Clin Neurophysiol Mot Control 105, 156–164

Graven-Nielsen T, Arendt-Nielsen L, Svensson P, Jensen TS (1997b): Experimental muscle pain: A quantitative study of local and referred pain in humans following injection of hypertonic saline. J Musculoskelet Pain 5, 49–69

Greene HS, Cholewicki J, Galloway MT, Nguyen C V, Radebold A (2001): A history of low back injury is a risk factor for recurrent back injuries in varsity athletes. Am J Sports Med 29, 795–800

Handwerker HO, Kobal G (1993): Psychophysiology of experimentally induced pain. Physiol Rev 73, 639–71

Hashmi JA, Baliki MN, Huang L, Baria AT, Torbey S, Hermann KM, Schnitzer TJ, Apkarian AV (2013): Shape shifting pain: Chronification of back pain shifts brain representation from nociceptive to emotional circuits. Brain 136, 2751–2768

Hernandez A, Gross K, Gombatto S (2017): Differences in lumbar spine and lower extremity kinematics during a step down functional task in people with and people without low back pain. Clin Biomech 47, 46–52

Hill A V. (1950): The series elastic componet of muscle. Proc R Soc London Ser B - Biol Sci 137, 273–280

Hitt R, Bunn JY (2013): Stiffening strategy to maintain upright posture. J Electromyogr Kinesiol 22, 13-20

Hodges P (2001): Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. Exp Brain Res 141, 261–266

Hodges PW, Richardson CA (1999): Altered trunk muscle recruitment in people with low back pain with upper limb movement at different speeds. Arch Phys Med Rehabil 80, 1005–1012

Hodges PW, Tucker K (2011): Moving differently in pain: A new theory to explain the adaptation to pain. Pain 152, S90–S98

Hodges PW, Smeets RJ (2015): Interaction between pain, movement, and physical activity. Clin J Pain 31, 97–107

Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC (2003): Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res 151, 262–271

Hodges PW, Coppieters MW, MacDonald D, Cholewicki J (2013): New insight into motor adaptation to pain revealed by a combination of modelling and empirical approaches. Eur J Pain 17, 1138–1146

Jacobs J V, Henry SM, Nagle KJ (2009): People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments. Behav Neurosci 123, 455–458

Jacobs J V, Henry SM, Jones SL, Hitt JR, Bunn JY (2011): A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance. J Neurophysiol 106, 2506–2514

Johansson H, Sojka P (1991): Pathophysiological mechanisms involved in genesis and spread of muscular tension in occupational muscle pain and in chronic musculoskeletal pain syndromes: A hypothesis. Med Hypotheses 35, 196–203

Jones MD, Taylor JL, Booth J, Barry BK (2016): Exploring the mechanisms of exercise-induced hypoalgesia using somatosensory and laser evoked potentials. Front Physiol 7

Jones SL, Hitt JR, DeSarno MJ, Henry SM (2012a): Individuals with non-specific low back pain in an active episode demonstrate temporally altered torque responses and direction-specific enhanced muscle activity following unexpected balance perturbations. Exp Brain Res 221, 413–426

Jones SL, Henry SM, Raasch CC, Hitt JR, Bunn JY (2012b): Individuals with non-specific low back pain use a trunk stiffening strategy to maintain upright posture. J Electromyogr Kinesiol 22, 13–20

Jubany J, Danneels L, Angulo-Barroso R (2017): The influence of fatigue and chronic low back pain on muscle recruitment patterns following an unexpected external perturbation. BMC Musculoskelet Disord 18, 161

Kerr MS, Frank JW, Shannon HS, Norman RW, Wells RP, Neumann WP, Bombardier C (2001): Biomechanical and psychosocial risk factors for low back pain at work. Am J Public Health 91, 1069–1075

Kim M, Yi C, Kwon O, Cho S, Cynn H, Kim Y, Hwang S, Choi B, Hong J, Jung D (2013a): Comparison of Lumbopelvic Rhythm and Flexion-Relaxation Response Between 2 Different Low Back Pain Subtypes. Spine (Phila Pa 1976) 38, 1260–1267

Kim M, Yoo W, Choi B (2013b): Differences between two subgroups of low back pain patients in lumbopelvic rotation and symmetry in the erector spinae and hamstring muscles during trunk flexion when standing. J Electromyogr Kinesiol 23, 387–393

Koes BW, van Tulder MW, Thomas S (2006): Diagnosis and treatment of low back pain. BMJ 332, 1430–1434

Konrad P: Emg-Fibel. 2011

Kosek E, Lundberg L (2003): Segmental and plurisegmental modulation of pressure pain thresholds during static muscle contractions in healthy individuals. Eur J Pain 7, 251–258

Kramer M, Ebert V, Kinzl L, Dehner C, Elbel M, Hartwig E (2005): Surface electromyography of the paravertebral muscles in patients with chronic low back pain.

Arch Phys Med Rehabil 86, 31–36

Kubo K, Akima H, Kouzaki M, Ito M, Kawakami Y, Kanehisa H, Fukunaga T (2000): Changes in the elastic properties of tendon structures following 20 days bed-rest in humans. Eur J Appl Physiol 83, 463–468

Lamoth CJC, Meijer OG, Daffertshofer A, Wuisman PIJM, Beek PJ (2006): Effects of chronic low back pain on trunk coordination and back muscle activity during walking: changes in motor control. Eur Spine J 15, 23–40

Lee JK, Desmoulin GT, Khan AH, Park EJ: A portable inertial sensing-based spinal motion measurement system for low back pain assessment; in: 2011 Annual International Conference of the IEEE Engineering in Medicine and Biology Society; IEEE 2011, 4737–4740

Lethem J, Slade PD, Troup JDG, Bentley G (1983): Outline of a fear-avoidance model of exaggerated pain perception—I. Behav Res Ther 21, 401–408

Lu D, Le P, Davidson B, Bing HZ, Lu Y, Patel V, Solomonow M (2008): Frequency of cyclic lumbar loading is a risk factor for cumulative trauma disorder. Muscle and Nerve 38, 867–874

Lund JP, Donga R, Widmer CG, Stohler CS (1991): The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 69, 683–694

Madeleine P, Leclerc F, Arendt-Nielsen L, Ravier P, Farina D (2006): Experimental muscle pain changes the spatial distribution of upper trapezius muscle activity during sustained contraction. Clin Neurophysiol 117, 2436–2445

Magnusson ML, Aleksiev A, Wilder DG, Pope MH, Spratt K, Lee SH, Goel VK, Weinstein JN (1996): European Spine Society ?The Acromed Prize for Spinal Research 1995 Unexpected load and asymmetric posture as etiologic factors in low back pain. Eur Spine J 5, 23–35

Marras WS, Parnianpour M, Ferguson SA, Kim J-Y, Crowell RR, Bose S, Simon SR (1995): The classification of anatomic- and symptom-based low back disorders using motion measure models. Spine (Phila Pa 1976) 20, 2531–2546

Mathiassen SE, Möller T, Forsman M (2003): Variability in mechanical exposure within and between individuals performing a highly constrained industrial work task. Ergonomics 46, 800–824

Merletti; R, Parker P: Electromyography: Physiology, engineering and noninvasive Applications; in: Climate Change 2013 - The Physical Science Basis, volume 53; ed. by Intergovernmental Panel on Climate Change; Cambridge University Press, Cambridge 2013, 1–30

Merletti R, Farina D: Surface Electromyography : physiology, engineering, and applications. John Wiley & Sons, Inc., Hoboken, New Jersey 2016a

Merletti R, Farina D: Surface electromyography: physiology, engineering and applications. John Wiley & Sons 2016b

Merletti R, Knaflitz M, De Luca CJ (1990): Myoelectric manifestations of fatigue in voluntary and electrically elicited contractions. J Appl Physiol 69, 1810–1820

Merletti R, Rainoldi A, Farina D (2001): Surface electromyography for noninvasive characterization of muscle. Exerc Sport Sci Rev 29, 20–25

Merletti R, Holobar A, Farina D (2008): Two dimensional high density surface EMG (HD-

EMG) technology and applications. 1–4

Misra G, Paris TA, Archer DB, Coombes SA (2014): Dose-Response Effect of Isometric Force Production on the Perception of Pain. PLoS One 9, e88105

Mitchell K, Porter M, Anderson L, Phillips C, Arceo G, Montz B, Levy S, Gombatto SP (2017): Differences in lumbar spine and lower extremity kinematics in people with and without low back pain during a step-up task: a cross-sectional study. BMC Musculoskelet Disord 18, 369

Mitnitski AB, Yahia LH, Newman NM, Gracovetsky SA, Feldman AG (1998): Coordination between the lumbar spine lordosis and trunk angle during weight lifting. Clin Biomech 13, 121–127

Moore BD, Drouin J, Gansneder BM, Shultz SJ (2002): The differential effects of fatigue on reflex response timing and amplitude in males and females. J Electromyogr Kinesiol 12, 351–360

Moseley GL, Hodges PW (2005): Are the changes in postural control associated with low back pain caused by pain interference? Clin J Pain 21, 323–329

Müller G (2001): Diagnostik des Rückenschmerzes. 435–441

Mumenthaler M, Mattle H: Kurzlehrbuch Neurologie, 1. Thieme 2006

Naugle KM, Fillingim RB, Riley JL (2012): A Meta-Analytic Review of the Hypoalgesic Effects of Exercise. J Pain 13, 1139–1150

Nilges P, Essau C (2015): Die Depressions-Angst-Stress-Skalen. Der Schmerz 29, 649–657

Nöllenheidt C, Brenscheidt SArbeitswelt im Wandel. 2015

Ostelo RWJG, Deyo RA, Stratford P, Waddell G, Croft P, Von Korff M, Bouter LM, de Vet HC (2008): Interpreting Change Scores for Pain and Functional Status in Low Back Pain. Spine (Phila Pa 1976) 33, 90–94

Paquet N, Malouin F, Richards CL (1994): Hip-spine movement interaction and muscle activation patterns during sagittal trunk movements in low back pain patients. Spine (Phila Pa 1976) 19, 596–603

Pavlaković G, Petzke F (2010): The Role of Quantitative Sensory Testing in the Evaluation of Musculoskeletal Pain Conditions. Curr Rheumatol Rep 12, 455–461

Pengel LHM (2003): Acute low back pain: systematic review of its prognosis. BMJ 327, 323–0

Pfingsten M (2004): Angstvermeidungsüberzeugungen bei Rückenschmerzen Gütekriterien und prognostische Relevanz des FABQ. Schmerz 18, 17–27

Plamenig P: Zahlen, Daten & Fakten; in: Qualitäts- und Risikomanagement im Gesundheitswesen; 2018, 75–88

Potvin JR, Bent LR (1997): A validation of techniques using surface EMG signals from dynamic contractions to quantify muscle fatigue during repetitive tasks. J Electromyogr Kinesiol 7, 131–139

Radebold A, Cholewicki J, Panjabi MM, Patel TC (2000): Muscle response pattern to sudden trunk loading in healthy individuals and in patients with chronic low back pain. Spine (Phila Pa 1976) 25, 947–54

Raspe H (2012): Gesundheitsberichterstattung des Bundes - Rückenschmerzen. Robert Koch-Institut 1–36

Reddy KS kumar, Naidu MUR, Rani PU, Rao TRK (2012): Human experimental pain models: A review of standardized methods in drug development. J Res Med Sci 17, 587–595

Rojas-Martínez M, Mañanas MA, Alonso JF (2012): High-density surface EMG maps from upper-arm and forearm muscles. J Neuroeng Rehabil 9, 85

Rolke R, Campbell KA, Magerl W, Treede R-D (2005): Deep pain thresholds in the distal limbs of healthy human subjects. Eur J Pain 9, 39–48

Rolke R, Magerl W, Campbell KA, Schalber C, Caspari S, Birklein F, Treede RD (2006a): Quantitative sensory testing: A comprehensive protocol for clinical trials. Eur J Pain 10, 77–88

Rolke R, Baron R, Maier C, Tölle TR, Treede RD, Beyer A, Binder A, Birbaumer N, Birklein F, Bötefür IC, et al. (2006b): Quantitative sensory testing in the German research network on neuropathic pain (DFNS): Standardized protocol and reference values. Pain 123, 231–243

Ross GB, Mavor M, Brown SHM, Graham RB (2015): The effects of experimentally induced low back pain on spine rotational stiffness and local dynamic stability. Ann Biomed Eng 43, 2120–2130

Sahrmann S, Azevedo DC, Dillen L Van (2017): Diagnosis and treatment of movement system impairment syndromes. Brazilian J Phys Ther 21, 391–399

Sánchez-Zuriaga D, López-Pascual J, Garrido-Jaén D, de Moya MFP, Prat-Pastor J (2011): Reliability and Validity of a New Objective Tool for Low Back Pain Functional Assessment. Spine (Phila Pa 1976) 36, 1279–1288

Scholtes SA, Gombatto SP, Van Dillen LR (2009): Differences in lumbopelvic motion between people with and people without low back pain during two lower limb movement tests. Clin Biomech 24, 7–12

Shum GLK, Crosbie J, Lee RYW (2005): Effect of Low Back Pain on the Kinematics and Joint Coordination of the Lumbar Spine and Hip During Sit-to-Stand and Stand-to-Sit. Spine (Phila Pa 1976) 30, 1998–2004

Shum GLK, Crosbie J, Lee RYW (2007): Movement coordination of the lumbar spine and hip during a picking up activity in low back pain subjects. Eur Spine J 16, 749–758

Tanii K, Masuda T (1985): A kinesiologic study of erectores spinae activity during trunk flexion and extension. Ergonomics 28, 883–893

Thomas JS, France CR, Sha D, Vander Wiele N, Moenter S, Swank K (2007): The effect of chronic low back pain on trunk muscle activations in target reaching movements with various loads. Spine (Phila Pa 1976) 32, E801-8

Truini A, Romaniello A, Galeotti F, Iannetti GD, Cruccu G (2004): Laser evoked potentials for assessing sensory neuropathy in human patients. Neurosci Lett 361, 25–28

Tucker K, Falla D, Graven-Nielsen T, Farina D (2009): Electromyographic mapping of the erector spinae muscle with varying load and during sustained contraction. J Electromyogr Kinesiol 19, 373–379

Vaegter HB, Hoeger Bement M, Madsen AB, Fridriksson J, Dasa M, Graven-Nielsen T (2017): Exercise increases pressure pain tolerance but not pressure and heat pain thresholds in healthy young men. Eur J Pain (United Kingdom) 21, 73–81

Valeriani M, De Tommaso M, Restuccia D, Le Pera D, Guido M, Iannetti GD, Libro G, Truini A, Di Trapani G, Puca F, et al. (2003): Reduced habituation to experimental pain

in migraine patients: A CO2 laser evoked potential study. Pain 105, 57–64

van den Hoorn W, Hodges PW, van Dieen JH, Hug F (2015): Effect of acute noxious stimulation to the leg or back on muscle synergies during walking. J Neurophysiol 113, 244–254

van der Hulst M, Vollenbroek-Hutten MM, Rietman JS, Schaake L, Groothuis-Oudshoorn KG, Hermens HJ (2010): Back muscle activation patterns in chronic low back pain during walking: a 'guarding' hypothesis. Clin J Pain 26, 30–37

van Dieen JH, Toussaint HM, Thissen C, van de Ven A (1993): Spectral analysis of erector spinae EMG during intermittent isometric fatiguing exercise. Ergonomics 36, 407–414

Vasseljen O, Woodhouse A, Bjørngaard JH, Leivseth L (2013): Natural course of acute neck and low back pain in the general population: The HUNT study. Pain 154, 1237–1244

Villemure C, Bushnell MC (2002): Cognitive modulation of pain: How do attention and emotion influence pain processing? Pain 95, 195–199

Vos T, Flaxman AD, Naghavi M, Lozano R, Michaud C, Ezzati M, Shibuya K, Salomon JA, Abdalla S, Aboyans V, et al. (2012): Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990-2010: A systematic analysis for the Global Burden of Disease Study 2010. Lancet 380, 2163–2196

Willer JC, Roby A, Bars D Le (1984): Psychophysical and electrophysiological approaches to the pain-relieving effects of heterotopic nociceptive stimuli. Brain 107, 1095–1112

Yavuz ŞU, Şendemir-Ürkmez A, Türker KS (2010): Effect of gender, age, fatigue and contraction level on electromechanical delay. Clin Neurophysiol 121, 1700–1706

Yavuz UŞ, Negro F, Falla D, Farina D (2015): Experimental muscle pain increases variability of neural drive to muscle and decreases motor unit coherence in tremor frequency band. J Neurophysiol 114, 1041–1047

Zhou S, Lawson DL, Morrison WE, Fairweather I (1995): Electromechanical delay in isometric muscle contractions evoked by voluntary, reflex and electrical stimulation. Eur J Appl Physiol Occup Physiol 70, 138–145

Zhou S, McKenna MJ, Lawson DL, Morrison WE, Fairweather I (1996a): Effects of fatigue and sprint training on electromechanical delay of knee extensor muscles. Eur J Appl Physiol Occup Physiol 72, 410–6

Zhou S, McKenna MJ, Lawson DL, Morrison WE, Fairweather I (1996b): Effects of fatigue and sprint training on electromechanical delay of knee extensor muscles. Eur J Appl Physiol Occup Physiol 72, 410–416

Zwarts MJ, Stegeman DF (2003): Multichannel surface EMG: basic aspects and clinical utility. Muscle Nerve 28, 1–17

Acknowledgements

At this point I would like to thank all the amazing people who have supported and encouraged me on my way to the final thesis.

First of all, I thank my doctoral supervisor Professor Petzke and Doctor Erlenwein for always standing by with advice and action.

Secondly, I would like to thank my outstanding supporter Doctor Utku Yavuz who did not only help me with the statistics and analysation but also has always encouraged and supported me in any occasion.

Curriculum vitae

My name is Franziska Butterwegge. I was born on 10th July, 1994 in Kassel. In Kassel I completed Primary school from 2000-2004 and secondary school from 2004-2013. My specialised courses were English and Chemistry. In winter 2013/14 I started studying medicine at the University of Göttingen. I completed the preliminary medical examination in September 2015. In August 2016 I started with the experiments for my Doctor Thesis, which I performed under the supervision of the Pain Clinic of the Universitätsmedizin Göttingen. In spring 2019 I completed my second state examination (M2). During my internship I worked among others in the Anaesthesiology of the Unfallkrankenhaus Salzburg und spent the half of a Tertial in the Inner Medicine of the Kantonsspital Luzern. In June 2020 I completed the final medical examination (M3). In October of the same year I started working as a doctor in the department of Inner Medicine in the Elisabethkrankenhaus Kassel.