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Introduction
The aim of this paper is to present a model for the assessment and treatment of musculoskeletal dysfunction. The model is based on the role of proprioception in sensorimotor incongruence and suggests how a number of symptoms, including but not limited to pain, arise and can be treated. The model builds on Moseley's "Imprecision hypothesis of chronic pain" (Moseley 2015). We have expanded on Moseley's theory to include acute pain, non-painful motor dysfunction and sensory dysfunction.

In recent years substantial amounts of data on how nociception interacts with proprioception has been gathered in studies of acute and chronic musculoskeletal pain disorders throughout the body (Baliki 2015, Bank 2013a, Bank 2013b, Bank 2013c, Bank 2014, Bray 2011, Daenen 2012, Gilpin 2015, Lewis 2012, Lotze 2015, Moseley 2012b, Tsay 2015, Wand 2013, Röijezon 2015). What is becoming clear is that there seems to be a mismatch between motor output and sensory feedback that cause pain, motor inhibition and sensory disturbances (Bank 2013c, Bank 2014, Bray 2011, Daenen 2012, Flor 1997, Moseley 2012b, Moseley 2015, Röijezon 2015, Tsay 2015, Wand 2013).

Proprioception
Sufficient proprioception includes three stages of information handling: The first stage is detection and transmission of afferent inputs. The second stage is integration of information from peripheral and central sources and the third stage is interpretation of the integrated data in relation to a body schema (Bank 2013c, Longo 2010, Proske 2012). Body schema is described as: "the actual performance of the body in its environment, which involves an integration of proprioceptive, vestibular, somatosensory and visual input from the periphery that interrelate with motor system" (Daenen 2012). The motor-control system normally works below our conscious level, and we only become aware of it when incongruence in the system occurs. When incongruence occurs, the person is vulnerable to more sensory disturbances, which may further exacerbate the problem (McCabe 2007a) and may lead an increased risk of injury and progression of secondary dysfunctions (Röijezon 2015).

For example in patients with Chronic Regional Pain Syndrome (CRPS), the body schema has not been updated to the changed status of the body part. The maximum range of motion of the joints is still considered the same as it was before developing CRPS and does not accurately match the actual status, which creates a mismatch (Bank 2013c, Gay 2007, Lewis 2012, McCabe 2007a). Similar mismatches also occur in patients with fibromyalgia (McCabe 2007b), whiplash associated disorder (Daenen 2012), osteoarthritis (Gilpin 2015), chronic low back pain (Bray 2011, Wand 2013). Furthermore, healthy subjects experience sensory disturbances when exposed to incongruent proprioceptive input (Blankenburg 2006, Daenen 2010, McCabe 2005, Moseley 2006).

A potentially dangerous situation
In situations that are potentially dangerous, nociceptive and proprioceptive information is sent to the brain (Baliki 2015, Liang 2013, Lotze 2015, Moseley 2015, Tsay 2015). When that information arrives at the brain, the brain needs to answer the very important question: "How dangerous is this?" (Moseley 2007). To be able to answer in the best way, the brain values
every bit of information it has access to, such as: other sensory input, previous experiences, knowledge, expectations about consequences, etc. (Baliki 2015, Lotze 2015, Moseley 2007, Moseley 2015). When the brain has evaluated the situation, the response deemed most suitable is chosen (Baliki 2015, Lotze 2015, Moseley 2015). If the evaluation concludes that the situation is dangerous, pain often occurs. Since pain is erroneously interpreted as a measure of tissue damage, we assume that the tissues have been damaged. If pain persists, we automatically assume that tissue damage remains, which is often wrong (Baliki 2015, Lotze 2015, Moseley 2007, Moseley 2015). If the evaluation concludes that the situation is dangerous, the accompanying proprioceptive information (body position, type of movement, direction of the adverse force, etc.) is tagged as a potential danger for future reference (Baliki 2015, Moseley 2015, Tsay 2015).

**Motor inhibition and sensorimotor incongruence**

Nociception can inhibit motor signals (Bank 2013a, Graven-Nielsen 2008, Hodges 2011, Lund 1991, Nijs 2012). The inhibition often remains, most likely because the accompanying proprioceptive information has been tagged as a potential danger (Tsay 2015). Examples of this can be seen in patients with chronic pain, where often body perception disturbances are seen (Frettö 2006, Hirakawa 2014, Lewis 2007, Moseley 2012a, Punt 2013). Current evidence indicates that a decrease of pain alone is not sufficient to normalize the neuromuscular changes in patients with chronic pain (Falla 2006, Falla 2007, Falla 2008, MacDonald 2010).

For the inhibition to be cancelled the brain needs to receive new proprioceptive information that neutralises the stored information to remove the "danger tag" (McCabe 2005, Moseley 2005, Moseley 2012b, Pleger 2005). Research suggests that the information should be sent from the area where the nociceptive input originated and includes the same level of innervation (McCabe 2007a, Moseley 2005). When the new information arrives at the brain and is analysed in the same way as earlier, the conclusion this time is that it is not dangerous. Changes are seen immediately since the danger is now deemed to be over, and a new response is sent out. In addition to the cancellation of the motor inhibition, sensory disturbances may be restored when the proprioception is normalized (Daenen 2012, Moseley 2012b, Tsay 2015, Wand 2013).

**Restoring joint motion and normalizing proprioception**

One factor that is essential in normalizing proprioception is to restore joint motion at the level of innervation, since restored joint motion improves proprioception (Cuomo 2005). One way of restoring joint motion is manipulation/mobilisation, which is suitable since it can have an immediate and significantly beneficial effect on proprioceptive feedback (Clark 2015) and result in plastic changes from sensorimotor integration (Haavik 2012). Acute decreases in pain following manipulation may allow more active participation in exercise and functional retraining earlier in the rehabilitation process (Wassinger 2015). Since soft tissues also are richly innervated with mechanoreceptors, some soft tissue techniques may also be useful in normalizing proprioception (Clark 2015). Another modality that can be useful in improving proprioception is vibration (Beinert 2014, Beinert 2015).

**Assessment and treatment**

In addition to restoring joint motion, further attention is often needed to ensure normal proprioception. Considering that most manipulation/mobilisation techniques utilize a force vector from posterior to anterior, patients who had an injury where the direction of the adverse force were from posterior to anterior, may not have the proprioceptive danger tag removed, even if the joint motion is restored.
There are some manipulation/mobilisation techniques that utilize a force vector from anterior to posterior (Jackson 2005, Maitland 2005, Minardi 2006) which could be used when the direction of the adverse force were from posterior to anterior. It is very important that the techniques are pain free, otherwise the nociceptive input will prevent a normalizing of proprioception and removal of motor inhibition, due to the mechanisms discussed above. To assess which direction stimuli should be applied, a combination of stimuli and motor tests can be used.

**Case study**
The patient came to the clinic six weeks after an incident that happened when he was performing seated rowing (weight training) at the gym. He had overextended his low back and immediately experienced an acute stabbing pain in the right side of the low back. At the clinic he presented with pain in the right side of the lumbar spine and weakness in lumbar extension during sitting.

Palpation revealed tenderness and reduced springing at L1 on the right side. Myotome testing revealed a markedly weak hip flexion (L1-L2) on the right side. After manipulation of L1 on the right side the pain was reduced by 90%. No change was seen on the myotome weakness. After stimulation of the L1 segment on the right hand side on the anterior side of the trunk the myotome test normalized and the weakness in lumbar extension in sitting was slightly better.

On the return visit one week later the low back pain was gone. The patient still experienced weakness in lumbar extension in sitting. Myotome testing was negative. After stimulation of the L1 segment on the right hand side on the anterior side of the trunk, with simultaneous lumbar extension in sitting the strength was restored. During the next visit the patient was pain free and had full strength in all movements.

The interpretation, according to the presented model, is that at the time of injury, proprioceptive information that L1 was subjected to an adverse force from posterior to anterior on the right side and that the joint lost mobility, was sent to the brain together with the nociceptive information. The manipulation restored the mobility of L1, but there was still a danger tag that needed to be addressed in order to turn off the motor inhibition.

The stimulation of L1 on the right hand side on the anterior side of the trunk supplied some proprioceptive information but not enough to neutralize the danger tag. The stimulation of L1 on the right hand side on the anterior side of the trunk with simultaneous lumbar extension in sitting provided the proper proprioceptive information to neutralize the danger tag. A plausible reason for this is that the stimulation was performed during the same movement as the injury occurred.

**Summary**
Moseley's "Imprecision hypothesis of chronic pain" posits: "pain as a conditioned response to the multisensory and meaningful events that routinely coincide with, or preempt, nociceptive input. Moreover, imprecise encoding of those multisensory and meaningful events leads to overgeneralization of the response, such that an adaptive and protective process becomes maladaptive, distressing, and disabling chronic pain" (Moseley 2015).

We have presented a model that also includes acute pain, non-painful motor dysfunction and sensory dysfunction (see picture 1 below). The model is based on the role of proprioception in sensorimotor incongruence and suggests how a number of symptoms,
not only pain, arise and can be treated. Evidence strongly suggests there is a need for rehabilitation strategies that target sensorimotor incongruence to improve function (Aman 2015, Bank 2013c, Bank 2014, Gay 2007, Gilpin 2015, Luomajoki 2011, Mace 2008, McCabe 2007a, Moseley 2005, Moseley 2012b, Pleger 2005, Röijezon 2015, Wand 2011). More studies are required, and if the model is correct, it will open up new treatment possibilities of pain, non-painful motor dysfunctions and sensory dysfunctions.

Referenser


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Picture 1. A = Injury, B = Treatment


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