The traditional mechanistic paradigm in the teaching and practice of manual therapy : Time for a reality check.

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Abstract

The actual mainstream paradigm for manual physical therapy in the treatment of pain mostly relies on a biomechanical model. At its core, the premises of the model are that biomechanical dysfunctions in the form of hypo or hypermobility, poor postural behavior and sub-optimal motor control are causal and/or perpetuating factors for many painful musculoskeletal (MSK) conditions. In this model, it is believed that manual therapy effectively decreases or eliminates pain, at least in great part, by «correcting» the dysfunctions via a lasting mechanical effect on different connective tissues and by changing the person's postural habits. While this model has been around for years and has some proof for its effectiveness, there is either a lack of evidence or evidence against many of the assertions it makes. Meanwhile, evidence in favor of an alternate neurophysiological model has been emerging in the past decade. In the following discussion, the author will present what he considers the many shortcomings of the biomechanical model (BMM) and will briefly introduce an emerging neurophysiological model (NPM) that could better explain the effects manual therapy has on pain.

Manual therapy is frequently utilised in the treatment of several musculoskeletal (MSK) painful conditions. There is a growing body of evidence for its effectiveness in the treatment of numerous such conditions[1,2,3,4]. Although the effect size in many studies is reported to be modest[1,4,5] there is often a significantly positive effect on pain and on other outcome measures in many trials. Even though proven effective, manual therapies have not yet been consistently proven substantially superior to other form of conservative care such as NSAID and general physician care[1,4,6].

There is no official consensus as to the mechanisms through which manual therapies exert their effects on painful conditions. The most frequently proposed mechanism is that manual therapies achieve such pain relieving effects through a mechanical cascade of events. The rationale behind most of this logic originates from the notion that mechanical dysfunctions of different forms are causal and/or perpetuating factors in painful conditions. Such biomechanical dysfunctions are then addressed by different manual therapy interventions in hope to eliminate or attenuate these adverse biomechanical faults. The alleviation of the pain would be the result of the correction of such dysfunctions. This rationale is largely hypothetical as the relation between the positive outcomes in the studies has not yet convincingly been linked to lasting biomechanical changes[8-14,38,51]. On the other hand, there has been in the past decade an emergence of evidence for a neurophysiological mechanism [7,15-20,24,25,51] for manual therapy in the treatment of pain. The evidence is growing, but while the exact mechanism through which the neurophysiologic effects are achieved is not completely known, there is evidence for the implication of both the peripheral and central nervous systems for combined effects that are possibly both specific and non-specific to the applied modalities.

Despite a growing body of evidence and scientific knowledge against the BMM of manual therapy and the emerging and convincing evidences for a NPM of effectiveness, there seems to be a lot of hesitation for a paradigm change in both the clinical reasoning associated with, and subsequent application of, manual therapy by clinicians. Not only that, but there also seem to be a lag between what is taught in manual therapy program[21] and the actual paradigm shift suggested by the scientific literature. Several reasons might explain such reluctance

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to change the model upon which clinical reasoning is made in manual therapy but it is astounding that there does not even seem to be an actual open debate on the question among clinicians. The idea of a neurophysiological effect with manual therapy is briefly discussed in the many courses leading to the Canadian certification in manual therapy and the clinical reasoning still taught remains dominated by mechanical notions on segmental dysfunctions and the subsequent need for their correction[21].

In the present text, the author will succinctly present his understanding of the current state of the literature about both the BMM and NPM. Subsequently, he will suggest adjustments that should be made in the teaching of manual physical therapy in the context of the paradigm shift suggested by many in the literature[7,22,23,24,26,27,39,51]

The questionable biomechanical model of manual therapy

To summarize this model, it is proposed that biomechanical dysfunctions characterized by a combinations of segmental joint hypo or hypermobility , suboptimal postures, muscle weakness and/or poor muscle control play a significant role in the emergence of painful MSK conditions by putting too much strain on different tissues which would ultimately lead local and/or distant tissues to sustain damage or to function sub optimally. The end result of this dysfunctional state would then often be pain. The role of manual therapy in such a model is to find these aforementioned dysfunctions and treat them via manual mobilisations or manipulations, stabilisation exercises and postural corrections among others. It is proposed that the manual mobilisations or manipulations will restore the joint play by restoring tissues optimal lengths or by reducing a fixation or sub-luxation and thus, restoring optimal joint function which, in turn, will lead to the resolution of the dysfunction and thus, the pain. The neurophysiological effects are now progressively being added to this model as some type of bonus to the primarily sought biomechanical effects. While very seductive, this model has several shortcomings.

The first one is that the findings on which the treatments are based largely rely on a motion palpation and observational exam that is greatly lacking in validity and reliability. Motion palpation tests, positional faults assessments, clinical postural assessments and many specific tests all have been found to have, at best, poor to fair reliability [28-37]. This casts serious doubt on the validity of the findings and the subsequent conclusions that are drawn from such an evaluation. In light of this, the value of the treatments originating from this kind of evaluation is, at least in part, questionable. Then there is the issue that to date, the literature has not been able to consistently demonstrate a lasting mechanical effect secondary to the application of manual therapy. Most observed effects seem to only be transient [8-9-10-11-12-13-14,38]. A transient effect could possibly be the result of creep deformation of the targeted tissues. It is demonstrated that creep elongation is transient and that the elongated tissues will return to their normal pre-treatment lengths after a certain time interval has elapsed[41]. Such transient effects are not so surprising when one looks at prior biologic plausibility. The forces used in manual therapy have been demonstrated to lack the necessary magnitude to provoke plastic changes on the targeted tissues[14,40,41] for several reasons. These reasons include, among others, the dissipation of the applied forces before it reaches the targeted tissue and the high forces required for plastic deformation to occur in mature connective tissues compared with the relatively low forces employed in the application of manual therapy. Certainly, sustainable changes may be possible via a gene expression mechanism if one is willing to undergo very frequent treatments and exercise on a daily basis over a long period of time[42-45]. Even then, this regimen would have to be sustained permanently for the changes to last. A short 4 week abstinence from the regimen could reverse all the changes[46].

On the basis of this information alone, it is questionable that the positive outcome observed following a course of a dozen manual therapy sessions (and often less) are the result of structural changes (tissue length or position) that would have «corrected» biomechanical dysfunctions. If the dysfunctions the therapist was aiming to correct were really the cause of the problem and the intervention only had a transient effect on them, then why would there be a lasting pain relief? One answer could be that the mechanical theory may be mostly wrong.

The BMM also proposes that the intervention must be highly tailored to the findings of the motion palpation assessment to be effective. If this was true, manual treatments applied specifically to the segment considered dysfunctional, both in terms of level and direction, would be more effective than manual therapy applied on random levels and in a random direction. There are few studies designed specifically for comparing this, but the few that are seem to reveal that manual therapy tailored on the motion palpation exam's findings are no more effective than random application of manual therapy in the area where the pain is[47,48,49,86]. Certainly, there are studies that tell us that some subgroups will respond better to tailored treatments. For instance a subgroup of acute low back pain (LBP) patients will respond favourably to a non-specific lumbar manipulation[3]. And patients with directional movement preferences will respond more favourably to treatments in these preferred directions[50]. Yet, patient's allocation to the right subgroup can be done without a thorough lumbar biomechanics exam in both these cases and the treatment applied does not have to be segment specific to be effective. In fact, in both these cases, subgroup allocation cues have little to do with the traditional findings of a specific manual palpation examination. The subgrouping issue is often brought up by the many proponents of the BMM of manual therapy to account for the small effect sizes reported in the literature. Although this argument has some value, it is interesting to note that the emerging subgroup classification for LBP treatments is not based on precise lumbar biomechanics and is not the result of the traditional biomechanical dysfunction reasoning. So, if past research for non-specific LBP (NSLBP) had subgrouped patients in groups concordant with the BMM, chances are that the groups formed would not be like the ones that were shown to have a significant impact on outcome measures. It could then be argued that the studies outcomes would not have been much different. Most likely, NSLBP is a heterogeneous condition but the search for the right subgroups remains in part elusive and the known demonstrated subgroups are not based on the traditional manual therapy's clinical reasoning. A good article by Wand and O'Connell[23] discusses the subgrouping issues in a brilliant way and proposes alternative views on this subject that certainly questions many of the traditional assumptions of the BMM for LBP.

Another note on the need of highly tailored and specific treatments is the fact that if such an assertion is true, more experienced and better skilled therapists should get better outcomes because of more precise treatments. So far, the evidence in the literature is that there is no statistically or clinically relevant difference between the outcomes obtained by inexperienced and uncertified therapist and those of the more experienced and certified ones[89].

Assumptions of the mechanical model

Another issue with the BMM revolves around what its proponents consider being causal factors in the genesis of painful conditions. Among these factors it is proposed that occupational postures or postural habits are important contributing factors to painful conditions. Another example is the case of LBP and degenerative disc disease (DDD). It is proposed that occupational activities, repetitive flexions, segmental hypomobility and other motion palpation findings are all important contributors to DDD and its severity. Both the examples are discussed below.

Posture and pain

The assumption that posture behaviours are contributing to pain is often made because some postures would increase the load on specific tissues and thus cause tissue damage and/or pain. Despite seemingly intuitive, this is a theory that is largely unsubstantiated. The body of literature on the subject is equivocal as to the correlation between posture and pain. Some studies reveal a significant correlation between both[54,55,57,58,59,61] while others do not[53,56,57,60]. Most of the studies that denoted a correlation found only a small difference in the postures of the people with pain. It is questionable that these small postural changes are significant enough to contribute to tissue damage or pain[61] let alone being reliably detected by a routine observational clinical exam. But the most important thing to remember here is that correlation does not equal causation. The relationship between pain and posture is not necessarily causal and the adoption of a different posture could just as easily be caused by the pain or the fear of experiencing it. There is some evidence in the literature to support that[62,63,64] as it was shown that postural changes happen after the admini-

stration of a painful stimulation or injection. In addition to that, different but yet similar cervical flexor strengthening exercises have been demonstrated to be of equal efficacy for pain relief even if one of the exercise regimens did not alter neck postures[52]. So the pain relief doesn't appear to be related to significant postural changes. The debate is still open on this regard but the role posture plays in the genesis of painful conditions might not be so important after all and, even if it were so, the manual interventions aiming at changing postures are most likely unable to do so by changing tissue length.

DDD and genetics

A recent long term study on twins has brilliantly demonstrated that almost up to 70% of the variance of DDD could be caused by genetic factors[65] and that only a small percentage (<10%) of the remaining 30% was influenced by factors like segmental mobility, occupational activities and other physical factors. It is also demonstrated that the correlation between degenerative changes on the MRI and X-rays is either unrelated with pain or only correlated with pain in the most severe cases only[66-70]. In light of this, a manual intervention whose goal is to limit degenerative changes and subsequently pain via a modification of physical parameters has little chance of being effective. Plus, there still is no consensus as to what physical parameters are more detrimental. For instance, it is still to be determined which of the stoop or squat lifting method is safer for the lower back[72]. Also, the ideal sitting posture still remains only in the realm of suppositions[71].

Other assumptions

There are a number of other assumptions that are made in the BMM of manual therapy. For this model to be accurate, one has to assume them to be true. For example, the sub-luxation or fixation has no credible proof for neither its existence nor its causal relation to pain[73,74,75]. Then there is the truly debatable facilitated segment concept which proposes that segmental hypomobility alone can be a trigger of neurophysiological changes, such as hypertonicity, leading to pain in local or distant tissues[62]. The assumption that segmental hypomobilities are themselves a dysfunction that can cause pain is another. Often these assumptions are challenged by researchers and when results cast serious doubts on the veracity of the assumptions, such results seem to either go unnoticed or get severely criticized. For instance, a recent study[76] has demonstrated that segmental hypomobilties are a feature of LBP but that they decrease during the course of treatment regardless if they were treated or not. Moreover, the pain intensity and subsequent relief were not correlated with the initial stiffness severity and its following decrease. Overall, with so many assumptions, many of which are questionable, it is difficult to understand why this model remains so prominent and largely unchallenged by the vast majority of physical therapists. This is especially true at the educational level. The biomechanical paradigm is still the dominant model taught in the Universities manual therapy courses and is definitively the main determinant of the clinical reasoning taught and encouraged by the Orthopaedic Division of the CPA via the Canadian manual therapy syllabus.

The general assumption that a number of biomechanical «dysfunctions» or features may play a role in the genesis of pain may find some support in the literature. Some studies may have demonstrated a correlation between different biomechanical features and pain. Again, we should be cautious before concluding that the relationship in such cases is a causal one. Certainly it is conceivable that mechanical features play a role in MSK pain but the fact of the matter is, that so far, tissue length, form, position or symmetry remain poor predictors of pain[56,76,77,65]. Even it were so, as commented above, the forces applied during common manual treatments for pain generally lack the necessary magnitude and specificity to achieve enduring changes in mature tissue length, form or symmetry.

The mechanical model's specificity and linearity

The fact that this model strongly relies on biomechanical characteristics for clinical reasoning and treatment, combined with the high prevalence in the asymptomatic population of what the model calls «biomechanical dysfunctions», introduces a specificity issue. Indeed, under such circumstances, the model will almost always find something biomechanically wrong in people with pain just as it would in people without pain. It then becomes

nearly impossible to accurately decipher which of these findings would really play a significant role in a person's painful condition. Another important issue is that the model does not account for the complexity of pain. Pain was shown by many authors to be a complex bio-psycho -social phenomenon both in terms of cause and experience. To associate almost every MSK pain with mechanical dysfunctions and «wear and tear» issues bypasses this known fact. The model does not take the psycho-social considerations much further than considering these factors as mere aggravating factors adding to the causal biomechanical dysfunctions. Such narrow consideration of important neurophysiological processes prevents this model from acknowledging other competing mechanisms that could account for part or all of the manual treatment's effectiveness.

Other considerations

It is truly possible that mechanical effects are expected and desired in some MSK conditions. For instance in sub acute post trauma injuries. Movement restrictions in joints post severe sprains that were immobilised possibly will benefit from a more traditional biomechanical type of manual therapy. The capsular and peri-articular tissues in such cases are in a healing and remodelling process and so these connective tissues are more likely to sustain permanent changes with the forces of manual therapy. But the main complaints in these conditions is not pain. One could question whether the restricted ROM is really the result of connective tissues adhesions/ restrictions rather than a protective reaction from the central nervous system (CNS). It might be a mixture of both. Nevertheless, perhaps in such cases the BMM might apply better. Consequently, this model is not entirely wrong and thus it would be cautious not to throw the baby out with the bath water. It could be argued, however, that in the cases where the main complaint is pain, the BMM seems largely erroneous. And finally, in the cases were the main complaint is not pain but restricted mobility, it is questionable that any permanent changes can be achieved with manual therapy if the limitations are the result of mature connective tissues restrictions. In such cases, one should be cautious before suggesting a permanent ROM improvement can be achieved with manual therapy.

The neurological model

When the preceding flaws of the BMM are mentioned, it is often counter argued that the BMM has demonstrated effectiveness. One could stress that the effect sizes in much cases tend to be modest but yet, it is effective. In response to these arguments, it could be highlighted that evidence of effectiveness is just that : evidence it has a positive effect. Evidence of effectiveness should not be mistaken with evidence of a sound, plausible and valid scientific theory. In support of that, comparable results can be achieved with other seemingly completely different treatments ie. NSAID, acupuncture, behavioural therapy and non-specific exercices[5,6]. All these treatments, just like manual therapy, show an effectiveness in the treatment of pain but all propose different rationales for their effectiveness. Why is that so? Obviously, these different rationales cannot all possibly be right. Conceivably, a NPM could unite all these different rationales.

As said earlier, there's a growing body of evidences demonstrating the presence of neurophysiological effects following the application of manual therapy. Numerous studies demonstrate immediate positive effects on pain after the application of non-specific manual therapy whether in the form of manipulation or mobilisation[3,19,24,48,49,78,79,80]. Lumbar manipulation has been demonstrated to produce a change in temporal summation resulting in an increase in thermal pain threshold in the leg in asymptomatic subjects[80]. Thoracic thrusts are demonstrated to provoke sympathetic changes in the hand[20]. Mobilisations or manipulations away from the painful area produce immediate changes in pain ratings[24,79,80]. These effects are either achieved with non-specific techniques or via a random selection of vertebral segment. These proposed neurophysiological effects could very well be a combination of both specific and non -specific effects in the peripheral (PNS) and CNS. Evidence for a non-specific effect in the CNS following a lumbar thrust was presented by Bialosky et al in a study[80] where the subjects' anticipation was determining the outcome of the manipulation for pain in the low back. In that same study, regardless of expectation, the thermal pain threshold in the leg was increased after the lumbar thrust which could be some evidence for a peripheral effect on temporal summation.

The complete neurophysiological mechanisms through which manual therapy achieves pain relief remains unknown but current knowledge of the neurophysiology of pain could help us elaborate a model for this mechanism. Bialosky et al. have recently proposed a very comprehensive model for the neurophysiological effect of manual therapy in the treatment of pain[7]. The actual pain science literature has demonstrated that numerous humoral, chemical, physical, social and behavioural factors all contribute to a subject's pain experience. Melzack proposes that all these factors interact and participate to the painful sensation by influencing what Melzack's calls the subject's own neuromatrix[81]. This model proposes that pain is, in fact, a brain's output in response to various stimuli including but not limited to nociceptive inputs. The decision of the brain to output pain is based on the perceived threat of the numerous factors mentioned above and their relative particular context. Anything that would increase the perceived threat is then likely to increase pain as well. Different factors such as stress, anxiety, hypervigilance, fear avoidance behaviours, memory, emotions and context are all potential variables that could influence how the brain perceives the stimuli it has to analyse[81-86]. In such a complex scenario, the same nociceptive stimulus is likely to yield very different responses by the brain from a person to another and from a situation to the next. Also, in order for the brain to output pain, a noxious stimulus is not necessary and pain can persist without such stimulation[82,83]. Phenomenas like peripheral and central sensitization produce conditions like primary and secondary hyperalgesia and allodynia and are the very manifestation of the complexity and the neuroplasticity involved in pain and chronic pain[82,83,87]. A good understanding of these notions of pain neurophysiology sheds a new light on many of the clinical manifestation of pain and should allow physical therapists to rationalise very differently many painful conditions they see clinically[87]. To fully grasp the rationale behind a manual therapy neuromatrix' approach to treating pain, one needs to perfect one's understanding of pain neurophysiology. The astute reader will surely be interested in the work of Moseley, Melzack, Wall, Butler, Benedetti and many others to perfect this kind of essential knowledge.

Pain *is* in the brain, thus in the central nervous system. It only makes sense that the conceptual model for the treatment of pain by manual therapy espouses this very fact and lets go of its traditional overtly mechanical paradigm. Combining the present knowledge of pain neurophysiology, evidence of a neurophysiological effect and lack of evidence for a lasting mechanical effect following manual therapy (along with the many other shortcomings) should allow manual therapists to redefine their model toward a more neurophysiological one. For instance, a simple explanation for a good part of the effectiveness of manual therapy could be that the novel stimulation introduced in the CNS by manual therapy *may* help the brain downregulate the perceived threat of current stimuli and thus decrease the pain by means of descending inhibition and other peripheral and central mechanisms (which include a placebo response). By this same mechanism, the brain could change the (mal)adaptive motor responses it was outputting because of the pain. In combination with that, reflexive reactions at the spinal cord level, via an influence on temporal summation, could also provide some temporary pain relief which could help in downregulating the threat some more. It is likely that the context in which the treatment is given, the treatment act itself, time and other variables will all account for the effect seen clinically after a course of manual therapy. In summary, in a neurophysiological model for manual therapy, both specific and non-specific reactions, including peripheral reflexive reactions and central/cortical processing could account for most, if not all the outcomes of a manual treatment aiming at reducing pain. Some part of the BMM could be salvaged, for instance, a competing mechanical effect of manual therapy could exist, in the form of improved perfusion and/or temporary alleviation of detrimental pressure on neural tissues in anatomical tunnels. Such a model implies that usual mechanically oriented manual therapies might be sufficient for pain relief but are not necessary. Different forms of manual care could then achieve the same kind of pain relief provided they succeed in decreasing the perceived threat and don't hinder the body's self healing mechanism (in the case of a real tissue injury). The therapist in such a model then becomes more an interactor than an operator.

This kind of neurophysiological model remains hypothetical, incomplete and might not be proven more effective in

terms of outcome, but, nevertheless, it is powered by up to date pain science based on serious references[7,15-20,24,25,51,80-87]. Furthermore, it is a more plausible one than the traditional biomechanical model that has too many shortcomings, lacks biologic plausibility and is too reliant on folkloric traditions. It is also a more encompassing one as all the components of the bio-psycho-social model of pain can be included in it.

What to do next?

It is demonstrated that knowledge of pain neurophysiology greatly changes how clinicians manage painful conditions and that there is a current lack of such knowledge by most clinicians[88]. It would be wise for educational institutions to place much more importance on pain neurophysiology. It would then be a lot easier to convince students of the many shortcomings of the biomechanical model as they would have an alternate explanation for what they see clinically. As for the biomechanical model's teaching, well, its proponents should accept and recognize its many flaws, let go of any hubris and embrace the new knowledge. Surely, this would mean we admit to more uncertainty, but that is what science is all about. The basis for a more plausible neurophysiological model should be presented and the clinical reasoning should be adjusted accordingly.

Sadly, that is not what seems to be happening. It is the observation of the author that different form of mechanically based treatment are growing in popularity among physical therapists, many of which are scientifically implausible or lack evidence for effectiveness (cranio-sacral therapy, myo-fascial release or visceral therapy). Furthermore, the material presented in many courses and seminars really does not reflect current pain science knowledge and omits criticizing the BMM as the biomechanical paradigm is often presented as if it had little known shortcomings.

Considering the present knowledge, it is the opinion of the author that there should be a shift in the teaching of manual therapy from the actual BMM towards a more neurophysiological one.

Conclusion

The BMM for the effectiveness of manual therapy in the treatment of pain makes many unproven assumptions, has many obvious demonstrated flaws and lacks prior biologic plausibility and thus, should seriously be questioned. An emerging NPM should be considered instead and gradually developed as new knowledge and evidences are gathered. The many educational institutions teaching manual therapy should acknowledge these facts and change their courses syllabus and manuals to reflect this current scientific knowledge.

Competing interests

Frédéric Wellens is the owner of Clinique Physio Axis, a privately owned physical therapy clinic.

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