When I was completing my training in the mid nineties it was commonly taught that small amplitude joint mobilizations were used to treat stiffness and that large amplitude mobilizations were used to treat pain. While a number of biomechanical based theories were taught to explain the possible mechanisms by which manual therapy improved hypomobile structures, Melzack and Wall’s pain gate theory appeared to be the only neurophysiological theory taught to explain pain modulation. Even at this time research had started to appear in therapy related journals suggesting that the brain played a role in manual therapy induced pain modulation, activating a supraspinal inhibitory pathway. Wright’s (1995) review on this topic found that a number of studies demonstrate manual therapy to cause both hypoalgesia, as measured by an increase in pain pressure thresholds, and concurrent sympathetic excitatory responses, as measured by changes in indicators such as skin conductance, skin temperature, blood pressure and respiratory rate. The author proposed that such responses, seen in human subjects, mirror the findings of previous animal studies in which an area of the brain know as the periaquaductal grey matter had been stimulated.

Since this review a steady stream of research has continued to be published relating to this area. What stands out about this body of work is the overall high quality of the research, with for example both placebo and control groups being commonly employed. Overall it suggests that the question of whether sufficient evidence exists to support the role of the central nervous system in manual therapy induced pain modulation is worth considering. In addition, if this position can be supported it is also worth considering what the clinical implications of this research may be. My essay this month aims to briefly summarize the research to date and attempt to answer the previously posed questions.

In the spine much of the research to date has investigated joint mobilization techniques applied to the neck. Schmid et al’s (2008) systematic review identified 15 relevant cervical studies, of which the overall quality of the studies was considered high, and reported that the evidence consistently supported findings of concurrent hypoalgesia, sympathetic nervous system excitation and changes in motor function. The authors conclude that these findings support the hypothesis that a supraspinal mechanism within the central nervous system (CNS) is responsible for the effects seen in response to the joint mobilizations. So what is it about the responses observed in these studies that suggest that they are controlled by the CNS higher up than at the level of the spinal cord? From Schmid et al’s (2008) review the strongest arguments appear to be based on two findings. Firstly, the increase in pain pressure thresholds, indicative of a hypoalgesic response, is not limited to the site of treatment but occurs in a widespread extrasegmental distribution in the upper extremities. Secondly, as concurrent sympathetic responses are also noted it would suggest that a higher center within the CNS is likely to be mediating this response.

Consistent with these findings in the neck, studies investigating joint mobilization responses in the lumbar spine, elbow and knee have also demonstrated significant hypoalgesic and sympathetic responses (Krouwell et al 2010, Perry and Green 2008, Moss et al 2007, Paungmali et al 2003). Evidence within these studies also supports a widespread hypoalgesic response, with for example the knee mobilization study finding an increase in pain pressure thresholds measured distally at the foot to be of a similar magnitude to that observed at the knee (Moss et al 2007). It is also noteworthy to consider Krouwell et al’s (2010) finding that pain pressure
threshold values were not affected by whether a large amplitude or small amplitude mobilization was used in the lumbar spine. To date this study appears to be the only research that has included mobilizations of different amplitudes, with grade III mobilizations being used for the majority of the other studies.

Based on the collective findings of 19 studies it appears reasonable to contend that pain modulation in responses to joint mobilizations is at least in part controlled by the CNS by means of a supraspinal inhibitory pathway. If a normal joint can be mobilized causing a hypoalgesic response in a distal body region, this poses a challenge to some of our clinical beliefs. For example, clinical situations often appear in which, even following a comprehensive examination, it is unclear if spinal structures are referring pain distally into the extremities. Treatment to the spine resulting in measurable changes to distal reassessment markers would previously have been suggested as evidence supporting the spine as the pain generator. Current research would suggest the need to recognize that this may be an error in clinical reasoning. Vicenzino’s (1998) research best illustrates this in a patient population. In this study mobilization to C5/6 was applied to a group of patients with lateral epicondylalgia, who had no known cervical pathology according to the screening tests performed as part of the inclusion criteria. The results of the study show that mean pain pressure threshold scores taken at the elbow had improved by 26% and that mean pain free grip values had increased by 29%.

Other potential clinical applications can also be drawn from this body of research. In the area of treatment selection, Schmid et al (2008) suggests that it provides reasonable rationale for the option of applying treatment to a proximal joint in clinical presentations in which treatment to the target joint is excessively painful or where manual therapy to the target joint is contraindicated. It also provides an alternative theory as to why some manual therapy studies show improvement in the outcome measures of neck pain patients when treatment is applied to the thoracic spine (Cleland et al 2005).

I hope you have found this essay informative and thought provoking. While this topic adds to the knowledge base of theories explaining the potential mechanisms by which manual therapy works, it by no means negates the existing neurophysiological and biomechanical theories. I suspect that this topic is likely to be one of the trends in the research world for the coming years. It appears likely that research regarding the effect of therapeutic interventions on the CNS and management strategies aimed specifically at modulating CNS responses will continue to filter into physical therapy training and continuing education.

References


