Joint Mobilization Overview

Joint mobilization (JM) is a passive therapeutic technique that is employed to enhance the flexibility of soft tissues and the mobility of joints. 1 Joint mobilization techniques became popular among physicians in the 1950s for the purpose of restoring joint range of motion, often involving joint manipulation under anesthesia. 2,4 The association of JM with strong manual thrusts originate in the 1970s, when controlled motion and patient cooperation during treatment was introduced. 2,4 The cause of lost joint mobility may be pain or progressive stiffness. 2,4 JM is often indicated for reversible joint hypomobility, maintenance of joint mobility when full-range active motion is not possible, avoidance of progressive stiffness from disuse, and relieving or diminishing pain. 2,4

Many theories exist concerning the mechanisms by which various JM techniques increase ROM and enhance function (Table 1). 2,5 The term “osteokinematics” refers to physiological movements of that can be actively performed through voluntary control of muscles. 1,5 The term “arthrokinematics” refers to accessory movements at the joint articular surfaces that must occur for unrestricted joint ROM. 1,5 Arthrokineumatic displacements that may be affected by manual JM techniques include roll, spin, and glide of the joint articular surfaces. 1-5 Maitland classified JM techniques intended to normalize arthrokineumatic displacements as static and oscillatory movements. 2,5 Maitland further classified oscillatory movement as the following: Grade I, small-amplitude movement performed at the beginning of the range; Grade II, large-amplitude movement performed within the range, but not reaching the limit of the range (any part of the range that is not restricted by stiffness or muscle spasm); Grade III, large-amplitude movement performed to the limit of the range; and Grade IV, small-amplitude movement performed at the limit of the range. 2,5

Articular Receptor Overview

Neurology provides the theoretical basis for specific JM techniques. 4 Four categories of afferent nerve fibers conduct impulses that originate from receptors located in synovial joints, which are designated as Type I, Type II, Type III, and Type IV. Ruffini endings (REs),
Pacinian corpuscles (PCs), and Golgi ligament endings (GLEs) are mechanoreceptors that convey information to the central nervous system by means of Type I, Type II, and Type III nerves. Unmyelinated Type IV nerves carry afferent pain impulses that originate from free nerve endings, which are termed nociceptors.\(^2\)\(^-\)\(^8\) Mechanoreceptor activation varies on the basis of the thickness of capsules that encase the receptor.\(^2\) Each mechanoreceptor has unique characteristics in terms of adaptation, threshold, and discharge frequency (Table 2).

Ruffini endings continuously generate afferent impulses, both at rest and during movement.\(^3\)\(^,\)\(^4\) They play a role in maintenance of muscle tone and awareness of static joint position.\(^3\)\(^,\)\(^4\)\(^,\)\(^7\) REs generate impulses at a frequency of about 10 to 15 Hz during maintenance

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| Cyriax | • Theorized that pain is due to a lesion and treatment must reach the lesion.  
• Evaluation involves a search for the type of tissue causing the complaint.  
• Employs strong movements (including manipulation) to return ROM. |
| Kaltenborn | • Developed from Cyriax’s approach.  
• Treats the lesion found in evaluation.  
• Treatment focuses on the joint capsule.  
• Traction and joint glide are used to increase joint mobility. |
| Maitland | • Treats the symptom without the confusion over diagnostic terminology.  
• Examination focuses on function and study of all anatomical structures.  
• Problems are grouped into PAIN/stiff and Stiff/pain. Problems may change from one group to the other.  
• Treatment uses grades of motion and two types of joint motion: glide and traction. |
| Mennell | • Joint play evaluation was developed by Mennell as an evaluation tool.  
• Joint mobilization is used to treat joint dysfunction.  
• Muscle pain is generally not assessed.  
• Assessment is to rule out contraindications to mobilization.  
• Treatment is sharp thrust to return normal range (muscle reeducation to maintain new range). |

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of a static joint position.3 During joint movement, the impulse discharge frequency increases in proportion to the stretch of the capsule.3,4 Having a low activation threshold, a small force (approximately 3 grams) can activate REs.4 They are primarily located in joints that control posture and locomotion, such as the neck, shoulder, and hip joints6 but are found in the superficial layers of all joint capsules.2-7 Because REs adapt slowly to a sustained stimulus, a slow and prolonged application of tension maximizes their response.

Pacinian corpuscles are known as acceleration mechanoreceptors, because their activation relates to velocity of joint motion at the beginning and end of displacement.3,4,7 They are located in the inner layers of fibrous capsules2-7 and fat pads7 and respond to a rapid increase in joint capsule tension with a discharge that lasts less than 0.5 seconds.4 PCs are inactive when the joint is immobilized or otherwise maintained in a static position.4 Because they generate a brief reflexive increase in the muscle tone when activated,2 PCs are classified as reflexogenic mechanoreceptors.7 Large numbers of PCs are found in joints that require fine motor control, such as the foot, hand, and lumbar spine.6 These joints respond best to JM that involves fast oscillation techniques at the beginning and end ranges of motion.

Golgi ligament endings are present in all intrinsic and collateral ligaments, except spine ligaments, and are categorized as reflexogenic mechanoreceptors.3-7 They generate afferent neural impulses that convey information about ligament tension, direction of joint movement, and joint position during active contraction or passive stretch. Golgi ligament endings have a higher activation threshold than REs and PCs, which occurs near the end-range of joint motion.2,7 Therefore, the GLE is activated by tension generated by JM at the end-range of movement,2-7 which requires a force of a few kilograms.6 Because GLE activation only occurs in response to extreme joint displacement, trauma could result from a lack of proficiency in administration of a JM technique that targets the GLE.

Nociceptors generate pain impulses in response to abnormal chemical or mechanical stimuli.3 Chemical stimuli include prostaglandin-E, potassium irons, polypeptides, histamine, and lactic acid.3 Mechanical stimulation results from abnormally high tissue tension.3,4 Nociceptors are non-encapsulated free nerve endings that are present in joint capsules, ligament fibers, fat pads, and the walls of blood vessels,6,7 but they are not found in the superficial layers of articular cartilage, menisci, synovial tissue, inner layers of the annulus fibrosus, and nucleus pulposus.3,8 They are not typically activated by JM techniques.

Joint Mobilization for Pain Control

Joint mobilization can be used for pain modulation, which involves mechanoreceptor activation for modification of nociceptor-generated pain impulse transmission.8 Essentially, inhibition of nociceptive input to the central nervous system depends on the volume and type of mechanoreceptive input.4

Joint mobilization creates tension within joint capsules.4 A static traction force produces the tension throughout all portions of a joint capsule.4 Although PCs only respond at the initiation of the traction force, REs convey information to the central nervous system about the amount of tension applied to the joint capsule.4 Therefore, traction is an appropriate option for joints such as the shoulder, neck, and hip.

When a gliding force is applied to the articular surface of a joint, the mechanoreceptors in the stretched joint capsule produce a diphasic response.4 The PCs respond with a brief burst of impulses. The REs within the portion of the capsule that is stretched are activated by the transition from a static to dynamic state, while the activation level of those within the nonstretched portion of the capsule decreases.4 The direction, velocity, and amplitude of the joint motion is perceived through the integration of input from both the stretched and slackened portions of the joint capsule.4

Both REs and PCs are present in all synovial joints. A widely utilized technique advocated by Maitland for the pain modulation is brief and repetitive mid-range joint oscillations induced by a small amount of force,1,2 which activates REs and PCs. Targeting mechanoreceptors located in the more dense tissues of a joint is most efficient for production of a large volume of mechanoreceptor input to the central nervous system. The knee joint capsule contains both REs and PCs, the infrapatellar fat pad contains PCs, the anterior cruciate ligament contains more PCs and GLEs than REs, and the outer two-thirds of the menisci and the peri-meniscal tissues contain REs, PCs, and GLEs.9 Hence, targeting activation of PCs with brief, low-magnitude oscillations may be suitable for knee joint treatment.6

Although the distribution of each mechanoreceptor type has not been determined for every joint, the
research literature suggests that a long, slow hold is suitable for the neck, shoulder, and hip, all of which contain a large proportion of REs. In contrast, short, fast movements are more appropriate for joints with a large proportion of PCs, such as the knee and the joints of the feet, hands, and lumbar spine.

**Reflex Effects on ROM**

Alteration of the mechanoreceptor input to the central nervous system can adversely affect kinesthetic awareness and postural balance. Musculoskeletal pathology and the inflammatory response to injury can increase some types of afferent input to the central nervous system and may decrease others. The responsiveness of the alpha motoneurons in the anterior horn of the spinal cord be either facilitated or inhibited by the altered afferent input received by the posterior horn of the spinal cord. Neural facilitation may cause muscle contractions to exceed the force demand of a task and to be sustained for a longer period time, which would increase circulatory and metabolic demands. Muscle tone and the responsiveness of stretch reflexes can be reduced by complex multisynaptic routing of afferent signals from mechanoreceptors and nociceptors, which activates inhibitory interneurons within the spinal cord that depress the responsiveness of alpha. Under some circumstances, the application of JM may contribute to a reflexive increase in muscle tone by facilitating the activation of motor units. Under other circumstances, JM may promote muscle relaxation, thereby increasing joint ROM.

**Conclusion**

JM can be effective for pain reduction and increased range of motion. The JM technique chosen for treatment of a particular joint should be based on the type of mechanoreceptor present. Theoretically, activation of a large volume of mechanoreceptors is a key factor for pain modulation.

**References**


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