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Neuro-Ocular Release: A New Osteopathic Technique For Resolving Somatic Dysfunction

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SPECIAL COMMUNICATION

Abstract

Neuro-ocular release(NOR) is a new osteopathic treatment modality that can be used in conjunction with any indirect osteopathic technique. It is proposed that NOR utilizes the recruitment of the visual system to access the descending pathways, while counterstrain access the ascending pathways, resulting in a resetting of the central and peripheral nervous systems. This resetting allows for dampening of the potentiation of somatic dysfunction (SD). The ascending pathways integrate with many of the vision and ocular reflex pathways influencing the descending response to the peripheral tissues, the location of palpable somatic dysfunction. The authors purport the NOR technique allows for more time efficient and effective treatment by changing the central nervous system entrainment of SD.

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Introduction

Osteopathic manipulative techniques continue to evolve with the discovery of neurological mechanisms and their interactions between the somatic and nervous systems. Each osteopathic manipulative treatment (OMT) procedure has its benefits. In the case of counterstrain (CS), it is an effective indirect treatment method, but efficiency (time) is an issue. To address this deficit, the principal author founded this new technique, neuro-ocular release (NOR).

CS, an indirect positional release technique developed in the 1950s by Lawrence H. Jones, DO, FAAO, has broad applications for acute or chronic somatic dysfunction (SD) in all age groups. The principles of treatment are to modulate neuromuscular dysfunction effectively, reducing hypertonicity of a muscle in spasm.[1] The physician palpates a tenderpoint in the affected area, monitoring the tenderpoint while placing the patient in a position of ease (disengaging the restrictive barrier) resulting in softening of the tenderpoint. The position is held for 90 to 120 seconds, the patient is passively returned to a neutral position, and the tenderpoint reassessed, with the goal of pain reduction, improved motion, and physiology.

NOR is a new osteopathic manipulative procedure discovered by the primary author to treat neurologic model SD more efficiently by 3 vs. 90-120 seconds and effectively by addressing entrainment of SD. NOR utilizes the visual/ocular nervous system in conjunction with indirect OMT. Recruiting the visual system during OMT itself is not novel, e.g., oculocephalogyric recruitment during cervical muscle energy; however, NOR is a novel combination using ocular reflexes, a fixed stare, closing eyes, and indirect positioning. NOR utilizes CS positioning, then the physician directs the patient to visually focus (stare) at a specific point in the room that when aligned correctly, results in further softening of the tenderpoint. The authors propose ocular recruitment allows for a reset of the central nervous system (CNS) memory (entrainment) of the SD, allowing for a "warm reboot of the computer," so to speak, of the CNS. This technique theoretically provides a more complete activation/deactivation of the neuro-chemical complexity of SD centrally and peripherally.

Many patients present to primary care with pain, and the majority have neuromuscular pain associated with SD.[2] SD is impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial and myofascial structures, and their related vascular, lymphatic, and neural elements.[3] The

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principle treatment for SD is OMT. NOR is specifically designed to address the pain pathway as well as CNS components that maintain the feedback loop (dysfunction-pain-dysfunction) found in SD. In 2016, the primary author had difficulty treating a patient's shoulder SD/pain and decided to use a CS technique with the thought, "If I align the whole body including the visual system in the position of injury, it may facilitate a more complete and rapid release." And it did in ~3 seconds.

NOR's resetting of the biological mechanisms that sustain the SD are specific, complex and sophisticated. SD is identified and reflected in the soma, but it is learned/maintained centrally, in the spinal cord, sensory and motor cortex. Most manual manipulative techniques have focused on peripheral symptoms, signs, and effects. However, there are few osteopathic manipulative procedures that address the treatment of both peripheral (PNS) and central (CNS) components of SD.

SD is found and maintained peripherally through the effect of muscle tone, muscle spindles, neurological apparatus, and muscle twitch (slow/fast) fibers. The peripheral-central interplay begins in the PNS where the neural gamma efferent nerves, stretch receptors, and afferent sensitization responses, including chemical, mechanical, long term/sensitizing effects of pressure and edema, are the actors that cause learned and maintained SD.[4] These structures are thought to be modulated by the CNS, particularly the spinal cord, where learned patterns of behavior and central entrainment

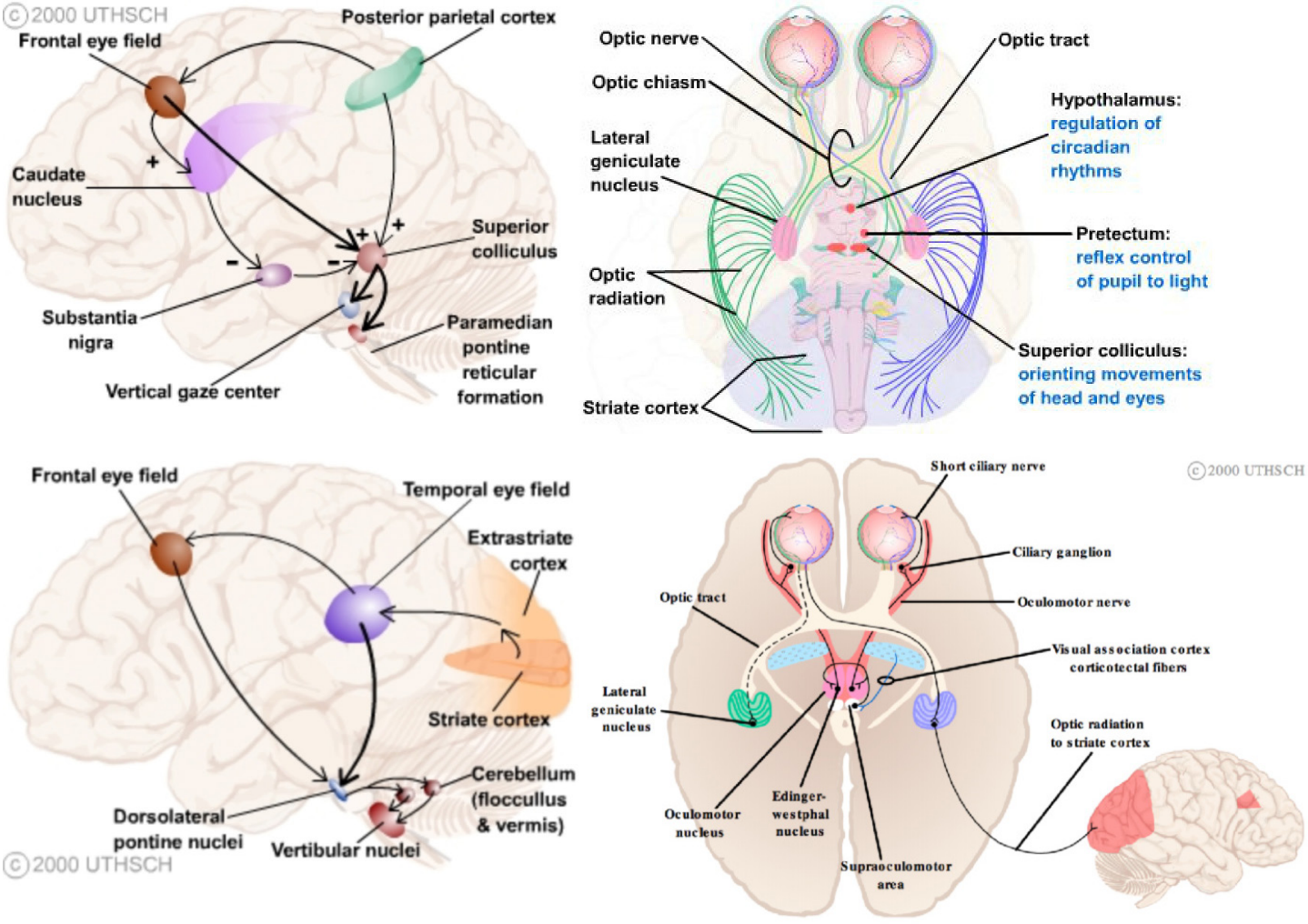

Image 1. **Neuro Ocular Anatomy***: Images reprinted from Neuroanatomy Online, an open access educational resource created by McGovern Medical School at UTHealth[6,7].*

- **Top Left:** *The voluntary saccades circuit.*
- **Top Right:** *The inferior surface of the brain illustrating the visual pathway.*
- **Bottom Left:** *The smooth pursuit pathway.*
- **Bottom Right:** *The accommodation pathway including the supraoculomotor area, and parasympathetic neurons (i.e., the Edinger-Westphal neurons) of the oculomotor nucleus.*

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occurred, reinforced electrically and chemically. NOR is unique in addressing the central and peripheral SD interplay using osteopathic indirect methods via peripheral tenderpoints and NOR alignment technique.

Once the indirect method of OMT is initiated, such as CS, then the NOR procedure is introduced by having the patient stare at a point in the room. The point is usually in the direction of injury, verified by an increased softening of the tenderpoint under the physician's finger when the eyes are correctly aligned. The authors suspect the recruitment of multiple neural pathways when the patient's body and ocular reflexes are aligned to a position of ease, suggesting there may be differing functions of reflex mechanisms when different regions of the nervous system are recruited.[5]

Theory of the Mechanism

The mechanism begins with a position of ease accompanied by an optical reflex and stare. The image passes through the fovea centralis, optic nerve, optic chiasm, optic tract, lateral geniculate ganglion, optic radiation, and visual cortex, which has diffuse synapses with the thalamus, amygdala, hypothalamus, superior colliculus and corticospinal tract, the ventral horn affecting the peripheral nervous control of SD and the feedback through the dorsal horn anterior and lateral spinothalamic tract to motor cortex and thalamus, hypothalamus (see image 1).[4] The electrical and chemical balancing of the CNS is likely key in resolving SD more completely than traditional segmental joint biomechanical procedures.

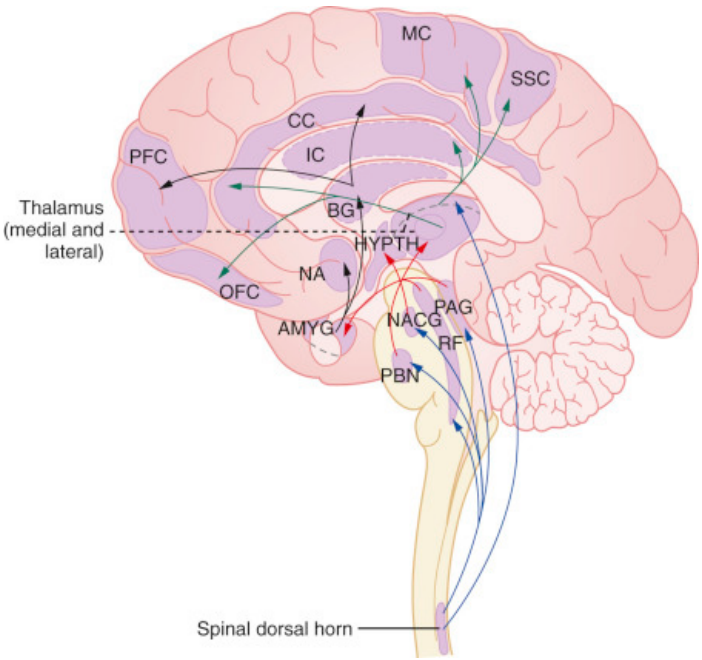

***Image 2.* Afferent Ascending Pathways:** *reprinted with permission from Pharmacology and Physiology for Anesthesia (Second Edition): Foundations and Clinical Application*[12].

Ascending Pain Pathway

The induction of neuromuscular pain begins in the skin and joints. Various stimuli generate impulses through different receptors in the periphery of the body, especially the skin. Receptors located in the dermal, epidermal, and subcutaneous tissue provide specialized somatosensory functions.[4] However, sensory qualities cannot be isolated to individual receptors at all times. Nociceptors consist of free nerve endings making up nearly 50% of all receptors. These impulses are transmitted through specific tracts, described below, to the sensory cortex of the brain. Proprioceptors found in muscle spindle fibers, tendons, and joint mechanoreceptors allow for the sense of position in space, motion, and force. General sensory afferent (GSA) signals coalesce within the cord and are transmitted to the cortex by a chain of neurons and axons located at specific sites in the CNS (see image 2). The primary somatosensory cortex (S1°) in the postcentral gyrus processes the conscious perception of pain including localization, quality, and intensity. The secondary somatosensory cortex(S2°) has proposed implications in the memory of sensory input.[4,8]

Somatic pain is conducted by myelinated A-fibers for temperature, pain, and position. Unmyelinated fibers transmit temperature and pain in the trunk and limbs. The GSA fibers, located in the spinal ganglion, terminate at the posterior horn of the spinal cord gray matter in Rexed laminae I, II, IV, V, and VI.[4] They synapse with the anterolateral system, composed of neospinothalamic and the paleospinothalamic tracts of the pain pathway.

The neospinothalamic tract synapses in the ventral-posterolateral nucleus of the thalamus and projects to the S1°and S2° cortex, through the internal capsule and corona radiata. Additional neurons project to the ventral-posteroinferior nucleus, intralaminar nuclei, and collaterals to the reticular formation. Intralaminar nuclei neurons ascend to the striatum, S1°, S2°, and cingulate gyrus, as well as the prefrontal cortex.[4,6,7]

The paleospinothalamic path synapses in the reticular formation send projections to intralaminar nuclei of the thalamus, hypothalamus, as well as the limbic region and are primarily responsible for the emotional component of pain. The neurons ascending from the Rexed laminae are the spinomesencephalic and the spinoreticular tracts. Spinomesencephalic tracts terminate mainly in the periaqueductal gray (PAG), while others terminate in the midbrain raphae nuclei. The spinoreticulothalamic extends to the reticular formation

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in the medulla containing the gigantocellular nucleus and nucleus raphes magnus. These reticulothalamic fibers transmit pain impulses to the medial thalamus, the hypothalamus and the limbic system, components of the reticular activating system (RAS).[4,6,7]

The third order neurons of the sensory pathway, located in the thalamus, send projections to the postcentral gyrus of the S1° cortex. Ascending axons of the spinal lemniscus, anterior and lateral spinothalamic tracts, travel alongside axons of the pyramidal tract in the dorsal part of the internal capsule.[4,9]

The pathways for pain and temperature initiate at the skin, travel to the spinal ganglion of the afferent nerve fibers and terminate at the posterior horn of the spinal cord gray matter in the Rexed laminae. The pathway ascends the lateral spinothalamic tract to the ventral-posterolateral nucleus of the thalamus with termination in the postcentral gyrus sensory cortex.

Unconscious proprioception starts at the muscle spindles, tendon, joint, and skin receptors to the spinal ganglion. The primary neuron of the afferent nerves within the spinal ganglion sends axons terminating at the dorsal column gray matter beginning the anterior spinocerebellar tract. Neurons run directly to the cerebellum, crossed and uncrossed, passing through the superior cerebellar peduncle to the vermian part of the spinocerebellum. The posterior spinocerebellar tract begins at the base of the posterior horn gray matter and travels to the cerebellum without crossing, passing through the inferior cerebellar peduncle terminating in the spinocerebellar vermis.[4,9]

The pathways for position sense, conscious proprioception, vibration, and touch begins in the Vater-Pacini corpuscles of the skin, muscle, and tendon receptors traveling to the spinal ganglion. The pathway ascends the cord to the nucleus gracilis and nucleus cuneatus, of the lower medulla, synapsing and ascending, crossing midline, and traversing the medial lemniscus to the ventral-posterolateral nucleus of the thalamus with neurons traveling to the postcentral gyrus sensory cortex.[4,8,9]

The vestibulo-ocular reflex (VOR) allows for gaze stabilization during head rotation, keeping the object in focus on the fovea. The slow phase opposes head rotation to keep gaze steady whereas the fast phase saccades allow for re-centering if the slow phase overshoots. The pathway for this reflex is initiated in the semicircular canals. Bipolar cells in the canals, via CN VIII, synapse on premotor cells in the vestibular nuclei. Premotor neurons of the nucleus synapse with motor neurons in the CN nuclei, which in turn act upon the extrinsic muscles of the eye.[10]

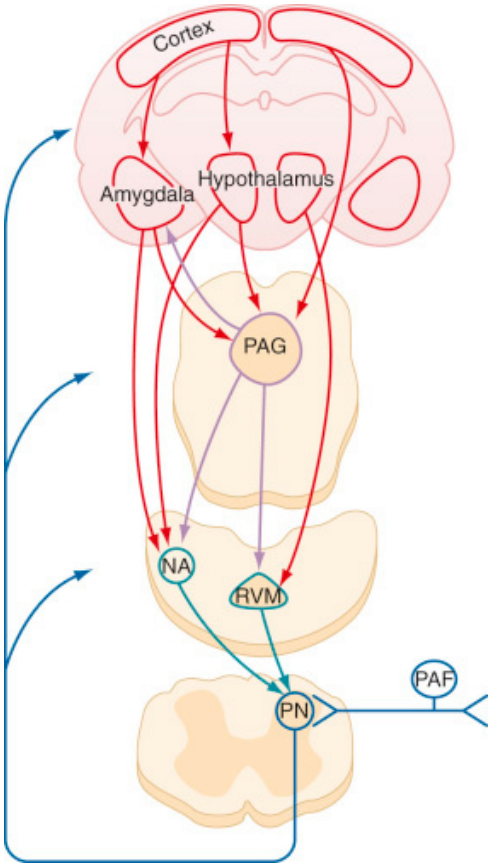

***Image3.* Efferent Descending Pathways:** *reprinted with permission from Pharmacology and Physiology for Anesthesia (Second Edition): Foundations and Clinical Application*[12].

The oculocervical reflex connects the vestibular system with proprioceptive afferent and efferent communication through the spinal tract, producing trunk rotation in response to a stimulus. This pathway plays a lesser role in a person with an intact vestibular system; however, it plays a more active role in patients with vestibular impairment.[10]

Optokinetic reflex maintains a moving object on the retina while the head remains stable. It produces optokinetic nystagmus consisting of alternating slow, compensatory movement in line with the object movement, and fast, anti-compensatory, movement opposite the movement of the object.[11] This reflex is mediated by the geniculo-transcortical-floccular pathway **and** works in conjunction with the VOR.[10]

Major components of the nociceptive system. An overview of the major anatomic components that process nociceptive information from the periphery to cortical structures. Its critical integrative function is underlined by connections to specific cortical and

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subcortical structures modulating emotional and behavioral processes. *AMYG,* amygdala; *BG,*Basal Ganglia; *CC,* Cingulate Cortex; *HYPTH,* hypothalamus; *IC,* Insular Cortex; *MC,* Motor Cortex; *NA,* Nucleus Accumbens; *NACG,*Noradrenergic Cell Group; *OFC,* Olfactory Cortex; *PAG,* Periaqueductal Grey; *PBN,* Parabrachial Nucleus; *PFC,* Prefrontal Cortex; *RF,* Reticular Formation; *SSC,* Somatosensory Cortex.

Descending Pain Pathway

The PAG receives inputs from the cortex and is capable of activating a powerful analgesic effect. The rostroventromedial medulla (RVM) can facilitate or inhibit nociceptive inputs and acts as the relay station for descending pain signals.[12] These structures provide a mechanism through which cortical and subcortical sites can influence nociception (see image 3). Faull and Pattinson revealed activation and resting connectivity between the PAG and the visual cortex, S1°, the thalamus, medulla, PFC, occipital cortex, cerebellar lobes, and other higher cortical regions.[13] Neuropsychiatric studies suggest that dynorphin and their associated kappa receptors have a dynamic influence over the emotional component of pain as well as modulation of the peripheral pain response.[14] The cortex sends efferent fibers to the PAG, the Raphe Nucleus Magnus (RNM), and the RVM. The RNM contains monoamine pathways projecting to the dorsal horn, which can exhibit excitatory and inhibitory functions. The PAG-RVM pathway is the target of opioids and opioid-like substances and is influenced by stress and emotion. The effect is inhibition of the presynaptic primary afferents and postsynaptic inhibition at the spinal projections.[12]

The interrelationship of major neuroanatomical components exerting descending nociceptive control is shown. The periaqueductal gray*(PAG)* and rostral ventral medulla *(RVM)* integrate input from cerebral structures and relay to the spinal dorsal horn. Noradrenergic pontine and medullary nuclei*(NA)* directly project to the dorsal horn. Presynaptic and postsynaptic mechanisms modulate nociceptive information from primary afferents *(PAF)*to spinal projection neurons *(PN).* Inhibition or excitation of spinal interneurons. Cortical areas, amygdala, and the hypothalamus exert top-down control. These structures modulate pain experienced by stress, emotion, and cognition.

The above pathways, afferent proprioception, vestibular afferents, and ocular feedback coalesce to produce efferent neuromuscular and pain modulating responses throughout the body. The afferent pathways ascend in close proximity within the spinal cord and synapse throughout the cortex and CNS. Disruptions along any of the pathways have the potential to influence adjacent pathways and ultimately the descending CNS response. This complex network has been previously described as the Pain Matrix, which is much more entangled than a simple stimulus and response loop.[8,15] Functional MRI studies have revealed that pain memory and integration occur throughout the brain and is further compounded by comorbid conditions, polypharmacy, and genetics.[12,15,16]

Current osteopathic treatments targeting nervous system response focus mostly on the general afferent pain and efferent response pathways. NOR integrates the vestibular and ocular components' activating overlapping CNS tracts that help modulate the descending response. The authors propose NOR resets the neurovascular-somatic system to neurological normalcy by using the optical tract to influence the descending motor tracts, attempting to reset the cerebellum and cerebrum for normalized or optimized function (i.e., pre-SD state).[17]

Application

The primary author has created a video presented online outlining the procedure of the NOR technique (https://youtu.be/eBYE-C7lCbuU). The video demonstrates treatment for upper thoracic complaints, but NOR can be used in place of, or in conjunction with, any indirect osteopathic treatments.

For example, to use NOR to treat the anterior shoulder, the physician first palpates a tenderpoint. The patient is instructed and assisted to a position of ease, and tenderpoint pain has resolved; in this case, placing the affected side hand on the frontal over the coronal suture. The physician continues to monitor the tenderpoint while directing the patient to align their eyes with the position of ease, asking the patient to stare at the ipsilateral elbow. Correct alignment occurs when the tenderpoint softens more completely under the physician's finger. The patient is instructed to stare at their elbow for 3 seconds, then close their eyes, straighten up to their neutral position, and open their eyes. The tenderpoint is again palpated with the localized goal of complete resolution of pain.

Indications include:

- Somatic and visceral pain associated with acute, subacute, and chronic SD
- Neural, myofascial, and arthrodial pain associated with SD
- Appendicular and axial pain associated with SD commonly having tender points

NOR Requirements

- Cooperative patient
- Patient with some vision (even 20/400)
- Patient with the ability to follow directions and focus

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- Identifiable somatic dysfunction (tenderpoint)
- Highly skilled practitioner of Indirect OMT

NOR Contraindications

- Relative
  - No SD
  - No palpable tenderpoint
- Absolute
  - Uncooperative patient
  - Patient unable to focus/stare or with no orbital contents
  - Acute fractures and other acute medical emergencies

Conclusion

Neuro-ocular release (NOR) is an advanced, indirect osteopathic manipulative procedure that puts into play the visual/ocular nervous system utilizing the CNS as described above to deactivate entrainment, central facilitation, and local somatic dysfunction. By utilizing the neuro-ocular system in the manner described above, the inhibition of the presynaptic primary afferents and postsynaptic inhibition at the spinal projections modulate pain and diminish somatic dysfunction. The authors purport that NOR offers a faster and more complete treatment of somatic dysfunction that osteopathic clinicians should consider, particularly for recurrent neuromusculoskeletal pain with SD.

Limitations

A controlled study of this technique as compared to sham and other indirect OMT has not been performed. However, the primary author has performed NOR on 3000+ patients for the past 3 years with patient-reported 98% pain reduction following treatment. Additionally, the proposed mechanism of action of NOR above is theoretical, not proven.

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