Inclusion of Primal Reflex Release Technique™ (PRRT™)

in the plan of care for shoulder pain: A case report

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ABSTRACT:

Shoulder pain in physical therapy is a common occurrence; however, literature is mixed when it comes to the most efficacious treatment approach. The purpose of this paper was to introduce a new technique, Primal Reflex Release Technique™ (PRRT™), into the realm of shoulder care. This article describes the management of a 55 year old male patient with bilateral shoulder pain during elevation which was insidious in nature. The plan of care involved both traditional therapeutic approaches (mainly strengthening) and PRRT™. Following treatment with PRRT™, the patient no longer had pain with elevation and was able to immediately begin strengthening exercises to address the underlying cause of pain. Literature to explain the mechanism of action for PRRT™ or provide validity/reliability data is currently absent. Future studies need to focus on attempting to answer these questions.

INTRODUCTION:

As an outpatient physical therapist, the number of shoulder patients with limited range of motion or pain one will see during their career is immense, and for every one encountered there is likely a different treatment strategy/technique that could be used and could be successful. Included in this laundry list are various manual therapy techniques, modalities, therapeutic exercises, and

passive stretching, however, the most efficacious combination or stand-alone treatment to improve shoulder range of motion and/or decrease pain has yet to be established in the literature.

The purpose of this paper was to attempt to present a new technique into the realm of shoulder treatment, Primal Reflex Release Technique™ (PRRT™), which is currently absent from the literature, but showing promising anecdotal results. The main goal of this treatment technique is aimed at decreasing patient's pain. This is achieved through stimulation (facilatory tapping or quick stretch) of either the contralateral agonist or ipsilateral antagonist muscle groups of identified reactive sites (areas that are painful to fingertip transverse pressure). The mechanism of action behind this technique is yet to be understood, nevertheless, there are several theories as to how it could work that are currently being seen in other techniques throughout healthcare. However, before we consider the mechanism through which PRRT™ may work, we must first consider pain itself and what may result from it.

Pain is defined as "a more or less localized sensation of discomfort, distress, or agony, resulting from the stimulation of specialized nerve endings."[1] Once free nerve endings are stimulated, there are two pathways for which pain can travel to the central nervous system: fast-sharp neospinothalamic pathway (senses mechanical or thermal pain and transmits signals via small A delta fibers) or slow-chronic paleospinothalamic pathway (senses chemical pain and transmits signals via type C fibers). The majority of the fast-sharp pain fibers terminate in the thalamus with very few terminating in the reticular areas of the brainstem. The slow-chronic pain fibers however, mainly terminate in the reticular areas of the brainstem with only $^{1}/_{10}$ to ¼ of fibers terminating at the thalamus. These reticular areas of the brainstem are not only important in pain

perception (communicate with cerebellum, cerebral cortex, and basal ganglia), but also are excitatory in nature resulting in both increased arousal throughout the brain and increased gamma efferent activity. Gamma efferent activity is responsible for innervating intrinsic musculature (specifically muscle spindles which sense and maintain muscle length). Thus, increased pain results in increased bulboreticular excitation causing increased facilitory gamma efferent signals and therefore increased intrinsic muscle activity (AKA tone).

With a basic understanding of pain, the pathways that it uses, and the lasting effect, we can now consider how PRRT™ may work. The main theory as to why many of the PRRT™ techniques may work is based largely on Sherrington's second law, which states "when a muscle receives a nerve impulse to contract, its antagonist receives simultaneously an impulse to relax."[1] This is more commonly known as reciprocal innervation. Either cutaneous or nociceptive stimulation of a body area (results in change of muscle length and tension which stimulates activity from muscles spindles and golgi tendon organs) will cause a reflexive excitation resulting in contraction of the underlying muscle along with an inhibitory signal to the ipsilateral antagonist (AKA flexor reflex).[2] Simultaneously, as the nerve impulse synapses in the spinal cord to send the contraction message down the motor neuron to the side stimulated, it also has synapses that cross the spinal cord and cause inhibition of the agonist muscle group and excitation of the antagonist (AKA crossed extensor reflex).[2] For example, cutaneous or pain stimulus to the right biceps would cause the right biceps to contract and the right triceps to relax, while also resulting in left biceps relaxation and left triceps contraction. This reflex fatigues within seconds,[3] but afterdischarge can prolong the time it takes for the muscle to return to baseline contraction status (largely dependent on the strength of the stimulus with a stronger stimulus resulting in an

increased afterdischarge).[2] Many of the PRRT™ techniques use cutaneous stimulation of either the ipsilateral antagonistic or contralateral agonist muscle groups to accomplish reciprocal inhibition of select muscle groups.

The above-theorized mechanism of action can explain the majority of PRRT™ techniques, however, along with reciprocal inhibition, there can also be stimulation of the negative stretch reflex. A muscle that is suddenly shortened causes fewer nerve impulses to be sent from the underlying muscle spindles (reflecting the shortened muscle length) and results in relaxation of the muscle in order to maintain the resting length.[2] Both reciprocal innervation/inhibition and, to some extent, the stretch reflex are common mechanisms of action behind many current "traditional" physical therapy techniques (for example proprioceptive neuromuscular facilitation).

A second theory that may come into play involves disrupted gamma activity (facilitory to inhibitory and/or efferent to afferent). As described above, the slow-chronic pain pathway eventually results in increased facilitory gamma efferent activity. It is proposed that the normal resting balance between gamma activity is the result of presetting from higher control centers such as the cerebellum, basal ganglia, and cerebral cortex. Due to the close communication between the bulboreticular areas and those higher control centers it is theorized that recalcitrant pain could lead to a higher preset of gamma efferent activity secondary to motor learning. There are currently several osteopathic techniques that use this particular theory as the basic mechanism of action, stating that correcting inappropriate proprioceptive information and thereby decreasing gamma efferent signaling resets the balance between afferent and efferent

ativity and ultimately leads to a more harmonious movement pattern.[4] In this theory, the PRRT™ techniques would provide an influx of proprioceptive information (afferent activity) to allow for comparison to current preset efferent activity and therefore adjustments made to correct for the heightened baseline activity.

A third theory, based on the thought that people can remain in heightened states of sympathetic activity following injury or recalcitrant pain,[5] argues that several of the PRRT™ techniques assist in stimulating the vagus nerve (parasympathetic nervous system), which helps to restore homeostasis and/or decrease pain perception. There have been several studies that have looked at the effect of low level vagal nerve stimulation in regard to people with depression or chronic pain and have found that low level stimulation results in pro-nociceptive effect (decreased pain perception).[6,7] The difficulty applying this theory to PRRT™ is how exactly the vagal nerve is being stimulated and can short term stimulation produce the same results as a continuous stimulation which was used in the studies.

Although these are three of the main theories behind why PRRT™ may work, one must also consider everything else that is being fired while attempting to stimulate select muscles (for instance, golgi tendon organs, muscle spindles, cutaneous receptors, joint receptors, possibly nociceptors, etc).[8] It is possible that all of the tapping/stroking/quick stretch is simply gaiting pain. The point is, why or how PRRT™ works is still unknown. However, despite this, PRRT™ is being used and is resulting in positive anecdotal results. The following case study is one such example.

THE CASE:

Patient History

The patient was a 55 year old sedentary African American male with chief complaint of intermittent right shoulder pain/numbness that extended down his lateral arm to his elbow with over-shoulder level elevation or cold weather. The patient rated an average pain of 4-5/10 and worst of 7-8/10 and best of 0/10 (with rest) on a 10mm visual analog scale (VAS). The patient was referred to physical therapy by his primary physician with a diagnosis of bilateral shoulder tendonitis; however, per the patient there was only difficulty with the right shoulder. No specific mechanism of injury was reported, but rather a gradual onset 2 years prior which had been worsening over the past year. The patient sought treatment via his physician a year ago when the symptoms began to worsen and was prescribed pain medication (patient couldn't recall the name of the medication) which had minimal to no effect. No imaging had been taken and no other health problems, major accidents or surgeries were specified. The patient reported a history of smoking and stated he was taking Naproxen PRN (as needed). The patient had retired from the air force nine years prior and stated that he had led a very sedentary lifestyle since. The patient stated his goal for therapy was to regain full, pain-free use of his right upper extremity for over-shoulder level motions to allow for lifting/carrying of objects.

Although this case was not unique in presentation, this particular patient was selected because his case was relatively uncomplicated (no co-morbidities/confounding factors) which helps to eliminate biases and make for an easier match to the general population. Despite the limited complications, one factor that initially appeared as though it may affect the outcome of this

treatment was that this patient wasn't entirely comfortable about being treated by a student physical therapist and needed reassurance about credentials prior to beginning the initial evaluation.

Examination

On first observation, the patient presented with poor to fair sitting/standing posture with rounded shoulders, protruded head position (majority of ear protruding in front of the patient's shoulders) and winging scapula bilaterally (left > right). All of these measurements were based solely on subjective observation as there are no objective quantification techniques to my knowledge.

Following general observation of the patient, the cervical spine (CSP) was evaluated first given its proximity to the shoulders and the symptom described as "numbness." A McKenzie screen was used (single test of CSP flexion, extension, protrusion, retraction, bilateral sidebend and rotation followed by repetitions of single motions) and revealed cervical range of motion (ROM) within normal limits (WNL), except minimal limitation (<25%) with cervical retraction. Repeated cervical retraction (20 repetitions) in sitting abolished numbness in the patient's right lateral arm, however did not relieve the pain associated with over-shoulder level motion. With a positive partial abolishment of symptoms following cervical movement, it was evident that a quick screen for neurological involvement was indicated. A quick screen for intact sensation and reflexes was performed. Using light finger-tip brushing on both upper extremities at the same time, the patient was asked to identify if it felt the same or different side to side to indicate any altered sensation to light touch. Patient identified no altered sensation. Reflexes for the upper

extremities were carried out in a conventional manner with the patient seated. Bilateral biceps brachii, triceps brachii and brachioradialis reflexes were normal (2+).

Since cervical retraction accounted for only a portion of the patient's symptoms and there did not appear to be any other neurological involvement, the patient's shoulders were evaluated next. Shoulder active range of motion (AROM), which was taken in sitting (**see Table 1**), revealed full painful elevation bilaterally with compensatory strategies used on the right (positive shoulder shrug). The patient also displayed disrupted scapulo-humeral rhythm (left > right). Manual muscle testing, performed as described by Reese, 2005[9], revealed decreased strength and pain with resisted elevation (right > left), compensatory strategies on the right with elevation, decreased scapular strength (right > left), and decreased rotator cuff musculature (**see Table 2**). Passive range of motion (PROM) with the patient in supine was WNL bilaterally and without pain.

At this point in the examination, there appeared to be several options that could be the root cause of this patient's symptoms including an impingement syndrome, acromioclavicular pathology, or contractile dysfunction of the rotator cuff musculature. Special tests with moderate to good reliability/validity were selected to assist with ruling in/out the possible causes for the painful elevation and compensatory strategies being observed. All special tests were executed as described in Dutton, 2004[10]. Patient displayed a positive AC shear (pain replication) and Near impingement test (superficial and superior pain reproduction) on the left and a positive O'Brien's Active Compression test (superficial pain reproduction), Cross-over Impingement test (superficial pain both superiorly and in the pectoral region), Hawkins-Kennedy test (pain

replication within the shoulder), and Neer impingement test (pain replication within the shoulder) on the right (refer to **Table 3** for accuracy, sensitivity and specificity measures for these tests[10, 11, 12]). These tests seemed to point to an impingement syndrome on the right and an AC pathology on the left.

The last part of the examination was palpation, which was performed in accordance with the PRRT™ examination. For this, the patient was positioned in supine with head supported on a pillow and palpated for tenderness/reactive areas by using fingertips to gently rub transversely across select muscles alternating from right to left. The patient was asked if there was tenderness and if so which was worse (right or left), while also being observed for signs of discomfort (audible noise, facial expression, or pulling away from the stimulus). The following areas were palpated in this manner: suboccipitals, masseters, sternocleidomastoids, scalenes, upper trapezius, levator scapula, middle trapezius/rhomboids, infraspinatus, supraspinatus, and pectoralis minor. The patient was reactive on the right > left in all of the above areas except no tenderness over the middle trapezius/rhomboids or the pectoralis minors. Tenderness/reactivity to this mild stimulus suggested that this particular patient could be a good candidate for PRRT. In addition to the reactivity, this patient also had a history of recalcitrant pain (possibly more effectiveness based on theories of action) and was not displaying specific patterns of restriction.

In summary, following the entire examination/evaluation the physical therapy working diagnosis was secondary impingement syndrome in the right shoulder and acromioclavicular pathology in the left shoulder. In addition to those diagnoses, the patient had increased sensitivity/reactivity

on the right throughout the cervical/shoulder complex which could have been contributing further to his secondary impingement.

Intervention/Outcome

PRRT™ was started immediately following the examination with patient consent after explaining that the following intervention was about using reflexes to turn off muscles and in order to achieve this he must be caught off guard. The patient was skeptical but stated he would give it a try. With the patient in supine with head supported on a pillow, he was treated using PRRT™ to address the increased tenderness/reactivity on the right through either stimulation of the contralateral agonist or ipsilateral antagonist musculature (**see appendix for description of techniques**). The techniques were performed in the order they are presented in the appendix and after performing each technique the initial tender areas were re-palpated to assess for change. If the initial technique didn't result in abolishment of the increased reactivity, a different technique was attempted (for example, switching from ipsilateral agonist to contralateral antagonist). This was repeated until the patient was no longer tender or reactive at any of the locations as was initially.

Once the patient no longer had any increased reactivity, he was asked to sit up and move his upper extremities though elevation. The result was full, pain-free ROM bilaterally, except 1/10 pain in left shoulder at end-range elevation. The disrupted scapulo-humeral rhythm was still present (left > right), however, there were no longer any compensatory strategies being used on the right as was initially seen. The patient also moved through full elevation range of motion at a much quicker/spontaneous pace than was initially observed. Since the patient no longer had pain

with elevation, the rest of the initial treatment consisted of strengthening exercises for the scapular and rotator cuff musculature (included thera-band rows, internal and external rotation, 2# dumbbell flexion/scaption/extension to 90', prone shoulder extension with arms by sides, doorway pec stretch, and UBE), repeated cervical retraction in sitting, and education on neutral posture. The patient was extremely pleased with his progress at that point, but was warned about possible soreness given his current level of activity.

Per the MD, the patient was to be seen two times per week for four weeks. At the next visit two days later, the patient still had full and pain-free ROM bilaterally. If the patient had returned with pain a quick re-examination (same as initial examination) would have been performed to test for reactivity and PRRT™ would have been used again. If the patient had not show any signs of improvement following use of the PRRT™ within 2-4 sessions (no decrease in reactivity or no lasting effects), it would no longer have been included in the plan of care. Subsequent treatments for the patient consisted of exercises to improve scapulo-humeral rhythm, proprioceptive awareness, strength and posture. The patient was provided with a thera-band and a home-exercise program (HEP) that included the majority of the strengthening and stretching he was completing in therapy.

Although this patient was making quick progress and was to be seen for four weeks, he was only seen for five visits total. He was independent in his HEP within three visits which was promising; however, he missed his third week due to a death in the family and his fourth week secondary to some type of infection that required hospitalization and antibiotics. Following these two missed weeks, he came to only one more therapy visit because he was feeling so good

and was independent in his HEP and essentially self-discharged. The patient was reminded that without complying with his exercise program to continue strengthen musculature required to maintain neutral posture, restore scapulo-humeral rhythm and arthrokinematics, his symptoms would likely return. The patient acknowledged understanding this.

In summary, at the conclusion of treatment the patient had full and pain-free ROM bilaterally and had >4/5 strength with all scapular/shoulder motions (see **table 4**). The patient's postural awareness had improved and he was able to maintain neutral posture for >15 minutes without cuing and no longer had limitation with cervical retraction. In addition, following the first two visits, the patient had not experienced the "numbness" symptom that was initially described.

DISCUSSION:

The purpose of this paper was simply to introduce a new technique into the realm of shoulder treatment. There is currently no literature regarding PRRT™ and only speculation into its mechanism of action. However, despite the lack of literature, there are positive anecdotal results being seen. PRRT™ does not appear to be a stand alone treatment, but does offer a new technique as an adjunct to traditional physical therapy. As in this case, PRRT™ appeared to allow for a quicker transition to pain-free strengthening which addressed the suspected root cause of this patient's symptoms. Again, how or why PRRT™ works is not fully understood and is only speculative at this time. However, if hypothesizing, if the increased reactivity was due to imbalanced gamma gain and resulted in increased tone, it is possible this was adding to the current problem of the secondary impingement syndrome. By abolishing the hyper-reactive

areas and resetting gamma gain, it may have allowed for improved awareness of movement/control, increased sub-acromial space secondary to decreased humeral superior translation, or decreased resistance to movement. All of which would/could result in decreased pain with elevation.

Future studies need to begin to delve further into the mechanism of action behind PRRT™ and complete comparative studies to attempt to establish validity and reliability data. Since PRRT™ appears to be best suited for decreasing pain, possibly pairing it against a modality such as electrical stimulation may be a good place to start.

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Table 1: Active rage of motion measurements for a 55 year old male with chronic shoulder pain.

	Left Shoulder	Right Shoulder
Flexion	WNL with pain (pain begins at 110')	WNL with pain (begins at 94') and (+) shrug sign
Abduction	WNL with pain (pain begins at 90')	WNL with pain (begins at 90') and (+) shrug sign
Internal Rotation	T7	T10 with pain
External Rotation	WNL	WNL

Table 2: Manual muscle test measurements for a 55 year old male with chronic shoulder pain.

	Left	Right
Shoulder Flexion	5/5	3/5 with pain and (+) shrug sign
Shoulder Abduction	4+/5 with pain	3/5 with pain and (+) shrug sign
Shoulder Internal Rotation	5/5	5/5
Shoulder External Rotation	5-/5	5-/5
Upper Trapezius	5/5	5/5
Middle Trapezius	4+/5	4+/5
Rhomboids	4+/5	4/5
Lower Trapezius	3/5	3-/5

Table 3: Accuracy, sensitivity, and specificity for acromioclavicular joint (ACJ) and shoulder impingement tests.

	Accuracy	Sensitivity	Specificity
O'Brien's active compression test	.92-.97[12] for ACJ pathology	.16-1.0[12] for ACJ pathology	.9-.925[12] for ACJ pathology
Cross-over impingement/Horizontal Adduction Test	.79[12] for ACJ pathology .48[11] for subacromial impingment	.77-1.0[12] for ACJ pathology .23[11] for subacromial impingement	.79[12] for ACJ pathology .82[11] for subacromial impingement
Neer Impingement Test	.68[11] for subacromial impingement	.46-.93[10,11] for subacromial impingement	.69[11] for subacromial impingement
Hawkins-Kennedy Impingement test	.70[11] for subacromial impingement	.62-.78[10,11] for subacromial impingement	.66[11] for subacromial impingement

Table 4: Summary of 55 year old male with chronic shoulder pain on initial versus at discharge in regards to ROM, MMT, and pain rating via VAS.

	Initial		Final	
STRENGTH	**Left**	**Right**	**Left**	**Right**
Shoulder Flexion	5/5	3/5 with pain and (+) shrug sign	5/5	4+/5
Shoulder Abduction	4+/5 with pain	3/5 with pain and (+) shrug sign	5-/5	4+/5
Shoulder Internal Rotation	5/5	5/5	5/5	5/5
Shoulder External Rotation	5-/5	5-/5	5/5	5/5
Upper Trapezius	5/5	5/5	5/5	5/5
Middle Trapezius	4+/5	4+/5	5-/5	5-/5
Rhomboids	4+/5	4/5	4+/5	4+/5
Lower Trapezius	3/5	3-/5	4/5	4/5
ROM	**Left**	**Right**	**Left**	**Right**
Flexion	WNL with pain (pain begins at 110')	WNL with pain (begins at 94') and (+) shrug sign	WNL (1/10 pain with full elevation)	WNL
Abduction	WNL with pain (pain begins at 90')	WNL with pain (begins at 90') and (+) shrug sign	WNL	WNL
Internal Rotation	T7	T10 with pain	T7	T10
External Rotation	WNL	WNL	WNL	WNL
PAIN RATING (VAS)	7-8/10 with over-shoulder level elevation		Occasionally 1/10 on left only with end range elevation	