CHIROPRACTIC CARE FOR A PATIENT WITH SPASMODIC DYSPHONIA ASSOCIATED WITH CERVICAL SPINE TRAUMA

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ABSTRACT

Objective: To discuss the diagnosis and response to treatment of spasmodic dysphonia in a 25-year-old female vocalist following an auto accident.

Clinical features: The voice disorder and neck pain appeared after the traumatic incident. Examination of the cervical spine revealed moderate pain, muscle spasm and restricted joint motion at C-1 and C-5 on the left side. Cervical range of motion was reduced on left rotation. Bilateral manual muscle testing of the trapezius and sternocleidomastoid muscles, which share innervation with the laryngeal muscles by way of the spinal accessory nerve, were weak on the left side. Pre and post accident voice range profiles (phonetograms) that measure singing voice quality were examined. The pre- and post-accident phonetograms revealed significant reduction in voice intensity and fundamental frequency as measured in decibels and hertz.

Intervention and outcome: Low-force chiropractic spinal manipulative therapy to C-1 and C-5 was employed. Following a course of care, the patient's singing voice returned to normal, as well as a resolution of her musculoskeletal complaints.

Conclusion: It appears that in certain cases, the singing voice can be adversely affected if neck or head trauma is severe enough. This case proposes that trauma with irritation to the cervical spine nerve roots as they communicate with the spinal accessory, and in turn the laryngeal nerves, may be contributory in some functional voice disorders or muscle tension dysphonia. (*J Chiropr Med* 2005; 4:19–24)

Key Indexing Terms: Chiropractic; Voice Disorders; Whiplash Injuries

INTRODUCTION

Human voice production involves the coordination of optimal vocal fold positioning and control of the airflow from the lungs to the oropharynx. Laryngeal function must be coordinated, efficient, and physiologically stable to produce a normal voice. Any imbalance in this delicate system affects vocal quality. When vocal quality deteriorates and examination shows no anatomic or neurologic etiologic factors, then benign spasmodic dysphonia (SD) or the subclassification of muscle tension dysphonia (MTD), should be suspected.^{1,2}

Functional voice disorders such as MTD account for at least 10% of the cases of dysphonia referred to multidisciplinary voice clinics. They occur predominantly in women, are frequently transient, and commonly develop after an upper respiratory infection, and psychological issues are frequently present. These voice disorders can be misdiagnosed because they can have multiple causative factors.^{1,2} When a patient's history revealed that a voice disorder had developed concomitant with neck trauma or whiplash, and considering the mutually shared innervation, manual muscle testing was viewed as a possible diagnostic or screening procedure and incorporated into the initial examination.

This case report examines the result of testing bilaterally, the trapezius and sternocleidomastoid (SCM) muscles, as a diagnostic indicator for possible laryngeal involvement in a case of muscle tension dysphonia. When these muscles were found to be weak unilaterally, cervical spine subluxation was suspected, and structural correction or spinal adjustment was attempted, and seemed to be successful when the symptoms of voice dysfunction were resolved.

CASE REPORT

A 25-year-old female speech-language pathology graduate student and church soloist, began to have difficulty turning her head to the left, and experi-

enced neck pain, stiffness, headaches and dizziness following an auto accident. She was seen immediately after the accident by emergency room staff, and diagnosed with a strained neck or whiplash associated disorder (WAD). Tylenol was recommended. When her symptoms persisted, she saw her family physician, who also prescribed pain medication.

After 8 weeks her condition had not improved. She then sought care from a chiropractor. She still had all of the above symptoms, as well as a clicking sensation in her left laryngeal area during jaw movement. Since the accident she had also noticed a partial loss of voice control, or break in register when singing, while still maintaining her normal speaking voice.

Orthopedic provocative and gross neurological tests were normal, except for a positive Romberg sign, which was found on the left side, and was consistent with the dizziness experienced by the patient following the accident. This finding indicated abnormal afferent innervation. Palpation of the cervical spine revealed restricted movement at C-1 and C-5 with hypertonicity and moderate pain on the left side. Cervical range of motion was limited to 40 degrees on left rotation.

Because the patient's history revealed that a voice disorder had developed concomitant with neck trauma and that some neck and phonatory muscles mutually share innervation, manual muscle testing was viewed as a possible diagnostic procedure and incorporated into the initial examination. Bilateral manual muscle testing³ elicited weakness of the left trapezius and sternocleidomastoid (SCM) muscles.

After a discussion about the effects of WAD on the spine, muscles and nerves, and how it may have affected her singing voice, she stated that the speech-language pathology course of study included participation in a voice assessment program, known as Voice Range Profile (VRP). She stated that her profile, taken before the accident, was on file at the department. A post-accident profile was performed prior to the commencement of treatment (Figs 1 and 2).

The profile is a reliable and valid⁴ diagnostic tool designed to help vocal clinicians better understand

voice registers, classifications and disorders, by establishing a vocalist range of intensity from the lowest to the highest fundamental frequencies. It is a display of vocal intensity versus fundamental frequency. It is plotted in linear or logarithmic units on an XY diagram. A primary feature of the display is the manner in which the range of intensity covaries with the range of fundamental frequency to create an area of characteristic shape.⁵

The characteristic shape of her pre- and post-profiles, as visually measured within the XY diagram, show the post accident VRP to be significantly reduced, and indicated potential vocal stress.

A treatment plan to structurally correct or reduce the suspected spinal subluxations or spinal joint lesions at C-1 and C-5 was initiated with the patient's consent.

Chiropractic specific spinal adjustment or spinal manipulative therapy was performed at C-1 and C-5, utilizing cervical diversified technique. The spinal adjustments were performed with the patient lying supine. The adjusting stance was from her left side and a low force thrust was delivered with the left hand contacting the left transverse process of C-1 and the left posterior arch of C-5. The low-velocity, variable-amplitude thrust was delivered appropriate for structural correction of the vertebral subluxation complex or functional spinal lesion, as was indicated when the affected muscles were strong when re-tested.

At each visit manual muscle testing was performed bilaterally and if the SCM or trapezius muscles were unilaterally weak, treatment was performed. Following treatment, the weak SCM and trapezius muscles were re-tested and found to be strong and functioning normally.

On follow-up visits, treatment was not performed if the affected muscles had remained strong. Over an 8-week period of 10 treatments, there was resolution of all objective findings and subjective complaints, including the return of her normal singing voice. As a point of interest, the positive left Romberg sign and her dizziness resolved as well. This finding may indicate a possible relationship between vertigo, WAD, and abnormal afferent input from

Figure 1. Pre-accident voice range profile. Intensity is plotted vertically as sound pressure level in decibels (dB), and horizontally as fundamental frequency in hertz (Hz).

neck muscles and reflexes that control muscle tone, head orientation, and posture.

DISCUSSION

Spasmodic dysphonia and muscle tension dysphonia are synonyms for functional voice disorder and consensus is lacking regarding their classification and etiology. Spasmodic dysphonia is found in cases with specific kinds of voice interruptions. There are a variety of diseases that produce laryngeal dystonia. For example, postural imbalance following head and neck injury, especially whiplash, can affect or cause compensatory vocal strain. Spasmodic dysphonia is also subclassified as adductor or abductor types.¹

Adductor spasmodic dysphonia is most common and is characterized by hyperadduction or sudden involuntary muscle spasm of the vocal folds producing an irregularly interrupted, effortful, strained,

staccato voice. It is generally considered neurologic and a focal dystonia.

Abductor spasmodic dysphonia is similar to adductor spasmodic dysphonia except that the laryngeal muscle spasms cause the vocal folds to open and not vibrate. The voice is then interrupted by breathy, unphonated bursts, rather than constricted and shut off. Both abductor and adductor spastic dysphonia usually progress gradually and are aggravated by stress. Mixed spasmodic dysphonia involves muscles that open the vocal cords as well as muscles that close them and therefore has features of both disorders.

In these types of spasmodic dysphonia, the voice may be normal or more normal during laughter, coughing, crying or other non-voluntary vocal activities, or during singing, but when speaking vocal breaks occur at the beginning of words.⁶

Because of the possibility of serious underlying neurologic dysfunction, a complete neurological and

Figure 2. Post-accident voice range profile.

neurolaryngeal examination is advised. Muscle tension dysphonia is seen as an excessive or "imbalanced" activity of the intrinsic and extrinsic laryngeal muscles affecting phonatory function, and is seen as benign in nature. Patients usually complain of neck and shoulder tightness, excess vocal effort and fatigue, with symptoms intensifying on extended use.² Many cases of MTD are also characterized by vocal breaks occurring randomly, with no apparent pattern.⁶

Various therapeutic methods are employed to treat spasmodic dysphonia or muscle tension dysphonia, including speech therapy, muscle relaxants, botulinum toxin injections,⁷ surgery and external manual laryngeal massage,² but they are not always satisfactory.¹

As noted, some voice disorder cases are linked to head or neck trauma. It should be of interest that functional spinal lesion, or vertebral subluxation

complex, are also factors commonly found in cervical spine trauma cases.^{8,9}

I believe that this patient experienced vertebral subluxation complex or functional spinal lesions at the C-1 and C-5 motor segments, mechanically disrupting the normal motor-reflex activity of the vagus (CN X) and spinal accessory (CN XI) nerves in their course and approximation of the upper cervical spine. The initial examination found hypertonic neck muscles and restricted movement at C-1 and C-5, indicating the possibility of abnormal joint function affecting the associated spinal nerve roots. Specifically, the spinal roots C-1 and C-5 of the accessory nerve (ramus externus), which innervate the SCM and upper trapezius muscles, could be involved. If the spinal roots to the spinal accessory nerve were involved due to mechanical irritation, treatment would allow the unilaterally weak muscles to function normally, relieving her symptoms.

This spinal nerve root irritation could also influence the accessory portion (ramus internus) of the spinal accessory nerve as it supplies, by way of the vagus and recurrent laryngeal nerves, the intrinsic muscles of the larynx (except the cricothyroid), which is innervated by the superior laryngeal nerve. Research also indicates that the thyroarytenoid laryngeal muscle can also be cross-innervated by the external division of the superior laryngeal nerve,¹⁰ and could be a factor.

A non-traumatic case of vertebral subluxation complex or functional spinal lesion, and its relationship to spasmodic dysphonia, is cited in 1 of 2 cases in the literature.¹¹ The author states that vertebral subluxation complex may affect axoplasmic transport, impulse conduction, and neurocellular morphology.¹² This axonal compression, neurobiological and neurochemical reaction can compromise the motor efferents of the vagus-recurrent laryngeal and accessory nerve complex.

This paper offers the possibility that, manual muscle testing may show that innervation to the trapezius, SCM, and intrinsic laryngeal muscles can be mechanically irritated, allowing unilateral weakness of these muscles. Spasm of the opposing trapezius and SCM muscles, with hyperadduction of the opposing vocal cords or intrinsic laryngeal muscles, may follow. Voice disorder SD/MTD, painful and fatigued neck muscles can then be a sequelae. The vagus parasympathetic nerve to the diaphragm can also be involved, affecting overall voice control.¹³

Following neck injury or trauma, and if indicated, specific spinal adjustment applied to the cervical spinal joint dysfunction, or vertebral subluxation complex, may reduce mechanical irritation to the spinal nerve roots of the spinal accessory nerve, and in this case, appeared to allow normal innervation of the affected laryngeal, trapezius and SCM muscles. The opposing intrinsic laryngeal and cervical muscles that are hyperadducted, or in compensatory spasm, perhaps return to normal, alleviating symptoms of muscle tension dysphonia or voice disorder and neck and shoulder pain.

It should be noted that spontaneous remission can occur in some MTD cases, but if found to be persistent, they are generally viewed clinically as being musculoskeletal with various causations. This excessive muscle activity has been attributed to many sources, including 1) psychological and/or personal-

ity factors that tend to induce tension, 2) technical misuses of the vocal mechanism in the context of extraordinary voice demands, 3) learned adaptations following an upper respiratory tract infection, and 4) compensation for underlying benign disorder.²

It appears that vertebral subluxation complex or functional spinal lesion following cervical spine trauma may have disrupted the spinal roots C-1 and C-5 of the spinal accessory nerve, especially the accessory portion or (ramus internus) in its innervation of the laryngeal muscles unilaterally, allowing compensation, or hyperadduction of the opposing vocal folds. This may have resulted in the patient's inability to sing with the same intensity or voice sound pressure level as the post-accident voice range profile would tend to indicate.

Given the favorable response to specific chiropractic spinal adjustments in relation to muscle tension dysphonia, the need for more research is indicated. A study investigating the possibility that a voice range profile could be effective in determining a link between vertebral subluxation complex due to cervical spine trauma and muscle tension voice disorder would be of interest. Incorporating surface electromyography into this research study might also help to establish objectivity when testing for abnormal muscle activity of the trapezius and SCM muscles.

CONCLUSIONS

With the possibility that cervical spine trauma, due to personal injury or auto accident, and certain voice disorder cases could have a common link, the question is whether mechanical irritation to the cervical spine can negatively impact the vagus and spinal accessory nerves as they innervate the intrinsic laryngeal muscles and the upper trapezius and SCM muscles.

This case report suggests that specific chiropractic spinal adjustment, with its efficiency and safety,¹⁴ may be a therapeutic alternative not previously considered in cases of trauma associated SD/MTD, and that manual muscle testing may be a valuable aid to diagnosis and treatment in these cases.

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