Trans-Cricothyroid Injections for Intractable Mutism

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Abstract

Objectives: To discuss the relationship between the physiologic and psychologic control of laryngeal function.

Study design: Single subject retrospective research investigation.

Methods: Here we present the case history of a highly intelligent, normally developing teenager from a middle-class family background who suffered idiopathic recurrent bouts of mutism, which were originally and erroneously diagnosed as manifestations of a paradoxical vocal fold movement disorder. The patient was refractory to standard respiratory retraining and voice therapy exercises. Based on our previous experiences and research with other patients, we employed trans-cricothyroid Lidocaine and saline injections on separate occasions to restore functional voice control.

Discussion: The physiology of normal voice production depends upon discreet integration of complex cortical, auditory, sensori-motor, and end organ activities. There is a subset of patients with non-organic dysphonia who exhibit respiratory and laryngeal muscle hypertension as a consequence of underlying psychological disequilibrium. Voice difficulties in this clinical population may range from intermittent harsh vocal quality to protracted aphonia. Lidocaine injection after the first and second occurrence of mutism and saline injection after the third manifestation had immediate and demonstrably positive results following each treatment session. Alternative physiogenic versus psychogenic explanations for these outcomes are presented.

Conclusion: There is a complex and poorly understood relationship between the physiologic and psychologic factors leading to conversion reaction mutism. We found that disruption of a potentially dysfunctional sensori-motor feedback loop, via trans-cricothyroid injections, quickly resulted in return of normal voice.

INTRODUCTION

The larynx facilitates numerous critical biological, automatic, and reflexive functions, such as airway patency, airway protection, and tracheal-bronchial clearance. All mammals have the innate voluntary ability to drive and control vocal fold vibrations to generate both voiced and voiceless sounds, which can be used to communicate thoughts and feelings. Humans possess advanced oropharyngeal anatomical and neuromuscular controls to convert these sounds into articulated words for the purposes of speech production. Intelligible speech depends upon the coordinated balance of multiple muscular subsystems, including those of the respiratory, phonatory, articulatory, and resonatory mechanisms. These principal biomechanical activities are regulated by complex cognitive, linguistic, and psychological processes, which are largely responsible for word choice and speech intonation patterns that uniquely differentiate speakers from one another. Several clinical researchers have suggested that the larynx is the window to the soul via audible expression of emotions; that powerlessness and silence are interrelated [1-9].

The mechanism of normal voice production has been the subject of extensive research for nearly 200 years since the pioneering inquiry by Manuel Garcia. Sensori-motor integration is unquestionably essential for all of the aforementioned laryngeal functions. Histologic examinations have revealed abundant nerve endings and mechanoreceptors within the supraglottic larynx to the vocal folds, contained within the superior laryngeal nerve; afferents from the subglottis travel via the recurrent laryngeal nerve. Afferent signals traveling from the receptors in the supraglottis to the vocal folds are contained within the superior laryngeal nerve; afferents from the supraglottis travel within the recurrent laryngeal nerve. Sensory fibers, with their cell bodies located in the inferior jugular
ganglion, terminate in the solitary nucleus within the medulla. Polysynaptic connections are then formed between sensory fibers, terminating in the solitary nucleus, and efferent motor neurons of the laryngeal muscles, located within the nucleus ambiguous of the medulla. These communicating pathways form the servo-reflex system of the larynx [13-14]. Additionally, afferents from mechanoreceptors within the laryngeal muscles themselves create a myotatic reflex, which responds to and balances sensory output. Lastly, although the mechanism has not yet been fully elucidated, auditory feedback is believed to play a critical role in vocal accuracy and fundamental frequency variations associated with normal intonation patterns during extemporaneous speech [3].

Disturbances in voice production can result from many different isolated or interrelated pathological conditions. In some cases, the resultant dysphonia may be mild without compromise to speech intelligibility. In others, the problem may be more advanced with notable negative impact on the ability to communicate effectively. In each case, the underlying etiology of dysphonia may be functional or organic; either due to abnormal speaking behaviors with no known associated pathologies, or to observable lesions, diseases, or injuries involving any of the aforementioned speech subsystems or their neurologic substrates. There is an unusual subgroup of individuals who present to otolaryngologists with histories suggestive of psychogenic dysphonia. That is, the voice difficulties have no associated endoscopic evidence of laryngeal pathology, and they may range from intermittently hoarse vocal quality to protracted aphony. On a very rare occasion a patient may present with mutism; the inability to generate audible speech, not even a whisper. This profound communication disorder is almost always attributable to a deep seated emotional struggle, manifested via conversion to a form of laryngeal lockdown [15-19]. Under this subconscious disconnected condition, discussion about the psychological state of mind can be avoided by the patient. In turn, such avoidance behavior may cause cyclic reinforcement of the disorder and coexisting feelings of helplessness. Diagnostic terms like muscle tension dysphonia, hysterical or conversion reaction dysphonia, psychosomatic or psychogenic dysphonia, non-organic dysphonia, and functional dysphonia have all been used extensively and interchangeably to categorize patients who present with significant voice difficulty without definitive or observable explanations.

The synonymous diagnostic terms “paradoxical vocal fold movement disorder” and “vocal cord dysfunction (VCD)” have received substantial attention in the scientific voice literature over the past decade [20-23]. The typical clinical presentation is that of recurring, yet episodic voice and breathing difficulties. Intermittently abnormal adduction of the vocal folds during inspiratory activities results in gasping sensations and substantial emotional and physical stress. Episodes of transient dyspnea may coexist, characterized by bursts of laryngeal stridor, strained vocal quality, and limited pitch and volume control. Not infrequently, with such occurrences patients seek immediate medical attention for relief. The presumed, but faulty diagnosis is often asthma, with no notable improvement in symptoms with standard pharmacotherapy or breathing treatments. If and when laryngoscopy and bronchoscopy are performed the results are not conclusive for an underlying contributing pathology; the default diagnosis of VCD is usually rendered thereafter with recommendations for respiratory retraining exercises and voice therapy.

We report a case of a very bright, well-mannered teenager who was referred to our voice center by a pulmonologist who had rendered 3 months of unsuccessful treatments for the presumed diagnosis of asthma. At the end of this treatment period he turned his attention to the possible diagnosis of VCD, largely due to the failures of standard asthma care. During the last two months of this treatment course the patient was persistently and completely mute, despite an intervening comprehensive voice therapy program, which included inhalatory and exhalatory retraining exercises, both forceful and easy onset voice production techniques, laryngeal massage, audible cough prolongation methods, and humming strategies. This report describes in detail the unusual medical history and extraordinary repetitive therapeutic and medico-surgical interventions that we employed to restore voice and speech ability.

**METHODS**

This is a single subject retrospective research investigation of a well-developed 15 year old patient. At the time of this report, the patient was an honor student, a competitive swimmer, and a talented French horn musician. Past medical history included 1) an atrial septal defect, which was surgically repaired at the age of three years, 2) recurrent shoulder pain, and 3) allergic rhinitis. Approximately one year ago the patient presented to a pulmonologist with complaints of chronic dry cough, pleuritic chest pain, shortness of breath, intermittent wheezing, gasping for air during swimming activities, and globus sensations. There were no known antecedent medical or environmental triggers. Referral was made to a pediatric otolaryngology colleague whose clinical and laryngoscopic findings suggested normal anatomy and physiology of the larynx. The pulmonologist empirically prescribed Albuterol (p.r.n), with the presumed diagnosis of adolescent onset asthma. The patient administered this medication at least three times daily for six weeks with no noticeable improvements in symptoms. Throughout this period of time, there were no associated speech or voice difficulties.

Pulmonary function testing was conducted two months after the patient’s initial diagnosis of juvenile asthma; use of albuterol ceased one week prior to this evaluation. The bronchodilator response elicited during testing supported the earlier diagnosis of asthma. Two puffs of Qvar (40mcg) B.I.D. was prescribed in lieu of the albuterol. Additionally, Omeprazole (20mg) once daily was prescribed to address the globus sensation, with the presumptive clinical diagnosis of laryngopharyngeal reflux, though no signs of this condition were observed during the aforementioned laryngoscopy examination. One month later (i.e, three months after the initial examination by the pulmonologist), the patient reported both mild breathing improvements during exercise and less noticeable globus sensations. However, the dry hacking cough persisted throughout this treatment period. The medication regimen was maintained for the next three months.

Six months after initial presentation to the pulmonology service, the patient suffered an acute upper respiratory infection
accompanied by a moderate to severe bout of laryngitis with associated odynophagia, ear pain, and hoarse-breathy dysphonia. An appropriately dosed penicillin-based antibiotic was prescribed for 10 days along with a strict vocal hygiene regimen. With the exception of the voice difficulty all other signs of infection subsided markedly within five days. Approximately 10 days after the onset of the original signs and symptoms, the patient became aphonie. Within a couple of days thereafter neither whispering nor voluntary oral airflow was possible; the patient was essentially frozen with respect to normal physiologic requirements of aerodynamic control for speech and voice production. As a consequence, communication was achieved via mouthing words, written messages, gesturing, or various combinations of these techniques. It is noteworthy that notwithstanding this profound communication disorder, all biologic laryngeal functions were normally preserved, including maintenance of airway patency during rest and physical exertion, airway protection during swallowing, and coughing ability.

Repeat laryngoscopy by another otolaryngologist demonstrated no discernable anatomic explanations for the patient's mutism; at rest, no vocal fold lesions were identified. Upon request to generate voice and speech behaviors during the laryngoscopy examination the patient was unable to produce any audible sound. The vocal folds moved only slightly toward the midline during each effort. A large glottal chink prevailed, and any airflow dynamics were imperceptible. It was as if the vocal folds were mechanically frozen in the abducted position. Moderate hypertonic contractions of the strap musculature of the neck were notable during this examination. Interestingly, involuntary vocal fold coughing behaviors were repetitively provoked when the tip of the flexible scope was deliberately pressed against the lingual surface of the epiglottis. No paradoxical VCD motion artifacts were observed during a variety of breathing maneuvers. All of these findings were consistent with the diagnosis of psychogenic conversion aphonia or mutism. The aforementioned comprehensive voice therapy program was then initiated, at two sessions per week. One month later, with persistence of these signs and symptoms, the patient was referred by the treating pulmonologist to our Voice Laboratory at the Detroit Medical Center for another opinion. Because causally related psychological or emotional factors were not yet being considered in the differential diagnosis, referral to a clinical psychologist or psychiatrist was delayed pending the scheduled voice lab results.

The patient presented with a parent, and there was no observable friction between them. The aforementioned characteristics of mutism were exhibited; neither voluntary voice nor whispering was possible. Attempts to blow a pinwheel were unsuccessful as well. Both swallowed and voluntary coughing abilities were unimpaired. Independent examinations by a board certified otolaryngologist and a Ph.D. level speech-language pathologist, with more than 30 years of experience evaluating vocal pathologies, was next conducted using a 70 degree pathologist, with more than 30 years of experience evaluating certified otolaryngologist and a Ph.D. level speech-language pathologist that such treatment has been found to be quite helpful in other cases with similar speech difficulties. Consistent with extensive reports in the voice therapy literature for both muscle tension and conversion dysphonias, the intervention applications included 1) laryngeal massage, 2) use of a stethoscope for biofeedback of phonation from the examiner, 3) non-speech inhalatory-exhalatory coordination exercises, with focus on diaphragmatic support for upstream airflow regulation, 4) various easy onset voice techniques, 4) gentle humming activities, and 5) prolongation of cough into continuous voice attempts, using video endoscopic biofeedback of the patient's laryngeal inlet and vocal folds during this technique [24-26]. This exercise program was very similar to that which was unsuccessfully attempted prior to the patient's visit with us.

Both the attending speech therapist and the otolaryngologist independently concluded that the patient was completely unresponsive to these conservative therapeutic techniques. Discussions were then rendered about additional potential treatments, including any one or more of the following standard or experimental alternatives: 1) psychotherapy, 2) continuation of the aforementioned standard voice therapy program, and 3) Lidocaine or saline injection directly into the subglottic space either to anesthetize the laryngeal interior or induce a deliberate placebo effect, as previously described for the treatment of patients with intractable MTD [27-29]. Neither the patient nor the parent was interested in the first or second treatment recommendations; they preferred to proceed with the experimental option of laryngeal injection after learning that the actual technique is usually minimally discomforting and takes about 1 minute to perform. Consent was obtained from the patient and parent to proceed with either or both of the laryngeal injection procedures noted above, at our administration discretion. After alcohol swabs to the neck, the cricothyroid membrane location was marked with a surgical pen in preparation for the injection. We arbitrarily elected to place 5cc of 4% topical Lidocaine into a 10cc syringe attached to a 26 gauge needle. Once the cricothyroid membrane was pierced and the needle entered the subglottis space, as determined by aspiration of air into the syringe, 1cc increments of the solution was injected every 5 seconds until the entire amount was released, as planned. As expected, each such
dose induced vigorous coughing behavior by the patient, which in turn distributed the solution into the laryngeal inlet for topical anesthesia purposes. The entire injection procedure lasted less than 1 minute, with no reported discomfort by the patient. Next, easy onset voice exercises and humming techniques were quickly reintroduced to stimulate phonation output. Within 2 minutes the patient was able to generate near normal voice characteristics. Two minutes later, with continuous voice practice via conversation, normal voice and speech skills were recovered. Follow-up phone calls one month later revealed that the patient was doing well, with no recurrence of voice or speech difficulties.

Two weeks after the follow-up phone conversation the patient suffered a relapse of the mutism condition and returned to our Voice Center. There were no known identifiable triggers, with the possible exception of the stress of impending midterm examinations. We followed the same evaluation methodology that was initially employed for definitive diagnosis and treatment. The patient was mute and unable voluntarily to initiate voice or verbal speech through whisper. Video stroboscopic examination results were identical to the original findings two months earlier. Another stint of voice therapy was administered with hopes of a clinical breakthrough, to no avail. Both the patient and parent requested treatment with one of the experimental injections, as described earlier. Once again, without forewarning about which solution was chosen, we elected to administer Lidocaine rather than saline. As with the previous injection, the patient’s response was immediate and dramatic, with no reported discomfort. Voice and speech ability were restored to normal levels within 2 minutes post-injection. The patient was discharged and instructed to 1) seek the counsel of a clinical psychologist or psychiatrist to help resolve possible causally related emotional conflicts or anxiety, 2) try to become more philosophical about stresses in her life, and 3) consider the art of Yoga or meditation for general body relaxation exercises.

Over the course of five months following this last treatment session, we maintained correspondence via email to keep abreast of the patient’s status. Reports indicated no communication setbacks, notwithstanding the fact that none of the recommended treatments or exercises were employed during this post-treatment time frame. In concert with the pulmonologist’s guidance the patient continued using Qvar and Omeprazole p.r.n.

One month later, approximately 10 months since our originally evaluation, the patient contracted a moderately severe upper respiratory infection with associated left otalgia,odynophagia, and laryngitis. Antibiotic therapy, a Medrol dose pack, and Flonase for nasal congestion were prescribed by the ER attending. On day 7 of this condition, most of these symptoms subsided substantially. However, the dysphonia converted into aphonia. On day 8, the patient became mute, unable to vocalize or whisper. Upon presentation to our Voice Center the patient exhibited the exact same communication deficits as observed the previous two visits to our clinic. Videostroboscopy of the larynx demonstrated normal tissue appearances; no signs of laryngitis were detected. Strap muscle hypertonicity was noted during faulty phonation attempts. There were no signs of VCD during various vigorous breathing activities. These findings supported our original hypothesis that the patient’s recurring history of mutism was likely of non-organic origin, and for unclear reasons was not responsive to behavior modification voice or breathing exercises in the past. The patient elected to undergo another laryngeal injection in lieu of an initial stent of voice therapy. Whereas on the two previous treatment occasions we selected Lidocaine for the laryngeal injections, this anesthetic solution did not offer unequivocal diagnostic information for more definitive clinical counseling of the patient. In view of this fact, and because there are no unequivocal standard treatments for this rare speech disorder, on this treatment occasion we elected to use a 5cc solution of .9% injectable sterile sodium chloride with expectations of a possible placebo effect. As with the two previous injections with Lidocaine, neither the patient nor parents were aware of which of the two experimental solutions were chosen. The procedure took less than 1 minute to perform, with no reported discomfort by the patient. Immediately post-injection the patient experienced restoration of voice and speech abilities to normal levels, without therapeutic prompts from the treatment team. Discussions were rendered about this placebo effect and the clinical implications thereof. At the time of this writing, 4 months post-injection, the patient continues to enjoy consistently normal communication behaviors.

**DISCUSSION**

A case study is distinct from experimental research. In general, it lacks methodological control of extraneous variables that may account for the findings observed. Notwithstanding this notable limitation, case study investigations can offer important information to the scientific literature. Although they do not necessarily test hypotheses, unique cases can direct future research inquiry on larger population bases with similar clinical histories. In this vein, we presented a very interesting adolescent patient with a bizarre recurrent history of mutism. The treating pulmonologist formed an initial diagnosis of profound VCD, secondary to various asthmatic sequelae, including chronic cough, wheezing, dyspnea, and potential steroidal use laryngeal irritability. The literature is replete with investigations linking asthma and VCD to common pathognomonic mechanisms with many parallel and interrelated symptoms [20-22]. The patient’s referring otolaryngologists suggested that the mutism behavior was likely the most extreme form of MTD he had ever observed. In MTD there is inappropriate recruitment of accessory muscles leading to vocal strain, breaks and fatigue, change in fundamental frequency, sore throat, and visible cervical muscle tension. Aberrant laryngeal sensation leading to vocal cord dysfunction may stem from LPR, URI asthma, smoking, high stress, excessive voice use, and poor breath support [23]. The referring otolaryngologist did not consider conversion reaction in his differential diagnostic algorithm. During his exam, and later corroborated in our voice laboratory, the patient exhibited observably pronounced hyperactivity of the ventricular vocal folds and strap musculature of the neck during all futile efforts to phonate. These behaviors were probably compensatory to overcome profound tonic contractions of the true vocal folds, which rendered these structures frozen in the abducted position throughout the endoscopic voice examination. Consistent with the psychogenic diagnosis of MTD was the patient’s involuntary and consistently normal approximation of the true vocal folds, firmly at the midline of the glottis, during stimulus induced

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coughing and swallowing. Based on the presenting symptoms, signs, and history, this diagnosis was also at the top of our list of possible non-organic etiologies. Such suspicions supported our recommendation to attempt Lidocaine injection into the immediate subglottic space after the patient failed to respond to voice therapy measures. We previously reported excellent and reliable results with this treatment technique in other patients with therapeutically intractable MTD [27,29].

The results of this investigation have literally produced solutions in search of answers. We postulate two alternative but likely inter-dependent theoretical explanations for our patient’s complete responses to Lidocaine to anesthetize the larynx and saline to evaluate a placebo effect: 1) a bonafide sensori-motor integration and cortical re-regulation response, secondary to the induced laryngeal anesthesia, and/or 2) a psychological or emotional emancipation effect, because of the theater of the clinical operating suite and the perceived definitive medical intervention. Each of these possible explanations will be discussed below.

A sensorimotor response?

Deafferentation research has demonstrated that overall gross voice motor skills are unimpaired under conditions of deliberate laryngeal anesthesia [30-32]. These experimental findings were supportive of the long-standing hypothesis that experientially ingrained volitional voice behavior is an "open-loop" phenomenon that is generally independent of continuous sensory feedback for successful production [33]. In essence, these clinical researchers have demonstrated that underlying voice ability is not significantly threatened if the sensory arm of the complex neuromuscular feedback loop of the phonation subsystem is temporarily disrupted. This well-documented finding not only lent support to our investigatory use of topical Lidocaine to desensitize the larynx and disrupt potentially aberrant sensori-motor feedback operations in other patients in the past [27,29], but it also contributed to employing this procedure with the current patient. As we previously reported, the exact mechanism of action responsible for the immediate improvements in phonation control following the Lidocaine injection is not clear. We believe that this topical anesthetic bath may act on the laryngeal and tracheal mucosal mechanoreceptors to impoverish sensory feedback during phonation attempts. Under this condition the patient may be able to experience a break in the possible vicious cycle of hyper functional laryngeal and cervical musculature contractions that contribute to the associated dysphonia. This so-called sensory trick may support initial gains in voice motor control, which in turn enables the patient to "re-set" the larynx to premorbid vocal fold position, length, and tension levels required for more natural voice production. If voice fluency and control is jump started through this rehabilitation technique, perhaps respiratory-phonatory subsystem reintegration promptly begins, activated in part by associated muscle memories. In the current patient, as with our previously treated patients, the quick voice improvements may have occurred solely as a consequence of these potential neuromotor speech remodeling processes. With continued practice and satisfactory rehearsal of voice and speech behaviors thereafter, perhaps communication balance was regained. What

insidiously weakens this particular treatment effect theory is the fact that the patient demonstrated precisely the same response following injection of a placebo solution, which could not have had any medicinal beneficial impact on performances of a proposed aberrant laryngeal servomotor mechanism.

A psychological response?

To support this conclusion it was first necessary to establish a definitive diagnosis and etiology. Without addressing these variables, our extrapolations of the findings would not have been productive. Based on the patient’s age, overall good health, generally unremarkable vocal history, mutism features, and videostroboscopy observations we felt reasonably confident that the recurring communication disorder was likely the result of an aberrant conversion response. What corroborated this presumed diagnosis was the actual treatment effect we obtained with placebo injection; that is, prompt recovery of normal voice and speech ability. Because our recommendations for psychological evaluations and treatments were shunned as unnecessary by the patient and parents, we unfortunately do not have a corroborated diagnosis of a conversion disorder. This omission presented an unavoidable challenge to our hypothesis of a psychological disorder. Notwithstanding this notable limitation, the robust and unequivocal placebo effect helped to minimize our concern about possible misdiagnosis. At the close of each treatment session we pressed the patient and parents to schedule an official psychological evaluation for the purposes of ensuring an accurate diagnosis and exploring the beneficial role of psychotherapy in reducing possible mutism recurrence in the future. The literature is replete with investigations on the important role of psychological counseling in young patients with selective mutism, which is considered by many clinical researchers to be a manifestation of an underlying anxiety disorder [34-36]. Although we cannot unequivocally substantiate this emotional association in our patient, we recognize the possible causal interrelationship between this common factor and mutism variants.

Freud described a conversion reaction as a sign of repressed energy that ultimately is transformed into variable sensory, motor, or visceral symptoms, without an underlying organic cause [37]. Causal factors of somatoform mutism may include repressed sexual energy, emotional conflicts, and subconscious fear of possible humiliation or embarrassment if the "wrong" things are said. Whereas conversion mutism has not been discussed extensively in the vocal pathology literature because it is a very rare condition, conversion aphony has been encountered and described by countless otolaryngologists and voice therapists [2, 4, 6, 9]. In this clinical population, the abilities to phonate and speak normally are neither intentional nor feigned, and they are frequently exacerbated by intense psychological or physical stress. In some cases, excessive shyness and a genetic predisposition to anxiety disorders, owing to complex family histories, trigger vulnerability to the development of this condition. When the patient with a conversion disorder receives notable attention and sympathy from others, these responses usually reinforce the emotional need for such secondary gains. Historically, conversion reaction has also been classified as a form of hysterical neurosis. The objective of psychotherapy as the primary form of treatment
is to help the patient resolve causally related conflicts. The goal of voice therapy is to rehabilitate normal integration of the respiration and phonation speech subsystems for the production of effortless, fluent, and intelligible voice and speech behaviors. In most cases such therapeutic intervention is successful. However, there is a subset of individuals, not unlike our patient, who are refractory to standard conservative therapies. Although reasons for such treatment failures often remain elusive, at some point this clinical population may require alternative more aggressive intervention techniques to restore communication abilities. Not only will the timing of such decisions vary from case to case, so too will the treatments of choice. For our patient, we opted to intervene with laryngeal injection immediately after failed responses to various voice therapy techniques; similar therapeutic failures in the past were also supportive of this treatment decision. For others with MTD or conversion aphonias who demonstrate even subtle improvements with voice therapy, psychological counseling, or both, we usually continue with such treatments until and unless no further gains are appreciated and the dysphonia remains disabling. At that time, we recommend considering an injection procedure for potentially definitive reversal of the problem, followed by a brief stint of behavioral intervention to reinforce the anticipated gains.

In the current patient, the actual trigger responsible for the recurrent bouts of mutism remains elusive. Contrary to the aforementioned descriptions of a conversion disorder, our young patient had no history of perverse sexual experiences, behavioral inhibitions, social anxiety, or other notable emotional turmoil that may have materialized into a phobic state of mutism. What’s more, until the past year, the speech communication history has been completely normal. Neither the referring physicians nor the parents reported that the patient had ever suffered underlying anxieties, fears, or neurologic trauma that would provoke prolonged selective or involuntary inability to speak. Rather, all of these individuals described the patient as a very intelligent, articulate, motivated, athletic, and well-adjusted individual, with many friends and extracurricular interests. We continue to contemplate whether our patient’s honor roll status and extreme drive for academic success, enthusiastically cheered by loving parents, may have induced a level of performance anxiety and psychological disequilibrium that eventually lead to somatization. [38]. This process is known as the “transmutation of energy,” wherein there is a tendency to break stride in the rigorous pursuit of success owing to subconscious physical and emotional disassociation from pressure packed or difficult situations [5]. Mutism in our patient’s case may have been a conversion reaction to the inability to cope with fearful feelings that emerge during certain confrontational speaking situations. Because this condition eliminated the need to talk about anything with anyone, perpetual relief from such fears may have been gained as a consequence of the disorder. Curiously, two of the three bouts of mutism developed shortly after the patient suffered from symptoms of a severe URI, with notable signs of laryngitis, otalgia, and rhinorrhea. Though on each occasion most symptoms resolved within one week with various prescribed medications, the laryngitis converted into a state of mutism. Combined with usual life stresses and the inherent high need achiever personality, this coexisting confluence of factors may have been sufficient to produce the perfect storm; a somatoform reaction. The patient was refractory to respiratory retraining and voice production biofeedback exercises. The parents were quite concerned, and pressure was mounting at home and at school for to speak normally so that focus could be placed on daily routines without the stigma of being mute. We suggest that because of all of these factors the patient was emotionally primed to get better at the time of our first evaluation and treatment session. In addition, our success initially may have been partially attributable to the fact that the referring physicians effectively pumped the patient with psychological reassurances about our excellent track record helping others with similar types of communication disorders.

In retrospect, it is difficult to know with certainty whether or not our patient would have responded as favorably to saline had we used this solution instead of Lidocaine at our first treatment session. As described above, perhaps the anesthetic effect was indispensable initially to induce physiologic sensorimotor rebalancing at the outset; analogous to the importance of applying primer paint before any other application for successful repair of a severely damaged wall. The optimal timing of lidocaine or saline injection for patients with psychogenic muscle tension or conversion reaction dysphonias cannot be answered without prospective, blinded, research studies.

CONCLUSION

It should be acknowledged that the results of this case study are bound by three important limitations. First, the treatment outcomes are based on a single case experience. Replication with many other patients who present similar communication difficulties is necessary to substantiate the validity of the procedures employed, results obtained, and explanations rendered. Second, corroborative diagnosis of conversion or selective mutism by a pediatric psychiatrist was not achieved. Third, psychotherapy was never attempted as a definitive line of treatment, in lieu of our treatment procedures. As a consequence, it remains unclear whether or not the patient would have responded as favorably to such counseling. To address these paramount concerns, we are in the process of designing a between group factorial research design methodology for comparative analyses of four different independent variables relative to initial treatment outcomes: Lidocaine injection, saline injection, traditional voice therapy techniques, and psychological counseling. Results of this anticipated investigation may help establish more definitive intervention algorithms for these complex and multi-varied clinical populations.

REFERENCES

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