To promote awareness and understanding among brass musicians of focal task-specific embouchure dystonia (FTSED)—a misunderstood and debilitating occupational disorder—a comprehensive review of literature from prominent medical, scientific, and musical sources was undertaken to determine the definition, causes, and treatment of the condition. A case study was also included with the intention of providing a source for reference and exploring possible pedagogical influences on FTSED. FTSED can be defined as a neurologically-based movement disorder characterized by abnormal random or sustained involuntary muscle contractions, initiated during playing, that cause embouchure dysfunction. A muscle spasm that occurs in a trumpet player’s lips when attempting to start a note, a horn player’s inability to sustain a tone without a rapidly shaking embouchure, or a tubist’s jaw inexplicably clamping shut when attempting to play octave leaps are all examples of FTSED.

Chapter One contains a brief introduction and definition of FTSED, a discussion of embouchure terminology and function, and an overview of key neurological concepts. Chapter Two includes further investigation of the definition of FTSED, with consideration of the causes, symptoms, diagnosis, and treatment of the disorder. Chapter Three presents summations of empirical studies of FTSED and similar dystonias, while Chapter Four presents summations of case studies of musicians with focal dystonia. The final chapter includes a summary of key points, suggestions for future research, and guidelines for recovery. A case study of FTSED is contained in the appendix, including
specific details of the initial appearance and progression of symptoms, pre-diagnosis symptom management strategies, a week-long intensive re-training program, and subsequent methods and routines leading to a return to public performance.

The exact causes of FTSED are unknown and current treatment options provide only minimal benefits. Often career-ending, FTSED has no known cure and medical research and insight with regard to the disorder are limited. Additionally, trends in brass pedagogy may contribute to the development of embouchure dystonia. Despite the minimal reports of successful long-term outcomes in clinical studies, recent findings indicate that FTSED may, in fact, be treatable and preventable, yet research must be undertaken to test such assertions. Improving the prognosis for FTSED and facilitating rehabilitation necessitates increased awareness among performers and teachers, a re-thinking of brass pedagogy, and the development and testing of effective treatment programs.
THE EFFECT OF FOCAL TASK-SPECIFIC EMBOUCHURE DYSTONIA UPON BRASS MUSICIANS: A LITERATURE REVIEW AND CASE STUDY

by

Seth David Fletcher

A Dissertation Submitted to the Faculty of The Graduate School at The University of North Carolina at Greensboro in Partial Fulfillment of the Requirements for the Degree Doctor of Musical Arts

Greensboro
2008

Approved by

Committee Chair
© 2008 by Seth David Fletcher
To Allison. Your patience, persistence, and eternally transcendent sense of humor are inspiring. I love you dearly. And to all those afflicted with focal dystonias. The human spirit has no boundaries that are not self-imposed.
APPROVAL PAGE

This dissertation has been approved by the following committee of the Faculty of

The Graduate School at The University of North Carolina at Greensboro.

Committee Chair

Committee Members

March 24, 2008
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GLOSSARY


Autonomic Nervous System B: Component of the peripheral nervous system responsible for the control of involuntary muscles and those bodily functions that are not consciously directed, including regular beating of the heart, intestinal movements, sweating, salivation, etc.

Axon B: A nerve fiber extending from the cell body of a neuron carrying nerve impulses away from it. An axon may be over a meter in length in certain neurons.

Basal Ganglia A: An area deep inside the brain that is believed to play a major role in the coordination of voluntary muscle movement. The basal ganglia are a group of structures that include the globus pallidus, thalamus, and subthalamic nucleus.

Brainstem B: The enlarged extension upwards within the skull of the spinal cord, consisting of the medulla oblongata, the pons, and the midbrain. The pons and medulla are together known as the bulb, or bulbar area. Attached to the midbrain are the two cerebral hemispheres.

Central Nervous System A: The brain and spinal cord.

Cerebral Cortex B: The intricately folded outer layer of the cerebrum, making up 40% of the brain and composed of an estimated 15 thousand million neurons. This is the part of the brain most directly responsible for consciousness, with essential roles in perception, memory, thought, mental ability, and intellect, and it is responsible for initiating voluntary activity. It has connections, direct or indirect, with all parts of the body.

Cerebellum B: The largest part of the hindbrain, located behind the pons and the medulla oblongata and overhung by the occipital lobes of the cerebrum. The cerebellum is essential for the maintenance of muscle tone, balance, and the synchronization of activity in groups of muscles under voluntary control, converting muscular contractions into smooth coordinated movement.

Dendrite B: One of the shorter branching processes of the cell body of a neuron, which makes contact with other neurons at synapses and carries nerve impulses from them into the cell body.
Dyskinesia\textsuperscript{A}: A general term to describe any kind of involuntary muscle movement.

Dystonic / Dystonia\textsuperscript{A}: Dystonic movements are typically patterned and repetitive, causing twisting movements and abnormal postures. Dystonia occurs when opposing muscles are involuntarily contracting simultaneously. The activation of these muscles may “overflow” to other muscle groups.

Ganglia\textsuperscript{B}: Any structure containing a collection of nerve cell bodies and often also numbers of synapses.

Glial Cell\textsuperscript{B}: The special connective tissue of the central nervous system, composed of different cells, including the oligodendrocytes, astrocytes, ependymal cells and microglia, with various supportive and nutritive functions.

Idiopathic\textsuperscript{A}: With regard to dystonia, this term is used to describe a form of the disorder in which no direct cause (such as brain injury due to trauma, medications, another disorder or condition, or a specific gene mutation) can be identified.

Kinesigenic\textsuperscript{A}: With regard to movement disorders, symptoms that are triggered by sudden body movements such as a startle or by specific activities may be described as kinesigenic.

Motor Cortex\textsuperscript{B}: The region of the cerebral cortex that is responsible for initiating nerve impulses that bring about voluntary activity in the muscles of the body. It is possible to map out the cortex to show which of its areas is responsible for which particular part of the body. The motor cortex of the left cerebral hemisphere is responsible for muscular activity in the right side of the body.

Movement Disorder\textsuperscript{B}: A movement disorder is a chronic neurological condition that affects the ability to control muscle movement. The three most common movement disorders are tremor, Parkinson’s disease, and dystonia.

Myelin\textsuperscript{B}: A complex material formed of protein and phospholipid that is laid down as a sheath around the axons of certain neurons, known as myelinated nerve fibers. Myelinated nerves conduct impulses more rapidly than non-myelinated nerves.

Neural Pathway\textsuperscript{A}: The brain communicates through connections of individual brain cells that fire signals at each other in circuits or patterns. The signals are messages needed to complete a task. These circuits are how areas of the brain communicate with one another and with the rest of the body. In an individual with dystonia, the circuits that facilitate movement are disrupted by abnormal activity.

Neuron / Nerve Cell\textsuperscript{B}: A neuron; one of the cells that makes up the nervous system.
Neurotransmitter\(^\text{A}\): A chemical in the body that serves as a “messenger” and transmits signals between nerve cells (also called neurons) or between the nerves and muscles or organs.

Nervous System\(^\text{A}\): The body’s system to receive and interpret stimuli and send instructions to the organs and peripheral parts of the body. The nervous system includes the brain, spinal cord, and nerves.

Neurological Disorder\(^\text{A}\): Any disease or condition that affects the nervous system.

Pathology\(^\text{B}\): The study of disease processes with the aim of understanding their nature and causes. Clinical pathology is the application of the knowledge gained to the treatment of patients.

Pathophysiology\(^\text{B}\): The disordered physiological processes associated with disease or injury.

Peripheral\(^\text{A}\): With regard to dystonia, a phenomenon (such as trauma or surgery) that impacts an area of the body away from the central nervous system, such as nerve endings or muscles.

Plasticity\(^\text{B}\): Change in the efficacy or connections of the synapses between neurons in the nervous system. It is a crucial process that underlies modification of an animal’s behavior during development and in response to previous activity or experience, including learning and memory.

Primary Dystonias\(^\text{A}\): Those forms of the disorder that occur without the symptoms of any other neurological or metabolic disease.

Proprioception\(^\text{B}\): The form of sensation through which one is aware of the position and orientation of one's body relative to the direction of gravity, of one's body parts relative to one another, and of acceleration and changes in position, the information being supplied by sensory receptors called proprioceptors.

Psychogenic\(^\text{A}\): A term used to describe physical symptoms that originate from a psychological or psychiatric condition.

Secondary Dystonias\(^\text{A}\): Those forms of dystonia that are attributed to an outside factor such as physical trauma, exposure to certain medications, and additional neurological or metabolic diseases.

Sensorimotor Cortex\(^\text{B}\): A generic name for both the somatosensory cortex and the motor cortex, separated by the central sulcus.
Sensory Trick\textsuperscript{A}: A phenomenon where a person with dystonia may temporarily reduce symptoms by gently touching part of the body. Common examples include a person with cervical dystonia placing a finger under the chin to straighten the head, or a person with dystonia of the jaw placing a toothpick in the mouth to reduce symptoms.

Somatic\textsuperscript{B}: Relating to organs and tissues of the body other than the gut and its associated structures. The term is applied especially to voluntary muscles, the sense organs, and the nervous system.

Somatosensory Cortex\textsuperscript{B}: Areas of the cerebral cortex devoted to processing information from the somatic receptors. The primary somatosensory cortex is an area in which parts of the body are mapped contralaterally, with disproportionately large representations of hands, lips, and tongue. The small secondary somatosensory area in the parietal lobes responds specifically to painful stimuli relayed by the peripheral nervous system.

Stereognosis\textsuperscript{B}: Recognition of the three-dimensional shape of an object by touch alone. A function of brain association areas located in the parietal lobe.

Visceral\textsuperscript{B}: Relating to the internal organs (the viscera) of the body as opposed to somatic structures.
CHAPTER I
INTRODUCTION

Professional and aspiring professional musicians spend countless hours performing and practicing, seeking to refine their art and achieve greater technical dexterity and musical sophistication. If one is a musician, then the effort and dedication required to perform at the highest levels is well known. Consider the following scenario: a trombone player earns a seat in one of the nation’s premier orchestras. One day in rehearsal she notices that she cannot articulate some middle-register notes cleanly. The following week this same difficulty recurs and is noticed by the conductor. Naturally, she increases her practice and focuses on the source of the problem.

Unfortunately, she then develops an uncontrollable tremor in her embouchure when playing sustained tones. Over the course of the next few months her ability to play rapidly declines to the point that she is forced to stop playing. What would one do if faced with such a dilemma? To whom would one turn for help? And what would one do when several medical professionals all provided conflicting diagnoses?

Musicians with focal task-specific dystonias often confront these questions on a daily basis. The term dystonia is derived from the Greek dys (abnormal) and tonos (tension) and is applied to neurological disorders that cause unnatural body postures

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1 This is an imagined scenario for illustrative purposes only and does not refer to an actual medical case.
and/or spasms. Focal dystonias affect an isolated part of the body and task-specific dystonias occur only when engaged in a certain action. Focal task-specific dystonias can occur with any part of the body that engages in a controlled, repetitive motion. In brass players, the embouchure can be susceptible to this affliction. Focal task-specific embouchure dystonia (FTSED) is one of the most devastating occupational disorders affecting wind musicians today.

In a recent study by Frucht, neurologist at the Columbia Presbyterian Medical Center and co-founder of “Musicians With Dystonia,”3 only two of twenty-six subjects diagnosed with embouchure dystonia were able to continue their full-time professional performance schedule. This same study asserts that “once present, symptoms of embouchure dystonia did not improve.”5 In a separate study, Lederman, of the Medical Center for Performing Artists at the Cleveland Clinic Foundation, suggested that FTSED is one of the rarest and least-studied disorders afflicting musicians today.6 Retired physician and horn player Dalrymple also comments on the rarity of the disorder,


3 “Musicians With Dystonia” is a special program of the Dystonia Medical Research Foundation that was founded by Dr. Frucht and one of his patients, professional horn player Glen Estrin, with the goals of promoting research and providing support and information for musicians diagnosed with dystonia. For more information, see http://www.dystonia-foundation.org/pages/musicians_with_dystonia/180.php; accessed 14 March 2008.


5 Ibid., 903.

estimating that only 1% of musicians with medical problems are diagnosed with FTSED.\(^7\) Despite the potential severity and perhaps partially due to its rarity, FTSED is not a commonly-known or well-understood disorder among brass performers, pedagogues, and students. During two recent presentations at the 2007 Southeast and Southwest Regional Tuba-Euphonium Conferences, an informal poll revealed that three-quarters of audience members were unfamiliar with FTSED, and of those who were aware of the term, only two could provide a basic description.\(^8\) Similarly, a 1999 study of Canadian music faculty from various universities concluded that the subjects were not knowledgeable about focal dystonia.\(^9\) FTSED is not a common subject for research; fewer than thirty published sources that discuss embouchure dystonia exclusively or prominently were identified in this study.

Unawareness of FTSED in the music community—and the deficiency of research in the medical community—potentially leave those suffering from inexplicable embouchure dysfunction with many more questions than answers. In a striking case, Frucht described one of his patients diagnosed with FTSED as having been “evaluated by


\(^8\) Seth D. Fletcher, “Focal Task-Specific Embouchure Dystonia: Diagnosis, Treatment, Recovery, and Prevention,” (lecture presented at the Southeast Regional Tuba-Euphonium Conference, Western Carolina University, Cullowhee, NC, 16 March 2007); Fletcher, “An Insider’s Perspective on Focal Dystonia,” (lecture presented at the Southwest Regional Tuba-Euphonium Conference, University of Arizona, Tucson, AZ, 14 April 2007).

\(^9\) Kelly Dawn Barrowcliffe, “The Knowledge of Playing-Related Injuries Among University Music Teachers” (M.Sc. diss., The University of Western Ontario, 1999).
as many as 30 other individuals” before learning the true nature of his affliction.¹⁰

Increased knowledge of FTSED among brass instrumentalists and general practitioners
could decrease the possibility of such misdiagnoses. This prospect provides the impetus
for the present study.

**Purpose of Study**

The purpose of this study is to promote awareness and understanding of focal
task-specific embouchure dystonia (FTSED) among brass musicians. An examination of
current scientific, medical, and professional literature was undertaken in an attempt to
answer the following questions:

1. In the simplest terms possible, what is focal task-specific embouchure
dystonia?

2. What are the symptoms of FTSED and are there any “warning signs” that can
aid in early detection?

3. What causes FTSED?

4. How is FTSED diagnosed and treated?

5. Can FTSED be prevented?

6. What is the state of current research concerned with FTSED?

Additionally, a case study of FTSED is included as an appendix with the intention of
providing a source for reference and exploring possible pedagogical influences on
FTSED.

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Characteristics of the Embouchure

The discussion of FTSED must essentially be preceded by a brief discussion of the brass instrumentalists’ embouchure. *The Merriam-Webster Dictionary* defines embouchure as “the position and use of the lips, tongue, and teeth in playing a wind instrument.”¹¹ Traditionally, this definition is altered by brass pedagogues to include the lips only, plus surrounding facial muscles. The functions of the tongue and oral cavity are often considered separately from the embouchure, though they work interdependently with the embouchure during sound production. *Grove Music Online* provides a thorough discussion of both woodwind and brass embouchures, defining embouchure generally as “the coupling mechanism, during the playing of a wind instrument, between the air supply of the player and the instrument.”¹² Frucht gives a slightly more technical definition, describing embouchure as “the set pattern of perioral (surrounding the mouth) and jaw muscles used to initiate and control the amplitude and force of airflow into the mouthpiece of a woodwind or brass instrument.”¹³

According to Frucht, twelve muscles are used in the formation of the embouchure, the most prominent being the *orbicularis oris*, the curved muscle directly above and below the lips (Figures 1 and 2).¹⁴ Iltis and Givens of the Department of Movement Science at Gordon College suggested a similar embouchure anatomy, stating that “no

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¹⁴ Ibid., 900-901.
fewer than seven pairs of muscles...shape the aperture of the lips.”

Both sources include the following muscles as belonging to brass embouchure function: the *orbicularis oris*, *zygomaticus major/minor*, *levator anguli oris*, *depressor anguli oris*, *levator labii superioris*, and *depressor labii inferioris*.16

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16 Detailed descriptions and diagrams of these muscles can be found online on “The Muscle Master List,” an interactive tutorial by the Loyola University Medical Network, see http://medi-smart.com/tut-24.htm; Internet; accessed 25 March 2008.
In brass players, these muscles perform complicated movements that are analogous to the complex motor tasks of the hands in pianists and string instrumentalists. Muscles of the embouchure, however, are only one part of the complex task of tone production on a brass instrument, a process succinctly summarized as follows:
Several factors are involved in producing a tone on a brass instrument: air quantity, speed and direction (which are affected by the back of the tongue, the angle of the instrument as it is held to the mouth, mouthpiece placement and teeth alignment); the ‘push-pull’ of the muscles surrounding the centre of the lips; the ‘harmony’ of the facial mask; the efficiency of the lips as they meet naturally; the structure of the teeth; the ability to achieve correct intonation without ‘lipping’ notes into tune; and a concept of sound focusing on pitch centre, fullness and intensity, and sheer beauty of tone.\textsuperscript{17}

While a complete investigation into brass instrument tone production and the varying theories and definitions of embouchure is beyond the scope of this study, it is imperative to note that the embouchure in and of itself does not produce sound on a brass instrument, but is one part of the tone production process. The importance and function of the embouchure within that process, as well as the best methods for teaching proper embouchure function, vary widely among pedagogues. The role that these differing pedagogical concepts and practices play in relation to brass instrumentalists with FTSED has to date been unexplored.

The specific characteristics of the embouchure necessarily vary, depending on the particular instrument played as well as an instrumentalist’s personal anatomy and pedagogical background. These subjective variations in embouchure appearance, function, and pedagogy require that, for the purposes of this study, the term embouchure be defined as simply and generally as possible. Lederman, of the Department of Neurology and Medical Center for Performing Artists at the Cleveland Clinic, provides such a definition, describing the embouchure simply as “the configuration of facial

muscles utilized in playing a wind instrument.“ Just as the embouchure merits preliminary investigation in this study, certain neurological terms and concepts warrant examination with regard to definitions and functions, as such information is not typical content of brass performance research and is central to the content of this study.

**Key Neurological Concepts**

The human brain is arguably the most complex and elusive study subject that the human race has yet encountered. Although much is known about the brain and new information and insights are reported frequently, more exists that is not known and remains to be discovered. The study of the brain, neurology, is a relatively young discipline, perhaps dating back only 150 years. Despite this short history of neurology, the amount of information available is considerable, as evidenced by more than one hundred professional journals devoted to the topic. While providing a complete and thorough overview of basic neurology is beyond the scope of this study, a summation of the various parts of the nervous system is included.  

The nervous system can be described as the control unit of the human body, responsible for regulating bodily functions—both conscious and unconscious—and the rapid transmission of information. The nervous system can be divided into three main parts: the central nervous system (CNS), the peripheral nervous system (PNS), and the autonomic nervous system (ANS). The CNS (Figure 3) is comprised of the brain and the

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spinal chord. Its primary function is to generate appropriate reactions to sensory signals, from inside or outside the body. The PNS includes all of the nervous system outside of the brain and spinal chord, such as the 12 pairs of cranial nerves and 31 pairs of spinal nerves, that link the CNS with the rest of the body. The ANS is located in both the CNS and the PNS, and is the part of the nervous system which is responsible for involuntary, or automatic functions. Though the CNS and PNS have both ascending sensory pathways (Figure 4) and descending motor pathways (Figure 5), the ANS is thought to utilize descending pathways only.20

Figure 3. The Central Nervous System. Image courtesy of the National Institute of Health.

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Four main “building block” components form the nervous system: neurons, blood vessels, glia, and sensory organs. Nerve cells, or neurons, are the most important component of the nervous system because they transmit essential information in the form of electrical impulses. Two main types of neurons exist: sensory neurons that link to
form ascending pathways and motor neurons that link to form descending pathways. A neuron (Figure 6) is comprised of a main cell body containing the nucleus; dendrites surrounding the cell body, responsible for passing received impulses to the cell body; and an axon which sends impulses on to other cells. The blood vessels of the nervous system supply necessary nutrients to its cells and remove wastes as well. Glial cells are the most numerous cells in the nervous system and act as “glue” that connects and protects. These cells include myelin, the protective insulation around each neuron’s axon, analogous to the outer covering of electrical wire. Finally, the sensory organs and free nerve endings in the skin have receptors that transmit ascending information to the CNS.\footnote{Ibid.}

Figure 6. A Typical Neuron Structure. Image courtesy of the U.S. National Cancer Institute.

Since a full neurological investigation into the specific process of brass instrument performance has not been undertaken, a detailed description is not available.
A concise description of the general process of muscle movement and the parts of the brain involved is, however, possible. Theirl, a board certified chiropractic neurologist at the Functional Restoration Clinic in New York City, provided a helpful summary of that process in an article entitled “It Really is All Connected.” According to Theirl, muscle movement is a four-step process. First, one decides what one wants to move. Next, one decides how the movement should be made. Then, the movement itself is initiated. Finally, one senses the movement that took place. The brain areas (Figure 7) activated in this process are (Step 1) the prefrontal association cortex, (Step 2) premotor cortex, (Step 3) primary motor cortex, and (Step 4) the primary somatosensory cortex. Table 1 outlines specific brain areas with their functions.

Figure 7. Lobes of The Human Brain. Image courtesy of the U.S. National Cancer Institute.

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Table 1. Brain Area Function. Data from Scott Theirl, “It Really is All Connected,” [article on-line]; available from http://www.functionalrestoration.com.

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<th>OTHER FUNCTIONS</th>
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<td>Prefrontal Assoc. Cortex</td>
<td>Decides What to Move</td>
<td>Focus, Concentration, Planning</td>
</tr>
<tr>
<td>Premotor Cortex</td>
<td>Decides How to Move</td>
<td>None</td>
</tr>
<tr>
<td>Primary Motor Cortex</td>
<td>Initiating Movement</td>
<td>None</td>
</tr>
<tr>
<td>Primary Somatic Sensory</td>
<td>Proprioception</td>
<td>Coordinates Movements</td>
</tr>
<tr>
<td>Cortex</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Posterior Parietal Cortex</td>
<td>Coordinates Expected Sensations with Actual Sensations</td>
<td>None</td>
</tr>
<tr>
<td>Primary Visual Cortex</td>
<td>Processes Sight</td>
<td>None</td>
</tr>
<tr>
<td>Higher-Order Visual Cortex</td>
<td>Attaches Meaning to Sight</td>
<td>None</td>
</tr>
<tr>
<td>Parietal-Temporal-Occupipital Association Cortex</td>
<td>Coordinates Feeling, Hearing and Seeing</td>
<td>Sends Information to Prefrontal Assoc. Cortex</td>
</tr>
<tr>
<td>Auditory Cortex</td>
<td>Processes Sounds</td>
<td>None</td>
</tr>
<tr>
<td>Limbic Association Cortex</td>
<td>Coordinates Movements and Senses with Emotion</td>
<td>Sends Information to Prefrontal Assoc. Cortex</td>
</tr>
<tr>
<td>Brainstem</td>
<td>Relay between Brain, Cerebellum and Body</td>
<td>Coordinates Several Bodily Functions</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>Coordinates Muscle Movements</td>
<td>Coordinates Balance, Muscle Rhythm and Timing, Eye Movements, Neck and Back Muscles</td>
</tr>
<tr>
<td>Basal Ganglia</td>
<td>Processes Muscle Movement</td>
<td>Processes Emotions</td>
</tr>
</tbody>
</table>

The preceding discussion merely hints at the barest essentials of human neurology, yet provides important context for this study because FTSED is defined by medical professionals as a neurological disorder. Consulting the aforementioned resources for further information is highly recommended. Dr. Joe Dispenza, an author, researcher, and practitioner featured in the movie *What the Bleep do We Know!* notes
how complex the human brain truly is, stating that “if we were to compare the number of
connections in all of the telecommunication systems in the entire world to the number of
connections in the neurological network of the brain, they would appear the size of a pea
in relation to the size of the human brain.”23 Expecting brass players and pedagogues to
fully comprehend an area of research that scientists and medical professionals find
daunting is probably unreasonable. A basic knowledge of neurology, however, may
benefit brass instrumentalists not only in recognizing and managing FTSED, but also in
general performance and pedagogy as well.

**Organization of the Text**

Chapter One contains a brief introduction and definition of FTSED, a discussion
of embouchure terminology and function, and an overview of key neurological concepts.
Chapter Two includes further investigation of the definition of FTSED, with
consideration of the causes, symptoms, diagnosis, and treatment of the disorder. Chapter
Three presents summations of empirical studies of FTSED and similar dystonias, while
Chapter Four presents summations of case studies of musicians with focal dystonia. The
final chapter includes a summary of key points, suggestions for future research, and
guidelines for recovery. The appendix contains a case study of FTSED, including
specific details of the initial appearance and progression of symptoms, pre-diagnosis
symptom management strategies, a week-long intensive re-training program, and
subsequent methods and routines leading to a return to public performance.

CHAPTER II
DEFINING FOCAL TASK-SPECIFIC EMBOUCHURE DYSTONIA

Definitions and Classifications of Dystonia

To arrive at a complete understanding of FTSED, consideration the origins and development of the terminology, classification and diagnosis of dystonia in general are important. According to Fahn et al., Hermann Oppenheim originally coined the term “dystonia” in 1911 and defined it as a state in which “muscle tone is hypertonic at one occasion and in tonic muscle spasm in another, usually but not exclusively elicited upon volitional movements.”24 What Oppenheim emphasized was his observation of chronic muscle cramps that seemed to occur without the presence of provoking movements. This view was soon replaced by an emphasis upon the disfigured and sustained postures that seemed to characterize the condition.25 Since these early definitions, the understanding of dystonia and related disorders has expanded and advanced significantly.26 In February 1984, an ad hoc committee of the Dystonia Medical Research Foundation proposed that dystonia be understood as a neurologically-based syndrome of sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal

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25 Ibid., 333.

postures.\textsuperscript{27} Dystonic movements can be observed in almost all parts or areas of the body and may occur when that body part or area is at rest or engaged in voluntary motor function. Therefore, dystonia may consist of dystonic movements, dystonic postures, or a combination of both.\textsuperscript{28}

The modern classification of dystonia begins with the division of the disorder into two groups based on the etiology or causes of the dystonia, as shown in Table 2. The first group, idiopathic dystonias, is comprised of dystonia that is itself the primary condition of the patient. In other words, the dystonia causes the patients’ symptoms. The second group encompasses instances of dystonia that are secondary to another condition. In these instances, the dystonia is caused by an outside factor. These conditions are known as symptomatic dystonias. For the greater portion of the twentieth century a third category of psychological etiology was also included in this classification.\textsuperscript{29} Despite extensive research disproving this notion, evidence exists that as late as the 1990s that some psychologists considered some forms of dystonia to be psychosomatic in nature.\textsuperscript{30} Researchers now agree on the causal divisions of dystonia into the two categories of idiopathic and symptomatic.\textsuperscript{31}


\textsuperscript{28} Ibid., 3.

\textsuperscript{29} Ibid., 4.


\textsuperscript{31} Extremely rare cases have been documented in which dystonia is thought to be caused by psychosis due to the elimination of any other criterion. These are the exceptions, however, and not, as once was thought,

<table>
<thead>
<tr>
<th>Idiopathic Conditions (Primary)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Generalized Dystonia</td>
</tr>
<tr>
<td>Hereditary</td>
</tr>
<tr>
<td>Idiopathic Torsion Dystonia</td>
</tr>
<tr>
<td>Segmental Dystonia</td>
</tr>
<tr>
<td>Affecting Two or More Body Areas</td>
</tr>
<tr>
<td>Focal Dystonia</td>
</tr>
<tr>
<td>Occupational Cramps</td>
</tr>
<tr>
<td>Blepharospasm (Eyes)</td>
</tr>
<tr>
<td>Oromandibular Dystonia (Mouth)</td>
</tr>
<tr>
<td>Torticollis (Neck)</td>
</tr>
<tr>
<td>Symptomatic Conditions (Secondary)</td>
</tr>
<tr>
<td>Assoc. with Other Neurological Disorders</td>
</tr>
<tr>
<td>Wilson’s Disease</td>
</tr>
<tr>
<td>Huntington’s Disease</td>
</tr>
<tr>
<td>Hallervorden-Spatz Disease</td>
</tr>
<tr>
<td>Etc.</td>
</tr>
<tr>
<td>Other Causes</td>
</tr>
<tr>
<td>Prenatal Brain Injury</td>
</tr>
<tr>
<td>Brain Trauma, Tumor, or Injury</td>
</tr>
<tr>
<td>Toxin Induced</td>
</tr>
<tr>
<td>Drug Induced</td>
</tr>
<tr>
<td>Psychological</td>
</tr>
</tbody>
</table>

Further classification of dystonia employs two additional criteria: age at onset and distribution of the dystonic movements (Table 3). The age at which the dystonia first appears is important for serving as an indication of the severity and possible spread of the symptoms to other parts of the body. In general, the younger the age at onset, the greater the chance that the condition will spread to other parts of the body and develop with

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the rule. In the first half of the twentieth century disorders such as FTSED were routinely dismissed as psychological problems.
increased severity as time progresses. The distribution of dystonic movements refers to how much and what parts of the body are affected. Distribution can be discussed in the following terms: focal, segmental, multifocal, generalized, and hemidystonic. Focal means that only a single part or area of the body is affected. Common types of focal dystonia include blepharospasm—eyelids affected, torticollis—neck affected, and some occupational cramps. Segmental dystonia affects two or more contiguous body parts and multifocal dystonia affects two or more noncontiguous parts of the body. Generalized dystonia affects one or both legs plus some other region of the body. Finally, dystonia affecting an entire half of the body is deemed hemidystonia.

A clear definition for FTSED can then be derived from these methods of dystonia classification. Firstly, “focal” refers to the localized area of the body affected, in the case of this document, the embouchure. Next, the term “task-specific” characterizes the nature of the dystonic movements as present only during the execution of a specific task. “Embouchure” refers to the facial muscles utilized in wind instrument tone production as defined in Chapter One. And, to reiterate, “dystonia” is the neurologically-based syndrome of involuntary muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures. A muscle spasm that occurs in a trumpet player’s lips when attempting to start a note, a horn player’s inability to sustain a tone without a

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33 For an in-depth perspective into all the focal dystonias, please see Advances in Neurology Vol. 50: Dystonia 2, ed. by Stanley Fahn, C. David Marsden, and Donald B. Calne. Several chapters devoted to the various types of focal dystonia can be found between pp. 457-537.

34 Fahn, “Concept and Classification of Dystonia,” 4-5.
rapidly shaking embouchure, or a tubist’s jaw inexplicably clamping shut when attempting to play octave leaps are all examples of FTSED.


<table>
<thead>
<tr>
<th>Age at Onset</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Childhood Onset, 0-12 Years</td>
<td></td>
</tr>
<tr>
<td>Adolescent Onset, 13-20 Years</td>
<td></td>
</tr>
<tr>
<td>Adult Onset, Older than 20 Years</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Cause</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic (Primary)</td>
<td></td>
</tr>
<tr>
<td>Symptomatic (Secondary)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Location or Distribution</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Focal</td>
<td></td>
</tr>
<tr>
<td>Segmental</td>
<td></td>
</tr>
<tr>
<td>Multifocal</td>
<td></td>
</tr>
<tr>
<td>Generalized</td>
<td></td>
</tr>
<tr>
<td>Hemidystonia</td>
<td></td>
</tr>
</tbody>
</table>

Symptoms of FTSED

The first signs of focal task-specific dystonias in musicians may be so subtle that they pass completely unnoticed—for example, a slightly less controlled violinist’s vibrato, or a pianist’s slight cramps or spasms while playing soft passages. In wind and brass players, FTSED can first appear as evidence of fatigue: unclear articulation, poor tone quality in an isolated register, difficulty with lip slurs, etc. These initial indications are often attributed to lack of practice or to having a “bad playing day.” In FTSED, these first signs are often limited to one range or specific style of playing.\(^\text{35}\) The inexplicable nature of initial symptoms of embouchure dystonia can lead the afflicted performer to

self-doubt, intense frustration, and depression, which will be discussed later. Regardless, certain symptoms and signs can serve as warnings that embouchure dystonia may in fact be the cause. Greater awareness of dystonia is crucial so that these symptoms can be recognized as soon as possible, and a proper diagnosis determined.

According to Frucht, the symptoms of embouchure dystonia can be divided into three main categories: embouchure tremor, involuntary lip movements, and involuntary jaw movement. These categories were derived from observation of symptoms after the condition had been present for some time. As previously noted, the initial symptoms are usually described vaguely as difficulty in performing. Other initial reports include such descriptions as loss of embouchure control, lip fatigue, lip tremor, and involuntary facial movements. The presence of pain in embouchure dystonia is quite rare despite the assertion of physical discomfort by most patients. In the case of embouchure dystonia the symptoms often remain specific to musical performance. However, it is possible for symptoms to spread beyond the initial embouchure-related response. In Frucht’s study, 27% of patients experienced a spread of the dystonia to other oral tasks. The reasons behind the isolation or spread of symptoms are unknown.36

Embouchure tremor consists of a shaking of the lips and various embouchure muscles, resulting in a correlating “wobble” in the player’s sound. This type of symptom is the most common found in embouchure dystonia. The tremor usually begins at the onset of a sound but may occur at any time during performance. In some instances the initial sound is good and the tremor increases the longer a note is held. The oscillations

36 Ibid., 901.
are typically very rapid and extremely noticeable. Most often both lips are involved in the tremor. While involuntary lip movements and involuntary jaw closure tend to be instrument-specific, embouchure tremor affects all types of brass players.\textsuperscript{37}

Involuntary lip movements can be described as either a lateral pull or a closure of the lips (“lip lock”).\textsuperscript{38} Lateral pull is an uncontrolled, usually rapid, movement away from the embouchure shape that is manifest at the onset of the sound or shortly thereafter. Lateral pull affects either one or both lips and/or one or both corners of the mouth. Closure of the lips is characterized by a sealing of the lips at the moment of tone production. As the lips seal shut, the airflow is obstructed. This results in increased effort to force air through the lips producing a delayed note onset and (substantial) lack of clarity of articulation. Lateral pull seems to be specific to trumpet and horn players while “lip lock” is most often observed in trombone and tuba players, although the reasons for such distribution are unknown.\textsuperscript{39}

The final category of symptoms, involuntary jaw movement, is more likely to spread to other activities than the other types of symptoms. Like embouchure tremor, however, it does not seem to be instrument-specific. Involuntary jaw movement can further be divided into jaw closure and jaw tremor. These cases are similar to “lip lock” and embouchure tremor, respectively, but they are expressed in the physically larger context of the jaw. Jaw closure usually occurs at the initial onset of a note and produces

\textsuperscript{37} Ibid.

\textsuperscript{38} Ibid.

\textsuperscript{39} Ibid.
similar effects as “lip lock.” Jaw tremor is also most often present at the onset of a tone and produces rapid variations in pitch.\footnote{Ibid., 901-3.}

A few other general observations can be made about the symptoms of embouchure dystonia. Cases have been reported by players of all ages, but the majority of cases appear in patients between 35 and 45 years old. Next, the development of the symptoms often begins in a specific register. This being said, evidence of a correlation between specific registers on specific instruments and Frucht’s various symptoms types does not exist—i.e., tremor does not exclusively begin as middle-register symptoms, etc. Similarly, the symptoms often begin as articulation-specific. For example, difficulty playing staccato notes. As with register-specific onset, there is no evidence of any symptomatic correlations with articulation-specific onset. In all cases, after the initial onset of symptoms the disorder develops and progresses to various stages of severity (Table 4).\footnote{Adapted from Frucht et al., “The Natural History of Embouchure Dystonia,” 903 and Jan Kagarice, “A Pedagogical Approach to the Issue of Focal Task Specific Dystonia of the Embouchure,” (presentation at the International Trombone Festival, June 2004).}

<table>
<thead>
<tr>
<th>Stage 0</th>
<th>Unable to Play</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stage 1</td>
<td>Plays several short notes, but stops because of blockage or lack of facility</td>
</tr>
<tr>
<td>Stage 2</td>
<td>Plays short sequences without rapidity and with unsteady fingering</td>
</tr>
<tr>
<td>Stage 3</td>
<td>Plays easy pieces, but is unable to perform more technically challenging pieces</td>
</tr>
<tr>
<td>Stage 4</td>
<td>Plays almost normally, but difficult passages are avoided for fear of motor problems</td>
</tr>
<tr>
<td>Stage 5</td>
<td>Able to play normally and returns to concert performances</td>
</tr>
</tbody>
</table>

**Causes of FTSED**

Several possible causes of dystonia exist: genes, brain lesions, injury and trauma, and behavioral causes, among others. In the case of FTSED, however, a growing amount of current research asserts that the condition is a product of overuse resulting in a disorder in the brain’s sensory feedback system, the somatosensory cortex. Described as use-dependent cortical reorganization, the premise is that the motor cortex is “ rewired” due to over-stimulation from the senses. A description of the basic neurology behind motor and sensory function as related to brass performance is necessary to gain a better understanding of the sensory overload that may play a role in causing embouchure dystonia.

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Neurons, the basic functional units of the brain, are specialized cells that transmit information to muscle, gland, or other nerve cells. The human brain contains between one billion and one trillion neurons, which facilitate all brain function.\textsuperscript{45} Neurons communicate through the transmission of electrical impulses that create connections with other cells. The neuron consists of three main parts: cell body, axon, and dendrites. The cell body contains the nucleus, the axon sends electrical signals, and the dendrites receive signals from other neurons. The point of connection between the axon of one neuron and a dendrite of another is called a synapse (Figure 8).\textsuperscript{46} A chain of synapses creates a neural pathway. A neural pathway can be likened to a dirt path that has been worn in a field of grass: the more times it has been traveled and the more attention focused upon the path, the stronger the pathway becomes. In instrumentalists, the concentrated and repetitive practice required creates strong synapses and pathways from the motor cortex, the part of the brain that controls movement, to the specific muscles utilized in playing.\textsuperscript{47} A dysfunction in these pathways is one potential cause of FTSED in musicians.\textsuperscript{48}


\textsuperscript{46} Ibid., 5.

\textsuperscript{47} For a discussion of synapse strength and development, see Schwarz, \textit{The Mind and the Brain}, 106-110.

Figure 8. Synapses. Image courtesy of the National Institute of Health.

The healthy neurological process required for brass playing can be simply described in two steps: auralization and actualization. The player internally hears what he or she wants to play, based upon a specific sound concept—i.e., the ideal tone quality, articulation style, rhythmic value, dynamic level, etc. The body then creates a sound utilizing the instrument through an attempt to match the sound concept. This process
creates a neural pathway, or circuit, in which the concept of sound is the intention that directs the motor cortex to transmit signals to the muscles needed to do the actions that will create a sound matching the concept. This output pathway facilitates all the necessary actions of playing a brass instrument: inhalation, embouchure formation, speed of exhalation, tongue movements, etc. At the same time, the brain is receiving sensory information—the hearing of the actualized sound and the kinesthetic sense of playing—which creates an input neural pathway. These two pathways, motor cortex output and somatosensory input, create a neural circuit that controls and regulates motor functions. While the sensory input can be useful for making adjustments, it is the auralization that produces the output function that is the force behind healthy brass playing (Figure 9).\footnote{Richard J. Lederman, “Neurophysiology and Performance,” in \textit{Medical Problems of the Instrumentalist Musician}, ed. Raoul Tubiana and Peter C. Amadio (London: Martin Dunitz, 2000), 121-33.}

![Figure 9. Healthy Neural Pathways in Brass Playing. Image courtesy of Jan Kagarice.](image-url)
If, however, sensory input becomes the focus of a player’s attention, then a disruption of the motor cortex pathway may occur. Professional musicians often practice or perform on their instruments for several hours a day. If these hours of practice become a vehicle for overuse, misuse or intense focus upon sensory input, then overactive impulses in the sensory pathway can become problematic. Signals from the sensory input can interfere and fuse with motor cortex output causing uncontrolled involuntary movements or FTSED (Figure 10). Is there a degree of focus on sensory input that can lead to embouchure dystonia? The answer may be found in current philosophies of brass pedagogy.

Figure 10. Disrupted Neural Pathways in Brass Playing. Image courtesy of Jan Kagarice.

Physicians describe most illnesses in terms of the symptoms that are manifest in the body. Similarly, many brass pedagogues teach descriptions of what appears to happen when one plays well. Physiology is often used as a method. For example, in a
well-functioning embouchure the corners of the mouth can usually be observed to be firm. Using this observation, teachers will tell students to keep their corners firm when playing. Similarly, when a relaxed breath is taken the abdomen appears to rise. Many teachers tell their students to breathe low, into their abdominal cavities. But one will find that you can have firm corners and a rising abdomen and have neither a working embouchure nor a relaxed breath. A description of the physical appearances of good brass playing is not a prescription for good brass playing. In fact, it may be just the opposite. Perhaps Dennis Wick, the English trombonist and pedagogue, says it best: “The players/teachers do what they do. They tell the students what they think they do. The students then try to do what they think the teachers (think they) said about what they think they do.”

Diagnosis of FTSED

The definition and classification of dystonia is complex. The preceding discussion serves to demonstrate some of the confusion and mystery that has clouded dystonia. It is understandable, given the elusive nature of the disorder, that obtaining a correct diagnosis has been difficult for musicians with FTSED. Additionally, the stigma of such a diagnosis, which can be damaging to a musician’s career, may lead to the avoidance of any diagnosis altogether. Although any physician may diagnose dystonia, seeking such an opinion from a neurologist is preferable and recommended. A physician who suspects dystonia will typically refer a patient to the appropriate specialist.

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Unfortunately, due to the difficulty of diagnosing dystonia in musicians, several opinions may be required before a sufficient diagnosis is rendered.

The diagnosis of dystonia is a three-stage process. The first stage is the recognition of abnormal movements associated with dystonia. The second stage is the classification of these movements by age of onset and location, followed by determining the patient’s history with the problem. Finally, a thorough investigation into the cause of the dystonia is undertaken.\(^{51}\) According to Tubiana, two equal parts comprise the examination of a musician suspected of being afflicted with focal dystonia: a thorough history, which determines the greater part of the diagnosis and prognosis, and a complete physical examination that is both orthopedic and neurological in nature.\(^{52}\) Naturally, diagnosis of embouchure dystonia, as well as any occupational disorder, involves examination of the patient’s symptoms while performing the task in question—in this case, playing the instrument. The thoroughness of further examinations as described above is determined on a case-by-case basis, depending on specific symptoms present and the patient’s history. In comparison to other forms of dystonia, the diagnosis of focal dystonia in musicians is particularly difficult for several reasons. As Wilson notes, the subjectivity of early symptoms, the variation in the manifestation of the disorder with

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regard to the instrument being played, the high demands for accuracy, and subtle variables in individual performance often make it difficult to obtain a diagnosis.\(^3\)

The onset, diagnosis, and prognosis of brass musicians diagnosed with FTSED can be described in a generic case history model (Table 5).\(^4\) This case history begins with a description of personal traits of the patient. The patient is most often a natural player, meaning he or she plays by instinct and by feel, not normally intellectualizing the playing process. These players are highly talented and have achieved measurable levels of success, most being full-time performers. Universally present is a preoccupation with perfection and a high level of commitment and dedication to practice and improvement. Additionally, most players with embouchure dystonia would be accurately described as musically intuitive and expressive performers.

The first event in the case history is a major change in the sensation of playing one’s instrument. This change can be the result of many possible factors: a new instrument or mouthpiece, injury, change in technique, new job, increase in performances, and/or added stress from any number of factors. Subsequently, a symptom develops that is similar to those described previously: range-specific or style-specific difficulties in performance. The player then attempts to resolve the symptom by way of physiology, as taught by most pedagogues. He or she visits leading teachers and reads pedagogical texts, trying to understand and intellectualize what is going wrong.


\(^4\) This case history model has been developed by Jan Kagarice, adjunct professor of trombone at the University of North Texas, member of the International Trombone Association’s (ITA) Pedagogy Committee and chair of ITA’s ad hoc committee on FTSD.
Table 5. Case History Model of Player Diagnosed with FTSED. Data from Jan Kagarice, “A Pedagogical Approach to the Issue of Focal Task Specific Dystonia of the Embouchure,” Presentation at the International Trombone Festival, June 2004.

<table>
<thead>
<tr>
<th>Personal Traits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Natural Player</td>
</tr>
<tr>
<td>Considered Talented and Successful</td>
</tr>
<tr>
<td>Perfectionist Personality</td>
</tr>
<tr>
<td>Committed and Dedicated to Practice and Improvement</td>
</tr>
<tr>
<td>Naturally Expressive and Intuitively Musical</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Change in the Feel of Playing</th>
</tr>
</thead>
<tbody>
<tr>
<td>New Equipment</td>
</tr>
<tr>
<td>New Job</td>
</tr>
<tr>
<td>Increase in Performance, Practice, or Difficulty o Repertoire</td>
</tr>
<tr>
<td>Increase in Stress for Various Reasons</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms Develop</th>
</tr>
</thead>
<tbody>
<tr>
<td>Often Range- Specific</td>
</tr>
<tr>
<td>Often Style-Specific</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Attempts to Remedy Symptoms with Physiological Approach</th>
</tr>
</thead>
<tbody>
<tr>
<td>Visits Leading Pedagogues</td>
</tr>
<tr>
<td>Reads Pedagogical Texts</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Focus Solely on the Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased Practice Time</td>
</tr>
<tr>
<td>Increased Repetitions on Problematic Exercises</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms Worsen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased Anxiety and Stress</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Notice of Symptoms or Perception of Notice by Colleagues</th>
</tr>
</thead>
<tbody>
<tr>
<td>Feelings of Embarrassment</td>
</tr>
<tr>
<td>Increased Self-Consciousness</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symptoms Continue to Worsen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression May Develop</td>
</tr>
<tr>
<td>Anxiety Increases</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Condition Becomes Clinical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Quits Playing</td>
</tr>
<tr>
<td>Seeks Medical of Other Treatment</td>
</tr>
</tbody>
</table>

The symptoms become the complete focus of the player’s attention and the practice regimen is increased. Unfortunately, despite the player’s best efforts, the problem worsens while fear, anxiety and stress continue to build. At this stage, the player is either afraid others will notice the problem (or the problem has already been noticed)
and embarrassment and self-consciousness become constant. The next result is generally the onset of depression as the problem continues to worsen until it reaches a clinical state. Often the dystonia progresses until playing is virtually impossible.

This general progression of embouchure dystonia is then followed by one of two actions: the player either stops playing or consults a medical professional and begins some form of treatment. Understanding that the physically observable symptoms of FTSED are not the extent of the disorder is an important realization. FTSED in musicians can cause severe depression and a loss of identity that is difficult to overcome.\(^\text{55}\) Several neurologists propose that treatment of embouchure dystonia (and indeed all FTSD) should be holistic in nature and aimed at total wellness.

**Treatment of FTSED**

No known cure exists for FTSED or any other form of dystonia. Rather, treatments for dystonia seek to mitigate symptoms. To date, no medical treatment has been found to be universally successful for FTSED, and in fact, all medical treatments have yielded only minimal success.\(^\text{56}\) A variety of methods have been used to treat FTSED, both traditional and non-traditional. Traditional medical treatments include *botulinum toxin* injections, trihexyphenidyl administered orally, psychotherapy, chiropractic treatment, prolonged rest, physical therapy and surgery. Non-traditional treatments include biofeedback, acupuncture, herbal therapy, massage therapy, dental

\(^{55}\) For a stirring account of one musician dealing with injury, see Jennifer Buller, “What is it like to be an Injured Musician?” *Canadian Music Educator* 43, no. 4 (Summer 2002): 20-3.

prosthetics, constraint induced movement therapy, aquatic therapy, and dietary changes.\textsuperscript{57} According to Frucht, the most effective treatment for embouchure dystonia is a re-training of the embouchure.\textsuperscript{58} Still, some of the aforementioned therapies deserve closer inspection.

Perhaps the most common medical treatment for FTSED is \textit{botulinum toxin}. As with all dystonia treatments, \textit{botulinum toxin} is prescribed with the intent of alleviating symptoms and has no ability to cure the disorder. A small amount is injected directly into the specific muscles exhibiting dystonic movements and has a weakening effect on those muscles. This reversible effect lasts approximately two to three months. Since the effects do not last, repeated injections are needed if any benefits are to be maintained. There has been some success with \textit{botulinum toxin}, the most famous case of which is the pianist Leon Fleischer, who recently began to play with both hands again after being limited to playing with the left hand for thirty years due to hand dystonia.\textsuperscript{59} Frucht noted in his study that in seven patients with embouchure dystonia only one showed significant improvement with \textit{botulinum toxin} injections.\textsuperscript{60} The overall ineffectiveness of this treatment in musicians likely is due to the difficulty in achieving the proper level of


\textsuperscript{58} Ibid., 904.


\textsuperscript{60} Frucht et al., “The Natural History of Embouchure Dystonia,” 904.
dosage. Administering too much *botulinum toxin*, a frequent mistake, causes highly impaired muscle function.\(^{61}\)

*Botulinum toxin* may be an option worth exploring for some patients, primarily those with hand dystonia, but it has proven ineffective as a treatment for embouchure dystonia. Additional traditional treatments have proven ineffective as well. *Trihexyphenidyl* has been shown to produce results in child-onset segmental dystonia when administered within five years of onset, but has not been proven to show any results in other forms of dystonia.\(^{62}\) Prolonged rest and surgery also have not been effective in treating embouchure dystonia. Chiropractic treatment, psychotherapy and physical therapy have provided some relief but have not been proven to significantly reduce the symptoms of FTSED or other dystonias. Chiropractic treatment may be helpful since there is some evidence showing a correlation between FTSED and posture issues; psychotherapy can be helpful in treating the depression that often accompanies FTSED in musicians; and traditional physical therapy may help to recreate healthy neural pathways.\(^{63}\)

Non-traditional treatments likewise can provide some benefits and relief but also fail to provide a viable treatment strategy for FTSED. Biofeedback, acupuncture, herbal therapy, aquatic therapy, and dietary changes may provide some distraction from the symptoms of FTSED as well as non-related benefits, but these practices have not been

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\(^{61}\) Lim, Altenmüller, and Bradshaw, “Focal Dystonia: Current Theories,” 898.


\(^{63}\) Lim, Altenmüller, and Bradshaw, “Focal Dystonia: Current Theories,” 898.
shown to have any clinical effect on dystonia. Dental prosthetics have also not been successful. In at least one case, however, massage therapy has alleviated embouchure dystonia symptoms, but this result has not been substantiated elsewhere.\textsuperscript{64} Constraint-induced movement therapy has been shown to be useful in the treatment of hand dystonia and in the rehabilitation of stroke victims.\textsuperscript{65} In this unique treatment, unaffected areas such as arms or fingers are restrained while the patient is required to complete a task using the affected body part.\textsuperscript{66} This approach has shown considerable short-term benefits in patients with hand dystonia, though more studies are needed and to date it has not been utilized in the treatment of embouchure dystonia.\textsuperscript{67}

**Summary**

To arrive at a more complete understanding of FTSED, the classifications of dystonia, as well as symptoms, causes, diagnosis, and treatment of the disorder were considered. FTSED can be described as a neurologically-based movement disorder characterized by abnormal random or sustained involuntary muscle contractions initiated during playing that cause embouchure dysfunction. Initial symptoms are often dismissed as signs of fatigue, lack of practice, or simply having a bad playing day—unclear articulation, poor tone quality in an isolated register, difficulty with lip slurs, etc.—and

\textsuperscript{64} Frucht et al., “The Natural History of Embouchure Dystonia,” 904.

\textsuperscript{65} Lim, Altenmüller, and Bradshaw, “Focal Dystonia: Current Theories,” 898.


\textsuperscript{67} Lim, Altenmüller, and Bradshaw, “Focal Dystonia: Current Theories,” 899.
three categories of developed symptoms can be described: embouchure tremor, involuntary lip movements, and involuntary jaw movement.

A thorough diagnosis of FTSED is best obtained from a neurologist and consists of a detailed medical and musical history and a complete physical and neurological examination. Diagnosis of embouchure dystonia is potentially difficult due to the subjectivity of early symptoms, the high demands for accuracy in musical performance, and variables in daily performance. The precise causes of FTSED are unknown and it is likely that many factors contribute to its development. One possible theory suggests that overuse and overload of sensory input causes a “rewiring” of the somatosensory cortex, resulting in disruption of natural motor function. No proven or typical medical plan of treatment for FTSED exists and the only treatment shown to be effective is a retraining of the embouchure, although little documentation exists to substantiate this assertion. Traditional treatment options include *botulinum toxin* injections, oral medications, psychotherapy, chiropractic treatment, prolonged rest, physical therapy and surgery.

The following chapter provides summations of empirical research of focal dystonias in musicians, including further examination of symptoms, possible causes, diagnosis, and treatment. Four studies of FTSED in brass musicians and nearly 50 studies of other musicians affected with focal dystonia were identified. The literature reviewed includes extensive clinical observation reports and experimental research.
CHAPTER III
EMPIRICAL STUDIES OF FOCAL DYSTONIAS

The preceding discussion provided a general overview of FTSED encompassing the definition and classification, symptoms, causes, diagnosis, and treatment of the disorder. Further insight may be gained from detailed examination of empirical studies related to FTSED. The purpose of this chapter is primarily to identify what methods and analyses have been previously implemented as a means of reference and as an aid to future study design. There are two categories of empirical studies included: those that are experimental in design and those that provide long-term observations of clinical practices. The literature reviewed is divided into the following categories: studies involving brass musicians and studies involving other musicians.

Studies Involving Brass Musicians

Published empirical research specifically concerned with FTSED affecting brass musicians is severely limited in scope. This study identified four such papers; two of which provide clinical observations and two of which are experimental in design. Literature will be discussed in order of publication. The first study to address FTSED affecting brass musicians was written by Lederman of the Department of Neurology and Medical Center for Performing Artists at the Cleveland Clinic Foundation.68

In this study, “Embouchure Problems in Brass Instrumentalists,” Lederman chronicles the clinical history of 81 brass instrumentalists over a period of fifteen years between 1985 and 2000. The records of these patients were reviewed and compared for demographics, symptoms, possible causes of symptoms, results of examination, management strategies, and treatment outcomes. Treatment outcomes of patients with FTSED and those with other diagnoses were then compared. Lederman identified 43 patients (of 81 total brass player patients) seen for embouchure disorders, 18 of whom were diagnosed with FTSED (42% of those with embouchure disorders, 22% of total brass player patients). Of the 18 patients diagnosed with FTSED, 16 were men. Horn and tenor trombone players accounted for 5 each these 18. Six were trumpet players and there was one each of bass trombone and tuba players. Symptoms of those diagnosed with FTSED included impaired control, loss of lip seal, poor articulation, decreased flexibility, spasms, poor tone quality, stiffness, and tremor.

Lederman reported that diagnosis of FTSED and other embouchure problems often relies more on what is heard rather than seen. Only 7 of the 18 patients with FTSED were observed to have visible abnormality of lip or facial movement and two patients were diagnosed from their descriptions of the onset and evolution of symptoms, since no change in sound or appearance was clinically observed. Regarding treatment, only 1 of the 18 (6%) patients with FTSED resumed unlimited playing after treatment, compared with 14 out of 25 (56%) patients with other embouchure disorders. Treatment for FTSED patients included technical re-training, mouthpiece alteration, and medication
with anticholinergic drugs—medications blocking the neurotransmitter acetylcholine\textsuperscript{69}—such as trihexyphenidyl. Specifics of the technical re-training utilized were not provided. Lederman concluded that “with dystonia, technical re-training is the desired method of treatment but seems even more difficult to accomplish than with limb dystonia, and results have been particularly disappointing.”\textsuperscript{70}

The second study of FTSED affecting musicians is Frucht’s “The Natural History of Embouchure Dystonia.”\textsuperscript{71} In this study, Frucht describes twenty-six patients suffering from FTSED with the stated purpose to “describe and demonstrate the phenomenology of embouchure dystonia, to bring to attention a rare but professionally-disabling condition, and to propose a method of classifying the specific defects in patients with embouchure dystonia.”\textsuperscript{72} Patients reported a complete history and underwent neurological examination, although specifics of each are not detailed. Additionally, patients responded to a written questionnaire on the following topics: previous medical evaluations and diagnoses, musical training, current professional engagements, prior playing history, past injuries and medical history, family medical history, initial and current symptoms, spread of dystonia to other tasks, response to treatment, and long-term follow-up. Nineteen patients were videotaped both “buzzing” on the mouthpiece alone and playing their instruments.


\textsuperscript{70} Lederman, “Embouchure Problems in Brass Instrumentalists,” 57.

\textsuperscript{71} Steven Frucht et al., “The Natural History of Embouchure Dystonia,” 899-906.

\textsuperscript{72} Ibid., 900.
This study included patients playing the following instruments: 11 horn, 5 trumpet, 5 tuba, 2 trombone, 2 flute, and 2 clarinet. In these patients, symptoms of FTSED began to emerge an average of 26 years after beginning to play their instrument (mean was 38 years of age), and they were evaluated at the authors’ clinics at an average age of 46 years. It was reported that most had received prior evaluation and diagnoses for a range of disorders including trismus—jaw muscle spasms, often a symptom of tetanus, that keep the jaw in a closed position—a temporomandibular joint dysfunction, neuropathy, muscle strain, depression, hysteria, and Bell’s palsy. Patients described their initial symptoms vaguely as “difficulty performing,” and Frucht mentioned the following symptoms observed: “loss of embouchure control, lip fatigue, lip tremor, and other involuntary facial movements.” Pain was uncommon, symptoms were initially reported to appear in a specific register, and loss of self-esteem and/or depression was observed in most patients. Four distinct classes of FTSED were postulated: lip tremor, lateral pulling of one or both lips, involuntary lip closure, and involuntary jaw movement.

The treatment of patients in this study incorporated both traditional and non-traditional approaches including acupuncture, herbal therapy, chiropractic treatment, massage, dental prosthetics, botulinum toxin injections, and embouchure re-training. One


75 Ibid., 901

76 Ibid.
patient reported mild effectiveness from massage therapy and another gained significant improvement from *botulinum toxin* injection (*masseters, medial pterygoids, and temporalis*). Embouchure re-training was reported to be somewhat effective for patients experiencing lateral lip pull, but not for those with tremor (details of the embouchure re-training were not described). The study did not identify any potential risk factors for FTSED, but only that certain evidence (three patients’ development of writer’s cramp prior to FTSED) suggests the possibility of predisposition to FTSED. Frucht concluded that two factors play a role in the difficulty in diagnosing FTSED: the reluctance of musicians to seek medical attention and the unfamiliarity of health care professionals with embouchure function in instrumentalists.

The first empirical study of brass musicians with FTSED, published in 2004, investigated the organization of lip representation in the patients’ somatosensory cortex—a part of the brain dedicated to processing sensory information, with clearly defined areas representing specific body areas. The authors cite previous studies of focal hand dystonia that display evidence of abnormal somatosensory representation of patients’ fingers and seek to examine similarities in the somatosensory representation of the lips in patients with FTSED. Magnetoencephalography (MEG), a non-invasive brain imaging technique that records magnetic fields produced by the electrical activity of neurons,\(^7\)

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was utilized to compare the somatosensory representation of patients with a control group. Additionally, a psychophysical test of touch sensitivity was performed.

The study subjects included eight former brass players, all male, who were previously diagnosed with FTSED and had not undergone any form of treatment: two trumpet players, four horn players, and two trombonists. Their ages ranged from 28 to 43 years-of-age and none had dystonic symptoms that had spread beyond playing their instrument. The control group for the somatosensory representation portion of the experiment comprised eight male non-musicians between the ages of 23 and 38. An additional control group for the psychophysical test included four healthy male musicians and two healthy female musicians—all brass players. Subjects first underwent a psychophysical test (comparing the psychological magnitude of sensations and the physical amplitude of stimuli applied)\textsuperscript{79} of gap detection in their fingers and lips. They subsequently received tactile stimuli in the same areas recorded by MEG.

Results of the gap detection test indicated that patients and controls showed no difference in sensitivities between fingers in each hand when viewed as a group or when compared between groups. Data for lip sensitivities indicated that healthy musicians demonstrated a higher sensitivity threshold (i.e., ability to perceive weaker stimuli) than controls and that patients’ upper lips showed lower sensitivity in most cases—10 of 16, with 2 others showing lower sensitivity in their lower lip—than healthy musicians or control subjects. The MEG data showed that subjects’ lip representations of their

affected/unaffected lips were respectively similar and that both fingers and lips were
mapped in the same order as in the normal somatosensory homunculus. While the order
of representation was shown to be normal—little finger, ring finger, middle finger, index
finger, thumb, . . ., lips—the spatial representation between digits and lips was found to
be closer together in patients than in controls. Specifically, the point of cortical
representation of the thumb was laterally closer to the lips in patients.

The authors draw three main conclusions from the data analysis. First, the
reduced distance in cortical representation of space between the thumb and lips is
consistent with plastic brain reorganization through intense training as demonstrated by
similar results in studies of musicians with focal hand dystonia. Second, the
psychophysical test demonstrated a positive correlation between the repetitive
movements of the upper lip in brass playing and the involuntary movements observed in
patients with FTSED. Finally, “it is reasonable to infer that there is probably a close
relationship between decreased sensitivity of the upper lip and occurrence of embouchure
dystonia.”

The second, most recent empirical study of FTSED, published in 2005, sought to
quantify and qualify selected embouchure-muscle activity in horn players—with and
without FTSED—through the use of surface electromyography (EMG). EMG is the
continuous recording of the electrical activity of a muscle by means of electrodes inserted

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into the muscle fibers, the tracing of which is displayed on an oscilloscope.\textsuperscript{82} Surface EMG utilizes non-invasive electrodes placed directly on the skin. In addition to EMG data, audio and video recording was utilized for comparative purposes. Iltis and Givens suggest that the lack of similar research may be due to the perceived difficulty of isolating muscles utilized in embouchure formation, non-consensus of appropriate dependent variables for comparison, and the non-existence of standardized methods for using EMG in qualitative and quantitative study. The authors proposed that their study addresses those issues.

Five horn players participated as subjects in the study, four of whom were healthy (two males, two females) and one who was diagnosed with FTSED. Healthy subjects ranged in age from 21 to 25 years and were accomplished students at the Aspen Festival School of Music. The dystonic subject was a 49-year-old freelance performer who experienced the onset of symptoms after thirty-seven years of playing.\textsuperscript{83} Each subject was tested on two occasions, each session being recorded by EMG, audio and video.\textsuperscript{84} EMG data was gathered by two pairs of surface electrodes placed on subjects’ left levator labii superioris and depressor anguli oris muscles. Sound recording was done with a Yamaha Silent Brass system and video was taken with a digital camcorder that streamed data into a laptop and synchronized the recording with EMG input. Each testing session


\textsuperscript{83} Though not disclosed in this study, the subject with FTSED was in fact Dr. Iltis, as referenced in the September 2002 edition of \textit{The Instrumentalist}, v. 57, 38-39.

\textsuperscript{84} Two videos, one example of normal performance and one example of FTSED are available on Dr. Iltis’ faculty website at Gordon University, http://faculty.gordon.edu/ns/mv/Peter_Iltis/dystonia/index.cfm.
consisted of three parts. First, subjects performed four iterations of concert-pitch C5 (523.28 Hz), each note played for four beats at 60 b.p.m. with four beats rest between each note played. Next, subjects repeated the same process with concert-pitch C4 (261.64 Hz), a note that was predetermined to produce lip tremor in the subject with FTSED. Finally, a “fatigue trial” consisting of 60 seconds of loud, sustained C5 (with necessary breaths) was carried out to examine the reliability of specific variables.

The data collected was subject to rigorous statistical analysis and a descriptive comparison between audio and imaging data. Analysis showed that each tested variable proved “highly reliable” within each of the two testing sessions. In assessing the reliability between the two testing sessions, variables showed “fair to good” reliability with the exception of the power calculation of the levator labii superioris muscle. The EMG signal of the dystonic subject was found to have rapid explosions of high output mixed with points of very low output, which corresponded to the unsteady and weak tone production observed through audio analysis and visible as a tremor during playing. The explosive activity in the dystonic subject was observed in both muscles tested, but was more prominent in the levator labii superioris. The EMG data from healthy subjects showed, in contrast, more regular and sustained muscle output throughout, corresponding with an observed evenness and strength in tone quality. EMG signal strength was “homogenous” for both muscles tested in the healthy subjects.

85 Ilitis and Givens, “EMG Characterization of Embouchure Muscles Activity,” 29. See figures 3, 4, and 5 for visual depictions of increased, spastic muscle output in a subject with FTSED.

86 Ibid., 30.
The authors draw several conclusions from both results, observed and analyzed. They suggest that their quantitative analysis demonstrates acceptable reliability and that testing of the variables utilized in this study may be useful in comparing horn players with FTSED to those that are healthy. The qualitative analysis of video and audio recording synchronized with EMG data provides substantiated evidence that suggests these methods are useful as a diagnostic tool. Finally, because only one subject with FTSED was examined, the authors recommend that additional studies examine a greater number of subjects and also patients with various incarnations of FTSED.

**Studies Involving Other Musicians**

Empirical studies of task-specific dystonias affecting musicians other than brass players are concerned primarily with hand dystonia (FTSHD). Research exploring FTSHD is far more bountiful than that concerned with FTSED—nearly fifty studies have been identified. The findings of these studies will be discussed according to study focus: large case-series reviews, anxiety as a causative factor, the effect of muscle fatigue, sensory perception, somatosensory organization, sensorimotor integration, development of quantification methods, drug therapies, and re-training programs.

Case series review studies typically provide patient demographic information, description of presenting symptoms, discussion of patient history and etiological factors, and the application and outcome of treatment strategies. Three types of case series reviews have been identified: general overviews of all patients seen in a particular practice, analyses of a general overview in examination of a specific question, and series that focus on one instrumental group. Professional musicians are the predominant
subjects of these studies, although students, amateurs, and part-time professionals are also included.

In the earliest of the large case series reviews identified in this study, Newmark and Hochberg describe “isolated painless manual (hand) incoordination” present in 57 musicians out of 450 total musicians seen during a five-year period. Of those 57 patients, 42 were male with an average age of 42 years. Thirty-five patients were pianists; and other instruments represented were guitar, violin, viola, cello, clarinet, flute, bassoon, trumpet, harp, and percussion. Three stereotypical symptoms were described, accounting for 32 cases: “flexion of the 4th and 5th fingers in pianists,” “flexion of the 3rd finger in guitarists,” and “extension of the 3rd finger in clarinetists.” The remaining 25 patients exhibited various dysfunctions ranging from individual finger movements to movements involving the entire hand. Thirty-seven patients reported a “triggering incident” of either trauma, inflammation, or significant increase in practice prior to onset of symptoms. Treatment options administered included physical therapy, various drug trials, and biofeedback. Three patients experienced improvement from physical therapy and three additional patients found drug therapies to be beneficial. Treatment of all other cases was deemed unsuccessful.

A later study of similar scope to Newmark and Hochberg’s exhibited many of the same results, but with one key difference: the absence of the prevalence of three stereotypical symptoms. Lederman described a series of 42 musicians diagnosed with

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focal task-specific dystonia out of 507 total musicians seen in his practice through 1990.\textsuperscript{88} Patients were predominantly male (29 of 42) with an average age at onset of 35 years. The study included patients from every major instrumental category, with a prevalence of pianists (9). Presenting symptoms included impaired control, stiffness or cramping, involuntary movements, rapid fatigue, tremor, loss of mouthpiece seal (embouchure), and muscle weakness. Additionally, eight patients reported pain. Detailed descriptions of symptoms of each patient are provided. The presence of a “triggering incident” was reported in 60% of cases and included trauma, nerve entrapments, significant increase in playing time (N=12), and a period of increased stress. A “sensory trick,” or maneuver that reduces symptoms, was observed in seven patients. Treatment options in this series included \textit{botulinum toxin} injections as well as slow practice and re-learning. Detailed descriptions of treatment strategies are not provided. Only four of the 42 patients have continued playing with little to no impairment.

Brandfonbrener published a third case series of similar scope, detailing reports of 58 musicians diagnosed with focal task-specific dystonias out of 3,918 instrumental musicians seen between 1985 and 1995.\textsuperscript{89} A 3-to-1 ratio of males to females was reported, with an average age of 38 years at onset. Pianists comprised the largest instrumental group in the study and violin, viola, guitar, percussion, accordion, flute, clarinet, oboe, bassoon, trumpet, trombone, and horn were also represented. The embouchure was affected in four woodwind-playing patients and all seven brass patients.


All other subjects experienced symptoms in the hand or specific fingers. Handedness was not found to have any correlation with presentation of symptoms. Detailed tables providing the specifics of each subjects’ symptoms are provided. Certain “triggering factors” are reported in 43 cases, including (in order of prevalence): sudden increase in practice/performance, return to graduate-level study, radical change in technique, nerve entrapment, physical trauma, psychological trauma, and a new instrument. Treatment strategies employed and patient outcomes are not mentioned.

Two publications chronicle the same series of musicians diagnosed with focal task-specific dystonias between 1994 and 2001 at the Institute of Music Physiology and Musician’s Medicine at the University of Hannover (Germany).90 The details of 144 patients are presented, again exhibiting a prevalence of males (81%) with a mean age at onset of all patients of 33 years. The instrumentalists most represented were pianists, followed closely by guitarists. The following instruments were also represented: organ, harpsichord, violin, viola, cello, double-bass, flute, clarinet, saxophone, oboe, bassoon, recorder, trombone, trumpet, horn, tuba, electric bass, and harp. Those patients exhibiting FTSHD (N=124) presented typically with involuntary hand or finger movements.91 The remaining twenty patients presented symptoms affecting the embouchure area. Detailed descriptions of symptoms were provided. In contrast to Brandfonbrener’s study, a correlation was identified between handedness and the affected


91 Interestingly, this group included a trombone player with dystonic symptoms affecting the left arm, perhaps the lone documented instance of a non-embouchure dystonia in a brass musician.
limb in FTSHD. Although “triggering events” of individual patients were not specifically discussed, the authors provide a thorough analysis of the possible development of musician’s dystonia as a combination of predisposition and extrinsic and intrinsic triggers (Table 6). Treatments included trihexyphenidyl, botulinum toxin injections, ergonomic changes, pedagogical re-training, and non-specific instrumental exercises. Extensive details of treatment were outcomes are provided.

Table 6. Possible Predisposition and Intrinsic and Extrinsic Triggering Factors in the Development of Musician’s Dystonia. Data from Jabusch and Altenmüller, “Focal Dystonia in Musicians: From Phenomenology to Therapy,” Advances in Cognitive Therapy 2, nos. 2-3 (2006), 213.

<table>
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<td>Intrinsic Triggering Factors</td>
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Two large case series address a specific issue with respect to the overall population studied. The first explored the potential of certain instrumental groups and gender as risk factors.\(^{92}\) Data collected from a survey of 2,661 healthy musicians was compared with data collected from 183 patients diagnosed with FTSHD or FTSED.

Woodwind and guitar players were determined to be the most likely groups to develop focal dystonia while percussionists and strings players were the least susceptible. When gender was considered, an unexpected ratio of male musicians to female musicians was observed, suggesting that males exhibit some predisposition that does not present in females. These analytical findings confirm similar data reported in previous studies.

The other large case series publication addressing a specific concern explored the presentation of secondary motor disturbances in patients treated at the Institut de Fisiologia i Medicina de l’Art in Terrassa, Spain. A review of 101 cases of musicians diagnosed with focal dystonia out of 771 total musicians seen during a five-year period included clinical history analysis, neurological examinations, and instrumental performance observations. Over half (53.5%) of all patients reported secondary motor disturbances, which included difficulty playing a similar second instrument, typing on a keyboard, gripping actions between thumb and index finger, and writing. In 20 patients, the task-specific dystonia and secondary motor problem presented simultaneously, while the remaining 32 experienced a delay between one month and twelve years. Secondary motor disturbances presented in the following instrumental groups—in order of prevalence: plucked strings, keyboard, woodwind, brass, and strings. Further details and analyses of these secondary motor disturbances are provided. Rosset-Llobet concluded that the high percentage of secondary motor disturbances in musicians with task-specific dystonias suggests that the disorder is, in fact, more movement-specific than task-specific

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and that longer follow-up assessments may reveal greater occurrences of these disturbances.

The final group of identified case series reports are investigations of smaller populations of specific instrumental groups: woodwind, strings, and percussion. Schuele and Lederman report on twenty-four woodwind instrumentalists seen between 1985 and 2001 with specific reference to long-term outcome, treatment benefits, and the impact on patients’ careers.94 Fifteen subjects were male (62.5%) with an average age at onset of 34. Instruments represented included flute (4), clarinet (10), oboe (3), bassoon (2), and bagpipes (4). Eighteen were diagnosed with FTSHD and six with FTSED (2 clarinet, 2 flute, 2 bassoon). Thirteen patients reported “triggering events” including trauma, stress, new teacher, increased playing, or illness. Treatments for patients with FTSED included acupuncture, re-training, rest, instrument modifications, trihexyphenidyl, and bromocriptine. Treatments for patients with FTSHD included Alexander technique, Feldenkrais therapy, re-training, trihexyphenidyl, and botulinum toxin injections. Treatment outcomes proved successful in less than half of all patients.

The next of the instrument-specific series, incidentally by the same authors, reports data from 21 string instrumentalists seen between 1986 and 2001.95 Subjects included 18 men and 3 women, with a mean age of 34 and instrument distribution of 15 violinists and 6 violists. All were diagnosed with FTSHD following a complete medical history review, examination, and playing observation, with 16 presenting symptoms in


the fingering hand and 5 in the bowing hand. A “triggering event” was noted in 12 cases, including change of technique, instrument, or teacher, increased practice, or minor trauma. Treatments administered included physical therapy, re-training, trihexyphenidyl, *botulinum toxin* injections, splint immobilization, and surgery. Only 6 of 16 patients affected in their fingering hand maintained their performing career and none of those whose bowing hand was affected continued to perform professionally. *Botulinum toxin* injections had virtually no benefit in string players while previous studies have shown that it is somewhat effective in woodwind and piano players with FTSHD. Schuele and Lederman postulated that the multilateral finger action required of string players is a factor.

The final case series to be considered here provides detailed summaries of 6 percussionists out of a total of 139 musicians diagnosed with focal dystonia at the Cleveland Clinic Medical Center for Performing Artists. Percussionists are among the least documented groups affected by FTSHD: Lederman identified only 21 cases reported in the United States and Europe. The study included five males and one female ranging from 21 to 51 years-of-age at onset of symptoms. Three were orchestral percussionists, two were jazz/rock drum set players, and one played drum set for country music groups. Symptoms primarily affected the wrist and forearm. Treatments administered included rest, re-training, physical therapy, body awareness techniques, trihexyphenidyl (THC), *botulinum toxin* injections, and limb immobilization. Three of the six returned to

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performance: one improved on a regiment of THC, another by restricting performance to mallet instruments only, and yet another after successful limb immobilization therapy.

Several studies cited previously considered the possible role of psychological factors in the development of focal dystonias in musicians, but detailed psychological analyses were not performed. A group of researchers from the Institute of Music Physiology and Musician’s Medicine (Hannover, Germany) recently published findings of just such an analysis, specifically comparing the effects of anxiety and perfectionism of musicians diagnosed with focal dystonia to those with chronic pain.\(^7\) Subjects consisted of 20 dystonic musicians, 20 musicians with chronic pain syndrome, and 30 healthy musicians. Participants completed four different questionnaires: the Freiburg Personality Inventory, the Questionnaire for Competence and Control Orientations, and two questionnaires designed specifically for the study that explored perfectionism and anxiety disorders, respectively. A statistical analysis of questionnaire results showed that musicians with focal dystonia displayed more anxiety than controls and those with chronic pain. The same group also demonstrated higher levels of perfectionism. Anxiety and perfectionism were concluded to be aggravating factors during the development of focal dystonias in musicians and further research into the role of the limbic system in such disorders was deemed necessary.

Another infrequently investigated topic in focal dystonia research is the effect of muscle fatigue on the presenting symptoms. One such study pertaining to musicians

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exists, published in the November 2001 issue of *Movement Disorders*. Because muscle fatigue reduces motor performance in healthy individuals and focal dystonia exhibits an increase of motor output, it was considered that fatigue-induced changes may be observable in individuals with focal dystonia. Subjects included ten musicians diagnosed with FTSHD (nine men, one woman), three musicians with a non-dystonic hand motor impairment, and five healthy musicians. The testing procedure consisted of three parts: first, a base-line assessment of the performance of a short musical passage; second, the subject tightly grasped a spring handgrip until the point of muscle failure; third, after a brief rest, the same musical passage was performed again. Performance assessment was based upon a rating system considering finger-movement accuracy, musical execution, amount of abnormal movements, and movement speed. Statistical analysis showed that musicians with FTSHD demonstrated improved motor performance for a period of five minutes after the fatigue test. The three musicians with non-dystonic movement impairment showed no improvement and the healthy musicians all demonstrated decreased motor facility after the fatigue test. Fatigue inducement was determined to have considerable value in the evaluation and diagnosis of FTSHD and that the development of similar therapeutic strategies may be possible.

Perception is another area of investigation that has been relatively unexplored in musician’s dystonia research. Lim and colleagues at the University of Hannover compared the perception of sequential stimuli by musicians with FTSHD and patients

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with writer’s cramp. Both auditory and tactile stimuli were used to investigate whether any timing problems were localized or general in nature. Subjects included two groups of patients: eight professional musicians—either guitarists or pianists—and five patients with writer’s cramp. There were three groups of controls: eight musicians and eight non-musicians for comparison with the dystonic musicians and five healthy controls for comparison with writer’s cramp patients.

Subjects underwent seventy-five trials each of tactile and auditory trials. Trials consisted of a sequence of six pulses, either auditory or tactile, in which the final pulse was irregular by a period between 200 and 300 msec. Subjects were asked to rate the timing on a scale between “1=definitely early” and “6=definitely late.” Statistical analysis of results showed that musicians with FTSHD were more accurate than all other groups when detecting early stimuli, both tactile and auditory, and less accurate than all other groups when detecting late auditory stimuli—with no significant difference in tactile stimuli. Patients with writer’s cramp did not demonstrate any significant difference from controls in either auditory or tactile domains. Lim concluded that musicians with FTSHD exhibit a general timing deficiency not present in patients with writer’s cramp suggesting a different pathophysiology of the two disorders. A follow-up study led by Dr. Lim investigated electrophysiological responses of musicians with

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FTSHD to simple auditory stimuli and found further evidence of general nervous system alterations of perception in that population.\textsuperscript{100}

The possibility of alterations of the somatosensory cortex presenting in focal dystonias was first explored in a primate model developed by Byl et al. in the late 1990s.\textsuperscript{101} Subsequent investigation of somatosensory representation in patients with focal dystonia has been undertaken and Byl identified several studies that explored this possibility in musicians. In 1998, Elbert et al. published their findings in such a study, using a 37-channel biomagnetometer to record trials of tactile stimuli administered to all digits of eight professional musicians diagnosed with FTSHD.\textsuperscript{102} Compared with controls, dystonic musicians presented a smaller distance, or fusion, between somatosensory representations of the fingers of their affected hands. Elbert postulated that a treatment strategy designed to reverse the fused representations would be effective.\textsuperscript{103}


\textsuperscript{103} Such a treatment, “Constraint-induced Movement Therapy,” was designed by Candia and will be discussed later in this chapter along with other experimentally investigated treatments.
In a smaller study comparing the somatosensory representations of a healthy flutist with those of a flutist with FTSHD, Byl found similar results.\textsuperscript{104} Not only were the locations of the dystonic patient’s finger representations distorted in relation to the healthy subject, but also—unlike Elbert’s findings—they were in the wrong order.

Neither study, however, determined whether alterations of somatosensory representation in musicians with FTSHD was a causative or consequential factor. A 2003 study led by McKenzie reinforced the findings of previous research, again showing a fusion of digits in dystonic patient’s somatosensory representations as well as an abnormal spatial ordering.\textsuperscript{105} These findings—along with similar results in studies of non-musician patients—suggest that neuroplasticity, the ability of the brain to adapt from training and experience, may play a role in the development of focal dystonias.

Documented evidence suggests that neuroplasticity is a continuous part of human development\textsuperscript{106} and several studies provide evidence suggesting that musicians demonstrate greater neuroplasticity than other populations.\textsuperscript{107} The most recent study


investigating alterations of the somatosensory representation in patients with focal
dystonia and the possibility of healthy neuro-plastic alterations derived from re-training
was published in the January 2008 issue of Neurology.\textsuperscript{108} Rosenkranz et al. explored the
ability of proprioceptive training in the form of varied pulse vibrations to alter abnormal
somatosensory organization in musicians with FTSHD and patients with writer’s cramp,
a possibility suggested by a previous study confirming the role of proprioception in task-
specific dystonias.\textsuperscript{109} Baseline assessment showed alterations in cortical representation
among dystonic patients as compared to controls. Post-treatment evaluation, however,
showed that musicians with FTSHD exhibited a more normal somatosensory
representation, whereas patients with writer’s cramp demonstrated no effect.
Rosenkranz concluded that the results further strengthened the case for different
pathophysiology (suggested by previous research)\textsuperscript{110} of the disorders and that similar
interventions should be explored in the treatment of musicians with FTSHD.

The work of Rosenkranz and colleagues demonstrates consideration of not only
somatosensory cortical representations, but sensorimotor cortex—the somatosensory
cortex and the motor cortex—function as well. Three other studies specifically examine

\textsuperscript{108} K. Rosenkranz, K. Butler, A. Williamon, C. Cordivari, A. Lees, and J.C. Rothwell, “Sensorimotor

\textsuperscript{109} K. Rosenkranz, S. Siggelkow, R. Dengler, and E. Altenmüller, “Alteration of Sensorimotor Integration

\textsuperscript{110} K. Rosenkranz, A. Williamon, K. Butler, C. Cordivari, A.J. Lees, and J.C. Rothwell,
the sensorimotor cortex as a whole in musicians with FTSHD. The first consisted of a functional MRI study of guitarists utilizing a specially designed instrument for imaging purposes.111 Guitarists with FTSHD demonstrated a significant increase in sensorimotor cortical activity when playing as compared to a control group. A separate study of guitarists in 2003 showed similar results with EMG data.112 Sensorimotor disruption was concluded to be possibly a result of “abnormal excitability or deficient inhibition in the basal-ganglia-thalamo-cortical network.”113 A later study by some of the same researchers confirmed these findings and further suggested a greater role of deficient inhibition.114

Before empirical studies of various treatment options applied to FTSHD are examined, a consideration of evaluation methods for musician’s dystonia is warranted. A review published in the February 2007 issue of Movement Disorders suggests that “the dearth of appropriately evaluated methods makes intervention studies of musician’s dystonia difficult to interpret.”115 The vast majority of studies of focal dystonia treatments employ rating systems, typically subjective, which use non-specific language


113 Ibid., 202.


(Table 4: Tubiana Scale, p. 25) and were not evaluated for validity and reliability. Examples of ratings scales include the Arm Dystonia Disability Scale (ADDS),\textsuperscript{116} the Tubiana and Chamagne Score,\textsuperscript{117} and the Burke-Fahn-Marsden Scale (BFM).\textsuperscript{118} To date, only three studies have been primarily concerned with the development of a valid and reliable quantification assessment tool for musicians with FTSHD.

The first, by Jabusch, Vauth, and Altenmüller of the University of Hannover, was developed specifically for evaluation of pianists with FTSHD.\textsuperscript{119} Subjects performed sequences of 10 to 15 C-major scales on a Kawai MP 9000 digital piano at a medium dynamic and legato style in sixteenth notes at a tempo of quarter note $= 120$ b.p.m. Scales were performed over two octaves, ascending and descending, hands separate. 

\textit{Musicator Win} software was used to record their performances and generate MIDI files and a special software program (MIDI-based Scale Analysis Software) was designed to measure key velocity, tone duration, inter-onset intervals between notes, and tone overlaps. Statistical analysis compared data from dystonic patients with controls and significant differences were identified between the two groups. Patients were then treated with \textit{botulinum toxin} injections and subjected to a follow-up assessment. Results at follow-up showed significant patient improvement. A second round of injections was

\begin{itemize}
\item \textsuperscript{116} S. Fahn, “Assessment of Primary Dystonias,” In \textit{Quantification of Neurological Deficit}, T.L. Munstat, ed. (Boston: Butterworth’s, 1989: 241-270) : 0=normal, 1=mild difficulty, 2=moderate difficulty, 3=marked difficulty.
\end{itemize}
administered after benefits decreased with time and follow-up results again verified improvements. MIDI-based Scale Analysis “was found to be an effective and reliable tool for quantification of focal dystonia in pianists and for monitoring treatment effects.”\textsuperscript{120}

The next study to report the development of an evaluation tool for FTSHD in musicians was geared toward wider application. Jabusch and Altenmüller explored “Three-Dimensional Movement Analysis” of a flutist with FTSHD.\textsuperscript{121} Colored markers were attached to the patient’s dystonic fingers and three digital video cameras were placed at varying angles throughout the testing area. The subject performed ten sequences of a five-note scale pattern (c-d-e-f-g, ascending and descending) in sixteenth notes at a tempo of quarter note = 120 b.p.m. Movement analysis of the third and fourth fingers was performed using SIMI Motion 3D software. The same procedure was carried out for six control subjects. Statistical analysis showed significant, specific differences between the patient and controls. The patient subsequently received four administrations of botulinum toxin injections over a six-month period and a follow-up assessment motion analysis confirmed significant improvement. Three-dimensional movement analysis was concluded to be a “useful parameter” in the quantification of FTSHD in musicians.

The final study concerned with development of a valid, reliable assessment tool for musician’s dystonia described a method in which the frequency of dystonic and

\textsuperscript{120} Ibid., 179.

compensatory movements are assessed by multiple raters.\textsuperscript{122} This Frequency of Abnormal Movements (FAM) rating method is based upon a paradigm of dystonic impairment in which fingers in flexion are considered “primary dystonic movement” and adjacent fingers that extend are considered “compensatory.” A single digital video camera recorded 18 subjects’ performance of two musical excerpts on their respective instruments (piano, trumpet, percussion, violin, flute, or clarinet). After initial assessment, patients underwent intensive “sensorimotor retuning” therapy, with sessions of three hours daily for seven days. Patients then completed six months of self-therapy followed by a second videotaped assessment.

Two raters, a health care professional and a professional musician, scored the frequency of abnormal movements of each subject using the FAM rating method. Additionally, the raters scored the videos using the aforementioned ADDA and BFM (see p. 63) methods with repeat viewings. While none of the assessment methods found any significant improvements in subjects’ performance after the applied treatment strategy, the FAM scale was found to have “good intra- and inter-rater correlation, concordance and internal consistency.” The authors acknowledged certain limitations of the study and provided future study designs to address these concerns. Despite the three tested methods discussed above, there is no standardized method for focal dystonia assessment, making evaluation of treatment methods difficult and accurate comparison of results among studies virtually impossible.

Of the various treatment strategies utilized in focal dystonia management, drug therapies are perhaps the most common. While previously discussed research, primarily large case-series reviews, has included results from such therapies, four studies were identified to have specific focus on pharmacological options for treating musicians with FTSHD. The first presents the case of a flutist reporting significant relief after taking pseudoephedrine, a common ingredient in over-the-counter decongestants.\textsuperscript{123} The patient was a 51-year-old female flutist diagnosed with focal dystonia of the right hand whose symptoms began after she experienced a ruptured cerebral aneurysm. Therapy with antihistamines was unsuccessful. The patient noticed a reduction of symptoms one morning after having taking a decongestant the night before. A double-blind placebo-controlled study was designed to determine if the effect was reproducible. Results showed that intake of pseudoephedrine or pseudoephedrine plus carbinoxamine produced significant improvements in performance as rated by two independent blind observers. It is noted that although improvement was observed the dystonic movement was not eliminated.

The remaining three studies report on large numbers of patients receiving trials of \textit{botulinum toxin} injections. The first, by researchers in the Motor Control Section of the National Institute of Neurological Disorders, included 18 patients diagnosed with FTSHD (14 men, 4 women) with an average age of 43.\textsuperscript{124} Injections were targeted to the affected muscles, located by EMG if clinical observation proved inconclusive. Improvement was


judged by each patient and based upon a subjective rating scale.\textsuperscript{125} Results of initial injections were positive: 7 reported major improvement, 6 moderate improvement, and 2 with minor improvement. By the end of the study period, however, 16 of the 18 patients had withdrawn, with only two reporting success at a long-term follow-up. The next study, again conducted by the Motor Control Section of the National Institute of Neurological Disorders, reviewed data of injection sessions of 120 patients with FTSHD.\textsuperscript{126} Results were similarly discouraging, with 59.4\% reporting no benefit from their last injection and 15.6\% reporting marginal benefit. Withdrawal rate was also high in this study and was attributed to dissatisfaction with treatment and inconvenience of travel to the NIH clinic.

The most recent study of botulinum toxin injection treatment of musicians with FTSHD was a joint effort between researchers at the Cleveland Clinic Foundation and Institute of Music Psychology and Musician’s Medicine (Hannover, Germany).\textsuperscript{127} Eighty-eight musicians diagnosed with focal dystonia—3 with embouchure dystonia, all others with hand dystonia—and treated with botulinum toxin injections between 1995 and 2002 underwent a retrospective chart review and a telephone survey utilizing a standardized questionnaire. Eighty-four patients responded to the survey: 74 men and 10 women.

\textsuperscript{125} 0=no improvement, 1=minimal improvement, 2=moderate improvement, 3=major improvement.


Patients were asked to rate their playing ability subsequent to treatment: 26 of 84 reported a decline or no change, 20 reported mild improvement, 25 moderate improvement, and 13 marked improvement. Six patients discontinued injections after four sessions because improvement was so dramatic that treatment was no longer considered necessary. In contrast to previous studies, this study provides evidence that long-term benefit is possible in more than just a handful of cases. Despite the relatively low numbers indicating effectiveness, botulinum toxin injections can yield a significant benefit for some patients. Because no studies reported any adverse effects other than localized temporary muscle weakness, such a treatment option seems worthy of consideration for affected musicians.

Although oral medications and/or botulinum toxin injections have been shown to be effective for a small number of patients, evidence from large case series, case studies, and other sources suggests that re-training therapies comprise the best options for treatment of musicians with focal dystonia. Since 1999, several studies have been published documenting the effectiveness of six distinct methods. The first documented method, constraint-induced movement therapy, was developed primarily by Taub as a rehabilitation method for stroke patients. Candia adapted this technique in the treatment of musicians with FTSHD.¹²⁸

In Candia’s initial study, three pianists and two guitarists with FTSHD underwent 1.5-2.5 hour sessions of therapy daily for a period of eight consecutive days. Non-dystonic fingers were immobilized with splints while the dystonic finger carried out

repetitive exercises alone or in coordination with other digits. Patients continued self-treatment at home for one hour daily combined with un-splinted normal repertoire practice. Results were measured with a dexterity displacement device and a subjective rating scale. All patients exhibited improvement in performance ability by the end of treatment. At final follow-up one non-compliant patient subsequently regressed, one showed stable results, and three experienced further improvement. Two reported a return to concert performances.

A subsequent study provided further details regarding the splinting procedure, included more subjects, and re-named the technique as Sensory Motor Retuning (SMR).[^129] Eleven musicians with FTSHD received the same treatment and evaluation protocols as described above: six pianists, two guitarists, two flutists, and one oboist. In this study, each patient exhibited one finger that was determined to be dystonic and one or two fingers that performed compensatory movements during performance in response to the dystonic dysfunction. Splints that immobilized the primary compensatory finger were custom-made for each patient, allowing for independent movement of the dystonic finger. Each of the six pianists and two guitarists demonstrated significant improvement as a result of treatment and four of those patients continued performance at a level close to pre-dystonic ability. The lack of success in the three wind players was attributed to two possibilities: one, that the finger-mouth coordination necessary for wind playing had altered somatosensory representation in a way that was not addressed by SMR, and two,
that the therapy does not account for both the force of holding the instrument and complex finger movements.

A third study headed by Candia employed MEG to examine the somatosensory representations of ten patients before and after SMR therapy.\textsuperscript{130} Prior to treatment, patients were found to have different representations in the affected and non-affected hands. Subsequent to treatment, the previously altered representations had apparently been somewhat repaired, changing to resemble the non-affected representation. These findings supported the behavioral improvement of patients after SMR therapy.

In a study published in 2003, Byl rigorously examined the outcome of a 12-week supervised treatment program aimed at sensorimotor re-training.\textsuperscript{131} Subjects consisted of three musicians diagnosed with FTSHD: two female flutists and one male bagpipe player. Evaluation methods consisted of MEG imaging, sensory, and motor tests before and after treatment; data was subject to statistical analysis and compared with age-matched controls. Treatment consisted of several components: first, patients were asked to stop all activity that produced the dystonic movements; second, a wellness program was implemented incorporating stress management and regular exercise; third, musculoskeletal problems were addressed through physical therapy and massage; and finally, a guided sensorimotor training program was begun.


The sensorimotor training consisted of “attended, goal-oriented, rewarded activities” that progressed in complexity over time. Training sessions occurred once a week for approximately two hours each and patients were given exercises to complete at home in the interim. Subsequent to treatment, patients demonstrated average improvement of 87% for somatosensory hand representation and 117% for target-specific performance tasks. Test results for fine motor skills, sensory discrimination, and musculoskeletal skills increased by 23%, 32%, and 32%, respectively. Although all three patients reported improved performance ability after treatment, one did not return to public performance. Of the other two, one continued to perform with a modified schedule, and the other completed conservatory studies uninhibited.

In the December 2003 issue of Medical Problems of Performing Artists, Tubiana published a study outlining a program of prolonged neuromuscular rehabilitation for musicians with FTSHD developed in conjunction with physiotherapist Phillipe Chamagne. This study examined the treatment results of 145 patients seen between 1992 and 1999 and summarized the rehabilitation process. Pre- and post-treatment assessment was based upon a standardized rating scale. Neuromuscular rehabilitation treatment consists of a series of four phases, each with a specific focus.


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The third phase, “Re-Teaching Movement of Posture,” develops and corrects specific complex motions, especially those utilized in playing an instrument. The final phase, “Return to the Instrument,” integrates work of the previous stages in a progressive manner. Practice in front of a mirror is employed as are orthotic devices on occasion. The total process lasts two years on average and is reportedly influenced by patient motivation. Of the 110 patient who finished rehabilitation, 25 showed no improvement and 85 showed some improvements. Of those 85, 39 showed significant improvement and return to concert performance. Although results were “far from satisfactory,” FTSHD was determined to be, in fact, “not incurable.”

A more simple and effective treatment method was developed by Sakai of the Biomechanics Laboratory at Utsunomiya University in Utsunomiya, Japan. Sakai published findings of a study of Slow-Down Exercise (SDE) in treating pianists with FTSHD in the March 2006 issue of *Medical Problems of Performing Artists.*

Twelve patients diagnosed with FTSHD—10 male, 10 female—with an average age at onset of 28 underwent a 5-step, 6-week program of SDE. Subjects were selected for inclusion only if their dystonic movements disappeared during playing at slow rates of motion. Assessment was based upon the Arm Dystonia Disability Scale (ADDS) and the Tubiana Scale.

SDE treatment consists of five steps. First, patients choose a piece, to be used during treatment, that provokes dystonic movement when performed at tempo. Second, performance speed of the chosen piece is reduced to a point at which symptoms disappear

and the resultant metronome marking is noted. Third, patients perform the study piece for a half-hour daily at that tempo. Fourth, if possible without recurrence of symptoms, tempo is increased up to 20%. Finally, after two weeks practice at the faster speed, speed is further increased as far as possible. On average, the final resultant speed attained was within 12.4% of the normal speed for each patient’s chosen repertoire. Post-treatment ADDS scores improved to normal (0) for 12 subjects and mild (1) for 8. Tubiana scale assessment improved from an average of 2.2 to 4.6. Despite the successful treatment method, patients reported “extreme dislike” for the exercises. Sakai concluded that “It is logical that the SDE employed in this study reduced the stratum of neural memories of physical movements to lower grades and repaired the memories associated with dystonia.”

Two additional treatment methods of musicians with focal dystonia have been reported in publication. One, a completely different approach than Sakai’s to treating pianists with focal dystonia, was documented in the September 2006 issue of *Medical Problems of Performing Artists.* In this study, three pianists underwent “Pianism Re-training,” a biomechanically-sound performing technique utilizing minimal tension. Subjects attended a minimum of 10 one-hour sessions, over a two-week period, that consisted of rigorous and detailed physical therapy while at the piano, concentrating on postural and movement awareness. The specifics of therapeutic practice were reported.

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134 Ibid., 28.

All three patients reported significant improvement after treatment as assessed by subjective rating scales and objective, blind aural assessment.

The final and most recently reported treatment method was developed by Farias and Sarti-Martinez of the International University of Andalucia.\textsuperscript{136} At the 2007 \textit{Congreso de la Sociedad Anatómica Espanola}, he presented findings regarding 90 musicians who underwent a six-month treatment period. All subjects were diagnosed with FTSHD and included guitarists (classical, flamenco, and electric), bass guitarists, violinists, pianists, violists, clarinet players, and flute players. The rehabilitation program consisted of “standardized slow and rhythmic finger, hand, and wrist movements” practiced for 30 minutes daily over a sixth-month period.

Assessments were made bi-monthly over the course of treatment through comparative analysis of recorded variables such as performance speed, time of performance without dystonic movements, playing-related loss of control, involuntary movements, and abnormal postures. Results were expressed as percentages of recovery: 4 reported 100% recovery, 11 reported 95% recovery, 39 reported 90% recovery, 20 reported 85% recovery, 13 reported 80% recovery, and 3 reported 0% recovery. Farias concluded that specific motion re-education is a key component in the treatment of musician’s dystonia. While reported results of this study are promising, details are lacking as to specific training and assessment methods employed, and further investigation is necessary.

Summary

Although the meager number of research studies (4) demonstrates that embouchure dystonia is an under-studied phenomenon, the literature reviewed provides valuable information. First, documented occurrences of FTSED in brass musicians are quite rare, but this may be due to several factors including: unfamiliarity with the disorder among physicians, neurologists, and brass players; unfamiliarity with the nature of brass instrument performance among medical professionals; a lack of standard testing procedures for diagnosis; the propensity of musicians to attempt to hide their symptoms rather than seek medical advice; and a general lack of research.

Additionally, evidence shows a prevalence of male patients with FTSED, which correlates with the evidence of male prevalence among musicians with focal hand dystonia. This apparent gender bias of FTSED remains unexplored. The studies by Lederman and Frucht reveal not only the ineffectiveness of current treatments, but also the lack of well-planned, documented and tested re-training programs, which are said to be the most effective treatment option available. Frucht’s study does, however, provide a clear and concise classification of different manifestations of FTSED, which may prove useful for physicians in determining a proper diagnosis.

The experimental studies reviewed provide a framework that further studies could readily follow. Hirata’s study shows that MEG can be an effective tool in FTSED research and provides evidence for the role of distorted sensory feedback as a contributing factor in embouchure dystonia. Reproductions of this study with larger numbers of subjects may help solidify the findings and yield further insights, particularly
pertaining to the role of plasticity in somatosensory representation. Although the EMG study by Iltis and Givens was lacking in number of patient subjects, the data collected suggests that EMG may be a reliable tool in the evaluation of embouchure dysfunction and FTSED in particular. The protocol that was developed, if proven reliable in other trials, could be applied not only as a diagnostic tool, but also in treatment.

FTSED research is certainly in its infancy and the literature examined in this study provides few definitive answers. What is provided, however, are models for study that can be reproduced which may lead to more answers and more well-focused questions. The most pressing needs highlighted by the current state of FTSED research are awareness among medical professionals and brass players, the development of standard diagnostic protocols, and the development and testing of embouchure re-training programs.

The preceding summation of empirical research pertaining to FTSHD in musicians shows that although this area of study is more developed than FTSED research, it too is lacking in many ways. Specifically, standard assessment tools that are valid and reliable have yet to be developed, making comparison of results among studies troublesome. Some treatments show great promise, but often are missing sufficient detail and studies of treatments lack replication. Additionally, the roles of anxiety and muscle fatigue have received limited inspection in FTSHD research. Continued investigation of these areas along with the application of relevant diagnostic techniques (such as MEG mapping of somatosensory representations) to FTSED will likely yield progressively improved results as scientific understanding of musician’s dystonia is expanded.
The next chapter contains a review of case study literature with regard to both brass players and other musicians. Nine instances of FTSED affecting brass musicians and documented cases of focal dystonia affecting 17 other musicians were identified. These case studies provide more individual patient characteristics than is available with empirical research, and the personal accounts often include descriptions of the subjects’ emotional reactions to their condition.
CHAPTER IV

CASE STUDIES OF FOCAL DYSTONIAS

Individual case study reports provide detailed information not always given in larger patient reviews and empirical studies. Additionally, personal patient accounts yield a different perspective than those offered by physicians and researchers. Both clinical case study reports and individual narratives will be examined in this chapter. It is hoped that further discussion of such narratives will encourage others with focal dystonias to share their experiences publicly. The literature reviewed will be divided into the following categories: studies involving brass musicians and studies involving other musicians.

Studies Involving Brass Musicians

As with empirical research of FTSED, literature concerned with case reports of the disorder is also limited. Nine case studies of brass musicians with FTSED were identified: six personal accounts and three clinical observations. Of the personal accounts, three are by horn players, one by a trombonist, and two by tuba players. The clinical accounts include a study of one trumpet player, one study of two horn players, and a study of one tuba player. The literature will be reviewed chronologically by type, personal or clinical, and analyzed for comparative features.
The earliest published individual account of FTSED was written by Peter Iltis and appeared in the September 2002 edition of The Instrumentalist.\textsuperscript{137} In this report, Iltis, a professor of horn and movement science at Gordon College, described the onset and development of symptoms, response to playing difficulties, and diagnosis of his own case as well as providing a brief overview of FTSED. The initial presentation of symptoms was traced to an increase in practice of scales and arpeggios at extreme dynamics in the middle and lower registers in an attempt to improve a perceived deficiency in low-register playing ability. These exercises were practiced for several hours daily and eventually led to an embouchure tremor, rendering sustained tones impossible. In an attempt to combat the development of tremors, Iltis increased warm-up time, although exact details were not provided.

As the condition worsened, the opinion of a performing arts medicine researcher was sought by Iltis, resulting in a diagnosis of FTSED. While the researcher apparently offered no suggestions for treatment, Iltis proposed that break times should be planned into practice—5 minute breaks for every 30 minutes of practice—and that practice should be planned to include varied exercises in every range. Additionally, Iltis discouraged intensive repetitions of exercises targeting technical weaknesses.

The next personal narrative of FTSED was written by accomplished tubist Ron Munson and published in the Spring 2003 issue of the International Tuba-Euphonium

Association Journal. Munson chronicled his nearly thirty years of struggle with what was ultimately diagnosed as FTSED with the stated purpose “... to help others make an early discovery of this most devastating condition in order to avoid wasting valuable time in getting on with their lives.” The first sign of playing difficulties appeared in May of 1972 in the form of a weakness in tone quality, inability to sustain low-register pitches and an involuntary tremor in some low-register playing. Symptoms progressed to include articulation problems and eventually led to an abandonment of tuba performance.

Munson was able to return to ensemble performance later through development of a new embouchure for the lower register and increased practice of up to five hours daily with no days off. Any reduction in practice was met with an increase of symptoms. Excessive frustration and depression were reported and other career options were explored, leading to the building of a private studio teaching all brass instruments. Munson mounted several attempts at returning to full-time performance between 1978 and 2000, trying various approaches to practice, including brief stints playing trumpet and horn. Although he was able to make several solo appearances, these were not considered complete successes and were manageable only with extensive practice time. In 2000, Munson contacted Frucht, who gave a diagnosis of FTSED based upon a video examination and questionnaire responses. Munson concluded that while it may be possible for those with milder cases to continue playing, individuals with excessive difficulty should use their imagination in fashioning a new career.


139 Ibid., 51.
Horn player Glen Estrin briefly recounted his experiences with FTSED in the Summer 2003 edition of *The Flutist Quarterly* in an article with Frucht that provided an overview of the disorder and information with regard to the Musicians With Dystonia Program (founded by Frucht and Estrin). While a freelance artist in New York City, Estrin experienced a lack of responsiveness in his lower lip. This unresponsiveness developed into muscle spasms in the lips and jaw areas while playing and became clinical within a period of six months. Several medical professionals were consulted, including oral surgeons, facial chiropractors, massage therapists, muscle therapists, and acupuncturists. Estrin received several diagnoses including TMJ syndrome and trismus, although no treatment prescribed offered any improvement. He then sought the opinion of a neurologist and was diagnosed with FTSED after a description of symptoms and history of their progression only. Estrin retired from horn playing as a result of FTSED. The article makes no mention of further examination or treatment strategies attempted.

The first published account of an individual recovering from FTSED was by Janine Gaboury-Sly in the February 2004 issue of *The Horn Call*.140 Gaboury-Sly first experienced performance difficulties in the fall of 1998 during a return to her regular duties as associate professor of horn at Michigan State University after a summer of “a very light performance and practice schedule.” In addition to her teaching schedule, she performed in concert with the Detroit Symphony and the Michigan Opera Theatre and completed preparations for two upcoming solo recitals. Those commitments, combined with regular faculty brass quintet rehearsals, resulted in some days with up to ten hours of

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rehearsal and performance. During this period of intense performance activity she noticed a twitch in her top lip while in a rehearsal. Although initially attributed to fatigue, this spasm developed into a more pronounced cramp, occurring while playing or when forming an embouchure without the instrument.

Gaboury-Sly was aware of FTSED because it had afflicted a colleague, Curtis Olsen, a few years earlier. As she became concerned that her symptoms were similar to his, she consulted with Olsen to ensure that she did not follow the same missteps that led to his eventual inability to even buzz a single note on the mouthpiece. Since Olsen had chosen to significantly increase practice time and focus on the particular issues that were troublesome, Gaboury-Sly decided to instead take some time away from playing. After three weeks off during the Christmas season of 1998, she began playing again, this time for a three-week run of a musical and a commitment to solo with the Michigan State University Orchestra. These performances were both considered unsuccessful and a subsequent period of rest was undertaken during the summer of 1999. At this point, Gaboury-Sly consulted Lederman, a neurologist at the Cleveland Clinic, and was diagnosed with FTSED. Lederman suggested a low dosage of the Parkinson’s drug Artane and starting over, re-learning to play horn with a new embouchure.

Gaboury-Sly did begin a regimen of Artane, but instead of learning a new embouchure, she developed a new practice routine in which she concentrated all mental focus on her lower lip and jaw. The idea was that relaxing the upper lip and complete focus on the lower embouchure would allow the upper lip to return to normal function. With this in mind, she practiced the warm-up routines in Verne Reynolds’s The Horn
Handbook\textsuperscript{141} on a daily basis and experienced minor improvements in a matter of days. Endurance and flexibility improved first, followed by extreme dynamics and middle-range slurs by the summer of 2000, although tone quality and accuracy remained problematic. In the fall of 2000, Gaboury-Sly returned to solo performance, sharing a recital with a colleague at Michigan State, and by 2001 she considered her recovery complete.

David Vining, professor of trombone and euphonium at Northern Arizona University, has documented his experiences with FTSED in an article published on his personal website.\textsuperscript{142} In this account, Vining describes his symptoms and the practices that led to his complete recovery over the course of four years. Vining reports being diagnosed with FTSED in 2002, although he does not mention the diagnosis procedure or the medical professional consulted. Symptoms presented included a closing of the lips at various junctures in playing, either at the beginnings of notes or in the middle, often causing a “hiccup” effect or the abrupt cessation of tone. The initial onset of symptoms began with “chipped” articulations of specific pitches that then spread to encompass a greater range. Eventually, public performance was abandoned.

Vining sought out non-medical treatment options, beginning with the study of Alexander technique, Body Mapping, and Feldenkrais therapy. He identified the single most important principle in these practices as “to retrain the affected area, retrain the whole body.” Vining also consulted with Kagarice, subsequently developing a new

\textsuperscript{141} Verne Reynolds, \textit{The Horn Handbook} (Portland, OR: Amadeus Press, 1997).

\textsuperscript{142} David Vining, “My Recovery from Embouchure Dystonia,” [article on-line]; available from www.davidving.net/narrative.html; Internet; accessed 21 January 2008.
definition of embouchure: “a three-dimensional entity in motion which only exists if fed by the airflow. The air blows the tissue into the right shape and size to produce the right note.” Utilizing concepts and exercises gathered from his study of movement therapies and work with Kagarice, Vining was able to return to public performance by September of 2006 and continues to maintain an active performance schedule.

In addition to the preceding case studies, a brief collection of three cases published online by the Spanish brass pedagogue Joaquín Fabra is worthy of mention.\textsuperscript{143} Fabra is reported to have recovered from FTSED through his own efforts, although no details of his experience are provided. Though it should be noted that Fabra is not a medical professional, he does claim to have helped several individuals diagnosed with FTSED to a full recovery. His “Embouchure Dystonia Dossier” includes short narratives describing three such individuals: a tuba player, a bass trombonist, and a trumpet player. All three musicians report a sudden awareness of a difficulty in sound production that deteriorated into an inability to continue public performance after increasing practice time to compensate for the difficulty. Symptoms described were consistent with other case reports of FTSED and all three described a full recovery after working with Fabra, although details of their recovery are not provided. In addition to these written accounts, “before-and-after” videos claiming to show players prior to and subsequent to working with Fabra are available online.\textsuperscript{144} The evidence of recovery provided in these cases is minimal, detailed descriptions of the exercises utilized are non-existent, and the writing is


\textsuperscript{144} See www.embouchuredystonia.com/videos.htm.
difficult to understand at best. Despite these limitations, the accounts published on Fabra’s website do serve as likely descriptions of FTSED.

The first clinical account of patients with FTSED was published in the January 1999 issue of *Movement Disorders* by Frucht, Fahn, and Ford of the Columbia Presbyterian Medical Center. In this report, details of the onset and progression of symptoms, treatment strategies, and treatment outcomes of two horn players with similar backgrounds are discussed. The first patient was a 26-year-old female, playing professionally, who began experiencing symptoms after a significant increase in playing time and difficulty of repertoire followed by an alteration of lead-pipe angle on the instrument. The second patient was a 20-year-old female student at a large music conservatory who first experienced symptoms after an embouchure alteration intended to improve tone quality. Both players were referred to the authors’ practice by the same individual: their teacher.

The presentation of symptoms in both patients was quite similar. The first patient demonstrated an involuntary upward pulling of both lips when playing or blowing into the mouthpiece, resulting in a separation of the lips and loss of seal with the mouthpiece. An upper lip tremor causing an audible shake in tone was also observed. The second patient demonstrated an involuntary upward pull of the top lip and downward pull of the bottom lip when playing or blowing into the mouthpiece, also resulting in a separation of the lips and loss of seal with the mouthpiece. In the case of the second patient, lip tremor was not observed, but symptoms worsened when playing in the lower

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register. Both patients exhibited a spilling of liquid from the corners of the mouth when attempting to drink from a soda bottle, but no other spreading of symptoms was indicated. Neither patient reported a family history of dystonia, trauma, or significantly problematic medical history.

The only treatment options offered to the patients were trials of oral medication, not specifically identified in the study, or botulinum toxin A injections. These options were declined by both patients. Instead, both patients underwent a re-training program suggested by their teacher consisting of embouchure alteration and practice with a trombone mouthpiece. Specifically, the patients employed what was described as a trombone embouchure, fixing the lips in a more down-turned position. This technique was considered moderately successful in the first patient because she had continued to perform, albeit with difficulty, in her professional orchestral position at the time of publication. The second patient experienced an improvement in her upper-register playing, but the lower register remained significantly affected. Specific details of the re-training process were not provided, or apparently investigated, nor was inquiry made into the specific practice regimens and pedagogical philosophies prescribed by the patients’ shared teacher. Embouchure re-training was concluded to offer the best hope for improvement in cases of FTSED.

The next clinical account of FTSED was published in the March 2000 edition of Movement Disorders, again by Frucht, Fahn, and Ford.146 This report described a 17-year-old female trumpet player diagnosed with FTSED, which presented after an incident

146 Steven Frucht, Stanley Fahn, and Blair Ford, “Focal Task-Specific Dystonia Induced by Peripheral Trauma,” Movement Disorders 15, no. 2 (March 2000): 348-350.
of peripheral trauma. The patient had begun study of the trumpet at age nine and anticipated a career in music performance. During a high school marching band rehearsal the bell of her trumpet was reportedly impaled with the slide of an aberrant trombone player, causing her mouthpiece to forcefully strike against her lips and teeth, resulting in significant pain and swelling of both lips. The symptoms of trauma reportedly subsided within a week and the patient returned to normal playing, although at a lower level of proficiency. Over the course of six months after returning to performance subsequent to the trauma incident, the patient’s playing deteriorated to the extent that performance became impossible. She was evaluated by an oral surgeon, dermatologist, and three neurologists with no diagnosis rendered prior to consultation at the Columbia Presbyterian Medical Center.

Examination revealed a pronounced tremor of both lips that manifested immediately upon tone production in all registers. Presentation of tremor was initially reported in the upper register and subsequently developed throughout the instrument’s range. Involuntary flaring of the nostrils and puckering of the lips were also observed. Dystonic symptoms did not spread to other tasks and the patient reported no pain. No family history of dystonia existed and the patient had no history of other oral trauma, dental problems, or an increase in practice and performance or change of instrumental technique. Treatment strategies included unspecified rest and embouchure re-training, both of which proved unsuccessful. The patient received counsel to pursue an alternative career path based upon her age and prognosis and it was not disclosed if she ceased all trumpet or musical study subsequent to diagnosis. This case documented an instance of
FTSED triggered by peripheral trauma. Frucht et al. argue that the repetitive, intense daily practice routines of musicians causes predisposition to focal dystonia, and that trauma to the specific body area most utilized in repetitive practice can increase this predisposition.\textsuperscript{147}

The most recent published clinical case report of FTSED was written by Kim, An, and Lee of The Catholic University of Korea and Kim of Hanyang University Medical College, published in the November 2007 issue of \textit{Movement Disorders}.\textsuperscript{148} This report describes a tuba player diagnosed with FTSED experiencing a temporary relief from symptoms after exposure of the affected area to cold temperature. The patient was a 22-year-old male attending a college of music and had been playing tuba for twelve years with a reported practice regimen of five hour-long sessions daily since beginning instrumental study. The onset of symptoms was preceded by a significant increase in playing time and difficulty of repertoire due to a college entrance examination. Initial symptoms presented as uncomfortable lip contractions in low-register playing, which progressed to involuntary tremors in both lips and a lateral pulling of the right side of both lips, evident in clinical observation. The patient was treated with oral medications (\textit{levadopa}, \textit{trihexiphenidyl}, and \textit{baclofen}) and \textit{botulinum toxin} injections with no improvement. Any additional treatments or the patient’s subsequent performance activities are not described.

\textsuperscript{147} The authors cite Nancy Byl et al., “A Primate Model for Studying Focal Dystonia and Repetitive Strain Injury: Effects on the Primary Somatosensory Cortex,” \textit{Physical Therapy} 77, no. 3 (March 1997): 269-284, as evidence for their hypothesis.

The possibility of cold temperature providing relief of the patient’s FTSED was prompted by a previous study of writer’s cramp. Needle EMG was used to measure the dystonic symptoms and the effect of cooling in the depressor anguli oris muscles. The tremor induced by tuba performance was a regular, rhythmic pulse between 1.5 and 2 Hz. The patient received a five-minute “ice massage” of the embouchure area and then attempted to play after an intermittent period of ten to twenty seconds. Tremor was not visible, audible, or evident in EMG data while playing after embouchure cooling. The relief of symptoms through cooling lasted for approximately one minute. No other sensory tricks, such as touch or pressure, produced any effect. Two possible explanations were proposed: first, that cold temperature reduced the muscle activity, counteracting the abnormal movements, or second, that the alteration of sensory feedback allowed the muscles to function normally.

Several important conclusions may be drawn from the case study literature described above. First, the initial onset of FTSED symptoms coincides with either an increase in practice/performance time and difficulty of repertoire, a change in instrumental technique or equipment, trauma to the embouchure area, or a combination of these factors. Second, an increase in practice time with focus directed on alleviating symptoms is not effective and typically accelerates the deterioration of playing ability. Third, correct diagnosis is often difficult to obtain—patients often receive multiple diagnoses by multiple medical professionals before FTSED is confirmed. Fourth, drug treatment and rest are ineffective. And finally, medical professionals have thus far failed

149 C. Pohl, J. Happe, and T. Klockgether, “Cooling Improves the Writing Performance of Patients with Writer’s Cramp,” Movement Disorders 17, no. 6 (December 2002): 1341-1344.
to adequately design and test embouchure re-training programs, despite published conclusions that such re-training programs offer the best chance for a patient’s recovery.

Of the nine studies reviewed, three reported individuals achieving complete recovery from FTSED. These accounts were individual narrative reports, however, and not written or clinically evaluated by medical professionals. Specific details of these recovery regimens were not outlined, although Gaboury-Sly and Vining do provide general descriptions. The documented cases of those not finding any relief or moderate relief of symptoms are also missing detail of any re-training regimens utilized. Also lacking is the consideration of the role that specific pedagogical philosophies and practice exercises might play in the development of FTSED. The literature available suggests that although the causes of each case of FTSED may be specific to each patient, similarities do exist in many areas, and such factors regarding the onset and progression of symptoms may be relevant.

Studies Involving Other Musicians

With one exception,150 published case studies of non-brass instrument playing musicians with focal task-specific dystonias all reported instances of hand dystonia (FTSHD). While the amount of literature detailing and studying these disorders is greater than the meager documented study of FTSED, it is still limited in scope. Case studies of seventeen individuals were identified: seven personal accounts and ten clinical observations. The personal accounts include those of the following instrumentalists: one

pianist, one violinist, three guitarists and two flutists. Clinical observations include studies of one Nadaswaram player, one pianist, one cellist, one violist, four guitarists, and one study of two percussionists.

Whereas only half of the personal accounts of FTSED reported successful treatment of symptoms, six of the seven individual cases of FTSHD reported successful return to concert performance. The most prominent of these individuals, and perhaps the most publicly visible of any patient suffering from any type of focal dystonia, is pianist Leon Fleischer. Fleischer’s case is well-documented and four brief articles discussing his experiences were identified for the purposes of this study.\textsuperscript{151} Additional newspaper and magazine articles have been published about Fleischer and he was the subject of an Academy-Award-nominated documentary, \textit{Two Hands}. In an effort to promote awareness of FTSHD, he has also given several radio and television interviews in recent years.

Fleischer first noticed symptoms in 1963 after an injury he incurred while “practicing Schubert’s ‘Wanderer’ Fantasy for eight or nine hours a day.”\textsuperscript{152} After several days of rest to heal his injured right thumb, he became aware of involuntary flexion in the third and fourth fingers of the same hand. The initial reaction was to practice more in an attempt to correct the problem, a course of action that ultimately exacerbated the symptoms. By the following year, symptoms had progressed to a clinical


\textsuperscript{152} Sacks, “Hand Delivered,” 69.
state and Fleischer had to abandon a concert tour of the Soviet Union with the Cleveland Orchestra. In 1964, FTSHD was an even less-known phenomenon than today and a correct diagnosis was not determined until nearly thirty years later. During that time, Fleischer tried a plethora of treatments, “from aromatherapy to Zen Buddhism,” none of which proved successful.\textsuperscript{153} By the 1970s, he had reinvented his career by teaching, by conducting, and also by performing literature written for left-hand alone.

Despite the severity of symptoms and the lack of a positive diagnosis, Mr. Fleischer continued to attempt to use his right hand to play on a daily basis, thinking “the way it came upon me might be the way it would leave me.”\textsuperscript{154} Although such a spontaneous remission never occurred—and has yet to occur in any documented case of focal dystonia—he did achieve a return to public performance, playing with both hands. After being diagnosed with FTSHD by Hallet, Fleischer agreed to a trial of \textit{botulinum toxin} and received his first injection in 1995. That same year he performed in concert with two hands for the first time since 1964, playing Mozart’s \textit{Piano Concerto in A Major, K. 414}. Fleischer credits the re-acquired use of his right hand when playing to not only the \textit{botulinum toxin} injections, but also to Rolfing\textsuperscript{155} therapy and maintains that despite his return to public performance that he is not cured.\textsuperscript{156}

\textsuperscript{153} Brubach, “A Pianist for Whom Never Was Never an Option,” 2.25.

\textsuperscript{154} Sacks, “Hand Delivered,” 70.

\textsuperscript{155} Rolfing is a holistic system of soft tissue manipulation and movement therapy founded by Dr. Ida Rolf, aimed to improve body functionality. See www.rolf.org for more information.

\textsuperscript{156} Isacoff, “Classical Musicians Suffer for Their Art,” D10.
Fleischer has continued to perform with both hands to this day, maintaining a regimen of *botulinum toxin* injections, Rolfing, and finger stretching before and after practice or performance. Over the course of his four-decade-long struggle with FTSHD, he has developed ideas about what may trigger the disorder and about the nature of music performance in general. He postulates the notion of three “personalities” of the performer:

Person A hears before they play. They have to have this ideal in their inner ear of what they’re going to try and realize. Person B actually puts the key down, plays and tries to manifest what person A hears. Person C sits a little apart and listens. And if what C hears is not what A intended, C tells B to adjust to get closer to what A wanted.\(^{157}\)

According to Fleischer, mindless repetition was the most important factor leading to his development of FTSED. He cautioned that “whatever you do with your fingers and your hands must be in the service of an idea . . . if you put the key down for a single note, unless you have a goal for that note, it’s an accident.”\(^{158}\)

Fleischer is not the only world-renowned musician to speak publicly about his affliction with FTSHD. Peter Oundjian, former first violinist with the Tokyo String Quartet, has discussed his experiences briefly in several interviews, although precise details of the development, progression and treatment of his symptoms are not readily available.\(^{159}\) Oundjian believes his dystonia was caused by a combination of an

\(^{157}\) Brubach, “A Pianist for Whom Never Was Never an Option,” 2.25.

\(^{158}\) Ibid.

overbooked schedule—up to 130 concerts annually with the Tokyo String Quartet—and a small maladjustment of his violin’s strings that changed their vertical alignment slightly.\textsuperscript{160} He also suspected that personality played a role in the development of FTSHD, specifically his own case and that of Leon Fleischer:

> When I think about Leon, with whom I have spoken a lot, I realize that we are in some ways similar personality types. Playing the music of Beethoven, Brahms, and Shostakovich, we put perhaps too much of ourselves into it. This situation (FTSHD) rarely happens to someone who keeps a more objective approach. You have to have your soul in the music, of course, but not every muscle of your body. It is so important to use minimum contractions.\textsuperscript{161}

Oundjian first began experiencing trouble with his hands in the late 1980s and reported that by 1993 a serious problem had developed. In 1995 he took a leave of absence from the Tokyo String Quartet and permanently left the ensemble, and violin performance, in 1996. He then proceeded to explore a career in conducting, finding success in guest appearances before earning a permanent post with the Nieuw Sinfonietta of Amsterdam in 1998. He continues a successful conducting career today as music director of the Toronto Symphony and has apparently foregone aggressive attempts at returning to violin performance.

Another prominent musician afflicted with FTSHD and the first to claim a complete recovery—the other being horn player Janine Gauboury-Sly—is guitarist David


\textsuperscript{161} Isacoff, “Classical Musicians Suffer for Their Art,” D10.
Leisner.\textsuperscript{162} Leisner first experienced difficulty with his right hand in 1984, specifically, painless flexion of the third, fourth, and fifth fingers, which progressed to the point that he was no longer able to continue public performance by 1985. He spent the next five years traveling across the U.S. to various medical professionals and other experts undergoing numerous treatment options, all of which proved fruitless. Having given up hope after these endeavors, he abandoned attempts to resolve the condition and focused on his career as a composer.

Some time later, Leisner accidentally discovered that he was able to perform most repertoire by using just the index finger and thumb of his right hand. In 1991 he performed his first public concert in six years utilizing this two-fingered technique. The following year, he had the idea to employ the larger muscle groups of the upper arm and shoulder in strumming the strings. This yielded amazing results: “within five minutes of doing this, I was able to use my ring finger that I hadn’t used for eight years.”\textsuperscript{163} Leisner continued to refine his large muscle technique and gained further use of his middle finger within a year. Another year of this practice led to the use of his ring finger in concert performance and he considered himself completely recovered by 1997. Contrary to the accepted understanding of FTSHD as a neurological disorder, Leisner believes the problem is a physical one. He stated, “I found out—and I’m quite sure of this now—that

\textsuperscript{162} Colin Cooper, “Journey of Discovery,” \textit{Classical Guitar} 15, no. 10 (June 1997): 11-17.

\textsuperscript{163} Ibid., 12.
the focal dystonia place is here, in the back of the shoulder, at the apex of where the arm meets the torso.”

Two additional brief accounts of guitarists with FTSHD come from Brazilian artist Badi Assad and Minnesota native Billy McLaughlin. While neither of these accounts offer much detail, they do serve as examples of musicians who found ways to continue their performance careers. Assad related that upon receiving a diagnosis of FTSHD that she initially decided to quit playing. She noted that the beginning of her recovery coincided with the belief that “the music is in me, not in my hands.” After a two-year hiatus beginning in 1994, she returned to public performance and recording in 1996. Assad was able to re-learn how to play guitar by cultivating new movements and hand positions that did not provoke the dystonic symptoms. McLaughlin first experienced difficulties in guitar performance after an unspecified injury to his left hand in 1997. By 1999, spasms in his left hand had made public performance impossible and in 2001 he was diagnosed with FTSHD. After five years away from playing, McLaughlin successfully attempted to re-learn the guitar while playing with the opposite hand. His new technique consists of using both hands, primarily the right to produce sound by tapping the fingerboard of an amplified acoustic guitar.

164 Ibid.
166 Mark Davis, “Fearless!,” 58.
167 Videos depicting this technique are available on McLaughlin’s website: www.billymacmusic.com.
returned to public performance at the end of 2006 and continues to maintain an active
schedule utilizing his new playing technique.

The first of two narratives chronicling a flutist with FTSHD was written by
Ernestine Whitman, a faculty member at Lawrence University. Whitman described the
onset of her affliction as a gradual loss of control of the distal joints in her left hand. This
problem was initially solved by plugging the holes of her flute’s A and G keys, then later
by utilizing finger splints to set her fingers in the proper position. This gradual loss of
control was followed by a more sudden development of involuntary straightening of the
second and third fingers of her left hand when attempting to depress the corresponding
keys. Whitman was pregnant during this development, and her obstetrician diagnosed
carpal tunnel syndrome which was expected to dissipate following the pregnancy. A year
later the symptoms had not subsided, and Whitman underwent carpal tunnel surgery.
Surgery was not successful and a series of visits to several hand specialists afforded no
relief. She eventually consulted doctors at the Mayo Clinic and was diagnosed with
FTSHD.

Following her diagnosis at the Mayo Clinic, Whitman tried two different
approaches to regaining her former playing ability. The first was a device created to hold
the flute in an attempt to allow for a new left-hand position. Unfortunately, the device
was not able to sufficiently support the weight of the flute. The second approach was a
hiatus from the flute, lasting about one year, in which time she took up study of the
bassoon. While playing the bassoon was helpful at first, Whitman reported that once she

was able to play scales and arpeggios easily her symptoms returned. The setbacks led to severe depression, which was eventually alleviated with the help of a therapist.

Whitman then turned to Pascarelli, of the Miller Health Care Institute for Performing Artists at Roosevelt Hospital in New York, who suggested that she make some physical adjustments to her flute to allow for a more natural hand position. The result was a clip-on attachment that allowed her wrist to remain straight while still being able to hold the flute effectively. She then began to re-learn hand technique, slowly, under the supervision of another doctor at the clinic. Although Whitman did not claim 100% recovery—she uses the middle of her fingers, instead of fingertips, to depress the G and A keys—she was able to return to a full performance schedule in just over a year’s time. Whitman’s narrative provides a detailed account of the psychological challenges of a musician afflicted with focal dystonia and a creative solution to the problem. She concluded:

While my case may be extreme, I do think all of us too readily allow our sense of dignity and self-worth to be dependent upon our success as musicians. . . . I hope that I carry with me this newfound conviction that one’s value as a person exists quite apart from one’s ability as a performer.  

The second account of a flutist with FTSHD was written by Roger Martin, professor of flute at Tennessee Technological University. Martin first experienced symptoms in his left hand, described as “wooden and unresponsive,” while preparing to

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169 Ibid., 48.

play the Leibermann *Concerto*, a work he had performed in concert several times previously. He managed to complete his concerto engagement “with desperate determination” and subsequently consulted a hand specialist who diagnosed FTSHD and explained that there was no treatment that was known and that the symptoms would continue to progress. Though Martin did not divulge further details of symptoms or any prior medical consultation, and did not claim to be cured, he did outline specific practices that have allowed him to continue playing professionally.

Martin realized that the development of FTSHD had resulted in a complete focus of attention on the physical aspects of playing and that prior to symptoms he had split his attention equally between physical sensation and the sound produced while playing. As the growing focus on the physical problem had exacerbated symptoms, he tried a radically different approach: “When I ignored my hands and put all my attention on hearing the motion of pitches, there was improvement.”

This insight prompted him to explore directed attention further, first by writing a script that was read to him in a hypnotherapy session and repeated aloud during subsequent daily practice, and second by developing practice methods to hone “tonal imagination.”

These methods included singing practice materials with accurate pitch in long tones (minus rhythm), singing phrases as written with exaggerated dynamic shaping, an exercise from *Top-Tones for Saxophone* by Sigurd Rascher, and imagining all aspects of sound (color, volume, vibrato) prior to tone commencement. Rascher’s exercise, entitled

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171 Ibid., 31.

172 “I now hear each note clearly before I play it and allow my body to produce the music in the most effortless way possible.”
“Tonal Imagination,” consisted of playing long tones in patterns of ascending fifths and descending fourths. As each tone was first imagined and then played, the next tone was then imagined for a time before moving physically to the new pitch, refining the ability to concentrate on inner hearing rather than reacting to the auditory sensation of the actual sound produced. Martin also discussed the psychological part of the recovery process, suggested that taming one’s inner dialogue is an important factor in improvement. He noted that critical self-analysis while playing detracts from focus on the desired sound. “Make a commitment to improving your tonal imagination,” he suggested, “the results can only be rewarding.”\(^{173}\)

One clinical study of FTSED exists documenting a case in a professional Nadaswaram player.\(^{174}\) The Nadaswaram, or Nāgasvaram, is a conical double-reed instrument, resembling the oboe, utilized in traditional Indian music.\(^{175}\) The subject was a 72-year-old male who had started playing the instrument at age 15 and first experienced symptoms at age 69. The initial symptoms were task-specific tremor of the lips and right forearm causing a disruption in tone while playing. A year after presentation of these symptoms, postural tremors of the head and upper limbs developed, later diagnosed as essential tremor. A 2-mg daily dose of clonazepam—an anticonvulsant typically used to

\(^{173}\) Ibid., 33.


treat epilepsy\textsuperscript{176}—resulted in a 60% improvement in the patient’s resting tremor symptoms. EMG was used to record lip and right forearm muscle activity both at rest and while playing.

The EMG data collected showed no tremor while the subject was at rest. When the instrument was brought near the mouth and while playing, a 5-5.5 Hz rhythmic pulse was observed in the right forearm. Increased EMG output was recorded as irregular pulse in the lip muscles only while playing. On occasion, the forearm and lip pulse were synchronous. The patient had a positive family history for essential tremor, but not task-specific tremor or dystonia. No report of other injury or increased performance activity prior to initial onset of symptoms was mentioned. No treatment of the FTSED is mentioned and the subject continues to experience symptoms. Ragothaman conclude that “our patient provides an important pathophysiological link between task-specific tremor, essential tremor, and task-specific dystonia.”\textsuperscript{177}

The lone case report of a pianist with FTSHD documents the possible effectiveness of Δ9-tetrahydrocannabininal (THC), the main active ingredient of cannabis, as a treatment option.\textsuperscript{178} The subject was a 38-year-old male professional pianist with a ten-year history of FTSHD. He had ceased public performance six years prior to the present study although daily practice had been maintained. Specific symptoms included


\textsuperscript{177} M. Ragothaman, et al., “Embouchure Dystonia and Tremor,” 2134.

flexion of the third, fourth and fifth fingers of the right hand. The subject had undergone unsuccessful treatment previously with trihexyphenidyl and botulinum toxin. The lack of successful treatment options led to a search for novel approaches. THC had been found to have anti-dystonic effects in an animal-based trial\(^\text{179}\) and in anecdotal reports of musicians experiencing temporary relief from dystonic symptoms after smoking cannabis. Additionally, THC has been found to be safe and to have no neuropsychological effects at dosages of 10 mg daily.\(^\text{180}\)

A placebo-controlled single-dose trial was undertaken, with two days of testing separated by a seven-day interval. The testing sessions consisted of an initial playing assessment, then the administration of THC or placebo followed by a three-hour playing period with rest breaks every 45 minutes. The playing analysis measured motor control of the fingers as assessed through MIDI-scale analysis and was carried out 30, 60, 180, and 300 minutes after administration of THC or placebo.\(^\text{181}\) Specifically, ten to fifteen C-major scales were played on a digital piano by each hand separately to a set tempo.

The initial playing assessment showed impaired motor control in the affected hand, with normal readings for the unaffected hand. After medication with THC a significant improvement in motor control was observed and measured, with the effect slowly decreasing after 180 minutes. The unaffected left hand showed no alteration in


performance. The subject was able to play difficult literature that had been unplayable before treatment during the first two hours after THC administration. During the testing session in which a placebo was administered, the subject showed no improvement in motor control. THC intake was concluded to be a potentially useful treatment option for musicians with focal dystonia and further study was suggested. A difference in the effects of THC on musicians with focal dystonia and patients with generalized and segmental dystonia was hypothesized to indicate a possible difference in pathophysiology.

In contrast to treating FTSHD with medication, Ackermann and Adams explored the role of proprioception in the case study of a professional cellist.\textsuperscript{182} The purpose of this study was to evaluate finger movement discrimination by testing proprioceptive sensation with a unique psychophysical method. The subject was a 47-year-old male with a three-year history of FTSHD. Initial symptoms were described as a loss of fluency in performance, which progressed to observable involuntary movements of the second and third fingers of the left hand. Playing difficulty eventually resulted in cessation of public performance and the subject was subsequently diagnosed with FTSHD by a neurologist. To test the processing of proprioceptive information, the authors built a “pseudo-cello” mimicking the dimensions of a real cello, but allowing for the

manipulation of string height and tension on one string (the D string) by a motorized rod.\footnote{Ibid.}

The subject sat with the “pseudo-cello” in a normal playing position and was instructed to press the tensioned string with a specified finger and judge the tension on a scale of 1 to 5, with 5 being the most tension. String height and tension was determined randomly by a computer program, as was the order of fingers tested. Each finger underwent fifty evaluations. After the initial testing session, the subject underwent an intensive ten-day sensorimotor re-training period with Ackermann and a cello teacher—the latter of which had apparently cured himself of FTSHD after eight years of self-retraining. The subject’s re-training period included sensory discrimination training (Braille identification, stereognosis, texture sensitizing), constraint-induced movement therapy, a general conditioning program, and cello-specific technique exercises. A final testing session with the “pseudo-cello” was then administered.\footnote{Ibid.}

The results of each testing session showed an improvement in string tension perception in the subject’s second, third, and fourth fingers (left hand) after the period of sensorimotor re-training. Additionally, the subject reported an improved score on the Candia dystonia evaluation scale.\footnote{Victor Candia, et al. “Constraint-Induced Movement therapy for Focal Dystonia in Musicians,” \textit{The Lancet} 353, no. 9146 (January 1999): 42.} Tests that require less than full playing movement, such as the one devised in this study, were suggested to be valuable in the assessment and treatment of focal dystonias. Ackermann and Adams acknowledged that the relevance of
their data has yet to be determined and that a control subject was not utilized in the study. Ongoing study was planned, aimed at addressing these issues and determining possible further uses of the testing procedure in diagnoses and treatment.\textsuperscript{186}

In the November 1997 issue of \textit{Manual Therapy}, physiotherapist Jane Kember reports a case of FTSHD in a professional violist who was successfully treated with physical therapy.\textsuperscript{187} The patient was a 30-year-old male who demonstrated an uncontrollable, painless, flicking of the thumb in his right hand when playing—specifically, when beginning a bow stroke—resulting in an unstable grip on the bow. He had been diagnosed with FTSHD at a performing arts clinic and referred to the author’s practice for treatment. The player was able to play for three to four minutes at a moderate tempo before expression of symptoms, although symptoms presented more quickly at slower tempos. Symptoms were also observed when the patient attempted to pick up a cup. Playing difficulties were initially experienced five years prior to treatment, after a period of increased performance, teaching, and practice. The patient quit playing for one year and subsequently changed his teacher in an attempt to correct a perceived improper technique. A return to playing with a new technique produced minor improvement, but a recurrence of symptoms to various degrees again resulted in the abandonment of performance. A severe depression was reported.

The patient previously had been examined by a neurologist, treated by a chiropractor, and treated by a physiotherapist who prescribed weightlifting and shoulder

\textsuperscript{186} Ibid. 

exercises. He also was examined by two additional medical professionals who concluded that there was no underlying pathology. Kember confirmed the previous diagnosis of FTSHD following examination and identified many alterations from normal joint and movement function, including postural abnormalities, limited cervical spine movement, muscle imbalance in the shoulder girdle and decreased movement in the right carpo-metacarpal joint (wrist/fingers).\textsuperscript{188}

A regimen of intense physical therapy was implemented that consisted of four sessions of hands-on treatment and the application of self-performed exercises. Details of each session were provided, improvements were observed by the third session, and by the fourth session the flicking motion of the right thumb had been reduced to the mere presentation of tension, although complete control was not achieved. The patient continued with prescribed therapeutic exercises as well as a warm-up/warm-down sequence of exercises before and after playing and reported a limited return to professional performance two weeks after the end of treatment. Within a few months, the patient had resumed a full-time performance and teaching schedule.\textsuperscript{189}

Perhaps the earliest published case study of a musician with focal dystonia was written in 1997 and reported in the \textit{Journal of Behavior Therapy and Experimental Psychiatry}.\textsuperscript{190} In this account, Peter Roxburgh describes a patient with symptoms resembling a description of FTSHD, and although that precise terminology is not used,

\textsuperscript{188} Ibid.

\textsuperscript{189} Ibid.

the diagnosis given is occupational cramp, a term used in more recent literature as interchangeable with focal dystonia. The patient was a 35-year-old self-taught male guitar player/teacher with a 10-month history of progressive cramp in the right hand—only when playing—that had resulted in an inability to perform publicly. Specifically, when attempting to play individual strings the fingers involuntarily extended and the wrist flexed. The onset of playing difficulty occurred after a car accident and coincided with a career move to become a full-time professional guitarist and a reported difficult personal relationship situation. Prior treatments, all unsuccessful, included psychotherapy, acupuncture, chiropractic manipulation, and physical therapy.191

Roxburgh postulated that the patient’s involuntary hand spasms were the result of a conditioned anxiety response brought about by the patient’s attempts to increase performance ability too rapidly. Intensive focus on physical relaxation was thought to divert attention from the anxiety response and perhaps improve motor function. To that end a two-step training program was implemented. The first step was a guided relaxation session in which the patient was directed to cultivate sensations of “relaxation, warmth, and heaviness.” A tape recording was made of the verbal directions for use in at-home daily practice. The second step was to hold the guitar while maintaining the achieved relaxed state through directed mental focus. Progressively more difficult musical exercises were performed, with frequent rest periods during which the guided relaxation technique was repeated. This process was carried out in a total of three treatment sessions, and a complete remission of symptoms was observed by the final session.

191 Ibid.
Success was attributed to “the explicit instruction to adhere to a positive criterion for the
maintenance of relaxation rather than awareness of incipient anxiety.”\textsuperscript{192}

The second of four case reports of guitarists examined in this study appeared in
the October 2005 edition of the \textit{Journal of Clinical Rheumatology}.\textsuperscript{193} The patient was a
56-year-old male professional guitar player with a ten-year history of painless
involuntary contractions of the thumb, second, and fourth fingers while playing. The
progression of symptoms eventually resulted in discontinuation of public performance
and coincided with the development of several soft tissue masses in the forearms,
subsequently found to be benign lipomata, or tumors. A few years after onset, dystonic
symptoms spread from guitar playing only to interference with daily tasks such as
writing. Both the patient’s personal and family medical history were reported as
unremarkable.

The standard physical and neurological examinations of the patient proved normal
with the exception of the lipomata and FTSHD. Additionally, EMG data was recorded
and an ultrasound examination administered. EMG confirmed abnormal contractions of
the extensor muscles of the right forearm and ultrasound revealed no nerve entrapment
evident from the lipomata. The patient had been previously diagnosed with nerve
entrapment syndrome, carpal tunnel syndrome, psychosomatic disorder, and Parkinson’s
disease. Treatments administered previously included \textit{risperidone} (an antipsychotic drug
often used to treat schizophrenia), \textit{clonazepam, levodopa, chlorzoxazone} (a muscle

\textsuperscript{192} Ibid., 218.

relaxer and pain reducer), anti-inflammatories, vitamins, herbs, and physical therapy. No additional treatment was offered and the patient’s eventual outcome was unclear.\footnote{Ibid.}

A more clear, yet unsuccessful outcome was reported in a case of FTSHD by Dillon, Higgins, and Curtin in the \textit{Irish Medical Journal}.\footnote{J.P. Dillon, T. Higgins, and J. Curtin, “Focal Dystonia in a Professional Musician.” \textit{Irish Medical Journal}, IMJ Online [article on-line]; available from http://www.imj.ie/Issue_detail.aspx?issueid=+&pid=2738&type=Contents; Internet; accessed 7 January 2008.} A 39-year-old professional male guitarist was referred to their practice with a six-year history of difficulty controlling the right-hand thumb while playing. Upon attempting to strum the strings, the patient’s thumb was observed to involuntarily flex inward. Symptoms had progressed to the point where the patient was unable to continue a performance career. Clinical examinations and radiological evaluation of the hand both showed normal results, and the presentation of symptoms led to a diagnosis of FTSHD. A six-week trial of thumb immobilization in a cast yielded no significant reduction of symptoms. The patient was then referred for neurological evaluation and subsequently underwent EMG guided \textit{botulinum toxin} injections which also proved ineffective at an eight-week follow-up.

The most recently published account of FTSHD in a guitarist also reports unsuccessful treatment, as well as a likely example of trauma as an etiological factor.\footnote{J.N.A.L. Leijnse, and M. Hallet, “Etiological Musculo-Skeletal Factor in Focal Dystonia in a Musician’s Hand: A Case Study of the Right Hand of a Guitarist,” \textit{Movement Disorders} 22, no. 12 (2007): 1803-1808.} The patient was a 44-year-old male classical guitarist with a seven-year history of FTSHD diagnosis and several unsuccessful treatments. Symptoms presented as a painless involuntary flexing of the thumb and index fingers toward each other.
Symptoms were restricted to guitar playing and found to occur primarily in slower playing and not in more rapid performances. The involuntary thumb contractions occurred most often when the thumb was not involved in string activation. Initial examination showed no evidence of nerve compression, musculo-skeletal abnormalities, anatomical limitations, or joint damage. During questioning, the patient revealed a deep splinter wound which punctured the web-space between the thumb and index finger, incurred while cleaning a wooden floor two years prior to the onset of symptoms.

The patient was subjected to a detailed functional evaluation of the right thumb, which revealed a slight misfunction at the metacarpophalangeal thumb joint—in the middle of the thumb. Specifically, the joint was not able to flex independently of the other thumb joints, in contrast to such an ability in the left thumb. The conclusion reached was that the splinter trauma had incapacitated the right-hand flexor pollicis muscle. This abnormal function was thought to have been missed in prior examinations, due to normal-appearing thumb function and the patient’s trauma experience being previously unreported. The dystonic muscular contractions likely developed as a compensatory mechanism for the loss of motion caused by the incident of trauma. Specifically, the loss of the ability to maintain equilibrium in the thumb explains the prevalence of symptoms in slow playing, where the patient was attempting to maintain a static thumb position.¹⁹⁷

The possibility of re-training thumb use in slower playing to more closely resemble the fluid motion present in faster playing was considered, but dismissed without

¹⁹⁷ Ibid.
attempted treatment due to the notion that without repair to the functional disability such re-training would be impossible. Additionally, surgery, joint immobilisation, and *botulinum toxin* injections were considered as possible options but rejected for various reasons without attempted treatment. The patient sought treatment elsewhere, but these endeavors, not specified, proved unsuccessful and he eventually discontinued performance activities.\(^{198}\)

One case report of FTSHD affecting percussionists was identified and chronicles the disorder as evident in the right hand of two professional tabla players.\(^{199}\) The first patient was a 32-year-old male, with 21 years of professional experience, exhibiting an inability to extend second, third and fourth fingers of the right hand when attempting to play and reporting pain associated with this dysfunction. This motor impairment was not present during other tasks. Normal movements of the left hand during tabla playing, however, resulted in involuntary extension of the right thumb and flexion of the affected fingers. The patient had no family history of neurological disease, and examination was otherwise normal. A treatment of *botulinum toxin* injections was prescribed. The patient reported a reduction in pain after one month and the return of normal playing ability after four months, resulting in a return to public performance.

The second patient was a 47-year-old male, with 25 years of professional experience, reporting a two-year history of lateral tremor of the right hand while playing the tabla. In addition to task-specific tremors, the patient also demonstrated involuntary

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\(^{198}\) Ibid.

flexion of the first, second, and third fingers in the right hand during rapid movements made by the left hand. He was able to write and perform other tasks normally, and pain was not reported. The patient had no family history of neurological disorders, and the examination was normal apart from the described symptoms. A regimen of botulinum toxin injections was prescribed that proved only minimally effective. No other treatments were reported.  

**Summary**

Several important observations may be made from the examination of the case study literature described. First, the initial onset of FTSED symptoms can be traced to coincide with either an increase in practice/performance time and difficulty of repertoire, a change in instrumental technique or equipment, trauma to the embouchure area, or a combination of these factors. Second, an increase in practice time with focus directed on alleviating symptoms is not effective and typically accelerates the deterioration of playing ability. Third, correct diagnosis is often difficult to obtain as evidenced by patients often receiving multiple diagnoses by multiple medical professionals. Fourth, drug treatment and rest are generally ineffective. And finally, medical professionals have failed to adequately design and test embouchure re-training programs, despite published conclusions that such re-training programs offer the best chance for a patient’s recovery.

Of the nine studies of brass musicians with FTSED reviewed, three reported individuals achieving complete recovery from FTSED. These accounts were individual narrative reports, however, and not written or clinically evaluated by medical

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200 Ibid.
professionals. Regardless, specific details of these recovery regimens were not outlined, although Gaboury-Sly and Vining do provide general descriptions. The documented cases of those not finding any relief or moderate relief from symptoms are missing details of any re-training regimens utilized. Also lacking is the consideration of the role that specific pedagogical philosophies and practice exercises might play in the development of FTSED. The literature available suggests that although the causes of each case of FTSED may be specific to each patient, similarities do exist in many areas, and such factors may be relevant.

As noted with case studies of FTSED in brass musicians, the initial onset of FTSHD symptoms can usually be traced to coincide with either an increase in practice/performance time and difficulty of repertoire, a change in instrumental technique or equipment, trauma to the affected hand, or a combination of these factors. Also, an increase in practice time with focus directed on alleviating symptoms is not effective and typically accelerates the deterioration of playing ability. Additionally, correct diagnosis is often difficult to obtain as evidenced by patients receiving multiple diagnoses by various medical professionals. In contrast to FTSED of brass musicians, some FTSHD cases have been documented to respond positively to either physical therapy or botulinum toxin injections.

The case study literature regarding focal task-specific dystonias afflicting musicians provides examples of successful recoveries, dismal failures, and the dramatic effect the disorder can have on one’s career. What seems missing in many cases, however, are specific details that may yield insight into possible causes and a viable
course of treatment. It seems clear from the evidence provided that each case is extremely subjective and should be evaluated and treated as such. That said, the development of a systematic evaluation process as well as a systematic progression of treatment options may prove beneficial. For example, in the literature surveyed, only one report documented the specific line of questioning employed in the assessment of focal dystonia. Application of a tested and refined standard questioning procedure could aid in identifying possible causes, complicating factors, and additional information that could enhance the treatment process. Regardless, case reports provide a useful perspective in focal dystonia research and continued investigation is needed.

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CHAPTER V
CONCLUSIONS

Summary of Key Points

The purpose of this study was to promote awareness and understanding of focal
task-specific embouchure dystonia (FTSED) among brass musicians. An examination of
current scientific, medical, and professional literature was undertaken in an attempt to
answer the following questions:

1. In the simplest terms possible, what is FTSED?

FTSED is a neurological movement disorder affecting the facial muscles utilized
in brass instrument tone production in which abnormal involuntary movements
occur when playing, inhibiting performance ability to varying degrees.

2. What are the symptoms of FTSED and are there any “warning signs” that can aid in
early detection?

Perhaps the most useful classification of symptoms is given by Dr. Steven Frucht:
lip tremor, lateral pull, lip lock, and involuntary jaw movements. As symptoms
initially present as common playing problems, they can be quite difficult to detect.
Potential warning signs may include inexplicable playing difficulties of a kind not
previously encountered, inexplicable decline in playing ability despite increased
or steady practice, and small abnormal facial movements that interrupt or inhibit
playing to a significant degree.

3. What causes FTSED?

The exact causes of FTSED are unknown. The most likely theory is that the
disorder is a result of maladaptive neuroplasticity. In other words, the brain
develops a dysfunctional response as a consequence of distorted or damaged
sensory-motor integration. Research has identified several factors that may
contribute to the development and progression of symptoms: perfectionism,
stress and anxiety, increase in practice and performance time, increase in difficulty of repertoire, dramatic life event, and psychological or physical trauma. The specific possible influences of these factors are undetermined.

4. How is FTSED diagnosed and treated?

Definitive diagnosis is best attained from a neurologist familiar with the disorder, although other medical professionals accustomed to the treatment of musicians may be able to provide positive diagnosis as well. The diagnosis is typically determined from a combination of physical examination, neurological examination, and playing observations. No definitive or standard testing procedure exists. Treatments include oral drug therapies, botulinum toxin injections, unspecified re-training regimens and various alternative therapies. The only treatment reported to be effective is pedagogical re-training. However, documentation of the details and effectiveness of such programs is non-existent.

5. Can FTSED be prevented?

Current research does not address the possibility of FTSED prevention. Since body use, practice habits, pedagogical techniques, and personal, physical, and psychological health seem to play a role in the progression of symptoms, addressing these areas may be wise preventative measures.

6. What is the state of current research concerned with FTSED?

This study identified fewer than fifteen sources specifically concerned with FTSED. While more thorough investigation of FTSHD exists and the body of literature studying dystonia in general is growing, FTSED research remains limited. This is likely due to the low number of reported cases, difficulty in studying embouchure musculature, the unfamiliarity of medical researchers with brass instrument performance, and the unfamiliarity of brass players and pedagogues with FTSED.

Focal task-specific embouchure dystonia is a devastating condition that can afflict professional musicians, students, and amateurs alike. Often career-ending, FTSED has no known cure and medical research and insight with regard to the disorder are limited. Unfortunately, trends in brass pedagogy may contribute to the development of embouchure dystonia. Despite this seemingly bleak outlook, new medical and
pedagogical developments in recent years offer hope for victims of FTSED. Embouchure dystonia may, in fact, be treatable and preventable, yet clinical studies must be undertaken to test such assertions. Improving the prognosis for FTSED and facilitating rehabilitation necessitates increased awareness among performers and teachers, a re-thinking of brass pedagogy, and the development and testing of effective treatment programs. A determined effort to accomplish these goals is needed in order to provide effective solutions for embouchure dystonia.

Suggestions for Further Study

The most pressing issue regarding FTSED is awareness among brass players and pedagogues, since it is unlikely that extensive medical research will be undertaken without the impetus, interest, and expertise of the brass community. There has been no published research assessing brass players’ and pedagogues’ current knowledge and understanding of FTSED. While the present study aims to increase awareness through the simplification of concepts, a summation of research, and presentation of a case study, one logical next step would be to assess awareness in the brass community and determine and implement appropriate awareness strategies.

The development and testing of embouchure re-training programs is another pressing issue that deserves attention. While current research suggests that such programs are the only treatment option offering any benefit, there is little information available. FTSHD treatment regimens may serve as a model for study, particularly those developed by Byl (2003), Candia (2002), and Tubiana (2003). Although specific hand exercises do not apply, the organization, treatment components, and general philosophies
of those studies are certainly transferable. Additionally, some individual accounts, such as documented in this study, offer insights into possible successful treatment designs. Anecdotal reports of individuals who have succeeded with re-training have been encouraging. However, until embouchure re-training programs are thoroughly outlined, tested, and publicly reported, recovery from FTSED will likely remain a substantially difficult endeavor.

Conclusions

A consideration of the current treatments for embouchure dystonia reveals that there is no known cure and that the treatments available do not provide a clinically proven method for effective recovery. Does this mean that no hope exists for those afflicted with FTSED? Should those diagnosed with the disorder find other musical outlets or give up music altogether? The answer to these questions is a resounding “no!” Each individual confronted with FTSED must determine what is best for themselves and evidence exists that effective treatment is possible. Additionally, with a reconsideration of the pedagogy and philosophy of brass playing and musicianship, there is hope that FTSED can be prevented and perhaps eventually eradicated. What then can be done about FTSED?

Several studies report that embouchure dystonia becomes an intellectual, emotional, and physical problem. Treatment and recovery should therefore take an holistic approach integrating therapy aimed at these areas with a pedagogically-based re-

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202 Notably those by Altenmüller, Byl, Candia, Chamagne, Jabusch, and Tubiana.
training of the embouchure. A four-step program developed by Raoul Tubiana to treat hand dystonia in musicians consisting of body-image restructuring, selective muscle differentiation and relaxation training, individual muscle re-training, and technical re-training at the instrument has proven quite effective. This program, which took about a year to complete for each participant, boasted at least partial improvement in all but 57 of 438 patients. Ninety-five of those patients returned to concert performance. The development and subsequent clinical testing of a similar multi-faceted holistic approach to embouchure dystonia is long overdue.

Perhaps the first aspect of FTSED that should be addressed during treatment is the intellectual. One of the most difficult things about embouchure dystonia is the feeling that there is no logical explanation for what is going on, particularly before or soon after a diagnosis of focal dystonia. This inexplicability often leads those afflicted into a state of confusion, drawing conclusions that are as irrational as the symptoms they exhibit. The simple act of naming the problem and knowing that it is an actual neurological disorder can be very therapeutic. Musicians should be informed about basic neurology, dystonia in general, FTSED, possible causes of their disorder, the difference between physiology and pedagogy, possible treatments, and current philosophies and trends in research. Becoming educated about dystonia provides a rational grounding for what can seem a very irrational experience.


Given the devastating nature of dystonia, the lack of a known cure, and its propensity for ending careers in brass musicians, there can be no surprise that emotional problems and depression often compound the problem and hinder recovery. Lack of information regarding embouchure dystonia, the absence of visible physical problems and pain, and the refusal by many to recognize FTSED as a legitimate problem can add to the emotional instability of a patient. Colleagues, friends, and family are often unaware of FTSED and may be unable or unwilling to comprehend what the patient is experiencing. While some may be dismissive, others may simply not know what can be done to help. It is imperative that patients with FTSED seek immediate professional help to deal with their anxieties, depression and emotional distress. Without treatment, the accompanying depression and psychological problems can be as devastating as FTSED itself and in extreme cases much more so. Furthermore, a healthy emotional state appears to provide the best environment for recovery from FTSED. Effective management of emotional problems can enhance the recovery effort by renewing the patient with a positive self-image and the ability to persevere.\textsuperscript{205}

FTSED can also have an effect on other body movements and physical activity in general.\textsuperscript{206} It is important to address general body use and physical well-being in order to further create an environment that is conducive to healing. Body relaxation and proper air flow are essential first ingredients in the embouchure-rebuilding process and as such


\textsuperscript{206} Wilson, “Current Controversies on the Origin, Diagnosis, and Management of Focal Dystonia,” 317.
need to be properly addressed. To this end there are two methods that can be studied and practiced which can offer great benefits: the Alexander technique and the Feldenkrais method. Both of these techniques aim to heal the body through proper awareness and movement. The Alexander technique is a practical method for improving ease of movement, balance, support, flexibility and coordination. Its aim is to heighten kinesthetic sensitivity and improve specific actions or use of a particular body part through improving use of the whole body. The Feldenkrais method has perhaps more lofty goals: mental, physical, and overall human improvement. Through focused awareness, Feldenkrais addresses self-image, learning, and movement through a practical and philosophical approach. Patients seeking to recover from embouchure dystonia are encouraged to study either one of these or some other form of movement-based body therapy.

The final step in embouchure dystonia recovery is a systematic re-training of the embouchure. As in all parts of FTSED treatment, re-training should be guided by a competent professional. In embouchure re-training a competent professional would best be described as a brass player/teacher who has a knowledge and understanding of FTSED and the misconceptions in pedagogy that contribute to the disorder. This facilitator needs the ability and insight to personalize a step-by-step process based on focused awareness

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207 Jan Kagarice, “A Pedagogical Approach to the Issue of Focal Task Specific Dystonia of the Embouchure.”


of sound and an inhibition of sensory input. Because individuals with FTSED manifest the disorder in different ways, each needs a personalized therapy. No one-size-fits-all method of re-training exists. However, all re-training should share certain fundamental characteristics.

Re-training should be systematic, progressive and begin with relaxation and a sense of ease of movement. The systematic nature of re-training suggests the employment of a simple format. For example, re-training sessions might start with body relaxation, followed by airflow studies, then proceed to specific personalized exercises and end with playing tunes by ear. A progressive approach means that each step in the process is built on the success of the previous steps. The concept of progression is crucial: each new step must build on the previous without becoming too far removed from the previous success. The recovery process needs to be challenging, but a sense of accomplishment and capability is imperative. A push for fast results can potentially sabotage recovery. The focus on building by small successes, however, allows for the return to steps that are successful after an apparent setback. Imagery, stretching, breathing techniques, guided visualizations and meditation can all be helpful in facilitating relaxation and ease of movement.

The critical components to re-training are a focus on auralization and an inhibition of sensory feedback. Giving the brain conscious direction—in other words, an internal sound to match—is the surest way to success. The most effective way to develop the inner ear is through playing simple tunes from memory in various keys. This actively engages the mind in a creative process as opposed to a reactive one. If one can hear it,
one can play it. Given a task to accomplish, the tune one “hears” to play, the body can re-learn to play well through trial and error. The other aspect of this focus is the inhibition of sensory input. Sensory input is likely part of the cause of FTSED and appropriate reactions to sensations while playing must be re-learned. Fortunately, to inhibit focus on this area one simply turns one’s focus to another: auralization. In all exercises utilized in embouchure re-training the emphasis on auralization must be the key factor.\textsuperscript{210}

Regrettably, clinical research and development of effective treatments for embouchure dystonia are virtually non-existent. The four-part approach detailed in the preceding pages—including intellectual education, emotional treatment, body-use therapy, and embouchure re-training—is a possible model for development and study which, given current knowledge and understanding of FTSED, may provide an avenue for success. Although several success stories exist from those undergoing similar treatments, very few of these are documented.\textsuperscript{211} It is an obvious conclusion that further research regarding embouchure dystonia and effective, proven treatment is necessary.

\textsuperscript{210} The discussion of embouchure re-training is derived from the author’s personal experience of a re-training session with Jan Kagarice that took place in July of 2004 at the Chautauqua Institute in Chautauqua, New York. Karagice and Joaquin Fabra (www.embouchuredystonia.com) are considered, within the music world, to be leading experts in embouchure re-training.

\textsuperscript{211} See http://www.davidvining.net/ and http://www.embouchuredystonia.com for accounts of embouchure dystonia recovery.
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Books


Dissertations


Internet Resources


APPENDIX

A CASE STUDY OF FTSED
In June of 2004 Seth Fletcher was diagnosed with focal task-specific embouchure dystonia (FTSED) by Dr. Arthur Fowle at the British Association for Performing Arts Medicine clinic in London. The following account chronicles the initial appearance and progression of symptoms to a clinical state, strategies employed prior to diagnosis, a week-long intensive re-training program following diagnosis, and subsequent methods and routines that eventually resulted in a return to public performance. Additionally, certain pedagogical implications of Fletcher’s experience will be proposed. There are several purposes of this account: to document a case of FTSED for reference, to explore possible pedagogical strategies that may be preventative or therapeutic in nature, and to create awareness of FTSED while encouraging others to share their experiences.

Onset and Progression of Symptoms

The complication of symptoms to the point of significantly impaired public performance and practice was evident in the late autumn of 2003. Fletcher had commenced postgraduate study at the Royal Northern College of Music (RNCM) in Manchester, England that September and had been in preparations for an upcoming audition with the U.S. Marine band in January of 2004. The onset of symptoms had been gradual and had not significantly interfered with performance to that point. By the beginning of December 2003, however, erratic unresponsiveness in the middle register—specifically between B-flat and F in the bass clef staff—occasional lack of clarity with
repeated and/or rapid articulations in the same register, and an uneven tone quality across registers had been observed and commented on by colleagues and teachers. Performance in pitch ranges above and below the noted area were unaffected. Fletcher also observed the development of subtle, uncharacteristic facial movements, and experienced a general sense of discomfort while playing in the affected register.

Although the culmination of symptoms to a disruptive state occurred in November of 2003, in retrospect the initial presentation of symptoms was first observed in late January of that same year. As a member of the Tennessee Tech Tuba Ensemble (TTTE), Fletcher was involved with the group’s concert tour to Carnegie Hall and subsequent recording\(^\text{212}\), serving as principal euphoniumist and featured soloist. Fletcher played the top part for the duration of the program in addition to performing the finale from Vladimir Cosma’s *Euphonium Concerto*. The program was presented in two parts: the first, approximately 1 hour in duration, was comprised of ten “classical” pieces either arranged for or specifically composed for tuba ensemble and the second, approximately 30 to 45 minutes in duration, consisted of several jazz transcriptions. Between January 20 and 28, that program was performed in concert a total of seven times in addition to a two-day recording session on January 26 and 27. Prior to the tour, extensive ensemble rehearsals and sectionals resulted in several days of playing in excess of eight hours per day from January 7 to 19.

The initial presentation of symptoms was first observed during the TTTE’s recording session on January 27 and consisted of the same symptoms described

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previously—erratic unresponsiveness and occasional lack of clarity with repeated and/or rapid articulations in the middle register—albeit to a far lesser degree than their subsequent development. Instances of these symptoms did not interfere with the recording process, were not noticed by anyone but Fletcher, and were attributed to fatigue. During the last concert of the tour on the following day, the symptoms presented with more frequency, but similar performance anomalies were demonstrated by the vast majority of the ensemble and were again attributed to fatigue resulting from the intensive schedule of the tour. After a period of rest lasting a few days, Fletcher resumed a normal playing schedule through the Spring of 2003 with no further occurrence of symptoms.

The intervening months between May of 2003 and Fletcher’s arrival at the RNCM in early September saw no significant recurrence of symptoms. Performance and practice time during these months did decline, however, and two sudden increases in performance at the end of July for the Avalon Brass Band Camp and at the end of August due to preparations for the fall term did result in brief reappearances of symptoms. In each case, the unresponsiveness and lack of clarity were mild and presented concurrent with previously experienced limited endurance and range issues typically following a period of decreased practice and performance. Normal playing resumed without issue after a few days of adjustment to the increased playing schedule.

Over the course of the first few weeks of the fall term at the RNCM, playing and practice time increased significantly as Fletcher began performing with the Tintwistle Brass Band, several college ensembles, and the Elision Euphonium Quartet. The exact point of the onset of symptoms cannot be determined, but the development was gradual
from no earlier than late October, progressing until noticed by others around the
beginning of December. Disruption of normal performance had degraded to the point
that Fletcher was uncertain of the ability to attend the aforementioned Marine Band
audition. An increase in practice over the winter break period with special attention to
long tones, lips slurs, scales, and articulations yielded marked reduction of symptoms and
the audition was attended. Normal playing resumed with the beginning of the term in
January 2004 and a gradual return of symptoms occurred over the course of the next two
months. Fletcher was able to perform as soloist with the Tintwistle Brass Band during
this time period, with approximately four performances of F. Bryce’s Rondoletto.
Playing complications, however, resulted later in discontinuation of solo performances.

By the end of March 2004, symptoms had progressed significantly, surpassing
previous levels of playing disruption. Fletcher participated in two performances as
principal euphoniumist in both the RNCM Wind Orchestra (for the 2004 British
Association of Symphonic Bands and Wind Ensembles conference) and the RNCM Brass
Band, and experienced inexplicable difficulty with two solo passages in particular. The
first involved a solo entrance on a middle F (in the bass clef staff), while the second
entailed rapid articulations across the middle and upper registers. Although both
performances were acceptable, these concerts marked the beginning of a rapid
deterioration of playing ability. Previously described symptoms intensified and spread to
encompass a larger range of notes (from G at the bottom of the bass clef staff to C above
the staff), and a new symptom developed: the uncontrollable closing or clamping of the
lips immediately prior to tone production in the original range affected (B-flat and F in
the bass clef staff). Symptoms progressed to a degree that made normal practice of
fundamentals—long tones, scales, arpeggios, lip slurs, etc.—virtually impossible. The
focus of private study with Fletcher’s teacher, Steven Mead, naturally turned to an array
of strategies intended to relieve symptoms. After several weeks of experimentation
affording no improvement, it was suggested that a prolonged period of rest may be
helpful and Fletcher spent the last week of April and first week of May 2004 completely
away from the instrument. The return to practice in May afforded no relief from
symptoms and normal execution of fundamentals remained unsuccessful.

Continual decline in performance and the rapidly approaching date for degree
recitals in June led to the suggestion to seek help from additional sources. Fletcher
consulted renowned tubists Roger Bobo (then visiting professor at the RNCM) and James
Gourlay (then head of Winds and Percussion at the RNCM) on several occasions during
March and April of 2004. The term “focal dystonia” was first heard by Fletcher during a
session with Bobo, who explained what he knew about the disorder, suggested that it may
be useful to seek medical attention, and provided a few practical exercises focused on
breathing and tone production. Gourlay found the presented symptoms quite
inexplicable, took several photographs of the involuntary facial movements demonstrated
when playing, and suggested a few simple buzzing and tone production exercises. After
a few meetings, Gourlay proposed that Tom Clough, an accomplished former trombonist
and certified Alexander technique practitioner who had personally experienced similar
embouchure difficulties early in his career, might be of assistance.
Clough provided an overview of Alexander technique philosophy—with which Fletcher was previously familiar—and specific exercises and thought processes that allowed for an increased sense of ease, affording longer practice sessions and an improved attitude. Clough was also instrumental in helping Fletcher schedule an appointment with the British Association for Performing Arts Medicine to explore a possible diagnosis of focal dystonia, as first suggested by Bobo. The most notable benefits of practices implemented after consultations with Bobo, Gourlay, and Clough were the apparent isolation of symptoms to a range of approximately an octave (B-flat to B-flat within the bass clef staff) and the improvement of playing above and below that range.

While the development of symptoms appeared to reach a plateau by the end of May, the level of playing disruption made performance awkward at best and impossible at worst. In the most-affected octave, attempts at starting notes (either tongue-articulated or with air alone) resulted in either no discernible tone or an explosion of noise. In every case, extensive involuntary facial movements were present. Since the best efforts of Fletcher and his teachers had provided no significant improvement, it seemed imperative to seek a medical opinion. With the assistance of Clough, he scheduled an appointment at the British Association for Performing Arts Medicine (BAPAM) clinic in London. BAPAM is a charitable organization promoting health in artists of all disciplines by offering free clinics, referrals, health awareness training, and other services in addition to funding research. Fletcher’s appointment was with Dr. Arthur Fowle, a general physician
serving on BAPAM’s Medical Committee and attending to patients regularly at their clinic in London.

The examination was comprised of two parts: a general evaluation and a performance evaluation. Fletcher’s blood pressure, temperature and weight were measured and he was then asked to describe symptoms and their development. This description was followed by a series of reflex and movement tests apparently designed to gauge flexibility and strength. Leg, arm, hand, and facial movements were examined. Fowle then observed the manifestation of symptoms during performance. Fletcher played long tones, scales, and lip slurs in all ranges as well as a few solo excerpts. Fowle concluded that a diagnosis of focal dystonia best explained the symptoms presented. According to Fowle, the next step was to consult a neurologist to determine what, if any, treatment options were available. He advised continuing the study of Alexander technique in the interim and suggested a change in mouthpiece and/or embouchure. BAPAM provided Fletcher with a referral to a neurologist accustomed to evaluating musicians with focal dystonia, Dr. Karin Rosenkranz. Unfortunately, due to Rosenkranz’s research activities, the next available appointment was not until the following September, at which point Fletcher was to have returned to the U.S. Economic constraints made returning to England unfeasible.

Fletcher was diagnosed on June 9, 2004, only a few weeks before scheduled degree recital examinations. Given the state of playing dysfunction it was decided to defer the degree recital to a later date, despite the poor prognosis for patients diagnosed with focal dystonia (the recital examination was subsequently completed in the fall of
Upon learning of the diagnosis, Clough referred Fletcher to Dennis Wick, who offered much-needed encouragement and recommended study with Jan Kagarice, a pedagogue with reported success in helping players with embouchure dysfunction. Fletcher contacted Kagarice and scheduled a session for the last week in July. Fletcher spent the remainder of the summer term at the RNCM working with Clough and continuing with the practice routines previously implemented.

**Pre-diagnosis Strategies**

Subsequent to the significant disruption of performance, and prior to Fletcher’s diagnosis of FTSED, several practices were employed in an attempt to alleviate symptoms. Although precise records detailing specifics of these practices were not kept, their documentation here may perhaps serve as aid in the design of experimental research which may yield insight into their effectiveness, or lack thereof. The strategies utilized may be divided into the following categories: breathing exercises, facial muscle exercises, mouthpiece buzzing, and tone production exercises. Additionally, the application of Alexander technique will be discussed.

Breathing exercises had been a staple of Fletcher’s practice regimen prior to the onset of symptoms. A change was initiated in specific exercises and the focus of these exercises, however, in attempts to alleviate symptoms. Notably, attention was directed toward the physical characteristics of the breathing mechanism as opposed to the motion, shape and direction of the airflow itself. Fletcher was directed to engage abdominal muscles with increased effort in both inhalation and exhalation as a matter of conscious attention. Previous breathing practice consisted of exercises typical of those found in *The
Breathing Gym\textsuperscript{213} and while practice of these was encouraged to be continued, four additional exercises were implemented.

The first new exercise consisted of slow breathing through the instrument. A metronome was set at 60 b.p.m. and the lips placed in the mouthpiece with an aperture too large to produce sound. Air was inhaled evenly over twenty seconds and then exhaled evenly over twenty seconds; the entire process was repeated three times. The next exercise involved the use of the Ultrabreathe\textsuperscript{214} device, which is designed to strengthen breathing musculature through increased resistance training. The device features adjustable resistance of both inhalation and exhalation and was utilized in various combinations (in for 2 counts, out for 2 counts, etc.) with increased resistance over time. The third additional breathing exercise was termed “pitched air” and entailed blowing air in pre-determined pitch patterns utilizing several syllables (‘aw,’ ‘oh,’ ‘ah,’ ‘oo,’ and ‘ee’). Pitch patterns included scales, arpeggios, and short excerpts from solo and etude material. The final exercise, “mouthpiece whistling,” was a variation on the “pitched air” concept.\textsuperscript{215} The mouthpiece was placed on the lips with an aperture slightly smaller than the rim and air is blown to achieve a whistle that was controllable using the syllables described above. Long tones, scales, and arpeggios were the main focus of this exercise.

\textsuperscript{213} Sam Pilafian and Pat Sheridan, The Breathing Gym (Focus On Excellence, Inc., 2002).

\textsuperscript{214} See http://www.ultrabreathe.com.

\textsuperscript{215} A complete description of mouthpiece whistling can be found in Ken Amis’ The Brass Player’s Cookbook: Creative Recipes for a Successful Performance (Meredith Music, 2006).
The next types of exercise employed were facial muscle stretches and isometric facial contractions intended to counteract the involuntary spasms occurring during tone initiation. It was theorized that perhaps these spasms were the result of muscle weakness and that strengthening the facial muscles would resolve their occurrence. These exercises were performed every other day in conjunction with breathing practice to allow adequate time for muscle recovery. First, facial muscles would be tensed inward, toward the nose, for a period of five seconds and then completely relaxed (repeated three times). Next the face would be stretched outward, similar to yawning, and then allowed to relax (also repeated three times). Two types of isometric exercises would then be practiced. The first consisted of forming an embouchure around the shank of a mouthpiece, then tightening the face muscles around the aperture. This was done both with and without synchronized blowing and repeated approximately ten times. The size of the mouthpiece would be changed for each day’s session. The second type of isometric exercise consisted of holding an object (a straw, pencil, or stylus) between the lips so that it stayed parallel to the ground for a specified amount of time (between 5 and 30 seconds). A rest period of no shorter than one hour would follow sessions of facial muscle training.

Mouthpiece buzzing was also practiced in the effort to combat symptoms. Prior to initiation of these exercises in 2004, buzzing was never an integral part of Fletcher’s practice routine, although it was utilized on occasion. The series of exercises employed began with air-articulations of long tones, glissandi of 5th and octaves, scales, and arpeggios. A similar series of exercises was then performed with regular tongue articulations. Finally, exercises were performed alternating immediately between air
articulation and tongue articulation. The size of the mouthpiece used was varied between trumpet, baritone horn, small euphonium (Wick 4AL), large euphonium (Wick SM3), and tuba. Typically two different mouthpieces were used per session. Buzzing practice was performed mostly in the range of B-flat in the staff to F above the staff (bass clef), although some practice was extended beyond those ranges.

As symptoms progressed further, the bulk of practice time was spent on tone production exercises. By June 2004, very little else was possible and even starting and sustaining long tones was difficult, as rarely could they be initiated without explosive facial spasms. Tone production practice would typically begin with air-articulated long tones either above or below the prominently affected area and then ascending or descending, respectively, through the problem areas. Metronome use was occasional, as its use seemed more of a hindrance as symptoms progressed. At one point it was suggested that significant time, at least one hour, be spent daily in air-articulating one tone in the difficult range, moving to a different note the next day. This practice was implemented for approximately two weeks in May, but abandoned when the notes practiced in this manner became even more troublesome. After air-articulated long tones, the same process would be repeated with tongue articulations. Next, simple rhythmic patterns (four quarter notes or quarter, two eighths, quarter, etc.) would be played first with air, then tongue articulations. Again, these would begin in a playable register and ascend or descend through the problem areas. Scales and arpeggios would be practiced next, very slowly, both slurred and articulated. Finally, a series of pedal tones would be played to end each session.
The last of the strategies employed to treat symptoms prior to diagnosis were private lessons in Alexander technique (AT). Fletcher studied with Clough, whose credentials included not only AT certification and positions as trombonist with the BBC, Royal Philharmonic and London Symphony Orchras, but also personal experience with severe embouchure dysfunction. Clough’s unique perspective and constant optimism were invaluable. The Alexander technique is basically “a simple and practical method for improving ease and freedom of movement, balance, support, flexibility, and coordination.”

Fletcher’s study of AT consisted of re-learning the function of what is called “Primary Control” and the inhibition of reflexes and other movements. A series of mental directions and visualizations were employed prior to playing long tones and other simple exercises. It was thought that if the involuntary facial movements disrupting performance had become an ingrained reflex, that conscious control and inhibition of reflexes could reverse the process. The immediate benefit of AT study was a relaxed approach to practice and a sense of enjoyment in the process of performance rehabilitation, despite the continued severity of symptoms. Over the course of several weeks, it was noticed that while the primary area affected was unimproved, upper-register and lower-register tone production became more reliable and efficient.

It is clear from the practices outlined above that the focus of attention to combat symptoms prior to diagnosis was directed on the physical processes involved with brass performance. With the exception of AT study, all practices concentrated on “what

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happens” when playing as opposed to “how to play.” As the development of symptoms continued, it was apparent that these strategies were ineffective at best and possibly exacerbated the problem. Upon being diagnosed with FTSED, Fletcher concluded that new strategies were necessary if recovery was ever to be achieved. Initial research showed that embouchure re-training was the only treatment shown to have any positive effect on FTSED.

Re-training

From July 19 to 23, 2004, Fletcher studied with Jan Kagarice at the Chautauqua Institution in New York. The purpose of this study was to re-train the embouchure in the hope of reducing or eliminating symptoms, which in effect became a complete re-learning and re-conceptualization of brass instrument performance. The typical day of re-training involved two to three sessions from two to three hours in length, with an average of six hours of work daily. The primary concepts of the re-training will be discussed along with specific exercises and thought processes utilized, including: body relaxation, air flow, embouchure concept, and playing “by ear.” Although this intensive period of re-training did not result in a spontaneous remission of symptoms, it did provide some immediate benefits, specifically in improved tone quality, enhanced ease of production in less-affected ranges, reduction in severity of involuntary facial spasms, and increased endurance. Fletcher credits this re-training with providing the foundation for the eventual return to public performance.

The primary concept of Kagarice’s philosophy was quite simple: playing a note on a brass instrument is achieved by blowing air past lips that are touching and internally
singing the intended pitch. FTSED was suggested to be a breakdown of muscle function
due to disagreement between kinesthetic sensory and auditory sensory information, a
notion supported by medical research, as detailed in the present study. The hypothesis
was proposed that if attentive mental focus was directed toward specific healthy actions
of brass playing—i.e. singing the intended pitch “in your head,” before blowing—then
proper motor function could be re-learned with repetition. Another important concept
was that intellectual understanding is insufficient and that proper function must be
experienced to be repeated and learned (Hebbian learning). To that end, the re-training
process was slow and deliberate, with each session beginning with the same focus,
progressing through previous steps covered and ending with a new step.

The re-training process began with a discussion of the onset and progression of
symptoms, strategies employed for managing symptoms, and Fletcher’s attitudes and
mental states during this time period. Prior to the scheduled sessions in New York,
Kagarice had requested a detailed written history of Fletcher’s playing experiences. This
written history and initial conversations revealed that he demonstrated several
stereotypical characteristics of players with FTSED that had been observed in most other
consulting players. Specifically, these included being a “natural,” self-taught player in
the formative years, being a self-described perfectionist and “workaholic,” experiencing a
significant change resulting in a negative effect on playing—in Fletcher’s case periods of
intense playing in excess of 8 hours per day—increasing practice to combat playing
difficulty by focusing on the problem, and increasing feelings of self-doubt as symptoms
progressed. In addition to providing Kagarice with a sense of Fletcher’s personality and
mindset, these discussions, in retrospect, served to help re-contextualize the FTSED from an “end of the world” type of problem to the opportunity to surpass previous levels of performance with new concepts of brass performance, pedagogy and music in general.

The foundation for the re-training process was cultivating body relaxation. Each session began with this topic and it was repeated if and when a sense of relaxation was lost at any time during practice. The general process, consisting of a series of mental suggestions, was as follows. First, eyes were closed and attention directed to awareness of the present environment—ambient sounds, smells, temperature, spatial sense, etc. Next, attention was directed to follow the natural flow of air during breathing. Then, large body areas were directed to relax, beginning with the feet and continuing upward to the head. Finally, eyes were opened and simple movements performed—steps in a specific direction, trunk twists, toe touches, etc.—while allowing the body to be as relaxed as possible. Only when a sufficient state of relaxation was achieved would the next step, air flow, be implemented.

The development of a consistent air flow centered around the concept of “blowing” as opposed to “breathing,” a subtle, yet important distinction. Instead of focusing on the physiology of respiration (“fill up from the bottom up,” “lift here,” “squeeze there,” etc.), or breath control (“in 4 counts, out 4 counts;” resistance training, etc.), attention was focused on the action required for brass tone production, blowing air past the lips. To this end, two types of exercises were utilized. The first consisted of rapid, repeated exhalations and inhalations gradually slowing and lengthening, often accompanied by corresponding hand motions. This “air turn-around” exercise reinforced
the sensation of air moving past the lips and mirrored the change of air direction necessary in brass instrument tone production. The second type of exercises were those employing visualizations, again directed at proper exhalation. These simply consisted of blowing while imagining various images such as: a target posted a specified distance away, a barrel tumbling over Niagara falls, a giant wave rushing forward at 100 m.p.h., a feather flying in the wind, and many others. These exercises always followed body relaxation focus and were also utilized intermittently throughout each re-training session.

The next topic addressed was the concept of embouchure, about which three main points were made. First, embouchure is the function of lips interrupting the air flow. Second, an embouchure is formed only when the air flow blows the lips into position (i.e., embouchure does not exist without air flow). And finally, the ear, or inner hearing, controls pitch, not physical manipulations (i.e. tightening corners, dropping jaw, etc.). These statements were demonstrated by a most interesting display of free-buzzing (without the mouthpiece) in which Kagarice sang a pitch, then proceeded to buzz the pitch with various contorted “embouchures,” concluding by producing the note with an inhalation. This intellectual discussion of embouchure was followed by practical application, first with a free buzz, then mouthpiece buzzing, then playing on the instrument.

The primary exercise of embouchure formation consisted of three steps: 1. hearing a pitch internally; 2. blowing to establish a stable air stream; and 3. allowing the lips to close until the imagined pitch was produced. These steps were repeated several times while buzzing the lips alone, then while buzzing the mouthpiece, and finally while
playing on the instrument. Rest time was taken between repetitions, particularly of the free- and mouthpiece buzzing, and airflow exercises were frequently revisited. Long tones were the first exercises used in playing the instrument, starting both above and below the staff and progressing through the middle register. First, one tone was played, then two (Do, Re), then three (Do, Re, Mi), etc. As these exercises became more fluid over the course of the week, new steps were added such as descending slurred scales, ascending slurred scales, lip slurs, and arpeggios. If and when symptoms prohibited the continuation of an exercise, the initial steps of hearing, blowing, and closing of the lips were rehearsed, usually resulting in sufficient decrease of facial spasms.

The final focus in the re-training process was playing “by ear.” This process was a continuation of the previous tone production practices extended to all aspects of music performance—articulations, dynamics, tone color, phrasing, etc. As in all other re-training steps, this was a sequential process, practiced by starting with the most basic level and progressing in small increments. Simple melodies, mostly stepwise in construction, were performed from memory in various keys with directed attention focused on internal hearing of the melody. Examples of melodies used include “Mary Had a Little Lamb,” “Row, Row, Row Your Boat,” “Twinkle, Twinkle, Little Star,” “Three Blind Mice,” “Ode to Joy,” “America,” and “My Country ’Tis of Thee,” among many others. The first step was to choose a melody and key in which it was to be performed. The next step was to sing the melody out loud and then internally. Then, the melody would be performed slurred, without any articulations. This process would be repeated in two or three additional keys. Articulations would be added next. The melody
would be sung again, this time with clearer and precise articulations as appropriate, then immediately performed in the same manner. It would subsequently be transposed to two or three different keys, not necessarily the same ones used previously. This progression would continue, gradually adding dynamics, phrasing, tone color and other expressive features.

As described above, the re-training process that took place from July 19 to 23, 2004 was a sequential and progressive progress based upon the concepts of internal hearing and air flow. Each session consisted of body relaxation, air flow, embouchure concept/tone production, and playing by ear. This series of exercises was performed incrementally, always revisiting and advancing upon previous successes. The end results of this re-training period were improved tone quality, enhanced ease of production in less-affected ranges, reduction in severity of involuntary facial spasms, increased incidence of successful tone production in the middle register, and increased endurance. Most importantly, Fletcher gained the belief that recovery was, in fact, possible.

Return to Performance

In August of 2004, Fletcher began doctoral studies at the University of North Carolina Greensboro (UNCG) under the guidance of Dr. Dennis AsKew. Although symptoms were still present at this point and playing impaired to a clinical state, the recent re-training session and subsequent progress, combined with knowledge of AsKew’s pedagogical philosophies, suggested the feasibility of this course of action. It was reasoned that recovery from FTSED would possibly be aided by continuing with future plans as if the disorder had not manifested in the first place. Although Fletcher
does not claim to have completely recovered from FTSED, a return to public solo performance was, in fact, achieved.

In September of 2005, Fletcher completed the degree recital requirements for the M.M. degree at the RNCM with a video and audio taped performance at UNCG. In October of the same year, a work from that recital was again performed in addition to a duet with a colleague on a studio recital. These performances marked the first solo appearances in over 18 months. Subsequently, recital requirements for the Doctor of Musical Arts degree were completed with performances on April 24 and October 28, 2007 and February 7, 2008.\textsuperscript{218} Fletcher attributes the successful return to public solo performance to the continuation of exercises learned in re-training with Kagarice and several practices implemented by AsKew, including study of materials in the \textit{Concepts of Euphonium Technique}\textsuperscript{219} etude book, an extensive period of time devoted to playing trombone only, the use of “wind patterns” (see p. 168) in solo and etude practice, and not least an attitude of patient persistence.

\textit{Concepts of Euphonium Technique} outlines a method for the development of instrumental technique based upon the principles of muscle memory and delineation. The book specifically outlines a pattern of variables—articulation, speed, dynamics, and range—that are applied to each individual etude, resulting in several repetitions of all possible combinations of variables for each. This systematic and sequential approach, when combined with focused inner hearing, served as a natural extension of tone

\textsuperscript{218} Programs and sound clips from these performances may be found online at www.euphoniumunlimited.com.

production and playing-by-ear exercises continued from embouchure re-training. The possible impact of muscle memory from healthy repetitions cannot be overlooked. If FTSED is in fact a result of learned sensorimotor malfunction, can proper sensorimotor function be re-learned through directed mental focus on inner hearing and the cultivation of muscle memory through systematic, delineated practice? This method was employed in the practice of etudes, solos, ensemble music, and melodic playing by ear. Over the course of time, this practice appeared to have a profound positive effect in the reduction of symptoms.

Perhaps the most dramatic tactic employed after re-training was the abandonment of the euphonium in favor of trombone for several weeks in the spring of 2005. While there were no major setbacks in the fall of 2004, progress had seemed to plateau. AsKew suggested that playing trombone only, for a time, might provide another avenue for directed mental focus, diverting attention away from the symptoms of FTSED. This seemed quite reasonable, especially since Fletcher had previously been an able trombonist, having performed as a soloist in recital and as a member of several concert and jazz bands, but had not played the instrument regularly since 2002. To further explore this idea of unfamiliarity, new study materials—including scale studies with varied articulation, legato etudes, and Jack Gale’s *12 Jazz Duets*—were used in conjunction with the basic practices continued from re-training. This temporary change of instrument proved to be quite helpful. A reduction in the affected range by a fourth was observed (to between B-flat and F in the bass clef staff) as well as improved clarity

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of articulation and ease of facility in performing lip slurs. These benefits continued when
euphonium playing resumed. While the exact mechanism for these improvements was
not known, it was surmised that the act of playing trombone, specifically the distraction
of executing fluid slide technique, increased Fletcher’s ability to focus directed mental
effort toward playing by ear and airflow. Perhaps over time this allowed for muscle
memory re-learning of healthy patterns, resulting in a reduction of symptoms.

The final therapeutic practice initiated in Fletcher’s study with AsKew was the
use of “wind patterns” in solo and etude study. A wind pattern consists of simply
blowing the rhythmic pattern of the music being practiced, incorporating articulations,
dynamics, phrasing, and fingerings. This method may be utilized in a progressive
manner, first focusing on rhythm alone, then adding the inner hearing of pitch, then
specific articulation, dynamics, and so on. Wind patterns may be employed with or
without the instrument, allowing for increased practice time with less fatigue and the
conditioning of an airflow reflex. This method was utilized extensively in recital
preparation, both in the learning of repertoire and as an additional means of maintenance.

Finally, the role of positive attitude and patience displayed by AsKew deserves
mention as it certainly directly affected Fletcher’s return to performance. As documented
in several studies, musicians with FTSED typically experience emotional distress, often
leading to depression. Fletcher’s case was no different, although clinical depression was
not diagnosed. In maintaining a relaxed, open-minded, and positive demeanor, AsKew
allowed the focus to be on the recovery process and progress made and not the disorder
itself or any setbacks incurred. Indeed, this environment was conducive to the re-training
and recovery process and contributed significantly to Fletcher’s return to public solo performance.

**Pedagogical Implications**

Personally experiencing a disorder such as FTSED is nothing less than a life-changing event, resulting in feeling of confusion, frustration, and presenting difficult decisions. As with any difficulty, however, experiencing FTSED provides extensive possibilities for learning, as well as for personal and professional growth. Four years of personal experience with—and research of—FTSED has profoundly impacted Fletcher’s performance and pedagogy, in his own opinion, for the better. He has had the privilege of studying privately with many of the most well-regarded performers and pedagogues in the tuba-euphonium and trombone communities, in addition to attending and performing in countless international, national and regional conferences and master classes, and presenting at regional conferences. Fletcher has observed that the vast majority of pedagogical focus rests on describing the symptoms of what happens when a brass instrument is played well and that many of these descriptions are vague or incomplete at best and grossly incorrect at worst.

The practice of utilizing descriptions of the symptoms of good brass playing as a pedagogical method may be helpful to some players, but simple reasoning shows it is more likely to be misleading. For example, it is often said that to play higher pitches, one should make the embouchure’s aperture smaller and blow faster air. But how exactly does one make the embouchure’s aperture smaller? Should one only close the lips themselves or use the cheek muscles or both—by the way, smaller in which direction?
And how is the air blown faster: do you push with the abdominal muscles or raise the
tongue to increase the internal mouth pressure and therefore the airspeed as well? Also,
is one supposed to calculate the exact aperture size and airspeed necessary for each
desired pitch? Another example would be the direction to expand one’s rib cage to allow
for a proper inhalation. Unfortunately, it is very easy to expand one’s rib cage without
achieving an inhalation sufficient for proper brass playing. Brief consideration of two
eamples clearly shows that certain observations of what happens when a brass
instrument is played well are not necessarily the same as directions for how to play a
brass instrument well. More importantly, research identified in this study suggests that
directed attention on sensory feedback may play a role in the development of focal
dystonias. It may be that “symptomology pedagogy” is not only ineffective, but also
harmful.

While it is beyond the scope of the present study to experimentally test and
compare various pedagogical strategies, Fletcher’s personal experience and the literature
reviewed suggest that certain practices may contribute to the onset and development of
FTSED, or exacerbate the problem once present. And although no medical studies have
documented a successful treatment of FTSED, the methods used in Fletcher’s case (as
well as those described by Vining and Fabra) exhibit similarities to methods utilized in
medical research demonstrating success with focal hand dystonia. Pedagogical practices
focused on internal hearing and cultivation of a consistent air flow may provide a
healthier paradigm for brass instrument performance and instruction.
In the same manner that visual focus provides direction for skilled actions such as pitching a baseball and shooting an arrow from a bow, inner hearing guides brass instrument performance—and all musical performance. How likely would it be for one to hit the bull’s-eye of an invisible target? Probably about as likely as it would be for one to hit a note on the trumpet without being able to hear the intended pitch. The importance of the ability to internally hear what one intends to play cannot be overestimated in brass performance. The more details one can imagine in the mind’s ear, the more details can be expressed in performance. Sight-singing practice utilizing solfège along with dictation exercises can be invaluable in cultivating internal auditory perception. Singing as a diagnostic tool in private instruction is also invaluable as there is no other reliable method to determine the internal hearing of a student. In instrumental practice, internal hearing may be developed through playing-by-ear exercises (as described previously) and through audiation prior to and during playing at all times. While the “if you can sing it, you can play it” axiom may be debatable, it is a most certain fact that if one cannot hear it, one will not be able to play it as effectively.

If inner hearing is analogous to a rocket’s directional computer, then airflow is the fuel catalyzing its propulsion to the intended target. Sound cannot be produced on a brass instrument without the action of air moving past the lips. Therefore, the majority of breathing practice should focus on the cultivation of a consistent, relaxed airflow in terms of the action of blowing. The “air turn-around,” wind patterns, and other blowing exercises described above provide sufficient means to this end. With regard to inhalation, Alexander technique study suggests that body mapping—the learning of
correct anatomy and physiology—combined with relaxed, controlled movements provides an optimum paradigm for breathing. Simply put, in the absence of an incorrect perception of respiratory function all that is necessary for sufficient inhalation during brass performance is a relaxed body and the sense of air moving inward past the lips. In effect, the air inhaled already “knows where to go.” Directed mental focus on the action of blowing and the allowing of inhalation to be a natural continuation of that process frees the respiratory system to function subconsciously and effectively.

Two additional concepts integral to Fletcher’s return to public performance are deserving of mention and may be applicable to a healthy brass pedagogy: un-attachment and variation in practice. The various symptoms that present in FTSED may quickly become the focus of an afflicted player’s attention. Indeed, studies suggest that the first impulse of afflicted musicians is to increase practice aimed at the problem areas. The outcome, or the manifestation of symptoms when playing, becomes the focus. Becoming un-attached from this outcome—emotional un-attachment and attention un-attachment—allows for the complete direction of mental focus on inner hearing and the action of airflow. The development of an attitude of indifference toward the actual sound produced during practice and performance, whether successful or otherwise, proved quite useful. Finally, variations in exercises practiced were implemented to counteract the possibility that numerous exact repetitions may contribute to FTSED or aggravate symptoms.

The exact causes of FTSED remain unknown. Research suggests, however, that a pedagogy centered around descriptions of observations as means to good brass
performance play a role in the development and progression of the disorder. Fletcher’s personal experience supports this theory and advocates a pedagogy based upon directed mental focus toward inner hearing and airflow, combined with the cultivation of unattachment and varied daily practice. Preventing or curing FTSED may not be possible, but this account provides an example of an alleviation of symptoms that allowed for a return to public solo performance. The strategies and practices utilized in this endeavor may prove successful for others, whether they are afflicted with FTSED or not.