Focal Dystonia Causes and Treatments: A Guide for Pianists

Juan Nicolás Morales Espitia

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FOCAL DYSTONIA CAUSES AND TREATMENTS: A GUIDE FOR PIANISTS

by

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DEDICATION

This document is dedicated to all the musicians suffering from focal dystonia.
ACKNOWLEDGEMENTS

I greatly appreciate the guidance that my thesis advisor, Dr. Scott Price, provided me for the completion of this document. I am forever indebted to Ms. Edna Golandsky, who patiently showed me a pathway to continue my professional career, and my piano teachers Dr. Inesa Sinkevych and Mr. Phillip Bush, who always believed in me and did not allow me to give up on playing two-hand repertoire. Finally, I am immensely grateful to my wife, Joo Yeon Park, and my family, who unconditionally supported me at all times.
ABSTRACT

Focal dystonia is a movement disorder that precludes the execution of fine motor skills. Although research on focal dystonia has been intensified during the last few decades, particularly because it has affected eminent musicians such as pianists Leon Fleisher and Gary Graffman, violinist Peter Oundjian, and oboist Alex Klein, there are still several answered questions, as well as numerous misconceptions, commonly associated with this disorder. This document comprehensively explores dystonia’s mechanism of action, causes, potential risks, and treatments. The publicly available statements of three prominent concert pianists who successfully recovered from focal dystonia are also included in this document. Additionally, the document comprises the author’s insights after utilizing the Taubman Method to control the symptoms caused by this movement disorder.
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<thead>
<tr>
<th>Abbreviation</th>
<th>Full Form</th>
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<tbody>
<tr>
<td>BG</td>
<td>Basal Ganglia</td>
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<tr>
<td>BoNT</td>
<td>Botulinum Toxin</td>
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<tr>
<td>CNS</td>
<td>Central Nervous System</td>
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<tr>
<td>CTDs</td>
<td>Cumulative Trauma Disorders</td>
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<td>DBS</td>
<td>Deep Brain Stimulation</td>
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<tr>
<td>DMRF</td>
<td>Dystonia Medical Research Foundation</td>
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<tr>
<td>DRD</td>
<td>Dopa-Responsive Dystonia</td>
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<td>DYTn</td>
<td>Dystonia Gene</td>
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<tr>
<td>FHD</td>
<td>Focal Hand Dystonia</td>
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<tr>
<td>fMRI</td>
<td>Functional Magnetic Resonance Imaging</td>
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<td>FTSD</td>
<td>Focal Task-Specific Dystonia</td>
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<tr>
<td>GB</td>
<td>Globus Pallidus</td>
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<tr>
<td>MSM</td>
<td>Manhattan School of Music</td>
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<tr>
<td>OOS</td>
<td>Occupational Overuse Syndrome</td>
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<tr>
<td>R&amp;R</td>
<td>Rehabilitation and Retraining</td>
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<tr>
<td>RSI</td>
<td>Repetitive Strain (stress) Injuries</td>
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<tr>
<td>rTMS</td>
<td>Transcranial Magnetic Stimulation</td>
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<tr>
<td>SMRs</td>
<td>Skeletal Muscle Relaxants</td>
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<tr>
<td>tDCS</td>
<td>Cathodal Direct Current Stimulation</td>
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CHAPTER 1

INTRODUCTION

Nothing I was doing seemed to change anything in my hand. It wasn’t fair. It wasn’t fair. I wondered if there was any point to living, if I wasn’t able to play. I gave serious thought to ending my life. That, at least, would be an action that would bring a solution, and an end to the suffering.¹

These are some of the memories of the celebrated American pianist Leon Fleisher from 1965 when he was forced to cancel his concerts and tours with the New York Philharmonic and the Cleveland Orchestra, as well as all of his scheduled recitals during that season. Fleisher reported experiencing the symptoms of a neurological disorder known as Focal Task-Specific Dystonia (FTSD) in the fourth and fifth fingers of his right hand. His case is, unfortunately, only one of several involving world-class pianists who developed this condition.

The list of leading concert pianists that have reported suffering from this disorder includes American pianist Gary Graffman, French pianists Michel Beroff and Jean-Efflam Bavouzet, Dutch pianist Rian de Waal,² Chinese pianist Hung-Kuan Chen,³ New

Zealander pianist Michael Houstoun, and American pianist James Litzelman, among other distinguished performers. In addition, Frank R. Wilson, based on the information from Glenn Gould’s personal diary, concluded that Gould’s symptoms matched those of FTSD during his two major crises in 1959 and 1977. Gould’s description is quite detailed:

During the 2nd TV taping (first week of June) lack of coordination was immediately apparent – Opening theme of Casella was unbalanced and notes appeared to stick and scale-like passages were uneven and uncontrolled. At this period problem appeared primarily in dynamically restrained passages. During the next two weeks problems increased. It was no longer possible to play even Bach Chorales securely – parts were unbalanced, progression from note to note insecure.

FTSD symptoms have been seen even in some historical figures. Eckart Altenmüller’s research suggests that Robert Schumann also developed FTSD in the third finger of his right hand, causing the end of his career as a virtuoso pianist. Paradoxically, the percentage of pianists presenting the symptoms of FTSD has been shown to be significant in more recent studies, including both piano students and professional pianists. Sakai’s (2002) research on 200 injured pianists concludes that eighteen of them showed the symptoms of FTSD, 9% of the cases.

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6 Tubiana and Amadio, Medical Problems of the Instrumentalist Musician, 380.
7 Tubiana and Amadio, 313.
FTSD symptoms have been discussed by neurologists since the nineteenth century. One of the earliest references to the symptoms that characterize this disorder was provided by the French neurologist Guillaume-Benjamin-Amand Duchenne in 1861:

I define functional spasm as an affection characterized by pathological continuous contractions, either painful or painless, which manifest as clonic contractions, tremors or sometimes paralysis; this disorder is evident only during certain voluntary movements and can appear in any region of the body… this spasm which is provoked by abuse of muscular function and appears only during this function, has been named “Schreiberskrampf” (writer’s cramp) in Germany. This nomenclature which has been used by all authors who have written on this unusual disorder reinforces the fact it is often localized in the muscles of the hand and particularly in the muscles used in writing… But I have also seen it in pianists, florists, tailors, boot makers, fencing masters etc. It may also occur in many other regions of the body apart from the hand. ¹⁰

Although research on FTSD has intensified since the 1970s, particularly with the rise of the field of performing arts medicine,¹¹ the medical community has not yet come to an agreement on the contributing factors or treatments and whether there is a definite cure. In this research, the author will provide an overview of the symptoms commonly linked to this disorder and the treatments that have been used in patients with FTSD, while due to their relatively recent emergence, most of them still remain within experimental stages.

The author was diagnosed with FTSD in 2013 when he noticed the loss of control of his second finger in the right hand. The key symptom was characterized by involuntary movements causing his index finger to curl into the palm while playing the piano. After an extensive search including a diverse variety of medical approaches, the author found the Taubman Method (TM) the most efficient and long-lasting alternative to mitigate the

¹⁰ Tubiana and Amadio, Medical Problems of the Instrumentalist Musician, 329.
effects caused by the disorder, and in the long term, a guideline to ultimately achieve a complete recovery.

**Historical Overview**

The term dystonia was first used by the German neurologist Hermann Oppenheim in 1911. However, before the term was officially coined, his observations on musicians had described some of the symptoms frequently associated with Focal Task-Specific Dystonia (FTSD):

Piano-player’s cramp consists, as a rule, in abnormal muscular contractions, from which a finger or several remain lifted from the keys or are pressed upon them… Violinist’s cramp may affect the bowing hand or the hand pressing upon the strings, occasionally both… An occupational neurosis of the labial muscles was seen by me in a horn-blower. As soon as he placed the mouth piece to his lips a spasm came on in the orbicularis oris, so that the patient was unable to bring out a tune.\(^{12}\)

Through the course of the twentieth century and the beginning of the twenty-first century, several neurologists have contributed to narrow the scope of the definition of dystonia. Nevertheless, it was only in 1975, at the First International Dystonia Symposium, when the clinical characteristics of several forms of focal dystonia were finally measured. A decade later, under the auspices of the Dystonia Medical Research Foundation, an ad-hoc committee was gathered to formulate the first consensus definition of dystonia. This definition outlines dystonia as a syndrome consisting of “sustained muscle contractions, frequently causing twisting and repetitive movements, or abnormal postures.”\(^{13}\)

\(^{12}\) Dressler, Altenmüller, and Krauss, 195.

One of the most current definitions of dystonia was achieved through the collaboration of an international Consensus Committee organized by the National Institutes of Health in 2013. This Committee stated the following definition for dystonia:

A movement disorder characterized by sustained or intermittent muscle contractions, causing abnormal, often repetitive, movements, postures or both. Dystonic movements are typically patterned, twisting and may be tremulous. Dystonia is often initiated or worsened by voluntary action and associated with overflow muscle activation.\(^{14}\)

**Classification**

Dystonia’s classification requires consideration of diverse factors that are solely present in each specific type of dystonia. For instance, dystonias can be classified depending on the age at onset, its body distribution, temporal pattern, nervous system pathology, and whether it is inherited or acquired.\(^{15}\) The type of dystonia affecting pianists is normally addressed as either Focal Hand Dystonia (FHD) or Focal Task-Specific Dystonia (FTSD). The word focal denotes a dystonia that affects only a specific part of the body, and Task-Specific implies that the symptoms appear only through the execution of a specific action. Yet, although focal dystonias may appear when performing a variety of activities such as writing, painting, playing golf or darts, recent studies have shown that musicians trained in classical music are the most likely to develop this condition.\(^{16}\) In fact, a study conducted in 2006 with 144 musicians revealed that the pianists were the instrumentalists with the largest population diagnosed with FTSD.\(^{17}\)

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\(^{14}\) Albanese et al., 6.  
\(^{16}\) Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 3.  
\(^{17}\) Altenmüller and Jabusch, 5.
Phenomenology

The current knowledge of dystonia’s phenomenology is abundant due to numerous reports and studies conducted on musicians diagnosed with this condition. The American neurologist Steven Frucht (2018), based on reported cases of over 1000 patients, all of them musicians, identified some of the common aspects observed in musicians’ dystonia phenomenology. More specifically in pianists, there are several recurring features. The hand most commonly affected is the right hand, due perhaps to the notorious higher technical demands that the piano literature often places on this hand. The dystonic movements affect initially one finger, though there are reported cases where the dystonic movements expand to adjacent fingers over time. These involuntary movements can occur either through uncontrolled flexion or extension of the affected finger, although flexion predominates over extension.

Dystonic movements often have a technical trigger such as ascending scales, octaves, chords, or repeated notes. Still, although the undesired motion is clearly detectable, the patients are unable to prevent it from reoccurring. Despite dystonic movements being the most evident symptom of FTSD, patients have also reported a “lack of control,” “lack of fine touch,” or “having to think about what was previously automatic.” The author correlates this lack of automaticity with a loss of “natural sensations” on the keyboard as if the proper motion of his index finger had been somehow “forgotten.”

19 Dressler, Altenmüller, and Krauss, 198.
20 Dressler, Altenmüller, and Krauss, 198.
A distinguishing factor in musician’s dystonia is the absence of pain, in contrast with most muscle-related affections.\textsuperscript{23} Likewise, the dystonic movement is perceived just within seconds of playing. However, the author has observed that although the involuntary motion is indeed painless, after hours of practicing, the sustained tension generated by the dystonic movement may result in pain or muscular fatigue.

**Coordination of Muscle Activity in FTSD**

Every movement requires a complex and constant interaction between the central nervous system and the muscles directly involved in the movement. Depending on the vector of the movement, some muscles are responsible for actively producing the movement, while some muscles delimitate the range of movement. These groups of muscles are denominated agonist and antagonist muscles, respectively. This phenomenon was discovered and further studied by the English neurophysiologist Charles Sherrington, who identified it as the principle of reciprocal inhibition. He defined it as: “a reflex of ‘simultaneous double sign,’ with excitation of one or more agonist muscle(s) and simultaneous inhibition of the antagonists acting at the same joint.”\textsuperscript{24}

In a pianist’s hand, for example, the muscles in charge of flexion movements are called flexors, while the muscles in control of extension movements are called extensors. A precise control of the activity in both muscle groups is necessary to achieve a smooth and efficient motor pattern. Several studies have shown that the absence of inhibition, preventing the antagonist muscles from blocking the movement produced by the agonist

\textsuperscript{23} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 3.
muscles, is observed in patients with FTSD, resulting in simultaneous contraction of agonist and antagonist muscles, and therefore in the dystonic movement. This anomalous lack of inhibition between the agonist and antagonist muscles is also known as co-contraction. Furthermore, studies have also demonstrated, through the use of wire electromyographic recording, that the hand of a pianist with FTSD shows “abnormally prolonged muscle firing, with co-contraction and overflow of activation of inappropriate muscles.”

Risk Factors

The research of Eckart Altenmüller and Christos I. Ioannou (2015) has categorized the triggering factors of the musician’s dystonia into sensory-motor and psychological. The sensory-motor triggering factors are related directly with the instrumental performance. This category, among others, includes biomechanical obstacles, workload, technique, as well as the instrument itself, ultimately leading the performer into motor fatigue and muscular overuse. This category is highly significant, as pianists often tend to overlook possible flaws in the way they physically approach their instrument. The American pianist Gary Graffman, still active as piano faculty at the Curtis Institute of Music and the teacher of a generation of outstanding pianists including Yuja Wang and Lang Lang, shared his experience with FTSD in 1986:

Until my problem appeared, I’d never given the slightest thought as to how I play the piano. It was like brushing teeth or walking. Or even which fingers I used: if a colleague asked how I fingered a section of a certain piece of music, I’d have to


26 Lin and Hallett, 2.

play to see what my fingers have been doing. So it was only until I was forced to acknowledge that I could no longer traverse the bravura passages of my daily repertoire that I consciously realized I’d been doing something weird— and for a long time, too.28

The second category, the psychological triggers, focuses entirely on the performer. The psychological demands of the classical musician are notorious as they involve accurate execution of extremely complex and fast motions, in addition to the interpretive difficulties inherent to every musical work.29 Factors such as anxiety, perfectionism, stress, and others belong to this category. However, the author agrees with the researchers Altenmüller and Ioannou (2015) when pointing out hereditary susceptibility as an ultimately decisive factor in the musicians that develop FTSD.30 Despite the overwhelming challenges that classical musicians face on a daily basis, continuous practice does not result for every performer in developing this neurological disorder.

**Purpose of the Study**

The purpose of this document is to contribute to the research that has been conducted on FTSD while presenting the author’s experience and insights on both facing and living with the disorder. It is the hope of the author that this document may prove valuable to pianists who have been diagnosed with FTSD and have been struggling to overcome it. As part of this research, observations on piano technique and biomechanics will be provided to support the utilization of the Taubman Method (TM), not only as a

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29 Raab et al., *Performance Psychology*, 103.
30 Raab et al., 110.
recovery method, but as a method to play the piano with more efficient utilization of the body’s resources, resulting in a highly coordinated use of the body as a whole.

**Need for the Study**

The availability of sometimes accurate but often fraudulent medical information on the internet allows musicians who may suspect they are suffering from FTSD to do research and self-diagnosis before scheduling an appointment with a physician. It has been estimated that when using search engines with the keywords Focal Task-Specific Dystonia, the results will show “more than 500 useless references.”

Although much that has already been said about FTSD is common knowledge, there is still a significant amount of misinformation, as well as erroneous theories and assumptions commonly associated with this disorder. Likewise, even with the increasing attention that FTSD has received during recent decades, there are still several questions that remain unanswered.

The relatively small percentage of pianists affected by this condition and, in some cases, their reluctance to make their condition known to the public, has made it difficult to collect enough information to approach more conclusive results. This reaction is understandable since it is often the case that a tacit negative connotation exists in relation to physical injuries. Pianists often associate injuries with a poor technique, or even the lack of the minimum physical conditions to sustain a career as a performer.

Consequently, this document is meant to present a comprehensive guide to FTSD as a very delicate but still treatable condition while demystifying some of the misconceptions commonly linked with this disorder, in addition to introducing a variety of alternatives.

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Limitations of the Study

The present document is limited to an examination of the history, possible causes, and the currently recognized treatments for FTSD. The study includes publicly available cases of three classical pianists who have recovered from FTSD and descriptions of how they accomplished their recovery. Lastly, the author’s experiences with FTSD and the TM are introduced.

Literature Review

There is a significant amount of research conducted on dystonia since it is a syndrome that contains a wide variety of conditions. Despite the fact that dystonia-related disorders were reported by British neurologist William Gowers as early as in 1888, dystonia was still cataloged as a rare and untreatable condition as late as the 1990s. Fortunately, significant advances in the current understanding of dystonia have come as a result of neurophysiologic and brain imaging techniques. Likewise, new therapeutic approaches have demonstrated efficiency at reducing the severity of the symptoms that characterize dystonia.

The diagnosis of dystonia, in contrast, still remains as a symptom-based clinical evaluation. To date, neither brain imaging nor laboratory tests serve as a tool to diagnose this disorder. Consequently, misdiagnosis of dystonia-related symptoms has been a

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33 Mitchell F. Brin, Cynthia Comella, and Joseph Jankovic, *Dystonia: Etiology, Clinical Features, and Treatment* (Lippincott Williams & Wilkins, 2004), 1.
34 Brin, Comella, and Jankovic, 1.
35 Jankovic, *Dystonia*, 1.
typical scenario, even in recent years. Connections between the symptoms of dystonia and stressful work environments have been made in the past by psychoanalysts, concluding that the origin of FTSD has a psychosomatic root.\textsuperscript{36} This assumption classifies dystonic movements as an unconscious psychological response to threatening career-related events.

Notwithstanding, numerous studies, through the use of Functional Magnetic Resonance Imaging (fMRI) on patients with FTSD, have shown that the somatosensory representation of single fingers is compromised. The brain images of these patients demonstrate fusion between the areas responsible for processing individual finger movements. Dissimilarly, the images from the non-affected hand in the opposite hemisphere show a distance of about 2.5 cm between the movement centers that process incoming sensory stimuli from individual fingers.\textsuperscript{37} Still, psychological factors and overuse have been highlighted by neurologists as possible triggering factors for FTSD, although these implications remain subjective since healthy musicians face the same challenges as musicians who have developed FTSD.

While the causes of FTSD are yet to be discovered, a variety of treatments have been implemented on diagnosed patients. These treatments include strengthening of the metacarpophalangeal joints, behavioral modification, rest and relaxation techniques, movement therapies, gymnastics, chiropractic manipulation, ultrasound, acupuncture,

\textsuperscript{37} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 6.
rehabilitative splints, and transcutaneous nerve stimulation, between others. Nevertheless, those procedures have proven to be insignificant or of no benefit to the patients.\textsuperscript{38} 

Intramuscular botulinum toxin injection (BoNT) is one of the treatments that has shown significant efficiency at decreasing the intensity of dystonic movements.\textsuperscript{39} BoNT is injected into the muscles that activate the dystonic movement. While weakening the targeted muscles, partial relief from undesired muscular contractions is experienced by the patients. This treatment has proven to be beneficial, particularly for patients suffering from blepharospasm, torticollis, and spasmodic dysphonia.\textsuperscript{40} However, this may not be the case for patients of FTSD. The author received BoNT injections over the course of a year, with insufficient results. Whereas the BoNT successfully weakened the muscles that triggered the dystonic movement, the same muscles became significantly less responsive, and their speed and power were reduced substantially. The author concluded that the negative side effects of this treatment were greater than its benefits.

The research on dystonia has brought substantial findings that have changed the way both neurologists and patients perceive this syndrome. At the time of the writing of this paper, literature is abundant in describing this disorder in detail, principally how and where the dystonic movements are originated. Nevertheless, it is rather speculative in terms of its causes, as well as in depicting a successful sequence of steps to follow for pianists that have been diagnosed with FTSD. The purpose of this document is to fill that void, present the insights of pianists that have achieved significant recovery, together

\textsuperscript{38} Tubiana and Amadio, \textit{Medical Problems of the Instrumentalist Musician}, 305.
\textsuperscript{39} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 206.
\textsuperscript{40} Tubiana and Amadio, \textit{Medical Problems of the Instrumentalist Musician}, 305.
with the author’s experience at applying the TM as a means to recover from FTSD, and to provide the reader with a comprehensive guide to FTSD.

**Design and Procedures**

This paper comprises four chapters, a bibliography, and appendices. Chapter one consists of the introduction of the study, including the purpose, need, limitations, review of literature, and design and procedures. Chapter two provides a historical overview as well as observations on the risk factors connected with FTSD. Chapter three examines known treatments, in addition to specific approaches utilized by pianists who achieved significant recovery. The final chapter is a summary of the research and its findings.
CHAPTER 2

GENERAL OVERVIEW

Dystonia is a hyperkinetic movement disorder with main symptoms portraying sustained co-contractions between agonist and antagonist muscles. Movement disorders belong to the broader category of neurological syndromes. These syndromes are characterized by either excess or lack of voluntary and automatic movements. In the medical literature, the movement disorders that produce excess of movement are commonly denominated as hyperkinetic, dyskinetic, and as abnormal involuntary movements. The five main categories of hyperkinetic movement disorders are dystonia, chorea, tics, tremor, and myoclonus. Nonetheless, within this classification, dystonia remains as one of the most common movement disorders.

Movements can be categorized as voluntary, automatic, and semi-voluntary. This distinction is relevant due to its intrinsic implications in the case of pianists diagnosed

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43 Rapoport et al., *Handbook of Neurological Therapy*, 320.
with FTSD. Voluntary movements are conscious, as they serve an intentional purpose. Piano playing begins as sequences of voluntary movements, though through repetition the required motion patterns become automatic. This means that the basic movements required to play the instrument turn into learned motor behaviors that do not require a conscious effort. This process is often the result of years of practice and technical training. Consequently, a pianist suffering FTSD loses control over the automatic movement patterns necessary for piano playing. The author recalls the sensation as if the affected finger had “forgotten what to do or how to move.” Understanding how dystonia affects a pianist’s hand, from a neurological perspective, offers valuable clues in terms of what will be necessary to regain control of automatic movements. This approach will be further explained in Chapter 3 of the present document, under the treatments section.

Many movement disorders, dystonia included, are the result of pathologic alterations to the basal ganglia (BG) area of the brain. The BG is the group of gray matter nuclei located between the cerebral hemispheres, the diencephalon, the mesencephalon, and the mesencephalic junction. In the past, the BG’s structure comprised the corpus striatum, claustrum, and amygdaloid complex. However, more current definitions of the BG limit it to the corpus striatum, the subthalamic nucleus, substantia nigra, and pedunculopontine nucleus.44 As the striatum receives information from the axis of the central nervous system, the globus pallidus (GB) and the pars reticularis of the substantia nigra take the information gathered by the striatum and deliver it into the thalamus.45


45 Standring, 366.
Notwithstanding, the complex organization and interactions between the structures forming the BG are not yet fully understood; for instance, the function of one of its structures, the claustrum, is still unknown.\textsuperscript{46}

The function of the basal ganglia can be described as guiding intention into action. It produces and sustains motor patterns, as well as inhibiting undesired movements.\textsuperscript{44} Disorders of the basal ganglia bring either the inability to execute desired movements as in Parkinson’s disease, or the inability to prevent unwanted movements as

\textsuperscript{46} Standring, 364.  
\textsuperscript{47} Standring, fig. 24.2.  
\textsuperscript{48} Standring, 369.
in FTSD. Traditionally, dystonia has been associated with the putamen area of the basal ganglia.\(^{49}\)

![Figure 2.2. Axial (A) and coronal (B) magnetic resonance images of the brain showing the basal ganglia, thalamus and internal capsule\(^{50}\)](image)

\(^{49}\) Fahn, Jankovic, and Hallett, *Principles and Practice of Movement Disorders*, 2.
\(^{50}\) Standring, *Gray’s Anatomy*, fig. 24.1.
The BG carries not only motor functions and motor coordination, but these structures are also actively involved in processes related to cognition, emotion, and motivation. BG’s role is crucial in allowing cognitive controls to take over motor patterns, resulting in the performance of more efficient goal-directed movements. After a movement is targeted, the BG breaks the movement into simpler and more manageable gestures. Subsequently, when sequences of simpler gestures are learned, they start occurring automatically while allowing the subject to focus on environmental or external cues with direct implications in the movement. This process is followed by an assessment of the efficiency of the movement, based on the predicted outcome and its actual result. Small adjustments through repetition of the motor pattern refine it and retune it to obtain the exact sequence of gestures that require the least effort for its performance.

Early neuroimaging studies confirmed that the characteristic muscle contraction and overflow present in dystonia were the consequence of impaired inhibition of the BG. These studies exposed a lack of activity in the internal segment of the globus pallidus (GP), a subcortical structure responsible for inhibiting the thalamus. The results consolidated the hypothesis that inactivity in the GP leads to overactive thalamocortical projections. In accordance with these findings, recent fMRI studies conducted on FTSD

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52 Soghomonian, 117.
53 Soghomonian, 118.
patients also demonstrated a disrupted somatotopic organization of digits in the putamen. Ultimately, these studies have led researchers to formulate a new hypothesis in which dystonia is the product of abnormalities during movement preparation rather than during movement execution.55

HISTORICAL OVERVIEW

In 1833, the Scottish neurologist Charles Bell described in detail the fluency and movement efficiency of the human hand:

The human hand is so beautifully formed, it has so fine a sensibility, that sensibility governs its emotions so correctly, every effort of the will is answered so instantly, as if the hand itself was the seat of that will; ...we use it as we draw our breath, unconsciously.56

Ironically, during the same year of the publication of his celebrated treatise on the human hand, Bell observed in his practice the earliest documented case of a writer’s cramp,57 a condition which would be diagnosed today as FTSD.58

More than a century later, in 1911, the German neurologist Hermann Oppenheim used the expression “dystonia musculorum deformans” to identify a condition detected in four of his patients. Nevertheless, this term has become obsolete. “Musculorum” implies that the disorder is primarily a muscle disorder, while fluctuating muscle tone is not always an essential feature of the disorder. Additionally, “deformans” suggests that all patients develop deformities, and this is also imprecise.59

55 Kurstot and Forsström, 202.
56 Tubiana and Amadio, Medical Problems of the Instrumentalist Musician, 311.
57 Fahn, Jankovic, and Hallett, Principles and Practice of Movement Disorders, 3.
The same disorder was also observed during the same year by Polish neurologists Edward Flatau and Wladyslaw Sterling. However, the term they used to describe this condition is perhaps more accurate to the movement disorder recognized today: “progressive torsion spasm.” Eight years after Oppenheim’s publication, German neurologist Kurt Mendel published a summary of all the known cases and modified Oppenheim’s term to “torsion dystonia.” Mendel’s contribution was significant because it recognized, for the first time, dystonia as a separate disease entity while laying out a distinction with other movement disorders such as chorea, myoclonus, athetosis, and hysteria. Although Mendel’s term is intrinsically redundant since torsion is a central component of the definition of dystonia, the term dystonia was rapidly adopted by neurologists and has served since to describe a clinical syndrome presenting that particular motor phenomenology.

The next major published research on dystonia was made by American neurologist Ernst Herz in 1944. Through a massive trilogy of papers that included 15 personal cases and 105 cases from the literature, Herz successfully demonstrated that dystonic movements involve the simultaneous firing of agonist and antagonist muscles while other movement disorders such as chorea only show contractions of agonist muscles. By means of utilizing moving pictures and frame-by-frame analysis, Herz also

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60 Dressler, Altenmüller, and Krauss, Treatment of Dystonia, 2.
61 Jankovic and Tolosa, Parkinson’s Disease & Movement Disorders, chap. 28.
62 Fahn, Jankovic, and Hallett, Principles and Practice of Movement Disorders, 259.
showed that the movements were slow, long-sustained, as well as depicting irregular contortion patterns.\textsuperscript{64}

In the following thirty years, although the definition of dystonia remained static, its etiology was further developed. In 1976, American neurologist Stanley Fahn and British neurologist Paul Eldridge completed an etiological classification of dystonia. Their research outlined dystonias as either primary or secondary, based on how the disease was acquired. Primary dystonias represented those inherited or sporadic, and secondary dystonias containing those with heredodegenerative disease or environmental insults.\textsuperscript{65} The same year, British neurologist David Marsden contributed an anatomical classification proving that the term dystonia could be applied to patients that suffered not only generalized dystonia but also focal or segmental dystonia.\textsuperscript{66}

The first research foundation focused entirely on dystonia was created in British Columbia, Canada, in 1978 and named the Dystonia Medical Research Foundation (DMRF). As part of the organization’s efforts to unify the current definitions of dystonia, an ad hoc committee was authorized to conform and revise the definition in 1987 as follows: “Dystonia is a syndrome of sustained muscle contractions, frequently causing twisting and repetitive movements or abnormal postures.”\textsuperscript{67} This definition was not updated until 2013: “A movement disorder characterized by sustained or intermittent muscle contractions, causing abnormal, often repetitive, movements, postures or both. Dystonic movements are typically patterned, twisting and may be tremulous. Dystonia is

\begin{itemize}
\item\textsuperscript{64} Kurstot and Forsström, \textit{Dystonia}, 201.
\item\textsuperscript{66} LeDoux, 416.
\item\textsuperscript{67} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 3.
\end{itemize}
often initiated or worsened by voluntary action and associated with overflow muscle activation,"\textsuperscript{68} also discussed in the first chapter of this treatise.

Also encountered within the most recent advances in the research on dystonia are the results from investigations of clinical and molecular genetics. This approach has made it possible to identify specific mutated genes present in patients with different types of dystonia. These genes have been named as DYTn genes (currently from DYT1 to DYT27).\textsuperscript{69} Nonetheless, although the most current DYTn list cannot be considered as a complete classification system since there are several forms of dystonia missing, identification of DYTn genes has paved the beginning of a pathway toward a more comprehensive classification based on etiology.\textsuperscript{70}

\textbf{CLASSIFICATION}

As with the evolution of the definition of dystonia, its classification has been continuously evolving as the disorder is more deeply understood. Currently in the medical literature, there is no definitive classification system of dystonias. The majority of authors converge on certain categories, while the grouping of these categories may vary. The classification system utilized in this treatise does not follow strictly that of any author, but it is proposed as a means to summarize some of the classification systems found more commonly in the movement disorder literature.

The following headings are the most frequently used parameters when determining and diagnosing a specific type of dystonia:

\begin{itemize}
  \item \textsuperscript{68} Albanese et al., “Phenomenology and Classification of Dystonia,” 6.
  \item \textsuperscript{69} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 9.
  \item \textsuperscript{70} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 261.
\end{itemize}
**Body Distribution**

This category includes focal, segmental, multifocal, hemidystonia, and generalized dystonia, ranging from affecting a single body part to the entire body.\(^{71}\) The importance of this category relies on its implications for diagnosis and therapy. While botulinum toxin (BoNT) may be advisable for some cases of focal and segmental dystonias, generalized dystonias most likely will require medication and surgical intervention.\(^{72}\)

**Temporal Pattern**

Generally, dystonia’s symptoms are continual, but there are instances in which the symptoms’ appearance may fluctuate.\(^{73}\) This category also determines whether the dystonic movements are produced suddenly as in paroxysmal dystonia, as opposed to task-specific dystonia in which the symptoms are always triggered by the same conditions. Persistent, task-specific, diurnal, and paroxysmal are examples of different types of dystonias with varying temporal patterns.

**Age at Onset**

Depending on the age at onset, there are recurrent patterns that can be observed in the later development of the disorder.\(^{74}\) When dystonia appears at an early age, it usually becomes generalized, whereas dystonia developed during adulthood is more likely to remain focal or segmental. In addition, there are specific types of dystonia associated

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\(^{71}\) Hubert H. Fernandez, ed., *A Practical Approach to Movement Disorders: Diagnosis and Medical and Surgical Management* (New York: Demos Medical Pub, 2007), 124.


\(^{73}\) Jankovic and Tolosa, *Parkinson’s Disease & Movement Disorders*, chap. 28.

with particular ages. For instance, dystonia that develops at ages 1 or 2 is probably linked to an inherited metabolic disorder, while sporadic focal dystonia is more often diagnosed in patients after 50 years of age. In the case of musicians, it has been estimated that FTSD develops at a mean age of 33, although there have been reported cases in which the disorder was developed between ages 16 to 75.

Etiology

Depending on the causes, dystonias can be classified as primary, secondary, or secondary-plus. Primary dystonias are those of which the origin is idiopathic or unknown, and they are generally genetically inherited. FTSD is cataloged as a primary dystonia due to its unknown cause. In contrast, secondary dystonias are the consequence of external factors inciting the development of the disorder. These factors include, but are not limited to, brain injuries, vascular causes, infections, medication-induced side effects, and direct impact of chemical agents such as manganese, copper, methanol, cyanide, carbon monoxide, and ephedrone. In addition, secondary-plus dystonias are those that appear in conjunction with other disorders such as dystonia plus myoclonus or dystonia plus parkinsonism.

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PHENOMENOLOGY

In medicine, phenomenology refers to a first-person narrative of the resulting experience after the specific symptoms of an illness make appearance. Phenomenology is crucial to achieving an accurate diagnosis of a given disease, as well as to deepen the knowledge related to the development of a particular disorder. Although the causes of some types of dystonia, including FTSD, are not yet fully understood, medical literature clearly delineates dystonia’s phenomenology due to the numerous reported cases of patients suffering from this neurological disorder.

FTSD is known to manifest itself in musicians that have had extensive training and whose instruments require maximal fine-motor skills development. FTSD’s main characteristic is the loss of voluntary motor control. It causes the affected area in the body to distort so completely that performing becomes close to impossible. The initial symptoms include, but not exclusively, loss of control in technically challenging passages. Involuntary flexion or extension of the fingers while performing are observed in pianists and violinists, while lack of control and coordination of the lips, tongue, and facial muscles is common in brass and wind players.

Paradoxically, the appearance of these early symptoms is often misunderstood by musicians as a deficiency in practice or technique rather than as a health problem.

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81 Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 3.
83 Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 3.
resulting in a worsening of the condition.  

Similarly, the results of recent research conclude that classical musicians are the most likely population to develop FTSD due to the continuous execution of complex repetitive movements over the course of many years. FTSD’s frequency of appearance in the general population is 1:3,400 while its rate in professional musicians is approximately 0.5%–8%. Furthermore, studies have shown that between 8% to 14% of musicians visiting performing arts clinics are ultimately diagnosed with FTSD.

Figure 2.3. Typical patterns of dystonic posture in a pianist, violinist, flutist, and trombone player

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89 Altenmüller and Jabusch, “Focal Dystonia in Musicians,” fig. 1.
American pianist Leon Fleisher recalls his experience with FTSD in the fourth and fifth fingers of the right hand:

“When I started practicing again, things didn’t feel quite right on my right side. My fourth and fifth fingers seemed to want to curl under. I practiced even harder, not listening to my body . . . Things got progressively worse and in less than a year those two fingers were completely curved under, sticking into the palm of my hand. No way could I play the piano.”^90

In addition to previously described features of FTSD, such as simultaneous contraction of agonist and antagonist muscles as well as the absence of pain, there is one more unique feature present in FTSD: its susceptibility to “sensory tricks,” also referred as *geste antagonistes*.^91* Geste antagonistes* or antagonist gestures were first found when observing patients with generalized dystonia in the nineteenth century.^92 These are sensory stimuli that, for unidentified reasons, dampen the effects of the dystonic contractions.^93 By means of modifying the incoming sensory information received by the brain, a pianist diagnosed with FTSD can perceive immediate relief or even complete restoration of motor control.^94 This effect can be achieved by ‘tricking’ the brain into feeling that the pianist is not actually playing on the keyboard. Several reported cases

confirm that pianists with FTSD were capable of recovering partial or almost complete motor control when they wore latex gloves while performing.\textsuperscript{95}

Nonetheless, the same \textit{geste antagonistes} do not work in all cases. For instance, the author did not experience the mentioned relief when wearing latex gloves. Still, he did experience the effects of \textit{geste antagonistes} when playing while his hand was submerged in a large bucket full of water. Although the results are undoubtedly remarkable, to say the least, \textit{geste antagonistes} work only for a short period of time until the brain correlates the new incoming sensory information with actual piano playing.

Another distinctive clinical feature of FTSD is the presence of tremor, although its prevalence in patients with dystonia ranges from 14\% to 86.67\%, according to recent studies.\textsuperscript{96} It may occur in the affected part of the body, also named as dystonic tremor, or unaffected body regions, commonly addressed as tremor associated with dystonia.\textsuperscript{97} Recent studies have shown that dystonic tremor appears either when sustaining a particular posture, during movement, and in some rare cases while resting.\textsuperscript{98} Dystonic tremor is defined in medical literature as a “spontaneous, oscillatory, rhythmical, although often inconstant, patterned movement produced by contractions of dystonic muscles.”\textsuperscript{99}

\begin{flushright}
\textsuperscript{95} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 6.
\textsuperscript{97} Pandey and Sarma, 3.
\textsuperscript{98} Pandey and Sarma, 4.
\end{flushright}
The presence of dystonic tremor in patients suffering from FTSD presents a challenge for its diagnosis since its clinical features partially overlap with those of the most common movement disorders: essential tremor, functional tremor, and Parkinson’s disease. Additionally, in the case of pianists, dystonic tremors can be mistakenly identified with an apparent weakness in the fingers affected by dystonia. Although part of the symptomatology may be similar, the key to successfully differentiate dystonic tremor from other movement disorders is to identify the unique symptoms only present in dystonia patients such as responsiveness to sensory tricks or *geste antagonistes*, display of dystonic postures when playing the instrument or performing a similar activity (such as typing), and tremor movements that are characteristically irregular and asymmetrical in opposition to coherent oscillations.\(^{100}\)

FTSD’s phenomenology also comprises mirror and overflow movements.\(^{101}\) Mirror movements are those that occur when the opposite hand attempts to perform the same task that triggers the dystonic movement in the affected hand. The author recalls mirror movements, particularly when performing left-hand repertoire; his right hand occasionally displayed dystonic movements while resting over his thigh. Overflow movements refer to the spread of dystonia to adjoining areas of the hand. Likewise, the author experienced overflow movements when he was initially diagnosed. After

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increasing the intensity of his practice sessions, the dystonic movements seemed to expand from his index finger to cover also the thumb of the right hand.

Finally, there are cases of patients diagnosed with FTSD in which the dystonic movements spread to involve contiguous body parts.\textsuperscript{102} Although it is estimated that it occurs in a small percentage of patients, in those cases the effects of dystonia escalate from a merely occupational disability to a significant disability that also affects their daily routines. Furthermore, notwithstanding the fact that the initial manifestation of FTSD is unilateral, it has been estimated that up to 25\% of FTSD patients may eventually develop dystonic symptoms on the opposite hand, especially when the unaffected hand performs the same task that triggers the dystonic symptoms in the affected hand.\textsuperscript{103}

Recognizing this affliction as a neurological disorder and not as a performance problem or practice deficiency will offer the opportunity to treat it accordingly, leading to an early diagnosis and the creation of a long-term treatment that may prevent the spreading of dystonia in those patients, and hopefully leading to a partial or full recovery.

**RISK FACTORS**

FTSD is classified as a primary dystonia; therefore, its causes are still unknown. Nonetheless, continued research and studies on this disorder have brought new insights while providing clues to achieve a deeper understanding of this condition. One of the most frequent associations to FTSD is the one that connects it with occupational overuse syndrome (OOS), repetitive strain (stress) injuries (RSI), or cumulative trauma disorders.

\textsuperscript{102} Fahn, Jankovic, and Hallett, *Principles and Practice of Movement Disorders*, 262.

Although the terminology varies, this conception suggests that FTSD may be a conceivable outcome of “biomechanical strain due to tension, pressure, or friction which is excessively forceful, repetitive, or prolonged.” Since it is estimated that the minimum number of hours required to achieve instrumental mastery is 10,000 in ten years of deliberate practice, the relationship between FTSD, repetitive movements, and occupational disorders such as OOS, RSI, or CTDs seems worthy of consideration.

A study conducted on primates in 1996 elucidates important remarks on the results of intensive training based on repetitive movements. In this study, two monkeys were trained over the course of 20 weeks. The training required them to close a handpiece, with between 3-400 repetitions per day. According to the study, “one monkey used a highly articulated hand-squeezing strategy, and the other monkey used a proximal arm-pulling strategy.” The results showed that the monkey utilizing the hand-squeezing strategy developed motor deterioration and dedifferentiation of the typically highly segregated representation of the hand in the brain. These results are consistent with the brain images observed in pianists diagnosed with FTSD, where the sensory inputs from individual fingers overlap. In contrast, the monkey using the proximal arm-pulling strategy showed mild motor deterioration, though with no motor dysfunction.

Since only the monkey that performed the task while actively utilizing both the muscles in the hand and the arm showed no motor dysfunction, it is reasonable to hypothesize that the motor dysfunction displayed by the second monkey was the result of performing the same task while utilizing only the muscles of the hand. Therefore, the results of this study show that training based on repetitive movements does not necessarily lead the subject to develop a motor dysfunction. Instead, the results lead to point out the physical approach utilized to perform the task as the cause of the motor disability rather than the task itself or the training process. Likewise, reported cases of patients diagnosed with dysphonia, writer’s cramp, and leg dystonia did not go through excessively repetitive intense training to learn to talk, write, or to walk.\textsuperscript{108} In other words, the development of dystonia in those cases was not the consequence of intensive training.

These results are of high relevance to the author of this paper when correlating them with his own experience with FTSD. The author started playing the piano at age 8 and pursued continuous formal musical training from age 10 to age 26 when he was diagnosed with FTSD. The author received piano instruction with primary emphasis on independent finger motion and independent finger strengthening while completely avoiding forearm rotational movements or wrist induced attacks. In his playing at the time, each attack was initiated and conducted by each finger in an isolated manner. He found that the recovery process started only when a more organic and holistic approach was utilized to perform on the instrument. Through the Taubman Method, the author received guidance from Edna Golandsky, one of the foremost specialists in this method.

to utilize his body in a more coordinated and efficient manner that allowed him to
perform again.

Observations on the appearance and development of FTSD have shown that its
presence occurs primarily after skill acquisition, instead of during skill acquisition. It
may, therefore, be logical to assume that FTSD occurs once dexterity has been
developed, perhaps as a result of maladaptive plasticity of the central nervous system
(CNS). The CNS operates as a composite network formed by structurally and
functionally interconnected regions. Plasticity is the capacity of the CNS to modify and
reorganize existing functional structures while generating new structures in the process.
The learning process of playing a musical instrument, through experience and training,
takes place simultaneously with significant plastic adaptations of the CNS. Studies have
shown that performing on an instrument “enhances myelination, gray matter growth, and
fiber formation of brain structures involved in the specific musical task.” Despite brain
plasticity being necessary for the successful acquisition of any given skill, the negative
aspect is that when the new-formed structures are inadequate, this may result in the
process of movement adaptation that leads eventually to FTSD.

109 Altenmüller and Ioannou, “Maladaptive Plasticity Induces Degradation of Fine
Motor Skills in Musicians,” 85.
110 G. Lubrini et al., “Brain Disease, Connectivity, Plasticity and Cognitive
Therapy: A Neurological View of Mental Disorders,” Enfermedad Cerebral,
Conectividad, Plasticidad y Terapia Cognitiva. Una Visión Neurológica Del Trastorno
Mental (Spanish; Castilian) 33, no. 3 (April 1, 2018): 189,
111 Altenmüller and Ioannou, “Maladaptive Plasticity Induces Degradation of Fine
Motor Skills in Musicians,” 81.
112 Leijnse, Hallett, and Sonneveld, “A Multifactorial Conceptual Model of
Peripheral Neuromusculoskeletal Predisposing Factors in Task-Specific Focal Hand
Dystonia in Musicians,” 109.
When learning to play an instrument, the CNS must find efficient and physiologically sustainable movements that correspond to the particular performance requirements of the instrument. During this process, the CNS also compensates for diverse anatomical restrictions such as different finger length and strength, joint motion range, or the lack of finger independence due to interconnected finger tendons. Recent findings in the research of FTSD suggest that abnormal overcompensation is the product of movement patterns that do not converge to physiologically sustainable muscle activation patterns. Compensation movements are still within the range of normal movement optimization and learning behavior. However, abnormal overcompensation relates to the execution of movements that require effort beyond the muscles’ short- and long-term physiological capacity.113

These observations are consistent with the experiences of three classical pianists, presented in detail in chapter three of this paper, that were diagnosed with FTSD and found a way to recover partially or completely. In each case, the recovery process required some kind of readjustment to their physical or technical approach toward the instrument. Paradoxically, although brain plasticity favored the creation of motor patterns that exceeded the muscle’s capacity and ultimately triggered the appearance of dystonic movements, it is also brain plasticity that could lead toward recovery. Since studies in neuroscience have shown that the brain is capable of reassigning functions to different zones after the primary structures that conduct a specific neurocognitive task have been

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113 Leijnse, Hallett, and Sonneveld, 123.
compromised,\textsuperscript{114} it would be feasible to propose that pianists diagnosed with FTSD could recover partially or completely through a retraining process that emphasizes an alternative physical approach, therefore creating new motor patterns that supply those that were corrupted with the appearance of the dystonic gestures.

Preliminary studies conducted on musicians suffering from dystonia have revealed that approximately half of the cases show profiles with higher levels of anxiety, perfectionism, and neuroticism,\textsuperscript{115} making them more vulnerable to experience negative emotions such as depression, irritability, anger, and sadness.\textsuperscript{116} These studies have suggested that the marked tendencies in the psychological profiles of musicians suffering from dystonia may act as an extrinsic triggering factor to the development of this neurological disorder.\textsuperscript{117}

However, more recent studies conducted in populations of healthy musicians and musicians diagnosed with dystonia have demonstrated that there are no significant differences in the psychological profiles between the two groups.\textsuperscript{118} Furthermore, the results revealed that musicians who showed higher levels of anxiety and perfectionism

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\textsuperscript{117} Christos I. Ioannou and Eckart Altenmüller, “Psychological Characteristics in Musician's Dystonia: A New Diagnostic Classification,” \textit{Neuropsychologia} 61 (August 1, 2014): 80, https://doi.org/10.1016/j.neuropsychologia.2014.05.014.
\textsuperscript{118} Ioannou and Altenmüller, 82.
\end{flushleft}
could be found in both groups, although the probabilities of musicians diagnosed with dystonia to develop the mentioned psychological traits are 5.9 greater than those in healthy musicians.\textsuperscript{119} Still, these studies did not measure the psychological profiles of musicians diagnosed with focal dystonia before the appearance of the motor disorder. Consequently, these studies could not determine if the anxiety levels presented by musicians with FTSD increased as a consequence of the impossibility of performing on the stage with the same quality levels prior to developing FTSD. In this sense, the author recalls that although he did not perceive significantly increased anxiety levels after experiencing the effects of FTSD, he did observe a substantial loss of confidence when performing on the stage. It can be concluded that since major life events such as being diagnosed with FTSD can significantly influence personality profiles,\textsuperscript{120} the results of these studies must be interpreted with caution.

Finally, analysis of epidemiologic studies conducted in patients with dystonia has established specific patterns in terms of the populations that are most likely to develop it, as well as gender differences. Focal dystonia has been reported in a variety of professions besides music, all of them requiring fine motor skills: “painters, artificial flower-makers, turners, watchmakers, knitters, engravers, masons, compositors, enamellers, smiths, cigarette makers, shoemakers, millers, and money counters.”\textsuperscript{121} Several cases have been

\textsuperscript{119} Ioannou and Altenmüller, 86.
\textsuperscript{120} Susanne Steinlechner et al., “Personality Profiles Are Different in Musician’s Dystonia and Other Isolated Focal Dystonias,” \textit{Psychiatry Research} 266 (August 1, 2018): 28, \url{https://doi.org/10.1016/j.psychres.2018.05.017}.
\textsuperscript{121} Tubiana and Amadio, \textit{Medical Problems of the Instrumentalist Musician}, 300.
reported in physical activities such as golf, archery, darts, pitching, billiards, tennis and table tennis, petanque, pistol shooting, cricket, long-distance runners, and baseball.

In music, the instrumentalists that develop this condition more often are pianists, guitarists, and brass players. However, the disorder can affect any part of the body required for playing the instrument, including the hand, upper arm, embouchure and tongue, or even foot in the case of drummers. The music genre being performed also appears to have an incidence in the musicians that develop FTSD. Studies have established that musicians that perform written music are more likely to develop FTSD than musicians specializing in more improvisatory genres such as jazz.

One of the most striking findings in FTSD’s epidemiology is the proportion in which the disorder affects men and women. Studies conducted in musicians have shown that men are four times more susceptible to develop FTSD than women. Although the reasons are still unknown, it has been hypothesized that the noticeable difference could

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126 Ioannou and Altenmüller, “Psychological Characteristics in Musician's Dystonia,” 80.
128 Ioannou and Altenmüller, “Psychological Characteristics in Musician's Dystonia,” 81.
be connected with hormonal cycles in women by changing cortical excitability and neuronal plasticity, making them more adaptable to their environment.\textsuperscript{129} Again, brain plasticity seems to play a fundamental role in the development of FTSD, which in turn could also explain why not all diagnosed musicians share a similar psychophysiological profile.

\textsuperscript{129} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 258.
CHAPTER 3

TREATMENT

There are a variety of treatments for musicians suffering from FTSD. Nonetheless, excluding the application of botulinum toxin injection (BoNT), the majority have been applied only to small groups of musicians who are enrolled in clinical trials. Although some of the treatments show encouraging results, logistic and financial factors play an important role in access to those treatments. Treatments being explored in clinical trials frequently involve multiple sessions within a span of 6-12 months, and even though they are often free of charge due to their experimental approach, musicians are still required to be physically present to gain admission into the clinical trial. At the time of the writing of this paper, no treatment has yet demonstrated conclusive effectiveness in reversing the underlying changes in the motor cortex that result as a consequence of FTSD,\textsuperscript{130} although there are available treatments and therapies that have shown strong evidence at restoring partial or complete motor control in FTSD patients.

The following section provides an overview of different approaches utilized in a variety of treatments developed over the last three decades to combat the effects of

\textsuperscript{130} Dressler, Altenmüller, and Krauss, 256.
FTSD. Although none of the treatments provides an ultimate cure,\textsuperscript{131} they offer important findings that could eventually lead to an integrated multi-approach with a higher percentage of efficacy.

**Botulinum Toxin Injection (BoNT)**

The most potent biologic toxin, botulinum toxin, also known as botox, is the resultant protein of the bacterium *clostridium botulinum*. BoNT was first used in the 1980s to treat ocular disorders, but soon thereafter became the first-line treatment for many different conditions ranging from several types of dystonia to gastrointestinal, urologic, dermatologic, secretory, and cosmetic disorders.\textsuperscript{132} The objective of BoNT therapy is to reach a balance between inducing enough muscular weakness to reduce the symptoms of dystonia while remaining insufficient to obstruct function.\textsuperscript{133} BoNT’s action consists of preventing the release of the neurotransmitter acetylcholine, interrupting the normal neuromuscular transmission, and resulting in muscle weakness.\textsuperscript{134} After approximately three months, the neuromuscular synapse is naturally restored by returning acetylcholine levels back to normal.\textsuperscript{135} This means that the therapeutic effects produced by BoNT dissipate after that period, obliging subsequent applications of the toxin for continued action.


\textsuperscript{132} Fahn, Jankovic, and Hallett, *Principles and Practice of Movement Disorders*, 298.

\textsuperscript{133} Dressler, Altenmüller, and Krauss, *Treatment of Dystonia*, 106.

\textsuperscript{134} Okun, *The Dystonia Patient*, 37.

BoNT as therapy has demonstrated noteworthy efficacy in the treatment of dystonias such as cervical, blepharospasm, laryngeal, oromandibular, spasmodic dysphonia, clonic hemifacial spasm, writer’s cramp, and dystonic tremor. Furthermore, the systematic application of BoNT’s injections has resulted in significant improvement in the patients’ quality of life. For example, patients suffering from spasmodic dysphonia exhibit limited intelligibility in their speech while being often perceived as extremely nervous individuals due to their involuntary voice breaks. Similarly, patients diagnosed with oromandibular dystonia often display both disfiguring jaw and perioral facial movements that can affect mastication and speech. In musicians with embouchure dystonia, dystonic movements can expand to everyday tasks, namely drinking from a bottle, eating, or speaking. In such cases, BoNT treatment offers the possibility for the patients’ return to their normal daily routines.

BoNT’s efficacy relies primarily on the following factors: accurate localization of the muscles involved in the dystonic spasm, application of the appropriate doses, and the experience, skills, and techniques utilized by the clinician performing the procedure.

139 Bhattacharyya and Tarsy, “Impact on Quality of Life of Botulinum Toxin Treatments for Spasmodic Dysphonia and Oromandibular Dystonia,” 391.
140 Falup-Pecurariu et al., Movement Disorders Curricula, 378.
141 Değirmenci, “A Glance into Botulinum Toxin Outpatient Clinic in Movement Disorders Practice,” 164.
While the muscles affected by dystonia can be anatomically determined by a specialist depending on the vector of the involuntary movement, the required dose differs between patients and the correct dosage is determined by initially injecting a small amount of the toxin and gradually increasing it, usually within two-week intervals, until achieving the desired effect. Expert practitioners are recommended for this procedure, as BoNT must be applied directly into the affected muscles, which may be problematic to reach due to their location in the body. For instance, in a pianist’s case, the muscles located in the forearm are tightly intertwined, making the application of BoNT’s injections particularly difficult. Under those circumstances, the use of electromyogram guidance may be highly recommended to increase the success rate when targeting muscles of difficult access.

Side effects of BoNT, although temporary, may include “excessive muscle weakness, shortness of breath, head drop, ptosis, diplopia, and dysphagia.” Meanwhile, the most unwanted side effect of BoNT therapy is the spreading of toxin over adjacent muscles, causing weakness in muscles unaffected by the dystonia. Another aspect that needs to be considered before beginning this treatment is that BoNT therapy focuses on reducing dystonic movements by weakening the muscles that generate the movement, even when those muscles play an active role in the execution of fine motor skills in performance on a musical instrument. By decreasing strength and consequently speed in the muscles affected by FTSD, a significant reduction of the performer’s ability and dexterity is equally inevitable. As mentioned previously in this paper, BoNT therapy only

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142 Falup-Pecurariu et al., *Movement Disorders Curricula*, 375.
143 Falup-Pecurariu et al., 376.
144 Değirmenci, “A Glance into Botulinum Toxin Outpatient Clinic in Movement Disorders Practice,” 164.
alleviates, up to a certain level, the symptoms produced by dystonia while the source of the motor disability remains intact. In fact, studies conducted on pianists diagnosed with FTSD who have received BoNT therapy show that only 10-15 percent of them have been capable of successfully continuing high levels of professional activity.\textsuperscript{145}

To conclude, BoNT is undoubtedly an invaluable resource for patients in which dystonia has severely affected their everyday life. For musicians suffering from FTSD, it acts mainly as a mild palliative, although its use in conjunction with other therapies and treatments may offer a more effective approach in the search of a partial to full recovery from FTSD.

\textbf{Oral Medications}

The use of oral medications in the treatment of dystonia largely preceded the application of BoNT injections as a mechanism to lessen the symptoms of dystonia. Concurrently, through the first half of the twentieth-century, dystonia was seen in the psychiatric profession as a manifestation of conversion disorder, based on Freudian hypotheses in psychoanalysis.\textsuperscript{146} Later technological developments, including positron emission tomography and single-photon emission computed tomography that measured regional distribution of administered radioactivity in the brain,\textsuperscript{147} wire electromyographic recording which allowed observation of simultaneous contraction of agonist and antagonist muscles, and magnetic resonance imaging that showed internal brain lesions in patients with dystonia were necessary to scientifically corroborate the existence of

\textsuperscript{146} Dressler, Altenmüller, and Krauss, 88.
\textsuperscript{147} Dressler, Altenmüller, and Krauss, 36.
dystonia as a neurological disorder. As a consequence, oral medications were first developed in other movement disorders such as Parkinson’s disease.

Levodopa was first used as a dopamine replacement to treat Parkinson’s disease in the 1960s.\textsuperscript{148} In the 1990s, molecular DNA and biochemical studies detected a type of secondary-plus dystonia, later denominated dopa-responsive dystonia (DRD),\textsuperscript{149} for which low doses of levodopa result in a substantial reduction of the dystonic contortions.\textsuperscript{150} DRD is a type of generalized dystonia with an early age onset affecting predominantly females.\textsuperscript{151} Unfortunately, patients with idiopathic dystonias such as FTSD have rarely shown improvement with dopaminergic therapy.\textsuperscript{152}

Another type of oral medication often used to treat dystonia patients is the group of drugs classified as anticholinergic agents. Anticholinergics have abundant effects both in the central and peripheral nervous systems. Therefore, they are utilized to control a variety of symptoms, including involuntary movements, bradycardia, bronchodilatation, prevention of motion-induced nausea, and gastric disorders, between others.\textsuperscript{153} Anticholinergic agents have a similar action to BoNT in the sense that they counteract the effects of the neurotransmitter acetylcholine, albeit their action is more generalized.

\textsuperscript{149} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 295.
\textsuperscript{150} Okun, \textit{The Dystonia Patient}, 7.
\textsuperscript{151} Frucht, Fahn, and SpringerLink (Online service), \textit{Movement Disorder Emergencies}, 213.
\textsuperscript{152} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 295.
affecting muscles of the gastrointestinal and genitourinary tract, salivary glands, heart, eyes, lungs, and skin.\textsuperscript{154}

Although multiple studies have reported anticholinergics producing positive results in the treatment of several types of dystonias, their use in the treatment of focal dystonias, among them FTSD, has greatly declined while being replaced by BoNT therapy.\textsuperscript{155} This change took place because anticholinergics also produce uncomfortable side effects such as “blurred vision, constipation, dry mouth, urinary disturbances, and tachycardia.”\textsuperscript{156} In addition, more recent studies have found anticholinergic drugs to cause cognitive dysfunction, memory impairment, and visual hallucinations.\textsuperscript{157}

An alternative oral medication used extensively in the past for the treatment of dystonia were the dopamine receptor-blocking drugs. Also known as neuroleptics and antidopaminergic therapy, these drugs operate principally by blocking dopamine receptors in specific pathways in the brain, and they are typically utilized in the treatment of schizophrenia.\textsuperscript{158} Nevertheless, their potential benefits in the treatment of dystonia have been exceeded by their potential unwanted side effects. These may include tardive dyskinesia, parkinsonism, and more predominantly sedation.\textsuperscript{159} Currently, their use for

\textsuperscript{155} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 296.
\textsuperscript{156} Ogino et al., “Benefits and Limits of Anticholinergic Use in Schizophrenia,” 38.
\textsuperscript{157} Ogino et al., 39.
\textsuperscript{159} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 295.
the treatment of dystonia is not recommended, though since they continue to be utilized for the treatment of several psychiatric conditions, future generations of neuroleptics might reduce their adverse effects.

The last type of oral medication utilized to control some of the symptoms caused by dystonia is the skeletal muscle relaxants (SMRs), often called antispastic or simply muscle relaxers. SMRs are classified into neuromuscular blocking agents when acting peripherally, whereas SMRs acting on the central nervous system are categorized as directly acting agents. SMRs were first used to treat muscle spasms produced by a neurological condition referred to as spasticity, and subsequently to treat patients suffering from dystonia. Although the exact mechanism of action of SMRs is still unknown, it is believed that by “inhibiting the polysynaptic reflexes in the central nervous system,” muscle tone is therefore reduced as a result. In addition, certain benzodiazepines and SMRs such as Diazepam, Lorazepam, and Baclofen employ a neurotransmitter termed as y-aminobutyric acid to support relaxation of the skeletal muscles. Although studies have shown improvement in dystonia patients when taking SMRs and benzodiazepines, before getting this type of medication, it is essential to be aware that some of their side effects may include sedation, weakness, and memory loss.

**Surgical Treatment**

Due to the risks and possible complications involved in these procedures, surgical treatments in the past were generally recommended only for extremely severe cases of dystonia in which neither oral medications nor BoNT therapy was effective. The two

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most common procedures are thalamotomy and pallidotomy. Both procedures consist of the destruction of specific areas in the brain through thermocoagulation, a technique in which cerebral tissue is exposed to sustained high temperatures over short intervals of time. Thalamotomies target the thalamus, aiming to interrupt the abnormal outflow with the prefrontal motor cortex, which results in co-contraction and overflow of activation of inadequate muscles. The goal of the pallidotomy procedure is to create a lesion in the globus pallidus internus, in order to restore the thalamus’ normal function, at least partially, by disrupting the irregular inhibitory signals sent by the globus pallidus internus. Adverse side effects and complications of these procedures may include speech disturbances (dysarthria), partial weakness on one side of the body (hemiparesis), inability to control facial movements (pseudobulbar palsy), abnormal sensations of the skin (paresthesia), personality changes, surgical site infections, and postoperative chronic subdural hemorrhage.

The irreversible nature of the thalamotomy and pallidotomy procedures in combination with their potential undesired outcomes, led to a generalized preference in the medical community for other surgical procedures to treat dystonia patients, more predominantly Deep Brain Stimulation for which the effects are reversible. Nonetheless,

162 Fahn, Jankovic, and Hallett, *Principles and Practice of Movement Disorders*, 306.
studies conducted since the early 2000s, principally in Japan\textsuperscript{166} and India,\textsuperscript{167} have shown extraordinary improvement in terms of the ventro-oral thalamotomy’s safety and effectiveness when treating patients suffering from FTSD, due to the development of new technological tools.\textsuperscript{168} Such advances in technology include computed tomography scan and cranial magnetic resonance imaging that enable more precise target alignment in conjunction with the utilization of a Gamma Knife that generates a three-dimensional coordinate system. The use of these devices suppresses the necessity of a craniotomy while also making the procedure less invasive.

During a ventro-oral thalamotomy, the patient must remain conscious. Consciousness allows the patient to perform on their musical instrument during the procedure, and to immediately report any change in the intensity of the symptoms as the surgeon monitors the presence of any side effect. This mechanism helps the specialist to determine the precise target area in the ventro-oral nucleus of the thalamus, as well as the number of micro-lesions necessary for each patient to achieve recovery. Localization of those specific zones is reached through electrical stimulation with a monopolar radiofrequency probe (electrode), which is introduced through a small hole made in the skull by the surgeon. Notwithstanding, two major advantages of this surgical procedure are that it does not require post-operative management and that the results are immediately perceived by the patient.

\textsuperscript{167} Doshi et al., “Stereotactic Thalamotomy for Task-Specific Dystonia,” 245.
Possibly the most notable study showing the efficacy of ventro-oral thalamotomy on patients suffering from FTSD and most certainly the study with the largest population of musicians was held at the Neurological Institute of the Tokyo Women’s Medical University between October 2003 and February 2017.\(^{169}\) This study included 171 FTSD patients of which 58 were musicians, including pianists, guitarists, percussionists, wind players, and string players. The thalamotomies performed in this study were MRI-guided focused ultrasound in combination with a Leksell stereotactic frame. To measure the results of the procedure, patients were evaluated based on the Tubiana’s Musician’s Dystonia Scale.\(^{170}\) This scale comprises five possible scores according to the musician’s performance ability: “inability to perform affected tasks (very severe, score of 1), perform only easy affected tasks (severe, score of 2), perform affected tasks with marked difficulty (moderate, score of 3), nearly normal function (mild, score of 4), and normal functioning (nil, score of 5).”\(^{171}\)

A percentage of immediate postoperative relief was reported by 97.7% of the patients enrolled in the study, of which 80.2% were good responders and 17.5% partial responders. The average patient score in the Tubiana’s Musician’s Dystonia Scale before and after the surgery was 1.72 and 4.38, respectively, and the effect remained stable through a mean follow-up period of 47.36 months. In addition, similar studies conducted on musicians diagnosed with FTSD have reported patients showing no sign of dystonic


symptom recurrence as long as 108 months after the surgical procedure was performed.\textsuperscript{172} Still, careful consideration must be taken before undergoing this procedure since studies have also shown that a percentage of up to 3.5\% of patients developed permanent side effects as previously described.\textsuperscript{173} Nonetheless, the evidence collected in these studies implies that stereotactic ventro-oral thalamotomy is currently the procedure with the fastest action and highest recovery rate when treating musicians diagnosed with FTSD.

The last surgical procedure employed to treat dystonia patients is known as deep brain stimulation (DBS). Deep brain stimulators work similarly to pacemaker devices, only that the electrical wires are placed within the brain. The electrical signals sent by the electrode cause the brain cells to modify their normal activity, and they are used to treat various movement disorders, including Parkinson’s disease, dystonia, and essential tremor.\textsuperscript{174} Although the exact DBS mechanism of action is still unknown, the electrical stimulation accomplishes a similar result to thermocoagulation by causing a functional blockade over the targeted area.\textsuperscript{175} A DBS system is composed of a stimulator, an extension wire, and a DBS lead. The stimulator originates electrical signals that are carried through the extension wire and delivered into specific areas of the brain by the

\textsuperscript{172} Horisawa et al., “Long-Term Improvement of Musician’s Dystonia after Stereotactic Ventro-Oral Thalamotomy,” 650.
\textsuperscript{173} Horisawa et al., “Safety and Long-Term Efficacy of Ventro-Oral Thalamotomy for Focal Hand Dystonia A Retrospective Study of 171 Patients,” E375.
DBS lead. The entire system is implanted inside the patient’s body, and the DBS system remains programmable, as its action can be modified or suspended with a remote control that is outside of the patient’s body.\textsuperscript{176}

For the treatment of dystonia, DBS focuses mainly on the stimulation of the posteroverentral lateral globus pallidus internus.\textsuperscript{177} Several studies have demonstrated pallidal DBS as a beneficial treatment specifically to counteract the symptoms from segmental dystonia, cervical dystonia, hemidystonia, and generalized dystonia.\textsuperscript{178} Even in such cases, symptom improvement is not immediate as DBS requires sustained therapy (average of three to six months) for management and optimization of the stimulator before the patient can perceive notable relief from the dystonic contractions.\textsuperscript{179}

Additionally, small studies conducted in South Korea\textsuperscript{180} and Japan\textsuperscript{181} have also shown positive results in the application of thalamic DBS to FTSD patients suffering from writer’s cramp. In both studies, the patients reported significant improvement while reaching almost normal to normal writing. At the time of the writing of this paper, no published study has tested the effects of DBS when treating musicians diagnosed with

\textsuperscript{176} Chou, Patil, and Grube, \textit{Deep Brain Stimulation}, 11.
FTSD. However, since writer’s cramp is also a type of FTSD, it would be plausible to suggest that similar positive results could be expected in musicians suffering from FTSD. Accordingly, the effects of DBS therapy in musicians suffering from FTSD remains to be furtherly researched and studied.

DBS possible complications and long-term adverse effects may include intracerebral hemorrhage, implant infections, DBS lead dislocation and/or fracture, dysarthria (reduced verbal fluency), paresthesia (abnormal sensations of the skin), ataxia (lack of voluntary coordination of muscle movements), diplopia (perception of two images of a single object), mood changes and depression. In addition, DBS requires periodical replacement of the stimulator’s generator (battery), as well as occasional electrode revisions and removals. That having been said, two of DBS’ therapy most critical advantages are that its action is reversible and that once implanted, the stimulation intensity and the target area can be amplified or reduced as needed.

**Rehabilitation and Retraining**

These types of therapies are based on the premise that FTSD affects musicians by increasing motor cortex excitability while decreasing its normal inhibition, resulting in the disturbance of sensorimotor integration and a substantial lack of motor control. Also known as a behavioral treatment, rehabilitation and retraining (R&R) therapies

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operate under the principle that the neurological dysfunction caused by FTSD can be reversed through context-specific, intensive training-based remediation. Several studies conducted with musicians suffering from FTSD have shown clinical amelioration, including reversed somatosensory map distortions after undergoing behavioral therapy.

In addition to retraining exercises, R&R therapies frequently involve immobilization and splinting of the fingers in the affected hand. Nonetheless, due to the rising number of emerging studies analyzing the efficacy of R&R therapies at restoring normal motor control in patients diagnosed with FTSD, a variety of approaches have been employed in these studies. The following section provides brief descriptions of the methods and techniques utilized in some of the studies applying R&R therapies to pianists suffering from FTSD.

A retrospective descriptive study conducted over four years on fifty-four pianists at the University of Music, Drama, and Media in Hannover (Germany) indicated that 79.2% of the subjects reported improvements in motor performance, although only 5.6% reported a complete recovery. The retraining process in this study was framed in three stages. The first stage, named deprograming, focuses on the identification of incorrect movements and the establishment of a correct hand position. The next stage aims at building mental representations of simple movements, while weak muscle groups are

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186 Jabusch and Altenmüller, “Focal Dystonia in Musicians: From Phenomenology to Therapy,” 211.
strengthened. The final stage begins when the dystonic movements are controlled, and the muscular equilibrium is reestablished. 87% of the subjects in this study employed R&R therapies, although alternative simultaneous therapies such as relaxation techniques (38%), physiotherapy (30%), acupuncture (21%), psychotherapy (23%), hand therapy (42%), and body techniques (21%) were also used by some of the subjects.

A study conducted on twenty pianists showed the result of another R&R approach termed by the author as a “slow-down exercise.” Since the dystonic movements often disappear when playing slowly, this study concentrates on guiding the subjects to play with a tempo that is slow enough to eliminate involuntary movements while gradually increasing the performing tempo. The pianists in the study were required to practice 30 minutes per day with the assistance of a metronome for two weeks. After the initial two weeks, subsequent increments of 10% to 20% in the speed were applied each successive two weeks. If the dystonic movements reappeared, patients were advised to return to a slower speed and to repeat the process. In the results of this study, twelve patients reported significant improvement and eight patients reported mild improvement, although this study did not include a long-term follow-up period to measure the sustained effectiveness of the slow-down exercise nor the extent to which pianists in the study were able to successfully return to their professional careers.

A study conducted on ten musicians suffering from focal hand dystonia at the University of Konstanz (Germany) applied an R&R therapy labeled as sensory-motor

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This therapy uses a hand splint designed to fit the hand anatomy of each patient. By means of the hand splint, one or more non-affected fingers are immobilized while remaining in their characteristic rest position when performing under normal circumstances on the instrument. This device allows the affected finger to participate in repetitive alternating individual finger movements. The subjects were required to practice diverse finger combinations during two-hour practice periods over the time span of 8 consecutive days. The results of this study showed evidence of both symptomatic improvement and cortical reorganization of the affected hand. Despite results having been collected over a short time period, these findings are meaningful because they confirm that motor cortex reorganization is potentially achievable by FTSD patients.

An analogous study was conducted by the School of Music at the University of Auckland (New Zealand), on three pianists diagnosed with FTSD. This study emphasized the necessity of establishing new motor pathways as a critical requirement to return to normal playing. With that purpose, the subjects underwent a pianism retraining program based on the utilization of an efficient biomechanical approach to perform on the instrument with the elimination of unnecessary tension. The core of this technical approach to perform on the instrument was outlined in this study as follows:

We hypothesize that the retraining improved pianism by the following mechanisms: establishing a balanced posture, refining the movement patterns so that the fingers were aligned with the keys, removing the tension in the wrist and forearm, maintaining freedom of movement in the elbow and shoulder, enabling

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the fingers to carry the weight of the arm, and releasing each note immediately after playing.191

Starting with awareness and reinternalization of body posture and hand position, the retraining sessions progressed through the performance of single notes, five-finger patterns, scales, one-hand parallel minor thirds, and diminished-seventh arpeggios. All of the exercises were performed initially at a slow tempo, which was gradually increased as long as the subjects continued to employ the newly learned technical principles. The subjects in this study participated in a minimum of ten one-hour retraining sessions over a time period of two weeks. According to the scales developed by the researchers of this study to measure performance improvement, all the pianists participating showed noticeable improvement in the technical areas explored during the R&R process, thus providing evidence that FTSD can be successfully treated through pedagogical retraining.

R&R therapies have also been combined with non-invasive brain stimulation procedures such as transcranial magnetic stimulation (rTMS)192 and cathodal direct current stimulation (tDCS).193 In those cases, brain stimulation was applied with the intention of either reducing excitability in the motor cortex (tDCS) or increasing its inhibition (rTMS) in FTSD patients,194 expecting to facilitate or amplify the effects of R&R. Participants reported moderate to significant improvement in both cases, although,

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191 De Lisle et al., 110.
given the short time frame and the small population sample in both studies, further research on the application of tDCS and rTMS as an aid for R&R therapies in the treatment of FTSD is needed in the future to consolidate these results.

R&R therapies present a promising horizon for patients suffering from FTSD. The results from numerous studies have confirmed that task-specific retraining breaks apart the cortical fusion in FTSD patients while allowing normal cortical segregation to be reestablished, thus restoring normal representations of the hand in the motor cortex.\footnote{Berque et al., “A Combination of Constraint-Induced Therapy and Motor Control Retraining in the Treatment of Focal Hand Dystonia in Musicians,” 157.}

Common elements found in different types of R&R therapies integrate identification and application of a long term sustainable biomechanical approach to perform on the instrument and a retraining period centered on slow practice with gradual increases in speed occurring only if the dystonic symptoms remain nonexistent. Advantages of R&R therapies include the absence of side effects, which are a common denominator present in all other types of treatments for FTSD, long term improvement, and the possibility of permanently adopting a more efficient biomechanical approach when performing on the instrument.\footnote{De Lisle et al., “Effects of Pianism Retraining on Three Pianists with Focal Dystonia,” 110.}

R&R therapies require the patients to have a comprehensive understanding of their condition to ensure full commitment through the process since it has been estimated that a minimum of eight months is necessary to observe a statistically significant decline in the frequency of dystonic contractions.\footnote{Berque et al., “A Combination of Constraint-Induced Therapy and Motor Control Retraining in the Treatment of Focal Hand Dystonia in Musicians,” 157.}

Furthermore, researchers have emphasized
the need for consistency within the retraining process: “Learning needs to be progressive so that the foundations are perfected, and reinforced by focused repetition.”\textsuperscript{198} Studies have also shown that when undergoing R&R therapies, the efficacy of the treatment is contingent upon the patient’s complete avoidance of concert playing during the retraining process.\textsuperscript{199} As the purpose of R&R therapies is to create new motor patterns that eventually replace the old disrupted motor patterns, performance on the stage might retrieve disrupted motors patterns while weakening the newly learned motor patterns. To conclude, it is possible to speculate that in FTSD patients the disrupted motor patterns were acquired through years of training; therefore, complete internalization and automatization of new motor patterns might ultimately take an equivalent time frame.

**Three Prominent Pianists Recovering from FTSD**

The following section presents publicly available accounts from three pianists diagnosed with FTSD, their descriptions of the process they underwent to recover from FTSD, and the author’s annotations referencing those descriptions to content previously presented in this document.

**Michael Houstoun**

Michael Houstoun was born in Timaru (New Zealand) in October 1952. His career as an international concert pianist was launched at the age of twenty upon receiving the Bronze Medal at the Van Cliburn International Piano Competition in 1973. Mr. Houstoun was also a laureate pianist at the Leeds International Piano Competition in


\textsuperscript{199} De Lisle, Speedy, and Thompson, 127.
1975 and the Tchaikovsky International Piano Competition in 1982. Since then, he remained active on the stage while performing a robust repertoire spanning from J. S. Bach to the present day. Some of his most highlighted performances include two presentations of the complete Beethoven sonatas in a seven-concert cycle and the 48 Preludes and Fugues of Bach's “Well-tempered Clavier” in two-concert events. Mr. Houstoun has recorded for Rattle Records since 1999, and his albums have earned the Classical Record of the Year award on five occasions.200

In 2000, the development of FTSD resulted in his inability to continuously perform on the stage. FTSD affected the third and fifth fingers of his right hand, thus compromising his playing of scales, chords, and octaves. He describes the symptoms as follows:

…I could get a clean signal through to my 3rd finger, but not to my 4th (and eventually my 5th). Movements – and even intentions of movements – in those fingers triggered the involvement of my 3rd finger… If I rested my hand on a table with all the fingertips touching the tabletop, I simply could not raise my 3rd finger at all unless I raised the 4th finger at the same time. My playing lost its clarity, evenness, and I was exhausted after only a few minutes. Through all this there was no pain, only dysfunction.201

These symptoms reflect evidence of simultaneous firing of agonist and antagonist muscles202 due to lack of differentiation in the representation of the digits in the primary somatosensory cortex,203 as well as abnormally prolonged muscle firing;204 all of them

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201 “Michael Houstoun.”
203 Kurstot and Forsström, Dystonia, 201.
204 Lin and Hallett, “The Pathophysiology of Focal Hand Dystonia,” 2.
were characteristic symptoms displayed by FTSD patients. After a thorough examination, Mr. Houstoun was finally diagnosed with FTSD, as he recalls:

After extensive nerve conduction tests two neurologists separately confirmed focal dystonia as the diagnosis. Both agreed it was incurable and could not recommend any way for me to treat it. (Ultimately our team concluded that I must have been on the verge of developing FD for some time and it just took some particularly obsessive practising – Chopin Op.10 No.2 – to kick it in. We cannot think it came suddenly out of nowhere.)

The road to recovery for Mr. Houstoun started first by taking his muscles back to a healthy state. This process included numerous physiotherapy and acupuncture sessions to remove all the deep knots located in his forearm: “It took well over a year of treatment before my muscles and postural alignment approximated a normal condition.” A multidisciplinary team of specialists, including a sports doctor, an osteopath, a doctor with extensive experience treating injuries in musicians, and a physiotherapist/acupuncturist led the way to a crucial conclusion necessary for the creation of a recovery plan: “Eventually I/we settled on the idea that the involuntary movements could only be countered through relaxation and that this was primarily a mental matter. And the changes which needed to be made had to be made at the piano.” Although relaxation techniques alone are not effective to suppress the resultant dystonic muscle spasms caused by FTSD, careful reconsideration of technical principles in the keyboard is a cornerstone of all the studies that have successfully applied rehabilitation and retraining therapies (R&R) in pianists diagnosed with FTSD.

Mr. Houston’s recovery process on the keyboard was guided by Dr. Rae de Lisle, currently an Associate Professor of Piano at the University of Auckland:

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205 “Michael Houstoun.”
206 “Michael Houstoun.”
I showed my playing to a respected and trusted colleague who gradually guided me to a remodelling of my RH technique. We freed up my elbow, realigned my arm and hand, lowered my wrist, changed the way I raise my fingers. I learned how to release what was not in use. This took a long time and required a lot of patience, as indeed had all the treatments. Five years after the first signs of FD I played my first full solo recital.\textsuperscript{207}

FTSD occurs typically in experienced musicians, musicians who have undergone several years of intensive musical training. Consequently, closely reevaluating the technical foundation is a difficult task requiring humility and forbearance, as Mr. Houstoun remembers:

Rae de Lisle was the final piece in the puzzle. It was not easy for a man of my experience to yield a technique that had ‘paid the bills’ for 30 years. But Rae’s beautiful gentle manner, her deep understanding of the mechanics of piano playing, her quick and accurate eye, her ability to listen to countless ‘wrong ones’ in the search for the ‘right one’ – this all enabled a gradual and powerful remodelling of the way I use my RH, a remodelling from which I could not easily slip backwards.\textsuperscript{208}

\textbf{Hung-Kuan Chen}

Hung-Kuan Chen was born in Taipei (Taiwan). One of the leading pianists of his generation, Mr. Chen earned top prizes in the Arthur Rubinstein, Busoni, and Geza Anda International Piano Competitions and at the Young Concert Artists International Piano Auditions. He was also a laureated pianist at the Queen Elisabeth, Montreal International Musical and Van Cliburn International Piano Competitions.\textsuperscript{209} Currently, Mr. Chen is on the faculty of The Juilliard School and is a visiting professor at the Music Department of Yale University.

\textsuperscript{207} “Michael Houstoun.”
\textsuperscript{208} “Michael Houstoun.”
Mr. Chen was diagnosed with FTSD in 1992, affecting the fourth and fifth fingers of his right hand. He describes the symptoms as follows:

It appears as if the body is making strange decisions, disobeying one's wishes! In reality it is the result of the accumulation of complex and faulty movements. From the body’s point of view, it may even be to fulfill our needs, or even to —rescue us. This is the how I perceive Focal Dystonia… When I realized that the medical world was in the dark about FD in 1992, I decided to begin my own research.\textsuperscript{210}

After being diagnosed with FTSD, Mr. Chen concluded that FTSD was the result of emerging disrupted movement patterns overlapping with the original movement patterns that were established before the appearance of the dystonic spasms. He states that recovering from FTSD is possible when retrieving the original set of movement patterns: “It is a process of re-acquainting oneself with the original movement memory set, rather than creating a new one. By re-energizing the original set, we bring it to the foreground. This is of course, more easily said than done!”\textsuperscript{211}

Mr. Chen’s views on FTSD are in accordance with the research on dystonia conducted by Joaquín Farias, a Spanish neurologist. Farias calls the first phase in the rehabilitation process of FTSD synaptic plasticity. This phase requires the patient to consciously inhibiting the dystonic response while performing voluntary movements instead.\textsuperscript{212} Farias proposes that previously learned motor patterns do not need to be

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\textsuperscript{211} Chen. \\
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relearned but regained: “The control has been with us all the time and has not been lost; it has merely been temporarily distorted.”

Mr. Chen recovered control over his movements by developing a comprehensive muscular awareness while performing on the keyboard from the simplest to the most complicated tasks:

I started from the last key in the highest register; the lightest in weight, and cautiously placed my index finger on surface of the key… Then, I started to press down the key — just enough pressure to register a slight key movement— about half of a millimeter or so… I took regular breaks; at each successive attempt, the finger pressed deeper. After two hours I was able to press down all the way without triggering FD movement… At this point I believed I had successfully removed the unwanted new compensatory movements caused by the faulty set of memory in the vertical direction. And this was the beginning of my recovery.

Although Mr. Chen and Dr. Farias’ understanding of FTSD conceptually differs from the basic premise in rehabilitation and retraining (R&R) therapies, the process followed to achieve recovery is equivalent. It starts by slowly reestablishing an effective physical approach when performing on the instrument:

Traditional piano exercises have often included some notoriously mechanical aspects such as practicing loud and hard or with excessive movements. After doing a new kind of soft-touch practice, I found it to be highly effective and less annoying. The results in my students are surprisingly good.

Similarly, Mr. Chen also emphasizes on the importance of repetition to consolidate efficient movement patterns, another fundamental notion applied to R&R:

Repeating these steps in endless positions and combinations… This takes time, yet is highly effective. The brain and body has a way to register these movements on its own, all we have to do is to stay there long enough for it to register…

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213 Joaquín Farias, Rebellion of the Body: Understanding Musician’s Focal Dystonia (Sevilla: Galene Editions, 2006), sec. CONTROL.
214 Chen, “My FD Story Revised.”
215 Chen.
initial improvement was gratifying, but the gradual and slow improvement that followed, required great patience.\textsuperscript{216}

Finally, Mr. Chen’s recovery process also required a re-examination of certain technical principles:

Occasionally I notice certain playing habits, which induce FD response in me. One typical one is playing with heavy arm weight and grabbing or stretching action. Although it might be interesting and expressive in slow playing, when used at higher speed, it can cause overlapping commands and inducing this affliction. I have experimented with certain approach and noticed the immediate FD response.\textsuperscript{217}

\textbf{James Litzelman}

James Litzelman is an American pianist, teacher, lecturer, and adjudicator. He is currently director of the graduate program in piano pedagogy at the Catholic University of America in Washington, DC.\textsuperscript{218} Dr. Litzelman has sustained an active career as a performer and lecturer throughout the United States, Mexico, Europe, China, and Russia.\textsuperscript{219} He was diagnosed with FTSD in 1993. The key to Dr. Litzelman’s recovery was the development of a retraining technique, which he has denominated symmetrical inversion. This type of practice consists of mirroring exercises that equally match the distribution of black and white keys in both hands: “If you play a D-major five-finger pattern in your right hand while playing a G-minor five-finger pattern in your left hand, starting on the thumbs in each hand, you are playing in symmetrical inversion.”\textsuperscript{220}

\textsuperscript{216} Chen.
\textsuperscript{217} Chen.
\textsuperscript{220} Litzelman, “Recovering from Focal Dystonia,” 60.
Symmetrical inversion exercises allow a pianist suffering from FTSD to perform identical tasks in both hands while providing immediate visual, aural, and tactile feedback from both hands. In addition, this mechanism permits the pianist to “mirror” natural movements from the unaffected into the affected hand. Since neurologists in the past have identified the hand of an FTSD patient as: “a hand that has forgotten its cunning,” symmetrical inversion exercises seem to offer the possibility of restoring normal mobility by transferring normal motor patterns from the unaffected hand into the affected one.

Through his experience with FTSD, Dr. Litzelman also noticed the importance of developing body awareness to eliminate any unnecessary tension:

… since I was so focused on my fingers, to the point of obsession, really, I wasn’t aware of what was happening to my body. This hindered my recovery because I was learning to play with tension in all sort of places—my mouth, face, shoulders and upper back, and even in my left hand—and all of this had to be unlearned as I continued my recovery.222

**Juan Nicolás Morales – My Own Story With FTSD**

The following section contains the study author’s personal account of his diagnosis and continuing recovery process from FTSD:

I was born in Bogota (Colombia) in 1987. My formal musical training started at age ten at the National Music Conservatory. In 2012, I completed my Bachelor of Music degree, after a gratifying graduation recital in which I performed, among other pieces, Beethoven’s sonata Op. 57, Balakirev’s *Islamei*, and Stravinsky’s *Three Movements from Petrushka*. I took one year off before applying for a master’s degree, but in the meantime,

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222 Litzelman, “Recovering from Focal Dystonia,” 60.
I applied to a piano competition in Europe. As part of the preparation process, I gave a recital in April of 2013, including Ravel’s *Gaspard de la Nuit*, Chopin’s etude Op. 10 no. 4, and Liszt’s Transcendental etude no. 4, *Mazeppa*. The recital went fairly well, as I was feeling in pretty good shape for the competition. I remember taking one day of break and then coming back to my daily practice routine. As soon as I placed my right hand on the keyboard, the symptoms became evident. Despite my best efforts, I was not able to prevent my second finger from curling to touch the palm of my right hand while I was playing. That was when the nightmare began.

Perhaps one of the most challenging aspects of FTSD is that it is not a condition with a gradual curve of deterioration. The symptoms appeared without giving me any sign, at least that I could understand at the time, of what was coming. There was no time for adjusting or assimilating my new reality. It literally felt as if my whole pianism was suddenly gone overnight. After thorough psychological, psychiatric, physical, and neurological examination, I was finally diagnosed with FTSD a couple of months later. The emotional impact was devastating: “… This condition is incurable, though since you are a young man, I suggest you do something else with your life…”, said the neurologist who diagnosed me.

It was a violinist friend who helped me realize that first I needed to see myself from a different perspective. Musicians often develop and consolidate our confidence and identity based on the fact of being performers. Consequently, when, even temporarily, being a performer is no longer a possibility, we tend to lose our sense of purpose as members of a community while leaving aside the fact that we still can be valuable colleagues, teachers, friends, sons, siblings, or parents. Understanding that developing
FTSD was not my fault and that there was nothing to be ashamed of was essential in starting a recovery process.

Notwithstanding FTSD, I continued pursuing my dreams in music, hoping to find a solution along the way, and applied for a master’s degree in piano performance. The fact that the auditions were short, in general between 10 to 20 minutes, helped me to get through and I ended up gaining admission to the Manhattan School of Music (MSM). Unfortunately, during this process, the symptoms worsened to the point in which my thumb was flexing simultaneously with my second finger as I was playing. Just a couple of months before moving to New York, I vividly recall teaching a Clementi sonatina to a beginner student and not being able to play the scales in the first movement when trying to demonstrate some of the formal aspects of that piece.

My first year at MSM was really tough. Surviving in a highly competitive conservatory without being able to play even the simplest scalar passages meant touching bottom for me. I had tried every therapy and procedure I could, including botulinum toxin injections, acupuncture, Alexander Technique, homeopathy, physical therapy, therapeutic massages, and visualization techniques, but none of them helped me. That was when I first met Edna Golandsky. In the beginning, she was not pleased about my trying to recover while still performing concert repertoire, but I had no other choice. Ms. Golandsky, one of the foremost specialists in the Taubman Method (TM), patiently guided me in exploring new horizons that ultimately would allow me to continue my career.

Based on physiological research, Mrs. Dorothy Taubman developed a set of technical principles, later known as the Taubman Method (TM), that could be applied
consistently by pianists in performance with the intention of putting their bodies under the least possible amount of effort and stress by means of an efficient and coordinated utilization of the body’s biomechanical resources. She believed that “anyone could learn to play at an expert level with the correct understanding of function and movement.”

When referring to Ms. Taubman and the TM on an interview conducted in 2010, Leon Fleisher agreed upon her understanding of what a healthy pianistic technique is and what it is not:

I went and saw her, and she really understood how the hand moves, what is healthy for the hand, what is not healthy for the hand. What I found not healthy for the healthy hand as she also found not healthy for the hand is what you might call “Russian technique”, which is treating the fingers like little hammers and to develop them to be as strong as they could possibly be. That has nothing to do with music making. Eventually it might take you to the circus, and you can show how strong you are. Maybe you can play a note and the thing could go up and hit a bell.

In an interview conducted in 2012, Ms. Golandsky outlined two of the basic principles of the TM: “don't isolate muscles, or choose movements that cause them to work in opposition; and don't curl your fingers, or stretch them.” A similar approach is pursued by Dr. Rae de Lisle, the therapist who guided Michael Houstoun into his recovery process from FTSD, in her description of what an ideal pianistic posture involves:

… a four-point balance between the ischial tuberosities and the feet, enabling free movement of the body; active abdominal muscles that support the body, enabling the back to remain straight; shoulders dropped and relaxed; and elbows and wrist

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225 Smith, “Edna Golandsky on Taubman Technique,” 51.
floating and free of tension. The forearm is used for transferring the weight from the shoulder to the hand without pressure; the shoulder opens freely, without excessive ulnar or radial deviation of the wrist; the fingers should be as closely aligned to the axes of the keys as possible; the metacarpophalangeal and interphalangeal joints should be softly flexed, and each finger should be released immediately after playing.226

Over the first few months of lessons with Ms. Golandsky, I realized that in the past I played without any awareness of how I was using my body to perform on the piano. In my mind, every technical challenge needed to be solved with hard work and discipline. It worked for several years, though probably only because I was very young, and my body could still endure such harsh and unnatural training. Mr. Fleisher, in the same interview, describes the type of approach to the instrument that I was using in the past:

I think not only do most musicians practice their instruments too much, I think there is much too much mindless work. In other words, the approach that is taken is that they think it’s a physical activity, so they have to train their muscles, even the little muscles of the fingers and in the hand. They feel they have to train them and strengthen to make them as strong as their biceps, triceps and quads – that would be the ideal thing. That’s all wrong.227

During my time at MSM, I concluded that my notions of what I often called a “good technique” were somehow misguided. I used to think that any pianist that could play evenly and consistently fast scales, arpeggios, octaves, etc., had a “good technique.” Before my diagnosis of FTSD, it did not matter to me how it was achieved or what the consequences could be for the body in the future, as I was probably looking for results at any cost. Only after developing FTSD, I concluded that a true good pianistic technique

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227 Woo, “Focal Dystonia in Pianists,” 91.
must be a reliable and systematic approach to utilize one’s body and consistently meet all playing requirements while also being biomechanically sustainable in the long term.

The fundamental pillar of the TM relies on the use of the muscles located in the forearm – pronator and supinator – that enable rotations to either side. Ms. Golandsky defines it as follows:

Rotation is a motion of the forearm, which could be compared to turning a doorknob. This movement is the foundation of Mrs. Taubman's work. There are single rotation motions, to play notes going in opposite directions, and double rotation, for notes moving in the same direction. These motions enable the forearm to assist the fingers with arm weight and covering larger intervals.\(^{228}\)

In accordance with this line of thought, studies have also determined that the utilization of pronation–supination movements when playing the piano are the result of motor pattern adaptations, which are “driven by normal movement optimization and learning behavior”\(^{229}\) while allowing larger muscles to control and support finger actions. In the aforementioned interview, Mr. Fleisher also reflects on the importance of the use of forearm rotations as a base for an efficient pianistic technique:

But she [Mrs. Taubman] had a much more sane and similar approach physically, similar to Schnabel. Her power came not from the individual [finger] because there’s a limit. These are really tiny muscles. You can work on them twelve hours a day for twenty years; they will only get so strong, because they are so small. Someone like Dorothy [Taubman] understood that the power, the real power, comes from rotation. These two muscles, the supinator and the pronator, are very powerful muscles, and you get the power from those, not from the individual fingers, and the weight from the upper part of the body.\(^{230}\)

\(^{228}\) Smith, “Edna Golandsky on Taubman Technique,” 51.
\(^{230}\) Woo, “Focal Dystonia in Pianists,” 94.
Seven years after being diagnosed with FTSD, I sincerely believe that achieving full recovery is possible through a thorough technical retraining process. In retrospect, being diagnosed with FTSD pushed me to learn much more about my body and how to use it in an effective and coordinated way when performing the piano. I also discovered that although FTSD impaired my right hand’s dexterity, it did not affect my artistry and understanding of music. FTSD gave me the opportunity of studying repertoire that I had neglected for many years, which in turn helped me to grow as an artist and to improve my tone and sound control.

Another positive outcome of FTSD, as could be expected, is that the relearning process of technical fundaments occurs much faster in the non-affected hand. Studies have shown that the retraining process of the affected hand also has a marked positive effect in the non-affected hand by an unconscious transference of the newly learned technical principles into the non-affected hand resulting in an overall pianistic improvement.\(^{231}\) I found these conclusions to be true in my case since my left hand improved remarkably through the last few years allowing me to perform satisfactorily with orchestra the Ravel Concerto for the Left Hand both in Colombia (2019) and the United States (2020), although when I just started learning this concerto, I had to fight relentlessly against my old technical habits until the technical retraining process was more consolidated.

Slow practice is crucial to achieving a full recovery from FTSD. Because of diverse professional and financial reasons, since I was diagnosed with FTSD in 2013, I

\(^{231}\) De Lisle et al., “Effects of Pianism Retraining on Three Pianists with Focal Dystonia,” 110.
was never able to truly slow down my piano practice to focus on feeling that each movement in my playing was solid, that each movement felt right. There was always an upcoming concert or an accompaniment part to study, and I pushed my way again through it while undoubtedly hindering my recovery process. Complete recovery from FTSD requires time and patience to consolidate the new technical fundaments while giving the body time enough to internalize and automatize the new movement patterns. Since the old technical habits were acquired through years and probably hundreds of hours of practice on the instrument, retrieving those motor patterns is quite easy.

Meticulous observation and awareness of each movement when practicing point out the road to recovery, as one of the pianists participating in an R&R study acknowledges in the interview after the completion of the study:

> I grew up with focal dystonia so now I have to be very patient, rigorous and disciplined because sometimes some old reflexes are coming back to the surface [...] I have to work on this. Each order has to be well executed and no movement has to be done without order. That’s the way of mastering dystonia: giving order again and again. So you have to be patient and rigorous. If you had bad habits for many years it’s not in one week of work you’re gonna adopt good reflexes and erase all the rest. It’s a long work.\(^{232}\)

For the reasons previously exposed, I would highly recommend avoidance of repertoire learned before the diagnosis of FTSD, at least until the recovery process is completed. Repertoire pieces that were learned before the appearance of FTSD can easily trigger the dystonic spasm since they were coded into the brain while using the old motor patterns.

Although my recovery process has not been ideal, through the application of principles from the TM I have been able to regain significant motor control which permitted me to perform on the stage technically challenging works such as Liszt’s *Après une lecture du Dante: Fantasia quasi Sonata* or Bartok’s sonata Sz. 80, in addition to the successful completion of all the performance requirements for my master's and doctoral degrees. Only at this point in my career I finally have the privilege of taking the time necessary to fully recover, and this time I am planning on giving my body all the time it needs to do so. A final thought on FTSD, the recovery process from FTSD also entails recognizing and appreciating even the smallest steps and improvements, as Hung-Kuan Chen recalls:

What gave me the drive and courage to find a cure? On one side was the curiosity about the human body, awareness and consciousness; and on the other, my desire to continue my art. This was the biggest learning curve I had ever encountered. It meant having to detach from ego and ambition. It taught me to embrace all that comes to me and be extremely grateful…to notice the tiny things – those details which create a full life and are often missed by most people. To be ‘in the moment’ sounds clichéd but is not. And as part of the search for meaning, the joy of being able to play again – that was a true miracle.  

This chapter presented several of the currently available treatments for FTSD, in addition to the accounts of pianists who have resumed their professional careers after undergoing a retraining process that, although difficult, allowed them to regain the motor control and dexterity lost as a result of FTSD. Moreover, this chapter demonstrates that recovery from FTSD is possible, as well as, it provides evidence that new treatments are currently being developed meanwhile showing highly encouraging results for the future.

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233 Chen, “Hung-Kuan Chen Biography.”
CHAPTER 4
SUMMARY AND CONCLUSIONS

What is FTSD?

Focal task-specific dystonia (FTSD) is a movement disorder characterized by simultaneous contraction (co-contraction) of agonist muscles (the muscles actively producing a movement) and antagonist muscles\textsuperscript{234} (the muscles delimitating the range of a movement), resulting in sustained or intermittent muscle contractions as well as abnormal movements and/or postures, which are worsened by voluntary action.\textsuperscript{235} FTSD produces a disruption of the precise balance of muscular excitation and inhibition necessary for the execution of fine motor skills. The lack or absence of inhibition displayed by FTSD patients is evidenced as the central nervous system is not actively inhibiting the action produced by antagonist muscles and therefore impeding movement accuracy and fluency while allowing activation of uninvolved muscles in a movement pattern.\textsuperscript{236}

Several movement disorders, FTSD included, are the result of pathologic alterations to the basal ganglia (BG) area of the brain. The function of the BG can be defined as leading intention into action. It produces and sustains motor patterns,

\textsuperscript{235} Albanese et al., “Phenomenology and Classification of Dystonia,” 6.
\textsuperscript{236} Lin and Hallett, “The Pathophysicsiology of Focal Hand Dystonia,” 109.
meanwhile inhibiting undesired movements.\textsuperscript{237} Disorders of the BG cause either the incapability to perform desired movements as in Parkinson’s disease, or the incapacity to prevent unprompted movements as in FTSD. Neuroimaging studies conducted on dystonia patients have shown that the impaired inhibition of the BG occurs as a consequence of lack of activity in the globus pallidus (GP), a subcortical structure in charge of inhibiting the thalamus, leading to overactive thalamocortical projections.\textsuperscript{238} Although the BG’s organization and the interactions among its inner structures are yet to be further investigated,\textsuperscript{239} the current knowledge on FTSD suggests that the deficiency in the BG’s function to enable cognitive controls take over motor patterns ultimately results in loss of motor control as experienced by FTSD patients.

FTSD differs from other types of dystonias because it is triggered only by specific tasks, and it exclusively affects a single body part. Likewise, FTSD is a painless condition as opposed to most muscle-related affections,\textsuperscript{240} although the continuous tension originated from the dystonic spasm might eventually cause muscular fatigue. FTSD can be temporarily dissuaded through a mechanism known as “sensory trick” or \textit{geste antagonistes}.\textsuperscript{241} Through the modification of incoming sensory information received by the brain, pianists diagnosed with FTSD have been able to recover partial to almost complete motor control when utilizing \textit{geste antagonistes} such as wearing latex gloves while performing.\textsuperscript{242} Unfortunately, the effect from \textit{geste antagonistes} works only

\textsuperscript{237} Standring, \textit{Gray’s Anatomy}, 369.  
\textsuperscript{238} Kurstot and Forsström, \textit{Dystonia}, 201.  
\textsuperscript{239} Standring, \textit{Gray’s Anatomy}, 364.  
\textsuperscript{240} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 3.  
\textsuperscript{241} Frucht, Fahn, and SpringerLink (Online service), \textit{Movement Disorder Emergencies}, 4.  
\textsuperscript{242} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 6.
for a very short period of time, until the brain recognizes the incoming sensory information as actual piano playing.

FTSD symptoms include, in some cases, sporadic tremor particularly when sustaining specific postures or during the execution of particular movements.\textsuperscript{243} Whereas FTSD tremor deviates from other movement disorders due to its distinctively irregular and asymmetrical oscillations.\textsuperscript{244} Another phenomenon subsequent to FTSD symptoms is the development of mirror movements occurring when the unaffected hand is performing tasks similar to those that trigger the dystonic spasm in the affected hand.\textsuperscript{245} In such cases, the affected hand will display dystonic contortions even while resting. Lastly, studies have indicated that as far as 25\% of pianists diagnosed with FTSD develop FTSD symptoms in the unaffected hand\textsuperscript{246} while in some cases, the dystonic spasms expand to adjacent fingers over time\textsuperscript{247} or even spread to involve contiguous body parts.\textsuperscript{248}

Research on FTSD has demonstrated that the most likely population to develop FTSD is classical musicians,\textsuperscript{249} and within that population, pianists comprise the highest number of patients diagnosed with FTSD.\textsuperscript{250} Pianists diagnosed with FTSD experience loss of voluntary motor control while exhibiting involuntary flexion or extension of

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\textsuperscript{243} Pandey and Sarma, “Tremor in Dystonia,” 4. \\
\textsuperscript{244} Schwingenschuh and Deuschl, “Chapter 19 - Functional Tremor,” 232. \\
\textsuperscript{245} Fahn, Jankovic, and Hallett, \textit{Principles and Practice of Movement Disorders}, 262. \\
\textsuperscript{246} Doshi et al., “Stereotactic Thalamotomy for Task-Specific Dystonia,” 245. \\
\textsuperscript{247} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 198. \\
\textsuperscript{248} Ettinger, Weisbrot, and Cambridge University Press, \textit{Neurologic Differential Diagnosis}, 158. \\
\textsuperscript{249} Jabusch and Altenmüller, “Focal Dystonia in Musicians: From Phenomenology to Therapy,” 211. \\
\textsuperscript{250} Altenmüller and Jabusch, “Focal Dystonia in Musicians,” 5. 
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fingers during performance.\textsuperscript{251} Despite the unwanted motion being clearly detectable by the performer, FTSD patients are unable to prevent it from reoccurring.\textsuperscript{252} As a result of these involuntary movements, performance has been deemed close to impossible for instrumentalists suffering from FTSD.\textsuperscript{253} In musicians, FTSD symptoms have been reported in patients ranging from ages 16 to 75.\textsuperscript{254} In terms of its occurrence, it has been estimated that up to 8\% of professional musicians are affected by FTSD.\textsuperscript{255} Furthermore, recent studies have confirmed that as much as 14\% of musicians that visit specialized performing arts clinics suffer from FTSD.\textsuperscript{256}

\textbf{Which Are the Causes of FTSD?}

FTSD is categorized in neurology as a primary dystonia; consequently, its cause remains unknown. Researchers in the past have suggested that FTSD in musicians may be the result of occupational overuse syndrome (OOS), repetitive strain (stress) injuries (RSI), or cumulative trauma disorders (CTDs). Notwithstanding, based on the fact that FTSD has also been reported in patients performing common tasks such as the use of a spoon,\textsuperscript{257} walking, talking, or writing;\textsuperscript{258} it seems conceivable to conclude that intensive

\textsuperscript{251} Altenmüller and Jabusch, 3.
\textsuperscript{252} Tubiana and Amadio, \textit{Medical Problems of the Instrumentalist Musician}, 336.
\textsuperscript{253} Huettenrauch, “Health Resources for Performers,” 330.
\textsuperscript{258} Dressler, Altenmüller, and Krauss, \textit{Treatment of Dystonia}, 198.
and prolonged training of a specific task is not necessarily a detonating factor for FTSD. Another reason to discard OOS, RSI, and CTDs as possible causes for FTSD is that in contrast to those afflictions, FTSD patients do not experience any improvement or relief from the symptoms after resting for extended periods of time.

Researchers in the past have also linked the development of FTSD with psychological profiles presenting a marked tendency of high anxiety levels, perfectionism, and neuroticism.\textsuperscript{259} However, these conclusions were the result of examining musicians after being diagnosed with FTSD, omitting the possibility that their psychological profiles could have been greatly affected due to the diagnosis of FTSD. Moreover, research conducted on spasmodic dysphonia patients, another type of FTSD, has also shown that: “…psychological and emotional symptoms and an overall poor quality of life in patients with spasmodic dysphonia were found to be secondary to, rather than the cause of, the voice disorder.”\textsuperscript{260}

Considering that pianists who have recovered successfully from FTSD through the application of rehabilitation and retraining (R&R) therapies which emphasized the utilization of a more effective biomechanical approach when performing on the instrument, a hypothesis worthy of examination is that FTSD could be the result of a suboptimal technical approach to the instrument that could no longer be sustained by the body.\textsuperscript{261}

\textsuperscript{259} Ioannou, Furuya, and Altenmüller, “The Impact of Stress on Motor Performance in Skilled Musicians Suffering from Focal Dystonia,” 227.
\textsuperscript{260} Bhattacharyya and Tarsy, “Impact on Quality of Life of Botulinum Toxin Treatments for Spasmodic Dysphonia and Oromandibular Dystonia,” 391.
\textsuperscript{261} De Lisle et al., “Effects of Pianism Retraining on Three Pianists with Focal Dystonia,” 110.
Current Treatments for FTSD

The treatments utilized in the past to control the FTSD symptoms include, among others, the application of botulinum toxin injection (BoNT), oral medications, surgical treatments, and rehabilitation and retraining (R&R) therapies. After scrupulous consideration of the balance between their effectiveness in the treatment of FTSD symptoms and their potential side effects, the following are the current treatments that show the utmost promising overall results:

*Stereotactic ventro-oral thalamotomy* – Although the precise reason for the success of this procedure when treating FTSD patients is not yet fully understood, its purpose is to interrupt the abnormal outflow between the thalamus and the prefrontal motor cortex, which results in co-contraction (agonist and antagonist muscles) and overflow of activation of inadequate muscles. This procedure involves the generation of micro-lesions in the ventro-oral nucleus of the thalamus through exposure to sustained high temperatures over short intervals of time.

The most noteworthy study showing the efficacy of ventro-oral thalamotomy on patients suffering from FTSD was conducted in Japan between 2003 and 2017. This study included a population of 171 FTSD patients of which 58 were musicians, containing pianists and other instrumentalists. 97.7% of the patients enrolled in this study reported immediate postoperative relief while regaining on average nearly normal to

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normal motor function, and the results persisted through a mean follow-up period of 47.36 months.

*Deep Brain Stimulation (DBS)* – A three-part system, DBS involves the placement of an electrode inside the brain, an extension wire, and a stimulator that produces electrical signals which are sent to the electrode through the extension wire. Its action is similar to that of the stereotactic ventro-oral thalamotomy by causing a functional blockade over the targeted area.

In the treatment of FTSD, studies in South Korea and Japan have targeted the nucleus ventrooralis and the ventralis intermedius nucleus of the thalamus through the application of DBS when treating patients suffering from writer’s cramp, another type of FTSD, and the results have been equivalent to those seen when performing a stereotactic ventro-oral thalamotomy. However, at the time of the writing of this paper, no published study has confirmed the effects of DBS as an effective treatment for musicians diagnosed with FTSD, although it appears to be likely that equally favorable results could be expected in these musicians.

*Rehabilitation and Retraining* – These therapies act under the basis that the loss of voluntary movement control caused by FTSD can be reverted through context-specific, intensive training-based remediation. Numerous studies conducted with musicians

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suffering from FTSD have identified major symptom improvement, including reversed somatosensory map distortions after undergoing R&R therapies.\textsuperscript{270}

Although slightly different approaches are applied in each R&R therapy, they all share some basic paradigms. First, biomechanically inefficient postures and movements when performing on the instrument are observed and substituted by more effective mechanisms that are sustainable in the long term. Thereafter, assimilation and internalization of the new movement patterns are consolidated through slow practice with gradual speed increases applied only when the FTSD symptoms remain nonexistent.

In addition to the evidence provided by studies conducted on musicians diagnosed with FTSD who underwent R&R therapies, this treatise presented the publicly available testimonies of three prominent concert pianists who successfully recovered from FTSD after undergoing a retraining process that followed stages similar to those previously described. In addition, the author included his insights after undergoing a retraining process to recover from FTSD based on the fundamentals of the Taubman Method (TM), a technical approach that provides a step by step instruction to acquire a coordinated and highly efficient use of the body’s physiological resources when performing on the piano.

With the presentation of this information, it is the author’s intent that pianists and other instrumentalists diagnosed with FTSD can comprehensively understand this movement disorder, its implications, its effects and mechanisms of action, and the possible roads to recovery.

\textsuperscript{270} Jabusch and Altenmüller, “Focal Dystonia in Musicians: From Phenomenology to Therapy,” 211.


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APPENDIX A

RECITAL PROGRAMS

UNIVERSITY OF SOUTH CAROLINA
School of Music

presents
JUAN NICOLAS MORALES, piano

in
GRADUATE RECITAL

Wednesday, December 5, 2018
6:00 PM • Recital Hall

Sonata in B-Flat Major, D.960
  Molto moderato
  Andante sostenuto
  Allegro vivace con delicatezza
  Allegro ma non troppo

Franz Schubert (1797-1828)

Après une lecture du Dante: Fantasia quasi Sonata

Franz Liszt (1811-1886)

Mr. Morales is a student of Phillip Bush.
This recital is presented in partial fulfillment of the requirements for the Doctor of Musical Arts degree in Piano Pedagogy.
presents

JUAN NICOLAS MORALES, piano

in

DOCTORAL LECTURE RECITAL

A Brief Journey through the Left-Hand Repertoire

Wednesday, April 25, 2018
6:00 PM • Recital Hall

Prelude & Nocturne for the Left Hand, Opus 9  Aleksandr Scriabin  
(1872-1915)

Etude Opus 10, No. 6 in E-Flat Minor  Frederic Chopin  
(1810-1849)

Studies on Chopin’s Etudes, No. 13  Leopold Godowsky  
after Chopin’s Opus 10, No. 6  (1870-1938)

Etude for the Left Hand, Opus. 36  Felix Blumenfeld  
(1863-1931)

Piano Concerto for the Left Hand  Maurice Ravel  
(1875-1937)

Joo Yeon Park, piano

Mr. Morales is a student of Phillip Bush.  
This recital is presented in partial fulfillment of the requirements  
for the Doctor of Musical Arts degree in Piano Pedagogy.