Spasmodic Dysphonia:

The Disorder and the Mystery

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Abstract

This paper will attempt to give a comprehensive understanding of the nature of spasmodic dysphnia (SD) in all of its forms, including abductor SD, adductor SD, and mixed SD. It will also examine its parent disorders, dystonia and focal dystonia, and discuss possible causes of the disorder as well as its incidence. This has caused quite a big debate among specialists as to whether the disorder is truly neurological, or merely psychogenic, and this paper will evaluate both arguments in order to attempt to reach a conclusion. This paper will also explore various symptoms of the disorder in terms of vocal physiology and how SD is recognized and diagnosed by specialists. Finally, this paper will present several means of treatment for SD and assess the effectiveness of each treatment, in addition to the prognosis of those living with the condition that do not get treated. This discussion will include an examination of the effectiveness of invasive treatments, as well as a look into the role of speech pathologists and traditional speech therapy.

Spasmodic Dyphonia:

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Carmen is 40 years old. Although she is extremely bright, her speech is often very hard to understand. Her friends worry about her because occasionally, when she attempts to speak, it sounds as though she is being choked. Other times, her voice sounds extremely breathy and barely audible. Carmen's description is consistent with someone who suffers from the vocal disorder known as "spasmodic dysphonia", specifically mixed (adductor and abductor) spasmodic dysphonia (SD). This condition is one of the more controversial vocal disorders that exists, in that it's cause is not known with absolute certainty. A great deal of speculation exists, but the true origin still remains a mystery, and there is much debate among speech and language specialists about whether the disorder is truly neurological, or psychogenic. As such, many different treatment options exist as well. This paper will discuss the various theories held by experts about spasmodic dysphonia in an attempt to gain a better understanding of the nature of the disorder, as well as the possible courses of action to take when one is identified as having it.

What is Spasmodic Dysphonia?

In order to gain a better understanding of the nature of spasmodic dysphonia, one must first begin by examining the anatomy of vocal production. According to the Washington Voice Consortium (2003), there are three subsystems of the vocal mechanism. Those subcategories are the air pressure system, which includes the diaphragm and lungs, the vibratory system, which includes the larynx, and the resonating

system, which includes the pharynx and nasal passages. Air is first moved from the lungs into the vocal tract. Simultaneously, the arytenoid cartilages, which are connected to the vocal ligaments, are brought together by the lateral cricoarytenoid and inter arytenoid muscles, resulting in the vocal folds being brought together at the midline. As air moves past the vocal folds, they are forced open and closed at an extremely fast rate due to the Bernoulli Effect, causing the air to begin to vibrate. This now vibrating column of air then moves into the pharynx, and the nasal and oral cavities, where these vibrations are amplified into discernable speech. For people suffering from spasmodic dysphonia, however, the muscles in the vibratory system are affected; the muscles do not contract or relax in the way that most people's would during speech, making the everyday action of speaking that most people take for granted enormously difficult and exasperating.

According to the National Spasmodic Dysphonia Association (2013), one of the most widely accepted views about the condition is that it is a subset of a class of neurological disorders called dystonias. A dystonia "is a movement disorder that causes muscles to contract and spasm involuntarily." (NSDA, 2013) Some dystonias are more generalized, in that they affect the entire body. Other dystonias are considered to be "focal." This type of disorder affects the muscles in localized areas on the body. The most common focal dystonia, for example, is called cervical dystonia, in which the neck twists and contorts involuntarily. In one form of focal dystonia, the lateral cricoarytenoid and inter arytenoid muscles in the larynx are affected, specifically. When these muscles spasm, the voice can either be choked off, or lowered to a breathy whisper. This specific type of movement disorder is what is known as spasmodic dysphonia.

There are three subdivisions of spasmodic dysphonia: adductor SD (ADSD), abductor SD (ABSD), and mixed SD. The first classification, ADSD, is also the most common. In this type of SD, spasms in the laryngeal muscles cause the arytenoid cartilages to "overlap or overadduct." (Kieschnick & Powell, 1996) Symptoms of this specific form of SD include the voice being cut off mid-speech, or, often times, a strained or choked sound to one's voice. This vocal quality is often accompanied with various tremors and tics – the person looks like they are physically straining to get the words out, and this can cause major feelings of frustration and disheartenment. The second manifestation, ABSD, may present itself in a person whose voice suddenly drops to a whisper mid-sentence, or whose voice is intermittently soft and breathy. This characteristic is due to the fact that, in this type of SD, spasms in the muscles cause the arytenoid cartilages not to meet and the vocal folds not to come together at the midline, as is necessary for a full speaking tone. The third type of SD is mixed, in which a person encompasses the characteristics of both of the other types of SD, sometimes having episodes of vocal fold over-adduction, and other times having episodes of vocal fold over-abduction. Obviously, all forms of this condition can be quite debilitating, and can take away or severely limit one of the most basic facets of human life: speech.

One of the most frustrating elements of the condition, besides the obvious symptoms, is the fact that, although some scientists have traced symptoms of the disorder to dystonic movement in the laryngeal muscles, there is still no concrete evidence about what specifically causes the dystonia in the first place. According to Neychev, Fan, Mitev, Hess, & Jinnah (2008), the "traditional views [have placed] responsibility for dystonia with dysfunction of basal ganglia circuits." (2499) The basal ganglia are a

collection of nuclei in the brain thought to play a significant role in controlling posture and voluntary movement. (Mink, 1996) In fact, several studies have demonstrated the correlation between basal ganglia damage and various movement disorders, such as Parkinson's disease, which is the most common movement disorder, closely followed by Essential Tremor and Dystonia. However, recent tests utilizing functional imaging studies have revealed that abnormal activity in the cerebellum can also play a role in dystonia. The cerebellum, located in the back of the brain, also plays a role in coordinating balance and muscle coordination for voluntary movements. (Hahn, 2013) As such, a more recent focus has not been to examine one structure without the other, but rather the two together as well as how they interact with each other to generate movement. More current research certainly suggests that both play a major role in the development of dystonia and, consequently, spasmodic dysphonia, but more research is needed to assert that claim with certainty. (Neychev et al, 2008) Scientists are a lot closer to uncovering the secrets of spasmodic dysphonia, but as of now the mystery still continues unresolved.

One of the most baffling facets of SD is that it is task specific. In other words, symptoms of the disorder only present themselves when a person is speaking. When a person laughs, sings, swallows, or breathes, the larynx behaves normally, with no unusual adduction or abduction of the muscles. This fact has led some to believe that the condition is not so much neurological in nature, but rather psychogenic, meaning that the physical symptoms are purely the result of mental and/or emotional stress. According to Sapir (1995), many cases of SD present with no obvious signs of a neurological disease. Additionally, there was evidence of depression and/or anxiety preceding or coinciding

with the SD in many patients. Sapir, however, argues that not all patients were shown to have any mental stress or emotional trauma, and, in fact, the distress in those that did have it was secondary to or even the result of the SD, rather than its cause. As such, it is unreasonable to make the assumption that all people who suffer from SD are experiencing the symptoms solely as a conversion reaction to some other harrowing event. That is not to say that the condition cannot be psychogenic in some patients. In fact, it is not unheard of for the brain to cope with an enormous amount of emotional strain by manifesting the stress physically. In rare cases, this manifestation can even mimic a movement disorder, such as dystonia, ataxia, dysarthria, or spasmodic dysphonia. Authors and specialists have thereby separated the condition into neurologic SD and psychogenic SD (PSD). Although the question of why SD is task specific still remains unknown, it seems that both sides of the debate are correct in some respect: SD can be either a neurological or a psychogenic disease, depending on each patient. But an exact cause for either type still eludes experts to this day.

How Common is the Disorder and How is it Diagnosed?

Spasmodic Dysphonia is estimated to affect about 50,000 people in North America. (NSDA, 2013) However, this number is purely an estimation. Due to the unclear nature of the condition, SD is commonly misdiagnosed and/or undiagnosed. As such, this representation of incidence may not be entirely accurate. Studies have shown, however, that the vast majority of SD cases present in patients between 30 and 50 years of age. Additionally, more women appear to be afflicted than men, although, once again, scientists are not sure why. (Castrogiovanni, 2008)

As previously stated, diagnosis of spasmodic disphonia is especially tricky, because people with SD often exhibit symptoms similar to those of other vocal diseases. Typically, onset of SD is gradual for a period of about 18 months, with no structural abnormalities in the larynx as an indicator of the disorder. After this amount of time, the disorder appears to be chronic and maintain a consistent severity unless otherwise treated. Again, an explanation for the course of this condition is nonexistent at this time. (NSDA, 2013) Typically, diagnosis of SD requires a careful examination by a team of specialists. This team includes "an otolaryngologist…a speech-language pathologist…and a neurologist. The otolaryngologist examines the vocal folds for other possible causes of the voice disorder. The speech-language pathologist evaluates the types of voice symptoms to see if they are characteristic of spasmodic dysphonia or other voice disorders and voice quality. The neurologist evaluates the patient for signs of other muscle movement disorders." (NIDCD, 2010) Because of the uncertainty of SD, this extreme level of evaluation and cooperation is the only way to accurately identify a vocal disorder as spasmodic dysphonia and not anything else.

What Are the Courses of Action?

Unfortunately, since no explanation for spasmodic dysphonia can be found, a cure for it does not exist either. Various treatment options do exist, each one with its own level of success. However, these treatment options only serve to reduce the effects of spasmodic dysphonia. A person suffering from the condition can be helped to become a functional speaker. However, the disorder can never be eradicated from him or her completely. As such, the communication disease specialist, in this case a speech-language pathologist (SLP), is usually not the only or most effectual specialist to play a

role in the lives of people with spasmodic dysphonia. Initially, the SLP, as part of the aforementioned team of specialists, evaluates the patient to determine whether or not the symptoms are truly indicative of SD. Once the patient is diagnosed, he or she can discuss various treatment options with his or her speech pathologist. One treatment option is traditional voice therapy with the SLP. This, however, because of the neurological nature of the disease, has only been somewhat effective with extremely mild cases. In many of these cases, vocal rehabilitation and vocal psychotherapy were used to teach breathing exercises, help establish optimum pitch, and improve the patient's vocal image, which can affect any of their vocal qualities. In one study, the SLPs even evaluated the effectiveness of inspiratory speech versus expiratory speech in overcoming the effects of SD. This method was shown to increase functionality of the speaking voice in some of the mildest cases. (Kieschnick et al., 1996) SLPs can also discuss the use of augmentative and alternative communication devices, such as voice amplifiers for patients with ABSD, and devices that translate text into synthetic speech for patients with ADSD, with their patients. (NDSA, 2013)

Interaction with the SLP has really only been shown as an effective treatment option for those with extremely mild SD. For more severe cases, a more invasive approach is required if the patient is to have any hope of improvement. One of these more invasive options is known as a Recurrent Laryngeal Nerve Section (RLN Section). The RLN section is a surgical procedure that consists of the "removal of a small segment of one side of the RLN. The goal…is the reduction of glottic tightness…thereby allowing for easier vocal production and intelligibility." (Kieschnick et al., 1996, 15) This procedure has been shown as effective in minimizing or even eliminating the

symptoms of SD for an extended period of time in a handful of patients. In some cases, it even resulted in improved phonation during postoperative recurrences of the disorder. As stated earlier, though, complete removal of SD is impossible at this stage in our understanding of it, and so the symptoms do tend to return after surgery, although potentially not for an extended period of time, as is the ambition of the procedure. For that reason, as well as others, this treatment option is frequently met with opposition. Other reasons that RLN sections are not very popular include the fact that the surgery is irreversible. The section of nerve will permanently be gone if surgically removed. Additionally, surgical procedures always present some danger of unwanted side effects. This procedure, in particular, has been shown to cause breathiness, and even aspiration in some patients. As such, this method is more commonly objected to than endorsed.

The current primary treatment for SD symptoms is Botulinum Toxin (BOTOX) injections. For many patients, this treatment option has been extremely effective in temporarily treating the overadduction of the laryngeal muscles to alleviate strain in the speaking voice. In one study, 80% of patients reported improved quality and less effort in their speaking voice within 24-28 hours of the BOTOX injection. (Kieschnick et al., 1996, 16) At this time, however, there is not enough research to determine the long-term effects of the treatment. However, one observable disadvantage to this treatment is the short-lived relief that it offers. Patients usually must return for injections every 3-6 months. However, at this time, this course of treatment does appear to be the most reliable and safest method for temporarily treating the symptoms of SD.

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