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Spasmodic Dysphonia is a Neurological Disorder
Current Evidence and References

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Many studies over the last three decades have demonstrated that Spasmodic Dysphonia is a neurological disorder. There is a high rate of other forms of focal dysphonia in patients with the disorder (Aminoff, Dedo, & Izdebski, 1978; Blitzer, Lovelace, Brin, Fahn, & Fink, 1985; Brin, Blitzer, & Stewart, 1998; Schaefer, 1983), many patients have a high incidence of other neurological disorders (Aminoff et al., 1978; Aronson, Brown, Litin, & Pearson, 1968, 1968; Finitzo et al., 1987; Rosenfield et al., 1990; Roth, Glaze, Goding, & David, 1996), abnormalities in brainstem reflexes and other aspects of neurological function have been a consistent finding in spasmodic dysphonia over many different laboratories world wide (Cohen et al., 1989; Deleyiannis, Gillespie, Bielamowicz, Yamashita, & Ludlow, 1999; Devous et al., 1990; Finitzo-Hieber, Freeman, Gerling, Dobson, & Schaefer, 1981; Hall & Jerger, 1976; Hirano et al., 2001; Ludlow & Connor, 1987; Ludlow, Schulz, Yamashita, & Deleyiannis, 1995; Schaefer et al., 1985) and genetic studies have found an association between Spasmodic Dysphonia and other forms of dystonia (Blitzer & Brin, 1991; Brin et al., 1998).

Others have suggested that Spasmodic Dysphonia is not a neurological disorder as some patients' symptoms can be reversed with voice therapy (Chevrie-Muller, Arabia-Guidet, & Pfauwadel, 1987). This is most likely a confusion in the diagnosis of Spasmodic Dysphonia and not distinguishing it from other forms of hyperfunctional voice such as a disorder referred to as muscular tension dysphonia which is often misdiagnosed as spasmodic dysphonia (Roy, Ford, & Bless, 1996).

The major difference is that Spasmodic Dysphonia, as the name suggests, involves involuntary transient muscle spasms which intermittently interfere with voice production producing involuntary voice breaks either within vowels in the adductor form of the disorder (Bielamowicz & Ludlow, 2000; Nash & Ludlow, 1996) or during voiceless consonants in the rarer abductor form of the disorder (Cyrus, Bielamowicz, Evans, & Ludlow, 2001). Patients with other hyperfunctional voice disorders do not have intermittent voice disruptions but rather a consistent strained voice production (Morrison, 1997; Morrison, Nichol, & Rammage, 1986; Morrison & Rammage, 1993; Morrison, Rammage, Belisle, Pullan, & Nichol, 1983; Roy, Gouse, Mauszycki, Merrill, & Smith, 2005; Sapienza, Walton, & Murry, 2000).

Therapeutic evidence has demonstrated that patients with muscular tension dysphonia respond to voice therapy alone (Roy, Ford, & Bless, 1996) while those with Spasmodic Dysphonia do not (Roy et al., 1996) except when therapy is provided along with botulinum toxin (Murry & Woodson, 1995). The assertion that Spasmodic Dysphonia is not a neurological disorder because it can be reversed by voice therapy is most likely due to a confusion in the initial diagnosis of the disorder.

No evidence in the literature to date has shown that persons with symptoms commensurate with Spasmodic Dysphonia will have a long term response to voice therapy alone. On the other hand, ample evidence is available in the literature demonstrating that patients with muscular tension dysphonia respond well to voice therapy (Roy, Bless, Heisey, & Ford, 1997; Roy et al., 1996).

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