APPEAL NO. 000157

On December 14, 1999, a contested case hearing (CCH) was held. The CCH was held under the provisions of the Texas Workers' Compensation Act, TEX. LAB. CODE ANN. § 401.001 *et seq.* (1989 Act). The issue at the CCH was whether appellant's (claimant) compensable injury is a producing cause of claimant's spasmodic dysphonia. Claimant requests that the hearing officer's decision that the compensable injury is not a producing cause of claimant's spasmodic dysphonia be reversed and that a decision be rendered in his favor. Respondent (carrier) responds that the hearing officer's decision is supported by the evidence and requests that the hearing officer's decision be affirmed. Carrier also contends that claimant's request for review was not timely filed. The hearing officer's decision was distributed on January 5, 2000; the claimant received the decision on January 10, 2000; and claimant's request for review was filed on January 25, 2000, within the 15-day period provided for in Section 410.202(a). Claimant's request for review was timely filed.

DECISION

Affirmed as modified herein.

Claimant testified that he was employed (employer) for eight years, that in June 1996 he became hoarse and went to Dr. M, that his symptoms were made worse by talking on the telephone all day when doing his telemarketing job, and that his left leg drags and he does not have the full use of his left arm because of polio he had when he was two years old. The parties stipulated that on _____, claimant was the employee of employer and that he sustained a vocal cord strain that was accepted by carrier.

According to a medical article in evidence, spasmodic dysphonia is a neurological condition affecting the vocal muscles of the larynx; its symptoms may be aggravated by continual talking on the telephone; in most cases its cause is not known; in some cases it appears that its onset may be triggered by trauma to the vocal cords; and, regardless of the cause, its symptoms are thought to be due to abnormal functioning in the basal ganglia area of the brain.

Dr. M, whose letterhead indicates he is in family practice, wrote in November 1998 that in the fall of 1996 claimant presented to him with severe laryngitis and he referred claimant to Dr. P, an otolaryngologist, who made a diagnosis of spastic dysphonia. Dr. P wrote in October 1996 that claimant's voice quality sounded like spastic dysphonia. Dr. P wrote in December 1996 that claimant has a significant vocal cord abnormality that requires speech therapy and that his voice abnormality is related to excessive voice use and appears to be aggravated by his work as a telemarketer. Dr. P also wrote in December 1996 that claimant's initial examination demonstrated chronic laryngitis with early polypoid formation, that he could not rule out spastic dysphonia, and that it was his impression that claimant's work was a contributing factor to his dysphonia. SS, the speech therapist Dr. P referred claimant to, wrote in December 1996 that claimant presented to her with a medical

diagnosis of polypoid degeneration and ventricular dysphonia and that he also has vocal strain and laryngeal spasm suggestive of spasmodic dysphonia. Dr. P wrote in January 1997 that spastic dysphonia is unrelated to voice use and is an idiopathic neurologic laryngeal abnormality. SS wrote in December 1998 that, while the exact cause of claimant's voice problem cannot be determined, the constant phone use would increase the severity of the problem and his job would be a factor in the severity of the problem.

Dr. S, an otolaryngologist, wrote in October 1997 that he agreed with a diagnosis of adductor spasmodic dysphonia and recommended Botox injections, which claimant underwent.

At carrier's request, Dr. A, an otolaryngologist, examined claimant in March 1999 and she wrote that she agrees with a diagnosis of spasmodic dysphonia; that the cause of claimant's spasmodic dysphonia has not been determined; that people in many types of occupations and unemployed people are afflicted with spasmodic dysphonia; that it cannot be verified that claimant's spasmodic dysphonia was caused by his employment; that claimant's current condition appears to be a continuation of a problem which is three years old and is in a continuum of a disease state and treatment; that while extensive voice use will demonstrate spasmodic dysphonia, she does not feel that there is any support to say that talking actually causes claimant's or anyone's spasmodic dysphonia; that while it would be reasonable to say that speaking demonstrates claimant's disease process, she does not feel that it necessarily exacerbated it; and that she failed to see that the spasmodic dysphonia was actually caused by claimant's employment. Dr. A noted that claimant's Botox injections were done through his group health insurance, and health insurance claim forms indicate that that was the case.

Dr. C, whose specialty is physical medicine and rehabilitation, examined claimant in August 1999 and wrote that claimant's intense speech as a telemarketer aggravated his preexisting polio condition, that claimant is experiencing a post-polio syndrome as related to his speech-producing mechanisms, and with the manifestation of the post-polio syndrome by the intense speech required in telemarketing, claimant would qualify as having a work injury and not an ordinary disease of life. In a deposition on written questions, Dr. C stated that he found claimant to have symptoms compatible with spastic dysphonia with an underlying causative factor most likely secondary to post-polio syndrome, and that spastic dysphonia may be aggravated by use of the voice.

In a deposition on written questions, Dr. P answered that spastic dysphonia is an idiopathic neurologic laryngeal voice disorder, that the cause of spastic dysphonia is unknown, that it is still his opinion that the diagnosis of spastic dysphonia is unrelated to voice use, that initially claimant had mild chronic laryngitis that resolved by February 1997, that spastic dysphonia is manifested only when speaking, that he concurs with Dr. A's conclusion that spasmodic dysphonia is not aggravated by voice use but rather is demonstrated by voice use, and that spasmodic dysphonia can happen to anyone regardless of occupation or voice use.

Dr. M wrote in May 1999 that there is no evidence that spastic dysphonia is related to voice use.

Claimant appeals the hearing officer's finding that the injury claimant sustained while working for employer is not a producing cause of his spasmodic dysphonia and the hearing officer's conclusion and decision that the compensable injury is not a producing cause of claimant's spasmodic dysphonia. Claimant contends that the hearing officer's decision is against the great weight and preponderance of the evidence and that carrier accepted symptoms of spasmodic dysphonia, thus the compensable injury is a producing cause of that condition. There is conflicting evidence in this case regarding the disputed issue of whether the compensable injury is a producing cause of claimant's spasmodic dysphonia. The 1989 Act makes the hearing officer the sole judge of the weight and credibility of the evidence. Section 410.165(a). As the trier of fact, the hearing officer resolves conflicts in the evidence and determines what facts have been established from the evidence presented. We conclude that the appealed finding, conclusion, and decision are supported by sufficient evidence and that they are not so contrary to the overwhelming weight of the evidence as to be clearly wrong and unjust. Cain v. Bain, 709 S.W.2d 175 (Tex. 1986).

We modify Stipulation 1.A. to reflect that (employer) was the employer, as was stipulated to by the parties.

As modified herein, the hearing officer's decision and order are affirmed.

Robert W. Potts
Appeals Judge

CONCUR:

Elaine M. Chaney
Appeals Judge

Dorian E. Ramirez
Appeals Judge