Chapter 30
V2 Rhizotomy

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ABSTRACT

Trigeminal neuralgia is a facial pain syndrome characterized by excruciating, paroxysmal, electric shock-like pain attacks in the sensory distribution of the trigeminal nerve. Medical management remains the first line of treatment. When this fails, surgical management needs to be considered. Percutaneous interventional procedures such as glycerol rhizotomy, radiofrequency (RF) thermocoagulation, and balloon compression of the trigeminal ganglion and its branches are some of the most commonly used procedures as they avoid exposure to general anesthesia, provide successful short-term results, and are available to people with significant co-morbidities. Of these, RF is the most often used. The V2 and V3 branches of the trigeminal nerve are most commonly affected, and are thus the most frequent targets for RF interventions. These procedures may be performed using conventional fluoroscopic, ultrasound, or CT-guided imaging, including combined flat-panel CT and fluoroscopy. This chapter summarizes these common ablation techniques targeting the V2 branch of the trigeminal nerve.

INTRODUCTION

Trigeminal neuralgia (TN) is a severe, neuropathic pain condition affecting the distribution of the trigeminal nerve. The pain is often characterized as electric and sharp as if the face is being stabbed. Pain is often accompanied by a unilateral grimace, which led to the synonym “tic douloureux.”(Burchiel & Slavin, 2000). Attacks are most common in the second and third trigeminal divisions, on the right side of the face (Harness & Chase, 1990) . The attacks usually recur with the same intensity and distribution.

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A brief refractory period of relief typically follows that lasts seconds to hours. This pain can be triggered by stimulating a specific facial area (e.g by light touch or tooth brushing) or can occur spontaneously (Rasmussen, 1991) . The etiology of TN is not fully understood. Dysfunction of the myelin sheath has been the focus of much investigation (Dubner, Sharav, Gracely, & Price, 1987).

Clinical examination, imaging studies, and laboratory tests are usually unremarkable in classic TN (Rasmussen, 1990a). When the signs and symptoms of TN are secondary to another disease process it is called ''symptomatic TN''. The basic pathophysiology of TN remains largely unknown.(Bowsher, 1997; Canavero, Bonicalzi, & Pagni, 1995; Rasmussen, 1990b). Many TN patients suffer pain attacks for months or years before the condition is appropriately diagnosed. Over time, these severe pain attacks have a devastating impact on patients' lives (Rasmussen, 1991). Timely and accurate diagnosis of TN speeds treatment with medication and surgical interventions which can help to limit this negative impact (Tartaro, Stroffolini, & Lepore, 1979).

Aretaeus of Cappadocia is credited with the first clinical description of TN. At the end of the first century, he discovered *heterocrania,* which he described as spasm causing "distortion of the countenance" (Cappadocia.), 1856; Mosberg, 1960). In 1677, the noted American physician and philosopher, John Locke, identified the major clinical features of TN in his work Countess of Northumberland (Mosberg, 1960). It was the first time a physician found that this pain was not caused by dental pathology. In 1773, John Fothergill, an English physician, clearly established the disorder as a discrete syndrome (Eboli, Stone, Aydin, & Slavin, 2009; Pearce, 2003). In 1912, Sir William Osler, one of the four founding professors of Johns Hopkins Hospital, gave the most complete medical description of trigeminal neuralgia for his colleagues, giving new understanding of the disease's pathophysiology (Casey & Jannetta, 2010). Dr. Osler found that specific trigger zones anywhere within the trigeminal distribution, could initiate these severe attacks and tics in the face (Dubner et al., 1987). He found this to be the reason patients avoided touching, washing, and shaving their faces. He also identified intraoral trigger zones, which were stimulated by chewing. This was important because most other facial pains were treated with massage or by applying heat and cold (Türp & Gobetti, 1996).

The overall incidence rate is estimated at approximately three to five cases per year per 100,000 persons. Patients are more likely to be female and older than 50-60 when the first attacks begin (Massager et al., 2007). The age and sex distribution are interesting as most other common chronic headache and facial pain disorders occur at an earlier age (Fraioli, Lisciani, & Fraioli, 2014; Maarbjerg, Di Stefano, Bendtsen, & Cruccu, 2017). The paroxysmal pain usually worsens over time. While there are familial examples, TN does not appear to be increased in any particular ethnic group, geographic region, or climate (Duff, Spinner, Lindor, Dodick, & Atkinson, 1999; Smyth, Greenough, & Stommel, 2003; Maarbjerg et al., 2017).

PATHOPHYSIOLOGY

Compression of the trigeminal nerve root by benign tumors and vascular anomalies can produce symptoms, which are indistinguishable from classic TN. This implies that injury to the nerve root is an important factor in the evolution of the disease (Devor, Amir, & Rappaport, 2002). Early research showed that surgical decompression of the nerve root from vascular anomalies often effectively alleviated the symptoms (Barker, Jannetta, Bissonette, Larkins, & Jho, 1996; Haines, Jannetta, & Zorub, 1980). Damage to the sensory neurons of the trigeminal nerve ganglion causes cascade of changes in the brainstem and