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Advances in microvascular decompression for hemifacial spasm

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Abstract

Primary hemifacial spasm (HFS) is a disorder that causes frequent involuntary contractions in the muscles on one side of the face, due to a blood vessel compressing the nerve at its root exit zone (REZ) from the brainstem. Numerous prospective and retrospective case series have confirmed the efficacy of microvascular decompression (MVD) of the facial nerve in patients with HFS. However, while MVD is effective, there are still significant postoperative complications. In this paper, recent technological advances related to MVD (such as lateral spread response, brainstem auditory evokes potential, three dimensional time of flight magnetic resonance angiography, intraoperative neuroendoscopy) are reviewed for the purposes of improving MVD treatment efficacy and reducing postoperative complications.

Keywords: Hemifacial spasm (HFS); Microvascular decompression (MVD); Lateral spread response; Three dimensional time of flight magnetic resonance angiography; Neuroendoscopy

Hemifacial spasm (HFS) is a disorder characterized by involuntary contractions of facial muscles, usually on one side of the face that can be intermittent, rhythmic or sustained. The contraction often starts from orbicularis oculi and spreads to multiple facial expression muscles, and can be triggered or exacerbated by emotional excitability, stress, fatigue or excessive speech. Overseas epidemiology surveys suggest a prevalence of 0.78/100 000 (Auger and Whisnant, 1990), more often seen in women (male:female = 1:2) and rare in children (Titlić et al., 2006). Most cases involve one side of the face, often on left (left:right = 3:2), and bilateral involvement is seen in less than 1% of cases. Primary HFS usually results from microvascular compression of the facial nerve at the root exit zone (REZ). Long term pressure and irritation from the offending vessel cause local demyelination and "shortening" between nerve fibers, leading to ectopic bioelectric transmission (Zhu et al., 2012). About 1–2% of HFS cases are secondary to space occupying lesions in the cerebellopontine angle or posterior fossa. Familial cases have been reported, but in general HFS is not considered hereditary (Lagalla et al., 2010). Many treatments for HFS have been reported, including pharmacological agents, botulinum toxin injection, facial nerve blockage, physical therapy, radiofrequency ablation, acupuncture, as well as facial nerve combing and microvascular decompression (MVD). In 1959, Gardne first reported a case of trigeminal neuralgia caused by vascular compression (Gardner and Miklos, 1959). In 1966, based on Gardne's findings, Jannetta suggested a theory of neural circuitry shortening as a result of demyelination of facial nerve root from vascular compression that could be the cause for more than 95% of HFS cases, and started treating HFS with MVD. Following Jannetta, experiences from others confirmed the efficacy of MVD in treating HFS, which has now become the first choice of treatment for HFS (Jannetta et al., 1977). A review that included 22 reports and 5685 cases of MVD with an average follow up of 2.9 years showed rates of complete symptom resolution as high as 91.1% (Miller and Miller,

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2012). Chung et al. reported 1169 cases of MVD for HFS with an average follow up of 28.3 months and overall effective rate of 95% (Chung et al., 2001). At this time, MVD enjoys well established techniques and confirmed efficacy, but certain postoperative complications remain, including hearing loss (0.8–16.2%) and facial paresis (1.2–16.2%) (Jannetta et al., 1977). Many continue to work to improve MVD outcomes and reduce complications. Currently most efforts aim at application of new technologies, such as intraoperative electrophysiological monitoring, neuroimaging three dimensional reconstruction and neuroendoscopy.

1. Intraoperative neuroelectrophysiological monitoring

Lateral spread response (LSR), also known as abnormal muscle response, is an abnormal neuromuscular response recorded in HFS patients from muscles supplied by a facial nerve branch different than the one being stimulate (Thirumala et al., 2011). Its mechanisms involve bidirectional conduction of neural impulses. When the mandibular branch of the facial nerve is stimulated in patients with HFS, neural signals can be transmitted not only to the orbicularis oris, causing its contraction, but also in a retrograde fashion toward the facial nucleus in the brainstem, where abnormalities along the proximal facial nerve or inside the facial nucleus can generate abnormal impulses that can return along a different facial nerve branch (zygomatic branch for example) and arrive at orbicularis oculi, resulting a signal with a latency of 8–10 ms (delayed by 4–5 ms compared to the primary orbicularis oris response). This abnormal muscle response indicates abnormal cross connection between different branches of the facial nerve, which is currently believed to be the etiological basis for HFS. Intraoperatively, disappearance of LSR following separation of the offending vessel can be used to confirm the offending vessel and predict postoperative results. Kong et al. (2007) performed MVD in 300 cases of HFS and recorded LSR in 263 cases (87.7%), of which LSR disappeared in 230 cases (87.4%) and persisted in 33 cases (12.5%). At one year follow up, those in whom LSR disappeared during surgery showed significantly better results than those in whom LSR persisted. Neves et al. (2009) studied 32 cases of HFS and concluded that use of intraoperative LRS monitoring could not only predict short term outcomes but also impact long term treatment results. Reports from Kang et al. (2012) and Kim et al. (2010) also support the value of LSR monitoring in MVD. Thirumala et al. (2011) performed electrophysiological monitoring in 293 cases of HFS that underwent surgical treatment and recorded LSR in 259 of these cases (87.7%), of which LSR disappeared following decompression in 207 cases (Group 1) but persisted in 52 cases (Group 2). Group 1 showed a rate of symptom resolution of 94.7% within 24 h following surgery and 93.3% at discharge from hospital, and the rate was 67.3% and 76.9% respectively in Group 2 ($P < 0.0001$). At 54.5 months following surgery, the rate of symptom resolution was 93.3% in Group 1 and 94.4% in Group 2 ($P = 1.000$). They therefore concluded that LSR monitoring might predict treatment effects immediately following surgery but not over long term. Joo et al. (2008) also studied the value of LSR in MVD and questioned its value in indicting long term treatment outcomes. In China, extensive studies seem to confirm a positive role of LSR monitoring in MVD (Gao and Zhao, 2013a; Liu et al., 2010; Ying et al., 2011). The authors have used LSR monitoring in relevant surgeries and believe that it is to a certain extent helpful in confirming offending vessels and predicting treatment effects (Fig. 1).

Brainstem auditory evokes potential (BAEP), nerve action potential (NAP) and electrocochleography (EcochG) are the three main techniques for intraoperative monitoring of the auditory nerve. BAEP is non-invasive and capable of assessing function of auditory pathways from the periphery up to the brainstem, and therefore is probably the most commonly used. BAEP is short latency auditory responses representing neuroelectric activities up to the brainstem level in response to acoustic stimulation. It has defined temporal relationship to the stimulus and is not affected by sleep or anesthesia. In an MVD procedure, the surgeon operates in the cerebellopontine angle area where the facial and auditory nerves are located extremely closely to each other and the auditory nerve and neighboring vessels can be impacted by surgical maneuvers (Dou et al., 2009). Rates of hearing impairment at an early time following surgery as high as 14–18% have been reported Jannetta and Kassam (1999), Murai et al. (1991). Usage of intraoperative electrophysiological monitoring and improvement in microscopic ear surgery have greatly reduced hearing

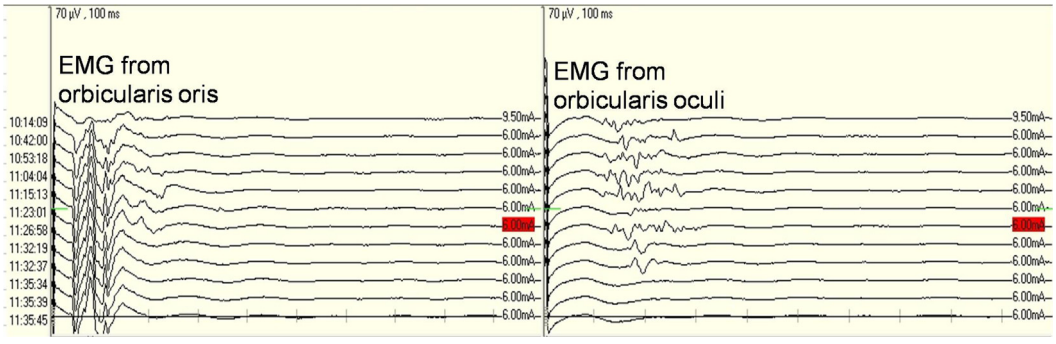

Fig. 1. Triggered EMG from orbicularis oris (left) and orbicularis oculi (right). Note the disappearance of EMG activities from orbicularis oculi shortly after separation of the offending vessel from the facial nerve (red mark).