



## Sympathetic nerves bridge the cross-transmission in hemifacial spasm

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### ARTICLE INFO

#### Article history:

Received 21 January 2012

Received in revised form 30 March 2012

Accepted 9 April 2012

#### Keywords:

Hemifacial spasm  
Facial nerve  
Sympathetic nerve  
Offending artery  
Cross-transmission

### ABSTRACT

The pathophysiologic basis of hemifacial spasm is abnormal cross-transmission between facial nerve fibers. The author hypothesized that the demyelinated facial nerve fibers were connected with the sympathetic nerve fibers on the offending artery wall, and thus the latter function as a bridge in the cross-transmission circuit. This hypothesis was tested using a rat model of hemifacial spasm. A facial muscle response was recorded while the offending artery wall was electrically stimulated. The nerve fibers on the offending artery wall were blocked with lidocaine, or the superior cervical ganglion, which innervates the offending artery, was resected, and meanwhile the abnormal muscle response was monitored and analyzed. A waveform was recorded from the facial muscle when the offending artery wall was stimulated, named as “Z-L response”. The latency of Z-L response was different from that of abnormal muscle response. When the nerve fibers on the offending artery wall were blocked by lidocaine, the abnormal muscle response disappeared gradually and recovered in 2 h. The abnormal muscle response disappeared permanently after the sympathetic ganglion was resected. Our findings indicate that cross-transmission between the facial nerve fibers is bridged by the nerve fibers on the offending artery wall, probably sympathetic nerve fibers.

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### 1. Introduction

Hemifacial spasm (HFS) is a neuromuscular disorder characterized by frequent, involuntary facial muscle contractions that cause physical discomfort and embarrassment, and impair the life quality. Although there are many different etiologies such as Bell's palsy, facial nerve injury, demyelinating lesions and hereditary [18], it's generally accepted that the major etiology of hemifacial spasm is vascular compression of the seventh cranial nerve (the facial nerve, Fig. 1A) at its root exit zone (REZ) [2,6]. The compression is caused by offending vessels, such as the anterior inferior cerebellar artery, posterior inferior cerebellar artery, and

vertebral artery [2,6,9]. Up till now, the pathogenesis of HFS is unclear yet.

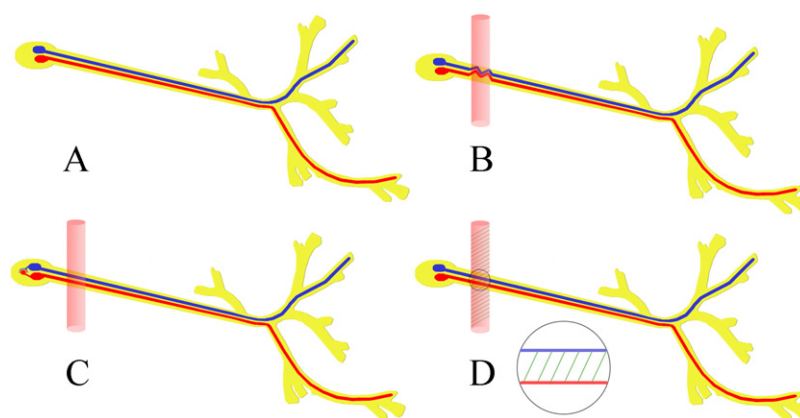
In patients with HFS, electrical stimulation of one branch of the facial nerve on the affected side can elicit a delayed response from the muscles supplied by other branches; this has been called an “abnormal muscle response” (AMR), also known as “lateral spread response” (LSR). The presence of an AMR has been documented only in patients with HFS, so an AMR serves as a “marker” of HFS and is useful for the diagnosis and intraoperative monitoring during microvascular decompression surgery [17]. An AMR suggests that abnormal cross-transmission takes place between facial nerve fibers, and this is believed to be the main pathogenic basis for HFS [13,17,19]. However, the location and style of cross-transmission are still controversial. Two hypotheses have prevailed. One supposes that ephaptic transmission occurs between individual facial nerve fibers at the site of neurovascular compression because compression injures the myelin [16] and brings bare axons close together [4,15]. The other hypothesis states that chronic injury to the facial nerve may make the facial motor nucleus hyperexcitable and cause the opening of dormant synapses, which could cause cross-transmission in the facial motor nucleus [7,8,13,14]. In this paper, the former is referred to as the “ephaptic transmission hypothesis” (Fig. 1B) and the latter as the “hyperexcitable nucleus hypothesis” (Fig. 1C). The advantages and shortcomings of these two hypotheses are discussed later.

**Abbreviations:** AMR, abnormal muscle response; ZLR, Z-L response; ECA, external carotid artery; EMG, electromyogram; HFS, hemifacial spasm; MVD, microvascular decompression; REZ, root exit zone.

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**Fig. 1.** Models of three hypotheses regarding the cross-transmission of hemifacial spasm. (A) The normal status of facial nerve. The blue line represents a facial nerve fiber in the temporal branch. The red line represents a facial nerve fiber in the marginal mandibular branch. (B) Ephaptic transmission hypothesis supposes that ephaptic transmission occurs between individual facial nerve fibers just at the site of neurovascular compression. (C) Hyperexcitable nucleus hypothesis claims that chronic injury to the facial nerve may cause the opening of dormant synapses that would cause cross-transmission in the facial motor nucleus. (D) Sympathetic bridge hypothesis assumes that at the site of neurovascular compression, cross-transmission between the facial nerve fibers is bridged by the sympathetic nerve fibers (the green lines) on the offending artery wall. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

In our clinical practice, we have often found that once the offending vessel is detached from the facial nerve, the AMR disappears immediately, and such patients generally have immediate spasm relief after surgery. This observation is consistent with the literature [11,12,20]. However, neither the ephaptic transmission hypothesis nor the hyperexcitable nucleus hypothesis successfully explains this very common and important clinical phenomenon. It seems inconceivable that hyperexcitability of facial motor neurons would decrease immediately after detaching the offending vessel. Similarly, it is impossible that the histological changes at the sites of compression, such as demyelination, vacuolization of the myelin sheath, and partial degeneration of axons [16], will disappear immediately after decompression. Based on these two hypotheses, it might be expected that most cases of HFS would resolve gradually after MVD surgery. However, this is not the case. The abnormal cross-transmission seems dependent on the close contact between the facial nerve and the offending vessel. Thus, we hypothesized that the offending vessel itself may be part of the cross-transmission circuit, so the circuit is broken as soon as the offending vessel is detached from the facial nerve. Nerve impulses (action potentials) can only be conducted through nerve fibers, and there are abundant sympathetic nerve fibers on the artery wall [3]; thus, we further hypothesized that the sympathetic nerve fibers on the offending artery wall may connect to the demyelinated facial nerves and function as a “bridge” in the cross-transmission of HFS; named as “sympathetic bridge hypothesis” (Fig. 1D).

If the sympathetic bridge hypothesis is true, there follows three deductions: (1) If the sympathetic nerve fiber on the offending artery wall is electrically stimulated, the impulse will be conducted to the facial nerve, and then a facial muscle response can be recorded; (2) If the sympathetic nerve fiber on the offending artery wall is blocked with lidocaine, the cross-transmission circuit will be broken, and then AMR will be eliminated, but the facial nerve conduction remains intact; (3) If the sympathetic ganglion which innervates the offending artery is removed, the cross-transmission circuit will be broken permanently. Following these deductions, the hypothesis was tested in animal experiments.

## 2. Methods

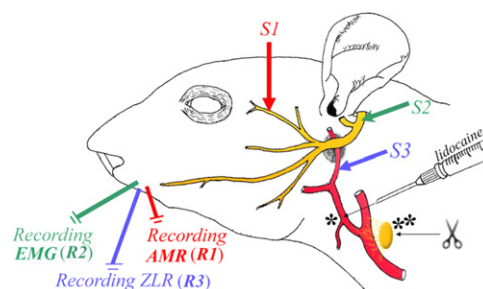
### 2.1. Animal model of HFS

Experiments with animals were performed in accordance with the legal requirements of Xinhua Hospital. Rat models of HFS

were produced under anesthesia with pentobarbital (40 mg/kg, intraperitoneally), following the methods reported by Kuroki and Moller [8]. At the first stage, the extracranial portion of the facial nerve was exposed under a Moller–Wedel surgical microscope; and then the main trunk of the facial nerve was dissected. Afterwards a chromic gut suture was placed in the gap between the facial nerve and the temporal artery, which runs just beneath the facial nerve at a right angle. At the second stage (2 weeks later), the chromic gut suture was removed, and then the temporal artery was transposed with a teflon felt so that it came in close contact with the facial nerve (Fig. 2). Four weeks after the second stage, abnormal muscle response was recorded [8] from the mentalis muscle in response to electrical stimulation of the ipsilateral temporal branch of the facial nerve (S1 and R1, Fig. 2), using an evoked potential system (Medtronic Keypoint 4, Dantec, Denmark) with the stimulation intensity of 3–10 mA  $\times$  0.1 ms. Only those AMR-positive rats were kept for further study. Those failed to induce AMR were rejected.

### 2.2. Test the first deduction

Two groups of rats were included in this experiment. The HFS group ( $n = 10$ ) underwent 2 stages of operation as mentioned above, and were confirmed AMR-positive. The control group of rats



**Fig. 2.** Schematic diagram of the animal experiment, using a rat model of hemifacial spasm established by Moller et al. To record abnormal muscle response (AMR): stimulating the temporal branch of the facial nerve (S1) and recording from the mentalis muscle (R1). To record electromyogram (EMG): stimulating the main trunk of the facial nerve at the stylomastoid foramen (S2) and recording from the mentalis muscle (R2). To record Z-L response (ZLR): stimulating the temporal artery wall (S3) and recording from the mentalis muscle (R3). \*The beginning of the external carotid artery (ECA; upstream of the temporal artery) was exposed, and 1 mg/50  $\mu$ L lidocaine was injected around the external carotid artery to block the nerve fibers on the artery wall. \*\*The superior cervical ganglion was resected.

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