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Is hemifacial spasm affected by changes in the heart rate? A study using heart rate variability analysis

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HIGHLIGHTS

- A transient increase in the heart rate occurred a few seconds before the onset of hemifacial spasm.
- Fluctuations in the heart rate were significantly enhanced during hemifacial spasm.
- The etiology of hemifacial spasm may involve changes in pulsatile frequency in offending arteries.

ABSTRACT

Objective: Hemifacial spasm (HFS) is caused by arterial conflict at the root exit zone of the facial nerve. As the offending artery is pulsatile in nature, this study investigated the association of heart rate fluctuation with HFS.
Methods: Twenty-four preoperative patients underwent simultaneous recordings of facial electromyogram and electrocardiogram overnight. Series of R-wave to R-wave intervals (RRIs) in the electrocardiogram were analyzed across subjects in relation to HFS. The degree of heart rate fluctuation was quantified by analyzing the heart rate variability (HRV). The sleep stage was evaluated during the period of HFS.
Results: A 0.1 Hz fluctuation in RRIs by 5% compared to the baseline preceded a few seconds the onset of the HFS, indicating that a significant increase in the heart rate coincided with HFS. HRV analysis demonstrated that fluctuations in the heart rate were significantly enhanced during HFS. Wake or light sleep stages were more often accompanied by HFS, suggesting an association with autonomic activities.
Conclusion: Our findings suggest that the etiology of HFS is more than just a mechanical compression of the facial nerve and may involve changes in pulsatile frequency in offending arteries.
Significance: We propose the etiology of HFS from a unique standpoint.

Abbreviations: AMR, abnormal muscle response; ECG, electrocardiogram; EEG, electroencephalogram; EMG, electromyogram; HF, high frequency; HFS, hemifacial spasm; HRV, heart rate variability; LF, low frequency; NN intervals, normal to normal intervals; REM, random eye movement; REZ, root exit zone; RMSSD, square root of the mean squared difference of successive NN intervals; RRI, R-wave to R-wave interval; RVLM, rostral ventro-lateral medulla; SDNN, standard deviation of the normal to normal intervals; SWS, slow wave sleep.

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1. Introduction

Hemifacial spasm (HFS), a movement disorder characterized by involuntary muscle contraction of one side of the face. HFS impairs not only daily activities but also social activities of patients. Because the central etiology of HFS is mechanical compression by offending arteries to the root exit zone (REZ) of the facial nerve (Campbell and Keedy, 1947; Gardner, 1962; McLaughlin et al., 1999), microvascular decompression is a standard surgical treatment, in which the arteries are microsurgically separated from the facial nerve REZ, and provides favorable outcome (Barker et al., 1995; Chung et al., 2000; Samii et al., 2002). However, a recent notable finding using high resolution MRI indicated that arterial conflict to the facial nerve was visualized in more than 70% of asymptomatic cases (Kakizawa et al., 2008). Given that the prevalence of HFS is 7.4–9.8 per 10,000 individuals (Auger and Whisnant, 1990; Mizobuchi et al., 2017), it is assumed that an arterial conflict to the facial nerve REZ is not sufficient to develop HFS. Although there are likely unknown mechanisms underlying the occurrence of HFS in addition to simple mechanical compression, only a few reports have addressed this issue.

Heart rates fluctuate periodically even at the resting or sleep state, a feature defined as heart rate variability (HRV) (Malik et al., 1996) The heart rate time series is thus characterized by a variability in the R-peak to R-peak interval (RRI, i.e., the time between adjacent R-waves in the electrocardiogram [ECG]) over a wide range and is primarily regulated by a balance between the sympathetic and parasympathetic nervous systems (Thayer et al., 2012). The arterial compression to the facial nerve is pulsatile, raising the hypothesis that the HFS is induced by chronological changes in the heart rate. We designed a study where the facial electromyogram (EMG) is recorded simultaneously with the ECG to investigate whether fluctuation in heart rates is associated with the HFS.

2. Methods

2.1. Patients enrolled in the study

Patients with HFS, who were referred to the Kumamoto University Hospital for microvascular decompression between March 2014 and December 2016, were given information detailing the purpose and protocol of this observational study before surgery. Patients with diabetes mellitus, chronic heart disease, and those treated with adrenoceptor blocking agents or anti-arrhythmic medication were excluded from this study because these conditions are known to have a significant impact on HRV (Malik et al., 1996). Twenty-four patients met our criteria and agreed to participate in this study. Informed consent was obtained from all the patients. This study protocol was approved by the institutional review board for scientific research of the Kumamoto University Hospital (Approval number, 1802).

2.2. Overnight recordings of facial EMG and ECG

Simultaneous recordings of facial EMG and ECG were performed during sleep because recording in the daytime would acquire numerous artifacts of facial EMG due to daily activities such as speaking, eating and laughing. As reported previously, the spasm persists during sleep (Wang and Jankovic, 1998). The electrodes were positioned and recording was started before the patients went to sleep. Two or three plate-type electrodes (Nihon Koden, Tokyo) were placed on each side of the face using designated paste (Fig. 1A). To fix facial electrodes, we took care to avoid facial skin erosion on their removal by using a less-adhesive tape. For ECG, the right arm (RA) and left arm (LA) electrodes ware placed on the right and left chest, respectively as per usual protocol. Simultaneous digital recordings of facial EMG and ECG were performed using the EEG-1200 system (Nihon Koden) at a sampling rate of 500–1 kHz (Fig. 1B). Once the recording was finished, the elec-

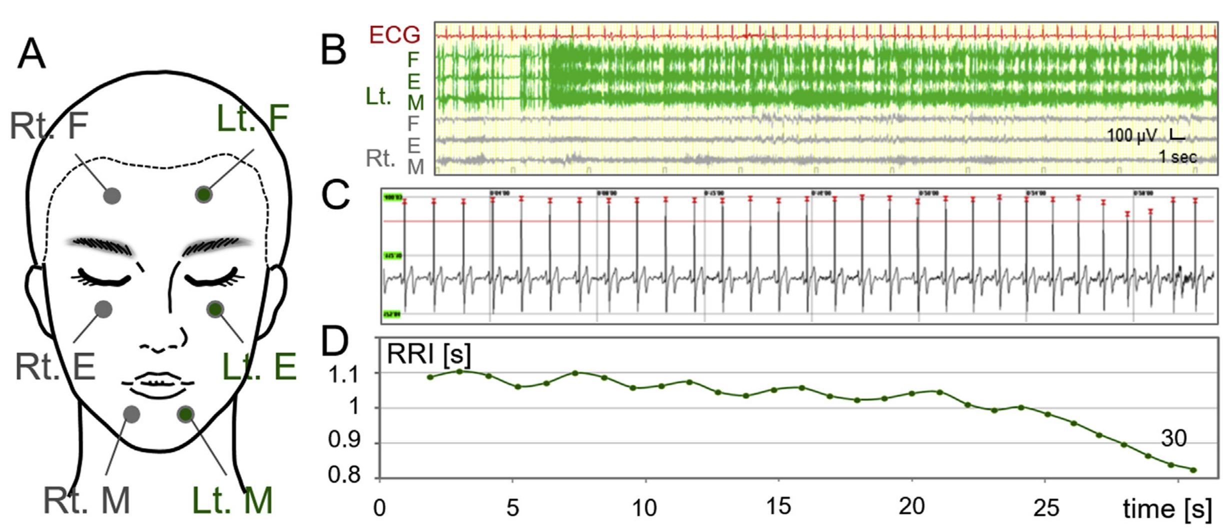

Fig. 1. Simultaneous recordings of electrocardiogram (ECG) and facial electromyogram (EMG) and analysis of the R-R interval (RRI). (A) A schematic drawing showing the bilateral positioning of the plate-type electrodes for the frontal muscle (F), orbicularis oculi muscle (E), and mentalis muscle (M) of the face. (B) A typical pattern obtained from the simultaneous recording of ECG and EMG in a patient with left hemifacial spasm. Facial EMG signals are seen on the left (green) but not on the right (gray). Vertical and horizontal scale bars indicate the voltage of EMG (100 μV) and time (1 s), respectively. (C) Measurement of RRIs in ECG. Using the extracted ECG data, the QRSTool software (see Section 2) identified R peaks (red marks) above a threshold red line and calculated the time between adjacent two R peaks. (D) An example of the RRI tacogram. The x and y axes are time and RRI, respectively. In this 30-s epoch, RRIs gradually decreased (i.e., the heart rates increased) over the last 10 s. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)