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Resting cranial and upper cervical muscle activity is increased in patients with migraine

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HIGHLIGHTS

- We applied a new method for quantifying electromyogram in scalp recordings.
- We measured scalp muscle activity in headache-free migraine patients and in controls.
- Scalp muscles exhibited more activity in migraineurs than in controls.

ABSTRACT

Objective: To compare comprehensive measures of scalp-recorded muscle activity in migraineurs and controls.
Method: We used whole-of-head high-density scalp electrical recordings, independent component analysis (ICA) and spectral slope of the derived components, to define muscle (electromyogram-containing) components. After projecting muscle components back to scalp, we quantified scalp spectral power in the frequency range, 52–98 Hz, reflecting muscle activation. We compared healthy subjects (n = 65) and migraineurs during a non-headache period (n = 26). We also examined effects due to migraine severity, gender, scalp-region and task (eyes-closed and eyes-open). We could not examine the effect of pre-ictal versus inter-ictal versus post-ictal as this information was not available in the pre-existing dataset.
Results: There was more power due to muscle activity (mean ± SEM) in migraineurs than controls (respectively, −13.61 ± 0.44 dB versus −14.73 ± 0.24 dB, p = 0.028). Linear regression showed no relationship between headache frequency and muscle activity in any combination of region and task. There was more power during eyes-open than eyes-closed (respectively, −13.42 ± 0.34 dB versus −14.92 ± 0.34 dB, p = 0.002).
Conclusions: There is an increase in cranial and upper cervical muscle activity in non-ictal migraineurs versus controls. This raises questions of the role of muscle in migraine, and the possible differentiation of non-ictal phases.
Significance: This provides preliminary evidence to date of possible cranial muscle involvement in migraine.

1. Introduction

The International Classification of Headache Disorders (ICHD) considers muscle to be relevant in tension-type headache, but does not address its relevance in migraine (Headache Classification Committee of the International Headache, 2013). There have been many studies since the 1970s examining the role of muscle in

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migraine as well as in tension-type headache, mostly qualitative (Bakal and Kaganov, 1977; Tfelt-Hansen et al., 1981; Bakke et al., 1982; Clifford et al., 1982; Lous and Olesen, 1982; Ahles et al., 1988; Celentano et al., 1990; Lebbink et al., 1991; Jensen et al., 1993; Blau and MacGregor, 1994; Burnett et al., 2000; Hagen et al., 2002; Ebinger, 2006; Leistad et al., 2006; Fernández-de-las-Peñas et al., 2008; Hung et al., 2008; Oksanen et al., 2008; Blaschek et al., 2012; Watson and Head, 2012; Didier et al., 2015; Landgraf et al., 2015). Their conclusions disagree, but many do conclude there is a link between migraine and muscle activation. Here we limit ourselves to reviewing quantitative studies of migraine.

While many studies have focussed on tension-type headache, they sometimes have included migraine groups for comparison. The methods of quantitation of muscle activity in these studies differ, such that it is difficult to make robust comparisons and identify a clear conclusion. There are differences in recording electromyogram (EMG) (surface or needle recordings), differences in muscles sampled, differences in headache phase (during inter-headache or headache periods) and differences in activity state (at rest or during instructed contraction or head postures). The extracted measures of electromyographic activity include median or mean frequency and root mean square power.

The findings have not pointed to a consistent alteration in muscle activity. Bakal and Kaganov (1977) reported migraine patients had higher frontalis EMG as well higher neck EMG activity than tension-type headache patients and headache-free controls. McArthur and cohen (1980) reported migraine patients had higher frontalis EMG activity than tension-type headache patients and headache-free controls. Anderson and Franks (1981) reported that frontalis EMG does not distinguish between tension-type and migraine headaches. Clifford et al. (1982) reported during attacks, that migraineurs had activity in the anterior temporal muscles which exceeded the patient's own baseline recordings and that all muscles were activated more strongly than in the control period. Similarly, Bakke et al. (1982) reported a rise in activity from control levels shortly before migraine patients experiencing maximal pain. Ahles et al., (1988) reported higher levels of muscle activity (frontalis, trapezius) in three headache groups, including migraineurs, than in the non-headache group, but which did not differ from each other. Jensen et al. (1994) could not identify EMG measures corresponding to 'migraine severity in the previous year' (though increased EMG measures were seen in patients with 'chronic headache'), and there was no relationship between muscle activity and migraine generally.

Given modern methods of signal analysis, it is now possible to extract EMG activity and other contaminants (such as electrooculogram (EOG), electrocardiogram (ECG), mains noise, white noise and other artefacts) from scalp electrical recordings, usually with the aim of extracting clean electroencephalogram (EEG). Using such methods, therefore, it is equally feasible to obtain 'clean' EMG and accurately quantitate power corresponding to EMG activity. We now report a topographic quantitative study on cranial and cervical muscle activity in a group of migraine sufferers and headache-free controls using recordings from an EEG cap with 128 electrodes covering the scalp. The cap included electrodes over muscles such as the frontalis, orbicularis and temporalis, and also close to nuchal (upper cervical) muscles. At frequencies above 10–20 Hz, EMG signals are the largest contributors to scalp electrical recordings, with the highest power typically in the range 60–90 Hz (Goncharova et al., 2003; Whitham et al., 2007; Whitham et al., 2008; Zhang et al., 2015). Recent research indicates that this is true even when the subject is relaxed and phasic muscle has been excised from the recordings (Whitham et al., 2007; Whitham et al., 2008; Pope et al., 2009; Yilmaz et al., 2014).

A standard approach in identifying components in electrical scalp recordings is to use Independent Components Analysis (ICA) to reveal components that correspond to separate, different sources of electrical activity, e.g. neurogenic, myogenic and others (Makeig et al., 1996; Vigário et al., 2000; Delorme et al., 2007; Prakash and Roy, 2016). The artefactual components (e.g. non-neurogenic sources if analysing EEG) are then discarded, and "clean" scalp EEG recordings can be reconstructed. In this study, we utilise the myogenic sources from ICA, enabling us to quantitate muscle activation in migraineurs versus headache-free controls. We then tested for differences in the level of muscle activity due to gender (female and male), region (frontal, left-temporal, central, right-temporal, occipital), task (eyes closed and eyes open), and condition (migraine, headache-free control). Finally, we tested for a linear relationship between muscle activity and headache severity.

2. Subjects and procedure

2.1. Subjects

The data used in this study was drawn from an existing dataset collected from participants with a range of neurological and psychological disorders and controls, whose purpose was to investigate changes in brain rhythms with disease. Both studies were approved by the Clinical Research Ethics Committee of Flinders Medical Center and Flinders University, application number: OFR # 382.13, and each participant signed a consent form. All participants were recruited from the clinics and staff of the Flinders Medical Center, or their relatives, between 2004 and 2007. All patients were evaluated by a neurologist and those with a single neuropsychiatric diagnosis were included. A power analysis for the original study recommended recruiting 10 migraine participants with aura and 10 without. Additionally, some participants recruited as controls were diagnosed as migraineurs after the initial medical examination by a neurologist. For this study, we recorded sufficient clinical information for accurate diagnosis; we did not use migraine diaries, so the full suite of migraine expression was not known. In addition, while all patients were without headache on the day of recording, we do not know how long they remained headache-free. Diagnosis was validated by another neurologist using the 2013 ICHD-III-beta diagnostic criteria (Headache Classification Committee of the International Headache, 2013), based on review of their records.

This resulted in a dataset consisting of two groups: 65 healthy participants with no history of headache and 26 migraine participants. Table 1 shows the demographic details of participants which shows gender distributions for migraineurs and controls that are close to the population expectations of 67% and 50% female. Given the inter-individual variability of muscle activity, we chose to include the maximum number of participants. The migraine participants all described their pain intensity as three or four out of five (moderate or severe), mean intensity of 3.9, and the maximum number of attacks per year was 104. 50% had migraine with aura. About 70% of the migraine participants had a frequency of one or two attacks in a month, and the mean frequency was 0.9 per

Table 1
Subject demographics.

Participants	Age (years) (mean ± SEM)	Females	Males	Number of EEG channels
Migraine	48.6 ± 13	19	7	124
Control	46.2 ± 17.2	33	32	124