Heterogeneous activity level of jaw-closing and -opening muscles and its association with arousal levels during sleep in the guinea pig

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Kato T, Masuda Y, Kanayama H, Nakamura N, Yoshida A, Morimoto T. Heterogeneous activity level of jaw-closing and -opening muscles and its association with arousal levels during sleep in the guinea pig. Am J Physiol Regul Integr Comp Physiol 298: R34–R42, 2010. First published October 21, 2009; doi:10.1152/ajpregu.00205.2009.—Exaggerated jaw motor activities during sleep are associated with muscle symptoms in the jaw-closing rather than the jaw-opening muscles. The intrinsic activity of antagonistic jaw muscles during sleep remains unknown. This study aims to assess the balance of muscle activity between masseter (MA) and digastric (DG) muscles during sleep in guinea pigs. Electromyograms (EMGs) of dorsal neck, MA, and DG muscles were recorded with video during sleep-wake cycles. These variables were quantified for each 10-s epoch. The magnitude of muscle activity during sleep in relation to mean EMG activity of total wakefulness was up to three times higher for MA muscle than for DG muscle for nonrapid eye movement (NREM) and rapid-eye-movement (REM) sleep. Although the activity level of the two jaw muscles fluctuated during sleep, the ratio of activity level for each epoch was not proportional. Epochs with a high activity level for each muscle were associated with a decrease in δEEG power and/or an increase in heart rate in NREM sleep. However, this association with heart rate and activity levels was not observed in REM sleep. These results suggest that in guinea pigs, the magnitude of muscle activity for antagonistic jaw muscles is heterogeneously modulated during sleep, characterized by a high activity level in the jaw-closing muscle. Fluctuations in the activity are influenced by transient arousal levels in NREM sleep but, in REM sleep, the distinct controls may contribute to the fluctuation. The above intrinsic characteristics could underlie the exaggeration of jaw motor activities during sleep (e.g., sleep bruxism).

Although motor systems are generally inactive or suppressed during sleep, jaw muscles are found to be activated during the normal sleep of humans and animals (8, 9, 11, 27, 31). The jaw motor activities can be associated with physiological functions, such as swallowing and respiration, and with activities that cannot be identified as specific functions or behaviors. The amount of intrinsic jaw muscle activity during sleep compared with the amount of activity during wakefulness can fall within the physiological range of recuperative function because patients suspected of having increased jaw muscle activity during sleep (e.g., sleep bruxism) complain of jaw muscle symptoms such as pain or fatigue (10, 49). Postexercise soreness or fatigue due to the overloading of the jaw muscles can be a contributing factor to the development of jaw muscle symptoms (55). Moreover, jaw muscle symptoms are often reported in the jaw-closing muscles [e.g., masseter (MA) and temporalis muscles] but rarely in the jaw-opening muscles [e.g., digastric (DG) muscle] (10, 49).

Our current knowledge about changes in the magnitude of spontaneous EMG activity of jaw-closing and -opening muscles during sleep is still limited. Previous studies have shown that state-dependent modulations of EMG activity for oromandibular muscles differ from those of the muscles in distinct anatomical or functional segments during sleep-wake cycles in humans and animals (15, 27, 40, 44). Jaw-closing and -opening muscles are antagonistic muscles, which usually exhibit alternating activations during usual oromandibular behaviors (e.g., chewing and drinking) in waking states (41, 59). Anatomical and functional differences between the motoneurons of jaw-closing and -opening muscles have been reported regarding neural connections with premotor neurons (42, 45, 54, 62). In addition, there is a different modulation in the excitability of jaw reflexes for jaw-closing and -opening muscles during sleep-wake cycles (5–7, 24). These findings suggest that jaw-closing and -opening muscles, located in the same anatomical segments (Vth cranial nerve), would be differently activated during sleep in experimental animals.

In addition to the state-dependent modulation, EMG activity and reflex excitability of jaw muscles have been reported to fluctuate within a given state of vigilance (6, 8, 24, 27, 40). The fluctuation is associated with the transient changes in sleep states, such as transient arousals during sleep; motor events in the jaw and other body segments during sleep are associated with signs of transient changes in heart rate and cortical EEG activity (e.g., micro-arousals) (14, 29, 30, 48, 53) and intense activations in a single or multiple muscles frequently occur during intense arousals, such as intermittent awakenings during sleep (19, 29). The destruction of sleep by frequent and intense arousals can be a risk for increased jaw motor activity because patients with jaw muscle pain frequently report sleep disturbance (38). On the other hand, sleep bruxism has been proposed to be a form of heightened jaw responsiveness to arousals rather than increased arousals (28). In patients with sleep bruxism, most jaw muscle events are associated with leg/body movements but sleep structures are normal (21, 35, 36). Thus, the clinical conditions with increased jaw motor activity may reflect a variety of associations between jaw motor activity and arousal levels during sleep (19, 35). However, jaw motor activity in relation to arousal levels is not well studied in experimental animals.

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Characterizing the magnitude and fluctuation of muscle activities in jaw-closing and opening muscles during sleep/wake cycles in animals will provide us with information to explore physiological mechanisms underlying an exaggeration of the jaw-closing muscle activity and associated jaw muscle symptoms in a future study. In this study, we attempt to demonstrate that 1) the magnitude and timing of the activity in MA and DG muscles are heterogeneous during sleep, and 2) the changes in cortical EEG and cardiac activity are associated with a level of muscle activity or with the number of muscles activated during sleep. The guinea pig was chosen as an experimental animal since these animals are often used for investigating the physiology of sleep and trigeminal motor systems (4, 12, 16, 32, 56, 57).

MATERIALS AND METHODS

Experiments were carried out on 10 adult male albino guinea pigs (Hartley) weighing 400 to 500 g. Animals were housed individually or in pairs in a meshed-floor cage, maintained on a 12:12-h light-dark cycle (06:00 to 18:00) with temperature and moisture at 22°C and 50%, respectively. They were allowed access to food and water. Animals were fed a standard guinea pig diet. All experimental procedures were reviewed and approved by the Animal Research Ethics Committee of the Matsumoto Dental University. Procedures also conformed to the APS guiding principles in the care and use of animals.

Surgical Preparation

Surgery was performed under pentobarbital sodium anesthesia (40 mg/kg ip) with an addition of atropine (0.04 mg/kg ip) at such a level that neither apparent corneal reflex nor spontaneous eye movements were present. All wound margins were anesthetized using small injections of lidocaine hydrochloride (Xylocaine; Fujisawa Pharmaceutical, Osaka, Japan). Rectal temperature was maintained between 36 and 38°C with a heating pad. An electrocardiogram was continuously monitored. The electrodes for electroencephalogram (EEG), electrooculogram (EOG), electrocardiogram (ECG), and electromyograms (EMGs) from dorsal neck (dNE), MA, and DG muscles were implanted as follows. To implant the EEG electrodes and settle the multiple-pin connectors, the surface of the skull was exposed. T-shaped stainless steel screws (diameter: 1.4 mm) were implanted into the skull; two screws for EEG were placed over the frontal cortex, another two in the right orbital bone for EOG, and one in the occipital bone for the ground. These screws were soldered with silicone-coated multistranded stainless wires (diameter: 0.05 mm) before surgery. For EMG recording, the skin of the submandibular and dNE areas was incised, and two pairs of wires were sutured onto the dNE muscles and onto the left MA and DG muscles. For ECG recording, a pair of wires was sutured onto the right and left sides of the rib cage after the skin on the sternum was incised. The EMG and ECG wires were tunneled subcutaneously to the exposed surface of the skull. The wires for EEG, EOG, ECG, and EMGs were soldered to a multiple-pin socket in the connectors. The connectors were fixed to the skull with dental acrylic resin. After all incisions were sutured, antibiotic ointment (gentamycin sulfate) was applied around the wound, and an antibiotic (10 mg/kg oxytetracycline) and analgesics (0.8 mg/kg flurbiprofen axetil (Ropion); Kaken Seiyaku, Tokyo, Japan) were injected intra-peritoneally for 3 days following surgery. The animals rested in a clean cage under a warm heating pad until full recovery from anesthesia was confirmed. They were then returned to the home cage.

Recording Procedures

Recovery from the surgery was confirmed by the presence of normal behaviors such as locomotion, grooming, and ingestion. Recording sessions started 5 to 7 days after postoperative recovery (4 or 5 days). During the entire recovery period, the animals were adapted to the acrylic recording chamber on at least three separate days under the same conditions as recording protocols. During recording, an animal was placed in the recording chamber in a noise-attenuated, electrically shielded cubicule. A lightweight shielded cable was connected to the multiple pins on the animal’s head. The other side of the cable was connected to the multichannel slip-ring so as not to disturb free movement. The recordings were made for 5 h during a light period (11:00 to 16:00). Animals had free access to food and water; pellets were contained in a plastic bait box (5 cm × 10 m × 2 cm), and the water bottle faucet was located 4 cm above the floor on the side opposite to the bait box. A one-way mirror and a video monitoring display enabled experimenters to observe the animal’s behavior without disturbing it.

The electrical signals were amplified and filtered (model AB-621G; NIHON-KODEN, Tokyo, Japan) with optimal bandwidths (EEG, EOG, and ECG: 0.3–100 Hz; EMGs: 100–1,000 Hz; 60-Hz hum filters for all), and the results were fed continuously into a personal computer by using a commercial program (SleepSign; KISSEI COMTEC, Matsumoto, Japan) with a sampling rate of 512 Hz for all channels. The animal’s behavior was simultaneously video taped for offline analysis. The video system consisted of a near-infrared camera located 20 cm in front of the recording chamber and an audio-video recorder.

Scoring States of Vigilance and Behaviors

The states of vigilance (e.g., wakefulness, NREM sleep, and REM sleep) were determined for 10-s epochs on the basis of EEG, dNE EMG, and EOG activities according to the previous studies (22, 27, 47, 56). In addition to the visual analysis, the cortical power spectrum of the delta activity (0.5–4.5 Hz) was also used to score NREM sleep. Wakefulness was scored when the integrated dNE EMG was high and the EEG power in the delta band was low. In NREM sleep, the dNE EMG activity was low, and the δEEG power band was increased. REM sleep was scored when the integrated EMG was lower than that scored in NREM sleep with an occasional occurrence of REMs and with a low δEEG power (Fig. 1). Each animal’s behavior was also

Fig. 1. State-dependent changes in δEEG and integrated EMG activities and heart rate during sleep-wake cycles. The δEEG (6 power) was high during NREM sleep. EMG activities in the dorsal neck (dNE), masseter (MA), and digastric (DG) muscles were high during wakefulness (W), while jaw muscle activations can occur in some sleep epochs. Mean heart rate (mHR) per epoch was low during NREM sleep.
scored during wakefulness in combination with polygraphic traces and video records. The periods of wakefulness, NREM sleep, and REM sleep periods were determined by the following criteria: periods of wakefulness were terminated by three consecutive NREM sleep epochs; NREM sleep periods ended if more than three consecutive epochs scored as wakefulness or REM sleep; REM sleep periods were terminated if followed by more than three consecutive wakefulness or NREM sleep epochs.

For the behavioral analysis, the oro-facial behaviors of each animal during the recording were scored for each 10-s epoch and classified into the following categories: chewing (CH), drinking (DR), grooming (GR), and other nonspecific behaviors (OT) (including yawning, gritting, gnawing on bait box, sniffing, etc.). When the animals were consuming food, they showed a series of oral behaviors; they first picked up one or more pellets in the mouth and transported the food to the molars before starting CH. These preparatory behaviors were included with CH and DR in calculations of the time spent on ingestive behaviors.

Quantitative EMG Analysis

EMG activities from muscles were first integrated for every 10-s epoch. To exclude the influence of the electrical baseline in the EMG data, the minimal integrated value was subtracted from all measurements for the three muscles (27, 40). Then EMG measurements were normalized by mean activity during total wakefulness (100%). To assess the variability of the normalized activity during sleep and wakefulness, standard deviations of normalized activity were calculated for each state of vigilance and behaviors (40). To evaluate the correlation of the muscle activities between MA and DG muscles, the activity balance of the two muscles was calculated as a logarithmic value of the ratio between normalized MA and DG muscle EMG activities [Log(DG EMG (%)/MA EMG (%))]. Thus, the ratio can be negative if the MA muscle activity is more dominant compared with that of the DG muscle at a given epoch, and it becomes zero when the activity of the two muscles is equal. Standard deviations of the ratio for vigilance and behavioral states were used to assess the range of the activity balance; a large standard deviation indicates heterogeneity of activity between two muscles in the same recording epoch.

EEG and Heart Rate Analysis

Mean heart rates were calculated from the mean values of RR intervals for 10-s epochs using the SleepSign software package. To quantify EEG activity, power spectral analyses were performed with the software package, which computes fast-Fourier transforms on 10-s epochs with a cosine window tapering. The median power of the delta intervals for 10-s epochs using the SleepSign software package. To assess the variability of the normalized activity during sleep and REM sleep periods, out of the 3-h recording time, the percentage of waking periods was 51.8 ± 2.0% (93.1 ± 3.0 min) and those of NREM and REM sleep periods were 38.1 ± 1.4% (68.6 ± 2.5 min) and 10.1 ± 0.7% (18.3 ± 1.2 min), respectively. The animals usually became quiet for a while after active wakefulness and then fell into NREM sleep followed by REM sleep before returning to wakefulness (Fig. 1).

Of the total wakefulness, 41.9 ± 4.4% (41.0 ± 5.1 min) was spent on ingestive behaviors [preparatory behavior before CH: 0.7 ± 0.3% (0.66 ± 0.1 min); CH: 35.6 ± 3.1% (34.3 ± 3.9 min); DR: 6.2 ± 1.7% (6.1 ± 1.7 min)]. Grooming occupied 2.0 ± 0.6% (1.85 ± 0.5 min). The duration of quiet wakefulness without motor activity was 5.4 ± 1.1% of total wakefulness (5.0 ± 1.0 min). The remaining periods were associated with OT, such as sniffing, yawning, gritting, and gnawing with or without walking.

Magnitude of Muscle Activity

During wakefulness. The muscle activity for CH, DR, GR, and OT differs significantly between the three muscles (muscle effect: P < 0.01; behavior effect: P < 0.01; interaction: P < 0.01, Fig. 2). Among the behaviors analyzed, the MA muscle was most active during CH (280.2 ± 24.3%) (behavior effect: P < 0.001; post hoc tests: P < 0.05). DG muscle activity did not differ between CH (205.0 ± 24.1%) and DR (173.4 ± 19.0%) (behavior effect: P < 0.001; post hoc tests: P < 0.05). The dNE muscle activity was highest during DR (216.3 ± 34.4%) and GR (156.1 ± 20.9%) (behavior effect: P < 0.001; post hoc tests: P < 0.05). The mean activity balance between MA and DG muscles differed significantly between the four behaviors (behavior effect: P < 0.001, Fig. 3A). The ratios did not differ between CH (−0.001 ± 0.02) and DR (0.17 ± 0.09). The ratio was lowest for OT, while it was highest for GR (0.47 ± 0.07) (post hoc tests: P < 0.05). Variability in the activity balance was lowest for CH (0.11 ± 0.01) and second lowest for DR (0.18 ± 0.02), smaller compared with other behaviors (GR: 0.31 ± 0.05; OT: 0.59 ± 0.05) (behavior effect: P < 0.001; post hoc...
The magnitudes in EMG activities of MA and DG muscles differed between chewing foods (CH), drinking water from a faucet (DR), grooming (GR), and other behaviors (e.g., yawning, gritting, etc.) (OT). \( *P < 0.05; **P < 0.01; ***P < 0.001 \). Bars = means \( \pm SE \).

tests: \( P < 0.05 \), Fig. 3B), indicating that the activity balance between MA and DG muscles was most stable for CH behavior.

**During sleep.** The magnitude of the EMG activity for the three muscles decreased during sleep compared with wakefulness but state-dependent modulation of muscle activity from wakefulness to sleep states differed between muscles (Fig. 4). While no difference between the two sleep stages was noted for the magnitude of the activity of MA and DG muscles, the magnitude of dNE muscle activity was significantly lower in REM sleep than in NREM sleep (\( P < 0.01 \)). In NREM sleep, muscle activity for the MA (26.1 \( \pm \) 3.4\% ) and dNE (28.8 \( \pm \) 3.2\% ) muscles was higher than that of DG muscles (10.7 \( \pm \) 1.8\% ) (muscle effect: \( P < 0.001 \); post hoc tests: \( P < 0.01 \)). In REM sleep, MA muscle activity (27.6 \( \pm \) 7.1\% ) was higher than the muscle activity of the DG (9.4 \( \pm \) 2.9\% ) muscles (muscle effect: \( P < 0.05 \); post hoc test: \( P < 0.01 \)).

Compared to the variability of total wakefulness, variability in the magnitude of muscle activity decreased to levels three to ten times lower during sleep for the jaw and dNE muscles (muscle effect: \( P < 0.01 \); state effect: \( P < 0.001 \); interaction: \( P > 0.05 \)). Variability did not differ between sleep states for the jaw muscles, while the dNE muscle became less variable in REM than in NREM sleep (post hoc tests: \( P < 0.05 \)). In NREM sleep, the magnitude of MA muscle activity was two to four times more variable (39 \( \pm \) 6.9\% ) than that of the DG (9.3 \( \pm \) 1.8\% ) and dNE (15.7 \( \pm \) 2.2\% ) muscles (\( P < 0.05 \)). In REM sleep, the variability of MA muscle activity (30.7 \( \pm \) 8.2\% ) was approximately three times higher than activity in the DG (9.3 \( \pm \) 1.8\% ) and dNE (10.4 \( \pm \) 1.2\% ) muscles (\( P < 0.05 \)).

The mean activity balance between MA and DG muscle activity significantly differed between wakefulness and the two sleep states (state effect: \( P < 0.001 \); Fig. 5A). The ratio was significantly lower in NREM sleep (\( -0.32 \pm 0.23 \)) and REM sleep (\( -0.43 \pm 0.24 \)) compared with wakefulness (\( -0.02 \pm 0.1 \)) (post hoc tests: \( P < 0.01 \)). No difference was noted between the two sleep stages. The ratio was significantly more variable for NREM sleep (0.55 \( \pm \) 0.05) than for wakefulness (0.48 \( \pm \) 0.04) and REM sleep (0.41 \( \pm \) 0.04) (state effect: \( P < 0.05 \); post hoc tests: \( P < 0.05 \); Fig. 5B). These values were two to four times higher during sleep than during chewing and drinking.

**Association Between Muscular, Cortical and Cardiac Activities**

The delta power of EEG activity was higher in NREM sleep (561.6 \( \pm \) 72.4 \( \mu V \)\(^2\) ) than during wakefulness (194.9 \( \pm \) 23.4 \( \mu V \)\(^2\) ) or REM sleep (178.1 \( \pm \) 19.5 \( \mu V \)\(^2\) ) (state effect: \( P < 0.001 \); post hoc tests: \( P < 0.01 \)). The mean heart rate was lowest in NREM sleep (NREM sleep: 282.1 \( \pm \) 6.0 beats/min; REM sleep: 292.9 \( \pm \) 6.9 beats/min; wakefulness: 290.8 \( \pm \) 6.0 beats/min; state effect: \( P < 0.001 \); post hoc tests: \( P < 0.01 \)).

In NREM sleep (Fig. 6), \( \delta \) EEG power was significantly decreased as muscle activity level graded up from the 1st to the 4th quartile levels for all three muscles (activity effects: \( P < 0.01 \)); the reduction of \( \delta \) EEG power in relation to an increase of the activity levels for MA, DG, and dNE muscles was 14\%, 12\%, and 23\%, respectively (post hoc tests: \( P < 0.05 \)).

The mean heart rate was highest at the 4th quartile level of muscle activity for the MA muscle (activity effects: \( P < 0.01 \)),
no difference between the activity levels was observed in the other two muscles (Fig. 7). In REM sleep, the mean heart rate was not affected by the levels of muscle activity for the three muscles. During wakefulness, mean HR increased more as muscle activity was elevated to a high level for all muscles (activity effects: $P < 0.001$, Fig. 7).

The changes in EEG power and heart rate were more clearly pronounced with an increase in the number of muscles showing a higher level of activity at the same epoch in NREM sleep. EEG power was significantly lower and heart rate was significantly higher when the three muscles were activated at the 4th quartile activity level compared with that when less muscles were activated (muscle number effect: $P < 0.01$ for both variables) (Fig. 8, A and B). However, such correlations were not found for heart rate during REM sleep (Fig. 8C).

During wakefulness, the mean heart rate was significantly elevated as more muscles were activated from the 1st quartile level to higher levels (muscle number effect: $P < 0.001$) (Fig. 8D).

**DISCUSSION**

The results showed that the magnitude of the muscle activity during sleep relative to the magnitude during wakefulness was significantly higher for the MA muscle compared with the DG muscle, although the muscle activity decreased and varied for NREM and REM sleep. The fluctuations in muscle activity for the MA muscle were not in proportion to those for DG muscle. A high magnitude of jaw muscle activity was associated with a decrease in EEG power and a slight increase in heart rate during NREM sleep, while no changes were found in REM sleep. Thus, state- and time-dependent changes in the magnitude of the EMG activity of jaw-closing and -opening muscles in relation to cortical and autonomic activities are differently modulated during sleep and an increase in muscle activity for both muscles may be under the influence of distinct neurophysiological substrates between NREM and REM sleep.

**Jaw Muscle Activity During Wakefulness**

The large variation in muscle activity during all waking periods is due to the occurrence of various types of behaviors during wakefulness (18, 26). The discrepancy in muscle activity between the jaw-closing and -opening muscles in various behaviors results from different central motor controls to the two muscles (4, 16, 58). However, the muscle work ratio between the two muscles was very stable during chewing and drinking, suggesting that the relevant neural networks coordinate the activities of the two muscles for executing and completing the respective behavior appropriately (41, 59). Behavior-related variation in muscle activity was also observed in the dNE muscle during oromandibular behaviors because this muscle contributes to postural adjustments during jaw movements (23).

**Jaw Muscle Activity During Sleep**

Oromandibular behaviors are not present under the loss of consciousness during sleep. Therefore, the magnitude and var...
ability of jaw muscle activity during sleep was decreased compared with wakefulness. The decreased muscle activity during sleep is a contributing factor to the circadian and ultradian variations of the jaw muscle activities in sleep-wake cycles (18). However, jaw muscles can be activated within a certain level of the magnitude and variability during sleep in guinea pigs. The levels of intrinsic muscle activity for MA and DG muscles in this study may lie within recuperative ability for both muscles during sleep in guinea pigs because jaw muscle symptoms in the morning are associated with the increased level of muscle activity during sleep (10, 39, 49). The magnitude of intrinsic muscle activity during sleep relative to wakefulness in the MA muscle was approximately up to three times as high as the magnitude in the DG muscles. The MA and DG muscles in guinea pigs are composed of fast oxidative glycolytic or IIA fibers but the DG muscle include fast glycolytic fibers (50). Thus, the MA muscle may be more fatigue-resistant compared with the DG muscle and can therefore cope with a high magnitude of activity during sleep. However, the high magnitude of activity in the MA muscle during sleep is characterized by a larger range of variability; i.e., intense MA muscle contractions can occasionally occur compared with the DG muscle. This suggests the possibility that the MA muscle has a lower safety margin for recuperating from muscle fatigue when muscle activity is increased during sleep. This may explain why jaw muscle symptoms are often reported in the jaw-closing muscles in patients with the increased jaw muscle activity during sleep. The difference in muscle activity levels between the MA and DG muscles during sleep may be due to the distribution of heterogeneous excitatory and inhibitory influences to MA and DG motoneurons; the motoneurons that innervate the MA muscle have different patterns and amounts of premotor connections from those of the DG muscle (42, 45, 54, 60, 62).

Arousal Level and Motor Activation in NREM Sleep

In NREM sleep, the δEEG power decreased when a higher level of muscle activation occurred in the MA and DG muscles, supporting previous observations of the association between transient arousals and motor activities (19, 35, 37). Autonomic control is characterized by increased parasympa-
thetic and reduced sympathetic tone in NREM sleep. Cardiac activation is typical of transient arousals and is correlated with the level of arousal and with a decrease in δEEG activity in NREM sleep (3, 29, 33, 48, 52, 61). In this study, heart rate increase was only observed for the MA muscle. The heart rate increase detected in this study more likely reflects an intense (high or prolonged) cardiac activation because mean heart rate analysis is less sensitive for clearly detecting transient heart rate increase (3, 61). In addition, heterogeneous motor activation in time and magnitude among the muscles might obscure a slight heart rate increase with the activation of DG and dNE muscles. Whether this is the case or not, the results indicate that a high level of cardiac activation is associated with the occurrence of a high level of transient muscle activity in the MA muscle; this muscle exhibited larger variability in the magnitude of EMG activity compared with DG and dNE muscles. Considering that the changes in heart rate reflect physiological adjustments in autonomic activity during NREM sleep and wakefulness (1, 19, 46), the activity-dependent increase in heart rate can occur during NREM sleep, as seen in waking behaviors with high activity level in the jaw muscles (Fig. 2). Moreover, the changes in δEEG power and in heart rate are clearly correlated with a high activity level in all three muscles during the same time epoch. These results imply that the intensity of muscle activity in a given muscle and the recruitment of multiple muscles are associated with a hierarchy of arousal level in NREM sleep (19, 29, 52), probably because the descending excitatory influences increase as transient arousal activation becomes more intense in this period (13, 25, 35, 53).

Motor Activation and Heart Rate in REM Sleep

In REM sleep, no increase has been found for mean heart rate in association with the level of jaw muscle activity and the number of muscles activated. Distinct cardiac and motor controls in REM sleep from NREM sleep can be attributed to the lack of an association between heart rate increase and motor activity. The mean heart rate was as high in REM sleep as it was during wakefulness in this study, whereas autonomic controls are known to operate differently between REM sleep and other states, such as NREM sleep and wakefulness (1). In REM sleep, pontine inhibitory neurons tonically hyperpolarize the motoneurons in addition to a withdrawal of descending excitatory inputs (2, 8, 20, 25, 51). This system can contribute to less movement in REM sleep unless potent behavioral requirement (e.g., dangerous stimuli) does not occur, as previous studies showed a higher arousal threshold for motor activation in REM sleep in humans (28, 29, 34). Nonetheless, studies have suggested that brain stem neural systems, rather than the cerebral cortex, generate phasic excitatory inputs that overcome the tonic inhibition (2, 8, 43). A higher incidence of the phasic jaw muscle activation results in a lack of decrease in the magnitude of jaw muscle activities in REM sleep unlike those in the dNE muscle (2, 15). Thus, the magnitude and fluctuation of MA and DG muscles in REM sleep reflect the occurrence of phasic motor events that result from the inhibitory and facilitatory influences, operating under the open-loop regulations of the autonomic activity (1, 46).

Perspectives and Significance

A higher level of jaw-closing muscle activity compared with the jaw-opening muscle may be one of the physiological factors predisposing to muscle symptoms of jaw-closing muscles in patients with increased jaw muscle activity during sleep. The inter- and intrasegmental difference in motor activation
during sleep can imply that these muscles have distinct motor responsiveness in relation to arousal activities, because the activity levels of these muscles are associated with arousal levels. In addition, sleep state (NREM and REM sleep) also influences motor responsiveness during sleep. These results underscore the importance of understanding state-dependent changes in the balance between motor and arousal activities (cortical or autonomic) when assessing the exaggerated jaw motor activity during sleep in animals and humans. In this study, guinea pigs were used as an experimental animal since guinea pigs have been used for neurophysiological studies of sleep and trigeminal/jaw motor functions (4, 12, 16, 17, 32, 56). Future studies are needed to assess the effects of experimental disturbance of sleep or motor excitability on the patterns of balance between arousal and jaw motor activities, and to explore the neurophysiological substrates contributing to exaggerated jaw motor activities.

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DISCLOSURES

No conflicts of interest are declared by the author(s).

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