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Load compensatory response of external intercostal muscles during assisted ventilation in rabbits

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Short communication

Abstract. Phrenic and external intercostal motor responses to inspiratory load were studied in anaesthetized rabbits and after bilateral vagotomy. Animals were ventilated with a phrenic nerve driven respirator set at different gains, i.e. volume to phrenic-signal ratio. Load was imposed by occluding the trachea at the end of expiration for the period of one breath. It was found that vagally mediated low threshold facilitation may be observed in rabbits on late-inspiratory external intercostal motor units. After vagotomy, external intercostal EMG response to load depended on the given of the servorespirator set during the period of assisted ventilation.

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Since the work of Hering and Breuer (1868), it is known that inspiratory time and amplitude is controlled by inhibitory vagal input. In contrast, the rate of rise of inspiratory activity may be facilitated by afferent vagal inflow (Bartoli et al. 1975). This facilitatory effect is better manifested on external intercostal (EI) than phrenic (Phr) motor output (DiMarco et al. 1981). On the other hand, EI muscle activity is under control of extra-vagal load-compensatory reflex originated in muscle spindles (Sears 1964, von Euler 1973). Both these reflexes, vagally and spinally mediated, act in opposite directions on on-going inspiratory activity. Vagally mediated low-threshold facilitation was never demonstrated in rabbits. We hypothesized that in rabbits, during inspiratory loading, both these reflexes are in balance and they annul effects of each other. It is possible that, by changing the balance, the studied effects would be unmasked.

In this study, we consider the responses of EI muscles to tracheal occlusion before and after vagotomy in anaesthetized rabbits ventilated with a phrenic nerve driven respirator set at different levels of signal to volume ratio (Huszczuk 1970, Bartoli 1975). Ventilation with different gains of the servorespirator gave us the opportunity to change the balance between afferent and efferent signals.

The experiments were carried out on 6 male rabbits weighing 2.5-3.4 kg anaesthetized with an intravenous mixture of chloralose (33 mg/ml) and urethane (400 mg/ml) in the dose of 2 ml per kg b.w. Animals either breathed spontaneously or breathing was assisted by phrenic nerve driven respirator. Experiments with assisted ventilation and recordings were performed according to previously described methods by Romaniuk et al. (1976). The EMG of external intercostal (EI) muscles from 3 to 6 intercostal spaces was recorded with concentric needle electrodes. To measure the effects of phasic vagal input on inspiratory activities, the respirator was switched off (Bartoli et al. 1975) and tracheal occlusion was applied at the end of an expiratory pause and maintained through the following inspiratory effort ("no - inflation test"). We could also increase the gain of the servorespirator (i.e. for the same signal of Phr ENG, the volume of inflation was higher) for one breath or for a period of several minutes. Presented results are those which were consistent in all studied rabbits.

During tracheal occlusion in vagi intact rabbits (Fig. 1A), an amplitude and duration of inspiratory activity of both Phr (ENG) and EI EMG increases

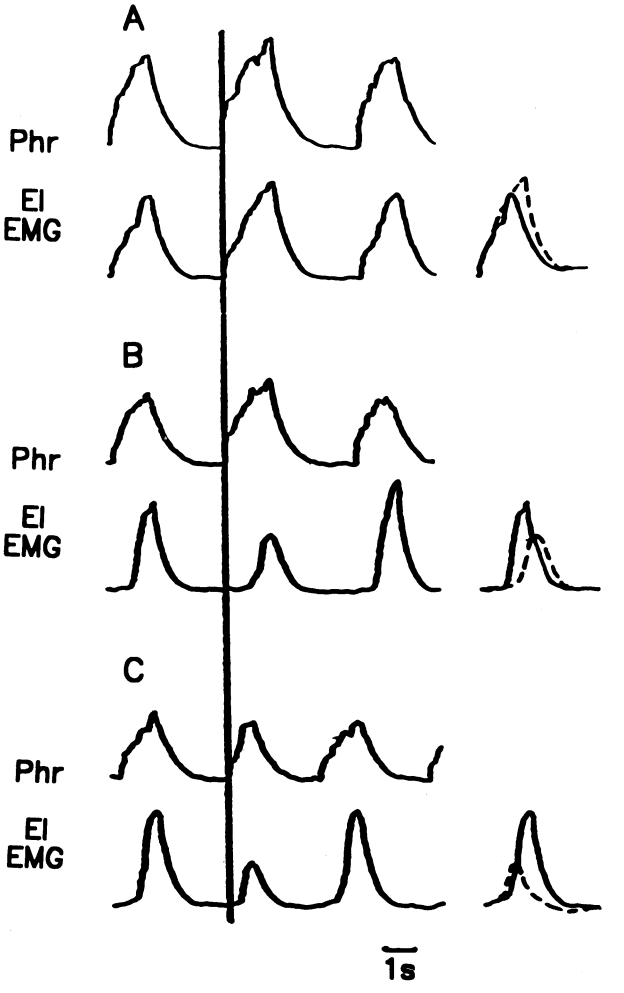

Fig. 1. The effect of tracheal occlusion on phrenic EMG activity and A, early-inspiratory and B, late-inspiratory external intercostal (EI) motor unit activity during assisted ventilation. In C, (late inspiratory EI motor units) the servorespirator gain was switched to a higher volume to signal ratio (increase 200%) for one breath. Right panel: EI EMG from the first (control) breath (continuous line) overlap with EI EMG recorded during tracheal occlusion (dashed line) to compare delay and rate of rise of EI activity. All manoeuvers were performed in the second breath. Vertical line denotes onset of inspiration. Vagi intact.

according to the classic Hering-Breuer inflation reflex. Figure 1A shows an example of integrated activity of early inspiratory EI units, with the onset of their activity simultaneous with the onset of Phr ENG. Figure 1B presents an example of late inspiratory EI units, with the onset of their activity delayed to the onset of Phr. The rate of rise of Phr (ENG) as well as EI EMG of early inspiratory units did not change tracheal occlusion (Fig. 1A). There was, however, a delay in the onset and decrease in both rate of rise and peak amplitude of EI EMG of late inspiratory units in response to tracheal occlusion (Fig. 1b, right panel). In contrast, increasing the gain of the servorespirator for one breath shortened inspiration and decreased delay of the onset of activity of late-inspiratory EI motor units (Fig. 1C).

Figure 2 presents the effect of tracheal occlusion performed during assisted ventilation set at two different servorespirator gains. Animals were not tested until the new steady state had been maintained for several minutes. In Fig. 2B (lower panel), the gain of the servorespirator was 40% higher than in the example shown in Fig. 2A (upper panel). During ventilation with higher gain, the amplitudes of control Phr and EI activities, as well as inspiratory duration, were smaller compared to the ventilation with lower gain. This was an effect of stronger inspiratory inhibition produced by the larger ventilatory volume. During tracheal occlusion, the rate of rise of EI motor units decreased. The decrease was larger for higher gain of the servorespirator. This confirms our hypothesis that changing the balance between afferent and efferent signals may reinforce the observed effect.

In our previous studies, inspiratory facilitation (DiMarco et al. 1981) was observed in rabbits only with impairment of brainstem function due to lesions (Romaniuk and Budzińska 1985) or cerebral ischemia (Pluta and Romaniuk 1990). In the present study, facilitation was observed but only on late-inspiratory EI units lung inflation. This finding could be compared with our earlier observation that late-inspiratory phrenic motoneurones are more sensitive to vagal input than early-inspiratory ones (Karczewski et al. 1980). Brainstem lesions or cerebral ischemia may lower the threshold for facilitatory vagal input (Fadiga et al. 1965) and, in these circumstances, the excitation by lung inflation may be observed also on Phr ENG.

The response of Phr ENG to tracheal occlusion was partically after vagotomy (Fig. 3). As in Fig. 3A, EI EMG activity also did not change during tracheal occlusion performed for one breath. However, after increasing the gain of the respirator and ventilating the animal with a higher volume to signal ratio, tracheal occlusion increase EI EMG activ-

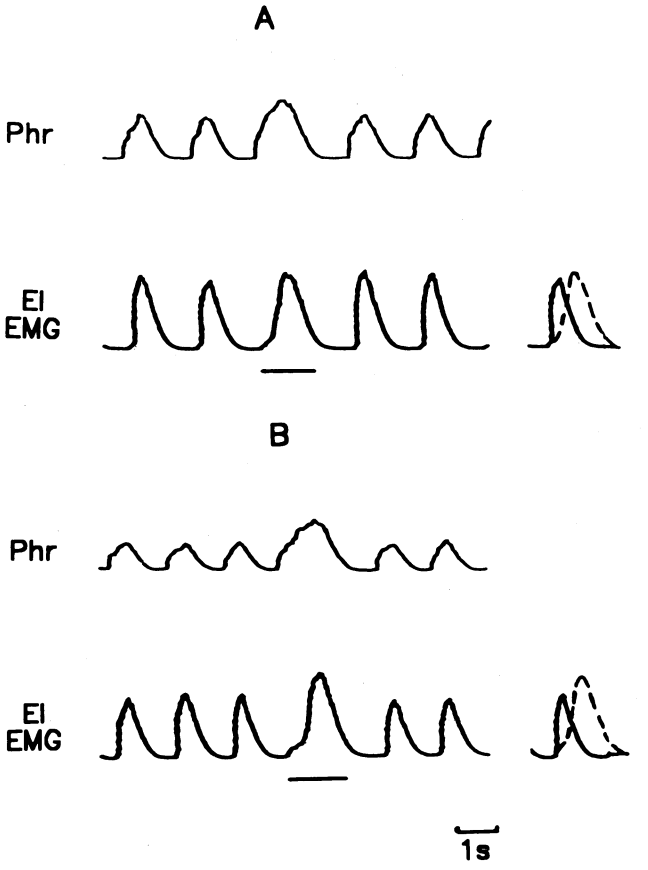

Fig. 2. The effect of tracheal occlusion (underlined) performed during assisted ventilation at two different servorespirator gain settings. In B, the gain of the respirator was 40% higher than in A. Vagi were intact. There was a stronger response of EI EMG activity during tracheal occlusion compared to preceding unoccluded breaths when ventilation was conducted with higher gain (B). Right panel: comparison of EI EMG recorded during tracheal occlusion (delayed with larger amplitude) and EI EMG during preceding control breath.

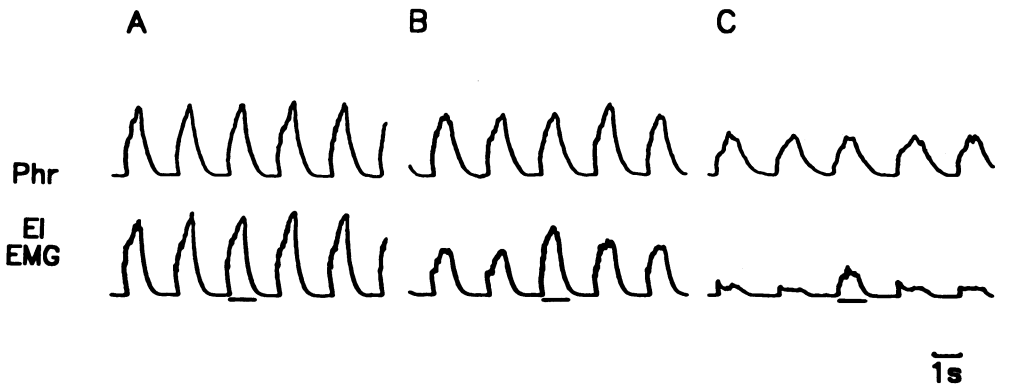

Fig. 3. The effect of tracheal occlusion performed during assisted ventilation at three different servorespirator gains: A, control; B, 50% gain increase and C, 100% gain increase. Vagi were cut. During assisted ventilation with higher gain, the response to tracheal occlusion compared to previous unoccluded breath was stronger (A to C).

ity (Figs. 3B and C). With higher gain, increase of peak EI EMG during occlusion was larger compared to preceding unoccluded breath. Control EI EMG activity decreased with increase of the respirator gain because of both hyperventilation and unloading of muscle spindles. In our experiments in dogs, we have also shown (Romaniuk, Kowalski, Supinski, DiMarco - not published) that increase in chemical drive reinforces the load compensatory response. In this paper, chemical drive was lower for higher gain of servorespirator. This suggests that increase of EI EMG response to load during high gain ventilation was not chemically mediated.

In spontaneously breathing animals, tracheal occlusion can produce a decrease of peak EI EMG activity compared to the preceding unoccluded breath (Fig. 4A). This effect is the opposite to vagal and spinal load compensatory reflexes and may be explained by inhibition originated from tendon organs (Shannon 1986). After unloading the muscle spindles by application of assisted ventilation with high gain for several minutes, the classical load compensatory response was restored (Fig. 4B).

Our recent studies, performed in dogs (Romaniuk et al. 1992), showed that load compensatory response depends on the difference in muscle length between occluded (with load) and unobstructed (free) breath. We suggest that different EI responses to tracheal occlusion in the present study could be explained by different levels of unloading the muscle spindles during assisted ventilation prior to occlusion.

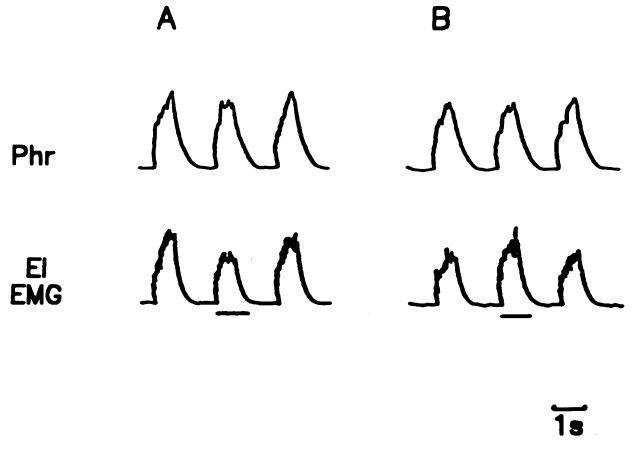

Fig. 4. The effect of tracheal occlusion during: A, spontaneous breathing and B, after switching to assisted ventilation with a high gain (300%). Vagi were cut. During tracheal occlusion in spontaneously breathing animals, there was a decrease in the EI EMG activity. After switching to assisted ventilation with a high gain, there was an increase of EI EMG in response to tracheal occlusion.

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