

Response of the Canine Internal Intercostal Muscles to Chest Wall Vibration

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Although high-frequency mechanical vibration of the rib cage reduces dyspnea, its effects on the respiratory muscles are largely unknown. We have previously shown that in anesthetized dogs, vibrating the rib cage during inspiration elicits a marked increase in the inspiratory electromyographic (EMG) activity recorded from the external intercostal muscles but does not affect tidal volume (V_T). In the present studies, we have tested the hypothesis that the maintenance of V_T results from the concomitant contraction of the internal interosseous (expiratory) intercostals. When the rib cage was vibrated (40 Hz) during hyperventilation-induced apnea, a prominent activity was recorded from the external intercostals but no activity was recorded from the internal intercostals, including when the muscles were lengthened by passive inflation. The internal intercostals remained also silent when vibration was applied during spontaneous inspiration, and the phasic expiratory EMG activity recorded from them was unaltered when vibration was applied during expiration. Thus, the internal interosseous intercostals in dogs are much less sensitive to vibration than the external intercostals, and they do not interfere with the action of these latter during rib cage vibration. This lack of sensitivity might be the result of a reflex inhibition of the muscle spindle afferents by afferents from external intercostal muscle spindles.

Although medical treatment of airflow obstruction has made significant progress in the last 20 years, dyspnea remains a major cause of disability in many patients with chronic obstructive pulmonary disease (COPD). Recent studies, however, have shown that this symptom could be relieved by the application of high-frequency mechanical vibration to the parasternal region of the rib cage during inspiration (1, 2). The relief, quantified on the basis of visual analog scales, averaged 38% during resting breathing (1) and 21% during CO_2 -induced hyperpnea (2). Similarly, vibration of the parasternal region has been shown to reduce the sense of effort in healthy subjects breathing CO_2 -enriched gas mixtures or breathing against inspiratory resistive loads (3, 4). Because high-frequency vibration is a potent stimulus of spindle primary endings in limb muscles (5, 6), this beneficial effect on dyspnea has been primarily attributed to increased afferent information from the intercostal muscles (1–4). The actual effects of rib cage vibration on the respiratory muscles, however, are largely unknown.

To approach this problem, we have initially examined the response to vibration of the inspiratory intercostal muscles in a group of anesthetized dogs (7). When vibration was applied during hyperventilation-induced apnea, the internal intercostal muscles of the parasternal region (the so-called parasternal intercostals) showed occasional, low-amplitude electrical activity. In contrast, a prominent activity was consistently re-

corded from the external intercostals in the rostral segments, including when the vibrator was applied to distant areas of the rib cage. Also, when vibration was applied during the inspiratory phase of the breathing cycle, there was a substantial increase in external intercostal inspiratory activity but no alteration in parasternal intercostal inspiratory activity (7). Thus the external intercostals are much more sensitive to vibration than the parasternal intercostals, and this difference is fully consistent with the known difference in spindle density between the two muscle groups. Indeed, histologic studies of intercostal muscles in cats (8) and electrophysiologic studies in dogs (9, 10) have clearly established that the external intercostals are abundantly supplied with muscle spindles and that the parasternal intercostals are poorly endowed.

In the dog, however, the external intercostals in the rostral interspaces cause a cranial displacement of the ribs and an increase in lung volume when they contract alone (11, 12), yet the increased inspiratory activity recorded from these muscles during rib cage vibration was not accompanied by any increase in tidal volume (V_T) (7). The hypothesis was raised, therefore, that rib cage vibration produces concomitant contraction of the internal interosseous intercostals situated in the caudal portion of the rib cage (7). As the external intercostals, these muscles in the cat are abundantly supplied with muscle spindles (8). In addition, they have a clear-cut expiratory action on the lung (12). Consequently, if these muscles contracted in response to vibration, they might balance the increased external intercostal activity and maintain V_T constant.

METHODS

The experiments were carried out on 13 adult mongrel dogs (13 to 25 kg) anesthetized with pentobarbital sodium (initial dose, 25 mg/kg intravenously). The animals were placed in the supine posture and intubated with a cuffed endotracheal tube, and a venous cannula was inserted in the forelimb to give maintenance doses of anesthetic. A catheter was also inserted in the femoral artery to monitor blood pressure and sample arterial blood periodically for blood gas analysis. The rib cage and intercostal muscles were exposed on the right side of the chest from the first through the tenth rib by deflection of the skin and underlying muscle layers, and the external intercostal muscle in the eighth interspace was severed from the costochondral junction ventrally to the angle of the rib dorsally to expose the internal intercostal muscle. Two experimental protocols were then followed.

Experiment 1

The electrical and mechanical response to vibration of the internal intercostal muscles was first studied in the absence of central respiratory drive in six animals. The electromyogram (EMG) of the muscle thus exposed was obtained with a pair of silver hook electrodes spaced 3 to 4 mm apart, and the changes in muscle length were measured with a pair of piezoelectric crystals (2 mm diameter) implanted 6 to 10 mm apart in a well-identified muscle bundle and connected to a sonomicrometer (Triton Technology, San Diego, CA). Detailed descriptions of this technique have appeared in previous reports (13, 14). Implantation of the EMG electrodes and crystals was made 10 mm apart midway between the angle of the ribs and the costochondral junctions. The EMG signal was processed with an amplifier (model 830/1;

(Received in original form April 17, 2000 and in revised form August 23, 2000)

Supported in part by a research grant from the Brussels School of Medicine.

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Am J Respir Crit Care Med Vol 163, pp 49–54, 2001
Internet address: www.atsjournals.org

CWE Inc., Ardmore, PA), bandpass filtered below 100 and above 2,000 Hz, and rectified before its passage through a leaky integrator with a time constant of 0.2 s.

The animal was connected to a mechanical ventilator (Harvard pump, Chicago, IL), and after a 30-min recovery period, the ventilator was set to deliver about the normal V_T (approximately 300 ml) at about twice the normal rate of breathing (i.e., 30 strokes/min). When the animal was disconnected from the ventilator, it was therefore apneic for 30 to 40 s, at which time trains of vibration of 2 to 3 s duration were applied at intervals to the internal intercostal muscle in the eighth interspace, either 2 cm ventral or 2 cm dorsal to the costochondral junctions. The vibrations were delivered using a commercially available vibrator (model V. 101; LDS, Royston, UK) which was adjusted so that the amplitude of movement of its moving element and the frequency of vibration were 1.5 to 2.5 mm and 40 Hz, respectively. The vibrator was held manually throughout, perpendicular to the muscle, so that the site and the force of application could be well controlled, and it was maintained in contact with the muscle via a 30-cm-long Plexiglass tubing. This tubing, combined with the band width selected for the EMG signal, essentially eliminated all vibration artifacts. The area of contact between the vibrator and the muscle was approximately 1.8 cm².

At least five trials of 40-Hz vibrations were obtained in each animal; three trials were also performed in which the frequency of vibration was set at 10 and 80 Hz. In four animals, 40- and 80-Hz vibrations were subsequently applied to the parasternal intercostals in the fifth, sixth, and seventh interspaces and to the internal intercostal in the ninth interspace. When this procedure was completed, a differential pressure transducer (Validyne, Northridge, CA) was connected to a side port of the endotracheal tube to measure airway opening pressure, and the animal was mechanically ventilated with a self-inflating bag so as to lengthen passively the internal intercostal muscle investigated (12). Indeed, studies on relaxed limb muscles in cats (5, 15) and in humans (6) have clearly shown that the sensitivity to vibration of spindle primary endings increases during muscle stretch and decreases during muscle shortening; similar observations have been made on isolated cat muscle spindles (16, 17). Vibration (40 Hz) was thus initiated just before the onset of ventilation, and it was maintained constant for five or six consecutive cycles. Five trials were obtained in each animal, after which the EMG electrodes and piezoelectric crystals were transferred to the external intercostal muscle in the third interspace. Electrode and crystal implantation in this muscle was also made midway between the rib angles and the costochondral junctions. Vibrations of 40 Hz frequency were finally applied to the third parasternal intercostal muscle, first during apnea at resting end-expiration, and then during mechanical ventilation.

Experiment 2

Seven animals were next studied to assess the effects of vibration on the EMG activity of the internal intercostal muscles during spontaneous breathing. The site of electrode implantation and the procedure used to amplify and filter the EMG signal were similar to those described in Experiment 1. In four animals, however, no phasic expiratory activity could be recorded from the middle portion of the muscle during resting, room air breathing. Electrode implantation in these animals was therefore made in the dorsal portion of the muscle close to the angle of the ribs, i.e., in the portion that receives the greatest expiratory neural drive (18). In each animal, a pair of EMG electrodes was also inserted in the parasternal intercostal muscle in the third interspace; this signal was continuously monitored on a loudspeaker to provide the investigator with a precise phase reference.

The animal was allowed to recover for 30 min after instrumentation, after which it was connected to a heated Fleisch pneumotachograph and a differential pressure transducer for the measurement of lung volume. Every five to 10 breaths, 40-Hz vibrations were then applied to the internal intercostal muscle in the eighth interspace. The vibrations were delivered either during inspiration (out-of-phase) or during expiration (in-phase). At least 10 breaths with in-phase vibration were obtained in each animal; vibration in these breaths was initiated after the cessation of parasternal intercostal activity and removed before the onset of the next parasternal inspiratory burst. Ten breaths with out-of-phase vibration were also obtained, the vibration

being then initiated shortly before the onset of parasternal intercostal activity and removed immediately after the onset of the expiratory pause.

The animals in both experiments were maintained under light surgical anesthesia throughout the measurements. Supplementary doses of anesthetic (1 to 2 mg/kg) were given at regular intervals to ensure that there was no spontaneous movement of the forelimbs or hindlimbs, no flexor withdrawal of the forelimb, and no pupillary light reflex; the corneal reflex, however, was kept present. Rectal temperature was also maintained constant between 36 and 38° C with infrared lamps. At the end of the experiment, the animal was given a lethal dose of anesthetic (30 to 40 mg/kg intravenously).

Data Analysis

The changes in internal and external intercostal muscle length induced by vibration during apnea at resting end-expiration (Experiment 1) were measured peak-to-peak and expressed in micrometers. The changes in muscle length induced by mechanical inflation were also measured peak-to-peak. However, they were first expressed as percentage changes relative to the muscle length at end-expiration (LR), and they were then divided by the time to peak airway opening pressure to determine the rate of change in muscle length. The values thus obtained for each muscle were averaged over the different trials.

The electrical response of the muscles to vibration during apnea, during mechanical ventilation, and during inspiration (Experiment 2) was evaluated qualitatively on the basis of the raw and integrated EMG traces. On the other hand, the effects of in-phase vibration on internal intercostal EMG activity were quantified by measuring (in arbitrary units) the plateau of the integrated EMG signal during each vibrated breath and that recorded during the immediately preceding nonvibrated (control) breath. To allow comparison between the different animals, the expiratory EMG activity recorded during each vibrated breath was then expressed as a percentage of the activity recorded during the control breath. In each animal, these measurements were also averaged over the 10 trials.

Data were finally averaged for the animal group, and they are presented as means \pm SE. Statistical comparisons between the changes in internal and external intercostal muscle length during apnea and during mechanical inflation, and statistical assessment of the effects of in-phase vibration on expiratory EMG activity were made using Student's paired *t* tests; the criterion for statistical significance was taken as $p < 0.05$.

RESULTS

Effects of Vibration during Apnea at Resting End-expiration

Vibrating the internal intercostal muscle during apnea at resting end-expiration induced significant changes in muscle length in each animal. With the frequency of vibration set at 40 Hz, the peak-to-peak amplitude of these changes averaged $92 \pm 8 \mu\text{m}$, and these were not different from those recorded in the external intercostal muscle during vibration of the parasternal intercostal in the third interspace ($88 \pm 15 \mu\text{m}$). However, whereas all animals consistently demonstrated clear-cut EMG activity in the external intercostal, none showed any activity in the internal intercostal (Figure 1). This absence of activity persisted when the amplitude of movement of the vibrator was altered to change muscle length from approximately 20 μm to approximately 200 μm . It also persisted when the frequency of vibration was set at 10 and 80 Hz as well as when the vibrations were applied to the more rostral and caudal interspaces.

Effects of Vibration during Mechanical Ventilation

As anticipated (12), the internal intercostal in the eighth interspace lengthened during inflation in every animal and shortened during deflation. The amount of muscle lengthening thus induced at peak inflation in the six animals was $12.9 \pm 1.5\%$ LR, and the rate of muscle lengthening averaged $26.4 \pm 5.4\%$ LR/s. As shown in Figure 2A, a few discharges were recorded in the last part of inflation in one animal, and these were the

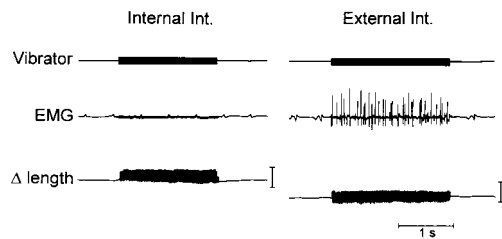


Figure 1. Response of the internal (eighth interspace) and external (third interspace) intercostal muscles to rib cage vibration during hyperventilation-induced apnea in a representative animal. Note that the two muscles showed similar changes in length (an upward deflection indicates muscle lengthening). However, electrical activity was recorded only from the external intercostal. Length calibration: 200 μm . Vibration frequency: 40 Hz.

result of the combined effects of muscle lengthening and vibration; indeed, when the same inflation was produced without vibration, the muscle remained electrically silent throughout (Figure 2B). In contrast, in five animals, vibrating the rib cage during inflation did not elicit any internal intercostal EMG activity (Figure 3A).

On the other hand, mechanical ventilation had a dramatic effect on the electrical response to vibration of the external intercostal muscle in all animals (Figure 3B). Thus, the muscle shortening induced by inflation was consistently associated with a progressive disappearance of EMG activity, and this activity came back whenever the (positive) pressure applied at the airway opening was released and the muscle was allowed to return to its end-expiratory length. Yet, although the changes in muscle length were in opposite direction to those observed in the internal intercostal, their magnitude was similar; for the six animals, the amount of external intercostal shortening at peak inflation was $12.0 \pm 1.5\%$ LR, and the rate of muscle shortening averaged $24.1 \pm 3.3\%$ LR/s.

Effects of Vibration during Breathing

Representative records of internal intercostal EMG activity during resting, control breathing and during out-of-phase vi-

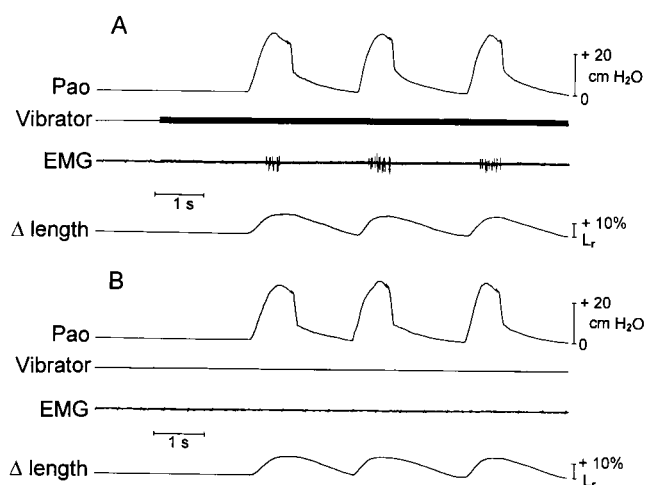


Figure 2. Response of the internal intercostal muscles to vibration during mechanical ventilation in the animal that had EMG activity at peak inflation (A). In the absence of vibration (B), the muscle remained electrically silent throughout, even though the changes in airway opening pressure (Pao) and the changes in muscle length were similar. The change in muscle length is expressed as a percentage change relative to LR.

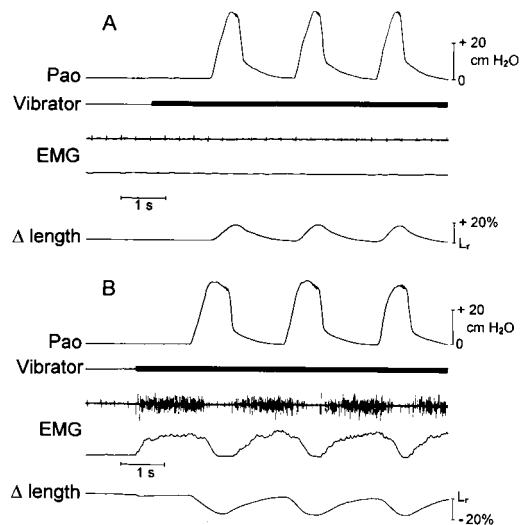


Figure 3. Effects of mechanical inflation on the response to vibration of the internal (A) and external (B) intercostal muscles in a representative animal. The raw and integrated EMG signals are shown for both muscles. The internal intercostal remained electrically silent throughout, including at peak inflation. In contrast, the external intercostal was active during vibration at resting end-expiration (left part of the record), and this activity was abolished at peak inflation. The changes in muscle length are expressed as percentage changes relative to LR.

bration of the eighth interspace are shown in Figure 4. The muscle showed phasic expiratory EMG activity in all animals, but it remained consistently silent during inspiration, regardless of whether vibration was applied or not. Similarly, no change occurred with in-phase vibration in any animal (Figure 5); for the animal group, expiratory EMG activity in the vibrated breaths averaged $100.6 \pm 0.9\%$ of the activity recorded in the control breaths (not significant [NS]). The peak inspiratory EMG activity recorded from the parasternal intercostal, inspiratory time, and V_T also remained unchanged.

DISCUSSION

High-frequency mechanical vibration, when applied to the belly of a relaxed limb muscle or to its tendon, is well known to produce an increased afferent input from spindle primary endings and to induce a reflex muscle contraction (5, 6). This muscle contraction is conventionally referred to as the tonic

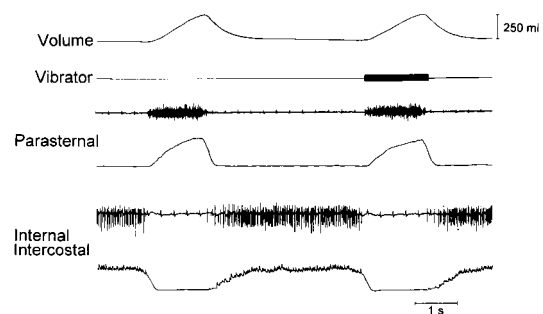


Figure 4. Electrical response of the internal intercostal muscle (eighth interspace) to out-of-phase vibration in a representative animal. The traces of lung volume and parasternal intercostal activity (third interspace) are also shown. The internal intercostal was electrically active during the expiratory phase of the breathing cycle but remained consistently silent during the inspiratory phase, including when vibration was applied.

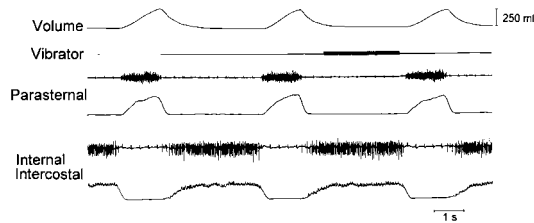


Figure 5. Response of the internal intercostal muscles to in-phase vibration in a representative animal. Same animal as in Figure 4. The phasic expiratory EMG activity recorded from the muscle remained unchanged during vibration.

vibration reflex (TVR), and one of the main results of the present studies is the demonstration that in the dog, vibration of the relaxed rib cage at end-expiration elicits a prominent TVR in the external intercostals but not in the internal interosseous intercostals (Figure 1). The changes in external and internal intercostal muscle length during vibration, however, were similar. Because these changes represent the combined effects of the displacement of the ribs and the muscle contraction, this similarity implies that the mechanical stimuli applied by the vibrator to the internal intercostals were at least equal to and probably greater than those applied to the external intercostals. Furthermore, when the respiratory system in our animals was passively inflated so as to shorten the external intercostals and to reduce the responsiveness to vibration of the corresponding spindle primary endings (5, 6, 15–17), the EMG activity caused by vibration decreased markedly and invariably (Figure 3). In contrast, when the internal intercostals were lengthened by inflation, they remained totally unresponsive to vibration in five of six animals even though the responsiveness to vibration of the spindle primary endings should be increased (5, 6, 15–17). The conclusion must be drawn, therefore, that the internal interosseous intercostals are much less sensitive to chest wall vibration than the external intercostals.

One explanation for this differential sensitivity to vibration could be a major difference in spindle density. Histologic studies of intercostal muscles have reported that in the cat, the external intercostal muscle in the third interspace contains 110 spindles per gram of dry muscle, whereas the internal interosseous intercostal in the eighth interspace has only 30 spindles per gram of dry muscle (8). However, the absolute number of spindles was approximately the same in the two muscles (18 and 24, respectively), presumably because the internal intercostal was thicker. Consequently, the total number of unitary excitatory postsynaptic potentials (EPSPs) induced by vibration in the internal and external intercostal α -motoneurons should also be about the same. In addition, the parasternal intercostals in cats contain a total of only 5 to 8 muscle spindles (8), and yet, in the dog, these muscles commonly show low-amplitude EMG activity during vibration at resting end-expiration (7). It would appear, therefore, that spindle density is not the major determining factor of the absence of TVR in the internal interosseous intercostals.

Another possibility would be that spindle primary endings in the internal interosseous intercostals have a higher threshold of activation than those in the external and parasternal intercostals; or, alternatively, the spindles in the canine internal interosseous intercostals might contain a higher proportion of secondary endings, which are less sensitive to vibration than primary endings (5, 6, 16). Previous studies in cats by Bolser and coworkers (19), however, have indicated that muscle spindles in the internal and external intercostals have a similar

sensitivity to vibration. According to these studies, a vibration amplitude of 100 μ m should, in fact, have triggered approximately 85% of the spindles in both the internal and the external intercostals in our animals, and the speculation was raised, therefore, that the internal intercostal α -motoneurons themselves have a higher activation threshold or a more negative membrane potential than the external intercostal α -motoneurons during hypocapnic apnea. If so, vibration might elicit the same number of EPSPs in the two sets of motoneurons and induce efferent α -motor activity only to the external intercostals.

Intracellular recordings from respiratory motoneurons in the thoracic spinal cord in cats have clearly established that during a normal respiratory cycle, the internal intercostal α -motoneurons depolarize during expiration to reach the threshold of activation and send efferent impulses to the muscles; this depolarizing phase is then followed by a hyperpolarizing phase during inspiration so that the motoneurons remain below the activation threshold and the muscles are kept silent (20, 21). These rhythmic fluctuations in membrane potential are of central origin and have, accordingly, been called central respiratory drive potentials (CRDPs). Also, earlier studies by Kirkwood and Sears (22) have shown that in the cat, the size of EPSPs from spindle afferents in internal intercostal motoneurons is modulated by the respiratory cycle, being greater during expiration than during inspiration. Consequently, if the absence of TVR in the internal intercostal muscles during hypocapnic apnea were the result of a higher activation threshold of the corresponding motoneurons, it would be expected that rib cage vibration would not elicit any internal intercostal EMG activity when applied during inspiration but would induce an increased EMG activity when applied during expiration.

In agreement with this prediction, no animal showed any internal intercostal activity during out-of-phase vibration (Figure 4). As a corollary, these muscles do not compensate for the increased external intercostal inspiratory activity during this procedure and do not play any role in maintaining V_T constant (7). However, all animals had phasic expiratory EMG activity in the internal intercostals, thus indicating that the α -motoneurons were well above their activation threshold during the expiratory phase of the breathing cycle, and yet in-phase vibration did not have any effect either (Figure 5). This result does not necessarily exclude a difference in membrane potential between the external and internal intercostal α -motoneurons during hypocapnic apnea but indicates that one or several additional factors play a major role.

The actual mechanism of the lack of response of the internal intercostals to in-phase vibration is uncertain, but two possible explanations come to mind. First, although vibration of a relaxed muscle stimulates exclusively or predominantly the spindle primary endings, it also stimulates the Golgi tendon organs when applied to a contracting muscle (5). Stimulation of tendon organs in the internal intercostals would, through autogenetic inhibition, reduce efferent activity to the muscles. Furthermore, recordings from medullary neurons in cats by Shannon and coworkers (23) have shown that stimulation of tendon organs in the external or internal intercostal muscles also causes inhibition of expiratory activities via supraspinal pathways. Although the peripheral (abdominal or intercostal) target of these medullary expiratory neurons was not readily identified, such supraspinal effects might further reduce the increased internal intercostal expiratory EMG activity that the spindle primary endings would produce otherwise. We have no data to exclude this possibility. However, Shannon and Bolser (19, 23) have also shown that a vibration amplitude of 90 to 100 μ m causes virtually exclusive activation of intercostal mus-

cle spindles and that these do not affect medullary expiratory or inspiratory activity. Indeed, as in our previous study (7), our animals did not show any alteration in the amplitude or timing of the parasternal intercostal inspiratory EMG activity during vibration, thus suggesting that the stimuli had little or no supraspinal effect. In addition, stimulation of Golgi tendon organs would not account for the fact that the internal intercostals did not respond to vibration during hypocapnic apnea. Finally, and perhaps more importantly, it would be surprising if the inhibitory effect of tendon organs and the excitatory effect of spindle primary endings canceled each other exactly. In the external intercostals, the latter effect is clearly dominant, such that the inspiratory EMG activity recorded from these muscles shows a twofold increase when the rib cage is vibrated during inspiration (7).

The second possible explanation for the lack of response of the internal intercostals to in-phase vibration would be related to the concomitant effect of vibration on the external intercostals. These muscles are very sensitive to vibration (7; see also Figures 1 and 3B), and although the muscle overlying the internal intercostal being studied was removed in our animals, the vibrations must have triggered external intercostal muscle spindles in adjacent interspaces. Also, it is well known that Ia afferent fibers from flexor limb muscles inhibit extensor α -motoneurons, and vice versa, so it is reasonable to conceive that stimulation of muscle spindles in external intercostals would cause reflex inhibition of their physiologic antagonists, the internal intercostal motoneurons. Sears (24) has been unable, in cats, to record inhibitory postsynaptic potentials (IPSPs) in internal intercostal motoneurons during electrical stimulation of Ia afferent fibers in external intercostal nerves. Furthermore, if in-phase vibration did elicit direct inhibition of internal intercostal motoneurons through stimulation of external intercostal muscle spindles, it would be expected that the procedure would induce a net decrease in internal intercostal expiratory EMG activity. Such a decrease was never observed in any animal (Figure 5), and this leads to the speculation that the inhibition occurs presynaptically, either along the Ia afferents from the internal intercostals or at the level of some interneurons. Indeed, it should be recalled that in the cat, the internal intercostal α -motoneurons in a given segment of the spinal cord receive not only monosynaptic excitation from the homonymous muscle spindles in the same segment but also polysynaptic excitation from homonymous muscle spindles in contiguous segments (20, 21); consequently, the response of these muscles to vibration depends to a large extent on polysynaptic pathways.

It is difficult to reconcile the current findings with the previous observation by Homma and coworkers (25) that vibration of the lower intercostal spaces in normal humans elicits internal intercostal EMG activity. Besides a species difference, two factors must be considered. First, the subjects studied by Homma and coworkers (25) were awake, whereas the animals of this study were anesthetized. It is well known that anesthesia, in particular barbiturate anesthesia, may depress spindle reflexes (26), so the possibility exists that the sensitivity to vibration of the internal intercostals in our animals was inadvertently reduced. It should be noted, however, that the external intercostals in these animals consistently showed a prominent TVR during apnea (Figure 1) and were very sensitive to changes in muscle length (Figure 3B). Furthermore, it is particularly striking that the internal intercostal activity recorded by Homma and coworkers (25) was continuous, independent of the phase of the respiratory cycle (that is, it was similar regardless of whether the α -motoneurons were depolarized or hyperpolarized). These investigators also observed

a concomitant, marked reduction in the phasic inspiratory EMG activity recorded from the parasternal intercostals in the upper interspaces (see Figure 2 in Reference 25), and this suggests that the perception of vibration in the rib cage or the abdomen may have been a key factor in these subjects. In the current study, all potential conscious reactions to vibration were eliminated.

The second important difference between the study of Homma and coworkers (25) and the current studies relates to the technique of electrode insertion and, with it, to the selectivity of EMG recording. Because our animals were anesthetized, the internal intercostal electrodes were implanted under direct vision; all the overlying muscles were deflected, so the risk of cross-contamination was eliminated. On the other hand, Homma and coworkers (25) inserted concentric needle electrodes transcutaneously. The internal intercostal electrode therefore passed through first the abdominal external oblique, then the external intercostal, and the activity thus recorded may have originated from either one of these two muscles, rather than the internal intercostal. Indeed, with the vibrator applied to the skin, a TVR was probably induced in the external intercostals.

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