Inspiratory and expiratory patterns of the pectoralis major muscle during pulmonary defensive reflexes

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Bolser, Donald C., and Paul J. Reier. Inspiratory and expiratory patterns of the pectoralis major muscle during pulmonary defensive reflexes. J. Appl. Physiol. 85(5): 1786-1792, 1998.—Experiments were conducted to determine the discharge pattern of the pectoralis major muscle during pulmonary defensive reflexes in anesthetized cats (n = 15). Coughs and expiration reflexes were elicited by mechanical stimulation of the intrathoracic trachea or larynx. Augmented breaths occurred spontaneously or were evoked by the same mechanical stimuli. Electromyograms (EMGs) were recorded from the diaphragm, rectus abdominis, and pectoralis major muscles. During augmented breaths, the pectoralis major had inspiratory EMG activity similar to that of the diaphragm, but during expiration reflexes the pectoralis major also had purely expiratory EMG activity similar to the rectus abdominis. During tracheobronchial cough, the pectoralis major had an inspiratory pattern similar to that of the diaphragm in 10 animals, an expiratory pattern similar to that of the rectus abdominis in 3 animals, and a biphasic pattern in 2 animals. The pectoralis major was active during both the inspiratory and expiratory phases during laryngeal cough. We conclude that, in contrast to the diaphragm or both the inspiratory and expiratory phases during laryngeal pattern in 2 animals. The pectoralis major was active during that of the rectus abdominis. During tracheobronchial cough, the pectoralis major also had purely expiratory EMG activity similar to the diaphragm, but during expiration reflexes the pectoralis major had inspiratory EMG activity similar to that of the pectoralis muscles during cough in intact animals. It is unknown whether the pectoralis major muscle contributes to the production of cough under normal conditions or may only participate in the production of this reflex after SCI (functional plasticity). Thus the present study was carried out to test the hypothesis that the pectoralis major muscle would have increased activity during cough in spinally intact animals. Our results indicate that this muscle participates in cough, as well as in other inspiratory and expiratory pulmonary defensive reflexes, in animals with intact spinal cords.

PULMONARY COMPLICATIONS are the leading cause of morbidity and death in patients with cervical spinal cord injuries (SCIs) (25). The main cause of this increased pulmonary morbidity is ineffective airway clearance, primarily due to an impaired cough reflex (25). The cough reflex is the primary mechanism for the clearance of mucus and foreign matter from the upper respiratory tract (2). The mechanism by which cough clears the upper airway is the generation of large expiratory airflows (18). Several methods have been developed to enhance expiratory airflow during cough in patients with SCIs (14). However, these methods only yield minimal increases in expiratory airflow during cough (14). There are other promising approaches to enhancing the effectiveness of pulmonary defensive reflexes, such as intraspinal electrical stimulation of abdominal motoneurons (8). However, progress in this area has been impeded by a lack of understanding of the underlying physiology and muscle motor patterning during cough.

Cervical SCI typically leads to an impaired cough reflex due to reduced motor drive to thoracic and lumbar expiratory spinal motoneurons (7, 26). Patients with lower cervical spinal involvement (C5–C7) can inspire via active control of the diaphragm and can produce weak expiratory efforts during cough (7, 9, 26). These patients have no contractile activity of their abdominal muscles during cough; rather, expulsion is accomplished through an active process involving contraction of the clavicular portion of the pectoralis major during the compressive and early expulsive phases of cough (7, 9, 26). Under these conditions, the action of this muscle compresses the upper chest to drive compression and expulsion (7, 9, 26). This pattern of activity of the pectoralis major during cough cannot be predicted from previous reports that this and other upper chest muscles have inspiratory activity during breathing (5), although electrical stimulation of this muscle in the dog can elicit either inspiratory or expiratory effects on chest wall mechanics (23). Furthermore, there have been no reports of the activity patterns of the pectoralis muscles during cough in intact animals. It is unknown whether the pectoralis major muscle contributes to the production of cough under normal conditions or may only participate in the production of this reflex after SCI (functional plasticity). Thus the present study was carried out to test the hypothesis that the pectoralis major muscle would have increased activity during cough in spina lingual isolated animals. Our results indicate that this muscle participates in cough, as well as in other inspiratory and expiratory pulmonary defensive reflexes, in animals with intact spinal cords.

METHODS

Cats (n = 15, weight 2.5–5.0 kg) were anesthetized with pentobarbital sodium (35 mg/kg iv) and given supplemental anesthetic as necessary. Atropine sulfate (1.0 mg/kg iv) was administered to block reflex tracheal secretions. Catheters were placed in a femoral artery and vein for monitoring blood pressure and administering drugs, respectively. A tracheal cannula was inserted through an incision at the fourth tracheal segment, and the animals were allowed to breathe spontaneously.

Bipolar stainless steel wire electrodes were placed in the diaphragm, pectoralis major, and rectus abdominis muscles according to the technique of Basmajian and Stecko (1). All electrodes were placed ∼4-mm apart in 5 animals and ∼1-mm apart in 10 animals. For the diaphragm, a small midline incision was made in the abdominal body wall just caudal to the xiphoid process and the electrodes were placed in the inferior surface of this muscle. The diaphragm electrodes were sealed in place with cyanoacrylate, and the incision was closed. For the rectus abdominis muscle, the electrodes were placed through a small incision in the skin ∼7 cm caudal to the xiphoid process and ∼2 cm lateral to the midline. An incision in the skin overlying the T1 intercostal space was made, and electrodes were superficially placed in the pectoralis major muscle ∼3 cm lateral to the midline and 2 cm caudal to the pectoantebrachialis muscle (13). The pectoantebrachialis muscle has no homolog in humans (13).
In each animal, an incision in the pectoralis muscle was made at T7, and a plastic sheet (8 × 5 cm) was placed underneath the pectoralis major muscle rostral to this site. This procedure electrically isolated the pectoralis major muscle so that EMGs from underlying muscles did not contaminate the pectoralis EMG.

EMGs from these muscles were amplified, band-pass filtered (0.1–5 kHz), monitored on an oscilloscope, and integrated with a resistance-capacitance circuit (100-ms time constant). These signals were displayed along with blood pressure on a chart recorder.

Cough is characterized by coordinated bursts of activity in inspiratory and expiratory muscles (18). Cough was defined as a burst of EMG activity in the diaphragm immediately followed by a burst of EMG activity in the rectus abdominis muscle (3). This definition differentiates augmented breaths, the aspiration reflex, and the expiration reflex from cough (28–30, 32). Augmented breaths were defined as a large EMG burst in the diaphragm with no subsequent activity in the diaphragm (26). Augmented breaths were defined as a large EMG burst in the diaphragm with no subsequent activity in expiratory muscles (32).

Cough, expiration reflexes, or augmented breaths were elicited by mechanical stimulation of the intrathoracic trachea or larynx with a small length of flexible plastic tubing for 10 s per stimulus episode (3). The intrathoracic trachea was accessed via the tracheal cannula, and the larynx was stimulated by inserting the plastic tubing 1–2 in. rostrally in the trachea.

All values are expressed as means ± SE. Statistical differences between means were evaluated with the Student’s t-test or one-way analysis of variance. Burst amplitudes of EMGs were expressed as a percentage of the largest amplitude burst of that particular EMG in each animal (4, 32). Relationships between normalized burst amplitudes of the EMGs were evaluated by linear regression analysis. Differences were considered significant if P < 0.05.

RESULTS

Coughs (n = 1,254 total, 1,155 tracheobronchial, and 99 laryngeal), expiration reflexes (n = 75), and augmented breaths (n = 43) were elicited in every animal. Augmented breaths were observed as spontaneous events or after mechanical stimulation of the larynx or trachea. Expiration reflexes were elicited during laryngeal stimulation. Coughs were elicited during either laryngeal or tracheobronchial stimulation.

The pectoralis major EMG was normally silent during eupneic breathing but was active during either purely inspiratory reflexes, such as augmented breaths, or purely expiratory reflexes, such as the expiration reflex (Fig. 1). Furthermore, the pectoralis major was active during cough, a reflex with both inspiratory and expiratory components (Figs. 2 and 3).

Augmented breaths. Diaphragm and pectoralis major EMG activity increased during augmented breaths (Fig. 1). The average increase in diaphragm EMG during augmented breaths was not different (59 ± 1% of the maximum diaphragm EMG burst) from the average increase in EMG burst amplitude of the pectoralis major (51 ± 10% of the maximum pectoralis major EMG burst; P < 0.25). Regression analysis indicated that there was no correlation between the peak burst amplitudes of the diaphragm and pectoralis major EMGs during augmented breaths (r = 0.13; slope = 0.15 ± 0.19; P < 0.45).

Expiration reflexes. Rectus abdominis and pectoralis major EMG activity increased during expiration reflexes (Fig. 1). The average increase in rectus abdominis EMG amplitude (32 ± 2% of the maximum rectus abdominis EMG burst) was not different from the increase in pectoralis major EMG amplitude (34 ± 2% of the maximum pectoralis major EMG burst; P < 0.3) during expiration reflexes. However, the average increase in pectoralis major EMG during expiration reflexes (34 ± 2% of the maximum pectoralis major EMG burst) was not as great as the increase in the EMG of this muscle during augmented breaths (65 ± 2% of the maximum pectoralis major EMG burst; P < 0.001). There was a weak correlation between the peak

Fig. 1. Example of discharge patterns of pectoralis major muscle EMG during an augmented breath and an expiration reflex in the same animal. Augmented breath is identified by a large diaphragm burst in the absence of rectus abdominis activity. Conversely, expiration reflex is characterized by a rectus abdominis burst without an increase in diaphragm activity. Pectoralis major is active during both of these reflexes. DIA, diaphragm; RA, rectus abdominis; P, pectoralis major.
burst amplitudes of the rectus abdominis and pectoralis major EMGs during expiration reflexes (r = 0.48; slope = 0.42 ± 0.1; P < 0.001).

Tracheobronchial cough. The burst pattern of the pectoralis major EMG during tracheobronchial cough had either an inspiratory pattern (n = 10 animals), an expiratory pattern (n = 3 animals), or a biphasic pattern (n = 2). In animals with an inspiratory pattern, the pectoralis major EMG increased during cough in a pattern similar to that of the diaphragm EMG (Fig. 2). In animals with an expiratory pattern, the pectoralis major increased during cough in a pattern similar to that of the rectus abdominis EMG (Fig. 3). In the animals with a biphasic pattern, the pectoralis major EMG increased during both the inspiratory and expiratory phases of cough, with a transient decrease in EMG activity at the phase transition.

The average EMG amplitudes of the diaphragm (52 ± 5% of the maximum diaphragm EMG burst), rectus abdominis (59 ± 4% of the maximum rectus abdominis EMG burst), and pectoralis major (58 ± 4% of the maximum pectoralis major EMG burst) during tracheobronchial cough were not significantly different from one another (P < 0.7). The average EMG ampli-

Fig. 2. Example of inspiratory discharge pattern of pectoralis major muscle EMG during cough. Dotted lines, end of inspiratory phase. During tracheobronchial cough, pectoralis major EMG activity peaks with diaphragm EMG activity. During laryngeal cough, pectoralis major EMG activity also peaks with diaphragm EMG activity, but more of pectoralis major EMG burst overlaps rectus abdominis EMG burst.

Fig. 3. Example of expiratory discharge pattern of pectoralis major muscle EMG during cough. Dotted lines, end of inspiratory phase. During tracheobronchial cough, there is little evidence of pectoralis major EMG activity when diaphragm is active. Pectoralis major EMG burst is coincident with rectus abdominis burst. During laryngeal cough, peak of pectoralis major EMG burst is coincident with that of rectus abdominis burst. However, unlike tracheobronchial cough, considerable pectoralis major EMG activity occurs during inspiratory phase.
tude of the pectoralis major muscle during tracheobronchial cough was not significantly different from the average EMG amplitude for this muscle during augmented breaths ($P < 0.3$). The average EMG amplitude for the diaphragm during augmented breaths was not different from the EMG amplitude of this muscle during tracheobronchial cough ($P < 0.11$). The average EMG amplitudes for the rectus abdominis and the pectoralis major muscles during tracheobronchial cough were significantly greater than the EMG amplitudes of these muscles during the expiration reflex ($P < 0.01$ and $P < 0.005$, respectively).

The peak EMG amplitudes of the diaphragm, rectus abdominis, and pectoralis major muscles were moderately correlated during tracheobronchial cough. Significant linear relationships with positive slopes existed between diaphragm, rectus abdominis, and pectoralis major EMG amplitudes during tracheobronchial cough. The average regression coefficient was $0.43 \pm 0.07$ for diaphragm-rectus abdominis, $0.59 \pm 0.06$ for diaphragm-pectoralis major, and $0.55 \pm 0.08$ for rectus abdominis-pectoralis major relationships. The average slope for each relationship was $0.57 \pm 0.15$ for diaphragm-rectus abdominis, $0.60 \pm 0.12$ for diaphragm-pectoralis major, and $0.53 \pm 0.10$ for rectus abdominis-pectoralis major relationships. An example of these relationships from a single animal during tracheobronchial cough is shown in Fig. 4.

Laryngeal cough. During laryngeal cough, the discharge pattern of the pectoralis major was not always consistent with the discharge pattern of this muscle during tracheobronchial cough. In animals with either inspiratory (Fig. 2) or expiratory (Fig. 3) discharge patterns, the pectoralis major had a phase-spanning burst pattern. That is, the EMG of this muscle was active during both the inspiratory and expiratory phases of laryngeal cough. Although the pectoralis major EMG most often peaked at the transition between the inspiratory and expiratory phases of laryngeal cough, differing patterns could be observed in successive laryngeal coughs in the same animal. For example, Fig. 3 shows three successive laryngeal coughs in which the pectoralis major EMG peaked in different phases. During the second laryngeal cough the pectoralis major EMG peaked during the transition from the inspiratory to expiratory phase, but in the first and third laryngeal coughs of the series this EMG peaked in the expiratory phase.

The average EMG amplitude of the diaphragm ($57 \pm 4\%$ of the maximum diaphragm EMG burst) was not significantly different from that of rectus abdominis ($54 \pm 4\%$ of the maximum rectus abdominis EMG burst; $P < 0.3$) during laryngeal cough. However, the pectoralis major EMG amplitude ($69 \pm 6\%$ of the maximum pectoralis EMG burst) was significantly greater than the EMG amplitudes of the diaphragm ($P < 0.04$) and rectus abdominis muscles ($P < 0.02$) during laryngeal cough. The EMG amplitudes for the diaphragm and rectus abdominis muscles during laryngeal cough were not significantly different from the EMG amplitudes of these muscles during tracheobronchial cough. By contrast, the average EMG amplitude of the pectoralis major muscle was significantly greater during laryngeal cough than during tracheobronchial cough ($P < 0.05$).

No attempt was made to determine the relationships among the peak EMG amplitudes of the pectoralis major, diaphragm, and rectus abdominis muscles because of the cough-to-cough variation in timing of the peak in pectoralis major EMG activity during laryngeal cough.

**DISCUSSION**

The major finding of this study is that the pectoralis major participates in both inspiratory and expiratory pulmonary defensive reflexes. This muscle is active...
during a purely inspiratory defensive reflex, the augmented breath; a purely expiratory defensive reflex, the expiration reflex; and a defensive reflex with both inspiratory and expiratory components, the cough reflex. During cough elicited from the tracheobronchial region, the pectoralis major in a given animal can have either inspiratory- or expiratory-phased activity. During laryngeal cough, the pectoralis major burst spans both the inspiratory and expiratory phases.

Contribution of the pectoralis major to the EMG responses during pulmonary defensive reflexes. The EMG responses observed in this study were specific to the pectoralis major muscle. The EMG electrodes were placed superficially, and this muscle is large (at least 5–10 mm thick). It is unlikely that the EMG responses that we recorded were contaminated by EMGs from underlying muscles (such as the scalenes, transversus costarum, rectus thoracis, or intercostals) because these muscles were electrically isolated from the pectoralis major with large segments of plastic in all animals.

Response of the pectoralis major during augmented breaths. Augmented breaths consist of an increase in motor drive to inspiratory muscles that results in a severalfold increase in tidal volume (22, 31). This increased tidal volume is thought to prevent atelectasis (22). Well-known inspiratory muscles, such as the diaphragm, external intercostals, and genioglossus, all have been shown to participate in the production of augmented breaths (31, 33). Our results indicate that the pectoralis major also participates in the production of augmented breaths. The fact that this muscle was active during augmented breaths indicates that the pectoralis major receives inspiratory motor drive. The source of this inspiratory motor drive is probably the brain stem, although reflex activation of this muscle from sensory afferents located in other inspiratory muscles cannot be ruled out. Indeed, we found no correlation between diaphragm and pectoralis major burst amplitudes during augmented breaths, suggesting that the discharge patterns of these two muscles are not simply due to a corollary output of the same pattern generator. However, van Lunteren et al. (33) have observed that the magnitudes of activation of upper and lower intercostal muscles or costal and crural regions of the diaphragm during augmented breaths are different. These investigators suggested that their findings could be explained by nonhomogeneous distribution of motor drive to different motoneuron pools or by differences in the organization of different motoneuron pools. These alternative explanations could also account for our observations as well.

Response of the pectoralis major during expiration reflexes. The expiration reflex is purely an expiratory defensive response that functions to prevent irritants or foreign material from passing through the glottis and into the lungs (15, 16, 27). It is usually elicited from the larynx and consists of a sudden increase in expiratory muscle activity, without a preceding inspiratory effort (15, 16, 27). The resultant expiratory airflow will remove irritating objects from the glottal area, maintaining a patent airway.

We found that the pectoralis major participates in the expiration reflex. It has largely been assumed that abdominal expiratory muscles were responsible for the expiration reflex. Indeed, lumbar nerve activity increases during expiration reflexes (27). However, the extent to which individual expiratory muscles are active during this reflex is unknown. Our findings indicate that the expiratory motor response during this reflex includes a large EMG burst in the rectus abdominis muscle, as well as in the pectoralis major. The extent to which other muscles participate in the expiration reflex is unknown, although Korpás and Tomori (18) showed evidence that midthoracic external intercostal muscles are active during expiration reflexes.

There was a weak relationship between the rectus abdominis and pectoralis major burst amplitudes during expiration reflexes. As discussed above, the distribution of expiratory motor drive to different muscles during expiration reflexes is poorly understood. Furthermore, the extent to which spinal reflexes may modify descending motor drive to different motoneuron pools is unknown. This observation may be due to a corollary descending expiratory drive to both the rectus abdominis and pectoralis major motoneuron pools or to reflex activation of one muscle by sensory afferents in another during expiration reflexes or both.

Although it is very similar to cough (without the inspiratory component), the expiration reflex is considered to be a different phenomenon. For example, cough, but not the expiration reflex, can be inhibited by codeine (15). The expiration reflex in cats also can be observed early in postnatal life, when cough is not yet present (17). Furthermore, the EMG burst amplitudes of the rectus abdominis and the pectoralis major muscles were larger during cough than during the expiration reflex. This observation is consistent with a previous report that intrapleural pressure changes during cough are greater than during the expiration reflex (15). Therefore, the regulation of pectoralis major activity during the expiration reflex probably represents a different control mechanism from that responsible for modulation of pectoralis major activity during cough.

Response of the pectoralis major during cough. Our findings indicate that the pectoralis major is active during cough under normal conditions in an animal model. The participation of respiratory muscles such as the diaphragm, intercostals, abdominals, and laryngeals in cough is well known (18). However, the participation of other accessory muscles of respiration is less well understood. The pectoralis major is active during voluntary cough in most tetraplegics and some normal subjects (9). Therefore, the increased activity of this muscle during cough is found in more than one species and probably represents an important component of this reflex.

The activity pattern of the pectoralis major EMG during tracheobronchial cough was consistent in a given animal, and the population of animals fell into three groups in which the activity of this muscle was primarily inspiratory, expiratory, or biphasic. During laryngeal cough, the pectoralis major was consistently
active throughout the inspiratory and expiratory phases in every animal, but the EMG of this muscle often peaked at different times in the cough cycle in a given animal. The reason(s) for this difference in activation patterns is unknown. In our experiments, expiratory airflow was directed out of the tracheal cannula, so there was no mechanical compression phase during cough. It is unlikely that the motor pattern for tracheobronchial and/or laryngeal cough would be different in a preparation in which the larynx participates in compression because 1) as Sant’Ambrogio and co-workers (24) have reported, the motor pattern for laryngeal muscles during tracheobronchial cough is not altered when the larynx is bypassed or denervated during cough, 2) humans with tracheostomies can still cough effectively (20), and 3) the larynx was bypassed in every animal and during tracheobronchial and laryngeal cough within a given animal. Therefore, it is unlikely that laryngeal bypass can account for the inter- and intra-animal differences in the motor pattern of the pectoralis major during cough.

Changes in forelimb position have been shown to alter the mechanical action of the pectoralis major muscle from inspiratory to expiratory in the dog (23). In the present experiments, the forelimbs were fixed in an extended position in all animals. Therefore, it is unlikely that forelimb position can account for either the inter-animal variability of pectoralis major muscle activity during tracheobronchial cough or the inspiratory and expiratory activity of the pectoralis major muscle during augmented breaths and expiration reflexes within individual animals. Furthermore, we observed in preliminary experiments that the pectoralis major muscle discharge pattern during tracheobronchial cough was unaffected by movement of the forelimbs from extended to flexed positions.

It is conceivable that the differences in the motor pattern of the pectoralis major between laryngeal and tracheobronchial cough are due to stimulation of sensory afferents from each region of the airway that can elicit multiple reflex responses. Mechanical stimuli applied to the intrathoracic trachea usually elicit augmented breaths and/or coughs (6, 18). Similar stimuli applied to the larynx can elicit cough, expiration reflexes, augmented breaths, apnea, and/or swallowing (18, 21, 34). Presumably, the central processing of laryngeal sensory information is sufficiently different from that mediating tracheobronchial reflexes to account for a different pectoralis major burst pattern during cough elicited from the two regions of the airway.

Functional relevance of the pectoralis major in pulmonary defensive reflexes. The pectoralis major muscle appears to participate in several different pulmonary defensive reflexes, and its activity pattern exhibits profound changes in a manner that is consistent with each specific reflex, or behavior, in which it participates. This situation contrasts with the regulation of the diaphragm, which also is active during several different pulmonary defensive reflexes. However, the diaphragm always has an inspiratory function and therefore is a monofunctional muscle. Similarly, abdominal expiratory muscles always have an expiratory function during pulmonary defensive reflexes. Their activity patterns do not change from inspiratory to expiratory during different reflexes (18). The differential responses of the pectoralis major muscle during different reflexes may represent different motor strategies involving several muscles acting in concert on the upper chest.

Electrical stimulation of the pectoralis muscles can have either an inspiratory or an expiratory action on the upper chest in anesthetized dogs (23). Although it is tempting to speculate about the inspiratory and expiratory EMG activity observed in the present study in terms of producing an inspiratory or expiratory mechanical effect on the chest wall during the different reflexes, we have no direct evidence that the EMG changes that we observed actually would have resulted in these sorts of alterations in chest wall mechanics.

Implications for mechanisms of cough production after SCI. On the basis of previous reports that the pectoralis major muscle was active during cough in humans with SCIs (7, 9, 10, 12), we speculated in the present study that this muscle would be active during cough in animals with intact spinal cords. Although one study (9) reported EMG activity of the pectoralis major muscle during cough in three of five normal subjects, the extent to which pectoralis major activity normally occurs during cough or is an adaptive response to SCI is unknown. Our findings indicate that pectoralis major activity is more likely a normal feature of the cough reflex. It is possible that the regulation of pectoralis major activity undergoes functional plasticity after SCI, resulting in an enhanced contribution of this muscle to cough effectiveness. Training of this muscle can increase expiratory reserve volume in tetraplegic subjects (11). The extent to which SCI may modify the control of pectoralis major motoneurons during cough is best addressed by further study in models of SCI.

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REFERENCES


