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RELATIONSHIP BETWEEN PARASTERNAL AND EXTERNAL INTERCOSTAL MUSCLE LENGTH AND LOAD COMPENSATORY RESPONSES IN DOGS

BY J. R. ROMANIUK*, G. SUPINSKI AND A. F. DIMARCO†

From the MetroHealth Medical Center, Pulmonary Division, 3395 Scranton Road, Cleveland, OH 44109, USA

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SUMMARY

1. The effects of tracheal occlusion on peak parasternal (PA) and external intercostal (EI) (3rd interspace) EMG activities were examined at different end-expiratory lung volumes both above and below functional reserve capacity (FCR) in anaesthetized, vagotomized and spontaneously breathing dogs.

2. Parasternal (PA) and external intercostal (EI) muscle lengths were monitored *in situ*. The difference in peak EMG activity between free and occluded breaths (test breaths) was related to the coincident peak change in intercostal muscle length (ΔL) for each muscle, respectively.

3. At FRC, tracheal occlusion resulted in compensatory augmentation of peak EI, but little change in peak PA EMG activities. At lung volumes below FRC, airway occlusion resulted in augmentation of both PA and EI activities. Responses to airway occlusion at lung volumes above FRC were variable. The magnitude and duration of these changes in EMG, however, could be linearly related to the value of ΔL. With $\Delta L = 0$, there was no change in peak EI or PA EMG; for values of $\Delta L < 0$, there was attenuation of EI and PA EMG; for $\Delta L > 0$, there was enhancement of EI and PA EMG activation.

4. The magnitude of the changes in EMG activity in response to tracheal occlusion was more prominent for the EI muscle compared to the PA, the latter of which are known to have much fewer muscle spindles than EI muscle.

5. Our results suggest that a difference in end-inspiratory muscle length between the control and occluded breaths is a stimulus for the intercostal response to applied loads implicating muscle spindles as the predominant receptor moderating these responses. We hypothesize that when $\Delta L = 0$, no change in EMG occurs since the spindles sense no change in muscle length. When $\Delta L < 0$ (i.e. peak muscle length during the occluded breath is shorter than control) muscle spindles would be disengaged, resulting in a disfacilitation of EMG activity. Where $\Delta L > 0$ (i.e. peak muscle length during the occluded breath is longer than control), muscle spindles are stimulated, resulting in enhancement of EMG activity.

* Visiting Professor from Polish Academy of Science, Medical Research Centre, Department of Neurophysiology, Warsaw, Poland.

† To whom all correspondence and reprint requests should be sent.

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6. Additional doses of Nembutal (20 mg), which produced significant changes in breathing pattern, did not affect the magnitude of the load compensatory responses.

INTRODUCTION

In vagotomized animals, airway occlusion at end-expiration results in an augmentation of inspiratory intercostal motor activity during the subsequent inspiratory effort (Critchlow & von Euler, 1963; Sears, 1964*a*, *b*; Corda, Eklund & von Euler, 1965). The precise mechanism of this phenomenon, also referred to as stretch reflex (cf. Glebovski, 1988), segmental autogenic facilitation or load compensatory reflex (cf. von Euler, 1973), is not clear. It is known that the reduction in inspiratory muscle shortening that occurs during contraction against a load such as airway occlusion or change in posture results in an increase in afferent discharge from muscle spindles (Critchlow & von Euler, 1963; Sears, 1964*a*; Hilaire, Nichols & Sears, 1983; Glebovski, 1988). It has been postulated that the consequent reflex increase in motoneurone output to the extrafusal intercostal muscle fibres results in an increased force of contraction to overcome the effects of the applied load and achieve the 'expected' or 'desired' degree of muscle shortening (Hammond, Merton & Sutton, 1956; Granit, 1975; DiMarco, Romaniuk & Supinski, 1990*a*). If this hypothesis is correct, the magnitude of intercostal facilitation during loaded breathing should be related to the difference between inspiratory shortening achieved during the contraction against the load and that which occurs during free breathing.

In the present investigation, we sought to test this hypothesis by evaluating the responses of the inspiratory intercostal muscles of the upper rib-cage to airway occlusion. As pointed out by von Euler (1973), these muscles provide a unique opportunity to study muscle spindle responses since they are activated phasically and reproducibly with each inspiration and are abundantly supplied with muscle spindles. Furthermore, recent developments allow for their length to be measured directly (Newman, Road, Bellemare, Clozel, Lavigne & Grassino, 1984; DiMarco, Romaniuk & Supinski, 1990*b*). The relationship between changes in end-inspiratory intercostal muscle length and peak intercostal EMG before and during airway occlusion, therefore, was examined. To achieve a variety of changes in muscle length, airway occlusion was applied over a wide range of lung volumes below and above FRC. The responses of the parasternal intercostal muscles, which have only few muscle spindles (Jung-Caillol & Duron, 1976), were compared with those of the external intercostal muscles which are known to be richly supplied with these receptors.

METHODS

Thirteen mongrel dogs, weight 15–20 kg, were studied in the supine posture. Anaesthesia was provided by the initial administration of 25 mg/kg pentobarbitone intravenously and supplemented with 20–40 mg, as needed. Anaesthetic level was monitored by the corneal reflex, which was left intact. A cuffed endotracheal tube was sutured into the trachea in the cervical region. Bilateral cervical vagotomy was performed in each animal.

Body temperature was maintained at $38 \pm 0{\cdot}5$ °C with a heating blanket. A cuffed endotracheal tube was placed in each animal. Catheters were placed in the femoral vein and artery for administration of intravenous fluids and anaesthetic, and to monitor arterial blood pressure,

respectively. Changes in end-tidal P_{CO_2} were monitored at the tracheal opening via a CO_2 analyser (Beckman LB-2). Arterial P_{O_2}, P_{CO_2} and pH were also measured intermittently (Arterial Blood Gas Analyzer, IL 813). Tidal volume was recorded by electrical integration of the flow signal from a pneumotachograph (Fleisch No. 1). Airway pressure was monitored by a pressure transducer (MP-45; Validyne Co., Northridge, CA, USA) connected to the airway opening.

Intercostal muscle length was assessed by sonomicrometry using piezoelectric crystals (Model No. 120, Triton Technology, San Diego, CA, USA) according to previously described techniques (Newman *et al.* 1984). Pairs of piezoelectric crystals, spaced 5–12 mm apart and oriented along the axis of muscle fibres, were sewn into a parasternal intercostal muscle and an external intercostal muscle, both in the 3rd intercostal space. Just proximal to the placement of the crystals, pairs of unipolar teflon-coated stainless-steel recording electrodes were inserted directly into the intercostal muscles to record electromyographic activities. Mass efferent activities of each muscle were amplified, rectified and processed by RC circuits containing third-order low-pass filters with a time constant of 100 ms (Charles Ward Enterprises, Ardmore, PA, USA) to provide moving averages of activity.

Protocol

In each animal, peak integrated EMG activity and change in intercostal muscle length were assessed during occluded inspiratory efforts at functional reserve capacity (FRC) and compared to that of the prior control free breath. Airway occlusion was always applied during the expiratory phase of the control breath. To achieve a wide range of differences in intercostal muscle length between occluded and unoccluded breaths, airway occlusion was also applied over a wide range of different pre-contractile muscle lengths. In each animal, changes in pre-contractile muscle length were accomplished by passive inflations and deflations with a volume syringe over a wide range of lung volumes between 0·3 l below and 1·0 l above FRC (mean number of manoeuvres was 17 ± 2; range, 10–35). After each change in lung volume, which was always performed during the expiratory phase of the control breath, the airway was occluded for a single breath. End-inspiratory intercostal muscle EMG and length during the occluded breaths were compared to the average of the same respective parameters during the preceding three breaths at FRC.

Regarding this methodology, both the change in lung volume as well as airway occlusion can potentially alter the level of neural drive during the occluded breath. The purpose of the present study, however, was to assess the effect of changes in intercostal muscle length, as the independent variable, on neuromuscular drive. Since passive changes in lung volume result in progressive changes in resting intercostal muscle length, lung volume changes as well as the effects of airway occlusion were reflected in the measured end-inspiratory length following airway occlusion. Furthermore, this method provided a very similar control for each test breath and allowed us to evaluate the effect of differences in end-inspiratory length over a wide range. It should also be noted that inspiratory rib movements during lung inflation are similar to those during spontaneous breathing (DaSilva, Sayers, Sears & Stagg, 1977).

In five animals, studies were repeated after bilateral section of the phrenic nerves in the cervical region. These studies were performed to (a) evaluate intercostal muscle responses at an increased level of neural drive and degree of inspiratory intercostal muscle shortening and (b) assess intercostal responses independent of the potential compounding effects of diaphragm contraction on intercostal muscle length changes.

Data analysis

The difference in inspiratory muscle shortening between occluded and free inspiratory efforts was used as an index of the difference between the 'expected' and actual degree of inspiratory shortening (see Fig. 1). Peak integrated EMG activity during the occluded breath was expressed as a percentage of the average of peak EMG of the three preceding free breaths. Statistical comparisons were made using the t test for two paired samples and linear regression analysis. A P value of less than 0·05 was considered significant.

RESULTS

The effects of tracheal occlusion at FRC on parasternal (PA) and external intercostal (EI) EMG are displayed for one animal in Fig. 1. While peak EI activity

increased in response to the occlusion, there was little change in peak PA EMG activity. As expected in a vagotomized animal, inspiratory time was unchanged by tracheal occlusion (Fig. 1*B*). Both PA and EI muscles shortened during the control inspiration while both muscles demonstrated a small degree of lengthening during the occluded breath. Similarly, in another example (Fig. 2*A*), tracheal occlusion resulted in no appreciable change in peak parasternal intercostal EMG whereas there was marked enhancement of external intercostal activation in response to airway

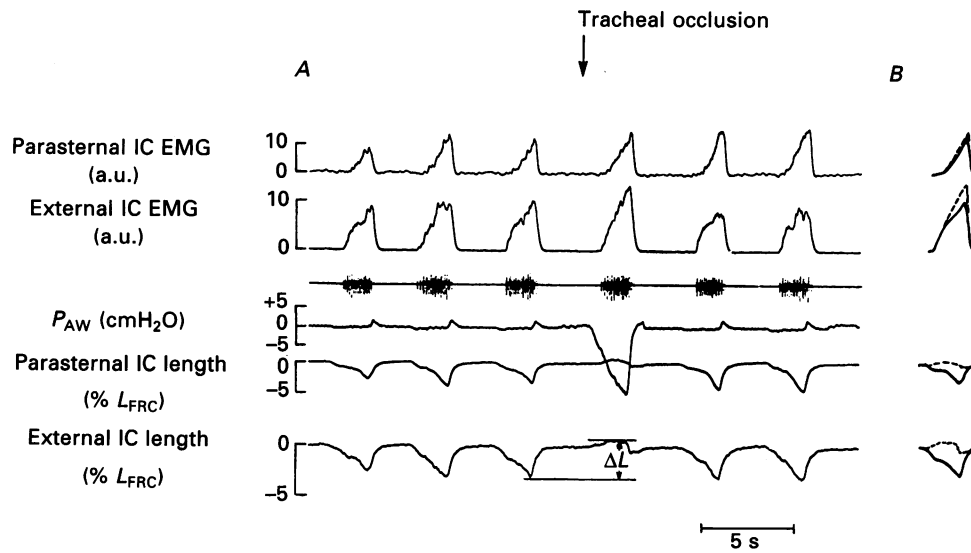

Fig. 1. *A*, effects of tracheal occlusion performed at FRC for a single breath on parasternal and external intercostal EMG and length. The method by which the difference in end-inspiratory muscle length between occluded and free inspiratory breaths (ΔL) was measured is shown. *B*, superimposed traces of integrated EMG activities and muscle length for occluded (dashed line) and preceding free breath (continuous line). Airway occlusion resulted in an increase in external intercostal activity and little change in peak parasternal activity compared to the previous unoccluded breaths. Both parasternal and external intercostals lengthened slightly during the occluded breaths. See text for further explanation. a.u. = arbitrary units; P_{AW} = airway pressure.

occlusion. During the occluded effort, parasternal length was biphasic whereas the external intercostal muscle lengthened. Bilateral phrenicotomy resulted in a marked enhancement of parasternal and external intercostal EMG with associated increases in the magnitude of their inspiratory shortening (Fig. 2*B*). Whereas both PA and EI often lengthened prior to phrenic section, they consistently shortened during airway occlusion after phrenicotomy. As shown in this animal, the response to airway occlusion following phrenicotomy was qualitatively similar to that observed pre-phrenicotomy, i.e. there was marked enhancement of external intercostal EMG but no change in parasternal EMG.

The effect of tracheal occlusion on PA and EI muscle EMG and length at different lung volumes is shown for one animal in Fig. 3. In response to tracheal occlusion at

FRC, peak EI EMG increased markedly, whereas there was a smaller increase in PA EMG. These changes were associated with a small degree of lengthening of both the PA and EI muscles during the occluded effort. With passive deflation (−200 ml), both PA and EI muscles passively lengthened. The subsequent inspiratory effort was

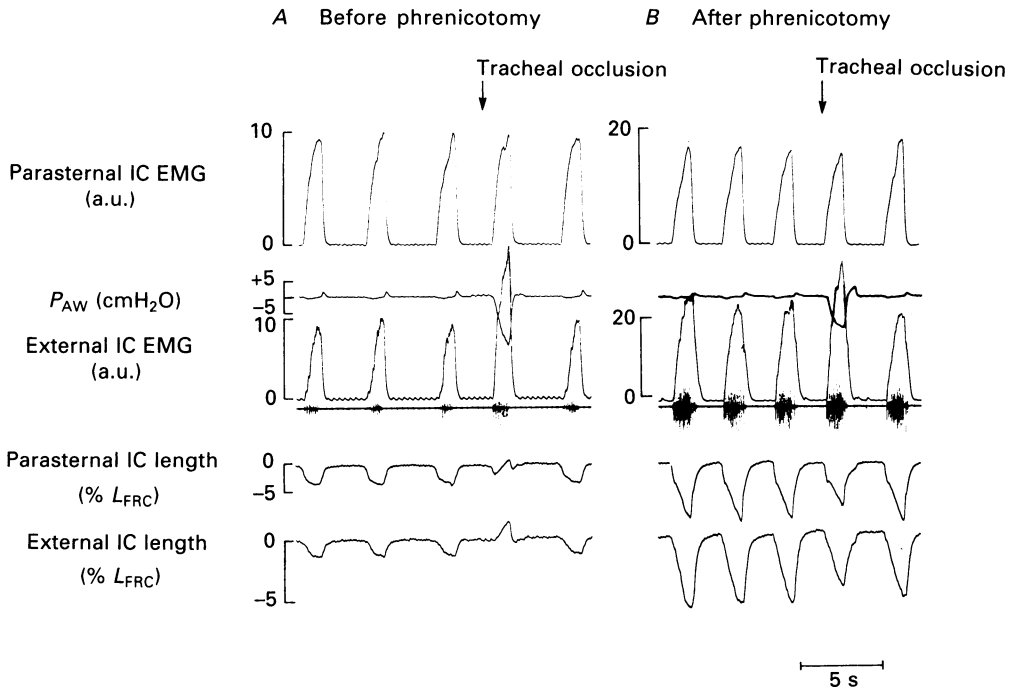

Fig. 2. Effects of tracheal occlusion at FRC for a single breath on parasternal and external intercostal EMG and length before and after phrenicotomy. The responses prior to phrenicotomy were similar to those described in Fig. 1. After phrenicotomy (*B*) parasternal and external intercostal muscle EMG increased substantially (note change in scale of arbitrary units, a.u.). This was associated with a marked increase in the degree of parasternal and external intercostal muscle shortening. Following phrenicotomy, tracheal occlusion resulted in enhancement of external intercostal EMG but little change in parasternal EMG. The parasternal and external intercostal muscle shortening was less following airway occlusion. See text for further explanation. P_{AW}, airway pressure.

associated with further lengthening of these muscles and marked increases in PA and EI activation. Lung inflation to +800 ml resulted in passive shortening of both the PA and EI muscles. The subsequent occluded inspiratory effort was associated with further muscle shortening and marked reductions in both peak PA and EI EMG compared to the previous free breath.

Very large inflations, usually greater than 1·0 l, often resulted (seven of thirteen animals) in a much greater initial enhancement of both parasternal and external intercostal activation as shown in Fig. 4. These EMG trajectories had a strikingly different pattern compared to the gradual rise in activity which peaked just prior to

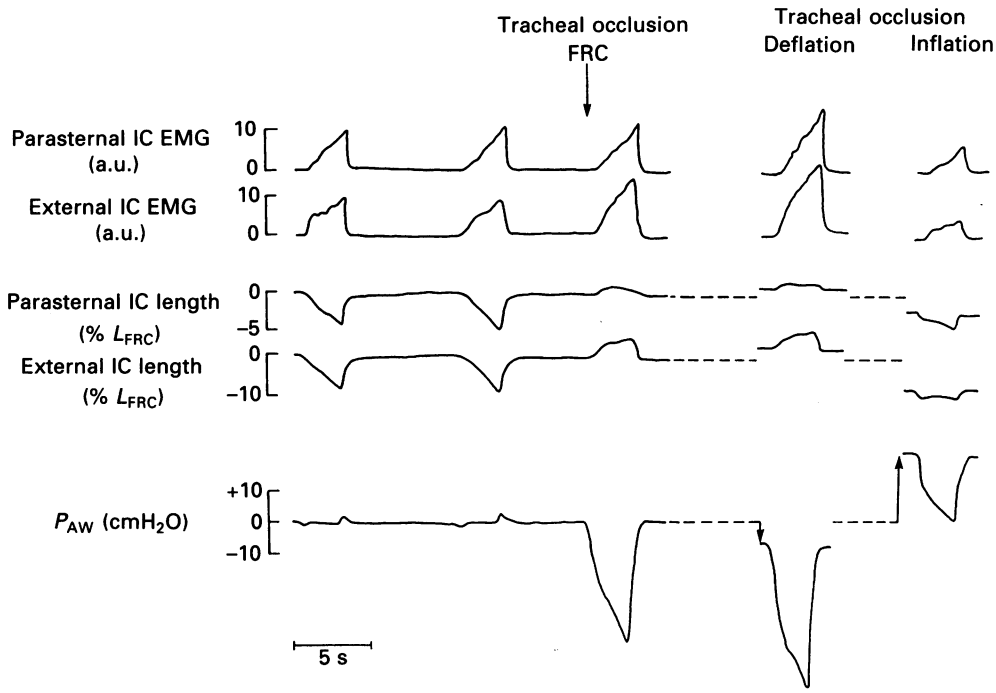

Fig. 3. Effect of tracheal occlusion at different lung volumes on parasternal and external intercostal EMG and muscle length. Airway occlusion was performed at FRC after deflation (−200 ml) and inflation (+800 ml) of the lungs. Deflation produced enhancement of reflex activation of intercostal muscles during tracheal occlusion while inflation produced diminution of their activity. See text for further explanation. a.u., arbitrary units; P_{AW} airway pressure.

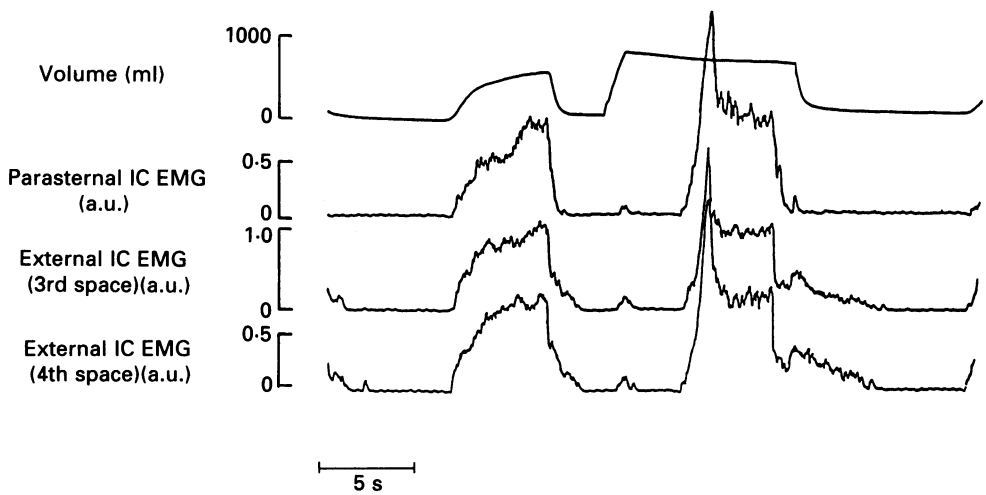

Fig. 4. Effect of large lung inflation on parasternal and external intercostal muscle EMG. With large inflations, an enhancement of intercostal activities was observed in early inspiration. See text for further explanation. a.u. arbitrary units.

inspiratory termination following smaller inflations. Since these effects were qualitatively very different from those occurring with smaller inflations, we limited our analysis to lung inflations below that which elicited this type of response.

The relationship between ΔL (the difference between the end-inspiratory length achieved during the occluded breath and that during the preceding free breath) and

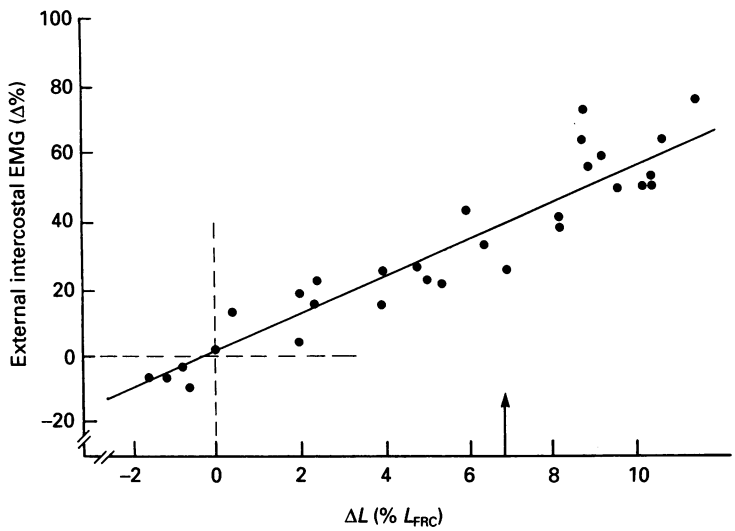

Fig. 5. Relationship between external intercostal muscle length change (expressed as percentage of resting length at FRC) and the percentage change in external intercostal muscle EMG during occluded inspiratory efforts at different lung volumes (●). Each point represents the comparison between a single pair of occluded and free breaths. This relationship could be fitted by a linear function with a correlation coefficient (r) of 0·94. The value of ΔL during tracheal occlusion at FRC is indicated by the arrow. ΔL decreased with inflation and increased with deflation.

the percentage change in peak EI EMG between these same breaths, is shown for one animal in Fig. 5. With deflation, there were increases in ΔL for the EI muscle and corresponding increases in the level of EI EMG activity. With inflation, there were decreases in ΔL and corresponding decreases in EMG activity. As shown by the dashed lines in Fig. 5 when end-inspiratory length was shorter during the occluded breath than that achieved during the preceding control (i.e. ΔL had a negative value), there was disfacilitation, i.e. peak intercostal EMG activity was less than control values. The relationship between ΔL and percentage change in EMG could be fitted by a linear function with a correlation coefficient in this animal of 0·94. The correlation coefficients, slope and Y-intercepts in each animal for both the PA and EI muscles are provided in Table 1. The relationship between EI ΔL and percentage change in EMG could be fitted by a linear function with a positive slope in each animal (mean correlation coefficient of 0·90). In those animals in which the effects of phrenicotomy were assessed ($n = 5$), a significant correlation was still demonstrated; however, the mean slope of the relationship decreased from 7·69 to 3·87 for EI muscle

($P < 0.05$). While the relationship between PA ΔL and percentage change in EMG could also be fitted by a linear function, the degree of correlation (0·74 for PA) and magnitude of the response (slope = 3·73) were significantly less ($P < 0.05$) than that for the EI muscle. In one animal, the correlation was very poor ($r = 0.34$) and the slope had a negative value. As with the EI response, the mean slope also fell

TABLE 1. Correlation coefficients, Y-intercepts and slopes of the relationships between changes in external and parasternal intercostal EMG and ΔL for each animal

	External			Parasternal		
	r	i	s	r	i	s
1	0·93	60·1	10·85	0·95	60·1	10·46
2	0·92	116·9	1·64	0·71	97·3	0·39
3	0·86	101·2	1·92	0·41	98·3	0·78
4	0·89	102·2	11·56	0·82	91·5	10·55
5	0·79	110·2	2·44	0·74	100·1	0·40
6	0·91	109·2	11·35	0·96	99·4	3·84
7	0·95	111·9	6·08	0·63	93·2	1·22
8	0·94	100·3	5·76	0·70	104·5	2·90
9	0·78	92·9	9·53	0·80	88·1	6·27
	(0·95	(98·1)	(3·8)	(0·33)	(94·9)	(−1·0)
10	0·92	102·9	3·9	0·34	87·8	−2·28
	(0·95)	(98·1)	(3·7)	(0·70)	(89·5)	(2·7)
11	0·99	128·9	5·89	0·83	92·2	4·59
	(0·74)	(92·1)	(1·6)	(0·17)	(89·3)	(0·64)
12	0·97	119·4	12·33	0·80	93·3	2·00
	(0·93)	(97·8)	(5·1)	(0·77)	(94·9)	(−1·66)
13	0·95	91·1	6·82	0·95	80·5	7·36
	(0·64)	(109·4)	(5·14)	(0·88)	(87·9)	(6·72)
Mean	0·90	103·6	6·93	0·74	91·3	3·73
S.E.M.	0·02	4·7	1·07	0·05	3·1	1·10
Mean before phrenicotomy ($n = 5$)	0·92	107·0	7·69	0·74	88·4	3·59
S.E.M.	0·04	7·4	1·47	0·10	2·3	1·72
Mean after phrenicotomy ($n = 5$)	0·84	99·1	3·87	0·57	91·3	1·48
S.E.M.	0·06	2·8	0·64	0·14	1·5	1·51

Numbers in parentheses are results after phrenicotomy. r = correlation coefficient, i = intercept, s = slope.

significantly following phrenicotomy (from 3·59 to 1·48; $P < 0.05$). Furthermore, the degree of correlation between ΔL and EMG was significantly less compared to the pre-phrenicotomy condition ($P < 0.05$).

In three animals, we examined the effects of supplemental doses of pentobarbitone anaesthesia on the relationship between ΔL and change in EMG. The results for one animal are shown in Fig. 6. After administration of anaesthesia (20 mg pentobarbitone), there was a marked decrease in breathing frequency while amplitude of inspiratory activity was less affected. The slope, Y-intercept and correlation coefficient of this relationship were not significantly affected by supplemental anaesthesia. Similar results were obtained in two other animals.

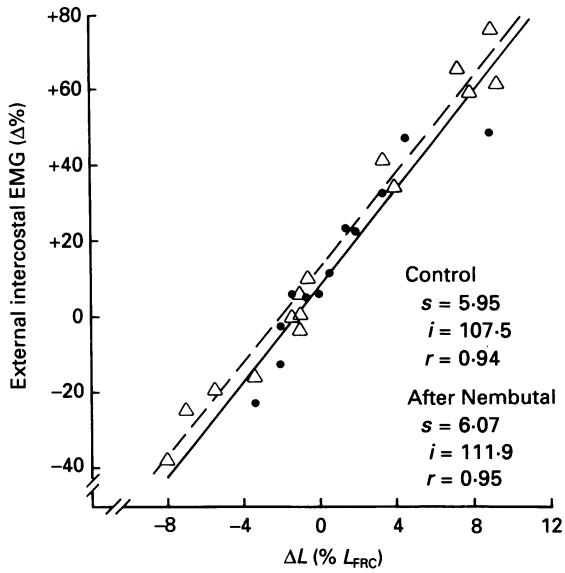

Fig. 6. Effect of Nembutal (20 mg total dose) on the relationship between the percentage change in muscle length (ΔL) and percentage change in external intercostal EMG. The additional dose of Nembutal did not result in any change in this relationship. See text for further explanation. s = slope; i = intercept; r = correlation coefficient.

DISCUSSION

In vagotomized animals, a wide variety of responses in terms of intercostal motor activation can occur in response to airway occlusion. The results of this study demonstrate that the magnitude of the change in intercostal motor activation induced by airway interruption is directly related to the difference between the degree of muscle shortening achieved during the occluded breath compared to that achieved during free breathing at the same level of chemical drive. Furthermore, a relationship between changes in muscle length and EMG was also demonstrable after phrenicotomy when the control levels of electrical activation and degree of shortening are known to be significantly greater (Ninane, DeCramer & DeTroyer, 1986). Our results, therefore, support the well-described hypothesis that the increase in intercostal neural output in response to loaded breathing occurs to achieve the 'desired' degree of muscle shortening (cf. Hammond *et al.* 1956; Altose, Stanley, Cherniack & Fishman, 1975; D'Angelo, Garzaniti & Bellemare, 1988; Greer & Stein, 1989). In addition, our results also describe the relationship between changes in intercostal muscle length and the change in the degree of intercostal muscle activation. For both the external and parasternal intercostals, the percentage change in peak intercostal EMG activity was linearly related to the peak inspiratory difference in muscle length between test and control breaths. Interestingly, when the end-inspiratory length during the test breath was shorter than the control breath, in which case muscle spindles would be disengaged, peak inspiratory activity was reduced compared to the control breath.

It should be emphasized that the range of inflation did not encompass the entire vital capacity range. While progressive inflations up to approximately 1·0 l resulted in progressive reductions in ΔL and consequent reductions in the degree of intercostal enhancement, larger inflations frequently resulted in marked excitation of the initial portion of the intercostal EMG. The mechanism of the latter response is not clear but may relate to activation of the sympathetic nervous system (Prabhakar, Marek & Loeschcke, 1985). The specific characteristics of this response were not systematically evaluated in the present study.

Methodology

The degree of electrical activation of the intercostal muscles was assessed in this study by electromyographic recordings. It is possible, however, that the changes in muscle length alone could have altered the magnitude of EMG signals without any actual change in neural activation. This could have resulted from changes in the number of muscle fibres in close proximity to the recording electrode that occurs with alterations in muscle length. For this reason, a greater EMG signal would be expected to occur under conditions of muscle shortening whereas the EMG signal would decrease under conditions of muscle lengthening. In the present study, however, shorter muscle lengths were associated with reductions in EMG activity and longer muscle length with increases in EMG activity, i.e. the opposite of that predicted by muscle length changes alone. Therefore, while this effect may have had some influence on the magnitude of the changes in muscle activation, it did not alter the overall qualitative aspects of the described responses.

The shape of the EMG trajectories, which are often non-linear, was not assessed in the present study. D'Angelo *et al.* (1988) have shown, however, that the shape of the EMG trajectory in any given muscle remains unchanged with alterations in chemical drive or airway occlusion at end-expiration. The determination of peak activity, therefore (as measured in the present study), provides a reliable index of changes in the degree of neuromuscular activation.

At first glance, one might argue that the comparison of free breaths at FRC to occluded breaths at lung volumes other than FRC may not be completely valid. As mentioned above, since passive changes in lung volume result in progressive changes in resting intercostal muscle length, lung volume changes as well as the effects of airway occlusion were reflected in the measured end-inspiratory length following airway occlusion. Furthermore, the near-linear relationship between changes in end-inspiratory length and EMG activity lends support to our contention that the major independent variable was, in fact, differences in end-inspiratory length achieved by this methodology.

Previous studies

Von Euler & Fritts (1963) also compared peak external intercostal EMG activity during occluded breaths to peak EMG activity during the preceding free breaths at different lung volumes. In their paper, they presented results from one animal the data of which are qualitatively similar to those of the present study. These investigators observed enhancement of external intercostal EMG during airway occlusion at FRC; at a certain point above FRC, EMG activity during free breathing

was equal to that of the occluded breath (i.e. tracheal occlusion had no effect on peak EMG activity); and, at lung volumes above this value, EMG activity during the occluded breath was less than that of the free breath. Importantly, the observed changes in intercostal motor activation resulting from airway occlusion at different lung volumes were totally abolished following section of the dorsal roots in the cervical and thoracic regions. This indicates that the observed changes in EMG activity at different lung volumes in that study as well as the present one were not secondary to complicating methodological concerns but mediated reflexly via spinal mechanisms. Changes in load-compensatory reflex response due to changes of volume gain in vagotomized rabbits ventilated by a servorespirator were also described by Romaniuk, Celichowski & Kowalski (1985).

Previous authors, however, could only speculate on the effects that changes in lung volume and airway occlusion would have on muscle length. They postulated that increases in lung volume were associated with muscle shortening and decreases in lung volume with muscle lengthening but that muscle length was constant during airway occlusion. They assumed, therefore, that the passive length changes secondary to changes in lung volume accounted fully for the observed phenomenon. More recent studies (van Lunteren & Cherniack, 1986; Greer & Stein, 1989) and the present one clearly demonstrate, however, that the inspiratory muscles may shorten or lengthen considerably even while contracting under occluded conditions. It is evident, therefore, that the actual difference in end-inspiratory length between occluded and free breaths cannot be determined without direct length measurements. Furthermore, the fact that there was essentially no enhancement in intercostal EMG when ΔL was equal to 0, excitation when end-inspiratory shortening was less than control and disfacilitation when greater than control (both before and after phrenicotomy), strongly suggests that differences in muscle length during the test and control breaths were the true stimuli for the observed responses.

Several previous investigations have demonstrated facilitatory vagal influences attributed to increments in lung volume during normal tidal breathing in anaesthetized animals (Bartoli, Cross, Guz, Huszczuk & Jefferies, 1975; DiMarco, von Euler, Romaniuk & Yamamoto, 1981). This phenomenon has been assessed in these studies by carefully comparing the trajectory of inspiratory motor activity before and after eliminating vagal volume-related feedback. During spontaneous breathing, airway occlusion for a single breath and the consequent prevention of lung expansion results in a reproducible reduction in the slope of electrical activity to both the diaphragm and intercostal muscles, as shown in Fig. 7*A*. The present study and others (cf. Sears, 1964*a*; von Euler, 1973; D'Angelo *et al.* 1988) clearly demonstrate the existence of non-vagal excitatory reflex effects on inspiratory intercostal motor activity, as well. Following vagotomy in the same animal (Fig. 7*B*), the increase in the EMG trajectory during airway occlusion indicates an excitation of intercostal motor activation. Intercostal neuromuscular activity during occluded breaths in the intact preparation, therefore, is determined by the balance between the depressant effect produced by the loss of vagal volume-related feedback and the excitatory effects mediated by non-vagal factors. Furthermore, the reduction in EMG trajectory observed in the intact preparation following airway occlusion suggests that the effect of vagal feedback is greater than non-vagal factors.

It should also be mentioned that the vagal facilitatory effects have been shown to be easily suppressed by supplemental anaesthesia (DiMarco *et al.* 1981), whereas the non-vagal facilitatory effects described in the present study are not. Consequently, the relative magnitude of these different reflexes may be dependent on the prevailing

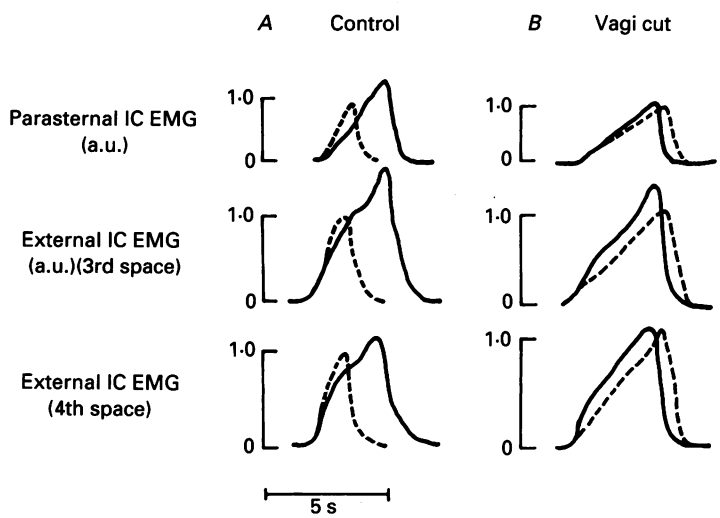

Fig. 7. Effect of tracheal occlusion on parasternal and external intercostal EMG before and after vagotomy at FRC. Occluded inspiratory trajectories (continuous line) are superimposed upon the preceding free breaths (dashed line). *A*, before vagotomy (control) tracheal occlusion resulted in a prolongation of inspiration, increase in end-inspiratory EMG amplitude, and decrease in the rate of rise of intercostal inspiratory activity. *B*, after vagotomy, tracheal occlusion resulted in a slight decrease in inspiratory time and enhancement of external intercostal and parasternal intercostal EMG. The increase in peak intercostal activity was achieved by an increase in rate of rise of activity.

level of anaesthesia. The reason for this difference in sensitivity to anaesthesia may be due to the different portions of the CNS involved in mediating these reflex effects. Load compensatory reflexes are organized at a spinal level (von Euler, 1973), while vagal facilitatory input is processed in the brain stem. Spinal segmental reflexes are probably less affected by pentobarbitone than reflexes organized on brain stem level.

Mechanism of changes in intercostal activation

Clearly, chest wall mechanoreceptor feedback and its corresponding potential effects on muscle activation is a dynamic intrabreath phenomenon (cf. Houk, Rymer & Crago, 1981; Greer & Stein, 1989, 1990). It is likely, therefore, that the relationship between changes in intercostal muscle length and intercostal activation also varied during the breath. This relationship, however, was not evaluated in the present study. Rather, we chose to measure end-inspiratory changes in muscle length and level of muscle activation for the following reasons. First of all, peak intercostal EMG activity reproducibly occurs at end-inspiration where EI motoneurones have the lowest threshold for afferent input (Sears, 1964*b*) and any reflex effects on intercostal motor drive are likely to be fully developed at this point. Secondly, as a result of the

vagotomized state of the animals, inspiratory duration was relatively constant. Consequently, both EMG and length were always measured at a relatively fixed interval from the onset of inspiration at which time the magnitude of central neural input to intercostal α- and fusimotor motoneurones should have remained constant between control and test breaths. A recent study of Greer & Stein (1990) showed that the previously described tonic and phasic fusimotor activity of intercostal nerves could be related to static and dynamic fusimotor drive, respectively. It has been postulated that tonic activity subserves predominantly a postural function, whereas phasic activity subserves a respiratory function. Our results do not allow us to differentiate the potential role of specific γ-fibres.

It is unlikely that the changes in EMG activity observed in the present study were chemically mediated since airway occlusion was maintained for only a single breath. Furthermore, although the absolute level of chemical drive may have varied during the course of the experiment, each occluded test breath was compared to preceding free breaths to control for the prevailing level of chemical drive at that moment. This allowed for a comparison between test breaths in each animal at a constant level of chemical drive. Since the changes in the level of muscle activation were non-chemical in origin, and the animals were vagotomized, the observed responses were most likely mediated by non-pulmonary neural mechanisms, presumably via chest wall mechanoreceptors.

The predominant muscle mechanoreceptors are muscle spindles and tendon organs. Muscle spindle endings are considered to be length receptors and tendon organs force receptors (cf. Shannon, 1986). Activation of muscle spindle afferents are generally thought to produce excitatory reflex effects and tendon organs inhibitory one (Kirkwood & Sears, 1974, 1980). The responses described in the present study were largely excitatory in nature and directly related to differences in actual compared to 'expected' length changes (ΔL). These results strongly suggest that these excitatory responses were secondary to activation of receptors sensitive to changes in muscle length, i.e. muscle spindle endings. Interestingly, the responses of the PA muscles, which are known to have far fewer muscle spindles (Duron, 1973; Jung-Caillol & Duron, 1976), were significantly less than those of the EI in terms of the magnitude of the EMG changes for the same ΔL. The differential responses of parasternal, external intercostal muscle and diaphragm activity (D'Angelo *et al.* 1988) to tracheal occlusion indicate that the common central inputs (Sears, 1964*b*) to the chest wall motoneurones are governed by different peripheral control mechanisms. For example, in contrast to the intercostals, the diaphragmatic motoneurones are inhibited by excitation of external intercostal muscle spindles (Remmers, 1970; von Euler, 1979; Romaniuk, Supinski & DiMarco, 1990). Furthermore, stimulation of intercostal tendon organs may excite (cf. Shannon, 1986) while diaphragmatic tendon organs inhibit (Cheeseman & Revellette, 1990) phrenic motoneurones.

We cannot exclude in our study some interaction with potential tendon organ stimulation (cf. Sant'Ambrogio & Widdicombe, 1965; Newsom Davis & Sears, 1970; Hilaire *et al.* 1983). This was particularly true for responses in which there was no excitation or actual reductions in motor activation in response to airway occlusion. These latter responses occurred when end-inspiratory length during the occluded

breaths was shorter than that during the free breath. Although muscle force was not measured in the present study, it is conceivable that the developed force was greater under these conditions resulting in activation of tendon organs, thereby causing reflex inhibition of intercostal activity. It should be noted, however, that the reduction in EMG activation fell on the same linear relation as the excitation of motor activity suggesting that these responses may represent a disfacilitation of intercostal motor activation (possibly due to disengagement of muscle spindles rather than true inhibition). This also suggests that intercostal α-motoneurones are under continuous facilitation via primary muscle afferents (Nathan & Sears, 1960; Sant'Ambrogio & Widdicombe, 1965). Lung inflation or stretching the muscle by postural changes (Duron, 1973; Hilaire *et al.* 1983; Goldman *et al.* 1984) may decrease the effects of centrally transmitted depolarization (Sears, 1964*b*) to such an extent that respiratory activity could be abolished.

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