

Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers

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Hodges, Paul W., Simon C. Gandevia, and Carolyn A. Richardson. Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. *J. Appl. Physiol.* 83(3): 753–760, 1997.—The influence of respiratory activity of the abdominal muscles on their reaction time in a postural task was evaluated. The electromyographic (EMG) onsets of the abdominal muscles and deltoid were evaluated in response to shoulder flexion initiated by a visual stimulus occurring at random throughout the respiratory cycle. Increased activity of the abdominal muscles was produced by inspiratory loading, forced expiration below functional residual capacity, and a static glottis-closed expulsive maneuver. During quiet breathing, the latency between activation of the abdominal muscles and deltoid was not influenced by the respiratory cycle. When respiratory activity of the abdominal muscles increased, the EMG onset of transversus abdominis and internal oblique, relative to deltoid, was significantly earlier for movements beginning in expiration, compared with inspiration [by 97–107 ms ($P < 0.01$) and 64–90 ms ($P < 0.01$), respectively]. However, the onset of transversus abdominis EMG was delayed by 31–54 ms ($P < 0.01$) when movement was performed during a static expulsive effort, compared with quiet respiration. Thus changes occur in early anticipatory contraction of transversus abdominis during respiratory tasks but they cannot be explained simply by existing activation of the motoneuron pool.

postural reaction; abdominal muscles; trunk; motor control; respiratory loading; motoneuron pool

CONTRACTION OF THE MUSCLES OF THE anterior abdominal wall occurs as part of the postural adjustments before the movement of the upper limb (15) and voluntary loading of the trunk (6). Rapid flexion of the upper limb produces a brief challenge to postural stability of the trunk as a result of reactive forces acting equal and opposite to those producing the movement (4, 16). Movement of this type is associated with contraction of transversus abdominis (TrA) preceding the activation of deltoid, whereas contraction of internal oblique (IO), external oblique (EO), and rectus abdominis (RA) occurs shortly after the muscle responsible for initiation of the movement (i.e., deltoid) (15). These contractions are considered to contribute to the maintenance of postural equilibrium and stabilization of the trunk (3, 6, 11, 15). However, the contribution of the abdominal muscles to postural control is likely to be complicated by their role in respiration.

Although electromyographic (EMG) activity of the abdominal muscles is rarely recorded during quiet breathing, abdominal muscles are activated toward the end of expiration once ventilation is increased (12). Contraction of the abdominal muscles contributes to

the regulation of the length of the diaphragm (7), end-expiratory lung volume (14), and expiratory airflow (1). When ventilation is increased voluntarily, contraction of TrA, IO, EO, and RA produces expiration below functional residual capacity (FRC) with a subsequent increase in inspiratory volume (8, 12). In contrast, involuntary increases in ventilation due to hyperoxic hypercapnia or loaded inspiration provoke contraction of TrA and IO at a lower minute volume than the other abdominal muscles (8, 25). The influence of respiratory contraction of the abdominal muscles on their contribution to postural control has not been considered. Abdominal expiratory muscles are depolarized in expiration and thought to be hyperpolarized in inspiration (20). These changes could mean that the latency to onset of postural contraction of abdominal muscles changes with the phase of respiration and is shorter when the motoneuron pool is already recruited.

Therefore, this study was designed to determine whether 1) timing of activation of specific abdominal muscles would be altered in postural tasks according to the phase of quiet respiration; 2) timing of activation of specific abdominal muscles would be altered in postural tasks when respiratory drives to motoneurons were increased; and 3) timing of activation of abdominal muscles changed during tonic activation of their motoneurons in an expulsive effort.

MATERIALS AND METHODS

Subjects. Five volunteer subjects (4 men and 1 woman) 26.6 ± 1.4 yr old and of average height (176.4 ± 5.0 cm) and weight (69.0 ± 3.6 kg) were studied. Subjects were excluded if they had any major neurological or respiratory condition. The study was approved by the Medical Research Ethics Committee of the University of Queensland. Subjects were unaware of the experimental hypotheses and were not trained in respiratory maneuvers.

Recordings. Fine-wire bipolar EMG electrodes [75 μ m, insulated stainless steel (A-M Systems), 1-mm insulation removed] were inserted into the left TrA, IO, and EO under the guidance of real-time ultrasound imaging (Advanced Technology Laboratory, Australia). The technique has been described in detail elsewhere (5, 8). The locations for electrode insertion are identified in Fig. 1. The electrodes were placed 2 cm medial to the proximal end of a line drawn from the anterior superior iliac spine to the inferior border of the rib cage for TrA; 2 cm medial and superior to the anterior superior iliac spine for IO; and midway between the iliac crest and inferior border to the rib cage in the midaxillary line for EO. Surface electrodes (Ag/AgCl) were placed over RA on either side of a line drawn between the right and left anterior superior iliac spine 2 cm lateral to the midline and over the muscle belly of deltoid in parallel with the muscle fibers. Skin impedance was reduced to <5 k Ω before attachment. Control

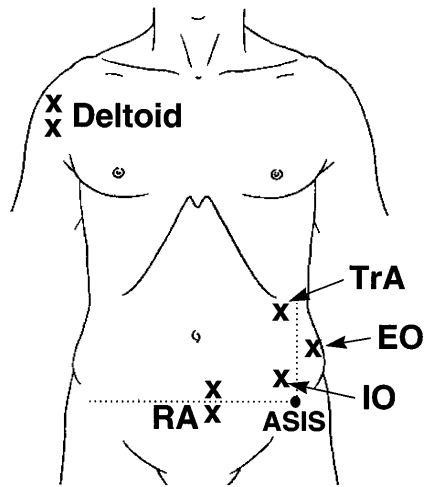


Fig. 1. Location for insertion of fine-wire electrodes into transversus abdominis (TrA), internal oblique (IO), and external oblique (EO) and placement of surface electrodes for rectus abdominis (RA) and deltoid. Dashed lines indicate line drawn vertically from anterior superior iliac spine (ASIS) and horizontally between right and left ASIS for identification of electrode sites.

studies revealed that the onset of EMG for deltoid and RA could be measured accurately with surface electrodes.

The respiratory cycle was monitored by using an air-filled tube strapped to the chest at the level of the nipples. Movement of the rib cage was measured by changes in pressure within the device throughout the respiratory cycle.

Experimental conditions. The subjects stood on a force platform (SMS Healthcare, UK) that provided auditory feedback of equality of weight bearing between the two legs. This was necessary, since variation in weight bearing has been suggested to influence the activation of postural muscles in limb movement tasks (18). The sensitivity of the device was set to alarm if the imbalance between the legs exceeded 4% of total body weight. Subjects performed 40 trials of unilateral shoulder flexion to 60° from the resting position by the trunk. Movement was performed as fast as possible in response to a visual stimulus in each of four different conditions presented in random order. Each set of limb movements was completed in 15 min for each condition. The conditions were as follows.

1) Quiet breathing: subjects breathed at a self-selected volume and rate.

2) Inspiratory loading: in an attempt to activate TrA preferentially during expiration (8), subjects inspired through a narrow tube (ID 5.5 mm, length 190 mm). Expiration was performed without resistance. After a loaded inspiration, the EMG activity of TrA and IO increased during the expiratory phase. Subjects were unaware of possible respiratory responses to loading.

3) Expiration below FRC: to evaluate the influence of respiratory activation of the abdominal muscles on their reaction time latency during the postural limb movement task, the activity of each of the abdominal muscles was increased during expiration by voluntary forceful expiration below FRC. It was necessary for this technique to be voluntary, since involuntary methods to increase expiratory airflow differentially influence the abdominal muscles (8). Subjects were instructed to maintain a normal respiratory rate throughout the procedure.

4) Static expulsive maneuvers: subjects were asked to stop breathing and made a submaximal forced expiration against a closed glottis to evaluate the effect of a static contraction of the abdominal muscles.

Throughout the trials, the subjects maintained a relaxed standing position. The visual stimulus to move was provided by a green light placed 1.5 m in front of the subject at eye level and was preceded by a warning stimulus by a random period of between 0.5 and 3 s. Five practice movements were performed in each condition, with feedback from the experimenters, to ensure consistency of response. In all conditions, except the static expulsive maneuver, the timing of the movement stimuli was presented at random during the respiratory cycle so that the phase in which the limb movement occurred would vary. This resulted in ~10 stimuli occurring at end expiration, midinspiration, end inspiration, and midexpiration (Fig. 2A). In each static expulsive maneuver, the subject performed two repetitions of shoulder movement in response to the visual stimulus, and 10 trials were recorded.

Data collection and analysis. EMG data were sampled at 2,000 Hz by using 12 bit analog-to-digital conversion, band-pass filtered at 20–1,000 Hz, and stored on computer for later analysis. The time to onset of the increase in EMG activity formed the basis of the analysis. The onset of increased EMG of each muscle was determined automatically by using an algorithm following full-wave rectification of the trace. The algorithm identified onset as the point where the mean of 50 consecutive samples deviated by >3 SDs from the mean baseline activity recorded for the 100 ms before the warning stimulus. The trials were separated into four groups based on the phase of respiration, as assessed by the motion of the rib cage. The phases of respiration were arbitrarily defined as (Fig. 2A): end expiration (300 ms before the onset of rib cage expansion), midinspiration (300 ms either side of the point halfway between end expiration and end inspiration), end

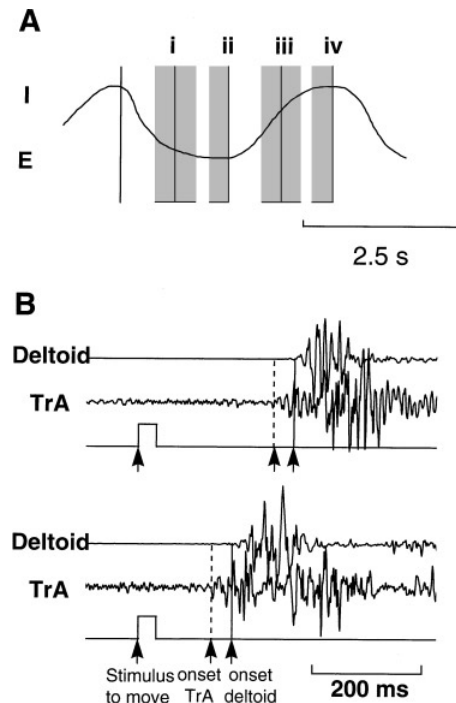


Fig. 2. A: identification of phases of respiration. I, inspiration; E, expiration; i, midexpiration; ii, end expiration; iii, midinspiration; iv, end inspiration. B: 2 trials from 1 set of rapid arm movements for a single subject, demonstrating variation in time from stimulus to move to EMG onset (arrows). Onset of deltoid EMG is denoted by unbroken line and onset of TrA EMG by broken line. Note difference in absolute latency of responses but relatively consistent period between onset of EMG of each muscle.

inspiration (300 ms before the cessation of rib cage expansion), and midexpiration (300 ms either side of the point halfway between end inspiration and end expiration). The allocation of each trial to one of the four groups was determined by the time of onset of the increase in deltoid EMG. Recordings were discarded if the onset of the increase in deltoid EMG occurred outside the defined respiratory phases.

The absolute reaction time from the stimulus to move to the onset of increased EMG was measured for deltoid and each abdominal muscle. In addition, the onset of increased EMG of each of the abdominal muscles was measured relative to the onset of the increased deltoid EMG. Negative values indicated the onset of increased abdominal muscle EMG before the onset of the increase in deltoid EMG. The absolute reaction time for deltoid and the abdominal muscles varied between trials within each condition, and this may obscure any changes in timing of the abdominal muscles relative to the arm movements in the different respiratory phases and different experimental conditions (Fig. 2*B*). In contrast, the difference in latency between the activation of the abdominal muscles and deltoid was relatively constant within conditions (Fig. 2*B*; see also Ref. 15). Hence, the main analyses were performed on the reaction times expressed as the latency difference between the onset of activity in the deltoid and the various abdominal muscles.

The main analysis relied on pooled measurements from each single trial within each respiratory phase (i.e., one value per condition per subject). However, the general conclusions were unaltered if measurements were made when all trials in a particular condition were averaged after rectification of the EMG signals (within or across subjects). Rectification and averaging have been used for illustrative purposes (see Fig. 5). An analysis of variance and Duncan's multiple-range test were used to evaluate changes in the relative time of onset of the increase in EMG of each muscle between the respiratory phases for each condition. For the expulsive maneuver, the reaction time latency of each muscle was compared with that averaged over the four respiratory phases in the quiet-

breathing condition. Throughout the text results are expressed as means \pm SE. Statistical significance was set at the 5% level.

RESULTS

As judged by the absence of single and multiunit discharges in the EMG records, all of the abdominal muscles were "silent" during quiet breathing in the relaxed standing position (Fig. 3*A*). This contrasts with earlier studies reporting activity of the abdominal muscles in standing (7, 10, 23). This can be explained by our instruction to relax in the standing position: when tonic activity was occasionally observed, it ceased when subjects were reminded to relax. In the loaded-inspiration condition, the activity of TrA and also IO, but not EO and RA, occurred throughout expiration (Fig. 3*B*). This is consistent with previous findings (8). In contrast, the activity of all abdominal muscles increased during voluntary expiration below FRC (Fig. 3*C*). In each subject, a similar pattern of muscle activation occurred during the different experimental conditions.

Quiet breathing. When subjects breathing quietly received a visual signal to flex their shoulder rapidly, an increase in TrA EMG occurred before contraction of deltoid (Figs. 4*A* and 5). This "anticipatory" activity of TrA occurred 20 ± 14 ms before the increase in deltoid EMG. The onset of increased EMG of IO, EO, and RA followed that of deltoid (Figs. 4*A* and 5). The absolute latencies for each muscle are presented in Table 1, and the relative latency between the onset of deltoid EMG and that of each of the abdominal muscles is presented in Table 2. There were no significant differences in the absolute latencies across the respiratory phases for any of the muscles. Given that the variation in the absolute latencies was larger than the variation in the relative

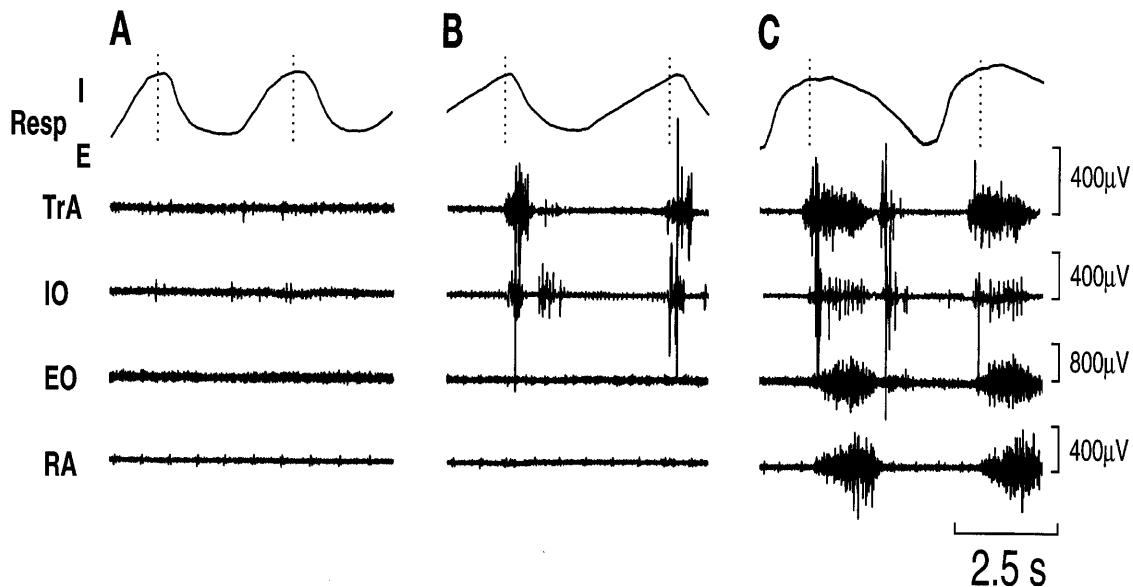


Fig. 3. Representative raw EMG traces for each muscle from a typical subject during quiet breathing (*A*), breathing with an inspiratory load (*B*), and breathing with voluntary expiration below functional residual capacity (FRC) (*C*). Note absence of overt muscle activity during quiet breathing, phasic increase in TrA and IO EMG during expiration with inspiratory loading, and phasic EMG increase in all abdominal muscles during voluntary expiration below FRC. Resp, respiration. Dashed lines indicate end of inspiration.

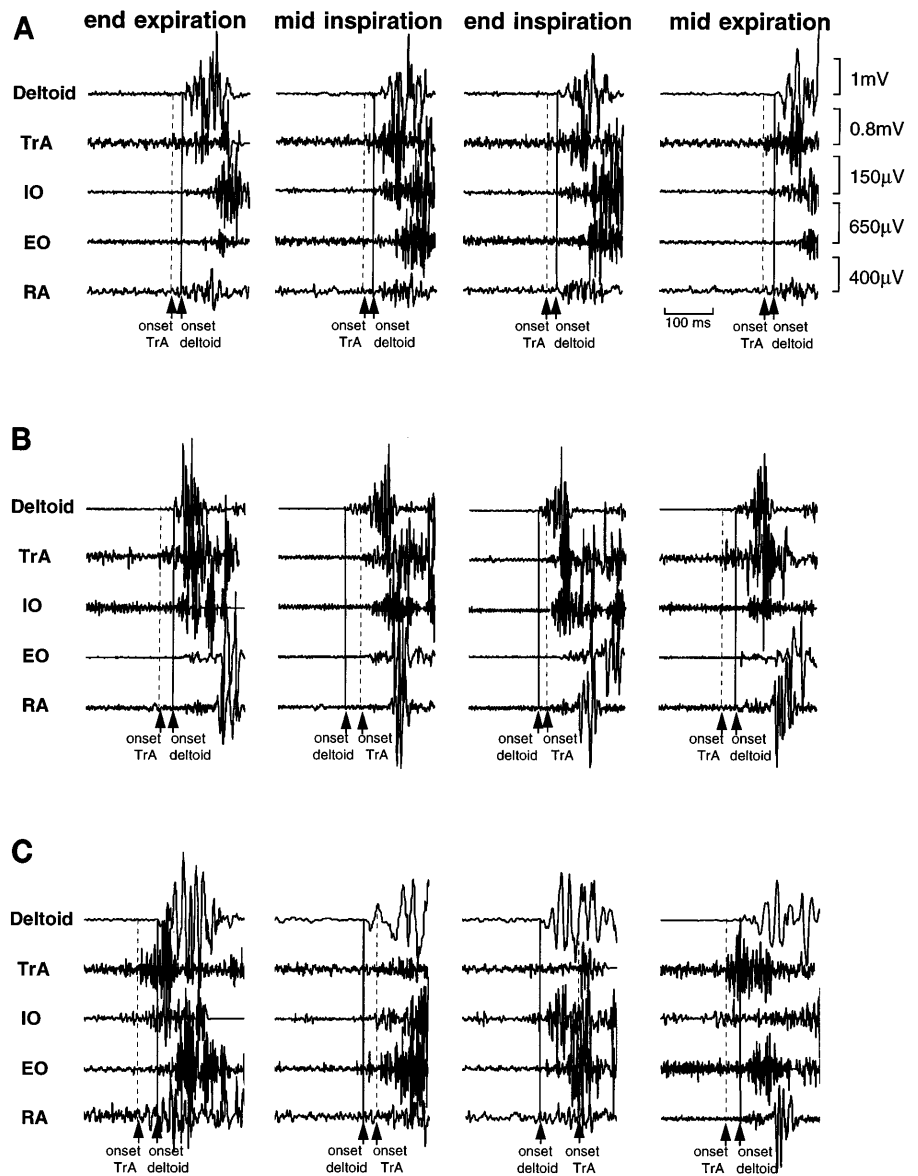


Fig. 4. Representative raw EMG traces for all muscles for a typical subject for trials during 4 respiratory phases with each respiratory condition. *A*: quiet breathing; note increase in TrA EMG before onset of increased deltoid EMG and constant relationship between onset of increased EMG of each of abdominal muscles for each respiratory phase. *B*: breathing with an inspiratory load; note change in relationship between onset of increased EMG of TrA and IO relative to deltoid with TrA, preceding deltoid by greatest amount during midexpiration and following deltoid during inspiration. *C*: breathing with voluntary expiration below FRC; note change in relationship between onset of increased EMG of TrA and IO relative to that of deltoid. TrA precedes deltoid during midexpiration and follows deltoid during inspiration. EMG scales are consistent between each group of trials.

latency between the abdominal muscles and deltoid, the onset of EMG in the abdominal muscles is subsequently reported relative to the onset of deltoid (Fig. 2*B*; see MATERIALS AND METHODS). When the subjects were breathing quietly, no significant difference in the latency between the onset of increased EMG of the abdominal muscles and deltoid occurred between the respiratory phases (Table 2).

Inspiratory loading. When subjects breathed with an inspiratory load, the onset of the increase in TrA EMG preceded that in deltoid EMG only when the movement occurred during the mid-expiratory phase of the respiratory cycle (Figs. 4*B* and 5 and Table 2). However, some individual subjects (including those whose data are presented in Figs. 4 and 5) also showed activation of TrA before deltoid in end expiration. The onset of the increase in EMG of TrA relative to that of the deltoid EMG occurred earlier in both midexpiration and end expiration than in midinspiration and end inspiration (Figs. 4*B* and 5 and Table 2), although, for the group,

there was no significant difference between end expiration and midinspiration. The onset of increased EMG of TrA preceded the onset of the increase in deltoid EMG by -38 ± 13 ms in midexpiration but followed the onset of deltoid by 59 ± 21 ms at end inspiration. This resulted in a mean difference of 97 ± 23 ms in the activation times of TrA between these two respiratory phases. There was no significant change in the absolute reaction time of deltoid between respiratory phases [$F(4,3) = 1.13, P = 0.38$].

The onset of the increase in EMG of IO preceded the onset of the increased deltoid EMG by -2 ± 32 ms in midexpiration but followed it by 62 ± 26 ms at end expiration. No significant difference was noted between the respiratory phases for EO or RA.

Voluntary expiration below FRC. When subjects breathed voluntarily and deliberately expired below FRC, the onset of the increased TrA EMG occurred before the increase in deltoid EMG when movement occurred during both the mid- and end-expiratory

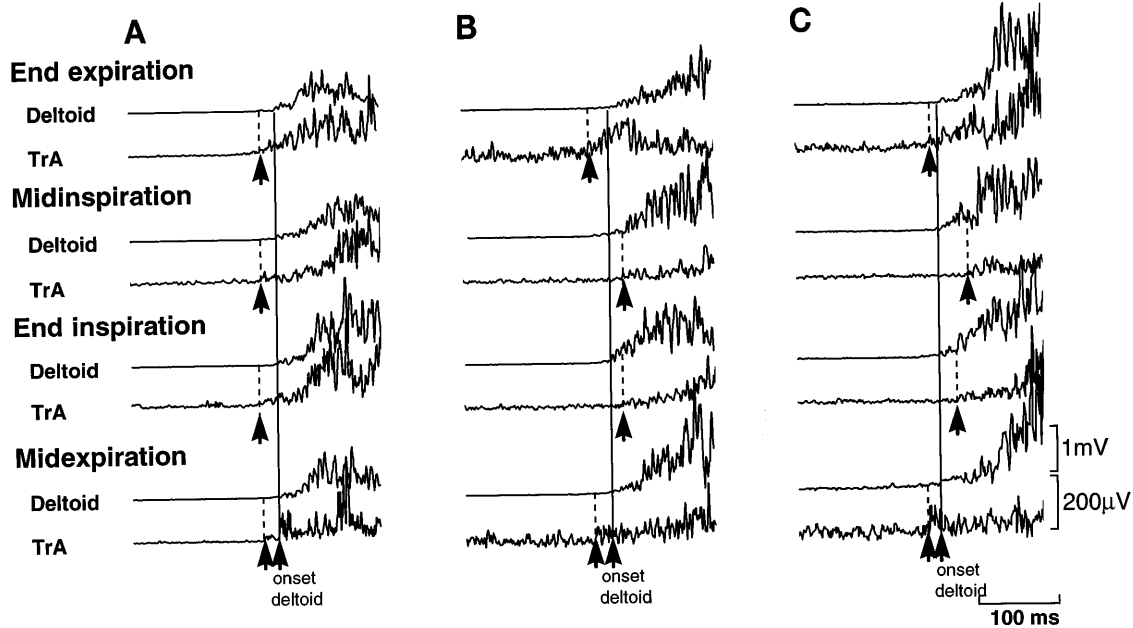


Fig. 5. Mean of rectified EMG for 5 repetitions in 1 subject for TrA and deltoid for trials during each phase of respiration during quiet breathing (A), breathing with an inspiratory load (B), and breathing with voluntary expiration below FRC (C). Trials were aligned to onset of deltoid EMG before averaging. Onset of deltoid EMG is denoted by unbroken line and that of TrA is noted by broken line. Note consistent relationship between mean onset of EMG of deltoid and TrA between respiratory phases with quiet breathing and variation in relationship between phases when breathing with an inspiratory load and breathing with voluntary expiration below FRC.

phases of the respiratory cycle. In midexpiration, the increase in EMG of IO also occurred before deltoid EMG (Figs. 4C and 5 and Table 2). The onset of the increase in TrA EMG in response to limb movement was significantly earlier when deltoid EMG was initiated during end expiration compared with midinspiration and for midexpiration compared with end inspiration, end expiration, and midinspiration (Figs. 4C and 5 and Table 2). Across subjects, the mean difference in latency of onset of the increased TrA EMG between midexpiration and midinspiration was 107 ± 23 ms. The onset of the increase in IO EMG was significantly earlier during midexpiration compared with midinspiration (Table 2). The onset of the increase in EMG of EO and RA occurred after that of deltoid and was not significantly different between movements initiated in each of the respiratory phases.

Static expulsive maneuver. Subjects stopped breathing, and all of the abdominal muscles contracted during a static expulsive maneuver. In contrast to the three earlier conditions, the onset of increased TrA EMG

followed that of deltoid by 22 ± 8 ms (Fig. 6) when the upper limb was moved. When compared with the mean onset latency for the four phases of quiet breathing, the onset of the increase in TrA EMG occurred significantly later when the movement was initiated during a static expulsive maneuver than during quiet respiration by a mean of 41 ± 13 ms (Fig. 6).

During the expulsive maneuver, the onset of the increase in EMG of IO, EO, and RA followed the onset of the increase in deltoid EMG. Although the onset of increased EMG of IO relative to that of deltoid was only of borderline significance when compared with quiet respiration, the relative onset of EO and RA occurred significantly later when movement was initiated during the static expulsive maneuvers. The mean delay in onset of EMG of EO and RA, compared with quiet breathing, was 42 ± 18 and 45 ± 29 ms, respectively.

DISCUSSION

The present study has evaluated the influence of respiratory activation of the abdominal muscles on their timing of onset of contraction in a brief postural task. The results provide evidence that the preparatory activation of TrA and IO occurs earlier relative to the contraction of the arm muscle when added respiratory activity is present in these muscles, as a result of inspiratory loading or voluntary expiration below FRC. Respiratory modulation of the preparatory contractions is not evident for quiet breathing. In contrast, the onset of TrA activation was delayed relative to deltoid when motoneurons of the abdominal muscles were activated by a static expulsive maneuver. This contradictory influence of activation of specific abdominal

Table 1. Absolute reaction times of abdominal and deltoid muscles with each respiratory phase in the quiet-breathing condition

Respiratory Phase	Deltoid, ms	TrA, ms	IO, ms	EO, ms	RA, ms
End expiration	163 ± 17	145 ± 17	208 ± 17	240 ± 10	284 ± 28
Midinspiration	178 ± 16	160 ± 17	208 ± 25	239 ± 27	274 ± 39
End inspiration	149 ± 13	140 ± 20	185 ± 29	210 ± 19	260 ± 34
Midexpiration	148 ± 16	116 ± 12	169 ± 21	237 ± 25	278 ± 40

Values are means ± SE; times are in ms from the visual stimulus to move. TrA, transversus abdominis; IO, internal oblique; EO, external oblique; RA, rectus abdominis.

Table 2. Relative latency between onset of EMG of deltoid and each of abdominal muscles, with each respiratory phase and in each of the respiratory conditions

Respiratory Condition	Phase	TrA	IO	EO	RA
Quiet respiration	EE	-18 ± 12	49 ± 30	77 ± 12	119 ± 26
	MI	-20 ± 15	33 ± 19	65 ± 31	96 ± 37
	EI	-9 ± 17	38 ± 26	63 ± 21	112 ± 38
	ME	-32 ± 13	23 ± 19	89 ± 19	129 ± 34
<i>F</i> (4,3)		0.58	0.56	0.44	0.89
Inspiratory load	EE	6 ± 26	13 ± 13	122 ± 15	168 ± 25
	MI	53 ± 18	36 ± 14	92 ± 17	144 ± 57
	EI	59 ± 21	62 ± 26	108 ± 21	140 ± 51
	ME	-38 ± 13	-2 ± 32	123 ± 26	187 ± 83
<i>F</i> (4,3)		8.62†	2.57*	0.52	0.57
Expiration below FRC	EE	-18 ± 10	13 ± 27	118 ± 32	129 ± 32
	MI	44 ± 18	61 ± 24	30 ± 98	123 ± 38
	EI	22 ± 11	18 ± 22	115 ± 27	129 ± 45
	ME	-63 ± 22	-29 ± 29	97 ± 19	110 ± 38
<i>F</i> (4,3)		12.74†	2.15*	0.43	0.53
Voluntary expiration below FRC		22 ± 8	70 ± 11	116 ± 19	158 ± 31
	<i>F</i> (4,1)	8.80†	3.97	4.55*	4.91*

Values are means ± SE. Times are in ms from onset of deltoid EMG. Negative values indicate onset of abdominal muscle EMG before that of deltoid. *F* values for comparison between each respiratory phase for conditions of quiet respiration, inspiratory loading, and expiration below FRC are presented along with the *F* values for comparison between static expulsive maneuver and mean onset in quiet-breathing condition. EE, end expiration; MI, midinspiration; EI, end inspiration; ME, midexpiration. * $P < 0.05$, † $P < 0.01$.

muscles on their timing of EMG onset with limb movement cannot be explained simply by reduced time needed to discharge the abdominal motoneurons due to their concurrent activity. Additional, presumably supraspinal, factors must be involved.

The study confirms that one or more of the abdominal muscles contract before the agonist limb muscles when movement of the arm is performed while standing. This "preparatory" activation of TrA and IO is likely to be reprogrammed as their onsets occur before any relevant afferent activity can initiate them. A recent study documented the preparatory contractions of TrA in

reaction-time movements of the arm but did not assess the influence of respiration on them (15).

The earlier contraction of TrA and IO during expiration (when the neural drive to these muscles is increased) compared with inspiration in the inspiratory-loading and expiration-below-FRC conditions is consistent with the influence of changes in membrane potential associated with the respiratory cycle. Thus the depolarization of the abdominal motoneurons that occurs during expiration (22) should decrease the time needed to discharge them in a postural task, leading to earlier activation of TrA and IO. Whereas these changes in membrane potential (i.e., central respiratory drive potentials) have been identified in the cat (22), their existence cannot be confirmed directly in human studies. Consistent with the proposed relationship between postural activity and the respiratory neural drive, Rimmer and colleagues (21) considered that changes in intercostal muscle activity during respiration combined with trunk rotation in humans may reflect the sum of the central respiratory drive potentials and voluntary drive to the muscles. Rimmer et al. (21) showed decreased postural contraction of the internal intercostal muscles during inspiration and increased inspiratory contraction of the external intercostal muscles when the trunk was rotated.

However, the results of the present study cannot be explained simply by changes in background neural drive to the abdominal motoneurons. This is because the activation of the same muscles was relatively delayed during a static maneuver where the activation of the abdominal motoneurons was increased. Second, the change in timing of activation is specific to particular muscles: being prominent for the TrA and IO but not for RA and EO, despite the respiratory activity of all muscle being increased in the expiration-below-FRC

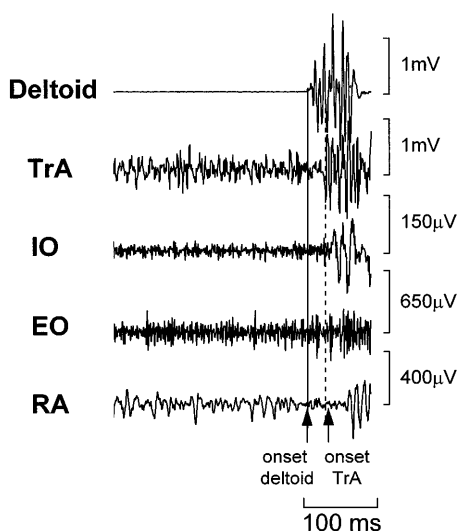


Fig. 6. Representative raw EMG from abdominal muscles for a single trial of rapid shoulder flexion occurring during performance of an expulsive maneuver with glottis closed. Onset of deltoid EMG is denoted by an unbroken line and that of TrA by broken line. Note delayed onset of TrA EMG following that of deltoid.

condition. In the previous study of Rimmer and colleagues (21), it was hypothesized that afferent input from muscle spindles and tendon organs may also contribute to the variation in intercostal muscle activity. However, it is difficult to delineate the role of muscle and other afferents in the later modulation of the output to the various abdominal muscles associated with arm movements. Although they are unlikely to determine the onset times for the responses in TrA and IO, they may well contribute to the later modulation of the EMG. Such an integration of afferent and descending input is likely to involve spinal (2, 17) and supraspinal sites, including the respiratory centers (9, 20). However, because of the task dependence of the responses and because they may sometimes involve coactivation of the diaphragm (see below) and abdominal muscles (TrA and IO), it is unlikely that the anticipatory response of the abdominal muscles is mediated purely via output from classic pontomedullary respiratory centers.

An alternate way to analyze the results is to consider the mechanism by which the abdominal muscles act on the trunk. One function attributed to the preparatory contraction of the abdominal muscles is the production of intra-abdominal pressure to assist in the stabilization of the trunk and to control postural equilibrium disturbed by the movement of the arm (6, 15). The potential mechanisms for this include an increase in tension of the thoracolumbar fascia through which TrA attaches to the spine (24) as well as the increase in intra-abdominal pressure itself (5, 19). To be effective, these mechanisms require contraction of the diaphragm to prevent its passive lengthening and the displacement of the abdominal contents. Preliminary recordings from the diaphragm with intramuscular electrodes are consistent with its activation together with TrA in similar tasks to those used here (P. W. Hodges and S. C. Gandevia, unpublished observations). The results obtained during the static expulsive maneuvers support the view that the preparatory contractions are involved in the mechanical stabilization of the trunk. Here there is already an elevation of intra-abdominal pressure due to the co-contraction of the diaphragm and abdominal muscles. Hence, when the arm is required to move, there may be less need to provide a more stable platform as this has already been ensured by the contraction of the abdominal muscles. In this situation, the preparatory contraction of TrA and IO was delayed until after the onset of the movement. TrA and IO are likely to be the more important abdominal muscles in any mechanical stabilization produced via an increase in abdominal pressure as they are more effective in this task than RA or EO (5, 6).

This analysis is also consistent with the observations during the phases of respiration when the activity of some of the abdominal muscles was increased. The preparatory contractions of TrA and IO occurred earlier when the arm movement began during expiration rather than during inspiration. During quiet inspiration, the abdominal pressure may have already been elevated sufficiently as a result of diaphragmatic con-

traction (see Ref. 13) so that there was less need for an anticipatory contraction of the abdominal muscles.

In summary, the reported changes in timing of contraction of the abdominal muscles in a brief postural task between phases of respiration cannot be simply explained by the influence of conventional respiratory drive or ongoing voluntary drive to abdominal motoneurons. The findings are consistent with the complex interaction of respiratory and postural demands on the abdominal muscles to optimize trunk control during rapid movement of the upper limb. It seems that supraspinal structures are able to select the appropriate output to abdominal and other truncal muscles before limb movement based on the biomechanical need for an increase in abdominal pressure.

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