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Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm

PAUL W. HODGES AND SIMON C. GANDEVIA
Prince of Wales Medical Research Institute, University of New South Wales, Sydney, New South Wales 2031, Australia

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Hodges, Paul W., and Simon C. Gandevia. Changes in intra-abdominal pressure during postural and respiratory activation of the human diaphragm. *J Appl Physiol* 89: 967–976, 2000.—In humans, when the stability of the trunk is challenged in a controlled manner by repetitive movement of a limb, activity of the diaphragm becomes tonic but is also modulated at the frequency of limb movement. In addition, the tonic activity is modulated by respiration. This study investigated the mechanical output of these components of diaphragm activity. Recordings were made of costal diaphragm, abdominal, and erector spinae muscle electromyographic activity; intra-abdominal, intrathoracic, and transdiaphragmatic pressures; and motion of the rib cage, abdomen, and arm. During limb movement the diaphragm and transversus abdominis were tonically active with added phasic modulation at the frequencies of both respiration and limb movement. Activity of the other trunk muscles was not modulated by respiration. Intra-abdominal pressure was increased during the period of limb movement in proportion to the reactive forces from the movement. These results show that coactivation of the diaphragm and abdominal muscles causes a sustained increase in intra-abdominal pressure, whereas inspiration and expiration are controlled by opposing activity of the diaphragm and abdominal muscles to vary the shape of the pressurized abdominal cavity.

postural control; abdominal muscles; spinal stability

ACTIVITY OF THE HUMAN DIAPHRAGM and intercostal muscles is coordinated for both respiratory and postural functions (22, 28). Although the diaphragm is the principal muscle of inspiration, it is also active when the spine is perturbed (19). For instance, when stability of the trunk is challenged in a controlled manner by reactive moments from movement of a limb, electromyographic (EMG) activity of the diaphragm increases before the limb movement (19). This response is related to the amplitude of the forces that perturb the spine (19, 22) and has been confirmed from direct measurement of muscle shortening by using ultrasound imaging and changes in transdiaphragmatic pressure (Pdi) (19). With a sustained challenge to the posture of the trunk, by repetitive movement of an upper limb, diaphragm EMG is still modulated to maintain respiration, but activity also develops tonically (i.e., during expiration) with superimposed modulation at the frequency of limb movement (22). Thus there are at least two drives to diaphragm motoneurons during limb movement, one related to inspiration and the other to the movement. The mechanical output from the diaphragm and the relationship between this output and stabilization of the trunk during this complex situation have not been established.

Contraction of the diaphragm produces inspiratory airflow via depression of its central tendon and elevation of the lower ribs to increase the vertical and transverse diameters of the thoracic cavity (12). In addition, the diaphragm assists in the mechanical stabilization of the spine via increased intra-abdominal pressure (gastric pressure; Pga) in conjunction with contraction of the abdominal and pelvic floor muscles (4, 9, 17). If these two tasks are to occur concurrently, then the activity of the diaphragm must modulate intrathoracic pressure [pleural or esophageal pressure (Pes)] for respiration and Pga in association with limb movement. This dual function must involve coordination of the diaphragm and other muscles surrounding the abdominal cavity and may compromise the respiratory motion of the rib cage and abdomen.

The aims of the present study were as follows: *1*) to identify whether the multiple "inputs" to diaphragm motoneurons during repetitive limb movement result in respiratory modulation of Pes, a tonic increase in Pga, and modulation of Pga (with limb movements); *2*) to investigate whether the abdominal muscles, which have an opposite respiratory function but work with the diaphragm for Pga production, are similarly coordinated to the diaphragm for both postural and respiratory functions; *3*) to investigate whether diaphragm and abdominal muscle activity and their mechanical outputs are related to the resultant forces imposed on the spine from repetitive limb movement; and *4*) to evaluate whether the pattern of abdominal and rib cage movement during respiration is altered by the dual functions performed by the diaphragm during sustained repetitive arm movements.

Address for reprint requests and other correspondence: P. Hodges, Prince of Wales Medical Research Institute, Barker St., Randwick, NSW 2031, Australia (E-mail: p.hodges@unsw.edu.au).

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METHODS

Subjects. EMG studies were conducted on seven male subjects. The mean age, height, and weight of the subjects were 37 ± 6 (SD) yr, 1.76 ± 0.05 m, and 66 ± 9 kg, respectively. Pressure recordings were made from six of these subjects. Evaluation of changes in respiratory pattern with limb movement was undertaken on 10 subjects. Three subjects were involved in both experiments. Informed consent was obtained, and all procedures were approved by the institutional research ethics committee and conducted in accordance with the Declaration of Helsinki.

EMG. Recordings from the right costal diaphragm were made by using bipolar fine-wire electrodes fabricated from multistrand Teflon-coated stainless steel wire (7 strand, 110 μm; A-M Systems). The two wires were gently twisted, and the insulation was fused by heating. The cut ends of the wires were exposed, and the tips were bent back 0.5–1 mm to form a hook. The electrodes were threaded into a hypodermic needle (0.70 × 50 mm) and then inserted into the diaphragm via the seventh or eighth intercostal space in the midclavicular line under the guidance of real-time ultrasound imaging by using a 3-MHz vector array transducer (model 128XP/4, Acuson). Before needle insertion, the diaphragm was visualized by using the ultrasound transducer aligned parallel to the intercostal space to identify the approximate depth of the inner border of the diaphragm and to confirm that the selected site remained below the pleural reflection during deep inspiration (14, 19). Approximately 0.5–1 ml of lidocaine (2% with epinephrine) was injected along the proposed path to the external surface of the diaphragm. The location of the electrode was confirmed by evaluation of the EMG signal. EMG activity recorded during inspiration but not during forced expiration through pursed lips confirmed the electrode's location in the diaphragm. In one subject, additional recordings were made from the internal intercostal muscle from an electrode that was intended for placement in the diaphragm but exhibited activity on forced expiration and was silent with inspiration.

Recordings were made from the abdominal muscles by using bipolar fine-wire electrodes fabricated from two strands of Teflon-coated stainless-steel wire (75 μm; A-M Systems) that were gently twisted and inserted into a hypodermic needle (32 × 0.63 mm). The Teflon coating was removed from the distal 1 mm of each wire and bent back at 1 and 2 mm to form a hook. Electrodes were inserted under ultrasound guidance into transversus abdominis (TrA) on both sides (left: $n = 5$, right: $n = 7$) and the right obliquus internus abdominis (OI) ($n = 2$), obliquus externus abdominis (OE) ($n = 5$), and rectus abdominis ($n = 3$). Electrodes were inserted midway between the anterior superior iliac spine and the distal border of the rib cage. Surface EMG electrodes (1-cm diameter, Ag/AgCl disks) were placed over deltoid (anterior and posterior portions) and erector spinae (ES; $n = 5$) adjacent to the L_3 spinous process. A ground electrode was placed on the right shoulder.

Pressure recordings. Pga and Pes were recorded with a pair of pressure transducers (Gaeltec) inserted via the nose into the esophagus and stomach. The transducers were separated by 200 mm to ensure that they remained in the appropriate cavity despite motion of the diaphragm. Accurate location of the pressure transducers was confirmed when opposite pressure changes were recorded during a sniff before and after experimental tasks and by observation of opposite pressure changes during the respiratory cycle in tasks in which breathing was maintained. Preliminary data were collected by using a multilumen gastroesophageal catheter with a proximal balloon in the esophagus and a distal balloon inflated in the stomach (26). With this device, the possibility of movement of the sensors between cavities is minimal and any artifactual change in Pga recording as a result of movement of the catheter to different depths within the stomach are reduced because part of the balloon is likely to remain in the gastric air pocket. The results were the same as those recorded with the thin-film resistive strain-gauge sensors.

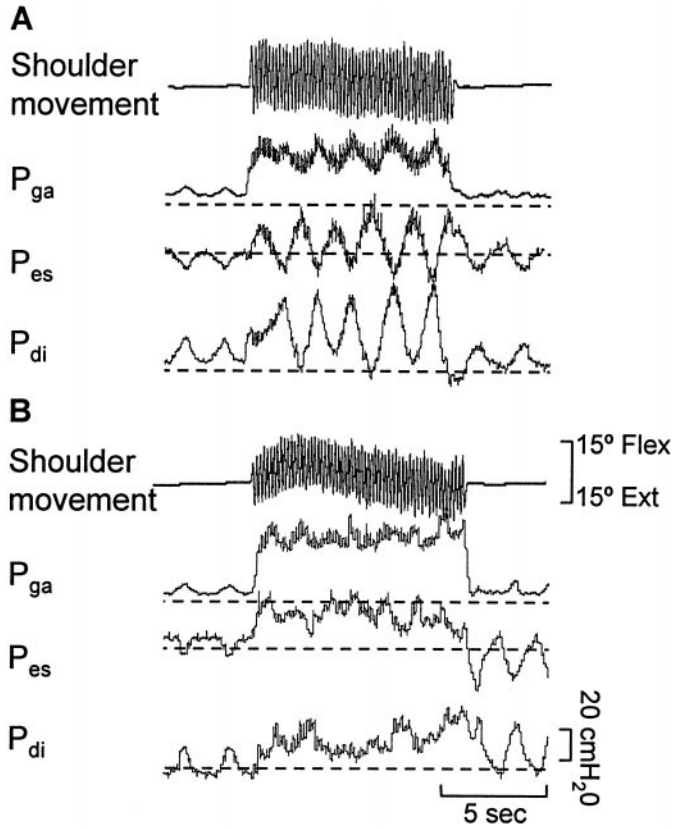

Fig. 1. Pressure changes during repetitive limb movement. Raw data from a representative subject for repetitive upper limb movement with (*A*) and without (*B*) breathing. Two breaths before and after upper limb movement are shown. Opposite direction of changes in intra-abdominal (gastric; Pga) and intrathoracic (esophageal; Pes) pressures indicates that the transducers remained in the appropriate cavities. Note sustained increase in Pga with movement and the respiratory- and movement-related modulation of amplitude of Pga, Pes, and transdiaphragmatic pressure (Pdi). Sustained elevation of Pga in the trial without breathing may be a result of closure of the glottis. Flex, flexion; Ext, extension.

Shoulder movement. Angular displacement of the left shoulder was measured with a potentiometer attached to a lightweight bar that was strapped to wrist of the subject with the axis of rotation aligned to that of the glenohumeral joint. Motion of the arm was displayed on an oscilloscope along with markers that indicated the required angular range of motion.

Respiration. Expansion of the rib cage was monitored with an inductance plethysmograph (Respitrace, Ambulatory Monitoring, Ardsley, NY) placed around the chest. In trials without EMG recordings from the trunk muscles, the respiratory movement of the abdomen was recorded with an additional band placed around the abdomen and the gains of the signals were adjusted by using an isovolume maneuver (6). Volume at the mouth was measured with a pneumotachograph.

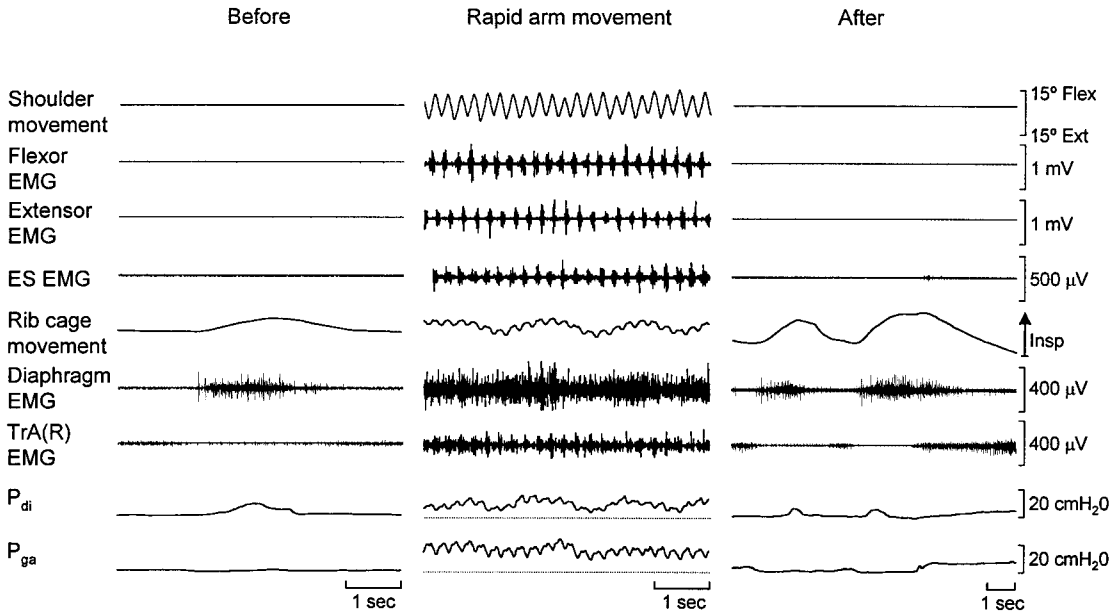

Fig. 2. Raw electromyogram (EMG), pressure, and movement data from a typical subject before, during, and after rapid upper limb movement. Subject breathed quietly before limb movement. In the period after movement, subjects were instructed to breath normally and then perform a deep inspiration followed by a deliberate expiration through pursed lips. EMG activity recorded from the diaphragm intramuscular electrodes during inspiration (Insp) but not forced expiration (*right*) indicates that the electrodes remained in the diaphragm. During repetitive movement of the upper limb as fast as possible, the diaphragm and transversus abdominis (TrA) EMG activity is tonic and modulated with respiration and movement. Similarly, Pga and Pdi are tonically elevated and modulated with respiration and movement. In this subject, there was only small amplitude variation in Pga and Pdi before and after the upper limb movement. ES, erector spinae; R, right.

Procedures. Subjects adopted a relaxed standing position with the feet placed shoulder-width apart. Each trial commenced with three quiet breaths followed by a period of limb movement. Subjects moved their right arm at the shoulder in the sagittal plane between 15° flexion and 15° extension "as fast as possible" for 10–30 s. At the completion of the limb movement, three further tidal breaths were recorded followed by two large breaths with forced expiration below functional residual capacity to ensure that the fine-wire electrodes remained in the diaphragm. Respiration was maintained throughout shoulder movement. In additional trials, movement was performed with increasing frequency between 15° flexion and 15° extension starting at 1 cycle/s and increasing to maximal speed (~3 Hz) over ~20 repetitions. In these trials, the subjects voluntarily stopped breathing and maintained their usual end-expiratory level.

In a separate trial, rib cage and abdominal movements and volume were recorded during arm movement from 10 subjects. The procedure for these trials was identical to that described for the main experiments and each subject moved his upper limb for a minimum of 10 s both while breathing and with breathing voluntarily stopped at the subjects usual end-expiratory level.

Data analysis. Diaphragm EMG was band-pass filtered from 53 Hz to 3 kHz and sampled at 5 kHz. EMG data from the abdominal, ES, and deltoid muscles were band-pass filtered from 53 Hz to 1 kHz and sampled at 2 kHz. Pressure and rib cage movement data were sampled at 2 kHz. Data were collected by using Spike2 (Cambridge Electronic Design) and exported for signal processing with Matlab (MathWorks).

For each subject, the average amplitudes of Pga and transdiaphragmatic pressure (Pdi) were measured for a period of three complete respiratory cycles before movement and for three breaths in the middle of the upper limb movement. The result was not different if the mean pressure was measured for the initial, middle, or final three breaths of the movement period. Peak-to-peak variation in pressure across the respiratory cycle was measured for the same three quiet breaths and the breaths during movement. Peak-to-peak variation in pressure was also calculated for five repetitions of limb movement in the middle of the movement period. The measurements of peak-to-peak pressure change with respiration and movement were averaged for each subject.

For comparison of the amplitude of diaphragm, abdominal, and ES EMG between inspiration and expiration, the root mean square (RMS) EMG amplitude was calculated for 500 ms epochs immediately before the maximum and minimum expansion of the rib cage. The baseline noise was subtracted from the RMS EMG amplitude, and all values were expressed as a percentage of the peak level recorded during respiratory cycle. Student's *t*-tests were used to compare EMG amplitudes between respiratory phases for each muscle.

For identification of the changes in EMG and pressure during the movement cycles, data were averaged across movement repetitions for each subject. Trials were triggered from the shoulder movement trace at the onset of shoulder flexion.

The relationships among the EMG, pressure, and movement data were also analyzed in the frequency domain. The power spectral densities of the autocorrelations of the EMG, pressure, and movement signals were calculated to identify the frequency of EMG bursts and the frequency of shoulder and rib cage motion. To remove any nonstationarity from the data due to low-frequency drift, and to remove any movement artifact, the EMG data were high-pass filtered at 100 Hz (4th-order zero-lag Butterworth filter) and then rectified and low-pass filtered at 30 Hz (4th order zero-lag Butterworth filter). The EMG, angular displacement of the shoulder, and movement of the rib cage

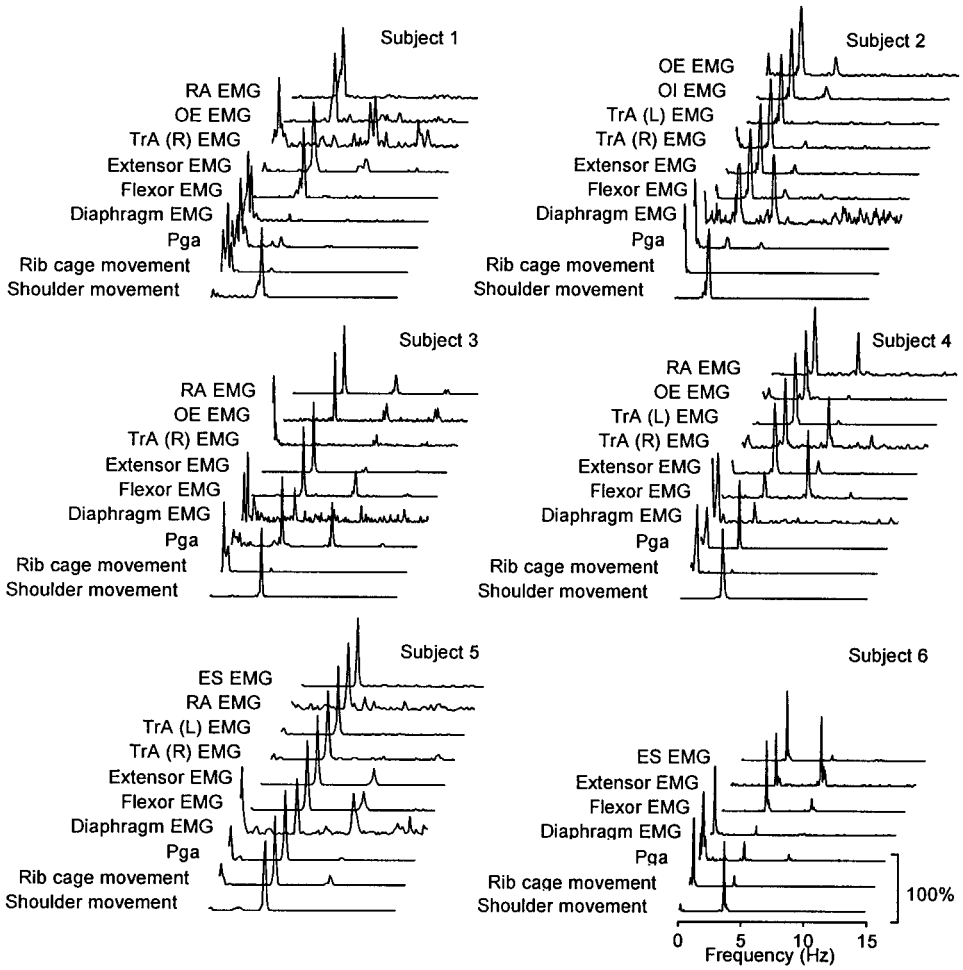

Fig. 3. Power spectral densities for individual subjects. Power spectra (calculated for 15-s periods) are presented for all subjects for trials with upper limb movement during breathing. Note peak in the upper limb movement signal at ~3 Hz and corresponding peaks in all EMG signals at this frequency and smaller peaks at twice the movement frequency for the majority of subjects. In addition, note peak in the rib cage, diaphragm, TrA, and Pga signals at the respiratory frequency of ~0.3 Hz. All power spectra are normalized to the largest peak present in each spectrum. Thus the relative amplitudes of the peaks indicate distribution of power between frequencies for each muscle. RA, rectus abdominis; OE, obliquus externus abdominis; OI, right internus obliquus abdominis; L, left.

were then resampled at 1 kHz so that all data were at the same sampling frequency and could be directly compared with identical resolution for the Fourier transform analysis. Spectral analysis was performed by using a Hanning window with no overlap. Because the high-frequency components within the multiunit EMG were removed, only the low-frequency EMG bursts in association with the shoulder movement and respiration were evaluated. Analysis of the filtered and rectified EMG signal by using the "runs" test indicated that the data satisfied the conditions of stationarity (5). An additional factor that supports the validity of this analysis is that many of the findings were confirmed by analysis of data averaged with the onset of the movement used as the trigger (see Fig. 3). For comparison of the amplitude of the power spectral densities among muscles and among subjects, the data were normalized. The peaks in power spectra were presented as a percentage of the maximal peak to indicate the relative distribution of the power at each frequency. However, the amplitudes of the peaks in the power spectra were not evaluated, and only the frequencies at which peaks occurred were used for analysis. Evaluation of the raw and averaged data provided a more precise measure of the amplitude of any modulation with breathing and movement.

In trials with arm movement at increasing frequency, the relationships between shoulder acceleration and the amplitude of EMG or pressure were assessed with Pearson's correlation coefficient between peak shoulder acceleration for consecutive arm movements and the corresponding RMS EMG or pressure (both measured over a 100-ms epoch).

The respiratory volume and the rib cage and abdominal movements were measured as a peak-to-peak change and averaged over three breaths, with and without upper limb movement. A multiple regression equation was calculated to calibrate the rib cage and abdominal movements to volume. The contribution of rib cage and abdominal expansion to the volume of air displaced with and without limb movement was calculated as a proportion of the sum of the two signals.

RESULTS

Changes in Pga. Rapid repetitive limb movement during breathing increased mean Pga by 26.3 ± 2.0 cmH_2O. This increase was maintained until the end of the task (Figs. 1*A* and 2). Similarly, the mean pressure difference across the diaphragm increased by 20.7 ±

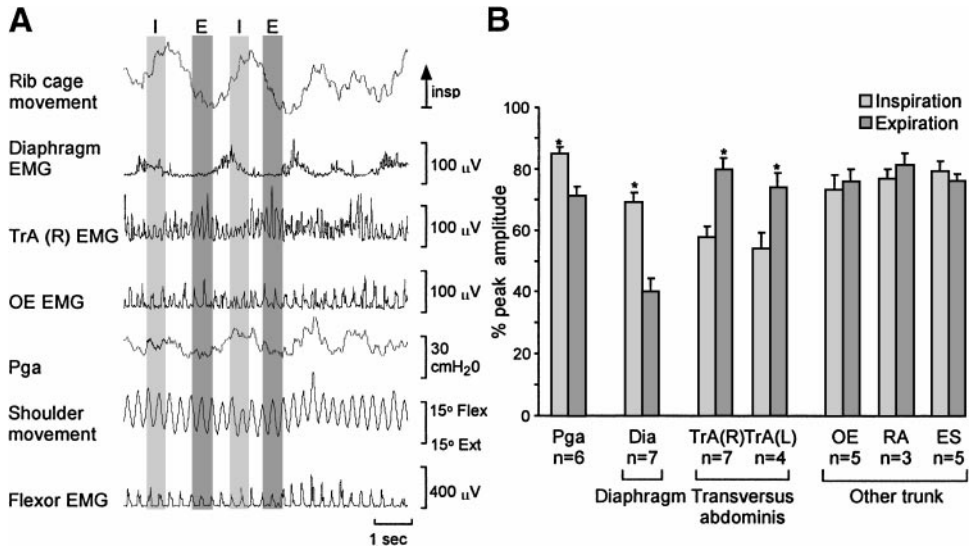

Fig. 4. Changes in EMG activity and pressure between phases of respiration during limb movement. Raw data for a representative subject (*A*) and data averaged across all subjects (*B*; means ± SD; *n*, no. of subjects included in each average) are presented. In *A*, shaded areas indicate the final 500 ms of inspiration (I; light bars) and expiration (E; dark bars) selected for measurement of amplitudes. OE activity is included for comparison with that of TrA. Note the variation in amplitudes of EMG between respiratory phase for TrA and diaphragm (Dia) but not the other trunk muscles.

2.0 cmH_2O (i.e., Pes increased by 5.7 ± 2.2 cmH_2O and may have been augmented by partial or complete closure of the glottis). In trials in which breathing continued, peaks were present in the power spectra of Pga and Pdi at the frequency of limb movement (3.5 ± 0.2 Hz) and respiration (0.3 ± 0.1 Hz) (Fig. 3). In all subjects, the majority of power was at the frequency of limb movement. Occasionally an additional peak was found at twice the frequency of limb movement. These peaks partly resulted from small changes in pressure at twice the movement frequency and partly because the pressure modulation was not precisely sinusoidal and therefore peaks are expected at the harmonic frequencies. The respiratory changes in Pga and Pdi during quiet breathing before limb movement were 8.2 ± 1.3 and 13.8 ± 2.2 cmH_2O, respectively. They increased to 19.4 ± 1.8 and 28.3 ± 5.1 cmH_2O, respectively, with shoulder movement. These pressure changes with respiration were 74 ± 5 and 136 ± 53% of the amplitude of the mean increase in Pga and Pdi with movement, respectively. Figure 4*B* shows that the Pga amplitude was significantly different between respiratory phases when averaged across subjects. With each shoulder movement, there was a mean peak-to-peak variation in Pga and Pdi of 14.2 ± 2.7 and 7.4 ± 1.5 cmH_2O, respectively (Fig. 5).

When breathing voluntarily ceased at the usual end-expiratory level before limb movement, similar increases in Pga and Pdi (28.1 ± 8.9 and 18.1 ± 5.9 cmH_2O, respectively) occurred during the upper limb movement (Fig. 1*B*). In addition, a single peak in the power spectrum was identified, and this occurred at the frequency of upper limb movement. The amplitudes of the fluctuations in Pga and Pdi with each movement were 11.3 ± 4.3 and 8.4 ± 2 cmH_2O, respectively, and were similar in size to the fluctuations when the subject was breathing during the arm movement. The timing of the peak increase in Pga (or, in several cases, 2 peaks) in relation to the arm movement was similar for all subjects and was aligned to the start of shoulder flexion, except in *subject 6*, in whom an additional larger peak in Pga was aligned with the peak of shoulder flexion (Fig. 5).

Muscle activity with repetitive limb movement. Contraction of the diaphragm and the trunk muscles occurred during the repetitive limb movement (Fig. 2). In contrast to before the period of arm movement, activity of the diaphragm occurred throughout the respiratory cycle for the duration of the repetitive shoulder movement (Fig. 2). In addition to the tonic activity, Fig. 2 shows modulation of diaphragm EMG with respiration. The amplitude of diaphragm EMG was higher in inspiration than expiration (Fig. 4). The opposite pattern of activity modulation was found for both the right and left TrA (Fig. 2). Similar to the diaphragm, TrA was active throughout the respiratory cycle and was modulated with respiration, but the amplitude of TrA EMG was higher during expiration (Fig. 4). In one subject, activity of the internal intercostal muscle (an expiratory muscle) was recorded and showed identical modulation to TrA. In contrast, the other trunk muscles (OE, RA, and ES) did not have variation in EMG amplitude between respiratory phases (Fig. 4). In two subjects in whom OI EMG was recorded (not illustrated) the activity was not modulated by respiration.

A major peak in the EMG power spectrum for each of the trunk muscles occurred at the same frequency as the movement of the upper limb (Fig. 3). In two subjects, a larger peak for TrA EMG was identified at twice the movement frequency (Fig. 3). On the basis of

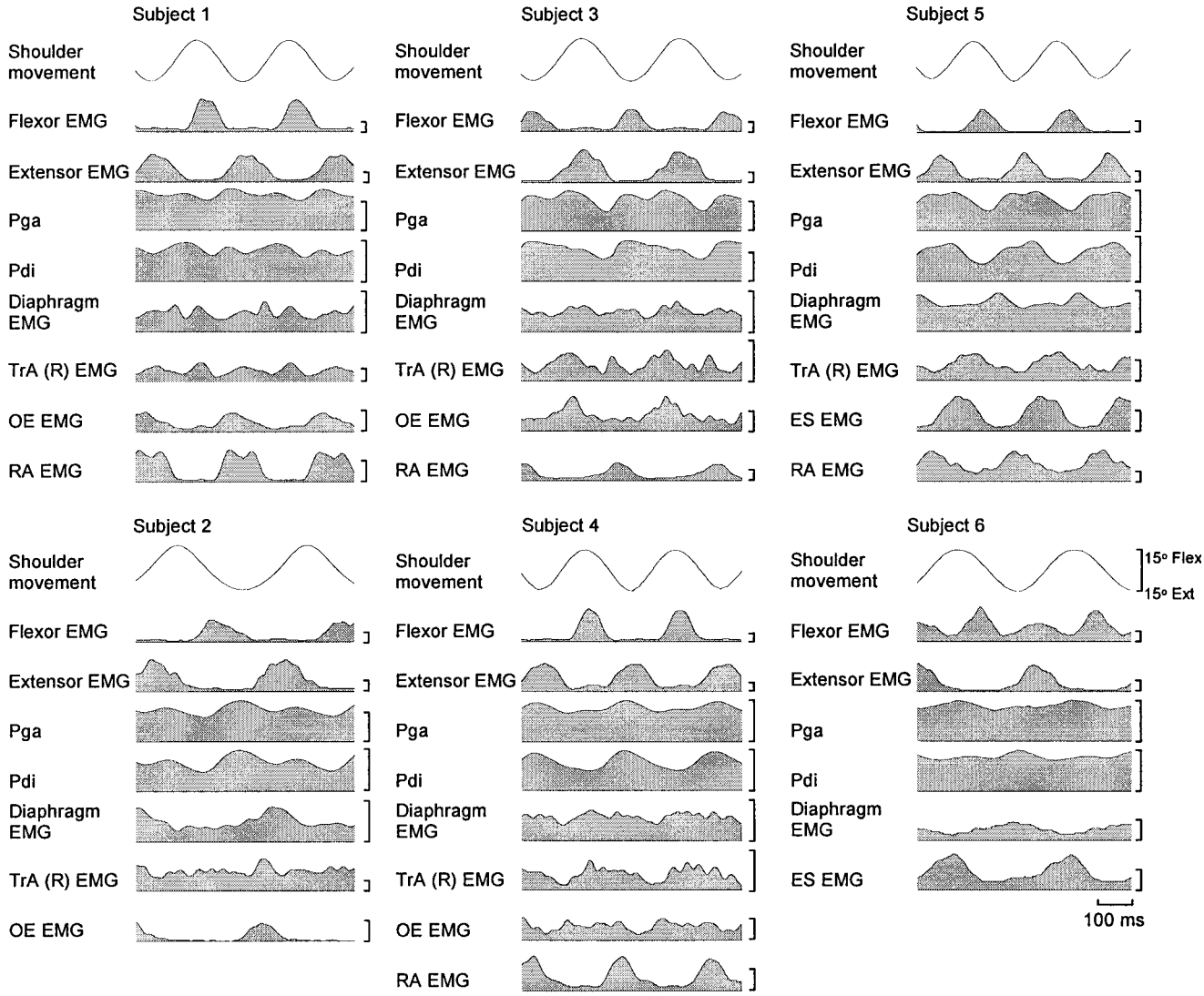

Fig. 5. Averaged data for individual subjects. Averaged data are presented for all subjects for upper limb movement during breathing. Data were aligned to onset of upper limb flexion. In *subjects 1–5,* peak Pga was aligned to start shoulder flexion, whereas in *subject 6* the peak was aligned to peak shoulder flexion. Note phasic bursts in diaphragm and TrA activity with each movement repetition. Vertical calibrations for the averaged EMG data: 50 μV, pressure data: 25 cmH_2O. Upper limb movement range: −15–15°.

averages triggered from the limb movement, there was modulation of EMG amplitude associated with the movement of the limb in all subjects (Fig. 5). The pattern of the modulation in EMG amplitude varied among muscles. For TrA and the diaphragm, this occurred as a phasic change in amplitude superimposed on tonic background activity. In contrast, the phasic activity of OI, OE, RA, ES, and the anterior and posterior deltoid occurred as relatively discrete bursts.

The temporal and spatial characteristics of EMG modulations produced by limb movements varied among subjects (Fig. 5). Across subjects, the peak amplitudes of diaphragm and TrA activity occurred within ~100 ms in most subjects. In addition, the peak activity of these two muscles was loosely aligned with a peak in the Pdi, but, when multiple peaks were present, the peak EMG was not always aligned to the largest peak in Pdi. The data presented in Fig. 5 show that the temporal relationship between the phasic changes in amplitude of Pga and that of the trunk muscles (including TrA and the diaphragm) also varied among subjects. In five subjects the peak Pga aligned with the start of shoulder flexion and corresponded with the peak trunk flexor EMG (RA) (Fig. 5). In *subject 6* the peak Pga aligned with peak shoulder flexion and peak EMG of the trunk extensor (ES).

Shoulder movement with increasing frequency. When the speed of limb movement increased, the change in Pga amplitude was consistent with the change amplitude of the predicted reactive moment from limb movement. Figure 6 shows that, when upper limb movement was performed with increasing frequency, the amplitude of Pga and trunk muscle EMG increased. For all subjects, the size of the EMG and pressure were corre-

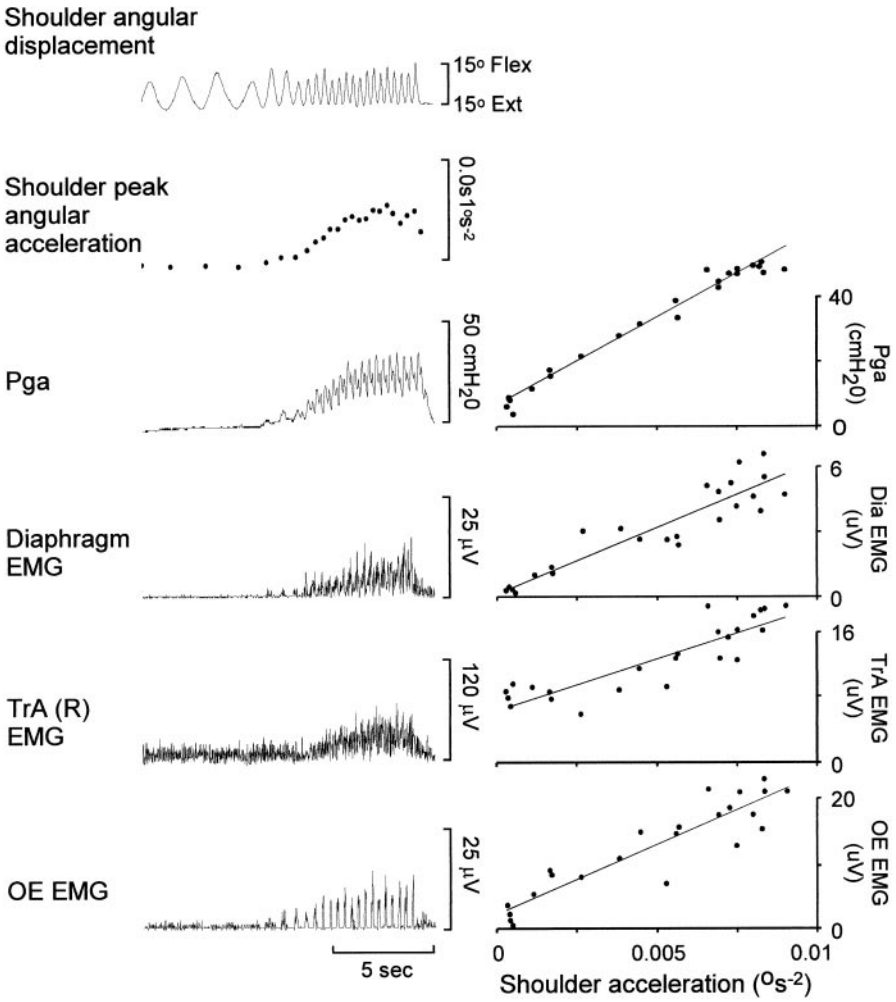

Fig. 6. Relationship between trunk muscle EMG, Pga, and acceleration of upper limb for a representative subject. *Left,* diaphragm, TrA and ES EMG, and Pga with the upper limb displacement and peak angular acceleration for a trial in which the frequency of upper limb movement increased from ~1 Hz to movement performed as fast as possible. *Right*: EMG and pressure measurements plotted against peak shoulder acceleration. There is a linear relationship between peak upper limb acceleration and EMG and pressure amplitudes ($P < 0.001$).

lated with the peak acceleration for shoulder movement recorded for each repetition of shoulder movement (Table 1).

Changes in respiration with repetitive shoulder movement. When movement and respiration were combined there was a 58 ± 15% increase in the volume of air displaced during the respiratory cycle (Fig. 7). Although there were small changes in airflow with each repetition of shoulder movement, it was not possible to determine whether this was due to actual changes in airflow or movement of the mouthpiece. In association with the increase in breath size, movement of the rib cage and abdomen increased by 76 ± 27 and 62 ± 19%, respectively. However, the proportion of rib cage and abdominal movement was not changed by the performance of limb movement and remained at ~40% abdominal and 60% rib cage motion (i.e., 41:59 for abdominal vs. rib cage movement when subjects breathed without movement and 39:61 when subjects moved their upper limb while breathing). Although the proportion of movements remained constant, there was a tonic reduction in abdominal circumference equivalent to a reduction in volume of 0.32 ± 0.09 liter (averaged across the respiratory cycle) and a corresponding increase in the rib cage circumference of 0.50 ± 0.07 liter. At the end of expiration, the abdominal volume decreased by 0.47 ± 0.12 liter and the rib cage volume increased by 0.15 ± 0.12 liter compared with breathing without limb movement.

Table 1. *Correlation coefficients for the relationship between the peak acceleration of the upper limb and the amplitudes of trunk muscle EMG and Pga when subjects moved their limb with increasing frequency*

Parameter	*r*
Pga	0.96 ± 0.03
Diaphragm	0.87 ± 0.07
Transversus abdominis (right)	0.91 ± 0.02
Transversus abdominis (left)	0.88 ± 0.02
Obliquus internus abdominis	0.83 ± 0.01
Obliquus externus abdominis	0.88 ± 0.03
Rectus abdominis	0.92 ± 0.03
Erector spinae	0.94 ± 0.01

Values are means ± SD. Pga, intra-abdominal (gastric) pressure.

DISCUSSION

The present data indicate that tonic activity of the diaphragm associated with repetitive upper limb

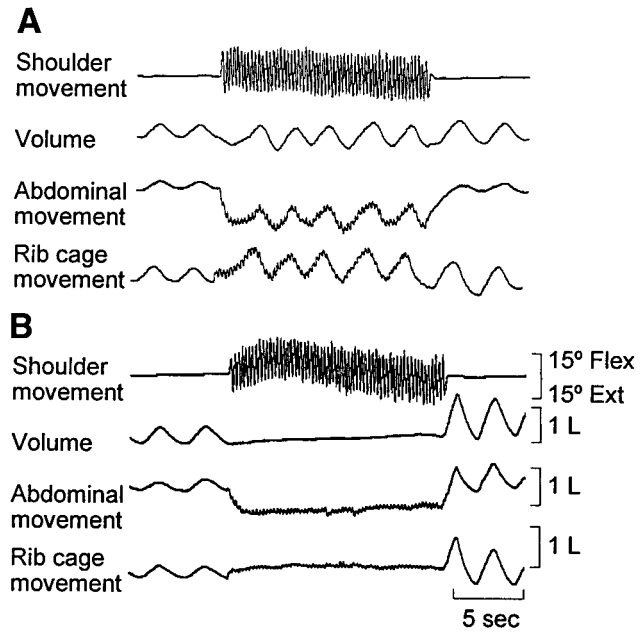

Fig. 7. Movement of rib cage and abdominal wall during rapid upper limb movement. Raw data from a representative subject of rib cage and abdominal movement and volume with repetitive limb movement during breathing (*A*) and with breath held at the normal end-expiratory volume (*B*). During limb movement, there is a reduction in mean abdominal volume and an increase in mean rib cage volume. Movement of both cavities is maintained during breathing, and small deviations occur with limb movements. These may be due to actual changes in volume or to distortion of the bands from superficial trunk muscle contraction.

movement increases Pga. This increase continues throughout movement, with modulation in the amplitude of the pressure increase with each limb movement and with respiration. Of the abdominal muscles, only TrA activity is modulated in conjunction with both the respiratory and postural demands during the limb movement.

Coordination of postural and respiratory functions of the trunk muscles. The dual task to regulate Pga and Pes concurrently was achieved by coordinated contraction of the diaphragm and abdominal muscles. The changes in diaphragm EMG identified with the addition of limb movement (i.e., tonic activity with phasic modulation at the frequencies of respiration and limb movement) corroborate our previous findings from a study in which a similar task was used (22). Corresponding changes were identified in TrA but not in the other abdominal muscles. Although OI, OE, and RA showed no respiration-related modulation in EMG amplitude, the amplitude of TrA was modulated with respiration and was out of phase with the modulation in amplitude of diaphragm EMG.

Several earlier studies have reported differentiation in the function of the abdominal muscles with respiratory tasks. Although the activity of TrA is not modulated with quiet breathing (13, 31), it is the first abdominal muscle recruited when expiration is increased with chemical drive or elastic loading (1, 13). In addition, the timing of the postural contraction of TrA, but not RA and OE, varied across the respiratory cycle when respiration was challenged by an inspiratory load (23). More recently, it has been argued that TrA, but not the other abdominal muscles (OI, OE, and RA), may have separate populations of motor units for postural and respiratory functions (27). Such an organization provides a potential mechanism to simplify the coordination of postural and respiratory functions, but this hypothesis has been based on investigation of a limited population of motor units and requires corroboration. The function of various abdominal muscles also changes with postural demand. TrA, unlike the other abdominal muscles, acts in a manner that is not affected by the direction of the perturbation to the trunk (9, 10, 24).

Tonic activity of the diaphragm and TrA with superimposed respiratory modulation is consistent with the dual postural and respiratory demands placed on these muscles with repetitive limb movement. At the initiation of limb movement, the diaphragm and abdominal (particularly TrA) muscles cocontract tonically to elevate the Pga (19). Respiratory airflow is then achieved by cyclic changes in the shape of the pressurized abdominal cavity as a result of alternate modulation of ongoing diaphragm and TrA activity. Thus, during inspiration, shortening of the diaphragm displaces the abdominal contents caudally while TrA is lengthened, and, during expiration, the opposite sequence occurs. This proposal is supported by the preservation of rib cage and abdominal movement during repetitive limb movement. Although the circumference of the abdomen was reduced and that of the rib cage was increased at the end of expiration, the relative contribution of both components to tidal volume was unchanged. Other studies have also identified a consistent relationship between abdominal and rib cage motion with exercise (2). This occurred despite the reduction in compliance of the abdominal contents as a result of tonic contraction of the abdominal muscles (2, 12). The change in abdomen and rib cage circumference would also lengthen the diaphragm. Although this would place the diaphragm at a more favorable point on the length-tension relationship (30) and may also increase passive contribution to Pdi as a result of stretch of the diaphragm, the effect of these factors on respiration or Pga production cannot be determined from the present results. Other factors contributing to respiratory movement of the rib cage and abdomen, such as activity of the intercostal and other accessory respiratory muscles, are also likely to change during repetitive limb movement. Changes in the activity of the muscles that act on the rib cage and abdomen during exercise have been investigated in several studies (2, 29). Although the results of these studies suggest that muscle activity is coordinated to reduce distortion of the rib cage and assist the flow-generating function of the diaphragm (2), the additional demand for postural control of the trunk with exercise has not been addressed.

The neural mechanisms underlying coordination of the dual functions of the diaphragm and TrA muscles are complex and not completely understood. However, the present data are consistent with summation of

multiple inputs to the respiratory motoneurons from either central or peripheral sources (see Ref. 22 for discussion).

Mechanical contribution of Pga to spinal stabilization. The sustained increase in Pga associated with the tonic activity of the diaphragm is consistent with a contribution of the diaphragm to mechanical stabilization of the trunk. This contribution is supported by the correlation between the increase in Pga and diaphragm EMG and the peak acceleration of the shoulder (and therefore the reactive moment imposed on the spine as a result of movement of the limb). A similar relationship between forces acting on the trunk and Pga has been identified in association with other static (11, 18, 25) and dynamic tasks (8).

Increased Pga may assist in mechanical stabilization of the spine either by production of an extensor moment (4, 9, 17) or by regulation of abdominal muscle shortening (16). A recent study has provided direct evidence that an extensor moment at the lumbar spine is produced by increased Pga (in the absence of abdominal muscle activity) (20). The present results provide further support for this function as the peak Pga was aligned temporally to the period of shoulder flexion. Although no subject had a simple Pga waveform, the extensor moment resulting from this increase in Pga is appropriately timed to counteract the flexor moment resulting from flexion of the upper limb (15, 21). However, it is less obvious why the increase in Pga is sustained. There are three possible explanations. First, tasks that require maintenance of a static joint orientation generally involve coactivation of opposing muscle groups (7). The combination of an extensor moment from the tonically increased Pga and the opposing flexor moments produced by the abdominal muscle activity and limb movement may provide an analogous situation. Second, Pga may be increased to restrict shortening of the abdominal muscles to increase their force generation. Third, Pga may directly influence the stiffness of the spine and control intervertebral motion (3, 10, 24).

Conclusion. The results of the present study provide evidence that the diaphragm and TrA muscles continuously contribute to respiration and postural control. The mechanical consequence of contraction of the diaphragm and TrA is an opposing action on the rib cage and abdomen but a shared function for pressurization of the abdominal cavity. As a result, the combined tonic and phasic activity of these muscles provides a mechanism for the central nervous system to coordinate respiration and control of the spine during limb movements.

This postural function is relevant to changes in respiratory muscle function with exercise. Although repetitive arm movement used in this study is not a normal functional task, the data provide evidence, in a controlled situation, of the mechanism for coordination of postural and respiratory functions of the trunk muscles and provide a method to investigate this coordination when respiration and postural control are compromised.

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