The impact of aging and habitual physical activity on static respiratory work at rest and during exercise

Rungchai Chaunchaiyakul,1,2 Herb Groeller,1 John R. Clarke,3 and Nigel A. S. Taylor1

1Department of Biomedical Science, University of Wollongong, Wollongong, New South Wales 2522, Australia; 2College of Sports Science and Technology, Mahidol University, Nakhonpathom 73170, Thailand; and 3Navy Experimental Diving Unit, Panama City, Florida 32407-7001

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QUANTITATIVE STUDIES of the respiratory system indicate that aging lungs contain fewer, but larger, alveoli (1, 40), respiratory muscle strength is reduced (5, 10), there is an increased calcification of the intrathoracic joints (9, 11), and the diaphragmatic contribution to ventilation is elevated, particularly during exercise (35). These changes are likely to affect the mechanics of the respiratory system and, in turn, modify the static work of breathing. Accordingly, the literature contains several reports that aging humans experience a reduction in lung tissue elastic work (14, 15, 38).

We have had particular interest in the possible effect that a greater calcification of the aging rib cage joints may have on chest wall stiffness, since a more rigid chest wall would increase the static work of breathing, similar to that seen in restrictive pulmonary disorders or chest strapping (8). Such changes may possibly result in a ventilatory limit to exercise. However, there has been very little research focusing on changes in chest wall mechanics in aging humans (27, 29, 35), with only one study specifically concerned with changes in chest wall elastic work during senescence (21); this project demonstrated that aging was accompanied by an increase in chest wall elastic work. Indeed, we are unaware of any investigation in which the separate mechanical attributes of the lung and chest wall have been simultaneously studied within the same aging sample. This has forced readers to combine data from separate studies to gain an overall understanding of the mechanics of the complete respiratory system during aging, thereby treating these independent observations as if they were drawn from the same population sample. Such a treatment is credible only if the subjects from these studies had equivalent pulmonary and thoracic function, health and disease status, stature, lifestyle, and exercise habits. Evidence to support such assumptions cannot be acquired from these earlier reports. Therefore, the first aim of this project was to concurrently investigate the thoracopulmonary mechanics of the aging respiratory system within the same population sample.

It is well established that a sedentary lifestyle is associated with an acceleration of age-related physiological deterioration (13, 39), such that the effects of aging and habitual inactivity may be additive. Some of these changes will influence the mechanical properties of the respiratory system, but they can possibly be delayed in habitually active older adults, or perhaps reversed when older sedentary people undertake regular physical training. For example, physical training can increase respiratory muscle function, maximal voluntary ventilation, maximal minute ventilation, and static lung volumes (3, 7, 17). In older, habitually active people, Hagberg et al. (16) have demonstrated that the age-related deterioration in pulmonary function is slower when long-term exercise habit is sustained. Thus such a long-term physical activity habit may also result in greater mechanical efficiency of the entire thoracopulmonary pump, relative to age-matched sedentary adults. The second aim of this study was to investigate this interaction.

However, with the exception of the work of McClaran et al. (19), who studied lung function in physically active elderly subjects, there is a dearth of information concerning the interaction of physical activity habit and thoracopulmonary mechanics in aging adults. We already know that regular exercise plays a beneficial role in the prevention of skeletal muscle and

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joint dysfunction with aging (39). If chest wall mechanics are adversely affected by a more sedentary lifestyle, then we similarly need to know whether or not those changes are able to be reversed through behavioral modification. However, despite the importance of the chest wall to the mechanical behavior of the total respiratory system, the interaction of habitual exercise on aging chest wall mechanics remains unexplored. We have recently developed new methods for analyzing respiratory mechanics and their impact on respiratory muscle work (34). A novel aspect of these methods involves the use of simultaneous analyses of lung tissue and chest wall mechanics to derive respiratory muscle work from the task-specific, end-expiratory lung volume (EELV). Thus the third aim of this project was to investigate changes in thoracopulmonary mechanics and their impact on respiratory muscle work at rest and during exercise across aging, physically active adults.

It was first hypothesized that age-related stiffening of the chest wall would increase the elastic work of chest wall expansion in older adults, and this would coexist with more compliant lungs. Our second hypothesis was that habitual physical activity would ameliorate age-related changes in the chest wall but not those of the lung tissue. Therefore, we simultaneously investigated the separate effects of aging on the elastic properties of the lung tissue and chest wall, we fractionated the contributions of these components to the static (elastic) work of breathing at rest and during exercise, and we tested the hypothesis that habitual physical activity could offset possible age-related effects on chest wall elastic work.

METHODS

This study used a cross-sectional design, where the interaction of aging and habitual physical activity was investigated using 29 chronically sedentary and 29 habitually active subjects, aged 20 – 83 years. The elastic properties of the lung and chest wall compartments were simultaneously investigated while resting subjects performed a series of expiratory pressure-volume relaxation maneuvers, commencing from total lung capacity and terminating at residual volume. From these data, lung tissue, chest wall, and total respiratory compliance curves were constructed for each subject and used to calculate and fractionate the elastic work of breathing, including inspiratory muscle work after (34) and during both resting and exercising tidal volume (Vt) excursions. These methods have not previously been used to investigate respiratory mechanics in aging adults. All methods were approved by the Human Research Ethics Committee (Univ. of Wollongong), with subjects providing written, informed consent.

Subjects. Fifty-eight healthy, asymptomatic nonsmokers with normal lung function histories (and without diagnosed cardiorespiratory disease) were recruited into three age groups: young (Y, 20 – 30 years, n = 20), middle-aged (M, 40 – 50 years, n = 19), and older adults (O, > 60 years, n = 19). Medical histories and physical examinations were used to exclude those with probable heart or cerebrovascular disease, those using cardiorespiratory medication, and those with abnormal resting or exercising electrocardiograms or with a resting systolic blood pressure > 160 mmHg. Gender and racial covariance was eliminated by recruiting Caucasian males. All subjects were either retired, current university employees, or full-time students, and their past or current occupations were sedentary in nature and could be broadly classified as involving either office or technical work. Accordingly, regular physical activity was achieved entirely through their involvement in voluntary exercise. Two subgroups of adults were recruited for each age division: sedentary and habitually active.

Differentiation of voluntary exercise habit was achieved using a screening questionnaire developed from the literature (6, 12, 37). Such screening was designed not to correlate with physical fitness but rather to separate active from inactive subjects. Accordingly, a sedentary subject was defined as one who was not currently involved, or had not been previously involved, in regular physical activity at > 50% of maximal capacity more than once per week, either within the last 10 years or, in the case of the young adults, for longer than 2 years since turning 20 years old. Habitually active subjects were currently active or had a long history of regular physical training, at an intensity of at least 50% maximal capacity three to four times per week over the last 10 years or for longer than 2 years since turning 20 (young adults). Each age group was, therefore, subdivided into chronically sedentary [young sedentary (YS, n = 10); middle-aged sedentary (MS, n = 10); and older sedentary (OS, n = 9)] and habitually active subgroups [young physically active (YA, n = 10); middle-aged physically active (MA, n = 9); and older physically active (OA, n = 10)]. Each group originally contained 10 subjects, but 2 subjects (1 OS and 1 MA), in whom evidence of asthma was found, were subsequently excluded. The physical characteristics and lung function of these subjects are summarized in Table 1.

Table 1. Physical characteristics and lung function of adult subgroups, classified by age and physical activity habit

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>YS</th>
<th>YA</th>
<th>MS</th>
<th>MA</th>
<th>OS</th>
<th>OA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>23.1 ± 3.3c</td>
<td>25.7 ± 4.7c</td>
<td>46.9 ± 5.3b</td>
<td>42.8 ± 5.1b</td>
<td>65.0 ± 4.1</td>
<td>66.0 ± 7.1</td>
</tr>
<tr>
<td>Mass, kg</td>
<td>68.5 ± 16.1</td>
<td>81.4 ± 6.7</td>
<td>77.6 ± 12.6</td>
<td>80.7 ± 4.7</td>
<td>75.2 ± 10.0</td>
<td>74.6 ± 13.3</td>
</tr>
<tr>
<td>Height, m</td>
<td>1.73 ± 8.8c</td>
<td>1.84 ± 8.1</td>
<td>1.73 ± 9.4c</td>
<td>1.80 ± 5.4</td>
<td>1.76 ± 5.1</td>
<td>1.77 ± 7.3</td>
</tr>
<tr>
<td>Tidal volume</td>
<td>0.78 ± 0.05</td>
<td>0.95 ± 0.07</td>
<td>0.97 ± 0.1</td>
<td>1.21 ± 0.12</td>
<td>0.99 ± 0.08</td>
<td>0.94 ± 0.04</td>
</tr>
<tr>
<td>Vital capacity</td>
<td>4.61 ± 0.24c</td>
<td>5.66 ± 0.16c</td>
<td>4.41 ± 0.26c</td>
<td>5.52 ± 0.26b</td>
<td>4.21 ± 0.17</td>
<td>4.39 ± 0.18</td>
</tr>
<tr>
<td>FEV1,0 (%)</td>
<td>3.83 ± 0.24c</td>
<td>4.81 ± 0.22c</td>
<td>3.28 ± 0.24c</td>
<td>4.44 ± 0.21b</td>
<td>2.94 ± 0.25</td>
<td>3.23 ± 0.20</td>
</tr>
<tr>
<td>FEV1.0/FVC (%)</td>
<td>83.67 ± 2.8c</td>
<td>83.07 ± 2.1c</td>
<td>76.2 ± 2.8</td>
<td>78.68 ± 2.2</td>
<td>72.21 ± 3.0</td>
<td>73.2 ± 2.0</td>
</tr>
<tr>
<td>Compliance (%kPa⁻¹, means ± SE)</td>
<td>1.36 ± 0.12</td>
<td>1.54 ± 0.13</td>
<td>1.28 ± 0.06</td>
<td>1.79 ± 0.17</td>
<td>1.17 ± 0.12</td>
<td>1.47 ± 0.07</td>
</tr>
<tr>
<td>Lung tissue: static</td>
<td>2.63 ± 0.35</td>
<td>3.39 ± 0.37</td>
<td>3.40 ± 0.42</td>
<td>4.41 ± 0.46</td>
<td>3.95 ± 0.74</td>
<td>3.52 ± 0.64</td>
</tr>
<tr>
<td>Lung tissue: dynamic</td>
<td>1.99 ± 0.15#c</td>
<td>3.36 ± 0.35</td>
<td>2.31 ± 0.22#c</td>
<td>3.16 ± 0.28#</td>
<td>2.36 ± 0.23#</td>
<td>2.76 ± 0.18</td>
</tr>
<tr>
<td>Chest wall: static</td>
<td>3.32 ± 0.44</td>
<td>4.06 ± 0.85</td>
<td>2.09 ± 0.26</td>
<td>3.37 ± 0.28</td>
<td>2.30 ± 0.53</td>
<td>3.74 ± 0.76</td>
</tr>
</tbody>
</table>

Young sedentary (YS, n = 10); young habitually active (YA, n = 10); middle-aged sedentary (MS, n = 10); middle-aged habitually active (MA, n = 9); older sedentary (OS, n = 9); older habitually active (OA, n = 10); FEV1.0, forced expired volume (1 s); FVC, forced vital capacity (l/kPa). Componental compliance was computed over 1-liter tidal volume from functional residual capacity. Significant differences (P < 0.05): c young vs. middle-aged groups; b middle-aged vs. older groups; # young vs. older groups; * sedentary vs. habitually active groups; # static vs. dynamic compliance.
Methodological overview. Each subject attended three to four experimental sessions, at least 48 h apart, but at the same time of the day and in the same relation to previous meals. Subjects were first trained to provide reproducible resting, static pressure-volume relaxation curves and were familiarized with exercise and lung-volume maneuvers. During subsequent visits, the mechanical attributes of the lung tissue and chest wall were simultaneously determined during seated rest, along with both resting and exercising lung volumes, determined at each of three exercise intensities. Subjects were asked to avoid strenuous physical activity on each test day. All measurements were made in an air-conditioned laboratory at the host institution (Univ. of Wollongong) with subjects closely monitored to ensure maintenance of the prescribed workload and to verify normal cardio-vascular responses.

Lung volume measurement. At rest and during steady-state exercise, subjects performed spontaneous tidal breathing (1 min) followed by the standard maximal inspiratory and expiratory maneuvers to quantify the following static volumes: inspiratory capacity, expiratory reserve volume, and vital capacity (Table 1). Total lung capacity was derived using vital capacity and the age-predicted residual volume. End-inspiratory lung volume (EILV) and EELV were then determined. Standard flow-volume loops were also used to quantify dynamic lung volumes (Table 1).

Static pressure-volume measures and analysis. Static pressure-volume maneuvers were always completed at rest using the classic relaxation technique (23, 30) and performed over the entire vital capacity range. Commencing from total lung capacity, subjects exhaled a small and variable air volume before relaxing against an occluded airway. Using a stopcock, the experimenter controlled each expired volume by transiently occluding the airway (1–3 s). At each occlusion, subjects were trained to completely relax the primary respiratory and accessory muscles and the muscles of the oropharyngeal region and to hold the glottis open. Thus, at lung volumes above the intrinsic relaxation volume of the respiratory system (functional residual capacity), the elastic nature of the respiratory structures compressed the occluded lung volume, generating positive alveolar and airway pressures at each relaxation. At volumes below the functional residual capacity, the lung volume was decompressed on relaxation, resulting in negative alveolar and airway pressures. Respiratory pressure-volume data for each relaxation point were collected while subjects relaxed against a transiently occluded airway. With this technique, subjects provided 5–10 relaxation points during each complete maneuver, with an average of >30 reproducible pressure-volume points being obtained for each subject across the vital capacity excursion, from 5–10 successive maneuvers.

A hand-dipped esophageal balloon and catheter, connected to a differential pressure transducer (DP45–30, ± 8.6 kPa; Validyne, Northridge, CA), was inserted 40 cm beyond the external nares and used to approximate intrapleural pressure during these maneuvers. Static alveolar pressure was determined from mouth pressure at zero airflow using a second pressure transducer (DP45–14, ± 0.22 kPa; Validyne). Both transducers were coupled to a carrier demodulator (CD19A; Validyne). Respiratory flows were measured using a heated pneumotachograph (model 8430; Hans Rudolph, Kansas City, MO) and differential pressure transducer (DP45–30, ± 8.6 kPa; Validyne). Data were sampled at 50 Hz (DAS 1602, Metabyte; Keithley Data Acquisition, Taunton, MA), simultaneously visualized using an oscilloscope, and saved on computer. Flow and pressure calibrations were performed before each trial. Data collected included expired flow and transmural pressures for the lung (transpulmonary pressure, Ptp), chest wall (transthoracic pressure, Pm), and total respiratory system (transrespiratory pressure, Prs).

Respiratory flows and relaxation pressures were converted to pressure-volume coordinates for the lung tissue, total respiratory system, and the chest wall (by subtraction) for each subject using flow integration software (32, 34) and modeled mathematically using first-third order polynomials (Tablecurve; Jandel Scientific). Equation coefficients, the correlations of which always exceeded 0.9, were entered into analysis software to calculate static total respiratory system [Cst(tot)], lung tissue [Cst(tl)], and chest wall compliance [Cst(wt); Table 1] and lung tissue, chest wall, and total respiratory elastic work (34).

Respiratory static work components were computed over a 1-liter VT, commencing from either the resting or exercise-specific EELV (V1), which was superimposed on each subject’s resting static pressure-volume curve. This approach is justified since the elastic properties of the system had already been identified and should remain constant for a given (upright) posture and set of environmental conditions. These calculations are illustrated in Fig. 1 using schematic pressure-volume curves for the lung tissue, chest wall, and total respiratory system. Classically (Fig. 1A), a 1-liter volume displacement (line V1V2) defines the hypothetical V1, and the area between any one pressure-volume curve and the zero pressure axis will have the dimensions of work (work = pressure × volume). Using integration, one may compute the area under each of the transrespiratory pressure-volume curves. These areas quantify the elastic work performed on each tissue compartment during a volume change. For instance, during inspiration, energy is used to expand the lung tissue, so lung tissue elastic work will be defined by the area V1LUV2. Similarly, chest wall elastic work equates with area V1RCV2, which falls entirely to the left of the zero pressure axis, resulting in negative chest wall elastic work. That is, at EELV (V1), the chest wall has an inherent tendency to expand, and its stored elastic energy will assist lung tissue expansion. However, such stored energy is generally inadequate to entirely drive inspiration, so the inspiratory muscles are recruited to perform the remaining elastic work involved with lung

Fig. 1. Partitioning static work of the respiratory system over a 1-liter tidal volume, commencing at the end-expiratory lung volume (EELV or Vt). Lung tissue elastic work is defined by the area V1LUV2, chest wall elastic work equates with area V1RCV2, and total respiratory elastic work is the area V1SV2. In A, the EELV corresponds with the relaxation volume (VR) of the total respiratory system (VR = V1), but in B, it is below this relaxation volume (VR > V1; see text for details).
expansion. This work, known as the total respiratory elastic work, is the area under the transrespiratory pressure-volume curve ($V1SV2$) and represents the difference between lung tissue and chest wall elastic work. In this example (Fig. 1A), static inspiratory work equals area $V1SV2$.

However, the above analysis assumes that the EELV coincides with the intersection of the transrespiratory pressure-volume curve and the zero pressure axis ($V1$ in Fig. 1A), the so-called relaxation volume of the respiratory system ($Vr$). We have previously demonstrated that this assumption is not universally appropriate (34) and that the EELV is sometimes defined at points other than the intrinsic relaxation volume of the respiratory system (33). Accordingly, it is our view that one should always analyze respiratory elastic work commencing at the actual EELV rather than at the respiratory $Vr$. Figure 1B illustrates such an analysis, where the EELV is below the $Vr$. The line $V1V2$ still quantifies volume change, whereas lung tissue elastic work (area $V1LUV2$) and chest wall elastic work (area $V1RCV2$) remain similarly defined. However, two areas of total respiratory elastic work now exist: an area of negative work ($V1TVr$) and one of positive work ($VpSV2$). The absolute area of these regions corresponds with the area that is classically used to define static respiratory work ($V1SV2$), but the physiological impact of these separate regions is quite different, so each area must be measured separately. For example, inspiratory muscle work is defined by the area $VpSV2$ in Fig. 1, $A$ (where $Vr = V1$) and $B$, and expiratory muscle work is now present (area $V1TVr$, Fig. 1B only).

**Dynamic measures.** To separate inspired and expired air during exercise, and to minimize dead space, two sets of low-resistance, two-way, nonrebreathing valves (model 2700B; Hans Rudolph) were inserted between the mouthpiece and pneumotachograph. Dynamic lung tissue compliance ($C_{d,y}n$), computed over the same tidal range as $C_{d,y}$, was measured using the spontaneous $Vt$ and the $Pao$ difference between the points of end-inspiratory and end-expiratory zero airflow (20), with data collected at rest and during exercise. Oxygen uptake and carbon dioxide production were determined over 20-s intervals, using an open-circuit spirometry system (SensorMedics 2900). Gas analyzers were calibrated daily. Cardiac frequency was continuously monitored from the ear lobe using a pulse oximeter (Ohmeda Biox 3700e; BOC Health Care, Louisville, CO).

**Exercise protocol.** Subjects cycled at three steady-state intensities (15–20 min) on an electronically braked ergometer (50 rev min$^{-1}$; Excalibur Lode, Groningen, Netherlands) at one absolute (oxygen uptake 1.5 l min$^{-1}$) and two relative work rates [40 and 60% of the cardiac reserve (age-predicted maximal minus resting cardiac frequency)]. Workloads were administered in a balanced order across three visits and used to measure the intensity-specific EILV and EELV. In each trial, exercise commenced at 25 W (2 min), increasing in steps of 6–10 W every 20–30 s, until the target work rate was achieved. Data collection commenced after steady-state conditions were satisfied. At each exercise intensity, subjects performed spontaneous tidal breathing (1 min) followed by maximal inspiratory and expiratory maneuvers to measure lung volumes.

**Design and analysis.** This project was based on a two-factor design, with three levels of factor one (age: 20–30 years, 40–50 years, and >60 years) and two levels of the second factor (physical activity habit: sedentary and habitually active), with subjects nested within each cell. Data were analyzed using repeated-measures ANOVA and Tukey’s multiple-comparison post hoc procedure. Static and dynamic lung compliance were compared using paired $t$-tests. Alpha was set at the 0.05 level for all analyses. Data are reported as means ± SE unless stated otherwise. The use of a cross-sectional design, in combination with the current sample sizes and the physical activity screening methods, may impact on data interpretation and its extrapolation to the aging adult relative to data obtained using a longitudinal design.

**RESULTS**

**Static and dynamic compliance.** Resting static and dynamic lung volumes were within the ranges 93–114% and 84–126% of age-predicted values, respectively (Table 1). Representative pressure-volume data are displayed in Fig. 2. In general, static pressure-volume curves for the older subjects covered a smaller volume range, confirming a reduction in vital capacity with aging (Table 1). Maximal transmural pressures also decreased with aging, in particular at residual volume. Therefore, aging affected the shape of these pressure-volume relationships, showing a tendency for the chest wall curve to move rightward, with a simultaneous leftward displacement of the pulmonary curve.

Static compliance ranged from 1.17–1.79 l kPa$^{-1}$ for total respiratory system, 2.63–4.41 l kPa$^{-1}$ for the lung, and 2.09–4.06 l kPa$^{-1}$ for the chest wall (Table 1) and were within normal ranges for asymptomatic subjects (14, 31). This, in combination with normal lung function and our screening procedures, verified that we were studying the effects of aging in asymptomatic subjects.

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**Fig. 2.** Representative static pressure-volume curves for the total respiratory system (●), lung tissue (○), and chest wall (▼) obtained from young, middle-aged, and older subjects. Lung volumes are total lung capacity (TLC), functional residual capacity (FRC), and residual volume (RV).
Elastic work of breathing at rest. When averaged across physical activity habit, no significant differences in the total static work of breathing were found among the three age groups ($P > 0.05$; Table 2); $Y = 0.33 \pm 1^{-1} (\pm 0.03)$; $M = 0.39 \pm 1^{-1} (\pm 0.03)$; and $O = 0.43 \pm 1^{-1} (\pm 0.03)$. However, elastic work performed on both the lung ($Y = 0.79 \pm 0.05$; $M = 0.47 \pm 0.05$; $O = 0.43 \pm 0.05 \pm 1^{-1}$) and chest wall ($Y = -0.49 \pm 0.06$; $M = -0.12 \pm 0.07$; $O = 0.04 \pm 0.05 \pm 1^{-1}$) changed significantly with age ($P < 0.05$). The decline in lung tissue elastic work was $\sim 41\%$ from $Y$ to $M$ and $46\%$ from the $Y$ to $O$ groups. Chest wall elastic work became more positive with age ($P < 0.05$), changing by $\sim 75\%$ ($Y$ to $M$) and $110\%$ ($Y$ to $O$), indicating less elastic energy was being stored within the chest wall. Because absolute work reflects $V_T$ change, work was normalized to a $1$-liter $V_T$, and this same pattern persisted. Therefore, these observations were not the result of variations in breathing pattern but represented age-related changes in the mechanical properties of tissues, with aging affecting lung tissue and chest wall elasticity in opposite directions. Thus we accepted our first hypothesis, and we have demonstrated that, even in the absence of significant compliance changes, aging appears to affect chest wall mechanics to a greater extent than it does lung tissue mechanics.

An effect of habitual physical activity on the resting static work of breathing was only evident for the total respiratory system of the older groups, where the sedentary subjects had a greater static work than the habitually active subjects (Table 2; $P < 0.05$). No significant interaction between aging and habitual physical activity was evident for either lung tissue or chest wall elastic work ($P > 0.05$). Thus these results led us to reject our second hypothesis, and habitual physical activity appeared to not significantly interact with age-related changes in chest wall mechanics. Accordingly, we combined the sedentary and physically active subjects within each age category for subsequent analyses.

Elastic work of breathing during exercise. Elastic work of breathing, across physical activity habit, was affected by aging, with the total respiratory static work of breathing being significantly elevated in the middle-aged ($1.5 \cdot \text{min}^{-1}$ oxygen uptake, $P < 0.05$) and older subjects (all work rates, Table 2, $P < 0.05$). Absolute chest wall elastic work (reported in $J$) of the young and middle-aged groups increased $\sim 50\%$ from rest to exercise, whereas that of the older subjects increased approximately fivefold, with $V_T$ doubling. When work was normalized to a $1$-liter $V_T$, nonsignificant reductions in lung tissue elastic work were concurrently observed (Table 2; $P > 0.05$), with significantly more positive chest wall elastic work across age groups (Table 2; $P < 0.05$): $\sim 85\%$ ($Y$ to $M$) and $\sim 175\%$ ($Y$ to $O$). This general pattern was evident at each exercise intensity, replicating that seen at rest and indicating less energy was being stored within the chest wall at the EELV. Indeed, as age increased, the amount of energy storage at both EILV and EELV differed.

Chest wall elastic work is not always entirely defined by pressure-volume points falling to the left of the zero pressure axis. Because tidal excursions during exercise may cross this axis, producing positive $P_{th}$, chest wall elastic work can represent the summation of both negative and positive components (34), and we sought evidence for both components within each age group. In Fig. 1, the resting state is presented, and the thoracic relaxation volume is $4.4 \text{ l}$ above residual volume and...
Table 2. Static compliance and elastic work of breathing at rest and during steady-state exercise at an intensity of 40 and 60% of cardiac reserve and at an oxygen uptake of 1.5 l·min⁻¹ for 6 subgroups classified by age and physical activity habit

<table>
<thead>
<tr>
<th>Subject Group</th>
<th>YS</th>
<th>YA</th>
<th>MS</th>
<th>MA</th>
<th>OS</th>
<th>OA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rest</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total work</td>
<td>0.32 ± 0.04*</td>
<td>0.35 ± 0.04</td>
<td>0.39 ± 0.05</td>
<td>0.39 ± 0.04</td>
<td>0.50 ± 0.04</td>
<td>0.37 ± 0.23</td>
</tr>
<tr>
<td>Lung tissue</td>
<td>0.85 ± 0.07</td>
<td>0.74 ± 0.08</td>
<td>0.42 ± 0.06</td>
<td>0.52 ± 0.09</td>
<td>0.48 ± 0.08</td>
<td>0.37 ± 0.05</td>
</tr>
<tr>
<td>Chest wall</td>
<td>-0.56 ± 0.08*</td>
<td>-0.43 ± 0.09*</td>
<td>-0.05 ± 0.09</td>
<td>-0.18 ± 0.09</td>
<td>-0.04 ± 0.08</td>
<td>0.05 ± 0.06</td>
</tr>
<tr>
<td>40% Cardiac reserve C(\text{st(rs)})</td>
<td>1.23 ± 0.11</td>
<td>1.77 ± 0.14</td>
<td>1.44 ± 0.20</td>
<td>1.89 ± 0.21</td>
<td>1.49 ± 0.30</td>
<td>1.74 ± 0.18</td>
</tr>
<tr>
<td>C(\text{st(t)})</td>
<td>2.54 ± 0.49</td>
<td>2.99 ± 0.22</td>
<td>3.81 ± 0.62</td>
<td>4.44 ± 0.64</td>
<td>3.67 ± 0.75</td>
<td>3.54 ± 0.59</td>
</tr>
<tr>
<td>C(\text{st(w)})</td>
<td>3.40 ± 0.59</td>
<td>4.58 ± 0.59</td>
<td>2.59 ± 0.53</td>
<td>3.32 ± 0.51</td>
<td>3.19 ± 0.92</td>
<td>4.01 ± 0.62</td>
</tr>
<tr>
<td>Total work</td>
<td>0.51 ± 0.13*</td>
<td>0.89 ± 0.20</td>
<td>0.61 ± 0.11</td>
<td>0.56 ± 0.12</td>
<td>1.02 ± 0.17</td>
<td>0.83 ± 0.08</td>
</tr>
<tr>
<td>Lung tissue</td>
<td>0.96 ± 0.11</td>
<td>1.01 ± 0.12</td>
<td>0.53 ± 0.06</td>
<td>0.65 ± 0.07</td>
<td>0.69 ± 0.11</td>
<td>0.64 ± 0.05</td>
</tr>
<tr>
<td>Chest wall</td>
<td>-0.49 ± 0.10*</td>
<td>-0.10 ± 0.16</td>
<td>0.08 ± 0.11</td>
<td>-0.04 ± 0.15</td>
<td>0.31 ± 0.13</td>
<td>0.18 ± 0.08</td>
</tr>
<tr>
<td>60% Cardiac reserve C(\text{st(rs)})</td>
<td>1.16 ± 0.11</td>
<td>1.72 ± 0.12</td>
<td>1.37 ± 0.09</td>
<td>1.85 ± 0.25</td>
<td>1.41 ± 0.29</td>
<td>1.56 ± 0.11</td>
</tr>
<tr>
<td>C(\text{st(t)})</td>
<td>2.53 ± 0.52</td>
<td>3.07 ± 0.26</td>
<td>3.54 ± 0.39</td>
<td>4.35 ± 0.60</td>
<td>3.70 ± 0.77</td>
<td>3.60 ± 0.64</td>
</tr>
<tr>
<td>C(\text{st(w)})</td>
<td>3.30 ± 0.58</td>
<td>4.26 ± 0.57</td>
<td>2.27 ± 0.23</td>
<td>3.44 ± 0.55</td>
<td>3.16 ± 0.94</td>
<td>3.29 ± 0.41</td>
</tr>
<tr>
<td>Total work</td>
<td>0.45 ± 0.11*</td>
<td>0.89 ± 0.20</td>
<td>0.77 ± 0.10</td>
<td>0.61 ± 0.09</td>
<td>0.98 ± 0.16</td>
<td>0.84 ± 0.09</td>
</tr>
<tr>
<td>Lung tissue</td>
<td>0.93 ± 0.09</td>
<td>1.01 ± 0.13</td>
<td>0.58 ± 0.06</td>
<td>0.68 ± 0.07</td>
<td>0.68 ± 0.10</td>
<td>0.65 ± 0.06</td>
</tr>
<tr>
<td>Chest wall</td>
<td>-0.50 ± 0.10*</td>
<td>-0.10 ± 0.16</td>
<td>0.18 ± 0.11</td>
<td>-0.04 ± 0.12</td>
<td>0.28 ± 0.12</td>
<td>0.18 ± 0.08</td>
</tr>
<tr>
<td>1.5 l·min⁻¹ oxygen uptake C(\text{st(rs)})</td>
<td>1.18 ± 0.10</td>
<td>1.76 ± 0.15</td>
<td>1.48 ± 0.24</td>
<td>1.79 ± 0.19</td>
<td>1.40 ± 0.30</td>
<td>1.69 ± 0.17</td>
</tr>
<tr>
<td>C(\text{st(t)})</td>
<td>2.55 ± 0.51</td>
<td>2.95 ± 0.22</td>
<td>3.56 ± 0.43</td>
<td>4.32 ± 0.65</td>
<td>3.40 ± 0.57</td>
<td>3.61 ± 0.63</td>
</tr>
<tr>
<td>C(\text{st(w)})</td>
<td>3.79 ± 0.59</td>
<td>4.67 ± 0.67</td>
<td>2.61 ± 0.51</td>
<td>3.09 ± 0.48</td>
<td>3.17 ± 0.94</td>
<td>3.79 ± 0.63</td>
</tr>
<tr>
<td>Total work</td>
<td>0.42 ± 0.11*</td>
<td>0.85 ± 0.20</td>
<td>0.78 ± 0.11</td>
<td>0.49 ± 0.13</td>
<td>1.02 ± 0.17</td>
<td>0.82 ± 0.08</td>
</tr>
<tr>
<td>Lung tissue</td>
<td>0.92 ± 0.09</td>
<td>0.98 ± 0.12</td>
<td>0.59 ± 0.05</td>
<td>0.62 ± 0.08</td>
<td>0.74 ± 0.05</td>
<td>0.63 ± 0.05</td>
</tr>
<tr>
<td>Chest wall</td>
<td>-0.51 ± 0.09*</td>
<td>-0.12 ± 0.17</td>
<td>0.18 ± 0.12</td>
<td>-0.09 ± 0.16</td>
<td>0.31 ± 0.15</td>
<td>0.18 ± 0.08</td>
</tr>
</tbody>
</table>

Young sedentary (YS, n = 10); young habitually active (YA, n = 10); middle-aged sedentary (MS, n = 10); middle-aged habitually active (MA, n = 10); older sedentary (OS, n = 9); and older habitually active (OA, n = 10). Data are mean ± SE. Compartamental compliance was computed over 1-liter tidal volume commencing at functional residual capacity. Significant differences (P < 0.05) are indicated: * young vs. middle-aged groups; vs. † young vs. older groups; ‡ sedentary vs. habitually active groups. Total respiratory, \(C_{\text{st(rs)}}\); lung tissue, \(C_{\text{st(t)}}\); chest wall, \(C_{\text{st(w)}}\).

above \(V_2\). During exercise, large \(V_T\) can move the point \(V_2\) above this intrinsic relaxation volume. At rest, as predicted, lung tissue elastic work, normalized to \(V_T\), was entirely composed of positive components, whereas chest wall work contained predominantly negative components (Fig. 4). Thus the lungs gained, whereas the chest wall dissipated energy during inspiration.

During exercise, the young subjects required the greatest amount of elastic work to inflate the lung, but they also had more energy stored within the chest wall (Fig. 4; \(P < 0.05\)). Each age group contained individuals who required positive (inspiratory muscle) work to be performed on the chest wall during rest and exercise (Fig. 4). However, during exercise, and unique to this investigation, the older subjects experienced a greater shift from performing predominantly negative chest wall elastic work at rest to accomplishing more positive (inspiratory muscle) work when exercising (Fig. 4; \(P < 0.05\)). This change reflected a modification in the manner in which energy was being stored within the chest wall over the respiratory cycle, as reflected in the ratios of positive-to-negative chest wall elastic work components. At rest, these ratios were: 0.04 (Y), 0.64 (M), and 1.00 (O). During exercise, the ratios of these elastic work components increased for each group but then remained stable in the young group (0.49, 0.42, 0.42; for 1.5 l·min⁻¹, 40 and 60% of cardiac reserve, respectively). In the middle-aged subjects, these positive and negative work components were approximately equal, eliciting ratios of 0.94 (1.5 l·min⁻¹), 1.32 (40%), and 1.12 (60%). In the older subjects, positive chest wall elastic work was increased to be two- to threefold greater than the negative chest wall elastic work: 3.44 (1.5 l·min⁻¹), 2.57 (40%), and 2.87 (60%). Therefore, energy stored within the chest wall of the younger person fulfilled similar inspiratory and expiratory roles during exercise. However, positive chest wall work, which assists expiration, became a major component during exercise in the two older groups.

Static inspiratory muscle work is derived from the summation of all the positive and negative elastic work components of the lung and chest wall during a tidal inflation (34). Each age group had similar inspiratory muscle work at rest (\(P > 0.05\)), which increased during exercise (Fig. 4). However, these changes were both exercise intensity and age independent, resulting in each group performing similar inspiratory muscle work during exercise (\(P > 0.05\)), but modified the respiratory phase in which this work was performed.

Lung volumes during exercise. Minute ventilation was equivalent across the three age groups at rest and at each exercise intensity (\(P > 0.05\)), but there was a significant interaction with physical activity habit, with sedentary subjects achieving a lower exercise ventilation (\(P < 0.05\)) due to lower absolute workloads. The young subjects produced greater respiratory frequencies, both at rest and during each of the exercise intensities, than either the middle-aged (\(P < 0.05\)) or older subjects (\(P < 0.05\)). Conversely, \(V_T\) were smaller in this age group (\(P < 0.05\)). Whereas the young and middle-aged habitually active subjects increased \(V_T\) more than their sedentary counterparts, this trend was not apparent for the older subjects (\(P > 0.05\)); at 60% exercise intensity, YS 1.94 (±0.1)
vs. YA 2.52 (±0.09) l of BTPS, and MS 2.26 (±0.14) vs. MA 2.87 (±0.13) l of BTPS (all \( P < 0.05 \)).

During steady-state exercise, EELV showed little change from rest (Fig. 5). On the contrary, EILV for each group increased during exercise, with this change being significantly more apparent for the young and middle-aged habitually active subjects (\( P < 0.05 \)). Thus \( V_T \) increases during exercise were mainly accomplished by encroaching upon the inspiratory reserve volume. Clear differences were apparent between the vital capacities and EILV of the sedentary and habitually active subjects in both the young and middle-aged adults (Fig. 5; \( P < 0.05 \)). The failure to observe this pattern in the older subjects is intriguing but remains inexplicable.

DISCUSSION

To our knowledge, this is the first investigation in which chest wall and lung tissue mechanics were simultaneously measured in aging adults, across levels of habitual physical activity. Our principal observation was that aging was accompanied by a redistribution of the manner in which elastic energy was stored within the chest wall during the respiratory cycle. With increments in both age and work rate, a parallel displacement of the chest wall pressure-volume curve resulted in a shift from energy being stored primarily during expiration to an equivalent or greater energy storage during inspiration. This means that, in asymptomatic aging adults, less elastic energy was used during inspiration, but more stored energy was used to drive expiration, particularly as exercise intensity elevated. Therefore, it appeared that reciprocal changes in lung tissue and chest wall elastic work with senescence, although deviating significantly from that which existed in young adults, did not result in a significant elevation in the static work performed by the inspiratory muscles. Instead, these changes merely represented a redistribution of the tissues on which, and the phase of the respiratory cycle during which, this work was performed. Because this pattern was also evident when elastic work was normalized for differences in \( V_T \), these changes, then, are believed to represent a physiological consequence of aging in asymptomatic individuals, which was largely independent of age-related changes in ventilatory pattern. This appears to occur concomitantly with age-related reductions in pulmonary elastic work and increased expiratory flow resistance and appears to be largely unaffected by physical activity habit.

Fig. 4. Chest wall, lung tissue, and inspiratory muscle static work of breathing in young (Y), middle-aged (M), and older (O) adults normalized to a 1-liter tidal volume and recorded at rest (top) and during 3 levels of steady-state exercise (bottom 3 panels): 40 and 60% of cardiac reserve and oxygen uptake of 1.5 l·min\(^{-1}\). a, b, and c, Significant differences between the young and middle-aged, between middle-aged and older, and between the young and older subjects (\( P < 0.05 \)).

Fig. 5. Changes in vital capacity (VC), end-inspiratory lung volume (EILV), and EELV of sedentary (open symbols) and habitually active (closed symbols) young, middle-aged, and older adults measured at rest and during 3 levels of steady state: 40 and 60% of cardiac reserve and oxygen uptake of 1.5 l·min\(^{-1}\). *Significant difference between subjects of a different physical activity habit within each age group (\( P < 0.05 \)).
Aging and static respiratory mechanics. Aging resulted in the horizontal displacement of the static pressure-volume relationships of the lung and chest wall toward the zero pressure axis, without affecting compartmental compliance. As a result, the static recoil pressures of the lung tissue and chest wall decreased, and elastic work was altered. These data show that less pressure was required to expand the aging lung, whereas the aging chest wall possessed a lower inherent recoil pressure at the EELV. Previously, Pierce and Ebert (27) and Thomas et al. (36) reported similar displacements of the pulmonary and thoracic compliance curves. However, those data were not simultaneously measured in the same sample but were independently approximated in the absence of information relating to the other compartment.

The lower $P_{TV}$ in the older subjects, across lung volumes, was possibly associated with an age-related degeneration of the alveolar elastic matrix and small airways (4, 18, 22). Our observations reveal these changes appeared to affect the lungs equally over the entire $V_T$ range so that lung tissue compliance remained stable. When lung tissue elastic work was normalized for $V_T$, it was clear these age-related changes largely occurred before the fifth decade while tending to remain somewhat stable thereafter. Such a linear, age-related changes in pulmonary function have previously been observed (19). During exercise, lung tissue elastic work for each group increased approximately one- to twofold, and roughly in proportion to the change in $V_T$, but consistently remained lower in the two older groups. These are not novel findings. However, what is new is the observation that chest wall mechanics changed when the younger adults exercised and in aging adults during both rest and exercise.

In the young group, elastic energy stored within the chest wall was transferred to the lungs during a resting inspiration, with this reciprocal energy exchange minimizing total respiratory static work. This pattern has been known for $>50$ years (23, 25). However, on average, the chest wall compliance curves of the middle-aged and older groups crossed the zero pressure axis at lower lung volumes, and positive $P_{RV}$ were observed within the $V_T$ range. This change may be related to a reduced mobility of the aging chest wall, accompanying changes to intrathoracic joint function (9, 28, 29), and it resulted in the performance of positive chest wall elastic work during inspiration.

Consequently, the nature of energy storage within the resting chest wall was altered, becoming less negative (more positive) with increasing age. This phenomenon was not observed to a significant extent in the young subjects until they commenced exercise, whereas exercise made this feature more pronounced in the middle-aged and older subjects. Through the use of more sophisticated analyses (34), we have now demonstrated that negative elastic energy, apparently lost from the aging chest wall at rest (Fig. 4), appears as positive elastic energy and on the opposite side of the zero pressure axis. This elastic energy is a consequence of inspiratory muscle work performed on the chest wall and is necessary to inhale above its inherent, albeit reduced, relaxation volume.

All groups exhibited positive chest wall work during exercise, which increased with aging, while negative work decreased. This stored positive energy drives expiration, particularly as exercise intensity is increased. Whereas the young subjects consistently displayed the same ratio of more negative and less positive work across all exercise intensities, this ratio in the middle-aged subjects approached unity, and it became more positive and less negative in the older group. Indeed, the increase in stored positive energy occurred at an equivalent rate to the reduction in negative elastic energy. Thus total stored elastic energy was modified in both its form and magnitude with aging. We suggest that the mechanism driving this mechanical change was associated with increments in the $V_T$ and EELV relative to vital capacity. Specifically, in the older group, the EELV was more likely to be located on the flatter portion of the chest wall compliance curve and above the intrinsic relaxation volume of the chest wall.

The physiological significance of this modified chest wall elastic work and its reciprocal energy storage is found within its impact on inspiratory muscle work, which is most pronounced during exercise. Stored elastic energy in the aging respiratory system appears to contribute less to lung inflation. However, since less work is required to inflate the aging lung, aging did not significantly affect inspiratory muscle work (Fig. 5).

Ventilatory responses during exercise. Exercise-induced increments in minute ventilation were achieved via modifications in $V_T$ and breathing frequency, with the latter increasing its contribution only after $V_T$ approached its asymptote. The balance between these ventilatory components is achieved in such a manner that flow-resistive and elastic work are combined to minimize total respiratory work (23, 24). In the current study, early $V_T$ increments were primarily attributable to recruitment of the EILV, rather than the EELV, in agreement with previous reports (2, 31, 41). Because all three compliance curves for each age group were linear over the lower $V_T$ range, we speculated that $V_T$ excursions during exercise would fall mostly over the more compliant regions of these curves. However, since the EILV of the older subjects was close to the vital capacity, this placed the EILV on the flatter portion of the total respiratory curve. More recently, Teramoto et al. (35) have shown that, during exercise, aging adults also displace the abdominal content to a greater extent than do younger adults. Thus, although static inspiratory muscle work did not differ at the current work rates, the additional chest wall elastic work may represent a mechanical impediment to heavier exercise in the older subjects.

Conclusion. The first aim of this project was to simultaneously quantify age-related changes in lung tissue and chest wall mechanics. We found that aging caused parallel and opposite displacements of the static pressure-volume relationships of these two compartments, without changes in compliance. We have shown that aging affects energy storage within the chest wall, which is modified both in its size and form (negative vs. positive) with increasing age, such that less elastic energy was used during inspiration, but more stored energy was available to drive expiration. These changes are physiological consequences of asymptomatic aging, as they were largely independent of differences in ventilatory pattern. However, although such changes did not alter inspiratory muscle work, they did lead to a redistribution of this work between tissue compartments. The second aim of this project was to test the hypothesis that habitual physical activity would counteract age-related changes in chest wall mechanics, eliciting a relative reduction in the static work of breathing. We found no evidence to support this supposition.
REFERENCES


