Postural changes in lung volumes and respiratory resistance in subjects with obesity

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Watson, R. A., and N. B. Pride. Postural changes in lung volumes and respiratory resistance in subjects with obesity. J Appl Physiol 98: 512–517, 2005. First published October 8, 2004; doi:10.1152/japplphysiol.00430.2004.—Reduced functional residual capacity (FRC) is consistently found in obese subjects. In 10 obese subjects (mean ± SE age 49.0 ± 6 yr, weight 128.4 ± 8 kg, body mass index 44 ± 3 kg/m²) without respiratory disease, we examined 1) supine changes in total lung capacity (TLC) and subdivisions, 2) whether values of total respiratory resistance (Rrs) are appropriate for mid-tidal lung volume (MTLV), and 3) estimated resistance of the nasopharyngeal airway (Rnp) in both sitting and supine postures. The results were compared with those of 13 control subjects with body mass indexes of <27 kg/m². Rrs at 6 Hz was measured by applying forced oscillation at the mouth (Rrs,mo) or the nose (Rrs,na); Rnp was estimated from the difference between sequential measurements of Rrs,mo and Rrs,na. All measurements were made when subjects were seated and when supine. Obese subjects when seated had a restrictive defect with low TLC and FRC-to-TLC ratio; when supine, TLC fell 80 ml and FRC fell only 70 ml compared with a mean supine fall of FRC of 730 ml in control subjects. Values of Rrs,mo and Rrs,na at resting MTLV in obese subjects were about twice those in control subjects in both postures. Relating total respiratory conductance (1/Rrs) to MTLV, the increase in Rrs,mo in obese subjects was only partly explained by their reduced MTLV. Rnp was increased in some obese subjects in both postures. Despite the increased extrapulmonary mass load in obese subjects, further falls in TLC and FRC when supine were negligible. Rrs,mo at isovolume was increased. Further studies are needed to examine the causes of reduced TLC and increases in Rrs,mo and sometimes in Rnp in obese subjects.

METHODS

Subjects

Ten obese subjects with a body mass index of ≥30 kg/m² and 13 control subjects with a body mass index of ≤27 kg/m² were studied. None of the subjects had clinical evidence of pulmonary disease, abnormality of the chest radiograph, or evidence of airflow obstruction [forced expiratory volume in 1 s (FEV1)/vital capacity (VC) of >70%] (Table 1); one of the obese subjects was aware of nasal obstruction but had not sought treatment for this. Three of the obese subjects were smokers, and three were former smokers. The control subjects were younger and shorter than the obese subjects and had never smoked. Written consent was obtained from all subjects, and the protocol was approved by the Research Ethics Committee of this medical school.

Methods

Spirometry was measured with a dry spirometer (Fukuda Spiro-analysen ST-250). TLC and subdivisions were measured in the erect and supine positions in the 10 obese subjects and in 5 of the control subjects using a Morgan Benchmark and the multibreath helium dilution technique (9). Subjects were positioned comfortably with the mouthpiece, and when relaxed and breathing regularly, they were turned on to the circuit at the end of a tidal expiration. Occasional deep inhalations were made, and helium equilibrium was reached in ~3 min, and the subject was then asked to breathe in to TLC followed by a slow VC. In all 13 controls, TLC was measured in the seated position using body plethysmography (14).

Forced Oscillation Technique

Forced oscillation was applied at the airway opening during normal tidal breathing to measure respiratory impedance. The subject supported the cheeks and floor of the mouth with the palms of the hands to minimize dissipation of the applied flow in the upper airway. The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.
head and neck were kept in a neutral to slightly extended position, while the subject maintained tidal breathing via a large-bore mouthpiece and with a nose clip in place.

The oscillation apparatus consisted of a loudspeaker attached to a tube leading to a screen pneumotachograph. A complex signal of sinusoidal soundwave oscillation, containing all harmonics of 2 to 26 Hz, was applied by the loudspeaker (25). The signal was presented as preprogrammed pseudorandom noise, the sequence being repeated every 0.5 s for a 16-s period. During oscillation, airway opening pressure (applied peak-to-peak signal of <2 cmH2O) and airflow were recorded by two identical differential transducers (Validyne MP45) with carefully matched short tubing, which gave adequate matching up to 30 Hz. Pressure and flow signals were fed into a Fourier analyzer, ensemble averaged in the frequency domain over the measurement period of 16 s, and calculated to give the values of impedance at 2-Hz intervals from 2 to 26 Hz. The derived values were the time-averaged mean of inspiratory and expiratory impedance over the several breaths of the 16-s period. The in-phase component of impedance, Rrs, is an index of airflow resistance analogous to resistance derived by other methods, such as body plethysmography (16); ns, not significant; VC, vital capacity; RV, residual volume; FRC, functional residual capacity; FEV1, forced expiratory volume in 1 s; %pred, percent predicted; M, male; F, female.

Mouth pressure was measured via a sidearm just distal to the mouthpiece. The nose was clipped. For Rrs,na, a small, tightly fitting, transparent nasal mask (as used for continuous positive airway pressure) was worn while the mouth was held shut. The oscillation apparatus was coupled to the central opening in the nasal mask, and a port in the mask allowed the airway opening pressure to be measured. Care was taken to ensure that there was no leak around the mask and that the mask did not compress the nasal alae. Preliminary tests showed that dissipation of the forcing signal within the mask was negligible. To estimate Rnp, averaged results from three 16-s periods of Rrs,mo measurement were subtracted from a similar set of three 16-s periods of Rrs,na obtained either immediately before or immediately after the measurement of Rrs,mo. Care was taken to ensure the monitored breathing pattern was similar during mouth and nasal breathing. The assumption that Rnp = Rrs,na − Rrs,mo implies that resistance of the oral cavity is negligible when the mouth is held open by a large-diameter mouthpiece. For measurements in the supine posture, the oscillation apparatus was supported by a gantry over the subject, who lay supine on a couch. Care was taken to ensure that a similar, slightly extended position of the head and neck was sustained in this position. The two differential pressure transducers were positioned in the perpendicular axis so that their orientation was not altered between sitting and supine postures. In both postures at the end of each 16-s oscillation measurement, a full inspiration was made to relate tidal volume to TLC and so calculate absolute MTLV and FRC.

### Data Analysis and Statistics

Reported values of Rrs are the mean of three 16-s periods of oscillation. To assess the influence of different lung volumes (VT), we calculated specific Rrs (SRs; Rrs × MTLV). In addition, because in normal subjects there is an approximately linear fall in airway conductance (Gaw) as VT falls with an intercept close to residual volume (RV) (26), we plotted total respiratory conductance (Grs), the reciprocal of Rrs, against the absolute MTLV at which Rrs was measured. Differences between postures and groups were assessed by paired and unpaired t-tests, respectively. P values ≥0.5 were considered not significant (ns).

### RESULTS

#### Postural Changes in TLC and Subdivisions

**Control subjects.** The five subjects who had TLC and subdivisions measured in both positions using multibreath helium dilution were older, taller, and heavier (mean age 57 yr, height 1.77 m, weight 71 kg) than the total control group and had a mean TLC of 6.55 liters (97% of predicted), FRC of 3.58 liters (104% of predicted), VC of 4.85 liters (118% of predicted), RV of 1.70 liters (75% of predicted), FEV1 of 3.81 liters (116% of predicted), and FEV1/VC of 79%. On adopting the supine position, there were small mean falls in TLC (190 ml), VC (10 liters), and RV (30 ml) (all P >0.05) and a fall in FRC of 730 ml (P = 0.048) (Fig. 1). This small fall in TLC duplicated numerous previous studies (1, 4, 29, 38), so in the total group of 13 control subjects whose seated volumes are shown in Table 1, supine FRC was obtained by measuring supine inspiratory capacity and assuming the individual’s supine TLC was 200 ml smaller than the seated TLC measured by plethysmography. Mean supine FRC was 2.69 ± 0.2 liters (SE), giving a mean fall in FRC of 740 ml (P < 0.0001, seated vs. supine).

**Obese subjects.** When seated, TLC, VC, FRC, expiratory reserve volume, and FEV1, expressed as %predicted, were all smaller than in control subjects (P < 0.05); RV and FEV1/VC were similar in control and obese subjects, but %FRC/TLC

### Table 1. Characteristics and lung volumes of subjects

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Control</th>
<th>Obese</th>
<th>Supine</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>n</td>
<td>13</td>
<td>10</td>
<td></td>
<td></td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>23 ±1</td>
<td>24 ±1</td>
<td>24 ±1</td>
<td></td>
</tr>
<tr>
<td>TLC, liters</td>
<td>5.82 ±0.3</td>
<td>5.30 ±0.3</td>
<td>5.22 ±0.3</td>
<td>0.021</td>
</tr>
<tr>
<td>%pred</td>
<td>100 ±1</td>
<td>86 ±1</td>
<td>91 ±1</td>
<td></td>
</tr>
<tr>
<td>VC, liters</td>
<td>4.23 ±0.3</td>
<td>3.49 ±0.3</td>
<td>3.37 ±0.3</td>
<td>0.043</td>
</tr>
<tr>
<td>%pred</td>
<td>109 ±6</td>
<td>89 ±6</td>
<td>91 ±5</td>
<td></td>
</tr>
<tr>
<td>RV, liters</td>
<td>1.59 ±0.1</td>
<td>1.81 ±0.1</td>
<td>1.85 ±0.1</td>
<td></td>
</tr>
<tr>
<td>%pred</td>
<td>92 ±6</td>
<td>91 ±5</td>
<td>91 ±5</td>
<td></td>
</tr>
<tr>
<td>FRC, liters</td>
<td>3.43 ±0.2</td>
<td>2.29 ±0.2</td>
<td>2.22 ±0.2</td>
<td></td>
</tr>
<tr>
<td>%pred</td>
<td>115 ±4</td>
<td>72 ±4</td>
<td>94 ±3</td>
<td>0.0002</td>
</tr>
<tr>
<td>FRC/TLC, %</td>
<td>69 ±2</td>
<td>54 ±3</td>
<td>54 ±3</td>
<td>0.00001</td>
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<tr>
<td>FEV1, liters</td>
<td>3.51 ±0.2</td>
<td>2.80 ±0.3</td>
<td>2.75 ±0.3</td>
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<tr>
<td>%pred</td>
<td>108 ±6</td>
<td>86 ±7</td>
<td>81 ±2</td>
<td></td>
</tr>
<tr>
<td>FEV1/VC, %</td>
<td>84 ±2</td>
<td>82 ±2</td>
<td>82 ±2</td>
<td></td>
</tr>
</tbody>
</table>

Values are means ± SE; n, no. of subjects. BMI, body mass index; TLC, total lung capacity measured by multibreath helium *equilibrium or body plethysmography (16); ns, not significant; VC, vital capacity; RV, residual volume; FRC, functional residual capacity; FEV1, forced expiratory volume in 1 s; %pred, percent predicted; M, male; F, female.
was much lower in obese subjects ($P = 0.0001$) (Table 1). Hence, obese subjects had a moderate restrictive defect with low FRC/TLC. On adopting the supine position, the mean falls in TLC of 80 ml ($P = \text{ns}$) and in VC of 120 ml ($P = \text{ns}$) were slightly smaller than in control subjects. The mean fall in FRC was only 70 ml ($P = \text{ns}$) and contrasted with the fall of 740 ml in control subjects ($P < 0.0001$).

**Postural Changes in Rrs and Conductance**

**Control subjects.** Seated values were typical for healthy subjects (30). When supine, mean Rrs,mo rose by 39% ($P < 0.0001$), whereas mean MTLV fell by 0.82 liter ($P < 0.0001$) and SRrs at the mouth (SRrs,mo) rose by 9% ($P = \text{ns}$). Plotting mean sitting and supine values of Grs at the mouth (Grs,mo) against absolute Vt indicated that, assuming the Grs-Vt slope had an intercept on the volume axis close to RV, the supine fall in Grs,mo appeared appropriate for the fall in MTLV (Fig. 2).

**Obese subjects.** Mean values of seated Rrs,mo were twice those in control subjects. Some of this increase in Rrs,mo was due to the smaller MTLV in obesity. Because mean MTLV in seated obese subjects and supine control subjects was nearly identical (Table 2), mean values of Grs,mo in the two groups could be compared at isovolume (~2.84 liters). At this volume, Grs,mo in the obese subjects was only 65% of the mean value in supine control subjects ($P = 0.0058$) (Table 2, Fig. 2). When supine, Rrs,mo rose by 38% ($P = 0.0022$) and MTLV fell by a mean of 0.21 liter (somewhat greater than the 0.07-liter fall in supine FRC), giving a 27% rise in SRrs,mo ($P = 0.065$). The fall in Grs,mo was somewhat larger than expected for the small fall in MTLV (Fig. 2).

**Comparison of Rrs Breathing via the Nose and via the Mouth**

The obese subject who reported nasal obstruction had a very high Rrs,na and Rnp (12.44 sit, 19.08 cmH$_2$O·l$^{-1}$·s supine), which was much greater than any of the other values so that his results have been excluded from the comparison with control subjects. Mean Rrs,na in the nine remaining obese subjects was approximately twice the value in control subjects in both sitting and supine postures, similar to the proportionate differences in Rrs,mo. All of these differences between control and obese subjects were statistically significant ($P < 0.007$) (Table 3). Part of the increase in Rrs,na in obesity reflects the higher value of intrathoracic resistance. However, the derived value of seated Rnp in obesity was significantly higher than in control subjects when seated and nearly so when supine ($P = 0.055$); four subjects had values greater than or equal to control mean + 2 SD. The absolute increase in mean supine Rnp was similar in both groups but was only statistically significant in the control subjects.

**DISCUSSION**

In the present study, we found that in obese subjects 1) TLC and FRC were both reduced when seated, but further falls when supine (80 and 70 ml, respectively) were smaller than in control subjects; 2) Rrs,mo was increased in part due to

<table>
<thead>
<tr>
<th>Grs,mo, cmH$_2$O·l$^{-1}$·s</th>
<th>Control (n = 13)</th>
<th>Obese (n = 10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting</td>
<td>Supine</td>
<td>Sitting</td>
</tr>
<tr>
<td>2.02 ± 0.1</td>
<td>2.80 ± 0.1</td>
<td>4.53 ± 0.5</td>
</tr>
<tr>
<td>0.52 ± 0.03</td>
<td>0.38 ± 0.01</td>
<td>0.23 ± 0.02</td>
</tr>
<tr>
<td>3.64 ± 0.1</td>
<td>2.86 ± 0.1</td>
<td>2.84 ± 0.2</td>
</tr>
<tr>
<td>7.22 ± 0.4</td>
<td>7.91 ± 0.4</td>
<td>12.77 ± 1.5</td>
</tr>
<tr>
<td>0.14 ± 0.01</td>
<td>0.13 ± 0.01</td>
<td>0.09 ± 0.01</td>
</tr>
</tbody>
</table>

Values are means ± SE; *n*, no. of subjects. Rrs,mo and Grs,mo, total respiratory resistance and conductance, respectively, while breathing via the oral route; TLC, midtidal lung volume at which measurements of respiratory resistance were made; SRrs,mo and SGrs,mo, specific Rrs,mo and Grs,mo, respectively. $\dagger P < 0.0001$, $\ddagger P < 0.004$ between postures within each group of subjects. $*P < 0.0006$, $\ddagger P < 0.003$ between obese and control subjects in the same posture.

Fig. 1. Postural changes in total lung capacity (TLC) and subdivisions as measured by multibreath helium dilution. See text for further details of volume changes. FRC, functional residual capacity; RV, residual volume.

Fig. 2. Postural changes in mean total respiratory conductance (Grs) plotted against the midtidal lung volume (MTLV) in 10 obese subjects and 13 control subjects, both sitting and supine. Symbols on horizontal axis indicate mean values of RV in control (●) and obese (○) subjects. TLC in control subjects was from records of tidal volume and inspiratory capacity, assuming that TLC measured by body plethysmography in the seated position fell by 200 ml when supine. In the obese subjects, TLC measured by multibreath helium dilution in both postures was used as the reference volume to derive MTLV.
The normal plot of Gaw vs. VL as the reference curve. Not by that change, Briscoe and DuBois recommended using a catheter may induce contraction of the muscles of the pharyngeal wall. The present method, unlike most other methods for measuring Rnp, estimates the time-averaged resistive load imposed by the nasopharynx over several breaths (including any effects of linearity of nasal flow resistance), which can be directly compared with the resistive load during oral breathing. Because measurements of Rrs,mo and Rrs,na were consecutive, we needed to be sure that the breathing pattern and MTLV were similar for the 16 s of each measuring period. In the present experiments, values of Rrs,na were obtained at a slightly higher MTLV than Rrs,mo, slightly reducing the contribution of intrapulmonary resistance to Rrs,na. This would counter the overestimate of Rnp that results from ignoring the contribution of the resistance of the oral cavity (0.2–0.5 cmH2O·L−1·s−1 with a large-diameter mouthpiece) (2, 12) to total Rrs,mo. Oral cavity resistance does not change when moving from sitting to supine posture (2). With a small FRC, parts of the lung may not be ventilated during multibreath helium equilibration. We tried to avoid this by taking occasional deep inhalations during equilibration. In four seated obese subjects, a direct comparison of body plethysmography and multibreath helium dilution gave mean values of FRC of 2.50 and 2.51 liters and of TLC of 5.12 liters (89% predicted) and 4.96 liters (86% predicted), respectively.

breathing at smaller VL but in addition Rrs,mo was increased at isovolume; and 3) Rnp was sometimes increased.

Technical

There have been few previous studies of airflow resistance in both sitting and supine postures or estimates of the proportion of total airflow resistance provided by the nasopharyngeal airway during normal tidal breathing. The forced oscillation method we used averages inspiratory and expiratory resistance over several breaths, as is usually the case with the esophageal balloon-catheter method used to measure total pulmonary resistance. Forced oscillation has major advantages over the latter method in being noninvasive and in avoiding the problem of obtaining reliable measurements of esophageal pressure in the supine posture, but the airflow resistance measured includes that of the chest wall (Rw) and lung tissue as well as Raw. At an oscillation frequency of 6 Hz at FRC, however, lung tissue resistance is negligible and Rw in normal subjects is ≈0.5 cmH2O·L−1·s−1 (5, 7, 21, 27); similar values of Rw have been found at 0.7 liter below and 1.1 liter above FRC (5). In considering airway dynamics at isovolume, we mainly describe the relation of airway dynamics to Vl. We have compared absolute values of Gs and Vl based on the classic observation of Briscoe and DuBois (8) that the relation between Gaw and Vl at differing Vl in an individual was constant over a very wide range of absolute values of Gaw in normal adults of different height, age, and gender. A consequence is that values of specific Gaw (Gaw/Vl) at FRC are also similar. To examine whether an intervention that changes Vl can be explained or not by that change, Briscoe and DuBois recommended using the normal plot of Gaw vs. Vl as the reference curve.

The noninvasiveness of the forced oscillation technique is of particular value in measuring Rnp where an intrapharyngeal catheter may induce contraction of the muscles of the pharyngeal wall. The present method, unlike most other methods for measuring Rnp, estimates the time-averaged resistive load imposed by the nasopharynx over several breaths (including

<table>
<thead>
<tr>
<th>Table 3. Postural changes in total respiratory resistance when breathing via the nose and derived nasopharyngeal resistance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control (n = 13)</td>
</tr>
<tr>
<td>Sitting</td>
</tr>
<tr>
<td>Rrs,na, cmH2O·L−1·s−1</td>
</tr>
<tr>
<td>MTLV,na, liters</td>
</tr>
<tr>
<td>Rrs,mo, cmH2O·L−1·s−1</td>
</tr>
<tr>
<td>MTLV,mo, liters</td>
</tr>
<tr>
<td>Rnp, cmH2O·L−1·s−1</td>
</tr>
</tbody>
</table>

Values are means ± SE. Rrs,na, total respiratory resistance while breathing via the nose; Rrs,mo, total respiratory resistance while breathing via the mouth; MTLV,na and MTLV,mo, mean MTLV at which measurements were made in the 2 postures. Rnp, nasopharyngeal resistance as Rrs,na−Rrs,mo. \(^a\)One of the obese subjects had a very high Rrs,na and Rnp and has been excluded (see text). \(^b\)P < 0.0001, \(^c\)P < 0.0004 between postures within each group of subjects. \(^d\)P < 0.0007, \(^e\)P < 0.05 between obese and control subjects in the same posture.

Changes in TLC and Subdivisions

Seated. The best recognized abnormalities in seated obese subjects, confirmed in this study, are reduction in FRC and expiratory reserve volume. In normal subjects, FRC during muscle relaxation is reduced when intra-abdominal volume and pressure are increased experimentally, although the reduction is less than expected from the respective compliances of the lung, ribcage, and abdomen (19). In obesity, mass loading of the thorax and abdominal surface of the diaphragm presumably increases intra-abdominal pressure, and there is a reduction in total compliance of the respiratory system and particularly of the chest wall (28); these changes are thought to reduce the relaxation volume of the respiratory system. Most studies have found also a modest reduction in seated TLC (20, 28, 34, 38, 40, 41), although this is not entirely consistent (6, 32, 33). Recent studies have found systematic falls in VC as weight increases above predicted values (39) and rises in VC as excess weight is lost (11). Because any changes in RV are small, reduction in TLC therefore should occur when weight rises above the normal value. In the present results, the reduction in TLC of 14% of predicted values is slightly smaller than the reduction of 17% of predicted values in our laboratory’s earlier study (40). The mechanism reducing TLC in obesity has not been precisely defined. It is usually attributed to the same extrapulmonary factors that reduce FRC, with the caudal movement of the diaphragm being restricted by the enlarged abdomen at full inflation. However, in pregnancy (18), there is very little reduction in TLC, despite a large increase in intra-abdominal volume. In obesity, the major load is the subcutaneous abdominal fat, which is on average three to four times greater in volume than fat within the abdominal cavity (37). The extra-abdominal and extrathoracic fat presumably acts as a threshold inspiratory load (35), regardless of whether there is any effect on abdominal wall compliance. In obese subjects, maximum inspiratory pressure at FRC is relatively well preserved both seated (23, 40) and supine (40), so there is no a priori reason to suspect that impaired performance of inspiratory muscles is a factor in preventing full inflation.

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Supine. The mechanism of reduction in FRC on adopting the supine posture in healthy subjects appears to be twofold. First, gravity displaces the static pressure-volume curve of the relaxed chest wall to a smaller volume so that its neutral position falls from ~50% VC to 30% VC (~62–50% TLC); the pressure-volume curve of the lungs does not change, except perhaps close to RV. Second, there is a small reduction in TLC and VC due to an increase in volume of intrathoracic blood in the supine position (36). The combined effect is to reduce supine relaxation volume by ~0.7–0.8 liter. FRC in both postures in healthy subjects is believed to correspond to relaxation volume.

In obese subjects, when supine, gravity similarly would be expected to increase the mass effect of the abdomen on the abdominal surface of the diaphragm, but, as reported in a few previous studies (4, 38, 40), we found only a very small further fall in FRC and TLC. The mechanism of the lack of supine change in FRC and TLC requires further investigation; lack of further fall in FRC has a favorable effect in restricting increases in inspiratory work of breathing and deterioration in gas exchange during tidal breathing when supine.

Airflow Resistance Breathing via the Mouth

Because airway dimensions are dependent on Vt and lung recoil pressure, it is not surprising that the supine fall in FRC in normal subjects leads to an increase in airflow resistance. This effect was studied comprehensively by Linderholm (26) using upright and horizontal body plethysmographs to measure Raw, and an esophageal balloon catheter to measure pulmonary resistance over a wide range of Vt and lung recoil pressures. He found that changing from upright to horizontal postures did not alter significantly the mechanical properties of the lungs. In addition, he confirmed that, at volumes below seated FRC, the ΔGaw-ΔVt relationship was linear, with an intercept on the volume axis close to RV. Due to the additional effect of conductance across the chest wall, values of Grs at any Vt are systematically lower than values of Gaw; hence the ΔGrs-ΔVt plot is displaced to the right of the ΔGaw-ΔVt curve with a decreasing ΔGrs-ΔVt slope as Vt is increased toward TLC. However, at and below FRC, the normal ΔGrs-ΔVt slope approaches the ΔGaw-ΔVt slope, because, as Gaw falls, the relative importance of conductance across the chest wall declines, allowing the normal ΔGrs-ΔVt slope at small volume to be used as a reference curve for values of Grs in obesity (Fig. 2). In contrast, measuring SGrs (or SRRs) at small volumes can give misleading information because the intercept of the ΔGrs-ΔVt slope on the volume axis results in falls in SGrs for a given volume decrement progressively increasing as RV is approached.

In obesity, Rs,mo was about twice that in control subjects, both in the seated and supine positions, confirming our laboratory’s earlier study (40). The Grs-Vt plot shows that the reduction in Grs,mo in seated obese subjects was larger than can be explained by the smaller MLTV (Fig. 2). Supine, there was a small fall in FRC and a slightly larger fall in MLTV; the accompanying fall in Grs was slightly greater than expected from the reduction in MLTV. Although the small scale of these changes limits confidence in this finding, in our laboratory’s earlier study (40) we also found a fall in Grs when supine, despite supine MLTV being unchanged.

A few earlier studies have found an increase in airflow resistance (34, 40, 41) and in SRrs (40, 41) in obesity, but it is not possible from the way these results were presented to determine whether the increase was solely due to the reduced Vt. The site of this additional increase in Rs,mo and fall in Grs,mo at isovolume could be in any part of the extra- or intrathoracic airways, the lung tissue, or the chest wall. Although an increase in Rw appears an obvious possibility, the value of Rw measured at 6 Hz is small and much lower than the value that would be found during tidal breathing; furthermore, two recent studies (40, 41) have found only small differences between Raw and Rs in seated obese subjects at FRC. Potential causes of an increase in intrapulmonary resistance include airway closure (22) and tidal expiratory flow limitation (31). Mass loading of the supralaryngeal airway by fat in the neck could narrow the airway when supine (3, 24), but this seems unlikely when seated. The present experiments do not allow us to distinguish between these possibilities.

Airflow Resistance of the Nasopharynx

In normal subjects, Rnp accounted for just less than one-half the total airflow resistance during tidal breathing via the nose both when seated and when supine. This proportion is similar or slightly lower than that reported before for the seated (10, 15, 17) and supine postures (15). Estimated Rnp increased in the supine posture, as observed previously with this (15) and other methods (13). This increase has been attributed to gravitational increases in vascular pressure leading to swelling of the mucosa of the nasal cavity, particularly at the nasal valve (13). Other sites, such as narrowing of the velopharynx, may contribute. The combined effect of these changes was that total airflow resistance during supine nasal breathing (as during sleep) was more than twice the values measured in routine laboratory assessment when seated breathing via a large bore mouthpiece.

The obese subject who had a very high Rnp was aware of nasal obstruction, but this had not been treated. Excluding this subject, average seated Rnp was still higher in the obese subjects than in the control subjects, but there was considerable between-subject variation. Supine increases in absolute mean Rnp were similar in the obese and control subjects. We do not know whether the sites of Rnp differed in the two groups in either posture. Much more extensive studies are required to examine whether measurements of Rnp using this simple noninvasive technique when awake can help to predict the risk of obstructive sleep apnea.

In summary, although mass loading and reduced compliance of the respiratory system are the presumptive causes of the restrictive lung disease that is found in obese subjects when seated, neither TLC nor FRC show the expected further fall when recumbent. The observed increase in airflow resistance in obese subjects is only partly explained by the smaller Vt. The cause and site of the additional resistance at isovolume is uncertain. An increased Rnp was found in some obese subjects.

GRANTS

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