

# special communication

# **Breathing Patterns\***

# 1. Normal Subjects

Martin J. Tobin, M.D.; Tejvir S. Chadha, M.D.; Gilbert Jenouri, B.A.; Stephen J. Birch, B.S.; Hacik B. Gazeroglu, B.S.; and Marvin A. Sackner, M.D., F.C.C.P.

Ventilatory monitoring devices that require mouthpiece breathing produce a rise in tidal volume  $(V_T)$ , a fall in frequency (f) and alterations in periodicity and variability of breathing components. Together with the introduction of the respiratory inductive plethysmograph, a reliable noninvasive monitoring device of ventilation, major advances have taken place in understanding the significance of the components of the breathing pattern. We measured the breathing pattern of normal subjects utilizing respiratory

In the past decade, major advances have taken place in the interpretation of the significance of the various time and volume components of the breathing pattern.<sup>13</sup> For example, it has been shown that the tidal volume divided by inspiratory time (mean inspiratory flow) reflects respiratory center drive when mechanics of breathing are normal or only moderately impaired. 45 Relative contributions of the rib cage and abdominal compartmental excursions to tidal volume and the phase relationship between the two compartments provide information on respiratory muscle fatigue.<sup>3,6</sup> Mouthpiece breathing to and from a spirometer or pneumotachograph is known to increase tidal volume and decrease respiratory frequency compared to natural breathing monitored with an external device.<sup>79</sup> Further, volume and timing relations from the rib cage and abdominal compartmental contributions to tidal volume can only be measured with the latter such as the respiratory inductive plethysmograph.<sup>10,11</sup>

The interpretation of alterations of the time and volume components of the breathing pattern during natural breathing has not been translated into clinical practice because quantitative data have been lacking. In this study, breathing patterns of normal subjects were investigated with respiratory inductive

202

inductive plethysmography and continuously processed these data with a microprocessor system. The mean values of the breathing pattern components in normal subjects were not affected by age, but the rhythmicity was more irregular in the elderly. The values of breathing pattern components obtained noninvasively by respiratory inductive plethysmography in normal subjects are fairly predictable in limits similar to other tests of pulmonary function.

plethysmography to provide a basis for comparison to disease states reviewed in a subsequent publication.<sup>18</sup>

#### MATERIALS AND METHODS

## Subjects

The breathing pattern in the supine position was recorded utilizing the respiratory inductive plethysmograph in 47 young ( $\leq$ 50 years) and 18 old ( $\geq$ 60 years) normal subjects (Table 1). Subjects rested in the supine position a minimum of ten minutes prior to recording the breathing pattern for 15 minutes.

#### Theoretical Background

The traditional equation of minute ventilation  $(\dot{V}_{uh})$  indicates equality to the product of tidal volume  $(V_T)$  and respiratory frequency (f) such that  $\dot{V}_{uh} = V_T \times f$ . This can be rewritten so that  $\dot{V}_{uh} =$  $V_T/T_1 \times T_T/T_{TOT}$ . The first parameter,  $V_T/T_1$ , has been termed mean inspiratory flow, and the second,  $T_T/T_{TOT}$  the fractional inspiratory time or effective timing ratio.<sup>13</sup>

With respiratory inductive plethysmography, three signals are recorded: the calibrated ribcage (RC) and abdominal (ABD) excursions and their electrical sum signal, the latter being equivalent to tidal volume (V<sub>T</sub>). The sum of the absolute values of the peak-to-peak excursions of RC and ABD irrespective of their timing to the tidal volume signal is termed the "maximum compartmental amplitude (MCA)." When the peak-to-trough amplitudes of the RC and ABD signals are in phase, the ratio of MCA to  $V_T$  (MCA/ $V_T$ ) is equivalent to 1.00. When the peak-to-trough amplitudes of the RC and ABD move out of phase with each other, MCA/ $V_T$  exceeds 1.00. Thus, this index provides information on overall phasic relations between RC and ABD excursions.

#### Apparatus

Detailed descriptions of the respiratory inductive plethysmograph (Respitrace, Non-Invasive Monitoring Systems, Inc, Ardsley, NY) have been published.<sup>91</sup> Briefly, the device consists of two coils

<sup>\*</sup>From the Jane and Edward Shapiro Pulmonary Suite, Division of Pulmonary Disease, Department of Medicine, Mount Sinai Medical Center, Miami Beach.

Supported in part by National Heart, Lung and Blood Institute Grant HL-10622 and a contract from Electric Power Research Institute RP-1373.

Reprint requests: Dr. Sackner, 4300 Alton Road, Miami Beach 33140

Table 1—Pulmonary Function Tests and Breathing Pattern Components in Normal Subjects\*

	Young	Old
Number (M/F)	47 (31/16)	18 (9/9)
Age (years)	$28.6 \pm 5.3$	$68.9 \pm 6.5$
VC (L)	$5.32 \pm 1.12$	3.48±.84
% Pred normal	$117 \pm 18$	$118 \pm 25$
FRC (L)	$3.05 \pm .80$	$3.01 \pm .64$
% Pred normal	$110 \pm 25$	$118 \pm 24$
TLC (L)	$6.91 \pm 1.32$	$5.81 \pm 1.17$
% Pred normal	$110 \pm 13$	111 ± 18
FEV <sub>1</sub> (L)	$4.16 \pm .73$	2.64 ± .64
% Pred normal	$106 \pm 17$	$121 \pm 28$
$R_{m}$ cmH <sub>2</sub> O/L×sec	1.7±.5	1.5±.4
% Pred normal	$147 \pm 46$	$143 \pm 47$
f (breaths/min)	$16.7 \pm 2.7$	$16.6 \pm 2.8$
V <sub>T</sub> (ml)	$383 \pm 85$	$382 \pm 108$
V <sub>min</sub> (L/min)	$6.02 \pm 1.32$	$5.92 \pm 1.59$
T <sub>I</sub> (sec)	$1.60 \pm .30$	1.67±.35
T/Tmr	$.424 \pm .032$	$.413 \pm .034$
$V_{T}/T_{I}$ (ml/sec)	$249 \pm 54$	$253 \pm 69$
RC/V <sub>T</sub> (%)	$42 \pm 3$	$46 \pm 14$
MCA/V <sub>T</sub>	1.04 ± .04	$1.07 \pm .05$

#### $Mean \pm SD$

of Teflon-insulated wire sewn onto elastic bands encircling the rib cage (RC) and the abdomen (ABD) which are connected to an oscillator module. Changes in cross-sectional area of the RC and ABD compartments alter the self-inductance of the coils and the frequency of their oscillations, which, after appropriate calibration, reflect tidal volume measured by spirometry. Assuming that the respiratory system moves with two degrees of freedom, *ie*, the rib cage and abdominal compartments can be considered as the only two moving components, the device is calibrated using RC, ABD and spirometric (SP) volumes and the equation RC/SP+ABD/SP=1.<sup>10</sup> The subject breathes into a spirometer in two body postures to produce a spread of values of rib cage and abdominal contributions to tidal volume and the equation is solved graphically to achieve the scaling factors for the electrical gains of the RC and ABD amplifiers.

The signals from the respiratory inductive plethysmograph were recorded on a Grass polygraph recorder (Grass Instruments, Quincy, MA) and a Z-80 A based microprocessor system (Respicomp, Non-invasive Monitoring Systems, Inc, Ardsley, NY) which sampled the signals at 20 points/sec. It continuously calculated respiratory frequency (f), tidal volume ( $V_{T}$ ), minute ventilation ( $\dot{V}_{min}$ ), inspiratory time ( $T_{t}$ ), fractional inspiratory time ( $T_{t}/T_{TOT}$ ), mean inspiratory flow ( $V_{T}/T_{t}$ ), the percentage of rib cage contribution to tidal volume ( $\Re RC/V_{T}$ ), and the ratio of the maximum compartmental amplitude to tidal volume (MCA/ $V_{T}$ ).

# RESULTS

Forty-seven of the normal subjects were 20 to 50 years of age and 18 from 60 to 81 years of age (Table 1). Differences in age did not affect the mean values of the various breathing pattern components. The average f for all the normals was  $16.6 \pm 2.8$  breaths/min,  $V_T$   $383 \pm 91$  ml and  $\dot{V}_{min}$   $6.01 \pm 1.39$  L/min. Inspiratory time,  $1.62 \pm .31$  sec, was briefer than expiratory time, as reflected by a  $T_I/T_{TOT}$  of  $.421 \pm .033$ . Values of  $T_I/T_{TOT}$  did not correlate with respiratory frequency. Respiratory center drive as reflected by  $V_T/T_I$  was  $250 \pm 58$  ml/sec. No significant correlations (r values between .02 and .14) could be established for f,  $V_T$ ,  $\dot{V}_{min}$  and  $V_T/T_I$  on the one hand and body height on the other.

A typical analog tracing of the breathing pattern in a normal young adult is shown in Figure 1. One or no sighs (defined as three times mean  $V_{T}$ ) occurred over the 15-minute observation period in the young subjects and zero to 3 in the older subjects. Breathing was generally more regular in young than the old normal adults. Figure 2 depicts examples of the wide range of rhythmicity of breathing in the older subjects. Exaggerated intermittent periods of crescendo and decrescendo tidal volume fluctuations and end-expiratory level oscillations were seen in the analog recordings of six of the 47 young and six of the 18 old subjects. Brief central apneas of eight to ten seconds' duration were observed occasionally in the recordings of the old normal adults.

The recording of the breathing pattern was obtained in the supine position in which the rib cage contribution to tidal volume  $(42 \pm 17 \text{ percent})$  was less than





abdominal contribution, but as evidenced by the large standard deviation, many normal subjects displayed rib cage predominance. There was no sexual difference as to which compartment predominated. The rib cage and abdominal compartments moved in synchrony or almost so and their maximal compartmental amplitude was close to tidal volume (MCA/V<sub>T</sub> ratio of  $1.05 \pm .04$ ).

# DISCUSSION

# **Respiratory Rate**

In our normal subjects, aging did not affect respiratory frequency. The rate was  $16.7 \pm 2.7$  breaths/min in the young subjects  $(28.6 \pm 5.3 \text{ years})$  and  $16.6 \pm 2.8$  in the older subjects ( $68.9 \pm 6.5$  years). A slight fall in frequency and rise in tidal volume with aging has been reported for mouthpiece breathing, but inspection of individual data points revealed wide scatter.<sup>13</sup> Previous authors have indicated that mean rates as low as 11<sup>14</sup> or 12 breaths/min<sup>15</sup> were present in normal subjects. Such measurements were obtained with subjects breathing through a mouthpiece attached to a spirometer or pneumotachograph, whereas rates similar to ours, viz 17.0 and 17.5 breaths/min, were found on counting the respirations of subjects unaware of being observed<sup>16</sup> and from recordings obtained with a rib cage pneumograph,<sup>17</sup> respectively.

Respiratory rate is set at a particular frequency which is least costly to the average force developed by the respiratory muscles. Mead<sup>10</sup> derived an equation for this frequency which is a function of the time FIGURE 2. Types of breathing patterns in old normal adults. A) Fairly regular rhythm; B) irregular rhythm with short end-expiratory pauses; C) irregular rhythm with small tidal volumes interposed among normal tidal volume breaths (same subject as in B); D) sigh near onset of recording followed by an end-expiratory apnea. The flattened appearance on the SUM ( $V_T$ ) tracing is artifactual and due to a limitation of excursion of the recording pen; E) Cheyne-Stokes-like breathing pattern with paradoxic movements between RC and ABD compartments immediately prior to second hyperpneic episode; and F) undulating fluctuations of tidal volume.

constant of the respiratory system, *ie*, resistance times compliance, and the ratio of alveolar ventilation to dead space. Mead<sup>16</sup> used a mean value for the time constant of nasally breathing normal subjects, assumed an alveolar ventilation of 6,000 ml/min and dead space of 200 ml, and calculated this value as 19 breaths/min. However, if a dead space of 150 ml is assumed and our mean value for minute ventilation of 6,000 ml/min is used, then alveolar ventilation will be 3,600 ml. Substitution of these values into the Mead<sup>16</sup> equation leads to a predicted respiratory rate of 17.4 breaths/ min, a value close to the value of 16.6 breaths/min we observed.

# Tidal Volume

In our subjects, the tidal volume of  $383 \pm 91$  ml (mean + SD) was markedly lower than recorded for normal subjects in other studies,  $840 \pm 120$  ml (supine),<sup>18</sup>  $490 \pm 60$  ml (supine))<sup>19</sup> and  $710 \pm 159$  ml (posture not stated).<sup>14</sup> This difference relates to direct measuring techniques which employ breathing through a mouthpiece with the nose clipped. The importance of this factor in causing an increase in V<sub>T</sub> was first shown by Gilbert et al<sup>7</sup> using magnetometers, and later confirmed with the canopy-spirometry system<sup>8</sup> and the respiratory inductive plethysmograph.<sup>9</sup> When comparing breathing patterns from different studies, body posture must also be taken into account. In six normal subjects whose breathing pattern was monitored with respiratory inductive plethysmography in our labora-

tory,  $V_{\tau}$  increased from  $364 \pm 60$  ml in the supine position to  $443 \pm 107$  ml in the sitting position and  $V_{\tau}/T_{\tau}$ increased from  $219 \pm 26$  ml/sec to  $243 \pm 51$  ml/sec. But values for these components were still much less than observed in seated normal subjects breathing through a mouthpiece to a spirometer.

### Minute Ventilation

As mentioned above, mouthpiece breathing promotes an increase of  $V_{T}$  and a decrease in f.  $\dot{V}_{min}$  remains unchanged or slightly increases. With a significant apparatus deadspace, there is a rise of  $\dot{V}_{min}$ . In our normal subjects, the  $\dot{V}_{min}$  of  $6.01 \pm 1.39$  L/min (mean  $\pm$  SD) was lower than reported by several investigators in subjects breathing through a mouthpiece, viz,  $7.81 \pm .10$  L/min,<sup>14</sup> 12.7  $\pm 1.4$  L/min,<sup>18</sup> and  $7.30 \pm .90$  L/min.<sup>19</sup>

#### Mean Inspiratory Flow

Barcroft and Margaria<sup>20</sup> wrote some 50 years ago that attempts to elucidate mechanisms controlling respiration should analyze minute ventilation in terms of "1) the duration of the phases of respiration . . . , and 2) upon the rates at which air is taken in and out during the various phases." They measured  $V_T/T_1$  and  $\dot{V}_{min}$  at rest, during CO<sub>2</sub> inhalation, and with exercise and showed a linear relationship between the two measurements. Our slope of  $(V_T/T_1 \text{ [ml/sec]})/(\dot{V}_{\min} \text{ [L]})$  of 37.8 in normal subjects was similar to their value of 35.7. For the next 40 years, this analytical approach lay dormant until the investigations of Clark and Von Euler,<sup>1</sup> and Milic-Emili and Grunstein.<sup>2</sup> In this analysis, minute ventilation was regarded as the product of mean inspiratory flow  $(V_T/T_1)$  and fractional inspiratory time  $(T_{I}/T_{TOT})$ . Since neural impulses controlling inspiratory drive were independent of reflex activity once inspiration has begun,  $V_T/T_1$  reflected respiratory center drive as it is the mechanical transformation of phrenic nerve activity. Comparison of  $V_{\tau}/T_{1}$  to standard indices of respiratory center output, such as the mouth occlusion pressure generated during the first 0.1 sec after the onset of unanticipated inspiration against an occluded airway  $(P_{01})$ , showed a close correlation between the two measurements.<sup>4</sup> This relationship was linear up to a  $V_T/T_1$  of 1 L/sec,<sup>21</sup> a value much greater than encountered in our resting subjects.

# **Rib Cage-Abdominal Compartmental Excursions**

Although there was slight predominance of abdominal contribution to tidal volume in our normal supine subjects, there was wide variation so that several subjects had predominantly rib cage movement. The compartments generally moved almost synchronously as reflected by the maximal compartmental amplitude ratio which approached unity.

### References

- Clark FJ, von Euler C. On the regulation of depth and rate of breathing. J. Physiol 1972; 222:267-95
- 2 Milic-Emili J, Grunstein MM. Drive and timing components of ventilation. Chest 1976; 70:131-33
- 3 Macklem PT. Respiratory muscles: The vital pump. Chest 1980; 753-58
- 4 Derenne JP, Couture J, Iscoe S, Whitelaw WA, Milic-Emili J. Occlusion pressures in men rebreathing CO<sub>2</sub> under methoxyflurane anesthesia. J Appl Physiol 1976; 40:805-14
- 5 Chadha TS, Birch S, Schneider A, Ford D, Sackner MA. Effect of graded bronchospasm on breathing pattern. Am Rev Respir Dis 1982; 125:218 (abstract)
- 6 Ashutosh K, Gilbert R, Auchincloss JH, Peppi D. Asynchronous breathing movements in patients with chronic obstructive pulmonary disease. Chest 1975; 67:553-57
- 7 Gilbert R, Auchincloss JH Jr, Brodsky J, Boden W. Changes in tidal volume, frequency, and ventilation induced by their measurement. J Appl Physiol 1972; 33:252-54
- 8 Askanazi J, Silverberg PA, Foster RJ, Hyman AI, Milic-Emili J. Effects of respiratory apparatus on breathing pattern. J Appl Physiol 1980; 48:577-80
- 9 Sackner JD, Nixon AJ, Davis B, Atkins N, Sackner MA. Effects of breathing through external dead space on ventilation at rest and during exercise. II. Am Rev Respir Dis 1980; 122:933-40
- 10 Chadha TS, Watson H, Birch S, Jenouri GA, Schneider AW, Cohn MA, et al. Validation of respiratory inductive plethysmography using different calibration procedures. Am Rev Respir Dis 1982; 125:644-49
- 11 Cohn MA, Rao ASV, Broudy M, Birch S, Watson H, Atkins N, et al. The respiratory inductive plethysmograph: a new noninvasive monitor of respiration. Bull Europ Physiopathol Respir 1982; 18:643-58
- 12 Tobin MJ, Chadha TS, Jenouri GA, Birch SJ, Gazeroglu HB, Sackner MA. Breathing patterns: 2. Diseased subjects. Chest (in press)
- 13 Jammes Y, Auran Y, Gouvernet J, Delpierre S, Grimaud C. The ventilatory pattern of conscious man according to age and morphology. Bull Europ Physiopathol Respir 1979; 15:527-40
- 14 Savoy J, Dhingra S, Anthonisen NR. Role of vagal airway reflexes in control of ventilation in pulmonary fibrosis. Clin Sci Molecular Med 1981; 61:781-84
- 15 Comroe JH, Forster RE, DuBois AB, Briscoe WA, Carlsen E. The lung. Chicago: Year Book, 1962; 323
- 16 Mead J. Control of respiratory frequency. J Appl Physiol 1960; 15:325-36
- 17 Bendixen H, Smith GM, Mead J. Pattern of ventilation in young adults. J Appl Physiol 1964; 19:195-98
- 18 Sorli J, Grassino A, Lorange G, Milic-Emili J. Control of breathing in patients with chronic obstructive lung disease. Clin Sci Molecular Med 1978; 54:295-304
- 19 Renzi G, Milic-Emili J, Grassino AE. The pattern of breathing in diffuse lung fibrosis. Bull Europ Physiopath Resp 1982; 18:461-72
- 20 Barcroft J, Margaria R. Some effects of carbonic acid on the character of human respiration. J Physiol (London) 1931; 72:175-85
- 21 Almirall JJ, Miserocchi G, Whitelaw WA, Grassino A, Milic-Emili J. Effect of changes in the expiratory activity on occlusion pressure (P<sub>0.1</sub>) during CO<sub>2</sub> rebreathing in sitting and supine subjects. Proc Int Union Physiol Sci 1980; 14:296