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ORIGINAL ARTICLE

Respiratory pattern, thoracoabdominal motion and ventilation in chronic airway obstruction

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ABSTRACT: ***Respiratory pattern, thoracoabdominal motion and ventilation in chronic airway obstruction. M. Fernandes, A. Cukier, N. Ambrosino, J.J. Leite, M.I. Zanetti Feltrim.***

Background. **Patients with chronic obstructive pulmonary disease (COPD) present abnormal respiratory mechanics, but its relation to ventilation variables at rest is not fully understood.**

Methods. **We evaluated breathing pattern, thoracoabdominal motion, and ventilation in moderate and severe COPD patients by means of respiratory inductive plethysmograph and analysis of respiratory metabolism in semi-recumbent position at rest. Diaphragmatic movement was measured using radiographs.**

Results. **COPD patients showed an increase in mean inspiratory flow, minute ventilation, dead space ventilation, oxygen and carbon dioxide ventilatory equivalents and reduction of respiratory times and pulse oxymetry. These findings were more pronounced in severe COPD. Changes in ventilatory efficiency were correlated with decreased respiratory times, reduced diaphragmatic movement, and lower oxygen uptake.**

Conclusions. **Rapid shallow breathing and reduced diaphragmatic movement have led to ventilatory inefficiency without changes in thoracoabdominal motion.**

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Keywords: Breathing pattern, chronic obstructive pulmonary disease, pulmonary ventilation, respiratory muscles, thoracoabdominal motion.

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Introduction

Patients with COPD may experience changes in respiratory pattern in the form of rapid, shallow breathing. These changes have been related to hypercapnia [1] muscle weakness; [2] increased pleural pressure variation during breathing; and [3] the perception of inspiratory effort and dyspnoea.

The mechanisms underlying these changes have not yet been fully clarified [2], but are related to the capability of respiratory muscles to sustain increases in inspiratory load [5].

Several studies have described the breathing pattern and thoracoabdominal motion in severe COPD patients [1, 3, 6]. However, mechanisms involved in the genesis of changes in breathing pattern and thoracoabdominal motion and their relation to ventilatory variables at rest have not been fully clarified. This study aims to assess the breathing pattern, thoracoabdominal motion, and ventilation in COPD patients during spontaneous breathing.

Methods

Patients

The study population included COPD patients admitted at the Heart Institute of the Clinical Hospital of the São Paulo University Medical School from 2002 to 2003. Diagnosis of COPD was made according to American Thoracic Society guidelines [7]: history of smoking (> 10 pack-year), a post-bronchodilator forced expiratory volume in one second (FEV_1) < 80% of predicted and a forced expiratory volume at first second (FEV_1) / forced vital capacity (FVC) ratio < 0.7. Upon admission, all patients had stopped smoking for 1 to 38 years, were in stable conditions, as assessed by stability in blood gas values, and were free from exacerbations in the four weeks prior to study. Patients with a post bronchodilator (200 mcg of salbutamol) change in FEV_1 > 15% or > 200 ml were excluded from the study as well as patients with other organ failure, cancer or inability to cooperate. All patients received regular treatment

with inhaled bronchodilators and inhaled steroids according to current guidelines for their disease stage [8].

The study included 29 COPD patients divided into two groups: a moderate COPD group (FEV_1 35 to 50% predicted) and a severe COPD group (FEV_1 < 35% predicted), as well as a control group with 15 healthy subjects. The study was approved by the Research Committee on Human Investigation at the authors' institution, and informed written consent was obtained after the purposes and potential risks of the investigation were fully explained and understood by each patient.

Protocol

First day: Following informed consent, patients underwent conventional pulmonary function tests.
Second day: An assessment of the diaphragmatic movement was undertaken. Third day: Breathing pattern, thoracoabdominal motion and metabolic gas analyses were conducted.

Lung Function

Spirometry and lung volumes were assessed according to the American Thoracic Society (ATS) and European Respiratory Society (ERS) guidelines [9]. Dynamic lung volumes were calculated by means of flow espirometer (Puritan-Bennett PB 100, Pleasanton, CA, USA). The predicted values according to *Knudson et al*. 1983 [10] were used.

Respiratory muscle function

Maximal inspiratory (MIP) and expiratory (MEP) pressures were measured at the level of Functional Residual Capacity (FRC) [9], using a respiratory module system (Marshall Town - Instrumentation Industries, Bethel Park, PA). The predicted values according to *Neder et al*. 1999 [11] were used.

Diaphragmatic Movement

Diaphragmatic movement (DM) was obtained using chest radiography under full inspiration and expiration in orthostatic position [12, 13]. We performed two radiographs exposures of the same film in posterior-anterior and lateral view. Images were digitalised by a computerised system. We used software (Image Tool for Windows, version 1.28) to calculate the area between the right dome of the diaphragm in full inspiration and expiration using a posterior-anterior view (DM_{PA}) and the area between the 2 domes in lateral view (DM_L) expressed in square centimeters. The calibration of the computer software was made based on a five-centimeter line [12, 13].

Breathing pattern, thoracoabdominal motion, and exhaled gas analysis

Breathing pattern and thoracoabdominal motion were recorded using a respiratory inductive plethysmography system (Respitrace, Non-invasive Monitoring Systems, Miami, FL). The Least Squares Method calibration technique was used [14]. During the study, the tidal volume (V_T), respiratory frequency (f), inspiratory and expiratory times (T_I,T_E), the fractional inspiratory time (T_I/T_{TOT}), the mean inspiratory flow (V_T/T_I), the percentage of rib cage contribution to tidal volume (%RC/V_T), and the ratio of the maximum compartmental amplitude to tidal volume (MCA/V_T) were calculated continuously.

Ventilatory variables were obtained breath-by-breath by using a gas metabolic analysis system with a pneumotachograph (Cardiopulmonary Exercise System CPX, Medical Graphics Corporation, St. Paul, MN) by means of a mouthpiece and nose clip. The system was calibrated before measurements by using the manufacturer's recommended procedures. Oxygen uptake (VO_2), carbon dioxide output (VCO_2), minute ventilation (V_E), ventilatory equivalents (V_E/VCO_2, V_E/VO_2), end tidal CO_2 and O_2 ($ETCO_2$, ETO_2), dead space ventilation (V_D/V_T) and oxygen saturation (SpO_2) were measured. Alveolar ventilation (V_A) was calculated using $V_A = V_T (1 - [V_D/V_T])$.

The subjects were comfortably placed in a 45º semi-recumbent position and following stabilisation, breathing at rest was measured for 4 minutes. The modified Borg scale (0 to 10) was used to assess the feeling of breathlessness in the first minute [15].

Statistical analysis

Data is presented as means and standard deviations (SD). One-way analysis of variance and Kruskal-Wallis one-way analysis of variance were utilised to determine differences between groups. The Tukey test was used for multiple comparisons and Pearson's coefficiency was obtained to assess the correlation between variables; $p < 0.05$ was considered statistically significant.

Results

Lung and respiratory muscle function

Significant difference between groups was found in the body mass index (BMI); severe COPD patients showing a significant reduction. The mean functional pulmonary values between groups were different whereas the respiratory muscle function was similar in COPD and control groups. Diaphragmatic movement was significantly reduced in both COPD groups (table 1).

Breathing pattern and thoracoabdominal motion

Severe COPD group showed a breathing pattern characterised by significantly reduced T_I resulting in increase in V_T/T_I. No other significant difference in breathing pattern and thoracoabdominal motion was found among groups. The perception of breathing effort (modified Borg scale) did not differ between groups (table 2).

Ventilatory Variables

Variables obtained from the respiratory gas analysis are shown as following (table 3). VO_2, VCO_2, ETO_2 and $ETCO_2$ were similar in all groups. Compared with control mean V_E was significantly higher in moderate but not in severe COPD. In the severe COPD group V_D/V_T, V_E/VO_2

Table 1. - Anthropometric data, lung and respiratory muscle functions

	Control Group	Moderate COPD Group	Severe COPD Group
Sex M/F	7/8	11/3	14/1
Age yr	60±7	63±7	60±8
Height cm	167±9	166±11	164±6
BMI kg/m^2	26±4	24±5	21±4*
FEV_1 L	2.91±0.81	1.20±0.27*	0.73±0.17*†
% pred	102±13	44±4*	27±6*†
FVC L	3.65±0.89	2.66±0.76*	2.12±0.57*†
% pred	104±11	77±14*	63±18*†
FEV_1/FVC	0.79±0.05	0.45±0.07*	0.36±0.08*†
MIP cmH_2O	89±21	80±30	88±23
MIP % pred	145±36	113±36	120±36
MEP cmH_2O	103±17	102±24	111±13
MEP % pred	166±29	97±23	141±18
DM_{PA} cm^2	74±19	53±19*	41±14*
DM_L cm^2	132±41	95±42*	66±32*

M/F: male/female; BMI: body mass index; DM_{PA}: Diaphragmatic movement postero-anterior view; DM_L: Diaphragmatic movement lateral view; FEV_1: forced expiratory volume in one second; FVC: forced vital capacity; MIP: maximal inspiratory pressure; MEP: maximal expiratory pressure. * $p<0.05$ versus control group, † $p<0.05$ versus moderate group. Values are mean ± SD.

Table 2. - Breathing pattern, thoracoabdominal motion, and dyspnoea

	Control Group	Moderate COPD Group	Severe COPD Group
V_T mL	411±187	384±179	476±191
f breaths/min	13±3	17±5*	16±3
T_I s	1.85±0.48	1.51±0.46	1.38±0.25*
T_E s	2.90±0.95	2.33±0.75	2.36±0.49
T_I/T_{TOT}	0.39±0.06	0.40±0.04	0.37±0.06
V_T/T_I mL/s	223±80	271±146	346±134*
%RC/V_T %	35±12	36±16	30±9
MCA/V_T	1.03±0.05	1.07±0.06	1.03±0.03
Borg	1±0.8	1±1.1	1±1.3

Borg: dyspnoea index; f: respiratory frequency; MCA/V_T: ratio of the maximum compartmental amplitude to tidal volume; T_I: inspiratory time; T_E: expiratory time; T_I/T_{TOT}: fractional inspiratory time; V_T: tidal volume; V_T/T_I: mean inspiratory flow; %RC/V_T: percentage of rib cage contribution to tidal volume. * $p<0.05$ versus control group. Values refer to mean ± SD of a period of four minutes.

Table 3. - Ventilatory variables

	Control Group	Moderate COPD Group	Severe COPD Group
VO_2 mL/min	193±69	212±56	170±45
VCO_2 mL/min	165±70	181±45	147±40
V_E L/min	8.4±2.6	10.5±1.6*	9.9±1.8
V_E/VO_2	44±9	53±12	60±9*
V_E/VCO_2	55±10	61±13	71±14*
ETO_2 mm Hg	103±7	104±5	101±7
$ETCO_2$ mm Hg	40±4	37±4	38±5
V_D/V_T	0.37±0.07	0.42±0.06	0.47±0.05*
SpO_2 %	96±1	94±2	91±2*†
V_A L/min	5.31±1.9	6.22±1.1	5.35±1.2

ETO_2: end tidal oxygen; $ETCO_2$: end tidal carbon dioxide; SpO_2: oxygen saturation; VO_2: oxygen uptake; VCO_2: carbon dioxide output; V_E: minute ventilation; V_E/VO_2: ventilatory equivalent to oxygen; V_E/VCO_2: ventilatory equivalent to carbon dioxide; V_D/V_T: ratio dead space volume under tidal volume; V_A: alveolar ventilation. * $p<0.05$ versus control group, † $p<0.05$ versus moderate group. Values refer to mean ± SD of a period of four minutes.

and V_E/VCO_2, were significantly increased and SpO_2 was reduced.

Ventilatory Efficiency

In the moderate COPD group, changes in ventilatory efficiency (V_E/VO_2 and V_D/V_T) were related to lower respiratory times (decreased T_I and increased f), decreased diaphragmatic mobility seen on the chest radiography lateral view and lower oxygen consumption (table 4 and figure 1). In the severe COPD group these correlations have been seen in minor intensity (table 4 and figure 2).

Discussion

This study identifies breathing pattern changes in COPD patients. Although thoracoabdominal motion was within normal limits, specific mechanisms are possibly associated with these findings promoting adaptation to the chronic increase in the load imposed on the respiratory muscle system. An association between variables allows us to identify specific characteristics pertaining to these patients that, when present, suggest ventilatory impairment.

The behaviour of V_T and f was different between patient groups in this study. Past studies suggest that the mechanical load imposed on the respiratory muscle system and the available muscle strength may determine changes in the breathing pattern in COPD [1, 3]. However, V_T and f may adjust to minimize muscle load according to the predominant change either in the elastic or the resistive component of the system at any given moment in the disease [2]. The major interference of the elastic work in the moderate COPD group may have led a lower V_T. It is known that the use of a mouthpiece promotes increase in tidal volume and reduction of respiratory rate in healthy subjects and in patients with COPD [16, 17]. Thus, the increase in breathing rate observed in our patients may be minimized by the use of this device. Greater differences in respiratory pattern could have been observed without its use.

The correlation results of T_I/T_{TOT} were similar in all groups. Our findings may be associated to flow inspiratory and ventilation. The imbalance between flow and demand may promote a secondary adjustment, namely a decrease in T_I/T_{TOT}, which reveals progressive muscle system deterioration [18] and which is related to dyspnoea [19]. Inspiratory muscles in our patients probably were operating under a still tolerable inspiratory load with no need for additional adjustments in the effective inspiration time. Thereby, the capacity of the system to generate inspiratory flow maintained relatively preserved. Decreases in T_I and T_{ToT} were probably the ventilatory strategy adopted to adapt the muscles to stress, keeping them below the fatigue zone [5].

Our patients tended to have an increased abdominal component. Our findings may be associated with specific chronic diaphragmatic adaptation, like maintenance of a relatively constant diaphragmatic geometry [20] and radius of curvature [13] in hyperinflation, leading diaphragmatic excursion and shortening preserved in respiration at rest [21, 22]. Other mechanisms may help the diaphragm to continue contributing to the generation of V_T, such as increased diaphragmatic motor unit discharge during tidal breathing and expiratory muscle recruitment, increasing diaphragmatic lengthening, thus improving the diaphragmatic force-length relationship and accumulating abdominal elastic energy that could support inspiratory abdominal expansion [23] (figure 3).

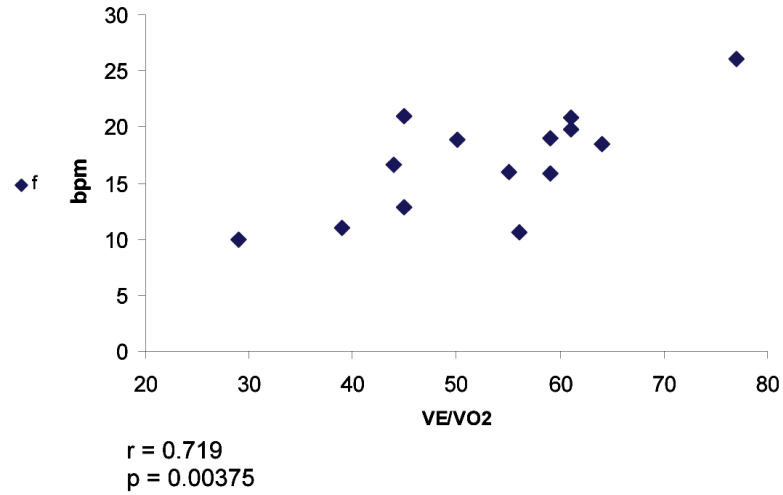
The COPD groups had coordinated rib cage and abdominal motion, even though other studies have demonstrated out of phase motion between compartments in COPD subjects [24, 25]. The paradoxical motion takes place as a function of the intensity of peak inspiratory pleural (P_{pl}), and transdiaphragmatic (P_{di}) pressure during breathing at rest [26]. Normal respiratory muscle strength provided our patients with an adequate ratio between muscle work executed and muscle reserve, preserving pressure generating capabilities. Therefore, we believe that normal muscle strength and efficient diaphragmatic motion avoid chest wall distortion. The adequate function of the respiratory muscles provides adjust between the respiratory motor command from the central nervous system and afferent feedback of the respiratory system [27]. It may have contributed to lower dyspnoea indexes in our patients.

The balance between mechanical impediments to breathing and the response of the inspi-

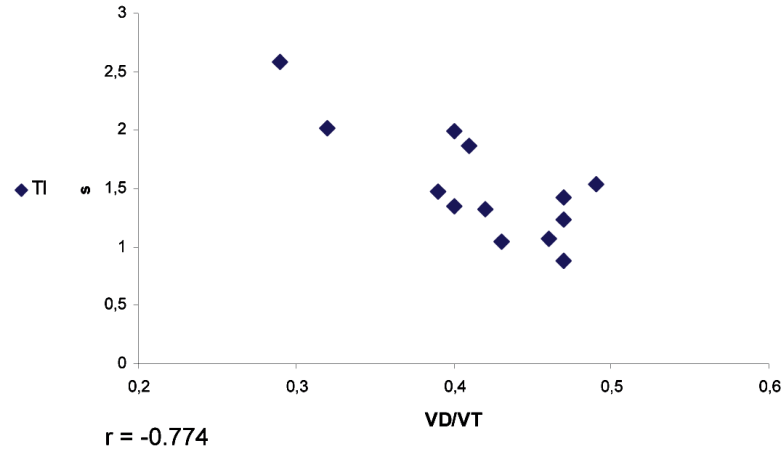
Table 4. - Ventilatory efficiency

	Moderate COPD Group				Severe COPD Group			
	V_E/VO_2		V_D/V_T		V_E/VO_2		V_D/V_T	
	r	p	r	p	r	P	r	p
T_I	-0.789	0.000786	-0.774	0.00116	-0.327	0.234	-0.538	0.0384
DM_L	-0.587	0.0272	-0.622	0.0176	-0.111	0.705	-0.239	0.411
VO_2	–	–	-0.873	0.0000454	–	–	-0.467	0.0796

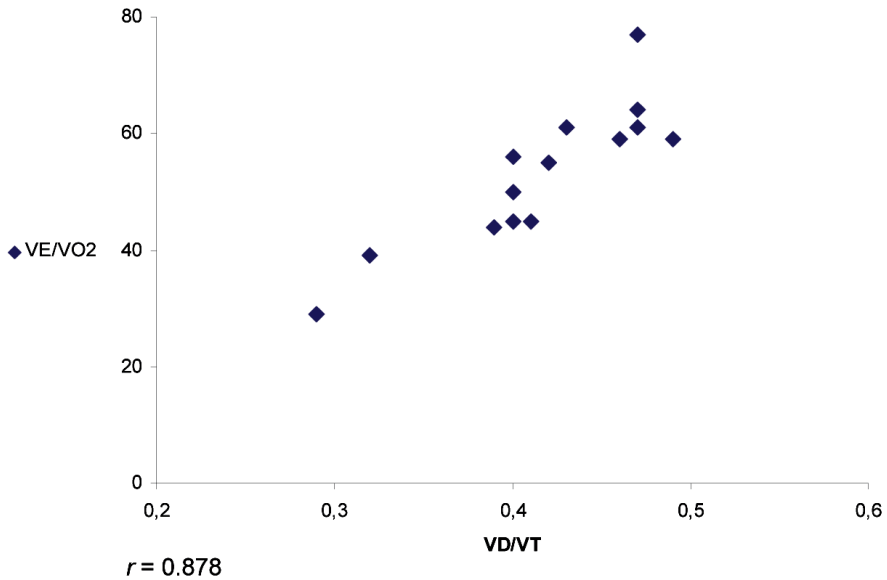
Plot of f (respiratory frequency), T_I (inspiratory time), DM_L (diaphragmatic movement in lateral view) and VO_2 (oxygen uptake) versus V_E/VO_2 (ventilatory equivalent to oxygen) and V_D/V_T (dead space volume under tidal volume ratio) to moderate and severe groups.



r = 0.719
p = 0.00375



r = -0.774
p = 0.00116



r = 0.878
p = 0.0000367

Fig. 1. - Plot of f versus V_E/VO_2 and T_I, V_E/VO_2 versus V_D/V_T in patients with moderate COPD.

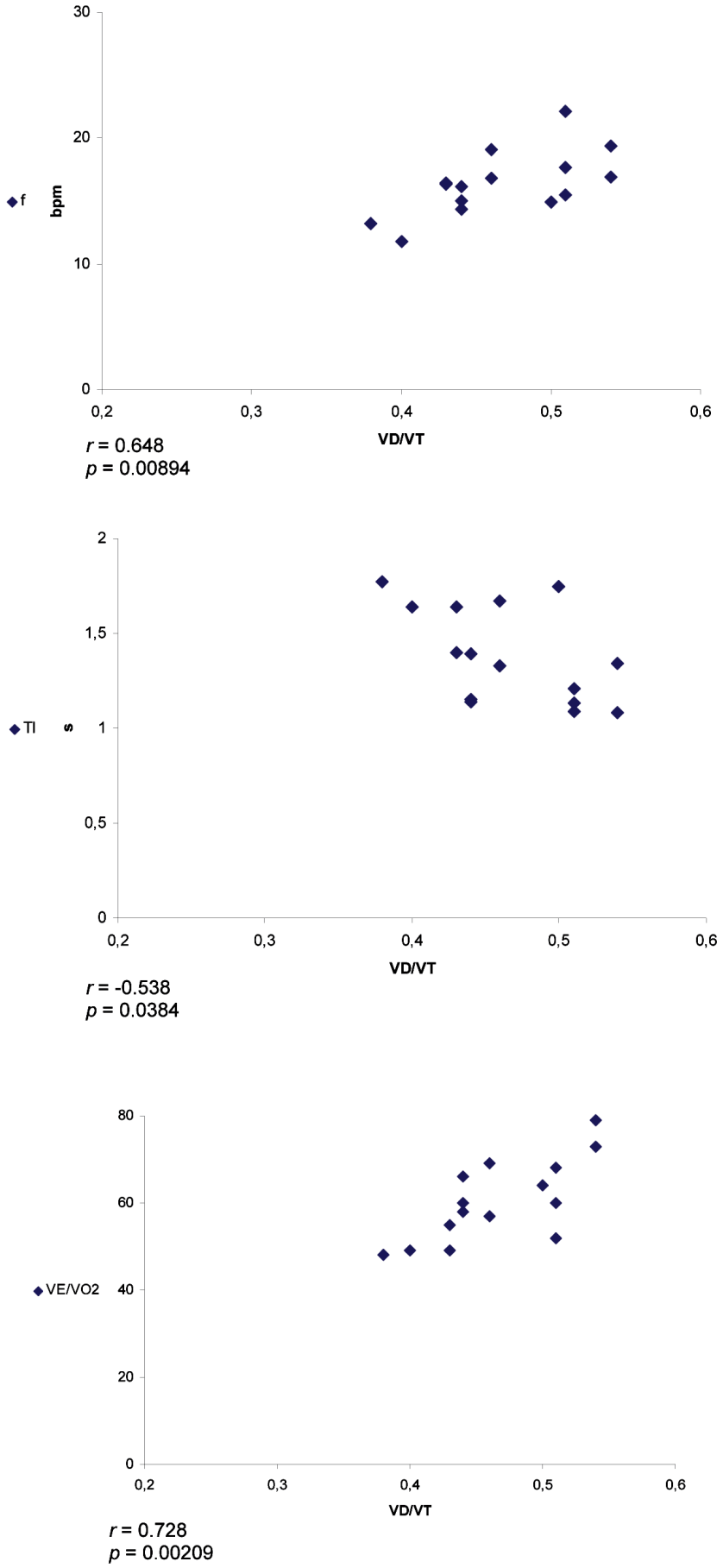


Fig. 2. - Plot of f, T_I and V_E/VO_2 versus V_D/V_T in patients with severe COPD.

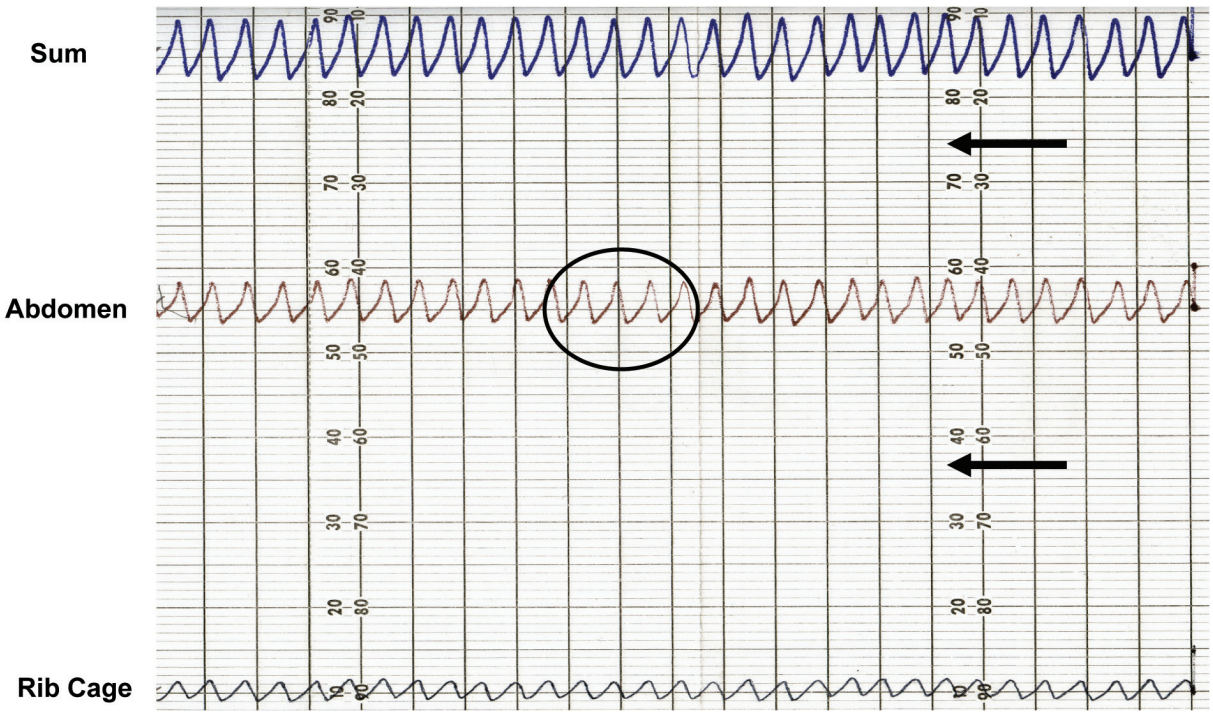


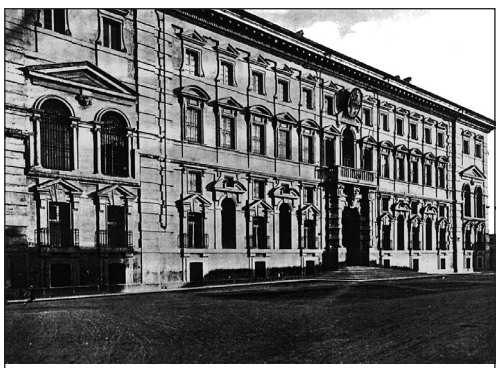
Fig. 3. - Respitrace record - severe COPD patient. Notice the pattern of prolonged expiration in the abdominal excursion curve, which may be related to expiratory muscle recruitment.

ratory muscles, expressed as inspiratory pressure under maximal inspiratory pressure (P_I/MIP), is shifted negatively (increased work/decreased muscle reserve) in COPD. Under such conditions, patients choose to reduce P_I by reducing V_T. A reduced V_T may reflect reduced central respiratory drive, mechanical limitation and/or inspiratory muscle dysfunction [2]. Adjustments in the breathing pattern seen in our patients probably occurred to improve energy efficiency, although rapid shallow breathing may lead to an increased V_D/V_T ratio. We observed an increase in the V_D/V_T ratio in COPD patients; alveolar ventilation (V_A), however, was unchanged. This may have maintained the oxygenation within acceptable clinical levels in the 3 groups. Correlations seen in the moderate COPD group reinforce the hypothesis that adjustments in respiratory times (lower T_I, higher f) and changes in diaphragmatic motion could be the determining factors leading to impaired V_E. However, at advanced stages of the disease, adjustments in respiratory times may have less influence on ventilatory efficiency. We have noticed that in four patients (two in the moderate group and two in the severe group), respiratory rate was reduced and V_D/V_T was increased. This data is possibly associated with limited capacity of V_T and V_T/T_I increase, which led to increase in T_I in the two moderate cases. Structural changes in the pulmonary parenchyma present in patients with severe COPD may have contributed to increasing V_D/V_T, which occurred in the presence of f mean close to that observed in the moderate group. The impact of ventilation strategies adopted in CO_2 blood pressure was not analyzed because of the absence of this variable. However, we observed $ETCO_2$ variation in many patients which was possibly a result of the oscillation of f and V_T. Even though $ETCO_2$ does not properly represent arterial CO_2 because of heterogeneity of ventilation in COPD, it is likely that the adopted strategies could have influenced arterial CO_2 in the presence of adequate balance between ventilation and perfusion.

In conclusion, the increased dead space ventilation and lower diaphragmatic motion are associated to impairment of ventilatory function and changes in breathing patterns. This is initially seen as rapid and shallow breathing with no changes in thoracoabdominal motion. As the disease progresses, greater neural respiratory drive stimulation occurs, seen as an increase in the mean inspiratory flow. These changes may lead to ventilatory inefficiency, although they defer the onset of respiratory muscle deterioration, frequently associated with chronic obstructive pulmonary disease. Respiratory pattern and thoracoabdominal motion characterization at different COPD stages, and its association to ventilation and diaphragmatic function, may provide a better pathophysiological understanding and adequate therapeutic planning at the future.

References

1. Gorini M, Misuri J, Corrado A, *et al*. Breathing pattern and carbon dioxide retention in severe chronic obstructive pulmonary disease. *Thorax* 1996; 51: 677-83.
2. Roussos C, Koutsoukou A. Respiratory failure. *Eur Respir J* 2003; 22: 3-14.
3. Duranti R, Misuri G, Gorini M, Goti P, Gigliotti F, Scano G. Mechanical loading and control of breathing in patients with severe chronic obstructive pulmonary disease. *Thorax* 1995; 50: 127-33.
4. American Thoracic Society. Dyspnoea. Mechanisms, assessment, and management: A Consensus Statement. *Am J Respir Crit Care Med 1999*; 159: 321-40.
5. Bellemare F, Grassino A. Effects of pressure and timing of contraction on human diaphragm fatigue. *J Appl Physiol* 1982; 53: 1190-5.
6. Martinez FJ, Couser JI, Celli BR. Factors influencing ventilatory muscles recruitment in patients with chronic airflow obstruction. *Am Rev Respir Dis* 1990;142: 276-82.
7. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 1995; 152: 77-120.
8. Sociedade Brasileira de Pneumologia e Tisiologia. I Consenso Brasileiro de Doença Pulmonar Obstrutiva Crônica. *J Bras Pneumol* 2000; 26: 1-52.
9. American Thoracic Society/European Respiratory Society. Statement on Respiratory Muscle Testing. *Am J Respir Crit Care Med* 2002; 166: 518-624.
10. Knudson RJ, Lebowitz MD, Holberg CJ, Burrows B. Changes in the maximal expiratory flow-volume curve with growth and ageing. *Am Rev Respir Dis* 1983; 127: 725-734.
11. Neder JA, Andreoni S, Lerario MC, Nery LE. References values for lung function tests. II. Maximal respiratory pressures and voluntary ventilation. *Braz J Med Biol Res* 1999; 32: 719-27.
12. Simon G, Bonnell J, Kazantzis G, Waller RE. Some radiological observations on the range of movement of the diaphragm. *Clin Radiol* 1969; 20: 231-3.
13. McKenzie DK, Gorman RB, Tolman J, Pride NB, Gandevia SC. Estimation of diaphragm length in patients with severe chronic obstructive pulmonary disease. *Respir Physiol* 2000; 123: 225-34.
14. Chadha ST, Watson H, Birch S, *et al*. Validation of respiratory inductive plethysmography using different calibration procedures. *Am Rev Respir Dis* 1982; 125: 644-9.
15. Borg GV. Psychophysical bases of perceived exertion. *Med Sci Sports Exercise* 1982; 14: 377-81.
16. Gilbert R, Auchincloss Jr JH, Brodsky J, Boden W. Changes in tidal volume frequency and ventilation induced by their measurement. *J Appl Physiol* 1972; 33: 252-4.
17. Loveridge B, West P, Anthonisen NR, Kryger MH. Breathing patterns in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1984; 130: 730-3.
18. Tobin MJ, Chadha TS, Jenouri G, Birch SJ, Gazeroglu HB, Sackner MA. Breathing patterns. 2. Diseased subjects. *Chest* 1983; 84: 286-94.
19. Gigliotti F, Romagnoli I, Scano G. Breathing retraining and exercise conditioning in patients with chronic obstructive pulmonary disease (COPD): a physiological approach. *Respir Med* 2003; 97: 197-204.
20. Gauthier AP, Verbanck S, Estenne M, Segebarth C, Macklem PT, Paiva M. Three-dimensional reconstruction of the in vivo human diaphragm shape at different lung volumes. *J Appl Physiol* 1994; 76: 495-506.
21. Gorman RB, Mckenzie DK, Pride NB, Tolman JF, Gandevia SC. Diaphragmatic length during tidal breathing in patients with chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2002; 166: 1461-9.
22. Kleinman BS, Frey K, Vandrunen M, *et al*. Motion of the diaphragm in patients with chronic obstructive pulmonary disease while spontaneously breathing versus during positive pressure breathing after anesthesia and neuromuscular blockade. *Anesthesiology* 2002; 97: 298-305.
23. Laghi F, Tobin MJ. Disorders of the respiratory muscles. *Am J Respir Crit Care Med* 2003; 168: 10-48.
24. Sackner MA, Gonzalez H, Rodriguez M, Belsito A, Sackner DR, Grenvik S. Assessment of asynchronous and paradoxic motion between rib cage and abdomen in normal subjects and in patients with chronic obstructive pulmonary disease. *Am Rev Respir Dis* 1984; 130: 588-93.
25. Gilmartin JJ, Gibson GJ. Abnormalities of chest wall motion in patients with chronic airflow obstruction. *Thorax* 1984; 39: 264-71.
26. Gilmartin JJ, Gibson GJ. Mechanisms of paradoxical rib cage motion in patients with COPD. *Am Rev Respir Dis* 1986; 134: 683-7.
27. Ambrosino N, Scano G. Measurement and treatment of dyspnoea. *Respir Med* 2001; 95: 539-47.



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