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Respiratory muscle insufficiency in acute respiratory failure of subjects with severe COPD: treatment with intermittent negative pressure ventilation

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Respiratory muscle insufficiency in acute respiratory failure of subjects with severe COPD: treatment with intermittent negative pressure ventilation. A. Corrado, G. Bruscoli, E. De Paola, G.F. Ciardi-Dupre', A. Baccini, M. Taddei.
ABSTRACT: Nine subjects with severe chronic obstructive pulmonary disease (COPD) in acute respiratory failure (ARF) and with marked weakness of the respiratory muscles (Group A) underwent intermittent negative pressure ventilation by means of an iron lung (8 h daily for 7 days). Seven subjects with COPD in stabilized chronic respiratory failure (Group B) were studied as controls and submitted to the same medical therapy without ventilator treatment. Functional respiratory tests were performed before and after 7 days of treatment. After ventilatory treatment, Group A showed an increase of maximum inspiratory pressure (PImax), maximum expiratory pressure (PEmax), vital capacity (VC), arterial oxygen tension (Pa_{O_2}), pH and a decrease of residual volume (RV), total lung capacity (TLC) and arterial carbon dioxide tension (Pa_{CO_2}) (all statistically significant). No improvement was ascertained in the functional parameters of Group B. The expiratory muscles seem to play a determining role in ARF. We conclude that the iron lung is a useful therapeutic defence in removing muscular fatigue and in restoring a good level of respiratory compensation of ARF in severe COPD.
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In chronic obstructive pulmonary disease (COPD) complicated by chronic respiratory insufficiency, the respiratory muscles become progressively weaker because of the hyperinflation secondary to bronchial obstruction and the reduced energy supply linked to chronic hypoxaemia. The weakness of inspiratory muscles causes a malfunction of the respiratory pump with a progressive retention of CO_2 [1]. Correlation between CO_2 retention and inspiratory muscle fatigue was found in patients affected by COPD, in which the inspiratory external workload was increased [2] and in subjects mechanically ventilated during the weaning period [3].

Muscular respiratory fatigue can be removed by: 1) reducing the resistive load by means of bronchodilators that lower the airways resistance; 2) increasing the contractile efficiency of muscles by means of medication [4–6] and/or an adequate energy contribution *e.g.* supplying more oxygen; 3) resting the inspiratory muscles in order to obtain a physiological recovery [7–11].

The aim of this research was to estimate the effects of a ventilatory treatment (acting externally and by intermittent negative pressure) on respiratory muscle fatigue in acute respiratory failure (ARF) in subjects affected by severe COPD. The data reported by other authors on this treatment were observed only in subjects with COPD in a clinically stable state.

Patients and method

Nine subjects were studied (Group A), eight males and one female, mean age 61±7.7 yrs, affected by severe COPD in ARF, stage 1 according to the classification of Weitzenblum [12]. No patient presented clinical or laboratory signs of bronchopneumonia. Rales were not found at physical chest examination. The leucocyte count was normal and the chest X-ray showed the characteristic features of COPD (hyperinflation, prominent pulmonary trunk, large hilar vessels). All patients presented signs of peripheral oedema and revealed an increase of the exertional dyspnoea a few days before hospital admission. Five patients were followed in the Outpatient Department, four were transferred to the Intensive Care Unit from other departments because of their deteriorating clinical respiratory conditions. Before admission, the five outpatients practised home oxygen therapy for more than 18 h per day with a liquid oxygen system. Three of them declared misuse of the oxygen therapy (less than 15 h per day), the other two used the oxygen as prescribed. All of the patients had taken drugs which could have influenced muscular functions, such as theophylline and/or $beta_2$-agonist. This treatment was associated with cardiokinetic, mucolytic and diuretic agents. The same therapy was continued in the hospital.

All of the patients performed respiratory function tests in a sitting position: vital capacity (VC) and forced expiratory volume in one second (FEV_1) were obtained by a pneumotachograph (Jaeger) by means of flow integration. Residual volume (RV), total lung capacity (TLC) and airway resistance (Raw) were determined by means of a constant volume body plethysmograph (Body Screen, Jaeger). The maximum inspiratory pressure (Pimax) at RV and the maximum expiratory pressure (Pemax) at TLC were measured at the mouth by means of a pressure transducer and according to the method of Black and Hyatt [13]. Arterial oxygen tension (Pao_2), arterial carbon dioxide tension ($Paco_2$) and pH were determined while the patients breathed air, on the basis of an arterial blood sample taken from the radial artery and analysed on an ABL 300 (Copenhagen Radiometer). All parameters were measured in the basal condition and repeated 12 h after the end of a cycle of 7 days of ventilatory treatment carried out with an intermittent negative pressure ventilator acting externally (Iron lung, Pulmolife Mod.). The ventilator was set to deliver pressures ranging from -50 to -60 cmH_2O at a rate of 15 breaths·min^{-1}. The pressures used were needed in our patients to obtain a tidal volume (recorded at the mouth by means of Wright's ventilograph) of more than 500 ml. Arterial blood gases were determined intermittently during the treatment sessions in the iron lung. The ventilatory treatment was carried out intermittently during the day (8 h per day, with cycles of 2 h each) while patients were awake. Oxygen was supplied 24 h a day by means of nasopharyngeal catheters at a mean flow rate of 2 l·min^{-1} according to each patient's need.

In order to improve the clearance of secretions during treatment sessions, the iron lung was set in a way which permitted postural drainage. The patients were also helped by nurses to expectorate by means of directed coughing.

Seven subjects (Group B), five males and two females, mean age 61±6.6 yrs, affected by COPD in stable chronic respiratory failure (CRF), were studied as controls. This group presented a similar degree of respiratory muscle weakness and volumetric and respiratory mechanical impairment compared to Group A, but a lower hypoxaemia and hypercapnic acidosis severity. This group underwent the same medical therapy including oxygen therapy 24 h a day as Group A, but did not undergo ventilatory treatment. The choice of patients in stable condition as control group was motivated by the ethical impossibility of withholding ventilatory treatment to patients suffering from ARF.

For both groups the same parameters were determined in air, after oxygen had been disconnected for an hour, both at the beginning and after 7 days. Statistical analysis was performed using Student's t-test for paired data within the same group and nonpaired data when groups were compared.

All of the subjects were thoroughly informed of the aims of the research and gave their consent.

Results

In table 1, the mean values for both Groups of VC, FEV_1, RV, TLC, expressed as percentage of predicted [14], and of Raw, Pao_2, $Paco_2$, pH, Pimax and Pemax, expressed as absolute values, are shown. In Group A the analysis of the data revealed a marked degree of bronchial obstruction (FEV_1 26.1±8.3%, Raw 9.5±3.5 $cmH_2O·l^{-1}·s$), associated with marked hyperinflation (RV 206±41.7%). The mean value of the VC was reduced (55±10.3%), whilst that of the TLC was slightly increased (110±15.5%). The Pao_2 (5.8±1.2 kPa) showed the presence of a very severe arterial hypoxaemia associated with a decompensated respiratory acidosis ($Paco_2$ 9.2±1.1 kPa; pH 7.33±0.028). The low Pimax (-46±25.1 cmH_2O) and Pemax (72±31.3 cmH_2O) values indicated a marked reduction of the respiratory muscle strength. The baseline data collected from the control group (Group B) demonstrated an obstructive ventilatory damage similar

Table 1. – Basal respiratory function data of the two groups of subjects affected by COPD in ARF (Group A) and in stable state (Group B)

	Group A 8 M, 1 F		Group B 5 M, 2 F		
	Mean	SD	Mean	SD	p
Age yrs	61	7.7	61	6.6	
VC % pred	55	10.3	61	17.5	NS
FEV_1 % pred	26	8.3	27	7.7	NS
RV % pred	206	41.7	169	59.1	NS
TLC % pred	110	15.5	100	17.1	NS
Raw $cmH_2O·l^{-1}·s$	9.5	3.5	9.7	4.4	NS
Pao_2 kPa	5.8 (43.2)*	1.2 (8.7)*	8 (60.3)*	0.64 (4.8)*	<0.001
$Paco_2$ kPa	9.2 (68.5)*	1.1 (7.9)*	7 (52.8)*	0.71 (5.3)*	<0.001
pH	7.33	0.028	7.36	0.02	<0.01
Pimax cmH_2O	-46	25.1	-49	19.6	NS
Pemax cmH_2O	72	31.3	96	43.8	NS

*: mmHg; COPD: chronic obstructive pulmonary disease; ARF: acute respiratory failure; M: male; F: female; SD: standard deviation; VC: vital capacity; FEV_1: forced expiratory volume in one second; RV: residual volume; TLC: total lung capacity; Raw: airway resistance; Pao_2 and $Paco_2$: arterial oxygen and carbon dioxide tension, respectively; Pimax and Pemax: maximum inspiratory and expiratory pressure, respectively; NS: nonsignificant.

Table 2. – Respiratory function data of the subjects with COPD in ARF (Group A), before and after seven days of treatment with intermittent negative pressure ventilation (INPV)

	Before INPV		After INPV		
	Mean	SD	Mean	SD	p
VC % pred	55	10.3	62	13.7	<0.01
RV % pred	206	41.7	170	38.2	<0.01
TLC % pred	110	15.5	101	13.2	<0.02
Raw $cmH_2O \cdot l^{-1} \cdot s$	9.5	3.5	8.3	4.21	NS
Pao_2 kPa	5.8 (43.2)*	1.2 (8.7)*	8.2 (61.5)*	0.75 (5.6)*	<0.001
$Paco_2$ kPa	9.2 (68.5)*	1.1 (7.9)*	7.1 (53.0)*	0.61 (4.6)*	<0.001
pH	7.33	0.028	7.37	0.018	<0.01
Pimax cmH_2O	-46	25.1	-63.5	27.8	<0.001
Pemax cmH_2O	72	31.3	95.7	38.3	<0.05

*: mmHg. For definitions of other abbreviations see legend to table 1.

Table 3. – Respiratory function data of the subjects affected by COPD in a stable state of chronic respiratory failure (Group B), before and after seven days of medical therapy without intermittent negative pressure ventilation (INPV)

	Basal values		After seven days		
	Mean	SD	Mean	SD	p
VC % pred	60	17.5	63.8	15.8	NS
RV % pred	169	59.1	161.6	70.4	NS
Raw $cmH_2O \cdot l^{-1} \cdot s$	9.7	4.4	11	5.3	NS
Pao_2 kPa	8 (60.3)*	0.64 (4.8)*	8.4 (63.3)*	0.8 (5.9)*	NS
$Paco_2$ kPa	7 (52.8)*	0.71 (5.3)*	6.9 (52.0)*	0.61 (4.58)*	NS
pH	7.36	0.02	7.37	0.03	NS
Pimax cmH_2O	-49	19.6	-47	16.4	NS
Pemax cmH_2O	96	43.8	93.3	44.9	NS

*: mmHg. For definitions of other abbreviations see legend to table 1.

to that observed in Group A, a global respiratory insufficiency partially compensated and a reduced contractile strength of the respiratory muscles.

In table 2 the mean values and the results of the statistical analysis of all the parameters found in Group A, before and after assisted mechanical ventilation, are shown.

All of the parameters, except Raw, showed a net improvement after the ventilatory treatment. The Pimax rose from a mean value of -46±25.1 to -63.5±27.8 cmH_2O ($p<0.001$); the Pemax underwent an analagous increase from the average basal value of 72±31.3 to 95.7±38.3 cmH_2O ($p<0.05$). The VC increased from 55±10.3 to 62±13.7% ($p<0.01$), while the RV and the TLC diminished. The RV dropped from the basal mean value of 206±41.7 to 170±38.2% ($p<0.01$), and the TLC from 110±15.5 to 101±13.2% ($p<0.02$). The Pao_2 and pH rose after treatment from the basal mean value of 5.8±1.2 to 8.2±0.75 kPa ($p<0.001$) for the Pao_2, and 7.33±0.028 to 7.37±0.018 ($p<0.01$) for the pH, respectively.

The $Paco_2$ underwent a net reduction after ventilatory treatment from 9.2±1.1 to 7.1±0.61 kPa ($p<0.001$). The Raw did not change significantly after treatment: from 9.5±3.5 to 8.3±4.2 $cmH_2O \cdot l^{-1} \cdot s$ (NS). In table 3 the data collected before and after 7 days of exclusively pharmacological treatment of Group B are shown. A comparison of data does not reveal any significant statistical variation. We have, furthermore, looked for an eventual correlation between Pimax and Pemax and the various parameters studied in the group which underwent assisted mechanical ventilation. The Pimax correlated only with VC both before ($r=0.69$, $p<0.05$) and after treatment ($r=0.79$, $p<0.05$), with CO_2 in basal condition ($r=-0.87$, $p<0.01$) and with Pemax ($r=0.94$, $p<0.001$) in basal condition. The Pemax correlated in basal condition with $Paco_2$ ($r=-0.82$, $p<0.01$), Pao_2 ($r=0.68$, $p<0.05$), pH ($r=0.84$, $p<0.01$) and with the Pimax ($r=0.94$, $p<0.001$). The values of Pimax and Pemax were corrected for the RV (Pimax/RV, Pemax/RV): the two ratios were significantly correlated both before ($r=0.84$, $p<0.01$) and after ventilatory treatment ($r=0.72$, $p<0.05$).

Discussion

The action of INPV on the patients studied determined: 1) improved respiratory muscle contractile efficiency (increase of both Pimax and Pemax); 2) improvement in the exchange of gases (increase of the Pao_2) and ventilatory efficiency (reduction of the $Paco_2$); 3) significant variation of the pH from decompensated acidosis to metabolic compensation; 4) increase of VC and decrease of RV; 5) Raw unchanged before and after treatment.

An improved respiratory muscle contractile efficiency was noted in subjects affected by COPD in stable

clinical condition, who were treated with INPV in short-term [11, 15] or in long-term [9, 10]. INPV was found to reduce the electrical activation of the diaphragm even 20 min after the start of ventilatory treatment [8]. Data relating to these patients confirm the findings reported by other authors about the action of INPV on recovering the contractile strength of the respiratory muscles. The improvement of the Pimax and Pemax in the group of ventilated subjects and the absence of variation in the control group proves that ventilatory treatment is efficient in removing muscle fatigue, being the only difference in treatment between the two groups. Nevertheless, our patients varied from those described by other authors in the degree of clinical and functional impairment.

In healthy subjects an acute respiratory acidosis equivalent to a $Paco_2$ of about 54 mmHg was found to predispose the respiratory muscles to fatigue [16]. In patients with COPD an inverse correlation was found between Pimax <55 cmH_2O and $Paco_2$ >50 Torr [17]. The combined alterations of these parameters is therefore an index of the respiratory muscle weakness.

Before ventilatory treatment, our patients were probably in a state of muscular fatigue (Pimax -46±25.1 cmH_2O and $Paco_2$ 68.5±7.9 mmHg) which was sustained by energetic (hypoxaemia), metabolic (acidosis) and mechanical (hyperinflation) factors. Hyperinflation causing the shortening of inspiratory muscles and the lengthening of expiratory muscles placed them far from their geometric point of optimum efficiency. The strength of the respiratory muscles is in fact directly related to the resting length of their fibres and inversely related to lung volume [18, 19]. The increase of RV in our patients was probably due to the increase of bronchial resistance and in part to a state of continuous inspiratory muscle activity as MARTIN *et al.* [20] found in asthmatic subjects. The decrease of RV found after ventilatory treatment associated with unchanged Raw, but with a net improvement in the contractile force of the inspiratory muscles, supports this hypothesis. In patients with COPD, the Pimax is determined both by generalized muscle weakness, of which the Pemax represents the marker, and by mechanical drawbacks in the inspiratory muscles as a result of hyperinflation [17].

The reduction of Pemax in our patients could be related both to the increase of the TLC that determined an excessive lengthening of the muscle fibres, and to the arterial hypoxaemia associated with hypercapnia and decompensated respiratory acidosis. The noncorrelation, however, between Pemax and TLC and the high degree of correlation between Pemax and Pao_2 ($p<0.05$) and Pemax and pH ($p<0.01$) led us to deduce that Pemax was conditioned predominantly by energetic and metabolic factors. Since the Pimax did not correlate with Pao_2 and pH but presented a high degree of correlation with Pemax before treatment, we deduced that in our patients the expiratory muscles conditioned the function of the inspiratory ones. The mechanism is not clear. We can hypothesize that the lack of counterbalancing between inspiratory and expiratory muscles moves the elastic point of balance of the chest-lung system towards the TLC with a consecutive shortening of the inspiratory muscles. The ventilatory treatment, removing respiratory muscle fatigue, induced a net increase of the contractile force of the expiratory muscles. The latter, bringing the chest-lung system towards its point of elastic equilibrium (reduction of the RV), permitted the inspiratory muscles to take up a position at a better degree of length in order to develop strength. In our opinion, this is supported by the high degree of correlation found between Pimax and Pemax after ventilatory treatment, when the parameters were corrected respectively for the RV ($p<0.01$).

The iron lung, even though now not greatly used, was proved to be useful by several authors [21–23], for removal of acute respiratory acidosis in subjects affected by COPD.

Our results demonstrate that the INPV used in acute respiratory decompensation in subjects with severe COPD exerts a vicarious ventilatory action and restores a good level of respiratory compensation removing muscle fatigue. The expiratory muscles in this phase of the illness become very important: as they recuperate contractile force after ventilatory treatment, they move the chest-lung system towards a better point of elastic equilibrium with consequent reduction of the energy cost of the work of breathing.

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Insuffisance musculaire respiratoire dans la décompensation respiratoire aiguë de sujets atteints de BPCO sévère: traitement par ventilation sous pression négative intermittente.
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RÉSUMÉ: Neuf sujets (âge moyen de 61±7.7 années, 8H, 1F), atteints de BPCO grave en décompensation respiratoire aiguë (groupe A) et atteints en outre d'une faiblesse marquée des muscles respiratoires, ont subi une ventilation sous pression négative intermittente (INPV). Cette INPV a été effectuée grâce à un poumon d'acier (pendant 8 heures par jour, pendant 7 jours), les pressions du ventilateur s'étendant de -50 à -60 cmH_2O. Sept sujets (âge moyen de 61±6.6 années, 5H, 2F), atteints de BPCO avec décompensation respiratoire chronique stabilisée (groupe B), ont servi de contrôles et ont été soumis au même traitement médical que le groupe A, mais sans ventilation. Des tests fonctionnels respiratoires ont été pratiqués dans des conditions basales, puis après 7 jours de traitement. Après traitement ventilatoire, le groupe A montre une augmentation significative de PImax ($p<0.001$), de PEmax ($p<0.05$), de CV ($p<0.01$), et une réduction significative de VR ($p<0.01$) et de TLC ($p<0.02$). Une augmentation significative de la Pao_2 ($p<0.001$) et du pH ($p<0.01$), est associée à une diminution nette de la $Paco_2$ ($p<0.001$). Raw reste pratiquement inchangé (p=NS). L'on n'a pas observé d'amélioration des paramètres fonctionnels dans le groupe B. La corrélation observée dans le groupe A entre PEmax: Pao_2, pH, $Paco_2$, suggère que les muscles expiratoires dans la décompensation respiratoire aiguë subissent des altérations métaboliques et un déficit nutritionnel plus marqués que les muscles inspiratoires, et que dès lors ils jouent un rôle déterminant en conditionnant le fonctionnement de ces derniers. Le traitement ventilatoire au poumon d'acier s'avère une mesure thérapeutique utile, en supprimant l'état de fatigue musculaire et en restaurant un niveau satisfaisant de compensation respiratoire dans les décompensations respiratoires aiguës chez les sujets atteints de BPCO avancée.

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