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P0480

Changes of breathing pattern and mouth occlusion pressure in patients with airway obstruction after bronchodilator inhalation

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It is known that neuromuscular drive is increased in patients with airway obstruction. The aim of the study was to estimate an influence of beta-agonist on breathing pattern and mouth occlusion pressure (P0.1) in patients with reversible and non-reversible airway obstruction. Ventilatory function tests, pattern of breathing, mouth occlusion pressure (P0.1) were measured in 23 patients (15 with bronchial asthma, 8 with COPD), age 48.4 ± 16.7 . Their mean FEV1 was 1.6 ± 0.7 l/s, mean Raw 0.79 ± 0.36 kPa/s/l. Control group consisted of 20 healthy subjects, aged 38.2 ± 8.6 years. Pattern of breathing in patients was changed in comparison to control group – the tidal volume and minute ventilation were increased (0.81 ± 0.19 vs 0.67 ± 0.16 l and 13.3 ± 3.7 vs 9.9 ± 1.9 l/s), mean inspiratory flow (Vt/Ti) was higher, inspiratory and total time were shortened but these differences did not reach statistical significance. Also mouth occlusion pressure and inspiratory impedance were significantly increased (P0.1 3.6 ± 1.6 vs 1.6 ± 0.3 cmH2O and P0.1/Vt/Ti 6.6 ± 2.3 vs 3.8 ± 1.0 cmH2O/l/s).

In all patients these measurements were repeated 20 minutes after β -agonist inhalation (0.2 mg fenoterol). Patients were divided in two groups according to reaction to the drug. Gr.A consisted of 15 pts. (mostly with bronchial asthma), who responded to bronchodilator (increase in FEV1 $\geq 15\%$) and revealed significant decrease of P0.1 (3.9 ± 1.8 vs 2.6 ± 1.2 cmH2O) and P0.1/Vt/Ti (6.6 ± 2.4 vs 4.2 ± 1.1 cmH2O/l/s). Gr.B consisted of 8 pts (mostly with COPD) who did not respond to bronchodilator (Δ FEV1 $\leq 15\%$) and did not present changes in P0.1 and P0.1/Vt/Ti.

The reduction of neuromuscular drive and inspiratory impedance in responsive patients (Gr.A) is a consequence of diminishing of nonelastic respiratory load and seems to be an additional advantage for the patient.

P0481

The problem of measuring perception of dyspnoea

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Background: During recent decades we have become more interested in how people perceive their dyspnoea. Research in this area is focused on the relationship between physiologic changes and the associated sensation. One of the problems has been the difficulty in quantification of this relationship. In literature several different measurements for this quantification are being used among each other: (1) The 'evoked sensation/physical change' slope; (2) the correlation between these two parameters and (3) any increase in magnitude of the evoked sensation for a given physical change. These three measurements are indexes of dyspnoea sensitivity for each subject: the steeper the slope or the stronger the correlation the better the perception. The third measurement considered any increase in magnitude of the evoked sensation as a good perception. However, the question is: are the results of these different methods comparable. We have studied the perception of dyspnoea during a histamine provocation test in asthmatic patients by three different methods to assess the comparability of these techniques.

Methods: In 154 asthmatic patients a histamine provocation test was obtained. The FEV₁ was measured after each inhalation of histamine, and the subjects were asked to rate their symptoms of dyspnoea on a Visual Analogue Scale (VAS). VAS score were dichotomized as follows. Any increase in VAS score was coded as good perception; no change in VAS score was coded as no perception. For further analyses, correlations and slopes were used to analyze the relationship between VAS score and the reduction in FEV₁. The results of these three different methods were compared.

Results: Dyspnoea as indicated by the VAS scale increased as the FEV₁ decreased in 150 subjects (97.4%). The correlation and slope between increase in VAS score and decrease in FEV₁ could be analyzed respectively in 137 and 122 subjects. The group mean correlation was 0.83 ± 0.30 SD. The group mean slope of perception of dyspnoea/percentage fall in FEV₁ was 1.11 ± 0.75 SD. After dichotomy between good and bad perceivers, we obtained different proportions for each method (Table 1). Although the relation between the individual slopes and correlation was significant ($p > 0.001$), the correlation between the methods was not very convincing ($r = 0.41$).

Table 1: Percentages of good and bad perceivers for each method.

Method	Good perception	No good perception
Slope	71.3%	28.7%
Correlation	89.1%	10.9%
Any increase in VAS score	97.4%	2.6%

Conclusion: In this study we have shown that obtained perception of dyspnoea during a histamine provocation test by three different methods leads to different results. Therefore, the qualification 'good perceiver' depends on the kind of measurement.

P0482

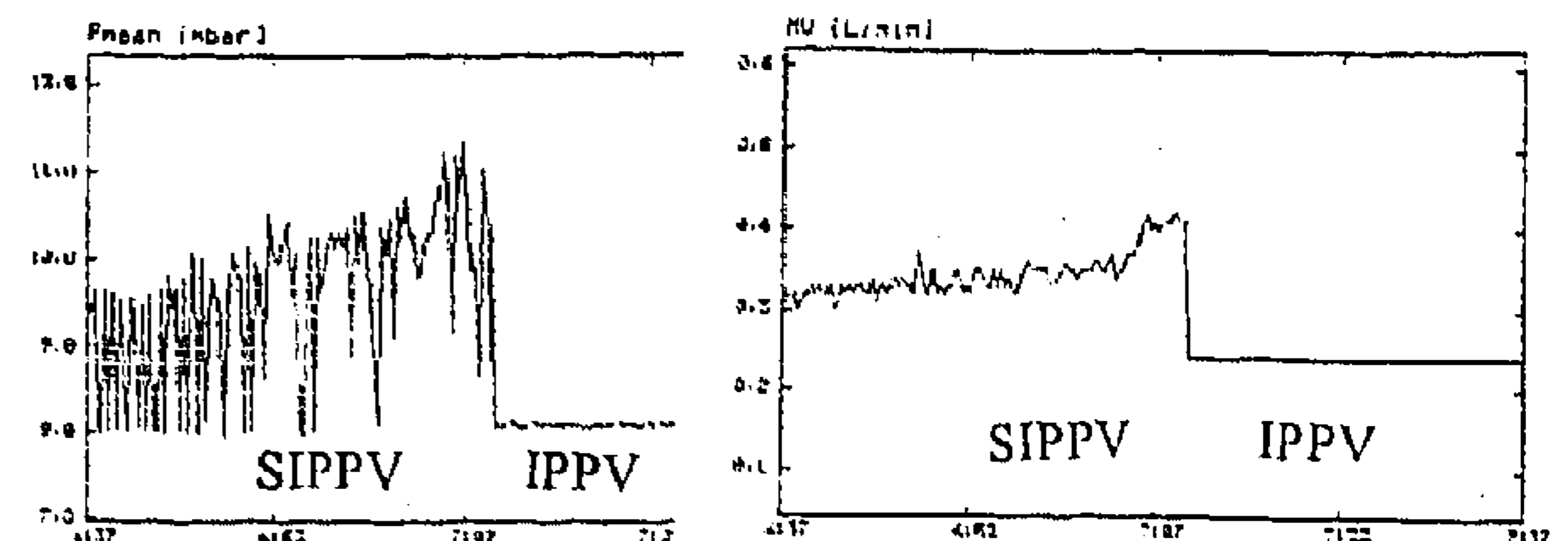
Triggered hyperventilation – Is it safe?

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Objective: We tried to establish whether the hyperventilation created by a ventilator during Synchronised IPPV evoked by fluid in the ventilatory hoses could or could not be safe for the brain and lungs.

Methods: We measured the cerebral blood flow velocity (CBFV) in 3 neonates with pulsed Doppler during SIPPV. In all neonates we measured the pneumotachogram with Baby link Dräger 8000. We compared the cerebral blood flow velocity with pCO₂, ventilatory setting, minute ventilation (MV) and clinical condition.

Results: Changes in CBFV were found in all neonates early after the increase of minute ventilation created by hyperventilation during SIPPV, with an appropriate response of CBF to the decrease in pCO₂. The monitoring of MV can indicate instability of the volumes related to the hyperventilation created by triggered ventilation. The bubbling of water in the hoses could increase both the ventilatory rate and MV, decrease of CBFV, mean velocity and pCO₂.



Conclusion: It is very important to have more and continual informations about changes in blood gases or brain perfusion during conventional ventilation. The evaluation of pneumotachograms could help to detect uncontrolled hyperventilation, especially in neonates with SIPPV. Uncontrolled hyperventilation generated by the fluid in the hoses could increase the risk for both hypoperfusion of the brain and barotrauma.

Pulmonary gas transfer and exercise

P0483

Factors underlying the relationship of transfer factor (TI, CO) to alveolar volume (VA)

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Background and Aims: In normal subjects TI, CO is described more accurately by age, stature (St) and VA than by age and St alone (Chinn et al, *Eur Respir J* 1996; 9: 1269–1277). TI, CO reflects the diffusing capacity of the alveolar capillary membrane (Dm) and volume of blood in alveolar capillaries (Vc); hence our question: Is the contribution of VA to TI, CO mediated via an effect on Dm or Vc or both variables?

Methods: Measurements of Dm and Vc by the single breath CO method for 46 asymptomatic men (33 smokers and 13 non-smokers) from a previous study (Cotes et al. *Br J Industr Med* 1983; 40: 13–21) were re-analysed. The mean age was 41.7 (range 29–72) yr. To avoid co-linearity with St, VA was expressed as VA.St⁻². Dm and Vc were analysed in the reciprocal forms in which they were derived.

Results: TI, CO was related to VA, St⁻², stature and smoking ($p < 0.05$); age contributed in the absence of VA.St⁻². 1/Dm was negatively related to VA.St⁻². St was significant in the absence of VA. 1/Vc was related to St but not to VA. St⁻².

Conclusions: VA contributes to between subject variation in TI, CO by affecting Dm. This result resembles that within subjects measured at different levels of VA (Hamer NJ, *Clin Sci* 1963; 24: 275–285).

P0484

Consequences of carbon monoxide from tobacco smoke on hemoglobin

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We studied 132 consecutive subjects (77% males), age 60.4 ± 12.7 yr (22–85 yr) sent to our lab for blood gas analysis and with PaO₂ higher than 65 mmHg. They answered a questionnaire asking for age, number of cigarettes smoked per day the last fifteen days, number of hours per day they stayed in a room where other people were smoking, their occupations and if they had gas or fuel heating systems at home. 3 ml blood samples were anaerobically drawn from the radial artery. Blood samples were tested twice with an Instrument Laboratories (Lexington, MA) IL 482 co-oxymeter. Patients were divided in non-smokers ($n = 46$), passive smokers ($>$ two hour in a room where somebody was smoking) ($n = 29$), smokers $<$ 10 ($n = 12$), smokers $>$ 10 $<$ 20 ($n = 21$), smokers $>$ 20 $<$ 40 ($n = 25$) and smokers $>$ 40 ($n = 9$).