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Head helmet versus face mask for non-invasive continuous positive airway pressure: a physiological study

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Abstract *Objective:* To assess selected physiological effects of non-invasive continuous positive airway pressure delivered by head helmet ($CPAP_H$), a special interface device designed to completely contain the head of the patient, compared to face mask ($CPAP_M$). *Design:* Randomized physiological study. *Setting:* University research laboratory. *Patients:* Eight healthy volunteers. *Intervention:* Continuous positive airway pressure delivered by face mask and $CPAP_H$ in random order. Three gas flow rates (20-30-40 l/min and 30-45-60 l/min, respectively, for $CPAP_M$ and $CPAP_H$) and four CPAP levels (0-5-10-15 cmH_2O) were employed in a randomized sequence. *Measurements and results:* In each patient we monitored airway pressure, esophageal pressure, expiratory flow, and inspiratory and expiratory CO_2 concentration. End-expiratory lung volume changes from CPAP 0 were measured by inductance plethysmography. The application of increased levels of CPAP resulted in a significant increase in end-expiratory lung volume, similar for $CPAP_H$ and $CPAP_M$. Inspiratory changes of airway pressure were comparable for the two CPAP modes. Inspiratory CO_2 concentration was higher during $CPAP_H$ (significantly decreased at increased gas flow rates), compared to $CPAP_M$. *Conclusions:* Continuous positive airway pressure delivered by head helmet is as effective as $CPAP_M$ in increasing end-expiratory lung volume and in compensating for airway pressure changes without the need of a reservoir bag. Higher gas flow rates are necessary to maintain a relatively low inspiratory CO_2 concentration.

Keywords Continuous positive airway pressure · Head helmet · Face mask · Hypoxemic respiratory failure · Healthy volunteers

Introduction

Patients with acute hypoxemic respiratory failure often benefit from positive airway pressure therapy. In order to decrease the need for endotracheal intubation, non-invasive continuous positive airway pressure (CPAP) delivered by face or nasal mask has been widely applied in patients affected by this condition, be it cardiogenic [1, 2, 3, 4] or non-cardiogenic [5, 6, 7, 8, 9, 10, 11, 12, 13]. Despite a number of encouraging reports, several factors limit the application of non-invasive CPAP and of non-invasive ventilation in general. The use of a face mask, which is the most commonly used interface in the acute adult setting [14, 15, 16], appears to be particularly relevant among those factors. Inability to fit the mask to the patient properly and to avoid relevant air leaks, indeed, accounts for a large proportion of non-invasive ventilation failures [16]. The need for a specifically trained team is another major limitation of non-invasive ventilation [16, 17]. Moreover, patient discomfort, nasal pain and ulcerations of the nose bridge may limit continuous or long-term application of non-invasive ventilation, thus decreasing its efficacy [16, 18].

At our institution, to overcome most of these problems, we provide CPAP by a head helmet ($CPAP_H$) instead of a face mask ($CPAP_M$) [19]: the helmet is a special interface device designed to contain the head of the patient completely and it provides a seal all around the patient's neck. We have been using $CPAP_H$ for patients with acute hypoxemic respiratory failure for more than 15 years. Given a minor amount of personnel training and appropriate settings, $CPAP_H$ has proved feasible in most of the hospital wards, as well as in the emergency department. An internal protocol has been developed for $CPAP_H$ as first-line treatment in acute cardiogenic edema, in both intra- and extra-hospital settings [19]. Using the helmet, Antonelli et al. successfully delivered non-invasive pressure support ventilation in patients with acute hypoxemic respiratory failure [20].

The aim of this study was to assess selected physiological effects of $CPAP_H$, compared to $CPAP_M$, in healthy volunteers. We specifically investigated the extent to which $CPAP_H$ may increase end-expiratory lung volume (EELV) and may maintain a constant airway pressure, so as to minimize the work of breathing [21]. Moreover, since a certain level of CO_2 rebreathing is expected during $CPAP_H$ [22], we measured the inspiratory CO_2 concentration ($PiCO_2$).

Methods

We studied eight healthy non-obese, non-smoking, young volunteers (3 females; age 27±3 years) after obtaining informed consent. All subjects were unaware of the purpose of the study. The study was approved by the institutional ethics committee.

Experimental setup

Equipment

The helmet (Sea-Long Medical Systems, Louisville, USA) is made of two parts: (1) a transparent PVC hood which contains the entire subject's head is fitted tightly on (2) a rigid acrylonitrile butadiene styrene ring supporting a latex-free rubber neckseal. Two pipe-connectors are fitted in the upper and lower parts of the hood for the expiratory and the inspiratory limbs of the circuit. The face masks (Harol, Gibeck, Vital Signs) were individually selected for each subject, aiming at the best compromise between comfort and air tightness.

The CPAP circuit (Fig. 1) consisted of (1) flexible 20 mm ID PVC pipes (Mallinckrodt, Mirandola, Modena, Italy), (2) an adjustable flowmeter and (3) a selected spring-loaded, positive end-expiratory pressure (PEEP) valve (5, 10, 15 cmH_2O; Vital Signs, Totowa, NJ). When $CPAP_M$ was applied, a T-piece was inserted in the inspiratory limb and connected to a 10-l high compliance rubber bag (Harol, S. Giuliano, Milan, Italy), serving as a compliant reservoir, to minimize inspiratory pressure drops [21, 23]. Non-humidified medical air was used throughout the experiment as the inspiratory gas.

Monitoring

Airway pressure (Paw) was measured by a small bore (2 mm ID) plastic tube fitted to the anterior part of the mask or through the neckseal of the helmet, with the tip placed close to the mouth of the subject. Esophageal pressure (Pes) was measured by an esophageal balloon catheter (Smartcath, Bicore Monitoring System, Irvine, CA) inserted through a nostril and positioned in the mid-esophagus. The correct position of the esophageal balloon was checked by means of the airway occlusion technique [24]. Paw and Pes lines were connected to calibrated pressure transducers (Bentley Trantec, Armstrong, CA). The expiratory flow was measured by a calibrated heated pneumotachograph (Fleisch #2) placed on the expiratory limb and connected to a differential pressure transducer (Validyne MP45; Validyne, Northridge, CA).

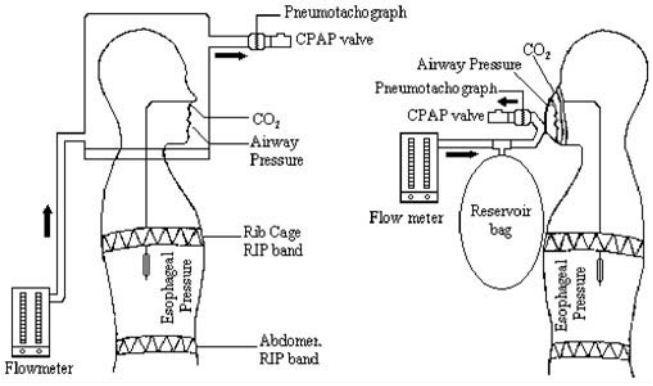

Fig. 1 Continuous positive airway pressure circuit

The CO_2 concentration was measured by a capnometer (Datex Normocap, Instrumentarium, Helsinki, Finland) sampling by a thin catheter inserted through an adapter on the mask or through the neckseal of the helmet, and advanced for approximately 1 cm in a nostril.

We measured changes in EELV by means of respiratory inductive plethysmography (RIP) (Respitrace Plus, NIMS, Miami Beach, FL) operating in DC mode. We first let the Respitrace respiratory monitor perform a 5-min auto-calibration to proportionate the electrical gains of the rib cage and abdomen coil signals correctly [25]; we then connected the subjects to a Fleisch pneumotachograph through a mouthpiece and the RIP sum signal (the sum between the rib cage and abdomen signals) was calibrated against the pneumotachograph [25]. The RIP calibration was then checked by comparing tidal volumes (Vt), measured by RIP and by pneumotachograph, over several breaths. Calibration was repeated if the RIP and pneumotachograph differed by more than 5%. Signals (Paw, Pes, flow, PCO_2, thorax, abdomen and sum motion) were conditioned through an 8-channel polygraph (5900 Signal Conditioner Cage, Gould Instruments, Cleveland, OH), digitalized at 100 Hz (12-bit resolution PCI-MIO-16E-4, National Instruments, Austin, TX) and then recorded by a PC (Labview 6.0.2, National Instruments, Austin, TX) for storage and analysis.

Experimental protocol

All the subjects, while sitting on a chair, received both $CPAP_M$ and $CPAP_H$ in random order.

For both $CPAP_M$ and $CPAP_H$ each subject received three different gas flows and four different CPAP levels (0, 5, 10, 15 cmH_2O). In order to investigate the CO_2 rebreathing issue and the effect of gas flow rate on $PiCO_2$, gas flow rates higher than those commonly required during $CPAP_M$ (provided with a reservoir bag), were used during $CPAP_H$. To this end $CPAP_H$ was conducted at 30, 45 and 60 l/min, but $CPAP_M$ at 20, 30 and 40 l/min. With both systems, first we randomized the gas flow rates and then, for each gas flow rate, we randomized the CPAP levels. At a

given flow, each CPAP level was applied for at least 5 min to obtain a stable respiratory pattern and this was followed by 5 min with CPAP 0 cmH_2O. The gas flow rate delivered by the flowmeter was checked, temporarily connecting the pneumotachograph to the inspiratory limb of the circuit before each flow step. At the end of each flow step, the RIP calibration was checked against the pneumotachograph and, if a difference of more than 5% was found, mask or helmet was removed and a complete RIP calibration procedure was performed as described above. The subjects were free to rest whenever desired between different gas flow rates. Moreover, after completing each set of either $CPAP_H$ or $CPAP_M$, the subjects were allowed to rest for at least 10 min. After all resting periods, calibration of the RIP was repeated.

Measurements and calculation

For each step, all parameters were obtained from a selected 1-min recording, closest to the end of the 5-min run. The inspiratory (Ti) and expiratory (Te) time, Vt, inspiratory and expiratory flow (computed as Vt/Ti and Vt/Te, respectively), respiratory rate [RR, obtained as 60/(Ti+Te)], were averaged from the 1-min selected recording of RIP sum traces. Minute ventilation (Ve) was computed as Vt*RR.

An ideal CPAP system should maintain a constant airway pressure throughout respiration [21]. We therefore measured the inspiratory trough (ΔPaw_i, the inspiratory drop) and the expiratory peak (ΔPaw_e), both as differences from the zero flow end-expiratory airway pressure level (all data averaged in each individual over 1 min). Inspiratory $PiCO_2$ was identified by inspection as the plateau inspiratory value of the capnogram and the end-tidal PCO_2 ($PetCO_2$) as the highest expiratory value. The contribution of rib cage to Vt was computed as the ratio between inspiratory rib cage changes and Vt (RC%).

Changes in EELV for the rib cage component ($\Delta EELV_{RC}$), for the abdomen component ($\Delta EELV_{AB}$) and for the whole respiratory system ($\Delta EELV$) were computed as differences between the average end-expiratory values over 1 min during CPAP and the average end-expiratory values at CPAP 0 obtained from all breaths in the minute immediately before CPAP application. On the same breaths, we measured and averaged the changes in end-expiratory esophageal pressure (ΔPes_{ee}), as defined by the zero-flow crossing.

Statistical analysis

The effects of the different CPAP modes, CPAP levels and gas flow rates on all the variables studied were tested by means of 3-way analysis of variance for repeated measurements. The Bonferroni correction was used for multiple comparisons. Linear regression analysis was used when required. Statistical analysis was done using SPSS software (SPSS, Chicago, IL). The data are expressed as averages ± SD unless otherwise specified.

Results

No adverse effects were observed or reported in any subject. Pes recording, however, was missed in one subject because of intolerance to the esophageal balloon catheter insertion. None of the subjects required resting periods during $CPAP_H$; four subjects required at least one resting period during $CPAP_M$. In two of the seven subjects equipped with the esophageal catheter, the analysis of Pes waveform showed indirect evidence of expiratory muscle contraction [26, 27], which was more pronounced at higher CPAP levels with no apparent differences between the two CPAP modes.

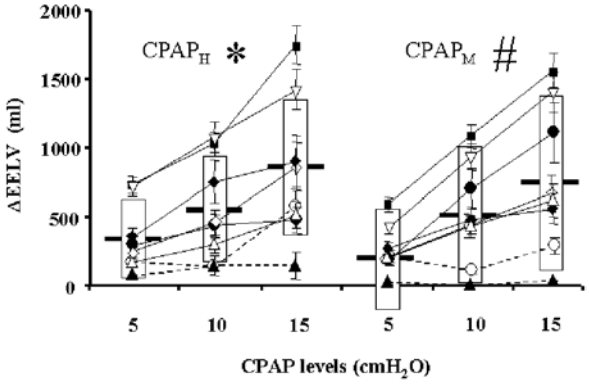

Fig. 2 Effect of different continuous positive airway pressure (CPAP) levels on end-expiratory lung volume (EELV) during CPAP with a helmet ($CPAP_H$, *left side*) and with a face mask ($CPAP_M$, *right side*) in each subject. *Dotted lines* refer to subjects with evidence of expiratory muscle contraction. *effect of CPAP levels during $CPAP_H$ $p<0.05$; # effect of CPAP levels during $CPAP_M$ $p<0.05$

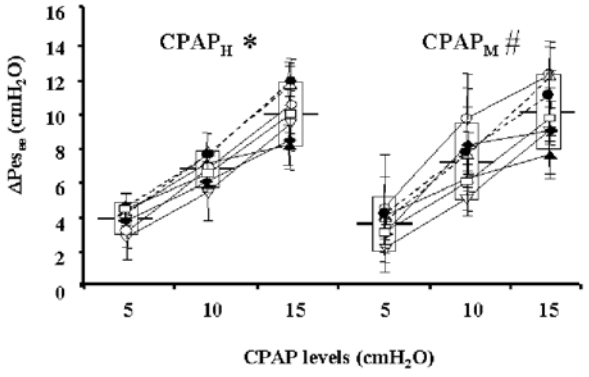

Fig. 3 Effect of different continuous positive airway pressure (CPAP) levels on end-expiratory esophageal pressure at increasing CPAP levels (ΔPes_{ee}) during CPAP with a helmet ($CPAP_H$, *left side*) and CPAP with a face mask ($CPAP_M$, *right side*) in each subject. *Dotted lines* refer to subjects with evidence of expiratory muscle contraction. *effect of CPAP levels during $CPAP_H$ $p<0.05$; # effect of CPAP levels during $CPAP_M$ $p<0.05$

End-expiratory lung volume changes

Application of an increasing level of CPAP resulted in a significant lung volume increase from baseline EELV ($\Delta EELV$ was 271±84 ml, 541±116 ml and 823±172 ml, respectively, at CPAP 5, 10 and 15 cmH_2O, $p<0.001$). EELV changes induced by CPAP were similar between $CPAP_H$ and $CPAP_M$ (Fig. 2, Table 1). The CPAP effect was significant both for $\Delta EELV_{RC}$ ($p<0.01$, Table 1) and

Table 1 Main effects of continuous positive airway pressure (CPAP) mode, CPAP levels and gas flow rate on end-expiratory lung volume (EELV) and end-expiratory esophageal pressure (Pes_{ee}) changes

	Flows (l/min)		Helmet			Mask		
	Helmet	Mask	CPAP-5	CPAP-10	CPAP-15	CPAP-5	CPAP-10	CPAP-15
$\Delta EELV_{AB}$ (ml)*	30	20	63±118	169±240	274±174	81±177	95±152	140±174
	45	30	73±209	237±142	330±121	117±114	306±557	435±572
	60	40	238±126	295±95	453±114	122±127	212±169	300±253
$\Delta EELV_{RC}$ (ml)*	30	20	192±147	334±303	527±409	164±153	266±228	454±329
	45	30	238±180	282±170	508±359	174±195	306±334	414±451
	60	40	210±233	386±387	531±471	103±82	361±314	576±466
$\Delta EELV$ (ml)*	30	20	254±246	502±479	800±535	245±274	361±310	594±443
	45	30	311±275	519±238	839±441	291±279	612±689	850±805
	60	40	450±336	681±459	985±565	225±137	573±449	876±603
ΔPes_{ee} (cmH_2O)*	30	20	4.2±0.8	7.2±0.9	10.7±1.6	3.8±1.7	7.5±2.4	10.0±2.6
	45	30	3.8±1.2	6.2±1.3	10.2±1.5	3.7±0.9	7.6±2.5	10.2±2.5
	60	40	3.7±0.5	6.6±0.8	8.7±1.7	3.1±2.0	6.2±1.7	9.7±1.8

Average values of changes in EELV (*ΔEELV*), the rib cage component of EELV ($\Delta EELV_{RC}$), the abdomen component of EELV ($\Delta EELV_{AB}$), and changes in end-expiratory esophageal pressure (ΔPes_{ee}) at different CPAP levels and gas flow rates during CPAP delivered by helmet ($CPAP_H$) and by face mask ($CPAP_M$).
*CPAP levels main effect $p<0.05$.

Table 2 Main effects of continuous positive airway pressure (CPAP) modes and CPAP levels on relevant variables

		CPAP levels (cmH_2O)			
		0	5	10	15
ΔPaw_i (cmH_2O)§ †	Helmet ‡	0.6±0.4	0.9±0.4	1.0±0.3	1.2±0.5
	Mask	1.0±0.4	1.1±0.4	1.2±0.4	1.3±0.4
ΔPaw_e (cmH_2O)[a,b]	Helmet[c]	0.3±0.2	0.5±0.2	0.7±0.3	0.8±0.4
	Mask	0.9±0.6	0.7±0.4	0.8±0.6	0.8±0.6
Vt (ml)*	Helmet	423±157	482±146	493±147	498±175
	Mask	490±150	571±142	593±184	632±228
$\%RIP_{RC}$	Helmet	38.8±7.7	40.9±12.1	42.4±14.6	47.4±16.2
	Mask	44.8±8.1	44.7±9.9	47.2±11.5	51.3±14.0
Ti (s)	Helmet	1.43±0.43	1.66±0.63	1.71±0.65	1.74±0.70
	Mask	1.69±0.48	1.81±0.52	1.69±0.44	1.73±0.59
Te (s)[a]	Helmet	2.47±0.81	2.89±1.01	3.28±1.25	3.09±1.17
	Mask	2.55±0.94	3.64±2.11	3.60±2.19	3.61±1.89
RR (bpm)[a]	Helmet	16.4±4.2	14.3±4.2	13.3±4.5	14.0±4.9
	Mask	15.6±4.9	13.4±6.1	13.9±6.2	13.7±6.3
Ve (l/min)	Helmet	7.2±4.09	6.9±3.19	6.6±3.32	6.8±3.55
	Mask	7.7±3.79	7.3±3.16	7.5±2.80	7.8±3.19
Vt/Ti (ml/s)	Helmet	318±148	311±113	307±108	301±115
	Mask	313±130	333±101	355±78	371±109
Vt/Te (ml/s)	Helmet	199±128	188±104	177±108	189±115
	Mask	222±127	201±109	204±100	209±105
$PetCO_2$ (mmHg)*,[a]	Helmet	28.7±2.3	28.9±3.1	30.5±3.1	30.9±3.6
	Mask	25.3±3.6	27.6±4.1	28.5±4.8	29.6±5.5
$PiCO_2$ (mmHg)*	Helmet	3.2±0.7	3.1±0.8	3.1±1.0	3.1±1.1
	Mask	0.7±0.8	0.8±0.8	0.8±0.7	0.9±0.9

Average values of main variables at different CPAP levels during $CPAP_H$ and $CPAP_M$
See text for abbreviations
*$CPAP_H$ vs $CPAP_M$ $p<0.05$
[a] CPAP levels main effect $p<0.05$
[b] Interaction between CPAP modes and CPAP levels $p<0.05$
[c] Effect of CPAP levels during $CPAP_H$ $p<0.05$

$\Delta EELV_{AB}$ ($p<0.01$, Table 1). There were no significant differences in $\Delta EELV_{AB}$ or $\Delta EELV_{RC}$ between $CPAP_H$ and $CPAP_M$ and no significant interaction between CPAP modes and either flow or CPAP levels (Table 1). ΔEELVs were smallest in the two patients with evidence of expiratory muscle activation (Fig. 2).

End-expiratory Pes (Pes_{ee}) increased significantly at increasing CPAP levels (ΔPes_{ee} was 3.7±1.2 cmH_2O, 6.9±1.7 cmH_2O and 9.9±2 cmH_2O, respectively, at CPAP 5, 10 and 15 cmH_2O, $p<0.001$), mirroring the increase in EELV (Fig. 3, Table 1). In the two patients with evidence of expiratory muscle activation, no significant

Table 3 Main effects of continuous positive airway pressure (CPAP) mode and gas flow rate on relevant variables

	Helmet			Mask		
Flow (l/min)	30	45	60	20	30	40
ΔPaw_i (cmH_2O)[c]	1.0±0.5	0.9±0.5	0.9±0.4	1.0±0.4	1.2±0.4	1.3±0.4
ΔPaw_e (cmH_2O)[a,b]	0.5±0.3	0.6±0.4	0.6±0.3	0.7±0.5	0.8±0.5	0.8±0.7
Vt (ml)*	492±171	495±160	435±135	579±169	584±202	552±184
$\%RIP_{RC}$	42.6±13.7	42.8±11.6	41.8±14	46.6±10.9	46.4±12.0	48.1±11.1
Ti (s)	1.59±0.40	1.83±0.88	1.48±0.38	1.81± 0.57	1.71±0.51	1.67±0.44
Te (s)	2.81±1.01	3.38±1.3	2.61±0.81	3.57±1.74	3.31±1.80	3.17±2.12
RR (bpm)[a]	14.8±4.2	13.0±4.7	15.7±4.4	13.1±5.5	14.4±6.2	15.0±5.9
Ve (l/min)	7.2±3.55	6.5±3.60	6.9±3.44	7.2±3.12	7.9±3.73	7.6±2.76
Vt/Ti (ml/s)	317±114	302±130	308±120	337±105	356±118	335±98
Vt/Te (ml/s)	201±119	175±111	189±110	196±106	221±129	211±93
$PetCO_2$ (mmHg)*	29.1±2.9	30.4±3.0	29.8±3.4	28.0±5.2	27.7±5.1	27.4±4.1
$PiCO_2$ (mmHg)*,[a]	4.1±0.6	2.8±0.5	2.5±0.6	1.0±0.9	0.6±0.6	0.7±0.9

Average values of main variables at different gas flow rates during $CPAP_H$ and $CPAP_M$
See text for abbreviations
*$CPAP_H$ vs $CPAP_M$ $p<0.05$

[a] Gas flow rate main effect $p<0.05$
[b] Effect of gas flow rate during $CPAP_H$ $p<0.05$
[c] Effect of gas flow rate during $CPAP_M$ $p<0.05$

correlation was found between ΔEELV and ΔPes_{ee}. In the other subjects (5), who showed no evidence of expiratory muscle activation, there was a significant correlation between ΔEELV and ΔPes_{ee} (average correlation coefficient 0.76±0.06, $p<0.001$).

Airway pressure inspiratory trough (ΔPaw_i) and expiratory peak (ΔPaw_e)

Both ΔPaw_i and ΔPaw_e were slightly lower (statistically not significant) during $CPAP_H$ (Tables 2 and 3). Both ΔPaw_i and ΔPaw_e increased at higher CPAP levels ($p<0.05$; Tables 2 and 3). The analysis of interactions showed that (1) the effect of CPAP levels on ΔPaw_i was significant only during $CPAP_H$ ($p<0.01$), (2) during $CPAP_M$ ΔPaw_i increased with increasing gas flow rates ($p<0.05$) while, during $CPAP_H$, ΔPaw_i decreased slightly but not significantly at higher gas flow rates and (3) the effect of CPAP level on ΔPaw_e was significant only during $CPAP_H$.

Respiratory pattern

Compared to $CPAP_H$, $CPAP_M$ resulted in a significantly higher Vt (572±40 ml vs 474±68 ml, $p<0.05$) and a slightly, but not significantly, lower RR (Tables 2 and 3). These resulted in a non-significant increase in Ve (7.58±1.25 l vs 6.86±1.19 l, $p<0.01$). The contribution of rib cage was not significantly different between the two CPAP modes. No significant differences between CPAP modes were found on Ti, Te or Vt/Te, while Vt/Ti showed non-significantly lower values during $CPAP_H$ ($p=0.085$).

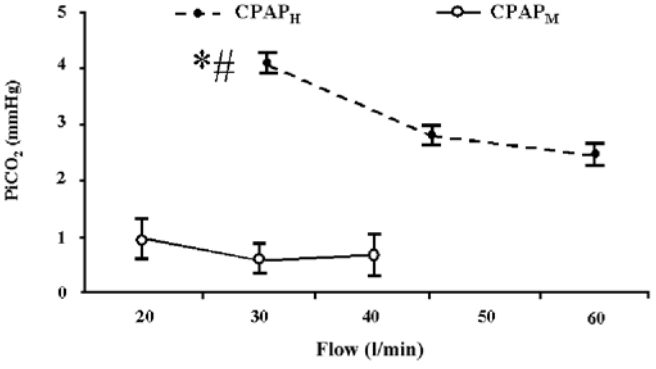

Fig. 4 Inspiratory levels of PCO_2 at different flows during continuous positive airway pressure with a helmet ($CPAP_H$, *closed circles*) and continuous positive airway pressure with a face mask ($CPAP_M$, *open circles*). *effect of gas flow rates during $CPAP_H$ $p<0.05$; # $CPAP_H$ versus $CPAP_M$ $p<0.05$

Carbon dioxide wash-out

The inspiratory CO_2 concentration was higher during $CPAP_H$ (3.1±0.15 mmHg) compared to $CPAP_M$ (0.8± 0.3 mmHg) at all flows and CPAP levels ($p<0.001$; Tables 2 and 3, Fig. 4). During $CPAP_H$ $PiCO_2$ significantly decreased at increasing gas flow rates ($p<0.001$) (Fig. 4).

Discussion

In this study on healthy subjects, we have shown that $CPAP_H$ is at least as effective as $CPAP_M$ in increasing EELV and in minimizing respiratory airway pressure oscillations, while avoiding the need of a reservoir bag.

However CO_2 may accumulate inside the helmet and higher gas flow rates are necessary to improve CO_2 wash-out and maintain low $PiCO_2$ levels.

We have used a different range of gas flow rates for $CPAP_H$ and $CPAP_M$. During $CPAP_M$ we used gas flow rates commonly used for a continuous flow CPAP system provided with a reservoir bag. During $CPAP_H$ we used higher gas flow rates to investigate the effect of flow on CO_2 accumulation in the helmet. Because of the important role that the flow levels can play in affecting the CPAP performance of both modes, the interactions between CPAP mode or CPAP levels and CPAP flows will not be considered and gas flow effects will be discussed separately for the two CPAP systems.

The increase in lung volume associated with the application of a positive airway pressure is the main physiological mechanism justifying the use of CPAP in patients with acute hypoxemic respiratory failure. In our study, the EELV changes induced by CPAP during $CPAP_H$ were comparable to those measured during $CPAP_M$. We used RIP to measure changes in EELV. Some authors have shown that, in spontaneously breathing subjects, measurements of EELV by RIP may be not sufficiently accurate (±10%) if compared to pneumotachograph measurements [28]. By repeatedly checking the RIP calibration to maintain the difference with the pneumotachograph smaller than 5%, we aimed at limiting possible errors in this study. Despite this possible limitation, the increase in Pes_{ee} with increased CPAP level and the high correlation between the RIP-measured ΔEELV and the ΔPes_{ee} support the reliability of our findings, at least in the subjects with no evidence of expiratory muscle activation. Since we lacked measurement of gastric pressure, the presence of expiratory muscle contraction was inferred by analysis of esophageal pressure and RIP signals [26, 27]. We found indirect evidence of expiratory muscle contraction in two subjects. These subjects obtained the first part of inspiratory lung inflation by expiratory muscle relaxation [26, 27].

An ideal CPAP system should maintain a constant airway pressure throughout the respiratory cycle [21]. To minimize Paw changes, continuous flow CPAP systems are best fitted with a reservoir bag along the inspiratory limb of the CPAP circuit [23]. We did not use a reservoir bag during $CPAP_H$, since we expected the helmet to work as a compliance reservoir itself. Inspiratory and expiratory pressure oscillations were comparable to those of an effective CPAP system [21, 23]. There were no statistical differences between the two systems despite slightly lower inspiratory as well as expiratory pressure oscillation during $CPAP_H$, which may be partially explained by the higher flow rates used with the helmet. The effect of CPAP level upon ΔPaw_i and ΔPaw_e differed between $CPAP_H$ and $CPAP_M$. This may be due to the fact that the bag of $CPAP_M$ and the helmet of $CPAP_H$ have different mechanical performances as reservoirs. Ideally a good reservoir bag should have a high compliance (400–700 l/cmH_2O) to face large volume changes with minimal pressure variations [23]. The helmet compliance, however, decreases sharply as pressure increases, explaining the lower performance at higher CPAP levels and the higher ΔPaw_i.

Tidal volume was significantly higher during $CPAP_M$. Though not significant, RR was lower and Ve higher with $CPAP_M$. These findings may be due to a psychological effect as well as to the increased dead space of the face mask, as shown by other studies which systematically investigated this issue [29].The $PetCO_2$ levels measured suggest that, though to a smaller extent, all subjects tended to hyperventilate during $CPAP_H$ as well.

As expected, CO_2 wash-out was critical during $CPAP_H$. This can, however, be improved by increasing the gas flow. Our contention is that this might be an advantage over $CPAP_M$. At 30 l/min flow rates, the average $PiCO_2$ was 4 mmHg (0.56%). In a subject ventilating at 6 l/min at 12 breaths/min, with a physiological dead space of 150 ml and a CO_2 production of 200 ml/min, Ve needs to increase only from 6 to 6.45 l/min to maintain a constant alveolar CO_2 pressure. By contrast, if, instead of the helmet, a mask dead space of 100 ml is added, the same subject will now need to increase Ve from 6 to 7.2 l/min to achieve a constant $PaCO_2$. Dead space volumes of commercially available face masks are in the order of 150–250 ml [30]. This may explain why, despite the higher $PiCO_2$, minute ventilation was lower, though not significantly so, during $CPAP_H$. The clinical relevance of CO_2 rebreathing with the helmet, as opposed to the additional mask dead space, is unknown and requires further studies.

Accumulation of CO_2 inside the helmet and rebreathing is a critical point of helmet application. Though, in terms of imposed work of breathing, an effective CPAP may be obtained with a helmet at gas flow rates similar to those used with a face mask, higher gas flows should always be considered to improve CO_2 wash-out and decrease CO_2 rebreathing.

We have shown in healthy volunteers that CPAP may be delivered effectively by a head helmet. Since this study was conducted in healthy subjects, its results may only partially apply to patients with respiratory distress and further studies are necessary to verify our results under pathological conditions. However, applications of the helmet in the clinical setting are encouraging [19, 20]. The helmet may have several advantages compared to other interfaces. It allows relatively free movement of the head while maintaining a good seal without compression on the face or head. In this study the helmet was effective in providing CPAP levels as high as 15 cmH_2O. The lack of pressure points on the face avoids the main complications associated with the use of a face mask: intolerance, pain and skin necrosis. In a recent randomized control trial on acute hypoxemic non-hypercapnic respiratory insufficiency by Delclaux et al. [10], CPAP delivered by a face

mask failed to show a reduction in the need for intubation and improvement in the outcome compared to standard oxygen therapy, despite early physiological improvement. In that study, mask intolerance was probably one of the major causes of the short duration of the CPAP treatment and may account, in part, for the CPAP failure. With the helmet, CPAP treatment may be applied continuously rather than intermittently, and this may be a determinant factor in the success of non-invasive CPAP.

Easy assembling and application allows the use of the helmet by personnel with a comparatively short specific training and it also has the advantage of a leak-proof system. Thus, it allows the use of CPAP outside intensive care units, in wards and in emergency rooms, and even by out-of-hospital emergency medical systems [19]. The possibility of early treatment may increase the efficacy of CPAP.

In conclusion, $CPAP_H$ provides physiological effects at least comparable to those provided by a mask. Higher gas flow rates should be used to improve CO_2 washout.

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