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Effects of non-invasive ventilation on middle ear function in healthy volunteers

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Abstract *Objectives:* To evaluate the effects of non-invasive ventilation (NIV) with facial mask or helmet on middle ear (ME). *Design:* Prospective, randomised study. *Setting:* University hospital. *Participants:* Ten healthy subjects randomly allocated in two groups of five subjects each. *Interventions:* NIV for 1 h, with helmet (group H) or facial mask (group M). Flow-triggered pressure support was 10 cmH_2O, PEEP 5 cmH_2O, FiO_2 0.21. *Measurements and results:* Impedenzometry was performed before NIV and 5 min after NIV ended; it was repeated 60 min later. In group H the acoustic compliance increased after NIV from 2.0±.6 ml to 2.3±.6 ml (P<.01), suggesting that the tympanic membrane became less stiff; 1 h later the compliance returned to basal values (2.0±.7 ml); in group M the compliance was unaffected (from 2.0±.5 ml to 2.0±.4 ml; 1.9±.4 ml 1 h later). The acoustic reflex, i.e., the contraction of the stapedial muscle in response to an auditory stimulus, involving the acoustic and facial nerves, was also evaluated during impedenzometry at 250 Hz, 500 Hz, 1,000 Hz, and 4,000 Hz; no significant change of the threshold was observed. *Conclusions:* The tympanic membrane is tighten by the tensor tympani and a reversible loosening suggests muscle fatigue in response to the application of intermittent positive pressure applied to the external ear during NIV with helmet. The loss of tensor tympani protective action could theoretically predispose the middle and inner ear to mechanical damage during NIV with helmet, suggesting the use of protective devices (ear plugs) in selective cases requiring long-term, high-pressure treatment.

Keywords Non-invasive ventilation · Helmet · Facial mask · Impedenzometry · Tympanometry · Acoustic reflex

Introduction

In recent years, non-invasive ventilation (NIV) has gained increasing popularity in the treatment of acute and chronic respiratory failure. A French multicenter survey found that NIV was applied in a percentage of patients varying from 14% to 50%, in relation to the cause of respiratory failure [1]. For many years, NIV has also been used successfully for long-term non-invasive ventilation in patients with musculoskeletal disorders [2]. More recently, NIV has been proved to be effective in hypercapnic acute respiratory failure due to chronic obstructive pulmonary disease (COPD) exacerbation [3, 4], hematologic malignancies [5], and solid organ transplantation [6, 7], including bilateral lung transplantation [8].

The availability of more comfortable patient-ventilator interfaces has contributed to this increasing popularity. Recently, a new device – the helmet – has been successfully employed in ICU patients affected by acute hypoxemic respiratory failure [9]. In that study no patient complained of ear disturbances; however, the whole head is exposed to positive pressure during NIV with a helmet and tympanic membranes are therefore challenged with intermittent overpressure. In this study, impedenzometry – a simple technique aimed to study middle ear (ME) functionality – was utilized to investigate the short-term effects of NIV with a facial mask or a helmet on ME in a group of healthy volunteers.

Material and methods

After obtaining the approval of the local Ethical Committee and written informed consent, ten healthy subjects, aged 24–48 years (seven males and three females), were included in the study and randomly divided into two groups, H (helmet) and M (facial mask). Criteria of exclusion were a medical history positive for ear diseases, any abnormal finding at otoscopy or at fiberoptic examination of upper airways, and the presence of a curve other than A at tympanometry (see below).

Each subject underwent continuous NIV for 1 h by a Servo Ventilator 300 (Siemens-Elema, Sweden) in flow-triggered pressure support (PS); FiO_2 was 0.21, PS was 10 cmH_2O, and PEEP was 5 cmH_2O. Such duration of application and level of pressure support were chosen: a) to prevent any risk of damage in healthy volunteers; and b) because intermittent 1-h NIV applications are often employed in clinical practice [7]. Group H was interfaced to the ventilator by a helmet (CaStar, Starmed, Italy); medium or large sizes were chosen according to the size of the subject. Group M was interfaced by a facial mask of appropriate size (Koo Med Equipment, China).

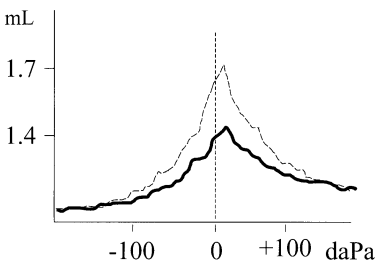

Fig. 1 Two "bell shaped" tympanograms (Jerger's type A) registered in a healthy subject included in this study and treated with NIV with helmet (before NIV: *black line*; after NIV: *dotted line*)

Impedenzometry was performed separately and sequentially in each ear by means of an impedance audiometer Q.I.Master (Quasar Medical, USA) before starting NIV and 5 min after disconnecting the subject from the ventilator. The examination was repeated 1 h later. Impedenzometry includes two measurements: tympanometry and acoustic reflex threshold assessment.

Tympanometry registers the acoustic impedance of ME, i.e., the amount of a probe sound reflected by the tympanic membrane, while the pressure in the external ear is varied from −200 daPa to +200 daPa. Conventionally, impedance audiometers supply acoustic compliance instead of acoustic impedance. Acoustic compliance, defined as the correspondent theoretical volume of the ME, is the reciprocal of impedance and corresponds to the fraction of the probe sound that is absorbed by the ME. In healthy subjects the relationship between the acoustic compliance and the pressure in the external ear corresponds to a bell-shaped curve (Fig. 1), classified as type A by Jerger [10]. Acoustic compliance is considered to be normal when the peak of the bell-shaped curve (which occurs when the pressure applied in the external ear canal equals the pressure in the ME) is obtained when the pressure applied in the external ear is 0 daPa. Abnormal shapes of tympanograms have been associated with several diseases of the ME. In general terms, the departure of maximal acoustic compliance from 0 unveils abnormal ME pressure (i.e., in Eustachian tube occlusion, maximal compliance shifts to negative pressure values reflecting ME depression), and abnormally high or low acoustic compliance values suggest reduced or increased stiffness of the tympanic membrane. However, acoustic compliance depends on ME size and shape so that a range of normality is not available.

The acoustic reflex is the contraction of the stapedial muscle in response to an auditory stimulus; it involves the acoustic and facial nerves. The pressure in the external ear is set at the value that corresponds to the maximal acoustic compliance and auditory stimuli of growing intensity are given. When the contraction of the stapedial muscle is elicited, a sudden change of acoustic compliance is registered due to the movement of the tympanic mem-

Table 1 Acoustic compliance values (ml) recorded before NIV (*b*) and 5 min and 60 min after NIV applied with helmet (*H*; five subjects) or mask (*M*; five subjects)

	H				M			
		b	5 min	60 min		b	5 min	60 min
	1 Right	1.4	1.7	1.2	6 Right	2.0	2.0	2.0
	Left	2.0	2.0	2.1	Left	2.8	2.6	2.8
	2 Right	2.4	2.5	2.2	7 Right	2.0	2.3	2.0
	Left	1.4	1.6	1.5	Left	2.1	2.3	2.1
	3 Right	2.8	3.3	3.3	8 Right	1.1	1.5	1.1
	Left	1.2	1.4	1.4	Left	1.8	1.5	1.8
	4 Right	2.2	2.6	2.8	9 Right	2.3	2.2	2.2
	Left	2.5	2.5	2.5	Left	1.8	2.0	1.9
	5 Right	1.3	1.9	1.2	10 Right	2.3	2.0	2.1
	Left	2.8	3.2	2.2	Left	1.5	1.6	1.5
Mean		2.0	2.3[a]	2.0		2.0	2.0	1.9
SD		0.6	0.6	0.7		0.5	0.4	0.4

[a] Comparison with basal values: *P*<.01

Table 2 Acoustic reflex thresholds at 250 Hz, 500 Hz, 1,000 Hz, and 4,000 Hz in group H (helmet) and M (mask) before and after non-invasive ventilation (NIV). Ten values were obtained from five patients since examination was performed bilaterally. In both groups thresholds were determined again 1 h after the end of NIV. Data are given as mean (standard deviation)

Group H (*n*=5)				
Hz	250	500	1,000	4,000
Base	90 (7)	85 (4)	93 (7)	90 (7)
5 min after NIV	95 (6)	93 (9)	95 (6)	93 (7)
60 min after NIV	89 (6)	88 (7)	94 (7)	91 (7)
Group M (*n*=5)				
Hz	250	500	1000	4000
Base	90 (7)	85 (10)	92 (7)	88 (9)
5 min after NIV	92 (9)	84 (13)	93 (7)	91 (9)
60 min after NIV	89 (6)	86 (9)	93 (6)	89 (7)

brane. The acoustic reflex threshold has been determined at 250 Hz, 500 Hz, 1,000 Hz, and 4,000 Hz; it is the weakest sound that elicits the stapedial reflex and is given as dB.

Each group of values were examined graphically and by Kolmogorov-Smirnov test for major deviations from normal distribution; if no major deviation was observed, means and standard deviations were reported. A *t*-test corrected according to Bonferroni was employed to compare values obtained after NIV to basal values. *P* values <.05 were considered as statistically significant.

Results

All the healthy volunteers included in this study presented normal findings at basal impedenzometry and tolerated NIV application with helmet or mask well. In particular, none of them complained of ear disturbances.

No significant departure from normal distribution was observed in the values taken into account. Acoustic compliance values are reported in Table 1. In group H the compliance increased from 2.0±.6 ml to 2.3±.6 ml after NIV ($P<0.01$), but returned to basal values 1 h later (2.0±.7 ml). By contrast, the compliance did not change after NIV in group M (from 2.0±.5 ml to 2.0±.4 ml; 1.9±.4 ml 1 h later). In both groups maximal acoustic compliance values remained near to 0 daPa. The acoustic reflex threshold at the frequencies tested is reported in Table 2. Slight, but statistically not significant, increases of the mean threshold values were observed in group H after NIV.

Discussion

The acoustic compliance of the ME measures the fraction of a probe sound that is absorbed by the tympanic membrane and the ossicular chain. The compliance is decreased by pathological conditions that make the system stiffer (such as otosclerosis) and is increased by conditions that make the system looser, for instance, by the disconnection of the tympanic membrane from the ossicular chain. In this study the acoustic compliance did not change following NIV applied with facial mask, but increased following NIV with helmet. Physiologically, the compliance is regulated by two ME muscles, the tensor tympani and the stapedial muscle. The increase in the compliance, which was transient since it returned to basal values in 1 h, could possibly originate from the exhaustion of ME muscles. These muscles regulate the stiffness of the tympanic membrane and contract in response to potentially dangerous auditory stimuli. According to the so-called "fixation theory", one of the purposes of the ME muscles is the stabilization of the ossicular chain against changes of the pressure in the external ear [11, 12]. The observed differences between helmet and facial mask could possibly originate from different effects on external and middle ear pressure. In addition, an influence of noise within the helmet cannot be ruled out, but this is possibly less important since several hours exposure to high-intensity noise are usually needed to induce ME alterations [13].

ME pressure is regulated by the Eustachian tube, which is functionally obstructed or collapsed at rest. When pressure is higher in the pharynx than in the ME, the tube opens spontaneously and pressures equalize. On the other hand, when pressure is higher in ME than in the pharynx, the tube opens spontaneously only at gradients higher than 25 cmH_2O or when the tube is opened by the muscular contraction of tensor veli palatini, levator palatini, or salpingopharyngeus during Valsalva or Toynbee manoeuvres (respectively, a forced expiration or swallowing with the mouth and the nose closed) [14]. During NIV with facial masks, positive pressure is directly applied to upper airways; air enters the ME during inspiration, but leaves it with more difficulty during expiration since the patient cannot perform a Valsalva or Toynbee manoeuvre. As a consequence, theoretically, during NIV with a facial mask the tympanic membrane could be constantly hyperinflated, a condition named hyperectasis [15]. At the time that the second impedenzometry was performed, 5 min after NIV cessation, no alteration was observed in tympanograms from group M. This finding could be explained because, as soon as NIV is stopped, ME overpressure (which should have been apparent on tympanograms) equilibrates with external pressure after a single swallowing effort.

When NIV is administered by a helmet, positive pressure is applied to the patient's whole head. Positive pressure is immediately transmitted to ME by the inward displacement of the tympanic membrane, while pharyngeal pressure probably grows more slowly because of the initial negative airway pressure that triggers pressure support. According to this hypothesis, for the entire duration of NIV application, the tympanic membrane is pushed inward during inspiration and returns to neutral position during expiration while the ME pressure oscillates from peak pressure to PEEP; pharyngeal pressure never exceeds ME pressure; and the Eustachian tube remains closed during the entire respiratory cycle. ME muscle fatigue can therefore result from muscle contraction in response to periodic external ear overpressure, in order to stabilize the ossicular chain.

The regulation of ME pressure is complex and many factors can influence the effects of NIV. Positional changes [16] and hypo- or hypercapnia [17] affect ME pressure by changing the size of the vessels or by varying gas reabsorption. In addition, the mastoid, which is air-filled, has an ME pressure buffering capability: the larger its volume, the better its buffering capability [18]. In this study NIV with helmets caused a limited increase of acoustic compliance and no significant depression of acoustic reflex, possibly because NIV was applied with relatively low inspiratory pressure and for a short time, or because buffering mechanisms, such as mastoid pneumatization, minimized the effects of NIV on ME pressure. Hypothetically, however, the periodic displacement of the tympanic membrane could involve the ossicular suspensory ligaments and the annular ligament of the footplate and damage the middle and internal ear, particularly if some protective mechanisms, such as the muscular contraction of tensor tympani, are depressed by fatigue.

In conclusion, when NIV is applied with a helmet, both external and middle ears are directly exposed to inspiratory positive pressure. The effects pointed out by this study are limited and probably of little clinical importance, but further studies are needed to investigate middle and inner ear function during longer NIV treatments with helmets, although a recent study did not report any clinically-apparent ear damage in patients that were treated with this technique [9]. Subjects with hearing deficits (e.g., elderly patients with stiffer ossicular chains) could be particularly at risk for ME alterations, and tympanic membrane and Eustachian tube function should probably be evaluated before applying NIV with helmet. In selected cases (long-term continuous application, high inspiratory pressure), the use of simple devices such as ear plugs could easily avoid external ear overpressure and some kind of muffler could usefully attenuate the noise within the helmet.

References

1. Carlucci A, Richard JC, Wysocki M, Lepage E, Brochard L (2001) Noninvasive versus conventional mechanical ventilation. An epidemiologic survey. Am J Respir Crit Care Med 163:874-880
2. Baydur A, Layne E, Aral H, Krishnareddy N, Topacio R, Frederick G, Bodden W (2000) Long-term non-invasive ventilation in the community for patients with musculoskeletal disorders: 46 years' experience and review. Thorax 55:4–11
3. Brochard L, Mancebo J, Wysocki M, Lofaso F, Conti G, Rauss A, Simonneau G, Benito S, Gasparetto A, Lemaire F, Isabey D, Harf A (1995) Noninvasive ventilation for acute exacerbations of chronic obstructive pulmonary disease. N Engl J Med 333:817–822
4. Plant PK, Owen JL, Elliott MW (2000) Early use of non-invasive ventilation for acute exacerbations of chronic obstructive pulmonary disease on general respiratory wards: a multicentre randomised controlled trial. Lancet 355:1931–1935
5. Conti G, Marino P, Cogliati A, Dell'Utri D, Lappa A, Rosa G, Gasparetto A (1998) Noninvasive ventilation for the treatment of acute respiratory failure in patients with hematologic malignancies: a pilot study. Intensive Care Med 24:1283–1288
6. Antonelli M, Conti G, Bufi M, Costa MG, Lappa A, Rocco M, Gasparetto A, Meduri GU (2000) Noninvasive ventilation for treatment of acute respiratory failure in patients undergoing solid organ transplantation: a randomized trial. JAMA 283:235–241
7. Hilbert G, Gruson D, Vargas F, Valentino R, Gbikpi-Benissan G, Dupon M, Reiffers J, Cardinaud JP (2001) Noninvasive ventilation in immunosuppressed patients with pulmonary infiltrates, fever, and acute respiratory failure. N Engl J Med 344:481–487
8. Rocco M, Conti G, Antonelli M, Bufi M, Costa MG, Alampi D, Ruberto F, Stazi GV, Pietropaoli P (2001) Noninvasive pressure support ventilation in patients with acute respiratory failure after bilateral lung transplantation. Intensive Care Med 27:1622–1626
9. Antonelli M, Conti G, Pelosi P, Gregoretti C, Pennisi MA, Costa R, Severgnini P, Chiaranda M, Proietti R (2002) New treatment of acute hypoxemic respiratory failure: noninvasive pressure support ventilation delivered by helmet. A pilot controlled trial. Crit Care Med 30:602–608
10. Jerger J (1970) Clinical experience with impedance audiometry. Arch Otolaryngol 92:311–324
11. Marquet J (1985) Stapedotomy technique and results. Am J Otolaryng 6:63–67
12. Huttenbrink KB (1988) The mechanics of the middle-ear at static air pressures: the role of the ossicular joints, the function of the middle-ear muscles, and the behaviour of stapedial prostheses. Acta Otolaryngol Suppl 451:1–35
13. Borg E, Nilsson R, Lidèn G (1982) Fatigability of the stapedial reflex in industrial noise. Acta Otolaryngol 94:385–393
14. Uzun C, Adali MK, Tas A, Koten M, Karasalihoglu AR, Devren M (2000) Use of the nine-step inflation/deflation test as a predictor of middle ear barotrauma in sports scuba divers. Br J Audiol 34:153–163
15. Sade J (2001) Hyperectasis:the hyperinflated tympanic membrane: the middle ear as an actively controlled system. Otol Neurotol 22:133–139
16. Gaihede M, Kjaer D (1998) Positional changes and stabilization of middle ear pressure. Auris Nasus Larynx 25:255–259
17. Ikarashi F, Takahashi S, Yamamoto Y (1999) Carbon dioxide exchange via the mucosa in healthy middle ear. Arch Otolaryngol Head Neck Surg 125:975–978
18. Elam M, Harell M, Luntz M, Fuchs C, Sade J (1998) Middle ear pressure variations during 50% N2O anesthesia as a function of mastoid pneumatization. Am J Otol 19:709–711