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**EUHA**

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# Efficacy of continuous positive airway pressure on middle ear atelectasis: A double-blind placebo-controlled clinical trial

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## ABSTRACT

**Objective:** To investigate the effects of continuous positive airway pressure (CPAP) treatment on patients with middle ear atelectasis.

**Study design:** Prospective, double-blind, randomised, placebo-controlled study.

**Methods:** Fifty four patients with middle ear atelectasis were randomised to receive CPAP treatment with a pressure level of either 14 cm H<sub>2</sub>O (CPAP Group) or 0 cmH<sub>2</sub>O (Placebo Group) once per week for a period of three hours for four sessions. Outcome measures included otomicroscopic examination, tympanometric and audiometric evaluation. Patients were followed for six months.

**Results:** The CPAP Group included 35 atelectatic ears and the Placebo Group included 32 atelectatic ears. More ears recovered to normal tympanic membrane or regressed to Grade 1 atelectasis in the CPAP Group than in the Placebo Group during all follow-up visits ( $p < .05$ ). There was a statistically significant increase in the middle ear pressure values of the patients in the CPAP Group compared to the Placebo Group at Week 5, Month 3, and Month 6 ( $p < .05$ ). There was no significant difference in middle ear pressure values between follow-up visits in the CPAP Group ( $p > .05$ ). Significant improvement of pure-tone air-conduction threshold averages were found in the CPAP Group compared to the Placebo Group at Month 6 ( $p < .05$ ).

**Conclusion:** CPAP is a safe, well-tolerated way of applying positive pressure to the middle ear for patients with middle ear atelectasis. It contributes to significant improvement in middle ear pressure of these patients, also resulting in an improved degree of atelectasis.

**Key-words:** Continuous positive airway pressure; CPAP; middle ear atelectasis; middle ear pressure; tympanometry; audiometry.

**Level of evidence:** Level 1b

## INTRODUCTION

Middle ear atelectasis is a condition in which the normal contour and elasticity of the tympanic membrane are lost. Research suggests that it results from persistent negative middle ear pressure caused by Eustachian tube (ET) dysfunction and imbalance of absorption and secretion by mastoid and middle ear mucosa<sup>1,2</sup>. It predisposes the patient to adhesive otitis, tympanic membrane atrophy, cholesteatoma formation, and ossicular erosion.

Establishing normal middle ear ventilation and aeration is the main goal in order to prevent retraction of the atelectatic tympanic membrane. Many alternatives are available to manage a patient with middle ear atelectasis including medical and surgical alternatives. Medical management includes decongestants, nasal steroids, antihistamines, Valsalva's manoeuvre, and nasal balloon inflation<sup>3,4</sup>. Surgical alternatives are ventilation tube insertion, cartilage tympanoplasty with or without prophylactic tube insertion, and laser tympanoplasty<sup>5-8</sup>. In spite of this range of alternatives, middle ear atelectasis still remains a difficult middle ear pathology to treat.

Continuous positive airway pressure (CPAP) has recently been used in the standard treatment protocol of obstructive sleep apnoea syndrome (OSAS) patients. It prevents the collapse of the upper airway with pressurised air during sleep. The effects of CPAP on middle ear pressure have recently been a subject of research<sup>9-11</sup>. It was demonstrated that positive airway pressure directed to the nasopharynx with CPAP was delivered to the middle ear and increased middle ear pressure<sup>9-11</sup>. Yung suggested that CPAP could be used to reinflate the retracted tympanic membrane in middle ear atelectasis<sup>9</sup>. In his study, more than two thirds of the 30 atelectatic tympanic membranes were found to be reinflated immediately after a single treatment session of CPAP<sup>9</sup>. We conducted this prospective study to assess the effects of longer CPAP treatment on mild to moderate, but not severe, middle ear atelectasis and also to document six months of follow-up results.

## MATERIALS AND METHODS:

### Patients and study design

Institutional Review Board approval was obtained from our institution before the study commenced. Patients who had been diagnosed with middle ear atelectasis between November 2012 and December 2014 were asked to participate in this double-blind, randomised, placebo-controlled study. The patients were informed about the study and their signed written consents were obtained. Exclusion criteria included age below 18 years, presence of snoring or daytime sleepiness and an Epworth Sleepiness Scale score higher than 5, allergic or vasomotor rhinitis, sinonasal, or nasopharyngeal pathology, and planned air flights or diving during the following six months. Finally, patients who had cholesteatoma or accumulation of keratin debris on tympanic membrane, and those with Grade 4 atelectasis according to Sade's classification were also excluded<sup>12</sup>.

Patients were randomly assigned to two groups according to the CPAP pressure level applied at a 1:1 ratio. All the patients and investigators remained blinded to the randomisation throughout the entire study. The CPAP pressure applied was set to 14 cm H<sub>2</sub>O in the CPAP Group or 0 cm H<sub>2</sub>O in the Placebo Group. CPAP application was conducted by an experienced technician in the hospital's sleep laboratory. Nasal masks were selected according to the patient's comfort and choice. Each patient came to the hospital's sleep laboratory once per week on the same day of four consecutive weeks. CPAP was applied in the laboratory for a period of three hours under the surveillance of a sleep technician. Patients were awake in an upright sitting position during the use of CPAP. Drinking, eating, and chewing gum were not permitted, while swallowing with usual pattern was allowed. Any discomfort during the use of CPAP was noted.

### Outcome measures

Outcome measures included sequential otomicroscopic examination and tympanometric and audiometric evaluation. For each patient, the study consisted of a baseline assessment and three follow-up visits at Week 5 (i.e., one week after the fourth CPAP session), Month 3, and Month 6. Otomicroscopic examination for the presence and grading of atelectasis and tympanometric measurement were done at the baseline and at each follow-up visit. Audiometric evaluation was performed at the beginning and at the Month 6 visit.

Pure tone audiometry was performed in a sound-proof booth using pure tones (250 ms duration, 25 ms rise/fall time, 50% duty cycle) at octave frequencies from 125 Hz to 8000 Hz with a maximum intensity of 120 dB SPL with a two-channel AC40 clinical audiometer (Interacoustics, Assens, Denmark). Air conduction thresholds were measured under headphones at 125 Hz, 250 Hz, 500 Hz, 1 kHz, 2 kHz, 4 kHz, 6 kHz, and 8 kHz. Bone conduction thresholds were measured on 250 Hz, 500 Hz, 1 kHz, 2 kHz, 3 kHz, and 4 kHz. Hearing results were analysed according to the Guidelines of the Committee on Hear-

ing and Equilibrium of the American Academy of Otolaryngology-Head and Neck Surgery<sup>13</sup>. Accordingly, we calculated the mean, standard deviation (SD), and range of the air-conduction thresholds, bone-conduction thresholds and air-bone gaps. Thresholds at the frequencies 500 Hz, 1 kHz, 2 kHz, and 3 kHz were used for calculation. The air-bone gap was calculated as the air-conduction pure-tone average minus the bone conduction pure-tone average.

The tympanometric measurements were performed after swallowing using a 220 Hz probe tone with an Impedance Audiometer AZ 7 (Interacoustics, Assens, Denmark). Tympanograms were classified according to Campbell in types A, B and C<sup>14</sup>. When the compliance peak was between +50 daPa and -100 daPa, the tympanogram was considered as type A; when the peak was between -100 daPa and -350 daPa as type C; and when there was no peak, the curve was interpreted as type B (1 daPa = 1.02 mm H<sub>2</sub>O)<sup>14</sup>.

### Statistical analysis

The statistical data was conducted using IBM SPSS Statistics (IBM Co., NY, USA). Metric data are presented as means  $\pm$  SD. Descriptive statistics were calculated for numeric variables. Analysis of the results was performed by the Student's paired t test, the ANOVA, the Mann Whitney U, and the Kruskal Wallis test. A *p* value < .05 was considered to be statistically significant.

## RESULTS

During the study period, 54 patients diagnosed with middle ear atelectasis agreed to participate in this study. Three patients who could not follow the weekly sequential CPAP schedule and one patient who had an unplanned air flight during the study course were dropped from the study. Three other patients who missed more than one follow-up visit were also excluded from the study.

The remaining 47 patients' data were enrolled and analysed. The CPAP Group included 24 patients with 35 atelectatic ears (including eleven patients with bilateral atelectasis), and the Placebo Group included 23 patients with 32 atelectatic ears (including nine patients with bilateral atelectasis). The baseline characteristics of the study population are presented in Table 1. The ages ranged between 20 and 63 years; the mean age in the CPAP Group was  $37.87 \pm 10.83$ , whereas it was  $36.03 \pm 11.19$  in the Placebo Group ( $p > .05$ ). The degree of atelectasis, hearing levels, and mean middle ear pressure values showed that both groups of this study were statistically identical ( $p > .05$ ).

	CPAP group (n=35)	Placebo group (n=32)	p value
Age (yr; mean $\pm$ SD) (median)	37.87 $\pm$ 10.83 (39)	36.03 $\pm$ 11.19 (33)	0.251
Gender (Male/female) (n <sup>o</sup> )	10/14	12/11	0.552
Degree of atelectasis <sup>a</sup> , n (%)	35	32	
Grade 1	15 (42.8%)	14 (43.8%)	0.923 923.000
Grade 2	12 (34.2%)	11 (34.4%)	
Grade 3	8 (22.8%)	7 (21.8%)	
Tympanometric pressure (daPa) (mean $\pm$ SD; min, max; median)	-284.97 $\pm$ 138.96 (-400, -32) (-400)	-262.65 $\pm$ 133.54 (-400, -48) (-285)	0.508
Audiometry			
Air-conduction pure-tone average (dB) (mean $\pm$ SD; min-max; median)	28.54 $\pm$ 12.36 (12-63) (26)	29.06 $\pm$ 13.56 (10-60) (24,5)	0.870
Air-bone gap (dB) (mean $\pm$ SD, min-max; median)	12.9 $\pm$ 7.14 (2, -30) (12)	11.9 $\pm$ 5.35 (3, -27) (11)	0.496
Word recognition score (%) (median)	90 (90)	92.12 (91)	0.596
<sup>a</sup> According to Sade's classification of 0 to 4 degrees SD indicates standard deviation; CPAP, continuous positive airway pressure; n, number of ears. <sup>o</sup> This n indicates number of patients.			

Table 1: Baseline demographic and clinical data of the study groups.

In the CPAP Group, one patient had an earache during all the CPAP sessions, and one other patient suffered from mild claustrophobia. But they both were able to complete the CPAP therapy. The rest of the patients in both groups tolerated the CPAP device well.

Otomicroscopic examination revealed normal tympanic membrane (Grade 0 atelectasis) in some ears in the CPAP Group during follow-up visits: Grade 0 atelectasis was shown to be in five ears at Week 5, in nine ears at Month 3, and in seven ears at Month 6. The distribution of the degree of atelectasis in the groups is presented in Table 2. The number of

Degree of atelectasis <sup>a</sup>	Initial		Week 5		Month 3		Month 6	
	CPAP group (n=35)	Placebo group (n=32)	CPAP group (n=35)	Placebo group (n=32)	CPAP group (n=32)	Placebo group (n=26)	CPAP group (n=32)	Placebo group (n=32)
Grade 0, n	0	0	5	0	9	0	7	2
Grade 1, n	15	14	20	14	17	10	15	7
Grade 2, n	12	11	6	9	5	8	7	14
Grade 3, n	8	7	3	6	1	7	2	8
Grade 4, n	0	0	1	3	0	1	1	1

<sup>a</sup> According to Sade's classification of 0 to 4 degrees  
n indicates number of ears.

Table 2: Distribution of degree of atelectasis in the study groups.

ears with Grade 0 and Grade 1 atelectasis was higher in the CPAP Group than the Placebo Group during all follow-up visits. The difference between the groups based on degree of atelectasis was statistically significant at Week 5, Month 3, and Month 6 ( $p < .05$ , Figure 1).

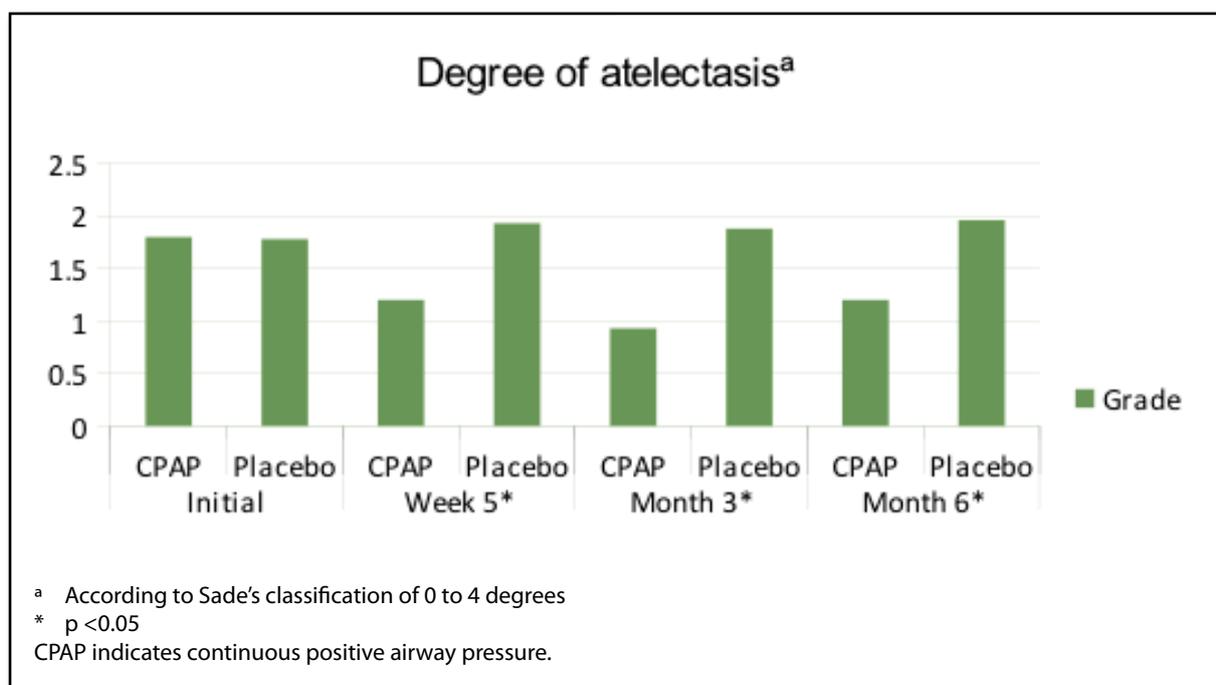


Figure 1: Change in the mean degree of atelectasis in study groups.

In the CPAP Group, the mean grade of atelectasis was the lowest at the follow-up visit at Month 3. This was significantly lower than the mean grade of atelectasis at Week 5 ( $p < .05$ ), but not significantly different from the mean grade of atelectasis at Month 6 ( $p > .05$ ).

The change in otomicroscopic evaluations of the patients during follow-up is presented according to group in Table 3. For the CPAP Group, 12 of the total of 24 patients (eight patients with unilateral and four patients with bilateral atelectasis) at Week 5; 14 of 22 patients (eight patients with unilateral and six patients with bilateral atelectasis) at Month 3; and eleven of 22 patients (seven patients with unilateral and four patients with bilateral

Comparison to the initial evaluation <sup>a</sup>	Week 5		Month 3		Month 6	
	CPAP group (n=24)	Placebo group (n=23)	CPAP group (n=22)	Placebo group (n=18)	CPAP group (n=22)	Placebo group (n=32)
Better, n	12	0	14	0	11	2
Same, n	9	19	5	12	5	10
One side better, other side same, n	2	1	2	1	5	9
Worse <sup>o</sup> , n	1	3	1	5	1	2

<sup>a</sup> According to Sade's classification  
<sup>n</sup> indicates number of patients.  
<sup>o</sup> For bilateral atelectatic ears, "worse" is accepted as higher in degree of atelectasis in at least one of the ears.

Table 3: Change in degree of atelectasis in the study groups.

atelectasis) at Month 6 demonstrated a better degree of atelectasis compared to the initial evaluation. Nine patients (five patients with unilateral and four patients with bilateral atelectasis) kept the same degree of atelectasis at Week 5 compared to the initial evaluation. At Month 3, five patients (four patients with unilateral and one patient with bilateral atelectasis) and at Month 6, five patients (all of them with unilateral atelectasis) demonstrated the same degree of atelectasis compared to the initial evaluation.

In the CPAP Group, none of the patients with unilateral atelectasis progressed to a worse (i.e. higher) degree of atelectasis compared to the initial evaluation during all follow-up visits. One patient with bilateral Grade 3 atelectasis progressed to a Grade 4 atelectasis on the left side and kept the Grade 3 atelectasis on the other side at Week 5. This patient missed the follow-up at Month 3, but at Month 6, the patient kept the same degrees of atelectasis as at Week 5. Another patient of the CPAP Group with bilateral atelectasis (right ear with Grade 1 atelectasis, left ear with Grade 3 atelectasis) kept the same degrees of atelectasis at Week 5, but demonstrated Grade 2 atelectasis on both ears at Month 3; in other words, there was a regression of degree of atelectasis on the left side, and a progression to a higher degree of atelectasis on the right side at Month 3. This patient had Grade 1 atelectasis on the right side and Grade 2 atelectasis on the left side at Month 6.

In the Placebo Group, no patient regressed to a lower degree of atelectasis compared to the initial evaluation at Week 5 and Month 3 (Table 3). Two patients with unilateral Grade 1 atelectasis regressed to normal tympanic membrane at Month 6. At Week 5, 19 of total 23 patients (13 patients with unilateral and six patients with bilateral atelectasis); at Month 3, twelve of 18 patients (nine patients with unilateral and three patients with bilateral atelectasis); and at Month 6, ten of 23 patients (seven patients with unilateral and three patients with bilateral atelectasis) kept the same degree of atelectasis compared to the initial evaluation. Progression to a worse (i.e. higher) degree of atelectasis during the follow-up visits was high in the Placebo Group. Three patients (one patient with unilateral and two patients with bilateral atelectasis) at Week 5; five patients (one patient with unilateral and four patients with bilateral atelectasis) at Month 3; and nine patients (five

patients with unilateral and four patients with bilateral atelectasis) at Month 6 progressed to a higher degree of atelectasis – for bilateral atelectasis, progression in at least one of the ears – compared to the initial evaluation.

The mean middle ear peak (MEP) pressure of the patients in the CPAP Group increased during follow-up period (Table 4). This increase was statistically significant at Week 5, Month 3 and Month 6 compared to the initial evaluation ( $p < .05$ ). Although reduced compared to normal ears, the highest mean MEP pressure was observed at Month 3 in the CPAP Group. However, there was no significant difference in the mean MEP pressure in this group of the patients between Week 5, Month 3, and Month 6 ( $p > .05$ ). The mean MEP pressure of the patients was significantly higher in the CPAP Group than in the Placebo Group during all follow-up visits ( $p < .05$ , Table 4). The difference between the groups based on mean MEP pressure of the patients was statistically significant at Week 5 ( $p < .02$ ) and statistically highly significant at Month 3 and Month 6 ( $p < .01$ ).

Typanometric pressure mean±SD (daPa)		CPAP group	Placebo group	<i>p</i> value
Initial	n	35	32	
	mean±SD (median) (daPa)	-273.34±149 (-400)	-252.18±141.98 (-260)	0.508
Week 5	n	35	32	
	mean±SD (median) (daPa)	-170.80±130.5 (-180)	-280.15±123.19 (-297.5)	0.002*
Month 3	n	32	26	
	mean±SD (median) (daPa)	-143.15±136.97 (-88.5)	-299.07±129.06 (-370)	0.001*
Month 6	n	32	32	
	mean±SD (median) (daPa)	-165.9±150.72 (-106.5)	-320.15±126.0 (-400)	0.001*

\*  $p < 0.05$   
SD indicates standard deviation; CPAP, continuous positive airway pressure; n, number of ears.

Table 4: Evaluation of tympanometric pressure in the study groups.

The results of audiometric evaluation in the groups are presented in Table 5. Significant improvement of pure-tone air-conduction threshold averages was reported in the CPAP Group compared to the Placebo Group at Month 6 ( $p < .05$ ). However, there was no significant difference between the two groups regarding the air-bone gap average and the mean word recognition scores at Month 6 ( $p > .05$ ). The average air-conduction threshold gain was 6.7 dB in the CPAP Group at Month 6. There was no air-conduction threshold gain in the Placebo Group. In fact, the mean pure-tone air-conduction threshold average was worse in the Placebo Group at Month 6 compared to the initial evaluation.

		Pure-tone average <sup>o</sup> (dB)		Air-bone gap average (dB)		WRS (%)	
		Initial	Month 6	Initial	Month 6	Initial	Month 6
CPAP group	mean±SD (median) (daPa),	28.54 ±12.36 (26)	21.8 ± 6.92 (22)	12.9± 7.14 (12)	9.32± 5.22 (8)	90±3.5 (90)	92.33± 5.48 (91)
Placebo group	mean±SD (median) (daPa)	29.06 ±13.56 (24,5)	33.67± 14.9 (30)	11.9± 5.35 (11)	11.22± 5.25 (10)	92.12± 3.8 (91)	90.56± 5.33 (90)
<i>p</i> value		0.870	0.001*	0.496	0.158	0.596	0.202
<sup>o</sup> Pure-tone air conduction threshold average; * <i>p</i> < 0.05 SD indicates standard deviation; CPAP, continuous positive airway pressure; WRS, word recognition scores.							

Table 5: Results of audiometric evaluation in the study groups.

## DISCUSSION

Patients who use CPAP experience an increased level of pressure in their nasopharynx and middle ear. This study has demonstrated a significant increase in the middle ear peak pressure of patients with middle ear atelectasis who had four weekly CPAP sessions. In contrast, there was no change in the middle ear pressure of the patients in the Placebo Group.

The increase of middle ear pressure in the CPAP Group is most likely through the Eustachian tube (ET). The ET functions to regulate the pressure and ventilation of the middle ear. It stays closed during rest and opens actively during swallowing, chewing and yawning. The tensor veli palatini muscle opens the collapsed part of the ET leading to an area that has relatively lower pressure than the middle ear and nasopharynx. Air flows into this lower-pressure ET area mainly from the nasopharynx, as the proximal end of the ET lumen dilates first and is then followed by dilation of the distal end. Following this active dilation, ET then collapses passively to return to its resting position which leads the air to travel into the middle ear, thereby equalising gas pressure between the middle ear and atmosphere<sup>15</sup>.

It has been shown that middle ear pressure reaches suprathysiologic levels during CPAP use in normal subjects at even minimal CPAP settings<sup>10</sup>. It has also been demonstrated that increases in middle ear pressures directly correlate with rising pressure levels of a CPAP device<sup>10,11</sup>. Middle ear pressures of OSAS patients were found to have increased after CPAP use for six months<sup>11</sup>. Patients using CPAP at a higher pressure level tended to have a larger middle ear pressure change. The increase in middle ear pressure of the patients using CPAP at 12 to 14 cm H<sub>2</sub>O level was found to be higher than that in patients using CPAP at the 8 to 10 cm H<sub>2</sub>O pressure level<sup>11</sup>. In our study, the CPAP pressure level chosen for the CPAP Group (14 cm H<sub>2</sub>O) is based on these results.

Yung studied the short-term effects of single CPAP use on 30 atelectatic and 30 normal middle ears<sup>9</sup>. In his study CPAP was applied with a pressure level of 10 cm H<sub>2</sub>O for three hours. Examination of tympanic membranes and pure-tone audiometric evaluations were performed immediately after the use of CPAP in atelectatic middle ears; unfortunately, grading of the degree of atelectasis could not be done. It was stated that a total 22 of 30 atelectatic tympanic membranes were reinflated immediately after the CPAP use. But long-term results were not given for all of the patients. Seven patients were in an early postoperative period of a tympanoplasty. Six of these postoperative atelectatic tympanic membranes were reinflated, and they preserved middle ear ventilation for six months as well. The middle ear pressures of atelectatic tympanic membranes were not measured in his study<sup>9</sup>. We want to emphasise that our study is the first reported study to present the effects of CPAP treatment on middle ear pressure of patients with middle ear atelectasis. Our results are promising for CPAP use on middle ear atelectasis, but further research appears warranted to confirm them.

Sivri et al. stated that eleven of twelve ears which revealed opaque tympanic membrane with absence of light reflex and five of six slightly retracted tympanic membranes recovered to normal tympanic membrane after six months of CPAP use. Besides that, four of five retraction pockets in tympanic membranes were totally recovered, and one retraction pocket had regressed from stage 3 to stage 2 in their study<sup>11</sup>.

In our study, more ears recovered to normal tympanic membrane or regressed to a lower degree of atelectasis in the CPAP Group than in the Placebo Group during all follow-up periods. The distribution of degree of atelectasis in both groups was found to be significantly different. In the CPAP Group, the number of ears with normal tympanic membrane, and correspondingly the mean grade of atelectasis, were the highest at Month 3. Supporting this, the mean middle ear pressure was also measured to be the highest at Month 3 in the CPAP Group.

The average air-conduction threshold gain was 6.7 dB in the CPAP Group at Month 6 in our study. But the air-bone gap averages and the word recognition scores did not significantly differ between the groups at Month 6. It is possible that hearing gain and other audiometric parameters would have been different if audiometric evaluation had also been done at Month 3. They may possibly have been better as the grade of atelectasis and middle ear pressure were best at Month 3 in the CPAP Group.

In middle ear atelectasis, there is a risk that the atelectatic membrane may adhere to the incudostapedial joint and the promontory, which can lead to ossicular resorption, accumulation of squamous debris, and development of cholesteatoma. To prevent retraction of the atelectatic membrane, equalisation of the negative middle ear pressure is recommended. This may be achieved by performing Valsalva's manoeuvre, nasal balloon inflation, or by inserting a ventilation tube into the tympanic membrane<sup>3,4</sup>. Inserting ventilation tubes in an atelectatic tympanic membrane could sometimes be problematic because of the poor height of the middle ear cavity. Unsuccessful attempts of ventilation tube insertion could bring increased possibility of permanent perforation in an atelectatic tympanic membrane. There are no adequate studies that show hearing improvement from ventilation tube insertion for middle ear atelectasis<sup>8</sup>. Surgical treatment is usually recommended for Grade 3 or Grade 4 atelectasis. Otologists tend to perform cartilage tympanoplasty with/without prophylactic tube insertion at the time of surgery<sup>5-7</sup>. Recurrent atelectasis is not uncommon after cartilage tympanoplasty. It has been reported that more than 50% of completely retracted tympanic membranes eventually re-collapsed after a cartilage tympanoplasty<sup>16</sup>. Laser myringoplasty is recently presented as a minimally invasive technique for certain atelectatic tympanic membranes<sup>8</sup>. However, all these surgical procedures have associated risks and morbidity.

The side effects mostly seen with CPAP use are related to the upper respiratory tract, most often nasal congestion, dryness of mouth and nose, epistaxis, skin irritation, and discom-

fort owing to the mask<sup>17,18</sup>. These side effects are generally observed with long-term use of CPAP. In our study, otalgia occurred in one patient and mild claustrophobia due to the mask occurred in another patient. Vertigo and nystagmus due to CPAP use is very rare. Yung reported mild vertigo in two patients with single CPAP use<sup>9</sup>. In contrast, none of the patients in our study experienced any vertigo. CPAP is a safe, well-tolerated way of applying positive pressure to the middle ear. It can be self-administered and it can be used for relatively long periods. Future studies would be worthwhile to investigate the effects of CPAP use on middle ear atelectasis with different regimes.

One of the limitations of our study is the small sample size. Further studies including more patients would be beneficial to support our results. CPAP can be an alternative way of establishing normal middle ear ventilation through the Eustachian tube. However, as Yung stated in his paper, "It is just another way of trying to put more air into the middle ear"<sup>9</sup>. It should be kept in mind that gas exchange through the Eustachian tube is not the only mechanism for regulation of middle ear ventilation. Gas diffusion through the middle ear mucosa and physiologic properties of mastoid air cell system are as important as the Eustachian tube<sup>19</sup>. In our study, patients were not evaluated for these parameters; further research including these may be warranted.

## CONCLUSION

This study demonstrated a significant increase in the middle ear pressure of patients with middle ear atelectasis who had four weekly CPAP sessions. In contrast, no change was observed in the middle ear pressure of the patients in the Placebo Group. Although CPAP does not correct the underlying problem of middle ear atelectasis or Eustachian tube dysfunction, our data suggest that it contributes to significant improvement in middle ear pressure of patients with middle ear atelectasis. Our study also showed that more ears recover to normal tympanic membrane or regress to a less severe atelectasis of the tympanic membrane in the CPAP Group than in the Placebo Group during six months of follow-up. At 6 Months, the CPAP Group showed a statistically significant increase when compared to the Placebo Group in average pure-tone air conduction thresholds, but the average air-bone gap was not significantly different between the groups.

CPAP is a safe, well-tolerated way of applying positive pressure to the middle ear in middle ear atelectasis. Further research is needed to confirm our results and also to determine the clinical significance of CPAP use in the treatment of middle ear atelectasis.

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