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- Nonsurgical home treatment of middle ear effusion and associated hearing loss in children. Part I: Clinical trial
- Nonsurgical home treatment of middle ear effusion and associated hearing loss in children. Part II: Validation study

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Nonsurgical home treatment of middle ear effusion and associated hearing loss in children. Part I: Clinical trial

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Abstract

We conducted a randomized, controlled clinical trial to investigate the efficacy of treatment of persistent middle ear effusion (MEE) and associated hearing loss with a modified Politzer device used in the home setting over a 7-week period. Efficacy was determined by comparing pre- and posttherapy air-conduction thresholds, tympanometric peak pressures, and otoscopic findings. The study group was made up of 94 children (174 ears), aged 4 to 11 years, who had at least a 2-month history of MEE and associated hearing loss. At study's end, patients in the treatment group experienced statistically significant improvements in all measured outcomes; no significant improvements were seen in the control group in all measured outcomes. At study's end, the hearing sensitivity of 73.9% of the treated ears was within normal limits, compared with only 26.7% of the control ears. These findings demonstrate that home treatment of children with persistent MEE and associated hearing loss with the modified Politzer device is highly efficacious.

Introduction

Methods of managing middle ear effusion (MEE) have included (1) periodic observation during efforts to reduce environmental risk factors, (2) decongestants, (3) antihistamines, (4) corticosteroid therapy, (5) antimicrobial agents, (6) myringotomy with or without placement of tympanostomy tubes, (7) adenoidectomy with or without tonsillectomy, either alone or in combination with myringotomy with or without placement of tympanostomy tubes, and (8) insufflation of the eustachian tube/middle ear system by the Valsalva maneuver or the Politzer method.

The efficacy of treatment with drugs or surgery has been questioned. For example, Cantekin et al concluded that decongestant and antihistamine treatment was not beneficial in the management of MEE.[1] In a meta-analysis of 33 studies, Williams et al concluded that the beneficial effect of antimicrobial treatment is minor and only short-term.[2]

If medication fails, most otolaryngologists resort to surgical placement of tympanostomy tubes.[3] One survey showed that 40% of otolaryngologists believe that tubes are used too frequently.[4,5] There are several arguments against the surgical treatment of MEE with tympanostomy tubes:

- MEE resolves spontaneously in 80 to 90% of children within 3 months.[6,7]
- A significantly negative tympanometric peak pressure (TPP) may persist after surgical treatment with tympanostomy tubes.[8]
- Ventilation tubes frequently fall out prematurely, often in 4 to 7 months.[9,10] In such cases, 40% of patients experience a recurrence of MEE,[3] and 33 to 75% undergo repeat surgery to replace them.[10,11]
- The risk of one or more episodes of otorrhea following tube placement is three times higher than the risk following simple myringotomy.
- Patent tubes allow bacteria, viruses, and allergens to migrate into the middle ear cavity.[12]
- Other reported complications of tympanostomy tube placement include (1) tympanic membrane retraction, (2) postsurgical infection, (3) localized foreign-body reaction, (4) granulation, (5) hyalinization, (6) tympanosclerosis, (7) temporary or permanent hearing impairment of varying

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degrees, (8) persistent tympanic membrane perforation, (9) dislocation of the tube into the middle ear cavity, (10) tube blockage, and (11) cholesteatoma.[6,13,14]

A clinical practice guideline published in 2004 advises that antibiotics and decongestants are ineffective medical approaches to the treatment of MEE.[7] Likewise, antimicrobials, with or without corticosteroids, are not recommended for the routine treatment of MEE because they do not provide long-term benefits, although they may confer some short-term benefits. This guideline recommends that children who have MEE that has persisted for 4 months or longer and who have persistent hearing loss or other signs and symptoms should be considered for surgical insertion of tympanostomy tubes.

Insufflation of the eustachian tube/middle ear system involves forcing air under pressure through the eustachian tube (retrograde approach) and into the middle ear. The assumption underlying this approach is that frequent repetition of this procedure over a short period of time (on the order of days) can result in the normalization of negative middle ear pressure and the elimination of MEE. The two most common methods of insufflation are the Valsalva maneuver and the Politzer method. The Valsalva maneuver involves performing forced nasal expiration with the nose and lips closed. The Politzer method involves inserting the tip of a rubber air bulb into a patient's nostril, simultaneously compressing the other nostril with a finger, and having the patient swallow as the rubber bulb is compressed.

Valsalva maneuver. Cantekin et al evaluated the Valsalva maneuver in 66 children between 2 and 6 years of age who had recurrent or chronic otitis media and functioning tympanostomy tubes.[15] None of these children was able to open the eustachian tube with the Valsalva maneuver. The failure of the Valsalva maneuver was attributed to excessive tubal compliance in this age group.

One modification of the Valsalva maneuver involves inserting a balloon into one nostril while compressing the other nostril.[16] When the maneuver is successful, the balloon is inflated. Blanshard et al evaluated the balloon modification of the Valsalva maneuver in 85 children, aged 3 to 10 years, who had bilateral MEE and who were candidates for surgical placement of tympanostomy tubes.[17] Approximately one-half of these children were treated with autoinflation (experimental group), and the remainder were assigned to a control group. Patients in the experimental group achieved a beneficial effect after 2 to 4 weeks of autoinflation. The disadvantages of autoinflation in this study included difficulty in performing the procedure, as 43% of the experimental group performed it irregularly and 12% were unable to perform it at all.

Because of its limitations, the Valsalva maneuver does not hold promise as a treatment for otitis media with effusion and related conditions.

Politzer method. The Politzer method (politzerization) was patterned after an inflation technique described by Shea.[18] Schwartz et al modified the Politzer method by forcing air through the nostril with a 1-oz infant nasal syringe equipped with a plastic tip that was inserted into a nostril.[19] In a controlled study, they measured the effectiveness of politzerization by performing tympanometry 5 and 10 minutes after politzerization in 24 adults and children with MEE and associated tympanic membrane retraction. In the active-treatment group, the mean shift in TPP following politzerization was –9 mm H_2O.

An important advantage that the Politzer method has over the Valsalva maneuver is that the forced air is initiated by an external source. The limitations of the Politzer apparatus and modifications thereof include (1) their cumbersome design, (2) a failure to produce continuous and nonfluctuant air-pressure flow, (3) a failure to coordinate the air-pressure stream with swallows, and (4) an absence of air-pressure and air-flow volume controls, which might lead to the generation of either harmful or ineffective air pressures into the middle ear via the nose. Politzerization has generally not been well accepted by patients, and it traditionally has not been suitable for use in the home setting. These limitations are attributable to the design of the currently existing Politzer devices rather than to any facet of the method itself.

An optimum insufflation device would (1) be portable, (2) be operable by a patient or parent, (3) allow for coordination of the air-pressure stream with swallows, and (4) be equipped with air-pressure and air-volume flow controls so that air pressures introduced into the middle ear through the nose are sufficient yet not harmful.[20,21]

We designed a modified Politzer autoinsufflation device that overcomes the aforementioned limitations of extant devices (figure 1).[20,21] Our handheld, battery-operated device emits controlled air pressure and flow that can be adjusted according to the patient's age and magnitude of hearing loss.[20,21] We conducted two earlier studies of treatment with a device very similar to this and found that approximately 75% of patients with MEE and associated hearing loss recovered following treatment.[20,21] However, a major limitation of these studies was that patients were required to come to a physician's office for treatment 2 or 3 times a week over a period of 6 to 7 weeks. This routine placed a burden on patients in terms of convenience and direct and indirect costs.

In an attempt to obviate the disadvantages of office-based politzerization, we conducted a National Institutes of Health–supported, randomized, controlled study of the feasibility of using our modified Politzer device to treat persistent MEE and associated hearing loss in the home

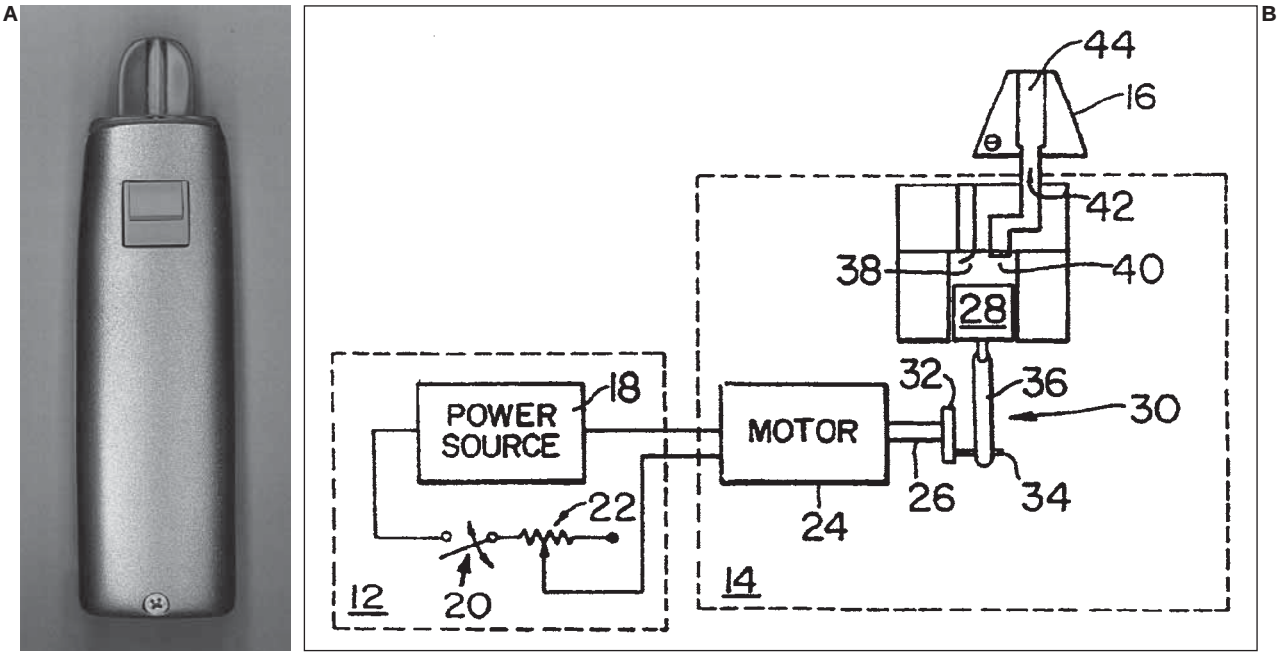


Figure 1. ***A:*** *Photograph shows the external appearance of our modified Politzer device.* ***B:*** *Schematic depicts the inner components of the apparatus used in this study. The "activation means" (12) includes a power source (18), switch (20), and a "power variation means" (22). The compressor (14) is activated by the activation means. The compressor's components include a motor (24) that turns a motor shaft (26). The shaft is connected to a piston (28) by a rotating disk (32), pin (34), pivoting linkage (30), and arm (36). The arm pivotally drives the piston as a result of the rotary motion of the shaft and disk. Oscillation of the piston affects the operation of two flutter valves (38 and 40). Deflection of the flutter valves creates air flow through an exit port (42). The exit port communicates with a channel (44) in a nostril plug (16).* Source: Silman S, Arick D. Efficacy of a modified politzer apparatus in management of eustachian tube dysfunction in adults. J Am Acad Audiol 1999;10:496-501.

setting. Efficacy was determined by comparing changes in pretherapy (pretest) and posttherapy (posttest) air-conduction thresholds, TPPs, and otoscopic findings.

Patients and methods

Instrumentation. Our modified Politzer device was very similar to the one that we used in our previous two studies.[20,21] The federal Food and Drug Administration has classified our modified apparatus as a 510(k) prescriptive medical device. The device emits a controlled air pressure and volume velocity sufficient to effect improvement (based on the results of our feasibility study) without discomfort. The device has two settings. Setting 1 delivers an air pressure of 5.2 psi at a volume velocity of 1,524 ml/min; setting 2 delivers an air pressure of 2.5 psi at a volume velocity of 1,690 ml/min (figure 2).

Patients. More than 600 children were referred to the Center for Auditory Research at Brooklyn (N.Y.) College for possible inclusion in the study. Suitable candidates were those who had furnished informed consent and who satisfied six study criteria: (1) age 4 to 11 years, (2) at least a 2-month history of MEE and associated hearing loss as documented by a physician, (3) pure-tone air-conduction thresholds of 20 dB HL or more at 3 frequencies between 500 and 4,000 Hz with air-bone gaps of 15 dB or more at these frequencies *or* pure-tone air-conduction thresholds of 25 dB HL or more at 2 frequencies between 500 and 4,000 Hz with air-bone gaps of 15 dB or more at these frequencies at the final pretest, (4) a TPP of –100 daPa or less at the final pretest, (5) an otologic diagnosis of MEE at the final pretest, and (6) an absence of enlarged adenoids, acute otitis media, and other ear abnormalities at the final pretest otologic examination.

Procedures. Treatment was initiated on the day of the final pretest. Each patient's parent administered the treatment twice daily—in the morning before breakfast and again in the evening after supper. During each of these two sessions, treatment was administered to one nostril and then to the other, and then the procedure was repeated approximately 10 minutes later. During each treatment, the patient was instructed to be in a sitting position. To deliver therapy, the parent inserted a pediatric probe tip (coupled to the device) into one nostril while compressing the other nostril with a finger. Each participant had his or her own device. The parent was given alcohol-soaked wipes and instructed to clean the tip before each use. The child then

held a small amount of water in the mouth without swallowing it. The parent then turned on the device, thereby introducing air flow into the nostril at a constant volume velocity. After 1 or 2 seconds of air flow, the parent asked the child to swallow the water.

Parents were provided with a daily log to foster and track compliance. Most patients were seen in the office for monitoring at least once near the midpoint of the treatment period, which lasted 7 weeks.

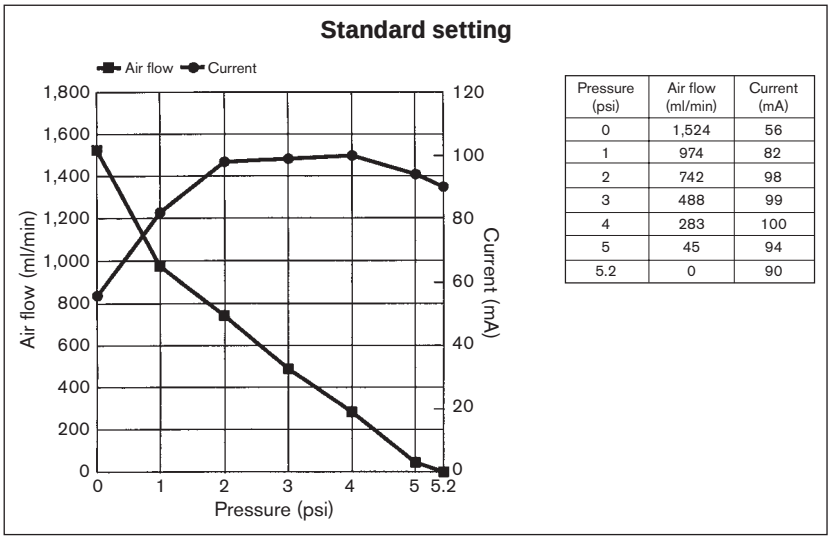
Initially, the air pressure was set to 5.2 psi. As the study progressed, a small proportion of the children (~3 to 4%) in the experimental group, particularly those whose hearing had been improving markedly, began to experience a slight discomfort during sensations of ear popping at this setting; none had experienced any discomfort at this setting at the beginning of the study. The discomfort appeared to reflect a decrease in the fluid level of the middle ear. In response, we modified the device to incorporate a second air-pressure setting of 2.5 psi. Lowering the psi from 5.2 to 2.5 resolved the discomfort. This circumstance forced us to modify our protocol so that the device was set at 2.5 psi for younger children (≤7 yr) during the first week and increased to 5.2 psi (if tolerated) for the remainder of the study. For older children (>7 yr), the device was set to 5.2 psi throughout the study. In both of these age groups, the setting was lowered to 2.5 psi if any participant experienced discomfort.

Audiometry was performed by audiologists certified by the American Speech-Language-Hearing Association and licensed by New York State. Otologic evaluations were performed by board-certified otolaryngologists with at least 15 years of experience. Audiologic evaluations (air- and bone-conduction threshold testing and tympanometry) and otolaryngologic evaluations (otoscopy) were performed upon enrollment in the study and 4 weeks after the completion of the treatment (i.e., 11 wk following study entry). However, parents were advised to discontinue treatment if a child developed a cold or upper respiratory infection and to complete the treatment after the cold or upper respiratory infection resolved. As a result, a small number of children were seen later than 11 weeks after study entry.

The patients in the control group did not undergo sham treatment with a dummy device because their parents would have immediately recognized it as such. These parents were instructed to contact the investigators immediately if they noticed any worsening of hearing status.

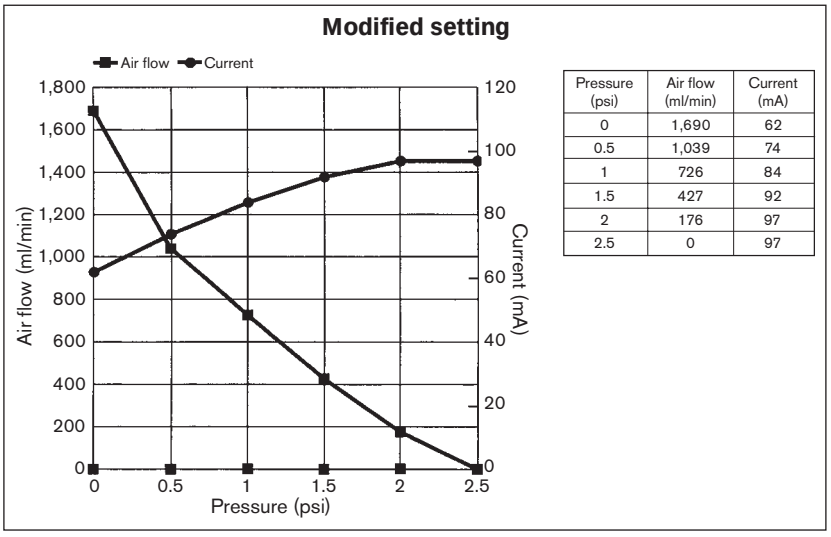
Audiologists were blinded to each patient's otologic findings, and otolaryngologists were blinded to each patient's audiometric findings. At the posttest, audiologists and otolaryngologists were blinded to each patient's disease status. The statistician was blinded as to whether test results were obtained before or after therapy and to the disease status of each patient.

A



Pressure (psi)	Air flow (ml/min)	Current (mA)
0	1,524	56
1	974	82
2	742	98
3	488	99
4	283	100
5	45	94
5.2	0	90

B



Pressure (psi)	Air flow (ml/min)	Current (mA)
0	1,690	62
0.5	1,039	74
1	726	84
1.5	427	92
2	176	97
2.5	0	97

Figure 2. ***A:*** *Graph illustrates the response characteristics of the device (5.2 psi) used in this study. The relation between volume velocity (air flow) and pressure (psi) is shown.* ***B:*** *Graph illustrates the response characteristics of an additional setting (2.5 psi) that was added to the device during the second half of the study. The relation between volume velocity (air flow) and pressure (psi) is shown.*

Sample size determination was made using power formulae for Student's *t* tests.[22] To achieve a power of 90% or more for each of our hypotheses, the sample size to be recruited was 110 (55 for each group, a number calculated to include a possible 10% attrition rate). An alpha level of 0.05 was used for all statistical tests. For statistical analysis, an absent TPP was coded as –450 daPa.

All patients in the control group who had not spontaneously recovered by the posttest were given the option of receiving treatment with the study device upon the conclusion of their involvement in the study. The outcomes of these controls will be presented in another report.

Results

A total of 94 children (174 ears), aged 4 to 11 years, met the inclusion criteria and were entered into the study. Of this group, 47 patients (88 ears) were randomly assigned to the experimental group and 47 patients (86 ears) were assigned to the control group.

Compliance. Complete compliance with the treatment protocol was demonstrated by 46 of the 47 experimental patients (97.9%), based on inspection of their daily logs and our assessment of their familiarity with the device; in the remaining patient, compliance was judged to be moderate. Our device appeared to be easy to use at home and was well tolerated by patients and parents.

Air-conduction thresholds and TPPs. For each ear, the mean pretest air-conduction thresholds for the experimental and control groups were similar—that is, within 3.0 dB at 500 Hz, 5.1 dB at 1,000 Hz, 4.2 dB at 2,000 Hz, and 1.4 dB at 4,000 Hz (table 1). At the pretest, the mean 4-frequency pure-tone average (PTA) in the experimental group was 29.6 in the right ear and 32.6 dB HL in the left ear; in the control group, the mean for both ears was 29.3 dB HL. Overall, the mean PTAs in both ears in both groups were symmetrical to within 3.3 dB.

The mean pretest TPPs in both ears in both groups were symmetrical to within 40.3 daPa (table 1). All posttest mean air-conduction thresholds in the experimental group were less than 20 dB HL. By contrast, in the control group, all but 1 posttest mean air-conduction threshold were higher than 20 dB HL. The exception was seen at 2,000 Hz in the left ear, where the mean threshold was 17.6 dB HL; in the right ear at that frequency, the mean (20.7) was only slightly higher than 20 dB HL.

Comparisons of the pre- and posttest measures for each ear in each group were obtained using paired Student's *t* tests. In the experimental group, these comparisons revealed statistically significant improvements ($p < 0.001$) in mean air-conduction thresholds across the entire frequency range in both ears (table 2). No statistically significant improvements in mean air-conduction thresholds occurred at any frequency or in either ear in the control group ($p > 0.05$). Likewise, the experimental group experienced statistically significant improvements ($p < 0.001$) in mean TPPs in both ears, while no such improvements were seen in the control group ($p > 0.05$).

Comparisons of the differences in scores (posttest

Table 1. Mean (standard deviation) air-conduction thresholds (dB HL) and TPPs (daPa)

Group	Test	Ear	n	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz	TPP
Experimental	Pre	Right	43	33.0	32.1	23.8	29.4	–300.9
				(10.9)	(10.1)	(11.0)	(12.1)	(147.7)
		Left	45	35.3	37.7	26.0	31.4	–334.8
				(11.4)	(10.8)	(12.2)	(11.7)	(135.2)
Control	Pre	Right	45	32.7	32.4	21.2	30.8	–320.7
				(7.8)	(9.3)	(10.7)	(11.2)	(139.0)
		Left	41	32.3	32.6	21.8	30.4	–294.5
				(8.3)	(12.0)	(11.5)	(13.8)	(169.5)
Experimental	Post	Right	43	18.3	15.5	7.9	12.0	–188.7
				(7.6)	(8.4)	(7.9)	(9.8)	(131.3)
		Left	45	19.0	17.3	8.7	14.7	–178.1
				(10.3)	(11.0)	(8.2)	(8.1)	(136.7)
Control	Post	Right	45	29.7	30.3	20.7	28.1	–283.9
				(11.7)	(13.2)	(13.9)	(14.9)	(154.3)
		Left	41	30.1	29.0	17.6	26.3	–318.9
				(10.8)	(13.8)	(12.5)	(14.6)	(133.7)

Table 2. Mean differences (confidence intervals) and significance levels (*p*) between pre- and posttest air-conduction thresholds (dB HL) and TPPs (daPa)*

Group	Ear	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz	TPP
Experimental	Right	–14.8	–16.6	–15.9	–17.4	112.2
		(–18.1, –11.4)	(–20.2, –13.1)	(–19.9, –12.0)	(–21.6, –13.3)	(71.1, 153.3)
		$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$
	Left	–16.3	–18.3	–17.3	–16.8	156.7
		(–19.7, –13.0)	(–21.5, –15.2)	(–20.7, –13.9)	(–20.4, –13.1)	(105.2, 208.1)
		$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$
Control	Right	–3.1	–2.3	–0.9	–3.1	36.7
		(–7.1, 0.9)	(–6.6, 2.0)	(–5.1, 3.4)	(–8.1, 1.9)	(–17.4, 90.8)
		$p = 0.12$	$p = 0.28$	$p = 0.68$	$p = 0.22$	$p = 0.18$
	Left	–2.2	–3.5	–4.3	–4.0	–24.4
		(–5.8, 1.4)	(–7.4, 0.3)	(–8.8, 0.3)	(–9.7, 1.6)	(–90.3, 41.5)
		$p = 0.22$	$p = 0.07$	$p = 0.06$	$p = 0.16$	$p = 0.46$

** Negative mean values indicate improvement at the posttest.*

minus pretest) for each ear between the two groups were obtained using independent sample Student's *t* tests (assuming equal variances). These comparisons revealed statistically significant differences ($p < 0.001$) between the two groups in the difference scores throughout all frequencies in both ears, indicating that all mean improvements in air-conduction thresholds from pre- to posttest in the experimental group were significantly greater than those for the control group (table 3). Similarly, the mean posttest improvement in TPP in the right ear in the experimental group was significantly greater ($p < 0.03$) than that in the right ear in the control group. In the left ear, the contrast was even more striking ($p < 0.001$), as the results reflected not merely an improvement in mean TPP in the left ear in the experimental group, but an actual deterioration in mean TPP in the left ear in the control group. Although the observed difference in change in the mean TPPs from pre- to posttest between the two groups was greater in the left ear (181.1 daPa) than the right ear (75.5 daPa), there is no reason to conclude that the true effect is different for the two ears. However, these values are only point estimates of the differences in mean change scores, and the confidence intervals overlap substantially. Therefore, the apparent discrepancy between the results in the right and left ears is not statistically significant.

Hearing sensitivity. Overall, hearing sensitivity returned to within normal limits in at least one ear in 40 of the 47 patients (85.1%) in the experimental group, compared with only 15 of the 47 patients (31.9%) in the control group (table 4). In terms of the number of ears, recovery was seen in 65 of 88 experimental ears (73.9%) and 23 of 86 control ears (26.7%).

Table 3. Means (confidence intervals) and significance levels (*p*) for the difference between groups (experimental minus control) in the difference (posttest minus pretest) in air-conduction thresholds (dB HL) and TPPs (daPa)

Ear	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz	TPP
Right	–11.7	–14.3	–15	–14.3	75.5
	(–16.8, –6.5)	(–19.8, –8.8)	(–20.8, –9.3)	(–20.8, –7.9)	(8.1, 142.9)
	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$
Left	–14.1	–14.8	–13.1	–12.8	181.1
	(–19.0, –9.3)	(–19.7, –9.9)	(–18.6, –7.5)	(–19.3, –6.2)	(99.5, 262.6)
	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$	$p < 0.001$

At the pretest, bilateral hearing loss was present in 41 experimental patients; at the posttest, hearing had been restored to within normal limits in at least one ear in 37 patients (90.2%). Of these 37 patients, 25 (67.6%) achieved normal hearing in both ears and 12 (32.4%) in one ear. In the control group, 39 patients had a bilateral hearing loss at the pretest, and 12 (30.8%) achieved restoration of hearing. Of these 12 controls, 8 (66.7%) regained hearing in both ears and 4 (33.3%) regained hearing in one ear.

If we define a significant hearing loss for this age group as a PTA of 35 dB HL or more, 38 of the 88 experimental ears (43.2%) and 41 of the 86 control ears (47.7%) demonstrated such a hearing loss at the pretest. At the posttest, only 3 of the 38 experimental ears (7.9%) still had a PTA of 35 dB HL or more, compared with 36 of the 41 control ears (87.8 %).

Otoscopic findings. We examined the relationship between hearing sensitivity in the 65 experimental ears that had achieved normal hearing at the posttest and findings on pneumatic otoscopy. Normal tympanic membrane mobility was seen in 49 of these ears (75.4%), moderate mobility in 12 (18.5%), and no mobility in 4 (6.2%). Thus, either normal or moderate mobility was observed in 61 of the 65 ears (93.8%). This finding further substantiates the efficacy of our device for improving middle ear function and hearing sensitivity in children with MEE. The absence of tympanic membrane mobility in 4 normal ears suggests that an immobile eardrum is not necessarily associated with hearing loss.

Follow-up. Upon completion of participation in the study, all parents were advised to notify us if they or others noticed any change in hearing status. The parents of 6 children who had improved following active treatment did contact us. One child developed MEE and hearing loss after 3 months; his hearing sensitivity was restored to within normal limits by an additional 2 weeks of treatment. Another parent called after 7 months, and we found that the child had developed recurrent MEE and hearing loss; this child's hearing also returned after an additional 2 weeks of treatment. Another parent called after 9 months, but her child's hearing sensitivity was found to be normal.

Calls from 3 other parents—2 of which came more than 1 year after treatment—indicated that tympanostomy tubes had been placed in 2 children. One of these children had already received 2 sets of tubes prior to enrollment in our study. Following the study, the child's hearing sensitivity and otologic status, which had been monitored by an otologist, had remained within normal limits for 1 year. Subsequently, however, the child experienced a recurrence of MEE and associated hearing loss, and a third set of tubes was inserted. Nevertheless, MEE with hearing loss recurred again 3 months later. The patient underwent a repeat course of home treatment with our device, and his hearing began to improve. Two children were diagnosed with enlarged adenoids; 1 underwent an adenoidectomy without tympanostomy tube placement and his hearing returned to normal, while the other underwent an adenoidectomy with insertion of tympanostomy tubes.

We also placed telephone calls to 10 randomly selected parents of children in the experimental group. These calls were made 12 to 18 months following the completion of the child's participation in the study. All of these parents reported satisfaction with the outcome of the treatment.

Table 4. Number (%) of experimental patients and controls whose posttest hearing returned to within normal limits according to the type of pretest hearing loss (bilateral or unilateral)

	Experimental group	Control group
Bilateral hearing loss at pretest		
Normal ears posttest	62/82 (75.6)	20/78 (25.6)
Normal patients posttest	37/41 (90.2)	12/39 (30.8)
Unilateral hearing loss at pretest		
Normal ears posttest	3/6 (50.0)	3/8 (37.5)
Normal patients posttest	3/6 (50.0)	3/8 (37.5)
Total		
Normal ears posttest	65/88 (73.9)	23/86 (26.7)
Normal patients posttest	40/47 (85.1)	15/47 (31.9)

Discussion

The results of our investigation indicate that twice-daily home treatment of persistent MEE and associated hearing impairment over a period of 7 weeks with a modified Politzer device that controls air flow and air pressure is highly efficacious in children aged 4 to 11 years. All mean posttest air-conduction thresholds and TPPs were markedly better than the mean pretest values in the experimental group but not in the control group; the pre- and posttest differences in the experimental group were statistically significant ($p < 0.001$). Also, the differences between groups in the mean change from pre- to posttest were significant for all air-conduction and TPP measures, and these improvements were markedly greater in the experimental group.

The Agency for Health Care Policy and Research's Otitis Media Guidelines Panel recommends that candidacy for surgical placement of tympanostomy tubes in children with persistent MEE should be based on the presence of a hear-

ing loss.[7] In our study, hearing impairment was present in all ears in both groups at the pretest. Hearing sensitivity was judged to be within normal limits if the air-conduction thresholds were less than 20 dB HL at any of 3 frequencies *or* less than 25 dB HL at either of 2 frequencies.

Although posttest hearing sensitivity did not improve to within normal limits in either ear in 7 of the 47 experimental patients (14.9%), reports by parents, teachers, and patients themselves indicated that these patients' listening and attention behaviors suggested that some degree of improvement in hearing status had occurred during treatment. It is possible that their hearing sensitivity actually did improve during treatment only to subsequently deteriorate because of a recurrence of MEE prior to the posttest; the posttest was not performed until 4 weeks after the completion of treatment. In a pilot study that we conducted prior to this investigation, audiologic evaluations were performed immediately before and after treatment, and we observed immediate improvement in hearing sensitivity in all patients after each administration. These pilot data support our hypothesis that the 7 treatment failures were attributable to the lag between the end of treatment and the posttest. The audiologic, otologic, and demographic characteristics of the 7 experimental patients whose hearing was not restored were no different from the data on those whose hearing did recover to within normal limits. We also considered the possibility that the duration of treatment was insufficient for these 7 patients, and we offered them a 2- to 3-week extension. The results of the extended treatment will be reported in a separate article.

In conclusion, the results of our study indicate that the use of our modified, automated Politzer device for the home treatment of MEE in children aged 4 to 11 years was highly successful. Marked improvement was reflected by evaluations of pure-tone air-conduction thresholds, TPPs, and tympanic membrane mobility at the posttest 4 weeks following the completion of treatment. Future research is needed to generalize findings to children younger than 4 years, to teenagers, and to adults. Further study is also needed to explore long-term outcomes. Still, our anecdotal observations suggest that there is indeed a long-term benefit.

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Nonsurgical home treatment of middle ear effusion and associated hearing loss in children. Part II: Validation study

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Abstract

In this prospective follow-up investigation, we examined the efficacy of a modified Politzer device in the home treatment of persistent middle ear effusion (MEE) and associated hearing loss in children who had previously participated in a similar clinical trial. Our study group was made up of 38 patients who had been either (1) untreated control participants in the previous study whose hearing in one or both ears had not returned to normal within 11 weeks of their initial audiologic pretest ("former control group"; n = 30), or (2) active-treatment participants in the previous study whose hearing sensitivity in at least one ear had not improved to within normal limits after treatment and who elected to undergo a continuation of treatment ("extended-treatment group"; n = 8). Treatment efficacy was determined by comparing differences in pre- and posttreatment air-conduction thresholds and otoscopic findings. Following treatment, the former control group experienced significant improvements in hearing sensitivity at all frequencies; at the posttreatment test, hearing sensitivity was within normal limits in 43 of 60 ears (71.7%), and normal or moderate tympanic membrane mobility was observed in 30 of 34 otoscopically examined ears (88.2%). In the extended-treatment group, hearing sensitivity returned to within normal limits in 9 of 10 impaired ears (90.0%). These findings further substantiate the efficacy of our modified Politzer device in improving middle ear function and hearing sensitivity in children with MEE, and they support the reliability of the findings reported in our previous study. These results also indicate that many patients in whom initial treatment is not successful may benefit from extended treatment.

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Introduction

We recently reported the results of a randomized controlled clinical trial of a modified Politzer device for the home treatment of persistent middle ear effusion (MEE) and associated hearing loss.[1] Our handheld, battery-operated device emits controlled air pressure and air flow that can be adjusted in accordance with the degree of hearing loss and the patient's age.

In our previous study, we found that twice-daily home treatment over a period of 7 weeks was highly efficacious in restoring hearing in children between the ages of 4 and 11 years who had MEE. At the posttreatment audiologic test, hearing sensitivity had returned to normal in 65 of 88 treated ears (73.9%) and in at least one ear in 40 of 47 treated patients (85.1%); the corresponding figures for the control group were only 23 of 86 (26.7%) and 15 of 47 (31.9%). We also reported that pneumatic otoscopy revealed normal or moderate tympanic membrane mobility in 61 of 65 successfully treated ears (93.8%).[1]

At the conclusion of our previous study, we offered the controls whose hearing had not recovered the opportunity to undergo active treatment. We also offered extended treatment to those patients in the active-treatment group who had not completely improved. In this article, we report the findings of our prospective follow-up study.

Patients and methods

Instrumentation. The design and function of our modified Politzer device are described in the report of our previ

ous study.[1] The device emits a controlled air pressure and volume velocity sufficient to effect improvement without discomfort. The device has two settings. Setting number 1 delivers an air pressure of 5.2 psi at a volume velocity of 1,524 ml/min; setting 2 delivers an air pressure of 2.5 psi at a volume velocity of 1,690 ml/min. The device was very similar to one that we had used in two other studies reported in 1999[2] and 2000.[3]

Patients. Our study population was made up of patients who had furnished informed consent and who satisfied six study criteria: (1) age 4 to 11 years, (2) at least a 2-month history of MEE and associated hearing loss as documented by a physician, (3) pure-tone air-conduction thresholds of 20 dB HL or more at 3 frequencies between 500 and 4,000 Hz with air-bone gaps of 15 dB or more at these frequencies *or* pure-tone air-conduction thresholds of 25 dB HL or more at 2 frequencies between 500 and 4,000 Hz with air-bone gaps of 15 dB or more at these frequencies at the final pretest, (4) a tympanometric peak pressure of –100 daPa or less at the final pretest, (5) an otologic diagnosis of MEE at the final pretest, and (6) an absence of enlarged adenoids, acute otitis media, and other ear abnormalities at the final pretest otologic examination.

Former control group. At the completion of our previous study, 32 of the 47 controls had not achieved normal hearing in at least one ear. Of these 32 patients, 30 accepted our offer to undergo active treatment.

Extended-treatment group. Eight patients in the active-treatment group of our previous study who had not achieved recovery of hearing in one or both ears accepted our offer to continue treatment in the current study.

Procedures. The procedures used in this study were the same as those followed in our previous report, except that we did not include any untreated controls. All patients in this follow-up study received active treatment.

Each patient's parent administered treatment in the morning before breakfast and again in the evening after supper. Each participant had his or her own device. The parent was given alcohol-soaked wipes and instructed to clean the tip of the pediatric probe, which was coupled to the device, before each use. During each treatment, the patient was instructed to be in a sitting position. To deliver therapy, the parent inserted the tip into one nostril while compressing the other nostril with a finger (figure). The child then held a small amount of water in the mouth without swallowing it. The parent then turned on the device, thereby introducing air flow into the nostril at a constant volume velocity. After 1 or 2 seconds of air flow, the parent asked the child to swallow the water. Immediately thereafter, the same therapy was administered to the other nostril. Approximately 10 minutes later, the procedure was repeated in both nostrils.

The planned duration of treatment for the former control group was 7 weeks. The patients in the extended-treatment group (who had already received 7 weeks of treatment) were scheduled to undergo an additional 2 weeks of treatment. Two weeks after the completion of extended treatment, they underwent an audiologic assessment. Those whose hearing was not within normal limits in one or both ears were given another 2 weeks of treatment and assessed again 2 weeks after its completion. This treatment-and-assessment cycle continued until either hearing sensitivity was restored to within normal limits or no further improvement in hearing sensitivity was observed.

Figure. The parent inserts the tip of the modified Politzer device into one nostril while she compresses the other nostril with her finger.

Parents were provided with a daily log to foster and track compliance. Most patients were seen in the office for monitoring at least once near the midpoint of the treatment period. Parents were advised to discontinue treatment if the child developed a head cold or infection and to resume treatment only after obtaining clearance from a study otologist or the child's pediatrician.

Audiometry was performed by audiologists certified by the American Speech-Language-Hearing Association and licensed by New York State. Otologic evaluations were performed by board-certified otolaryngologists with at least 15 years of experience. Audiologic evaluations (air- and bone-conduction threshold testing) and otolaryngologic evaluations (otoscopy) had been performed on all patients upon their enrollment in the previous study. Follow-up audiologic and otologic evaluations were performed at the midpoint of treatment for the former controls and biweekly during treatment for the extended-treatment patients. The final audiologic and otologic evaluations were conducted on all patients 4 weeks after the completion of each patient's final treatment. Hearing sensitivity was judged to be within normal limits if the air-conduction thresholds were less than 20 dB HL at any of 3 frequencies between 500 and 4,000 Hz *or* less than 25 dB HL at either of 2 of these frequencies. Audiologists were blinded to each patient's otologic findings, and otolaryngologists were blinded to each patient's audiometric findings. An alpha level of 0.05 was used for all statistical tests.

Table 1. Mean (standard deviation) pre- and posttest air-conduction thresholds (dB HL) in the former control group in the current study and in the active-treatment group in the previous study

Group	Test	Ear	n	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Current study: Former control group*	Pre	Right	30	31.0 (8.9)	32.0 (9.2)	22.2 (10.6)	28.8 (11.7)
		Left	30	30.7 (10.1)	32.7 (12.7)	23.0 (11.6)	28.7 (14.7)
Previous study: Active-treatment group†	Pre	Right	43	33.0 (10.9)	32.1 (10.1)	23.8 (11.0)	29.4 (12.1)
		Left	45	35.3 (11.4)	37.7 (10.8)	26.0 (12.2)	31.4 (11.7)
Current study: Former control group	Post	Right	30	18.5 (12.3)	18.2 (12.8)	11.8 (11.3)	15.0 (11.6)
		Left	30	18.7 (8.2)	16.3 (9.7)	11.0 (9.9)	14.2 (11.8)
Previous study: Active-treatment group	Post	Right	43	18.3 (7.6)	15.5 (8.4)	7.9 (7.9)	12.0 (9.8)
		Left	45	19.0 (10.3)	17.3 (11.0)	8.7 (8.2)	14.7 (8.1)

** The former control group was made up of 30 patients who had served as untreated controls in the previous clinical trial.[1] Upon conclusion of their participation in that study, they elected to receive active treatment.*
† Data from the active-treatment group in the previous study[1] are presented for comparison purposes.

Results

Former control group. Of the 30 former controls, 27 underwent 7 weeks of treatment and 3 underwent 9 weeks.

We obtained pre- and posttreatment mean air-conduction thresholds (pretest and posttest, respectively) in both ears in the former controls and compared them with the same values for the active-treatment group in the previous study (table 1). The pretest values for the former controls were obtained 11 weeks following their enrollment in the previous study, and their posttest values were obtained 4 weeks following the completion of treatment in the current study. The mean pretest air-conduction thresholds in the 2 groups were within 4.6 dB at 500 Hz, within 5.0 dB at 1,000 Hz, within 3.0 dB at 2,000 Hz, and within 2.7 dB at 4,000 Hz. In the former control group, mean pretest 4-frequency pure-tone averages were 28.5 and 28.8 dB HL in the right and left ears, respectively; in the previous active-treatment group, the corresponding figures were 29.6 and 32.6 dB HL. Thus, the mean pure-tone averages were symmetrical within 4.1 dB for both ears of both groups.

Analysis of the mean differences in pre- and posttest air-conduction thresholds for both ears in the former control group revealed that treatment resulted in a statistically significant ($p < 0.001$) improvement in hearing in both ears, ranging from 10.3 to 16.3 dB, across the frequency range (table 2).

We conducted between-group comparisons to determine if treatment was more or less effective in the former control group than it had been in the active-treatment group in the previous study. We found no statistically significant differences (table 3). This finding can be interpreted as supporting the reliability of the findings reported in our previous study.

Hearing sensitivity. In the current study, recovery of hearing sensitivity to within normal limits following treatment occurred in 43 of the 60 ears (71.7%) in the former control group. Results of the previous study were similar, as active treatment restored hearing in 65 of 88 ears (73.9%).[1]

Otoscopic findings. We performed posttest pneumatic otoscopy in 34 of the 43 ears in the former control group in which hearing had been restored to within normal limits. Of these 34 ears, tympanic membrane mobility was found to be normal in 29 (85.3%), moderate in 1 (2.9%), slight in 3 (8.8%), and absent in 1 (2.9%). Overall, normal or moderate tympanic membrane mobility was observed in 30 of these ears (88.2%). In our previous study, normal or moderate tympanic membrane mobility was observed in 61 of 65 ears (93.8%).[1] This similarity further substantiates the efficacy of our device in improving middle ear function and hearing sensitivity in children with MEE, and it further supports the reliability of the findings reported in our previous study.

Extended-treatment group. The duration of extended treatment ranged from 2 to 4 weeks, bringing the total duration of all treatment to 9 to 11 weeks. We compared the pure-tone thresholds obtained following the completion of the initial treatment and the completion of extended treatment.

Following the initial treatment period, 6 of these patients had a unilateral hearing impairment (patients 2, 3, 4, 5, 6, and 7) and 2 patients had a bilateral hearing impairment (patients 1 and 8)—a total of 10 impaired ears (table 4). Following extended treatment, normal hearing was restored in 9 of the 10 ears (90.0%). Normal hearing was restored in 5 of the 6 patients with unilateral impairment and in both ears of the 2 patients with bilateral impairment.

Table 2. Mean (95% confidence interval) differences (posttest minus pretest) in air-conduction thresholds (dB HL) in the 30 former controls*

Ear	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Right	–12.8 (–17.5, –8.1)	–13.8 (–18.5, –9.2)	–10.3 (–14.3, –6.4)	–13.8 (–19.1, –8.5)
Left	–12.0 (–15.6, –8.4)	–16.3 (–21.2, –11.5)	–12.0 (–17.0, –7.0)	–14.5 (–20.0, –9.0)

** All differences are statistically significant ($p < 0.001$) according to the Student's paired t test. The negative values represent improvements in hearing sensitivity following treatment.*

When the number of ears that recovered after initial plus extended treatment (9) is added to the number that recovered after the initial treatment (65), normal hearing overall was restored in a total of 74 of 88 ears (84.1%).

Follow-up. Upon the completion of their children's participation in the current study, parents were advised to notify the investigators if they or others noticed any change in hearing status. One parent of a former control whose hearing was restored to within normal limits contacted us 1 month later to report a recurrence of the child's hearing impairment. Audiologic and otologic evaluations revealed that this child had experienced a recurrence of MEE. The parent was instructed to resume treatment for 3 weeks and to return for evaluation 2 weeks after the completion of that treatment. Although the treatment was completed, the parent and patient did not return until 4 months later. At that time, the child's hearing was normal in both ears.

In the report of our previous study, we noted that 3 children whose hearing had been restored by treatment subsequently experienced recurrent MEE with hearing loss. The hearing in all 3 of those patients returned to within normal limits after treatment had been reinstituted for 2 weeks. These findings suggest that the use of our modified Politzer device may be effective for the treatment of recurrent MEE in patients who have already responded to previous treatment. Larger studies are needed to confirm the efficacy of our device in treating recurrences.

Discussion

A limitation of this investigation was the absence of a control group. This study, however, represents a follow-up of the participants from our previous study whose hearing sensitivity did not recover to within normal limits following treatment, so we did not recruit another control group. Therefore, the contribution of spontaneous recovery to the improvement observed cannot be entirely ruled out. Recall that the former control group and the extended-treatment group demonstrated close similarity in pretreatment air-conduction thresholds and in several other measures, including the degree of improvement from pre- to posttest, the percentages of ears demonstrating recovery of hearing sensitivity to within normal limits at the posttest, and the percentages of ears with normal-to-moderate tympanic membrane mobility. The close similarity between the two groups in (1) pretest hearing sensitivity, (2) the degree of improvement from pre- to posttest, (3) the percentages of ears demonstrating recovery of hearing sensitivity within to normal limits at the posttest, (4) the substantial and significant ($p < 0.001$) improvement in hearing sensitivity from pre- to posttest, and (5) the lack of change in hearing sensitivity in the former control group throughout the 11-week period in our previous study followed by improvement only after initiation of the home treatment (with increasingly greater improvement observed with the progression of treatment as evidenced by the results of monitoring during the treatment) suggests that the major factor accounting for the improvements seen at the posttest was home treatment rather than spontaneous recovery.

Table 3. Mean (95% confidence interval) differences between groups (values for the previous study's active-treatment group minus values for the current study's former control group) in the changes (posttest minus pretest) in air-conduction thresholds (dB HL)*

Ear	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Right	–1.9 (–7.4, 3.5) $p = 0.48$	–2.8 (–8.5, 2.9) $p = 0.33$	–5.6 (–11.3, 0.1) $p = 0.053$	–3.6 (–10.2, 2.9) $p = 0.28$
Left	–4.3 (–9.3, 0.6) $p = 0.09$	–2.0 (–7.4, 3.4) $p = 0.46$	–5.3 (–11.1, 0.4) $p = 0.07$	–2.3 (–8.5, 3.9) $p = 0.47$

** Statistical significance was determined according to an independent samples t test (assuming equal variances). The negative values represent improvements in hearing sensitivity following treatment.*

Table 4. Pure-tone thresholds for 500 through 4,000 Hz in 8 patients following initial treatment and following extended treatment

		Frequency (Hz)							
		Right ear				Left ear			
Pt.	Test	500	1,000	2,000	4,000	500	1,000	2,000	4,000
1	After 7 wks of initial Tx*	30	25	15	25	25	25	5	25
	After extended Tx†	10	10	15	10	10	10	5	0
2	After 7 wks of initial Tx	15	5	5	5	35	25	15	30
	After extended Tx	10	10	0	5	20	15	5	15
3	After 7 wks of initial Tx	25	30	25	20	25	20	10	15
	After extended Tx	20	15	15	10	15	15	15	10
4	After 7 wks of initial Tx	30	25	30	25	15	10	10	15
	After extended Tx	10	15	15	10	10	10	10	10
5	After 7 wks of initial Tx	30	30	10	10	15	15	5	15
	After extended Tx	25	15	15	15	25	20	5	15
6	After 7 wks of initial Tx	10	20	10	10	25	30	20	25
	After extended Tx	15	15	5	10	15	15	15	10
7	After 7 wks of initial Tx	15	25	20	10	55	60	45	40
	After extended Tx	10	0	10	10	35	45	30	15
8	After 7 wks of initial Tx	35	30	20	20	35	35	20	15
	After extended Tx	20	20	0	15	15	10	10	0

** Previous study. Posttest was performed 4 weeks following the conclusion of the 7-week treatment course. Note that these patients had been treated for 7 weeks in the previous clinical trial, but they did not reach the criteria for complete recovery. Their pure-tone thresholds upon initial enrollment in the previous clinical trial were poorer than those indicated in this table.*

† Current study. Posttest was performed 2 weeks following the conclusion of extended treatment.

In a recent prospective follow-up study of children with persistent MEE and hearing loss who had been treated early in life with tympanostomy tubes, Valtonen et al concluded that parents must be advised of the necessity of lengthy (5 yr) follow-up monitoring, the possible need for repeated surgical placement of tympanostomy tubes, and the possibility that middle ear complications may require other surgical management.[4] Use of our device might serve as an alternative to repeated surgical insertions of ventilation tubes; further research is needed to investigate this possibility.

In conclusion, the findings of our current study further substantiate the efficacy of our modified Politzer device in improving middle ear function and hearing sensitivity in children with MEE, and they support the reliability of the findings of our previous study.

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