Reduced Auditory Efferent Activity in Childhood Selective Mutism

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Background: *Selective mutism is a psychiatric disorder of childhood characterized by consistent inability to speak in specific situations despite the ability to speak normally in others. The objective of this study was to test whether reduced auditory efferent activity, which may have direct bearings on speaking behavior, is compromised in selectively mute children.*

Methods: *Participants were 16 children with selective mutism and 16 normally developing control children matched for age and gender. All children were tested for pure-tone audiometry, speech reception thresholds, speech discrimination, middle-ear acoustic reflex thresholds and decay function, transient evoked otoacoustic emission, suppression of transient evoked otoacoustic emission, and auditory brainstem response.*

Results: *Compared with control children, selectively mute children displayed specific deficiencies in auditory efferent activity. These aberrations in efferent activity appear along with normal pure-tone and speech audiometry and normal brainstem transmission as indicated by auditory brainstem response latencies.*

Conclusions: *The diminished auditory efferent activity detected in some children with SM may result in desensitization of their auditory pathways by self-vocalization and in reduced control of masking and distortion of incoming speech sounds. These children may gradually learn to restrict vocalization to the minimal amount possible in contexts that require complex auditory processing.*

Key Words: Auditory processing, middle ear acoustic reflex, otoacoustic emission, selective mutism, social anxiety, vocalization

Selective mutism (SM) is a psychiatric disorder of childhood characterized by consistent reluctance or inability to speak in specific social situations despite the ability to speak normally in other situations (American Psychiatric Association 1994). This condition typically involves severe impairments in social and academic functioning. Common complications include school failure, social difficulties in the peer group, and aggravated intrafamilial relationships (e.g., Bergman et al 2002; Kristensen 2001; Meyers 1984; Remschmidt et al 2001; Steinhausen and Juzi 1996). Although selective mutism has been described in the medical and psychological literatures for many years (Kussmaul 1877; Tramer 1934), the etiology of the disorder remains unknown (Dow et al 1995).

From the earliest descriptions of SM, shyness, timidity, and social withdrawal have been almost universally mentioned as characteristic of the disorder. Steinhausen and Juzi (1996) reported that 85 of the 100 selectively mute children whom they had studied were described by their parents as shy, and about two thirds were described as anxious. Black and Uhde (1992, 1995) reported an overwhelming incidence of avoidant disorder or social phobia in children with SM, leading these authors to argue forcefully that SM should be treated as an extreme variant manifestation of social phobia rather than a separate diagnostic entity. This position has been challenged recently by data indicating that parents, teachers, and clinicians do not report greater social anxiety in children with SM compared with children with social anxiety (Manassis et al 2003) and that children with SM do not report greater social anxiety than children with social phobia alone during the performance of behavioral tasks that involve conversational interaction with a same-age peer (Yeganeh et al 2003). It should be noted that although many children with SM display shy temperament and social anxiety, only a small portion of socially anxious children meet DSM-IV diagnostic criteria for SM. These considerations suggest that SM may involve a unique component that is absent in typical manifestations of social phobia. Although social anxiety may produce reduction or even temporary paucity of speech in social contexts, other factors more directly associated with auditory processing mechanisms and their potential effect on speaking behavior may be involved in SM.

Any organism that produces vocalizations is confronted with two fundamental sensory problems. First, it needs to discriminate whether its sensory organs are being stimulated by self-generated or by external auditory signals. Second, when vocalizing, the organism's auditory system needs to prevent desensitization to simultaneous and subsequent external signals due to potential over-stimulation by its own vocalization (Hoy 2002). Animal research describes various mechanisms that inhibit the auditory system during vocalization. Some animals have sound sensitivity reflexes that temporarily reduce eardrum compliance and middle-ear transmission, making them less responsive to sound when stimulated by their own calls. Others deploy neural inhibition to suppress the effect of self-vocalization on the auditory system and so prevent desensitization to external stimulation centrally (McCasland and Konishi 1981; Metzner 1989, 1993, 1996; Muller-Preuss and Ploog 1981; Poulet and Hedwig 2002; Schuller and Radtke-Schuller 1990; Suga and Schlegel 1972; Suga and Shimozawa 1974). In humans, the most studied efferent feedback pathways to the auditory periphery are the middle-ear acoustic reflex (MEAR) and the olivocochlear (OC) efferent system (Liberman and Guinan 1998).

The neural circuit of the MEAR controls the contraction of the stapedius and tensor-tympany middle-ear muscles upon presentation of loud low-frequency sounds. Contraction of the stapedius stiffens the motion of the ossicular chain in the middle ear and thus attenuates the transmission of low-frequency sound through the middle ear by approximately 10–30 dB (Borg and Counter 1989; Moller 1994). It has been shown that the stapedius

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muscle is activated as a part of the vocalization process in humans (Borg and Zakrisson 1973, 1975) and that it could play an important role in reducing distortion, nonlinearities, and upward spread of masking (Borg and Zakrisson 1974). During self-vocalization, an individual's own ears are stimulated primarily by the low-frequency components of the speech, which reach the cochlea through both air and bone conduction. Therefore, it is assumed that the activity of the stapedius during vocalization has the effect of decreasing the masking influence of the speaker's own voice. This results in an improved capacity of the speaking person to hear other external sounds while vocalizing (Borg et al 1984).

Olivocochlear efferent suppression relies on efferent innervations to the organ of Corti from neurons originating in the olivary complex (Rasmusen 1946; Warr and Guinan 1979). Lateral OC neurons project mainly ipsilaterally onto afferent neuron dendrites near the inner hair cells. Medial OC neurons primarily project contralaterally and synapse at the base of the outer hair cells. It has been shown that contralateral acoustic stimulation can attenuate, through fibers of the medial OC bundle, the acoustic energy generated by outer hair cells contraction, known as transient evoked otoacoustic emissions (TEOAEs; Maison et al 1998, 2000; Ryan and Kemp 1996; Ryan et al 1991). The suppressor stimulus may be thought of as a masker that could be presented by an external source (e.g., clinical testing probes, speech of social partners) or by ones own vocalization. It has been suggested that the efferent activity of the medial OC bundle plays a significant role in improvement of signal-to-noise ratio and speech intelligibility in noise (Dewson 1968; Giraud et al 1997; Micheyl and Collet 1996).

Inefficient functioning of the MEAR and diminished TEOAE efferent suppression during vocalization could result in excessive masking of external stimuli and desensitization of the auditory pathway to incoming sounds. Such physiology could lead to reduced speech in the afflicted individual. Furthermore, reduced vocalization in such individuals would be particularly expected in situations that require highly efficient environmental sound processing such as in conversation with peers or classroom discussions. Therefore, the objective of our study was to test the hypothesis that auditory efferent activity may be reduced in selectively mute children.

Methods and Materials

Participants

Sixteen children with SM (age range of 5–16 years) were recruited for this study. DSM-IV criteria for selective mutism were ascertained through a semistructured interview with the parents. Normal speech production at home was verified through homemade videotapes of the children fluently conversing with members of their nuclear family. Fifty-six percent of the children had a history of evaluation and treatment by a mental health professional, and none were medicated during the study's assessments.

Comorbid diagnoses were assigned on the basis of a structured research diagnostic interview (Schedule for Affective Disorders and Schizophrenia for School Age Children—Present and Lifetime Version; K-SADS-PL; Kaufman et al 1997) conducted with the parents. Children with insidious developmental delays or mental retardation were excluded.

Participants with SM were individually matched for age and gender with a comparison group of 16 normally developing and freely speaking children recruited from the community and screened for psychiatric symptoms using parental reports on the Child Behavior Checklist (CBCL; Achenbach 1991). None of the children from the control group had surpassed clinical cutoff scores (Zilber et al 1994) on any of the narrow- or broad-band scales of the CBCL. Parents of children in the SM group reported significantly higher levels of mutism (M = 3.11, SD = .39) on the total score of the Selective Mutism Questionnaire (Bergman et al 2002) compared with parents of control children (M = 1.41, SD = .43), $t(30) = 9.71$, $p < .0001$. Table 1 provides a summary of participants' characterization data.

Table 1. Characteristics of Study Participants

	Selective Mutism ($n = 16$)	Normal Control ($n = 16$)
Age (Years)	8.21 (3.46)	8.22 (2.79)
Sex, (male/female)	5/11	5/11
SMQ	3.11 (.39)	1.41 (.43)
Comorbidity, *n* (%)		
Social phobia	10 (62.5)	—
Simple phobia	4 (26.7)	—
Enuresis	3 (18.8)	—
Simple motor tics	2 (12.5)	—
Dysthemia	1 (6.3)	—
CBCL		
Internalizing	—	−.72 (.44)
Externalizing	—	−.55 (.63)

Mean (SD) unless otherwise specified.
SQM, Selective Mutism Questionnaire; CBCL, Child Behavior Checklist.

Procedure

The study protocol was approved by the Tel Aviv University review board and the Chaim Sheba Medical Center ethics review committee. Following a national television segment on SM inviting parents to contact our laboratory, we received a large number of calls. Parents were briefly interviewed over the phone to determine plausible diagnosis of SM. Parents of children who seemed eligible were invited to an interview at the university, where a detailed explanation of study procedures was given and parental informed consent obtained. Older children were requested to add their signed consent as well. The parents were interviewed regarding their child's SM symptoms, and DSM-IV diagnostic criteria were ascertained. Other psychiatric conditions were assessed using the K-SADS-PL.

Children from both groups who met the study's psychiatric inclusion criteria were invited for a day of audiologic assessment. All children were first tested with pure-tone audiometry and tympanometry. These preliminary tests revealed two children with SM and two control children with serous otitis media. These children were excluded from the study. Participants who had normal air-conduction thresholds (i.e., pure-tone average of .5, 1, and 2 kHz $\leq$ 15 dB hearing level [HL]) in both ears (American Speech-Language-Hearing Association Audiologic Assessment Panel 1996, 1997) as well as type A tympanograms were further tested for speech reception thresholds, speech discrimination of phonetically balanced monosyllabic words, and efferent auditory activity using measures of MEAR thresholds, MEAR decay function, and suppression effect of TEOAE. In addition, afferent auditory function at the acoustic nerve and brainstem levels was assessed using auditory brainstem response (ABR).

Acoustic Reflex Testing

A Grason-Stadler GSI-33, middle ear (V.2) analyzer was used for measurements of MEAR pure-tone thresholds at .5, 1, and 2 kHz and for reflex decay testing. Threshold was defined as the lowest intensity level of the tone needed to elicit a .02-mL

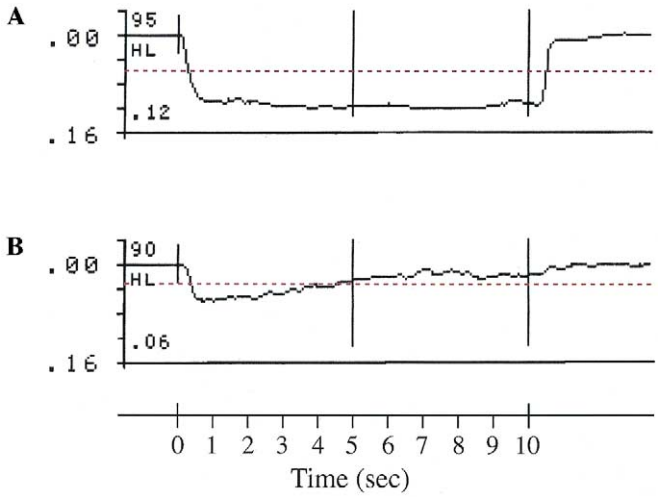

Figure 1. Illustration of (**A**) normal middle-ear acoustic reflex (MEAR) decay function and (**B**) abnormal MEAR decay functions taken from representative participants who were stimulated with 10 sec of 1-kHz tones. Dashed red line represents 50% of reflex's initial amplitude.

decrease in middle ear admittance on at least two of three trials. The initial presentation level was 70 dB HL and levels were elevated by 5-dB increments until threshold was detected. In accord with the GSI-33 protocol, the maximum stimulus output was 120 dB HL for contralateral recordings for all tested frequencies, 110 dB HL for ipsilateral recording at .5 and 1 kHz, and 105 dB HL for ipsilateral recording at 2 kHz. For ipsilateral testing, the stimulus was presented to the same ear where immittance measurements were made. For contralateral testing, immittance measurements were made in one ear while the stimulus was presented to the opposite ear. Following Wiley et al (1987) abnormal MEAR threshold was defined as $>$ 100 dB HL.

Reflex decay function was measured ipsi- and contralaterally to .5- and 1-kHz pure tones. During each test run, the eliciting stimulus was presented 10 dB above the MEAR threshold for a period of 10 sec at the tested frequency. Following Gelfand (2002), normal decay function was noted for full 10 sec of stable reflex action (Figure 1A). Abnormal decay was defined as a $\geq$ 50% decrease from the peak's initial amplitude within the testing epoch (Figure 1B).

Testing of TEOAEs and TEOAE Suppression Effect

The TEOAEs were recorded and analyzed using an ILO92 Echoport OAE analyzer V.4.2 (Otodynamics, Hatfield, United Kingdom) with a SDG-type probe. The quick screen mode was used. This mode employs click stimuli produced by 80-μsec rectangular electric pulses presented at a rate of 80/sec in the nonlinear mode of stimulation. Noise rejection level was set at 54.9 dBpe sound pressure level (SPL). Emissions in response to 260 low-noise samples were averaged. A recording window of 2.5–12.5 msec poststimulus was used for the analysis.

Before testing for TEOAE suppression effects, each subject's emissions were recorded in both ears using a stimulus level set at 80 $\pm$ 3 dBpe SPL. The presence of normal TEOAEs was determined by a whole reproducibility level $\geq$ 50%, and signal-to-noise ratio $\geq$ 3 dB in three of four frequency bands (1.6, 2.4, 3.2, and 4 kHz).

All subjects were tested for TEOAE suppression effect via six successive TEOAE measurements, alternately without and with contralateral acoustic stimulation (CAS). For all subjects, the first measurement was always without CAS. The click level for suppression of TEOAE measurements was set at 74 $\pm$ 1 dBpe SPL. The CAS consisted of a continuous white noise presented to the contralateral ear via an SM-N insert earphone. Threshold to the noise was measured in both ears, and CAS was presented at 40-dB sensation level. Amplitudes of the three TEOAE recordings with CAS and the three TEOAE recordings without CAS were averaged separately. The suppression effect was calculated by subtracting the mean TEOAE with CAS from the mean TEOAE without CAS. It should be noted that the procedure for documenting the suppression effect requires subjects to remain still for a long period of time. Because of the young age of many of the participants in the study and their limited ability to cooperate with the demand to remain still for prolonged periods of time, we chose the quick screen mode because it permits a shorter duration of procedure.

Recording of ABR

We recorded ABRs using a Biologic Navigator-Pro Evoked Potential System (Bio-logic Systems Corp., Mundelein, Illinois). Electrodes were placed at Cz and the earlobe ipsilateral to the stimulated ear. A ground electrode was placed at the contralateral earlobe. Impedance was kept below 5 kΩ. Reponses were amplified with a gain of 100,000 and digitally filtered with a bandwidth of .1–3 kHz. Each ear was stimulated by two blocks of alternating 85 dB HL clicks with presentation rates of 21/sec or 51/sec, and two blocks of alternating 65 dB HL clicks with presentation rates of 21/sec or 51/sec. Clicks were delivered using ER-3 insert earphones. Responses in each condition were averaged over 2000 individual sweeps, with a sweep time of 16 msec. Peak absolute latencies of ABR waves I, III, and V were reliably obtained from all participants. Interpeak latencies of waves I–III and I–V were also calculated.

Data Analysis

Missing data were excluded from analyses on a test-by-test basis. Analyses of between-group differences on standard clinical abnormality cutoff scores were supplemented, when possible, by analyses of these same measures using continuous scales. For between-group differences on dichotomous variables, Fisher's Exact Test was used. This test is most useful when the total sample size and the expected values are small, as in our sample. In tests of group differences involving continuous variables, *t* tests were used with one-sided or two-sided alphas reported for planed or exploratory contrasts respectively.

Results

All children had normal speech reception thresholds, normal speech discrimination, and within normal range ABR wave latencies. *t* test analyses revealed no significant between-group differences for any of the measures. Table 2 presents means and SDs of absolute and interpeak ABR wave latencies at an intensity level of 85 dB HL and click presentation rate of 21/sec for the SM and control children. For brevity, only the standard (85 dB HL, 21/sec) ABR data are fully reported. Reducing the intensity to 65 dB HL or increasing the click presentation rate to 51 clicks per second did not yield significant between-group differences in ABR latencies.

MEAR Thresholds and Decay Functions

Of 16 children from the SM group, 9 displayed no MEAR at maximum stimulation level in at least 2 of 12 assessment conditions (2 ears $\times$ 3 frequencies $\times$ 2 stimulation side) compared with only two children in the control group. Of the nine

Table 2. Mean Auditory Brainstem Response Peaks and Interpeak Latencies in Msec by Ear for the Selective Mutism (SM) and Control Groups (Ctrl)

	Wave I		Wave III		Wave V		Interpeak I–III		Interpeak I–V	
	SM	Ctrl	SM	Ctrl	SM	Ctrl	SM	Ctrl	SM	Ctrl
Right Ear	1.42 (.10)	1.45 (.10)	3.56 (.12)	3.59 (.14)	5.38 (.13)	5.44 (.15)	2.14 (.14)	2.13 (.16)	3.96 (.15)	3.98 (.19)
Left Ear	1.43 (.13)	1.47 (.11)	3.64 (.20)	3.61 (.14)	5.45 (.21)	5.42 (.12)	2.21 (.23)	2.14 (.17)	4.02 (.24)	3.95 (.17)

Data are given as mean (SD).

selectively mute children with no MEAR, six failed to show MEAR in both ears, whereas the two children from the control group showed no MEAR in the left ear only. Figure 2 presents abnormal MEAR thresholds for individual subjects by group, ear, stimulation side, and stimulation frequency. Nonparametric analysis were performed to assess between-group differences in abnormal (absent or greater than 100 dB HL) versus normal (equal or lower than 100 dB HL) MEAR threshold. Higher incidence of abnormal right ear ipsi- and contralateral MEAR thresholds at 1 and 2 kHz were found for the SM group compared with the control group (Fisher's Exact Tests = .009, .04, .02, and .007, respectively). The SM group displayed higher incidence of abnormal left ear contralateral MEAR at .5, 1, and 2 kHz compared with the control group (Fisher's Exact Tests = .053, .05, and .008 respectively). Finally, the SM group had higher incidence of abnormal left ear ipsilateral MEAR threshold than control subjects at 2 kHz (Fisher's Exact Tests = .005). No significant between-group differences were found for both ispi- and contra-lateral right ear MEAR thresholds at .5 kHz and for ipsilateral left ear threshold at 1 kHz.

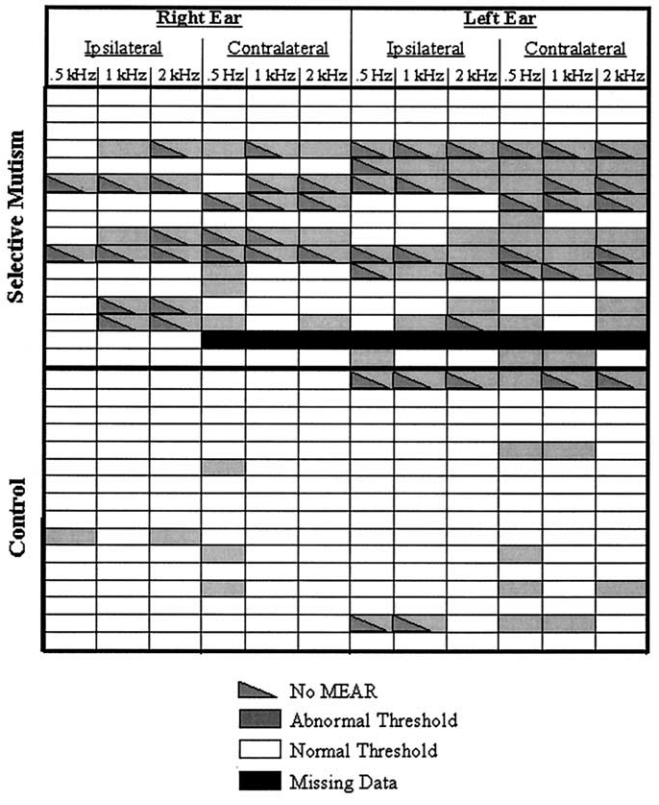

Figure 2. Abnormal middle-ear acoustic reflex (MEAR) thresholds (≥ 100 dB HL, in gray) by ear, stimulation side, and stimulation frequency for the SM and control groups. Each row represents one participant. No MEAR at maximum stimulation level is flagged.

To obtain an estimate of the between-group differences in MEAR threshold magnitude, tested frequencies that repeatedly failed to elicit MEAR at the maximum stimulation level were entered into the analyses as 120, 110, or 105 dB "threshold" values in concurrence with the maximum output for the tested frequency. This analytic strategy truncates the actual MEAR thresholds on such trials, which are, in fact, higher. Table 3 summarizes the means of ipsi- and contralateral MEAR thresholds of the selectively mute and control groups. Compared with children in the control group, children with SM showed significantly higher right ear ipsi- and contralateral MEAR thresholds for both 1- and 2-kHz tones. This effect was marginally significant for right ear ipsi- and contralateral thresholds for .5-kHz tones. For the left ear, children with SM showed significantly higher ipsi- and contralateral MEAR thresholds for all stimulation frequencies (i.e., .5, 1, and 2 kHz).

To further elucidate our findings, between-group differences in normal versus abnormal MEAR decay function were assessed using nonparametric analyses. Table 4 summarizes the number of abnormal MEAR decay functions for each group by ear, frequency, and laterality. Compared with children in the control group, selectively mute children displayed greater incidence of abnormal right ear MEAR decay functions for ipsilateral stimulation at .5 and 1 kHz, as well as contralateral stimulation at .5 kHz. In addition, a marginally significant finding of higher incidence of abnormal left ear ipsilateral MEAR decay function at .5 kHz

Table 3. Mean Acoustic Reflex Thresholds in dBHL by Ear, Frequency, and Laterality for the Selective Mutism and Control Groups

	Selective Mutism	Normal Control	t	*df*[a]	*p* Value (one-tailed)
Right Ear					
Ipsi					
500 Hz	94 (10)	90 (7)	1.34	30	.10
1000 Hz	96 (12)	87 (5)	2.88	30	.004
2000 Hz	95 (10)	87 (7)	2.57	30	.008
Contra					
500 Hz	103 (12)	97 (5)	1.61	29	.06
1000 Hz	102 (14)	92 (5)	2.56	29	.01
2000 Hz	100 (13)	90 (4)	2.88	29	.004
Left Ear					
Ipsi					
500 Hz	98 (10)	92 (8)	1.73	29	.05
1000 Hz	97 (11)	89 (9)	2.07	29	.03
2000 Hz	97 (10)	87 (8)	3.11	29	.002
Contra					
500 Hz	106 (11)	99 (9)	3.69	29	.04
1000 Hz	104 (14)	94 (9)	4.15	29	.02
2000 Hz	106 (14)	94 (10)	4.39	29	.005

Data are given as mean (SD).

[a]One participant with selective mutism did not complete right ear contralateral and left ear contra and ipsilateral middle-ear acoustic reflex threshold assessment.

Table 4. Number of Children Showing Abnormal Middle-Ear Acoustic Reflex (MEAR) Decay Function by Ear, Frequency, and Laterality for the Selective Mutism and Control Groups

	Selective Mutism (Abnormal/Total)	Normal Control (Abnormal/Total)	Likelihood Ratio Value	Fisher's Exact (one-sided)
Right Ear				
Ipsi				
500 Hz	6/13	2/16	4.16	.05
1000 Hz	5/11	1/16	5.97	.03
Contra				
500 Hz	7/13	3/16	4.15	.05
1000 Hz	4/11	2/16	2.13	ns
Left Ear				
Ipsi				
500 Hz	4/9	1/14	4.52	.06
1000 Hz	5/11	3/14	1.64	ns
Contra				
500 Hz	5/10	4/16	1.68	ns
1000 Hz	4/9	2/15	2.85	ns

Due to technical failure, one child from the selective mutism group did not have MEAR decay data. Reflex decay function was not measured for ears and frequencies that failed to produce MEAR at maximum stimulation levels.

was noted for children in the SM group compared with control children.

TEOAEs and Suppression of TEOAE

We contrasted TEOAE suppression among 9 and 12 right ears and 9 and 10 left ears in SM versus control participants, respectively. Four left ears and three right ears in the SM group as well as two left ears and one right ear in the control group were not tested because of either technical failures or children's inability to comply with the testing procedure. Three right ears and three left ears in the SM group and three right ears and four left ears in the control group displayed no recordable TEOAEs. It is worth noting that most of the lost TEOAE data pertained to children who were younger than 6 years of age. It may be the case that these young children had greater difficulty complying with the restrictive demands of the TEOAE recording procedure.

Table 5 summarizes TEOAE amplitude and suppression values for the SM and control groups. Nonsignificant between-group differences were found for TEOAE amplitudes in both ears; however, as shown in Figure 3, the SM group displayed significantly lower TEOAE suppression in the right ear compared with the control group. Nonsignificant between-group difference in TEOAE suppression was found for the left ear.

Ten participants from the control group and seven from the SM group had valid TEOAE suppression values in both ears. Further exploration of the frequency of participants per group showing left or right ear predominance for contralateral suppression indicated that in the control group, seven participants showed greater right ear suppression effect, two had equal TEOAE contralateral suppression values in both ears, and only one participant presented with greater left ear suppression value. In contrast, four and three participants from the SM group showed right and left ear suppression effect predominance respectively. It should be noted, however, that this tendency for a greater frequency of right ear suppression predominance in the control group and no clear tendency for a greater frequency of

Table 5. Mean Amplitude of TEOAE and TEOAE Suppression Effect in dB for the Selective Mutism and Control Groups

	Selective Mutism	Normal Control	*t* Value	*df*	*p* Value (one-tailed)
Right Ear					
TEOAE	11.12 (3.52)	12.54 (4.16)	.82	19	ns
TEOAE Suppression	.93 (.78)	1.87 (.90)	2.50	19	.03
Left Ear					
TEOAE	10.92 (2.78)	13.90 (4.34)	1.76	17	ns
TEOAE Suppression	1.32 (1.02)	1.52 (1.52)	.33	17	ns

Data are given as mean (SD).
TEOAE, transient evoked otoacoustic emission.

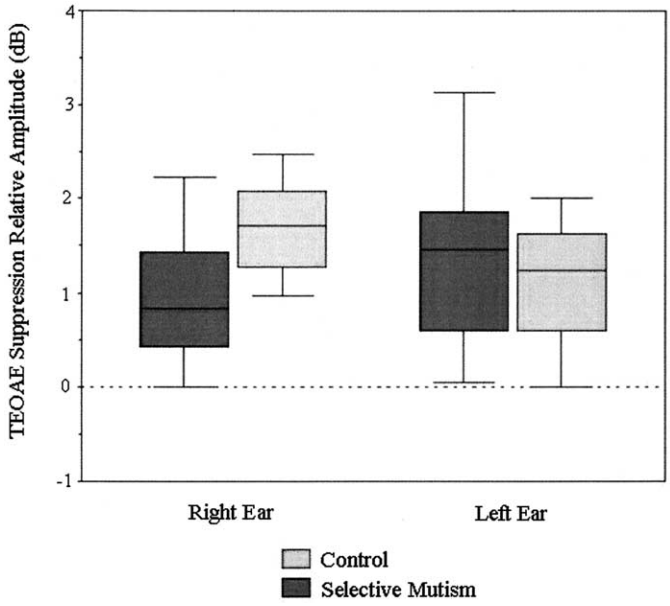

Figure 3. Box plot comparison of right and left ears transient evoked otoacoustic emission suppression for selectively mute and normally developing control children. The upper and lower boundaries of the standard box plots are the 25th and 75th percentiles; the whiskers extending from the box represent the highest and lowest values. The line across the box indicates the median.

right or left ear suppression predominance in the SM group was only at a trend level of significance, $\chi^2 = 3.39$, $p = .09$.

Clinical abnormality estimates of reduced TEOAE suppression effect were derived following Prasher et al (1994) in which TEOAE suppression magnitudes lower than 1 dB SPL were considered abnormal. Nonparametric analyses revealed that for the right ear, 5 of 9 children in the SM group had TEOAE suppression magnitudes lower than 1 dB SPL, whereas only 1 of 12 children in the control group had such abnormal suppression effect (Fisher's Exact Test, $p < .05$). No significant between-group differences were found in the left ear.

Dicussion

The findings from this study provide evidence for aberrations in MEAR function and diminished right ear TEOAE suppression in selectively mute children. These aberrations in auditory efferent activity in children with SM appear along with normal pure-tone and speech audiometry and normal brainstem transmission as indicated by ABR latencies. Based on the literature describing the possible effects of self-vocalization on hearing, it may be the case that reduced efferent activity during self-vocalization hinders selectively mute children's ability to process incoming auditory signals simultaneously.

The specific efferent deficiencies found in the selectively mute children from this study seem to match the reported perceptual and behavioral outcomes forming the core symptoms of the disorder. It is our conjecture that faced with the detrimental consequences of self-vocalization on external sound processing, children with SM may gradually, and probably unconsciously, adapt by whispering, restricted vocalization, and speech avoidance in contexts that require careful monitoring and complex auditory processing of incoming sounds.

Furthermore, the reported reductions in efferent activity and their functional implications in the control of masking seem to be consistent with various reports in the literature citing children with SM explaining their lack of speech with phrases such as, "my voice sounds funny, and I don't want others to hear it," or "my brain won't let me speak because my voice sounds funny" (Black and Uhde 1992; Boon 1994). Such subjective experience of self-vocalization, along side the masking and distortion of subsequent external signals, may lead to selective mutism in some children. Although it will be important for future studies to test whether children with SM display notable difficulties in auditory processing during self-vocalization, the specific neuroacoustic substrates revealed in our study make this particular hypothesis highly plausible.

In addition, our TEOAE data seem to be in accord with previous studies reporting right ear predominance for the suppression effect of TEOAE in healthy subjects (Khalfa and Collet 1996; Morlet et al 1999). The study of TEOAE suppression effect in psychiatric populations is scarce (cf. Collet et al 1993; Khalfa et al 2001; Veuillet et al 2001). As such, our data provide a contribution to the extant literature.

Although it is tempting to interpret the diminished efferent activity in children with SM as an etiologic factor for the disorder, the association between reduced auditory efferent activity, selective mutism, and social anxiety requires further elucidation. Specifically, abnormal MEAR function has been reported for introverted and socially withdrawn adults (Bar-Haim 2002), and reduced inhibitory cortical neural processes have been reported for socially withdrawn and anxious children (Bar-Haim et al 2003). Thus, at this point, at least three models of the association between SM and social anxiety deserve further consideration: 1) reduced auditory efferent inhibition at the brainstem level may lead to SM but not to social anxiety, 2) reduced auditory efferent inhibition may be associated with both SM and social anxiety in a parallel and unrelated way, or 3) the association between SM and reduced auditory efferent activity may be mediated by social anxiety. The third model seems to have the potential to accommodate both the findings of high comorbidity between SM and social anxiety on one hand and the uniqueness of SM symptoms on the other.

Selective mutism is a perplexing disorder to clinicians and parents. It is many times difficult to discern a clear pattern of situations and conditions in which the child with SM does or does not speak. How can we make sense of the fact that children with SM speak normally in some situations but not in others, and that within the same environment a child with SM may speak to some individuals but not to others? According to our suggested model, reduced auditory efferent activity may lead to auditory processing difficulties of incoming sounds during self-vocalization. Whether the child with SM may or may not speak in certain situations or with certain individuals as a function of his or her efferent deficiency may depend on the adaptive significance the child assigns to the accurate processing of incoming auditory information in that particular context. For instance, it may be "safe" to miss out on a word or two while speaking to a good and predictable friend in class, but it may prove more problematic during class discussion when addressing a teacher's question. We propose that children with SM are many times faced with the dilemma of choosing between speaking their minds and making sure they reliably follow the conversational context. We further propose that for some children, particularly those with shy temperament, this dilemma is often solved by mutism. Clearly, reduced auditory efferent activity does not deterministically lead to selective mutism in all children. In fact, the formulation of a single etiologic factor for SM seems implausible. First, some children may adapt differently to the auditory condition of reduced efferent activity. Second, our data indicate that approximately one forth of the children with SM do not display any abnormalities in auditory efferent activity. For these children, social anxiety, oppositional behavior, or another factor may be the cause of mutism; however, for approximately three fourths of children with SM, it may be the case that the combination of reduced auditory efferent inhibition during self-vocalization, along with shy, socially anxious, and inhibited temperament, culminates in fully diagnosable SM.

Further clarification of these possibilities and more careful analysis of the specific contexts in which children with SM do or do not speak may bear practical significance for the development of more efficient therapies for SM, which has established itself as a difficult-to-treat disorder. Specifically, potential pharmacotherapies could focus on procedures that directly target the functioning of the deficient auditory neural circuits outlined earlier.

To date, only a small number of studies has reported pharmacotherapy for childhood selective mutism using selective serotonin reuptake inhibitors (Black and Uhde 1994; Carlson et al 1999; Dummit et al 1996; Golwyn and Sevlie 1999). These studies have shown some success in alleviating social anxiety symptoms in children with SM but report only limited improvement in speech behavior. Taking into account the findings from our study, it is interesting to note that although there are some indications for the involvement of serotonin in auditory processing, (Gopal et al 2000; Thompson et al 1998), the primary neurotransmitter involved with inhibitory efferent activity from the OC bundle, as well as with mediation of neuromuscular

junction activation of the MEAR, is in fact acetylcholine (Godfrey et al 1990; Simmons 2002). Therefore, pharmacotherapy targeting cholinergic systems may prove effective for the treatment of childhood SM.

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