Resistant Bacteria in the Adenoids

A Preliminary Report

John E. McClay, MD

Objective: To determine the incidence of resistant bacteria in adenoid cultures from children with and without middle ear disease and rhinosinusitis symptoms.

Design: Children meeting the requirement for tympanostomy tube placement underwent an adjuvant adenoidectomy for symptoms of adenoid hypertrophy or recurrent rhinosinusitis. Adenoid tissue and coexisting middle ear fluid, if present, were cultured.

Setting: Tertiary referral children’s hospital with community-based satellite clinics.

Patients: Forty-six patients ranging in age from 1 to 11 years (68% <3 years) with recurrent or persistent otitis media and symptoms of adenoid hypertrophy or rhinosinusitis (study patients) underwent tympanostomy tube placement and adenoidectomy with culture of the adenoids and middle ear effusions. Eighteen patients with adenoid hypertrophy without ear disease or rhinosinusitis were used as controls.

Interventions: Tympanostomy tube placement and adenoidectomy.

Main Outcome Measures: Presence or absence of resistant bacteria.

Results: Resistant bacteria were found in cultures of the adenoids in 56% (26/46) of the study group compared with 22% (4/18) of the control patients (P<.02). Also, strains of Streptococcus pneumoniae, Haemophilus influenzae, or Moraxella catarrhalis were found in cultures from 78% (36/46) of the study group, compared with 44% (8/18) of those from the control group (P<.01). Resistant isolates were found in 65% (23/35) of the S pneumoniae, 37% (18/49) of the H influenzae, and 100% (19/19) of the M catarrhalis cultures from the adenoids or middle ear spaces.

Conclusion: Resistant bacteria are present in significant amounts in the adenoids of children with middle ear disease and rhinosinusitis symptoms compared with patients without those diseases or symptoms.

Arch Otolaryngol Head Neck Surg. 2000;126:625-629

Resistant bacteria that can cause infections in the middle ear, nasal cavity, and respiratory system have been an increasing concern over the past 10 years. The rapid progression of the resistance of the 3 most common pathogens of otitis media and rhinosinusitis (Streptococcus pneumoniae, Haemophilus influenzae, and Moraxella catarrhalis) has been alarming. In 1997, in more than 11 300 isolates evaluated from 434 institutions in 45 states and the district of Colombia, 30% to 40% of the H influenzae and S pneumoniae strains and 90% of the M catarrhalis strains were resistant.1

Placement of tympanostomy tubes has been an effective way to treat patients with recurrent otitis media and persistent otitis media with effusion. The bacteria infecting the middle ear space have their origin in the nasopharynx, where the adenoids reside. In fact, pathogenic bacteria have been cultured simultaneously from the middle ear space and the nasopharynx in almost 70% of patients, and pathogenic bacteria have been found in more than 95% of the nasopharynges of children with acute otitis media.2 When the adenoids have been evaluated specifically, more pathogenic bacteria have been found in patients with recurrent or persistent otitis media than in controls,3,5 indicating that the adenoids may act as a reservoir of infection. In several notable prospective studies with large numbers of patients, adjuvant or primary adenoidectomy has been reported to decrease the persistence or recurrence of otitis media in children.6-8

Similarly, Lee and Rosenfeld9 describe the effectiveness of adenoidectomy for signs and symptoms of sinusitis, regardless of the weight of the adenoids. The pathogenic bacteria they cultured from the adenoids in children with rhinosinusitis are similar to the bacteria found in the adenoids of children with otitis media. In the past 10 years, the most

From the Division of Pediatric Otolaryngology, Department of Otolaryngology–Head and Neck Surgery, University of Texas Southwestern Medical Center at Dallas.
SUBJECTS AND METHODS

Children undergoing tympanostomy tube placement for recurrent otitis media (>4 episodes in 6 months or >6 episodes in the past year) or persistent otitis media (bilateral middle ear fluid present for >3 months) or both were evaluated for adenoidectomy. No other historical factors, such as day care or secondhand smoke, were recorded. If the surgeon thought that adenoidectomy was indicated, based on preoperative signs of sinusitis or adenoid hypertrophy (mouth breathing, snoring, nasal airway obstruction), then adenoidectomy was performed.

At the time of surgery, the external auditory canal was cleaned with alcohol. After the myringotomy, the middle ear fluid was aspirated via a 15-gauge blunt needle attached to a tuberculin syringe connected to the suction tubing. Fluid present in the middle ear space was classified as absent, purulent, serous, or mucoid. The syringe was capped and sent directly to the microbiology laboratory. The adenoids were removed with a curette. An adenoid specimen of at least 5 mm³ was placed into a culturette, and the medium was activated. Most specimens were larger than 1 cm³. The specimens were also sent directly to the microbiology laboratory.

Cultures were performed from March 1997 to September 1997. Initially, specimens from the middle ear were sent for aerobic and anaerobic culturing. After 20 consecutive anaerobic cultures were negative, only aerobic cultures were attempted. Only aerobic bacteria were cultured from adenoid tissue. No attempt was made to culture anaerobic bacteria from adenoid tissue. The adenoid tissue was homogenized in a small amount of brain-heart infusion broth or sterile physiological nonbacteriostatic saline. An aliquot of the homogenized tissue was then inoculated on a Trypticase soy agar medium that contained 5% sheep blood, a chocolate agar plate, and a MacConkey agar plate. The plates were then incubated in 5% to 8% carbon dioxide at 35°C for 24 hours. For middle ear fluid, several drops were inoculated on the same types of media and incubated under the same conditions.

Plates were removed and examined for sufficient growth. If there was no growth or insufficient growth, the plates were returned for an additional 24 hours. Plates were held for 72 hours before being reported as no growth. Plates were examined for the potential pathogens S pneumoniae, H influenzae, M catarrhalis, and group A β-hemolytic streptococcus. Gram-negative rods and S aureus or other potential pathogens with moderate to abundant growth over other microbial flora were recorded as pathogenic.

Identification and susceptibility testing was performed on strains of S pneumoniae, gram-negative rods, and strains of S aureus. Organisms were determined to be sensitive or resistant to oxacillin sodium. In this preliminary investigation, if resistance was found, no attempt was made to specify whether it was an intermediately or highly resistant strain. β-Lactamase testing was performed on strains of H influenzae and M catarrhalis. Mixed oropharyngeal flora was reported if multiple morphotypes of bacteria were observed with no clearly predominant potential pathogen. Organisms included as normal flora were α-hemolytic streptococci and enterococci, coagulase-negative staphylococci, and Corynebacterium, Neisseria, Haemophilus, Micrococcus, and Stomatococcus species. Gram-negative rods, S aureus, and yeast were considered normal flora if they were found in small numbers. Statistical analysis was performed with the paired t test.

RESULTS

In 46 patients with recurrent or persistent otitis media or both and signs of rhinosinusitis or adenoid hypertrophy, an adjuvant adenoidectomy was performed in addition to placement of tympanostomy tubes (study group) (Table 1). Eighteen patients with no signs of recurrent ear disease or sinusitis within the last year underwent an adenoidectomy in conjunction with tonsillectomy for obstructive sleep apnea (control group). The average age of the study population was 3 years (age range, 9 months to 11 years). The average age of the control group was 5.8 years (age range, 2.5-13 years). Sixty-eight percent of the study population was younger than 3 years. Only 1 patient (6%) in the control group was younger than 3 years.

All resistant bacteria cultured from patients in the study or control group were isolates of S pneumoniae, H influenzae, or M catarrhalis. None of the other types of bacteria cultured showed resistance. Twenty (55%) of the 36 cultures from study patients with evidence of these 3 pathogens yielded 2 or more of these 3 bacterial types. Of the 20 patients with multiple pathogens present, 18 (90%) yielded at least 1 type of resistant strain and 10 (50%) yielded at least 2 types of resistant strains. In comparison, of the 8 patients in the control group who had either S pneumoniae, H influenzae, or M catarrhalis isolated in their adenoids, 5 (60%) had multiple pathogenic strains present, a similar percentage to the study group.

Of the 46 patients who underwent tube placement and adenoidectomy, 17 had both middle ear spaces clear at the time of tube placement. Twenty-nine patients had fluid present, and 9 (31%) of the 29 patients had the same fluid in both middle spaces. Twenty patients had different types of fluid in their middle ear spaces, combining purulent, serous, mucoid, or absent fluid. Thirteen (45%) of the 29 patients in the study group had at least 1 middle ear space with purulence, and 16 (55%) had a combination of mucoid, serous, or absent fluid.

Only 10 of the 29 patients with middle ear effusions had positive cultures for bacteria in one or both middle ear spaces. Eight (80%) of the 10 patients with bacteria recovered from one or both middle ear effusions had H influenzae, S pneumoniae, or M catarrhalis, and 7 (88%) of the 8 patients had the same bacteria present in the adenoids. The 1 patient with M catarrhalis recovered from the middle ear space did not have M catarrhalis in the adenoids, even if
though *M. catarrhalis* was found in 18 of the 46 adenoid cultures in the study patients.

The breakdown of bacteria found in the adenoids is listed in Table 2. The percentage of resistant isolates for each pathogen was essentially the same, independent of the patient group. The true significance between groups lies in the total number of patients from each group that had 1 of the 3 main pathogens of otitis media—*S. pneumoniae*, *H. influenzae*, or *M. catarrhalis*—cultured, since those bacteria had the only isolates showing resistance. The control group had a greater percentage of isolated group A β-hemolytic streptococcus or normal oropharyngeal flora present.

Furthermore, the percentage of resistance was essentially the same whether the bacteria were cultured from the adenoids or middle ear effusions. The total number of isolates is listed in Table 3. Occasionally, the middle ear space had more than 1 pathogen cultured when both pathogens were present, since there are more isolates listed in Tables 2 and 3 than there were positive cultures obtained. Overall, 35 *S. pneumoniae* isolates, 49 *H. influenzae* isolates, and 19 *M. catarrhalis* isolates were identified.

### Table 1. Bacteria Cultured in the Adenoids*

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Recurrent or Persistent Otitis Media (n = 46)</th>
<th>Adenotonsillar Hypertrophy (n = 18)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pathogenic bacteria cultured</td>
<td>41 (89)</td>
<td>12 (67)</td>
<td>&gt;.05</td>
</tr>
<tr>
<td><em>Streptococcus pneumonia</em>, <em>Haemophilus influenzae</em>, and/or <em>Moraxella catarrhalis</em></td>
<td>36 (78)</td>
<td>8 (44)</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Resistant bacteria</td>
<td>26 (56)</td>
<td>4 (22)</td>
<td>&lt;.02</td>
</tr>
<tr>
<td>Multiple pathogenic bacteria</td>
<td>20 (43)</td>
<td>5 (28)</td>
<td>. . .</td>
</tr>
</tbody>
</table>

*Each value describes the number of patients with that bacteria type cultured. Ellipses indicate not evaluated.

### Table 2. Bacterial Isolates Cultured in the Adenoids*

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Study Group</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pneumoniae</em></td>
<td>19 (63)</td>
<td>5 (60)</td>
</tr>
<tr>
<td><em>Haemophilus influenzae</em></td>
<td>24 (33)</td>
<td>7 (28)</td>
</tr>
<tr>
<td><em>Moraxella catarrhalis</em></td>
<td>18 (100)</td>
<td>1 (100)</td>
</tr>
<tr>
<td>Group A β-hemolytic streptococcus</td>
<td>5 (0)</td>
<td>4 (0)</td>
</tr>
<tr>
<td><em>Staphylococcus aureus</em></td>
<td>5 (0)</td>
<td>3 (0)</td>
</tr>
<tr>
<td>Others†</td>
<td>4 (0)</td>
<td>1 (0)</td>
</tr>
<tr>
<td>None</td>
<td>5</td>
<td>6</td>
</tr>
</tbody>
</table>

*Numbers in parentheses indicate the percentage of resistant strains.
† Includes Pseudomonas, groups C and F streptococci, and α-hemolytic streptococcus.

### Table 3. Bacterial Isolates Recovered From All Sites

<table>
<thead>
<tr>
<th>Bacteria</th>
<th>Resistant</th>
<th>Sensitive</th>
<th>% Resistant</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Streptococcus pneumoniae</em></td>
<td>23</td>
<td>12</td>
<td>65</td>
</tr>
<tr>
<td><em>Haemophilus influenzae</em></td>
<td>18</td>
<td>31</td>
<td>37</td>
</tr>
<tr>
<td><em>Moraxella catarrhalis</em></td>
<td>19</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

The resistant rate for the pathogens isolated in this study was higher than in previous reports for *S. pneumoniae* only, in which a resistant rate of 65% was seen, independent of the site where the resistant bacteria were cultured. The resistance of this pathogen is concerning, since it is a common cause of bacteremia and meningitis, and a highly resistant organism could result in death. In this study, the rates of resistance for *H. influenzae* and *M. catarrhalis* were similar to those reported previously.

The qualification of pathogenic vs nonpathogenic bacteria in this study is also similar to that in other studies. In this evaluation, *H. influenzae* was the most common pathogen cultured in the adenoids, similar to other reports. In fact, current research using in situ hybridization and polymerase chain reaction has found *H. influenzae* in 82% to 100% of the adenoids in patients with and without adenoid hypertrophy or chronic otitis media with effusion. The second most common pathogen in the adenoids in this study was *S. pneumoniae*. The amount of this bacteria cultured in the adenoids has varied from common to rarely found in previously published reports. The interaction between pathogens has been thought to be synergistic.

The concept that the adenoids act as a reservoir of infectious bacteria is controversial, with conflicting reports. Supporting this concept, this study showed that *H. influenzae, S. pneumoniae*, and *M. catarrhalis* are cultured more often from the adenoids removed from patients with recurrent or persistent otitis and rhinosinusitis or adenoid hypertrophy than from patients with adenoid hypertrophy and no middle ear or sinus disease. Similarly, Pillsbury et al demonstrated more pathogenic bacteria in the adenoids of patients with recurrent otitis media than in those with persistent serious otitis media or hypertrophy alone. Brodsky and Kock also cultured more bacteria from the adenoids of patients with
either recurrent or persistent otitis media than from the adenoids of patients without infections in the head and neck.

However, Brodsky and colleagues and others also found the same amount of pathogenic bacteria in the adenoids, regardless of size, in patients with otitis media and rhinosinusitis as in those of patients with adenoid hyperplasia alone. In contrast, Tomonaga et al showed more pathogenic bacteria in enlarged adenoids of patients with otitis media with effusion than in patients with hyperplasia of the adenoids alone. Even more confusing is the fact that Maw and Speller found the same amount of pathogenic bacteria in the adenoids and tonsils of patients with otitis media with effusion as in patients without any head and neck disease.

Regardless of the pathophysiology, adenoidectomy, independent of the size of the adenoids, has been shown to be effective in resolving chronic persistent otitis media with effusion and possibly recurrent otitis media in children older than 4 years. Adenoidectomy in children younger than 3 years has been shown to be safe, but its effectiveness in treating recurrent otitis media or rhinosinusitis has not been proved.

Similarly, Lee and Rosenfeld believed that the adenoids act as a nidus of infection in children with rhinosinusitis. They showed that bacteria were recovered from the adenoids and that patients improved after adenoidectomy, independent of the weight of the adenoids. Certainly, a critical consideration in determining the effectiveness of adenoidectomy for rhinosinusitis symptoms would seem to be the relationship of the adenoids to the nasopharynx of the individual choana. Since there are nasopharynges of different sizes, the weight may not be a true comparison between patients.

Most patients (68%) in this study group were younger than 3 years. The difference in age between the control group and the study group is concerning. Age appears to have an impact on the amount of sensitive and resistant bacteria found in the nasopharynx, with the frequency of pathogens in the nasopharynx decreasing with age. In fact, when carrier rates for resistant S pneumoniae were recently evaluated in adults and children in Sweden, prolonged carrier rates were noted in children younger than 1 year, in children with more than 6 ear infections, and in children who had their first ear infection before the age of 1 year. During the study period, only 1 child younger than 3 years who had airway obstructive symptoms as a result of adenotonsillar hypertrophy met the inclusion criteria for the control group, which excluded any cases of otitis media or rhinosinusitis in the past year. Obtaining biopsy specimens from the adenoids in children younger than 3 years who are undergoing surgical procedures at other sites may yield better controls.

Colonization of the nasopharynx seems to be transient. The Swedish study evaluated the duration of resistant S pneumoniae in 678 adults and children. The average duration of colonization was 19 days (range, 3-267 days). However, 94% of the individuals had spontaneous disappearance of the resistant S pneumoniae within 3 months of detection. Also, a French study found that 60% of children aged 4 months to 41/2 years carried S pneumoniae in their nasopharynges and that 42% were resistant strains.

The carrier rate of resistant bacteria did not change after treatment with cefpodoxime proxetil or amoxicillin clavulanate. However, 75% of the sensitive strains were eradicated, leaving 75% of the remaining strains resistant. Antibiotic therapy most likely selects out the difficult-to-treat individuals with the worst diseases. Colonization with resistant organisms in the nasopharynx or the adenoids seems to be harder to treat medically, which could result in the need for adenoidectomy.

Resistant bacteria were recovered from the adenoids in children with recurrent or persistent otitis media and signs and symptoms of rhinosinusitis in significant amounts compared with children who had hyperplasia alone. In this preliminary report, the clinical significance of their presence and the role of adjuvant adenoidectomy are undetermined. However, the possibility that the adenoids are acting as a reservoir of infection in these children who are refractory to antibiotic therapy is a concern. Prospective clinical trials are needed to determine the effect of adenoidectomy on the resolution and prevention of further otitis media and/or rhinosinusitis in children who harbor resistant bacterial strains in their adenoids, especially in children younger than 3 years, who appear to be more greatly affected by recurrent ear infections and signs and symptoms of rhinosinusitis.

Accepted for publication December 3, 1999.

Presented in part at the American Society of Pediatric Otolaryngology, West Palm Beach, Fla, May 13, 1998.

Special thanks to Toni Biggs, MTASCP, for her help in gathering the microbiological information. I would also like to thank Karen Krisher, PhD, D(ABMM), Department of Microbiology/Virology, Children’s Medical Center Dallas, and the Department of Pathology, University of Texas Southwestern Medical Center at Dallas.

Corresponding author: John E. McClay, MD, Pediatric Otolaryngology, Department of Otolaryngology–Head and Neck Surgery, University of Texas Southwestern, 5323 Harry Hines Blvd, Dallas, TX 75235-9035.

REFERENCES

7. Paradise JL, Bluestone CD, Rogers KD, et al. Efficacy of adenoidectomy for re-