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Meningitis following Cochlear Implantation: Pathomechanisms, Clinical Symptoms, Conservative and Surgical Treatments

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Abstract

Pneumococcal otogenic meningitis is a rare postsurgical complication that can develop following stapedectomy or after cochlear implantation. The bacterial infection can be fatal in some instances. A recent increase in the incidence of otogenic meningitis among cochlear implant wearers is of concern. The majority of meningitis cases are associated with a 2-component electrode manufactured by one cochlear implant company. The device with the added 'positioner' component has been withdrawn from the market (FDA Public Health Web Notification: Cochlear Implant Recipients may be at Greater Risk for Meningitis, Updated: August 29, 2002, www.fda.gov/cdrh/safety/cochlear.html). Not all cases have been subsequent to otitis media and symptoms have developed from less than 24 h up to a few years after implantation. The purpose of this paper is to review and discuss the pathogenesis, pathology/bacteriology and to elaborate on some clinical features of otogenic meningitis in implanted children and adults. Essential aspects of surgery, electrode design, and cochleostomy seal are discussed. Conclusions are drawn from the available data and recommendations are made for good practice in cochlear implantation and follow-up.

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Introduction

The Food and Drug Administration (FDA) has recently reported about 87 cases of meningitis in patients implanted with the three FDA-approved cochlear implant devices: Advanced Bionics devices (53 cases), Cochlear Corporation devices (33 cases) and MED-EL Corp devices (1 case). A total of 17 deaths have resulted from these meningitis cases (as of October 2, 2002).

According to the FDA, the majority of the Cochlear Corporation and the MED-EL cases had predisposing factors for meningitis unrelated to the implant (e.g. Mondini inner ear deformity, preimplantation history of meningitis).

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Cerobrospinal fluid (CSF) culture results from 21 cases in the United States have shown the following distribution of bacteria: *Streptococcus pneumoniae* (15), *Streptococcus viridans* (2), *Haemophilus influenzae* (3), *Escherichia coli* (1). Although vaccination is usually protective against both pneumococcus and *Haemophilus influenzae,* 2 cases of pneumococcal meningitis and 2 cases of *H. influenzae* meningitis developed after the patient had received the appropriate vaccine.

In some of the reported cases of meningitis in cochlear implant recipients may have had overt or subclinical otitis media prior to surgery or before the meningitis developed. According to the FDA, physicians are encouraged to consider appropriate prophylactic perioperative antibiotic treatment and to diagnose and treat otitis media promptly in patients with cochlear implants.

Since otogenic meningitis is rare in industrial countries, an update of pathogenesis, pathology, bacterial spreading, diagnostic criteria and treatment would appear to be timely, and is provided herein.

Pathogenesis, Pathology and Clinical Symptoms of Otogenic Meningitis

Bacteria can reach the meninges either by hematogenic spread (primary bacterial meningitis) or by extension from nearby infections (secondary bacterial meningitis, e.g. sinusitis, acute or chronic otitis) or by communication of CSF with the exterior (e.g. spontaneous due to meningocele, penetrating injuries, oto- or neurosurgical procedures). Many bacteria can cause meningitis, but *Neisseria meningitidis* (meningococcus), *Streptococcus pneumoniae* (pneumococcus) and *Haemophilus influenzae* type b are the three 'primary pathogens'. Factors such as age, history of head trauma with CFS leak, ear or sinus surgery and immune status may help predict the causative agent.

In a retrospective review of 79 adult patients suffering from otogenic meningitis, 32 patients had acute otitis media (AOM), 29 patients chronic otitis or cholesteatoma and 18 patients a cerebrospinal fluid leak [Barry et al., 1999]. In this study, *Streptococcus pneumoniae* was the most common cause of meningitis complicating acute otitis media (69%) or CSF leak (50%). A review of 4,162 cultures of aspirates from the middle ear of individuals with AOM from the USA [Klein, 1980], Finland and Sweden [Rohn et al., 1980] revealed that the predominant organisms are *S. pneumoniae* (33.9%), *H. influenzae* (20.2%), *Streptococcus pyogenes* (7.2%) and *Staphylococcus aureus* (1.9%). The bacteriology of AOM in infants less than 6 weeks of age differed slightly from that of older children and adults, in that gram-negative bacteria represented a significant percentage of the positive cultures: *S. pneumoniae* (18.3%), *H. influenzae* (12.4%), *S. aureus* (7.7%), *S. pyogenes* (3.0%), *E. coli* (5.9%) [Klein, 1980; tables 1, 2]. The bacteriology of AOM in older children is not significantly different from that of younger children; *H. influenzae* is still a significant pathogen in the older age group [Schwartz and Rodriguez, 1981]. Even though these data are more than 20 years old, this spectrum of bacteria has not changed.

It is likely that AOM results from the entrance of pathogenic bacteria from the nasopharynx into the Eustachian tube and middle ear cleft by direct extension. Rare but serious complications of otitis media are labyrinthitis and meningitis or intracranial empyema. In those complications, the pattern of bacteria causing meningitis is the same as in acute otitis media [Nadol and Arnold, 1987].

It seems that meningitis has recently emerged as an extremely rare but significant cause of morbidity and mortality following cochlear implantation. The onset of meningitis may be in the days following implantation or as long as several years later. Surprisingly only in some of these meningitis cases has there been a clinical evidence of acute otitis media. However, the most common offending organisms were *S. pneumoniae* and *Haemophilus influenzae,* indicating that the infection must have taken the route of the middle ear through the cochlea to the meninges.

Dahm et al. [1994] studied the pathogenesis of labyrinthitis following pneumococcal otitis media in nonimplanted and implanted cat cochleas. Already at that time, the authors stated that 'pneumococcal otitis media is frequent in young children and could lead to labyrinthitis post-implantation'. The authors further stated that in the course of implant surgery, the barrier between the middle and inner ear is broken down by incising the round window membrane or by fenestration of the cochlear wall. The insertion of an electrode array into the scala tympani could provide a pathway for bacteria to enter the cochlea. To assess the risk and methods of minimizing this risk by proper grafting to the cochleostomy around the electrode entry point the authors have used a cat animal model of pneumococcal otitis media. They proved that a properly sealed cochleostomy (connective tissue graft around the electrode) is the best protection from pneumococcal meningitis. The findings indicated that cochlear implantation in general does not increase the risk of labyrinthitis following pneumococcal otitis

Table 1. Review of cultures of aspirates from middle ears of patients with AOM

Organism	Number	Percentage
Streptococcus pneumoniae	1,399	33.9
Haemophilus influenzae	834	20.2
Streptococcus pyogenes	298	7.2
Branhamella catarrhalis	161	3.9
Staphylococcus aureus	80	1.9
Mixed	72	1.7
Others (including gram-negative bacterial infection)	209	5.1
No growth	1,068	25.9
Total	4,121	99.8

From Klein and Teele [1976].

Table 2. Percentage of bacteria in positive AOM cultures

Organism	Percentage
Streptococcus pneumoniae	18.3
Heamophilus influenzae	12.4
Streptococcus pneumoniae and *Haemophilus influenzae*	3.0
Staphylococcus aureus	7.7
Streptococcus pyogenes	3.0
Branhamella catarrhalis	5.3
Escherichia coli	5.9
Klebsiella (Enterobacteriaceae)	5.3
Pseudomonas aeruginosa	1.8
Miscellaneous (gram-negative)	5.3
No growth/nonpathogens	32.0

From Klein [1980].

media, but the authors strongly recommended to use fascia as a graft around the entry point of the electrode (fig. 1a). The risk of postimplantation meningitis was addressed 8 years ago.

Furthermore, several authors have described early or late complications of stapedectomy including fatal meningitis [Sheehy and House, 1962; Clairmont et al., 1975; Wolff, 1964; Palva and Kaerjae, 1972; Benitez, 1977]. Moon [1970] reported on 48 fistulas (2.3%) in 2,091 patients who underwent stapes surgery. He called attention to the concept of primary and secondary fistulas. A primary fistula is one which is considered to have existed from the time of the original procedure. This group includes cases with continuous fluid leaks from the co-

chlea vestibule to the middle ear. A secondary fistula is one which occurs after a tissue seal closure has existed for a period of time, either by atrophy of the connective tissue or by bio-incompatibility of the prosthesis material.

Similar to undiagnosed posttraumatic or spontaneous CSF leakage so-called 'silent fistulas' may occur following stapedectomy or stapedotomy without any clinical symptoms. Many stapes surgeons remember a perilymph fistula discovered during revision surgery in patients with good sensorineural hearing and no vestibular symptoms [Althaus, 1981]. Nadol [2002] recently reported on histopathologic findings in temporal bones from individuals who had undergone cochlear implantation. Here is demonstrated a case where a cochleostomy originally sealed with fascia had changed to an extremely thin membrane infiltrated with inflammatory cells. Bacterial transport through a membrane across the round window has recently been demonstrated [Paparella et al., 2002]. This observation suggests that the pathways for infection from the middle ear to the meninges or brain are similar to the path set up following AOM after stapedotomy or stapedectomy: bacteria may migrate through a weak or imperfectly sealed cochleostomy into the labyrinth. The organisms infiltrate the cochlear turns along the electrode, entering Schuhknecht's bony channels and following perineural and/or perivascular pathways into the internal auditory canal. This spreading is identical to that of the classic routes of infection for labyrinthitis and otogenic meningitis following acute or chronic otitis media [Friedmann and Arnold, 1993].

Electrode development in the last few years has seen the emergence of different electrode concepts. Temporal bone studies have shown that the currently existing concepts of the larger, stiffer and preshaped electrodes are responsible for more basilar membrane perforation and spiral lamina fractures [Richter et al., 2002; Roland et al., 2002; Gstoettner et al., 2001; Fayad et al., 2002; Sutton et al., 1980]. The continuous pressure of an electrode which is preshaped, too thick or excessively stiff and/or the pressure of a positioner transmitted to the surface of the modiolus might cause necrosis of the tissue (locus minoris resistentiae) facilitating the central spread of infection.

If during a common cold bacteria migrate through the Eustachian tube into the middle ear of an implanted individual and the sealing of the cochleostomy is incomplete, then these bacteria might enter the inner ear, following the fluid pathways in Schuhknecht's channels and the auditory canal to the meninges [Schachern et al., 1992].

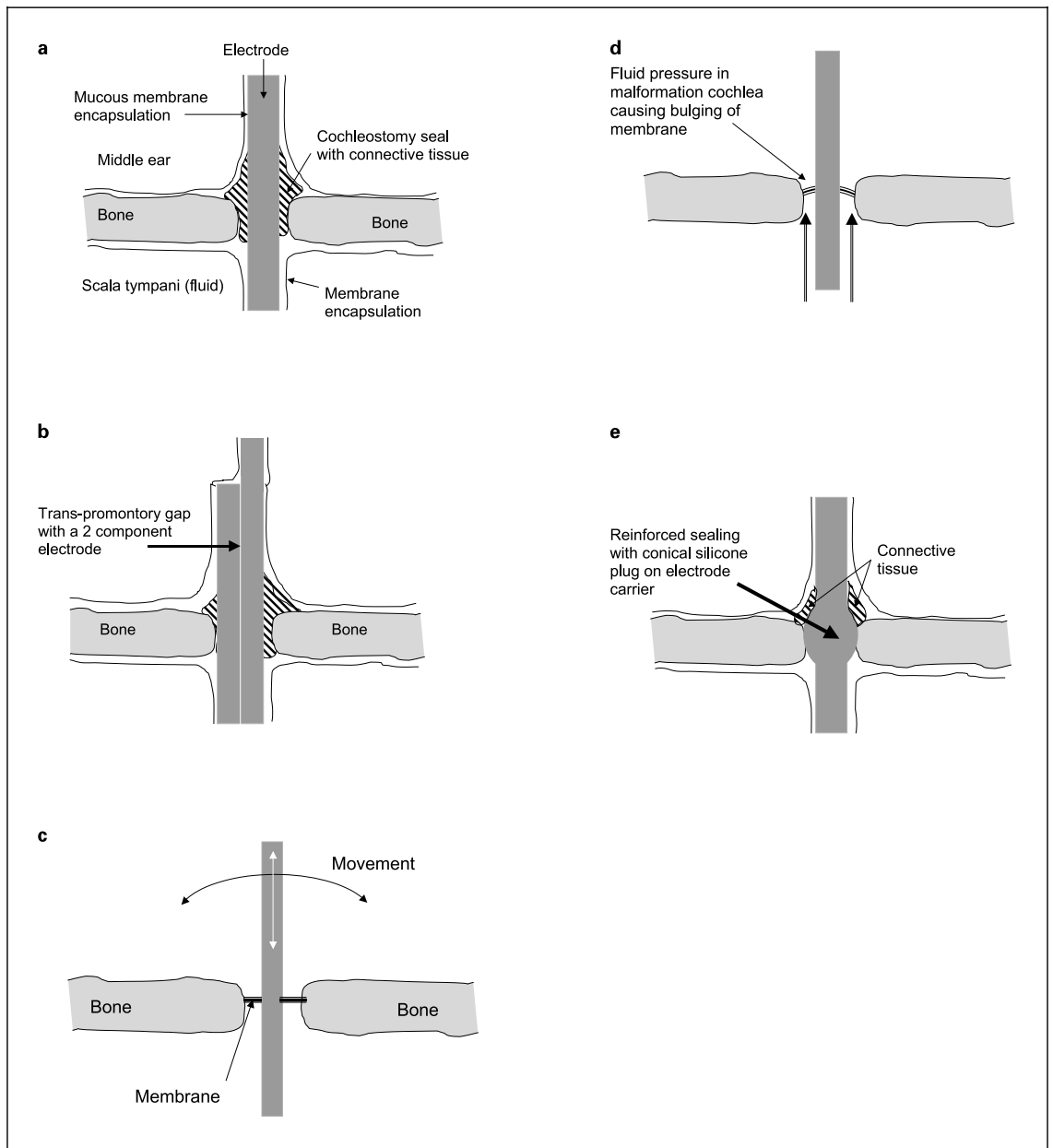Surprisingly, not all cases of meningitis following cochlear implantation are associated with AOM. However,

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Fig. 1. a–e Appearance of the sealed cochleostomy site under different conditions with a normal electrode and the 2-component electrode.

the most common offending organism was *S. pneumoniae*. This raises the pathetical question whether in those cases the contamination might have been hematogenic with an intracochlear mini-abscess or empyema formation on the necrosed tissue (from continuous pressure on the surface of the modiolus), which then spread intracranially via the internal auditory canal. This could also explain why in some cases the septic process was so aggressive and did not respond to treatment and why the mortality was so high in cases implanted with electrodes with a positioner.

Furthermore, it is possible that complete sealing of the two silicone elements, namely the electrode and the positioner is hard to achieve. With incomplete sealing, a (fluid) leak might be persistent, allowing bacteria from the middle ear to enter the cochlear fluid space. The two sili-

cone components squeezed together through the cochleostomy are the unique feature of the electrode in question. One component has a concave profile on one side. The two parts originate in the middle ear, traverse the cochleostomy and continue in the scala tympani. Any silicone-to-silicone pressure would effectively prevent tissue growth between the two components (fig. 1b). Encapsulation of the electrode should take place around the dual system. However, a transpromontory gap will persist between the components. A de facto silent perilymphatic fistula may be created. Packing of such a fistula with additional connective tissue may be unrealistic since no room may be available between the two components of the electrode (fig. 1b).

Especially in malformations of the cochlea CSF fluid pressure may be exerted against the thin membrane encapsulation causing rupture. A silent perilymphatic fistula may occur such as sometimes found [Goodhill, 1981] or after stapedectomy. Finally it should be noted that the movement of an electrode surrounded by just a very thin layer of connective tissue sealing the cochleostomy could induce rupture, creating a fluid leak (fig. 1c, d).

As a further possible transmission pathway an open aqueductus cochleae must be considered. The cochlear aqueduct originates internally in an orifice on the medial wall of the scala tympani near the origin of the latter and terminates on the undersurface of the petrous portion of the temporal bone in the superior border of the jugular fossa, between the jugular bulb and the inferior surface of the internal auditory canal [Anson et al., 1965]. The question of patency of the cochlear aqueduct in humans has long been a subject of discussion. A number of experiments on animals have demonstrated that this channel is open in certain mammals [Neiger, 1968]. The cochlear aqueduct is larger in animals than it is in man, and although this passage is open in the human fetus and young children, it usually is filled with a network of arachnoidal connective tissue in the adult [Bast and Anson, 1949]. A temporal bone study by Włodyka [1978] indicates that this aqueduct is open in 75% of individuals under the age of 21, but in only 32% of the elderly. It is possible that an abnormally large or immature cochlear aqueduct, as described by Igarashi and Schuhknecht [1962], is an important route of pressure transduction from CSF to the cochleostomy seal as well as a route of bacterial migration to the meninges. Since the cochlear aperture of the aqueduct to the scala tympani is topographically closer to the cochleostomy than the internal auditory canal, the route of infection through the cochlear aqueduct must be seriously considered in such cases

where it offers an open fluid connection to the subarachnoidal space, e.g. in certain malformed cochleae. This may be part of the explanation why meningitis occurs more frequently in cases of cochlear malformation [Tom et al., 1992; Stevenson et al., 1993; Ohlms et al., 1990].

Clinical Symptoms and Signs

A prodromal respiratory illness, sore throat or earache often precedes the fever, headache, stiff neck and vomiting that characterise acute meningitis. Adults may become desperately ill within 24 h and children even sooner. In older children and adults, changes in consciousness progress through irritability, confusion, drowsiness, stupor and coma. Seizures and cranial neuropathies may occur. Dehydration is common and vascular collapse may lead to shock and the Waterhouse-Friderichsen syndrome, especially in meningococcal septicemia. Hemiparesis and other focal deficits can result from cerebral infarction, but their early occurrence is relatively uncommon in meningitis rather suggesting a brain abscess.

In infants between 3 months and 2 years of age symptoms and signs are less predictable. Fever, vomiting, irritability, convulsions, a high-pitched cry and bulging or tight fontanelles are common; stiff neck may be absent. In infants and young children, subdural effusions may develop after several days. Typical signs are seizures, persistent fever and enlarged head size. Subdural taps through the coronal sutures detect the high protein content in the subdural fluid.

Because acute bacterial meningitis, especially streptococcal, can be lethal within hours, accurate diagnosis and swift treatment are paramount. When otogenic bacterial meningitis following cochlear implant surgery is suspected, antibiotics should be given immediately without waiting for diagnostic test results. Because specific bacteriologic diagnosis is important, every effort should be made to obtain spinal fluid for bacteriologic culture and sensitivity testing.

Prophylaxis and Treatment of Otogenetic Meningitis

Perioperative Antimicrobial Prophylaxis for Cochlear Implantation
The anatomical basis for infection is a connection between the CSF and a colonized middle ear cavity. This can be temporary or occasionally persistent.

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The microorganisms that cause ascending meningitis under such conditions are predominantly pneumococci, followed by other streptococci, *H. influenzae* and less frequently, other organisms that may colonize the middle ear.

Active immunization against *S. pneumoniae* (conjugated for children and 23-valent polysaccharide vaccine for adults) and *H. influenzae* type b (conjugated vaccines for children) may benefit any prospective implant candidate as well as patients with an implant in place. Such timely immunizations will certainly decrease the 'background' incidence of meningitis in this population. Recommendations concerning various licensed age-appropriate vaccines are detailed in the FDA notification (www.fda.gov/cdrh/safety/cochlear.html).

Considerations regarding perioperative antimicrobial prophylaxis need to address two aspects: one is a gram-positive skin flora during implantation of the implant package, the other is control over middle ear flora. Prophylaxis for postoperative wound infections is routinely achieved by short-term administration of a first- or second-generation *cephalosporin*. At centers where community-acquired methicillin-resistant *S. aureus* are observed or a patient has been previously hospitalized, *vancomycin* may be given (15–20 mg/kg/dose, over 1 h × 1; adults 0.5–1 g), with the infusion completed 1 h prior to surgery. However, penetration and the pharmacokinetics of *vancomycin* have not been studied in middle ear fluid.

Implantation should be avoided in the presence of middle ear fluid. If middle ear fluid is found at the time of cochlear implant surgery, special care should be taken to reduce any risk of infection. The use of high volume irrigation, administration of local, topical antibiotics into the middle ear space at the time of surgery and a more prolonged postoperative antibiotic regimen are highly recommended. This may cause difficulties in scheduling surgery in some young children. The bacteria commonly found in middle ear fluid in the presence of an intact tympanic membrane are *S. pneumoniae, H. influenzae* (predominantly nontypable) and *Moraxella catarrhalis.* A regimen that would cover this flora including most penicillin-resistant pneumococci would be *ceftriaxone* 50–75 mg/kg (adults 1–2 g). It should be given about 3 h prior to insertion to allow time for penetration. *Ceftriaxone* would also cover skin flora in areas where community-acquired methicillin-resistant *S. aureus* strains are not yet of concern.

The short-term oral administration of *amoxicillin/clavulanate* with the higher dose of *amoxicillin* of 80 mg/kg/day, or *rifampin* 20 mg/kg × 2 q 24 h, has also been suggested [Dagan et al., 2000]. Oral administration may however be unreliable prior to surgery, and the influence of other perioperative medications on absorption is unknown. Based on these considerations, oral perioperative antimicrobial prophylaxis is not recommended.

If there is a tympanic membrane perforation, the middle ear may be colonized in addition to skin flora with *Pseudomonas aeruginosa* or other gram-negative organisms. The additional prophylactic antimicrobial agents in such cases should be tailored to sensitivity patterns and administered at doses that would cover CNS infections.

The accepted principles of perioperative antimicrobial prophylaxis are to provide adequate antimicrobial tissue and fluid concentrations at the time of intervention. Although tempting, perioperative antimicrobial prophylaxis should not be extended beyond that time.

Choice of Empiric Antimicrobial Agents for Cochlear Implant Patients Suspected of Having Meningitis

The spectrum of organisms that cause ascending meningitis is believed to be similar to that causing nonotogenic meningitis in children, apart from the predominance of pneumococci and the possibility to encounter strains of nontypable *H. influenzae.*

The choice of empiric antimicrobial treatment of meningitis therefore does not need to deviate from the usual recommendations, providing tympanic membrane perforation and grommets are absent. These recommendations apply to the spectrum of microorganisms found in patients over 3 months of age. Cochlear implants are currently not used earlier.

These current recommendations for empiric treatment of meningitis are:

Vancomycin given at 60 mg/kg/day, infused over 1 h every 6 h (adults: 2 g/day divided q 6 h), in combination with cefotaxime at 200–300 mg/kg/day every 8 h (adults: 8–12 g/day every 4–6 h), or ceftriaxone given at 100 mg/kg/day every 12 h (adults up to 4 g/day divided q 12 h).

In cases where the tympanic membrane is perforated (acutely or chronically), the possibility of colonization of the middle and inner ear with *P. aeruginosa* has to be considered. *Meropenem* or *cefepime* may be given empirically instead of *cefotaxime* or *ceftriaxone.* Experience in the treatment of ascending meningitis due to *P. aeruginosa* with these agents is limited and treatment has not always been successful. An optimization of dosage and regimen (e.g. longer infusion times) of these agents, that have both been studied for nonpseudomonas meningitis, should be discussed with an expert in infectious diseases. The doses studied in children for the treatment of conventional

meningitis were 120 mg/kg/day every 8 h for *meropenem*, and 150 mg/kg every 8 h for *cefepime*, which currently lacks regulatory approval for the treatment of meningitis. An *aminoglycoside* should be added if the CSF gram stain shows gram-negative organisms or if growth of pseudomonas is confirmed.

Single high-dose administration of aminoglycosides has not been studied for the treatment of meningitis and should be avoided. *Ceftazidime*, another possible option (always in conjunction with vancomycin as with the others), would be given at 150 mg/kg/day every 8 h.

In adults, during pregnancy and in immunocompromised patients, the possibility of a nonotogenic meningitis caused by *Listeria monocytogenes* should be considered and *ampicillin*, 400 mg/kg/day every 6 h (adults: 12 g/day every 4 h) added.

Surgical Treatment

If a cochlear implant patient presents with meningitis, the most likely cause can be assumed to be otogenic. The pathogenesis has been discussed above.

Examination of the ear can show a bland tympanic membrane, but with increased vascularization and thickening of the tympanic membrane. Classic acute otitis media with a bulging tympanic membrane is also possible. The latter is an indication of an acute inflammatory process of the middle ear cavity. With the help of imaging techniques, such as high-resolution CT or MRI, soft tissue masses in the region of the mastoid and the middle ear can be detected as well as osteolytic areas in the region of the cochlea.

A middle ear and mastoid revision operation should be performed in any event, both to drain the infected middle ear and mastoid and to exclude a fistula. If there are signs of inflammation in the region of the mastoid or middle ear, the bone should be removed generously down to the sigmoid sinus and, if applicable, down to the dura of the middle fossa.

All inflammatory granulations of the middle ear cavity should be carefully removed. In order to clearly identify outflow of fluid from the cochlea, the cochleostomy where the active electrode array enters the cochlea must be cleaned. Inflammatory granulations found in this region should be removed. If purulent secretions exit from the cochleostomy next to the electrode array, the surgeon must decide whether the electrode array should be removed and a partial labyrinthectomy performed. The latter is necessary in order to remove the focus of the menin-

gitis. Removal of the electrode and a labyrinthectomy must also be performed if the high-resolution CT scans show a clear osteolytic destruction of the labyrinth. In such cases, the whole middle ear should be obliterated with a big flap of the temporalis muscle or a free muscle transfer.

If the surgeon identifies only a few inflammatory granulations in the middle ear but a perilymph fistula in the region of the cochleostomy, it may be sufficient to retract the electrode only partially, inject highly effective antibiotics into the cochlea, reinsert the electrode array into the cochlea and seal the cochleostomy carefully with connective tissue. One can try to preserve the functionality of the device by initiating a high-dose parenteral antibiotic regimen (as described above) over several days until the symptoms of meningitis disappear. In any case, the mastoid should be drained for a couple of days, and local antibiotics, selected on the basis of culture and sensitivity testing, can be administered through a drainage tube.

In principle, the treatment of otogenic meningitis follows classic principles of this pathogenesis. The decision whether the electrode should be left in place or removed depends on the severity of the disease, the intraoperative findings and on the individual experience of the surgeon.

Conclusions

A possible reason for otogenic meningitis following cochlear implantation is a fluid leak around the cochleostomy caused by: (a) incomplete cochleostomy packing, (b) a mismatch between cochleostomy and electrode diameter, (c) rupture of the cochleostomy seal as a consequence of electrode movement or of an increased CSF fluid pressure (common with cochlear malformation such as common cavity) and (d) by the use of a two silicone component electrode or any other electrode that features a gap or a channel through the cochleostomy which cannot reliably be sealed with connective tissue.

At least in some instances the connective tissue applied between the electrode and the cochleostomy opening atrophies slowly and a very thin connective tissue membrane will result (fig. 1c). In case of bacterial otitis media, this thin atrophic membrane does not provide robust biologic protection against bacterial passage. Spread of bacteria through the remaining labyrinthine fluid spaces to the subarachnoid space may be possible.

Complete packing of the cochleostomy after insertion of the electrode, sufficient perioperative antibiotic prophylaxis and secure fixation of the electrode to exclude

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postoperative movement are important measures for the prevention of otogenic meningitis.

It is not known with certainty whether or in which cases the cochleostomy opening closes completely around the electrode through bone regrowth. Hence, it is advantageous to match cochleostomy size and electrode size and close the cochleostomy opening with a conical shaped electrode shaft before adding additional connective tissue for a secure seal (fig. 1e).

Cochlear implant users and their relatives should know that middle ear infection entails a small chance of bacterial progression to the meninges. Thus, an experienced otologist or the surgeon should be consulted in case of an earache.

The cochlear implant user and his/her relatives should be informed about the risk and past occurrence of meningitis after cochlear implantation.

Upon the first symptoms of meningitis, the physician should start antibiotic treatment in accordance with the guidelines described above and immediately consult an experienced otosurgeon. To exclude osteolysis of the cochlea and to identify soft tissue masses in the middle ear and mastoid, high resolution CT and MRI must be performed. In case of substantiated suspicion of otogenic meningitis in a cochlear implant patient a surgical revision of middle ear and mastoid is mandatory. In case of a purulent labyrinthitis the intracochlear electrode has to be removed and a labyrinthectomy must be performed with obliteration of the middle ear.

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