Cochlear Implant Infections

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Introduction

Infections associated with cochlear implantation surgery are uncommon but can have devastating effects. Persistent infection may require removal, with or without device replacement, causing significant patient morbidity and expense. This chapter will review the epidemiology, microbiology, diagnosis, treatment, and prevention of cochlear implant infections.

Cochlear Implant Surgery

A cochlear implant is a prosthesis that provides direct electrical stimulation to the auditory nerve in order to evoke the perception of sound. It is the gold-standard treatment for deafness in the severe-to-profoundly hearing impaired where a hearing aid does not provide adequate speech recognition for daily communication. The surgery involves a mastoidectomy approach that allows access to the middle ear, and the implantation of a long, thin electrode array into the scala tympani of the cochlea (Fig. 8.1). This electrode is attached to an implant body, known as the "receiver stimulator," that is placed beneath the temporalis muscle (in the subperiosteal plane), posterosuperior to the pinna. These components are fully implanted, with no direct physical communication through the skin. This receiver-stimulator communicates through a radiofrequency coil to the externally worn "speech processor." The processor has a microphone(s) to detect sound and extracts components of the acoustic signal pertinent to the understanding of speech. These signals are encoded as a series of electrical pulses. This code is transmitted to the receiver-stimulator across the skin by the radio-frequency link, then via the electrode into the cochlea where electrical stimulation of the inner ear is relayed via the auditory nerve to the brain.

Infections in cochlear implantation relate to the surgical incision, the implant body or electrode, including the intracochlear electrode, and meningitis. The risk of meningitis is increased partly because of the potential connection between the cochlea and the meninges. The cochlea is filled with perilymph and communicates with the cerebrospinal fluid (CSF) via the

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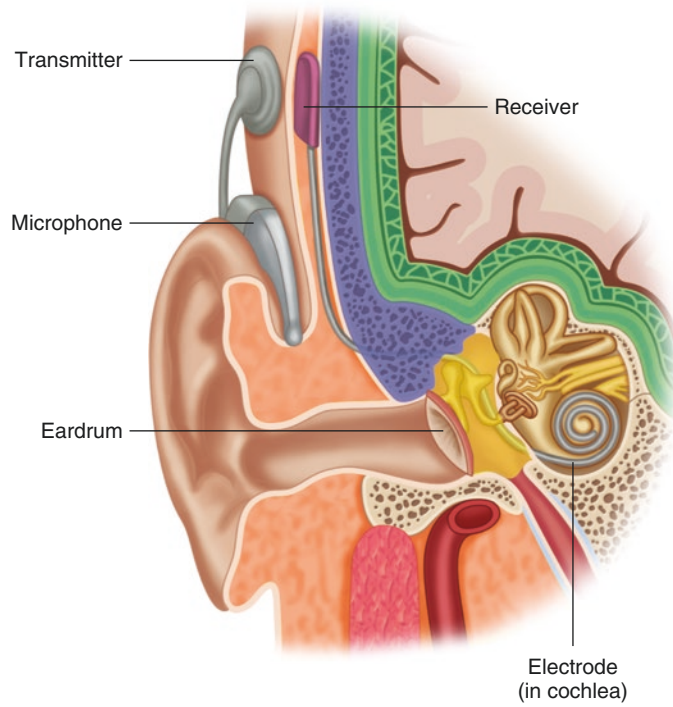

Fig. 8.1 Anatomy of the ear with a cochlear implant. Yellow = middle ear; blue = temporal bone and mastoid; green = meninges

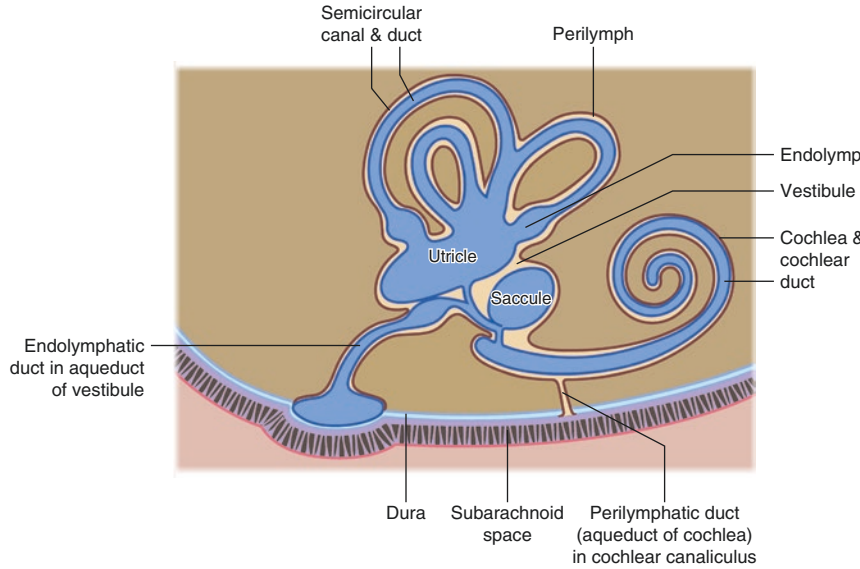

Fig. 8.2 Relationship of the cochlear aqueduct to the central nervous system. Adapted from O'Rahilly R, Müller F, Carpenter S, Swenson R. *Basic Human Anatomy*: *A Regional Study of Human Structure* (online textbook developed at Dartmouth Medical School), www.dartmouth.edu/~humananatomy. With permission

cochlear aqueduct. The cochlear aqueduct, which is located in the scala tympani near the round window, is very narrow and of variable patency in humans [1] (Fig. 8.2). The cochlea can also communicate with the CSF in a dysmorphic cochlea [2], or if the surgery causes significant trauma to the medial (modiolar) wall of scala tympani.

Epidemiology

Infections associated with cochlear implantation occur in 1.7–4.1% of cases and include wound infection, otitis media and mastoiditis, implant body infection, cochlear infection, and meningitis [3–5]. Infections may occur at any point after surgery, but the majority of infections occur >30 days postoperatively, often months to years after surgery. Table 8.1 notes the overall incidence and mean time of onset for various infections. A history of chronic otitis media may increase the risk of post-implantation infection. Similarly, patients with a history of meningitis, bacterial labyrinthitis, or cochlear dysmorphism (which may have caused the hearing loss necessitating the cochlear implant) are more at risk of developing meningitis post-operatively compared with patients who have no history of meningitis [6].

Microbiology

Cochlear implant surgery is typically classified as a "clean" operation and most of the microorganisms responsible for infection in cochlear implant surgery are skin flora, *Staphylococcus aureus* in particular. Upper respiratory tract microorganisms such as *Streptococcus pneumoniae* and *Haemophilus influenzae* may be the cause of cochlear implant infections associated with otitis media or meningitis [7]. Table 8.2 lists pathogens commonly cultured in various types of cochlear implant infections.

Cunningham et al. evaluated reported wound cultures from 15 of 30 patients with infections and found that most (73%) were due to *S. aureus* (11 cases); other pathogens included Group A streptococci (1) and Gram-negative bacilli (3, *Escherichia coli*, *Klebsiella*, *Pseudomonas*) [5]. Hopfenspirger et al. reported 26 organisms grew in cultures from 22 wound infections, and the major pathogens included *S. aureus* (12 cases), *Pseudomonas* (5), coagulase-negative staphylococci (4), *Alcaligenes* (2), and *Candida albicans* [8]. Neither Cunningham nor Hopfenspirger evaluated cultures by time of onset of infection. Gawecki et al. reviewed 19 late-onset skin flap and implant-related infections, and of 16 with positive cultures, 69% were due to *S. aureus*, either alone (50%) or in combination (19%) with Gram-negative bacilli such as *Pseudomonas*, *Klebsiella*, *Enterobacter* [9]. Zawawi et al. reviewed the literature and found 43 cases of postoperative mastoiditis, with mean onset 17.2 months; results of cultures are listed in Table 8.2 [10]. Reefhuis reviewed 26 cases of meningitis following cochlear implantation in 4262 children and found that 65% of meningitis cases developed >30 days postoperatively and all of these late onset cases were due to *S. pneu-*

Table 8.1 Incidence and time of onset of cochlear implant infections

Study	Total # patients (% pediatric)	Study years	Incidence of infection (major)	% early onset (≤30 days)	Mean onset (months)
Cunningham [5]	734 (37%)	1993–2002	4.1%	27%	11.2
Hopfenspirger [8]	268 (100%)	1990–2007	8.2%	27%	7.3 for late-onset cases
Zawawi [10]	N/A (100%)	2000–2013	N/A (all mastoiditis)	N/A	17.2
Reefhuis [7]	4262 (100%)	1997–2002	0.6% (all meningitis)	35%	11.8 for late-onset cases
Yu [4]	241 (N/A)	1990–2000	1.7%	25%	24 for late-onset cases
Gawecki [9]	1076 (59%)	1994–2013	1.8%	none	33.2

Cunningham [5], Hopfenspirger [8], Yu [4], Gawecki [9] reviewed wound and implant-related infections; Zawawi [10] reported mastoiditis cases, Reefhuis [7] reported meningitis cases.

Table 8.2 The microbiology of cochlear implant infections

Surgical site infections	Otitis media/mastoiditis	Meningitis
Staphylococcus aureus	*Streptococcus pneumoniae*	*Streptococcus pneumoniae*[a]
Coagulase-negative staphylococci (indent)	*Streptococcus pyogenes*	*Haemophilus influenzae*[a]
Streptococci	*Staphylococcus aureus*	*Acinetobacter baumannii*
Pseudomonas aeruginosa	*Pseudomonas* species	*Escherichia coli*
Escherichia coli	*Haemophilus influenzae*	*Enterococcus* species
Klebsiella pneumoniae		
Alcaligenes xylosoxidans		
Candida albicans		

Data from several series of cochlear implant infections [5, 7, 8, 10]
[a]Data from Reefhuis, et al. [7] Late onset meningitis cases (>30 days post-implant) were all due to *Streptococcus pneumoniae* and *Haemophilus influenzae*, while early onset cases were caused by one of the five organisms listed

moniae or *H. influenzae* [7]. Early onset cases (<30 days postoperatively) also included enterococci and Gram-negative bacilli.

Clinical Manifestations and Management

Operative Management and Wound Infection

Surgical site infections are the most common infectious complications after cochlear implant surgery. However, as the surgical technique has improved, the rate of wound infections has decreased. Early cochlear implant incisions had an inverted "C" shape that commenced behind the ear, curved upward and backward and then inferiorly [11]. The concept was to expose the region where the implant body would sit. This approach was associated with a high incidence of wound infection, likely due to interference with the occipital blood supply [12]. More recently, incisions have become more linear, or curvilinear, running essentially vertically, in order to avoid disruption to the scalp circulation [12, 13]. Disruption of the scalp blood supply has been reduced further by the introduction of shorter ("minimally invasive") incisions. Another important concept in designing an incision is that it should not cross the implant body, and to facilitate this, incisions should be close to the postauricular sulcus. The development of surgical access that reduces the risk of infection has been helped considerably by improvements in the design of the implant body, which is now thinner, smaller and requires less bone-drilling to seat the device in the cortical bone of the cranium. These revised surgical requirements have subsequently reduced the size of the surgical incision. Figure 8.3 illustrates historical and current types of surgical incisions.

Patients with any active infections, such as otitis externa or otitis media, should be treated and cleared of these infections prior to cochlear implant surgery. At the time of surgery, prophylactic intravenous antibiotics should be started within 1 h prior to the surgical incision (within 2 h for antibiotics with long half-lives such as intravenous vancomycin). The major risk is of wound contamination with *S. aureus* [5, 8], so the antibiotic chosen should cover staphylococci. There is no consensus on the optimal prophylactic antibiotic for cochlear implant surgery, nor whether the type of prophylaxis should differ for children and adults. A 2010 policy statement from the American Academy of Pediatrics discussed prophylaxis but did not make a specific recommendation [14]. The guidelines published in 2013 by a group of U.S. national organizations regarding antibiotic prophylaxis for various types of surgeries did not specifically mention cochlear implant surgery, but recommended a single preoperative dose of either cefazolin or cefuroxime for clean procedures in the head and neck involving an implant [15]. These guidelines noted that a single preoperative dose of vancomycin may be added to the chosen prophylactic regimen for patients known to be colonized with methicillin-resistant *S. aureus* (MRSA). A first generation

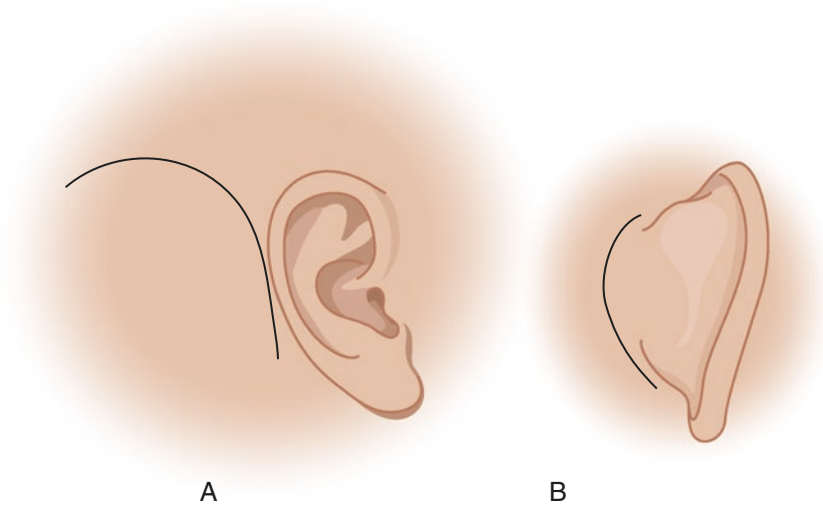

Fig. 8.3 Incisions used in cochlear implant surgery, past and present. (**a**) This is a historical incision, while (**b**) represents the current type of cochlear incision, which is smaller, straighter, and closer to the postauricular sulcus than (**a**)

cephalosporin, such as cefazolin, is effective against methicillin-sensitive staphylococci. This was the antibiotic given as prophylaxis in 83% of 98 cochlear implant surgeries (1991–2005) in a series by Hirsch et al.; 15% of the patients were children [16]. No patient developed a major early (≤30 days) postoperative infection and only one patient developed a minor infection (incisional cellulitis). For pediatric patients in particular, some surgeons prefer to use cefuroxime, since this agent covers common otitis pathogens such as *S. pneumoniae* and *H. influenzae* in addition to *S. aureus*. For patients colonized with MRSA, an antibiotic such as intravenous vancomycin with activity against MRSA should be included in the prophylactic regimen (e.g., vancomycin plus cefuroxime, each given as a single dose preoperatively and started in the appropriate window of time as discussed above). Patients with dermatological conditions such as seborrheic dermatitis or psoriasis, where excessive scaling of the skin prevails, should be treated aggressively for their skin conditions prior to surgery.

During surgery, the cutaneous and the muscle/periosteal incisions are off-set, so that a wound infection will be less likely to track down to the implanted device [17]. Similarly, each of these layers is closed separately at the end of the procedure. This provides a layer of vascularized tissue (i.e., fascia, muscle) between the skin and the implant body. Subperiosteal dissection (for the placement of the implant body) should be no more extensive than required, to reduce the risk of a subperiosteal hematoma developing as this will increase the risk of a wound infection. Similarly, a compression bandage ("mastoid dressing") must be applied to reduce the risk of a subperiosteal collection developing and becoming infected. In this regard, it is important to appreciate the posterior and superior extent of the implant body. To provide compression over the implant, the mastoid dressing needs to be more extensive than that applied in conventional mastoid surgery.

Surgical site infections may present as a stitch abscess, localized cellulitis, or infection of the implant body. Provided that the precautions outlined above have been undertaken, a stitch abscess or localized cellulitis is unlikely to lead to a spread of infection to the receiver-stimulator. A stitch abscess is treated along conventional lines, with the removal of an infected stitch and oral antibiotic therapy to cover staphylococci as needed. A localized area of cellulitis may also respond to oral antibiotics, but more extensive cellulitis or systemic symptoms will require admission to hospital and intravenous antibiotics.

Fluctuance over the implant heralds an infection of the receiver-stimulator, and this may not present until several weeks after surgery. The patient may not complain of discomfort or pain and is usually systemically well. If the ear is discharging, a sample should be sent for microbiology to facilitate targeted therapy. One needs to exclude mastoiditis, and although this does not usually co-exist with an infection of the implant body, it should be considered. Computerized tomography (CT) imaging in the first 2–3 weeks after surgery may be of limited value because the mastoid will be filled with blood. Therefore, clinical assessment is of greater importance in diagnosis. Mastoiditis may be anticipated if the patient is systemically unwell, or the focus of the fluctuance and/or post-auricular discharge is not in the vicinity of the implant body. The presence of pus in the middle ear (rather than blood) would confirm the diagnosis of mastoiditis, as would a mucopurulent discharge from a middle ear ventilation tube. The management of implant body infection and mastoiditis is considered in greater detail in the following sections.

Implant Body Infection

Infections of the implant body occur most frequently in the perioperative period, but may occur at any time in the life of the implant. Refractory infections are likely to involve biofilms on the surface of the implant: biofilms have been demonstrated on several devices removed because of persistent infection [18, 19]. The likely cause of the infection will depend on the circumstance. Perioperative infections are thought to arise from contamination of the implant body at the time of surgery by cutaneous pathogens, and *S. aureus* is the most common cause of surgical site infections. Anything that causes a hematoma in the vicinity of the implant body postoperatively, such as poor compression over the implant or perioperative anticoagulation, will increase the risk of a postoperative infection. Late-onset infections may occur following head trauma involving the implant. The hematoma may be initially sterile and later become superinfected. An extensive hematoma must be drained, but compression may be sufficient to treat a small, uninfected hematoma and thus, compression is the preferred option early after surgery. Drainage may involve either aspiration (in a sterile field), or surgical incision and drainage. The aspirate or surgical drainage sample should always be sent for microscopy and culture. If a new incision is to be made, then this should not overlie the implant body. If an infection is found, the wound will always need to be opened and explored.

Implant body infection may present as a relatively painless swelling over the implant, with or without a purulent discharge from the surgical incision in the vicinity of the prosthesis [20]. In some cases, the patient's main complaint is that their transmitter does not fit correctly, or that it is constantly falling off. When wounds in these types of infections are surgically explored, granulation tissue is typically found surrounding the device.

An infected implant is notoriously difficult to treat. Patients will often have little to no improvement with antibiotic treatment. Surgical exploration is required, and if, as anticipated, there is granulation tissue on the implant device, the implant body should be removed. This is because direct washing of an infected device is ineffective at removing biofilms and therefore at eliminating infection [19]. The electrodes are left within the cochlea until re-implantation, which may be undertaken several months later. At this time both the implant body and the intracochlear electrode can be replaced.

Studies on explanted prostheses have shown evidence of biofilms (particularly *S. aureus*) within the depressed areas on the receiver/stimulator that most likely to act as a reservoir [19]. These observations have led to revisions of implant design, minimizing the risk of further infections.

Extrusion of a cochlear implant is a special case. This presents as a gradual breakdown of the skin, revealing the implant beneath (Figs. 8.4 and 8.5). This condition is associated with excessively thin or poorly vascularized skin, and will often break down along an incision line when the

device has been placed immediately beneath this. It may also occur when there has been excessive pressure across the skin between the antenna on the receiver-stimulator and the coil of the speech processor. There is typically no granulation tissue. The implant might be salvaged by rotating a skin flap over the implant and treating with a course of antibiotics, but this approach is not always effective in salvaging the implant. Geraghty et al. reported three adults with cochlear implants who developed device exposure; one had postoperative wound dehiscence that was successfully treated with flap rotation, but two others failed flap coverage and antibiotic treatment and the implant was removed [21]. Gawecki et al. evaluated the experience of 632 children and 444 adults with cochlear implants placed between 1994 and 2013 and found that a major skin flap complication, including some with device extrusion, occurred in 2% of children and 1.4% of adults [9]. Attempts to treat with antibiotics and primary wound closure failed; rotational skin flaps succeeded in 50% of the cases. Parkins et al. reported seven cases of threatened device extrusion out of 74 cochlear implants (9.4%) performed in their center over a 13-year period. Four of these seven were in patients who had undergone a "magnet upgrade" procedure. They reported successful rotational flaps in two of the three single surgery device extrusions, and one of the four "magnet upgrade" patients [22]. Hoffman et al. described a case of partial migration of the electrode array from the cochlea resulting in loss of speech perception with no overlying skin changes. This was able to be managed with re-opening of the cochleostomy and replacement of the existing electrode array with a resultant return to preoperative function [23].

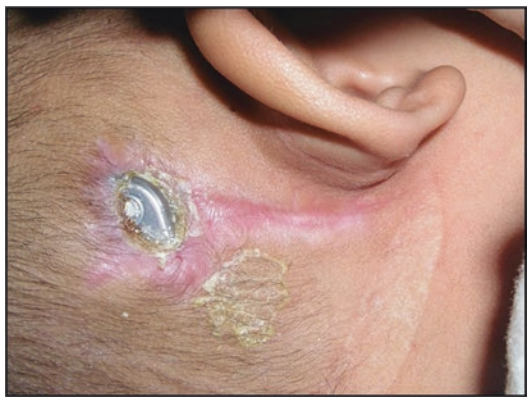

Fig. 8.4 Cochlear implant receiver exposure with minimal evidence of active infection. Photograph courtesy of Dr. Robert Briggs, University of Melbourne, Royal Victorian Eye & Ear Hospital, Melbourne, Australia

Otitis Media

Otitis media is associated with a potential risk of meningitis when it occurs in an implanted ear. However, acute otitis media is common in

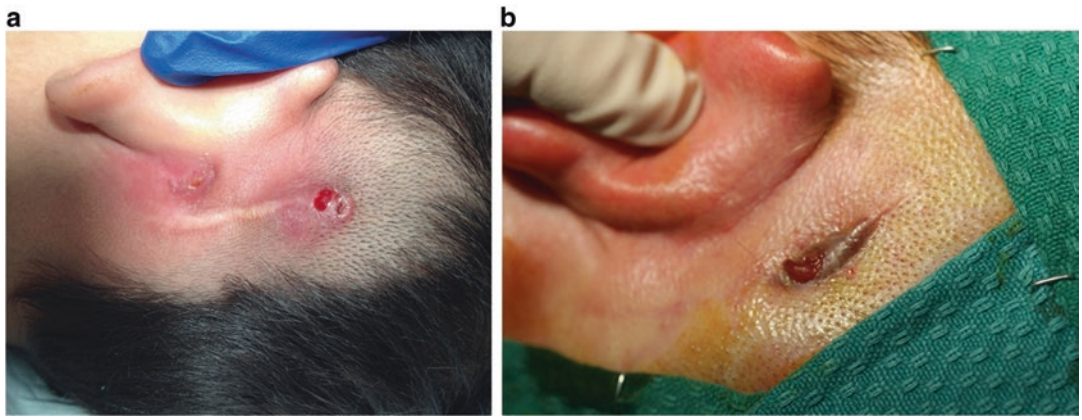

Fig. 8.5 Device exposure with chronic active infection in two different patients. Photographs courtesy of Dr. Robert Briggs, University of Melbourne, Royal Victorian Eye & Ear Hospital, Melbourne, Australia

younger children and seldom causes complications in children with cochlear implants. Uncomplicated otitis media in children with a cochlear implant is not an emergency. There is a lower threshold for prescribing oral antibiotics than usual; we caution against the "wait and watch" approach advocated for otitis media seen in the wider community [24]. Once the infection has settled the associated middle ear effusion is not a risk to the implant, and is treated conventionally, with a ventilation tube if it persists for 3 months or more. The effusion may however change the electrical field potential between the stimulating electrode to the auditory nerves, and (for reasons that are poorly understood) reduce the current required to elicit a perceptual threshold [25]. This means that sounds are too loud, and a child's reluctance to use the implant may be the first clinical sign of a middle ear effusion in a child with an implant. Patients with recurrent otitis media should have a tympanostomy tube inserted, and this is particularly recommended prior to implantation in patients with a history of recurrent acute otitis media.

Mastoiditis

Mastoiditis occurs in approximately 1% of pediatric cochlear implant recipients [26]. This is most prevalent in the first few weeks after implantation. Complicated wound infections with signs of a fluid collection or a discharging wound are indicative of mastoiditis and require hospitalization for treatment with intravenous antibiotics, and may require surgical drainage/washout with or without device explantation. As described above, the location of the wound discharge and the presence or absence of fluctuance over the implant can help to differentiate mastoiditis from an implant body infection. Infected fluid or tissue should be taken for microbiology and then empirical antibiotics should be commenced. Intracranial involvement (such as lateral sinus thrombosis or an intracranial collection) should be excluded. Surgical intervention is recommended in the following circumstances: if it has been established that there are complications, when there is severe infection, or a failure to respond to antibiotic therapy. A tympanostomy tube together with intravenous antibiotics may be all that is required (to drain the purulent discharge), or it may be necessary to perform a formal mastoid exploration for the removal of the inflammatory tissue. Where possible, the implant should be left in place, however failure to improve with the above measures may indicate implant body infection. A prolonged course of antibiotics is indicated when mastoiditis occurs early after cochlear implantation; input from an infectious diseases physician is recommended.

Meningitis

Meningitis following cochlear implantation surgery is a rare but devastating complication. This may present as early as within 24 h post-implantation or as late as several years after, with the highest risk within the first 2 months.

An increased risk of meningitis has been found to be associated with structural anomalies of the inner ear, especially when there is an abnormal communication between the perilymph and the internal auditory meatus. Other risk factors include immune deficiency, the presence of neurological prostheses, cerebrospinal fluid (CSF) leak, and a history of basilar skull fracture or meningitis [27]. One cochlear implant design was associated with a significant increase in the incidence of meningitis in the early 2000s [6, 7]. This device required the use of a positioner—a plastic spacer that was inserted lateral to the cochlear electrode to push it medially. The rationale was that this should bring the electrode contacts closer to the modiolus. The device was voluntarily recalled by the manufacturer in 2002. It is still debated whether the meningitis was caused by the degree of cochlear trauma seen with this electrode design, which far exceeded that of other implants, or the presence of two prosthetic materials in close proximity within the cochlea. The latter may have caused a dead space within which bacteria could grow while evading immune surveillance [28].

Streptococcus pneumoniae accounts for the majority of cases of meningitis. Similarly, *S. pneumoniae* is associated with meningitis under other conditions where the dura has been breached, such as a skull base fracture or CSF leak [27]. Meningitis may be caused by several mechanisms following cochlear implantation. The most obvious is spread of infection along the electrode within the cochlea and from there, to the CSF via the route(s) described above. This is the major concern, especially in the first few weeks after surgery. To mitigate this risk, patients should be vaccinated against *S. pneumoniae* prior to surgery. In addition, a prophylactic antibiotic should be started within the appropriate window of time prior to incision as discussed above, and after the insertion of the electrodes into the cochlea via a cochleostomy, fascial tissue may be packed around the cochleostomy to seal off the cochlea from the middle ear. In animal studies, adding this fascial tissue has been shown to reduce the subsequent risk of infectious spread from otitis media [29]. In the event that a CSF "gusher" is encountered at surgery, the seal is reinforced with muscle, the patient is rested head-up, and on rare occasions consideration may need to be given to the insertion of a lumbar drain [30]. All the patients undergoing cochlear implantation should be advised that potential symptoms of meningitis (e.g., fever and/or headache) at any point postoperatively should prompt urgent medical assessment because of the increased risk of meningitis in cochlear implant patients.

Meningitis can occur months or years after surgery. If a patient with a cochlear implant presents with suspected meningitis, their initial management follows conventional lines: urgent antibiotics are administered, a lumbar puncture is performed and the CSF should be sent for cell counts, Gram staining, and culture. The major clinical decision for the cochlear implant team is to ascertain whether the infection has arisen from the implanted ear. This can be confirmed by the observation of a purulent middle ear effusion in the implanted ear, or the presence of mastoid and middle ear opacification on CT scanning. Magnetic resonance imaging (MRI) will likely be required, but because of the magnet in the receiver-stimulator's coil, the image quality may be too poor in the vicinity of the middle ear to discern the presence of an effusion. MRI may be used in some cochlear implant recipients with additional stabilization measures in place to minimize the risk of displacing the implant or causing significant pain due to force pressures. The risk of displacement differs with different types of cochlear implants due to varying torque measurements between models, and this should be managed on an individual basis in consultation with the device manufacturer [31]. In the event that otitis media is confirmed in the implanted ear, the surgeon may need to consider whether surgical drainage of the fluid or removal of the device is warranted. These are complex matters that will depend on the specific circumstances. It is worth keeping in mind that the origin of the meningitis may not be related to the cochlear implant, and if that is the case, the only impact of the implant on management will be the complexity of performing safe MRI imaging and the image distortion that results from the cochlear implant magnet [32]. If need be, the implant magnet can be removed through a minor surgical procedure prior to the MRI, and then replaced later on.

Meningitis also has an influence on the possibility of *future* cochlear implantation. Hearing loss is frequently a complication of meningitis. When caused by *H. influenzae* there is a chance that the hearing may recover spontaneously in the months after infection, but this seldom occurs when the causative organism is *S. pneumoniae*. The main concern regarding cochlear implantation is that the scala tympani may fibrose and then ossify. This may occur as early as a month after meningitis or may not become clinically apparent until several months later [33]. The fibrosis and ossification have been attributed to injury to the cochlear endosteum [34]. This pathological change can make cochlear implantation impossible, as the lumen to receive the device is obliterated. In light of this, sequential MRI scanning is required after meningitis when there has been severe hearing loss, with urgent cochlear implantation if the cochlea begins to ossify. Once implanted, the

device will function quite well, even if the scalar ossification continues.

To decrease the risk of meningitis, all children should be immunized with vaccination against *H. influenzae* and *S. pneumoniae* prior to implantation. Guidelines for specific vaccinations have been published [14].

Special Considerations

Acute Otitis Media

Acute otitis media (AOM) is the most common bacterial infection in children and many pediatric cochlear implant candidates have a history of otitis media. Given the benefit of early cochlear implantation for speech and language development, delaying implantation until the child is older to reduce the likely incidence of AOM is not recommended. Control of AOM is required very early on in the assessment, as it is difficult to ascertain whether the patient is an audiological candidate for implantation if their evaluation is complicated by the conductive loss associated with a middle ear effusion. Furthermore, it is not possible to proceed safely to implant surgery if there is a middle ear effusion, especially when purulent. Tympanostomy tube placement is recommended for these patients and we have a low threshold for also recommending an adenoidectomy, to reduce the risk of subsequent otitis media. Patients with a dry and clean tympanostomy tube can safely proceed to cochlear implantation. Studies have demonstrated that good control of AOM before implantation reduces the risk of AOM post-implantation [35].

Acute otitis media at the time of surgery increases the risk of device contamination and has the potential to increase the risk of meningitis [27]. If a patient has otitis media at the time of implantation, we advocate that surgery be delayed 6 weeks until the infection has resolved and the inflammation has settled. Some surgeons are of the opinion that implantation may proceed without complication in the presence of otitis media with effusion, but we view this as taking an unnecessary risk. Placement of a tympanostomy tube provides much greater certainty concerning the safe timeline for implantation after a delay for treating otitis media. This approach is of particular value in young children (under 3 years of age) who are in the critical stage of speech and language acquisition.

Chronic Otitis Media

Cochlear implantation is contraindicated in patients with untreated chronic otitis media, due to the potential for device infection or chronic middle ear inflammation spreading into the cochlea. With the latter, granulation tissue spreads into the inner ear and cochlear implant function deteriorates and/or fluctuates [36]. The implant electrode must be removed and the inner ear, which will then undergo fibrotic change, cannot be implanted again. Interestingly, in these patients the disease extends no further than the cochlea, and does not present as otitic meningitis.

Management is determined by the activity of the chronic otitis media. Cochlear implant candidates with inactive disease (i.e., a dry ear) may present with either a tympanic membrane perforation or a dry mastoid cavity. Tympanic membrane perforations are closed prior to implantation to reduce the risk of device and middle ear infection through contamination via the external ear canal. Autologous cartilage is the preferred graft tissue in these cases as the risk of retraction of the graft onto the implant electrode is lower than with fascia. However, myringoplasty failures are not uncommon and it is recommended that cochlear implantation be delayed until the tympanic membrane is both intact and stable, which is a minimum of 3 months post-myringoplasty. Tiny tympanic membrane perforations may not require a myringoplasty as the risk of exposure to the external environment is considerably less than with a large perforation; in this regard a small perforation is similar to a tympanostomy tube. The over-riding consideration with a small perforation is whether the ear ever discharges—if so it should be closed. Dry mastoid cavities may be implanted by rotating a vascularized fascial flap in over the implant electrode [37]. Alternatively, the mastoid can be obliterated with blind closure of the exter-

nal ear canal [38]. Cochlear implantation follows 3–6 months later or when deemed appropriate; implantation can be performed at the first operation as a single-stage procedure.

Chronic active otitis media (also known as chronic suppurative otitis media) implies that there is aural discharge. Treatment of these ears is always staged. The first stage aims to dry up the ear, and eliminate middle ear/mastoid infection and/or granulation or cholesteatoma. A canal wall up mastoidectomy may be appropriate, provided that the surgeon is confident that the ear will remain stable, and in particular that the tympanic membrane will not retract around the implant. If a mastoid cavity needs to be created, then the mastoid should be obliterated together with a blind sac closure of the external ear canal.

Conclusion

Cochlear implantation is a common and safe procedure with a relatively low incidence of infectious complications. Surgical site infections are the most common postoperative infectious complication; most are minor and can be treated with oral antibiotics in an outpatient setting. Children with cochlear implants may have episodes of AOM and these should be managed with conventional antibiotics; tympanostomy tube placement should be considered in children with recurrent episodes of AOM. Less common but more severe complications including mastoiditis, implant body infection, and meningitis require hospital admission for intravenous antibiotics and surgical management. Device removal is rarely necessary but if required, efforts should be made to preserve cochlear architecture to allow for possible re-implantation in the future.

References

1. Gopen Q, Rosowski JJ, Merchant SN. Anatomy of the normal human cochlear aqueduct with functional implications. Hear Res. 1997;107(1-2):9–22.
2. Phelps PD, King A, Michaels L. Cochlear dysplasia and meningitis. Am J Otol. 1994;15(4):551–7.
3. Hoffman RA, Cohen NL. Complications of cochlear implant surgery. Ann Otol Rhinol Laryngol. 1995;104(Supp; 166):420–2.
4. Yu KCY, Hegarty JL, Gantz BJ, Lalwani AK. Conservative management of infections in cochlear implant recipients. Otolaryngol Head Neck Surg. 2001;125:66–70.
5. Cunningham CD, Slattery WH, Luxford WM. Postoperative infection in cochlear implant patients. Otolaryngol Head Neck Surg. 2004;131(1):109–14.
6. Wilson-Clark SD, Squires S, Deeks S, Centers for Disease Control and Prevention (CDC). Bacterial meningitis among cochlear implant recipients – Canada, 2002. MMWR Suppl. 2006;55:20.
7. Reefhuis J, Honein MA, Whitney CG, Chamany S, Mann EA, et al. Risk of bacterial meningitis in children with cochlear implants. N Engl J Med. 2003;349(5):435–45.
8. Hopfenspirger MT, Levine SC, Rimell FL. Infectious complications in pediatric cochlear implants. Laryngoscope. 2007;117:1825.
9. Gawecki W, Karlik M, Borucki L, Szyfter-Harris J, Wrobel M. Skin flap complications after cochlear implantations. Eur Arch Otorhinolaryngol. 2016;273:4175–83.
10. Zawawi F, Cardona I, Akinpelu OV, Daniel SJ. Acute mastoiditis in children with cochlear implants: is explantation required? Otolaryngol Head Neck Surg. 2014;151:394.
11. Clark GM, Pyman BC, Bailey QR. The surgery for multiple-electrode cochlear implantations. J Laryngol Otol. 1979;93(3):215–23.
12. Ajalloueyan M, Amirsalari S, Radfar S, Tavallaee A, Khoshini S. Flap outcome using "C" shaped and "new" incisions in pediatric cochlear implantation. Iran Red Crescent Med J. 2014;14(4):218–21.
13. Gibson WPR, Harrison HC, Prowse C. A new incision for placement of cochlear implants. J Laryngol Otol. 1995;109(9):821–5.
14. Rubin LG, Papsin B, Committee on Infectious Diseases and Section on Otolaryngology-Head and Neck Surgery. Cochlear implants in children: surgical site infections and prevention and treatment of acute otitis media and meningitis. Pediatrics. 2010;126(2):381–91.
15. Bratzler DW, Patchen Dellinger E, Olsen KM, et al. Clinical practice guidelines for antimicrobial prophylaxis in surgery. Am J Health Sys Pharm. 2013;70:195–283.
16. Hirsch BE, Blikas A, Whitaker M. Antibiotic prophylaxis in cochlear implant surgery. Laryngoscope. 2007;117(5):864–7.
17. O'Leary S. Biofilm and prostheses in otology. In: Kania R, Ars B, editors. Biofilms in otitis. Amsterdam: Kugler Publications; 2015.
18. Antonelli PJ, Lee JC, Burne RA. Bacterial biofilms may contribute to persistent cochlear implant infection. Otol Neurotol. 2004;25:953–7.
19. Pawlowski KS, Wawro D, Roland PS. Bacterial biofilm formation on a human cochlear implant. Otol Neurotol. 2005;26:972–5.

20. Lalani T, Sexton DJ, Tucci DL. Cochlear implant infections. In: Post TW, Calderwood SB, Kaplan SL, Thorner AR, editors. UptoDate; 2016. Available from http://www.uptodate.com/contents/cochlear-implant-infections Accessed 17/01/2017.
21. Geraghty M, Fagan P, Moisidis E. Management of cochlear implant device extrusion: case series and literature review. J Laryngol Otol. 2014;128(Suppl 2):S55–8.
22. Parkins CW, Metzinger SE, Marks HW, Lyons GD. Management of late extrusions of cochlear implants. Am J Otol. 1998;19:768–73.
23. Hoffman RA, Cohen N, Waltzman S, Shapiro W, Goldofsky E. Delayed extrusion of the nucleus multichannel cochlear implant. Otolaryngol Head Neck Surg. 1991;105(1):117–9.
24. Spiro DM, Arnold DH. The concept and practice of a wait-and-see approach to acute otitis media. Curr Opin Pediatr. 2008;20(1):72–8.
25. Dixon JF, Shinn JB, Adkins M, Hardin BD, Bush ML. Middle ear disease and cochlear implant function: a case study. Hear Bal Commun. 2014;12(3):155–8.
26. Kempf HG, Tempel S, Johann K, Lenarz T. Complications of cochlear implant surgery in children and adults. Laryngothinootologie. 1999;78(10):529–37.
27. Arnold W, Bredberg G, Gstottner W, Helms J, Hildmann H, et al. Meningitis following cochlear implantation: pathomechanisms, clinical symptoms, conservative and surgical treatments. ORL J Otorhinolaryngol Relat Spec. 2002;64(6):382.
28. Wei BPC, Shepherd RK, Robins-Browne RM, Clark GM, O'Leary SJ. Pneumococcal meningitis post cochlear implantation: potential routes of infection and pathophysiology. Otolaryngol Head Neck Surg. 2010;143(5,s3):S15–23.
29. Dahm MC, Clark GM, Franz BKH, Shepherd RK, Burton MJ, et al. Cochlear implantation in children. Labyrinthitis following pneumococcal otitis media in unimplanted and implanted cat cochleas. Acta Ototlaryngol. 1994;114:620–5.
30. Wootten CT, Backous DD, Haynes DS. Management of cerebrospinal fluid leakage from cochleostomy during cochlear implant surgery. Laryngoscope. 2006;116(11):2055–9.
31. Teissl C, Kremser C, Hochmair ES, Hochmair-Desoyer JH. Magnetic resonance imaging and cochlear implants: compatibility and safety aspects. J Magn Reson Imaging. 1999;9:26–38.
32. Baumgartner WD, Youssefzadeh S, Hamzavi J, Czerny C, Gstoettner W. Clinical application of magnetic resonance imaging in 30 cochlear implant patients. Otol Neurotol. 2001;22(6):818–22.
33. Novak MA, Fifer RC, Barkmeier JC, Firszt JB. Labyrinthine ossification after meningitis: it's implications for cochlear implantation. Otolaryngol Head Neck Surg. 1990;103(3):351–6.
34. Brodie HA, Thompson TC, Vassilian L, Lee BN. Induction of labyrinthitis ossificans after pneumococcal meningitis: an animal model. Otolaryngol Head Neck Surg. 1998;118(1):15–21.
35. Luntz M, Teszler CB, Shpak T. Cochlear implantation in children with otitis media: second stage of a long-term prospective study. Int J Pediatr Otorhinolaryngol. 2004;68(3):273–80.
36. Benatti A, Castiglione A, Trevisi P, Bovo R, Rosignoli M, et al. Endocochlear inflammation in cochlear implant users: case report and literature review. Int J Pediatr Otorhinolaryngol. 2013;77(6):885–93.
37. Tong MCF, van Hasselt CA. Cochlear implant surgery in a modified radical mastoidectomy cavity reconstructed utilizing the Hong Kong vascularized temporalis fascia flap technique. In: Honjo I, Takahashi H, editors. Cochlear implant and related sciences update. 1st Asia Pacific Symposium, Kyoto; 1996.
38. Leung R, Briggs RJS. Indications for and outcomes of mastoid obliteration in cochlear implantation. Otol Neurotol. 2007;28(3):330–4.