Tinnitus is a common symptom of many cochlear or auditory system pathologies. Since tinnitus is frequently associated with a sensorineural hearing loss, it is not surprising that a large proportion of profoundly and totally deaf patients describe tinnitus as a symptom. The clinical management of severe tinnitus in these patients is discussed with particular emphasis on the use of electrical stimulation. While cochlear implants appear to provide a measure of relief when being used, significant improvements in the management of severe tinnitus will only occur when we have a greater understanding of the underlying pathophysiology, diagnostic procedures that can accurately establish the site of tinnitus generation, and more objective clinical trial procedures that include the use of controls.

Tinnitus aurium is a common symptom of many cochlear or central auditory system pathologies that involve sensory cell or neural damage. In contrast, tinnitus is relatively uncommon in cases of conductive hearing loss where the pathological changes occur in the middle ear and are of a mechanical origin (Hazell 1987).

A recent British survey found that up to 9% of the general population described their tinnitus as a problem. One percent of the population surveyed reported severe tinnitus, with 0.5% describing their tinnitus as so severe that it resulted in their inability to lead a normal life (MRC Institute of Hearing Research 1987). Given a similar level of industrialization in Australia, we can expect the incidence of severe tinnitus in this country to match that experienced in Britain.

While the careful psychological management of tinnitus patients remains an important form of treatment (Wilson 1993), until recently there were few alternative options available for the clinical management of severe tinnitus. A number of procedures that have been investigated for some time may soon be available clinically; these include drug treatment, acoustic masking and the use of electrical stimulation. While all of these treatments suffer limitations, they provide hope of improved tinnitus management in some patients.

Since tinnitus is frequently associated with a sensorineural hearing loss, it is not surprising that a large proportion of profoundly to totally deaf patients describe tinnitus as a symptom. However, it is this group of patients who are considered the most difficult to treat (Hazell 1987). It is the purpose of the present paper to review the clinical management of tinnitus in the profoundly and totally deaf with particular emphasis on the use of electrical stimulation. The first task, however, is to review current theories on the mechanisms of tinnitus generation.

Theories of Tinnitus Generation

Tinnitus aurium is generally thought to arise as a result of abnormal neural discharge patterns generated within the cochlea or CNS. As such, some forms of tinnitus may be derived from mechanisms that are also common to other neural pathologies such as neuralgia. It is not surprising that the treatment of pain relief in neuralgia includes procedures that are also used in the treatment of tinnitus, such as anaesthetic block and electrical stimulation (Maiman et al 1985, Nissen 1987).

Several of the proposed mechanisms of tinnitus generation are based on an increase in the spontaneous activity of auditory nerve fibres in cochleae with surviving hair cells. For example, experimental studies have shown that when animals are given salicylate at doses sufficient to induce tinnitus in humans, the spontaneous firing rates of auditory nerve fibres increase beyond the range found in normal animals (Evans et al 1981). More recently, Jastreboff and Brennan (1992) have used salicylate to induce tinnitus in animals and have shown that salicylate intoxication results in a disturbance of the calcium homeostasis within the cochlea. Their work has also indicated that calcium supplements in the diet could reduce salicylate induced tinnitus in experimental animals.

Eggermont (1984) has postulated that tinnitus associated with Meniere’s disease is a result of the displacement of the basilar membrane toward the scala tympani following endolymphatic hydrops. Such displacement can result in increased spontaneous activity via depolarization of residual hair cells. Since tinnitus in
Meniere’s disease is typically characterized by a low pitch roar (Hazell 1987), it is tempting to speculate that the most extensive basilar membrane displacement in endolymphatic hydrops is likely to occur towards the apex of the cochlea. It is worth noting that the basilar membrane in this region of the cochlea is at its most pliable (Gummer et al. 1981) and therefore, presumably, most susceptible to distortion in the case of endolymphatic hydrops.

The pitch of tinnitus in subjects with a noise induced high frequency hearing loss is typically localized to the steeply rising border of the lesion (Penner 1980). Such tinnitus can usually be masked by a band of noise surrounding the frequency of the tinnitus – this is used clinically in tinnitus maskers (Hazell et al. 1985).

Significantly, experimental studies in animals with a high frequency noise induced hearing loss have shown that auditory nerve fibres in the damaged region of the cochlea have higher spontaneous rates than adjacent fibres innervating undamaged regions (Liberman and Kiang 1978, Salvi and Ahroon 1983). It has been suggested that this so-called “edge effect” – between spontaneous rates of normal and lesioned regions of the cochlea – may be the source of tinnitus generation in hearing loss (Penner 1980).

Kiang et al. (1970) have used a similar argument to propose the generation of tinnitus in cochleae damaged by ototoxic drugs. Unlike lesions associated with a noise induced hearing loss, several studies have shown significantly reduced spontaneous activity in cochleas damaged with aminoglycosides (Kiang et al. 1970, Shepherd and Javel, unpublished observations). Kiang proposed that tinnitus generation in this model was again a result of an abrupt change in spontaneous activity in the region of the cochlear lesion. In this case, however, the auditory nerve fibres projecting to the damaged organ of Corti would exhibit lower spontaneous activity compared with nerve fibres innervating the undamaged region.

Auditory nerve fibres innervating cochleae devoid of hair cells show little, if any, evidence of spontaneous activity (Shepherd and Javel, unpublished observations). It seems reasonable to suggest that, in such pathology, tinnitus is generated from more central structures within the auditory pathway. Here too, “contrast” in spontaneous activity between silenced and spontaneously active regions of the auditory brainstem may play a role in tinnitus generation. For example, while the absence of hair cells or the sectioning of the auditory nerve abolishes spontaneous activity in both the auditory nerve and the ventral cochlear nucleus (Durham et al 1989), quite high levels of spontaneous activity can still be recorded in the dorsal cochlear nucleus (Koerber et al. 1966). It is conceivable that this significant difference in spontaneous activity may result in the generation of tinnitus by some comparator network, either via intrinsic connections within the cochlear nucleus itself (Snyder and Leake 1988), or within some higher auditory centre that receives input from both subdivisions of this nucleus.

Finally, Eggermont (1984) has suggested that the efferent pathway may play an important role in the control of tinnitus, as the efferent system is known to act at many levels of the auditory pathway to modify neural firing patterns. He has proposed that the amelioration of tinnitus using such procedures as relaxation and distraction may be controlled by the efferent pathway.

Management of tinnitus in the profoundly-totally deaf

Current tinnitus management techniques are palliative in nature. They generally rely on procedures that temporarily abolish (e.g. anaesthesia block) or mask (e.g. tinnitus maskers, electrical stimulation) the abnormal spontaneous activity thought to be the source of tinnitus generation. While these management techniques are considered temporary, lasting only for the duration of the treatment, long-term ‘residual inhibition’ of tinnitus is occasionally reported. Interestingly, a similar effect is sometimes observed in the management of neuralgia (Maiman et al. 1985). Techniques to induce residual inhibition in tinnitus subjects should be one of our long-term goals. However, at present the mechanisms giving rise to such an effect are far from clear.

Before describing the potential management techniques available, it is important to note that, as a result of the subjective nature of this complaint and the importance of the subject’s psychological state in the perception of tinnitus, the use of carefully controlled clinical studies are considered important in tinnitus management trials (Duckert and Rees 1984, Dobie et al. 1993). A major criticism of many early trials was the lack of an appropriate control (Cotter 1981). The design of a study must be carefully considered when evaluating therapeutic trials for tinnitus.

1. Drug Therapy

Lignocaine is the most frequently cited drug used to ameliorate severe tinnitus (Melding et al 1978, Duckert and Rees 1983). Its use for the diagnosis of central pain in neuralgia resulted in some patients reporting a temporary relief from their tinnitus (Melding et al. 1978). Melding and his colleagues then evaluated intravenous lignocaine in 78 subjects using dose rates of 1-2 mg/kg. Although the study did not have a control, 35% of the subjects reported complete abolition of their tinnitus, 28% reported good relief (>60% reduction of tinnitus), 12% partial relief (30%-60% reduction), while 26% reported no relief. The duration of relief extended from 10 minutes to three days. The subjects most responsive to IV. lignocaine were those with a partial sensorineural hearing loss. However, as commonly found in tinnitus management, there was no other indication of predictive outcome. These authors reported that no subject suffered an adverse reaction in response to the treatment.

More recent controlled studies have generally supported these encouraging results (Duckert and Rees 1983, Hulshof and Vermeij 1984) although side effects, including an increase in tinnitus, have been reported in some subjects.

However, the intravenous administration of lignocaine is not an acceptable tinnitus management technique. An oral analogue of lignocaine was therefore developed and evaluated. Unfortunately, controlled studies have demonstrated that this treatment is not effective (Hulshof and Vermeij 1984).
More recently, antidepressants have been evaluated for their effectiveness in reducing severe tinnitus. Dobie et al (1993) reported that nortriptyline (a tricyclic antidepressant) can help reduce tinnitus in some subjects, particularly depressed subjects or subjects with insomnia. Dobie suggests that nortriptyline did not directly affect the tinnitus sensation but helped reduce the stress related depression known to exacerbate tinnitus (Dobie et al 1993). Their study also indicated a statistically significant placebo effect, emphasizing the importance of using controlled trials.

Although drug therapy for the management of tinnitus has been investigated for many years, the techniques have not developed into routine clinical procedures. Indeed, no drug has yet been approved by the U.S. Food and Drug Administration (FDA) for use in tinnitus management (Marion and Cevette 1991). While results using lignocaine continue to be encouraging, a more suitable technique for delivering the drug to the cochlea must first be developed. In addition, further studies are required to evaluate the use of antidepressants. However, they appear to have some potential in a small group of patients.

2. Surgical Treatment for Tinnitus

It is clear that surgery plays only a minor role in the management of tinnitus (Barrs and Brackmann 1984, Silverstein et al 1986, Hazell 1987, Nissen 1987). While tinnitus associated with certain specific disease processes such as otosclerosis, cholesteroloma, acoustic neuroma and venous malfunctions may be alleviated by surgery for the underlying etiology, surgery involving the sectioning of the auditory nerve is only recommended when associated with severe vertigo (Hazell 1987, Nissen 1987).

Retrospective studies in patients who have had their auditory and/or vestibular nerves sectioned as a result of severe vertigo, have shown that VIIIth nerve sectioning does not necessarily ameliorate tinnitus. For example, Barrs and Brackmann (1984) reported on 110 patients who underwent nerve sectioning for vertigo with tinnitus as a secondary complaint. More than half the patients (62%) who underwent a complete vestibular and auditory nerve section described their tinnitus as improved postoperatively. Fourteen percent of these patients reported that their tinnitus had completely disappeared. However, 12% of patients described their tinnitus as worse. In patients who underwent vestibular nerve section only, 49% described their tinnitus as improved, including 10% who reported that their tinnitus had completely disappeared. The mechanism by which the sectioning of the vestibular nerves results in the amelioration of tinnitus is not understood and warrants investigation. Significantly, however, 19% of patients that underwent vestibular nerve section only, felt that their tinnitus was worse following surgery.

Because these treatments provided only partial success, and since they obviate the use of potentially effective treatments in the future, surgical intervention for tinnitus is rarely considered.

Finally, as we will note in some detail below, the great majority of cochlear implant patients describe a reduction in tinnitus when using the device (Pyman et al 1990, Gibson 1991, McKerrow et al 1991, Souliere et al 1992, Ward et al 1992) with few, if any, reporting a worsening of their tinnitus. Therefore, while cochlear implants are primarily communication aids for the profoundly to totally deaf, they also appear to provide some help in the management of tinnitus in this group of patients.

3. Tinnitus Maskers

Tinnitus maskers are often used to obtain relief from tinnitus in the partially deaf by generating an acoustic masker signal set in frequency and intensity to approximately match the perceived pitch and loudness of the subject’s tinnitus. While clinical trials indicate they are an effective technique for managing tinnitus in some subjects, there is concern that such devices may have adverse effects on residual hearing and ultimately exacerbate the tinnitus (Cotter 1981). Because they are of very limited value or no use in the profoundly to totally deaf, they will not be considered in any detail here. For further information the reader is directed to an extensive study by Hazell et al (1985).

4. Electrical Stimulation

The use of electrical stimulation to suppress tinnitus has a long history (for an excellent review see Vernon 1987). In 1801, Grapengiesser applied direct current (DC) to electrodes placed within the external meatus or on the mastoid and reported temporary relief of tinnitus in some subjects. Significantly, anodal currents tended to reduce tinnitus while cathodal currents generally increased tinnitus and evoked auditory sensations. Because the procedure also resulted in nausea and pain, it was not continued (Feldman 1984, Vernon 1987).

A similar study was performed by Hatton et al (1960), initiating our recent interest in the use of electrical stimulation in tinnitus management. Hatton and his colleagues reported 45% of their subjects observed a reduction in their tinnitus when anodal current was applied to electrodes ipsilateral to the tinnitus. The intensity of tinnitus generally decreased as the current was increased, and the suppression would last for the duration of the stimulus. Like Grapengiesser, Hatton found that with the cathode placed on the tinnitus side of the head, the tinnitus would increase in loudness as the current increased. The majority of subjects whose tinnitus could be affected by the DC had severe hearing losses. This important finding has since been supported in more recent studies. Hatton and his colleagues made no attempt to use their findings in the clinical management of tinnitus. This was presumably because they were aware of the damaging effects of DC.

Using pulse DC stimuli during promotory stimulation to select patients for cochlear implantation, Cazals et al (1978) reported 6 of 7 patients had indicated that their tinnitus ipsilateral to the stimulating electrode had been suppressed when using anodal pulses. The Bordeaux group confirmed their findings in additional studies (Portmann et al 1979). However, they ultimately abandoned the use of DC stimuli after demonstrating that it could cause extensive tissue damage within the cochlea (Aran et al 1983).

A number of experimental studies during this period essentially confirmed the clinical observation of the effects of DC on tinnitus. These studies demonstrated...
significant reductions in auditory nerve spontaneous activity using anodic currents and increases in spontaneous activity and evoked neural activity when using cathodic currents within the scala tympani (Konishi et al 1969, Schreiner et al 1986). These changes were attributed to hyperpolarization of the inner hair cell and auditory nerve fibres associated with an anodic current and their depolarization in cathodic currents.

More recently, investigators have used charge balanced alternating currents (AC) in an attempt to ameliorate tinnitus in both hearing and profoundly deaf patients. These stimuli do not produce damaging electrochemical reactions and are therefore considered safe (Shepherd et al 1990). A variety of electrode sites have been investigated, including transcutaneous (mastoid, external auditory meatus), transtympanic (promontory, round window) and intracochlear.

On the basis of tinnitus suppression among their cochlear implant patients, Chouard et al (1981) used a variety of waveforms to stimulate electrodes placed in the vicinity of the ipsilateral ear in subjects with severe tinnitus. More than one-third of the 53 subjects experienced considerable relief, although in no case was the tinnitus completely abolished. These investigators also reported no incidence of vertigo or increased tinnitus, and described residual inhibition in some subjects lasting several weeks. While subjects found a variety of stimulus waveforms useful, the most efficacious was an anodic pulse capacitively coupled to provide no nett DC. Finally, there appeared to be no relationship between predictive outcome and factors such as aetiology, age and gender.

Somewhat less encouraging results were obtained when this work was repeated by Vernon and Fenwick (1985). Only 22% of 23 subjects reported a reduction of 40% or more in their tinnitus and in each case only one of the three waveforms trialled was found to be effective. Vernon and Fenwick observed shorter periods of residual inhibition – on average 3 hours – although in no case was the tinnitus worsened.

More recently, trials of a prototype device designed for transtympanic electrical tinnitus suppressor have shown that they are effective for a small proportion of patients with both sensorineural hearing loss and severe tinnitus (Dobie et al 1986, Shulman 1987). However, the production of these devices has recently ceased and they are therefore no longer available for tinnitus management.

Other investigators have opted for devices in which the stimulating electrode is placed either on the promontory or the round window. Graham and Hazell (1977) implanted promontory stimulating electrodes in 13 severe to profoundly deaf subjects and stimulated with low frequency (10 and 30 Hz) sinusoidal current waveforms. Only two subjects (15%) reported relief from tinnitus, although one experienced a period of residual inhibition of four hours. Side effects with this stimulus included vertigo in four subjects.

This group has continued development of a transtympanic tinnitus suppressor and have recently reported improved results using an electrode located on the round window (Hazell et al 1993). Testing a variety of frequencies of sinusoidal current, they obtained their best results using low frequency stimuli (20 Hz). Seven of nine subjects (78%) with unilateral total deafness and severe tinnitus were able to obtain total suppression of their tinnitus for the duration of the stimulation period. However, in no case was it possible to suppress tinnitus using a stimulus that did not also evoke a hearing sensation. This group now has three subjects using permanent devices and report “good results” for tinnitus suppression over a period of 3.5 years. While this study, like the majority in this type of trial, was uncontrolled, the results are encouraging and support the underlying belief that the likelihood of success of these devices increases as the stimulating electrode is placed closer to the cochlea.

During the past six years, Cochlear Pty. Ltd. has been carrying out trials both in Australia and overseas using a stimulator capable of outputting a number of different charge balanced AC waveforms at a variety of stimulus frequencies. Their objective has been to evaluate the effectiveness of electrical stimulation for tinnitus suppression at current levels below auditory threshold, using both transcutaneous and transtympanic stimulating electrode sites. Subjects with normal to profound hearing losses and chronic severe tinnitus were acutely stimulated using an electrode located on the round window, promontory or within the external auditory meatus (Staller et al, in press). Of the 92 subjects acutely stimulated using an ear canal electrode, approximately 18% reported a significant reduction (>40%) in tinnitus loudness, and a further 16% reported changes that were not significant. Of the 65 subjects receiving promontory stimulation, only 14% reported a significant reduction in their tinnitus during the stimulation period. A further 29% reported tinnitus reductions that were less significant. These authors also reported little overlap in significant tinnitus reduction between promontory and ear canal electrodes, with only one subject indicating significant suppression at both stimulus sites. The reason for this somewhat surprising result is not clear. Finally, a number of subjects reporting significant tinnitus reduction also described periods of residual inhibition lasting from minutes to several months.

A greater proportion of subjects reported a reduction in their tinnitus when the stimulating electrode was placed on the round window. Of the 24 subjects in this trial, 33% reported a significant reduction and a further 29% reported a non-significant reduction in their tinnitus (Staller et al, in press). Moreover, all subjects that obtained a significant reduction in their tinnitus also exhibited some degree of residual inhibition. Similar results were observed when round window stimulation was used in a 5-10 day chronic trial, although the degree of tinnitus suppression tended to vary during this period. In addition, approximately 9% of the patients in this trial demonstrated a placebo effect. It is clear, however, that the round window was the most effective stimulation site tested in this study.

Consistent with other reports, Staller et al (in press) found no significant predictive factors related to electrical stimulation tinnitus suppression. Moreover, no specific stimulus waveform was demonstrated to be most effective. However, the frequency of stimulation did appear to be an important factor, with low frequencies
(50-60 Hz) producing suppression in the largest proportion of patients. This finding is consistent with the observations of Hazell et al (1993).

This work has expanded to include eight clinics throughout the world. However, due to the relatively small number of subjects apparently receiving significant benefit, the probable need to implant stimulating electrodes close to the cochlea, the potential of adverse side effects (pain, vertigo, facial nerve response), the lack of any predictive factors, and concerns with patient acceptance, Cochlear Pty. Ltd. has recently discontinued its program.

In conclusion, it is clear that electrical stimulation specifically for the suppression of tinnitus will provide significant relief for a small number of patients. However, at this point no commercially available device is available for clinical trials. Before viable devices become commercially available, a greater understanding of the mechanisms underlying electrical tinnitus suppression, an improved diagnostic technique that will provide some indicator of predictive outcome, the optimization of stimulus waveforms, electrodes and stimulation sites, and a thorough safety evaluation are required.

Tinnitus Suppression following Cochlear Implantation

While the primary objective of cochlear implants is to restore useful levels of hearing to the profoundly and totally deaf, experience has shown that these devices also help to relieve tinnitus in a large proportion of implant patients.

As already noted, patients with a profound-total hearing loss have a high incidence of tinnitus. Published results from the pre-operative evaluation of implant patients indicate that approximately 80% experience some level of tinnitus (Table I), significantly higher than the 36% who experience tinnitus within the general population (MRC Institute of Hearing Research, 1987).

The implantation of a scala tympani electrode array has the potential to exacerbate tinnitus postoperatively. However, this does not appear to be a significant problem. Experience in our clinic has shown no indication of a worsening of tinnitus following surgery (Pyman et al 1990). Gibson (1991) evaluated tinnitus in 54 of his implant patients and reported 9.3% had indicated an increase in their tinnitus postoperatively. In these cases the increase in tinnitus was temporary, typically lasting for periods of up to three months. Significantly, Gibson also reported that 41% of his patients with tinnitus prior to cochlear implantation observed a reduction postoperatively — including some patients that reported total relief. A reduction in tinnitus following implantation has also been reported by other groups (Pialoux et al 1979).

There is clear agreement among implant groups that tinnitus suppression is observed in the majority of patients while using the device (Table I).

Thedinger et al (1985), using the House single channel cochlear implant, reported "approximately half of the patients obtained significant tinnitus reduction" when using the device. They also noted that less than 8% reported a worsening of their tinnitus while the remainder observed no change.

Many clinics using the Cochlear Pty. Ltd. multiple channel implant have described reductions in tinnitus among patients during device use. Laszig et al (1989) noted that 31 of their 39 (80%) implant patients with tinnitus preoperatively had reported its suppression when using the device. Six patients (15%) described no change and two (5%) reported a reinforcement of their tinnitus.

In a controlled study using stimuli that evoked no hearing sensations, these authors reported that 6 of the 15 patients (40%) taking part in the trial had observed a suppression in their tinnitus. One of these patients also reported tinnitus suppression using a placebo. Similar results have recently been reported by a second group using a cochlear implant developed by the University of California, San Francisco (McKerrow et al 1991). These findings indicate that electrical stimulation per se — in this case the subthreshold stimulus — can effectively reduce tinnitus in a small number of patients. However, combined with the masking produced by the hearing sensations evoked when using their speech processor, a far greater number of patients observed tinnitus relief.

Thus masking would appear to play an important role in tinnitus suppression among cochlear implant patients. Whether this masking is more effective when information carrying stimuli such as speech is used, is an important question yet to be addressed.

Of the thirty two implant patients in our clinic that had reported tinnitus preoperatively, 69% indicated that their tinnitus had improved with use of the device (Pyman et al 1990). No patient described their tinnitus as worse following implantation. Gibson (1991) and Souliere et al (1992) also reported reductions in tinnitus among the majority of their patients while using the device. Both authors also described residual inhibition in some patients which typically lasted between one minute to several hours. In the majority of cases, however, tinnitus immediately returned when power to the device was removed.

Table I
Pre- and Post-operative Incidence of Tinnitus in Cochlear Implant Patients

<table>
<thead>
<tr>
<th>Patients with tinnitus</th>
<th>Patients with some relief using cochlear implants</th>
<th>Author</th>
</tr>
</thead>
<tbody>
<tr>
<td>preoperatively</td>
<td></td>
<td></td>
</tr>
<tr>
<td>67% (n=58)</td>
<td>79% (n=39)</td>
<td>Laszig et al, 1989</td>
</tr>
<tr>
<td>89% (n=36)</td>
<td>69% (n=32)</td>
<td>Pyman et al, 1990</td>
</tr>
<tr>
<td>72% (n=54)</td>
<td>84% (n=39)</td>
<td>Gibson, 1991</td>
</tr>
<tr>
<td>83% (n=6)</td>
<td>80% (n=5)</td>
<td>McKerrow et al, 1991</td>
</tr>
<tr>
<td>85% (n=33)</td>
<td>74% (n=28)</td>
<td>Souliere et al, 1992</td>
</tr>
<tr>
<td>76% (n=211)</td>
<td>66% (n=149)</td>
<td>Ward et al, 1992</td>
</tr>
</tbody>
</table>

*31% reported relief following implant surgery and 53% reported relief when using the device.
A review of clinical data by Cochlear Pty. Ltd. has basically confirmed these findings (Ward et al 1992). Of 149 implant patients reporting tinnitus prior to implantation, 66% described a decrease in their tinnitus with use of the device, 29% reported no change and 5% observed an increase in their tinnitus.

While the great majority of patients describe their tinnitus suppression as ipsilateral to the ear being stimulated, a small number of patients have reported bilateral tinnitus suppression (McKerrow et al 1991, Solliere et al 1992, Staller et al, in press). This would indicate that, at least in these patients, tinnitus is of a central origin.

In summary, while the majority of reports describing tinnitus in cochlear implant patients are derived from uncontrolled trials or patient surveys, and are therefore quite subjective, the consistency of these reports indicates that a considerable proportion of patients receive relief from their tinnitus when using their device. This relief appears to be a result of the effects of the electrical stimulus per se in addition to the masking properties of the electrically evoked auditory percepts.

Provided the patient is suitable psychologically to undergo cochlear implant surgery and the subsequent rehabilitation, and that there is a motivation on the part of the patient to use the device as an aid to communication, it would appear that the use of the cochlear implant would currently provide the most efficacious treatment of severe tinnitus in the profoundly and totally deaf. It should be emphasised, however, that should the patient view the procedure solely as a means of ameliorating tinnitus, there is a slight risk of exacerbating the condition. Moreover, unrealistic expectations of tinnitus amelioration may lead to an unfavourable outcome in cases where tinnitus is reduced but not eliminated.

**Conclusion**

Despite considerable research in this field, it is clear that the pathophysiology of tinnitus remains relatively obscure. This is due to the subjective nature of this complaint and the difficulty of identifying the multiplicity of potential generator sites within the auditory pathway. Significant improvement in the clinical management of tinnitus will only follow research directed at broadening our understanding of the underlying pathophysiology, and establishing safe means of moderating aberrant neural firing patterns in experimental animals. Our knowledge in this area can be complemented by recent experimental and clinical advances in the management of chronic pain. Because of the general lack of any predictive factors in the treatment of tinnitus, a more extensive diagnosis will be required in order to accurately establish the site of tinnitus generation. It is to be hoped that when we have achieved these goals we will be able to prescribe specific treatments for each of the many forms of auditory system pathology responsible for generating tinnitus.

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SHEPHERD, R.K.


Author/s: Shepherd, R. K.

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