Long-Term and Frequent Cellular Phone Use and Risk of Acoustic Neuroma

Ahmed Harith Salahaldin¹ and Abdulbari Bener^{2,3}

¹Department of Ear, Nose, and Throat, Audiology Unit, Rumailah Hospital and Hamad General Hospital, and ²Department of Medical Statistics and Epidemiology, Hamad General Hospital, Hamad Medical Corporation, Doha, Qatar, and ³Department of Evidence for Population Health Unit, School of Epidemiology and Health Sciences, University of Manchester, Manchester, United Kingdom

> Abstract: Human exposure to radio frequency radiation has increased dramatically during recent years from widespread use of mobile phones, and in some studies this exposure has been linked to the development of acoustic neuroma. The aim of our study was to describe the epidemiology of acoustic neuroma in a newly developed country, Qatar. We reviewed all cases of acoustic neuroma registered at the Hamad Medical Corporation during the period 2004-2005. We collected and assessed the sociodemographic information, presenting complaints, audiological evaluation, and laboratory investigations. During the study period, we diagnosed acoustic neuroma in 13 patients (10 women, median age 55 years, and 3 men, median age 49 years). Most of the cell phones were used daily for an average of 14 times per day (range, 8-20 times) and had been used for the duration of more than 5 years, with the exception of 3 patients who had used the cell phone excessively (>20 minutes per call more than five times daily) owing to the nature of their jobs. The total incidence rate for Qatar was found to be 17.2 per million population. In conclusion, the incidence of acoustic neuroma in Qatar is slightly higher than that in other countries. Despite the presence of facilities in Qatar, no proper screening and management protocol is available. This study highlights the need for the development and implementation of a national registry plan whereby effective care services can be delivered and high-risk groups can be targeted.

Key Words: acoustic neuroma; cellular phone use; epidemiology

An acoustic neuroma (AN) is a benign growth or tumor on the eighth cranial nerve (acoustic nerve) that leads from the inner ear to the brain [1]. Although not generally a life-threatening condition, ANs (sometimes also called *vestibular schwannomas* or *neurolemmomas*) expand in size and can push against the brain, while not actually invading it [2]. ANs are generally slow-growing, having average growth rates of 0.2 cm per year. However, growth rates in excess of 2 cm per year have been documented and, if a neuroma is not treated, it is potentially lethal. Gradual enlargement can occur, leading to indentation of the brainstem, increased intracranial pressure, and death over a course of 5–15 years [3]. The exact cause of ANs is unknown, and most occur spontaneously.

Most people with diagnosed ANs are between the ages of 30 and 60 years, and the incidence of ANs is slightly higher among women (60%) than among men (40%) [4]. Modern imaging tests now permit us to diagnose ANs earlier, when most tumors are smaller. Advances in microsurgery, including intraoperative monitoring of facial and cochlear function, have greatly reduced the risks of facial paralysis and hearing loss. Today, many tumors can be treated effectively with both surgery and radiotherapy.

Several authors have described and documented the association between ANs and cellular phone use, which, by exposing humans to radio frequency radiation, poses

<u>Reprint requests</u>: Prof. Abdulbari Bener, Department of Medical Statistics and Epidemiology, Hamad General Hospital, PO Box 3050, Doha, Qatar. Phone: 974-439 3765; 974-439 3766; Fax: 974-439 3769; E-mail: abener@hmc. org.qa; abaribener@hotmail.com

an important public health problem [2,5,6]. These authors have failed, however, to establish an actual link between the two, mainly because most of the studies were characterized by a small sample size, short duration of exposure and, sometimes, methodological problems.

Handheld mobile phones were introduced in Qatar during the late 1990s and soon became relatively common among the population [7]. Recently, Qatar's population increased dramatically as a result of revolutionary industrial and trade development associated with considerable economic growth and scientific development in that country. These changes also incited an increase in the use of cellular phones. Conversely, a steady increase in the number of patients with ANs—involving both local and expatriate populations—was detected. The aim of this study was to describe the epidemiology of ANs in newly and rapidly developing Qatar.

PATIENTS AND METHODS

This study was based at Hamad General and Rumailah Hospitals in Doha, Qatar. Both hospitals provide comprehensive tertiary health care services for the residents of Qatar; hence, they act as an ideal center for population-based studies. All patients with various hearing difficulties are treated at the audiology and otolaryngology departments of these hospitals.

We collected and recorded sociodemographic information, such as age at presentation, gender, and ethnicity, and data on presenting complaints that included hearing-loss tinnitus, dizziness, and other problems; duration of onset; mode of presentation; clinical findings that included ear, nose, and throat findings; and otoneurological evaluation history. Audiological evaluation included pure-tone audiometry, speech audiometry, a cochlear-retrocochlear test battery, tympanometry, videocomputer nystagmography, and testing for otoacoustic emissions, auditory brainstem evoked response, caloric and acoustic reflex, and reflex decay. We performed a series of laboratory investigations that included radiological imaging (e.g., skull, x-ray, computed tomography scan, magnetic resonance imaging [MRI], and Doppler study of the head and neck vascular system). We also analyzed a special report on MRI tumor size, extension, and the presence of pressure effect.

Data on follow-up, type of surgical procedure used, biopsy results, postoperative results, and information on the use of a cellular phone were also presented. It is worthwhile to note that in the year 2004, the population of Qatar was 755,163, and the total number of registered mobile phones was 532,141. In the total Qatar population, 70.5% have access to a cellular phone [7]. Most of these mobile phones were manufactured by Nokia in Sweden.

RESULTS

During the study period, we diagnosed AN in 13 patients (10 women, 3 men). The age range of female subjects was 34–66 years (median age, 55 years) and, for men, 34–66 years (median age, 49 years). All but 1 of the 13 patients had been complaining of hearing loss in one ear or the other; 12 had complained of gradual onset, and only 1 reported sudden onset. Tinnitus was a common complaint and occurred in 11 patients. Duration of onset of symptoms was 3 weeks in one patient, 7–8 months in two patients, and 1–10 years in four patients; it was unknown in six patients. The mode of progression of symptoms was mild in one patient, gradual in nine, sudden in one, and unclear in two.

Table 1 describes the audiometric configuration about the size and shape of the tumor. We carefully recorded a patient history of cellular phone use, and apparently all patients had used them. Most of the cell phones were used daily on an average of 14 times per day [range, 8– 20 times] and for a duration of more than 5 years, with the exception of three patients who had used the cell phone excessively (>20 minutes per call and more than five times per day) owing to the nature of their job.

We conducted MRI with enhancement on all patients; the results are shown in Table 2. The diagnosis of AN in 11 patients, based on clinical and foregoing investigation results and postoperative findings, was found to

Table 1.	Audiometric Configuration About the Size and	
Shape of	the Tumor	

Variables	Frequency	Percentage
Side		
Left	11	84.6
Right	2	15.4
Shape		
No response	1	7.7
Slope	8	61.5
Flat	4	30.8
Average hearing level (ISO)/speech		
No response	1	7.7
Normal	2	15.3
Mild	4	30.8
Moderate	3	23.1
Severe	3	23.1
Average hearing level (ISO)/ high frequencies		
No response	1	7.7
Normal	0	0.0
Mild	3	23.0
Moderate	4	30.8
Severe	4	30.8
Very severe	1	7.7
Glomus jugulare	2	15.3

ISO = International Standards Organization.

sureT1 WCT2 WCHomogeneity/ HeterogeneityectFindingsFindingsHeterogeneitypeduncleHypodenseHyperdenseHetero, enhancedpeduncleHypodenseHyperdenseIntense homo, postcontrastprodenseISO signalIntense homo, postcontrastIntense homo, postcontrastISO signalISO signalIntense homo, postcontrastIntense homo, postcontrastInterve VMixed flow streakISO signalIntense enhancementInerve VMixed Iso streakISO signalIntense enhancementInerve VIMixed Iso signal intensityIntense enhancementInerve VIILow signal intensityHigh signal intensityIntense enhancementstandReduced signal intensityHigh signal intensityIntense enhancement						
(mm)ShapeSiteEffectFindingsFindingsHetrogeneity $12 \times 20 \times 30$ $LCPA, IAM$ Cerebellar peduncleHypodenseHyperdenseHetro, enhanced -10 $LCPA, IAM$ $LCPA, IAM$ Cerebellar peduncleHypodenseHyperdenseIterse homo, postcontrast -10 $LCPA, IAM$ $R LAM$ $R CPA, IAM$ IsO signalISO signalIterse homo, postcontrast 3.4 $R LAM$ $R CPA, IAM$ $R CPA, IAM$ IsO signalIterse homo, postcontrast 3.4 $Leccream coneLCPA, IAMR CPA, IAMIsO signalIterse homo, postcontrast3.0Leccream coneLCPA, IAMR CPA, IAMEnhanced flow streakIsO signalIterse homo, postcontrast3.0LCPA, IAMR, CPA, IAMR, CPA, IAMRind IntensityIterse enhancement3.0LCPA, IAMR, CPA, IAMR, CPA, IAMRind IntensityIterse enhancement3.0LCPA, IAMR, CPA, IAMRind IntensityRigh Isignal intensityPromo2.0 \times 30 \times 30LCPA, IAMPons, CeHLow signal intensityRigh Isignal intensityPromo2.0 \times 2.09LCPA, IAMPons, CeH, andReduced signal intensityRigh Isignal intensityPromo2.0 \times 2.09LCPA, IAMPons, CeH, andReduced signal intensityRigh Isignal intensityReduced signal intensity3.0 \times 2.09LCPA, IAMPons, CeH, andReduced signal intensityRigh Isignal intensity$	Pressure	T ₁ WC	$T_2 WC$	Homogeneity/	Hypodense	
× 30L CPA, IAMCerebellar peduncleHypodenseHyperdenseHetero, enhancedL CPA, IAML CPA, IAMIntense homo, postcontrastIntense homo, postcontrastIntense homo, postcontrastR IAMR CPA, IAMISO signalISO signalIntense homo, postcontrastR CPA, IAML JF, CPA, IAMISO signalIntense homo, postcontrastL JF, CPA, IAML JF, CPA, IAMEnhanced flow streakISO signalIntense homo, postcontrastL JF, CPA, IAML CPA, IAMStrainal nerve VMixed ISO nitenseMixed bright ISO signalIntense enhancementx 30L CPA, IAMPons, CeHIow streakISO signalIntense enhancementx 30L CPA, IAMPons, CeHIow streakMixed bright ISO signalIntense enhancementx 30L CPA, IAMPons, CeHIow signal intensityHigh signal intensityIntense enhancementx 30L CPA, IAMPons, CeHIow signal intensityHigh signal intensityIntense enhancementLocarent ConeL CPA, IAMPons, CeHReduced signal intensityHigh signal intensityIntense enhancementL CPA, IAMPons, CeHPons, CeHReduced signal intensityIntense enhancementLocarent ConeL CPA, IAMPons, CeHReduced signal intensityIntense enhancementL CPA, IAMPons, CeHReduced signal intensityHigh signal intensityIntense enhancementL CPA, IAMPons, CeHReduced signal intensityHigh signal intensityIntense enhancement <th>Effect</th> <th>Findings</th> <th>Findings</th> <th>Heterogeneity</th> <th>Areas</th> <th>Ventricles</th>	Effect	Findings	Findings	Heterogeneity	Areas	Ventricles
LCPA,IAM LCPA,IAM Intense homo, postcontrast RIAM RIAM ISO signal ISO signal Intense homo, postcontrast RCPA,IAM RCPA,IAM Enhanced flow streak ISO signal Intense homo, postcontrast Ice-cream cone L.Fr,CPA,IAM Enhanced flow streak ISO signal Intense enhancement L.Fr,CPA,IAM Enhanced flow streak ISO signal Intense enhancement L.Fr,CPA,IAM BS,cranial nerve V Mixed ISO signal Intense enhancement ×30 L.CPA,IAM BS,cranial nerve V Mixed ISO signal Intense enhancement Rounded LCPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Rounded LCPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Rounded LCPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Ice-cream cone LCPA,IAM Pons,CeH, and Reduced signal intensity Intense enhancement Ice-cream cone LCPA,IAM Pons,CeH, and Reduced signal intensity High signal intensity Intense enhancement </td <td></td> <td>le Hypodense</td> <td>Hyperdense</td> <td>Hetero, enhanced</td> <td></td> <td></td>		le Hypodense	Hyperdense	Hetero, enhanced		
R1AM R1AM ISO signal ISO signal Intense homo, postcontrast R CPA, IAM R CPA, IAM Strong homo enhancement Strong homo enhancement L JF, CPA, IAM L JF, CPA, IAM Enhanced flow streak ISO signal Intense enhancement L JF, CPA, IAM L JF, CPA, IAM Enhanced flow streak ISO signal Intense enhancement A: JF, CPA, IAM L JF, CPA, IAM Mixed ISO intense Mixed bright ISO signal Intense enhancement X: Onded L CPA, IAM BS, cranial nerve V Mixed ISO intense Mixed bright ISO signal Intense enhancement Rounded L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH Iso signal intensity High signal intensity Intense enhancement I CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement I CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement	M	1		Intense homo, postcontrast		
RCPA,IAM Strong homo enhancement Ice-cream cone L.Fr,CPA,IAM Enhanced flow streak ISO signal L.Fr,CPA,IAM L.Fr,CPA,IAM Enhanced flow streak ISO signal X-30 L.Fr,CPA,IAM BS,cranial nerve V Mixed ISO signal Intense enhancement X > 0 L.CPA,IAM BS,cranial nerve V Mixed ISO signal Intense enhancement X > 0 L.CPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Rounded L.CPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Rounded LCPA,IAM Pons,CeH Low signal intensity High signal intensity Intense enhancement Rounded LCPA,IAM Pons,CeH Now signal intensity High signal intensity Intense enhancement I.CPA,IAM Pons,CeH Reduced signal intensity High signal intensity Intense enhancement I.CPA,IAM Pons,CeH Reduced signal intensity High signal intensity Intense enhancement I.CPA,IAM Pons,CeH Reduced signal intensity High signal intensity Intense enhancement		ISO signal	ISO signal	Intense homo, postcontrast		
Ice-cream cone L CPA, IAM Intense enhancement L JF, CPA L JF, CPA, IAM Enhanced flow streak ISO signal L JF, CPA, IAM L JF, CPA, IAM Mixed ISO intense Mixed bright ISO signal X 30 L CPA, IAM BS, cranial nerve V Mixed ISO intense Mixed bright ISO signal X 40 L CPA, IAM BS, cranial nerve V Mixed ISO intense PC enhancement Rounded L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH Reduced signal intensity Intensity Intense enhancement I CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement I CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement I CPA, IAM Pons, CeH Reduced signal intensity High signal intensity	Μ			Strong homo enhancement		
LJF, CPA LJF, CPA, IAM Enhanced flow streak ISO signal LJF, CPA, IAM BS, cranial nerve V Mixed ISO intense Mixed bright ISO signal X 20 L CPA, IAM BS, cranial nerve V Mixed ISO intense PC enhancement PC L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement CPA, IAM Pons, CeH ISO & reduced signal intensity High signal intensity Intense enhancement CPA, IAM Pons, CeH ISO & reduced signal intensity High signal intensity Intense enhancement CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement Ic-cream cone I.CPA, IAM Pons, CeH Pons, CeH Reduced signal intensity Intensity	M			Intense enhancement		
LJF, CPA, IAM LJF, CPA, IAM × 30 L CPA, IAM BS, cranial nerve V Mixed ISO intense Mixed bright ISO signal Intense hetero PC enhancement L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement CPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement LCPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement LCPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement LCPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement LCPA, IAM Pons, CeH Reduced signal intensity High signal intensity Intense enhancement		Enhanced flow streak	ISO signal			
 × 30 L CPA, IAM BS, cranial nerve V Mixed ISO intense Mixed bright ISO signal Intense hetero PC enhancement L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity R Pons, CeH, and Reduced signal intensity High signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity L CPA, IAM Pons, CeH ISO & reduced signal intensity 	IAM					
L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement L CPA, IAM Pons, CeH, and Reduced signal intensity High signal intensity Intense enhancement L CPA, IAM Pons, CeH IceAcrean cone ICPA, IAM Pons, CeH Ice-crean cone I. CPA IAM Revisem Revisem		V Mixed ISO intense	Mixed bright ISO signal	Intense hetero	Areas with	Fourth ventricle
L CPA, IAM Pons, CeH Low signal intensity High signal intensity Intense enhancement Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense enhancement L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity Intense L CPA, IAM Pons, CeH, and Reduced signal intensity High signal intensity Intense L CPA, IAM Pons, CeH Intense VII Intense Intense L CPA, IAM Pons, CeH Pons, CeH Intense Intense Intense Loc-cream cone I. CPA, IAM Revisetem Revisetem Intense Intense				PC enhancement	cystic component	
Rounded L CPA, IAM Pons, CeH ISO & reduced intensity High signal intensity L CPA, IAM Pons, CeH, and Reduced signal intensity High signal intensity L CPA, IAM Pons, CeH, and Reduced signal intensity High signal intensity L CPA, IAM Pons, CeH Intensity High signal intensity L CPA, IAM Pons, CeH Intensity Intensity Locareant cone L CPA, IAM Revised Revised		Low signal intensity	High signal intensity	Intense enhancement		
L CPA, IAM Pons, CeH, and Reduced signal intensity High signal intensity cranial nerve VII L CPA, IAM Pons, CeH Ice-cream cone 1. CPA IAM Brainstern		ISO & reduced intensity	High signal intensity			Fourth ventricle
L CPA, IAM Po Ice-cream cone T. CPA TAM Br		Reduced signal intensity	High signal intensity		Small cystic lesion,	Sixth ventricle
L CPA, IAM Ice-cream cone L CPA IAM	cranial nerve V				hypointense	
Ice-cream cone I. CPA. IAM						
	M Brainstem					

involve the left cerebellopontine angle (CPA) and internal auditory meatus (IAM) in 3 men. ANs in eight female patients affected the left ear in six and the right ear in two. Two additional female patients were found to have glomus jugulare tumors involving the CPA and IAM. MRI evaluation of the size of the lesions revealed small lesions (<10 mm) in three patients (27.3%), moderate lesions (20–29 mm) in another three patients (27.3%), and large lesions (\geq 30 mm) in five patients with AN (45.4%).

DISCUSSION

Overall prevalence of AN in our study was approximately 17.2 per million, as compared with 10.4 per million in northwest England in 1999, increasing to a rather comparable incidence of 14 per million in the last 5 years in England [8]. In our study, AN incidence was markedly higher among women (76.9%) than among men (23.1%), consistent with another reported study [4].

Most recently, a study by Lin et al. [9] showed that the prevalence of incidental ANs appears to be roughly 2 in 10,000 people. A recent study from the United States by Propp et al. [1] described an incidence of 0.6–0.8 per 100,000 person-years based on data from the Central Brain Tumor Registry and the Los Angeles Cancer Surveillance Program during the period 1995–1999. The various mean incidences ranging from 2 to 20 per million inhabitants per year from various periods and places indicate that the real incidence of AN is unknown and that huge differences in reported incidences exist today [10].

According to most recent studies [11,12], including our study, MRI with proper diagnostic criteria seems to be the most reliable single test both for the diagnosis and differential diagnosis of the disease and for early detection. The size of the tumor was small to moderate in diameter (<29 mm) in 54.6% of patients, whereas in 45.4% of patients the tumor was slightly larger (≥30 mm). The smallest lesions (<10 mm) were recently detected (in the last year), and this finding coincides well with other findings [13].

CONCLUSION

The incidence of ANs in Qatar is slightly higher than that in other countries (17.2 per million population). Despite the presence of facilities in Qatar, no proper screening and management protocol is available. This study highlights the need for the development and implementation of a national registry plan so that effective care services can be delivered and high-risk groups can be targeted.

ACKNOWLEDGMENT

We are very grateful to Dr. Ganesh Shanmugam, Department of Ear, Nose, and Throat, Rumailah Hospital, for providing and referring to us some patients.

REFERENCES

- Propp JM, McCarthy BJ, Davis FG, Preston-Martin S. Descriptive epidemiology of vestibular schwannomas. J Neurol Oncol 8(1):1–11, 2006.
- Christensen HC, Schuz J, Kosteljanetz M, et al. Cellular telephone use and risk of acoustic neuroma. *Am J Epidemiol* 159:277–283, 2004.
- 3. Hirsch BE, Cass SP, Sekhar LN, Wright DC. Translabyrinthine approach to skull base tumors with hearing preservation. *Am J Otol* 14(6):533–543, 1993.
- 4. http://www.acousticneuroma-info.com/guide_detail.php? gid= TL001&a=a. Accessed on August 9, 2006.
- Lonn S, Ahlbom A, Hall P, Feychting M, Swedish Interphone Study Group. Long-term mobile phone use and brain tumor risk. *Am J Epidemiol* 161:526–35, 2005.
- 6. Hardell L, Hansson MK, Sandstrom M, et al. Vestibular

schwannoma, tinnitus and cellular telephones. *Neuroepi*demiology 22(2):124–129, 2003.

- Bener A, Lajunen T, Ozkan T, Haigney D. The effect of mobile phone use on driving style and driving skills. *Int J Crashworthiness* 11(4):1–7, 2006.
- Evans DG. Incidence of vestibular schwannoma and neurofibromatosis 2 in the northwest of England over a 10year period: Higher incidence than previously thought. *Otol Neurootol J* 26(1):93–97, 2005.
- Lin D, Hegarty JL, Fischbein NJ, Jackler RK. The prevalence of "incidental" acoustic neuroma. Arch Otolaryngol Head Neck Surg 131(3):241–244, 2005.
- Tos M, Stangerup S, Caye-Thomasen P, et al. What is the real incidence of vestibular schwannoma? *Arch Otolaryngol Head Neck Surg* 130(2):216–220, 2004.
- 11. Gillespie JE. MRI screening for acoustic neuroma. *Br J Radiol* 73:1129–1130, 2000.
- 12. Zealley IA, Cooper RC, Clifford KM, et al. MRI screening for acoustic neuroma: A comparison of fast spin echo and contrast enhanced imaging in 1233 patients. *Br J Radiol* 73:242–247, 2000.
- Stangerup SE. Increasing annual incidence of vestibular schwannoma and age at diagnosis. J Laryngol Otol 118(8):622–627, 2004.