American Academy of Otolaryngology—
Head and Neck Surgery Foundation

Geriatric Care Otolaryngology

American Academy of Otolaryngology—Head and Neck Surgery
Working for the Best Ear, Nose, and Throat Care
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Introduction

Americans Are Growing Older—Are Physicians Ready?

Data from the U.S. Bureau of the Census, the World Health Organization, and the United Nations on U.S. and global trends in aging indicate that, in the United States, the proportion of the population aged 65 and older is expected to increase from 12.4 percent in 2000 to 19.6 percent in 2030, which translates into approximately 71 million persons. The number of those 80 years and older is expected to increase from 9.3 million to 19.5 million in 2030.

Concurrent with this substantial growth in the elderly population will be a requirement that physicians caring for geriatric patients must take into account the physiological changes in this demographic group. The senior patient often presents co-morbidities, many of which occur only in old age. Illnesses in the elderly can also exist with unusual symptoms or without common symptoms, and medical therapy may be difficult to prescribe because of possible adverse effects resulting from a combination of necessary medications. Surgical procedures in the elderly should be performed with caution, but outcome studies have proven time and again that healthy elderly patients continue to have surgical procedures performed without suffering major complications. Hence, age should not always be a deterrent in performing needed surgery on an elderly patient.

There is other good news regarding medical care for America's senior citizens. Of significance is that elderly Americans are becoming more knowledgeable regarding their health and treatment options. Already we find that an informed population of senior citizens will not only seek treatment for life-threatening medical disorders, but will also seek treatment for medical conditions that have

a significant impact on their quality of life. Many of these disorders occur in the ear, nose, throat, and related structures of the head and neck, thereby requiring the support of an otolaryngologist—head and neck surgeon.

The otolaryngologist—head and neck surgeon plays a key role in the diagnosis and treatment of disorders in the head and neck area. These specialists provide early detection of cancers; address vestibular and hearing problems; perform facial plastic and reconstructive procedures that can improve a patient's outlook; and conduct needed treatment of middle ear infection, sinusitis, and upper respiratory infections that can improve quality of life.

The American Academy of Otolaryngology—Head and Neck Surgery, the national medical society for more than 12,000 ear, nose, and throat specialists, previously produced *Primary Care Otolaryngology*, a guide for clinicians regarding the unique requirements demanded in diagnosing and treating ear, nose, and throat disorders. The Geriatric Committee of the Academy, with the support of the John Hartford foundation of the American Geriatric Society, now introduces an online publication, Geriatric Care Otolaryngology, as a companion to that monograph. This publication aims to provide expert guidance regarding the unique requirements for the diagnosis and treatment of ear, nose, and throat disorders in the elderly. The publication includes self-tests to add to the reader's educational experience.

A collection of essays that address the special considerations necessary for the diagnosis and development of treatment paradigms for the elderly are included in this online primer. They are authored by leading clinicians in the treatment of ear, nose, and throat disorders.

As the average age of our population increases, both otolaryngologist—head and neck surgeons and primary care providers will treat more elderly patients for head and neck disorders. The challenge for both specialists and generalists will be to offer the appropriate diagnosis and treatment for a patient who has undergone significant physiological changes. This online primer is an important first step for the American Academy of Otolaryngology—Head and Neck Surgery in identifying the special requirements for effective treatment of ear, nose, and throat disorders for the geriatric patient. It is the hope that Academy members and their physician colleagues will find the essays by experts in the field useful in their administration of patient care.

The essays are:
Essay 1.
Hearing
When Surgery Is Appropriate for Age-Related Hearing Loss
More than 28 million Americans have some degree of hearing loss. In some, hearing loss is caused by a medical disorder that can be treated by a surgical intervention. This essay discusses, among other things, the age when such surgery can be safe and effective.

Essay 2.
Head and Neck Cancer
Quality of Life following Chemoradiation Therapy for Head and Neck Cancer
A combination of chemotherapy and radiation has given hope to those diagnosed with this most deadly of cancers, in which more than one-half of all patients are older than 65 at the time of original diagnosis. The author discusses the severe impact this treatment has on quality of life, and the need to share this information with patients.

Essay 3.
Voice
Dysphonia and the Aging Voice
The loss of voice quality can be as devastating to the elderly patient as hearing loss. A leading expert in voice care discusses the diagnosis and treatment of this condition.

Essay 4.
Swallowing
Patient Safety and Medicinal Therapy for Ear, Nose, and Throat Disorders
Swallowing disorders can be debilitating or may necessitate a visit to an emergency room. This essay covers the risk factors and incidence of dysphagia among the elderly.

Essay 5.
Facial Plastic Surgery
The Aging Face—
Benefits and Pitfalls of Botox® and Laser Skin Treatments
An important discussion of cosmetic surgery procedures and nonablative techniques that have proven to be highly popular with an aging American population.

Essay 6.
Rhinosinusitis
Surgical Management of Chronic Rhinosinusitis in the Geriatric Patient
Thirty-seven million Americans suffer from acute or chronic rhinosinusitis. When medical therapy fails, surgery may be necessary. A leading expert discusses when the senior patient is a suitable surgery candidate and the appropriateness of functional endoscopic sinus surgery.

Essay 7.
Sleep Disorders
The Most Effective Treatments for Snoring and Sleep Apnea
New studies reveal that obstructive sleep apnea, prevalent among the elderly and obese, can lead to more severe illnesses and even death. New treatments are now available, but who should be a candidate for these new procedures?

Essay 8:
Geriatric Polypharmacy in Otolaryngology
Patient Safety and Medicinal Therapy for Ear, Nose, and Throat Disorders
Often medical therapy is the most effective treatment for ear, nose, and throat disorders. In suggesting therapies, consideration must be given to medications prescribed for other medical disorders.

Hearing

When Surgery Is Appropriate for Age-Related Hearing Loss

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After reading this presentation the reader will understand the general operation and the audiometric and surgical indications for semi-implantable hearing aids, and be able to identify which patients meet the audiometric criteria for cochlear implantation.

Historical Perspective

Traditional teaching has been that surgery was for conductive hearing loss and hearing aids were for Sensorineural Hearing Loss (SNHL). This notion is changing. This paper will provide information on most of the current indications for semi-implantable hearing aids and cochlear implants. Hearing improvement devices (hearing aids, surgical improvements, cochlear implants) will see increasing clinical application, so knowledge about their application is essential for otolaryngologists.

There are four main roles possible for surgery in SNHL:

1. cochlear implants and auditory brainstem implants;

2. implantable hearing aids and semi-implantable hearing aids;

3. bone-anchored hearing aids, although the main indication is conductive hearing loss; and

4. application of medications to the inner ear with wicks and sponges, which are not discussed in this article.

The results of auditory brainstem implants are less impressive than those of cochlear implants and the patient numbers is small so this article will not deal with auditory brainstem implants. The severity of hearing loss as summarized in Table 1 is the most important factor in determining which modality is appropriate.
The common vernacular for cochlear implantation indication (which is now outdated) as a severe-to-profound hearing loss means that the loss is greater than 70 dB.

New implantable hearing devices must undergo the Food and Drug Administration (FDA) approval process.[1] After the sponsor (manufacturer, physician, or medical center) develops the idea, the sponsor typically meets with FDA staff, who make recommendations on how to proceed depending on the nature of the new device. A new device is granted an Investigational Device Exemption (IDE) to conduct a trial on a small number of patients with approval of the investigator's Institutional Review Board (IRB). If the pilot study results show no particular safety issues, then a multi-institutional study is performed, which may lead to a premarket approval (PMA) study. The PMA study is an examination of the safety and efficacy of the device and can lead to what is typically referred to as FDA approval. For devices that are similar to currently approved products, the FDA may issue a clearance that also allows sale of the device.

Table 1: Hearing loss severity

Severity of Loss	Threshold (dB HL)
Normal	20
Mild	21-40
Moderate	41-55
Moderately severe	56-70
Severe	71-90
Profound	90

Cochlear Implants

The key concept of cochlear implants is that the hair cells and/or spiral ganglion neurons are stimulated electrically rather than by sound. Hearing aids amplify sound and stimulate the cochlea acoustically. This difference in stimulation mode means that the cochlear implant is not a hearing aid.

The concerns and uses are significantly different.

A typical cochlear implant consists of the following components:

1. A microphone, which converts sound energy to electrical energy. Some systems have two directional microphones but most have one omnidirectional microphone. The microphone(s) can be connected by cable or directly to the speech processor.

2. A speech processor, which contains a computer chip to digitize the electrical signal from the microphone and process it according to variable programmed instructions. The speech processor connects to the behind the ear housing if it is not physically part of the housing, and then to the transmitter.

3. A transmitter, which is an externally worn object that transmits the signal to the underlying receiver/stimulator. Transmission is usually transcutaneous through intact skin via FM radio waves, but in the past was percutaneous (through a surgical opening in the skin). It is held in place with a magnet.

4. A receiver/stimulator, which is implanted under the skin, typically superior and posterior to the pinna. It receives the FM electrical signal and connects to the electrodes in the cochlea. Some receiver/stimulators have ground electrode and neural response telemetry capability.

Indications for Cochlear Implants

Evolution of the indications for cochlear implant are reflected in the latest recommendations from the FDA[2]. Initial criteria were bilateral "severe to profound hearing loss" (90 dB) then "severe to profound" (70 dB) but currently the main criterion is poor speech understanding using sentence material. There are many cochlear implant candidates who have better thresholds than 70 dB but have poor speech recognition.

Adults Criteria

1. Be 18 years or older, with bilateral, severe to profound sensorineural hearing loss, i.e., 70 dB or greater PTA at 500, 1000, and 2000 Hz;

2. Have tried but have limited benefit from an adequately fitted binaural hearing aid; or

3. Have sentence recognition score of 50 percent or less in the ear to be implanted and 60 percent or less in the contralateral ear in best-aided conditions using HINT or CUNY tests.

Pediatric Criteria

1. Be 12 months to 17 years of age.

2. Infants age 12-24 months should have bilateral, profound hearing loss with thresholds of 90 db or greater at 1000 Hz.

3. Children 24 months to 17 years should have bilateral severe to profound (greater than 70 dB) hearing loss.

4. Infants and older children should demonstrate lack of progress in simple auditory skills in conjunction with appropriate auditory amplification and participation in intensive aural habilitation for three to six months. Less than 0.14520 percent correct on the Multi-syllabic Lexical Neighborhood Test (MLNT) or Lexical Neighborhood Test (LNT), depending on the child's cognitive and linguistic abilities.

5. A three- to six-month trial of appropriate hearing aids is required. If meningitis is the cause of hearing loss or if there is radiologic evidence of cochlear ossification a shorter hearing aid trial and earlier implantation may be reasonable.

Note: The earliest time for implantation is now as low as 12 months. Difficulty in determining the severity of the loss and a meaningful trial of hearing aids can be problems at this age.

Cochlear Implant Results

Results of cochlear implantation have been impressive. An important measure of success of cochlear implantation is the ability to understand speech in the absence of speech-reading or other cues, such as when using a telephone.

Cray found that 95 percent of cochlear implant recipients could identify a dial tone, a busy signal, and voices.[3] Average telephone use per week was 5.4 hours and 85 percent could interact with strangers on the telephone within five months of receiving the sound processor.

Approximately 30 percent communicated via a cellular phone for personal use. Telephone use had increased over the past decade. Of course these excellent results may not always be achievable. Outcomes depend greatly on the nature of patients implanted, severity of hearing loss, quality of post-implant rehabilitation, and a variety of other factors. In fact, if results are too good it can be argued that implant criteria may be too strict, eliminating some candidates who may not be "stars" but still have significant benefit.

Cochlear Implant Issues

Some cochlear implant controversies persist. Sometimes it is not clear which ear to implant when one ear has more residual hearing than the other.
The role of cochlear implants in children with multiple handicaps or in prelingually deafened patients is challenging. The deaf community still has some reservations about the use of cochlear implants as reviewed by Hintermair and Albertini [4]. They felt that parents are being forced to make decisions for their children without adequate information about alternatives and that they are unprepared for the consequences of these decisions.

Cochlear Implant Complications

In one representative study, 2 of 30 children who received a CI developed meningitis.[5] In a survey of Latin American cochlear implant centers, of 3,768 cases reviewed, the following complications occurred: migration, 13 cases (0.35 percent); extrusion, 15 cases (0.4 percent); implant failure secondary to trauma, 18 cases (0.48 percent); device failure, 86 cases (2.28 percent); skin inflammation by magnet, 35 cases (0.9 percent); and infection, 26 cases (0.7 percent).[6] Even though cochlear implantation is the only reasonable option for many patients, the possibility of complications and poor results should be kept in mind.

Future of Cochlear Implants

Cochlear implant technology continues to evolve but the main reason for improvements in cochlear implant performance appears to be the liberalization of patient selection criteria. Indications for cochlear implants have started to overlap with those for hearing aids. It is unlikely that large numbers of patients will choose cochlear implantation if a hearing aid would serve them just as well. For this reason it is likely that technology rather than patient selection will drive further improvements. Promising research regarding hair cell regeneration and cochlear physiology will likely impact cochlear implantation in the future.

Implantable and Semi-implantable Hearing Aids

Sound is the sensation of vibration. Mechanical stimulation of the cochlea with an implantable device is a natural, logical way to augment hearing.

Loudspeakers and hearing aids introduce acoustic distortion so that "high-fidelity" amplification is problematic. The physical basis for middle ear hearing devices has been reviewed by Spindel.[7]
Hearing aids, including implantable ones, sense sound energy using electrical techniques, amplify and/or otherwise process it, and then convert the energy back to a mechanical form that stimulates the cochlea.

Conventional hearing aids provide acoustic stimulation whereas implanted hearing aids typically provide direct mechanical drive to an ossicle using some attached device.

The two main electromechanical techniques used in implantable hearing aids are *piezoelectric and electromagnetic transduction*.

Piezoelectric transduction exploits the reciprocal relationship between electrical current and physical movement in certain materials. By bonding two piezoelectric materials together, a bimorph is created that vibrates in proportion to the current or voltage applied. Piezoelectric transducers can be of the "diaphragm" or "springboard" types but the principle is similar.

Piezoelectrics are precise, small, and accurate. Typically they have low power requirements so battery life can be prolonged. They also have disadvantages. For example, one part of the transducer must be rigidly fixed to the skull and the other to an ossicle, which causes ossicular

loading. If there is no current or insufficient current, the piezoelectric component is stiff and does not move. In the middle ear, this means that the device could actually affect hearing adversely. "No current" situations could occur if the battery is depleted or removed, if the user removes the external part of the device, or if the device fails. Device failure has occurred in some early models and is of concern. Some designs require surgical ossicular disruption, which should be considered carefully in case the device fails or must be removed.

Electromagnetic transduction relies on the movement of a magnet in an electrical field provided by wire coil as in a home stereo speaker. One end of the electromagnet device can be attached to an ossicle. Unlike the piezoelectric device, the other end does not need to be attached to the skull. The magnet can be outside the wire coil, called "extra-coil," or inside the coil, called "intra-coil."

The surgical technique for electromagnetic devices can be simpler than for piezoelectric devices depending on design. The magnet can remain in place and the rest of the unit removed in case of device failure. The magnet is attached to an ossicle and ossicular disruption may not be needed for electromagnetic devices. The magnet can be very lightweight so there is minimal loading on the ossicle.

The electromagnetic design also has disadvantages. In general, greater power is required for electromagnetic than for the piezoelectric design, so battery life is shorter. For optimal efficiency the coil and the magnet axes must be co-axial. In the intra-coil design this is inherent, but in the extra-coil design this can be a problem. In one extra-coil design only the magnet is implanted into the middle ear with some distance between the coil placed in the ear canal and the magnet, which can be attached to an

ossicle. The distance between the coil and the magnet must be very small, because the power drops off in proportion to the square of the distance between them. Minimizing the distance between the coil and magnet means that such a device has to be worn deep in the ear canal. If the user does not consistently insert the device to its full depth, adverse function can occur.

The intra-coil design permits perfect alignment of the coil and the magnet and removes the variability produced by the distance between them. The intra-coil device requires that the coil and magnet be attached to an ossicle whereas the extra coil design requires attachment of only the magnet. This design difference means that the intra-coil device is heavier than the extra-coil device and may produce some "loading" weight on the ossicle with resulting hearing loss. In addition, the intra-coil device must be hardwired to the processor, making the surgical implantation more complex.

Advantages of the implanted or semi-implanted hearing aids over conventional in-the-ear aids include :

1. better sound clarity,
2. avoidance of the occlusion effect,
3. reduced feedback,
4. greater functional gain,
5. perceived benefit in many listening situations (reverberation, background noise, sound distortion, and speech perception), and
6. reduced problems with cerumen and moisture issues because the external ear canal is open

Disadvantages of implanted or semi-implanted hearing aids include the following:

1. Surgery is required so implantation mandates greater concern about choosing a hearing aid than for conventional, removable hearing aids.
2. Surgical implantation may require ossicular loading and/or ossicular disruption, which could be irreversible. The usual risks of infection and other surgical complications are present.
3. Battery power and longevity of the device may be poor.
4. Long-term performance is unknown.
5. The required physical dimensions of the device must be small. This limits acoustic output so the devices are less useful as hearing loss becomes more severe.

6. Most implantable hearing aids are not compatible with magnetic resonance imaging (MRI) scans.

7. Implantable aids are typically more expensive than conventional aids. Medical insurance for repair and surgical removal for replacement are problems. Reinsertion may not provide results similar to those obtained after initial application.

8. Many implantable or semi-implantable hearing aids are not FDA approved.

Bone Conduction

Bone conduction hearing aids must drive the entire skull so their power requirements are great. They have been traditionally used for conductive hearing losses but expanded indications for bone-anchored hearing aids are developing for sensorineural hearing loss as well. Single-sided deafness (SSD) from pathology, such as acoustic neuromas, or sudden sensorineural hearing loss are recent rationales made for bone conduction aids. This indication is controversial. Reported benefits for SSD patients include better sound localization and better sound clarity. Classic thinking has been that bone stimulates both cochleae simultaneously. If one cochlea does not work, it seems that the functioning cochlea would be fully stimulated or perhaps even overstimulated before effects are seen on the deaf side. It has been argued that a contralateral routing of signals (CROS) hearing aid would have the same effect.

On the other hand, many persons with SSD report improved speech understanding when using a special bone-anchored hearing aid test device. Are they impressed by a novel toy with a different frequency response than they are used to? Do they like the bone-anchored device because the occlusion effect and acoustic distortion produced by hearing aids are absent? If bone-anchored hearing aids provide better hearing for patients with SSD, why wouldn't they be better for bilateral sensorineural losses as well? Currently the answers are not known but evidence is accumulating and studies should soon be available to address the issue.

Devices

Bone-Anchored Hearing Aid (BAHA)

The principal use for bone conduction hearing aids, including the BAHA, is conductive hearing loss. Traditional bone conduction hearing aids must deliver the sound stimulus across the skin and other soft tissues before reaching the bone, which attenuates the sound. Although increased power may partially overcome this attenuation, it is frequency dependent so distortion may occur. The magnitude of the problem varies among different patients because the tissue thickness and acoustic impedance properties vary. Variable acoustic impedance caused by skin results in variable phase shifts and intensity attenuations so amplification becomes complex. Electrical power needs for bone conduction aids are great, so battery life and user satisfaction are problems.

The BAHA stimulates the bone directly with a metal screw through the skin and into the skull. Surgery is required to place the metal fixture. A screw that is

osseointegrated provides better results that a simple screw. Osseointegration allows bone to grow tightly to the artificial implant. The titanium screw is called the "fixture" and this attaches to an "abutment" to which the patient attaches the external device.

For most patients the surgical procedure has been simplified from a two-stage procedure to a one-stage procedure. A very thin skin flap is created, currently with a dermatome, followed by careful drilling and tapping of a hole in the skull. In the one-stage procedure a dermatome is used to elevate a thin skin flap so that the device will be 50-55 mm from the external auditory canal. Then subcutaneous tissue deep to the flap is removed with a wide margin. A hole, three or four millimeters deep, is drilled with the guide drill, then countersunk. The fixture is placed with a slowly rotating drill. Finally, a biopsy punch is used to make an opening in the flap before closure. The procedure is followed by a waiting period of three to four months to allow osseointegration before using the device. The two-stage procedure is still recommended for children, and for patients with poor or thin bone or irradiated bone. After placement of the device the skin flap is replaced for three to six months to allow osseointegration. Then the fixture is identified, a cover screw removed, and the abutment fitted. The processor lasts about five years.

The BAHA is FDA approved for conductive hearing losses caused by congenital auditory atresia or conductive hearing loss in patients who have chronic ear infections (chronic otitis media or external otitis), in whom placing a traditional hearing aid would cause repeated infections. It is approved for children older than five years of age with single-sided sensorineural deafness.

The device is contraindicated for an average bone threshold worse than 45 dB at 0.5, 1, 2, 3 kHz for BAHA Classic 300, and Compact, and worse than 70 dB for the more powerful, body-worn Cordelle. It is also not recommended for persons who are noncompliant or have psychological problems.

In one study, all seven previous bone conduction aid users were satisfied with a BAHA. Five of 16 (31 percent) air conduction hearing aid users were not happy with their BAHA and reverted to their aid. There were no predictive factors.[8]

Recent reports indicate that persons who hear in only one ear can achieve improved sound localization and clarity of sound using BAHA on the same side of the skull as the hearing loss. In general there should be a credible effort to use a conventional hearing aid before BAHA is performed. The use of BAHA for tinnitus treatment has been recommended as well, but the rationale for this application is unclear and the literature thus far is not strongly supportive.

Does BAHA improve sound localization? CROS aids were compared to BAHA by Niparko et al., who found that BAHA delivered superior performance than the CROS hearing aid. However, they recommended longer follow-up to assess whether the advantages of the bone-anchored hearing aid outweigh the disadvantages of implantation surgery, costs, and device maintenance.[9] Wazen et al. reported that sound localization was not improved in BAHA patients implanted for SSD.[10]

The advantages of the BAHA over other hearing aids include:

1. greater sound clarity and comfort than other bone conduction aids,
2. avoidance of the occlusion effect,
3. absence of feedback,
4. absence of any foreign objects in the ear canal that may cause external otitis,
5. the low risk of the surgery, and the compatibility of the device with MRI scans.

BAHA has disadvantages that include the following:

1. surgery is required. Infection is possible but rates are low;
2. bone conduction thresholds must be good, otherwise stimulation is ineffective;
3. battery life is short;
4. occlusion effect is absent or minimal in conductive hearing losses.;
5. device may fail to osseointegrate. This may occur in 1-2 percent of normal persons and in up to 25 percent of children or of persons with poor-quality bone or thin skulls.

6. there can be psychological adjustments to having a screw in the skull.
7. some care to the operative site is needed and the patients typically cannot see that site themselves.
8. battery life is short (but batteries are readily available and changeable).
9. If the user does not use the device, removal can be difficult.

Soundbridge

Med-El Corporation is now responsible for the Vibrant Soundbridge device. It is a semi-implantable hearing aid that received FDA approval in 2000. The device uses an intra-coil type of electromagnetic Floating Mass Transducer (FMT).

The FMT is surgically attached to the incus and connects to the internal processor via a hardware.
Batteries last 12-16 days.

The Soundbridge consists of two parts:

1. the internal implant called the vibrating ossicular prosthesis (VORP) (implanted receiver unit, conductor link, and FMT); and
2. an external amplification system called the audio processor (microphone, sound processing system, modulator circuit, and battery).

The use of any hearing device mandates that a hearing loss be present; however, if the hearing loss is too severe, power and physical limitations may make a semi-implantable device, such as the Vibrant Soundbridge

preferable to a fully implanted device. According to FDA (2000) recommendations, a Soundbridge device may be indicated in persons 18 years of age and older with moderate to severe (40 to 70 dB) sensorineural hearing loss who desire an alternative to an acoustic hearing aid.[11] It is recommended that individuals have experience with a properly fitted acoustic hearing aid. Implantation should be done in the worse ear.

Detailed audiometric indications for the Vibrant Soundbridge are listed below.

1. Air conduction thresholds are in the range shown in table 2.

2. The pure tone average (500, 1000, and 2000 Hz) is greater than 30 dB in the ear to be implanted and the asymmetry of PTA between the two ears is less than 20 dB.

3. Air/bone gap is less than 10 dB.

4. Speech recognition score is 50 percent or greater.

5. Appropriate hearing aids have been used for four hours a day for at least three months.

6. The candidate has normal anatomy, has not undergone ear surgery, is 18 years or older and psychologically stable, and has no other ear disorders.

Contraindications for Soundbridge include conductive hearing loss, retrocochlear pathology, pychological problems, mental retardation, inability to follow up, or

skin conditions that would be aggravated by the magnet. The device appears to have a small ossicular loading effect, on the order of 2 dB.[12]

Table 2: Air conduction range for Soundbridge device

Frequency (Hz)	500	1000	2000	3000	4000	6000
Lower limit (dB HL) 10	10	15	25	40	40	
Upper limit (dB HL) 65	75	80	85	85	85	

Soundtec ® Direct System

The Soundtec® Direct System device is an extra-coil electromagnetic device. A rare-earth magnet is surgically fixed to the incus and an attachment ring in the incudostapedial joint space. Placement requires ossicular disarticulation. The inductive coil is placed in a fitted ear mold deep in the ear canal to come as close as possible to the magnet. Although the occlusion effect could be present, there should be no acoustic feedback because the stimulus is not acoustic. The surgical proce
dure is easier than for any other implanted hearing aid.

Indications for The Soundtec ® Direct System device are:[13]

1. bilateral, symmetric, moderate to moderately severe sensorineural hearing loss (see table 3).

2. bone conduction thresholds within 15 dB of air conduction thresholds.

3. high-frequency (1, 2, and 4 kHz) averages between 35 and 70 dB.

4. discrimination scores 60 percent or better.

5. duration of hearing loss for two years without fluctuation.

6. at least six months of hearing aid use and at least 45 days in the ear to be implanted.

7. adequate ear canal size, motivation, cognitive skills, age 21-80 years.

8. dissatisfaction with conventional hearing.
Exclusion criteria for The Soundtec ® Direct System ® device include otitis externa, otitis media, retrocochlear pathology, malformations, previous middle ear surgery or disabling tinnitus, asymmetry of the high frequency average greater than 15 dB conductive, and unilateral or fluctuating hearing loss.

Table 3: Air conduction range for Soundtec device

Frequency (Hz)	500	1000	2000	3000	4000	6000
Lower Limit (dB HL) 0	10	35	50	50	40	
Upper limit (dB HL) 60	70	75	75	80	110	

Compared with optimally fit hearing aids, Hough reported that, for the subjects reviewed, The Soundtec ® Direct System ® DDHS provided an average improvement of 52 percent in functional gain (250-6000 Hz), 22 percent in aided thresholds, 3.8 percent for speech discrimination in quiet, 17 percent for speech in noise, 13.1 percent in articulation index scores, 28 percent in aided benefit, 27.3 percent in sound quality of speech, and a 16.7 percent

increase in overall subject satisfaction.[14] In addition, with The Soundtec ® Direct System ® DDHS, subjects reported absence of acoustic feedback, little or no occlusive effects, and more natural sound perception.

Envoy Device

The Envoy device (St. Croix Medical) uses two piezoelectric crystals—the "driver" and the "sensor"—but is not yet FDA approved. The sound sensor is attached to the incus. This piezoelectric detects sound, converts it to an electrical signal, and sends the electric signals to the sound processor. The sound processor amplifies, filters, and otherwise processes the electric signal and then sends the modified electrical signal to the second "driver" piezoelectric on the stapes.
Division of the incudostapedial joint and resection of part of the incus is required. The entire device is implanted but can be modified externally. The battery must be replaced under local anesthesia every three to five years. The device is recommended for mild to severe sensorineural hearing loss.

In a phase I trial with the Envoy device, Chen found that patients perceived benefits from the device including communication in background noise and word recognition.[15] Functional gain and speech reception thresholds were similar with the Envoy device and conventional hearing aids. Five of seven patients with Envoy implantable hearing aids had working devices at two months postactivation The authors concluded that feasibility was demonstrated but it appears that more data are needed on this device.

Otologics Device

The Otologics Middle Ear Transducer (MET) Ossicular Stimulator device involves the placement of an ossicular

stimulator directly into the body of the incus with a laser-drilled hole. Access is obtained via a mastoidectomy. The Otologics device has the CE mark but is not FDA approved. Several screws are placed into the simulator to firmly anchor it to the skull. The patient wears an external button device that contains a microphone, battery, and digital signal processor. The button detects sound, converts it to electrical energy, and transmits it across the skin to the internal processor. The internal processor connects to the MET.

Indications for the MET Ossicular Stimulator include:

1. bilateral moderate to severe (40-90 dB), nonfluctuating, nonprogressive, symmetric sensorineural hearing loss.
2. normal tympanogram.
3. no middle ear disease.
4. speech recognition scores better than 20 percent at 65 dB SPL.
5. postlingual, English speaker with good cognitive function.
6. auditory thresholds within limits shown in table 4.

Table 4: Air conduction ranges for the Otologics device (adapted from manufacturer's graph)

Frequency (Hz)	500	1000	1500	2000	3000	4000
Lower Limit (dB HL) 15 Upper limit	45	55	55	55	55	

Note that the MET Ossicular Stimulator has a larger upper limit threshold criteria than for implantable hearing aids so it may be useful in patients with larger hearing loss.

The Otologics device appears to provide high gain and wide-band amplification, but has some apparent disadvantages. The surgical procedure is elaborate and the large amount of implanted metal is discouraging. The instrument drives the incus directly so it may be useful for larger hearing losses (compare tables 1 and 2) but ossicular loading is expected.

Other Devices

The German Implex AG device involves stimulation of the incus by a diaphragm-type piezoelectric driver. There were some problems with feedback so a piece of the malleus was removed but the device is not on the market today. The Rion device from Japan uses a piezo-ceramic rod that is connected to the stapes with a hydroxylapatite strut and transcutaneous electrical stimulation of an internal receiver with an external audio processor. This device in not FDA approved.
The RetroX device from Belgium is a microphone, amplifier, processor, and titanium tube that connects the external auditory canal to the postauricular area.
The ear canal is unoccluded so that sloping high frequency hearing losses may be particularly well aided. One study reported gains of only 9-10 dB, but improvement in understanding of speech in noise.[16,17]

Of 25 patients implanted with the RetroX device, another report indicated that four required explantation because of granulation tissue, two patients complained of acoustic feedback and needed supplementary fitting, and yet 23 of 25 subjects were either satisfied or even extremely

satisfied with the RetroX device. They reported improved hearing at 1, 2, 4, and 8 kHz. In quiet, the speech reception thresholds improved by 10 dB SPL. Speech intelligibility in noise improved by 15 percent for signal-to-noise ratios between -5 dB and +5 dB.[17]. Overall, it appears that the RetroX device needs further evaluation. The device is not FDA approved.

The Tubingen device is a totally implantable communication assistance (TICA) device. The microphone picks up the sound signal transcutaneously from the external auditory canal, and amplifies and transduces the signal to vibrate the ossicular chain.

Future of Surgery for Sensorineural Hearing Loss

There may be an increasing role for surgery in treatment of sensorineural hearing loss. It seems likely that implantable aids will be increasingly common as technology improves, but they will never replace conventional hearing aids.

The technology of cochlear implants will continue to improve, but the criteria are unlikely to overlap further with that of hearing aids. Probably the greatest advances in sensorineural hearing loss treatment in the future will be found in the application of drugs to the inner ear to promote hair cell growth. Such drugs may be administered either systemically or via tympanotomy, in which case surgery will be required.

Quiz

1. A 50-year old man asks if a cochlear implant would help. He has bilateral sensorineural hearing loss and is having lots of trouble using his hearing aids. His thresholds are in the 60-65 dB range but his speech recognition scores are only 20 percent. Is he a candidate?

Answer: His thresholds are better than the usual criteria allow, but his speech recognition scores are below 50 percent so he is a suitable candidate.

2. What is the role for a bone-anchored hearing aid in sensorineural hearing loss?

Answer:
Although the BAHA is most widely accepted for conductive hearing loss, it has been applied with success for single-sided deafness due to many causes—acoustic neuroma, trauma, sudden sensorineural hearing loss, or others.

3. A 45-year-old woman is not happy with her speech understanding with her in-the-canal hearing aid. Her thresholds are symmetric at about 45 dB. Are there surgical options for her?

Answer:
A semi-implantable hearing aid may reduce the occlusion effect and is an option. Her auditory thresholds are appropriate.

4. What are the current audiometric indications for cochlear implantation in adults?

Answer:
The main indications are severe to profound hearing loss (PTA at 500, 1000, and 2000 Hz 70 dB or greater) OR a speech recognition score of 50 percent.
The latter indication is becoming more common.

5. How do semi-implantable hearing aids work?

Answer:
Electomagnetic or piezoelectric techniques stimulate the ossicular chain and sometimes act as sound sensors as well.

6. What implantable options are there for an adult with chronic ear disease and symmetric, bilateral, mixed hearing loss with thresholds in the 60 dB range and bone thresholds at 30 dB?

Answer:
Assuming that the conductive hearing loss is not amenable to traditional otologic surgery, the patient meets the criteria for the BAHA implant. Implantable hearing aids are excluded by the conductive hearing loss. The thresholds are in the range of acceptability for a regular BAHA.

Glossary

Electromagnetic coils
Devices that take advantage of the fact that current is generated in a coil if it is moved in a magnetic field. Conversely, a magnetic field is generated by applying current to the coil. Application of amplified current to a coil allows enhancement of the physical movement of ossicles if they are attached rigidly to a magnet. There are two general designs for electromagnetic coils: (1) extra-coil, where a magnet is outside the wire coil so that there is some distance between the coil and the magnet, and (2) intra-coil, where a magnet is inside the wire coil. The magnet can be attached to a structure such an ossicle outside the coil, causing it to move.

Floating mass transducer
An electromagnetic design where the magnet and coil are in a single device fixed to an ossicle in an intra-coil design.

Functional gain
The difference between unaided and aided thresholds. Functional gain is an important concept for hearing aids. Conventional hearing aids can provide large gains but some users do not use all available gain because of distortion and feedback at greater volumes.

Implantable hearing aid
A device that uses acoustic energy (physical movement) to drive the ossicles to improve hearing in which there is no external component, only internal component(s). Strictly speaking there are very few truly implantable hearing aids; most often they are semi-implantable aids.

Occlusion effect
An effect produced by occlusion of the external auditory canal inducing a conductive

Hearing loss
This effect is a common problem in fitting hearing aids that must occlude the external ear canal. The occlusion effect causes a feeling of ear fullness or hollowness but slightly improved bone conduction thresholds. The occlusion effect is frequency-dependent, and is greater for low frequencies than high frequencies. Speech discrimination may be adversely impacted. Venting or open ear molds typically prevent the occlusion effect and are useful in attempting to reduce the low frequency gain in patients with high-frequency hearing loss. Venting may induce feedback in the hearing aid, typically around 3 kHz.

Ossicular loading
Refers to the situation where mass applied to an ossicle may cause conductive hearing loss when the device is not being used. Ossicular loading is important for devices that require a piece of hardware to be attached to an ossicle. Residual hearing is a related term that refers to the amount of conductive hearing loss induced by ossicular loading. If the user decides not to use the device, he or she may be forced to use it anyway if the device induces a conductive hearing loss. When batteries fail, the device fails, or the user removes the external hardware, implantable hearing aids are not activated and this may be a detriment to hearing.
Surgical removal of the implanted device may be required, although it is possible that conductive hearing loss may persist. Some device manufacturers have considered ossicular loading more seriously than others.

Piezoelectrics
Materials that generate electrical current in proportion to physical application of force. Conversely they undergo physical change as a result of application of electrical energy.

Typically two such materials are bonded together to create a piezoelectric bimorph that generates electric output in proportion to mechanical deflection. Piezoelectrics can act either as sound sensors, providing an electric output depending on sound stimulation, or transducers, which produce physical deformation in proportion to electric current. In ear-related applications the two most likely types of piezoelectrics are the diaphragm type and, more commonly, the springboard type. Some other applications of piezoelectrics include microphones, record needles, and singing greeting cards.

References

1. Jaffee IS. Implantable middle ear hearing devices: Food and Drug Administration review process. Otolaryngol Clin North Am. Apr 2001;34(2):515-517.

2. Doyle T. New Device approval. Department of Health and Human Services; Food and Drug Administration [letter].
Available at: http://www.fda.gov/cdrh/pdf/P000025a.pdf. Accessed Aug. 20, 2001.

3. Cray JW, Allen RL, Stuart A, Hudson S, Layman E, Givens GD. An investigation of telephone use among cochlear implant recipients. Am J Audiol. Dec 2004;13(2):200-212.

4. Hintermair M, Albertini JA. Ethics, deafness, and new medical technologies. J Deaf Stud Deaf Educ. Spring 2005;10(2):184-192.

5. Callanan V, Poje C. Cochlear implantation and meningitis. Int J Pediatr Otorhinolaryngol. May 2004;68(5):545-550.

6. Goycoolea MV. Latin American experience with the cochlear implant. Acta Otolaryngol. May 2005;125(5):468-473.

7. Spindel JH. Middle ear implantable hearing devices. Am J Audiol. Dec 2002;11(2):104-113.

8. Browning GG, Gatehouse S. Estimation of the benefit of bone-anchored hearing aids. Ann Otol Rhinol Laryngol. Nov 1994;103(11):872-878.

9. Niparko JK, Cox KM, Lustig LR. Comparison of the bone anchored hearing aid implantable hearing device with contralateral routing of offside signal amplification in the rehabilitation of unilateral deafness. Otol Neurotol. Jan 2003;24(1):73-78.

10. Wazen JJ, Ghossaini SN, Spitzer JB, Kuller M. Localization by unilateral BAHA users. Otolaryngol Head Neck Surg. Jun 2005;132(6):928-932.

11. Crompton M. Vibrant Soundbridge -- P990052. Food and Drug Administratioin, Center for Devices and Radiological Health [Device approval letter]. Available at: http://www.fda.gov/cdrh/pdf/P990052b.pdf. Accessed Oct 27, 2000.

12. Vincent C, Fraysse B, Lavieille JP, Truy E, Sterkers O, Vaneecloo FM. A longitudinal study on postoperative hearing thresholds with the Vibrant Soundbridge device. Eur Arch Otorhinolaryngol. Oct 2004;261(9):493-496.

13. Makanani D. SOUNDTEC® Direct System - P010023. Food and Drug Adminstration, Center for Devices and Radiological Health. Available at: http://www.fda.gov/cdrh/pdf/p010023.html, Accessed 2001.

14. Hough JV, Dyer RK, Jr., Matthews P, Wood MW. Early clinical results: SOUNDTEC implantable hearing device phase II study. Laryngoscope. Jan 2001;111(1):1-8.

15. Chen DA, Backous DD, Arriaga MA, et al. Phase 1 clinical trial results of the Envoy System: a totally implantable middle ear device for sensorineural hearing loss. Otolaryngol Head Neck Surg. Dec 2004;131(6):904-916.

16. Garin P, Genard F, Galle C, Jamart J. The RetroX auditory implant for high-frequency hearing loss. Otol Neurotol. Jul 2004;25(4):511-519.

17. Garin P, Genard F, Galle C, Fameree MH, Jamart J, Gersdorff M. Rehabilitation of high-frequency hearing loss with the Retrox auditory implant. B-Ent. 2005;1(1):17-23.

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Chapter 2

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Head and Neck Cancer

Quality of Life following Chemoradiation Therapy for Head and Neck Cancer

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It is estimated that in 2005 there will be more than 40,000 newly diagnosed cancers of the head and neck, and that this year more than 10,000 patients will die of head and neck cancer (HNC).[1] Despite multiple modalities of treatment such as surgery, radiation, and chemotherapy, head and neck cancers continue to have one of the lowest five-year survival rates.[2]

The treatment algorithms for advanced HNC have changed significantly over the past 10 years. Current treatment protocols for advanced laryngeal cancers usually involve either chemotherapy with radiation (organ preservation) or total laryngectomy (TL) with postoperative radiation. Both treatment modalities have equivalent control rates and are widely used today for advanced laryngeal cancers.[3] Recent data also support the use of chemoradiation (CRT) for HNC in other sites, such as the oropharynx and hypopharynx.[4] As such, an increasing number of HNC patients with primary tumors in the oropharynx and hypopharynx, as well as larynx, are being treated with a CRT protocol rather than the standard surgical resection with postoperative radiation.

Although CRT protocols usually allow preservation of the functional organ, there are still significant acute morbidities, as well as long-term, following CRT. Because of the increasing number of patients with HNC undergoing CRT, there have been numerous studies examining the quality of life (QOL) for such patients during and after their treatment. QOL used as a clinical outcome measurement is often not measured or carefully defined, but rather broadly defined to encompass an individual's perception of his or her emotional, physical, social, and sexual state. Simply, it is the satisfaction and well-being that a patient experiences on a daily basis.[5] QOL issues will differ depending on tumor site, stage, and type of treatment rendered.

QOL Instruments

Quality of life is usually assessed through questionnaires, called instruments. Issues pertaining to quality of life are called domains. The four basic domains are psychological, social, occupational, and physical.[6] Each domain or issue is given a score and, in general, a higher score signifies a better quality of life. Multiple instruments have been utilized to study QOL in head and neck cancer patients, ranging from global (applicable to any disease and containing general physical, social, and psychological questions), to specific head and neck questionnaires, to performance questionnaires that address specific functions such as speech or swallowing.

Commonly used instruments in head and neck cancer patients are the European Organization for Research and Treatment of Cancer Quality of Life Questionnaire Core 30

(EORTC-C30), the Head and Neck Core 35 (EORTC-HN35), the Head and Neck Radiotherapy Questionnaire (HNRQ), the University of Michigan Head and Neck Quality of Life Questionnaire, and the University of Washington Head and Neck Disease Specific Measure (UW-QOL).[7]

The University of Washington Quality of Life Instrument, version 4 (UW-QOL, v 4) is a short questionnaire, assessing functional items such as swallowing, pain, and speech. However, psychological issues are not addressed in this instrument. The first section consists of 12 domains that pertain to the degree of quality of life in the categories of pain, appearance, activity, recreation, swallowing, chewing, speech, shoulder function, taste, saliva, mood, and anxiety. The second part of the instrument asks the patients which of the previous issues have been the most important to them in the past seven days. In the final part of the instrument, patients are given three general questions comparing their (1) current health-related quality of life to one month before developing cancer, (2) health-related quality of life during the past seven days, and (3) overall quality of life during the past seven days. Higher scores in each category signify better quality of life. This instrument is simple to use and well-suited for rapid assessment of QOL in HNC patients. A comparison of the UW-QOL with the EORTC QOL C33, the Medical Outcomes Short Form 36, and the EORTC HN35 found that the UW-QOL was a broad measure suitable for low-cost assessment of disease-specific functional status.[8] Because psychological issues are not included in the UW-QOL questionnaires, this limits its usefulness in assessing those issues.

Global questionnaires include the Medical Outcomes Short Form 36, the Karnofsky Performance Index, and the Hospital Anxiety and Depression Scale.[9] The M.D. Anderson Dysphagia Inventory and the Performance Status Scale for Head and Neck Cancer are performance questionnaires addressing specific functions such as swallowing, speech intelligibility, and the ability to eat.[10]

QOL in HNC Patients Prior to Treatment

Patients with head and neck cancer often have multiple medical co-morbidities that will affect their quality of life even prior to undergoing treatment. There is usually a history of heavy smoking and alcohol use. Because of the deleterious effects of smoking, most patients with HNC will also have chronic lung disease. Coronary artery and liver disease are also common in this patient population. A cancer in the upper aerodigestive tract may have an adverse effect on chewing, swallowing, and eating, leading to weight loss and malnutrition. Other chronic illnesses such as hypertension, diabetes, and vascular disease may be present as well. Adverse socioeconomic factors and poor family support also diminish the QOL in HNC patients.

The diagnosis of HNC is often delayed, particularly when the primary site is located in the orophayrnx or hypopharynx. Because of the difficulty in examining such areas and the paucity of symptoms produced, the tumor may not be seen or diagnosed until it reaches significant size, or when a neck metastasis develops. Unfortunately, this means that many patients will present with an advanced stage of HNC at diagnosis, worsening their prognosis and QOL.

Acute Changes during and Immediately Following CRT

Most HNC patients undergoing CRT will receive a platinum-based regimen, such as cisplatin, with or without 5-FU. Patients receiving concurrent chemotherapy with radiation will experience significant mucositis, which may require a break in treatment. Additional acute side effects from CRT include anemia, neutropenia, infectious complications, renal toxicity, ototoxicity, skin changes, fatigue, and weight loss.[11] The mucositis and edema of the aerodigestive tract will lead to varying degrees of dysphagia. Some patients will require placement of a gastrostomy tube (G-tube) for feeding and/or a tracheotomy tube for securing the airway. Because of the many side effects, patients will experience a significant decrease in their overall QOL during treatment. The stress of undergoing chemotherapy and daily radiation treatments also contributes to a generalized depression in many of these patients.

Chronic Changes after CRT

Most of the acute side effects gradually subside after completion of treatment. Studies have shown that, 6 to 12 months after treatment, QOL scores rise to above pretreatment levels.[12] However, most patients report some long-term xerostomia and dysphagia. Because of dry mouth and sticky saliva, only 50 percent of patients in one study were able to resume a normal diet after treatment.[13] Some patients require long-term G-tubes for adequate nutrition. A significant number of patients also reported residual pain even after treatment.[14]

Other long-term side effects after CRT include hoarseness, decreased taste, and difficulty chewing. Further longitudinal studies are needed to determine whether late sequelae following CRT persist or worsen over many years, caused by progressive fibrosis of tissues.

Review of QOL Studies

Many studies have looked at quality-of-life issues for head and neck cancer patients following treatment. Schwartz et al. reviewed the published literature, evaluating the terminology, the design, and the interpretation of quality-of-life measurements in head and neck cancer patients. They specifically examined studies that compared head and neck cancer treatments at a point in time or reported changes over a period of time. They concluded that there were few hypothesis-driven studies, and clinical interpretations of quality-of-life outcomes were often not provided at the conclusion of the studies.[15] Terrell et al. reported on significant clinical predictors in head and neck cancer patients pertaining to quality-of-life issues. The most common predictors of QOL were tracheotomy and gastrostomy tubes, co-morbid conditions, chemotherapy, and neck dissections.[16] Another study evaluated patients with advanced head and neck cancer who underwent CRT on performance and QOL issues. Twelve months after treatment, patient's QOL issues had resolved except for the ability to eat a wide range of food. This data supported the use of intense chemoradiation therapy with minimal impact on QOL issues.[17]

Harrison et al. reported good QOL and oncological outcomes with patients who were treated for cancer of the base of the tongue.[18] Hammerlid and Bjordal et al. examined QOL issues in patients with head and neck cancer. Their studies reported that there were differences in QOL issues depending on the anatomic subsite of the head and neck cancer. They found that tumor stage and site had the most profound impact on individual patients' QOL. Their studies also reported that after one year from end of treatment date, patients returned to their pretreatment QOL state except in the senses, xerostomia, and sexuality.[19] Another study addressed the issue of swallowing after head and neck cancer treatment. The researchers concluded that patients undergoing CRT for oropharyngeal cancer had better functional outcomes related to swallowing than those undergoing surgery with postoperative radiation.[20]

It is often difficult to accurately compare QOL studies of HNC patients in the literature. Many studies examined patients with head and neck cancer in several different subsites, including oral cavity, oropharynx, larynx, and hypopharnyx. Care must be used when comparing QOL outcomes for patients with cancers in the different subsites in the head and neck after treatment. Morton et al. reported that anatomic subsites within head and neck cancer responded differently, and patients ultimately experienced different QOL issues depending on the subsite and treatment received. Organ preservation therapy did not necessarily lead to better QOL outcomes.[21] In addition, many different types of QOL instruments were used in these studies to identify varying domains pertaining to QOL, including mental, sexual, and physical functioning.

Improvement of QOL in Patients Treated with CRT versus Surgery

McDonough et al. compared patients with HNC who underwent CRT versus induction chemotherapy followed by surgery and postoperative radiation. There were significantly higher QOL scores in the nonsurgical group, with lower levels of social distress and avoidance because of better communication abilities and less physical disfigurement.[22] In another study, QOL issues were compared in laryngeal cancer patients who underwent CRT versus total laryngectomy with postoperative radiation (TL+XRT).[23] CRT patients had more problems with chewing, swallowing, and pain, while TL+XRT patients had worse speech and shoulder function. Hanna et al. recently reported that laryngeal cancer patients who underwent CRT had similar QOL scores compared with those who underwent TL+XRT, but functional subscale analysis revealed some differences. CRT patients had greater difficulties with dry mouth, while TL+XRT patients reported more problems with social functioning, sensory disturbances, use of painkillers, and coughing.[24]
Finizia et al. reported similar QOL issues pertaining to psychosocial adjustment and functional ability in patients who underwent CRT and TL+XRT. In the TL+XRT group, a lower level of QOL was reported in those patients who used an electrolarynx.[25] Terrell et al. assessed the QOL outcomes in patients in the Veterans Affairs Laryngeal Cancer Study who underwent CRT and TL+XRT. They reported that the CRT group experienced better QOL outcomes in freedom from pain, lower levels of depression, and less problems with work due to better overall emotional

well-being. Speech scores were similar in both the CRT and the TL+XRT groups.[26] Paleri et al. also assessed the QOL outcomes in patients who underwent CRT and TL+XRT. He reported similar scores between the two groups but a higher trend (better QOL) in the CRT group.[27] Major et al. reported similar QOL outcomes in CRT and TL-XRT patients in the domains of physical functioning, bodily pain, health perception, social functioning, energy/fatigue, and mental health. He reported that the CRT group had a higher QOL in their activities of daily life compared with those undergoing surgery with postoperative radiation.[28]

For laryngeal cancer patients, organ preservation protocols using CRT have the obvious advantage of retaining laryngeal speech. Many total laryngectomy patients undergo speech rehabilitation with the use of an electrolarynx, esophageal speech, or tracheal esophageal puncture. This may explain why in one study some TL patients reported that their speech was the same as always and in another study both TL and CRT patients who retained their larynx showed similar speech scores.[29] In contrast, most CRT patients did not generally receive any speech or swallowing therapy, and this group of patients may potentially benefit from use of intensive therapy in these arenas.
Recently the use of concurrent chemoradiation for organ preservation has been extended to nonlaryngeal sites, including the oropharynx and hypopharnx. Data from these studies confirm the efficacy of CRT for locoregional control of advanced oropharyngeal and hypopharyngeal cancers with acceptable morbidity.[30] Schrader et al. examined a series of patients with hypopharyngeal cancer who

were treated with hyperfractionated radiation therapy and concurrent chemotherapy, and found that late sequelae of treatment were tolerable and did not adversely impact patients' QOL.[31] In the University of Pennsylvania Phase II trial of oropharyngeal cancer patients treated with chemoradiation, organ preservation was attained in 77 percent of the patients and 90 percent did not require permanent tracheotomy or gastrostomy tubes. The eating-in-public and speech understandability scores were not greatly different from patients' pretreatment scores; however, scores for normalcy of diet declined.[32]

There is evidence of benefit from use of amifostine during CRT. This thiol compound protects normal tissues from radiation by binding of the sulfhydryl group with hydroxyl radicals. There is a high concentration of amifostine in the salivary glands after administration, which can decrease the severity of xerostomia after head and neck radiation.[33] Randomized studies demonstrated significant reduction of high-grade xerostomia in patients undergoing CRT for advanced HNC who were receiving amifostine.[34]
Recent advances in radiation therapy techniques such as intensity-modulated radiation therapy (IMRT) also show promise in decreasing the bothersome xerostomia[35]

Conclusion

The efficacy of chemoradiation protocols for locoregional control of advanced head and neck cancer has been demonstrated. HNC patients often have compromised quality of life at the time of diagnosis, even before beginning treatment. Depending on the primary site of the tumor, abnor-

malities in swallowing, chewing, speech, and appearance may occur. Coexisting morbidities such as cardiopulmonary disease, hypertension, diabetes, malnutrition, alcoholism, and poor social support contribute to low QOL.

Treatment with CRT carries significant acute and chronic morbidity. Acute toxicities include mucositis, anemia, neutropenia, renal toxicity, dysphagia, weight loss, fatigue, and depression. Much of the acute toxicity resolves after completion of treatment, and most patients return to pretreatment QOL levels by 6 to 12 months, except for the senses, xerostomia, and sexuality. Other long-term sequelae include continued difficulty with swallowing and chewing, decreased ability to eat a wide range of foods, sticky saliva, and hoarseness. Some patients are unable to eat a normal diet and may even be permanently G-tube dependent.
However, overall, QOL in HNC patients treated with CRT is relatively high, and compares favorably with patients who have undergone surgical resection of their tumor followed by postoperative radiation therapy. HNC patients treated with CRT may benefit from the use of amifostine during treatment. Intensity-modulated radiation therapy techniques may also reduce the incidence of long-term xerostomia. Intensive speech and swallowing therapy may aid in improving these functions following CRT.

Patients and physicians need to understand the differences in sequelae among treatment protocols so that better-informed decisions can be made. Supportive therapy for swallowing and speech function, as well as psychological support, are essential in treatment planning. Future prospective, longitudinal studies are needed to examine QOL issues for patients with advanced HNC.

Quiz

1. The most common acute toxicities associated with chemoradiation for head and neck cancer include all of the following EXCEPT

   a. mucositis
   b. anemia
   c. dysphagia
   d. neck fibrosis
   e. xerostomia

2. Most of the acute toxicities associated with chemoradiation for head and neck cancer resolve

   a. one month following end of treatment.
   b. three months following end of treatment.
   c. one year following end of treatment.
   d. two years following end of treatment.
   e. five years following end of treatment.

3. Chronic sequelae following chemoradiation for head and neck cancer usually include

   a. neutropenia
   b. permanent gastrostomy
   c. permanent tracheotomy
   d. sticky saliva
   e. none of the above

4. The following statement is supported in the literature:

   a. Laryngeal cancer patients who undergo chemoradiation with organ preservation have overall better quality of life than patients who undergo total laryngectomy.
   b. Total laryngectomy patients have worse quality-of-life scores in speech than laryngeal cancer patients who undergo chemoradiation with organ preservation.
   c. Most patients who undergo chemoradiation will require a tracheotomy and gastrostomy tube.
   d. Most patients who undergo chemoradiation are able to return to a normal diet within one year.
   e. Patients who undergo chemoradiation for head and neck cancer have less pain than those who undergo surgery.

5. The following may help to mitigate the adverse effects of chemoradiation treatment in head and neck cancer patients:

   a. amifostine
   b. intensity-modulated radiation therapy
   c. speech therapy
   d. swallowing therapy
   e. all of the above

Correct Answers:

1. d
2. c
3. d
4. a
5. e

References

1. Jemal A, Murray T, Ward E, et al. Cancer statistics, 2005. CA Cancer J Clin. January 2005;55:10-30.

2. See the following four studies: Garden AS, Harris J, Vokes EE, et al. Preliminary results of Radiation Therapy Oncology Group 97-03: A randomized phase II trial of concurrent radiation and chemotherapy for advanced squamous cell carcinomas of the head and neck. J Clin Oncol. 2004;22(14):2856-2864; Forastiere A, Koch W, Trotti A, et al. Head and neck cancer. N Eng J Med. 2001;345(26):1890-1900; Brockstein B, Haraf DJ, Rademaker AW, et al. Patterns of failure, prognostic factors and survival in locoregionally advanced head and neck cancer treated with concomitant chemoradiotherapy: A 9-year, 337-patient, multi-institutional experience. Ann Oncol. 2004;15(8):1179-1186; and Argiris A, Brockstein BE, Haraf DJ, et al. Competing causes of death and second primary tumors in patients with locoregionally advanced head and neck cancer treated with chemoradiotherapy. Clin Cancer Res. 2004;10(6):1956-1962.

3. The Department of Veterans Affairs Laryngeal Cancer Study Group. Induction chemotherapy plus radiation compared with surgery plus radiation in patients with advanced laryngeal cancer. N Eng J Med. 1991;324:1685-1690. Also see Gilbert J, Forastiere A. Organ preservation trials for laryngeal cancer. Otolaryngol Clin N Am. 2002;35:1035-1054.

4. See Calais G, Alfonsi M, Bardet E, et al. Randomized trial of radiation therapy versus concomitant chemotherapy and radiation therapy for advanced-staged oropharyngeal carcinoma. J Natl Cancer Inst. 1999;91:2081-2086; and Machtay M, Rosenthal DI, Hershock D, et al. Organ preservation therapy using induction plus concurrent chemoradiation for advanced respectable oropharyngeal carcinoma: A University of Pennsylvania phase II trial. J Clin Oncol. 2002;20:3964-3971.

5. Morton RP, Izzard ME. Quality-of-life outcomes in head and neck cancer patients. World J Surg. 2003;27:884-889.

6. Borghgraef K, Delaere P, Van den Bogaert W, et al. Quality of life in head and neck cancer patients. Acta Otorhinolaryngol Belg. 1997;51:69-72.

7. See the following four references: Bjordal K, Ahlner-Elmqvist M, Tollesson E, et al. Development of a European Organization for Research and Treatment of Cancer (EORTC) questionnaire module to be used as quality of life assessment in head and neck cancer patients. Acta Oncol. 1994;33:879-885; Browman GP, Levine MN, Hodson DL, et al. The head and neck radiotherapy questionnaire: A morbidity/quality of life instrument of clinical trials of radiation therapy in locally advanced head and neck cancer. J Clin Oncol. 1993;11:863-872; Terrell JE, Nanavati KA, Esclamado RM, et al. Head and neck cancer-specific quality of life: Instrument validation. Arch Otolaryngol Head Neck Surg. 1997;123:1125-1132; and Hassan SJ, Weymuller

EA. Assessment of quality of life in head and neck cancer patients. Head Neck. 1993;15:485-496.

8. Rogers SN, Lowe D, Brown JS, et al. A comparison between the University of Washington Head and Neck Disease-Specific measure and the Medical Short Form 36, EORTC QOQ-C33 and the EORTC Head and Neck 35. Oral Oncol. 1998;34:361-372.

9. See Ware JE Jr, Cherbourne CD. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. Med Care. 1992;30(6):473-483. Also see Karnofsky DA, Burchemajh JH. The Clinical Evaluation of Chemotherapeutic Agents. New York: Columbia University Press; 1949:191-205; and Zigmond AS, Snaith RP. The hospital anxiety and depression scale. Acta Psychiatr Scand. 1983;67:361-370.

10. List MA, D'Antonio LL, Cella DF, et al. The Performance Status Scale for Head and Neck Cancer Patients and the Functional Assessment of Cancer Therapy-Head and Neck Scale. A study of utility and validity. Cancer. 1996;77(11):2294-2301.

11. Nguyen NP, Sallah S, Karlsson U, et al. Combined chemotherapy and radiation therapy for head and neck malignancies: Quality of life issues. Cancer. 2002;94:1131-1141.

12. Murry T, Madasu R, Martin A, et al. Acute and chronic changes in swallowing and quality of life following intraarterial chemoradiation for organ preservation in

patients with advanced head and neck cancer. Head Neck. 1998;210:31-37. Also see List MA, Siston A, Haraf D, et al. Quality of life and performance in advanced head and neck cancer patients on concomitant chemoradiotherapy: A prospective examination. J Clin Oncol. 1999;17:1020-1028.

13. List MA, Mumby P, Haraf D, et al. Performance and quality of life outcomes in patients completing chemoradiotherapy protocols for head and neck cancer. Qual Life Res. 1997;6:274-284.

14. See List et al., 1997, note 13; and LoTempio MM, Wang KH, Sadeghi A, et al. Comparison of quality of life outcomes in laryngeal cancer patients following chemoradiation vs. total laryngectomy.
Otolaryngol Head Neck Surg. In press.

15. Schwartz S, Patrick DL, Yueh B. Quality-of-life outcomes in the evaluation of head and neck cancer treatments. Arch Otolaryngol Head Neck Surg. 2001;127:673-678.

16. Terrell JE, Ronis DL, Fowler KE, et al. Clinical predictors of quality of life in patients with head and neck cancer. Arch Otolaryngol Head Neck Surg. 2004;130:401-407.

17. See List et al., 1999, note 12.

18. Harrison LB, Ferlito A, Shaha AR, et al. Current philosophy on the management of cancer of the base of tongue. Oral Oncol. 2003;39(2):101-105.

19. See Hammerlid E, Bjordal K, Ahlner-Elmqvist M, et al. A prospective study of quality of life in head and neck cancer patients. Part I: At diagnosis. Laryngoscope. 2001;111(4 Pt 1):669-680; and Bjordal K, Ahlner-Elmqvist M, Hammerlid E, et al. A prospective study of quality of life in head and neck cancer patients. Part II: Longitudinal data. Laryngoscope. 2001;111(8 Pt II):1440-1452.

20. Gillespie MB, Brodsky MB, Day TA, et al. Swallowing-related quality of life after head and neck cancer treatment. Laryngoscope. 2004;114(8):1362-1367.

21. See Morton and Izzard, 2003, note 5.

22. McDonough EM, Varvares MA, Dunphy FR, et al. Changes in quality-of-life scores in a population of patients treated for squamous cell carcinoma of the head and neck. Head Neck. 1996;18(6):487-493.

23. See LoTempio et al., in press, note 14.

24. Hanna E, Sherman A, Cash D, et al. Quality of life for patients following total laryngectomy vs chemoradiation for laryngeal preservation. Arch Otolaryngol Head Neck Surg. 2004;130(7):875-879.

25. Finizia C, Bergman B. Health-related quality of life in patients with laryngeal cancer: A post-treatment comparison of different modes of communication. Laryngoscope. 2001;111(5):918-923.

26. Terrell JE, Fisher SG, Wolf GT. Long-term quality of life after treatment of laryngeal cancer. Arch Otolaryngol Head Neck Surg. 1998;124:964-971.

27. Paleri V, Stafford FW, Leontsinis TG, et al. Quality of life in laryngectomees: A post-treatment comparison of laryngectomy alone versus combined therapy. J Laryngol Otol. 2001;115(6):450-454.

28. Major MS, Bumpous JM, Flynn MB, et al. Quality of life after treatment for advanced laryngeal and hypopharyngeal cancer. Laryngoscope. 2001;111(8):1379-1382.

29. See Lotempio et al., in press, note 14; and Terrell, Fisher, Wolf, 1998, note 26.

30. See Calais et al., 1999, and Machtay et al., 2002, note 4.

31. Schrader M, Schipper J, Jahnke K, et al. Hyperfractionated accelerated simultaneous radiochemotherapy in advanced hypopharyngeal carcinomas. Survival rate, retained function, quality of life in a phase II study. HNO. 1998;46:140-145.

32. See Machtay et al., 2002, note 4.

33. Capizzi RL. The preclinical basis for broad-spectrum selective cytoprotection of normal tissues from cytotoxic therapies by amifostine. Semin Oncol. 1999; 26 (2 Suppl 7):3-21.

34. See Brizel DM, Wasserman TH, Henke M, et al. Phase III randomized trial of amifostine as a radioprotector for head and neck cancer. J Clin Oncol. 2000;18:3339-3345; and Suntharalingam M, Jaboin J, Taylor R, et al. The evaluation of amifostine for mucosal protection in patients with advanced loco-regional squamous cell carcinomas of the head and neck (SCCHN) treated with concurrent weekly carboplatin, paclitaxel, and daily radiotherapy (RT). Semin Oncol. 2004;31(6 Suppl 18):2-7.

35. See Rosenthal DI, Chambers MS, Weber RS, et al. A phase II study to assess the efficacy of amifostine for submandibular/sublingual salivary sparing during the treatment of head and neck cancer with intensity modulated radiation therapy for parotid salivary sparing. Semin Oncol. 2004;31(6 Suppl 18):25-28; Saarilahti K, Kouri M, Collan J, et al. Intensity modulated radiotherapy for head and neck cancer: Evidence for preserved salivary gland function. Radiother Oncol. 2005;74(3):251-258. Epub 2004 Dec 8; and Hong TS, Tome WA, Harari PM. Intensity-modulated radiation therapy in the management of head and neck cancer. Curr Opin Oncol. 2005;17(3):231-235.

Voice

Dysphonia and the Aging Voice

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The human voice is unique in the entire animal kingdom. The flexibility of the human voice allows us to portray our thoughts, emotions, joys, and fears. This extraordinary flexibility can be seen throughout life, beginning with the power of a baby's cry to the wonderful fullness and range of the world-class soprano or tenor. Each voice is unique and provides one of the signatures of the individual. The ancient Greeks felt that the voice was so important to a person's character that they thought that the voice actually originated in the heart.

These remarkable qualities of the voice are unfortunately not immune to the effects of human aging. It is rare that elite singers will continue to perform their most difficult roles even into their late 50s or 60s. As people enter their 80s and 90s, voices lose not only their range but also some of their strength and power, and male and female voices become less distinguishable.

This chapter will describe the multiple neurological, hormonal, and general effects of aging on the voice and also the opportunities that people have to allow their voices to remain strong and vibrant well into their senior years.

Brief Anatomy and Physiology of the Voice

The production of the voice is a complex interaction among a number of different body systems.

This discussion can be simplified by thinking of the voice as a musical instrument. For any instrument to produce sound, something must activate the sound (such as plucking a string on a guitar or blowing into a trumpet), something must vibrate (like the guitar string or the reed), and something must resonate (the body of the instrument). The lungs serve the role of the activator in human voice production. As one breathes in, negative pressure is produced that actually pulls air into the lungs. As we expire or exhale, that air serves as the source of power for setting up the vibration of the vocal folds. [1]

The vocal folds themselves serve as the vibrators, and it is the fine control of the movements of the vocal folds that allows for the flexibility of the speaking and singing voice. In the body, the face and sinuses and the chest serve as resonators, which give the voice its timbre or character.

In addition to these three important areas, other body parts and systems play important roles. Tension in the jaw or neck will reduce flexibility and increase the onset of fatigue. The voice is strongest when the body is in the upright position (not many opera roles allow the figure to recline) and musculo-skelatal problems can affect the best posture. In addition, the diaphragm and abdomen are important in the support of the voice, so abdominal conditions such as cramping or bowel disorder may have a negative impact on voice. The psychological system is

important in the confidence one has in his or her voice, as can be seen by the fluttering of the voice when a person is nervous or anxious. Finally, refined coordination of muscle movement and sensory control are adjusted by the neurological system.

Aging and Its Effects on the Larynx and Vocal Folds

The larynx goes through a maturation process that begins early in life and continues with changes that occur throughout life, with the most dramatic changes occurring early in life through puberty. Over time, however, the cartilages of the larynx begin to calcify and become slightly more rigid. The joints that allow for three-dimensional movement of the vocal folds become stiffer and the bulk of muscle of the vocal folds diminishes. [2] These findings are not dissimilar to those that can be seen in other muscles and joints in the body. Often, the vocal folds become slightly bowed, which prevents tight vocal fold closure, and result in a somewhat more breathy voice with a reduction in the very upper portion of the range. This in turn requires tighter closure and tension

The vibration of the vocal folds requires that there be moisture on the vocal fold surface. As people age, there is a decrease in mucous and saliva secretions not only in the mouth but in the larynx as well.[3] Smooth vibration is reduced. It is important to maintain good hydration and this can be supplemented with products that thin the mucous such as Humabid® or Mucinex®. In general, the major changes that occur with the laryngeal structures

begin in the 60s in men and may begin just after menopause in women. Fortunately, these changes tend to occur over a long period of time, so most people are able to maintain a good voice well after the beginning of changes caused by aging, although this is often more difficult for the singing than the speaking voice. The hormonal effects on the larynx will be discussed in more detail later.

Neurological Changes of the Voice with Age

The production of voice depends on a very sophisticated and integrated coordination of nerves of sensation to the larynx and nerves that control muscle movements. The nerves must also coordinate the activity of the lungs and resonating cavities. With aging, the speed of nerve transmission decreases and there is a reduction in coordination of muscular movement. Although in the absence of a true neurological disorder this does not play a major role, fine control of pitch and range may be affected. Specific neurological disorders increase in frequency with aging, but are still uncommon in comparison with the normal neurological changes that occur with the aging process. The most common specific voice disorder whose incidence increases with aging is voice tremor, which can occur independently (Primary Vocal Tremor) or with other diseases such as Parkinson's disease.[4] In addition, motion disturbances of the larynx such as neuromuscular paresis or paralysis, or joint movement problems, become more prevalent with ageing.[5] Early identification with a visit to an otolaryngologist (ear, nose and throat surgeon) may allow for either medications or voice training techniques

that can dramatically reduce the effects of the tremor on communication.

Gastrointestinal Disorders and Voice

The most common and most important gastrointestinal disorder that can negatively affect the voice is reflux disease. Most people associate the term reflux with gastroesophageal reflux, which is a term used for acid leaving the stomach and regurgitates up the esophagus. When the reflux reaches the throat, however, it is more appropriately called laryngopharyngeal reflux (LPRD).[6] LPRD can result in a number of nonspecific symptoms such as hoarseness, chronic cough, or chronic irritation with a mucous-sticking sensation. People will frequently clear their throats or may suspect that this problem is related to sinus disease because of the mucous sensation. Although multiple or prolonged episodes of reflux are usually necessary to result in esophageal disease, intermittent and short-duration LPR can result in symptoms. Many patients with LPRD have no symptoms of heartburn.
Although reflux can occur at any age, it tends to increase as people age, particularly in the face of a hiatal hernia. In many senior patients with dysphonia, reflux may be playing an important role and therefore should at least be considered. LPRD may also be more difficult to treat than gastroesophageal reflux and may require a combination of diet and lifestyle modifications, histamine (H2) blockers, and proton pump inhibitors (such as Prilosec).

Other gastrointestinal or abdominal conditions may also have either a direct or indirect effect on the quality of the voice. Abdominal surgery, cramping, constipation, or diarrhea may all influence the ability of the abdomen and diaphragm to support the voice. In addition discomfort from some of these conditions may limit abdominal strength and support.

Respiratory Disorders

As stated earlier, the initiation of sound and voice begins with inhalation and exhalation. Thoracic and pulmonary disorders may serve to limit vital capacity, which in turn will limit breath support and control necessary for efficient speaking and singing. Of particular note are restrictive pulmonary diseases such as Chronic Obstructive Pulmonary Disease (COPD), chronic bronchitis, and asthma. Appropriate prevention of controllable diseases such as COPD through early smoking cessation and early intervention for patients that develop these diseases will play a role in maintaining vocal strength and efficiency.

Hormonal Effects on the Aging Voice

Thyroid Hormones

Besides these mechanical elements discussed above, the decrease or changes in hormones may play a major role in changes in voice with age.[7] As a person ages there may frequently be a drop in the secretion of thyroid hormones—hormones that generate energy for the muscle

s and that hydrate the organism. We often observe a decrease in thyroid hormone or hypothyroidism. The thyroid hormones should be controlled systematically in presbyphonia after menopause or andropause. Patients with a sluggish thyroid often require appropriate therapeutic treatment to get them out of their lethargy and feeling energetic again.

Progesterone Action on the Envelope of Neurons

Progesterone is secreted by the ovaries and this was originally considered as a hormone involved only in reproductive functions. But Gago has demonstrated that it can be synthesized within the nervous system by neurons and glial cells.[8] The progesterone has promyelinating and neuroprotective effects. Moreover, it can be synthesized locally in the nervous system by neurons and glial cells and can thus be considered to be a "neurosteroid." It plays an astonishing role here. It activates the synthesis of the protective sheath of the neuron, the myelin sheath. The myelin sheath, a sort of protective sleeve that shields the nerve from all traumatic aggression and from differences in temperature, enables nervous impulses to be transmitted at a constant speed between the brain and its target organ. Nerves that have this myelin sheath conduct nervous impulses better and faster. As early as 1995, Ian Duncan of the University of Wisconsin lifted the veil on the action of progesterone on the brain, but not on its synthesis. The impact of this discovery has led to a better understanding of therapeutic approaches to treating neurological diseases or certain myopathies that alter the myelin sheath and therefore nerve conduction, such

as Lou Gehrig's disease or multiple sclerosis. It seems that progesterone significantly slows the evolution of these afflictions.

At menopause, the somewhat precipitous drop in progesterone results in a progressive slowing down of nerve conduction that's barely noticeable. This slowdown is caused by a relative lack of myelinization of peripheral nerves and, as a result, the voice is less well controlled, particularly in singing.

Menopause Outcome

During menopause, this cycle is progressively disrupted. But this menopause effect, which today is of interest to us all, has only relatively recently become topical.[7] In Greek civilization, four hundred years BCE, the menopausal woman didn't exist. She was an exception. The average life expectancy was 23 to 27 years of age. Menopause was still rare in the Middle Ages. Life expectancy was then 23 to 40 years. It was only in the nineteenth and twentieth centuries that menopause was common enough that consideration of its health implications began to be considered. Indeed, girls born in the 1980s can expect to live to the age of 92! Menopause now corresponds to practically half a woman's life. By the end of the twentieth century, France accounted for nearly 8.5 million menopausal women. The importance of the voice, the development of verbal communication, and interpersonal relationships all point to the essential problem that the voice and menopause are now beginning to pose.

Why These Changes in a Woman's Voice at This Period of Her Life?

During the perimenopause, ovarian activity strongly diminishes. Progesterone and estrogen levels are dramatically reduced. Similarly, the secretion of male hormones also drops off considerably. But their presence, now that they are no longer counterbalanced by feminine hormones, can sometimes cause the voice to become more masculine. Thus, the ovary becomes a simple endocrine gland with no reproductive function.

The menopausal phase normally lasts from the age of 47 to the age of 55. The impact that the sex hormones had on their various target organs disappears, not without consequences. However, these days, the administration of substitute hormones enables the unpleasant consequences of this lack of sex hormones to be delayed to an increasingly later age, saving many women from a trying experience that is both mentally and physically hard to accept. Our better understanding of endocrinology has provided therapies that may help some menopausal woman to have a better quality of life.

In some cases, hormone substitutes may be a contraindication. They are not recommended in cases of breast cancer, in patients with a high-risk family background, in certain cardiovascular pathologies, or in cholesterol-related afflictions. For this reason, a medical check-up is a prerequisite for women in their 50s considering their options.

Because estrogens are reduced, the receptors of sex hormones receive more androgens and become more receptive to them. As a result, the vocal cord mucous membrane thickens and exhibits a lack of tonicity and a deficiency of contour. The voice becomes deeper and more masculine. Meanwhile, the 60-year-old woman may develop symptoms such as increased hairiness, as an indirect consequence of androgens. A smear test of the cervix of the uterus indicates an atrophy of the epithelium. The same result is obtained from a smear test of the vocal cords: the parallelism is amazing

Menopause and the Nervous System

The neurological motor and sensation functions of the larynx are largely controlled by the vagus nerve. Its responsiveness is improved by estroprogesterone. Therefore, at menopause the radical drop in the secretion of estrogens and the complete halt in the secretion of progesterone induce slower nervous conduction from the brain to the larynx. As a result, vocal response slows down slightly, which can hamper rapid changes in frequencies when singing. Later, the vibrato (seven vibrations per second) cannot be maintained. The voice gradually gears down to the tremolo (four vibrations per second).

Androgens Turn into Estrogens

Since 1977, we know that in both men and women fat cells can turn androgens into estrogens. The relationship between obesity and a higher secretion of estrones

(estrogen derivatives) is also age related. It is higher in menopausal women. This is the work of a specific gene in our DNA (cytochrome 19 associated with P450 aromatase) that facilitates the transformation of androgen into estrogen in our adipose cells. Thus, the lower need for hormone substitutes of overweight woman is caused by the fact that her fat cells will transform her androgens into estrones. Meanwhile, the slim woman is more likely to need hormone substitute therapy, although the positive value of lower weight for many chronic diseases such as hypertension and diabetes would seem to be more important to most women.

With age, muscle mass also diminishes, adipose mass increases, and cells are redistributed differently about the body. Corticosteroids encourage the increase of fat cells. Therefore, menopause women need to be cautious about consuming them. A carefully considered hormone substitute therapy program, associated with vitamins and minerals, can bring considerable benefits to most females who have elite voice requirements, if their body can tolerate it. Many women thus treated are able to avoid developing a masculine voice as they age and are able to preserve a beautiful voice for significantly longer. It is impressive to see the sopranos who have kept the same tessitura until the age of 65.

Men and Andropause

The androgens secreted by the testicles have a direct effect on the voice. They certainly act on the bony tissues,

but also on the brain. Androgens increase blood flow in the organism and improve oxygenation and muscle performance and can produce some sense of euphoria. At the age of 70, andropause may appear. Blood analysis will diagnose the lack of androgens. If there is no contra indication such as cancer of the prostate, androgens treatment may be useful to recover the vocal folds shape, the tonicity of the resonators, and consequently a powerful voice with a satisfactory register.

The Paradox of the Aging Voice

In Women

As the menopausal woman advances in age, her new hormonal balance, with its absence of estrogens and its very mild secretion of testosterone due to the atrophy of her ovaries, is no longer able to sustain the tonicity and strength of the vocal cord muscles. What are the consequences of this? The two vocal cords atrophy progressively. The mucous membrane covering them becomes thinner and dehydrates.

Initially, the voice displays a narrower register, the higher harmonics are lost, and the voice is less powerful and tires faster. But a paradoxical effect sets in. Because the vocal cord has diminished in thickness and become finer, the voice, which had become a little deeper, now becomes higher, more delicate, sometimes even shrill. You often hear 80-year-olds speaking with a very high-pitched voice. One can thicken the vocal cords again by injecting

a substance into them, which may provide some reasonable timbre and vocal endurance. [10]

In Men

After the age of 70, men can present the same vocal symptoms in the male climacteric. Yet the vocal structure in this case behaves like an athlete in all respects. As with women, hormone therapy is indicated in conjunction with specific nutrition hygiene, and voice therapy and training. Androgenic hormonal therapy is rarely advisable because of its danger for an altered prostate. For men, vocal training is the best guarantee of keeping a young voice. Regular practice and communication with others stimulates the voice and preserves its timbre.

When Should a Patient Be Seen by an Otolaryngologist?

There are a number of different reasons for a referring physician to refer a patient to, or for that matter the patient themselves to consider being seen by, an otolaryngologist. Although dysphonia is rarely due to a worrisome cause such as a tumor or cancer, this diagnosis should be considered in someone with progressive hoarseness, particularly in a patient with a history of tobacco or alcohol use, or in the face of other symptoms such as progressive pain or discomfort, pain or difficulty swallowing, hemoptysis, or a neck mass. For intermittent hoarseness, or very gradual progression of dysphonia, in a patient that does not have the above more-worrisome history or symptoms, referral should be based in part on the level of concern of the patient and

their perceived degree of disability. In such cases it is probably best to have them seen and examined by an otolaryngologist to reassure the patient, confirm the diagnosis, and help in the coordination of treatment.

Treatment of the Disordered Aging Voice

The approach to treatment of the dysphonic voice that may occur with aging requires thoughtful assessment so that the treatment can be focused on the most fundamental causes and individualized to the needs of the specific patient. There are three major avenues of treatment. [11] The first is to treat a specific etiology, such as reflux or a neurological disorder. These will be specific to the underlying problem. The second is general medical and environmental treatments. These include things like avoidance of smoke and irritants, hydration, humidification, and mucolytics. This might also include anti-inflammatory therapy and at times even a short course of systemic steroids. The last recourse is to have the patient evaluated and treated by a speech-language pathologist, preferably by an individual with a specific interest in voice disorders (voice pathologist). In many cases they can work with the patient to optimize their technique, voice use environment, and the quantity and volume of voice use. Many patients have dramatic improvements in the quality, strength, and durability of their voice after just a few sessions with a voice pathologist.

Preserving a Youthful Voice: A Multifactorial Treatment

The key to preserving a youthful voice is to be serious about physical exercise,[12] hydration, lubrication of the vocal cords, dental hygiene, muscular activity, nutrition, vitamin and mineral supplements, possibly appropriate hormone therapy, and, often, anti reflux medication. The multiple potential etiologies of a voice problem in the aging patient may make specific identification and treatment difficult, because the disorder may be related to a number of different factors.

In general, people who are conscientious about their overall health will maintain good care of the health of their vocal cords. For the average person this should help to maintain a strong and vibrant voice. For the performer, they can most certainly retain an efficient vocal tessitura and timbre.

Hormonal treatment may be used. Thyroid testing may find a deficiency in thyroid hormones, more commonly found in women, which should be treated. [13]

Many patients who are unhappy with the quality of their voices may benefit from voice therapy. Some will prefer or add singing lessons and join a choir to strengthen their voices. This also allows them to belong to a team, to talk to others, and to routinely practice their voice.
Some will require an acute treatment for arthrosis (anti-inflammatory medicine or injection of steroid in the crico

arytenoid joint), or injection of material in the bowing vocal cord. Dental care is important to maintain good oral hygiene and lubrication. In some cases respiratory therapy may be valuable in improving the breath support of the voice.

Alternative medicine with vitamins, minerals, and antioxidants may not only play an important role in overall health but also in vocal health. Lubrication of the vocal tract is critical to optimizing voice quality. This can be accomplished through hydration and at times the use of mucous thinning medications (mucolytics).

If people do not take proper care of themselves, the voice will age. The vocal register will narrow, the voice will weaken, and the timbre will lose color and become metallic. This may be partially avoided by adopting a regular and constant healthy lifestyle, by taking antioxidants, vitamin C and E, minerals such as magnesium, and by keeping up physical and intellectual activities.

In sum, the human voice is not immune to the effects of aging. Vocal quality and strength can be affected by a number of different conditions that increase in prevalence with age. The memory and the activity of the brain are indispensable to keep a good voice. As time goes by, the register becomes narrow, and the brain command is slower due to loss of neurons.[14] Training the voice and developing vocal memory are important in sustaining a strong voice with age. Fortunately, with appropriate diagnosis and environmental and hygienic interventions, specific medical treatments, and voice therapy, most people can maintain a functional, quality voice through all of their lives.

Quiz

1. Which of the following generalizations about changes in voice with aging is true?
   a. Voice changes occur only at menopause in women.
   b. Laryngeal anatomic development is the same for men and women.
   c. Vocal fold bowing occurs infrequently with late voice aging.
   d. Modifications in voice may be due to a combination of neurological, muscular, and skeletal changes.
   e. Once a voice change occurs, there is little that can be done to improve voice quality.

2. Laryngopharyngeal reflux disease
   a. may be more difficult to treat than gastroesphageal reflux disease.
   b. decreases with age.
   c. is always associated with heartburn.
   d. will not cause any changes in voice.
   e. results in very specific symptoms.

3. Which of the following is not commonly associated with menopause?
   a. Masculinization of the voice
   b. Increased bulk of the vocal folds with increased vocal strength
   c. Decreased flexibility and range of the voice
   d. Reduced neuromuscular flexibility
   e. Increased adipose and changes in fat cell distribution

4. The treatment of a voice disorder in the aging patient
   a. is useless since voice change is inevitable.
   b. should focus more on the specific problem rather than general quality of life issues.
   c. is usually straightforward since the problem is often only from one cause.
   d. may be improved by the assessment and treatment from a voice pathologist.
   e. typically requires surgery.

5. Which of the following is an expected neurological change that occurs in relationship to the voice with aging?
   a. Specific and serious neurological disorders are the most common cause of a change in voice.
   b. Fine muscular coordination of vocal fold movement increases with age.
   c. Multiple neurological changes gradually occur which can reduce voice quality.
   d. Increased lubrication develops due to increased glandular secretion.
   e. Voice tremor is uncommon.

Answers:

1. d
2. a
3. b
4. d
5. c

References

1. Scherer RC, Rubin JS. Laryngeal physiology: Normal and disordered. In: Benninger MS, ed. Benign Disorders of Voice. Alexandria: American Academy of Otolaryngology—Head and Neck Surgery; 2002:29-44.

2. Von Leden H, Alessi DM. The aging voice. In Benninger MS, Jacobson BJ, Johnson AF. Vocal Arts Medicine: The Care and Prevention of Professional Voice Disorders. New York: Thieme Medical Publishers, Inc.; 1994:269-280.

3. See von Leden, 1994, note 2.

4. Benninger MS. Dysphonias secondary to neurologic disorders. J Singing. 1996;52:29-32. Also see Benninger MS, Schwimmer C. Functional neurophysiology. In: Gould J, Rubin P, eds. Diagnosis and Care of Vocal Disorders. Tokyo: Igaku-Shoin Publishers; 1995:105-121.

5. See Benninger, 1996, note 4.

6. Kaufman JA, Amin MR, Aviv JE, et al. Laryngopharyngeal reflux (LPR). Ear Nose Throat. 2002;81(9):1-32.

7. Woo P, Casper J, Colton R, Brewer D. Dysphonia in the aging: Physiology versus disease. Laryngoscope. 1992;102:139-144. Also see Malmgren LT, Jones CE, Bookman LM. Muscle fiber and satellite cell apoptosis in the aging human thyrarytenoid muscle: A sterological study with confocal laser scanning microscopy. Otolaryngol Head Neck Surg. 2001;125:34-39.

8. Gago N, et al. Progesterone and the oligo dendroglial lieage: Stage-dependant biosynthesis and metabolism. December 2001;36-3:295-308.

9. Lee JR, Hopkins, V. What Your Doctor May Not Tell You About Menopause. Warner Books; 1996.

10. Ford CN, Bless DM, Loftus JM. Role of injectable collagen in the treatment of glottic insufficiency: A study of 119 patients. Ann Otol Rhinol Laryngol. 1992;101:237-247. See also Hammond TH, Gray SD, Butler JE. Age- and gender-related collagen distribution in human vocal folds. Ann Otol Rhinol Laryngol. 2000;109:913-920.

11. Johnson AF, Jacobson BH, Benninger MS. Management of voice disorders. Henry Ford Hosp Med J. 1990;38:44-47. Also see von Leden, 1994, note 2.

12. Peres G. Nutrition des sportifs. Abregé de médecine du sport, 8ième ed. Masson; 2002.

13. Abitbol J, Abitbol P, Abitbol B. Sex hormones and the female voice. J Voice. 1999;13:424-446.

14. Kansaku K.,A. Yamaura, S. Kitazawa. Sex differences in lateralization revealed in the posterior languages areas. Cerebral Corte.x 2000;10:866-872.

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Chapter 4

American Academy of Otolaryngology—
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Swallowing

Patient Safety and Medicinal Therapy for
Ear, Nose, and Throat Disorders

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According to current predictions, 16.4 percent of individuals in the United States will be over the age of 65 by 2020, making them the fastest-growing segment of the population (U.S. Census Bureau, 1996). The presence of health problems in our aging population is also increasing, with 80 percent of individuals over the age of 65 having at least one chronic illness. The incidence of dysphagia in this aging population is expected to have a significant impact on health care costs and quality of life.

Swallowing is a highly coordinated process involving four phases—oral preparatory, oral, pharyngeal, and esophageal—allowing for the manipulation of a bolus in the oral cavity and safe passage through to the stomach. In the normal swallow, this process is rapid and dynamic involving the functional integration of these phases, which permits passage of liquids and solids without incident from the oral cavity to the stomach. Dysphagia is then any difficulty in the process of deglutition or disruption in any of the phases. Aspiration results when there is misdirection of oropharyngeal or gastric contents into the larynx and lower respiratory tract. The most serious outcome of the misdirected bolus is the development of aspiration pneumonia. The risk of aspiration pneumonia is greater in individuals with other risk factors including comorbidities of other medical illnesses, which are known to be more common in the elderly.

The exact incidence of dysphagia in the aging population is not known, although numerous studies have looked at specific aspects of its prevalence. Loeb and colleagues reported a 44 percent incidence of dysphagia in the elderly that was associated with significant morbidity and mortality. Groher and McKaig looked at the prevalence of dysphagia in institutionalized elderly and found that 31 percent were on a mechanically altered diet presumably due to dysphagia, with the most common underlying etiology being dementia in 42 percent, cerebral vascular accident (CVA) in 30 percent, deconditioning in 12 percent, and Parkinson's disease (PD) in 10 percent. Another study examined the occurrence of silent aspiration in asymptomatic "healthy" elderly individuals with community acquired pneumonia (CAP) versus a control group of age-matched persons using indium chloride scanning, and demonstrated a 71 percent incidence of silent aspiration in the CAP group, further highlighting the significant incidence of dysphagia in the aging population.

Risk Factors and Dysphagia in the Elderly

Does normal aging result in dysphagia or is it always a pathologic state? Elderly individuals are more likely to experience dysphagia from diseases or conditions associ-

ated with the aging process than because of normal aging alone. The general consensus is that subtle changes in healthy elderly persons do not compromise the efficiency of safe oral intake in the absence of other co-morbidities. Thus it is important to differentiate between dysphagia in the elderly caused by risk factors and that due to "normal aging."

Specific risk factors have been associated with the development of dysphagia in the elderly. The most common etiology of dysphagia in the elderly is CVA. The incidence of dysphagia after an acute CVA is estimated to be 40–70 percent. About 50 percent of elderly stroke patients aspirate in the immediate period after the insult, and about 25 percent die of aspiration pneumonia within the first year of rehabilitation. Studies looking at the localization of the CVA and the presence of dysphagia have shown that larger infarcts are associated with a higher likelihood of aspiration. Subcortical and periventricular white matter lesions are more associated with poor lingual coordination and dysphagia affecting the oral phase of swallowing. Infarcts in the brainstem area have a higher incidence of dysphagia and aspiration.

The presence of other neurological disorders associated with dysphagia also increase in incidence with aging. Parkinson's disease is the most common neuromuscular disease in the elderly with frequent occurrence of dysphagia. Bird and colleagues noted a 15–20 percent incidence of aspiration on radiologic study in asymptomatic individuals with PD. Dysphagia in PD is typically multifactorial and related to rigidity/bradykinesia-causing delays In addition, amyotrophic lateral sclerosis (ALS) is also more frequently seen in older individuals. ALS involves rapid and progressive degeneration of upper and lower motor neurons. It is important to note that asymptomatic individuals with the spinal or nonbulbar form of ALS may still present with dysphagia because of compromised breath support. Dray and colleagues have noted that while laryngeal sensation is typically maintained in individuals with ALS, allowing for aspiration to be sensate, voluntary clearance from the airway may be affected by decreased pulmonary/abdominal support.

Polypharmacy is another risk problem in the elderly with a strong association between certain classifications of medications and the development of dysphagia. Anxiolytics like benzodiazepines are frequently prescribed in older individuals and known to metabolize more slowly, making them more suspect for the development of dysphagia because of their sedating affects and the consequent depression of the central nervous system (CNS). In another study, the researchers witnessed incidents of aspiration in the elderly and noted that the ingestion of sedative medications presumably impairing the cough reflex were the most important risk factor. Other medications frequently consumed by the elderly with similar effects on the CNS include antihistamines, phenothizine-based antiemetics, anticonvulsants, antipsychotics, opiates, and lithium, which impair cognition and awareness. Neuroleptic medications may predispose to extrapyramidal effects further leading to problems with swallowing. Additionally, anticholinergic medications, which have a drying effect on the oral mucosa, may also interfere with swallowing by impairing bolus transport and are known to be dose-dependent with increasing age.

The presence of bacteria in the oral cavity predisposes individuals with dysphagia and aspiration to more serious consequences like pneumonia. In the elderly, increased oropharyngeal colonization with pathogens like staphylococcus aureus and aerobic gram-negative bacilli is more prevalent. Additionally, edentulous elderly institutionalized individuals have a lower risk of aspiration pneumonia versus dentate elderly because of poor dental hygiene. A relation between oropharyngeal aspiration and low serum albumin has been found to increase the risk for the development of pneumonia, highlighting the importance of adequate nutrition in the elderly.

Age-Related Changes and Dysphagia

Studies of changes in the swallowing mechanism caused by aging have demonstrated interesting findings. Changes in skeletal muscle strength are noted with age and may include reduced facial muscle strength resulting in poor cup drinking and decreased masticatory strength; decreased lingual pressure to drive the pharyngeal swallow; increased connective tissue within the body of the tongue restricting bolus control with repetitive tongue movements needed to clear the bolus; and frequent premature spillage of material over the base of the tongue.

Many of the physiologic changes that occur with aging are subtle, progressing slowly over time, which may allow the "healthy elderly" to adapt with compensatory mechanisms without incidents. It is also known that base-of-tongue propulsion is critical to bolus transit into the upper esophageal sphincter (UES). Robbins and colleagues have studied lingual pressures in the elderly and found that pressures do not decline with age but differences between maximum isometric and swallow pressures decrease, suggesting that as individuals age they accommodate by working harder to maintain critical pressures for safe and effective bolus passage.

A delay in triggering the pharyngeal swallow has also been reported with normal aging. A study of older versus young asymptomatic individuals found that the older group was more likely to have a delayed swallow response with multiple swallows needed to clear a bolus. They additionally found that older individuals had a three-fold increase in the action of inspiration versus exhalation after the swallow, increasing the likelihood of laryngeal penetration caused by residual material remaining in the hypopharynx after the first swallow.

Oral and pharyngeal transit times are another area of swallowing physiology that is subject to change with aging. Older individuals have been found to have prolonged oral bolus transit times, and increased pharyngeal bolus transit times, placing them at increased risk of aspiration. Other authors have found increased duration of the swallow with longer closure and opening of the airway and UES with increased age in asymptomatic women. In addition, Robbins' study showed that the speed of swallowing gradually slows after age 45 with a significant difference between individuals below 45 versus over 70 years of age.

Timely laryngeal motion, both superiorly and anteriorly, is critical to airway protection during swallowing. Studies have suggested changes in the strength and timing of laryngeal elevation with secondary effects on cricopharyngeal relaxation. Logemann and colleagues studied laryngeal movement in older individuals and found gender differences. Older men were found to have reduced hyoid motion while older women retained muscular reserve. Additionally, they found that cricopharyngeal relaxation was reduced in the males implicatinga greater risk for dysphagia in normal older men because of their lower muscular reserve. However, the degree of reduced motion and its relation to dysphagia is not as clear. Reduced resting tone in the upper esophageal sphincter resulting in delayed UES relaxation is seen in older individuals, with decreased cross-sectional area in the esophageal inlet. An additional consequence of the change in UES function is the increased incidence of Zenker's diverticulum in the elderly.

A connection between breathing and, more specifically, increased apnea duration during the swallow has been found with aging and is felt to be a protective mechanism because it enables the system to compensate for other age-related changes including longer oropharyngeal and hypopharyngeal transit times and delayed initiation of maximum hyolaryngeal excursion. In contrast, another study found longer swallowing apnea duration in women versus men, which, in fact, decreases with age. Further evidence of changes in breathing and swallowing with advanced age is found in a study showing that SpO2 levels were lower only in elderly individuals with dysphagia. Oxygenation during swallowing was not affected in healthy, asymptomatic older individuals.

The cough reflex is an important respiratory defense mechanism against aspirated material. Changes in the cough reflex with age have been reported by some authors, placing the elderly at greater risk for aspiration pneumonia. Other researchers have not been able to replicate this decline. Nakazawa and colleagues studied three groups of elderly individuals: healthy and asymptomatic, dementia but no suspected dysphagia, and history of aspiration pneumonia. The only group that had significant changes in the cough reflex was the one with a history of aspiration pneumonia, which demonstrated increased latencies in reaction to inhaled citric acid versus the other two groups. This was confirmed by another study that also found no significant change in the cough reflex in asymptomatic older individuals, and theorized that the results from the other studies may have been related to other risk factors prevalent in that population.

Weight loss is a frequent sign of dysphagia. However, eating history and weight stability are not always consistent. Some of the above-mentioned physiologic changes that occur with aging happen gradually, allowing the individual to adapt and compensate. This is frequently accomplished by taking nutritional supplements or more calorie-rich-type foods like shakes and puddings, thus disguising their underlying difficulty swallowing. Additionally, changes in the sense of taste and smell may affect the diet of the elderly. Flavor preferences have been shown to shift in older individuals as they perceive greater concentrations of sugar and salt as being pleasant, despite the fact that these substances are frequently restricted from

their diets. A loss of chemosensory perception altering the sense of smell is also found with age, and is related to degeneration of the olfactory nerve and mucosa that may interfere with older individuals having difficulty identifying blended foods. Thus, it appears that older individuals may benefit from stronger-flavored foods to compensate for their decreased perception, which in turn may have a positive effect on appetite and nutrition.

In summary, dysphagia is an increasing problem in the elderly. Although specific physiologic changes occur with the normal aging process, these are frequently well compensated for in the healthy older person. It is further critical to recognize the relationship between other co-morbidities and the normal physiologic changes, because these co-morbidities significantly increase the likelihood of developing dysphagia and its potential complication of aspiration pneumonia.

References

1. U.S. Census Bureau. Current population Reports: Population Projections of the United States by age, sex, race, and Hispanic origin—1996–1050. 1996;P25-1130.

2. Nolan L, O'Malley K. Prescribing for the elderly, II: Prescribing patterns—differences due to age. J Amer Ger Soc. 1988;36,245-254.

3. Agree EM, Freedman VA. Implications of Population Aging for Geriatric Health. Baltimore, MD: Johns Hopkins Population Center, Papers on Population. 1998;WP98-105.

4. Robbins J, Hamilton J, Lof G, Kempster G. Oropharyngeal swallowing in normal adults of different ages. Gastroenterology. 1992;103:823-829.

5. Dodds WJ. The physiology of swallowing. Dysphagia. 1989;3:171-178; see also Miller A. Neurophysiological basis of swallowing. Dysphagia. 1986;1:91-100.
See Miller, 1986, note 5.

6. Marik PE. Aspiration pneumonitis and pneumonia: A clinical review. N Engl J Med. 2001;344:665-672.

7. Loeb M, McGreer A, McArthur M, Walker S, Simor AE. Risk factors for pneumonia and other lower respiratory tract infections in the elderly residents of long-term care facilities. Arch Intern Med. 1999;27:2058-2064.

8. Groher ME, McKaig, TN. Dysphagia and dietary levels in skilled nursing facilities. J Am Geriatr Soc. 1995;43:528-532.

9. Kikuchi R, Watabe N, Konno T, et al. High incidence of silent aspiration in elderly patients with community-acquired pneumonia. Am J Respir Crit Care Med. 1994;150:251-253.

10. Kuklemeier KV. Epidemiology and dysphagia. Dysphagia. 1994;9:209-217. See also Baum BJ, Bodner L. Aging and oral motor function: Evidence for altered performance among older persons. J Dent Res. 1983;62:2-6.

11. Logemann JA. Swallowing physiology and pathophysiology. Otolaryngol Clinics of N Amer. 1988;21:613-623. Also see Buchholtz DW, Robbins, JA. Neurologic disease affecting oropharyngeal swallowing. In: Perlman AL, Delrieu K, eds. Deglutition and Its Disorders. San Diego, CA: Singular Publishing Group; 1997:319-342.
Schindler JS, Kelly JH. Swallowing disorders in the elderly. Laryngoscope. 2002;112:589-602.

12. Gordon C, Langton-Hewer R, Wade DT. Dysphagia in acute stroke. Br Med J. 1987;295:411-414. See also Kidd D, Lawson J, Nesbitt R, et al. Aspiration in acute stroke: A clinical study with videofluoroscopy. Quar J Med. 1993;86:825-829; and Mann G, Dip GP, Hankey GJ, Cameron D. Swallowing function after acute stroke: Prognosis and prognostic factors at 6 months. Stroke. 1999;30:744-748. See Kidd et al., 1993, note 14.

13. Dray TG, Hillel AD, Miller RM. Dysphagia caused by neurologic deficits. Otolaryngol Clinics of N Amer. 1998;31:507-524. See Kidd et al., 1993, note 14.

14. Daniels SK, Brailey F, Foundas AL. Lingual discoordination and dysphagia following acute stroke. Dysphagia. 1999;14:85-92.

15. Kim H, Chung S-D, Lee K-H, Robbins J. Aspiration subsequent to a pure medullary infarction: Lesion sites, clinical variables and outcome. Archive of Neurology. 2000;57:478-483. See Schindler and Kelly, 2002, note 13.

16. Bird MR, Woodward MC, Gibson EM, Phyland DJ, Fonda D. Asymptomatic swallowing disorders in elderly patients with Parkinson's disease: A description of the findings on clinical examination and videofluoroscopy in sixteen patients. Age and Ageing. 1994;23:251-254.
See Dray et al, 1998, note 16. See Schindler and Kelly, 2002, note 13.

17. Vergis EN, Brennen C, Wagener M, et al. Pneumonia in long-term care: A prospective case-control study of risk factors and impact on survival. Archives of Internal Medicine. 2001;161:2378-2381. See Schindler and Kelly, 2002, note 13.

18. Palmer L, Albulak K, Fields S, et al. Oral clearance and athogenic oropharyngeal colonization in the elderly. AJRCCM. 2001;164:464-468.

19. Terpenning M, Bretz W, Lopatin D, Langmore S, et al. Bacterial colonization of saliva and plaque in the elderly. Clinics of Infectious Disease. 1993;16(Suppl 4): S314-S316.

20. Riquelme R, Torres A, El-Ebiary M, de la Bellacasa JP, et al. Community-acquired pneumonia in the elderly: A multivariate analysis of risk and prognostic factors. AJRCCM. 1996;154:1450-1455.

21. Fucila S, Wright PM, Chan I, Yee S, et al. Functional oral-motor skills: Do they change with age? Dysphagia. 1998;13:195-201.

22. Jaradeh S. Neurophysiology of swallowing in the aged. Dysphagia. 1994;9:218-220.

23. Nicosia MA, Hind JA, Roecker EB, Carnes, M. et al. Age effects on the temporal evolution of isometric and swallowing pressure. J Gerontol A Biol Sci Med Sci. 2000;55(11)M634-640; also see Yokoyama M, Mitomi N, Tetsuka K, Tayama N. Role of laryngeal movement and effect of aging on swallowing pressure in the pharynx and upper esophageal sphincter. Laryngoscope. 2000;110:434-439. See Jaradeh, 1994, note 30. See Schindler and Kelly, 2002, note 13.

24. Robbins JA, Levine R, Wood J, Roecker EB, Luschei E. Age effects on lingual pressure generation as a risk factor for dysphagia. Journal of Gerontology. 1995;50A: M257-M262.

25. Nilsson J, Ekberg O, Olsson R, et al. Quantitative aspects of swallowing in an elderly nondysphagic population. Dysphagia. 1996;11:180-184.

26. Shaw DW, Cook IJ, Dent J, Simula ME, et al. Age influences oropharyngeal and upper esophageal sphincter function during swallowing. Gastroenterology. 1990;98: A390. See also Cook IJ, Doods WJ, Dantas RO, Kern MK, et al. Timing of videofluoroscopic, manometric events, and bolus transit during the oral and pharyngeal phases of swallowing. Dysphagia. 1989;4:8-15.

27. Tracy JF, Logemann JA, Kahrilas PJ, Jacob P, et al. Preliminary observations on the effects of age on oropharyngeal deglutition. Dysphagia. 1989;4:90-94. See also Cook et al., 1989, note 36.

28. Johnson ER, McKenzie SW, Sievers A. Aspiration in stroke. Archives of PM&R. 1993;74:1-4.

29. These authors include Rademaker AW, Pauloski BR, Colangelo LA, Logemann JA. Age and volume effects on liquid swallowing function in normal women. JSLHR. 1998;41:275-284; Sonies BC, Baum BJ, Shawker TH. Tongue motion in elderly adults: Initial in situ observations. J Gerontol.1984;39:279-283; Sonies BC, Parent IJ, Morrish K, Baum BJ. Durational aspects of the oral-pharyngeal phase of swallow in normal adults. Dysphagia 1988;3:1-10; and Robbins, et al., 1992, note 4.

30. Logemann JA, Pauloski BR, Rademaker AW, Kahrilas PJ. Oropharyngeal swallow in younger and older women: Videofluoroscopic analysis. JSLHR. 2002;45:434-445.

31. Fulp SR, Dalton CB, Castell JA, Castell DO. Aging-related alterations in human upper esophageal sphincter function. Am J of Gastroenterology. 1990;85:1569-1572.

32. Shaker R, Ren J, Zamir Z, et al. Effect of aging, position and temperature on the threshold volume triggering pharyngeal swallows. Gastroenterology. 1994;107:396-402.

33. Jordan PH. Dysphagia and esophageal diverticula. Postgraduate Medicine. 1977;61:155-161.
See Robbins et al., 1992, note 4.

34. Hiss SG, Treole K, Stuart A. Effects of age, gender, bolus volume, and trial on swallowing apnea duration and swallow/respiratory phase relationships of normal adults. Dysphagia. 2001;16:128-135.

35. Colodny N. Effects of age, gender, disease, and multisystem involvement on oxygen saturation levels in dysphagic persons. Dysphagia. 2001;16:48-57.

36. Sekizawa K, Ujiie Y, Itahashi S, et al. Lack of cough reflex in aspiration pneumonia. Lancet. 1990;335:1228-1229.
See also Nakajoh K, Nakagawa T, Sekizawa K, et al. Relation between incidence of pneumonia and protective reflexes in post-stroke patients with oral or tube feeding. JIM, 2000;247:39-42.

37. Nakazawa H, Sekizawa K, Ujiie Y, et al. Risk of aspiration pneumonia in the elderly. Chest. 1993;103:1636-1637.

38. Katsumata U, Sekizawa K, Ebihara T, et al. Aging effects on cough reflex. Chest. 1995;107:290-291.
Gustafson B, Tibbling L. Dysphagia: An unrecognized handicap. Dysphagia. 1991;6:193-199.
Murphy C. Senescence and clinical changes in the olfactory system: Psychophysical consideration. (NIDCD Monograph), Development, Growth, and Senescence in the Chemical Senses. 1992;3:153-160.

39. Doty RL, Shamon P, Allekbaum SL, Giberson R, et al. Smell identification ability: Changes with age. Science. 1984;226:1441-1443.

40. Cain WS, Stevens JC. Uniformity of olfactory loss in aging. Annals of the New York Academy of Science. 1989;561:29-38.

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Chapter 5

American Academy of Otolaryngology—
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Facial Plastic Surgery

The Aging Face— Benefits and Pitfalls of Botox® and Laser Skin Treatments

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The effects of aging on the skin are a result of a combination of many factors. The most obvious manifestations of aging are recognizable in the face. Most adults can instantly estimate a person's age at a glance, based on the quality and certain characteristics of the facial skin and the underlying facial contours. We have a mental image of what a person looks like in the 40s or 50s or beyond, and most of us strive to look "good" for our actual age. Although aging skin is a fact of life, and some of the effects of aging are related to genetic factors beyond our control, many treatments and techniques are available today through facial plastic surgeons and other skin care specialists to help reduce the effects of aging on skin. In particular, there are advances in nonsurgical management of senescent skin that are increasingly effective in combating the signs of aging, such as injectable agents like Botox® (botulinum toxin A) and laser skin treatments. Proper matching of techniques with specific age-related changes is important for successful management.

Effects of Aging on Skin

The appearance of the aging face is a result of many factors, both genetic and environmental. There are intrinsic factors of cutaneous aging that are characterized by a loss of skin elasticity and volume. There is a loss of the rete pegs in the cutaneous dermal-epidermal junction and atrophy of the dermis, resulting in thinning, laxity, and fine wrinkling of the skin. In addition, there is a loss of the underlying adipose tissue in the face, known as lipoatrophy, resulting in a thinner, increasingly gaunt appearance to the face. The roundness of the cheeks and lips associated with youth gives way to thinning of the lips, hollowing of the cheeks and orbits, and creping of the eyelid skin.

Photoaging of the skin causes the most pronounced negative impact on the senescent face. Overexposure to ultraviolet light from the sun is the cause of the majority of the damage associated with the aging face. Glogau classified photoaged skin into four subtypes based on the clinical manifestations of photoaging, including changes in skin color, wrinkling, and texture.[1] Photodamage is the cause for "age spots," dyschromias, lentiges, telangiectasias, and the progressive sallow yellow color and overall splotchy pigmentation of the senile facial skin. Poikilodermia of Civatte—the "redneck" appearance noted on photoaged cervical and cheek skin—is primarily the result of solar damage. With increasing sun exposure and photodamage, surface growths and scaling such as actinic keratoses, seborrheic keratoses, and even skin

cancers appear on the face, giving the skin surface a rough, uneven texture. Premature senescent changes of the skin are also associated with other environmental factors, most notably smoking and poor nutritional status.

Expression lines or laugh lines are dynamic, hyperkinetic rhytids of the face resulting from repetitive facial muscle movements. Because the muscles of the face insert into the deep layers of skin, movements of these muscles cause crinkling of the overlying skin. Over the years, this repetitive crinkling of the skin causes permanent creases or deep rhytids on the face. These are most noticeable in the area around the large muscles of the eyes, forehead, and mouth, where smiling, laughing, and frowning cause permanent changes in the dermis. The vertical frown lines in the glabella region between the eyebrows result from contraction of the corrugator muscles, while the horizontal rhytids at the root of the nose are due to contraction of the procerus muscle. Crow's feet lines form with contraction of the orbicularis oculi, and the large horizontal rhytids across the forehead result from brow elevation by the frontalis muscle. Vertical lines form around the lips, especially in smokers, from repetitive puckering and sucking.

Last, but not least, gravity and its constant downward pull on the soft tissues of the face is a factor in aging, and the facial structures are not immune to its effect. With increasing age, as the skin intrinsically becomes progressively thinner and less elastic, gravity acts on the facial soft tissues. The brow lines droop, the lower eyelids sag, jowls form along the jaw line, and the malar eminence (or

prominence of the cheek) crowds downward toward the nasolabial fold. In addition, the nasal tip begins to point downward and the earlobes elongate, giving adage to the old children's song, "Do your ears hang low."

Treatment Options

A variety of treatment options are available to help diminish the signs of aging. Certainly, many surgical options are available to improve one's appearance, such as rhytidectomy, blepharoplasty, and brow lift, and these procedures are the primary armament in the correction of the gravitational effects of aging. The focus of this discussion, however, is on minimally invasive procedural treatments for the face to improve age-related skin changes. These include injectable medications and materials that can reduce the signs of aging, such as botulinum toxin A, as well as laser resurfacing treatments used to improve skin quality and contour.

It is important to note that the prevention of further skin damage by avoiding overexposure to the sun and using sun screen regularly should be the first-line treatment for all patients. Smoking cessation and maintaining good nutrition and hydration will also help reduce age-related skin changes. In addition, a multitude of medical skin treatments and cosmetic products are available that can be modestly beneficial in reducing fine rhytids and pigment irregularities with long-term use, such as topical applications of antioxidant products, retinoic acids, glycolic acids, or growth factor serum.

Botox ®

A main focus of this paper is a discussion of the use of botulinum toxin in management of the aging face. Botox is the trade name for purified botulinum toxin A made by the Allergan corporation. Although botulinum toxin is considered one of the most lethal known poisons, in small therapeutic doses it is extremely safe and effective in treating hyperkinetic expression lines. It has been used safely in clinical applications since the 1970s to treat functional disorders like strabismus, blepharospasm, and spasmodic dysphonia. In 2002, Botox was approved by the FDA for cosmetic purposes for the treatment of the vertical and transverse frown lines of the glabella between the eyebrows. These are caused by contraction of the corrugator and procerus muscles, respectively. Since its FDA approval for cosmetic use, many new uses for Botox have been explored and touted by physicians.

Botulinum toxin causes a temporary weakness of the targeted facial muscle, which can last for three to five months. It is a neuromuscular blocking agent that works locally at the neuromuscular junction to block the release of acetylcholine, a nerve transmitter, at the presynaptic membrane of the nerve terminals. [2] Because acetylcholine is blocked, the force of muscular contraction is reduced. In the region of the injection, it effectively causes a reversible denervation of the injected muscle tissue. This results in muscle atrophy in the region of the injection, with reduction of the muscle fiber diameter and weakness. Because the neurotoxic effects are localized, only parts of the muscle are affected, and because only small amounts of toxin are used clinically, the muscle is not completely paralyzed (contrary to some representations in the media). With weaker and less repetitive movement of the muscle, there is less crinkling of the overlying skin, and the expression lines smooth out or are even eliminated.

Botulinum toxin A is most frequently used in the upper third of the face to treat the dynamic expression lines of the glabella, forehead, and periorbital region. Excellent results have been reported in reducing the hyperkinetic rhytids in these areas. [3] The effects of Botox are noted approximately 72 hours to one week after injection. Many doctors have used the toxin effectively to treat other hyperkinetic lines, most notably nasal scrunch or "bunny" lines, vertical lip rhytids or "smoker's lines," and the marionette lines along the melolabial folds. Botulinum toxin has also been found to be useful in treating the muscle hypertrophy seen in platysmal banding of the neck.

Although Botox has an excellent safety record, it is important to weigh the desired clinical effect with injection at any of these sites with the potential for overtreatment and complications. Botulinum toxin is produced by the bacteria Clostridium botulinum, which is the cause of botulism. Botulism is most commonly linked to food contamination, and can result in a generalized flaccid paralysis leading to death if respiratory support is not provided. Fortunately, the chemodenervation that occurs after botulinum toxin injection is temporary. There have been no long-term complications reported in the use of Botox

in the face, and there have been no life-threatening allergic reactions reported. [4]

The recent cases of injection-induced botulism reported in Florida, in 2004, were reportedly caused by injections with exceedingly large doses of an unlicensed botulinum toxin product labeled for animal research. The average patient injection for aesthetic purposes generally ranges between 25 and 50 units. Each vial of Botox contains only 100 units of purified toxin, while the estimated minimum lethal dose of neurotoxin would be equivalent to approximately 3,000 units.

Minor, regional complications of Botox injections may occur following treatment. Ecchymosis and pain at the site of the injection are the most common reported complications, and may be dependent upon the injection technique itself. Headaches, dry mouth, nausea, and flu-like symptoms can arise after Botox injections, as well as localized skin dryness due to decreased sweat gland activity.[5] Allergic reactions to Botox are extremely rare. Botulinum toxin injections are contraindicated in patients with a skin infection in the area of the proposed injection, and should not be used in patients with peripheral neuropathic or neuromuscular disorders such as amyotrophic lateral sclerosis or myasthenia gravis.

The majority of the remaining potential complications can be classified as localized functional or esthetic deficits secondary to diffusion or improper placement of the toxin during injection. Blepharoptosis is the most frequently reported deficit resulting as an unintentional side effect from periorbital toxin injections, with an estimated incidence of 1 to 3 percent. [6] Injection outside the orbital rim is recommended to prevent inadvertent toxin effects on the levator palpebrae superioris muscle. Digital pressure on the orbital rim as the injection is performed can also reduce accidental diffusion of toxin into the orbital zone. Botulinum toxin-induced blepharoptosis is temporary but, if necessary, can be managed with a topical alpha adrenergic agonist. Aproclonidine 0.5 percent eye drops can be applied to help stimulate Mueller's muscle to raise the lid 1 mm to 2 mm.

Other areas that may develop unintentional functional deficits include brow ptosis following injection of the frontalis muscle. Because this is the only brow elevator, unopposed action by the brow depressor muscles (corrugator, procerus, and orbicularis oculi) can cause an apparent droop in the eyebrows. Cautious, conservative injection of the frontalis, along with simultaneous injection of the corrugator, can help prevent this effect. The use of botulinum toxin in the perioral area can be complicated by oral incompetence or asymmetries. Keeping melolabial injections 1 cm away from the oral commissure and injecting superficially along the vermilion will smooth out vertical lip rhytids, while reducing the risk of incompetence. Additionally, injection too deeply into the platysmal bands can result in dysphagia or alterations in the voice by affecting the strap and cricothyroid muscles.

Laser Facial Rejuvenation

Although botulinum toxin injections are aimed primarily at dynamic, hyperfunctional rhytids of the upper third of the face, laser treatments to the skin are aimed at correcting the effects of photoaging. Differentiation between static and dynamic wrinkles and the degree of photoaging should be ascertained before attempting laser resurfacing. Rhytids secondary to facial muscle contraction are more impervious to laser resurfacing than nondynamic wrinkles. Improvement in nondynamic wrinkles, skin elasticity, dyschromias, and skin texture irregularities are the expected benefits of laser resurfacing. Laser treatment rejuvenation modalities can be classified as either ablative or nonablative.

Ablative Modalities

The goal of ablative skin resurfacing is to remove the photodamaged epidermis and replace it with a new, undamaged epidermal layer, while enhancing and tightening the dermis through increased collagen and elastin formation. Ablative skin resurfacing techniques denude the epidermis and papillary dermis through selective photothermolysis and controlled tissue vaporization. Re-epithelialization occurs within 7 to 14 days due to the abundance of skin appendages (sweat glands and hair follicles) in the face that provide a source for epidermal cells that migrate upward to form new epithelium. The new skin is fresher, younger looking, smoother, and with fewer pigmentary irregularities. The most commonly used

ablative resurfacing lasers include the CO2 and erbium: yttrium-aluminum-garnet (Er:YAG) lasers.

CO2 Laser. The pulsed CO2 laser emits a 10,600-nm wavelength of infrared light, which targets intra- and extracellular water in the skin tissue. Because skin is 70 percent water by volume, the laser can effectively and selectively vaporize the skin tissue. Histologic evaluation after laser resurfacing with the CO2 laser reveals it can ablate approximately 50µm to 150µm of tissue per pass, so that two or sometimes three passes are sufficient for removing the epidermis and papillary dermis.[7] Advances in laser technology have allowed delivery of the CO2 laser pulse at short pulse durations of less than 1 millisecond. Because this pulse duration is shorter than the thermal relaxation time of skin, the pulsed CO2 laser leaves a relatively thin zone of nonspecific thermal damage in the residual dermis, when compared to a nonpulsed CO2 laser.

There are associated thermal effects of this ablation that are desirable—that lead to collagen shrinkage, wound contraction, and subsequent skin tightening. There is also photothermal coagulation at the wound base, so there is little bleeding or wound seepage in the immediate post-treatment period. Although the degree of thermal injury caused by the CO2 laser plays a significant role in the extent of postlaser collagen remodeling, this thermal damage can extend up to 100 µm, which may lead to complications such as prolonged erythema, hyper- or hypopigmentation, or even scarring.[8] Other potential complications of laser resurfacing include wound infection, milia formation, contact dermatitis, and delayed healing.

Wound healing after ablation proceeds with re-epithelialization and new collagen and elastin fiber formation in the dermis. The end result reveals significant improvement in skin quality, with smoother, firmer, and tighter skin. However, postprocedure erythema may last for several weeks to months. Although the reduction in photodamage and wrinkling of the skin following CO2 laser treatments is excellent, the enthusiasm for the procedure is tempered by both the long healing time and the degree of anesthesia often required for patient comfort during the procedure.

Er:YAG Laser. The erbium laser has a wavelength of 2,940 nm, which has ten times greater water absorption than the CO2 laser.[9] Consequently, it ablates less tissue per pass (20µm to 40 µm), and has a narrower zone of thermal necrosis (5 to 10 µm per pass). Almost all of the erbium laser energy is converted to water vaporization, leaving less tissue debris and thermal damage. Because it produces less thermal injury than the CO2 laser, faster healing time can result, with less risk of complications. Alternatively, because it results in less thermal injury, it also produces less collagen remodeling and therefore less significant skin-tightening effect. It is not as effective as the CO2 laser as a single modality for correcting moderate facial rhytids. However, when used in combination therapy along with the CO2 laser, it can be a very effective tool in treatment of the photoaged skin.[10] By combining the two lasers, tissue ablation is maximized while detrimental thermal injury effects are minimized.

Nonablative Laser Rejuvenation

Nonablative laser rejuvenation avoids some of the potential risks of ablative procedures, by inducing a dermal inflammatory healing response without causing appreciable injury to the epidermis. There is marked reduction in recuperative time following nonablative laser rejuvenation, compared with ablative techniques. Because there is significantly less postoperative edema and erythema, patients can be treated and return to work the same day. In addition, there is much less pain associated with the procedure, essentially eliminating the requirements for periprocedural anesthesia. Although these nonablative technologies have been shown to diminish rhytids to a variable degree the dermal remodeling effect is not as significant or pronounced as ablative modalities. The nonablative technologies are not as efficacious as the carbon dioxide or Er:YAG lasers for reduction of moderate and severe rhytids, but can be ideal for modest cutaneous improvement in patients with early photoaging, who desire little or no downtime following the procedure.

The most effective modalities for nonablative facial rejuvenation use mid-infrared lasers, such as the 1320-nm neodyminium:yttrium-aluminum-garnet (Nd:YAG) laser or the 1064-nm Q-switched Nd:YAG laser. Mid-infrared laser light penetrates to the papillary and upper reticular dermis to promote collagen remodeling in the dermis, while bypassing the epidermis. However, there is still nonselective heat transfer to the epidermis. Combining these lasers with a cooling mechanism for the skin surface helps

limit epidermal injury. Cooling is accomplished in several ways: nondynamic precooling of the skin, a dynamic coupling of the laser pulse with a cooling cryogen, and/or skin cooling delivered immediately after the laser pulse delivery (quenching). This allows for better control of the surface temperature, maintaining skin temperatures from 40°C to 48°C, while allowing dermal temperatures to reach 60°C to 70°C, at which thermal-induced activation of dermal fibroblasts and dermal remodeling can occur. Cooling protects the epidermis and reduces the risk of pigmentary changes and scarring, even in Fitzpatrick skin types IV and V.[11]

In sum, many options are available to improve the effects of aging on facial skin. Botox and laser facial rejuvenation are two of the most common nonsurgical adjuncts in the treatment spectrum. When selecting a treatment modality, it is important to assess each patient to ascertain both the etiology of their senescent skin pathology and their expectations of treatment. The physician must appropriately match these expectations with the technique most likely to effect the desired change. Frank communication and education of the patient is essential for directing patient expectations. As in all cosmetic procedures, proper patient selection is key to a successful outcome.

Quiz

1. Vertical, dynamic wrinkles in the glabellar region of the forehead are best treated with

   a. nonablative laser facial rejuvenation therapy with the Nd:YAG laser.
   b. Botox injection of the frontalis muscles.
   c. ablative laser resurfacing with the Er:YAG laser.
   d. Botox injection of the corrugator muscles.

2. The most effective treatment of lax jowling along the lower border of the mandible would be

   a. Botox injection of the orbicularis oris muscle.
   b. a surgical rhytidectomy procedure.
   c. topical retinoic acid treatment.
   d. ablative laser resurfacing with the pulsed CO2 laser.

3. Photodamage of the skin is evidenced by

   a. blepharoptosis of the eyelids.
   b. lipoatrophy in the cheeks.
   c. solar keratoses on the forehead.
   d. laugh lines around the mouth.

Answers:

1. d
2. b
3. c

References

1. Glogau RG. Chemical peeling and aging skin. J Geriatr Dermatol. 1994;2:5-10.

2. Lam S. The basic science of botulinum toxin. Facial Plast Surg Clin N Am2003;11:431-438.

3. Kim DW, Cindiff J, Toriumi DM. Botulinum toxin A for the treatment of lateral periorbital rhytids. Facial Plast Surg Clin N Am. 2003;11:445-451.

4. Kwak CH, Hanna PA, Jankovic J. Botulinum toxin in the treatment of facial tics. Arch Neurol. 2000;57(8):1190-1193.

5. Bulstrode NW, Grobelaar AQ. Long-term prospective follow-up of botulinum toxin treatment of facial rhytids. Aesthetic Plast Surg. 2002;26:356-359.

6. Frankel AS. Botox for rejuvenation of the periorbital region. Facial Plast Surg. 1999;15:255-262.

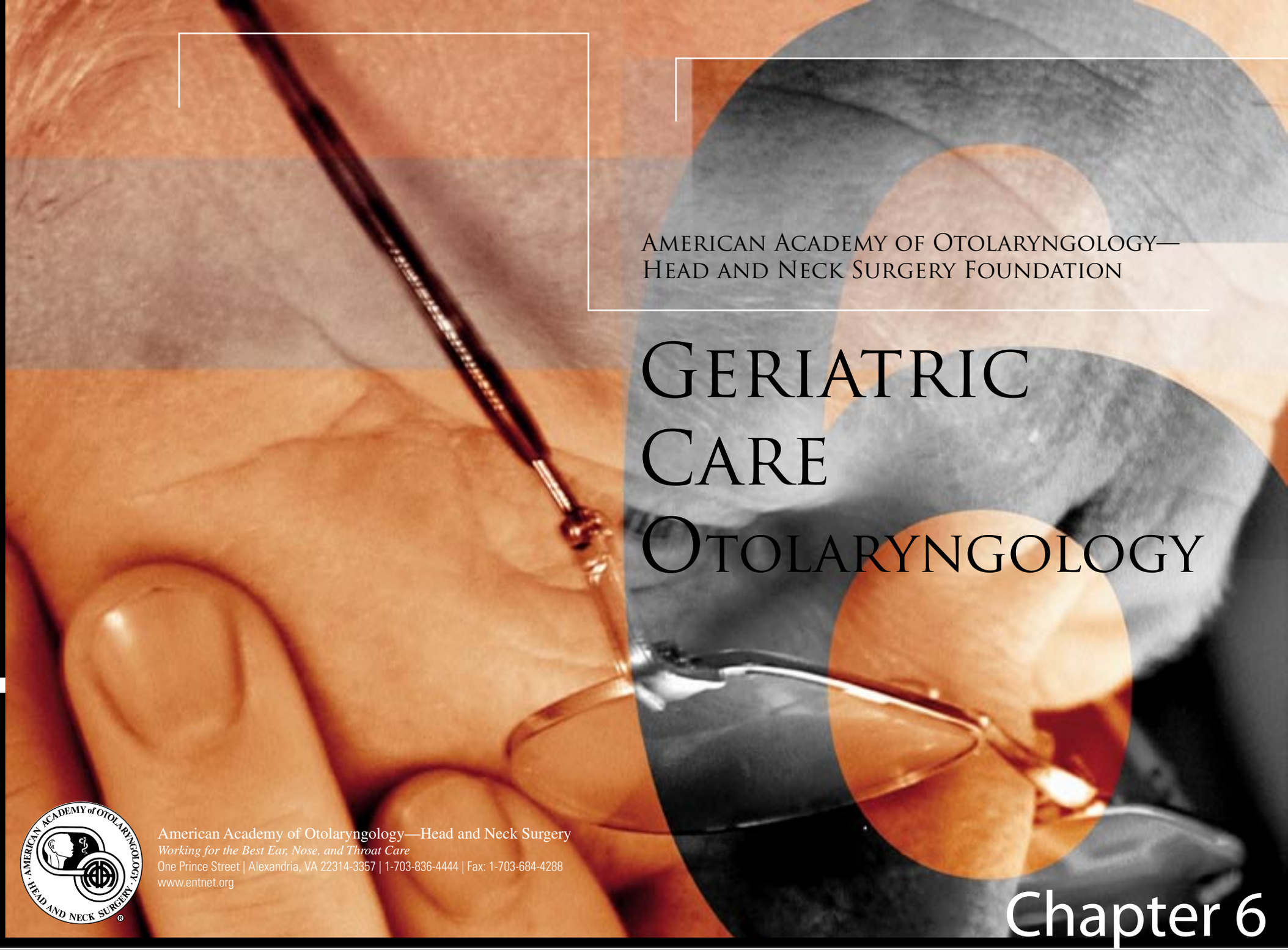
7. Stuzin JM, Baker TJ, Baker TM, et al. Histologic effects of the high-energy pulsed CO2 laser on photoaged facial skin. Plast Reconstr Surg. 1997;99:1036-2050.<<AU: pages look wrong – more than a thousand pages???>>

8. Rubach BW, Schoenrock LD. Histologic and clinical evaluation of facial resurfacing using a carbon dioxide laser with the computer pattern generator. Arch Otolaryngol-Head Neck Surg. 1997;123:929-933.

9. Clark RA. Biology of dermal wound repair. Dermatol Clin. 1993;11:647-666.

10. Goldman MP, Manuskiatti W, Fitzpatrick RE. Combined laser resurfacing with the UPCO2 + Er:YAG lasers. Dermatol Surg. 1999;25:160-163.

11. Pham RT. Nonablative laser resurfacing. Facial Plast Surg Clin N Am. 2001:9:303-910.



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Chapter 6

American Academy of Otolaryngology—
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Rhinosinusitis

Surgical Management of Chronic Rhinosinusitis in the Geriatric Patient

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As our population ages and continued advances in medicine occur, more and more of our patients are moving into the senior age range defined as 65 years of age or older. These people are increasingly susceptible to common respiratory tract infections. The U.S. population has grown by 39 percent in the past 30 years, but the segment older than 65 years has grown by 89 percent and the segment older than 85 years by 232 percent. It is estimated that, by the year 2030, 70 million people will be older than 65 years of age, and comprise approximately 20 percent of the U.S. population.[1]

Questions about Aging and Health

1. Does immune status decrease with age?

The immune system undergoes continuous changes throughout life, with a decline of cell and antibody-mediated immune responses with age.[2] This process is well documented and called immunosenescence. Some evidence in a recent study of healthy centenarians suggests that the immunologic changes observed during aging are consistent with a reshaping, rather than a generalized deterioration, of the main immune functions.[3] The consequences of impaired immune function in the elderly include increased susceptibility to infectious diseases as well as an extended post-illness recovery period.[4]

2. Do respiratory tract infections increase with age?

As people age, they become more susceptible to infections. Infection is a common cause of illness in old age and the fourth most frequent cause of death.[5] Increasing frequency of infections in the elderly has commonly been attributed to a decrease in their immune status and to nonspecific host factors, alterations in the skin and mucosal barriers, as well as nutritional factors and underlying chronic diseases.[6] Some T-cell functions decrease with age while others fail to respond to new antigen challenges. Humoral function appears to be affected by an increase in IgG and IgA and variable changes in IgM levels. A sluggish T-helper cell response along with B-cell defects may explain why the elderly do not have as vigorous response to immunization with vaccines.[7]

Skin changes associated with aging include poorer hydration and loss of depth of the rete pegs, increasing the likelihood of shearing damage to the epidermis. Impairment of mucociliary clearance with aging also contributes to increased risk of respiratory infections.

3. What other co-morbidities are associated with aging?

Malnutrition: Malnutrition frequently occurs in the elderly. Specifically, depletion of muscle mass (sarcopenia) is responsible for the frailty of the elderly. Not only does reduction in muscle mass affect mobility, it results in a diminished capacity for responding to the increase in protein synthesis necessary for fighting disease and infection.[8] Protein-energy malnutrition impairs several aspects of the immune system, including cell-mediated immunity, ability of phagocytes to kill ingested bacteria and fungi, several components of the complement system, mucosal secretory IgA antibodies, and the affinity of antibodies.[9]

Studies demonstrate that even a modest increase in dietary supplements can improve the immune response. Administration of zinc sulfate to patients over 70 years of age for one month increased the number of circulating T cells, delayed cutaneous hypersensitivity to certain purified protein derivatives, and improved serum IgG antibody response to tetanus toxoid.[10] Vitamin C supplementation for a similar time period in the elderly enhanced lymphocyte proliferation responses in vitro and skin reactivity to tuberculin in vivo.[11] Nutritional support is essential in this patient population and should be paramount in the primary care physician's health care plans.

Restricted lifestyle: Few studies have actually studied the role of lifestyle as it affects the immune response in the elderly. Tsukamoto and colleagues investigated the role of lifestyle and neutrophil functions in the elderly.[12] They concluded that stress-coping activities and relationships (for example, hobbies, pets, and close family relationships)

were a positive influence and play a role in the balance between phagocytosis and subsequent superoxide production, which can improve neutrophil function. Total serum protein levels were also noted to play an important role in prevention of infections in this population.

Chronic Rhinosinusitis

Epidemiology

Chronic rhinosinusitis (CRS) is currently the most common chronic ailment in the United States. (134.4 cases per 1,000 Americans), accounting for 35 million cases and 11.9 million patient visits a year. Risk factors for development of CRS include allergy, paranasal anatomic anomalies,[13] compromised immune status, and mucociliary dysfunction.

Up to one-third of patients with chronic rhinosinusitis have associated asthma. Although the prevalence of asthma in the elderly is similar to younger adults, the morbidity and mortality are greater in older patients. The death rate due to asthma is 14 times higher for those 65 and older. Symptoms of asthma in the elderly appear to be more consistent, with less mild symptoms and symptom-free periods.[14] There was also a higher prevalence of reported allergy (62 percent), Chronic Obstructive Pulmonary Disease (COPD), and sinusitis in these asthmatics.

Pathophysiology

The inciting stage for the development of CRS typically comes from either viral or allergic inflammation. Allergic rhinitis causes an increase in mucus production and a decrease in mucociliary function and nasal mucous membrane. If ostial obstruction develops, a secondary bacterial infection may ensue. With persistent obstruction, poor aeration results in oxygen resorption in the sinus cavity and carbon dioxide accumulation. Further changes in the sinus membranes occur with mucous membrane fibrosis and ciliary injury resulting from the persistent ostial obstruction, which, if persistent, becomes chronic rhinosinusitis.

Signs and symptoms of CRS can be very subtle and result in chronic postnasal drainage, nasal congestion, and a chronic cough. A dull, aching pain or sinus headache may be present as well. The diagnosis is confirmed with a noncontrast CT of the sinuses.

Cognitive function may also be impaired in the elderly with chronic rhinosinusitis. One study reported subtle changes in cognitive function in elderly patients with CRS compared with age-matched controls using the Mini-Mental State Examination.[15] The researchers found either a decrease in the power of concentration in these patients or an effect on specific cognitive functions. They recommended early medical intervention for neglected CRS to sustain cognitive function in the elderly.
Medical treatment is aimed at reducing intranasal inflammation, promoting drainage, and treating the underlying

bacterial infection. Medications include but are not limited to a combination of nasal steroids, decongestants, long-term antibiotics (three-plus weeks), and mucolytic agents. Oral steroids have been advocated in some cases as well.

Surgical Options

Failure of medical therapy for the symptomatic patient with radiologic evidence of chronic sinus disease is the main indication for sinus surgery. By understanding the pathophysiology, limited mucosal-sparing surgery may be performed using endoscopic techniques to remove disease at the ostiomeatal complex, an area along the lateral nasal wall where there is a confluence of sinuses. This technique, first introduced by Messerklinger in 1978 and known as functional endoscopic sinus surgery (FESS),[16] is relatively safe in the hands of a properly trained otolaryngologist.[17] FESS has become the procedure of choice for the surgical management of chronic rhinosinusitis.

Surgical assessment of the elderly is far more critical than in the younger, healthier patient. The goal of a preoperative assessment is to identify and determine surgical or anesthesia risk factors and to assist in the management of those recognized problems before and after surgery.[18] As with any operation, the benefits must outweigh the risks. Chronic rhinosinusitis is not a life-threatening disease but often significantly affects the quality of life of our patients.

Outcomes

Patients with upper respiratory tract infections have measurable, significant decrements in health-related quality of life.[19] A positive impact on symptoms and quality of life in the majority of patients undergoing functional endoscopic sinus surgery for CRS has been established.[20]

Many published studies have demonstrated improvement in symptom scores and quality of life following functional endoscopic sinus surgery for chronic rhinosinusitis.[21] Most studies report success rates ranging from 76–95 percent in adults.[22] Complication rates also vary from 2–35 percent, and are directly dependent on the experience and expertise of the surgeon.[23] Unfortunately, most studies lack the methodology of the gold standard research protocol: randomized, double-blind placebo control.

In 2001, Lund published an excellent review of 24 ESS studies through 1998 and explained eloquently most of the studies' shortcomings.[24] Fortunately, several studies using outcome measures (level 2 evidence) have confirmed success utilizing FESS. Durr performed a longitudinal study on 51 patients undergoing FESS for recurrent acute rhinosinusitis, chronic rhinosinusitis, and nasal polyposis.[25] Patients were evaluated preoperatively and three months postoperatively with a disease-specific health status questionnaire (Rhinosinusitis Outcome Measure) and a general health status questionnaire (Medical Outcome Study 36-Item Short-Form Health Survey). Their short-

term results showed statistically significant improvement in quality of life after endoscopic sinus surgery in both the disease-specific and the disease-generic health status questionnaires.

Finally, Lieu and Piccirillo reviewed 518 FESS studies through 2001 looking specifically at methodology to determine efficacy of FESS.26 Of those published studies, only 35 met their stringent criteria for further assessment of methodological criteria. Taking the limitations of most of the studies into account, patient improvement following FESS ranged from 68.9 percent (good outcome) to 94 percent (at least 50 percent improvement).

Performing a Medline search revealed only one article in the medical literature specifically addressing endoscopic sinus surgery in the geriatric population. Ramadan and VanMetre retrospectively reviewed their experience with FESS over an 11-year period.27 Of the 568 cases studied, 8.1 percent (46 patients) were older than 65 years of age. In evaluating their results, Ramadan found complication rates similar to the younger adults. However, in those elderly patients undergoing revision sinus surgery (N=11), the complication rate was significantly higher and related to breach of the lamina papyracea with herniation of orbital fat (27 percent versus 8 percent), hemorrhage (18 percent versus 3.4 percent), or periorbital ecchymosis (9 percent versus 1 percent).

A new, validated, 10-question, health-related, quality-of-life instrument (Rhinosinusitis Quality of Life Survey—Rhino Qol) for patients with sinusitis is now available and should make outcome studies easier to perform.[28]

In sum, chronic rhinosinusitis is a common and often debilitating disease that frequently requires surgical management after failure of medical therapy. With the increasing age of our population, more elderly patients will require some form of surgical management for CRS. Clinical experience and outcome measures show high success rates for FESS. Careful attention to co-morbid factors and proper preoperative assessment and planning should allow for successful management of these patients. Future studies are necessary to thoroughly evaluate this patient population.

Quiz

1. Which of the following do not affect the risk of infection in the elderly?

a. immunosenescence
b. sarcopenia
c. nutritional imbalance
d. lifestyle
e. none of the above

2. The medical management of chronic rhinosinusitis does not usually include

a. decongestants
b. nasal steroid sprays
c. antihistamines
d. antibiotics
e. mucolytic agents

3. Evidence for efficacy of FESS in the management of CRS is currently best shown by

a. reviews of retrospective studies.
b. randomized, double-blind, placebo-controlled study.
c. expert opinion.
d. outcome studies.
e. none of the above.

Answers:
1. e
2. c
3. d

References

1. Williams J. The U.S. population: A fact sheet. CRS Report. InEnvironment. 1995; 95-705

2. Ramos-Casals N, Garcia-Carrasco M, Brito M, López-Sgnificance of autoimmune manifestations in the elderly. Lupus. 2003;12:341-355.

3. Ginaldi L, De Martinis M, et al. Immunological changes in the elderly. Aging (Milano). 1999;11:281-286. Also see Ginaldi L, De Martinis M, et al. Cell proliferation and apoptosis in the immune system in the elderly. Immunol Res. 2000;21:31-38; and Ramos-Casals et al., 2003, note 2.

4. See the excellent review of this topic by Ramos-Casals et al., 2003, note 2.

5. Fox R, ed. Immunology and Infection in the Elderly. Edinburgh: Churchill Livingstone; 1984:289.

6. Terpenning M, Bradley S. Why aging leads to increased susceptibility to infection. Geriatrics. 1991;46:77-78.

7. See Terpenning and Bradley, 1991, note 6.

8. Sixty-five Plus in America [Special Studies]. Washington, DC. Government Printing Office; 1999.

9. Chandra R. The relation between immunology, nutrition and disease in elderly people. Age and Ageing. 1990;19:S25-31.

10. Duchateau J, Delepress G, et al. Beneficial effects of oral zinc supplementation on the immune response of old people. Am J Med. 1981;70:1001-1007.

11. Kennes B. Effect of vitamin C supplements on cell-mediated immunity in old people. Gerontology. 1983;29:305-311.

12. Tsukamoto K, Suzuki K, Machida K, et al. Relationship between lifestyle factors and neutrophil functions in the elderly. J Clin Lab Anal. 2002;16:266-272.

13. Caughey R, Jameson M, Gross CW, Han JK. Anatomic risk factors for sinus disease: Fact or fiction. Am J Rhinol. 2005;19:334-339.

14. Diette G, Krishman J, Dominici F, et al. Asthma in older patients: Factors associated with hospitalization. Arch Intern Med. 2002;162:1123-1132.

15. Matsui T, Arai H, Nakajo M, Maruyama M, Ebihara S, Sasaki H, et. al. Role of chronic sinusitis in cognitive functioning in the elderly. J Am Geriatr Soc. 2003;51:1818-1819.

16. Messerklinger W. Endoscopy of the nose. Munich: Urban & Schwarzenberg; 1978:49-50.

17. Kennedy D. Functional endoscopic sinus surgery: Technique. Arch Otolaryngol Head Neck Surg. 1985;111:643-649. Also see Kennedy D. The functional endoscopic approach to inflammatory sinus disease: Current perspectives and technique modifications. Am J Rhinol. 1988;2-3:89-96.

18. Johnson J. Surgical assessment in the elderly. Geriatrics. 1988;43:83-90.

19. Linder J, Singer D. Health-related quality of life of adults with upper respiratory tract infections. J Gen Intern Med. 2003;18:802-807.

20. Damm M, Quante G, Jungehuelsing M, et al. Impact of functional endoscopic sinus surgery on symptoms and quality of life in chronic rhinosinusitis. Laryngoscope. 2002;112:310-315.

21. Lund V, Scadding G. Objective assessment of endoscopic sinus surgery in the management of chronic rhinosinusitis: An update. J Laryngol Otol. 1995;109:693-694.

22. See Levine H. Functional endoscopic sinus surgery: Evaluation, surgery and follow-up of 250 patients. Laryngoscope. 1990;100:79-84; Matthews B, Smith L, Jones R, et al. Endoscopic sinus surgery: Outcome in 155 cases. Otolaryngology Head & Neck Surgery. 1991:104; Rice DH. Endoscopic sinus surgery: Results at two-year follow-up. Otolaryngology Head & Neck Surgery. 1989;99:476-479; Sharp H, Rowe-Jones J, Mackay I. The outcome of endoscopic sinus surgery: Correlation with computerize tomography score and systemic disease. Clin Otolaryngol. 1999;24:39-42; Sobol S, Wright E, Frenkel S. One-year outcome analysis of functional endoscopic sinus surgery for chronic sinusitis. J Otolaryngol. 1998;27:252-257; and Damm et al., 2002, note 20.

23. See Ramadan H, Allen G. Complications of endoscopic sinus surgery in a residency training program. Laryngoscope. 1995;105:376-379; Stankiewicz J. Complications of endoscopic intranasal ethmoidectomy. Laryngoscope. 1987;97:1270-1273; Stankiewicz J. Complications in endoscopic intranasal ethmoidectomy: An update. Laryngoscope. 1989;99:686-690; and Vleming M, Middelweerd R, deVries N. Complications of endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg. 1992;118:617-623.

24. Lund V. Evidence-based surgery in chronic rhinosinusitis. Acta Otolaryngol. 2001;121:5-9.

25. Durr D, Derosiers M. Evidence-based endoscopic sinus surgery. J Otolaryngol. 2003;32:101-106.

26. Lieu J, Piccirillo J. Methodologic assessment of studies on endoscopic sinus surgery. Arch Otolaryngol Head Neck Surg. 2003;129:1230-1235.

27. Ramadan H, VanMetre R. Endoscopic sinus surgery in geriatric population. Am J Rhinol. 2004;18:125-127.

28. Atlas S, Gallagher, P, Wu Y, et al. Development and validation of a new health-related quality of life instrument for patients with sinusitis. Qual Life Res. 2005;14:1375-1386.

Additional Resources

1. Surgery of the Paranasal Sinuses—AAO-HNS Monograph
2. Endoscopic Sinus Surgery—AAO-HNS SIPAC

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Chapter 7

American Academy of Otolaryngology—
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Sleep disorders

The Most Effective Treatments for Snoring and Sleep Apnea

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Obstructive sleep apnea-hypopnea syndrome (OSAHS) is a prevalent disorder characterized by intermittent cessation of airflow during sleep that results in excessive daytime sleepiness. Common presenting symptoms include habitual snoring, witnessed apneas, nighttime gasping or arousals, daytime sleepiness or fatigue, unrefreshing sleep, and morning headaches. The diagnosis of OSAHS is established if the patient has both an apnea-hypnea index (AHI) score ≥ 5 on overnight monitoring, and evidence of excessive daytime sleepiness or fatigue. OSAHS is a prevalent disorder in Western society estimated to affect up to 5 percent of the adult male and 3 percent of the adult female population.[1] The focus of this review is the growing body of evidence that suggests that the burden of OSAHS is especially high in older adults.

Epidemiology of OSAHS in Older Adults

Several large population-based cohort studies have demonstrated that undiagnosed sleep apnea is higher in the ≥ 60-year-old age group compared with younger age groups.[2] It is estimated that the rate of AHI ≥ 5 is 50-60 percent in older men and 30-40 percent in older women, which is three times greater than the general adult population.[3] Menopausal status appears to be a major determinant of the rate of OSAHS in women. In the large population-based Wisconsin Sleep Cohort Study, postmenopausal women were found to be 3.5 times more likely than premenopausal women to have AHI scores ≥ 15.[4] A number of theories have been proposed to explain the increased prevalence of OSAHS in older age groups, including ongoing loss of neuromuscular tone,[5] decreased hormone levels,[6] and changes in the ratio of fat to lean body mass.[7]

Although the prevalence of OSHAS appears to increase steadily with age, the overall prevalence of the disorder appears to level off after the age of 65 years.[8] The reason for the plateau after age 65 is not clear but may be explained in several ways: (1) the incidence of new cases of OSAHS decreases after age 65; (2) the mortality rate of OSAHS cases increases after age 65; or (3) OSAHS remits at older age. Because there is little evidence to support death caused directly by OSAHS or spontaneous remission of OSAHS, a reduction in incidence after age 65 is the currently favored explanation;[9] however, further investigation is needed to clarify this issue.

Sleep Patterns in Older Adults

Changes in sleep duration and architecture occur as a normal part of the aging process.[10] The duration and need for sleep appears to lessen with age. The average 70-year-old sleeps only 6 hours per night but may make up for loss of nocturnal sleep with daytime naps of 1 to 2 hours.[11] In addition, many older adults have increased difficulty falling to sleep once in bed (increased sleep latency), and have greater difficulty staying asleep (decreased sleep efficiency).[12] The number of co-morbid medical conditions that have an impact on sleep increases with age and includes depression, arthritis, gastroesophageal reflux, prostate hypertrophy, and renal and pulmonary disorders.[13] In addition, older adults are more likely to take medications such as diuretics that cause nocturia. As a result, up to 40 percent of older individuals complain of sleep disturbance and undesired daytime fatigue.[14]

The challenge for the physician presented with complaints of sleep disturbance in older individuals is to determine the degree to which symptoms are related to normal age-related changes in sleep pattern versus an underlying medical disorder or a primary sleep disorder. A thorough review of the patient's past medical history, medications, and alcohol and stimulant (caffeine, nicotine) use is required. Improving the management of co-morbid conditions (for example, reflux, arthritic pain, or prostate hypertrophy) will often result in sleep improvement. A thorough sleep history (table 1) helps identify patients at risk of the primary sleep disorders seen most commonly

in older adults: OSAHS, restless leg syndrome, insomnia, and sleep maintenance insomnia. It is important to note that sleep maintenance insomnia, which is characterized by early morning awakenings, is highly associated with either alcohol abuse or depression in older individuals.[15] Patients suspected of a primary sleep disorder should undergo a full-night polysomnography in order to establish the presence and severity of a sleep disorder. The diagnosis of OSAHS can be established in patients with daytime sleepiness or fatigue who are found to have an AHI ≥ 5 on overnight polysomnography.

Table 1: Elements of sleep history

Time in bed
Time of sleep
Number of awakenings
Rising time
Presence of snoring, witnessed apneas, gasps, or choking spells
Presence of involuntary leg motion or jerking
Level of daytime sleepiness or fatigue (Epworth Scale Score)
Time, duration, and number of daytime naps

Clinical Sequelae of OSAHS

Untreated OSAHS has been associated with reduced quality of life as well as a number of serious health conditions.

Reduced quality of life. Daytime sleepiness caused by OSAHS often results in decreased energy, loss of concentration,

poor job performance, and reduced social interaction. In addition, the loud snoring that often accompanies OSAHS may result in poor sleep for the sufferer's bed partner. An AHI ≥ 5 has been associated with concentration difficulty but not memory tasks on self-assessment exams.[16] OSAHS patients demonstrate significant improvement in daytime sleepiness,[17] and in numerous quality-of-life parameters including physical functioning, social functioning, vitality, and general health perception after treatment.[18] The relationship between sleepiness and level of AHI is not well defined, especially in older populations, with many people with AHI ≥ 5 reporting minimal or no daytime sleepiness on self-reported examination.[19]

Cardiovascular morbidity and mortality. Evidence supports an increase in cardiovascular mortality five years after diagnosis in untreated patients with severe OSAHS patients compared with treated patients.[20] Another study found that men under age 60 with snoring and excessive daytime sleepiness were twice as likely to die over a 10-year study period compared with subjects without snoring or snoring without sleepiness.[21] Two large prospective studies suggest that untreated, loud, habitual snorers have a 30-40 percent greater risk of myocardial infarction or stroke relative to nonsnorers.[22] In contrast, a study by Jennum et al., which contained greater numbers of older individuals (age 54-74 years), failed to demonstrate a significant association between untreated snoring and cardiovascular morbidity or mortality.[23]

Therefore, it has been hypothesized that the relationship between OSAHS and cardiovascular morbidity and mortality may be stronger in younger OSAHS patients compared with older patients.

Hypertension. It has been hypothesized that systemic arterial hypertension is the cause of the increased cardiovascular morbidity and mortality observed in patients with sleep-disordered breathing.[24] Laboratory evidence has demonstrated that sustained arterial hypertension can be induced in animal models subjected to intermittent airway occlusion during sleep.[25] The current epidemiological evidence shows a strong and consistent association between OSAHS and hypertension. Four large population-based cross-sectional studies found that the odds of hypertension were 1.4 to 2.5 times greater in patients with an AHI ≥ 5 compared with controls.[26]
A prospective analysis of the Wisconsin Sleep Cohort study found that even minimal elevation in AHI scores was associated with a 42 percent increased risk of developing hypertension over a four-year period.[27] Currently it is unclear the degree to which blood pressure can be lowered by treatment of OSAHS with CPAP or other methods.[28]

Motor vehicle accidents. Epidemiological and laboratory evidence both suggest that patients with OSAHS are at greater risk for motor vehicle accidents (MVA). Patients with an AHI ≥ 15 were 7.3 times more likely to have had multiple MVAs in the five years before their study, compared with those with lower or no apnea.[29] Hospitalized MVA victims were found to be 6.3 times more likely to have an AHI ≥ 5 than community controls.[30]

In addition, patients with severe OSAHS performed significantly worse on a driving simulator than controls without OSAHS.[31]

All patients ≥ 65 years should be screened by history and physical examination for sleep-disordered breathing given the extensive prevalence of at least mild apnea (AHI ≤ 15) in this age group and the severe potential health and quality-of-life consequences of undiagnosed sleep apnea. The evidence supports treatment of OSAHS in individuals with the following findings:

- AHI ≥ 5 with excessive daytime sleepiness
- AHI ≥ 5 with cardiovascular co-morbidities
- AHI ≥ 15 with or without excessive daytime sleepiness

Patients with an AHI between 5 and 15 who do not have excessive daytime sleepiness are likely to be at low risk for cardiovascular sequelae and unlikely to be compliant with therapy, because they have no self-perceived sleepiness. Treatment selection depends largely on the level of sleep apnea, presence of medical co-morbidities, presence of anatomical deformities, and patient preference.

Quiz

1. Common primary sleep disorders in older adults include all of the following EXCEPT:

    a. Obstructive sleep apnea-hypopnea syndrome
    b.. Cataplexy
    c. Insomnia
    d. Sleep maintenance insomnia
    e. Restless leg syndrome

2. Changes in sleep pattern with age include which of the following:

    a. Increased sleep requirement
    b. Improved sleep efficiency
    c. Fewer nocturnal arousals
    d. Higher rates of insomnia
    e. Less need for napping

3. Which of the following statements is supported by current evidence:

    a. OSAHS is a major cause of systemic hypertension.
    b. OSAHS is a major cause of memory loss in older individuals .
    c. Treatment of OSAHS significantly improves self-perceived quality of life.
    d. The association between cardiovascular mortality and OSAHS is stronger in older individuals.
    e. The treatment of OSAHS significantly lowers systemic high blood pressures.

Answers:

1.
2.
3.

References

1. Davies RJO, Stradling JR. The epidemiology of sleep apnea. Thorax. 1996;51:S65-S70.

2. See the following articles: Bixler E, Vgontzas A, ten Have T, Tyson K, Kales A. Effects of age on sleep apnea in men. Am J Respir Crit Care Med. 1998;157:144-148; Bixler E, Vgontzas A, Lin H, Ten Have T, Rein J, Vela-Bueno A, Kales A. Prevalence of sleep-disordered breathing in women. Am J Respir Crit Care Med. 2001;163:608-613; Duran J, Esnaola S, Rubio R, Iztueta A. Obstructive sleep apnea-hypopnea and related clinical features in a population-based sample of subjects aged 30 to 70 yr. Am J Respir Crit Care Med. 2001;163:685-689; and Duran CJ. Prevalence of obstructive sleep apnea-hypopnea and related clinical features in the elderly: A population-based study in the general population aged 71-100. World Conference 2001 Sleep Odyssey, October 21-26 2001, Montevideo, Uruguay.

3. Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea. Am J Respir Crit Care Med. 2002;165:1217-1239.

4. Young T, Finn L, Austin D, Peterson A. Menopausal status and sleep-disordered breathing in the Wisconsin Sleep Cohort Study. Am J Respir Crit Care Med. 2003;167:1181-1185.

5. Klawe JJ, Tafil-Klawe M. Age-related response of the genioglossus muscle EMG-activity to hypoxia in humans. J Physiol Pharmacol. 2003;54:14-19.

6. See Young, Finn, Peterson, 2003, note 4.

7. See Young, Peppard, Gottlieb, 2002, note 3.

8. Young T, Shahar E, Nieto FJ, et al. Predictors of sleep-disordered breathing in community dwelling adults: The Sleep Heart Health Study. Arch Intern Med. 2002;162:893-900. Also see Bixler et al., 1998, and Bixler et al., 2001, note 2.

9. See Young, Peppard, Gottlieb, 2002, note 3.

10. Piani A, Brontini S, Dolso P, et al. Sleep disturbances in elderly: A subjective evaluation over 65. Arch Gerontol Geriatr Suppl. 2004;9:325-331.

11. Cauter EV, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep in relationship with growth hormone and cortisone levels in healthy men. JAMA. 2000;284:861-868.

12. Boselli M, Parrino L, Smeireri A, Terzano MG. Effect of age on EEG arousals in normal sleep. Sleep. 1998;21:351-357.

13. Foley D, Ancoli-Israel S, Britz P, Walsh J. Sleep disturbances and chronic disease in older adults: Results of the 2003 National Sleep Foundation Sleep in America Survey. J Psychosom Res. 2004;56:497-502.

14. Bliwise DL. Sleep in normal aging and dementia. Sleep. 1993;16:40-81.

15. Barthlen GM. Obstructive sleep apnea syndrome, restless legs syndrome, and insomnia in geriatric patients. Geriatrics. 2002;57:34-39.

16. Jennum P, Sjol A. Self-assessed cognitive function in snorers and sleep-apneics: An epidemiological study of 1504 females and males age 30-60 years. The Dan-MONICA II Study. Eur Neurol. 1994;34:204-208.

17. Ballester E, Badia JR, Hernandez L, et al. Evidence of the effectiveness of continuous positive airway pressure in the treatment of sleep apnea/ hypopnea syndrome. Am J Respir Crit Care Med. 1990;159:495-501. Also see Engelman H, Kingshott RN, Wraith PK, et al. Randomized-placebo controlled crossover trial of continuous positive airway pressure for mil sleep apnea/ hypopnea syndrome. Am J Respir Crit Care Med. 1999;159:461-467.

18. Pichel F, Zamarron C, Magan F, et al. Health-related quality of life in patients with obstructive sleep apnea: Effects of long-term positive airway pressure treatment. Respir Med. 2004;98:968-976.

19. See Young, Peppard, Gottlieb, 2002, note 3.

20. He J, Kryger MH, Zorick FJ, et al. Mortality and apnea index in obstructive sleep apnea: Experience in 385 male patients. Chest. 1988;94:9-14. Also see Partinen M, Jamieson A, Guilleminault C. Long-term outcome for obstructive sleep apnea syndrome patients: Mortality. Chest. 1988;94:1200-1204.

21. Lindberg E, Christer J, Svardsudd K, et al. Increased mortality among sleepy snorers: A prospective population based study. Thorax. 1998;53:631-637.

22. Hu FB, Willett WC, Manson JE, et al. Snoring and the risk of cardiovascular disease in women. J Am Coll Cardiol. 2000;35:308-313. Also see Koskenvuo M, Kaprio J, Telakivi T, et al. Snoring as a risk factor for ischemic heart disease and stroke in men. Br Med J. 1987;294:16-19.

23. Jennum P, Hein HO, Suadicani P, Gyntelberg F. Risk of ischemic heart disease in self-reported snorers. Chest. 1995;108:138-142.

24. Lavie P, Herer P, Peled R, et al. Mortality in sleep apnea patients: A multivariate analysis of risk factors. Sleep. 1995;18:149-157.

25. Tilkian AG, Guilleminault C, Schroeder JS, et al. Hemodynamics in sleep-induced apnea: Studies during wakefulness and in sleep. Ann Intern Med. 1976;85:714-719.

26. See Nieto FJ, Young TB, Lind BK, et al. Association of sleep-disordered breathing, Sleep apnea, and hypertension in a large community-based study. JAMA. 2000;283:1829-1836; Bixler EO, Vgontzas AN, Lin HM, et al. Association of hypertension and sleep-disordered breathing. Arch Intern Med. 2000;160:2289-2295; Young TE, Peppard P, Palta M, et al. Population-based study of sleep-disordered breathing as a risk factor for hypertension. Arch Intern Med. 1997;157:1746-1752; and Duran et al., 2001, note 2.

27. Peppard PE, Young TE, Palta M, et al. Prospective study of the association between sleep-disordered breathing and hypertension. N Engl J Med. 2000;342:1378-1384. See also Peppard PE, Young T. Sleep-disordered breathing and hypertension: Reply. N Engl J Med. 2000;343:967.

28. See Young, Peppard, Gottlieb, 2002, note 3.

29. Young TB, Blustein J, Finn L, Palta M. Sleep-disordered breathing and motor vehicle accidents in a population-based sample of employed adults. Sleep. 1997;20:608-613.

30. See Davies and Stradling, 1996, note 1; Young, Peppard, Gottlieb, 2002, note 3; and Young et al., 1997, note 29. Also see Teran-Santos J, Jimenez-Gomez A, Cordero-Guevara J. The association between sleep apnea and the risk of traffic accidents. Cooperative Group Burgos-Santander. N Engl J Med. 1999;340:847-851.

31. Findley FJ, Fabrizio MJ, Knigth H, et al. Driving simulator performance in patients with sleep apnea. Am Rev Respir Dis. 1989;140:529-530.

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Chapter 8

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Geriatric Polypharmacy in Otolaryngology

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The term "polypharmacy" refers to "the administration of many drugs." In today's world, polypharmacy probably applies to drug use within most of the population. Considering over-the-counter pain relievers, sinus and cold remedies, vitamins, alternative medications, and "magic herbs," most people consume some form of medication on a daily basis. Because the vast majority of the population is seen by a physician at least once a year, it is easy to see how the number of medications taken either by prescription or otherwise can rapidly become a meaningful statistic, thus justifying the term polypharmacy.

Recent data demonstrate that the elderly (population aged 65 years or more) represent approximately 12 percent of the U.S. population. However, this same 12 percent of the population accounts for approximately 32 percent of annual prescriptions. The average elderly person uses two to six prescription drugs and one to more than three over-the-counter medications on a daily basis.[1]
Looking into the future, it should be noted that during the twentieth century the number of people in the United States less than 65 years of age tripled. During the same period, the number of people over 65 increased 11 times. Current population projection studies estimate that by the year 2050 the elderly population will have more than doubled. At the midpoint of the twenty-first century, one in five Americans will be considered elderly.[2]
When one considers that up to one-third of the patients seen by the average otolaryngologist practicing in the United States are aged 65 or older, it is readily understandable why it is necessary to have a good foundation in geriatric otolaryngologic polypharmacy.

Common Pathologies in Geriatric Otolaryngology

It is important to realize that many otolaryngologic disorders can be seen in almost any age group. However, age predicts to a varying degree the prevalence of disease in certain chronologic populations. It is with this in mind that the following list of geriatric otolaryngologic disorders is presented. The disorders presented below reflect the most common geriatric otolaryngologic manifestations.

Otologic

Hearing Loss

- Sensory
- Conductive

a. cerumen impaction
b. serous otitis
c. otosclerosis

• Mixed

Balance Disorders

• Benign Paroxysmal Positional Vertigo
• Labyrinthitis
• Meniere's
• Multiple Medications

Rhinologic

• Rhinitis
• Epistaxis
• Nasal Obstruction
• Olfactory Dysfunction
• Sinusitis

Oropharyngeal

• Dysphagia
• Aspiration
• Xerostomia
• Burning Mouth Syndrome
• Laryngopharyngeal Reflux

Laryngeal

• Voice Disturbance

Other

• Thyroid
• Head And Neck Cancer
• Cosmetic

Brief Description of Otolaryngologic Geriatric Disorders and Possible Treatments

The following description of the above disorders and their treatments is not meant to serve as a definitive reference for diagnosis and treatment of geriatric otolaryngologic disease. Rather it is simply a brief outline to serve as a foundation to better understand polypharmacy in the elderly otolaryngologic patient.

Otologic

Hearing loss is the most common otolaryngologic manifestation in the geriatric population.[3] Approximately 60 percent of the population in the United States, aged 65 and older, has at least a 25dB compromise in hearing. Hearing in this patient group may be divided into sensory, conductive, and mixed hearing loss. Sensory hearing loss, the most common type of hearing loss in the elderly patient, is almost universally treated with amplification or a hearing aid. Medications associated with this might include topical steroid creams in the event of sensitivity to hearing aid ear molds. Conductive hearing loss is typically caused by cerumen impaction or middle ear fluid. Cerumen impaction is the most common and most treatable cause of conductive hearing loss in the elderly. A recent study of 29 elderly patients in a skilled nursing facility demonstrated that 19 patients (65.5 percent) had at least one ear occluded by 50 percent or more with cerumen. Hearing improved in 80 percent of the ears after cerumen removal.[4] A variety of over-the-counter cerumenolytic agents are readily available to treat this problem.

Serous otitis is most commonly a result of eustachian tube dysfunction. A variety of possible causes include infection, allergy, and mechanical obstruction. Medications most often used in the treatment of these disorders include antibiotics, antihistamines, decongestants (oral and topical), nasal steroid sprays, and oral steroids. Otosclerosis is most commonly treated with surgery or amplification.

Balance disorders often result in seriously incapacitating injuries. It has been estimated that in a one-year period, 30-50 percent of people 65 and older fall at least once. That number increases to 50 percent after age 80.[5] In 1 percent of this age group, falls result in hip fractures. An overall 5 percent fracture rate is seen. Approximately half of the hip fractures never regain normal function. Balance disorders have many etiologies including cardiovascular disease, neurologic disease, multiple medications, and, of course, vestibular pathology. Vestibular disorders include benign paroxysmal positional vertigo (BPPV) and Meniere's. BPPV is treated with vestibular exercises, canalith repositioning, and numerous medications including meclizine and benzodiazepines for symptomatic relief. Meniere's may be treated with a low-salt diet, diuretics, and vestibular suppressants.

Rhinologic

Many forms of rhinitis have been described. Allergic rhinitis and nonallergic rhinitis have been treated with a combination of medications including antihistamines, decongestants, nasal steroid sprays, oral steroids, and nasal saline and nasal antihistamine sprays. Vasomotor rhinitis has been treated with ipratroprium, nasal steroid sprays, and antihistamine sprays. Epistaxis may be treated with moisturizing agents (saline nasal sprays), antibiotics, and lubricants. Many elderly people with nosebleeds are taking anticoagulant medication, which must be considered in the overall treatment plan. Nasal obstruction results from multiple causes and therefore has multiple treatments. Obstruction from trauma is most often addressed with surgical intervention. Allergic and vasomotor rhinitis may result in obstruction. Their treatments have been presented. Rhinitis medicamentosa (rebound rhinitis) is treated with discontinuation of the offending topical agent, application of a topical nasal steroid spray, and a short tapering dose of oral steroids.

Olfactory function has been shown to decline in people more than 60 years old.[6] More than 50 percent of people over age 80 have dysfunctions of smell.[7] Because the sense of smell is probably the least understood of all the senses, there are not a lot of treatment options. Treatment is according to underlying cause outlined above for obstructive and other inflammatory disorders. Sinusitis is relatively common in the aged population and frequently is overlooked because symptoms are often more subtle than in younger people. Treatment of sinusitis is typically accomplished with varying combinations of antibiotics, decongestants, mucolytics, hydration irrigations, and occasionally antihistamines.

Oropharyngeal

Dysphagia may occur in 10–30 percent of elderly patients.[8] The list of potential causes of dysphagia in the geriatric population is long. Systemic causes include musculoskeletal, connective tissue, autoimmune, cardiovascular, neurologic, and general disorders. Medications that can cause dysphagia include anticholinergics, antidepressants, antihypertensives, diuretics, and phenothiazines. Other possible etiologies of geriatric dysphagia relate to local disorders of the oropharynx, hypopharynx, and esophagus. Treatments of these disorders may include multiple medications, which in turn can cause further problems.

Aspiration and in particular silent aspiration in the elderly is a frequent cause of pneumonia. Pneumonia is treated with antibiotics, which may also contribute to polypharmacy in the elderly. Xerostomia is a common symptom in up to 20 percent of the elderly population. Again, the administration of multiple medications in itself is a common cause of this problem in the elderly. Other causes of xerostomia include diabetes, psychotropic drugs, and radiation therapy.
Burning mouth syndrome has not been definitively linked to any specific etiology. Varying degrees of treatment success have been realized employing symptomatic treatment with low dosages of tricyclic antidepressants, benzodiazepines, and anticonvulsants.[9] Laryngopharyngeal reflux is seen in all age groups but certainly is recognized in the elderly. It is treated primarily with diet, proton pump inhibitors, and H2 blockers.

Laryngeal

Vocal dysfunction is seen in approximately 12 percent of the geriatric population.[10] Speech therapy is the mainstay of treatment although other underlying causes such as essential tremor, Parkinson's disease, stroke, myasthenia gravis, and thyroid disease may introduce new medications.

Other

Thyroid disease is usually treated with synthroid or other similar medications. Head and neck cancer may involve numerous medications. A number of chemotherapeutic agents, including cisplatin, bleomycin, vincristine, vinblastine, and nitrogen mustard, may produce ototoxicity. Finally, let us not forget that with the appearance of aging comes the desire for the disappearance of aging. Botox, fillers, and a plethora of cosmetic antiaging drugs have found their way into the market in recent years. The geriatric population accounts for some of the most avid users of these drugs.

Correctly Medicating the Geriatric Population in a Polypharmacy World

Based on the presentation above of the more common geriatric otolaryngologic disorders and their suggested possible treatments, the otolaryngologist should be aware of the several medications that can be used for elderly patients, and of the possible problems that can be caused by the harmful interaction of some of these particular drugs. A partial list of medications available to the otolaryngologist is provided in table 1.

Table 1.
Partial List of Medications in the Otolaryngologist's Arsenal

Topical Steroid Creams	Antibiotics
Betamethasone	Amimnoglycosides
Mometasone	Neomycin
Fluocinonide	Tobramycin
Triamcinolone	Gentamycin
Fluocinolone	

Macrolides	Cephalosporins
Azithromycin	Cefazolin
Clarithromycin	Cephalexin
Erythromycin	Cefaclor
	Cefuroxime
	Cefprozil
	Cefoxitin

Penicillins	Quinolones
Amoxicillin	Levofloxacin
Amoxicillin/Clavulanate	Moxifloxacin
	Ciprofloxacin

Other Antimicrobials	Cerumenolytics
Clindamycin	Various OTC Preparations
Metronidazole	

Sedating	Decongestants
Hydroxyzine	Pseudoephedrine
Diphenhtdramine	Ephedrine
Cetirizine	Guaifenesin

Antihistamines	Nasal Sprays
Nonsedating	Steroids
Fexofenadine	Beclomethasone
Desloratadine	Fluticasone
Loratadine	Triamcinolone
	Mometasone
	Budesonide

Oral Steroids	**Other**
Prednisone	Oxymetazaline
Methylprednisolone	Azelastine
	Ipratropium

Diuretics	Anti-Vertiginous
Hydrochlorothiazide Dyazide	Meclizine Benzodiazepines
Gastrointestinal	**Chemotherapeutics**
Proton Pump Inhibitors Rabeprazole Esomeprazollansoprazole Omeprazole Pantoprazole **H2 Blockers** Nizatidine Cimetidine Famotidine Ranitidine	Cisplatin Bleomycin Vincristine Vinblastine Nitrogen Mustard
	Cosmetic Applications
	Botox Dermal Fillers

Potentially Problematic Drug Group Interactions

Although it is not within the scope of this essay to present all possible drug interactions, the more common drug group interactions are discussed below.

Aminoglycosides and Cephalosporins. Nephrotoxicity, or the quality or state of being toxic to kidney cells, may result from the combined use of aminoglycosides and cephalosporins.[11] The mechanism of action is unknown. Predisposing factors include older age, preexisting renal compromise, and large doses of either drug. The fact that elderly patients are considered to be at increased risk in

this group is a reflection of the large number of patients in this group who are taking multiple medications, have multiple medical disorders, and have preexisting renal impairment. Renal functions should be closely monitored in this group if alternative regimens are not possible.

Quinolones and Corticosteroids. The quinolone group of antibiotics is known to possibly cause arthropathy.[12] Corticosteroids increase the risk of tendon rupture and or tendonitis.[13] The exact mechanism of action has not been demonstrated. However, the geriatric population is known to be at increased risk for adverse effects from these drug interactions. These interactions are easily and often overlooked. Large numbers of geriatric patients already have arthritis and other arthropathies. Therefore this drug interaction can readily be mistakenly attributed to other ongoing disease or simply explained by the expectation of joint pain and pathology in older patients. The combined use of these medications is not unlikely to be seen in the geriatric patient population. It should be kept in mind that the interactions may be generated by topical nasal corticosteroids as well as oral IV and IM steroids. The prescribing practitioner is therefore urged to keep this interaction in mind and use this combination of drugs only when other combinations are not applicable.

Levofloxacin and QT Prolonging Agents. The QT interval is the time from electrocardiogram Q wave to the end of the T wave corresponding to electrical systole. The concurrent use of levofloxacin and drugs that prolong the QT interval may result in synergistic or additive effects on the

QT interval.[14] This may result in life-threatening cardiac arrhythmias, including torsades de pointes. The risk of QT prolongation increases with age. It can therefore again be seen that the elderly are particularly at risk. Because the incidence of cardiac arrhythmias tends to increase with age, it is again an easy mistake to overlook the drug interaction of these life-threatening conditions and ascribe arrhythmias to normal events of aging. It is important to keep this in mind when prescribing these drugs in the elderly.

Importance of Understanding Pharmacokinetics and Aging

It could be concluded that harmful drug interactions or effects are predictable by virtue of the known pharmacology of the drugs and that they are therefore avoidable. But in order to properly understand and effectively work with polypharmacy in the geriatric population, it is essential to establish a basic knowledge of pharmacokinetics and aging. Four physiologic events can give a firm foundation to achieving this goal. They are (1) absorption, (2) distribution, (3) metabolism, and (4) renal excretion.

Absorption in the elderly plays only a minor a role in prescribing patterns. Age-related gastrointestinal and skin changes surprisingly do not usually significantly alter absorption patterns with most medications. An error that should be avoided is to alter dosing based solely on the idea of decreasing absorption rates in the geriatric patient. Distribution of medications often changes with aging.

Aging usually results in a decrease in lean body mass and total body weight. This in turn results in an increase in the overall percentage of body fat. This is important to consider because of the increase in volume of distribution for lipophillic medications such as sedatives that penetrate the central nervous system.

Metabolism is variable in the elderly as it is in other age groups. Even though liver function tests do not demonstrate large changes with aging, there is a degree of general decline in metabolic capacity. This results from decreased liver mass and hepatic blood flow. This must be kept in mind when prescribing for the elderly and it is suggested that prescribing guidelines be checked regularly.

Renal excretion is a well-established parameter in prescribing methods. Age-related decrease in renal blood flow and glomerular filtration rate are well recognized. Regular precautions in this area are encouraged.

Necessity of Recognizing Inappropriate Medications for the Elderly

Although careful scrutiny should be employed when prescribing all medications to the geriatric population, it is also important for physicians to know that certain medications are potentially dangerous as stand-alones. In 1991, Mark Beers headed a group of investigators at the University of California, Los Angeles, in formulating a list of criteria for determining the appropriate use of medica-

tions in the elderly living in nursing homes. He updated the list in 1997.[15] The list includes 28 medications or classes of medications that were considered inappropriate for use in the elderly. Fourteen of the 28 medications were considered to have potentially severe adverse reactions when used in the geriatric population. This list was updated by Fick et al. in 2003.[16] Table 2 (below) lists the drugs presented by Fick et al. in their update.

Potentially Inappropriate Medications for the Elderly According to the Revised Beers Criteria

For detailed information on herbs, supplements, or generic or brand name medications, reference MedlinePlus (http://www.nlm.nih.gov/medlineplus/druginformation.html), a service of the U.S. National Library of Medicine and the National Institutes of Health.

CLICKABLE

A

- alprazolam (Xanax)
- amiodarone (Cordarone)
- amitriptyline (Elavil)
- amphetamines
- anorexic agents

B

- barbiturates
- belladonna alkaloids (Donnatal)
- bisacodyl (Dulcolax)

C

- carisoprodol (Soma)
- cascara sagrada
- chlordiazepoxide (Librium, Mitran)
- chlordiazepoxide-amitriptyline (Limbitrol)
- chlorpheniramine (Chlor-Trimeton)
- chlorpropamide (Diabinese)
- chlorzoxazone (Paraflex)
- cimetidine (Tagamet)
- clidinium-chlordiazepoxide (Librax)
- clonidine (Catapres)
- clorazepate (Tranxene)
- cyclandelate (Cyclospasmol)
- cyclobenzaprine (Flexeril)
- cyproheptadine (Periactin)

D

- dessicated thyroid
- dexchlorpheniramine (Polaramine)
- diazepam (Valium)
- dicyclomine (Bentyl)
- digoxin (Lanoxin)
- diphenhydramine (Benadryl)
- dipyridamole (Persantine)
- disopyramide (Norpace, Norpace CR)
- doxazosin (Cardura)
- doxepin (Sinequan)

E

- ergot mesyloids (Hydergine)
- estrogens
- ethacrynic acid (Edecrin)

F

- ferrous sulfate (iron)
- fluoxetine (Prozac)
- flurazepam (Dalmane)

G

- guanadrel (Hylorel)
- guanethidine (Ismelin)

H

- halazepam (Paxipam)
- hydroxyzine (Vistaril, Atarax)
- hyoscyamine (Levsin, Levsinex)
- hioridazine (Mellaril)

I

- indomethacin (Indocin, Indocin SR)
- isoxsuprine (Vasodilan)

K

- ketorolac (Toradol)

L

- lorazepam (Ativan)

M

- meprobamate (Miltown, Equanil)
- mesoridazine (Serintil)
- metaxalone (Skelaxin)
- methocarbamol (Robaxin)
- methyldopa (Aldomet)
- methyldopa-hydrochlorothiazide (Aldoril)
- methyltestosterone (Android, Virilon, Testrad)
- mineral oil

N

- naproxen (Naprosyn, Avaprox, Aleve)
- neoloid
- nifedipine (Procardia, Adalat)
- nitrofurantoin (Microdantin)

O

- orphenadrine (Norflex)
- oxaprozin (Daypro)
- oxazepam (Serax)
- oxybutynin (Ditropan)

P

- pentazocine (Talwin)
- perphenazine-amitriptyline (Triavil)
- piroxicam (Feldene)
- promethazine (Phenergan)
- propantheline (Pro-Banthine)
- propoxyphene (Darvon) and combination products

Q

- quazepam (Doral)

R

- reserpine (Serpalan, Serpasil)

T

- temazepam (Restoril)
- thioridazine (Mellaril)
- ticlopidine (Ticlid)
- triazolam (Halcion)
- trimethobenzamide (Tigan)
- tripelennamine

In a study completed at Duke University, it was determined that, in a one-year period, 20 percent of elderly citizens whose benefits were processed by a large pharmaceutical benefits manager filled a prescription for at least one medication classified in the Beers list as a "drug of concern."[17] Five drugs from this list have particular relevance in the otolaryngologic geriatric patient pool. They are alprazolam, amitriptyline, diazepam, diphenhydramine, and propoxyphene.

Alprazolam and diazepam may result in side effects including drowsiness, light-headedness, fatigue, dizziness, irritability, and confusion. These are all symptoms that can mistakenly be attributable to "old age." Therefore treatment results can be easily misinterpreted and can result in dangerous consequences. Amitriptyline is a frequently employed mood elevator. Like alprazolam and diazepam it too may result in confusing and misleading symptoms, especially in the elderly. These symptoms include slow or difficult speech, dizziness or faintness, crushing chest pain, seizures, and visual and auditory hallucinations. Diphenhydramine should be generally avoided in the elderly. It commonly results in dry mouth, confusion, urinary retention, and constipation. It is found frequently in over-the-counter preparations for sleep, upper respiratory infections, and allergy. Propoxyphene should probably not be used at all in older adults. Elderly people are especially sensitive to the effects of narcotic analgesics. This may increase the chance of side effects, especially breathing problems, during treatment.

The Problem of Adverse Drug Reactions and the Elderly in a Polypharmacy World

Polypharmacy in today's world is becoming the standard as opposed to the exception. This is seen much more in the elderly population than in any other group of individuals. A number of facts contribute to this statistic. It is well accepted that with increased age comes increased potential for disease. In addition, the complexity and severity of disease rises with age. The number of diseases seen in the geriatric population on an individual basis is also recognized to be overall much greater than in the general population. Finally it can be readily seen that all of the above facts are amplified by recognition that the elderly population is living longer with the progression of medical knowledge and skills. Based on all of the above it is easy to understand the reasons for the marked increase in drug use in the elderly.[18]

The potential for serious problems arises with the increasing number of drugs used in any given individual patient. These problems are referred to as adverse drug reactions (ADRs). A linear relationship has been well established between the number of drugs taken and the increased potential for ADRs.[19] It is alarming to realize that at least 80 percent of ADRs resulting in hospital admissions are classified as type A (dose-related) in nature.[20] ADRs are seen much more often in the elderly patient for multiple reasons. With increasing age comes decreasing natural defense mechanisms leading to greater susceptibility to ADRs. In addition, individual drug toxicities are sometimes magnified when combined with other medications. Multiple medications used in combination in a single patient can produce ADRs not seen by the use of these same medications used individually.

Adverse drug reactions and adverse drug interactions are a common cause for elderly patients to be admitted to the hospital.[21] While in the hospital, adverse drug reactions are seen more commonly in geriatric patients and may contribute to morbidity and death.[22] Lazarou et al. demonstrated that even after eliminating errors in noncompliance, drug abuse, drug administration, overdose, and therapeutic failures, the overall incidence of serious ADRs of the general hospitalized patient population in the United States was 6.7 percent.[23] It was further concluded that the incidence of fatal ADRs was 0.32 percent in patients from 39 prospective studies included in these authors' meta-analysis. ADRs are therefore likely to be between the fourth and sixth most common cause of death in the United States. ADRs in Europe are more prevalent than in the United States. Wiffen et al. demonstrated the incidence of ADRs in Europe to be two times those in the United States before 1985. In studies carried out more recently, the ADR rates for both Europe and the United States in the geriatric environment were 20 percent greater than in the pre-1985 studies.[24]

In sum, polypharmacy is a growing concern throughout the world, especially as it concerns the elderly. Prevention of complications of polypharmacy in the geriatric population may be best accomplished by recognition and compliance with prescribing guidelines specifically outlined

for older patients. Failure to recognize the differences in older adults has disastrous potential for error. Older adults should only be prescribed medications that are absolutely necessary. Medication lists in the elderly are frequently a compilation of multiple physicians. These lists should be reviewed often and unnecessary medications should be discontinued. It is extremely important, when diagnosing and treating the elderly, to recognize that drug reactions may result in effects that mimic the conventional concept of "growing old." These effects include but are not limited to loss of balance, drowsiness, incontinence, dizziness, falls, confusion, depression, nervousness, fatigue, malaise, and insomnia. Adverse drug reactions such as dizziness from antihypertensives must not be treated with other drugs such as meclizine. Prescribing errors may also be minimized by prescribing medications that are taken only once a day and limiting the use of PRN or as-needed medications.

Quiz

1. People over age 65 living in the United States comprise approximately 32 percent of the population. They account for what percentage of the annual prescriptions written in the United States?
   a) 18%
   b) 32%
   c) 43%
   d) 52%

2. Dysphagia occurs in up to 30 percent of elderly patients. It is important to realize that this may be induced by medications including:
   a) Anticholinergics
   b) Antidepressants
   c) Antihypertensives/diuretics
   d) Phenothiazines
   e) All of the above

3. Arthritis and other arthropathies are commonly seen in the geriatric population. It is therefore a very easy error to overlook the iatrogenic etiology of these diseases by prescribing which of the following drug combinations?
   a) Dyazide and meclizine
   b) Ipratrpium and synthroid
   c) Ciprofloxicin and methylprednisolone
   d) Prednisone and rabeprazole
   e) Famotidine and Cisplatin

4. Understanding four physiologic mechanisms can help to prevent adverse drug reactions (ADRs) in the elderly. These mechanisms include all of the following except:

a) Chelation
b) Renal excretion
c) Absorption
d) Metabolism
e) Distribution

5. Drugs from the Beers list of inappropriate drugs for the elderly that have particular relevance in the geriatric otolaryngologic population include all of the following except:

a) Amitriptyline
b) Alprazolam
c) Diphenhydramine
d) Ciprofloxacin
e) Diazepam

Answers

1. b
2. e
3. c
4. a
5. d

References

1. Kane RL, Ouslander JG, Abrass IB, eds. Essentials of Clinical Geriatrics. 4th ed. McGraw-Hill; 1984.

2. U.S. Census Bureau. 1994 U.S. Census Data and Projections. www.census.gov.

3. Gates GA, Cooper JC. Incidence of hearing decline in the elderly. Acta Otolaryngol. 1991;111:240–248.

4. Moore, AM, Voytas J, Kowalski D, Maddens M. Cerumen, hearing, and cognition in the elderly. J Am Med Dir Assoc. 2002;3(3):136–139.

5. Dominguez RO, Bronstein AM. Assessment of unexplained falls and gait unsteadiness: The impact of age. Otolaryngologic Clinics of North America. 2000;33(3):637–651.

6. Doty RL, Shaman P, Applebaum SL, Giberson R, Sikorski L, Rosenberg L. Smell identification ability: Changes with age. Science. 1984;226:1441–1443.

7. Murphy C, Schubert CR, Cruickshanks KJ, Klein BE, Klein R, Nondahl DM. Prevalence of olfactory impairment in older adults. JAMA. 2002;288:2307–2312.

8. Barczi SR, Sullivan PA, Robbins J. How should dysphagia care of older adults differ? Establishing optimal practice patterns. Semin Speech Lang. 2000;21:347–361.
8 American Academy of Family Physicians. Am Fam Physi
American Academy of Family Physicians. Am Fam

9. Physician. 2002;65:615–620,622. Copyright© 2002. cian. 2002;65:615–620,622. Copyright© 2002.

10. Shindo ML, Hanson DG. Geriatric voice and laryngeal dysfunction. Otolaryngologic Clinics of North America. 1990;23(6):1035–1044.

11. Bobrow SN, Jaffe E, Young RC. Anuria and acute tubular necrosis associated with gentamicin and cephalothin. JAMA. 1972;222(12):1546–1547.

12. Lipsky BA, Baker CA. Fluoroquinolone toxicity profiles: A review focusing on newer agents. Clin Infect Dis. 1999;28(2):352–364.

13. See Bayer Corporation. Cipro (ciprofloxacin hydrochloride) US prescribing information. September 2005; Bristol-Myers Squibb Company. Tequin (gatifloxacin) US prescribing information. January 2006; Ortho-McNeil Pharmaceutical, Inc. Levaquin (levofloxacin) US prescribing information. July 2005; Pfizer. Maxaquin (lomefloxacin hydrochloride) US prescribing information. January 2005; Bayer Pharmaceuticals Corporation. Avelox (moxifloxacin hydrochloride) US prescribing information. December 2005; Sanofi-Synthelabo Inc. NegGram (nalidixic acid) US prescribing information. June 2004; Merck & Co., Inc. Noroxin (norfloxacin) US prescribing information. July 2004; and Ortho-McNeil Pharmaceutical Inc. Floxin (ofloxacin) US prescribing information. Revised May 2004.

14. Hanlon JT, Schmader KE, Ruby CM, et al. Suboptimal prescribing in older inpatients and outpatients. J Am Geriatr Soc. 2001;49:200–209.

15. Beers MH. Explicit criteria for determining potentially inappropriate medication use by the elderly: An update. Arch Intern Med. 1997;157:1531–1536.

16. Fick DM, Cooper JW, Wade WE, Waller JL, Maclean JR, Beers MH. Updating the Beers criteria for potentially inappropriate medication use in older adults: Results of a US consensus panel of experts. Arch Intern Med. 2003;163:2716–2724.

17. Curtis LH, Østbye T, Sendersky V, et al. Inappropriate prescribing for elderly Americans in a large outpatient population. Arch Intern Med. 2004;164:1621–1625

18. See Hanlon, Schmader, Ruby, et al. 2001, note 14.

19. Grymonpre RE, Mitenko PA, Sitar DS, Aoki FY, Montgomery PR: Drug-associated hospital admissions in older medical patients. J Am Geriatr Soc. 1988;1092–1098.

20. Blackwell Synergy. Adverse drug reactions in elderly patients. Br J Clin Pharmacol. 57(2):121–126.

21. See Cunningham G, Dodd TRP, Grant DJ, Murdo MET, Richards RME. Drug-related problems in elderly patients admitted to Tayside hospitals, methods for prevention and subsequent reassessment. Age Ageing. 1997;0:375–382;

Mannesse CK, Derkx FH, de Ridder MA, Man in 't Veld AJ, van der Cammen TJ. Adverse drug reactions in elderly patients as contributing factor for hospital admission: Cross sectional study. Br Med J. 1997;315:1057–1058.

22. Mannesse CK, Derkx FH, de Ridder MA, Man in 't Veld AJ, van der Cammen TJ. Contribution of adverse drug reactions to hospital admission of older patients. Age Ageing. 2000;29:35–39.

23. Lazarou J, Pomeranz BH, Corey PN. Incidence of adverse drug reactions in hospitalized patients: A meta-analysis of prospective studies. JAMA. 1998;279:1200–1205.

24. Wiffen P, Gill M, Edwards J, Moore A. Adverse drug reactions in hospital patients. Bandolier Extra. 2002;1–15.