accompanied by red maculae on the hard palate and without lesions on other parts of the body.[5–9]

Herpes virus (HHV-8) is known to be involved in the pathogenesis of KS. Other factors such as genetics, immunosuppression and deregulation of cytokines, growth factors, and adhesion of molecular functions cooperate with HHV-8 to start developing KS.[2,8–10] In this report, the patient had anemia, but there is no evidence in the literature to support this association.

Thus, this report highlights the importance of paying attention to elderly patients, who may have asymptomatic lesions in the oral cavity signaling other infections, including the formation of malignant tumors. Health professionals and caregivers should be able to identify changes in these patients to reach an early diagnosis favoring the prognosis and quality of life for this group of individuals.

David Braga, DDS
Wagner Hespanhol, PhD
Maria Aparecida Cavalcante, PhD
Department of Oral and Maxilofacial Surgery
Universidade Federal do Rio de Janeiro, Rio de Janeiro RJ, Brazil

Taciria Bezerra, DDS
Department of Pediatric Dentistry, Universidade Federal do Rio de Janeiro, Rio de Janeiro, RJ, Brazil

Virginia de Matos
Pharmaceutical-biochemistry, Cytopathology Laboratory
Universidade Federal do Rio de Janeiro, Rio de Janeiro RJ, Brazil

Fernanda S. Cavalcante, MSc
Pricilla Matos, MSc
Dennis de Carvalho Ferreira, PhD
Instituto de Microbiologia Paulo de Góes, Universidade Federal do Rio de Janeiro, Rio de Janeiro, RJ, Brazil

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REFERENCES

1. Ramirez VA, Anaya GS, Martinez GM. Kaposi's sarcoma of the head and neck: A review. Oral Oncol 2010;46:135–145.
2. Leão JC, Porter S, Scully C. Human herpesvirus 8 and oral health care: An update. Oral Surg Oral Med Oral Radiol Endod 2000;90:694–704.
3. Ferreira DC, Paiva SS, Carmo FL et al. Identification of herpesviruses types 1–8 and human papillomavirus in acute apical abscesses. J Endod 2011;37:10–16.
4. Sullivan RJ, Pantanowitz L, Casper C et al. HIV/AIDS: Epidemiology, pathophysiology, and treatment of Kaposi sarcoma-associated herpesvirus disease: Kaposi sarcoma, primary effusion lymphoma, and multicentric Castleman disease. Clin Infect Dis 2008;47:1209–1215.
5. Centeno A, Danielo C, Campana R et al. Tumores malignos de boca. Med Cutan Iber Lat Am 2010;38:221–228.
6. Solivetti FM, Elia F, Latini A et al. AIDS-Kaposi sarcoma and classic Kaposi sarcoma: Are different ultrasound patterns related to different variants. J Exp Clin Cancer Res 2011;30:40.
7. Dal ML, Polesel J, Ascoli V et al. Classic Kaposi's sarcoma in Italy, 1985–1998. Br J Cancer 2005;92:188–193.
8. Schwartz RA, Micali G, Nasca MR et al. Kaposi sarcoma: A continuing conundrum. J Am Acad Dermatol 2008;59:179–206.
9. Seleit I, Attia A, Maraee A et al. Isolated Kaposi sarcoma in two negative patients. J Dermatol Case Rep 2011;2:24–25.
10. Jakob L, Metzler G, Chen KM et al. Non-AIDS associated Kaposi's sarcoma: Clinical features and treatment outcome. PLoS ONE 2011;6: e18397.

AGE AND SPEECH PRODUCTION: A 50-YEAR LONGITUDINAL STUDY

To the Editor: Speech changes with age, affecting quality of life.[1,2] Underlying degenerative processes include laryngeal neuromuscular degeneration through atrophy and dystrophy and edema in the vocal fold cover.[3–6] Because voice production structures share physiological territory with the aerodigestive tract, age-related degeneration of the voice may coincide with degeneration of other important functions such as breathing, swallowing, and airway protection. Historically, age-related voice studies have been cross-sectional in nature, identifying age-related vocal characteristics by comparing an elderly group with a younger group. Although such studies provides general trends, longitudinal case studies may provide additional insights by tracking the progression of voice, swallowing, and breathing characteristics with age without the effects of intersubject statistical averaging and variability.

The current case study used 50 years (1958–2007, aged 48–98) of speech recordings. The subject is a male lay leader of an international church. In addition to the unique longitudinal breadth of his speeches, this subject and his body of speeches are unique because he received no training as a public speaker and used none of the traditional rhetorical characteristics of sermons; he avoided smoking, coffee, and alcohol, common vocal irritants that might obfuscate age-specific changes to the voice; the acoustical environment was consistent, one of two multipurpose university arenas; and all of the speeches were long enough to provide a sustained representative voice sample for analysis. Two types of analyses were employed: speech fundamental frequency to reveal the current health of the laryngeal physiology, and length of speech breath groups to indicate efficiency of laryngeal valving and vital lung capacity.

Overall, the participant's voice changed significantly in the mid to latter part of his sixth decade (Figure 1), which could be traced to age-related physiological processes. Generally, speech fundamental frequency decreased until about age 68 (Figure 1A). From age 68 to 98, average pitch increased from 140 to 160 Hz, and range (interquartile range) decreased 20%. Because speech fundamental frequency depends on the physiology of the vocal folds and control of the musculature of the larynx, changes

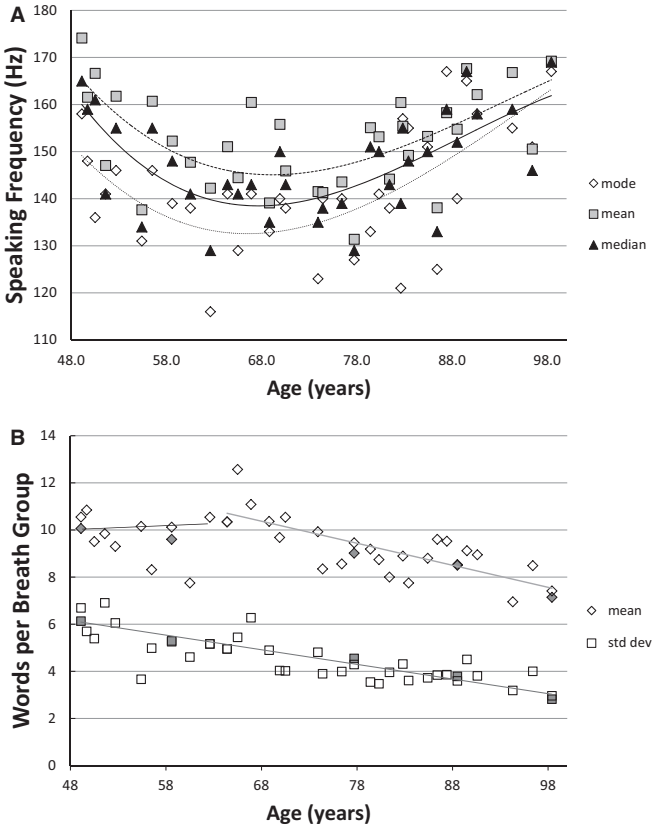

Figure 1. (A) Speaking fundamental frequency changes over a lifetime: mode, mean, and median. (B) Average (diamonds) number of words per breath group and standard deviation (squares) of words per breath group, as counted by a reviewer. Solid filled symbols represent a second reviewer for rater reliability testing.

in mean and range may suggest a deterioration of the state of the tissue and general motor control with age. For example, age-related loss of mass by itself would increase the average speech fundamental frequency, but loss of mass in the vocal folds could cause the vocal folds to begin to bow.[7] Furthermore, if the participant adjusted for the bowing by increasing the stretch of the vocal fold to assist with glottal closure during phonation, this would also raise average speech fundamental frequency.

Changes in speech fundamental frequency corresponded with a reduction in breath group length. The participant increased the number of breath groups per minute (6.3% per decade), losing approximately 6% to 6.5% of speech breath group length per decade (Figure 1B). This change was almost imperceptible until the sixth decade. Simultaneously, the standard deviation of words per breath group decreased nearly linearly throughout the observation period. Thus, the participant could not sustain the same number of words in a breath group and needed to breathe more frequently while speaking. This change might have been caused by a less-flexible rib cage and the loss of vital capacity. It may have been caused by increased glottal chink or bowing of the vocal folds,[8] resulting in more air leakage during speaking and reducing the air available.

Although the longitudinal breadth of the study period makes these results valuable, they are nevertheless preliminary because only one individual was examined. It is possible the results were affected by variations of recording environment, recording equipment, and audio compression, none of which were controlled. Nevertheless, the effects were probably minimal because the venues and communication context were similar, the metrics used would be less sensitive to these variabilities, and the results were similar to other reports in the literature.

Systemic neuromuscular changes can be inferred from changes in speech fundamental frequency and speech breathing. Other changes, such as increased risk of dysphagia (the inability to swallow safely and efficiently), may also correlate with these changes. Additional studies may identify indicators of when further assessments and treatments of age-related changes (e.g., dysphagia, dysphonia) are needed or when preventative exercise may assist in slowing age indicators.[9,10] Future longitudinal studies using more participants (of both sexes) may further understanding of normal changes due to aging versus pathology, but such a corpus of recordings must first be filtered based on communicative intent, venues, knowledge of vocal coaching, and related information.

Eric J. Hunter, PhD
Mara Kapsner-Smith, MS
Patrick Pead
Megan Z. Engar, BS
Wesley R. Brown, MD
National Center for Voice and Speech, Salt Lake City Utah

Eric J. Hunter, PhD
Center on Aging, University of Utah, Salt Lake City, Utah
Division of Otolaryngology–Head and Neck Surgery
School of Medicine, University of Utah
Salt Lake City, Utah

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REFERENCES

1. Golub JS, Chen PH, Otto KJ et al. Prevalence of perceived dysphonia in a geriatric population. J Am Geriatr Soc 2006;54:1736–1739.
2. Schneider S, Plank C, Eysholdt U et al. Voice function and voice-related quality of life in the elderly. Gerontology 2011;57:109–114.
3. Hirano M, Kurita S, Nakashima T. Growth, development, and aging of human vocal folds. In: Bless D, Abbs JH, eds. Vocal Fold Physiology. San Diego, CA: College Hill Press, 1983, pp 22–43.
4. Honjo I, Isshiki N. Laryngoscopic and voice characteristics of aged persons. Arch Otolaryngol 1980;106:149–150.
5. McMullen CA, Andrade FH. Functional and morphological evidence of age-related denervation in rat laryngeal muscles. J Gerontol A Biol Sci Med Sci 2009;64A:435–442.
6. Baken RJ. The aged voice: A new hypothesis. J Voice 2005;19:317–325.
7. Bloch I, Behrman A. Quantitative analysis of videostroboscopic images in presbylarynges. Laryngoscope 2001;111(11 Pt 1):2022–2027.
8. Linville SE. Source characteristics of aged voice assessed from long-term average spectra. J Voice 2002;16:472–479.
9. Burkhead LM, Sapienza CM, Rosenbek JC. Strength-training exercise in dysphagia rehabilitation: Principles, procedures, and directions for future research. Dysphagia 2007;22:251–265.
10. Thomas LB, Harrison AL, Stemple JC. Aging thyroarytenoid and limb skeletal muscle: Lessons in contrast. J Voice 2008;22:430–450.

A CURIOUS CASE OF *LACTOBACILLUS CASEI* IN A PROSTHETIC JOINT: WAS IT THE YOGURT?

To the Editor: Although declining, the incidence of hip fractures in elderly adults remains a significant cause of morbidity and mortality.[1] The mainstay of treatment remains hip replacement. The prevalence of prosthetic hip joint infection ranges from 0.5% to 1.3%.[2] Treatment of such infections usually requires a combination of surgery and long-term antibiotic therapy. The most common bacteria isolated from prosthetic hip infections are staphylococci, streptococci, and enterobacteraceae.[3]

The case of a 95-year-old white woman with a history of hypertension, insulin-dependent diabetes mellitus, coronary artery disease, chronic systolic heart failure, and dementia who underwent left total hip replacement after a fall resulting in a displaced intertrochanteric fracture of the left proximal femur 1.5 years before this admission is presented. Three months before this admission, she began experiencing a functional decline and complained of left hip discomfort. Plain films obtained at her home revealed no radiographic evidence of fracture or change in the position of her prosthetic hardware. Three days before this admission she was noted to have a stage III ulcer along the lateral aspect of her left thigh, erythema, and a moderate amount of serosanguinous drainage. She was admitted to the hospital.

On admission, she was afebrile and normotensive, without tachycardia or tachypnea. Laboratory tests were notable for a high white blood count (WBC) (11,500/μL) with a differential of 76% polymorphonuclear cells, a mildly high erythrocyte sedimentation rate (46 mm/h; normal 0–30 mm/h), and a high C-reactive protein (66.9 mg/L; normal 0–5 mg/L).

Computed tomography images of her left lower extremity demonstrated an elongated, heterogeneous, complex collection in the left thigh (Figure 1); ultrasound-guided diagnostic aspiration of the fluid collection produced <1 mL of thick, hemorrhagic material that was diluted in sterile saline and sent for microbiology. Because of suspected prosthetic hip infection, vancomycin and ceftriaxone were started for empirical coverage of common pathogens, including methicillin-resistant *Staphylococcus aureus* (MRSA).

Over the next few days, despite antibiotic therapy, her WBC rose to 15,000/μL, and she developed a low-grade fever of 99.5°F, with increasing somnolence. Given her clinical deterioration despite antibiotics, after discussion with her and her son, who was her healthcare proxy, she was taken to the operating room where she underwent extensive debridement of the left hip, excision of a sinus tract, and modular exchange of the hemiarthroplasty components. Cultures were obtained from the intracapsular synovium, extracapsular tissue, and superficial sinus tract.

Superficial wound cultures grew methicillin-sensitive *Staphylococcus aureus* (MSSA). Deep cultures from the initial ultrasound-guided aspiration fluid and the intracapsular synovium grew lactobacillus species, eventually identified as *Lactobacillus casei* and *L. paracasei*. The isolate was sensitive to penicillin, gentamicin, erythromycin, clindamycin, and linezolid but resistant to vancomycin. The antibiotic regimen was changed, and she completed 4 weeks of intravenous penicillin G followed by a prolonged course of suppressive therapy with oral

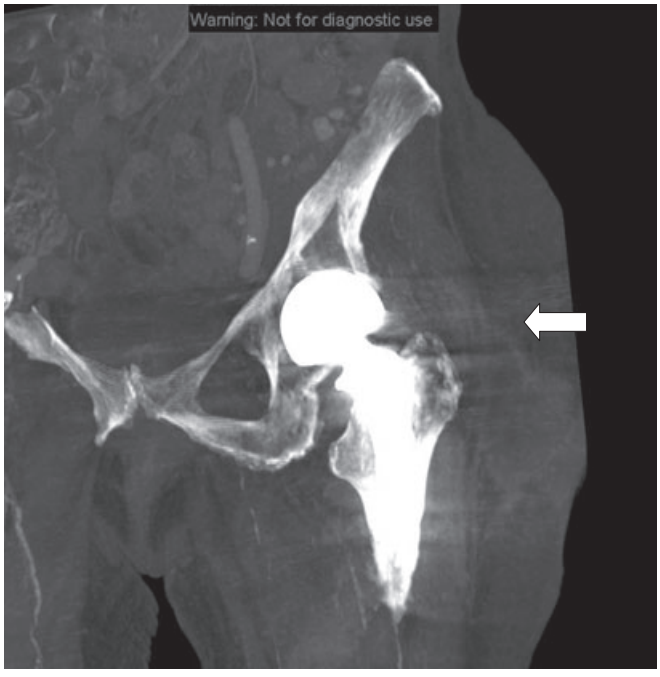

Figure 1. Three-dimensional reconstruction of the patient's left hip demonstrating the sinus tract communicating from the prosthetic hip replacement to the skin, as indicated by the arrow.