

DEVELOPMENT OF VOICE IN HEARING-IMPAIRED INDIVIDUALS: OVERVIEW OF PHYSIOPATHOLOGICAL ASPECTS

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A Study design/planning
B Data collection/entry
C Data analysis/statistics
D Data interpretation
E Preparation of manuscript
F Literature analysis/search
G Funds collection

Abstract

This article presents an overview of information on the development of voice, based on a detailed analysis of the literature. The data demonstrate voice abnormalities in individuals with hearing impairment as compared to individuals with normal hearing. Many researchers believe that the main factor leading to voice abnormalities among hearing-impaired individuals is incorrect auditory feedback resulting from higher hearing thresholds. This lack of feedback leads to abnormal encoding of neuronal patterns in cortical regions of the brain, which results in a disrupted control of internal and external laryngeal muscles.

Key words: hearing impairment • voice disorders • auditory feedback • acoustic voice parameters • aerodynamic parameters • voice development

ROZWÓJ GŁOSU U OSÓB Z ZABURZENIAMI SŁUCHU: WYBRANE ASPEKTY FIZJOPATOLOGII GŁOSU

Streszczenie

Niniejszy artykuł prezentuje przegląd informacji dotyczących rozwoju głosu w oparciu o szczegółową analizę doniesień literaturowych. Przedstawione dane opisują nieprawidłowości głosu u osób z zaburzeniami słuchu w porównaniu do głosu osób prawidłowo słyszących. Zdaniem większości badaczy głównym czynnikiem prowadzącym do zaburzeń głosu u osób niesłyszących i niedosłyszących jest nieprawidłowa kontrola słuchowa głosu wynikająca z podwyższonego progu słuchu. Brak właściwej kontroli słuchowej prowadzi do kodowania w korze mózgowej nieprawidłowych wzorców neuronalnych, a przez to do zaburzeń kontroli czynności wewnętrznych i zewnętrznych mięśni krtani.

Słowa kluczowe: wada słuchu • zaburzenia głosu • kontrola słuchowa • parametry akustyczne głosu • parametry aerodynamiczne • rozwój głosu

Introduction

The human voice is a sound generated in the larynx and further modified by resonance organs including the throat, nasal cavity, and sinuses. The basic tone generated in the glottis is characterized by specific physical features. In the upper level of the vocal tract, the voice receives unique subjective and individual features that depend upon the structure of the larynx and facial part of the skull. Measurable, physical voice parameters correspond well with subjective features. Thus, physical voice features such as frequency, amplitude, and spectral structure are perceptively represented by pitch, loudness, and timbre. Thus, the voice is a source of information and conveys human emotions.

The voice appears in newborns immediately after birth as a reflex. Voice continues to develop in further stages of life and, together with the development of speech, voice becomes a useful tool of interpersonal communication. Studies demonstrate that the production of voice is controlled by the central nervous system and the auditory system. Thus, disorder in either of those control systems

can lead to abnormalities in voice quality and changes in acoustic structure.

Purpose

This paper aims to synthesize the current literature on the development of the human voice in individuals with hearing impairment as compared to individuals with normal hearing.

Materials and methods

We conducted a literature search with the following key words: voice development, acoustic voice parameters, auditory control, voice disorders, vocalization, multi-dimensional acoustic voice analysis, nasalance, audiogenic dysphonia, and audiogenic dyslalia. Polish and English key words were used. Publications were found from the following databases: Pubmed, Web of Science, Scopus, and the Warsaw Medical University Library Polon system. A total of 51 articles (14 in Polish, 37 in English) were retrieved and used in final analyses.

Voice development in hearing impaired individuals

Compared to normally hearing individuals, the voice of hearing-impaired individuals is marked by an increase in average fundamental frequency (F0), an increase in the variation of the fundamental frequency (vF0), an increase in the variation of amplitude, and a decrease in phonation time [1–4]. The data suggests that hearing-impaired individuals have difficulty in maintaining long-term frequency and amplitude control during phonation. Perceptively, this difficulty results in vocal instability which can take the form of tremor, poor modulation, and elevated pitch. Those voice features are described as ‘audiogenic dysphonia’. The speech of hearing-impaired individuals is marked by incorrect patterns of articulation, or ‘audiogenic dyslalia’. Voice quality depends on adequate functioning of the auditory organs and the respiratory tract. With appropriate hearing, a correct voice range develops, and with an ability to change voice intensity, development of correct acoustic voice parameters is possible. Appropriate hearing thus enables the development of speech and prosody.

Voice production is controlled by auditory feedback that takes place within the central nervous system and any disturbance in this feedback results in abnormalities in acoustic voice parameters. Appropriate functioning of the respiratory system is necessary to ensure aerodynamic parameters for the production of a healthy human voice.

Early in life, hearing impaired children show no differences in the larynx compared to healthy children. Differences appear in the first years of life and the level of differences depends on various factors, including type and profoundness of hearing impairment, as well as the method and effects of rehabilitation. Voice disorders in poorly hearing or deaf children are usually functional [5].

For individuals with hearing impairment, changes can be seen in voice development within the first few months of life as compared to individuals with normal hearing. In particular, voice is typically higher pitched, shows greater fluctuations, and is less stable. Although the major part of initial vocalization in the first few months of life comes from intrinsic motivation (i.e., with no external sound stimulation), the role of auditory feedback increases in later developmental stages [6,7]. Observation of the vocal functions of hearing-impaired children in the first few months of life allows for the testing of differences in vocalizations compared to normally hearing children. In the first year of life, the occurrence of canonical babbling is delayed among hearing-impaired children. In contrast, children with normal hearing produce much more canonical syllables. Development of this ability in hearing-impaired children is usually delayed by 4–6 months [8–10]. In addition, the total time of this delay was found to vary based on the severity of the hearing impairment [11,12].

Apart from the quality of vocalization, an impairment in hearing can also affect the number of episodes [13,14]. Various studies have demonstrated that hearing impaired or deaf children produce significantly less vocalizations than their healthy peers, and the inhibitory influence of hearing impairment is strictly related to its severity. In deaf

children, the number of vocalizations is less than in those in children with partial hearing impairment. In both groups, however, the number of vocalizations is substantially lower than in their normally hearing peers [14,15]. Research at later stages of development (16–24 months) shows that the transition from babbling to words is also delayed among hearing-impaired children. Moreover, as speech develops, hearing-impaired children show delayed and/or a more limited use of syllables which contain consonants, incorrect vowel articulation, and/or a poorer range of vocabulary compared to children with normal hearing [11,13–15].

Changes in the acoustic structure of voice among hearing impaired individuals are observed at every developmental stage. Changes in the acoustic structure are primarily related to voice frequency. In particular, deaf and partially deaf individuals use a narrower range of frequencies and tend to have less control over the pitch of their voices [16–24]. The voice of hearing-impaired children is frequently dull, fluctuating, and harsh, and is accompanied by high muscle tension; it is puffy, toneless, silent, monotonous, and devoid of melody. Thus, the presence of a hearing impairment disrupts the self-regulation of pitch, volume, rhythm, and timbre of the voice [22,23,24].

Other studies demonstrate abnormalities of resonance – often in the form of nasalance – among individuals with hearing disorders [25–27]. In these individuals, voice and speech becomes nasal, dull, and dark. As a reason of the nasalance, most researchers consider there is inappropriate central nervous system control of velopharyngeal muscle function due to a disruption of auditory control [28–30]. Another potential cause of nasalance may be a slower rate of speech among the hearing impaired [31–33]. Elevated nasal resonance is observed in perceptive and acoustic examinations, whereas electromyography (EMG) results of palatal muscles remain unchanged compared to standard values [29,34].

Several research centers have conducted studies aimed at explaining the mechanisms underlying voice abnormalities among individuals with hypoacusis and deafness. These studies demonstrate that a lack, or inappropriate, auditory control of voice leads to abnormal voice characteristics by inducing changes in aerodynamic parameters. These changes arise as a result of disturbed coordination between internal and external muscles of the larynx, and due to abnormalities in the tension and relaxation of antagonistic muscles. Comparative studies of normal and hearing-impaired individuals have revealed statistically significant differences in the vital capacity of lungs (VC), maximum sustained phonation (MSPT), and fast adduction–abduction rate (FAAR). In particular, hearing-impaired individuals show significantly lower VC, a shorter MSPT time, and decreased FAAR compared to normally hearing individuals. Although peak expiratory flow (PEF) is numerically lower among hearing-impaired individuals as compared to normally hearing individuals, this difference did not reach statistical significance [35,36].

Parallel research conducted on mechanism of respiration and chest mobility suggest abnormalities in the coordination of chest and abdomen movements among deaf and poorly hearing individuals. In particular, deaf and

hearing-impaired individuals tend to initiate phonation with incorrect (either too large or too small) air volume in the lungs. In addition, the mean air amount per syllable is doubled among deaf or hearing-impaired individuals versus normally hearing subjects. These data suggest that deaf or hearing-impaired individuals speak out fewer syllables for each expiration phase, which causes breaks for aspiration at grammatically inappropriate moments during speech.

Radiological examinations of the larynx (X-ray) in hearing-impaired individuals reveal that the glottis closes with excess muscle tension (i.e., hyperadduction) [36–38]. Changes in the larynx are already present during childhood for those born with hypoacusis, such as elevated or lowered vocal fold tension, glottis insufficiency, organic vocal fold changes (edema, nodules), or arytenoid asymmetry. Deaf children develop changes in the form of lower vocal fold tension with glottis insufficiency (i.e., incomplete closure). Handzel analyzed the larynx of deaf children and identified asymmetry, a lack of pneumatization of the piriform recess, a tendency to true folds atrophy, hypertrophy of vestibular folds, and dilatation of the rima glottidis [39]. Higgins et al. also noted increased air pressure in the oral cavity and higher resistance in larynx among deaf children [38]. The changes are accompanied by abnormalities in peripheral speech organs. Further, among deaf children, performance of the tongue was found to be weaker, movements of the right and left sides of the palate are asymmetric, motor function of the lips and jaw is uncoordinated, and function of the velopharyngeal muscle is disrupted, which causes nasalance. According to some researchers, these abnormalities are likely due to weakened superficial and deep sensibility in the soft palate [38].

The aforementioned differences in aerodynamic voice parameters can affect the acoustic voice characteristics of individuals with hypoacusis, and are already present during childhood and persist in later years of life. However, the degree of severity of these abnormalities strongly depends upon the time of onset, as well as the type and efficacy of the audiological intervention. Unequivocally, the best approaches for correcting voice and speech quality are achieved by early cochlear implantation or early administration of a hearing aid [41,42].

Discussion

The structure of the larynx originates in cartilages of the IV and VI branchial arch and the muscles arise from corresponding muscular buds. During embryonic development, all structures of the respiratory system originate from an abdominal prominence in the anterior part of the primary intestine. The upper part of the respiratory system ultimately transforms into the superior aperture of the larynx. Further development gives rise to two buds: (1) the buccopharyngeal bud, which later transforms into the epiglottis, and (2) the tracheobronchial bud, which later transforms into the glottis and subglottis [43].

Voice production arises from a cascade of neuronal activity in the brain. First, pre-motor cortex and supplementary motor cortices are activated. Next, the stimuli are transmitted to cortical centers that are responsible for controlling vocalization, located in the anterior part of the callosal gyrus

(Brodmann areas 24 and 33) and supplementary motor area 6 in the upper and middle frontal gyrus. The superior center of motor control for speech is localized to the posterior part of the inferior frontal gyrus (areas 44 and 45, Broca center), which coordinates the functioning of muscles that produce voice [44].

Vocal function of the larynx appears immediately following birth. Voice of the newborn is a cry of 400–600 Hz frequency and of 80–110 dB loudness. Voice production at this early stage is a reflexive activity that results from temperature differences sensed by the newborn and plays a protective role. These vocalizations enable an increase in VC of the lungs and eliminate the remaining amniotic fluid from the respiratory tract. At this stage, modulation is weak, and voice acoustic parameters are poorly differentiated. The fundamental tone and lower components are also weak and formants in the range of 1.4 to 3 kHz are considered to be high. There is an observable difference in the basic tone between male and female newborns such that the basic tone is usually higher in males compared to females. By the end of the first year in infants with normal hearing, the range of the newborn's voice becomes wider by 5–6 half-tones, and two main vowel formants appear. Next, the ability to phonate combined vowels appears, and newborns begin to babble [45]. In the first 3 months of life, the child produces sounds that are not reminiscent of speech, but rather are akin to growling and screaming. This stage is considered to be learning of phonation control and simple articulation. Vowels appear between 3 and 7 months of life, and this stage is considered to be critical for learning to control the resonance function of the vocal tract. At 7 months of life canonical babbling occurs, with more and more simple syllables forming longer sequences [46]. After 10 months, as a child hears sounds from the outside, they start to imitate these sounds. By taking part in social and family life and interacting with peers and adults, the child is confronted with different sounds that serve to enrich speech with prosodic elements such as rhythm, dynamics, and melody. Prior to puberty, the voice itself achieves a wider range from 1 octave up to 1.5 octave. At the same time, the mean position of the voice decreases and the ability to modulate the voice is enhanced. Both modulation ability and voice range may continue to grow, depending on exposure to a variety of sounds and individual features [46–49].

In the first months of life, hearing impaired children produce fewer and more limited vocalizations compared to the vivid vocalizations of their normally hearing peers. Babbling appears later, and similarly the transition from babbling to words is delayed. An analysis of acoustic and aerodynamic parameters shows many differences between hearing-impaired and normally hearing children. Whereas normally hearing children produce lower frequency vocalizations, hearing impaired children show frequency increases. Fundamental frequency variation (vF0) and peak variation of amplitude (vAm) are also increased among hearing-impaired children compared to normally hearing children. Early in development, the voice of hearing-impaired children is higher, more tremorous, and poorer in melody. In normally hearing children, in contrast, voice is melodic, lower, more stable, and contains fewer voice breaks and less subharmonic components. Healthy children also

demonstrate correct nasal resonance. In contrast, hearing-impaired children produce nasalance. Increased nasal resonance is a result of a weakened function of the pharyngeal muscles, which allows for small amounts of air from the oral cavity to enter the nose, thus giving the voice a nasal timbre.

Normally hearing children present with correct coordination of the internal and external muscles of the larynx, whereas coordination is incorrect among deaf and poorly hearing children. Handzel proved that deaf or poorly hearing children also show morphological abnormalities in the larynx, including asymmetry of cartilages, muscles, and piriform recesses, as well as functional changes (e.g., edematous vocal folds, vestibular fold hypertrophy). These functional changes are not present in the larynx of normally hearing children unless they are caused by other factors (e.g., inborn abnormalities, functional changes, vocal nodules, central auditory processing disorders).

The voice of healthy children is produced softly and without tension, and the timbre is light and sonorous. In the case of hearing-impaired individuals, uncoordinated muscles result in a voice that is tense, hard, or puffy, and is typically darker and harsh.

In summary, the voice of poorly hearing or deaf children develops over time; however the pace of this development

is slower and less dynamic compared to normally hearing children. Further, most measurable voice parameters in hearing-impaired individuals achieve values that differ significantly from those observed in healthy children. Early provision of a hearing aid supports voice and speech development and thus increases the chances for keeping pace with normally hearing children. Early intervention with a hearing aid is now considered a standard procedure [50,51].

Conclusions

At every stage of development the voice of hearing-impaired individuals shows abnormalities compared to normally hearing individuals. For individuals with hearing loss, the formation of the voice depends largely upon the type, profoundness, and time of onset of hypoacusis. Analysis of aerodynamic parameters shows a decrease in the VC of lungs and shortening of MPST time. Abnormalities in the acoustic structure of voice refer primarily to frequency and amplitude parameters, voice irregularities, and the presence of noise components. Early intervention with a hearing aid, along with audiological and phoniatric rehabilitation, improves acoustic structure and the individual's interpersonal communication. Improvement in voice acoustic parameters is a useful indicator of better auditory control.

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