Hearing Loss in Stroke–Pathways Society

by Nehzat Koohi, University College London

Stroke is the most common cause of neurological disability (MacDonald, Cockerell, Sander, & Shorvon, 2000) and about 1 in 3 stroke survivors are functionally dependent after one year (Murray and Lopez 1996). The majority of stroke survivors need rehabilitation (MacDonald et al., 2000), requiring them to be adequately informed of the nature, prognosis, and proposed treatment of their illness. Hearing plays an important role in effective communication between patients and healthcare professionals (Bensing, 2000), therefore hearing impairment may restrict participation in rehabilitation programs, leading to a lower level of physical performance (Landi et al., 2006). Both ischaemic and haemorrhagic strokes may disturb all levels of the auditory pathway and lead to hearing deficits that start acutely before, during, or shortly after the stroke. Nevertheless, hearing deficits after stroke have not been as extensively investigated as visual deficits, possibly due to the potentially “invisible” nature of this impairment compared to more obvious symptoms (e.g. dysphasia or motor loss).

Sensorineural hearing loss (SNHL) may be highly prevalent in stroke survivors (Edwards et al., 2006; Formby, Phillips, & Thomas, 1987; O’Halloran, Worrall, & Hickson, 2009); this may be due to pathology of the inner ear (H. Lee, 2012), auditory nerve, or cochlear nuclei, i.e. the part of the central auditory pathway before the crossing of the auditory fibers at the superior olivary complex brainstem level (Luxon, 1980). SNHL is distinctly different from the “Central Deafness”, i.e., the rare and dramatic occurrence of “deafness” with an additional attentional impairment component that is attributed to bilateral cortical damage, in the presence of relatively preserved cochlear and neural function (Timothy D. Griffiths, 2010). The observed association between hearing loss and stroke could be attributed to age related changes of the inner ear or the auditory nerve, as the majority of stroke sufferers are usually over the age of 60 (Jacquin et al., 2012), while risk factors for stroke such as cigarette smoking, atherosclerosis and others have also been associated with a more insidious onset of hearing impairment with advancing age (Yamasoba et al., 2013), or alternatively, to the hearing pathways being directly affected by the stroke (H. Lee et al., 2006). Furthermore, if the stroke involves the central auditory pathway in the brain, patients may suffer from additional auditory processing deficits that are not reflected by the hearing thresholds (D.-E. Bamiou et al., 2012; D. E. Bamiou et al., 2006).

This review aims to determine the type of hearing loss documented on Pure Tone Audiometry, after infarction of different cerebral artery territories.

Sudden hearing loss after stroke is less common than other neurological impairments, but it may be the initial presentation of vertebrobasilar ischemia, which is more common in the presence of vascular risk factors (Kim & Lee, 2009; H. Lee & Baloh, 2005; H. Lee & Cho, 2003; H. Lee et al., 2002; H. Lee, Whitman, Lim, Lee, & Park, 2001). Anterior Inferior Cerebellar Artery territory stroke is the leading cause, and reported to account for 83% of cases, while Posterior Inferior Cerebellar Artery stroke accounts for 12% (H. Lee et al., 2002). The type of hearing loss in both AICA and PICA is reported to be predominantly cochlear (H. Lee et al., 2002; H. Lee & Yi, 2008) but can also be mixed cochlear/retrocochlear, and less

frequently retrocochlear only (H. Lee et al., 2002). However, it should be noted that with the exception of tests such as auditory brainstem evoked responses and acoustic reflexes, very few studies have conducted any psychoacoustic tests, such as localization and temporal resolution, that may help decide if the hearing loss is peripheral or central (Ulbricht, 2003). Furthermore, the presence of severe to profound hearing loss confounds interpretation of auditory brainstem evoked responses and acoustic reflexes. Despite these limitations, there is clear evidence that AICA and PICA territory strokes may lead to mixed cochlear/retro-cochlear, and less frequently retro-cochlear only patterns of hearing loss (H. Lee et al., 2002). Hearing loss for both AICA and PICA infarcts is mostly unilateral. However, bilateral hearing loss is also possible when there is more extensive damage (Chang et al., 2013; C. C. Lee et al., 2011).

Hearing loss due to brainstem and midbrain lesions is rare and has been reported in less than 1% of isolated brainstem strokes (H. Lee & Yi, 2008). This is due the fact that the ascending pathway partially decussates in the brainstem, thus an extensive bilateral brainstem lesion is required to cause hearing loss, and such lesions are rarely compatible with life (Timothy D. Griffiths, 2010). Hearing deficits after Superior Cerebellar Artery (SCA) ischemic infarction are usually contralateral (Murakami et al., 2005). The SCA branches penetrate into the superior cerebellar peduncle, the dentate nucleus, and about two thirds of the cerebellar deep white matter (Marinkovic, Kovacevic, Gibo, Milisavljevic, & Bumbasirevic, 1995). In SCA infarction, the ischemic lesion occurs in the area where fibers from the nucleus have already crossed, and therefore sensory hearing loss is observed in the contralateral side (Doyle, Fowler, & Starr, 1996; Murakami et al., 2005).

Hearing deficits after SCA infarction can also be bilateral. Cerrato et al (Cerrato et al., 2005) report a case with bilateral hearing loss (left more than right) as one of the dominant symptoms at presentation. There are case studies of ipsilateral to the stroke hearing deficits. Lee and Yi (H. Lee & Yi, 2008) reported two patients with upper brainstem infarction on MRI, with a hearing loss ipsilateral to the stroke. One of these two cases had a cochlear type hearing loss, while the other probably combined.

There are reports of hearing loss after haemorrhagic brainstem stroke. Cohen et al. (Cohen, Luxon, & Rudge, 1996) reported a bilateral symmetrical hearing loss in a patient with medial brainstem involvement and unilateral hearing loss in a patient with a brainstem lesion prior to the decussation in the pons.

Hearing loss may also present due to stroke of higher-level subcortical structures. Musiek and Baran (Musiek et al., 2007) reported a case of a patient with a subarachnoid bleed affecting both inferior colliculi. The patient suffered from total “central” deafness during the first week post-admission but the hearing was recovered considerably two and a half weeks after the admission. Eventually, audiological testing showed a progressive recovery, in which PTA ended up within normal range. Although the auditory evoked potentials improved, they did not recover to the normal status.

The majority of patients with cortical deafness, due to functional abnormalities of the pulvinar, may suffer from some degree of attentional deficit (Pandya, 1995). Thus, obtaining accurate hearing thresholds may be a challenging task for clinicians, as patients are required to sustain

attention for a simple task. Consequently, the degree of hearing loss in such patients may have been overestimated. Nevertheless, audiological assessment procedures were not consistently employed, even where objective measures would have led to a more complete definition of the auditory deficits. Accordingly, audiological examinations that evaluate the peripheral auditory system, auditory nerve, and brainstem are highly essential for the diagnosis of cortical deafness.

In conclusion, peripheral hearing loss is observed in the vast majority of Anterior Inferior Cerebellar Artery infarction but rarely in Posterior Inferior Cerebellar Artery infarctions. Such hearing loss may occur in isolation and could also be prodromal symptom that responds well to early treatment. Brainstem and higher subcortical lesions involving the ascending auditory pathways may also affect the hearing thresholds depending on the site and size of the lesion. Finally, hearing loss is also reported in patients with lesions in primary and secondary auditory cortices. However, the so-called cortical deafness may partly be due to attentional deficit in stroke patients.

Hearing loss is not routinely assessed after stroke. Furthermore, because of other severe symptoms patient may not be aware of their hearing loss at the time of stroke. Consequently, hearing loss can remain undetected yet may have important implications for rehabilitation. Hearing loss after stroke may be an important unmet need for stroke patients and further research into prevalence, patterns, detection, and treatment are required.

Nehzat started working as an Audiological Scientist in the Department of Neuro-otology at the National Hospital for Neurology and Neurosurgery in 2009. Initially, she was particularly interested in Vestibular Science and gained extensive knowledge in this field working in Professor Linda Luxon's team. After a period of APD training provided by Dr Bamiou in her clinics, she decided to focus on Auditory Processing Disorders. She then enrolled on a PhD programme at the University College London and Dr Bamiou became her PhD supervisor. Her research focuses on hearing and auditory function in individuals with strokes. She examines hearing and auditory processing in this patient population, and her research question is how the disordered auditory processing in stroke patients can be remediated by the use of Personal Frequency Modulation (FM) systems. Recently, she had the exciting opportunity of visiting Professor Frank Musiek's Neuro-Audiology lab at the University of Connecticut where she improved her knowledge about auditory processing and auditory training of the brain.

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