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Maternal Drug Abuse and Neonatal Hearing Impairment

Michael McKenna

SUNY Plattsburgh, Department of Communication Disorders and Sciences - Spring 2015

Introduction

Infants that have been prenatally exposed to drugs or alcohol are at an increased risk for hearing impairment. Early onset hearing impairment profoundly affects the development of speech perception and language (Grimmer, Bührer, Aust, & Obladen, 1999). In addition to alcohol and tobacco, cocaine is one the drugs most commonly used by pregnant women.

General Effects of Alcohol, Tobacco Smoke, and Cocaine on Auditory Development

- Alcohol has been shown to be embryotoxic and is known to cause sensorineural hearing loss (SNHL) in the immature ear. Moreover, prenatal alcohol exposure (PAE) may result in fetal alcohol syndrome (FAS) (Church & Gerkin, 1988).
- Maternal cigarette smoking during pregnancy was associated with the reduction of efficiency of central auditory processing (Jacobsen, Slotkin, Mend, Frost, & Pugh, 2007).
- When used during pregnancy, cocaine has been shown to alter neurosensory transmission through the brainstem (Grimmer et al., 1999). Cocaine also has a direct effect on the organ of Corti, which is located in the cochlea and contains the hair cells that transduce mechanical sound vibrations into nerve impulses. Cocaine has been found to cause damage to the organ of Corti during critical periods of development (Tan-Laxa, Sison-Switala, Rintelman, & Ostrea, 2004).

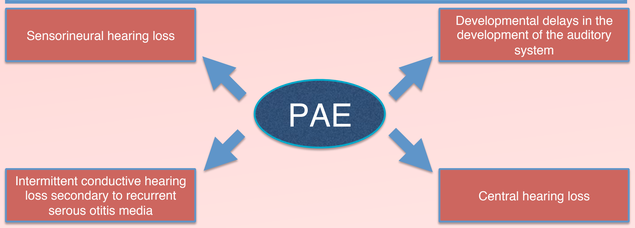

Figure 1: Four types of hearing disorders that can result from prenatal alcohol exposure

Prenatal Alcohol Exposure and Hearing

PAE can lead to FAS, which refers as a pattern of malformations occasionally seen in children born to women who consume large amounts of alcohol during pregnancy. FAS has been found to occur in 4% of all children born to alcoholic women (Church & Kaltenbach, 1997).

Because FAS is a syndrome of the neuroectoderm, it is characterized by malformations of brain tissue, ocular abnormalities, SNHL, and other sensory disorders. Cranio-facial anomalies are a distinguishing feature of FAS (Cone-Wesson, 2005). Some cranio-facial anomalies associated with FAS include microtia, stenosis of the ear canal, and low-set and/or posteriorly rotated ears (Church & Kaltenbach, 1997). Recurrent otitis media with effusion (OME) is common among those with such abnormalities. Recurrent OME is associated with conductive hearing loss, which occurs when there is an issue with the outer and/or middle ear. It is common for children with FAS to experience conductive hearing loss as a result of recurrent OME. The high rates of recurrent OME in these children is likely due to eustachian tube dysfunction, another aspect of the cranio-facial dysmorphism (Cone-Wesson, 2005).

There is a seemingly high incidence of bilateral SNHL in children with FAS. SNHL results from damage to the sensory end organs and/or auditory nerve. It is thought to be due to the fact that FAS is a syndrome of the neuroectoderm, which can be caused by exposure to ototoxic agents, such as alcohol. This neuroectoderm syndrome is characterized, in part, by congenital abnormalities of the central nervous system and sensory organs (Church & Gerkin, 1988). Pure tone audiograms indicated that about one-half of FAS children with SNHL have predominately high-frequency hearing loss, as ototoxic agents are known to typically cause high-frequency hearing losses. These hearing losses are typically classified in the mild to moderate range and warrant the use of hearing amplification (Church & Kaltenbach, 1997).

Damage to any one of the brainstem auditory nuclei, tracts, thalamocortical radiations, primary and secondary auditory cortices, and portions of the corpus callosum, can result in a central hearing disorder (See figure 1). Central hearing disorders can range from an impaired ability to understand speech sounds (despite a normal pure tone audiogram) to complete deafness. Excessive alcohol exposure can damage the central auditory system, and dysfunction may extend beyond the brainstem region (Church & Kaltenbach, 1997).

Prenatal Cocaine Exposure and Hearing

When cocaine is used during pregnancy, it crosses the placenta to the fetus. Cocaine affects the maternal cardio-vascular and autonomic systems, and thus has an indirect effect on the fetus (Cone-Wesson, 2005). Its use has been associated with alterations in the developing monoaminergic neurotransmitter systems, with implications for both anatomical and physiological aspects of fetal brain development (Morrow et al., 2004).

It has been found that cocaine blocks the reuptake of norepinephrine at the nerve endings and results in an excess of circulating catecholamines. These issues result from the poor development of the monoaminergic transmitter system, which works directly to modulate norepinephrine and catecholamine levels (Tan-Laxa et al., 2004).

Anoxia may be a factor resulting in the prolonged auditory brainstem response (ABR) latencies seen in infants that have been prenatally exposed to cocaine (Cone-Wesson, 2005). The vasoconstrictive effects of cocaine on the uterine vessels decreases blood flow to the fetus and thus can result in placental insufficiency and ischemic/hypoxic injury to the developing brain, brainstem auditory system, and the entire central nervous system, all of which are extremely sensitive to oxygen deprivation (Tan-Laxa et al., 2004).

Several other factors may be influential in the effects of PCE. Factors such as the timing of cocaine exposure during pregnancy, other drugs that may be taken during pregnancy, and the amount of exposure to cocaine, all may lead to different, and possibly additional, effects on the auditory system (Tan-Laxa et al., 2004).

A normal ABR Is dependent on both an intact peripheral hearing mechanism and an auditory brainstem. It is common that infants with PCE that have been tested via ABR show significantly prolonged absolute peak latencies in most of the potentials; this suggests abnormalities in the peripheral auditory system (middle ear, cochlea, and auditory nerve) (Tan-Laxa et al., 2004). The prolonged latencies for later peaks of the ABR are consistent with central nervous system effects of cocaine, because wave III and V are dependent upon the integrity of the brainstem auditory nuclei (See figure 2). The I-V inter-peak latencies represent central brainstem conduction time from the auditory nerve to the inferior colliculus in the midbrain and is used as a measure of brainstem maturation. For PCE infants, the waves I-V inter-peak latencies were frequently prolonged when tested, suggesting delaying brainstem maturation (Lester et al., 2003). These prolongations may also suggest that infants with PCE have increased susceptibility to neural adaptation, possibly due to neurotransmitter depletion causing synaptic insufficiency (Cone-Wesson, 2005).

Prenatal Exposure to Tobacco Smoke and Hearing

Deficits in auditory processing may be one of the neurodevelopmental consequences of maternal cigarette smoking during pregnancy that leads to later language and reading deficits (Kable, Coles, Lynch, & Carroll, 2009).

According to results from transiently evoked otoacoustic omissions (TEOAE) testing performed on infants who have been prenatally exposed to maternal cigarette smoking, it has been found that maternal smoking during pregnancy may have a detrimental effect on the developing cochlea. Subtle effects of cigarette smoke exposure on the inner ear are already apparent and assessable at birth. Maternal cigarette smoking during pregnancy also alters the function of the auditory brainstem (Katbamna, Klutz, Pudrith, Lavery, & Ide, 2013). When tested via ABR, these infants are typically found to have significantly reduced transmission times associated with a decreased latency; however, the relationship is dose-responsive (Kable et al., 2009).

Observations show that reductions in cortical cholinergic neurotransmission resulting from prenatal cigarette smoke exposure may lead to reduced efficiency of central auditory processing by reducing the selectivity of auditory perception, as well as decreased efficiency of the neurocircuitry that supports auditory attention (Jacobsen et al., 2007).

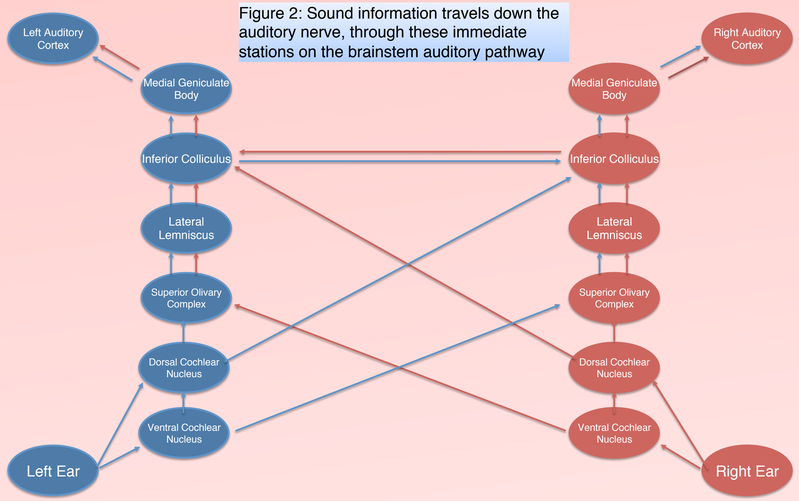

Figure 2: Sound information travels down the auditory nerve, through these immediate stations on the brainstem auditory pathway

Conclusion

It is probable that prenatal exposure to environmental toxins such as alcohol, cocaine, and tobacco smoke, will result either directly or indirectly, in the underdevelopment of auditory abilities. Maternal tobacco smoking elevates the risk of cognitive and auditory processing deficits (Jacobsen et al., 2007). The use of cocaine by pregnant women may result in neonatal retrocochlear hearing loss and abnormalities in central auditory processing. Prenatal alcohol exposure can result in FAS; due to the cranio-facial anomalies associated with FAS, these individuals are prone to recurrent OME and conductive hearing loss (Cone-Wesson, 2005).