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The Challenge and Opportunity of New Technology in Otolaryngology

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Description

Hyperbilirubinemia is common in the new born owing to their special metabolism characteristics. Physiologic bilirubin levels are protective because of their antioxidant effects [1], while pathological jaundice is harmful to the health of the neonate. Previously, most pathological studies on the jaundice of neonates have concentrated on central nuclei in the auditory brainstem. Later, epidemiological investigation has demonstrated that the pathology is highly related with auditory neuropathy. That is to say, hyperbilirubinemia can also cause injury to peripheral hearing pathways. The present review mainly illustrates the neurotoxic effects on the hearing system by bilirubin and treatment aspects of auditory neuropathy.

Metabolism of Pilirubin

Bilirubin is a kind of pigment containing tetrapyrrol structure and the final decomposition product of proteins containing heme, such as hemoglobin, myoglobin and cytochrome. Average daily production of bilirubin is about 250 ~ 350 mg in adults. When the red blood cells are engulfed and digested by mononuclear macrophages or rectuloendothelial cells, heme is released from hemoglobin and turned into biliverdin under the action of various enzymes (heme oxygenase, NADPH, cytochrome c reductase) in the cytomicrosome. Bileverdin is then turned into Unconjugated Bilirubin (UCB) by biliverdin reductase. UCB is fat-soluble and is carried to the liver when bound with plasma albumin. In the smooth surfaced endoplasmic reticulum, UCB is turned into conjugated bilirubin under the catalysis by UDP-Glucuronosyl Transferase (UDPGT). Conjugated bilirubin is water-soluble and can be filtrated from the glomerulus and then exhausted in the urine. Under normal circumstances, only a small part of unconjugated bilirubin is not combined with albumin which is not harmful to the nervous system.

Hyperbilirubinemia and Injury of Bilirubin to the Hearing System

Albumin can act as the carrier of UCB and prevent bilirubin from penetrating cell membrane when they combine together. The concentration of albumin is usually far higher then that of bilirubin. When they combine tightly at the ratio of 1:1, the concentration of free bilirubin does not rise [1]. Under some situations, such as when red blood cells break in large scales as in the new born baby whose liver DUPGT is not mature with less than fully developed blood-brain-barrier, or as in pathological hemolysis or use of certain drugs (sulfa drugs, non-steroidal anti-inflammatory drugs, biliary contrast agent and free fatty acid) that compete for binding to plasma albumin, increase of plasma UCB can happen in the newborn baby's body. As UCB is not only lipid-soluble, but also able to pass through the blood-brain barrier and cell membrane, its' neurotoxic property can lead to neuronal damage.

Bilirubin is harmful at multiple sites in the auditory system, including central auditory nuclei such as dorsal and ventral cochlear nucleus, superior olivary nucleus, trapezoid body and lateral lemniscus [2]. Long exposure to bilirubin can result in extensive neuronal injury in the hearing system. Previous studies suggested that the injury was confined to the central auditory system and that the cochlea and the auditory nerve were not affected [3,4] but latest epidemiologic study evidence revealed that hyperbilirubinemia was highly associated with auditory neuropathy and that the incidence is positively correlated with bilirubin concentration in blood. Studies on the peripheral hearing system of Gunn rats with nuclear jaundice revealed that spiral ganglion neurons showed severe degeneration, especially those in large nerve fibers with myelin, while no morphological change was observed in hair cells. Ye et al. [5] demonstrated that bilirubin could cause demyelination and nerve fibers decrease in the Habenula perforate. The synapses between hair cells and afferent nerve ends were also damaged without visible change in the inner or outer hair cells. Although nobody has demonstrated that bilirubin affects the excitement of spiral ganglion neurons, injury on the SGN by bilirubin will inevitably cause neuronal dysfunction, leading to mild to profound hearing loss based upon exposure time and intensity. The reason why SGN is particularly sensitive to the toxic effects of bilirubin is unclear.

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