Dissertation on

"A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR"

submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

In partial fulfillment of the regulations For the award of degree of

M.S., OTORHINOLARYNGOLOGY

(BRANCH IV)

REGISTRATION NUMBER:220420102004

DEPARTMENT OF OTORHINOLARYNGOLOGY

GOVERNMENT KILPAUK MEDICAL COLLEGE & HOSPITAL

CHENNAI -600010

MAY 2022

BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled **"*A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR*"** is a bonafide record of the research work done by **Dr.V.R.VINOTHAN,** Post graduate in the Department of Otorhinolaryngology, Government Kilpauk Medical College, Kilpauk, Chennai-10 in partial fulfillment of the regulations laid down by The Tamil Nadu Dr.M.G.R. Medical University for the award of **M.S. Degree in Otorhinolaryngology (Branch IV),** under my guidance and supervision during the academic year from 2020-2022.

Dr.S.MUTHUCHITRA
M.S(ENT).,DLO.,
Professor & HOD,
Department of
Otorhinolaryngology,
Govt. Kilpauk Medical College
and Hospital, Kilpauk,
Chennai-10.

DR.R.SHANTHIMALAR MD.,DA.,DME (OSD)
The Dean,
Govt. Kilpauk Medical College and
Hospital, Kilpauk,
Chennai-10.

CERTIFICATE FROM GUIDE

This is to certify that the dissertation titled **"*A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR*"** is a bonafide original work done by **Dr.V.R.VINOTHAN,** Postgraduate in Otorhinolaryngology, under my direct supervision and guidance in the Department of Otorhinolaryngology, Govt. Kilpauk Medical College, Chennai,in partial fulfillment of the regulations of the Tamilnadu Dr. M.G.R. Medical University for the award of **M.S. Degree in Otorhinolaryngology (Branch-IV)** during the academic period of 2020-2022**.**

Dr.S.MARY NIRMALA DNB(ENT).,DLO.,

Associate Professor ,

Department of Otorhinolaryngology,

Govt. Kilpauk Medical College and Hospital,

Kilpauk, Chennai-10.

DECLARATION

I, **Dr.V.R.VINOTHAN,** solemnly declare that the dissertation titled **"*A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR*"** is the bonafide work done by me at the Department of Otorhinolaryngology, Govt. Kilpauk Medical College and Hospital, Chennai, under the guidance of **Dr.S.MUTHUCHITRA M.S(ENT).,DLO.,** Professor and Head, Department of Otorhinolaryngology, Chennai.

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the University regulation for the award of **M.S degree in Otorhinolaryngology (Branch-IV)** examinations to be held in May-2022.

Station: Chennai-10 **Dr.V.R.VINOTHAN**

Date: **(Reg .No: 220420102004)**

ETHICAL COMMITTEE CERTIFICATE

GOVT. KILPAUK MEDICAL COLLEGE,

CHENNAI-10

Protocol ID. No. 414/2020 Meeting held on 12/11/2020

Reg.No. ECR/1385/Inst/TN/2020

CERTIFICATE OF APPROVAL

The Institutional Ethics Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for "A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR" Submitted by Dr. VINOTHAN V.R, Post-graduate, Department of ENT, Govt. Kilpauk Medical College, Chennai-10.

Proposal is APPROVED.

The Institutional Ethics Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

For DEAN

DEAN

Govt. KILPAUK MEDICAL COLLEGE

CHENNAI - 600 010.

ACKNOWLEDGEMENT

I wish to express my sincere thanks to **PROF. Dr.R.SHANTHIMALAR MD.,DA.,DME(OSD).,** The DEAN, Government Kilpauk Medical College and Hospital for having permitted me to utilize the facilities of the hospital for conducting this study.

My heartfelt gratitude to Prof**. DR.S.MUTHUCHITRA M.S(ENT)., D.L.O.,** Professor, Department of Otorhinolaryngology, Government Kilpauk Medical College and Hospital for her constant motivation, valuable suggestions, and expert supervision during the course of this study.

I thank, **PROF. Dr.S. MARY NIRMALA DNB (ENT).,DLO., AND PROF. Dr.S.GANAPATHY M.S(ENT).,DNB.,** for their guidance and support.

I wish to thank my Assistant Professors, **DR.P.THAMIZHARASAN MS, DLO, DR.K.SANJAYKUMAR MS(ENT)., DR.G.UDAYACHANDRIKA MS(ENT)., DR.C.BALAJI MS.,DLO DR.P.SIVARAM DLO., DR.V.MANIMEHALAI DLO.,** for their valuable tips and support.

I am grateful to the department of pediatrics and audiologists.

I wish to thank my senior **Dr.Mohammed Rashid.H** , my fellow colleagues **Dr.Indu .S.P. , Dr.Sharumathi R.R , Dr.Kousalya.P , Dr.Naadia.R** and my junior post graduates who most willingly helped me during this study period. I also thank the staff nurses, OPD staff, Department of Otorhinolaryngology, Government Kilpauk Hospital for their cooperation and assistance in the conduct of this study. I wish to extend my gratitude to my statistician for his expert assistance.

Last but not the least, I am indebted and grateful to all the Patients who constitute the backbone of this study, who most willingly and selflessly subjected themselves to this study for the sake of the benefit of their community and without whom this study would not have been possible.

Date: **Dr.V.R.VINOTHAN**

Place: Chennai

PLAGIARISM CERTIFICATE

Analyzed document	Dr Vinothan V R dissertation.docx (D125467440)
Submitted	2022-01-19T10:53:00.0000000
Submitted by	Vinothan
Submitter email	drvinoent@gmail.com
Similarity	14%
Analysis address	drvinoent.mgrmu@analysis.urkund.com

Sources included in the report

SA	**Tanmaya Tyagaraj.docx** Document Tanmaya Tyagaraj.docx (D119563724)	7
SA	**Tanmaya Tyagaraj.docx** Document Tanmaya Tyagaraj.docx (D119253390)	3
SA	**Tanmaya.docx** Document Tanmaya.docx (D119704857)	3
W	URL: http://52.172.27.147:8080/jspui/bitstream/123456789/6981/1/Shenoy%20Shwetha.pdf Fetched: 2021-11-15T05:04:16.7670000	4
W	URL: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5675031/ Fetched: 2019-09-25T11:40:05.3400000	26
W	URL: https://www.frontiersin.org/articles/655625 Fetched: 2021-11-20T05:15:21.4770000	1
W	URL: http://repository-tnmgrmu.ac.in/7056/1/200700311venkataramanan.pdf Fetched: 2021-11-15T05:03:46.0830000	18
W	URL: https://emedicine.medscape.com/article/836646-overview Fetched: 2019-10-09T09:42:23.6830000	2
SA	**Tamil Nadu Dr. M.G.R. Medical University / Dr SARANYA.docx** Document Dr SARANYA.docx (D31303290) Submitted by: ssaranyasavi@gmail.com Receiver: ssaranyasavi.mgrmu@analysis.urkund.com	1

PLAGIARISM CERIFICATE

This is to certify that this dissertation work titled ***"A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR"*** of the candidate **Dr.V.R.VINOTHAN** with registration Number **220420102004** for the award of **M.S. Degree** in the branch of **OTORHINOLARYNGOLOGY.** I personally verified the urkund.com website for the purpose of checking plagiarism. I found that the uploaded thesis file contains contents from introduction to conclusion and result shows **14%** of plagiarism in the dissertation.

DATE:
PLACE: Chennai

Dr. S. MUTHUCHITRA M.S. (ENT)., DLO.,
Professor & HOD,
Department of Otorhinolaryngology
Govt. Kilpauk Medical College and
Hospital, Kilpauk,
Chennai-10.

.

ABBREVIATIONS

DPOAE	– DISTORTION PRODUCT OTOACOUSTIC EMISSION
AABR	– AUTOMATED AUDITORY BRAINSTEM RESPONSE
SNHL	- SENSORINEURAL HEARING LOSS
AC/BC	- AIR CONDUCTION/ BONE CONDUCTION
HL	- HEARING LOSS
IHC	- INNER HAIR CELL
OHC	- OUTER HAIR CELL
OAE	- OTOACOUSTIC EMISSION
AN	- AUDITORY NERVE
DCN	- DORSAL COCHLEAR NUCLEI
VCN	- VENTRAL COCHLEAR NUCLEI
AVCN	- ANTEROVENTRAL COCHLEAR NUCLEI PVCN
	- POSTERO VENTRAL COCHLEAR NUCLEI MSO
	- MEDIAL SUPERIOR OLIVARY COMPLEX LSO
	- LATERAL SUPERIOR OLIVARY COMPLEX MNTB
	- MEDIAL NUCLEUS OF TRAPEZOID BODY NLL
	- NUCLEUS OF LATERAL LEMNISCUS
CNIC	- CENTRAL NUCLEUS OF INFERIOR COLLICULUS
MGBv	- VENTRAL MEDIAL GENICULATE BODY
AC	- AUDITORY CORTEX
Rt/ Lt	- RIGHT/LEFT

CONTENTS

"A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR"

INTRODUCTION

Congenital hearing loss arises when the ear's ability to transfer the vibratory mechanical wave of sound into electrical energy in the form of nerve impulses is hindered. Sensorineural hearing loss occurs when parts of the auditory nerve or the central auditory pathway are damaged. Conductive hearing loss occurs when the outer or middle ear is compromised. Conductive and sensorineural hearing loss are both included in the term "mixed hearing loss." Hearing loss due to conductive causes occurs when sound waves can't travel through the ear properly due to problems with the middle ear, the external ear, or both. Hearing loss caused by damage to the hair cells in the inner ear is classified as sensory hearing loss, while those caused by damage to the central auditory pathway are classified as central hearing loss. Disorders of the Auditory Nervous System. Acoustic Neuropathy Spectrum Disorder encompasses a wide variety of clinical diseases marked by otoacoustic emissions and a cochlear microphonic in conjunction with aberrant or absent auditory brainstem responses, which leads in impaired speech discrimination. As a result of an inner hair cell lesion, Auditory Neuropathy Spectrum Disorder could be induced by damage to neuronal networks of an intervening synapse in the auditory nerve.[1]

Neonatal hearing surveillance are readily available in the majority of industrialised nations for children born with this common problem. Within a month of birth, these programmes want to test all infants. Improved developmental outcomes later in infancy are made possible by early diagnosis, early intervention, and early therapy. Neonatal hearing screening programmes may miss children having progressive hearing loss since it is possible for hearing loss to progress over time. At-risk newborns should be screened again at regular intervals. Congenital hearing loss is treated medically and supportively depending on the cause and kind of hearing loss. Genetic factors, cranio-facial abnormalities, and congenital infections are the most common causes of hearing loss. This includes both non-syndromic forms, wherein hearing loss is the sole clinical symptom, and syndromes as Usher or Jervell as well as Lange–Nielsen syndromes.[2]

Most hospitals in India now do not do hearing screenings on newborns. The prevalence of congenital hearing loss in newborns exceeds the prevalence of all metabolic diseases now detected through blood tests. Profound hearing loss is among the most common birth defects, accounting for around a quarter of all cases. Approximately one in every thousand newborns is born profoundly deaf, with severe or profound bilateral hearing loss accounting for four times as many births. In comparison to the general population, neonates in intensive care facilities have a 10–20-fold increased risk of substantial hearing loss.[2]

The National Institutes of Health (NIH) released a consensus statement in 1993 recommending universal newborn hearing screening from the age of three months. The statement also suggested using otoacoustic emission as a screening tool.[3]

Hearing loss affects 1 to 6 people in every 1000, and high-risk screening only finds half of the babies with the condition. In addition, the average age of diagnosis for children with hearing loss is 2.5 years old. Eventually, early detection and treatment of hearing loss by 6 months will lead to better outcomes.[3]

Absence of auditory input during the child's first year of life, which is important for brain development, causes major delays in the child's overall development. High-risk newborns have a greater chance of suffering from substantial hearing loss than the general public. As a result, newborn hearing screening programmes, which are already in place in industrialised nations, are needed to detect hearing loss at an early stage.[4]

The purpose of this study is to determine the prevalence as well as severity of hearing impairment in high-risk newborns by utilising the DPOAE and AABR tests.

AIM AND OBJECTIVES

AIM

To study the prevalence and severity of hearing impairment among the high risk infants using DPOAE and AABR in a tertiary care teaching hospital, Chennai.

OBJECTIVES

- To assess the hearing impairment of all high risk infants using DPOAE and AABR.

- Hearing impairment if present – Early referral of hearing impaired children for rehabilitative measures.

REVIEW OF LITERATURE

Hearing impairment is more common than any other disability in Indian children aged 0–4 years (0.60%) as well as 5–9 years (0.28%). The NHS newborn hearing screening programme is not extensively used, despite the fact that two-thirds of all people with hearing loss come from developing countries. There are many other life-threatening public health challenges in these countries, therefore hearing loss has not got adequate attention in these regions.[1]

Detection and treatment of infant hearing loss can be more effective if done at a young age. Considering that only a small fraction of newborns have hearing loss in the general population, screening all of the healthy babies would be expensive. The Joint Committee on Infant Hearing (JCIH) established a list of risk factors based on a review of all studies published to identify newborns who are most likely to have hearing impairment. Congenital infections, hearing impairment in the family, birth weight, morphological anomalies (including craniofacial anomalies), bacterial meningitis, hyperbilirubinemia, and perinatal asphyxia are a few of the risk factors to observe for. As a result of these recommendations, medical facilities can screen neonates who show any of the risk indicators in an effort to find children who have hearing loss more quickly.[2]

EMBRYOLOGY OF THE EAR.

External Ear.

The pinna begins to form in the fourth week of pregnancy, when tissues from the mandibular as well as hyoid arches condense and appear near the distal end of the first branchial groove. The pinna develops as a result of ridges known as Hillock's of His. Except for the tragus and also the anterior external auditory meatus, which come from the mandibular arch, the pinna develops from the hyoid arch. By the fifth month, the ear has fully developed into an adult configuration.[6]

External Auditory Canal.

The external auditory canal develops from the first branchial groove's dorsal portion. This groove's ectoderm meets the tubotympanic recess's endoderm. A cord of epithelial cells forms the Meatal Plate as the canal deepens in the second month, growing medially into the mesenchyme. The tympanic membrane's lamina propria is made up of mesenchyme close to the meatal plate. The first pharyngeal pouch mucosa supplies the medial layer of the tympanic membrane.[7]

Eustachian Tube.

The development of the Eustachian tube is from the Tubotympanic recess. By the 30th week of pregnancy, it has widened, elongated, and undergone mesodermal chondrification, resulting in the development of the fibrocartilaginous tube.[7]

Ossicular Chain.

The first ossicle forms in the fourth week of pregnancy. This structure arises as a result of the development of an inter-branchial bridge between the mandibular as well as hyoid arches. The mandibular arch (Meckel's cartilage) gives rise to the malleus, while the hyoid arch (Reichert's cartilage) gives rise to the incus and stapes suprastructures. It is the otic capsule that gives rise to the stapes' foot plate.[8]

The ossicles have grown to adult size by the 15th week of pregnancy, and ossification commences in the incus, malleus, and stapes. The middle ear's muscles also grow at the same time. By the completion of the 20th week of pregnancy, the ossicles have fully developed.[8]

Inner Ear.

The formation of the saccule and cochlear duct comes after the semicircular canals as well as utricle (pars superior), which are phylogenetically

older. The superior pars' immunity to developmental abnormalities is thought to be due to its evolutionary development, as opposed to the inferior pars'.

There will be a thickening of the surface epidermis dorsally, called the "otic placode," by mid-third week, which looks like a plaque. The Auditory Pit forms within days of invasion into the underlying mesenchyme. The otocyst is formed by the enlargement of the auditory pit as well as the fusing of tissue above it (otic vesicle). In the future, the otocyst will be surrounded by mesenchymal tissue that will also differentiate.

The next stage of development comprises the elongation of the otocyst and the formation of three deepening folds, that delineate the utricle, semicircular ducts, the endolymphatic sac and duct, as well as the saccule with its cochlear duct. The cochlear duct keeps growing in a spiral fashion, and by the eighth week of pregnancy, it has made two and a half complete turns. It has been found that a number of cochlear irregularities reflect the phase at which normal development has been disturbed.

By the 20th week, the fetus's organ of Corti has developed to the point that it can "hear" and react to fluid-borne noises. By the 25th week of pregnancy, the Corti organ resembles the adult form.[8]

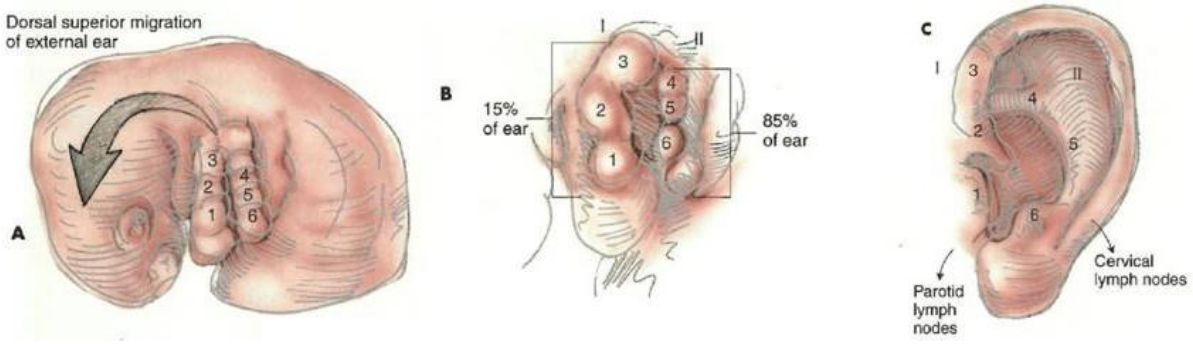


Figure 1. Embryology of external ear.[9]

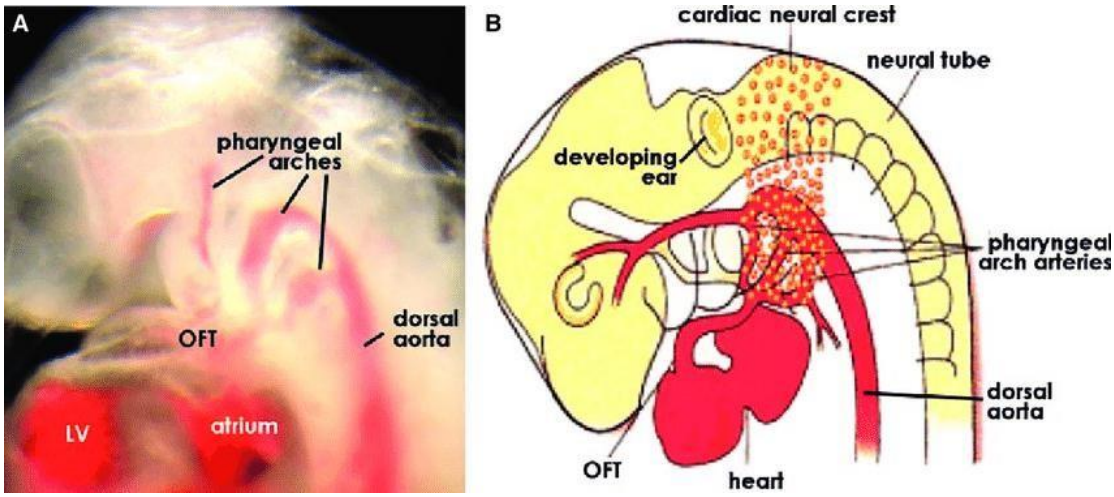


Figure 2. Embryology of ear.[10]

EAR ANATOMY.

There are three parts to the ear: the outer ear, the middle ear, and the inner ear.

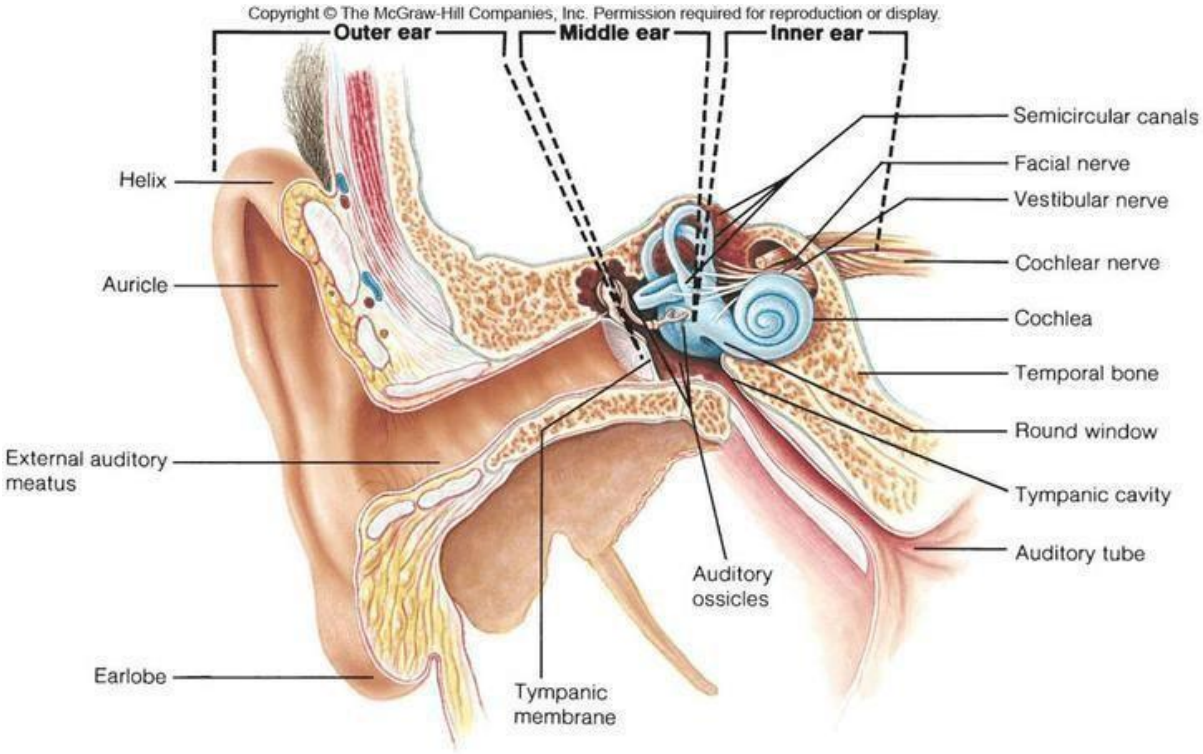


Figure 3. Anatomy of ear.[11]

External Ear.

The pinna, an extended part of the external ear, and the external acoustic meatus make up the external ear.[12]

Pinna:

The wider end of the pinna points upward. In addition to its irregularly concave lateral surface, which faces slightly forward, this feature has various eminences and depressions that have been given names. There is only one piece of yellow elastic cartilage in the pinna, which contributes to the remainder of the pinna's hardness and flexibility.[12]

The External Acoustic Meatus:

External Acoustic Meatus stretches from the base of the concha to the membrane of the tympanic cavity. The tragus extends around 4 centimeters from the tip of the ear. After passing outward and backward (pars externa), it turns into an S-curve before heading inward, forward, then slightly upward (pars media). Finally, it returns downward (pars interna). The tympanic membrane, which shuts the inner extremity of the meatus, is positioned in an oblique direction. The cartilaginous section is 8 millimeters in length. and the osseous portion measures 16 millimeters in length and is much narrower.[12]

Tympanic Membrane:

Translucent membrane that comprises the middle ear's lateral wall is known as the "tympanic membrane." The fibrous annulus secures it to the bone tympanic sulcus. It also connects to the malleus' handle at the lateral process

and the umbo formed by the manubrium's tip. The medial as well as lateral malleolar folds split the tympanic membrane into a superior pars flaccida or Sharpnell's membrane and a pars tensa. Three layers make up the tympanic membrane. On the lateral surface, squamous epithelium forms a protective barrier, whereas the mucosal layer continues on the medial side. The lamina propria, a fibrous layer, sits in the middle of the two main layers.[12]

Ossicular Chain.

The Malleus, Incus, and Stapes comprise the ossicular chain. The tympanic membrane sends sound waves to the cochlea via these tiny bones. Malleus has a neck and anterior as well as lateral processes, and is the most lateral ossicle.

The malleus is lateral to the incus, which is the largest of the ossicles. The incus has three processes: a long process, a short process, and a lenticular process. The long process is the largest and most complex. Stapes is the smallest ossicle. There's an oval window between the stapes' footplate and the cochlea, which articulates with the stapediovestibular ligament on the footplate. The stapedial head is connected to the footplate by an anterior and a posterior crus of the stapes arch.[13]

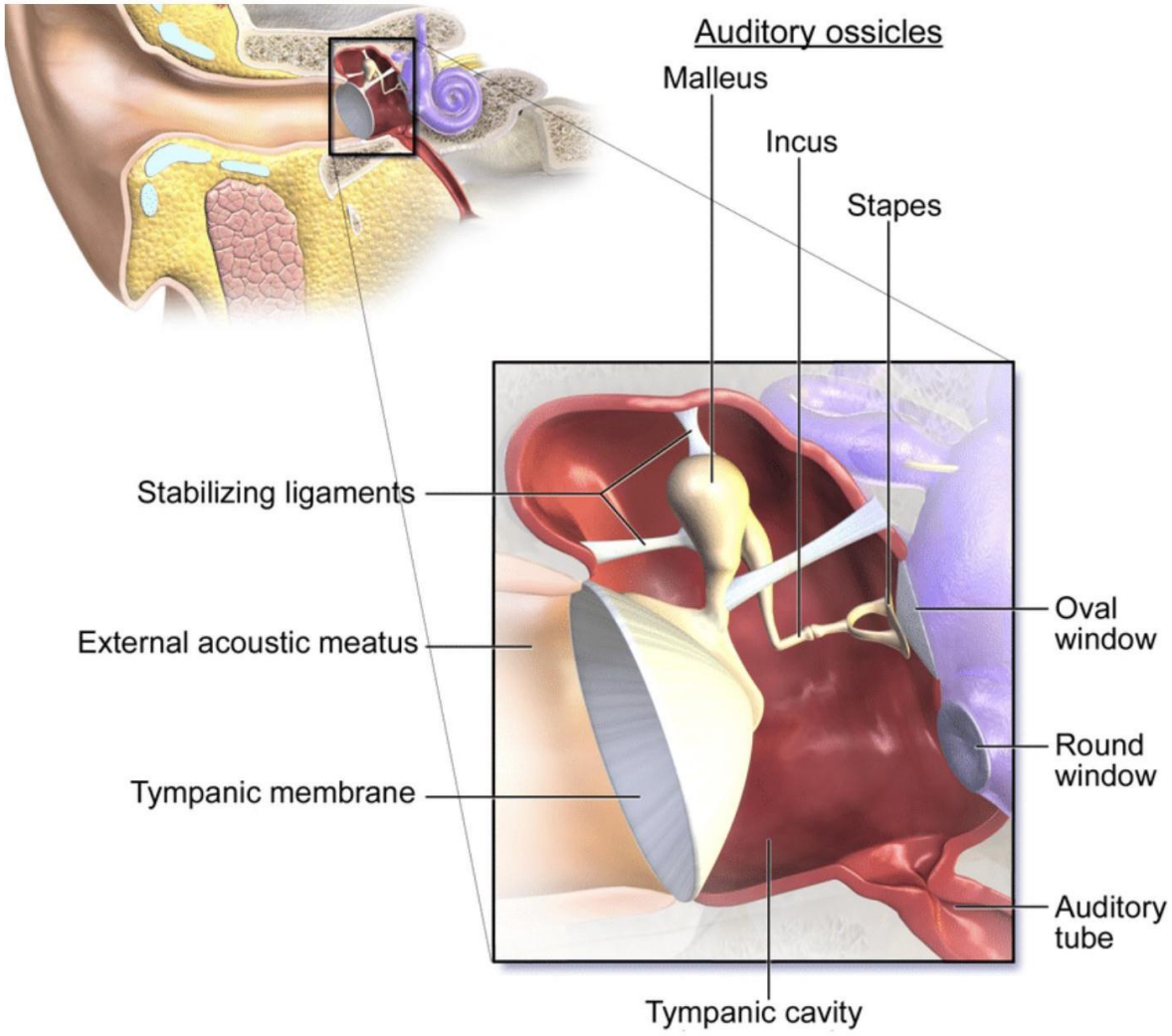


Figure 4. Anatomy of middle ear.[14]

Muscles in the middle ear:

The trigeminal nerve innervates the tensor tympani, which arises from the greater wing of the sphenoid as well as eustachian tube and joins to the malleus' handle and neck. The facial nerve innervates the stapedius, which extends from the pyramid to the stapes' posterior crus.[14]

The Eustachian tube

The eustachian tube is about 35 mm long and connects the tympanic cavity's anterior and posterior halves. Mucociliary cells, which are found in abundance in the tube's lining mucosa and play a key role in its clearance function, line the inside of the tube. About two-thirds of the anteromedial end of the eustachian tube is made up of fibrocartilaginous tissue, with the rest being bone. The middle ear's tympanic opening is located in the front wall. The tube is normally closed during rest; to open it, the tensor veli palatini muscle is used, which is controlled by the trigeminal nerve. The fibrocartilaginous tube has a fat pad on the lateral side, known as the Ostmann lateral fat pad.[15]

Inner ear

The sensory organs and soft tissue components of the inner ear are housed in the bony labyrinth. The cochlea, three semicircular canals, and the vestibule make up the inner ear. As its center axis, the cochlea's modiolus revolves two and a half times. In order to facilitate passage of the Cochlear Nerve, a hole is drilled in the fundus of the Internal Auditory Canal, which is 5mm in height.

Lateral, horizontal, and posterior are the names of the three semicircular canals. These are orthogonal to one another and cover a 240-degree arc. The

ampullated and non-ampullated ends are joined to the utricle by the sphincter of Oddi. The utricle and the saccule are housed in the vestibule's two recesses.[16]

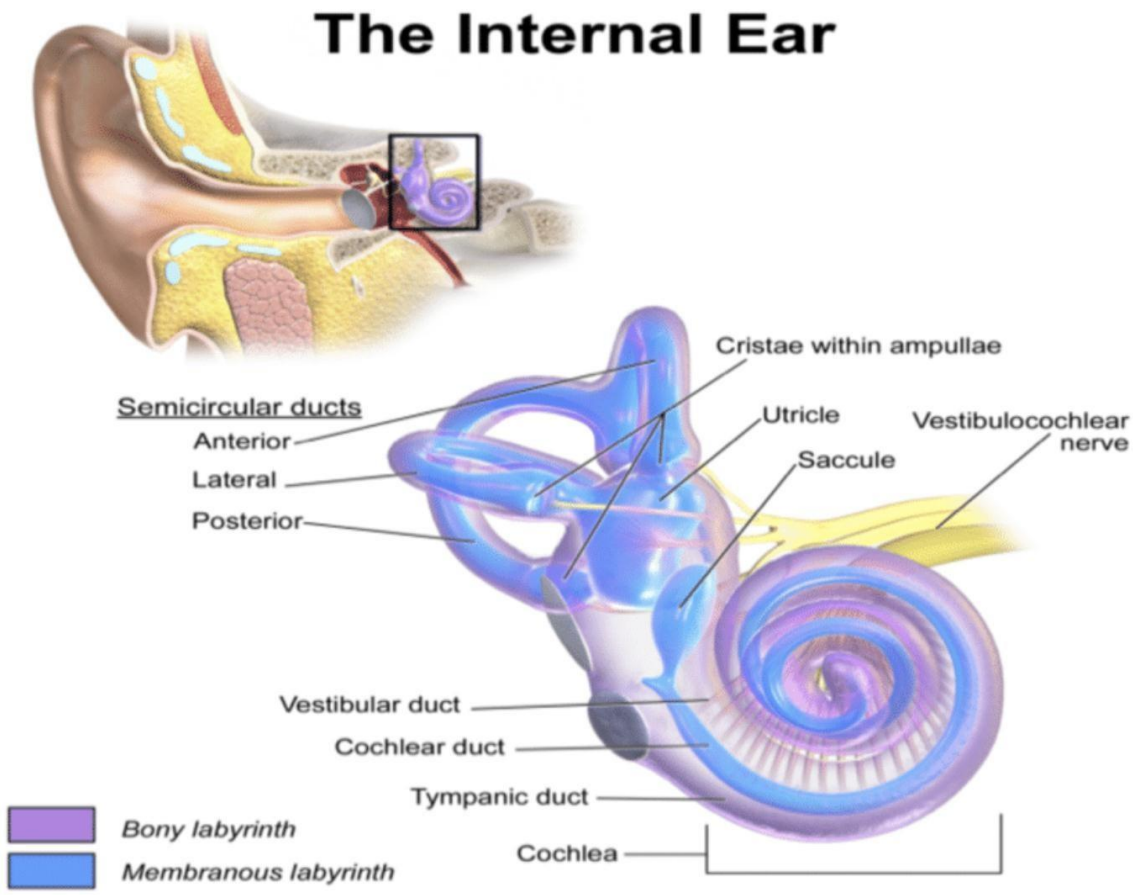


Figure 5. Anatomy of Inner ear.[17]

Internal auditory canal.

The superior and inferior vestibular nerves, the cochlear nerve, the facial nerve, the intermediate nerves, and the labyrinthine artery as well as vein are all located within the internal auditory canal.[17]

Auditory Pathway and Hearing Physiology.

The air transmits acoustic signals to the inner ear, which is filled with fluid. Air and fluid have different relative impedances, which affects how much sound power can be transmitted through an air–fluid interface. Only 30 dB of the power density of an incoming sound wave is transported to the fluid in the inner ear, making it ineffective for speech understanding.

By boosting the sound pressures which reach the inner ear at specific frequencies, the external and middle ears better match the sound-conducting characteristics of air and cochlear fluid. Traditionally, the peripheral auditory system has been divided into three peripheral components to better comprehend the primary sensitivity, timing, and frequency tuning functions it performs. This is how the exterior, middle, and inner ears all come together to make a distinct contribution.[18]

The External Ear.

External ears are like funnels, collecting and crudely filtering sound through them. With the pinna, it's easier to locate sounds, especially in areas where interaural temporal delays don't help. The Pinna works in concert with the external auditory canal to raise the acoustic pressure there at tympanic membrane with in 1.5 to 5 kHz speech frequency range. The external ear adds 2-7 kHz to the sound pressure gain.[18]

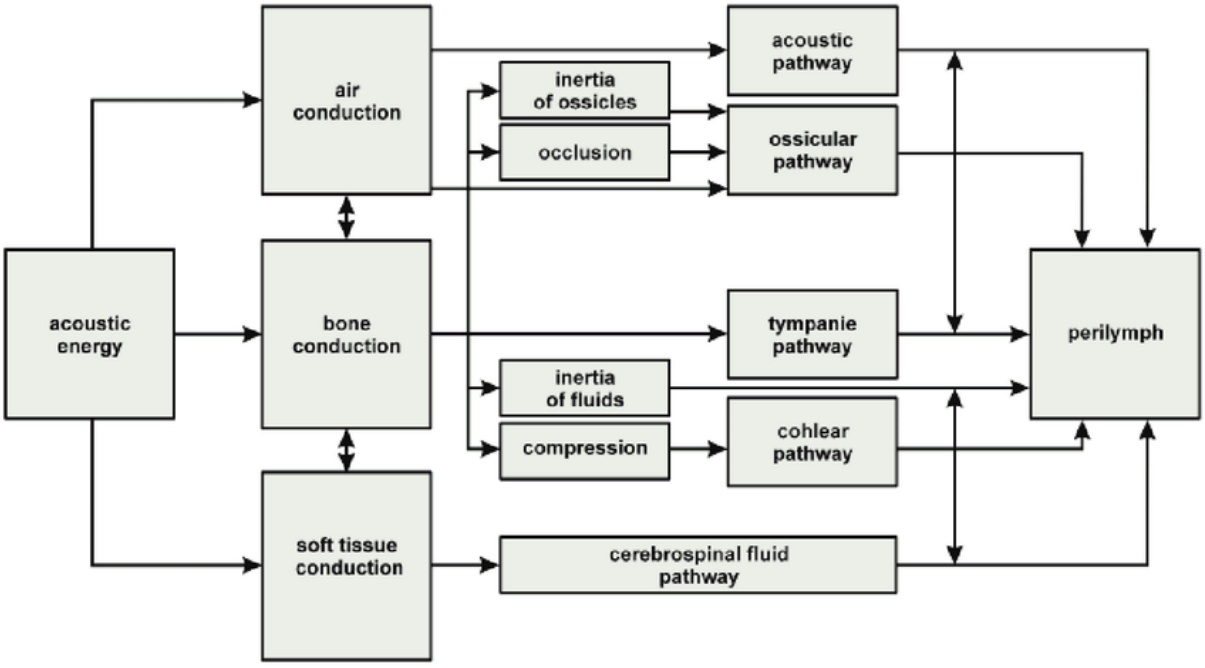


Figure 6. Mechanism of hearing receptor stimulation.[19]

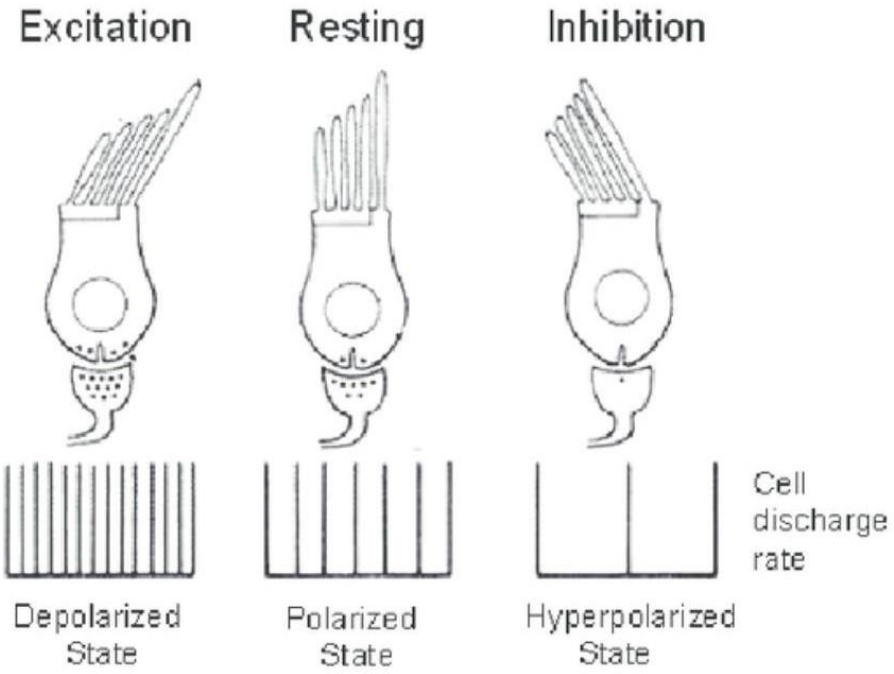


Figure 7. Hair cell physiology.[20]

The Middle Ear.

It is through the ossicles, not the tympanic membrane, that sound energy travels to the inner ear for processing. Ossicular chain is a lever system. By moving the malleoincudal joint as well as the stapes via the tympanic membrane, the oval window can move back and forth. The oval window receives sound selectively, and the round window bulges outward in reaction to an inward movement of the stapes footplate and inward when the stapes travels away from the oval window. Movement of the basilar membrane is caused by variations in the pressures communicated to the perilymph. For each rise in footplate sound pressure, a drop in stapes volume velocity occurs due to the middle ear acting as a transformer.[20]

Area ratio:

The tympanic membrane to stapes footplate area ratio is a significant middle-ear transformer mechanism (the area ratio). The tympanic membrane collects sound throughout its full surface before transferring it to the stapes' smaller footplate. For a "perfect" transformer action, since pressure equals force per area, the sound pressure applied here to inner ear by stapes footplate must be 20 times — or 26 dB — greater than the sound intensity at the human tympanic membrane.[20]

Ossicular lever:

When the malleus and incus rotate around the ossicular axis, they exert a lever action that can be described as an ossicular lever.[20]

Impedence Matching :

When the round window moves out and the oval window moves in due to the swaying of the foot plate, this is known as impedance matching. Acoustically separated windows are required for effective sound energy transfer.[21]

Inner Ear.

There are three chambers in the cochlea, which is a coiled tube. Fluids are basically incompressible, hence moving the stapes footplate around in the oval window causes fluids to flow around as well. The round window goes out when the stapes footplate moves in. The differential in sound pressure there at two cochlear windows is critical in stimulating the inner ear because of this coupling of the round as well as oval windows by its incompressible cochlear fluids.[22]

The basilar membrane, the organ of Corti, the scala media, and the Reissner's membrane make up the cochlear partition in the inner ear. When the stapes moves inward, the motion is transferred almost instantly through the

cochlear fluids, causing the round window to move outward. A travelling wave is formed as a result of this wave configuration.. The wave's maximum displacement is tonotopically structured so that high-frequency sounds cause the most displacement near the stiff, thick base, while low-frequency sounds generate the most displacement near the compliant, thin apex.[22]

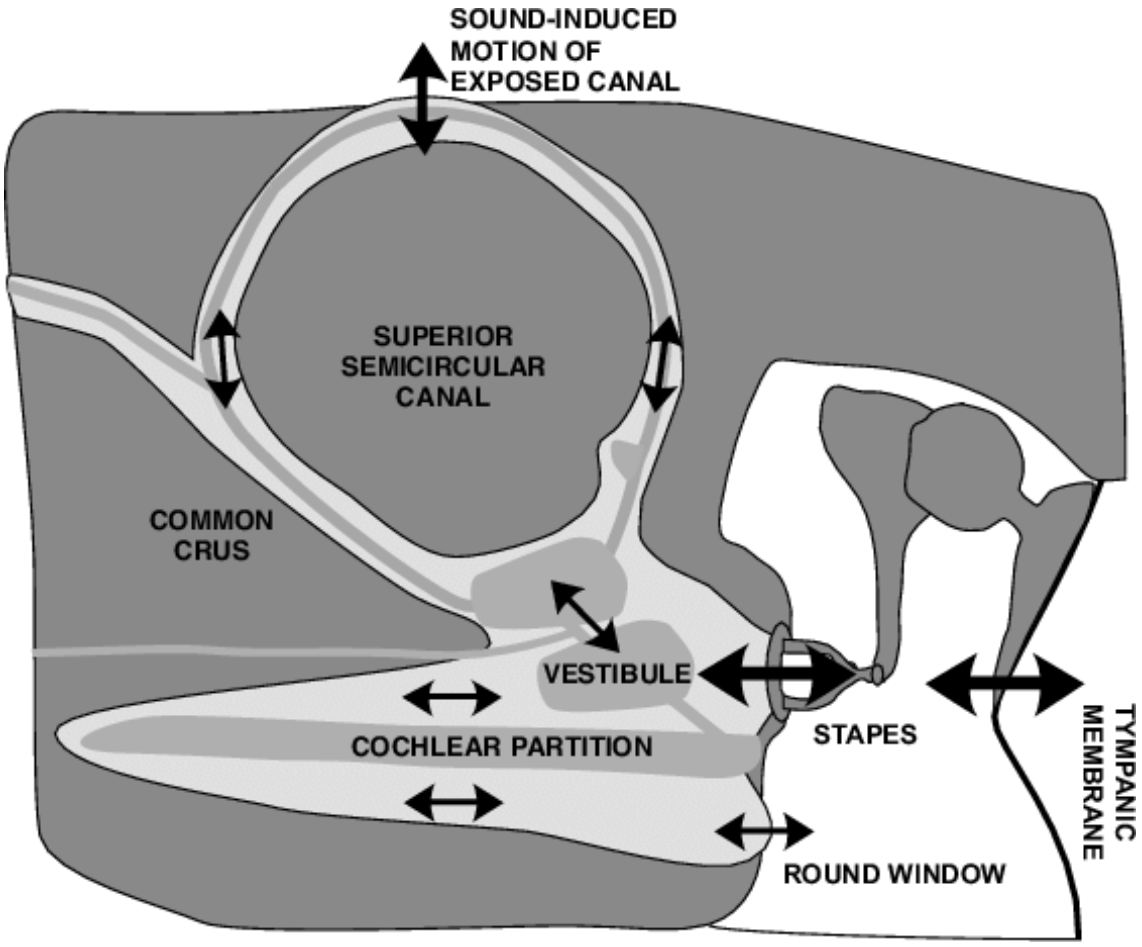


Figure 8. Response of Inner ear to sound.[23]

The tectorial membrane of the organ of Corti is sheared as the basilar membrane shifts with the motion of the cochlear fluid. The epithelium's outer hair cells are organised in three rows. It has been found that sensory hair cells have a sterociliary bundle in the upper region of the cell that is sensitive to shearing. The synaptic pole at the basal end is rounded, and the afferent and efferent nerve fibers pass through it to link with the cochlear nerve.[23]

The apical surface of the inner hair cells is flattened or concave. These cells are flask-shaped, having a large central portion and a narrowing base. Each of the inner hair cells has one row with a slender notch between them, giving the appearance of an even smaller W than the outer hair cells do.[23]

Mechanism for Impulse Transduction.

Tectorial membrane sliding across outer hair cells causes the stereocilia to deflect toward longest one in bundle because shearing force is created by movement of cochlear fluids. When stereocilia come into contact, changes in membrane potential and plasma membrane protein structure occur. As a result, calcium-gated channels are opened, generating impulses to be conveyed through the afferent neurons that invigorate the outer hair cells.[24]

The organ of Corti's Innervation.

Type I and type II neurons supply energy to the hair follicles. The inner hair cells are innervated by Type I neurons, while the outside hair cells are innervated by Type II neurons. Type I neurons make up the vast majority of the brain's cells (95 percent) The inner hair cells are reached by type I neurons entering the organ of Corti through the foramen nervosum in the habenula perforata. They enter the organ of corti via the habenula perforate, as do type II neurons, which contain unmyelinated fibres. Type II neurons, on the other hand, are not the principal channel from the cochlea for signalling.[24]

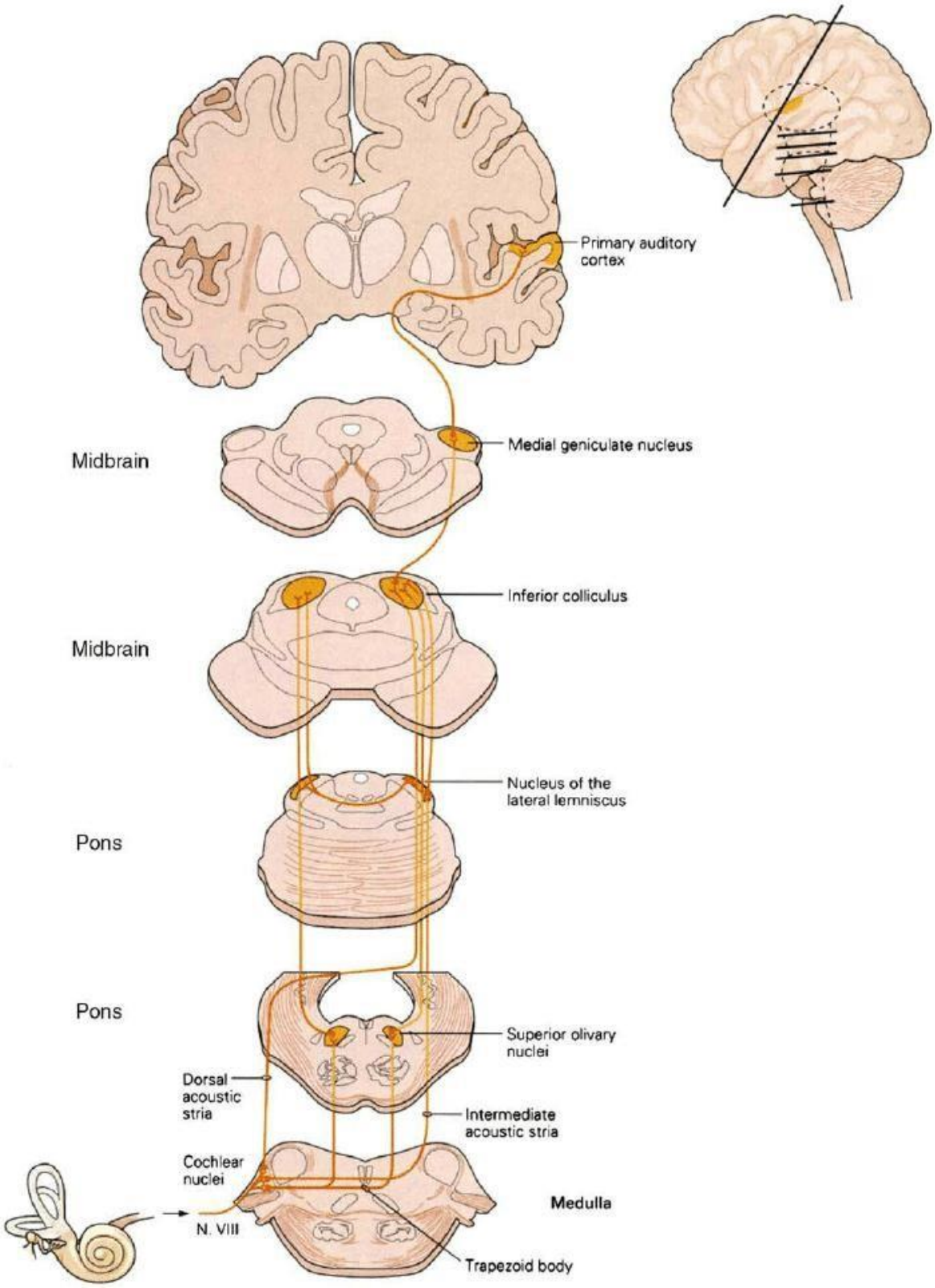


Figure 9. Auditory pathway.[25]

Auditory Pathway:

Ascending Auditory Nervous System - Classic system.

The cochlear nucleus, located at the top of the conventional ascending auditory pathway, is where synaptic transmission carries information from all of the auditory nerve fibres. Anterior Ventral Cochlear Nucleus, Posterior Ventral Cochlear Nucleus, and Dorsal Cochlear Nucleus are the three major components of the cochlear nucleus (DCN). The three acoustic striae as well as the lateral lemniscus connect the three cochlear nucleus divisions to the inferior colliculus. The inferior colliculus is the auditory pathway's primary midbrain nucleus, receiving input from various peripheral brainstem nuclei as well as the auditory cortex. The inferior colliculus processes the signals received and transmits fibres to the thalamic medial geniculate body. The primary auditory cortex receives input from neurons in the ventral region of the medial geniculate body.[25]

Ascending Auditory System - non-classic system.

There is a "adjunct" to the classic auditory system, which is the nonclassic ascending auditory pathway. These connections connect it to the inferior colliculus, inferior colliculus's external nucleus, and inferior colliculus's dorsal cortex, where it receives its auditory information. Medial and dorsal Medial Geniculate Bodies receive ascending fibres from these areas, which in

turn send them to neurons there. These neurons subsequently send out connections to the association cortex and limbic system regions. Only the cochlea provides input to the classical ascending auditory system, while other sensory systems provide information to the nonclassical auditory system.[25]

Congenital Hearing Loss.

One of the most frequent birth defects nowadays is congenital hearing loss, with a rate of permanent hearing loss between 2-3/1000 live births. Based on data of 44 state screening programmes, the CDC determined a prevalence of permanent hearing loss of 1.09/1000 people. Permanent hearing loss in newborns is defined differently among countries, ranging from 40dBHL in the UK to 35dBHL in the US.[26]

A unilateral or bilateral permanent hearing loss in the speech frequency range averaging 30-40dB is defined by the Joint Committee on Infant Hearing (JCIH, 2000) as the target group for infant screening programmes. The targeted screening population also includes people with conductive hearing loss due to outer or middle ear abnormalities.[26]

The significance of detecting and intervening in hearing loss as in early stage.

Early universal newborn auditory screening programmes uncovered a number of deaf or hard-of-hearing infants who went on to demonstrate that early detection and care can lead to almost normal language development by the age of three. Numerous demographic parameters were examined by the researchers (such as degree of hearing loss, race/ethnicity, socioeconomic position, and gender) and it was discovered that early detection was the key to better language outcomes in these individuals. Early identification that would result in normal speech as well as language development had a cutoff age of six months.[26]

Plasticity of the brain.

It is possible to "rewire" sections of the central nervous system in order to adjust brain processing to aberrant settings (such as cochlear implants). Sprouted axons and altered synaptic effectiveness can both cause such rearrangement. Unconscious learning manifests itself in the form of neural plasticity. Because the central nervous system is most malleable throughout childhood, early detection and placement of hearing aids are critical.

As a result, it's reasonable to conclude that neural plasticity played a significant influence in the high level of speech discrimination reported in early-identified implant patients. In order to get the best outcomes from implants, learning in the traditional sense is crucial as well. Adequate stimulation is necessary for optimal development of the nervous system during childhood, even if the auditory nerve system changes as a result of aberrant events.[27]

Epidemiology.

According to latest projections, persistent hearing loss of greater than 25 decibels hearing level in the poorer ear is present in at least 4 (1.1 to 6) people out of every 1000 people with hearing loss. Nearly half of these newborns have no known risk factors for hearing loss, making it difficult to detect hearing loss until the kid falls behind in other developmental areas. Hearing loss is more common in high-risk newborns, with a prevalence of 2-to-10 percent. Using a newborn hearing screening programme, doctors can look for hearing loss in newborns that ranges from 30 to 40 decibels or more in the frequency range of 500 to 4000 hertz (Hz). Speech acquisition is primarily affected by hearing loss in this range. Prevalence of hearing loss in newborns (both at risk and not at risk in India) ranges from six to sixty per thousand, with an average of four per thousand. According to another study conducted in India, 4 out of every 1000 newborns suffer significant hearing loss.[28]

Risk factors

The American Academy of Pediatrics' Joint Committee on Infant Hearing has identified many risk factors for early or late-onset congenital hearing loss in children. Persistent congenital hearing loss runs in the family, although the body of research supporting this is small, with only 1.43 percent of children with a positive family history suffering from hearing loss as proof. When a premature baby is born at 24–31 weeks gestation and their birth weight is between 750–1500 g, admission to a neonatal intensive care unit (NICU) is a significant risk factor because hearing loss is more common. Interventions such as assisted ventilation, venous access, and aminoglycoside use in the neonatal intensive care unit can raise the risk of permanent hearing loss in newborns. Hospitalization for less than 12 days, as well as a history of high-frequency ventilation treatment, have been linked to hearing loss in this group. Infants in this situation may also have a delayed maturation of their auditory system, according to previous research.[29]

In the vast majority of children with hearing loss, the condition is hereditary, generally resulting from a single gene flaw. These flaws can be inherited in many ways and manifest themselves differently depending on where you live. Hearing loss can be categorized based on whether or not it is accompanied by other physical or laboratory symptoms (syndromic hearing loss) or not (non-syndromic hearing loss). Hearing loss that isn't caused by a

genetic condition is also incredibly diverse. Eighty percent of all hereditary cases of hearing loss are autosomal recessive non-syndromic; the remaining twenty percent are autosomal dominant non-syndromic, which commonly progresses with age of onset. X-linked and maternal mitochondrial DNA-related patterns of inheritance are uncommon.[29]

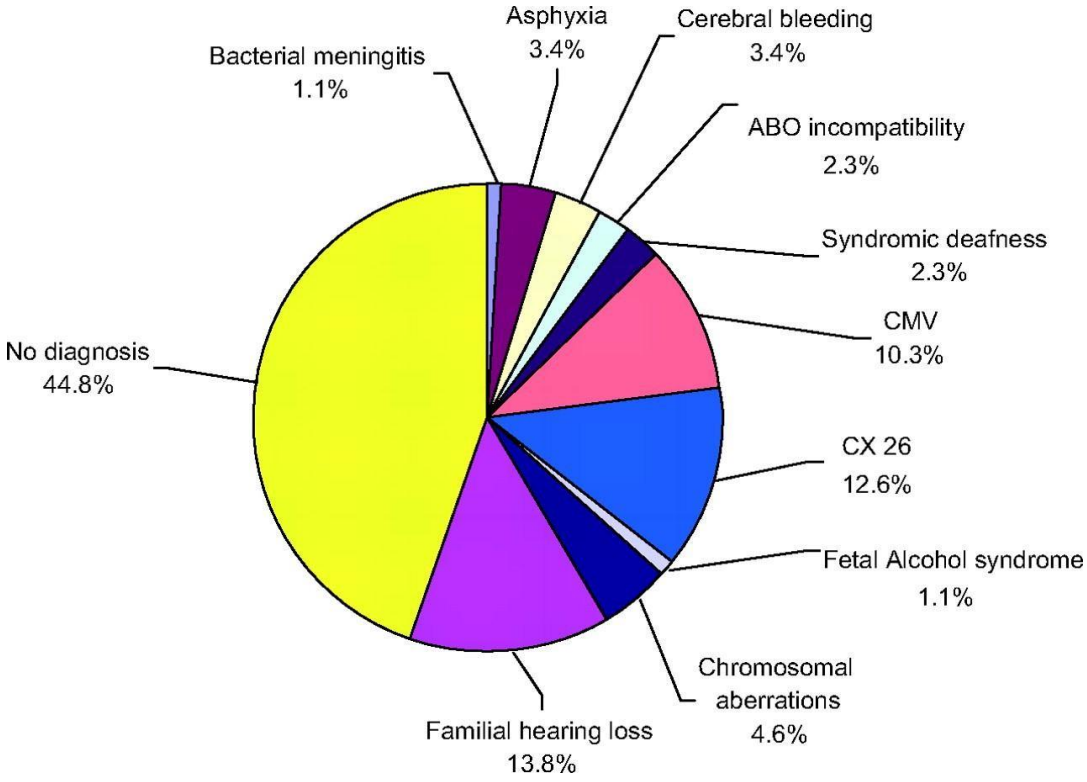


Figure 10. Etiology of congenital hearing loss.[30]

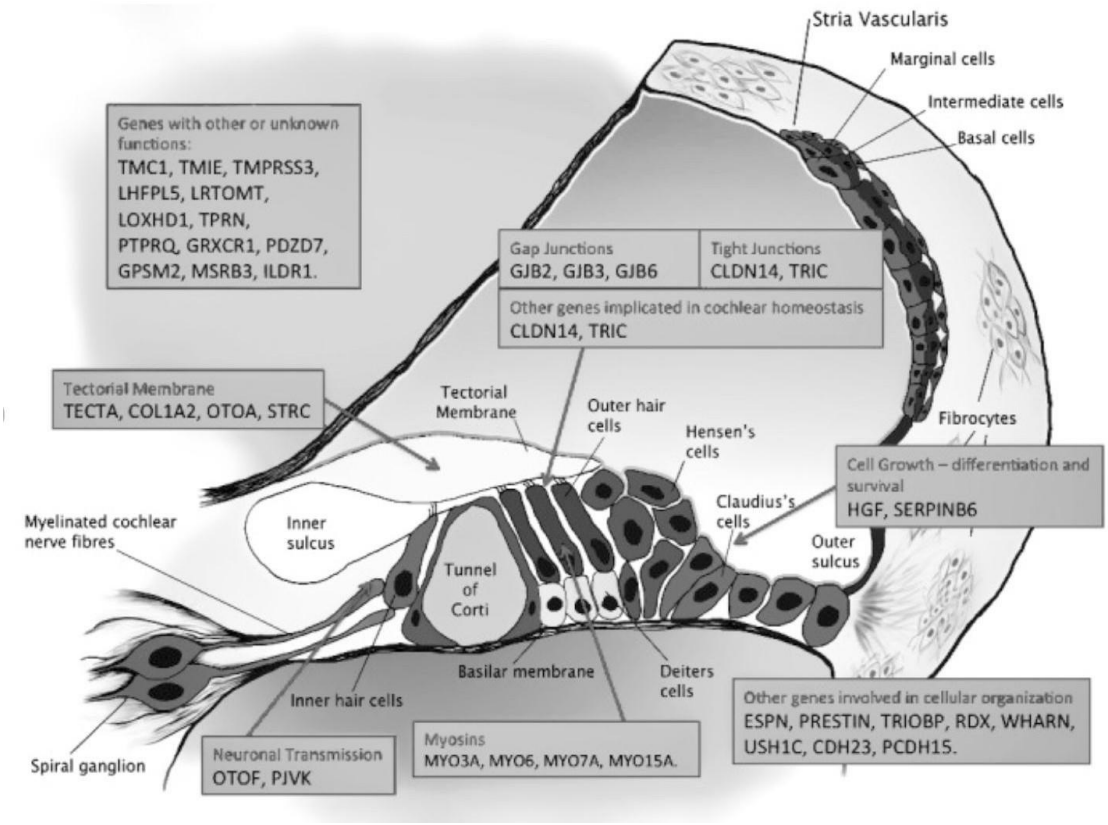


Figure 11. Genes in Autosomal recessive hearing loss.[31]

Despite the fact that the frequency of causal genes varies depending on the population and ethnicity, a mutation in the gap junction protein beta 2 gene is the most common genetic cause of severe to profound autosomal recessive and non-syndromic hearing loss (GJB2). About half of all occurrences of autosomal recessive non-syndromic hearing loss in white people in Europe and the United States are caused by mutations in this gene.[30]

Clinical evaluation may reveal a syndromic aetiology in some cases. Symptoms such as pre-auricular pits and tags, branchial cysts or fistulae, or dystopia canthorum (the lateral displacement of the medial corners of the eyes, giving the appearance of a wider nasal bridge), heterochromia iridis, and pigmentary abnormalities may be linked to syndromes proven to cause hearing loss, which must also be considered. There have been over 400 of these syndromes identified. Genetic testing is available for a large number of diseases since the relevant genes have been identified.[30]

The most prevalent non-genetic cause of sensorineural hearing loss is congenital infection, with congenital cytomegalovirus (CMV) disease standing out. Compared to industrialised countries, where the frequency of congenital CMV disease is 0.58 percent, developing countries with a high maternal seroprevalence have a frequency of 1–6 percent. Viruses such as CMV can be spread through sexual contact or through contact with bodily fluids of children who are infected with the virus. The virus can be shed in urine, saliva, and blood. Congenital infection-induced hearing loss risk may be influenced by factors such as socioeconomic status (congenital CMV infection), accessibility of prevention techniques such as immunisation (congenital rubella), or hygiene practices (congenital toxoplasmosis). Congenital rubella infection is the most common cause of congenital hearing loss in nations without a rubella vaccination programme.[31]

Congenital hearing loss surveys, which divide causality among genetic and environmental variables, cover all of the aforementioned risk factors. However, in most investigations, a definitive reason for hearing loss could not be found in a significant number of youngsters.[32]

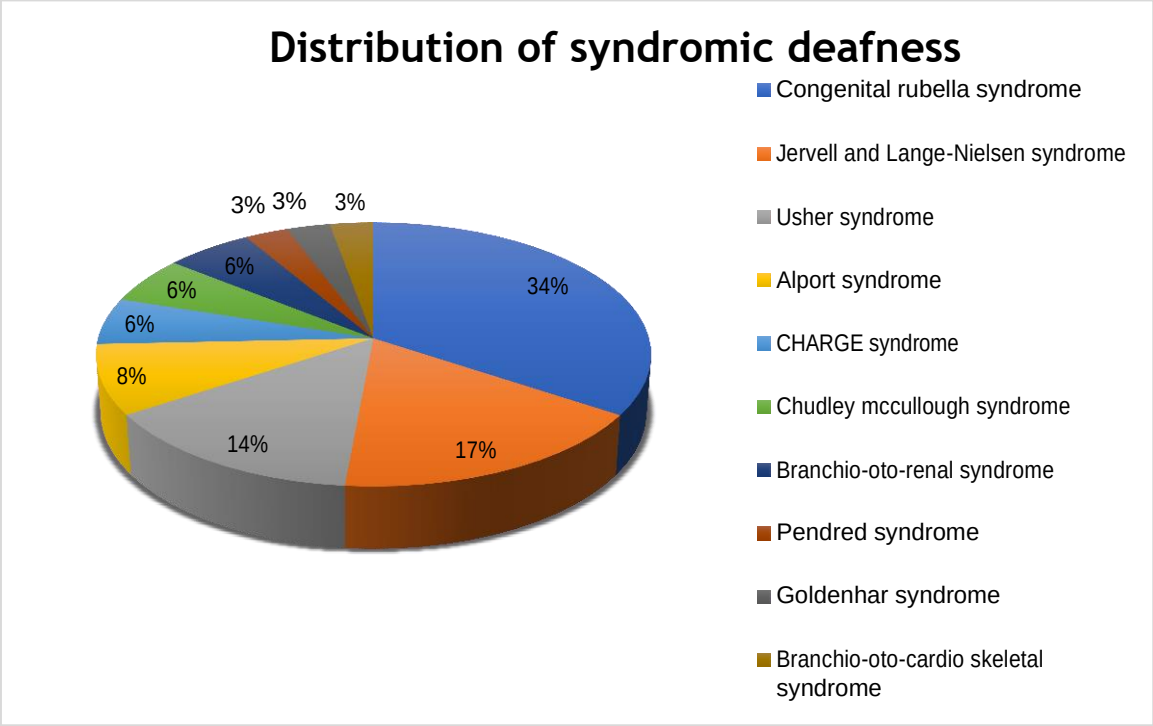


Figure 12. Syndromic hearing loss distribution.[32]

Pathophysiology.

There are two types of congenital hearing loss: hereditary and acquired. These two types have vastly different processes and pathology.[33]

Hearing loss that develops early in life due to a gene mutation.

Understanding normal auditory function as well as the pathophysiological mechanisms that might disturb it has improved tremendously as a result of research into hearing loss that has a genetic basis. Any part of the auditory system can be mutated genetically. While the majority of genes implicated in syndromic hearing loss are linked to a specific syndrome, non-syndromic hearing loss loci are traditionally termed with a prefix followed by an integer suffix: DFNA for autosomal dominant locus, DFNB for autosomal recessive loci, and DFNX for X-linked loci.[33]

Stria vascularis as well as endolymph homeostasis - inner ear

The stria vascularis, which is found on the cochlear duct's lateral wall and is critical for inner ear homeostasis. One unique fluid produced by this highly-specialized tissue is called endolymph and it is critical for auditory transduction because it bathes the inner ear's sensory hair cells in it. As a result of the endolymph's high potassium (K+) and low sodium (Na+) ion concentrations, as well as its high positive endoochlear potential (+80–100 mV), several channels, pumps, and gap junctions all work together to make it what it is. Border, intermediate, and basal cells make up the stria vascularis. The lateral wall's marginal cells face the endolymph, while the intermediate and basal cells below them connect to each other and to the fibrocytes of the supporting spiral

ligament (a thick periosteum that forms the cochlear duct's outer wall) via gap junctions. The gap junction protein beta 2 as well as 6 genes, GJB2 and GJB6, help transfer ions between cells and electrically couple the cells in this network of gap junctions. 29 Many people with severe to profound autosomal recessive congenital hearing loss have mutations in GJB2, which is the most prevalent gene to be affected.[34]

Two tight junction barriers limit the passive passage of ions in the intrastrial space (the intermediary cell layer and capillaries), which is isolated from the marginal cell layer as well as the basal cell layer. In humans, the claudins and MARVEL domain-containing protein 2 (commonly known as tricellulin) encoded by CLDN14 and MARVELD2 cause autosomal recessive non-syndromic hearing loss since they are both components of the tight junction. thirty-one In addition to the EAST syndrome (which causes epilepsy, ataxia, sensorineural hearing loss, and renal tubulopathy) and Bartter syndrome (which causes renal tubulopathy as well as hearing loss), mutations in a handful of other genes expressed inside the stria vascularis that are crucial for ionic homeostasis in the endolymph cause a variety of syndromic and non-syndromic forms of hearing loss (for example, DFNB73).[35]

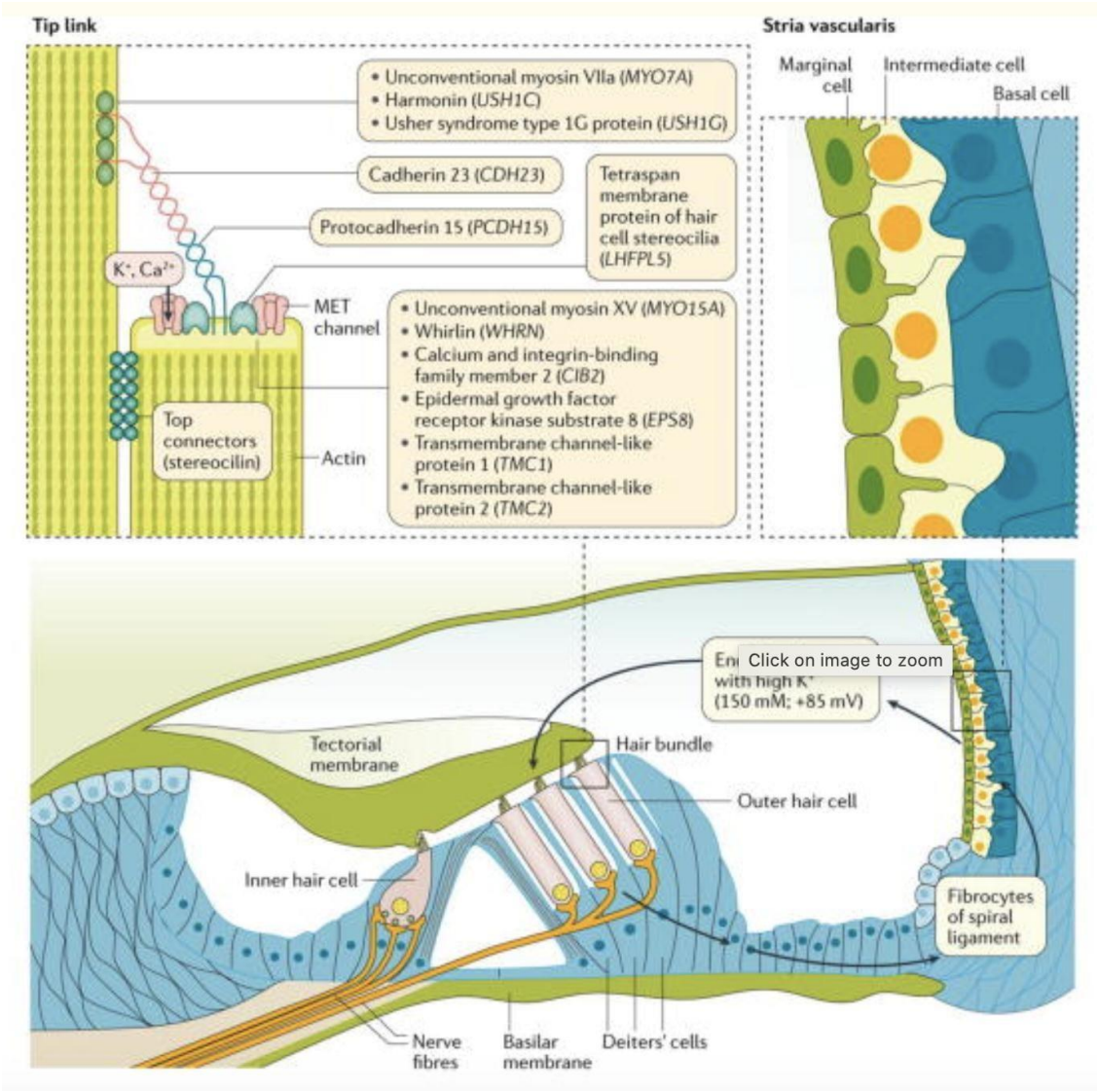


Figure 13. Stria vascularis – inner ear homeostasis.[1]

Inner ear homeostasis necessitates proper pH balance in the endolymph. If you have a genetic mutation for an enzyme or pump that regulates the endolymph's pH and ionic composition, you could develop Pendred syndrome (which includes hearing loss and goitre), distal renal tubular acidosis with deafness, or non-syndromic early-onset severe to profound hearing loss, which

is linked to enlarged vestibular aqueducts. About one-third of people with enlarged vestibular aqueducts have variable hearing loss, whether the condition is syndromic (e.g. Pendred syndrome) or not. Endolymphatic hydrops (large buildup of endolymph in the cochlea and vestibular system) may be responsible for these variations, although the specific process causing hearing loss and rapid dips in hearing is unknown.[35]

Stereocilia perform mechano-electrical transduction.

The inner and stereociliary bundle of inner as well as outer hair cells, the cells that transform mechanical stimuli into electrical activity, are affected by several additional types of hereditary hearing loss. Stair-like stereocilia on the apical surface of hair cells are actin-rich projections that are linked together by protein linkages to form a stereocilia ladder structure. Outside hair cells' stereocilia are embedded in tectorial membranes, which are formed of non-collagenous glycoproteins such alpha-tectorin, beta-tectorin, otogelin, and otolin-1. These glycoproteins are also found at the terminals of the outside hair cells' tallest stereocilia. Consequently, a shearing motion is caused by an elongated basilar membrane as it moves as a result of sound waves striking the hair cells.[36]

Stereocilial displacement physically opens mechano-electric transduction channels on the stereocilia's apical surface, and K+ flows through into sensory hair cells under an electrochemical gradient. After the K+ inflow depolarizes the hair cells, a series of processes occur, including activation of the auditory nerve fibres. The potassium voltage-gated channel subclass Q member 4 gene encodes channels that release K+ from the basolateral surface of hair cells (KCNQ4). Autosomal dominant non-syndromic progressive hearing loss is caused by KCNQ4 gene mutations, which are rather frequent.

Several proteins, including espin, encoded by the ESPN gene, cross-link longitudinal actin fibres within the stereocilium to increase their strength and rigidity. Due to the fact that it is autosomal recessive, hearing loss caused by ESPN mutations can be either non-syndromic (autosomal recessive), or progressive (autosomal dominant), with or without balance issues (lack of reflexes). 35 In the cell body, the actin filaments produce rootlets at the base of the stereocilia, where they are firmly packed to create rootlets. A non-syndromic recessive hearing loss is caused by a mutation in the cytoskeleton-associated TRIO as well as F-actin binding protein (TRIOBP), which prevents actin filaments from bundling together densely (DFNB28). 36 Tip connections connect the shorter stereocilium's apical surface to the lateral surface of its taller neighbouring cell, where an electron-dense anchor is made up of numerous interacting proteins crucial for hearing at the other end of the stereocilium. At

the base of inner hair cells lies the ribbon synapses, a specific type of synapses with thousands of vesicles containing the glutamate neurotransmitter (released by calcium-dependent exocytosis of the vesicles). There are thousands of vesicles in this structure that can be released quickly and continuously to accurately encode speech perception's requirements for sound strength and temporal accuracy. Hearing loss occurs in Otof knockout mice because inner hair cells do not exocytose, which is required for otoferlin (encoded by OTOF) to participate in this process. People with Auditory Neuropathy Spectrum Disorder have OTOF gene mutations.[36]

Acquired congenital hearing loss.

Acquired congenital hearing loss can be caused by a variety of infectious agents. Because congenital Zika virus infection is now known to be a major cause of foetal harm and disabilities in newborns, researchers have discovered that this virus can also cause congenital hearing loss in children. Seven percent of newborns with microcephaly and Zika virus infection in Brazil were found to have sensorineural hearing loss, according to the study.[37]

Cytomegalovirus.

CMV is the most prevalent prenatal infectious agent that can be debilitating and belongs to the Herpesviridiae family. CMV shedding persists

for months after infection, especially in the saliva or urine of young children, and poses an exposure risk for pregnant women because viral DNA can be detected in these body fluids for months after infection (CMV shedding). With a 32% chance of vertical transmission during pregnancy, the risk of congenital CMV disease is highest after primary infection. During reactivation or reinfection, however, the probability of vertical transmission is only 1.4% in seropositive mothers Given that women of reproductive age in developed nations have a seropositivity rate of 50% for CMV, one in every 100–200 live infants will be infected with CMV congenitally.[38]

It's unknown what causes sensorineural hearing loss in children who have had congenital CMV infection. Viral antigens have been discovered in the spiral, organ of Corti, scala media, and Reissner's membrane, as well as in the temporal bone where studies have shown inflammation and edoema of the cochlea and spiral ganglion. Infection and cytolysis of labyrinth components, including hair cells, have been demonstrated in mouse models. After an experimental challenge in another CMV-infected mouse model, hearing loss was linked to shrinkage of spiral ganglion neurons. As well as evidence supporting viral infection's direct cytolytic effect, there is also evidence of immunological damage, which is mediated by both the host immune response and the production of viral genes encoding pro-inflammatory chemokines The virus' virulence and the mother's, foetus', and placenta's immune responses all

play a role. About 10% of CMV-infected infants are symptomatic at birth, and the neonate's risk of symptoms is highest when the mother is infected around conception or throughout the first trimester of pregnancy.[38]

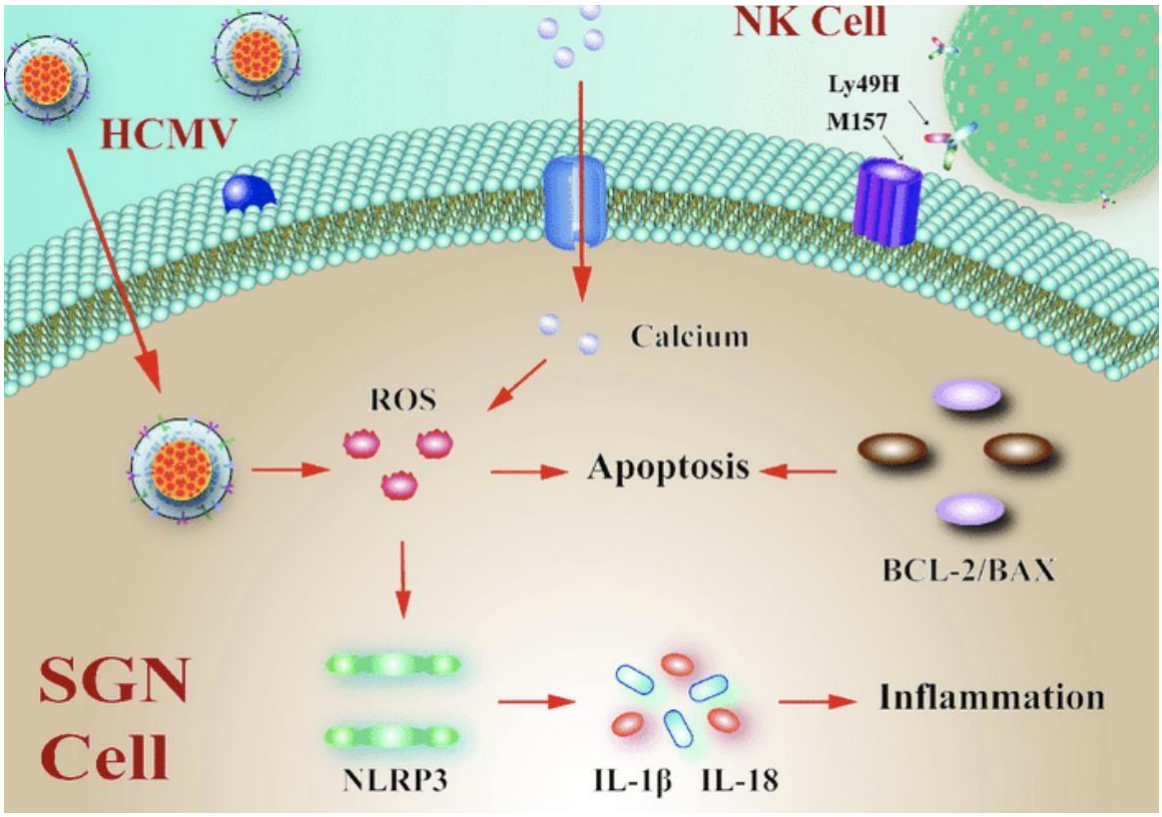


Figure 14. Human cytomegalovirus (HCMV) infection mechanisms and routes in spiral ganglion neurons (SGN).[39]

Rubella.

Rubella virus-infected neonates are more likely to be born at term, although their birth weight is often lower than that of uninfected newborns of the same gestational age. Hearing loss is the most common side effect of a congenital rubella infection. Cataracts, hepato-splenomegaly, and microcephaly

are also common findings. This trifecta of symptoms includes hearing loss, cataracts, and congenital heart disease but can vary in severity depending on when the foetus was exposed to rubella virus. Many rubella-related problems (including congenital heart disease including hearing loss) were found in nine newborns who had been exposed to the virus by the 11th gestational week in a prospective investigation of pregnant mothers with confirmed rubella infection. Only one complication was observed in 35% (9 out of 26) of children born to mothers who were infected between 13 and 16 weeks of pregnancy: hearing loss. It's possible that hearing loss linked to congenital rubella syndrome won't manifest until a child is an adult. 51 It's still unclear how rubella infection causes hearing loss, however the virus can cause damage to the inner ear, cell death in the organ of Corti, and changes in the endolymph's composition after strial injury.[40]

Diagnosis, early detection and prevention.

Targeted screening was used exclusively in the past century for infants deemed to be at high risk of hearing loss (that is, infants admitted to the neonatal ICU as well as those with a family history of hearing impairment or craniofacial anomalies). Because data shows that detecting hearing loss in children as early as possible is critical to their development, nationwide neonatal hearing screening programmes have now been adopted. It is currently common practice in affluent countries to conduct a two-phase neonatal hearing

screening programme (that is, two electrophysiological measurements done sequentially).[1]

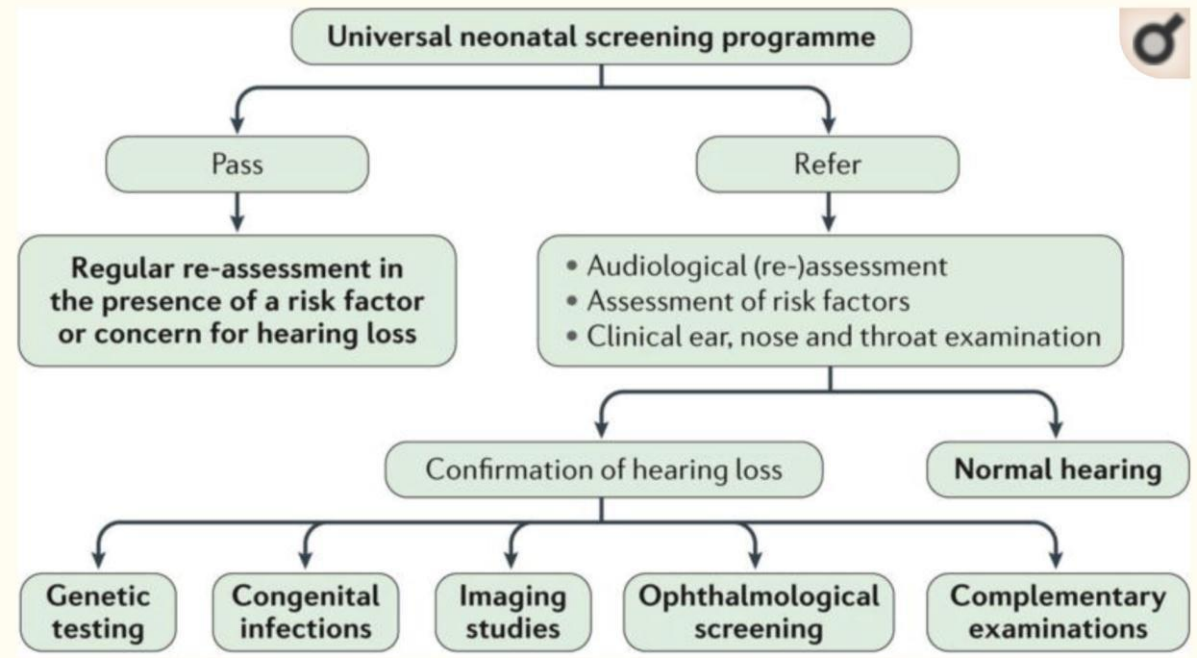


Figure 15. Hearing assessment.[1]

Assessment of hearing

Most screening methods include measuring oto-acoustic emissions twice, measuring oto-acoustic emissions as well as automated auditory brain stem responses, or measuring automated auditory brain stem responses twice. Children who do not pass the test should be evaluated by an audiologist and a doctor to determine if they have a hearing loss, preferably before the age of

three months. Even if you pass the neonatal hearing test, you may still have a congenital hearing loss that is progressive, late-onset, and less severe (30–40 dB hearing loss), which is missed by most newborn hearing screening programmes. Hearing loss can occur later in life if a child passes a neonatal hearing screening but has risk factors for it or if their parents express concern about their child's hearing abilities.

In cases when hearing loss has been detected during neonatal screening, a full audiometric evaluation is needed to determine the severity and location of the hearing loss (unilateral or bilateral). In order to determine the degree of hearing loss, the better-hearing ear should be used to average out frequencies of 500 to 4,0Hz. Hearing loss can be mild (20–40 dB), moderate (41–70 dB), severe (71–95 dB), or profound (> 95 dB) based on its laterality and severity.

Electrophysiological (oto-acoustic emissions estimate the function of outer hair cells and auditory brain stem responses estimate the function of inner hair cells as well as the integrity of hearing pathways) and behavioural (audiometry) tests are included in the audiometric assessment of the auditory nerves. A variety of tests are used in clinical settings to determine whether or not the ear is functioning properly in order to cross-check the results of physiological and behavioural measurements.[41]

Oto-acoustic emissions.

Sounds produced by the movement of outer hair cells in response to auditory stimuli are known as oto-acoustic emissions. A click stimulus causes transient evoked oto-acoustic emissions. Transient induced oto-acoustic emissions responses show an oscillatory sound pressure waveform, which corresponds to the tympanic membrane (eardrum) moving back and forth due to variations in fluid pressure in the cochlea. A tiny probe inserted into the external auditory canal can monitor transient evoked oto-acoustic emissions responses, which can provide a frequency-specific indication of cochlear health. Rather than being used in isolation to determine whether or not someone has normal hearing, oto-acoustic emissions should be interpreted in conjunction with other tests such as tympanometry, otoscopy, and assessment of the auditory brain stem responses.[42]

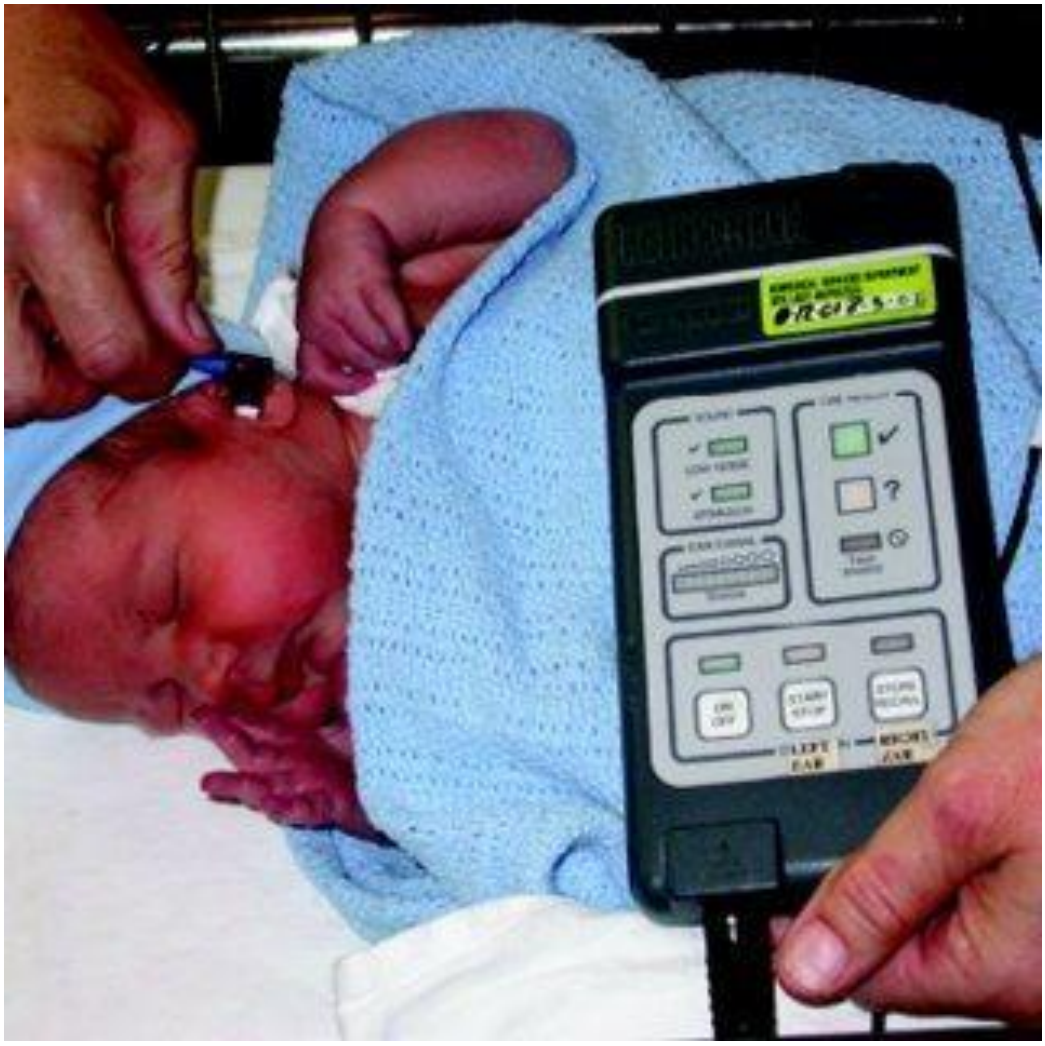


Figure 16. OAE screening.[43]

Auditory brain stem responses or auditory steady state responses.

These reactions originate in the auditory brain stem, where auditory stimuli evoke electrical potentials that represent neuronal activity along the auditory pathway. Computer-averaging techniques are used to record the activity from scalp electrodes. When evaluating hearing in babies and children of all ages, click- or tone burst-triggered auditory brain stem responses are the

gold standard. For higher frequencies (2,000–4,000 Hz), the thresholds measured are frequently within 10 decibels of the behavioural auditory thresholds.

Tonal stimuli modulated by AM/FM generate steady-state auditory responses. To collect threshold data in children with substantial hearing loss (>90 dB), auditory steady state responses are more beneficial than click stimuli because they provide greater average sound pressure levels. Lack of auditory steady state response thresholds suggests no useable hearing and predicts poor hearing aid efficiency. Most of the time, the difference between the thresholds determined with the two approaches is less than 10 decibels (dB) for all forms of hearing loss. Click auditory brain stem responses as well as average auditory steady state responses are closely related for all types of hearing loss. To assess bone conductive hearing thresholds and separate conductive hearing loss versus sensorineural hearing loss, auditory steady state responses are also relevant.[44]

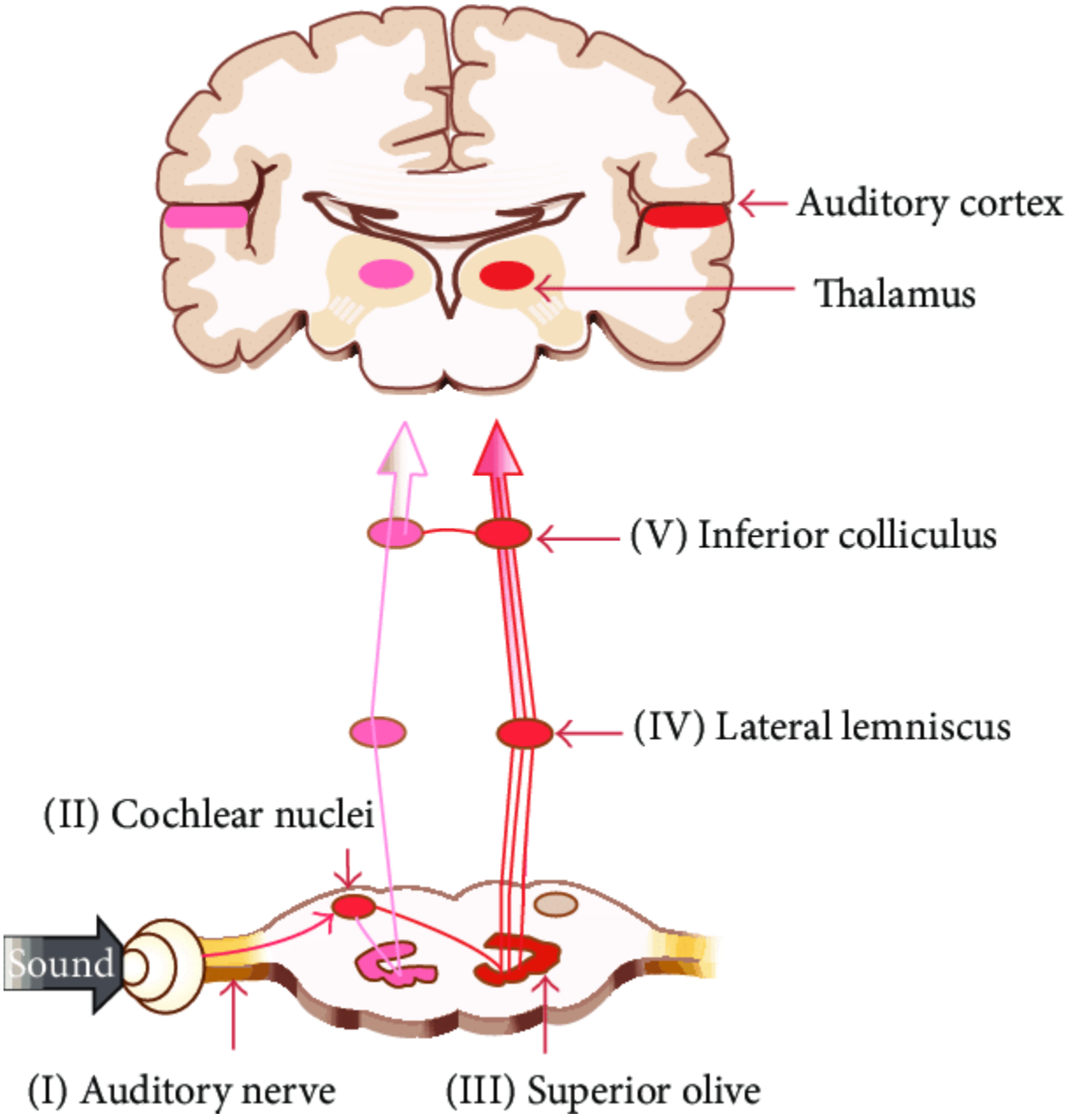


Figure 17. ABR neural generators.[45]

Automated auditory brainstem response (AABR).

This test covers the entire auditory system from the ear all the way down to the brainstem and beyond (including middle ear, inner ear as well as eighth nerve). Electrodes are put on the forehead, nape of neck, and shoulder during AABR procedure. When using AABR screening, a click stimulus is delivered to each of a child's ears at the same volume level. Responses from children with normal hearing are compared to those from a sample of typically developing children. if the answers match, the youngster passes, if not, he or she has hearing loss. After delivery, AABR screening can be conducted with a strict statistical pass criterion that removes interpretation bias. For infants older than 34 weeks of gestation and younger than 6 months, the AABR is a useful screening tool.

The automated screener employs a response algorithm to generate a PASS or REFER result based on the averaged responses to many stimulus presentations.

The threshold for passing is set at 35 dB. Babies are sedated during testing to reduce muscular activity's effect on the results. Within the first 10 milliseconds of a human heartbeat, seven waves are generated. It is possible to achieve Waves I, III, and V consistently in all age categories. Waves II and IV are much less frequent occurrences than Wave I. Each wave's latency (the time it takes for the wave peak to appear after the stimulus first appears) increases when

stimulus intensity or loudness drops, while wave amplitude decreases. Although the specific anatomic site of wave origin is still a matter of debate, the following parts are thought to be responsible.[46]

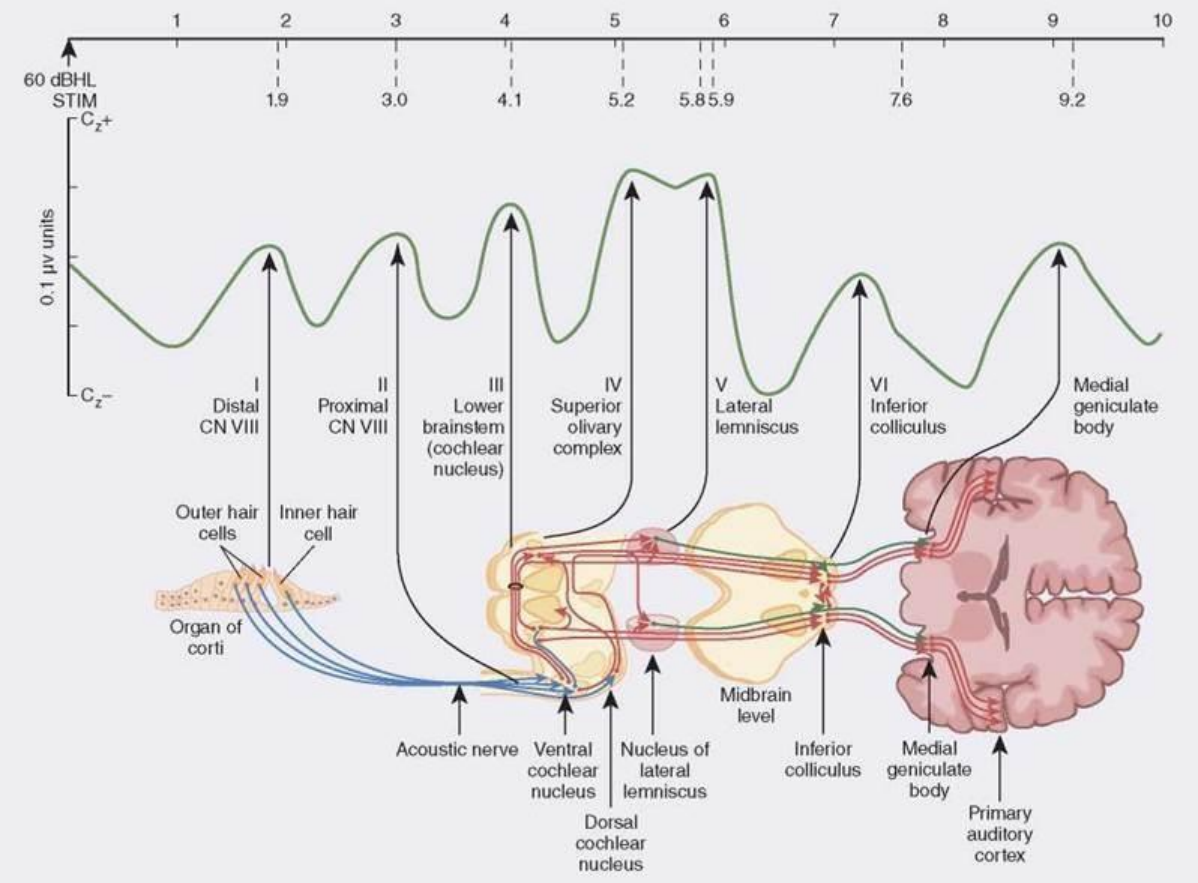


Figure 18. AABR wave and corresponding structure.[46]

Audiometry.

Children aged 6–24 months can have their hearing tested using visual re-enforcement audiometry. A new sound source will cause an orienting reaction towards the sound in children who have adequate hearing. Audiologists who are

proficient in their field can produce dependable results. Children between the ages of 2 and 4 can benefit from the usage of play audiometry since it trains them to respond to auditory stimuli through play. A bone-conduction transducer (such as an earphone) or air-conduction transducer (such as an earphone) is commonly employed after the age of four for conventional audiometry (Figure 3). First, the auditory system as a whole is tested, while second, the skull is vibrated to activate the cochlea directly, skipping the outer and middle ears entirely Sensorineural hearing loss and conductive hearing loss can be distinguished using air conduction and bone conduction thresholds, which are typically measured at octave frequencies between 250 and 8,000 Hz.[47]

Investigation into aetiology

The search for an inherent aetiological diagnosis becomes necessary if a diagnosis of bilateral persistent congenital hearing loss is made. Screening for congenital infections, imaging, and genetic testing are all options. GJB2 and GJB6 mutations are the most common first-line genetic tests. Other tests based on clinical findings are used in addition to ophthalmologic screening to look for ocular signs of congenital infection or specifics of the syndrome, kidney ultrasonography to look for congenital malformations, and an electrocardiogram to diagnose long-QT syndrome (as seen in Jervell Lange-Nielsen). The aetiological work-up can be reduced in unilateral hearing loss to a

comprehensive clinical examination for a syndromic etiology of hearing loss, study of probable congenital infections and inner ear imaging.[48]

Even though next-generation DNA sequencing technologies and extensive genetic testing employing gene panels are included in several guidelines for aetiological workups for congenital hearing loss, they are not widely used. Using targeted genomic amplification with massively parallel DNA sequencing for comprehensive genetic testing has modified the diagnostic algorithm, and the requirement for additional tests may be determined in the future by the suspected diagnosis as well as the results of the comprehensive genetic testing. New guidelines from the American College of Medical Genetics and Genomics (ACMG) encourage using gene panels because of the value they provide. A multidisciplinary approach can identify an etiological cause in around half of the patients with bilateral congenital hearing loss by using a sequential diagnostic strategy based on the degree of hearing loss.[48]

Genetic testing and analysis.

A pedigree is always the first step in a genetic diagnostic. A clear understanding of inheritance mode is critical since it helps to narrow the field of possible causal genes. Environmental factors of hearing loss should be examined when looking at a patient with no other affected family members.[49]

Non-syndromic hearing loss DNA testing is difficult since there are so many different genes that could be involved and so few diagnostic indications based on phenotype. As a result, diagnostic use of genes associated to hearing loss has behind scientific advancement for a long time. When it came to diagnosing non-syndromic hearing loss, diagnostic labs around the world focused on a few key genes, notably the most often altered one, GJB2. Only 10–20% of patients having non-syndromic hearing loss were found to have the disease-causing gene using this method. The simultaneous examination of many genes has been made possible by technological breakthroughs such as next-generation DNA sequencing. There are currently multiple labs that offer low-cost genetic testing for broad panels of genes connected to both syndromic and non-syndromic forms of hearing loss using these new approaches." Once hearing loss is confirmed through audiometry, comprehensive genetic testing currently offers the greatest diagnosis rate of any test.[49]

Acquired congenital hearing loss diagnostics.

Congenital CMV infection is the primary non-genetic etiology of congenital hearing loss in developed nations, hence any newborn showing indications of infection should be checked for CMV infection. Even healthy and apparently unaffected children with hearing loss should be tested for this virus. Microcephaly, jaundice, and intrauterine growth retardation are just a few of the signs and symptoms of CMV infection. About 30% of children with

symptoms of CMV infection will also have sensorineural hearing loss. A diagnostic examination is necessary in these situations. Congenital CMV infection can be diagnosed prenatally using CMV PCR on amniotic fluid (the positive predictive value is nearly 100 percent). As soon as a baby is born, samples of the newborn's blood, urine, saliva, or throat should be taken (within 3 weeks of delivery, since viral shedding after such time point may show postnatally acquired infection rather than congenital infection) and analyzed. Only in retrospect, utilizing saved dried newborn blood spots as the basis of template for PCR-based diagnosis can congenital CMV infection be established in children receiving assessment of the aetiology of sensorineuroal hearing loss after the age of three weeks. An early blood test for metabolic, endocrine, and other diseases is performed frequently throughout the developed world within the first week of life. All of the blood that's left is collected as dried blood clots. Local storage policies determine the availability of these samples, and they also have a lower diagnostic sensitivity than saliva or urine samples collected in "real time," making them less useful for diagnosis.[50]

Neuroimaging (cerebral ultrasonography as well as MRI), visual function testing, and hearing testing are all required in cases where congenital CMV infection is suspected. However, congenital CMV infection is practically asymptomatic in 90% of babies. Although these children have fewer neurodevelopmental issues than those who are born with the condition, 10% of

them will still suffer from significant sensorineural hearing loss during their lifetime.[50]

Only 12 months after birth can a conclusive clinical diagnosis of congenital rubella infection be obtained. There are four criteria for diagnosing rubella infection: a positive anti-rubella IgM titre (conceivably measured with enzyme immunoassays), a significant rise in anti-rubella IgG titer 2–3 weeks following acute phase of the infection or high titers persisting far beyond what is expected from passive maternal antibody transfer, rubella virus isolation in cultures from throat, nasal, blood or cerebrospinal fluid specimens, or the detection of the virus in these samples (from congenital cataracts, isolated from the lens).[50]

Management.

Pathogen-associated hearing loss is currently treated non-surgically with a focus on two important areas of intervention: specialized antimicrobial medicines and anti-inflammatory medications to diminish the host's immunological reaction to infection and, as a result, the cochlea's damage. Infectious disease-related hearing loss may be better understood if new therapeutics such free radical scavengers, anti-oxidants, and nanoparticle systems are developed. In some circumstances, surgery might help close an air-bone gap. Special education with sign language are two examples of non-medical help.[51]

Hearing rehabilitation

There are various methods for restoring hearing, including implantable and non-implantable technologies, such as conventional hearing aids and cochlear implants.[51]

Hearing aids.

Children with hearing loss can benefit from hearing aids designed specifically for them. A skilled paediatric audiologist should fit and programme hearing aids for infants and children. Hearing aids for hearing-impaired youngsters can be BTE (behind the ear) or ITE (in the ear). Hearing aids using ITE technology are entirely hidden inside the ear canal. Older children may benefit from ITE hearing aids, but newborns and young children should avoid them. The size and form of a child's ears alter as he or she grows older. Hearing aids using BTE technology are more reliable and last longer. Infants as young as two months old may be equipped with hearing aids.[52]

Cochlear implants.

When used improperly, hearing aids might be ineffective. The use of cochlear implants may be an option for those who suffer from severe sensorineural hearing loss. An electronic device, a cochlear implant is. There are two parts: an exterior one and an inside one that must be surgically inserted.

Electrical impulses are generated externally by picking up sound and converting it into electronic signals that can be transmitted internally. Auditory nerve fibres receive impulses from the receiver and transmit them directly to the brain.

Pneumococcal meningitis, a major side effect of cochlear implants, occurs at an alarmingly high rate. Pneumococcal vaccination is required for all children receiving a cochlear implant.[53]

Workings of a cochlear implant

- The microphone picks up sound.
- A precise pattern of electrical impulses is generated from the "coded" signal.
- The coil transmits the impulses to the implant through the skin.
- The electrodes in the cochlea receive a series of electrical impulses from the implant.
- An electrical impulse is picked up by the Auditory nerve, which transfers it to the brain.
- These impulses are interpreted as sounds by the brain.[53]

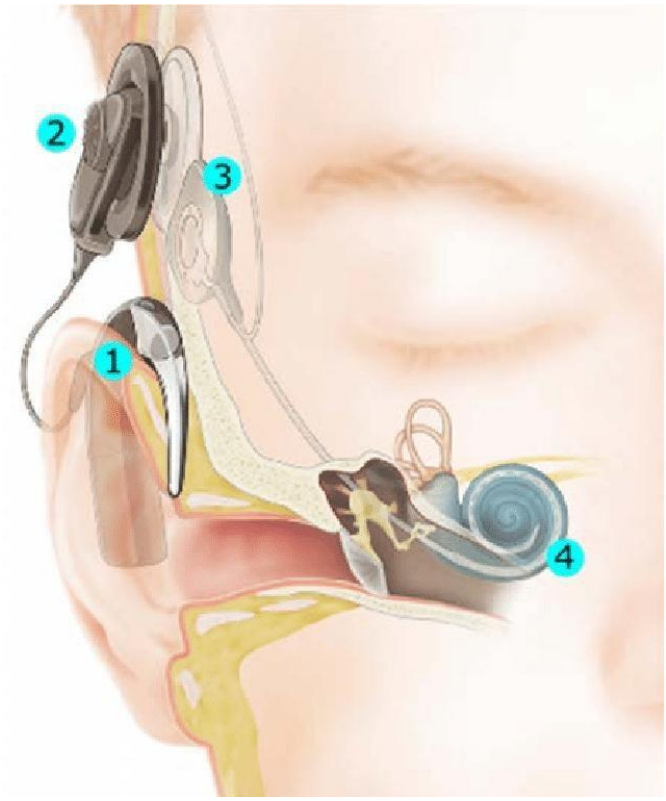


Figure 19. Cochlear implant.[54]

Kennedy et al. in 1991 in 370 infants compared Evoked Otoacoustic Emissions and Standard and Automated Analysis of Auditory Brainstem Responses. They observed that tests performed by an automated OAE device were swift (median time of 12-55 mins) and non-intrusive (no scalp electrodes were used). An automated OAE failed 30 percent of the time with a stimulus 35-36dB over normal hearing threshold, whereas an ABR failed just 3 percent of the time, while an automated ABR failed 2 to 7 percent of the time. In terms of detecting a hearing impairment, the automated OAE test was the most accurate.[55]

Norton et al. in 2000 studied a total of 4911 infants, including 4478 newborns who graduated from neonatal intensive care units, 353 healthy babies

with one or more predictors for hearing loss (Joint Committee on Infant Hearing, 1994), and 80 healthy babies without risk factors who failed one or more neonatal tests, were targeted as a potential subject pool for the research. Using TEOAEs and DPOAE response to two sensory inputs (L1 = L2 = 75 dB SPL as well as L1 = 65 dB SPL L2 = 50 dB SPL), and also ABR in response to a click of 30 dB nHL. As a whole, referral rates were low for all three neonatal hearing screening tests, especially if referral seeking follow-up were limited to those in which stopping requirements were not satisfied in both ears The sensitivity of each test rose in direct proportion to the severity of the hearing loss being assessed.[56]

Barker et al. in 2000 noted false-positive rates with DPOAE screening range from 11% to 35%. As a result of this variation in pass and refer rates when different criteria are employed, newborn hearing screening programmes need to be standardised and further compared to determine appropriate pass criteria.[57]

Meier et al. in 2004 noted that OAE measurements were faster and easier to relate the subject to than AABR measures. The Algo 3 recorded AABR at a pass rate of 98 percent, whereas the Beraphone MB11 recorded AABR at a rate of 92 percent, but the differences between the two were not statistically significant.[58]

Johnson et al. in 2005 observed that A-ABR hearing screening have detected about 23% of infants with PHL at age 9 months if all were screened

with the 2-stage OAE/A-ABR newborn auditory screening procedure presently employed in many hospitals. An important reason why infants with mild hearing loss are missed during A-ABR screening is because of the technology used.[59]

Xu et al. in 2005 used DPOAE as well as automated ABR (AABR) screening at the age of a few days, followed by otoscopy, measurement of middle ear impedance, and click induced ABR at the age of two months. Babies with abnormal DPOAE or DPOAE-AABR screening results were referred for further testing. 24.5% of the 200 NICU babies were identified after a single DPOAE screening session. The DPOAE-AABR screening detected nine of them (4.5 percent). Six NICU babies (about 3 percent) showed substantial hearing loss assessed on click-evoked ABR when they were two months old. No hearing loss was identified in any of the infants indicated by DPOAE but who passed the AABR hearing test.[60]

Lin et al. in 2007 studied effective hearing screening programme using one-step TEOAE, two-step TEOAE as well as AABR, as well as one-step AABR programmes, which drastically reduced referral rates from 5.8 percent to 1.6 percent and then 0.8%. There was no discernible variation in the accuracy of their congenital hearing loss recognition rates.[61]

Vignesh et al. in 2015 in their study found reduced referral rates in newborn screening programmes by utilising a two-step approach, particularly

AABR and DPOAE at the beginning of testing. They also observed that, AABR reduces the number of false-positive results, enhancing the screening program's overall efficiency.[62]

Dumanch et al. in 2017 in a retrospective data review found that ninety-six percent of children with risk factors had normal hearing by age three; one percent had congenital hearing loss; and three percent had persistent hearing loss by the age of three; Neurodegenerative illnesses, syndromes, and congenital infections were the leading causes of congenital hearing loss in children. Children with congenital CMV, syndromes, and craniofacial deformities had the highest risk of acquiring persistent postnatal hearing loss.[63]

Howell et al. in 2019 in their study, an analysis of children in Virginia who had universal newborn hearing screening (UNHS) from 2010 to 2014, indicated that neonatal indicators (69 percent), craniofacial deformities (30 percent), risk of HL syndromes (14 percent), and family history were the most common UHL risk factors (14 percent). Neonatal indications (49%) and family history (27%), risk of HL syndromes (19%), and craniofacial deformities were the most common risk factors in BHL (16 percent). Positive family history was associated with an increased risk of BHL in children, while cranial abnormalities were associated with an increased risk of UHL in those same children (P .001). There was a high rate of neonatal indicators in UHL and BHL populations. Craniofacial malformations were substantially more common in UHL children, while hearing loss ran in the family for BHL children.[64]

MATERIALS AND METHODS

STUDY DESIGN

Cross sectional study

SAMPLE SIZE CALCULATION

From the previous study, the prevalence of hearing loss detected by OAE among the high-risk infant was 6%

Considering 6% as prevalence, assuming 95% confidence with 5% allowable error, the sample size is calculated by the formula N= 4pq/E2 where p is prevalence, q is 1-p, and E is allowable error of P.

$$N = \frac{4\times0.0.06\times (1\text{-}0.0.06)}{0.05\times0.05}$$

$$= \frac{4\times0.06\times0.94}{0.0025}$$

$$= 90.52$$

To account for non-response rate (about 10%) were added to the sample size

90+9= 99.5

Thus, a total of 100 participants will be included in the study.

SAMPLING METHOD

Simple random sampling without replacement will be done to achieve the estimated sample size within the study duration.

PLACE OF STUDY

Government Kilpauk medical college and hospital, Chennai 10.

DURATION OF STUDY

1 Year.

INCLUSION CRITERIA

- Infants with atleast one of the following high-risk factor will be taken into the study.
- High risk criteria
- Parental or caregiver concern regarding hearing, speech, language, and/or developmental delay.
- Family history of congenital or delayed onset childhood sensorineural hearing loss
- Maternal infections-toxoplasmosis, syphilis, rubella, cytomegalovirus, herpes Craniofacial abnormalities
- Birth weight <1500g
- Hyperbilirubinaemia at a level exceeding indication for exchange transfusion
- Ototoxic drugs (aminoglycosides) during NICU / PUCU admission
- Bacterial meningitis

- Severe respiratory depression at birth (birth asphyxia)
- Stigmata or other findings associated with a syndrome known to include
- Sensorineural hearing loss (e.g. Waardenburgs or Ushers syndromes)

EXCLUSION CRITERIA

- High risk infants whose parents do not give consent.
- Infants on ventilator who are severely ill.

METHODOLOGY

The present study was conducted in the Department of Otorhinolaryngology, Kilpauk medical college and hospital, Chennai from September 2020 to September 2021.The study was done on High risk infants who are on followup attending the pediatric OPD in GOVT KILPAUK Medical College, and referred to our department of ENT and Head and Neck surgery for hearing Assessment. High risk infants referred to the department of ENT between September 2020 to September 2021 were evaluated for hearing loss by DPOAE and AABR. The case history and clinical examination including otoscopy will be done for all cases. After getting informed and written consent from parents' infants are subjected to screening simultaneously with both DPOAE and AABR.

STATISTICAL ANALYSIS

The quantitative data will be expressed in mean and SD. Qualitative data will be expressed in proportions. P value less than 0.05 will be considered as significant.

RESULTS

During this study,100 high risk babies were subjected to OAE testing.

Table 1: Descriptive data variables of the study participants (N=100)

Slno	Variable	Minimum	Maximum	Mean	Std. Deviation
1	AGE (MONTHS)	1	12	6.89	3.55
2	GESTATIONAL AGE AT BIRTH	38	42	39.91	1.33
3	BIRTH WEIGHT (GRAMS)	901	3492	2422.85	707.01
4	LENGTH	44.06	50.68	48.49	1.48
5	HEAD CIRCUMFERENCE	29.85	34.00	32.92	1.19
6	CHEST CIRCUMFERENCE	26.62	32.92	30.85	1.58
7	APGAR 1 MIN	3	6	4.56	1.08
8	APGAR 5 MINS	3	7	5.89	1.12
9	PEAK LEVEL OF TOTAL BILIRUBIN	1.10	17.00	7.10	4.15

The age of the study group ranged between 1 months to 12 months. 42 babies(42%) were male and 58 babies(58%) were female. The gestational age of the study group ranged between 38 to 42 weeks. Birth weight varied between 901g and 3492g.

Total babies screened initially by OAE = 100

Total babies who passed screening by OAE = 83 babies on right ear and 92 babies on left ear

Total babies who failed screening by OAE = 17 babies on right ear and 8 babies on left ear

Total babies subjected to AABR=100

Total babies who passed AABR=96 babies on right ear and 98 babies on left ear

Total babies who failed AABR=4 babies on right ear and 2 babies on left ear

Babies for whom OAE could not be done due to craniofacial malformation =2 babies

Four babies were diagnosed to have hearing impairment out of 100 high risk babies. The incidence rate is 4.0% which is similar to other studies done (2.5 -10%).

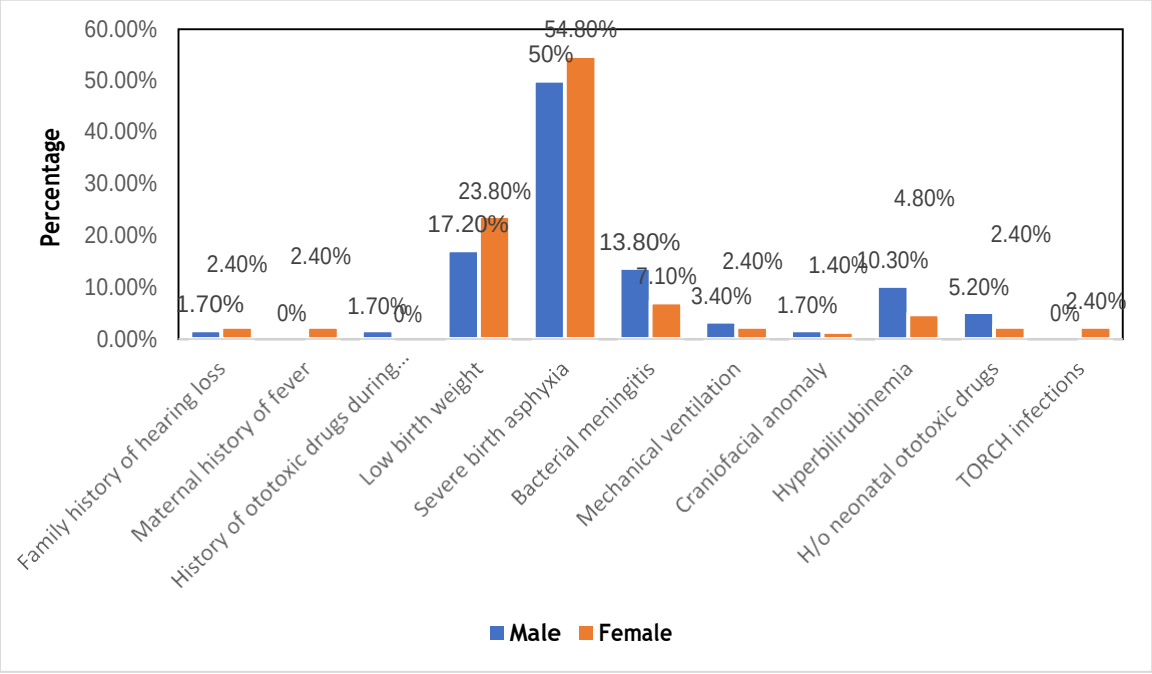
Table 2: Distribution of risk factors among the study participants (N=100)

Slno	Variable	Female (n=58)	Male (n=42)	X^2, (df), p
1	**Type of delivery**			
	AVD	1 (1.7)	1 (2.4)	0.350 (2)
	LSCS	10 (17.2)	9 (21.4)	0.840
	NVD	47 (81)	32 (76.2)	
2	**Family H/O hearing**			0.05 (1),
	Loss	1 (1.7)	1 (2.4)	0.817
	Present	57 (98.3)	41 (97.6)	
	Absent			
3	**Maternal H/O fever**			
	with rash	0	1 (2.4)	1.395 (1),
	Present	58 (100)	41 (97.6)	0.238
	Absent			
4	**H/O ototoxic drugs**			
	during pregnancy			0.731 (1)
	Present	1 (1.7)	0	0.392
	Absent	57 (98.3)	42 (100)	

5	**Low birth weight**			
	Present	10 (17.2)	10 (23.8)	0.657 (1)
	Absent	48 (82.8)	32 (76.2)	0.42
6	**Severe birth asphyxia**			
	Present	29 (50)	23 (54.8)	0.221 (1)
	Absent	29 (50)	19 (45.2)	0.64
7	**Bacterial meningitis**			
	Present	8 (13.8)	3 (7.1)	1.100 (1)
	Absent	50 (86.2)	39 (92.9)	0.29
8	**Mechanical ventilation**			
	Present	2 (3.4)	1 (2.4)	0.095 (1)
	Absent	56 (96.6)	41 (97.6)	0.76
9	**Craniofacial anomaly**			
	Present	1 (1.7)	1 (1.4)	0.054 (1)
	Absent	57 (98.3)	41 (97.6)	0.82
10	**Hyperbilirubinemia**			
	Present	6 (10.3)	2 (4.8)	1.032 (1)
	Absent	52 (89.7)	40 (95.2)	0.31

11	**H/O Neonatal ototoxic drugs** Present Absent	 3 (5.2) 55 (94.8)	 1 (2.4) 41 (97.6)	 0.494 (1)
12	**TORCH infections** Present Absent	 0 58 (100)	 1 (2.4) 41 (97.6)	 1.395 (1) 0.24

Figure 1: Distribution of risk factors among the study participants (N=100)



The distribution of risk factors were uniform in both the genders.

Table 3: Distribution of screening of severe birth asphyxia (n=52)

Slno	Babies	Screening by OAE	AABR
1	Total babies	52	52
2	Normal hearing	45	49
3	Impairment in Right ear	7	3
4	Impairment in both ears	5	2

Fifty two babies with severe birth asphyxia of which seven babies had hearing impairment in screening by OAE and three out of seven babies had hearing impairment in the AABR.

Table 4: Distribution of screening of low birth weight (n=20)

Slno	Babies	Screening by OAE	AABR
1	Total babies	20	20
2	Normal hearing	16	19
3	Impairment in Right ear	4	1
4	Impairment in both ears	1	0

Twenty babies with low birth weight of which four babies had hearing impairment in screening by OAE and one out of four baby had hearing impairment in the AABR.

Table 5: Distribution of screening of bacterial meningitis(n=11)

Slno	**Babies**	**First screening by OAE**	**AABR**
1	Total babies	11	11
2	Normal hearing	8	11
3	Impairment in Right ear	3	0
4	Impairment in both ears	1	0

Eleven babies with bacterial meningitis of which three babies had hearing impairment in first screening by OAE and all the eleven babies passed the AABR.

Table 6: Distribution of screening of mechanical ventilation (n=3)

Slno	Babies	First screening by OAE	AABR
1	Total babies	3	3
2	Normal hearing	2	3
3	Impairment in Right ear	1	0
4	Impairment in both ears	0	0

Three babies with mechanical ventilation was screened, one baby had hearing impairment in first screening by OAE and that three babies passed the AABR.

Table 7: Distribution of screening of craniofacial anomaly(n=2)

Slno	Babies	First screening by OAE
1	Total babies	2
2	Normal hearing	1
3	Impairment in Right ear	1
4	Impairment in both ears	0

Two of the babies were born with craniofacial malformation. OAE could not be performed on one baby due to atresia of the auditory canal. The other infant passed the OAE's initial screening.

Table 8: Distribution of screening of maternal history of fever with rash (n=1)

Slno	Babies	First screening by OAE
1	Total babies	1
2	Normal hearing	1
3	Impairment in Right ear	0
4	Impairment in both ears	0

One baby with maternal history of fever with rashes was screened and the baby passed the first screening by OAE.

Table 9: Distribution of screening of H/o ototoxic drugs during pregnancy (n=1)

Slno	Babies	First screening by OAE
1	Total babies	1
2	Normal hearing	1
3	Impairment in Right ear	0
4	Impairment in both ears	0

One baby with history of ototoxic drugs during pregnancy was screened and the baby passed the first screening by OAE.

Table 10: Distribution of screening of hyperbilirubinemia (n=8)

Slno	Babies	First screening by OAE	AABR
1	Total babies	8	8
2	Normal hearing	6	8
3	Impairment in Right ear	2	0
4	Impairment in both ears	1	0

Eight babies screened for hyperbilirubinemia of which two babies had hearing impairment in first screening by OAE and the eight babies passed the AABR.

Table 11: Distribution of screening of H/o neonatal ototoxic drugs (n=4)

Slno	Babies	First screening by OAE
1	Total babies	4
2	Normal hearing	4
3	Impairment in Right ear	0
4	Impairment in both ears	0

Four babies with history of neonatal ototoxic drugs was screened and all the four babies passed the first screening by OAE.

Table 12: Distribution of screening of H/o seizures (n=3)

Slno	Babies	First screening by OAE	AABR
1	Total babies	3	3
2	Normal hearing	2	3
3	Impairment in Right ear	1	0
4	Impairment in both ears	1	0

Three babies screened for H/o seizures of which one baby had hearing impairment in first screening by OAE and three babies passed the AABR.

Table 13: Distribution of screening of H/o TORCH infections (n=1)

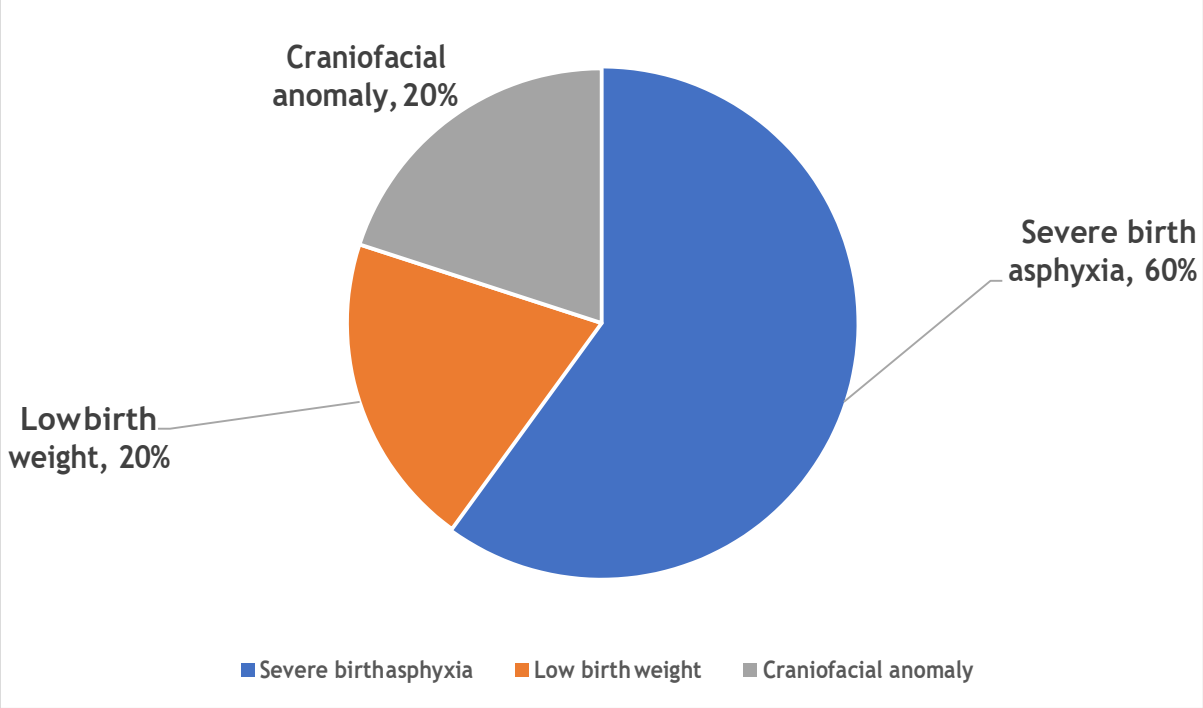
Slno	Babies	First screening by OAE
1	Total babies	1
2	Normal hearing	1
3	Impairment in Right ear	0
4	Impairment in both ears	0

One baby with history of torch infection was screened and the baby passed the first screening by OAE.

Table 14: Distribution of risk factors of failed AABR screening tests (n=5)

Slno	Risk factor	Number of cases AABR done	Hearing status
1	Severe birth asphyxia	3	Abnormal
2	Low birth weight	1	Abnormal
3	Craniofacial anomaly	1	Normal

Figure 2: Distribution of risk factors of failed AABR screening tests (n=5)

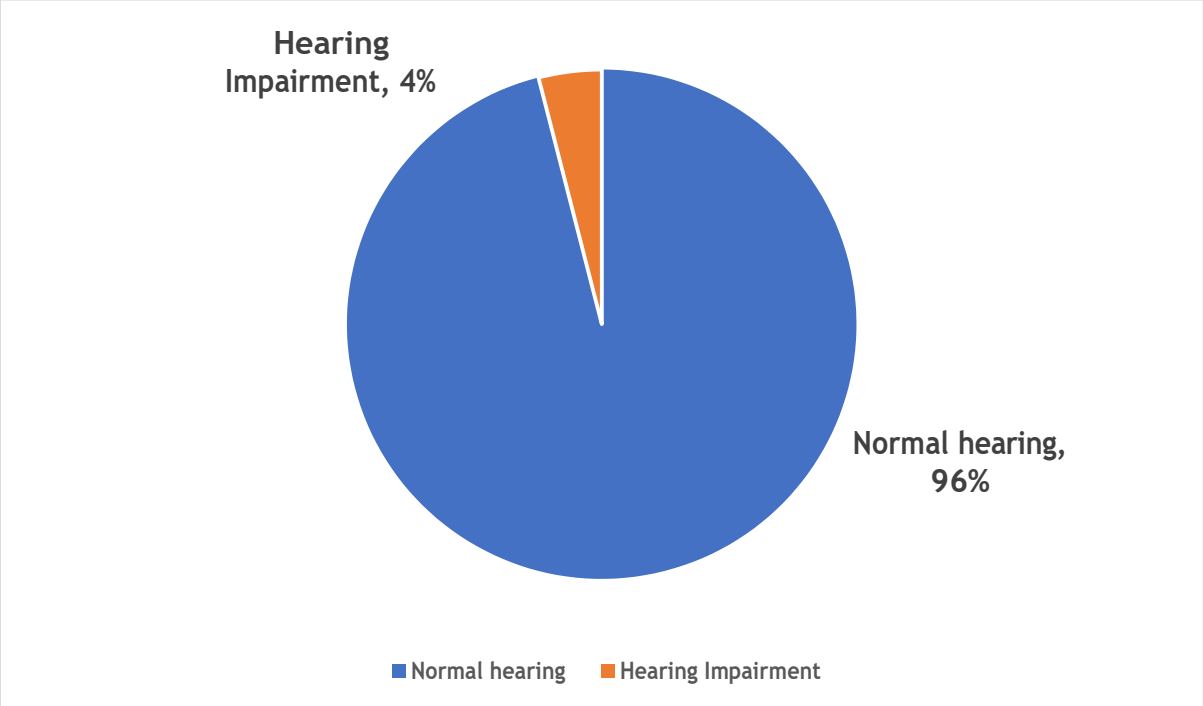


Four babies were subjected to AABR and it was abnormal in all four babies.

Table 15: Distribution of final outcome of screened infants (N=100)

Slno	Outcome	Number of cases	Percentage
1	Normal hearing	96	96%
2	Hearing Impairment	4	4%

Figure 3: Distribution of final outcome of screened infants (N=100)

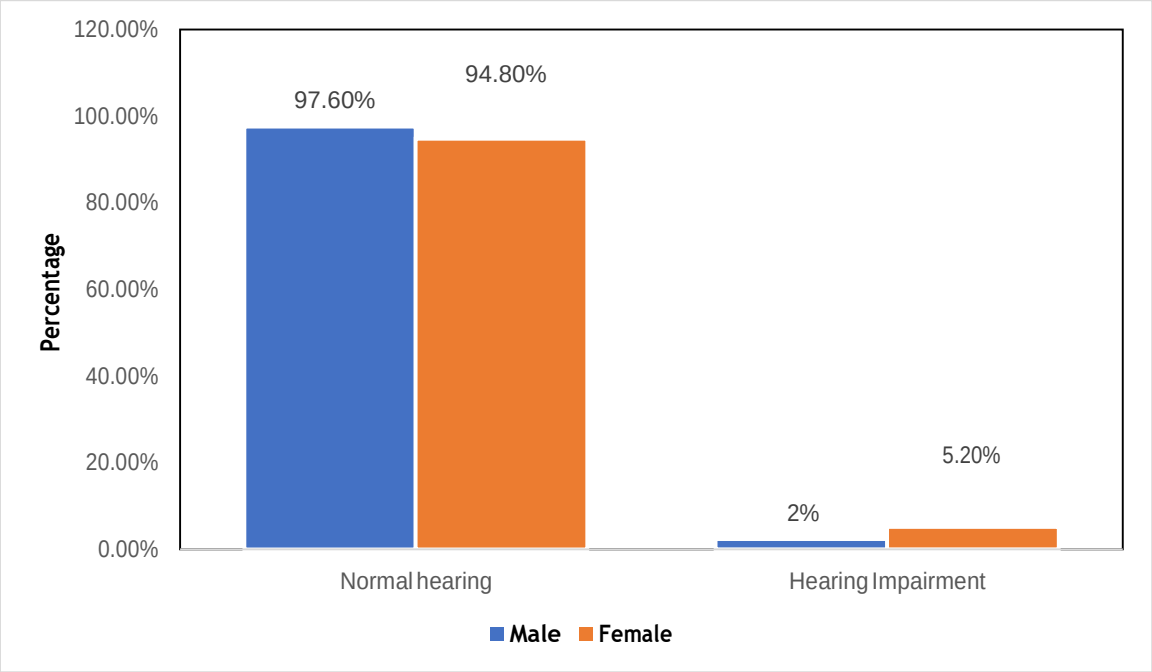


Total four babies had hearing impairment out of 100 babies screened.

Table 16: Distribution of final outcome of screened infants with gender (N=100)

Slno	Outcome	Male	Female	X^2, (df), p
1	Normal hearing	41 (97.6)	55 (94.8)	0.494 (1) 0.482
2	Hearing Impairment	1 (2.4)	3 (5.2)	

Figure 4: Distribution of final outcome of screened infants with gender (N=100)

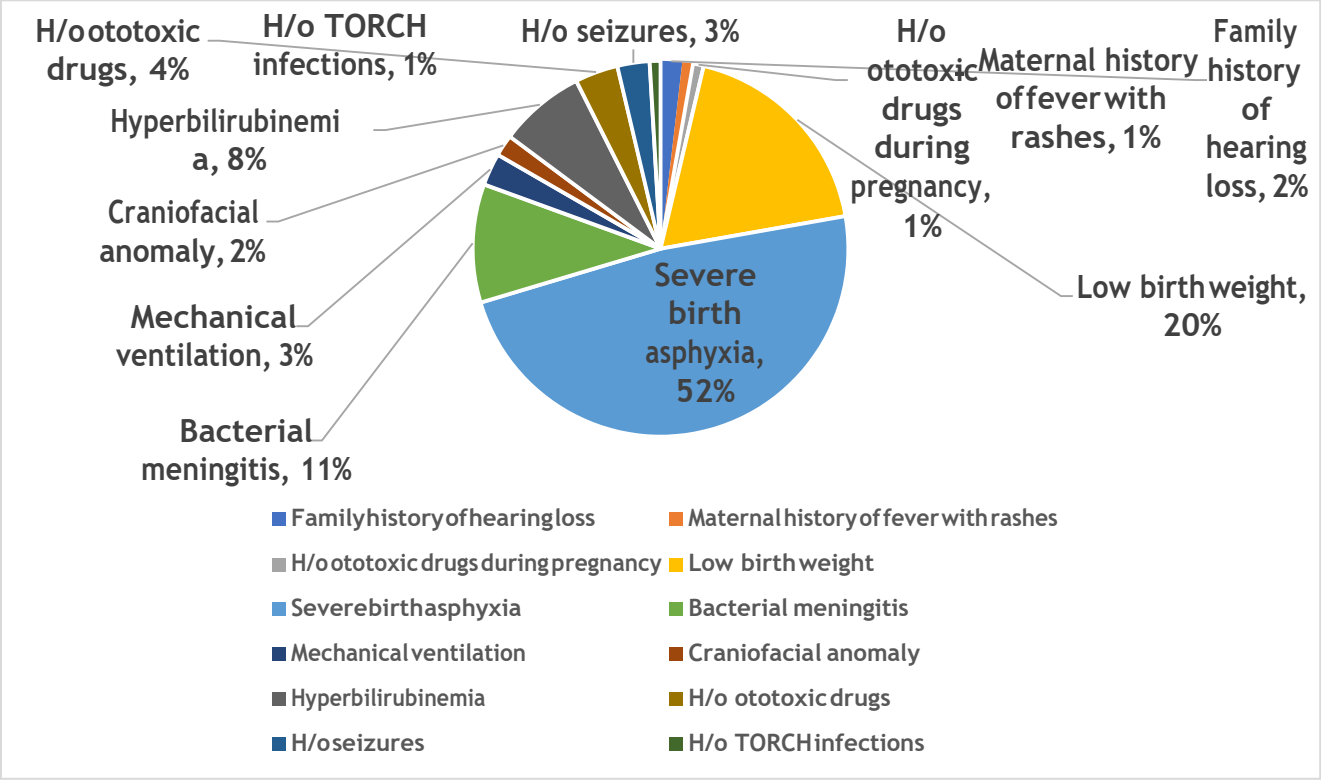


Out of four babies who had hearing impairment, three were female babies and one was male baby.

Table 17: Distribution of final outcome of screened infants with risk factors (N=100)

Slno	Risk factor	Total number of cases	Normal hearing	Hearing impairment	X^2, (df), p
1	Family history of hearing loss	2	0	2	48.98(1), <0.001
2	Maternal history of fever with rashes	1	1	0	0.042 (1), 0.83
3	H/o ototoxic drugs during pregnancy	1	1	0	0.042 (1), 0.83
4	Low birth weight	20	19	1	0.065 (1), 0.79
5	Severe birth asphyxia	52	49	3	1.883 (1), 0.04
6	Bacterial meningitis	11	11	0	0.515 (1), 0.47
7	Mechanical ventilation	3	0	0	0.129 (1), 0.72
8	Craniofacial anomaly	2	0	0	0.085 (1), 0.77
9	Hyperbilirubinemia	8	0	0	0.362 (1), 0.55
10	H/o ototoxic drugs	4	0	0	0.174 (1), 0.67
11	H/o seizures	3	0	0	0.129 (1), 0.72
12	H/oTORCH infections	1	1	0	0.042 (1), 0.837

Figure 5: Distribution of final outcome of screened infants with risk factors (N=100)



There is statistical significance in family history of hearing loss and severe birth asphyxia as significant risk factor for hearing impairment.

DISCUSSION

Prior to being discharged from the hospital where they were born, all new borns should be screened for hearing loss. It's not always possible in a developing country like ours with limited resources. As a result, all new borns with risk factors should have their hearing tested done at the least. According to various statistics, a hearing impairment occurs in 2.5 percent to 10 percent of high-risk neonates. [65] As with other studies, this study found a 4.0% incidence rate.

In our study 100 babies were initially screened and subjected to OAE testing. A total of 17 babies failed at first screening by OAE on right ear and 8 babies on left ear. They include 7 with severe birth asphyxia, 4 babies with low birth weight, 3 babies had bacterial meningitis, one had mechanical ventilation, two needed transfusion because of hyperbilirubinemia and one had history of seizures. Total babies subjected to AABR were17. Out of which 13 babies passed AABR screening. Four babies failed after AABR tests**.** Among them three had severe birth asphyxia and one had low birth weight. The baby with craniofacial anomaly passes AABR and other four babies were diagnosed with hearing impairment. Four babies were diagnosed to have hearing impairment out of 100 high risk babies. The incidence rate is 4.0% which is similar to other studies done (2.5 -10%).[65]

BIRTH ASPHYXIA

In our study fifty two babies with severe birth asphyxia of which seven babies (13.46%) had hearing impairment in first screening by OAE and three out of seven babies had hearing impairment in the second screening by AABR. 86.54% had normal hearing out of 52 babies of birth asphyxia screened.

Among 51 babies who had suffered severe asphyxia during birth, one had hearing loss, according to a study by Nagapoornima et al.[66] A study by Christine Ohl et al found four babies with hearing impairment after screening 12 babies who had suffered severe birth asphyxia.[67]

LOW BIRTH WEIGHT

In our study twenty babies with low birth weight of which four babies had hearing impairment in first screening by OAE and one out of four baby had hearing impairment in the second screening by AABR. We found no association of low birth weight with hearing impairment.

Low birth weight (LBW) was not linked to hearing impairment in Christine Ohl et als study, which found babies with LBW had normal hearing.[67] We found that LBW was not associated with hearing loss in studies by Finckh Kramer U et al, and Hess M et al.[68,69]

BACTERIAL MENINGITIS

In our study eleven babies with bacterial meningitis of which three babies had hearing impairment in first screening by OAE and all the three babies passed the second screening by AABR.

Despite screening 14 meningitis babies, none of them had hearing impairment, as found in our study by Nagapoornima et al.[66]

HYPERBILIRUBINEMIA

In our study eight babies screened for hyperbilirubinemia of which two babies had hearing impairment in first screening by OAE and the two babies passed the second screening by AABR.

Even though Nagapoornima et al looked for hearing impairment in 38 babies with severe hyperbilirubinemia who needed exchange transfusions, no such issues were found. This is because hyperbilirubinemia was discovered early and treated effectively.[66]

OTOTOXIC DRUGS

In our study four babies with history of neonatal ototoxic drugs was screened and all the four babies passed the first screening by OAE. One baby with history of ototoxic drugs during pregnancy was screened and the baby passed the first screening by AABR.

According to Finckh Kramer U et al, aminoglycosides do not pose a significant health risk.[68] It was found that aminoglycoside use did not increase the risk of hearing loss by Hess M et al.[69]

MECHANICAL VENTILATION

In our study three babies with mechanical ventilation was screened, one baby had hearing impairment in first screening by OAE and that one baby passed the second screening by AABR.

M D Mohd Khairi et al conducted a 2-stage hearing assessment in 401 at-risk neonates and concluded that mechanical ventilation for more than 5 days was not an independent risk factor for hearing impairment.[70]

CRANIOFACIAL MALFORMATION

In our study two of the babies were born with craniofacial malformation. OAE could not be performed on one baby due to atresia of the auditory canal. The other infant passed the OAE's initial screening.

Nagapoornima et al screened 24 babies with craniofacial malformation but found no evidence of hearing impairment, whereas in our study, one of the two (50%) babies with craniofacial malformation had hearing impairment.[66]

TORCH INFECTION

In our study one baby with history of torch infection was screened and the baby passed the first screening by OAE. Six babies were screened by Nagapoornima et al for the TORCH infection, but none of them had hearing loss, as was the case in our research.[66]

So in our study of 100 babies screened, 4 babies had hearing impairment (4.0%) and it is exceeding the incidence found by Nagapoornima et al, who found three high-risk babies in a sample of 279 (1.07 percent).[66]

We found 4.55 percent of the 1461 at-risk babies to be having hearing impairment, in Christine Ohl et al which is similar to our study findings.[67]

Finckh Kramer U et al examined 1062 at-risk newborns and found that only 1.3% of them had hearing impairment, which is lower than the percentage found in our investigation.[68]

Around 150 high-risk infants were screened by Sayed Hossein Fakhraee et al, and 42 of them (or 28% of the total) had varying degrees of hearing loss.[71]

CONCLUSION

- In this study of 100 babies were screened, 4 babies(4.0%) had hearing impairment.
- Of the 12 risk factors screened, severe birth asphyxia, family history of hearing loss seem to be associated with hearing impairment.
- 13.46%(7 out of 52) of babies with severe birth asphyxia and 100%(2 out of 2) of babies with family history of hearing loss had hearing impairment.
- Meningitis, hyperbilirubinemia, ventilated babies, and those who received ototoxic drugs did not show any hearing impairment, which is most likely due to early and effective treatment.
- It is for this reason that early detection and intervention will help deaf and hard of hearing kids develop language skills during the critical period of neural plasticity, preventing them from being cast into a socially isolated existence and an educational future full of misery.

REFERENCES

1. Korver AM, Smith RJ, Van Camp G, Schleiss MR, Bitner-Glindzicz MA, Lustig LR, Usami SI, Boudewyns AN. Congenital hearing loss. Nature reviews Disease primers. 2017 Jan 12;3(1):1-7.
2. Wake M, Hughes EK, Collins CM, Poulakis Z. Parent-reported health-related quality of life in children with congenital hearing loss: A population study. Ambulatory Pediatrics. 2004 Sep 1;4(5):411-7.
3. White KR, Maxon AB. Universal screening for infant hearing impairment: simple, beneficial, and presently justified. International journal of pediatric otorhinolaryngology. 1995 Jul 1;32(3):201-10.
4. Marschark M. Consensus on early identification of hearing loss?. The Journal of Deaf Studies and Deaf Education. 1998 Mar 1;3(2):173-5.
5. Brookhouser PE. Sensorineural hearing loss in children. Pediatric Clinics of North America. 1996 Dec 1;43(6):1195-216.
6. Moneta LB, Quintanilla-Dieck L. Embryology and anatomy of the ear. Operative Techniques in Otolaryngology-Head and Neck Surgery. 2017 Jun 1;28(2):66-71.
7. Wright CG. Development of the human external ear. Journal of the American Academy of Audiology. 1997 Dec 1;8(6).

8. Mansour S, Magnan J, Ahmad HH, Nicolas K, Louryan S. Comprehensive and clinical anatomy of the middle ear. Springer International Publishing; 2019 Jul 4.
9. Ordon A, Wolfswinkel E, Shauly O, Gould DJ. Aesthetic otoplasty: principles, techniques and an integrated approach to patient-centric outcomes. Aesthetic plastic surgery. 2019 Oct;43(5):1214-25.
10. Sizarov A, Baldwin HS, Srivastava D, Moorman FM. Development of the heart: Morphogenesis, growth, and molecular regulation of differentiation. Moss and Madams' Heart Disease in Infants, Children, and Adolescents Including the Fetus and Young Adult, 9th edition. Philadelphia, Wolters Kluwer. 2016:1-54.
11. Raja J, SA S, TN SB. Dual AGC Model Implementation of the Inner Hair Cell and Auditory Nerve IC in Neuromorphic VLSI.
12. Alvord LS, Farmer BL. Anatomy and orientation of the human external ear. Journal of the American Academy of Audiology. 1997 Dec 1;8(6).
13. Marchioni D, Molteni G, Presutti L. Endoscopic anatomy of the middle ear. Indian Journal of Otolaryngology and Head & Neck Surgery. 2011 Apr;63(2):101-13.
14. Atmaja BT. The Physiology, Mechanism, and Nonlinearities of Hearing.
15. Proctor B. Anatomy of the eustachian tube. Archives of Otolaryngology. 1973 Jan 1;97(1):2-8.
16. Davis H. Biophysics and physiology of the inner ear. Physiological reviews. 1957 Jan 1;37(1):1-49.

17. Hussain B, Ali M, Qasim M, Masoud MS, Khan L. Hearing impairments, presbycusis and the possible therapeutic interventions. Biomedical Research and Therapy. 2017 Apr 20;4(4):1228-45.
18. Irvine DR. Physiology of the auditory brainstem. InThe mammalian auditory pathway: Neurophysiology 1992 (pp. 153-231). Springer, New York, NY.
19. Baliński S, Morawska-Kochman M, Bochnia M. Hearing and stomatognathic system: Searching for a link. Dental and Medical Problems. 2017;54(4):417-22.
20. Emanuel D, Sumalai M, Letowski T. Auditory function: Physiology and function of the hearing system. Helmet-Mounted Displays: Sensory, Perceptual, and Cognitive Issues, Edition. 2009;1:307-34.
21. Killion MC, Dallos P. Impedance matching by the combined effects of the outer and middle ear. The Journal of the Acoustical Society of America. 1979 Aug;66(2):599-602.
22. Alberti PW. The anatomy and physiology of the ear and hearing. Occupational exposure to noise: Evaluation, prevention, and control. 2001:53-62.
23. Songer JE. *Superior Semicircular Canal Dehiscence: Auditory Mechanisms* (Doctoral dissertation, Harvard-MIT Division of Health Sciences and Technology).
24. Flock Å. Sensory transduction in hair cells. InPrinciples of receptor physiology 1971 (pp. 396-441). Springer, Berlin, Heidelberg.
25. Graven SN, Browne JV. Auditory development in the fetus and infant. Newborn and infant nursing reviews. 2008 Dec 1;8(4):187-93.

26. Ching TY. Is early intervention effective in improving spoken language outcomes of children with congenital hearing loss?. American journal of audiology. 2015 Sep;24(3):345-8.
27. Takesian AE, Hensch TK. Balancing plasticity/stability across brain development. Progress in brain research. 2013 Jan 1;207:3-4.
28. Parving A, Hauch AM, Christensen B. Hearing loss in children--epidemiology, age at identification and causes through 30 years. Ugeskrift for laeger. 2003 Feb 1;165(6):574-9.
29. Kountakis SE, Skoulas I, Phillips D, Chang CJ. Risk factors for hearing loss in neonates: a prospective study. American journal of otolaryngology. 2002 May 1;23(3):133-7.
30. Declau F, Boudewyns A, Van den Ende J, Peeters A, van den Heyning P. Etiologic and audiologic evaluations after universal neonatal hearing screening: analysis of 170 referred neonates. Pediatrics. 2008 Jun 1;121(6):1119-26.
31. Paludetti G, Conti G, Di Nardo W, De Corso E, Rolesi R, Picciotti PM, Fetoni AR. Infant hearing loss: from diagnosis to therapy Official Report of XXI Conference of Italian Society of Pediatric Otorhinolaryngology. Acta Otorhinolaryngologica Italica. 2012 Dec;32(6):347.
32. Arumugam SV, Paramasivan VK, Murali S, Natarajan K, Kameswaran M. Syndromic deafness-prevalence, distribution and hearing management protocol in Indian scenario. Annals of Medicine and Surgery. 2015 Jun 1;4(2):143-50.

33. Mahboubi H, Dwabe S, Fradkin M, Kimonis V, Djalilian HR. Genetics of hearing loss: where are we standing now?. European Archives of Oto-Rhino-Laryngology. 2012 Jul;269(7):1733-45.
34. Ciuman RR. Stria vascularis and vestibular dark cells: characterisation of main structures responsible for inner-ear homeostasis, and their pathophysiological relations. The Journal of Laryngology & Otology. 2009 Feb;123(2):151-62.
35. Nesterova AP, Klimov EA, Zharkova M, Sozin S, Sobolev V, Ivanikova NV, Shkrob M, Yuryev A. Diseases of the Ear. Disease Pathways. 2020:297.
36. Michalski N, Petit C. Genetics of auditory mechano-electrical transduction. Pflügers Archiv-European Journal of Physiology. 2015 Jan;467(1):49-72.
37. Kenna MA. Acquired hearing loss in children. Otolaryngologic Clinics of North America. 2015 Dec 1;48(6):933-53.
38. Barbi M, Binda S, Caroppo S, Ambrosetti U, Corbetta C, Sergi P. A wider role for congenital cytomegalovirus infection in sensorineural hearing loss. The Pediatric infectious disease journal. 2003 Jan 1;22(1):39-42.
39. Xia W, Yan H, Zhang Y, Wang C, Gao W, Lv C, Wang W, Liu Z. Congenital Human Cytomegalovirus Infection Inducing Sensorineural Hearing Loss. Frontiers in Microbiology. 2021 Apr 14;12:824.
40. OJALA P, VESIKARI T, ELO O. Rubella during pregnancy as a cause of congenital hearing loss. American journal of epidemiology. 1973 Nov 1;98(5):395-401.

41. Cunningham M, Cox EO, Committee on Practice and Ambulatory Medicine. Hearing assessment in infants and children: recommendations beyond neonatal screening. Pediatrics. 2003 Feb 1;111(2):436-40.
42. Probst R, Lonsbury-Martin BL, Martin GK. A review of otoacoustic emissions. The Journal of the Acoustical Society of America. 1991 May;89(5):2027-67.
43. Coates H, Gifkins K. Newborn Hearing Screening—the Ultimate Early Detection Strategy for Hearing Loss. InHearing Impairment 2004 (pp. 105-112). Springer, Tokyo.
44. Cone-Wesson B, Dowell RC, Tomlin D, Rance G, Ming WJ. The auditory steady-state response: comparisons with the auditory brainstem response. Journal of the American Academy of Audiology. 2002 Apr;13(04):173-87.
45. Emami SF, Gohari N. The vestibular-auditory interaction for auditory brainstem response to low frequencies. International Scholarly Research Notices. 2014;2014.
46. Van Straaten HL. Automated auditory brainstem response in neonatal hearing screening. Acta Paediatrica. 1999 Dec;88:76-9.
47. Walker JJ, Cleveland LM, Davis JL, Seales JS. Audiometry screening and interpretation. American family physician. 2013 Jan 1;87(1):41-7.
48. Parving A. Epidemiology of hearing loss and aetiological diagnosis of hearing impairment in childhood. International Journal of Pediatric Otorhinolaryngology. 1983 Jan 1;5:151-65.

49. Sloan-Heggen CM, Bierer AO, Shearer AE, Kolbe DL, Nishimura CJ, Frees KL, Ephraim SS, Shibata SB, Booth KT, Campbell CA, Ranum PT. Comprehensive genetic testing in the clinical evaluation of 1119 patients with hearing loss. Human genetics. 2016 Apr 1;135(4):441-50.
50. Lammens F, Verhaert N, Devriendt K, Debruyne F, Desloovere C. Aetiology of congenital hearing loss: a cohort review of 569 subjects. International journal of pediatric otorhinolaryngology. 2013 Sep 1;77(9):1385-91.
51. Lieu JE, Kenna M, Anne S, Davidson L. Hearing loss in children: a review. JAMA. 2020 Dec 1;324(21):2195-205.
52. Dillon H. Hearing aids. Hodder Arnold; 2008.
53. House WF. Cochlear implants. Annals of Otology, Rhinology & Laryngology. 1976 May;85(3_suppl):3-.
54. Sundström S. Prosodic and Phonological Ability in Children with Developmental Language Disorder and Children with Hearing Impairment. Linköping University Electronic Press; 2018 May 15.
55. Kennedy CR, Kimm L, Dees DC, Evans PI, Hunter M, Lenton S, Thornton RD. Otoacoustic emissions and auditory brainstem responses in the newborn. Archives of Disease in Childhood. 1991 Oct 1;66(10 Spec No):1124-9.
56. Norton SJ, Gorga MP, Widen JE, Folsom RC, Sininger Y, Cone-Wesson B, Vohr BR, Mascher K, Fletcher K. Identification of neonatal hearing impairment: evaluation of transient evoked otoacoustic emission, distortion

product otoacoustic emission, and auditory brain stem response test performance. Ear and hearing. 2000 Oct 1;21(5):508-28.

57. Barker SE, Lesperance MM, Kileny PR. Outcome of newborn hearing screening by ABR compared with four different DPOAE pass criteria.

58. Meier S, Narabayashi O, Probst R, Schmuziger N. Comparison of currently available devices designed for newborn hearing screening using automated auditory brainstem and/or otoacoustic emission measurements. International journal of pediatric otorhinolaryngology. 2004 Jul 1;68(7):927-34.

59. Johnson JL, White KR, Widen JE, Gravel JS, James M, Kennalley T, Maxon AB, Spivak L, Sullivan-Mahoney M, Vohr BR, Weirather Y. A multicenter evaluation of how many infants with permanent hearing loss pass a two-stage otoacoustic emissions/automated auditory brainstem response newborn hearing screening protocol. Pediatrics. 2005 Sep 1;116(3):663-72.

60. Xu Z, Li J. Performance of two hearing screening protocols in the NICU. B-ent. 2005 Jan 1;1(1):11-5.

61. Lin HC, Shu MT, Lee KS, Lin HY, Lin G. Reducing false positives in newborn hearing screening program: how and why. Otology & Neurotology. 2007 Sep 1;28(6):788-92.

62. Vignesh SS, Jaya V, Sasireka BI, Sarathy K, Vanthana M. Prevalence and referral rates in neonatal hearing screening program using two step hearing screening protocol in Chennai–A prospective study. International journal of pediatric otorhinolaryngology. 2015 Oct 1;79(10):1745-7.

63. Dumanch KA, Holte L, O'Hollearn T, Walker E, Clark J, Oleson J. High risk factors associated with early childhood hearing loss: a 3-year review. American journal of audiology. 2017 Jun 13;26(2):129-42.
64. Howell JB, Appelbaum EN, Armstrong MF, Chapman D, Dodson KM. An analysis of risk factors in unilateral versus bilateral hearing loss. Ear, Nose & Throat Journal. 2019 Jul;98(6):330-3.
65. Abraham paul K. Hearing loss in neonates and infants : Need for early detection and intervention. Pediatrics Today Vol.XII No.4 July-August 2009 : 157-160.
66. Nagapoornima P, Ramesh .A, Srilakshmi, Suman Rao, Patricia. P.L. and Madhuri Gore. Universal Hearing Screening. Indian Journal of Paeditrics.2007 Jun;74(6):545 – 9
67. Christine Ohl, LilianeE Dornier, Cecile Czajka, Jean-Claude Chobaut and Laurent Tavernier. International Journal of Paediatric OtorhinoLaryngology.73 (2009) 1691 - 1695.
68. Finckh-Kramer U, Gross M,Bartsch M, Kewitz G, Versmold H and Hess M. Hearing screening of high risk newborn infants. HNO.2000 Mar : 48(3) 215 - 20.
69. Hess M, Finckh-Kramer U, Bartsch M,Kewitz G, Versmold and Gross M. Hearing screening in at-risk neonate cohort. Int J Paediatr Otorhinolaryngol.1998 Nov15 ; 46(1-2) : 81-9.

70. Mohd Khairi , Din ,Shahid and Normastura .Hearing screening of infants in Neonatal Unit using otoacoustic emissions.
Journal of Laryngology & Otology (2005), 119:9:678-683
71. Sayed Hossein Fakhraee, Mohammad Kazemian,and Amir- Ali Hamidieh. Hearing assessment of the high risk neonates. Arch Iranian Med. 2004 . **7**(1): 44 – 46.

INFORMED CONSENT FORM

STUDY:A STUDY ON EVALUATION OF HEARING IN HIGH RISK INFANTS USING DPOAE AND AABR

STUDY CENTRE: Department of Otorhinolaryngology,

Govt.Kilpauk Medical College Hospital, Chennai.

PATIENTS NAME :

PATIENTS AGE :

I.P NO. :

Patient may check ( ) these boxes

I confirm that I understood the purpose of the procedure for the above study.( )

I had the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ( )

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.( )

I understand that the ethical committee members and the regulatory authorities will not need my permission to look at my health records, both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. ( )

However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. ( )

I agree not to restrict the use of any data or results that arise from the study. ( )

I agree to take part in the above study and to comply with the instructions given during the study and faithfully co-operate with the study team and to immediately inform the study staff if I suffer any unexpected or unusual symptoms. ( )

I hereby consent to participate in this study. ( )

My signature below indicates that I have decided to let my baby participate, that I have read (or been read) the information provided above, that I was given the opportunity to ask questions and that they have answered to my satisfaction, and that I have received a copy of this signed consent form. ( )

Signature / thumb impression of the patient:

Patient's name and address:

Place:

Date:

Signature of the investigator:

investigator's name:

Place:

Date:

சுயஒப்புதல்படிவம்

ஆர ாய்ச்ச ிநிலை யமம்:கீ

ழ்ப்ப ாக்கமம்மருதத்துவகக்கல்லூரிஅரசுமருதத்துவம

னை,சென்னை.

பங்குபபறுபவரின்பபயர்:

பங்குபபறுபவரின்எண்:

பங்குபபறுபவர்இதலை( )குறிக்கவும்

மமமேகுறிப்பிட்டுள்ளமருத்துவஆய்வின்விவரங்கள்ளைக்குவிளக்கப்ப

ட்டது.என்னுனடயெந்மேகங்கனளக்மகட்கவும்,அேற்காைேகுந்ே

விளக்க ங்கனளப்சபறவும்வாய்ப்பளிக்கப்பட்டது. ( )

நானம்இவ்வாய்வில்ேனம்ைிச்னெயாகத்ோனம்பங்ம

கறம்கிமறனம்.எந்ேக்காரண

த்ேினைாமேோஎநம்ேக்கட்டத்ேிலுமம்எந்ேஎட்டெிக்கலுக்க

ூமம்உட்படாமல்நான்

இவ்வாய்வில்இருந்துவிேகிக்சகாள்ளோம்என்றும்அறிந்து

சகாண்மடன். ( )

இந்ேஆய்வுெம்மந்ேமாகவும்,மமலும்இதுெோர்ந்ேஆய்வுமமற்ச

காள்ளும்

மபாதும்,இந்ேஆய்வில்பங்குசபறும்மருத்துவர்என்னுனடயமருத்து

வஅ

றிக்னகனளப்பார்ப்பேற்குஎன்அனுமேிமேனவயில்னேஎ

ைஅறிந்துசகா

ள்கிமறன்.நான்ஆய்வில்இருந்துவிேகிக்சகாண்டாலும்இதுச

பாருந்தும்எ

ைஅறிகிமறன். ( )

இந்ேஆய்வின்மூேம்கினடக்கும்ேகவல்கனளயும்,ப

மெொேனைமுடிவுகனளயுமம்மறற்முமம்ெிகிச்னெசேொ

டர்பாைேகவல்கனளயு

மம்மருதத்துவர்மமற்சகாள்ளுமம்ஆய்விலம்பயனம்படுதத்ேிகம்

சகாள்ளவுமம்,அனே

ப்பிரசுரிக்கவும்என்முழுமைதுடன்ெம்மேிக்கிமறன். ( )

இந்ேஆய்வில்பங்குசகாள்ளஒப்புக்சகாள்கிமறன்.ளைக்குக்சகாடுக்கப்
பட்
டஅறிவுனரகளின்படிநடந்துசகாள்வதுடன்,இந்ேஆய்னவமமற்சகா
ள்ளூ
ம்மருத்துவஅணிக்குஉண்னமயுடன்இருப்மபன்என்றும்உறுேியளி
க்கி
மறன்.என்உடல்நேம்வழக்கத்ேிற்குமாறாகமநாய்க்குறிசேன்பட்
டாமோ
உடமனேஅனேமருத்துவஅணியிடம்சேரிவிப்மபன்எனேஉறுேி
அளிக்கிமறன். ( )
இந்ேஆய்வில்ளைக்குமருத்துவப்பரிமொேனைசெய்துசகாள்ளமற்
றும் ஆய்வில்பங்மகற்கநான்முழுமைதுடன்ெம்மேிக்கிமறன். (
)

பங்மகற்பவரின்னகசயாப்பம்/கட்னடவிரல்மரனக:

_____

இடம்:

_____

மேேி:_____

பங்மகற்பவரின்சபயர்மற்றும்விேோெம்::

ஆய்வாளரின்னகசயாப்பம்_____

_____ இடம்_____

மேேி_____

ஆய்வாளரின்சபயர்_____

MASTER CHART

S NO	NAME OF MOTHER	AGE (MONTHS)	SEX	TYPE OF DELIVERY	GESTATIONAL AGE AT BIRTH	BIRTH WEIGHT (GRAMS)	LENGTH	HEAD CIRCUMFERENCE	CHEST CIRCUMFERENCE	APGAR 1 MIN	APGAR 5 MINS	FAMILY H/O HEARING LOSS	MATERNAL H/O FEVER WITH RASH	H/O OTOTOXIC DRUGS DURING PREGNANCY	LOW BIRTH WEIGHT	SEVERE BIRTH ASPHYXIA	BACTERIAL MENINGITIS	MECHANICAL VENTILATION	CRANIOFACIAL ANOMALY	HYPERBILIRUBINEMIA	PEAK LEVEL OF TOTAL BILIRUBIN	H/O NEONATAL OTOTOXIC DRUGS	H/O OTHER CONGENITAL MALFORMATION	H/O SEIZURES	TORCH INFECTIONS	PREAURICULAR AREA	PINNA	POST AURICULAR AREA	EXTERNAL AUDITORY CANAL	TYMPANIC MEMBRANE	DPOAE RIGHT	DPOAE LEFT	AABR RIGHT	AABR LEFT
1	SARITHA	9	Male	LSCS	40	1373	44.67	31.46	29.29	4	4	NO	NO	NO	YES	NO	NO	NO	NO	NO	2.1	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
2	VAISHNAVI	5	Female	LSCS	40	1285	46.05	29.85	26.62	4	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	10.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
3	SURYA	1	Male	NVD	38	1386	45.67	30.47	27.06	3	4	NO	NO	NO	YES	NO	NO	NO	NO	NO	11.1	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
4	KALEESWARI	10	Female	NVD	41	1172	46.02	30.91	28.53	5	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	3.1	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
5	LEEMA	3	Male	LSCS	41	1463	46.78	30.31	28.9	3	3	NO	NO	NO	YES	NO	NO	NO	NO	NO	3.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
6	RADHA	1	Male	NVD	40	1275	45.61	31.56	28.87	3	5	NO	NO	NO	YES	NO	NO	NO	NO	NO	2.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
7	FATHIMA	4	Male	NVD	42	1009	44.06	30.09	26.97	4	4	YES	NO	NO	YES	NO	NO	NO	NO	NO	9.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	REFER	PASS
8	RATNAM	8	Female	LSCS	41	1372	45.54	31.37	29.14	3	3	NO	NO	NO	YES	NO	NO	NO	NO	NO	4.8	NO	NO	NO	NO	TAG	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
9	KAYAL	3	Male	NVD	38	1075	44.71	30.74	28.91	4	5	NO	NO	NO	YES	NO	NO	NO	NO	NO	11.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
10	GEETHA	4	Female	LSCS	38	971	46.24	30.24	27.66	6	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	9.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
11	RUKKU	9	Male	NVD	40	1202	46.84	30.05	27.48	6	7	NO	NO	NO	YES	NO	NO	NO	NO	NO	3.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
12	PARIMALA	3	Female	LSCS	40	1246	46.73	31	28.94	5	5	NO	NO	NO	YES	NO	NO	NO	NO	NO	4.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
13	KARPAGAM	12	Male	NVD	40	1150	46.2	30.32	29.33	6	7	NO	NO	NO	YES	NO	NO	NO	NO	NO	9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
14	ANJU	12	Female	NVD	39	1496	44.78	31.01	27.87	5	7	NO	NO	NO	YES	NO	NO	NO	NO	NO	12	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
15	THILAGA	12	Male	NVD	38	1343	46.61	31.26	29.4	4	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	2.6	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
16	DEEPA	11	Female	LSCS	40	1085	44.71	30.74	29.37	5	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	4.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
17	RAMYA	6	Female	NVD	41	901	46.24	30.24	27.89	4	5	NO	NO	NO	YES	NO	NO	NO	NO	NO	8.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS
18	NISHA	12	Female	NVD	41	1429	46.84	30.05	28.53	6	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	8.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
19	ARCHANA	1	Female	NVD	42	1440	46.73	31	27.27	4	5	NO	NO	NO	YES	NO	NO	NO	NO	NO	5.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
20	NITHYA	9	Male	LSCS	39	1252	46.2	30.32	27.35	6	6	NO	NO	NO	YES	NO	NO	NO	NO	NO	4.6	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
21	ABINAYA	11	Female	NVD	41	2396	49.35	33.88	30.19	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	11.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
22	CHITRA	10	Male	NVD	41	2907	49.2	33.94	32.24	6	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	7.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
23	DEEPA	2	Male	NVD	41	3376	48.41	34	31.94	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	7.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
24	GEETHA	11	Male	LSCS	39	2851	50.68	33.48	30.02	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
25	GOMATHI	11	Female	NVD	39	2898	49.4	33.41	30.24	4	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	1.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
26	ABRAMI	12	Female	NVD	40	2820	49.97	33.79	31.33	3	4	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
27	BANI	10	Male	NVD	39	2508	49.74	33.76	30.63	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
28	SRUTHI	7	Male	LSCS	40	2560	49.74	33.94	31.32	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	11.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
29	SNEHA	2	Male	NVD	41	2362	48.7	33.73	30.83	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	11.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
30	PREMA	4	Male	NVD	39	2145	48.7	33.86	32.89	3	4	NO	NO	NO	NO	YES	NO	NO	NO	NO	1.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
31	PREETHI	8	Female	NVD	38	2170	49.42	33.25	31	3	3	NO	NO	NO	NO	YES	NO	NO	NO	NO	6.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
32	MANJULA	12	Male	NVD	38	3451	49.46	33.04	31.4	3	3	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
33	SANJANA	4	Female	LSCS	38	2400	48.28	33.53	30.99	4	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
34	KANNAGI	6	Female	NVD	41	2645	49.19	33.71	32.06	3	3	NO	NO	NO	NO	YES	NO	NO	NO	NO	2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
35	LAVANYA	4	Female	NVD	38	2514	49.76	33.17	32.92	3	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.6	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
36	MEERA	11	Female	NVD	39	2541	49.22	33.61	31.97	3	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
37	SANGAVI	2	Female	NVD	41	2070	48.37	33.58	31.6	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	8.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	REFER	PASS
38	TEENA	6	Female	NVD	41	3100	48.41	33.24	32.79	5	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	11.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
39	KANITHA	7	Male	LSCS	39	3183	48.88	33	32.46	4	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
40	RAMYA	2	Male	NVD	40	3109	49.64	33.71	32.13	3	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
41	GAYATHRI	2	Female	NVD	39	2900	48.24	33.29	32.27	4	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
42	BAVYA	10	Male	NVD	38	3316	49.35	33.99	30.4	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	7.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
43	RINITA	6	Male	LSCS	38	3028	49.23	33.24	30.53	4	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
44	KANAGA	11	Male	NVD	38	2651	49.5	33.08	30.65	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	9.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
45	VALLI	10	Female	LSCS	39	2494	49.85	33.1	30.28	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.5	NO	NO	NO	NO	TAG	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS

46	PRABHA	2	Female	NVD	40	3387	48.39	33.49	30.19	3	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	6	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
47	FATHIMA	7	Male	NVD	42	2630	49.72	33.68	31.21	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.1	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
48	FAHINA	1	Female	NVD	41	2436	50.25	33.46	32.38	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	7.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
49	DIVYA	6	Male	LSCS	39	3058	49.44	33.56	30.69	4	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
50	DARSHINI	9	Female	NVD	41	2139	48.92	33.05	30.13	4	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	3.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
51	DHARANI	12	Female	NVD	39	2498	48.35	33.41	30.86	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	REFER	REFER
52	BHAGYA	8	Female	NVD	38	3408	48.89	33.01	30.48	4	7	NO	NO	YES	NO	YES	NO	NO	NO	NO	10.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
53	MUTHU	4	Male	NVD	40	3259	48.59	33.13	32.68	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
54	RATNA	3	Female	NVD	41	2887	49.11	33.27	32.65	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	2.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
55	RENUKA	7	Female	LSCS	40	3400	50.24	33.22	32.1	6	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
56	DEVAKI	3	Male	NVD	39	2182	49.31	33.6	32.49	3	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	5.1	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
57	NANDINI	1	Female	LSCS	41	2288	48.56	33.55	31.56	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	8.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
58	SHANTHI	6	Male	NVD	39	3057	48.67	33.15	32.51	5	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	8.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
59	KALAI	7	Female	NVD	41	2859	49	33.92	31.08	5	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	10.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
60	JAMUNA	6	Female	NVD	38	3458	50.26	33.3	31.93	4	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	11.4	NO	NO	NO	NO	SINUS	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
61	RANI	11	Male	NVD	42	2813	48.33	33.58	31.61	5	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	8.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
62	RENU	3	Female	NVD	38	2741	48.4	33.66	32.11	4	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	6.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS
63	RESHMA	12	Male	NVD	40	3481	48.2	33.27	30.19	6	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	3.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
64	RAGAVI	12	Female	NVD	42	2754	49.66	33.31	32.24	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	3.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
65	CHANDRA	4	Male	NVD	39	2420	50.1	33.88	32.4	3	4	NO	NO	NO	NO	YES	NO	NO	NO	NO	11	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
66	SUGUNA	7	Female	LSCS	39	2659	49.98	33.04	31.15	6	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	3.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
67	SARGUNA	8	Female	NVD	40	3171	50.01	33.62	32.23	3	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
68	MARIAMMAL	9	Female	NVD	39	2165	49.56	33.18	30.93	5	5	YES	NO	NO	NO	YES	NO	NO	NO	NO	6.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	REFER	REFER
69	MUNIYAMMA	9	Female	NVD	40	2003	49.42	33.89	30.23	3	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	9.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
70	MUNISWARI	2	Male	NVD	40	2149	48.63	33.54	31.66	3	5	NO	NO	NO	NO	YES	NO	NO	NO	NO	5.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
71	NATHIYA	7	Male	NVD	38	2718	49.47	33.72	31.78	5	6	NO	NO	NO	NO	YES	NO	NO	NO	NO	5.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS
72	ESWARI	7	Female	NVD	41	3391	48.28	33	30.38	5	7	NO	NO	NO	NO	YES	NO	NO	NO	NO	4.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
73	ESHA	11	Male	LSCS	41	3256	49.88	33.06	30	5	5	NO	NO	NO	NO	NO	YES	NO	NO	NO	6.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
74	MEENA	8	Male	NVD	42	2592	49.69	33.48	31.4	6	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	2.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
75	MANJU	3	Female	NVD	38	2103	49.64	33.22	31.91	4	5	NO	NO	NO	NO	NO	YES	NO	NO	NO	11.7	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
76	MENAKA	10	Female	NVD	41	2694	48.6	33.68	31.97	6	6	NO	NO	NO	NO	NO	YES	NO	NO	NO	5.7	NO	NO	YES	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
77	MAHALAKSHMI	6	Female	NVD	42	2532	48.96	33.76	30.15	5	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	5.6	NO	NO	YES	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS
78	SANJULA	12	Female	NVD	39	3220	49.39	33.28	32.7	5	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	10.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
79	HEMA	6	Female	NVD	40	2106	49.35	33.83	31.55	6	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	1.7	NO	NO	YES	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
80	MENAKA	3	Female	NVD	40	2928	48.97	33.47	32.66	5	6	NO	NO	NO	NO	NO	YES	NO	NO	NO	3.9	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
81	SRUTHIKA	5	Male	NVD	39	2603	48.21	33.62	31.56	6	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	6.2	NO	NO	NO	NO	SINUS	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
82	KARTHIGA	7	Female	AVD	41	2367	48.3	33.22	32.21	3	7	NO	NO	NO	NO	NO	YES	NO	NO	NO	12	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
83	KAVI	9	Female	NVD	42	2590	48.86	33.46	30.34	6	6	NO	NO	NO	NO	NO	YES	NO	NO	NO	1.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
84	AKSHARA	5	Female	NVD	41	2167	48.42	33.75	30.07	5	6	NO	NO	NO	NO	NO	NO	NO	NO	YES	14.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
85	NIMMY	11	Male	NVD	40	2149	49.05	33.98	32.75	3	7	NO	NO	NO	NO	NO	NO	NO	NO	YES	16.2	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
86	NISHA	5	Male	AVD	42	2914	48.84	33.08	32.78	4	7	NO	NO	NO	NO	NO	NO	NO	NO	YES	15	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
87	SWAHA	6	Female	NVD	42	2304	49.62	33	32.19	6	7	NO	NO	NO	NO	NO	NO	NO	NO	YES	14.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
88	KAMATCHI	11	Female	NVD	38	2259	48.56	33.84	30.72	6	6	NO	NO	NO	NO	NO	NO	NO	NO	YES	15.3	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
89	NITHYA	11	Female	NVD	38	2820	48.91	33.88	32.62	4	7	NO	NO	NO	NO	NO	NO	NO	NO	YES	17	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
90	SARANYA	11	Female	NVD	39	3014	48.81	33.15	30.98	5	5	NO	NO	NO	NO	NO	NO	NO	NO	YES	16.8	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	REFER	PASS	PASS
91	CHITRA	8	Female	NVD	41	2035	48.89	33.14	30.26	4	5	NO	NO	NO	NO	NO	NO	NO	NO	YES	15.5	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
92	DEEPA	12	Female	NVD	40	2886	49.45	33.28	32.13	5	7	NO	NO	NO	NO	NO	NO	NO	NO	NO	1.5	YES	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
93	GEETHA	2	Female	NVD	42	2840	48.3	33.53	32.67	3	6	NO	NO	NO	NO	NO	NO	NO	NO	NO	4.3	YES	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
94	GOMATHI	8	Male	NVD	42	2475	49.89	33.56	31.59	4	7	NO	NO	NO	NO	NO	NO	NO	NO	NO	1.5	YES	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
95	ABRAMI	2	Female	NVD	39	3492	49.8	33.12	31.11	4	6	NO	NO	NO	NO	NO	NO	NO	NO	NO	1.1	YES	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
96	BANI	6	Male	NVD	39	2204	49.07	33.99	32.01	6	6	NO	NO	NO	NO	NO	NO	YES	NO	NO	4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
97	SRUTHI	3	Female	NVD	42	2097	48.76	33.91	32.79	6	6	NO	NO	NO	NO	NO	NO	YES	NO	NO	4.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
98	SNEHA	12	Female	NVD	40	3111	50.12	33.84	31.02	5	6	NO	NO	NO	NO	NO	NO	YES	YES	NO	11.6	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	REFER	PASS	PASS	PASS
99	PREMA	2	Male	NVD	38	2885	48.66	33.79	30.23	4	5	NO	NO	NO	NO	NO	NO	NO	YES	NO	2.4	NO	NO	NO	NO	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS
100	PREETHI	5	Male	NVD	42	2911	48.95	33.35	32.67	3	7	NO	YES	NO	NO	NO	NO	NO	NO	NO	9.4	NO	NO	NO	YES	NORMAL	NORMAL	NORMAL	NORMAL	INTACT	PASS	PASS	PASS	PASS