Introduction and Rationale

The inner ear is an organ which has two functions; the cochlea is responsible for hearing and the vestibular system for balance. Alterations in these organs can cause major difficulties for human beings, such as reduction in the capacity to react to environmental sounds, to keep an effective communication with the environment or even to maintain body balance (Schmidt et al., 2010).

The hormonal alterations which happen during pregnancy can result in changes in the homeostasis of labyrinthine fluids, since they have a direct influence on the enzymatic process and the action of neurotransmitters. The compromise of labyrinth fluid characteristics, as well as the interference on the sensitivity of enzymatic receptors influences the basal metabolism of the inner ear. These alterations may be asymptomatic or associated with otologic symptoms (Bittar, 1999).

Symptoms such as tinnitus, dizziness and sudden hearing loss (HL) are often associated with the action of estrogen and progesterone on the cochlea, posterior labyrinth and central auditory pathway. Dizziness is more frequent in the first two trimesters of pregnancy. Nausea is the main symptom associated with dizziness in pregnant women. When the gestational trimesters are compared, nausea is more frequent in the first gestational trimester and it reduces as the pregnancy progresses (Schmidt et al., 2010).
The most frequent audiological complaint during pregnancy is tinnitus with proposed theories including hyperdynamic circulation, increase in perilymphatic fluid pressure and hormonal changes. More significantly, tinnitus considered as an early sign of preeclampsia (PE) (Al-Zubiadi, 2012; Haas et al., 2007).

Sudden sensorineural hearing loss (SSNHL) may occur during pregnancy, but its prevalence is very low. It is suggested that SSNHL is closely related to the changes in the cardiovascular system, hematological system, endocrine system, and/or some other systems due to pregnancy. These changes possibly evoke disorders of cochlear circulation or cochlear fluid homeostasis leading to SSNHL (Hou and Wang, 2011).

Since, both the clinical manifestations caused by vestibular as well as hearing disorders affect the life routine; family, social and professional relations. This cause loss of self-confidence, concentration and performance causing frustration and depression. So this assay aims at clarifying the importance of evaluation of audio-vestibular findings during pregnancy and explanation of the causes.
Aim of the Study

- To highlight on the auditory and vestibular findings in pregnant women.
Functional Anatomy of the Audio-vestibular System

The human ear consists of the outer ear (pinna or concha, external ear canal, tympanic membrane), the middle ear (middle ear cavity with the three ossicles malleus, incus and stapes) and the inner ear (cochlea which is connected to the three semicircular canals by the vestibule, which provides the sense of balance). The cochlea is connected to the brain stem via the eighth cranial nerve, i.e. the vestibular cochlear nerve or nervus statoacusticus. Subsequently, the acoustical information is processed by the brain at various levels of the auditory system. An overview about the anatomy of the ear is provided by figure 1 (Kollmeier, 2008).

Figure (1): An overview of the anatomy of the outer ear, middle ear & inner ear (Kollmeier, 2008).
1- **External Ear:**

The pinna and the initial part of the external auditory canal are constructed from elastic cartilage and the more internal part of the external ear canal is surrounded by bone. In its rest position, the external ear canal is a bit winded so that the ear drum can only be inspected from outside if this winding is removed (e.g. by softly pulling the pinna to the back and upward). This is utilized when performing otoscopy. The permeability of the external auditory canal, the shape and consistency of the ear drum, and any suspicious changes in the visible structures are considered by an inspection using otoscope, a magnifying glass or a microscope. The function of the pinna is to sample the incident sound wave from a larger area into the smaller area of the ear canal (funnel principle, especially for high frequencies). Also, a spectral change (filter) is provided as a function of the incidence direction. The resulting change in timbre as a function of incidental direction can be utilized for localization of the sound source from where the sound is emitted. Additionally, interaural differences in level and in arrival time occur that are utilized by the brain to perform a very exact localization in the horizontal plane by comparing the input to both ears (*Kollmeier, 2008*).

2- **Middle Ear:**

The middle ear is located in the air-filled tympanum which is connected via the Eustachian tube (ET) to the nose/throat cavity (figure 1). This narrow tube can actively be opened by muscle tension when swallowing or yawning. The opening produces a pressure release on both sides of the tube which provides the same atmospheric pressure both from
outside and inside the tympanic membrane. The maintenance of equilibrium is important for the free movement of the tympanic membrane as well as the successive middle ear ossicles. A strong displacement of the tympanic membrane (e.g. by changes of the air pressure) can also be compensated by the joint between malleus head and incus that fixates itself into a new position (*Hudde and Engel, 1997*).

The function of the middle ear is the impedance adjustment between the sound propagation in air (low impedance) and the quite high input impedance of the liquid-filled inner ear (high impedance). This is primarily achieved by the ratio between the large area of the tympanic membrane and the small area of the stapes footplate attached to the oval window. An additional contribution arises from the leverage of the long processes of the malleus connected to the ear drum and the shorter processes of the incus connected to the stapes. The total transformation effect produces an approximate increase of the force per unit of area by a factor of 50 at the stapes footplate (*Kollmeier, 2008*). At very high sound pressure levels, the stapedius muscle comes into action which is attached to the head of the stapes and produces a change of middle ear transmission efficiency that can be measured by acoustical impedance measurements.

A pathological change of the middle ear function usually results in a conductive hearing loss (CHL). This may be caused by insufficient ventilation through the Eustachian tube as well as fluid or infections in the middle ear cavity. Even though the type of the pathological change can be very different (i.e. otosclerosis with a fixation of the stapes footplate to the surrounding bone, disruption of the ossicular chain, defect
in the tympanic membrane, etc.), the resulting dysfunction is very similar. For a functional diagnostics of the middle ear, impedance audiometry is utilized. Tympanometry tests the static air pressure delivered to the ear canal that yields the best transition (i.e. maximum acoustical compliance of the ear drum) (Kollmeier, 2008).

3- Inner Ear:

The inner ear includes a hearing apparatus (the cochlea) as well as a balance apparatus (the vestibular system) (Kinne, 2015).

Cochlea is a coiled shell-shaped tube which is embedded in the very hard temporal bone and consists of three compartments. The oval window connects to the scala vestibuli which is connected at the upper end of the cochlea, the so-called helicotrema, with the scala tympani. The intermediate compartment (scala media) is filled with endolymph and is separated from the scala tympani by the basilar membrane (figure 2). The lateral width of the basilar membrane increases steadily from the oval window to the helicotrema and simultaneously decreases in its stiffness (De Boer, 1980).

If a sound excites the oval window, a pressure difference occurs orthogonal to the basilar membrane between the scala vestibuli and the scala tympani which produces a movement of the basilar membrane leads to a peculiar wave pattern on the basilar membrane, the so-called travelling wave. It propagates with a comparatively slow velocity and a very high dispersion along the basilar membrane from stapes toward the helicotrema and displays maximum amplitude for high frequencies close to the stapes and for low frequencies close to the helicotrema (frequency-
place transformation). Many theories have been developed in the past to explain this pattern, including a linear theory (De Boer, 1980) and three-dimensional finite element models (Bohnke and Arnold, 1999).

**Figure (2):** Schematic of the cochlea and its cross section. The three fluid-filled ducts named scala vestibuli (SV), scala media (SM), and scala tympani (ST) are separated from each other by Reissner’s membrane and the basilar membrane (BM). The organ of Corti (OC) contains sensory cells that detect sound (Murakoshi et al., 2015).

The displacement of the basilar membrane produces a shear movement in relation to the tectorial membrane which is detected by the inner and outer hair cells embedded on top of the basilar membrane (figure 3). The stereocilia at the end of the hair cells are connected with each other with tiny links (“tip links”) (figure 4) that produce a change in ion permeability, if a mechanical stress is applied (Hudspeth et al., 1998; Dallos et al., 1991).
Functional Anatomy of the Audio-vestibular System

Figure (3): Schematic of structure of the organ of corti (OC). The OC contains two types of sensory cells, inner hair cells (IHCs) and outer hair cells (OHCs). The tectorial membrane is an extracellular matrix and covers the OC. The BM is located beneath the OC, which is composed of the fibrous layer and the hyaline matrix. Scale bar equals 50μm (Murakoshi et al., 2015).

Figure (4): Scanning microscope photography of an intact hair cell bundle (stereocilia) located on a top of a hair cell. Tip links (arrows) connect shorter stereocilia to their taller neighbours (Dallos et al., 1991; Hudspeth et al., 1998).
Hence, the hair cells detect a shear movement by a change in their intracellular potential. While the inner hair cells are each connected to a large number of afferent nerve fibers (that transfer excitation from the receptors to the brain), the three rows of outer hair cells are primarily connected to efferent fibers (i.e. slowly changing adaptation information from the brain to the hair cells). The outer hair cells are capable of actively contracting themselves if an external voltage is applied. This is used to amplify vibrations at low acoustical input levels, so that an active feedback loop simultaneously increases the sensitivity and the frequency specificity of the basilar membrane response (Gummer et al., 2002; Dallos et al., 1997).

A pathological change of the inner ear usually causes a sensorineural hearing loss that may have various reasons (excessive noise exposure, age-related detriment of the hair cells, metabolic diseases, etc.). The function of the inner ear can be tested with a tuning fork or with the audiogram using both bone-conduction transducers in comparison to measuring the sensitivity to air-borne sound with headphones. The presence of otoacoustical emissions (OAEs) is an indication of a (nearly) normal auditory system, while the threshold test and speech audiometry can be used to assess suprathreshold distortions of the auditory perception due to a hearing loss (i.e. recruitment phenomenon or decreased speech intelligibility in noise). Also, an “objective” auditory test which tests both the function of the inner ear and parts of the brain, the brain stem audiometry (brain stem-evoked response audiometry, BERA) is available for clinical diagnostics (Kollmeier, 2008).
Auditory Pathway:

The auditory nerve originates in the middle of the cochlea and runs through the meatus acusticus internus (inner ear duct) to the brain stem where it reaches the cochlear nucleus. From this brain nucleus, several links exist to other nuclei in the brainstem (i.e. superior olive, nucleus accessories and to the nuclei of the lateral lemniscus). The auditory pathway goes up through the lateral lemniscus and the medial lemniscus into the inferior colliculus and the medial geniculate body until it finally reaches the primary auditory cortex which is located in area 41, 42 of the temporal lobe in the cortex (Figure 5). Several linkages exist at each of these stages to the respective other side (Langner and Schreiner, 1988).

The function of the auditory nerve and the auditory pathway is the coding and processing of all acoustical information into neural excitation patterns in the appropriate structures that represent auditory information to the brain. The auditory nerve codes acoustical information by the synchronicity in the spiking pattern of different neural fibers as well as increasing the rate of the respective fibers with increasing stimulus level. In the brain stem, already complex auditory features are evaluated. For example, an interaural comparison to detect interaural arrival time and intensity differences is already performed in the superior olive complex as a basis for localization of sound sources. In addition, a modulation frequency analysis is already performed in the inferior colliculus (Langner and Schreiner, 1988).

The common modulation of the intensity in different frequency bands seems to be an important feature for an auditory object, so that a modulation analysis is assumed to take place already at a comparatively early processing
stage of the auditory system. Furthermore, the whole auditory pathway is characterized by its tonotopic organization, i.e. neighbouring acoustical frequencies yield excitations in neighbouring structures within the whole auditory pathway. A similar spatial organization is supposed for the spatial organization of sound sources in space (spatiotopic organization) and for a regular ordering of the different modulation frequencies in the brain (periodotopic mapping) (Dau et al., 1997).

Pathological changes of the functions of the auditory nerves and the more peripheral parts of the central auditory system (e.g. acoustic neuroma that mechanically damages the auditory nerve or hearing deficits connected with other neurological symptoms as a consequence of a stroke or other neurological disease) are denoted as retrocochlear or neural hearing disorders. They can be detected with brain stem audiometry where a prolonged delay time is observed between the electrical waves originating from the cochlea (i.e. waves I and II) and the more centrally originating parts of the evoked response audiometry (ERA). Alternatively, imaging techniques like functional MR imaging or positron emission tomography can be used. More centrally located damages to the auditory system (for example due to blood circulation deficits or lesions) can result in various neurological symptoms, among which the aphasic syndromes are most prominent. Several central language tests have been developed and applied in order to improve the diagnostic properties here (Kollmeier and Koch, 1994).
Figure (5): Schematical view of the central auditory pathway (Kollmeier, 2008).
The two main components of the vestibular system are (1) the peripheral vestibular component and (2) the central vestibular component. The peripheral vestibular component is composed of three semicircular canals, two otolith organs, and cranial nerve VIII (Figure 6). The three semicircular canals are the anterior canal, the lateral canal, and the posterior canal. The purpose of the semicircular canals is to detect rotational movements of the head. For example, the semicircular canals are activated when you turn your head left and right or when you tilt your head up and down. The semicircular canals are able to respond to rotational head movements in the following way. When a rotational head movement occurs, it causes the fluid in the semicircular canals to move. The movement of the fluid causes hair cells in the semicircular canals to bend, and the bending of the hair cells stimulates cranial nerve VIII (Kinne, 2015).

Fig (6): Anatomical Review of the Vestibular System (Levine, 2015).
The two otolith organs are the utricle and the saccule. The purpose of the otolith organs is to detect linear movements of the head. For example, the otolith organs are activated when you walk forward and backward or when you jump up and down. When a linear head movement occurs, it causes the fluid in the otolith organs to move but otoconia (calcium carbonate crystals or “ear rocks”) in the otolith organs to lag behind. The lagging behind of the otoconia causes hair cells in the otolith organs to bend, and the bending of the hair cells stimulates cranial nerve VIII. Cranial nerve VIII is comprised of the cochlear nerve (for hearing) and the vestibular nerve (for balance). The purpose of the vestibular nerve is to transmit balance-related information from the semicircular canals and the otolith organs to the central nervous system (Kinne, 2015).

Fig (7): Sensors of the otolith organs and semicircular canals (Levine, 2015).
The central part is composed of the vestibular nuclei, the ascending tract, and the descending tract. The purpose of the vestibular nuclei is to process the balance-related information from the peripheral vestibular system (along with visual information from the eyes and somatosensory information from the muscles). Once the vestibular nuclear complex has made sense of all of this incoming information, it transmits outgoing information along the ascending tract to control the movement of the eyes and along the descending tract to control the movement of the muscles. Therefore, the two primary functions of the vestibular system are to stabilize the eyes during movements of the head and to stabilize the body during movements of the head. The eyes are primarily stabilized through a mechanism known as the vestibulo-ocular reflex (VOR), and the body is primarily stabilized through a mechanism known as the vestibulo-spinal reflex (VSR) (Kinne, 2015).
Hormonal and Physiological Changes during Pregnancy

Introduction:

Pregnancy is a dynamic, anabolic condition. As within several weeks of it, a new endocrine organ, the placenta, is formed and becomes ready for secreting hormones that affect the metabolism of all nutrients of the body. These changes in nutrients metabolism, in addition to changes in the anatomy and physiology of the mother, support fetal growth and development while maintaining maternal homeostasis and preparing for lactation (King, 2000).

Hormones of pregnancy:

- Some of these changes in pregnancy may be due to various protein and steroid structured hormones produced by fetoplacental unit and increased activity of maternal pituitary, thyroid and adrenal glands. While protein structured placental hormones are Human Chorionic Gonadotropin (HCG), Human Placental Lactogen (HPL), Human Somatomammotropin, Human Chorionic Thyrotropin and Human Chorionic Corticotrophin, steroid ones are progesterone and estrogen (Akkoca et al., 2014).

- The corpus luteum and placenta secrete hormones that maintain pregnancy and influence metabolism. Human chorionic gonadotropin (HCG) is detected in the serum and urine within a few days of implantation. Its serum concentration increases rapidly during early pregnancy to reach its highest level between 10-12 weeks of gestation (Akkoca et al., 2014) and declines by the 20th week, once estrogen and
progesterone levels increase. HCG maintains the corpus luteum in early pregnancy; it has few known effects on non-reproductive tissues (King, 2000).

- **Human placental lactogen (HPL):** in contrast to HCG, the production of human placental lactogen by the trophoblast does not start until the seventh to eighth week of gestation (Schindle, 2005); it is present only during pregnancy, with maternal serum levels rising in relation to the growth of the fetus and placenta. Maximum levels are reached near term, typically to 5–7 mg/L. Higher levels are noted in patients with multiple gestation. Little HPL enters the fetal circulation. HPL affects the metabolic system of the mother in the following manners:

  - In a bioassay human placental lactogen mimics the action of prolactin, yet it is unclear whether hPL has any role in human lactation.
  - **Metabolic**
    1) Decrease maternal insulin sensitivity (insulin resistance) leading to an increase in maternal blood glucose levels.
    2) Decrease maternal glucose utilization, which helps ensure adequate fetal nutrition. Chronic hypoglycemia leads to a rise in hPL.
    3) Increase lipolysis with the release of free fatty acids. With fasting and release of HPL, free fatty acids become available for the maternal organism as fuel, so that relatively more glucose can be utilized by the fetus. Also, ketones formed from free fatty acids can cross the placenta and be used by the fetus (Guyton and Hall, 2005).
These functions support fetal nutrition even in the case of maternal malnutrition. This hormone has weak actions similar to those of growth hormone, causing the formation of protein tissues in the same way that growth hormone, but 100 times more HPL than growth hormone is required to promote growth (Guyton and Hall, 2005).

**Estrogen and progesterone:**

Biosynthesis of the estrogens (ie, estrone, estradiol, and estriol) is a complicated process involving the mother, fetus, and placenta. In addition to influencing the uterus and other reproductive organs, estrogens cause a rise in certain binding hormones, which result in the elevation of total hormone concentrations, whereas the amounts of unbound and biologically active hormones remain unchanged. Estrogens also influence carbohydrate, lipid, and bone metabolism (Hytten and Chamberlain, 1980).

Estrogen levels start to rise shortly after implantation, with an additional rise at the sixth and seventh weeks of gestation, reflecting the takeover of estradiol production and secretion by the placenta. Estrogen levels continue to rise during the second and third trimester until delivery (Schindle, 2005). Its concentration reaches levels 3–8 times higher compared with levels in non-pregnant women. Estrogen-level increase during pregnancy is the result of a unique transaction between mother and fetus. The fetus produces adrenal dehydroepiandrosterone and dehydroepiandrosterone sulfate by using the pregnenolone produced by the placenta. Thereafter, the placenta metabolizes these hormones to produce androstenedione. Lastly, they are transformed to estrone and estradiol and released into maternal circulation (Doria et al., 2006).
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Progesterone concentrations rise progressively throughout pregnancy, of which the initial source is the corpus luteum, but placental sources of progesterone predominate later during pregnancy. Progesterone is the central hormone during the first part of pregnancy, and serves as a precursor for some fetal hormones. During physiologic pregnancy, progesterone levels are 4–6 times higher than in non-pregnant women. Deoxicorticosterone, one of its metabolites, is found in concentrations 1,000 times higher than in non-pregnant women, but the physiologic role of this hormone is still not known (Doria et al., 2006). Progesterone levels continue to rise until delivery in uncomplicated pregnancies (Schindle, 2005).

Progesterone relaxes smooth muscle, which causes atony of the uterus, gastrointestinal and urinary tracts. Although fetal demand for nutrients occurs primarily during the last half of gestation when more than 90% of the fetal growth occurs, adjustments in nutrient metabolism are apparent within the first weeks of pregnancy (Hytten and Chamberlain, 1980).

- **Thyroid and parathyroid glands**: They also change during pregnancy. Hypertrophy of the thyroid gland can be seen as a result of a relative iodine deficiency during pregnancy. This leads to increased secretion of thyroid hormones in the second trimester, resulting in an increased basal metabolic rate (BMR) (Nussbaum and Benedetto, 2006).

- Thyroxine-binding globulin (TBG) concentrations rise due to increased estrogen levels.
Hormonal and Physiological Changes during Pregnancy

- T4 and T3 increase over the first half of pregnancy but there is a normal to slightly decreased amount of free hormone due to increased TBG-binding.

- TSH production is stimulated, although in healthy individuals this is not usually significant. A large rise in TSH is likely to indicate iodine deficiency or subclinical hypothyroidism.

- Serum calcium levels decrease in pregnancy, which stimulates an increase in parathyroid hormone (PTH).

- Cholecalciferol (vitamin D3) is converted to its active metabolite, 1, 25 dihydroxycholecalciferol, by placental 1α-hydroxylase (Lazarus and Premawardhana, 2005).

**Pancreas:**

Changes in carbohydrate metabolism during pregnancy are achieved through increased production of insulin combined with resistance to its action, which increases with placental enlargement and the release of insulin antagonists such as HPL. These changes may be adaptive, providing an optimal environment for fetal growth and development, as glucose is the major substrate for the fetus. Maternal blood glucose levels determine fetal levels, which are normally 10–15% lower. Pregnancy is thus a diabetogenic state and susceptible individuals are at risk of developing gestational diabetes (Carlin and Alfirevic, 2008).
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**Pituitary:**

Like the other endocrine glands, the pituitary expands during pregnancy, increasing in size by 50%. Along with a significant weight increase of the anterior pituitary gland, there is an increased output of gonadotropins, corticotropin, adrenocorticotropic hormone (ACTH), and melanocyte-stimulating hormone (Gonzalez et al., 1988).

Prolactin (PRL) levels increase throughout pregnancy reaching its peak at term, and further changes may occur in the puerperium if breastfeeding is established. Prolactin appears to prepare the breasts for lactation by stimulating glandular epithelial cell mitosis and increasing production of lactose and lipids. Microprolactinomas (<10 mm) generally cause no problems in pregnancy, with the risk of symptomatic expansion of the order of 1.5%. Macroprolactinomas (>10 mm) can be more troublesome, with symptomatic expansion in 4% of treated and 15% of untreated patients (Molitch, 1985).

**Adrenal glands:**

Hypertrophy of the adrenal cortex can be seen with increased production and secretion of hormones, including cortisol, aldosterone, and dehydroepiandrosterone (Nussbaum and Benedetto, 2006). This rise in serum cortisol also causes relative gestational immunosuppression which leads to reactivation of latent viral infections (Torsiglieri et al., 1990).
 **Haematological system:**

In pregnancy, the haematological system undergoes changes in order to meet the demands of the developing fetus and placenta.

- **Blood volume:**

Plasma volume increases rapidly up to 10% above baseline by 7 weeks of gestation, and plateau by 32 weeks at 45–50% (Clapp et al., 1988). Red cell mass expansion also occurs but to a lesser degree and that cause accounts for the dilutional anaemia of pregnancy despite adequate stores (Cavill, 1995).

It is unclear whether or not this combination of changes gives a survival advantage as the decrease in blood viscosity may improve placental perfusion and reduce the risk of local thrombosis in early stage of pregnancy (Koller, 1982).

Plasma volume may also be important for normal fetal development as pregnancies complicated by growth restriction have measurably lower mean maternal plasma volumes compared with normal fetuses (Carlin and Alfirevic, 2008). There is also evidence that obstetric outcome and birth weight are correlated with the amount of plasma volume expansion (Murphy et al., 1986).

- **Blood components:**

The red cell ‘mass’ equates to the total volume of red blood cells in the circulation. It increases by 18–25% (Hyten, 1985), perhaps secondary to a rise in erythropoietin, in early pregnancy, and then falls after delivery (Harstad et al., 1992).
The increase in red cell volume provides for the extra oxygen demands of the mother and fetus. The lower end of the normal range for haemoglobin in pregnancy is 11–12 g/dL, and World Health Organization (WHO) recommends supplementation at levels <11.0 g/dL. White cell count increases in pregnancy from the first trimester and plateau at approximately 30 weeks of gestation as a result of selective marrow erythropoiesis. This causes a left shift with granulocytosis and more immature white cells in the circulation. The normal range for pregnancy is 5000–12000/mm³, although values as high as 15000/mm³ are not uncommon. The platelet count usually falls in pregnancy, possibly due to a dilutional effect and/or increased consumption secondary to endothelial-mediated activation (Carlin and Alffirevic, 2008).

- **Coagulation system:**

Pregnancy is a procoagulable state with alterations in both coagulation and fibrinolysis. The changes in the coagulation system during pregnancy appear to be aimed at minimizing blood loss at delivery. Unfortunately, these changes also predispose to thromboembolism, particularly in those with additional risk factors (Duhl et al., 2007).

In terms of absolute risk, pregnancy is associated with a four to six folds increase in venous thromboembolism compared with non-pregnant age-matched controls (Heit et al., 2005).

The circulating levels of factors VII, VIII, IX, X, XII, fibrinogen and Von willibrand factor increase, factor XI decreases, prothrombin and factor V remain unchanged. The natural anticoagulants antithrombin III and protein C levels are unchanged or increase and protein S levels fall (Hellgren, 1996).
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Fibrinolytic activity is known to decrease, mainly due to the marked increase in plasminogen activator inhibitors (PAI-I and PAI-2), and the combination of all these changes increases the risk of thrombosis during pregnancy and the puerperium (Davis, 2000).

- **Blood pressure:**

  An alternative method of exploring the hemodynamic changes is to examine the neurohormonal response to pregnancy. Nitric oxide (NO) and the prostaglandins are vasodilators that may be responsible for the observed drop in peripheral resistance, blood pressure and for changes in uterine and renal blood flow. These hemodynamic changes initiate additional baroreceptor-mediated neurohormonal events, including activation of the renin–angiotensin–aldosterone and sympathetic nervous systems, and release of natriuretic peptides. The renin–angiotensin system regulates salt and water hemostasis in the body so, there is an increase in both renin and angiotensin levels during pregnancy (August et al., 1990). This paradoxical increase in renin secretion occurs despite the normal expansion of extracellular volume during pregnancy.

  Activation of the sympathetic nervous system typically occurs in response to a decrease in peripheral vascular resistance and arterial pressure. During pregnancy, both opposing influences are active and the findings vary with regard to the extent and nature of net sympathetic activation during normal pregnancy and in patients with hypertensive disorders of pregnancy (Natrajan et al., 1982; Davey and Macnab, 1981).
The natriuretic peptides are involved in integration of cardiovascular and renal function. Atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) are released in response to volume overload states (atrial and ventricular distension), respectively. In healthy pregnant women, ANP and BNP levels increase during the course of pregnancy (Yoshimura et al., 1994).

**Changes in the Cardiovascular System:**

An increase in cardiac output is one of the most important changes of pregnancy. Cardiac output increases by 30–40% during pregnancy, and the maximum increase is attained around 24 weeks’ gestation (Mashini et al., 1987). The increase in heart rate occurs first (by the end of the first month of pregnancy) and plateaus at an increase of 10–15 beats per minute by 28–32 weeks’ gestation. Stroke volume increases by mid first trimester and progressively increases through the second trimester. Cardiac output can vary depending on the uterine size and maternal position at the time of measurement. The enlarged gravid uterus can cause aortocaval compression and reduced cardiac filling while the pregnant woman is in the supine position, cardiac output, heart rate, and stroke volume return to non-pregnant levels within 6–8 weeks after delivery (Robson et al., 1987). Diastolic and systolic blood pressure tend to fall during mid-pregnancy and then return to normal by week 36 (Thornburg et al., 2000).

The physiological changes in preload and afterload are accompanied by remodeling of the ventricles and atria. All four cardiac chambers increase in size from the first trimester to the end of the third trimester.
The dimensions decrease to baseline levels in the postpartum period. Left ventricular remodeling also manifests as increases in left ventricular wall thickness and mass. Increases in atrial size may contribute to atrial arrhythmias during pregnancy (Mone et al., 1996).

➢ *Changes in the Respiratory System:*-

The respiratory tract undergoes many changes during pregnancy, mediated initially by changes in the endocrine system and later by the enlarging uterus, in order to provide oxygen for increased maternal demands and for fetal physiology. These changes act to lower maternal pCO2 to half that of the fetus, thereby facilitating more effective gas exchange (Contreras et al., 1991).

Oxygen consumption increases by 30–50 mL/min, two-thirds of which covers additional maternal requirements (mainly the kidneys) and one-third is for the developing fetus. Despite this increase, pCO2 does not vary greatly and is approximately 13.6 kPa at term. PCO2 is also lower in the supine position; therefore, blood gases should be collected while sitting. Increased oxygen consumption is associated with a greater increase in carbon dioxide production, presumably due to the increase in carbohydrate to fat metabolism in pregnancy. Progesterone may lower the threshold and/or increase the sensitivity of the respiratory centre to carbon dioxide, or act independently as a primary stimulant of these two mechanisms (Carlin and Alfirevic, 2008).

➢ *Changes in the Renal System:*-

The glomerular filtration rate is increased during pregnancy because of increased renal plasma flow. A rise in the filtration rate
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decreases plasma blood urea nitrogen (BUN) and creatinine concentrations by about 40–50%, to approximately 8–9mg/dL and 0.5–0.6 mg/dL, respectively. Tubular reabsorption of sodium is increased. However, glucose and amino acids might not be absorbed as efficiently; hence glycosuria (up to 300 mg/day) and aminoaciduria may develop in normal gestation. The renal pelvis and ureters are dilated, and peristalsis is decreased. Physiological diuresis during the postpartum period occurs between the second and fifth days. The glomerular filtration rate (GFR) and BUN concentration slowly return to non-pregnant values by the sixth postpartum week (Jeyabalan and Conrad, 2007).

➢ **Metabolic changes during pregnancy:**

During pregnancy there is increased basal metabolic rate (BMR) which is due to the increased oxygen consumption, increased cardiac output and expansion of blood volume. There is 4 litre increase in the total body water. In the first and second trimester, it’s mainly the plasma volume and third trimester; it’s the extra-vascular fluid volume. This causes boggy mucus membrane and dependent extremity edema. Postpartum, there is a rapid decrease in the plasma volume and slow decrease in the interstitial fluid (Torsiglieri et al., 1990).

➢ **Musculoskeletal:**

Neuromechanical adaptations to pregnancy refer to the change in gait, postural parameters, as well as sensory feedback, due to the numerous anatomical, physiological, and hormonal changes women experience during pregnancy. Such changes increase their risk for musculoskeletal disorders and fall injuries. Musculoskeletal disorders
include lower-back pain, leg cramps, and hip pain. Pregnant women fall at a similar rate (27%) to women over age of 70 years (28%). Most of the falls (64%) occur during the second trimester. The root causes for these falls are not well known. However, some factors that may contribute to these injuries include deviations from normal posture, balance, and gait (Dunning et al., 2003). To positionally compensate the additional load due to the pregnancy, pregnant mothers often extend their lower backs. As the fetal load increases, women tend to arch their lower backs, specifically in the lumbar region of their vertebral column to maintain postural stability and balance. The arching of the lumbar region is known as lumbar lordosis (Whitcome et al., 2007).
Hearing and Vestibular Disorders in Pregnancy

These sophisticated physiological changes, in the cardiovascular system, hematological system, endocrine system and others, especially for estrogen and progesterone levels influence the auditory system. They may impact the circulation of cochlea and/or cochlear fluid homeostasis (Al-Mana et al., 2008).

Hormonal fluctuations alter the maintenance of the chemical composition of both perilymph and endolymph in the inner ear, and the ion transport processes between them. The link between the perilymph and the endolymph is maintained by hydrostatic pressure via the cochlear aqueduct. Therefore, the patency of the cochlear aqueduct is key, to whether the effect on the hearing will be to a greater or lesser extent, with the changes in composition and pressure of the cerebrospinal fluid (CSF) during pregnancy (Goh and Hussain, 2012). They can cause cochlear and/or vestibular disorders as tinnitus, hearing loss, autophony, imbalance, vertigo and dizziness. They also can cause external ear canal disorders (otitis externa and bleeding polyps) (Havas and Conjoint, 2012; Schimdt et al., 2010).

It is important for audiologist to be aware of these conditions and the underlying process in order to be able to manage them effectively and safely considering the health of both the mother and the unborn child. With the potential for symptoms to be early warning signs for impending crisis, prompt recognition of these may prevent unnecessary harm and lead to an earlier diagnosis of conditions (Kumar et al., 2011).
1- **Tinnitus:**

Tinnitus is the perception of sound (ringing, whooshing, buzzing or pulsing) in the ears or head when no external source is present (*Cummings et al., 1998*). The impact on quality of life ranges from annoying to unbearable and disabling. The management of patients with tinnitus is a daunting task due to the vast number of potential etiologies. For example, tinnitus has been associated with otosclerosis, Meniere's disease, hypertension, autoimmune disorders, vascular pathology, intracranial neoplasms, acoustic neuromas (ANs), foreign bodies, psychiatric disorders, and among other conditions (*Jafek and Stark, 1996*).

Tinnitus is the more common audiological complaint during pregnancy with proposed theories including hyperdynamic circulation, increase in perilymphatic fluid pressure and hormonal changes. It is also may be due to Eustachian tube dysfunctions (EDTs) or alterations in the auditory pathway (*Sennaroglu and Belgin, 2001; Tsundo et al., 1999*).

The prevalence of tinnitus during pregnancy is reportedly 25%, or approximately double that in non-gravid controls (*Gurr et al., 1993*). Most of these episodes are benign and resolve with delivery. However, several medical conditions presenting with tinnitus also occur with increased severity and/or prevalence during pregnancy. Thus, it is essential that the physician identify patients who require a more extensive work-up for "ringing in the ears" (*Haas et al., 2007*).
More significantly, tinnitus has been speculated as an early warning sign of gestational hypertension or preeclampsia (PE), and it would be prudent for such patients to be carefully monitored (Shapiro et al., 1999).

Severe tinnitus has even led to an emergency cesarean delivery at 34 weeks, with total resolution postoperatively (Mukhophadhyay et al., 2007). As a result, physicians caring for gravid patients should be especially aware of all possible etiologies of this complaint. In the majority of patients, tinnitus resolves with delivery and represents no underlying pathology (Tsunoda et al., 1999).

2- **Autophony and Aural fullness:**

Eustachian tube dysfunction (ETD) has been estimated to affect between 5-30% of pregnant women and can be variable in terms of its symptomatology and this can be either tubal obstruction or patulous Eustachian tubes (PET). The pathophysiology is related to the edema of the mucosa (Derkay, 1988).

The signs and symptoms usually begin after first trimester and depend on which form of dysfunction exists. Women with tubal obstruction will complain of clogged or popping sensation in their ears with muffling of sounds and serous otitis media (SOM) may develop in severe cases. On the other hand women with patulous tubes usually manifest intermittent symptoms consisting of autophony and a roaring sensation in their ears that is synchronous with respiration and is worst in upright position or with exercise (Derkay, 1988).
3- **External canal disorders:**

Otitis externa in pregnancy occurs mainly in the third trimester. Eccrine activity may be noticeably increased during pregnancy. Although there is considerable individual variation, the rate of sebum production increased during pregnancy and return to normal after delivery. The wet external auditory canal with the overproduction of sebum are well-known predisposing factors for otitis externa (*Al-Zubiadi, 2012; Millington and Graham-Brown, 2010*).

External canal: Granuloma gravidarum or pregnancy tumor, describe pyogenic granuloma (bleeding polyp) occurring in the prenatal period. These benign lesions can occur up to 5% of pregnancies (*Sills et al., 1996*).

They occur more frequently in the second and third trimester. The pathogenesis of these lesions may be hormone mediated with the dilation and proliferation of blood vessels, while the sites of predilection tend to be the oral cavity (*Courney et al., 2003*).

4- **Hearing loss:**

Hearing loss manifesting in pregnancy does not appear to be a commonly reported problem (*Kumar et al., 2011*).

The pregnancy may be associated with a low frequency sensorineural hearing loss (SNHL) and tolerance problem mimicking cochlear pathology. There is a decrease in hearing level for 125, 250 and 500 Hz, beginning in the first trimester and increasing over second and third trimesters. For frequencies of 1000 Hz and above, there were no
significant difference in between the trimesters and the postpartum period. The speech audiometric findings are normal during pregnancy. However, this low frequency hearing loss returns to normal in the postpartum period (Sennaroglu and Belgin, 2001).

Sex hormones (estrogen and progesterone) exert regulatory influences on central nervous system (Sharma et al., 2011; Wharton and Church, 1990). Some research indicates that there are estrogen receptors (ERα and ERβ) in the cochlea in both humans and animal models (Stenberg et al., 2001). Estrogen receptors can be found in the spiral ganglion, outer hair cells, inner hair cells, stria vascularis, and cochlear vessels (Lee and Marcus, 2001).

They may modulate auditory transmission, fluid electrolyte balance, and blood flow in the cochlea. In addition, estrogen may impact auditory function at different levels of the central nervous system (CNS) by modulating GABA-ergic, serotonergic and glutamatergic systems (Woolley et al., 1997). It has an excitatory and protective effect on the auditory system (Guimaraes et al., 2004). However, progesterone has a mainly inhibitory action on the auditory system (Hou and Wang, 2011).

The production rates for these hormones in non-pregnant women (estrogen 0.02–0.1 mg/24 h and progesterone 0.1–40 mg/24 h, respectively) show considerable increase in near term pregnant women (estrogen 50–100 mg/24 h and progesterone 250–600 mg/24 h). While creating optimal conditions for pregnancy, these hormonal changes cause an increase of 6.5 litre in extra-cellular and 1.25 litre intra-cellular fluids. As a result of these osmotic changes in body, water and sodium retention
takes place. As it is evident from previous studies that circulating sex hormones affect the sensorineural hearing system, one may expect changes in hearing levels with so much fluid retention during pregnancy (Sharma et al., 2011).

It is also reported that peripheral edema can be induced by some physiological changes in most normal pregnancies. These changes include obstruction of venous return by the gravid uterus, peripheral vasodilatation, increased plasma volume, decreased colloid osmotic pressure, and increased vascular permeability. When peripheral edema occurs in the membranous labyrinth, and it affects the cochlea just like Meniere’s disease then sudden sensorineural hearing loss (SSNHL) may come about (Sennaroglu and Belgin, 2001).

Factors such as pregnancy related stress and immune-mediated disorders also may play a role in initiation of SSNHL (Hou and Wang, 2011; Schreiber et al., 2010).

As pregnancy is a hypercoagulable state with alterations in both the coagulation and fibrinolysis systems (Hellgren, 1996). These changes induce the risk of venous thromboembolism, which can rise four to six-fold. When a thromboembolic episode occurs in the cochlear arteries or veins, ischemia or congestion will result, and SSNHL may be evoked (Heit et al., 2005).

A perilymph fistula secondary to a fracture of the stapes footplate occurring at the time of exertion of parturition, may lead to a sudden onset of hearing loss (Whitehead, 1999).
SSNHL during pregnancy that is caused by the changes during pregnancy may be a new disease that is different from the usual SSNHL, similar to the definition of “pregnancy-induced hypertension.” This disease can be called “pregnancy-induced sudden sensorineural hearing loss (Hou and Wang, 2011).

Furthermore, SSNHL occurrence during pregnancy could occur again in the same patient who had SSNHL in a previous pregnancy (Pawlak-Osinska et al., 2009). Part of the SSHNL work up should include continued monitoring of the patient (Georgescu et al., 2014).

A study conducted by Lavy, (1998) reported sudden onset of sensorineural hearing loss in two cases associated with pregnancy.

Kanadys and Olesczok, (2005) also reported a case of sudden sensorineural hearing loss in 23-years-old healthy women during an uncomplicated pregnancy.

Hou and Wang, (2011) reported two cases of SSNHL in pregnancy, were investigated and their clinical features were analyzed. One patient had low-frequency fluctuating sensorineural hearing loss and the other had profound sensorineural hearing loss (SNHL). Both of them had hearing loss in the sixth week of pregnancy. Hearing of the first patient returned to normal after delivery, but hearing of the second patient showed little improvement. They supposed that their SSNHL was related to pregnancy.

On the other hand, Tsunoda et al. (1999) reported that pure tone audiometry showed normal hearing in all of his pregnant cases.
Sharma et al. (2011) studied hearing loss in pregnant women grouped in four groups (I, II, III the three trimesters, IV postpartum) and (V control group) which showed gradual decrease in hearing acuity at low frequencies (125, 250, 500, 1000) from the first to third trimester and returned to normal in the postpartum period (Figure 8).

**Figure (8):** Showing mean pure tone thresholds for pregnant and non-pregnant subjects (Sharma et al., 2011).

Recognition and proper management of SSNHL in pregnant women is important because emotional status of the patients might impede upon pregnancy development. Appropriate audiological evaluation is mandatory in order to exclude tumoral etiology of SSNHL. Hearing improvement after delivery in majority of cases is suggestive of reversible inner ear process (Georgescu et al., 2014).
The detection of hearing level and hormones should be carried out frequently before and during this period, and checking the amount of fluid in the cochlea postpartum. After that, we can obtain more substantial evidence to determine the etiologies and mechanisms of SSNHL during pregnancy (Hou and Wang, 2011).

SSNHL can be treated with Dextran 40, which has been reported to be safe and effective in improving hearing levels (Wang and Young, 2006), with the aim of decreasing blood viscosity and reducing cochlear hypoxia (Lamm and Arnold, 1999; Lacy and Wright, 1992). Accordingly, sudden hearing loss patients treated with intravenous dextran administration within 7 days after the onset of their hearing loss showed marked hearing improvement (Redleaf et al., 1995).

**Figure (9):** Revealed a low-frequency mild SNHL in 33 years old patient with a 31 week pregnancy.  
**Figure (10):** Audiometric evaluation revealed normal limit thresholds in all frequencies one week injection of 250 ml Dextran 40 twice a day (Georgescu et al., 2014).
5- Vestibular disorders

Manifestations of vestibular disorders include: imbalance, gait deviations, gait instability, a feeling of floating, rotation, falls, nausea and vomiting. These disorders affect the life routine; family, social and professional relations (Pedalini et al., 1999).

Vertigo and dizziness are frequently experienced during pregnancy and are among the most common complaint from pregnant women to primary care (Black, 2002).

The release of neurotransmitters can alter the biochemical control of the inner ear, since these mediators can be released during pregnancy; it is possible that there is an increase in neurotological symptoms. Such fact can be responsible for the frequent complaint of dizziness during pregnancy (Schmidt et al., 2010).

Pregnancy is characterized by numerous alterations which happen to women as hormonal, anatomical, cardiovascular and pulmonary changes, edema, and weight gain which can affect the muscle-skeletal system and posture (Ireland and Ott, 2000).

The increase in the instability complaint in the following trimesters and the tendency to fall in the third trimester can be explained by the increase in body weight and postural change which occurs and increases as gestation progresses (Ribas and Guirro, 2007).

Uchide et al. (1997) suggested that the coincidence of the drop in osmolarity and the increase in vertigo spells as being the possible effect factor of pregnancy on Meniere's disease. Therefore, changes in the osmotic fluid can affect the inner ear during pregnancy.
Nausea and vomiting, common symptoms during pregnancy often are regarded as an unpleasant but normal part of pregnancy during the first and early second trimesters. Nausea and vomiting of pregnancy (NVP) occurs in approximately 75–80% of pregnant women. The exact etiology and pathogenesis of NVP are poorly understood and are most likely multifactorial (Badell et al., 2006).

Nausea and vomiting of pregnancy (NVP) may be precipitated or influenced by the hormonal or fluid-volume changes occurring in the vestibular system (Black, 2002). A physiological reduction in serum osmolality may affect the vestibular system via changes in the perilymph and therefore lead to an exacerbation of nausea and vomiting during pregnancy (Uchide et al., 1997).

Also Koch, (1997) has shown that vestibular-mediated nausea and vomiting may be induced by hormonally mediated change in osmolarity in early pregnancy.

The period of pregnancy that corresponds to the onset of nausea and vomiting of pregnancy is characterized by a significant decrease in plasma osmolarity. This has been attributed to decreased sensitivity to vasopressin which may be linked to the action of HCG (Davison et al., 1988).

Two observations support the involvement of b-HCG in the etiology of NVP. One is the temporal relationship between the peak in maternal b-HCG levels and the peak of nausea and vomiting. The other is the more severe nausea and vomiting seen in women with conditions such
as multiple gestations or molar pregnancy which tend to involve elevated levels of b-HCG (Goodwin, 2002).

Estrogens, specifically estradiol, have also been implicated in influencing NVP. One group of authors demonstrated that women with hyperemesis gravidarum had statistically significant elevated levels of estradiol in the first trimester compared with controls (Depue et al., 1987). Lower estradiol levels are associated with cigarette smoking, so smokers are less likely to experience hyperemesis gravidarum (Bernstein et al., 1989).

**Exogenous estrogens** also have induced nausea and vomiting when given to some non-pregnant patients. For example, some women who take oral contraceptives are at increased risk for developing nausea and vomiting when pregnant (Whitehead et al., 1992).

**Progesterone** may disrupt gastrointestinal neuromuscular function resulting in nausea and vomiting. Progesterone decreases smooth muscle contractility and may also cause gastric dysrhythmias or delayed emptying (Depue et al., 1987).

In a certain study, Electrogastrograms of 32 pregnant women with nausea and vomiting indicated that 26 of the women had gastric dysrhythmias including bradygastrias or tachygastrias. These findings were confirmed by a trial in which non-pregnant women received estrogen and progesterone in levels equal to those of women during the first trimester of pregnancy. The authors found that the non-pregnant women experienced slow-wave dysrhythmias seen in pregnant women with nausea and vomiting (Walsh et al., 1996). Both of these studies
support the hypothesis that progesterone and/or estrogen at elevated levels during pregnancy might disrupt normal gastric myoelectric rhythms and contribute to the etiology of NVP.

Treatment of NVP ranges from dietary and lifestyle changes to vitamins, antiemetics and hospitalization for intravenous therapy. Treatment generally begins with non-pharmacologic interventions; if symptoms do not improve, drug therapy is added (Badell et al., 2006).

Postpartum vertigo is associated with a multitude of causes. Some cases reports suggest the abrupt changes in middle ear and intracranial pressure (ICP) secondary to Valsalva maneuver during labor, can lead to trauma to the vestibular system, including perilymph fistula formation (PLF) (Gleeson and Williams, 1989) and superior semicircular canal dehiscence (SSCD) (Watters et al., 2006). The resulting symptoms of both conditions can include vertigo, disequilibrium, aural fullness, autophony, tinnitus, and conductive hearing loss with enhanced bone conduction (Friedland, 2009; Ogutha et al., 2009).

Treatment can begin with bed rest, stool softeners, and avoidance of Valsalva maneuver. However, delayed treatment may result in permanent hearing loss and therefore, exploratory tympanotomy and repair should be undertaken as soon as possible (Pullen, 1992).
**Special Medical Disorders during Pregnancy and their Effects on Hearing & Balance**

**A- Pre-eclampsia**

Pre-eclampsia (PE) complicates 2–7% of pregnancies and may greatly contribute to maternal morbidity and mortality (Zhao et al., 2012). It is a systemic disease that affects multiple organs, such as the liver, kidneys, heart, and central nervous system (C.N.S). It is characterized at the cellular level by endothelial dysfunction, cell damage, activation of pro-inflammatory cytokines, coagulation cascade and reduced maternal systemic organ perfusion (Hakim et al., 2013).

Pre-eclampsia (PE) has recently been shown to be related to an imbalance of circulating angiogenic factors (high circulating levels of antiangiogenic and low levels of circulating proangiogenic factors, such as placental growth factor and vascular endothelial growth factor). The relatively high concentrations of antiangiogenic factors are believed to trigger vascular endothelial cell injury in the liver, kidney, and brain, as well as in the placenta itself. Endothelial dysfunction and injury most likely result in hypertension, proteinuria, and other systemic manifestations of the syndrome (figure 11) (Steinberg et al., 2009). Increased vascular resistance and multifocal vasospasms have been reported in pre-eclampsia cases (Zeeman, 2009; Suzuki et al., 2003).
Figure (11): Summary of the pathogenesis of preeclampsia: Immune factors (such as AT1-AA), oxidative stress, NK cell abnormalities, and other factors may cause placental dysfunction, which in turn leads to the release of anti-angiogenic factors (such as sFlt1 and sEng) and other inflammatory mediators to induce hypertension, proteinuria, and other complications of preeclampsia (Wang et al., 2009).
Normal blood supply to the cochlea is essential for sustaining endocochlear potential, ion transport, endolymphatic fluid balance and for preventing toxic substances from entering the cochlea (Shi, 2011).

Sensorineural hearing loss (SNHL) occurs when there is damage to the inner ear, cochleo-vestibular nerve or brain. The cochlea is principally supplied by the inner ear artery (labyrinthine artery) which is usually a branch of the anterior inferior cerebellar artery. Cochlear microcirculatory disorders associated with impaired local oxygenation have been considered to be a major pathogenic factor in sensorineural hearing impairment. So, reduced cochlear blood flow is a main contributor to hearing loss (Chau et al., 2012; Nakashima et al., 2003).

The majority of SNHL cases are caused by abnormalities in the hair cells (thousands of tiny nerves of the organ of corti in the cochlea) which are strikingly vulnerable to ischemia (Terzi et al., 2014).

It was previously believed that pre-eclampsia was a self-limiting condition that resolves after delivery of the placenta with insignificant long-term sequelae for the mother, although fetal morbidity might be great. However, recent data have shown that maternal endothelial dysfunction may persist for years after the episode, and that women who have suffered from preeclampsia have higher rates of cardiovascular morbidity and mortality than women who have not had the disease (Gastrich et al., 2010; Irgens et al., 2001).

Even if pre-eclampsia resolves after delivery, cochlear damage and permanent hearing loss remain unchanged in pre-eclamptic patients (Baylan et al., 2010).
Altuntas et al. (2012) studied cochlear function in 52 patients with preeclampsia during the postpartum period (within one week), and they reported hearing loss in only one (1.9%) patient.

Baylan et al. (2010) also studied cochlear function in 40 patients with pre-eclampsia and 30 control subjects during pregnancy and the postpartum period (within 3 weeks), and they found a significantly increased risk of hearing loss at low frequencies during pregnancy and the postpartum period in the patients with pre-eclampsia.

Bakhshaee et al. (2008) studied cochlear function in 37 patients with pre-eclampsia and 38 control subjects during pregnancy and the postpartum period (within 2 weeks), and they reported hearing impairment in five out of 37 (13.5%) patients during pregnancy. Cochlear function normalized in all of those patients during the postpartum period.

Gurr et al. (1993) theorized that hypertension (as seen in preeclampsia) could be the underlying etiology of tinnitus via increased intracranial pressure (ICP) influencing perilymphatic fluid pressure.

Resolution of preeclampsia by delivery may be curative. Nonetheless, evaluation of a patient with tinnitus and suspected preeclampsia should be confirmed for standard management for preeclampsia (Haas et al., 2007).

The risks to both mother and neonate can be reduced by appropriate supervision and therapy. Close monitoring of maternal and fetal welfare will help to determine the optimum time for delivery. Maternal hypertension should be controlled with agents considered to be well tolerated in pregnancy. Following the index pregnancy, all patients
with early and/or severe hypertension (HTN) should be investigated for an underlying cause (Siddiqui et al., 2010).

The supplementation with 1.5 gram of calcium per day appears effective as well in the prevention of preeclampsia, especially in the malnourished and young patents (Deruelle et al., 2010). Calcium supplementation appears to approximately halve the risk of pre-eclampsia (PE), to reduce the risk of preterm birth and to reduce the rare occurrence of the composite outcome “death or serious morbidity” (Hofmeyr et al., 2010).

Insufficient data is currently available to recommend antioxidant supplementation. Low molecular weight heparin is potentially beneficial in the prevention of preeclampsia. Nitric oxide (NO) or NO releasers are not effective and can cause headaches. Diuretics reduce the birth weight without improving the incidence of PE (Deruelle et al., 2010).
B- Otosclerosis.

Otosclerosis is a localized progressive disease of bone remodelling. The stapes bone has a footplate and a head joined by two arches. In otosclerosis, new spongy bone forms around the footplate and may cause the stapes to become fixed (figure 12). There is resorption of the stable otic capsule followed by a reparative phase with bone deposition. Hearing is impaired, as movements of the ossicles are essential for sound conduction through the middle ear. Otosclerosis may also involve the inner ear sensory organ (the cochlea) (Chole and McKenna, 2001).

![Otosclerosis](image-url)

**Figure (12):** Otosclerosis involving stapes footplates as demonstrated by a circle (Zarandy and Rutka, 2010).

The cause of otosclerosis is multifactorial; several dominant factors may coexist in the patient: genetics, exposure to the measles virus, vitamin D deficiency, race, and pregnancy. Genetic foci have been recently investigated and found in familial otosclerosis. In this autosomal
dominant type of otosclerosis, there are thought to be at least three gene foci (Van Den, 2002).

Hearing loss (HL) is conductive in stapedial otosclerosis, although an apparent sensorineural element may be noted at 2000 Hertz (Hz) on pure tone audiometry. This is the basis of the so-called Carhart's notch, an important diagnostic clue, and reasons for this being physiological and complex. This does not however indicate a cochlear involvement (Browning and Gatehouse, 1984). Generally, tympanogram findings will be normal or nearly normal (type As) figure 13 (Kotb, 2011).

![Tympanogram and Audiogram](image)

*Figure (13): Audiological investigations in Otosclerosis (Kotb, 2011).*

When the otosclerotic lesion extends from the footplate of the stapes to involve the cochlea, the hearing loss becomes of mixed type, the conductive element from stapes fixation and the sensorineural element from the cochlea. It may rarely be purely sensorineural. Tinnitus is usually on one side and is not necessarily associated with cochlear degeneration. It may be mild and is usually not troublesome. Dizziness
may occur. This is usually due to a sudden change in the position of the patient's head and is self-limiting. The duration of the vertigo is not quantifiable (*Hussain and Gibbin, 2008*).

The onset is usually insidious and disease progression is characteristically slow although it may be rapid at times especially in pregnancy. The age of onset, clinically, is usually between the ages of 20 and 30 years (*Hussain and Gibbin, 2008*).

Otosclerosis has been known to worse and/or present during pregnancy. *Barton, (1945)* initially reported this phenomenon stating that nearly 66% of patients with otosclerosis experienced an exacerbation of symptoms during pregnancy. Tinnitus was so severe in some cases that therapeutic abortion was recommended as treatment.

While the exact etiology is unknown, it is hypothesized that the rise in circulating estrogen during pregnancy favors osteosclerotic formation as estrogens are well-known stimulators of osteocytic activity and may play a dominant role during ossification of an otospongyotic bone lesion, thus increasing the fixation of the stapes to the oval window (*Hansen et al., 1986*).

This may explain the onset of a conductive hearing loss (CHL) due to otosclerosis during pregnancy. Some control of otosclerosis progression can be achieved by prevention of conception (*Arnold et al., 1996*).

A large, retrospective study of pregnant women studied by Gristwood and Venables reported on a subjective impression of deterioration of hearing, particularly with subsequent pregnancies. They reported that the chance in female patients with bilateral pregnancy-
related otosclerosis of subjective deterioration of hearing varied from about 33% after one pregnancy to about 63% after six pregnancies (*Gristwood and Venables, 1983*).

*Hall et al. (1974)* also demonstrated that only 8% of the otosclerotic patients had an aggravation of hearing problems during their pregnancy.

It was suggested that there was a relation between otosclerosis and the puerperium, and that otosclerosis was a true infective puerperal osteitis. Pregnancy or the puerperium possible intoxicated the temporal bone. The temporal bone might be affected by bony changes which occurred in the puerperium and in that way otosclerosis was produced (*Tange, 2013*).

Otosclerosis can be differentiated from benign tinnitus, most notably by a measurable hearing loss. Weber tuning-fork test findings will lateralize to the affected ear and there may be a hearing loss of 55 to 60 dB (*Jafek and Stark, 1996*).

Although this condition is generally irreversible, symptoms often improve postpartum. Referral to an audiologist for diagnosis and treatment is recommended, as hearing aids allow some return in function to the affected ear. Surgical treatment is available, but should be deferred until after delivery. Medical treatment with sodium fluoride is contraindicated in pregnancy secondary to potential adverse effects on fetal bone formation (*Torsiglieri et al., 1990; Hansen et al., 1986*).
**C-Meniere's disease**

Meniere disease, or endolymphatic hydrops, is characterized by a quartet of symptoms: low-pitched tinnitus, aural fullness, vertigo, and low-frequency hearing loss (fig 14a). It typically presents during the childbearing years (third and fourth decades) (*Hansen et al., 1986*).

Vertigo has been shown to occur more frequently during pregnancy, so complaints of vertigo and tinnitus do not confirm the diagnosis of Meniere's disease. Rather, the presence of a measurable hearing loss (with fluctuation over time) is necessary to establish Meniere's disease as the cause of the tinnitus (*Torsiglieri et al., 1990*).

**Figure (14a):** Audiogram (hearing test) typical of early Meniere's disease on the right side (x=left, o=right). There is a low-tone sensorineural hearing loss (*Hain, 2015*).
Figure (14b): Audiogram typical of middle-stage Meniere's disease, again on the right side. Hearing is reduced at all frequencies, but more so at high and low frequencies (Hain, 2015).

Figure (14c): Audiogram typical of late-stage Meniere's disease, again on the right side. Hearing is flat, and unaidable on the right side (Stages of Meniere's disease) (Hain, 2015).
Meniere's disease can definitely be intensified by pregnancy. *Uchide et al.* (1997) reported a 10-fold increase in the number of Meniere-associated vertigo attacks during early pregnancy. These authors speculate that exacerbation of Meniere's disease is secondary to the physiologic decrease in serum osmolality beginning in the fifth to 16th week of pregnancy. This drop in serum osmolality (of up to 10 mosm/kg) is hypothesized to cause an influx of water into the endolymphatic space (figure 15), thus exacerbating the endolymphatic hydrops (*Semaan et al.*, 2005).

It may have some association with hormonal changes (which cause fluid retention). That physiological reduction in serum osmolality may affect the vestibular system via changes in the perilymph and therefore lead to an exacerbation of symptoms during pregnancy (*Uchide et al.*, 1997).

![Figure 15: Meniere's disease (endolymphatic hydrops) (Yamane et al., 2010).](image_url)
SP/AP ratio in electrocochleography (ECoG) is the most sensitive parameter in diagnosing the cases of Meniere's disease. The ears with a SP/AP ratio larger than 0.37 are regarded as positive for Meniere's disease (*Shin and Yukio, 1994*). *Uchide et al. (1997)* also reported a case of 31 years old pregnant women, audiogram taken during and after pregnancy showed fluctuating hearing loss of the left ear. The eye tracking test showed normal pattern. ABR showed no significant difference between right and left ear. ECoG revealed negative dominant SP (-SP/AP = 41%) (figure 16) and the caloric test show no canal paresis except left directional preponderance.

![Figure 16: ECoG revealing negative dominant SP (-SP/AP = 41%) (Uchide et al., 1997).](image)

There is a definite relationship between ECOG and symptoms associated with Meniere's disease. Presence of hearing loss with aural fullness is a strongest predictor of a positive ECOG which shows enlarged SP/AP ratio (*Ferraro et al., 1985*).
The multifactorial pathology leading to Meniere's disease can pose difficulties in management in all patients, with pregnancy adding in additional considerations. If the diagnosis is uncertain and imaging is required, it is recommended to wait until the postpartum period before undertaking a magnetic resonance imaging (MRI) scan due to a lack of conclusive studies on the safety of MRI in pregnancy (Hylton, 2000).

Conservative methods of management of Meniere's disease, with respect to dietary changes of salt and caffeine reduction, pose no risk to the mother or fetus and could therefore be used as first line of management. Although there are no adverse event data published to preclude the use of betahistine in these patients, it should be used with caution. Prochlorperazine for acute episodes of vertigo should also be used with caution as antipsychotics have been linked to extrapyramidal effects in the neonate when used in the third trimester; however, most evidence indicates no increased risk (Kumar et al., 2011), also during acute attack of vertigo, dimenhydrinate (Dramamine) and meclizine (Antivert) can be safely given in pregnancy in minimal doses. Diuretics and histamines are avoided as it causes hypotension, hypovolemia and lowers cardiac output. For intractable vomiting metaclopromide can be used (Sherlie and Varghese, 2012).

Isosorbide is also said to be effective for the control of Meniere's disease (Kakigi et al., 1995), It is a non metabolizable dehydric alcohol formed by removing two water molecule from one sorbitol molecule and is said to be able to maintain an osmotic gradient between outer and inner sac for 4 hrs, resulting in relief of endolymphatic hydrops .The use of isosorbide is reported to be safe in pregnancy (Kumar et al., 2011).
**D. Patulous Eustachian Tube (PET).**

*Definition:*

The impairment of the physiological protection function of the Eustachian tube (ET) causes a distressing autophony and aural fullness, the so-called syndrome of the patulous Eustachian tube (PET) (*Iwano et al., 1991; Bluestone and Canteki, 1981*).

Eustachian tube (ET) is closed at rest and is actively opened only under controlled conditions during respiratory rest in the nasopharynx. If the Eustachian tube is opened outside this limited range of time, there is a pathological communication between the nasopharynx and the tympanic cavity. This pathological communication causes an intermittent or constant transfer of pressure fluctuations from the pharynx towards the middle ear (*Heermann, 1988*).

The intensity and frequency of complaints vary between individuals. In cases of rare symptoms or of low-intensity symptoms, it is enough to inform the patient of the diagnosis and of the harmlessness of the symptoms. On the other hand, there are patients who are so distressed by PET symptoms, that psychological problems arise, resulting in the inability to work. These patients need for initiation of specific diagnostics and management therapy (*Dornhoffer et al., 2014*).

Many of these patients can modify their symptoms by increasing the venous pressure of the mucosal vessels and the surrounding venous plexus of the Eustachian tube by compression of the jugular veins or by an inclined head position. In many cases “sniffing” is a useful maneuver
to actively release the distressing symptoms. During sniffing the Eustachian tube and the middle ear are evacuated by a forced nasal inspiration, causing stiffening of the eardrum and the ossicular chain. This effect is temporary, and sniffing will be repeatedly used. The risk of a long-term sniffing habit is to produce a chronic negative middle ear pressure and the development of middle ear diseases (Dornhoffer et al., 2014).

*Etiology:

Factors that may influence the occurrence of a PET include sudden weight loss, hormonal changes (e.g. as in pregnancy or use of birth control pills), stress, fatigue, the inappropriate use of decongestants, and temporo-mandibular joint (TMJ) syndrome (O'Conner and Shea, 1981).

Pregnancy can be a cause of patulous Eustachian tube due to the effects of pregnancy hormones on surface tension and mucus in the respiratory system (Hillman and Edawrd, 1995). Estrogen is supposed to have an impact upon the viscosity of the intratubal mucus as well as on the compliance of the tubal cartilage. In addition, increased estrogen causes increased fat metabolism leading to a reduction of the Ostmann’s fat pad. The reduction of the Ostmann’s fat is considered to be one of the main factors causing the symptoms of PET (Dornhoffer et al., 2014).

Miller, (1962) documented 17 female patients who during pregnancy developed symptoms of a PET. The largest series of patients was published by Plate et al. (1979) who examined 270 pregnant women. Of these, 19 appeared to have a closing failure of the Eustachian tube but only 5 showed the clinical symptoms of a PET. With the patient breathing through her nose, they noted the occurrence of impedance changes
synchronous with respiratory cycles. The symptoms were highly correlated to the level of estriol in the serum.

*Symptoms:

The most common PET symptom is a feeling of aural fullness and/or a plugged or blocked ear. Auditory symptoms include the report of autophony, or the phenomenon of being able to hear one's own voice more loudly than usual, and hearing a loud crackling sound when chewing. *Robinson and Hazell, (1989)* reported that vestibular symptoms and hearing loss can also occur because the PET allows excessive pressure changes to occur in the middle ear which are then transmitted to the inner ear through ossicular movement. They reported reducing vestibular symptoms in five of six patients by treatment of their PET condition. PET symptoms can either be continuous or episodic, occurring in one or both ears, and typically diminishing or disappearing when the patient is supine (*O'Conner and Shea, 1981*).

*Diagnosis:

Diagnosis of a PET is usually made through a combination of history and otoscopic or microscopic examination of the tympanic membrane (TM). It is usually possible to observe tympanic membrane movements occurring synchronously with inspiration and expiration; the result of transmission of intraoral air pressure changes to the middle ear through the patent Eustachian tube (*Henry and Di-Bartolomeo, 1993*).

Evaluation of the acoustic transfer function from the nasopharyngeal cavity to the middle ear via the patulous tube is important. Conventional ET function tests, such as tubo-tympano-aerodynamic-graphy (TTAG) and sonotubometry, or the recently reported
audiometry using nasally presented sound, can objectively evaluate the acoustic or pressure transfer functions via the patulous ET under the resting or swallowing conditions (Hori et al., 2007) and also nine-step inflation–deflation test and pressure chamber tests (Doyle et al., 2013).

MRI or CT can help confirm morphological changes of the paratubal structures (Figure 17) (Dornhoffer et al., 2014).

![Axial CT of the Eustachian tube with a patient suffering from a PET. m mastoid, ca carotid artery, et Eustachian tube, ms maxillary sinus, sn nasal septum (Dornhoffer et al., 2014).](image)

**Figure (17):** Axial CT of the Eustachian tube with a patient suffering from a PET. m mastoid, ca carotid artery, et Eustachian tube, ms maxillary sinus, sn nasal septum (Dornhoffer et al., 2014).

**Treatment:**

Fortunately, PETs are frequently transient, resolving when the condition causing them (e.g. weight loss or hormonal changes) resolves (Bluestone and Cantekin, 1981). In symptomatic pregnant cases, increased humidity, frequent Valsalva or Mueller maneuvers and injection of irritating substances into the peritubal areas relieve the symptom (Derkay, 1988).
E. Bell’s Palsy

The incidence of idiopathic facial palsy or Bell's palsy is higher in pregnancy. The pregnant women have a 3.3 times increased risk of developing this palsy which is more during the third trimester of pregnancy, 38–45 cases per 100,000 deliveries, compared with approximately 17 cases per 100,000 per year for non-pregnant women of childbearing age (Falco and Errikson, 1989).

This increased risk is due to edema of facial nerve and surrounding tissues due to increased interstitial fluid volume, which causes compression of the nerve (Deshpande, 1990). The third trimester is the time when the extracellular volume is at its maximum, supporting the theory that fluid retention leads to perineural edema followed by a facial palsy (Walling, 1993).

The other major hypothesis is viral, as the gestational immunosuppression induced by rise in cortisol levels, lead to reactivation of a latent herpes simplex virus (HSV) and herpes zoster virus (HZV) (This may be suppression of herpes simplex virus reactivation in early pregnancy and increase susceptibility to infection and reactivation in late pregnancy), inflammatory reactivation with subsequent demyelinization (Vrabec et al., 2007; Host and Mabry, 1983).

When a herpes eruption is present on the side of facial weakness, then Ramsay Hunt syndrome is diagnosed. In addition to the rash, hearing loss is much more common in Ramsay Hunt than most other causes of bell’s palsy (Pietersen, 2002). Ramsay Hunt syndrome type II is the reactivation syndrome of herpes zoster in the geniculate ganglion. It has variable presentation which may include a lower motor neuron lesion of
the facial nerve, hearing loss, vertigo, and pain. A triad of ipsilateral facial paralysis, ear pain, and vesicles in the auditory canal and auricle is typical for Ramsay Hunt Type II (Sweeney and Gilden, 2001).

ABRs are able to dependably detect early subclinical lesions of the auditory pathways. Therefore, one might expect that a pathological process in the internal acoustic canal or brain stem might also produce an abnormal ABR pattern. Due to clinical and histological observations that edema of the facial nerve in cases with idiopathic paralysis extends proximal to the geniculate ganglion (even into the internal auditory canal) can cause a compression of the eighth cranial nerve by this edema. ABR signs for a cochlear lesion are more commonly seen in such auditory disorders as sensorineural hearing loss. However, pathological ABRs with delayed brain-stem latencies either on the side of the paralysis or contralateral to it can still be caused by the same pathogenesis as the palsy. It is possible that these are signs for a common neuropathy in the central pathways of the auditory system and the facial nerve tracts (Welkoborsky et al., 1991).

Electroneurography (ENoG) refers to the electrical stimulation of the facial nerve. The purpose of ENoG testing is to quantify the percentage of facial nerve fibers that are electrically stimulable. The patient serves as the control because the weak side is compared to the normal side (Beck and Benecke, 1993).

Management of Bell’s palsy in pregnancy requires careful consideration of both the mother and fetus. It has long consisted of oral prednisolone, antiviral agents and supportive eye care (Kumar, 2011).
The usual course of steroids may be needed for treating this palsy. However, the steroid use early in pregnancy has an increased risk of cleft-palates, and infants born to mothers receiving exogenous steroids should be watched for adrenal hypofunction (Host and Mabry, 1983). So, corticosteroids can be used if it presents in third trimester, Prednisolone is given 1 mg/kg per day and tapered over 5 days. If HSV is the suspected cause then Acyclovir is used which is a category B drug (Gillman et al., 2002).

Prognosis for a full recovery in more severe palsies is poorer in pregnant women than in non-pregnant women (Peitersen, 2002), although the data supporting this theory may be biased as pregnant patients are less likely to receive treatment (Vrabec et al., 2007).

**Bell’s palsy and Tinnitus during Pregnancy: Predictors of Preeclampsia?!!**

Bell’s palsy and preeclampsia share the similar pathogenesis of extra-cellular edema. Hence, there is a strong association between them with 22% of women with Bell’s palsy developing preeclampsia in pregnancy (Katz et al., 2011; Shmorgun et al., 2002). Increased blood pressure may contribute to increased extracellular fluid volume, and therefore may lead to perineural edema (Deshpande, 1990; McGregor et al., 1987).

As a result, the facial nerve may become impinged within the bony canal. This theory of fluid retention has also been proposed as an explanation for the increased incidence of carpal tunnel syndrome during pregnancy (Graham, 1982).
Hypertension may also cause nerve compression through other mechanisms such as vasospasm, microemboli, or thrombosis of the vasa nervorum (Falco and Eriksson, 1989; Hansen et al., 1986).

Since the facial and acoustic cranial nerves as well as the inner ear are all tightly contained within a nerve sheath or labyrinth respectively, it is plausible that the edematous state often associated with preeclampsia may initially manifest with an isolated neurological deficit with affection on these parts. The explanation for an increased prevalence of tinnitus in pregnancy may be due to an increase in perilymphatic fluid pressure. It seems to follow, intuitively, that if the above explanations for tinnitus are plausible; it is expected to see an increase in tinnitus in those pregnant patients with preeclampsia (Shapiro et al., 1999).

Therefore, any woman presenting with Bell’s palsy should have screening for preeclampsia by performing urine dipstick for protein, measurement of blood pressure and carrying out renal and liver function tests. We must put in our mind that certain drugs like magnesium sulfate used in preeclampsia can actually worsen the recovery of Bell’s palsy (Lee et al., 1996).

Ragupathy and Emovon, (2013) reported two cases of Bell’s palsy in pregnancy in association with preeclampsia. First case, Caucasian primigravida presented at 39 weeks gestation with sudden onset of right sided facial weakness and numbness. They confirmed Bell’s palsy and gave steroids. Post admission, the blood pressure was labile with proteinuria and labetalol commenced. They induced labor 4 days later for preeclampsia. She delivered a 3.2 kg baby with good APGARS and cord gases. Second case, a 26-year-old Asian woman in her second pregnancy presented at 37 weeks of
gestation in early labor. She was already on steroids for Bell’s palsy (started by her general practitioner 8 days prior). She developed preeclampsia with elevated blood pressure, impaired liver and renal function tests. She had a normal vaginal delivery of a 3 kg baby. Her blood pressure stabilized on labetalol, 100 mg three times a day, and subsequently went home on third postnatal day.

Mylonas et al. (2005) reported a case of Bell’s palsy during early postpartum period in a patient with mild preeclampsia. Shapiro et al. (1999) have presented two cases of Bell’s palsy and one of tinnitus, all in third trimester, and all associated with preeclampsia.
**F. Brain tumors**

Brain tumors can become symptomatic during pregnancy, some appearing more frequently (meningiomas), and some only increasing in size and worsening symptomatology (meningiomas, hemangioblastomas, pituitary adenomas, and neurinomas) (Roelvink et al., 1987; Simon, 1988).

Tumors such as neurinomas and astrocytomas have been described to present for the first time during pregnancy (Klinken et al., 1990; Bardeguez et al., 1989). Since nausea, vomiting, headaches and vertigo are common complaints both during pregnancy and in the presence of brain tumors, the later ones may be under-diagnosed or even missed in the pregnant woman, until real neurological signs appear (Beni-Adani et al., 2001).

In the United States approximately 100 pregnant women per year would have a primary brain tumor, although not all such tumors would be diagnosed during pregnancy (Simon, 1988). Hormonal changes account for some of the dynamics in size of brain tumors and their clinical manifestations (Goldberg and Pappaport, 1987; Haas et al., 1986) especially in meningiomas (Schlehofer et al., 1992) and in vestibular schwannomas (Doyle and Luxford, 1994).

Vestibular Schwannoma is a benign tumor comprising 8% of all brain tumors and 78% of cerebellopontine-angle tumors (CPA), arising from the Schwann cells of the vestibular nerves, usually within the internal auditory canal, Diagnosis is based on clinical presentation, supporting evidence from various audiometric studies including brainstem evoked response potentials and neuroimaging (Awan et al., 2001).
Generally, acoustic neurinomas have been found to be large and more vascular during pregnancy. So, these tumors can present for the first time during pregnancy or symptoms may worsen during the last 3-4 months of pregnancy due to acute increase in size secondary to pregnancy changes (Gaughan and Harner, 1993).

There are two leading hypotheses regarding the mechanisms underlying increase in the size of these tumors during pregnancy. One focuses on the rapid expansion or engorgement of the vascular bed, which is presumably the result of the generalized increase in blood volume that occurs during pregnancy. The other hypotheses is that there is a direct hormonal effect on tumor growth rate mediated by the progesterone and estrogen receptors, also known to mediate growth of meningioma cells (Fugimoto et al., 1984; Glick et al., 1983).

Several metabolic changes associated with pregnancy may in fact, also be responsible for the observation that acoustic neurinomas are larger in pregnancy (Gaughan and Harner, 1993). Arterial hypertension or pre-eclampsia and the tendency to retain extracellular and intracellular fluid during pregnancy are considered to be additional predisposing factors for the development of increased intracranial pressure and cerebral edema (Olivi et al., 1992).

These changes may accentuate the symptoms associated with acoustic neurinoma or as sometimes reported, may result in their initiation. Spontaneous intratumoral hemorrhage has also been reported in acoustic neurinomas (Misra et al., 1995).

An explanation suggests that the brain tumor may undergo selective fluid accumulation and high blood pressure in the late stage of pregnancy contributing to increased intracranial pressure (ICP) and
symptomatology complicating the diagnosis is the clinical overlap between AN and preeclampsia (Bernard, 1998; Dutton et al., 1991).

Kasantikul et al. (1980) using immunohistochemical staining, found estrogen binding activity in eight acoustic neurinomas. They concluded that estrogen may promote the growth of acoustic neurinomas by inducing proliferation of vascular endothelium with a resultant increase in tumor vascularity.

Beatty et al. (1995) reported six cases of acoustic neurinomas during pregnancy where the tumor was removed in one patient while she was pregnant, in the other five subjects; the tumor was removed 2-10 months postpartum.

Hsiano et al. (1997) reported a case with twin pregnancy, presenting during the 30th week of pregnancy and whose symptoms exacerbated during the 36th week, when a caesarean section was performed and 2 weeks later craniotomy and tumor excision was done.

Thacker et al. (1995) reported a case of giant acoustic neurinoma presented with hyperemesis gravidarum during third trimester of pregnancy.

Small vestibular schwannomas usually present with only hearing abnormalities or tinnitus. However, larger tumors may present with headache and vomiting (due to obstructive or communicating hydrocephalus), ataxia (and other cerebellar signs), and cranial nerves involvement (including trigeminal, facial, glossopharyngeal and vagus nerves). Failure to diagnose a large lesion compressing the brain-stem may cause sudden deterioration and an attempted delivery in the presence
of untreated high ICP can be disastrous (Imrych and Imrychova, 1988; Deev and Gusev, 1990).

A strong clinical suspicion or advanced neurologic involvement may assist in diagnosis. If AN is highly suspected, the patient should be referred for magnetic resonance imaging (MRI) of the internal auditory canals with gadolinium contrast (figure 18). Diagnosed at an early stage, before ICP causes neurologic deterioration, proper management may allow for vaginal delivery with low maternal risk. Then, tumor resection can be performed postpartum (Gaughan and Harner, 1993). However, when diagnosis is made at a later stage with severe ICP, neurosurgical and obstetric cooperation is crucial for managing cesarean delivery and tumor resection. The diagnosis and treatment of vestibular schwannoma in the pregnant woman present a real challenge to the obstetrician, neurosurgeon and anesthesiologist regarding maternal and fetal well-being (Beni-Adani et al., 2001; Magliulo et al., 1995).

If diagnosis is made during the second half of gestation, fetal maturation can be accelerated by administration of corticosteroids. Lung maturity can be determined by lecithin-sphingomyelin index or by phosphatidylglycerol levels, (which is important for formation and stabilization of the surface-active layer, which prevents alveolar collapse and respiratory distress). Once lung maturity is established, early cesarean delivery can be performed to allow brain surgery (Isla et al., 1997).

In the past, prognosis in such cases was very poor. Allen et al. (1974) described seven women with acoustic tumors presenting during pregnancy with hearing abnormalities, mild cerebellar signs, but none with papilledema before delivery. The eighth case he described was a 36 year old woman with unsteady gait, slurred speech, facial weakness and
dysphagia. Six weeks before term, her clinical status deteriorated. Owing to threatened eclampsia, a cesarean section was performed with survival of a healthy child but deterioration of the mother who became blind and soon after became comatose and died. On autopsy, the diagnosis of acoustic neurinoma was confirmed.

![MRI Image](image_url)

**Figure (18):** MRI (T1-weighted image) demonstrating a huge right acoustic neuroma, strongly enhancing after gadolinium administration. The tumor is mostly solid (white round tissue), with some cystic components (dark areas surrounding parts of the tumor). It significantly compresses the brainstem, causing midline shift and obstructive hydrocephalus (Beni-Adani et al., 2001).
An audiogram that depicts the typical high-frequency sensorineural hearing loss, of female who was diagnosed with acoustic neuroma. She presented with a complaint of left-ear hearing loss and tinnitus (figure-19) (Battista and Messina, 2012).

![Audiogram of audiometer showing asymmetrical pattern of hearing loss with elevated thresholds at high frequencies, consistent with acoustic neuroma](image)

**Figure (19):** Female with asymmetrical audiogram and acoustic neuroma on the left side (Battista and Messina, 2012).

Another important aspect of the audiogram is acoustic reflexes. An audiologist may see absent or elevated responses with an acoustic neuroma but this is not the most sensitive measure as reflexes may still be within normal limits in patients with acoustic neuromas. Patients may also have absent reflexes for other reasons, so we cannot rely on this measure alone. Patient’s speech discrimination may often be worse than we expected based on the audiogram. The patient may present with mild or moderate hearing loss but have poor word understanding. This can be associated with an acoustic neuroma (Battista and Messina, 2012).
ABR testing can be important in the follow-up care of the patient. A characteristic finding on the ABR is a present wave I but absent waves III or V (figure 20); we may also see a delayed I-III absolute latency or a delayed wave V latency. It is also important to pay attention to the differences between ears and look for any significant interaural differences because that can indicate possible acoustic neuroma. There are high false-positive and false-negative rates associated with the ABR. Patients with small tumors can have normal ABRs. The ABR is more accurate the larger the tumor is, so it has 92 to 98% accuracy when the acoustic neuroma is greater than one-and-a-half centimeters but as it gets smaller than that, the accuracy drops to 60 to 70%. Even if a patient has a normal ABR, an acoustic neuroma cannot be ruled out (Schmidt et al., 2001).

![ABR tracings of patient with acoustic neuroma on the left side.](image)

_Figure (20): ABR tracings of patient with acoustic neuroma on the left side. Note left-ear diminished wave I and absent waves III and V (Battista and Messina, 2012)._  

Danger of obstructive hydrocephalus, with possible herniation can be predicted by the presence of papilledema. Papilledema, headache and vomiting are some of the alarming clinical presentations of high ICP, so ICP must be treated before delivery. The fastest and the safest operation
should be chosen. The options of CSF drainage include external ventriculostomy, endoscopic third ventriculostomy, or a shunt operation (usually ventriculoperitoneal). This is achieved best by a ventriculoperitoneal shunt procedure. Radical tumor surgery that might last long hours, should not be the means to decrease ICP if danger is real to mother and fetus. It should be delayed and performed in the best physiological conditions after delivery (Beni-Adani et al., 2001).

The traditional management for acoustic neuroma is surgical excision; however, gamma knife radio-surgery is becoming increasingly relevant as an alternative to microsurgical tumor resection as it combines an effective and safe minimal-invasive treatment (Muacevic et al., 2004). Gamma knife treatment is as efficient as microsurgery, but without risk of infection, bleeding or CSF leak. The patient can go back to work after a few days. By this, the radiation dose to all parts of the tumor and the surrounding structures was defined with a high degree of accuracy. It is offered to all patients with a tumor size of less than approximately 4 cm. Permanent arrest of growth, normal facial nerve activity, preservation of hearing and no complications are consequently fully sufficient goals of the gamma knife treatment (Noren et al., 1993).

In the gamma knife treatment, the converging gamma beams from 201 $^{60}$Co sources were collimated to a focus giving rise to a volume of sharply defined radiation. This volume was made to coincide exactly with the target by positioning the patient's head by means of the stereotactic frame in the gamma knife according to the precalculated co-ordinates. The whole radiation was delivered in one session usually taking 15 to 25 minutes. The dose delivered to the periphery of the tumor was previously usually 18 to 20 Gy but was reduced during the last three years to 10 to 15 Gy with a maximum of 15 to 25 Gy at the centre (Noren et al., 1993).
**G. Superior and posterior semicircular canals dehiscence presenting in pregnancy.**

Superior semicircular canal dehiscence (SSCD) is a rare condition in which the roof of the superior semicircular canal is absent or shows evidence of severe thinning. Superior semicircular canal dehiscence was first reported by Minor et al. (1998).

Posterior semicircular canal dehiscence is more infrequently reported compared to superior semicircular canal dehiscence. They observed that patients with posterior and superior semicircular canal dehiscence have similar auditory and vestibular features. However, they observed that the vertical component of the pressure and sound induced nystagmus in PCD beats in the opposite direction to that of superior canal dehiscence (Meehan et al., 2013). Ewald’s first law in humans where the axis of nystagmus should match the anatomical axis of the semicircular canal that created it (Cremer et al., 2000).

In semicircular canal dehiscence (SCD), an extra third mobile window is created in the bony labyrinth in addition to the oval and round windows. Under normal circumstances, sound pressure waves are transmitted through the oval window into the cochlea and activate the hair cells on the basilar membrane to create the sensation of sound. The sound pressure is then dissipated back into the middle ear via the round window. It has been hypothesized that if a third mobile window is present in the labyrinth, the sound waves will be transmitted into both the cochlea and the labyrinth with the subsequent stimulation of the vestibular hair cells and the production of vertigo (Meehan et al., 2013).
It is believed that a dehiscence may result from congenital underdevelopment of the temporal bone followed by an event later in life that breaks and opens the weak area. Precipitating events may involve high intracranial or middle ear pressures such as during weight lifting or pushing during labor, but most patients do not remember a specific time of onset of symptoms (Mikulec et al., 2004).

Symptoms of semicircular canal dehiscence syndrome include vertigo and oscillopsia, precipitated by loud sounds (Tullio’s phenomenon), aural fullness, tinnitus and hearing loss (HL) (Meehan et al., 2013). Symptoms can arise following sneezing, coughing, nose blowing, bending, exertion, applying pressure to the tragus and on carrying out a Valsalva maneuver (Cremer et al., 2000).

Some patients develop increased bone conduction sensitivity, as a consequence, they frequently report hearing their own heart beat (autophony), hearing their feet touch the ground when walking, hearing their joints or eye balls move and hearing a tuning fork placed on a distal extremity. Negative bone conduction thresholds are often found on pure tone audiometry, therefore an air-bone gap can exist (figure 21) and patients may be misdiagnosed with otosclerosis (Picavet et al., 2009).

However patients with SSC dehiscence may be distinguished from patients with otosclerosis by the presence of intact acoustic reflexes and increased vestibular evoked myogenic potentials (VEMPs) on the affected side (figure 22). It is therefore advised that when SSC dehiscence is suspected, the audiologist should check for negative bone conduction thresholds, acoustic reflexes and VEMPs (Merchant et al., 2007).
**Figure (21):** Conductive hyperacusis in patient with bilateral SCD (Yuen et al., 2009).

VEMPS (Vestibular evoked myogenic responses)

**Figure (22):** Left (figure B): VEMP obtained in an individual who has left sided superior canal dehiscence, using a Bio-Logic Navigator Pro. The left side is much larger than the right. Right (figure A): Threshold VEMP in same person, showing lower threshold on the left side (Watson et al., 2000; Brantberg et al., 1999).
The diagnosis of semicircular canal dehiscence is confirmed by high resolution (0.5 mm collimation or less) CT scan of the temporal bone. MRI scans with contrast are useful investigations in the diagnosis of vertigo and should be obtained to exclude a central pathology, but do not show semicircular canal dehiscence as clearly as high resolution CT scan (Meehan et al., 2013).

Smith and Phillips, (1998) reported patient with developed semicircular canal dehiscence symptoms at 22 weeks gestation, which may have resulted from the bone demineralization observed during pregnancy. The additional effects of sneezing during an upper respiratory tract infection may also have been a contributory factor to the dehiscence.

Meehan et al. (2013) presented the first case of a female pregnant patient with superior and posterior semicircular canal dehiscence. The diagnosis of semicircular canal dehiscence should be suspected in patients with sound induced vertigo. It should also be considered in patients with conductive hearing loss and intact acoustic reflexes. The diagnosis of semicircular canal dehiscence is based on clinical signs and symptoms and can be confirmed by high resolution CT scan (figure 23, 24).

The evidence to date from investigations supports the hypothesis that SSCD can cause a conductive hearing loss by acting as a ‘third’ window in the inner ear that results in elevation of thresholds for air-conducted sounds and reduction of thresholds for bone-conducted sounds (Rosowski et al., 2004).
**Figure (23):** Bone window CT of the petrous bone. The arrow demonstrates dehiscent left superior semicircular canal (Meehan et al., 2013).

**Figure (24):** The arrow demonstrates dehiscent left posterior semicircular canal (Meehan et al., 2013).
The main treatment for superior and posterior semicircular canal dehiscence is avoidance of the provoking factors. In the case of disabling disequilibrium, surgical intervention is recommended and usually involves resurfacing the area of dehiscence alone, or combined resurfacing and plugging, with occlusion of the canal lumen. The rate of recurrence of symptoms is reported to be significantly lower when occlusion of the lumen is performed (Crane et al., 2008).

Access to superior semicircular canal dehiscence can be made via a middle cranial fossa or via a less invasive transmastoid approach (Fiorino et al., 2010). In posterior canal dehiscence, a transmastoid approach with plugging of the posterior semicircular canal can be performed (Mikulec and Poe, 2006). Alternatively, a ventilation tube could be inserted for patients with symptoms induced by pressure in the external auditory canal. By reducing the movement of the tympanic membrane, ossicular chain and stapes footplate, this should lead to a reduced pressure in the inner ear fluids (Minor, 2005).

Disequilibrium in pregnant woman is notoriously difficult to diagnose and patients may go untreated for years. Obstetricians and gynecologists with patients complaining about new onset postpartum vertigo should inquire about symptom onset and focus their questions around events during the second stage of labor. Patients with symptoms of SSCD should be referred to an audiologist (Jacqueline et al., 2009).

The resulting symptoms of SCD and PLF can include vertigo, disequilibrium, aural fullness, autophony, tinnitus, and conductive hearing loss with enhanced bone conduction (Ogutha et al., 2009). Differentiating the two conditions (PLF & SSCD) from each other along
with Meniere's disease can pose a diagnostic challenge. Audiometric assessment must be undertaken at the time of presentation and serially afterward. Although only 50% of patients with PLF may experience hearing loss, a low-frequency SNHL may be suggestive of Meniere's disease as an alternative diagnosis (Friedland, 2009).

The fistula test (figure 25), observing for a positive Hennebert sign (pressure-induced nystagmus) may be demonstrable, but it is absent in a significant number of patients and can be present in up to 30% of patients with Meniere's disease, so it is therefore unreliable. A high-resolution computed tomography scan of the temporal bones may help diagnose SSCD; however, the sensitivity for the diagnosis of PLF is limited (Pullen, 1992).

Figure (25): A fistula test (Hosuk and Won, 2012).
Effect of Pregnancy on Auditory Evoked Potentials

During pregnancy levels of the steroid (sex) hormones increases which may interact with various neurotransmitters in the brain. This sex steroid neurotransmitter interaction is known to affect the morphology and latencies of various evoked potential responses (Yadav et al., 2003). These hormones (estrogen and progesterone) show significant increase specifically between weeks 12 to 24 of pregnancy compared to early pregnancy (Avizheh et al., 2015).

There are contradictory findings concerning the impact of the menstrual cycle on hearing levels. Hormonal influence on auditory brainstem responses (ABR) has been elaborated by different researchers differently during menstrual cycle. Resende et al. (2000); Howard et al. (1992) and Fagan and Church, (1986) reported no change in ABRs throughout the menstrual cycle while Tasman et al. (1999); Elkind-Hirsch et al. (1992) and Zani, (1989) reported increasing latencies of wave III and V in midcycle and decreasing latencies in mid luteal phase of menstrual cycle.

Elkind-Hirsch et al. (1994) postulated that central auditory pathways are modulated by the changing levels of hormones during menstrual cycle on the basis of the quantitative analysis of various hormones during different phases of menstrual cycle.

Bhatia et al. (1991) found increasing latencies in pre-menstrual phase. All of them reported shortest latencies during menstrual phase when hormonal levels are lowest in the body.
Earlier studies have revealed that latencies of visual evoked potentials (VEP) decreases \((Tandon \text{ and } Bhatia, 1991)\) while those of auditory brainstem responses (ABR) increases during pregnancy \((Tandon \text{ et al., 1990})\).

\textbf{Tandon et al. (1990)} were one of the first to study ABRs in pregnant women and they concluded that absolute peak latencies of waves I to V were similar between pregnant and non-pregnant control groups. However, interpeak latencies I-III, III-V and I-V were higher in pregnant women when compared with the control group.

\textbf{Tasman et al. (1999); Elkind-Hirsch et al. (1992) and Zani, (1989)} suggested that increased inter-peak latency I–V of ABRs during third trimester of pregnancy could be because of elevated levels of estrogen and progesterone or could be due to retention of water.

\textbf{Egeli and Gürel, (1997)} conducted a similar study and found that wave I was statistically different between groups, yet no other absolute nor interwave latencies reached significance.

Another study by \textbf{Sennaroglu and Belgin, (2001)} did not find any change in the waves of ABR in pregnancy. They suggested that hormonal changes are occurring over a long period of time which result in adaptation and sensitization of the brain. However they found that hearing level to low tone sounds decreases which could be because of excessive retention of sodium and water.

\textbf{Elkind-Hirsch et al. (1994)} stated that estrogen by increasing the synthesis of GABA in auditory pathways has an inhibitory role on auditory nerve synapses, leading to delayed synaptic conduction time.
**Stomati et al. (2002)** also demonstrated estrogen through increasing mediators levels like Allopregnanolone which leads to increase the inhibitory effect of GABA on the ABR latency.

It has also been hypothesized that estrogen is responsible for electrolyte imbalance and thereby disturbing the inner ear function (*David, 1996*) responsible for brainstem synaptic impairment, presumably by producing ischemic changes (*Ben David et al., 1995*) that may influence cochlear blood flow and potentially hearing functions (*Laugel et al., 1987*).

Mid-latency responses (MLRs) and slow vertex responses (SVRs) are better tool to represent the central auditory conduction electrophysiologically and hormonal levels changes markedly during third trimester of pregnancy (*Yadav et al., 2002*).

As pregnancy involves a number of neuroendocrine interactions, so *Tandon et al. (1996)* tried to see the effect of pregnancy on central level of auditory pathways by recording MLRs and SVRs. The increased levels of estrogen, progesterone and other placental hormones during pregnancy might play an important role in controlling the higher functions. So, pregnancy has been found to have an inhibitory influence on cognitive functions which could be due to increased levels of sex steroids and their interaction with the central nervous system.

The effect of pregnancy on mid-latency responses (MLRs) and slow vertex responses (SVRs) are the indicators of auditory information processing at the thalamo-cortical and cortical association areas respectively. *Yadav et al. (2003)* studied MLR waves in pregnant and
they are not showing any significant change in pregnant group as compared to the non-pregnant group (Figure 26). They can interpret that primary thalamo-cortical auditory pathway, supposed to be the generators for MLR might not get affected by the changing levels of estrogen and progesterone during pregnancy. All the waves of SVRs are getting significantly delayed during pregnancy in their study (Figure 27) which could be because of the interaction between estrogen and progesterone with the generators of SVRs.

Generators of P2 and N2 components of SVR are found to be located in various poly-sensory association areas- peri-cruciate gyrus, antero-lateral gyrus and medial supra-sylvian gyrus (Dickerson and Buchwald, 1992; Woods et al., 1987). N1 wave arises from sub-cortical sources receiving projections from inferior parietal lobule and P1 reflect an additional auditory processing system in parallel with the primary thalamo-cortical pathway. All these generators lie in various cortical areas, it can be said that auditory information at the central level is getting delayed. This could be because of the elevated levels of various hormones and their interaction with various neurotransmitters in the generator regions. P3 event related potential which is a late component of auditory evoked response has already been found to be prolonged in pregnancy whose generators lie in hippocampus (Tandon et al., 1996).

Retention of sodium and water during pregnancy is not likely to affect the SVRs because that is most likely to affect the peripheral auditory conduction rather than central, so Yadav et al. (2003) provide an electrophysiological evidence of slowing of information processing and perception at the cortical and association areas during third trimester of normal pregnancy.
Figure (26): Representative tracings of MLRs in non-pregnant (A) and pregnant (B) females (Yadav et al., 2003).

Figure (27): Representative tracings of SVRs in non-pregnant (A) and pregnant (B) females (Yadav et al., 2003).
Conclusions

Hormonal fluctuations in women during pregnancy can cause vestibular and/or cochlear disorders.

Tinnitus is a common audiological complaint during pregnancy and occurs in nearly 25% of pregnant women.

Red flags and indications for audiological evaluation include hearing loss, vertigo and facial weakness.

Vertigo and dizziness are frequently experienced during pregnancy and are among the most common complaint from pregnant women to primary care.

Low frequency SNHL may be beginning in the first trimester and increasing over second and third trimesters but it never reaches the pathologic levels and returns to normal in the postpartum period.

CHL in pregnant women may be due to otosclerosis, Eustachian tube disorders, middle-ear effusion & SSCD.

Exacerbation of otosclerosis in pregnancy due to the rise in circulating estrogen that favors osteosclerotic formation by stimulating of osteocytic activity and playing a dominant role during ossification of otospongeotic bone lesion.

Preeclampsia is a risk factor for cochlear damage and permanent hearing loss.

Exacerbation of Meniere disease during pregnancy is secondary to the physiologic decrease in serum osmolality that is associated with hormonal changes (which cause fluid retention).
Recommendations

- Always evaluate for signs/symptoms of preeclampsia in gravid women with tinnitus. Severe tinnitus has even led to an emergency cesarean delivery at 34 weeks, with total resolution postoperatively.

- Calcium supplementation appears to approximately halve the risk of pre-eclampsia, preterm birth and outcome 'death or serious morbidity'.

- Asymmetric hearing loss complaint requires careful audiological evaluation to rule out a retrocochlear lesion (ie, acoustic neuroma) in pregnant women.

- SSNHL in pregnant women can be treated by dextran 40 leading to significant hearing improvement.

- Conservative methods of management of Meniere's disease, with respect to dietary changes of salt and caffeine reduction could be used as first line of management. During an acute attack dimenhydrinate and meclizine (Antivert) can be safely given in pregnancy.
Summary

Pregnancy is a dynamic, anabolic condition. As within several weeks of it, a new endocrine organ, the placenta, is formed and becomes ready for secreting hormones that affect the metabolism of all nutrients of the body and also there is increased activity of maternal pituitary, thyroid and adrenal glands.

The hormonal alterations which happen during pregnancy can result in changes in the homeostasis of labyrinthine fluids, since they have a direct influence on the enzymatic process and the action of neurotransmitters. The compromise of labyrinth fluid characteristics, as well as the interference on the sensitivity of enzymatic receptors influences the basal metabolism of the inner ear. These alterations can be asymptomatic or associated with symptoms.

These hormonal changes in women during pregnancy can cause vestibular and/or cochlear disorders. They include: tinnitus, hearing loss, autophony, imbalance, nausea, vomiting, vertigo and dizziness. Vertigo and dizziness are frequently experienced during pregnancy and are among the most common complaint from pregnant women to primary care.

Tinnitus is the most common audiological complaint during pregnancy with proposed theories including hyperdynamic circulation, increase in perilymphatic fluid pressure and hormonal changes. It has been speculated as an early warning sign of gestational hypertension or preeclampsia.

Eustachian tube dysfunctions (ETDs) have been estimated to affect between 5 - 30% of pregnant women and can be variable in terms of its symptomatology and this can be either tubal obstruction or patulous ET.
Summary

The pregnancy is rarely associated with a low frequency SNHL beginning in the first trimester and increasing over second and third trimesters. Hearing loss never reaches the pathologic levels and returns to normal in the postpartum period. It may be due to sex hormones affect the sensorineural hearing system, changes in hearing levels with so much fluid retention during pregnancy, pregnancy related stress and immune-mediated disorders.

In preeclampsia, cochlear microcirculatory disorders associated with impaired local oxygenation have been considered to be a major pathogenic factor in sensorineural hearing impairment. Reduced cochlear blood flow is a main contributor to hearing loss.

It was reported a 10-fold increase in the number of Meniere-associated vertigo attacks during early pregnancy secondary to the physiologic decrease in serum osmolality of pregnancy. This drop in serum osmolality is hypothesized to cause an influx of free water into the endolymphatic space.

Otosclerosis has been known to worsen and/or present during pregnancy as estrogens are well-known stimulators of osteocytic activity and may play a dominant role during ossification of an otospongotic bone lesion, thus increasing the fixation of the stapes to the oval window. This may explain the onset of a CHL due to otosclerosis during pregnancy.

This increased risk of Bell's palsy is due to edema of facial nerve and surrounding tissues due to increased interstitial fluid volume, which causes compression of the nerve.
References


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الملخص العربي

الحمل هو حالة بنانية ديناميكية حيث أنه في خلال بضعة أسابيع يتكون عضو عديم جديد وهو المشيمة ويقوم بإفراز هرمونات تؤثر على عملية الأيض لجميع العناصر الغذائية للجسم بالإضافة إلى زيادة نشاط الغدة النخامية والدرقية والكظرية للمرأة الحامل.

التغييرات الهرمونية التي تحدث أثناء الحمل ينتج عنها تغييرات في توازن (إستتباب) السائل الليمي لتجويف الأذن حيث أن لها تأثير مباشر على العملية الإندويمية وعمل الناقلات العصبية. وهذا بدورة يؤثر على الأيض الأساسي للأذن الداخلية. وهذه التغييرات من الممكن أن تكون غير مصاحبة بأعراض أو تظهر لها أعراض.

هذه التغييرات الهرمونية في المرأة أثناء الحمل قد تسبب اضطرابات في السمع والإتزان. هذه الاضطرابات تشمل طنين الأذن، ضعف السمع، حس صوت الذات، اختلال التوازن، الدوار، الدوار، القلق. الدوار والدوام (الدوخة) يمكن ملاحظتهم كثيرا أثناء الحمل وتعد من أكثر شكاوى الحوامل من النساء إلى الرعاية الأولية.

من الشكاوى السمعية الأكثر شيوعًا أثناء فترة الحمل هو طنين الأذن، ومن النظريات المقترحة لحدوثه هي زيادة نشاط الدورة الدموية أو زيادة في ضغط السائل الليمفي المحيطي (المسال القوقعي) أو التغييرات الهرمونية. ومن الجدير بالذكر أن طنين الأذن يعد مؤشرا مبكرا لحدوث تسمم الحمل.

قد يحدث أيضا خلل وظيفي بقناة أوستاكيوس، يؤثر على 30% من النساء الحوامل. وقد ينقسم هذا الخلل الوظيفي تبعا للأعراض إلى انسداد الأذن الوسطى أو إرخاء قناة أوستاكيوس.
قد يحدث أيضاً ضعف سمعي عصبي حسي مع الحمل في الترددات المنخفضة ولكن نسبه حدوثه ضعيفة. بيدا هذا الضعف في الثلاثة أشهر الأولى ويزيد في الثلاثة أشهر الثانية والثالثة، ولكن هذا الضعف غالباً لا يستمر ويعود السمع للمنسوب الطبيعي بعد الولادة. وقد يحدث هذا الضعف بسبب تأثير الهرمونات، زيادة احتباس السوائل بالأذن الداخلية، التوتر والاهبال المصاحب للحمل أو اضطرابات جهاز المناعة.

يدخل اضطرابات (خلل) في الأوعية الدقيقة لفوقيحة الأذن أثناء تسمم الحمل وبالتالي يقل الدم المغذي للفوقيحة ويقل الأكسجين الذي يصلها وهذا يعد من الأسباب المؤدية لضعف السمع العصبي الحسي.

وجد أن نوبات الدوار المصاحبة لداء منيبر تزيد بمعدل عشر مرات في المراحل الأولى من الحمل ويرجع هذا للتغيرات الأسموزية في الحمل حيث أن نقص الأسماوزية للدم يؤدي إلى دخول الماء إلى النفايات الداخلية للأذن.

بعد الحمل من الأسباب المحتمل أن تزيد ضعف السمع الناتج عن تصلب عضيات الأذن (تكتل عظم الركاب) حيث أن الاستروجين هو محفز ويلعب دوراً في عملية التصليب. وقد يوضح هذا بداية الضعف السعبي التوسيع بسبب مرض تصلب الأذن بسبب الحمل.

تزيد أيضاً مع الحمل نسبة حدوث شلل العصب الوجهي (شلل بيل) ويحدث هذا بسبب احتباس السوائل بين الأنسجة فيحدث ورم في العصب الوجهي (العصب السابع) والأنسجة المحيطة فيضغط هذا الورم على العصب ويحدث شلل العصب الوجهي (العصب السابع).
Audio-vestibular findings in pregnancy

An essay

Submitted for the Partial Fulfillment
of Master Degree in
Audiology

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2016
السمع والإتزان في الحمل

بحث مرجعي توئم للفصول على درجة الماجستير

في السمعيات

مقدمة من الطبية/ آية صلاح عز الرجال

بكالوريوس الطب والجراحة

تحت إشراف

أ.د/ محمد عبد اللطيف الجوهرى

أستاذ السمع والإتزان
وحدة السمعيات
"قسم الألف والأذن والحنجرة"
"كلية الطب "بنات"
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د/ رضا محمد عبد الوهاب بحيري

مدير السمع والإتزان
وحدة السمعيات
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جامعة الأزهر

"كلية الطب بنات"
جامعة الأزهر

2016
Acknowledgement

First and foremost, thanks to ALLAH that this work came to its end.

Words cannot adequately express the feelings of gratitude that I have for those who helped me to complete this work.

It is a great honor to express my sincerest feelings and my cordial appreciation to PROFESSOR DR. MOHAMED ABDEL LATIF EL-GOHARY, Professor of Audiology, Faculty of Medicine for Girls, Al-Azhar University, for his generous help, valuable scientific assistance and kindly giving me much of his valuable time. That, I am honored to have his name on my research.

I am greatly indebted and grateful to DR. REDA MOHAMED ABDEL WAHAB BEHAIRY, Lecturer of Audiology, Faculty of Medicine, Al-Azhar University, for her great effort, precious advice and endless support to complete this work.

Last but not least, I would like to offer my sincere thanks to persons who give me all support, help and love to complete this work, who are my parents, my sister, my friends.

AYA SALAH EZZ EL-REGAL
2016
بسم الله الرحمن الرحيم
فالوا سبحانك لآ
علمنا إلا ما علمنا
إبن أنت العلماء
الكلهم
صر رحمته العظيم
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<tr>
<td>ABR</td>
<td>Auditory brainstem response</td>
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<td>ACTH</td>
<td>Adrenocorticotropic hormone</td>
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<td>ANP</td>
<td>Atrial natriuretic peptide</td>
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<td>ANs</td>
<td>Acoustic neuromas</td>
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<td>AP</td>
<td>Action potential</td>
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<td>BERA</td>
<td>Brain stem-evoked response audiometry</td>
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<td>BM</td>
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<td>BMR</td>
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<td>BNP</td>
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<td>CHL</td>
<td>Conductive hearing loss</td>
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<td>CPA</td>
<td>Cerebellopontine angle</td>
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<td>CSF</td>
<td>Cerebrospinal fluid</td>
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<td>CT</td>
<td>Computed tomography</td>
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<td>ECoG</td>
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<td>GABA</td>
<td>Gamma amino-butyric acid</td>
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<tr>
<td>GFR</td>
<td>Glomerular filtration rate</td>
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<td>ICP</td>
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<td>IHCs</td>
<td>Inner hair cells</td>
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<td>kPa</td>
<td>Kilopascal</td>
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<td>MLRs</td>
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<td>MRI</td>
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<td>NKc</td>
<td>Natural killer cells</td>
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<td>NO</td>
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<td>NVP</td>
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<td>OAEs</td>
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<td>OC</td>
<td>Organ of corti</td>
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<tr>
<td>OHCs</td>
<td>Outer hair cells</td>
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<td>PAI</td>
<td>Plasminogen activator inhibitors</td>
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<td>PCD</td>
<td>Posterior canal dehiscence</td>
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<td>PE</td>
<td>Preeclampsia</td>
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<td>PET</td>
<td>Patulous Eustachian tube</td>
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<td>PLF</td>
<td>Perilymph fistula</td>
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II
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<td>PRL</td>
<td>Prolactin</td>
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<td>PTH</td>
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<td>SCD</td>
<td>Semicircular canal dehiscence</td>
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<tr>
<td>SEng</td>
<td>Soluble endolgin</td>
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<tr>
<td>SFLT1</td>
<td>Soluble Fms like tyrosin kinase 1</td>
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<td>SM</td>
<td>Scala media</td>
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<td>SNHL</td>
<td>Sensorineural hearing loss</td>
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<td>SOM</td>
<td>Serous otitis media</td>
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<td>SP</td>
<td>Summation potential</td>
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<td>SSCD</td>
<td>Superior Semicircular canal dehiscence</td>
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<td>SSNHL</td>
<td>Sudden sensorineural hearing loss</td>
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<td>ST</td>
<td>Scala tympani</td>
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<td>SV</td>
<td>Scala vestibuli</td>
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<td>SVRs</td>
<td>Slow vertex responses</td>
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<td>T₃, T₄</td>
<td>Thyroid hormones</td>
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<td>TBG</td>
<td>Thyroxine-binding-globulin</td>
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<td>TM</td>
<td>Tympanic membrane</td>
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<td>TMJ</td>
<td>Tempromandibular joint</td>
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<td>TSH</td>
<td>Thyroid stimulating hormone</td>
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<td>TTAG</td>
<td>Tubo-tympano-aerodynamic-graphy</td>
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<td>VEMP</td>
<td>Vestibular myogenic evoked potential</td>
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<td>VEP</td>
<td>Visual evoked potential</td>
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<td>WHO</td>
<td>World health organization</td>
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