**(Slide 1) Lecture 13**

**Physiology of sensory systems. Part 2**

**(Slide 2)** Lecture plan:

1. Vestibular apparatus and equilibrium.

2. The ears and hearing.

3. Concept of pain. Classification of pain.

**(Slide 3)** The sense of equilibrium, which provides orientation with respect to gravity, is due to the function of an organ called the **vestibular apparatus**. The vestibular apparatus and a snail-shaped structure called the cochlea, which is involved in hearing, form the inner ear within the temporal bones of the skull. The vestibular apparatus consists of two parts: (1) the otolith organs, which include the utricle and saccule, and (2) the semicircular canals.

**(Slide 4)** The sensory structures of the vestibular apparatus and cochlea are located within the **membranous labyrinth**, a tubular structure that is filled with a fluid called **endolymph**. Endolymph is unlike any other extracellular fluid: it has a higher K+ concentration (higher even than in the intra-cellular compartment) and much lower concentrations of Na+ and Ca2+ than do other extracellular fluids. Partly because of this concentration gradient, depolarization of the mechanoreceptor hair cells is produced by the passive inflow of K+ , rather than of Na+ or Ca2+ as in other cells. This ion movement is also driven by the negative resting membrane potential of the hair cells, so that K+ moves down its electrochemical gradient into the hair cells when the K+ channels in the apical membrane of the cells are opened. The membranous labyrinth is located within a bony cavity in the skull, the bony labyrinth. Within this cavity, between the membranous labyrinth and the bone, is a fluid called perilymph. Unlike endolymph, perilymph is fairly typical of extracellular fluids such as cerebrospinal fluid.

**(Slide 5)** The utricle and saccule provide information about linear acceleration − changes in velocity when traveling horizontally or vertically. We therefore have a sense of acceleration and deceleration when riding in a car or when skipping rope. A sense of rotational, or angular, acceleration is provided by the semicircular canals, which are oriented in three planes like the faces of a cube. This helps us maintain balance when turning the head, spinning, or tumbling.

**(Slide 6)** The receptors for equilibrium are modified epithelial cells. They are known as hair cells because each cell contains 20 to 50 hairlike extensions. These are actually modified microvilli called stereocilia arranged in rows of increasing height. Touching the stereocilia of the tallest row is an even taller true cilium called a kinocilium. When the stereocilia are bent in the direction of the kinocilium, the plasma membrane is depressed and ion channels for K+ are opened, allowing K+ to passively enter and depolarize the hair cell. This causes the hair cell to release a synaptic transmitter that stimulates the dendrites of sensory neurons that are part of the vestibulocochlear nerve (VIII). When the stereocilia are bent in the opposite direction, the membrane of the hair cell becomes hyperpolarized and, as a result, releases less synaptic transmitter. In this way, the frequency of action potentials in the sensory neurons that innervate the hair cells carries information about the direction of movements that cause the hair cell processes to bend.

**(Slide 7)** The otolith organs, the **utricle** and **saccule**, each have a patch of specialized epithelium called a macula that consists of hair cells and supporting cells. The hair cells project into the endolymph-filled membranous labyrinth, with their hairs embedded in a gelatinous **otolithic membrane**. The otolithic membrane contains microscopic crystals of calcium carbonate (otoliths) from which it derives its name (oto = ear; lith = stone). These stones increase the mass of the membrane, which results in a higher inertia (resistance to change in movement). Because of the orientation of their hair cell processes into the otolithic membrane, the utricle is more sensitive to horizontal acceleration and the saccule is more sensitive to vertical acceleration. During forward acceleration, the otolithic membrane lags behind the hair cells, so the hairs of the utricle are pushed backward. This is similar to the backward thrust of the body when a car quickly accelerates forward. The inertia of the otolithic membrane similarly causes the hairs of the saccule to be pushed upward when a person accelerates downward in an elevator. These effects, and the opposite ones that occur when a person accelerates backward or upward, produce a changed pattern of action potentials in sensory nerve fibers that allows us to maintain our equilibrium with respect to gravity during linear acceleration.

**(Slide 8)** The three semicircular canals project in three different planes at nearly right angles to each other. Each canal contains an inner extension of the membranous labyrinth called a semicircular duct, and at the base of each duct is an enlarged swelling called the ampulla. The crista ampullaris, an elevated area of the ampulla, is where the sensory hair cells are located. The processes of these cells are embedded in a gelatinous membrane, the cupula, which has a higher density than that of the surrounding endolymph. Like a sail in the wind, the cupula can be pushed in one direction or the other by movements of the endolymph.

**(Slide 9)** The endolymph of the semicircular canals serves a function analogous to that of the otolithic membrane—it provides inertia so that the sensory processes will be bent in a direction opposite to that of the angular acceleration. As the head rotates to the right, for example, the endolymph causes the cupula to be bent toward the left, thereby stimulating the hair cells. Hair cells in the anterior semicircular canal are stimulated when doing a somersault, those in the posterior semicircular canal are stimulated when performing a cartwheel, and those in the lateral semicircular canal are stimulated when spinning around the long axis of the body.

**(Slide 10)** **Video. 2-Minute Neuroscience\_ Vestibular System**

**(Slide 11)** Stimulation of hair cells in the vestibular apparatus activates sensory neurons of the vestibulocochlear nerve (VIII). These fibers transmit impulses to the cerebellum and to the vestibular nuclei of the medulla oblongata. The vestibular nuclei, in turn, send fibers to the oculomotor center of the brain stem and to the spinal cord. Neurons in the oculomotor center control eye movements, and neurons in the spinal cord stimulate movements of the head, neck, and limbs. Movements of the eyes and body produced by these pathways serve to maintain balance and “track” the visual field during rotation.

**(Slide 12) Video. INSTANT NEURO - Vestibular Pathways.**

**(Slide 13)** When a person first begins to spin, the inertia of endolymph within the semicircular ducts causes the cupula to bend in the opposite direction. As the spin continues, however, the inertia of the endolymph is overcome and the cupula straightens. At this time, the endolymph and the cupula are moving in the same direction and at the same speed. If movement is suddenly stopped, the greater inertia of the endolymph causes it to continue moving in the previous direction of spin and to bend the cupula in that direction. Bending of the cupula affects muscular control of the eyes and body through the neural pathways previously discussed. During a spin, this produces smooth movements of the eyes in a direction opposite to that of the head movement so that a stable visual fixation point can be maintained. When the spin is abruptly stopped, the eyes continue to move smoothly in the former direction of the spin (because of the continued bending of the cupula) and then are jerked rapidly back to the midline position. This produces involuntary oscillations of the eyes called vestibular nystagmus. People experiencing this effect may feel that they, or the room, are spinning. The loss of equilibrium that results is called vertigo.

**(Slide 14)** Sound waves are alternating zones of high and low pressure traveling in a medium, usually air or water. Thus, sound waves cannot travel in space. Sound waves travel in all directions from their source, like ripples in a pond where a stone has been dropped. These waves are characterized by their frequency and intensity. The frequency is measured in hertz (Hz), which is the modern designation for cycles per second (cps). The pitch of a sound is directly related to its frequency − the greater the frequency of a sound, the higher its pitch.

The intensity, or loudness, of a sound is directly related to the amplitude of the sound waves and is measured in units called decibels (dB). A sound that is barely audible − at the threshold of hearing − has an intensity of zero decibels. Every 10 decibels indicates a tenfold increase in sound intensity; a sound is 10 times louder than threshold at 10 dB, 100 times louder at 20 dB, a million times louder at 60 dB, and 10 billion times louder at 100 dB. The ear of a trained, young individual can hear sound over a frequency range of 20 to 20,000 Hz, yet still can distinguish between two pitches that have only a 0.3% difference in frequency. The human ear can detect differences in sound intensities of only 0.1 to 0.5 dB, while the range of audible intensities covers 12 orders of magnitude, from the barely audible to the limits of painful loudness. Human hearing is optimal at sound intensities of 0 to 80 dB.

**(Slide 15)** Sound waves are funneled by the pinna, or auricle, into the external auditory meatus. These two structures form the outer ear. The external auditory meatus channels the sound waves to the eardrum, or tympanic membrane. Sound waves in the external auditory meatus produce extremely small vibrations of the tympanic membrane; movements of the eardrum during speech (with an average sound intensity of 60 dB) are estimated to be about the diameter of a molecule of hydrogen!

**(Slide 16)** The middle ear is the cavity between the tympanic membrane on the outer side and the cochlea on the inner side. Within this cavity are three middle-ear ossicles − the malleus (hammer), incus (anvil), and stapes (stirrup). The malleus is attached to the tympanic membrane, so that vibrations of this membrane are transmitted via the malleus and incus to the stapes. The stapes, in turn, is attached to a membrane in the cochlea called the oval window, which thus vibrates in response to vibrations of the tympanic membrane. The fact that vibrations of the tympanic membrane are transferred through three bones instead of just one affords protection. If the sound is too intense, the ossicles may buckle. This protection is increased by the action of the stapedius muscle, which attaches to the neck of the stapes. When sound becomes too loud, the stapedius muscle contracts and dampens the movements of the stapes against the oval window. This action helps to prevent nerve damage within the cochlea. If sounds reach high amplitudes very quickly, however − as in gunshots − the stapedius muscle may not respond soon enough to prevent nerve damage.

**(Slide 17)** Encased within the dense temporal bone of the skull is an organ called the cochlea, about 34 mm long (the size of a pea) and shaped like the shell of a snail. Together with the vestibular apparatus (previously described), it composes the inner ear. Vibrations of the stapes and oval window displace perilymph fluid within a part of the bony labyrinth known as the scala vestibuli, which is the upper of three chambers within the cochlea. The lower of the three chambers is also a part of the bony labyrinth and is known as the scala tympani. The middle chamber of the cochlea is a part of the membranous labyrinth called the cochlear duct, or scala media. Like the cochlea as a whole, the cochlear duct coils to form three turns, similar to the basal, middle, and apical portions of a snail shell. Because the cochlear duct is a part of the membranous labyrinth, it contains endolymph rather than perilymph.

**(Slide 18)** The perilymph of the scala vestibuli and scala tympani is continuous at the apex of the cochlea because the cochlear duct ends blindly, leaving a small space called the helicotrema between the end of the cochlear duct and the wall of the cochlea. Vibrations of the oval window produced by movements of the stapes cause pressure waves within the scala vestibuli, which pass to the scala tympani. Movements of perilymph within the scala tympani, in turn, travel to the base of the cochlea where they cause displacement of a membrane called the round window into the middle-ear cavity. This occurs because fluid, such as perilymph, cannot be compressed; an inward movement of the oval window is thus compensated for by an outward movement of the round window.

**(Slide 19)** When the sound frequency (pitch) is sufficiently low, there is adequate time for the pressure waves of perilymph within the upper scala vestibuli to travel through the helicotrema to the scala tympani. As the sound frequency increases, however, pressure waves of perilymph within the scala vestibuli do not have time to travel all the way to the apex of the cochlea. Instead, they are transmitted through the vestibular membrane, which separates the scala vestibuli from the cochlear duct, and through the basilar membrane, which separates the cochlear duct from the scala tympani, to the perilymph of the scala tympani. The distance that these pressure waves travel, therefore, decreases as the sound frequency increases. Sound waves transmitted through perilymph from the scala vestibuli to the scala tympani thus produce displacement of the vestibular membrane and the basilar membrane. Although the movement of the vestibular membrane does not directly contribute to hearing, displacement of the basilar membrane is central to pitch discrimination. Each sound frequency produces maximum vibrations at a different region of the basilar membrane. Sounds of higher frequency (pitch) cause maximum vibrations of the basilar membrane closer to the stapes.

**(Slide 20) Video. 2-Minute Neuroscience: The Cochlea**

**(Slide 21)** The sensory hair cells are located on the basilar membrane with their “hairs” projecting into the endolymph of the cochlear duct. The hairs are actually stereocilia, which are large, specialized microvilli arranged in bundles. The stereocilia increase in size stepwise within each bundle, as they do in the vestibular apparatus; however, unlike the case in the vestibular apparatus, the cochlear hair cells lack kinocilia.

**(Slide 22)** There are two categories of hair cells, inner and outer. Inner hair cells, about 3,500 per cochlea, form one row that extends the length of the basilar membrane. The hair bundles on the inner hair cells are mechanosensory: they transform sound waves in cochlear fluid into nerve impulses. Their stereocilia are interconnected near their tips with filaments that are coupled to mechanotransduction channels in the plasma membrane. These channels open when the stereocilia within each bundle are bent in the direction of their tallest members, allowing the movement of K+ across the plasma membrane as will be described shortly. Each of the inner hair cells is innervated by 6–20 sensory neurons of cranial nerve VIII from the spiral ganglion, which transmit sound information to the brain. The number of synapses with afferent neurons depends on the location of the inner hair cells along the basilar membrane, with those in the middle having the greatest number of synapses and the highest sensitivity to sound. There are also about 11,000 outer hair cells arranged in multiple rows: three rows in the basilar turn, four in the middle turn, and five in the apical turn of the cochlea. The outer hair cells are innervated primarily by motor neurons that originate in the olivary nuclei of the medulla oblongata. These depolarize or hyperpolarize the outer hair cells, causing them to shorten when they are depolarized or lengthen when they are hyperpolarized. Such movements are believed to aid the sensory function of the inner hair cells.

**(Slide 23)** The stereocilia of the hair cells are embedded in a gelatinous tectorial membrane ( tectum = roof, covering), which overhangs the hair cells within the cochlear duct. The association of the basilar membrane, inner hair cells with sensory fibers, and tectorial membrane forms a functional unit called the spiral organ, or organ of Corti. When the cochlear duct is displaced by pressure waves of perilymph, a shearing force is created between the basilar membrane and the tectorial membrane. This causes the stereocilia to bend, and this mechanical process opens K 1 channels in the plasma membrane covering the tops of the stereocilia.

**(Slide 24)** These K+ channels face endolymph, which uniquely has a high concentration of K 1 similar to that of the intracellular compartment. Also, the endolymph of the cochlea (but not the vestibular apparatus) has an amazingly high positive potential: + 100 mV. Combined with the negative resting membrane potential of the hair cells, this produces an extremely steep electrochemical gradient favoring the entry of K+. So, when the K+ channels in the bent stereocilia open, K+ moves passively down its electrochemical gradient into the hair cells. This depolarizes the hair cells and stimulates them to release glutamate, which stimulates the associated sensory neurons. The K+ that entered the hair cells at their apical surface can then move passively out through channels in their basal surface, which face perilymph in the scala tympani. Perilymph, as previously mentioned, has a low K+ concentration typical of extracellular fluids.

**(Slide 25)** The greater the displacement of the basilar membrane and the bending of the stereocilia, the greater the amount of transmitter released by the inner hair cell, and therefore the greater the generator potential produced in the sensory neuron. By this means, a greater bending of the stereocilia will increase the frequency of action potentials produced by the fibers of the cochlear nerve that are stimulated by the hair cells. Experiments suggest that the stereocilia need bend only 0.3 nanometers to be detected at the threshold of hearing! A greater bending will result in a higher frequency of action potentials, which will be perceived as a louder sound. As mentioned earlier, traveling waves in the basilar membrane reach a peak in different regions, depending on the pitch (frequency) of the sound. High-pitched sounds produce a peak displacement closer to the base, while sounds of lower pitch cause peak displacement further toward the apex. Those neurons that originate in hair cells located where the displacement is greatest will be stimulated more than neurons that originate in other regions. This mechanism provides a neural code for pitch discrimination.

**(Slide 26)** Pain is of a great significance in physiology, pathophysiology and in all fields of medicine. Understanding of physiological bases of pain permits to gain a deeper insight into the interactions of an organism with different internal and external stimuli including those leading to damage of tissues. Besides, knowing mechanisms of pain, a physician can timely and adequately use various medical means to improve the condition of a patient.

**(Slide 27)** Pain is a physiological phenomenon that informs us of hazardous influences which damage or potentially threaten an organism. Thus, pain is both a warning and protecting event. Currently the most popular is definition of pain given by the International Association for Study of Pain: “Pain is an unpleasant sensory and emotional experience associated with actual or potential damage to tissue or described in terms of such damage.”

**(Slide 28)** There are two main kinds of pain: physical pain and psychogenic pain. By cause, physical pain is classified into three categories:

1. Pain caused by external factors (except for pain caused by excessive adequate stimulations of sensory organs, for example, of vision, of audition). Such pain is characterized by the following features: it is superficial, brief except for the cases associated with breakage of the integrity of skin. Such pain can be easily localized and its cause identified. The external factor can be eliminated. The nervous system remains undamaged, its peripheral apparatus and central mechanisms remain intact.

2. Pain caused by internal processes. This kind of pain is associated with stimulation of any kind of receptors by different mechanisms. The resultant train of afferent impulses is perceived as pain. Skin usually does not participate in initiation of this pain, unless it is damaged or reflects pain. It is often impossible to localize this pain, identify its cause, and eliminate it partly or fully. It is of much longer duration. Here, the nervous system remains undamaged, because the focus of the pathological process is distal from receptors. Besides, afferent fibers retain the normal conduction, and pain modulating mechanisms function normally. By the type of the involved tissue, this kind of pain is categorized into ectodermal,

mesodermal, endodermal pain, and also pain caused by excessive loads on muscles.

3. Pain associated with damage to the nervous system, in particular to its afferent part. Although such pain is often accompanied by sensations on skin, it is nevertheless difficult or impossible to correctly identify the external causes and to localize the source of pain. This pain is long-term, may persist for years, and elimination of its source is impossible. The nervous system is damaged: defects in the conducting peripheral or central pathways are noted, as well as disturbances in pain modulating mechanisms. Damages causing this pain are proximal to receptors of peripheral nerves, spinal cord or higher nervous centers. This kind of pain may be local or systemic (neuralgia, causalgia, phantom pain, thalamic syndrome).

**(Slide 29)** Besides, physical pain may be:

- primary, or epicritic (rapid, acute, piercing) for example, pain caused by pricking the skin with a needle; it has a precise localization, persists for some time after elimination of the stimulus, does not elicit emotional response;

- **secondary**, or protopathic, pain (slow, intolerable, burning), which appears in 0.5-1 s after the primary pain, has no distinct localization, persists for some time after elimination of the stimulus, is accompanied by alteration in the functions of the cardiovascular and respiratory systems, may affect the personality, way of thinking (secondary pain also includes dull pain in the visceral organs and in deep somatic structures);

- **chronic pain** (physical pain, persisting for a long time in some patients with chronic diseases) is characterized by complex nervous mechanisms at the emotional, affective and behavioral levels manifested by reactive depression, which makes an individual disabled and radically changes his/her life.

**(Slide 30)** Psychogenic pain is associated with psychological and social factors, such as emotional condition of an individual, a specific situation, cultural traditions. It has no clearly defined onset and no evident reason. The nature of psychogenic pain remains unclear in many aspects. Often the acuity of pain as described by the patient does not correspond to his/her behavior. Pain may disappear at night. Pain may have poorly defined localization or may change localization, which sometimes does not correspond to the dermatome or the site of referred pain. Besides, psychogenic pain is not associated with external stimuli and can vary with the mood. It can be eliminated by intake of antidepressants and use of other means that relieve emotional stress.

**(Slide 31)** It follows from the above that the concept of “pain” includes:

- painful stimulus informing an individual about a past or a forthcoming damage to tissues;

- personal, or individual perception of a harmful factor;

- complex of responses aimed at protection of an organism against a noxious factor;

- category of experience based on numerous events accompanied by sensory and emotional conditions.

**(Slide 32)** Painful stimulations may arise from skin, deep tissues and internal organs. These stimulations are perceived by receptors (nociceptors) located throughout the body excluding the brain. Methods of microneurography confirmed the existence in humans and other mammalians of two types of pain receptors. The first type of these receptors is anatomically represented by free nerve endings that branch out in a tree-like manner (myelinated fibers). These are fast A-delta fibers conducting excitation at 6-30 m/s. These fibers are excited by high-intensity mechanical (a prick of a needle) and sometimes by thermal stimulations of skin. A-delta nociceptors are primarily located in skin. Besides, they are also found in joints.

**(Slide 33)** The other type of nociceptors is represented by dense non-capsulated glomerular bodies (unmyelinated C-fibers conducting excitation at 0.5-2 m/s). In humans and other primates these afferent fibers are represented by polymodal nociceptors that respond to mechanical, as well as thermal and chemical stimuli. They are activated by chemical substances produced in tissue damage, and at the same time act as chemoreceptors. C-Fibers are distributed throughout all tissues excluding the CNS. However, they are present in peripheral nerves as nervi nervorum. Fibers having receptors perceiving tissue damages, contain substance P which acts as a neurotransmitter. All types of nociceptors are characterized by low excitability. Bioactive substances that stimulate chemonociceptors, are called algogens. There are three groups of algogens: tissue algogens, plasma algogens and algogens released from the nerve endings.

**(Slide 34)** Tissue algogens are serotonin, histamine, some prostaglandins and high concentrations of K+ and H+ ions. Plasma algogens include bradykinin, kallidin and Hageman’s contact factor. The third group includes an oligopeptide – substance P.

**(Slide 35)** Opioid peptides and opioid receptors were discovered at the beginning of 70s of the 20th century. Of clinical importance are three classes of opioid receptors: μ-, kappa, and delta-receptors. Their distribution in the CNS is highly non-uniform. The highest density of receptors is found in the posterior horns of the spinal cord, in the mesencephalon and thalamus. As shown by immunocytochemical research, spinal opioid receptors have the highest concentration in the superficial layers of posterior horns of the spinal cord. Endogenous opioid peptides (enkephalin, endorphin, dynorphin) come into interaction with the opioid receptors each time when a painful sensation arises in result of overrunning the pain threshold. The fact that a great number of opioid receptors are localized in the superficial layers of the spinal cord means that opiates may easily penetrate into the spinal cord from the surrounding cerebrospinal fluid.

**(Slide 36)** In 1965 R.Melzack and P.Wall put forward the gate control theory of pain which is currently adopted by most researchers. The first statement of the gate theory is that transmission of nerve impulses from the afferent fibers to the neurons of the spinal cord that further transmit impulses to the brain, is modulated by the spinal gate mechanism which is a system of interneurons in the gelatinose substance of the spinal cord (the 2nd and the 3d laminae of the dorsal horn). It was found that neurons of these laminae receive axon terminals from numerous thin and thick afferent fibers, and also dendrites from the deeper laminae of dorsal horns of the spinal cord. This permits to suggest that gelatinose substance acts as the spinal gate modulating conduction of nerve impulses from peripheral receptors to the neurons of the spinal cord that project to the brain (relay neurons).

**(Slide 37)** According to the second statement of the gate theory, the spinal gate mechanism is controlled by a relatively large number of impulses arriving through thick and thin afferent fibers. The gate mechanism limits transmission of nerve impulses arriving at the relay neuron at high frequency through thick fibers (the gate afferent closes), and, conversely, facilitates passage of nerve impulses arriving at high frequency through the thin afferent fibers (the gate opens).

**(Slide 38)** The third statement of Melzack-Wall theory says that the spinal gate mechanism is influenced by nerve impulses coming from the cerebral cortex and the brainstem through the descending fibers. Such cognitive (evaluating) factors as attention, alertness, produce a powerful effect on the process of pain perception. The forth statement says that the selective cognitive processes modulating the properties of the spinal gate mechanism, are centrally controlled by a specialized system of large-diameter fibers with high conduction velocity. According to R. Melzack and P. Wall, this central triggering function is performed by posterior columns – the medial lemniscus and dorsolateral system.

**(Slide 39)** The fifth position states that if excitation of relay neurons of the spinal cord exceeds the critical level, they start to generate impulses stimulating nociceptive system. When excitation in relay neurons reaches the critical level, a train of impulses is transmitted to the brain, primarily through the anterolateral ascending system: through fibers of neospinothalamic pathway to the ventrobasal thalamus and further to the somatosensory cortex, and through fibers of the paleospinothalamic pathway to the reticular formation, medial intralaminar thalamus and structures of the limbic system. Activation of these brain structures determines the sensory, motivational and cognitive components of the general response to pain.

Thus, the basic principles of the gate theory take into account specificity of receptors, physiological mechanisms of convergence, summation, facilitation and inhibition, the role of ascending and descending systems of the brain and the spinal cord.

**(Slide 40)** Lesson assignment:

Lauralee Sherwood. Fundamentals of Human Physiology.

Pages: 145 – 146.

Pages: 161 – 176.

Questions that we will analyze for a lesson on this topic:

1. The structure of the vestibular apparatus.

2. Functioning mechanism of the Sensory hair cells of the vestibular apparatus.

3. Utricle and Saccule. The mechanism of their functioning.

4. Semicircular Canals. Its implications for vestibulation.

5. Neural pathways of the vestibular system.

6. Functional structure of the outer ear. Significance for the perception of sounds.

7. Functional structure of the middle ear. Significance for the perception of sounds.

8. Functional structure of the cochlea. Significance for the perception of sounds.

9. Structure and function spiral organ (organ of Corti).

10. The role of potassium in the mechanisms of the functioning of hair cells.

11. Concept of pain. Classification of pain

12. Modern concepts of pain sensory (nociceptive) system nociceptors.

13. Opioid Receptors and Mechanisms.

14 Modern Theories of Pain.

Finish for today

The full lecture is at the indicated website.

**Thank you for attention**