International Journal of Pharma and Bio Sciences
ISSN 0975 - 6299

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Proceedings of Third National B.D.S. Students seminar on Basic Medical Sciences
13th October 2007

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Abstract - Anat - 01

STEM CELLS

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Research on stem cells from humans and other animals is crucial to advancing our understanding of basic processes in developmental biology and also provides the potential to identify new drugs. Stem cell research may offer hope to those suffering from incurable degenerative diseases such as Alzheimer’s, Parkinson’s and Motor Neurone Disease. Stem cells can also be taken from umbilical cord blood just after birth. Of all stem cell types, autologous harvesting involves the least risk. By definition, autologous cells are obtained from one's own body, just as one may bank his or her own blood for elective surgical procedures. Adult stem cells are frequently used in medical therapies, for example in bone marrow transplantation. Stem cells can now be artificially grown and transformed (differentiated) into specialized cell types with characteristics consistent with cells of various tissues such as muscles or nerves. Embryonic cell lines and autologous embryonic stem cells generated through therapeutic cloning have also been proposed as promising candidates for future therapies the same time it also raises several scientific, ethical and social issues in the development of such applications. Apart from challenges of using the right kind of stem cells in the most appropriate way for a particular disease, there are also issues related to the use of human embryos to create human embryonic stem (hES) cell lines, potential for commoditization of human tissues and cells with inherent danger of exploitation of underprivileged people , and challenges related to prevention of human germ-line engineering and reproductive cloning.

Stem cells are most fascinating areas of biology today. It’s a remarkable potential. It develops into many different cell types in body. It serves as a sort of repair system for the body. They will divide without limit to replenish other cells. When a stemcell divides, each new cell has the potential to either remain as a stem cell or become another type of cell with more specialized function. Its ability to generate multiple organ – specific cell types and so they are described as multipotent. In stem cell healthy cell replaces the damaged cells. Stemcell that is most able to differentiate is the fertilized ovum. It’s a origin of all tissue types. the results of animal experiments are not directly applicable to humans and therefore research on human stem cells is necessary .A huge amount of research is needed however to understand exactly how stem cells work and how their potential can be harnessed for treatments.
Abstract- Anat - 02

DEVELOPMENT OF FACE

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Although a few developmental fMRI studies have shed some light on the neurological development of either object or spatial processing we still know very little about the development of the ‘what’ and ‘where’ processing systems. The external human face develops between the 4th and 6th weeks of embryonic development. Facial swellings arise on the frontonasal process (2 medial nasal and 2 lateral nasal processes) and the first pharyngeal arch (2 mandibular and 2 maxillary processes). By a process of merging and some localized fusion these processes come together to form the continuous surfaces of the external face. The primary palate is formed in this period by fusion/merging of the medial nasal and maxillary processes. Subsequently, between 6th and 12th embryonic/fetal weeks, the secondary palate is formed as the result of fusion between palatal processes growing from the oral surfaces of the maxillary processes. Each merging and fusion site is also the site of a potential facial or palatal cleft. The present findings have important implications for theories of visuospatial development. They suggest that the neural systems involved in face and location processing may undergo development and fine-tuning well into late childhood.

Face and neck development of the embryo refers to the development of the structures from the third to eighth week that give rise to the future head and neck. They consist of three layers, the ectoderm, mesoderm and endoderm, which form the mesenchyme (derived form the lateral plate and paraxial mesoderm), neural crest and neural placodes (from the ectoderm). The paraxial mesoderm forms structures named somites and somitomeres that contribute to the development of the floor of the brain and voluntary muscles of the craniofacial region. The lateral plate mesoderm consists of the laryngeal cartilages (arytenoid and cricoid). The three tissue layers give rise to the pharyngeal apparatus, formed by 6 pairs of branchia/pharyngeal arches, a set of pharyngeal pouches and pharyngeal grooves, which are the most typical feature in development of the head and neck. The formation of each region of the face and neck is due to the migration of the neural crest cells who come form the ectoderm. This cells determine the future structure to develop in each pharyngeal arch. Eventually, they also form the neurectoderm, which forms the forebrain, midbrain and hindbrain, cartilage, bone, dentin, tendon, dermis, pia mater and arachnoid mater, sensory neurons and glandular stroma.
Abstract - Anat - 03

CONJOINED TWINS

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Conjoined twins are two babies that are born physically connected to each other. Conjoined twins develop when an early embryo partially separates to form two individuals. Conjoined twins are fused twins resulting from incomplete division of a single blastocyst, 13 to 16 days post fertilization. Complete division of a human zygote within seven days of fertilization yields identical monozygotic twins. The embryological theory of the author is that all conjoined twins result from fusion of separate germ discs. The birth of conjoined twins has always fascinated mankind, with the public’s view of malformed children greatly influenced by the prevailing culture and religious beliefs. In prehistoric times, conjoined twins were depicted in cave drawings, on pottery, or as figurines. A rare phenomenon, the occurrence is estimated to range from 1 in 50,000 births to 1 in 200,000 births, with a somewhat higher incidence in Southwest Asia and Africa. Approximately half are stillborn, and a smaller fraction of pairs born alive have abnormalities incompatible with life. The overall survival rate for conjoined twins is approximately 25%. The condition is more frequently found among females, with a ratio of 3:1.

Two contradicting theories exist to explain the origins of conjoined twins. The older theory is fission, in which the fertilized egg splits partially. The second and more generally accepted theory is fusion, in which a fertilized egg completely separates, but stem cells (which search for similar cells) find like-stem cells on the other twin and fuse the twins together. Conjoined twins share a single common chorion, placenta, and amniotic sac, although these characteristics are not exclusive to conjoined twins as there are some monozygotic but non-conjoined twins that also share these structures in utero. This happens where the zygote of identical twins fails to completely separate. Conjoined twins occur in an estimated one in 200,000 births, with approximately half being stillborn. The overall survival rate for conjoined twins is between 5% and 25%. Conjoined twins are more likely to be female (70-75%). The mechanism of conjoined twins is still poorly understood. Identical twins are formed when the developing embryo splits in two during development. It is believed that conjoined twins form when the two separate eggs join back together somewhat later in development when cells begin to differentiate from stem cells. In many cases, depending on how closely the two twins are tied together and what tissue they share, they can be separated by surgery. However, such surgery is dangerous if the twins share common organs, particularly when they are joined at the skull.
Abstract- Anat - 04

DEXTROCARDIA

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Dextrocardia is a congenital defect in which the heart is situated on the right side of the body. There are two main types of dextrocardia: dextrocardia of embryonic arrest also known as isolated dextrocardia and dextrocardia situs inversus. Dextrocardia situs inversus is further divided. Dextrocardia is a condition in which the heart is pointed toward the right side of the chest instead of normally pointing to the left. It is present at birth (congenital). During the early weeks of pregnancy, the baby's heart develops. Sometimes, for reasons that are unclear, the heart develops and turns so that it points to the right side of the chest instead of the left side. There are several types of dextrocardia. Many types involve other defects of the heart and abdomen area. The simplest type of dextrocardia is one in which the heart is a mirror image of the normal heart, and no other problems exist. This condition is rare. Often in this case, the organs of the abdomen and the lungs will also be arranged in a mirror image of their normal position. For example, the liver will be on the left side instead of the right. Dextrocardia is a rare condition in which, instead of being in the left side of your chest, your heart is located in the right side.Dextrocardia is congenital, meaning that you are born with this abnormality. Less than one percent of the general population is born with dextrocardia, according to the Texas Dextrocardia situs inversus refers to the heart being a mirror image situated on the right side. For all visceral organs to be mirrored, the correct term is dextrocardia situs inversus totalis. Dextrocardia is believed to occur in approximately 1 in 12,000 people. Although statistically people with dextrocardia situs inversus do not have any medical problems from the disorder, they may be prone to a number of bowel, esophageal, bronchial and cardiovascular disorders. Certain cardiovascular and pulmonary disorders related to dextrocardia can be life-threatening if left unchecked . Kartagener syndrome may also be present in patients with dextrocardia situs inversus but also involves mirrored positioning of major internal organs and may include male infertility. There are various forms of dextrocardia, ranging from a normally configured heart that is positioned further to the right than normal (dextro-position) to so-called "mirror-image dextrocardia," in which the positions of the heart chambers and major vessels are exactly the reverse of the "normal" arrangement. Chest x-rays and an ECG (echocardiogram) may be used to determine which type of dextrocardia is present.
Abstract- Anat - 05

DEVELOPMENT OF PALATE

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The palate has two key stages of development during embryonic (primary) and an early fetal (secondary) involving the fusion of structures and a key epithelial to mesenchymal transition. The primary palate is formed by two parts: maxillary components of the first pharyngeal arch (lateral) & frontonasal prominence (midline). The secondary palate can also be divided in two anatomical parts: anterior hard palate - ossified (contributions from the maxilla and palatine bones)& posterior soft palate - muscular. The oral side of the palate is covered with a squamous stratified (pluristratified) epithelium. The surface of the hard palate of most mammalian species is further thrown into a series of transversal palatal ridges or rugae palatinae. Both the palatal ridge number and arrangement are also species specific. The development of the secondary palate commences in the sixth week of human embryological development. It is characterised by the formation of two palatal shelves on the maxillary prominences, the elevation of these shelves to a horizontal position, and then a process of palatal fusion between the horizontal shelves. The shelves will also fuse anteriorly upon the primary palate, with the incisive foramen being the landmark between the primary palate and secondary palate. This forms what is known as the roof of the mouth, or the hard palate. The formation and development of the secondary palate occurs through signalling molecules SHH, BMP-2, FGF-8 among others. Failure of the secondary palate to develop correctly may result in a Cleft palate disorder.

Tissues beneath the nasal sac enlarge & grow inferiorly to form the primary palate. It acquires the triangular shape due to the continuous growth of the maxillary process in a medial direction. During the deepening of the nasal sac & the formation of the primary palate, the ectoderm at the depth of the nasal sac proliferates to form a thickened ectodermal plate, the nasal fin, which then thins down to a thin double thickened membrane called the “oro-nasal membrane” (2 layers of ectoderm from stomodeum & nasal sac). The rupture of the oronasal membrane detaches the 1ry palate from the nasal cavity. 1ry palate & central parts of upper lip are one unit at first, then by 8wiu become separated by the vestibular lamina.
Abstract -Anat - 06

ANATOMY OF CAVERNOUS SINUS

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The cavernous sinus (or lateral sellar compartment), within the human head, is a large collection of thin-walled veins creating a cavity bordered by the temporal bone of the skull and the sphenoid bone, lateral to the sellatureica. The sinus may be joined by several anastomoses across the midline. The cavernous sinus receives blood via the superior and inferior ophthalmic veins through the superior orbital fissure and from superficial cortical veins, and is connected to the basilar plexus of veins posteriorly. The internal carotid artery (carotid siphon), and cranial nerves III, IV, V (branches V₁ and V₂) and VI all pass through this blood filled space. Infection from the face may reach the cavernous sinus through its many anastomotic connections, with severe consequences.

The cavernous sinus drains by two channels, the superior and inferior petrosal sinuses, ultimately into the internal jugular vein via the sigmoid sinus. Each cavernous sinus (one for each hemisphere of the brain) contains the following: vertically, from superior to inferior (within the lateral wall of the sinus) oculomotor nerve (CN III), trochlear nerve (CN IV), ophthalmic nerve, the V₁ branch of the trigeminal nerve (CN V), maxillary nerve, the V₂ branch of CN V.
Abstract- Anat - 07

TEMPEROMANDIBULAR JOINT

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The temporomandibular joint is the joint of the jaw and is frequently referred to as TMJ. The TMJ is a bilateral synovial articulation between the mandible and temporal bone. The name of the joint is derived from the two bones which form the joint: the upper temporal bone which is part of the cranium (skull), and the lower jawbone or mandible. There are six main components of the TMJ. They are Mandibular condyles, Articular surface of the temporal bone, Capsule, Articulardisc, Ligaments, Lateral pterygoid. The capsule is a dense fibrous membrane that surrounds the joint and incorporates the articular eminence. It attaches to the articular eminence, the articular disc and the neck of the mandibular condyle. The unique feature of the TMJs is the articular disc. The disc is composed of fibrocartilagenous tissue (like the firm and flexible elastic cartilage of the ear) which is positioned between the two bones that form the joint.

The TMJs are one of the few synovial joints in the human body with an articular disc, another being the sternoclavicular joint. There are three ligaments associated with the TMJ: one major and two minor ligaments. The major ligament is temporomandibularligament. The minor ligaments being stylomandibular ligament and the sphenomandibular ligament. To palpate the joint and its associated muscles effectively, have the patient go through all the movements of the mandible in relationship to the TMJ while bilaterally palpating the joint just anterior to the external acoustic meatus of each ear. This includes asking the patient to open and close the mouth several times and then to move the opened jaw to the left, then to the right, and then forward. To further assess the mandible moving at the TMJ, use digital palpation by gently placing a finger into the outer part of the external acoustic meatus. Auscultation of the joint can also be done.
Abstract- Anat - 08

MAXILLARY ARTERY

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The maxillary artery supplies deep structures of the face. It branches from the external carotid artery just deep to the neck of the mandible. The maxillary artery, the larger of the two terminal branches of the external carotid artery, arises behind the neck of the mandible, and is at first imbedded in the substance of the parotid gland; it passes forward between the ramus of the mandible and the sphenomandibular ligament, and then runs, either superficial or deep to the lateral pterygoid muscle, to the pterygopalatine fossa. It supplies the deep structures of the face, and may be divided into mandibular, pterygoid, and pterygopalatine portions.

The first or mandibular portion passes horizontally forward, between the neck of the mandible and the sphenomandibular ligament, where it lies parallel to and a little below the auriculotemporal nerve; it crosses the inferior alveolar nerve, and runs along the lower border of the lateral pterygoid muscle. The second or pterygoid portion runs obliquely forward and upward under cover of the ramus of the mandible and insertion of the temporalis, on the superficial (very frequently on the deep) surface of the lateral pterygoid muscle; it then passes between the two heads of origin of this muscle and enters the fossa. The third or pterygopalatine portion lies in the pterygopalatine fossa in relation with the pterygopalatine ganglion. This is considered the terminal branch of the maxillary artery.
Abstract - Anat - 09

MANDIBULAR NERVE

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The mandibular nerve (V₃) is the largest of the three branches or divisions of the trigeminal nerve, the fifth (V) cranial nerve. It is made up of two roots: a large sensory root proceeding from the inferior angle of the trigeminal ganglion, a small motor root (the motor part of the trigeminal), which passes beneath the ganglion, and unites with the sensory root, just after its exit through the foramen ovale.

The mandibular nerve gives off the following branches: From the main trunk, From the anterior division and from the posterior division. Its motor branches (here they are) go to the muscles of mastication: masseter, temporalis, and the pterygoid muscles. The mandibular nerve innervates: mylohyoid muscle and anterior belly of digastric muscle, mucous membrane of the anterior two-thirds of the tongue, the inside of the cheek (the buccal mucosa), teeth and mucoperiosteum of mandibular teeth, skin of the temporal region, auricula, lower lip, and chin, Muscles of mastication, the muscles tensor tympani and tensor velipalatini.
Abstract - Anat - 10

ORAL ANTRAL FISTULA

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A fistula is an abnormal pathway between 2 anatomic spaces or a pathway that leads from an internal cavity or organ to the surface of the body. A sinus tract is an abnormal channel that originates or ends in one opening. An orofacial fistula is a pathologic communication between the cutaneous surface of the face and the oral cavity. Oroantral fistula, an abnormal communication between the oral cavity and the maxillary sinus, is infrequently diagnosed radiologically.

The purpose of this study was to describe the CT findings and clinical features of oroantral fistula and to show that dental CT multiplanar reformatting programs can be instrumental in diagnosing this condition. A dental fistula which is also called gum boil or a parulis is an infection at the base of the tooth which forms inflamed pus. Dental fistula if not treated starts with a tooth abscess and normally ends on the gingiva or slightly in the oral vestibule.
Abstract - Anat - 11

CLEFT LIP & CLEFT PALATE

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Cleft lip (cheiloschisis) and cleft palate (palatoschisis), which can also occur together as cleft lip and palate, are variations of a type of clefting congenital deformity caused by abnormal facial development during gestation. A cleft is a fissure or opening—a gap. It is the non-fusion of the body's natural structures that form before birth. Approximately 1 in 700 children born have a cleft lip or a cleft palate or both. In decades past, the condition was sometimes referred to as harelip, based on the similarity to the cleft in the lip of a hare, but that term is now generally considered to be offensive. Clefts can also affect other parts of the face, such as the eyes, ears, nose, cheeks, and forehead. In 1976, Paul Tessier described fifteen lines of cleft. Most of these craniofacial clefts are even rarer and are frequently described as Tessier clefts using the numerical locator devised by Tessier.

A cleft lip or palate can be successfully treated with surgery, especially so if conducted soon after birth or in early childhood. If the cleft does not affect the palate structure of the mouth it is referred to as cleft lip. Cleft lip is formed in the top of the lip as either a small gap or an indentation in the lip (partial or incomplete cleft) or it continues into the nose (complete cleft). Lip cleft can occur as a one sided (unilateral) or two sided (bilateral). It is due to the failure of fusion of the maxillary and medial nasal processes (formation of the primary palate). Cleft palate is a condition in which the two plates of the skull that form the hard palate (roof of the mouth) are not completely joined. The soft palate is in these cases cleft as well. In most cases, cleft lip is also present. Cleft palate occurs in about one in 700 live births worldwide.
Abstract - Anat - 12

INTERNAL HYDROCEPHALUS OF CHILD

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Hydrocephalus is a brain condition that gets its name from the Greek word for water (meaning "hydro") and head (meaning "cephalus"). It occurs when cerebral spinal fluid (CSF) — the clear, water-like fluid that surrounds and cushions the brain and spinal cord — is unable to drain from the brain. It then pools, causing a backup of fluid in the skull. When everything is working normally inside the brain, CSF will flow through narrow passageways called ventricles and exit the brain through a small reservoir at the base of the brain called the cistern. CSF is responsible for delivering nutrients to the brain and taking waste away from sensitive areas, where it will eventually be absorbed into the bloodstream. If a blockage exists in any of the ventricles, CSF backs up and causes an excess of fluid in the brain, or hydrocephalus. This accumulation of fluid can also happen when the choroid plexus (the area of the brain that produces CSF) is in overdrive or if the fluid fails to be properly absorbed by the bloodstream. When hydrocephalus is present at birth, it can be the result of conditions like spina bifida.
Abstract - Anat - 13

CRANIAL BASE IN CRANIOFACIAL DEFORMATIES

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The cranial base is of crucial importance in integrated craniofacial development. As distinct from facial bones, it is formed through endochondral ossification. The posterior and anterior cranial bases are derived from distinct embryologic origins and grow independently—the anterior cranial base solely from the neural crest, the posterior cranial base from the paraxial mesoderm. The anterior cranial base has more prolonged and active growth and exerts more influence on facial growth than does the posterior cranial base. Cranial base angulation is a unique feature in modern human beings. Cranial base anomalies have been identified in many genetic and developmental disorders. The molecular basis of cranial base development and growth is being clarified. In this review, these aspects of cranial base are discussed in detail, with a focus on developmental features, roles in craniofacial growth, anomalies, and the genetic basis of development.
Abstract - Anat - 14

EAGLE’S SYNDROME

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A case of symptomatic calcification of the stylohyoid ligaments is described. The patient presented with head and neck pain, with the neck pain being reproduced by palpation of the styloid process through the tonsillar fossa. Calcification or ossification of the stylohyoid ligament is a frequent, often incidental finding on radiographs, however when the source of pain is from the styloid process or calcified stylohyoid ligaments it is referred to as Eagle’s syndrome. The symptoms may be confused with other causes of head and neck pain. This paper also discusses the pain patterns, clinical presentation, radiologic findings and treatment of Eagle’s syndrome. Elongated styloid process causing Eagle's Syndrome is a rare clinical entity. The diagnosis is often difficult because of its vague symptomatology. Here we present case reports of two patients, a male and a female, 46 years & 40 years respectively presented between January 2011 to October 2010. Both of the patients presented with pain in the throat, pain in the ear on swallowing for the last 06 months. Palpation of tonsillar fossa and radiological demonstration of the elongated styloid process confirms the diagnosis. Under General Anaesthesia, avulsion of the elongated processes were done through tonsillar fossa approach, after tonsillectomy.
Abstract - Anat - 15

TRIGEMINAL NEURALGIA

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The excruciating facial pain of Trigeminal Neuralgia is caused by compression of the trigeminal nerve at the root entry zone by a blood vessel in 96% of cases. The offending vessel is usually a tortuous loop of the superior cerebellar artery although other vessels can be involved. The neurosurgeon can perform direct microvascular decompression of the trigeminal nerve. This achieves long term complete pain relief in the majority of patients and avoids destruction of the trigeminal nerve, thereby avoiding the complications of facial numbness, dysesthesia, corneal anesthesia and ulceration as well as the dreaded anesthesia dolorosa (extreme constant burning pain after neural destructive procedures).

The pain of Trigeminal Neuralgia is distinctive when it manifests itself in full; in fact it is the history of the pain and only the history that can make the diagnosis. There is no diagnostic study, not even an MRI scan, that can make the diagnosis. Even the physical examination is most often normal with no evidence of sensory loss or motor weakness of the face, although touching “trigger points” on the face during the physical exam may reproduce the pain.Unfortunately, many patients with Trigeminal Neuralgia undergo unsuccessful dental procedures (tooth extractions, root canals) in the early stages of the disease. There is medical treatment for Trigeminal Neuralgia and the medication of choice is Tegretol, an anti-epileptic drug. In fact, an initial good response to Tegretol is another confirmatory characteristic of the correct diagnosis of Trigeminal Neuralgia. This fact can be very helpful in distinguishing Trigeminal Neuralgia from other conditions such as atypical facial pain. Unfortunately, the positive response of pain control that Tegretol has is just that – control, and not a cure. Although medications are extremely helpful in controlling pain for patients, they unfortunately do not cure pain and at higher doses, side effects are very common and often debilitating. The most gratifying neurosurgical procedures performed for pain are those that can completely eliminate the pain like the Microvascular Decompression procedure for Trigeminal Neuralgia. Please click on the link below for more detailed information on Trigeminal Neuralgia.
MOVEMENT OF EYE BALL

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In this work, we investigate eye movement analysis as a new sensing modality for activity recognition. Eye movement data were recorded using an electrooculography (EOG) system. We first describe and evaluate algorithms for detecting three eye movement characteristics from EOG signals—saccades, fixations, and blinks—and propose a method for assessing repetitive patterns of eye movements. We then devise 90 different features based on these characteristics and select a subset of them using minimum redundancy maximum relevance (mRMR) feature selection. We validate the method using an eight participant study in an office environment using an example set of five activity classes: copying a text, reading a printed paper, taking handwritten notes, watching a video, and browsing the Web. We also include periods with no specific activity (the NULL class). Using a support vector machine (SVM) classifier and person-independent (leave-one-person-out) training, we obtain an average precision of 76.1 percent and recall of 70.5 percent over all classes and participants. The work demonstrates the promise of eye-based activity recognition (EAR) and opens up discussion on the wider applicability of EAR to other activities that are difficult, or even impossible, to detect using common sensing modalities.
Abstract- Anat - 17

TONSIL

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Tonsils are clumps of tissue on both sides of the throat that help fight infections. Tonsils may swell when they become infected (tonsillitis). If you look down your child's throat with a flashlight, the tonsils may be red and swollen or have a white or yellow coating on them. Other symptoms of tonsillitis can include: sore throat, pain or discomfort when swallowing, fever, swollen glands (lymph nodes) in the neck. Enlarged tonsils without any symptoms are common among kids. Left alone, enlarged tonsils may eventually shrink on their own over the course of several years. Don't rely on your own guesses, though — it can be hard to judge whether tonsils are infected. If you suspect tonsillitis, contact your doctor. Recurrent sore throats and infections should also be evaluated by the doctor, who may order a throat culture to check for strep throat. Everybody's heard of tonsils. But not everyone knows what tonsils do or why they may need to be removed. Knowing the facts can help alleviate the fears of both parents and kids facing a tonsillectomy.
Abstract - Anat - 18

FACIAL NERVE

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The facial nerve carries the signals that control the movements of the facial muscles with exceptions of eye muscles innervated by third, fourth, fifth, and sixth cranial nerves, and jaw muscles innervated by the trigeminal nerve (CN V). The sensory portion of the facial nerve receives taste sensations from the anterior two-thirds of the tongue. The facial nerve originates in the brainstem in the pons very near the border with the medulla. The sensory and motor portions of the facial nerve exit the brainstem separately and do not join until the level of the internal acoustic meatus. The facial nerve travels with and shares connective tissue with the vestibulocochlear (CN VIII) to the internal acoustic meatus (near the inner ear). The facial nerve enters the temporal bone continues through the facial canal where it takes at least two very sharp turns. The nerve emerges from the stylomastoid foramen and passes through, but does not innervate, the parotid gland, where it divides into five major branches. Although these 5 branches are considered universal there are several variations.
Abstract - Anat - 19

ANATOMY OF SPEECH

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We have to first understand how human speech production works in order to create a model for machine speech. Our understanding of the anatomy of speech production can help us create a model for machine speech. In general, a speech signal is an air pressure wave that travels from the speaker's mouth to the listener's ears. Figure 1 is a schematic of the anatomy of speech production. The lung produces the initial air pressure that is essential for the speech signal; the pharyngeal cavity, oral cavity, and nasal cavity shapes the final output sound that is perceived as speech. The pharyngeal cavity and oral cavity (collectively known as the vocal tract) contracts and relaxes dynamically to create all sorts of sounds through resonance. The nasal cavity opens another air hole to create what linguists call nasal sounds (ie. /m/, /n/). Together, these cavities characterize the sounds we produce. A source-filter system produces human speech. Speech begins with a breathy source. The airflow beginning at the lungs causes sound to be produced through vibration and hissiness at the larynx (also referred to as your voicebox) in your throat. You then shape this sound through a filter, the passageways of the mouth and nasal cavity (nose). As you move your tongue around in your mouth to different areas, different tube-like vocal tract shapes are created. These shapes result in different sounds. Here are some important terms related to speech anatomy: Alveolar ridge: A bony ridge at the roof of your mouth about a half-inch behind your upper teeth. Glottis: The hole (or space) between the vocal folds in your throat. Larynx: Also referred to as the Adam's apple, it's the voice box made of cartilage in your throat that holds your vocal folds. Lips: Important for forming consonants such as in "pat," "bat," "mat," "fat," "vat," and "wet." They're protruded for some vowels. Palate: Roof of the mouth, divided into hard palate (front) and soft palate (back). Pharynx: A tube that connects the larynx to the oral cavity (mouth), located at the far back of your throat. Teeth: Used to make dental sounds such as /θ/ in "teeth" and /ð/ in "those." Tongue: The most important organ of speech production. A large muscle capable of amazing shape changes, used for speech and feeding.
Abstract - Anat - 20

PAROTID GLAND

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The parotid gland is a major salivary gland in humans. It is a bilateral structure, and the largest of the salivary glands. It is wrapped around the mandibular ramus, and secretes saliva through Stensen duct (or parotid duct) into the oral cavity, to facilitate mastication and swallowing and to begin the digestion of starches. The word 'parotid' (paraotic) literally means around the ear. The parotid glands are a pair of mainly serous salivary glands located inferior and anterior to the external acoustic meatus draining their secretions into the vestibule of oral cavity through Stensen duct or parotid duct. Each gland lies posterior to the mandibular ramus and anterior to the mastoid process of temporal bone.

The gland is effectively palpated bilaterally. Start anterior to each ear, move to the cheek area, and then inferior to the angle of the mandible. The gland is roughly wedge shaped when seen superficially and is also wedge shaped when seen on horizontal sections. The parotid duct or Stenson duct is a long excretory duct that emerges from the anterior border of the gland, superficial to the masseter muscle. The duct pierces the buccinator muscle, then opening up into the oral cavity on the inner surface of the cheek, usually opposite the maxillary second molar. The parotid papilla is a small elevation of tissue that marks the opening of the parotid duct on the inner surface of the cheek.
Abstract - Anat - 21

LARYNX – MOVEMENT OF VOCAL CORD

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The upper free border of the conus elasticus or crico vocal membrane is called vocal fold. Anteriorly it is attached to the angle of thyroid cartilage and posteriorly to the vocal process arytenoid cartilage. The space between the 2 vocal fold called rima glottidis. The anterior 3/5th of the rima glottidis is called inter membranous part and the posterior 2/5th is called inter cartilagenous part. The average length of the cord is 23mm in male and 17mm in female.

The vocal folds are sharp white fold of mucous membrane. They consist of yellow elastic tissue, it is lined by the stratified squamous epithelium. It has no submucous coat & blood vessels so they are pearly white in colour. The mucosa is firmly adherent to the underlying structure to prevent the vocal cord edema. Function of vocal cord is phonation or production of voice.

Movement of vocal card:
Tension of the vocal cord - Crico thyroid.
Relaxation of vocal cord - Thyro Arytenoid;
Abduction of vocal cord- Posterior crico arytenoid.
Adduction - Inter membranous closure by Lateral crico arytenoids, In 2nd stage Inter cartilaginlus closure by Inter or transverse arytenoids.
Abstract - Anat - 22

THYROID GLAND

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The thyroid gland or simply, the thyroid / in vertebrate anatomy, is one of the largest endocrine glands. The thyroid gland is found in the neck, below the thyroid cartilage (which forms the laryngeal prominence, or "Adam's apple"). The thyroid gland controls how quickly the body uses energy, makes proteins, and controls how sensitive the body is to other hormones. It participates in these processes by producing thyroid hormones, the principal ones being triiodothyronine (T<sub>3</sub>) and thyroxine which can sometimes be referred to as tetraiodothyronine (T<sub>4</sub>). These hormones regulate the growth and rate of function of many other systems in the body. T<sub>3</sub> and T<sub>4</sub> are synthesized from iodine and tyrosine. The thyroid also produces calcitonin, which plays a role in calcium homeostasis. Hormonal output from the thyroid is regulated by thyroid-stimulating hormone (TSH) produced by the anterior pituitary, which itself is regulated by thyrotropin-releasing hormone (TRH) produced by the hypothalamus. The thyroid gets its name from the Greek adjective for "shield-shaped", due to the shape of the related thyroid cartilage. The thyroid, from the Greek ‘thyreoeides’, meaning shield shaped, is made up of two lobes and a bridging ‘isthmus’. The gland consists of spherical ‘follicles’, which are composed of an outer basement membrane, a peripheral layer of follicular cells and a core of proteinaceous colloid. The thyroid hormones are synthesized and stored in these follicles and are dependent on an adequate iodine supply. The thyroid gland acts as a store of iodine and, in evolutionary terms, has allowed animals to migrate away from the ocean, the primary source of iodine. Thyroid hormone levels are regulated by a multiplex negative feedback loop with control from the hypothalamic–pituitary axis and autoregulation within the thyroid itself. The end product of this process is the production of the two thyroid hormones. Disease of the thyroid gland is the second most common endocrine disorder, after diabetes. Thyroid diseases ranges from the production of too much or too little of the thyroid hormones, to the development of neoplasia. Excessive release of thyroid hormones in the presence of normally functioning downstream pathways is referred to as hyperthyroidism. These patients tend to be hyperactive, heat sensitive and to lose weight. Insufficient thyroid hormone is called hypothyroidism, and is associated with a slow metabolism, making patients feel lethargic and gain weight.
Abstract - Anat - 23

MAXILLARY SINUS

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The pyramid-shaped maxillary sinus (or antrum of Highmore) is the largest of the paranasal sinuses, and drains into the middle meatus of the nose. It is present at birth as rudimentary air cells, and develops throughout childhood. The maxillary sinus was first discovered and illustrated by Leonardo da Vinci, but the earliest attribution of significance was given to Nathaniel Highmore, the British surgeon and anatomist who described it in detail in his 1651 treatise. The maxillary sinus is universally described as a pyramidal-shaped cavity in the maxilla. Hypoplasia, which can occur unilaterally or bilaterally, is graded by the authors by the degree of failure of descent below the nasal floor in achieving its position adjacent to the posterior dentition in the adult. Rarely, the sinus is excessively pneumatized in the nonpathologic state. Review of the literature failed to reveal a comprehensive study of the conditions that alter maxillary sinus volume and configuration. Based on a retrospective review of 600 high resolution Radiographs of the paranasal sinuses, the types and relative incidences of these conditions have been determined, and a classification system proposed. The mixed-sex sample group was comprised of nonpediatric (adolescent and adult) and was of a polyethnic composition. Results showed that enlargement of the sinus is uncommonly encountered, and is produced by air (pneumocele) and mucus (mucocele) entrapment, or by benign tumors which have arisen in the sinus or adjacent maxilla and have grown intracavitarily, with the sinus walls expanding and remodeling to accommodate them. Reduction in size and volume is more frequent. Heredo-familial syndromic conditions reduce sinus size by impaired facial growth centers, or obliteration by dense osteosclerosis. Irradiation for neoplastic disease in the pediatric population similarly, directly effect growth centers, or impairs pituitary function. Another iatrogenic cause, direct surgical intervention (Caldwell-Luc procedure) almost universally alters sinus volume and shape by osteoneogenesis. Midfacial fractures involving the sinus also produce distortion by sclerosis as well as by malpositioning of bone fragments. The principal systemic disorders, sickle cell anemia and osteopetrosis, which diffusely effect medullary bone, do so either through compensatory marrow proliferation or sclerotic new bone formation, thus serving to produce maxillary enlargement and sinus obliteration. The greatest source of maxillary sinus distortion and destruction are neoplasms.
DEPARTMENT OF ANATOMY
POSTER PRESENTATION

Abstract - Anat - 01

CARTILAGES OF LARYNX

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The larynx is located within the anterior aspect of the neck, anterior to the inferior portion of the pharynx and superior to the trachea. Its primary function is to protect the lower airway by closing abruptly upon mechanical stimulation, thereby halting respiration and preventing the entry of foreign matter into the airway. The cartilages of the larynx are thyroid cartilage, cricoid cartilage, arytenoids cartilage, epiglottic cartilage, interarytenoid cartilage, cuneiform process, corniculate cartilage. The thyroid cartilage is the largest of the laryngeal cartilages. The thyroid cartilage is a hyaline cartilage and forms most of the floor of the larynx. The fusion of the two lateral plates varies in different species. The rostral part forms the 'Adam's apple'. The thyroid cartilage articulates with the thyrohyoid bone and the cricoid cartilage. It becomes brittle as the animal ages. The cricoid cartilage is also a hyaline cartilage. It is signet ring shaped and is wider on the dorsal surface than the ventral surface. There is a crest on the midline of the dorsal surface and facets for arytenoid cartilages on the rostral edge. The cricoid cartilage articulates with the thyroid cartilage. The arytenoid cartilage is also a hyaline cartilage. It is paired and articulates with the rostral part of the cricoid cartilage. A vocal process is present on the caudal surface where the vocal folds attach; a muscular process extends laterally and a corniculate process extends dorsomedially. The epiglottic cartilage is an elastic cartilage, which is the most flexible and most rostral type of cartilage. The thinner stalk-like part is attached to the root of the tongue, the body of the thyroid cartilage and the basihyoid bone. The larger blade-like part lies behind the soft palate and points dorso-rostrally. During deglutition, the large blade part of the epiglottic cartilage partially covers the entrance to the trachea. The interarytenoid cartilage is a nodule of hyaline cartilage. It is located between the arytenoid cartilages dorsally. The cuneiform process is formed by elastic cartilage. It supports mucosal folds from the epiglottis to the arytenoid cartilages. It is not present in all species and can be free or fused with the epiglottic cartilages. The corniculate cartilages are 2 small, conical cartilages that articulate with the apices of the arytenoid cartilages, serving to extend them posteriorly and medially. They are located in the posterior parts of the aryepiglottic folds of mucous membrane.
Abstract - Anat - 02

MUSCLES OF EYE BALL

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The eye muscle is the fastest reacting muscle of the whole body, contracting in less than 1/100th of a second. In fact, the eye muscles work together to carry out no less than seven coordinated movements and allow the eye to track many different kinds of moving object. The intrinsic muscles of the eye serve to focus the eye and control the amount of light entering it. Each eye is held in place by three pairs of taut, elastic muscles (extrinsic muscles) that constantly balance the pull of the others. The superior rectus acts to roll the eyeball back and up, but it is opposed by the inferior rectus. In the same way, the lateral rectus pulls to the side, while the medial rectus pulls toward the nose, and the two oblique muscles roll the eye clockwise or counterclockwise. The muscles of each eye work together to move the eyes in unison. Because of the constant tension in the muscles, they can move the eye very quickly, much faster than any other body movement. The extraocular muscles are the six muscles that control movement of the eye (there are four in bovines) and one muscle that controls eyelid elevation (levator palpebrae). The actions of the six muscles responsible for eye movement depend on the position of the eye at the time of muscle contraction. Four of the extraocular muscles control the movement of the eye in the four cardinal directions: up, down, left and right. The remaining two muscles control the adjustments involved in counteracting head movement; for instance this can be observed by looking into one's own eyes in a mirror while moving one's head. The superior rectus muscle attaches to the top of the eye and moves the eye upward. The inferior rectus attaches to the bottom of the eye and moves the eye downward. The medial rectus attaches to the side of the eye near the nose and moves the eye toward the nose. The lateral rectus attaches to the side of the eye near the temple and moves the eye outward. The superior oblique is an extraocular muscle that comes from the back of the orbit and travels through a small pulley (the trochlea) in the orbit near the nose. It then attaches to the top of the eye. The superior oblique rotates the eye inward around the long axis of the eye (front to back). The superior oblique also moves the eye downward. The inferior oblique arises in the front of the orbit near the nose. It then travels outward and backward in the orbit before attaching to the bottom part of the eyeball. It rotates the eye outward along the long axis of the eye (front to back). The inferior oblique also moves the eye upward.
Abstract - Anat - 03

PAROTID GLAND

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The parotid gland is a major salivary gland in humans. It is a bilateral structure, and the largest of the salivary glands. It is wrapped around the mandibular ramus, and secretes saliva through the Stensen duct (or parotid duct) into the oral cavity, to facilitate mastication and swallowing and to begin the digestion of starches. The word 'parotid' (paraotic) literally means around the ear. The parotid, a serous compound tubulo-alveolar gland, is yellowish, lobulated, and irregular in shape. It occupies the interval between the sternomastoid muscle and the mandible. The parotid gland is enclosed in a sheath (parotid fascia) and is shaped roughly like an inverted pyramid, with three (or four) sides. It has a base (from which the superficial temporal vessels and auriculotemporal nerve emerge), apex (which descends inferior and posterior to the angle of the mandible), and lateral, anterior, and posterior (or posterior and medial) surfaces. The lateral surface is superficial and contains lymph nodes. The anterior surface is grooved by the ramus of the mandible and masseter, producing a medial lip (from which the maxillary artery emerges) and a lateral lip, under cover of which the parotid duct, branches of the facial nerve, and the transverse facial artery emerge. The posterior surface is grooved by (1) the mastoid process and the sternomastoid and digastric muscles and (2) more medially by the styloid process and its attached muscles. Medially, the superior part of the gland is pierced by the facial nerve and the inferior part by the external carotid artery. Structures that pass through the gland are from lateral to medial: (1) Facial nerve (2) Retromandibular vein (3) External Carotid artery (4) Superficial temporal artery (5) branches of the great auricular nerve. The gland is mainly irrigated by external carotid artery via the posterior auricular artery and the transverse facial. Venous return is to the Retromandibular vein. The gland is mainly drained into the preauricular or parotid lymph nodes which ultimately drain to the deep cervical chain. The gland has a capsule of its own of dense connective tissue but is also provided with a false capsule by investing layer of deep cervical fascia. The fascia at the imaginary line between the angle of mandible and mastoid process splits into the superficial lamina and a deep lamina to enclose the gland. Risorius is a small muscle embedded with this capsule substance. The gland has short striated ducts and long intercalated ducts. The intercalated ducts are also numerous and lined with cuboidal epithelial cells and have lumina larger than those of the acini. The striated ducts are also numerous and consist of simple columnar epithelium, having striations that represent the infolded basal cell membranes and mitochondria. Even though the parotid gland is the largest, provides only 25% of the total salivary volume. The serous cell predominates in the parotid, making the gland secrete a mainly serous secretory product.
Abstract - Anat - 04

FACIAL NERVE

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The facial nerve, or cranial nerve (CN) VII, is the nerve of facial expression. The pathways of the facial nerve are variable, and knowledge of the key intratemporal and extratemporal landmarks is essential for accurate physical diagnosis and safe and effective surgical intervention in the head and neck. The facial nerve is composed of approximately 10,000 neurons, 7,000 of which are myelinated and innervate the nerves of facial expression. Three thousand of the nerve fibers are somatosensory and secretomotor and make up the nervus intermedius. The motor part of the facial nerve arises from the facial nerve nucleus in the pons while the sensory and parasympathetic parts of the facial nerve arise from the nervus intermedius. The motor part and sensory part of the facial nerve enters the petrous temporal bone via the internal auditory meatus (intimately close to the inner ear) then runs a tortuous course (including two tight turns) through the facial canal, emerges from the stylomastoid foramen and passes through the parotid gland, where it divides into five major branches. Though it passes through the parotid gland, it does not innervate the gland (This is the responsibility of cranial nerve IX, the glossopharyngeal nerve). The facial nerve forms the geniculate ganglion within the facial canal at the genu, the first bend in the canal. Intracranial branches of the nerve includes greater petrosal nerve, nerve to stapedius, chorda tympani. Extracranial branches of the nerve distal to stylomastoid foramen are Posterior auricular nerve, Branch to Posterior belly of Digastric muscle as well as the Stylohyoid muscle and five major facial branches (in parotid gland) - from top to bottom (a helpful mnemonic being To Zanzibar By Motor Car): temporal, zygomatic, buccal, marginal mandibular, cervical branches. The facial nerve is developmentally derived from the hyoid arch (second pharyngeal branchial arch). The motor division of the facial nerve is derived from the basal plate of the embryonic pons, while the sensory division originates from the cranial neural crest. The main function of the facial nerve is motor control of most of the muscles of facial expression. In addition, the facial nerve receives taste sensations from the anterior two-thirds of the tongue via the chorda tympani. The facial nerve also supplies parasympathetic fibers to the submandibular gland and sublingual glands via chorda tympani. Parasympathetic innervation serves to increase the flow of saliva from these glands. It also supplies parasympathetic innervation to the nasal mucosa and the lacrimal gland via the pterygopalatine ganglion. The facial nerve also functions as the efferent limb of the corneal reflex.
Abstract - Anat – 05

EXTENSIVE EXPLORATIVE ANATOMIC EXPLOIT OF EXTRA ORDINARY TUMOUR ENTITY

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The past 20 years have seen an explosion in the understanding of how cancer cells work. Increasing knowledge of human carcinogenesis is providing many new opportunities for study of molecular targets modulated by drugs that might prevent cancer. Most of the insights have resulted from an improved understanding of the genetic basis of carcinogenesis. Specific molecules have been identified which cause the initiation and progressive growth of tumors. From this work, a fundamental re-ordering of the approach to drug discovery and development for the treatment and prevention of cancer has emerged. There is the opportunity to move away from screening agents by their effects on tumor cell growth, in vivo or in vitro, and targets that have been thoroughly exploited. While it remains true that these methods might continue to be the basis for the development of clinically useful agents, these agents may not be the best lead compounds that affect a particular pathway of biologic importance specific for cancer establishment or progression.

Drugs discovered by these early methods have historically demonstrated clear limitations in clinical efficacy. The hope is that drugs targeting new, specific molecular lesions in cancer cells will provide more selective and less toxic therapy or prevention approaches, alone or in combination with other agents. The focus of attention in this new approach to cancer drug discovery is a compound’s effect against a novel molecular target, or a target operating in a defined biochemical pathway, with the intent of causing a gain or loss in function to reverse, stop, or delay cancer progression. The cancer cell selectivity of lead compounds should be enhanced by screens based on a novel target or a critical pathway that represent a true Achilles heel. Some possible ways that preventive agents may modify molecular targets include interfering with tumor initiation by: 1) modifying carcinogen activation by enzymes responsible for activation or scavenging DNA reactive electrophiles or free radicals, 2) enhancing carcinogen detoxification processes by altering the activity of detoxifying enzymes, 3) repairing structural/functional genetic defects by enhancing endogenous DNA repair systems, 4) blocking tumor promotion and progression including altering the expression of genes involved in cell signaling, particularly those regulating cell proliferation, apoptosis and differentiation.
Abstract - Bio-01

SICKLE CELL ANEMIA

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Sickle cell anemia is one type of anemia. Anemia is a condition in which your blood has a lower than normal number of red blood cells. This condition also can occur if your red blood cells don't contain enough hemoglobin. Red blood cells are made in the spongy marrow inside the larger bones of the body. Bone marrow is always making new red blood cells to replace old ones. Normal red blood cells live about 120 days in the bloodstream and then die. They carry oxygen and remove carbon dioxide (a waste product) from your body. In sickle cell anemia, the abnormal sickle cells usually die after only about 10 to 20 days. The bone marrow can't make new red blood cells fast enough to replace the dying ones. Sickle cell anemia is an inherited, lifelong disease. People who have the disease are born with it. They inherit two genes for sickle hemoglobin—one from each parent. People who inherit a sickle hemoglobin gene from one parent and a normal gene from the other parent have a condition called sickle cell trait. Sickle cell trait is different than sickle cell anemia. People who have sickle cell trait don't have the disease. Like people who have sickle cell anemia, people who have sickle cell trait can pass the sickle hemoglobin gene to their children. Sickle cell anemia has no widely available cure. However, treatments to improve the anemia and lower complications can help with the symptoms and complications of the disease in both children and adults. Blood and marrow stem cell transplants may offer a cure for a small number of people. Sickle cell anemia varies from person to person. Some people who have the disease have chronic (long-term) pain or fatigue (tiredness). However, with proper care and treatment, many people who have the disease can have improved quality of life and reasonable health much of the time.
Abstract - Bio -02

IMMUNOGLOBULIN G

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An antibody also known as immunoglobulins is a Y shaped protein produced by B cell that is used by the immune system to identify and neutralise foreign body such as bacteria and viruses. The antibody recognizes a unique part of the foreign target called an antigen. Each tip of the "Y" of an antibody contains a paratope (a structure analogous to a lock) that is specific for one particular epitope (similarly analogous to a key) on an antigen, allowing these two structures to bind together with precision. Using this binding mechanism, an antibody can tag a microbe or an infected cell for attack by other parts of the immune system, or can neutralize its target directly (for example, by blocking a part of a microbe that is essential for its invasion and survival). The production of antibodies is the main function of the humoral immune system. Antibodies are secreted by a type of white blood cell called a plasma cell. Antibodies can occur in two physical forms, a soluble form that is secreted from the cell, and a membrane-bound form that is attached to the surface of a B cell. Antibodies are glycoproteins belonging to the glycoproteins. Antibodies are typically made of basic structural units—each with two large heavy chains and two small light chains. Antibodies can come in different varieties known as isotypes or classes. In placental mammals there are five antibody isotypes known as IgA, IgD, IgE, IgG, and IgM. Activated B cells differentiate into either antibody-producing cells called plasma cells that secrete soluble antibody or memory cells that survive in the body for years afterward in order to allow the immune system to remember an antigen and respond faster upon future exposures. IgG crosses placenta and provides immunity to the foetus.
Abstract - Bio -03

BIOCHEMISTRY OF AGEING

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In recent decades, interest in ageing has greatly accelerated, not only since the elderly form an ever-increasing percentage of the population, but because they utilize a significant proportion of the national expenditures. In addition, many people have come to the realization that one can now lead a very happy, active, and productive life well beyond the usual retirement age. Scientifically, ageing is an extremely complex, multifactorial process, and numerous ageing theories have been proposed; the most important of these are probably the genomic and free radical theories. Although it is abundantly clear that our genes influence ageing and longevity, exactly how this takes place on a chemical level is only partially understood. The accelerated ageing syndromes (i.e., Hutchinson-Gilford, Werner's, and Down's syndromes) are genetically controlled, and studies of them have decidedly increased our understanding of ageing. In addition, C. elegans and D. melanogaster are important systems for studying ageing. This is especially true for the former, in which the age-1 mutant has been shown to greatly increase the life span over the wild-type strain. This genetic mutation results in increased activities of the antioxidative enzymes, Cu-Zn superoxide dismutase and catalase. Thus, the genomic and free radical theories are closely linked. In addition, trisomy 21 (Down's syndrome) is characterized by a significantly shortened life span; it is also plagued by increased oxidative stress which results in various free radical-related disturbances. There is considerable additional indirect evidence supporting the free radical theory of ageing. Not only are several major age-associated diseases clearly affected by increased oxidative stress (atherosclerosis, cancer, etc.), but the fact that there are numerous natural protective mechanisms to prevent oxyradical-induced cellular damage speaks loudly that this theory has a key role in ageing [the presence of superoxide dismutase, catalase, glutathione peroxidase, and glutathione reductase, among others; various important intrinsic (uric acid, bilirubin, -SH proteins, glutathione, etc.) and extrinsic (vitamins C, E, carotenoids, flavonoids, etc.) antioxidants; and metal chelating proteins to prevent Fenton and Haber-Weiss chemistry]. In addition, a major part of the free radical theory involves the damaging role of reactive oxygen species and various toxins on mitochondria. These lead to numerous mitochondrial DNA mutations which result in a progressive reduction in energy output, significantly below that needed in body tissues. This can result in various signs of ageing, such as loss of memory, hearing, vision, and stamina.
Abstract - Bio -04

AIDS

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HIV infection / AIDS is a disease of the human immune system caused by infection with human immuno deficiency virus. It is transmitted primarily by unprotected sexual intercourse (anal/oral), contaminated blood transfusion, hypodermic needles, and from mother to child during pregnancy, delivery and breast feeding. Prevention of HIV infection, primarily through safe sex and needle exchange programme, is a key strategy to control the spread of the disease. There is no cure or vaccine, however anti retro viral treatment can slow down the cause of the disease and may lead to a near normal life expectancy. There are three main stages of HIV infection namely acute infection, clinical latency and AIDS. AIDS is defined in terms of either a CD4 T cell count below 200 cells or the occurrence if specific diseases in association with HIV infection. HIV is a retrovirus that infects components of human immune system such as CD4 T cells, macrophages and dendritic cells. Upon entering to the target cell, the viral RNA genome is converted into doubles stranded DNA by a virally encoded reverse transcriptase that is transported along with the viral genomes in the virus particles. The resulting viral DNA is then imported into the cell nucleus and integrated into the cellular DNA by a virally encoded integrated and host co factors. There are two types of HIV which are HIV 1 and HIV 2. HIV 1 is the virus that was originally discovered. It is more virulent, more infective and a major cause of HIV infection globally.
Abstract - Bio -05

INSULIN

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Insulin is a peptide hormone produced by the beta cells of the pancreas and it's central to regulating carbohydrate and fat metabolism of the body. It causes the cells in the liver, skeletal muscles and fat tissue to absorb glucose from the blood. Insulin stops the use of fat of energy source by inhibiting the release of glucagon except in the presence of the metabolic disorder, diabetes mellitus and metabolic syndrome insulin is provided in the body in a constant proportion to remove excess glucose from the blood which otherwise will be toxic. When control of insulin fails, diabetes mellitus can result. As a consequence, insulin is used medically to treat some forms of diabetes mellitus. Patient with type one diabetes depend on external insulin (inj sub cutaneous) for their survival as the hormone is no longer produced internally. Patients with type two diabetes, are often insulin resistant and because of such resistance they may suffer from a relative insulin deficiency. Human insulin protein is composed of 51 amino acids and has a molecular weight of 5808 Da. It is a dimer of e chain and b chain which are linked together by disulphide bond. Bio synthetic human insulin for clinical use is manufactured by recombinant DNA technology. Biosynthetic human insulin has increased purity when compared with extractive animal insulin enhanced purity reducing antibody formation. Unlike any medications insulin currently can't be take orally because like nearly all other proteins introduced into the GIT, it is reduce into fragments and the activity is lost. There has been some research into ways to protect insulin from the digestive tract so that it can be administered orally or sub lingually.
Abstract - Bio -06

DIABETES AND DENTAL DISEASE

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Diabetes mellitus affects an estimated 20 million of the population, and both the incidence and prevalence are increasing every year. The two main types of diabetes are classified primarily on the basis of their underlying pathophysiology. Type 1 diabetes, results from autoimmune destruction of insulin-producing β-cells in the pancreas, leading to total loss of insulin secretion. Insulin is used by the body to facilitate the transfer of glucose from the bloodstream into the target tissues, such as muscle, where glucose is used for energy. Because a person with type 1 diabetes no longer produces endogenous insulin, glucose is unable to enter target cells and remains in the bloodstream, resulting in sustained hyperglycemia. A patient with type 1 diabetes must take exogenous insulin to remain alive—hence, the former name “insulin-dependent diabetes. Type 2 diabetes results from insulin resistance rather than from total absence of insulin production. Autoimmune destruction of β-cells does not occur in type 2 diabetes, and patients retain the capacity to secrete some insulin, although production often diminishes over time. Patients with type 2 diabetes can remain undiagnosed for years because hyperglycemia appears gradually and often without symptoms. Insulin resistance results in a decreased capacity to transfer glucose into target cells; thus, hyperglycemia develops. A large evidence base suggests that diabetes is associated with an increased prevalence, extent and severity of gingivitis and periodontitis. Furthermore, numerous mechanisms have been elucidated to explain the impact of diabetes on the periodontium. While inflammation plays an obvious role in periodontal diseases, as a major component in the pathogenesis of diabetes and diabetic complications. Research suggests that, as an infectious process with a prominent inflammatory component, periodontal disease can adversely affect the metabolic control of diabetes. Conversely, treatment of periodontal disease and reduction of oral inflammation may have a positive effect on the diabetic condition, although evidence for this remains somewhat equivocal.
Abstract - Bio -07

POLYMERASE CHAIN REACTION

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The polymerase chain reaction (PCR) is arguably the most powerful laboratory technique ever developed in 1987 by Kary Mullis. With this technique it is possible to make virtually unlimited copies of a single DNA molecule even though it is initially present in a mixture containing many different DNA molecules. It is used to amplify a specific DNA (target) sequence lying between known positions (flanks) on a double-stranded (ds) DNA molecule. The polymerase chain reaction can be used to amplify both double and single stranded DNA. The ease with which it can be done, the relatively low cost, and its unique combination of specificity and sensitivity coupled with great flexibility has led to a true revolution in genetics. The advent of kinetic, or Real-Time PCR has served to add yet another dimension to this cognitive dissonance, particularly in the realms of experimental as well as primer and probe design along with optimization of experimental conditions. Moreover, as correctly pointed out by Bustin “The comparative ease and rapidity with which quantitative data can be acquired using real-time RT-PCR assays has generated the impression that those data are reliable and can be subjected to objective analysis”. The method was first formally presented at the American Society of Human Genetics Conference in October of 1985 and the first clinical application for PCR, an analysis of sickle cell anemia, was published the same year. The PCR components are as Water, PCR Buffer, MgCl₂, dNTPs, Forward Primer, Reverse Primer, Target DNA, Polymerase.

Water is present to provide the liquid environment for the reaction to take place. It is the matrix in which the other components interact. To maintain a sterile environment, deionized water is the choice. The primary purpose of PCR Reaction Buffer is for providing an optimal pH and monovalent salt environment for the final reaction volume. Many commercially supplied PCR buffers already contain magnesium chloride (MgCl₂). MgCl₂ supplies the Mg++ divalent cations required as a cofactor for Type II enzymes, which include restriction endonucleases and the polymerases used in PCR. The standard final concentration of this reagent for polymerases used in PCR is 1.5mM. deoxynucleotide triphosphates (dNTPs) are used for providing the only source of that energy is the β and γ phosphates of the individual dNTPs. The selection of the target DNA is important, because the target DNA should be as pure as possible but also it should be uncontaminated by any other DNA source. Plasmid DNA is small and highly enriched for the specific target sequence while genomic DNA will usually contain only one copy of the target sequence per genome equivalent.
Abstract - Bio -08

TOBACCO IN CANCER

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Tobacco is one of the most widely used predominantly in India. For both genders, cancer of the mouth and pharynx ranks sixth overall in the world; it is also the third most common site among males in developing countries. In industrialized countries, men are affected two to three times as often as women, largely due to higher use of alcohol and tobacco. Ethnicity strongly influences prevalence due to social and cultural practices, as well as socioeconomic differences. In population terms, survival rates around the world show little improvement. In terms of etiology, the effects of tobacco use, heavy alcohol consumption, and poor diet together explain over 90 percent of cases of head and neck cancer. It is significantly high in usage in, Both in chewable form or through usage of cigarettes. It is one of the predisposing agents for cancer. It can predispose to cancer in sites like the oral cavity, lip, lungs etc. There are various forms of chewable tobacco such as Smokeless Tobacco, Betel Quid, Areca nut, Oral snuff. The most comprehensive source of evidence for the carcinogenicity of tobacco smoke remains the IARC publication of 1986. A major difficulty in accurately quantifying smoking risks for aerodigestive tract cancer is its strong synergism with alcohol. Stomatitis nicotina, in the West is the most commonly associated with pipe smoking and both hard and soft palate are relatively uncommon sites of oral cancer. The major chemical components in tobacco smoke are tobacco specific nitrosamines (TSN), nitrosornicotine (NNN), nitrosopyrrolidine (NPyR), nitrosodimethylamine (NDMA), and 4-(methyl)nitrosouanine -1-(3-pyridyl)-1-butaneone (NNK). These are usually generated during pyrolysis, and also endogenously absorbed as smokeless tobacco. They act on the keratinocyte stem cells, and produce DNA adducts, especially 06 Methyl Guanine which interferes with DNA replication. There is damage to all replicating cells including those of the immune response. Metabolism usually involves utilization of cytochrome p450 enzymes and conjugation with glutathione S transferase enzyme. Polymorphism of p450 and GST genes are currently the markers for head and neck cancers. Winn et al. found increased risks associated with regular use of mouthwash, of 40 percent for men and 60 percent for women, after adjusting for alcohol drinking and tobacco use. Risks generally increased in proportion to frequency and duration of mouthwash use, and were only apparent when the alcohol content of the mouthwash percent increased to greater than 25%.
Abstract - Bio -09

ALZHEIMERS DISEASE

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It is a neurodegenerative disease, which means there is progressive brain cell death that happens over a course of time. The total brain size shrinks with Alzheimer's - the tissue has progressively fewer nerve cells and connections. While they cannot be seen or tested in the living brain affected by Alzheimer's disease, postmortem/autopsy will always show tiny inclusions in the nerve tissue, called plaques and tangles. Plaques are found between the dying cells in the brain - from the build-up of a protein called beta-amyloid (you may hear the term "amyloid plaques"). The tangles are within the brain neurons - from a disintegration of another protein, called Tau. Risk factors associated with Alzheimer's disease include older people (over-75), Family history (inheritance of genes), Having a certain gene (the apolipoprotein E or APOE gene) and a female sex predilection (more women than men are affected). There are certain modifiable factors such as Factors that increase blood vessel (vascular) risk - including diabetes, high cholesterol and high blood pressure, Low educational and occupational attainment, Prior head injury, Sleep disorders (the breathing problem sleep apnea), Estrogen hormone replacement therapy. The stages of Alzheimer's disease are 3 steps as follows; Preclinical (no signs or symptoms), Mild cognitive impairment and Dementia. The Alzheimer's Association has broken this down further, describing seven stages along a continuum of cognitive decline based on symptom severity - from a state of no impairment, through mild and moderate decline, and eventually reaching "very severe decline."
Free radicals (e.g., superoxide, nitric oxide, and hydroxyl radicals) and other reactive species (e.g., hydrogen peroxide, peroxynitrite, and hypochlorous acid) are produced in the body, primarily as a result of aerobic metabolism. Antioxidants (e.g., glutathione, arginine, citrulline, taurine, creatine, selenium, zinc, vitamin E, vitamin C, vitamin A, and tea polyphenols) and antioxidant enzymes (e.g., superoxide dismutase, catalase, glutathione reductase, and glutathione peroxidases) exert synergistic actions in scavenging free radicals. Dietary antioxidants are useful radioprotectors and play an important role in preventing many human diseases (e.g., cancer, atherosclerosis, stroke, rheumatoid arthritis, neurodegeneration, and diabetes). Free radicals are defined as molecules having an unpaired electron in the outer orbit. They are generally unstable and very reactive. Examples of oxygen free radicals are superoxide, hydroxyl, per-oxyl (RO•2), alkoxyl (RO’), and hydroperoxyl (HO•2) radicals. Nitric oxide and nitrogen dioxide (NO•) are two nitrogen free radicals. Oxygen and nitrogen free radicals can be converted to other non-radical reactive species, such as hydrogen peroxide, hypochlorous acid (HOCl), hypobromous acid (HOB), and per-oxynitrite (ONOO•). Calorie restriction reduces the generation of free radical species and retards aging in animals. Under physiologic conditions, approximately 1% to 3% of the O2 consumed by the body is converted into superoxide and other ROS. Throughout the life cycle, any person may be at a risk of oxidative stress induced by high rates of oxygen use (e.g., strenuous work and competitive sports), the autoimmune activation of immune system cells (e.g., respiratory burst of polymorphonuclear and mononuclear cells), and environmental factors (e.g., pollutants containing NO, nitro-gen dioxide, and hydroxyl radicals). Prolonged exposure to free radicals, even at a low concentration, may result in the damage of biologically important molecules and potentially lead to DNA mutation, tissue injury, and disease. The removal of free radicals is achieved through enzymatic and non-enzymatic reactions. The recent advances in biochemistry and molecular biology techniques provide new, powerful tools for studying the expression of tissue antioxidant enzymes and for elucidating the mechanisms of the actions of antioxidants. For example, a number of knockout mice models, such as SOD, glutathione peroxidase, and eNOS null mice, have been developed to determine the specific roles of the defense systems under physiologic conditions. This is an exciting time for antioxidant research in the space station era.
Abstract - Bio -11

THALASSEMIA

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Thalassemia is a genetic disorder in which one of the two proteins that make up hemoglobin in red blood cells is deficient. Hemoglobin helps carry oxygen through the blood to all parts of the body. Most forms of thalassemia produce a chronic lifelong anemia that begins in early childhood and often must be treated with frequent transfusions. Significant, often life-threatening complications are common in the most severe forms. Cooley’s anemia (beta thalassemia major) is a severe form of thalassemia that requires regular, often monthly, blood transfusions. An estimated 1,000 people have Cooley’s anemia in the United States, and an unknown number are carriers – people who have the genetic trait and can pass it on to their children. Thalassemia is most common among people of Mediterranean descent, such as Italians and Greeks, and is also found among people from the Arabian Peninsula, Iran, Africa, Southeast Asia, and Southern China. Because many affected families are recent immigrants belonging to these ethnic groups they face cultural and language challenges that may impede their ability to seek appropriate care and understand the resources available for living with thalassemia. The thalassaemias result from inherited defects in the synthesis of the globin chains of haemoglobin. Humans have different haemoglobins at various stages of development. Normal adults have a major haemoglobin (Hb) called HbA, comprising about 90% of the total, and a minor component, HbA₂, which accounts for 2–3%. The main haemoglobin in fetal life is HbF, traces of which are found in normal adults. There are three embryonic haemoglobins. The genetics of a thalassaemia is complicated because normal humans receive two a genes from each parent, a genotype that is written aa/aa. There are two main classes of a thalassaemia. First, there are the a- thalassaemias, in which both a genes are deleted; that is, all or part of the gene is missing. On the other hand, in the a-thalassaemias only one of the a genes is lost. B- thalassaemia, like a- thalassemia, may result from a partial or complete deletion of the b globin gene. Some of these mutations cause an absence of b-chain production and the resulting disease is called b-thalassaemia, whereas others result in a reduced output of b chains, b-thalassaemia. Some of the latter forms are extremely mild and may not be identifiable in carriers; most heterozygotes for b-thalassaemia have very mild anaemia and a raised level of HbA₂.
Abstract - Bio -12

RECOMBINANT DNA TECHNOLOGY

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Recombinant DNA is a form of artificial DNA that is made through the combination or insertion of one or more DNA strands, therefore combining DNA sequences as per your requirement. The mRNA transcription from this DNA fragment followed by translation involving rRNA and tRNA that carries the amino acid must be clearly understood before partaking in Recombinant DNA technology. Recombinant DNA technology has been used in various non-medical fields such as Agriculture, such as growing crops resistant to pesticide, fruits with attractive colors, and other uses such as artificial insulin drug delivery, gene and antiviral therapy, vaccination, fluorescent fishes, etc. The advantages of Recombinant DNA technology are that it provides substantial quantity, developing resistance to natural inhibitors, it is economical, it has unlimited utilization, and the products are tailor-made according to the use. Some of the drawbacks of Recombinant DNA technology are since it has become widely used, it has become more commercial than practical usage, affects natural immunity of the body, it has the ability to destroy the natural ecosystem, and chance to produce mutation within a given system. The process of Recombinant DNA technology is to isolate DNA followed by cutting with restriction enzymes and ligating into cloning vector. This is then transformed into recombinant DNA molecule and introduced into host cell following which each transformed cell will divide many times to form a colony of millions of cells, each of which carries the recombinant DNA molecule (DNA clone). The vectors commonly used in gene cloning were originally derived from two natural sources Plasmids and Viruses. Many naturally occurring plasmids have selectable markers. Restriction enzymes are made naturally by many species of bacteria. They protect bacterial cells from invasion by foreign DNA, particularly that of bacteriophage. Restriction enzymes bind to specific DNA sequences. These are typically palindrome. Some restriction enzymes digest DNA into fragments with “sticky ends”. These DNA fragments will hydrogen bond to each other due to their complementary sequences. Other restriction enzymes generate blunt ends. The net result of gene cloning is to produce an enormous amount of copies of a gene. During transformation, a single bacterial cell usually takes up a single copy of a vector. Amplification of a cloned gene occurs in two ways: The vector gets replicated by the host cell many times. This will generate a lot of copies per cell (25-50 for plasmids) & The bacterial cell divides approximately every 20 minutes.
Abstract - Bio -13

GENE THERAPY IN CANCER

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Gene therapy promises a number of innovative treatments that are likely to become important in preventing deaths from cancer. Immunotherapy uses genetically modified cells and viral particles to stimulate the immune system to destroy cancer cells. Oncolytic virotherapy, which uses viral particles that replicate within the cancer cell to cause cell death, is an emerging treatment modality that shows great promise, particularly with metastatic cancers. Gene transfer is a new treatment modality that introduces new genes into a cancerous cell or the surrounding tissue to cause cell death or slow the growth of the cancer. This treatment technique is very flexible, and a wide range of genes and vectors are being used in clinical trials with successful outcomes. As these therapies mature, they may be used alone or in combination with current treatments to help make cancer a manageable disease. The systemic toxicity of chemotherapy regimens, while not as severe as they once were, still often result in acute and delayed nausea, mouth ulcerations and mild cognitive impairments. In addition, long-term side effects from chemotherapy can include an increased risk of developing other types of cancers. Less serious, but potentially just as debilitating, side effects can also occur. Treatment for metastatic prostate cancer, while prolonging life, often causes hot flashes, impotence, incontinence and an increased risk of bone fractures. Immunotherapy, or the concept of boosting the immune system to target and destroy cancer cells, has been a goal of cancer treatment for over several years. Currently gene therapy is being used to create recombinant cancer vaccines. Unlike vaccines for infectious agents, these vaccines are not meant to prevent disease, but to cure or contain it by training the patient’s immune system to recognize the cancer cells by presenting it with highly antigenic and immunostimulatory cellular debris. Initially cancer cells are harvested from the patient (autologous cells) or from established cancer cell lines (allogeneic) and then are grown \textit{in vitro}. These cells are then engineered to be more recognizable to the immune system by the addition of one or more genes, which are often cytokine genes that produce pro-inflammatory immune stimulating molecules, or highly antigenic protein genes.
Abstract - Bio -14

JAUNDICE

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Jaundice, a clinical condition characterized by yellowish pigmentation of the skin, mucous membrane and the sclera is caused by the elevated serum bilirubin level in the blood. The term jaundice is derived from a French word which means yellow. It is also called as icterus which is derived from the Greek word icteric. The normal concentration of the bilirubin in blood is usually less than 1mg/dl. Increase in the concentration of the serum bilirubin above 2mg/dl leads to jaundice. This hyperbilirubinemia leads to increased bilirubin level in the extra cellular fluid. Jaundice manifests initially as yellowish discoloration of the conjunctival membranes of the eye which is referred to as Conjunctival icterus. Jaundice is often seen as a manifestation of hepatitis or liver cancer. It may also indicate leptospirosis or obstruction of the biliary tract. Depending upon the physiology affected, jaundice is categorised as Haemolytic jaundice (pre-hepatic) , Hepatocellular jaundice (hepatic) and Cholestatic jaundice (post-hepatic). Pre hepatic jaundice or haemolytic jaundice is caused following an increased rate of haemolysis in some of the diseases like thalassemia, sickle cell anaemia, glucose 6 phosphate deficiency. It is presented by an increase in the formation and excretion of urobilinogen with an elevated serum unconjugated bilirubin. Hepatic jaundice or Hepatocellular jaundice can be caused by liver diseases like acute or chronic hepatitis, hepatotoxicity, cirrhosis, drug induced hepatitis and alcoholic liver disease. It is characterised by an increased level of unconjugated bilirubin due to the inability of the necrotising liver cells to metabolise bilirubin. It is also presented with increased activities of Alkaline phosphatase, alanine transaminase and aspartate transaminase. Obstructive jaundice or post hepatic jaundice is caused by an obstruction to the of bile duct which can be due to gallstones, pancreatic cancer, liver flukes, biliary atresia, cholangiocarcinoma and pancreatitis. In case of complete obstruction of the bile duct, no urobilinogen is found in the urine whereas there is excretion of conjugated bilirubin.
Abstract - Bio -15

IMMUNOGLOBULINS

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An antibody (Ab), also known as an immunoglobulin (Ig), is a large Y-shape protein produced by B cells that is used by the immune system to identify and neutralize foreign objects such as bacteria and viruses. The antibody recognizes a unique part of the foreign target, called an antigen. Each tip of the "Y" of an antibody contains a paratope that is specific for one particular epitope on an antigen, allowing these two structures to bind together with precision. Using this binding mechanism, an antibody can tag a microbe or an infected cell for attack by other parts of the immune system, or can neutralize its target directly. The production of antibodies is the main function of the humoral immune system. Igs are produced by the lymphocytes and are found in fraction of blood called gamma globulin. Gerald M. Edelman and Rodney Robert Porter are the notable researchers who worked extensively on purification and structural analysis of Igs, particularly the IgG type.

The basic structure of immunoglobulins are as follows- it consists of 2 identical light chains and 2 identical heavy chains, the heavy and light chains are joined together by interchain disulphide bonds and non-covalent interactions. The number of interchain disulphide bonds varies among different Igs. Within the polypeptide chains i.e. the heavy and light chains there are also present intra-chain disulphide bonds. Amino acid sequence of both heavy and light chains of an Ig characterizes two distinct regions of the chains based on variability of the amino acid sequence, known as VARIABLE (V) and CONSTANT (C) regions. Light and heavy chains are composed of both a variable and constant region designated VL and CL (light chains) and VH and CH (heavy chains). The amino acid sequence of the variable region form the N-terminal ends of the chains and determine antigenic specificity of the Igs. Constant regions are the same for each specific class of Ig and carry the effector sites. Light chain-VL-about 100-110 amino acids, CL-100-110 amino acids. There are two types of light chains, kappa and lambda, (κ and λ) the κ are twice as much as λ. Heavy chains-VH-110 amino acids, CH-330-440 amino acids. There are 5 types of heavy chains which defines the class of Igs, namely, Alpha, Gamma, Miu, Delta and Epsilon (α,γ,µ,δ,ε). The heavy chains are between 53-75KDa. The variable region makes up a quarter of the entire heavy chain while ¾ of the remaining chain is the constant region.
Abstract - Bio -16

BIO INFORMATICS

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Bioinformatics is the application of statistics and computer science to the field of molecular biology. It is a field of science which formulates methods for storing, retrieving, organizing and analyzing the biological data. Paulien Hogeweg coined the term Bioinformatics in 1979. It is primarily used in genomics and genetics, involving large-scale DNA sequencing from the 1980’s. Bioinformatics includes various sectors such as computer science, statistics, mathematics and engineering to process and organise the biological data. Nowadays complex machines are used to scan in the biological data at a much faster rate. Databases and information systems are the storage material used for storing and organizing biological data. Algorithms of artificial intelligence, soft computing, data mining, image processing, and simulation based on discrete mathematics, control theory, system theory, information theory, and statistics are used in analyzing the biological data. The actual process of analyzing and interpreting data is referred to as computational biology. Important sectors involved in bioinformatics and computational biology include the development and implementation of tools that enable access various types of information and the development of new algorithms (mathematical formulas) and statistics to assess relationships among members of large data sets. Major research efforts in the field include sequence alignment, gene finding, genome assembly, drug design, drug discovery, protein structure alignment, protein structure prediction, prediction of gene expression and protein–protein interactions, genome-wide association studies, and the modelling of evolution. The current progress of bioinformatics includes the creation and advancement of databases, algorithms, computational and statistical techniques, and theory to solve formal and practical problems arising from the management and analysis of biological data. Common activities in bioinformatics include mapping and analyzing DNA and protein sequences, aligning different DNA and protein sequences to compare them, and creating and viewing 3-D models of protein structures which is done by two approaches which are the static and the dynamic approach.
Abstract - Bio -17

NATURAL ANTIOXIDANTS

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An antioxidant is a molecule which inhibits the oxidation of other molecules. Oxidation reactions produce free radicals which in turn, start Chain reaction chain reactions that can cause damage or death to the cell. Antioxidants terminate these chain reactions by scavenging the free radical production. They are also called reducing agents. Antioxidants are classified into two broad divisions which are the water soluble and the lipid soluble. The different antioxidants present at a wide range of concentrations in the body include glutathione ubiquinone, uric acid etc. Antioxidants that are naturally found in the food include polyphenols, tocopherols, ascorbic acid and carotenoids. A polyphenol antioxidant contains a polyphenol or natural phenol. The main source of polyphenols is dietary, honey; legumes; fruits such as apples, pomegranate, grapes, strawberries, broccoli, cabbage, onion etc. It helps in scavenging free radicals and up-regulate certain metal chelation reactions. Ascorbic acid is well known for its antioxidant activity in liquids. It has an impact on cardiovascular disease, hypertension, chronic inflammatory diseases. The ascorbate ion is oxidized to form a radical cation and dehydroascorbic acid. It reacts with oxidants of the reactive oxygen species, such as the hydroxyl radical. Carotenoids are organic pigments that are found in the chloroplasts and chromoplasts of plants and some other photosynthetic organisms, including some bacteria and some fungi. Carotenoids are efficient free-radical scavengers, and they enhance the immune system. Epidemiological studies have shown that people with high β-carotene intake and high plasma levels of β-carotene have a significantly reduced risk of lung cancer. As an antioxidant, vitamin E or tocopherol, a fat soluble vitamin acts as a peroxyl radical scavenger, preventing the propagation of free radicals in tissues, by reacting with them to form a tocopheryl radical, which will then be reduced by a hydrogen donor (such as vitamin C) and thus return to its reduced state. It is incorporated into cell membranes as a protection to oxidative damage. Vitamin E has also found use as a commercial antioxidant in ultra high molecular weight polyethylene (UHMWPE) used in hip and knee replacements, to help resist oxidation.
Abstract - Bio -18

PROSTAGLANDINS

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The prostaglandins are a group of lipid compounds that are derived enzymatically from fatty acids. Every prostaglandin contains 20 carbon atoms, including a 5-carbon ring. They have strong physiological effects, such as regulating the contraction and relaxation of smooth muscle tissue. They differ from hormones in that they are produced in many places throughout the human body. The prostaglandins, the thromboxanes and prostacyclins constitute the prostanoid class, a subclass of eicosanoids. The eicosanoids are used as signalling molecules. The prostaglandins are synthesized from arachidonic acids. An intermediate arachidonic acid is created from diacylglycerol via phospholipase-A2, then brought to either the cyclooxygenase pathway or the lipoxygenase pathway to form either prostaglandin and thromboxane or leukotriene respectively. The cyclooxygenase pathway produces thromboxane, prostacycline and prostaglandin D, E and F. Alternatively, the lipoxygenase enzyme pathway is active in leukocytes and in macrophages and synthesizes leukotrienes. Prostaglandins are produced following the sequential oxidation of AA, DGLA or EPA by cyclooxygenases (COX-1 and COX-2) and terminal prostaglandin synthases. Prostaglandins ligate a sub-family of cell surface seven-transmembrane receptors, G-protein-coupled receptors. The functions involved by the prostaglandin are brought about by the combination of the types of the prostaglandins which include PGI, PGF, and PGE. The functions include the control of the hormonal and cell growth regulation, termination and induction of pregnancy, regulate the calcium metabolism and transport, decreases the immunological function, controls the thermoregulatory center in the hypothalamus and also causes pain in association with histamine and bradykinin. They are also used for the treatment of gastric ulcers by inhibiting the acid secretion, aggregation of the platelets and vice versa, constriction or dilation in vascular smooth muscle cells.
Abstract - Bio -19

TRANSLATION

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The biosynthesis of a protein or a polypeptide in the cellular ribosome is referred to as the translation. The gene expression stored in the DNA is transferred to the RNA through the process of transcription which is coded in the form of proteins through translation. The ribosomal complex decodes the information from the messenger RNA to produce a specific amino acid sequence, or polypeptide, which complexes to form an active protein. Translation occurs in various sites in various organisms. In bacteria, translation occurs in the cell’s cytoplasm in which the ribosome are located whereas in eukaryotes, translation occurs in the endoplasmic reticulum (vectorial synthesis). The ribosome stimulates the tRNA to bind with complementary anti-codon sequences of the mRNA. The specific amino acids of the tRNA are chained together into a polypeptide as the mRNA passes through and is read by the ribosome in a pattern called the ticker tape. Translation includes four phases which are initiation, elongation, translocation and termination. The growing cells produce larger amounts of protein eg. Liver cells. The amino acid is attached to the enzyme, by using ATP which is later transferred to the 3’ end of the tRNA to form a primary complex which activates the reaction. Initiation involves the binding of the small subunit of the ribosome to the 5’ end of mRNA with the help of initiation factors (IF) and The elongation of the polypeptide chains is by sequential addition of amino acids which is determined by the codons in the specific mRNA. the Termination of the polypeptide happens when the A site of the ribosome faces a stop codon. The three nucleotide base sequences in mRNA that code the amino acids constitute the genetic code. The codons AUG and GUG are the chain initiating codons whereas UAA, UAG and UGA are the stop codons.
DEPARTMENT OF BIOCHEMISTRY

POSTER PRESENTATION

Abstract - Bio -01

OSTEOPOROSIS-A SILENT THIEF

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Osteoporosis is a silent, multifactorial disease characterized by low bone mass and an increased risk of fracture. A variety of risk factors have been associated with the disease and include menopause, aging, oxidative stress, genetics and lifestyle. It is a serious public health problem that affects 25 million people in the United States, 80% of whom are women. It is often referred to as the ‘silent thief’ as it remains asymptomatic until the incidence of fracture. Osteoporosis does not discriminate by age; in fact, it is a geriatric disease with anadolescentonset.Osteoporosis affects every bone in the body, but the most common places where fractures occur are vertebral(32%), lower arm(16% and laps(15%). Osteoporosis develops in three stages: 1) Bone building, from childhood through early adulthood, 2) Osteopenia, when evidence of reduced bone mass is detected and 3) Osteoporosis, when bone loss is unmistakable. The multifactorial nature of the disease combined with complex molecular mechanistic interactions underlying its pathology, necessitate development of a system level understanding of the disease. Although current models for osteoporosis take into account roles of only a fraction of the many risk factors associated with the disease, they provide the framework for developing integrated, holistic models which can help us design high efficacy preventative and therapeutic strategies against the disease.
Abstract - Bio -02

Type 1 And Type 2 Diabetes

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Type 1 (insulin-dependent) diabetes occurs worldwide and can appear at any age. The genetic susceptibility is strongly associated with HLA-DQ and DR on chromosome 6, but genetic factors on other chromosomes such as the insulin gene on chromosome 11 and the cytotoxic T-lymphocyte antigen gene on chromosome 2 may modulate disease risk. Numerous studies further support the view that environmental factors are important. Gestational infections may contribute to initiation, whereas later infections may accelerate islet β-cell autoimmunity. The pathogenesis is strongly related to autoimmunity against the islet β cells. Markers of autoimmunity include autoantibodies against glutamic acid decarboxylase, insulin, and islet cell antigen-2, a tyrosine phosphatase-like protein. Type 2 diabetes mellitus is a chronic metabolic disorder that results from defects in both insulin secretion and insulin action. An elevated rate of basal hepatic glucose production in the presence of hyperinsulinemia is the primary cause of fasting hyperglycemia; after a meal, impaired suppression of hepatic glucose production by insulin and decreased insulin-mediated glucose uptake by muscle contribute almost equally to postprandial hyperglycemia. Type 2 diabetes can cause severe complications, affecting the eye, the nervous system and the kidney. The overall risk of cardiovascular disease is more than doubled, and life expectancy is reduced by an average 7 years. Type 1 and type 2 diabetes are characterized by progressive β-cell failure. Apoptosis is probably the main form of β-cell death in both forms of the disease. It has been suggested that the mechanisms leading to nutrient- and cytokine-induced β-cell death in type 2 and type 1 diabetes, respectively, share the activation of a final common pathway involving interleukin (IL)-1β, nuclear factor (NF)-κB, and Fas. Management of diabetes is best undertaken in the context of a multidisciplinary health team and requires continuing attention to many aspects, including insulin administration, blood glucose monitoring, meal planning, and screening for comorbid conditions and diabetes-related complications.
Abstract - Bio -03

Diagnosis Of Oral Cancer

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Oral squamous cell carcinoma is the sixth most common cancer for both sexes worldwide. The 5-year survival rate of the disease is currently about 50%. Oral cancer is often diagnosed only after it has advanced to an untreatable stage where the cancer cells have become aggressive and immune to therapeutic drugs. Detecting oral cancer at its earliest is thus vital for improving the survival rate of this disease. Current clinical diagnosis of most epithelial cancers, including oral cancer, typically involves performing invasive needle biopsies followed by histological examination on the excised tissue. The procedure may present psychological trauma and risk of infection to patients. Furthermore, biopsy is usually performed only under the condition that the lesions are spotted and appear abnormal. Yet, pre-cancerous lesions can appear innocuous or occur in hidden sites such as the crypts in the base of tongue, and can therefore easily go undetected even with white-light endoscopy. Furthermore, conventional histopathological diagnosis is based on morphological and structural changes at the cellular or tissue level, which may not be obvious for early-stage tumors. Taken together, it is clear that a diagnostic method for detecting early stage oral cancer is highly desired.

Recently, an increased amount of efforts has been made to develop less-invasive early diagnostic modalities for oral cancer, of which the in vivo high resolution imaging of oral epithelial tissues using novel optical systems and the chemical analysis of saliva hold great promises as valuable tools. Although advanced optical systems for in vivo imaging such as optical coherence tomography (OCT) and confocal reflectance endomicroscopy are designed to image cell and stromal morphology for non-invasive clinical diagnosis in real time, the contrast between neoplastic and normal tissues is often too low to be of any clinical value. Gold nanoparticles also possess other favorable physicochemical properties for use as optical probes for early diagnostics. A cancer diagnostic algorithm, light-induced autofluorescence spectroscopy using double excitation wavelengths, was also employed for distinguishing between cancerous and normal oral mucosa.
Abstract - Bio -04

Multiple Myeloma

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Multiple myeloma is a malignant disease characterized by plasmacytosis, paraprotein production, bone lesions, hypercalcemia, susceptibility to infections, and renal impairment. The underlying pathophysiologic phenomena of the clinical features include suppression of humoral- and cell-mediated immunity, elevation of IL-6, abnormalities of the bone marrow microenvironment, and increased osteoclastic activity. Overwhelming predictors of prognosis include albumin, β2-microglobulin, and chromosomal karyotype. With modern, intensive therapy including autologous hematopoietic stem cell transplantation, the median survival is approximately 5 yr. The disease is incurable and eventually relapses; requiring salvage therapy. The development of newer agents such as thalidomide, bortezomib, and lenalidomide—drugs that interfere with several of the complex pathophysiologic steps—has improved the outlook of relapsed disease significantly. Recent studies have characterized the molecular mechanisms by which multiple myeloma cell–host bone-marrow interactions regulate tumour cell growth, survival and migration in the bone-marrow microenvironment. These studies have not only enhanced our understanding of disease pathogenesis, but have also provided the framework for a new treatment model that targets the multiple myeloma cell in its bone-marrow microenvironment to overcome drug resistance and improve patient outcome. Current studies are directed at exploring the use of these novel agents earlier in the course of therapy, development of newer targeted therapies, and the use of gene expression profiling to individualize therapy.
Abstract - Bio -05

Insulin

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Insulin remains an important treatment for patients with type 1 and type 2 diabetes. Insulin is given to patients with type 1 diabetes as a form of hormone replacement therapy to replace the loss of endogenous insulin secretion. Intensive insulin treatment with either continuous subcutaneous insulin infusion or basal–bolus therapy reduces diabetic complications, including macrovascular complications. For patients with type 2 diabetes, insulin therapy is given to try and overcome the combination of insulin resistance and beta-cell dysfunction that are the pathological hallmarks of the disease. There are concerns that weight gain and hypoglycaemia, which are common side-effects of intensive insulin therapy, may reduce or negate direct benefits of controlling hyperglycaemia on macrovascular outcomes. The best insulin regimen for patients with type 2 diabetes is not clear, and treatment should aim to minimise weight gain and the occurrence of hypoglycaemia. Resistance to insulin-stimulated glucose uptake is present in the majority of patients with impaired glucose tolerance (IGT) or non-insulin-dependent diabetes mellitus (NIDDM) and in ~25% of nonobese individuals with normal oral glucose tolerance. In these conditions, deterioration of glucose tolerance can only be prevented if the β-cell is able to increase its insulin secretory response and maintain a state of chronic hyperinsulinemia. When this goal cannot be achieved, gross decompensation of glucose homeostasis occurs. The relationship between insulin resistance, plasma insulin level, and glucose intolerance is mediated to a significant degree by changes in ambient plasma free-fatty acid (FFA) concentration. Patients with NIDDM are also resistant to insulin suppression of plasma FFA concentration, but plasma FFA concentrations can be reduced by relatively small increments in insulin concentration. Consequently, elevations of circulating plasma FFA concentration can be prevented if large amounts of insulin can be secreted. If hyperinsulinemia cannot be maintained, plasma FFA concentration will not be suppressed normally, and the resulting increase in plasma FFA concentration will lead to increased hepatic glucose production. Because these events take place in individuals who are quite resistant to insulin-stimulated glucose uptake, it is apparent that even small increases in hepatic glucose production are likely to lead to significant fasting hyperglycemia under these conditions.
Abstract - Bio -06

Alzheimer’s Disease

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Alzheimer’s disease is one of the most common causes of mental deterioration in elderly people, accounting for around 50%-60% of the overall cases of dementia among persons over 65 years of age. The past two decades have witnessed a considerable research effort directed towards discovering the cause of Alzheimer’s disease with the ultimate hope of developing safe and effective pharmacological treatments. Alzheimer's disease is characterized by a widespread functional disturbance of the human brain. Fibrillar amyloid proteins are deposited inside neurons as neurofibrillary tanglesand extracellularly as amyloid plaque cores and in blood vessels. The major protein subunit (A4) of the amyloid fibril of tangles, plaques and blood vessel deposits is an insoluble, highly aggregating small polypeptide of relative molecular mass 4,500. The same polypeptide is also deposited in the brains of aged individuals with trisomy 21 (Down's syndrome).Studies have resulted in the discovery of an association between a decline in learning and memory, and a deficit in excitatory amino acid (EAA) neurotransmission, together with important roles for the cholinergic system in attentional processing and as a modulator of EAA neurotransmission. Accordingly, although there is presently no “cure” for Alzheimer’s disease, a large number of potential therapeutic interventions have emerged that are designed to correct loss of presynaptic cholinergic function. A few of these compounds have confirmed efficacy in delaying the deterioration of symptoms of Alzheimer’s disease, a valuable treatment target considering the progressive nature of the disease. Indeed, three compounds have received European approval for the treatment of the cognitive symptoms of Alzheimer’s disease, first tacrine and more recently, donepezil and rivastigmine, all of which are cholinesterase inhibitors.
Abstract – Bio- 07

Single cell Protein

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Single-cell protein (SCP) typically refers to sources of mixed protein extracted from pure or mixed cultures of algae, yeasts, fungi or bacteria (grown on agricultural wastes) used as a substitute for protein-rich foods, in human and animal feeds. Single-cell proteins develop when microbes ferment waste materials (including wood, straw, cannery, and food-processing wastes, residues from alcohol production, hydrocarbons, or human and animal excreta). The problem with extracting single-cell proteins from the wastes is the dilution and cost. They are found in very low concentrations, usually less than 5%. Engineers have developed ways to increase the concentrations including centrifugation, flotation, precipitation, coagulation, and filtration, or the use of semi-permeable membranes. The single-cell protein must be dehydrated to approximately 10% moisture content and/or acidified to aid in storage and prevent spoilage. The methods to increase the concentrations to adequate levels and the de-watering process require equipment that is expensive and not always suitable for small-scale operations. It is economically prudent to feed the product locally and soon after it is produced. Microorganisms have a high rate of multiplication and, hence, rapid succession of generations (algae: 2–6 hours, yeast: 1–3 hours, bacteria: 0.5–2 hours). They can be easily genetically modified for varying the amino acid composition. A very high protein content 43–85% in the dry mass. They can utilize a broad spectrum of raw materials as carbon sources, which include even waste products. Thus, they help in the removal of pollutants also. Strains with high yield and good composition can be selected or produce relatively easily. Microbial biomass production occurs in continuous cultures and the quality is consistent, since the growth is independent of seasonal and climatic variations. Land requirements is low and is ecologically beneficial. A high solar-energy-conversion efficiency per unit area. Solar energy conversion efficiency can be maximized and yield can be enhanced by easy regulation of physical and nutritional factors. Algal culture can be done in space that is normally unused and so there is no need to compete for land.
Abstract - Bio -08

Jaundice

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Jaundice  also known as  Icterus, is a yellowish  pigmentation of the skin,sclera and other mucous membrane .Jaundice itself is not a disease, but  a sign of one of many possible underlying pathological processes that occur at some point along the normal physiological pathway of the metabolism of bilirubin in blood. Normal concentration of bilirubin in blood plasma is normally 1.2 mg/dl, when increase of 2.5 mg/dl results in hyper bilirubinemia.Jaundice can be categorised as prehepatic, hepatic, or posthepatic, and this provides a useful framework for identifying the underlying cause. Neonatal jaundice is the serious condition because that leads to the complication like kernicterus. inadukts, jaundice is associated with severe chronic liver and biliary disease which alters the normal metabolism of bilirubin.Therefore, adequate diagnosis and serological examination to be investigated and that paves the way for proper treatment of jaundice.
Abstract - Bio -09

DNA Finger Printing

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DNA fingerprinting, also called DNA typing, in genetics, method of isolating and making images of sequences of DNA (deoxyribonucleic acid). The technique was developed in 1984 by the British geneticist Alec Jeffreys, after he noticed the existence of certain sequences of DNA (called minisatellites) that do not contribute to the function of a gene but are repeated within the gene and in other genes of a DNA sample. Jeffreys also determined that each organism has a unique pattern of these minisatellites, the only exception being multiple individuals from a single zygote (e.g., identical twins). The procedure for creating a DNA fingerprint consists of first obtaining a sample of cells containing DNA (e.g., from skin, blood, or hair), extracting the DNA, and purifying it. The DNA is then cut at specific points along the strand with substances called restriction enzymes. This produces fragments of varying lengths that are sorted by placing them on a gel and then subjecting the gel to an electric current (electrophoresis): the shorter the fragment the more quickly it will move toward the positive pole (anode). The sorted, double-stranded DNA fragments are then subjected to a blotting technique in which they are split into single strands and transferred to a nylon sheet. The fragments undergo autoradiography in which they are exposed to DNA probes—pieces of synthetic DNA that have been made radioactive and that bind to the minisatellites. A piece of X-ray film is then exposed to the fragments, and a dark mark is produced at any point where a radioactive probe has become attached. The resultant pattern of these marks can then be analyzed. An early use of DNA fingerprinting was in legal disputes, notably to help solve crimes and determine paternity. The technique was challenged, however, over concerns about sample contamination, faulty preparation procedures, and erroneous interpretation of the results. Efforts were made to improve its reliability, and today the technique has been refined through the use of more-specific and more-sensitive probes and better blotting membranes.
Abstract - Bio -10

Sickle Cell Anemia

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Sickle-cell disease is one of the most common severe monogenic disorders in the world. Haemoglobin polymerisation, leading to erythrocyte rigidity and vaso-occlusion, is central to the pathophysiology of this disease, although the importance of chronic anaemia, haemolysis, and vasculopathy has been established. Clinical management is basic and few treatments have a robust evidence base. One of the main problems of sickle-cell disease in children is the development of cerebrovascular disease and cognitive impairment, and the role of blood transfusion and hydroxycarbamide for prevention of these complications is starting to be understood. Recurrent episodes of vaso-occlusion and inflammation result in progressive damage to most organs, including the brain, kidneys, lungs, bones, and cardiovascular system, which becomes apparent with increasing age. Most people with sickle-cell disease live in Africa, where little is known about this disease; however, we do know that the disorder follows a more severe clinical course in Africa than for the rest of the world and that infectious diseases have a role in causing this increased severity of sickle-cell disease. More work is needed to develop effective treatments that specifically target pathophysiological changes and clinical complications of sickle-cell disease.
Abstract - Bio -11

Electron Transport Chain

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Electron transport chains are used for extracting energy via redox reactions from sunlight in photosynthesis or, such as in the case of the oxidation of sugars, cellular respiration. The electron transport chain is a process in which the NADH and [FADH₂] produced during glycolysis, β-oxidation, and other catabolic processes are oxidized thus releasing energy in the form of ATP. The mechanism by which ATP is formed in the ETC is called chemiosmotic phosphorylation. Cytochromes, ubiquinone, iron sulphur proteins are involved in electron transport chain. Uncouplers inhibit electron transport chain. They uncouple electron transport chain and oxidative phosphorylation. Ionophores inhibit electron transport chain and formation of ATP is inhibited.
DEPARTMENT OF MICROBIOLOGY

ORAL PRESENTATION

ABSTRACT -Micro -01

BIOFILMS ON TEETH

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Periodontitis and caries are infectious diseases of the oral cavity in which oral biofilms play a causative role. Moreover, oral biofilms are widely studied as model systems for bacterial adhesion, biofilm development, and biofilm resistance to antibiotics, due to their widespread presence and accessibility. Despite descriptions of initial plaque formation on the tooth surface, studies on mature plaque and plaque structure below the gum are limited to landmark studies from the 1970s, without appreciating the breadth of microbial diversity in the plaque. The data showed convincingly the dominance of Actinomyces sp., Tannerella forsythia, Fusobacterium nucleatum, Spirochaetes, and Synergistetes in subgingival plaque. The latter proved to be new with a possibly important role in host-pathogen interaction due to its localization in close proximity to immune cells. The present study identified for the first time in vivo that Lactobacillus sp. are the central cells of bacterial aggregates in subgingival plaque, and that Streptococcus sp. and the yeast Candida albicans form corncob structures in supragingival plaque. Finally, periodontal pathogens colonize already formed biofilms and form microcolonies therein. These in vivo observations on oral biofilms provide a clear vision on biofilm architecture and the spatial distribution of predominant species. New technologies have provided novel insights into how dental plaque functions as a biofilm. Confocal microscopy has confirmed that plaque has an open architecture similar to other biofilms, with channels and voids. Bacteria communicate via small diffusible signalling molecules (e.g. competence-stimulating peptide, CSP; autoinducer 2); CSP induces both genetic competence and acid tolerance in recipient sessile cells. Thus, rates of gene transfer increase in biofilm communities, and this is one of several mechanisms that contribute to the increased antimicrobial resistance exhibited by bacteria in biofilms. Oral bacteria in plaque do not exist as independent entities but function as a co-ordinated, spatially organized and fully metabolically integrated microbial community, the properties of which are greater than the sum of the component species. A greater understanding of the significance of dental plaque as a mixed culture biofilm will lead to novel control strategies.
ABSTRACT -Micro -02

STREPTOCOCCUS MUTANS AND DENTAL CARRIES

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Streptococcus mutans is the leading cause of dental caries (tooth decay) worldwide and is considered to be the most cariogenic of all of the oral streptococci. S. mutans metabolizes a wide variety of carbohydrates via nonoxidative pathways, and all of these pathways have been identified, along with the associated transport systems whose genes account for almost 15% of the genome. Virulence genes associated with extracellular adherent glucan production, adhesins, acid tolerance, proteases, and putative hemolysins have been identified. Plaque samples from caries-active subjects showed a higher incidence of S. mutans than plaque samples from caries-free subjects. This was especially evident in approximal incisor plaque. S. mutans serotype d was almost exclusively present in approximal plaque obtained from caries-active subjects. Tooth surfaces infected with S. mutans still harbored this microorganism 10 months later, while uninfected tooth surfaces remained free of S. mutans. Caries development predominantly occurs on those tooth surfaces which harbor relatively high percentages of S. mutans (> 5%). It is unlikely that serum or saliva antibodies against S. mutans play a major role in the protection against dental caries in these caries-free subjects since subjects with the greatest number of decayed surfaces showed the highest antibody titre as measured by haemagglutination or by the enzyme-linked immuno sorbent assay (ELISA). Although it is one of the most common ailments on the planet, dental caries, more commonly known as cavities, remains a poorly understood disease. Caries are caused by a complex interplay of factors, especially patient diet and the presence of the bacteria Streptococcus mutans on the teeth. In spite of the fact that poor dental health has been linked to multiple full-body conditions and diseases, such as Multiple Sclerosis and Heart Disease, and that eighty percent of all American adolescents will be diagnosed with caries, there are still few successful preventative treatments. Rampant caries diseases are especially common among lower-income populations, such as the devastating pediatric disease Early Childhood Caries, which has reported rates of as high as ninety percent in some subpopulations. Recent research into the intricate microbial ecology of the mouth and the other risk factors that may play a role in caries formation has provided insight into new treatment and prevention possibilities for this extremely common infectious disease.
ABSTRACT -Micro -03

PRIONS

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A prion in the Scrapie form (PrPSc) is an infectious agent composed of protein in a misfolded form. This is the central idea of the Prion Hypothesis, which remains debated. This would be in contrast to all other known infectious agents (virus/bacteria/fungus/parasite)—which must contain nucleic acids (either DNA, RNA, or both). The word prion, coined in 1982 by Stanley B. Prusiner, is derived from the words protein and infectious. Prions are responsible for the transmissible spongiform encephalopathies in a variety of mammals, including bovine spongiform encephalopathy (BSE, also known as "mad cow disease") in cattle. In humans, prions cause Creutzfeldt-Jakob Disease (CJD), variant Creutzfeldt-Jakob Disease (vCJD), Gerstmann–Sträussler–Scheinker syndrome, Fatal Familial Insomnia and kuru.

Prions are not considered living organisms but may propagate by transmitting a misfolded protein state. If a prion enters a healthy organism, it induces existing, properly folded proteins to convert into the disease-associated, prion form; the prion acts as a template to guide the misfolding of more proteins into prion form. These newly formed prions can then go on to convert more proteins themselves; this triggers a chain reaction that produces large amounts of the prion form. All known prions induce the formation of an amyloid fold, in which the protein polymerises into an aggregate consisting of tightly packed beta sheets. Amyloid aggregates are fibrils, growing at their ends, and replicating when breakage causes two growing ends to become four growing ends. The incubation period of prion diseases is determined by the exponential growth rate associated with prion replication, which is a balance between the linear growth and the breakage of aggregates. This altered structure is extremely stable and accumulates in infected tissue, causing tissue damage and cell death. This structural stability means that prions are resistant to denaturation by chemical and physical agents, making disposal and containment of these particles difficult. Prions come in different strains, each with a slightly different structure, and, most of the time, strains breed true. Prion replication is nevertheless subject to occasional epimutation and then natural selection just like other forms of replication.
ABSTRACT - Micro -04

YEAST IN APICAL PERIODONTITIS

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Yeast are eukaryotic microorganisms with 1,500 species currently described (estimated to be 1% of all fungal species). Yeasts are unicellular, although some species with yeast forms may become multicellular through the formation of strings of connected budding cells known as pseudohyphae, or false hyphae, as seen in most mould. Microbiological reports of apical periodontitis have revealed that yeasts can be isolated from approximately 5-20% of infected root canals. They occur either in pure cultures or together with bacteria. Almost all isolated yeasts belong to the genus Candida, and the predominant species is C. albicans. Phenotypic and genotypic profiles of C. albicans isolates show heterogeneity comparable with those of isolates from other oral sites. C. albicans expresses several virulence factors that are capable of infecting the dentin-pulp complex, including dentinal tubules. This causes, consequentially, an inflammatory response around the root apex, which suggests a pathogenic role for this organism in apical periodontitis. Yeasts are particularly associated with persistent root canal infections that do not respond favorably to conservative root canal therapy. This may be due to the resistance of all oral Candida species against a commonly used topical medicament, calcium hydroxide. However, other antimicrobial agents may offer alternative therapeutic approaches and improve the treatment of these persistent cases of apical periodontitis.

Studies indicate differences in the composition of the flora in retreatment cases compared with primary necrotic cases. The microbiota associated with persistent secondary infections is usually composed of a single species or at least by a low number of species. Gram-positive bacteria are predominant and Enterococcus faecalis is frequently isolated from retreatment cases. Previously treated teeth with persistent periapical lesions might be preserved with nonsurgical retreatment or endodontic surgery. Evidence-based dentistry recommends selection of alternate treatment options on the basis of the best available evidence. The outcome of retreatment of teeth with apical periodontitis is inferior to the success rate of primary treatment. Systematic review of nonsurgical endodontic retreatment results demonstrates success rate from 70.9% to 83.0% depending on the duration of observation period.
ABSTRACT-Micro-05

LEPTOSPIRAL INFECTIONS IN INDIA

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Leptospirosis is an acute anthropo-zoonotic infection of worldwide significance caused by spirochaete Leptospira interrogans which has 23 serogroups and >200 serovars. Various factors influencing the animal activity, suitability of the environment for the survival of the organism and behavioral and occupational habits of human beings can be the determinants of incidence and prevalence of the disease. The disease was considered inconsequential till recently, but it is emerging as an important public health problem during the last decade or so due to sudden upsurge in the number of reported cases and outbreaks. Since isolation rate of the microorganism from clinical specimens is low due to prior indiscriminate use of antibiotics, serological techniques remain the cornerstone of diagnosis. Leptospirosis is a zoonosis of ubiquitous distribution. The term is used for diseases caused by all leptospira regardless of serotype. Primarily a disease of wild and domestic mammals, man is infected through contact with an infected animal either directly or indirectly by water or soil contaminated with the urine of an infected animal. The spectrum of disease ranges from subclinical infection to a severe syndrome of multiorgan dysfunction characterized by headache, fever, myalgia, jaundice, hepatomegaly and convulsions. Leptospirosis was first reported from the Andaman Islands in 1929, and has since affected all parts of India. Although national incidence data are not available, leptospirosis has been recognized as a major health problem. Natural disasters and poor sanitary conditions have contributed to the multiple epidemics reported and several outbreaks of the disease have been reported in recent years. Although studies have highlighted the epidemicity and prevalence of leptospirosis in India, reports of human leptospirosis from northern India are few. The diagnosis of this disease in man and animals is investigated by direct and indirect laboratory methods. Direct methods includes immunofluorescence staining, immunoperoxidase staining, silver staining and various methods of Polymerase Chain Reaction; while the indirect methods includes various types of ELISA tests, the spot agglutination test or methods reliably identifying the infecting serovars, such as the microscopic agglutination test. Human leptospirosis can be controlled by reducing its prevalence in wild and domestic animals. Leptospirosis in domestic animals can be controlled through vaccination with inactivated whole cells. Sanitation and control of rodents are also important for prevention and control. The reported prevalence values of animal infection across the world are between 2 and 46% depending on the animal species.
ABSTRACT -Micro -06

ORAL CANCER DETECTION BY SALIVARY TEST

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Oral cancer refers to all malignancies that arise in the oral cavity, lips and pharynx, with 90% of all oral cancers being oral squamous cell carcinoma. Despite the recent treatment advances, oral cancer is reported as having one of the highest mortality ratios amongst other malignancies and this can much be attributed to the late diagnosis of the disease. Saliva has long been tested as a valuable tool for drug monitoring and the diagnosis systemic diseases among which oral cancer. The new emerging technologies in molecular biology have enabled the discovery of new molecular markers (DNA, RNA and protein markers) for oral cancer diagnosis and surveillance which are discussed in the current review. MicroRNAs (miRNAs) in human saliva have recently become an emerging field in saliva research for diagnostics applications and its potential role in biological implications. miRNAs are short noncoding RNA molecules that play important roles in regulating a variety of cellular processes. Dysregulation of miRNAs are known to be associated with many diseases. miRNAs were found present in the saliva of OSCC patients and could serve as potential biomarkers for oral cancer detection. Understanding the biological function of miRNAs in association with diseases is important towards utilizing miRNAs as diagnostic markers. There are currently a variety of profiling methods available for detecting miRNA expression levels. In this chapter, we overview the Applied Biosystem Stem-loop RT based Taqman MicroRNA Assay for salivary miRNA profiling. Using this highly sensitive and specific assay, miRNAs in saliva are profiled with only a few nanograms of starting RNA. This method is also applicable for studying biomarkers in other body fluids or clinical samples that contain small amounts of RNA. With monitoring of biomarker levels determined by the saliva biomarker test, cancer development in patients can be detected far earlier than previously possible. On average, one person in the United States dies every hour from oral cancer; but it’s not because the cancer is difficult to discover or diagnose. It’s because the cancer is often detected late in its development.
ABSTRACT -Micro -07

BIOTERRORISM

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Bioterrorism is terrorism involving the intentional release or dissemination of biological agents. These agents are bacteria, viruses, or toxins, and may be in a naturally occurring or a human-modified form. For the use of this method in warfare, see biological warfare. Bioterrorism attack is the deliberate release of viruses, bacteria, toxins or other harmful agents used to cause illness or death in people, animals, or plants. These agents are typically found in nature, but it is possible that they could be mutated or altered to increase their ability to cause disease, make them resistant to current medicines, or to increase their ability to be spread into the environment. Biological agents can be spread through the air, water, or in food. Terrorists tend to use biological agents because they are extremely difficult to detect and do not cause illness for several hours to several days. Some bioterrorism agents, like the smallpox virus, can be spread from person to person and some, like anthrax, cannot. Bioterrorism is an attractive weapon because biological agents are relatively easy and inexpensive to obtain, can be easily disseminated, and can cause widespread fear and panic beyond the actual physical damage they can cause. Military leaders, however, have learned that, as a military asset, bioterrorism has some important limitations; it is difficult to employ a bioweapon in a way that only the enemy is affected and not friendly forces. A biological weapon is useful to terrorists mainly as a method of creating mass panic and disruption to a state or a country. While any germ, bacteria, or virus could potentially be utilized by terrorist, there are a number of biological agents that have been recognized as being more likely to be utilized. The reason for these agents being of concern is based on their availability to terrorists and the ease by which these agents can be disseminated. The U.S. Centers for Disease Control and Prevention (CDC) has developed a classification system for biological terror agents. The classification is based on the likelihood of the agent being used and the risk posed by each agent. There are a number of bacteria and bacterial toxins that could potentially be used to infect the food supply. It is more important for the general public to understand the risk of bioterrorism and the appropriate response to a terrorist attack.
ABSTRACT -Micro -08

HANTA VIRUS - THE FOUR CORNERS DISEASE

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Hantaviruses are single-stranded, enveloped, negative sense RNA viruses in the Bunyaviridae family. Humans may become infected with hantaviruses through contact with rodent urine, saliva, or feces. Some strains of hantaviruses cause potentially fatal diseases in humans, such as Hantavirus hemorrhagic fever with renal syndrome (HFRS) and hantavirus pulmonary syndrome (HPS), while others have not been associated with known human disease. Human infections of hantaviruses have almost entirely been linked to human contact with rodent excrement, but recent human-to-human transmission has been reported with the Andes virus in South America. The name hantavirus is derived from the Hantan River area in South Korea, for which Hantaan virus is named. It was isolated in the late 1970s by Karl M. Johnson and Ho-Wang Lee. In 1993, an outbreak of Hantavirus pulmonary syndrome occurred in the Four Corners region in the southwestern United States. It causes two major clinical syndromes: Haemorrhagic fever with renal syndrome and Hanta virus pulmonary syndrome. Hemorrhagic fever with renal syndrome (HFRS) is a group of clinically similar illnesses caused by species of hantaviruses from the family Bunyaviridae. It is also known as Korean hemorrhagic fever, epidemic hemorrhagic fever, and nephropathis epidemica. Prodromal symptoms include flu-like symptoms such as fever, cough, myalgia, headache, and lethargy. It is characterised by a sudden onset of shortness of breath with rapidly evolving pulmonary oedema that is often fatal despite mechanical ventilation and intervention with potent diuretics. It has a fatality rate of 38%. There is no known antiviral treatment, but natural recovery from the virus is possible with supportive treatment. Patients with suspected hantavirus are usually admitted to the hospital and given oxygen and mechanical ventilation support to help them breathe during the acute pulmonary stage. As the virus can be transmitted by rodent saliva, excreta, and bites, control of rats and mice in areas frequented by humans is key for disease prevention. General prevention can be accomplished by disposing of rodent nests, sealing any cracks and holes in homes where mice or rats could get in, setting up traps, laying down poisons or using natural predators such as cats in the home.
DENTAL PLAQUE AS A BIOFILM

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Dental plaque is the diverse microbial community found on the tooth surface embedded in a matrix of polymers of bacterial and salivary origin. Once a tooth surface is cleaned, a conditioning film of proteins and glycoproteins is adsorbed rapidly to the tooth surface. Plaque formation involves the interaction between early bacterial colonisers and this film (the acquired enamel pellicle). To facilitate colonisation of the tooth surface, some receptors on salivary molecules are only exposed to bacteria once the molecule is adsorbed to a surface. Subsequently, secondary colonisers adhere to the already attached early colonisers (co-aggregation) through specific molecular interactions. These can involve protein-protein or carbohydrate-protein (lectin) interactions, and this process contributes to determining the pattern of bacterial succession. As the biofilm develops, gradients in biologically significant factors develop, and these permit the co-existence of species that would be incompatible with each other in a homogeneous environment. Dental plaque develops naturally, but it is also associated with two of the most prevalent diseases affecting industrialised societies (caries and periodontal diseases). Future strategies to control dental plaque will be targeted to interfering with the formation, structure and pattern of development of this biofilm. Dental plaque is a structurally- and functionally-organised biofilm. Plaque forms in an ordered way and has a diverse microbial composition that, in health, remains relatively stable over time (microbial homeostasis). The predominant species from diseased sites are different from those found in healthy sites, although the putative pathogens can often be detected in low numbers at normal sites. In dental caries, there is a shift toward community dominance by acidogenic and acid-tolerating species such as mutants streptococci and lactobacilli, although other species with relevant traits may be involved. Strategies to control caries could include inhibition of biofilm development (e.g. prevention of attachment of cariogenic bacteria, manipulation of cell signalling mechanisms, delivery of effective antimicrobials, etc.), or enhancement of the host defences. New technologies have provided novel insights into how dental plaque functions as a biofilm. Confocal microscopy has confirmed that plaque has an open architecture similar to other biofilms, with channels and voids. Gradients develop in areas of dense biomass over short distances in key parameters that influence microbial growth and distribution. Bacteria exhibit an altered pattern of gene expression either as a direct result of being on a surface or indirectly as a response to the local environmental heterogeneity within a biofilm.
Staphylococcus aureus is a bacterium that can reside on the skin or can be found in the nose of about one third of healthy individuals. It is generally non-pathogenic except where it gains access to deep tissues such as broken skin, resulting in surgical site or wound infection, the bloodstream leading to bloodstream infection or bacteraemia, and to the lungs causing for example ventilator-associated pneumonia. Early penicillin antibiotics such as flucloxacillin were effective in the treatment of infections caused by Staphylococcus aureus but since the late 1960s many strains have become resistant, but as methicillin was amongst the first anti-staphylococcal agents used, these strains have subsequently been known as MRSA. The prevention and control of MRSA is a challenge in hospitals and in the community throughout the world. MRSA has been prevalent in many Irish hospitals since the early 1970s. Considerable work was undertaken on the epidemiology and clinical importance of MRSA, which has significantly contributed to the world literature. At that time, most MRSA isolates were recovered from burns, surgical wounds and traumatic skin lesions, and invasive infection such as bloodstream infection, deep wound sepsis and osteomyelitis, was rarely seen during that early period. However, the importance of MRSA and its contribution to hospital-acquired infection was not widely acknowledged at the time, despite the efforts of those involved in describing their clinical experiences and in undertaking significant laboratory research. Nonetheless, our knowledge of MRSA, and in particular its contribution to hospital morbidity and mortality, owes much to this seminal body of work and to others.
ABSTRACT -Micro -11

BACTERIAL INVASINS

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To establish and maintain a successful infection, microbial pathogens have evolved a variety of strategies to invade the host, avoid or resist the innate immune response, damage the cells, and multiply in specific and normally sterile regions. Based on their capacity to deal with these critical issues, bacteria can be grouped in different categories. Here we review the so-called invasive bacteria, i.e., bacteria that are able to induce their own phagocytosis into cells that are normally nonphagocytic. The invasion of a host by a pathogen may be aided by the production of bacterial extracellular substances which act against the host by breaking down primary or secondary defenses of the body. Medical microbiologists refer to these substances as invasins. Most invasins are proteins (enzymes) that act locally to damage host cells and/or have the immediate effect of facilitating the growth and spread of the pathogen. The damage to the host as a result of this invasive activity may become part of the pathology of an infectious disease. The extracellular proteins produced by bacteria which promote their invasion are not clearly distinguished from some extracellular protein toxins ("exotoxins") which also damage the host. Invasins usually act at a short range (in the immediate vicinity of bacterial growth) and may not actually kill cells as part of their range of activity; exotoxins are often cytotoxic and may act at remote sites (removed from the site of bacterial growth). Also, exotoxins typically are more specific and more potent in their activity than invasins. Even so, some classic exotoxins (e.g. diphtheria toxin, anthrax toxin) may play some role in colonization or invasion in the early stages of an infection, and some invasins (e.g. staphylococcal leukocidin) have a relatively specific cytopathic effect.
ABSTRACT -Micro -01

RECENT ADVANCES IN DIAGNOSIS OF TUBERCULOSIS
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Despite the discovery of the tubercle bacillus more than a hundred years ago, and all the advances in our knowledge of the disease made since then, tuberculosis still remains one of the major health problems facing mankind, particularly in developing countries. Early diagnosis of tuberculosis and initiating optimal treatment would not only enable a cure of an individual patient but will also curb the transmission of infection and disease to others in the community. Since there is no cure for some multidrug-resistant strains of Mycobacterium tuberculosis, there is concern that they may spread around the world, stressing the need for additional control measures, such as new diagnostics, better drugs for treatment, and a more effective vaccine. Pulmonary TB can be diagnosed by its symptoms, chest radiography, sputum smear microscopy and by cultivation of M. tuberculosis, which is considered as the gold standard. Nonconventional diagnostic approaches proposed include the search for biochemical markers, detection of immunological response and early detection of M. tuberculosis by methods other than colony counting. Advances in the detection of Mtb include new tools like light-emitting diode fluorescence microscopy, nucleic acid amplification of Mtb and drug-resistant strains, and more rapid liquid culture with adjunct drug susceptibility testing. In the detection of latent tuberculosis infection, interferon γ release assays offer improved accuracy over the tuberculin skin test. The biggest advance in recent years has been the development of in vitro T-cell-based interferon-γ release assays (IGRAs) that use antigens more specific to M. tuberculosis than the purified protein derivative used in the TST. Besides high specificity, other potential advantages of IGRAs include logistical convenience, avoidance of poorly reproducible measurements, such as skin induration, need for fewer patient visits and the ability to perform serial testing without inducing the boosting phenomenon. Overall, due to its high specificity, IGRAs may be useful in low-endemic, high-income settings where cross-reactivity due to BCG might adversely impact the utility of TST. For drug resistance, new tools include line-probe assays, bacteriophage-based assays, molecular beacons and microscopic observation drug susceptibility assays. Interferon-γ (interferon-gamma) release assays (IGRAs) are new developments in TB infection testing. IGRAs are based on the ability of the Mycobacterium tuberculosis antigens for early secretory antigen target 6 (ESAT-6) and culture filtrate protein 10 (CFP-10) to stimulate host production of interferon-gamma. Because these antigens are not present in non-tuberculous mycobacteria or in any BCG vaccine variant, these tests can distinguish latent tuberculosis infection (LTBI).
ABSTRACT -Micro -02

CHIKUNGUNYA AND IT'S COMPLIANCES

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Chikungunya (in the Makonde language "that which bends up") virus (CHIKV) is an arthropod-borne virus, of the genus Alphavirus, that is transmitted to humans by virus-carrying Aedes mosquitoes. There have been recent breakouts of CHIKV associated with severe illness. CHIKV infection causes an illness with a similar mode of transmission as dengue fever, with an acute febrile phase lasting two to five days, followed by a longer period of joint pains in the extremities. The pain associated with CHIKV infection of the joints may persist for weeks or months, or in some cases years. Prevention is via mosquito control and preventing bite by infected mosquitoes. There is no specific treatment with medications used to help with symptoms. The incubation period of chikungunya disease ranges from one to twelve days, usually two to three. Its symptoms include a fever up to 40 °C (104 °F), a petechial or maculopapular rash of the trunk and occasionally the limbs, and arthralgia or arthritis affecting multiple joints. Other nonspecific symptoms can include headache, nausea, vomiting, conjunctivitis, slight photophobia and partial loss of taste. Ocular inflammation from Chikungunya may present as iridocyclitis, and have retinal lesions as well. Pedaloedema (swelling of legs) is observed in many patients, the cause of which remains obscure as it is not related to any cardiovascular, renal or hepatic abnormalities. Typically, the fever lasts for two days and then ends abruptly. However, other symptoms—namely joint pain, intense headache, insomnia and an extreme degree of prostration—last for a variable period; usually for about five to seven days. Patients have complained of joint pains for much longer time periods; some as long as two years, depending on their age. Recovery from the disease varies by age. Younger patients recover within 5 to 15 days; middle-aged patients recover in 1 to 2.5 months. Recovery is longer for the elderly. The severity of the disease as well as its duration is less in younger patients and pregnant women. In pregnant women, no untoward effects are noticed after the infection.
ABSTRACT -Micro -03

ORAL MANIFESTATION OF SYSTEMIC DISEASES

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The oral cavity is an important anatomical location with a role in many critical physiologic processes, such as digestion, respiration, and speech. It is also unique for the presence of exposed hard tissue surrounded by mucosa. The mouth is frequently involved in conditions that affect the skin or other multiorgan diseases. In many instances, oral involvement precedes the appearance of other symptoms or lesions at other locations. The oral cavity is the site of much infectious and inflammatory disease which has been associated with systemic diseases such as diabetes, cardiovascular disease and pre-term low births. This article emphasizes on the oral-systemic disease connection which is now a rapidly advancing area of research. The possible systemic diseases which arise from oral microorganisms are hereby focused. Oral manifestations of systemic diseases are potential indicators of an array of conditions. Truly the oral cavity is a mirror that reflects and unravels many of the human body's internal secrets. Some of these manifestations are disease specific and help raise a high degree of suspicion for the alert clinician. Because oral manifestations may accompany many systemic diseases, it is essential that these are appropriately recognized to provide correct diagnosis and referral for treatment and patient care. Multiple entities involving the various areas of the oral cavity like the soft palate, hard palate, tongue, gingiva, oral mucosa, the dentition, periodontium, and the salivary gland tissue have been enlisted. Although this article is not all-inclusive, the authors highlight lesions or conditions that are directly related to or are caused by some of the more common systemic diseases, and hope to provide ample insight for physicians, dentists, and clinicians in to laryngologic practice. There have been a very large number of studies conducted to test the validity of the relationships, for example, between periodontal destruction and prevalence of myocardial infarction and stroke. Further studies on periodontal infections and systemic inflammation as a cause or as an exacerbating factor for the progression of cardiovascular diseases, metastasis from primary tumors in the oral cavity and if there is a link of immunological diseases is needed. Increase in inter-professional collaboration and communication between dental hygienists and other health professionals is the need of the hour. We need to emphasize that the investigation into oral-systemic disease connections is a rapidly advancing area of research, and that the early identification of oral disease may contribute to the early diagnosis and treatment for a number of systemic diseases.
DEPARTMENT OF PATHOLOGY

ORAL PRESENTATION

Abstract - Path – 01

Turmeric – An Innovative Stain

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Turmeric (Curcuma longa) is a rhizomatous herbaceous perennial plant of the ginger family, Zingiberaceae. It is native to tropical Tamilnadu, in southeast India, and needs temperatures between 20 °C and 30 °C. The most important chemical components of turmeric are a group of compounds called curcuminoids, which include curcumin (diferuloylmethane), demethoxycurcumin, and bisdemethoxycurcumin. The best studied compound is curcumin, which constitutes 3.14% (on average) of powdered turmeric. In addition there are other important volatile oils such as turmerone, atlantone, and zingiberene. Some general constituents are sugars, proteins, and resins. Crude ethanolic extract and column chromatographic fractions of the Allepey cultivar of Curcuma longa were used as a stain for tissue sections. Staining was carried out under basic, acidic and neutral media conditions. Inorganic and organic dissolution solvents were used. The stain was used as a counterstain after alum and iron haematoxylins. C. longa stained collagen fibres, cytoplasm, red blood cells and muscle cells yellow. It also stained in a fashion similar to eosin, except for its intense yellow colour. Preliminary phytochemical evaluation of the active column fraction revealed that it contained flavonoids, free anthraquinone and deoxy sugar. A cheap, natural dye can thus be obtained from Curcuma longa. The use of non-allergic, non-toxic and eco-friendly natural dyes has become a matter of significant importance due to the increased environmental awareness in order to avoid some hazardous synthetic dyes. Since Curcuma longa is an innovative stain which is being increasingly used.
Abstract - Path – 02

Acute Leukaemia
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Leukemia or leukaemia is a type of cancer of the blood or bone marrow characterized by an abnormal increase of immature white blood cells called "blasts". Acute leukemia or acute leukaemia is a family of serious medical conditions relating to an original diagnosis of leukemia. Forms of acute leukemia include Acute myeloid leukemia, Acute erythroid leukaemia, Acute lymphoblastic leukemia , T-cell acute lymphoblastic leukemia, Adult T-cell leukemia / lymphoma, Precursor T acute lymphoblastic leukemia / lymphoma. Acute myeloid leukemia (AML), also known as acute myelogenous leukemia or acute nonlymphocytic leukemia (ANLL), is a cancer of the myeloid line of blood cells, characterized by the rapid growth of abnormal white blood cells that accumulate in the bone marrow and interfere with the production of normal blood cells. Acute erythroid leukemia (or "acute Di Guglielmo syndrome") is a rare form of acute myeloid leukemia where the myeloproliferation is of erythroblastic precursors. Acute lymphoblastic leukemia (ALL) is a form of leukemia, or cancer of the white blood cells characterized by excess lymphoblasts. T-cell acute lymphoblastic leukemia can refer to: Precursor T acute lymphoblastic leukemia/lymphoma or Adult T-cell leukemia/lymphoma. Adult T-cell leukemia/lymphoma (ATL) is a rare cancer of the immune system's own T-cells. Precursor T-cell lymphoblastic leukemia is a form of lymphoid leukemia, in which too many T-cell lymphoblasts (immature white blood cells) are found in the blood and bone marrow. It is sometimes additionally classified as a lymphoma, as designated Precursor T-cell lymphoblastic lymphoma. There is no single known cause for any of the different types of leukemia. The few known causes, which are not generally factors within the control of the average person, account for relatively few cases. Leukemia, like other cancers, results from mutations in the DNA. Certain mutations can trigger leukemia by activating oncogenes or deactivating tumor suppressor genes, and thereby disrupting the regulation of cell death, differentiation or division. These mutations may occur spontaneously or as a result of exposure to radiation or carcinogenic substances. Viruses have also been linked to some forms of leukemia. Experiments on mice and other mammals have demonstrated the relevance of retroviruses in leukemia, and human retroviruses have also been identified. The first human retrovirus identified was human T-lymphotropic virus, or HTLV-1, which is known to cause adult T-cell leukemia.
Abstract -Path - 03

Malignant Childhood Neoplasms

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Neoplasm refers to an abnormal mass of tissue as a result of abnormal growth or division of cells and may be benign, pre-malignant (carcinoma in situ) or malignant (cancer). Over the last two decades, cancer in the age group, 0-14 years has become the second leading cause of death by disease, and is considered the leading cause of death among the age group of 5-14. The most common types of malignant neoplasms are as follows. Leukemias, which are cancers of the bone marrow and blood, are the most common childhood cancers. They account for about 31% of all cancers in children. The most common types in children are acute lymphocytic leukemia (ALL) and acute myelogenous leukemia (AML). Leukemia may cause bone and joint pain, fatigue, weakness, pale skin, bleeding or bruising, fever, weight loss, and other symptoms. Brain and central nervous system tumors are the second most common cancers in children, making up about 21% of childhood cancers. There are many types of brain tumors, and the treatment and outlook for each is different. This type of cancer occurs in infants and young children. It is rarely found in children older than 10. This tumor can start anywhere but is usually in the belly (abdomen) and is noticed as swelling. It can also cause bone pain and fever. Wilms tumor starts in one, or rarely, both kidneys. It is most often found in children about 3 to 4 years old, and is uncommon in children older than age 6. It can show up as a swelling or lump in the belly (abdomen). Sometimes the child might have other symptoms, like fever, pain, nausea, or poor appetite. Wilms tumor accounts for about 5% of childhood cancers. Lymphoma - These cancers start in certain cells of the immune system called lymphocytes. They most often grow in lymph nodes and other lymph tissues, like the tonsils or thymus. They can also affect the bone marrow and other organs, and can cause different symptoms depending on where the cancer is. Lymphomas can cause weight loss, fever, sweats, tiredness (fatigue), and lumps (swollen lymph nodes) under the skin in the neck, armpit, or groin. The 2 main types of lymphoma are: Hodgkin lymphoma (sometimes called Hodgkin disease) and non-Hodgkin lymphoma. Both types occur in children and adults. Hodgkin lymphoma accounts for about 4% of childhood cancers. It is more common, though, in 2 age groups: early adulthood (age 15 to 40, usually people in their 20s) and late adulthood (after age 55). Hodgkin lymphoma is rare in children younger than 5 years of age. This type of cancer is very similar in children and adults, including which types of treatment work best. Non-Hodgkin lymphoma makes up about 6% of childhood cancers. It is more likely to occur in younger children than Hodgkin lymphoma, but it is still rare in children younger than 3. The most common types of non-Hodgkin lymphoma in children are different from those in adults. These cancers often grow quickly and require intensive treatment, but they also tend to respond better to treatment than most non-Hodgkin lymphomas in adults.
Abstract -Path - 04

Stem Cells And Disease

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Stem cells are undifferentiated biological cells that can differentiate into specialized cells and can divide (through mitosis) to produce more stem cells. They are found in multicellular organisms. In mammals, there are two broad types of stem cells: embryonic stem cells, which are isolated from the inner cell mass of blastocysts, and adult stem cells, which are found in various tissues. In adult organisms, stem cells and progenitor cells act as a repair system for the body, replenishing adult tissues. In a developing embryo, stem cells can differentiate into all the specialized cells—ectoderm, endoderm and mesoderm (see induced pluripotent stem cells)—but also maintain the normal turnover of regenerative organs, such as blood, skin, or intestinal tissues. Stem cells can also be taken from umbilical cord blood just after birth. Of all stem cell types, autologous harvesting involves the least risk. By definition, autologous cells are obtained from one's own body, just as one may bank his or her own blood for elective surgical procedures. There are three accessible sources of autologous adult stem cells in humans: Bone marrow, which requires extraction by harvesting, that is, drilling into bone (typically the femur or iliac crest), Adipose tissue (lipid cells), which requires extraction by liposuction, and Blood, which requires extraction through apheresis, wherein blood is drawn from the donor (similar to a blood donation), and passed through a machine that extracts the stem cells and returns other portions of the blood to the donor. Medical researchers believe that stem cell therapy has the potential to dramatically change the treatment of human disease. A number of adult stem cell therapies already exist, particularly bone marrow transplants which are used to treat acute leukaemia. In the future medical researchers anticipate being able to use technologies derived from stem cell research to treat a wider variety of diseases including cancer, Parkinson's disease, spinal cord injuries, Amyotrophic lateral sclerosis, multiple sclerosis, and muscle damage, amongst a number of other impaired conditions. Stem cell technology gives hope of effective treatment for a variety of malignant and non-malignant diseases through the rapid developing field that combines the efforts of cell biologists, geneticists and clinicians.
Abstract -Path - 05

Diagnostic Advancements In Pathology – DNA Probe

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It has introduced radical changes in concepts of disease causation and in classification of disease states affecting humans and other organisms. In addition, molecular pathology represents a "new" diagnostic technology with many potentials that have been heretofore untapped. This overview provides a discussion of the use of DNA probes in the study of human diseases. The role of detectable genetic abnormalities in pathogenesis will be considered, as well as their possible impact on nosology and disease classification. Hybridization is becoming an important new adjunct to conventional methods for the diagnosis of infectious diseases, inherited conditions, and neoplasia. Applications of this technology require very small quantities of tissue or body fluids because the DNA probes used in the hybridization assays detect minute amounts of homologous DNA sequence in the test material. Under the proper conditions, these DNA probes are absolutely specific for the pathogen or gene being examined, and hybridization with them usually yields objective answers that require little interpretation. The relatively minor inconveniences currently associated with DNA hybridization are related to the use of radioactivity as a detection signal and the time and labor required to obtain diagnostic data. In the future, technical improvements currently being developed and the preparation of new probes for additional human and microbial genes are likely to create an increasingly larger role for DNA hybridization in diagnostic pathology. Recombinant DNA techniques are contributing to the understanding of the pathogeneses of genetic, neoplastic, and viral diseases, and are used in the diagnosis of certain genetic and viral diseases. Such techniques will have wider application in the future and will play an increasing role in the clinical laboratory. The technology of this field rests upon the cleavage of DNA by certain enzymes, restriction endonucleases, and upon the ability to locate specific sequences of nucleotides in a cleaved DNA sample by using known fragments of DNA ("probes") labeled with radioisotopes or biotin. To produce useful probes, one "clones" multiple copies of the same DNA fragment in bacteria. The use of DNA probes in the clinical laboratory is valuable in antenatal diagnosis, genetic counseling, and post-natal diagnosis of genetic diseases, especially hematologic diseases and inborn errors of metabolism. DNA probes can also be used to detect viral genetic material in clinical specimens. It has introduced radical changes in concepts of disease causation and in classification of disease states affecting humans and other organisms.
Abstract - Path – 06

Organic Mental Disorders Associated With Infection

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An organic mental disorder (OMD), also known as organic brain syndrome or chronic organic brain syndrome, is a form of decreased mental function due to a medical or physical disease, rather than a psychiatric illness. This differs from dementia. While mental or behavioral abnormalities related to the dysfunction can be permanent, treating the disease early may prevent permanent damage in addition to fully restoring mental functions. An organic cause to brain dysfunction is suspected when there is no indication of a clearly defined psychiatric or "inorganic" cause, such as a mood disorder. Organic brain syndrome can be divided into 2 major subgroups: acute (delirium or acute confusional state) and chronic (dementia). A third entity, encephalopathy (subacute organic brain syndrome), denotes a gray zone between delirium and dementia; its early course may fluctuate, but it is often persistent and progressive. Damage to brain functioning could be due not only to organic (physical) injury (a severe blow to the head, stroke, chemical and toxic exposures, organic brain disease, substance abuse, etc.) and also to non-organic means such as severe deprivation, abuse, neglect, and severe psychological trauma. Infections - Any sudden onset (acute) or long-term (chronic) infection, Blood poisoning (septicemia), Brain infection (encephalitis), Meningitis (infection of the lining of the brain and spinal cord), Prion infections such as mad cow disease and Late-stage syphilis. Treatment is based on the etiology. Prognosis of some forms of disease are short term and treatable and some disorders get worse with time.
Abstract -Path – 07

Role Of Intermediate Filaments In Soft Tissue Tumours

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Intermediate filaments (IFs) are cytoskeletal components found in metazoan cells. They are composed of a family of related proteins sharing common structural and sequence features. Intermediate filaments have an average diameter of 10 nanometers, which is between that of 7 nm actin (microfilaments), and that of 25 nm microtubules, although they were initially designated 'intermediate' because their average diameter is between those of narrower microfilaments (actin) and wider myosin filaments found in muscle cells. Most types of intermediate filaments are cytoplasmic, but one type, the lamins, are nuclear. IFs are rather deformable proteins that can be stretched several times their initial length. The key to facilitate this large deformation is due to their hierarchical structure, which facilitates a cascaded activation of deformation mechanisms at different levels of strain. Diseases arising from mutation in IF genes are Arrhythmogenic right ventricular cardiomyopathy (ARVC), mutations in the DES gene, Epidermolysis bullosa simplex; K5 or K14 mutation and Laminopathies are a family of diseases caused by mutations in nuclear lamins and include Hutchinson Gilford Progeria Syndrome and various lipodystrophies and cardiomyopathies among others. Intermediate filaments have been used as cell type-specific markers for histologic and cytologic evaluation of normal and pathological tissues. Antibodies that discriminate between different proteins of intermediate filaments can be used to identify tumor cell type and origin. Cytokeratin, vimentin, and desmin are present in epithelial, mesenchymal, and muscle cells, respectively. Intermediate filament antibodies were used in this study to detect cytokeratin (in cells of epithelial origin), desmin (primarily in smooth, skeletal, and cardiac muscle cells), and vimentin (in cells of mesenchymal origin) of intermediate filaments from different animal species have a high degree of amino acid homology. Antibodies to these proteins have been used to evaluate tissues from several animal species although their application in diagnostic cytology has been limited to human pathology.
Abstract -Path – 08

Role Of Growth Factors In Healing Of Wounds

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Wound healing, as a normal biological process in the human body, is achieved through four precisely and highly programmed phases: hemostasis, inflammation, proliferation, and remodeling. For a wound to heal successfully, all four phases must occur in the proper sequence and time frame. Many factors can interfere with one or more phases of this process, thus causing improper or impaired wound healing. This article reviews the recent literature on the most significant factors that affect cutaneous wound healing and the potential cellular and/or molecular mechanisms involved. The factors discussed include oxygenation, infection, age and sex hormones, stress, diabetes, obesity, medications, alcoholism, smoking, and nutrition. A better understanding of the influence of these factors on repair may lead to therapeutics that improve wound healing and resolve impaired wounds. Wound healing is a complex biological process which requires cellular interactions between a variety of cells like fibroblasts, myofibroblasts, smooth muscle cells, endothelial cells, keratinocytes and immune cells. These interactions are mediated by numerous growth factors namely epidermal growth factor (EGF), fibroblast growth factor (FGF), insulin-like growth factor (IGF), keratinocyte growth factor (KGF), platelet-derived growth factor (PDGF), transforming growth factor (TGF) and vascular endothelial growth factor (VEGF). Growth factors are hormone-like molecules that interact with specific cell surface receptors to control the process of tissue repair. Even trace quantities of these growth factors exert a powerful influence in the wound healing process. By the third day tissue macrophages migrate into the wound and serve as the principal cell, controlling and regulating wound healing. These macrophages control wound healing through the production of growth factors such as platelet-derived growth factor (PDGF), transforming growth factor (TGF), interleukin (IL), and tumor necrosis factor (TNF). The phase of wound healing called fibroplasia begins as the number of macrophages and fibroblasts increase in number which enables the process of matrix formation and collagen synthesis. Fibroplasia begins about 5 days after injury and may continue for as long as 2 weeks. Fibroblasts migrate into the wound and replicate in response to mediators released during inflammation. These mediators include C5a, fibronectin, PDGF, fibroblast growth factor (FGF), and TGF. Therefore growth factors play fundamental roles in wound healing process by stimulating chemotaxis and cellular proliferation, by providing signaling among cells of the same and different type, by controlling extracellular matrix formation and angiogenesis, by regulating the process of contraction and by re-establishing tissue integrity.
Abstract - Path – 09

Premalignant Lesions

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Oral carcinogenesis proceeds through a stepwise accumulation of genetic damage over time. Because the oral cavity is easy to examine and risk factors for oral cancer are known, there is great opportunity to improve patient outcomes through diagnosis and treatment of premalignant lesions before the development of invasive oral carcinoma. The technological and therapeutic advances are much needed to improve the poor outcomes associated with oral cancer due to our inability to diagnose and treat this disease at an early, curable stage. Oral squamous cell carcinoma (SCC) is one of the ten most common cancers worldwide. Despite therapeutic advances, survival rates for patients with oral SCC remain at approximately 50% and have not improved over several decades. Persistent failure to diagnose and treat oral cancer at an early stage is a key factor limiting advances in outcome. Improving detection, diagnosis, and treatment of precancerous changes and early asymptomatic cancers is imperative to increase survival and improve functional outcomes for persons at risk to develop oral cancer. Survival rate has not significantly improved over the past several decades and still hovers at about 50-60%. Many oral SCCs develop from premalignant conditions of the oral cavity. A wide array of conditions have been implicated in the development of oral cancer, including leukoplakia, erythroplakia, palatal lesion of reverse cigar smoking, oral lichen planus, oral submucous fibrosis, discoid lupus erythematosus, and hereditary disorders such as dyskeratosis congenital and epidermolysis bullosa. Despite the general accessibility of the oral cavity during physical examination, many malignancies are not diagnosed until late stages of disease. In order to prevent malignant transformation of these precursor lesions, multiple screening and detection techniques have been developed to address this problem. The early detection of cancer is of critical importance because survival rates markedly improve when the oral lesion is identified at an early stage. Erythroplakia (also known as "Erythroplasia" is a flat red patch or lesion in the mouth that cannot be attributed to any other pathology.
Abstract -Path – 01

Teratoma

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The tissues of a teratoma, although normal in themselves, may be quite different from surrounding tissues and may be highly disparate; teratomas have been reported to contain hair, teeth, bone, very rarely, more complex organs or processes such as eyes, torso, and hands, feet, or other limbs. Usually, however, a teratoma will contain no organs but rather one or more tissues normally found in organs such as the brain, thyroid, liver, and lung. Sometimes, the teratoma has within its capsule one or more fluid-filled cysts; when a large cyst occurs, there is a potential for the teratoma to produce a structure within the cyst that resembles a fetus. Because they are encapsulated, teratomas are usually benign, although several forms of malignant teratoma are known and some of these are common forms of teratoma. A mature teratoma is typically benign and found more commonly in women, while an immature teratoma is typically malignant and is more often found in men. Teratomas are thought to be present at birth (congenital), but small ones are often not discovered until much later in life. Teratomas belong to a class of tumors known as nonseminomatous germ cell tumor (N.S.G.C.T.). All tumors of this class are the result of abnormal development of pluripotent cells: germ cells and embryonal cells. Teratomas of embryonic origin are congenital; teratomas of germ cell origin may or may not be congenital (this is not known). The kind of pluripotent cell appears to be unimportant, apart from constraining the location of the teratoma in the body. Teratomas derived from germ cells occur in the testes in men and ovaries in women. Teratomas derived from embryonic cells usually occur on the subject's midline: in the brain, elsewhere in the skull, in the nose, in the tongue, under the tongue, and in the neck (cervical teratoma), mediastinum, retroperitoneum, and attached to the coccyx. However, teratomas may also occur elsewhere: very rarely in solid organs (most notably the heart and liver) and hollow organs (such as the stomach and bladder), and more commonly on the skull sutures. The treatment of choice is complete surgical removal (i.e., complete resection). Teratomas normally are well encapsulated and non-invasive of surrounding tissues, hence they are relatively easy to resect from surrounding tissues. Exceptions include teratomas in the brain, and very large, complex teratomas that have pushed into and become interlaced with adjacent muscles and other structures. Prevention of recurrence does not require en bloc resection of surrounding tissues.
Abstract-Path – 02

Malignant Melanoma

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Malignant melanoma is a neoplasm of melanocytes or a neoplasm of the cells that develop from melanocytes. Although it was once considered uncommon, the annual incidence has increased dramatically over the past few decades. Surgery is the definitive treatment for early-stage melanoma, with medical management generally reserved for adjuvant treatment of advanced melanoma. Melanomas originate from melanocytes, which arise from the neural crest and migrate to the epidermis, uvea, meninges, and ectodermal mucosa. The melanocytes, which reside in the skin and produce a protective melanin, are contained within the basal layer of the epidermis, at the junction of the dermis and epidermis. Melanomas may develop in or near a previously existing precursor lesion or in healthy-appearing skin. A malignant melanoma developing in healthy skin is said to arise de novo, without evidence of a precursor lesion. Many of these melanomas are induced by solar irradiation. Melanoma also may occur in unexposed areas of the skin, including the palms, soles, and perineum. Certain lesions are considered to be precursor lesions of melanoma, including the common acquired nevus, dysplastic nevus, congenital nevus, and cellular blue nevus. A changing spot may be a problem, but not every change is a problem. A mole may appear and then get bigger or become raised but still be only a mole. Most public health information about melanoma stresses the so-called ABCDs: Asymmetry: One half of the mole is different from the other half. Border irregularity: The spot has borders which are not smooth and regular but uneven or notched. Color: The spot has several colors in an irregular pattern or is a very different color than the rest of your moles. Diameter: The spot is larger than the size of a pencil eraser. The main types of melanoma are (1) Superficial spreading melanoma: This type accounts for about 70% of all cases of melanoma. The most common locations are the legs of women and the backs of men, and they occur most commonly between the ages of 30 and 50. These melanomas are often barely raised and have a variety of colors. Such melanomas evolve over one to five years and can be readily caught at an early stage if they are detected and removed. (2) Nodular melanoma: About 20% of melanomas begin as deeper, blue-black to purplish lumps. They may evolve faster and may also be more likely to spread. (3) Lentigo maligna: Unlike other forms of melanoma, lentigo maligna tends to occur on places like the face, which are exposed to the sun constantly rather than intermittently. Lentigo maligna looks like a large, irregularly shaped or colored freckle and develops slowly. It may take many years to evolve into a more dangerous melanoma.
DEPARTMENT OF PHARMACOLOGY

ORAL PRESENTATION

Abstract -pharma -01

PREGNANCY AND DRUGS

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Treatment of common illnesses in early pregnancy is complicated because of the risk of teratogenic effects of drugs on the fetus. The period of greatest risk is between the first and eighth week of pregnancy. Since much of this period occurs before a diagnosis of pregnancy is made, care must be used in treatment of common illnesses in all women susceptible to becoming pregnant. Few, if any, drugs have been tested for teratogenicity in controlled clinical trials. Risk must therefore be based on epidemiological studies, individual case reporting and extrapolation from animal studies. Sufficient information is available on commonly used drugs to establish such risks. It is important that drugs of least known risk but adequate efficacy be used in treating intercurrent illness in the first trimester. Women and health care providers commonly overestimate the teratogenic risk of medications in pregnancy. With regard to nausea and vomiting of pregnancy, this leads to an underutilization of pharmacologic therapy. Pregnancy termination has even been elected in some circumstances when safe and effective alternatives for nausea have not been attempted. The misperception of teratogenic risk is partly the result of the way data on safety are presented. An awareness of this can lead to more balanced patient and physician educational materials. To facilitate rational use of medicinal and nonmedicinal therapy for nausea and vomiting of pregnancy, we have developed an algorithm based on a recent systematic review of safety and efficacy in management of the problem. The hierarchical use of medication is based on strength of evidence of fetal safety. Most women with active epilepsy need treatment with antiepileptic drugs during pregnancy. Antiepileptic drugs are also frequently used for other indications, such as migraine, pain syndromes, and disorders, which are prevalent among women of childbearing age.
STOMATITIS MEDICAMENTOSA

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Abstract -pharma -02

Allergic inflammatory changes in the oral soft tissues associated with the use of drugs or medicaments, usually those taken systemically. Possible manifestations include asthma, skin rashes, urticaria, pruritus, leukopenia, lymphadenopathy, thrombocytopenic purpura, and oral lesions. The causes of stomatitis vary widely, from a mild local irritant to a vitamin deficiency or infection by a possibly dangerous disease-producing organism. Inflammation may arise from actual injury to the inside of the mouth, as from cheek-biting, jagged teeth, tartar accumulations, and badly fitting dentures. Irritating substances, including alcohol, and tobacco, may also cause stomatitis. Other causes are infectious bacteria, such as streptococci and gonococci or those causing necrotizing ulcerative stomatitis, diphtheria, and tuberculosis; the fungus causing thrush; or the viruses causing herpes simplex and measles. Extreme vitamin deficiencies can result in mouth inflammation, as can certain blood disorders. Poisoning with heavy metals, such as lead or mercury, can also cause stomatitis. There is generally swelling and redness of the tissues of the mouth, which may become quite sore, particularly during eating. The mouth may have an unpleasant odor. In some types of stomatitis the mouth becomes dry, but in others there is excessive salivation. Ulcerations may appear, and, in extreme cases, gangrene (gangrenous stomatitis). Other forms of stomatitis may occasionally cause more severe symptoms, including chills, fever, and headache. Sometimes bleeding or white patches in the mouth can be seen. In thrush, the symptoms themselves may be slight (white spots in the mouth resembling milk curds) but the disease may give rise to serious infections elsewhere in the body. In some cases, stomatitis causes inflammation of the parotid glands. Stomatitis resulting from certain diseases presents special identifying symptoms. Syphilitic stomatitis produces painful ulcers in the mouth; in scarlet fever the tongue first has a strawberry color, which then deepens to a raspberry hue; in measles, Koplik's spots appear. The treatment varies according to the cause. When the inflammation is caused by anemia, vitamin deficiency, or any infection of the body, both the underlying disease and the stomatitis are treated.
GLAUCOMA AND ITS MANAGEMENT

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Major drug classes for medical treatment of POAG includes alpha-agonists, beta-blockers, carbonic anhydrase inhibitors, miotic agents, and prostaglandin analogs. Basic science research continues on other possible pharmacologic sites of action, including nitric oxide and cannabinoid pathways, although no topical product has been evaluated in US FDA trials of yet. Medical marijuana is not indicated for glaucoma treatment, as marijuana lowers IOP minimally and its duration of action is very short. In the future, topical derivatives that affect cannabinoid M receptors governing aqueous dynamics may be effective, but this is still under early investigation. The other drug classes mentioned have much more documented duration of action and efficacy without the systemic cannabinoid adverse effects. Furthermore, other options to treat ocular pain from end-stage glaucoma have arisen (eg, trans-scleral or endoscopic cyclophotocoagulation, absolute alcohol [ethanol] or chlorpromazine retrobulbar injections), which directly and more effectively alleviate the problem than in the past when marijuana was used for eye pain from end-stage glaucoma. The goal of treatment is reduction of the pressure before it causes progressive loss of vision. Considering the high average monthly cost of glaucoma medication, along with the possible risks of adverse effects or toxic reactions from drugs, inconvenience of use, and incidence of noncompliance, a strong reason not to treat indiscriminately exists. The current options available to treat open angle glaucoma are limited to methods of lowering IOP which include medical, laser and surgical treatments. The options available in the UK include topically applied beta antagonists, various miotics, a carbonic anhydrase inhibitor, alpha receptor agonists and a prostaglandin analogue. The most extensively tested of all these is the non-selective beta antagonist timolol. Timolol will produce a long term IOP lowering of 4-5mmHg. Argon laser trabeculoplasty is of use as a supplement to medical treatment, and may produce a further IOP reduction with some if not all of the topically applied preparations. It is most effective in eyes with trabecular pigmentation, and the elderly, but only 50% of those initially responding will continue to do so at 5 years. Repeat laser treatment does not usually last and is rarely useful. The treatment is of most use in the elderly, the arthritic, and the amnesiac patient for whom a moderate IOP fall is needed. Fistulising surgery (trabeculectomy) should be considered for all patients when the 'target IOP' is not met with other therapeutic options, and the expected rate of visual loss will affect the patient during their lifetime.
Abstract -pharma -04

DENTAL LOCAL ANAESTHESIA

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The numbing drug is only one part of what's injected. The liquid in the injection also can include, a type of drug called a vasoconstrictor. This drug narrows your blood vessels. This makes the numbness last longer. A chemical that keeps the vasoconstrictor from breaking down. Sodium hydroxide, which helps the numbing drug work. Sodium chloride, which helps the drugs get into your blood. There are two kinds of numbing injections. A block injection numbs an entire region of your mouth, such as one side of your lower jaw. An infiltration injection numbs a smaller area. This is the area near where the injection was given. If you need local anesthesia in order to have your dental treatment done, your dentist will dry part of your mouth with air or cotton. Many dentists then swab the area with a gel to numb the skin. Then, your dentist will slowly inject the local anesthetic. Most people don't feel the needle. Instead, the sting they feel is caused by the anesthetic moving into the tissue. An injection of local anesthesia can last up to several hours.

Local anesthesia is produced by the application or injection of a pharmacologic agent to eliminate pain or sensation in a specific area in the mouth for a short period of time. These agents are commonly used for most dental procedures to ensure patient comfort and safety, based on the professional judgment of the dentist. The ability to anaesthetize, or “numb”, areas of the mouth is of great benefit to patients who would otherwise experience unpleasant sensations during some types of dental treatment. In dentistry, the term “local anaesthesia” refers to a procedure where a “local anaesthetic”, which is a drug which numbs a small area of tissue, is delivered to block sensations from an area of the mouth.
Abstract -pharma -05

ORAL DRUG REACTIONS

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Oral drug reactions have many clinical manifestations and are produced by numerous medications. These reactions may be the result of an allergic reaction to systemically administered drugs or as an indirect effect of the action of the drug on other tissues. Other oral drug reactions may be the result of local or topical medications. These reactions are either a result of an allergic, delayed-type hypersensitivity, or a local primary irritation. The appearance may be nonspecific or it may resemble several distinct clinical entities. The diagnosis of these oral drug reactions is made with a good clinical history and examination, along with a high index of suspicion. Often there are multiple factors involved that complicate the clinical picture. The clinician who is familiar with the types of oral drug reactions caused by medications, the mechanisms by which these reactions occur, and which medications are most likely to cause the reaction will be prepared to make the correct diagnosis and treatment recommendations. Drug-induced side effects are a frequent occurrence. Many commonly available drugs are capable of causing untoward reactions. Such adverse effects may be seen in all age groups and present in many different forms. The oral drug reactions are often nonspecific, but they may mimic specific disease states such as pemphigus, erythema multiforme, or lichen planus. In such cases a high index of suspicion is required to make a correct diagnosis. Drug-induced side effects are a frequent occurrence. Many commonly available drugs are capable of causing untoward reactions. Such adverse effects may be seen in all age groups and present in many different forms. The oral drug reactions are often nonspecific, but they may mimic specific disease states such as pemphigus, erythema multiforme, or lichen planus. In such cases a high index of suspicion is required to make a correct diagnosis. Side effects may be quite characteristic, as is the case with phenytoin and gingival hyperplasia. Oral drug reactions manifest in a variety of patterns. This article will briefly describe the common presentations and mechanisms of oral drug reactions. The drugs most commonly responsible for these reactions will then be discussed, along with specific treatments. Finally, general clinical management and therapies will be addressed.
Abstract -pharma -06

TERATOGENIC EFFECTS OF THALIDOMIDE AND ITS REACTIONS

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More than half of all pregnant women experience morning sickness. With this alarmist statistic, a company based in Germany called Grunenthal developed a drug for expecting mothers in the late 1950s called Thalidomide. Soon after its introduction, over 10,000 babies were born with severe birth defects. These malformities included stunted or missing limbs, deformed eyes and ears, and the absence of a lung. A majority of births were still-born or had infants dying shortly after birth. This devastation created a huge scare and sparked decades of research in order to figure out what caused these severe birth defects and how to prevent this from occurring again. However, after years of study, in 2006, thalidomide was approved by the FDA due to its effective responses against multiple myeloma with its anti-inflammatory and antiangiogenic properties. Thalidomide is playing a significant role in cancer research particularly due to drug interactions seen with enzyme to substrate binding. Thalidomide must not be taken by women who are pregnant or who could become pregnant while taking this medication. Even a single dose of thalidomide taken during pregnancy can cause severe birth defects (physical problems present in the baby at birth) or death of the unborn baby. A program called Thalidomide REMS(formerly known as the System for Thalidomide Education and Prescribing Safety [S.T.E.P.S.®]) has been approved by the Food and Drug Administration (FDA) to make sure that pregnant women do not take thalidomide and that women do not become pregnant while taking thalidomide. All people who are prescribed thalidomide, including men and women who cannot become pregnant, must be registered with Thalidomide REMS®, have a thalidomide prescription from a doctor who is registered with Thalidomide REMS®, and have the prescription filled at a pharmacy that is registered with Thalidomide REMS® in order to receive this medication. This paper focuses on the teratogenic effects of Thalidomide and its reactions.
NEWER ANTI CANCER DRUG DELIVERY METHOD

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Despite advances in diagnostic imaging and drug discovery, primary malignant brain tumors remain fatal. Median survival for patients with the most severe forms is rarely past eight months. The severity of the disease and the lack of substantial improvement in patient survival demand that new approaches be explored in drug delivery to brain tumors. Recently, local delivery of chemotherapy to brain tumors has provided a way to circumvent the blood-brain barrier, allowing delivery of chemotherapy drugs directly to malignant cells in the brain. Two methods of local delivery have been developed: polymeric-controlled release and convection-enhanced delivery. Controlled release utilizes degradable or non-degradable polymers as carriers of chemotherapy; polymer implants or microparticles are implanted locally to introduce a sustained source of drug for periods of days or months. Convection-enhanced delivery employs the bulk flow of drugs dissolved in fluid, which is introduced intracranially using a catheter and pump. The convective fluid flow is capable of delivering drugs great distances within the brain, potentially treating invasive cells at a distance from the catheter infusion site. These two new delivery strategies are capable of delivering both standard chemotherapeutic drugs and new methods of anti-cancer therapy. Taken individually, or used in tandem, they represent a potential revolution in brain cancer treatment.

Each year, approximately 14,000 people are stricken with brain cancer. The disease occurs across both social and economic lines, with incidence rates peaking both in childhood and later in old age. Despite advances in imaging technology — which has led to earlier diagnosis of many tumors — the ability to treat the most aggressive form of brain cancer, glioblastoma multiforme (GBM), has not improved since 1980. The one-year survival rate for invasive central nervous system (CNS) cancer was 57.9 percent in 2002, and survival for GBM in particular is even lower. Over the past two decades, a variety of approaches to enhance the activity of systemically delivered chemotherapy drugs have been tested. Hyperosmolar BBB disruption has been used to enhance BBB transfer of chemotherapy agents, with mixed results. One study, using PET imaging to evaluate a combination of methotrexate and hyperosmolar BBB disruption, indicated a negligible effect in brain tumors, which is echoed by the marginal findings in clinical trials [8,9]. A variety of approaches have been tested for enhancing BBB permeability of systemically administered drugs — by modification with hydrophobic side groups, conjugation to ligands with known BBB carriers, such as transferrin, or encapsulation in liposomes or nanoparticles — but none of these approaches have impacted clinical treatment of glioma.
Abstract -pharma -08

ANTICARIES AGENTS

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Many potential anticaries agents other than fluoride have been identified in a range of laboratory models. This review covers only those agents which have demonstrated significant activity in either animal caries models, in situ models, or human clinical trials, including those measuring plaque acid formation. The agents which so far have been identified can be divided into 5 categories: phosphorus-containing agents, calcium-containing agents, antimicrobials and antibiotics, metals, and miscellaneous agents. Although many potential agents have been identified in various models, very few have been taken forward to full anticaries testing in humans. Chlorhexidine is an exception, and certain agents such as calcium glycerophosphate have been shown to result in greater anticaries activity when added to fluoride. In the future, non-fluoride agents which modify the production of acid in plaque—either antimicrobially, biochemically, or directly—appear to have the most promise for use in topical products and may prove to be effective anticaries systems. Phosphates have shown encouraging activity as diet additives. Although the use of fluorides has been successful in reducing dental caries, the need remains to develop and evaluate new approaches and promising products for caries prevention. Comprehensive caries-prevention protocols should encompass fluoride and other agents affecting the de-/remineralization balance but also antimicrobial strategies. Different from the traditional restorative approach, the current opinion is that caries should be detected and monitored in its earliest stages, when a nonsurgical reversal can still be achieved. This paradigm shift has implications for methods of caries diagnosis, the choice of preventative materials and the design of randomized clinical trials. This article summarizes the highlights of a special conference dedicated to the topic of novel anticaries and remineralizing agents (ICNARA 2), and identifies the current consensus and remaining questions on pivotal issues in this field. The present invention relates to oral compositions containing anti-caries agents distributed in an oral vehicle. In particular, the present invention provides oral compositions containing calcium, arginine and a cariostatic anion distributed in an oral vehicle. A method for preparing oral compositions containing anti-caries agents is also provided by the present invention.
**Abstract -pharma -09**

**PHARMACOTHERAPY OF ROOT CANAL IRRIGATION**

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The success of endodontic treatment depends on the eradication of microbes from the root-canal system and prevention of reinfection. The root canal is shaped with hand and rotary instruments under constant irrigation to remove the inflamed and necrotic tissue, microbes/biofilms, and other debris from the root-canal space. The main goal of instrumentation is to facilitate effective irrigation, disinfection, and filling. Several studies using advanced techniques such as microcomputed tomography (CT) scanning have demonstrated that proportionally large areas of the main root-canal wall remain untouched by the instruments, emphasizing the importance of chemical means of cleaning and disinfecting all areas of the root canal. There is no single irrigating solution that alone sufficiently covers all of the functions required from an irrigant. Optimal irrigation is based on the combined use of 2 or several irrigating solutions, in a specific sequence, to predictably obtain the goals of safe and effective irrigation. Irrigants have traditionally been delivered into the root-canal space using syringes and metal needles of different size and tip design. Clinical experience and research have shown, however, that this classic approach typically results in ineffective irrigation, particularly in peripheral areas such as anastomoses between canals, fins, and the most apical part of the main root canal. Therefore, many of the compounds used for irrigation have been chemically modified and several mechanical devices have been developed to improve the penetration and effectiveness of irrigation. This article summarizes the chemistry, biology, and procedures for safe and efficient irrigation and provides cutting-edge information on the most recent developments.

Irrigation has a central role in endodontic treatment. During and after instrumentation, the irrigants facilitate removal of microorganisms, tissue remnants, and dentin chips from the root canal through a flushing mechanism. Irrigants can also help prevent packing of the hard and soft tissue in the apical root canal and extrusion of infected material into the periapical area. Some irrigating solutions dissolve either organic or inorganic tissue in the root canal. In addition, several irrigating solutions have antimicrobial activity and actively kill bacteria and yeasts when introduced in direct contact with the microorganisms. However, several irrigating solutions also have cytotoxic potential, and they may cause severe pain if they gain access into the periapical tissues. Sodium Hypochlorite EDTA and CA Chlorhexidine Digluconate. Other irrigating solutions used in endodontics have included sterile water, physiologic saline, hydrogen peroxide, urea peroxide, and iodine compounds.
LIPOSOMES AS A DRUG DELIVERY SYSTEM

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The name liposome is derived from two Greek words: 'Lipos' meaning fat and 'Soma' meaning body. A liposome can be formed at a variety of sizes as uni-lamellar or multi-lamellar construction, and its name relates to its structural building blocks, phospholipids, and not to its size. In contrast, the term Nanosome does relate to size and was coined in the early 1990s to denote special liposomes in the low nanometer range; liposome and Nanosome are not synonyms. A liposome does not necessarily have lipophobic contents, such as water, although it usually does. A liposome is a tiny bubble (vesicle), made out of the same material as a cell membrane. Liposomes can be filled with drugs, and used to deliver drugs for cancer and other diseases. There are three types of liposomes MLV (multilamellar vesicles) SUV (Small Unilamellar Vesicles) and LUV (Large Unilamellar Vesicles). These are used to deliver different types of drugs. Liposomes are used as models for artificial cells. Liposomes can also be designed to deliver drugs in other ways. Liposomes that contain low (or high) pH can be constructed such that dissolved aqueous drugs will be charged in solution (i.e., the pH is outside the drug's pI range). As the pH naturally neutralizes within the liposome (protons can pass through some membranes), the drug will also be neutralized, allowing it to freely pass through a membrane. These liposomes work to deliver drug by diffusion rather than by direct cell fusion. Another strategy for liposome drug delivery is to target endocytosis events. Liposomes can be made in a particular size range that makes them viable targets for natural macrophage phagocytosis. These liposomes may be digested while in the macrophage's phagosome, thus releasing its drug. Liposomes can also be decorated with opsonins and ligands to activate endocytosis in other cell types. The use of liposomes for transformation or transfection of DNA into a host cell is known as lipofection. In addition to gene and drug delivery applications, liposomes can be used as carriers for the delivery of dyes to textiles, pesticides to plants, enzymes and nutritional supplements to foods, and cosmetics to the skin. The use of liposomes in nano cosmetology also has many benefits, including improved penetration and diffusion of active ingredients, selective transport of active ingredients, longer release time, greater stability of active, reduction of unwanted side effects, and high biocompatibility. The therapeutic advantages of liposomal drug delivery, such as the ability of long-circulating liposomes to accumulate preferentially at disease sites, including tumors and sites of inflammation, are well recognized. In cases in which a single active has more than one liposome product available, formulation changes leading to differences in pharmacokinetics, toxicity, and clinical efficacy are described.
Abstract -pharma -11

CYCLOSPORIN IN DENTISTRY

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Cyclosporin is an immunosuppressant drug widely used in organ transplantation to prevent rejection. It reduces the activity of the immune system by interfering with the activity and growth of T cells. It was initially isolated from the fungus Tolypocladium inflatum (Beauveria nivea), found in a soil sample obtained in 1969 from Hardangervidda, Norway, by Dr. Hans Peter Frey, a Sandoz biologist. Most peptides are synthesized by ribosomes, but ciclosporin is a cyclic nonribosomal peptide of 11 amino acids and contains a single D-amino acid, which are rarely encountered in nature. Human organ transplantation, a topic of science fiction in past years, is now a reality. Development of various immunosuppressive agents such as cyclosporine (cyclosporine A (or CyA) and tacrolimus (FK 506) has minimized the significance of obtaining a perfect tissue match between donor and recipient. These advances will further increase the clinical application of human-to-human organ transplantation in coming years. To ensure optimal clinical outcomes of organ transplantation, it is critical for medical, dental, and allied healthcare professionals to be familiar with prevention, identification, and/or treatment of the oral complications associated with pre- and post-organ transplant. The oral/facial complications associated with immunosuppressive drugs include Gingival hypertrophy/enlargement, Mouth ulcers, Dysphasia, Salivary gland enlargement, Gingival bleeding, Gingivitis, Xerostomia, Glossitis, Abnormal taste, Mouth odor, Fungal (Candida) infections, Herpes simplex, Esophagitis, Oral moniliasis, Stomatitis, Nonmelonoma skin carcinoma, Lymphadenopathy, Petechia, Increased gingival hemorrhage, Anemia. In addition to drug-induced gingival enlargement, other prevalent yet often unrecognized threats to successful organ transplantation arise from the risk of developing other oral/facial complications, including certain types of de novo malignancies, as well as viral, bacterial, or fungal infections associated with periodontal disease. Therefore, collaborative care is critical to the success of organ transplantation, as reflected in concordant dental care recommendations for patients before and after organ transplantation. Cyclosporine is a potent immunosuppressant used in organ transplant recipients, with side effects including nephrotoxicity, neurotoxicity, hepatoxicity, hypertrichosis, hypertension, and gingival overgrowth.
Abstract -pharma -12

ANAESTHETIC DRUG MANAGEMENT IN ORAL AND MAXILLOFACIAL SURGERY

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The choice of anaesthetic technique will be influenced by the airway evaluation, the skills of the anaesthetist and surgeon, and the available equipment. Conventional i.v. induction of general anaesthesia with muscle relaxation, inhalation route without muscle relaxation, insertion of trans-tracheal catheter under local anaesthesia before induction, awake fibreoptic intubation or tracheostomy under local anaesthesia have been techniques described. Adequate pre-oxygenation, i.v. access, and monitoring are paramount irrespective of the technique used. Trismus secondary to infection might not improve with induction of anaesthesia. In cases where mouth opening is limited and swelling is confined to the oral cavity, there should be a low threshold for the use of awake nasal fibre optic intubation. In the recently published NAP 4 report, failure to consider awake fibreoptic intubation as the primary airway technique led to direct harm in a proportion of reported patients. The avoidance of muscle relaxation when a difficult laryngoscopy is also anticipated, by using either a gaseous induction or a TIVA technique led to a feeling of false reassurance and contributed to adverse outcome in some cases. This, however, should be balanced against those cases where these techniques were used successfully and so, were not included in the project. It should be recognised that all primary techniques may fail and that clear rescue plans are in place before commencing anaesthesia. The location of anaesthetic management also needs consideration. The NAP 4 report recommends the anaesthetic management of any case which may involve surgical tracheostomy as a rescue technique should start in the operating theatre. Once a secure airway is established, anaesthesia can be maintained by either gaseous or i.v. routes. A saline-soaked throat pack is placed in the oropharynx, if possible, to absorb pus, secretions, and blood. Antibiotics are given according to local microbiology guidance. I.v. dexamethasone may also be required. The operating table may need to be rotated to 90° or 180° with regards to the anaesthetic machine. Pain relief can be provided by paracetamol, non-steroidal anti-inflammatory drugs, intra-oral local anaesthesia, and long acting opioids.
Abstract - Pharma -13

DRUG MANAGEMENT FOR PERIODONTAL DISEASE

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Periodontal diseases essentially comprise a group of oral infections whose primary aetiologic factor is dental plaque. Removal of the cause (and its effects) is the primary aim of both non-surgical and surgical treatment regimens, although the infective nature of the diseases has led to the widespread use of antimicrobials as an adjunct to mechanical debridement. A controlled release drug delivery system for placement in the periodontal pocket, gingival sulcus, tooth socket, wound or other cavity within the mouth. The system incorporates drug-containing microparticles in a fluid carrier medium, and is effective in the environment of use for up to 30 days. The role of local drug delivery systems in the management of periodontal diseases. The efficacy of several local delivery devices (i.e., tetracycline fibers, metronidazole and minocycline gels, chlorhexidine chips, and doxycycline polymer) which are either commercially available in the United States or abroad, or are currently under consideration for Food and Drug Administration (FDA) approval are discussed. The drug delivery systems are assessed with regard to their functional characteristics, effectiveness as a monotherapy, as compared to scaling and root planing, and ability to enhance conventional therapy. The subgingival microbiologic composition of diseased periodontal sites was evaluated by darkfield microscopy before and after scaling or local delivery of tetracycline. A standardized sampling and counting method using a crevicular washing technique was developed to determine both numbers and proportions of morphotypes using darkfield microscopy. Tetracycline-loaded hollow fibers established an initial intrasulcular concentration of 200,000 µg/ml, which decreased exponentially to 15 µg/ml in 24 hours. Repetitive intrasulcular placement of these fibers at periodontitis sites produced an incremental reduction in bacterial counts over a 10-day period. Monolithic fibers made of ethylene vinyl acetate loaded with 25% tetracycline hydrochloride provided sustained release for 10 days under in vitro test conditions. Ten patients were treated in a study comparing the effects of these fibers with scaling. Fibers were placed subgingivally to fill pockets to their probable depth and covered with a periodontal dressing which was maintained for 10 days. The average intrasulcular tetracycline concentration measured at the end of the 10-day period was 643 µg/ml. At these sites, total counts, spirochetes, motile rods and nonmotile rods were significantly reduced immediately following treatment. In comparison, scaling produced much smaller alterations of darkfield counts which were not statistically significant.
Abstract -pharma -14

ALLERGIC MUCOSAL REACTION DUE TO SYSTEMATIC DRUG ADMINISTRATION

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An adverse reaction to a drug or a biological agent is an undesirable and usually unanticipated response independent of the intended therapeutic or diagnostic purpose of the medication. Cutaneous drug eruptions are one of the most common types of adverse reaction to drug therapy, with an overall incidence rate of 2–3% in hospitalised patients. Almost any medicine can induce skin reactions, and certain drug classes, such as non-steroidal anti-inflammatory drugs (NSAIDs), antibiotics and antiepileptics, have drug eruption rates approaching 1–5%. Although most drug-related skin eruptions are not serious, some are severe and potentially life-threatening. Serious reactions include angioedema, erythroderma, Stevens–Johnson syndrome and toxic epidermal necrolysis. Drug eruptions can also occur as part of a spectrum of multiorgan involvement, for example in drug-induced systemic lupus erythematosus. As with other types of drug reaction, the pathogenesis of these eruptions may be either immunological or non-immunological. Certain patient groups appear to be predisposed to cutaneous adverse drug reactions (ADRs). There is a high incidence of hypersensitivity reactions in patients with altered immune status, for example due to viral infections (Epstein–Barr virus or HIV). A well-documented example is the increased risk of co-trimoxazole hypersensitivity in HIV patients. As with ADRs in general, altered drug handling due to organ impairment or genetic factors may play a part; for example, slow-acetylator status may predispose to sulfonamide reactions. The role of atopy in predisposing to drug reactions is controversial. It may be important in reactions to iodinated contrast material, but not in those to penicillins or during anaesthesia. The term multiple drug allergy syndrome has been used to describe patients who have a propensity to react against different, chemically unrelated drugs. Most drugs are associated with a spectrum of skin reactions, although some agents seldom cause skin reactions. Some types of skin rash are very rarely drug induced, for example eczema.
DEPARTMENT OF PHARMACOLOGY

POSTER PRESENTATION

Abstract -pharma -01

ROUTES OF DRUG ADMINISTRATION

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The route of administration is the way through which the dosage form is administered into the body for treatment of various diseases and disorders. Various routes of administrations play a marked role in the bioavailability of the active drug in the body. In present review these routes are included with their advantages and limitations. This is an attempt for the initials of field to familiarize with the routes of administrations with their significances. Oral Route is considered to be the most common route of drug delivery to obtain a systemic effect. However, with the recent developments in the field of drug delivery, it has been found that delivery through alternative routes is sometimes more beneficial. This article deals with the salient features, advantages and disadvantages of some of the alternative routes of drug administration- Transdermal, Pulmonary and Parenteral routes. Though the mechanisms of action of drugs delivered by these routes are different, they offer a common advantage- increased Therapeutic Index with simultaneously decreased side effects. Route of Administration shall consist of an alphabetic term which has a maximum length shall be restricted to 60 characters, with the hyphen and virgule being only punctuation permissible. Codes representing these Routes of Administration shall consist of three digits. In addition, since the prefixes intra- and endo- both mean within, the NSC generally felt that most US clinicians prefer the intra- prefix rather than the endo- prefix for route terms, with some exceptions (e.g., endotracheal). Some general terms (e.g., parenteral) should be reserved for instances when a particular route of administration is unknown (e.g., MedWatch forms). When possible E2B terms should take precedence. However, uptake of drugs administered orally may also occur already in the stomach, and as such gastrointestinal (along the gastrointestinal tract) may be a more fitting term for this route of administration. Furthermore, some application locations often classified as enteral, such as sublingual (under the tongue) and sublabial or buccal (between the cheek and gums/gingiva), are taken up in the proximal part of the gastrointestinal tract without reaching the intestines. Strictly enteral administration (directly into the intestines) can be used for systemic administration, as well as local (sometimes termed topical), such as in a contrast enema, whereby contrast media is infused into the intestines for imaging. However, for the purposes of classification based on location of effects, the term enteral is reserved for substances with systemic effects.
Abstract -pharma -02

DRUG TOXICITY-POSTER

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The toxicity of antiretroviral therapy is an increasingly important issue in the management of patients with human immunodeficiency virus (HIV). With sustained major declines in opportunistic complications, HIV infection is increasingly a more chronic disease, and so more drugs are being used in more patients for longer periods. However, permanent and near-perfect adherence to antiretroviral therapy is needed to maximize its long-term benefits. Although adverse reactions to antiretroviral therapy are common, and profoundly affect its clinical efficacy by limiting adherence, many such reactions are poorly studied and analysed, and are under-reported. This article summarizes adverse events associated with antiretroviral therapy, and discusses weaknesses and possible solutions in the study, analysis and reporting of adverse events that could improve antiretroviral drug development.

Preclinical drug development studies currently rely on costly and time-consuming animal testing because existing cell culture models fail to recapitulate complex, organ-level disease processes in humans. We provide the proof of principle for using a biomimetic microdevice that reconstitutes organ-level lung functions to create a human disease model-on-a-chip that mimics pulmonary edema. The microfluidic device, which reconstitutes the alveolar-capillary interface of the human lung, consists of channels lined by closely apposed layers of human pulmonary epithelial and endothelial cells that experience air and fluid flow, as well as cyclic mechanical strain to mimic normal breathing motions. This device was used to reproduce drug toxicity–induced pulmonary edema observed in human cancer patients treated with interleukin-2 (IL-2) at similar doses and over the same time frame. Studies using this on-chip disease model revealed that mechanical forces associated with physiological breathing motions play a crucial role in the development of increased vascular leakage that leads to pulmonary edema, and that circulating immune cells are not required for the development of this disease. These studies also led to identification of potential new therapeutics, including angiopoietin-1 (Ang-1) and a new transient receptor potential vanilloid 4 (TRPV4) ion channel inhibitor (GSK2193874), which might prevent this life-threatening toxicity of IL-2 in the future.
Abstract -pharma -03

ORAL COMPLICATIONS OF CANCER CHEMOTHERAPY

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Aggressive treatment of malignant disease may produce unavoidable toxicities to normal cells. The mucosal lining of the gastrointestinal tract, including the oral mucosa, is a prime target for treatment-related toxicity by virtue of its rapid rate of cell turnover. The oral cavity is highly susceptible to direct and indirect toxic effects of cancer chemotherapy and ionizing radiation. This risk results from multiple factors, including high rates of cellular turnover for the lining mucosa, a diverse and complex microflora, and trauma to oral tissues during normal oral function. Although changes in soft tissue structures within the oral cavity presumably reflect the changes that occur throughout the gastrointestinal tract, this summary focuses on oral complications of antineoplastic drugs and radiation therapies. It is essential that a multidisciplinary approach be used for oral management of the cancer patient before, during, and after cancer treatment. A multidisciplinary approach is warranted because the medical complexity of these patients affects dental treatment planning, prioritization, and timing of dental care. In addition, selected cancer patients (e.g., status posttreatment with high-dose head-and-neck radiation) are often at lifelong risk for serious complications such as osteoradionecrosis of the mandible. Thus, a multidisciplinary oncology team that includes oncologists, oncology nurses, and dental generalists and specialists as well as dental hygienists, social workers, dieticians, and related health professionals can often achieve highly effective preventive and therapeutic outcomes relative to oral complications in these patients. While oral complications may mimic selected systemic disorders, unique oral toxicities emerge in the context of specific oral anatomic structures and their functions. The most common oral complications related to cancer therapies are mucositis, infection, salivary gland dysfunction, taste dysfunction, and pain. These complications can lead to secondary complications such as dehydration, dysgeusia, and malnutrition. In myelosuppressed cancer patients, the oral cavity can also be a source of systemic infection. Radiation of the head and neck can irreversibly injure oral mucosa, vasculature, muscle, and bone, resulting in xerostomia, rampant dental caries, trismus, soft tissue necrosis, and osteonecrosis. Severe oral toxicities can compromise delivery of optimal cancer therapy protocols. For example, dose reduction or treatment schedule modifications may be necessary to allow for resolution of oral lesions.
Brain death is the irreversible end of brain activity (including involuntary activity necessary to sustain life) due to total necrosis of the cerebral neurons following loss of brain oxygenation. It should not be confused with a persistent vegetative state. Patients classified as brain-dead can have their organs surgically removed for organ donation. Even after brain death, the working of the heart might continue at a slow pace, but there will be no respiratory effort. Brain death is used as an indicator of legal death in many jurisdictions, but it is defined inconsistently. Various parts of the brain may keep living when others die, and the term "brain death" has been used to refer to various combinations. For example, although a major medical dictionary says that "brain death" is synonymous with "cerebral death", the US National Library of Medicine Medical Subject Headings (MeSH) system defines brain death as including the brainstem. The distinctions can be important because, for example, in someone with a dead cerebrum but a living brainstem, the heartbeat and ventilation can continue unaided, whereas, in whole-brain death, only life support equipment would keep those functions going. Brain death occurs when a person no longer has any activity in their brain stem and no potential for consciousness, even though a ventilator is keeping their heart beating and oxygen was circulating through their blood. When brain stem function is permanently lost, the person will be confirmed dead. Once brain death occurs it is often possible to still remove organs from the body that can be used in, often life-saving, organ transplants. Deciding whether to carry out a transplant can be a difficult decision for partners and relatives. Hospital staff is aware of these difficulties and will try to ensure the issue is handled sensitively and thoughtfully.
Abstract -physio -02

Parkinson’s disease

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Parkinson's disease (also known as idiopathic or primary Parkinsonism, hypokinetic rigid syndrome/HRS, or paralysis agitans) is a degenerative disorder of the central nervous system. Parkinson's disease is more common in older people, with most cases occurring after the age of 50. The motor symptoms of Parkinson's disease result from the death of dopamine-generating cells in the substantia nigra, a region of the midbrain; the cause of this cell death is unknown. The early symptoms of the disease are movement-related; these include shaking, rigidity, slowness of movement and difficulty with walking and gait. Later, thinking and behavioral problems may arise, with dementia commonly occurring in the advanced stages of the disease, whereas depression is the most common psychiatric symptom. Other symptoms include sensory, sleep and emotional problems. The main motor symptoms are collectively called Parkinsonism, or a "parkinsonian syndrome". Parkinson's also affects the voice, and sense of smell. Modern treatments are effective at managing the early motor symptoms of the disease, mainly through the use of levodopa and dopamine agonists. As the disease progresses and dopaminergic neurons continue to be lost, these drugs eventually become ineffective at treating the symptoms and at the same time produce a complication called dyskinesia, marked by involuntary writhing movements. Diet and some forms of rehabilitation have shown some effectiveness at alleviating symptoms. Surgery and deep brain stimulation have been used to reduce motor symptoms as a last resort in severe cases where drugs are ineffective. Research directions include investigations into new animal models of the disease and of the potential usefulness of gene therapy, stem cell transplants and neuroprotective agents. Medications to treat non-movement-related symptoms of PD, such as sleep disturbances and emotional problems, also exist. The common causes of the disease are environmental factors, and genetics. A number of environmental factors includes: pesticide exposure, head injuries, and living in the country or farming. Implicated agents include insecticides, primarily chlorpyrifos and organ chlorines and pesticides, such as rotenone or paraquat, and herbicides, such as Agent Orange. Heavy metals exposure has been proposed to be a risk factor, through possible accumulation in the substantia nigra. PD traditionally has been considered a non-genetic disorder.
Abstract -physio -03

Physiology of lustation & its applied aspect

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The human species is the only one among mammals in which breastfeeding and weaning are not governed only by instinct. Therefore, breastfeeding and weaning have to be learned. Currently, especially in modern societies, women have few opportunities to learn something about breastfeeding because their traditional sources of learning more experienced women in the family were lost as extended families were replaced by nuclear families. Consequently, women become mothers with little or no ability to breastfeed, which makes them more vulnerable to difficulties during the process. Health professionals play a crucial role in the prevention and management of such difficulties, but to do that, they need specific knowledge, attitudes and skills. Lactation describes the secretion of milk from the mammary glands and the period of time that a mother lactates to feed her young. The process can occur with almost all post-pregnancy female mammals, although it predates mammals. In humans the process of feeding milk is also called breastfeeding or nursing. In most species, milk comes out of the mother's nipples; however, the platypus (a non-placental mammal) releases milk through ducts in its abdomen. In only one species of mammal, the Dayak fruit bat, is milk production a normal male function. Galactopoiesis is the maintenance of milk production. This stage requires prolactin (PRL) and oxytocin. Newborn infants often produce some witch's milk. Galactorrhea is milk production unrelated to nursing, it can occur in males and females of many mammal species as result of hormonal imbalances or unusual physiological stimuli. Lactation is the physiologic completion of the reproductive cycle. Human infants at birth are the most immature and dependent of all mammals, except for marsupials. The marsupial joey is promptly attached to the teat of a mammary gland in an external pouch. The gland changes as the offspring develops, and the joey remains there until able to survive outside the pouch. In humans, throughout pregnancy the breast develops and prepares to take over the role of fully nourishing the infant when the placenta is expelled. There are two stages in the initiation of lactation: secretory differentiation and secretory activation. Pang and Hartman said it best: “Secretory differentiation represents the stage of pregnancy when the mammary epithelial cells differentiate into lactocytes with the capacity to synthesize unique milk constituents such as lactose.” In contrast to most organs, which are fully developed at birth, the mammary gland undergoes most of its morphogenesis postnatally in adolescence and adulthood. Lactation is an integral part of the reproductive cycle of all mammals, including humans. The hormonal control of lactation can be described in relation to the five major stages in the development of the mammary gland: (1) embryogenesis; (2) mammogenesis, or mammary growth; (3) lactogenesis, or initiation of milk secretion; (4) lactation (stage III lactogenesis), or full milk secretion; and (5) involution.
Abstract -physio -04

Tooth for vision

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Osteo-odonto-keratoprosthesis (OOKP) (also known as "tooth in eye" surgery) is a medical procedure to restore vision in the most severe cases of corneal and ocular surface patients. It includes removal of a tooth from the patient or a donor. After removal, a lamina of tissue cut from the tooth is drilled and the hole is fitted with optics. The lamina is grown in the patients' cheek for a period of months and then is implanted upon the eye. An operation to graft the OOKP is undertaken in severe pemphigoid, chemical burns, Stevens–Johnson syndrome, trachoma, Lyell syndrome and multiple corneal graft failure. There is a significant risk of anatomical failure of lamina in the long term, estimated at about 19% in a small study, with the main risks being laminar resorption, particularly in allografts, and glaucoma. The complex surgery is a two-part procedure. First, the tooth and part of the jaw are removed, and a lens is inserted into the tooth using a drill. The tooth and lens are then implanted under the eye socket. After a few months, once the tooth has grown tissues and developed a blood supply, comes the second step: part of the cornea is sliced open and removed and the too is stitched into the eye socket. Since the tooth is the patient’s own tissue, the body does not reject it.
Abstract -physio -05

Morbidity rate of MI patients with diabetes mellitus

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Diabetes mellitus (diabetes), in particular type 2 diabetes, constitutes one of the largest emerging threats to health in the 21st century. It is estimated that by 2030 as many as 360 million people worldwide will be affected. The cause of death in those with diabetes is dominated by coronary heart disease, accompanied by increased rates of stroke and peripheral vascular disease: so called macrovascular complications. At least two-thirds of deaths are attributable to these cardiovascular diseases and their sequelae. A true picture of the extent of macrovascular complications is obscured, however, by inaccurate death certification and diagnostic criteria based on the development of microvascular complications (retinopathy, neuropathy, nephropathy). The Euro Heart Survey demonstrated that if one applied oral glucose tolerance tests to those presenting with all forms of acute coronary syndrome, two-thirds display impaired glucose regulation. Although unstable coronary artery disease is the most common reason for admission to a coronary care unit, the long-term prognosis of patients with this diagnosis is unknown. This is particularly true for patients with diabetes mellitus, who are known to have a high morbidity and mortality after an acute myocardial infarction. Diabetic subjects are more likely to experience a myocardial infarction and have worse outcomes compared to non-diabetic subjects. The underlying pathophysiology of the atherosclerotic process is not significantly different in diabetic subjects, but the prothrombotic and procoagulant state with which diabetes is associated is thought to contribute to the higher incidence of and worse prognosis after myocardial infarction. Difficulties of re-establishing vessel patency by thrombolytic or mechanical means contribute to the high morbidity and mortality. The diffuse nature of arterial disease with accompanying metabolic derangement contribute to impaired compensatory mechanisms, increased infarct size and a disproportionately more substantial impairment of left ventricular function. The newer adjuvant antithrombotic and anticoagulant agents have particular roles in management therefore and careful modulation of glucose metabolism in the acute and follow-up phase of an infarct may favourably influence outcome.
Heart failure

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Heart failure (HF), often called congestive heart failure (CHF) or congestive cardiac failure (CCF) occurs when the heart is unable to provide sufficient pump action to maintain blood flow to meet the needs of the body. Heart failure can cause a number of symptoms including shortness of breath, leg swelling, and exercise intolerance. The condition is diagnosed by patient physical examination and confirmed with echocardiography. Blood tests help to determine the cause. Treatment depends on severity and cause of heart failure. In a chronic patient already in a stable situation, treatment commonly consists of lifestyle measures such as smoking cessation, light exercise, dietary changes, and medications. Sometimes, depending on etiology, it is treated with implanted devices (pacemakers or ventricular assist devices) and occasionally a heart transplant is required. Common causes of heart failure include myocardial infarction and other forms of ischemic heart disease, hypertension, valvular heart disease, and cardiomyopathy. Heart failure is a common, costly, disabling, and potentially deadly condition. In developed countries, around 2% of adults suffer from heart failure, but in those over the age of 65, this increases to 6–10%. Heart failure does not mean the heart has stopped working. Rather, it means that the heart's pumping power is weaker than normal. With heart failure, blood moves through the heart and body at a slower rate, and pressure in the heart increases. As a result, the heart cannot pump enough oxygen and nutrients to meet the body's needs. The chambers of the heart respond by stretching to hold more blood to pump through the body or by becoming more stiff and thickened. This helps to keep the blood moving for a short while, but in time, the heart muscle walls weaken and are unable to pump as strongly. As a result, the kidneys often respond by causing the body to retain fluid (water) and sodium. If fluid builds up in the arms, legs, ankles, feet, lungs or other organs, the body becomes congested, and congestive heart failure is the term used to describe the condition. Heart failure develops over time as the heart's pumping action grows weaker. The condition can affect the right side of the heart only, or it can affect both sides of the heart. Most cases involve both sides of the heart. Right-side heart failure occurs if the heart can't pump enough blood to the lungs to pick up oxygen. Left-side heart failure occurs if the heart can't pump enough oxygen-rich blood to the rest of the body. Right-side heart failure may cause fluid to build up in the feet, ankles, legs, liver, abdomen, and the veins in the neck. Right-side and left-side heart failure also may cause shortness of breath and fatigue (tiredness).
Abstract -physio -07

Circulatory shock

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Circulatory shock, commonly known simply as shock, is a life-threatening medical condition that occurs due to inadequate substrate for aerobic cellular respiration. In the early stages this is generally an inadequate tissue level of oxygen. Circulatory shock is not related to the emotional state of shock. Circulatory shock is a life-threatening medical emergency and one of the most common causes of death for critically ill people. Shock can have a variety of effects, all with similar outcomes, but all relate to a problem with the body's circulatory system. For example, shock may lead to hypoxemia (a lack of oxygen in arterial blood) or cardiac arrest. One of the key dangers of shock is that it progresses by a positive feedback mechanism. Once shock begins, it tends to make itself worse, so immediate treatment of shock is critical to the survival of the sufferer. Depending on its severity, shock can be divided into three separate stages: (1) the nonprogressive or compensated stage, (2) the progressive stage, and (3) the irreversible stage. All types of circulatory shock exhibit one or more of these stages, regardless of their causes. There are several causes of shock. The typical signs of shock are low blood pressure, a rapid heartbeat and signs of poor end-organ perfusion or "decompensation/peripheral shut down" (such as low urine output, confusion or loss of consciousness). The first changes seen in shock is an increased cardiac output followed by a decrease in mixed venous oxygen saturation (SmvO2) as measured in the pulmonary artery via a pulmonary artery catheter. Central venous oxygen saturation (ScvO2) as measured via a central line correlates well with SmvO2 and are easier to acquire. If shock progresses anaerobic metabolism will begin to occur with an increased blood lactic acid as the result. While many laboratory tests are typically performed there is no test that either makes or excludes the diagnosis. A chest X-ray or emergency department ultrasound may be useful to determine volume state. Shock is a common end point of many medical conditions. It has been divided into four main types based on the underlying cause: hypovolemic, distributive, cardiogenic and obstructive.
Abstract -physio -08

Physiology of salivation & xerostomia

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Saliva is produced by the major salivary glands: the parotid, the sublingual, and the submandibular glands. The remaining 10% of saliva comes from numerous minor salivary glands that are scattered throughout the submucosa of the oral cavity. Each salivary gland produces either a serous, a seromucous, or a mucous secretion; the definition of these three types of saliva is based on the glycoprotein content of the gland’s final secretory product. In humans and most other mammals, the parotids produce a serous (i.e., low glycoprotein content) secretion, the sublingual and submandibular glands produce a seromucous secretion, and the minor salivary glands produce a mucous secretion. As the salivary secretion is a reflex response controlled by both parasympathetic and sympathetic secretomotor nerves, it can be influenced by several stimuli. Moreover, patients taking medication which influences either the central nervous system or the peripheral nervous system, or medication which mimic the latter as a side effect, will have an altered salivary composition and salivary volume. Patients suffering from certain systemic diseases may present the same salivary alterations. The circadian rhythm determines both the volume of saliva that will and can be secreted and the salivary electrolyte concentrations. Dietary influences and the patient's age also have an impact on composition and volume of saliva. The latter implies a wide variation in composition both inter- and intra-individually. Sampling must therefore be performed under standardized conditions. The greatest advantage, when compared to blood sample collection, is that saliva is readily accessible and collectible. Consequently, it can be used in clinically difficult situations, such as in children, handicapped and anxious patients, where blood sampling could be a difficult act to perform. Xerostomia is the medical term for the subjective symptom of dryness in the mouth, which may be associated with a change in the composition of saliva or reduced salivary flow (hyposalivation) or have no identifiable cause. This symptom is very common and is often seen as a side effect of many types of medication. It is more common in older people (mostly because this group tend to take several medications) and in persons who breathe through their. Dehydration, radiotherapy involving the salivary glands, and several diseases can cause hyposalivation or a change in saliva consistency and hence a complaint of xerostomia. Sometimes there is no identifiable cause, and there may be a psychogenic reason for the complaint.
Abstract -physio -09

Physiology pathway to space flight

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The Effect of Space Flight on Innate Immunity to Respiratory Viral Infections (Mouse Immunology-2) investigates the effects of microgravity on immune function to fight Respiratory Syncytial Virus (RSV). In microgravity, crewmembers experience changes in immune function. These studies will help scientists determine the biological significance of space flight induced changes in immune responses. Drosophila normally displays a negatively gravitaxic behavior (locomotion against the gravity vector), shown to be altered by mutations in genes with a role in neuronal pathfinding, synaptic structure and peripheral nervous system development. Several of these genes have strong homology and relevance to humans in vestibular and balance related functions. Work from Baker et al is starting to link the perception of gravity through the antennal glomerular tract with the processing and control of locomotor activity by the central nervous system. The genes and brain regions of the CNS that affect gravitaxis have been mapped in Drosophila in ground experiments. Clues for the molecular mechanism that may trigger adaptation to changes in the gravity environment come from ground hypergravity studies that indicate that the cAMP pathway in the brain is essential, and that learning and memory may be implicated in behavioral adaptations. Neuronal genes represent one of the major categories of genes that undergo expression changes during adaptation to both microgravity as well as readaptation to 1g. Experiments in the microgravity environment can therefore reveal not only the molecular pathways required for adapting to altered gravity environments, but the mechanism of neuronal plasticity required during a normal behavior at 1g, which would otherwise not be uncovered. Vertebrates sense gravity and acceleration from inner ear otolith organs containing biomineral crystalline deposits of calcium carbonate called otoconia. A widely considered mechanism by which the nervous system responds to change in amplitude of gravity vector is a change in weight-lending otoconia. In this study, scientists apply electron microscopic techniques to image the otoconia mass of mice subjected to microgravity in the Mouse Immunology II mission and the corresponding ground control mice. Intracranial microcirculatory adaptations might also occur in astronauts, involving an increase in the turnover rate of catecholamines, i.e., norepinephrine (NE) and its precursor, Dopamine (DA). DA is known to inhibit prolactin (PRL) release and to enhance growth hormone (GH) secretion by the pituitary. Therefore, increased brain dopaminergic activity would result into lower circulating PRL concentrations. At the same time, plasma levels of GH and of its effector insulin-like growth factor-1 (IGF-1) would increase during flight.
Abstract -physio -10

Child older to mother: Progeria syndrome

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Progeria (Hutchinson–Gilford progeria syndrome, HGPS, progeria syndrome) is an extremely rare genetic disorder wherein symptoms resembling aspects of aging are manifested at a very early age. Progeria is one of several progeroid syndromes. It is a genetic condition that occurs as a new mutation, and is rarely inherited; as patients usually do not live to reproduce. The earliest symptoms may include a failure to thrive and a localized scleroderma-like skin condition. Limited growth, full-body alopecia, and a distinctive appearance (a small face with a shallow recessed jaw, and a pinched nose) wrinkled skin, atherosclerosis, kidney failure, loss of eyesight, and cardiovascular problems are all characteristics of progeria. Mental development is not adversely affected. The cause of progeria was discovered to be a point mutation in position 1824 of the LMNA gene, in which cytosine is replaced with thymine. Mutations in the LMNA gene cause Hutchinson-Gilford progeria syndrome due to production of an abnormal are lamin A protein. People inherit the disease; only one copy of the LMNA gene is enough to cause the disease because it is an autosomal dominant gene. Treatment focuses on reducing complications (such as cardiovascular disease) with coronary artery bypass surgery or low-dose aspirin. Children may also benefit from a high-energy diet. Growth hormone treatment has been attempted. The use of Morpholinos has also been attempted in order to reduce progerin production. Antisense Morpholino oligonucleotides specifically directed against the mutated exon 11–exon 12 junction in the mutated pre-mRNAs were used. As there is no known cure, few people with progeria exceed 13 years of age. At least 90% of patients die from complications of atherosclerosis, such as heart attack or stroke. Progeria syndrome is very rare; only about 130 individuals have been diagnosed since 1886.
Trigeminal neuralgia

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Neuralgia is pain in the distribution of a nerve or nerves, as in intercostal neuralgia, trigeminal neuralgia, and glossopharyngeal neuralgia. Under the general heading of neuralgia are trigeminal neuralgia (TN), atypical trigeminal neuralgia (ATN), occipital neuralgia, glossopharyngeal neuralgia and postherpetic neuralgia (caused by shingles or herpes). The term neuralgia is also used to refer to pain associated with sciatica and brachial plexopathy. Atypical trigeminal neuralgia (ATN) is a rare form of neuralgia and may also be the most misdiagnosed form. The symptoms can be mistaken for migraines, dental problems such as temporomandibular joint disorder, musculoskeletal issues, and hypochondriasis. ATN can have a wide range of symptoms and the pain can fluctuate in intensity from mild aching to a crushing or burning sensation, and also to the extreme pain experienced with the more common trigeminal neuralgia. The pain can be described as heavy, aching, and burning. Sufferers have a constant migraine-like headache and experience pain in all three trigeminal nerve branches. This includes aching teeth, ear aches, feeling of fullness in sinuses, cheek pain, pain in forehead and temples, jaw pain, pain around eyes, and occasional electric shock-like stabs. Unlike typical neuralgia, this form can also cause pain in the back of the neck. Pain tends to worsen with talking, facial expressions, chewing, and certain sensations such as a cool breeze. Vascular compressions of the trigeminal nerve, infections of the teeth or sinuses, physical trauma, or past viral infections are possible causes of ATN. Glossopharyngeal neuralgia consists of recurring attacks of severe pain in the back of the throat, the area near the tonsils, the back of the tongue, and part of the ear. The pain is due to malfunction of the glossopharyngeal nerve (CN IX), which moves the muscles of the throat and carries information from the throat, tonsils, and tongue to the brain. Glossopharyngeal neuralgia, a rare disorder, usually begins after age 40 and occurs more often in men. Often, its cause is unknown. However, glossopharyngeal neuralgia sometimes results from an abnormally positioned artery that compresses the glossopharyngeal nerve near where it exits the brain stem. Rarely, the cause is a tumor in the brain or neck. Occipital neuralgia, also known as C2 neuralgia, or Arnold's neuralgia, is a medical condition characterized by chronic pain in the upper neck, back of the head and behind the eyes. By understanding the neuroplastic changes following nerve damage, researchers may be able to better understand the mechanism of hyper excitability in the nervous system that is believed to cause neuropathic pain.
Abstract -physio -02

Leukemia

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Leukemia (American English) or leukaemia (British English) is a type of cancer of the blood or bone marrow characterized by an abnormal increase of immature white blood cells called "blasts". Leukemia is a broad term covering a spectrum of diseases. In turn, it is part of the even broader group of diseases affecting the blood, bone marrow, and lymphoid system, which are all known as hematological neoplasms. Leukemia is a treatable disease. Most treatments involve chemotherapy, medical radiation therapy, hormone treatments, or bone marrow transplant. The rate of cure depends on the type of leukemia as well as the age of the patient. Children are more likely to be permanently cured than adults. Even when a complete cure is unlikely, most people with a chronic leukemia and many people with an acute leukemia can be successfully treated for years. Sometimes, leukemia is the effect of another cancer, known as blastic leukemia, which usually involves the same treatment, although it is usually unsuccessful. Leukemia can affect people at any age. In 2000 approximately 256,000 children and adults around the world had developed some form of leukemia, and 209,000 have died from it. About 90% of all leukemias are diagnosed in adults. The word leukemia, which means 'white blood', is derived from the disease's namesake high white blood cell counts that most leukemia patients have before treatment. The high numbers of white blood cells are apparent when a blood sample is viewed under a microscope. Frequently, these extra white blood cells are immature or dysfunctional. The excessive number of cells can also interfere with the level of other cells, causing a harmful imbalance in the blood count. Some leukemia patients do not have high white blood cell counts visible during a regular blood count. This less-common condition is called aleukemia. The bone marrow still contains cancerous white blood cells which disrupt the normal production of blood cells, but they remain in the marrow instead of entering the bloodstream, where they would be visible in a blood test. For an aleukemic patient, the white blood cell counts in the bloodstream can be normal or low. Aleukemia can occur in any of the four major types of leukemia, and is particularly common in hairy cell leukemia. Diagnosis is usually based on repeated complete blood counts and a bone marrow examination following observations of the symptoms, however, in rare cases blood tests may not show if a patient has leukemia, usually this is because the leukemia is in the early stages or has entered remission. A lymph node biopsy can be performed as well in order to diagnose certain types of leukemia in certain situations.
Abstract -physio -03

Dialysis

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Dialysis is a process for removing waste and excess water from the blood, and is used primarily as an artificial replacement for lost kidney function in people with renal failure. Dialysis may be used for those with an acute disturbance in kidney function (acute kidney injury, previously acute renal failure), or progressive but chronically worsening kidney function—a state known as chronic kidney disease stage 5 (previously chronic renal failure or end-stage renal disease). The latter form may develop over months or years, but in contrast to acute kidney injury is not usually reversible, and dialysis is regarded as a "holding measure" until a renal transplant can be performed, or sometimes as the only supportive measure in those for whom a transplant would be inappropriate. The kidneys have important roles in maintaining health. When healthy, the kidneys maintain the body's internal equilibrium of water and minerals (sodium, potassium, chloride, calcium, phosphorus, magnesium, sulfate). The acidic metabolism end-products that the body cannot get rid of via respiration are also excreted through the kidneys. The kidneys also function as a part of the endocrine system, producing erythropoietin and calcitriol. Erythropoietin is involved in the production of red blood cells and calcitriol plays a role in bone formation.[3] Dialysis is an imperfect treatment to replace kidney function because it does not correct the compromised endocrine functions of the kidney. Dialysis treatments replace some of these functions through diffusion (waste removal) and ultrafiltration (fluid removal). There are three primary and two secondary types of dialysis: hemodialysis (primary), peritoneal dialysis (primary), hemofiltration (primary), hemodiafiltration (secondary), and intestinal dialysis (secondary). In hemodialysis, the patient's blood is pumped through the blood compartment of a dialyzer, exposing it to a partially permeable membrane. The dialyzer is composed of thousands of tiny synthetic hollow fibers. The fiber wall acts as the semipermeable membrane. Blood flows through the fibers, dialysis solution flows around the outside of the fibers, and water and wastes move between these two solutions. The cleansed blood is then returned via the circuit back to the body. Ultrafiltration occurs by increasing the hydrostatic pressure across the dialyzer membrane. This usually is done by applying a negative pressure to the dialysate compartment of the dialyzer. This pressure gradient causes water and dissolved solutes to move from blood to dialysate, and allows the removal of several litres of excess fluid during a typical 4-hour treatment. In peritoneal dialysis, a sterile solution containing glucose (called dialysate) is run through a tube into the peritoneal cavity, the abdominal body cavity around the intestine, where the peritoneal membrane acts as a partially permeable membrane.
Abstract -physio -04

Hashimoto’s disease

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Hashimoto's thyroiditis or chronic lymphocytic thyroiditis is an autoimmune disease in which the thyroid gland is attacked by a variety of cell- and antibody-mediated immune processes. It was the first disease to be recognized as an autoimmune disease. It was first described by the Japanese specialist Hakaru Hashimoto in Germany in 1912. Hashimoto's thyroiditis very often results in hypothyroidism with bouts of hyperthyroidism. Symptoms of Hashimoto's thyroiditis include weight gain, depression, mania, sensitivity to heat and cold, paresthesia, chronic fatigue, panic, bradycardia, tachycardia, congestive heart failure, high cholesterol, reactive hypoglycemia, constipation, migraines, muscle weakness, joint stiffness, menorrhagia, myxedematous psychosis, cramps, memory loss, vision problems, infertility and hair loss. The thyroid gland may become firm, large, and nodular in Hashimoto's thyroiditis, but changes in the thyroid can also be nonpalpable. Enlargement of the thyroid is due to lymphocytic infiltration and fibrosis rather than tissue hypertrophy. Physiologically, antibodies against thyroid peroxidase (TPO) and/or thyroglobulin cause gradual destruction of follicles in the thyroid gland. Accordingly, the disease can be detected clinically by looking for these antibodies in the blood. It is also characterized by invasion of the thyroid tissue by leukocytes, mainly T-lymphocytes. It is associated with lymphoma family history of thyroid disorders is common; with the HLA-DR5 gene most strongly implicated conferring a relative risk of 3 in the UK. In addition Hashimoto's thyroiditis may be associated with CTLA-4 (Cytotoxic T-lymphocyte Associated-4) gene polymorphisms that result in reduced functioning of the gene's products, which are associated with negative regulation of T-lymphocyte activity. Down regulatory gene polymorphisms affecting CTLA4 are also associated with autoimmune pathology seen in development of Type I diabetes. The strong genetic component underscoring this theory is borne out in studies on monozygotic twins, with a concordance of 38-55%, with an even higher concordance of circulating thyroid antibodies not in relation to clinical presentation (up to 80% in monozygotic twins). Neither result was seen to a similar degree in dizygotic twins, offering strong favour for high genetic aetiology. There are multiple suggested mechanisms by which the pathology of Hashimoto's Thyroiditis develops. Various autoantibodies may be present against thyroid peroxidase, thyroglobulin and TSH receptors although a small percentage of patients may have none of these antibodies present. As indicated in various twin studies a percentage of the population may also have these antibodies without developing Hashimoto's thyroiditis. Diagnosis is made by detecting elevated levels of anti-thyroid peroxidase antibodies in the serum.