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# MSS Disorders

| Disorder | Symptoms | Description | Important Info |
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| Myasthenia gravis | -closed eye<br>-diplopia<br>-general fatigue | Autoimmune disease, Abs against Ach receptors affecting NMJ, disruption of contraction cascade, reduction in no. of nicotinic Ach receptors (AChRs) + voltage gated Na channels "due to complement damage to postsynaptic membrane". | Treatable, affecting females more. |
| Dysostosis | | -**Aplasia**→ lack of bone synthesis<br>-**Supernumerary digit**→ extra digit<br>-**Syndactyly**→ fusion of digits<br>-**Craniosynostosis**→ abnormal skull sutures (absence of fontanelles). | CONGENITAL, Abnormal condensation & migration of mesenchyme,<br>Genetic abnormalities of homeobox genes" MSS development", affecting cytokines and its receptors. |
| Dysplasia | **1.**big head, prominent forehead, short hands and limbs.<br>**2.**big head, small hands, very severe restriction in chest wall "collapsing chest wall = respiratory insufficiency".<br>**3.**too little fragile bone, multiple fractures, blue sclera, hearing loss, teeth abnormalities.<br>**4.**hard rigid very white bone, fractures, leukopenia = pancytopenia→ immune deficiency with opportunistic infections. | **1.Achondroplasia (Dwarfism)**→ most common, mutations in FGFR3, no impact on longevity – intelligence – reproduction, associated with advanced paternal age, affects bone formation.<br>**2.Thanatophoric dysplasia** (severe bad Dwarfism)→ most common lethal (utero, at birth, shortly after birth) dwarfism, FGFR3 mutation in other locus.<br>**3.Ostogenesis Imperfecta (Brittle bone disease)**→ most common inherited disorder of CT, group AD disorders, deficient collagen1 synthesis, 7 types with different clinical features + severity of deficiency "type 1 → normal life + supportive therapy, type 2 → lethal in utero – after birth".<br>**4.Osteopetrosis (Marble bone disease) (Stone bone)**→ group of rare disorders, impaired osteoclastic activity + reduced bone resorption => diffuse sclerosis, diagnosed by Xray ((OPPOSITE TO OSTEOPOROSIS)). | CONGENITAL, Disorganized bone & cartilage, Gene mutations in development and remodelling, not premalignant, can't be treated. |

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| Metabolic disorders – Osteopenia / Osteoporosis | -Femur + Pelvic fractures: immobility, Pulmonary embolism ''silent killer of hospitalized patients '' → increases the risk of DVT, Pneumonia.<br>-less bone trabeculae, less thickness.<br>- most common complication is fractures more commonly in central bones and vertebral column ''compression fractures''. | -Osteopenia (Mild Osteoporosis)→ 1-2.5 SD below M.<br>-Osteoporosis (Severe Osteopenia)→ more than 2.5 SD below M.<br>A. Generalized ''more common'', localized.<br>B. Primary: more common, aging + PM, Secondary: less common, hyperthyroidism, malnutrition, steroids. | Decreased bone mass + density, Diagnosis: special imaging technique, bone mineral density (BMD scan): dual-energy X-ray absorptiometry (DXA or DEXA scan) or bone densitometry, prevention: Exercise, Calcium & V.D, TX: Bisphosphonates: reduce osteoclast activity and induce its apoptosis, Denosumab: anti-RANKL; blocking osteoclast activation, Hormones (estrogen): risking DVT and stroke. |
| Metabolic disorders – Rickets + Osteomalacia | -pain, dental issues, fragile bone ''can't stand'', MSS deformities, poor growth and development.<br>-Bowing of legs. | -V.D deficiency / abnormal V.D metabolism→ decreased mineralization / unmineralized bone matrix→ increase risk of fractures. | -rare nowadays. |

Metabolic disorders – HPT

## Hyperparathyroidism classification

*Different causes and features of hyperparathyroidism - raised parathormone (PTH).*

We should memorize this table

| | primary | secondary | tertiary |
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| pathology | Hyperfunction of parathyroid cells due to hyperplasia, adenoma or carcinoma. | Physiological stimulation of parathyroid in response to hypocalcaemia. | Following long term physiological stimulation leading to hyperplasia. |
| associations | May be associated with multiple endocrine neoplasia. | Usually due to chronic renal failure or other causes of Vitamin D deficiency. | Seen in chronic renal failure. |
| serum calcium | high | low / normal | high |
| serum phosphate | low / normal | high | high |
| management | Usually surgery if symptomatic. Cincacalcet can be considered in those not fit for surgery. | Treatment of underlying cause. | Usually cinacalcet or surgery in those that don't respond. |

Complications: -Secondary Osteoporosis. -Osteitis Fibrosa Cystica/ osteitis fibrosa/ osteodystrophia fibrosa/ von Recklinghausen's disease of bone ''abnormal bone formation, with severe recurrent brown tumors in bone, inflammatory response, less white bone'' -Brown tumor''not a neoplasm, primary HPT, lack of minerals, weak trabeculae→intramedullary bleeding+fractures, after 3-6 months → sacs of multipe blood filled cysts form around it''.

-Primary: more common, problem in the organ itself, hyperplasia in more common than carcinoma''rare'' and adenoma, MEN has 3 types.

-PTH stimulates osteoblasts and regulate ca+2 levels.
-complications are rare but can manifest with severe prolonged HPT.

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| Paget disease of bone / Osteitis Deformans | -microscopic feature of mosaic pattern of lamellar bone.<br>-mostly asymptomatic and mild.<br>-pain by microfractures and nerve compression.<br>-Leontiasis Ossea / Lion face / Platybasia→ severe paget disease of skull, secondary osteoarthritis, fractures, osteosarcoma.<br>-3 phases '' can occur together'':<br>1.Lytic ''increased interstitium and osteoclastic activity, decreased bone trabeculae, upper part of femur, like osteoporosis, blacker''.<br>2.Mixed ''middle part of femur, black and white''.<br>3.Sclerotic ''increased bone trabeculae and osteoblastic activity, lower part of femur, like Marble bone disease, whiter''. | -increased badly bone structure'' all abnormal bone structure''.<br>-DX: x-ray, increased serum Alk P (enzyme/ unspecific), Normal Ca andPO4 with NO PTH or Vitamin D problems.<br>-no need for biopsy. | -unknown etiology.<br>-factors: genetics, environmental, nutrient, viruses ''measles + RNA viruses''.<br>-50% of Familial Paget + 10% of Sporadic Paget→ have SQSTM1 mutations ''+ RANK, -OPG, mainly in lytic phase''.<br>-increased risk of secondary Osteosarcoma from abnormal bone.<br>-lytic metastasis more common than blastic (sclerotic).<br>-85% polystotic, 15% mono.<br>-axial bone more affected'' femur''. |
| Blisters | creating a pocket filled with fluid. | Result from repeated rubbing of the skin. The epidermis is pulled away from the dermis. | |

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| Calluses & corns | | Constant pressure on skin will stimulate this layer (Stratum corneum) to grow thicker. | |
| Psoriasis | The extra skin cells form scales and red patches that are itchy and sometimes painful. | common skin condition that speeds up the life cycle of skin cells. causing cells to build up rapidly on the surface of the skin. Accelerated keratinization. | |
| Albinism | leads to someone having very light skin, hair, and eyes. | inherited condition of less melanin production in skin. | |
| ecchymosis(bruise) | | Haemorrhage from cutaneous blood vessels in dermis. | |
| Comedo(blackheads) →Acne | | -clogged hair follicle (pore) in the skin. Keratin combines with oil to block the follicle.<br>-Acne is caused by the infection by some type of bacteria of the accumulated sebum in the skin. | - common during puberty. |
| Black eye / Racoon eye / Panda eye | | -haemorrhage in the 4th layer of the scalp'' loose CT'', blood accumulated in the layer spreading over the entire extent of the aponeurosis reaching the eyelids. | |
| Cephalhematoma / subperiosteal hematoma | | -fluid collection under the pericranium taking the shape of the related bone. | -no treatment wait for months it'll drain.<br>-limited no lateral spread.<br>-happens when BVs get damaged in vacuum / forceps – assisted birth. |
| Bell's Palsy | -face dropping of the eyelid, inability to close the eye on the affected side, sagging of the angle of the lip on the affected part, loss of forehead wrinkles, overflow of the tear, loss of nasolabial fold, drooling of saliva from the angle, face pulled to healthy side, can't open his mouth or raise his eyebrow. | -facial muscle paralysis due to facial nerve damage in 1. Internal acoustic meatus by tumor 2. Middle ear by infection or operation 3. Fracture in the course 4. Compression in stylomastoid 5. Facial nerve canal (perineuritis) 6. Parotid gland by tumor or operation 7. Laceration of the face. | |

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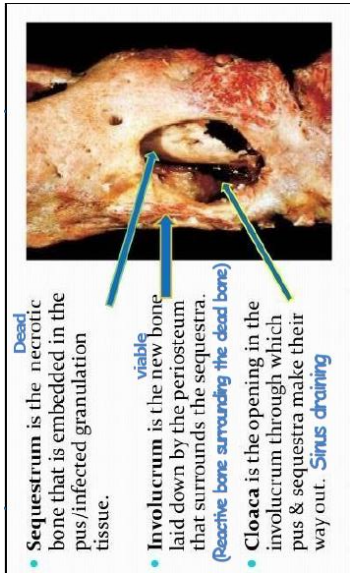
| Trigeminal neuralgia | | Common condition in which the patient experiences excruciating pain in the distribution of the mandibular or maxillary division, with the<br>ophthalmic division usually escaping. | A physician should be able to map out accurately on a patient's face the distribution of each of the divisions of the trigeminal nerve. |
| Osteonecrosis (Avascular necrosis) | -triangular / pyramidal, yellow, base wedged towards the joint, more avascular toward the joint.<br>-painful. | -Infarction (ischemic necrosis) of bone and marrow.<br>-associated conditions: 1. Vascular injury: trauma, vasculitis'' thromboembolic phenomena'' 2. Drugs: steroids 3. Systemic disease: Sickle'' frequent thrombosis'' 4. Radiation'' obstruction of BVs'' → ppl more experiencing radiation have high risk of developing avascular necrosis. | -most affected femur bone.<br>-mostly due to ischemia.<br>-necrotic part should be removed and replaced with an artificial joint. |
| Osteomyelitis / Acute Osteomyelitis / Acute bacterial Osteomyelitis / Acute Pyogenic Osteomyelitis<br>  <br> | -Hematogenous OM: systemic manifestations, fever, malaise, chills, leukocytosis, throbbing pain locally (hint). -Infants: subtle Adults: local pain. | -infections spread by the emissary vein(valveless) in the subaponeurotic space to intracranial venous sinuses + skull bone.<br>-Inflammation of bone/marrow due to infection. -1. Hematogenous spread'' most common'' (children) 2. Extension from contiguous site (adults, diabetic foot) 3. Direct implantation after compound # or orthopaedic procedure. -Long bones: metaphysis & epiphysis in adults; in children: epiphysis or metaphysis (not both). **-acute inflammation of BM→ spread of exudate→ thrombosis of vessels→ compression→ necrosis of bone→ liquefaction→ lifting of periosteum (late on xray )→ further necrosis.**<br>-DX: high index of suspicion; X-ray maybe normal in early phases (should not wait till we see x ray lytic changes), blood culture.<br>-Tx: admission, IV antibiotics and sometimes surgical drainage of pus. | - any organism can cause it. -Pyogenic osteomyelitis: bacteria, staph. aureus (80-90%). - E. Coli, Pseudomonas & Klebsiella are more common when UTI or IV drug abuse. - Neonates: Haemophilus influenzae & Group B Strept -Sicklers: Salmonella.<br>-common tissue necrosis and thrombosis. -severe pyogenic inflammation should be diagnosed early on before necrosis and breakdown take place. **- normal xray never rules out the probability of acute osteomyelitis.** |

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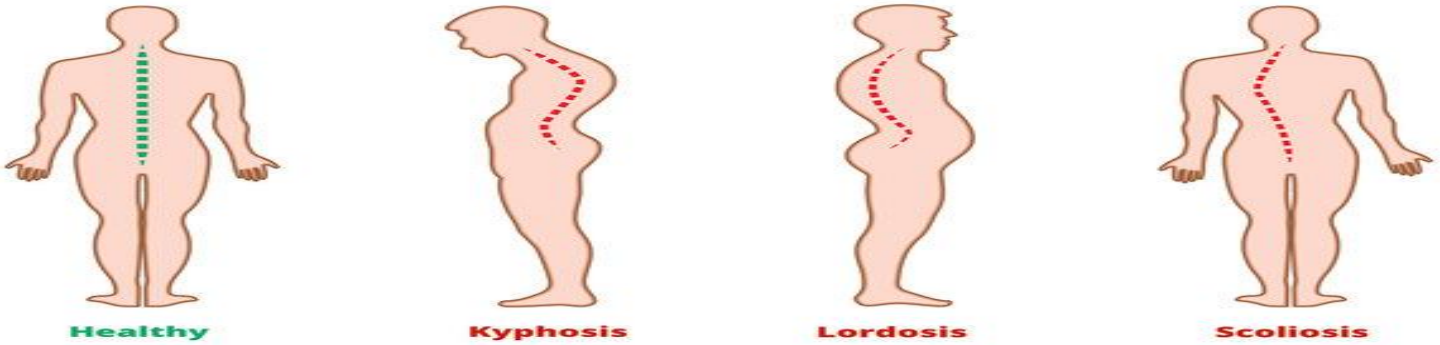
| Chronic Osteomyelitis | | -Causes: 1. Delay in diagnosis 2. Extensive necrosis / aggressive pathogen 3. Inadequate therapy (A. biotics or surgery) 4. Weakened host immunity '' cancer / steroids''. -complications of CH. OM: 1. Pathologic #s 2. Secondary amyloidosis (protein deposition in orans→ organ failure, associated with chronic diseases) 3. Endocarditis 4. Sepsis 5. SQ. cell Carcinoma of draining sinus (rare) 6. Sarcoma of bone. | - 5-25% of Acute OM persists as chronic OM - Very bad debilitating disease. |
| Mycobacterial Osteomyelitis | - maybe subtle and chronic course, necrotizing (caseating) Granulomas, central necrosis. **-TB Spondylitis (Pott Disease):** Destructive spine TB, Difficult to treat, may lead to #s, neurologic deficit, scoliosis, kyphosis. | -Hematogenous or direct spread.<br>**-TB Spondylitis (Pott Disease):** chronic TB osteomyelitis of vertebral bodies causing pain and compression fractures'' detected by xray''. | -1-3% of pts with pulmonary or extrapulm TB: can have bone involvement. |
| Intervertebral disc Herniation | --Leads to compression of spinal nerves and neurological manifestations. Sensory like numbness pain or sensory loss or Motors as muscle weakness. | -a fragment of the disc nucleus that is pushed out of the annulus, into the spinal canal through a tear or rupture in the annulus. | -can be corrected by laminectomy (removal of part or all the vertebral bone (lamina)). |
| Intervertebral disc Degeneration | -ageing and disc degeneration →limiting the ability of the disc to absorb shock. | -breakdown (degeneration) of one or more of the discs. | -can't be treated. |

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| Kyphosis | | -increase in the sagittal curvature present in the thoracic Part. -Causes: by muscular weakness or by structural changes in the vertebral bodies or by intervertebral discs. -may be CONGENITAL. | **1-Acute angular kyphosis**: due to Crush fractures or tuberculous destruction of the vertebral bodies.<br>**2- Senile kyphosis**: due to osteoporosis and or degeneration of the intervertebral discs involving the cervical, thoracic, and lumbar regions.<br>**3- Round-shouldered**: due to weak muscle tone with long hours of study or work over a low desk can lead to a gently curved kyphosis of the upper thoracic region. |
| Lordosis | | -an exaggeration in the sagittal curvature present in the lumbar region. | -Causes: An increase in the weight of the abdominal contents, as with the gravid uterus or a large ovarian tumor, Disease of the vertebral column such as spondylolisthesis'' one of the bones in your spine, known as a vertebra, slips out of position''. |
| Scoliosis | | -lateral deviation of the vertebral column. -mostly found in the thoracic region. | **-** Causes: Paralysis of muscles caused by poliomyelitis. Congenital hemivertebra'' deformation of the spine''. |
| Dislocations of the Vertebral Column | | -without fracture occur only in the cervical region. -commonly occur between the 4th and 5th or 5th and $6^{th}$ cervical vertebrae. -with fracture occur in thoracic and lumbar regions. | **-**Unilateral dislocations cause spinal nerve injury producing severe pain. Bilateral dislocations cause spinal cord injury.<br>-Bilateral upper cervical dislocations cause death due to injury of phrenic nerves C3 to 5 leads to diaphragm paralysis. |

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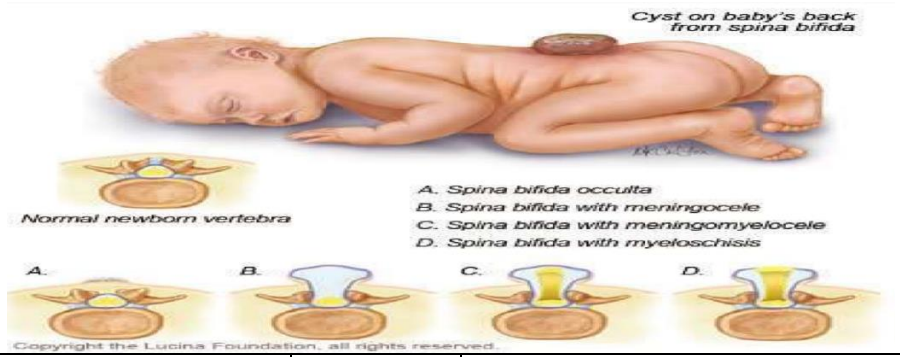
| Spina Bifida (CONGENITAL)<br>  <br><br> | -Failure of fusion of the two dorsal processes resulting in midline defect usually in lumbosacral region. | -**Spina bifida occulta**: The defect is covered with skin only.<br>**Spina bifida cystica**:<br>**A) Meningocele**: There is an opening in the spine where the meninges come through a sac. The sac is filled with cerebrospinal fluid and there is usually no nerve damage.<br>**B) Myelomeningocele**: The meninges and spinal nerves come through the open part of the spine. causing nerve damage and severe disabilities.<br>**C) Myeloschisis**: the neural tissue is exposed to the surface. |
| Sacralization of 5th lumbar vertebra (CONGENITAL) | | -The 5th lumbar vertebra fuses with the sacrum. | |
| Lumbralization of 1st sacral vertebra (CONGENITAL) | | -separation of the 1st piece of sacrum to form a separate vertebra. | |
| Hemivertebra (CONGENITAL) | | -failure of one of the chondrification center to appear so failure of half of vertebra to form→ defective vertebra produces scoliosis (lateral curvature). | |
| Hypotonia (Flaccidity) | | -decreased or lost muscle tone. -Flaccid muscles are loose and appear flattened rather than rounded. -Certain disorders of the nervous system and disruptions in the balance of electrolytes (Na, Ca, to a lesser extent, Mg) may result in flaccid paralysis. -characterized by loss of muscle tone, loss or reduction of tendon reflexes, and atrophy (wasting away) and degeneration of muscles. | |
| Hypertonia | | -increased muscle tone. -expressed by **spasticity or rigidity**. -Spasticity: increased muscle tone (stiffness) associated with an increase in tendon reflexes and pathological reflexes. -Rigidity: increased muscle tone in which reflexes are not affected. | |

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| Muscle fatigue | | -Prolonged and strong contraction of a muscle leads to it. - muscle fatigue increases in almost direct proportion to the rate of depletion of muscle glycogen. - **fatigue of the neuromuscular Junction (RARE):** stimulation of the nerve fibers at rates greater than 100 times per second for several minutes diminishes the number of acetylcholine vesicles→ impulses fail to pass into the muscle fibers (depletion of acetylcholine as a result of rapid stimulation of the muscle and in large quantities, which leads to the exit of all acetylcholine from its stores →there is no acetylcholine left to restimulate the muscle). -**CNS fatigue:** the cause and mechanism of its occurrence have not been determined. | -Causes: 1. inability of the contractile and metabolic processes of the muscle fibers to continue supplying the same work output (depletion in the sources that provide us with the energy molecules needed to carry out muscle contraction). 2. Interruption of blood flow (within 1 or 2 minutes because of loss of nutrient supply, especially O2). 3. most important cause is the accumulation of the final products of the glycolysis process. |
| Muscle hypertrophy | | -increase of the total mass (size) of a muscle. -results from an increase in the number of actin and myosin filaments in each muscle Fiber→ enlargement of the individual muscle fibers. - increasing size of myofibrils + enzyme systems (enzymes for glycolysis) that provide energy also increase →rapid supply of energy during short-term forceful muscle contraction. | **-Adjustment of Muscle Length:** type of hypertrophy occurs when muscles are stretched to a greater than normal Length→ causing new sarcomeres to be added at the ends of the muscle fibers, where they attach to the tendons. |
| Muscle atrophy | | -muscle remains unused for many weeks→ rate of degradation (by ATP dependent ubiquitin-proteasome pathway) of the contractile proteins is more rapid than the rate of replacement→ decrease in total muscle mass. | -Muscle Denervation →Rapid Atrophy (muscle loses its nerve supply → no longer receives the contractile signals that are required to maintain normal muscle size. -After about 2 months, degenerative changes also begin to appear in the muscle fibers. |

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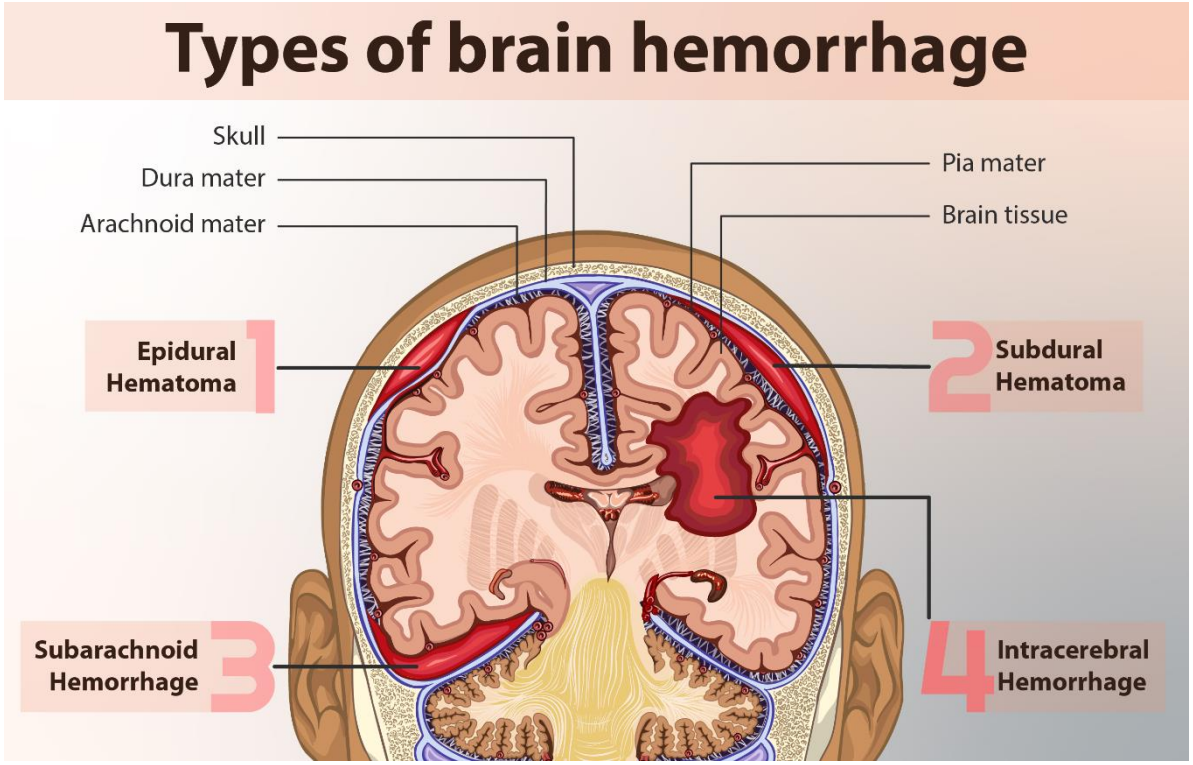
| Septic cavernous sinus thrombosis / Cavernous sinus syndrome | -Ophthalmoplegia with diminished pupillary light reflexes, Venous congestion leading to periorbital edema, Exophthalmos, Pain, or numbness of the face. | -results from sepsis from the central portion of the face (Danger area of the face), teeth, nose or paranasal sinuses→ damage to any of the cranial nerves that pass through it. -life-threatening and requires immediate treatment (antibiotics and sometimes surgical drainage). | -pterygoid venous plexus drains to nasal sinuses, teeth, ears, nose and deep structures→ Infection spreading from the nose, sinuses, ears, or teeth→ Septic cavernous sinus thrombosis. -Staphylococcus aureus and Streptococcus are often the associated bacteria. |




| Epidural | Subdural |
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| Between the skull and dura matter | Between dura and arachnoid matter |
| Rupture to meningeal vessels (**middle meningeal A**) | Rupture to cerebral veins (**bridging veins**) while approaching the venous sinus (superior cerebral veins) |
| Lense shaped (Biconvex) | Crescent shaped |
| Well localized | Poorly localized |
| Mostly arterial | Mostly venous |

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| Extradural hemorrhage/ epidural | - unconsciousness, respiratory arrest→ death, nausea, vomiting, seizures, weakness of the extremities. -limited laterally. -**CNIII (Oculomotor) Palsy:** complication of Epidural Hematoma that will present with "down and out" eyes and mydriasis due to compression of the Oculomotor Nerve via herniation. | -blow (Trauma (blow on skull, car accidents, falls....)) to the side of the head→ fracture of the skull in the region of Pterion→ Bleeding and strips up the dura from the kull bone→ hematoma expands, intense headache→ ICP rises→ the brain to shift, to be crushed against the skull, or herniate→ quickly compress the brainstem, causing unconsciousness. - To stop the hemorrhage, the torn artery or vein must be ligated or plugged. The burr hole through the skull wall should be placed about 1 to 1.5 in. (2.5 to 4 cm) above the midpoint of the zygomatic arch. -causes temporal lobe herniation (moving from supratentorial to infra)→ blood accumulates→ compress intracranial structures, which may impinge on the third cranial nerve, causing a fixed and dilated pupil on the side of the injury, eye will be positioned down and out, Compression of occulomotor nerve (III) → ipsilateral pupil dilation since the parasympathetic fibers that supply the constrictor pupillae are located on the outside of the nerve and are inactivated first by compression. | - Hallmark of epidural hematoma (Lucid interval) (talk and die syndrome): Often there is loss of consciousness (due to concussion) following a head injury, a brief regaining of consciousness (appear completely normal), and then loss of consciousness again, temporary improvement in a patient's condition after a traumatic brain injury, after which the condition deteriorates (followed by death), lasts for minutes or hours. - lucid interval is especially indicative of an epidural hematoma. |
| Subdural hemorrhage | -hugs the contour of the brain. | - excessive anteroposterior displacement of the brain within the skull (violent shaking of the head (e.g., child abuse or car accident)) and commonly occurs in alcoholics and elderly. | -causes tentorial herniation. |
| Subarachnoid hemorrhage | | - Extravasation of blood into the subarachnoid space between the pia and arachnoid. - ruptured cerebral aneurysm (balloon in BV ruptures with hypertension) or arteriovenous malformation. - The diagnosis is established by withdrawing heavily blood-stained cerebrospinal fluid through a lumbar puncture (spinal tap). | |
| Cerebral hemorrhage | | - Caused by bleeding within the brain tissue itself. - Mostly caused by hypertension rupturing cerebral arteries. | |

| Category | Behavior | Tumor Type | Common Locations | Age (yr) | Morphology |
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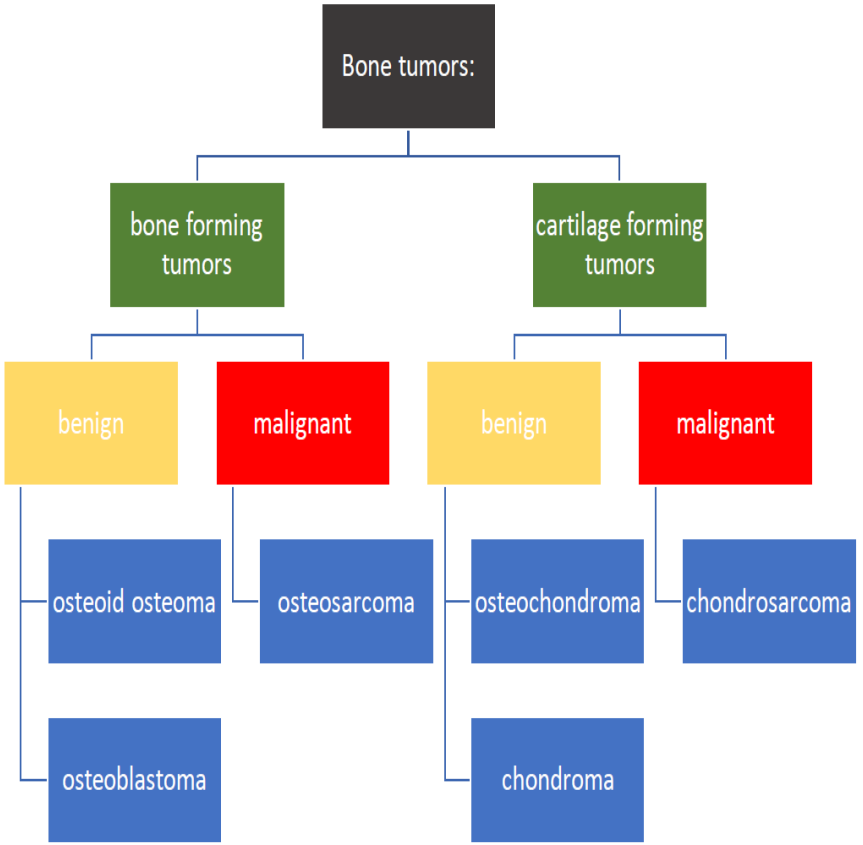
| **Cartilage forming** | Benign | Osteochondroma | Metaphysis of long bones | 10–30 | Bony excrescence with cartilage cap |
| — | — | Chondroma | Small bones of hands and feet | 30–50 | Circumscribed hyaline cartilage nodule in medulla |
| — | Malignant | Chondrosarcoma (conventional) | Pelvis, shoulder | 40–60 | Extends from medulla through cortex into soft tissue, chondrocytes with increased cellularity and atypia |
| **Bone forming** | Benign | Osteoid osteoma | Metaphysis of long bones | 10–20 | Cortical, interlacing microtrabeculae of woven bone |
| — | — | Osteoblastoma | Vertebral column | 10–20 | Posterior elements of vertebra, histology similar to osteoid osteoma |
| — | Malignant | Osteosarcoma | Metaphysis of distal femur, proximal tibia | 10–20 | Extends from medulla to lift periosteum, malignant cells producing woven bone |
| **Unknown origin** | Benign | Giant cell tumor | Epiphysis of long bones | 20–40 | Destroys medulla and cortex, sheets of osteoclasts |
| — | — | Aneurysmal bone cyst | Proximal tibia, distal femur, vertebra | 10–20 | Vertebral body, hemorrhagic spaces separated by cellular, fibrous septae |
| — | Malignant | Ewing sarcoma | Diaphysis of long bones | 10–20 | Sheets of primitive small round cells |



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| OSTEOID OSTEOMA | - Severe nocturnal pain , mediated by (PGE2) relieved by aspirin & NSAIDS. | - Femur & tibia; nidus with surrounding bone reaction.<br>- rim of reactive bone with some hemorrhage and reactive giant cells, you don't see atypia. | -much more common than osteoblastoma, < 2 cm (radiologically), young men. Treated by: radiofrequency, ablation or surgery. |
| OSTEOBLASTOMA | - Pain unresponsive to aspirin and NSAIDS. | - Posterior vertebrae; no rim of bone reaction.<br>- rim of reactive bone with some hemorrhage and reactive giant cells, you don't see atypia. | -> 2 cm (radiologically). -Treated by curetting. |
| OSTEOSARCOMA (osteogenic sarcoma) | -Progressive pain, pathological fracture. - progressive pain in the lower limb. | - Malignant osteogenic tumor (malignant osteoblast forming the abnormal woven bone), large destructive and infiltrative lesions with Codeman triangle. -lesions start in the bone then go deep into the medulla and outside into the periosteum→ elevating it forming an angle called codeman triangle. -limb salvage resection: biopsy (malignant osteoid haphazard abnormal mitosis)→ Neoadjuvant chemotherapy (avoid metastasis before surgery)→ surgery / local radiation→ local radiation / surgery → chemotherapy. -histological appearance: malignant osteoid by malignant osteoblast, all woven bone. - Hematogenous spread to lungs. -5 year survival reaches 60-70%. -Presence of mets at diagnosis is a bad prognostic factor (stage4). | - is the most common primary malignant tumor of bone after hematopoietic malignancies. -75% adolescents (between 5-15), older (after55)→ (secondary osteosarcoma). - Males > females. -Metaphysis of long bones (distal femur & proximal tibia) (Around the knee joint). - Genetic abnormalities: mutations in RB gene, TP53 gene, CDKN2A (p16 &p14 ), MDM2, CDK2.<br>- codeman triangle is not specific for osteosarcoma (not pathogonomic ). |
| Osteochondroma (benign exostoses) | -can cause pathologic fractures and pain. | -around the cartilaginous growth plate of the long bones (but it can occur in any bone). -normal cartilage, normal periosteum, and normal bone marrow. -cartilage does not appear on X-ray→ smaller exostosis. -exophytic pedunculated mass with normal cartilaginous cap, no destruction of the surrounding tissue, no infiltration or Codman triangle. | -Asymptomatic in most cases, solitary (85%) / multiple hereditary exostoses (MHE): EXT1, EXT2 gene mutations. -Rare (<3-5%) transformation to chondrosarcoma (more common in MHE). |

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| Medullary enchondroma / Cortical chondroma / Chondroma / Enchondroma | | -Benign hyaline cartilage tumors in bones with endochondral origin. -Multiple enchondromas: **Ollier disease. -Maffucci syndrome**: multiple enchondromas + skin hemangiomatosis. - On x-ray: 1. No destruction or infiltration 2. Cartilaginous appearance 3. No elevation in periosteum (no Codemans triangle) 4. Appears on small bones of hand and feet. -Histologically: taken by curetting looks normal cartilage. - bubble soap appearance. | -Mostly Solitary metaphyseal lesions; 20 50 years (mostly in: small bones of the hands and the feet). -IDH 1 & IDH 2 gene mutations. |
| Chondrosarcoma | | -Malignant tumors producing malignant cartilage. -Large destructive masses; shoulder, pelvis, ribs, skull, vertebral body. -infiltrating into the bone marrow and the soft tissue outside, and it is elevating the periosteum causing Codman triangle. -cartilaginous characteristics of the tumor. -bubble soap appearance in CT scan. -histological appearance: malignant lobulated cartilage. | -50 %the incidence of osteosarcoma. -40 50 years of age, M:F 2:1.- Genes: EXT, IDH1, IDH2, COL2A1, CDKN2A1. -Px: depends on grade. -Tx: surgical, chemotherapy. |
| Chalazion | -painless cyst on eyelid. | -blockage of meibomian gland (tarsal) by its oily material internally in the inner eyelid. -if infected will collect pus and be painful. | -blockage of zeis gland ducts→ external collection of oily material under the skin. |
| Ptosis | | -dropping of the upper eyelid. -**complete ptosis:** Loss of oculomotor nerve [III] function. - **partial ptosis:** loss of sympathetic innervation to the superior tarsal muscle. | - **Exophthalmos/ proptosis:** increased overall volume of orbital fat, e.g. hyperthyroidism (Graves' disease), may lead to forward protrusion of the eyeball. |
| Horner's Syndrome | | - lesion in the sympathetic trunk in the neck that results in sympathetic dysfunction. | 1-Pupillary constriction: paralysis of the dilator pupillae muscle. 2-Partial ptosis: paralysis of the superior tarsal muscle of the levator palpebrae superioris. 3-Absence of sweating (anhidrosis): ipsilateral side of the face and the neck. 4-flushing of skin. |
| Papilledema | | - swollen optic disc caused by increased intracranial pressure. -Any increase in intracranial pressure results in increased pressure in the subarachnoid space surrounding the optic nerve. | - seen when retina is examined using an Ophthalmoscope. -in cavernous sinus thrombosis, venous congestion within the retina may cause papilledema. |