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{only for PG Entrance Examinations Preparation}

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1. **Fetal or maternal growth hormone is not essential for fetal growth in utero.(Maximally affected by INSULIN).**
2. **Stem stature index : The upper : lower segment ratio at 2 yrs of age in a normal child is 1.5:1.**
3. The brain enlarges rapidly **during the latter months of fetal life and "early months of postnatal life".**At birth ,the head size is about 65 to 70 percent of the expected head of size in adults.It reaches 90 percent of the adult size by the age of 2 years**.Maximum growth of brain occurs during infancy among various postnatal and later periods.**
4. **Perinatal Period :- 22 weeks of gestation to 7 days after birth.The maximum no. of deaths in children occur in First 7 days.**
5. **Post Natal Period :- NewBorn → First 4 Weeks (1 month)after birth , Infancy -→ First Year , Toddler -→1 to 3 years , Preschool child →3- 6 years,School age 6- 12 years.so < 1 month is neonate,1month-12 years is infancy and childhood, 12-18 years are adolescent,18-35 years is young adults, > 35 years is older adults,> 65 years are elderly.**
6. **Weight :-** 2 times(2 ×3) his birth weight (approx 3 kg, LBW is less than 2.5 kg irrespective of the gestational period) at 5 months, **3 times at 1 year**, 4 times at 2 year, 5 times at 3 year, 6 times at 5 year, 7 times at 7 years,10 times at 10 years.During the first year,weight increases by 7 kg .After that the increase in weight is not so fast-only about 2.5 kg during the second year and from then until puberty by about 2 kg per year.
7. **Length** : The baby measures **50 cm at birth. 75 cm at 1 year of age**. Increase in length in 1st year is 25 cm (**50% increase in length in 1st year**.). 100 cm at the age of 4 and a half years (**Doubles the birth length at 4 and half years**).Thereafter 5 cm in height every year,until the age of 10 years.
8. **Head circumference** :- at birth -35 cm, at 12 months- 45 cm**. (10 cm increase in first year, *cf point 3*).**

9. **Circumference of the chest** :- The circumference of chest is about 3 cm less than the head circumference at birth. **The circumference of head and chest are almost by the age of 1 year.Thereafter the chest circumference exceeds the head circumference.**
10. **Water content in infant is 75 - 80%**
11. **Eruption of teeth :- 20 DECIDUOS TEETH , The lower central incisor appears, between the ages of 5 and 8 months**.The upper central incisors appear a month later. **By 2 years all the milk teeth are erupted.** The temp teeth begin to shed from the 6th to the 7 th year,after the eruption of the **1st permanent molar** behind the second temporary molar teeth**.** (a child at the age **of 6-7 years** till 10-12 years have total **24 teeth** ; 20 temp or replaced by permanent + 4 first molars at a new place**)** .**Therefore 1st permanent Molar erupt or first permanent tooth erupt at 6 years.The period of mixed dentition persists till about twelve to thiteen years.(6-11 years is a period of mixed dentition in most).**

Temporary Teeth	Eruption Time	Complete Root Calcification
Central incisors (lower) 2	6-8 months	1 1/2-2 years
Central incisors (upper) 2	7-9 months	1 1/2-2 years
Lateral incisors (upper) 2	7-9 months	1 1/2-2 years
Lateral incisors (lower) 2	10-12 months	1 1/2-2 years
First molars 4	12-14 months	2-2 1/2 years
Canines 4	17-18 months	2-2 1/2 years
Second molars 4	20-30 months	3 years

Permanent Teeth	Eruption Time	Complete Root Calcification
First molars 4	6-7 years	9-10 years
Central incisors 4	6-8 years	10 years
Lateral incisors 4	8-9 years	11 years
First bicuspid 4	9-11 years	12-13 years
Second bicuspid 4	10-12 years	13-14 years
Canines 4	11-12 years	13-15 years
Second molars 4	12-14 years	14-16 years
Third molars ('wisdom') 4	17-21 years	18-25 years

 # 20 permanent teeth and 4 Temporary teeth at the age of 10-12 years. All temporary teets are replaced except 4 canines.(total is still 24)(see table:- 10-12 years eruption time)

#A girl of 10 years will have 16 permanent teeth and 8 temporary teeths.(total is still 24) (see table:- 9-11 years eruption time)

#A child at the age of 7 years has 24 teeths.

#Second temporary molars and first temporary molars are replaced by biscupids

Only First second and third permanent molars are in new positon.

#1st and 2nd permanent molars add to total number of teeth,others are replaced.

2nd permanent appears very late. *****So total number of teeth remains 24 from the appearance of first molar i.e. 6-7 years and upto 11-12 years i.e. before the appearance of second molars (period of mixed dentition).

12. **The best method to determine age upto 14 years is Dentition.**
13. **<u>Bones</u> :- At birth ,the lower end of femur shows a centre of ossification about ½ cm in diameter. Ossification centre appearing just before the birth is lower end of femur. Capitate and hammate ossify at the age of 4 months, So before that , no bone is seen on radiography but after 4 months of age 2 carpal bone will be seen on radiography.(sutton) Between 2 and 6 years,the number of carpal bones present on x ray represents the approximate age in years.For examplae 4 carpal bones means 4 years. By 16-18 years,all the epiphysis at the elbow (except the medial epicondyle),head of the femur ,and lower end of the tibia join the respective shafts in males. BUT ,The age of 16 year old female is best determined by the radiograph of LOWER END OF RADIUS AND ULNA. :-Lower end of radius and ulna fuse at 16-17 years in girls(18-19 in boys).The age of 15 year old female is best determined by radiography of upper end of radius and ulna??????. In the females,the epiphysis at the elbow join their respective shafts by 13-14 years.(15-17 in boys).The age of a 17 year old female is best determined by upper end of humerus which fuse at 17-18 years (19-20 in boys). The age of 18 year old female is best determined by crest of ilium. (see table below). Patella completely ossifies by the age of puberty.**

Region	Girls	Boys
Elbow	13-14	15-17
Wrist	16-17	18-19
Shoulder	17-18	19-20
Crest of ilium	18-19	20-21
Ischial tuberosity	21-22	23-24
Inner end of clavicle	21-22	23-24

The indication of age is based on the union of epiphysis as per the following data.

14. **Motor development :-From the age of 4 weeks to 12 weeks,the infant learns to lift and control his head in the horizontal plane and then above the horizontal plane. Head movement is possible in an infant by 4 weeks(1 month),at first he can do it momentarily in horizontal plane for few seconds. Total head control(Neck holding) is possible by 12 weeks(3 Months),plane of face almost reaches to 90˚ to couch for several seconds.**

Age	*Milestone*
3 months	Neck holding
5 months	Sitting with support
8 months	Sitting without support
9 months	Standing with support
10 months	Walking without support
11 months	Crawling (Creeping)
12 months	Standing without support
13 months	Walking without support
18 months	Running
24 months	Walking upstairs
36 months	Riding tricycle

Age	*Milestone*
4 months	Grasps a rattle or rings when placed in hand
5 months	Reaches out to an object and holds it with both hands (intentional reaching with *bidextrous* grasp)
7 months	Holding objects with crude grasp from palm (*palmar* grasp)
9 months	Holding small object, like a pellet, between index finger and thumb (*pincer* grasp)

Age	*Milestone*
1 month	Turns head to sound
3 months	Cooing
6 months	Monosyllables (*'ma', 'ba'*)
9 months	Bisyllables (*'mama', 'baba'*)
12 months	Two words with meaning
18 months	Ten words with meaning
24 months	Simple sentence
36 months	Telling a story

By the age of 8 months he crawls in head.By the age of 10 months he creeps keeping his abdomen off the ground. Bidextrous grasp :- 5 months, Pincer grasp:- 9 months , At 9 months bisyllables (mama,baba).

Eye cordination:-By the age of 6 weeks,he follows the object by several unsteady excursions of the eyes,by the age of 2-3 months,the infant follows the object with steady movement of the eyes,by this time he can also converge and focus his eyes (Fix Gaze). Binocular Vision:- develops by 3-6 months,Depth perception begins by 6-8 months but is not very accurately established till the age of 6-7 years..

Social smile at 2 months.

Intellectual development:- 1) Sensorimotor (0-2 years) 2) Concrete thinking (2-7 years) ***3) Abstract or conceptual thinking (7-11 years) a child takes self decision at the of 7 years..*** *4) Adolescent or formal operational begins at 11 years.*

By the age of 18 months uses 10 words with meaning. Hand to mouth coordination:- By 18 months he can feed himself from a cup with only slight spilling.

At 3 years he can withhold and postpone his bowel movement. Preference of use of one hand is evident by 3 years.(Handedness develops at 36 months).. AT 36 Months knows gender. ***The child copies a circle by the age of 3 years***

. A child can **hop by 4 years** and walk in a straight line back and forth or balance on one foot for 5-10 seconds by 5 years.

A 5 year child can remember numbers upto 5 digits.

Which of the following is considered normal in a 4 yr old child :- a) Tics× b)Hyperexcitibality? c)Anxiety ? d) Hyperkinesis×????

15. **Max growth spurt in male occurs at 10-12 ml of testicular volume or stage III of pubic hair(Tanners) .Increasr in muscle mass is probably due to adrenal hormones. Maximum growth spurt in females occurs at Breast stage III (tanners) or at the time of menarche.First sign of puberty in males is increase in testicular volume(growth of testes) while in females is thelarche (breast development).**
16. **The fetal length is affected if the mother has undernutrition at any time during the pregnancy.**
17. **Short Stature :→In hypothyroidism the ratio between the upper and lower segments is immature.Dwarfism with disproportionate body proportion is seen in hypothyroidism(while in GH def. Dwarfism is proportionate). Bone age is markedly delayed. Epiphseal development is severely retarded. While bone age is normal in Familial intrinsic short stature.There is delayed bone age in constitutional delay in growth. There is also delayed bone age in psychosocial short stature. Bone growth is influenced maximum by the GH.In general ,delayed bone age in a child with short stature is suggestive of a hormonal or systemic disorder ,whereas normal bone age in a short child is more likely to be caused by a genetic growth plate disorder.(cf harrison). Bone age is normal in genetic short stature/or familial short stature.Glucocorticoid excess(supraphysiologic levels attenuate growth *often associated with obesity) is ass with delayed bone age. While sex steroid def. after***

10-11 years impairs growth.*Isolated GH deficiency is characterised by short stature ,micropenis,increased fat,high pitched voice,and a propensity to hypoglycemia. *All of the following hormones can affect growth of a child except :- a) GH b) Insulin C) ACTH d) Somatostatin.??????

18. **50th percentile of the normal weight is normal.**
19. **Adjustment mechanism used most commonly by children is PROJECTION.(Unconscious attribution of one`s own attitudes and urges to other person(s) , because of intolerance or painful affect aroused by those attitude and urges.) A universal phenimenon though occurs more commonly in children.**
20. **Breadth holding spells :-** Reflexive events in which typically there is a provoking agent that causes anger ,frustration and child starts to cry. The crying stops at full expiration when the child becomes **apneic** and **cyanotic** or pale.In some cases ,the event continues and **the child may loose consciousness** and muscle tone and fall to the ground. Ocassionally **child may have seizure**. These spells are seen generally between **6-18 months of age**. **True breadth holding spells generally do not occur after 5 years**. Treatment involves parental reassurance **that the spells will not harm the children**.
21. **Nail biting and thumb suckling :-** ***Must not be treated vigorously in first year of sucking*. If parents ignore this harmless behaviour most children spontaneously relinquish it between the ages 4 and 5 years.If parents however criticises ecessively,children may stubbornly persist longer that they would otherwise. It can be associated with number of sequalae if it persists beyond 4-6 years.Finger sucking should be terminated by 8 years to avoid displacement of permanent teeth.(permanent teeth starts erupting 6- 8 years).Finger sucking is a source of pleasure,it is a sign of insecurity and it can lead to malocclusion.** Treatment is indicated for thumbsucking in children older than 4 years of age who suck their thumb or fingers in multiple settings or both during day and at night.
22. **Temper tantrum :-** Also **gradually subsides** in between 3 to 6 years.
23. **Evening colic :-It can persist till 3-4 months of age, Colic generally disappears by 4 months.** Exact cause onot known.
24. **Preschool age problems**:- **Stuttering** usually begins between the ages **of 2 and 5 years** ,a period in which there is non-fluency of speech.Stuttering during the phase of non-fluent speech between the ages of 2 and 5 years will pass off. They should not show undue concern.
25. **Oedipal/phallic phase(3 to 5 years)** is also **a preschool age phase. While oral phase(Birth to 1-1½ years) seen in infancy, Anal phase(1-1½ years to 3 years) seen in toddler .Psychiatric symptoms theorised to result from fixation at the regression to these stages are as follows**::- **Neurotic,hysteria fixation is phallic**. **OCD fixed at Anal stage**.Schizophrenia,severe mood disorders,personality disorders theorised to result from fixation at the regression to oral phase.
26. **Behavior in middle childhood(school years):- Fears :-The typical fears are fear of *death and injury*.**

27. **Enuresis :-** It is defined as normal nearly complete evacuation of the bladder at a wrong place and time at least twice a month after the fifth year of life.Most children will have **complete diurnal and nocturnal control** by **five years of age**.It is primary enuresis if child has never been dry in night and secondary if the child had been dry for several months and again starts bedwetting. The commonest cause is always **Psychological stress.**
28. **Attention –Deficit Hyperactivity Disorder also called as "Minimal Brain Dysfunction"/ hyperkinetic disorder :- Inattention,hyperactivity - impulsivity. Disturbance causes significant distress in learning and adaptability and impairment in academic and behaviour pattern.He often talks excessively and often has difficulty awaiting turn(symptoms under hyperactivity-impulsivity category).He has difficulty in sustaining attention in tasks and fails to give close attention to details or makes careless mistakes in schoolwork,work or other activities(symptoms under Inattention category). Treatment :- Stimulants are used :- Dextroamphetamine and methylphenidate are the DOC. ***** *Barbiturates are contraindicated as they increase hyperactivity.***
29. **Autistic Spectrum Disorders :- ASD share a Triad of impaired social interaction (marked impairment in reciprocal social and interpersonal interaction) , communication(marked impairment in language and non-verbal communication) , and imagination. The most noticeable characteristic is inability to develop normal social skill with lack of eye contact,gestures and facial expression.They understand little or no language.They therefore fail to acquire speech.Young Children with autism also have deficient comprehension and communicative use of speech and gesture. Early accurate diagnosis is of extreme importance. Parents must understand that they are not responsible for their child`s condition.**
30. **A homosexual stage in early adolescence is considered to be normal phenomenon.(Nelson). The behaviour of an adolescent is probably best described as being paradoxical. Lymphocytes are important protective influence against childhood infections.After puberty neutropils replace lymphocytes as the predominant leucocyte.**
31. **Water content in infant is 75-80%. 50% in women and 60% of body weight in man.**
32. **Sodium is the major determinant of plasma osmolality. Urea is ineffective osmole. Normal plasma osmolality is 255-290 mosmol/kg and the average osmotic threshold for thirst is approximately 295 mosmol/kg. Water intake regulated by thirst through osmoreceptors. Water excretion is regulated by AVP,arginine vasopressin (antidiuretic hormone) acting on principal cells in collecting ducts.The major stimulus for AVP secretion is hypertonicity.An increase in tonicity is sensed by osmoreceptors leading to enhanced secretion of AVP and vice versa.**
33. **Hyponatremia:- Leads to increased ICF volume,specifically brain swelling or cerebral edema.Therefore the symptoms are primarily neurologic.:- Headache ,lethargy,confusion and obtundation.**

34. **The major symptoms of Hypernatremia are also neurologic:- A decreased brain cell volume is associated with an increased risk of subarachnoid or intracerebral hemorrhage leading to altered mental status,weakness ,neuromuscular irritability,focal neurologic deficits.CDI and NDI can lead to hypernatremia.CDI and NDI can generally be distinguished by Desmopressin;the urine osmolality should increase by atleast 50% in case of CDI but there is no change in case of NDI(as there is defect at receptor levels not in the deficiency of AVP). The ICF depletion results in a peculiar doughy feel of the skin and a woody consistency of the tongue. Causes :- Osmotic diarrhoea, Accidental Salt Administration in ORS or Infant feeds.(intake of fluids with more salts) and in viral gastroenteritides.(in contrast secretory dioarrhoea as caused by cholera,carcinoid,Vipoma have a fecal osmolality similar to that of plasma and present with ECF volume contraction and a normal plasma Na Conc or hyponatremia).**
35. **In Darrows solution K is 36 meq/l. In hartmann`s solution (Ringer`s lactate) K is 20 meq/l.**
36. **Infants Require on an average 103 kcal/kg/day. A child of 1- 2years of age requires 1200 kcal/day.**
37. **People eating seafood have LOW TXA2.** ,Omega -3 fatty acids especially DHA and EPA are plentiful in fish. EPA produces the series-3 prostanoids e.g. PG 3,thromboxane 3(TX3), and PGI 3 .PG3 and TX 3 inhibit formation of series-2 prostanoids (PG 2 and TX2),which favor atherogenesis. **Total fat intake should provide nomore than 30 percent of daily energy intake. Carbohydrates should contribute 55-60 percent of total energy intake.It is safer to obtain 10 to 15 % of calories from the dietary protein. Human breast milk has approx 15 calories/ounce/28.35 gm. All are complications of formula fed over breast fed baby like necrotising enterocolitis,ottitis media, hypocalcemia except vit deficiency as breast milk contains very little vit k but formula fed is rich in it.The normal avearge stool sodium content in a new born who is fed on bresat milk alone is:- 19 meq/l. Brest milk is rich in polyunsaturated fatty acids. Fatty acid necessary during 0-6 months of age is linolenic acid out of other EFA.**
38. **<u>Nutritional content of breast milk</u> :-calories 67 kcal, proteins 1.1 g, fat 3.5 g, lactose 7.0 g all per 100 gm. lactose max in breast milk.**

Table 23–10. Composition of colostrum and milk.[1] (Units are weight per deciliter.)

Component	Human Colostrum	Human Milk	Cows' Milk
Water, g	…	88	88
Lactose, g	5.3	6.8	5.0
Protein, g	2.7	1.2	3.3
Casein:lactalbumin ratio	…	1:2	3:1
Fat, g	2.9	3.8	3.7
Linoleic acid	…	8.3% of fat	1.6% of fat
Sodium, mg	92	15	58
Potassium, mg	55	55	138
Chloride, mg	117	43	103
Calcium, mg	31	33	125
Magnesium, mg	4	4	12
Phosphorus, mg	14	15	100
Iron, mg	0.09[2]	0.15[2]	0.10[2]
Vit A, µg	89	53	34
Vit D, µg	…	0.03[2]	0.06[2]
Thiamine, µg	15	16	42
Riboflavin, µg	30	43	157
Nicotinic acid, µg	75	172	85
Ascorbic acid, mg	4.4[2]	4.3[2]	1.6[2]

		Buffalo	Cow	Goat	Human
Fat	(g)	6.5	4.1	4.5	3.4
Protein	(g)	4.3	3.2	3.3	1.1
Lactose	(g)	5.1	4.4	4.6	7.4
Calcium	(mg)	210	120	170	28
Iron	(mg)	0.2	0.2	0.3	
Vitamin C	(mg)	1	2	1	3
Minerals	(g)	0.8	0.8	0.8	0.1
Water	(g)	81.0	87	86.8	88
Energy	(kcal)	117	67	72	65

Source : *(157)*

***********$$$$$$$ "LACTOSE" AND WATER MAXIMUM IN HUMAN BREAST MILK. PROTEIN,FAT AND CALCIUM MAX IN BUFFALO MILK ;next is cow**

milk in these nutrients value. LACTOSE AND ENERGY MINIMAL IN COW`S MILK. VITAMIN A AND VITAMIN C MAXIMUM IN COLOSTRUM; next is human milk in these nutrients value. Colostrum contains excess of proteins,minerals,immunoglobulins but less of fat when compared to human breast milk. Casein lactalbumin ratio is 1: 2 in human milk. Advantage of cows milk over breast milk and colostrum:- Less lactose,good proteins,Good minerals,Good calcium(1200 MG/LITRE) and phosphorus,good riboflavin and thiamnine but less vit A and ascorbic acid.(VIT A and Ascorbic acid are max in colostrum and breast milk)

39. **Breast feeding should be initiated within half an hour after *normal delivery* and four hours after *cesarean section*.Breastfeeding alone is *sufficient* food for first 6 months. Nutritional supplement should be introduced in the diet of infants after the age of 6 months.Indian mothers secrete 450-600ml of milk daily.**
40. **Protein Energy Malnutrition :-Earliest indicator is underweight for age. 1) IAP classification based on weight for age(More than 80% of expected for age as normal) : Grade I :- (71-80 %), II (61-70 %), III (51-60%), IV (≤ 50%). Mid Arm Circumference doesn't show much change in 1-4 years. 2) WHO classification is based on NCHS &CDC standards,USA according to ghai (based on 50th percentile (median)of wt. for age/height or height for age of american standards).It is able to distinguish stunting(chronic course of malnutrition) from wasting (acute onset malnutrition). Wasting;index of severity of malnutrition measured by deficit in weight for height.Severe wasting is Less than 70% of expected(median). Stunting;index of duration of malnutrition measured by deficit in height for age.These two deficits are measured in WHO classification of malnutrition. Gomez classification is based on weight for age like IAP classification and reference is of boston/harvard standards.Waterlow classification is like WHO classification & defines stunting and wasting.Height is a stable measurement of growth as compared to body weight.Whereas weight reflects only the present health status of the child,height indicates the events in the past also. Mid arm circumference doesn't show much change in 1- 5 years. So it is age independent anthropometric index. PCM is typically associated with low serum levels of Vitamin A, Zinc & Magnesium.**
41. **Marasmus :- refers to the generalized starvation with loss of body fat and protein. In marasmus;The buccal pad of fat is preserved till the malnutrition become extreme. The baby appears alert but often irritable. Whereas in Kwashiorkar{condition seen in displaced child according to ghanian language} refers to selective protein malnutrition with edema and fatty liver.Markedly retarded growth .psychomotor changes and EDEMA are three essential features. Child is lethargic,listless and apathetic and appears miserable (in marasmus he is alert but irritable).Flaky paint appearance of skin is seen in kwashiorkar.(Flaky paint rash of lower extremity indicates Zinc Deficiency; Harrison15th ed. 458). Flaky paint deramtoses is called because it appears like old paint flaking off the surface of the wood. Flag sign (alternate bands of normal and light colour) is also seen.**

Hyperpigmentation is also seen(Niacin Def). Fructose is not used in I/V infusion as it causes lactic acidosis and hepatocellular dysfunction. In Malnutrition there is diminished activity of xanthine oxidase. Pancreatic acini are atrophied and zymogen granules are reduced.The salivary glands atrohy .The liver shows fatty infiltration. In malnutrition there is diminished activity of xanthine oxidase.Most common cause of death in starvation is hepatic failure.Early death can occur due to hypoglycemia,hypothermia,infections,fluid and electrolyte imbalance, severe anemia, liver failure, cardiac failure, dehydration, or overhydration.The principal tasks during initial treatment are to detect,treat and prevent common and life threatening complications.

42. **The disadvantage of elemental diet in children is Hypertonic Dehydration.**
43. **MC cause of blindness in children is vitamin A def.**
44. **WHO classification of xerophthalmia(123NeedForSpeed) :- X1A:- Conjunctival Xerosis , X1B :- Bitot`s spots ,X2:- Corneal xerosis ,X3A:- Corneal ulceration < 1/3rd , X3B:- Corneal ulceration > 1/3rd Of cornea, XN:-Night Blindness , XF :- Fundal changes , XS :- Corneal scarring. In Xerophthalmia ,Anterior segment of eye is initially involved. Infants who are not breast fed should receive *a 50,000 I.U. supplement of vitamin A by two months of age* in areas of endemic vitamin A def.Therefore ORAL Dose of neonate(0-2 MONTHS) is 27.5 mg of retinol palmitate containing 50,000IU of vit A or 15 mg of retinol.(1 mcg of retinol = 3.33 IU = 1 RE ; & 110 mg of retinyl palmitate=60 mg of retinol).Every infant should be administered one dose of 1 lac units(DOSE in children less than 12 months of age) of vitamin A along with measles vaccine at 9 months followed by Four more doses of 2 lac I.U.(Dose in children more than 12 months of age) each at 18,24,30 and 36 months. SO total NO. of doses of 5 should be given to every child/infant. Recommended 'Daily' intake of Vitamin A For CHILDREN 7 to 12 years of age is 600 mcg.For 0-12 months is 350 mcg and for adults is 600 mcg or 600 RE or 2000 IU (according to harrison it is 1000 RE or mcg of retinol in adult males and 800 in adult females?????? RDA for infants and children are same as of parks;ICMR) . Symptoms of Vitamin A def include hyperkeratotic skin lesions also apart from xerosis ,bitots spots and corneal ulceration. *****Vitamin A def leads to hyperkeratotic skin lesions while hypervitaminosis leads to dry skin,cheilosis,glossitis and alopecia.Vitamin A in the strictest sense refers to retionl.however its oxidized metabolites,retinaldehyde and retinoic acid are also biologically active compounds.The term retinoids include synthetic molecules that are chemically related to retinol. Retinaldehyde is required for normal vision.Retinoic acid is required for normal morhogenesis,growth and cell differentiation.Retinol is involved in reproduction. Hypervitaminosis:- Acute toxicity leads to Pseudotumour cerebri [increased ICT] Chronic toxicity leads to vomiting,alopecia,bone pain,features of pseudotumour cerebri with increased Intracranial pressure,papilledema,portal hypertension.** High doses of carotenoids do not result in toxic symptoms.However carotenemia which is characterised by a yellowing of the skin (creases of palms and soles) but not the sclerae,may be seen after ingestion of > 30 mg of β-carotene on a daily basis. **Hypothyroid patients are particularly susceptible to the development of Carotenemia. In West ,vit A def is caused by malabsorption.**

45. **The daily requirement of iodine for adults is placed at 150 mcg per day.The % of total body iodine in the thyroid gland is 90%. The adult human body contains about 50 mg of iodine.The spectrum of iodine def disorders ranges from mental deficiency to intrauterine death.In india the level of iodisation is fixed under prevention of food adulteration act(PFA) and is not less than 30ppm(or 1 in 30000) at the production point ,and not less than 15 ppm of iodine at the consumer level.Currently No less than 140 Million people are estimated to be living in goitre-endemic regions of the country.In national goitre control prog iodine supplementation is implemented in subhimalayas + Northern regions. Iodized oil:- IM injection of poppy seed oil of 1ml dose will provide protection for 4 years.Children who are hypothyroid from birth or before are called cretins. Endemic cretinism :- It is ass with endemic goitre and characteristic clinical features which include deaf-mutism,squint,mental retardation,charactersistic spastic or rigid neuromotor disorder(spastic diplegia) and dwarfism.**
46. **Zinc:- Zinc def leads to growth retardation,hypogonadism,anorexia,alopecia,increased susceptibility to infections secondary to defective cell mediated immunity,liver and spleen enlargement,hypogusia(decreased taste sensation),hyposmia,stunted growth in children,dwarfism.Acrodermatitis enteropathica is a rare autosomal disorder characterised by abnormalities in zinc absorption.;clinical manifestations include diarrhoea,alopecia,muscle wasting,depression,irritability,and a rash involving the extremeties ,face and perineum. Hypopigmentation is also seen. Zinc toxicity:- zinc fumes from welding may also be toxic and cause fever chills,excessive salivation,sweating and headache.(*Metal fume fever* (self limited influenza like illness) results from exposure to fumes or smoke of zinc,copper,magnesium,and other volatilized metals.harrison15th ed 1472)**
47. **Cheilosis:- Vit B6,iron and B2 ,THIAMINE IS NOT ASSOCIATED WITH DERMATITIS.**
48. **Triad of glossitis,cheilosis, and corneal vascularisation –B2 deficiency.(oro-oculo-genital syndrome :- angular stomatitis,atrophic glossitis,genital dermatitis, corneal vasularisation).Riboflavin is extremely sensitive to light,and milk should bestored in containers that protect against photodegradation).**
49. **Niacin(vitamin B3):-The term Niacin refers to nicotinic acid and nicotinamide(serve as precursors of coenzymes NAD and NADP). Def causes PELLAGRA ; DDD (Dermatitis ,dementia, diarrhoea) ,The drug isoniazid inhibits the conversion of tryptophan to niacin.Seen in alcoholics,hartnup disease(congenital defects of intestinal and kidney absorption of trptophan) and in patients with carcinoid syndrome(increased conversion of tryptophan to serotonin leading to niacin def). Epithelial skin changes includes a characteristic skin rash that is pigmented and scaling,particularly in skin areas exposed to sunlight.This rash is known as "Casal`s Necklace.",when it rings the neck.Also spastic paretic syndrome seen.**

50. **Vitamin B6 (Pyridoxine) :-Seborrhiec Dermatitis,glossitis,,stomatitis, and cheilosis like with other Vitamin B deficiencies.(Thiamine is not ass with dermatitis), Peripheral neuropathy, In infants ,Diarrhoea ,Seizures and anemia have been reported. Microcytic, hypochromic anemia (Since the first enzyme involved in heme biosynthesis (ALA synthase) requires PLP (pyridoxal phosphate) as a cofactor). Vitamin B6 dependent syndromes that requires pharmacologic doses of ViT B 6 are sideroblastic anemia. (others are gyrate atrophy with chorioretinal degeneration due to decreased activity of the ornithine aminotransferase and cystathionine β - synthase deficiency).**
51. **Vitamin C (Scurvy) :- Deficiency:- impaired formation of maure connective tissue and include bleeding into skin (petechiae,ecchymosis, perifollicular hemorrhages),inflamed and bleeding gums, and manifestations of bleeding into joints,the peritoneal cavity, pericardium, and adrenal glands.In scurvy there are flattened cork screw hairs with surrounding hemorrhage on the lower extremities ,in addition to gingivitis due to lysyl hydroxylase defect as vitamin c is a cofactor for this enzyme,an enzyme involved in posttranslational modification of procollagen that is necessary for cross link formation. In children Vitamin C deficiency may cause impaired bone growth :-{ Wimburger sign in which epiphyseal centres of ossification are surrounded by a white ring }. Costochondral junction becomes prominent in children. (Costochondral jn swelling is also seen in chondrodystrophy)Metaphyseal fractures are common in scurvy.Deficiency of vitamin C is best estimated by buffy coat estimation**. A scorbutic child is listless,anorexic,fretful, and cries on being handled. Bones are tender and the infant is **reluctant to move his limbs**,which are kept in a **frog like position.**This may be mistaken for paralysis**(PSEUDOPARALYSIS).**
52. **Vitamin D and Rickets :-An increase in osteoid volume and thickness and a decrease in calcification of the mineralization front.Therefore mineralisation of the organic matrix is defective..In rickets the growing skeleton is involved;defective mineralisation occurs both in the bone and cartilaginous matrix of the growth plate. The term osteomalacai is usually used for this mineralization disorder in the adults in whom the epiphyseal growth plates are closed. Since vit D DEF leads to insufficient intestinal calcium absorption and hypocalcemia.Since calcium phosphate is necessary for deposition of calcium in the growing bones,decrease In blood levels of calcium,phosphorus,or both,interferes with calcification of the osteiod tissue.A compensatory increase in the osteoblastic activity results in elevation of serum alkaline phosphatse level. Calcification of osteiod tissue is irregular and incomplete.Markers of bone resorption increase when there is secondary hyperthyroidism and excessive bone resorption .In rickets,the most prominent radiologic alteration is seen at growth plates which is increased in thickness(epiphyseal widening),cupped, and hazy at the metaphyseal border owing to decreased calcification of the hypertrophic zone and inadequate mineralisation of the primary spongiosa.Cuppind and splaying on xray is diagnostic of rickets. There is defective mineralisation in epiphysial growth cartilage. Craniotabes is the earliest manifestation of rickets. Costochondral**

junction becomes prominent to give appearance of a rosary (rachitic rosary).The sternum projects forwards (pigeon breasts). Wind swept deformity is seen.Epiphysis of long bones are widened. Quant`s sign (a T shaped depression in the occipital bone) may be present in rickets. Earliest evidence of healing in rickets is provided by radiological examination of growing bone ends.Radiologic and bone densitometric changes are indistinguishable from those in osteoporosis in osteomalacia..Radiolucent bands ,called pseudofractures or loosers zone are seen in osteomalacia.They occur most often where major arteries cross the bones and are thought to be due to the pulsation of these vessels in the undermineralised area.In osteomalacia a decrease in bone density is usually associated with loss of trabeculae and thinning of cortices. Demineralisation is seen in osteoporosis due to imbalance in the resorption and formation of bone. WHILE in osteomalacia there is undermineralisation.

53. **CALCIUM:- An intake of 500 to 800 mg per day is adequate for the body needs from the age of 1 to 10 years.**
54. **Chromium :- *potentiates the action of insulin* in patients with impaired glucose tolerance.chromium def has been reported to cause glucose intolerance,peripheral neuropathy and confusion.**
55. **SELENIUM :- keshan disease is an endemic cardiomyopathy found in children and young woman residing in regions of china where dietary intake of selenium is low.concomitant def of iodine and selenium may worsen the clinical manifestations of cretinism.**
56. **Asphyxia:- The heart rate begins to fall in primary apnoea and continues to do so in case of secondary apnoea.In both instances the infant is not breathing and heart may be below 100 beats per minute. One should not insert the catheter deep in mouth or nose for suction.stimulation of posterior pharynx during the first few minutes after birth can produce a vagal response,causing severe bradycardia or apnoea.**
57. **Bag and mask ventilation :- causes abdominal distension as air or oxygen not only enters the lung ,but also escapes into the stomach via esophagus. Distended stomach presses on the diaphragm and compromises ventilation.So absoluteely contraindicated in cases of diaphragmatic hernia as ventilation was already compromised and has become more after BMV.**
58. **Peripheral cyanosis (acrocyanosis) may be present for a short while after birth even in normal term infants.*BUT CENTRAL CYANOSIS is NOT A NORMAL FINDING in the newborn.*Urine is usualaly passed during or shortly after birth.Systolic murmur may also be a normal finding. Almost 50 percent of children around the age of 5 years may have soft ejetion systolic murmur. If it is accompanied with a normal second sound then it is unlikely to be significant.Before discarding a murmur as of no significance,it is necessary to obtain ECG and thoracic roentenography. Presence of central cyanosis always indicates presence of heart disease if lung disease has been excluded. A number of primitive neonatal primitive reflexes can be elicited in a healthy neonate :- rooting,suckling,grasp,moro`s reflex.Moro reflex is a cochlear reflex. Other minor clinical clinical problems are Erythema**

toxicum(rash disappears spontaneously), Mongolian spots (not related to down`s syndrome,no Rx required ,sseen on lumbosacral region,disappear before first bday,not related to down`s syn), Breast engorgement, vaginal bleeding,hymenal tags(these are normal), Physiological phimosis,Cephalohematoma(never present at birth but gradually develops after 12-24 hours,the swelling is limited by suture lines,it is fluctuant,the rim may persist for as long as 6 months,Release of bilirubin from Hb present in the cephalhematoma can caues exaggeration of physiological jaundice),Caput succedaneum (not limited by suture line,doesnot have well defined margins ,crosses midline,disappears 24 hours after birth). Liver is palpable but not kidneys in newborn.

59. **Expressed Breast Milk:- can be stored at room temp for 10 hours, in a refrigerator for 24 hours and a freezer at -20˚ C for 3 months.**
60. **All are seen in hy[othermia in early neonates except :- a) Bradycardia b) sclerema c) Excess shivering d) Metabolic acidosis .Ans is c) excess shivering not seen ,Heat gain in newborn is by conduction,convection,and radiation ,in addition *to NON Shivering thermogenesis*.**
61. **Incubators for the management of hypothermia :- Convection warmed incubators, Radiant incubators. *There are no conduction based incubators.***
62. **Clinical Hazrads of Prematurity because of physiological handicaps:- The functional immaturity of the liver leads to hyperbilirubinemia.As the preterm infant is deficient in brown fat,it cannot produce heat in response to cold stress.Therefore preterm babies are more prone to develop hypothermia. The glomerular filtration rate and the concentrating ability of the kidney tubules are reduced. These infants are more prone to develop acidosis.**
63. **Management of LBW infants :- Birth weight > 1800 g : home care if the baby is otherwise well. Birth weight 1500-1800 g : secondary level newborn unit . Birth weight < 1500 g : Tertiary level newborn care for intensive care.**
64. **Neonatal Sepsis :- Early onset and late onset sepsis.Meningitis is the most common manifestation of late onset type.Group B streptococcus (strep agalactiae,also produces CAMP factor) is the most common organism isolated in early onset sepsis and also is the most common cause of bacterial meningitis.(Robbins 471 6th ed).Preterm Delivery,Early rupture of membranes(>24 hr before delivery),Prolonged labour,Fever,or chorioamnionitis are some of the risk factors for group B strep infection. Usual source of the organisms infecting a neonate is the mother`s birth canal. Lethargy in a 2 year old infant strongly suggests infection.*The most common and characteristic manifestation is an alteration in the established feeding behaviour*.(ghai 161) Bacterial meningitis in neonates in the newborn and the first 4 to 6 months of life has many atypical features Neck Rigidity and kernig`s sign are seldon prominent.Anterior Fontanelle may or may not be bulging.Infants present with fever lethargy and poor feeding;it is the most common sign in neonatal meningitis.**Symptoms and signs which should arouse suspicion of bacterial meningitis in children are :- 1)Vacant Stare;2) alternating irritability and drowsiness;3) persistent vomiting with fever 4) refusal to suck 5)

poor tone 6) poor cry 7) shock 8) fever or hypothermia 9) tremor or convulsions 10) neurological deficits of varying type. **Q:- In neonatal septicemia all of the following are used as diagnostic indicators except A) Leucocytosis B) Positive CRP C) Immature neutrophils 20% total D) ESR raised ans. is A) ???????**

65. **Necrotising Enterocolitis :-Earliest change seen in Xray is non-specific dilatation.But the most characteristic finding in Xray is gas in the intestinal wall(pneumatosis intestinalis).Pneumoperitoneum on abd xray may be seen due to intestinal perforation. Bowel sounds are diminished,abdominal distension present. Breast milk is protective for NEC. *Almost all patients of neonatal necrotising enterocolitis are artificially fed prior to the onset of illness.***
66. **Intrauterine infections :- Congenital syphilis :- Transmission of T pallidum from a syphilitic woman to her fetus across the placenta may occur at any stage of pregnancy,but the lesions of congenital syphilis generally develop after the fourth month of gestation when fetal immune response begins to develop.In Congenital syphilis best test is IgM FTA-ABS. Three types of congenital syphilis according to their timing of manifestations. ♠ 1) Early manifestations (early syphilis)(like secondary syphilis) :- Appear *within first 2 years* (often *between 2 and 10 weeks* of age) are infectious and resemble the manifestations of severe secondary syphilis in the adult ; The earliest sign is Rhinitis or snuffles which is soon followed by vesicles and later condylomata lata.The most common early manifestations are bone changes including osteochondritis,osteitis,and periostitis. ♠ 2) Late Manifestations (late cong syphilis)(like tertiary or late syphilis):- is that which remains untreated *after 2 yrs of age*. Cardiovascular syphilis rarely develops in late congenital syphilis but is common in acquired late syphilis of adult whereas interstitial keratitis is much more common in congenital late syphilis.Eighth nerve deafness and recurrent arthropathy are commonly ass with interstitial keratitis. Bilateral knee effusions called Clutton`s joints are present.Gummatous periostitis are also present.Neurosyphilis also seen. ♠ 3) Residual stigmata include :- Hutchison`s teeth(centrally notched ,widely spaced ,peg shaped upper central incisors,Mulberry molars,Saber shins(anterior tibial bowing), Rhagades (linear scars at the angle of mouth and nose that are caused by secondary bacterial infection of the early facial eruption). Congenital Rubella :-The age group most severely affected by rubella infection is UNBORNchild. Fetal infection and malformations not only are more common after maternal infection in the first trimester but also tend to be more severe and to involve more organ systems. Percentage of fetuses infected when maternal infection occurs in < 11 weeks of gestation :-90 % according to park. According to greenwood 70% , according to harrison 50% .but both says about first trimester as a whole and included 12th week also.?????. In the fourth month of pregnancy the risk reduces to approximately 20% and the only abnormality likely to be seen is sensorineural deafness. After 16 week(4th month) of pregnancy,although fetal infection still ocuurs,congenital abnormalities are very infrequent and no more likely to occur than in an apparently uncomplicated pregnancy. Before conception is unlikely to harm the fetus. Pepper salt retinitis seen in congenital rubella. Most common**

defect in congenital rubella is deafness.After Major cardiovascular manifestations are :- PDA, Pulmonic valvular and /or arterial stenosis, atrial septal defects. {NO VSD}. Cataracts ,deafness ,microcephaly,thromboctopenia are major non cardiac manifestations .Congenital toxoplasmosis :- In pregnancy if the mother becomes infected during the first trimester the incidence of transplacental infection is lowest,but the disease in the neonate is most severe.If maternal infection occurs during the third trimester,the incidence of transplacental infection is greatest but the infant is usually asymptomatic at birth.There is essentialy no risk if the mother becomes infected ≥ 6 months before conception. If conception is acquired < 6 months before conception,the likelihood of transplacental infection increases as the interval between the infection and conception decreases.Chorioretinitis, Diffuse cerebral calcification. Congenital CMV infection :- The most common cause of congenital viral infection . Petechiae ,hepatosplenomegaly,and jaundice are the most common presenting features. Microcephaly with or without cerebral calcifications, IUGRand prematurity are seen in 30-40% of cases. Inguinal hernias and chorioretinitis are less common.*It is impossible to predict which fetus will sustain life threatening CMV infection.***Congenital and neonatal herpes:- If herpes seroconversion occurred early in pregnancy,the risk of transmission to the newborn was very low.In women who acquired genital herpes shortly before delivery,the risk of transmission was high.It is recommended that pregnant women with active genital herpes lesions at the time of presentation in labour be delivered by cesarean section.**

67. **Respiratory Disorders :- HMD :- RDS almost always occurs in *preterm babies* often less than 34 weeks of gestation.It is the commonest cause of respiratory *in a preterm neonate.* In addition to prematurity, asphyxia ,acidosis, *maternal diabetes* and cesarean section can increase the risk of developing RDS. In the absence of surfactant,surface tension increases and alveoli collapse during expiration(Surfactant helps to reduce surface tension in alveoli.) RDS usually occurs within First 6 hours of life. Radiological features include *reticulonodular pattern,ground glass opacity,*low lung volumes, *air bronchogram*, and a whiteout lung in severe distress. Prenatal diagnosis can be made my determining L/S ratio in the amniotic fluid. Phosphatidyl glycerol estimation provide a reliable diagnosis.The best test in diabetic mothers to diagnose baby lung maturity. Meconium aspiration syndrome :- Usually occurs in Post mature and SFD babies. Chest X ray shows hyperinflation and *patchy infiltrates*. Transient Tachypneas of newborn:- Onset of resp. distress is immediately after birth and it rarely lasts beyond 48 hours.Occuring usually in term neonates. It is usually due to delayed clearance of lung fluid. Xray shows *prominent interlobar fissure* and prominent vascular markings. Prognosis is good. Diaphragmatic hernia :- should be suspected in any neonate who has *severe RD* and has a scaphoid abdomen. *Pulmonary hypoplasia* is also seen. This condition can be detected during antenatal USG. Chest x ray shows *presence of bowel loops in the thoracic cavity*.**

68. **Neonatal Jaundice :-*One Gram Hb Yields 35 mg Of Bilirubin.* Slight increases in serum bilirubin are best detected by examining the sclerae which have a particular affinity for bilirubin due to their high elastic content. The presence of scleral icterus indicates a serum bilirubin of atleast 3.0 mg/dl .However in Newborns jaundice is detected only when the bilirubin level is more than 5 mg/dl. In jaundice yellow discoloration of the skin is uniformly distributed over rhe body,in carotenoderma the pigment is concentrated on the palms ,soles, forehead, and nasolabial folds. Carotenoderma can be distinguished from jaundice by the sparing of the sclerae. Bilirubinuria,dark colour of urine indicates an elevation of the direct serum bilirubin fraction and therefore the presence of liver disease(conjugated).Unconjugated bilrubin is never transferred in urine nomatter how high the content is.Bilirubin formed in RE cells (mainly in spleen and some in liver) is virtually insoluble in water and therefore noncovalently bind to albumin. It is soulubilized in ER of liver cells by conjugation. While bilirubin is a tetrapyrrole pigment,Urobilinogen is a colourless tetrapyrrole.Urobilinogen is unconjugated bilirubin formed by the action of normal gut bacteria when the conjugated reaches the distal ileum , colon and hydrolyzed there and about 10-20% of it is absorbed passively completing the enterohepatic circulation by reaching the liver through portal vein.About 80-90% of urobilinogen is excreted in feces either unchanged or oxidised to orange derivatives called Urobilins. A Small fraction of urobilinogen (usually less than 3mg/dl) escapes the hepatic uptake ,filters across the renal glomerulus and is excreted in urine.**
69. **ISOLATED HYPERBILIRUBINEMIA (only bilirubin is elevated,other liver tests normal):- Indirect (unconjugated) : Crigler-najjar types I & II , & Gilbert`s syn(a very mild type) Direct (conjugated) :- Dubin johnson and Rotor . Causes of isolated direct hyperbilirubinemia are only two. But of isolated unconjugated i.e. indirect are many others apart from these hereditary causes like hemolytic disorders, ineffective erthropoiesis,Drugs and various causes of decreased hepatic clearance of bilirubin in newborn(physiologic jaundice,breastmilk jaundice,drugs like novobiocin gentamicin etc.)**
70. **<u>Hyperbilirubinemia with other liver tests elevated</u> :- Hepatocellular conditions(viral hepatistis,drugs,alcohol etc) , cholestatic conditions(intrahepatic & extrahepatic) . Patients with a hepatocellular process generally have a disproportionate rise in the aminotransferases compared to the alkaline phosphatase.**
71. **<u>1) Physiological jaundice :-</u> Most neonates develop mild unconjugated hyperbilirubinemia between 2 and 5 days after birth.Peak levels are typically 5-10 mg/dl,never more than 12 in term or never more than 15 in preterm and decline to normal adult concentraion within 2 weeks when the neonate liver assume full resposibility for bilirubin clearance and excretion. Many aspects of hepatic physiology are incompletely developed at birth and while in uterus maternal liver through placenta is involved in bilirubin clearance. Once confirmed physiological jaundice doesn`t require any treatment. 2) <u>This</u>**

physiologic jaundice can exaggerate if the neonate is premature(as if liver is also more profoundly immature in additon to prematurity) and in hemolysis(erthroblastosis fetalis) or in cephalhematoma ,hypothyroidism,vitamin K induced hemolysis with very high unconjugated bilirubin levels in some of these conditions. A rapidly rising levels in excess of 20mg/dl puts the infant at risk to kernicterus.Kernicterus is a condition in which bilirubin crosses an immature blood brain barrier and precipitates in the basal ganglia and other areas of brain.Cerebral cortex is generally spared. In newborn,Brain damage may be associated with *serum bilirubin protein ratio of more than 3.5.* 3) Acquired Conjugation Defects :- A modest reduction in bilirubin – conjugating capacity may be observed by the use of various drugs such as pregnanediol,novobiocin,chloramphenicol, and gentamicin by inhibiting UGT1A1 activity. Finally ,certain fatty acids and the progestational steroid 3 α,20 β – pregnanediol, identified in breast milk but not the serum of mothers whose infants have excessive neonatal hyperbilirubinemia,This is known as breast milk jaundice.It should be differentiated from Lucey-Driscoll syndrome(trasient familial neonatal hyperbilirubinemia) in which a UGTA1A1 inhibitor is found in maternal serum.

72. **Hereditary defects in bilirubin conjugation. :- Unconjugated :- A) CN type 1 characterised by *striking*,very severe unconjugated hyperbilirubinemia of about *20 to 45 mg/dl*. No response to phenobarbital and bile consists of unconjugated bilirubin mostly(>90%) and therefore colourless. .Bilirubin UDP glucoronyltransferase (UGT1A1)activity ABSENT . B) CN type II characterised by marked unconjugated hyperbilirubinemia of usually less than 20 (6-25) mg/dl with a *characteristic increase in monoglucoronides* in bile.UGT1A1 activity ≤ 10% normal. Phenobarbital decreases bilirubin by > 25%. C) Gilberts syndrome is characterised by Mild Unconjugated hyperbilirubinemia of *usually ≤ 4mg/dl/* in the absence of fasting and hemolysis..More elevated values like those of CN type II are associated with stress,fatigue,alcohol use, reduced caloric intake and intercurrent illness.While increased caloric intake and the use of enzyme inducers like phenobarbital produce bilirubin levels to almost normal. UGT1A1 activity is 10-35% of normal.There is also a *moderate increase of lipofuscin pigement* in some in the liver.**
73. **FAMILIAL Defects in hepatic excretory function :- Conjugated (Bilirubinuria will be present characteristically here as hyperbilirubinemia is conjugated) :- A) Dubin johnson syndrome`s cardinal feature is the accumulation of dark,coarsely granular pigment in the lysosomes of centrilobular hepatocytes.**Mutation in MRP2 gene.Biliary excretion of number of anionic compounds is also compromised like in case of BSP ,a synthetic dye with aa rise in its plasma conc.(due to reflux of conjugated BCG).Dyes such as Indocyanin which are not further conjugated or metabolised but are taken by hepatocytes do not show this kind of reflux phenomenon**. B) Rotor Syndrome :- Predominant rise in conjugated bilirubin.**Molecular basis remains unknown.Although the BSP clearance is decreased but there is no back reflux of

conjugated BSP.In rotor syndrome,the gall bladder is usually visualized on oral cholecystography but there is nonvisualisation of gall bladder in dubin johnson syndrome.

74. **Commonest cause of pathological hyperbilirubinemia in INDIA IS Rh incompatibility. Most common cause of jaundice in a newborn within 1st 24 hours of life is erythroblastosis fetalis.Idiopathic infantile hepatitis is commonest cause of cholestatic jaundice in a newborn. Neonatal jaundice First appearing in 2nd week : cause can never be Rh incompatibilty,also called erythroblastosis fetalis.(appears immediately,as early as 30 minutes after birth).**
75. **Persistent elevation of indirect bilirubin is the first sign of congenital hypothyroidism in neonates.This is due to decreased activity of UDPGT for weeks or months birth.**
76. **Phototherapy :- In phototherapy toxic 4 z- 15 z bilirubin is converted to 4z 15 E Called as E Photoisomerisation.. Complications of Phototheray :- 1) *Increase in insensible water loss* especially in premature infants.In addition , *stools tend to be more loose and frequent*. This loss must be compensated for by increasing the fluid intake by 25% over that required before phototherapy.Babies under phototherapy should have a regular measurement of temperature and get weiged twice daily. 2) Animal studies indicates retinal degeneration.*It is therefore,that the eyes of all newborns exposed to phototherapy be covered with sufficient layers of opaque material.* 3) *Bronze baby syndrome* :- The skin urine and the srum become brownish black after several days of phototherapy.It is seen more often in neonates with conjugated hyperbilirubinemia. Babies recover fully after several days once the therapy is discontinued.**
77. **Estimation of blood sugar is relevant in all except :- a) birth asphyxia b) large for date babies c) baby of hypothyroid miother d) Rh incompatibility . Answer is C) ??????????? Hypoglycemia may be secondary to perinatal stresses ,such as asphyxia,infection,respiratory distress and neurological disturbances. Classical transient neonatal hypoglycemia is observed in infants with small for gestational age. Relative hypersinsulinemia in infants of diabetic mothers (large for date babies) may lead to neonatal hypoglycemia.**
78. **An 80 day old baby developed jaundice with features suggestive of non-pathological jaundice.The level of bilirubin indicative of phototherapy is:- a) 9 b) 12 c) 15 d) 18 . Answer is 18 ??? (15-18 for term babies according to ghai 174).**
79. **Hypoxic ischemic encephalopathy (HIE) :- 8 DAY old neonate with extensor posture – H. I. E. Central nervous diseases that cause weakness generally produces spasticity an increase in tone due to uppermotor neuron disease predominantly affecting antigravity muscles i.e upper limb flexors and lower limb extensors , a type of decorticate rigidity because most of the brain stem is intact (in true decerbrate rigidity all four limbs show extensor response cf ganong 214..(in bats its opposite ☺ , just decerebrate them and the resonse in flexor rather than extensor in all four limbs.)**

80. Neonatal seizures :-Most common cause is HIE in neonates. Other important causes are hypocalcemia and hypoglycemia,Inbon error of metabolism like Phenylketonuria,urea cycle disorders,MSUD,GALACTOSEMIA,homocysteinemia.

81. <u>Inherited disorders of amino acid metobolism</u>:- Almost all are transmitted as autosomal recessive traits.in general these disorders are named for the compound that accumulates to highest concentration in blood or urine i.e. emias or urias. For many disorders(aminoacidopathies) ,the parent amino acid is found in excess ; for othres,generally referred to as organic acidemias,products in the catabolic pathways accumulate.*Metabolic acidosis ,often accompanied by hyperammonemias,is a frequent finding in the disorders of branched chain amino acid metabolism and also in glutaric aciduria type 2...*Organic acidemias include Glutaric aciduria (not a branched chain disorder but a catabolic pathway product accumulation of parent aminoacid leucine),and most of the branched chain disorders like isovaleric acidemia,methylmalonic acidemias,Maple syrup urine disease(classic branched chain ketoaciduria)(see harrison 2301 -2305).Methylmalonic acidemia:-methylmalonic aciduria is due to defect in metabolism of methylmalonic acid(methylmalonic acid is one of the product of branched chain amino acid isoleucine & valine,so parent amino acid is not accumulated but methylmalonic acid,that's y organic acidemias)..Sweaty feet syndrome is associated with glutaric aciduria(typeII) & isovaleric acidemia. Maple syrup odor is seen in Classic branched chain ketoaciduria(parent amino acid are valine ,leucine and isoleucine,all branched chain). Decerbrate rigidity is charactersitic feature of MSUD.there is rapid and progressive degeneration of nervous system.

 # In phenylketonuria accumulated amino acid is phenylalinine which itself is parent amino acid.There is tendeny to hypopigmentation and eczema.if untreated in infancy can lead to severe mental retardation.Thats why screening is done in ameica and europe using GUTHRIE BACTERIAL INHIBITION ASSAY(ferric chloride).

 # IN Homocystinuria , a sulfur containing amino acid homocystine is accumulated in blood and urine. It may be due to reduced activity of cystathonine β synthase , an enzyme in transulfuration pathway that converts homocsteine to cysteine and requires PLP.(Pyridoxal phosphate).These patients should be treated by restriction of methionine in the diet starting very early in life and large doses of pyridoxine. The other forms are due to impaired conversion of homocysteine to methionine , a reaction catalysed by methionine cysnthase and 2 essential cofactors,methyltetrahydrofolate (folic acid) and methylcobalamin(vit b12) ..Methionine should not be restricted in these two forms and folic acid,vit b 12 given. Homocysteine acts as an atherogenic and thrombophilic agent.(an independent risk factor).In addition hyperhomocstenemias and folate and vitamin B12 def. have been associated with an increased risk of NTDs in pregnant women. Methylmalonic acidemia is also noted in deficiency of cobalamin . Patients with type I i.e CBS def. have skeletal changes(oseoporosis), ectopia lentis and thromboembolic phenomenon. While

type 2 and 3 (vit b 1 and folate responsive respectively) have megaloblastic anemia & Mental retardation.

 # Alkapatonuria :- (Def of homogenistic acid oxidase leads to excretion of large amounts of homogenistic acid in urine and accumulation of oxidised homogenistic acid pigment in connective tissues(ochronosis).it is rarely noticed before the age of 20 to 30 years.so mainly recognised in middle life. Occasionaly patients develop pigmented prostatic or renal calculi. X rays of lumbar spine show degeneration and dense calcification of the intervertebral discs and narrowing of the intervertebral spaces.(bamboo like appearance).

 # Cystinosis :- In the infantile form abnormalities are usualy apparent by 6 to 10 months of age.cystinosis must be onsidered in any child with vitamin d resistant rickets,the fanconi syndrome or glomerular insufficiency. Cystine deposits in the cornea and retinal degeneration may be present. In contrast patients with adult form have only ocular abnormalties with photophobia,headache and burning or itching of the eyes.in adults glomerular and tubular function and the integrity of the retina is preserved..

TABLE 36
National Immunization Schedule

a. *For Infants*	
At birth	- BCG and OPV-0 dose
(for institutional deliveries)	
At 6 weeks	- BCG (if not given at birth)
	- DPT-1 and OPV-1
At 10 weeks	- DPT-2 and OPV-2
At 14 weeks	- DPT-3 and OPV-3
At 9 months	- Measles
b. At 16-24 months	- DPT and OPV
c. At 5-6 years	- DT - the second dose of DT should be given at an interval of one month if there is no clear history or documented evidence of previous immunization with DPT
d. At 10 and at 16 years	- *Tetanus Toxoid* - The second dose of TT vaccine should be given at an interval of one month if there is no clear history or documented evidence of previous immunization with DPT, DT or TT vaccines
e. *For Pregnant Women*	
Early in pregnancy	- TT-1 or Booster
One month after TT-1	- TT-2

82. Vaccination:-

At 10 And 16 Years :- TT vaccine is given.

1. **<u>Valid Contraindications</u> of DPT :- Encephalopathy within 72 hrs after dose ,seizures within 3 days after dose.Persistent inconsolable crying lasting ≥ 3 hr within 48 hr after dose.Temp more than 105 F Within 48 hr after dose.Collapse or shock like state (hypotonic-hyporesponsive state) within 48 hr after dose.**

82. <u>Invalid contraindications</u> :- Family history of convulsions. /sudden infant death syndrome/ family history of an adverse event following vaccinnation.

83. Anaphylactic reaction to vaccine or its constituent is valid contraindication to all general vaccines (DPT, OPV, IPV, MMR, Hib, HBV, influenza ,Pneumococcus, and varicella). Mild to moderate local reaction,mild acute illness,with or without low grade fever,current antimicrobial therapy,convalescent phase of illness and prematurity ,recent exposure to an infectious disease ,*history of penicillin or other nonspecific allergies or family history of such allergies are invalid contraindications.*

84. While most of the general vaccines can be used in HIV infected persons whether symptomatic or asymptomatic But in OPV there is increased risk of vaccine virus proliferation and paralytic polio;IPV used for household

contacts of HIV Infected persons. ***Live attenuated MMR vaccine can be administered to this group,but OPV cannot.*** Live attenuated vaccines are normally contraindicated in immunocompromised patients,including those with congenital immunodeficiency syndromes and those receiving immunosuppressive therapy. Passive immunisation with immunoglobulin preparations or antitoxins can be considered in individual cases, either as post exposure prophylaxis or as a part of treatment of established infection.

85. **Live attenuated vaccines :- BCG, Typhoid oral (Ty21A), Plague, Oral Polio, Yellow fever(17 D), Measles, Rubella, Mumps, Influenza, Epi Typhus. Inactivated or Killed Vaccines :- Typhoid(TAB), Cholera, Pertussis, Rabies, salk , influenza, hepatitis B, JE, KFD.**
86. **Diphtheria and Tetanus are Non human antisera (immunoglobulins) while hepatitis A & B,measles, rabies,mumps varicella are human immunoglobulins.** ***Antitoxins prepared from non human antisera(against tetanus, diphtheria,botulism, gas gangrene, and snake bite) are still the mainstay of passive immunisation.***
87. **Live Vaccines should not normally be given for 12 weeks (3 months) after an injection of normal human Ig and if a live vaccine has already been given, NHIg injection should be deferred for 2 weeks. High doses of immune globulin may inhibit the efficacy of measles and rubella vaccines, and an interval of atleast 3 months is recommended between the administration of immunoglobulin and that of MMR vaccine or its components.**
88. **Although Live virus vaccines in general should be withheld durig pregnancy,polio and yellow fever virus vaccines are exceptions.** ***Known pegnancy is considered contraindication to the receipt of rubella, measles,meninngococcal, mumps and varicella vaccines.*** **Pregnant women can safely receive tetanus as well as diphtheria toxoid.**
89. **Most effective vaccine is yellow fever vaccine.**
90. **Chickenpox:- Pleomorphic rash,Vesicles involve the corium and dermis,with degenerative changes characterised by ballooning. `Patients are infectious approx. 48 hour prior to the onset of the vesicular rash,during the period of vesicle formation(which generally lasts 4 to 5 days) and until all the vesicles are crusted. So scabs are not infectious.** Few patients deveops a prodorme 1 to 2 days before onset of exanthem**.(Quick prodormal and quick evolution of rash)** .Appear on the trunk and face**(centripetal in distribution). The most common infectious complication of varicella is secondary bacterial superinfection of the skin,which is usually caused by steptococcus pyogenes or staph aureus.The most common extracutaneous complication is the CNS. The syndrome of cerebellar ataxia and meningeal irritaion occurs. They are benign in children and doesn't require hospitalisation.** ***Aseptic meningitis*****,encepalitis,transverse myelitis,GBS,** ***and reye`s syndrome*** **can also occur.** ***Varicella pneumonia*** **is the most serious complication following chickenpox developing more commonly in adults. Perinatal varicella is associated with a high mortality rate when maternal disease develops** ***within 5 days before delivery*** **or wihin 48 hrs thereafter.**

91. **Herpes zoster also called shingles is characterised by a unilateral vesicular eruption within a dermatome,often associated with severe pain.The dermatomes from *T3 to L3* are *most frequently involved*.If the *ophthalmic branch of trigeminal* is involved,zoster ophthlamicus develops.**
92. **Unilateral vesicular lesions in a dermatomal pattern should lead rapidly to the diagnosis of herpes zoster.Patients with hodgkins disease and non hodgkin`s lymphoma are at greatest risk for progressive herpes zoster.Cutaneous dissmenination develops in about 40% of these patients. Patients with herpes zoster benefit from oral antiviral therapy,as evidenced by accelerated healing of lesions and resolution of zoster associated pain with acyclovir,valacyclovir,or famciclovir.**
93. **Complications of chickenpox are:- 1) Meningitis 2) Pneumonia 3) Reyes syndrome {no GIT complaints Like enteritis or pancreatitis} 4)Bleeding diathesis;thrombocytopenia 5) Myocarditis etc.**
94. **Measles:- (Morbillivirus) The virus cannot survive outside the body for any length of time.The only source of infection is a case of measles.Carriers are not known to occur. Infants are protected by maternal antibodies upto 6 months of age , in some,maternal immunity persists beyond 9 months. A line of conj. Inflammation on lower eye lid margin called steiner`s line seen in measles.Multinucleated giant cells with inclusion bodies in the nucleus and the cytoplasm (warthin finkeldey cells) are found in respiratory and lymphoid tissues and are pathognomonic of measles. Koplik`s spots also seen just before appearance of rash.Compy`s sign (White patches due to degenerated squamous epithelium occuring on buccal mucosa and gums) is also seen. The characteristic erythrematous, nonpruritic,maculopapular rash of measles begins at the hairline and behind the ears,spreads down the trunk and limbs to include the palms and soles and often becomes confluent.Death due to measles are almost always due to pneumonia. In young children,ottitis media is the most common complication. While pneumonia is more common in adults.Primary giant cells(hechts pneumonia) is most often documented in immunocompromised and or malnourished patients.Bronchopneumonia due to measles occurs due to immunomodulation. Postinfectious encephalomyelitis,transverse myelitis, SSPE also seen.Gastrointestinal complications include gastroenteritis,hepatitis,appendicitis,ileocolitis,mesentric adenitis.{Not pancreatitis}.Other rare complications are myocarditis ,thrombocytopenic pupura.Balognini`s symptom(a feeling of crepitation occuring from gradual increasing pressure on the abdomen) is seen in Measles. Treatment :- Antibiotics + oxygen+ supp measures..(Therapy is largely supportive and symptom based.)**
95. **Rubella :-Complications of post natally acquired rubella are uncommon. Most common complication is polyarthritis(60 % of adult females).**